Z.Kheladze,Zv.Kheladze

Standard requirements of treatment of Hemorrhagic insult for critical care medicine clinics of limited resources

Critical Care Medicine Institute,Tbilisi,Georgia

Here are discussed problem of ischemic insult and treatment standard of this disease in critical care medicine clinic. This standard includes diagnosis, care, treatment conditions and is supposed to be utilized by countries of limited resources in critical care medicine clinics. So, bed cost of each day is not more than 300,0-500,0 US.

Key Words: Ischemic insult, Critical patients, Blocks of information, Blocs of action, Standards

Term itself means blow. It expresses acute derangement of brain blood flow. Characterized by suddenly (in minutes and hours) developed focal neurological symptoms and general cerebral disorders. Most wide spread reason of ischemic stroke is brain vascular emboli. Source of emboli is most frequently heart, less frequently arterial-arterial embolus, which develops in vertebra-basilar or carotid system from atherothrombotic area. Reasons of cardiac genesis embolus are atrial fibrillation or other disorders of heart rhythm, myocardial infarction, bacterial endocarditis, prosthetic valves, myxoma, trichinellosis, etc. Reasons of noncardiac genesis emboli are atherosclerosis of aortic and carotid arteries, pulmonary vein thromboses, fat emboli, tumor emboli, air emboli, complications developed after surgery on thorax and abdominal cavity. In most cases of stroke there are preclinical manifestations, but in 20% of cases stroke may develop without previous manifestations. Ischemic stroke develops in the specific area of the brain after decrease of blood flow or full cessation. It is caused by angiospasm, thromboses or emboli. It is followed by disorder of brain matter blood supply in an irrigative area of obstructed arterial vasculature and development of softening, encephalomalacia, necrosis and stroke. During ischemic stroke following types are differentiated: athero-thrombotic – mechanism of development is damage of brachiocephalic arteries, large intracerebral arteries and aortic arch after thrombosis. Arterio-arterial emboli on the background of brachiocephalic artery atherosclerosis lead to an intracerebral arterial occlusion. According to the diameter of an occlusive blood vessel, develops stroke localized subcorticaly or in an anterior, middle and posterior pool of blood supply. Cardiac emboli – mechanism of development is by the result of intracardial thrombus formation developed cardiogenic embolus. Source of emboli is left atria or ventricle. Cardiogenic reasons for thrombus formation are infectious endocarditis, aortic aneurism, mitral stenosis, area of

myocardial infarction, postinfarction aneurism of heart, atrial fibrillation, etc. Vascular – based on disorder of cerebro-vascular blood supply during the decrease of arterial pressure (hypotensive medications, orthostatic arterial hypotonia, etc.) and decrease of cardiac output during myocardial ischemia. Lacunar stroke – mechanism is primary damage of the main arteries of brain, anterior, middle and posterior brain artery proximal branches small perforating arteries. Hemo-reological microocclusive type of stroke – mechanism of development is based on the disorder in haemostatic and fibrinolytic system. From hematologic changes should be mentioned erythrocytosis, hemoblastosis, hypercoagulation, polycythemia, sickle cell anemia, thrombocytopenic purpura and other diseases. In the development of stroke important role has blood viscosity, which is determined by hematocrit index. Its decrease leads to decrease of oxygen in blood. Increase of hematocrit is accompanied by the increase of blood viscosity and worsening of reological property, which during the ischemic stroke widens size of the stroke. Blood viscosity decreases in brain capillaries and comprises 2/3. Reological properties of thrombocytes and erythrocytes have influence on blood viscosity, which in turn depends on their cellular and membranous specificities – in particular, on deformation ability of erythrocytes. For example – appearance of more than 65% of rigid erythrocytes causes increase of the hematocrit (content of rigid erythrocytes are regulated by spleen). In the changes of blood viscosity, major role plays also ability of platelets to aggregate. In large vessels erythrocyte aggregation is reversible and depends on many factors – electrical properties of cell, content of fibrinogen, osmolality of blood plasma, blood pH and blood flow velocity gradient. In laminar flow of the blood maximal numbers of aggregates are formed in the central stream and breaks down near blood vessel wall. During increase of erythrocyte aggregation, thrombocytes move into the center of the stream, where they get mechanical damage, as a result blood viscosity increases. As thrombocytes practically never deform, blood viscosity depends on their concentration. Thrombocytic aggregates develop reversely less; they obstruct vascular lumen or increase intravascular coagulation.

Preserving optimal parameters of brain bleeding during the change of arterial pressure is limited. Its auto regulation depends on perfusion pressure, normal value of which is 75-90mmHg. Cerebral perfusion pressure (CPP) represents difference between mean arterial arterial pressure and cerebral venous pressure. Latter almost corresponds to CSF pressure and normal value is 150-80mmH2O. During ischemic stroke volume of dead neurons depends on perfusion pressure. Decrease of perfusion occurs by the influence of different factors. Brain cells are particularly vulnerable to hypoxia. During the decrease of blood flow less than 55ml/100g.min. protein synthesis is deranged and manifests reversible neurological disorders and during the decrease to less than 35ml/100g.min glycolysis is activated, malonic acid is

accumulated and develops lactic acidosis. Blood rheological properties and microcirculation are deranged. General peripheral resistance of blood vasculature increases. Decrease of brain blood circulation to less than 20ml/100g.min. (upper limit of ischemia) causes acceleration of neuromediators (glutamate and aspartate) discharge. Adhesion and aggregation of thrombocytes are reinforced and blood clotting ability increases. And during the decrease below 12ml/100g.min (lower limit for ischemia) ATP synthesis is suppressed, cell membrane function is deranged; there is disorganization of K/Na pump, which determines accumulation of Ca in cell. During such decrease of blood flow, in corresponding brain area after 6-8 minute develops “nuclear area” of infarction. It is an irreversible damage area, which during 3-6 hour is surrounded by ischemic area. In letter blood flow is impaired 20-40ml/100g.min. It is called ischemic “half-shadow zone”. Duration of this zone is individual, but usually is determined by 3-6 hour and during this time disorder of neuron function has reversible nature. Duration of presence of “ischemic half-shadow zone” determines maximal effective period of treatment. Main part of the infarction zone is formed after 6±2 hour from manifestation of the first symptoms of stroke. Final formation of foci lasts 3-7day.

“Brain ischemia” and “brain infarction” is non identical. Brain ischemia may lead to the formation of infarction or determine temporary disconnection of function of neuronal structure. In addition, reversible nature of brain function disorder not always indicates truly restoration of damaged structure and non existence of their morphological defects. Brain ischemia leads to the deficit of energetical – macroergic “ATP, phosphocreatine”, which is accompanied by inhibition of aerobic utilization of glucose and stimulation of anaerobic utilization. Letter is transformed into malonic acid. Malonic acid excess leads to the development of lactic acidosis, which causes vasodilatation, (vasoparesis) and hyper perfusion in an ischemic zone. It deepens metabolic disorders and there is disorganization of ion transporting ferment system. Vasoparesis is one of the leading pathogenetic factors of ischemic stroke, so use of vasodilatation enhancing factors and such perfusion of ischemic tissue is unjustifiable. Disorganization of ion transporting ferment system and disorder of metabolism breaks respiration of cell mitochondria, also their membranous potential and causes depolarization. Cell depolarization is accompanied by the increase of cell membrane permeability. Depolarization also activates oxidation recovery and arachidonic acid cycle, causes disorganization of Na, K pump, accumulation of fatty acids and free radicals, increase of neurotransmitters (glutamate and aspartate) production. These processes conduct sodium and potassium ions, also flow of water molecules in neurons, which causes swelling of the neurons and disorganization of their function. During ischemic stroke brain swelling is maximally expressed in infarction nucleus. At the beginning swelling has intracellular and then vasogenic

nature, due to the damage of blood-brain barrier. Swelling causes increase of brain volume, its pressure on vasculature and decrease of perfusion pressure, hypoperfusion, hypoxia and increase of swelling is accompanied by the dislocation of stem structure and strangulation.

In the pathogenesis of stroke sequence of mutual provoked changes is determined as “ischemic Cascade” or “Glutamate Calcium Cascade” and consists of eight stages: first stage-impairment of blood flow (till the upper limit of ischemia), second stage-glutamate “excitement toxicity” (hyper production and accumulation of exciting – irritating mediators – glutamate amino acids), third stage – intracellular accumulation of calcium ions, also water and sodium (result of the cytotoxic effect of aspartate and glutamate), fourth stage – intracellular activation of enzymes (protein kinases and others) by the derangement of mitochondrial function, fifth stage-enhancement of NO synthesis and development of oxidative stress, sixth stage-expression of early response apoptosis inductor genes (derangement of oxidation-reduction processes at the expense of blocking mitochondrial complexes, interaction of calcium excess with enzymatic systems and nitric oxide. irreversible damage of intracellular structures and neurons itself), seventh stage-far results of ischemia (local inflammatory reaction by releasing inflammatory agents, which increases risk of transforming ischemic area into hemorrhagic. microvascular disorders, damage of blood-brain barrier), eighth stage is apoptosis.

Stroke is preceded by following signs: dizziness, loss of consciousness, loss of vision, general weakness and transient paresthesias in different parts of the body. But stroke may also develop without preceding signs. And in 20% of cases stroke is asymptomatic (included hemorrhagic). Ischemic stroke is more frequent at >50 years of age. It develops more frequently at night. Consciousness is spared, there is no head ache, or it may be weakly expressed. Arterial pressure may be normal or even decreased. Skin and mucous is pale. Temperature may be normal or subfebrile. There are no meningeal signs or is weakly expressed. In contrast to general cerebral signs ischemic stroke is characterized by the dominance of typical focal signs. Speed of the symptom development depends on the mechanism of stroke and on the subtype species. And the signs of focal damage depend on which vasculature is damaged –anterior, middle or posterior cerebral artery, spinal artery or other vasculature. Sometimes there may be coincidence of damage of different vasculature.

Anterior brain artery atherosclerosis in the proximal segment rarely causes ischemia. Occlusion is compensated by collateral blood flow from anterior connecting artery. Probability of ischemia increases when there is a congenital atresia of collateral blood flow or there is atherosclerotic changes in the distal part of the artery. Occlusion is frequently resulting from emboli. Occlusion of the general branch causes such neurology, which is determined by the

loss of blood circulation in both arterial pools. Atherosclerotic damage of middle brain artery may cause ischemic symptoms in one or more distal artery segment of lenticular nucleus and striped body, which supplies deeply situated areas of white matter and subcortical notches. Occlusion develops as a result of emboli and stenosis of this artery with thrombus is rare. Total occlusion of middle cerebral artery opposite to the damage causes hemiplegia, with predominant upper limb paralysis. Right sided hemiplegia is accompanied by aphasia of different degree and nature. During the occlusion of separate branches of middle vertebral artery develops incomplete syndromes: during upper branch-motor aphasia (Broca's aphasia), with contralateral hand paresis and paresis of the mimic muscle of the lower half of the face; during lower branches-sensory aphasia (wernicke's aphasia) thrombosis of deep branches causes capsular hemiplegia, which may be accompanied by hemianesthesia. Atherosclerotic damage in carotid artery pool causes stroke in the beginning of blood vessel, less in the carotid siphon or proximal segment of middle and anterior carotid artery. An atherosclerotic thrombosis of an extra cranial part of the carotid artery is localized in neck area, in bifurcation area or in the area of carotid sinus. It is followed by the development of alternative optic-pyramidal syndrome, loss of vision on the side of pathological processes and on the opposite side hemiparesis. During the occlusion of common carotid artery in addition to the full and partial vision loss there is changes at the bottom of the eye. Atherosclerotic damage of the proximal part of the internal carotid artery is located at first 2cm of the posterior edge. Often it spreads downward at the distal part of the common carotid artery. In 80% of cases such damage manifests as small stroke or transition ischemia. Small thrombocytic emboli of internal carotid artery obstruct distal branches of middle cerebral artery or orbital artery. They cause transition vision loss or asymptomatic infarction. Large emboli may obstruct primary or secondary branches of middle cerebral artery. Some of the emboli are so large that they obstruct proximal branch of the middle cerebral artery and cause ischemia of middle cerebral artery pool. During atherosclerotic thrombus developed in the siphon area of internal carotid artery, on the opposite side develops hemiplegia, hemianesthesia, hemianopsia and aphasia because of the brain edema and dislocation of brain stem. Frequently there is deterioration of vital functions. Ischemic process may develop simultaneously on both side or separately. Develops pseudo bulbar paresis; pyramidal deficits; and oral automatism; strengthening of facial and soft palate reflexes; involuntary laughing and crying. Patient has ability to swallow and has voice. Total obstruction of the posterior part of the cerebral artery causes multiple clinical pictures, separate obstruction is less symptomatic. Syndromes of middle cerebral, subthalamic and thalamic injuries are connected with stenoses of pre common segments of posterior cerebral artery or distal parts of its penetrating branches. Syndromes of cortex injury are connected with occlusion of post common segment of posterior cerebral artery. During occlusion of posterior

cerebral artery stem develops unilateral or bilateral infarction of subthalamus and medial thalamus. Also damage of the brain peduncles and mid brain injury with accompanying clinical symptoms. During atresion of posterior connecting artery develops symptoms of peripheral injury. High level injury is characterized by the injury of red nucleus and dento-rubro-thalamic pathway and manifests as contra lateral ataxia. Low level injury manifests as paralysis of III cranial nerve and contralateral ataxia (Klode syndrome) or contralateral hemiplegia (Weber syndrome). Occlusion of subthalamic paramedian artery causes paralysis of sight above, abulia and euphoric condition. Occlusion of thalamic and thalamic-genicular branch causes contralateral sensory loss. Sometimes disorders are seen in facial, hand, body, leg and foot areas. Rarely, infarctions in the visual hill may be caused by the occlusion of posterior thalamic penetrating branch of post common part of the posterior cerebral artery. Occlusion of the peripheral part of the posterior cerebral artery frequently causes development of infarction with hemianopsia at critical surface of temporal or medial part of the occipital area. Bilateral infarction at distal part of the posterior cerebral artery blood supply pool causes cortical blindness. At spinal artery pool as the result of failure of blood supply picture of ischemia is multiple. On the damaged side develops paralysis of soft palate and vocal cord, Horner's syndrome and vestibular-cerebral symptoms. Opposite to damage there are dissociation anesthesia, swallowing and speech disorders. Infarction of cerebrum together with brain swelling may cause sudden stop of breathing which is connected with an increased pressure in the posterior skull cavity. Not so rarely with the stoppage of breathing. It may also manifest by hypersomnia, babinski's symptom, dysarthria and bilateral weakness of facial muscles. Early symptoms during this period may be loss of coordination, dizziness, seizure and vomiting. Stroke developed as the result of atherotrombotic occlusion causes bilateral syndromes of stem injury. Sometimes we can think of bilateral ischemia when there is visual paresis or nuclear ophthalmoplegia together with ipsilateral hemiparesis. When atherosclerotic occlusion of common artery branch causes clinical manifestation, it is accompanied by the symptoms of sensory, motor pathway and unilateral cranial nerve injury. Occlusion of upper artery of cerebrum causes ipsilateral ataxia and contralateral failure of temperature sensation. Sometimes partial hearing loss, ataxic tremor in upper limbs and Horner's syndrome is possible. Occlusion of anterior-inferior artery of cerebrum causes ipsilateral deafness, weakens of mimic muscle, cerebral ataxia, Horner's syndrome and paresis of horizontal gaze. On contralateral side body looses pain and temperature sense. Indicator of ischemia at base artery is coma, Cheyne-Stokes respiration, hyperthermia, hormeotonic type of contracture on all four limbs and bulbar disorder. Lacunar strokes may manifest by motor hemiparesis.

Bloc of information

Code: 5353	Critical	Treatment period : 14 bed-days
Level of medical support : II - III - IV		
I63	Brain infraction (critical condition)	

level	code	Pharmacologic treatment			number	
		Name	dosage	unit	Essential	Recommendation
	B__01	Block N 1-1, 1-2 – reception of patient			1	1
	B__02	Block N 2 -1, 2-2, 2-3, 2-4, 2-5 - diagnosis			1	1
	B__03	Block N 3-1, 3-2 – basic and aiding means			5	5
	B__04	Block N 4 - treatment of patient			2	2
	B__05	Block N 5 – water and electrolytes			2	2
	B__06	Block N 6 – correction of metabolic acidosis			1	1
	B__08	block N 8-2, 8-3, - nutrition			4	4
	B__11	block N11-pain management			2	2
	B__12	BlockN12-sedation			2	2
	B__23	BlockN23-treatment of hyperthermia			2	2
	B__25	block N 25-3 – antibacterial therapy			6	6
	B__29	block N 29-1, 29-2, 29-3 – respiratory correction APV			1	1
	B__34	block N 34 – non-differentiated therapy			1	1
	B__46	block N 46 – protection of intestinal mucosa			6	6
	B__47	block N 47 stimulation of digestion			3	3
	B__48	BlockN48-liquidation of bronchial spasm			2	2
	B__49	blockN490-enteric nutrition			3	3

Result:	Recovery of vital functions, improvement of clinic-laboratorial analysis, Elimination of critical condition

block N 1-1	
name	reception of patient:
subject:	Critical care medicine doctor, nurse, sanitarian
controlling:	Head of critical care medicine service
dates of implementation :	First hour of patient in clinic
characteristics of work:	Replace patients into bed
	Cleaning up of breathable ways
	Oxygen provision
	Attaching with monitor
	Catheterization of peripheral vein
	catheterization of urine bladder
	Send blood and urine for lab tests
	ECG monitoring
Implement:	block 1-2
Indication:	All critical patients
Contradiction:	No
Result of implementation:	A patient is placed into bed, under permanent monitoring and all actions are performed as written in protocol
Notes:	Cancellation of block, work or conditions of change
Signing: doctor:	

block N 1-2	
name	reception of patient:
subject:	Critical care medicine doctor, nurse, sanitarian
controlling:	Head of critical care medicine service
Dates	the first hour of patient in clinic
Characteristics of work:	Evaluation of patient's condition by severity in aids of analogous digital scale, monitoring
Indication:	Evaluation of all patients according to digital-analogous scale
Contradiction:	No
Result:	Patient is involved in monitoring, condition is evaluated as---- points
Notes:	Cancellation of block, work or conditions of change
Signing: doctor	

block N 2-1	
name	diagnosis
subject:	Critical care medicine doctor, nurse, sanitarian, lab assistant, nurse assistant, radiologist
controlling:	Head of critical care medicine service
Dates:	First 6 hours of patient in clinic
	Following tests must be carried out:
	ECG
	X-ray of chest
	Blood test

	Urine test
	Coagulogram
	Electrolytes
	Balance of airs in blood and acid-alkaline
	Creatinine
	Sugar
	Bilirubine
	Total albumin
	Glasgow scale
	APACHE II scale
	Calculation of blood components in circulation
	Total analysis of liquor in pleura
Indication:	All critical patients
	Analysis of liquor is performed when there is a doubt about meningeal encephalitis
	Pleural punctate test is conducted when there is more than 700ml liquid in pleural cavity or in order to determine processes in pleura
Contradiction:	Take of liquor and pleural pulctate can be postponed if there are acute respiratory failure and failure of blood circulation
	Liquor test can be postponed if there is a doubt on voluminous process in brain until CT of brain
Note:	Each tests are conducted once a week and in this period parameters that are sharply deviated from norm are also studied or it is doubtful that they are changed after one week of treating
Result:	Patients has all tests according to diagnosis
Signature : doctor	
block N 2-2	
name	diagnosis
subject:	Critical care medicine doctor, nurse, radiologist, endoscopist
controlling:	Head of critical care medicine service
Dates:	First 24 hours in clinic
Description:	
Following tests must be carried out:	Echoscope
	Echocardioscope
	ECG
	Transcranial Doppler
	Fibro-gastro-deudenoscopy
	Bronchoscopy
Indication:	Echocardioscopy and transcranial Doppler must be conducted with all patients
	EEG at the time of post-anoxia injury, also in case of convulsive syndrome სინდრომების დროს.
	Gastroscopy+gastro-duodenal bleedings
	Bronchoscopy: urgent pathology of bronchi, impossibility to determine disorder of traumatic ways in aids of less invasive method.:
Contradiction:	Gastroscopy and bronchoscopy can be postponed in patients above 70 years because of Acute respiratory disorder or blood circulation problems after decision of endoscopist.

result of work:	The studies were carried out to all patients in the study, except -
Notes:	These diagnostic methods are used in the case diagnostic tools used when found ineffective or less informative
Block, Cancellation, suspension or change working conditions.	
Signature: doctor	
block N 2-3	
name	diagnosis
subject:	Nurse radiologist, doctor radiologist
controlling:	Head of critical care medicine service
dates of implementation :	First 72 hour in clinic
Description:	CT
	Nuclear magnetic resonance test
	angiography or CT angiography
indication:	These studies are done with all of the critical patients when damaging or organs and tissues are likely to be.
	Nuclear magnetic resonance test is conducted when CT and other tests are not informative enough
	CT angiography is conducted in case of likelihood of aneurism
Contradiction:	MRI is not subjected to artificial ventilation of lungs patients or patients with any metal in the body at the time
result of work:	Necessary test is conducted with patient so that to diagnose a pathology
Notes:	Examinations are financed by the insurance company and the patient's owner.
Block, Cancellation, suspension or change working conditions	
Signature: doctor	
block N 2-4	
name	diagnosis
subject:	Critical care medicine doctor, nurse, laborer
controlling:	Head of critical care medicine service
Dates:	First 72 hours in clinic
Description:	Bacteriologic test
	Viral test
	Immunologic test
	Diagnostics of myocardial attack
	Diagnostics of severe sepsis
	Toxicological tests
	Hematologic test
indication:	These studies need to be done infectious, viral, immunological, toxicological, hematological pathologies, as well as myocardial infarction or sepsis are suspected.
Contradiction:	No
Result:	Patient has following analysis.
notes:	Examinations are financed by the insurance company and the patient's owner.

Signature: doctor	
block N 2-5	
name	diagnosis
subject:	Consultant
controlling:	Head of critical care medicine service
Dates:	48 hours after confirmation of consultation
Descriptions:	Following consultations must be carried out:
	Therapeutist
	Cardiologist
	Neuropathologist
	Neurosurgeon
	General surgeon
	Endocrinologist
	Hematologist
	Angiologist
	Oncologist
	Other specialist
indication:	Indication of consultation can be such problem or which cannot be identified or the implementation of the Protocol or its holding beyond the boundaries of critical care medicine doctor's competence, such as surgery, chemotherapy, and other.
contradiction: :	no
result of work:	Patient has mentioned consultation with doctor.
Notes:	The patient has consultations depending on the most severe symptoms.
Block, Cancellation, suspension or change working conditions	
Signature: doctor	
block N 3-1	
name	Provision with basic means
subject:	Critical care medicine doctor, nurse
controlling:	Head of critical care medicine service
Description:	Patient must be provided with basic means
	Catheter (one-time)
	Tube of tracheostomy
	Gastral pump
	Needle for lumbar puncture
	Intubation tube
	Catheter of Foley
indication:	All critical patients
Contradiction:	no
Result:	Provision with basic and aiding means
notes:	
Block, Cancellation, suspension or change working conditions	
Signature doctor:	

block N 3-2	
name	Provision with basic means
subject:	Critical care medicine doctor, nurse, sanitarian
controlling:	Head of critical care medicine service
characteristics	Patient must be provided with aiding means
	Cotton
	Bandage non-sterile
	Syringe (onetime)
	Alcohol
	Iodine
	Glove
	System of transfusion
	Syringe (10ml)
	Syringe(15ml)
	Syringe(20ml)
	Plaster
	poliviline tube
	Butterfly
indication:	All critical patients
contradiction: :	No
Result:	Basic and aiding means
Notes:	
Block, Cancellation, suspension or change working conditions	
Signature:doctor:	
block N 4	
name	Take care of patient
subject:	Critical care medicine nurse, sanitarian
controlling:	Head of critical care medicine service
Dates:	Once a day 10.00
Characteristics:	Cleaning up mouth with antibacterial solutions
	Prevention of bed sores (by means of camphoric alcohol, washing procedures)
	Bowel movement per2-3 days and if necessary enema
	Enteric nutrition 4 times a day according to defined calories in advance Take care of vein and urine bladder catheters
	Usage of gloves and onetime means
indication:	All critical care patients
Contradictions:	No
Result:	Patient is cleaned up, maintained
notes:	Onetime cleaning up procedure of patient with wet wipes Block, Cancellation, suspension or change working conditions
Signature: doctor	

block N 5	
name	Provision with water and electrolytes
subject:	Critical care medicine doctor, nurse
controlling:	Head of critical care medicine service
Dates:	
Description:	Sodium chloride 0,9% 500მლ.
	Potassium chloride 4% 200მლ.
	Calcium chloride or gluconate 10-30მლ.
	Magnesium sulphate 25% 15ml per day
Indication:	All critical care patients
Contradiction:	A higher than normal rate of electrolyte
result of work:	Water and electrolyte balance is corrected
Notes:	Block, Cancellation, suspension or change working conditions
Signature: doctor	
block N 6	
name	Correction of metabolic acidosis
subject:	Critical care physician, critical care nurse.
controlling:	Head of critical care medicine service
Dates:	
Description:	
indication:	Metabolic acidosis
contradiction: :	Metabolic alkalosis
Result:	Correction of metabolic acidosis
notes:	Bicarbonate dose is calculated by a special formula individually.
Block, Cancellation, suspension or change working conditions	
Signature: doctor:	
block N 8 -2	
name	Enteral feeding
subject:	Critical care medicine doctor, transfussiologist, nurse
controlling:	Head of critical care medicine service
Dates:	
Description:	About 3500-4500kkal
	-1 G per kilogram of body weight. Protein, 1 g. Fat and 4 g. Hydrocarbons.
indication:	All critical patients
Contradictions:	No
result of work:	Critically ill patient gets energetic feeding
Notes:	Food is provided orally, Nazo / or gastric probe and gastrostomy
Block, Cancellation, suspension or change working conditions	
Signature: doctor:	
block N 8 -3	
name	Compound nutrition
subject:	Critical care medicine doctor, transfussiologist, nurse
controlling:	Head of critical care medicine service
Dates:	
Description:	About 3500-4500kkal
	-1 G per kilogram of body weight. Protein, 1 g. Fat and 4 g. Hydrocarbons.

	500 ml of 40% glucose and insulin in 40, 2 times a day with the infusion rate of 100 ml / h
	The amino acid mixture - 500 ml. 1-2jer day parallel to glucose 100 ml / hr
indication:	All critical patients
contradiction: :	No
Dates:	Critically ill patient gets energetic feeding
notes:	Intravenous infusion, per orally, naso-gastral probe or gastrostomy, after calculating calories
Signature: doctor:	Block, Cancellation, suspension or change working conditions
block N 11-1	
name	Pain relief drugs and psychotropic medicines
subject:	Critical care medicine doctor, nurse
controlling:	Critical care medicine doctor
შესრულების დრო:	First 30 minutes after pain phenomenon
სამუშაოს დახასიათება პაციენტს უკათდება:	Fentanyl dose of 1-3 mg / kg intravenously slow. 1-2 min after the injection. Infusion: 1-3 Mcg / kg / hr. Validity of 0,5-1 hours.
	Morphine dose 0.1-0.2mg / kg, the infusion: 10-80 mcg / kg / min. Duration 3-4 Hours.
	Ketamine dose: 20-50mg. Iv Infusion: 0.5-2mg / kg / hr. Validity Start 1 min.
	Midazolam (Dormicum) intravenously 2.5 mg (initial dose).
indication:	Traumatic shock, myocardial infarction, postoperative period, any pain that requires analgesia.
Contradictions:	Fentanyl Contraindications: bradycardia, chest rigidity, vasodilatation, hypoventilation, vomiting, nausea, constipation. Morphine Contraindications: vasodilatation, hypoventilation, vomiting, nausea, constipation. Ketamine contradictions: head injury, intracranial hypertension, ischemic heart damage, hypertension. Ketorolac Contraindications: dyspepsia, nausea, vomiting, hallucinations, insomnia, hypertension.
result of work:	Reach to anesthesia
Notes:	Consumption of narcotic analgesics must be indicated in the registration journal, which indicates a time, series, the patient's name and number of the history, signature of doctor
Block, Cancellation, suspension or change working conditions	
Signature: doctor:	
block N 11-2	
name	Pain management with non-addictive drugs
subject:	Critical care medicine doctor, nurse
controlling:	Head of critical care medicine service
Dates:	First 30 minutes from pain phenomenon
	Ketorolac (Toradol) dose: 30 mg. one time per 6 hours 120mg per day, action time 10 min
	analgin 50% 2,0ml intravenously
	Nonsteroidal anti-inflammatory drugs for example: (diclofenac) 75 mg. IM
indication:	Postoperative period, any pain that requires analgesia.
contradiction: :	
Ketorolac Contraindicatio ns	Dyspepsia, nausea, vomiting, hallucinations, insomnia, hypertension. The asteroid ignition anti-drug: gastric ulcer and ulcer of duodenum.

Dates:	anesthesia
notes:	Consumption of narcotic analgesics must be indicated in the registration journal, which indicates a time, series, the patient's name and number of the history, signature of doctor
Block, Cancellation, suspension or change working conditions	
Signature: doctor:	
block N 25-3	
name	Antibacterial therapy
subject:	Critical care medicine doctor, nurse
controlling:	Head of critical care medicine service
Dates:	15-21 days
Description:	fosfomicin (monaural) 3gr per day
	Or dominal 400 gr per day
indication:	Existence of inflectional process
Contradictions:	Allergic reactions towards any antibiotic
result of work:	Prevention and treatment of inflectional process
Notes:	Or according to antibacterial sensibility
Block, Cancellation, suspension or change working conditions	
Signature: doctor:	
block N 29-1	
Name	Oxygenation of critical patient on spontaneous breathing
subject:	Critical care medicine doctor, nurse
controlling:	Head of critical care medicine service
Dates:	
Description:	Oxygen is supplied in the following ways: 1. Nasal cannula, 2. Simple maks, 3 mask of venture, 4. Mask with reservoir-24-60% concentration, by 1,6l/min
indication:	All critical patients
contradiction: :	no
Result:	Showings of external respiration are satisfactory
Notes:	Oxygenation indicators must be maintained for 5 l / min in light of SO O2-90% - = high. Otherwise, moving to pulmonary ventilation.
Block, Cancellation, suspension or change working conditions	
Signature: doctor:	
block N 29-2	
name	Oxygenation of patient on spontaneous respiration by CPAP face mask.
subject:	Critical care medicine doctor, nurse
controlling:	Head of critical care medicine service
Dates:	
Description:	Patient has pulmonary ventilation by menas of CPAP mask on the background of oxygen provision FIO2 60%. 15-30min
	Then oxygenation with cannula again
indication:	All critical care patients
Contradictions:	no
result of work:	Showings of external respiration are satisfactory

notes:	Oxygenation indicators must be maintained for 5 l / min in light of SO O2-90% - = high. Otherwise, moving to pulmonary ventilation.
Block, Cancellation, suspension or change working conditions	
Signature: doctor:	
block N 29-3	
name	Artificial pulmonary ventilation
subject:	Critical care medicine doctor, nurse
controlling:	Head of critical care medicine service
Dates:	
Description:	Initial parameters of ventilation in adults
	FIO2100% maintaining < 60%, to avoid toxicity of oxygen and pulmonary injury
	Respiration rate (ლღ) 8-12min. 18-24/min to reach therapeutic ventilation
	Regimes SMV, SIMV, SIPAP, BYPAP
	Inhalation volume (TV) 6-8ml/kg.
	Inhalation flow speed (IFR) 60ლ/წთ
	Inhalation/exhalation correlation (I/E) 1/2½- 1/3.
	Plateau pressure <35სმ H2O must be maintained low that maximal showing to avoid barotrauma
	(PIP)<45 სმ. H2O
	pressure support and positive end-expiratory pressure (PEEP) 5sm. H2O.
	Cardiac volume in patients with hypovolemia
indication:	Respiratory failure caused from various pathologies
contradiction: :	Bullous lungs
Result:	Patient has artificial pulmonary respiration and all necessary procedures
Notes:	Applications of all parameters is not easy during named regimes because technical characteristics of various respiratory apparatus are different: rate >20 can be increased by PEEP. Elevated volume can cause danger of barotraumas and cause damage associated with ventilator. Extremely low showing of IFR can increase PEEP because of lack of inhalation time. Extremely high showing of IFR can increase PIP. Increased correlation can be effective at the time of acute obstruction or COPD. Transverse correlation (2:1) is used for elevation of PAO2 at the time of severe hypoxia. PIP must be maintained at low levels(is less important than pressure of plateau)
Block, Cancellation, suspension or change working conditions	
Signature: doctor:	
block N 34	
name	Non-differentiated therapy
subject:	Critical care medicine doctor, nurse
controlling:	Head of critical care medicine service
Dates:	
description	

drug	dosage	unit
Diazepam	10mg/2ml	amp
Ketamine	500mg/10ml	bottle
Atropine	1mg/1ml	amp
Adrenaline	1mg/ml	amp
Prednisolone	30mg/ml	amp
Clonidine	0,15mg	injection
Digoxin	50mkg/ml	1ml
Dopamine	200mg/5ml	bottle
Forudemid	20mg/2ml	amp
Insulin short action	400 IU/10 ml	bottle
Neostigmine	2,5mg	bottle
Aminophylline injection	250mg/10ml	amp
Calcium glukonate	0.1	amp
Lidocaine injection	10% 100mg/5ml	amp
indication:		All critical patients
Contradiction:		no
result of work:		
notes:		
Block, Cancellation, suspension or change working conditions		
Signature: doctor:		

block N 45	
name	Recover of receptor's sensibility
subject:	Critical care medicine doctor, nurse
controlling:	Head of critical care medicine service
Dates:	
Description:	prednisolone 30mg 1ml amp
	dexamethasone 4მგ.1ml amp
indication:	Intoxications of various kinds, collagenoses, myasthenia, polumieloradiculoneuritis, chronic obstruction disease of lungs, hydrothorax
contradiction: :	Disease of gastro-duodenal tract
Result :	Recovery of sensitiveness of receptors
Notes:	On the background of protection of stomach mucosa
Block, Cancellation, suspension or change working conditions	
Signature: doctor:	

block N 46	
name	Protection of stomach mucosa
subject:	Critical care medicine doctor, nurse
controlling:	Head of critical care medicine service
Date:	Permanently
Description:	Blocker of hydrogen ions (omeprazole, lansoprazole 1 caps per day, in the morning before meal),.
	Patients of gastro-intestinal disease in anamnesis need inhibitor of H2 receptors: for example: Zantac 25mg-1ml 2 times a day
	Gastric mucosa exhibition means Simalgel 10ml 4 times or sucralphate 1gr 2 times 20 min before meal 1გრ 2ჯერ კვების წინ 20 წუთით ადრე.
indication:	All patients especially one with pathologies of gastro-intestinal tract.

Contradictions:	no
result of work:	Protection of stomach mucosa
notes:	
Block, Cancellation, suspension or change working conditions	
Signature: doctor:	
block N 47	
name	Stimulation of bowel movement
subject:	Critical care medicine doctor, nurse
controlling:	Head of critical care medicine service
Dates:	Per 2-3 days
Description:	Guttalax
	cleaning enema at at 2-3 days
	If peristaltic is slow then proserin 1ml amp before 20 min before enema ით ადრე.
indication:	All critical patients
contradiction: :	No
	Regulation of bowel movement
notes:	Enema must be conducted carefully in case of cardiac pathologies and thrombosis and prozerine- epilepsy,hyperkinesis, bronchial asthma, stenocardia and intestinal obstruction of mechanical character
Block, Cancellation, suspension or change working conditions	
Signature: doctor:	

References:

1.Z.Kheladze,Zv.Kheladze-“Critical Care Medicine”, First book, Tbilisi, Georgia, 2015,-300pp.

2.Z.Kheladze,Zv.Kheladze-“Critical Care Medicine”, Second book, Tbilisi, Georgia, 2016,-320pp

ზ.ხელაძე,ზვ.ხელაძე

იშემიური ინსულტის მკურნალობის სტანდარტული მოთხოვნები შეზღუდული რესურსების მქონე კრიტიკული მედიცინის კლინიკებისთვის

კრიტიკული მედიცინის ინსტიტუტი,თბილისი,საქართველო

განხილულია იშემიური ინსულტის პრობლემა.მოტანილია აუცილებელი ინფორმაცია იშემიური ინსულტის შესახებ.აქვეა წარმოდგენილი კრიტიკული მედიცინის კლინიკაში იშემიური ინსულტის მკურნალობის სტანდარტი. ეს უკანასკნელი მოიცავს ავადმყოფის დიაგნოზის,მოვლის და მკურნალობის პირობებს, სტანდარტი გამიზნულია შეზღუდული რესურსების მქონე ქვეყნების კრიტიკული მედიცინის კლინიკებისათვის.ამ თვალსაზრისით თიოთოეული საწოლ დღის ღირებულება არ აღემატება 300,0-500,0 აშშ. დოლარს