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1st Symposium of Anaesthesiologist and Reanimatologists in FB&H with International Participation

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ASSOCIATION OF MEDICAL DOCTORS ANESTHESIOLOGISTS - REANIMALOGISTS IN THE FEDERATION OF BOSNIA AND HERZEGOVINA

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GUEST LECTURER

Fluid treatment of dehydration, hemorrhage and uncontrolled hemorrhage

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Dehydration

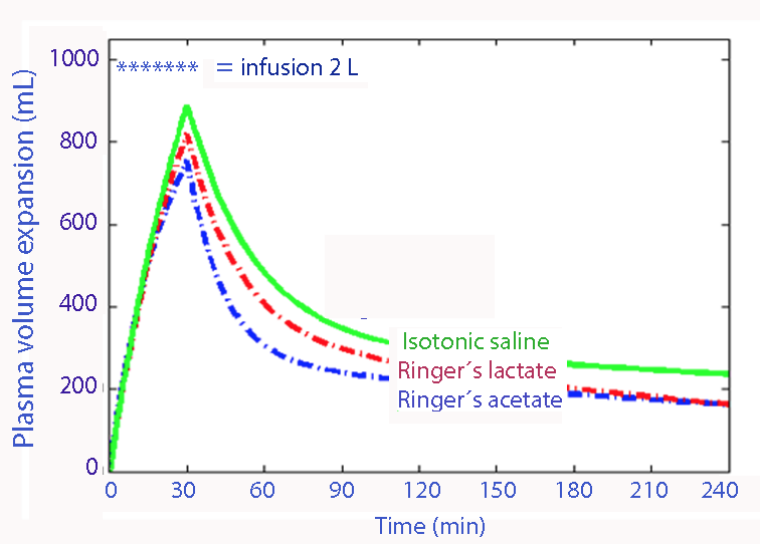
Humans need to ingest at least 1 ml/kg/hour of water to compensate for evaporation, which occurs mostly from the airways, and baseline urinary excretion. If intake is low or losses occur we talk about dehydration. There are two different forms of dehydration:

1. A common form of dehydration among debilitated elderly humans is due to insufficient intake of water. Here, the lack of volume is distributed over all body fluid spaces. This water deficit causes the serum osmolality to increase. At the same time the urine becomes concentrated, i.e. we see increasing urine osmolality and a higher creatinine concentration (normal is 600 mosmol/kg and about 10 mmol/L).

As the goal is to rehydrate all body fluid compartments, the appropriate intravenous fluid therapy consists of dextrose, preferably in a 2.5% or 5% solution (called *maintenance fluid*). Without the addition of 80 mmol of sodium per day the patient might develop clinical hyponatremia. The rate of infusion cannot be higher than 4 L in 6 hours due to the risk of hyperglycema, which is particularly at risk in trauma because of associated insulin resistance. Even with this rate, the expected plasma glucose level becomes 9 mmol/L, and higher values should not be allowed (1).

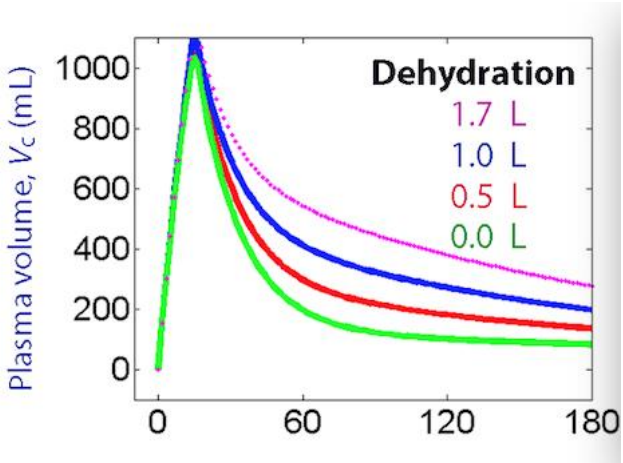
This rate is very slow in the trauma situation and, therefore, the therapy is usually combined with *resuscitation fluids* as priority is given to restoration of the blood and extracellular fluid volumes.

2. Another type of dehydration consists of losses of extracellular fluid only. This type is also called volume depletion and may be the consequence of diarrhea, vomiting, or excessive diuretic therapy. Furosemide causes a classical form of volume depletion. Here, the appropriate infusion fluid is lactated or acetated Ringer´s solution, except in pure vomiting where isotonic saline should be used.



The kinetic profiles of these three fluids are shown above (2, 3). They have a distribution phase where most of the volume accumulates in the plasma, whereafter distribution throughout the extracellular fluid space takes place. At the of a 30-min infusion the plasma volume expansion is about 50% of the administered volume, but this soon decreases as the infusion is discontinued.

In the dehydrated human the kinetics of Ringer´s does not look much different. Kinetic analyses have been performed of the fluid shifts occurring after infusion after using furosemide to dehydrate volunteers by almost 2 L (3, see figure below). Here, focus is on the central (the plasma) volume when 1.5 L of fluid is infused over only 15 min. As shown in the figure, distribution occurs fairly independently of the degree of dehydration. If the sum of the plasma and interstitial volumes are considered, the infused becomes *excreted* which makes the volume depletion to return after some time.

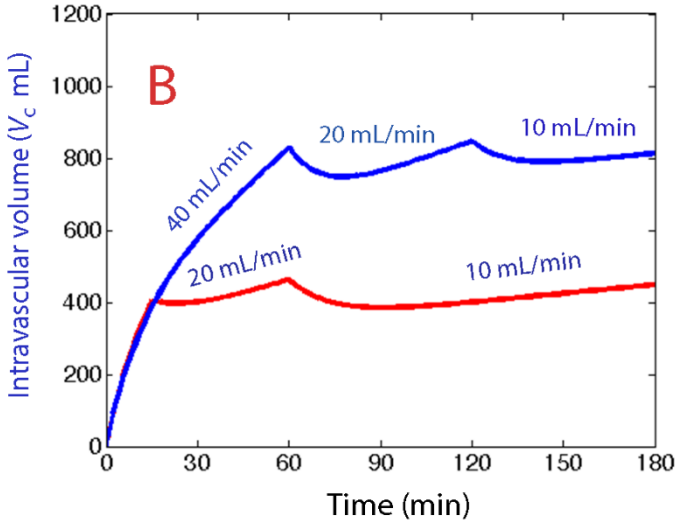


Therefore, the appropriate therapy is to infused Ringer´s at a medium rate, such as 40 mL/min for 30-60 min, and then to reduce the rate by 75% over the subsequent 2 hours.

Hemorrhage

The loss of blood volume is a crucial event and results in hypovolemic shock if more than 1 L is lost in an adult. Up to that limit the adrenergic system maintains the blood pressure, while cardiac output falls. Fluid therapy can be given as Ringer´s, colloid fluid, or blood products. The use of Ringer´s is effective due to the prominent distribution phase, but the infused fluid

doesn´t do the job well for very long as it is excreted, albeit a bit more slowly than in a normovolemic person. Depending on the degree of hypovolemia, kinetic analyses of the fluid shifts show that the following rates are appropriate if one wants to maintain normovolemia over time, after hemorrhage volumes of 450 and 900 mL (4):



How crystalloid fluid behaves in hypovolemic hypotension is not well known. However, it is likely that the clinical efficacy of Ringer´s is improved because low arterial blood pressure restricts the urinary excretion (5).

An alternative is to infuse a colloid fluid. The two currently recommended colloid fluids in trauma are 5% albumin and gelatin. In volunteers they expand the plasma volume by as much as the infused volume, and it is likely to be so also in hypovolemia.

The body has an intrinsic capacity to restore hypovolemia. The mechanism is called *capillary refill.* About 1/3 of a hemorrhage is compensated by fluid from the interstitial fluid space within 30 min, and the rest is compensated over several hours. However, this is too slow for the body; if fluid treatment is not instituted, hypovolemic shock enters an irreversible state after 1 hour.

Uncontrolled hemorrhage

This type of hemorrhage implies that bleeding is still ongoing through an opened major vessel. Here, arterial hypotension and a blood flow rates serve a purpose, namely to allow the coagulation system to plug the leak. However, the clot is still immature and might be washed away if the arterial pressure and blood flow rate is increased. In this setting vigorous fluid therapy can be a killer as it opens the injured vessel and allows the bleeding to continue ("re-bleeding"). Many animal experiments show that optimal survival is obtained by judicious fluid administration. Here, the goal is NOT to restore normal hemodynamics, but to balance successfully between preventing irreversible shock and fatal rebleeding. A good rule is to infuse half as much fluid half as fast. In the pig, crystalloid fluid given in proportion 3:1 to the blood loss causes severe rebleeding; 2:1 or 1:1 is associated with a better survival (6). A sudden drop in arterial pressure implies the onset of rebleeding, and further fluid therapy should be instituted at a lower infusion rate.

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COAGULATION AND HEMOSTASIS

1

Further look at the coagulopathic bleeding in trauma

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Introduction

So far, many studies have been done in the field of traumatic coagulopathy and according to them, recommendations for its treatment have been adopted. However, the pathomechanism of traumatic coagulopathy remains unclear. More research has recently been done on the pathogenesis and mechanism in order to improve the treatment and reduce the mortality associated with this condition. This review will in fact present new views on pathomechanism, as its understanding is a crucial for successful treatment.

Definition, incidence, pathogenesis and mechanism

Several terms are used in the literature to refer to coagulopathic bleeding in trauma, including trauma-induced coagulopathy (TIC) (1), acute traumatic coagulopathy, early coagulopathy of trauma, and the acute coagulopathy of trauma-shock. TIC is a phenomenon of acquired coagulopathy associated with increased total mortality in trauma patients. The prevalence of TIC according to different coagulation tests is 25%-42% of all trauma patients (2). TIC starts early after injury (3) and one of 4 trauma patients have coagulopathy at admission (4). The modern concepts of the pathogenesis of coagulopathy after traumatic injury imply that there is early acute coagulopathy in trauma and it is independent predictor for mortality. The early acute coagulopathy associated with traumatic injury has recently been recognized as a multifactorial primary condition that results from a combination of bleeding-induced shock, tissue injury- related thrombin-thrombomodulin-complex generation and activation of anticoagulant and fibrinolytic pathways. Early examination of the predictive factors for coagulopathy in trauma patients is important. Routine detection of posttraumatic coagulopathy is recommended which includes early, repeated and combined prothrombin time (PT) measurement, activated partial thromboplastin time (APTT), fibrinogen and platelets levels (Grade 1C). PT and PTT remain independent predictors of mortality in multiple logistic regression analysis. Viscoelastic methods can also be performed to help characterize coagulopathy and guide haemostatic therapy (Grade 1C) (5). In the mechanism of TIC, beside dilution of coagulation factors, acidosis, anemia, massive transfusion, hypothermia, consumpting coagulopathy, hyperfibrinolysis (due to reduced fibrin utilization under systemic hypoperfusion) play remarkable role (6). The detection of hyperfibrinolysis is essential as it is often undetected, undeclared or unexplored. Its incidence in trauma is 2-7%, depending on

the type of trauma and the severity of hypoperfusion (7). Hyperfibrinolysis can be detected at the earliest with ROTEM and is associated with higher mortality in severe trauma (8). More recently there are also reports that TIC may not be a disseminated intravascular coagolopathy (DIC). Within 24 hours of trauma, most severely injured patients have DIC scores "suggestive for" or of "overt DIC" but no anatomopathologic evidence of DIC. Considering pathologic findings as the gold standard diagnosis, DIC is exceptionally uncommon and the DIC scores should not be used for trauma (9). And so DIC with the fibrinolytic phenotype as a pathophysiologic mechanism for TIC has been definitively denied by researchers emphasizing activation of the thrombomodulin-protein C system as a principle pathway mediating TIC. Thence, “DIC hypothesis with a fibrinolytic phenotype” is a confusion of terms and should be abandoned. It is suggested that a state in which fibrinolytic activity exceeds the capacity of the haemostatic system to make stable clots, resulting in excess or uncontrolled hemorrhage, should be termed systemic activation of fibrinolysis with poor haemostasis (10). Further point of interest is acquired trauma coagulopathy as a phenomenon responsible for most post-operative traumatic bleeding. This coagulopathy might be present later due to the blood loss as a consequence of inadequate surgical haemostasis plus coagulation factors consumption, acidosis and hypothermia. In severe trauma with active bleeding, on the one hand we have tissue injury with coagulation activation, dilated coagulopathy resulting from fluid administration, and additional massive transfusion. When we have the triad of coagulopathy, acidosis and hypothermia, the patient enters a circulus viciousus from which it is difficult to get out of. Trauma patients with haemorrhage have injuries that require emergency surgery to control bleeding, and almost 80% of trauma death occurs in the operating room as a result of haemorrhage. Surgical bleeding is not a well-defined term, but can be broadly described as injuries that relate directly to operative visualization and are controlled by suture, packing, pressure, or application of haemostatic agents. If surgical bleeding is not treated in this way, it is likely to be fatal. Surgical bleeding originates from a recognizable site at the site of the trauma. Not all bleedings can be surgically controlled and there are various injuries and situations that fall into the category of non-surgical bleedings. These condition are area where surgical intervention has limited or no control of bleeding and where attempts of surgery can exacerbate traumatic hemorrhage and lead to severe bleeding and coagulopathy (e.g. pelvic fractures that have to be treated with angiographic embolization and bleeding due to coagulopathy treated with the establishment of normal haemostasis). Surgery in these conditions can lead to catastrophic consequences and lead to further blood loss, physiological disorder and possibly death. Changes in understanding TIC go back to the past when TIC was understood as a clinical syndrome featuring coagulopathy which occur in the early stage of trauma and is caused by activation of coagulation, fibrinolytic, and anti-coagulation pathways due to various factors, such as tissue injury and bleeding, but recently, it is proved in growing evidence that the endogenous coagulation disturbance in trauma itself is the activating factor of TIC, rather than dilution or other acquired coagulopathy (11). Tissue injury with endothelial cell damage and glycocalyx shedding, release of heparin-like substances and autoheparinisation (selfheparinization) is important mechanism for enchanging endogenous anticoagulation and are endogenous drivers of TIC. This, plus hypoperfusion, inflammation and sympatho –adrenergic activation, lead to TIC. Furthermore, the activated protein C pathway is one of the main initiating factors for TIC. Activation of protein C in the circulation occurs after

protein C binds to the protein C receptor in endothelial cells, which inhibits coagulation by affecting factors V and VIII, and promotes fibrinolysis by affecting plasminogen inhibitor-1 (PAI-1). Increased severity of injury and hypoperfusion are related to protein C level, coagulation parameters (PT and PTT), and hyperfibrinolysis. A decrease in the factor V and VIII levels was closely related to the severity of injury and the degree of hypoperfusion, which suggests that the activated protein C pathway plays an important role in TIC (12). Activated protein C may lead to degradation of fibrinolysis inhibitors, thence, the levels of coagulation factors might be relatively low in the presence of enhanced fibrinolysis. However, some researches prefer to believe that it is the result of the tissue-type plasminogen activator (tPA) secreted by the endothelial cells after being activated by stresses, such as injury. We need more molecular research to determine the exact mechanism of TIC.

Role of fibrinogen in major trauma

Normal plasma fibrinogen concentration is 1.8-4.3 g/L. The role of fibrinogen as a hemostatic agent in the management of traumatic hemorrhage is underestimated and there are several reasons for this. The presence of plasma expanders or high values of fibrin degrading products give artificially high values of fibrinogen. Decreased fibrinogen values are shown after the use of plasma expanders such as colloid, gelatin or dextran, with the resulting coagulopathy characterized as acquired hypofibrinogenemia and abnormal fibrin polymerization. Critical level of fibrinogen is 1 g/L (but it is significantly higher in practice). Reduced fibrinogen leads to acquired coagulopathy, but on the other hand systemic hypoperfusion is associated with decreased fibrin utilization and hyperfibrinolysis. The role of thrombin is to activate clotting proteins, to trigger platelet aggregation, and to convert fibrinogen to fibrin. Thrombin generation (TG) studies indicate that trauma patients with TIC show dysregulated hemostasis characterized by excessive non-wound-related TG due to a combination of circulating procoagulants capable of activating coagulation systemically and reduced inhibitor levels allowing systemic TG to continue once started (13). While the physiologic response to injury is to upregulate plasma procoagulant activity, the patients with reduced TG required more transfusions and had poorer outcomes. Measuring TG may provide an exquisitely sensitive tool for detecting disturbances in the enzymatic phases of coagulation in critically injured patients (14).

Platelets in trauma

Platelet (PTL) number in trauma patients initially is not critically reduced. PTL count < 150.000/µL is found in 4% of trauma patients with ISS score = 5, and in 18% of patients with ISS score > 45. The number of PTL is not their function. It is possible that PTL dysfunction is one of the earliest and most sensitive indicators of TIC. The specific mechanism is unknown but may involve “PTL exhaustion,” where PTL become activated en masse and are refractory to stimulation for up to 24 hours afterward (1). Other potential reasons for PTL dysfunction in trauma might be complement activation or inflammation. Minor decreases in PTL function increase mortality in patients with trauma.

Diagnosis of TIC

According to the European guidelines for management of bleeding and coagulopathy following major trauma (5) routine detection of post-traumatic coagulopathy which includes early repeated and combined measurement of PTT, aPTT, fibrinogen and PTL are recommended. Viscoelastic methods may also be performed to help characterize the coagulopathy and in guiding/ management of haemostatic therapy. Rotational thrombelastometry is used to promptly assess coagulation, but when it is not available, routine laboratory-based coagulation tests may be used for assessment of fibrinolysis.

Treatment of TIC

The treatment strategies should focus on achieving haemostasis as soon as possible and correcting coagulopathy; otherwise efforts at resuscitation are likely to be useless. Rapid control of the source of bleeding with damage control techniques instead of complete repair, interventional radiology especially in abdominal-pelvic trauma, use of blood derived products and haemostatic agents are essential. Early coagulation support may prevent acquired coagulopathy. The recommendations to avoid critical bleeding are constantly updated but in many situations, due to limited resources they cannot be applied completely. Target haemostatic therapy with lysine derivates and substituted coagulation factors in order to avoid packed red blood cells and fresh frozen plasma, are of vital importance. Protocol for the treatment of patient with major bleeding will depend on rational strategy which relies on the resources available. Tranexemic acid should be given in massive bleeding, even in the absence of clinically diagnosed hyperfibrinolysis. Fibrinogen and platelets have to be administered in order to provide thrombin formation when hyperfibrinolysis is considered. Bleeding that continues despite fibrin supplementation and adequate number of platelets may be a result of insufficient thrombin formation; in this case prothrombin complex concentrates may be the first line therapy for thrombin deficiency. Strategy in treatment of trauma patients relies on resource availability (15).

Conclusion:

New insights into the pathogenesis and mechanism of TIC will lead to more successful treatment and will reduce mortality in trauma patients.

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2

Coagulation Cascade Modulating Agents and Trauma

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Main Groups of Drugs That Modulate Coagulation
1. Antiplatelets
2. Anticoagulants
3. Thrombolytics
4. Herbal supplements

I will mainly focus on oral antiplatelets, on direct Factor Xa inhibitors, and vitamin K antagonists.

Antiplatelets

Antiplatelets can be divided to 4 groups according to their mechanisms of action:
1. COX inhibitors: Aspirin, and other NSAIDs
2. P2Y12 inhibitors: Clopidogrel, prasugrel, ticagrelor, cangrelor, and ticlopidine
3. GP IIb/IIIa inhibitors: Abciximab, eptifibatide, and tirofiban
4. Phosphodiesterase inhibitors: Cilostasol, and dypridamole

In Table 1 mechanisms of action or target molecules, route of administrations, and times for discontinuation before surgery are summarized while in Table 2 main indications and treatment recommendations of antiplatelet agents are given.

Table 1: Mechanism of Action/Target Molecule, Route and Discontinuation before Surgery

	Mechanism of Action/Target Molecule	Route of Administration	Discontinuation before surgery
Aspirin	Irreversible Inhibition of COX-1 and COX-2	oral/iv bolus	0-5 days
Clopidogrel	Irreversible inhibition of P2Y12 ADP receptor	oral	5-7 days
Prasugrel	Irreversible inhibition of P2Y12 ADP receptor	oral	7-10 days
Ticagrelor	Reversible inhibition of P2Y12 ADP receptor	oral	5-7 days
Cangrelor	Reversible inhibition of P2Y12 ADP receptor	iv continuous	1-6 hours

Abciximab	Reversible inhibition of GP IIb/IIIa receptor	iv bolus + continuous	48 hours
Eptifibatide	Reversible inhibition of GP IIb/IIIa receptor	iv bolus + continuous	8 hours
Tirofiban	Reversible inhibition of GP IIb/IIIa receptor	iv bolus + continuous	8 hours

Adapted from: Curr Opin Anesthesiol 2017, 30:466-473 and Anaesth Crit Care Pain Med 2018; 37: 379-389)

Some basic information about antiplatelets should be kept in mind in order to decrease risk of bleeding or other avoidable complications during surgery and anesthesia practice:

1. The aspirin-induced bleeding risk could be lower than that with clopidogrel, since thromboxane A2 plays a lesser role in platelet activation than ADP.
2. The bleeding risk is greater with dual therapy (aspirin + P2Y12 inhibitor) than with monotherapy (most often aspirin).
3. The bleeding risk due to clopidogrel is lower than that with prasugrel and ticagrelor.

Table 2: Treatment Recommendations for Special Conditions

Condition	Treatment Recommendation
Primary prevention	Aspirin in general not recommended
ACS (without PCI)	Aspirin (life-long) + Ticagrelor/Clopidogrel ≥ 12 months
ACS (with BMS/DES)	Aspirin (life-long) + Prasugrel/Ticagrelor/Clopidogrel ≥ 12 months
SIHD after DES	Aspirin (life-long) + Clopidogrel ≥ 6 months
SIHD after BMS	Aspirin (life-long) + Clopidogrel ≥ 1 month
Recent stroke	Aspirin and/or Clopidogrel
Past stroke	Aspirin (Clopidogrel)
PVD	Aspirin (Clopidogrel)

ACS, acute coronary syndrome; BMS, bare metal stent; DES, drug eluting stent; PCI, percutaneous coronary intervention; PVD, peripheral vascular disease; SIHD, stable ischemic heart disease (Adapted from: Curr Opin Anesthesiol 2017, 30:466-473)

When we meet a trauma patient on dual antiplatelet therapy we must decide with the surgeon the emergency level of surgery. If the surgery is really emergent (laparotomy, thoracotomy, pelvic packing, splenectomy etc.) to use damage control resuscitation techniques is important in order to avoid the development of lethal triad (coagulopathy, hypothermia, and acidosis). Always availability of alternative technique such as angiography should be kept in mind in order to decrease the risk of negative laparotomy.

If a viscoelastic method (VEM) is available then the resuscitation can be guided according to the results of it in polytrauma cases or in massive transfusion situations. One potential limitation of VEM may be the lack of sensitivity in detecting and monitoring platelet dysfunction due to antiplatelet drugs. If platelet dysfunction is expected, point of care (POC) platelet function tests, for example whole-blood impedance aggregometry, should be used in addition to VEM.

Treatment with platelet concentrates

1) If platelet dysfunction is documented in a patient with continued bleeding who has been treated with APA treatment with platelet concentrates is an option.

2) Administration of platelets in patients with intracranial hemorrhage (ICH) who have been treated with APA and will undergo surgery can be suggested.

The administration of desmopressin (0.3 μg/kg) can be considered in patients treated with platelet-inhibiting drugs or von Willebrand disease.

If the surgery can be postponed for 48-72 hours such as hip fracture surgery than every day 10-15% of platelets will be regenerated and when 20% of platelets are functional than adequate hemostasis can be achieved; but still platelet transfusions can be necessary especially in patients with continued bleeding intraoperatively and postoperatively.

After years of vitamin K antagonists (VKAs) being the only oral anticoagulative alternative, 'new' oral anticoagulant (NOAC) or 'direct' oral anticoagulant (DOAC) substances such as dabigatran, rivaroxaban and apixaban have become available. There are several beneficial characteristics of the so-called DOACs compared with warfarin and low molecular weight heparin (LMWH). While the drugs having xa on the middle of their names act by inhibiting factor Xa dabigatran inhibits thrombin complex (factor IIa). DOACs have a close dose-dependent effect; concentration and plasma peak concentrations are measurable a few hours after being administered orally. These facts supersede the need for routine drug monitoring.

The pharmacological properties of DOACs are quite complex: bioavailability, plasma protein binding ratios or elimination/excretion are quite different from each other; fortunately times to maximal concentrations (2-4 hours) and half-lives (10-14 hours) are similar. So in elective cases if there is a low bleeding risk and normal renal function, a discontinuation for 2–3 half-times (≈ 1 day) will be enough; but if a higher bleeding risk is expected, pausing for 4–5 half-lives (≥2 days) is more logical.

In the fifth addition of the European guideline on management of major bleeding and coagulopathy following trauma the measurement of plasma levels of oral direct anti-factor Xa agents such as apixaban, or rivaroxaban in patients treated or suspected of being treated with one of these agents is suggested (Grade 2C). If measurement is not possible or available, advice from an expert hematologist is recommended (Grade 2C). If bleeding is life-threatening, administration of tranexamic acid (TXA) 15 mg/kg (or 1 g) intravenously and that the use of prothrombin complex concentrate (PCC) (25–50 U/kg) be considered until specific antidotes are available (Grade 2C).

If bleeding is life-threatening in those receiving dabigatran, treatment with idarucizumab (5 g intravenously) (Grade 1B) and with tranexamic acid (TXA) 15 mg/kg (or 1 g) intravenously (Grade 2C) can be recommended.

Warfarin is the most commonly used VKA in the world, and the coumarins have a similar effect but a shorter (acenocoumarol) or longer (phenprocoumon) half-life. The most common indication for VKAs is the prevention of stroke in patients with atrial fibrillation. Other indications include prevention of thrombosis in those with previous venous or arterial thromboembolism or with mechanical heart valves.

There are three therapeutic options for the reversal of VKAs such as warfarin: vitamin K, PCC and fresh frozen plasma (FFP).
In the bleeding trauma patient, the emergency reversal of vitamin K-dependent oral anticoagulants with the early use of both PCC and 5 mg iv phytomenadione (vitamin K_1) (Grade 1A) is recommended.
Four-factor PCC (Factors II, VII, IX, and X) is administered intravenously in a dose of 25–50 U/kg, an algorithm can be used with which to calculate the most appropriate dose based on bodyweight and INR level. A stepwise dosage may be more appropriate, e.g. 25 U/kg if INR is 2–4.0, 35 U/kg if INR is 4–6.0 and 50 U/kg if INR is > 6.0.
The alternative to PCC is FFP, which also contains all the missing coagulation factors. Large volumes of FFP are required, and reversal is not always achieved and there are risks of transfusion-associated circulatory overload (TACO) and transfusion-related acute lung injury (TRALI). Again a stepwise dosage may be more appropriate, in our institution 2-3 U FFP are transfused if INR is 2–4.0, and 3-4 U of FFP are transfused if INR is >4.0 with vitamin K administration if PCC is not available. After the transfusion is completed INR is rechecked and 1 or 2 U can be transfused according to the result.

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3

Recent development in blood transfusion strategies in trauma

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Blood banking and transfusion of blood was initiated on a wide scale during World War I. Whole blood was the only product used up to the 1960s, when component therapy gained widespread acceptance. The task was then to maintain the blood volume with clear fluids, i.e. Ringer´s and colloids, until a pre-determined "transfusion trigger" was reached, after which erythrocytes were transfused. The trigger is set individually in the blood Hb range between 60 and 100 g/L, depending on the patient´s cardiac reserve (a lowered blood Hb is compensated with an increase in stroke volume). Transfusion of plasma follows other rules, but was mainly governed by not allowing the plasma fibrinogen concentration fall below 1 g/L. Hence, blood products were administered in a series, starting with crystalloids, erythrocytes, and then plasma. Platelets were finally administered when the entire blood volume had been substituted. This "component therapy" is still predominant in elective surgery. Here, the separate blood products are transfused according to their precise indications.

In trauma settings, these rules easily lead to patients being over-resuscitated with crystalloid fluids and receiving too little plasma. Therefore, in the past decade there has been a shift back to administration of more blood products in a balanced combination, with less emphasis on crystalloid fluid.

"Massive transfusion" is a term often used in trauma, and implies that 10 or more units of erythrocytes have been transfused within 24 hours. The mortality in these patients is between 20% and 50% (**1-3**). Death is most likely in the presence of the triad *hypothermia, metabolic acidosis,* and *coagulopathy*. The coagulopathy has been the most difficult to treat. Sadly, it is aggravated by infusion of clear fluids. Due to the complexity of the health threat, the transfusion strategy in trauma is more governed by outcome and "what works" rather that the precise indication for each blood component.

The strategy that has been favored during the past decade is to provide blood *products in the ratio 1:1:1,* i.e. to give units of plasma, erythrocytes, and platelets in the sample quantity. This strategy, originally suggested by Borgman et al. (**4**), has been validated in several studies, and has been shown to save lives (**4-7**).

Large trauma centers now store a number of universal donor blood in the emergency room in the proportion 1:1:1 to allow effective early therapy to be instituted in case a trauma patient needs acute massive transfusion.

Today, the reluctancy to infuse crystalloids has also spread to the *prehospital setting*. If possible, blood products should be brought out to the trauma scene with ambulance and helicopter, and plasma and erythrocytes be transfused on site. Coagulopathy and early survival has improved by such strategy (**8-10**).

Fibrinogen plays a crucial role in the success of massive blood transfusion in trauma. Fibrinogen promotes platelet aggregation and seems to be the key to the reduced incidence of coagulopathy (**11**). Therefore, there is a trend towards early use of plasma and, in particular, coagulation factor concentrates (**12**) and to target a plasma fibrinogen concentration of 1.5-2.0 g/L rather than 1 g/L (**13**). A retrospective military study reported decreased mortality among seriously injured soldiers treated with a high versus low fibrinogen to erythrocyte ratio (**14**). In one study of trauma-related hemorrhage, first-line treatment with coagulation factor concentrates versus fresh-frozen plasma resulted in a much reduced incidence of massive transfusion and the need for rescue therapy among those who received the coagulation factor concentrate (15).

Conventional coagulation tests, such as fibrinogen, activated prothrombin time, and international normalized ratio (INR), are less useful in trauma than tests of whole blood that integrate the coagulation cascade, such as thromboelastography. Here, the hemostasis is divided into initiation, amplification, and propagation. The integrated view of the coagulation is now a widely accepted in trauma care. Results are obtained fast and can guide critical decisions about further transfusions.

In conclusion, in trauma-induced hemorrhage one cannot rely on component therapy as we do during elective surgery. Better outcomes are obtained by a strict combination of plasma (preferably fresh-frozen), erythrocytes, and platelets according to a 1:1:1 scheme. The early use of coagulation factor concentrates is encouraged. They do not require cross-matching and further improve the outcome.

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4

Tranexamic acid in urgent medicine

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Trauma is one of the most leading causes of mortality worldwide. Among those trauma patients, haemorrhage is responsible for 30 to 40% of mortality. Effective and early resuscitation is an important and challenging task for all physicians involved in emergency care. To reduce bleeding, the effect of antifibrinolytic drug such as tranexamic acid (TXA) has been studied extensively.

Tranexamic acid (TXA) is an antifibrinolytic agent that has been initially used as an intravenous injection to reduce hemorrhage during tooth extractions, and later to reduce the need for blood transfusion in elective surgery. In 2011, results from a multi-center, randomized, and placebo-controlled trial (CRASH-2 trial) showed that TXA reduces mortality in bleeding trauma patients.

Giving TXA between one and 3 h of injury reduces the risk of hemorrhage death by about one-third without increasing the rates of vascular occlusive events. TXA is now being used in trauma patients who have or are at risk for severe hemorrhage. However, administration of TXA was not without controversy.

Key words: tranaxemic acid, polytrauma

BRAIN AND SPINE INJURIES

5

Traumatic brain injury, what's new

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Traumatic brain injury (TBI) is a leading cause of death and disability. It contributes to 30% of all injury-related deaths in the United States, [1] while many survivors live with significant disabilities, resulting in major socioeconomic burden. The highest rates of TBI were observed in older adults, very young (0 to 4 years) and young adults, males, while the most common mechanisms of TBI were falls, being struck by an object and motor vehicle crashes [1].
TBI is a heterogeneous disease and there are many different ways to categorize patients. TBI has traditionally been classified using injury severity scores, and the most commonly used is the Glasgow Coma Scale (GCS). It is universally accepted for TBI classification because of its simplicity and as predictive tool for overall prognosis. Using the GCS TBI is categorized as mild, moderate, and severe. Except this classification there is an alternative scoring system FOUR (Full Outline of UnResponsiveness), that includes a brainstem examination.
The pathophysiology of TBI includes primary and secondary brain injury.
Primary brain injury occurs at the time of trauma, damage includes focal contusions and hematomas, shearing of white matter tracts (diffuse axonal injury) along with cerebral edema and swelling. Secondary brain injury in TBI results from a cascade of molecular injury mechanisms that are initiated at the time of initial trauma and continue for hours or days. Secondary brain injury can be exacerbated by systemic events such as cerebral edema and increased ICP, hypotension, hypoxia, fever, seizures and hyperglycemia. The identification, prevention, and treatment of secondary brain injury are the principle focus of neurointensive care management for patients with severe TBI.
In the recent years international guidelines were developed for standardized approach in management of TBI [2]. The role of these guidelines was mainly to provide recommendations for management by using existing evidence. Neurointensive care units that operate based on guideline-driven protocols, have better patient outcomes.

Prehospital and early hospital management
The most important thing in prehospital management for severe TBI is the prevention and treatment of hypotension and hypoxia, two systemic events that are known to be major causes of secondary injury after TBI [3]. The injured brain is especially vulnerable to secondary insults in the first 24 hours. The study of Fuller from 2014 on 5057 patients with TBI entered into a European trauma registry, show that odds of death were 1.5 times greater at systolic blood pressure <120 mmHg, doubled at SBP<100 mmHg, tripled at SBP<90 mmHg, and six times greater at SBP<70 mmHg [4].
The aim of prehospital management is to normalize oxygenation and blood pressure:
-Prehospital endotracheal intubation is recommended in some patients with TBI. It is indicated in patients with GCS <9, an inability to protect their airway, or a SpO_2 <90% despite the supplemental oxygen [5].

-Prevention of hypotension in the prehospital setting is best achieved by fluid resuscitation using isotonic crystalloids.

-A prehospital assesment of GCS and appropriate precautions taken to stabilize and immobilize the spine during transport is a part of neurological assessment.

Early hospital admission phase (emergency department) of patients with TBI should be performed according to the Advanced Trauma Life Support (ATLS) protocol. This phase includes:

-Endotracheal intubation performed for all patients with a GCS score <9, inability to protect the airway, SpO_2 less than 90-93% with supplemental oxygen, or clinical signs of cerebral herniation. Adequate oxygenation (PaO_2 >60 mmHg) continues to be a priority.

-Monitoring of vital signs: heart rate, BP, pulse oximetry, capnography and temperature is obligatory. Hypoxia, hypoventilation, hyperventilation, and hypotension should be avoided [3].

-Neurological examination includes GCS, FOUR score. Computed tomography (CT) scan in all TBI patients with a GCS score of 14 or lower as soon as possible.

-Evaluation and management of increased intracranial pressure (ICP) should begin as soon as possible. Immediate lifesaving measures must be performed in patients demonstrating clinical signs of impending or ongoing cerebral herniation.

-Labaratory assessment (blood count, electrolytes, glucose, coagulation parameters).

-Transport of TBI patient to a hospital with neurosurgical services when they are hemodynamically stable.

Indications for surgical treatment after severe head injury are based on neurological status, or GCS, and findings on CT scan, such as large hematoma volume or thickness and evidence of mass effect including midline shift. Surgical guidelines have precise recommendation for surgical treatment of different neurological conditions.

But the very important thing is to perform the craniectomy when it is indicated as soon as possible. The study of Shackelford et al from December, 2018 concluded that postoperative mortality was significantly lower when craniectomy was initiated within 5.33 hours of injury [6].

Intensive care management

The most important goal of intensive care management of patient with severe TBI is to prevent secondary brain injury. The treatment is targeted to management of intracranial pressure (ICP) and maintenance of cerebral perfusion, to regulation of blood pressure and oxygenation and management of temperature, glucose, seizures and other potential secondary brain insults.

-Fluids- Isotonic fluids (normal saline) should be used to maintain euvolemia. Saline may be preferable to albumin.

-Avoidance of hypotension remains a priority in the ICU. Guidelines recommend maintaining the systolic BP ≥100 mmHg for patients 50 to 69 years old and ≥110 mmHg for patients 15 to 49 or >70 years old [2].

-Cerebral perfusion pressure is regulated through cerebral autoregulation, which can be disordered in 1/3 of TBI patients. An aim CPP of 60 to 70 mmHg is recommended to improve survival and favorable outcomes [2]. In order to regulate CPP, the first thing is to treat and lower the increased ICP.

-Ventilation - most patients with severe TBI are sedated and they are on artificial ventilation during the first several days. Acute hypercarbia may increase the ICP, while hypocarbia may

precipitate cerebral ischemia. Hypoxia should also be avoided, and the PaO_2 maintained >60 mmHg [3]. On the other hand, although hyperoxemia after TBI improves brain oxygenation and mitochondrial function, the use of hyperoxemia seems to be associated with increased in-hospital mortality [7].

The dilemma is when patients with TBI require higher levels of PEEP, because increased intrathoracic pressure will worsen venous return and increased ICP. One retrospective study from 2017 suggested that PEEP can be applied safely in patients with acute brain injury, although they did demonstrate a small but statistically significant positive relationship between PEEP and ICP [8]. The use of PEEP in TBI patients with acute respiratory distress syndrome (ARDS) does, however, seem to significantly improve brain tissue oxygenation, but with monitoring of ICP.

-Antiseizure drugs are generally recommended to prevent and treat post-traumatic seizures in the setting of severe TBI [2].

-Venous thromboembolism prophylaxis — Patients with TBI are at increased risk of venous thromboembolism (VTE), but, there is a potential risk of hemorrhage expansion, which is greatest in the first 24 to 48 hours. A recent systemic review from 2018 concluded that administering pharmacological VTE prophylaxis 24 to 48 hours postinjury may be safe for patients with low-hemorrhagic-risk TBIs [9].

-Management of coagulopathy – Around 1/3 of patients with severe TBI demonstrates a coagulopathy, which is associated with an increased risk of hemorrhage, poor neurologic outcomes, and death. Acute TBI produce coagulopathy through the systemic release of tissue factor and brain phospholipids into the circulation leading to inappropriate intravascular coagulation and a consumptive coagulopathy. The number of platelets is also important, some centers choose to maintain a platelet count >75,000/µl with platelet transfusions if necessary. In one cohort analysis from 2014, a platelet count of <135,000/microL was associated with a 12.4 times higher risk of hemorrhage expansion, while a platelet count of <95,000/microL identified patients who were 31.5 times more likely to require neurosurgical intervention [10]. Coagulation parameters should be measured in the emergency department (ED) in all patients with severe TBI. When a coagulopathy is identified, it is reasonable to use fresh frozen plasma (FFP), PCC, tranexemic acid.

-Glucose management - Avoidance of both hypo- and hyperglycemia is appropriate in patients with severe TBI, very high or low blood glucose levels should be avoided, with a range of 7.8 to 10 mmol/l.

-Temperature management- fever should be avoided, because it is associated with worse outcome. The goal is to maintain normothermia by use of antipyretic medications, surface-cooling devices, or endovascular temperature management catheters.

Elevated intracranial pressure (ICP) is associated with increased mortality and worse outcome. Initial treatment starts with simple techniques as soon as possible:

-30° head elevation to permit adequate venous drainage from the brain.

-All patients should be assessed for impending cerebral herniation. Clinical signs of impending herniation include significant pupillary asymmetry, unilateral or bilateral fixed and dilated pupils, respiratory depression, and the "Cushing triad" of hypertension, bradycardia, and irregular respiration. In such patients, the following measures must be taken immediately: endotracheal intubation, if not already performed; brief hyperventilation to a pCO_2 of approximately 30 mmHg, as a lifesaving measure; bolus dose of an osmotic agent capable of transiently reversing cerebral herniation; fluid resuscitation and vasopressors [2].

-ICP and CPP monitoring - Indications for ICP monitoring in TBI are GCS score ≤8 and an abnormal CT scan [2]. CPP is assessed through ICP monitoring, and recommended CPP of 60 to 70 mmHg appears to reduce mortality and morbidity [2]. A ventricular catheter, connected to a strain gauge transducer is the most accurate and cost-effective method of ICP monitoring and has the therapeutic advantage of allowing for cerebrospinal fluid (CSF) drainage to treat rises in ICP. Intraparenchymal ICP monitors are alternative method, but technically easier to place and associated with a lower risk of hemorrhage and infection. An ICP ≤22 mmHg is recommended [2]. In patients with a ventricular catheter, drainage of CSF is generally the first intervention for lowering ICP and it could be continuous or intermittent [2].

-Protocol-based analgosedation is recommended by 2018 guidelines to decrease duration of mechanical ventilation and ICU length of stay [11]. Intubated patient with severe TBI, with significant ICP elevation should be managed with an effective sedative agent in conjunction with the opioid infusion. Effective analgesia is very important, since patients with TBI often have pain that goes unrecognized. Fentanyl infusions are commonly used, while propofol may be the preferred sedating agent. In general, light sedation, is recommended when the ICP is adequately controlled.

-Osmotic therapy is generally used in TBI patients who are clinically symptomatic for cerebral edema or have documented ICP elevation that does not respond to initial measures such as CSF drainage, analgesia, and sedation. No specific hyperosmolar treatment protocol has been shown to improve function outcome or mortality in clinical trials. Hypertonic saline, from 3% to 23.4%, used in intermittent boluses or mannitol in boluses of 0.25 to 1 g/kg every four to six hours are used. The majority of studies do suggest improved ICP control with hypertonic saline, along with possible improvements in cerebral perfusion and brain tissue oxygenation [12].

Refractory ICP elevation — Patients with elevated ICP that is refractory to the measures described above may be managed with decompressive craniectomy, barbiturate coma, or hypothermia. Decompressive craniectomy is effective in controlling ICP and is potentially lifesaving in patients who have failed medical therapy, but many patients who require decompressive craniectomy will suffer severe disability.

Advanced neuromonitoring

Several technologies have been developed for the treatment of severe TBI, which allow the measurement of cerebral physiologic and metabolic parameters, with the goal of improving the detection and management of secondary brain injury. Many recent studies emphasize the importance of intraparenchymal probe for monitoring of brain tissue oxygen tension ($PbtO_2$) in detection of cerebral hypoperfusion after severe TBI and concludes that the management of severe TBI informed by multimodal ICP and $PbtO_2$ monitoring reduced brain tissue hypoxia with a trend towards lower mortality and more favorable outcomes than ICP-only treatment. Multimodal monitoring of brain physiology after TBI is also discussed as a possibility to lower mortality. Single intracranial access device with three lumens can measure simultaneously ICP, $PbtO_2$, brain temperature, cerebral microdialysis, cerebral blood flow sensor, or EEG monitoring electrode [13].

Prognosis

Cohort studies have suggested that patients with severe head injury (GCS ≤8) have approximately a 30% risk of death. Multiple studies indicate, however, that significant proportions (30 to 65%) of patients with severe TBI will regain independence, and that

functional recovery following severe TBI can occur very slowly, extending beyond even 6 to 12 months. Approximately 5 to 15% of patients with severe TBI are discharged from acute care in a vegetative state.

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6
Vetilatory strategies in head trauma patient

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Traumatic brain injury (TBI) is defined as an alteration in brain function or other evidence of brain pathology caused by external force. TBI is the most common cause of death under the age of 40 year. Severe TBI is diagnosed in the patient that has Glasgow Coma Scale (GCS) less than 9, after full resuscitation.

Central goals in head trauma, according to the Brain Trauma Foundation Guidelines, are prevention of hypoxic secondary insults through the maintained of an adequate cerebral perfusion pressure and cerebral oxygen delivery. Major issue is management of intracranial pressure (ICP), cerebral blood flow (CBF), and avoidance of secondary brain injury through the prevention of hypercarbia, hypoxemia and hypotension.

Mechanical ventilation is required in the majority of patients with severe traumatic brain injuries in order to control carbon dioxide (CO2) clearance, improve oxygen delivery, and facilitate resuscitation and injury management.

Conventional models of mechanical ventilation (assist/control AC, synchronized intermittent mandatory ventilations SIMV, pressure controlled ventilation PCV, airway pressure release ventilation-APRV or bilevel ventilation BIPAP) can be used in order to maintain low normocapnia and adequate oxygenation.

In the patient with the isolated severe brain injury, acute lung injury (ALI)/ acute respiratory distress syndrome ARDS occurs in 20-45% causing prolonged hospitalisation and higher mortality. It should be considered whether the selected mode of mechanical ventilation is being used to manage patient with isolated TBI or patient with ALI/ARDS, or to prevent ALI/ARDS.

Ventilatory strategies in these circumstances are challenging, because they can be in conflict. Therapeutic manoeuvres that improve oxygenation can have adverse effect on injured brain.

Non conventional models of mechanical ventilation of the lungs can be used in head trauma as salvage strategies (prone position, recruitment, high frequency oscillatory ventilation-HFOV, extracorporeal membrane CO2 removal-ECCO2R, extracorporeal membrane oxygenation-ECMO). Because of limited available evidence, these models of mechanical ventilation should be considered individually with monitoring of ICP and/or CBF.

Manipulating with CO2 by mechanical ventilation of lungs, we can strongly modulate CBF and ICP and directly have impact on outcome of TBI. TBI patients are special group in intensive care to which protective lung ventilation have to be adapted.

Keywords: ventilation, brain injury

7

Spinal cord injury – a challenge for anesthesiologist

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Spinal cord injury (SCI) remains a devastating event with often severe and permanent disability. The volume of SCI cases is not high and the nature of injury itself interfers with the anesthetic management, therefore it often represents a challenge for anesthesiologists.

Pathophysiology

As with the traumatic brain injury the mechanisms of spinal cord injury are considered as primary and secondary. The primary injury refers to the immediate effect of blunt trauma (forces of compression, contusion, and shear injury to the spinal cord with mostly partial spinal cord lesion) and penetrating trauma (usually produces a complete or partial transection of the spinal cord). The primary injury is followed by progressive secondary injury: ischemia, hypoxia, inflammation, oedema, excitotoxicity, disturbances of ion homeostasis, and apoptosis. Because of these secondary processes, spinal cord edema develops within hours of injury, becomes maximal between the 3rd and 6th day after injury, and begins to recede after the 9th day.

Clinical presentation

The usual symptom is pain at the site of spinal fracture, which can be masked by other brain and systemic injuries, which also complicate the initial evaluation and management of patients with SCI. Approximately half of SCIs involve the cervical cord and as a result present with quadriparesis or quadriplegia. There are different presentations of SCI:

- complete cord injury - spared sensory levels cranial to injury level, reduced sensation in the next caudal level, and no sensation in levels below. Similarly, there will be reduced muscle power in the level immediately below the injury, followed by complete paralysis in more caudal myotomes. In the acute stage, reflexes are absent, there is no response to plantar stimulation, and muscle tone is flaccid. A male with a complete SCI may have priapism. Urinary retention and bladder distension occur.
- incomplete cord injury — there are various degrees of motor function in muscles inervated by the levels of the spinal cord caudal to the injury. Sensation is also partially preserved in dermatomes below the area of injury, usually, to a greater extent than motor function. The bulbocavernosus reflex and anal sensation are often present.
- central cord syndrome — it is characterized by disproportionately greater motor impairment in upper compared with lower extremities, bladder dysfunction, and a variable degree of sensory loss below the level of injury after relatively mild trauma in the setting of preexisting cervical spondylosis.

- anterior cord syndrome — lesions affecting the anterior or ventral two-thirds of the spinal cord, sparing the dorsal columns, usually reflect injury to the anterior spinal artery. In trauma it is believed that this more often represents a direct injury to the anterior spinal cord by retropulsed disc or bone fragments rather than primary disruption of the anterior spinal artery.
- transient paralysis and spinal shock — Immediately after SCI a physiologic loss of all spinal cord functions caudal to the level of the injury, with flaccid paralysis, anesthesia, absent bowel and bladder control, priapism in males and loss of reflex activity can occur. This physiologic state may last several hours to several weeks and is referred to as a spinal shock. Clinical manifestations may normalize, but are more usually replaced by a spastic paresis reflecting more severe morphologic injury to the spinal cord.
- neurogenic shock: refers to hypotension, usually with bradycardia, attributed to interruption of autonomic pathways in the spinal cord causing decreased vascular resistance in large vascular beds (skeletal muscles, splanchnic vessels). This results in increased venous capacitance, decreased venous return to the heart and hypotension. At the same time there is predominance of parasympathetic activity, which is unopposed by sympathetic activity resulting in bradycardia, bradyarrhythmias and heart block. Bradycardia is the most common rythm, however, supraventricular tachycardia, ventricular tachycardia and cardiac arrest have also been reported. Neurogenic shock develops within 30 minutes following injury, peaks on day 4 and usually resolves over 2-6 weeks. It is more common with complete cervical SCI, but can appear also in high thoracic SCI (above Th 6). Besides hypotension and arrhythmias warm, flushed skin, priapism and diaphragmatic breathing with the injuries below C5 can be found. Injuries above C3 cause respiratory arrest.
- autonomic dysreflexia: is a constellation of signs and symptoms of excess sympathetic activity in response to a stimulus (pain) below the level of a SCI of T6 or higher. It is usually defined as an increase in systolic blood pressure (SBP) of >20 percent, often accompanied by bradycardia or arrhythmias, flushing, sweating, headache, blurred vision, and nasal congestion. Autonomic dysreflexia is more common in the chronic phase after injury, but it can occur in the acute phase as well. Autonomic dysreflexia should be treated immediately with removal of the stimulus and, if necessary, vasodilating medication in order to prevent complications of severe hypertension.

Initial evaluation and management

The primary assessment of a patient with SCI follows the ABCD prioritization scheme. If the patient has a head injury, is unconscious or confused, or complains of spinal pain, weakness, and/or loss of sensation, then a traumatic spinal injury should be assumed. Extreme care should be taken to allow as little movement of the spine as possible to prevent more SCI (rigid cervical collar, spinal bord for transfer and log-roll movements). Life-threatening priorities related to other injuries (bleeding, breathing difficulties…) can take precedence over the spinal cord injury.

Airway: The patient with a high cervical cord injury may require airway suction and/or intubation. Approximately one-third of patients with cervical SCI require intubation within the first 24h. Airway management may be difficult in patients with cervical spine injury because of immobilization and associated facial, head, or neck injuries. Rapid-sequence intubation with in-line spinal immobilization is the preferred method and if time is not an issue, intubation over a flexible fiberoptic laryngoscope may be a safer, effective option. Tracheostomy is

performed within 7 to 10 days, unless extubation is imminent. Patients with more severe cervical cord injuries are particularly likely to require tracheostomy. Futher details about airway management are described in anesthetic management section.

Breathing (respiratory) problems: Pulmonary complications, including respiratory failure, pulmonary edema, pneumonia, atelectasis, mucous plugging, and pulmonary embolism, are the most frequent complications during acute hospitalization after SCI with the incidence being highest with higher cervical lesions. Weakness of the diaphragm and chest wall muscles leads to impaired clearance of secretions, ineffective cough, atelectasis, and hypoventilation. The complications vary depending on the level and completeness of injury and preexisting respiratory status. The sympathectomy associated with injuries above T6 can result in bronchospasm and increased pulmonary secretions, which, when combined with the inability to produce an effective cough, lead to significant mucous plugging, obstruction of bronchioles, pneumonia, increased work of breathing, and ventilator failure. Respiratory complications in patients with thoracic SCIs are often related to direct chest trauma (traumatic pneumothorax, flail chest from rib fractures, rupture of the diaphragm or a bronchus, pulmonary contusions or lacerations, or hemopericardium).

Signs of impending respiratory failure, such as increased respiratory rate, declining forced vital capacity, rising pCO_2, or falling pO_2, indicate urgent intubation and ventilation with positive pressure support.

Circulatory complications: Spinal cord injury can produce a number of cardiovascular complications including hypotension, bradycardia, arrhythmias and early autonomic dysreflexia. Consequently patients presenting for anesthesia may be in an unstable hemodynamic condition. Besides, patients with SCI may have other injuries, therefore hemorrhagic shock, cardiac tamponade and pneumothorax should be ruled out as causes for hypotension. An adequate blood pressure is believed to be critical in maintaining adequate perfusion to the injured spinal cord and thereby limiting secondary ischemic injury. Albeit with few empiric supporting data, guidelines currently recommend maintaining mean arterial pressures of at least 85 to 90 mmHg and using intravenous (IV) fluids, transfusion, and pharmacologic vasopressors as needed. Administration of fluids should be guided by urinary output and hemodynamic monitoring, as excess fluids can cause further cord swelling and increase damage. Dopamine can be used alone or in combination with other vasoconstrictors (phenylephrine, norepinephrine). Bradycardia may require external pacing or administration of atropine.

Neurological status: Until spinal injury has been ruled out, immobilization of the neck and body must be maintained using cervical collar, straps, tape, and blocks. A neurologic examination should be completed as soon as possible to determine the level and severity of the injury, both of which impact prognosis and treatment.

Urinary complications: Patients with SCI are at risk for urinary retention, bladder distension injury, and consequently autonomic dysreflexia. They must be checked for bladder distension by palpation or ultrasound and urinary catheter should be inserted. Three or four days after injury, intermittent catheterization should be substituted, as this reduces the incidence of bladder infections.

Pressure Sores: most common on the buttocks and heels and can develop quickly (within hours) in immobilized patients. Backboards should be used only to transport patients with potentially unstable spinal injury and discontinued as soon as possible.

Gastrointestinal: Patients with SCI are at high risk of aspiration on induction of anesthesia. In the acute phase of SCI above the midthoracic level, gastric dilatation with delayed gastric

emptying, paralytic ileus, acute gastroduodenal ulceration and hemorrhage, and acute acalculous cholecystitis can all occur as part of spinal shock. Prophylaxis with proton pump inhibitors is recommended upon admission for four weeks.
Electrolyte disorders: Hyponatremia is common and may relate to disruption of the renal sympathetic pathways that regulate the renin–angiotensin response. Glucose tolerance may be impaired by the stress response or because of administration of glucocorticoids, especially in patients with preexisting diabetes.
Temperature control: The sympathectomy associated with high SCI can cause vasodilation and heat loss below the level of injury. Patients with neurogenic shock are frequently warm to the touch but hypothermic centrally. Core temperature should be monitored closely, with in-line fluid warmers and forced-air warming blankets used as necessary. Hyperthermia should be avoided, particularly in patients with associated traumatic brain injury.

Glucocorticoids — it has been suggested that methylprednisolone (30 mg/kg IV, followed by 5.4 mg/kg per hour over 23 more hours) might improve neurological outcome after SCI. However, according to results of studies its use is controversial. Some studies have shown benefit of its use (improved motor recovery), especially when given in the first 8h after injury, however in other studies there was no significant difference in neurologic function among treatment groups. Wound infections were somewhat more common in patients who received methylprednisolone. In 2013 the American Association of Neurological Surgeons and Congress of Neurological Surgeons stated that the use of glucocorticoids in acute spinal cord injury is not recommended. Some other surgical associations concluded that glucocorticoids are a treatment option and not a treatment standard. Since then the use of corticosteroids has declined. Contraindications to use of methylprednisolone is SCI with associated moderate to severe TBI. There are few data regarding the use of methylprednisolone in penetrating SCIs. However, retrospective studies suggest a higher rate of complications and no evidence of benefit. Because the neurologic benefits are uncertain, glucocorticoid therapy is not recommended in cases when there are clear risks associated with such therapy, such as penetrating injury, multisystem trauma, moderate to severe traumatic brain injury, and other comorbid conditions associated with risk of complications from glucocorticoid therapy. In other patients who present within eight hours of isolated, nonpenetrating cervical SCI, administration of IV methylprednisolone can be considered with knowledge of potential risks and uncertain benefits.

Surgery:

- Decompression and stabilization – there are currently no standards regarding the role, timing, and method of vertebral decompression in acute spinal cord injury. Options include closed reduction using traction and open surgical procedures. Closed reduction is an option for cervical spine fracture with subluxation. It involves use of longitudinal traction using skull tongs or a halo headpiece. Administration of a muscle relaxant or analgesic, such as diazepam or meperidine, may help facilitate reduction. Surgical reduction has the goal of reduction of dislocations as well as decompression of neural elements and stabilization of the spine. There are no evidence-based guidelines regarding the indications for or timing of surgery in SCI. In general, the specific management of cervical, thoracic, and lumbar spine and spinal cord injuries depends to a large extent on a surgeon's personal experience and practice norms in his or her center. Indications for cervical spine surgery include significant cord compression with neurologic deficits,

especially those that are progressive or that are not amenable or do not respond to closed reduction, or an unstable vertebral fracture or dislocation. Most penetrating injuries require surgical exploration to ensure that there are no foreign bodies imbedded in the tissue, and also to clean the wound to prevent infection.

- Timing of surgical intervention is not defined and remains controversial. Some studies suggested that early relief of spinal cord compression (within eight hours) leads to a better neurologic outcome, however, other have shown that early surgery led to increased rate of complications and poorer neurologic outcome, perhaps as a reflection of the vulnerability of the acutely injured cord. More contemporary studies suggest that medical complication rates are actually lower in patients who undergo early surgery, which allows for earlier mobilization and reduced length of intensive care unit and hospital stay. Most clinicians consider deteriorating neurologic function after incomplete SCI to be an indication to perform surgery as early as possible if there are no contraindications (hemorrhagic shock).

ANESTHETIC MANAGEMENT

Anesthesiologists are involved in treatment of patients with SCI in the emergency room (airway management), operating theatre (for spinal or other surgical procedures) and intensive care unit.

Preoperative evaluation — Preanesthesia evaluation should be as thorough as the urgency of the situation allows. Special consideration should be put on the mechanism of injury, possible additional injuries, course since injury, and medical history, besides an airway assessment and directed physical examination should be performed.

Injuries, that interfere with anesthetic management include facial fractures, traumatic brain injury (patient may not tolerate prone positioning for instrumentation of spinal fractures until cerebral edema has subsided), thoracoabdominal trauma, acute lung injury, and bony fractures. In many cases, the anesthesiologist, surgeon, and intensivist must discuss the relative risks and benefits related to the timing of surgery.

Airway evaluation — should be performed, including assessment of mouth opening, dentition, presence of other facial injuries, head trauma and blood in the airway. Until the cervical spine has been appropriately imaged to rule out an injury in a trauma patient, the anesthesiologist should assume that it is unstable (review spinal imaging). The patient may present with the cervical spine immobilized with a halo, a collar, or other devices that may affect the ability to mask ventilate or instrument the airway.

Choice of anesthetic technique — In most cases, patients with SCI will require general anesthesia for surgical procedure. Only very peripheral procedures (closed reduction of distal extremity fractures, closure of superficial extremity wounds) can be performed under peripheral nerve block, but only if it can be performed without moving the spine and is not otherwise contraindicated.

Monitoring — besides standard monitoring, patients with high thoracic and cervical spine injuries should have blood pressure (BP) monitored continually with an intraarterial catheter (placed before induction of anesthesia). Advanced hemodynamic monitoring is

also advised to guide fluid management. In case neuromonitoring is used during spine surgery, the choice of anesthetic agents must be modified to allow optimal monitoring (avoiding inhalational agents and muscle relaxants).

Venous access - Spine surgery may result in massive blood loss; we place two large-bore IV catheters (if possible, 14- or 16-gauge) for multilevel fusion and instrumentation. Central venous catheterization may be indicated when necessary for adequate access, and for administration of vasoactive drugs.

Premedication — Administration of anxiolytic and opioid medications should be done cautiously in SCI patients as they may have associated conditions that may predispose them to inadequate ventilation and oxygenation. Premedication should be administered in small doses or not at all, titrated to effect.

Preoxygenation — A head-up position is often beneficial, particularly for patients at risk for aspiration, however, patients with high SCI may become hypotensive when placed head-up. If airway management is anticipated to be difficult, and facemask preoxygenation also deemed difficult (patients with facial fractures) or ineffective, administration of O_2 via nasal cannulae during airway manipulation may delay oxygen desaturation.

Induction of anesthesia — Induction of anesthesia with IV medication is appropriate, choice of induction agents relates to the plan for airway management and other patient factors. Patients with thoracic and cervical SCI are at high risk for hypotension, therefore doses of induction agents should be reduced and a vasopressor infusion at a low dose should be started before induction (phenylephrine 20 to 40 mcg/min or ephedrine in bradycardic patients). Among neuromuscular blocking agents attention should be put on succinylcholine, which is contraindicated after 48-72h postinjury (possibility of severe hyperkalemia). If neuromonitoring is planned, rocuronium can be used for intubation and rapidly reversed with sugammadex to allow monitoring soon after intubation and/or patient positioning.
Rapid sequence induction and intubation (RSII) is often indicated. For patients with acute cervical SCI cricoid pressure during RSII should not be applied to avoid cervical spine motion. Patients with high thoracic and cervical spine injuries, especially those with preoperative bradycardia, are at high risk for severe bradycardia and even cardiac arrest with airway manipulation. Anticholinergic medication (atropine 0.4 mg IV, glycopyrrolate 0.2 - 0.4 mg IV) should be administered prior to induction of anesthesia.

Airway management — One of the most challenging procedures during management of a patient with cervical SCI is airway management. It may be difficult because of limited neck extension, facial or head injuries, retropharyngeal hemorrhage or oedema and the presence of spine stabilization devices. During airway management movement of the cervical spine must be avoided to avoid further injury of the spinal cord. No technique for airway management has been shown to be superior to others for prevention of neurologic deterioration in the patient with an unstable cervical spine. The technique should be chosen according to clinical circumstance, patient factors, and the expertise of the clinician. Experienced personnel and equipment for difficult airway management should be immediately available.

For patients with cervical or high thoracic SCI manual in-line stabilization (MILS) without applying traction should be used during all aspects of airway management. When a hard collar is in place in majority of cases it needs to be removed for intubation. MILS may prolong the time to intubation by worsening the view of the glottis; indirect laryngoscopy with a videolaryngoscope or flexible scope, or alternative airway management strategies may be required. Mask ventilation can cause significant cervical spine movement, therefore MILS should be performed at all times, only jaw thrust without chin lift should be used and an oral or nasal airway should be considered. The use of cricoid pressure for RSII is controversial, including for patients with cervical spine injury. Caution should be used especially in patients with lower cervical SCI, as it can increase distraction, angulation and translation of the spine. Use of gentle backward-upward-rightward pressure (BURP) maneuver can be considered if necessary to improve laryngoscopic view. For emergency procedures videolaryngoscope can be used to minimize spine motion. In nonemergency cases awake intubation is an option and the decision depends on the level of patient cooperation, and the expected degree of difficulty with all aspects of airway management. Blind nasal intubation can be performed, but for general anesthesia, flexible scope intubation (FSI) is the most commonly used awake technique. Retrospective reviews have reported no difference in neurologic outcome with asleep versus awake intubation for patients with cervical SCI. Advantages of awake intubation include the neutral position of the head and neck with minimal motion during airway management, maintenance of spontaneous ventilation and the possibility of neurological evaluation after airway management. However, usually it takes longer, coughing and gagging may occur, presence of secretions can make FSI difficult and it requires expertise. For patients with facial trauma or other features predicting difficult intubation, a surgical airway should be prepared.

Supraglottic airway devices can cause cervical spine motion and exert pressure on the cervical spine during insertion, inflation, and removal, therefore they should be used only as part of difficult or failed airway management.

Direct laryngoscopy with MILS is the most commonly used technique for emergency endotracheal intubation in patients with acute cervical SCI. It is often the intubating technique most familiar to the anesthesiologist and therefore the quickest and most sure method for securing the airway. The type of laryngoscope blade employed does not appear to significantly affect movement of the spine.

The use of a video laryngoscope (C-MAC, Airtraq, GlideScope, McGrath, or Bullard) with MILS improves the view of the glottis and reduce the degree of cervical spine motion compared with direct laryngoscopy. However, laryngoscopy and intubation may take longer with a videolaryngoscope compared with direct laryngoscopy.

Flexible scope intubation causes little motion of the cervical spine. However, coughing or gagging can occur during topicalization for awake intubation or during intubation if not adequately topically anesthetized, resulting in motion of the injured spine. Besides, it has a high failure rate in an emergency situation with providers who are inexperienced with this device.

Hemodynamic management — Acute SCI is often associated with hypotension as a result of sympathetic denervation, hypovolemia caused by associated injuries, or both. Blood pressure should be carefully managed in these patients to preserve spinal cord perfusion and to prevent secondary SCI. Spinal cord perfusion is dependent on mean arterial pressure (MAP) and is autoregulated over a wide range of systemic BP. Autoregulation may be lost

after SCI, rendering the cord more susceptible to ischemia with hypotension. Experts recommend the maintenance of a MAP of 85 to 90 mmHg for five to seven days after acute cervical SCI and avoidance of a SBP below 90 mmHg.

- Fluids: all patients with SCI require initial volume resuscitation starting with IV crystalloid, with addition of colloid and blood products as necessary. Volume overload should be avoided in order to prevent pulmonary and spinal cord edema. Acid–base status, lactate levels, estimated blood loss, and urine output should be utilized to guide fluid resuscitation. Of a good help could be also pulse pressure variation (PPV) of the arterial trace. During mechanical ventilation in the supine position, PPV > 11–13% predicts that the patient will respond to a fluid challenge with an increase in cardiac output.
- Intraoperative transfusion – The optimal hematocrit for patients with SCI has not been established. In general, the aim should be for a hematocrit of 21 - 30%.
- Vasoactive medications – With injury above the cardioaccelerator sympathetic innervation (T1-T4), vasopressors with inotropic and chronotropic properties in addition to vasoconstriction (dopamine, norepinephrine, epinephrine) are often required.

Temperature management — Temperature control is impaired in patients with SCI and core temperature is further reduced by general anesthesia. Fluid warmers and forced-air warming or cooling blankets should be used as necessary.

Emergence from anesthesia — The decision to extubate patients with partial and lower SCI depends on associated injuries, the length and position of the surgical procedure, blood loss, and IV fluid administration. The surgeon may want to perform a neurologic examination as soon as possible after surgery.

Postoperative pain control — The plan for postoperative pain control must be individualized and will be affected by the degree of spinal injury (level of sensory impairment), associated injuries, perioperative cognitive function, preoperative chronic use of opioids, and the need for postoperative ventilation and sedation. In majority of cases a multimodal approach to pain management will be appropriate. Chronic pain may develop in 26 – 96% of SCI patients.

Conclusions

Patient with spinal cord injury represents a challenge for anesthesiologist and requires experiences. The main goal of management is to prevent secondary injury to the spinal cord. Therefore, hypoxia should be prevented with the carefully planned airway mangement and hypotension with reasonable fluid administration and timely vasoactive medication application. The role of glucocorticoids is controversial and its use is decreasing.

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Organ donation in trauma victims – a case report

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Introduction

Organ transplantation is the most effective treatment for the terminal stage of organs failure. There is a huge imbalance between demand and supply of organs. In the United States, 20 people die every day waiting for a transplant, despite 36 528 transplants performed in 2018 (1). Most transplanted organs come from deceased donors. Up to 80-90% of organs are procured by donation after brain death (BD) and 10-20% via donation after cardiac death. Trauma victims account up to 30% of all deceased donors (2). In Bosnia and Herzegovina there is neither a national program of transplantation nor unique government's organ procurement organization. In the Entity of the Federation of Bosnia and Herzegovina, transplants of the kidney, liver and cornea have performed but the rate of living and deceased donations is very low. In such circumstances, are health care professionals able to manage the critical pathway of organ donation after BD of the trauma victims? We report a case of multi-organ donation after BD in trauma victim and first heart explantation in Bosnia and Herzegovina.

Case presentation

Anamnesis. A 19-year-old man (170 cm, 72.5 kg) was involved in a road accident and suffered severe multi-trauma. The patient was admitted to the Emergency Department of the Cantonal Hospital Zenica about 1 hour after the accident and was unconscious, without response to stimuli. The Glasgow Comma Scale was 3/15. Both pupils were medium-wide and fixed. Respiratory rate was six breaths per minute, peripheral O_2 saturation was 77%. Arterial blood pressure was 100/70 mmHg and heart rate was 120 per minute. The patient was immediately intubated without any medication required. After artificial ventilation onset, peripheral O_2 saturation was 94%. Three peripheral venous lines were inserted. Replenishing the circulating volume, analgesia and warming the patient was started.

Investigations. Cranial computed tomography (CT) revealed subarachnoid hemorrhage, severe general cerebral edema, multiple cerebral contusions, cranial base fractures, and facial bone fractures. Chest CT revealed lung contusions. Abdominal CT showed a retroperitoneal hematoma on the left side. Also, a fracture of the left tibia was diagnosed. Due to the severe and diffuse brain injury, a neurosurgical intervention was not possible. The patient was admitted in the Intensive Care Unit (ICU).

Treatment. Cerebral-protective intensive care therapy was immediately started: artificial ventilation, volume replacement, osmotic treatment of cerebral edema, antibiotic prophylaxis, proton-pump inhibitors and continuous analgosedation. Intensive monitoring

was performed: pulse-oximetry, invasive arterial pressure, central venous pressure (CVP), core temperature and urinary output. Four hours after ICU admission, control laboratory analysis showed a decrease in blood count and abdominal ultrasound showed free fluid in the minor pelvis. An exploratory laparotomy was performed. There was sequestration from retroperitoneal hematoma without bleeding from abdominal organs or vascular elements. Two units of concentrated erythrocytes and two units of fresh frozen plasma were administered. Twelve hours after ICU admission, control cranial CT found exacerbation of cerebral edema with compression over the ventricular system and morphological abolition of the brain structures. The patient's clinical condition met criteria of a possible organ donor. The analgosedation and osmotic therapy were stopped and intensive treatment was redirected to organ protection. Sufficient volume therapy with 6500 ml of crystalloid per 24 hour, supported by dopamine (5 μg/kg/min) in continuous infusion, secured optimal organ perfusion and oxygenation. Early substitutive administration of hydrocortisone was performed. Central diabetes insipidus was treated by repeated titrated application of desmopressin. Serum electrolytes, glucose and blood gases analysis performed every four hours and results determined the type of fluid and electrolyte management.

Twenty four hours after admission, all cranial reflexes were absent with no response to atropine. Clinical testing performed twice within three hours confirmed brain stem dead. The confirmatory test was a radionuclide scintigram that revealed no cerebral circulation. The patients was declared brain dead at 32 hours after hospitalization and his family consented to organ donation.

The patients had no earlier medical or surgical history and no risk behaviors. Serological tests were negative except cytomegalovirus IgG and Epstein-Barr virus nucleic acid. Biochemical markers of liver, renal and heart function were within range of normality. Ultrasound examination showed regular size, structure and function of the kidneys, liver and heart. At the time of procurement patient has been hospitalized for a total of 80 hours, hemodynamic stability was completely achieved. There were no signs of active infection (leukocyte, C-reactive protein, procalcitonin, cultures from blood, urine) and no signs of mineral imbalance. The patient became the donor of six organs. The heart and liver were transplanted at the German transplant center and the kidneys and corneas were transplanted at Bosnian transplant center.

Discussion

The first goal of the trauma management is to save the patient's life and create optimal recovery

conditions. In patients with devastating brain injury and obviously risk of death of neurological origin the most important is recognize the state of possible donor. Although prognosis of our patient was seen as extremely limited, life sustaining therapy was started as soon as possible. The recommendations of European Directorate for the Quality of Medicines & HealthCare were followed (3). When patient's clinical condition reached a state of possible organ donor, the treatment was redirected to organ protection. After declaration of BD, aggressive donor management was induced in order to prevent hypotension, central insipid diabetes, electrolyte disturbances, hypothermia, arrhythmias and coagulation disorders. Ensuring the vitality of the organs affected by pathophysiology of the trauma and BD is very challenging process.

Caring for relatives has a special significance in the process of organ donation. In our case, the first contact with the relatives included the most inaccurate prognosis and initial treatment.

Further, progression of injuries an expected BD was clarified. After declaration of BD, the parents were asked about their opinion about organ donation. Parental consent determined further activities to continue intensive donor management. Success in communication with relatives is the teamwork of the entire medical staff on shifts in the ICU (4).
Bosnia and Herzegovina is not a member of the Eurotransplant, but the transplant coordinator of the Federation of the Bosnia and Herzegovina registered eligible BD donor from our hospital in the Croatia Transplant. Our donor was offered in the Eurotransplant by mediating of the Croatia Transplant organization. The heart and liver were accepted from German transplant centre and their team of surgeon performed explantation in our institution.

Conclusion

To increase availability of organs for transplantation, it is critical to evaluate trauma victims as potential organ donors. In the developing countries, a higher rate of organ donation is possible through the combined efforts of healthcare professionals, government health institutions and population collaboration. Also, countries with high levels of the transplant programs could have a supportive role in this process, as our example has shown.

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PAIN MEDICINE

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Acute pain management

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ABSTRACT
Inspite of available pain killers, technical development of drug delivery pumps and different guidelines, acute postoperative pain is not sufficiently managed worldwide. There are different guidelines available for postoperative pain treatment strategies, procedure specific protocols can be tailored to individual needs. Effective postoperative pain relief enables early mobilization and recovery, protects immune system function and patient's wellbeing and prevents chronic postoperative pain. Pain measurement and supervision on surgical wards is crucial. Nurse – based anesthesiologist – supervised acute pain service (APS) introduced pain nurses and upgraded the role of surgical ward nurses and collaboration with surgeons to implement recovery after surgery protocols. Regular audits to improve postoperative outcome are crucial.
Key words: pain, acute, postoperative, management.

INTRODUCTION

Pain is defined by the IASP as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (1). Acute pain is defined as "pain of recent onset and probable limited duration. It usually has an identifiable temporal and causal relationship to injury or disease" Chronic pain "commonly persists beyond the time of healing of an injury and frequently there may not be any clearly identifiable cause". It has proven clinically useful to differentiate acute and chronic pain but it is important to recognise that classification based on time has limitations if the underlying pathophysiology is not taken into consideration (2).

EFFECTS OF UNEFFECTIVE POSTOPERATIVE PAIN RELIEF

Inadequately managed postoperative pain is associated with a broad range of negative consequences, including increased morbidity, development of chronic postoperative pain, impaired function, recovery from surgery, and quality of life, prolonged opioid use, and increased medical costs (3-6).

Morbidity

Inadequately managed acute postoperative pain is associated with effects related to aspects of both physiological and psychological function. Changes can occur in diverse organ systems, including the cardiovascular (coronary ischemia, myocardial infarction), pulmonary (hypoventilation, decreased vital capacity, pulmonary infection), gastrointestinal (reduced motility, ileus, nausea, vomiting), and renal (increases in urinary retention and sphincter tone, oliguria) systems. A negative impact may also be seen on immune function, the muscular system, coagulation, and wound healing.

Poorly controlled pain after surgery may impair sleep and have negative psychological effects, such as demoralization and anxiety (5,6).

Persistent postoperative pain

Poorly managed acute pain experienced after surgery may also result in the development of chronic pain. Based on reviews of the literature, persistent pain appears to affect between 10% and 60% of patients after common operations (3,4,6). There is some evidence for psychological factors (anxiety, depression, catastrophizing, overload stress) being predicting for the development of more severe and more prolonged pain after surgery. Fluctuations in reported incidence of chronic postsurgical pain are likely associated in part with the lack of a standardized definition of this complication, although it is frequently defined as enduring pain that has no other evident causes and lasts at least 2 months after surgery, past the expected healing period. The prevalence of chronic postsurgical pain varies by type of surgery and generally decreases with time. Over the last two decades, understanding of the mechanisms involved in the development of chronic pain has improved, with a combination of factors emerging as contributors, most notably inflammatory processes, tissue and nerve damage, and central sensitization. The development of chronic pain after surgical intervention has been shown to involve preoperative, intraoperative, and postoperative factors. Numerous studies of patients undergoing a broad variety of surgery types have demonstrated that the presence and intensity of acute postoperative pain are significant predictive risk factors for the development of chronic pain. Other potential determinants of persistent pain after surgery include younger age, female sex, obesity, genetic predisposition, preexisting pain, psychological factors (eg, preoperative anxiety and depression).

GUIDELINES

Guidelines advocate generalized pain management recommendations for the use of analgesic drugs and techniques (2,7-11). Such guidelines are derived from different surgical procedures with varying pain characteristics such as type, location, intensity and duration. In clinical practice it is well recognized that there are clear differences in perception of pain intensity and its consequences across different surgical procedures (4).

PROSPECT guidelines are an example of recommendations based on systematic reviews of the literature for a particular surgical procedure and include randomised studies that evaluate analgesic drugs and techniques and also anesthetic and surgical techniques on postoperative pain, taking into consideration also clinical routines and risk – benefit aspects. Guidelines are updated regularly are freely available online (9-11).

PAIN ASSESSMENT

Any guidelines are applied according to the intensity of pain, so pain assessment is crucial for the choice of analgesic drug and technique and also for evaluation of effectiveness of the chosen treatment. The ideal pain assessment tool would produce a numeric score or other objective metric, be easy to administer, be readily understood by patients, and yield reproducible results with good specificity and sensitivity. A number of clinically tested and validated pain scales exist (12).

Among the most frequently used pain scales is the visual analog scale (VAS). When using the VAS, the patient is shown a 100 mm line and asked to point to the area of the line that describes his or her pain, with the left end of the scale meaning “no pain” and the right end “the worst pain imaginable.” VAS score below 3 represents mild pain and successful pain relief.

ACUTE PAIN SERVICE

Nurse-based anesthesiologist-supervised acute pain service (APS) proved to be an effective and also a reasonably low-cost model (13). Pain management team consists of surgical ward nurses who assess and record pain, give analgesic drugs, monitor vital signs and side effects. Surgical ward nurses are trained by pain nurses who daily come to surgical wards and visit patients on PCA pumps for intravenous, epidural or peripheral nerve catheter analgesia. If needed pain nurses contact APS anesthesiologist to adjust analgesic regimen. APS is supervised by anesthesiologist who communicates with surgeons, physical therapists and also pharmacists.

Besides day-to-day responsibility for postoperative pain relief, APS pain nurses assume also training programmes of nursing and medical staff and regular auditing. According to the results obtained by monthly and annual analysis of pain scores, side effects and complications, APS anesthesiologist adjusts recommendations for pain treatment protocols. Patient satisfaction with the quality of postoperative pain management is also assessed and analysed.

MULTIMODAL ANALGESIA FOR ACUTE PAIN MANAGEMENT

To improve postoperative pain management, several concepts have been developed, including preemptive analgesia, preventive analgesia, and multimodal analgesia. Preemptive analgesia refers to the administration of an analgesic treatment before the surgical insult or tissue injury. Several randomized clinical trials have, however, provided equivocal evidence regarding the benefits of preincisional compared with postincisional analgesic administration. Current general consensus, therefore, indicates that use of preemptive analgesia does not translate into consistent clinical benefits after surgery. Preventive analgesia is a wider concept where the timing of analgesic administration in relation to the surgical incision is not critical. The aim of preventive analgesia is to minimize sensitization induced by noxious stimuli arising throughout the perioperative period. Multimodal analgesia consists of the administration of 2 or more drugs that act by different mechanisms for providing analgesia. These drugs may be administered via the same route or by different routes. Thus, the aim of multimodal analgesia is to improve pain relief while reducing opioid requirements and opioid-related adverse effects. Analgesic modalities currently available for postoperative pain control include opioids, local anesthetic techniques [local anesthetic infiltration, peripheral nerve blocks, and neuraxial blocks (epidural and paravertebral)], acetaminophen, nonsteroidal anti-inflammatory drugs, and cyclooxygenase-2-specific inhibitors as well as analgesic adjuncts such as steroids, ketamine, α-2 agonists, and anticonvulsants. (14).

CONCLUSIONS

APS led to an increase of appropriate use of specialized analgesic techniques. APS reduced the analgesic gaps that occurred during the transition from IV PCA or epidural PCA to oral analgesic therapy. Recorded and evaluated effectiveness of pain relief and possible side effects enables further possible improvement of postoperative analgesia. Several studies showed that implementations of APS was associated with a significant decrease in pain intensity and also lower incidence of postoperative complications. Multimodal analgesia remains the most widely recommended concept for postoperative pain management with the aim to improve pain relief while reducing opioid requirements and opioid-related adverse effects.

Nowadays undertreated postoperative pain is not due to the lack of effective drugs or techniques, but to the lack of an organized approach (APS) which uses existing treatments. The role of regular teaching programmes of ward nurses, implementation of standardized protocols, regular audits to improve outcomes cannot be overstated. Programmes for

education of patients are the next step to improve postoperative pain management and patient's satisfaction.

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10
Regional analgesia for pain management in traumatic patients

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Trauma is one of the leading cause of hospitalization in all age groups. It is always accompanied by pain. Pain is a good indication to determine the type of injury, but on the other hand, it can cause numerous compilations and deterioration of the injured patients. Therefore, it is crucial to know pathophysiology of pain and available methods of pain management in traumatic patients. Pain management is especially challenging in children and elderly due to multiple chronic medical conditions or heightened anxiety. Additionally, trauma patients can be presented with multiple injuries, substance abuse and emotional issues, which complicate care process.

Pain in traumatic patients is an acute pain and should be assessed and promptly treated. Traumatic patients usually have severe pain with score on visual analogue scale 7-10 out of 10. For pain, management administration of rapidly acting intravenous agents is usually recommended followed by multimodal pain management techniques.

Recently, techniques of regional analgesia such as neuraxial analgesia, peripheral nerve block, including intercostal blocks, interpleural catheter, plexus blocks and local anesthetic infiltration of incisions is widely used in everyday clinical practice for traumatic patients due lower side effect and effective long-lasting analgesia.

Providing effective pain management in trauma patients is crucial because it promote early healing, shorten hospital length of stay, lowers costs, prevents risk of chronic pain and ultimately reduces rate of disability and mortality.

11
Inadequate acute pain control and its consequences

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Pain is one of the most common causes for seeking health care: 20%–40% of the patients treated by general practitioners are estimated to suffer from different pain conditions . Millions of patients each year suffer from acute pain as a result of trauma, illness, or surgery. Pain is the most common reason for admission to the emergency department (ED), comprising more than 40% of the over 100 million ED visits annually [1]. Furthermore, a large proportion of cases in the ED setting involve pain of moderate to severe intensity [2]. Pain is defined as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (IASP 1986). Factors often associated with the development of long lasting pain include previous pain experiences, injuries, high age, female gender, and culture. Inadequate acute pain management has substantial consequences for patients. Unrelieved acute pain has consequences beyond the immediate perception of pain and can negatively impact patients' well-being on multiple levels.

Chronic pain (more than three-month duration) [1] is an acute and/or intermittent pain that persists and seems to be related to a complex interaction of risk and maintaining factors. Chronic pain has become a major health problem due to the high number of people affected. Apart from individual suffering, chronic pain is the cause of many social and financial pressures on society [2].

The causes of musculoskeletal chronic pain vary and are in several patients unknown. Some of the most common causes reported are injuries related to traffic accidents, falls, or sports injuries. In western countries, whiplash as the result of traffic injuries has a high annual incidence with 1.0 to 3.2/1000 per year [3, 4]. Whiplash is not only the term of the injury, it also describes the mechanism of energy—acceleration being transferred to the neck,—that results in soft tissue injury/distortion of the neck [5].

Chronic injury-related pain may be influenced by different physical and emotional factors that also affect daily life. During the last decade, attention has been paid to psychological factors such as depressive symptoms, anxiety, and negatively coloured cognitions that could be associated with future impairments [6, 7].

Posttraumatic stress reactions might play an important role in the persistence of symptoms after injuries and chronic pain after traffic accidents. In patients with traffic injury-related problems, 10–30% of the patients suffer from severe post-traumatic stress symptoms and receive a post-traumatic stress disorder (PTSD) diagnosis. Although the severity of post-traumatic stress decreases with time, it has been shown that about one-third of traffic victims

still suffer from post-traumatic stress reactions with anxiety and avoidance behaviour up to six years after the accident [8].

It is largely unknown how depression, anxiety, and posttraumatic stress interact in patients with injury-related chronic pain long after injury. A previous pilot indicated relationships between pain and stress, depression, and anxiety in patients with chronic pain [9]. Since most patients with chronic pain report poor-quality sleep that may influence the perceived level of depression and pain [10], the present study aimed to investigate all these factors in patients with chronic pain after trauma and who were referred to specialist care. A second aim was to examine differences between men and women.

BARRIERS TO PAIN MANAGEMENT

Numerous factors can contribute to inadequate pain management, including lack of sufficient physician training, lack of patient education about opioid use, as well as the side effects associated with certain analgesic therapy that contribute to noncompliance. Opioids are generally considered the treatment of choice for moderate to severe pain and are recommended for patients who are unresponsive to other types of analgesic agents [15,16]. However, a relatively low proportion of patients suffering from moderate to severe pain actually receive opioids to control their pain. In one study of patients who reported to the ED with closed fractures, among patients 16 to 69 years of age with moderate to severe pain, only about 60% received opioid analgesia [11].

Physician perceptions regarding analgesic therapy and lack of physician training in areas ranging from the recognition of inadequate pain management to the application of various available treatment modalities are factors that can contribute to inadequate pain management. Medical school and postgraduate training programs have historically placed a low educational emphasis on pain management , which has contributed to many physicians' negative attitudes about opioids and a reductance to prescribe them [12]. In contrast to the negative attitudes expressed by physicians and patients toward opioid therapy, studies have shown that when opioid analgesics are administered under proper physician supervision, treatment is associated with very low rates of opioid misuse. The apparent discrepancy between perceptions about the abuse potential of opioids and the actual risk of abuse supports the urgent need for improved physician and patient education with respect to the appropriate use of opioid analgesics for pain management[12].

It is evident that unrelieved acute pain can have negative consequences for various aspects of patients' health and quality of life. Conversely, effective pain management has been shown to mitigate these same sequelae of acute pain.

CONCLUSION

Inadequate management of pain unquestionably continues to have an unacceptable negative impact on patients' overall quality of life and ability to function both physically and mentally. Failure to administer appropriate analgesic treatment may result in worsening of pain and more frequent hospital readmissions, adding to the already high economic burden associated with pain therapy. Improved analgesic treatment that is well tolerated should provide more effective acute pain relief and is likely to benefit patients with respect to the various consequences associated with intense acute pain. Such measures are critical for facilitating early recovery and discharge, in addition to potentially reducing the risk for

development of chronic pain. In this respect, a multimodal approach to analgesia and establishment of acute pain services may have an important role in the future of acute pain management.

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12
Invasive procedures in chronic pain treatment

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Chronic pain is a major worldwide problem. In the United States, it is estimated that more than 100 million people suffer from chronic pain, with costs between $560 and $635 billion dollars per year [1]. The estimated prevalence of pain lasting at least three months is 14.6% [2]. The prevalence of chronic musculoskeletal pain conditions and frequent headaches is 43% [3]. Data from the 2012 National Health Interview Study estimated the prevalence of chronic daily pain to be 25.3 million people or 11.2% of the population [4]. These numbers do not describe the full impact that chronic pain has on productivity, quality of life, and human suffering.

To treat pain, the use of opioids has increased dramatically over the last several decades, with 9.6 to 11.5 million adults or approximately 3%–4% of the adult US population having been prescribed long-term opioid therapy [5]. Opioids have limited effectiveness for chronic pain and are accompanied by substantial risk of adverse outcomes including addiction, overdose, and deaths. Deaths from opioids now exceed deaths from motor vehicle accidents [6]. Thus, the need for nonpharmacological approaches for treating chronic pain has grown.

Invasive procedures (including surgery) might mitigate the need for chronic opioid and other pharmacological therapies and be viable options for chronic pain treatment. Procedures that completely replace damaged or arthritic joints or change major anatomical structures can produce long-term reduction in pain and improvement in function [7]. However, invasive procedures are increasingly being used for pain where the anatomical causes for the pain are not so clear.

The development of minimally invasive procedures has expanded the use of such interventions for treating a variety of chronic pain conditions such as low back pain [8], arthritis [9], and endometriosis [10]. In 2014, more than $45 billion was spent in the United States on surgical treatments for chronic low back pain (LBP). Arthroplasty costs for chronic knee pain topped $41 billion [11]. Invasive procedures are considered effective and are standard care for these two conditions. However, many types of invasive procedures are marketed, used, and paid for without evidence from rigorous study designs involving randomization, allocation concealment and blinding, or placebo controls. In the absence of these controls for common sources of bias, studies on invasive procedures may be giving a false impression of their true efficacy. Individual efficacy studies of invasive procedures have been published for LBP [12,13] and osteoarthritis of the knee [14], and a recent meta-analysis estimated the magnitude of the effects of sham surgery on subjective and objective outcomes [15]. However, no comprehensive systematic review of the current evidence on the safety and efficacy of invasive procedures compared with placebo treatment in chronic pain has been done.

The vast majority of injections done for the diagnosis or treatment of chronic pain are performed on an outpatient basis. Some are performed on inpatients, who may be already hospitalized for other reasons. All of them may be performed under fluoroscopic (x-ray) guidance but are sometime performed in the office without x-ray. For any nerve block, you

need to tell your doctor if you are allergic to contrast dye or if you think you may be pregnant. Below is a brief description of some of the more commonly performed nerve blocks by pain management specialists.

- **Epidural Steroid injection**: Epidural steroid injection is an injection performed in the back or neck in an attempt to place some anti-inflammatory steroid with or without a local anesthetic into the epidural space close to the inflamed area that is causing the pain. These injections are generally done for pain involving the back and leg or the neck and arm/hand. They may be done under x-ray guidance. Common side effects include soreness of the back or neck at the point where the needle enters the skin, there may be some temporary numbness in the involved extremity but persistent numbness or weakness (lasting over 8 hours) should be reported to your doctor. Epidural steroid injections may be placed in the lumbar (low back), thoracic (mid back), or cervical (neck) regions.
- **Facet Joint Injection**: The facet joints assist with movement of the spine both in the neck and back. Injection into these joints can provide relief of neck and back pain; these injections are always performed under x-ray guidance. Common side effects include soreness in the neck or back when the needle was inserted. You will be on your stomach for this injection if it is done for back pain; however you may either be on your stomach or back if the injection is performed for neck pain, depending on the preference of the physician. A needle is placed in your neck or back and advanced to the level of the joint under x-ray visualization. Contrast dye is used if the needle is put within the joint, and sometimes used if the injection is designed to numb the nerves to the joint. This block is often a diagnostic block and a more long lasting injection may be indicated if you have significant pain relief from this injection.
- **Lumbar Sympathetic Block**: A lumbar sympathetic nerve block is performed for pain in the leg that is thought to be caused by complex regional pain syndrome type I (or CRPS I). These injections are often performed under fluoroscopic (x-ray) guidance. Local anesthetic is placed near to the lumbar sympathetic chain in order to relieve the pain. Your leg will likely become warm immediately following the injection: this is an expected effect and not a complication. Back soreness is one of the more common side effects. If you feel any sharp pains down your leg or to your groin during the injection, you should let the physician know immediately. There may be some temporary numbness following the injection but if there is persistent numbness or weakness (> 8 hours) the doctor should be notified. You will be lying on your stomach for this injection. The injection is done from the back, in the lower aspect of the back. A needle is placed, often under x-ray guidance, to a spot just to the side and approaching the front part of the spine where the ganglion is located. If it is done under x-ray, a small amount of dye is injected to make sure the needle is in the right spot. After the doctor is satisfied that the contrast dye is in the right place, they will inject numbing medicine then remove the needle.
- **Celiac Plexus Block**: A celiac plexus block is generally performed to relieve pain in patients with cancer of the pancreas or other chronic abdominal pains. A needle is placed via your back that deposits numbing medicine to the area of a group of nerves called the celiac plexus. This injection is often performed as a diagnostic injection to see whether a more permanent injection may help with the pain. If it provides significant pain relief then the more long lasting injection may be done. This injection is usually performed under x-ray guidance. You will be lying on your stomach for this injection. The needle is place via the mid back and placed just in front of the spine. Contrast dye is injected to confirm that the needle is in the right spot; followed by some numbing medicine.

• **Stellate Ganglion Block**: A stellate ganglion block is an injection that can be performed for the diagnosis of complex regional pain syndrome of the arm or hand or for treatment of pain to that area. It can also be used to help to improve blood flow to the hand or arm in certain conditions that result in poor circulation of the hand. Side effects may include soreness in the neck where the needle was placed. In some instances the side effects may include droopiness of your eyelid on the side that is injected, along with a temporarily stuffy nose and sometimes temporary difficulty in swallowing. This injection is performed with or without x-ray guidance. You will be lying on your back for this injection with your mouth slightly open. It is very helpful to the doctor if you try not to swallow during the injection. If this injection is performed under x-ray the doctor will first inject a small amount of contrast to confirm the placement of the needle then inject some numbing medicine.

Epidurolysis procedure is used to loosen and dissolve some of the scar tissue from around entrapped nerves in the epidural space of spine, so that medications such as cortisone can reach the affected areas, and so that scar tissue is less painful.Scarring is most commonly caused from bleeding into the Epidural space following back surgery and the subsequent healing process. It is a natural occurrence following surgical intervention. Sometimes scarring can also occur when a disk ruptures and its contents leak out. The purpose of this procedure is to allow medications to reach affected nerves so that pain and other symptoms may be diminished, and to make scar tissue less painful. Epidurolysis requires a series of three injections. First, a catheter (a small tubing) inserted in the Epidural space up to the area of scarring. This is done in the operating room under sterile conditions using fluoroscopy (x-ray vision). This catheter is secured to the skin with dressings and tapes. The first injection of medications is made via this catheter. The patients are then kept in the hospital overnight. The second injection is done the following day; the catheter is injected and then removed. Will be sent home on the second day. The actual injections only take a few minutes. The Epidurolysis injection consists of a mixture of local anesthetic (like lidocaine or bupivacaine) and the steroid medication (triamcinolone – Aristocort® or methylprednisolone – Depo-medrol®, as well as x-ray contrast dye to visualize scarred space and hyaluronidase – and concentrated sterile salt solution to soften scar tissue and make it hurt less.

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AIRWAY MANAGEMENT

13

Airway management in pediatric trauma patient

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Summary

The main priority in managing of traumatized pediatric patients is establishing the airway which enables adequate oxygenation (1). A variety of issues of traumatized pediatric patients, including child' s anatomical, physiological and emotional specificity, need prompt response to provide adequate ventilation and oxygenation (1,2,3). Good knowledge of anatomical and physiological features in children is essential as well as experience of practitioners in procedures for rapid intervention (1,4). The most common pediatric emergences are traumatic injury (≈29%) (2,3,5). Traumatic head injury in children, either isolated or followed by other traumatic injuries of internal organs, limbs or spinal cord, represents the leading cause of serious morbidity and mortality (3,5). Neck injuries account for approximately 10% to 15% of serious injuries in children and can involve the aerodigestive, vascular and neurological systems (3). In particular, laryngotracheal injury is relatively uncommon in children, accounting for only approximately 0,5% of pediatric trauma admissions (3). External trauma of the laryngotracheal (LT) complex can be classified in to 2 general types external and internal (3). There are two types of the external trauma of the LT complex: blunt (bicycle/motor vehicle crashes, strangulation) and penetrating (gunshot/stab injuries, penetrating glass injuries) trauma (3). Mechanism of the internal LT complex trauma can be caused by trauma intubation injury, foreign body aspiration and caustic ingestion (3). Both of these types of neck trauma are capable of resulting in life-threatening airway compromise (3). Pediatric anesthesiologists are involved in preoperative management (resuscitation and stabilization in the emergency department, anesthesia for imaging), perioperative management and intensive care management of traumatized pediatric patients (1,2,3,5). Immediate control of the airway management, cervical spine, breathing and circulation are the most important initial interventions (2,3,5). Delayed management of the compromised airway, challenge of achieving proficiency for practitioners who infrequently perform pediatric airway management, is one of the most preventable cause of death in pediatric patients (1,2,3,5). Furthermore, there are numbers of syndromes and congenital anomalies, typically seen in the pediatric population presented with unique anatomical or functional specificity which can predict a difficult pediatric airway and which are always a challenge for airway management, even the experienced ones (1,2,3,6,7,8). Most pediatric cardiac arrests begin as respiratory failures or respiratory arrests (1). When the children "crashes" they will crash quickly with rapid progression to decompensated shock (1,2).

Child's anatomy and physiology

Children are not small adults. The main notable anatomic differences include (1,2,3,6,7,8):

1. Large head and prominent occiput;
2. Larg tongue;

Simple airway-opening techniques, such as triple maneuvers (head tilt, chin lift, jaw thrust, and/or oropharyngeal airway), usually enough to open the child's airway, all of them, are not recommended in trauma patients. If there is concern for C-spine injury, use a simple jaw thrust (1).

3. Cephalad larynx;
4. Epiglottis angled over vocal cord, long stiff and floppy;
5. The larynx is conically shape;
6. The trachea is short, in line with right bronchus and with small diameter so mucosal edema increase in resistance to air flow;

Differences in respiratory physiology between adults and pediatric patients have an effect on airway management (1,2,3,6,7,8). The pediatric patients have predisposition to hypoxemia:

1. Oxygen consumption of an infant is greater (6–8 mL/kg/min versus 4–6 mL/kg/min) than in adults just like the production of carbon dioxide (100-150mL/kg/min versus 60 mL/kg/min);
2. The respiratory rate in children is higher because of the need for higher minute ventilation so the CO_2 can be eliminated;
3. The closing capacity is larger than the FRC;

All this physiological features lead to low tolerance of apnea, shorter time to desaturation than adults, significant hypoxemia, bradycardia, acidosis and cardiac arrest.

Pediatric assessment coverage (2):

1. General assessment (pediatric assessment triangle-PAT)
2. Initial assessment (ABCDE's and transport decision)
3. Additional assessment (focus is on history and physical exam; *detailed physical exam if trauma)*
4. Ongoing assessment

Normal respiratory rates by age are:

- infant 30-60 breaths per minute
- toddler 20-30 breaths per minute
- preschool-aged child 20-30 breaths per minute
- school-aged child 20-30 breaths per minute
- adolescent 12-20 breaths per minute

Normal heart rates by age are:

- infant 100-160 beats per minute
- toddler 90-130 beats per minute
- preschool- aged child 80-120 beats per minute
- school-aged child 70-120 beats per minute
- adolescent 70-120 beats per minute

The technique used for rapid stabilization of the airway in trauma depends of the experience of the personnel present and practice patterns at a given institution (1,2,3,5,9). An orotracheal airway is the preferred technique to stabilize the airway before attempts to create a surgical airway (1,2,3). Orotracheal intubation in the pediatric trauma patient either a direct or indirect (video) laryngoscopy is favored in the literature as a safe and rapid technique particularly because emergency department personnel are experienced in and comfortable with this approach (1,2,3,9). If standard orotracheal intubation is not possible, rigid or flexible tracheo/bronchoscop, which are still the most reliable devices to establish the pediatric

airway, should be attempted next (1,2,3,9). It is far safer than intubation, because the airway is directly visualized and secretions and blood can be suctioned through the lumen of the rigid scope (1,2,3,9). It is also the most reliable devices of diagnosis, it allows the patient to be ventilated during the examination, and it helps determine the need for tracheotomy (1,2,3,9). If necessary, tracheotomy can then be performed over the bronchoscope (1,2,3,9). After airway stabilization, other priorities include consideration of other traumatic injuries of internal organs, limbs or spinal cord. A prearranged, logical, step-by-step approach by emergency department personnel toward the management of airway in pediatric patients in trauma is recommended in make efforts to reduce morbidity and mortality.

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14

Invasive Difficult Airway Management – Is There a Reason to Fear for Procedure Performance?

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Abstract: A can't intubate, can't oxygenate (CICO) scenario arises when attempts to manage the airway by tracheal intubation, face mask ventilation, and SAD have failed; it is rare but often poorly managed, with potentially disasters consequences. Emergency cricothyroidotomy is at temporary, life-saving procedures, indicated immediately when airway is obstructed and oxygen delivery is unable to be restore by other means. There are two techniques are used for emergency oxygenation through front-of-neck access: scalpel-bougie-tube or needle techniques .The effectiveness of either techniques as a rescue strategy also depend on a clinicians willingness to implement it. All anesthetists must obtain both theoretical and hands-on competence in all three small bore cannula, large bore cannula and scalpel-based) ways of performing front-of-neck-approach, so that they can choose the technique that suits the clinical situation best.

Key Words: airway, emergency oxygenation, cricothyroidotomia, CICO

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15

Percutaneous tracheotomy- our experiences

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Introduction:
Tracheotomy is a common surgical procedure used with critically ill patients in intensive care units. The morbidity of this procedure varies from 6 - 66% of patients (1). The mortality of this procedure according to literature reports ranges from 0 - 5% (2).
Minimally invasive procedures are widely used in many areas of surgery. Percutaneous tracheotomy (PCT) represents minimal invasive surgery compared to classic tracheotomy. It was first described by Toye and Weinstein in 1969 (3).
Two new methods suitable for performing percutaneous tracheotomy with a patient bed have been introduced based on the Seldinger technique. In 1985, Ciglia with his colleagues introduced the method with different diameter dilators, which is still one of the most popular methods nowadays(4).
Another technique described by Griggs et al. 1990, involves the use of the modified Howard-Kelly forceps as a tracheal dilator (5).

Indications:
In intensive care units, the most common indication for a tracheotomy is the need for longer mechanical ventilation. This need can occur with therapy-resistant pneumonia, severe chronic obstructive pulmonary disease, acute respiratory distress syndrome, severe brain injury, or multiple organ failure. The Critical Care Council, American College of Chest Physicians recommends tracheotomy in patients who expect mechanical ventilation for more than 7 days. The indications for percutaneous tracheotomy (PCT) are the same as those for standard open tracheotomy.
Airway obstruction can also occur due to the following conditions:
inflammatory disease
congenital anomalies (eg pharyngeal hypoplasia)
supraglottic or glottic pathological condition (eg tumors, bilateral paralysis of the vocal cords)
laryngeal trauma or stenosis
facial fractures that can lead to upper airway obstruction
edema (eg trauma, burn, infection, anaphylaxis)
the need for longer mechanical ventilation in cases of respiratory failure
the need to improve the pulmonary toilet
insufficient act of coughing due to chronic pain or weakness
prophylaxis (in preparation for extensive head and neck surgery)
severe sleep apnea

Contraindications:

What represents absolute and relative contraindications has become a matter of debate. Most of the published articles of neck injury, pediatric age, coagulopathies, and the urgent need to establish an airway are considered an absolute contraindication, while short, thick neck or obesity are relative contraindications. However, several reports point to the safety and feasibility of performing PCT in patients with the contraindications previously described. A retrospective study of Blankenship shows that percutaneous tracheotomy can be performed safely in morbidly obese patients in whom neck structures can be identified (6).
In a retrospective study, Gravvanis et al showed that PCT may be safer and faster in burned patients (7). PCT is also safe and feasible in patients with cervical spine fracture, as reported by the 2006 Ben-Nun study (8).
Cornblith et al published a study that included thousands of patients over a 10-year period and came to the conclusion that PCT is a safe procedure with minimal complications, even in the most at-risk patients (9).

Absolute contraindications:

Age of the patient (less than 8 years)
The need for emergency airway recovery
Major changes in neck anatomy due to hematoma, tumor and hyperthyroidism (second or third degree).

Relative contraindications:

obese patient who does not recognize the anatomical structures of the neck
coagulopathy
need for positive end expiratory pressure (PEEP) greater than 20 cmH2O
soft tissue infections of the neck at a future site of surgery

Advantages of PCT over classic tracheotomy:

a relatively simple technique for trained intensive care staff
it does not require an operating room with all the necessary human resources, but is performed on a hospital bed
the formation of a stoma between the tracheal rings reduces the possibility of bleeding both during the procedure itself and after the intervention
percutaneous tracheotomy infections were significantly reduced compared to the classical method (0 - 3.3 vs 36%
decreased incidence of tracheal stenosis
very small postoperative scar

PCT setup complications:

an endotracheal tube cuff may rupture during the procedure, which may lead to consequent hypoxia

pneumothorax and pneumomediastinum are the result of misplacement of the needle or dilator
damage to the posterior wall of the trachea and the possibility of a tracheoesophageal fistula larger bleeds are very rare and smaller ones can be treated with compression or suture

Late complications of PCT are reflected in a very small percentage of clinically significant subglottic stenoses, and the reason for their occurrence is laryngeal edema. In any case, the occurrence of subglottic stenoses is far less with PCT compared to the clasic tracheotomy

Patients and methods:

In the Blue Building Intensive Care Unit of the Anesthesia and Resuscitation Clinic of the Clinical Hospital Center Tuzla, from 2010 to 2012, 40 tracheotomies were performed, 31 of which were percutaneous tracheotomies. The indications for PCT were prolonged intubation and toilets of the tracheobronchal tree in patients with severe brain disease who did not have a purposeful expectoration.
In the intensive care unit, we use the Seldinger technique, which involves a wire guide, a plastic dilator, and a dilating forceps.

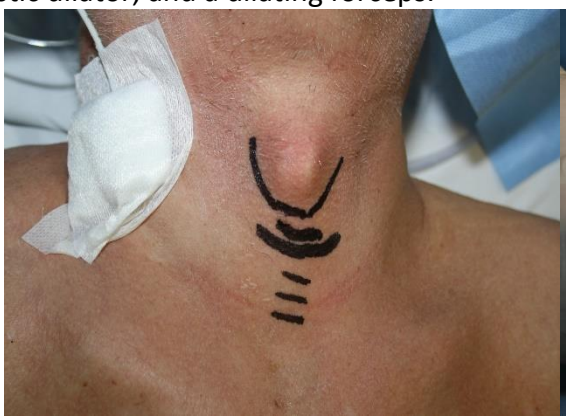

Figure 1.

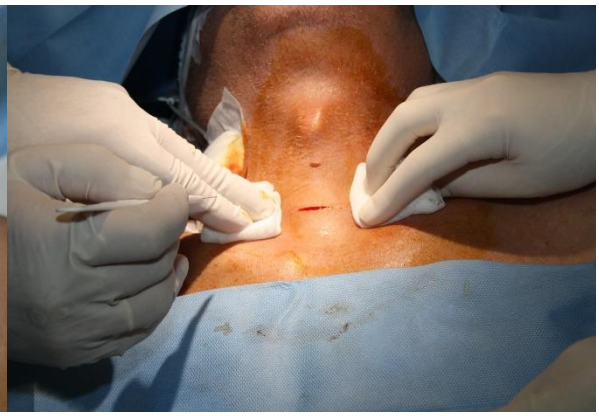

Figure 2.

After identification of the neck structures, a transverse incision is made in the projection between the second and third tracheal rings, a 2 cm incision is made on the skin (Figures 1 and 2).

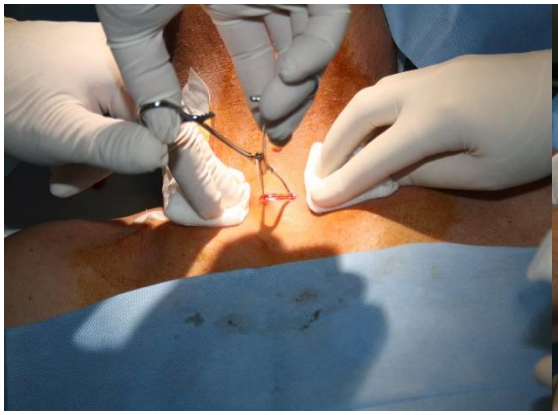

Figure 3.

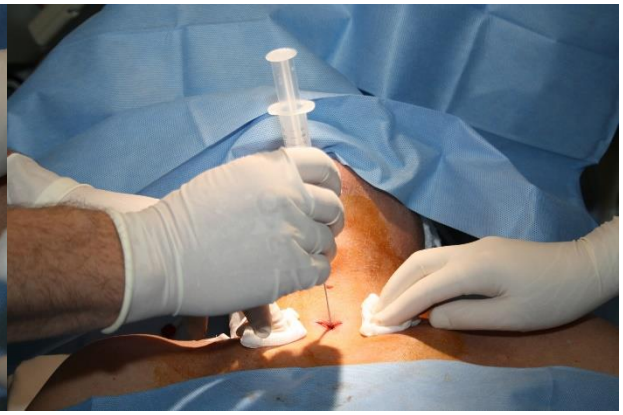

Figure 4.

With a small pen, we gradually dissect the subcutaneous tissue. With a test needle, we identify the position of the trachea and the tube. The original needle from the percutaneous tracheotomy set is then introduced (Figures 3 and 4).

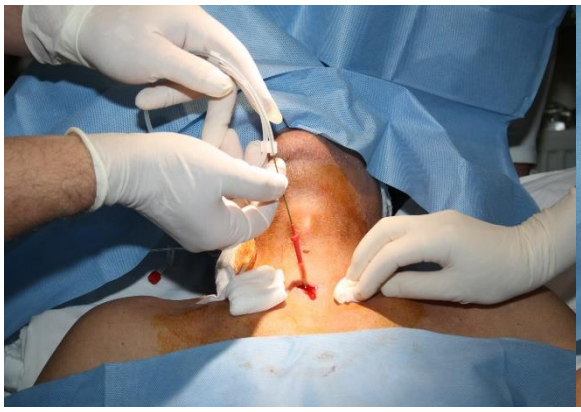

Figure 5.

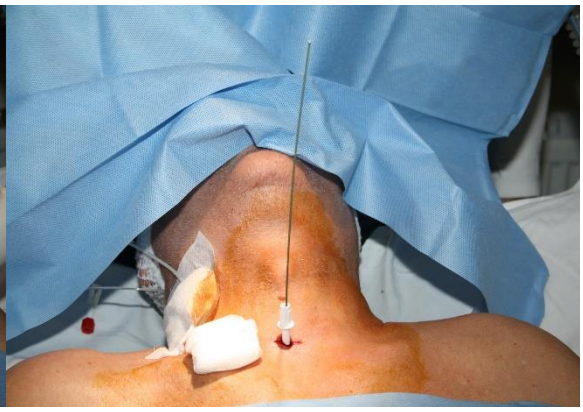

Figure 6.

A wire guide is introduced through the needle. We introduce a plastic dilator through the wire conductor (Figures 5 and 6).

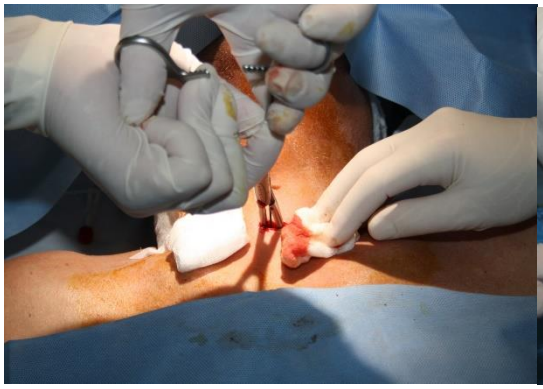

Figure 7.

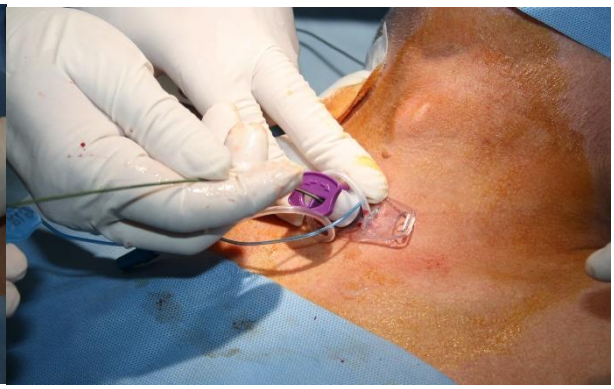

Figure 8.

Then, through the guide, we introduce the dilating forceps. When the trachea has been dilated sufficiently, a cannula is placed over the wire guide (Figures 7 and 8).

The results:

Intraoperative complications of PCT occurred in two cases. It was minor bleeding that was resolved by short-term digital compression. The other intraoperative and early postoperative complications described were not present. Unfortunately, we could not evaluate the late complications in ten patients because they had a lethal outcome due to the severity of the underlying disease. Other patients were decanilized after ten to twenty days. No patients were observed signs of tracheal stenosis. Tracheostomy opening infection was not recorded in any case. All tracheostomy openings healed very quickly per secundam, with an incredibly small scar.

Conclusion:

Percutaneous tracheotomy is the method of choice for critically ill patients in intensive care units. If the right patients are selected, they abide by the basic rules of antisepsis and train intensive care staff PCT is a method that with minimal trauma provides optimal treatment for the patient.

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16

Chest Trauma and MechanIcal VentIlatIon

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Thoracic trauma is among the causes of every four deaths from trauma. Blunt trauma is more common than penetrating trauma but both mechanisms of injury may detoriate respiratory function by effects on the chest wall, airways or lung parenchyma by contusion, fractures, rupture/penetration, thermal or chemical inhalational injury. Intrapulmonary injury due to blast, thermal or chemical injury may rapidly progess to Acute Lung Injury (ALI) or Acute Respiratory Distress Syndrome (ARDS). Haemorrhage into alveoli and parenchymal damage are maximal in the first 24 hours after injury and hypoxaemia and hypercapnia peak at about 72 hours.

Although thoracic trauma is relatively common, the incidence of isolated chest injury requiring thoracotomy is small. About 18% of patients require intercostals drains and about 2.5% need thoracotomy. Overall mortality is around 9% with low Glasgow Coma Scale score. Older age, presence of penetrating chest injury, long bone fractures, fracture of more than five ribs, and liver and spleen injuries are independent predictors of death in thoracic trauma. The management and care of thoracic trauma patients needs the close association of the thoracic surgeon and anesthesiologist. The surgeon and anesthesiologist share a “thoracic workspace” - the surgeon operating on vital thoracic structures and the anesthesiologist managing ventilation, oxygenation, and hemodynamics.

The principles of management of thoracic trauma are summarized below:

- Assessment and resuscitation: patient should be scaled on injury severity score (ISS). Any ISS more than 25 is severe.
- Physical examination (involving one side of chest or transmediastinal gunshot wound)
- Diagnostic studies
- Life-saving surgery

Anesthesiologists have to know a complete medical history and detailed physical examination of the thoracic trauma patients prior to surgery. The physical examination must be directed primarily to the cardiovascular and pulmonary systems. The presence of wheezing, rales, rhonchi, or other abnormal breath sounds suggests the need for further medical intervention. Any deviation of the trachea from the midline should alert the anesthesiologist to a potentially difficult intubation or to the possibility of airway obstruction during induction of anesthesia. The immediate management of thoracic trauma should follow standard Advanced Trauma Life Support guidelines aimed at identifying the most life threatening injuries in the order of threat to life. Although all victims of thoracic trauma should receive high concentrations of oxygen, Continuous Positive Airways Pressure (CPAP) may be useful if hypoxaemia persists, CPAP will cause surgical emphysema in patients with any loss of integrity of the airway.

In patients with blunt thoracic trauma, the common associated injuries include flail chest (12.5%-23.1%), pulmonary contusion (30%-35%), and pneumothorax (18%-40%).There are 12 lethal or potentially lethal thoracic injuries:

- Airway Obstruction
- Tension Pneumothorax
- Open Pneumothorax
- Massive Haemothorax
- Flail Chest
- Cardiac Tamponade
- Traumatic Aortic disruption
- Tracheobronchial rupture
- Myocardial contusion
- Diaphragmatic rupture
- Oesophageal rupture
- Pulmonary contusion

Lung protective strategies in thoracic injury: Anesthesiologists should be aware of lung protection during the care of thoracic trauma patients. These are mentioned as:

-*Lung Protective Ventilation*: A reduction in volutrauma can be achieved by the use of low tidal volumes and low peak inspiratory pressures. Classically relatively large tidal volumes (10-12 ml/kg) have been used to ensure normocapnia. Reducing the tidal volume to 6 - 8 ml/kg and limiting peak inspiratory pressure, results in a decreased incidence of overdistension and a reduction in trans-alveolar pressure.

-*Intraoperative lung recruitment*: Extrinsic PEEP will hold alveoli open, stop them collapsing totally at end expiration and limit shear forces. The level of PEEP applied to prevent collapse and recruit un-opened alveoli is difficult to calculate on an individual basis.

One Lung Anaesthesia and Alternative Techniques:

One lung anaesthesia (OLA) is a technique to collapse one lung to allow access to other structures within the chest such as trachea, oesophagus, major vessels or heart, to isolate a bleeding lung or to isolate a bronchial tear. Isolation of one lung may be achieved either with a double lumen tube or a bronchial blocker. A double-lumen endotracheal tube (DLT), most frequently left-sided one, is often used for lung isolation. Right-sided DLTs are more difficult to insert and require special care to keep the right upper lobe continuously patent. Because only 20% to 48% of DLTs are in the correct position after they are inserted, it is mandatory to confirm their anatomical and functional position. Specific problems encountered during double lumen tube placement in thoracic trauma include:

- Presence of cervical spine immobilization
- Presence of trauma to upper airway, jaw and mandible
- Bleeding into the trachea/bronchi may lead to poor views on bronchoscopy
- Need for right sided tubes in some conditions e.g. left tracheobronchial injury
- Concurrent pulmonary contusion worsening intra-pulmonary shunt during OLA

Other methods of lung separation include the use of a bronchial blocker, Univent tube (Fuji Systems, Tokyo, Japan), or an embolectomy catheter. A Univent tube is a single lumen tube in which there is a bronchial blocker housed in a small anterior lumen. It is possible to suction through the internal lumen of the blocker or to apply positive end-expiratory pressure (PEEP) to the collapsed lung. In all of these methods, placement
must be confirmed with bronchoscopy immediately after placement and after any change in patient positioning.

Management of hypoxaemia during OLA in thoracic trauma patients need special attention:

- At initiation of OLA the FiO_2 should be raised at least to 0.5, if not 1.0. This is simple, physiological and effective. The mechanisms diverting blood from the non-dependent lung take time and later in the period of OLA it is possible to reduce the FiO_2.
- Check the position of the DLT to make certain that all bronchopulmonary segments of the dependent lung are being ventilated.

- PEEP to the lower lung isn't reliably effective in thoracic surgery and may lower the PaO_2. Excessive PEEP may produce deleterious increases in pulmonary vascular resistance in the dependent lung.
- CPAP with 100% oxygen to the non-dependent lung is effective but inconvenient to the surgeon particularly in thoracoscopy.
- Manipulation of hypoxic pulmonary vasoconstriction (HPV). HPV response is a function of both alveolar and mixed venous oxygen tensions. When the lung is collapsed during OLA it is suggested that HPV decreases the blood flow to that lung by 50%.

High- frequency jet ventilation (HFJV) and High-frequency oscillation (HFO): These ventilation techniques are useful in specific types of thoracic trauma. HFO uses sub-dead space tidal volumes generated by pistons or electromagnetic diaphragms to produce oscillatory gas flows of between 150 and 3000 breaths per minute. The low volumes and low mean airway pressures are combined with alveolar recruitment regimes to prevent alveolar collapse. The technique is useful in patients with established barotrauma or broncho-pleural fistulae where very low mean airway pressures are advantageous.

Tube thoracostomy: This intervention may be needed for the management of pneumothorax or suspected bronchial or parenchymal injury. Chest tubes should be placed prior to the induction of general anesthesia to prevent a tension pneumothorax. It is important not to kink the tubes during the procedure or during positioning, especially a contralateral tube if the patient is placed in the lateral decubitus position. It is also important to maintain suction on the chest tubes throughout the procedure to ensure lung expansion and blood evacuation.

Analgesia in thoracic trauma: The normal mechanism of breathing requires co-ordinated mechanical movement of the chest wall musculature, ribs, lungs and diaphragm. All of these structures consequently have extensive sensory and motor nerve supply. Pain from injury to any of these structures may result from tissue and nerve damage, abnormal movement, or as the result of the treatment of such injuries by intercostal drain insertion or thoracotomy. Inadequate pain relief has adverse effects particularly in patients having respiratory or cardiac co-morbidity:

• Pain inhibits the patient's ability, or willingness, to take deep breaths, cough or mobilise.
• Decreased functional residual capacity
• Decreased tidal volume
• Decreased alveolar recruitment
• Increased atelectasis, sputum retention and infection.
• Hypoxaemia and hypercapnia
• Tachycardia and hypertension
• Increased oxygen demand, decreased oxygen delivery resulting in myocardial ischaemia and potential for dysrrhythmia.

Complications: Systemic air or gas embolism has been increasingly recognized as a complication of serious chest trauma and often presents with catastrophic circulatory and cerebral events. The classic findings are hemoptysis, sudden cardiac or cerebral dysfunction after initiation of positive pressure ventilation, air in the retinal vessels, and air in arterial aspirations. Diagnostic tools (TEE, Doppler, computed tomography) can detect intracardiac and cerebral air, but they may not be necessary to confirm the diagnosis. Spontaneous ventilation is preferred in any patient at risk for systemic air embolism. When positive pressure is necessary, the injured lung should be isolated. If oxygenation becomes a problem, the lowest possible airway pressure, in a form of continuous positive airway pressure, intermittent positive pressure ventilation, oxygen insufflation, or high-frequency ventilation can be used.

Postoperative care: Anesthesiologists should demonstrate special care postoperatively for respiratory support, fluide replacement and hypothermia.
In conclusion, the thoracic trauma patient are accepted as a challenge similarly to both the surgeon and anesthesiologist. Airway and ventilation management are the predominant problems. It is important to be well prepared and to review the fundamentals of securing an airway in many different traumatic scenarios. The key to a successful care and outcome in thoracic injury is mostly related to good communication between team members

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CARDIOVASCULAR SYSTEM AND TRAUMA

17
Hemodynamic monitoring and inotropic support in trauma patients

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Injuries are estimated to become the number-one cause of death for men and women under the age of 45 by the year 2020. Traumatic injuries kill more than five million people annually. Millions more suffer the physical and psychologic consequences of injury, which have an enormous impact on patients, their families, and society.
Active participation of anesthesiologists in the care of severely injured patients provides the best opportunity for improved outcome. We believe participation should not only include involvement in anesthetic management, but also the initial evaluation, resuscitation, and perioperative care of these patients. In these critical situations, anesthesiologists are often faced with the need to simultaneously address emergent airway management, resuscitation, massive blood loss, acidemia, coagulopathy, hypothermia, and the consequences of damage to various organs. The management of each of these conditions alone can be essential for survival, and their convergence presents a unique situation in which the likelihood of death or a bad outcome is real. Success in this stressful situation requires a sophisticated understanding of basic sciences and expertise in the clinical and technical skills of anesthetic management.

Monitoring of trauma patients during their treatment can be especially challenging. The functioning of even basic monitors may be problematic in settings of significant hypotension which is very often in trauma patients. The decision to use a monitor should be based on a number of factors including accuracy of the generated data, potential complications related to generating the data, clinical relevance of the data and impact of the data on clinical outcome.
Monitoring standards as outlined by the three major professional organizations are remarkably similar, the American Society of Anesthesiologists, the Australian and New Zealand College of Anaesthetists and the Royal College of Anesthetists. These standards are organized into physiologic variables that must be assessed on a regular basis:

1. Oxygenation (pulse oximetry, inhaled/exhaled gas analysis, blood gas analysis)
2. Ventilation (et CO_2 measurement, listening to the patient's breath sounds, monitoring the ventilator)
3. Circulation (ECG, blood pressure, ECHO, PA chateter measurements, urine output)
4. Temperature

Additional requirements include keeping adequate medical records, equipment with functioning alarm systems, and personnel. Proper use of hemodynamic monitoring involves not only the collection of data, but their interpretation in the context of the pathophysiological condition of the observed patient. If used properly, hemodynamic monitoring should improve the quality of treatment of trauma patients.

The goal of hemodynamic monitoring is to evaluate the function of the cardiovascular system and the compliance of the cardiac output with the metabolic needs of the organism, as well as to identify the components of hemodynamics that can be therapeutically affected during the patient's treatment.

What to think about when choosing hemodynamic monitoring?

1.The least invasive monitoring should be used to obtain the necessary data indicating the patient's hemodynamic condition
2. Continuous hemodynamic monitoring is better than intermittent monitoring
3. Active treatment of the patient is better than reactive treatment
4. There are no optimal hemodynamic parameters that are applicable to all patients
5. When interpreting the parameters, the absolute values and trends of the individual monitored parameters should be distinguished
6. The need for different hemodynamic monitoring may vary during the treatment of the patient, but also depends on their availability in the hospital

Hemodynamic monitoring can be non-invasive and invasive monitoring. Of course, implementing invasive monitoring carries with it the risk of complications. Therefore, the anesthesiologist / intensivist should evaluate the usefulness of one of the methods and the possible complications of invasive methods.
The Non-invasive monitoring consists of:
1. ECG: for monitoring heart rate, rhythm and myocardial ischemia
2. Puls oximetry: for SpO_2 and heart rate
3. Non-invasive Blood Pressure Monitoring: systolic, diastolic, mean, pulse paradoxus
4. The central venous pressure as assessed by observation of jugular vein distension and hepatojugular reflex
5. Capnography: presence of end-tidal carbon dioxide (EtCO2) indicates that ventilation and cardiac output are present, crucial in a patient with hemorrhagic shock.
6. Temperature Monitoring: sites for measuring include pulmonary artery, nasopharynx, esophageal, bladder and axillar temperature probe
7. Transesophageal Echocardiography (TEE): this minimally invasive technique can be used as a hemodynamic monitor and/or as a diagnostic device
8. Awareness Monitoring: all trauma patients should be monitored using a bispectral index (BIS) monitor
9. Urine output

The invasive hemodynamic monitoring involves the following parameters:
1. Placement of an arterial catheter for invasive continuous monitoring of blood pressure
2. Placement of central venous catheter for monitoring central venous pressure (CVP), analysis of its wave, $ScvO_2$, $PcvCO_2$
3. Using Pulmonary Artery Catheter (PA) . The information obtained from a PA includes CVP, PA pressure, PA occlusion pressure (“wedge”), cardiac output (CO) using thermodilution, continuous mixed venous oximetry SvO2, mixed venous blood gases (PvO2 and PvCO2) by intermittent sampling
4. The esophageal Doppler monitoring to monitor stroke volume (SV), CO, stroke volume variation (SVV)

To maintain good hemodynamics in trauma patients, among other treatment, we have to use volume - fluids and vasopressors as well as inotropes. The major vasopressors include phenylnephrine, norepinephrine, epinephrine and vasopressin. Dopamine is a vasopressor with inotrope properties that is dose-dependent, while dobutamine and milrinone are pure inotropes. As we know, vasopressors are contraindicated in the early management of hemorrhacig shock due to their deleterious consequences, although they may have a role in resuscitation when vasoplegic shock ensues and blood pressure cannot be maintained by fluids alone. Use of vasopressors is not recommended according to Advanced Trauma Life Support, and their usage remains contorversial with no clear guidelines on the timing, type and dose in hemorrhagic shock. Before we start to use vasopressors in early stage of hemorrhagic shock we have to get answers to many questions, still there is an insufficient clinical evidence to validate early vasopressor use in association with fluid infusion in hemorrhagic shock management: type of vasopressor and the precise timing, the use of norephinephrine advocated by some teams appears reasonable (expert idea), fluid loading is the first step to be considered in the management of hemorrhagic shock. European study conducted to assess the impact of vasopressin infusion as a salvage therapy in pre-hospital hemorrhagic shock that persists despite standard treatment, including a first line vasopressor (Vasopressin In Traumatic Shock (VITRIS) trial, may provide an answer.

Keeping in mind everything mentioned so far, trauma patients present unique challenges to anesthesiologists. Anesthesiologists are involved with trauma patients beginning with airway and shock resuscitation, continuing with intraoperative care during surgery, and extending on to pain management and critical care postoperatively.

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Trauma to the heart and great vessels

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ABSTRACT

Introduction: Thoracic great vessel and cardiac trauma are characterized by anatomic location and mechanism of injury: blunt or penetrating. Both the diagnosis and management of these injuries have evolved from more invasive to less invasive strategies paralleling the advent of sophisticated imaging tools and the development of endovascular therapies. The initial care of the trauma patient with cardiac injuries does not vary from standard Advanced Trauma Life Support (ATLS) protocols.

Penetrating trauma: Penetrating trauma to the heart most frequently occur with trauma to the anterior chest, but should also be suspected with wounds to the upper abdomen, chest, back, and neck. The protocol for treatment of patients with penetrating cardiac trauma can be further subdivided based upon the patient's vital signs upon presentation to the hospital. Unstable patients(systolic blood pressure less than 90 mm Hg) are taken directly to the operating room for exploration. If the diagnosis of penetrating cardiac injury is suspected but not confirmed, a subxiphoid pericardial window should be performed.

Blunt injury: Blunt cardiac injury (BCI) is a spectrum of traumatic heart diseases with severity that can range from myocardial contusion and EKG changes to septal rupture and death. The following mechanisms of injury may result in BCI: direct precordial impact, a crush injury between the sternum and spine, a deceleration injury causing injury from the fixation points of the aorta and vena cava, a hydraulic effect from an intraabdominal injury that sends force to the great vessels and heart, or a crush injury. Contusion is the most common type of injury with left atrial chamber rupture being least common.

Iatrogenic injury: As the fields of interventional and electrophysiology cardiology continue to increase the number of percutaneous procedures performed, there is a concomitant increase iniatrogenic injuries to the heart. Pacemaker and ICD placement, ASD occlusion devices, coronary catheterization, pericardiocentesis, and even central line placement can cause cardiac trauma. Fortunately these are rare complications but the incidence of iatrogenic injury has been reported as high as 6% for certain radiofrequency ablation procedures. Awareness and prompt recognition of an injury are essential to successful treatment.

Aortic trauma: The aortic injury is one of the most time-sensitive, life-threatening conditions , second only to head injury as a cause of death. Some predisposing factors for traumatic aortic injury include: penetrating chest injuries, deceleration injuries and blunt chest trauma. Hypotension, external evidence of trauma and altered mental status are common. The most common sites of injury are: aortic isthmus, distal to the origin of the left subclavian artery, ascending aorta and tethered site of the aorta. Traumatic aortic rupture (transection) is frequently fatal due to the profuse bleeding, this can quickly result in shock and death.

Injury of the pulmonary artery: Transection or rupture of even a small branch can cause fatal exsanguination in seconds, and yet it can also be easily controlled with gentle compression. Both of these characteristics are due to the low pressure in the PA circulation and to the thin,

compliant nature of the wall of the PA. A pseudoaneurysm may form when reepithelialization of the perforation does not occur, and delayed rupture can occur even months later. The most common cause of PA ruptures and pseudoaneurysms is PA catheters. Traumatic PA dissections (PADs) have also been described. The mechanism is likely similar to that seen in the aorta as a result of shearing forces and differential deceleration of the mediastinum and the spine.

Management: Since blunt cardiac injury describes a spectrum of disease states, the treatment depends on the actual problem. The primary priority is ensuring the patency of the airway and establishing adequate oxygenation and ventilation. This may include tube thoracostomy for drainage of hemothorax from the pleural space to allow re-expansion of the lung. Subsequently, the circulatory system is assessed. Priority is given to establishing intravenous access for the administration of crystalloid and/or blood products. If cardiac tamponade is suspected, this should be confirmed with sonographic confirmation of hemopericardium and/or right ventricular collapse during diastole. If tamponade physiology is present, treatment for immediate drainage of the pericardial space should be initiated. This can be accomplished percutaneously by pericardiocentesis or via open pericardial window. Arrhythmia can be managed medically with the caveat that anticoagulation needs to be used cautiously in trauma patients. Hemopericardium can be seen with or without hypotension or tamponade. If the patient is hypotensive and tamponade is expected then either a subxiphoid pericardial window or a thoracotomy can be performed. Traumatic aortic rupture is treated with surgery. Less ivasive option for treatment is endovascular repair. The patient should be closely monitored in an ICU setting. Management of traumatic PA injuries includes surgical repair, endovascular approaches, and observation. There are no strict guidelines, and the preferred approach depends on the lesion, the patient, and the institution.

Traumatic Cardiac Arrest: Most deaths due to trauma occur in the first five minutes following the actual traumatic event, and most of these deaths cannot be prevented, even with skilled and timely treatment. The common causes of preventable early death in trauma are: 60% haemorrhage, 33% tension pneumothorax, 10% cardiac tamponade, 7% airway obstruction. Immediate cardiopulmonary resuscitation (CPR) and Adrenaline do not increase survival in traumatic cardiac arrest and may hinder evidence-based treatments, so in this context they are considered secondary priorities. Trauma arrest is associated with a worse prognosis, however several recent studies have shown a 5.1 to 7.5% survival to discharge rate, though the neurological recovery varied (2% to 6.6%).

Anesthetic management of thoracic trauma: Thoracic injuries are dynamic. It is crucial for the anesthesiologist to continually reassess the patient, so that the manifestations of evolving injuries may be detected as early as possible and appropriate management decisions made. Up-to-date knowledge of injury patterns, mechanisms, pathophysiology, and operative and nonoperative management will facilitate optimal management of these patients. Initial resuscitation and surgical management of patients with thoracic trauma continue to evolve. Improvements in prehospital care and diagnostic techniques as well as development of minimally invasive interventions mean that the anesthesiologist may be required to provide care to unstable patients in an expanded range of scenarios and environments. Anesthetic management needs to address different clinical topics: management of diffcult airways, intrincis effects of anesthetics and mechanical ventilation on respiratory and cardiac function, the adequate replacement of blood loss. Interdisciplinary cooperation during diagnostic, treatment and in the perioperative course is a prerequisite for a successful management.

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Pulmonary embolism after trauma

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Pulmonary embolism (PE) is a potentially fatal complication after trauma. It is cause significant morbidity and mortality after injury and patients with pelvic or lower extremity fracture, severe head injury, or spinal cord injury are at greatest risk. Risk factors include thrombosis after endothelial damage and blood stasis, which may be caused by extended periods of immobility. Both conditions are associated with physical trauma.

Clinical presentation

The clinical presentation is variable and, depending on the extent of vessel obstruction, can range from asymptomatic to cardiogenic shock. Symptoms are often nonspecific, including chest pain (~ 50% of cases), worse with inspiration, coughing, dyspnea (> 50% of cases), tachycardia (~ 25% of cases), hypotension. Syncope and shock with circulatory collapse are possible in massive PE

Pathophysiology

Pathophysiologic response of the lung to arterial obstruction are:

1. Infarction and inflammation of the lungs and pleura causes pleuritic, chest pain and hemoptysis, leads to surfactant dysfunction → atelectasis → ↓ PaO2 and triggers respiratory drive → hyperventilation and tachypnea → respiratory alkalosis with hypocapnia (↓ PaCO2)

2.Impaired gas exchange lead mechanical vessel obstruction → ventilation-perfusion mismatch → arterial hypoxemia (↓ PaO2) and elevated A-a gradient

3.Cardiac compromise: elevated pulmonary artery pressure (PAP) due to blockage → right ventricular pressure overload → forward failure with decreased cardiac output → hypotension and tachycardia

Diagnosis

The diagnosis of PE is based primarily on the clinical findings and is confirmed by detection of an embolism in contrast CT pulmonary angiography (CTA). Arterial blood gas analysis typically shows evidence of respiratory alkalosis with low partial oxygen pressure, low partial carbon dioxide pressure, and elevated pH. Another commonly performed test is the measurement of D-dimer levels.

Wells criteria for pulmonary embolism

The Wells score is used as a diagnostic algorithm in stable patients for assessing the probability of PE.

Blood analysis

- Initial test: measure D-dimer levels (if suspicion for PE low)
    - D-dimers: fibrin degradation products detected in the blood after thrombus resolution via fibrinolysis;
    - ↑ troponin T and B-type natriuretic peptide (BNP): possible elevation from right ventricular pressure overload → poor prognosis
- Arterial blood gas (ABG) test

- Respiratory alkalosis : ↓ paO2 < 80 mmHg, ↓ paCO2, ↑ pH
- ↑ Alveolar-arterial (A-a) gradient : compares the oxygenation status of alveoli to arterial blood
- ↓ O2 saturation

A positive D-dimer is unspecific since it may be elevated anytime elevated fibrinolysis is occurring.

Imaging

Helical spiral CT/CT pulmonary angiography (CTPA): best definitive diagnostic test with contrast-enhanced imaging of the pulmonary arteries.

Chest radiography may indicate PE with atelectasis (visible collapse or incomplete expansion of the lung), pleural effusions and cardiomegaly.

Echocardiography: to detect right atrium pressure (RAP) signs. Venous reflux with dilation of inferior vena cava (also liver congestion in ultrasound of the abdomen), tricuspid regurgitation (tricuspid valve insufficiency), increase pulmonary artery systolic pressure and dilatation and hypokinesis of the right ventricle.

Ventilation/perfusion scintigraphy : Indication: alternative to CT angiography in patients with severe renal insufficiency or contrast allergy. Method: detects areas of ventilation/perfusion (V/Q) mismatch via perfusion and ventilation scintigraphy, Assessment: perfusion failure in normally ventilated affected pulmonary area (mismatch) suggests PE. Evidence of normal lung perfusion rules out PE → ventilation scintigraphy superfluous

Treatment

Initial management according to modified Wells criteria

Hemodynamically stable patients (systolic BP > 90 mmHg) with high probability of PE (Wells score > 4) → CTA for definitive diagnosis. Unless strongly contraindicated (e.g., high risk of bleeding, recent surgery), it has to start empiric anticoagulation before conducting a CTA.

If too unstable for CTA → it should perform bedside echocardiography obtain a presumptive diagnosis of PE (right ventricle enlargement/hypokinesis or visualization of clot) prior to empiric thrombolysis.

In patients with a low or medium probability of PE (Wells score ≤ 4) → measure D-dimer levels (+ ABG evaluation + CXR).

If positive (D-dimers ≥ 500 ng/mL) → CTA → evidence/exclusion of PE

If negative → PE unlikely → consider other causes of symptoms

General measures are: 45° reclining sitting posture, oxygen supplementation and intubation if respiratory failure, iv fluids and/or vasopressors in patients with hypotension and analgesics and sedatives.

Respiratory support — Supplemental oxygen should be administered to target an oxygen saturation ≥90 percent. Severe hypoxemia, hemodynamic collapse, or respiratory failure should prompt consideration of intubation and mechanical ventilation.

Hemodynamic support — The precise threshold that warrants hemodynamic support depends upon the patient's baseline blood pressure and whether there is clinical evidence of hypoperfusion. In general, small volumes of intravenous fluid (IVF), usually 500 to 1000 mL of normal saline, followed by vasopressor therapy should be administred.

Intravenous fluid – IVF is first-line therapy for patients with hypotension. However, in patients with right ventricular (RV) dysfunction, limited data suggest that aggressive fluid resuscitation is not beneficial, and may be harmful. The small volumes of IVF increase the cardiac index in

patients with PE, while excessive amounts of IVF result in RV overstretch (ie, RV overload), RV ischemia, and worsening RV failure.
Vasopressors – Intravenous vasopressors are administered when adequate perfusion is not restored with IVF. The optimal vasopressor for patients with shock due to acute PE is dobutamin and norepinephrine.

Non-life-threatening pulmonary embolism: therapeutic anticoagulation

- Empiric anticoagulation in patients with no absolute contraindication until definitive diagnosis has been made

1. **Initial anticoagulation (0–10 days)**
    - Low molecular weight heparin (LMWH) in stable patients without renal insufficiency, especially in cancer patients
    - Unfractionated heparin (UFH) in patients with renal failure and those who may still require thrombolysis

2. **Long-term anticoagulation and prophylaxis (3–6 months)**

Anticoagulation treatment is indicated for a minimum of three months after PE. The following agents may be used: Warfarin (target INR 2–3), LMWH and direct oral anticoagulants
Patients with a hypercoagulable state with DVT or PE: heparin followed by 3–6 months of warfarin for the first event, 6–12 months for the second, and lifelong anticoagulation for further events.

Massive, life-threatening pulmonary embolism: recanalization

- **Embolectomy**

Surgical embolectomy — The usual indication for surgical embolectomy is hemodynamic instability due to acute PE for patients in whom thrombolysis (systemic or catheter-directed) is contraindicated. Surgical embolectomy is typically limited to large medical centers because an experienced surgeon and cardiopulmonary bypass are required. It has a high mortality, particularly in the elderly (2 to 46 percent).

- **Inferior vena cava filter-** Medical device that is implanted into the inferior vena cava during a catheter intervention to prevent life-threatening PE. The device can trap large emboli and prevent them from traveling further to the heart and lungs.
- The IVC filters are, however, expensive, invasive, and associated with some complications. including erosion of the IVC, inducing thrombosis either above or below the filter, migration of the filter to the right atrium, and tilting or mal-positioning of the filter resulting in ineffective filtering of emboli and fatal PE.

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Vasopressin in sepsis

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Vasopressin has two primary functions. First, it increases the amount of solute-free water reabsorbed back into the circulation from kidney tubules of the nephrons. Second, Vasopressin constricts arterioles, which increases peripheral vascular resistance and raises arterial blood pressure. Vasopressin may play an important role in social behavior, sexual motivation, and maternal responses to stress.

In septic shock, norepinephrine is the most commonly used vasoconstrictor. Vasopressin infusions are also used as second line therapy for septic shock patients who are not responding to fluid resuscitation or infusions of catecholamines (e.g., dopamine or norepinephrine). Unfortunately, cardiac and vascular smooth muscle can become resistant, requiring increasing doses of norepinephrine. This produces adverse effects which include increasing tissue oxygen demand, reducing renal and mesenteric blood flow, pulmonary hypertension, and arrhythmias. In sepsis, there is an increased sensitivity to vasopressin. Vasopressin and norepinephrine are believed to have a synergistic action when used together.

The use of vasopressin is not without side-effects. Myocardial and splanchnic ischaemia may occur, but this effect is limited by avoiding high doses. However, in the literature, a dose range of 0.01–0.04 IU min^{-1} is commonly used to replace falling vasopressin levels. It is usually started when increasing norepinephrine doses are being used to maintain arterial pressure. The most common side effects during treatment with vasopressin are dizziness, angina, chest pain, abdominal cramps, nausea, vomiting, fever, water intoxication, pounding sensation in the head, diarrhea, sweating, and flatulence.

SUMMARY: There is growing evidence that vasopressin infusion in septic shock is safe and effective. Several studies published this year support the hypothesis that vasopressin should be used as a continuous low-dose infusion (between 0.01 and 0.04 U/min in adults) and not titrated as a single vasopressor agent.

SPECIFIC ENTITIES

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Trauma in pregnancy

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Occurrence of trauma in pregnancy is unknown, but it is estimated to complicate relatively high number of pregnancies (6-7%), causing significant foetal mortality and maternal morbidity. Trauma is considered as the leading nonobstetrical cause of maternal death worldwide. (1) Foetal status can be deteriorated following maternal trauma severely, mostly as a result of placental abruption. In some cases, premature rupture of membranes, preterm birth (PTB), spontaneous abortion and stillbirth, have been reported. (2) Therefore, injured pregnant patient should be evaluated by multidisciplinary team in order to provide the best possible outcome for both mother and fetus. Pregnancy influences significant physiologic and anatomic changes in the female organs and systems and it should be taken into account during the evaluation and interpretation of the diagnostic tests in traumatized pregnant patient.

The primary management goal is to stabilize the condition of the mother, as fetal outcomes are directly correlated with an early maternal resuscitation. Pregnant women (≥ 20 weeks' gestation), should be transported to a center that is capable of undertaking a timely and thorough trauma evaluation and treatment, including the delivery of the baby. Diagnostic radiologic imaging in pregnant trauma patients should be performed, if clinically indicated and not be withheld because of the fear of fetal effects. The most applied examinations in pregnancy include ultrasound, CT, and MRI. In the pregnant trauma patient, ultrasound is often easily accessible in emergency department and can provide important information regarding the fetal status, such as viability, gestational age, and potential placental abruption.

Perimortem cesarean section, defined as a cesarean section performed during maternal cardiac arrest and resuscitation, can be life-saving for both mother and fetus. (3) Cesarean delivery should be performed 4 minutes after properly performed cardiopulmonary resuscitation without success to restore circulation.

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Anesthesia in Major Pediatric Trauma

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Trauma remains the number one pediatric public health problem worldwide. According to the World Health Organization (WHO) reports almost 1 million children deaths occur each year from trauma. About 95% of these deaths occur in low and middle-income countries (LMICs). Pedestrian and bicycle accidents, falls, burns, and physical assault are the most common causes of injury in children. Child abuse, ongoing wars and disasters are also responsible for a significant number of childhood injuries. Anesthesia providers are part of the trauma teams in major trauma centers and often the main care taker perioperatively in smaller hospitals. Anesthesiologists may have to provide help for initial stabilization in the emergency department, anesthesia for imaging and surgical procedures, intensive care unit management and postoperative pain control. Anesthesia providers should be familiar with the principles of management of pediatric trauma as well as with age-related specific anatomical and physiological aspects of trauma care.

The major differences in pediatric and adult injuries are as follow. In children most injuries result from blunt trauma. Head injury is the most common cause of death and associated hypotension is uncommon (excluding infants). infants have additional protective areas (open fontanelles) to allow expanding intracranial masses. Rib fractures are less likely to occur due to incomplete calcification; therefore, significant thoracic force can be transmitted, resulting in a pulmonary contusion or tension pneumothorax. Psychological ramifications in the patient and family should be strongly considered. Significant blood loss can occur (30% of total blood volume) before hypotension may manifest. Pseudosubluxation of the cervical spine can be difficult to distinguish from a true injury. Ligamentous injuries to the spine occur more commonly in children and can only be definitively diagnosed by MRI.

Management of specific injuries

Traumatic Brain Injuries

Traumatic brain injuries (TBIs) are the major cause of death and morbidity in children. They affect over 3 million children annually, impacting every population and demographic group. Although falls are the leading cause of TBI when considering all children from 0 to 14 years of age, there are etiological variations. Motor vehicle accidents and assault become more prevalent with increasing age. Nonaccidental trauma (shaken baby syndrome or child abuse) should always be in the differential diagnosis in infants and young children. General anesthesia may be required for skull fracture, evacuation of hematoma, decompressive cranietomy or insertion of intracranial pressure monitoring. The mortality rate for TBIs patients in LMICs is

twice that in high income countries.The subsequent sequelae of TBI involve both primary and secondary injuries to the brain. Primary injury is caused by the initial trauma. It may result in skull fracture, brain contusion, intracranial (intraparenchymal, epidural, and subdural) hematoma, or diffuse axonal injury. The two major factors that result in secondary injury in patients with TBI are hypotension and hypoxemia.

The primary goals of anesthetic management are same as adults and can be summarized as follow 1) provide adequate anesthesia and analgesia. The Guidelines for the Acute Medical Management of Severe Traumatic Brain Injury in Infants, Children, and Adolescents 2012 highlight that choice and dosing of analgesics, sedatives, and neuro- muscular-blocking agents are left to the discretion of the treating physician. There are no evidence-based data to demonstrate a significant difference in outcome when comparing TIVA with inhalational agents. Avoid nitrous oxide which increases $CMRO_2$; 2) optimize surgical conditions with Neuromuscular blocking agents. Although concerns of undiagnosed myopathies leading to hyperkalemic cardiac arrest, and fasciculations and increased ICP exist, succinylcholine may be used; 3) avoid secondary insults including hypotension, hypoxemia, hypocarbia, hypercarbia, hypoglycemia, and hyperglycemia.; Maintain PaO_2 above 60 mmHg , PEEP may increase ICP, aim for PCO2 of 35-45 mmHg. Hyperventilation should only be used if impending herniation; 4) Monitor ICP, maintain an adequate CPP at >40 mmHg and avoid increases in ICP. ICP above 20 mmHg warrants intervention. Hyperosmolar therapy, with mannitol or hypertonic saline, is effective for intracranial hypertension (ICH). Possible concerns are natriuresis, dehydration, central pontine myelinolysis, and rebound in ICP. Aim for euvolemia. Normal saline should be used as maintenance fluid. Glucose containing fluids may be infused if the serum glucose is below 70 mg/dl. Maintain and monitor normothermia. There are potential adverse effects with induced hypothermia, including hypotension, bradycardia, arrhythmias, sepsis, and coagulopathy. Steroids do not provide benefit in TBI patients and may be harmful. Different from adults, DIC is relatively common in children with severe head injury, as the brain is particularly rich in thromboplastin. Monitor closely. The 2012 Guidelines recommend barbiturate therapy in hemodynamically stable patients when maximal medical and surgical therapy has failed to control ICP. Beware of reductions in blood pressure and cardiac output, as well as increased intrapulmonary shunts. When barbiturate therapy is used, close monitoring and cardiovascular support is necessary. The 2012 Guidelines recommend consideration of EVD to manage ICP CSF drainage via EVD resulted in ICP control in 87% of pediatric patients. Prophylactic anticonvulsant therapy should be given for the first 7 days posttrauma.

Cervical spine injury precautions

Children with TBI may also have cervical spine injury. younger children have injuries to the upper cervical spine, while older children have lower ones. Clearance requires radiographic assessment and a neurologic examination as well (7). In infants under 6 months of age, the head and cervi- cal spine should be immobilized using a spine board with tape across the forehead and blankets of towels around the neck. In infants greater than 6 months of age, the head should be immobilized in either the manner described above or using a small rigid cervical collar. Rigid cervical collar use is essential as it prevents cervi- cal distraction whereas soft collars can permit 5–7 mm distraction of cervical spine during laryngoscopy (8).

Resistance to using succinylcholine in pediatric TBI due to but these concerns must be weighed against the risks posed by a full stomach in trauma patients. Therefore, succinylcholine is not con- traindicated and is probably safer than high-dose rocuronium when there is concern for a difficult airway.

Soft Tissue Injuries

Soft tissue facial injuries are common in pediatric patients due to the prominence and relative size of the head in young children. Facial fractures are less frequent because of the elastic nature of the craniofacial skeleton in the pediatric population. A combination of sedation and local anesthesia can be useful when suturing facial lacerations in children. Midazolam syrup is helpful even when used in subtherapeutic doses. The addition of inhalational agents, such as nitrous oxide and/or sevoflurane, alone or in combination with oral agents can be helpful adjuncts during stimulating portions of the surgical repair, such as administration of local anesthesia or tissue advancement under tension. Intravenous benzodiazepines, opioids, or general anesthetic agents, such as propofol, can be used in an infusion or small bolus doses to deepen the sedation at appropriate points during the surgical repair. This advanced level of sedation necessitates increased levels of monitoring and airway support as well as increased support staff. Consideration should be made to complete more complex procedures in an operating room environment depending on the training and level of comfort of the surgical and the anesthesia support teams.

Abdominal Injuries

Abdominal injuries (AI) in children are frequent in motor vehicle-related crashes and falls. Clinical signs and symptoms concerning for AI include abdominal tenderness and distention, absent bowel sounds and peritoneal signs. Solid organ laceration or perforation may need surgical exploration. Anesthetic management may require meticulous hemodynamic monitoring and balanced fluid and blood replacement.

Thoracic Injuries

The majority are blunt thoracic injuries resulting from RTAs. Penetrating trauma as a result of gunshot wounds and other projectiles is less common. Young children have more compliant, cartilaginous chest walls and fractures are rare. However, associated pulmonary and cardiac contusions, pneumothoraces, hemothoraces and mediastinal injuries should be considered in children with blunt chest trauma.

Orthopedic Injuries

The causes of these injuries include falls, RTAs, workplace accidents, child abuse, and injuries sustained in conflicts or other disasters. Fractures in pediatric patients are distinct from those in adult patients. Young children have a growth plate, and physeal fractures represent ~18% of fractures. Four distinct types of fractures seen in children include plastic deformity, torus fractures, greenstick fractures, and physeal fractures. Peripheral nerve blocks together with general anesthesia provides better postoperative analgesia.

Some special considerations for traumatized children:

- A child with a Glasgow Coma Scale (GCS) score of <9 should have a definitive airway established (9,10).
- The pediatric trauma victims should always be considered as full stomach patients, and rapid sequence induction is preferred. The classic rapid sequence induction is difficult to perform in children. Although succinylcholine is associated with many unwanted effects, it should be considered for emergency intubation in children with full stomach.
- High volume, low pressure cuffs present a number of advantages: low fresh gas flow, reduced gas pollution, a decrease in repeated laryngoscopies, considerably reduced aspiration risk, and improved end-tidal gas monitoring.
- In infants, when blood loss approaches 50-75% of the preoperative volume (40-60 mL/kg), coagulative abnormalities are likely to appear.
- Age-appropriate equipment, diluted medicine in age-appropriate doses, and an ambient temperature of 26°C for infants and small children are required in the theatre.3
- Rapid-infusion devices, fluid warmers and infusion pumps should be available.
- Standard anterior-posterior chest X-rays is a cost-effective screening tool that will reveal most of the thoracic abnormalities.4
- From the specific testing, type and cross match blood, and hematocrit are indicated for a hemodynamically unstable patient. Serial hematocrits may help in the monitoring of solid organ injuries. Since coagulopathy is associated with trauma in general and with head injuries specifically, PT, PTT, and INR are useful tests in critically injured patients.
- Serial arterial blood gas testing is invaluable in assessing dynamic changes in hematocrit, oxygenation and acid-base status in critically ill children.
- A delay in fluid resuscitation may lead to a significant hypovolemia fast.
- All intravenous fluids and blood should be warmed to 37° to prevent hypothermia.

RESUSCITATION

Lastly, pediatric anesthesiologists also need to keep abreast of advances in acute pediatric resuscitation. Although intraoperative cardiac arrest is rare, emergency surgery has a higher risk for cardiac arrest. Anesthesiologists may also be involved in resuscitation outside the operating room.

the guidelines published by the International Liaison Committee on Resuscitation. the key points in pediatric resuscitation as follows:

1. Do not delay resuscitation while feeling for a pulse
2. Start chest compression in the presence of bradycardia before the pulseless state; a change of priority from airway-breathing-circulation to compression-airway-breathing
3. Use a compression to ventilation ratio of 30 : 2 for a sole rescuer but 15 : 2 when two rescuers are present
4. Use a compression rate of 100 per minute for all ages, pushing down 4cm for infants and 5cm for children, and minimizing interruption of compression
5. Aim for an end-tidal CO_2 of more than 10– 15 mmHg
6. If intubated, ventilate at 8–10 breaths per minute and do not pause compression for ventilation

7. If intravenous access is not available, establish intraosseous access rather than deliver drugs intratracheally
8. Give epinephrine 0.01 mg/kg, repeat if needed
9. Defibrillate with 4J/kg if initial 2J/kg is ineffective
10. If available, consider and organize extractor poreal life support early.

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Eras protocol for cesarean delivery (erac- enhanced recovery after cesarean) and implementation in serbian university hospital

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Introduction: Enhanced Recovery After Surgery (ERAS) is a protocol which has been established for more than 20 years in some Scandinavian countries, predominantly in colorectal surgery (1). Following colorectal surgery, urology and orthopedics, gynecology oncology was next for implementation of ERAS (2). In recent years some hospitals in the United Kingdom (UK) started the program with ERAS protocol for cesarean delivery. This program includes active participation of: surgeons (obstetricians), anesthesiologists, nurses, pediatricians and for the first time: PATIENTS! The whole medical team is involved in creating these protocols and implementing it in the hospital practice. The most important member of the team is a manager (3). The whole process starts in the obstetrics office and anesthesia pre-assessment clinic with oral and written information for patients. This process always starts preoperatively, continuous during surgery and finishes on patient's discharge home. Using ERAC protocol (Enhanced Recovery After Cesarean) showed that there was no increase in number of complications after discharge patient home, infections and readmissions to the hospital. First results were published and are encouraging for ERAC (4). ERAS is consisting of three parts: preoperative, intraoperative and postoperative part. The whole process starts in the moment when decision of surgery is made, where surgeon introduces ERAS to the patient, gives the patient written information and signs in the medical record that the patient will be in ERAS group. An anesthesiologist has to optimize patient's condition, give the information about preoperative fasting, what to expect following the surgery and about pain relief, IV lines, catheters, oral feeding, starting ambulation and about planning discharge home. In the written information paper patient must know what to do or who to call in case of some complications, which are in connection with mother or baby.

Methods: After Ethical Committee approval, we conducted two months electronic survey. Overall responses were recorded and the responses between those hospitals with ERAC protocols in place were compared to those who did not report having them. Actually, there is no one real ERAC protocol in Serbia, bur in many hospitals many of ERAS elements are in use. Pearson' chi square test was used where appropriate for comparisons between groups. (R version 3.3.3, R Core Team, R Foundation for Statistical Computing). Differences of $P \leq 0.05$ were considered significant.

Results: Responses were obtained from 46 of 49 hospitals. Results showed no one in Serbia has ERAC protocol, but in 24% of the hospitals some ERAS elements are implemented in everyday practice. More than 80% of patients for scheduled CD are admitted to the hospital the day before the CD, and 87% of patients have mechanical bowel preparation and DVT prophylaxis. Antibiotics prophylaxis is present in 73% of hospitals with some ERAS elements.

The use of NA for both elective and emergency CD was greater in the ERAS group. PO intake was allowed in over 90% of patients in the ERAS group within 12 hours of delivery, but 90% did not have solid food until the second day. 1/3 of ERAS group patients were discharged within 3 days of delivery. In non- ERAS hospitals hospital patients are not discharged till day 6 (4).

Following this survey, at Klinika za ginekologiju i akušerstvo was started a new survey, with the first ERAC protocol for cesarean delivery in Serbia and it is an on-going study.

Aluri (5) and Wrench (6) reported ERAS implementation at UK hospital and its influence on length of stay and maternal satisfaction, similar like Coates (7). Pilkington et al. (8) reported a possible reduction of 200,000 euros in hospitals expenses after implementation ERAS protocol for CD in their hospital.

Conclusion:

Successful ERAC protocol implementation in Serbian hospitals will require a great efforts of the multidisciplinary medical staff team and the outside community.

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Labor analgesia project in UKC Tuzla

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At the University Clinical Center Tuzla, there has been a great progress in anesthesia in obstetrics, not only in terms of the knowledge and skills of our anesthesiologists, but also in the organization of their work and the direction in which they go. A multidisciplinary approach to regional anesthesia for anesthetists and obstetricians aims for a safer and easier birth, the birth of alive and healthy baby, with maximum maternal safety at birth.
6 obstetric anesthesia schools were organized in cooperation with the American organization Kybele. Within the schools, lectures were held on topics in regional anesthesia in obstetrics, as well as practical workshops where all techniques of regional anesthesia and analgesia were mastered (spinal analgesia, combined spinal-epidural analgesia, as well as spinal anesthesia for cesarean section). For the implementation of the procedures for painless childbirth, it was necessary to train the entire staff of the Clinic for Gynecology and Obstetrics.
In the period from April 24,2017. by September 30,2019, there were a total of 8854 births at the Gynecology and Obstetrics Clinic. There were 6384 vaginal births, of which 676 (10.5%) were in birth analgesia. Caesarean section completed 2470 deliveries and 424 (17.1%) in spinal anesthesia. We had minimal complications as we adhered to standard protocols for procedures, in aseptic conditions, using sterile gloves, mask and cap. We used atraumatic pencil point needles 25 G and had no postspinal headaches. We have formed trolleys for postpartum analgesia and necessary non-invasive monitoring and equipment. All procedures are introduced into daily practice and we provide these services 24 hours. We produced a booklet on painless childbirth, organized lectures within the school for pregnant women in Tuzla and promoted painless childbirth on a local TV station, with the aim of getting to know pregnant women about painless childbirth. As a result of our activities, we had a total of 1,100 successfully performed delivery of analgesia on the our pregnant women. Our goals are to increase the number of procedures in regional analgesia and anesthesia and to become the reference center in BiH for all forms of obstetric anesthesia.
Keywords :spinal anesthesia, childbirth analgesia, obstetric anesthesia school

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Emergency department thoracotomy Life saving procedure

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In the literature data exist an absence of consensus and specific protocols concerning usefulness and indications of emergency department thoracotomy (EDT)[1].The appropriate indications for resuscitative thoracotomy (RT) are still debated in the literature and various guidelines have been proposed[2].

Cardiopulmonary resuscitation (CPR) is relatively novel branch of medical science. Before 1960s, half of century, EDT was commonly used for open heart massage. After 1960s closed cardiac massage became dominant, gold standard, with high quality closed chest compression and early defibrillation.

Numerious studies, human and animal, demonstrate how open chest cardiac compressions (OC-CPR) are superior in regard to sygnificantly improved hemodynamics and outcomes[3]. Despite that, resuscitative thoracotomeis and open cardiac massage in non-traumatic cardac arrest, have become a largely fogotten art[4].Some authors suggest that the concept of open chest direct cardiac compression in non-traumatic cardiac arrest should be revisited[4].

Concerning traumatic cardiac arrest the American Heart Association and the International Liaison Committee on Resuscitation recommend open cardiac massage under certain circumstances such as penetrating cardiac arrest[5]. EDT will permit a potentially lifesaving measure (clamping of a great vessel) in an extreme situation before proceeding to the operating theatre[6]. In Advanced Trauma Life Support (ATLS) guidelines indication for RT is penetrating torso trauma in extremis and RT by qualified surgeon. Performance criteria for EDT in practice are liberal in comparison with established guidelines[1]. EDT decision-making is more nuanced than previously described. Variation continues in the use of RT after loss of vital signs, in both blunt and penetrating trauma[7].

We present our experience, case report, of succeseful emergency department thoracotomy and open cardiac massage on young, 25 years old patient, sustaining ballistic trauma, with isolated, penentrating, thoracic injury and severe haemorrhagic shock leading to exanguinating cardiac arrest. Prompt initial resuscitation and quick descision for RT was made for a purpose of open cardiac massage and control of bleeding (vascular clamp on the ruptured pulmonary hilus). After successful resuscitation and restoration of spontaneus cardiac rythme, definite surgical treatment, right pulmenectomy, was made in the emergency operation theatre. Patient was discharged neurologically intact, after five months of hospitalisation.

In conclusion, emergency department thoracotomy, as a life saving procedure, is justified in the extreme emergencies on patients with isolited penetrant chest injury, leading to cardiac arrest.

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Hyperbaric Oxygen Therapy and Trauma

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BACKGROUND:Hyperbaric oxygen (HBO) is a clinical method for treatment with oxygen under elevated pressure. In the treatment of many diseases HBO is a self healing factor, while in the therapy of other enter into the complex of therapeutic measures. Hyperbaric oxygen therapy involves intermittent inhalation of 100% oxygen in the chambers under the pressure more than 1 atmosphere absolute (ATA). Hyperbaric oxygen is administered either in single or multiplace chambers. Single chamber accommodates only one patient and the chamber was pressurized to about 2 to 2.5 ATA with 100% oxygen. In multiplace chamber can accommodate several patients and / or medical personnel. The chamber is compressed by air up to 2.8 ATA, while the patient breathes 100% oxygen through an oxygen tent, a face mask or endotracheal tube. Several beneficial mechanisms are associated with intermittent exposure to hyperbaric doses of oxygen. Either alone, or more commonly in combination with other medical and surgical procedures, these mechanisms serve to enhance the healing process of treatable conditions.

1. **HYPEROXYGENATION** provides immediate support to poorly perfused tissue in areas of acutely compromised blood flow. The elevated pressure within the hyperbaric chamber results in a 10-15 fold increase in plasma oxygen concentration. This translates to arterial oxygen values of between 1,500 and 2000 mmHg, thereby producing a four-fold increase in the diffusing distance of oxygen from functioning capillaries..
2. **NEOVASCULARIZATION** represents an indirect and delayed response to serial hyperbaric oxygenation. Therapeutic effects include enhanced fibroblast division, neoformation of collagen, and capillary angiogenesis in areas of sluggish neovascularization such as late radiation damaged tissue, refractory osteomyelitis and chronic ulcerations in soft tissue.
3. Hyperoxia enhanced **ANTIMICROBIAL ACTIVITY** has been demonstrated at a number of levels. Hyperbaric oxygen causes toxin inhibition and toxin inactivation in Clostridial perfringens infections (gas gangrene). Hyperoxia enhances phagocytosis and white cell oxidative killing and aminoglycocide activity. It prolongs the post-antibiotic effect, when hyperbaric oxygen is combined with Tobramycin against Pseudomonas aeroginosa.
4. **DIRECT PRESSURE** utilizes the concept of Boyle's Law to reduce the volume of intravascular or other free gas. For more than a century this mechanism has formed the basis for hyperbaric oxygen therapy as the standard of care for decompression sickness and cerebral arterial gas embolism.
5. Hyperoxia-induced **VASOCONSTRICTION** is another important mechanism. It occurs without component hypoxia, and is helpful in managing intermediate compartment syndrome and other acute ischemias in injured extremities, and reducing interstitial

edema in grafted tissue. Studies in burn wound applications have indicated a significant decrease in fluid resuscitation requirements when hyperbaric oxygen therapy is added to standard burn wound management protocols.

6. **ATTENUATION OF REPERFUSION INJURY** is the most recently identified mechanism.

Oxygen, when breathed under increased atmospheric pressure, is a potent drug. Besides the beneficial effects discussed above, hyperbaric oxygen can produce noticeable toxic effects if administered indiscriminately. Safe time-dose limits have been established for hyperbaric oxygen exposure, and these profiles form the basis for today's treatment protocols.

In the USA in 1976 was established UMS, which was later renamed UHMS - Association of Underwater and Hyperbaric medicine. One of its tasks is to present indications for HBO:

1. Air and gas embolism.
2. Intoxications with carbon monoxide and cyanide.
3. Gas gangrene.
4. Crush syndrome, compartment syndrome and other traumatic injuries
5. Decompression sickness.
6. Difficult to heal wounds, incl. Diabetic foot.
7. Anemic conditions.
8. Necrotizing soft tissue infections.
9. Refractory osteomyelitis.
10. Radiation tissue damage.
11. Skin grafts.
12. Thermal burns.
13. Adjunctive hyperbaric therapy in intracranial abscess.

HBO is provided in mono – or multiplace hyperbaric chambers. Although it is a relatively expensive method with regard to the initial investment in equipment, HBO has been significantly developed in the last decades. Recent trends in hyperbaric medicine are primarily focused on the construction and use of multiplace chambers, mostly because their use is economically advantageous. Multiplace is also a decompression chamber at the Clinic of Anaesthesiology, Hyperbaric and Intensive medicine in the Naval Hospital - Varna. Since opening in 1992 has been continuous increase in the number of treated patients - around 6500 patients and about 70 000 treatment HBO sessions by the end of 2018.

METHODS: From January 1992 to September 2019, 114 trauma patients, except ordinary treatment, were treated with HBO. All patients were divided into 6 groups: 68 – with Decompression sickness (DCS), 19 – severe burn injury, 12 – abdominal trauma, 7 – air gas embolism(AGE), 5 - chest trauma, 3 - head trauma. Adequate treatment in conjunction with HBO therapy at 2.5 – 2.8 ATA for 60 -360 minutes, once or twice per day was used.

RESULTS: The average number of HBO treatments sessions was 13.6 by burn patients, 10,5 – by head-, 6,4 – by abdominal-, 5,6 - chest trauma patients, 2, 5 – DCS, 1,4 - AGE. The main problem is equalizing the pressure during compression. Therefore we apply a special methodology developed by us - "Treatment of Trauma patients in a multiplace hyperbaric chamber". It consists of several "Step by step" components:

a) Examination of the tympanic membrane.

b) X-ray of lungs to identify and objectification of his condition. It should exclude pneumothorax, cavities in the lung, bronchospasm.

c) CT of the skull and facial bones for sinusitis or craniocerebral trauma.

d) Neurological examination for objectification of coma and differential diagnosis.
e) ECG and if necessary consultation with a cardiologist.
f) Ensuring the presence of reanimation team inside hyperbaric chamber.
The following requirements are imposed in relations to the actions of the team in high pressure conditions:
1. The resuscitation team shall be composed of people physically and mentally fit to work with oxygen under high pressure in a confined space.
2. The equipment of the team must comply with the rules for work under high pressure oxygen environment.
3. The narrow space in the chamber does not affect the quality of the medical team actions.

Selection of staff is mandatory.. The resuscitation teams include physicians and nueses with resuscitation experience, mentally stable and physically healthy, with normal barofunction. In the equipment of the team are used tools that do not carry fire and explosion hazard. For safety reasons, items that can not be destroyed by pressure changes such as unopened ampoules, watches, blood pressure monitors, thermometers can not be used.

"Treatment of Trauma patients in a multiplace hyperbaric chamber"methodology includes the following algorithm:
1. Examination of the patient by an otolaryngologist and, if necessary, artificial paracentesis.
2. Resuscitation team inside the chamber. It is equipped with an Ambu, a foot aspirator and patient – specific medication, pre-drawn into a syringe.
3. Careful examination of the patient's medical supplies: iv catheter, intubation tube, urethral catheter, drains. The intubation tube balloon should be inflated with fluid. The urethral catheter should be sealed with a surgical instrument. If there is a closed drainage, it should be opened in advance.
4. In patients with coma and who have not undergone paracentesis, the physician should stimulate swallowing movements and mouth opening by hand.
5. Periodically should be ventilation performed to ensure a safe level of oxygen concentration in the chamber.
6. Team action in case of any complications:
a) if the patient gets a respiratory function, breathing with Ambu connected to an oxygen source is performed, urgent decompression and after completion and if necessary the patient is intubated;
b) at a stop of blood circulation, it is supported by artificial cardiac massage and medication, held, urgent decompression and after completion and defibrillation is performed if necessary.

There were no HBO related complications.

CONCLUSION: Hyperbaric oxygen therapy is effective and safe method for trauma patients provided that patients had received appropriate medical and surgical management.

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Abdominal compartment syndrome in trauma patients

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Abdominal compartment syndrome (ACS) is often unobserved clinical entity with high morbidity and mortality that has been described in critically ill patients including medical, surgical and trauma populations.[1,2,3] ACS is defined as sustained intra-abdominal pressure (IAP) exceeding 20 mmHg associated with multi-organ dysfunction or failure. Intra-abdominal hypertension (IAH) is a sustained or repeated IAP > than 12 mmHg. There are multiple physiological factors that can potentially alter IAP.[3] Blunt abdominal trauma with intra-abdominal bleeding from splenic, hepatic, and mesenteric injuries is the most common cause of IAH. Patophysiological effects of ACS can include ischemia within the splanchnic circulation resulting in a systemic inflammatory response leading to acute respiratory distress syndrome (ARDS), acute renal failure (ARF), increased intracranial pressure and multi-organ failure (MOF).[4,5,6] Patients with the risk for abdominal compartment syndrome should be frequently monitored. Monitoring IAP for signs of ACS is inexpensive and useful diagnostics tool for identifying complications. Treatment for ACS consists of relieving the increased IAP by nonsurgicaly metods or by urgent decompressive laparotomy.[3]

Keywords: Intra-abdominal hypertension, abdominal compartment syndrome, measurement of intra-abdominal pressure.

Introduction

Abdominal compartment syndrome (ACS) has been described in critically ill patients including medical, surgical, and trauma populations with high morbidity and mortality rates.[1] ACS in trauma patients is a potentially lethal condition caused by any event that produces intra-abdominal hypertension and ischemia of the peritoneal organs. The World Society of the Abdominal Compartment Syndrome (WSACS) has published the following definitions and recommendations.[2] The Grading of Recommendations, Assessment, Development, and Evaluation (GRADE) system for clinical practice guideline developers was used to provide consistency in identifying and rating the quality of available evidence and the strength of management suggestions and recommendations.[3]

Blunt abdominal trauma with intra-abdominal bleeding from splenic, hepatic, and mesenteric injuries is the most common cause of intra-abdominal hypertension. [4] Surgical placement of abdominal packing to control hemorrhage may also increase pressure within the peritoneal space.[3] Bowel distension as a consequence of hypovolemic shock and massive volume replacement are important causes of IAH, and subsequent ACS, in trauma patients.[5]

ACS is defined as sustained intra-abdominal pressure (IAP) exceeding 20 mmHg associated with new organ dysfunction or failure.[3] The normal IAP for critically ill adults is 5–7 mmHg [10, 11]. There are the following grades for IAH: Grade I for IAP of 12-15 mmHg, Grade II for IAP of 16-20 mmHg, Grade III for IAP of 21-25 mmHg, and Grade IV for IAP of more than 25 mmHg.

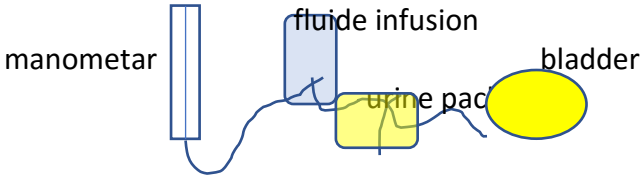
The pathophysiology of ACS is directly related to the increased IAP of the disease process and the consequent effects of this increased pressure on various organ systems. Distension of the abdomen from the increased IAP pushes up the diaphragm and compresses the thoracic cavity leading to impairment of respiration and ventilation. The increased IAP compresses the vena cava which causes a decrease in venous return (preload) and results in a decreased cardiac output. This decreased cardiac output also results in decreased perfusion to the kidneys resulting in impairment of renal function. In response to hypoxic tissue injury, proinflammatory cytokines are released. These molecules promote vasodilatation and increase capillary permeability, leading to the development of edema. After cellular reperfusion, oxygen free radicals are generated. These agents have a toxic effect on cell membranes that is further aggravated by the presence of cytokines, which stimulate the release of even more free radicals. Insufficient oxygen delivery to the tissues limits production of adenosine triphosphate and this decreased availability of adenosine triphosphate impairs energy-dependent cellular activities. Adverse effects of ACS can also include ischemia within the splanchnic circulation resulting in a systemic inflammatory response leading to acute respiratory distress syndrome (ARDS), acute renal failure (ARF), increased intracranial pressure, and multi-organ failure (MOF).[5,6,7]

ACS may arise due to condition in the abdomen, or outside the abdomen. Depending on the cause, ACS is classified as three main types: Primary ACS, which occurs due to an abdominopelvic pathology (e.g., penetrating trauma, intraperitoneal hemorrhage, pancreatitis); Secondary ACS, which occurs due to conditions not originating from the abdominopelvic region (e.g., large-volume resuscitation, burn injuries); and Recurrent ACS, which describes recurrence of ACS after a successful treatment of a previous episode.[8,9]

Some symptoms and signs of abdominal compartment syndrome include: pain in the abdomen, tense, tight abdomen, increase in girth of the abdomen, unstable hemodynamics that includes altered heart rate, blood pressure, reduction in urine output.[10]

Diagnosis of ACS syndrome is based on the measurement of the pressure in the urinary bladder via a urinary catheter with the patient in lying down position. This pressure reflects the intra-abdominal pressure.[3] Monitoring IAP and abdominal perfusion pressure (APP) for signs of ACS is inexpensive and useful diagnostics tool for identifying complications. APP=Mean arterial pressure (MAP)–IAP.[11,12] An upper limit of 25 mL fluid is introduced into the bladder during the procedure (Picture 1).

Picture 1. *Intraabdominal presure measurment using bladder technique*



Measurement of intra-abdominal pressure via the urinary bladder may not be possible in patients with bladder problems. In such patients, the intra-abdominal pressure may be measured by a tube inserted into the stomach, a catheter inserted via the femoral vein into the inferior vena cava, or direct measurement of the pressure in the abdomen. Patients at risk for ACS should be frequently monitored. Continuous measurement of the intraabdominal pressure is preferred to intermittent measurement in some cases. [3-5]

Treatment of abdominal compartment syndrome is carried out in a critical care unit. The intra-abdominal pressure is reduced so that the blood supply to the abdominal organs is not affected and damage is prevented.[6-10] The treatment includes the following supportive care

(the body should be positioned in such a way that the head end is at an angle of less than 30 degrees, artificial ventilation may be necessary for people who cannot breathe efficiently, antibiotics may be needed to treat infection, medications are required to relieve pain and thereby improve abdominal wall movement, blood flow to organs is maintained through drugs that increase blood pressure and the judicious use of fluid). Approaches to reduce intra-abdominal pressure: diuretics, dialysis, drainage of ascites, treatment of gastric distention and fecal impaction.

A surgical procedure (decompressive laparotomy) is used during which the abdomen is opened to relieve the abdominal pressure and is temporarily closed. Repeated such surgeries may be required before a final decision to close the abdomen permanently is taken. The final closure is often performed 6 to 12 months after the initial surgical decompression procedure to give time for the inflammation to subside.

Summary

ACS is associated with high morbidity and mortality among trauma patients. ACS is classified as an IAP greater than 20 mmHg with a new organ dysfunction. IAH and ACS occur frequently in patients in critical care unit and can alter organ perfusion and end organ function. Due to the prevention of complications in patients with the risk for ACS should be frequently monitored IAP (modified Kron technique) which may improve patient outcome of treatment and decrease hospital costs.

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Fat embolism and fat embolism syndrome

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Abstract

Dissipation of fat globules into the circulation with consequent manifestations mainly on respiratory function has been defined as fat embolism while severe forms with systemic manifestations have been denoted as fat embolism syndrome.

Etiologically, the highest incidence of fat embolism has been documented after major trauma and major surgical procedures of long bones and pelvic bones, while some other conditions and procedures are also associated with this clinical entity.

Speculations on the pathophysiology of fat embolism has lead to proposal of two theories, mechanical and biochemical. It has been widely accepted that both mechanical and biochemical changes induce impairment at circulatory and cellular level. After hydrolisation otherwise inert fat initiates direct damage to the cells of endothelium in different parts of circulatory system and also induce systemic inflammatory response with most prominent signs of alterations of function of respiratory and central nervous system, and changes in skin and retina.Establishing prompt diagnosis of fat embolism is complex and difficult despite numerous clinical criteria introduced by different authors.Early diagnosis enables institution of early supportive measures,which are the mainstay of the therapeutic approach besides early operative stabilization of long bone fractures and treating other conditions along with measures of prevention.

Key words : fat embolism, fat embolism syndrome, trauma, long bone fractures, polytrauma

Overview

Therapeutic approach for patients with major trauma and surgery implies great concern for possibility of serious complication of fat embolism and fat embolism syndrome. The incidence of fat embolism varies and it can turn up to 90% in patients who had sustained major injuries or had major surgical procedures on long bones and pelvic bones (1,2).

The incidence of fat embolism syndrome is in range of 2-5%, when clinical picture is very severe with systemic inflammatory response that has been initiated with multiple organ system dysfunction and complications and increased morbidity and mortality. Prompt diagnosis and supportive therapy are essential for critical care of these patients (1,2).

Although the clinical presentation and signs of fat embolism were described about two centuries ago and significant progress has been achieved in understanding and recognition of this condition, the issue of early diagnosis has remained troublesome. Despite the most commonly used numerous Gurd's criteria with major and minor clinical signs, the limitation of validation of the criteria has made a disadvantage, and currently it has been largely accepted that despite high incidence of fat embolization in patients with bone fractures clinical picture occurs in a small number of patients(3,4).

It is the clinical picture that is essential for the diagnosis of fat embolization and fat embolization syndrome, based on estimation of dyspnoea, skin petechiae and cognitive

alterations in the first few days after trauma, fractures of long bones or surgical procedures on long bones. Various laboratory tests and imaging modalities may contribute in diagnosis.

Assessing etiology the most common causes of fat embolization and fat embolization syndrome are fractures of long bones and pelvic bones and major surgical procedures on long bones and pelvic bones and they account for aboit 90% of cases(5). Other conditions that have been described to be associated in some cases with fat embolism include acute pancreatitis ,burns, liposuction, joint reconstruction, parenteral lipid infusion, sickle cell crisis, pathologic fractures , cardiopulmonary bypass, massive soft tissue damage after crush injury, bone marrow transplantation(5). Several risk factors have been recognized to increase the risk of fat embolism and they include: young age, multiple fractures, closed fractures, long period of conservative treatment of long bone fractures and the technique of surgical stabilization (5).

Epidemiology

According to published data there are great variations of the incidence of fat embolism in range from 67% to higher than 95%, many cases of mild fat embolism may not be recognized(6).The incidence of fat embolism syndrome in papers published in period of previous technique of surgical stabilization of trauma reached 19%, while in recent papers it is in range of 2-5% (7,8).

Pathophysiology

Based on animal studies and clinical picture there have been developed two theoretical approaches regarding the pathophysiology of fat embolism.

Gassling H.et al. postulated mechanical theory which is based on assumption that large fat droplets released after injury of bone medulla or adipose tissue enter venous system and circulate to pulmonary microvasculature where there is possibility to be deposited or to travel to systemic circulation through arteriovenous shunts. Significant proinflammatory and prothrombotic potential of fat globules trigger platelet aggregation eventually lodging in the pulmonary vasculature. Obstruction of pulmonary capillaries, interstitial haemorrhage and oedema lead to alveolar collapse and vasoconstriction. Massive fat embolization may result in circulatory failure.The process of deposition of fat globules in the pulmonary microcirculation lead to elevated pulmonary artery pressure, alterations in of oxygen exchange caused by ventilation-perfusion mismatch and systemic effects on end organs, with most prominent effects in the brain, kidney and skin. On the other hand, deposition of fat globules in the microcirculatory bed of brain initiates a cascade of chemical reactions with development of a systemic inflammatory response syndrome, local inflammation, alterations of perfusion and ischemia (10).

Baker PL and coworkers proposed biochemical theory for description of fat embolism syndrome. The focus in biochemical theory is on hormonal changes after trauma and proinflammatory state after release of glycerol and free fatty acids by tissue lipases. Intermediate products of fat metabolism may cause toxic injury to pneumocytes and pulmonary endothelial cells with development of cytotoxic oedema and haemorrhage. Damage of endothelial cells initiates pro-inflammatory cytokine cascade with consequent

acute lung injury or acute respiratory distress syndrome in the lungs and systemic inflammatory response in multiple organs (11).

It has been largely accepted that both mechanisms are involved starting with embolization with fat globule from injured tissue and subsequent biochemical degradation and initiating inflammatory response in different tissues.

Clinical presentation

Although there are some published evidence on fat embolism that occurred twelve hour after injury and even two weeks after the initial insult, in most cases clinical picture of fat embolism develops in the period after 24 h to 72 hours of the traumatic event or some planned procedures, what emphasizes the necessity of precise documentation of the time of injury or surgical procedures.

The most commonly cited criteria published by Gurd include major criteria that describe signs of respiratory dysfunction, neurologic deterioration and skin changes, while there are also numerous minor criteria that may be combined with major criteria (12). Lindeque B. proposed criteria based more on respiratory signs, and Schonfeld SA described a fat embolism index that is a sum of measuring seven clinical features (13,14).

Fat emboli mostly affect pulmonary circulation with dominant clinical signs of respiratory dysfunction in terms of tachypnea, dyspnoea, hypoxemia, with different severity of the signs in patients, while some may have progression of the condition to respiratory failure like in acute respiratory distress syndrome. There is usually high incidence of necessity for respiratory support in patients with fat embolism developed after long bone fractures (15).

The signs of affecting central nervous system manifest mostly in the early period and in most cases after development of respiratory distress, with a wide spectrum of changes from drowsiness and confusion to severe seizures and comatose state. There have been described also focal neurological signs like hemiplegia, aphasia, apraxia, different visual disturbances, with emphasis that most of neurological deficits are reversible (16).

Petechial rash may occur in up to 60% of patients as a result of embolization of small dermal capillaries and erythrocyte extravasation, mostly in the period of first 36 hours and disappearing within seven days. The location of petechial rash is specific, mostly in skin folds of upper body, neck, conjunctiva and oral mucous membrane (17).

Some other signs of fat embolism may be present in patients such as myocardial depression, electrocardiogram changes, high temperature, soft retinal exudates and visual disturbances, coagulation abnormalities and renal changes with oliguria, lipiduria, proteinuria, haematuria. Thrombocytopenia and anaemia may be common hematologic manifestations in patients with fat embolism.ž

Diagnostic approach

Despite development of numerous scales and criteria by different authors and better imaging modalities the diagnosis of fat embolism is made by assessing characteristic clinical syndrome in the context of imaging and history of the injury or disease (6,12,13,14).

Laboratory tests usually demonstrate anemia and thrombocytopenia in fat embolism syndrome, increased levels of urea and creatinine, metabolic acidosis. Analysis of the arterial blood gas usually demonstrates a low partial pressure of oxygen and hypoxemia with common finding of increased alveolar-arterial gradient. Lipase, free fatty acid, and phospholipase A2 may have elevated levels in fat embolism syndrome, but these findings are common in other pathologic conditions of the lung.Fat globules might be present in blood, urine, and sputum, but these findings are nonspecific for fat embolism and fat embolism syndrome.

Different radiological findings have been described but none of them are specific for fat embolism.The finding of bilateral patchy infiltrates on chest radiogram typical for acute respiratory distress syndrome is very common Ventilation/perfusion scans may show sub-segmental perfusion defects with a normal ventilatory pattern. In some cases, common findings on chest CT are ground glass opacification with interlobular septal thickening, that might be described as alveolar oedema, microhaemorrhage, and inflammatory response (18).Magnetic resonance of the brain may as a most sensitive test for demonstration of fat embolism reveal a star-field pattern of punctiform changes on diffusion-weighted imaging(19).There have been attempts to directly sample the respiratory system for fat particles with staining alveolar cells for neutral fat, one study had shown possibility of confirming the diagnosis of fat embolism syndrome, but clinical importance has not been approved(20).In the situations of available equipment there is also possibility of monitoring of releasing fat globules into the circulatory system by utilization of transesophageal echocardiography intraoperatively during the process of intramedullary nailing.

In the absence of specific diagnostic tests clinical diagnostic criteria remain gold standard for the diagnosis of fat embolism (6,12,13).

Therapeutic measures

At present, prevention, early diagnosis, adequate surgical technique , appropriate symptomatic treatment are essential, as there is no specific treatment for fat embolism or fat embolism syndrome. The mainstay of treatment of fat embolism syndrome consists of supportive measures that include adequate oxygenation and ventilation, maintenance of haemodynamic stability, adequate hydration, support with blood products as clinically indicated, thrombosis prophylaxis, adequate nutritional support.

There should be accentuated the emphasis on early immobilization of fractures by operative procedures what may reduce the incidence of fat embolism syndrome. Efforts to limit the elevation of intraosseous pressure during operative procedures on long bones have shown beneficial in terms of reduction of possibility for development of fat embolism. There could be also other measures that refine the process of fixation and stabilization of prosthetic materials (21).

Different studies of administration of different pharmacologic agents including dextrose infusion, heparin and other agents, with aim of decreasing mobilization of free fatty acids, have not shown clinical benefits (22).Therapy with corticosteroids has been extensively studied based on the proposed anti-inflammatory action with possibility of reducing perivascular hemorrhage, but data supporting administration of corticosteroids in already established fat embolism syndrome are controversial(14,23).Adequate support therapy and open reduction and internal fixation of long bone fractures may provide conditions for recovery from complications of fat embolism for most patients. Still, mortality rate approximates range of 5% to 10% (24).

A multidisciplinary team should be engaged in the process of diagnosis and treatment of patients with fat embolism. Limitation of diagnostic tests to clinical criteria and absence of specific treatment emphasize the necessity of preventive measures and prompt recognizing of the clinical syndrome of fat embolism and adequate supportive measures in terms of maintenance of respiratory and cardiocirculatory stability, correction of laboratory findings, maintenance of general supportive measures for critically ill patients.

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Burns – Anesthesia and Intensive Care

Selma Sijerčić

Major burns are systemic injuries requiring input from multiple specialities. Important considerations in the clinical outcome for these patients are early resuscitation, multidisciplinary team management, early surgical debridement, and prevention of complications. Anaesthesia in this group of patients can be challenging with profound hypermetabolism, pain management issues, alteration of drug pharmacokinetics, potential airway problems, temperature control, and substantial blood loss.
Overall, the mortality rate among hospitalized burn patients in a recent review of European data was 13.9% (4–28.3%). Major risk factors for death are older age, a higher total percentage of burned surface area, inhalation injury [mortality rate 27.6% (7.8–28.3%)], and chronic diseases. There appears to be no sex-related difference in survival after thermal injury. Multi-organ failure and sepsis are the most frequently reported causes of death. The main causes of early death (<48 h) are burn shock and inhalation injury.

- Crystalloid is the fluid of choice for resuscitation, volume calculated by the Parkland formula=4 ml kg^{-1} (% burn)$^{-1}$.
- Hypovolaemic shock in the first few hours after a burn injury is never due to the burn alone.
- Primary and secondary survey should follow ATLS principles (do not get distracted by the burn).
- Blood loss during debridement and grafting can be significant and insidious.
- Major burns require a dedicated multidisciplinary team for adequate management.

Primary survey
The initial management of a severely burnt patient is similar to that of any trauma patient. The burn injury must not distract from this sequential assessment, otherwise serious associated injuries may be missed.

Airway with cervical spine control
All burn patients should receive 100% oxygen (O_2) through a non-rebreathing mask on presentation. An assessment must be made as to whether the airway is compromised or is at risk of compromise. Initial compromise of the airway is almost always due to a low Glasgow Coma Score (GCS) and not the burn. Early tracheal intubation should be considered in the presence of any of the following features: stridor, hypoxaemia or hypercapnia, a GCS of <8, deep facial burns, full-thickness neck burns and oropharyngeal oedema. If intubation is required at this early stage, it is usually technically easy as swelling of the airway has not yet occurred. An uncut tracheal tube (size 8.0 mm or above) is used to allow subsequent bronchoscopy. Succinylcholine is safe in the first 24 h after a burn—after this time, its use is contraindicated due to the risk of hyperkalaemia leading to cardiac arrest, thought to be due to release of potassium from extrajunctional acetylcholine receptors. This can persist up to 1 year post-burn.

Inhalation injury
Inhalation injury is defined as the aspiration of superheated gases, steam, hot liquids, or noxious products of incomplete combustion. It is almost never seen in association with flash burns or other forms of brief, albeit high temperature, exposure. True inhalation injury is likely to be present if the burn was received in an enclosed space with delayed escape or rescue. Three distinct clinical entities are possible.

Upper airway thermal injury (above larynx)
This usually occurs above the glottis, as by the time hot gases reach the larynx, the heat energy has been dissipated. The pharynx and epiglottis may have significant thermal injury which can swell dramatically. Clinical signs include inspiratory stridor, change in voice/hoarseness, and a swollen uvula.

Lower airway thermal injury (below larynx)
Inhalation of products of incomplete combustion causes sloughing of the airway's epithelium, mucus secretion, inflammation, atelectasis, and airway obstruction. Clinical signs include dyspnoea, coughing wheezing, and the production of copious secretions. Findings at bronchoscopy include carbonaceous deposits, oedema, bronchial mucosal erythema, haemorrhage, and ulceration. Bronchial lavage with 1.4% bicarbonate solution has been used to neutralize acidic deposits and remove soot contamination.

Breathing
Breathing, chest movement, and tracheal position should be assessed clinically. There are several ways that a burn injury can compromise respiration.

Mechanical restriction of breathing
Deep dermal or full-thickness circumferential burns of the chest may severely restrict chest wall movement and relieving escharotomies may be necessary to allow adequate ventilation. These are rarely, if ever, needed before admission to a burn service.

Blast injury
Penetrating injuries can cause tension pneumothoraces, and the blast itself can cause lung contusions and alveolar trauma which may lead to adult respiratory distress syndrome.

Circulation
Two large-bore i.v. cannulae should be inserted through the unburnt skin if possible and baseline bloods sent. Traditional sites for i.v. access may be unavailable and unusual peripheral venous sites or central venous access is required (usual indications for central venous line insertion also apply). The groins are usually spared so femoral venous cannulation is often possible. Burns are not the cause of immediate hypovolaemia. If there are features of hypovolaemic shock, the patient is almost certainly bleeding from other injuries. Warmed Hartmann's solution should be started and titrated to cardiovascular signs, but if stable can be run slowly until the burn calculation is made.

Disability
A brief assessment of the conscious level should be made using the GCS score and pupils examined.

Exposure and estimation
Expose and ensure all jewellery and watches are removed from burnt limbs. The patient should be examined (including the back—log roll if appropriate) to get an accurate estimate of the burn area and to check for any concomitant injuries. Burn patients become hypothermic easily, so should be covered and warmed as soon as possible. Burns are classified by total body surface area (TBSA) and depth. A standard Lund–Browder chart is readily available in most emergency departments for a quick assessment of BSA burnt. If this is not available, the 'Rule of Nines' is fairly accurate in adult patients.

Fluids
I.V. fluid resuscitation is required in adults if the burn involves more than 15% BSA or 10% with smoke inhalation. The Parkland formula is the most widely used resuscitation guideline and is 4 ml kg^{-1} $(\%burn)^{-1}$ which predicts the fluid requirement for the first 24 h after the burn injury. Starting from the time of burn injury (not time of presentation), half of the fluid is given in the first 8 h and the remaining half is given over the next 16 h. The fluid of choice is Hartmann's solution. Any fluid already given should be deducted from the calculated requirement. A urinary catheter should be inserted and hourly urine output is a used as a guide to resuscitation. In adults, at least 0.5 ml kg^{-1} h^{-1} should be passed.

Analgesia
Full-thickness burns are painless; however, a mixed picture is common and the patient should receive i.v. morphine titrated against response.

Anaesthesia and surgery for burns
Inpatient burn injury care should be provided only by specialists trained in burn care in a burns unit. This is a reflection on the team approach to burn injuries, the resources, and infrastructure necessary to provide both critical care and the long-term management of the patient in terms of the planning and timing of surgical procedures.
Recent trends in patient care have focused on early excision and wound coverage, aiming to remove the full-thickness injury and get biological closure. This potentially reduces the risk of wound infection and the development of sepsis. The risk of this approach is the physiological insult of surgery to a patient who may well be deteriorating rapidly from their initial injuries. The most important and difficult clinical decisions are often made by the team at this stage.

Critical care management
In the first 24–48 h after resuscitation in patients with major burns, the hormonal response and inflammatory mediators cause hypermetabolism, immunosuppression, and SIRS.

Mechanical ventilation
Usual lung protection strategies apply, and routine ventilator-associated pneumonia prevention strategies should be implemented. Prophylactic corticosteroids or antibiotics have no role. In patients with inhalation injury, early tracheal intubation, aggressive pulmonary toilet, bronchodilator therapy, and bronchoscopic lavage are all important.

Psychological care/rehabilitation
A multidisciplinary team of physiotherapists, psychologists, nurses, councillors, and occupational therapists are vital to aid rehabilitation and reduce long-term impairment.

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POSTER PRESENTATIONS

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CytoSorb in a patient with politrauma and associated rhabdomyolysis - A case report

Ahmetović Djug Jasmina, Vikalo Lejla

Abstract: Politrauma is a serious injury to at least two organ systems where at least one injury or a combination of more than one endangers the patient's life. According to world statistics, it is the leading cause of death. Numerous scales are used to assess the severity of injuries, by which we can numerically express the severity of the injury. In order to indicate the importance of immune system dysfunction in the politraumatized, the presence of SIRS was added in defining the term polytrauma. Also due to skeletal muscle destruction and traumatic rhabdomyolysis, products from damaged muscle cells such as myoglobin, which can lead to acute renal failure, are released into the bloodstream. The use of extracorporal cytosorb blood purification is also used in the treatment of polytraumatized patients. By using cytosorb, the hyperinflammatory response of the organism is eliminated or reduced, thereby inhibiting SIRS and control over the course of the disease. Because cytosorb removes a wide range of molecules of size from 5 to 60 kDa, it has also found its use in eliminating other endogenous molecules eg myoglobin (17 kDa). In this paper, we present a case of a 41-year-old man with multiple serious injuries and consequent rhabdomyolysis who was hospitalized at the Intesive Care Unit of the Anesthesia Clinic and had cytosorb therapy successfully used for the first time in our hospital.

Keywords: polytrauma, SIRS, rhabdomyolysis, cytosorb

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The brucella aortitis with aortoduodenal fistula – a rare case report

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Abstract: Aim: The aim of this paper is to present a rare case report about primary aortoduodenal fistula (PADF) as result of the brucella aortitis.
Methods: The patients admitted with signs of gastrointestinal bleeding and during surgery the aortoduodenal fistula identified. The postoperative recovery was normal and at the seventh post-op day a fully recovered patient was transferred to another hospital for further Brucellosis evaluation.
Conclusion: The primary aorto-duodenal fistula is a rare complication of aneurysm of the abdominal aorta and a rare cause gastrointestinal bleeding, but about whom to think about. Therefore, detailed anamnesis, physical examination, early recognition of abdominal aneurysm rupture with CT angiography and emergency surgery are an algorithm for successfully resolving this life-threatening disease.

Keywords: aneurysm of aortae, aortoduodenal fistula, brucelosis

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Intercostal nerve block and awake sedation for intrapleural foreign body removal with VATS. A case report.

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Video assisted thoracoscopy (VATS) was until recently considered impossible without general anesthesia (GA) with one-lung ventilation (OLV).(1,2) Non-intubated VATS was introduced with purpose to avoid the adverse effects of GA and intubation.(3,4) Paravertebral and intercostal nerve block (INB) have been proposed as a safe and less invasive alternative for thoracic epidural anesthesia (TEA) and GA.(2,4)

This case report describes the benefits of non-intubated VATS under INB and awake sedation, in the extraction of the retained intrapleural foreign body.

A 66y old male was transferred on the Thoracic surgery department for a surgical foreign body extraction which was retained inside the right pleural cavity. The patient was previously admitted on the Cardiology department with signs of heart failure and atrial fibrillation. He had diabetes mellitus for 15 years. Echocardiography showed dilatative ischemic cardiomyopathy with ejection fraction of 20% . Chest X ray detected bilateral pleural effusion with partial atelectasis. The medical treatment failed and the persistent right side liqiudothorax was evacuated using Pleuracan® (Braun) catheter.(5) The position of the catheter was secured with suture and dressing. Because of the patient movement the catheter got disconnected and sank completely into the pleural cavity (confirmed on X ray and CT scan). Since the patient was hemodynamically unstable, hypotensive and tachyarrhythmic, the evacuation of the catheter was done using 2 port VATS, under combination of INB and awake sedation. The retained catheter was successfully removed, completely intact. The procedure went without any complications. The next day the patient was discharged.

We conclude that non-intubated VATS procedures using awake sedation and INB are reliable, safe, minimally invasive techniques for small lung resections and interventions.

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Postoperative quality of life after total gastrectomy compared with partial gastrectomy

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INTRODUCTION: Gastric cancer is the most common cause of death. In men, this percentage is (17.4%) and in women (7.8%).
According to statistics from 1995, the proportion of causes of gastric cancer mortality (EGC) was (28.6%.) However, this proportion increased to (57.6%) in 2009. Early detection of gastric cancer provides timely treatment. and better clinical outcome.
Although alternative treatments for gastric cancer have been developed, radical surgery remains the only treatment that offers the ability to cure. The quality of life after gastrectomy is changing and has been the subject of much research. The European Organization for Research and Treatment for Cancer (EORTC) has developed a specific questionnaire to assess quality of life after gastrectomy. Questionnaire (QLQ) -C30 has 30 questions regarding daily activities, weekly activities, and satisfaction with one's health. To date, this questionnaire has been translated into 81 languages and has been used in more than 3,000 studies worldwide.
AIM OF THE RESEARCH: The aim of the study was to evaluate the quality of life of patients at the Clinic for Abdominal Surgery UKC who underwent total gastrectomy and partial gastrectomy after confirmed gastric cancer using the QOL C-30 questionnaire.
RESEARCH AND RESEARCH METHODS: The study was conducted on 15 patients of the Clinic for Abdominal Surgery UKC between January 2018 and December 2018 who underwent total and partial gastrectomy. Surveys before surgery were used as a research method, after 1-month interview and then 6-month postoperatively, by telephone.
After the survey, the answers were entered into the QOL C-30 questionnaire and then statistically processed.
RESULTS: Patients ranged in age from 25 to 65 years. Of the 15 patients, 8 patients had total and 7 patients had partial gastrectomy. Patients with total gastrectomy had greater difficulty performing their daily activities, greater weight loss, and loss of appetite with a pronounced sense of weakness as well as greater dissatisfaction with their health. Patients with partial gastrectomy had more power to perform daily activities, weight loss and appetite were less and were less dissatisfied with their own health.
CONCLUSION: The quality of life of patients after partial gastrectomy is better than that of patients after total gastrectomy.

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Specific considerations of the anesthesiologists approach in cardiac autotransplant patients – Our experience

Edin Kabil, Ermina Mujicic, Nermir Granov

Cardiac autotransplantation is a method used for surgery on cardiac malignancy, and in cases of posterior location and difficulty of surgical exposure in mitral valve surgery.
Cardiac autotransplantation presents a rare and difficult anesthesiologist challenge. The role of the anesthesiologist as an integral member of the team in early preoperative considerations, during surgery and through the postoperative care development, is fundamental and crucial.
The left atrium plays a major role in cardiac physiology modulating left ventricular filling during diastole. Left ventricular diastolic dysfunction or mitral valve disease may lead to left atrial pressure or volume overload which chronically would lead to left atrial remodeling and enlargement. Left atrial enlargement has been proposed as a predictor of common cardiovascular outcomes usually manifested as atrial fibrillation, stroke, congestive heart failure, and cardiovascular death.
We have admitted 55 years old patient after previous history of valvular disease for 37 years, and history of cerebral stroke 25 years earlier, based on absolute arrhythmia. Patient has been avoiding surgical treatment and has been presented on admittance with severe mitral valve insufficiency, large atrial enlargement, multivessel coronary disease, pulmonary hypertension, chronic obstructive pulmonary disease, carotid artery occlusion and stenosis, and mechanically induced disarthria. Preoperative CT scan has shown megaatrium measuring 134x193x157mm. Patient has been prepared for a previous six months, and three days after arrival operation has been performed using cardiac autotransplantation. Patient has been in the Intensive care unit for another 20 days and on 35th day was released for a further physiatrist treatment. We herein describe the challenges in the management of this patient condition and resulted significant improvement in both cardiac and physical status and quality of life.

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Medicamentous and mechanical support of cardiac surgery patients - Case report

Edis Salihović, Suad Keranović

Introduction: Cardiac surgery patients often need inotropic support to establish hemodynamic stability and adequate oxygen supply. Catecholamines: Norepinephrine (noradrenaline), epinephrine (adrenaline), dopamine and dobutamine are still the backbone of inotropic support therapy. By acting on adrenoreceptors, they allow vasoconstriction through the alpha receptors, and increased cardiac out via the beta 1 receptor. Combinations of different catecholamines with one another or with other drugs, such as phosphodiesterase inhibitors or nitrates, lead to a wide range of possible hemodynamic effects. Among them, phosphodiesterase inhibitors appear to be the most promising drugs introduced to clinical use to date. The intra-aortic balloon pump has been the most commonly used device for mechanical circulatory support in critically endangered patients. The IABP mechanism is based on its inflation of the balloon during the diastole of the cardiac cycle, at the root of the aorta, which results in an increase amount of blood and oxygen in the coronary arteries. Prophylactic and postoperative administration of IABP is recommended, which is commonly used in high-risk patients undergoing cardiac surgery or percutaneous coronary intervention.
Methods: This case report includes 58 years old male patient who was admitted to our hospital due to planned coronary bypass surgery. He had a recent myoradial infarct, pulmonary embolism 15 years ago and primary hypertension.
Results: Applying inotropic support, IABP and adequate volume management during surgery and post surgery period in ICU has led to hemodynamic stability. Leading to the recovery of the patient and hospital discharge.
Conclusion: The outcome of cardiac surgery in the patients with coronary artery disease depends of high-quality preoperative preparation, the type of operation, and postoperative treatment that requires adequate hemodynamic and respiratory managment.

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Spinal anesthesia for cesarean section-our experience

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INTRODUCTION

Spinal anesthesia is method of injection local anestethic into the subarachnoid space, causing sympathetic, motor and sensory block.

At OBG clinic Tuzla in the past Cesarean Section were done strictly under general anesthesia. Number of elective C S was insignifficant, emergencies were mostly high level of emergency and urgent. On April 2017. in cooperation with Kybele organisation, 1.St school of obstetrics anesthesia was held. Intensive and continuous education, both, doctors and technicians starts and implementation of spinal anesthesia for C S

NEW AND UNKNOWN = FEAR AND RELUCTANCE

Improving our knowledge and skills we try to make our obstetricians and patients comfortable with spinal anesthesia as better, safer method of anesthesia for C S.

In the meantime 5 more schools of obstetrics anesthesia were held, using enormous experience of our colleges from Kybele we made a great progress increasing number of C S done in spinal anesthesia.

MATHERIAL AND METHODS

From April 2017. to October the first. 2019. at OBG clinic Tuzla were 8917 deliveries, out of it 2433 were finshed by C S. Out of that number 424 C S were done in spinal anesthesia. We retrospectively analysed those 424 cases.

Procedures were performed by the rules of profession, stirctly aseptic technique. We used local anesthetic with opioid.

RESULTS

Results are satisfying. Patients are satisfied, no serious complications with negative or permanent damages. We had 22 failed procedures, 10 cases of inadequate analgesia.

Side effects by frequency of occure were: hypotention, pruritus, bradicardia, nausea and/or vomiting.

Since maintaining of good uteroplacental flow is a goal of every birth we try to achieve only positive effects of spinal anesthesia to uteroplacental flow which is confirmed by APGAR SCORE of newborns 7/9 to 9/9.

CONCLUSION

Spinal anesthesia is safer, better method of anesthesia for C S. Considering it is obstetrics anesthesia great advantage is for often full stomach patients, at high risk of difficult or failed intubation, risk because of various disease. Specifi c advantage is awake mother establishing contact with baby, early breast feeding, less need for analgesia.

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Traumatic intercostal lung herniation in polytrauma patient – a case report

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Lung herniation is protrusion of lung tissue from thoracic cavity. It is extremely rare, potentially life threatening condition, which is caused by rapid increased in intrathoracic pressures coupled with defects in the chest wall. Because it is a extremely rare occurrence and has been reported mainly as case reports in the literature, the incidence and prevalence is unknown. Lung herniations are classified as spontaneous and acquired ones, with further classification of acquired subgroup to traumatic, spontaneous and pathological. Traumatic lung herniations are then divided into intercostal, cervical and diaphragmatic. We report the case of a 74-year-old man who was hit by 3.5 tons metal plate carried by crane and who suffered a glenohumeral joint dislocation associated with multiple rib fractures and herniation of the lung through an anterior chest wall defect. At admission to emergency department he was hemodinamically (BP 160/90 mmHg) and respiratory (RR 25/min) stable with normal laboratory findings including blood gas analysis (pO2 14.2 kPa, SatO2 0.97). Physical examination revealed subcutaneous emphysema on the left anterior chest wall, flail chest on the left, bilateral less audible breathing, left femur fracture and suspected dislocation of left shoulder. A computed tomography scan was notable for ventral intercostal left lung herniation rupture of the left hemi-diaphragm associated with herniation of the stomach and spleen into left chest, left- side fractures of 1st to 3rd and 4th to 11th rib that are significantly displaced, right-side fracture of 3rd to 6th rib, left - side haematothorax and pneumothorax, insignificant right - side pneumothorax, dislocation of left glenohumeral joint, and transtrochanteric fracture of the left femur. Thoracic surgery was consulted, and decision was made for emergency thoracotomy to repair lung herniation. Patient was immediately transferred to operation room, and intubation with a Robert Shaw endotracheal tube was done. The patient was positioned in right decubitus position and left thoracotomy was performed. The abdominal organs were reducted to their normal anatomic position and diaphragmatic defect was repaired. Left lung was repositioned and full reexpansion of left lung was achieved. At the end of the surgery patient was admitted to intensive care unit sedated and mechanically ventilated due to unstable left chest wall with thoracic drainage on the both sides. A month later after two more surgeries and after eighteen days on mechanical ventilation support he was discharged home with almost normal pulmonary function.

Because of rare occurrence and variety of etiologies and clinical presentations, early clinical diagnosis may be difficult and may present a daunting challenge for every clinician involved in managing thoracic trauma patients. Recognation and early consultation with a trauma or thoracic surgeon upon diagnosis of lung herniation is critical to ensure proper treatment and recovery.

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Anesthesiological treatment of trauma injury in children

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Anesthesiologic treatment of traumatic injuries in children is a great challenge for the anesthesiologist.At the same time,he is facing a serious task in relation to the anesthesia treatment of children for elective surgery.This involves a good knowledge of the problem, the experience of the anesthesiologist, and an adequate choice of resuscitation measures. It should be taken into consideration that the child may not have been in good health before the injury. In children, possible heart defects, respiratory dieseases and neurological disordes should bee considered. The approach and course of anesthesia in traumatic injury to a child depends on the urgency of the situation and state of consciousness. The aim of this work is to present the anestethic treatment of traumatic spleen injury in a 10-year -old-boy. The car of the boy begins from the moment of arrival of the Emergency Medical Service, by performing all diagnostic tests and labaratory findings at the Emergency Medicine Clinic for Anesthesia and Resuscitation.Anesthetic treatment adapts to the principles of traumatic surgery.Follow-up and further course of treatment after emergency surgery involves the team work of medical staf (anesthesiologist,pediatric surgeon, pediatrician intensivist),all with a view to the successful recovery of the child,as shown in this paper.

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Perioperative management of elderly patients with hip fractures

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Introduction. Hip fracture is a relatively common injury in elderly and geriatric patients. Early operative care and mobilization reduces morbidity and mortality.

Aim. In this paper we want to show a modern approach of anesthesiologic care of elderly and geriatric patients during surgical procedures for hip fracture.

Method. By reviewing the current literature, guides, and information on the web that address this problem, we aim to provide an overview of the most favorable anesthesia measures and procedures related to injuries that result in hip fracture and the consequent need for urgent fracture surgery. This study is for patients over 65 years of age of both sexes.

The result. The review research will result in a summary of contemporary guidelines that will guide anesthetists in their future work with such patients. Early surgical treatment with the most favorable anesthesia technique, with the ability to rapidly mobilize these patients, offers a chance to reduce morbidity and mortality. Surgery should be performed within 6 hours, with the permissible advantage of regional anesthesia and analgesia.

Conclusion. High levels of management of the injured with a hip fracture require a multidisciplinary approach. Care for this pathology should be performed by experienced surgeons and anesthetists.

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Anesthesia for Bilateral Lung Hydatid Cyst in a Childld

Lejla Dedić Simendić, Selma Sijerčić, Dzenita Ahmetašević

Hydatid disease is a parasitic infection coused by parasite, echinoccocus granulosus, caracterized by cystic lesion in the liver, lungs and reraly in other parts of the body. Bilateral hydatid cyst of lung in a child is a chalenge for surgeon and anestesiologist.
A 4 years female child, weighing 15,5kg, was hospitalized due to a verified bilateral hydatid cysts of lung. At admission to the hospital, the child had mildly dyspnoea, without edema and cyanosis. Preoperative preparation provides corticosteroids, antihistaminics and abendazole. The child premedicated with midazolam. Induction was with fentanyl, propofol and atracurium. Atraumatic intubation was performed with a 4,5mm diametar cuffed endotracheal tube. We were in a dilemma how to ventilate the child as both lungs were affected by pathological process. Considering all advantages and disadvantages of one lung ventilation we decided to proceed with two lung ventilation. During procedure we had no complications. In the case of billateral hydatid cysts in a 4 years child, two lung ventilation is a safer option.

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Pulmonary embolism after major trauma- Case report

Lejla Mujkić, Dženita Ahmetašević

Pulmonary thromboembolic events cause significant morbidity and motality after severe trauma (1). Strategies to prevent fatal pulmonary embolism are widely utilized, but te incidence and risk factors for fatal pulmoary tromboembolism are poorly understood.

Case report:
On 26th of November, 65 years old male patient was urgently admitted to the Surgical clinic with severe injuries caused by a car crash. Multiple injuries were diagnosed: Contusio pulmonum l.dex, PNTX l.sin, Exudatio pleurae bill, Fractura costtarum serriata No I-IX bill, Fractura manubrium et corporis sterni, Haemathoma retrosternale, Ruptura lienis traumatica, Haemathoma retroperitoneale, Fractura phalangis distalis pollicis l.dex (according to ISS-45 /74).
On the following day an urgent surgery of Laparatomia mediana superior et partim inferior, Splenectomio was performed and the patient was transferred to the ICU where he was attached to the controlled mechanical ventilation, hemodynamically and respiratory stable.
During his stay at the ICU, a CT chest scan showed pleural effusion as well as pneumonic infiltration and contusion focuses.
The day after because of hemodynamically unstability with tachyarrhythmia up to 170/ min a CT angiography of a lung artery was performed and showed a lung thromboembolism and therapy was included.
On the 10th of December surgical tracheotomy was performed. Regular checks by surgeon, thoracic surgeon, orthopaedist and pulmologist were performed. Postoperative recovery of the patient at the ICU lasted approximately 35 days. On the 31st of December the patient, hemodynamically and respiratory stable, with neat mental status was transferred to a Clinic for surgery.
Discussion:
Fatal pulmonary embolism appeared to be a potential preventable cause of late mortality after major trauma. Severity of injuries, co-morbidity and BMI were important risk factors for fatal PE after major trauma (2).
Conclusions:
Pulmonary thromboembolism is a dangerous complication of traumatic injury, with varied risk profiles and treatment options.

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Challenges In The Management of Trauma Patient

Senita Beharic

Management of trauma patients is clinically challenging and require multidisciplinary team approach regarding recognised fat embolism syndrome (FES), potentially fatal complication. We present a case of fit and healthy 27-year-old man that was admitted at Orthopaedic Dept with trauma femur fracture /injured after concrete wall fell on him/ fully conscious, respiratory and hemodynamically stable. Fifth day of hospitalisation Ostheosynthesis femoris cum clavo intramedularis was performed. There were no intraoperative complications and he was returned to orthopaedic ward. Few hours postoperatively became restless, petechial rash was noted on neck, developed tachypnea and tachycardia, but there was no focal neurological deficit. Pulse oximetry revealed oxygen saturations dropping to 75% on room air, 5L of oxygen via facial mask maintained saturation at 82-83%. His chest was clear to auscultation, respiratory rate was 28 and he was able to speak in full sentences. Chest X-ray showed ground-grass air space disease and arterial blood gas analysis confirmed hypoxemia. He was shifted to surgical ICU, where was put on mechanical ventilation (high-pressure PEEP). CT chest: bilateral lung contusion/consolidation. Patient remained on midazolam for sedation and fentanyl for pain control, and was provided supportive care. Intensive care management included continuous monitoring of vital signs, standard arterial blood gas analysis, complete blood cell count, coagulation profile, X-ray, echocardiogram (cardiac enzyme levels were negative), CT scan, volume resuscitation with crystalloid fluids. Albumins were administrated to retain blood volume and to bind fatty acids to decrease extent of lung injury. Due to applied complex intensive care treatment, patient health condition gradually improved: on 6th postoperative day he was weaned off pressure support ventilation, extubated and was stable on room air. Control chest X-ray: regression of intrapulmonary changes and pleural effusions. Patient was discharged awake and oriented without neurologic, respiratory or haemodynamic deficits to orthopaedic ward.

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Bleeding delayed due to splenic injury- our experience, case report

Abdulrahman Baker, Elvira Kazagic, Veldin Dzanko

Spleen rupture is the most frequent injury of parenchymatous organs in the abdomen and usually is connect with some fractures in the area of the left rib arch. Delayed bleeding, can lead to severe haemorrhagic juice even days after injury.

Objective: We aim to show our experience with delayed bleeding due to spleen rupuure in a patient after a traffic accident.

Case report:
Patient discharged from ward on admission day, where he had been treated for injuries sustained in a traffic accident nine days earlier. In the afternoon, he felt severe abdominal pain in his left shoulder followed by malaise and general weakness. Admitted to the ward with signs of haemorrhagic juice, an emergency CT scan of the chest and abdomen is done. Indication for emergency laparotomy under general anaesthesia on admission: pale, cold sweat, chilled, hypotensive, tachycardic, impaired pulse, respiratory noise slightly impaired breathing noises, abdominal pain sensitive in left hypochondrium, start with fluid intake and transfusions of DE in the surgical ward. Postoperative flow is complicated by other injuries initially sustained in a traffic accident (lung contusion, fracture of both pubic bones). Postoperative flow is complicated by other injuries initially sustained in a traffic accident (lung contusion, fracture of both pubic bones). During your stay at JIT, polarise effusion in the left thorax. Patient had compromises breathing and the left thorax is punctured and receives about 300ml of tear contents, which is examined microbiologically, finding the puncture neat. When the digestive function is spontaneously lowered, it starts with a orally intake. The patient is destroying postoperative day of transfer to surgical hemodynamic adjusted values of laboratory findings. From the surgery department discharged home recovered.

Conclusion:
With this case report, we wanted to highlight the importance of time for making a diagnosis of delayed bleeding from the spleen, to respond in time, because the rate of care is proportional to survival and complication development.

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Polytrauma with abdominal compartment syndrome

Selimović J., Žilić L.

Research shows that rolling car could severely damage a pedestrian, even if it moved very slowly. Blunt injuries are more often than penetrating injuries, up to 92,8% and the estimated prevalence of liver injury ranges from 1% to 8%. The high-grade liver injuries are demanding due to its massive bleeding in a difficult position to control area, associated with a profoundly altered physiology. An imbalance of dual immune responses, both systemic inflammatory response syndrome (SIRS) and compensatory anti-inflammatory response syndrome (CARS), seems to be responsible for organ dysfunction and increased susceptibility to infections.
We report the case of a 56-year-old female patient who, due to injuries to her chest and abdomen which she got as a pedestrian pressed by a car, was surgically treated at the hospital in Zvornik and then rushed into our institution in severe hemorrhagic shock, with fresh blood in her abdominal drains, and was taken to the operating room again with successfull recovery. Due to multiple abdominal injuries, predominantly liver injuries, she developed abdominal compartment syndrome and the operative wound remained open for 49 days in the intensive care unit, with surgeons bandanging it every 6 hours. The patient was hypotensive, with triple inotropic support and multiple recoupment of blood and blood products. The patient's condition was complicated by multiorgan failure. Appropriate infusion and medical therapy were ordained, laboratory findings were regularly corrected, hemodialysis treatment was performed, ventilation modes were changed according to respiratory abilities of the patient, antibiotic therapy was corrected according to microbiological findings. The patient was haemodynamically and respiratory stable when transferred to the surgical ward and today, 6 months after the injury, is waiting for plastic surgery of the anterior abdominal wall.
Proper management of the polytraumatised patients and a multidisciplinary approach to the patient increase the chances of survival.

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Venous access – that lasts longer-central venous ports

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Introduction

The use of CVC is also limited to 14 to 21 days, especially in immunocompromised or critically ill patients the use of CVC is limited due to their rapid colonization, therefore the use of venous ports took precedence over other venous approaches.

the tunneling venous port maybe with a subcutaneously implanted reservoir or tunneling venous port without a reservoir having surface access connectors.

The implantation of central venous port is an expensive and invasive method for venous access used strictly defined circumstances. The main indication for their use is chemotherapy in pediatric patients, or in the patient that should receive the therapy for a prolonged time as a patient with AIDS, use for prolonged parenteral nutrition and very rare for blood drawing in patients with difficult venous access.

We started implanting these devices as a result of the need for long-term treatment of pediatric patients undergoing chemotherapy.

This study aims to present our experience in the implantation of venous central ports.

Method

for a period of one year, 64 patients were admitted to have a venos implant. After the permission of the ethics committee, the study started. Written consent for every patient was obtained. We analyzed demographic characteristics, indications, difficulties depending on the site also we have compared the blind technique (anatomical approach) versus ultrasound technique and undesired complication.

Results

All procedures were made under general anesthesia. The patient was aged from new borne 24-hour old to 63-year-old. Main indication for implantation of venous port was long term treatment with chemotherapy, the presence of lymph nodes in the neck contributed to the displacement of anatomical structures which in itself caused a higher percentage of complications when using a blind technique, which complications were minimized by the use of an ultrasound guided technique, the second difficulty in implantation of the venous ports which could have contributed to an increase in the percentage of complications, and one requiring preoperative preparation of the patient, was the presence of thrombocytopenia. The most commonly applied site was the right jugular internal vein.

Conclusion

Implantation of the venous port is the only access for a patient with difficult venous access who need long term venous therapy. The use of ultrasound guided techniques contributed to the reduction of complications.

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Mechanical ventilation as therapy after surgical treatmen of a bronchogenic cyst

Šuhreta Tupajić, Lejla Žilić, Jasmila Jakupović

A bronchogenic cyst is the most common cystic formation in the mediastinum. Congenital in origin, it is due to an abnormal branching of the primitive intestine diverticulum in the course of embryogenesis. This is a case report of a male patient age 19 with bronchogenic cyst diagnosticed by CT scen. The patient was treated surgically (Uniportal VATS dex explorativa, Cystectomio, Sutturae bronchi dex cum pleuropatch, Drainage cavi pleurae dex) and a definitive diagnosis was made by a PH. 14-th postoperative day revision surgery was done due to the existence of bronchial fistula (Resutturae bronchi dex cum pleuropatch, Toilettae et drainage cavi pleurae dex duplex). Postoperatively the patient was placed in an intensive care unit and placed on mechanical ventilation for 48 hours as a form of therapy.

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