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SHOCK – Complications of shock and reanimation

Durić A.
Clinical Centre of Sarajevo University, Clinic for anaesthesiology and reanimation, Sarajevo

Abstract
Development of complications is possible in patients that survive severe shock of any origin as a consequence of pathophysiological events in shock syndrome by itself as well as techniques of aggressive reanimation required in current situation.
Complications are manifested as failure in functioning of organ and organic systems and its sequels. Although every organ may be damaged, the highest attention is focused on brain, heart, lung and kidney protection. The severest complication of hypovolemic shock is brain damage but most common one is renal function impairment. Non-invasive, even more precise standard replaces former standard of invasive monitoring in shock parameters estimation.
Indirect sign of GIT mucosa, sublingual capnography is in trend as well as impedance monitoring based on measurement of electrical resistance changes in chest. Resistance is proportional to aortic pulse wave strength related to systole and diastole.
Key words: shock, pathophysiology, reanimation, complications, monitoring

Introduction

Despite of modern courses of treatment and modern monitoring technology in shock syndrome, controversies still exist in definition of shock, therapeutically courses and decisions of reanimatologist: when to treat with fluid, which fluid and how much. This article due to limited space will not consider these questions.

Nevertheless to different causes of shock, common pathophysiological base is disturbance of sufficient perfusion and oxygen balance. Normal haemodynamical parameters, measured with standard indicators, do not include adequate microcirculation and oxygen balance maintenance in all organs. Hence, normovolemia with impaired distribution in pain and stress as well as persistent bleeding with normal blood pressure after vasoconstriction and cateholamine response introduce some organs in hypoperfusion and hypoxia with multiorgan dysfunction occurrence (MOF) and systemic body inflammatory response (SIRS). (1,2)

Pathophysiology

Changes in microcirculation are response to actual circulatory volume loss leaded to vasoconstriction, increase in vascular resistance, and redirection of circulatory volume from non-vital organic systems to vital organs.

Together with increased heart rate influenced with aortic arch, atria sensors and vasoconstriction, multisystemic humoral and catecholamine response occurs.

On smooth muscle of blood vessels, alpha 1 receptors mediate vasoconstriction influenced with vasoconstrictors: noradrenalin, angiotensine II, vasopressin, endotheline, thrombexane II, prostaglandine PGI2, nitric oxyde (NO), adenosine (local metabolic product). (3)

Balance between these vasoconstrictors and vasodilators determine local perfusion. Disturbance of microcirculation as a common response in late phase of shock whichever, results in cellular metabolic disturbances and organic dysfunction as a specific reaction of particular organs. Normal response to mild or moderate hypovolemia is attempt of intravascular volume restoration by hydrostatic pressure and osmolarity changes. (1)

Intravascular oncotic pressure remains constant or increase as a consequent to Starling law about fluid exchange; overall effect is fluid reapsorption in vascular pool.

Metabolic changes (hyperglicemia, glicolysis, lipolysis) increase extra cellular osmolarity, lead to osmolar gradient between cells and interstitial which increase interstitial volume and intravascular volume on account of intracellular volume.

With advancing of shock, vasodilator moments prevail over vasomotor tonus lead to hypoperfusion and hypotension.

Cells membrane dysfunction is common path in last phase of pathophysiological events in different types of shock.

Normal cellular transmembranic potential decrease, intracellular sodium and water increases, lead to cells oedema with further disturbance in microvascular perfusion.

Oxygen consumption in shock

Oxygen transport depends of:

- Microcirculation
- Capillary permeability
- Oxygen diffusion, CO2, nutritients, metabolic products
- Exchange of cells metabolic products

Inability of organism to provide adequate oxygen supply (DO2) and oxygen consumption (VO2) correlate to mortality. (1)

In resting person, oxygen needs VO2 is 100-160 ml/min/m2, remain constant over wide range of oxygen supply (normal values of DO2 are 500-720 ml/min/m2.) Tissues uptake is only 20-25% of provided oxygen, what is called oxygen extraction reserve.

Normally, there is large tissue potential for additional blood oxygen extraction if this is necessary. (1,4) Trauma and shock lead to increase in oxygen consumption, but this mechanism acts only while oxygen supply is more than 300 ml/min/m2. Below this level, tissues cannot increase oxygen extraction so further extraction is maximal. Therefore, oxygen consumption decreases because it's directly dependent on level of oxygen supply. (1,5)

Severe shock and complications of reanimation

In patients survived severe traumatic hypovolemic shock or septic shock, different organic dysfunctions may develop with consequent sequels.

Some of complications could be consequence of direct ischemia or caused by reanimation technique. In severe injured patients leading cause of death is multiple organic failure occurred in no more than 10% of cases with trauma score of 20. (6)

Reanimation require large amounts of crystalloids, colloids and blood, lead to fluid overload and electrolytic disbalance, pulmonary insufficiency and coagulopathy.
The severest complication of hypovolemic shock is brain damage and most common is renal function impairment.

When circulation is re-established after prolonged state of shock further problems are reperfusion and reoxygenation damage. (7)

Although every organ can be damaged in shock, the highest attention is directed to brain, heart, lung and kidney protection; if these vital organs are undamaged, other organs rarely suffer from dysfunction.

Haemorrhagic shock is tolerated on different way, depending of previous physiological status and patients age.

Very young and very old patients are prone to develop early decompensation after loss of circulatory volume.

Drugs and alcoholic intoxication could modify physiological response on trauma. (8)
Together with adequate fluid reanimation, early surgery is the most efficient in preventing of haemorrhagic shock consequences.

Early fluid reanimation decreases multiorganic failure.

In haemorrhagic shock, blood is to be given simultaneously to surgical haemostasis.

Damage of CNS

Previously egzisting cerebrovascular diseases influence on brain tissue reaction and shock by potentiating cerebral circulation changes and increase of vascular resistence.

Secondary changes of brain tissue are caused by cascade of biochemical changes begineing with hypoxia and resulting in interruption of normal balance between oxygen supply and consumption. (9) Excitatory amino-acid glutamate and decrease in ATP level follow cells ischemia.

Glutamate activate N-methyl D aspartate, propionate (AMPA) and metabolic receptors which activation results in cells ion pump disturbance with fast calcium and sodium inlet into cells. Several worsening effect are related to calcium, including vasospasm, mitochondrial dysfunction, protease and lipase activation. As an effect, lipase activation promote synthesis of arachidonic acid derivates-thromexane A2 (TxA2), prostaglandine PGI2 and leukotrienes. Derivates of this acid lead to platelets aggregation, vasodilation and free pxygen radicals synthesis in lipid peroxydation process. Loss of cell integrity after ion changes could result in cytotoxic oedema and vasogenic oedema due to cerebral capillary network. The severest complication of cerebral oedema is increased intracranial pressure which disturb autoregulation of brain circulation.

Secundary brain injuries could be prevented or limited by optimisation of cerebral reanimation.
Consensus of decisions for therapeutical guidelines for secondary brain injuries prevention in haemorrhagic shock and craniocerebral structures injury:

- maintenance of arterial pressure in refferal limits
- maintenance of cerebral perfusion
- mechanical ventilation in case of hypoxemic respiratory insufficiency caused by consciuous disorders, focal pulmonary contusions, pulmonary oedema
- oxygen saturation should exceed 92% and pO2 above 60 mm

- maintenance of circulatory volume with Hct around 30% and isotonic saline (colloids and hypertonic saline of 3% or 7.5% showed no significant advantages in reanimation or neurological outcome)
- hypotonic solutions sholud not be used in initial treatment due to possible sodium elevation and hypoosmolarity which could exacerbate oedema
- aim of treatment is maintenance of normovolemia without decrease in plasma osmolarity. In case that fluid tretment fail to establish mean AP, inotropic drugs and vasodilators could be applied (dopamine, fenilefrine, noradrenaline)
- maintenance of glicaemia in range 4-7 mmol/l
- prevention of hyperpyrexia (metabolic increase to 13%) by frictions or acetaminophen 650 mg every 4 hours
- pain, anxiety, cough, patients discordance with respiratory device, influence on oxygen consumption and intracranial pressure (analgetics, sedatives, relaxant drugs)
- specific treatment, drainage, hyperventilation, manitol, barbiturates, prophylaxis of epi seizures

Cardiac complications

Heart failure is rare in thraumatic shock, unless previous diseases exist. In multiple organ failure most common is combined respiratory and cardiac dysfunction. Heart failure could be caused by many factors during haemorrhage and reanimation. (10)

Elevated potassium, decreased calcium, hypophosphatemia and hypomagnesiemia could influence on cardiac contractility and rhythm. Prolonged shock enable platelets aggregation in coronary microcirculation, contributing myocardial ischemia and dysfunction. Reanimation itself, especially with blood derivates, could cause myocardial damage independent of haemorrhagic grade (11).

Severe brain damage could contribute myocardial depression and ireversibile shock. Mild hypoxia increase symphatetic tone and cardiac contractility while severe hypoxemia impair myocardial function same as severe acidosis do. Post haemorragic heart failure was attributed to «myocardial depression factors» with probable origin in pancreas and splanhic circulation in response to ischemia (12).
Vasopressors usually increase systemic vascular resistence by cardiac output decreasing, tissue perfusion and oxygen supply.(13)

If hypotension and bradicardia persist after adequate fluid supply, myocardial infarction is suspected, especially in diabetic patients and those with previous coronary disease.

Acute renal failure

Kidney flow is decreased for approx. 25% of cardiac outpuIn in response to haemorrhage and hypotension as well as in attempt to preserve effective volume.

Released renin influence synthesys of angiotensin II, potent vasoconstrictor. Increased osmolarity influence on ADH secretion.

Loss of volume for more than 15% surpasses kidney autoregulatory capacity and renal flow continue to decrease and being redistributed with disturbance of sodium and water retention.

Early renal insufficiency is often followed by tubular necrosis and myoglobinuria. They correlate with hypotension and myoglobinuria while late insufficiency correlate with development of sepsis and drugs intoxication (6,14).

Ventilatory support and general anesthesia could impair renal mechanisms of autoregulation together with NSAID usage which worsen vasoconstriction.

Prophylaxis and therapy of renal complication is based on diuresis maintenance by volume reconstitution, application of Henle loop diuretics, manitol and bicarbonates application.

Hyperpotassemia and enormus insufficiency require standard treatment, if that failes dialysis if needed.

Renal vasoconstriction is ceased after adequate reanimation but oliguria could persists after volume normalisation. In that situation diuretics sholud not be applied. Some authors used dopamine in small doses but numerous trials did not confirm that benefit (15).

Dialysis is indicated in overcoming of uremia and oliguria. Hence, incidence of renal failure with dialysis and adequate reanimation was decreased to less than 5%, according to references, even in severe trauma (16).

Pulmonary complications

Pulmonary insufficieny within MOF, in traumatic hypovolemic shock (late reanimation, patients older than 55) occures in 10% cases according to statistics (9).

Although shock, by itself, coud cause lung tissue ischemia with impared cilliar and surfactant activity, measure of extravascular water in lung tissue doesnt correlate with shock but with lung contusion and sepsis (17).

Over sufficient hydration could worsen ARDS.

Pathophysiology include inflammatory supstrate, arachidonic acid metabolites, oxygen radicals, increase in endothelial permeability of pulmonary capillars, leucocyte dyapedesys in capillars, endothelial oedema, thrombotic occlusions, C5 and C5a complement activation.

Liver

Liver disfunction is quite offten after traumatic and haemorrhagic shock, caused by hypotension and hypoxia with hepatocyte mitochondrial oedema, venous congestion, centrolobular necrosis and jaundince. Clinical fatures of liver dysfunction after hypovolemic hypotension are: alcaline phosphatase elevation for 2-3 times, SGOT elevation to 100-500 IU, jaundince, hepatomegaly, possible acholic stool also with alcaline pohosphatase and SGOT elevation.

Jaundince could also appear due to haemolytic reaction, bet rarely without liver dysfunction. Intravascular hemolysis is followed with occurence of free blood and urine hemoglobine and conjugate bilirubin elevation. Reanimation with massive doses of crystalloid solutions and transfusions could contribute to liver cells oedema (18).

Therapy is reanimation, volume supply, adequate flow and metabolism, hypernutrition with minimised proteine and proper selection of non hepatotoxic medicaments.

Mesenterial ischemia

Non occlusive mesenterial ischemia is caused by low flow which is the result of traumatic hypovolemic hypotensia.

Splanhic region has high alpha-adrenergic activity and its highly sensitive on hypoxia.

Integrity of gastrointestinal mucosa is compromitted. Splanhic organs could show refraction after persistens of arterial pressure on 45 mmHg for 30 minutes on fluid applied. The pH on gastric mucosa is significantly decreased during the shock, temporarely recovered after reanimation, but then followed with progressive

worsening (studied models of haemorrhagic shock). Values of MAP, pH and pCO2 imply hypoperfusion or reperfusion damage inspite of reconstituted normovolemia.

Inadequate or tardy reanimation cause vasoconstriction as a compensatory mechanism for purpose of vital organs perfusion.

This situation is worsen with cardiac diseases and application of inotropic medication.

Clinical manifestations of stress ulcers, perforation, colitis and septic shock are consequence of mucosal ischemia in gastrointestinal tract.

Haemorrhage from duodenal stress ulcer is usually late manifestation after hypovolemic hypoperfusion. Vasodilators, together with other reanimation procedures could be applied in treatment.

Coagulopathy

Drop in plasma coagulation factors was noticed immediate after haemorrhage. It was attributed to consumption and plasma dilution with fluids. Massive blood transfusions, colloids and crystal solutions could lead to so called dilution coagulopathy, lately compensated with increased liver synthesis and adequate supply. (19)

Hypothermia and acidosis contribute coagulopathy when massive transfusions are needed.

In general, more than 10 units of erythrocytes result in thrombopenia, decreased fibrinogen, prolonged prothrombic and PT time while more than 20 units result in coagulation defects in 70% of patients (20).

Disseminated intravascular coagulation usually follows multi-organ failure and SIRS.

Monitoring

Promptly recognized tissue hypo perfusion is basic in shock assessment. This cannot be recognised from usually assessed parameters: pulse, pressure, skin colour and diuresis.

Systemic pressure is normal, although patient could lose more than 500 ml of blood with some organs already hypo perfuse.

Skin colour often reflects skin and muscle circulation. They display hypo perfusion slowly (on 30% of blood loss skin is cold and marmoreal).

Comorbidity could also change significance of these parameters.

Techniques of invasive monitoring with venous system catheterisation, right heart or pulmonary arterial is still widely used. However, indications for invasive monitoring have been narrowed through occurrence of safe non-invasive techniques. (21)

Against prejudice of some clinicians, there is no direct relation between the stage of invasive techniques and precision of cardiac output (CO) measurement. PA catheter placement is complicated and invasive procedure in ICU and its been used for intracardial pressure measurement and CO from termodilution curve, followed with false measurement risk. Catheter and transducers must be adequately placed and calibrated. Pulmonary capillary pressure must be determined correctly and pulse frequency must be stabile. To obtain adequate parameters, patient must not receive inotropic and chronotropic drugs. (22)

Trans-oesophageal echocardiograph (TEE) is useful for structural and functional heart evaluation.
Modern technologies measure tissue perfusion by measurement of systemic oxygenation SO2. Adequate oxygenation is achieved with balance between DO2 supply and oxygen consumption VO2. DO2 is dependent of cardiac output, haemoglobin level, PaO2. VO2 is dependent of factors influence on energy requires: activity, pain, temperature and infection.

In normal circumstances, DO2 increase and decrease to meet oxygen needs of tissues and to preserve aerobic metabolism.

When DO2 is decreased or metabolism is increased, that subtle balance is interrupted, organism is switched to anaerobic cellular metabolism. Hence, integrated non-invasive monitoring identify early cardiac, pulmonary as well as other tissue perfusions and tissue peripheral saturation in general and allows real time therapeutically dosage.

Detection and measurement of sublingual CO2
Gastrointestinal mucosa is very sensitive on hypoperfusion and hypoxia, and it reacts on changes by increasing eliminated CO2 in whole gastrointestinal mucosa.

Similar neural pathways control vascular network in tongue and gastrointestinal tract. By measurement of CO2 partial pressure in gastrointestinal mucosa, evaluation of systemic tissue perfusion could be evaluated indirectly. This parameter is useful for clinicians in estimation of shock progression, which sometimes cannot be early detected with standard parameters: pressure, pulse, diuresis regarding compensatory catecholamine mechanisms.

Since tongue is the most proximal part of gastrointestinal tract, pCO2 measurement can be achieved with sublingual probe, small handy equipment. Studies have shown that is useful parameters in evaluation of peripheral blood supply. (23)

Principle of measurement with probe and fiber-optical technology is that only CO2 diffuse through semipermeabile probe then dissolute forming carbonic acid, changeing pH of solution. Fluorescent contrast intensity in solution is proportional to pH value.

Technique of bioelectrical impedance
The most impressive advance in non-invasive monitoring is measurement of thoracic bio-impedance.
Impedance curve is product of blood flow variations and volume in ascending aorta.

Impedance cardiograph

Provide haemodinamic data continually on simple way in every environment. (24)

It's functioning by measurement of thoracic electrical resistance changes during cardiac cycle, while blood mass in aorta increase and decrease.

Continual resistance measurement enables measurement, calculations and continual monitoring of whole cardiac cycle, including heart rate, cardiac output, parameters of contractility and thoracic fluid quantification. Two pairs of probes are placed on patient's neck and chest then connected on IQ monitor besides ECG monitoring. Changes in resistance occur in systole during the blood ejection from left chamber, with changes in thoracic aorta. IQ identifies initiation of electromechanical systole, aortic valve opening (B), maximal ventricular contraction (max curve B to C) and aortic valve closure (X).

IQ technology represents diagram of force, time and frequencies of impedance changes, forming three-dimensional form of cardiac function. Three-dimensional analysis discovers crucial information, previously hidden in traditional two-dimensional form. IQ analyse curve, measure cardiac parameters precisely, also with precise haemodinamical data without risk of invasive procedures, providing

clinicians with analysis of more patients data and also to evaluate more precisely overall circulatory function.

Simple functioning of IQ is provided by display showing ECG and thoracic resistance signals simultaneously. Clinicians could simply estimate whole spectrum of haemodinamical parameters and they trends including: left chamber performance (cardiac output, cardiac index, heart rate and frequency), myocardial contractility, pulmonary status and after load.

Apstrakt

ŠOK- komplikacije šoka i reanimacija

Pacijenata koji prežive stanje teškog šoka bilo koje etiologije moguć je razvoj komplikacija kao posljedica patofizioloških zbivanja samog sindroma šoka, a i kao posljedica tehnika agresivne reanimacije

kakvu zahtijeva postojeće stanje.

Komplikacije se manifestuju kao disfunkcija organa i organskih sistema sa posljedičnim sekvelama.Iako svaki organ može biti oštećen,najveća pažnja u toku liječenja usmjerena je na zaštitu mozga,srca,pluća i bubrega.Najteža komplikacija hipovolemičnog šoka je oštećenje mozga, a najčešća oštećenje renalne funkcije.Dosadašnji standardni invazivni monitoring u objektivizaciji procjene parametara šoka zamjenjuje neinvazivni koji čak može biti i precizniji.

U trendu su sublingvalna kapnografija kao indirektni pokazatelj perfuzije sluznice GIT-a i impedanca monitoring koji funkcioniše po principu mjerenja promjene električnog otpora u toraksu,a koji je proporcionalan jačini pulsnog talasa u aorti vezano za sistolu i dijastolu.

Ključne riječi: šok,patofiziologija,reanimacija,komplikacije,monitoring

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