# 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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**Marin E. Darsie, MD**
Assistant Professor of Emergency Medicine
Division of Neurocritical Care
University of Wisconsin School of Medicine and Public Health
Madison, WI, USA

**Asma M. Moheet, MD**
Assistant Professor of Neurology and Neurosurgery
Associate Director, Neuro Critical Care Unit
Cedars-Sinai Medical Center
Los Angeles, CA, USA

***For our families, our patients, and their families.***
***Thank you for teaching us.***





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# PREFACE

While staffing the Neurocritical Care Society booth at the 2016 SCCM Conference, members of the NCS Resident/Fellow Committee realized there was demand for a quick reference guide to neurocritical care when person after person inquired if we had any for sale. Over the next 15 months, the Resident/Fellow Committee developed a vision which has become The Pocket Guide to Neurocritical Care: A concise reference for the evaluation and management of neurologic emergencies.

Over 40 authors have contributed to this project. Residents, fellows, and APPs were recruited as junior authors to provide authorship opportunities for trainees at the beginning of their careers. They were paired with established leaders in the field of neurocritical care who served as the senior authors for each chapter.

The aim of this book was to create a resource for trainees and other members of the NCCU multidisciplinary team of varying backgrounds on bedside management and pearls for a variety of neurocritical care conditions. With guidance from the NCS Educational Products Committee, seventeen high-yield topics were identified which include the most commonly encountered neurologic emergencies and basics of critical care medicine explained through the neurocritical care perspective. This book is not intended to be a definitive reference text, rather it aims to arm the reader with a grasp of a neurocritical care topic in less than 15 minutes.

# ACKNOWLEDGEMENTS

This project would not have been possible without the time, efforts and vision of all the members of the NCS Resident/Fellow Committee, specifically Sherri Braksick, Tobias Kulik, Deepa Malaiyandi, and Anand Venkatraman for their assistance with editing.

We would like to recognize the members of the NCS Educational Products Committee for their oversight and support in developing this project, as well as their time spent editing and reviewing its content for accuracy.

We would like to thank Becca Stickney and Sara Memmen for all their help in launching the first edition of The Pocket Guide to Neurocritical Care.

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

Marin Darsie and Asma Moheet
August 2017

## 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 |
| **AED** | anti-epileptic drug |
| **AF** | atrial fibrillation |
| **AG** | anion gap |
| **AHA** | American Heart Association |
| **AICA** | anterior inferior cerebellar artery |
| **AIS** | acute ischemic stroke |
| **ALS** | amyotrophic lateral sclerosis |
| **AKA** | also known as |
| **ARDS** | acute respiratory distress syndrome |
| **aSAH** | aneurysmal subarachnoid hemorrhage |
| **ATLS** | Advanced Trauma Life Support |
| **$AVDO_2$** | arterio-venous difference of oxygen consumption |
| **AVM** | arteriovenous malformation |
| **BCx** | blood culture |
| **BBB** | blood-brain barrier |
| **BID** | twice daily |
| **BMP** | basic metabolic panel |
| **BP** | blood pressure |
| **BTF** | Brain Trauma Foundation |
| **BSAS** | Bedside Shivering Assessment Scale |
| **C** | Celsius |
| **$Ca^{2+}$** | calcium |
| **CABG** | coronary artery bypass graft |
| **CAS** | carotid artery stenting |
| **CBC** | complete blood count |
| **CBF** | cerebral blood flow |
| **CCM** | cerebral cavernous malformation |
| **CEA** | carotid endarterectomy |
| **CHF** | congestive heart failure |
| **CIDP** | chronic inflammatory demyelinating polyneuropathy |
| **COPD** | chronic obstructive pulmonary disease |
| **CIM** | critical illness myopathy |
| **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 |
| **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 |
| **CVAD** | central venous access device |
| **CVR** | cerebral vascular resistance |
| **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 |
| **DH** | decompressive hemicraniectomy |
| **DI** | diabetes insipidus |
| **DKA** | diabetic ketoacidosis |
| **DNI** | do not intubate |
| **DNR** | do not resuscitate |
| **DSA** | digital subtraction angiography |
| **DTR** | deep tendon reflexes |
| **DVT** | deep vein thrombosis |
| **EBV** | Epstein-Barr virus |
| **ECMO** | extracorporeal membrane oxygenation |
| **ED** | Emergency Department |
| **EDH** | epidural hematoma |
| **EEG** | electroencephalogram |
| **EKG** | electrocardiogram |
| **EMG** | electromyography |
| **EN** | enteral nutrition |
| **ENLS** | Emergency Neurologic Life Support |
| **ENT** | ear/nose/throat or otolaryngology |
| **EOM** | extraocular muscles |
| **ETT** | endotracheal tube |
| **EVD** | external ventricular drain |
| **FFP** | fresh frozen plasma |
| **FOUR** | Full Outline of Unresponsiveness |
| **FVC** | forced vital capacity |
| **GBS** | Guillain-Barré syndrome |
| **GCS** | Glasgow Coma Scale |
| **GI** | gastrointestinal |
| **GOSE** | Glasgow Outcome Scale-Extended |
| **GTC** | generalized tonic-clonic |
| **H** | hour |
| **HCG** | human chorionic gonadotropin |
| **HD** | hemodialysis |
| **Hgb** | hemoglobin |
| **HIT** | heparin-induced thrombocytopenia |
| **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 |
| **IgA** | immunoglobulin A |
| **IM** | intramuscular |
| **IV** | intravenous |
| **IVC** | inferior vena cava |
| **IVF** | intravenous fluids |
| **IVH** | intraventricular hemorrhage |
| **IVIg** | intravenous immunoglobulin |
| **IV tPA** | intravenous tissue plasminogen activator |
| **KCL** | potassium chloride |

| | |
|---|---|
| **LE** | lower extremity |
| **LFTs** | liver function tests |
| **LKWT** | last known well time |
| **LOS** | length of stay |
| **LP** | lumbar puncture |
| **MAP** | mean arterial pressure |
| **MC** | myasthenic crisis |
| **MCA** | middle cerebral artery |
| **MCS** | minimally conscious state |
| **MEP** | maximal expiratory pressure |
| **MG** | myasthenia gravis |
| **MH** | malignant hyperthermia |
| **MHS** | malignant hemispheric stroke |
| **MI** | myocardial infarction |
| **Min** | minute |
| **mL** | milliliter |
| **MOA** | mechanism of action |
| **MRI** | magnetic resonance imaging |
| **mRS** | modified Rankin Scale |
| **MV** | mechanical ventilation |
| **NCCU** | Neuro Critical Care Unit |
| **NCS** | nerve conduction study OR Neurocritical Care Society |
| **NCSE** | non-convulsive status epilepticus |
| **NG** | nasogastric |
| **NIF** | negative inspiratory force |
| **NIPPV** | noninvasive positive pressure ventilation |
| **NM** | neuromuscular |
| **NMBA** | neuromuscular blockade agent |
| **NMJ** | neuromuscular junction |
| **NOAC** | novel oral anticoagulant |
| **NS** | normal saline |
| **NSAID** | nonsteroidal anti-inflammatory drugs |
| **$O_2$** | oxygen |
| **OG** | orogastric |
| **OOB** | out of bed |
| **OSA** | obstructive sleep apnea |
| **OSM** | osmolar |
| **OT** | occupational therapy or therapist |
| **$P_{bt}O_2$** | brain tissue oxygen tension |
| **PCA** | posterior cerebral artery |
| **PComm** | posterior communicating artery |
| **PE** | pulmonary embolus |
| **PEEP** | positive end expiratory pressure |
| **PEG** | percutaneous endoscopic gastrostomy |
| **PFO** | patent foramen ovale |
| **PICA** | posterior inferior cerebellar artery |
| **PIV** | peripheral intravenous line |
| **PLEX** | plasmapheresis |
| **Pplat** | plateau pressure |
| **PRN** | pro re nata, as needed |
| **PRVC** | pressure regulated volume control |
| **PT** | physical therapy or therapist |
| **PVS** | persistent vegetative state |
| **QID** | four times daily |
| **QOD** | every other day |
| **RA** | rheumatoid arthritis |
| **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 |
| **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 |
| **SDH** | subdural hematoma |
| **Se** | sensitivity |
| **SE** | status epilepticus |
| **Sec** | second |
| **SIADH** | syndrome of inappropriate antidiuretic hormone secretion |
| **SLE** | systemic lupus erythematosus |
| **s/p** | status post |
| **Sp** | specificity |
| **$SpO_2$** | peripheral capillary oxygen saturation |
| **SSEPs** | somatosensory evoked potentials |
| **SSRI** | selective serotonin reuptake inhibitor |
| **SSS** | sick sinus syndrome |
| **ST** | speech therapy or therapist |
| **TB** | tuberculosis |
| **TBI** | traumatic brain injury |
| **TCD** | transcranial doppler ultrasound |
| **TH** | therapeutic hypothermia |
| **TIA** | transient ischemic attach |
| **TMJ** | temporomandibular joint |
| **TMP-SMX** | trimethoprim-sulfamethoxazole |
| **tPA** | tissue plasminogen activator |
| **TTM** | targeted temperature management |
| **U** | units |
| **UA** | urinalysis |
| **UCx** | urine culture |
| **UE** | upper extremity |
| **UH** | unfractionated heparin |
| **UMN** | upper motor neuron |
| **UTI** | urinary tract infection |
| **VF** | ventricular fibrillation |
| **$V_T$** | 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 will 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 reticular activating system 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 minimally conscious state (MCS) and vegetative states. 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 vegetative state, minimally conscious state (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 persistent vegetative state (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, such as medication or drug toxicities, generalized status epilepticus, metabolic disorders and meningoencephalitides can all cause poor responsiveness or coma, with or without focal neurologic findings. Coma may also be caused by brain lesions affecting the thalamus and brainstem, since these contain crucial arousal-supporting neurons. The latter 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 higher.

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

Limitations of GCS:

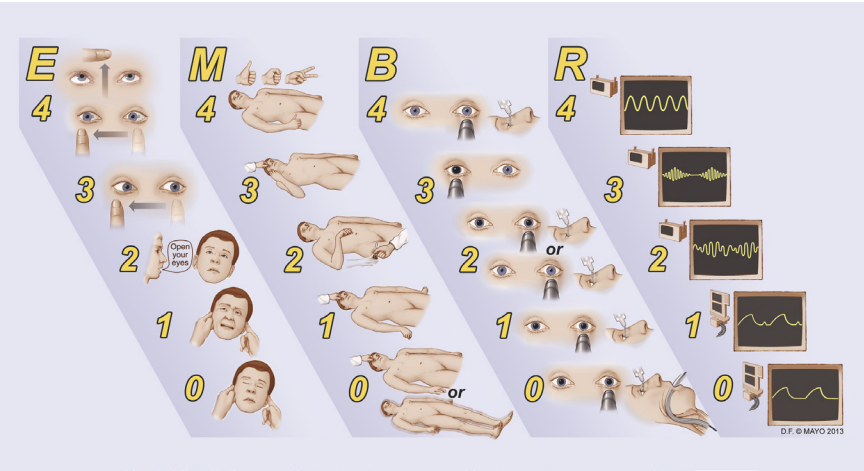
- Can miss locked-in states and subtle changes in consciousness
- Does not assess pupillary and other brainstem reflexes
- Patients with similar scores may go on to have different outcomes
- Assigns greater weight to motor response than eye opening and verbal responses.
- Intubated patients default to a T score on verbal

The Full Outline of UnResponsiveness (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 additionally described in Table 4.

**Figure 1.** Calculation of the FOUR Score




## 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
- 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 hemorrhage, organophosphate poisoning, clonidine overdose, pilocarpine eye drop use, and occasionally mirtazapine and olanzapine
- Sluggish pupils: neuromuscular blocking agents, 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 swab can also be used but use caution as with repeated testing this 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
  - Terson's syndrome: subhyaloid hemorrhage in SAH

Oculocephalic or "doll's eyes" reflex (OCR):
- *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

Cold calorics (oculovestibular response or OVR):
- *Afferent: CN VIII, Efferent: CN III and VI*
- Do this if OCRs are absent
- 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 of each
- 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 implies cerebral and 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

Cough reflex:
- *Afferent: CN X, Efferent: CN X*
- In an intubated patient can be tested by inserting a suction catheter into the patient's ETT or tracheostomy tube

<u>Motor</u>

A normal patient should follow commands. In a comatose patient it is often necessary to administer noxious stimuli centrally, 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 central noxious stimulus, peripheral stimulus (such as application of nailbed pressure) should be performed.

- 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.
- Postanoxic myoclonus is common in patients following cardiac arrest. Occasionally it may indicate ongoing seizure activity, EEG is recommended.

Tone and Reflexes

↑ 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.
- 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.

## 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:

- 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)
- Kussmaul: rapid, deep and labored breaths (metabolic acidosis)