THE POCKET GUIDE TO NEUROCRITICAL CARE:

A concise reference for the evaluation and management of neurologic emergencies

by the

NEUROCRITICAL CARE SOCIETY

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For our families, our patients, and their families.

Thank you for teaching us.

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PREFACE

The Pocket Guide to Neurocritical Care was first conceived by NCS members in training in 2016 after recognizing a need for a succinct reference that reviewed the basics of neurologic emergencies and neurocritical care. Spearheaded by the NCS Resident and Fellow Committee with support from the Educational Products Committee, 40+ resident, fellow, and APP authors were recruited to develop the product with the guidance of established leaders in the neurocritical care field. This publication has become a recognizable part of NCS courses, as well as some subspecialty training courses.

The success of *The Pocket Guide* can be attributed to the original mission of "written by trainees, for trainees." This unique characteristic ensures the content is high-yield, comprehensive, and readily accessible to multiple levels of learning. With this edition, we have continued that mission, taking feedback from trainees, and adding new authors in training including those in pharmacy training. We hope that this will be just the first of their many future publication opportunities.

This book compiles 18 chapters of the highest-yield information as suggested and recommended by providers and trainees across multiple disciplines who all have a common interest in caring for neurocritical care patients. It is not meant to be an exhaustive reference, but to give readers a strong fund of knowledge in neurocritical care to support their practice.

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A special thank you to Angel Gindele who coordinated all the authors, timeline, and editorial needs of this edition. Without her, this could not be the comprehensive and refined product before you.

Finally, we would like to thank our families. We are the best versions of ourselves because of your love and support.

Marin Darsie, Asma Moheet, and Winnie Lau
May 2020

ABBREVIATIONS

+	positive
–	decreased
↑	increased
AAN	American Academy of Neurology
Ab	antibody
ABCs	airway, breathing, circulation
ABG	arterial blood gas
AC	assist control
ACA	anterior cerebral artery
ACh	acetylcholine
AChEI	acetylcholinesterase inhibitor(s)
AChR	acetylcholinesterase receptor(s)
ACEI	angiotensin-converting enzyme inhibitor(s)
ACLS	Advanced Cardiac Life Support
AComm	anterior communicating artery
ADR	alpha delta ratio
AED	anti-epileptic drug
AF	atrial fibrillation
AG	anion gap
AHA	American Heart Association
AICA	anterior inferior cerebellar artery
AIDS	acquired immune deficiency syndrome
AIS	acute ischemic stroke
ALS	amyotrophic lateral sclerosis
AKA	also known as
ARDS	acute respiratory distress syndrome
aSAH	aneurysmal subarachnoid hemorrhage
ASIA	American Spinal Injury Association
ASV	adaptive support ventilation
ATLS	Advanced Trauma Life Support
$AVDO_2$	arterio-venous difference of oxygen consumption
AVM	arteriovenous malformation
BAL	broncheoalveolar lavage
BCx	blood culture
BBB	blood-brain barrier
BID	twice daily
BiPAP	bi-level positive airway pressure
BMP	basic metabolic panel
BP	blood pressure
BTF	Brain Trauma Foundation
BSAS	Bedside Shivering Assessment Scale
C	Celsius
Ca^{2+}	calcium
CAA	cerebral amyloid angiopathy
CABG	coronary artery bypass graft
CAS	carotid artery stenting
CBC	complete blood count
CBF	cerebral blood flow
CCM	cerebral cavernous malformation
CEA	carotid endarterectomy
cEEG	continuous EEG
CHF	congestive heart failure
CI	continuous infusion
CIDP	chronic inflammatory demyelinating polyneuropathy
COPD	**c**hronic obstructive pulmonary disease
CIM	critical illness myopathy
CIWA	Clinical Institute Withdrawal Assessment
CKD	chronic kidney disease
CMP	comprehensive metabolic panel
$CMRO_2$	cerebral metabolic rate of oxygen

CMV	cytomegalovirus
CN	cranial nerve
CNS	central nervous system
CO	cardiac output
CPAP	continuous positive pressure ventilation
CPP	cerebral perfusion pressure
CPR	cardiopulmonary resuscitation
CrCl	creatinine clearance
CSE	convulsive status epilepticus
CSF	cerebrospinal fluid
CSWS	cerebral salt wasting syndrome
CT	computerized tomography
CTA	CT angiography
CTV	CT venogram
CVR	cerebral vascular resistance
CVST	cortical vein sinus thrombosis
CXR	chest x-ray
D	day
DAI	diffuse axonal injury
dAVF	dural arteriovenous fistula
DBP	diastolic blood pressure
DCD	donation after circulatory death
DCI	delayed cerebral ischemia
DDAVP	desmopressin
DH	decompressive hemicraniectomy
DI	diabetes insipidus
DKA	diabetic ketoacidosis
DNI	do not intubate
DNR	do not resuscitate
DOAC	direct oral anticoagulant
DSA	digital subtraction angiography
DTR	deep tendon reflexes
DVT	deep vein thrombosis
EBV	Epstein-Barr virus
ECMO	extracorporeal membrane oxygenation
ECT	electroconvulsive therapy
ED	emergency department
EDH	epidural hematoma
EEG	electroencephalogram
EKG	electrocardiogram
EMG	electromyography
EMSE	epidemiology based mortality score
EN	enteral nutrition
ENLS	Emergency Neurologic Life Support
ENT	ear/nose/throat or otolaryngology
EOM	extraocular muscles
ETT	endotracheal tube
EVD	external ventricular drain
FDA	Federal Drug Administration
FFP	fresh frozen plasma
FiO_2	fraction of inspired oxygen
FOUR	Full Outline of UnResponsiveness
FVC	forced vital capacity
GBS	Guillain-Barré syndrome
GCS	Glasgow Coma Scale
GI	gastrointestinal
GOS-E	Glasgow Outcome Scale-Extended
GPD	generalized periodic discharge
GRE	gradient echo
GTC	generalized tonic-clonic
H	hour
HCG	human chorionic gonadotropin
HD	hemodialysis
Hgb	hemoglobin
HIT	heparin-induced thrombocytopenia

HIV	human immunodeficiency virus
HOB	head of bed
HSV	Herpes simplex virus
HTLV-1	Human T-lymphotropic virus type 1
HTN	hypertension
HTS	hypertonic saline
IBW	ideal body weight
ICA	internal carotid artery
ICH	intracerebral hemorrhage
ICP	intracranial pressure
ICU	intensive care unit
IDSA	Infectious Disease Society of America
IgA	immunoglobulin A
IIC	ictal-interictal continuum
IM	intramuscular
INR	international normalized ratio
IO	intraosseous
I & O	input and output
IV	intravenous
IVC	inferior vena cava
IVF	intravenous fluids
IVH	intraventricular hemorrhage
IVP	intravenous push
IVIg	intravenous immunoglobulin
IVP	intravenous push
IV tPA	intravenous tissue plasminogen activator
KCl	potassium chloride
LE	lower extremity
LCMV	lymphocytic choriomeningitis virus
LDH	lactate dehydrogenase
LFTs	liver function tests
LKWT	last known well time
LMWH	low-molecular-weight heparin
LOS	length of stay
LP	lumbar puncture
LR	Lindegaard ratio
LR	Ringer's lactate
MAP	mean arterial pressure
MCA	middle cerebral artery
MCS	minimally conscious state
MEP	maximal expiratory pressure
MFV	mean flow velocity
MG	myasthenia gravis
MH	malignant hyperthermia
MHS	malignant hemispheric stroke
MI	myocardial infarction
Min	minute
mL	milliliter
MMR	measles, mumps, rubella
MOA	mechanism of action
MRI	magnetic resonance imaging
mRS	modified Rankin Scale
MRV	magnetic resonance venogram
MV	mechanical ventilation
NCCU	Neurocritical Care Unit
NCS	nerve conduction study OR Neurocritical Care Society
NCSE	nonconvulsive status epilepticus
NG	nasogastric
NIF	negative inspiratory force
NIPPV	noninvasive positive pressure ventilation
NM	neuromuscular
NMBA	neuromuscular blockade agent
NMJ	neuromuscular junction
NMO	neuromyelitis optica
NMS	neuroleptic malignant syndrome
NORSE	new onset status epilepticus
NPi	neurologic pupillary index

NS	normal saline
NSAID	nonsteroidal anti-inflammatory drugs
O_2	oxygen
OG	orogastric
OHCA	out-of-hospital cardiac arrest
OOB	out of bed
OSA	obstructive sleep apnea
OSM	osmolar
OT	occupational therapy or therapist
$PbtO_2$	brain tissue oxygen tension
PCA	posterior cerebral artery
PCC	prothrombin complex concentrate
PComm	posterior communicating artery
PCR	polymerase chain reaction
PD	periodic discharge
PE	pulmonary embolus
PEEP	positive end expiratory pressure
PEG	percutaneous endoscopic gastrostomy
PFO	patent foramen ovale
PICA	posterior inferior cerebellar artery
PICC	peripherally inserted central catheter
PIP	peak inspiratory pressure
PIV	peripheral intravenous line
PLEX	plasmapheresis
P_{plat}	plateau pressure
PRBCs	packed red blood cells
PRES	posterior reversible encephalopathy syndrome
PRIS	propofol infusion syndrome
PRN	pro re nata, as needed
PRVC	pressure regulated volume control
PT	physical therapy or therapist
PTT	partial thromboplastin time
PVS	persistent vegetative state
QID	four times daily
QOD	every other day
RA	rheumatoid arthritis
RAAS	renin-angiotensin-aldosterone system
RAS	renin-angiotensin system
RAS	reticular activating system
RASS	Richmond Agitation and Sedation Scale
R/O	rule out
RCT	randomized control trial
RN	registered nurse
ROM	range of motion
ROSC	return of spontaneous circulation
RR	respiratory rate
RSE	refractory status epilepticus
RSI	rapid sequence intubation
RT	respiratory therapy or therapist
RTA	renal tubular acidosis
RVR	rapid ventricular response
SAH	subarachnoid hemorrhage
SBP	systolic blood pressure
SC	subcutaneous
SCA	superior cerebellar artery
SCD	sequential compression device
SCI	spinal cord injury
SDH	subdural hematoma
Se	sensitivity
SE	status epilepticus
Sec	second
SIADH	syndrome of inappropriate antidiuretic hormone secretion
SIMV	synchronized intermittent mechanical ventilation
SLE	systemic lupus erythematosus

s/p status post

Sp specificity

SpO_2 peripheral capillary oxygen saturation

SRSE super refractory status epilepticus

SSEPs somatosensory evoked potentials

SSRI selective serotonin reuptake inhibitor

SSS sick sinus syndrome

ST speech therapy or therapist

STESS status epilepticus severity score

SWI susceptibility-weighted imaging

TB tuberculosis

TBI traumatic brain injury

TCA tricyclic antidepressant

TCD transcranial doppler ultrasound

TH therapeutic hypothermia

TIA transient ischemic attack

TMJ temporomandibular joint

TMP-SMX trimethoprim-sulfamethoxazole

TOF train-of-four

tPA tissue plasminogen activator

TPN total parenteral nutrition

TSH thyroid stimulating hormone

TTM targeted temperature management

U units

UA urinalysis

UCx urine culture

UE upper extremity

UFH unfractionated heparin

UMN upper motor neuron

US ultrasound

UTI urinary tract infection

VALI ventilator associated lung injury

VBG venous blood gas

VC volume control

VF ventricular fibrillation

VPA valproic acid

VS vegetative state

V_T tidal volume

VT ventricular tachycardia

VTE venous thromboembolism

VZV Varicella zoster virus

w/ with

WFNS World Federation of Neurological Surgeons

WNV West Nile virus

w/o without

yo years old

CONTENTS

CHAPTER 1

COMPONENTS OF THE COMA EXAM

Anand Venkatraman & Edward Manno

The examination of a comatose patient is one of the most important responsibilities in the care of neurocritically ill patients. We describe key components of the coma exam and review common findings.

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DISORDERS OF CONSCIOUSNESS

Consciousness is comprised of 2 components: arousal and awareness. Two connected anatomic pathways coordinate consciousness: the ascending RAS within the brainstem, and arousal centers in the bilateral thalami which project diffusely to cortical neurons. Impairment of awareness can lead to a spectrum of disorders, which include MCS and VS. Coma, on the other hand, is caused by impaired arousal which leads to impaired awareness. Consciousness is not an all-or-nothing phenomenon, and gradations do exist. Newer technologies, such as functional MRI, are beginning to provide the ability to image and interpret brain processing in a more advanced and high-resolution fashion. This is shedding light on the gradations of consciousness and may alter how we evaluate and treat patients that may be “functionally locked-in” or have Unresponsive Wakefulness Syndrome, but the bedside neurologic exam remains a highly valuable standard assessment tool for all clinicians.

DIFFERENTIAL DIAGNOSIS

It is important to differentiate coma from other disorders of consciousness, including VS, MCS, and locked-in syndrome (Table 1). The prognosis of disorders of consciousness varies widely, and depends on clinical factors, cause of brain injury, and the duration of the consciousness impairment. For patients with PVS (defined as vegetative state with duration of > 1 month), the prognosis is poorest. Some patients with MCS will show recovery over time. Locked-in syndrome usually results from a lesion that interrupts the descending motor pathways, leaving cognitive function and consciousness intact, but with severe limitations on the patient’s ability to interact with the examiner.

POSSIBLE CAUSES OF COMA

Bihemispheric phenomena: medication or drug toxicities, SE or NCSE, metabolic disorders, meningoencephalitides (w/ or w/o focal neurologic findings).

Focal anatomic brain lesions: affecting the thalamus or brain stem which contain crucial arousal-supporting neurons. May be associated with focal neurologic findings.

It is essential to rule out reversible causes of coma in cases when the etiology is not known (Table 2).

NEUROLOGIC EXAM IN COMA

The initial exam is important for localization and identifying the cause of coma. Serial exams to assess interval change are equally important. Acute neurologic deterioration can signal AIS, ICH, seizure, worsening edema, hydrocephalus, or elevated ICP. Hourly vital sign assessments and neurologic checks are the norm in newly-admitted NCCU patients. In some, such as those admitted after surgical or endovascular procedures, the frequency of assessments may need to be more often.

We recommend the use of standardized scales to assess disorders of consciousness. The best known is the GCS, of which the arbitrary definition of coma is GCS ≤ 8 (E2V2M4). See Table 3 for reference.

Limitations of GCS:

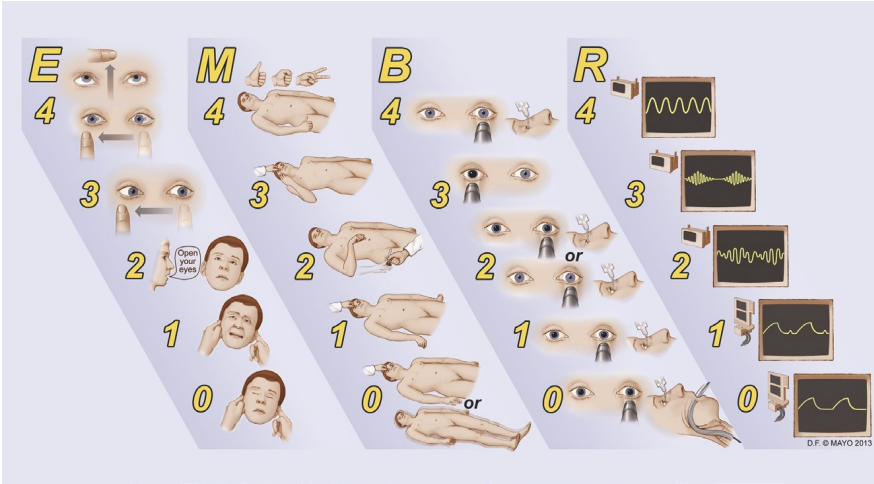
- Can miss locked-in states and subtle changes in consciousness
- Does not assess pupillary and other brainstem reflexes
- Patients with similar scores may go on to have different outcomes
- Assigns greater weight to motor response than eye opening and verbal responses
- Intubated patients default to a T score on the verbal component and aphasic patients have a low verbal score, each of which make the total GCS less reliable
- Some studies suggest only moderate inter-rater reliability, especially for motor response

FOUR score can also be used, and addresses some shortcomings of the GCS:

- Incorporates brainstem function and respiratory pattern, allowing for better localization
- Can help recognize a locked-in state
- Can recognize various stages of herniation

The calculation of the FOUR Score is illustrated in Figure 1 and is also described in Table 4.

Figure 1. Calculation of the FOUR Score (used with permission from the Mayo Foundation for Medical Education and Research)



SPECIFIC STEPS OF THE COMA EXAM

The patient's mental status, cranial nerve exam, motor exam (including response to noxious stimulus), tone, and reflexes should be assessed.

Cranial Nerves (CN)

Pupils:

- *Afferent: CN II, Efferent: CN III*
- Observe pupils in low light. Then, shine a light into both pupils alternately and observe for briskness of response. Assess for both direct and consensual light reflexes. A pupillometer is a useful adjunct, especially with abnormal pupils

Asymmetric pupils: consider compressive lesions of CN III, such as due to herniation and/or PComm aneurysms

- Unilateral dilated, non-reactive pupil: CN III dysfunction (rule out compression) vs unilateral medication effect or post-surgical pupil
- Nonreactive, dilated pupils: consider severe brainstem damage or medication side effect(Table 5)
- Pinpoint pupils: consider opioid use, pontine stroke or hemorrhage, organophosphate poisoning, clonidine overdose, pilocarpine eye drop use, and occasionally mirtazapine and olanzapine
- Sluggish pupils: NMBA, recent mydriatic administration, or albuterol use

Corneal responses:

- *Afferent: CN V, Efferent: CN VII*
- Gently hold the patient's eyelids open and drop 1-2 saline drops onto the cornea of each eye
- Cotton swabs can be used with caution as repeated testing with this method can lead to corneal ulceration

- There is a blinking response if this pathway is intact

Blink to threat:

- *Afferent: CN II, Efferent: CN VII*
- Briskly move your hand into the patient's visual field while holding his/her eyelid open. The patient should blink

Gaze:

- Hold eyes open and observe direction of gaze in neutral head position
- *Eye movements involve coordinated functioning of multiple CN, frontal lobe and brainstem centers*
- Gaze deviation also occurs due to involvement of frontal eye fields in each hemisphere: destructive lesions cause ipsilateral gaze deviation, stimulation causes contralateral deviation
  - Cortical ischemic stroke patients demonstrate gaze directed towards hemisphere of the stroke
  - Seizure patients demonstrate gaze directed away from seizing hemisphere, and may have gaze towards the hemisphere post-ictally
- Brainstem strokes can cause impaired gaze towards the side of the stroke
- Forced downgaze may be seen in thalamic hemorrhages, pineal mass lesions, and severe hydrocephalus
- Bilateral CN VI palsy seen in ↑ ICP

EOMs:

- *Innervation of extraocular muscles: Lateral Rectus CN VI, Superior Oblique CN IV, All others CN III*
- Fixation and tracking are normal findings
- Fixation: eyes looking at an object and not moving from that position
- Tracking: eyes moving as the object or the examiner moves, to follow them
- Roving eye movements: slow and conjugate to-and-fro movements
  - Can be seen in toxic and metabolic conditions where brainstem is intact. Light stages of sleep and lighter coma also cause this
- Nystagmus: fast, beating movements to one side (may indicate ongoing seizures)
  - Other causes: phenytoin toxicity, brain lesions like those seen in stroke or multiple sclerosis, inner ear disorders, and metabolic disorders like thiamine deficiency
  - Down-beating nystagmus may be seen in disorders of the craniocervical junction or cerebellar flocculus
  - Up-beating nystagmus may be seen in cerebellar vermis involvement, and sometimes in lesions of the medulla
  - Acute lesions in the pons can cause rapid downward jerking of the eyes with slow return to normal position, called ocular bobbing

Fundoscopy:

- Evaluate optic disc and nerve
- Blurring of optic disc margins is indicative of ↑ ICP, but absence of blurring does not automatically indicate normal ICP. Subhyaloid hemorrhages can also be seen with ↑ ICP. The presence of spontaneous venous pulsations implies a normal ICP, but the absence of these pulsations is uninformative
  - □ Terson syndrome: subhyaloid hemorrhage in SAH

Oculocephalic reflex or "doll's eyes":

- *Afferent: CN VIII and proprioceptive pathways from the cervical level, Efferent: CN III and VI*
- Confirm stability of cervical spine, then move head briskly in one direction and then the other with the eyelids held open
- Interpretation of OCR responses in a comatose patient:
  - □ In a normal OCR, eyes move conjugately in the direction opposite to head movement
  - □ In abnormal OCR, eyes stay in fixed position in the head, implying brainstem disease

Oculovestibular reflex or "cold calorics":

- *Afferent: CN VIII, Efferent: CN III and VI*
- Do this if OCRs are absent; also useful in cases where cervical spine instability is suspected
- Ensure patency of ear canal and ability of water to reach tympanic membrane
- Instill 50-60 mL of ice-cold water into each ear over 1 minute using a syringe
- Test each side individually with several minutes between testing each side
- Normal: slow conjugate deviation towards the irrigated side and fast horizontal nystagmus to the contralateral ear
- Abnormal: no fast nystagmus in patients with cerebral damage but intact brainstem reflexes. No slow deviation and no fast nystagmus imply brainstem damage

Gag reflex:

- *Afferent: CN IX, Efferent: CN X*
- Tested by stimulating the back of the patient's throat with a tongue depressor or suction catheter
- Gag reflex is of limited utility since many patients with normal brainstem lack a gag reflex. If a gag reflex is present, and on subsequent testing it is lost, that might be of clinical value

Cough reflex:

- *Afferent: CN X, Efferent: CN X*
- In an intubated patient can be tested by touching the carina with a suction catheter passed through the patient's ETT or tracheostomy tube

Motor

A normal patient should follow commands. In a comatose patient it is often necessary to administer noxious stimuli, which may include sternal rub or supraorbital ridge pressure. Do not perform supraorbital ridge pressure in the presence of facial fractures. If there is no response to this noxious stimulus, peripheral stimulus (such as application of nailbed pressure) should be performed. There is a range of movements which may be seen.

- Patients may localize to the stimulus, withdraw away from the stimulus, flex, extend, or have no response at all. Grimacing may also be observed
- Spinal reflexes may lead to lower extremity movements even in patients with severe brain damage or brain death (e.g. ***triple flexion*** response of hip, knee, and ankle flexion)
- Decorticate posturing: upper extremity flexion and lower extremity extension, typically from a lesion above the red nucleus of the midbrain
- Decerebrate posturing: upper and lower extremity extension is typically from a lesion below the red nucleus
- Unilateral or bilateral posturing may be seen based on location of lesion causing it
- Post-anoxic myoclonus is common in patients following cardiac arrest. Occasionally it may indicate ongoing seizure activity, and EEG is recommended

Tone and reflexes

Increased tone, brisk reflexes, and upgoing toes are indicative of a lesion in the spinal cord or brain.

- If unilateral, usually indicates a lesion on the opposite side
- Symmetric hyperreflexia can be normal, especially in young patients, but may also indicate bilateral lesions, especially in the brainstem and spinal cord. In rare instances, symmetric hyperreflexia might indicate conditions like serotonin syndrome
- Neuro-intact people with brisk reflexes usually do not have upgoing toes, so this can be a good way to differentiate pathological cases from physiologic hyperreflexia
- Brisk reflexes and ↑ tone in lower extremities but not upper extremities are indicative of lesion below the level of the cervical spinal cord
- Very early on, brain and spinal cord lesions might present with flaccid paralysis

RESPIRATORY PATTERNS IN COMATOSE PATIENTS

Medication side effects should be ruled out first. Sedating medications tend to cause slow regular breathing, whereas salicylate overdose can cause rapid breathing. In intubated patients, assess synchrony with the ventilator and degree of effort, including actual vs set respiratory rate. Abnormal breathing may manifest more prominently on spontaneous ventilator modes.

Types of abnormal breathing include:

- Cheyne-Stokes: oscillation between fast and slow breathing (multiple causes including bilateral hemispheric lesions, heart failure, etc.)

- Apneustic: rapid breathing with inspiratory pauses (pontine lesions)
- Biot's: quick shallow breaths followed by pause after four to five cycles (medullary damage). Also known as ataxic breathing
- Cluster: regular cycles of deep breaths with variable periodicity
- Kussmaul: rapid, deep and labored breaths (metabolic acidosis)

USEFUL ANCILLARY TESTS IN COMATOSE PATIENTS

- Laboratory tests: serum electrolytes, glucose, hormone levels (such as TSH), ammonia, and toxicology tests should be considered to evaluate for potentially reversible causes of coma (see Table 2)
- CT: primary value is to rule out ICH or large mass, and to assess for edema, hydrocephalus, and herniation
- MRI: requires significantly more time than CT and can be contraindicated or difficult to obtain in unstable patients, but is helpful in diagnosis of demyelinating lesions, meningoencephalitides, small strokes (especially evaluation of posterior fossa), and some metabolic disorders
- EEG: should be performed in all patients who are unresponsive without a clear etiology to evaluate for nonconvulsive or electrographic seizures. Most common finding in coma is "generalized slowing." Focal slowing can indicate a structural lesion. Triphasic waves are often seen in metabolic encephalopathies (classically in liver failure). Cefepime is another common cause of triphasic-like waves on EEG and alteration of consciousness in ICU patients, especially those with impaired renal function
- LP: measurement of opening pressure and diagnostic evaluation of CSF for infectious or inflammatory etiologies may assist in narrowing the differential diagnosis