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Less invasive advanced hemodynamic monitoring

Editorials and Case Studies
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Optimal management of critically ill patients demands accurate and continuous monitoring of their hemodynamic status. Such monitoring can be done by clinical assessment and by using a variety of available advanced monitoring techniques. It seems, however, that in recent years many clinicians have become wary of these techniques, and have either minimized or altogether stopped using any advanced methods of hemodynamic monitoring when managing critically ill patients.

An illustrative case of what I perceive to be insufficient monitoring has been presented in one of the Internet critical care discussion groups. The presentation described a 72 year old man with a significant cardiac history, who became septic (worsening acidosis, oliguria and hypotension) following major surgery that included the removal of a large renal tumor and a necrotic gallbladder. The patient had a positive fluid balance of 20L over 24h, received a ‘bit of noradrenaline’ and eventually had a sudden cardiac arrest. In answer to a comment that the patient may have been under-monitored, the response was: “He was actually on noradrenaline to achieve a target blood pressure of 70 mmHg… we actually monitored metabolic function of the liver (lactate), skin perfusion (clinical assessment), urine output - all good measures of organ function and perfusion rather than simply arbitrary pressures, volumes or flows… So what cardiac output is the right one for this patient? What level of preload is right? What level of lung water is right?... I would be happy to use more monitors if somebody could show me they made a difference…. The biggest problem with ALL the fancy numbers... is that... in the individual patient... you have NO idea what the “best” number is supposed to be”.

One of the main reasons for this ‘back to basics’ movement is the realization that physicians were generally confident of their clinical estimates of hemodynamic variables, but there was no relation between confidence and accuracy. Moreover, experienced physicians were no more accurate than less experienced ones, although they were significantly more confident. Hence the critical care community seemed to have learned at that time that clinical examination and vital signs alone are unreliable in the evaluation of the hemodynamic status and that more advanced monitoring tools may give us new important information that is relevant to patient care. The major value of the PAC lies in its ability to measure the cardiac output (CO), although the conflicting results of the various studies that were aimed at optimizing oxygen delivery made it unclear what target CO values we should aim for, it is still imperative to identify those instances in which a very low or a very high CO is unacceptable by clinical examination alone. However, although identifying a low CO is of great importance, it still does not necessarily point to the right therapeutic decision, e.g., fluids, inotropes, vasopressors. The next step in the hemodynamic assessment is the evaluation of the volume status, which, when the PAC is being used, is based on the measurement of the filling pressures (CVP, PAOP). This indeed is the major flaw of the PAC since filling pressures have been repeatedly shown to be unreliable in assessing the volume of the heart chambers and in predicting the response of the patient to fluid loading.

It is therefore unclear why so many clinicians still rely on filling pressures to guide fluid therapy. What is even more disturbing is the fact that pre-determined levels of filling pressures are being used as end-points of resuscitation, as is the case in both the 2004 Update of the Practice Parameters for Hemodynamic Support of Sepsis in Adult Patients and in the Surviving Sepsis Campaign Guidelines for Management of Severe Sepsis and Septic Shock. The current literature clearly shows that volumetric parameters of preload (e.g., the LV end-diastolic area measured by echo or the global end-diastolic volume – GEDV – measured by the PiCCO), reflect better the status of preload than the filling pressures. In addition, in patients who are on fully controlled mechanical ventilation, functional hemodynamic parameters (i.e., SPV, PPV, SVV, RSVT) are far superior to both filling pressures and volumetric parameters in the prediction of fluid responsiveness. Hence the information provided by the PAC is not reliable enough to identify latent hypovolemia, neither can it reliably prevent fluid overload or alert the care-giver to the development of pulmonary edema. This is especially true in the presence of increased pulmonary microvascular permeability, where aggressive optimization of the cardiovascular status may have grave pulmonary consequences. It is in these situations that a direct measurement of extra-vascular lung water is of utmost importance. These shortcomings of the PAC may explain the claim that the use of the PAC is frequently associated with an aggressive style of treatment which, in turn, leads to adverse outcomes. In particular the PAC was shown in a number of studies to be associated with a high positive fluid balance. Obviously the non-uniformity of patient management in response to hemodynamic data obtained from PAC both within and between institutions may have caused great impact on outcome. This confusion has led to the performance of a few large randomized trials which were aimed at elucidating the effect of the PAC on patient outcome. To date none of these studies have shown that the PAC has any beneficial effect on outcome. The conclusion of all this is that even with improved training in the insertion, interpretation, and implementation of the PAC and the data it generates, the PAC has inherent limitations as an advanced hemodynamic monitoring tool. This is why, as an alternative to expensive clinical trials on the PAC, it was proposed that our limited financial resources for clinical investigation be invested in the development of innovative techniques that may replace the PAC. These techniques are already out there, each trying to prove its superiority over the others. This of course is a very natural and very necessary process. Nevertheless, what is needed more is the realization that advanced hemodynamic monitoring should be further explored rather than completely abandoned. Admittedly, it is difficult to use published data as a basis from which to draw meaningful conclusions about the effects of any monitoring technique on outcome. However, these techniques provide us with a road map which, although incomplete by nature, may be more helpful than having no map at all, provided that one is aware of its pitfalls and limitations. The same logic is being employed whenever any physiological parameter is being measured. The ultimate study that will tell us, once and for all, how to best monitor hemodynamic status at any circumstance, at any time, is not out yet, and may never be. In the meantime we can either do nothing, or further explore the available technologies as well as our understanding of the pathophysiological processes that occur in the critically ill.
Case Study: ‘Failure to Thrive’ post abdominal laparotomy for gallstone ileus

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Patient Diagnosis:
Pulmonary aspiration and acute respiratory distress syndrome following an abdominal laparotomy for gallstone ileus.

Medical History:
An 85 year woman was admitted to the ICU following an abdominal laparotomy for the treatment of a gall stone ileus. A limited small bowel resection was performed and the patient was extubated after 48 hours. Over a period of 72 hours, she “failed to thrive” complaining of abdominal pain and distension. Abdominal examination revealed some rebound tenderness and the absence of bowel sounds. TPN was initiated on the 3rd post operative day and she required CPAP for respiratory support. On the 4th day, the intra-abdominal pressure was measured at 18 mmHg (bladder catheter technique using 100 mL 0.9% saline) and the patient had an abdominal CT scan with contrast that suggested the presence of a post-operative ileus. On her return from the CT scanner, the patient vomited and became very short of breath suggesting pulmonary aspiration. She required emergency intubation and gastric contents were aspirated from the airway. She was immediately bronchosco ped and her airway washed out with 250 ml of 0.9% saline. Subsequently, she was difficult to ventilate, requiring pressure controlled ventilation (inspiratory pressure of 20 cmH2O, PEEP of 15 cmH2O, inspiratory time 2 seconds, respiratory rate 15 breaths/minute, FiO2 of 0.8) to achieve a PaO2 of 58 mmHg. She was also hypotensive despite 7.5 mcg/kg/min of dobutamine (80/45, MAP 55 mmHg) and her CVP was 15 mmHg (central venous saturation 66%). An ECHO cardiogram (poor views) suggested a hyperdynamic left ventricle that was reasonably well filled. A chest X-ray demonstrated bilateral fluffy infiltrates consistent with an acute lung injury secondary to aspiration. Her urine output had fallen to less than 0.5 ml/kg/hr and had a metabolic acidosis with a blood lactate level of 2.9 mmol/l.

Clinical Course:
At this stage, the attending physician wished to measure this patient’s cardiac output, preload (GEDV/ITBV) and extravascular lung water so as to optimise cardiovascular performance. He especially wanted to know the lung water in order to understand how aggressive he should be with fluid therapy. Before obtaining any haemodynamic data other than the CVP and central venous saturation, he was inclined to give this lady at least 20 ml/kg of colloid (4% albumin) in an attempt to improve her haemodynamics. He was also concerned to administer a vasopressor (dopamine or nor-adrenaline) to such an elderly patient without some precious monitoring of cardiac output. The resident medical officer inserted a PICCO arterial catheter into the right femoral artery without difficulty. The first set of measurements (mean of three) was as follows: cardiac index 1.8l/min.m2, GEDI 880 ml/m2, ITBI 1100 ml/m2, SVV 7%, ELWI 18 ml/kg (see Table 1). The intensive care specialist interpreted these data as suggesting that although preload was probably adequate, flow was too low. Given the elevated ELWI (normal < 10 ml/kg), he did not think further fluid therapy was appropriate since she was unlikely to be “fluid responsive” (GEDI/ITBI at the upper limit of normal, SVV < 10% during controlled ventilation [tidal volume 7ml/kg]). Instead, the dose of dobutamine was increased to 15 mcg/kg/min and a low dose nor-adrenaline infusion (0.15 mcg/kg/min) was added to increase the MAP to greater than 70 mmHg. Three units of blood were administered to maintain a haematocrit of 32% and hydrocortisone 50 mg qid was also prescribed.

Subsequently:
The patient was treated by fluid restriction, a 20% albumin infusion (12 ml/hour to maintain serum albumin greater than 30 G/l) and a frusemide infusion (2-15 mg/hour to maintain urine output greater than 150 ml/hour). After 48 hours of therapy, the patient was in negative fluid balance and inotropic and vasopressor support could be reduced over the next five days as oxygenation improved. The patient was successfully extubated on day 8.

Summary:
Advanced haemodynamic monitoring with the PiCCO allowed the attending physicians to manipulate fluid therapy, inotropes/vasopressors and diuresis in such a way as to improve haemodynamics and pulmonary gas exchange. Excessive fluid therapy was avoided and active measures were taken to maintain colloid osmotic pressure and to obtain negative fluid balance.

Table 1: PiCCO Measurements

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Day 0</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 4</th>
<th>Day 5</th>
<th>Day 6</th>
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<td>CVP</td>
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<td>680</td>
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<td>825</td>
<td>830</td>
<td>790</td>
</tr>
<tr>
<td>ITBI</td>
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<td>850</td>
<td>1010</td>
<td>1030</td>
<td>1040</td>
<td>990</td>
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<tr>
<td>SVV</td>
<td>8</td>
<td>12</td>
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<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
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<td>1.8</td>
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<td>3</td>
<td>3.5</td>
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<td>3.1</td>
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<tr>
<td>ELWI</td>
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<td>18</td>
<td>14</td>
<td>12</td>
<td>10</td>
<td>9</td>
</tr>
<tr>
<td>PaO2/FiO2</td>
<td>95</td>
<td>125</td>
<td>210</td>
<td>270</td>
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<td>330</td>
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<td>PEEP</td>
<td>15</td>
<td>15</td>
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<td>12</td>
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</tbody>
</table>

Interventions
20 ml/kg Noradrenaline albumin off Frusemide infusion Dobutamine weaned 20% albumin Frusemide infusion off

CVP Central Venous Pressure; GEDI Global End Diastolic Volume Index; ITBI Intrathoracic Blood Volume Index; SVV Stroke Volume Variation; CI Cardiac Index; ELWI Extra Vascular Lung Water Index; PaO2/FiO2 Partial, Pressure Oxygen divided by Fraction of Inspired Oxygen; PEEP Positive End Expiratory Pressure.
Patient Diagnosis:
Acute respiratory failure on a background of acute myeloid leukemia.
Medical Diagnosis: A 55 year old man with a previous history of acute myeloid leukemia was admitted to the ICU because of acute respiratory failure. He had gained 7kg in weight the previous week on the ward where he was diagnosed as having a gastro-enteritis related to the chemotherapy (cytosar). His central venous pressure measured on the ward was 32cm H2O. The tentative diagnosis hence was acute lung edema and a bolus of 40 mg Frusemide was administered.

Clinical Course:
On admission to ICU he was in distress with a respiratory rate of 34 breaths per minute. Further examination of his vital signs showed a core temperature of 34.4°C, a MAP of 59 mmHg and a sinus tachycardia of 140 beats per minute. Because of clinical exhaustion, he was intubated and mechanically ventilated (machine rate 24 x 500ml, inspiration: expiration ratio 1:1 and a PEEP of 15) however oxygenation was poor with a pO2/FiO2 ratio of 115. Breaths sounds were diminished and fine crackles were heard over both lungs. The abdomen was tender, firm and distended with an intra-abdominal pressure of 26 mmHg. Neurological and extremities examination were unremarkable, however, the patient was oliguric. A PICCO catheter was inserted and confirmed the diagnosis of septic shock with a cardiac index of 5.1 l/min/m2 (normal range 3.0-5.0) and low SVRI. The CVP was 24 mmHg with a SVV of 15% and a GEDI of 650 ml/m2, confirming intravascular under-filling and fluid responsiveness, despite the high CVP. Blood cultures grew enterococcus faecalis and clostridium difficile toxins were positive on a recent stool sample. The patient's MAP was initially responsive to fluids together with doses of noradrenaline up to 1mcg/kg/min and dobutamine up to 15 mcg/kg/min, however he soon became anuric and the cumulative fluid balance was positive for another 12 l. Due to ongoing fluid resuscitation and profound capillary leak his pO2/FiO2 ratio further deteriorated to 75. At that time CVP was 29 mmHg, MAP 65 mmHg, SVV 13%, GEDI 780 ml/m2, IAP 28 mmHg, but ELWI increased from 12 initially to 17 ml/kg.

Subsequently:
The patient was diagnosed having an abdominal compartment syndrome with abdominal sepsis related to the toxic megacolon following diffuse clostridium difficile pseudomembraneus colitis. On abdominal CT the caecum diameter was 18cm with wall thickening up to 3.5cm, the whole colon was infiltrated and dilated. Therefore the option was taken to perform a total colectomy and decompressive laparotomy with temporal abdominal vacuum assisted fascial closure. After decompression despite the good CI and SVV parameters the patient was put on CVVH with aggressive ultra-filtration combined with albumin substitution because of the high ELWI and low pO2/FiO2 ratio. Over the following days his condition improved with a decrease in IAP to 16 mmHg and ELWI to 13 ml/kg and a concomitant rise in pO2/FiO2 ratio to 175. The CVP remained stable at 18 to 22 mmHg while SVV normalised at 10-13%.

Summary:
• Traditional filling pressures are erroneously increased in incidences of high intra-thoracic pressures (related to IAP or PEEP). In this situation volumes are better preload indicators.
• SVV is NOT an indicator of preload but a marker of fluid responsiveness (in fully ventilated patients).
• Measurement of flow (CI) does not allow you to discriminate between over or under-filling.
• After the initial resuscitation phase an even more important question that needs to be answered is: “when to stop filling?”
• ELWI can guide you to get rid of the excess fluids.

Monitoring of macro hemodynamics

Case Study:
Why filling pressures alone are not enough
Manu Malbrain, MD
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![Graph showing SV, SV change, volume responsiveness, target goal, volume overload, and ELWI](image_url)

Relation of preload and stroke volume in different fluid loading conditions

- SV: Stroke Volume
- ΔSV: Change in Stroke Volume
- ΔV: Change in Volume
- ΔS: Change in SV
- Target Goal
- Volume Responsive
- Volume Overload
- Preload

<table>
<thead>
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<th>Preload</th>
<th>SV</th>
<th>ΔV</th>
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<th>ΔS</th>
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<td>Volume Responsive</td>
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<tr>
<td>Target Goal</td>
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<tr>
<td>Volume Overload</td>
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</table>
Detection of tissue hypoxia

Editorial:

Central venous oxygen saturation:
Tackling tissue hypoxia at the front-line

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Mixed or central venous oxygen saturation (S(c)vO₂) in Sepsis has long been regarded as an end point of low impact in clinical decision making for the septic patient, because septic shock unlike other shock states was considered to be more or less exclusively “hyperdynamic” as reflected in a high venous saturation. It was clearly the merit of the “Early Goal Directed Therapy in Severe Sepsis and Septic Shock Study (EGDT)” by Emanuel Rivers that sensitized critical care providers to the fact that there is an early phase of supply dependency in sepsis during which central venous oxygen (ScvO₂) saturation is likely to be extremely useful. It was not only to detect but also to guide treatment of global tissue hypoxia (1).

Why is mixed venous saturation a good surrogate for ‘supplydependency’?

It is now well accepted that hemodynamic assessment by means of arterial blood pressure, heart rate, central venous pressure, and urinary output may fail to detect persistent tissue hypoxia, as patients who are resuscitated to having normal vital signs frequently continue to exhibit increased lactate levels – indicative of anaerobic glycolysis – along with a ScvO₂ below 70% (2). These signs of persisting tissue hypoxia indicate the need for additional resuscitation as low ScvO₂ was not only associated with a 57% in-hospital mortality rate, but more aggressive therapy to restore ScvO₂ with fluids, RBC transfusion, and inotropic agents reduced mortality to 44% in the EGDT trial.

The Fick equation, which defines O₂ consumption as the product of cardiac output times the arterio-venous O₂ difference (VO₂ = CO * ((CaO₂ – ScvO₂) can be transformed (if dissolved O₂ is neglected) to:

\[ \text{ScvO}_2 = \frac{\text{SaO}_2 - \text{VO}_2 / K^{*}\text{CO}^{*}\text{Hb}}{\text{K}^{*}\text{CO}^{*}\text{Hb}} \]

Thus, mixed venous saturation is influenced by SaO₂ on the one hand and by the balance between VO₂ and CO and Hb on the other. If (as it is usually the case in the ICU setting) SaO₂ is normal, then ScvO₂ reflects the oxygen supply-demand ratio of the tissues. In other words, any decrease in ScvO₂ below the normal range of approximately 70% is an alarm signal indicative of increased extraction, reflecting limited O₂ supply.

Does ScvO₂ reliably reflect VO₂? The question as to whether ScvO₂ is equivalent to VO₂ has been a matter of debate over the years and has been addressed in several studies (3,4,5). These studies have consistently shown that ScvO₂ values are approximately 5-10% higher than SvO₂ values in particular in shock states, which is likely to be secondary to the contributions of e.g. deoxygenated blood from the coronary sinus or increased extraction, e.g. in the hepatosplanchnic region. Recognizing this consistent difference is of primarily academic interest but does not argue against the clinical usefulness of ScvO₂. Unlike other end points of resuscitation ScvO₂-guided cardiocirculatory support has been shown to affect morbidity and mortality in a cohort of patients under conditions of an appropriately designed clinical severe sepsis trial (1) and has been associated with improved outcome as part of a Standard Operating Procedure (SOP) to treat septic shock (6). Consequently, the approximate 5% numeric difference between ScvO₂ and SvO₂ has prompted the writing committee of the Surviving Sepsis Campaign in its actualized version to recommend obtaining an SvO₂ of 65% or an ScvO₂ of 70%, respectively, in the resuscitation portion of its management of patients with severe sepsis and septic shock bundle (7).

ScvO₂ – is discontinuous equivalent to continuous monitoring? Systemic tissue oxygenation should be monitored and optimized in critically ill patients as many of these patients continue to have significant global ischemia and/or cardiac dysfunction as indicated by reduced ScvO₂ and elevated lactic acid concentrations (8). Once identified, these patients require aggressive management especially during the initial hours after admission. Thus, intermittent monitoring every 4-6 hours is unlikely to help to guide cardiocirculatory support in persistent tissue hypoxia in the window which determines generation of inflammatory mediators and mitochondrial impairment leading to cellular/tissue injury (1,9). Thus, while obtaining a central-venous specimen for blood gas analysis should be part of each placement of a central line in the critically ill, persistent signs of tissue hypoxia should then prompt the continuous monitoring of the ScvO₂.

Albeit not validated in prospective controlled studies, additional surrogates of impaired oxygen supply to peripher al tissues, such as the plasma disappearance rate of indocyanine green might help to unravel persistent tissue hypoxia, e.g. in the hepatosplanchnic area, because normalization of ScvO₂ improved outcome, but hospital mortality remained as high as 44% even in the interventional arm of the EGDT trial (1).

References


Each drop of the S(c)vO₂ below the normal range is an alarm signal of an increased oxygen extraction and hence a sign of restricted oxygen delivery to the tissues.

\[ \text{VO}_2 = \text{O}_2 \text{ consumption} \]
\[ \text{DO}_2 = \text{O}_2 \text{ delivery} \]
Detection of tissue hypoxia

Case Study: Early recognition of oxygen supply dependency by ScvO2

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Case history
A 63 year old female was admitted to a district general hospital’s medical ward with shortness of breath, feeling generally unwell and complaining of abdominal and chest pain. Her medical history revealed only well controlled hypertension. Diagnostic investigations found multiple pancreas pseudocysts, choledolithiasis and bilateral hydrothoraces. Ultrasound guided aspiration and drainage of the pseudocyst and the hydrothorax was performed. Five days later her general condition deteriorated and the abdominal CT showed free air under the diaphragm and refill of the pseudocysts. For an emergency operation and further care she was transferred to our tertiary care university hospital.

Clinical course
After arrival to the surgical ward and following the necessary investigations immediate laparotomy was performed. Although a communication between the pseudocyst and the pleural cavity was revealed gastro-intestinal perforation was not found. A previously inserted drain was removed, cholecystectomy was performed and the abdomen was thoroughly washed with saline. Following 24 hour intensive care observation the patient was discharged back to the surgical ward. Eight days later her condition suddenly deteriorated and she was again referred to ICU staff. Due to severe hypotension, altered mental status and oliguria she was immediately readmitted to the intensive care unit.

On arrival her SAPS II was 29 and by definition she had severe sepsis: high white cell count (40 G/l), tachycardia (>120/ min), hypotension (60/- mmHg) and suspected peritonitis. She was drowsy but on examination her Glasgow Coma Score was 13. Oxygen therapy as commenced, radial arterial and right internal jugular central venous lines were inserted. Whilst fluid resuscitation and norepinephrine treatment was started to regain acceptable blood pressure, transpulmonary invasive haemodynamic monitoring was also commenced (PICCO). After taking a blood sample for blood culture empiric antibiotic treatment was started with imipenem and amikacin. Due to severe hypotension, altered mental status and oliguria she was again referred to ICU staff. Due to severe hypotension, altered mental status and oliguria she was immediately readmitted to the intensive care unit.

However, her ScvO2 remained low and despite acceptable global haemodynamic and haemoglobin results the patient deteriorated and she was again referred to ICU staff. Due to severe hypotension, altered mental status and oliguria she was immediately readmitted to the intensive care unit. On arrival her SAPS II was 29 and by definition she had severe sepsis: high white cell count (40 G/l), tachycardia (>120/min), hypotension (60/- mmHg) and suspected peritonitis. She was drowsy but on examination her Glasgow Coma Score was 13. Oxygen therapy as commenced, radial arterial and right internal jugular central venous lines were inserted. Whilst fluid resuscitation and norepinephrine treatment was started to regain acceptable blood pressure, transpulmonary invasive haemodynamic monitoring was also commenced (PICCO). After taking a blood sample for blood culture empiric antibiotic treatment was started with imipenem and amikacin. Due to aggressive resuscitation (2500 ml crystalloid and 500 ml colloid) her haemodynamic parameters quickly stabilised (blood pressure increased to 100/70 mmHg) and urine output picked up from 30 ml/h to 100 ml/h within a few hours. The norepinephrine was stopped at that point. However, her ScvO2 remained low and despite acceptable global haemodynamic and haemoglobin results the patient received 3 units of packed red blood cells. Parameters before and after transfusion and on the following day are summarised in Table 1. Whilst none of the haemodynamic parameters changed after the transfusion ScvO2 improved as well as haemoglobin. Serum lactate and procalcitonin also decreased for the next day.

Her abdomen remained soft and neither clinical nor radiological findings indicated the need of acute surgical intervention. However, based on the result of the chest X-ray a right sided chest drain was inserted. On day-2 microbiology confirmed Stenotrophomonas maltophilia from blood culture and the pleural fluid sensitive for the antibiotics started earlier. During the following days the patient’s condition slowly improved and she was discharged to the surgical ward on day 5.

Conclusion
It is well accepted practice to allow haemoglobin to drop as low as 7 g/dl in septic patients without ischemic heart disease. However, we should not follow this 7 g/dl recommendation as the “golden pot under the rainbow”. There is increasing evidence that early resuscitation and goal directed therapy to stabilise oxygen demand and consumption is vital in the early phase of shock. In the current case, despite adequate fluid resuscitation, moderately low haemoglobin compromised the balance of oxygen delivery and consumption as indicated by low ScvO2 despite normal global haemodynamic and arterial blood gas figures. Although, we can seldom be certain that we have made the right decision, we have to make sure that we have done everything to achieve it. This case is an example of how ScvO2 indicated the possibility of haemodynamic instability due to low haemoglobin earlier than global haemodynamic parameters. There is increasing evidence that delaying adequate oxygen delivery by hours or even minutes to the tissues and organs in critically ill patients can cause irreversible damage and increase mortality. Measuring ScvO2 regularly or even continuously, as well as continuous monitoring of the vital functions is certainly the way forward in critical care, may help us in early decision making regarding haemodynamic support and fine tuning of the balance between oxygen delivery and demand.

Table 1

<table>
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<th></th>
<th>Before transfusion</th>
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<tr>
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<tr>
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Why do we measure Extravascular Lung Water? Many critical conditions lead to pulmonary edema. During systemic inflammation and sepsis, acute lung injury (ALI), burns, pancreatitis, multiple trauma with severe blood loss, ischemia-reperfusion injury and other states, the release of inflammatory mediators may enhance pulmonary microvascular pressure and permeability, thus promoting the accumulation of fluid in the lungs. In contrast to hyperpermeability states, during cardiac failure the main mechanism for edema includes increased hydrostatic pressure in the pulmonary circulation. However, both cardiogenic and non-cardiogenic origins of pulmonary edema have one common sign – increased extravascular lung water (EVLW). Moreover, both types of lung edema are accompanied by a high mortality rate that necessitates a search for strategies that will improve our therapy. Consequently, reliable tools for monitoring lung fluid balance are increasingly needed in modern intensive care.

The amount of edema is, however, difficult to estimate at the bedside. Clinical examination, chest radiography, and blood gases have proven to be of limited significance in quantifying pulmonary edema. Therefore, several techniques have been developed to assess EVLW. Among them, thermo-dye dilution and single transpulmonary thermodilution are used most frequently.

How can we determine Extravascular Lung Water? Originally, the thermo-dye dilution (TDD) was used for measuring lung water. This technique is based on the simultaneous detection of two indicators with different properties: a freely diffusible indicator (“cold”) and a dye (indocyanine green), which binds to the plasma albumin. Based on the Stewart-Hamilton principle, “cold” and dye allow the calculation of the intrathoracic thermal volume (ITTV) and the intrathoracic blood volume (ITBV), respectively. The difference between the two distribution volumes is used for estimation of EVLW (EVLW = ITTV – ITBV). The TDD method has been validated in animal models of lung edema and in the clinical setting. However, TDD is relatively time consuming, cumbersome and expensive, thus motivating the search for a reasonable bedside alternative.

Employing the PICCO technique based on the injection of a single thermo-indicator that can be detected with an indwelling arterial thermodilution catheter, is an appealing idea. EVLW determined by single transpulmonary thermodilution (STD) can be calculated using the specific analysis of the thermodilution curve. In addition to STD, combined with pulse contour analysis of cardiac output also gives the possibility of displaying a variety of cardiac pulmonary variables, thus expanding the options for hemodynamic monitoring.

Recent experimental and clinical studies have shown that EVLW assessed by STD demonstrates good reproducibility and close agreement on the double indicator technique and postmortem gravimetry. Compared with both TDD and right heart catheterization, STD is simpler to apply, less invasive and more cost-effective, all factors that make it more suitable for use at the bedside. However, the detection of EVLW by the thermodilution method can be impaired by several factors, for example, severe changes in cardiac output and pulmonary blood volume, accumulation of chest exudates, and positive end-expiratory pressure (PEEP). Therefore, determination of EVLW by TDD and STD requires repeated measurements.

What do we measure Extravascular Lung Water for? Several categories of both pediatric and adults intensive care patients have been shown to benefit from monitoring EVLW, including any patient who has cardiogenic and non-cardiogenic pulmonary edema, massive fluid shifts and severe changes in microvascular permeability. Thus, I consider any critical illness that results in shock and tissue hypoperfusion refractory to fluid resuscitation is a valid subject for EVLW monitoring. In addition, EVLW monitoring may also be of value in patients undergoing major surgical procedures, particularly, cardiothoracic surgery and organ transplantation.

In septic shock, invasive cardiovascular monitoring with arterial catheterization and “beat-to-beat” analysis facilitates the administration of large quantities of fluids, vasopressor/inotropic support, and ventilatory settings. Hence, such monitoring has recently been recommended as one of the guiding parameters for hemodynamic support in sepsis. During sepsis-induced pulmonary edema, accumulation of EVLW occurs before changes in blood gases, chest radiogram and pressure variables such as right atrial pressure (RAP) and pulmonary artery occlusion pressure (PAOP). It is important to emphasize that the latter variables are in fact unspecific diagnostic tools and influenced by a variety of factors. In contrast to RAP and PAOP, EVLW in severe sepsis correlates with markers of lung injury such as the oxygenation ratio, lung compliance, and the number of affected roentgenogram quadrants, as well as with the total lung injury score. During the onset of septic shock, EVLW is increased in three out of four patients. Therefore, in sepsis, EVLW serves as a marker of ALI, provides a valid estimate of the interstitial water content in the lungs and might become an alternative to RAP and PAOP in the management of fluid resuscitation.

To administer the correct therapeutic intervention in patients with systemic inflammatory response and concomitant heart failure, it is important to distinguish between cardiogenic and non-cardiogenic pulmonary edema. For these purposes, we can use the pulmonary vascular permeability index (PVPI) calculated in PICCOplus technology as EVLW / Pulmonary Blood Volume (PBV). When EVLW / PBV exceeds 3, permeability edema is suspected. In contrast, when EVLW/PBV is within the normal value (1-3) cardiogenic pulmonary edema should be suspected.

In critically ill patients, both EVLW and PVPI have important prognostic values and increase in non-survivors. When evaluated in combination with other cardiopulmonary parameters, EVLW may reduce the duration of mechanical ventilation and shorten the periods of stay in ICU and hospital. Moreover, measurement of EVLW can support the diagnosis and therefore improve the clinical outcome of pulmonary edema, if used cautiously in combination with treatment protocols known to hasten its resolution. In patients with increased EVLW, such protocols include fluid restriction, administration of diuretics and inotropes, PEEP, and so on.

Summary: Many critical states can be accompanied by the accumulation of EVLW and development of pulmonary edema. Among the various methods for measurement of EVLW at the bedside, single transpulmonary thermodilution may be most useful. Recent clinical studies have shown that EVLW correlates with the severity of lung injury and appears to have a prognostic value, especially in sepsis and ALI. Moreover, monitoring EVLW can be an important tool for prevention and directed treatment of pulmonary edema of both cardiogenic and non-cardiogenic origins. Thus, the success of our therapy often depends on the correct answers to the following questions: (1) how much water is in the lungs, (2) why is it there, and (3) what can we do to return lung water to the normal limits. I suspect that if we can answer these questions correctly, the measurement of EVLW and the individual therapeutic implications can contribute to improvement of outcome in many critically ill patients.
Case Study: Incipient pulmonary edema in systemic inflammatory response after multiple trauma

Professor Enrique Fernandes Mondejar, MD, Associate Professor
Intensive Care Unit,
Hospital Universitario Virgen de las Nieves
Granada, Spain

Medical History
A 28 year old man suffering multiple trauma after an accidental fall from approximately 10 meters.
Injuries included:
- Complex facial fractures
- Open fractures of left humerus and left tibia
- Closed fracture of right femur
- Transverse fracture of sacrum
- Absence of pulse in left arm

On arriving to our Trauma Centre Emergency Room (35 min after the accident) the patient was alert, conscious and breathing spontaneously. Pulse oxygen saturation was 96% with an oxygen mask of 40%. The hemodynamic status was strictly volume dependent and the patient was intubated and connected to a mechanical ventilator. Arterial blood gas analysis on mechanical ventilation with FiO2 0.4 and zero-PEEP: PaO2 175, PaCO2 33, pH 7.29, Base deficit -5.5, lactate 2.3 mmol/L and haemoglobin 9.8 gr/dl. After radiological and sonographic explorations to rule out internal injuries the patient was transferred to OR (approximately 30 minutes after admittance) for external fixation of long bone fractures and vascular repair of left axilla artery.

First 24 hours
The principal problems early after OR were:
1. Extreme hemodynamic instability requiring massive volume replacement (positive balance of 12 litres in the first 12 hrs) and vasoactive support with noradrenalin in increasing doses (from 0.35 to 2.7 µg/kg/min).
2. Coagulopathy requiring replacement of hemo-derivates:
   - Plasma 1500 ml, Red Blood Cells 2000 ml, platelets 12 units.

After 12 hrs the hemodynamic picture remained unstable with noradrenalin at 1.5 µg/kg/min (BP: 120/70 mmHg, CVP 175, PaCO2 33, pH 7.37, Base deficit -6.5, lactate 2.3 mmol/L and haemoglobin 9.8 gr/dl). After radiological and sonographic explorations to rule out internal injuries the patient was transferred to OR (approximately 30 minutes after admittance) for external fixation of long bone fractures and vascular repair of left axilla artery.

Fluid therapy for the next days
Based on a relatively low CVP in a patient with high vasopressor support and normal lung function, fluid therapy for the next 24 hours was planned to maintain the fluid infusion as necessary and to maintain the noradrenalin infusion or, if possible to reduce it. A PICCO catheter was inserted which provided the following data:
- Cardiac Index (CI): 4.7 l/min/m²
- Global End-Diastolic Volume (GEDV): 630 ml/m²
- Extravascular Lung Water (EVLW): 13 ml/kg
- Stroke Volume Variation (SVV): 17%

The optimum approach to fluid therapy is based on adequate interpretation of several physiologic parameters. EVLW gives unique and crucial information about lung fluid accumulation that can not be obtained in any other way.

Summary
1. Although this patient’s oxygenation and chest radiograph were completely normal, the lungs were moderately edematous (EVLW 13 ml/kg, normal <10 ml/kg). This is of no surprise as it is known that both oxygenation and chest radiograph are not sensitive when detecting incipient pulmonary edema.
2. Fluid therapy for the next days
   Based on a relatively low CVP in a patient with high vasopressor support and normal lung function, fluid therapy for the next 24 hours was planned to maintain the fluid infusion as necessary and to maintain the noradrenalin infusion or, if possible to reduce it. A PICCO catheter was inserted which provided the following data:
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   The optimum approach to fluid therapy is based on adequate interpretation of several physiologic parameters. EVLW gives unique and crucial information about lung fluid accumulation that can not be obtained in any other way.

3. Including EVLW monitoring in the fluid therapy decision tree allow us to take the right decisions according to the pathophysiological patient situation.
Case Study: Postoperative Volume Management (Total Hip Replacement)

Azriel Perel, MD,
Professor and Chairman
Department of Anesthesiology and Intensive Care
Sheba Medical Center, Tel Aviv University
Tel Hashomer, Israel

Assessment of pulmonary edema

Situation:
- 63 year old patient undergoing total hip replacement
- uneventful course of anaesthesia
- stable hemodynamics intraoperatively; after 4h surgery clinical signs of pulmonary edema with oxygen saturation (SaO₂) dropping below 80%
- recovery room: blood pressure 63/40 mmHg, heart rate 137 bpm
- stabilization of blood pressure with dobutamine and epinephrine
- blood gas analysis (ventilation with 100% oxygen): pH 7.23 (norm 7.35-7.45), pCO₂ 42 mmHg (norm 35-46), pO₂ 75 mmHg (norm 70-104), Hct. 37% (norm 40-52%)

Possible diagnoses:
- acute myocardial infarction with cardiac decompensation (cardiogenic shock)
- hypovolemia (hypovolemic shock)
- reaction to transfusion (anaphylactic shock)
- reaction to bone cement (anaphylactic shock)
- pulmonary embolism

Hemodynamic stabilization:
- installation of PICCO-system: CI 1.91, ITBI 779, ELWI 23, SVV 22%
- despite the high lung water, volume loading started because of low ITBI and high SVV
- reduction of catecholamine dosage with volume therapy
- TEE showed a hyperdynamic left ventricle with small end-diastolic volume

Further course:
- further decrease and eventually cessation of catecholamines
- fluid withdrawal after hemodynamic stabilization
- extubation on 2nd postoperative day with normal ELWI
- at the time of extubation there were still radiologic signs of significant pulmonary edema
- extubation was nevertheless successful

Conclusion:
- detection of hypovolemia by means of PICCO (low preload volume, high volume responsiveness) as the cause of hemodynamic instability.
- recognition and subsequent monitoring of pulmonary fluid accumulation.
- goal-oriented fluid- and catecholamine therapy with PiCCO: initial volume loading, and then volume withdrawal after circulatory stabilization.
- despite persisting radiologic signs of pulmonary edema the low ELWI showed an acceptable pulmonary water content allowing a successful extubation (Lung Water as a weaning parameter).
Systemic inflammatory response and developing multiple organ failure is present in a number of our intensive care admissions and is still the leading cause of death in critically ill patients. Thus it contributes to a considerable quantity of medical and financial resources spent today. The presence of hepatic dysfunction on admission (as high as 10-25%) or the development of hepatic dysfunction during the ICU stay (around 15% in an Austrian collective of 40,000 patients) negatively alters outcome to a greater extent than any other organ failure. In this context, recent clinical studies have focused on the ability of clearing and eliminating circulating endotoxins and bacteria, as well as on metabolism of endogenously produced biomarkers and mediators of inflammation, showing that these features are severely compromised even during early stages of sepsis and contribute to or limit recovery from these syndromes. It is thus interesting and astonishing that hepatic dysfunction has not received a comparable degree of attention from the critical care community.

The underlying reason for this may be twofold. Firstly, hepatic dysfunction was a well known phenomenon and it was commonly accepted that patients with a prolonged ICU stay developed jaundice during their stay without considering this derangement as potential deleterious. Secondly, it is still commonly believed among critical care clinicians – with exception of those actually involved – that hepatic (dys)function is difficult to assess, monitor and potential specific treatment is questionable whether we still consider it reasonable or appropriate to provide rational therapy for critically ill patients on the issue that splanchnic blood flow and concomitant acute hepatic dysfunction is intimately associated with fluid regimes (for e.g. hypovolemia or fluid restriction might actually cause a decrease in ICG metabolism) and other interventions such as catecholamine support. Thus it is clinically relevant to adapt therapeutic strategies accordingly. In conclusion, it is crucial to identify patients who are at risk of developing hepatic failure during their ICU stay. Therefore it is questionable whether we still consider it reasonable or appropriate to provide rational therapy for critically ill patients based on assumptions and derived parameters such as lactate metabolism and coagulation factors to quantify liver function, while ICG measured at the bedside with the LIMON has proved to be superior to conventional markers of hepatic dysfunction. Monitoring ICG metabolism by the LIMON represents an important step to translate research findings into improved care at the bedside.

ICG is an inert compound which binds mainly to albumin and lipoproteins in plasma. It is selectively taken up (energy independent) by hepatocytes and excreted into the bile via an energy dependent (ATP-consuming) transport system. ICG is neither metabolized nor does it undergo entero-hepatic recirculation. Thus, the disappearance rate of ICG from plasma into bile reflects hepatocyte excretory function and energy status. As a result of these features ICG was introduced to assess liver function in brain-dead organ donors and transplant recipients. Because of the precise estimate of liver (dys)function in this population, its use was also transferred into other critically ill entities, but failed to be adopted into widespread use due to the time consuming and workload intensive laboratory assessment technique. In the meantime it has become clinically feasible to monitor ICG metabolism as easily as pulsoxymetry with a finger clip – by pulse dye densitometry (LIMON-Technology), making the measurement non invasive. The results obtained have been shown to be as reliable as serial blood sampling and analysing methods. Together with the improvement of ICG measurement, indications and experience has grown and the use of ICG nowadays allows a modification of treatment algorithms, risk stratification and a prognostic estimate of a critically ill patient’s course. As well, these techniques have recently been implemented in the evaluation of partial hepatectomy for liver metastases and carcinoma resection surgery, and living-donor-liver-transplantation. Together with volumetric CT scans ICG evaluation has become one of the main parameters for the planning of the surgical approach in these conditions. From my own clinical experience I would like to reinforce the issue that splanchnic blood flow and concomitant acute hepatic (dys)function is intimately associated with fluid regimes (for e.g. hypovolemia or fluid restriction might actually cause a decrease in ICG metabolism) and other interventions such as catecholamine support.

**Editorial:**

**Bedside assessment of hepatic function and hepatic functional reserve – the time has come for all!**

Claus G. Krenn, MD, Professor, Austrian Society of Anaesthesiology, Reanimation and Intensive Care (ÖGARI), Department of Anaesthesiology and General Intensive Care, Medical University Vienna, Austria

ICG is an inert compound which binds mainly to albumin and lipoproteins in plasma. It is selectively taken up (energy independent) by hepatocytes and excreted into the bile via an energy dependent (ATP-consuming) transport system. ICG is neither metabolized nor does it undergo entero-hepatic recirculation. Thus, the disappearance rate of ICG from plasma into bile reflects hepatocyte excretory function and energy status. As a result of these features ICG was introduced to assess liver function in brain-dead organ donors and transplant recipients. Because of the precise estimate of liver (dys)function in this population, its use was also transferred into other critically ill entities, but failed to be adopted into widespread use due to the time consuming and workload intense laboratory assessment technique. In the meantime it has become clinically feasible to monitor ICG metabolism as easily as pulsoxymetry with a finger clip – by pulse dye densitometry (LIMON-Technology), making the measurement non invasive. The results obtained have been shown to be as reliable as serial blood sampling and analysing methods. Together with the improvement of ICG measurement, indications and experience has grown and the use of ICG nowadays allows a modification of treatment algorithms, risk stratification and a prognostic estimate of a critically ill patient’s course. As well, these techniques have recently been implemented in the evaluation of partial hepatectomy for liver metastases and carcinoma resection surgery, and living-donor-liver-transplantation. Together with volumetric CT scans ICG evaluation has become one of the main parameters for the planning of the surgical approach in these conditions. From my own clinical experience I would like to reinforce the issue that splanchnic blood flow and concomitant acute hepatic (dys)function is intimately associated with fluid regimes (for e.g. hypovolemia or fluid restriction might actually cause a decrease in ICG metabolism) and other interventions such as catecholamine support.

Thus it is clinically relevant to adapt therapeutic strategies accordingly. In conclusion, it is crucial to identify patients who are at risk of developing hepatic failure during their ICU stay. Therefore it is questionable whether we still consider it reasonable or appropriate to provide rational therapy for critically ill patients based on assumptions and derived parameters such as lactate metabolism and coagulation factors to quantify liver function, while ICG measured at the bedside with the LIMON has proved to be superior to conventional markers of hepatic dysfunction. Monitoring ICG metabolism by the LIMON represents an important step to translate research findings into improved care at the bedside.
A 35 year old male admitted from another hospital following probable acetaminophen (paracetemol) induced hepatic failure. He gave a history of having consumed excess acetaminophen over several days. His past medical history was that of a deep venous thrombus for which he was on warfarin. At presentation he had evidence of severe coagulopathy (INR 6.6, prothrombin time 92 seconds – poor treatment with intravenous vitamin K). His AST was elevated at 16,000, ALT 20,000, Bilirubin 91µmol/L (5.3 mg/dl), and creatinine 164 mmol/L. Over the next 24 hours his conscious level deteriorated to grade III encephalopathy requiring intubation and ventilation and transfer to a specialist unit. Findings on admission were those of young man with no stigmata of chronic liver disease, heart sounds were normal and chest clear on auscultation. There was no organomegaly (abnormal enlargement of the organs) but he was oligoanuric. Ultrasound of the abdomen was normal (liver echogenicity, spleen, vessels and pancreas with no free fluid). Blood investigations demonstrated ongoing liver dysfunction (INR 7.6, prothrombin time 110 seconds), Bilirubin 100µmol/L (5.8mg/dl), ALT 17,000, Albumin 30%, and creatinine 400mmol/L. He had a profound metabolic acidosis, (pH 7.05, PaCO2 30 mmHg, PaO2 105 mmHg, HC03 - 12), and anuric. In view of his clinical findings it was felt he fulfilled poor prognostic criteria and he was listed for urgent liver transplantation.

He underwent liver transplant at 36 hours post admission and received a full orthotopic graft. During reperfusion in theatre he had evidence of severe coagulopathy (INR 6.6, prothrombin time 21 secs). The PiCCO parameters at this time demonstrated ITBI 920, ELWI 12, SVV 10 %, CI 2.6 L/min/m2 and the gas exchange remained normal (pH 7.43, PaO2 5.5, PaCO2 10, HC03 22, FiO2 0.5, PEEP 6). LiMON ICG PDR was repeated and had fallen significantly to 8. Echocardiogram revealed normal left sided cardiac function, however the right heart was some what dilated and there was mild tricuspid regurgitation with elevated right sided pressures. Due to the complexity of the condition a PA catheter was inserted that showed a pulmonary pressure 50/33 (elevated), RV pressures 48/15, mixed venous saturation 70%. There was no evidence of pulmonary embolic disease. The deteriorating graft function was thought to be related to elevated right sided pressures and he was treated with nebulised epoprostenol (prostacyclin - vasodilator) and sildenafil (Viagra – increase pulmonary bed blood flow). He did not show any evidence of fluid overload in terms of fluid balance or clinical parameters and a neutral fluid status was maintained.

Over the next 48 hours his PA pressures fell and LiMON studies demonstrated a steady improvement (PA 35/17, ICG corr 12). Intra-abdominal pressure had stayed between 14-16 throughout this period and lactate and coagulation parameters had remained within the normal range.

Further monitoring was inserted including a PiCCO line, reverse jugular line and intra-cranial pressure bolt. He was commenced on haemofiltration. Intra-abdominal pressure at this time was 14. PiCCO monitoring revealed low intrathoracic blood volume index (ITBI) 600 (normal range 850 – 1000 ml/m2), normal extravascular lung water index (ELWI) 6 (normal range 3.0 – 7.0 ml/kg), high stroke volume variation (SVV) 20% (normal ≤10 %), normal Cardiac Index (CI) 4.8 (normal range 3.0 – 5.0 L/min/m2) and heart rate (HR) 130. Further fluid loading was undertaken and his SVV and CI fell to 12, with an increase in Stroke Volume Index (SVI) and CI (6.5l/min/m2). His lactate fell to 6 and the norepinephrine dose requirement decreased to 0.25µg/kg/min. His other investigations showed ongoing coagulopathy with INR 8 (prothrombin time 112 secs) and he remained anuric. In view of his clinical findings it was felt he fulfilled poor prognostic criteria and he was listed for urgent liver transplantation.

His clinical course thereafter was one of steady improvement. His PA catheter was removed at 72 hours post insertion and PiCCO monitoring some 2 days later. He no longer required pressor support by day 5 post liver transplant and his renal function returned at day 20. He was subsequently discharged to the ward and thence home 5 weeks post initial presentation. He remains well 2 years post liver transplant.

Clinical learning points:

- Volume depletion is common in patients with acute hepatic failure and PiCCO monitoring facilitates appropriate volume loading.
- CVP did not assist in any way in fluid guidance and ScvO2 was above normal despite the patient being fluid deplete. This is a common finding in patients with hyperdynamic states.
- Post liver transplant LiMON monitoring allowed dynamic tracking of graft function.
- This was especially pertinent in this case where the elevated CVP and right heart dysfunction was associated with significant graft dysfunction.
- It is important to note that at this time all other measures of graft function were reassuringly normal.
- Early intervention was undertaken and LiMON allowed tracking of the effects of these interventions. Using this technology earlier intervention could be considered before the standard measures of graft dysfunction were seen, and the patient made a steady improvement thereafter.
Case description
A 16 year old girl was brought to the ER after being injured in a car accident. She had sustained a severe head injury with intracerebral and subarachnoid hemorrhage and there was very extensive diffuse axonal injury. The patient also had a significant lung injury with bilateral lung contusion. A chest tube was inserted on the left side where there was a hemi-pneumothorax. Due to a hemoglobin concentration of 7 G/dl she received 3 units of packed cells and about 3 liters of crystalloids.

The patient underwent a laparotomy where a fractured spleen was removed and an Intracranial Pressure (ICP) monitor was inserted in the OR. Initial ICP was 20 mmHg at the time (normal range), the mean arterial blood pressure was 80 mmHg, and noradrenaline was initiated to maintain a Cerebral Perfusion Pressure of 65-80 mmHg.

Clinical course
The patient’s ICP increased over the next 2 days to a maximum of 35 mmHg. To assess her hemodynamics, a PICCO catheter was inserted and her Cardiac Index was high, at 4 L/Min., her Intrathoracic Blood Volume Index (ITBI) was normal at 800 ml/m², her Systemic Vascular Resistance (SVR) was high at 1500, and her Extravascular Lung Water Index (ELWI) was 15 ml/kg. The attending doctor decided to diurese her and attempt to keep her hypovolemic so that pulmonary and cerebral edema could be decreased. At the same time we were concerned about her splanchnic circulation so that a LiMON measurement (Liver Monitor, PULSION Medical Systems AG) was made, demonstrating a Plasma Disappearance Rate of 19%, well within normal values (18-23%).

The patient’s fluid intake was reduced and a diuretic was administered. Over the next day her blood pressure (BP) remained the same as did her urine output. ITBI decreased to 650 ml/m² and then to 500 ml/m². Her ICP decreased to 18-20 mmHg and her ELWI decreased to 12 ml/kg. Her PDR remained 18% and her lactate was normal. This regimen was maintained for the next two days.

On the 5th postoperative day, the patient’s PDR was measured and found to be 11%. At the time her ITBI was 550 ml/m², her ELWI was 10 ml/kg and her ICP was 18 mmHg. Lactate was still normal. Realizing that the patient’s splanchnic circulation was at risk, she was given a fluid bolus of 1000 ml of colloid to increase her perfusion. This had a minimal effect on her ICP, her BP increased, and her ITBI increased to 700 ml/m². ELWI remained at 9-10 ml/kg. Fluid administration was increased and her fluid balance over the next few days was maintained at a positive level. PDR returned to a value of 18%. The patient was stable for the next 2 days. Unfortunately, the patient developed a new septic episode 10 days after admission and died from multiple organ failure two weeks after admission.

Conclusion
This patient demonstrates the usefulness of monitoring splanchnic perfusion with the LiMON. It is not unusual for us to keep patients hypovolemic to try to decrease edema, whether pulmonary or cerebral. We are aware that “running patients dry” can lead to reduced perfusion of critical organ systems and cause unwanted oxygen debt. In this scenario, monitoring patients with regard to preload parameters, cardiac output and even fluid responsiveness may not be enough. Measuring lactate is one commonly used method to assess for reduced perfusion, but lactate may be a late sign of poor oxygen delivery. An indicator of adequacy of perfusion such as the plasma disappearance rate of ICG is helpful in the decision of the degree of hypovolemia that the patient can tolerate.
Editorial: Intra-abdominal pressure: It is now time to accept and promulgate!

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Clinical relevance of intra-abdominal pressure

Introduction
A compartment syndrome exists when the increased pressure in a closed anatomic space threatens the viability of surrounding tissue. When this occurs in the abdomen the impact on end-organ function within and outside the cavity can be devastating. It is only recently that abdominal compartment syndrome (ACS) has received heightened awareness. The development of intra-abdominal hypertension (IAH) and ACS are of tremendous importance in the care of critically ill, surgical and trauma patients. The impact of increased IAP on end organ function and especially the heart and the lungs can no longer be ignored!

Historical background
The effects of elevated IAP have been known since 1863, when Marey of Paris highlighted that “the effects that respiration produces on the thorax are the inverse of those present in the abdomen” (1). In 1890, Heinricius demonstrated that ACS was fatal to animals because of impairment of respiration, decreasing cardiac diastolic distension and hypotension. It wasn’t until 1911 that Emerson showed in dogs that elevated IAP increases systemic vascular resistance and can cause death from cardiac failure even before asphyxia develops. He concluded that “the distension of the abdomen with gas or fluid results in cardiac compromise due to an overloaded of the resistance in the splanchnic area” and that “removal of ascitic fluid results in relief of the labouring heart”.

Definitions
The World Society of Abdominal Compartment Syndrome (WSACS – http://www.wsacs.org/) was founded in 2004 to serve as a peer-reviewed forum and educational resource for all healthcare providers as well as industry who have an interest in IAH and ACS. The mission of the society is to foster education, promote research and thereby improve survival of patients with IAH and ACS. Recently the first consensus definitions report of the WSACS was published (2). Table 1 shows some of the consensus definitions.

Table 1: Consensus definitions (2)

<table>
<thead>
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<th>Definition</th>
<th>Description</th>
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<tr>
<td><strong>ACR</strong> = <strong>APF</strong> – <strong>IAP</strong></td>
<td><strong>ACR</strong> = abdominal perfusion pressure and is calculated as MAP – IAP</td>
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<td><strong>IAP</strong> is the steady-state pressure concealed within the abdominal cavity.</td>
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<tr>
<td><strong>APP</strong> = MAP – IAP</td>
<td>APP = abdominal perfusion pressure</td>
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<td><strong>IAP</strong> is graded as follows:</td>
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<tr>
<td>• Grade I: IAP 12-15 mmHg</td>
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<td>• Grade II: IAP 16-20 mmHg</td>
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<td>• Grade III: IAP 21-25 mmHg</td>
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<td>• Grade IV: IAP &gt; 25 mmHg</td>
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<td><strong>IAP</strong> is defined as a sustained IAP &gt; 20 mmHg (with or without an APP &lt; 60 mmHg) that is associated with new organ dysfunction / failure.</td>
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</table>

Table legend:
ACR = abdominal compartment syndrome; APP = abdominal perfusion pressure; FG = filtration gradient; GFP = glomerular filtration pressure; IAH = intra-abdominal hypertension; IAP = intra-abdominal pressure; MAP = mean arterial pressure

Recognition of ACS
Despite an escalation of the medical literature on the subject, there still appears to be an under-recognition of the syndrome. The results of several surveys on the physician’s knowledge of IAH and ACS have recently been published (3). The bottom line is that there is still a general lack of clinical awareness and many ICUs never measure the IAP. No consensus exists on optimal timing of measurement or decompression. In a recent editorial Ivatury stated that (4): “One potential exegesis of this widespread under-appreciation of these syndromes may be related to our rapidly evolving understanding of their pathophysiology. Our knowledge is no longer restricted to experimentally sound (isolated IAH) concepts, but is elevated to a true clinical phenomenon (IAH as a “second-hit” after ischemia-reperfusion).”

Diagnosis
Measurement of intra-abdominal pressure
Since the abdomen and its contents can be considered as relatively non-compressive and primarily fluid in character, behaving in accordance to Pascal’s law, the IAP measured at one point may be assumed to represent the IAP throughout the abdomen (5,6). In the strictest sense, normal IAP ranges from zero to 5 mmHg (7). Certain physiologic conditions, however, such as morbid obesity (8,9), ovarian tumours, cirrhosis or pregnancy, may be associated with chronic IAP elevations of 10-15 mmHg to which the patient has adapted with an absence of significant pathophysiology. In contrast, children commonly demonstrate low IAP values (10). The clinical importance of any IAP must be assessed in view of the baseline steady-state IAP for the individual patient. Different indirect methods for estimating IAP are used clinically because direct measurements are considered to be too invasive (5, 11). These techniques include rectal, uterine, gastric, inferior vena cavaal and urinary bladder pressure measurement. Only gastric and bladder pressures are used clinically.

Abdominal perfusion pressure (APP) measurement
Analogous to the widely accepted and clinically utilized concept of cerebral perfusion pressure, calculated as mean arterial pressure (MAP) minus intracranial pressure (ICP), abdominal perfusion pressure (APP), calculated as MAP minus IAP, has been proposed as a more accurate predictor of visceral perfusion and a potential endpoint for resuscitation (12-14).

**APP** = **MAP** – **IAP**

Pathophysiologic implications
IAH affects multiple organ systems in a graded fashion. In order to better understand the clinical presentation and management of disorders of IAH, one must understand the physiologic derangements within each organ system separately (15). Please refer to recently published reviews for a complete overview of the pathophysiologic implications of raised IAP (16, 17). In this editorial I will only discuss some key-messages relating to the most important organs, namely the heart and the lungs that will affect daily clinical practice.
Clinical relevance of intra-abdominal pressure

Cardiovascular function
Due to the cephalad movement of the diaphragm pleural pressure and intrathoracic pressure (ITP) will increase. When IAP rises above 10 mmHg cardiac output (CO) drops due to an increase in afterload (SVR) and a decrease in preload and left ventricular compliance (18-21). Mean arterial blood pressure may initially rise due to shunting of blood away from the abdominal cavity but thereafter normalise or decrease (14, 22).

- Accurate assessment and optimization of preload, contractility, and afterload are essential to restore end-organ perfusion and function.
- Traditional hemodynamic monitoring techniques and parameters, must, however, be re-evaluated in IAH/ACS since pressure-based estimates of intravascular volume as pulmonary artery occlusion pressure (PAOP) and central venous pressure (CVP) are erroneously increased.
- The clinician must be aware of the interactions between ITP, IAP, PEEP, and intracardiac filling pressures.
- Misinterpretation of the patient’s minute-to-minute cardiac status may result in the institution of inappropriate and potentially detrimental therapy.
- The Surviving Sepsis Campaign Guidelines targeting initial and ongoing resuscitation towards a CVP of 8 to 12 mmHg (23) and other studies targeting a MAP of 65 mmHg (24) should be interpreted with caution in the case of IAH/ACS to avoid unnecessary over- and under-resuscitation!
- Volumetric estimates of preload status such as SVV, PPV or SPV should be used to assess volume responsiveness (29).
- The cardiovascular effects are aggravated by hypovolemia and the application of PEEP (30-34), whereas hypervolemia has a temporary protective effect (35).

Pulmonary function
The interactions between the abdominal and the thoracic compartment pose a specific challenge to the ICU physician. Both compartments are linked via the diaphragm and on average a 50% (range 25-80%) transmission of IAP to the ITP has been noted in previous animal and human studies (21). Patients with primary ACS will often develop a secondary ARDS and will require a different ventilatory strategy and more specific treatment than a patient with primary ARDS (36, 37).

- IAH decreases total respiratory system compliance by a decrease in chest wall compliance, while lung compliance remains unchanged (38, 39).
- The ARDS consensus definitions should take into account PEEP and IAP values.
- The PAOP criterion in ARDS consensus definitions is futile in case of IAH and should be adapted (most patients with IAH and secondary ARDS will have a PAOP above 18mmHg).
- IAH increases lung edema, within this concept monitoring of extravascular lung water index (ELWI) seems warranted (40).
- The combination of capillary leak, positive fluid balance and raised IAP poses the patient at an exponential danger for lung edema.
- Body position affects IAP:
  - putting an obese patient in the upright position can cause ACS (41).
  - the abdomen should hang freely during prone positioning (42)
- the anti-Trendelenburg position may improve respiratory mechanics, however it can decrease splanchic perfusion (43).
- The use of curarisation should be balanced against the beneficial effect on abdominal muscle tone resulting in a decrease in IAP and improvement of APP, and the detrimental effect on lung mechanics resulting in atelectasis and sur-infection (44).

Conclusions
As first suggested in 1863 by Marey, ACS is a constellation of the physiologic sequelae of increased IAP, termed IAH. Recent observations suggest an increasing frequency of this complication in all types of patients. Even chronic elevations of IAP seem to affect the various organ systems in the body. The presence of IAH and ACS are significant causes of organ failure, increased resource utilization, decreased economic productivity, and increased mortality among a wide variety of patient populations (16, 45).

Despite its obvious clinical implications, attention is not paid to IAP, IAH and ACS. Only a few medical and surgical intensivists believe in the concept and actively attempt its prevention and treatment (4). It is time to pay attention, to accept, act and promulgate (17) as per the slogan of the 3rd World Congress on Abdominal Compartment Syndrome (WCACS2007) held in Antwerp, Belgium in 2007, March 22-24 (www.wcacs.org).

“The best way to predict the future is to invent it.”
Alan Kay; Conceiver of the laptop computer and windows operating system

“Look to the future and not to the past to find those things you want to make last: IAP is definitely here to stay!”
The author

| Table 2 |
|-----------------|-----------------|------------------|
| Cardiovascular effects related to IAP* | Cardiovascular effects related to IAP* |
| Diaphragm elevation | Pulmonary artery occlusion pressure |
| Difficult preload assessment | Central venous pressure |
| Transmural filling pressure | Right ventricular end-diastolic volume |
| Intra thoracic blood volume index | Right, global and left ventricular ejection fraction |
| Extra vascular lung water | Stroke volume variation |
| Pulse pressure variation | Systolic pressure variation |
| Inferior vena caval flow | Venous return |
| Venous return | Left ventricular compliance and contractility |
| Downward Starling curve shift to the right | Cardiac output |
| Systemic vascular resistance | Mean arterial pressure |
| Pulmonary artery pressure | Pulmonary vascular resistance |
| Heart rate | Lower extremity hydrostatic venous pressure |
| Venous stasis, edema, ulcers | Venous thrombosis |
| Venous embolism | Mixed venous oxygen saturation |
| Central venous oxygen saturation | |

* Cardiovascular effects are exacerbated in cases of hypovolemia, hemorrhage, ischemia, auto-PEEP or high PEEP ventilation # upon decompression
Table 3

<table>
<thead>
<tr>
<th>Pulmonary effects related to IAP</th>
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<tbody>
<tr>
<td>Diaphragm elevation ↑</td>
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<tr>
<td>Intrathoracic pressure ↑</td>
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<tr>
<td>Pleural pressure ↑</td>
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<tr>
<td>Functional residual capacity (FRC) ↓</td>
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<tr>
<td>All lung volumes (TLC, TV,...)↓</td>
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<tr>
<td>(~restrictive disease)</td>
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<tr>
<td>Extrinsic compression lung parenchym*↑</td>
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<tr>
<td>Auto-PEEP ↑</td>
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<tr>
<td>Compression atelectasis ↑</td>
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<tr>
<td>Peak airway pressure ↑</td>
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<tr>
<td>(volume controlled MV)</td>
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<tr>
<td>Mean airway pressure ↑</td>
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<tr>
<td>Plateau airway pressure ↑</td>
</tr>
<tr>
<td>Pulmonary vascular resistance ↑</td>
</tr>
<tr>
<td>Alveolar barotrauma = ↑</td>
</tr>
<tr>
<td>Alveolar volutrauma = ↑</td>
</tr>
<tr>
<td>Dynamic compliance ↓</td>
</tr>
<tr>
<td>Static respiratory system compliance ↓</td>
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<tr>
<td>Static chest wall compliance ↓</td>
</tr>
<tr>
<td>Static lung compliance =</td>
</tr>
<tr>
<td>Upper inflection point on PV curve ↓</td>
</tr>
<tr>
<td>Lower inflection point on PV curve ↑</td>
</tr>
<tr>
<td>Hypercarbia - pCO2 retention ↑</td>
</tr>
<tr>
<td>Pao2 ↓ and PaO2/FiO2 ↓</td>
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<tr>
<td>Alveolar oxygen tension ↓</td>
</tr>
<tr>
<td>Oxygen transport ↓</td>
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<tr>
<td>Dead-space ventilation ↑</td>
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<tr>
<td>Intrapulmonary shunt ↑</td>
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<tr>
<td>Ventilation perfusion mismatch ↑</td>
</tr>
<tr>
<td>Ventilation diffusion mismatch ↑</td>
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<tr>
<td>Oxygen consumption ↑</td>
</tr>
<tr>
<td>Metabolic cost and work of breathing ↑</td>
</tr>
<tr>
<td>Alveolar edema ↑</td>
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<tr>
<td>Extra vascular lung water (EVLW) =</td>
</tr>
<tr>
<td>Prolonged ventilation</td>
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<tr>
<td>Difficult weaning</td>
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<tr>
<td>Activated lung neutrophils ↑</td>
</tr>
<tr>
<td>Pulmonary inflammatory infiltration ↑</td>
</tr>
<tr>
<td>Pulmonary infection rate ↑</td>
</tr>
</tbody>
</table>

* Parenchymal compression is exacerbated in case of hemorrhagic shock or hypotension

Clinical relevance of intra-abdominal pressure

References
AVOID COMPLICATIONS

Protect your patient … against Abdominal Compartment Syndrome

CiMON
Clinical Relevance of Intra-Abdominal Pressure

Case Study: Primary abdominal compartment syndrome: A trauma case study

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Report of the case:
39-year-old male patient was admitted to our ER after a motorcycle accident. He had sustained severe blunt abdominal trauma and had required CPR and intubation on site of the accident. On arrival he was hemodynamically unstable and required mass-transfusion. Sonography showed free intraabdominal fluid. He underwent immediate emergency laparotomy that revealed a grade IV rupture of the right liver lobe and an extended retroperitoneal hematoma. After abdominal packing and primary closure a CT-scan showed no further injuries to head, spine, pelvis or extremities. Continuous bleeding required an immediate second look laparotomy with resection of liver segment IV, resection of right suprarenal gland, suture of right kidney, right-sided colectomy, discontinuous ileostomy and packing.

When admitted to ICU the patient needed further fluid resuscitation and noradrenalin support with up to 24 µg/min. A PICCO® catheter (Pulsion Munich) was inserted and revealed the following hemodynamic status: Cardiac output (CO) 8.7 L/min; intrathoracic blood volume index (ITBI) 890 mL/kg; extravascular lung water index (ELWI) 8.5 mL/kg; stroke volume (SV) 90 mL and systemic vascular resistance (SVR) 708 dyne*sec/cm5. A chest tube was inserted on the right side for hemothorax. 8 hours after admission urinary output ceased completely though CVP remained unchanged in the range of 10-12 mmHg, IAP was 23 mmHg and the patient had a persistent need for fluid resuscitation. Continuous veno-venous hemofiltration (CVVH) was begun. Noradrenalin support was increased from 23 to 33 µg/min and IAP increased from 23 to 33 mmHg.

The patient was diagnosed to have an abdominal compartment syndrome (ACS). He underwent decompressive laparotomy 14.5 hours after the second intervention and packing. When the abdomen was opened liver and intestine appeared to be insufficiently perfused. The packing was partly removed and shortly afterwards perfusion of liver and intestine improved. Bleeding had stopped and the abdomen was temporarily closed using a 30 x 30 cm vicryl® mesh (Ethicon, Norderstedt). In the operating room the patient’s condition already improved. The postoperative PICCO line the patient received noradrenalin support and massive fluid resuscitation in order to restore ITBI and SVR. Massive fluid resuscitation, damage control surgery and packing are well known risk factors for the development of an ACS due to capillary leak and third spacing of fluid.

According to these results, dosage of noradrenalin was increased and further volume was administered. The patient’s condition stabilized thereafter and volume as well as catecholamine support could be withdrawn. According to the mean arterial pressure, noradrenalin support was reduced to 8 µg/min during the following 24 hours. Urinary output did not pick up though stimulated with furosemide and the patient remained on CVVH.

On day 4 programmed laparotomy showed a stable condition. The abdomen was completely de-packed and again closed with a 30 x 30 vicryl® mesh (Ethicon, Norderstedt) mesh. GCS remained below 10 though sedation was stopped. Follow-up CCT and MRI showed no cerebral or spinal injuries. Prolonged ventilation required tracheotomy on day 13. During the course on the ICU the patient was successfully weaned and regained consciousness. Oral feeding was well tolerated. Physiotherapy was started as soon as the hemodynamic condition stabilized. Remaining problems were a constant rise in serum bilirubin and three septic episodes that required intermittent noradrenalin support and ventilation. Bilirubin rose over 20 (g/dL) but abdominal ultrasound and CT showed no intra- or extraperitoneal obstruction of the bide. This was followed by ERCP and the placement of a nasogastric tube. Serum bilirubin fell but remained elevated to 15 (g/dL). On day 29 the nasogastric tube had to be removed due to bleeding from the papilla vateri. Bilirubin remained at 17 (g/dL). On day 33 the patient was found to be colonized with MRSA. On day 43 after admission the patient deceased in the ICU due to an acute bleeding from his trachea and a blood loss of 4-6 l within 10 min. CPR was unsuccessful and stopped after 40 min. A post-mortem was performed. It was assumed that the bleeding originaed from an erosion of a bronchial artery. The reason for the erosion could not be determined.

Discussion:
In this case report, a patient developed an ACS in the course of a severe motorcycle accident with blunt abdominal trauma, liver rupture, retroperitoneal hematoma, resuscitation, damage control surgery and abdominal packing. The liver rupture and the retroperitoneal hematoma required abdominal packing and primary closure to stop the otherwise uncontrollable bleeding. According to the hemodynamic measurements via a PICCO line the patient received noradrenalin support and massive fluid resuscitation in order to restore ITBI and peripheral resistance. Massive fluid resuscitation, damage control surgery and packing are well known risk factors for the development of an ACS due to capillary leak and third spacing of fluid.

Although an increase of IAP was anticipated, a compromized perfusion in the abdominal cavity had to be accepted in order to gain time, control the bleeding and stabilize coagulation. The development of ACS was recognized early due to the implemented measurements and allowed for a programmed second look laparotomy and early de-packing.
Case Study: Secondary abdominal compartment syndrome

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Clinical Relevance of Intra-Abdominal Pressure

Secondary abdominal compartment syndrome

Case history
A 42 year old male with no relevant medical history was admitted to the surgical intensive care unit after resection of a malignant lesion by extrapleural pneumonectomy, pericardectomy and partial thoracic wall resection. During the operation, the patient developed cardiac arrhythmia with hypotension. At the end of the procedure anisocoria was noted, but both pupils were reactive to light. After admission to the ICU the patient remained hemodynamically unstable and a surgical revision was carried out on the first postoperative day (POD 1). During the revision diffuse oozing but no significant bleeding was encountered. Postoperatively, the patient developed a massive capillary leak syndrome over the first 5 days with an elevated stroke volume variation (SVV), fluid responsive hypotension, need for inotropics (dobutamine) and vasopressors (norepinephrine at a dose of 400ng/kg/min) and worsening renal function. A CT scan of the thorax revealed extensive infiltrates, for which endoscopic decompression was performed without further delay. It was followed immediately by a dramatic improvement in organ function (decreased O2 need, resuming diuresis, lower vasopressor need).

Immediately before decompressive laparotomy, the patient was treated with nitric oxide for pulmonary hypertension, he had a high CVP and high PEEP monitoring of this patient. Immediately before decompressive laparotomy, the patient was treated with nitric oxide for pulmonary hypertension, he had a high CVP and high PEEP was needed to maintain adequate oxygenation. The phenomenon that 20-80% of IAP is transmitted to the thorax has been described in animal studies before [1]. This phenomenon can lead to inaccurate assessment of the hemodynamic status of the patient when pressure monitoring (CVP, PCWP or PAOP) is used. Therefore, the use of volumetric monitoring methods (e.g.PiCCO) may be recommended in patients with intra-abdominal hypertension.

A CT scan of the brain showed a large ischemic lesion with secondary bleeding and cerebellar herniation and the patient was pronounced brain dead the same day.

Discussion and conclusion:
In this patient the abdominal compartment syndrome (ACS) was caused by massive fluid resuscitation leading to decreased compliance of the abdominal and thoracic wall. IAP was further increased by dilation of the colon, but no other intra-abdominal lesions were present. The development of secondary ACS was an indication for immediate decompressive laparotomy, but an attempt at lowering IAP by endoscopic decompression of the colon was made first, because of fear of impaired spontaneous breathing when the abdominal wall was compromised in a patient with a pneumonectomy and significant thoracic wall resection. This attempt was unsuccessful and decompressive laparotomy was performed without further delay. It was followed immediately by a dramatic improvement in organ function (decreased O2 need, resuming diuresis, lower vasopressor need).

A comment can be made regarding the hemodynamic monitoring of this patient. Immediately before decompressive laparotomy, the patient was treated with nitric oxide for pulmonary hypertension, he had a high CVP and high PEEP was needed to maintain adequate oxygenation. The phenomenon that 20-80% of IAP is transmitted to the thorax has been described in animal studies before [1]. This phenomenon can lead to inaccurate assessment of the hemodynamic status of the patient when pressure monitoring (CVP, PCWP or PAOP) is used. Therefore, the use of volumetric monitoring methods (e.g.PiCCO) may be recommended in patients with intra-abdominal hypertension.

Several recent surveys have demonstrated that awareness of primary ACS is generally good among surgeons and intensivists, but secondary ACS and the need for IAP monitoring in patients with non-abdominal risk factors (e.g. massive fluid administration, acidosis, hypothermia...) are less well recognised [2]. We believe that, in spite of the negative outcome, the clinical course of this patient illustrates the need for IAP monitoring in risk patients (as defined by the World Society for the Abdominal Compartment Syndrome) even when no apparent intra-abdominal pathology is present [3]. Also, decompressive laparotomy should be considered in any patient with an elevated IAP>20mmHg with new or progressing organ dysfunction regardless of the cause of the intra-abdominal hypertension [4]. However, the morbidity and mortality of secondary abdominal compartment syndrome remain high even when treated correctly [5].

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PiCCO_{2}

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