Non-traumatic coma

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INTRODUCTION

Coma or encephalopathy implies a disorder of consciousness (Plum and Posner, 1983). In the UK, in children under 16 years of age, the incidence is around 30 per 100 000 children per year (Wong et al, 2001). There are a range of non-traumatic conditions capable of inducing coma (Seshia et al, 1977). Acute hospital management now follows standardized procedures and protocols that will be familiar to those who have completed either the Advanced Paediatric Life Support or Pediatric Advanced Life Support courses; a requirement for most paediatric senior house officers. This approach is summarized in *Table 1*.

The rest of this article will focus on the causes, emergency assessment and investigation of comatose infants and children once management and stabilization has been initiated. Its purpose is to provide a framework for early investigation.

CAUSES OF COMA

The cause of coma may arise from within or outside the CNS (*Table 2*). Most causes produce a relatively stereotyped array of symptoms and signs that fall into one of four categories.

Generalized depression of hemisphere function

In these patients consciousness is depressed, motor tone becomes diminished, pupils are small but reactive, and reflex eye movements are disinhibited. Asterixis is one of the hallmarks of such metabolic encephalopathy.

Heightened excitability

In these patients heightened excitability of neural tissue occurs and results from a direct lowering of the threshold for neuronal excitability or because of a selective depression of inhibitory influences on neuronal function. Cheyne–Stokes respiration may result from bilateral hemispheric inhibition, and certain types of seizures from neuronal excitability.

Selective vulnerability

In these patients focal involvement of a specific brain region occurs as a result of a systemic metabolic insult. This may be a result of regional differences in tissue metabolic requirements or, alternatively, regional differences in neurotransmitters and receptors. It is not uncommon for focal findings to remain unexplained, e.g. those occurring during hypoglycaemia, hyperglycaemia, uraemia and hypercalcaemia.

Central syndrome

Progressive rostrocaudal deterioration with features and signs indicative of raised intracranial pressure (ICP) and brain tissue shifts may occur with any cause of cerebral oedema (such as toxic–metabolic encephalopathy; *Table 3*) or space-occupying lesion.

HISTORY

A careful history often provides clues to the diagnosis (Kirkham, 2001). Acute central deterioration is associated with metabolic disturbance, ingestions, cerebrovascular accidents or trauma. Cerebral deterioration over days or weeks is more compatible with infection, chronic intoxication or more slowly developing raised ICP (*Table 4*). The presence of focal neurological abnormalities in the child before the onset of central disturbance suggests cerebrovascular disease, an intracranial mass or focal encephalitis.

The past medical history may provide vital clues of recurrent disorders, such as seizures, migraine or sickle-cell disease. A family history of epilepsy or tuberculosis may be discovered, or the report of previous stillbirths or deaths in infancy may indicate inherited metabolic disease. The social history may suggest non-

TABLE 1.
Emergency assessment and management of the comatose child

Assessment	A		Establish Airway and administer oxygen, check for breathing
	B		Measure Blood pressure and resuscitate with saline
	C		Treat Cardiac state with inotropes if necessary
	D		Check Dextrostix and laboratory blood sugar; treat with glucose if low
	E		Evaluate level of consciousness with GCS score
	F		Assess Function of brainstem and Features of eyes and Fundi
Management	GCS	<9	Intubate and mechanically ventilate, thereafter treat accordingly (see below)
		9–12	Give mannitol (0.25 g/kg) if there is evidence of raised ICP
	Seizures		Treat both convulsive and non-convulsive episodes with benzodiazepine ± phenytoin
	Fever	<1 yr	Perform diagnostic lumbar puncture if not deteriorating level of consciousness
		>1 yr	If GCS >12 perform diagnostic lumbar puncture if not in non-convulsive status
Infection suspected or possible			Aciclovir
			Cefotaxime
			Erythromycin

GCS = Glasgow Coma Score; ICP = intracranial pressure

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TABLE 2.
Causes of acute encephalopathy or coma

Trauma	Accidental	Poisoning	
	Non-accidental	Vascular	Arteriovenous malformation
Hypoxic–ischaemic injury	Cardiorespiratory arrest		Hypertensive encephalopathy
	Sudden infant death syndrome		Embolism
	Near drowning		Migraine
	Smoke inhalation		Venous thrombosis
	Shock syndromes		Arteritis
Intracranial infection	Meningitis		Homocysteinuria
	Encephalitis	Endocrine dysfunction	Hypoglycaemia
	Post-infectious		Diabetes mellitus
Mass lesion	Haematoma		Diabetes insipidus
	Abscess	Respiratory failure	
	Tumour	Renal failure	
Electrolyte and acid–base disorders	Hypernatraemia	Hepatic failure	
	Hyponatraemia	Reye's syndrome	
	Water intoxication	Inherited metabolic disorder	Lactic acidosis
	Acidosis		Urea cycle disorder
	Alkalosis		Aminoacidopathies
Acute ventricular obstruction		Hypothermia/hyperthermia	
Seizure disorders		Iatrogenic	Overcorrection of acidosis
Complications of malignancy			Overhydration
Systemic infection	Sepsis syndrome		Drug overdosage

TABLE 3.
Toxic–metabolic causes of cerebral oedema

Inherited metabolic	Aminoacidopathies
	Organic acidaemias
	Hyperammonaemia
	Porphyria
	Non-ketotic hyperglycinaemia
Organ failure	Uraemia
	Hepatic failure
Electrolytes, minerals and vitamins	Hypercalcaemia
	Hypernatraemia
	Water intoxication
	Lead poisoning
	Vitamin A toxicity
Other	Hypoglycaemia
	Hypoxia–ischaemia

accidental injury or lead poisoning, or the family may recently have arrived from the tropics, introducing the possibility of a wide variety of intracranial infectious pathology.

CLINICAL EVALUATION

In common with assessing comatose adults, a full examination is essential in children. The general physical examination may provide signs of systemic disease, infection and trauma. The size and weight of the child might indicate failure to thrive, suggesting a long-standing metabolic disease, or emotional deprivation. The pulse, blood pressure and temperature should be measured and recorded and the breathing pattern observed.

Hyperpyrexia may be central in origin, but is most likely to be the result of infection. Hypothermia may result from shock, hypothalamic disturbance or inadequate measures for warming. Hypertension may be the cause of acute neurology, but conversely may be neurogenic in origin. Hypertension or bradycardia may be a result of increased ICP. Hypotension may be a result of shock or central causes.

Breath odours might suggest diabetic ketoacidosis, solvent abuse, or one of the rare aminoacidopathies. Examination of the skin might suggest a neurocutaneous disorder or yield valuable clues of sepsis, such as the minor abrasion leading to staphylococcal toxic shock, the petechial haemorrhages of meningococcaemia and, where prevalent, rickettsial disease. Bleeding into the skin or from any of the orifices might suggest a bleeding diathesis.

Examination of the head is particularly important in infants and young children. The fontanelles and sutures should be palpated and listening for bruits should not be forgotten. The head circumference should be measured and recorded and, if possible,

TABLE 4.
Early symptoms and signs of raised intracranial pressure

	Infant	Child
General state	Poor feeding	Anorexia and nausea
	Vomiting	Vomiting
	Irritability to coma	Lethargy to coma
	Seizures	Seizures
Head/eyes	Full fontanelle	False localizing signs
	Scalp vein distension	
	False localizing signs	
Other	Altered vital signs	Altered vital signs
	Hypertension	Hypertension
	Pulmonary oedema	Pulmonary oedema

comparison with earlier records made. Distension of the neck veins should be observed and, where there is a ventricular shunt or reservoir in situ, assessment of ventricular CSF pressure should be attempted if the child cannot be aroused.

Evidence of dysraphism should be looked for, particularly in the lumbosacral and occipital regions, as a sinus here might well be a clue to the presence of meningitis. Signs of meningeal irritation must be sought, although these may be absent in the very young infant or in the critically ill child, even in the presence of subarachnoid haemorrhage or meningitis. Inspection of the eyes might reveal proptosis, lid swelling or unilateral exophthalmos. Examination of the nose and external auditory meati might demonstrate CSF rhinorrhoea or otorrhoea. The mouth and pharynx should be examined for any evidence of trauma.

Last, a full neurological examination and fundoscopy should be completed as part of the initial clinical evaluation. The latter needs to be documented as part of the assessment for possible non-accidental injury.

SEVERITY ASSESSMENT AND SCORING

In patients needing frequent neurological review in order to monitor for significant change in level of consciousness, a screening assessment using the Glasgow Coma Scale score (GCS) can be carried out quickly by different bedside attendants (Teasdale and Jennett, 1974; Reilly et al, 1988) (*Table 5*). This scale is a modification of the 'central syndrome' that assigns scores for clinical findings that develop (in the course of deterioration) following head injury; many authors advocate its use in non-traumatic coma.

CLINICAL STRATEGY

General supportive care of the unconscious patient, management of raised ICP, and control of status epilepticus and seizures are the main emergency care problems encountered in the comatose child.

General care

The utmost priority in the management of the comatose child is preservation of vital function, i.e. airway protection, maintenance of ventilation, assurance of oxygenation and support of the circulation. The patient should be positioned to avoid aspiration, suffocation or physical injury. The neck should be protected if there is any question of trauma. Adequate aeration should be ensured, and a plastic airway may be placed if it can be done easily. The forced use of such an airway or the use of tongue blades or metal objects may cause severe oral injury and should be avoided.

In the presence of poor air exchange, the child should be intubated and mechanically ventilated. It is wrong to await the development of florid systemic complications such as cyanosis, severe acidosis or haemodynamic instability before proceeding to intubation. After intubation is accomplished, the child should be placed in 100% oxygen to avoid the development of hypoxia. Subsequent oxygen therapy should be guided by arterial blood gases and other appropriate oxygen monitoring.

General clinical surveillance should include regular observations of motor, sensory and pupillary function; blood pressure, pulse and electrocardiographic state; and oximetry and temperature. In the more severely affected patients, more complete and invasive monitoring should be undertaken, e.g. capnography and central venous pressure monitoring.

Neurological investigations

The initial investigation falls into two broad categories: those investigations which may lead to or confirm a specific diagnosis, and those which provide information which will help to give optimum supportive therapy during the child's illness (*Table 6*). The investigations must be tailored to the individual child's history and examination. For instance, if a clear history of hypoxia has occurred, to organize a full skeletal survey will not only be unhelpful, but may be detrimental, whereas in the child with retinal haemorrhages the investigation is essential. As a general rule, investigations such as arterial blood gases, blood sugar, urea, electrolytes and osmolality should be carried out on admission.

Lumbar puncture

Bacterial meningitis is an important cause of childhood mortality and the need for urgent lumbar puncture (LP)

TABLE 5.
Modified Glasgow Coma Scale

Score		>5 years	<5 years
Eye opening	4	Spontaneous	Spontaneous
	3	To voice	To voice
	2	To pain	To pain
	1	None	None
Verbal	5	Oriented	Alert, babbles, words – normal
	4	Confused	Less than usual ability, irritable
	3	Inappropriate words	Cries to pain
	2	Incomprehensible sounds	Moans to pain
	1	No response to pain	No response to pain
Motor	6	Obeys commands	Normal spontaneous movements
	5	Localizes pain	Localizes pain
	4	Withdraws to pain	Withdraws to pain
	3	Flexor response	Flexor response
	2	Extensor response	Extensor response
	1	No response	No response

if there is the slightest suspicion of this has been standard teaching until very recently (Kneen et al, 2002a,b; Riordan and Cant, 2002).

A number of reports on transtentorial or transforaminal herniation following LP in children with meningitis have appeared in the literature and there is debate about such a policy (Rennick et al, 1993). It is difficult to predict which children are at particular risk of tentorial herniation, but where the diagnosis of meningitis seems clear and there is impaired consciousness, or focal signs or other signs of incipient coning, or when the child has been ill for several days, many clinicians elect to treat the patient with antibiotics without obtaining confirmatory CSF findings.

Collective experience has shown that the great majority of children with pyogenic meningitis come to no harm from LP, but in the seriously ill child where consciousness is depressed, the decision is a difficult one and should be made only by the experienced clinician. In this instance some have recommended that a GCS below 8 is an absolute contraindication to the emergency LP (Rennick et al, 1993; Isaacs, 2002). Where a space-occupying lesion or severe cerebral oedema is expected, this investigation is of course contraindicated.

TABLE 6. Emergency investigations

Those that facilitate supportive therapy	Arterial blood gas
	Urea, electrolytes and creatinine
	Blood sugar
	Osmolality of plasma and urine
	Urine testing for ketones, sugar and pH
	Full blood count
	Coagulation screen
	Infection screen
	Blood levels of anticonvulsants
	Virology
	Chest X-ray
	Electrocardiogram
	Cerebral imaging
Those that might provide a specific diagnosis	CSF examination
	Toxicology screen
	Blood ammonia
	Blood pyruvate and lactate
	Amino acid profile
	Organic acid analysis
	Porphyrins
	Liver function tests
	Skeletal survey
	Cerebral imaging

Cranial imaging

Cranial imaging studies are indicated when the initial biochemical studies have failed to clarify the diagnosis, or if a structural lesion or poor cerebral function is suspected. Cerebral ultrasound scan is a sensitive tool for the detection of major intracranial abnormalities in young infants, but it is not as sensitive as cranial computed tomography (CT) scan in detecting smaller, although still clinically significant lesions such as focal infarcts, subarachnoid haemorrhages and small parenchymal haemorrhages.

Magnetic resonance imaging has advantages over CT scanning, particularly in the assessment of the posterior cranial fossa. In the comatose child cranial CT is invaluable when wanting to assess urgently potential underlying cerebral pathology and need for surgical intervention. In the context of metabolic encephalopathy, the CT scan is particularly useful in the evaluation of cerebral oedema and raised ICP (Tasker et al, 1990; Nadel et al, 1999). The likelihood of the latter can be determined by reviewing the presence or absence of CSF spaces both above and below the tentorium.

CONCLUSIONS

The key points for managing non-traumatic coma in children are listed below. Once specific causes have been identified and treated, the next stage is to ensure adequate support and intervention during recovery and rehabilitation. In many instances children will require detailed assessment which should be part of standard follow up. HM

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KEY POINTS

- Stabilize the child by ensuring adequate cardiorespiratory function and oxygenation.
- Correct and prevent metabolic imbalance of hydration, electrolytes, glucose and lactate.
- Control raised intracranial pressure.
- Stop seizures in a timely manner.
- Evaluate and treat specific causes.