

How to use arterial pressure to assess Hemodynamics ?



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Conflicts of interest

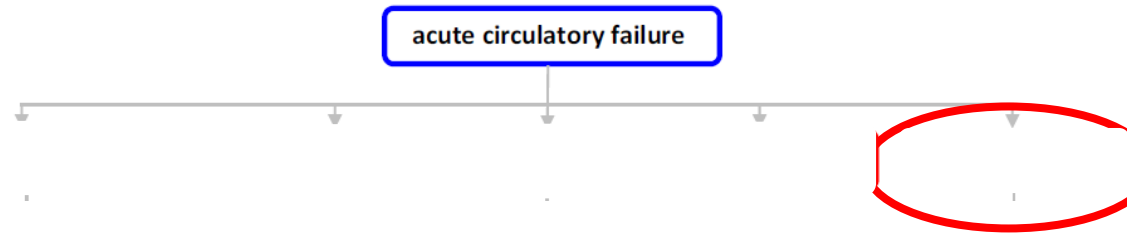
- Received Honoraria for Lectures from Baxter
- Received Honoraria From AOP for Consulting
- Member of the scientific board of Vitaris





Less invasive hemodynamic monitoring in critically ill patients

Jean-Louis Teboul^{1*}, Bernd Saugel², Maurizio Cecconi³, Daniel De Backer⁴, Christoph K. Hofer⁵, Xavier Monnet¹, Azriel Perel⁶, Michael R. Pinsky⁷, Daniel A. Reuter², Andrew Rhodes³, Pierre Squara⁸, Jean-Louis Vincent⁹ and Thomas W. Scheeren¹⁰

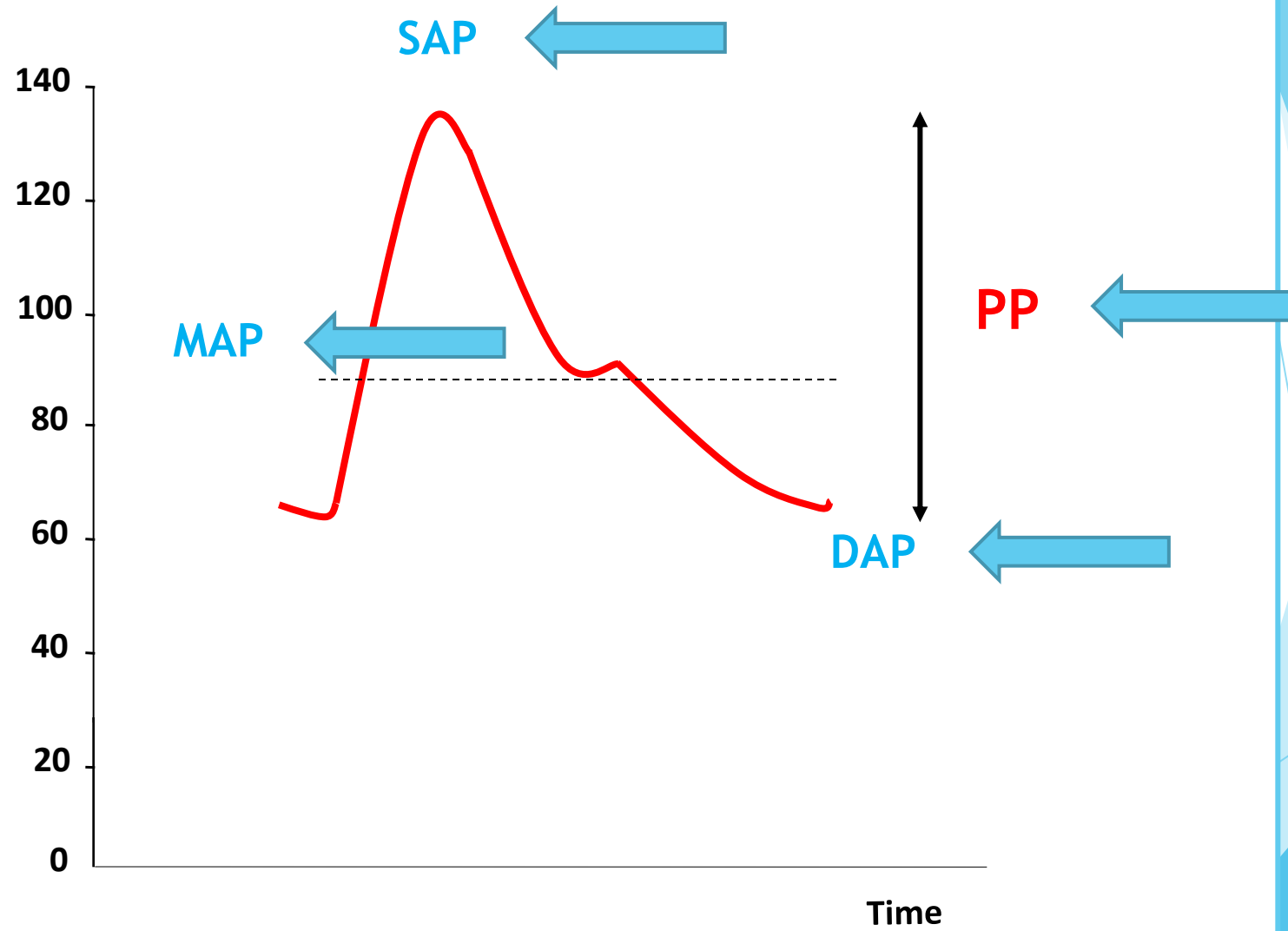


How can we use Arterial pressure?

- **Analysis of the static values**
- **Analysis of the dynamic variation of AP values**

- **Analysis of the static values**
- Analysis of the dynamic variation of AP values

Arterial pressure (mmHg)



Is it important to consider DAP in septic shock ?

1. DAP is a reflection of vasomotor tone arterial tone



a low DAP is mainly due to a depressed

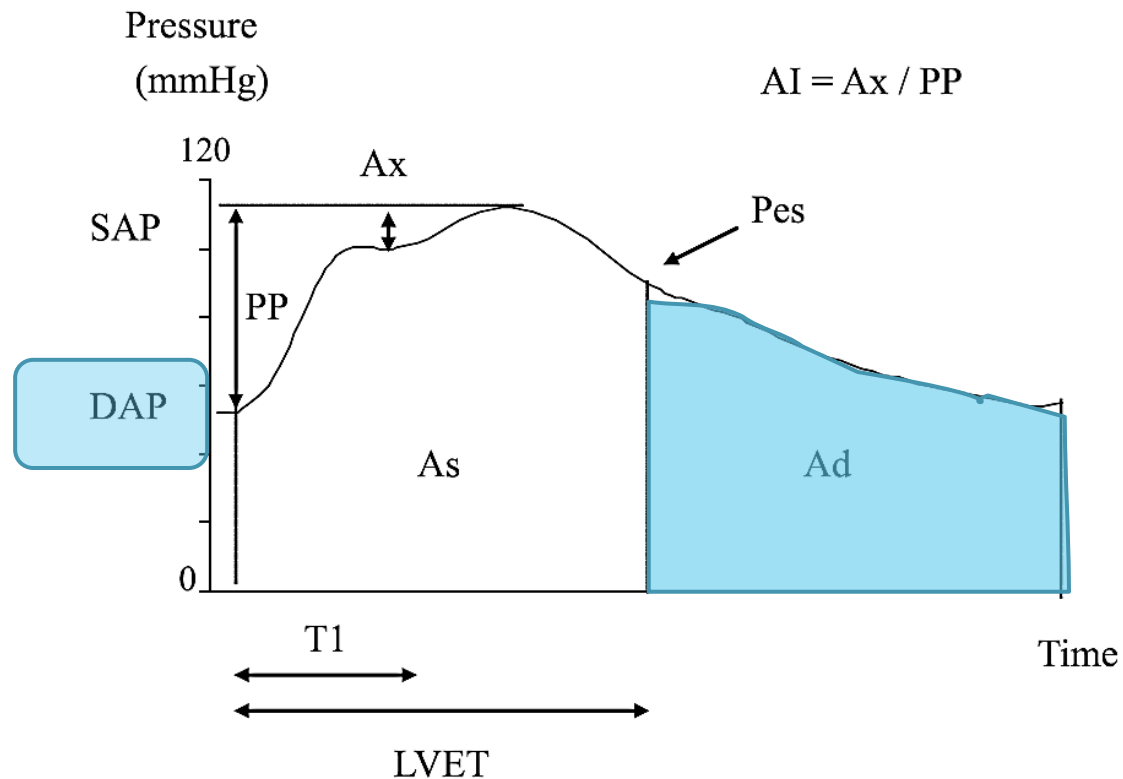


a DAP is mainly due to a depressed arterial to low tone

Bouchra Lamia
Jean-Louis Teboul
Xavier Monnet
David Osman
Julien Maizel
Christian Richard
Denis Chmela

Contribution of arterial stiffness and stroke volume to peripheral pulse pressure in ICU patients: an arterial tonometry study

67 ICU patients



Peripheral resistance

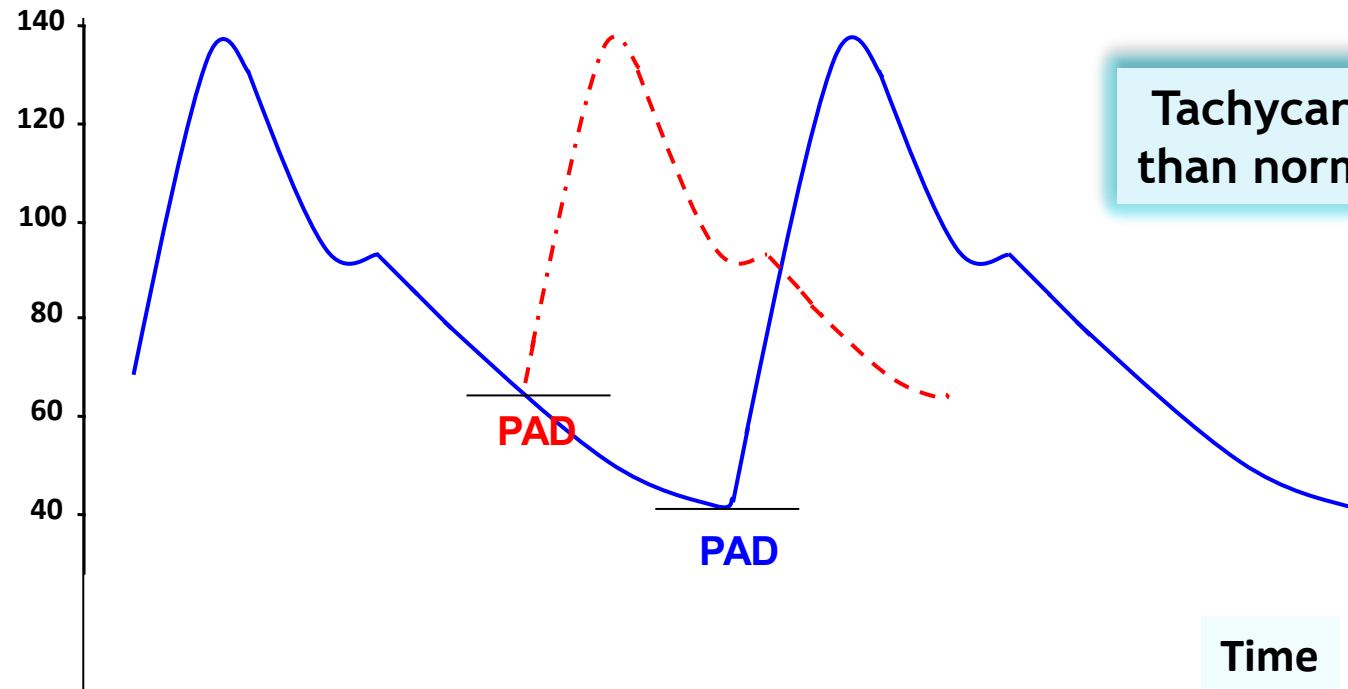
- related to peripheral **DAP** ($r = 0.71$) ($p < 0.001$)
- but not related to peripheral **SAP** ($r^2 = 0.04$) and **PP** ($r^2 = 0.02$)

Is it important to consider DAP in septic shock ?

1. DAP is a reflection of vasomotor tone \longrightarrow a low DAP is mainly due to a depressed arterial tone
BUT

Always consider heart rate

Pression artérielle(mmHg)



Tachycardia should theoretically result in a higher than normal DAP

Is it important to consider DAP in septic shock ?

1. **DAP** is a reflection of vasomotor tone a low **DAP** is mainly due to a depressed arterial tone
  a low **DAP** may be seen during **Bradycardia**

Is it important to consider DAP in septic shock ?

1. **DAP** is a reflection of vasomotor tone

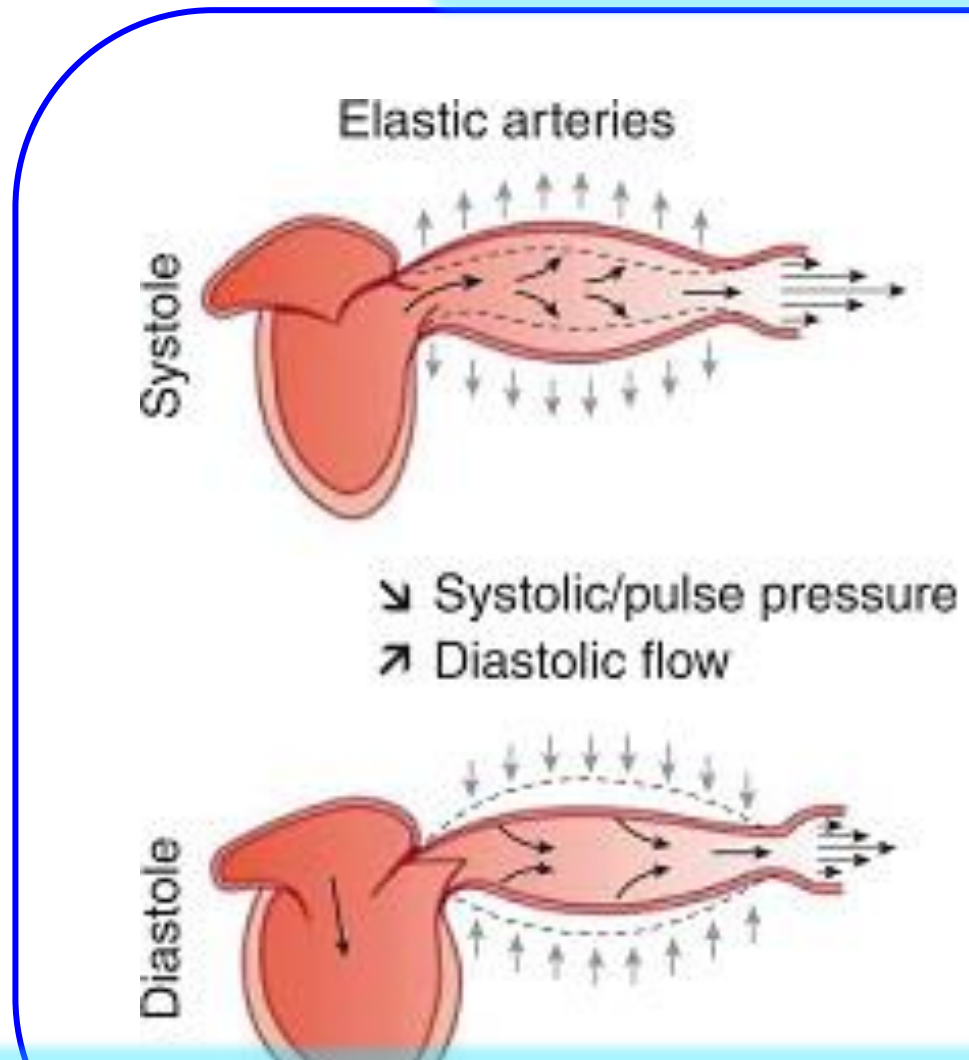
a low **DAP** is mainly due to a depressed arterial tone

a low **DAP** may be seen during Bradycardia



Arterial stiffness

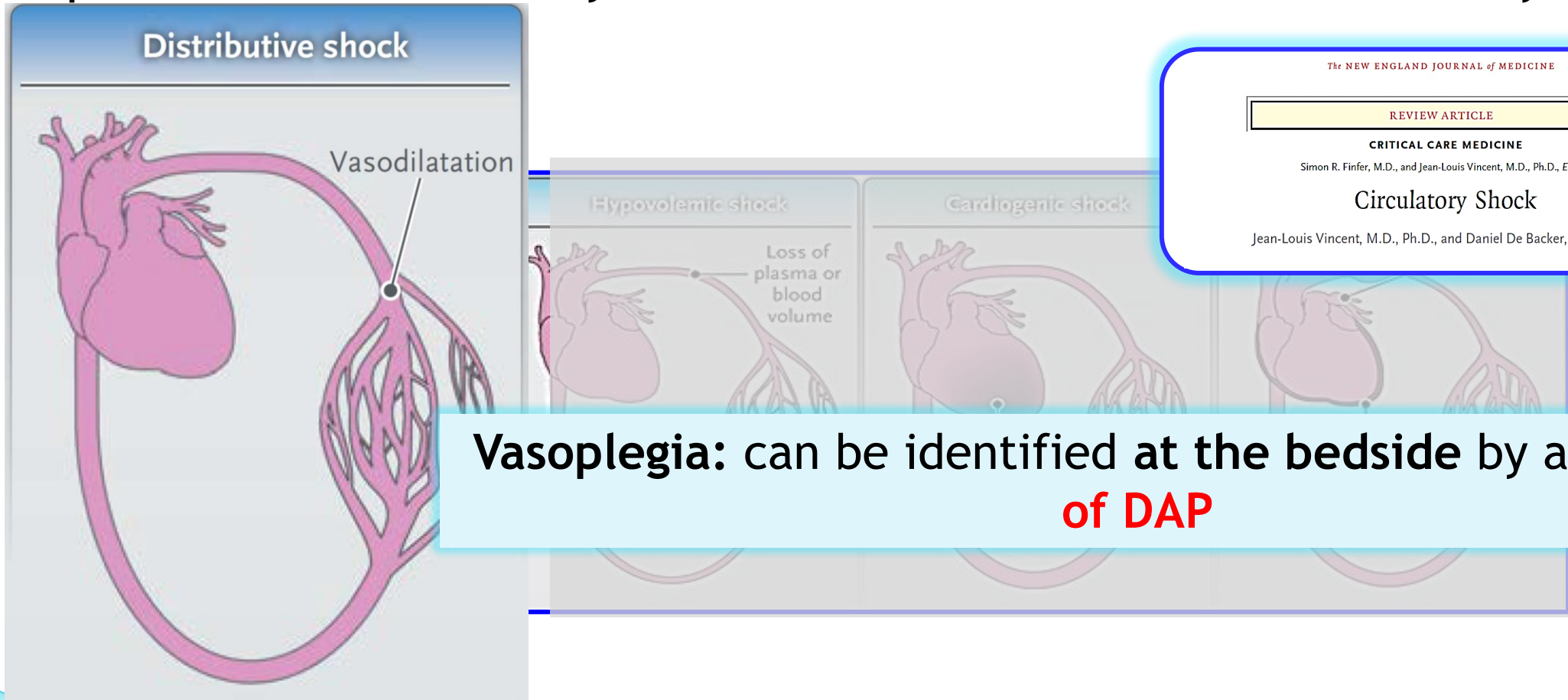
The cardiac pump generates a pulsatile flow



The arteries α In patients with stiff arteries the diastolic part of aortic pressure is reduced part of the stroke volume during the systole and by restituting it during the diastole

Is it important to consider DAP in septic shock ?

1. DAP is a reflection of vasomotor tone
2. Septic shock is characterised by vasodilation and a low vasomotor tone: DAP may help the



Vasoplegia: can be identified at the bedside by a **low value of DAP**

Is it important to consider DAP in septic shock ?

1. DAP is a reflection of vasomotor tone
2. Septic shock is characterised by vasodilation and a low vasomotor tone
3. **DAP is the major component Of MAP**

Review

Clinical review: Interpretation of arterial pressure wave in shock states

Bouchra Lamia¹, Denis Chemla², Christian Richard³ and Jean-Louis Teboul³

Critical Care 2005, 9:601-606

$$\text{MAP} = \text{DAP} + 1/3(\text{SAP} - \text{DAP}) \quad (3)$$

This formula can be rewritten as follows:

$$\text{MAP} = (2/3 \times \text{DAP}) + (1/3 \times \text{SAP}) \quad (4)$$

In other words, this rule of thumb implies that **DAP** contributes more to **MAP**, by a factor of two, than does **SAP**.

Frequently a **Low MAP** is mainly due to a **low DAP**

Is it important to consider DAP in septic shock ?

1. DAP is a reflection of vasomotor tone
2. Septic shock is characterised by vasodilation and a low vasomotor tone
3. DAP is the major component Of MAP
4. DAP is constant through the vascular tree

The peripheral AP is different from the central (aortic) AP

Central pressure wave

Peripheral pressure wave



femoral SAP and PP < radial SAP and PP

Not the case of
DAP

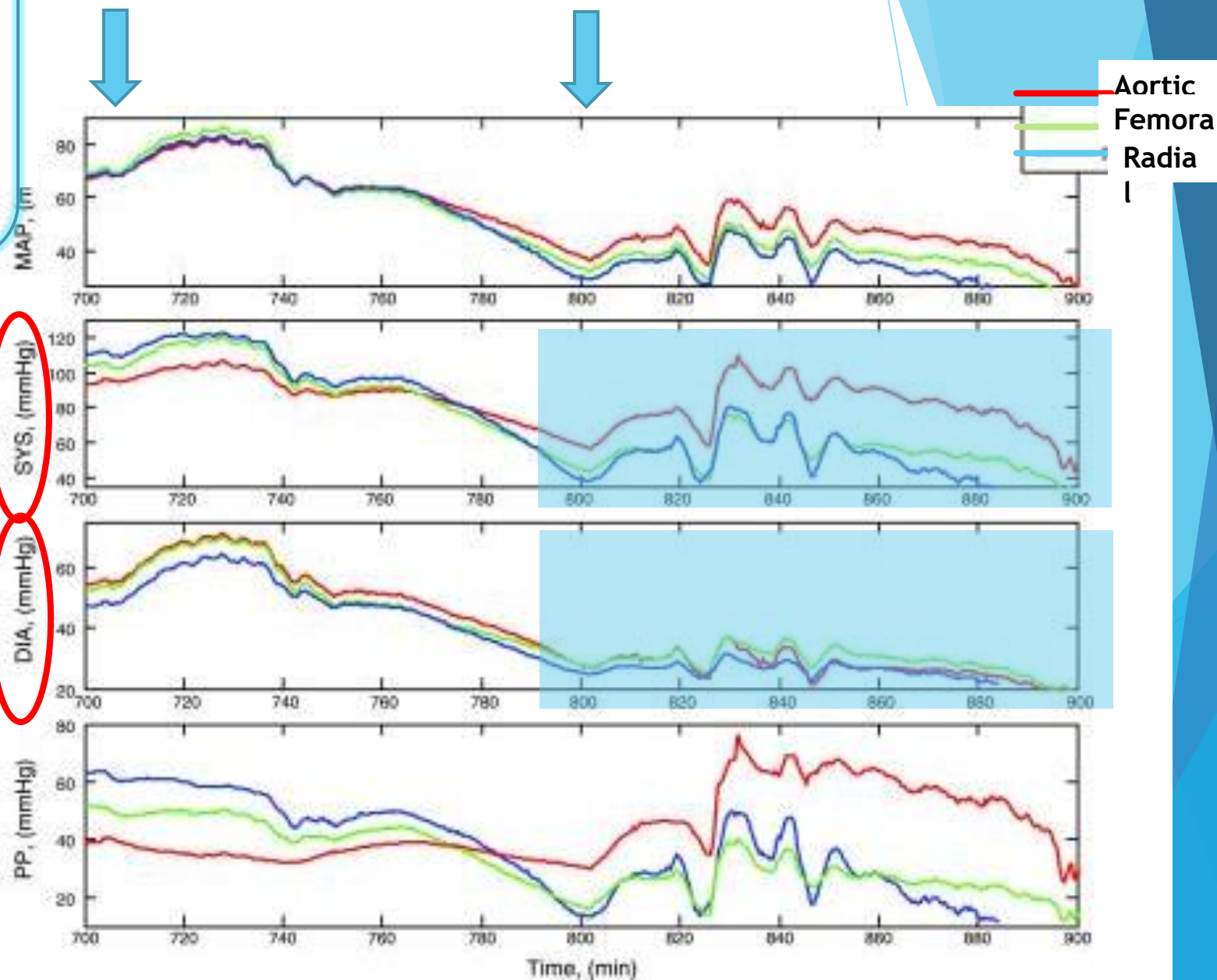
Peripheral vascular decoupling in porcine endotoxic shock

Feras Hatib, Jos R. C. Jansen, and Michael R. Pinsky

[J Appl Physiol \(1985\). 2011 Sep; 111\(3\): 853-860.](#)

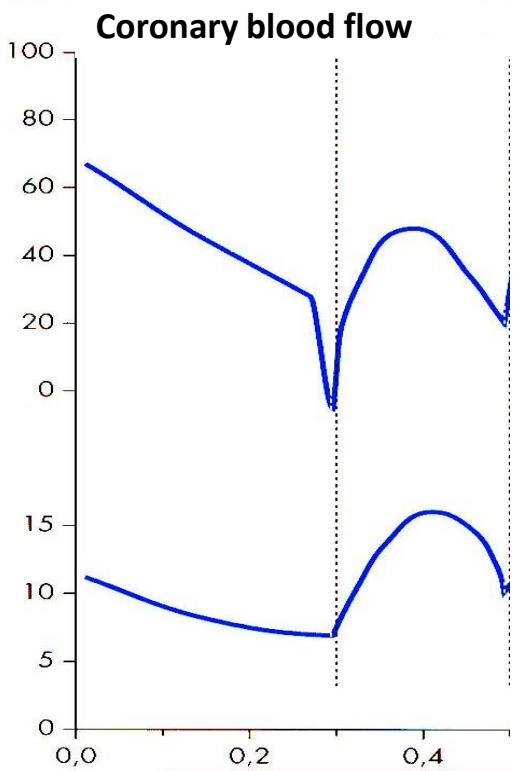
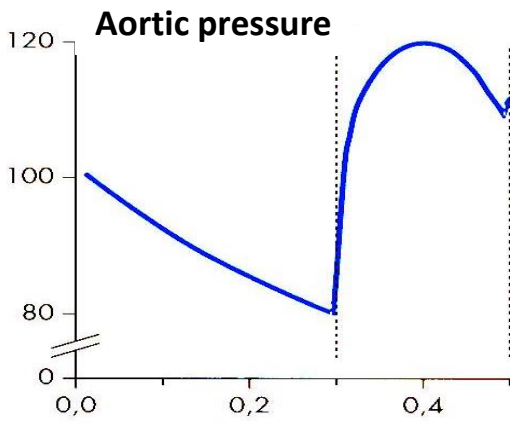
Endotoxin

Resuscitation



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4. DAP is constant through the vascular tree
5. **DAP is the upstream pressure for LV coronary perfusion**



DAP is the upstream pressure for LV coronary perfusion

Is it important to consider DAP in septic shock ?

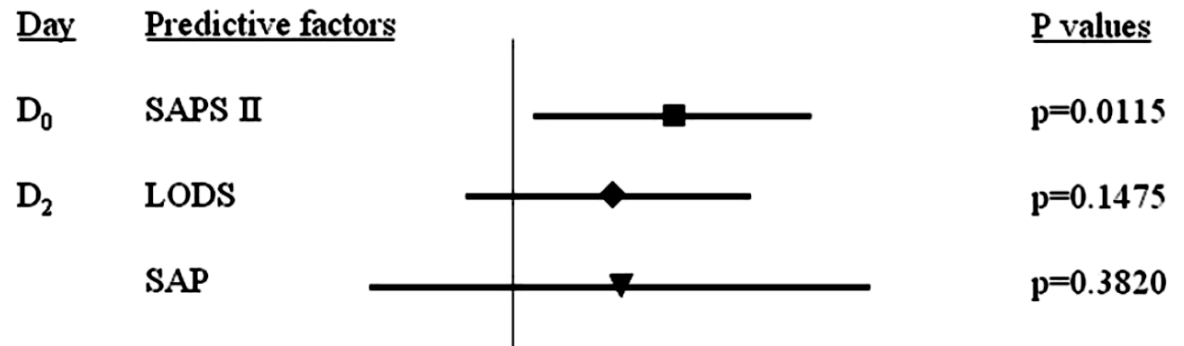
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6. **DAP is associated to outcome**

Diastolic Arterial Blood Pressure: A Reliable Early Predictor of Survival in Human Septic Shock

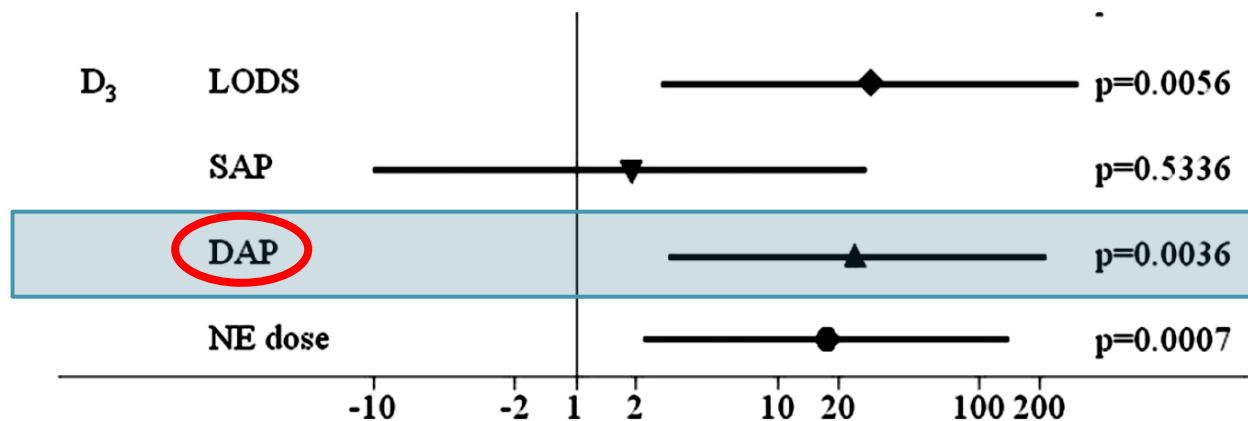
Samir Benckroune, MD, Peter C. J. Karpati, MD, Christine Berton, MD†, Cédric Nathan, MD, Joaquim Mateo, MD, Mansour Chaara, MD, Florence Riché, MD, Marie-Josèphe Laisné, MD, Didier Payen, MD, PhD, and Alexandre Mebazaa, MD, PhD

J Trauma. 2008;64:1188–1195

- 68 septic shock patients
- Receiving NE for at least 72 hrs
- Observational study



At D₃ **low DAP** (and not low SAP) was a predictor of **mortality**



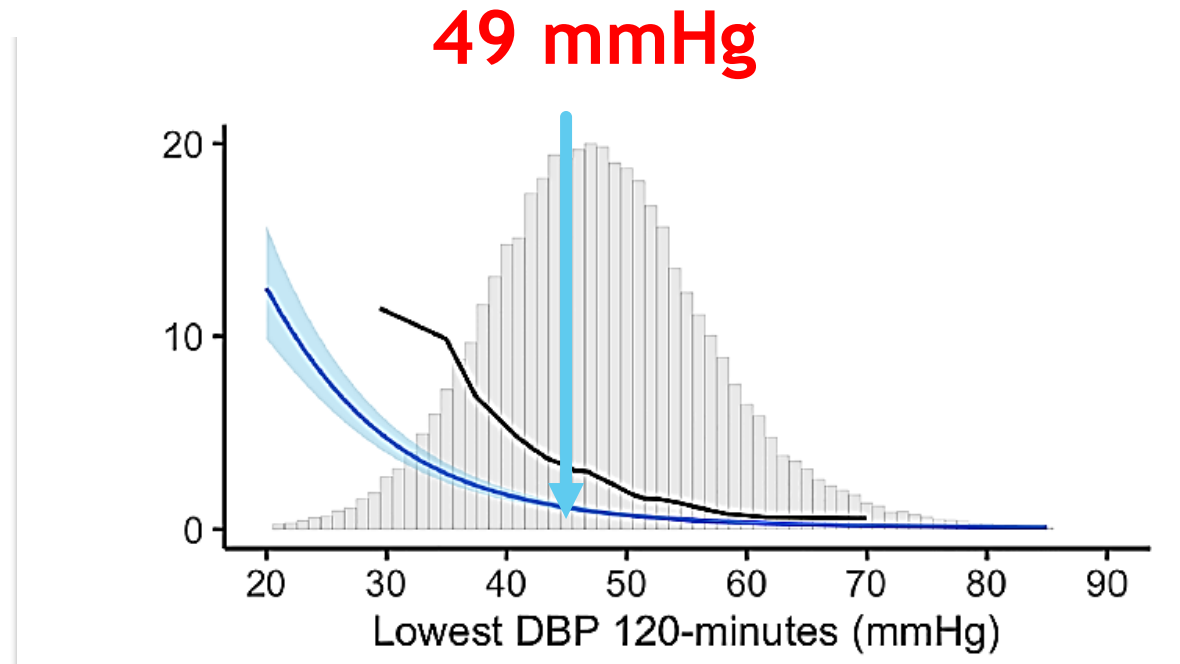


Association of systolic, diastolic, mean, and pulse pressure with morbidity and mortality in septic ICU patients: a nationwide observational study

Ashish K. Khanna^{1,2*}, Takahiro Kinoshita^{3†}, Annamalai Natarajan³, Emma Schwager³, Dustin D. Linn³, Junzi Dong³, Erina Ghosh³, Francesco Vicario³ and Kamal Maheshwari⁴

- **Retrospective observational study, 77,328** septic patients in 364 ICUs in the eICU
- **Primary exposure was the lowest cumulative value of each component; mean, systolic, diastolic, and pulse pressure, sustained for at least 120 min during ICU stay**

Predicted Probability of ICU Mortality in Septic Shock Patients (%)



In septic ICU patients, lower level of all blood pressure components including mean, systolic, diastolic and pulse pressure were associated with higher mortality, acute kidney injury and myocardial injury

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5. DAP is the upstream pressure for LV coronary perfusion
6. DAP is associated to outcome
- 7. DAP may be used as a trigger when managing septic shock patients**

Diastolic arterial pressure is important in septic shock: PRO

Olfa Hamzaoui Jean-Louis Teboul

Journal of Critical Care 51 (2019) 238–240

Patient of **70** years old and history of **CAD**
with **tachycardia** (heart rate: 100 beats/min)
and clinical signs of **septic shock** in spite of initial fluid resuscitation

Situation A



Situation B



DAP may be a trigger to start norepinephrine

Surviving Sepsis Campaign: International Guidelines for Management of Severe Sepsis and Septic Shock 2012

R. Phillip Dellinger, MD¹; Mitchell M. Levy, MD²; Andrew Rhodes, MB BS³; Djillali Annane, MD⁴; Herwig Gerlach, MD, PhD⁵; Steven M. Opal, MD⁶; Jonathan E. Sevransky, MD⁷; Charles L. Sprung, MD⁸; Ivor S. Douglas, MD⁹; Roman Jaeschke, MD¹⁰; Tiffany M. Osborn, MD, MPH¹¹; Mark E. Nunnally, MD¹²; Sean R. Townsend, MD¹³; Konrad Reinhart, MD¹⁴; Ruth M. Kleinpell, PhD, RN-CS¹⁵; Derek C. Angus, MD, MPH¹⁶; Clifford S. Deutschman, MD, MS¹⁷; Flavia R. Machado, MD, PhD¹⁸; Gordon D. Rubenfeld, MD¹⁹; Steven A. Webb, MB BS, PhD²⁰; Richard J. Beale, MB BS²¹; Jean-Louis Vincent, MD, PhD²²; Rui Moreno, MD, PhD²³; and the Surviving Sepsis Campaign Guidelines Committee including the Pediatric Subgroup*

Rationale. Vasopressor therapy is required to sustain life and maintain perfusion in the face of life-threatening hypotension, even when hypovolemia has not yet been resolved.

Adequate fluid resuscitation is a fundamental aspect of the hemodynamic management of patients with septic shock and should ideally be achieved before vasopressors and inotropes are used; however, using vasopressors early as an emergency measure in patients with severe shock is frequently necessary, as when diastolic blood pressure is too low.

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7. DAP may be used as a trigger when managing septic shock patients
8. **Development and use of markers of vascular hyporesponsiveness to NE:**

$$\text{VNERi} = \text{DAP} / (\text{HR} \times \text{NE dose})$$



Ability of diastolic arterial pressure to better characterize the severity of septic shock when adjusted for heart rate and norepinephrine dose

Antoine Goury^{1,2*}, Zoubir Djerada², Glenn Hernandez³, Eduardo Kattan³, Romain Griffon¹, Gustavo Ospina-Tascon^{4,5}, Jan Bakker^{6,7}, Jean-Louis Teboul^{2,8} and Olfa Hamzaoui^{1,2}

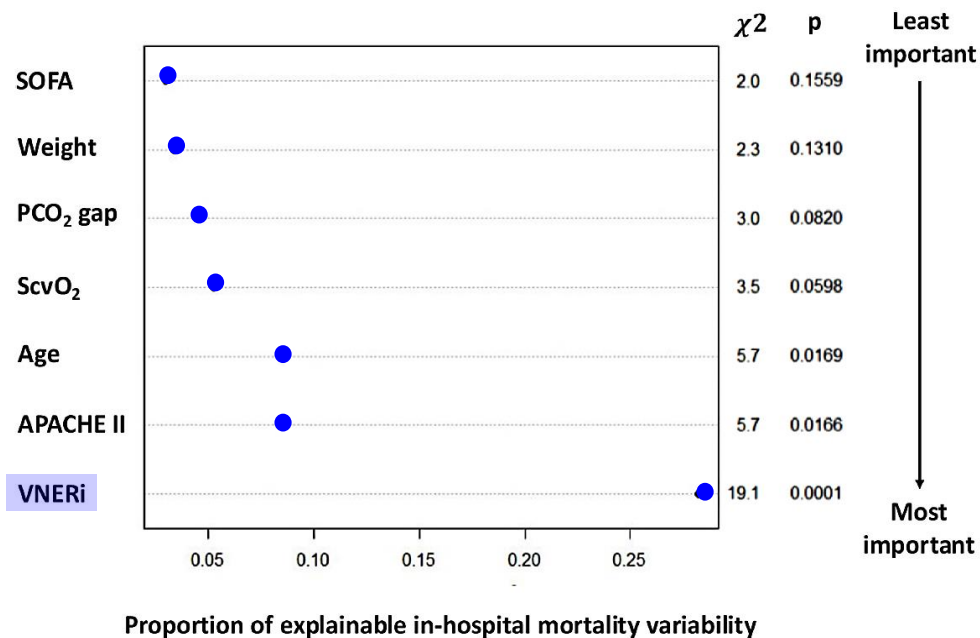
Annals of Intensive Care (2025) 15:43

$$\text{VNERi} = \text{DAP} / (\text{HR} \times \text{NE dose})$$

- **DAP**: marker of vascular tone
- **DAP/HR**: better marker of vascular tone than DAP



DAP/(HR x NE dose): marker of vascular responsiveness to NE





Ability of diastolic arterial pressure to better characterize the severity of septic shock when adjusted for heart rate and norepinephrine dose

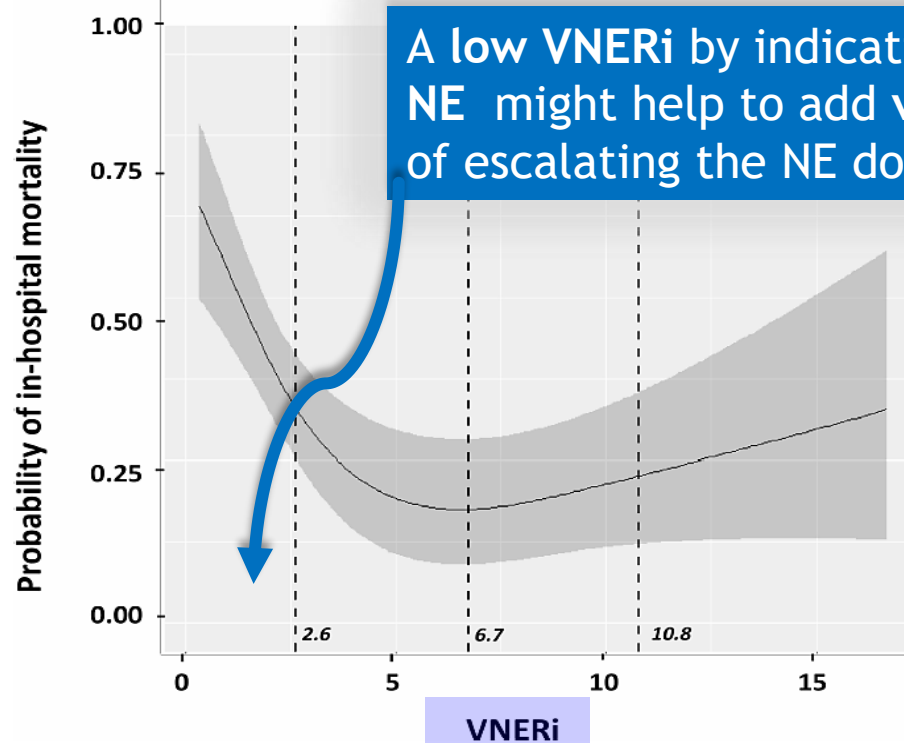
Antoine Goury^{1,2*}, Zoubir Djerada², Glenn Hernandez³, Eduardo Kattan³, Romain Griffon¹, Gustavo Ospina-Tascon^{4,5}, Jan Bakker^{6,7}, Jean-Louis Teboul^{2,8} and Olfa Hamzaoui^{1,2}

Annals of Intensive Care (2025) 15:43

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$$\text{VNERi} = \text{DAP} / (\text{HR} \times \text{NE dose})$$

DAP/(HR x NE dose): marker of vascular responsiveness to NE



A low VNERi by indicating hyporesponsiveness to NE might help to add vasopressin earlier instead of escalating the NE dose

Is it important to consider DAP in septic shock ?

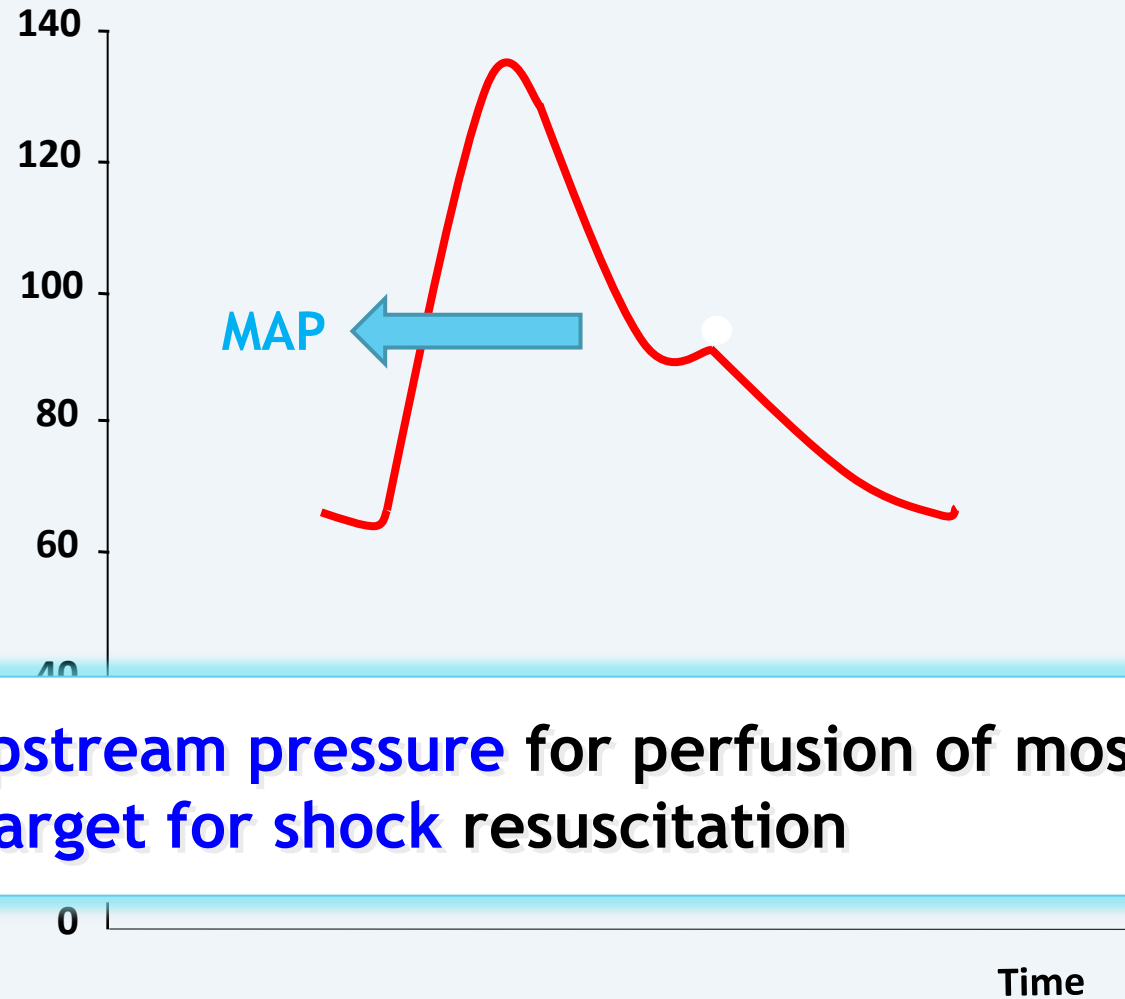
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$$\text{VNERi} = \text{DAP} / (\text{HR} \times \text{NE dose}) < 2.6$$



Add a second vasopressor: Vasopressin

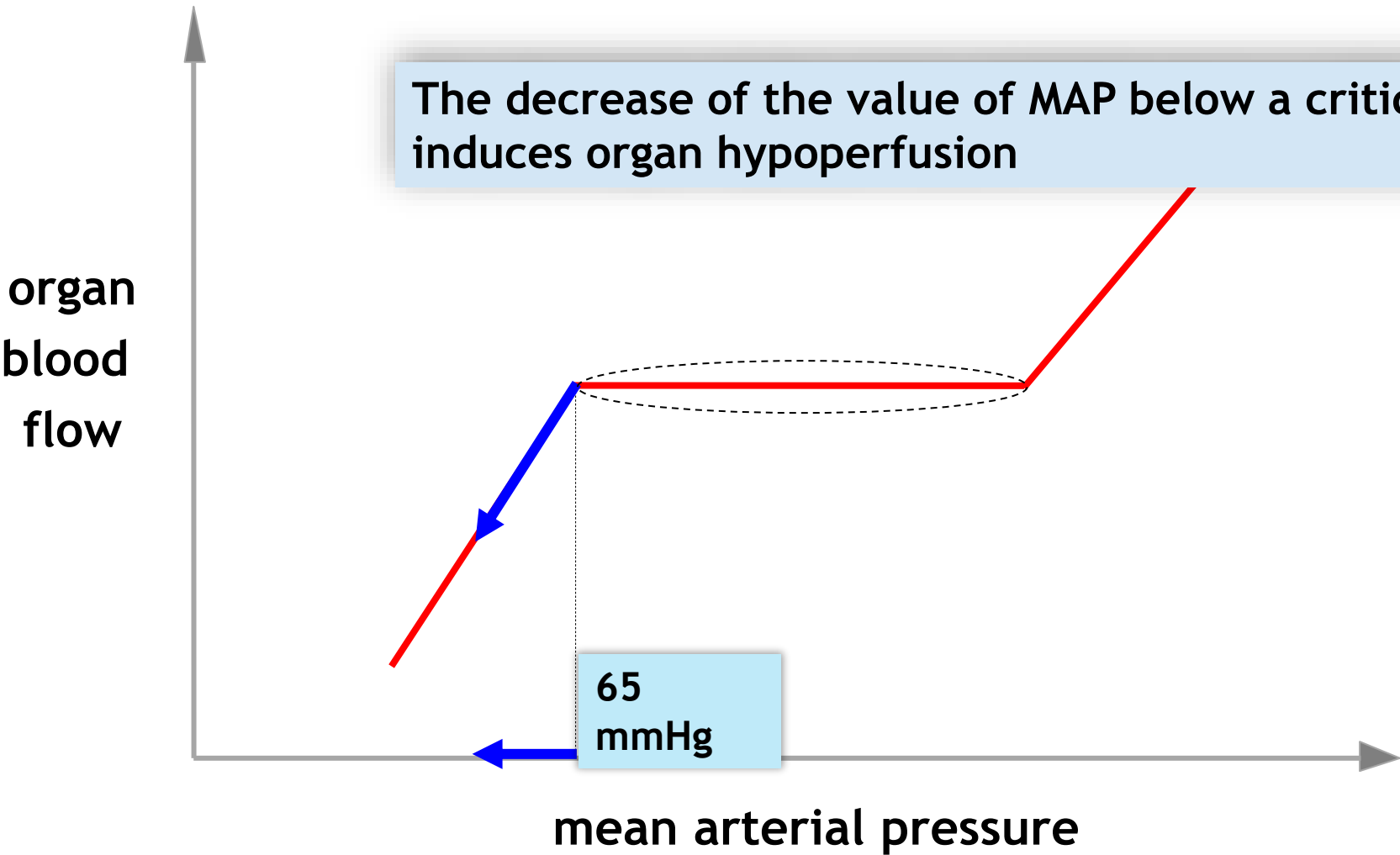
Arterial pressure (mmHg)



MAP: upstream pressure for perfusion of most vital organs
And a target for shock resuscitation

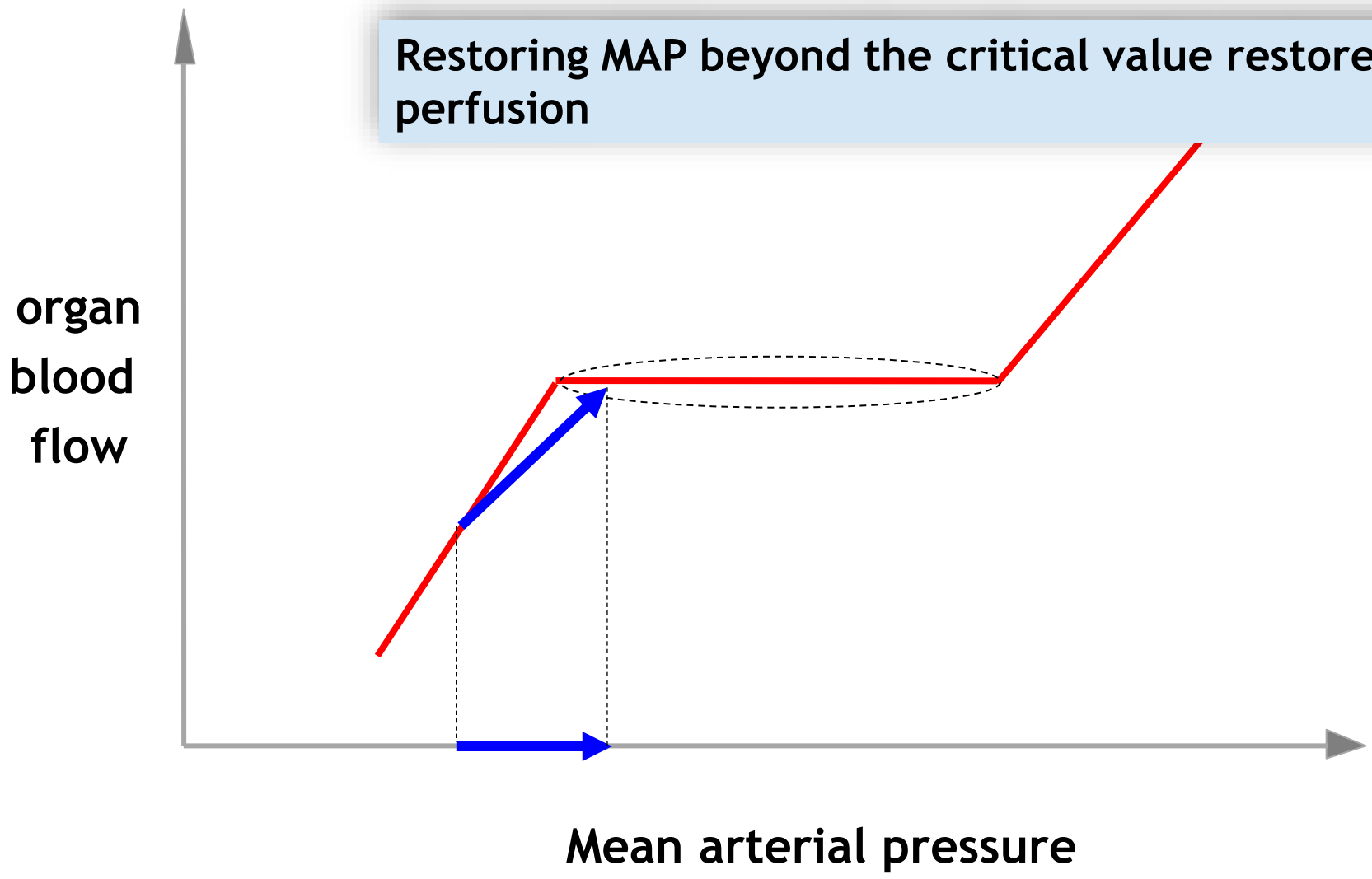
Autoregulation of organ blood flow

The decrease of the value of MAP below a critical value induces organ hypoperfusion



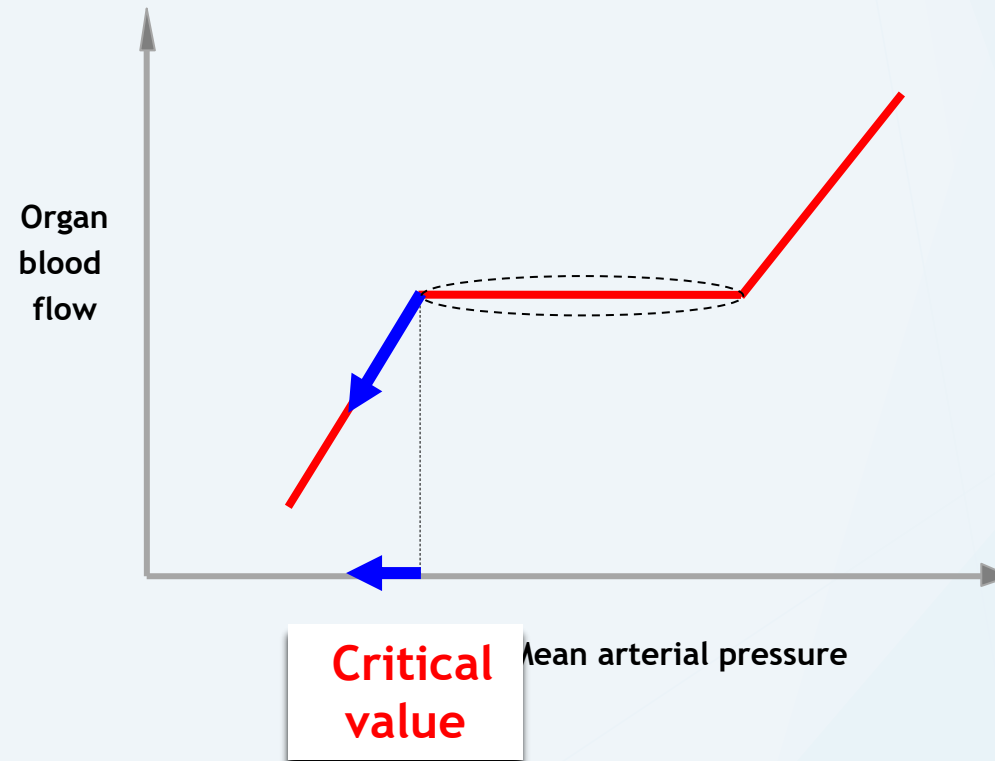
Autoregulation of organ blood flow

Restoring MAP beyond the critical value restores organs perfusion



MAP: Therapeutic goal

What is the cut off value?



GUIDELINES

Surviving sepsis campaign: international guidelines for management of sepsis and septic shock 2021



Intensive Care Med (2021) 47:1181–1247

MEAN ARTERIAL PRESSURE



MODERATE

9

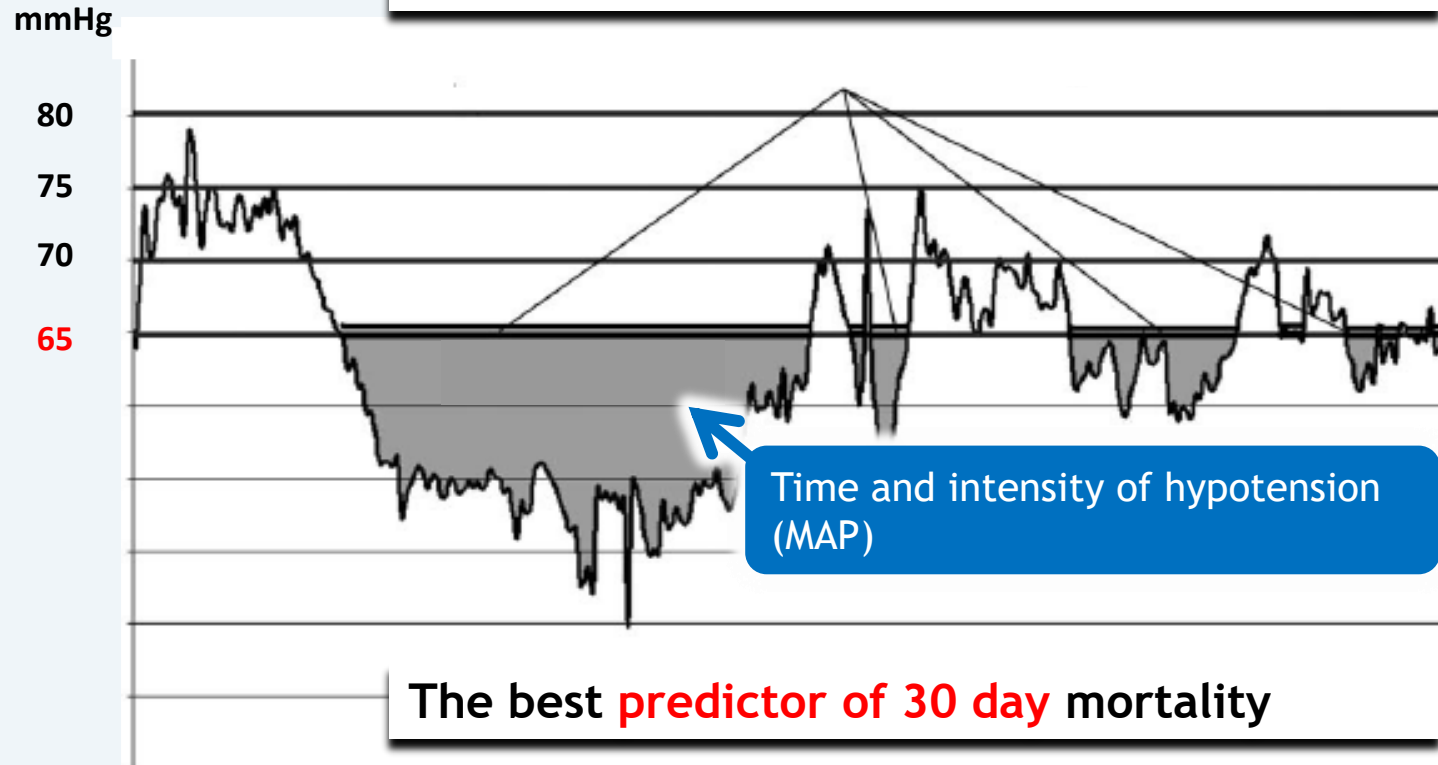
For adults with septic shock on vasopressors, we **recommend** an initial target mean arterial pressure (MAP) of 65 mm Hg over higher MAP targets.

Marjut Varpula
Minna Tallgren
Katri Saukkonen
Liisa-Maria Voipio-Pulkki
Ville Pettilä

Hemodynamic variables related to outcome in septic shock

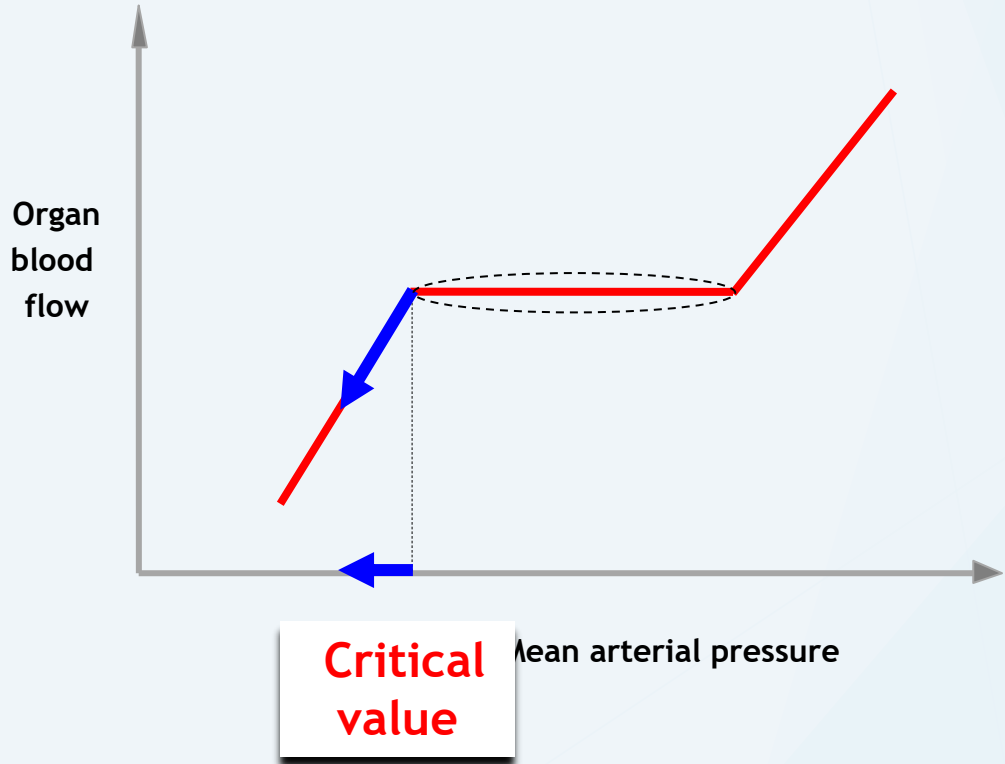
Retrospective cohort
111 septic shock patients
30-day mortality

Time under the value of **MAP 65 mmHg**



MAP: Treatment goal

What is the cut off value?
Is it the same for all the patients ?

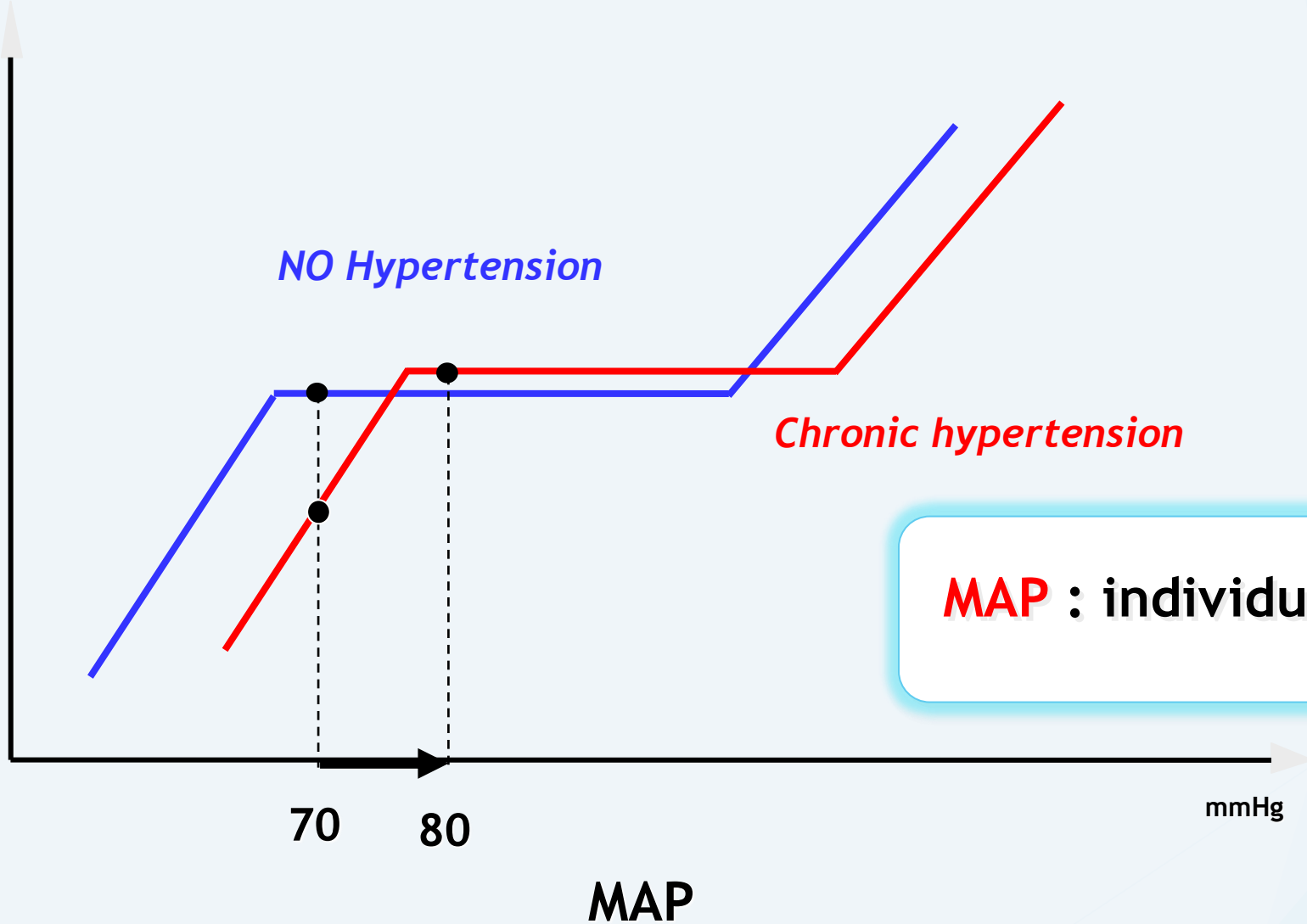


Autoregulation of Brain Circulation in Severe Arterial Hypertension

S. STRANDGAARD, J. OLESEN, E. SKINHØJ, N. A. LASSEN

British Medical Journal, 1973, 1, 507-510

Organ blood flow



MAP : individualize

The **NEW ENGLAND**
JOURNAL *of* **MEDICINE**

80-85 mmHg

ESTABLISHED IN 1812

APRIL 24, 2014

VOL. 370 NO. 16

65-70 mmHg

High versus Low Blood-Pressure Target in Patients with Septic Shock

Pierre Asfar, M.D., Ph.D., Ferhat Meziani, M.D., Ph.D., Jean-François Hamel, M.D., Fabien Grelon, M.D., Bruno Megarbane, M.D., Ph.D., Nadia Anguel, M.D., Jean-Paul Mira, M.D., Ph.D., Pierre-François Dequin, M.D., Ph.D., Soizic Gergaud, M.D., Nicolas Weiss, M.D., Ph.D., François Legay, M.D., Yves Le Tulzo, M.D., Ph.D., Marie Conrad, M.D., René Robert, M.D., Ph.D., Frédéric Gonzalez, M.D., Christophe Guitton, M.D., Ph.D., Fabienne Tamion, M.D., Ph.D., Jean-Marie Tonnelier, M.D., Pierre Guezennec, M.D., Thierry Van Der Linden, M.D., Antoine Vieillard-Baron, M.D., Ph.D., Eric Mariotte, M.D., Gaël Pradel, M.D., Olivier Lesieur, M.D., Jean-Damien Ricard, M.D., Ph.D., Fabien Hervé, M.D., Damien du Cheyron, M.D., Ph.D., Claude Guerin, M.D., Ph.D., Alain Mercat, M.D., Ph.D., Jean-Louis Teboul, M.D., Ph.D., and Peter Radermacher, M.D., Ph.D.,

388 pts

388 pts



High versus Low Blood-Pressure Target in Patients with Septic Shock

Pierre Asfar, M.D., Ph.D., Ferhat Meziani, M.D., Ph.D., Jean-François Hamel, M.D., Fabien Grelon, M.D., Bruno Megarbane, M.D., Ph.D., Nadia Anguel, M.D., Jean-Paul Mira, M.D., Ph.D., Pierre-François Dequin, M.D., Ph.D., Soizic Gergaud, M.D., Nicolas Weiss, M.D., Ph.D., François Legay, M.D., Yves Le Tulzo, M.D., Ph.D., Marie Conrad, M.D., René Robert, M.D., Ph.D., Frédéric Gonzalez, M.D., Christophe Guitton, M.D., Ph.D., Fabienne Tamion, M.D., Ph.D., Jean-Marie Tonnelier, M.D., Pierre Guezennec, M.D., Thierry Van Der Linden, M.D., Antoine Vieillard-Baron, M.D., Ph.D., Eric Mariotte, M.D., Gaël Pradel, M.D., Olivier Lesieur, M.D., Jean-Damien Ricard, M.D., Ph.D., Fabien Hervé, M.D., Damien du Cheyron, M.D., Ph.D., Claude Guerin, M.D., Ph.D., Alain Mercat, M.D., Ph.D., Jean-Louis Teboul, M.D., Ph.D., and Peter Radermacher, M.D., Ph.D.

Variable	Low-Target Group (N = 388)	High-Target Group (N = 388)	P Value
Primary outcome: death at day 28 — no. (%) [*]	132 (34.0)	142 (36.6)	0.57
Secondary outcomes — no./total no. (%)			
Death at day 90 [†]	164 (42.3)	170 (43.8)	0.74
Survival at day 28 without organ support [‡]	241 (62.1)	235 (60.6)	0.66
Doubling of plasma creatinine	161 (41.5)	150 (38.7)	0.42
No chronic hypertension	71/215 (33.0)	85/221 (38.5)	0.32
Chronic hypertension	90/173 (52.0)	65/167 (38.9)	0.02
Renal-replacement therapy from day 1 to day 7	139 (35.8)	130 (33.5)	0.50
No chronic hypertension	66/215 (30.7)	77/221 (34.8)	0.36
Chronic hypertension	73/173 (42.2)	53/167 (31.7)	0.046
Atrial fibrillation	11 (2.8)	26 (6.7)	0.02
Ventricular fibrillation or tachycardia	15 (3.9)	22 (5.7)	0.24
Digital ischemia	9 (2.3)	10 (2.6)	0.82
Mesenteric ischemia	9 (2.3)	9 (2.3)	1.00
Bleeding	42 (10.8)	31 (8.0)	0.22

Equilibrating SSC guidelines with individualized care



Jean-Louis Vincent^{1*} , Mervyn Singer², Sharon Einav³, Rui Moreno⁴ , Julia Wendon⁵, Jean-Louis Teboul⁶, Jan Bakker^{7,8,9,10}, Glenn Hernandez¹¹, Djillali Annane¹², Angélique M. E. de Man¹³, Xavier Monnet¹⁴, V. Marco Ranieri¹⁵, Olfa Hamzaoui¹⁶, Jukka Takala¹⁷, Nicole Juffermans^{18,19}, Jean-Daniel Chiche²⁰, Sheila N. Myatra²¹ and Daniel De Backer²²

Critical Care (2021) 25:397



We recommend **individualizing** arterial **blood pressure** levels.

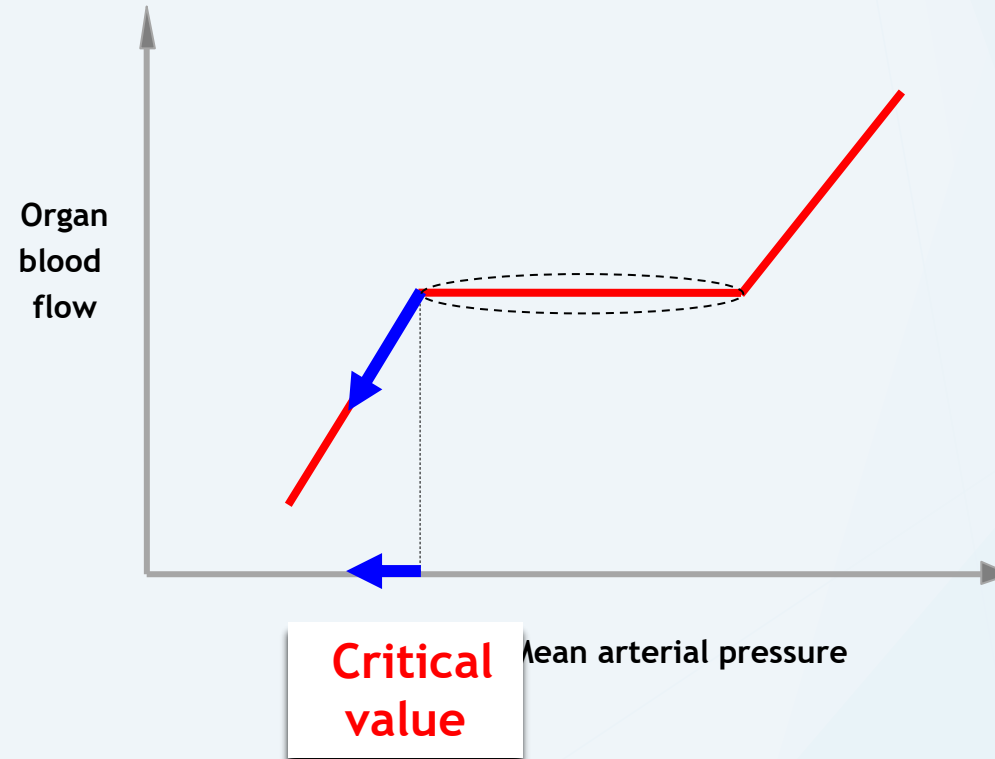
Although a mean value of **65 mmHg** may be recommended as an **initial goal**, the optimal level may be **higher** in patients with a history of **hypertension**, **atherosclerosis** or **chronic kidney disease**

MAP: Treatment goal

What is the cut off value?

Is it the same for all the patients ?

Is it the same in all the situations ?



SPECIAL ISSUE INSIGHT

Central venous pressure (CVP)

Olfa Hamzaoui^{1*}  and Jean-Louis Teboul^{2,3}



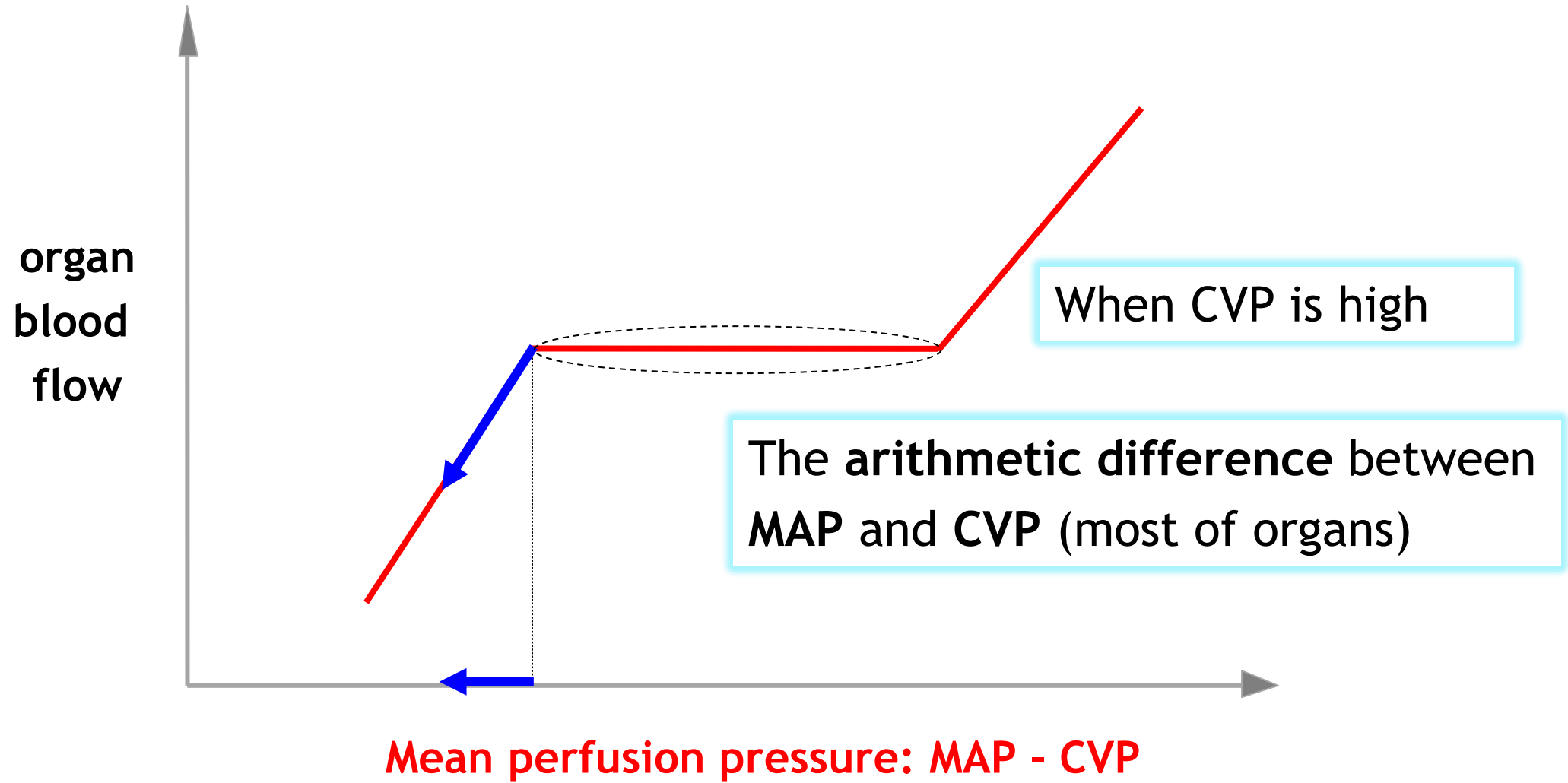
Intensive Care Med (2022) 48:1498–1500

CVP as the downstream pressure for organ perfusion

The **CVP** also reflects the **downstream pressure for perfusion of most vital organs** (e.g., brain and kidney). The **mean perfusion pressure (MPP)** of such organs is the **difference between mean arterial pressure (MAP) and CVP**.

MPP = MAP - CVP  **MAP may be a surrogate of MPP when CVP is low**

Autoregulation of organ blood flow



Low mean perfusion pressure is a risk factor for progression of acute kidney injury in critically ill patients – A retrospective analysis

Marlies Ostermann^{1*}, Anna Hall² and Siobhan Crichton³

BMC Nephrology (2017) 18:151

- Retrospective analysis
- 2118 patients: 790 patients (37%) who developed **AKI**
- 205 equipped with advanced hemodynamic monitoring.

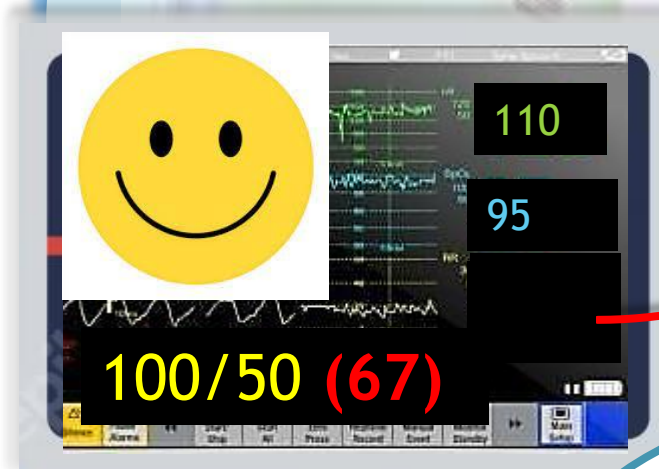
Table 2 Multivariable analysis: Risk factors for progression from AKI I to AKI III

Parameter	OR (95% CI) ^a	p-value
First arterial lactate following diagnosis of AKI I [mmol/L]	1.45 (1.12–1.89)	0.005
SOFA score on day of AKI I	1.20 (1.05–1.37)	0.01
First DO ₂ I in 12 h period after diagnosis of AKI I [ml/min/m ²]	0.997 (0.994–0.99)	0.01
First calculated MPP	0.995 (0.92–0.99)	0.03
Age [years]	1.02 (0.997–1.05)	0.09
Cumulative fluid balance on day of AKI I [ml]	1.00 (0.99–1.00)	0.98
MAP <65 mmHg for >1 h in first 12 h after diagnosis of AKI I	0.97 (0.48–1.96)	0.93

Mean perfusion pressure (MPP = MAP - CVP) but not MAP was an independent factor associated with AKI progression. A value of **MPP of 60 mmHg** was found as a cutoff.

Patient with pneumonia
ARDS and under mechanical
ventilation

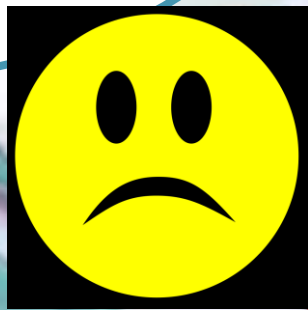
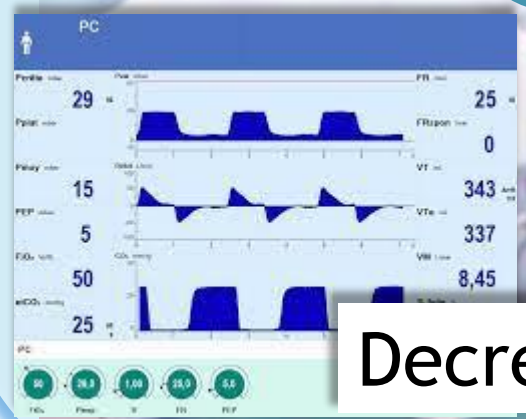
- Under NE: 0.4 microg/Kg/min
- Acute renal failure



$$\text{MPP} = \text{MAP} - \text{CVP}$$

$$\text{MPP} = 67 - 25$$

$$\text{MPP} = 42 \text{ mmHg}$$

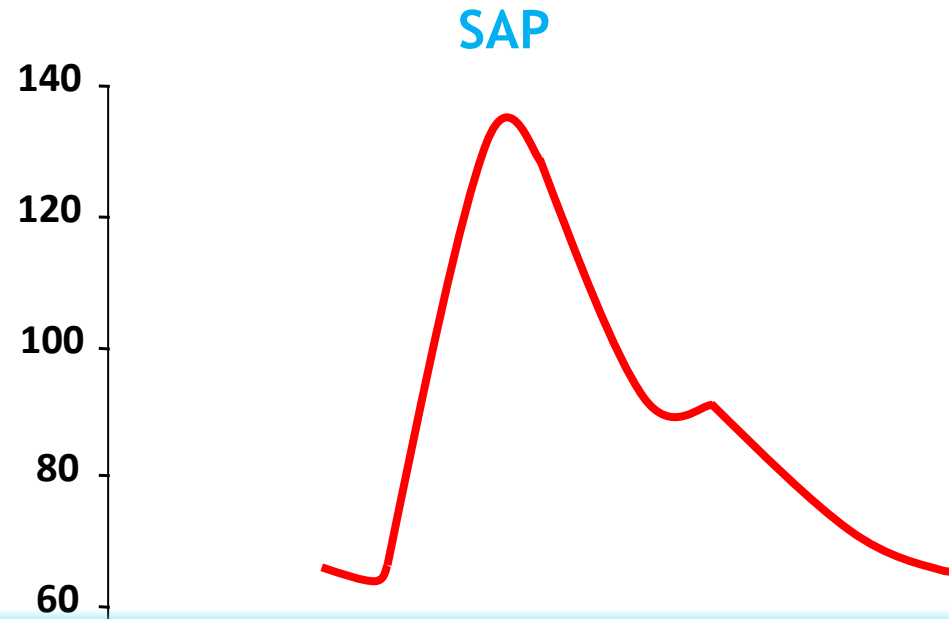


Decrease PEEP

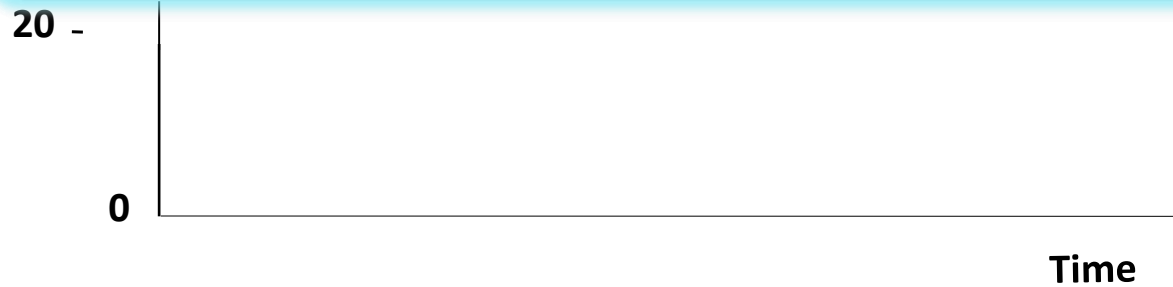
Increase NE



Arterial pressure (mmHg)

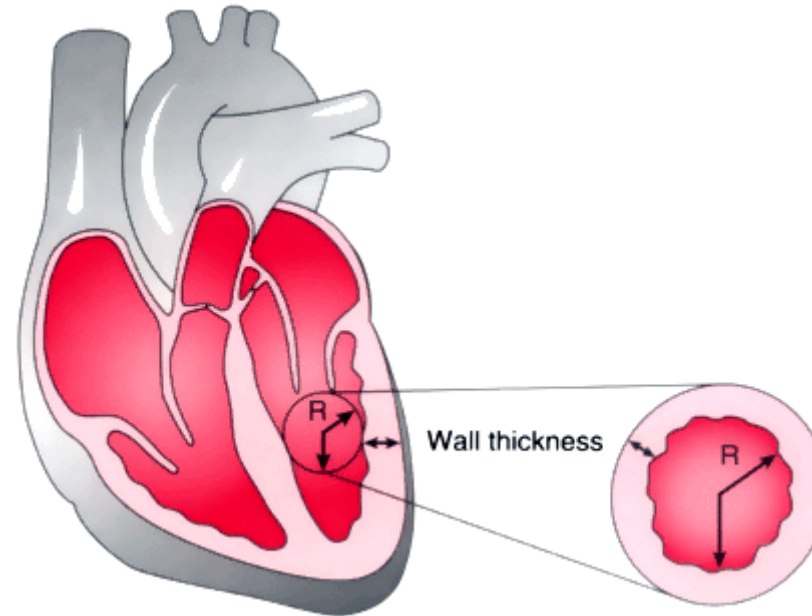


Is **peripheral SAP** an acceptable reflection of LV afterload?



LV afterload is defined by systolic wall stress

LV afterload is defined by the load that the LV must eject blood against and expressed as **systolic wall stress**



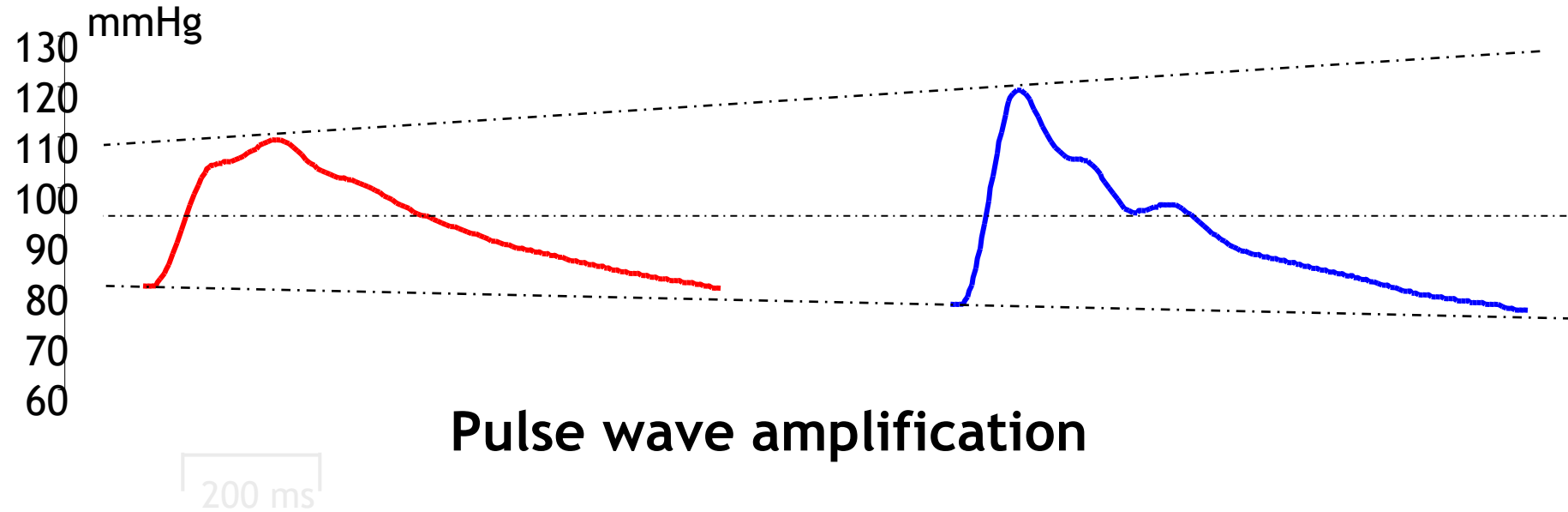
Laplace law

Systolic wall stress = ventricular systolic pressure \times LV radius / 2 \times LV wall thickness

Ventricular end-systolic pressure = 0.9 \times aortic SAP

Central pressure wave

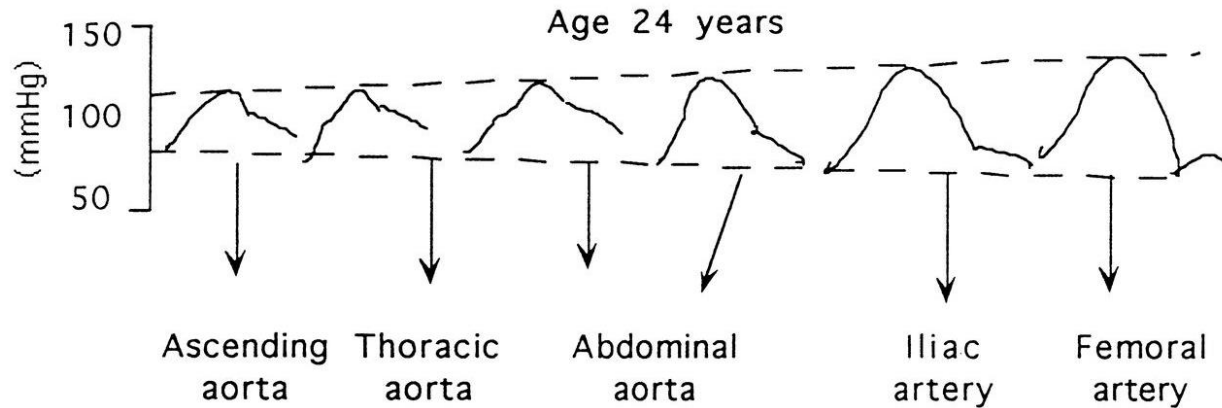
Peripheral pressure wave



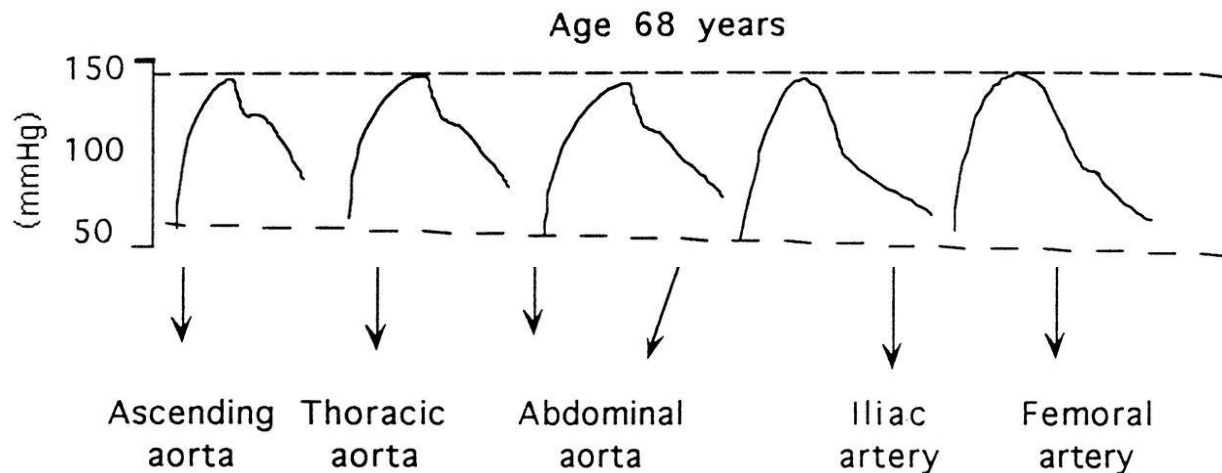
aortic SAP < **femoral SAP**

femoral SAP < **radial SAP**

Pulse wave amplification depends on age and arterial stiffness

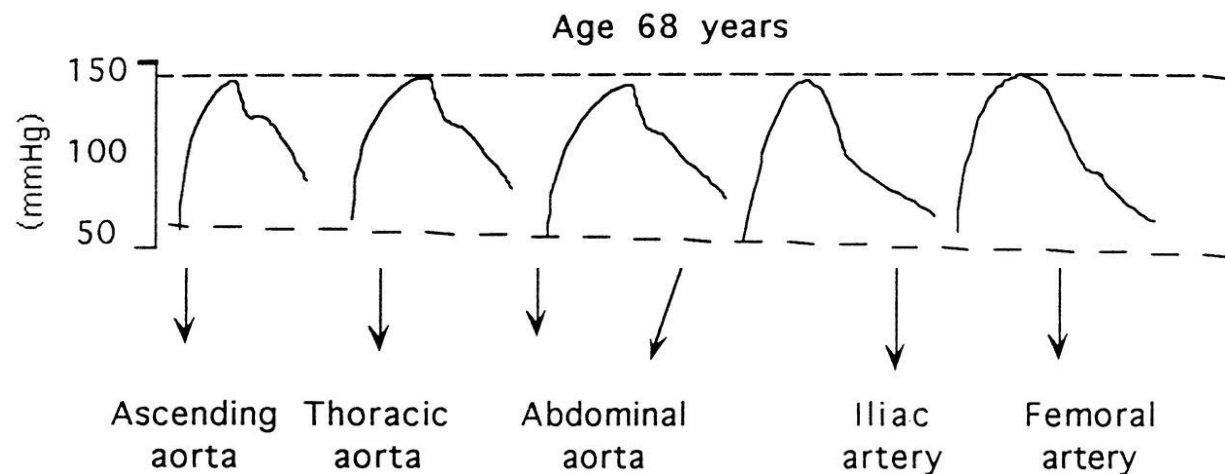


If you measure a **peripheral SAP** of **150** mmHg
the aortic SAP is lower
... and LV afterload not so much increased



If you measure a **peripheral SAP** of **150** mmHg
the aortic SAP is 150 and LV afterload is high

Pulse wave amplification depends on age and arterial stiffness



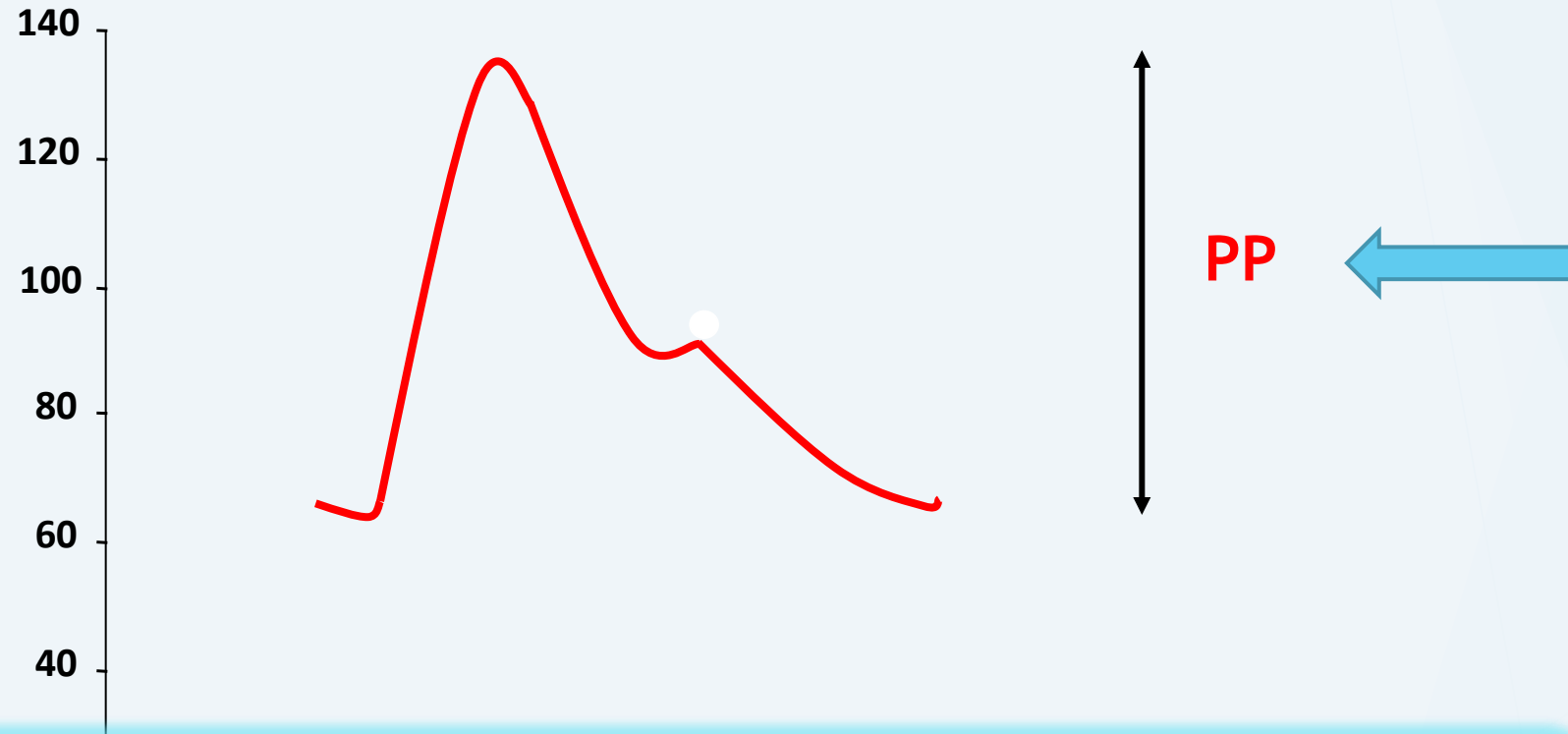
→ If you measure a **peripheral SAP** of **150** mmHg the aortic SAP is 150 and LV afterload is high

• Is **peripheral SAP** an acceptable reflection of LV afterload ?



- Yes, if low pulse wave amplification
- (**elderly, hypertensive, vasoconstricted**, etc)

Arterial pressure (mmHg)



Aortic pulse pressure = $SV / \text{aortic compliance}$

Chemla et al AJP 1998

Nicolas Dufour
 Denis Chemla
 Jean-Louis Teboul
 Xavier Monnet
 Christian Richard
 David Osman

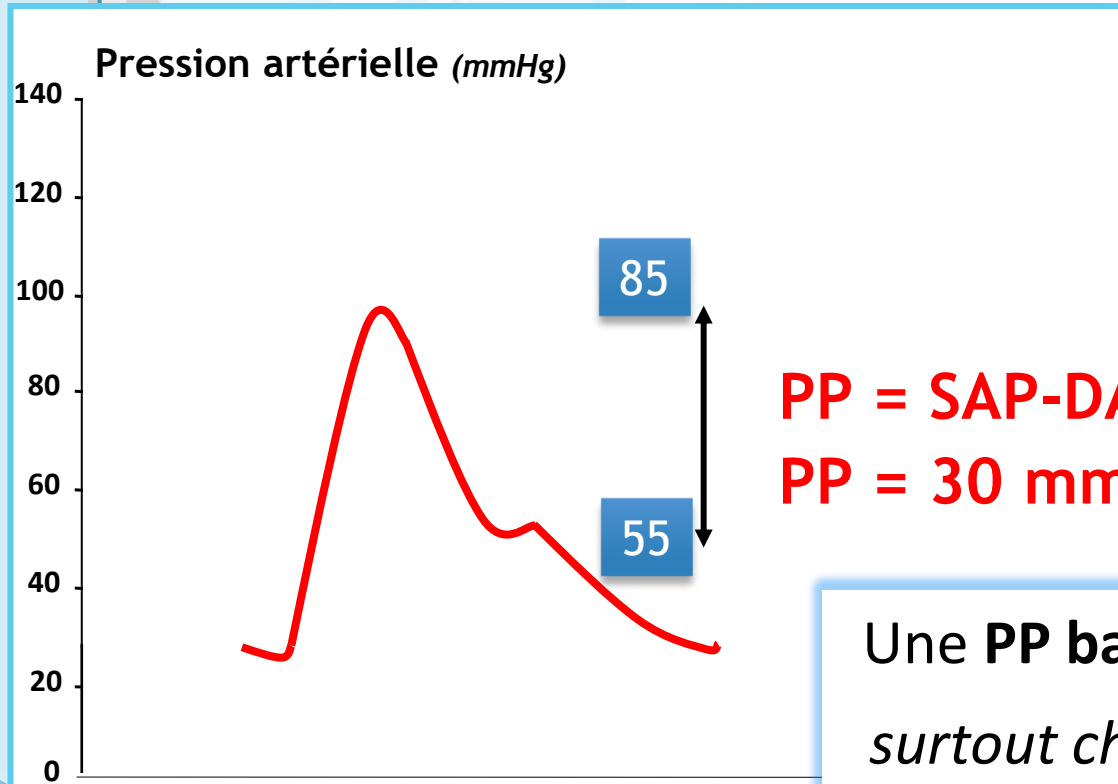
Changes in pulse pressure following fluid loading: a comparison between aortic root (non-invasive tonometry) and femoral artery (invasive recordings)

Easy bedside tool to identify low stroke volume

Table 5 Relationship between changes in PP and SV after volume expansion in young and elderly patients

PP versus SV relationship	Age <60 years		Age ≥60 years	
	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>
Femoral	0.46	0.03	0.75	<0.01

- Male (Mr Z)
- **75 years old**
- Medical history: **Hypertension**



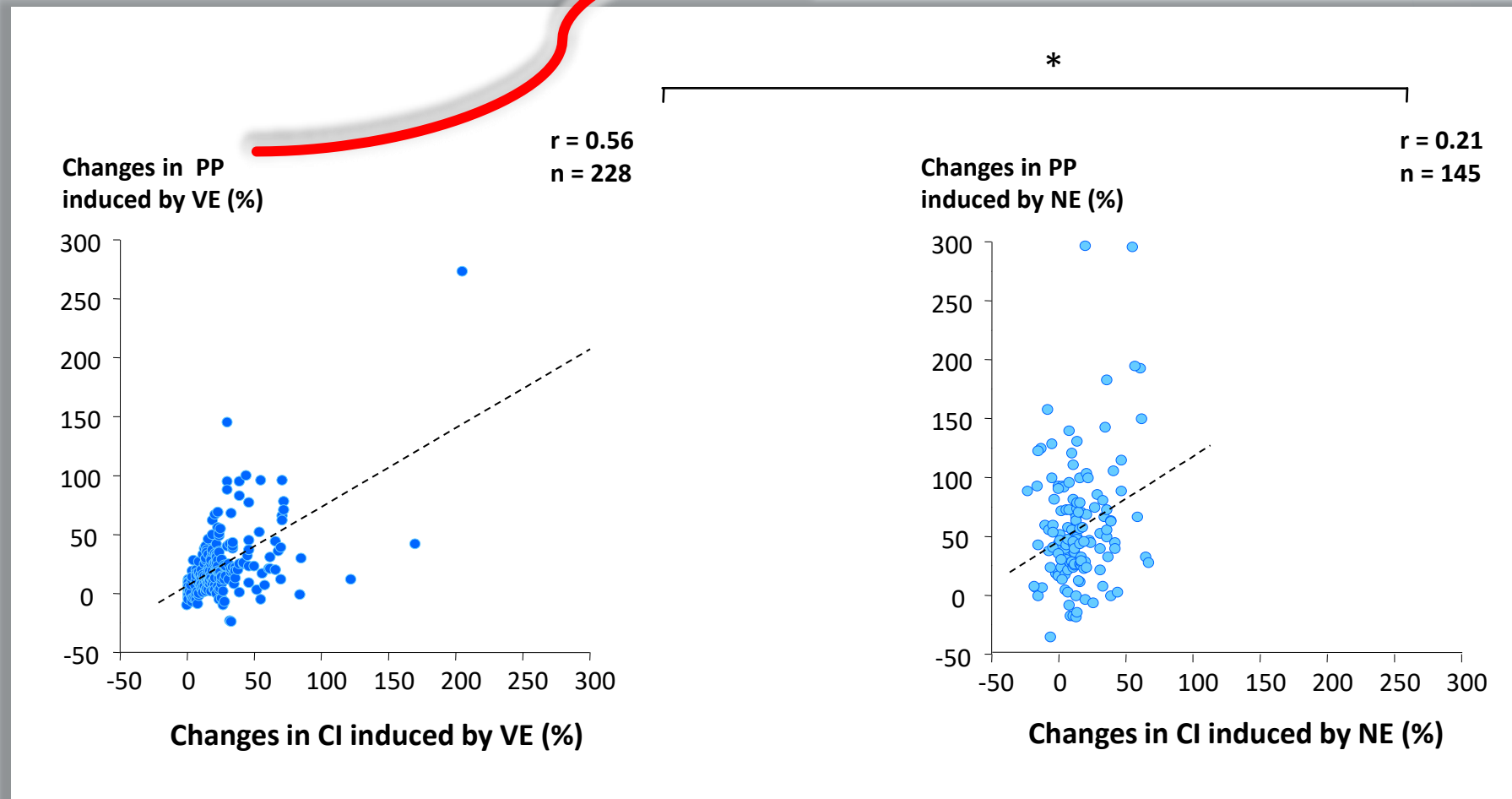
Une **PP basse** reflète un **bas volume d'éjection systolique** surtout chez les patients avec artères (supposées) rigides

Arterial pressure allows monitoring the changes in cardiac output induced by volume expansion but not by norepinephrine*

Xavier Monnet, MD, PhD; Alexia Letierce, PhD; Olfa Hamzaoui, MD; Denis Chemla, MD, PhD; Nadia Anguel, MD; David Osman, MD; Christian Richard, MD; Jean-Louis Teboul, MD, PhD

Crit Care Med 2011; 39:1394–1399

Easy bedside tool to test changes in low stroke volume



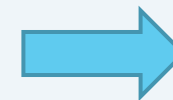
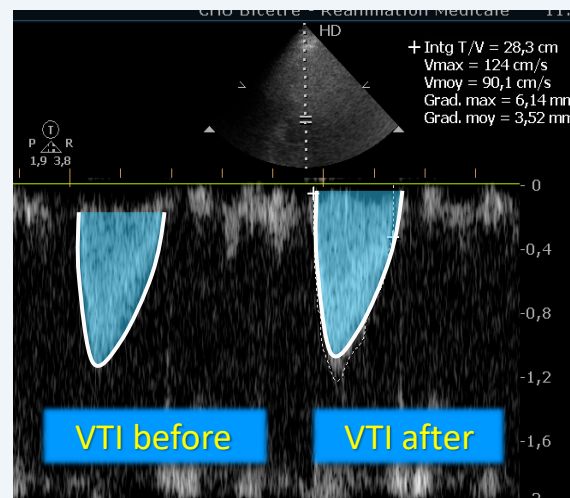
Dynamic changes of pulse pressure but not of pulse pressure variation during passive leg raising predict preload responsiveness in critically ill patients with spontaneous breathing activity

Rui Shi, MD, PhD^{a,b}, Francesca Moretto, MD^a, Dominique Prat, MD^c, Frederic Jacobs, MD^c, Jean-Louis Teboul, MD, PhD^{a,b}, Olfa Hamzaoui, MD^{c,*}

Journal of Critical Care 72 (2022) 154141

33 Patients ventilated with **pressure support mode or totally spontaneously breathing**

To **evaluate** whether **changes** in **pulse pressure (PP)** or in **pulse pressure variation (PPV)** during (PLR) can be used to evaluate preload responsiveness



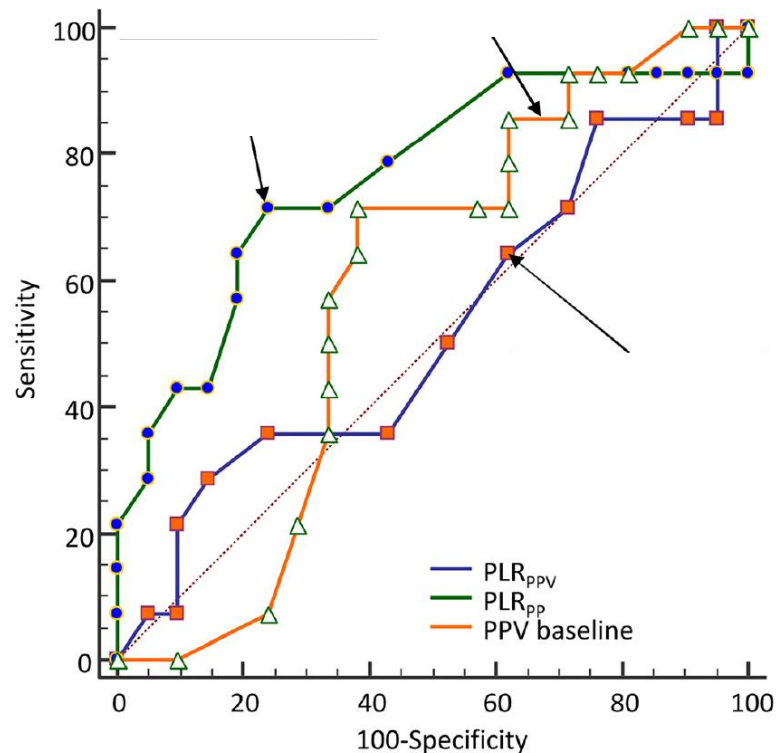
VTI >12%: PLR +

Dynamic changes of pulse pressure but not of pulse pressure variation during passive leg raising predict preload responsiveness in critically ill patients with spontaneous breathing activity

Rui Shi, MD, PhD^{a,b}, Francesca Moretto, MD^a, Dominique Prat, MD^c, Frederic Jacobs, MD^c, Jean-Louis Teboul, MD, PhD^{a,b}, Olfa Hamzaoui, MD^{c,*}

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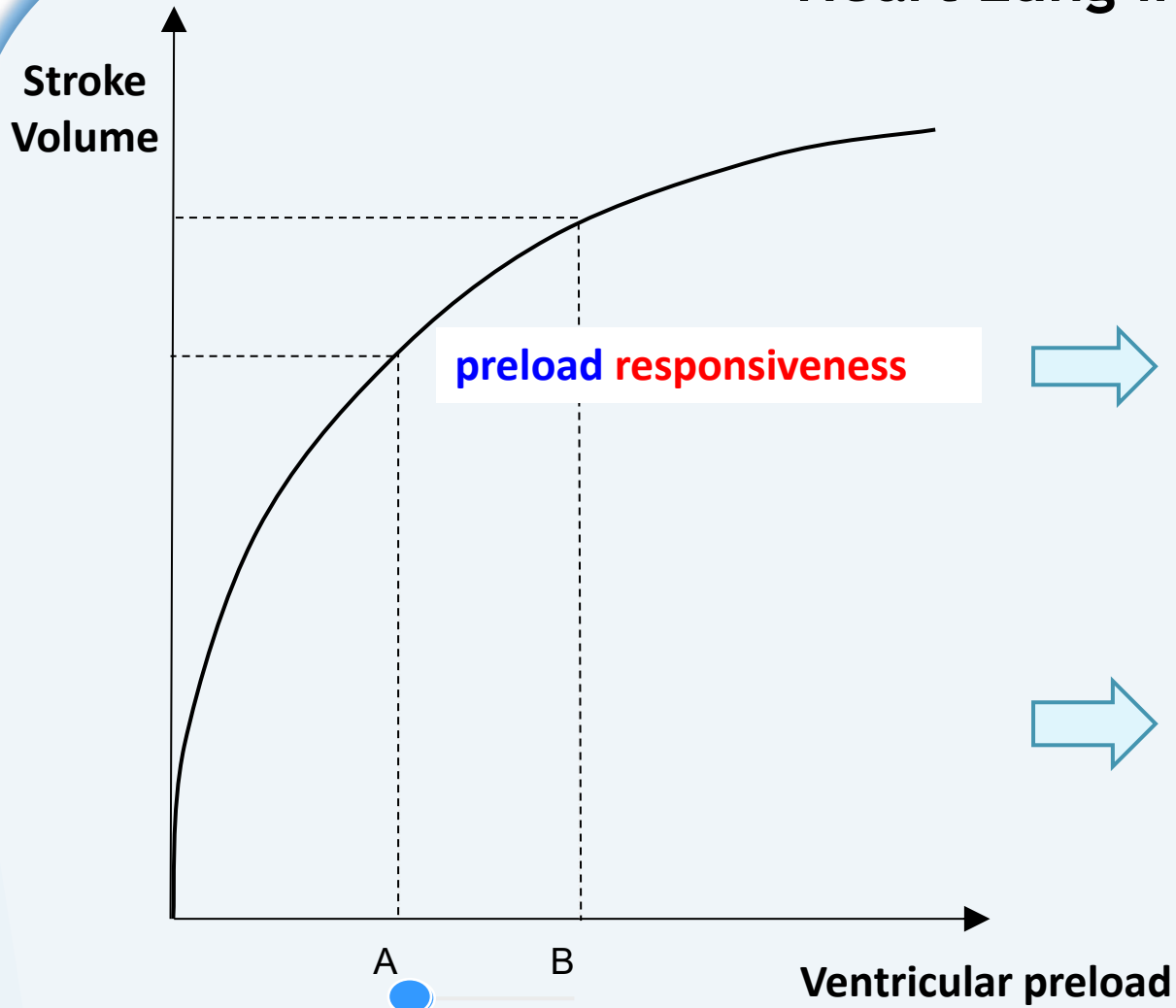
33 Patients ventilated with **pressure support mode or totally spontaneously breathing**



in patients with spontaneous breathing activity, **the increase in PP of equal to or higher than 2 mmHg during PLR** may be helpful to discriminate preload responders from non-responders with fair accuracy

- Analysis of the static values
- **Analysis of the dynamic variation of AP values**

Heart Lung interaction during MV



preload responsiveness

MV can induce cyclic changes in preload and ... in SV in cases of biventricular preload responsiveness

As aortic compliance does not change over the respiratory cycle, changes in PP (PPV) reflect changes in SV (SVV)

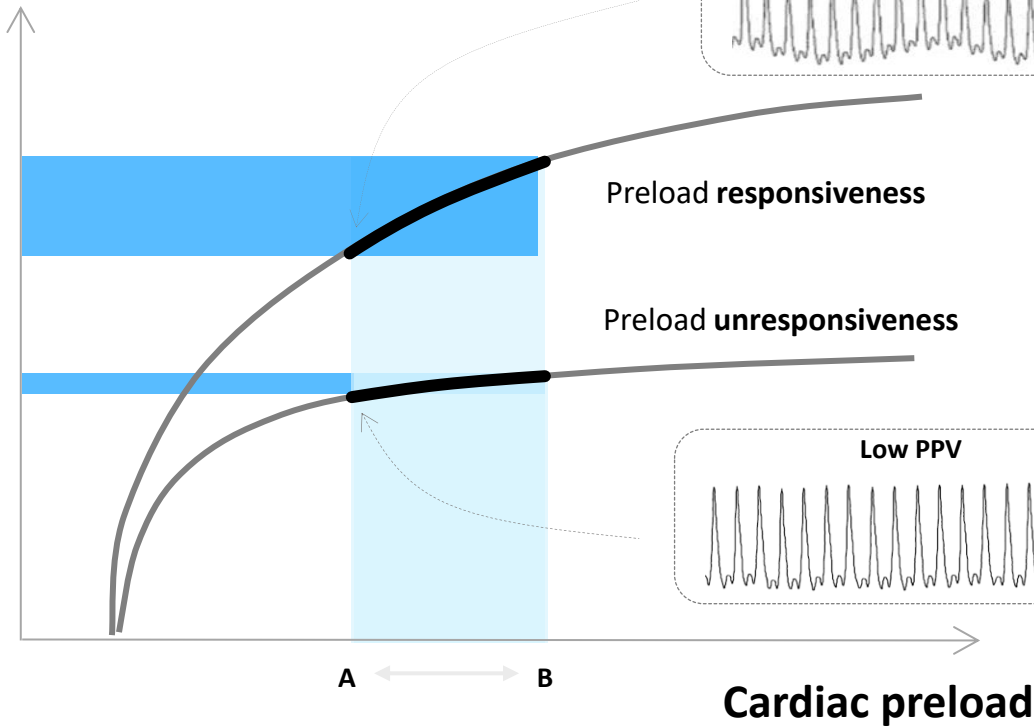


Arterial Pulse Pressure Variation with Mechanical Ventilation

Jean-Louis Teboul¹, Xavier Monnet¹, Denis Chemla², and Frédéric Michard³

Am J Respir Crit Care Med Vol 199, Iss 1, pp 22–31, Jan 1, 2019

Stroke
volume



Applied physiology

Applicability of pulse pressure variation: how many shades of grey?

Frederic Michard^{1*}, Denis Chemla² and Jean-Louis Teboul³

Critical Care (2015) 19:144

	False positive	False negative
L Low HR/RR ratio (Extreme bradycardia or high frequency ventilation)		✓
I Irregular heart beats	✓	
M Mechanical ventilation with low tidal volume		
I Increased abdominal Pressure (Pneumoperitoneum)	✓	
T Thorax open		✓
S Spontaneous breathing	✓	✓

Be Aware of the Limitations !

Take home messages?

Arterial pressure is a real time monitoring tool

❑ All the static components of arterial pressure (SAP, DAP, MAP, PP) are **important to consider** when managing critically ill patients

- SAP : reflects **LV afterload**
- DAP : may be used as a « trigger » to introduce vasopressors
- **VNERi =DAP/HRxDose de NAD**: may be used as a « trigger » to add a second vasopressor
- MAP : **Therapeutic target**
- PP : may be used as **surrogate of stroke volume**

Merci !

❑ The **variation of PP(delta PP)** under MV may be used as a marker of preload dependency

DU Hemodynamic management and monitoring of patients with shock (OPENING SOON)

Professor Olfa HAMZAOU

Merci !

Scientific Committee



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