RESUSCITATION OF A POLYTRAUMATIZED PATIENT WITH LARGE VOLUME CRYSTALLOID-COLLOID INFUSIONS – CORRELATION BETWEEN GLOBAL AND REGIONAL HEMODYNAMICS: CASE REPORT

Mirjana Lončarić-Katušin[1], Matija Belavić[1], Josip Žunić[1], Snježana Gučanin[1], Antonio Žilić[1] and Želimir Korać[2]

[1]Department of Anesthesiology, Intensive Medicine and Pain Medicine, [2]Department of Surgery, Karlovac General Hospital, Karlovac, Croatia

SUMMARY – Aggressive large volume resuscitation is obligatory to achieve necessary tissue oxygenation. An adequate venous preload normalizes global hemodynamics and avoids multiorgan failure (MOF) and death in patients with multiple injuries. Large volume resuscitation is associated with complications in minimally monitored patients. A properly guided resuscitation procedure will finally prevent MOF and patient death. Transpulmonary thermodilution technique and gastric tonometry are used in venous preload monitoring, calculating volumetric hemodynamic variables and estimating splanchnic perfusion as well. We present a 24-year-old man with multiple injuries resuscitated with large volume infusions and monitored by transpulmonary thermodilution technique and gastric tonometry. It is very important to monitor regional hemodynamics that enables clinician to maintain the required relations between global and regional hemodynamics. It prevents the development of MOF and patient death.

Key words: *Resuscitation – methods; Multiple trauma – complications; Hemodynamics; Emergency treatment – methods; Case report*

Introduction

Hemorrhagic shock is the main cause of shock in patients with multiple trauma[1]. Inappropriate treatment leads to cell hypoxia and death[2]. Intravenous infusion fluids are the main therapy for patient stabilization and reduction of shock effects[3].

The main goals of therapy are divided into three groups: (a) mean arterial pressure (MAP), heart frequency and diuresis optimization; (b) venous preload, cardiac output (CO), mixed venous oxygen saturation (SvO_2), oxygen delivery (DO_2) and regional hemodynamics (splanchnic perfusion) maintaining; and (c) hypoperfusion laboratory markers (lactates, pH, base loss) normalization[4]. Venous preload is defined as the level of venous preload that can achieve adequate cardiac function and systemic perfusion[5]. The lack of optimal venous preload predictors and empirical fluid infusions are closely connected with a high risk of overfilling, intravascular and extravascular compartment. Hypothermia, coagulopathy and abdominal compartment syndrome are the most common complications of large volume resuscitation[6]. According to some authors, aggressive volume resuscitation performed within the first 24 hours of trauma improves patient outcome[7].

Correspondence to: *Head Doctor Mirjana Lončarić-Katušin, MD*, Department of Anesthesiology, Intensive Medicine and Pain Medicine, Karlovac General Hospital, Andrije Štampara 3, HR-47000 Karlovac, Croatia
E-mail: mikatusi@inet.hr

Received March 8, 2010, accepted in revised form July 20, 2010

Methods

We used minimally invasive hemodynamic monitoring PiCCO (Pulsion, Medical System, Germany) and gastric tonometry (Tonocap, Datex-Ohmeda, USA). PiCCO is an abbreviation that means pulse contour cardiac output. PiCCO continuously analyzes the curve of invasive arterial pressure after initial measurement of CO by transpulmonary thermodilution technique without using pulmonary artery catheter. Thermistor probe is inserted through brachial artery. We give a bolus of 20 mL of cold water, temperature about 5 ^{0}C, through central venous catheter. Thermistor then measures change in the blood temperature and extrapolates hemodynamic calculations from the pulse contour and shape of the hemodynamic curve. It is also adjusted to monitor invasive pressure as an important parameter in global hemodynamics.

PiCCO data are related to: (a) venous preload: intrathoracic blood volume (ITBV), global end-diastolic volume (GEDV), stroke volume variation (SVV), pulse pressure variation (PPV); (b) heart contractility: stroke volume (SV), global ejection fraction (GEF); and (c) afterload: systemic vascular resistance (SVR).

The volume substitution was monitored by parameters that are related to venous preload: ITBV, GEDV and central venous pressure (CVP) measurement. These actions prevent overfilling and optimize volume load.

Gastric tonometry is a monitoring device for regional-splanchnic perfusion. It measures pH in gastric mucosa by diffusion of CO_2 from gastric content to cuff of tonometric probe. Gatric pH (pHi) is an indicator of stomach and splanchnic perfusion. pH values of 7.2 and lower are indicative of hypoperfusion. This method measures CO_2 gap in two ways, i.e. as a difference between pCO_2 in gastric mucosa and arterial blood ($PaCO_2$); and gastric mucosa and expired air ($EtCO_2$), in normal conditions lower than 10-15 mm Hg.

Case Report

A 24-year-old man was admitted to intensive care unit (ICU) after having sustained a motorbike accident. He had multiple injuries. His incoming vital parameters upon admission were as follows: Glasgow Coma Score (GCS) 12 points (3,3,6), noninvasive blood pressure (RR) 50/40 mm Hg, sinus tachycardia 165/minute, respiratory frequency 16/minute, arterial oxygen saturation (SaO_2) 90%, and afebrile.

Clinical examination followed by radiological diagnostic procedures revealed severe trauma with hemorrhagic shock; bilateral frontal intracerebral hematomas; paraplegic lower extremities; left femur complicated fracture with hematoma forming compartment syndrome; left lower leg complicated fracture with compartment syndrome; left femoral and saphenous vein and left femoral circumflex artery rupture; and left iliac and right pubic bone fracture.

He was immediately endotracheally intubated and mechanically ventilated (pressure controlled ventilation) after developing acute respiratory insufficiency. Therapeutic procedures were divided in two parts: first, resuscitation with large volume of intravenous fluids in order to maintain adequate venous preload and normalize global and regional hemodynamics; second, the patient was prepared for urgent surgical procedure that took four hours according to the criteria for damage control surgery[8].

Surgical procedure was performed as follows: incision of the left lower leg and evacuation of the hematoma; ligature of the left circumflex femoral artery; sutures of the left saphenous vein; fasciotomy of the left upper leg; and external fixation of the left femur and tibia. Simultaneously, volume resuscitation was guided by global hemodynamic parameters (PiCCO) and regional hemodynamic parameters (Tonocap). He was mechanically ventilated for 7 days. Additional surgical procedures were performed according to the criteria for damage control surgery[9].

Ultimate length of hospitalization in ICU was 21 days. Recurrent acute respiratory insufficiency de-

Table 1: Fluids for intravenous infusion used during the first 24 hours of resuscitation

Solution	Total (mL)
Hyperoncotic hyperosmolar (10 % NaCl)	200
Crystalloids	9300
Plasma alternatives (6% HAES)	2000
Blood	11550 (24 doses)
Fresh frozen plasma	2850
Total	25900
Intraoperatiyely	9860 mL

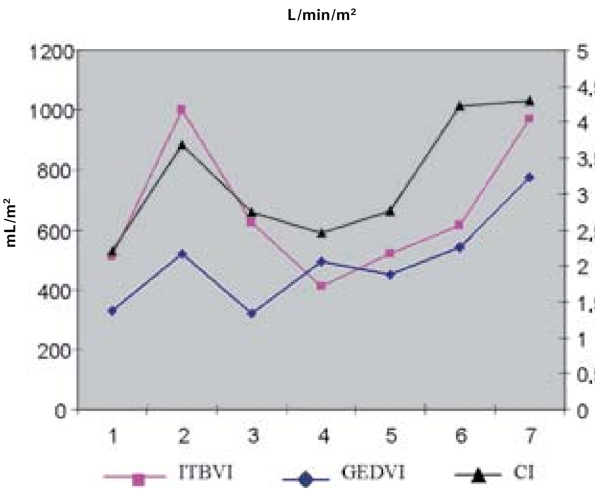

Fig. 1. Changes in intrathoracic blood volume index (ITBVI), global end diastolic volume index (GEDVI) and cardiac index (CI) during volume resuscitation.

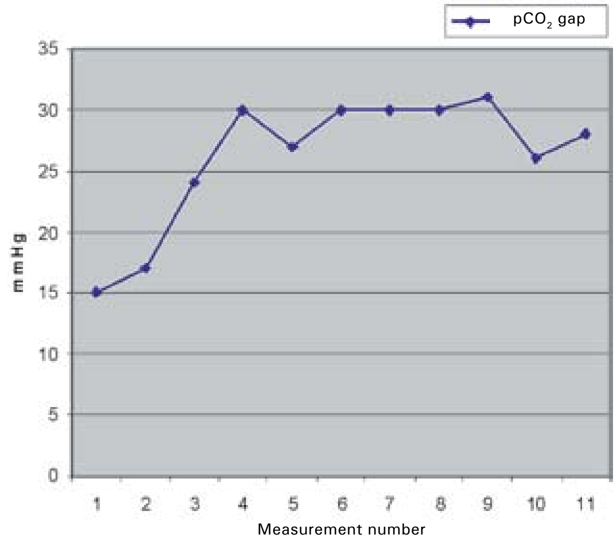

Fig. 2. pCO_2 gap during volume resuscitation.

veloped on postoperative day 14 due to pulmonary microembolism. The patient was reintubated and mechanically ventilated for the next two days. Left parietal ischemia resulted in right leg paresis.

After 21 days of ICU stay, the patient was transferred to Department of Traumatology. Three months later he started rehabilitation program.

During the first 24 hours, resuscitation volume load was 25 900 mL and 9860 mL intraoperatively (Table 1). The efficacy of volume resuscitation was guided by maintaining venous preload according to intrathoracic blood volume index (ITBVI) and global end diastolic volume index (GEDVI). Indexed values are absolute values *per* body surface. Minimal ITBVI during resuscitation was 410 mL/m^2 and GEDVI 328 mL/m^2. These values resulted in cardiac index (CI) of 2.74 L/min/m^2. CI could also be normalized by normalization of GEDVI and with conditional ITBVI more than 600 mL/m^2 (Fig. 1).

The response to volume load was monitored by SVV and PPV with consequential normalization of CI and MAP. The effect of volume load on splanchnic perfusion was monitored by pCO_2 gap; difference in partial pressure of carbon dioxide between gastric cells and arterial blood ($PaCO_2$); and estimated gastric mucosa acidity (pHi). In our case, we maintained pCO_2 gap 30 mmHg and lower, which was a sign of preserved splanchnic perfusion (Fig. 2). Lung function was estimated by monitoring extravascular lung water index (EVLWI) and pulmonary vascular permeability index (PVPI) to avoid pulmonary edema.

CI rising, SVV and PPV less than 10% were indicative of the patient's good response to volume load.

Discussion

In patients with multiple injuries, MOF is still very high despite advances in resuscitation, organ support techniques and better understanding of the metabolic response to trauma ranging from 14% to 42%[10].

Volume resuscitation is the basis of hemodynamic stabilization of patient in hemorrhagic shock[11]. Appropriate venous preload is the first step in maintaining adequate perfusion in patients with multiple traumas[12]. In this way, volume resuscitation has favorable effect on splanchnic perfusion monitored by gastric tonometry and results in better values of prognostic scores. In our case, we used Acute Physiology and Chronic Health Evaluation Score (APACHE II) and Multiple Organ Dysfunction Score (MODS) for status evaluation on admission[13]. On admission, APACHE II score was 28 with 63% predictive mortality; at 48 hours it was 10 with 11.3% predictive mortality. MODS was 9 in the first 24 hours (hospital mortality rate of 50%); at 48 hours MODS was 3 (hospital mortality rate of 11.3%). Standard therapeutic procedure during fluid resuscitation has to achieve correction of global hemodynamic variables that correlate with blood pressure, consequently leading to variable outcome of critically ill patients[14]. Outcome variability is the consequence of unrecognized shock masked by the patient compensatory mechanisms that lead to the state of shock despite normal blood pressure and heart frequency[15]. Corrected global hemodynamics is not a predictor of damaged tissue oxygenation recovery[16]. Regional variables of splanchnic perfusion are considered as better predictors of the existence of unrecognized shock[17].

Poeze *et al.*[18] showed that there was no superior hemodynamic variable in critically ill patients, which could be recognized as outcome predictor. The variables of splanchnic perfusion were found to be better predictors of outcome. In our case, the aforementioned variables also proved to be better predictors in relation to global hemodynamics, pressure and volume variables. On the other hand, supranormal volume resuscitation could result in intestinal perfusion reduction and development of abdominal compartment syndrome causing MOF and patient death[19]. Only carefully selected patients can benefit from volume resuscitation (preload responsiveness)[20].

Optimization of the circulating blood volume in a patient with multiple trauma is the most important point in normalization of global hemodynamics, which should be achieved as soon as possible, along with maintaining adequate splanchnic perfusion[21].

In most cases, correct diagnosis and therapy depend on precise measurement of hemodynamic parameters in critically ill patients[22]. Although pulmonary artery catheter is the "gold standard" of hemodynamic monitoring, less invasive methods such as PiCCO, Lithium Dilution Cardiac Output Measurement (LIDCO plus; LiDCO, Cardiac Sensor Systems, United Kingdom), transthoracic and transesophageal echocardiography are used for hemodynamic monitoring and volume status assessment in critically ill patients[23].

PiCCO as a less invasive method analyzes the curve of invasive arterial pressure after initial measurement of cardiac output using transpulmonary thermodilution technique.

Volumetric parameters such as ITBVI and GEDVI are the parameters that evaluate venous preload. Together with EVLWI, they make possible optimization of volume therapy in critically ill patients[24].

In routine assessment of volume status, cardiac load is assessed by measuring CVP and PCWP (pulmonary capillary wedge pressure). These filling pressures of the heart are indirect indicators of volume status; hence intravascular volume does not correlate with the values of the mentioned pressures. ITBVI is a better indicator of venous preload than CVP and PCWP in critically ill patients.

Determination of volumetric parameters was found to be a very good method in resuscitation of patients with multiple trauma. In our case, ITBVI reflected both changes, i.e. changes in volume status and cardiac output. ITBVI is an indicator of total venous preload. In septic patients with pulmonary edema, ITBVI and GEDVI as volume variables are better indicators of EVLW assessment than pressure variables[25]. Assessing ventricular filling and recognition of patients that will benefit from volume resuscitation preload are important determinants of good clinical outcome[20,26]. PiCCO hemodynamic monitoring allows for assessment of SVV. In patients on mechanical ventilation, this functional hemodynamic monitoring may identify those that will benefit from therapy with intravenous fluid-volume responsiveness. It shows the potential of cardiac output increase upon volume load[27].

Clinical studies[28,29] show that changes in cardiac index are in correlation with changes in ITBVI, but not with SVV. Continuous monitoring of SVV is a predictor, but also allows for opportune therapy as part of overall hemodynamic treatment[30].

Gastric tonometry assesses splanchnic perfusion and facilitates clinician's decision on the necessary supranormal values of global hemodynamic parameters. In life threatening hypovolemia and intestinal ischemia as early signs, it will protect from MOF development[31,32]. Gastric tonometry as a minimally invasive method and prognostic indicator of occult hypovolemia is a guide for appropriate therapeutic procedure. This way of intestinal perfusion monitoring can decrease the episodes of mesenterial ischemia and improve outcome in critically ill patients[33]. Gastric tonometry as a noninvasive method of intestinal mucosa perfusion (stomach or sigmoid colon) is sensitive for lower blood flow.

pHi under 7.2 indicates ischemia. Problems with pHi determination are due to the fact that equal arterial blood and gastric mucosa bicarbonate concentration with regional perfusion changes can disturb this relation.

Clinical studies show that low pHi in patients with multiple traumas is associated with the development of MOF and death[34]. Chang *et al.*[5] report that supranormal values of venous preload during resuscitation were connected with better patient outcome and lower incidence of MOF and death. Low pHi during resuscitation can be understood as an indicator of insufficient resuscitation and a mechanism of MOF development[35]. Studies focused on therapeutic treatment in order to increase pHi showed improvement in patient outcome[36]. Gomersall *et al.*[33] evaluated 210 patients with pH lower than 7.35 after resuscitation. This group of patients received colloid and dobutamine infusions. The authors conclude that therapy titrated towards high pHi in critically ill patients cannot be accepted. There was no difference in disease outcome. The reason for this is either disability to produce significant clinical change in pHi or it is simply a marker of disease rather than a factor of MOF pathogenesis[37].

Large volume resuscitation is obligatory in polytraumatized patients to achieve adequate tissue perfusion. It is associated with complications and minimally invasive monitoring is necessary for its optimization. Volumetric parameters allow for appropriate preload assessment, which is the basis of volume resuscitation. Regional hemodynamics monitoring is important and enables the clinician to maintain the relation between global and regional hemodynamics, thus preventing MOF and patient death.

Conclusion

In patients with severe trauma, aggressive volume resuscitation is an imperative to achieve adequate tissue oxygenation. It is associated with complications and minimally invasive monitoring carries the potential for its optimization. Volumetric parameters allow for an appropriate assessment of preload, which is the basis of volume resuscitation. At the same time, the methods of regional hemodynamics monitoring available such as tonometry enable the clinician to maintain proper relations between global and regional hemodynamics, thus reducing the occurrence of MODS and death in these patients.

References

1. PUYANA JC. Resuscitation of hypovolemic shock. In: FINK MP, ABRAHAM E, VINCENT JL, KOCHANEK P, eds. Textbook of critical care. 5th edition. Philadelphia: Saunders, 2005. p. 1933-43.
2. GUTIERREZ G, REINES HD, WULF-GUTIERREZ ME. Clinical review: hemorrhagic shock. Crit Care 2004;8:373-81.
3. JUKIĆ M, ŠALINOVIĆ K. Traumatsko-hemoragijski šok. Intenzivna medicina 2008;44:526-55.
4. WILSON M, DAVIS DP, COIMBRA R. Diagnosis and monitoring of hemorrhagic shock during the initial resuscitation of multiple trauma patients: a review. J Emerg Med 2003;24:413-22.
5. CHANG MC, MEREDITH WJ. Cardiac preload, splanhnic perfusion, and their relationship during resuscitation in trauma patients. J Trauma 1997;42:577-84.
6. SHAFI S, KAUDER DR, DONALD R. Fluid resuscitation and blood replacement in patients with polytrauma. Clin Orthop 2004;422:37-42.
7. SPAHN D, CERNY V, COATS TJ, *et al.* Management of bleeding following major trauma: a European guideline. Crit Care 2007;11:414.
8. BESMAN A, KIRTON O. Pelvic and major long bone fractures. Crit Care 2005;247:2097-102.
9. McARTHUR BJ. Damage control surgery for the patient who has experienced multiple traumatic injuries. AORN J 2006;84(6):992-1000 quiz 1001-2.
10. MOORE FA, SAVAIA A, MOORE EE, *et al.* Postinjury multiple organ failure: a bimodal phenomenon. J Trauma 1996;40:501.
11. DIEZ Ch, VARON AJ. Airway management and initial resuscitation of the trauma patient. Curr Opin Crit Care 2009;15:542-7.
12. HALJAMAE H. Fluid resuscitation in hemorrhagic shock and following blunt trauma; is there still a place for colloids? Acta Anaesthesiol Scand Suppl 2001;45 Suppl 115:17.
13. NGUYEN HB, RIVERS EP, HAVSTAD S, KNOBLICH B, *et al.* Critical care in the emergency department: a physiologic assessment and outcome evaluation. Acad Emerg Med 2000;7:1354-61.
14. SAMBASIVAN ChN, SCHREIBER MA. Emerging therapies in traumatic hemorrhage control. Curr Opin Crit Care 2009;15:560-8.
15. MAJNO G, JORIS I. General ischemia: shock. In: MAJNO G, JORIS I, eds. Cells, tissues, and disease: principles of general pathology. 2nd edition. Oxford: Oxford University Press, 2004. p. 715-25.
16. SCHLICHTING E, LYBERG T. Monitoring of tissue oxygenation in shock: an experimental study in pigs. Crit Care Med 1995;23:1703-10.

17. TAKALA J. Splanchnic perfusion in shock. Intensive Care Med 1994;20:403-4.
18. POEZE M, SOLBERG BCJ, GREVE JWM, RAMSAY G. Monitoring global volume related hemodynamic or regional variables after initial resuscitation: what is better predictor of outcome in critiically ill septic patients? Crit Care Med 2005;33:2494-500.
19. HOLTE K, SHARROCK NE, KEHLET H. Pathophysiology and clinical implications of fluid excess. Br J Anaesth 2002;89:622-32.
20. PINSKY MR. Assessment of indices of preload and volume responsiveness. Curr Opin Crit Care 2005;11:235-9.
21. SAKKA SG, REINHART K, MEIER-HELLMAN A. Does the optimization of cardiac output by fluid loading increase splanchnic blood flow? Crit Care Med 1993;21:1296-303.
22. LOPES AA, O'LEARY PW. Measurement, interpretation and use of hemodynamic parameters. Cardiol Young 2009;19: Suppl. 1:8-12.
23. SHOEMAKER W, WO C, CHAN L, *et al.* Outcome prediction of emergency patients by noninvasive hemodynamic monitoring. Chest 2001;120:528-37.
24. MICHARD F, ALAYA S, ZARKA V, BAHLOUL M, RICHARD C, TEBOUL JL. Global end-diastolic volume as an indicator of cardiac preload in patients with septic shock. Chest 2003;124:1900-8.
25. BOUSSAT S, JACQUES T, LEVY B, *et al.* Intravascular volume monitoring and extravascular lung water in septic patients with pulmonary edema. Intensive Care Med 2002;28:712-8.
26. FIEGL C, KEYSER A, PRASSER C, KEYL C. Assessment of fluid responsiveness in mechanically ventilated cardiac surgical patients. Eur J Anaesthesiol 2005;22:658-65.
27. REUTER DA, FELBINGER TW, SCHMIDT C, *et al.* Stroke volume variations for assessment of cardiac responsiveness to volume loading in mechanically ventilated patients after cardiac surgery. Intensive Care Med 2002;28:392-8.
28. BERKENSTADT H, FRIEDMAN Z, PREISMAN S, KEIDAN I, LIVINGSTONE D, PEREL A. Pulse pressure and stroke volume variations during severe haemorrhage in ventilated dogs. Br J Anaesth 2005;94:721-6.
29. DONATI A, NARDELLA R, GABBANELLI V, SCARCELLA M, ROMANELLI M, ROMAGNOLI L, *et al.* The ability of PiCCO *versus* LiDCO variables to detect changes in cardiac index: a prospective clinical study. Minerva Anestesiol 2008;74:367-74.
30. SAKKA SG, REINHART K, MEIER-HELLMAN A. Does the optimization of cardiac output by fluid loading increase splanchnic blood flow? Br J Anaesth 2001;86:657-62.
31. HEARD SO. Gastric tonometry. The hemodynamic monitor of choice (Pro). Chest 2003;123:469S-74S.
32. MILLER PR, KINCAID EH, MEREDITH JW, CHARA CD. Threshold values of intramucosal pH and mucosal arterial CO2 gap during shock resuscitation. J Trauma 1998;45:868-72.
33. GOMERSALL CD, JOYNT GM, FREEBAIRN RC, HUNG V, THOMAS A, TEIK E. Resuscitation of critically ill patients based on the results of gastric tonometry: a prospective, randomized controlled trial. Crit Care Med 2000;28:607-14.
34. KIRTON O, WINDSOR J, WEDDERBURN R, HUDSON-CIVETTA J, LYNN M, HERMAN M, *et al.* Failure of splanchnic resuscitation in the acutely injured trauma patient correlates with multiple organ system failure and length of stay in the ICU. Chest 1998;113:1064-9.
35. VENKATESH B, MEACHER R, MULLER MJ, MORGAN TJ, FRASER J. Monitoring tissue oxygenation during resuscitation of major burns. J Trauma 2001;50:485-94.
36. BARQUIST E, KIRTON O, WINDSOR J, HUDSON-CIVETTA J, LYNN M, HERMAN M, *et al.* The impact of antioxidant and splanchnic-directed therapy on persistent uncorrected gastric mucosal pH in the critically injured trauma patient. J Trauma 1998;44:355-60.
37. NIEUWENHUIJZEN GA, GORIS RJ. The gut: the 'motor' of multiple organ dysfunction syndrome? Curr Opin Clin Nutr Metab Care 1999;2:399-404.

Sažetak

REANIMACIJA BOLESNIKA S VIŠESTRUKIM OZLJEDAMA VELIKIM VOLUMENOM KRISTALOIDNIH-KOLOIDNIH INFUZIJA – KORELACIJA IZMEĐU OPĆE I REGIONALNE HEMODINAMIKE: PRIKAZ SLUČAJA

M. Lončarić-Katušin, M. Belavić, J. Žunić, S. Gučanin, A. Žilić i *Ž. Korać*

Agresivna reanimacija velikim volumenom je neophodna kako bi se postigla potrebna tkivna oksigenacija. Dostatnom prethodnom venskom opskrbom normalizira se opća hemodinamika i izbjegava višeorgansko zatajenje i smrt kod bolesnika s višestrukim ozljedama. Reanimacija velikim volumenom udružena je s komplikacijama u slučaju nedostatnog nadzora bolesnika. Ispravno vođen postupak reanimacije u konačnici će spriječiti višeorgansko zatajenje i smrt bolesnika. Tehnika transpulmonalne termodilucije i želučana tonometrija primjenjuju se u nadzoru venske opskrbe, izračunavanju volumetrijskih hemodinamskih varijabla i procjeni visceralne prokrvljenosti. Prikazuje se slučaj 24-godišnjeg mladića s višestrukim ozljedama, kod kojega je provedena reanimacija infuzijama velikog volumena i nadzor pomoću tehnike transpulmonalne termodilucije i želučane tonometrije. Vrlo je važno nadzirati regionalnu hemodinamiku, jer to kliničaru omogućuje održavanje potrebnih odnosa između opće i regionalne hemodinamike. Time se priječi razvoj višeorganskog zatajenja i smrt ovakvog bolesnika.

Ključne riječi: *Reanimacija – metode; Višestruke traume – komplikacije; Hemodinamika; Hitno liječenje – metode; Prikaz slučaja*