The process of hemodynamic stabilization

What's going wrong?

Differential diagnosis in hemodynamic failure

Azriel Perel

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

ISICEM 2007 Brussels

Disclosure

The speaker cooperates with the following companies

CritiSense

Drager-Siemens

iMDsoft

InSightec

Pulsion*

Preliminary statements

Critically ill patients do often have complex hemodynamics (hypovolemia, myocardial depression or both).

Only in rare case does the ‘diagnosis’ tell you what is the main physiological disturbance.

Co-morbidities often complicate the hemodynamic status.

Critically ill patients often present us with therapeutic conflicts (e.g., hemodynamic instability and ARDS).

In such situations therapeutic decisions are often critical since:

- **Occult hypoperfusion is associated with increased mortality.**
- **Volume overload may cause or worsen heart failure and/or pulmonary (and other organ) edema and is associated with increased mortality.**

HEMODYNAMIC MONITORING

- The ‘minimalist’ approach
- The ‘consensus’ approach
- The ‘advanced cardiopulmonary monitoring’ approach

Post from the ccm-l@list.pitt.edu 2003

"72yo man with a significant cardiac history who underwent removal of massive renal cell carcinoma and a necrotic gallbladder.

Following 24 hrs - oliguric, hypotensive but responsive to fluids. **20L positive balance over 24h.** Blew up like a balloon, but interestingly urine output still 45ml/hr. On a bit of noradrenaline…

I came into the unit at 0400 to start CVVHD... …. **whilst I was scrubbing he arrested."**

Question: "Don't you think that the patient may have been under-monitored?"

"The biggest problem with ALL the fancy numbers (and even the non-fancy numbers like CVP & MAP), is that in the individual patient…you have NO idea what the "best" number is supposed to be….

So then we get back to old fashioned clinical examination, measurement of indices of tissue function, and careful therapeutic trials, which may in some cases give us information later than the fancy machines, but at least the information is reliable."

I call this the "Back to Nature" movement….

The PiCClin Study
Part I: Clinicians' prediction of advanced cardiopulmonary variables in critically ill patients.

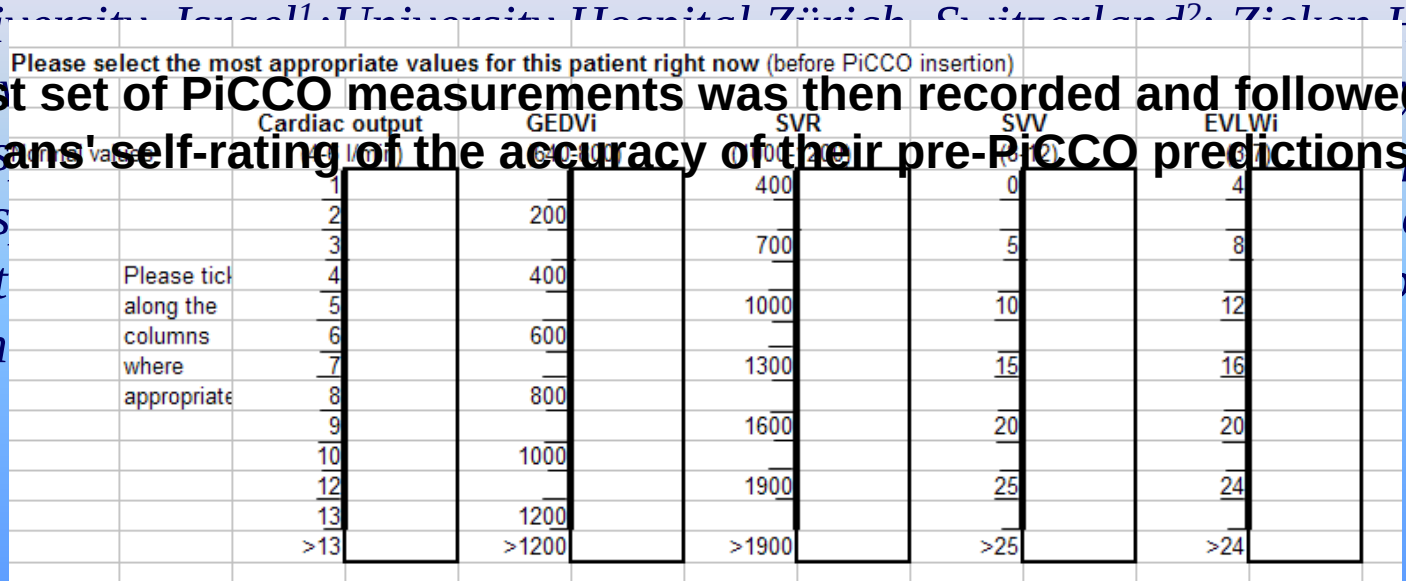
Methods: Cardiopulmonary assessment was done in critically ill patients from 12 European ICU's just before the use of the PiCCO monitoring system (Pulsion, Germany).

A Perel[1], M Maggiorini[2], M Malbrain[3], JL Teboul[4], J Belda[5], E Fernández Mondéjar[6], M Kirov[7], J Wendon[8], G Kourakin[1], R Lussmann[2], A Haller[2], R Stocker[2], M Lang[2], A Aguilar[5], A Smetkin[7], C Karvellas[8]

Intensive Care Units of the Sheba Medical Center, Tel Aviv Uni... Israel[1]; University Hospital Zürich, Switzerland[2]; Ziekenhuis Ant... Belgium[3]; Hôpital de Bicêtre, France[4]; Hospital Universitario La Paz, Madrid, Spain[5]; Hos... Stat... Lon...

One to four physicians per patient independently predicted the cardiac output (**CO**), systemic vascular resistance (**SVR**), preload (indexed global end-diastolic volume - **GEDVi**), fluid responsiveness (stroke volume variation during mechanical ventilation - **SVV**), and indexed extravascular lung water (**EVLWi**).

The first set of PiCCO measurements was then recorded and followed by physicians' self-rating of the accuracy of their pre-PiCCO predictions.



The patient population included 165 patients, which were evaluated by 135 residents and 122 specialists (total of 257 questionnaires).

The main reasons for using the PiCCO included:

- **Unclear fluid status (109)**
- **Suspected sepsis / septic shock (70)**
- **Respiratory failure (42)**
- **Cardiogenic shock (19)**
- **Renal failure (27)**
- **Other (18).**

The accuracy of predicted cardiopulmonary parameters

	CO (n=256)	SVR (n=254)	GEDVi (n=253)	SVV (n=214)	EVLWi (n=235)
Underestimation >20%	127 (49.6%)	46 (18.1%)	72 (28.0%)	85 (39.7%)	69 (29.4%)
Within ± 20%	99 (38.6%)	90 (35.4%)	93 (50.2%)	64 (29.9%)	93 (39.6%)
Overestimation >20%	30 (11.7%)	116 (45.7%)	73 (21.0%)	65 (30.4%)	73 (31.1%)

The PiCClin Study

Conclusions PiCClin (part I):

The ability of physicians to predict advanced cardiopulmonary parameters based on clinical evaluation and conventional monitoring alone has considerable limitations and is not improved by experience.

Intensivists often tend to assume that CO is lower, that SVR is higher and that preload is lower than the actually measured values. This may be due to a common misinterpretation of hypotension.

Intensive Care Med
DOI 10.1007/s00134-007-0531-4

INTERNATIONAL CONSENSUS CONFERENCE

Massimo Antonelli
Mitchell Levy
Peter J. D. Andrews
Jean Chastre
Leonard D. Hudson
Constantine Manthous
G. Umberto Meduri
Rui P. Moreno
Christian Putensen
Thomas Stewart
Antoni Torres

Hemodynamic monitoring in shock and implications for management

International Consensus Conference, Paris, France, 27–28 April 2006

Received: 13 October 2006
Accepted: 5 January 2007

Should we monitor preload and fluid responsiveness in shock?

How and when should we monitor stroke volume or cardiac output in shock?

What is the evidence for using hemodynamic monitoring to direct therapy in shock?

Intensive Care Med
DOI 10.1007/s00134-007-0531-4

14. a) We recommend frequent measurement of blood pressure and physical examination variables (including signs of hypoperfusion, urine output and mental status) in patients with a history and clinical findings suggestive of shock.

b) We recommend invasive blood pressure measurement in refractory shock. Level 1; QoE very low (D)

15. We do not recommend the routine use of the PAC for patients in shock. Level 1; QoE high (A)

Intensive Care Med
DOI 10.1007/s00134-007-0531-4

6. We recommend that preload measurement alone not be used to predict fluid responsiveness.
Level 1; QoE moderate (B)

7. We recommend that in shock, low values of commonly used static measures of preload such as CVP, RAP, PAOP (for example less than 4 mmHg) and ventricular volumes, should lead to immediate fluid resuscitation with careful monitoring.
Level 1; QoE low (C)

Surviving Sepsis Campaign Guidelines (Rivers, NEJM 2001)

Initial resuscitation *Begin resuscitation immediately in patients with hypotension or elevated serum lactate.*

Resuscitation goals:

- *Central venous pressure: 8-12 mm Hg*
- *Mean arterial pressure ≥ 65 mm Hg*
- *Urine output ≥ 0.5 mL.kg-1.hr-1*
- *Central venous or mixed venous oxygen saturation ≥ 70%*

If central venous oxygen saturation or mixed venous oxygen saturation of 70% is not achieved with a central venous pressure of 8-12 mm Hg, then transfuse packed red blood cells to achieve a haematocrit of ≥ 30% and/or administer a dobutamine infusion of up to a maximum of 20 µg.kg-1.min-1.

Practice parameters for hemodynamic support of sepsis in adult patients in sepsis

Task Force of the ACCCM and the SCCM, CCM 2004

“In most patients with septic shock, CO will be optimized at filling pressures between 12 and 15 mm Hg.”

Cardiac filling pressures are not appropriate to predict hemodynamic response to volume challenge*

David Osman, MD; Christophe Ridel, MD; Patrick Ray, MD; Xavier Monnet, MD, PhD; Nadia Anguel, MD; Christian Richard, MD; Jean-Louis Teboul, MD, PhD

CCM 2007 35:64-8

- The sign … d responsive … hat of CVP, as indicate … the ROC curves (0.5 …

- A CVP o … mm Hg predicted … positive predictive … pectively.

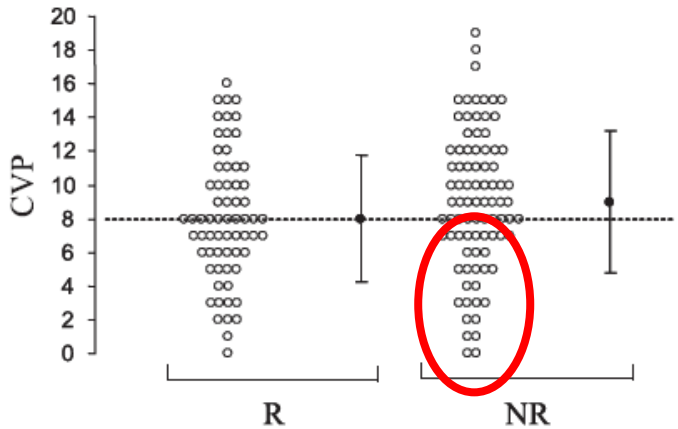
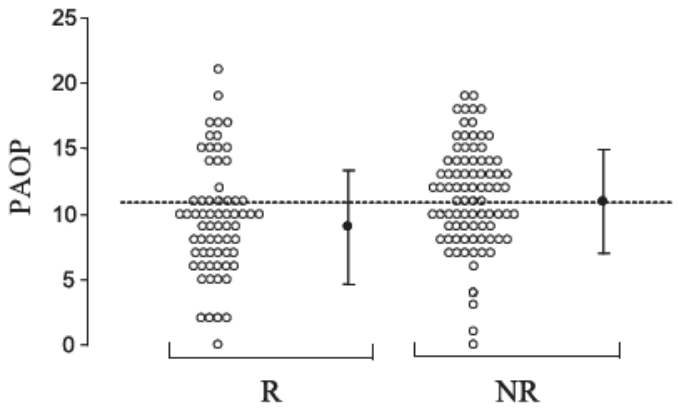


Figure 2. Individual values (*open circles*) and mean ± SD (*closed circles*) of pre-infusion central venous pressure (*CVP*) (both expressed in millimeters of mercury) in responders (*R*) and nonresponders (*NR*).



Intensive Care Med
DOI 10.1007/s00134-007-0531-4

8. We recommend a fluid challenge to predict fluid responsiveness…with a goal of obtaining a rise in CVP of at least 2 mmHg. A positive response includes measures of improved cardiac function and tissue perfusion.

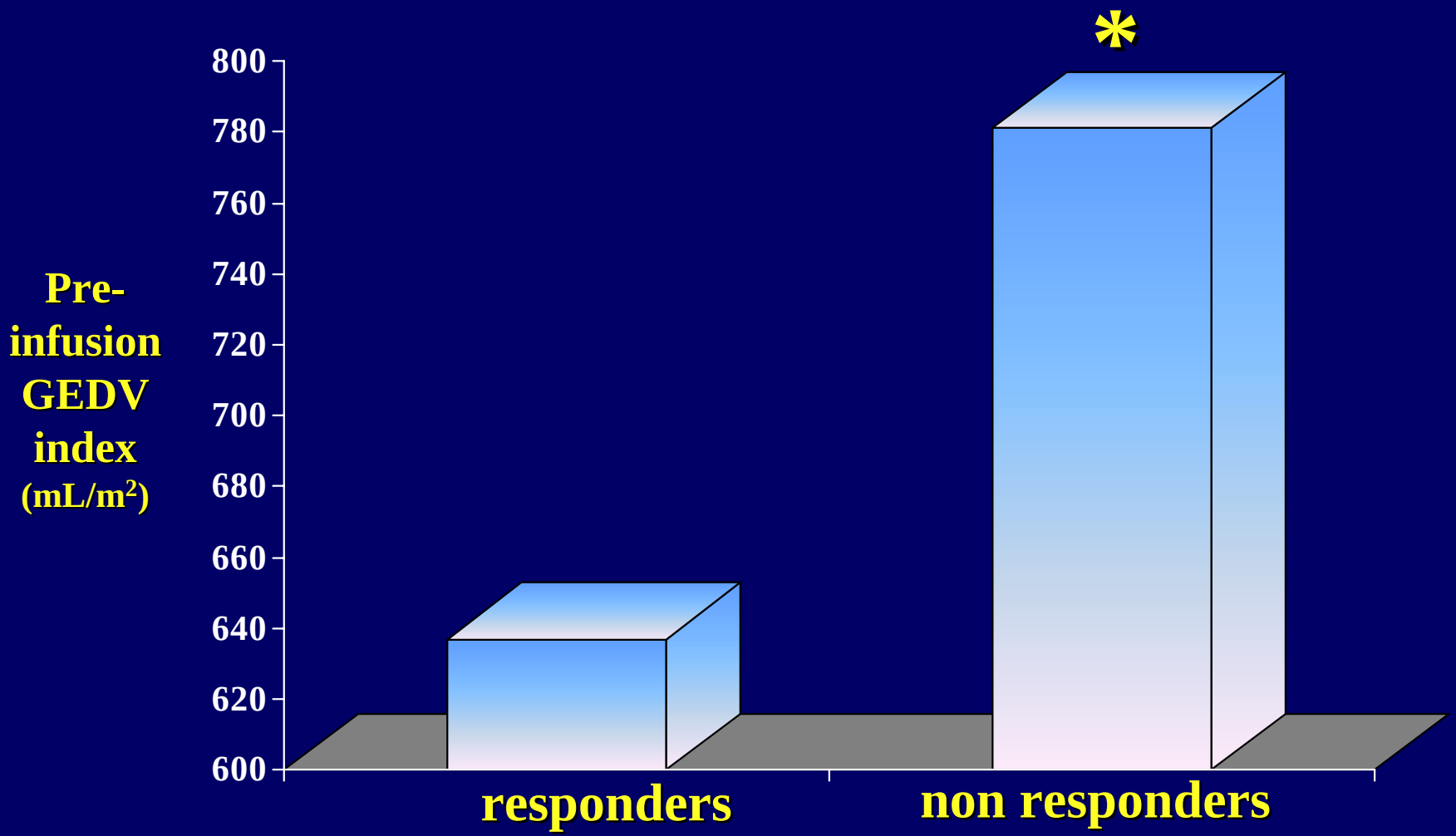
Level 1; QoE low (C)

50% of critically ill patients are loaded with fluids unnecessarily!
Hemodynamic response to fluid loading

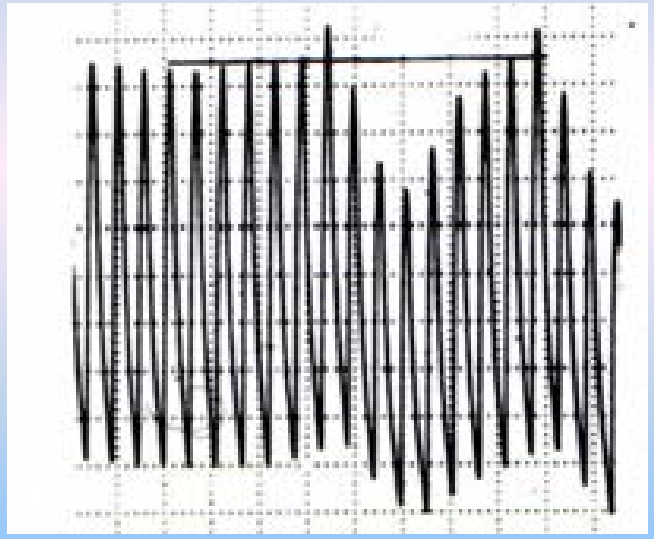
	Patients	Definition of Responders	N	Challenge	Responders
Preisman S (2005)	Cardiac surgery	> 15% SV	18	250 mL colloids	32/70 VLS (46%)
Hofer CK (2005)	Cardiac surgery	> 25% SVI	35	10 mL/kg (IBW) 6% HES	21 (60%)
Swensen CH (2006)	Abdominal surgery	Increase in CO	10	25 mL/kg of Ringer	4 (40%)
Tavernier B (1998)	Sepsis w. circulatory failure	> 15% SVI	15	500-1000 mL HES	21/35 VLS (60%)
Michard F (2000)	Sepsis w. circulatory failure	> 15% CI	40	500 mL HES	16 (40%)
Michard F (2003)	Septic shock	> 15% SVI	27	500 mL HES	32/66 VLS (48%)
Feissel M (2005)	Septic shock	> 15% CI	20	8 ml/kg HES	13/22 VLS (59%)
Monnet X (2005)	Critically ill w. circulatory failure	> 15% increase in ABF (Doppler)	38	500 ml NS	20 (53%)
Vallee F (2005)	Critically ill w. circulatory failure	> 10% increase in SVI	51	4 ml/kg colloid X 2	20 (39%)
Heenan S (2006)	Critically ill w. circulatory failure	> 15% in CO	21	1 L Ringer or 500 mL HES	9 (43%)
Lafanechère A (2006)	Critically ill w. circulatory failure	> 15% increase in ABF (Doppler)	22	PLR and 500 ml NS	10 (45%)
Osman D (2007)	Sepsis	> 15% in CO	96	500 mL HES	65/150 VLS (43%)

Global end-diastolic volume as an indicator of cardiac preload in patients with septic shock.

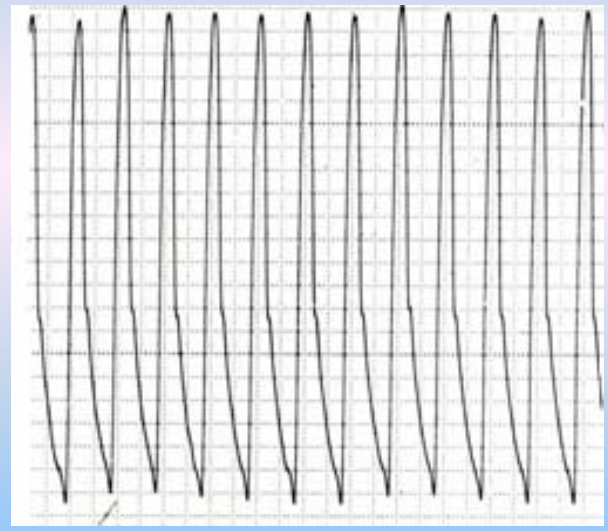
Michard F, et al. Chest 2003, 124:1900-8



Functional hemodynamic parameters (SPV, PPV, SVV) are the most sensitive parameters for the assessment of fluid responsiveness in mechanically ventilated patients



Responsive



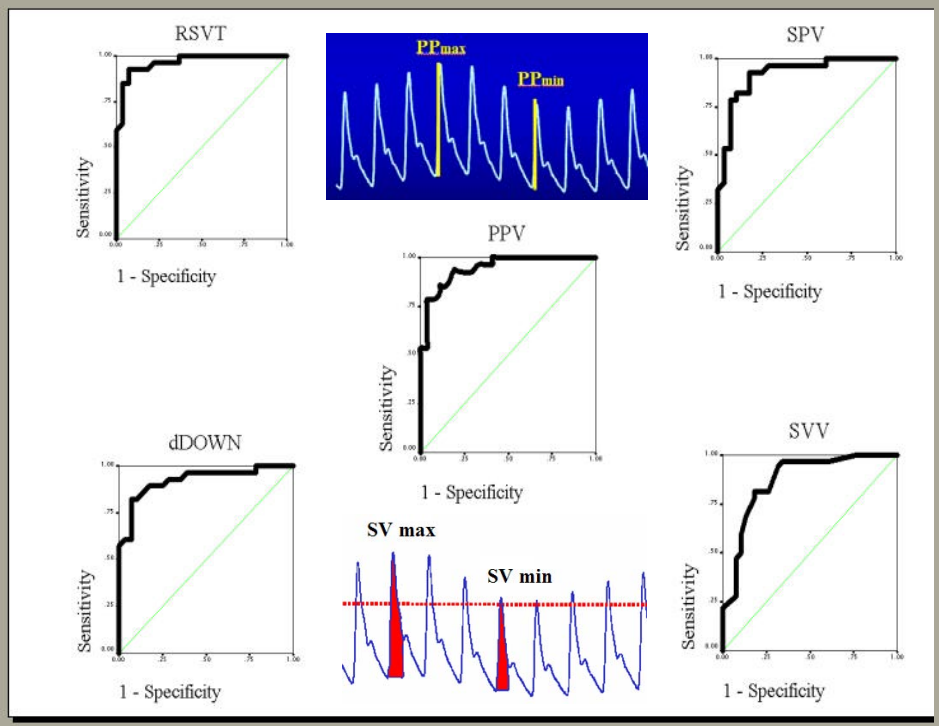
Non-responsive

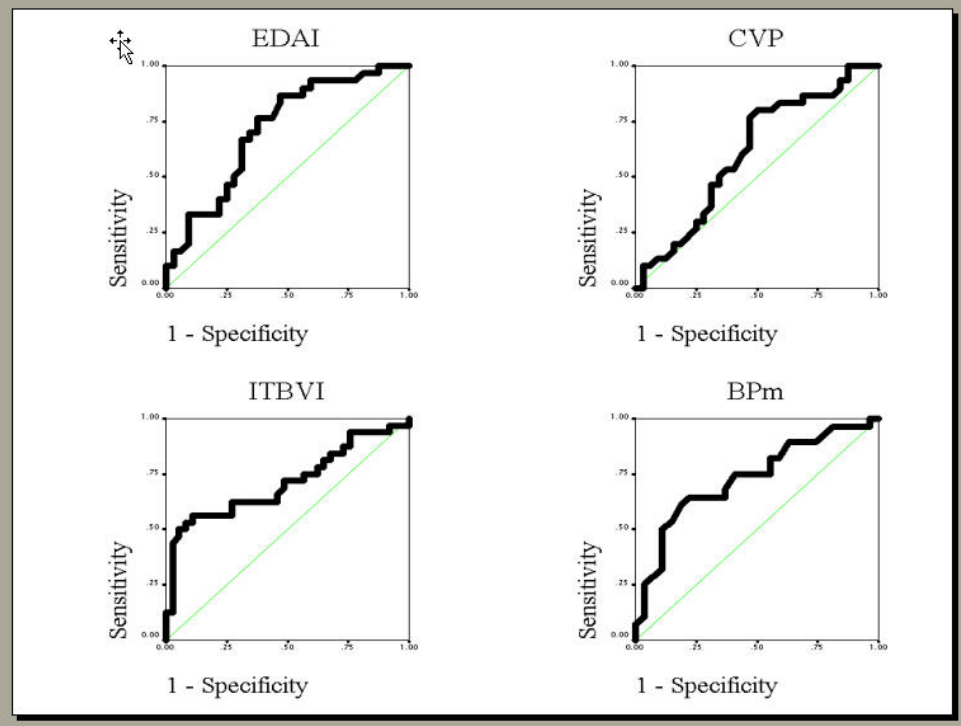
British Journal of Anaesthesia **95 (6)**: 746–55 (2005)
doi:10.1093/bja/aei262

BJA

Predicting fluid responsiveness in patients undergoing cardiac surgery: functional haemodynamic parameters including the Respiratory Systolic Variation Test and static preload indicators†

S. Preisman*, S. Kogan, H. Berkenstadt and A. Perel‡





Intensive Care Med
DOI 10.1007/s00134-007-0531-4

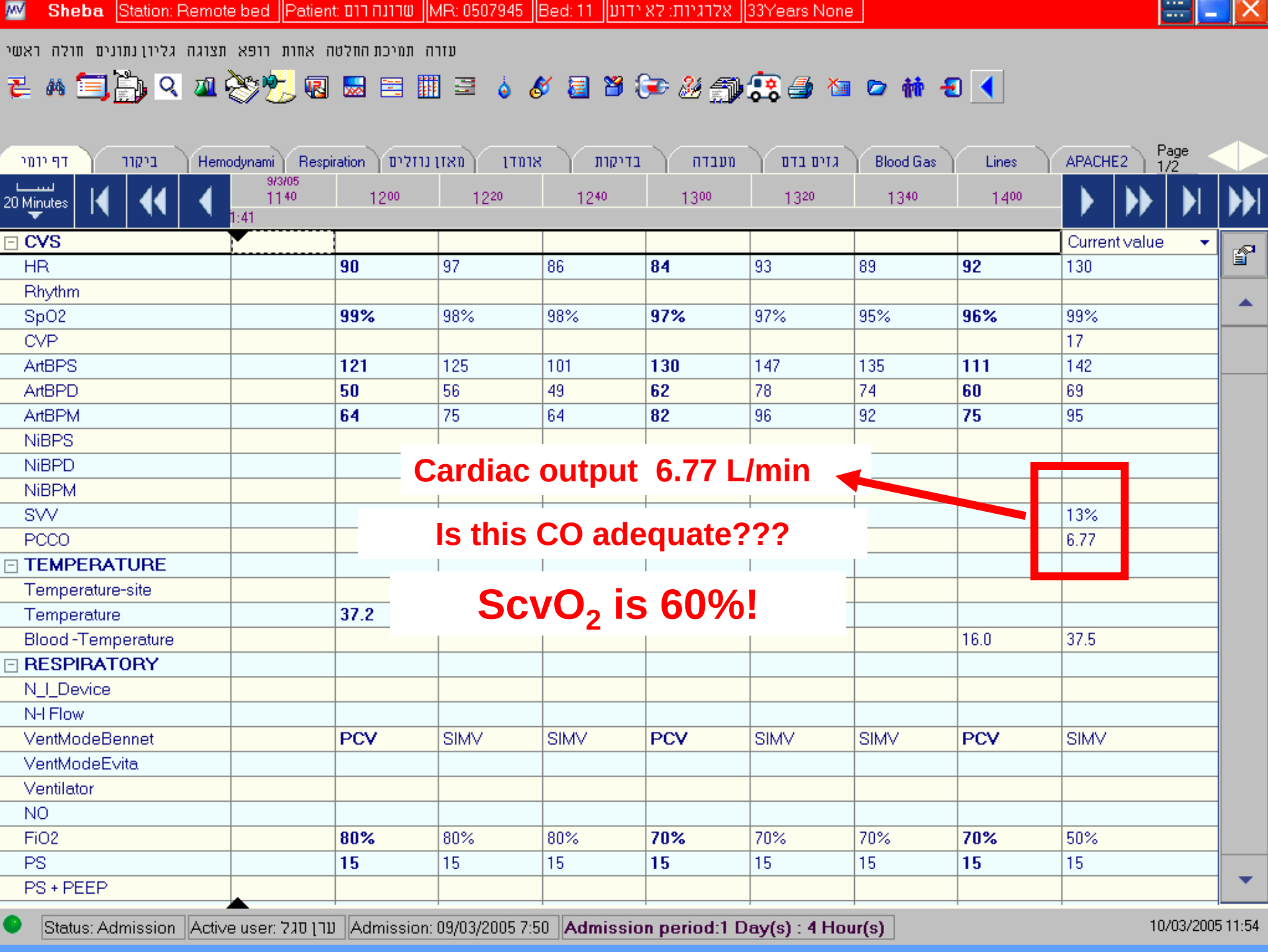
9. We do not recommend the routine use of dynamic measures of fluid responsiveness (including but not limited to pulse pressure variation, aortic flow changes, systolic pressure variation, respiratory systolic variation test, and collapse of vena cava).
Level 1; QoE high (A)

There may be some advantage to these measurements in highly selected patients.
Level 1; QoE moderate (B)

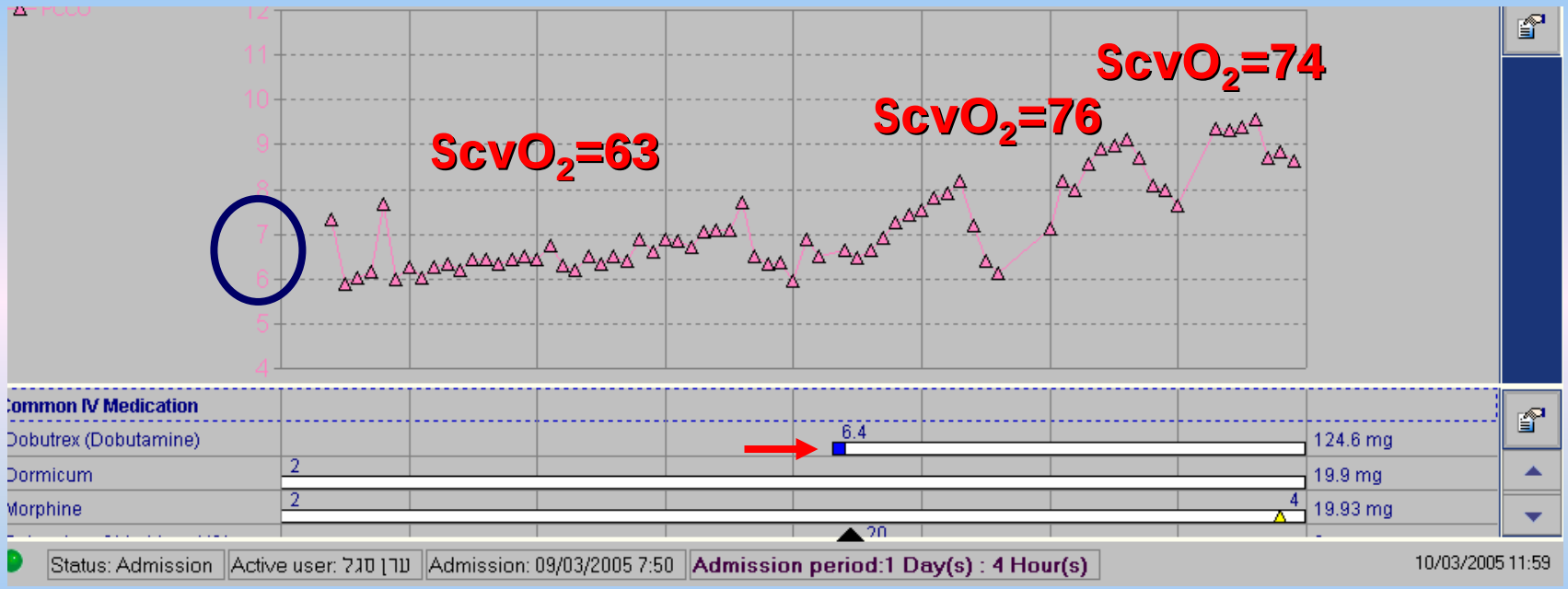
Intensive Care Med
DOI 10.1007/s00134-007-0531-4

10. We do not recommend routine measurement of CO for patients with shock.
Level 1; QoE moderate (B)

11. We suggest considering echocardiography or measurement of CO for diagnosis in patients with clinical evidence of ventricular failure and persistent shock despite adequate fluid resuscitation.
Level 2 (weak); QoE moderate (B)


Sheba
Station: Remote bed
Patient: שרונה רום
MR: 0507945
Bed: 11
אלרגיות: לא ידוע
33Years None
דף יומי
ביקור
Hemodynami
Respiration
מאזן נוזלים
אומדן
בדיקות
מעבדה
גזים בדם
Blood Gas
Lines
APACHE2
Page 1/2
20 Minutes
9/3/05
11:40
12:00
12:20
12:40
13:00
13:20
13:40
14:00
1:41
Current value
CVS
HR 90 97 86 84 93 89 92 130
Rhythm
SpO2 99% 98% 98% 97% 97% 95% 96% 99%
CVP 17
ArtBPS 121 125 101 130 147 135 111 142
ArtBPD 50 56 49 62 78 74 60 69
ArtBPM 64 75 64 82 96 92 75 95
NiBPS
NiBPD
NiBPM
SVV 13%
PCCO 6.77
TEMPERATURE
Temperature-site
Temperature 37.2
Blood -Temperature 16.0 37.5
RESPIRATORY
N_I_Device
N-I Flow
VentModeBennet PCV SIMV SIMV PCV SIMV SIMV PCV SIMV
VentModeEvita
Ventilator
NO
FiO2 80% 80% 80% 70% 70% 70% 70% 50%
PS 15 15 15 15 15 15 15 15
PS + PEEP
Cardiac output 6.77 L/min
Is this CO adequate???
ScvO2 is 60%!
Status: Admission
Active user: ערן סגל
Admission: 09/03/2005 7:50
Admission period:1 Day(s) : 4 Hour(s)
10/03/2005 11:54

Patient is given dobutamine



Conclusion: CO was high, but not high enough

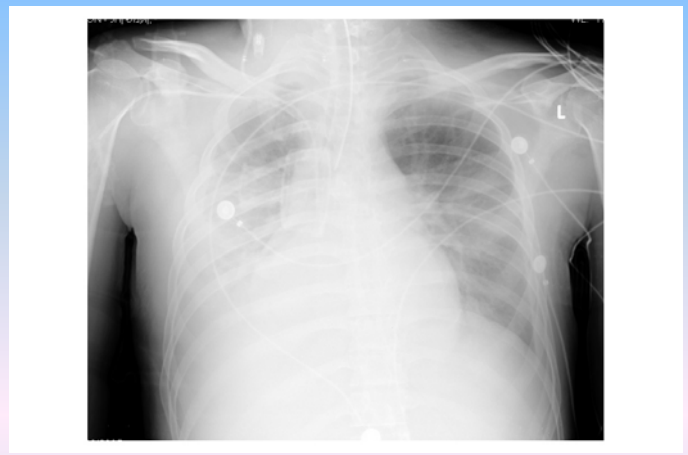
A man with fever and shortness of breath

$ScvO_2$ 72%

CVP 9 mmHg

Lactate 48

PaO_2/FiO_2 75 (PEEP 10)



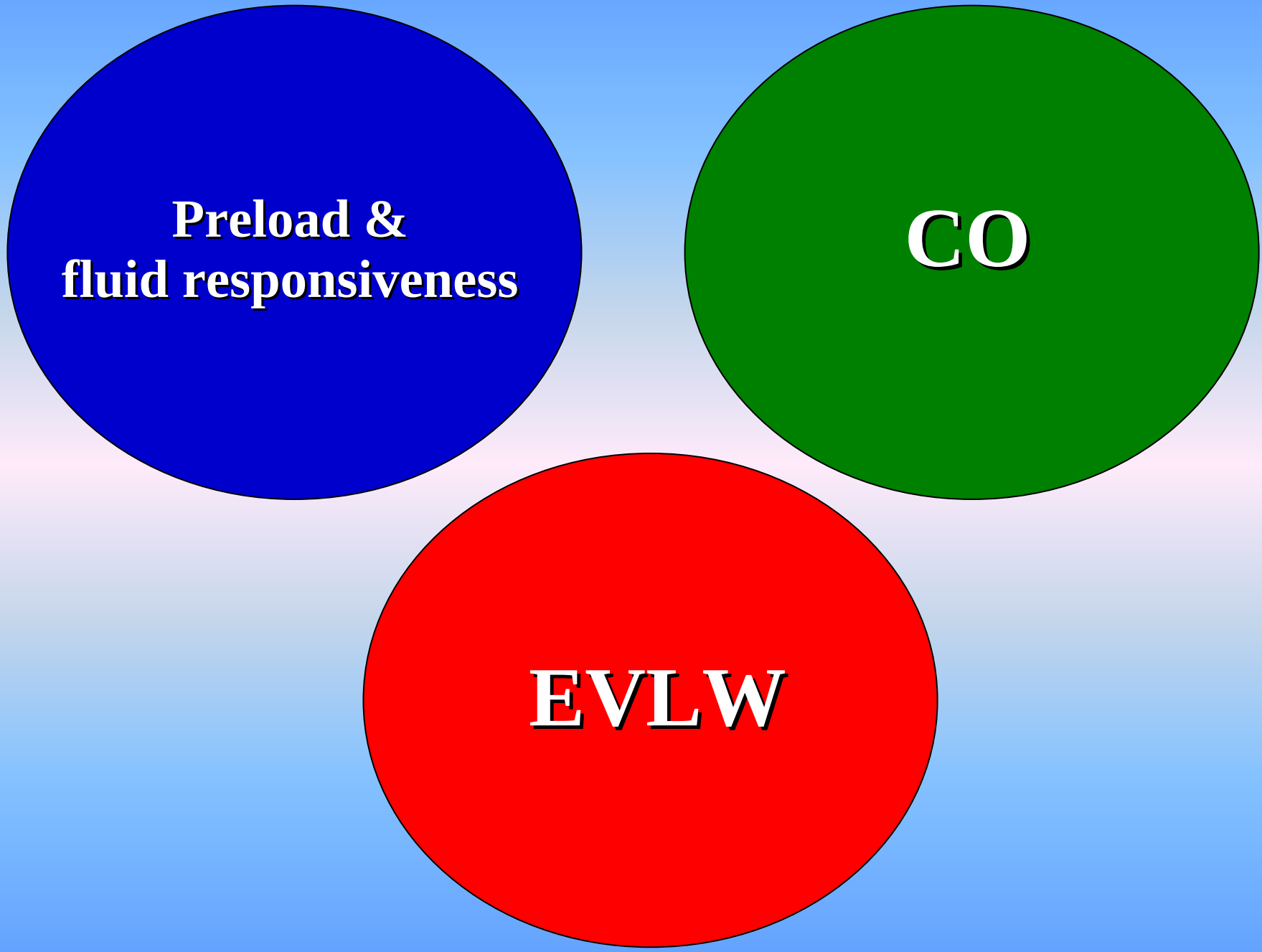
Following noradrenaline…

- Blood pressure increased to 120/65
- CO increased to 6.5 LPM (CI 3.7)
- $ScvO_2$ increased to 76%
- SaO_2 increased to 98% and FiO_2 decreased
- Urine output increased to 60 ml/h

• CO	3.8
• ITBVI	950
• EVLWI	15
• SVR	1100

The main reason to measure cardiac output is to identify those patients that have low (or high) CO values that are not evident clinically.

However, a low cardiac output value, like the $ScvO_2$, by itself, will tell you that something is wrong but not what is wrong and what should be done about it (fluids? inotropes?).


Preload &
fluid responsiveness
CO
EVLW

Practice parameters for hemodynamic support of sepsis in adult patients: 2004 update

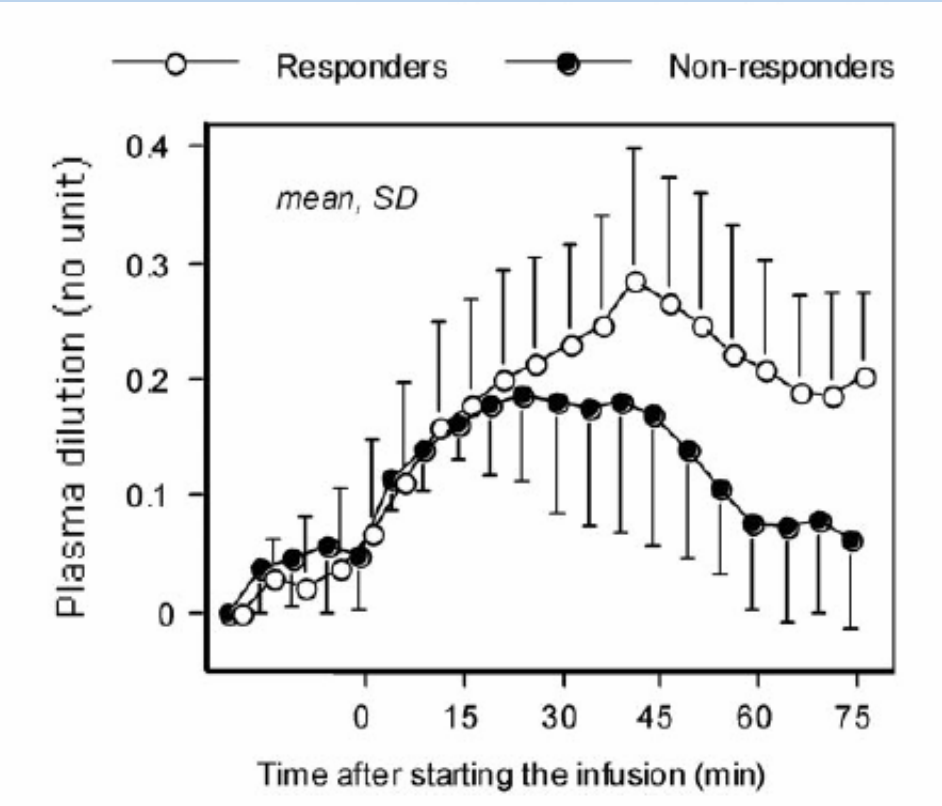
Task Force of the ACCCM and the SCCM, CCM Sept 2004

Pulmonary edema may occur as a complication of fluid resuscitation and necessitates monitoring of….

arterial oxygenation.

Intravascular Fluid Administration and Hemodynamic Performance During Open Abdominal Surgery

Svensen CH, Olsson J, Hahn RG. Anesth Analg 2006;103:671-6



In non-responders volume kinetic analysis suggested that 25% of the infused fluid resided in the central fluid space at the end of the infusion and only 3% at the end of the study.

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Comparison of Two Fluid-Management Strategies in Acute Lung Injury

The National Heart, Lung, and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network*

CONCLUSIONS

Although there was no significant difference in the primary outcome of 60-day mortality, the conservative strategy of fluid management improved lung function and shortened the duration of mechanical ventilation and intensive care without increasing nonpulmonary-organ failures. These results support the use of a conservative strategy of fluid management in patients with acute lung injury. (ClinicalTrials.gov number, NCT00281268.)

Should we ‘dry’ all patients that fulfil the ALI/ARDS criteria?

Michard F. et al. Chest 2004;125:1166-7

35% of ARDS patients had EVLW<7 ml/kg.

Patroniti N et al Crit Care Med 2005; 33:2547–54

Some ARDS patients had EVLWI values only slightly increased above normal.

Martin G et al Crit Care. 2005; 9:R74-82.

25% of ARDS patients had normal EVLW.

Research

Open Access

Extravascular lung water in patients with severe sepsis: a prospective cohort study

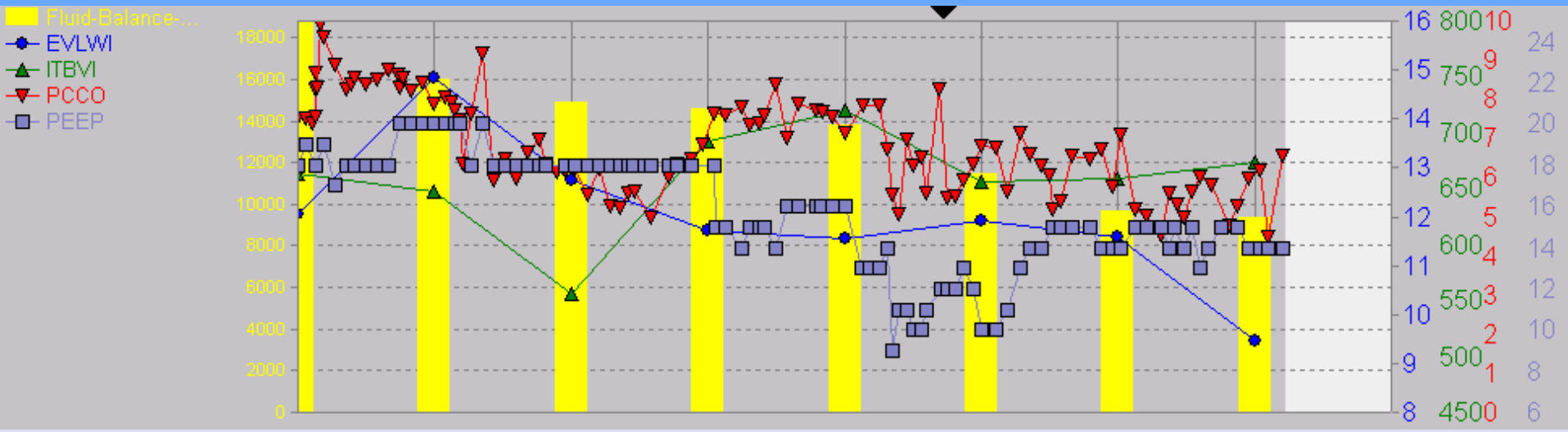
Greg S Martin[1], Stephanie Eaton[2], Meredith Mealer[3] and Marc Moss[4]

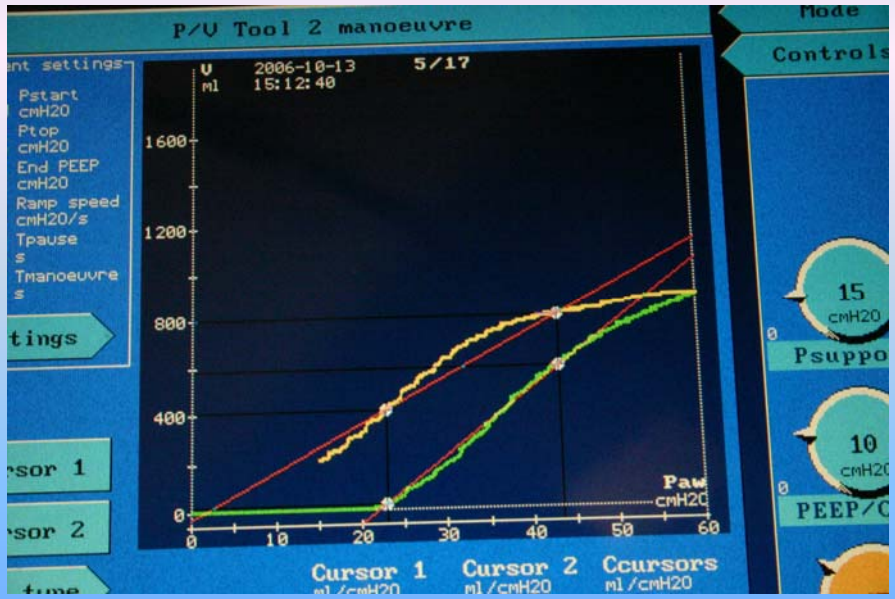
Critical Care 2005, **9**:R74-R82

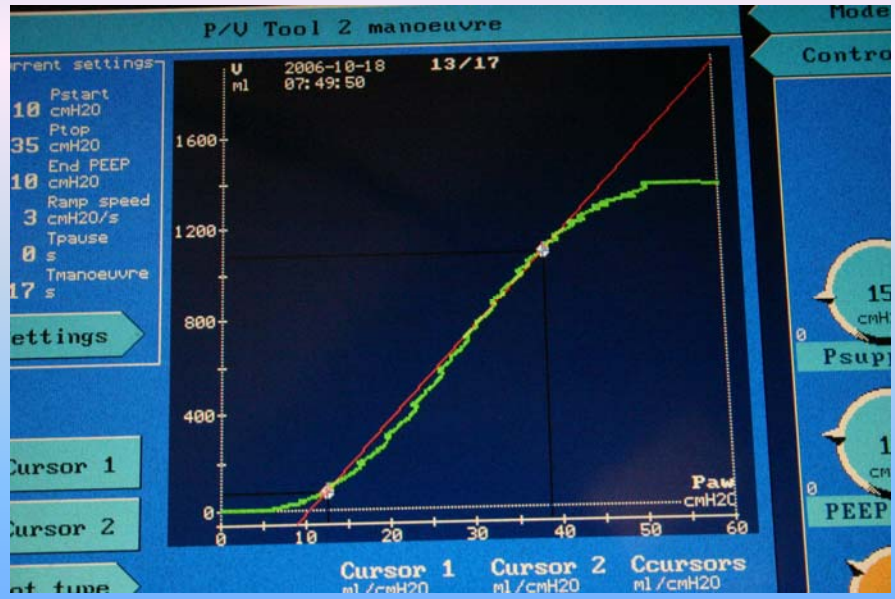
More than half of the patients with severe sepsis but without ARDS had increased EVLW, possibly representing subclinical lung injury.

Chronic alcohol abuse was associated with increased EVLW, whereas lower EVLW was associated with survival.

EVLW may improve both risk stratification and management of patients with severe sepsis.







13/10 - LIP 23

18/10 - LIP 12

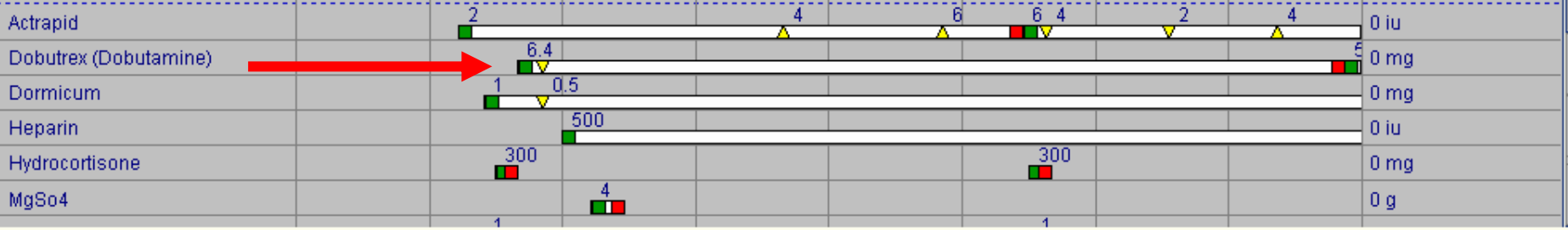
34 yr female; Very severe respiratory failure; Hemodynamic collapse; On noradrenaline.

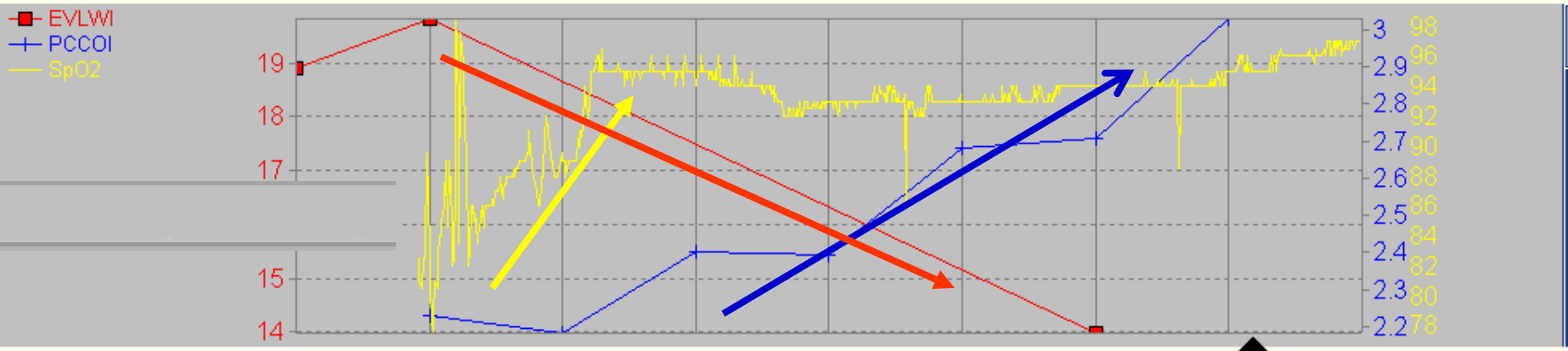
BP	113 / 67 mmHg	**CI**	2.7 l/min/m^2
HR	91 bpm	ITBVi	578 ml/m^2
Urine	Good	EVLWi	20 ml/kg
SaO_2	86% !!!	ICG PDR	6.7%
$ScvO_2$	80% !!!	(LiMON)	(18-25%)

Have we achieved initial resuscitation goals in this patient?

PICCO								ערך נוכחי
PCCOI		2.229	2.182	2.400	2.394	2.682	2.706	3.029
ITBVI	689	578					525	
EVLW	1228	1288					909	
EVLWI	18.9	19.8					14.0	
Picco SVRI		570.6	417.6	411.8	376.5	352.9	352.9	382.4
Picco_SV		47.18	40.52	44.62	42.38	46.9	46.53	54.66
Picco SVI		27.8	23.8	26.2	24.9	27.6	27.4	32.2

Common IV Medication





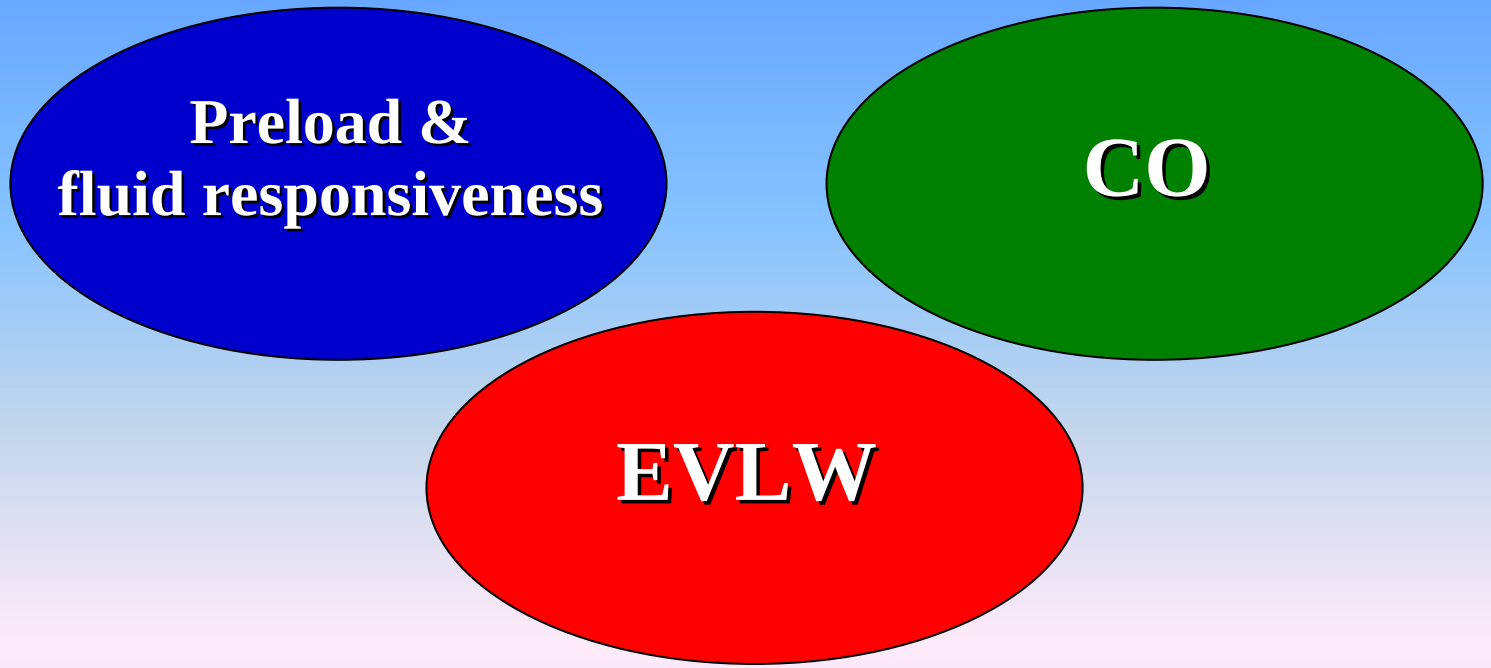
The PiCClin Study
Part II: Change of therapeutic plan following advanced cardiopulmonary monitoring in critically ill patients.

In the absence of further hemodynamic information, what would be your therapeutic decision?

Fluid loading	Red blood cells	Inotropic agent	Vaso-constrictor	Diuretic	Dialysis/ filtration	Other

The PiCClin Study
Part II: Change of therapeutic plan following advanced cardiopulmonary monitoring in critically ill patients.

	Original therapeutic plan	
	Pursued	**Changed**
Fluids (n=255)	68.2%	31.8%
Inotropes (n=257)	76.6%	23.3%
Vasoconstrictors (n=255)	76.5%	23.5%
Diuretics (n=254)	85.0%	15.0%



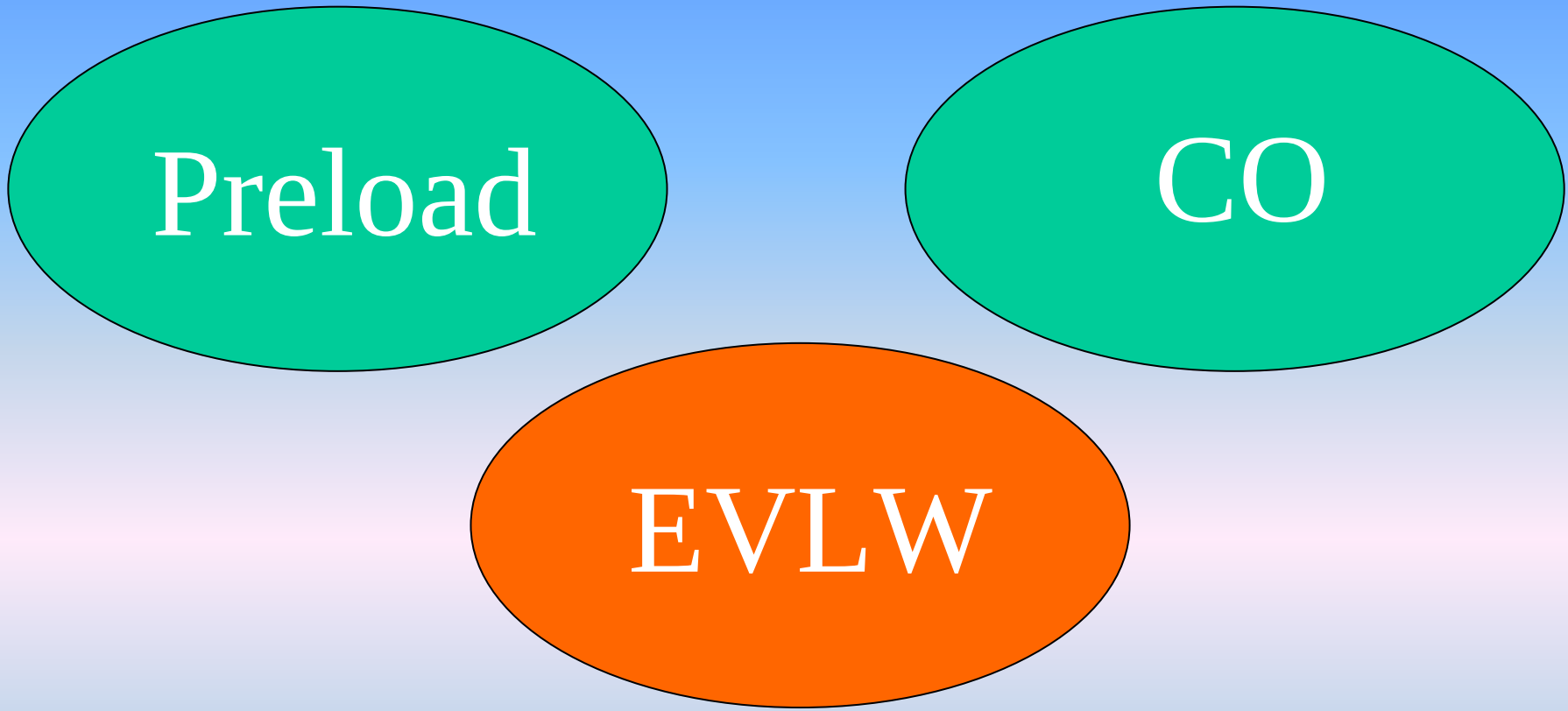
By monitoring this combination of parameters the PiCCO monitor offers most of the information necessary for hemodynamic management of critically ill patients, especially those who present significant therapeutic conflicts (e.g., heart vs. lungs).

The $ScvO_2$ and parameters of microcirculatory function may offer additio ation.

Hemodynamic monitoring should be kept as simple as possible

But not simpler!

Thank you!



Advanced hemodynamic monitoring should be applied to all patients that may be in low flow state and that may present significant therapeutic conflicts (e.g., heart vs. lungs)

The major problems with the interpretation of $ScvO_2$

- **Like the CO, a low SvO_2 tells you that something is wrong, but not what is wrong and what should be done about it (fluids? inotropes?).**

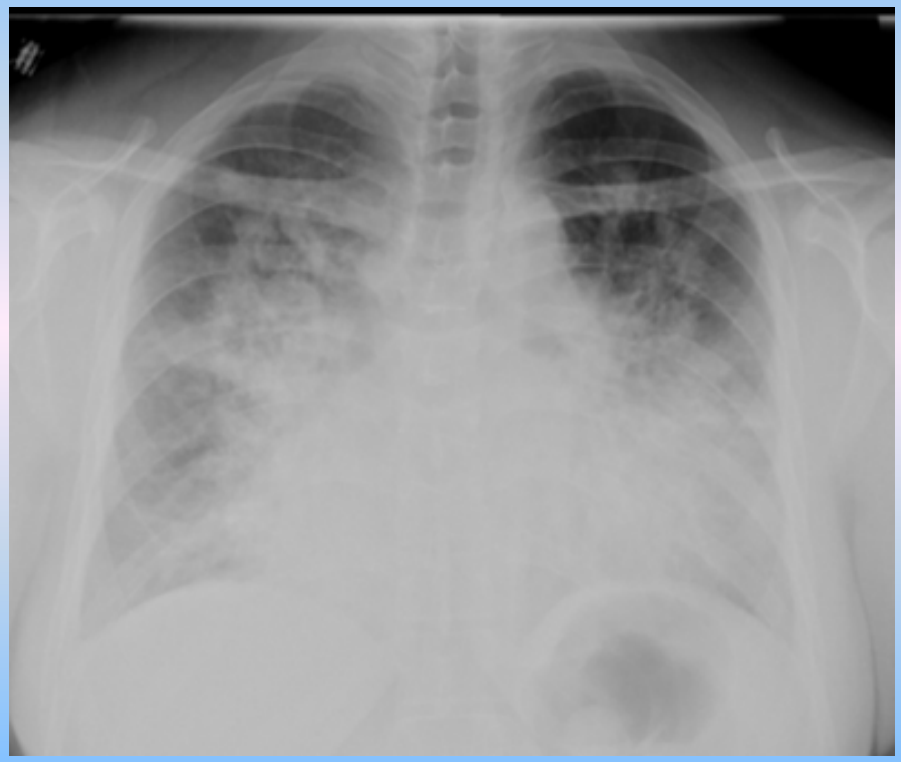
- **When the SvO_2 is normal or high - one cannot assume that all is well (e.g., CO normal) since in septic patients the $ScvO_2$ may be elevated due to an abnormally low O_2 extraction.**

Last but not least……

Even with the ‘best’ parameters it is not always easy to make the right decision.

- **A 31 year old patient with Down's syndrome is admitted to the ICU with severe pneumonia.**
- **s/p Closure VSD for CHF (age 6)**
- **The patient is an athlete and has just participated in a competitive swim in the San Francisco Bay.**
- **A few days later she started to have fever cough, white sputum, sore throat, and shortness of breath and was taken to the ER.**

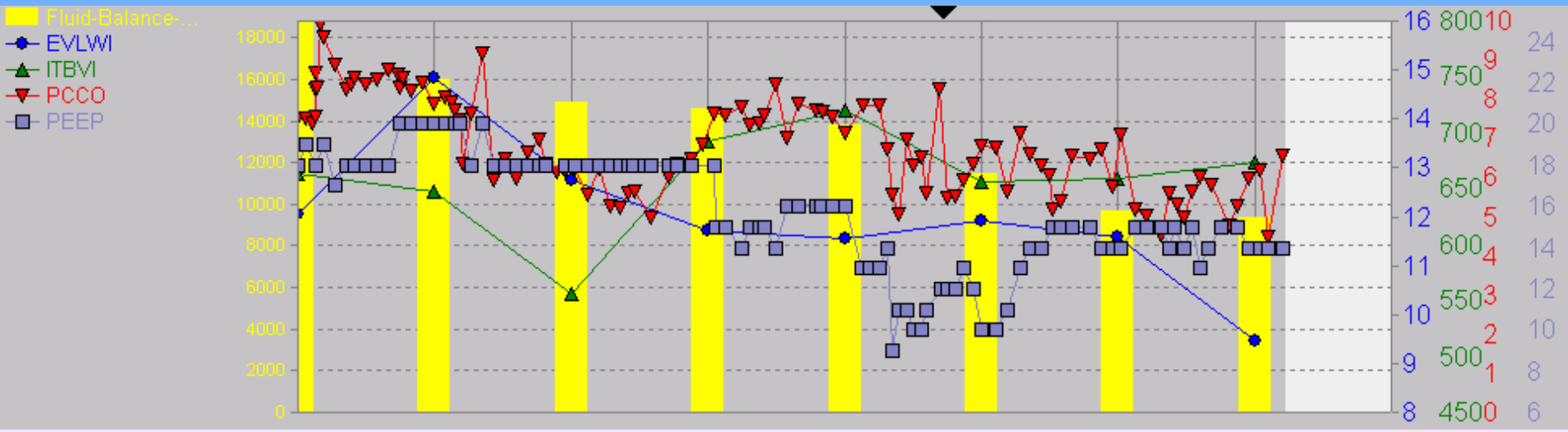
- **In the ER the patient was alert and oriented. She was short of breath and had very significant hypoxemia with an oxygen saturation of 60% (!) on room air.**



- **She was started on antibiotics and admitted to the ICU.**

In the ICU

- Stable hemodynamics with $ScvO_2$ 72 % and normal heart ECHO.
- Noradrenaline 0.02 mcg/kg/min due to SBP < 100.
- Decreased urine output < 700 cc/24 hr. Urine electrolytes consistent with a pre-renal state.
- **Positive fluid balance 12 L at Day 5 and 19 L at Day 7.**
- No improvement in respiratory status over the first 7 days (entered into surfactant study).
- Peep increased to 18 to maintain oxygenation.

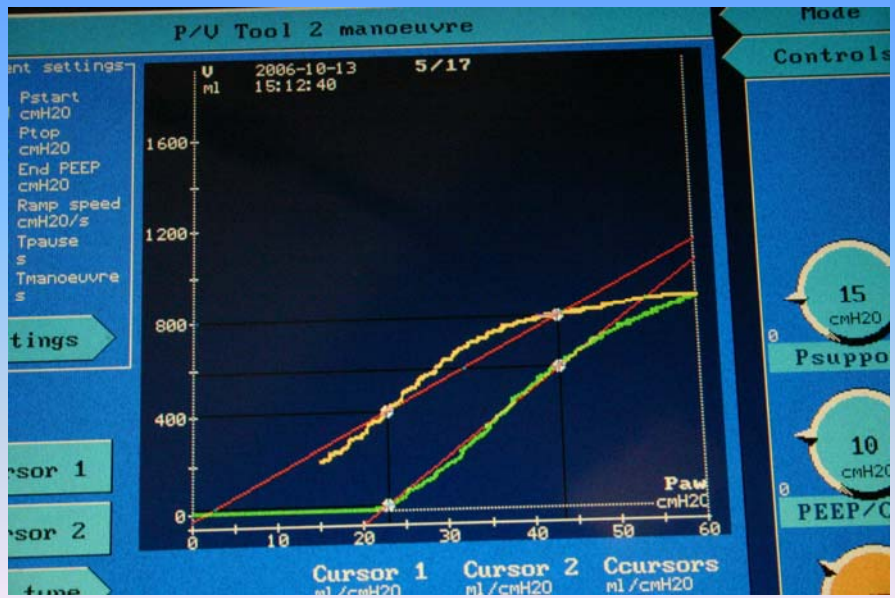


Even though the patient had low ITBV aggressive diuresis was started with daily negative fluid balance of 1-2.5 L.

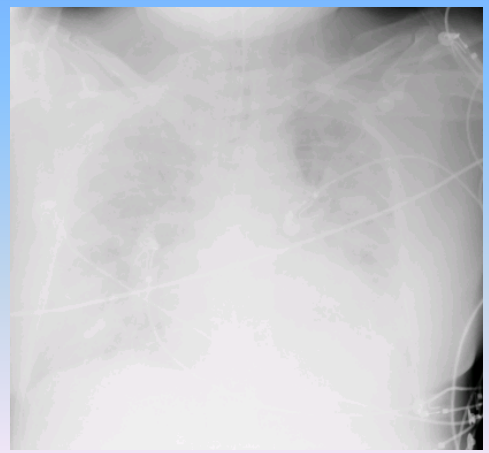
CO supported with dobutamine and later dopamine (due to bradycardia).

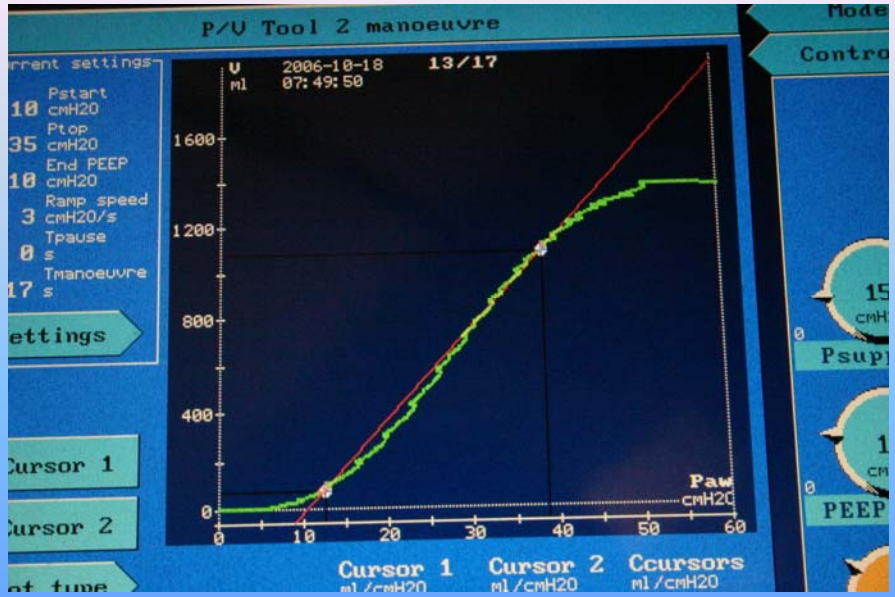
PEEP gradually reduced with decrease in EVLW and improved oxygenation.

ITBVi levels – unchanged!!

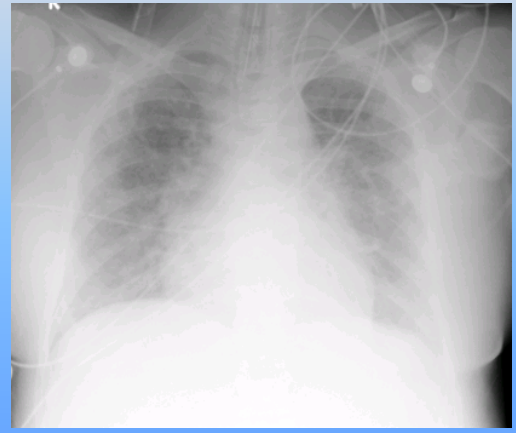


13/10 - LIP 23





18/10 - LIP 12



Patient was successfully extubated!

23rd October 2006

Conclusions (1)

The clinical interpretation of any monitored parameter

1. Necessitates a thorough understanding of the nature of the parameter itself.

2. Has to take into consideration all possible confounding factors.

Conclusions (2)

By monitoring a combination of parameters that include the CO, preload, fluid responsiveness and EVLW, the PiCCO monitor offers most of the information necessary for hemodynamic management.

The $ScvO_2$ and parameters of microcirculatory function offer additional important information.

What about outcome?

Unfortunately, it is difficult to separate between the effects of hemodynamic monitoring per se and the therapeutic philosophy, patient population, other therapeutic interventions and many other confounding factors.

The EBM police tends to target what is done, not what is not….

It is therefore much easier to do nothing and wait for the ultimate RCT….

The one that will tell us once and for all what to do at any circumstance at any time….

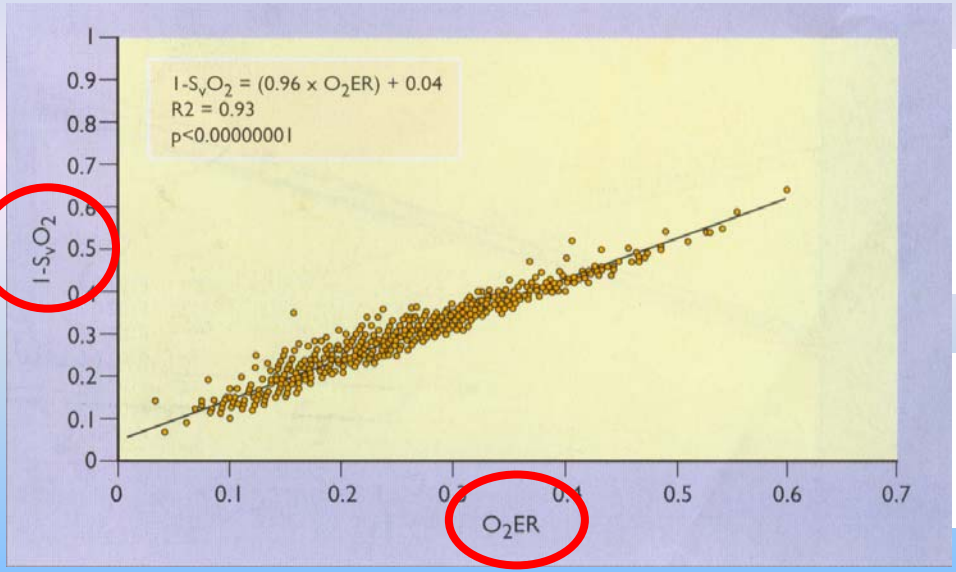
“The hottest places in hell are reserved for those who, in times of great crisis, do nothing”.

- Dante

Thank you!

Reliability of SvO_2 as an indicator of the oxygen extraction ratio (O_2ER) demonstrated by a large patient data set.

Keech J, Reed RL. J Trauma 2003 54:236-41



$$O_2ER = \frac{\dot{V}O_2}{\dot{D}O_2}$$

$$O_2ER \cong 1 - S_{\bar{v}}O_2$$

When oxygen extraction is low, $ScvO_2$ is high!!!

Identifying low cardiac output by itself is very important but not enough to guide therapy

What is the best way to measure cardiac output ?

Who Cares, Anyway ?

Caruso LJ et al, *Chest.* 2002;122:771-4

“Instead of asking which monitor is best for measuring CO, we might query why, in any event, CO is important to measure.”

Prediction of hemodynamics in ICU and ED patients by clinical evaluation alone is inaccurate and unreliable.

Clinical evaluation compared to pulmonary artery catheterization in the hemodynamic assessment of critically ill patients
Eisenberg PR et al, Crit Care Med 1984; 12: 349

Assessing hemodynamic status in critically ill patients: Do physicians use clinical information optimally?
Connors AF et al. J Crit Care 1987; 2: 174

Therapeutic impact of PAC in the ICU
Steingrub et al, Chest 1991; 99: 1451

PAC in critically ill patients: A prospective analysis of outcome changes associated with catheter-prompted changes in therapy
Mimoz O et al. Crit Care Med 1994; 22: 573

Hemodynamic and pulmonary fluid status in the trauma patient: are we slipping?
Veale WN Jr et al, Am Surg. 2005; 71: 621

Hemodynamic assessment in managing the critically ill: is physician confidence warranted?

Dawson NV et al. Med Decis Making 1993 ; 13: 258-66

Physicians were generally confident of their estimates, but there was no relation between confidence and accuracy.

Experienced physicians were no more accurate than less experienced ones, although they were significantly more confident.

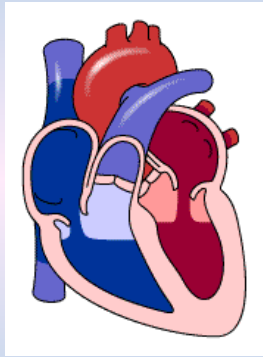
Physicians should not use their levels of confidence in their subjective estimates of cardiac function in deciding whether to base therapy on these estimates.

It seems that the many negative reports in recent years on the inadequacy of advanced methods of hemodynamic monitoring (mainly the PAC) have led to many instances of insufficient monitoring of critically ill patients.

I call this the
“Back to Nature” movement….

Heart Rate

PRELOAD



Afterload

Contractility

The PiCCClin Study - a multi-center European study

B. Change of therapeutic plan following advanced cardiopulmonary monitoring in critically ill patients.

In the absence of further hemodynamic information, what would be your therapeutic decision?

Possible therapeutic options:

Fluid loading	Red blood cells	Inotropic agent	Vaso-constrictor	Diuretic	Dialysis/ filtration	Other

Original therapeutic plan pursued

	Administered	Not administered
Fluids planned	109 (42.7%)	53 (20.8%)
Fluids not planned	28 (11.0%)	65 (25.5%)
Inotropes planned	33 (12.8%)	46 (17.9%)
Inotropes not planned	14 (5.4%)	164 (63.8%)
Vasoconstrictors planned	50 (19.6%)	36 (14.1%)
Vasoconstrictors not planned	24 (9.4%)	143 (56.9%)
Diuretics planned	19 (7.5%)	20 (7.9%)
Diuretics not planned	18 (7.1%)	197 (77.6%)

Original therapeutic plan changed

	Administered	Not administered
Fluids planned	109 (42.7%)	53 (20.8%)
Fluids not planned	28 (11.0%)	65 (25.5%)
Inotropes planned	33 (12.8%)	46 (17.9%)
Inotropes not planned	14 (5.4%)	164 (63.8%)
Vasoconstrictors planned	50 (19.6%)	36 (14.1%)
Vasoconstrictors not planned	24 (9.4%)	143 (56.9%)
Diuretics planned	19 (7.5%)	20 (7.9%)
Diuretics not planned	18 (7.1%)	197 (77.6%)

	Original therapeutic plan	
	Pursued	**Changed**
Fluids (n=255)	68.2%	31.8%
Inotropes (n=257)	76.6%	23.3%
Vasoconstrictors (n=255)	76.5%	23.5%
Diuretics (n=254)	85.0%	15.0%

The PiCClin Study

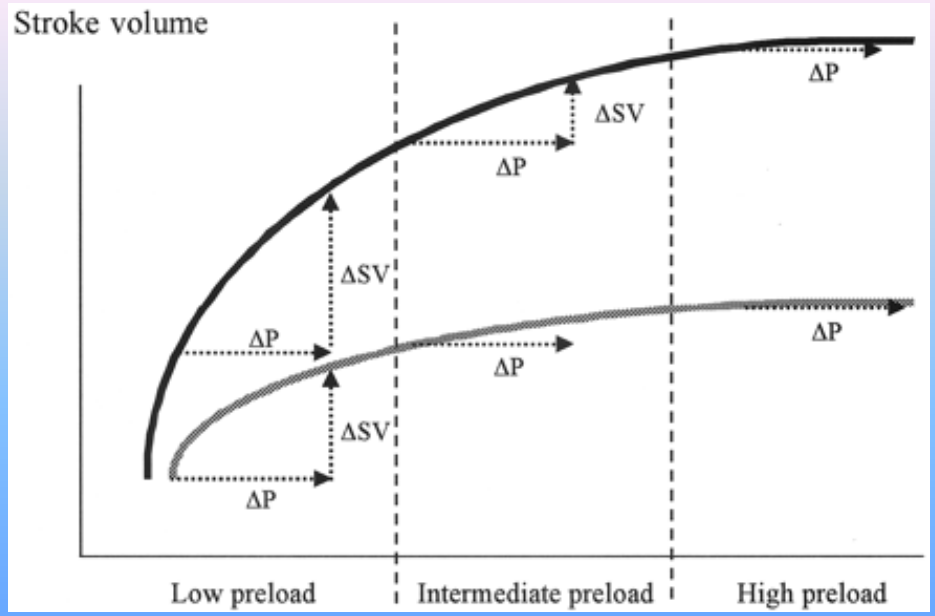
Conclusions (part II):

The measurement of advanced cardio-pulmonary parameters caused both specialists and residents to make considerable changes in therapeutic decisions that were made based on clinical judgment and conventional monitoring alone.

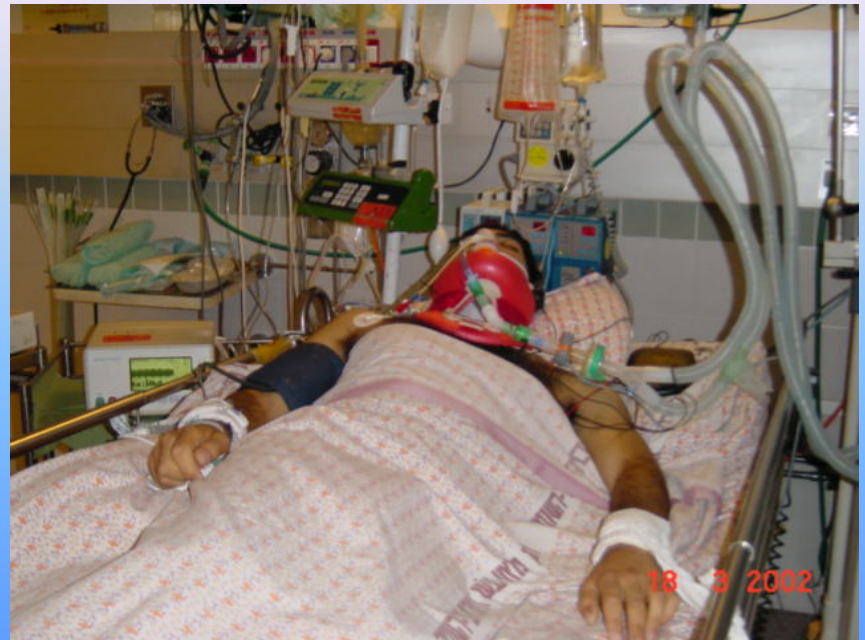
Practice parameters for hemodynamic support of sepsis in adult patients in sepsis

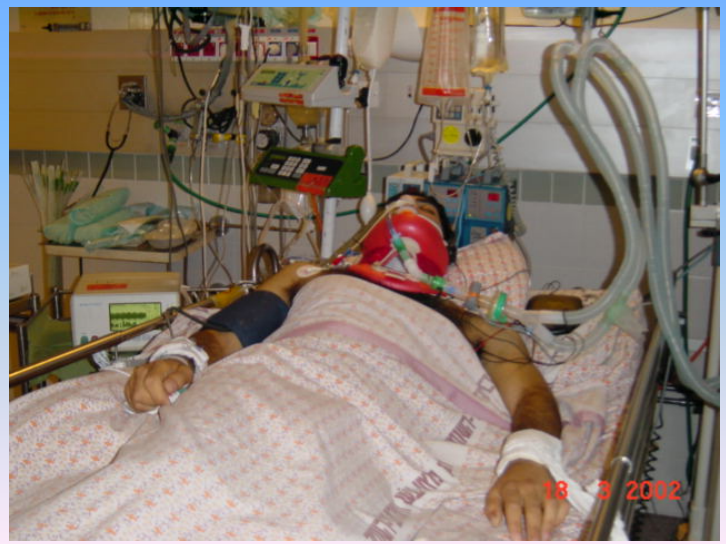
Task Force of the ACCCM and the SCCM, CCM 2004

"In most patients with septic shock, CO will be optimized at filling pressures between 12 and 15 mm Hg."



- **20 year old man after a motor vehicle accident.**
- **Neurological injury without improvement over the next 2-3 days.**





BP	70/40 mmHg
HR	155 bpm
CVP	5 cmH_2O
PaO_2/FiO_2	80 (PEEP 16)

Would you give fluids to this patient?

Aggressive diuresis was started!

- **CO = 12-15 L/min** — High !!!
- **SVR = 400-500** — Low !!!
- **ITBVI = 1200 ml/m² (800-1000)** — High !!!
- **EVLW = 19-23 ml/kg (4-7)** — High !!!

Intensive Care Med
DOI 10.1007/s00134-007-0531-4

INTERNATIONAL CONSENSUS CONFERENCE

3. In the absence of hypotension, when shock is suggested *by history and physical examination,* we recommend that a marker of inadequate perfusion be measured (decreased ScvO2, SvO2, increased blood lactate, increased base deficit, perfusion-related low pH).
Level 1; QoE moderate (B)

Early goal-directed therapy

16. We recommend instituting goal-directed therapy without delay, in patients presenting with septic shock, particularly where ScvO2 is below 70%.
Level 1; QoE moderate (B)

Surviving Sepsis Campaign Guidelines (Rivers, NEJM 2001)

Initial resuscitation

Begin resuscitation immediately in patients with hypotension or elevated serum lactate.

Resuscitation goals:

- *Central venous pressure: 8-12 mm Hg*
- *Mean arterial pressure ≥ 65 mm Hg*
- *Urine output ≥ 0.5 mL.kg-1.hr-1*
- *Central venous or mixed venous oxygen saturation ≥ 70%*

If central venous oxygen saturation or mixed venous oxygen saturation of 70% is not achieved with a central venous pressure of 8-12 mm Hg, then transfuse packed red blood cells to achieve a haematocrit of ≥ 30% and/or administer a dobutamine infusion of up to a maximum of 20 µg.kg-1.min-1.

A 40 year old woman with a large pancreatic tumor.
Two days following a long Whipple operation, the patient complains of shortness of breath, with SaO2 of 85% with an O_2 mask.

Vital signs:
HR 130 bpm, BP 140/65 mmHg, CVP 15 cmH_2O,
Urine output adequate.

Patient is ventilated with PCV, FiO_2 80%,
PEEP 14 cmH_2O, SaO_2 98%.

Mixed venous oxygen saturation in critically ill septic shock patients. The role of defined events

Krafft P et al, Chest 1993; 103:900-6

The SvO_2 of septic shock patients is mainly normal or even supra-normal.

$$\dot{O}_2ER \cong 1 - S_{\bar{v}}O_2$$

Fluid resuscitation in severe sepsis and septic shock: An evidence-based review

Jean-Louis Vincent, MD, PhD, FCCM; Herwig Gerlach, MD, PhD

(Crit Care Med 2004; 32[Suppl.]:S451–S454)

- **The normal SvO2 is 70-75% in critically ill patients, but it can be elevated in septic patients due to maldistribution of blood flow.**

- **Therefore, a normal or high SvO2 does not necessarily indicate adequate tissue oxygenation.**

A 31 year old patient with Down's syndrome is admitted to the ICU with severe pneumonia.

Stable hemodynamics with $ScvO_2$ 72 % and normal ECHO.

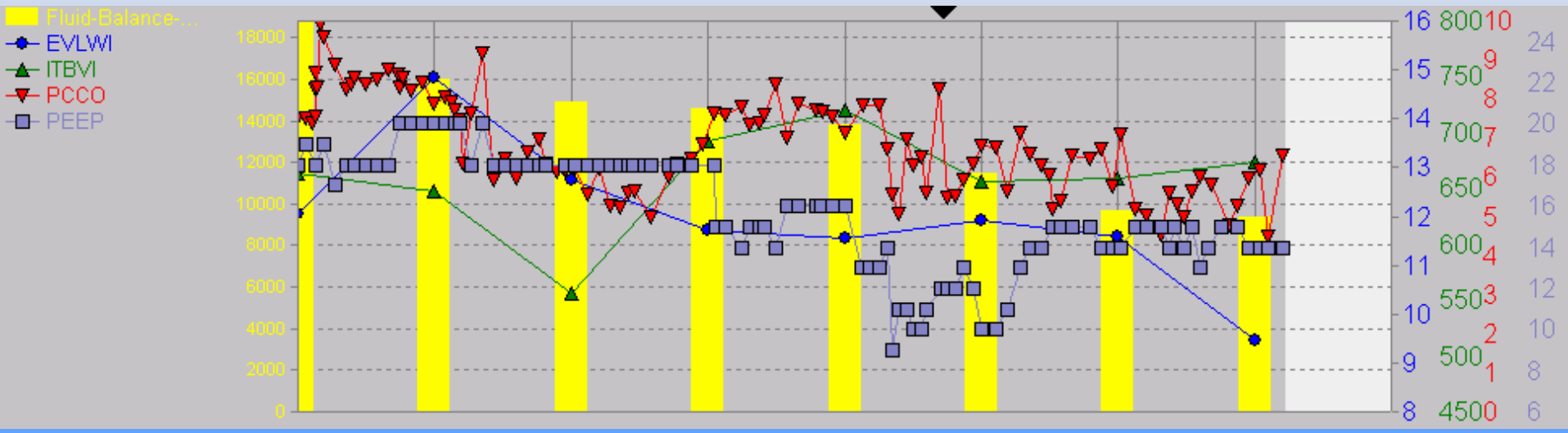
Noradrenaline 0.02 mcg/kg/min due to SBP < 100.

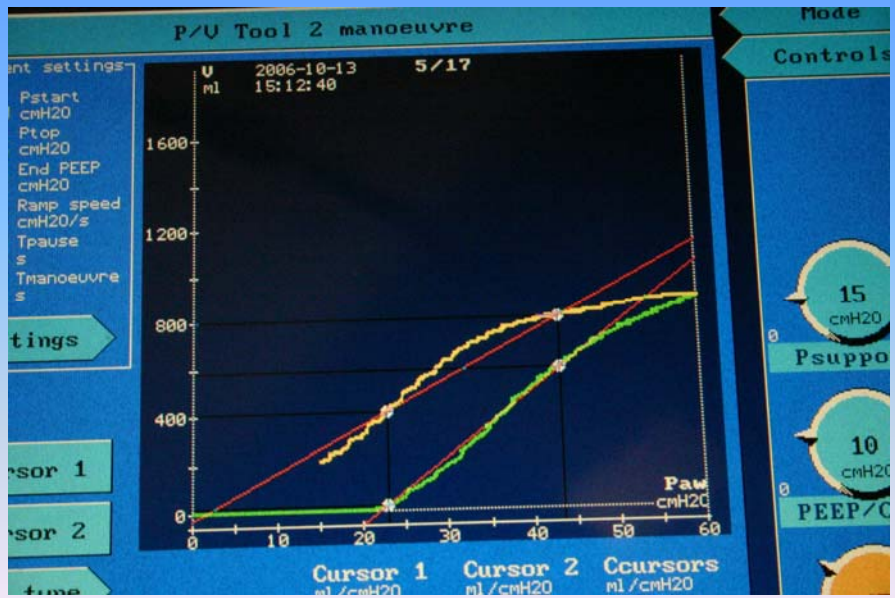
Positive fluid balance 12 L at Day 5 and 19 L at Day 7.

High EVLW, PEEP of 20 to maintain oxygenation.

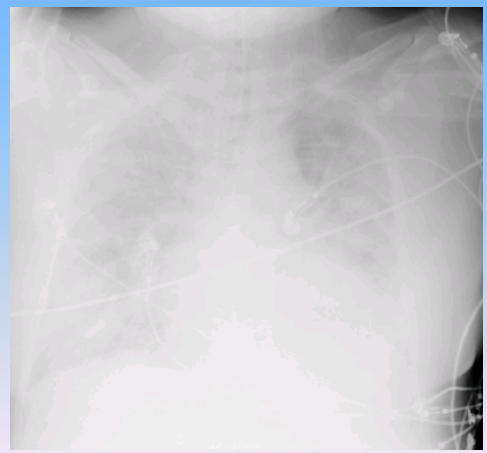
Even though the patient had low ITBV, aggressive diuresis was started with daily negative fluid balance of 1-2.5 L.

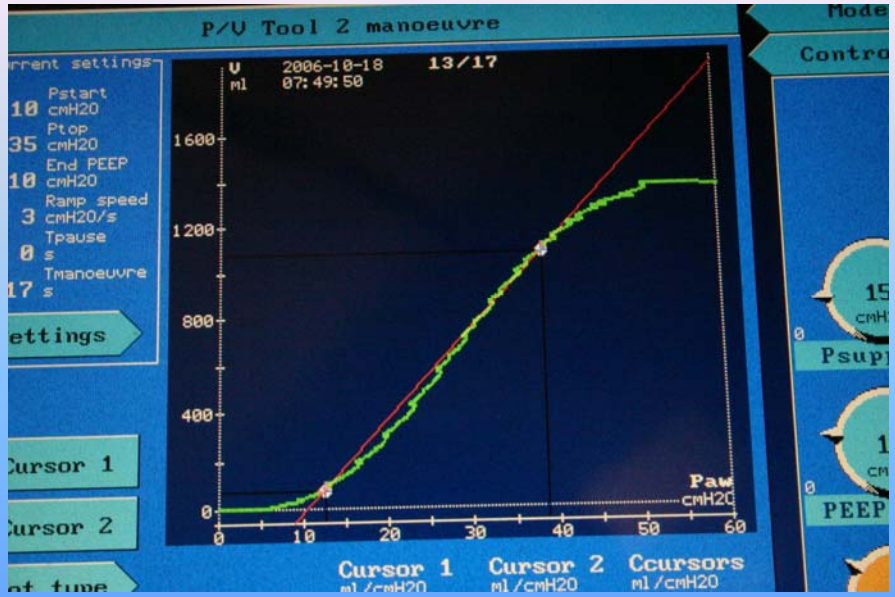
CO supported with dobutamine and later dopamine (due to bradycardia).





13/10 - LIP 23





18/10 - LIP 12

Sepsis

Most patients with severe sepsis exhibit a significant degree of myocardial depression, which is characterized by reversible flattening of the Frank-Starling curve, reduced inotropic responsiveness to catecholamines, and biventricular dilation and depression of the ejection fraction (EF).

Kumar A, et al Chest 2004;126;860

Nevertheless, many septic patients benefit from the administration of large fluid loads.

Hemorrhage

Rapid correction of hypovolemia is a key element in the treatment of hemorrhagic shock.

Nevertheless, correction of experimental hemorrhagic shock by retransfusion is associated with a global deterioration in LV function and a sharp decrease in LVEF. This phenomenon may be due to elevation of afterload and/or myocardial depression. *Preisman S, et al. BJA 2002;88:716*

Preliminary statements

Co-morbidities often complicate the hemodynamic status.

Critically ill patients often present us with therapeutic conflicts (e.g., hemodynamic instability and ARDS).

A man with fever and shortness of breath

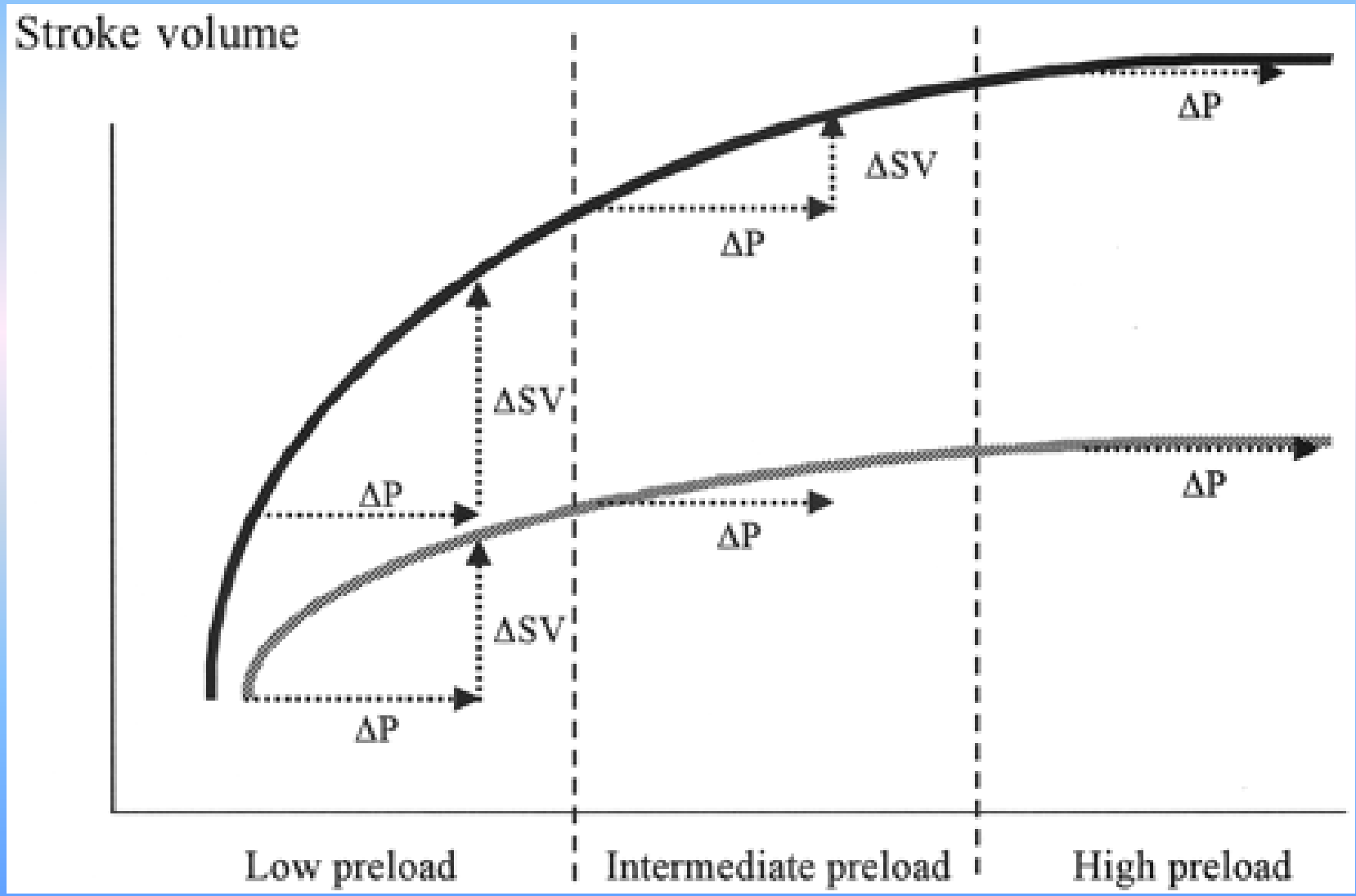
Lactate 48 (moderately elevated)

$ScvO_2$ 72%

CVP 9 mmHg

- **CO** **3.8**
- **ITBVI** **950**
- **EVLWI** **15**
- **SVR** **1100**

Static parameters of preload cannot accurately predict fluid responsiveness



Hemodynamic response to fluid loading

	Patients	Definition of Responders	N	Challenge	Responders
Preisman S (2005)	Cardiac surgery	> 15% SV	18	250 mL colloids	32/70 VLS (46%)
Hofer CK (2005)	Cardiac surgery	> 25% SVI	35	10 mL/kg (IBW) 6% HES	21 (60%)
Swensen CH (2006)	Abdominal surgery	Increase in CO	10	25 mL/kg of Ringer	4 (40%)
Tavernier B (1998)	Sepsis w. circulatory failure	> 15% SVI	15	500-1000 mL HES	21/35 VLS (60%)
Michard F (2000)	Sepsis w. circulatory failure	> 15% CI	40	500 mL HES	16 (40%)
Michard F (2003)	Septic shock	> 15% SVI	27	500 mL HES	32/66 VLS (48%)
Feissel M (2005)	Septic shock	> 15% CI	20	8 ml/kg HES	13/22 VLS (59%)
Monnet X (2005)	Critically ill w. circulatory failure	> 15% increase in ABF (Doppler)	38	500 ml NS	20 (53%)
Vallee F (2005)	Critically ill w. circulatory failure	> 10% increase in SVI	51	4 ml/kg colloid X 2	20 (39%)
Heenan S (2006)	Critically ill w. circulatory failure	> 15% in CO	21	1 L Ringer or 500 mL HES	9 (43%)
Lafanechère A (2006)	Critically ill w. circulatory failure	> 15% increase in ABF (Doppler)	22	PLR and 500 ml NS	10 (45%)
Osman D (2007)	Sepsis	> 15% in CO	96	500 mL HES	65/150 VLS (43%)