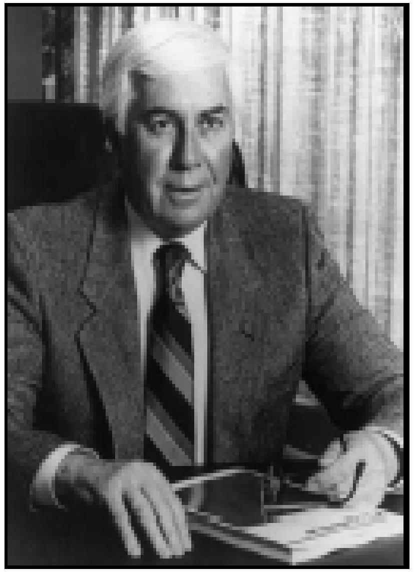
Monitoring

Michel Slama

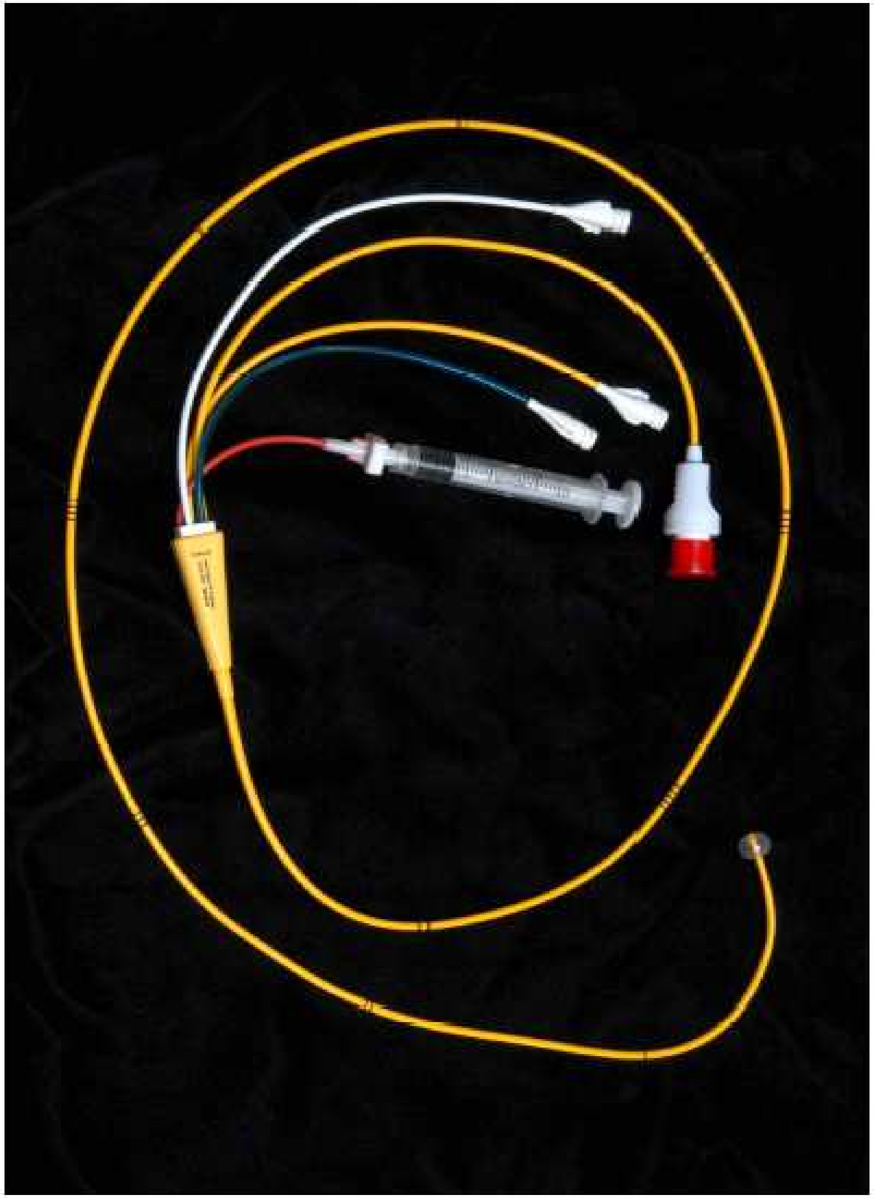
Amiens

France



H Jeremy C Swan

Respected cardiologist and co-inventor of the Swan-Ganz catheter. He was born on Jan 1, 1922, in Sligo, Ireland; he died on Feb 7, 2005, after a heart attack in Los Angeles, CA, USA, aged 82 years.



Impact of the Pulmonary Artery Catheter in Critically Ill Patients

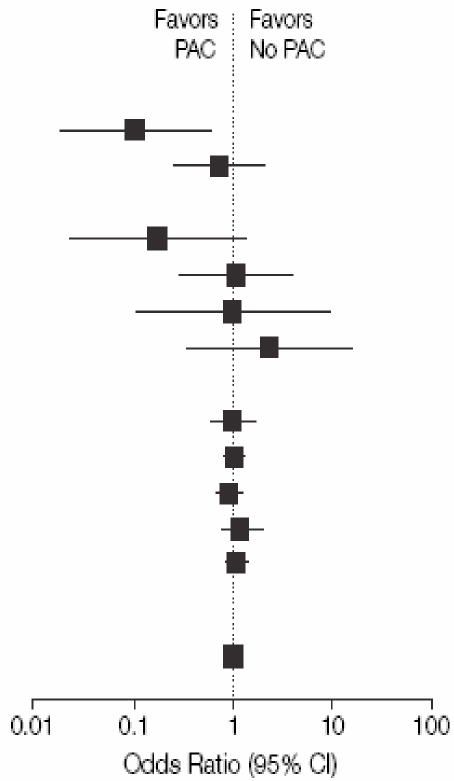
Meta-analysis of Randomized Clinical Trials

Monica R. Shah, MD, MHS, MSJ
Vic Hasselblad, PhD
Lynne W. Stevenson, MD
Cynthia Binanay, RN, BSN
Christopher M. O'Connor, MD
George Sopko, MD, MPH
Robert M. Califf, MD

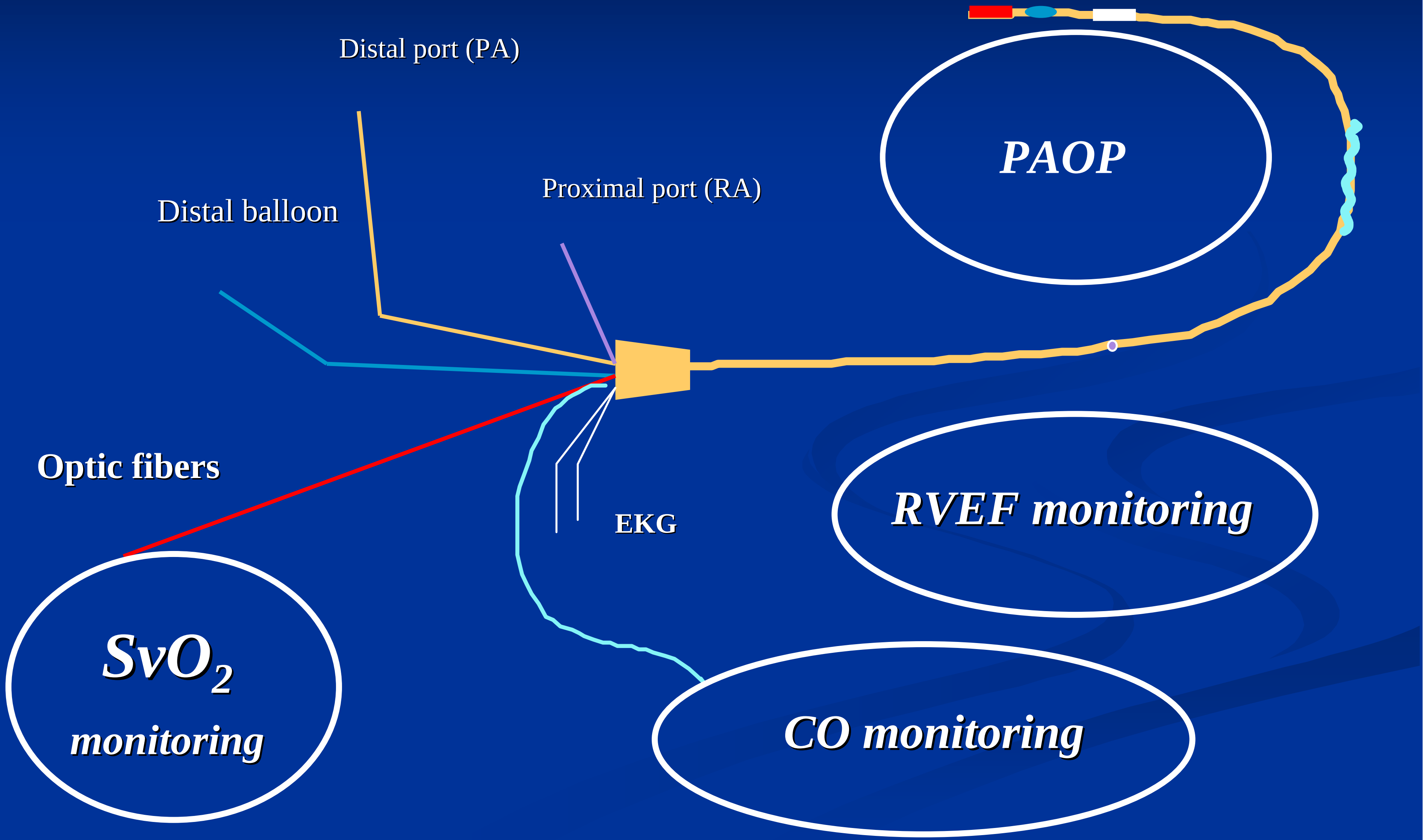
JAMA. 2005;294:1664-1670

Figure 2. Odds Ratio (PAC vs No PAC) for Mortality of RCTs Evaluating the Safety and Efficacy of the PAC

Source	No. of Deaths/Total No. of Patients: PAC	No PAC	Odds Ratio (95% CI)
Schultz et al,[15] 1985	1/35	10/35	0.11 (0.02-0.63)
Shoemaker et al,[16] 1988	11/58	7/30	0.76 (0.27-2.15)
Isaacson et al,[17] 1990	1/49	0/53	NA
Berlauk et al,[18] 1991	1/66	2/21	0.18 (0.02-1.42)
Guyatt,[19] 1991	10/16	9/17	1.10 (0.29-4.22)
Bender et al,[20] 1997	1/51	1/53	1.04 (0.11-9.95)
Valentine et al,[21] 1998	3/60	1/60	2.38 (0.35-16.29)
Bonazzi et al,[22] 2002	0/50	0/50	NA
Rhodes et al,[23] 2002	46/95	50/106	1.01 (0.58-1.76)
Sandham et al,[24] 2003	163/997	155/997	1.06 (0.83-1.35)
Richard et al,[25] 2003	199/338	208/343	0.93 (0.68-1.26)
ESCAPE,[10] 2005	45/215	38/218	1.25 (0.78-2.02)
Harvey et al,[14] 2005 (PAC-Man)	346/506	333/507	1.13 (0.87-1.47)
Combined			1.04 (0.90-1.20)



PAC technology



The New England
Journal of Medicine

Volume 333 | OCTOBER 19, 1995 | Number 16

A TRIAL OF GOAL-ORIENTED HEMODYNAMIC THERAPY IN CRITICALLY ILL PATIENTS

Luciano Gattinoni, M.D., Luca Brazzi, M.D., Paolo Pelosi, M.D., Roberto Latini, M.D.,
Gianni Tognoni, M.D., Antonio Pesenti, M.D., and Roberto Fumagalli, M.D.,
for the SvO_2 Collaborative Group*

**SAPS of 11 or higher
high risk after surgery,
massive blood loss,
septic shock or sepsis
syndrome,
acute respiratory failure,
acute respiratory failure
or multiple trauma.**

**Normal cardiac index
2,5-3,5 l/min/m^2**

**High cardiac index
> 4,5 l/min/m^2**

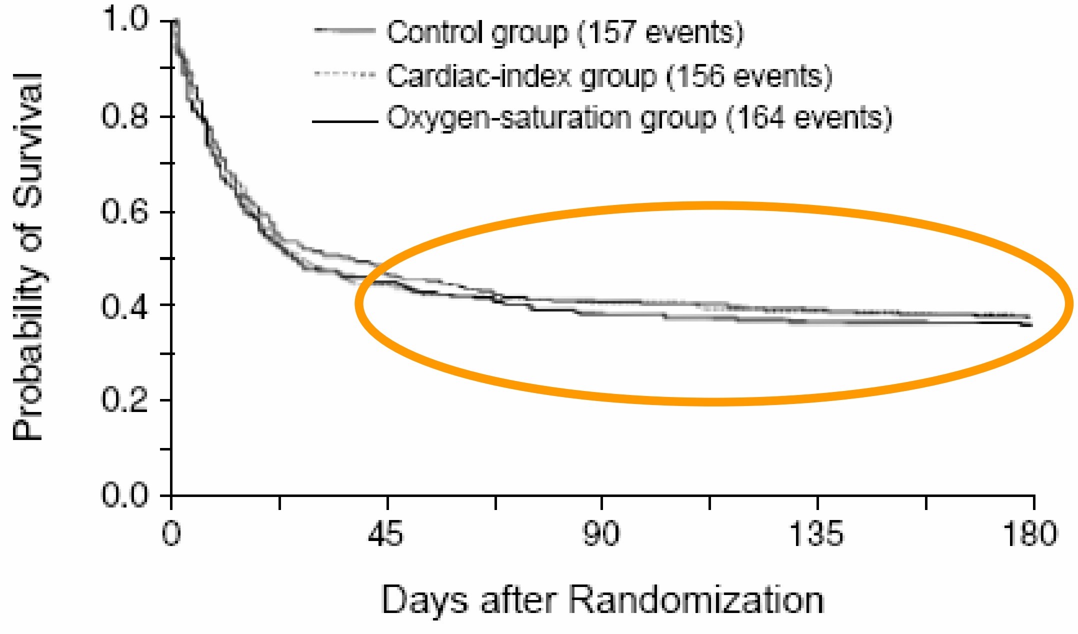
SvO2> 70%

Gattinoni NEJM 1995

Table 2. Characteristics of the Patients during the Five-Day Study Period.

Characteristic*	Control Group (N = 252)	Cardiac-Index Group (N = 253)	Oxygen-Saturation Group (N = 257)	P Value†
		mean ±SD		
Cardiac index (liters/min/m^2)	3.9±1.0	4.4±1.3‡§	4.1±1.2	**<0.001**
Oxygen delivery (ml/min/m^2)	575±164	641±184‡§	591±165	**<0.001**
Oxygen consumption (ml/min/m^2)	148±34	158±40‡¶	149±38	**0.006**
SvO_2 (%)	70.7±7.3	72.1±6.5	71.7±5.9	0.062
PaO_2/FiO_2‖	452±179	462±167	447±167	0.608
Mechanical ventilation (days)	3.5±1.8	3.2±1.9	3.6±1.8	0.092
Oxygen-extraction ratio (%)	27.1±6.9	25.8±6.3	26.2±5.4	0.056
Pulmonary arterial pressure (mm Hg)	27.7±7.5	26.4±6.4¶**	27.8±6.8	**0.042**

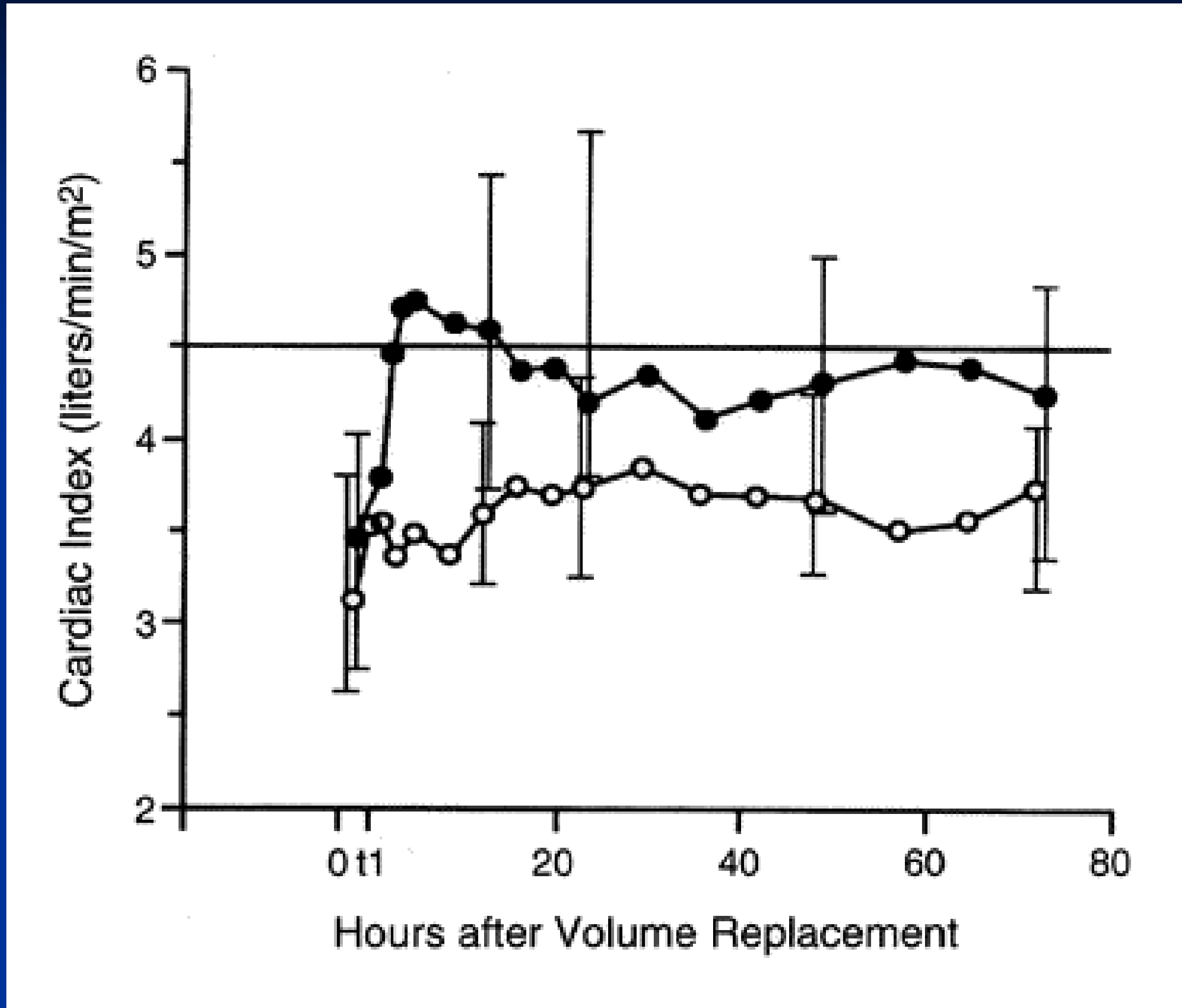
Gattinoni NEJM 1995



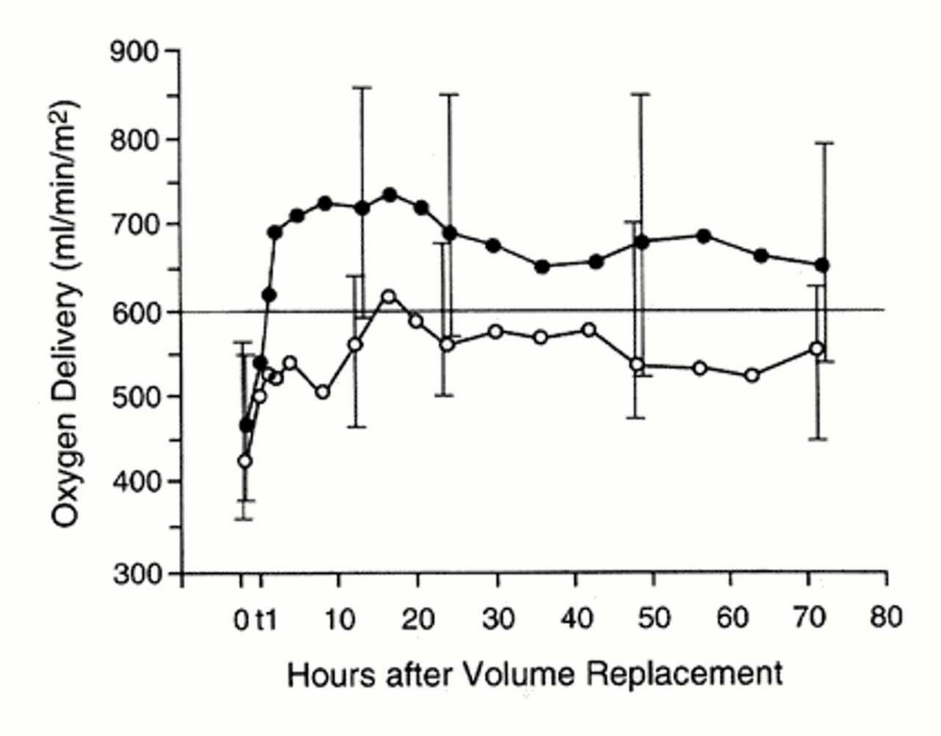
PATIENTS AT RISK (NO. OF EVENTS)

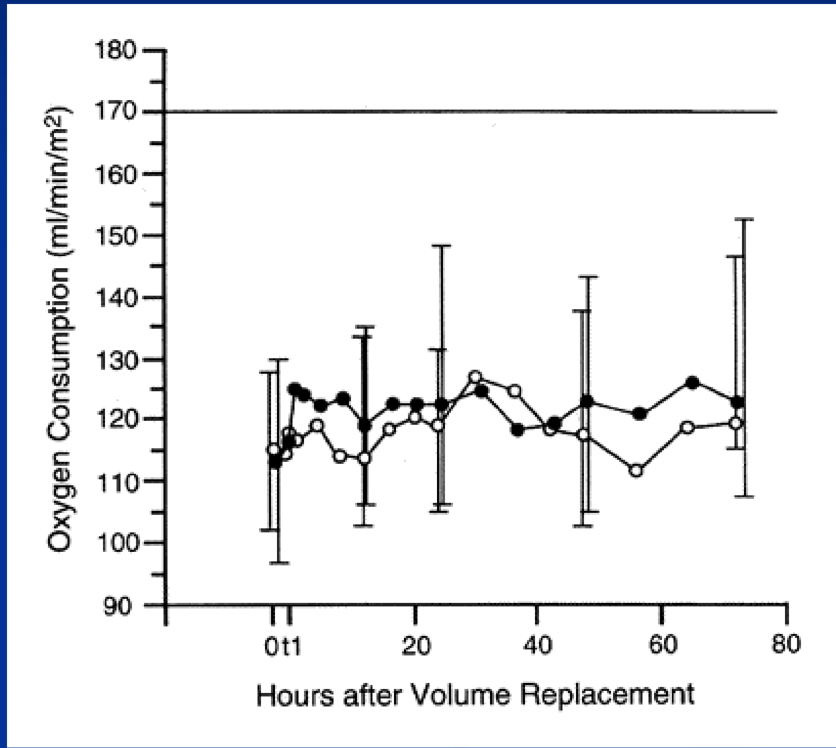
	0	45	90	135	180
Control group	252 (129)	108 (13)	94 (4)	90 (3)	87
Cardiac-index group	253 (133)	102 (8)	90 (4)	86 (3)	83
Oxygen-saturation group	257 (133)	106 (16)	89 (4)	85 (1)	84

Gattinoni NEJM 1995



Hayes NEJM 1994



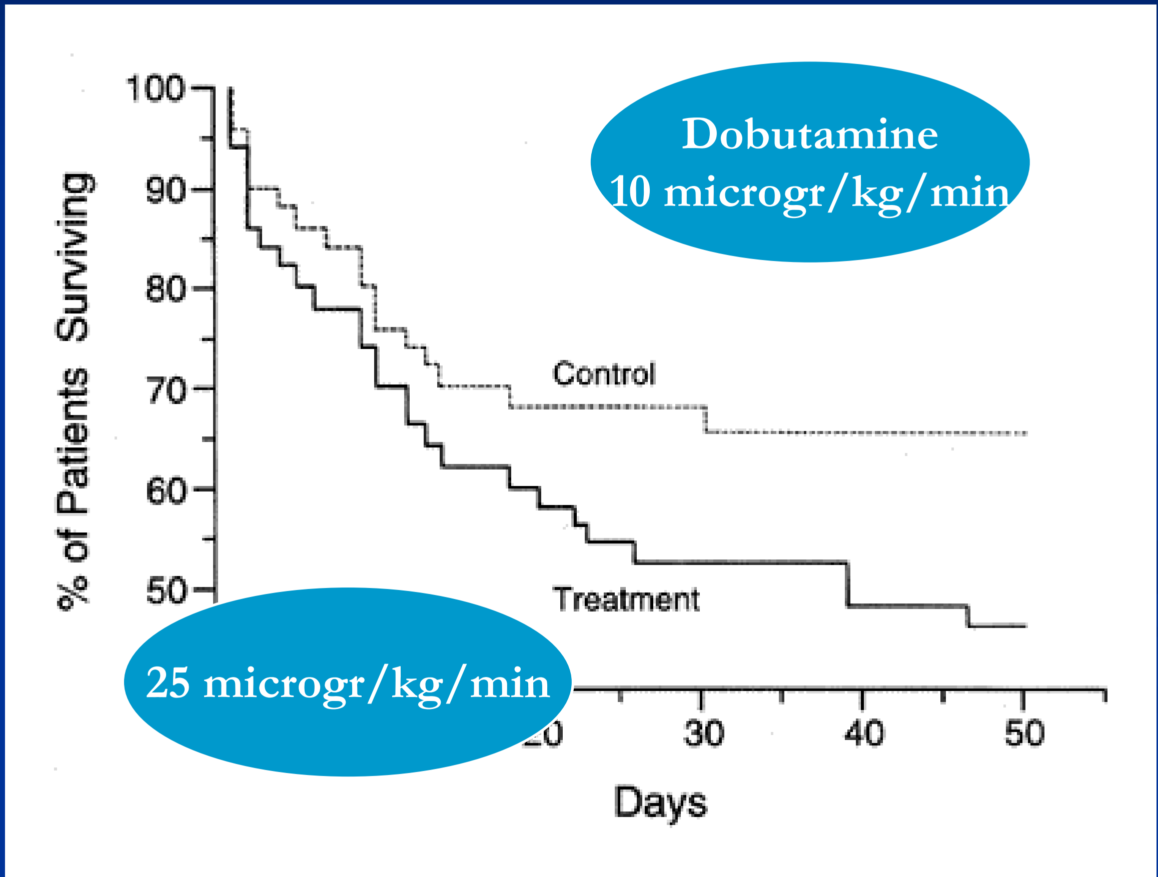


Hayes NEJM 1994

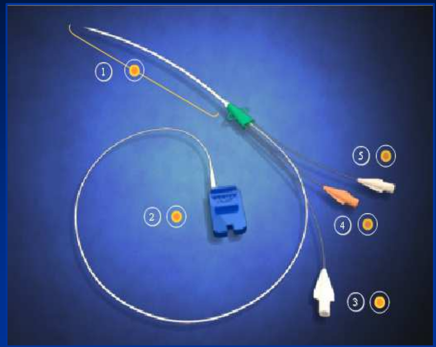
Outcome	Control Group (N = 50)	Treatment Group (N = 50)	Not Randomized (N = 9)
Days in unit — median (range)	10 (1–64)	10 (1–48)	10 (1–29)
Ventilation			
No. of days — median (range)	8 (0–54)	8 (0–41)	2 (0–26)
No. of patients	44	46	7
Days in hospital — median (range)	23.5 (1–244)	19 (1–187)	20 (11–102)
Mortality — %			
In intensive care unit	30	50*	—
In hospital	34	54*	—
Predicted risk of death — median % (range)	34 (3–91)	34 (3–85)	6 (3–32)
Cause of death — no. of patients			
Intractable hypotension	4	4	—
Cardiac event	2	4	—
Multiple organ failure	9	17	—

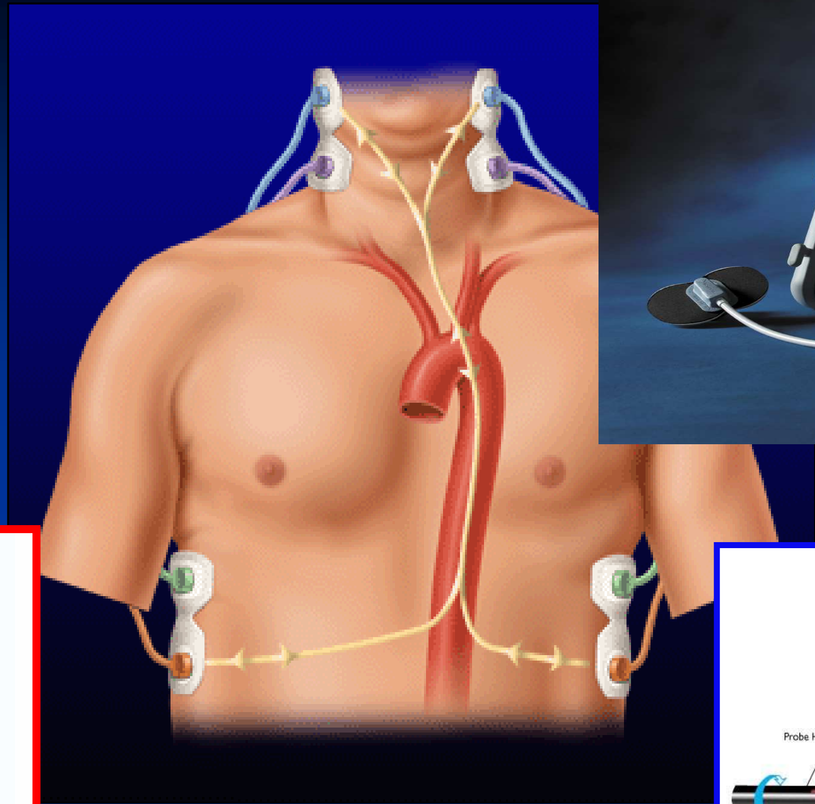
*P = 0.04 for the comparison between the control and treatment groups.

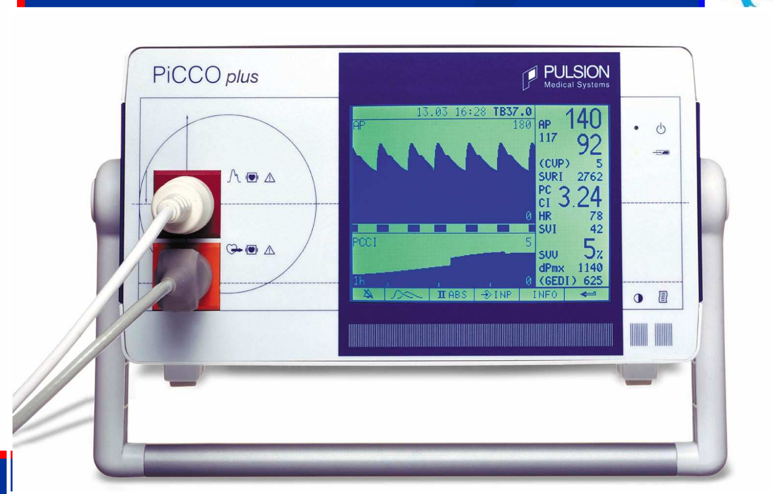
Hayes NEJM 1994

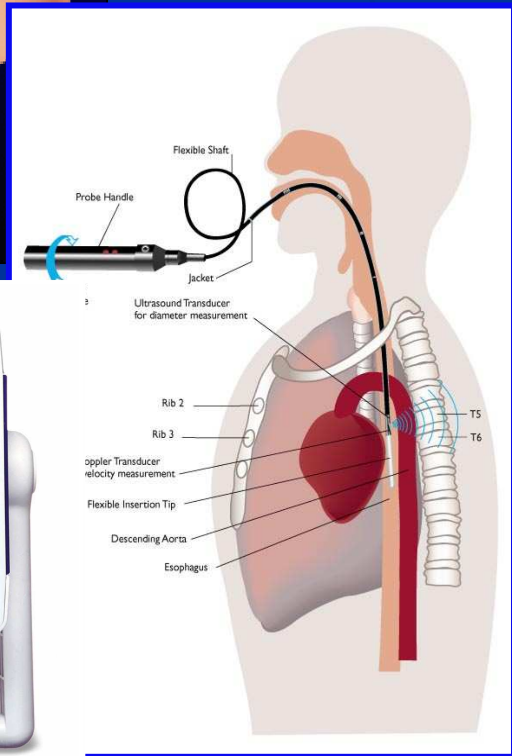


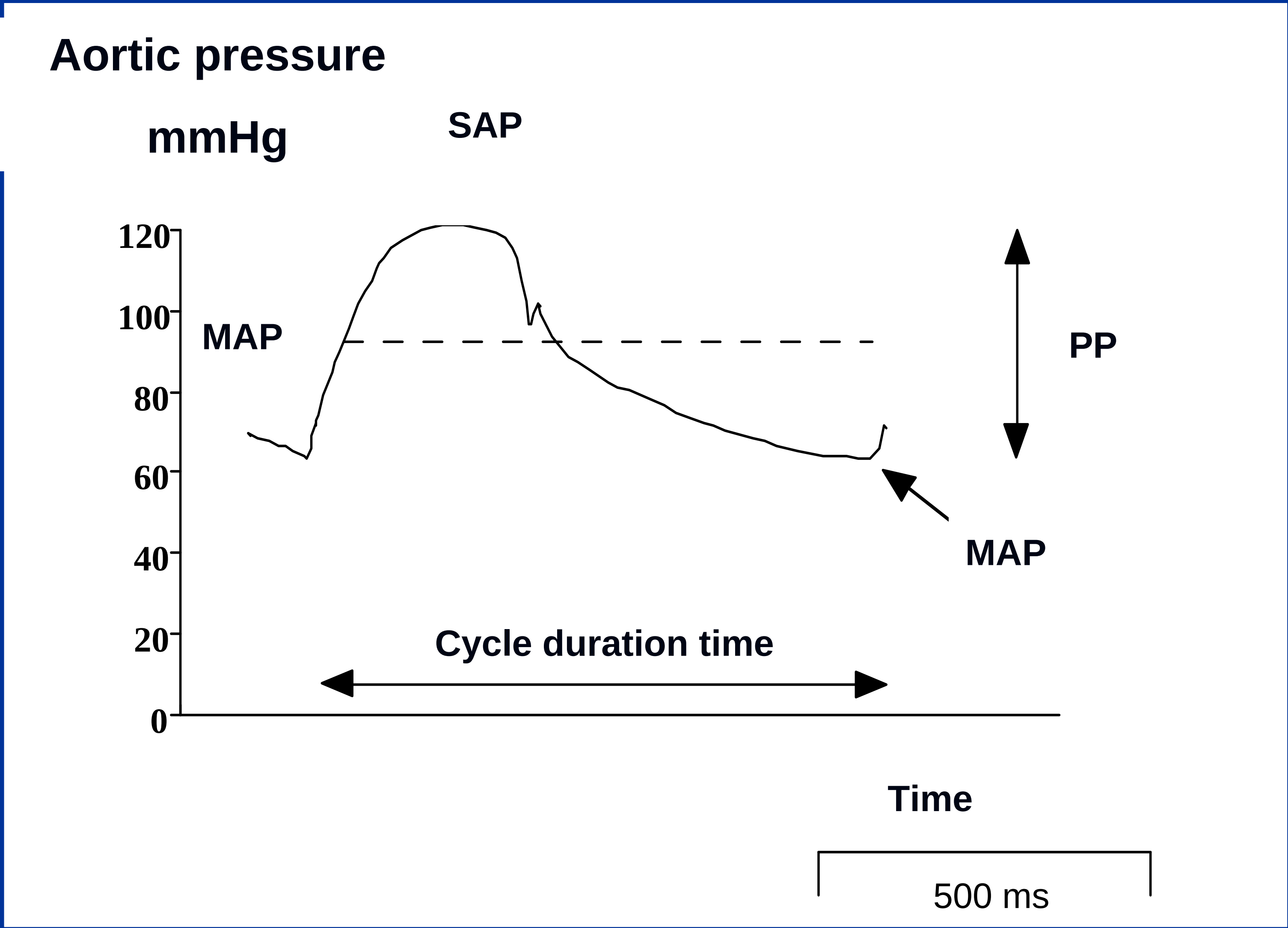
Hayes NEJM 1994



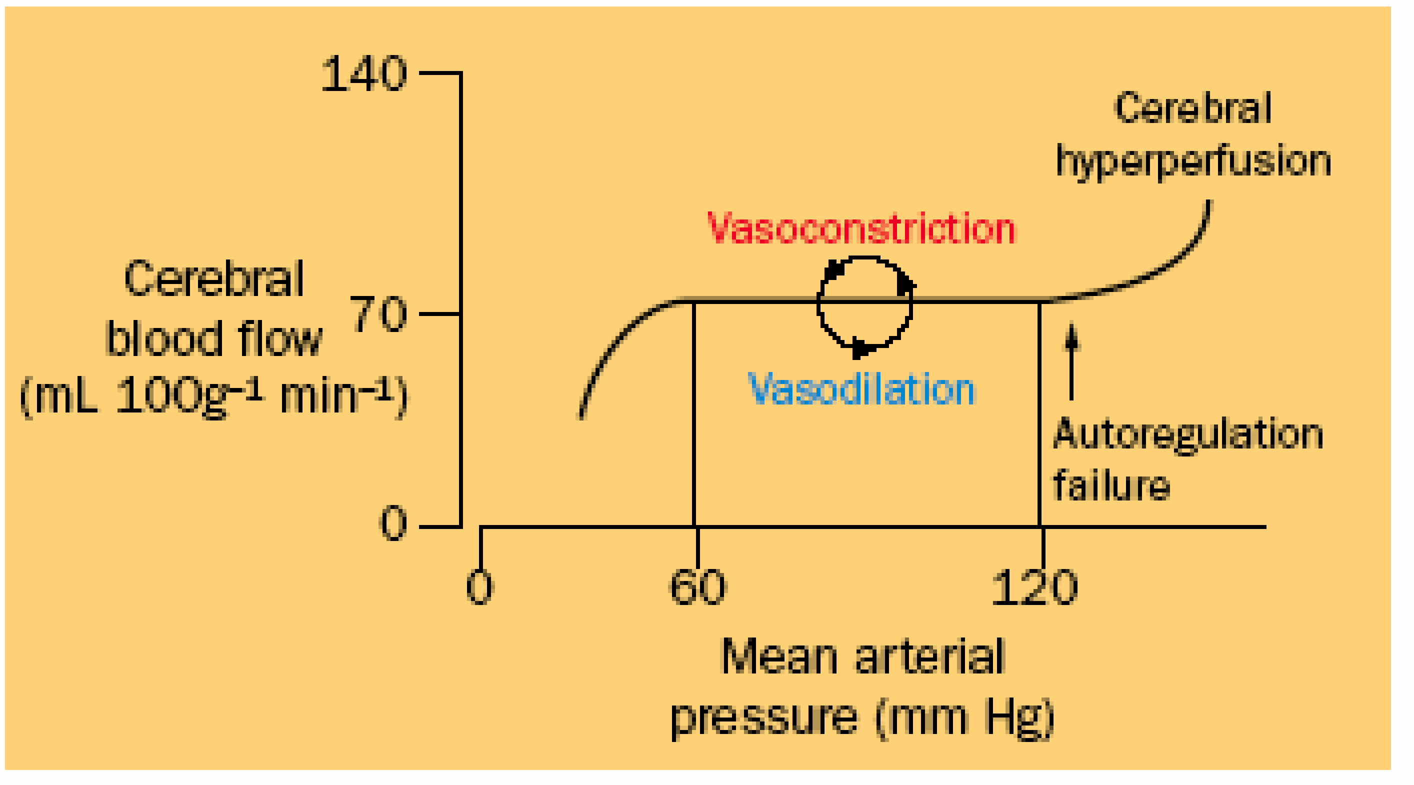



PiCCO plus
PULSION
Medical Systems

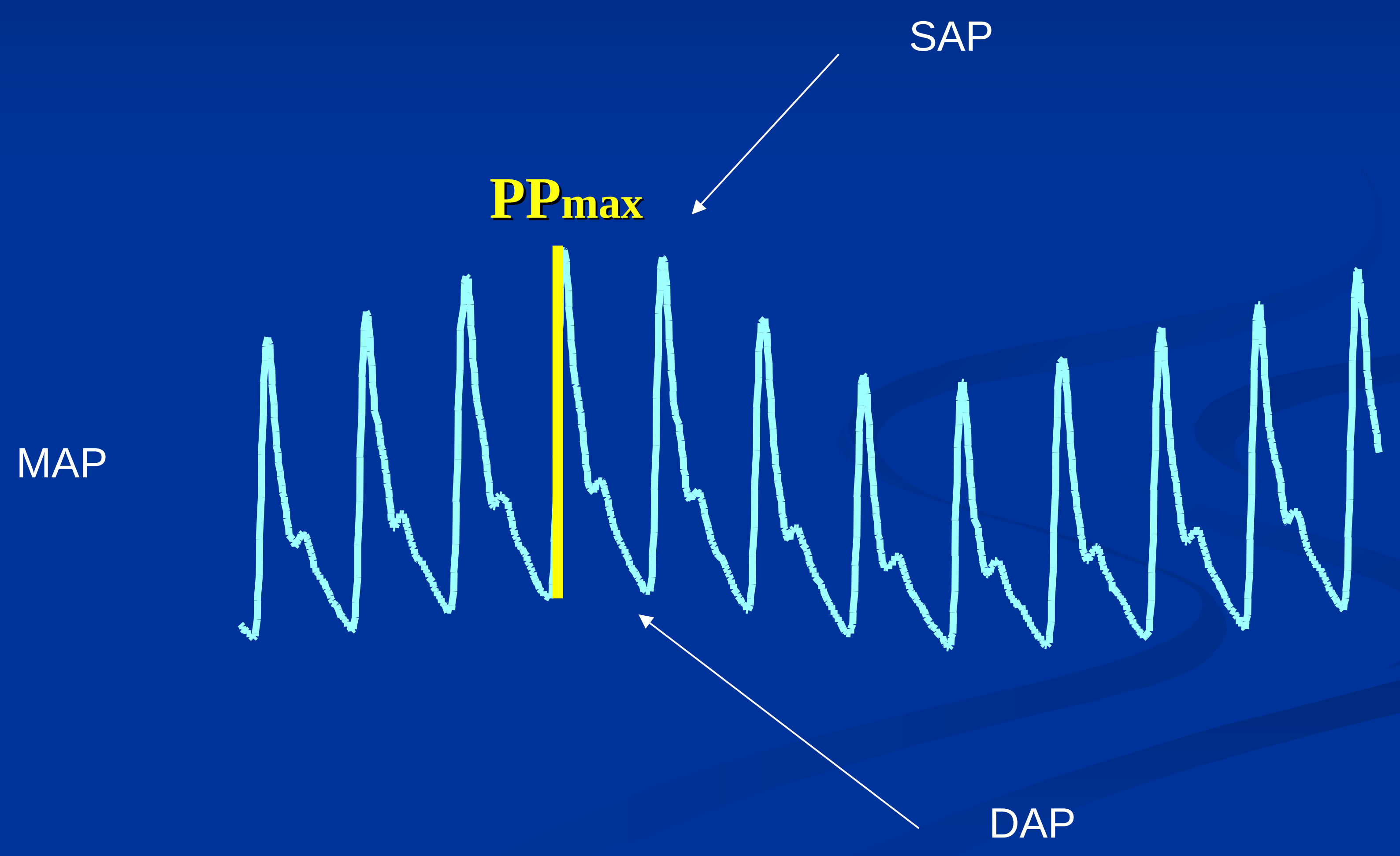

Flexible Shaft
Probe Handle
Jacket
Ultrasound Transducer
for diameter measurement
Rib 2
Rib 3
T5
T6
oppler Transducer
elocity measurement
Flexible Insertion Tip
Descending Aorta
Esophagus


Aortic pressure
mmHg
SAP
120
100
80
60
40
20
0
MAP
PP
MAP
Cycle duration time
Time
500 ms

Cerebral Autoregulation and MAP



Arterial Blood Pressure

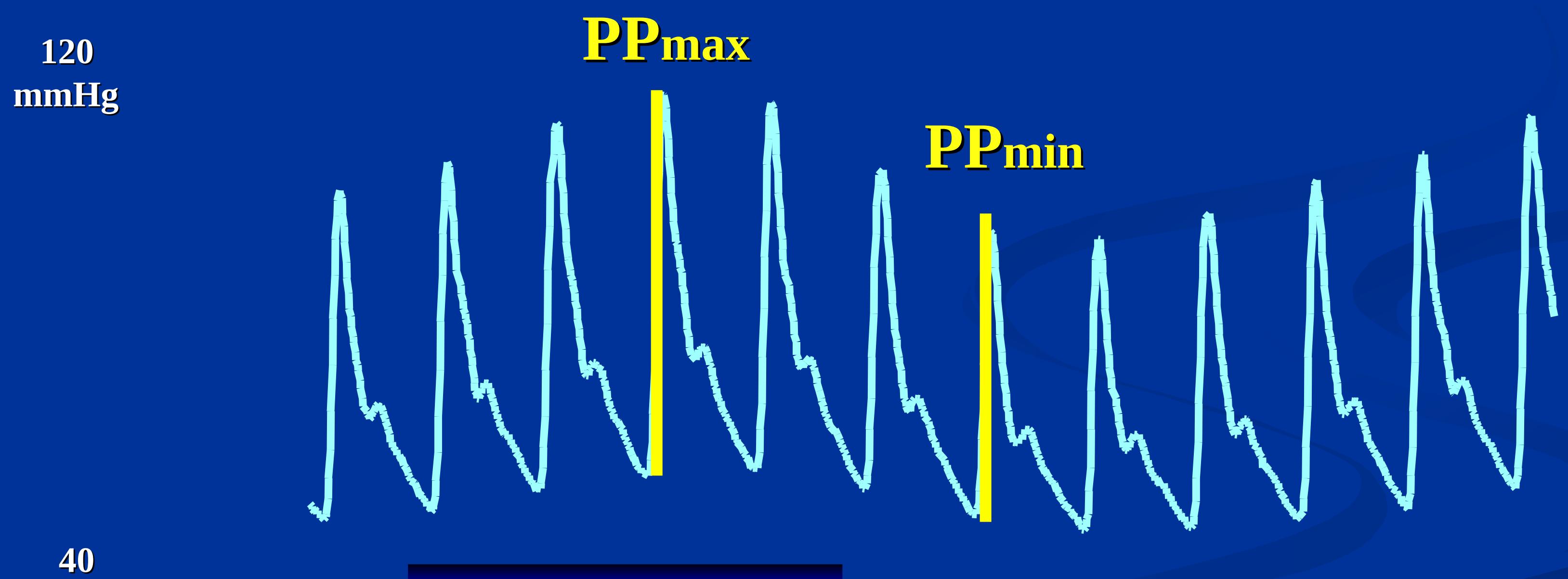


Relation between Respiratory Changes in Arterial Pulse Pressure and Fluid Responsiveness in Septic Patients with Acute Circulatory Failure

FRÉDÉRIC MICHARD, SANDRINE BOUSSAT, DENIS CHEMLA, NADIA ANGUEL, ALAIN MERCAT, YVES LECARPENTIER, CHRISTIAN RICHARD, MICHAEL R. PINSKY, and JEAN-LOUIS TEBOUL

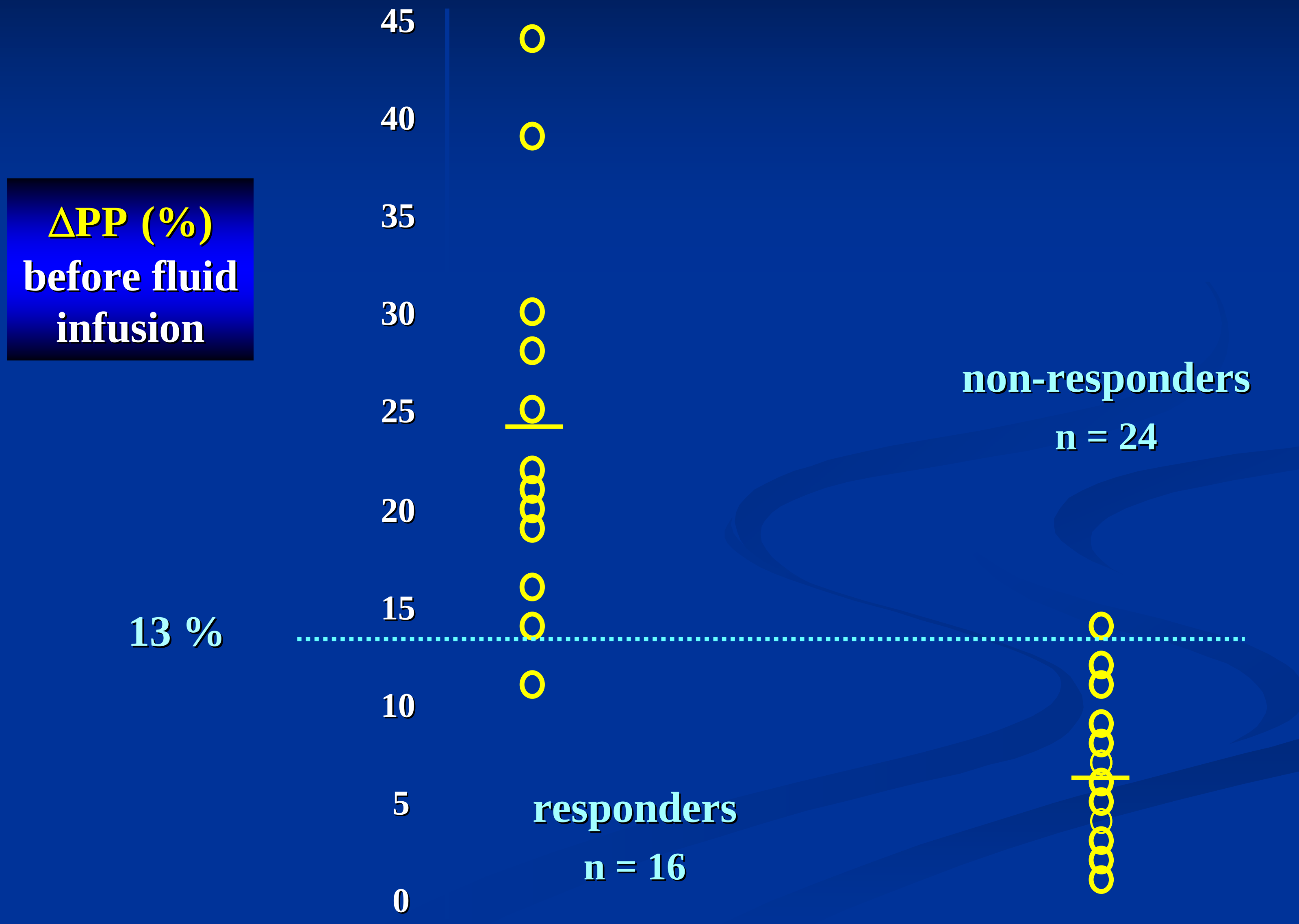
Am J Respir Crit Care Med 2000; 162:134-138

$$\Delta PP = \frac{PP_{max} - PP_{min}}{(PP_{max} + PP_{min})/2}$$

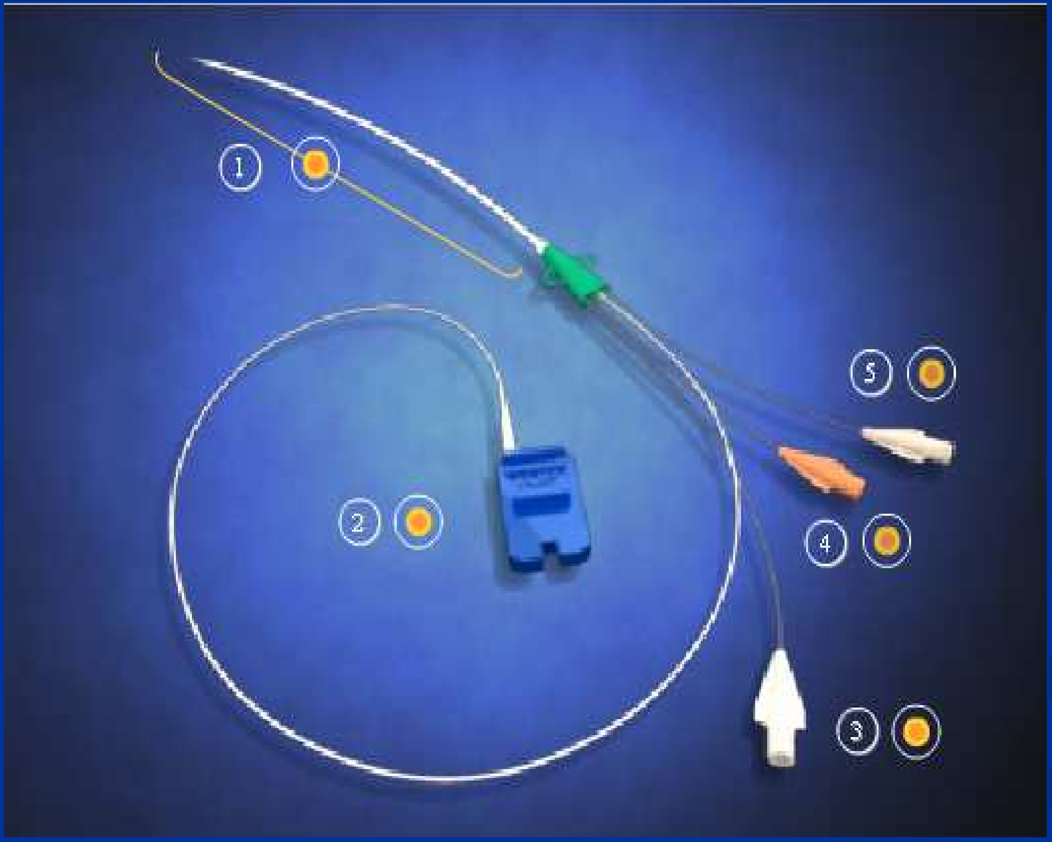


Arterial Pressure

Michard F, Boussat S, Chemla D, Anguel N, Mercat A, Lecarpentier Y, Richard C, Pinsky M, Teboul JL.
Relation between respiratory changes in arterial pulse pressure and fluid responsiveness in septic patients with acute circulatory failure. *Am J Respir Crit Care Med* 2000 ;162:134-138



ScvO2



$$VO_2 = (CaO_2 - CvO_2) \cdot Q$$

Fick…

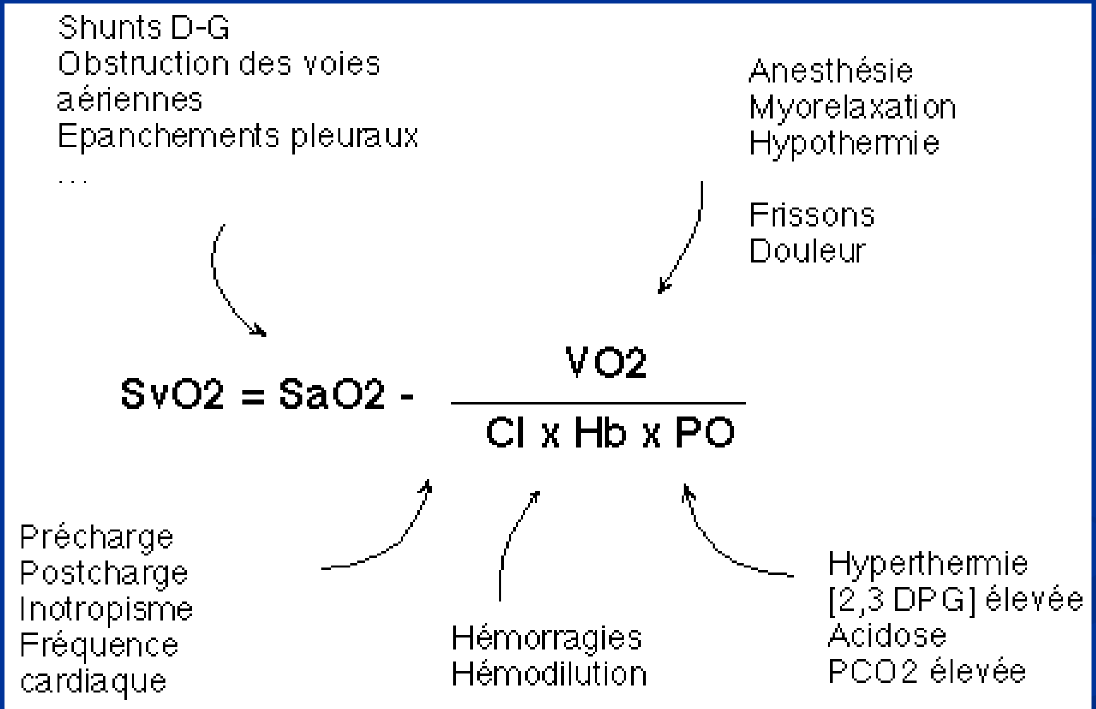
$$VO_2 \# (SaO_2 - SvO_2) \cdot (Hb \cdot 1{,}39 \cdot Q)$$

Considérant comme négligeable la quantité d'oxygène dissout dans le sang, l'équation de Fick peut être exprimée ainsi :

$$SvO_2 = SaO_2 - \frac{VO_2}{IC \times Hb \times 1.34}$$

Une variation de SvO_2 provient nécessairement de la variation d'un ou de plusieurs de ces déterminants …

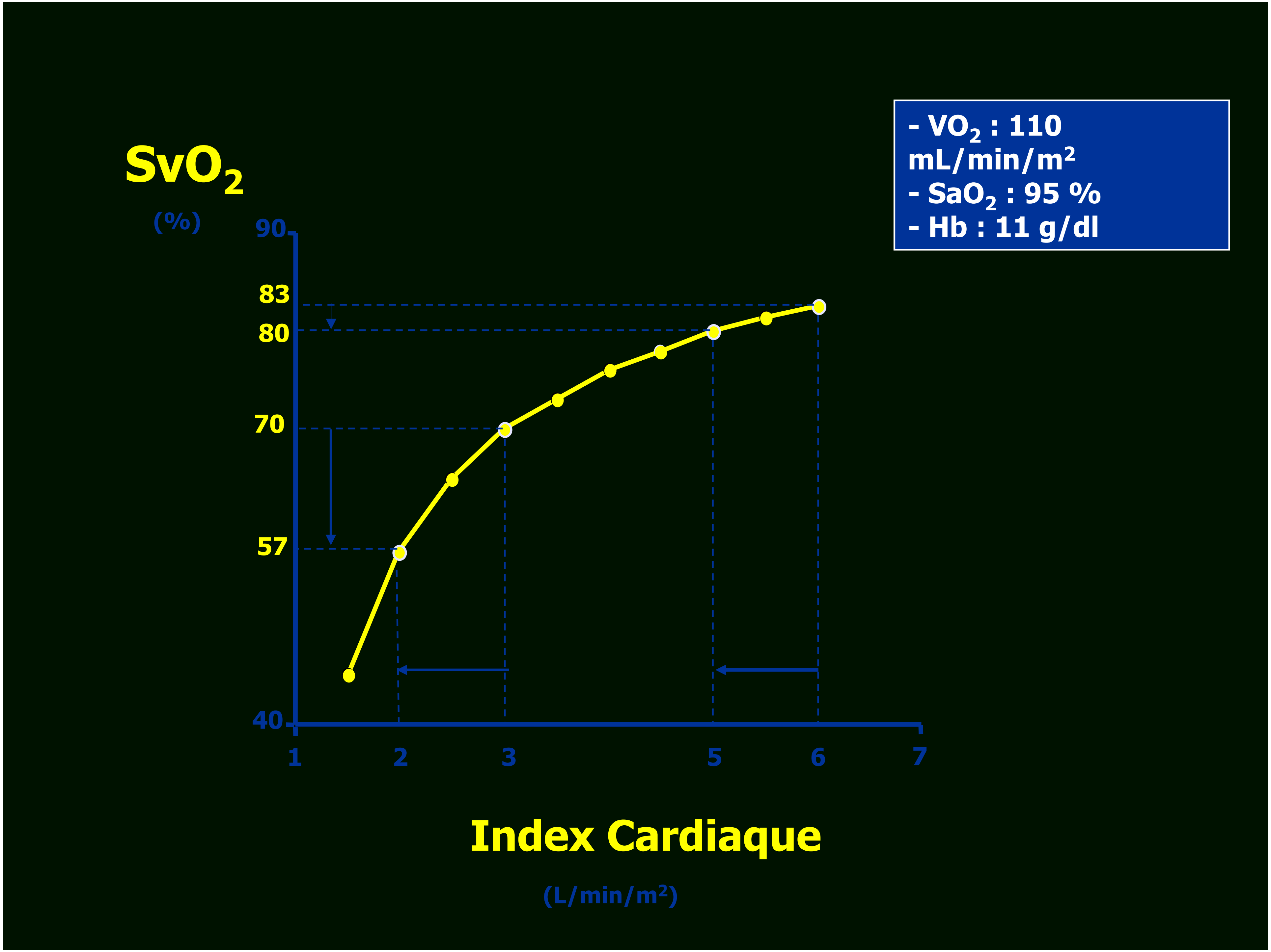
Facteurs susceptibles d'influencer la valeur de la SvO_2



Inégalité de variabilité des 4 déterminants de la SvO_2

- L'étendue numérique des variations de chaque déterminants détermine la variation numérique de la SvO_2
- Dans un orde décroissant, VO_2 ou IC (déterminants majeurs), puis Hb puis SaO_2 (déterminants mineurs)

La mesure continue de la SvO_2 reflétera mieux les variations des déterminants « majeurs » : IC et VO_2



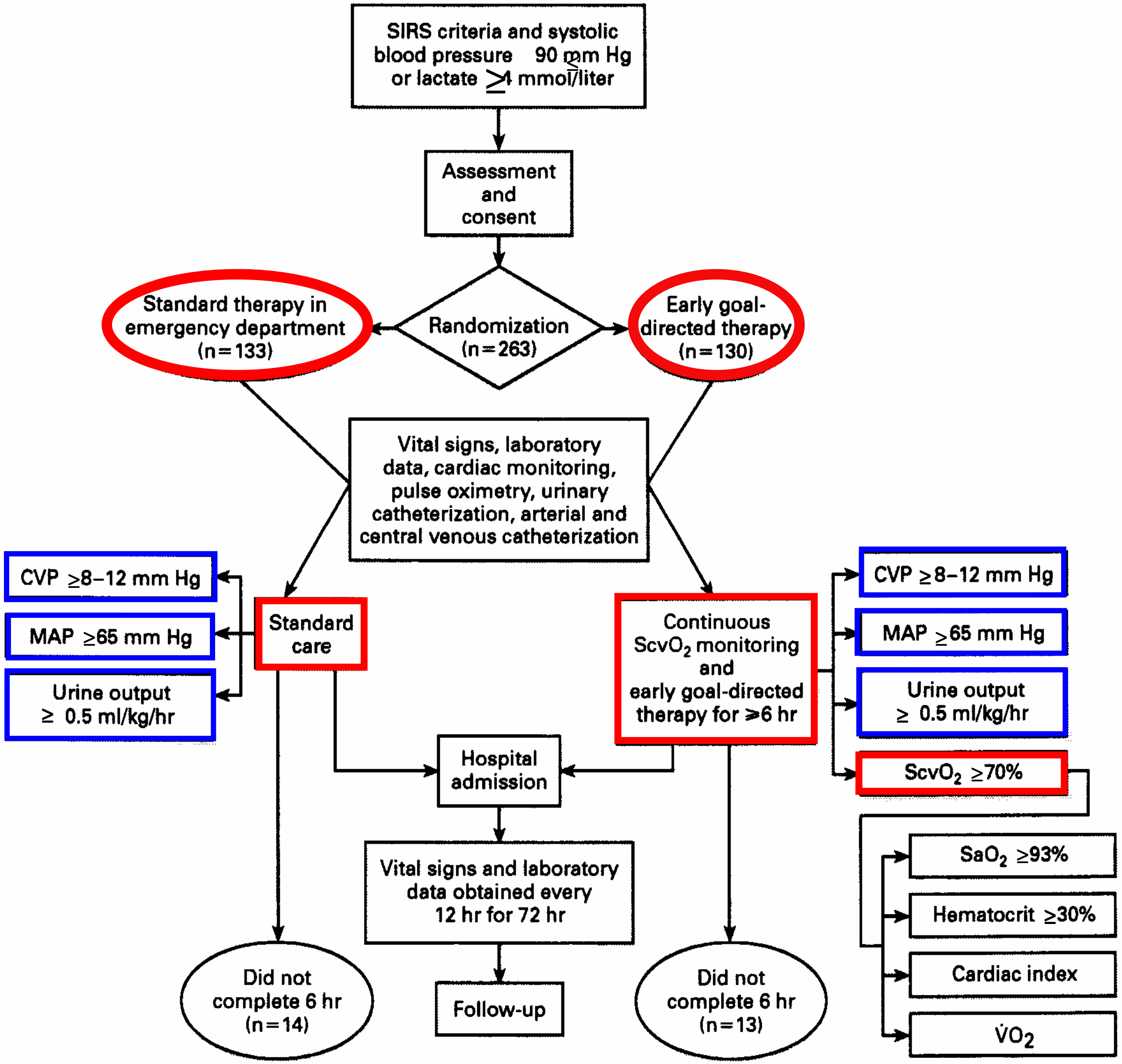
SvO2
(%)
90
83
80
70
57
40
1
2
3
5
6
7
Index Cardiaque
(L/min/m²)
- VO2 : 110 mL/min/m²
- SaO2 : 95 %
- Hb : 11 g/dl

The New England Journal of Medicine

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*

Rivers et al. *N Engl J Med* 2001;345:1368-77



Rivers NEJM 2001

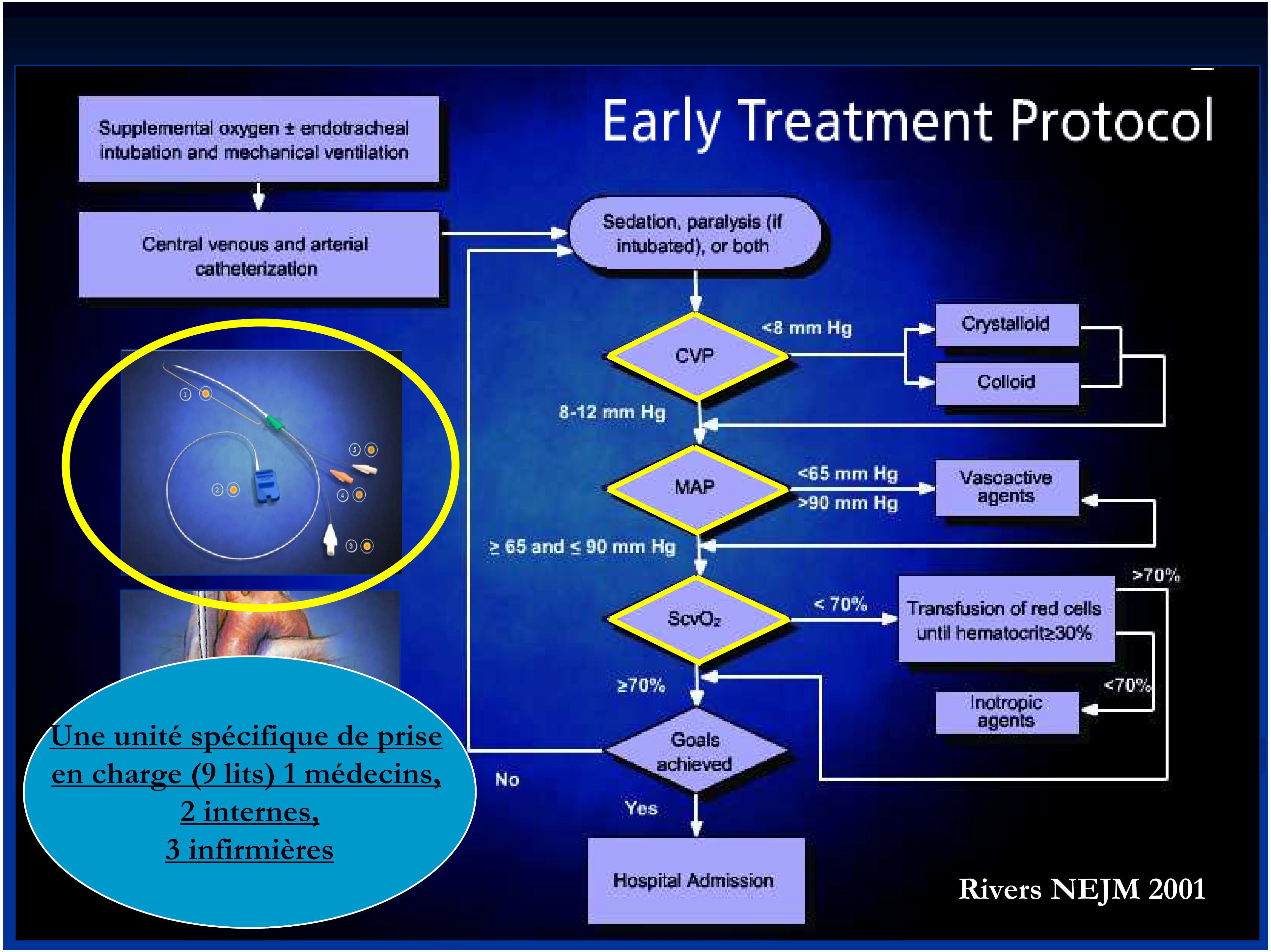

Early Treatment Protocol
Supplemental oxygen ± endotracheal intubation and mechanical ventilation
Central venous and arterial catheterization
Sedation, paralysis (if intubated), or both
CVP
<8 mm Hg
Crystalloid
Colloid
8-12 mm Hg
MAP
<65 mm Hg
>90 mm Hg
Vasoactive agents
≥ 65 and ≤ 90 mm Hg
ScvO2
< 70%
Transfusion of red cells until hematocrit≥30%
>70%
<70%
Inotropic agents
≥70%
Goals achieved
No
Yes
Hospital Admission
Une unité spécifique de prise en charge (9 lits) 1 médecins, 2 internes, 3 infirmières
Rivers NEJM 2001

TABLE 3. KAPLAN–MEIER ESTIMATES OF MORTALITY AND CAUSES OF IN-HOSPITAL DEATH.*

VARIABLE	STANDARD THERAPY (N=133)	EARLY GOAL-DIRECTED THERAPY (N=130)	RELATIVE RISK (95% CI)	P VALUE
	no. (%)			
In-hospital mortality†				
All patients	59 (46.5)	38 (30.5)	0.58 (0.38–0.87)	0.009
Patients with severe sepsis	19 (30.0)	9 (14.9)	0.46 (0.21–1.03)	0.06
Patients with septic shock	40 (56.8)	29 (42.3)	0.60 (0.36–0.98)	0.04
Patients with sepsis syndrome	44 (45.4)	35 (35.1)	0.66 (0.42–1.04)	0.07
28-Day mortality†	61 (49.2)	40 (33.3)	0.58 (0.39–0.87)	0.01
60-Day mortality†	70 (56.9)	50 (44.3)	0.67 (0.46–0.96)	0.03
Causes of in-hospital death‡				
Sudden cardiovascular collapse	25/119 (21.0)	12/117 (10.3)	—	0.02
Multiorgan failure	26/119 (21.8)	19/117 (16.2)	—	0.27

Rivers NEJM 2001

EARLY GOAL-DIRECTED THERAPY IN THE TREATMENT OF SEVERE SEPSIS AND SEPTIC SHOCK

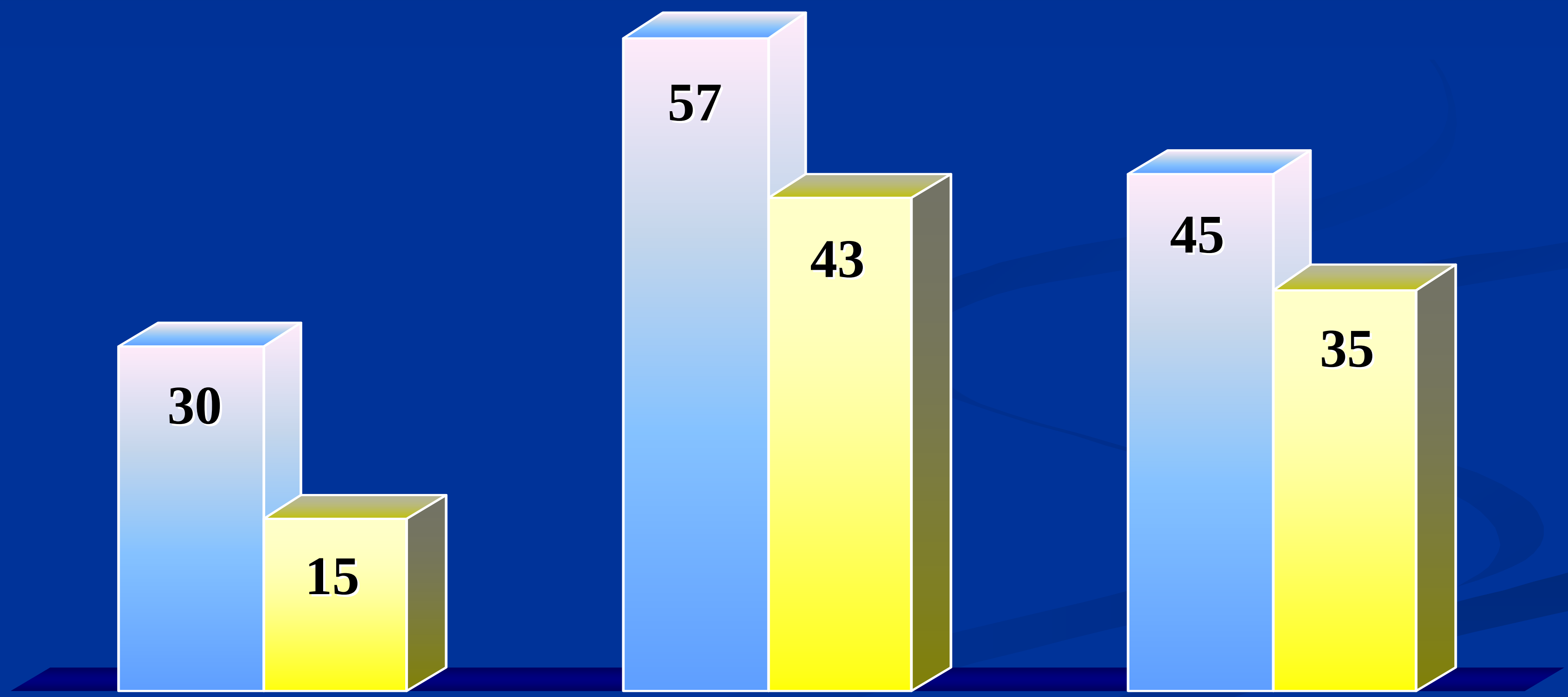
EMANUEL RIVERS, M.D., M.P.H., BRYANT NGUYEN, M.D., SUZANNE HAVSTAD, M.A., JULIE RESSLER, B.S., ALEXANDRIA MUZZIN, B.S., BERNHARD KNOBLICH, M.D., EDWARD PETERSON, PH.D., AND MICHAEL TOMLANOVICH, M.D., FOR THE EARLY GOAL-DIRECTED THERAPY COLLABORATIVE GROUP*

N Engl J Med 2001;345:1368-77

CONTROL

EGDT

In-hospital mortality



Severe sepsis | Septic shock | Sepsis syndrome

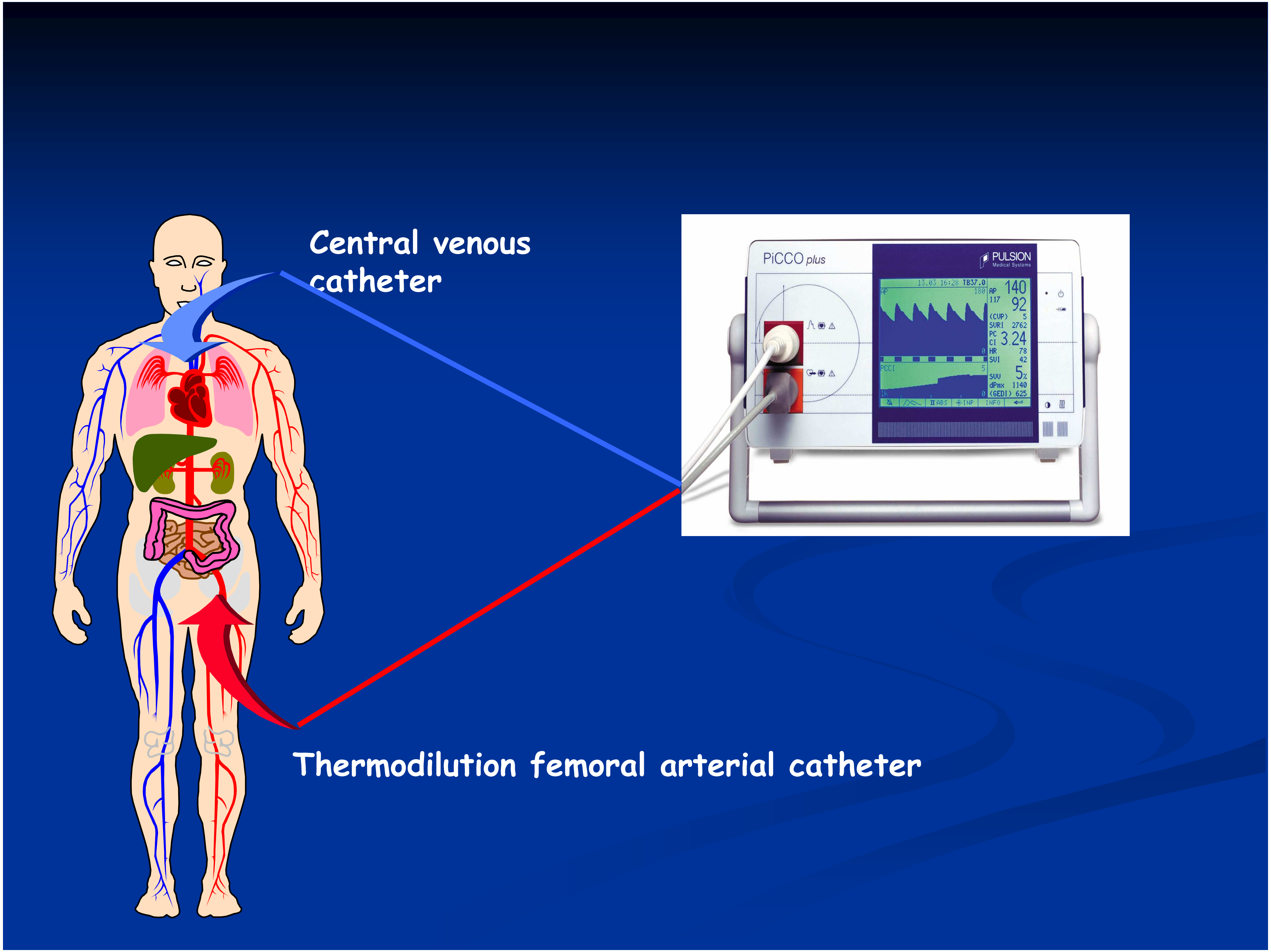
p value 0.06 | 0.04 | 0.07

PiCCO

Accuracy of the double indicator method for measurement of extravascular lung water depends on the type of acute lung injury*

Antoine Roch, MD; Pierre Michelet, MD; Dominique Lambert, MD; Stéphane Delliaux, MD; Christophe Saby, MD; Gilles Perrin, MD; Olivier Ghez, MD; Fabienne Bregeon, MD; Pascal Thomas, MD, PhD; Jean-Pierre Carpentier, MD; Laurent Papazian, MD, PhD; Jean-Pierre Auffray, MD

Conclusions: The double indicator method is useful for evaluation of pulmonary edema in indirect lung injury, as induced by oleic acid, but produces misleading values in direct lung injury, as produced by hydrochloric instillation. (Crit Care Med 2004; 32:811–817)


Central venous
catheter
Thermodilution femoral arterial catheter
PiCCO plus
PULSION

INTRODUCTION

Pulse Contour Cardiac Output

- 2 conditions:
  - un cathéter veineux central
  - un cathéter artériel

- 2 principes:
  - thermodilution transpulmonaire
  - analyse de contour de pouls

PRINCIPES

et paramètres obtenus

1. **<u>Méthode de thermodilution transpulmonaire:</u>** **mesures discontinues**
   - débit cardiaque
   - volume télédiastolique global VTDG
   - volume sanguin intrathoracique VSIT
   - eau pulmonaire extravasculaire EPEV
2. **<u>Analyse du contour de pouls:</u>** **mesures continues**
   - volume d'éjection
   - débit cardiaque
   - variation du volume d'éjection VVE
   - variation de la pression pulsée VPP
   - résistances vasculaires systémiques RVS

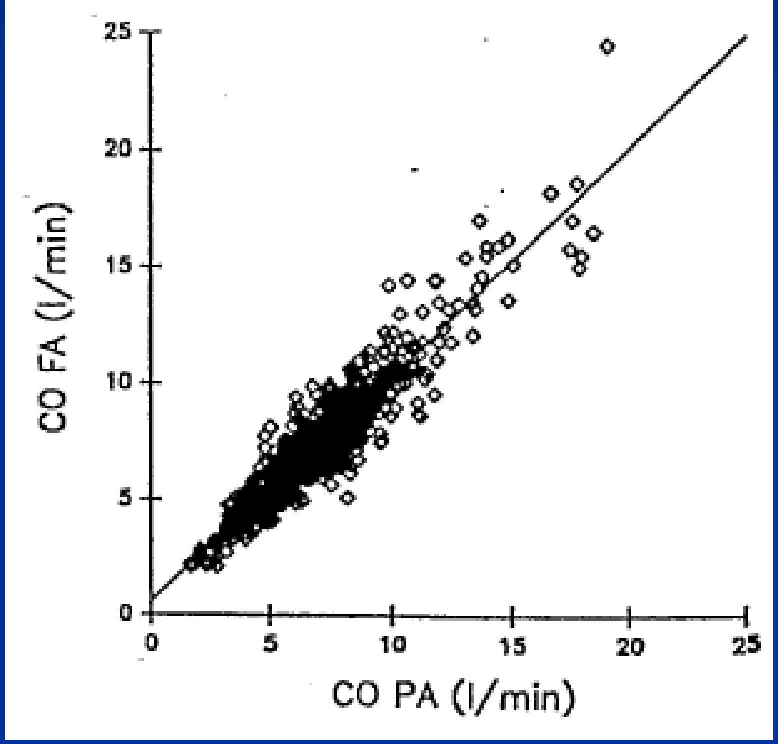
NORMES

Thermodilution transpulmonaire	
Débit cardiaque indexé	3.5 - 5.0 l/min/m²
VSIT	850 - 1000 ml/m²
VTDG	600 - 800 ml/m²
EPEV indexée	3.0 - 7.0 ml/m²
IFC	4.5 - 6.5 1/min
FEG	25 – 35 %
Analyse du contour de pouls	
VE indexé	40 - 60 ml/m²
VVE	< 10%
VPP	< 10%
RVSI	1200 - 2000 dyn.s.cm-5 m²

ETUDES DE VALIDATION

1. Débit cardiaque par thermodilution transpulmonaire

Débit cardiaque par thermodilution transpulmonaire *versus* thermodilution artérielle pulmonaire(1)



- **804 mesures chez 48 patients**

Biais: surestimation du DC de 7%

r = 0.94

Böck JC, *Cardiac output measurement using femoral artery thermodilution in patients*

J Critical care 1989, vol 4, No 2, June, p106-111

Débit cardiaque par thermodilution transpulmonaire *versus* thermodilution artérielle pulmonaire (2)

			biais	Intervalle de confiance	Cœfficient de corrélation
Acta Ped 1996	Mac Luckie A	9 enfants COLD	0.19l/min/m² (4.4%)		
Chest 1998	Godje O	150/30 COLD	0.16 l/min/m² (2.4%)	[-0.44; 0.79]	0.96
Intensive Care Med 1998	Perel A	123/12 PiCCO axillaire	0.47 ± 0.72 l/min		0.89
Intensive Care Med 1999	Sakka SG	449/37 COLD	0.68 ± 0.62 l/min	[0.06; 1.3]	0.97
Chest 2000	Goedje O	960/40 COLD	0.35 l/min/m²	[-0.06; 0.75]	0.98
Crit Care 2000	Bindels A	154/45 COLD	0.49 l/min/m²	[0.45; 0.53]	0.95
J.C.V.Anesth 2000	Sakka SG	51/12 COLD	0.73 ± 0.38 l/min		0.98

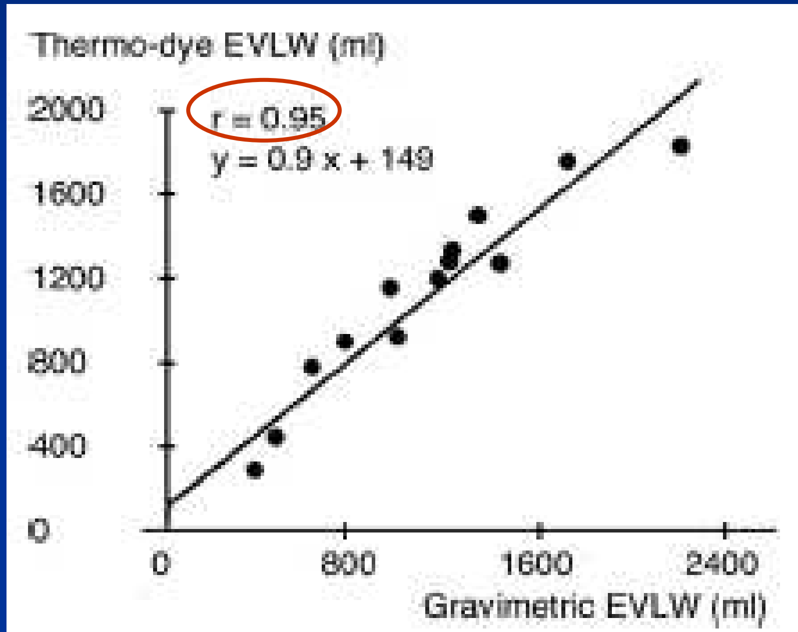
ETUDES DE VALIDATION

2. Eau pulmonaire extravasculaire par thermodilution transpulmonaire

Études expérimentales: EPEV par thermodilution transpulmonaire *versus* gravimétrie

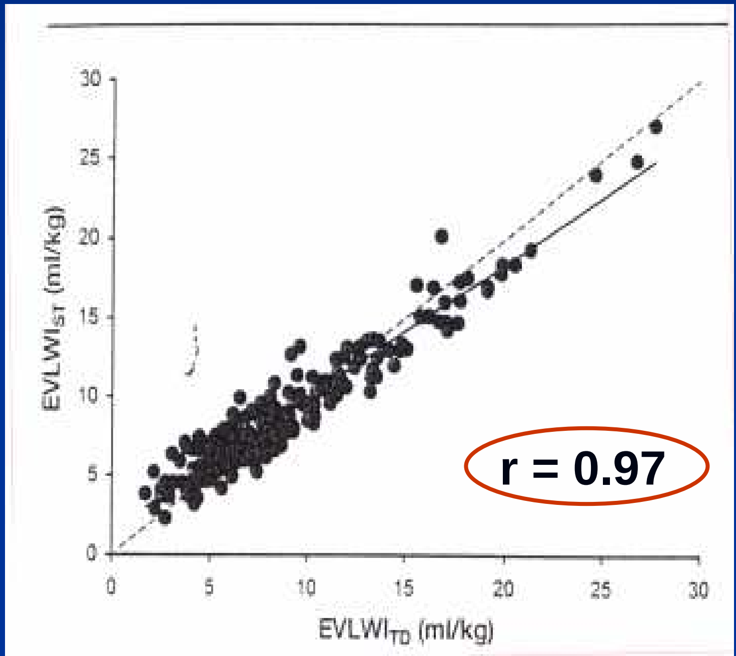
	auteurs	Modèle expérimental	Biais	Corrélation
Crit care Med 2004	Katzenelson	Acide oléique ou élévation de la pression hydrostatique	3.01 ± 1.34 ml/kg	r = 0.967
Crit care Med 2004	Kirov	LPS IV Acide oléique	4.9 ± 5.1 ml/kg	r = 0.85
Crit Care Med 2006	Rossi	Endotoxine IV	5.40 ml/kg	r = 0.94

Études cliniques: EPEV par double-dilution transpulmonaire *versus* gravimétrie



Sturm JA, *Development and significance of lung water measurement in clinical and experimental practice.*
Pratical Applications of Fiberoptics in Critical Care Monitoring 1990

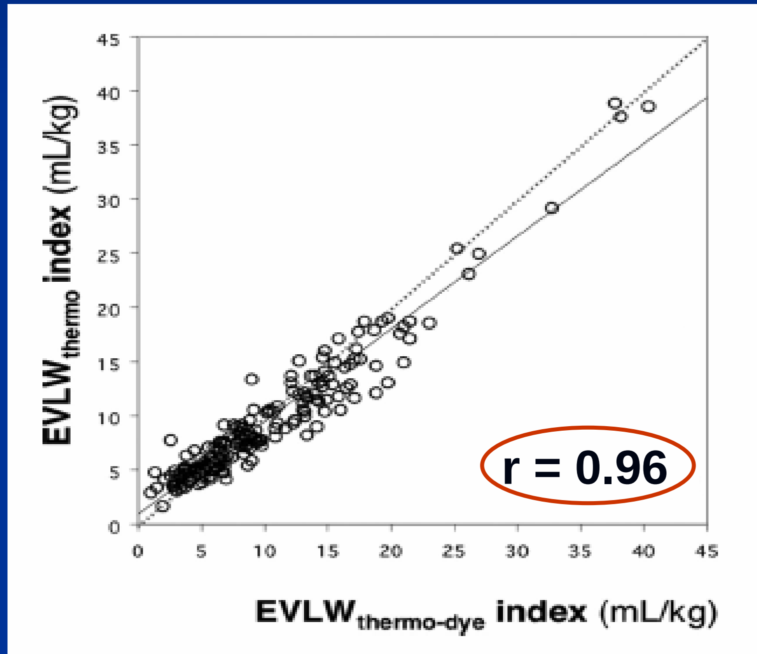
Études cliniques: EPEV par thermodilution transpulmonaire *versus* double-dilution transpulmonaire



209 patients

Biais = -0.2 ± 1.4 ml/kg

Sakka SG Intensive Care Med. 2000 Feb;26(2):180-7.



192 mesures chez 48 patients en post-opératoire

Biais = -0.5 ± 1.9ml/kg

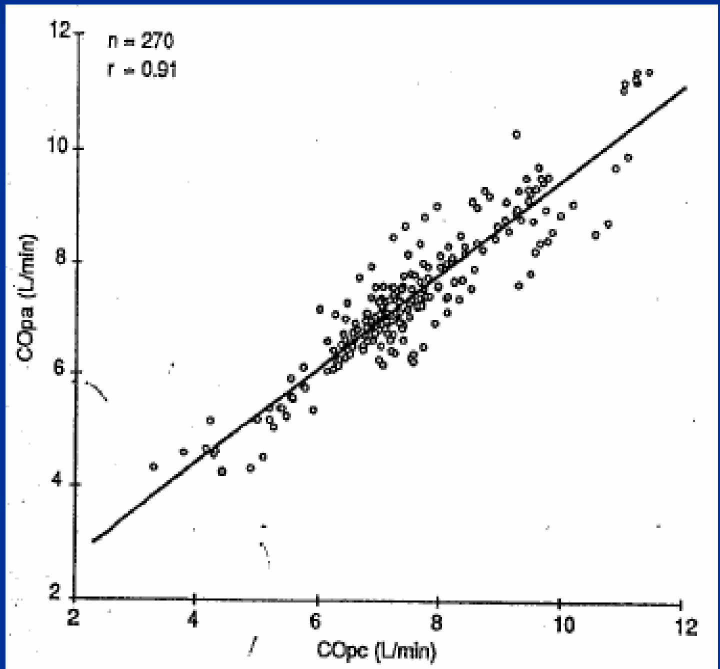
Michard F Crit Care Med. 2005 Jun;33(6):1243-7

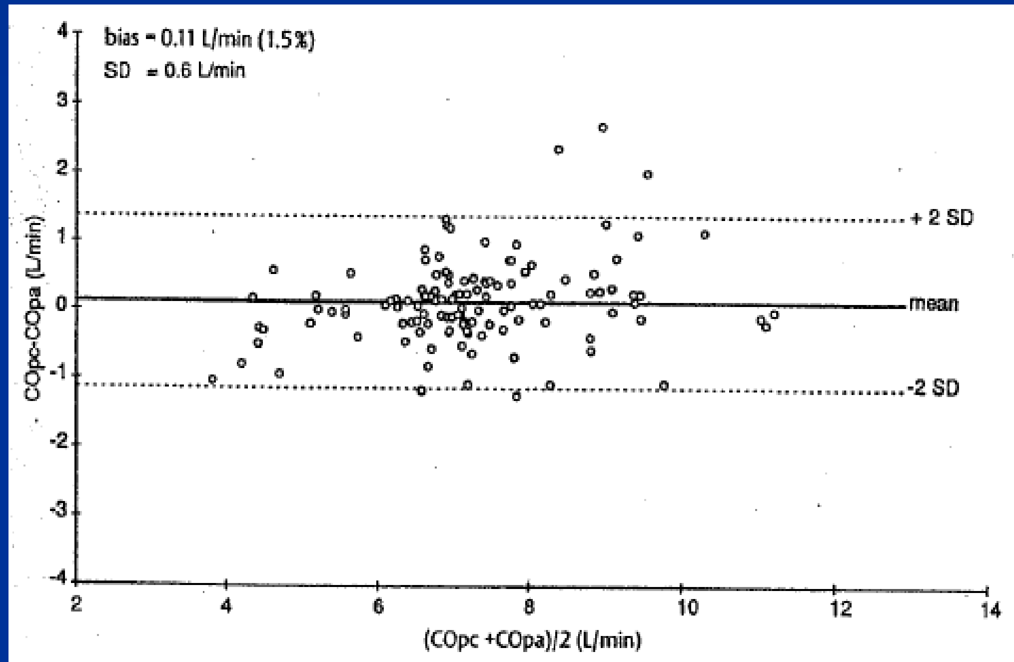
ETUDES DE VALIDATION

3. Débit cardiaque par analyse du contour de pouls

Débit cardiaque par contour de pouls *versus* thermodilution artérielle pulmonaire(1)

9 triples mesures chez 30 patients





Godje O, Thorac. Cardiovasc. Surgeon, 1998 (46), p242-249

Débit cardiaque par contour de pouls *versus* thermodilution artérielle pulmonaire (2)

			Biais (facteur de corrélation)
Thorac Cardiovasc Surg 1998	Godje O	270/30	0.11 ± 0.6 L/min (r = 0.91)
JCVA 1999	Buhre W	36/12	0.27 ± 1.16 L/min (r = 0.90)
Crit Care Med 1999	Goedje O	216/34	0.07 ± 1.4 L/min
Transp Proc 2003	Tzenkov IC	314/35	0.18 L/min

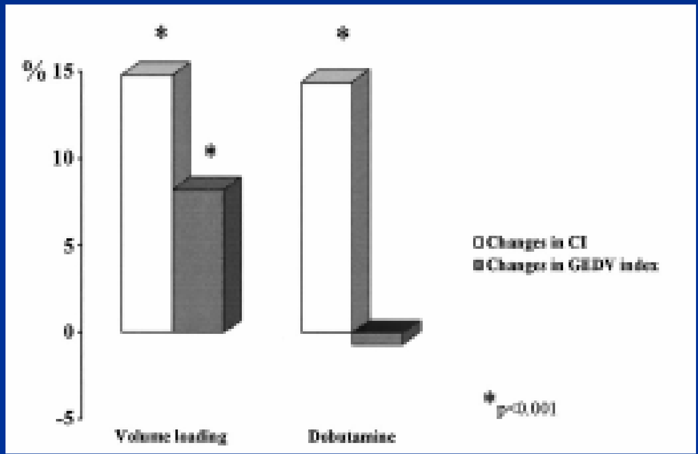
Débit cardiaque par contour de pouls: dérive au cours du temps(2)

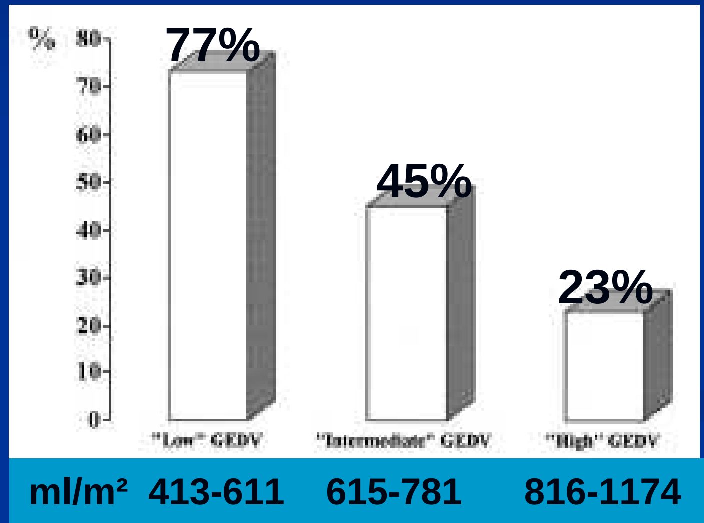
			Biais (facteur de corrélation)
Thorac Cardiovasc Surg 1998	Godje O	9/30 24h 1 calibration	0.15 ± 0.7 L/min (r = 0.90)
JCVA 1999	Buhre W	12 pré, per et post-opératoire 1 calibration	0.27 ± 1.16 L/min (r = 0.90)
Crit Care Med 1999	Godje O	34 h/24h	0.22 ± 1.58 L/min
JTCS 2007	Fakler U	7/24 enfants 24h	0.05 ± 0.4 L/min/m² (r = 0.93)

INTERET ET APPLICATIONS CLINIQUES

1. VTDG comme indicateur de précharge cardiaque

36 patients en choc septique





Corrélation △VTDG/△IC = 0.67

Réponse positive si augmentation du VE ≥15%

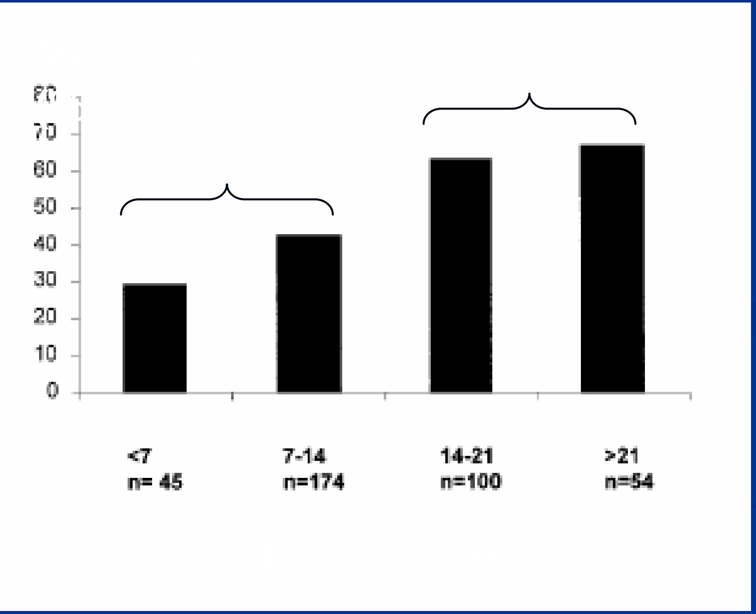
VTDG < 500ml/m²: 100%, <550: 89%, >950: 0%

Michard F, Alaya S, Zarka V, Bahloul M, Richard C, Teboul JL. ***Global end-diastolic volume as an indicator of cardiac preload in patients with septic shock.***
Chest. 2003 Nov;124(5):1900-8

2. EPEV: valeur pronostic

Étude rétrospective, n = 373 (1996-2000)

COLD système



< 10: mortalité = 33%

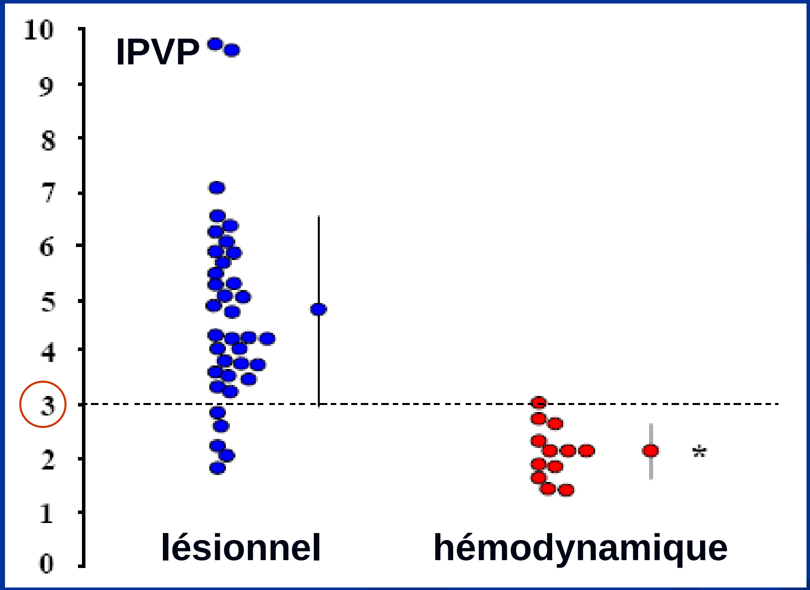
> 15: mortalité = 65%

Sakka SG. *Prognostic value of extravascular lung water in critically ill patients.* Chest. 2002 Dec;122(6):2080-6.

2. EPEV: diagnostic de l'œdème pulmonaire cardiogénique *versus* lésionnel

Index de perméabilité vasculaire pulmonaire: **IPVP = EPEV / VSP**

	ALI/SDRA	hydrostatique
n	36	12
FeVG (%)	51 ± 8	42 ± 8
BNP (pg/ml)	122 - 514	554 - 3057
IC (L/min/m²)	3.7 ± 1.0	4.2 ± 0.9
EPEVi (ml/kg)	22 ± 9	16 ± 4
IPVP	4.8 ± 1.7	2.1 ± 0.5



- **IPVP: Se = 95%; Sp = 100%**

Monnet X, Anguel N, Osman D, Hamzaoui O, Richard C, Teboul JL. Intensive Care Med. 2007 Mar;33(3):448-53. 2007 Jan 13

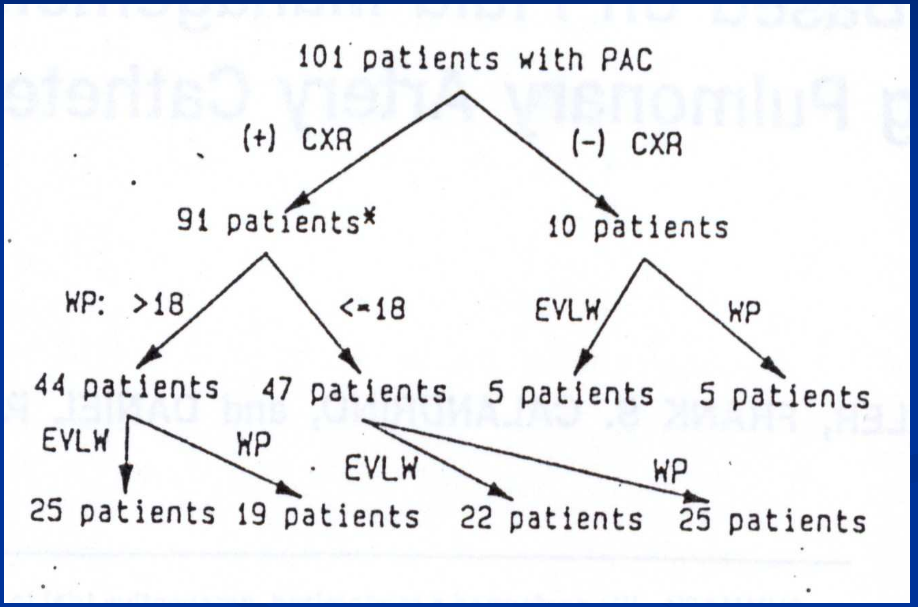
3. EPEV: prise en charge de l 'œdème pulmonaire

mesure par thermodilution artérielle pulmonaire

91 patients avec un syndrome instertitiel et/ou alvéolaire bilatéral

- groupe EPEV: remplissage jusqu'à EPEV > 7ml/kg, PAPO < 25mmHg et PaO2 maintenue

- groupe PAPO: remplissage pour PAPO = 18mmHg



➢ **diminution de la durée de la ventilation mécanique (9 *vs* 22j, *p = 0.047*), de la durée d'hospitalisation en soins intensifs (7 *vs* 16j, *p = 0.05*) et de la mortalité (35 *vs* 47%, *ns*)**

Mitchell JP et al, *Improved outcome based on fluid management in critically ill patients requiring pulmonary artery catheterisation*

Am Rev Respir Dis 1992; 145:990-998

3. VVE et prédiction de la réponse au remplissage

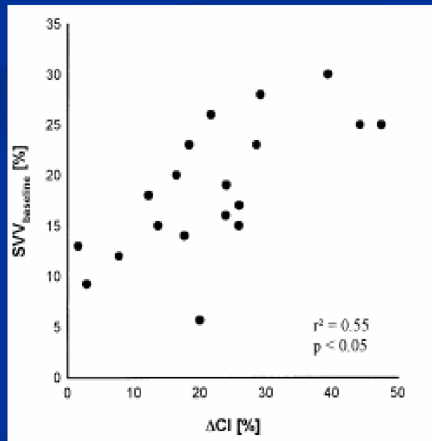
Expansion volémique

	0 min	3 min	6 min	25 min	28 min	31 min
HR (beats min^{-1})	89±10	90±10	89±10	90±9	89±9	89±9
MAP (mmHg)	78±11	78±11	78±10	90±12*	89±11*	87±12*
SAP (mmHg)	123±17	122±17	117±29	142±14*	141±14*	139±15*
SVRI (dyne s^{-1} cm^{-5})	1717±571	1717±576	1728±568	1596±529	1562±531	1590±541
CI_{td} (l min^{-1} m^{-2})	3.6±1	3.6±0.9	3.5±0.9	4.3±0.9*	4.3±0.9*	4.2±0.9*
SVV (%)	19.2±6.4	20.8±5.9	19.8±5.4	9.4±3.5*	9.2±3.1*	10.0±3.1*
CVP (mmHg)	6.6±2	6.8±2	7.0±2	11±3*	10.4±3*	10.2±2*
PAOP (mmHg)	5.9±2	6.3±2	6.6±3	11±5*	10.6±5*	11.0±5*
ITBVI (ml m^{-2})	860±201	848±198	852±212	919±182*	924±191*	908±193*
LVEDAI (cm^2 m^{-2})	7.9±1.9	7.9±2.1	7.9±1.9	9.7±2.4*	9.5±1.8*	9.5±1.9*
LVEFA (%)	0.69±0.15	0.70±0.16	0.70±0.16	0.68±0.15	0.67±0.15	0.68±0.15
Temperature (°C)	36.4±0.8	36.4±0.7	36.4±0.8	36.3±0.7	36.3±0.8	36.2±0.7

*$p<0.05$ vs. 0 min

20 patients en post-opératoire de chirurgie cardiaque

Ventilation: Vt = 14ml/kg, Ppic = 16.2cmH2O, Pep = 4cmH2O



r = 0.55

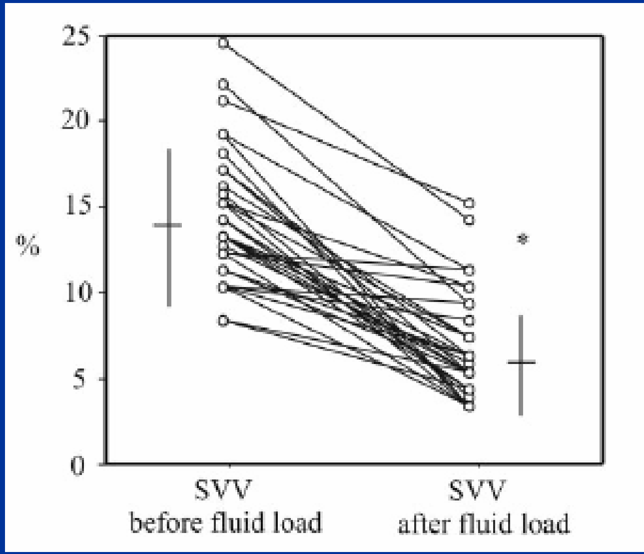
Reuter DA *Stroke volume variations for assessment of cardiac responsiveness to volume loading in mechanically ventilated patients after cardiac surgery.* Intensive Care Med. 2002 Apr;28(4):392-8. 2002 Mar 20

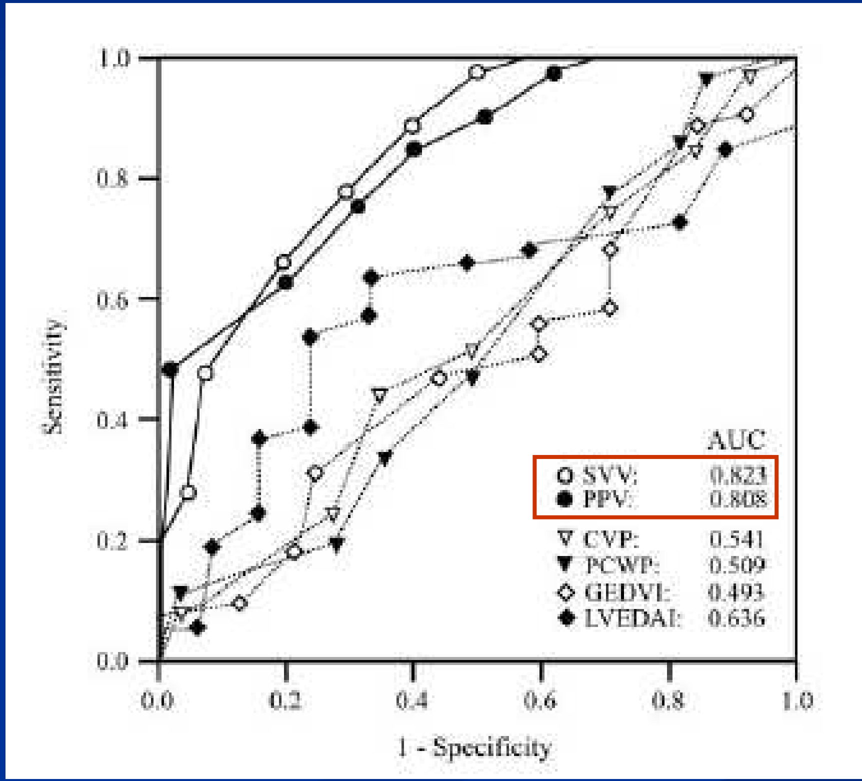
3. VVE et prédiction de la réponse au remplissage

n = 40 (3 mesures), critère de réponse positive: VEi ≥ 25%

- VVE > 12.5%: Se = 75%, Sp = 71%

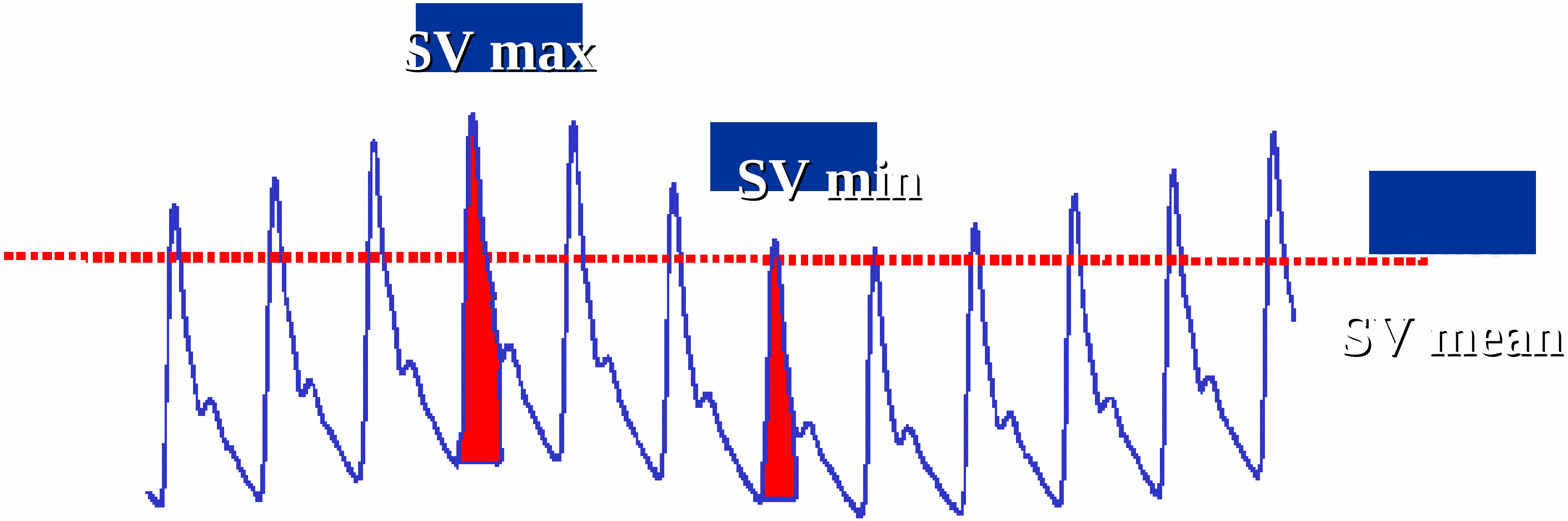
- VPP > 13.5%: Se = 72%; Sp = 72%





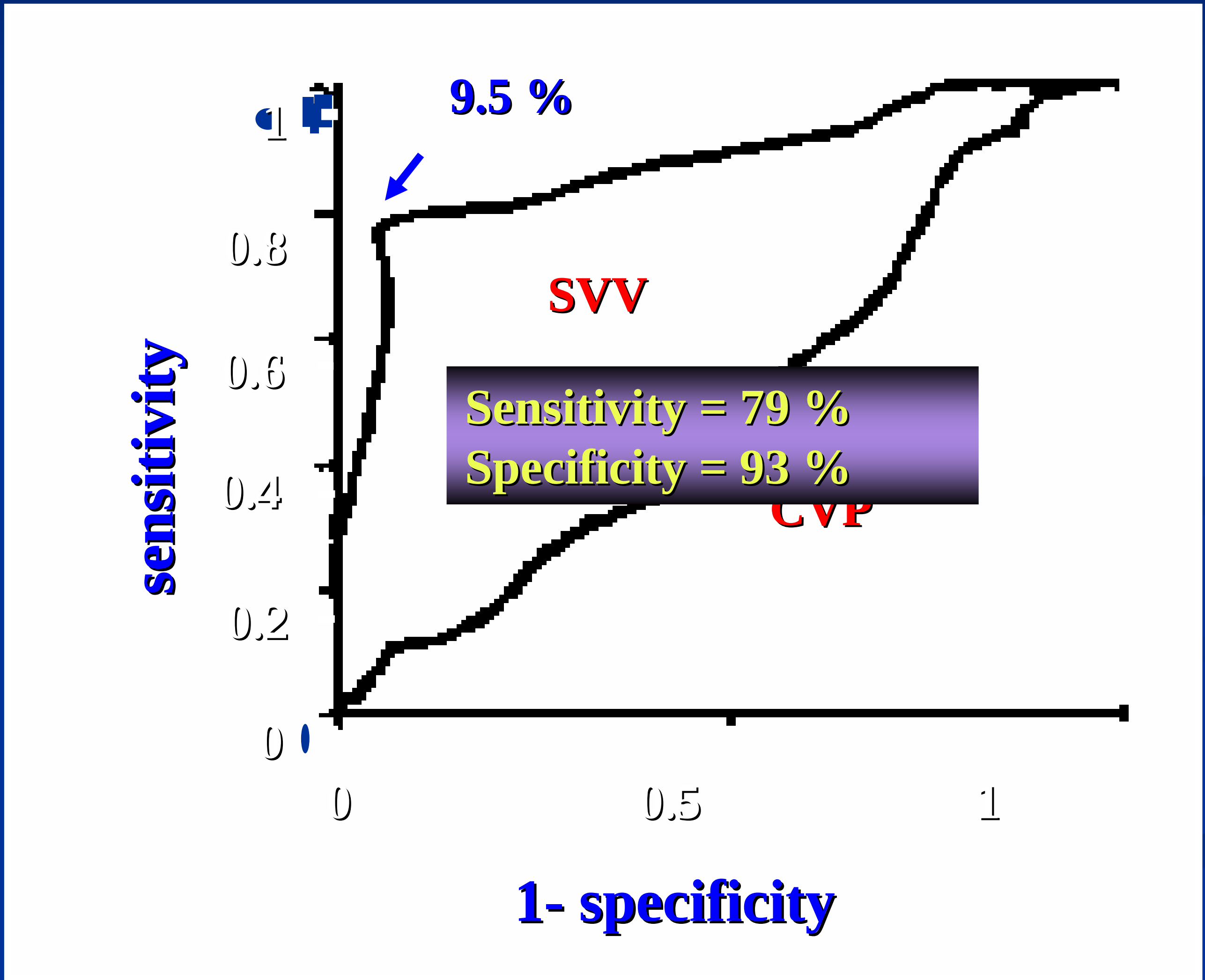
Hofer CK *Stroke volume and pulse pressure variation for prediction of fluid responsiveness in patients undergoing off-pump coronary artery bypass grafting.*
Chest. 2005 Aug;128(2):848-54

Stroke Volume Variation



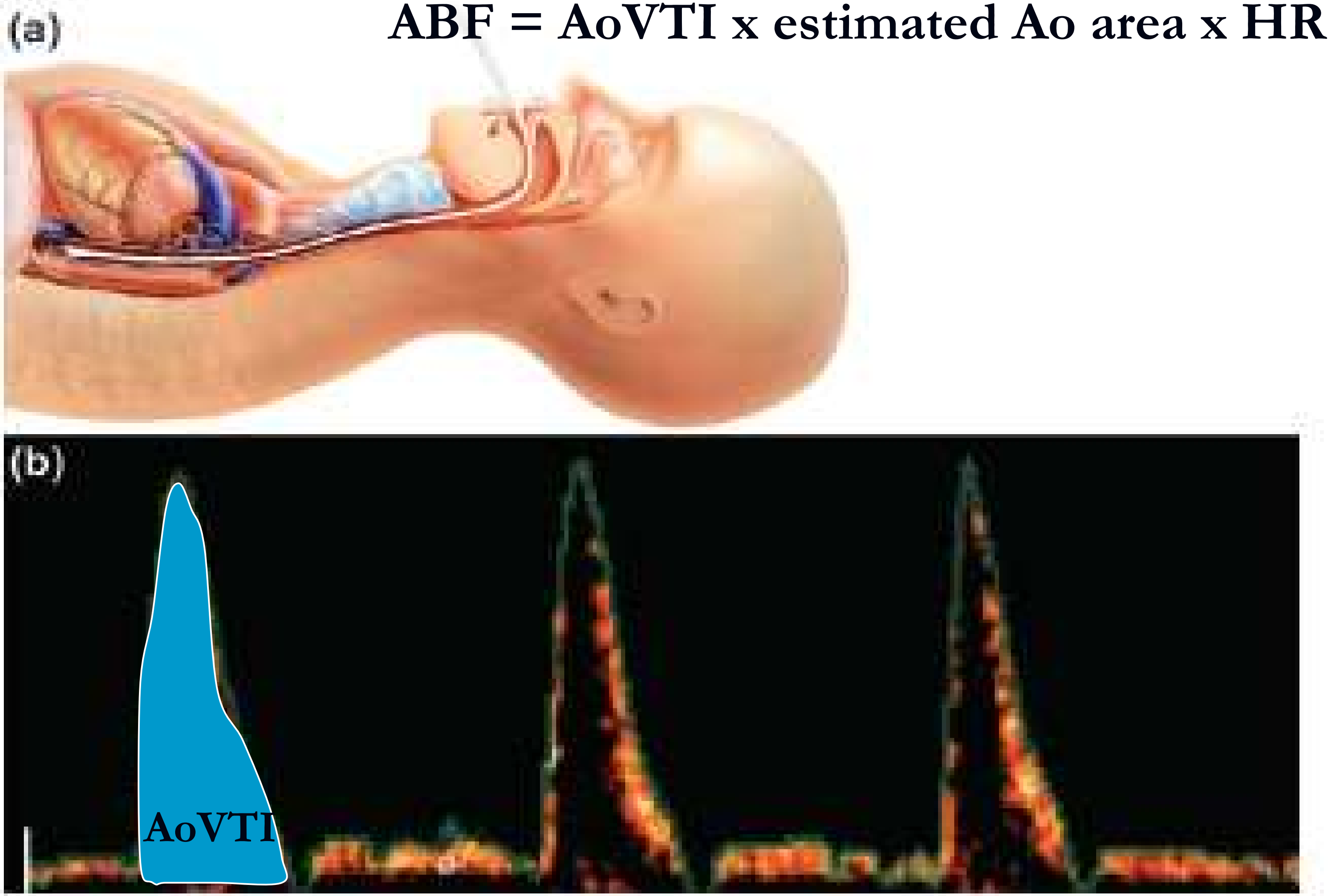
$$SVV = \frac{SV\ max + SV\ min}{2}$$

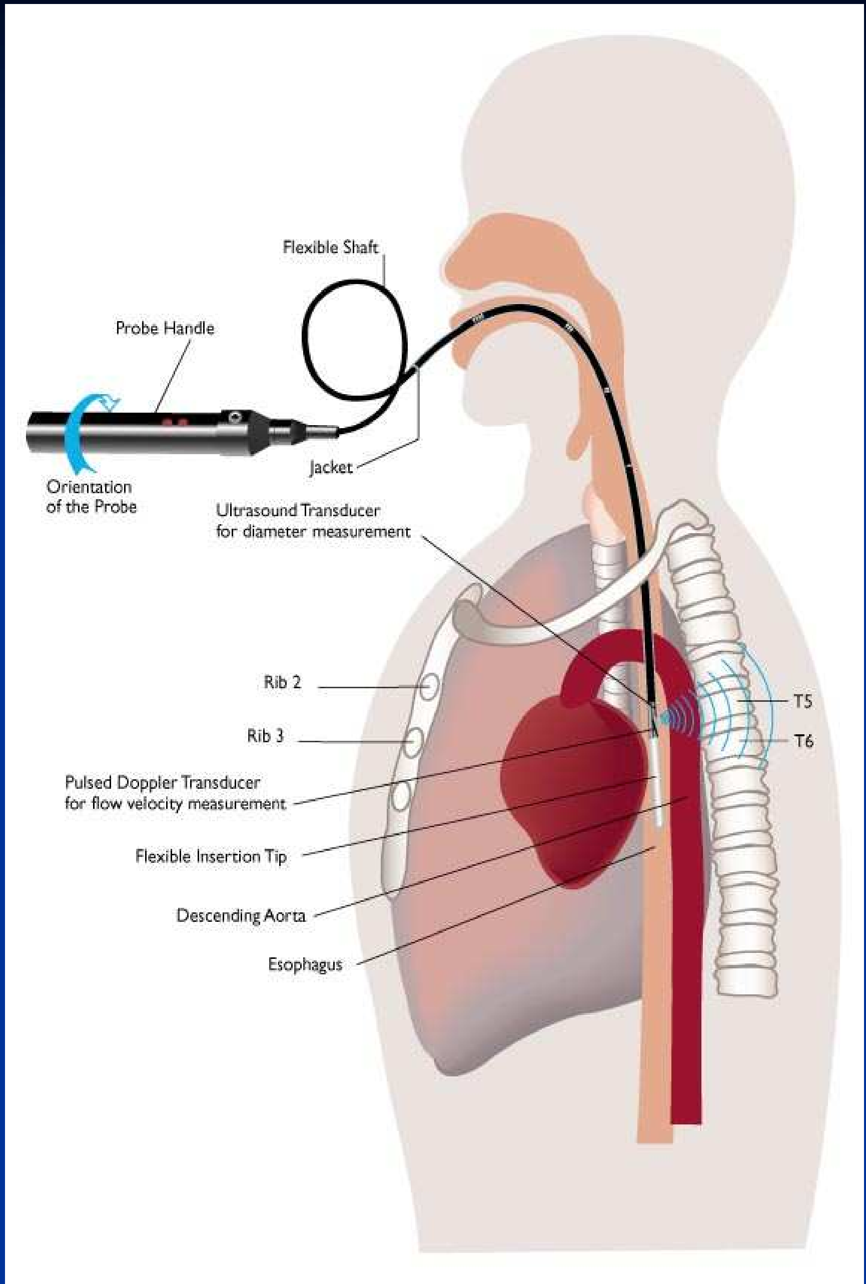
Stroke volume variation as a predictor of fluid responsiveness in patients undergoing brain surgery
Berkenstadt H. Margalit N, Hadani M, Friedman Z, Segal E, Vila Y, Perel A .
Anesth Analg 2001;92:984-9



Probe of 6mm or 7mm

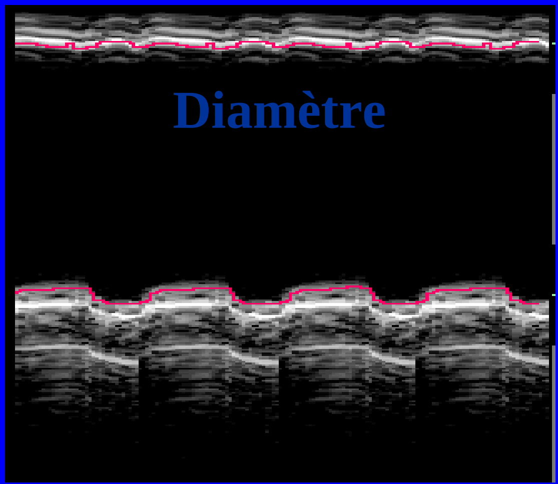
ABF = AoVTI x estimated Ao area x HR

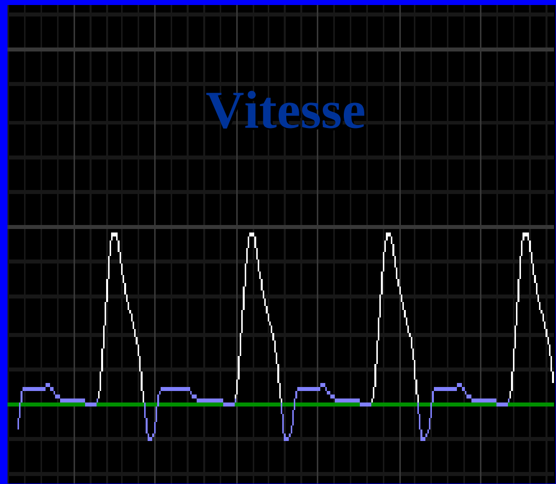



Flexible Shaft
Probe Handle
Jacket
Orientation
of the Probe
Ultrasound Transducer
for diameter measurement
Rib 2
Rib 3
T5
T6
Pulsed Doppler Transducer
for flow velocity measurement
Flexible Insertion Tip
Descending Aorta
Esophagus


HemoSonic100

Doppler trans-oesophagien



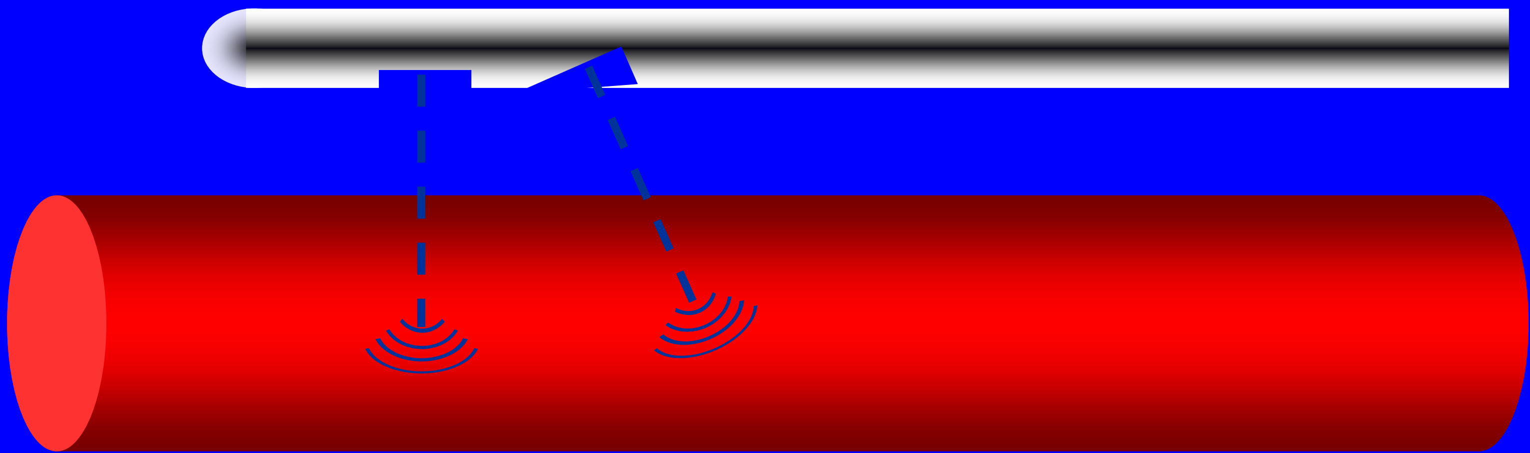


Débit aortique

DA = P D²/4 x V

3.2 l.min

HemoSonic100 Arrow®

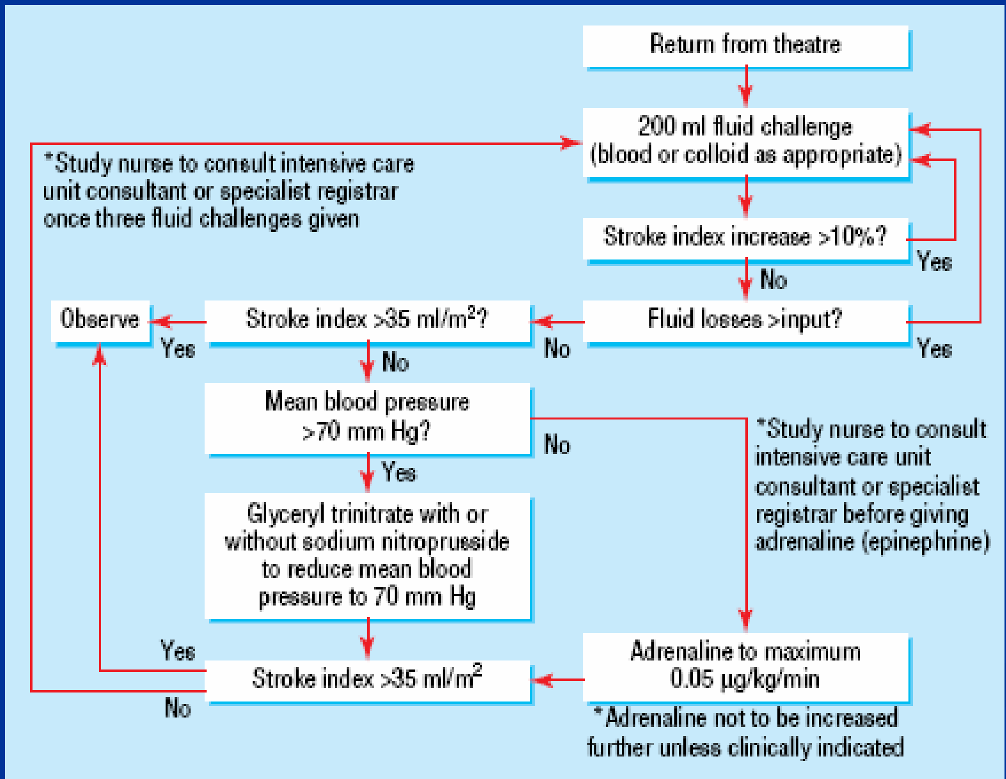


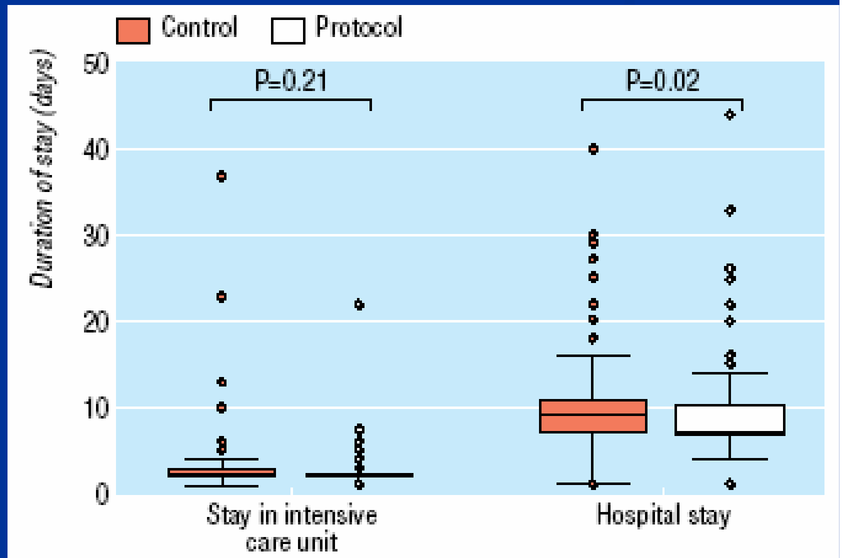
bmj.com

Randomised controlled trial assessing the impact of a nurse delivered, flow monitored protocol for optimisation of circulatory status after cardiac surgery

Moira McKendry, Helen McGloin, Debbie Saberi, Libby Caudwell, Anthony R Brady and Mervyn Singer

BMJ 2004;329;258-; originally published online 8 Jul 2004;
doi:10.1136/bmj.38156.767118.7C



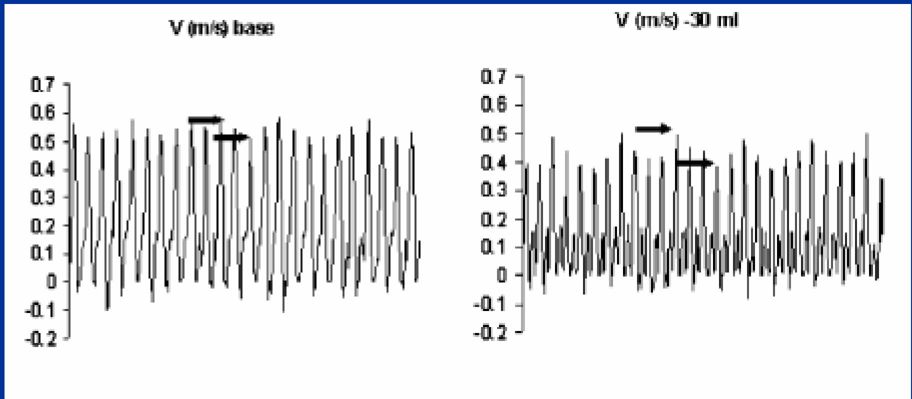


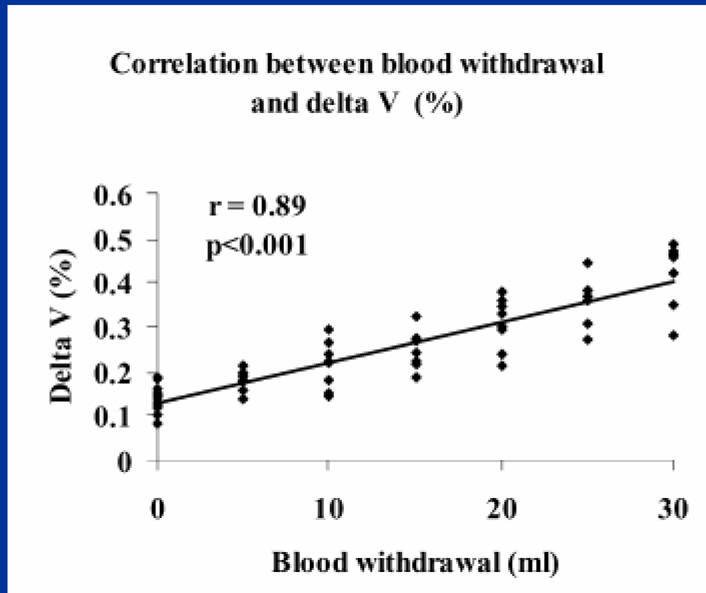
Intensive Care Med (2004) 30:1182–1187
DOI 10.1007/s00134-004-2190-z

EXPERIMENTAL

Monitoring of respiratory variations of aortic blood flow velocity using esophageal Doppler

Michel Slama
Henri Masson
Jean-Louis Teboul
Marie-Luce Arnould
Rachida Nait-Kaoudjt
Bouchra Colas
Marcel Peltier
Christophe Tribouilloy
Dinko Susic
Edward Frohlich
Michel Andréjak





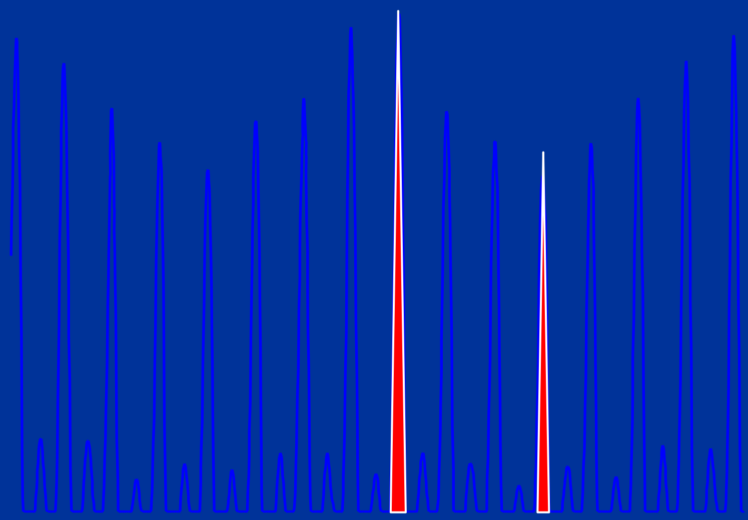
Intensive Care Med (2005) 31:1195–1201
DOI 10.1007/s00134-005-2731-0

ORIGINAL

Xavier Monnet
Mario Rienzo
David Osman
Nadia Anguel
Christian Richard
Michael R. Pinsky
Jean-Louis Teboul

Esophageal Doppler monitoring predicts fluid responsiveness in critically ill ventilated patients

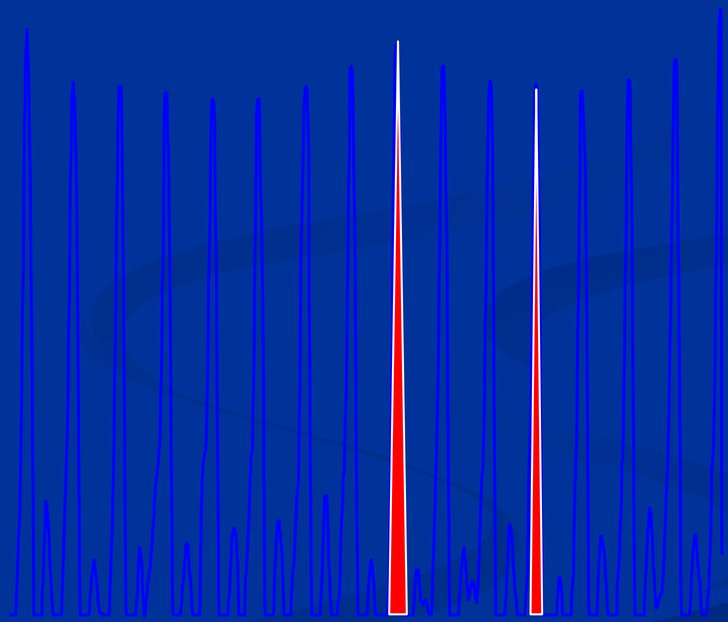
Base 2



Mean ABF = 2.30 L/min

ΔABF = 39%

Post VE



Mean ABF = 3.92 L/min

ΔABF = 13%

Intensive Care Med (2005) 31:1195–1201
DOI 10.1007/s00134-005-2731-0

ORIGINAL

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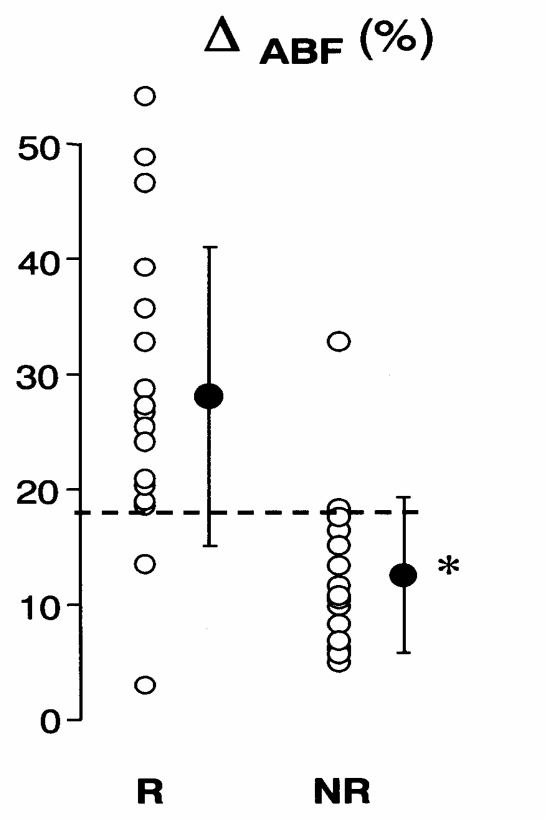
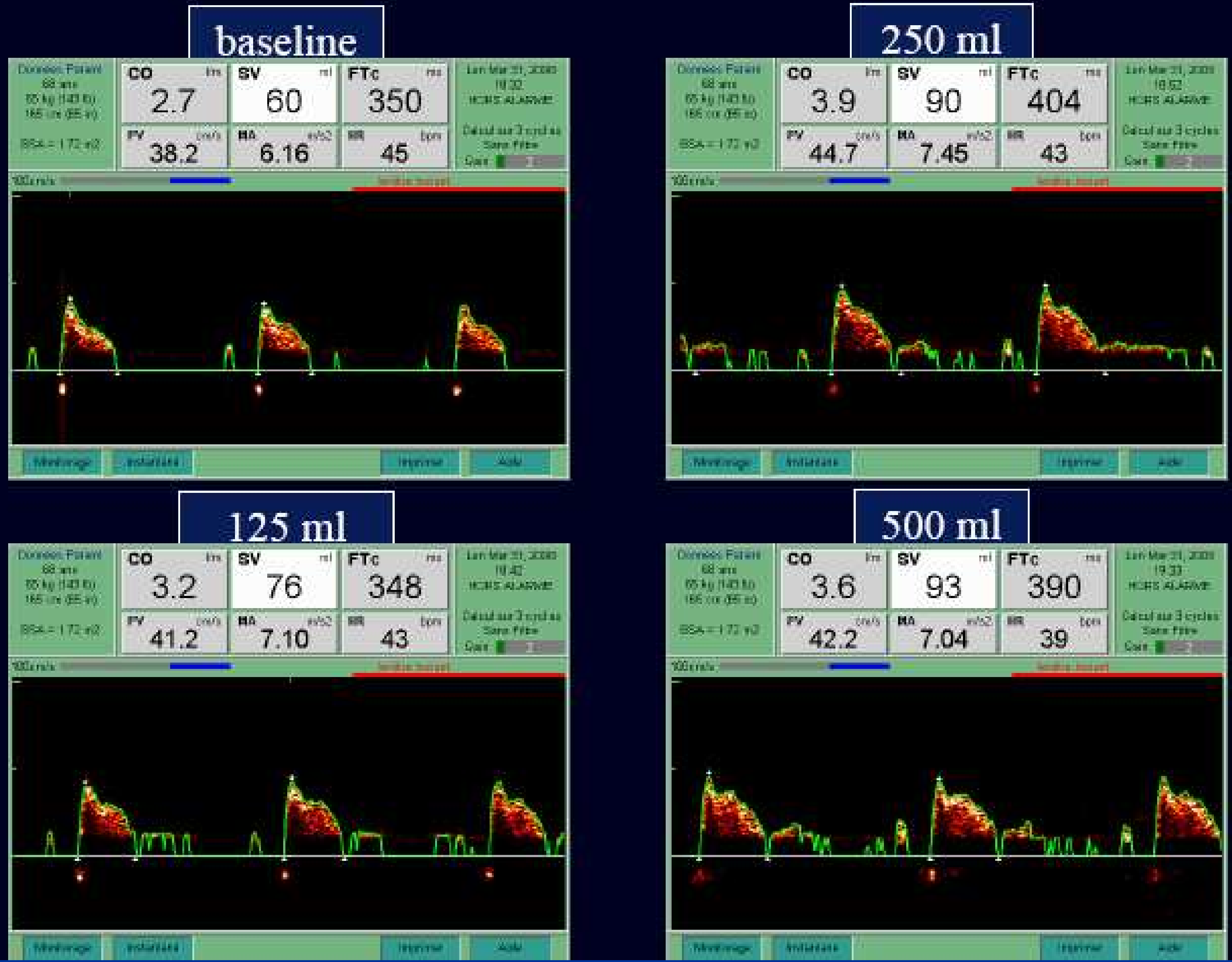
Esophageal Doppler monitoring predicts fluid responsiveness in critically ill ventilated patients




baseline
CO 2.7
SV 60
FTc 350
PV 38.2
MA 6.16
HR 45
250 ml
CO 3.9
SV 90
FTc 404
PV 44.7
MA 7.45
HR 43
125 ml
CO 3.2
SV 76
FTc 348
PV 41.2
MA 7.10
HR 43
500 ml
CO 3.6
SV 93
FTc 390
PV 42.2
MA 7.04
HR 39

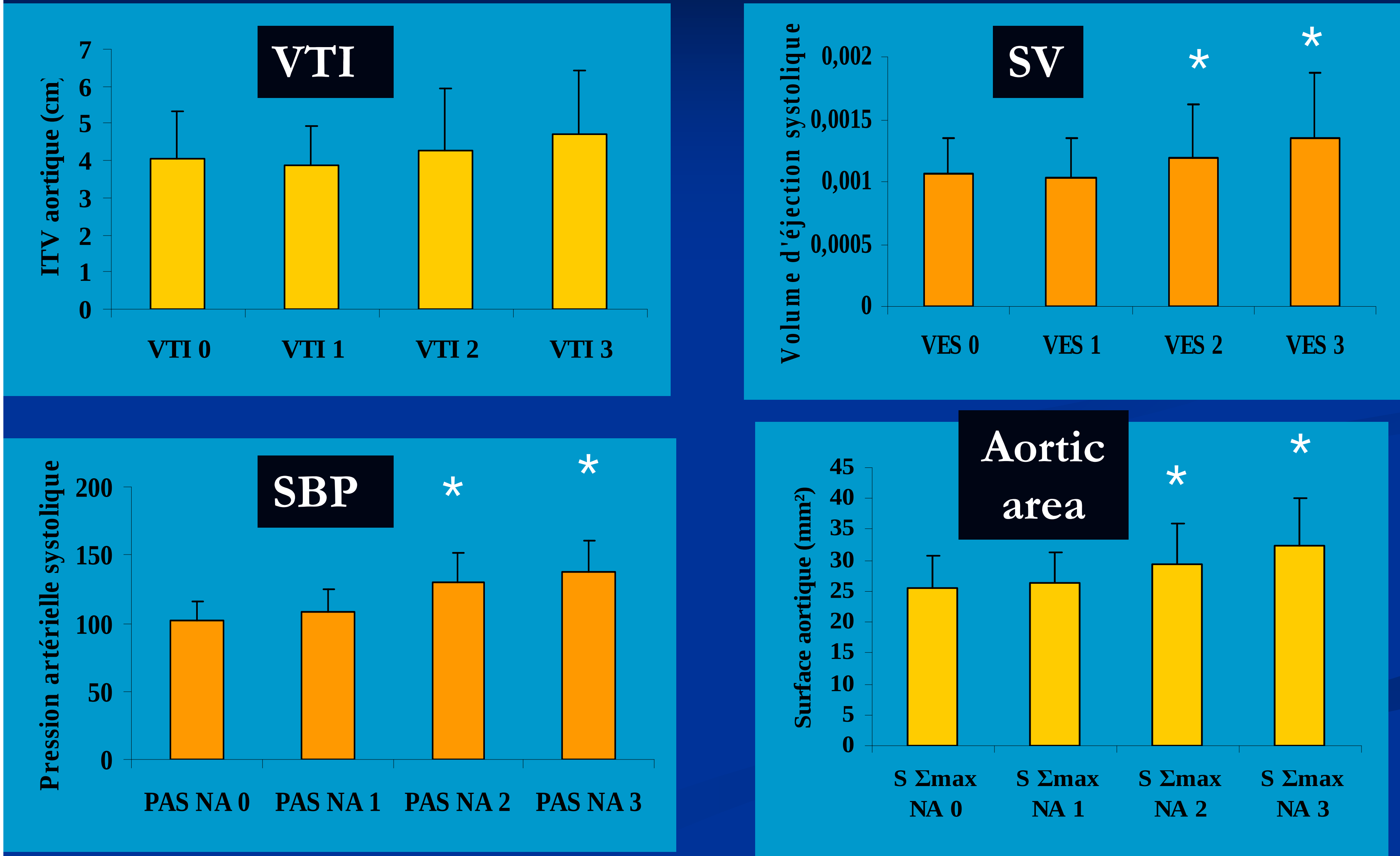
Septic shock with LV dysfunction

VTI 15 cm
SV without diameter : 47 ml
Aortic diameter 2 cm
SV with aortic diameter 47 ml

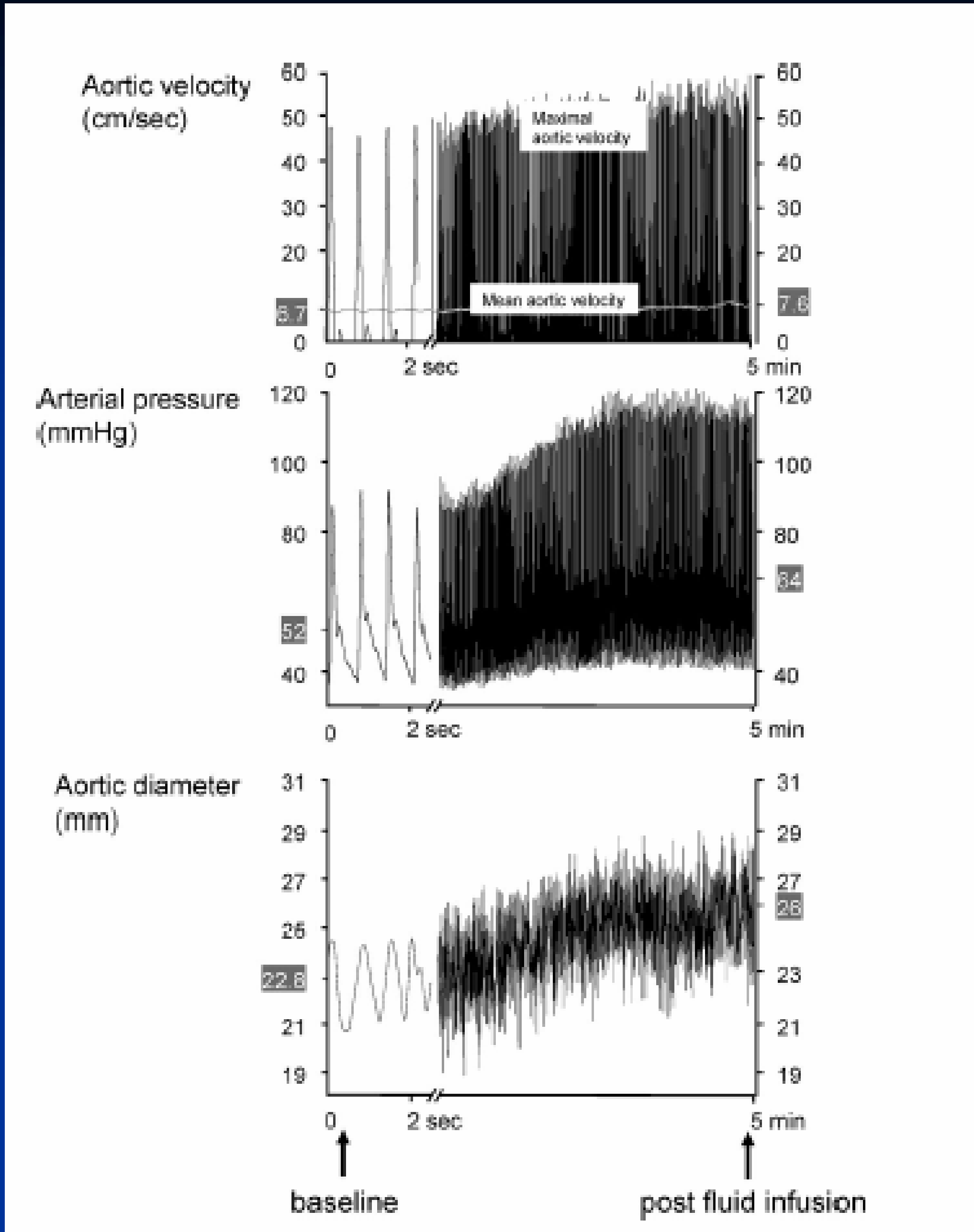
Dobutamine infusion

VTI 15 cm
SV without diameter 47 ml
Aortic diameter 2, 2 cm
SV with aortic diameter 57 ml
Increase of 25%

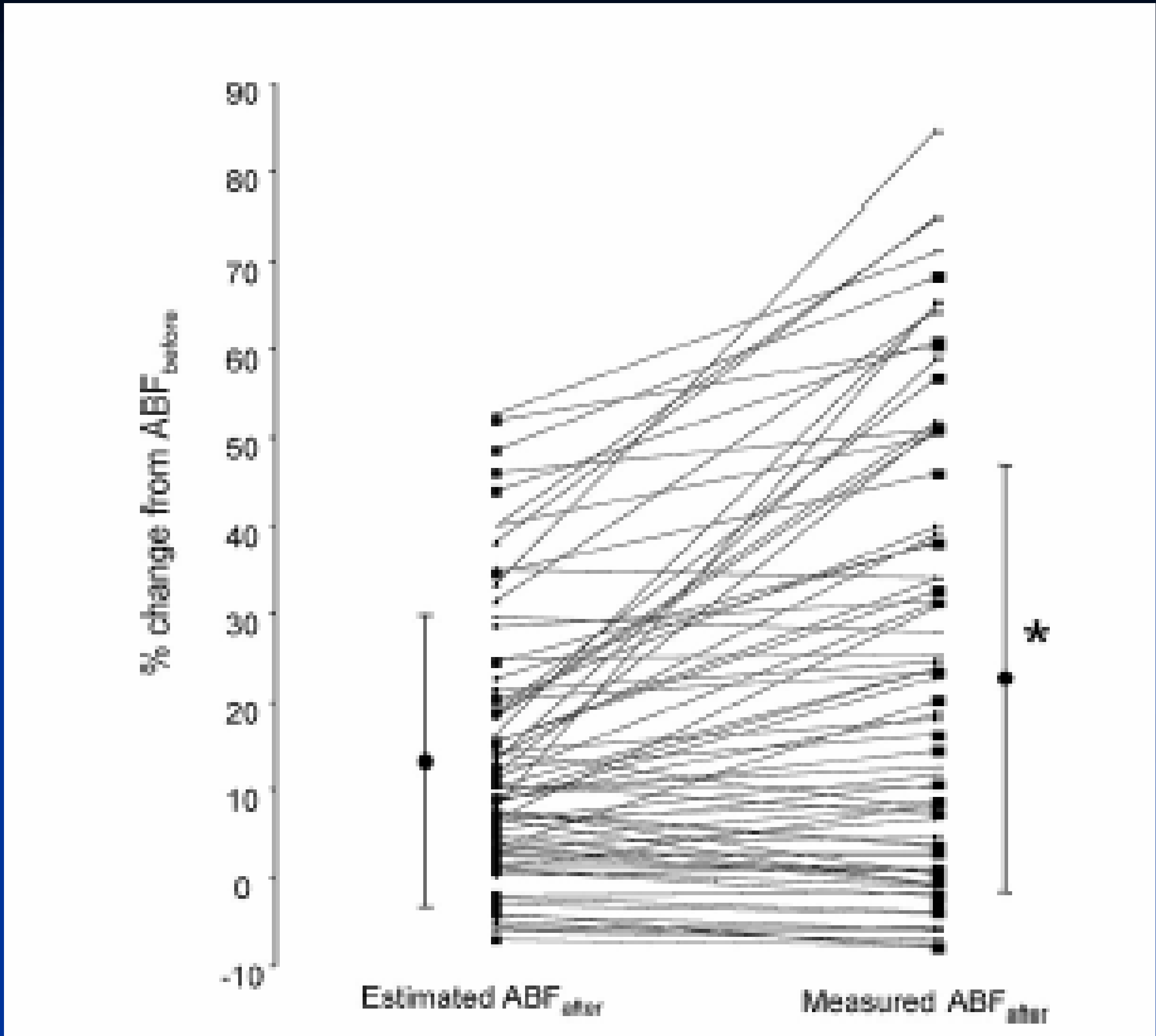
Noradrenaline infusion



Masson, Slama AHA 2005



Monnet Crit care med 2007



Without diameter measurement

With diameter measurement

Monnet Crit care med 2007

Table 2. Assessment of fluid responsiveness depending on method used for defining responders

	Assessment of Fluid Response by Measured ABF_{after}	Assessment of Fluid Response by Estimated ABF_{after}
Responders (n)	41	27
Nonresponders (n)	35	49

Monnet Crit care med 2007

The **InSpectra**™ **StO_2** Tissue Oxygenation Monitor

The InSpectra StO_2
Tissue Oxygenation Monitor

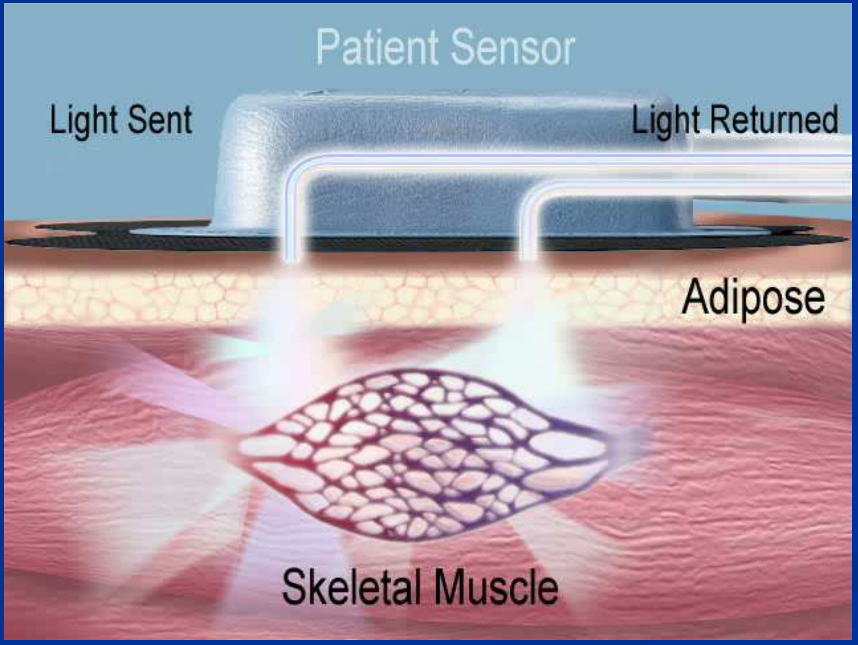
- A direct measure of tissue oxygen saturation (StO_2)
- Noninvasive
- Continuous trended display allows monitoring during resuscitation
- Designed by and for trauma physicians and nurses
- Fluid splash and drip resistant
- External data capture
  - 24-hour continuous data capture
  - with continuous or periodic PC connection

Features

- Displays StO_2 and Tissue Hemoglobin Index (THI)
- Sensor can stay on patient and be reconnected to other monitors throughout hospital
- Portable with minimum 2-hour battery life

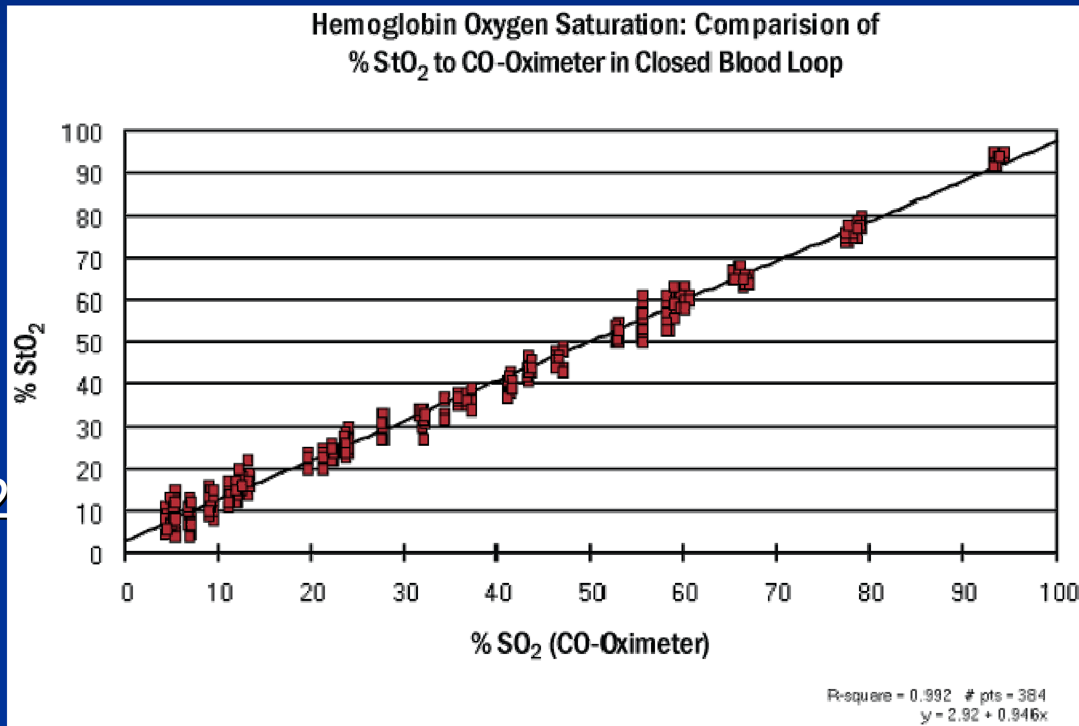
How it works

Deoxygenated hemoglobin (dark blue red) absorbs light differently than oxygenated (bright red) hemoglobin



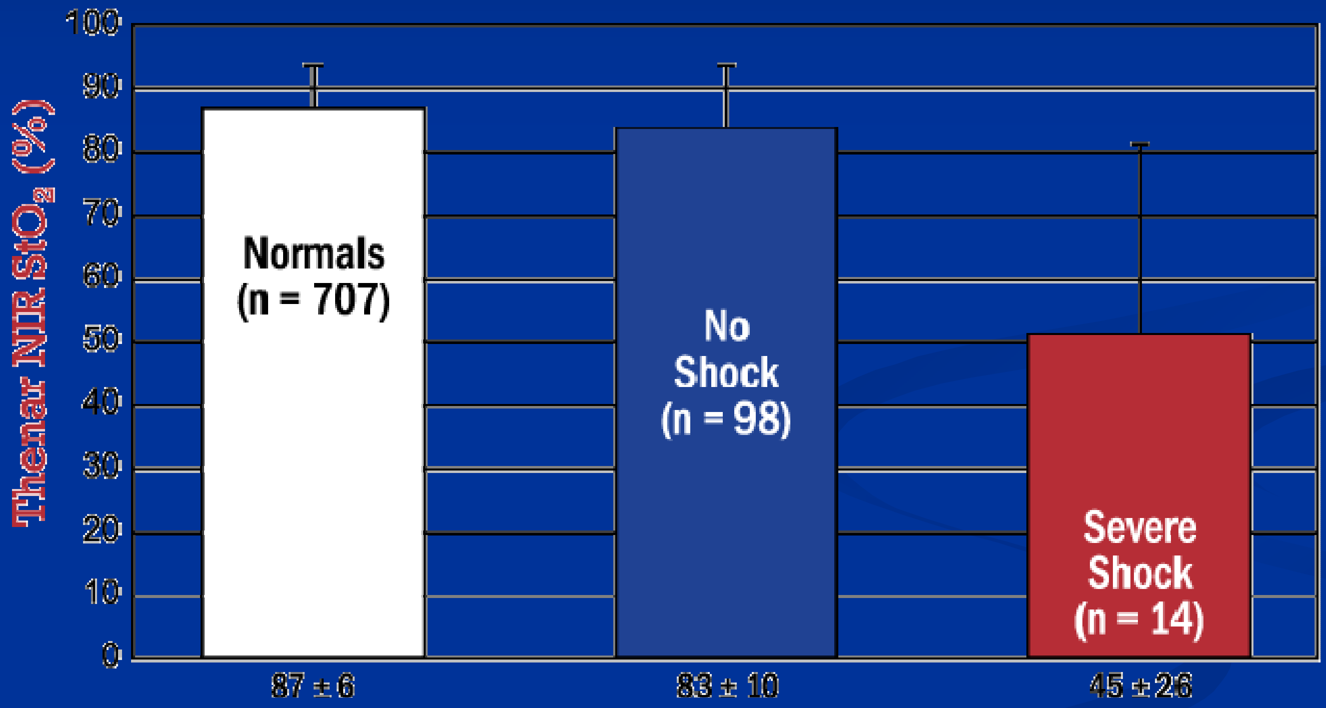
Validation of StO_2 measurement

- Closed circulating blood loop with blood pH and temperature held within normal ranges
- Key Results:
  - 384 paired readings
  - Correlation $r^2 = 0.992$
- Conclusion: Measurements of whole blood O_2 saturation with **InSpectra StO_2** System and 'gold standard' CO-Oximeter have a high degree of correlation



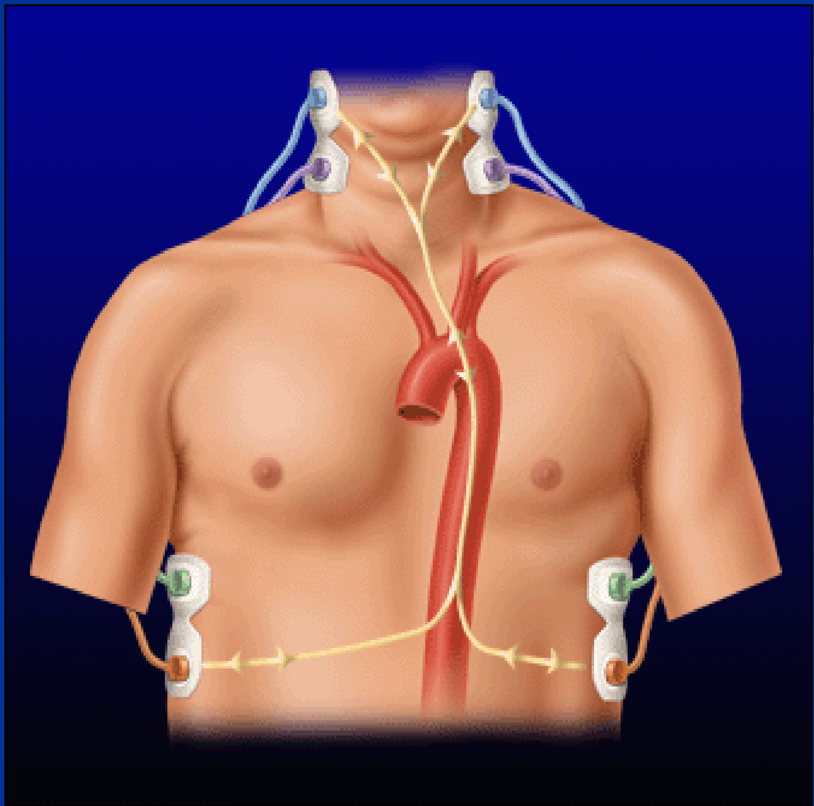
Myers DE et al. Noninvasive Method for Measuring Local Hemoglobin Oxygen Saturation in Tissue Using Wide Gap Second Derivative Near-Infrared Spectroscopy. *Journal of Biomedical Optics* May/June 2005.10(3);034017 1-18.

Thenar eminence StO_2 by shock class



Crookes et al. Can near-infrared Spectroscopy Identify the Severity of Shock in Trauma Patients?
J Trauma. 2005 June; 58(4):806-816

Cardio-impédancemétry

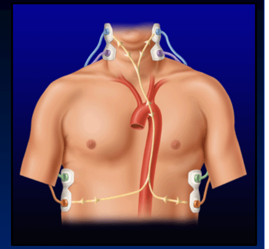


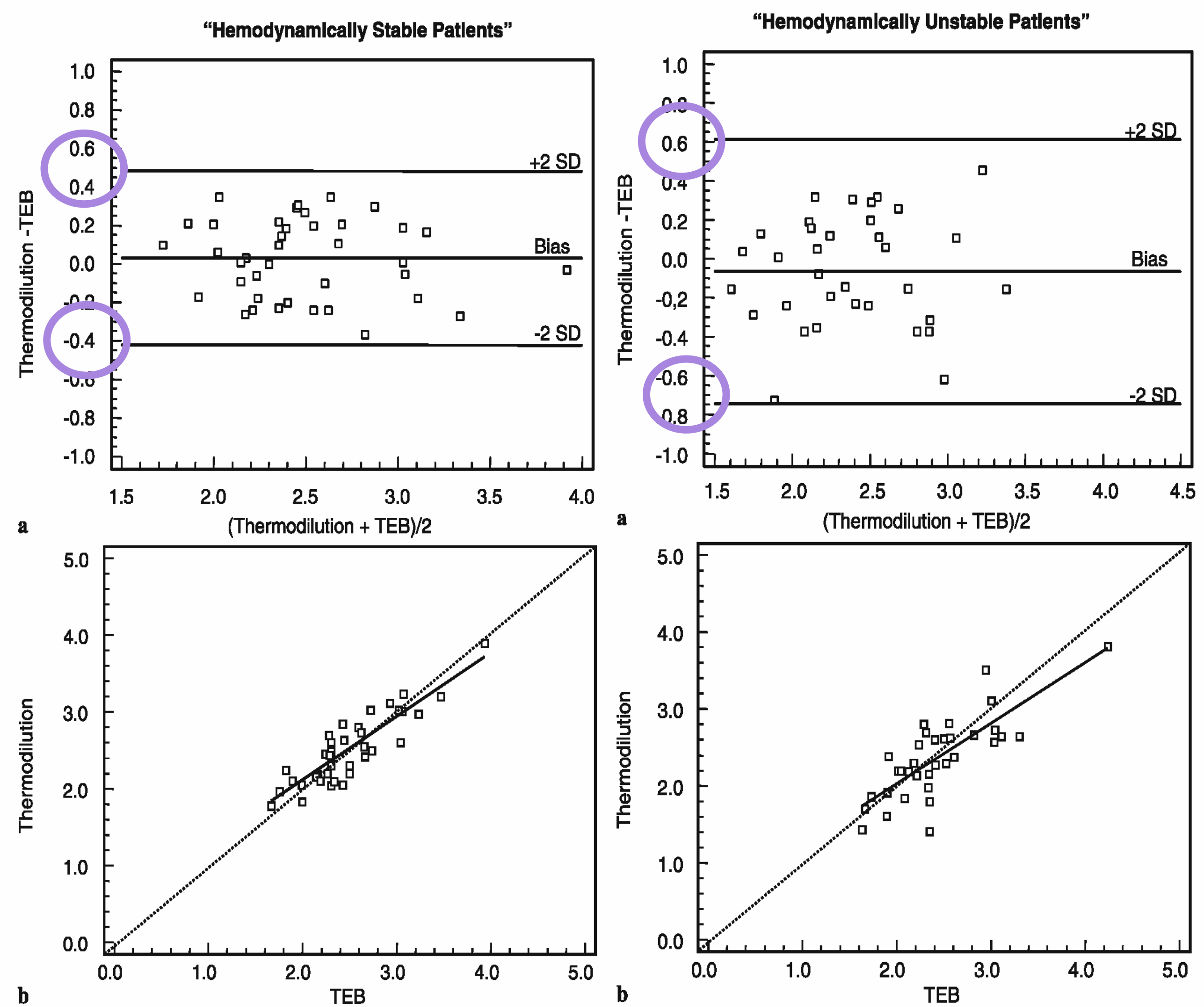
Intensive Care Med (2006) 32:2053–2058
DOI 10.1007/s00134-006-0409-x

BRIEF REPORT

Stefan Suttner
Thilo Schöllhorn
Joachim Boldt
Jochen Mayer
Kerstin D. Röhm
Katrin Lang
Swen N. Piper

Noninvasive assessment of cardiac output using thoracic electrical bioimpedance in hemodynamically stable and unstable patients after cardiac surgery: a comparison with pulmonary artery thermodilution





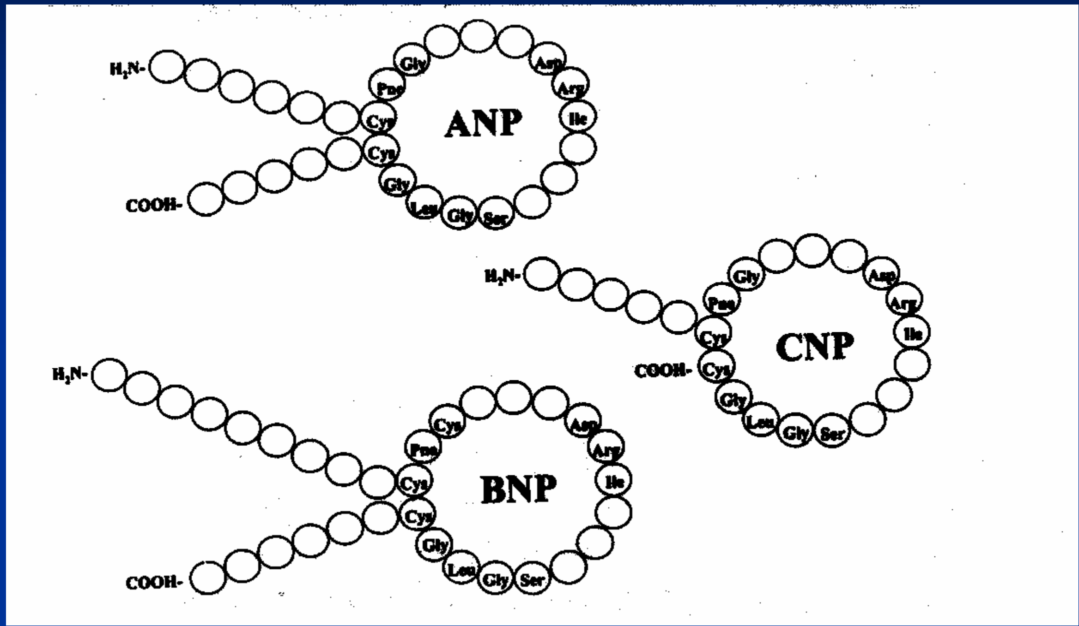
Echocardiography

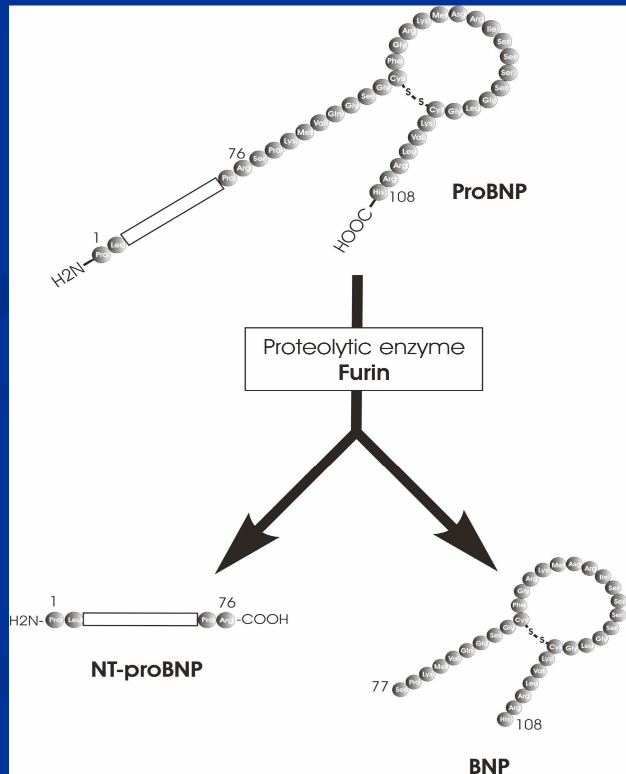
- Assess fluid-responsiveness
- Assess systolic function
- Assess cardiac output
- Assess PAOP
- Assess right cavities

Discontinuous monitoring

Biologie

Structures of natriuretic peptide family. Amino acids listed are common to all three hormones.



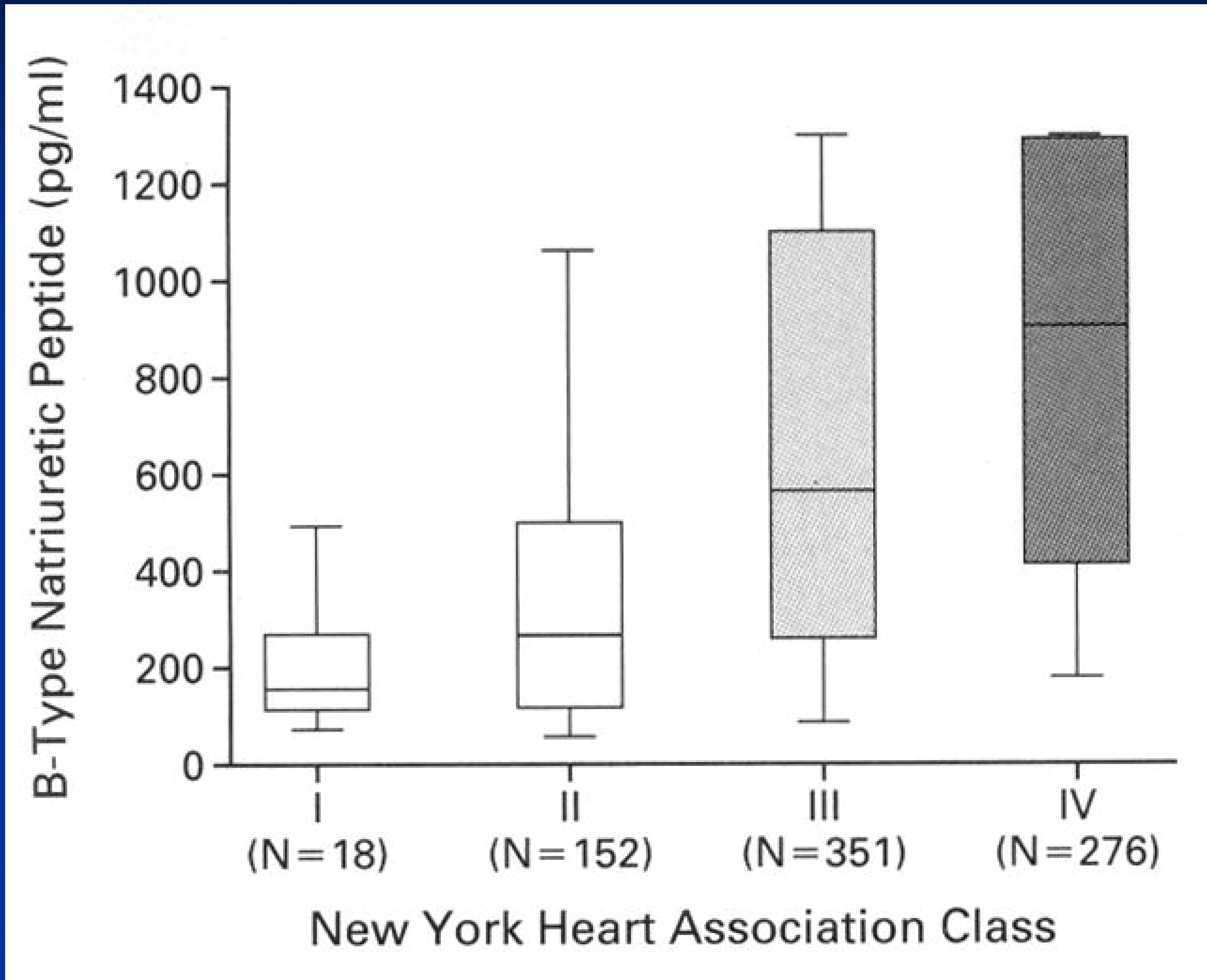


BNP 32 aminoacid-polypeptide identified from porcine brain

Brain NatriureticPeptide 1988

BUT highest levels in ventricle

now called B-type NP



Maisel AS, *N Engl J Med* 2002; 347:161–167

Increased BNP level is a strong predictor for cardiac dysfunction in ICU patients

Study:

- **121 consecutive admissions – Nepean ICU,Sydney**
- **cardiac evaluation by echocardiography**
- **Blood BNP levels by Biosyte immunoassay-range:0-1300 pg/ml**

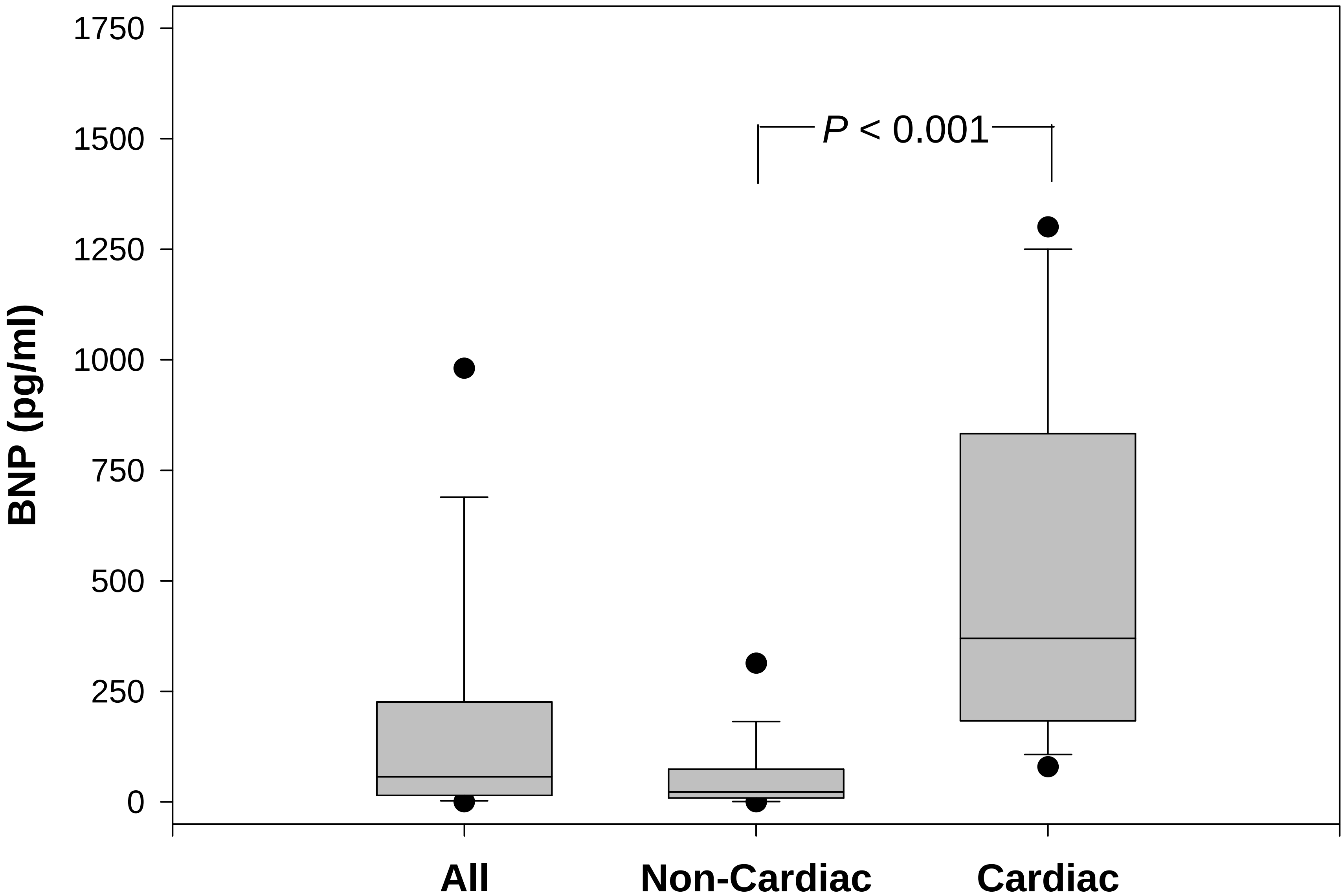
McLean A, Int Care Med 2003;31:21-27

BNP in the ICU: Nepean Study

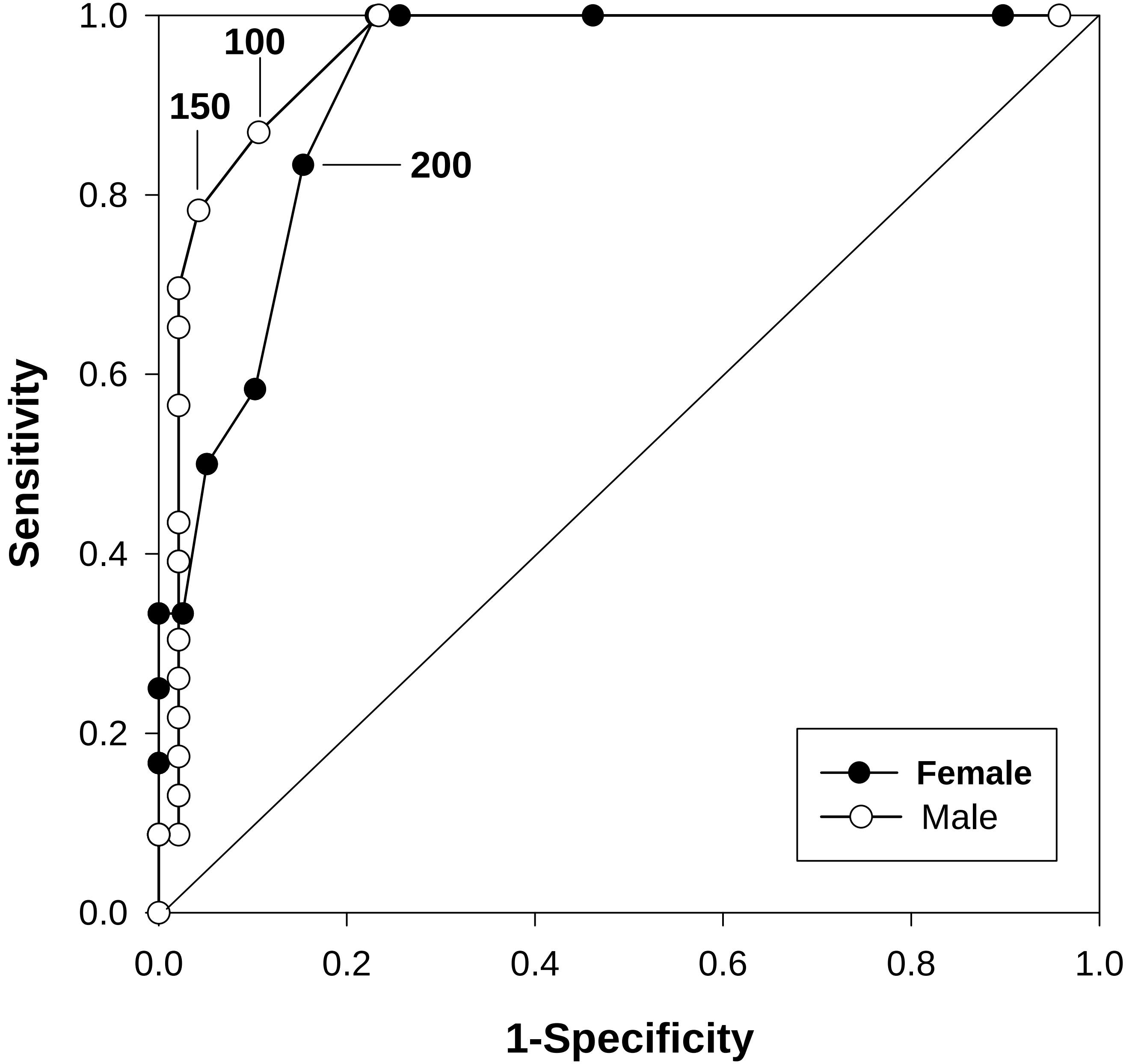
Cardiac dysfunction defined as - left ventricular systolic dysfunction
left ventricular diastolic dysfunction
right ventricular systolic dysfunction

Diagnosis of cardiac dysfunction made on - history/symptoms
ECG/CXR
echocardiography

Two Groups - Group I cardiac dysfunction
Group II non cardiac



BNP (pg/ml)
1750
1500
1250
1000
750
500
250
0
P < 0.001
All
Non-Cardiac
Cardiac



1.0
0.8
0.6
0.4
0.2
0.0
Sensitivity
100
150
200
Female
Male
0.0
0.2
0.4
0.6
0.8
1.0
1-Specificity

Sensitivity, Specificity, positive and negative predictive values for selected cutoff BNP Levels.

Age Group	Cutof (pg/ml)	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Accuracy (%)
All	74	100	62	51	100	62
	147	95	85	72	98	84
	220	84	96	89	94	94
	294	63	96	86	87	94
>55y	74	100	52	60	100	52
	147	100	80	78	100	80
	220	89	96	94	92	95
	294	67	96	92	80	95

PPV=positive predictive value NPV=negative predictive value

Increased BNP level is a strong predictor for cardiac dysfunction in ICU patients

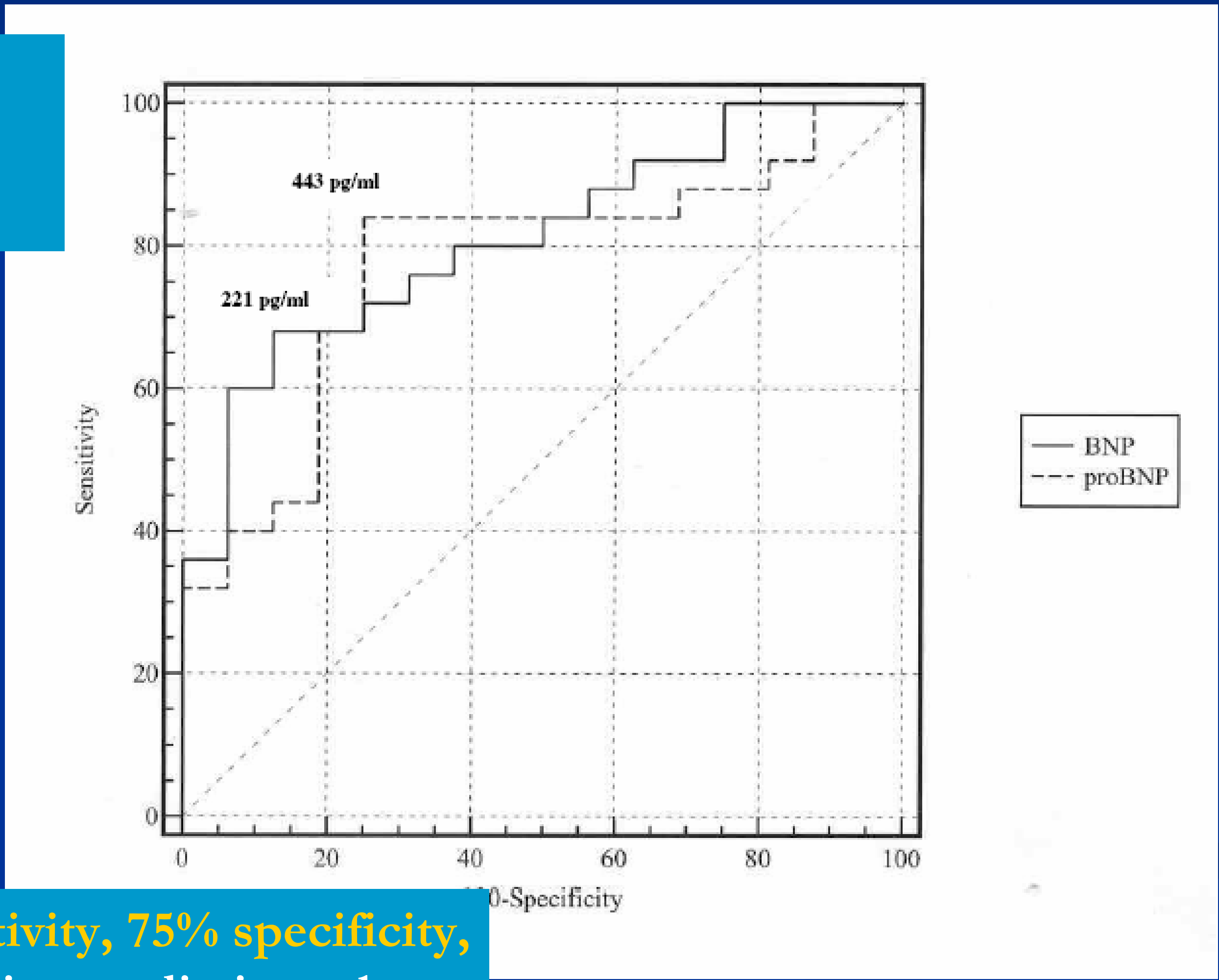
BNP > 147 pg/ml = sensitivity 95% specificity 85% NPV 98%

McLean A, Tang B, Nalos M, Huang S, Stewart D AnsInt Care 2003;31:21-27

Receiver-operating curve of B-type natriuretic peptide (BNP) and N-terminal pro-BNP (NT-proBNP) in patients with and without heart failure as assessed by echocardiography (patients with shock or respiratory distress)

N = 41

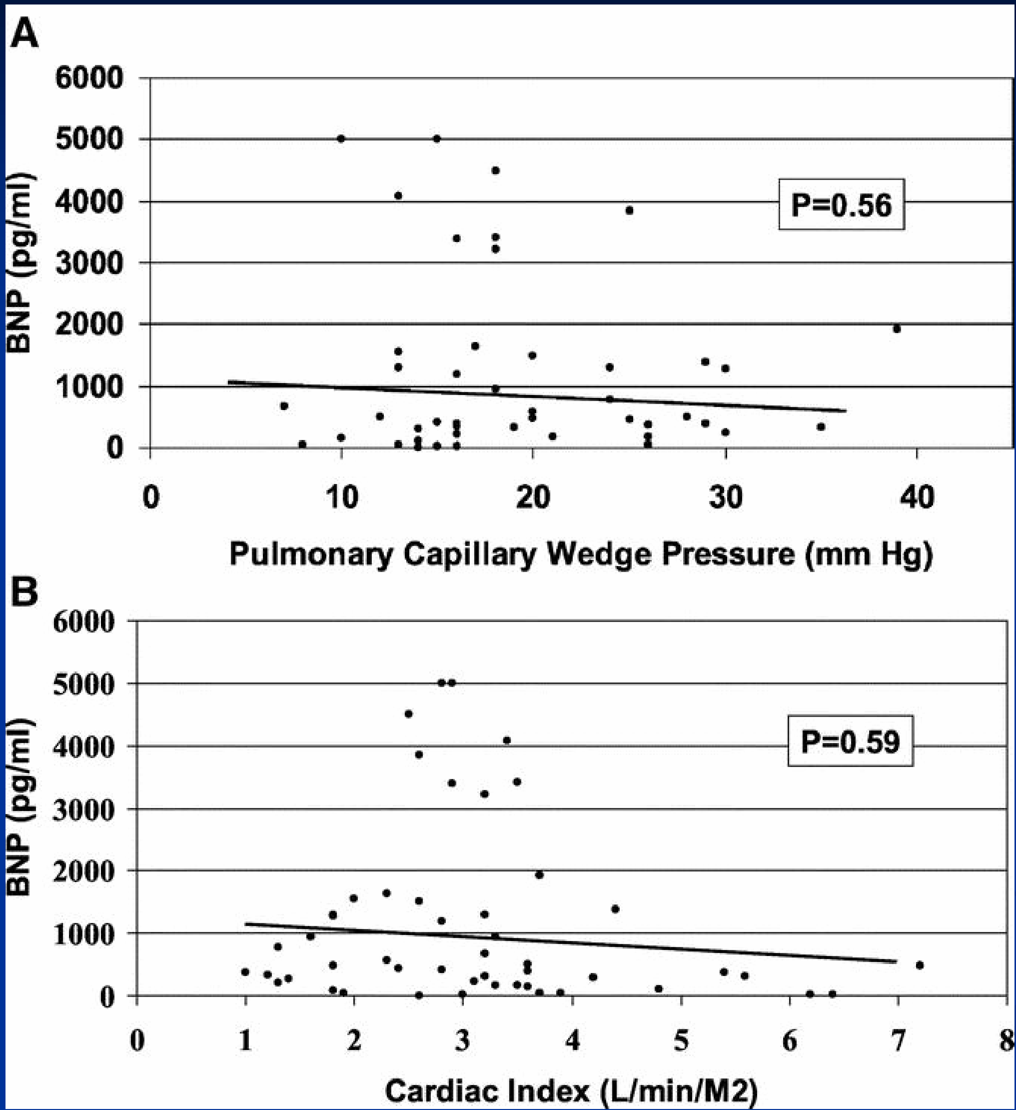
10 with RV dysfunction



84% sensitivity, 75% specificity, 75% negative predictive value

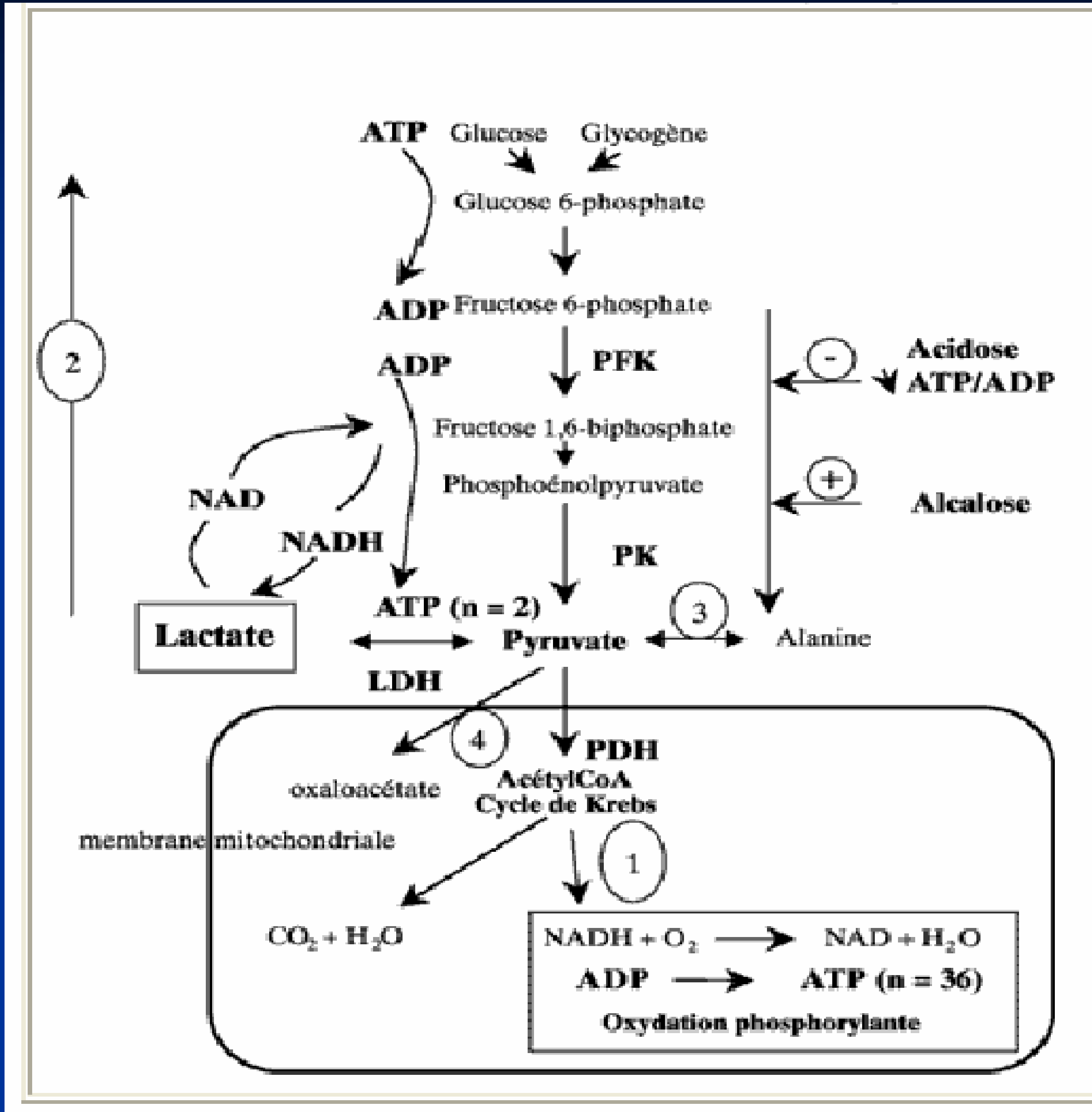
Bal Acta Anesth Scand 2006

Etat de choc



Tung: Crit Care Med, 32(8). 2004.1643-1647

Mêmes résultats Mekontso-Dessap A (Teboul) SRLF 2005


ATP
Glucose
Glycogène
Glucose 6-phosphate
ADP
Fructose 6-phosphate
ADP
PFK
Fructose 1,6-biphosphate
Phosphoénolpyruvate
NAD
NADH
PK
ATP (n = 2)
Lactate
Pyruvate
LDH
Alanine
Acidose
ATP/ADP
Alcalose
-
+
1
2
3
4
PDH
AcétylCoA
Cycle de Krebs
oxaloacétate
membrane mitochondriale
$CO_2 + H_2O$
$NADH + O_2 \longrightarrow NAD + H_2O$
ADP → ATP (n = 36)
Oxydation phosphorylante

Métabolisme

- Production : 1300 à 1500 mmol/j
  - Erythrocytes, intestin, cerveau, muscle,peau
- Métabolisme : foie (70%), reins, cœur
- Production hépatique si baisse du débit de 75%
- Rôle de la fonction hépatique : cirrhose
- Rein : cortex métaboliseur (néoglucogénèse) et médullaire productrice

Lactate et pronostic

- Relation bien établie entre morbidité/mortalité et intensité/durée de l'hyperlactatémie

Serial Blood Lactate Levels Can Predict the Development of Multiple Organ Failure Following Septic Shock

Jan Bakker, MD, Philippe Gris, MD, Michel Coffernils, MD, Robert J. Kahn, MD, Jean-Louis Vincent, MD, PhD, *Brussels, Belgium*

Am J Surg. 1996;171:221–226.

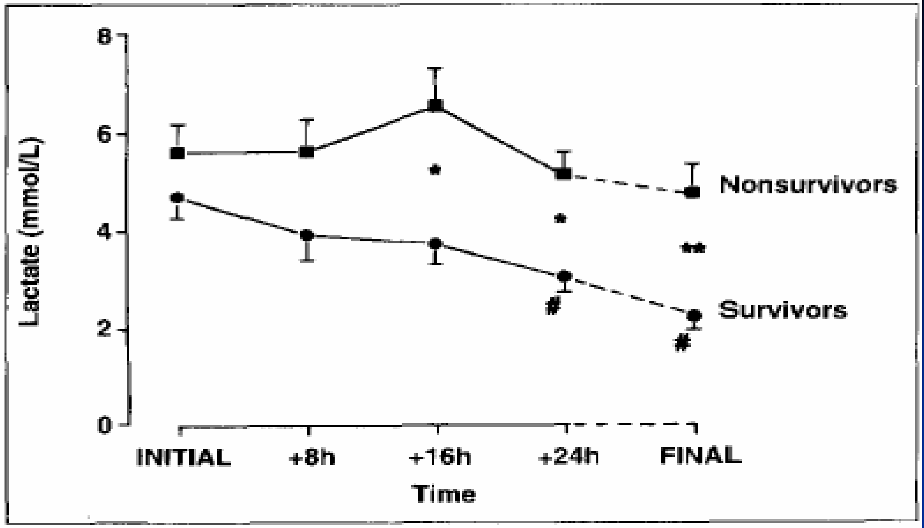
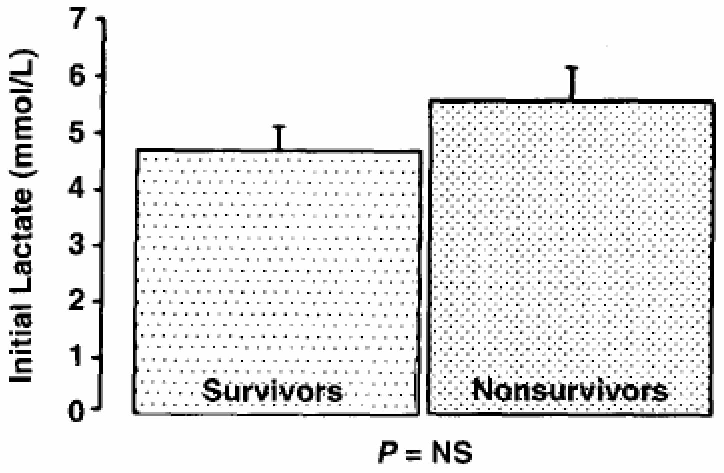


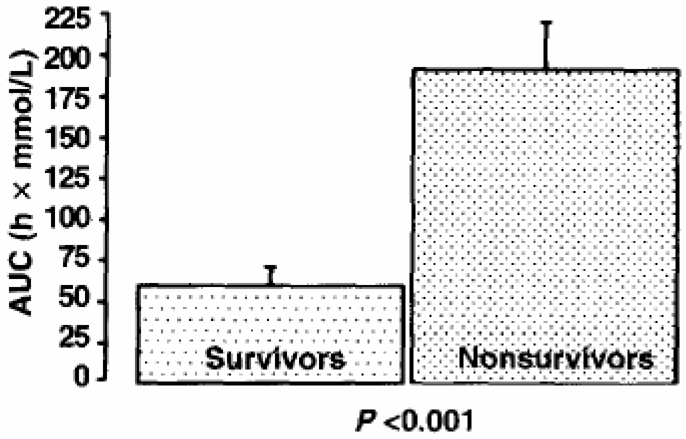
Figure 1. Time course of blood lactate levels for the 33 survivors and the 41 nonsurvivors. Initial values were taken at onset of shock, and the final values at time of recovery or before death (mean ± standard error of the mean). *$P<0.05$; **$P<0.01$ (survivors versus nonsurvivors); #$P<0.05$ versus initial blood lactate level.

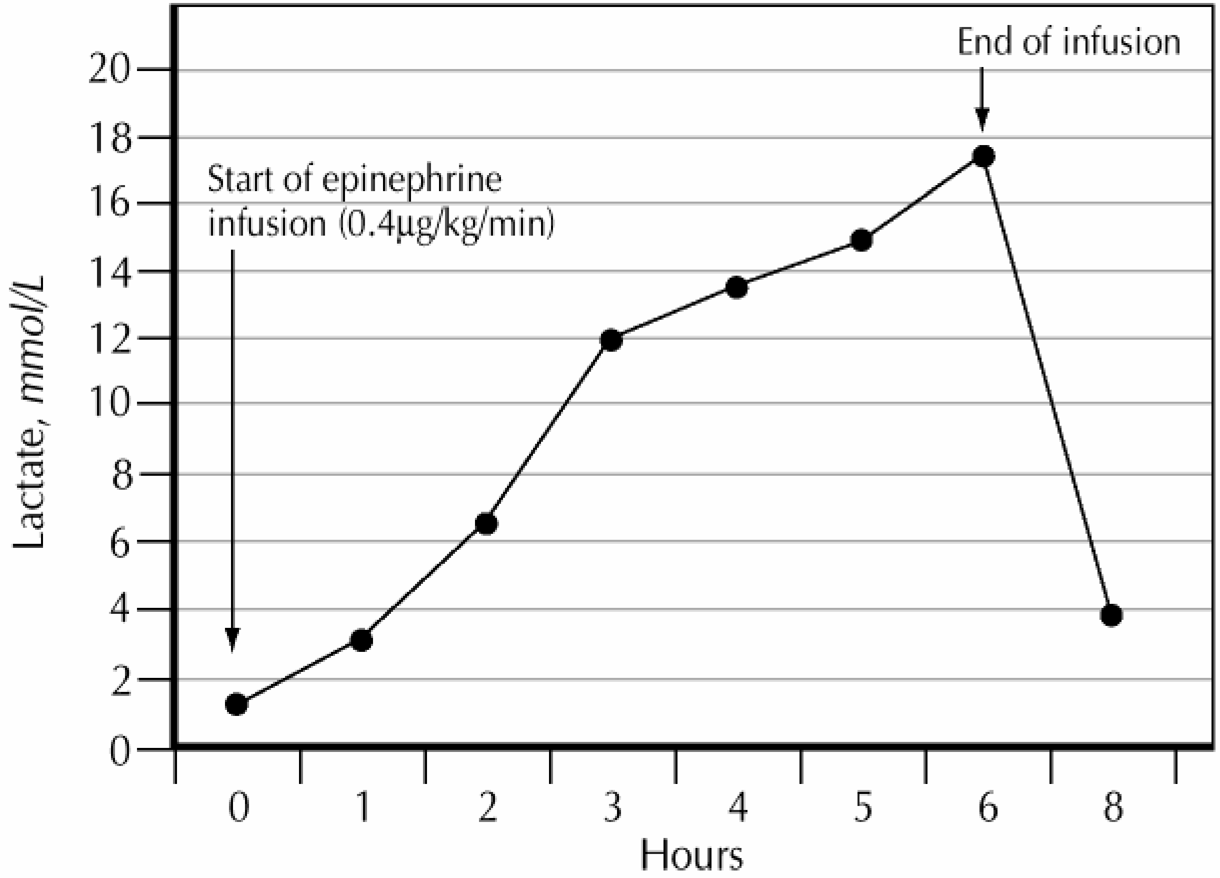
TABLE IV

Discriminants of Survival by Multiple Regression Analysis of All Measurements in the 74 Patients Surviving the First 24 Hours of Septic Shock

Variable	R^2 at End of Stepwise Regression Analysis	P Value of the R^2 Change
Lactime (h)	0.266	0.001
Age (y)	0.384	0.001
Heart rate (bpm)	0.440	0.02
Mean arterial pressure (mm Hg)	0.485	0.04







As can be seen, epinephrine infusion induces marked hyperlactatemia. During this time, systemic oxygen delivery is approximately doubled. This severe hyperlactatemia cannot be secondary to tissue hypoxia.

Lactate

- Reconsidération de la signification physiopathologique et du rôle du lactate dans le choc septique
  - Mécanisme adaptatif
  - Comburant préférentiel (cœur, cerveau)
  - Message métabolique
- Marqueur de gravité
- La production pulmonaire et digestive est probablement aussi d'origine inflammatoire

Finalement qu'est ce que je fais?

- Pression artérielle invasive : beaucoup d'informations.
- L'échocardiographie systématiquement une fois par jour si état de choc.
- Le BNP et le lactate
- Rarement le PICCO et plus jamais ni le cathétérisme cardiaque droit ni le Doppler oesophagien.
- J'évalue d'autres moniteurs : NIRS et Phlétysmographie

Conclusion

- Quelques arguments pour monitorer les patients
- Par de techniques non ou peu invasives
- Utiliser la technique que l'on connaît le mieux