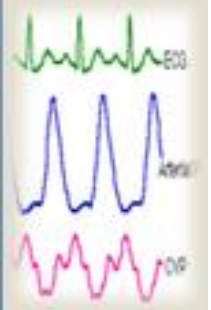
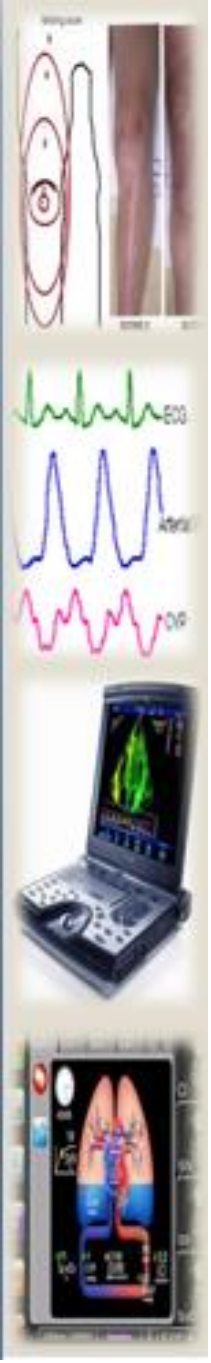
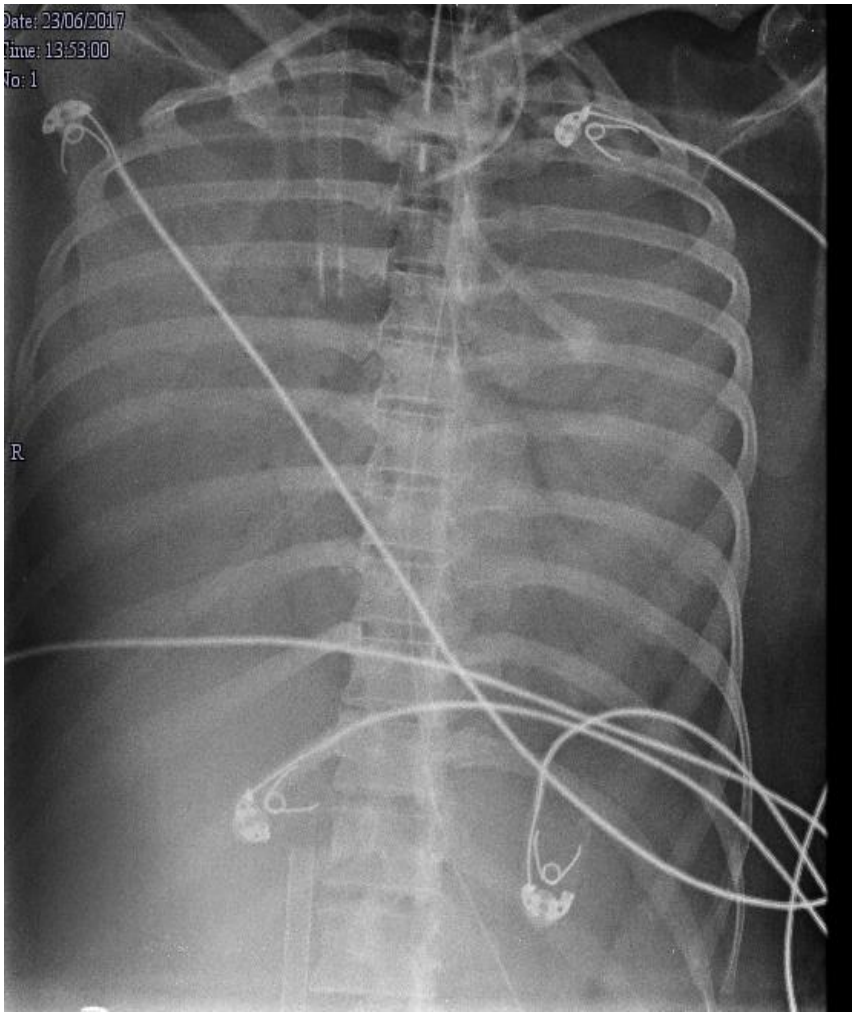
Dr Sameer Jog Consultant Intensivist,
Deenanath Mangeshkar Hospital, Pune
MD (Int Med) EDIC IDCCM

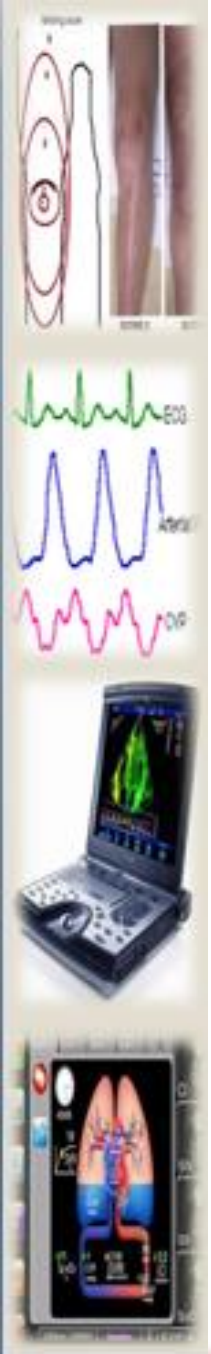


How I give fluids ? - Obvious !!

How I give fluids ?Not so obvious
Nobody is correct !!







ESICM
EUROPEAN SOCIETY OF INTENSIVE CARE MEDICINE
The Intensive Connection

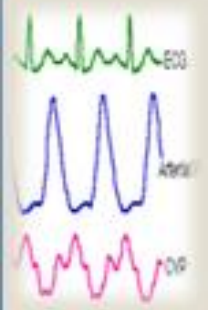
FENICE trial

FLUID CHALLENGES IN INTENSIVE CARE

How do we administer fluids in the ICU?

One week in 2013

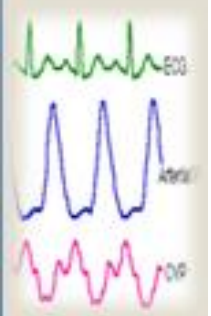
Multicentre observational study conducted by the ESICM Trials group.



FENICE data (ICM 2015)

2213 patients 46 countries, 311 centers 400 Indian patients

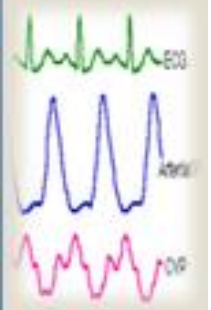
	Indication of fluid challenge	Percentage
1	Hypotension	58.7
2	Oliguria	18
3	Weaning vasopressor	7.1
4	Lactate	6.2
5	Skin mottling	1.7
6	Cardiac Output	3
7	SVO2 / ScvO2	0.5

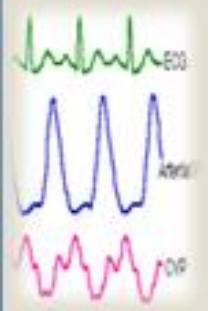
Don’t be surprised
FENICE data (ICM 2015)

2213 patients ,46 countries, 311 centers 400 Indian patients

	Hemodynamic Variable used to predict fluid responsiveness	Percentage
1	**No variable used**	**42**
2	Static Variable (CVP, PAOP ,GEDV, other)	35
3	Dynamic varibale used PPV , SVV,PLR,Echo	21.9
	Echo	2%



Fluid therapy = Toss a coin

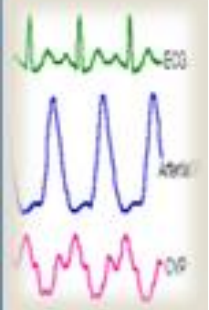
Shock Redefined

Shock is defined as circulatory and cellular dysfunction, manifested by markers of hypoperfusion (clinical or biochemical) with or without hypotension.

(Consensus Statement 2007, ICM)

Normotensive or Cryptic Shock

Shock with Hypertension-



Tools I shall Discuss

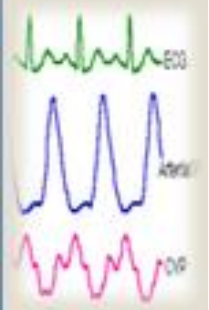
1) Clinical Assessment

2) Lactates and $ScvO_2$

3) CVP & FC and Art line

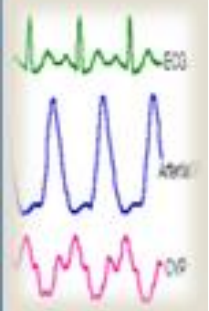
4) Trans-thoracic Echocardiography

5)Functional Hemodynamic monitoring concepts and gadgets



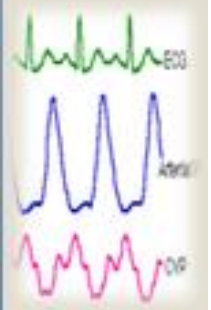
CASE 1

- Young male presented with fever, chills & rigors for 2 days
- No co-morbidities.
- Gametocytes of P.falciparum on peripheral smear, P.I.- 10%.
- BP 98/56mm (70mm), urine output < 0.5cc/kg/hr for last 6 hours
- O_2 sats – 94% on air. Art Lact 3.4 mmol/lit
- Hb 8 gm% and Platelets 125 $X10^3$
- Other Lab normal



Tools I shall Discuss

1) Clinical Assessment
2) Arterial Lactates and $ScvO_2$
3) CVP
4) Intra-arterial Pressure monitoring
5) Trans-thorasic Echocardiography
6) PA catheter
7) Functional Hemodynamic monitoring concepts and gadgets



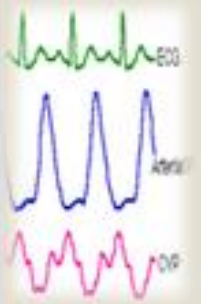
Clinical History is *the* Key

Hypovolemic

Distributive

Pump failure

Obstructive



Skin Mottling

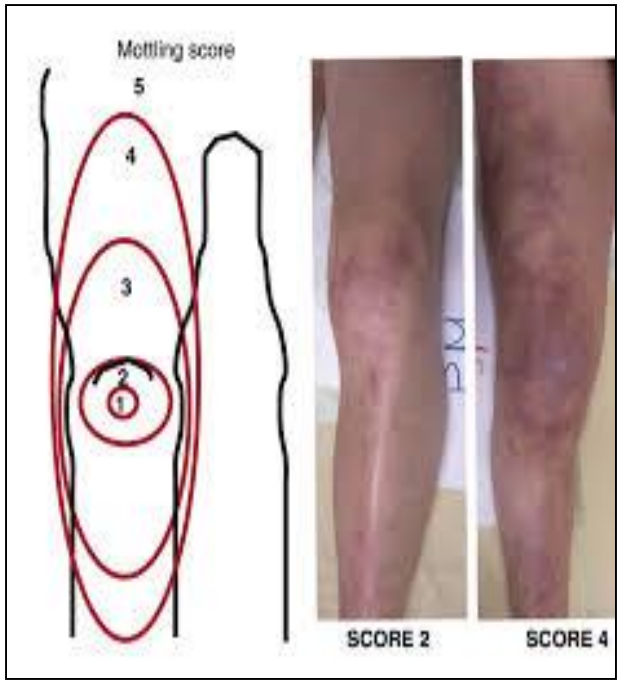
Easy ,reliable sign

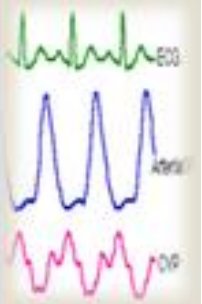
Score 1-5

Septic shock: vasopressors

14 day Mortality prediction

H. Ait-Oufella et al Intensive Care Med 2011





Capillary Refill Time

Quick test for hypoperfusion

Measurable response

Prognostic / Predictor value

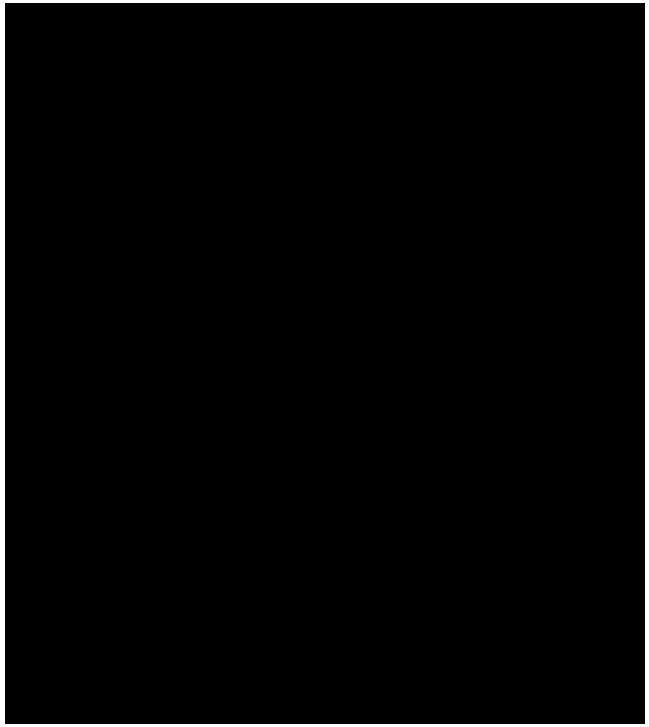
Int Care Med 2014

Limitations

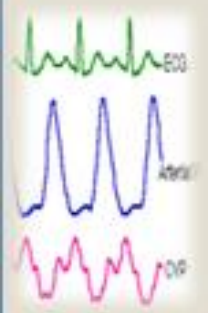
< 2 s in young individuals

Up to 4.5s in the elderly

Healthy Nurse



Patient in shock



Shock Index

HR / Systolic Pressure 80/ 120 = 0.66 {0.5 to 0.7}

Linear inverse co-relation with CO and SV

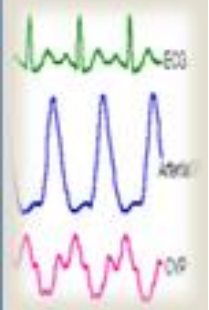
Validation in trauma

S.I. > 1 -- high mortality 140/92 = 1.5 {0.5 to 0.7}

Process study- S.I . guided resuscitation = EGDT

N Engl J Med 2014; 370:1683-1693

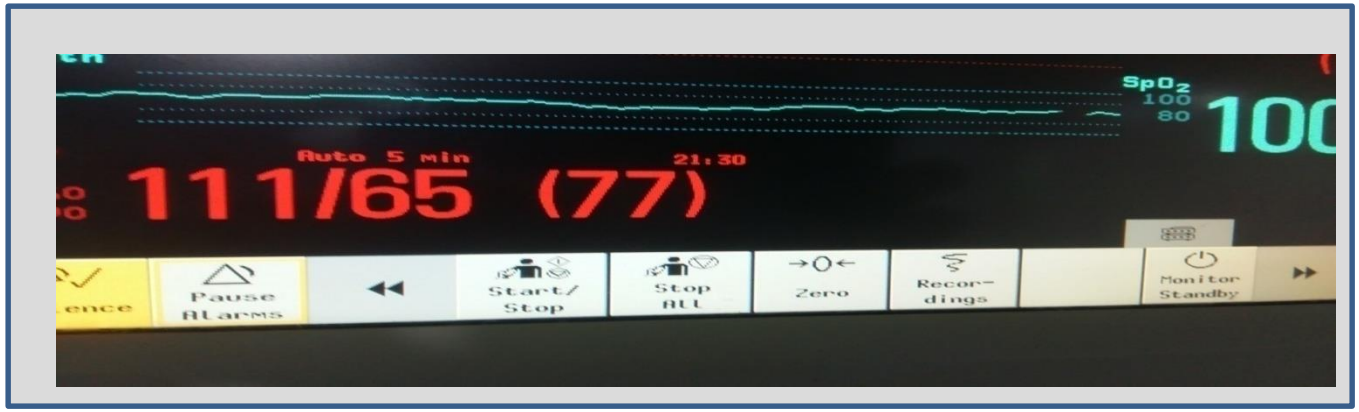
Septic Shock

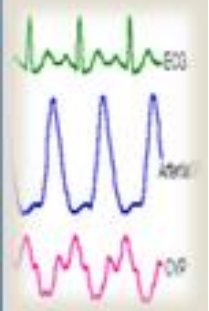


Watch the Pleth !!

Cardiogenic

Septic





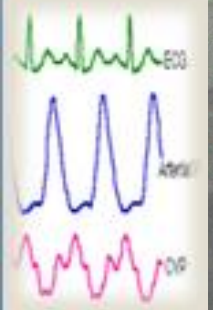
Urine Output

0.5 ml / kg / hour

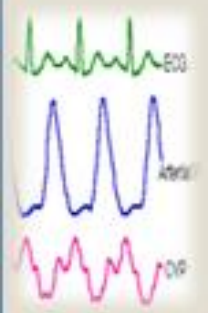
Response to fluid bolus

Common trigger for fluids

Limitations e.g RRT ptient

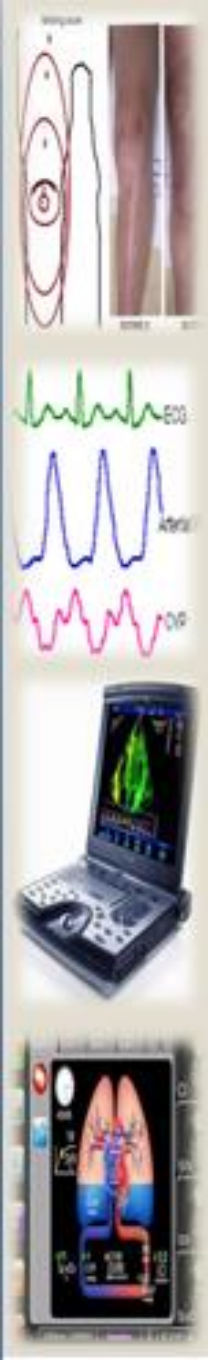


Best Catheter in ICU



Lactate levels

- Rapid and Reliable.
- Lactates- 1 to 1.5 mmol/lit
- Strong experimental and clinical association with tissue hypoperfusion.
- Increased blood lactate levels and their failure to normalize have been associated with increased morbidity and mortality.

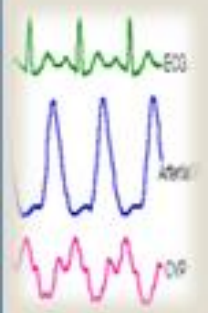


Basics
about Lactate

- Sustained hyperlactatemia means a large increase in mortality, regardless of status with respect to shock or hypotension.

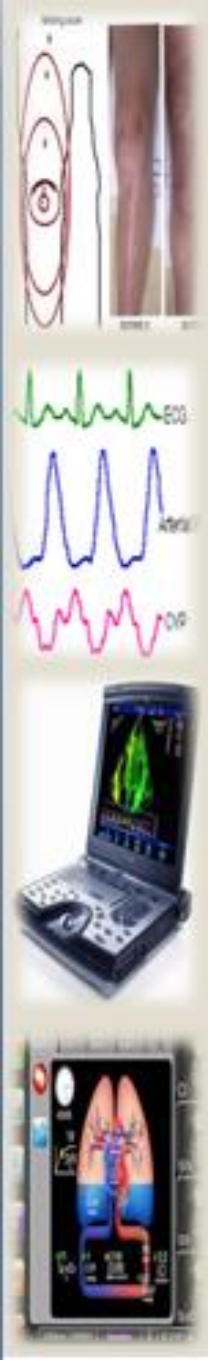
- Dose response relationship between lactate levels and mortality: the higher the level, the greater the risk of death.

- Lactate can be measured in arterial or venous blood, since the values are virtually interchangeable.

Hyperlactetemia ≠ Septic shock

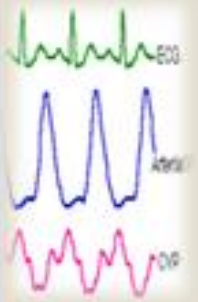
No.	Cause	Remark
1	Cardiogenic shock/ ADHF	Reduced Cardiac output. Reduced DO_2
2	Hemorrhagic shock	Drop in Hb Reduced DO_2
3	Septic shock	Cytopathy anaerobic metabolism, Reduced CO (initial) Endogenous Epinephrine β stimulation
4	Severe Hypoxia	PO_2 < 30 m Hg
5	Severe Anemia	Hb < 5 gm%
6	Seizure, shivering	High Oxygen consumption by muscles
7	Liver Disease	Poor clearance
8	Methanol, ethylene glycol, Metformin nRTI, Propofol	Interference with oxidative phosphorylation
9	Salbutamol	β stimulation Aerobic Glycolysis



How I give fluids –guided by Lactates

- High lactates means tissue hypoperfusion
- Think of fluids first **DESPITE**

CVP is high, edema present, B/L scattered crepts, LVEF is 40 %, creat is 3.1



The NEW ENGLAND JOURNAL of MEDICINE

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ORIGINAL ARTICLE

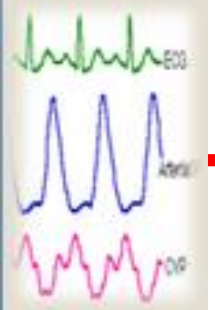
Early Goal-Directed Therapy in the Treatment of Severe Sepsis and Septic Shock

Emanuel Rivers, M.D., M.P.H., Bryant Nguyen, M.D., Suzanne Havstad, M.A., Julie Ressler, B.S., Alexandria Muzzin, B.S., Bernhard Knoblich, M.D., Edward Peterson, Ph.D., and Michael Tomlanovich, M.D. for the Early Goal-Directed Therapy Collaborative Group

N Engl J Med 2001; 345:1368-1377 | November 8, 2001

Fluids	**Vasopressors**	**Dobutamine**
Oxygen	**Mech Ventilation**	**RBC**

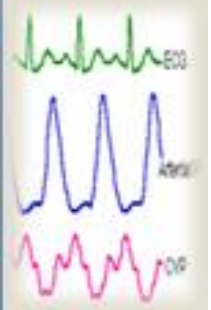
Target of $ScvO_2$ > 70 %

Mortality

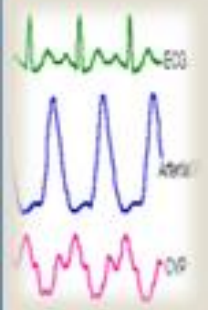
Rivers 2001- EGDT paper

	Control	EGDT	P-value
In-hospital	46.5	30.5	0.009
28-day Mortality	49.2	33.3	0.01
60-day Mortality	56.9	44.3	0.03

$ScvO_2$ Limitations

- Regional tissue hypoxia despite normal $ScvO_2$- Bowel gangrene
- Spurious low - Chronic heart failure
- Pseudonormalisation with rise in CO (Sepsis C.O. 10 lit)
- Falsely Elevated in VERY SICK shocks
  - microvascular shunting (ArterioloVenular shunting)
  - decreased cellular utilisation (apoptosis)

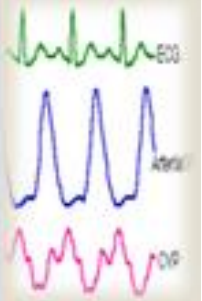


How I give Fluids – guided by ScvO2?

- In Septic(distributive) shock- if ScvO2 is low < 65 %

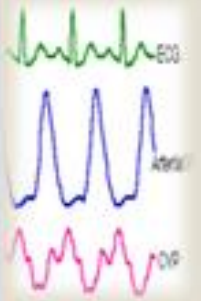
  Always consider fluids to improve tissue perfusion.

If ScvO2 is normal or high (≥ 70 %) , I cannot decide about fluids, will use other parameters.



CASE 2

- Elderly female, diabetic & hypertensive.
- Had burning micturation & fever with chills.
- Treated by a G.P, then better for 2 days
- Admitted with hypotension MAP 58 mm
- Had decreased urine output(700 cc in 24 hrs). Lact 1.3 mmol
- Became drowsy & developed a puffy face.



CASE 2

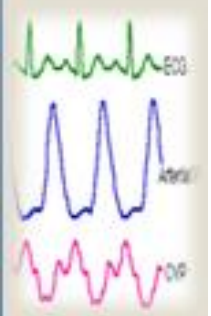
Concerns are

1)Fluid overload and pulmonary oedema.

2)Septic shock – hypovolemia

3)Renal Impairment

How to balance all these?

Tools I shall Discuss

1) Clinical Assessment
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3) CVP and FC
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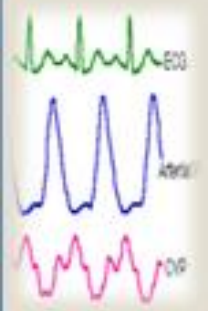


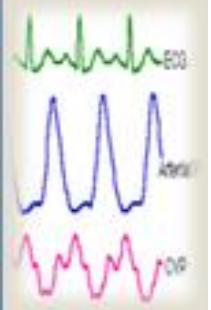
About CVP

Always Transduce for reliable continuous data.

Affected by MV , PEEP, IAP, RA/RV compliance and many more

Low CVP < 8 to10mm usually***(?)*** suggests hypovolemia (exceptions-anaphylaxis,AN Pancreatitis,DHS,Severe Septic shock)

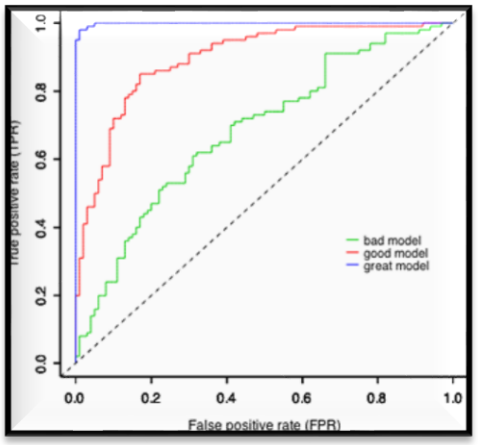
Normal or high CVP doesn't mean fluid status is adequate or overloaded.



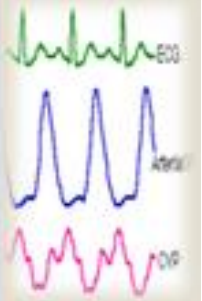
Assessing fluid responsiveness
ROC – Sensitivity & Specificity

Static pressure and volume parameters

(ROC ~0.5–0.6)



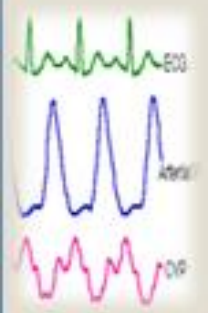
1. CVP
2. PAOP
3. IVC/SVC diameter
4. Right ventricular end-diastolic volume
5. Left ventricular end-diastolic volume
6. SVC/IVC variation during mechanical ventilation



Does the Central Venous Pressure Predict Fluid Responsiveness? An Updated Meta-Analysis and a Plea for Some Common Sense*

Conclusions: There are no data to support the widespread practice of using central venous pressure to guide fluid therapy. This approach to fluid resuscitation should be abandoned.

Paul Marik and Cavallazzi, Rodrigo Critical Care Medicine: July 2013

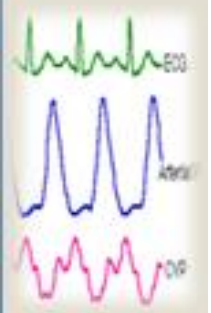
Myths about fluid challenge

1. *Fluid administration should be withheld because the central venous pressure is high*

2. *Fluid administration should be withheld because there is evidence of lung edema on the chest roentgenogram.*

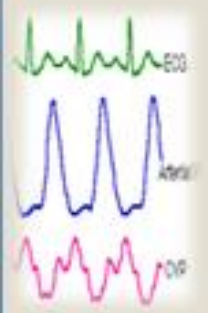
3. *Fluid administration should be withheld because the patient has already received a large volume in a short time interval.*

4. *I gave fluids to increase the central venous pressure to 12 mm Hg to exclude an underlying hypovolemia.*



Fluid challenge 35 years back!
Weil and Henning

- 250-500 ml fluid over 10 minutes
- Rise of 2-5 mm Hg rule for CVP and 3-7 rule for PAOP
- < 2 for CVP and <3 for PAOP – Continue
- 2-5 for CVP and 3-7 for PAOP- caution, reassess and go ahead
- > 5 for CVP and > 7 for PAOP Don’t

Downside of Fluid challenge

- Prognostic value of extravascular lung water in critically ill patients.

Chest 2002

- Sepsis in European Intensive Care Units: SOAP

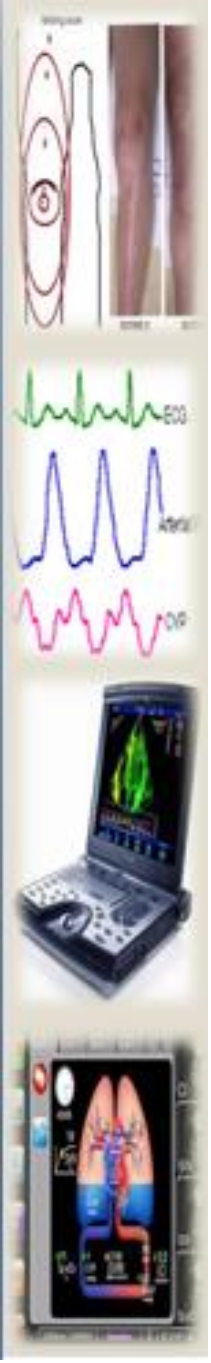
Crit Care Med 2006

- Comparison of Two Fluid-Management Strategies in Acute Lung Injury

N Eng J Med 2006

- Extra-vascular lung water is an independent prognostic factor in patients with acute respiratory distress syndrome.

Crit Care Med 2013



ORIGINAL

Fluid administration in severe sepsis and septic shock, patterns and outcomes an analysis

Paul E. Ma

May 2017

• 23,513
and sep

•Hospita
was 25.8

•Day 1 flu
No sh
Shock

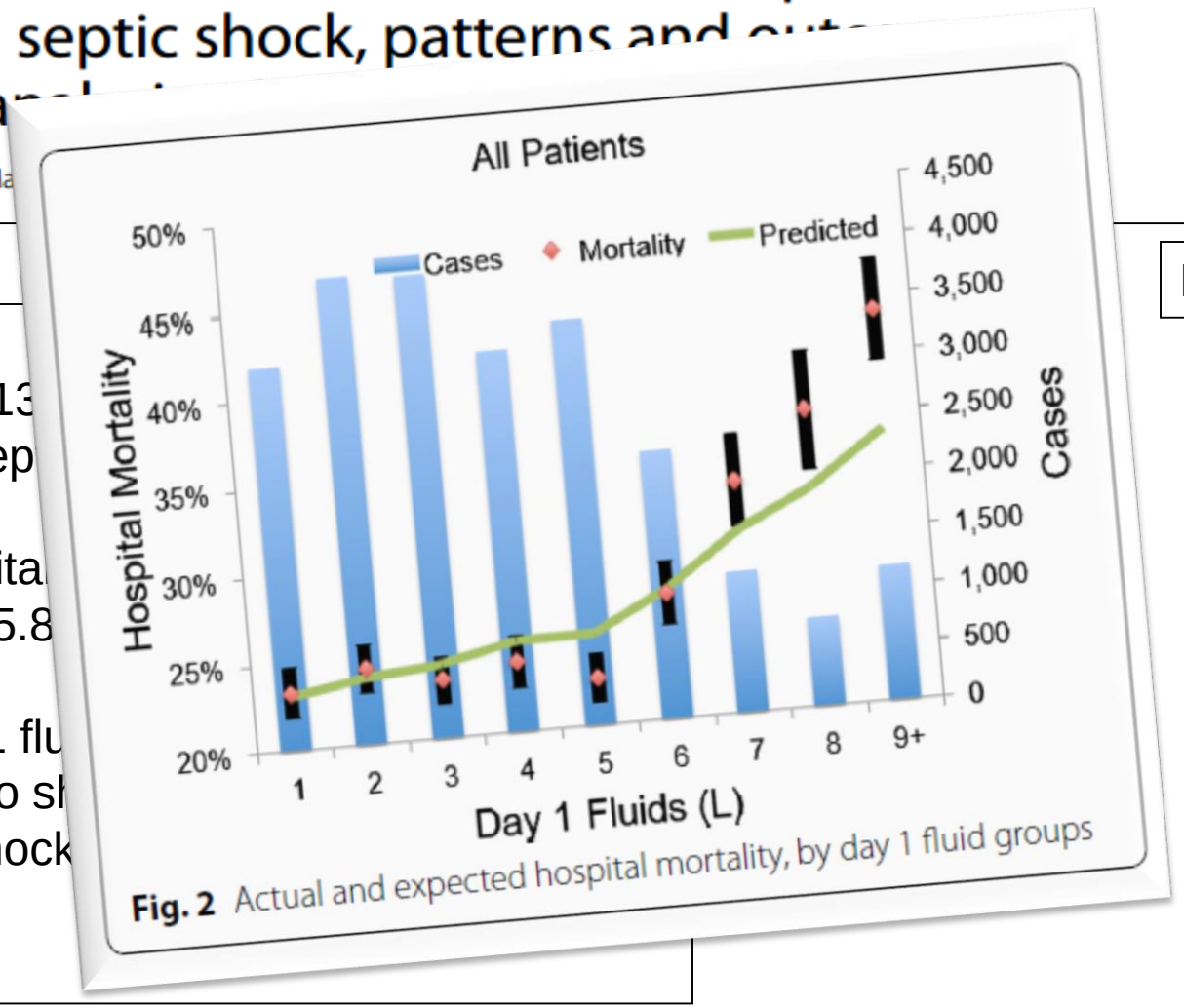
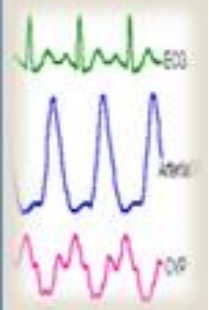


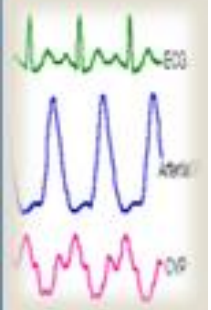
Fig. 2 Actual and expected hospital mortality, by day 1 fluid groups



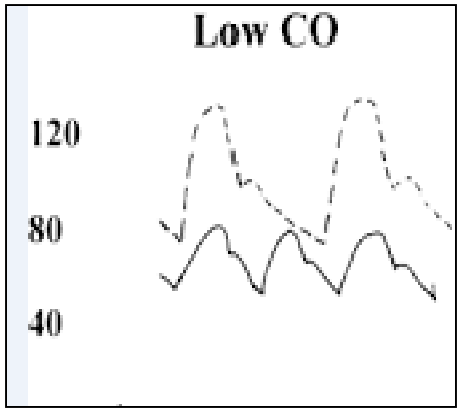
Arterial Line

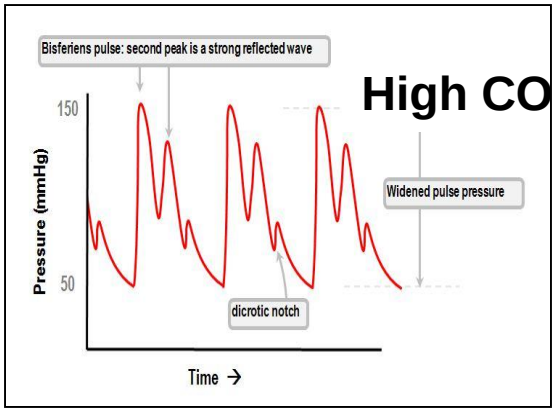
- “Gold Standard” for pressure monitoring.
- Especially in shock state.
- Proper installation and zeroing is must.
- Newer advanced hemodynamic algorithms are based on arterial line

e.g. Flotrac-Vigileo, PiCCO,LiDCO,PRAM



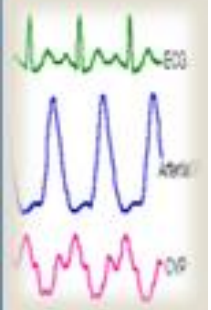
Pulse Pressure





- SBP -80, DBP 55, PP 25 mm
- Low SV/ CO
- Classic in cardiogenic shock
- Fluid challenge ?cautious

- SBP 150, DBP 55, PP 95 mm
- High SV/ CO
- Classic in distributive shock
- Fluid challenge



Low BP ≠ shock

- MAP
  shoul
  e.gPE

- Hype
  shoul
  e

Charalampos Pierrakos
Dimitrios Velissaris
Sabino Scolletta
Sarah Heenen
Daniel De Backer
Jean-Louis Vincent

Can changes in arterial pressure be used to detect changes in cardiac index during fluid challenge in patients with septic shock?

Intensive Care Med (2012)

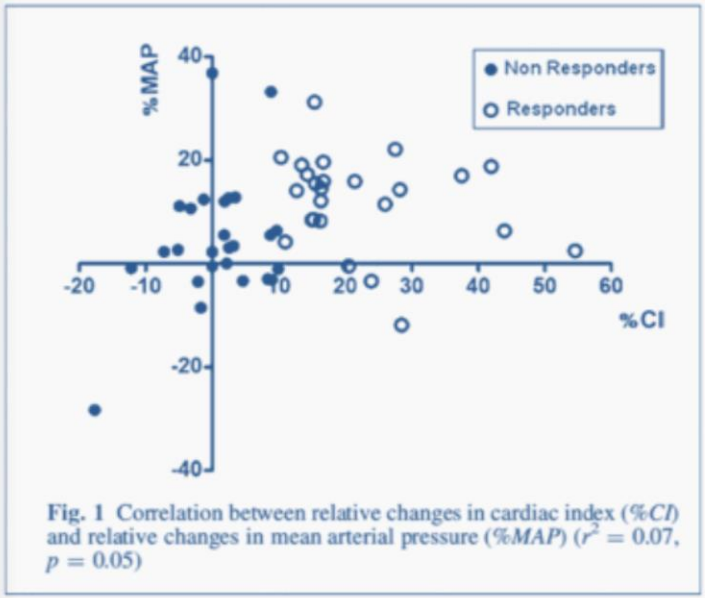
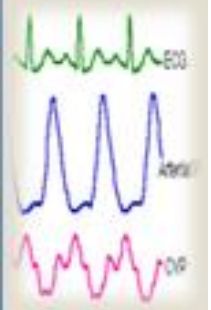


Fig. 1 Correlation between relative changes in cardiac index (*%CI*) and relative changes in mean arterial pressure (*%MAP*) ($r^2 = 0.07$, $p = 0.05$)

- Very weak correlation MAP and CI
- Sometimes changes in MAP without changes in CI

Target BP

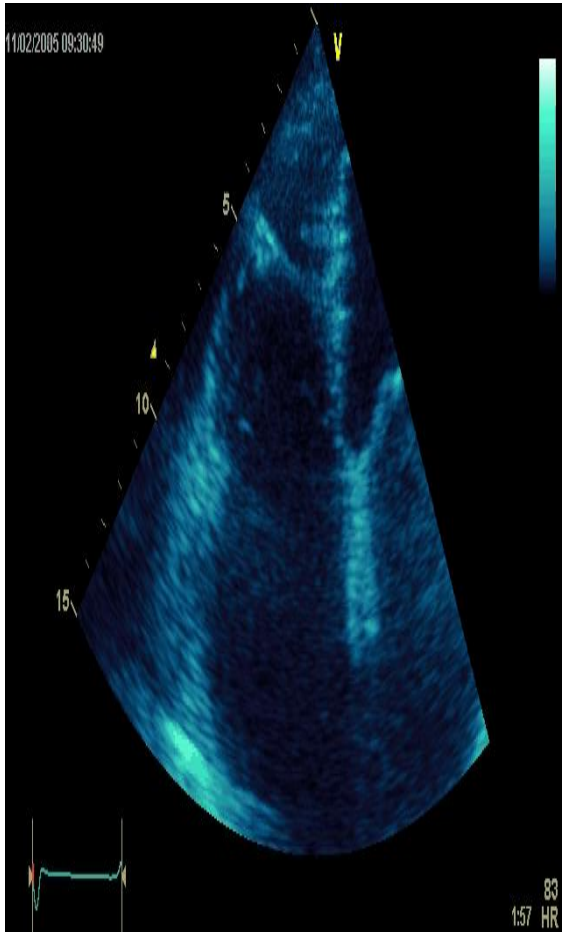
- No universal magic number
- Individualised target- CRT,urine output, Art lactate, sensorium
- MAP 65 mm is as arbitrary as CO of 5 lit
- SEPSISPAM study

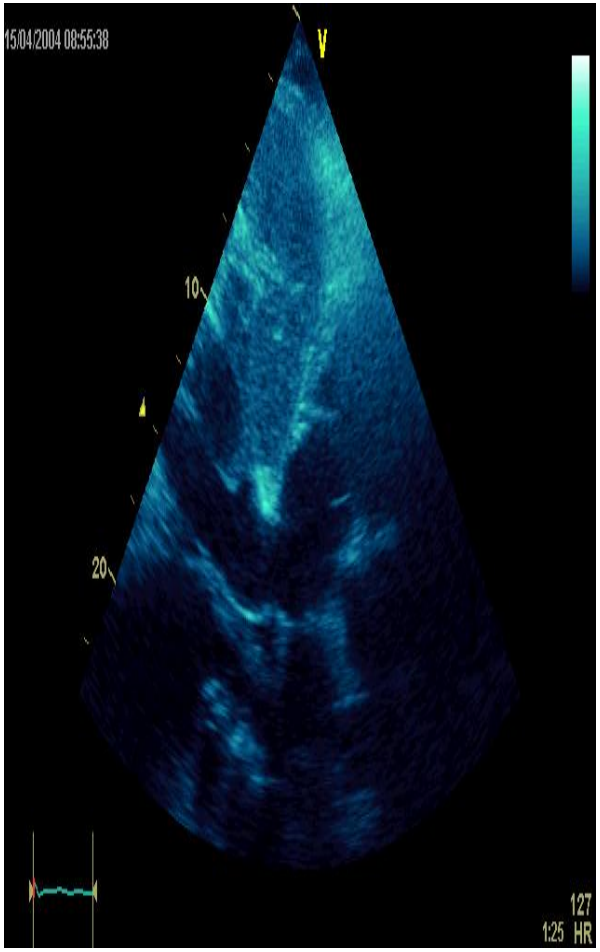
  80-85 Versus 65 -70 mm

NEJM 2014

Less RRT in HT

2017 - Echocardiography is the new stethoscope !





Echocardiography

Predominent right ventricular failure

- **RV ischaemia**
- **Pulmonary hypertension**

Global heart failure

- **TAMPONADE**
- Vegetations

Predominent left ventricular failure

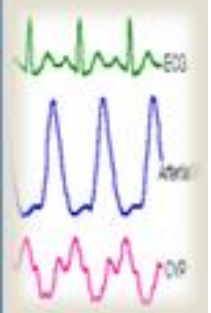
- **LV dysfunction**
- Massive Mitral regurg

Mebazaa et al. Intensive Care Med, 2004;30:185-96

Functional Echocardiography

Rough estimate of C.O.

Fluid Responsiveness – IVC collapse, Aortic Flow variation



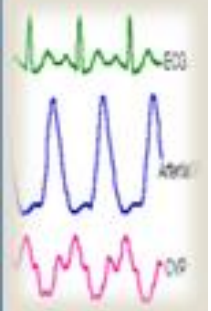
Fluids in cardiogenic shock ?

The clinical definition of cardiogenic shock is decreased cardiac output and evidence of tissue hypoxia in the **presence of adequate intravascular volume.**

Even patients with cardiogenic shock may benefit from fluids

Vincent, DeBacker NEJM 2013

May be judicious bolus of 200-300ml , clinical response– HR , BP, Urine ,pulse pressure Pulm edema



Fluids in Obstructive shock ?

European Heart Journal Advance Access published August 29, 2014

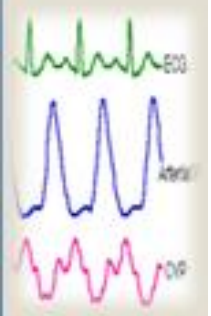
European Heart Journal
doi:10.1093/eurheartj/ehu283

ESC GUIDELINES

2014 ESC Guidelines on the diagnosis and management of acute pulmonary embolism

Aggressive volume therapy – may worsen RV function

Modest (500 mL) fluid challenge may help to increase cardiac index in patients with PE, low cardiac index, and normal BP

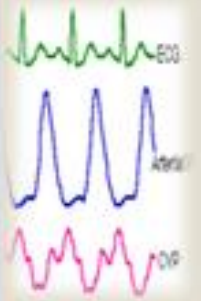


CASE- 3

- 60 yr male, diabetic, hypertensive, IHD with CABG 8 yrs ago.
- 2 admissions for LVF in past, on optimal anti-failure treatment.

- c/o cough, fever, breathlessness since 3 days.
- Admitted in tachypnea, desaturation(83% on NRBM) & hypotension & decreased urine output in last 12 hrs.

- Troponin I raised, Pro BNP raised, Procalcitonin 5ng/ ml, TLC 16000/ ccm, Creat 2.1, Lact 3.2

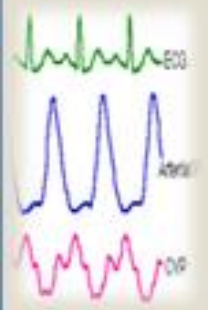


CASE- 3

1) Shock→ Cardiogenic AND / OR Septic?

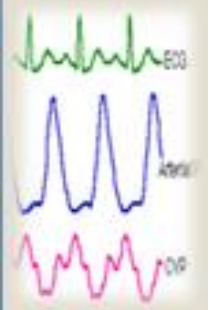
2)Hypoxic state→LVF AND / OR Acute lung injury?

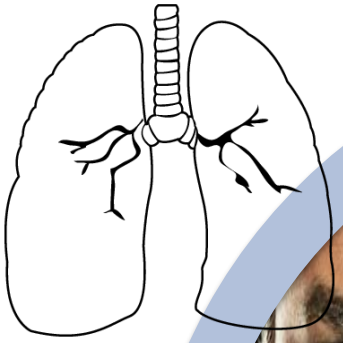
3)Acute renal impairment→ Septic MODS AND /OR hypoperfusional?

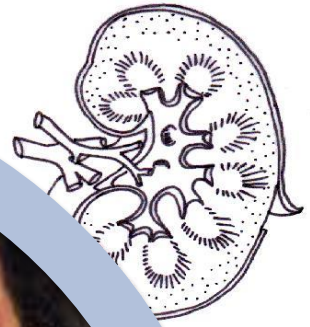



CASE- 3 Therapeutic Dilemma

1) Liberal Fluids - Pulmonary edema
2)No Fluids - Shock and AKI worsening
3)Vasopressors - Increased afterload, Renal vasoconstriction
4)Inotropes - Hypotension , tachycardia
5)Diuretics - Shock worsening
6)Aggressive ventilation – shock worsening

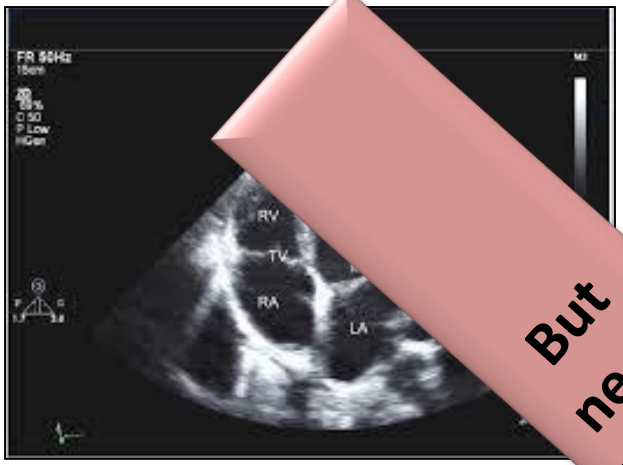




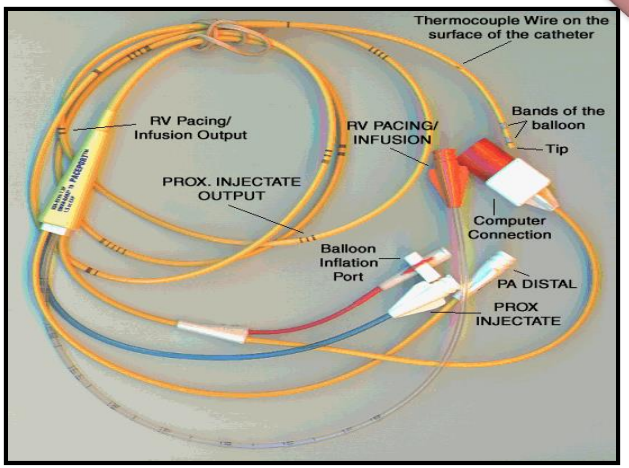


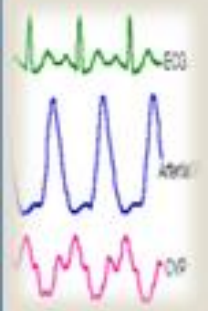
IDCCM, IFCCM,FNB,DM EDIC

You may not believe them

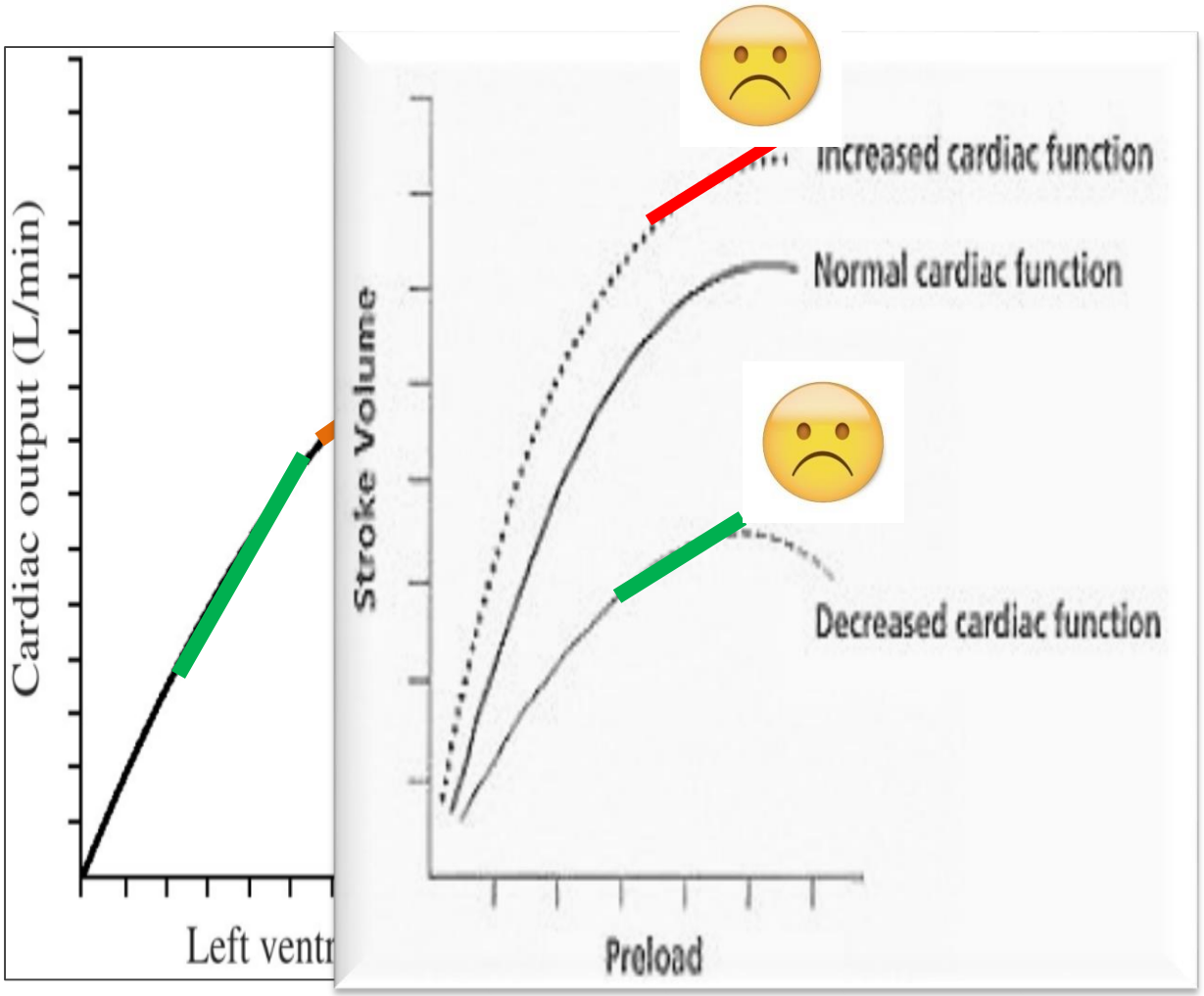


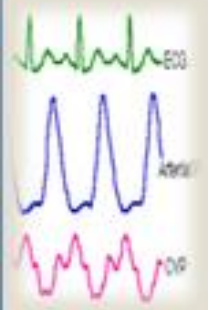
But need them!



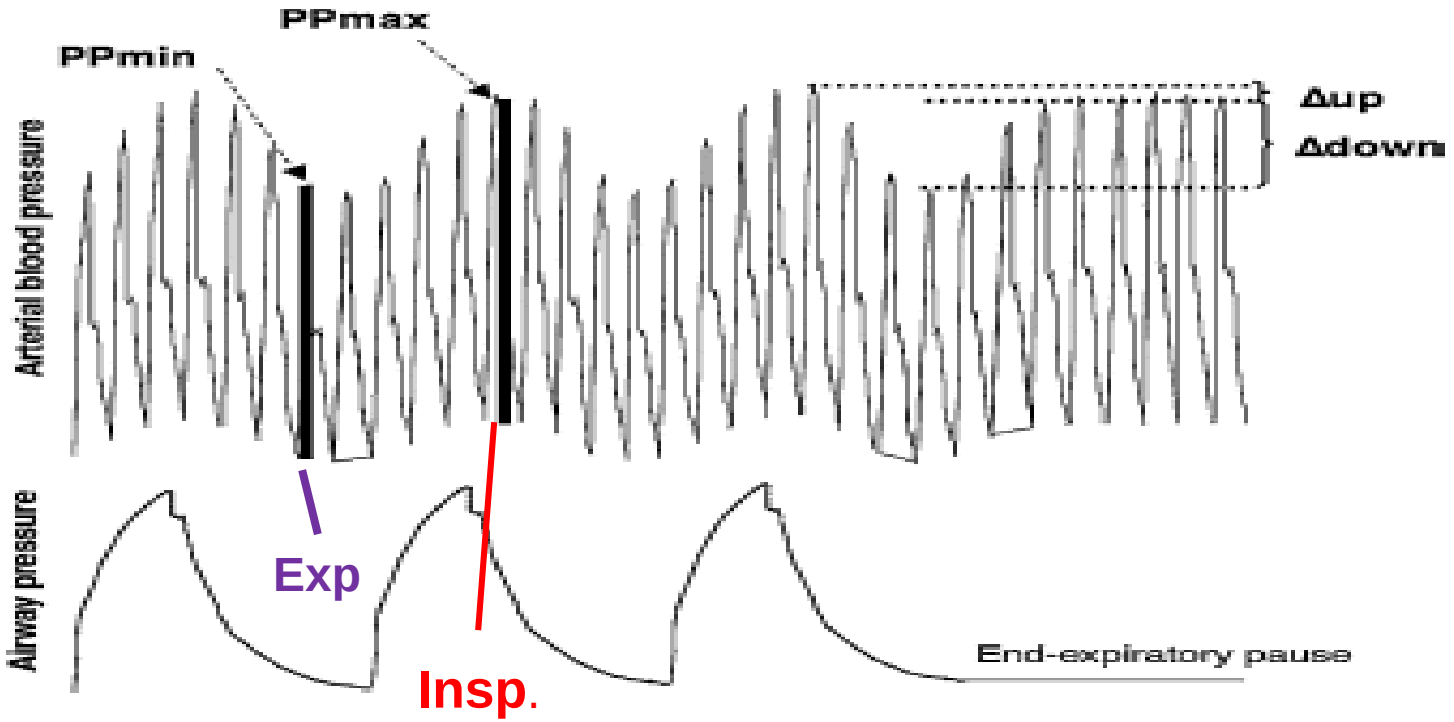



Frank- Starling

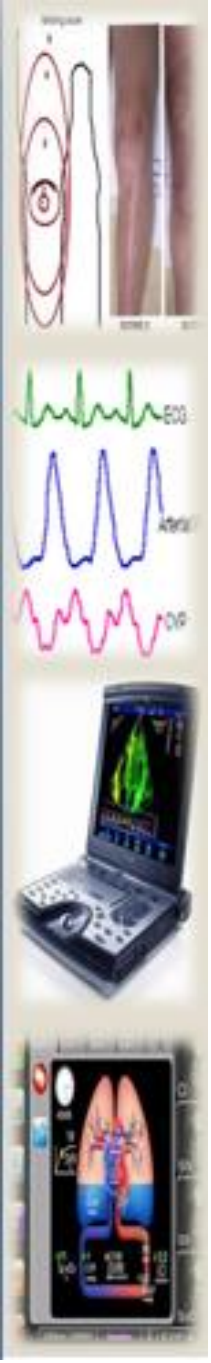




Respiratory Variation in PP



Reverse Pulsus Paradoxus during MV



Dynamic Variables

Controlled ventilation

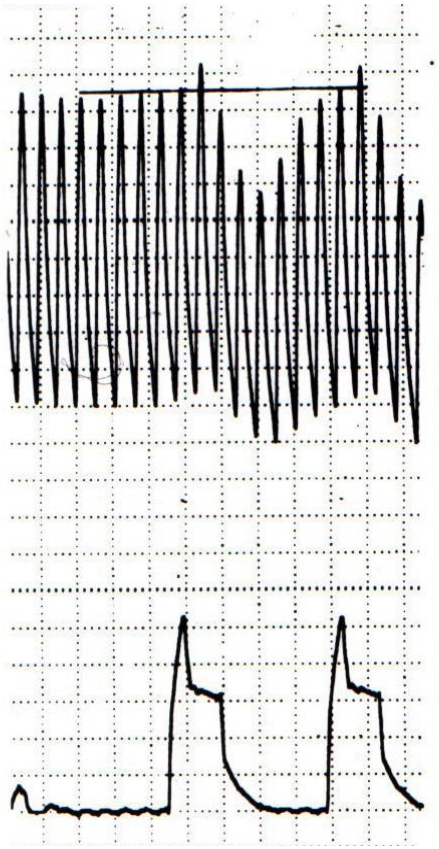
- Systolic pressure variation (SPV)
- Pulse pressure variation (PPV)
- Stroke volume variation (SVV)
- IVC distensibility index
- SVC collapsibility index
- Aortic flow velocity

Spontaneous/Assisted ventilation

- Passive leg raising
- Aortic blood flow velocity
- ITBV
- EVLWI

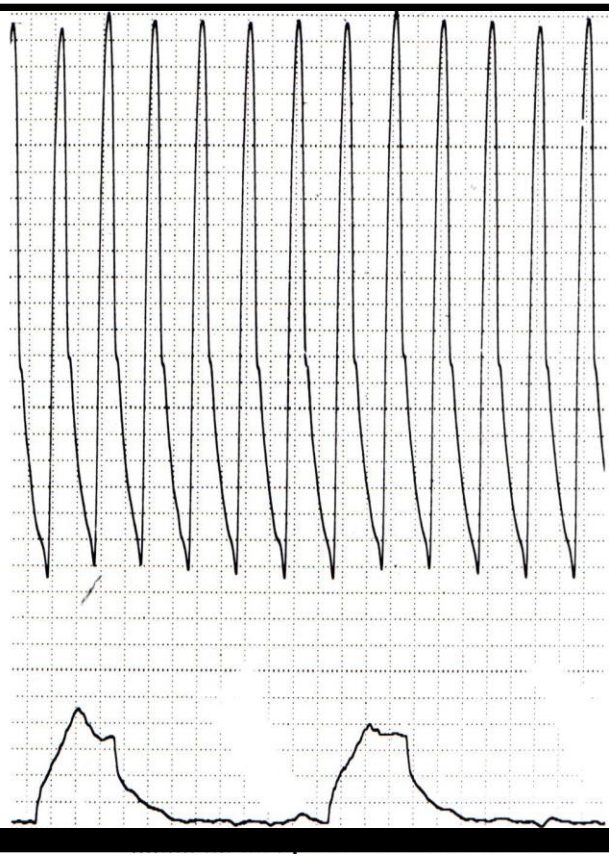
Fluid-responsive patient

SBP	137 mmHg
PCWP	5 mmHg
CI	1.26 l/min/m^2
LVEDA	5.4 cm^2
SPV	20 mmHg
△ up	4 mmHg
△ down	16 mmHg

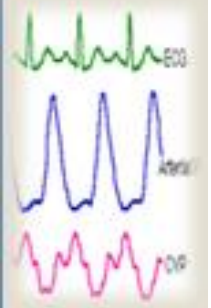


Fluid Non Responsive Patient

SBP	170 mmHg
PCWP	9 mmHg
CI	3.71 l/min/m^2
LVEDA	17,3 cm^2
SPV	2 mmHg
△ up	2 mmHg
△ down	0 mmHg



Coriat P et al, Anesth Analg 1994; 78: 46-53

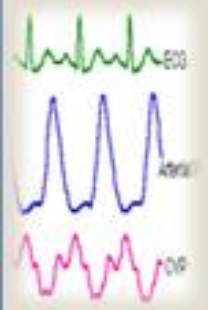




Advanced monitoring tools



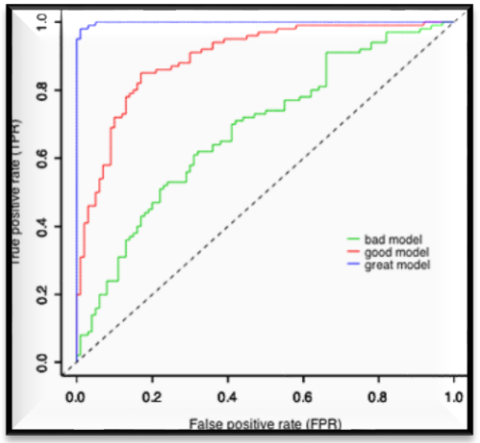




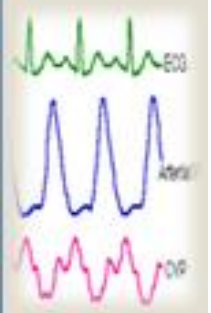



Assessing fluid responsiveness
ROC – Sensitivity & Specificity

Dynamic techniques based on heart–lung interactions during mechanical ventilation (ROC ~0.7–0.8)

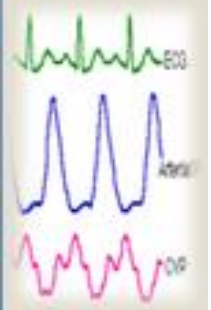


1. PPV
2. SVV
3. Pleth variability index
4. Aortic blood flow (Doppler or echocardiography)

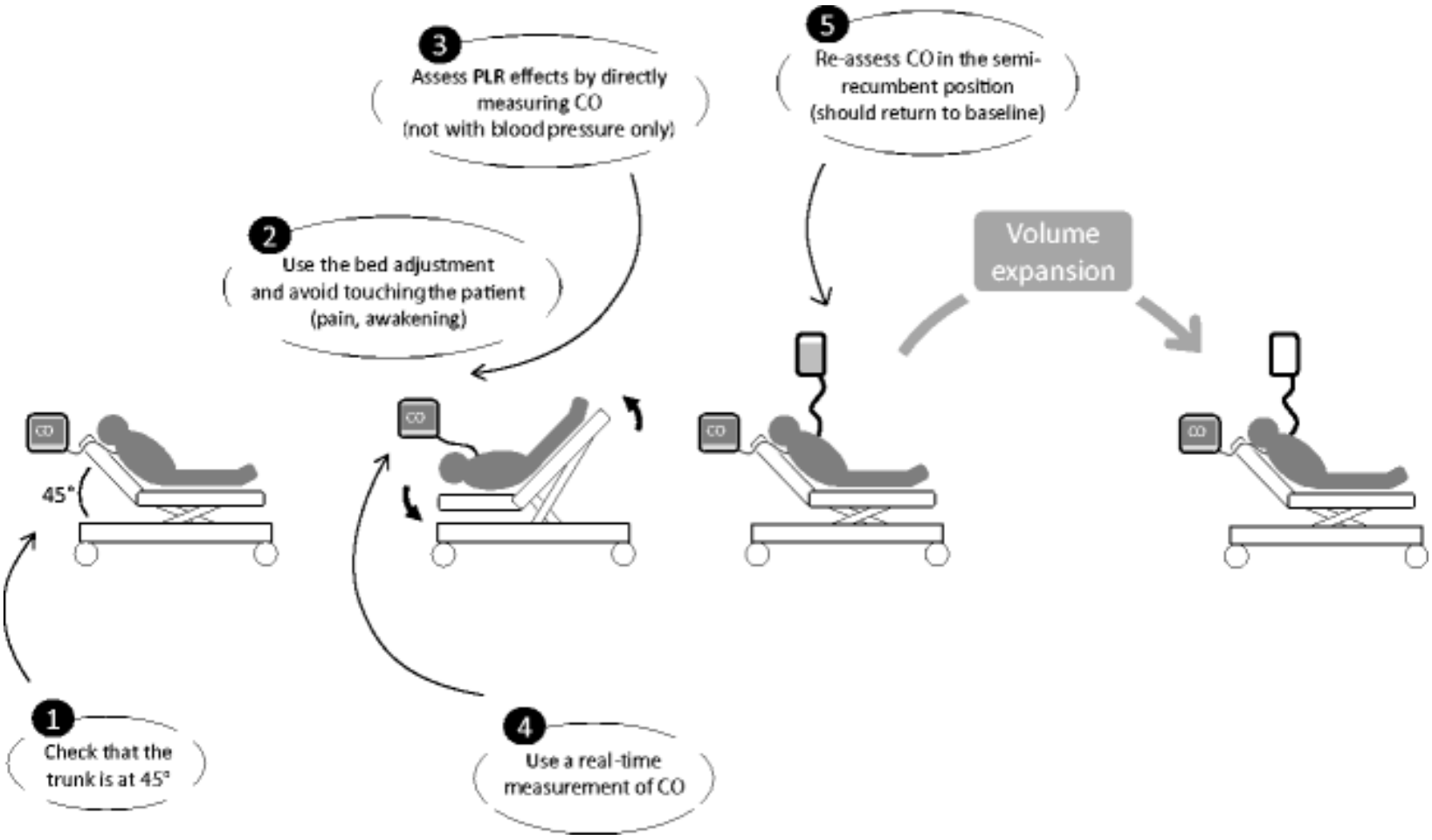



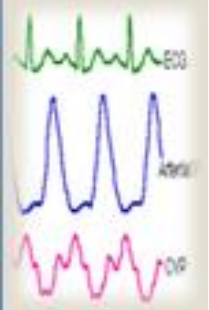
Limitations

- Spontaneously/Assisted breathing patients
- Cardiac arrythmia.
- TV of < 8 ml/kg.
- Low Compliance of respiratory system (<30ml/cmH2O)
- Severe pulm HT
- Open chest surgery
- IAH



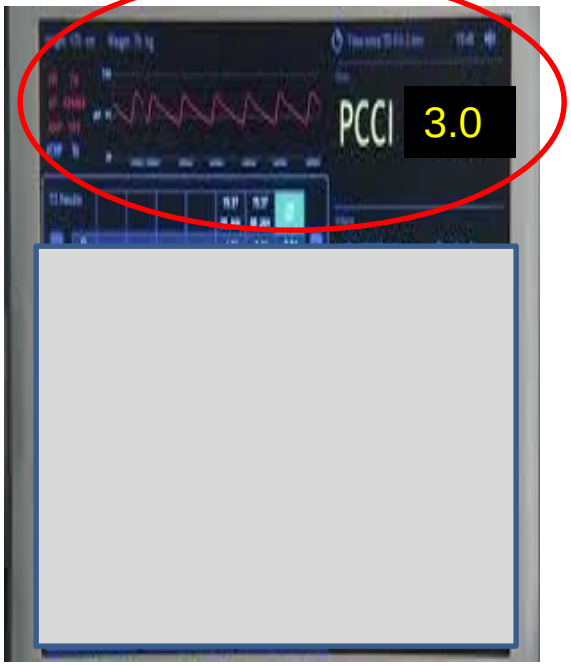
PLR


3
Assess PLR effects by directly measuring CO (not with blood pressure only)
5
Re-assess CO in the semi-recumbent position (should return to baseline)
2
Use the bed adjustment and avoid touching the patient (pain, awakening)
Volume expansion
45°
1
Check that the trunk is at 45°
4
Use a real-time measurement of CO

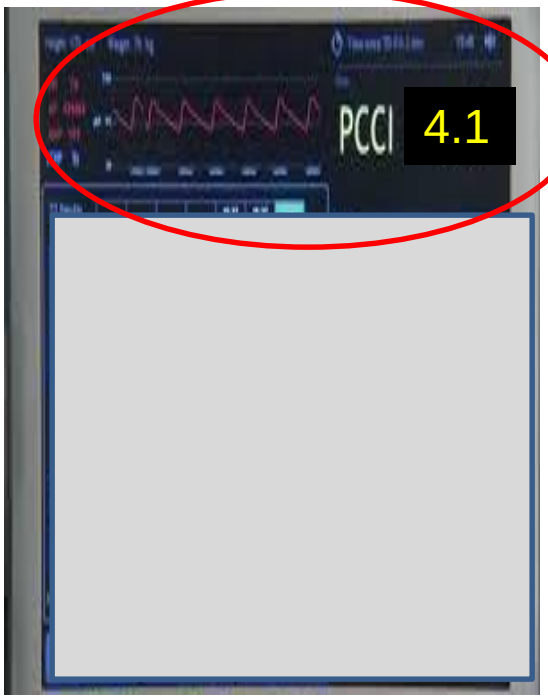


PLR- Wrong ways of doing and reading !

PLR- Right way of reading !

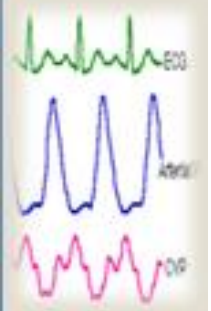


Passive leg raising



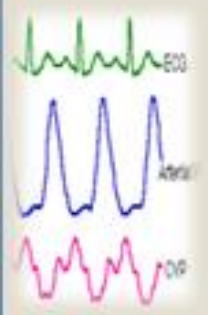
Rise of 10 % CO/ SV, Surrogate marker - Pulse Pressure

Never systolic, diastolic or Mean Pressure



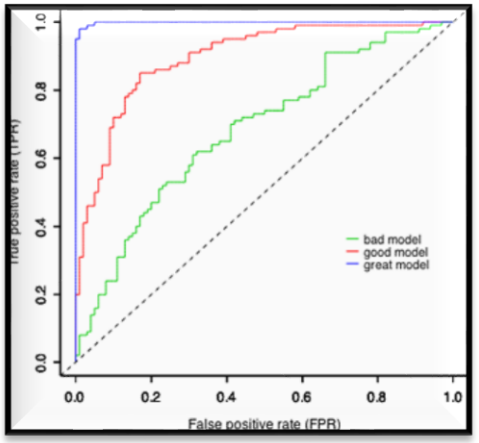
Limitations of PLR

- False negative - grossly volume depleted
- PLR induced changes in arterial pulse pressure are less accurate then CO/SV as variables.
- OR, leg fractures or hip fractures
- Intra-abdominal hypertension



Assessing fluid responsiveness
ROC – Sensitivity & Specificity

Techniques based on real or virtual fluid challenge (ROC~0.9)



1. PLR

2. Rapid fluid challenge (100–250 cc)

Dynamic Variables in monitoring
Fact / Fad / Fashion?

SSC 2016

- We suggest that dynamic over static variables be used to predict fluid responsiveness, where available **(weak recommendation, low quality of evidence)**.

RCT of PiCCO versus CVP

Effectiveness of treatment based on PiCCO parameters in critically ill patients with septic shock and/or acute respiratory distress syndrome: a randomized controlled trial

Zhongheng Zhang| Hongying Ni| Zhixian Qian

Original
Volume 41, Issue 3 / March , 2015
Pages 444 - 451

Conclusion

On the basis of our study, PICCO-based fluid management does not improve outcome when compared to CVP-based fluid management.

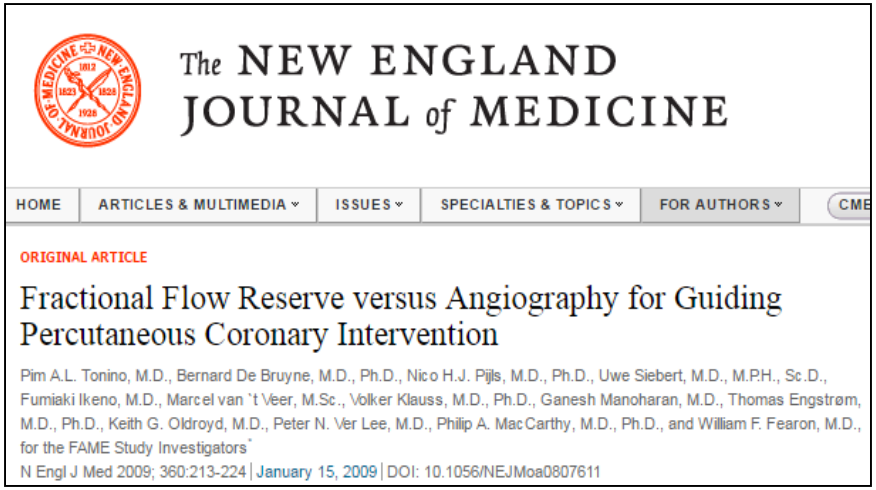
Can a diagnostic modality reduce mortality in RCT?

- Doing CT scan Versus not doing CT scan
- Using PA catheter versus not using PA catheter
- Using PiCCO (or any advanced hemodynamic monitoring) versus not using PiCCO (or any advanced hemodynamic monitoring) for fluid resuscitation

Can a diagnostic modality reduce mortality in RCT?

YES

but not in Critical Care !!



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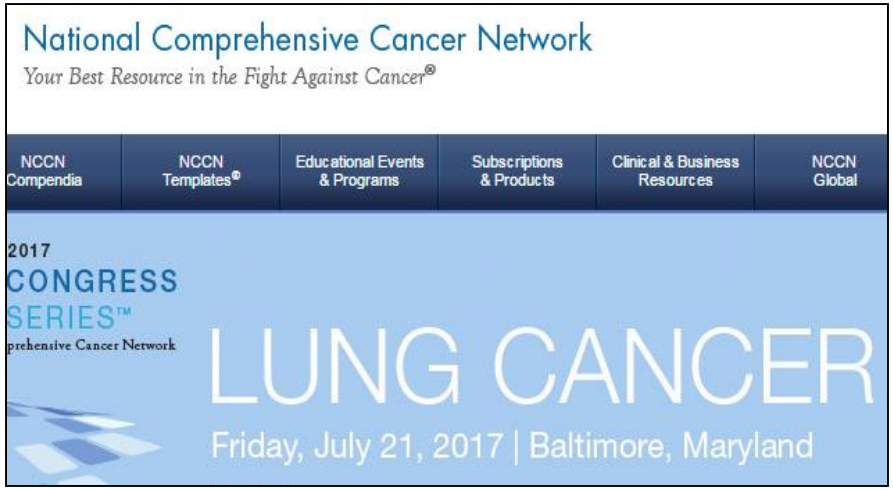
ORIGINAL ARTICLE

Fractional Flow Reserve versus Angiography for Guiding Percutaneous Coronary Intervention

Pim A.L. Tonino, M.D., Bernard De Bruyne, M.D., Ph.D., Nico H.J. Pijls, M.D., Ph.D., Uwe Siebert, M.D., M.P.H., Sc.D., Fumiaki Ikeno, M.D., Marcel van 't Veer, M.Sc., Volker Klauss, M.D., Ph.D., Ganesh Manoharan, M.D., Thomas Engstrøm, M.D., Ph.D., Keith G. Oldroyd, M.D., Peter N. Ver Lee, M.D., Philip A. MacCarthy, M.D., Ph.D., and William F. Fearon, M.D., for the FAME Study Investigators*

N Engl J Med 2009; 360:213-224 | January 15, 2009 | DOI: 10.1056/NEJMoa0807611



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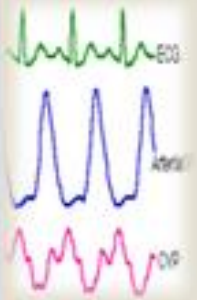
Friday, July 21, 2017 | Baltimore, Maryland

Conclusion - Routine measurement of FFR in patients with multivessel coronary artery disease who are undergoing PCI with drug-eluting stents significantly reduces the rate of the composite end point of death, nonfatal myocardial infarction, and repeat revascularization at 1 year.

Patients with advanced non–small-cell lung cancer with a mutant epidermal growth factor receptor (EGFR), **EGFR positive** EGFR tyrosine kinase inhibitors (TKIs) are the standard first-line therapy. ***Gefitinib***



How I give fluids ?

the Answer lies HERE !!

Echo

Urine

SVV

PLR

Lactates

V-A PCO_2

Thank You !

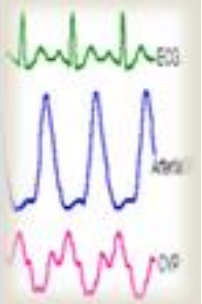
GEDV

CVP

PPV

CRT

FC



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www.dmhemcrit.com