

**MINISTRY OF HEALTHCARE OF UKRAINE  
HSEEU "Ukrainian Medical Stomatological Academy"**

**"Approved"**  
at the meeting of internal  
medicine №1 department  
Head of Department  
**Prof. Skrypnyk I.M.**

---

**Protocol № 1 from 29.08.2016**

**GUIDELINES  
FOR STUDENTS  
INDEPENDENT WORK  
IN THE PRACTICAL CLASSES PREPARING**

<i>Academic discipline</i>	Internal medicine
<i>Module</i>	Emergency conditions in clinic of Internal Medicine
<i>Content module</i>	Emergency conditions in clinic of Internal Medicine
<i>Study subject</i>	<b>Curation of the patient with coma</b>
<i>Course</i>	VI
<i>Faculty</i>	of foreign students training

Poltava 2016.

## I. CURRENT APPLICATIONS

Consciousness is defined as a state of being aware of both self and environment. It requires the continuous interaction of two intact components: awareness (content) mediated by the cerebral cortex and arousal (wakefulness) primarily supported by the brainstem structures (ascending reticular activating system [ARAS] and diencephalon). While the cortex is responsible for cognitive functions, the brainstem essentially activates and maintains cerebral responsiveness to the environment. The cortex has no intrinsic mechanism to generate or support arousal. Unconsciousness may be induced by failure of either or both the brainstem and cortex. Most commonly, acute loss of consciousness is caused by impaired arousal due to an insult to the brainstem. As a result of damage to the ARAS, the cortex persists in a state of inactivity, the patient presents unawakeful and it is impossible to assess his awareness. Whereas loss of arousal may develop after a small focal affection of brainstem structures, both diffuse and bilateral injury of cerebral hemispheres is required to produce complete unconsciousness, sparing the ARAS. Being able to produce significant neurological deficit, unilateral injury of the cerebral cortex does not result in a coma if the brainstem structures are intact.

**II. GOALS AND OBJECTIVES.** To expand student's knowledge of one of the most common emergency conditions, which there are in adults. To systematize the knowledge of early diagnosis and timely emergency assistance in comas.

### Know:

- features organization of prehospital emergency care
- a set of medications for emergency assistance at home

### Able to:

- to syndrome diagnosis of threatened conditions
- to administer adequate treatment in developing of main syndromes, which threatened the patient's life
- correctly deal with question of patient transport and emergency hospitalization in an intensive departments and profile centers

## III. INTERDISCIPLINARY INTEGRATION:

No	Name of section	Department
1.	Diagnosis and treatment of coma .	Department of propaedeutics of internal diseases Department of internal diseases Department of surgery with course anesthesiology and resuscitation
2.	Medications, which is using for treatment of urgent status	Department of Pharmacology

Comas can be caused by several medical complications and conditions, including:

- Traumatic brain injury
- Diabetes (hypoglycemic coma)
- Lack of oxygen
- Hemorrhage
- Swelling
- Infection
- Stroke
- Toxic exposure

One of the most common causes of coma is traumatic brain injury, which often occurs during assaults and transportation accidents. A traumatic brain injury may be classified as either an open head injury or a closed head injury and can cause focal brain damage, which affects isolated sections of the brain, or diffuse brain damage, which affects a broader area. Focal and

diffuse brain damage can influence consciousness, as well as cause intracranial pressure or swelling.

In addition to those suffering from traumatic brain injury, diabetic patients are at risk for coma from extreme levels of blood sugar. Conditions that block blood flow and lead to a lack of oxygen, such as hemorrhaging or stroke, can also cause patients to fall into a comatose state.

Even healthy individuals can fall into a coma if they are exposed to certain environmental factors. This includes infections, such as encephalitis and meningitis, and toxic poisoning, which can be caused by exposure to carbon monoxide and other toxins.

#### Coma Symptoms

One of the most obvious symptoms of a coma is a lack of consciousness. The onset of a coma can be gradual or immediate, depending on the cause. Patients who suffer from mild traumatic brain injuries may experience headaches, drowsiness, confusion, body aches, fever and dizziness before falling into a coma.

Patients in a coma lack awareness and wakefulness, which means they do not respond to external stimuli or pain. Although they may demonstrate reflexive movements, patients who do not exhibit purposeful movements are deemed unconscious. Comatose patients also keep their eyes closed, have impaired breathing and fail to exhibit a normal sleep-wake pattern.

#### Coma Diagnosis

##### Physical & Neurological Evaluation

Doctors use physical examinations as well as medical technologies to evaluate head injuries and diagnose comas. Once they have treated open wounds and established proper breathing and blood flow to the brain, they evaluate a patient's medical history to check for medications, conditions such as diabetes, and medical events such as strokes. Then, they check a patient's reflexes and pupil size to determine the level of consciousness. Doctors also take a blood sample to test blood count, electrolytes, and glucose levels, and look for any remnants of drugs or toxins such as carbon monoxide.

Once the physical evaluation is complete, several neurological examinations are performed. A patient's level of consciousness is assessed using the Glasgow Coma Scale or the Rancho Los Amigos Scale. These scales also help doctors evaluate a patient's chance of recovery.

#### *Glasgow Coma Scale*

**The Glasgow Coma Scale (GCS)** is the standard scoring system for assessment of the level of consciousness based on the best motor, verbal and eye-opening responses to external stimuli (Table 32.7).

**Originally introduced in 1974 for the** assessment of patients with traumatic brain injury (TBI), the GCS has gained worldwide acceptance as an easily performed and reproducible tool for the assessment of all acutely ill patients. As well as providing a quantitative documentation of the level of consciousness, it has powerful predictive value for survival and outcome in both traumatic and non-traumatic coma. However, the GCS does have limitations. The scale excludes assessment of many important neurological functions, requires serial observations to be effective and is limited to the best response in one limb. It cannot therefore identify asymmetry and has poor diagnostic value. In addition, combining the three values into a single total score can lead to disparities in assessment of true conscious level. Finally but importantly, a complete GCS cannot be obtained in patients who have eyelid swelling, are sedated and intubated with a tracheal tube or who are aphasic due to a dominant hemisphere lesion.

#### **The Glasgow Coma Score scale**

<b>Best eye-opening response</b>	<b>4</b>	<b>Spontaneous</b>
	<b>3</b>	<b>To speech</b>
	<b>2</b>	<b>To pain</b>

	1	None
Best motor response	6	Obeys command
	5	Localizes to pain
	4	Withdrawal
	3	Flexion posturing
	2	Extensor posturing
	1	None
Best verbal response	5	Orientated
	4	Confused speech
	3	Inappropriate words
	2	Incomprehensible sounds
	1	None

### ***The FOUR score***

**The recently introduced** Full Outline of UnResponsiveness (FOUR) score circumvents many of the limitations of the GCS (Table 32.8). The verbal score of the GCS is replaced by assessment of pupil reactions and respiratory pattern, making it more appropriate in the intubated patient.

### ***Brainstem reflexes and activity***

#### **Pupillary responses**

**The normal pupillary response to light** is a direct and consensual constriction and depends on functioning afferent (cranial nerve II) and efferent (cranial nerve III) pathways and the midbrain Edinger-Westphal nucleus. The presence of equal, light-reactive pupils indicates that the reflex pathway is intact. A normal pupillary reaction in a comatose patient suggests a toxic or metabolic aetiology rather than a structural one. Unilateral or bilateral miosis with normal reaction to light may be due to Horner's syndrome associated with lesions involving descending sympathetic pathways in the hypothalamus, midbrain, medulla or cervical spine (e.g. cervical carotid artery damage). Bilateral pinpoint pupils are seen with pontine lesions in the tegmentum, opioid overdose and anti-cholinesterase poisoning (e.g. in myasthenic cholinergic crisis). Unilateral pupillary dilation with either a sluggish or no response to light is caused by compression of the oculomotor nerve and is seen in uncal herniation, posterior communicating artery aneurysm rupture and with lesions affecting the cavernous sinus (e.g. pituitary apoplexy). Bilateral pupillary dilation with no response to light is seen in central herniation, extensive midbrain damage, hypothermia, drug intoxication (e.g. with anticholinergic agents) and brainstem death.

#### **The Full Outline of UnResponsiveness (FOUR) score**

<b>Eye response</b>	<b>4</b>	<b>Eyelids open, tracking or blinking to command</b>
	3	Eyelids open but not tracking
	2	Eyelids closed but open to a loud voice
	1	Eyelids closed but open to pain
	0	Eyelids remain closed with pain
<b>Motor response</b>	4	Thumbs-up, fist or peace sign
	3	Localizing to pain
	2	Flexion response to pain
	1	Extension response to pain
	0	No response to pain or generalized myoclonic status

Brainstem reflexes	4	Pupil and corneal reflexes present
	3	One pupil wide and fixed
	2	Pupil or corneal reflexes absent
	1	Pupil and corneal reflexes absent
	0	Absent pupil, corneal and cough reflex (using tracheal suction)
Respiration	4	Not intubated, regular breathing pattern
	3	Not intubated, Cheyne-Stokes breathing pattern
	2	Not intubated, irregular breathing pattern
	1	Breaths above ventilator rate
	0	Breaths at ventilator rate or apnoea

### ***Eye position and oculomotor disorders***

**Examination of the position of the eyes can** yield important lateralizing information. In the primary ocular position, the eyes may be conjugate in the midline, dysconjugate or deviated in a conjugated manner. Dysconjugate deviation is commonly seen in patients with impaired consciousness (and those on sedation) and has little localizing value.

### **Involuntary vertical eye movements in coma**

Eye movement	Clinical finding	Lesion identified
Ocular bobbing	Rapid downward jerks of both eyes followed by a slow return to the mid-position	Acute pontine lesion Metabolic and toxic
	Paralysis of both reflex and spontaneous horizontal eye movements	Extra-axial posterior fossa masses
Ocular dipping	Slow initial downward phase is followed by a relatively rapid return to the mid-position	Diffuse cerebral anoxia
		Following status epilepticus

**A complete IIIrd nerve palsy** is characterized by ptosis, pupillary dilation and deviation of the eye downward and laterally. It is most commonly seen as a manifestation of transtentorial herniation but may also be a feature of midbrain damage. A dysconjugate vertical gaze may be caused by IVth nerve palsy, which commonly occurs following trauma, drug intoxication or metabolic encephalopathy. VIth nerve palsy (inward deviation and failure of abduction of the eye) is often caused by trauma or raised intracranial pressure but has poor localizing value.

Tonic conjugate eye deviation is often seen in coma. In destructive hemispheric lesions, the eyes deviate towards the lesion and away from the hemiparesis and can usually be driven across the midline by vestibular stimulus. In contrast, in lesions below the pontomes-encephalic junction, the eyes deviate away from the side of the lesion and do not cross the midline.

### **Eye movements**

Spontaneous eye movements in coma tend to be either roving or involuntary, conjugate vertical movements. Roving eye movements are slow, purposeless lateral movements, either conjugate or dysconjugate. They imply intact oculomotor pathways and are usually associated with toxic or metabolic coma. A number of conjugate vertical eye movements are recognized and are classified according to the velocities of the upward and downward saccades. The most common are ocular bobbing and dipping, and they can identify anatomical and aetiological causes of coma.

## Oculovestibular reflexes

**Oculovestibular reflexes are involuntary eye movements** that occur after stimulation of the vestibular apparatus. the oculocephalic reflex is tested by sudden lateral rotation of the head while observing the motion of the eyes. Ocular responses to vertical stimulation are tested by flexing and then extending the neck. this reflex should not be tested if cervical spine instability is suspected. the oculovestibular reflex is elicited by irrigation of the tympanic membrane with cold (30°C) or warm (44°C) water. Normal and abnormal responses to oculocephalic and oculovestibular stimulation.

## Other cranial nerve reflexes

**The corneal reflex (blinking on lightly brushing the cornea with cotton wool)** tests the integrity of the afferent (trigeminal nerve), efferent (facial nerve) and brainstem (trigeminal and facial nerve pontine nuclei) pathways. However, the reflex may be lost with deep sedation. the gag and cough reflexes test the integrity of the glossopharyngeal and vagus nerves and associated medullary centres.

## Respiratory patterns

**Although distinct abnormal respiratory patterns** based on animal experiments suggest that individual patterns have localizing value, in practice early intervention with mechanical ventilation in comatose patients means that these patterns are not commonly seen in clinical practice. Primary central neurogenic hyperventilation is a rare condition characterized by rapid regular breathing that persists in the face of alkalosis, elevated arterial oxygen tension, low arterial carbon dioxide tension and in the absence of pulmonary or airway pathology. It is seen in damage to the midbrain tegmentum. Apneustic breathing consists of prolonged pauses at end inspiration and follows bilateral tegmental infarction and pontine demyelination. Cheyne-Stokes respiration consists of cyclical waxing and waning of tidal volume and respiratory rate separated by apnoeic episodes. It is seen in coma of varying aetiology and has little localizing value. Hiccups may occur as a result of structural or functional disorders of the medulla or its afferent or efferent connections with the respiratory muscles, and in this context may herald respiratory arrest.

## Oculocephalic responses

Oculocephalic reflex	Response	Cause
Horizontal rotation	Eyes remain conjugate and maintain fixation (move in opposite direction to head) No movement in either eye	Normal with reduced level of consciousness
		Low brainstem lesion
		Peripheral vestibular lesion
		Drugs
	Eyes move appropriately in one direction but do not cross the midline in the other	Anaesthesia
		Gaze palsy (unilateral lesion in pontine gaze centre)
		Pontine lesion
	One eye abducts but the other fails to adduct	IIIrd nerve palsy
		Internuclear ophthalmoplegia (lesion of the median longitudinal fasciculus)
	One eye adducts but the other fails to abduct	Vth nerve palsy
Vertical	Eyes remain conjugate and maintain	Normal with reduced level of

	fixation (move in direction opposite to head movement)	consciousness
	No movement in either eye	Low brainstem lesion
		Peripheral vestibular lesion
		Drugs
		Anaesthesia
	Only one eye moves	IIIrd nerve palsy
	Loss of upward gaze	Pretectal or midbrain tegmental compression

### Oculovestibular responses

Oculovestibular reflex	Response	Cause
Cold water instilled into the right ear	Nystagmus with slow phase to right and fast phase to left	Normal
	No response	Obstructed ear canal
		Labyrinthine damage
		Low brainstem lesion
	Tonic deviation towards stimulated side (slow phase to right, no fast phase)	Supratentorial lesion with intact pons
		Toxic/metabolic
		Drugs
		Structural lesion above brainstem
	Dysconjugate response	Brainstem lesion (usually in region of medial longitudinal fasciculus)
	Downbeat nystagmus	Horizontal gaze palsy
	Vertical eye deviation	Drug overdose
Warm water instilled into left ear after no response to cold	Slow phase to right, fast phase to left	Peripheral VIIIth nerve lesion
		Labyrinthine disorder on right

Ataxic breathing, characterized by irregular respiratory rate and tidal volume, is associated with severe medullary damage.

#### X-Rays and Imaging

Medical technologies allow doctors to identify the location and severity of the injury sustained. CT scans, MRI scans and EEG tests check for hemorrhaging, swelling, brain stem damage and non-convulsive seizures, an underlying cause of comas. In addition, they allow doctors to identify the level of consciousness and create appropriate treatment plans.

#### Coma Complications

Patients in a coma are at risk for a variety of complications. One of the most serious is progression into a persistent vegetative state. A coma lasting longer than two to four weeks is reclassified as a persistent vegetative state. Patients who fall into a persistent vegetative state typically have much lower chances of recovery.

Doctors always monitor comatose patients for any secondary brain injuries caused by brain swelling or intracranial pressure.

Due to inactivity, patients in a coma may experience bed sores, or skeletal or muscular atrophy. These complications can be overcome with appropriate rehabilitation plans.

### Coma Treatment

In order to successfully treat comatose patients, doctors must prevent secondary brain injuries from causing further damage, such as swelling and intracranial pressure.

#### Reducing Intracranial Pressure

To reduce intracranial pressure, doctors can prescribe medication or recommend surgery. A craniotomy may be performed to relieve pressure; this procedure creates holes in the skull to allow for excess blood and fluid to drain. Alternatively, the surgeon may insert a catheter to relieve pressure or remove a portion of the skull to create room for brain swelling.

#### Other Coma Treatments

Upon admission to a hospital, patients often receive antibiotics or glucose to treat suspected infections or diabetic comas. Doctors may also administer strong medications to control patients who suffer from non-convulsive seizures.

Because there is no cure for a coma, treatment options are limited to preventing further damage, treating underlying causes and maintaining proper blood flow and nutrition.

#### Coma Prognosis

A patient's chance of recovery is dependent on the severity of the personal injury sustained and the amount of time spent in a comatose state. Patients with mild head injuries often regain consciousness within a few weeks. Those with severe brain damage may slip into persistent vegetative states.

Patients who regain consciousness usually regain their cognitive and motor skills after rehabilitation. Through these programs, physical therapists and doctors help patients in their effort to recover the skills necessary to live independently.

To learn more about traumatic brain injury, or other states of consciousness associated with head injury, please refer to other articles on this site.

An unconscious patient presents with moist skin, shallow breathing. There are signs of previous injection on the shoulders and hips. BP- 110/70 mm Hg. Tonus of skeletal muscles and reflexes are increased. Cramps of muscles of the extremities are seen. What is the most likely disorder?

- hypoglycemic coma
- hyperglycemic coma
- hyperosmolar coma
- hyperlactacidotic coma
- stroke

A 23 year old man had acute respiratory viral infection. After it there appeared polydipsia, polyuria, weakness, nausea. Examination revealed the following symptoms: mental confusion, dry skin, soft eyeballs, Kussmaul's respiration, acetone smell from the mouth, muffled heart sounds, soft and painless abdomen. Blood sugar was 19millimole/l. What acute condition is it?

- ketoacidotic coma
- hyperosmolar coma
- cerebral coma
- hepatic coma
- acute renal insufficiency

A 28-year-old patient underwent endometrectomy as a result of incomplete abortion. Blood loss was at the rate of 900 ml. It was necessary to start hemotransfusion. After transfusion of 60 ml of erythrocytic mass the patient presented with lumbar pain and fever which resulted in hemotransfusion stoppage. 20 minutes later the patient's condition got worse: she developed adynamia, apparent skin pallor, acrocyanosis, profuse perspiration. *to*- 38, 5oC, Ps- 110/min, AP- 70/40 mm Hg. What is the most likely diagnosis?

- hemotransfusion shock
- hemorrhagic shock



septic shock  
anaphylactic shock  
DIC syndrome

A 58-year-old female patient complains about periodical headache, dizziness and ear noise. She has been suffering from diabetes mellitus for 15 years. Objectively: heart sounds are rhythmic, heart rate is 76/min, there is diastolic shock above aorta, AP is 180/110 mm Hg. In urine: OD- 1,014. Daily loss of protein with urine is 1,5 g. What drug should be chosen for treatment of arterial hypertension?  
inhibitor of angiotensin converting enzyme  
 $\beta$ -blocker  
calcium channel antagonist  
thiazide diuretic  
 $\alpha$ -blocker

A patient aged 18 with a cranial injury was in comatose state during several hours. In post-comatose period gets tired quickly, non-productive in dialog – in the beginning answers 2-3 questions, then gets tired and can not understand the point of the question. Which psychotropic should be given to the patient to prevent psychoorganic syndrome?  
nootropics  
neuroleptics  
stimulators  
tranquillisers  
antidepressants

A patient suddenly felt an acute chest pain irradiating to the left arm. Objectively: the patient is excited, with pale skin. Breathing rate - 38/min, AP - 180/110 mm Hg. Later the patient lost consciousness and fell down. Pulse on the great vessels was absent, the pupils were equally dilated. What is the most likely diagnosis?  
clinical death  
agonal state  
coma  
heart attack  
disorder of the cerebral circulation

An 18 year-old teenager with a 3-year history of diabetes was hospitalized in hyperglycemic coma. Specify the initial dose of insulin to be administered:  
0,1-0,2 U/kg of body weight per hour  
0,05 U/kg of body weight per hour  
0,2-0,3 U/kg of body weight per hour  
0,3-0,4 U/kg of body weight per hour  
0,4-0,5 U/kg of body weight per hour

You are a doctor on duty. A patient after a successful resuscitation (drowning) was delivered to an admission room. BP is 90/60 mm Hg, heart rate is 120/min., respiration rate is 26/min. The patient is unconscious, pupils are moderately dilated, general clonic and tonic convulsions are observed. Make the diagnosis:  
postresuscitation disease  
apparent death  
coma of unknown origin  
unconsciousness  
vegetative state

A patient is in a coma. The smell of ammonia breath is observed. A dry and pale skin is observed. History of disease: patient suffer by chronic glomerulonephritis during 10 years. Auscultation: pericardial friction noise. What type of coma do you find patient?

- uremic coma
- diabetic coma
- hypoglycemic coma
- alcohol coma
- hepatic coma

Patient, 43 y.o., was admitted to hospital in serious condition. Clinical and laboratory data are: coma, fruity breath odor, hypotension, dehydration, electrolyte disturbances, ketoacidosis. What is your diagnosis?

- diabetic ketoacidosis
- hyperosmolar hyperglycemic syndrome
- diabetes insipidus
- the syndrome of inappropriate secretion of antidiuretic hormone
- acute adrenal insufficiency

Patient A., 65 y.o., was hospitalized in the intensive care unit in a coma. She has been suffering from type II diabetes mellitus during 10 years. Severe polyuria, polydipsia were observed last 2 weeks. Glucose serum: 30 mmol / L, arterial pH - 7.3. Plasma osmolality - 350 mOsm / l. The patient newly diagnosed diabetic hyperosmolar coma. What is the main pathogenetic mechanism?

- dehydration
- hypocoagulation
- hyponatremia
- increased tubular reabsorption
- increased glomerular filtration

### **Recommended literature:**

#### **A. Main:**

1. "Harrison's principles of internal medicine", Editors: Anthony S. Fauci, Dennis L. Kasper, Stephen L Hauser, Dan L. Longo, Joseph Loscalzo, McGraw-Hill Education / Medical; 19 edition (April 8, 2015), 1-2 volumes, 3000 p.
2. CURRENT Medical Diagnosis and Treatment 2012, Fifty-First Edition (LANGE CURRENT Series) by Stephen McPhee, Maxine Papadakis and Michael W. Rabow (Paperback - Sep 12, 2011)
3. Davidson's Principles and Practice of Medicine: With STUDENT CONSULT Online Access, 21e (Principles & Practice of Medicine (Davidson's)) by Nicki R. Colledge BSc FRCP(Ed), Brian R. Walker BSc MD FRCP(Ed) and Stuart H. Ralston MB ChB MD FRCP FMedSci FRSE (Paperback - Mar 11, 2010) Kumar and Clark's Clinical Medicine, 7e (Kumar, Kumar and Clark's Clinical Medicine) by Parveen J. Kumar (Paperback - Jul 2, 2009)
4. 1000 Questions and Answers from Kumar & Clark's Clinical Medicine, 2e [Paperback] Parveen Kumar CBE BSc MD FRCP FRCP(Edin) (Editor), Michael L Clark MD FRCP (Editor)
5. Differential Diagnosis in Internal Medicine: From Symptom to Diagnosis by Walter Siegenthaler (Mar 21, 2007)
6. Symptom to Diagnosis: An Evidence Based Guide, Second Edition (LANGE Clinical Medicine) by Scott D. C. Stern (Sep 16, 2009)

7. CURRENT Diagnosis and Treatment Emergency Medicine, Seventh Edition (LANGE CURRENT Series) by C. Keith Stone (May 23, 2011)
8. Harrison's Gastroenterology and Hepatology by Dan Longo and Anthony Fauci (May 3, 2010)
9. Mayo Clinic Gastroenterology and Hepatology Board Review (Mayo Clinic Scientific Press) by Stephen Hauser (Jun 23, 2011)
10. Clinical Nephrology 2012 (The Clinical Medicine Series) by M.D., C. G. Weber (Sep 19, 2011) - Kindle eBook
11. Goldman's Cecil medicine / [edited by] Lee Goldman, Andrew I. Schafer.—24th ed. Elsevier Sanders. Rev. ed. of: Cecil medicine. 23rd ed. – 2012. p.
12. Sonographer's Handbook of Diagnostic Ultrasound by Jason R. Young M.D. (Feb 23, 2011)

**Additional literature:**

1. Kovalyova O.M., Asheulova T.V. Propedeutics to internal medicine. Part 1, Diagnostics. Vinnytsya, Nova Knyha, 2006, 424 p

**Composed by**

**N.P. Prikhodko**