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Case Report

Why should I bother about the ebb and flow phases of shock? An illustrative case report

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Abstract ***Introduction and background*** Preload and fluid responsiveness are two different things. In certain situations like patients with increased intrathoracic pressures, traditional barometric filling pressures as the central venous pressure are erroneously increased. In those circumstances volumetric preload indices better reflect the true preload conditions of the patient. Fluid management in these patients can be very tricky because adequate early initial resuscitation is mandatory however in order to prevent organ edema and secondary abdominal hypertension one must avoid ongoing futile fluid loading. We will illustrate opposite changes between barometric and volumetric preload indices in a patient with increased intrathoracic pressure. **Patients and methods** The case of a 26 year old man admitted to the ICU after general seizures described. This case was presented at the 32nd annual international symposium on intensive care and emergency medicine (ISICEM) in Brussels on March 20th and at the 2nd International Fluid Academy Day (IFAD) in Antwerp on November 17th. ***Results*** In this patient, that developed shock within 18 hours of ICU admission the dynamic evolution is presented. Despite initial normal (and thus adequate) filling pressures, further fluid resuscitation was needed to overcome the ebb phase (this was guided by functional hemodynamic parameters and volumetric preload indices). Diuretics were initiated after 24 hours to help the patient to transgress to the flow phase because of respiratory failure due to capillary leak as evidenced by increased extravascular lung water. ***Discussion*** This case nicely demonstrates the biphasic clinical course from ebb to flow during shock as well as the inability of traditional filling pressures to guide us through these different phases. It also provides answers to the four basic but crucial questions that need to be solved in order not to do any harm to the patient: (1) when do I start giving fluids, (2) when do I stop giving fluids, (3) when do I start to empty my patient, and finally (4) when do I stop emptying?

Key words barometric • critical care • de-resuscitation • ebb and flow phase • fluid management • fluids • preload • resuscitation • volumetric

Introduction

Preload and fluid responsiveness are two different things [24—26]. In certain situations like patients with increased intrathoracic pressure (ITP) related to increased intra-abdominal pressure (IAP) or the use of high positive endexpiratory pressures (PEEP) during lung protective ventilation, traditional barometric filling pressures like the central venous pressure (CVP) or pulmonary artery occlusion pressure (PAOP) are erroneously increased [2, 10]. In those circumstances volumetric preload indices like global enddiastolic volume index (GEDVI), right ventricular enddiastolic volume index (RVEDVI) or left ventricle enddiastolic area index (LVEDAI) better reflect the true preload conditions of the patient. Since increased ITP has an impact on the global ejection fraction (GEF), correction of GEDVI in relation to the GEF may further improve the predictive value of this preload parameter [14]. Fluid management in these patients can be very tricky because adequate early initial resuscitation is mandatory however in order to prevent secondary intra-abdominal hypertension (IAH) one must avoid ongoing futile (crystalloid) fluid loading [3, 5, 16, 18, 21]. We will illustrate opposite changes between CVP and GEDVI in a patient with increased ITP related to acute respiratory failure with pulmonary hypertension and a transiently opened foramen ovale [13].

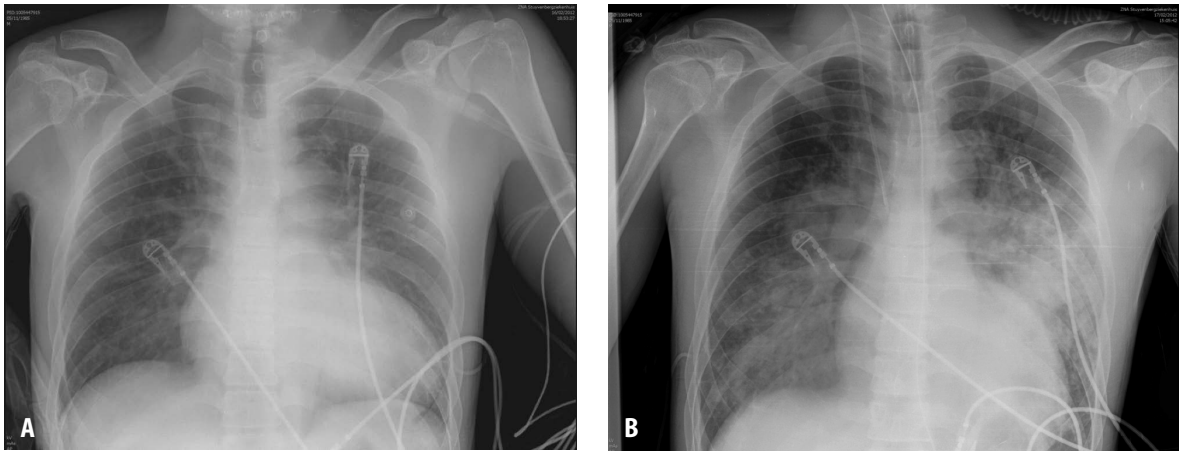

Fig. 1. Panel A. Chest X-ray obtained at admission. Panel B. Chest X-ray after 18 hours, obtained just after endotracheal intubation showing cardiomegaly with vascular crowding and bilateral interstitial infiltrates.

What are the risks of fluid overload?

When considering fluid administration it is important to know when to start giving fluids (what are the benefits of fluid administration), when to stop giving fluids (what are the risks of ongoing fluid administration), when to start removing fluids (what are the benefits of fluid removal), and when to stop fluid removal (what are the risks of removing too much fluid). The literature shows that a negative fluid balance increases survival in patients with septic shock [1]. Patients managed with a conservative fluid strategy also seem to have improved lung function, shorter duration of mechanical ventilation and intensive care stay without increasing non-pulmonary organ failure [30]. However, any measurement in the ICU will only be of value as long as it is accurate and reproducible, and no measurement has ever improved survival, only a good protocol can do this. Vice versa a poor treatment algorithm can result in potential harm to the patient [15]. Patients who are in the ebb or flow phase of shock have different clinical presentations and therefore different monitoring needs (targets) and different treatment goals [18, 21].

Methods

The case of a 26 year old man admitted to the ICU after general seizures is presented. This case was presented at the 32nd annual international symposium on intensive care and emergency medicine (ISICEM) in Brussels on March 22nd and at the 2nd International Fluid Academy Day (IFAD) in Antwerp on November 17th, both meetings were held in 2012.

A 5-item questionnaire was shown electronically to the participants. Each multiple choice question was shown during the case presentation lecture and participants were allowed to provide their feedback via a voting system (DIF Media). This case report will present the clinical case scenario as well as the results of the voting during both aforementioned meetings.

Case Study

Initial presentation

A 26 year old male is admitted to the intensive care unit with general seizures, syncope, non palpable blood pressure, and a suspicion of ventricular tachycardia whilst in the Emergency Room. The emergency room physician therefore (successfully) applied a DC shock to convert him to regular sinus rhythm. Afterwards the patient was alert and cooperative and he was transferred to the ICU for mere overnight "baby-sitting". From his previous history we know that he has been deprived of oxygen at birth, and consequently suffered a cerebrovascular accident (CVA) with left hemiparesis and seizures (managed with triple antiepileptic therapy, carbamazepine, topiramate and lamotrigine). Because of his cognitive deficit, he normally attends a special day care institution. For the last 9 years he had also been diagnosed with idiopathic cardiomyopathy with a left ventricular ejection fraction (LVEF) of 52% (treated with an angiotensin converting enzyme inhibitor) and a mild mitral regurgitation.

Overnight in the ICU, he was initially hemodynamically stable with no further seizures. However his need for supplemental oxygen increased from 2 litres via nasal cannula to 15 litres administered with a non-rebreathing mask. The patient was in respiratory distress with a respiratory rate of 34 breaths per minute. After failing a trial of non-invasive ventilation, he was intubated and mechanically ventilated within 24 hours of ICU admission, illustrating the dramatic chain of events. Respiratory rate was set at 24 breaths per minute and inspiratory pressures towards a tidal volume of 6 ml/kg predicted body weight (PBW). Figure 1 shows the chest X-ray on admission and just after intubation. He then became hemodynamically unstable. Therefore, a transthoracic cardiac ultrasound (US) was performed (Fig. 2) and the results are listed in Table 1 together with the ventilator settings and blood gas results.

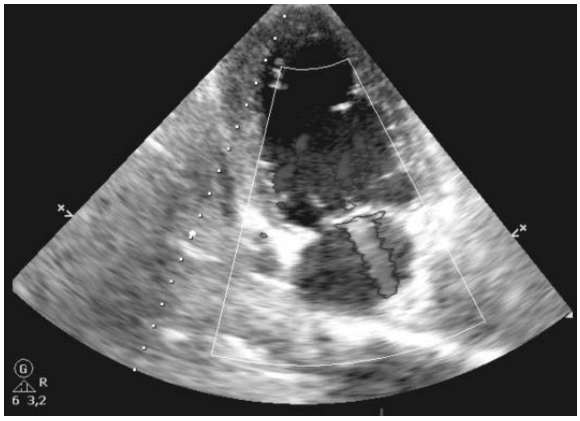

Fig. 2. Parasternal long axis image obtained during transthoracic cardiac ultrasound showing dilated left ventricle and 3 on 4 mitral regurgitation.

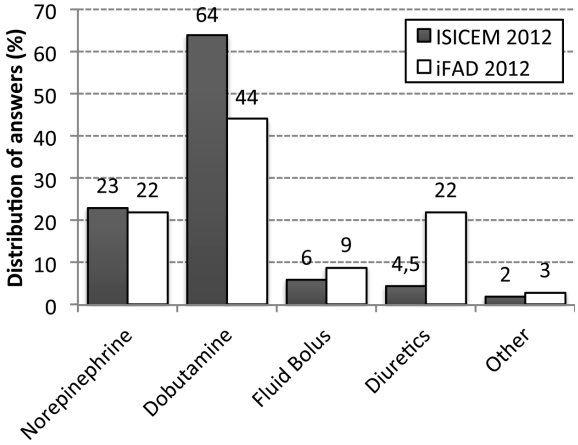

Fig. 3. Multiple choice question 1 (MCQ1): "Taking into account the results obtained with the transthoracic cardiac ultrasound, what is your treatment of choice at this stage?" Distribution of answers (in percentage) on MCQ1, grey squares denote the voting results of the ISICEM meeting while the white squares show the results of the iFAD meeting.

Multiple choice question 1

At this stage the participants of the ISICEM and IFAD meetings were asked the first multiple choice question (MCQ1): "Taking into account the results obtained with the transthoracic cardiac ultrasound, what is your treatment of choice at this stage?" Possible answers were: (1) norepinephrine; (2) dobutamine; (3) fluid bolus; (4) diuretics or (5) other. Fig. 3 shows the results of both votings. Based on the cardiac US findings physicians at ISICEM and iFAD seemed reluctant to fill the patient (only 6 to 9% stated to give a fluid bolus) and most of them were in favour of administrating dobutamine (44 to 64%).

Further course

The FiO_2 was increased to 100% and the PEEP was set according to the low flow pressure-volume (PV) loop (as can be automatically constructed with the Draeger Evita XL ventilator). Fig. 4 shows the PV loop with detection of a lower inflection point at 16 cmH_2O. During the PV loop that also acted as a recruitment maneuver his systolic blood pressure decreased to 40 mmHg, so norepinephrine was started and swiftly increased to 0.4 ug/kg/min. Dobutamine was also started at 4 ug/kg/min. Saturation remained poor at 88% and he was switched to high frequency percussive ventilation (HFPV) with the VDR4 ventilator (Percussionaire Corporation, Sandpoint, Idaho, U.S.A). A transpulmonary thermodilution PiCCO catheter (Pulsion Medical Systems, Munich, Germany) was inserted in the femoral artery at this point. The evolution of the hemodynamic parameters obtained after insertion of the PiCCO catheter together with the respiratory variables are listed in Tab. 2 and 3. The initial hemodynamic picture showed a normal cardiac index (CI) of 3.5 L/min.m^2 (normal range 3—5), a relatively low intravascular filling status with a GEDVI of 757 ml/m^2 (normal range 680—800), a very low global ejection fraction GEF of 13% (normal range 25—35) but a very severe capillary leak with high extravascular lung water index (EVLWI) of 38 ml/kg predicted body weight (normal range 3—7). The high EVLWI was

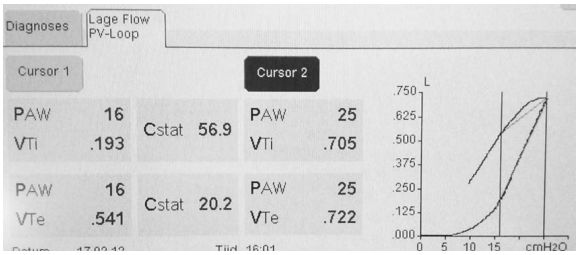

Fig. 4. Low flow pressure volume (PV) loop showing a lower inflection point at 16 cmH_2O and thus a best PEEP at 18 cmH_2O.

Table 1. Hemodyanmic profile obtained with transthoracic cardiac ultrasound, together with respiratory variables.

Parameter	Value
Mean arterial pressure, MAP (mmHg)	59
Central venous pressure, CVP (mmHg)	16
Cardiac index, CI (L/min.m^2)	3.5
Left ventricular enddiastolic pressure, LVEDP (mmHg)	25
Left ventricle ejection fraction, LVEF (%)	30
Left ventricle enddiastolic area index, LVEDAI (cm^2/m^2)	16.2
PaO_2 / FiO_2 ratio	74
Inspiratory airway pressure, IPAP (cmH_2O)	30
Positive endexpiratory pressure, PEEP (cmH_2O)	10
FiO_2	100
Lactate (mmol/L)	2.8

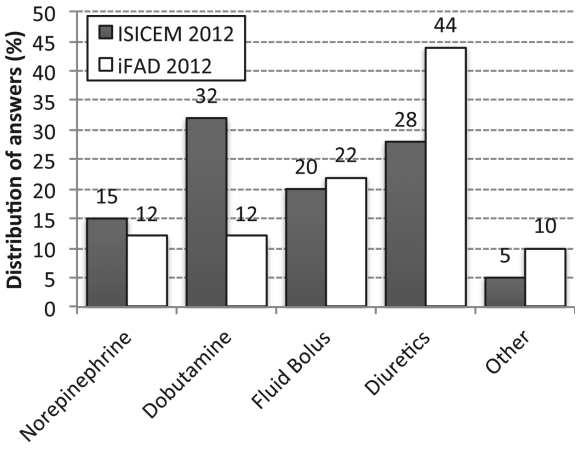

Fig. 5. Multiple choice question 2 (MCQ2): "Taking into account the results obtained with the transpulmonary thermodilution, what is your treatment of choice at this stage?". Distribution of answers (in percentage) on MCQ2, grey squares denote the voting results of the ISICEM meeting while the white squares show the results of the iFAD meeting.

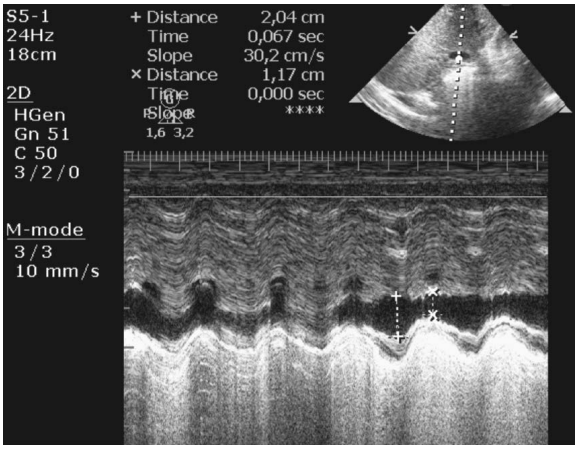

Fig. 6. Inferior vena cava collapsibility index (IVCCI) was calculated at 50%.

suggestive of hyperpermeability edema in view of the high pulmonary vascular permeability index (PVPI) of 7.4 (normal range 1—2.5) [27].

At the same time however the patient seemed to be fluid responsive with a high pulse pressure variation (PPV) of 19% (normal range <10). Heart rate was regular at 119 beats per minute with a MAP of 65 mmHg. The CVP was still 16 mmHg. His response to a passive leg raising (PLR) maneuver was positive (15% increase in CI and MAP) confirming that he was volume responsive despite the fact that he had such bad pulmonary edema (EVLWI 38) with a critical oxygenation status (P/F ratio of 57, at IPAP of 34 cmH_2O and PEEP of 15 cmH_2O).

Multiple choice question 2

At this stage the participants of the ISICEM and IFAD meetings were asked the second multiple choice question (MCQ2): "Taking into account the results obtained with the transpulmonary thermodilution, what is your treatment of choice at this stage?" Possible answers were: (1) norepinephrine; (2) dobutamine; (3) fluid bolus; (4) diuretics or (5) other. Figure 5 shows the results of both votings. Again physicians were reluctant to fill the patient initially (with only 20 to 22% indicating to give a fluid bolus). This patient had a relatively normal preload according to the volumetric preload indicator as was obtained by PiCCO (GEDVI 757 ml/m^2) but a high preload according to the barometric preload indicator (CVP 16 mmHg). Measurement of bladder pressure showed a slightly increased IAP of 12 mmHg [11]. The Surviving Sepsis Campaign Guidelines (SSCG) originally recommended that patients should be resuscitated towards a CVP range of 8—12 mmHg [7]. The latest revision of the SSCG still advocates initial fluid management based on CVP measurements [8]. However, using pressures to measure preload has been found to be inaccurate time and time again, particularly in patients ventilated with intermittent positive pressure ventilation (IPPV), (auto) PEEP, post cardiac surgery, obesity and those with intra-abdominal hypertension or abdominal compartment syndrome [2, 17]. Using a CVP threshold therefore may lead to over- but also under-resuscitation. Although it is re-assuring and noteworthy that the latest SSCG version does mention the effects of increased ITP and IAP on CVP: "In mechanically ventilated patients or those with known preexisting decreased ventricular compliance, a higher target CVP of 12 to 15 mmHg should be achieved to account for the impediment in filling. Similar consideration may be warranted in circumstances of increased abdominal pressure. Elevated CVP may also be seen with preexisting clinically significant pulmonary artery hypertension, making use of this variable untenable for judging intravascular volume status". Within this respect the compliance of the thorax and the abdomen are key elements in order to explain the index of transmission of a given pressure from one compartment to another: "The use of lung-protective strategies for patients with ARDS… has been widely accepted, but the precise choice of tidal volume… may require adjustment for such factors as the plateau pressure achieved, the level of positive end-expiratory pressure chosen, the compliance of the thoracoabdominal compartment…" [8]. This lead recently to the recognition of the polycompartment syndrome [19, 20].

Further course

In the case study the patient was given small volume resuscitation with hyperhaes (Fresenius Kabi) at a dose of 4 ml/kg given as a bolus over 10—15 minutes combined with 1000 ml of balanced colloids (Volulyte, 6% hydroxyethyl starch 130/0.4), following the results obtained with the transpulmonary thermodilution. He remained on a dobutamine infusion (9 ug/kg/min) and norepinephrine (0.4 ug/kg/min). The following day (day 2) his CI increased to 5.7 L/min.m^2, GEDVI increased to 900 ml/m^2 and EVLWI had decreased to 14 ml/kg PBW (Tab. 2).

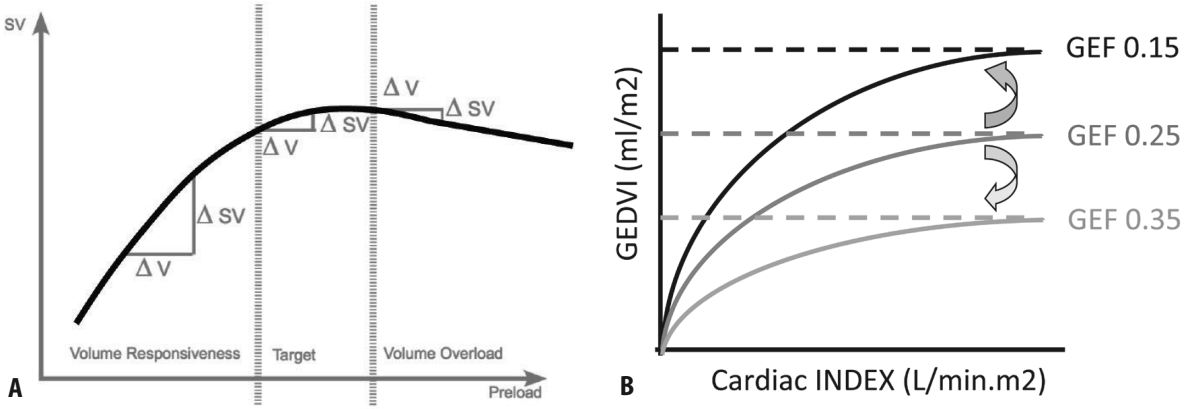

Fig. 7. Panel A. Relation between preload and stroke volume in different fluid loading conditions. Panel B. Ventricular function curves by global ejection fraction (GEF). The patient's GEDVI must be interpreted in conjunction with the patient's GEF (GEF – global ejection fraction, GEDVI – global end-diastolic volume index).

Table 2. Evolution of hemodynamic parameters obtained with transpulmonary thermodilution (PiCCO). Abbreviations and units: CI: cardiac index (L/min.m^2); CVP: central venous pressure (mmHg); EVLWI: extravascular lung water index (ml/kg PBW); GEDVI: global enddiastolic volume index (ml/m^2); GEF: global ejection fraction (%); HR: heart rate (bpm); MAP: mean arterial pressure (mmHg); PPV: pulse pressure variation (%); PVPI: pulmonary vascular permeability index.

Day	Time	CI	GEDVI	GEF	EVLWI	PVPI	PPV	HR	MAP	CVP
1	17:00	3,2	746	13	38	6,9	18	117	57	14
1	19:00	4,6	839	20	26	4,2	6	108	97	8
2	04:00	5,5	921	26	13	1,9	5	91	88	6
2	12:00	5	945	22	17	2,4	4	94	75	5
2	20:00	5,4	1025	23	19	2,5	6	93	79	6
3	04:00	4,8	1042	20	15	2,0	24	87	78	13
3	16:00	4,6	967	23	15	2,1	3	80	98	8
4	10:00	7	1073	24	14	1,8	5	107	103	10
4	18:00	5,9	977	26	12	1,7	4	101	85	9
5	10:00	4,6	1182	19	16	1,8	3	89	90	10
5	20:00	4,1	1060	17	13	1,7	4	80	86	14
6	04:00	3,1	893	16	14	2,1	5	79	76	9
6	11:00	3,3	972	17	14	2,0	4	80	95	6
6	17:00	3,2	900	16	12	1,8	3	84	109	5
7	06:00	3	882	20	11	1,7	10	65	72	10
7	12:00	3,8	908	21	10	1,5	17	144	100	6
7	18:00	5	829	25	12	2,0	6	88	69	4
8	05:00	4,9	1116	22	9	1,1	6	82	96	4
8	10:00	5,5	972	23	11	1,5	6	84	80	9
8	20:00	4,2	934	23	10	1,5	6	70	80	10
9	05:00	4,7	931	23	8	1,2	10	87	73	7

Despite the filling, his CVP decreased from 16 to 6 mmHg, illustrating the opposite changes between barometric and volumetric preload indices due to increased intrathoracic pressure.

This is an example of a therapeutic dilemma or conflict [22]. A therapeutic conflict is a situation where each of the possible therapeutic decisions carries some potential harm [29]. In high-risk patients, the decision about fluid administration should be

done within the context of a therapeutic conflict. Therapeutic conflicts are the biggest challenge for protocolized cardiovascular management in anesthetized and critically ill patients. A therapeutic conflict is where our decisions can make the most difference. Although the patient had evidence of severe pulmonary edema (EVLWI 38 ml/kg PBW) the decision was made to give fluids because the PPV was high and the PLR test was positive. Also, the GEDVI was relatively low in relation to the GEF, despite the increased CVP and increased left ventricular end diastolic area (from the ultrasound) [9, 14]. Cardiac US further showed that his inferior vena cava collapsibility index (IVCCI) was almost 50% [6] (Fig. 6).

What was really important to know for this patient was where he was on his Frank Starling curve (Fig. 7, panel A). Evidence shows that when the global end--diastolic volume and the right ventricular end-diastolic volume are corrected for the EF they correlate more closely especially when compared to the change in CVP or PAOP (Fig. 7, panel B) [14]. Observation of the transpulmonary thermodilution curve also allowed us to get further diagnostic clues (Fig. 8).

Multiple choice question 3

At this stage the participants of the ISICEM and IFAD meetings were asked the third multiple choice question (MCQ3): "What is the premature hump that appeared on the transpulmonary thermodilution curve?". Possible answers were: (1) Nothing to worry about, it is just an example of the crosstalk phenomenon; (2) It is related to thermal bolus mixing; (3) It may be an indicator of a right-to-left shunt due to pulmonary hypertension; (4) It is related to a wrong or false measurement technique; or (5) I don't know. Fig. 9 shows the results of both votings. The premature hump is evidence for a right to left shunt where an opening (foramen ovale) appears between the right and left atria. About half of the participants (41 to 53%) indicated the correct answer. Because the patient was extremely hypovolemic, the combination of positive pressure ventilation with high PEEP led to increased pulmonary vascular resistances, pulmonary hypertension and a propagation of West zone 1 conditions to zones 2 and 3. This phenomenon has been documented before [23].

Further course

By late afternoon of day 2, the patient had had a drop in urine output with production of only 350 mls over the last 12 hours despite a positive cumulative fluid balance of 4 litres. He was still on dobutamine 5 ug/kg/min, and norepinephrine 0.2 ug/kg/min. Other parameters are listed in Tab. 2 and 3, and can be summarized as follows: CI 5.4 L/min.m^2, MAP 79 mmHg, CVP 8 mmHg, PPV 6%, GEF 23%, GE-

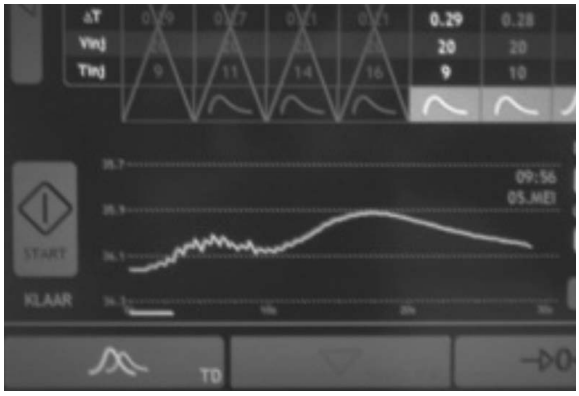

Fig. 8. Screen shot (obtained from a PiCCO$_2$ monitor) from the initial transpulmonary thermodilution curve, showing a premature hump

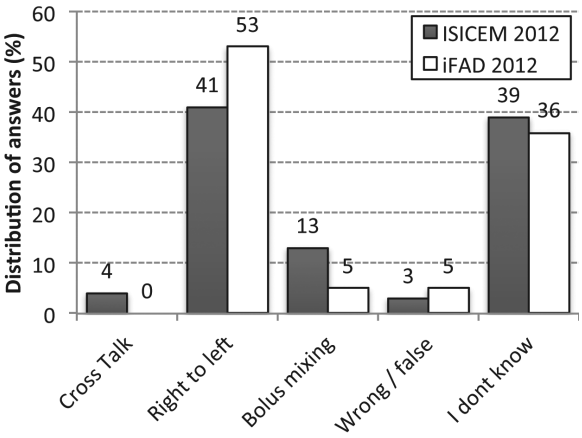

Fig. 9. Multiple choice question 3 (MCQ3): "What is the premature hump that appeared on the transpulmonary thermodilution curve?" Distribution of answers (in percentage) on MCQ3, grey squares denote the voting results of the ISICEM meeting while the white squares show the results of the iFAD meeting.

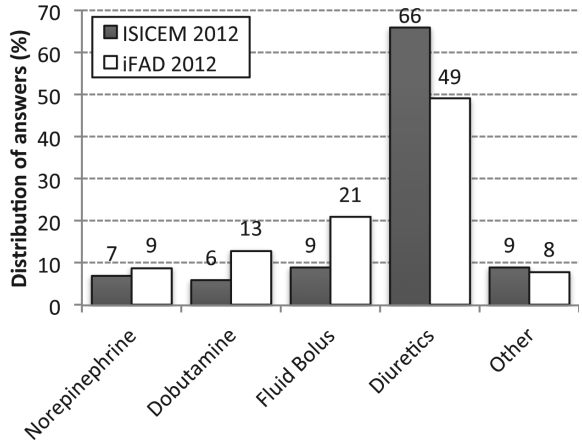

Fig. 10. Multiple choice question 4 (MCQ4): "Taking into account the new results obtained with the transpulmonary thermodilution and the drop in urine output, what is your treatment of choice at this stage?" Distribution of answers (in percentage) on MCQ4, grey squares denote the voting results of the ISICEM meeting while the white squares show the results of the iFAD meeting.

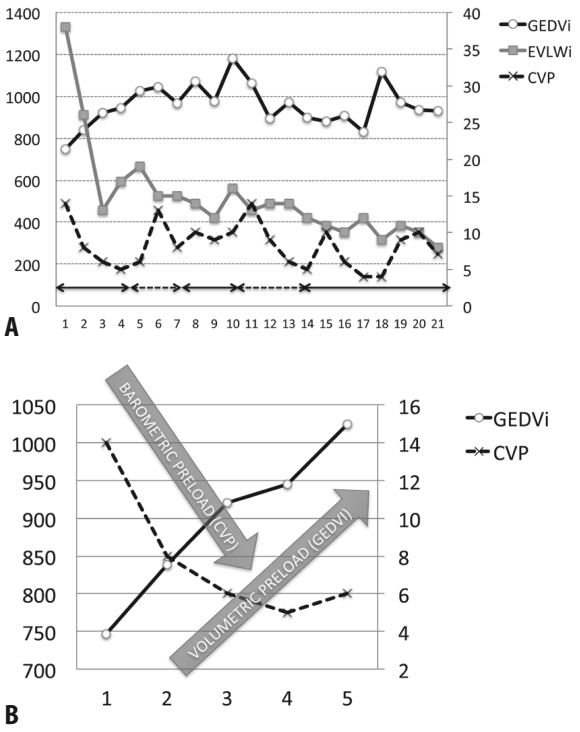

Fig. 11. Panel A. Evolution of barometric and volumetric indices during the first week of ICU stay. CVP: central venous pressure (mmHg); EVLWI: extravascular lung water index (ml/kg PBW); GEDVI: global enddiastolic volume index (ml/m^2). X-axis denotes different measurement time points, not days (corresponding to rows in Tab. 2). Arrows above X-axis indicate fluid administration (solid line) and diuretics (dotted line). Panel B. Detail during the first 2 days. X-axis shows first 5 measurements (see row 1 to 5 in Tab. 2). Opposite changes in volumetric (increase) and barometric (decrease) are observed during initial filling.

DVI 1080 ml/m^2, EVLWI 18 ml/kg PBW, conclusive with overfilling and worsening pulmonary edema in the absence of fluid responsiveness. Respiratory function deteriorated with a P/F ratio of 205, at an IPAP of 34 cmH_2O, PEEP 11 cmH_2O, while FiO_2 was increased from 45% to 65%. Lactate levels increased from 1.6 to 2.6 mmol/L.

Multiple choice question 4

At this stage the participants of the ISICEM and IFAD meetings were asked the fourth multiple choice question (MCQ4): "Taking into account the new results obtained with the transpulmonary thermodilution and the drop in urine output, what is your treatment of choice at this stage?" Possible answers were: (1) norepinephrine; (2) dobutamine; (3) fluid bolus; (4) diuretics or (5) other. Figure 10 shows the results of both votings. The majority of participants (49 to 66%) was now in favour of administration of diuretics. Finally the physicians fully understand the clinical situation of this patient who after the initial ebb phase of shock did not enter the flow phase spontaneously. His PEEP was increased to 18 cmH_2O, along with the administration of hypertonic albumin 20%, and he was given an infusion of lasic (frusemide, 60 mg/hr for 2 hours then followed by 10 mg/hr). This treatment was recently referred to as PAL [4]. By day 3 his cardiorespiratory condition improved with a drop in EVLWI to 15 ml/kg PBW, a PVPI of 1.9 and P/F ratio of 266 (with IPAP 34 and PEEP at 18 cmH_2O). Vasopressors and inotropes were titrated to norepinephrine doses of 0.11 ug/kg/min and Dobutamine at 3 ug/kg/min respectively, and he required less albumin 20% and less frusemide.

Further course

Things continued to fluctuate for the patient over the next few days but with two more episodes of frusemide infusions eventually his EVLWI came down to 8 ml/kg PBW on day eight. The patient was extubated on day 10 and left the ICU after 2 weeks. Fig. 11 shows the evolution of volumetric and barometric indicators during the first week (detail of first 2 days is shown in panel B showing opposite effects on volumetric and barometric preload indicators), while Fig. 12 shows the daily and cumulative fluid balance.

Multiple choice question 5

At this stage the participants of the ISICEM and IFAD meetings were asked the final multiple choice question (MCQ5): "What is your opinion on a positive cumulative fluid balance in septic shock?" Possible answers were: (1) Peripheral edema may look frightening for the relatives but it is just of cosmetic concern; (2) A cumulative fluid balance is always a biomarker of severity of illness; (3) A positive fluid balance is harmful and an independent predictor for morbidity and mortality; (4) Fluid balance must always be positive initially for a successful resuscitation of shock; or (5) I don't care. Figure 13 shows the results of both votings and it was re-assuring that the majority of participants (49 to 64%) were convinced that a positive cumulative fluid balance is indeed harmful.

In fact there is strong evidence to support conservative late fluid management in patients with septic shock, once the initial resuscitation is completed [28]. Hospital mortality was reduced in those patients who received adequate fluid resuscitation initially followed by conservative post resuscitation fluid management (defined as having 2 consecutive negative daily fluid balances within the first 7 days of ICU stay). In a meta-analysis of 40 studies that included 23625 patients, the mean cumulative fluid balance after 1 week was much lower in survivors than non survivors: 3,110 ml *vs* 7,738 ml (final manuscript under preparation) [18].

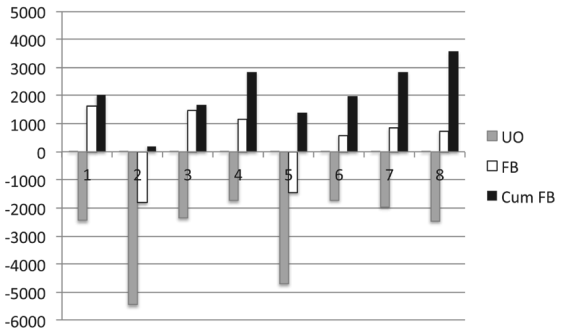

Fig. 12. Evolution of daily and cumulative fluid balance during the first week of ICU stay. Cum FB: cumulative fluid balance (ml); FB: fluid balance (ml); UO: urine output (ml); X-axis shows different ICU days.

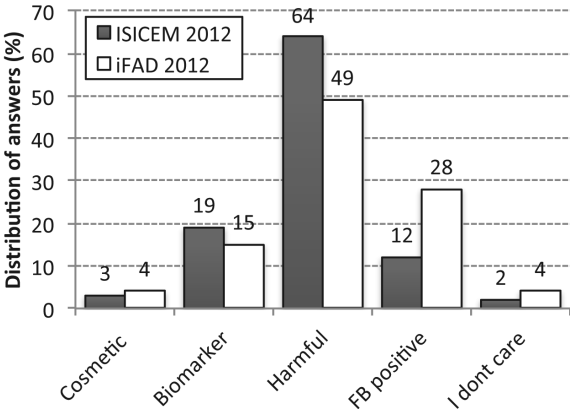

Fig. 13. Multiple choice question 5 (MCQ5): "What is your opinion on a positive cumulative fluid balance in septic shock?" Distribution of answers (in percentage) on MCQ5, grey squares denote the voting results of the ISICEM meeting while the white squares show the results of the iFAD meeting.

Fluid overload – an integrated approach

Patients do not die from anasarca (extreme edema), they die from multi-organ failure, and different organs need varying amounts of fluids to function. For example, lungs prefer to be dry but the liver cannot function if it is too dry. However when there is clinical evidence of capillary leak with peripheral edema then there will also be end-organ edema resulting in end-organ dysfunction, potentially leading to multiple organ dysfunction syndrome [12].

There are three phases or 'hits' a body takes when exposed to an inflammatory insult which includes trauma, infection, burns, sepsis or bleeding and this is summarized in Tab. 4. Recent evidence showed that the use of PAL treatment, combining PEEP with hypertonic albumin 20% and diuretics to initiate the flow phase (as we did in our patient) decreased EVLWI, IAP and daily and cumulative fluid balance, duration of mechanical ventilation and increased P/F ratio and survival in 57 patients with ALI compared to 57 matched controls [4]. PAL works as follows: the PEEP moves fluids from the alveoli into the interstitium (IS), thereby increasing interstitial hydrostatic pressure and decreasing interstitial oncotic pressure and moving IS fluids towards the capillaries. The hyperoncotic albumin 20% increases the intravascular oncotic pressure thereby removing fluids from the interstitium into the capillaries and finally the frusemide (Lasix) helps to remove the excess fluids from the patient.

Key messages

In this patient, that developed shock within 18 hours of ICU admission the dynamic evolution is presented. Despite initial normal (and thus adequate) filling pressures, further fluid resuscitation was needed to overcome the ebb phase (this was guided by functional hemodynamic parameters and volumetric preload indices). Diuretics were initiated after 24 hours to help the patient to transgress to the flow phase because of respiratory failure due to capillary leak as evidenced by increased extravascular lung water. It is interesting to see that based on barometric preload indicators many physicians were reluctant to start initial fluid resuscitation, this became clear once volumetric monitoring was performed with transpulmonary thermodillution. This case nicely demonstrates the biphasic clinical course from ebb to flow during shock as well as the inability of traditional filling pressures to guide us through these different phases. It also provides answers to the four crucial questions that need to be solved in order not to do any harm to the patient.

It is important to know and understand:
— when to start giving fluids (low GEF/GEDVI, high PPV and positive PLR, increased lactate);
— when to stop giving fluids (high GEF/GEDVI, low PPV, negative PLR, normalized lactate);
— when to start removing fluids (high EVLWI, high PVPI, raised IAP, low APP defined as MAP minus IAP, positive cumulative fluid balance).
— when to stop fluid removal (low ICG-PDR, low APP, low $ScvO_2$, neutral cumulative fluid balance).

However one must realize that the above mentioned thresholds are moving targets but also with moving goals (from early adequate goal directed therapy, over late conservative fluid management towards late goal directed fluid removal). And above all, one must always bear in mind that unnecessary fluid loading may be harmful. If the patient does not need fluids, don't give them, and remember that the best fluid may be the one that has not been given to the patient…!

It is essential to give the right fluid at the right time in the right fashion, and to use the correct monitor correctly.

Table 3. Evolution of respiratory and oxygenation parameters. Abbreviations and units: IPAP: inspiratory positive airway pressure (cmH_2O); PEEP: positive endexpiratory pressure (cmH_2O); P/F: pO_2 over FiO_2 ratio; RR: respiratory rate; TV: tidal volume (ml); VDR4: high frequency percussive ventilator; Vent: type of ventilator.

Day	Time	Vent	pO_2	pCO_2	P/F	lactate	pH	RR	TV	IPAP	PEEP
1	17:00	VDR4	82,5	42,8	121,3	1,82	7,25	24	442	30	6
1	19:00	VDR4	83,5	44,9	157,5	2,08	7,36	24	660	32	10
2	04:00	VDR4	86,5	32,7	192,2	3,56	7,41	16	680	32	11
2	12:00	VDR4	88,5	38,9	205,8	1,63	7,36	16	665	34	18
2	20:00	VDR4	167,6	37,8	316,2	2,44	7,48	16	670	35	20
3	04:00	VDR4	93,3	39,5	266,6	2,1	7,46	16	680	34	18
3	16:00	VDR4	103,9	35	494,8	1,79	7,48	16	710	34	16
4	10:00	EVITA	112	39,7	311,1	2,23	7,44	18	633	32	7
4	18:00	EVITA	76,8	33,8	295,4	1,45	7,48	16	899	30	6
5	10:00	EVITA	50,3	25,6	98,6	1,05	7,53	17	735	30	6
5	20:00	VDR4	102,3	28,7	292,3	1,65	7,58	17	640	32	14
6	04:00	VDR4	133,2	26,3	380,6	1,1	7,59	18	630	34	19
6	11:00	VDR4	143,2	24,6	477,3	1,04	7,59	17	630	33	14
6	17:00	VDR4	133,6	29,1	534,4	0,96	7,54	14	755	32	9
7	06:00	EVITA	92,3	41,7	355,0	0,77	7,44	12	641	30	9
7	12:00	EVITA	91,2	33,1	364,8	0,91	7,51	12	365	30	7
7	18:00	EVITA	66,7	28,2	115,0	1,58	7,55	13	850	28	7
8	05:00	EVITA	104,9	36	308,5	0,77	7,45	10	1020	28	7
8	10:00	EVITA	120,4	32,5	334,4	0,74	7,47	15	793	26	6
8	20:00	EVITA	107,6	32,2	358,7	0,59	7,44	11	900	26	6
9	05:00	EVITA	81,9	32,1	273,0	0,5	7,44	12	768	26	6

Table 4. The 3 hit model of shock.

HIT	First HIT – Acute Inflammatory Insult	Second HIT – Ischemia Reperfusion leading to MODS	Third HIT – if initial treatment unsuccessful
ORGAN FUNCTION	Leading to systemic inflammatory response, microcirculatory dysfunction, and distributive shock (hypotension, hypovolemia, oliguria, myocardial depression, interstitial edema formation, tissue hypoxia, increasing lactate levels)	Organ dysfunction could be acute lung injury, acute bowel injury, acute kidney injury, liver failure and central nervous system failure	Because of globally increased permeability syndrome, edema occurs in the lung, gut, kidneys, peripheries and brain with potentially devastating results
FLUIDS	Fluids are live saving	Fluids are considered as a biomarker for critical illness	Fluids become toxic and futile
FLUID BALANCE	Fluid balance should be positive	Zero fluid balance required	Fluid balance should be negative
TARGET	SVV, PPV, APP (MAP-IAP), PLR, TEO	EVLWI, PVPI, Intra Abdominal Pressure	ICG PDR, $ScvO_2$
GOAL	Early adequate goal directed fluid therapy	Late conservative fluid strategy	Late goal directed fluid removal

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