

Assessment of coma and impaired consciousness: A critical review

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Summary

Clinical scales has been evolved for assessing the depth and duration of impaired consciousness and coma. Three aspects of behaviour are independently measured: motor responsiveness, verbal performance, and eye opening. These can be evaluated consistently by doctors and nurses and recorded on a simple chart which has proved practical both in a neurosurgical unit and in a general hospital. The scales facilitates consultations between general and special units in cases of recent brain damage due to traumatic / non traumatic causes and is useful in defining the duration of prolonged coma, also helps in the diagnostic, prognostic and therapeutic index of altered consciousness.

Introduction¹⁻¹⁵

Coma is defined as a state of unconsciousness from which an individual can't be aroused by ordinary verbal, sensory, or physical stimuli. Prompt assessment, diagnosis and appropriate management may be life saving. A wide range of conditions may be associated with coma or impaired consciousness. Apart from acute brain damage due to traumatic, vascular or infective lesions, there are metabolic disorders such as hepatic or renal failure, hypoglycaemia or diabetic ketosis and also drug overdose and cerebral malaria. In gauging deterioration or improvement in the acute stage of such conditions, as well as in predicting the ultimate outcome, the degree and duration of altered consciousness usually overshadow all other clinical features is importance. It is therefore vital to be assess and to record changing states of altered consciousness reliably.

Coma scales, particularly the Glasgow coma scale (GCS), are widely used to assess patients with traumatic and non-traumatic coma. In patients with malaria, coma scaled are used to assess the degree of neurological dysfunction caused by the infection & the motor component of the GCS is used as a criterion for cerebral malaria, working in Blantyre coma scale (BCS). The BCS is like the GCS in that it has 3 components, but it measures different eye and 'verbal responses. This scale is widely used to define and assess the severity of cerebral malaria and to determine the prognosis and outcome of therapeutic trials.

This scale, however, has not been formally validated. Furthermore, neither the BCS nor the World Health Organization (WHO) definition of cerebral malaria takes into account the inability of young infants to localize pain, which is important in interpreting the clinical data in this age group.

Impaired consciousness is an expression of dysfunction in the brain as a whole. This may be due to agents acting diffusely, such as drugs or metabolic imbalance or to the combination of remote and local effects produced by brain damage which was initially focal. Such focal brain damage may affect some of the responses which are used to assess the level of consciousness, and any scale devised for general use must allow for this possibility. A simpler scale might suffice for metabolic or drug coma, when the likelihood of structural brain damage is small, but in an emergency there may be insufficient information to assign patients confidently to a particular diagnostic category. Moreover, coma of mixed origin is not uncommon, as when head injury is suspected of being associated with ingestion of drugs or alcohol or with a vascular accident. These seem good reasons for devising a generally applicable scheme of assessment. The development of equipment for monitoring various functions in critically ill patients has not altered the need for doctors and nurses to assess the level of

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consciousness. There is an abundance of alternative terms by which levels of coma or impaired consciousness are described and recorded. Systems for describing patients with impaired consciousness are not consistent. Indeed, many clinicians retreat from any formal scheme in favour of a general description to the patient's state, without clear guidelines as to what to describe and how to describe it.

In practice, such unstructured observations commonly result in ambiguities and misunderstandings when information about patients is exchanged and when groups of patients treated by alternative methods are compared, or reported from different centres. There is no general agreement about what terms to use, nor are those in common use interpreted similarly by different workers. Almost every report of patients in coma offers yet another classification. Most divide the spectrum of altered consciousness into a series of steps like stupor, coma etc., which in the reports reviewed the scales ranged from 3 to 15 and were often described in terms which defined clear definition. Many assume the existence of clinical features which are unique to each level, whilst others distinguish between coma and consciousness on the basis of only one aspect of behaviour.

The importance of careful and complete neurological examination in determining the nature and site of the lesion causing coma has been described at length by Fisher, Plum & Postner who emphasised that tests of brain stem function, not usually included in routine examination, can be useful in the diagnosis of stupor or coma. Neither, however, was primarily concerned with repeated bedside assessment of the degree of conscious impairment. We are reviewing the assessment of coma and impaired consciousness critically.

The brain and consciousness¹⁶⁻¹⁸

Research into the mechanics of the mind reveals how the brain creates consciousness. With the help of new techniques for mapping brain function such as magnetic resonance imaging (MRI) and positron emission topography (PET) which display images of

the brain at work and new psychological techniques to probe the relationship between mind and behavior, are gradually discovering the neurobiological tricks behind the brain's amazing ability to learn and remember, to use and comprehend language, and to experience happiness and grief, pleasure and pain. One by one nature is yielding the secrets underlying the biology of memory, perception and problem solving- even the biological underpinnings of social behavior and coming into the clear. Can the very mechanisms of consciousness itself be far behind?

Consciousness is not an illusion and neither is the mind. The subjective feeling we call "consciousness" is perfectly real and it is a reality that requires explanation. Although the study of subjective interior phenomena such as consciousness poses special problems, these can be overcome with the appropriate scientific methods cognitive experiments help as do MRI and PET technologies. Consciousness is unlikely to be a supreme and unsolvable scientific mystery forever elusive and impossible to explain. At least some of the secrets behind the critical mechanism of consciousness are gradually being revealed.

An intelligent conversation about consciousness cannot take place without first announcing clearly which phenomenon is being discussed. The need for sharp distinction is even more important for those actually investigating these phenomena. Consciousness is not conscience, nor is it merely the contents of the mind. Consciousness is the biological phenomenon that permits us to survey the contents of our minds: our feelings, thoughts and knowledge. Consciousness is not the feelings themselves, nor is it merely the accumulated thoughts and knowledge. It is an elaborate write of passage into these feelings, these thoughts and this knowledge into everything that makes us human.

Research on consciousness have found it helpful to make a distinction between essential consciousness or "awareness and extended consciousness or "consciousness" proper. Both are internal to the mind, but

awareness is more basic than consciousness and the latter depends on the former. Both awareness and consciousness occur automatically; an individual will neither make them happen nor prevent them from happening. Awareness lets a living organism sense that the contents of its thoughts. Obviously all human beings have awareness, but we would venture to say that many animals do as well. Consciousness proper encompasses a broader canvas of thoughts that portrays not just the organisms present, but its past and anticipated future too. Nonetheless, consciousness proper performs the same task for this larger mental faculty as awareness does for its much narrower range: it places mental contents in an individual perspective; and it gives the owner the sense that the owner can act on them.

These conditions are ideal for research on the neural mechanism underlying the difference between awareness and consciousness. Using PET or MRI imaging, it will be possible to determine with some reliability what permits him from point but prevents him from being aware of the thing to which he points.

In spite of the very real difficulties, the dream of understanding consciousness is likely to come true. It will probably happen gradually rather than suddenly, but are even now coming closer to discovering how we know that we know.

Coma in the paediatric patient¹⁷⁻²¹

Management of coma in children needs a thorough physical examination. The patient's airway & cardiorespiratory system must be examined immediately, and the vital signs must be recorded. If the patient is in shock or has had a cardiorespiratory arrest, the immediate management is directed to resuscitation & to the establishment of life support system. Generally, a child with a Glasgow coma scale of seven or less should be intubated and placed on a respirator. On the other hand, if the patient's vital signs and cardiovascular system are intact, attention may be directed to the history and physical examination.

The history may indicate the cause, but frequently the parent is unavailable or was not present at the onset of the coma. It is important to determine whether there has been a gradual change in personality & behavior or an abrupt loss of consciousness. The amount, type, and time of the last dose of insulin in the diabetic child is important to document. Because intoxication is a cause of coma in the toddler and adolescent patient, a careful review of medications & their location at home should be completed. Furthermore, the discovery of the child in close proximity to the medicine cabinet or storage area or the finding of pills and empty medication containers is overwhelming evidence of drug-induced coma. If there is any doubt about the history or if the clinical and laboratory findings do not support the history, a home visit may be invaluable. An altered state of consciousness in the newborn period associated with vomiting, failure to thrive, and seizures suggest an inborn error of metabolism. The patient in a postictal state following an initial seizure and the child with a history of chronic renal disease associated with hypertensive encephalopathy may present with coma. A child with severe pulmonary or heart disease or profound anemia may develop coma as a consequence of cerebral anoxia and ischemia. Rarely, brain tumors or cerebral abscess, particularly if there is rupture into the ventricular system, may produce sudden coma. These children may have a history of headache, vomiting, change in personality or congenital heart disease. Acute subarachnoid hemorrhage secondary to a bleed from an arteriovenous malformation causes a sudden alteration in consciousness.

The physical examination is helpful in distinguishing between a metabolic cause and structural cause for the coma. A slow, irregular pulse combined with systemic hypertension indicates increased intracranial pressure or hypertensive encephalopathy. The rate and rhythm of the respiratory pattern provide useful information about the etiology of the coma. Regular and deep hyperventilation (Kussmaul breathing) indicates metabolic acidosis; and irregular, ataxic respiration suggests cerebellar

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herniation. Cherry red discoloration of the face and cheeks is associated with carbon monoxide poisoning. A fruity breath is typical of diabetic ketoacidosis; a putrid odor indicates hepatic coma; and a sweet-smelling urine suggests maple syrup urine disease.

The examination should include a careful search for trauma and should test for the presence of nuchal rigidity. CSF rhinorrhoea, hematotympanum and Battle sign (bruising over the mastoid) are suggestive of a basilar skull fracture. Nuchal rigidity may indicate meningitis, encephalitis, subarachnoid bleed, or herniation of the cerebellar tonsils. Pinpoint pupils are associated with narcotics, barbiturate toxicity, organophosphates, and phencyclidine. Small and irregular pupils suggest a lesion in the pons, and dilated and unresponsive pupils are seen in the post-ictal state, with botulism, and with certain drugs including glutethimide, amphetamine, atropine, cocaine, ethyl alcohol, and mydriatics. A unilaterally dilated and unresponsive pupil in the comatose child indicates herniation of the uncus of the ipsilateral temporal lobe. Check to ensure that a mydriatic was not the cause of the abnormal pupil. The integrity of the extraocular muscles may be tested by the doll's eye maneuver. The fundi must be examined for the presence of papilloedema and retinal hemorrhages.

Brain stem function may be evaluated by ice water caloric testing (unless the tympanic is ruptured). The comatose child with an intact brain stem shows a fixed deviation of the eyes to the side of the stimulus, and the patient with irreversible coma has no response.

Focal neurological signs may be difficult to elicit in the comatose patient. Hemiparesis may be demonstrated by passively flexing the legs and hips. The examiner suddenly releases the extremities. The hemiparetic leg will rapidly fall to an externally rotated position, whereas the normal limb will slowly slide back to the original posture. This maneuver should be carried out with the patient supine and on a flat surface.

The quadriceps may be flattened, and the foot of the affected extremity is externally rotated owing to a decrease in muscle tone. Finally, the hemiparetic extremities may have altered reflexes, changes in muscle tone, and an extensor plantar reflex.

During the initial evaluation an I/V line is established and blood is obtained for a complete blood count, electrolytes, calcium, phosphorus, glucose, creatinine, blood gases, liver function studies, prothrombin and partial thromboplastin, ammonium level, and a toxic screen. It is important to collect and store an additional 5 ml of heparinized blood that can be utilized later if a specific metabolic disease becomes apparent. A thorough knowledge provides a framework to differentiate the various common causes of metabolic coma. If the initial Dextrostix suggests hypoglycemia, 2 ml / kg of 25% dextrose should be given I / V. A urinary catheter is inserted, the urine volume is noted; and a sample is examined for glucose, ketones, and further studies as indicated. A nasogastric tube is placed in position, and the stomach is emptied with care to prevent aspiration, particularly if a toxin is suspected the stomach contents may be analyzed in the laboratory for specific toxins. Structural causes of coma include concussion, contusion, subdural and epidural hematoma, cerebral edema, brain tumors and cerebral abscess.

The principles of treatment include maintenance of the respiratory status, normalization of cardiovascular function, and correction of acid-base, fluid, and electrolyte abnormalities. Seizures, increased intracranial pressure, and hyperthermia (or hypothermia) are managed appropriately. The primary goal of problem in a safe and controlled fashion. The use of invasive intracranial pressure monitoring should be considered for any infant or child with non traumatic coma and suspected increase in intracranial pressure to assess cerebral perfusion and to anticipate shifts in brain tissue. Cerebral perfusion is calculated as the difference between the mean arterial blood pressure and the mean intracranial pressure. Neurologic outcome is improved if the intracranial pressure can be

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reduced and maintained at 1.5 mmHg or less and if the cerebral perfusion pressure is above 50 mmHg. Poor Neurologic outcome or death is associated with intracranial pressures above 50 mmHg or cerebral perfusion pressures of less than 40 mmHg. Intracranial pressure in the child may be monitored by the use of a subarachnoid screw, a subdural pressure transducer, or a fluid-filled intraventricular catheter. Raised intracranial pressure may be lowered by paralysis and sedation with pancuronium, phenobarbital, morphine, or diazepam, mechanical hyperventilation (pCO₂ lowered to 30-35 mmHg), osmotherapy with I/V mannitol or furosemide, or drainage of CSF through the ventricular catheter. A decrease in cerebral perfusion pressure associated with a low systemic arterial pressure may be enhanced by infusions of octaloids or dopamine.

The induction of pentobarbital and the use of steroids does not appear to influence the neurologic prognosis in the comatose child. The prediction of coma outcome during the acute illness depends in part on the etiology of the condition; diabetic ketoacidosis has a more favorable outlook than Reye's syndrome. However, certain physical signs provide some indication of outcome before inducing paralysis and placement on the respirator. These signs include severity of the coma (i.e., modified Glasgow score), eye movement, pupil reaction, level of blood pressure, temperature, motor patterns, and the seizure type. The EEG is also useful to estimate the potential for neurologic recovery. For example, the reappearance of normal sleep spindles is an encouraging finding even if associated with high voltage slow waves that have no predictive value. EEG patterns associated with a poor prognosis include burst suppression and like activity, very low amplitude activity for age, and electrocerebral silence. Neurophysiologic studies have also been used to make a prognosis about comatose children, including brain stem auditory, visual and somatosensory evoked potentials. Generally the absence of all wave forms in these three modalities is associated with death of severe Neurologic residue. Somatosensory evoked potentials are the most

sensitive and reliable method for the evaluation of neurologic outcome in the comatose child.

TYPES OF COMA SCALE ¹⁻²⁷	
The Glasgow coma scale	Score
Eyes opening	
spontaneously	4
to speech	3
to pain	2
none	1
Best verbal response	
oriented	5
confused	4
inappropriate words	3
incomprehensible sounds	2
none	1
Best motor response	
obeys commands	6
localises pain	5
withdrawal (pain)	4
flexion (pain)	3
extension to pain	2
none	1
Total	3-15
Unrousable coma < 7	

Glasgow coma scale¹⁵⁻²⁷

To be generally accepted, a system must be practical to use in a wide range of hospitals and by staff without special training. But the search for simplicity must not be the excuse for seeking absolute distinctions where none exist: for that reason no attempt is made to define either consciousness or coma in absolute terms. Indeed, it is conceptually unsound to expect a clear watershed in the continuum between these states. What is required is an effective method of describing the various states of impaired consciousness encountered in clinical practice. Moreover, this should not depend on only one type of response because this may, for various reasons, be unstable. The three different aspects of behavioral response which we chose to examine were motor response, verbal response, and eye opening, each being evaluated independently of the other. These features in many previous reports on coma but not in the formal system we propose. This depends on identifying responses which can be clearly defined, and each of which can be accurately graded according to a rank order that indicates the degree of dysfunction.

Motor Responses: The ease with which motor responses can be elicited in the limbs, together with the wide range of different patterns which can occur, makes motor activity a suitable guide to the functioning state of the central nervous system. Indeed, every one of the reported scales which we reviewed included some aspect of motor responsiveness as a criterion. Obeying commands is the best response possible, but the observer must take care not to interpret a grasp reflex or postural adjustment as a response to command. The terms “purposeful” and “voluntary” are avoided because we believe that they cannot be judged objectively.

If there is no response to command, a painful stimulus is applied. The significance of the response to pain is not always easy to interpret unless stimulation is applied in a standard way and is maintained until a maximum response is obtained. Initially, pressure is applied to the finger nail bed with a pencil; this may result in either flexion or extension at the elbow. If flexion is observed stimulation is then applied to the head and neck and to the trunk to test for localization in brain death, a spinal reflex may still cause the legs to flex briskly in response to pain applied locally. For this reason, and because the arms show a wider range of responses, it is wise always to test them, unless local trauma makes this completely impossible.

A localizing response indicates that a stimulus at more than one site causes a limb to move so as to attempt to remove it. A flexor response may vary from rapid withdrawal, associated with abduction of the shoulder, to a slower, stereotyped assumption of the hemiplegic or deorticate posture with education of the shoulder. Experienced observers may readily distinguish between normal and abnormal flexion, but for general use in the first few days after brain damage has been sustained it is sufficient to record only that the response is flexor. Extensor posturing is obviously abnormal and is usually associated with adduction, internal rotation of the shoulder, and pronation of the forearm. The term “decerebrate rigidity” is avoided because it

implies a specific physioanatomical correlation. No response is usually associated with hypotonia and it is important to exclude spinal transection as an explanation for lack of response; and also to be satisfied that an adequate stimulus has been applied. When recording motor response as an indication of the functional state of the brain as a whole, the best or highest response from any limb is recorded. During a single examination some patients give variable responses, these usually becoming better as the patient becomes more aroused; responses from the right and left limbs may also differ. Any difference between the responsiveness of one limb and another may indicate focal brain damage and for this purpose the worst response should be noted. But for the purpose of assessing the degree of altered consciousness it is the best response from the best limb that is recorded.

Verbal Responses: Probably the commonest definition of the end of coma, or the recovery of consciousness, is the patient’s first understandable utterance; speech figured in nearly all the reported scales which we reviewed. Certainly the return of speech indicates the restoration of a high degree of integration within the nervous system, but continued speechlessness may be due to causes other than depressed consciousness (e.g., tracheotomy or dysphasia).

Orientation implies awareness of the self and the environment. The patient should know who he is, and why he is there; know the year, the season, and the month. The words “rational sensible” are avoided because they cannot be clearly defined. Confused conversation is recorded if attention can be held and the patient responds to questions in a conversational manner but the responses indicate varying degrees of disorientation and confusion. It is there that verbatim reporting of the individual patient’s responses can be useful. Inappropriate speech describes intelligible articulation but implies that speech is used only in an exclamatory or random way, usually by shouting and swearing; no sustained conversational exchange is possible.

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In comprehensible speech refers to moaning and groaning but without any recognizable words.

Eye Opening: Spontaneous eye opening, with sleep/ wake rhythms, is most highly scored on this part of the scale and it indicates that the arousal mechanisms in the brainstem are active. But arousal does not imply awareness, and we believe it is unwise to try to decide whether a patient is attentive on the basis of eye movements. Patients in the vegetative state, who are subsequently shown to be structurally decorticate, have often been believed by relatives, nurses, and even by doctors to be reacting visually to people around them; probably primitive ocular-following reflexes may be executed at subcortical level.

Eye opening in response to speech is a response to any verbal approach, whether spoken or shouted not necessarily the command to open the eyes.

Eye opening in response to pain should be tested by a stimulus in the limbs, because the grimacing associated with supraorbital or jaw-angle pressure may cause eye closure.

PAEDIATRIC COMA SCALE 1-12 Modified Glasgow coma scale *Eyes Opening*

Score	>1 Yr	<1Yr.
4	Spontaneously	Spontaneously
3	To verbal command	To shout
2	To pain	To pain
1	No response	No response

Best Motor Response

Score	>1Yr	<1Yr.
6	Obeys	Spontaneous
5	Localizes pain	Localizes pain
4	Flexion-withdrawal	Flexion-withdrawal
3	Flexion-abnormal (Decerebrate rigidity)	Flexion-abnormal (Decerebrate rigidity)
2	Extension (Decerebrate rigidity)	Extension (Decerebrate rigidity)
1	No response	No response

Best Verbal Response

The score of Glasgow coma scale has been modified to be applicable to children,

Score	>5Yr.	2-5 Yr.	0-23 Month
5	Oriented and converses	Appropriate words and phrases	Smiles, coos appropriately
4	Disoriented and converses	Inappropriate words	Cries, Consolable
3	Inappropriate words	Persistent cries or screams	Persistent inappropriate crying or screaming
2	Incomprehensible sounds	Grunts	Grunts, agitated or restless
1	No response	No response	No response

including those who have not learned to speak is the Blantyre coma scale (BCS).

Eyes movements:	directed (e.g. follows mother's face)	1
	not directed	0
Verbal response:	appropriate cry	2
	moan or inappropriate cry	1
	none	0
Best motor response:	localizes painful stimulus	2
	Withdraws limb from pain	1
	Nonspecific or absent response	0
Total		0-5
Unrousable coma ≤ 2		

These scales can be used repeatedly to assess improvement deterioration.

- Rub knuckles on patient's sternum.
- Firm pressure on thumb nail bed with horizontal pencil.

The child's level of consciousness and the response to stimuli should be carefully documented. A modification of the Glasgow coma scale is a useful tool for the grading of the degree of coma and the severity of the insult in infants and children. It is important to remember that the assessment of the verbal response is much different from that of the adult and the child's developmental level must be kept in mind during the evaluation. A coma score of less than five is associated with a grave prognosis, where as a score of five to eight may indicate a better prognosis in the child than in the adult.

The Glasgow Coma Scale is widely used to assess impairment of consciousness after head injury, both for clinicar purposes and i-

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international comparisons of methods of treatment. The scale measures three easily understood neurological reactions: eye opening, verbal responses, and limb movement. For each reaction, a score in points is given, the higher being the better. The three scores can be aggregated to give a coma score or sum, which indicates the level of responsiveness.

Below the age of 10 years, the verbal responses and to a lesser extent the motor responses, are not easily graded. A frightened child may not be willing to say that he knows where he is, though neurologically unimpaired, a normal infant will not speak or obey commands to move hind limbs. Normally a neonate cannot respond vocally to (by coos, babbles, hums) or locate painful stimuli. Only the eye-opening responses can be measured in the standard manner, even in neonates.

A paediatric modification of the Glasgow coma scale used in the Adelaide Children's Hospital since 1977 (Adelaide coma scale) takes neurological immaturity into account:

Adult scale	Paediatric scale (Adelaide coma scale)
(a) Eyes open spontaneously	4
to speech	3
to pain	2
none	1
(b) Best verbal response	As in adult scale
Orientated	orientated 5
Confused	words 4
Inappropriate words	vocal sounds 3
Incomprehensible sounds	cries 2
None	none 1
(c) Best motor response	As in adult scale
Obeys commands	5
Localize pain	4
Flexion to pain	3

We have assumed that, during the first 6 months of life, the best verbal response is normally a cry, though of course some infants make vocal responses during this period. The

expected normal score at this age is therefore 2. Between 6 and 12 months, the normal infant makes noises; the expected normal score is 3. After 12 months, recognizable words are expected, and the normal score is 4; orientation, defined as awareness of being in hospital, is expected by 5 years. Motor responses are recorded as in the original 5-point adult scale (the 6-point scale, which distinguishes withdrawal from abnormal flexion, has been chiefly used in special units). However, before the age of 6 months, the best normal response is usually flexion (score 3), while in the period 6 months to 2 years the infant will usually locate pain but obey commands (score 4).

Birth to 6 months :	9
>6 to 12 months :	11
>1 to 2 years :	12
>2 to 5 years :	13
>5 years :	14

Thus, the normal aggregate score will be as follows

The scales are arbitrary / but accord reasonably well with standard developmental screening tests.

Adelaide and Blantyre (BCS) coma scales compared

Adelaide coma scale	Blantyre coma scale
Motor	
5. Obeys commands	2. Localizes pain
4. Localizes pain	1. Withdraws from pain
3. Flexes to pain	0. No response
2. Extends to pain	
1. No response	
Verbal	
5. Aware of place, able to give name and age	2. Appropriate cry
4. Recognizable and relevant words	1. Inappropriate cry/ moan
3. Incomprehensible, but complex vocalization	0.No cry
2. Cry/ grunt	
1. None	
Eye response	
4. Spontaneous eye opening	1. Directed eye movements
3. Opens eyes to voice	

Discussion¹⁻²⁷

Apart from its practical use in the management of recently brain-damaged patient, this scales allow the duration of coma to be defined more precisely, in terms of how long different levels of responsiveness have persisted. There is evidence that this is a crucial criterion when it comes to predicting the ultimate outcome of coma, particularly after head injury. It would make it possible also to examine critically claims for good recovery after weeks or months "in coma," by enabling the alleged coma to be more accurately assessed. In such cases as we have scrutinized it has clear, even retrospectively, that there had been evidence of much earlier recovery, on at least one component of the coma scale, then had been recognized. By resolving the problem of defining "prolonged coma" the scale also makes it possible to distinguish between the various states which this term embraces, such as akinetic mutism and the persistent vegetative state.

Some may have reservations about a system which seems to undervalue the niceties of a full neurological examination. It is no part of our case to deny the value of a detailed appraisal of the patient as a whole, and of neurological function in particular, in reaching a diagnosis about the causes of coma, or in determining the probable site of brain damage. However, repeated observations of conscious level are usually made by relatively inexperienced junior doctors or nurses; these staff are not only few in number but they change frequently even during the course of a day. There are therefore good reasons for restricting routine observations to the minimum, and for choosing those which can be reliably recorded and understood by a range of different staff.

Different observers were able to elicit the responses in this scale with a high degree of consistency, and the likelihood of ambiguous reporting appears to be small. This was demonstrated by having several doctors and nurses examine the same group of patients. Disagreements were rare. This was in pronounced contrast to that happened when the observers were asked instead to judge

only whether patients were conscious or unconscious; one in five observers then disagreed with the majority opinion. This 20% disagreement-rate compared with rates of 20-35% which have been reported in various different clinical situations, whilst in one study extensor plantar responses showed only 50% consistency when observations were repeated.

One or other components of this scale may be unstable, and this fact can be recorded. Limbs may be immobilized by splints for fractures, tracheostomy may preclude speech, and eyelid swelling or bilateral third-nerve lesions make eye opening impossible. In the rare "locked-in syndrome," a patient with totally inactive limbs may obey commands to move the eyes and may even be able to signal his needs.

The nurses in our intensive-care unit have willingly adopted this method of formalizing observations which they previously used to record as a descriptive comment. They now plot them on a chart somewhat similar in format, but not content, to one proposed by Bouzarth, and which also provides for conventional recording of temperature, pulse and respiration, of the pupil size in mm and of focal motor signs. This method has already been adopted successfully for making observations on head injuries in a neighbouring general hospital. In such hospitals patients with head injuries form a considerable proportion of acute surgical admissions, and observations there depend on medical and nursing staff who have no special experience of neurology and neurosurgery.

The method for assessing patients with impaired consciousness that described almost a decade ago has been widely accepted, and in many centers the eye, verbal, and motor components are summed. Totals up to 8 relate to patients in coma with no eye opening or verbal responses, reflecting changes in motor response; scores from 9 to 15 depend more upon eye opening and verbal responses. Janine Jagger and her colleagues doubt if eye and verbal responses add predictive information. They studied the short-term

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outcome in head-injured patients assessed on admission only. Not surprisingly, they found the motor responses to be most informative; patients who, on admission, show eye-opening and comprehensible verbal responses ought not to die. Death can be expected only amongst patients already in coma due to severe established brain damage.

Such patients would have no eye opening and no comprehensible verbal responses so that their coma score would depend upon the motor response.

Changes in the eye and verbal responses, and thus higher overall scores, are useful in discriminating between patients with less severe impairment of consciousness. Although these patients would be expected to survive, this may be with differing degrees of disability. The Charlottesville group themselves found that increasing scores in the 9-15 range (reflecting improving eye and verbal performances) are associated with a doubling of the rate of good recovery in survivors of head injury. Furthermore, correlations have been established across the whole range of the coma score with cerebral metabolic rate for oxygen, evoked potential studies, and biochemical indices of brain damage.

Head-injured patients may change rapidly after admission, and the eye and verbal responses are useful in assessing improvement or deterioration to show whether a patient is in coma and how long he remains comatose. Scores obtained during the first few days after admission reveal much more about prognosis than do admission scores.

The analysis used by the Charlottesville group is not well suited to comparing the relative predictive power of different clinical features and can exaggerate minor differences. Moreover, they included information about pupil responses and about a haematoma, which could not have been known at the time of admission. Yet they have previously demonstrated correlations between higher coma scores and decreasing frequency of

abnormal pupil responses and CT scan abnormalities in moderately injured patients. Because of this, the inclusion of these features may have masked the information provided by the eye and verbal responses. Their analysis should have been restricted to the three aspects of the coma scale. They would then have found that knowledge of the eye and verbal responses in addition to the motor response does convey extra information, whether the three responses are considered separately or summed.

Although we cannot accept the Charlottesville group's reservations about the value of the eye and verbal components there are limitations inherent in the summation of the three responses. This step assumes an equal weighting for the three responses. More importantly, the information conveyed by the coma score is less than that contained in the three responses separately. This is because the same score may be made up in different ways. Indeed, in Glasgow patients under treatment are always described by the three separate responses and never by the total. The total score is merely a convenient method for summarizing data, especially for a series of patients. Therefore, while we do not favour its use in day-to-day clinical practice, we find no reason to doubt that it will continue to be used widely in the analysis and reporting of a series of patients with head injuries or other forms of acute brain damage.

In order to quantify the level of consciousness, a modification of the 'Glasgow' coma scale is developed that was suitable for use in children too young to have learned to speak the Blantyre coma scale (BCS). The scale uses motor and crying responses to pain, and includes ability to watch; from these three criteria a score can be calculated (minimum=0, maximum=5). Score <2 is taken as Unrousable coma.

For research purposes, however, we do not agree with the use of a summation of the Glasgow coma score (GCS) in the definition of cerebral malaria. The GCS was designed to monitor coma and does not provide a complete neurological description of the patient. The summation of the responses is

unfounded since their values are determined by their rank order and therefore do not depressant discrete quantities, and in summation of these values, information is lost. These criticisms are especially pertinent to the Adelaide modification if the GCS for children (who bear the brunt of cerebral malaria in Africa) since the responses vary with age. However, the scoring system devised by Molyneux et al is useful because of its simplicity and independence of age. The world health organization's definition identifies patients in whom the impairment of consciousness cannot be attributed to fever alone, while the score suggested by Leaver et al does not. Unlike Leaver and colleagues, it has found the motor response of the GCS to be the easiest to interpret and have used this in the initial assessment of consciousness in our young patients with malaria. This was also the experience with Thai adults. Thereafter it is reasonable to use coma scores to monitor progress. It is agreed that reports of neurological involvement in malaria should include a stratification of severity, but this should consist of a clinical description of each category and should not be reduced to a single score. Proposals for new definitions and classifications should include full details of the patients on whom these are based, including the parasitological diagnosis and exclusion of other diseases.

Compared with the Glasgow coma scale (GCS), which is objective, reproducible, easily taught, and of proven validity in traumatic and non-traumatic coma. Warrell et al defined unarousable coma as best motor response- localizes or worse; verbal response- incomprehensible sounds or nil; eye opening to pain or nil. This definition, when scored on GCS, gives score of 8 or less. Any upper limit is arbitrary, but it would argue that a patient with malaria and a GCS of 9 -11 also has cerebral involvement and requires the same management as patients with GCS 8 or less. Such patients would flex or localize in response to painful stimuli; any speech would be confused and disoriented, and the eyes would only open in response to commands or pain.

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