# **Clinical case**

**Prof. Jean-Louis TEBOUL** 

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#### **Conflicts of interest**

- Member of the Medical Advisory Board of Getinge
- Lectures for Edwards LifeSciences
- Lectures for Masimo

Mr R., **68** y.o.

- Medical history:
  - Chronic hypertension
  - COPD
- Cough and fever for three days,
- Increasing dyspnea since the last day

## at the ER (H<sub>0</sub>)

- mental confusion
- mottling
- crackles (right lung >left lung)
- fever 39° C

- RR = **40** /min
- HR = **104** /min
- AP = 69/34 (45) mmHg
- $SpO_2 = 90\% (O_2 10L/min)$

You decide to **intubate** the patient and infuse **500 mL crystalloids**.

Do you initiate norepinephrine at this stage?

 $Q_1$ 

- 1. Yes
- 2. No
- 3. I don't know

## at the ER (H<sub>0</sub>)

- mental confusion
- mottling
- crackles (right lung >left lung)
- fever 39° C

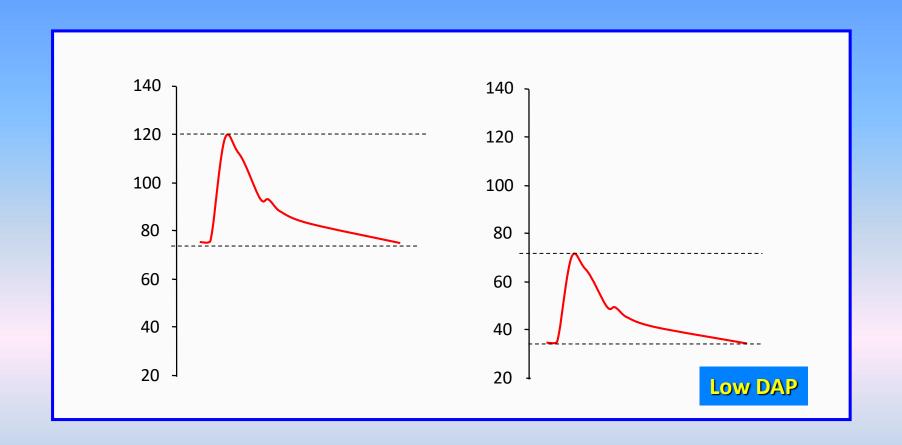
- RR = **40** /min
- HR = **104** /min
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- $SpO_2 = 90\% (O_2 10L/min)$

You decide to **intubate** the patient and infuse **500 mL crystalloids**.

Do you initiate **norepinephrine** at this stage?

 $Q_1$ 

- 1. Yes
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suggests a **decreased** arterial **tone**suggests the **septic** origin of shock
incites to administer a **vasopressor** 

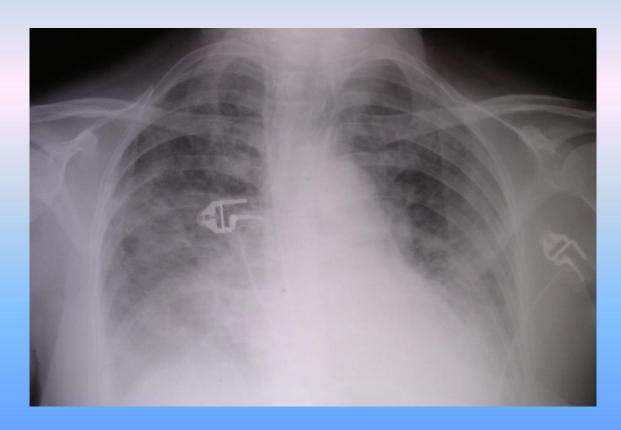
#### Take-home messages

1- During **septic** shock, **norepinephrine** should be initiated **early** when arterial **tone** is assumed to be **low**, e.g. when **DAP** is **low** (< 40 mmHg)

## at the ER (H<sub>0</sub>)

Lactate	<b>4.2</b> mmol/L
Urea	<b>23</b> mmol/L
Creat.	<b>210</b> μmol/L
CRP	<b>210</b> mg/L

(before intubation)	
рН	7.25
PaO <sub>2</sub>	<b>78</b> mmHg
PaCO <sub>2</sub>	<b>42</b> mmHg
HCO <sub>3</sub> -	<b>19</b> mmol/L



HR	<b>103</b> /min
AP	<b>80/38 (52)</b> mmHg
CVP	<b>10</b> mmHg
PPV	<b>7</b> %

**4.1** mmol/L

crystalloids	<b>750</b> mL
NE	<b>0.25</b> μg/kg/min
Propofol	<b>150</b> mg/h
Ceftriaxone + Levofloxacin	

RR	<b>18</b> /min
TV	<b>400</b> mL (6 mL/kg)
Pplat	<b>25</b> cmH <sub>2</sub> O
PEEP	<b>10</b> cmH <sub>2</sub> O
P/F	190

What do you do in terms of **treatment**?

- 1. Infuse fluids, first
- 2. Increase NE dose, first
- **3. Do both** at the same time
- 4. Dobutamine
- 5. Nothing else

 $Q_2$ 

HR	<b>103</b> /min
AP	<b>80</b> (38)(52) mmHg
CVP	<b>10</b> mmHg
PPV	<b>7</b> %

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What do you use for initial **hemodynamic assessment**?

- 1. Echocardiography
- 2. Uncalibrated CO monitoring
- 3. Transpulmonary thermodilution
- 4. PAC
- 5. Nothing more than AP monitoring



HR	<b>103</b> /min
AP	<b>80/38 (52)</b> mmHg
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#### Take-home messages

- 1- During **septic** shock, **norepinephrine** should be initiated **early** when arterial **tone** is assumed to be **low**, e.g. when **DAP** is **low** (< 40 mmHg)
- 2- **Echocardiography** is the **preferred** modality to **initially evaluate** the type of **shock** as opposed to more invasive technologies

Intensive Care Med (2014) 40:1795-1815

#### CONFERENCE REPORTS AND EXPERT PANEL

Maurizio Cecconi Daniel De Backer Massimo Antonelli Richard Beale Jan Bakker Christoph Hofer Roman Jaeschke Alexandre Mebazaa Michael R. Pinsky Jean Louis Teboul Jean Louis Vincent Andrew Rhodes

Consensus on circulatory shock and hemodynamic monitoring. Task force of the European Society of Intensive Care Medicine Intensive Care Med (2016) 42:1350–1359

#### **CONFERENCE REPORTS AND EXPERT PANEL**

Less invasive hemodynamic monitoring in critically ill patients



Jean-Louis Teboul<sup>1\*</sup>, Bernd Saugel<sup>2</sup>, Maurizio Cecconi<sup>3</sup>, Daniel De Backer<sup>4</sup>, Christoph K. Hofer<sup>5</sup>, Xavier Monnet<sup>1</sup>, Azriel Perel<sup>6</sup>, Michael R. Pinsky<sup>7</sup>, Daniel A. Reuter<sup>2</sup>, Andrew Rhodes<sup>3</sup>, Pierre Squara<sup>8</sup>, Jean-Louis Vincent<sup>9</sup> and Thomas W. Scheeren<sup>10</sup>

## Transthoracic echocardiography

LV size normal

**LVEF** 70%

RV function normal

HR	<b>104</b> /min
AP	<b>91/54(66)</b> mmHg
CVP	<b>11</b> mmHg
PPV	<b>7</b> %

**4.0** mmol/L

crystalloids	<b>750</b> mL
NE	<b>0.50</b> μg/kg/min
Propofol	<b>150</b> mg/h
Ceftriaxone + Levofloxacin	

RR	<b>18</b> /min
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Pplat	<b>25</b> cmH <sub>2</sub> O
PEEP	<b>10</b> cmH <sub>2</sub> O
P/F	190

What do you think about organ perfusion pressure?



- 1. Sufficient
- 2. Insufficient
- 3. I don't know

HR	<b>104</b> /min
AP	<b>91/54(66)</b> mmHg
CVP	<b>11</b> mmHg
PPV	<b>7</b> %

**4.0** mmol/L

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P/F	190

#### Why insufficient?

- 1. History of chronic hypertension
- 2. High CVP
- 3. Both
- 4. I don't know

 $Q_5$ 

HR	<b>104</b> /min
AP	<b>91/54(66)</b> mmHg
CVP	<b>11</b> mmHg
PPV	<b>7</b> %

**4.0** mmol/L

crystalloids	<b>750</b> mL
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P/F	190

#### Why insufficient?

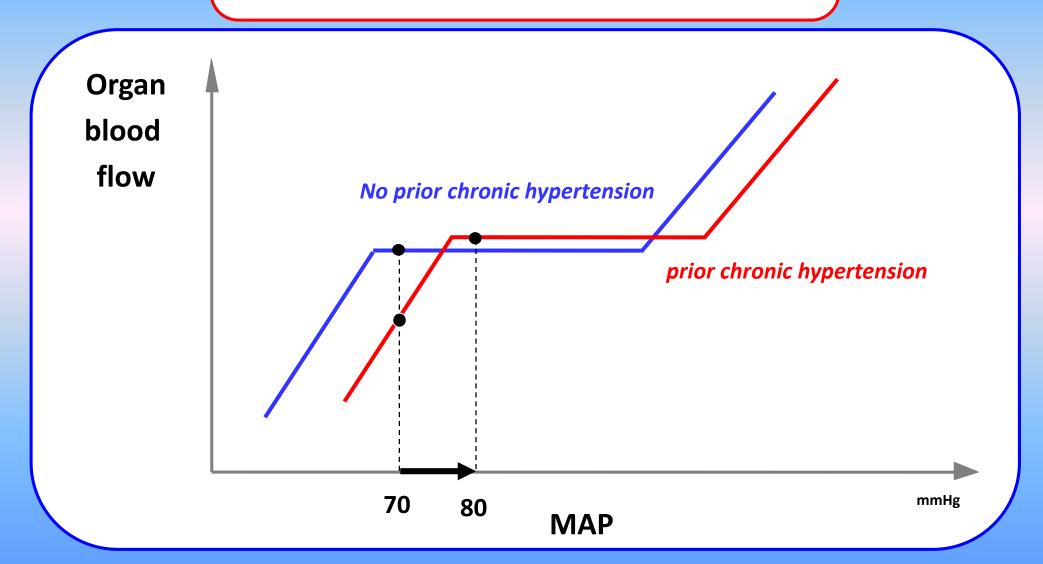
- 1. History of chronic hyp
- 2. High CVP
- 3. Both
- 4. I don't know

 $Q_5$ 

#### **Autoregulation of Brain Circulation in Severe Arterial Hypertension**

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

British Medical Journal, 1973, 1, 507-510



# The NEW ENGLAND JOURNAL of MEDICINE

80-85 mmHg

**ESTABLISHED IN 1812** 

APRIL 24, 2014

VOL. 370 NO. 17

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.,

Benefits in terms of kidney function with a high MAP target in patients with chronic hypertension

Alain Mercat, M.D., Ph.D., Jean-Louis Teboul, M.D., Ph.D., and Peter Radermacher, M.D., Ph.D., for the SEPSISPAM Investigators\*

**388** pts

**388** pts

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

Marlies Ostermann<sup>1\*</sup>, Anna Hall<sup>2</sup> and Siobhan Crichton<sup>3</sup>

BMC Nephrology (2017) 18:151

**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

#### Take-home messages

- 1- During **septic** shock, **norepinephrine** should be initiated **early** when arterial **tone** is assumed to be **low**, e.g. when **DAP** is **low** (< 40 mmHg)
- 2- Echocardiography is the preferred modality to initially evaluate the type of shock as opposed to more invasive technologies

3- The **organ perfusion pressure** should be estimated from the upstream organ pressure (MAP) and the downstream organ pressure (most often CVP) and **not from MAP alone** 

HR	<b>104</b> /min
AP	<b>91/54 (66)</b> mmHg
CVP	<b>11</b> mmHg
PPV	<b>7</b> %

crystalloids	<b>750</b> mL
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Pplat	<b>25</b> cmH <sub>2</sub> O
PEEP	<b>10</b> cmH <sub>2</sub> O
P/F	190
ScvO <sub>2</sub>	53%
Hb	<b>11</b> g/dL

Lactate	<b>4.0</b> mmol/L
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What do you do in terms of **treatment**?

- 1. Infuse fluids, first
- 2. Increase NE dose, first
- 3. Dobutamine
- 4. Nothing else
- 5. Need more information

 $Q_6$ 

HR	<b>104</b> /min
AP	<b>91/54 (66)</b> mmHg
CVP	<b>11</b> mmHg
PPV	<b>7</b> %

Lactate <b>4.0</b> mmol/L
---------------------------

crystalloids	<b>750</b> mL
NE	<b>0.50</b> μg/kg/min
Propofol	<b>150</b> mg/h
Ceftriaxone + Levofloxacin	

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P/F	190
ScvO <sub>2</sub>	53%
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What do you do in terms of **treatment**?

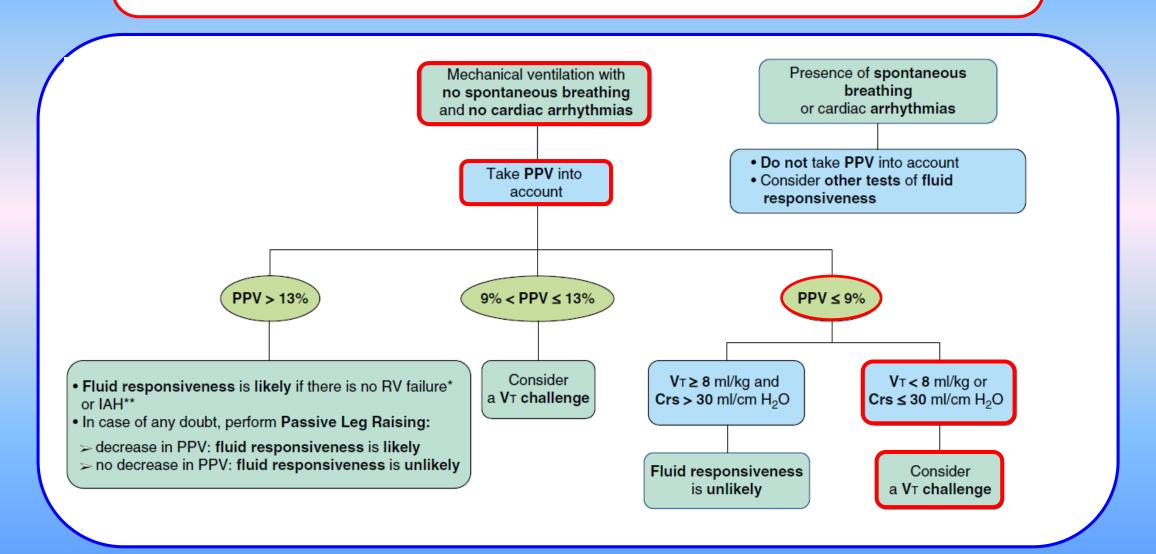
- Infuse fluids, first
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- 5. Need more information



#### **Arterial Pulse Pressure Variation with Mechanical Ventilation**

Jean-Louis Teboul<sup>1</sup>, Xavier Monnet<sup>1</sup>, Denis Chemla<sup>2</sup>, and Frédéric Michard<sup>3</sup>

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



The Changes in Pulse Pressure Variation or Stroke Volume Variation After a "Tidal Volume Challenge" Reliably Predict Fluid Responsiveness During Low Tidal Volume Ventilation\*

Sheila Nainan Myatra, MD, FCCM¹; Natesh R Prabu, MD, DM¹; Jigeeshu Vasishtha Divatia, MD, FCCM¹; Xavier Monnet, MD, PhD²; Atul Prabhakar Kulkarni, MD, FICCM¹; Jean-Louis Teboul, MD, PhD²

Crit Care Med 2017; 45:415-421

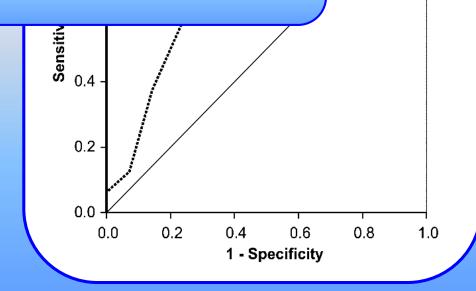
**Tidal vol** 

Very **helpful** in the absence of cardiac output monitoring

**Transient** 

in **tidal volume** 

from 6 to 8 mL/kg



HR	<b>104</b> /min
AP	<b>91/54 (66)</b> mmHg
CVP	<b>11</b> mmHg
PPV	<b>7</b> %

crystalloids	<b>750</b> mL
NE	<b>0.50</b> μg/kg/min
Propofol	<b>150</b> mg/h
Ceftriaxone + Levofloxacin	

RR	<b>18</b> /min
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PEEP	<b>10</b> cmH <sub>2</sub> O
P/F	190
ScvO <sub>2</sub>	53%
Hb	<b>11</b> g/dL

Lactate	<b>4.0</b> mmol/L
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What do you do in terms of **treatment**?

- Infuse fluids, first
- 2. Increase NE dose, first
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- 4. Nothing else
- 5. Need more information

 $Q_6$ 

TV challenge △PPV: 5 %

HR	<b>104</b> /min
AP	<b>90/52 (63)</b> mmHg
CVP	<b>10</b> mmHg
PPV	<b>7</b> %

Lactate	<b>4.0</b> mmol/L
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ScvO <sub>2</sub>	53%
Hb	<b>11</b> g/dL

crystalloids	<b>1500</b> mL
NE	<b>0.50</b> μg/kg/min
Propofol	<b>150</b> mg/h
Ceftriaxone + Levofloxacin	

HR	<b>102</b> /min
AP	110/55 (74)
CVP	<b>12</b> mmHg
PPV	5%
Lactate	<b>3.4</b> mmol/L
ScvO <sub>2</sub>	69%
Hb	<b>10.5</b> g/dL

#### Take-home messages

- 1- During **septic** shock, **norepinephrine** should be initiated **early** when arterial **tone** is assumed to be **low**, e.g. when **DAP** is **low** (< 40 mmHg)
- 2- Echocardiography is the preferred modality to initially evaluate the type of shock as opposed to more invasive technologies

3- The **organ perfusion pressure** should be estimated from the upstream organ pressure (MAP) and the downstream organ pressure (most often CVP) and **not from MAP alone** 

4- In cases of low  $V_T$ , changes in PPV during a  $V_T$  challenge can predict fluid responsiveness