I ask you to stand for silence minute in memory of Lord Rector of KNMU academician Tsyganenko Anatoly Yakovlevich

The traumatic shock. The prehospital management. The blood replacement in trauma patients.

Professor of the department of anesthesiology, traumatology and emergency medicine

Fesenko Olga Khamitovna

Specificity of battle trauma

The bleeding is the cause of death in 50%.

The management of patients on the battle field includes three stages:

- The first aid under gunfire according the protocol MARCH (massive bleeding, airway, respirations, circulation, head).
- The medical aid after stopping gunfire on the battlefield includes: cardio-pulmonary resuscitation, identification of severity of trauma, immobilization of fractures, wound bandage applying, analgesia, antibiotics. The main goal on this stage – hemodynamic stabilization by stopping the bleeding.
- Evacuation.

Grading the injured soldiers for evacuation

Marking the patients according to the severity of trauma:

- black – the pulse and breath are absent– resuscitation on the field; they should not been transported;
- red – life-threatening trauma – transportation in the first place;
- yellow – serious trauma, but not life-threatening– transportation in the second place;
- green – walking patients – transportation last of all.

RTS (Revised trauma score)

Signs	0	1	2	3	4
Consciousness (Glasgow coma scale)	3	4-5	6-8	9-12	13-15
Systolic blood pressure	0	1-49	50-75	76-89	>89
Respiration rate	0	1-5	6-9	>29	10-29

RTS = 0,9368 (GCS) + 0,7326 (BPs) + 0,2908 (RR)

Minimal score = 0, corresponds to the survival rate = 2,7%.
Maximal score = 7,8408, corresponds to the survival rate = 99%.
The injured patients with the score less than 4 might be transported immediately with the red marking.

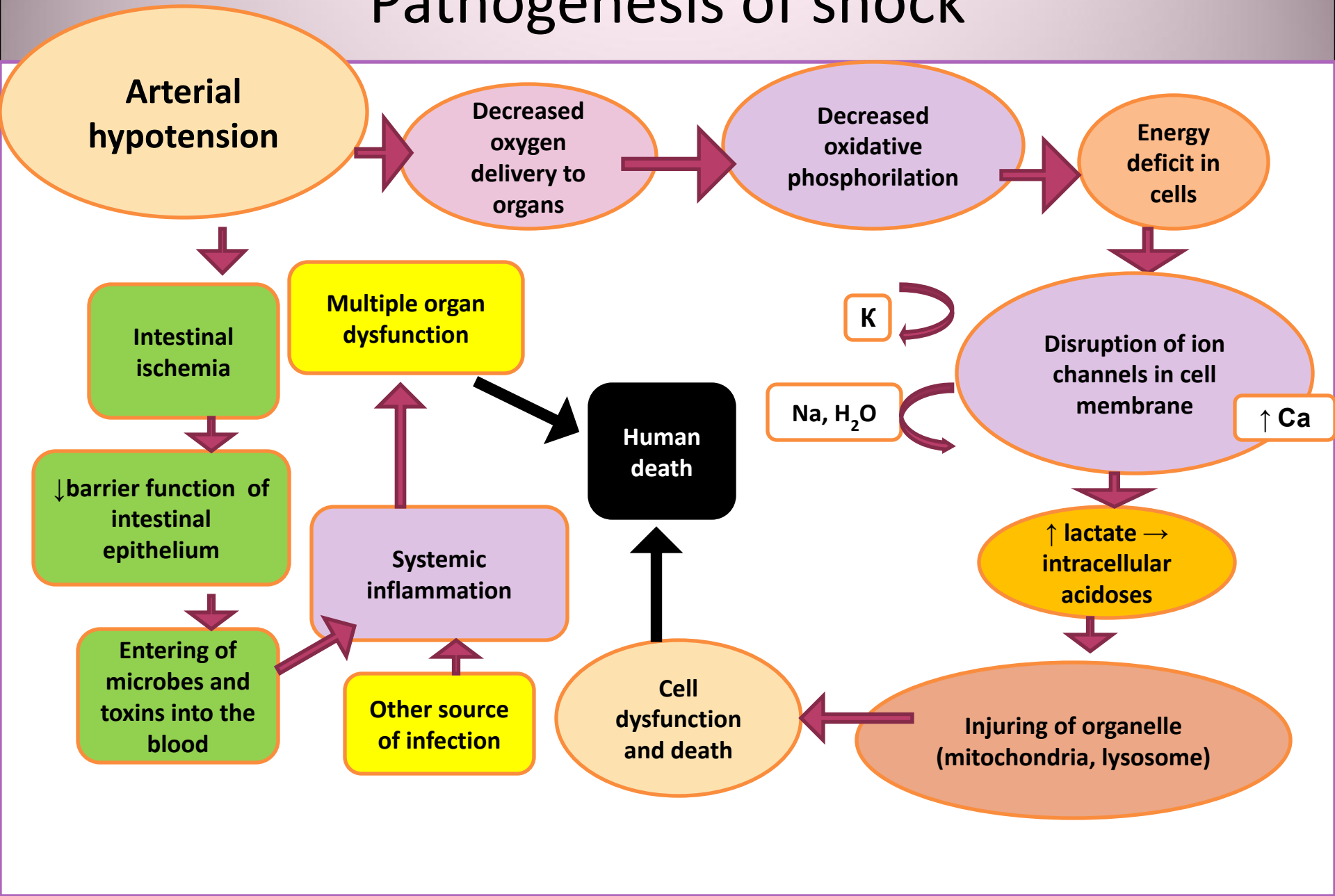
Shock

- Acute hemodynamic instability, which leads to organ dysfunction due to poor perfusion, with poor oxygen delivery and consumption.

Causes of traumatic shock

- Hypovolemia due to bleeding or dehydration in burned patients;
- Cardiac failure due to tension pneumotorax or cardiac tamponage;
- Vasodilatation due to spinal trauma.

Pathogenesis of shock



Clinical signs of shock

- Paleness
- Tachycardia
- Breathlessness
- Oliguria → anuria
- Impairment of consciousness
- Decreased blood pressure
- Poor perfusion (after the pressure to the nail the pink color is normally restored in 2 seconds, if the time of restoring is prolonged, this indicates the poor perfusion)

Stages of shock

- **Compensated**: the perfusion of vital organs (brain, lungs, heart) is maintained due to peripheral vasoconstriction.
- **Decompensated**: the compensatory mechanisms can not maintain adequate perfusion of vital organs. Clinical manifestation of shock.
- **Irreversible multiple organ failure**: massive cell death, multiorgan dysfunction.

Hemodynamic parameters

Parameters	Normal rate
Arterial blood pressure (BPa)	100-140/60-90 mm Hg
Mean arterial blood pressure (BPm) = diastolic BP + 1/3 pulse pressure (BPs – BPd)	70 – 105 mm Hg
Diastolic filling pressure in right atrium	1 – 7 mm Hg
Systolic pressure in right ventricle	15 – 30 mm Hg
Diastolic pressure in right ventricle	0 – 8 mm Hg
Systolic pressure in pulmonary artery	15 – 30 mm Hg
Diastolic pressure in pulmonary artery	4 – 12 mm Hg
Mean pressure in pulmonary artery (MPPA)	9 – 16 mm Hg
Pulmonary capillary closing pressure (PCCP):	
diastolic v-wave	2 – 12 mm Hg
systolic a-wave	3 – 15 mm Hg
Central venous pressure (CVP)	4 – 8 mm H_2O

Integral hemodynamic parameters

Parameters	Normal rate
Cardiac output (CO)	4,0 – 6,2 l/min
Stroke volume, systolic volume (SV)	20-35 ml
Cardiac index (CI) = CO / body surface	2,8 – 3,6 l/min/м^2
Peripheral vascular resistance (PVR) = (MBP-RAP) × 80 / CO	800 – 1500 dyn/S/см$^{-5}$
Index PVR = (MBP – RAP) × 80 / CI	1760 – 2600 dyn/S/см$^{-5}$ /м^2
Pulmonary vascular resistance (PVR) =[MPPA – PCCP] / CO	20 – 120 dyn/S/см$^{-5}$
Arterial oxygen content (C_aO_2) = Hb(g/l) × 1,34 × S_aO_2	
Mixed venous blood oxygen content (in pulmonary artery (C_vO_2) = Hb(g/l) × 1,34 × S_vO_2	
Oxygen delivery (DO_2) = CI × C_aO_2	520 – 720 ml/min
Oxygen consumption (VO_2) = [C_aO_2 - C_vO_2]× CI	110 – 140 ml/min
Oxygen extraction = VO_2 / DO_2	0,22 – 0,30 (22 – 30%)

Receptors of autonomic nervous system

Receptor	Localization	Result of stimulation
α_1	Myocardium Vascular wall	Increasing contractility Vasoconstriction
α_2	CNS Presynaptic membrane	Vasodilatation Decreasing contractility Decreasing pulse rate
β_1	Myocardium	Increasing contractility Increasing pulse rate
β_2	Bronchi Peripheral vessels	Bronchodilatation Vasodilatation
D_1	Renal vessels, Splanchnic vessels	Vasodilatation
D_2	Presynaptic membrane	Vasodilatation (inhibition of norepinephrine release)

Inotropic and vasopressor drugs

Drug	Action	Dose epinephrine
Epinephrine	Endogenic catecholamine, in small doses (0,04-0,1 mcg/kg/min) stimulates β_1 and β_2-receptors, in doses above 0,1 mcg/kg/min - α_1-receptors. Indicated in anaphylactic shock, blocks release of anaphylactic mediators from immune-cells. Side-effects: decreasing of renal perfusion, arrhythmia, hypercatabolism, hyperglycemia, hyperketonemia, miocardial ischemia, hypertension.	From 0,05 mcg/kg/min
Phenylephrine	α_1-adrenomimetic. Usefull for hemodynamic stabilization in patients with taxychardya, miocardial ischemia.	100-180 mcg/kg
Norepinephrine	Endogenic catecholamine, precursor of epinephrine, mediator in sympathetic nervous system. Stimulates α_1 and β_1-adrenoreceptors. In small doses (0,05-3 mcg/min) increases cardiac output and arterial BP, in doses above 4 mcg/min increases peripheral vascular resistance/ side-effects: tachycardia, increasing myocardial excitability, decreasing renal perfusion, oliguria. Indicated in patients with decreased peripheral vascular pressure: neurogenic shock (spinal trauma), septic shock, in tricyclic antidepressant poisoning.	Start from 0,05, to 24 mcg/min.

Inotropic and vasopressor drugs

Drug	Action	Dose
Dobutamine (dobutraxe)	Synthetic catecholamine, inotropic drug. Acts predominately on β_1 and less on β_2-receptors. Increases myocardial contractility, have minimal chronotropic effect. Indicated in cardiogenic shock.	2,5 – 10 mcg/kg/min
Dopamine (dophamine)	Endogenic catecholamine, precursor of norepinephrine, neurotransmitter. In small doses (0,5-3 mcg/kg/min) activates D_1-receptors (increasing renal perfusion), in mean doses (3 -7,5 mcg/kg/min) – stimulates β_1 and β_2-receptors (inotropic effect, increases oxygen requirement in myocardium), in high doses (>10 mcg/kg/min) - stimulates α_1-receptors (increasing peripheral vascular resistance).	2,5 – 5 mcg/kg/min
Dopexamine	New synthetic cathecholamine, stimulates more β_2-receptors, less β_1- receptors. Increases cardiac output without increasing oxygen requirement of myocardium, increases renal perfusion, splanchnic and peripheral perfusion. Very expensive drug.	0,5 – 6 mcg/min.

Inotropic and vasopressor drugs

Drug	Action	Dose
Isoproterenol	Stimulates β_1 and β_2-adrenoreceptors. Increases pulse rate, myocardial contractility, cardiac output, oxygen requirement in myocardium. Decreases peripheral vascular resistance. Used in patients with bradyarrythmya with shock, before pacing (implanting of cardiac pacemaker).	1 – 10 mcg/min
Amrinone, milrenone, enoxymone	Inhibitors of phosphodiaestherase. Markedly increase myocardial contractility, cause vasodilatation, bronchodilatation. No influence on pulse rate.	Amrinone 0,75 – 1,5 mg/kg.
Digitalis	Positive inotropic effect due to c-AMP-dependent increase of intracellular calcium via the action on Na-K and Ca-channels of membrane of myocardiocytes. Indicated in chronic cardiac failure, tachyarrhythmia. Limited use in shock.	

Reserve inotropic drugs

Drug	Action	Dos
Glucagon	Intravenous administration cause positive inotropic effect, improves atria-ventricular conductivity without stimulation of β_1-adrenoreceptors. Used for increasing BP in severe acidosis, β-adrenoblokers overdosing, in ineffective vasopressor therapy. Side-effects: nausea, vomiting, hyperkaliemia, hyperglycemia.	At the beginni 1 – 5 m then infusion 20 mg/hou
Vasopressine (ADG)	Potentiates the effect of norepinephrine, decreases the production of NO-synthetase and activity of ptoteinkynase C. Used lately in cases of cathecholamine-resistent septic shock: increases systolic BP, CI, CO, decreases the dose of norepinephrine. Side-effect: poor gastric and intestinal mucous perfusion.	0,01 – 0 unit/mi
Levosymendan	Used in acute cardiac failure. Increases cardiac output without increasing the oxygen requirement in myocardium. Cause vasodilatation. On the stage of clinical trial.	

Vasodilatative drugs

Drug	Action	Dose
Nitroglycerine	Organic nitrate. Relaxes smooth muscles of vascular walls due to increasing of NO production in endothelium. Cause generalized vasodilatation in pulmonary and systemic circulation. In doses up to 40 mcg/min predominantly cause venodilatative effect, in doses abive 200 mcg/min cause arteriodilatative effect. Decreases CVP, pressure in pulmonary artery without changing CO. in high doses decreases BP and CO. has antiaggregant effect. Side-effects: increased brain perfusion, intra-cranial pressure, pulmonary perfusion, intrapulmonary shunt (contraindicated in respiratory distress syndrom), methemoglobinemia.	start with 5 mcg/min, increasing of necessity
Sodium nytroprusside	vasodilator, donator of NO. effectively decreases BP, but can cause the steal syndrom (brain and myocardial ischemia).	

Intravenous fluid administration

Fluid	Advantages	Negatives
Colloids: albumin, dextrans, gelatins, hydroxyaethylstarch (HAES)	Less volume needed Prolonged volemic effect Less peripheral edema	More expensive Coagulopathy (dextrans >HAES) Pulmonary edema (in cases of capillary hyperpermeability) Descending of glomerular filtration Allergy
Crystalloids: saline solutions, dextrose	Less expensive Enhanced renal perfusion Interstitial volume replacement	Short volemic effect Peripheral edema (hypoalbuminemia) Pulmonary edema (hypoalbuminemia + high pulmonary capillary pressure)

Cristalloides

- Normal saline (isotonic 0,9% NaCl).
- Balanced electrolyte solutions: Ringer‘s lactate, sterofundyne, e.g.
- Hypertonic NaCl (from 1,6% to 10%).
- Dextrose 5-10%, is not recommended in brain edema, increases intracellular volume.

Synthetic colloides

- Dextrans: Poliglucine, Reopoliglucine
- Gelatins: Helafusine, e.g.
- Hydroxyaethylstarchs (HAES): Haecodez, Refortan, Stabizol, Voluven, Tetrastarch, e.g.

Combination: HAES + hypertonic NaCl:

for “small volume reanimation” – HyperHAES

Polyhydric alcohol solutions: Sorbilact, Reosorbilact, Xylite

- Crystalloid solutions.
- Volemic effect is short.
- Advantages:

1. Energy supply without insulin - useful in patients with intolerance to glucose.
2. Fast volemic effect due to hyperosmolarity.

- Side-effects: vascular oxalose in brain, liver and kidney → acute renal, hepatic failure. Therefore not used in USA, Europe, Australia.
- Unfortunately, in Ukraine these solutions are used widely.

Polivinylpirrolidones: peristoy, haemodez

- The first synthetic colloides.
- Repeated administration can lead to depression of reticule-endothelial system with severe immune depression. This side-effect is dangerous particularly in neonates and young children.
- Blastogenic effect.
- Accumulate in interstitial space leading to edema.
- Deposit toxins in liver, kidneys, spleen, lungs, bone marrow.
- Restrained for medical use in USA from 1958, in Ukraine from 1998, in Russia – from 2005.

Perfluorocarbons: Perftoran («blue blood»).

- The positive effects were exaggerated and did not confirmed in medical practice.
- Unfortunately, it is still in use in Ukraine.

Blood preparations

- Whole blood
- Packed red blood cells
- Fresh frozen plasma (FFP)
- Cryoprecipitate
- Platelets
- Albumin

Hemorrhagic shock

- Cause – blood loss.
- Hemodynamic changes:

Decreased volume of blood circulation, central venous pressure (CVP), cardiac index (CI), pulmonary capillary closing pressure (PCCP)

Increased peripheral vascular resistance (PVR)

Response to the blood loss

- Sympathetic activation: centralization of blood circulation, discharge of blood from spleen, liver, lungs, tachycardia, increased oxygen demand in myocardium.
- Stimulation of renin-angiotensin-aldosterone system: renal vasospasm, increased renal Na reabsorbtion → oliguria.
- Stimulation of vasopressin synthesis: increased renal water reabsorbtion → oliguria.
- Stimulation of osmoreceptors in hypothalamus: thirst.
- Stimulation of glucocorticoid production: increased sensibility of adrenoreceptors, membrane stabilization?
- Autohemodilution.
- Stimulation of renal erythropoietin production (in the late stage of shock).

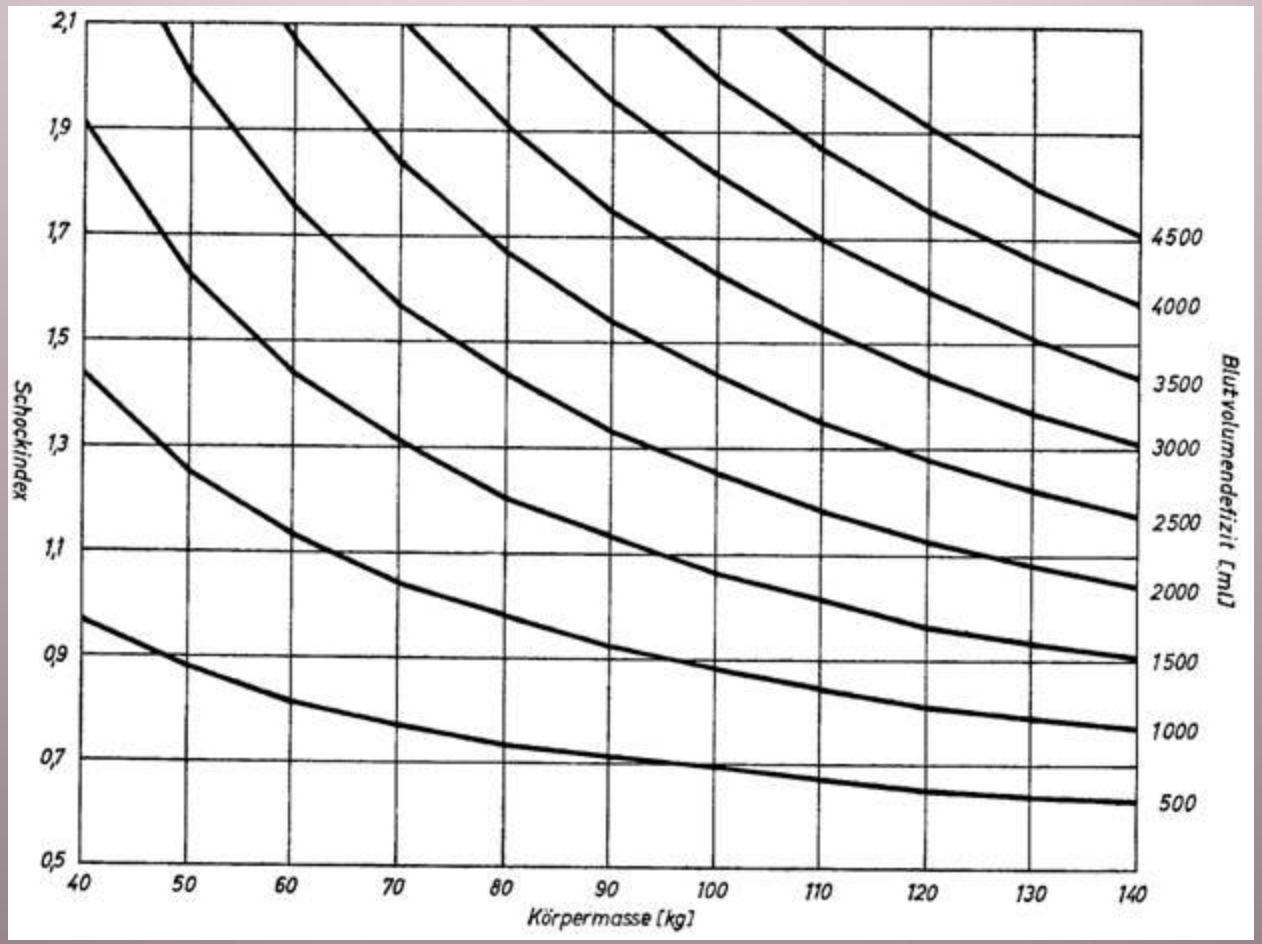
Classification of blood loss severity (American Association of Surgeons)

Parameters	Classes			
	I	II	III	IV
Volume of blood loss, % circulating blood volume	< 15	15 – 30	30 – 40	> 40
Pulse rate, bits/min.	< 100	>100	> 120	> 140
Blood pressure in horizontal position	Normal	Normal	Decreased	Extremely decreased
Diuresis, ml/h	> 30	20 – 30	5 – 15	< 5
Conscious level	Anxiety	Euphoria, excitation	Sopor	Coma
Replacement	Cristalloides	cristalloides, colloides, red blood cells	cristalloides, colloides, red blood cells	cristalloides colloides, red blood cells

At the prehospital stage Algover‘s shock index is useful for evaluating the volume of blood loss:
Shock index = Pulse rate / Systolic BP,
in normal 60 : 120 = 0,5.

Severity of shock	Shock index	Volume of blood loss
Shock I	0,8 – 1	< 1 liter
Shock II	1 – 2	1 – 2 liter
Shock III	> 2	2 – 3 liter
Shock IV	BP and pulse are undetectable	> 3 liter

Nomogram for calculating the blood volume deficit (Blutvolumendefizit [mL] – right vertical axis) according to the Algover's index (Schockindex – left vertical axis) and body mass (Korpermasse [kg], inferior horisontal axis)



Shock index:

- Is not informative in young children and old patients due to the age differences of hemodynamic parameters.
- Blood loss volume you can evaluate visually in cases of external bleeding.
- In cases of internal blood loss (intraabdominal, intrathoracic, interstitial bleeding) the shock index is usefull.

Hemorrhagic shock

Laboratory dates:

- Hb ↓.
- Ht ↓.
- Lactate ↑.
- Diuresis ↓.
- pH ↓.

Prehospital management of hemorrhagic shock

- Venous access – canulation of 2-3 veins;
- Crystalloid / colloid infusion;
- Oxygen inhalation;
- Analgesia if indicated (prefer non-narcotics);
- If systolic BP is < 90 mm Hg in spite of rapid infusion – begin vasopressors (norepinephrine or dobutamine;
- Transportation to the hospital in horizontal Fowler position with raised legs;
- Stop external bleeding (tourniquet, raised position of damaged extremity, pressing apply to the wound);
- Transport immobilization;
- Warming the patient (take off the wet clothes, cover with a blanket).

Infusion rate in continuous bleeding

- If the bleeding is not stopped the infusion rate must provide the minimally sufficient hemodynamic parameters (systolic BP = 80-90 mm Hg), but not the maximal replacement of blood loss.
- Such rule of reason decreases blood loss and accelerates the thrombi formation in the affected area.
- It helps to avoid the dilution syndrome with excessive bleeding.

War – epidemic trauma

- In the II word war all countries use the blood transfusion. But it doesn't survive the soldiers in many cases of traumatic shock.
- The experiments by C. Wiggers explain it: the replacement of blood loss with equivalent volume of blood transfusion in dogs did not survive them.
- In the late 1940-th in such experiments it was demonstrated that the replacement of blood loss with the twofold normal saline infusion survives dogs without the blood transfusion.

Change of field doctrine

- On the basis of understanding the pathophisiology of traumatic shock as the fluid sectors' shift and the significance of plasma volume deficit at the first the field doctrine was changed.
- In the Korean war for the first stage replacement of blood loss the synthetic colloid Macrodex (the soviet analogue – Poliglucine) was used widely. But it's maximal dose is restricted and the damages of lung and kidney were found in many cases.
- In the Vietnam war the rapid Ringer-lactate infusion was provided for the American wounded soldiers in the battlefield. The three vena infusion was continued during transportation on the helicopter to the hospital and to the stopping bleeding in the operating room. Then the blood transfusion began. The excess of fluid was eliminated with diuretics.
- This "liberal" regimen was provided also in the civil medicine: on the preoperative stage the 3-5-hold crystalloid saline volume to the blood loss was infused).

Change of doctrine

- The “liberal” regiment of crystalloid infusion gave rise to doubt in early 1990-th.
- The dilution of blood leads to coagulopathy with increasing blood loss.
- So there was provided “restrictive” infusion regiment and “permissible hypotension” for the prehospital stage to surgical hemostasis.
- Acquired immunodeficiency syndrome (AIDS) pandemia and detection of transmissive infections lead to the practice of early fractionating of donor blood. All these result to the fresh whole blood deficit in hospitals.
- So at the turn of the XX and XXI century the next staging was recommended in the protocols of blood loss replacement in many countries (including Ukraine).

Step-by-step in replacement of blood loss

- At the first stage: **saline cristalloids** in restrictive regiment (for detectable pulse on the radial artery. Advantages: replacement of all fluid sectors, enhanced renal perfusion. Side-effects: edema, dangerous in lungs and brain.
- At the second stage: **synthetic colloids** (Geleains, hydroxy-ethyl-starch - Tetraspan). Advantages: better volemic effect comparing to crystalloids. Side-effects: coagulopathy, neurotoxicity.
- At the third stage: transfusion of **packed red blood cells (PRBC)**, that requires some time for detection of blood group and rhesus and compatibility. Indicated in: Hb <70g/l and Ht<21%.
- At the forth stage: **fresh frozen plasma (FFP).** In acute massive blood loss the ratio of FFP to CRBC should be 1 : 1.
- At the fifth stage: **platelets** if the plates < 50×10^9/l.
- **Fibrinogen** (3-4 g) or **cryoprecipitate** (50 mg/kg body mass) are recommended in hypofibrinogenemia.

“Damage control”

- In XX century wars (in Persian Gulf 1990-1991 and in Vietnam 1961-1973) 24% of wounded American soldiers died.
- In XXI century wars (in Iraq and Afghanistan 2003-2009) only 10% of wounded American soldiers died.
- It was due to the change of medical strategy – american-british innovation called “damage control”.

“Damage control”

- For surgeons it means the physiological correction, but not anatomical correction. This includes stop bleeding, prevention of infection and further traumatic damage (immobility).
- For anesthesiologists it means anesthesia for this stage of surgery, “permissible hypotension” before stopping bleeding, “haemostatic resuscitation” and prevention of “lethal triad”: coagulopathy, acidosis and hypothermia.

Complete surgical correction

- Complete surgical correction may be provided in 1-2 days after the stabilization of the patients physical and metabolic state.


Thank you for your attention!
Questions?

THE PLAN

of **lectures** in surgery of extreme conditions and military surgery for the English-speaking medical students of IV course of stomatology faculty (autumn semester of 2012/2013 st. year) Lecture day: MONDAY.

Auditorium: The main 9-floor-building of Kharkiv Regional Hospital,

№	Date	Theme	Time	Lecturer
1	03.XII (December)	The traumatic shock. The prehospital management. The blood replacement in trauma patients.	9.20 – 11.00	Prof. Fesenko
2	10.XII (December)	Cranial and spinal trauma. Damage of the thorax. Pneumothorax, hemothorax, cardiac tamponade. The management of patients with multiple trauma. Burns. Crash-syndrom.	9.20 – 11.00	Prof. Fesenko

THE PLAN

of **practical classes** in surgery of extreme conditions and military surgery for the English-speaking medical students of IV course stomatology faculty (autumn semester of 2012/2013 st. year)

Auditorium: The main 9-floor-building of Kharkiv Regional Hospital, The 7-th floor. (Trinkler str.)

Teacher – prof. U.A. Fesenko

№	Themes	Hours
1.	Cardio-pulmonary resuscitation. Grading and evacuation of patients in catastrophe. Battle surgical trauma. The surgical treatment of gunshot wound.	5
2.	The traumatic shock. The blood replacement in trauma patients. Neurotrauma, damage of head, maxillofacial region and neck. The management of burned patients.	5
3.	Damage of the thorax. Pneumothorax, hemothorax, cardiac tamponade. Damage of the abdomen and pelvis, bones and joints. The management of patients with multiple trauma. Crash-syndrom. **Final test.**	6

№ of group	Date	Time	Teacher	№ of theme
1	25.12. 2012	12.25 – 16.45	Fesenko	1
	3.01. 2013	12.25 – 16.45		2
	11.01. 2013	12.25 – 17.35		3
2	26.12. 2012	12.25 – 16.45	Fesenko	1
	4.01. 2013	12.25 – 16.45		2
	14.01. 2013	12.25 – 17.35		3
3	27.12. 2012	12.25 – 16.45	Fesenko	1
	8.01. 2013	12.25 – 16.45		2
	15.01. 2013	12.25 – 17.35		3
4	28.12. 2012	12.25 – 16.45	Fesenko	1
	9.01. 2013	12.25 – 16.45		2
	16.01. 2013	12.25 – 17.35		3
5	31.12. 2012	12.25 – 16.45	Fesenko	1
	10.01. 2013	12.25 – 16.45		2
	17.01. 2013	12.25 – 17.35		3