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24.5.5 The unconscious patient 5901 of mortality in patients with syncope. Specific treatment of cardiac arrhythmia, valvular, and other structural disorders may be re- quired. Cardiac pacing may be indicated for certain arrhythmias, and a last resort in patients with recurrent neurally mediated syn- cope (the decision is now often aided by evidence from implantable loop recorders). Orthostatic hypotension induced by drugs (e.g. vasodilators, diuretics, phenothiazines, tricyclic antidepressants, monoamine oxidase inhibitors) may be improved by drug cessation or substitution. Autonomic failure, either primary or secondary, may be ameliorated by various strategies (e.g. volume expansion, fludrocortisone, graduated compression stockings, midodrine), and these options may also be explored as prophylaxis for recurrent syncope of noncardiac origin, although there is currently no clear evidence in favour of pharmacological treatment with the possible exception of midodrine. Selective serotonin reuptake inhibitors have been found helpful on occasion. A common issue after transient loss of consciousness due to syn- cope relates to driving. Different jurisdictions have differing rules with respect to fitness to drive, which may relate to both driver age and vehicle type (cars, motor cycles; lorries, buses). These stand- ards should be consulted by physicians before advising patients on driving. In the United Kingdom, the Driver and Vehicle Licensing Agency (DVLA) places no restrictions on patients suffering reflex vasovagal syncope, defined as episodes with definite provoking fac- tors, with associated prodromal symptoms, and which are unlikely to occur while sitting or lying. Even recurrent events evoke no sanc- tion provided the ‘3 Ps’ (provocation, prodrome, posture) apply on each occasion. However, if loss of consciousness is likely to be un- explained syncope, patients are debarred from driving for a vari- able period dependent on whether the risk of recurrence is deemed to be high or low, and the type of licence applied for. A licence is revoked or refused for one year for lorry/bus drivers with unex- plained syncope with high risk of recurrence. There are, in add- ition, particular standards for cough syncope (driving to cease until liability to attacks successfully controlled) and for individuals with pacemakers (for further information, see https://www.gov.uk/gov- ernment/uploads/system/uploads/attachment_data/file/435071/ aagv1.pdf). FURTHER READING Alboni P, Furlan R (2015). Vasovagal syncope. Springer, Heidelberg. Bajpai A, Camm AJ (2013). Cardiac causes of syncope. In: Mathias CJ, Bannister R (eds) Autonomic failure: a textbook of

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 be harbouring delayed effects of head injury such as subdural haematomas, or meningitis arising from a basal skull fracture. Investigations After performing resuscitation, obtaining a history (from a witness if necessary), physical examination and bedside tests (e.g. fingerprick blood glucose), further investigation depends on the clinical con- text: (1) coma with focal signs or evidence of head injury—urgent brain imaging by CT or MRI; (2) coma with meningeal irritation but without focal signs—urgent brain imaging and/or lumbar puncture is required; treat before investigation if clinical suspicion of menin- gitis (for example) is high; (3) coma without focal lateralizing neuro- logical signs and without meningismus—the probability of finding a focal abnormality is low; haematological/biochemical tests or a toxin screen

are most likely to provide the diagnosis. Prognosis Brainstem reflexes are the most important clinical signs in defining prognosis: in the absence of sedative drugs, the absence for 24 h of corneal or pupillary reflexes, or of oculovestibular responses, is al- most incompatible with recovery to independence, whatever the cause of coma. Treatment Specific treatment (if any) will depend upon the particular cause of coma, but—whatever the cause—long-term attention is required to the patient's respiration, skin, circulation, and bladder and bowel function, seizures must be controlled, and the level of consciousness should be regularly assessed and monitored. In patients in whom the prognosis is hopeless, the institution and continuation of resus- citative measures is inappropriate and will serve only to prolong the anguish of relatives and carers (see Chapter 17.10). Definition Normal consciousness Consciousness is the state of awareness of the self and the en- vironment when provided with adequate stimuli; normal consciousness is exhibited by those patients who are fully re- sponsive to stimuli and show appropriate behaviour and speech. Patients who are asleep can be roused and then perform normally. Normal consciousness depends on the integration of activity in the ascending reticular activating substance of the brainstem and the neuronal connections between areas of the cerebral cortex. The ascending reticular activating substance determines arousal, which is shown by awakening with eye opening, motor re- sponses, and verbal communication. The content of consciousness, which is the combination of psychological responses to feeling, emotions, and mental activity, is mediated by the cerebral cortex (Fig. 24.5.5.1). Coma Coma is a state of unrousable unconsciousness without any psy- chologically understandable response to external stimuli or inner need. The patient may appear to be asleep but is incapable of re- sponding normally to external stimuli other than by showing eye opening to pain, flexion or extension of the muscles in the limbs to pain, and occasionally grunting or groaning in response to painful stimuli. It occurs when there is damage to the ascending reticular activating substance or bilateral damage to areas of the cerebral hemispheres, or both (Figs. 24.5.5.2–24.5.5.5). Confusion Patients are usually disoriented with lowered attention, an inability to express thoughts, drowsiness, and defects in memory. There is clouding of consciousness characterized by an impaired capacity to think, understand, respond to, and remember stimuli. It is im- portant to differentiate acute confusion from dysphasia, amnesia, acute psychosis, severe depression, or dementia. Confusion is most commonly seen as the result of toxic or metabolic disturbances, par- ticularly in older people. Delirium There is motor restlessness, hallucination, disorientation, and delusion. The patient is often frightened and irritable, and the state can be regarded as profound confusion; both states should alert the doctor to impending coma. Delirium is most commonly seen in patients with toxic or metabolic disorders, but can be mimicked by degenerative brain disease, acute psychosis, and hypomania. Cerebral hemisphere Content of consciousness Brainstem Arousal (on–off switch of consciousness) Cerebellum
Fig. 24.5.5.1 Normal consciousness. Diencephalic herniation Uncal herniation- third nerve palsy
Fig. 24.5.5.2 Supratentorial mass.

 Stupor The patient appears to be asleep and will show little or no spontan- eous activity, respond only to vigorous stimulation, and then lapse back into somnolence. It may be difficult to differentiate stupor from catatonic schizophrenia or severe retarded depression, but in stupor due to organic disease the electroencephalogram (EEG) will always be abnormal. The vegetative state The patient breathes spontaneously, has a stable circulation, and shows cycles of eye opening and eye closure that may simulate sleep and waking, but he or she is unaware of self and environment. It can be seen transiently in the recovery from coma or it may persist to death. This state is usually seen in patients with diffuse bilateral cere-

bral hemisphere disturbance with an intact brainstem, although it can occur with bilateral damage to the most rostral part of the brain- stem. It is most commonly seen after head injury or as the result of hypoxic–ischaemic damage after cardiac arrest. The patient appears to be awake but is unaware, a condition that frequently causes dis- tress to carers and relatives (Fig. 24.5.5.6). The locked-in syndrome Damage to the ventral portion of the pons below the level of the third nerve nuclei causes total paralysis of the limbs and lower cra- nial nerves, but with intact consciousness (Fig. 24.5.5.7). The pa- tient can open, elevate, and depress the eyes but cannot move the eyes horizontally, and there is no voluntary movement or speech. The diagnosis is made when the doctor recognizes that the pa- tient is able to open the eyes voluntarily and allow them to close in response to command, and can therefore respond to verbal and sensory stimuli by blinking. The most common cause is infarction Destruction of cortex and hemispheres Intact ascending reticular activating substance Fig. 24.5.5.6 Vegetative state. Intact ascending reticular activating substance Damage to ventral pons Intact cortex Fig. 24.5.5.7 Locked-in syndrome. Fig. 24.5.5.3 Brainstem lesion—intrinsic. Tonsillar herniation Local brainstem pressure Fig. 24.5.5.4 Brainstem lesion—local pressure. Diffuse cortical injury Fig. 24.5.5.5 Bihemispheric damage.

section 24 Neurological disorders 5904 of the ventral pons, usually in a patient with hypertension, although it can also be seen with pontine tumours and multiple sclerosis, in central pontine myelinolysis after profound hyponatraemia, and after head injury. The prognosis is poor, although some patients re- cover, usually with residual spasticity. An EEG may help by showing an alert state, reactive to external stimuli, and neurophysiology can be used to exclude similar incapacities occurring in myasthenia gravis or the Guillain–Barré syndrome. Psychogenic unresponsiveness The term ‘pseudo coma’ or psychogenic unresponsiveness is used for patients who appear to be unconscious and in a coma but who are not. The simplest way to identify this condition is to undertake oculovestibular testing (see next), which will reveal the presence of nystagmus and indicate that the patient has an intact brainstem and cortex. The management of the patient in a coma History Once the patient is stable it is important to obtain as much infor- mation as possible from those who accompanied the patient to hos- pital or who observed the onset of the coma. The circumstances in which consciousness was lost are of vital importance in helping to identify the diagnosis. Generally, coma is likely to present in one of three ways: the predictable progression of an underlying illness; an unpredictable event in a patient with a previously known disease; or a totally unexpected event. In the first category are patients who deteriorate after focal brainstem infarction or those with known intracranial mass lesions who show similar deterioration. In the second category are patients with recognized cardiac arrhythmia or a known risk factor for sepsis. In the final category, it is important to distinguish whether there has been a previous history of seiz- ures, trauma, febrile illness, or focal neurological disturbances. The history of a sudden collapse in the midst of a busy street or office indicates the need for different investigations from those required when the patient has been discovered at home in bed surrounded by empty bottles that previously contained sedative tablets. Where there is uncertainty, a telephone call to relatives and medical attend- ants may be useful. Clinical assessment and examination Estimation of the temperature, pulse, blood pressure, and respira- tory rate, and examination of the skin, cardiovascular system, chest, and abdomen may often yield important clues in establishing the cause of a loss of consciousness. Fever, although not diagnostic, will usually indicate the presence of a systemic infection, menin- gitis, encephalitis, or abscess; seizures increase the likelihood of the last two diagnoses. Hypothermia is most commonly seen after exposure to low environmental temperatures, intoxication with al- cohol or barbiturates, the presence of peripheral circulatory

failure or profound myxoedema: tachy- or bradyarrhythmias, evidence for valvular heart disease, or peripheral emboli raise the possibility of a cardiogenic cause; bruits over the carotid vessels suggest cerebro- vascular disease; and splinter haemorrhages suggest endocarditis or collagen vascular disease. Hypotension raises the possibility of shock, myocardial infarction or septicaemia, and Addison's disease should be considered. Hypertension is less helpful as a clinical sign because it may be seen both as the result of cerebral insult or as an indicator of hypertensive encephalopathy. The odour of the breath of an unconscious patient may indicate the presence of alcohol, a ketotic fetor raises the possibility of dia- betes, and the fetor of hepatic or renal failure provides important clues. Clubbing of the fingernails suggests the possibility of a re- spiratory or gastrointestinal abnormality, and evidence of tracheal deviation, fluid in the chest, or collapse of the lung suggests the pos- sibility of a respiratory cause. In the abdomen the finding of enlarge- ment of an organ might indicate portal hypertension, polycystic kidneys, and an associated subarachnoid haemorrhage, or abnor- mality in the blood-forming organs. The general colour of the skin and mucous membranes might reveal anaemia, jaundice, cyanosis, or the pink discoloration of carbon monoxide poisoning. Purpura suggests a bleeding diathesis and bruising around the head indicates the possibility of trauma or a base-of-skull fracture. A rash may in- dicate an infective or inflammatory disease and hyperpigmentation raises the possibility of Addison's disease. The presence of puncture wounds might identify an individual who is diabetic or a recre- ational drug user. Neurological examination This requires observation and an assessment of reflex responses. The position, posture, and spontaneous movements of the patient should be noted; the skull and spine should be examined with testing for neck stiffness and Kernig's sign to identify meningeal irritation. Ophthalmoscopy will identify papilloedema, fundal haemorrhages, emboli, and subhyaloid haemorrhages; it must be remembered that the absence of papilloedema does not exclude raised intracranial pressure. The ears and oral cavity should be examined. Level of consciousness The level of consciousness must be documented by the initial ob- server and can then be monitored by medical and nursing staff to determine the progress of the patient and identify the need for fur- ther investigation, therapy, and decision. The most useful hierarch- ical grading scale to assess the level of consciousness is the Glasgow Coma Scale (GCS) in which the patient's response to graded stimuli of eye opening, motor response, and verbal response are recorded (Table 24.5.5.1); all four limbs are observed for responses to pain and the best response is recorded, although asymmetry should be noted and may identify lateralization. The scale measures conscious- ness and it is possible to score gradations from the fully conscious patient (eye opening, 4; motor response, 6; verbal response, 5) to the totally unresponsive patient (eye opening, 1; motor response, 1; verbal response, 1). If the level of consciousness can be shown to be improving, then urgent decisions may be delayed, but if de- terioration occurs it is imperative that a decision be made about management. Brainstem function The brainstem reflexes identify those lesions that affect the reticular activating substance and determine the viability of the patient. Most of the reflexes involve the eyes and the pattern of respiration,

 although the latter may be compromised by requirements of ventilation. Pupillary reactions Unilateral dilatation of a pupil with lack of a light response suggests uncal herniation of the temporal lobe over the tentorium cerebrum entrapping the third nerve or due to distortion of the brainstem; it may also be seen with a posterior communicating artery aneurysm or other third nerve damage. Midbrain lesions typically cause loss of the light reflex with pupils in the midposition, lesions in the pons cause small pupils with retained light responses, and fixed dilata- tion of the pupils suggests significant brainstem damage but must be differentiated

from the fixed dilatation caused by atropine-like agents instilled by earlier observers. Horner's syndrome may be seen with lesions in the hypothalamus or brainstem, but can also be seen with damage to the wall of the carotid artery. Small pupils that react briskly to light raise the possibility of metabolic causes of coma such as hepatic or renal failure; drug intoxications tend not to affect the pupillary light responses. Corneal responses The corneal reflex is usually retained until a very deep coma occurs; if absent in a patient who appears to be otherwise in a light coma, there is a distinct possibility that the cause may be drug intoxication. The loss of the corneal reflex in the absence of drug overdose is a poor prognostic indicator. Spontaneous eye movements Conjugate deviation of the eyes suggests a focal hemispheric or brainstem lesion, depression of the eyes is seen with damage to the midbrain at the level of the tectum, and skew deviation of the eyes suggests a lesion at the pontomedullary junction. Incoordinate eyes suggest damage to the ocular motor or abducent nerve in the brain- stem or pathways, but a minor degree of divergence of the eyes is normal in the unconscious patient. Patients in a light coma will often have normal roving eye movements, similar to those of sleep, which may be conjugate or dysconjugate. They cannot be mimicked and, when present, exclude the possibility of psychogenic unresponsive- ness, when eye movements are likely to be more jerky. Reflex eye movements are important in assessing brainstem ac- tivity. The oculocephalic response obtained by rotating the patient's head from side to side and observing the position of the eyes is likely to show doll's eye movements when the brainstem is intact, but the eyes will remain in the midposition of the head when the brainstem is depressed. Oculovestibular testing is undertaken by the installation of 50– 200 ml of ice-cold water into an external audi- tory meatus. The conscious patient, and those in psychogenic coma, will develop nystagmus with the quick phase away from the side of the stimulation, indicating an active pons and intact corticopontine connections. A tonic response with conjugate movement of the eyes towards the stimulated side indicates an intact pons and suggests a supratentorial cause for the coma, whereas a dysconjugate response or no response at all implies a lesion within the brainstem. Respiration The techniques of ventilation limit the value of observation of res- piration in patients with coma, but, if testing is possible before res- piration is controlled, then deep breathing suggests acidosis, regular shallow breathing is consistent with drug overdose, long-cycle Cheyne–Stokes respiration suggests damage at the level of the di- encephalon, and short-cycle Cheyne–Stokes respiration damage at the level of the medulla. Central neurogenic hyperventilation oc- curs with lesions in the low midbrain and upper pons, and reflex responses such as yawning, vomiting, and hiccoughing may occur with brainstem disturbances. Motor function Motor function is assessed as part of the level of consciousness in the GCS, but lateralizing abnormalities are important and indicate the likelihood of a focal cause, although they may occasionally be seen in the context of hepatic encephalopathy or hypoglycaemia. The presence of generalized or focal seizures implies hemispheric damage and may help in lateralization; multifocal myoclonus sug- gests a metabolic or anoxic cause with diffuse cortical irritation. Table 24.5.5.1 Neurological observation and assessment Glasgow coma scale Score Eye opening Spontaneous 4 To speech 3 To pain 2 Nil 1 Verbal response Orientated 5 Confused conversation 4 Inappropriate words 3 Incomprehensible sounds 2 Nil 1 Best motor response Obeys 6 Localizes 5 Withdraws 4 Abnormal flexion 3 Extension response 2 Nil 1 Brainstem function Pupillary reactions Corneal responses Spontaneous eye movements Oculocephalic responses Oculovestribular responses Respiratory pattern Motor function Motor response Muscle tone Tendon reflexes Seizures

 Investigation After performing the resuscitation, history, examination, and assess- ment, the doctor should identify one of the three following states Table

24.5.5.2). Coma with focal signs or evidence of head injury In such patients, whether the focal signs indicate a brainstem or supratentorial problem, a CT scan or MRI should be undertaken. A normal scan may be seen in patients with hypoglycaemia or hep- atic coma and the presence of structural pathology will be identified, allowing a decision to be made about the indications for surgery or other therapy. Coma with meningeal irritation but without focal signs Such patients most commonly have subarachnoid haemorrhage, acute meningitis, or meningoencephalitis as the cause of their coma. Brain imaging is the ideal investigation to identify the presence of subarachnoid blood and exclude the possibility of focal collections. Depending on the results of the scan, a lumbar puncture may be per- formed to provide diagnostic information. If the suspicion of men- ingitis is high, then treatment with antibiotics should be started and, in the absence of focal signs or papilloedema, lumbar puncture may precede imaging. Coma without focal lateralizing neurological signs and without meningismus Most patients will have suffered diffuse anoxic–ischaemic disease, metabolic derangement, or drug insult. It may be necessary to undertake imaging techniques but the probability of finding a focal abnormality is low, and it is more likely that haematological or bio- chemical tests or a search for toxins in the blood will provide the diagnosis or help identify an episode of ischaemia or hypoxia in the past. There may occasionally be an indication to undertake a lumbar puncture in such patients to exclude an inflammatory or infective cause. Patients who are in a coma as the result of a drug overdose will usually be identified from the history and the circumstances of dis- covery, but the possibility of drug-induced coma should always be considered in patients without focal signs and without meningismus. The discrepancy in marked depression of brainstem responses in a patient who appears to be in a relatively light coma suggests a drug- induced coma, the importance of which is that such patients have a good prognosis provided that they are given adequate respiratory and circulatory support during the coma. Prognosis The prognosis of individual patients depends on the aetiology, depth of coma, duration of coma, and certain clinical signs. Aetiology Following a head injury, prognosis depends on the presence of intra- cranial haematoma, the age of the patient, and the severity of the sys- temic injury and its effects. Patients in a coma after a drug overdose have, in general, a good prognosis provided that they are adequately resuscitated and protected. Patients who are in a coma as a result of causes other than head injury or drug overdose for a period of more than 6 h have only a 10% chance of making a good recovery. Those who have had a subarachnoid haemorrhage or stroke have a less than 5% chance of making such a recovery, and those with hypoxic Table 24.5.5.2 Investigations to identify the coma state Diagnostic category Investigations Causes Coma with focal signs ± Papilloedema CT scan or MRI Haematoma (extradural, subdural, parenchymal) Hemiparesis Chest radiograph Infarction Brainstem signs Tumour Focal seizures Abscess Rarely metabolic Coma with meningismus ± Fever ± Imaging Meningitis ± Fundal changes Lumbar puncture Encephalitis Blood tests Subarachnoid haemorrhage Diffuse head injury Cerebral malaria Hypertensive encephalopathy Coma alone History Blood tests Drug overdose Systemic signs ± Lumbar puncture Hypoxic ischaemia Electroencephalogram Metabolic (diabetes, hepatatic, renal, and so on) Toxic (alcohol, carbon monoxide) Epilepsy

 or ischaemic injury, typically after cardiac arrest, about 10% chance. Those with metabolic or infective causes have almost a 30% chance of making a good recovery. A vegetative state is most likely to occur after head injury or hypoxic–ischaemic damage. Depth of coma Patients with no response to eye opening, no focal response to pain, and a poor response to pain have a poorer outcome than those who respond with eye opening, grunting, and flexion of the limbs. Duration of the coma When patients have been in a coma for 6 h about 12% may make a good recovery, those who remain in a coma for 24 h have only a 10% chance of

recovery, and at the end of a week only 3% of patients can be expected to make a good recovery. In general, patients who remain in a coma for more than 7–14 days either die or enter a continuing vegetative state. Clinical signs Brainstem reflexes are the most important clinical signs in defining prognosis; the absence of corneal or pupillary reflexes or of oculovestibular responses for 24 h, in the absence of sedative drugs, is almost incompatible with recovery to independence whatever the cause of the coma. Most brainstem reflexes are useful indicators of a poor prognosis but some, such as the development of nystagmus and oculovestibular testing or vocalization of any recognizable word within 48 h, identify patients with a good chance of recovery. The value of investigations Although the bedside tests of eye opening, motor response, and brainstem reflexes proposed by Plum et al. and formulated by Levy (see Further reading) have been the mainstay of assessment of the patient in coma in the accident and emergency department and intensive care unit (ICU) for three decades, there is increasing evi- dence that biochemical values and neurophysiological tests have a role to play. Resting state EEG correlates with outcome early in coma but is relatively insensitive. The value of the N20 wave in somatosensory-evoked potential recording (N20 SSEP) and meas- urement of neuron-specific enolase (NSE) in peripheral blood at varying time intervals in coma have been shown to be prognostic in the PROPAC study in centres in the Netherlands. It is suggested that the bilateral absence of N20 SSEP, or the finding of NSE serum levels greater than 33 µg/litre, have a higher positive likelihood ratio and a lower false-positive rate than the clinical signs of absence of pupil- lary and corneal reflexes in predicting a poor outcome. There are reservations in accepting the conclusions reached from this study because only patients who remained unconscious at 24 h were included and ‘poor outcome’ was defined as death or remaining unconscious at 1 month, and it was assumed that the chance of re- covery of consciousness in patients remaining unconscious for 1 month was virtually nil. Nevertheless, there is logic in looking for biological parameters to identify prognosis in coma and, despite the difficulty in obtaining standardized measures of NSE and complex evoked potentials in the circumstances of most ICUs, there is the suggestion that the bilateral absence of N20 SSEP or the presence of serum NSE greater than 33 µg/litre might be used to define fu- tility of the continuance of care. At present using such criteria would result in litigation. It is, nevertheless, evident that a reproducible and validated biological marker would improve decision-making in the early management of the patient in a coma and avoid prolonging an insentient life. Further validation of such measures in large studies is warranted. Continuation of care The long-term care of patients in a coma may be undertaken in an ICU, on a specialist ward, in a rehabilitation unit, or in a long-stay hospital. It is important that those in whom prognosis is hopeless should not be permanently exposed to the rigors of intensive care medicine but should continue to receive basic care within routine hospital wards or a more long-stay environment. So long as patients are considered to have a potential for recovery they should be looked after in an ICU or a specialist ward. Their respiration, skin, circulation, and bladder and bowel function need attention, seizures must be controlled, and the level of consciousness should be regularly as- sessed and monitored. It is important that the mobility of joints and circulation to pressure areas are maintained during the long-term care of the patient, and the possibility of aspiration pneumonia, peptic ul- ceration, and other complications of long-term intensive care needs to be avoided. Techniques such as mechanical ventilation and steroid therapy should not be used routinely in the management of coma- tose patients; they do not improve prognosis and may compromise recovery. Investigations are of little help in identifying long-term prognosis because various types of EEG pattern have been recorded from patients in prolonged coma and CT scans simply show cortical atrophy with ventricular dilatation. Some somatosensory-evoked re- sponses have been reported to show loss of the cortical component in long-term unconsciousness and positron emission tomography (PET) is reported to show metabolic

underactivity but, at present, neither test can provide decisive information as to prognosis. Several studies have demonstrated an important role for func- tional neuroimaging in the identification of residual cognitive func- tion in the persistent vegetative state. These studies may be useful where there is concern about the accuracy of the diagnosis and the possibility that residual cognitive function is undetected, but the tests are extremely complex and subject to methodological and theoret- ical difficulties. Standardization of such techniques, including those assessing residual auditory function with a combination of PET and functional MRI (fMRI), remains a research tool and is unlikely to have a clinical role in the near future. Some authors believe fMRI can demonstrate speech perception, emotional processing, and con- scious awareness in some patients who behaviourally meet all the criteria that define permanent vegetative state. At present it is im- possible to estimate precisely the sensitivity and specificity of EEG- and fMRI-based technologies for the evaluation of consciousness. FURTHER READING Bates D (1991). Defining prognosis in medical coma. J Neurol Neurosurg Psychiatry, 54, 569–71. Bates D (1993). The management of medical coma. J Neurol Neurosurg Psychiatry, 56, 589–98.

Revision #1
Created 2026-01-22 16:43:37 UTC by Omar Ayman
Updated 2026-01-22 16:43:37 UTC by Omar Ayman