

# Cardiovascular Management of Septic Shock

**R. Phillip Dellinger, MD**

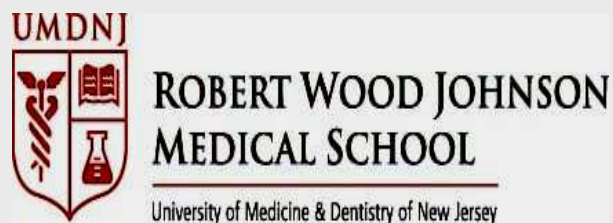
**Professor of Medicine**

**Robert Wood Johnson Medical School/UMDNJ**

**Director, Critical Care Medicine and Med/Surg ICU**

**Cooper University Hospital**

**Camden, New Jersey**



# Septic Shock — A Melting Pot of Shock Etiologies

- Hypovolemic (loss of cardiac filling)
  - Capillary leak (absolute hypovolemia)
  - Venodilatation (relative hypovolemia)
- Cardiogenic
  - Decrease in contractility
- Obstructive
  - Rise in pulmonary vascular resistance
- Distributive (hypoperfusion despite normal/increased cardiac output)

# MYOCARDIAL DYSFUNCTION IN SEPSIS

Left ventricular dysfunction

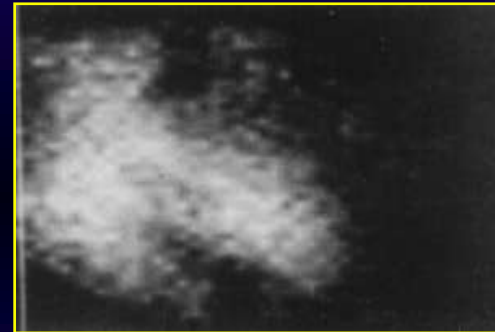
Right ventricular dysfunction

## During Septic Shock

Diastole



Systole

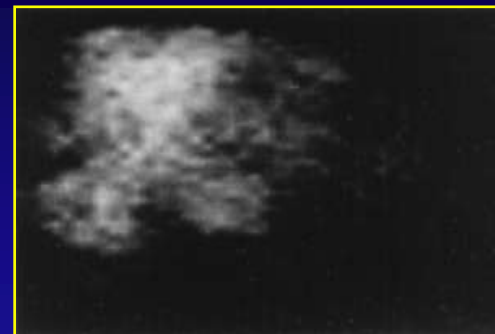


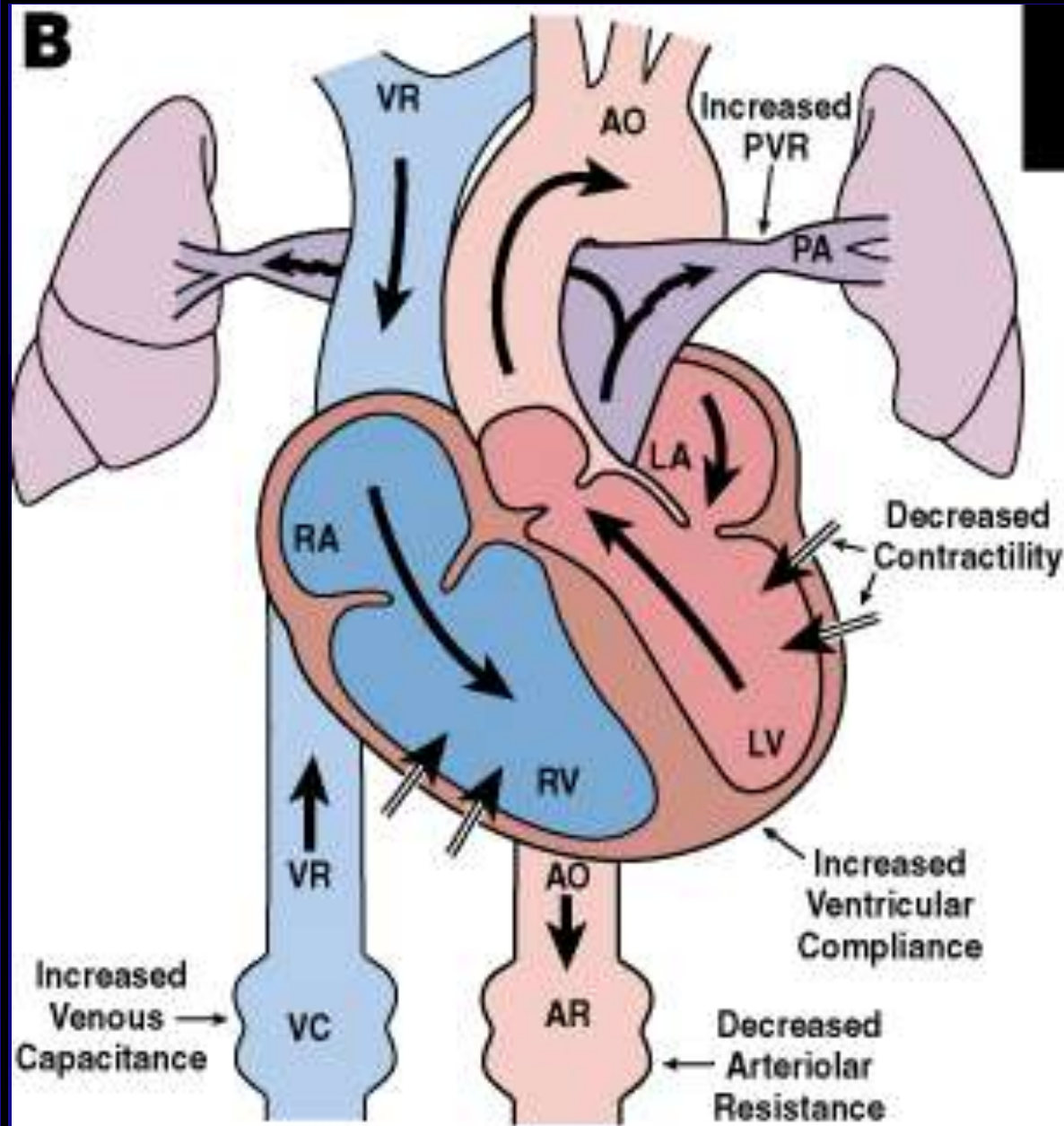
## 10 Days Post Shock

Diastole

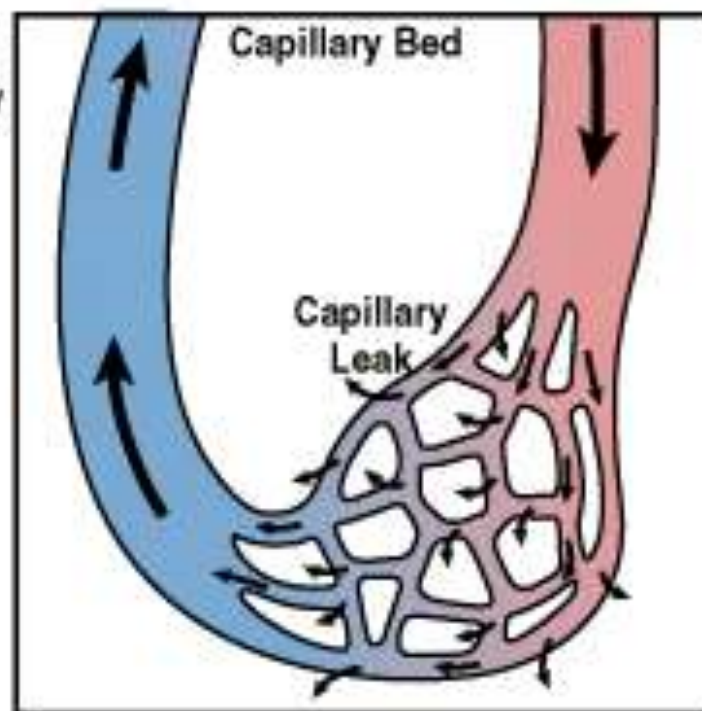
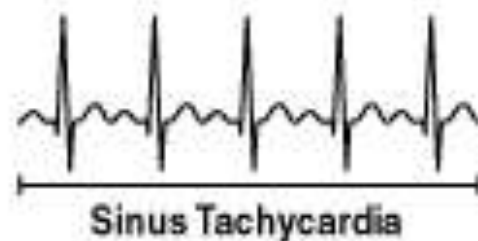


Systole



**B**

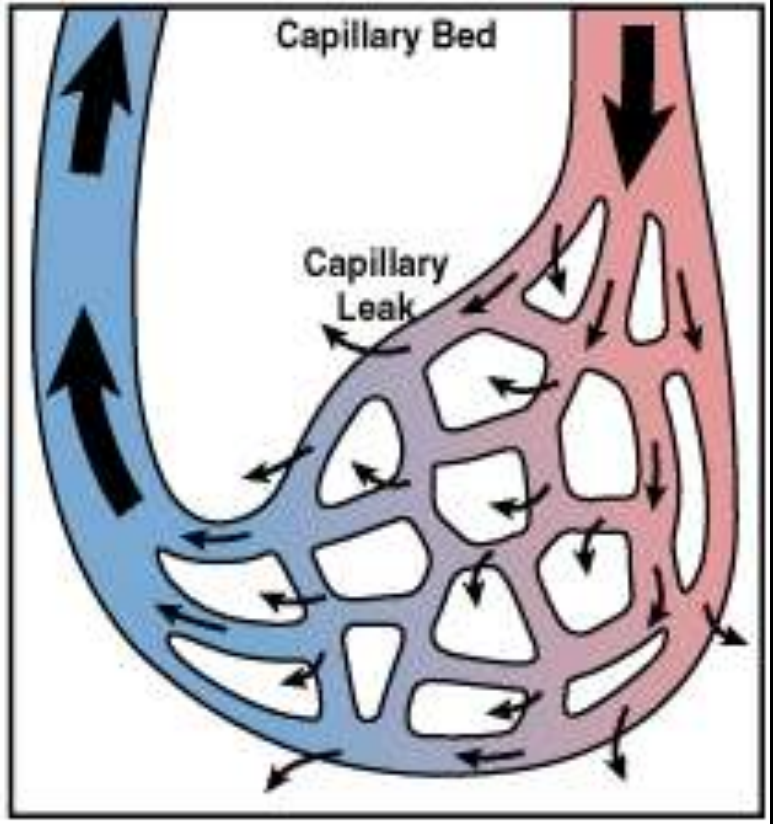
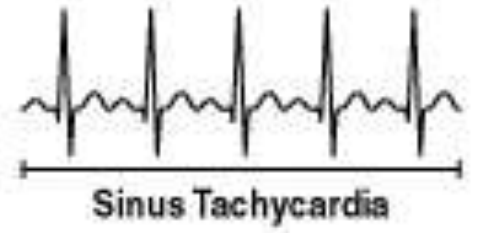
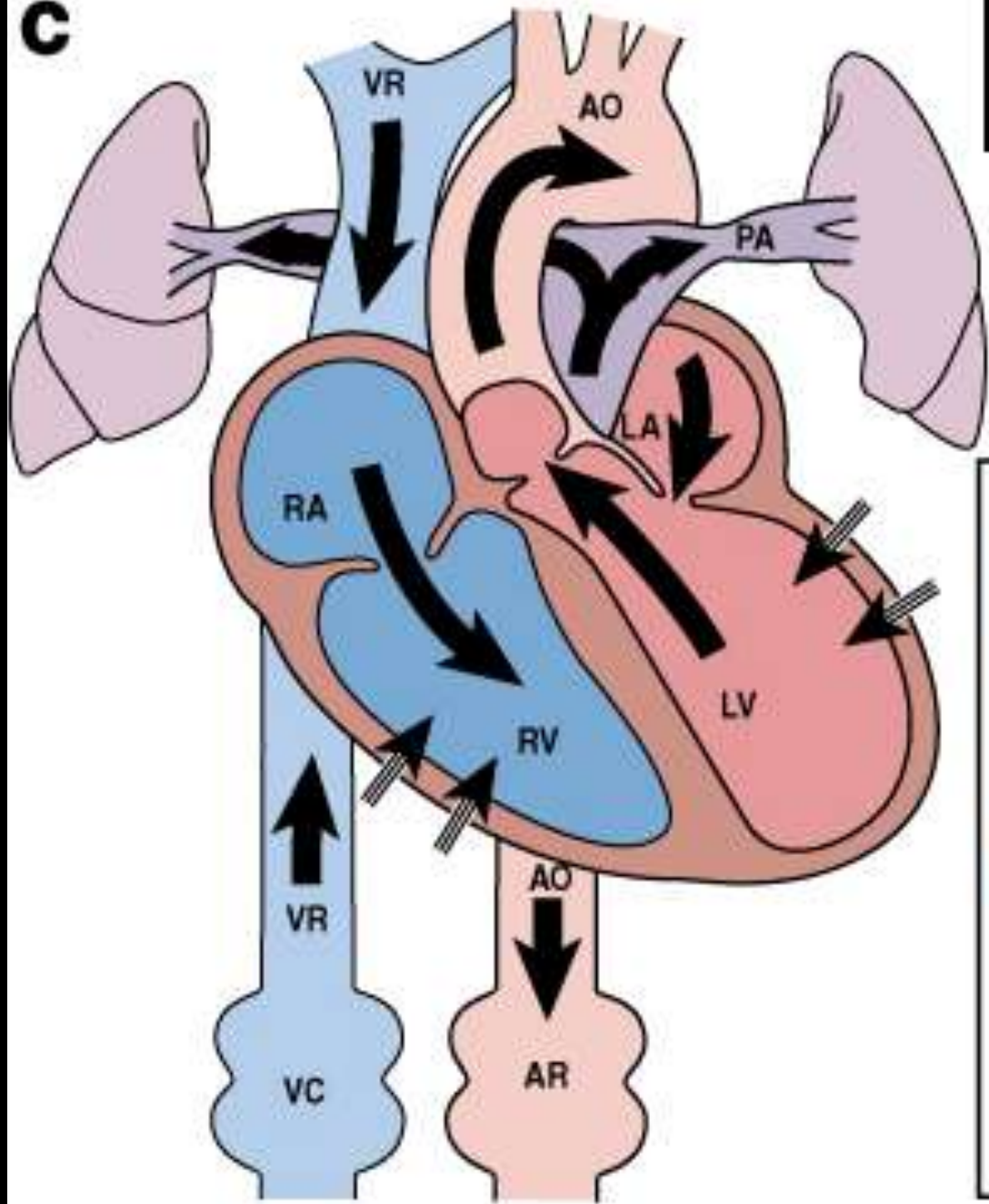
## Septic Shock Pre-Fluid Resuscitation





**C**

# Septic Shock Post-Fluid Resuscitation



## **Question:**

**If ejection fraction (contractility) is decreased in sepsis, why is cardiac output usually increased?**

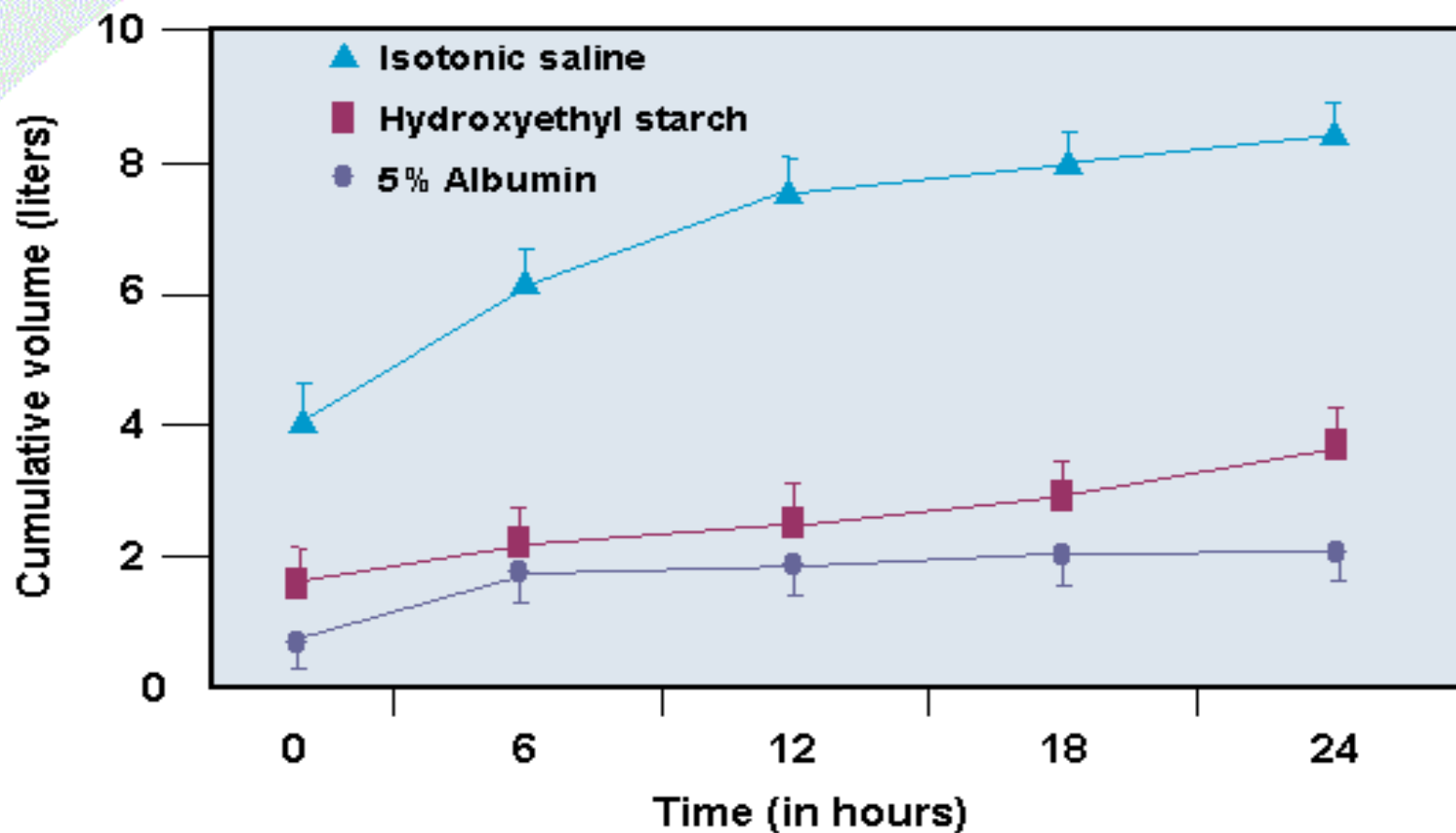
## **Reasons:**

- (1) Tachycardia**
- (2) Decreased LV afterload**
- (3) Increase in LV compliance**

# Hemodynamic Profile in Severe Sepsis and Septic Shock

- **Following volume resuscitation typically increased cardiac index with decreased systemic vascular resistance**

# Fluid Requirements in Sepsis



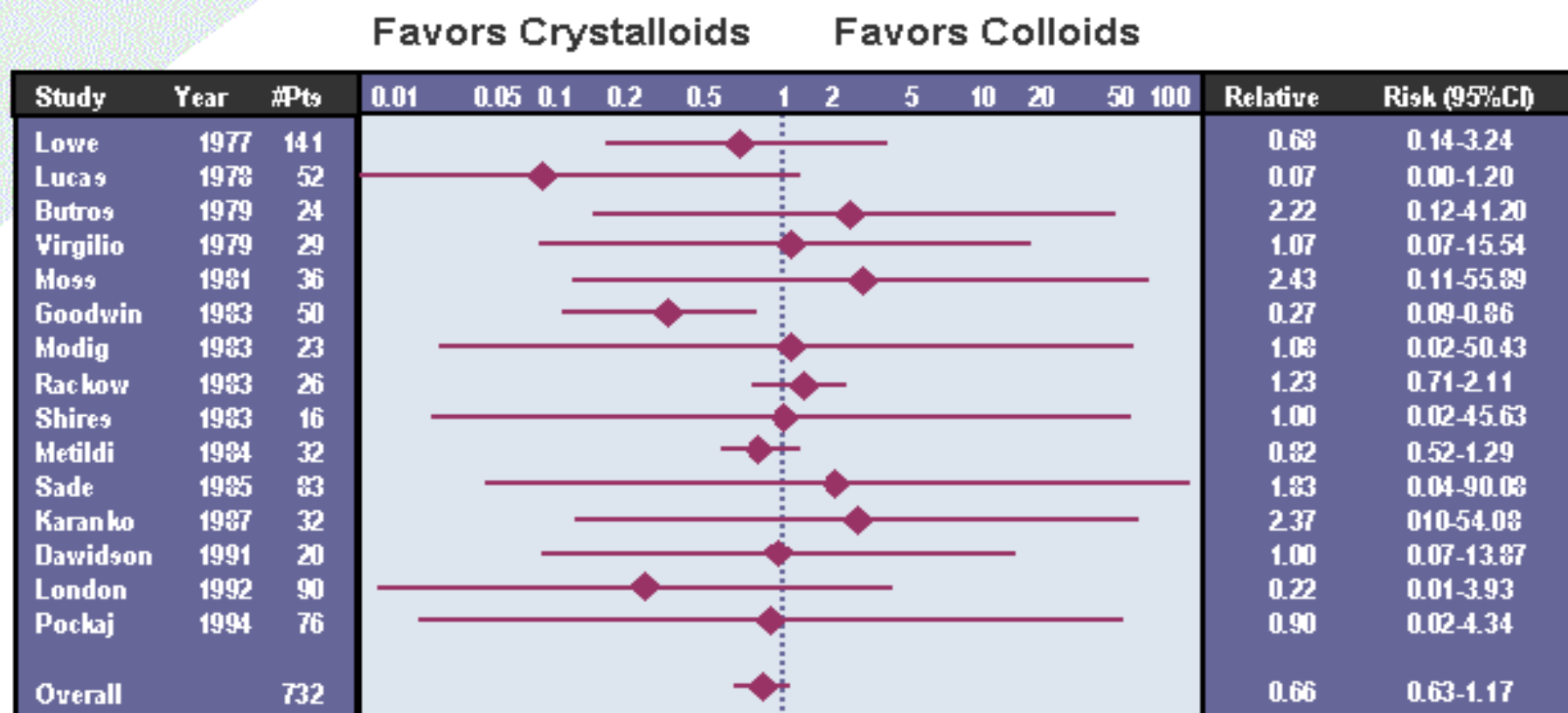
Rackow EC, et al. *Crit Care Med* 1983;11:839-50.

NATIONAL INITIATIVE IN SEPSIS EDUCATION





# What is the "Best" Fluid: Crystalloids vs Colloids?



Choi PTL, et al. *Crit Care Med* 1999;27:200-10; Cook D, et al. *Ann Intern Med* 2001;135:205-8;  
Schierhout G, et al. *BMJ* 1998 28;316:961-4; Wilkes MM, et al. *Ann Intern Med* 2001;135:149-64.



# Monitoring for Excessive Increase in Pulmonary Capillary Pressure

- **Physical exam**
- **Central venous pressure**
- **Pulmonary artery occlusion pressure**

# Hypotension Persists Despite Adequate Left Ventricular Preload

## No PA Cath

- Combined inotrope/vasopressor

## PA Cath

- Confirm adequate preload
- Inotrope targeted to maintain cardiac index 3.0
- Vasopressor targeted to maintain MAP 65 mm Hg

## CVP catheter?

**LeDoux D, Astiz ME, Carpati CM, Rackow EC**

**Effects of perfusion pressure on  
tissue perfusion in septic shock**

*Crit Care Med 2000; 28:2729-2732*

# MAP

	65 mm Hg	75 mm Hg	85 mm Hg	F/LT
<b>HR (beats/min)</b>	97 ± 4	101 ± 4	105 ± 5	.02/.02
<b>MAP (mm Hg)</b>	65 ± 0.5	75 ± 0.4	86 ± 0.4	.0001/.0001
<b>CI (L/min/m<sup>2</sup>)</b>	4.7 ± 0.5	5.3 ± 0.6	5.5 ± 0.6	.07/.03
<b>PAOP (mm Hg)</b>	14 ± 1	15 ± 1	16 ± 1	.18/.16
<b>LVSWI (g.m/m<sup>2</sup>)</b>	45 ± 3	52 ± 5.5	63 ± 7	.01/.01
<b>SVRI (dyne.sec/m<sup>2</sup>.cm<sup>5</sup>)</b>	998 ± 94	1065 ± 101	1216 ± 159	.09/.046
<b>Norepinephrine (µg/min)</b>	23 ± 22	31 ± 25	47 ± 39	.02/.016



# MAP

---

65 mm Hg

75 mm Hg

85 mm Hg

F/LT

---

**Urinary  
output (mL)**

49  $\pm$  18

56  $\pm$  21

43  $\pm$  13

.60/.71

**Capillary blood flow  
(mL/min/100 g)**

6.0  $\pm$  1.6

5.8  $\pm$  1.1

5.3  $\pm$  0.9

.59/.55

**Red Cell  
Velocity (au)**

0.42  $\pm$  0.06

0.44  $\pm$  0.06

0.42  $\pm$  0.06

.74/.97

**Pico<sub>2</sub> (mm Hg)**

41  $\pm$  2

47  $\pm$  2

46  $\pm$  2

.11/.12

**Pa-Pico<sub>2</sub> (mm Hg)**

13  $\pm$  3

17  $\pm$  3

16  $\pm$  3

.27/.40

---

# Traditional Vasopressor Therapy of Septic Shock

- Dopamine
- Norepinephrine
- Phenylephrine
- Epinephrine

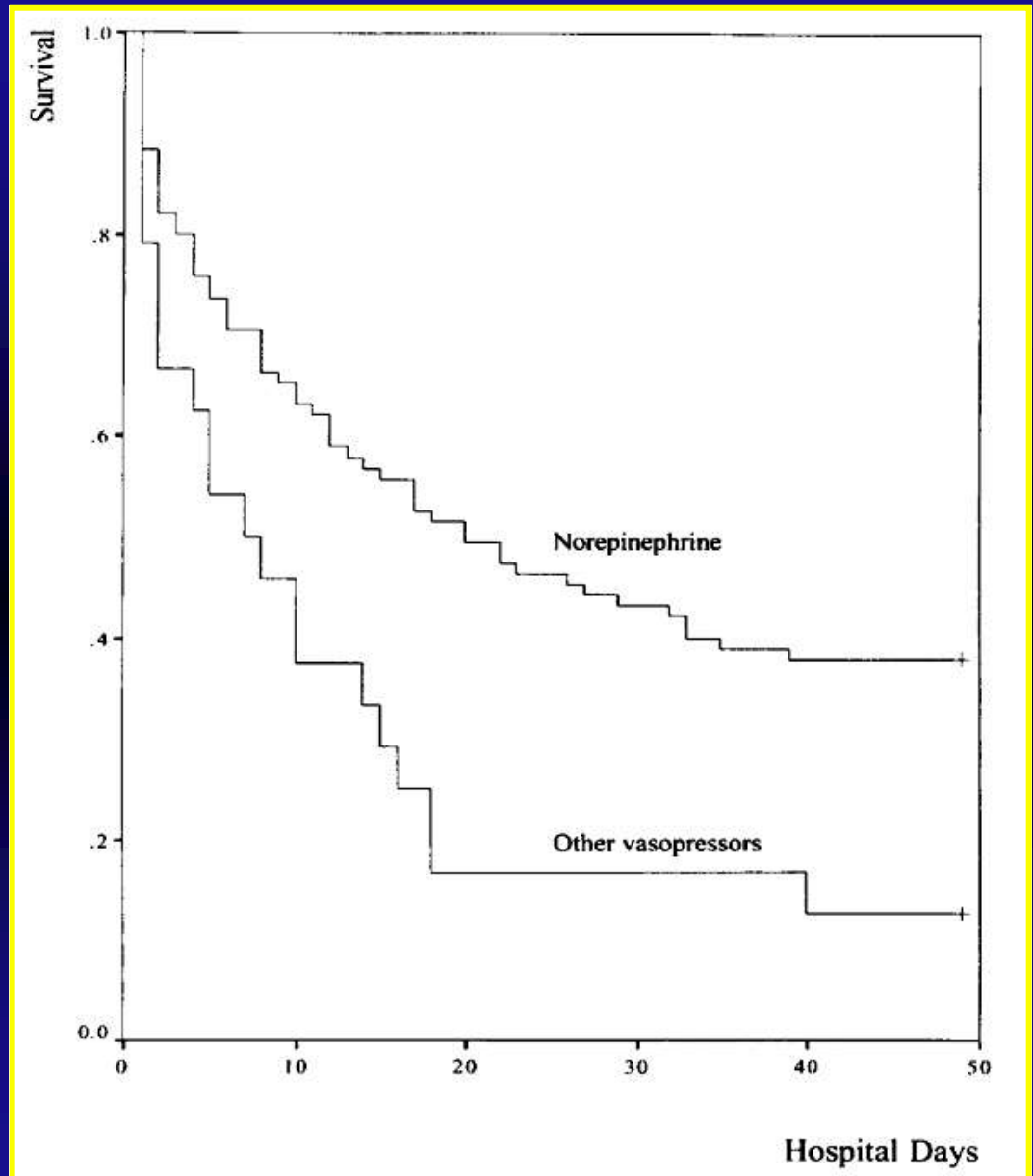
# ***Norepinephrine vs Dopamine***

## ***Norepinephrine***

- **Greater effect on efferent as opposed to afferent glomerular arteriolar resistance**
- **Better preserved splanchnic perfusion?**
- **Venous bed constriction**
  - **less ADH release**
  - **less tachycardia**
- **No effect on hypothalamic – pituitary axis or intracranial pressure**
- **More suppression of tumor necrosis factor**

**Martin C, Viviani X,  
Leone M, Thirion X.**  
Effect of norepinephrine  
on the outcome of septic  
shock. *Crit Care Med*  
2000; 28:2758-2765

**NE had significantly  
lower hospital  
mortality (62% vs  
82%)  $p < .001$**



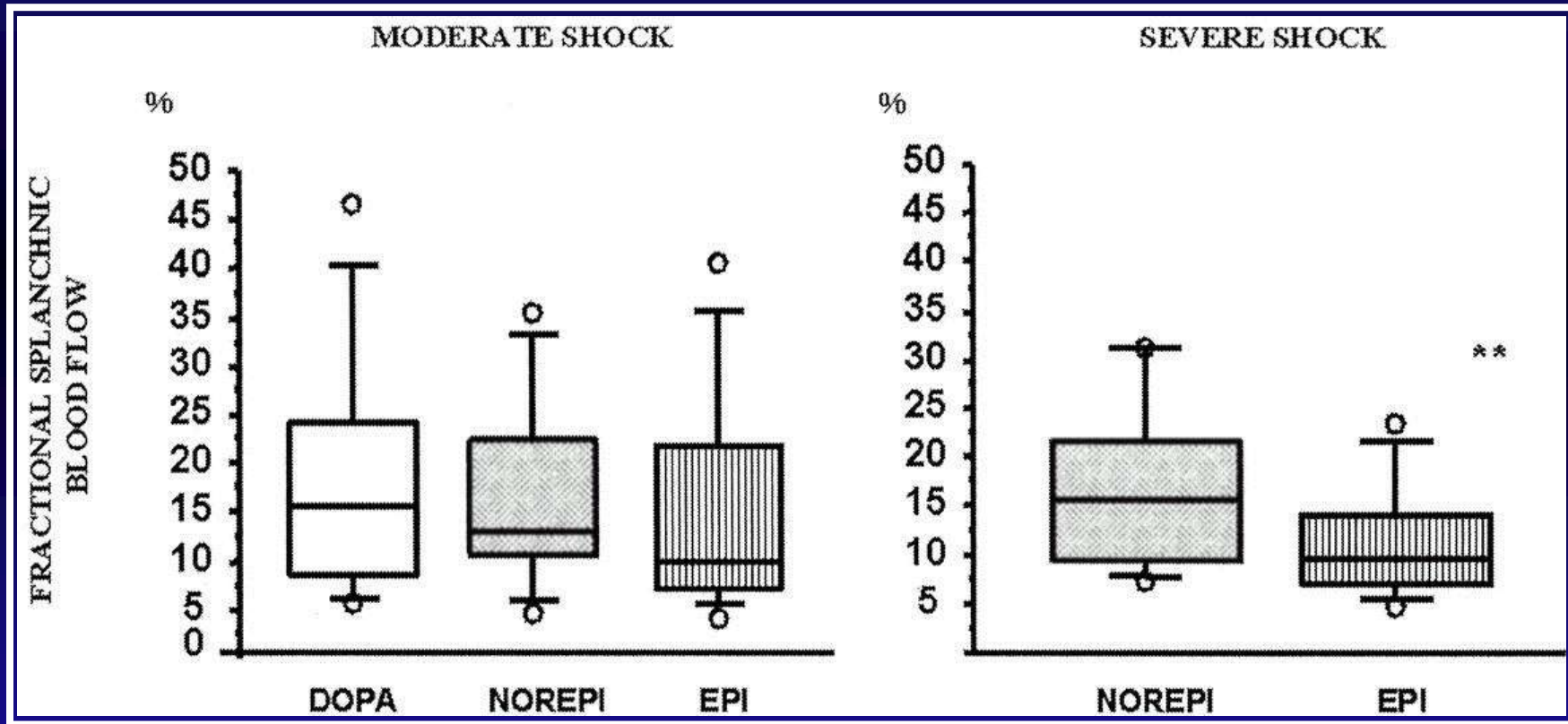
# Utility of Vasopressin in Septic Shock

## Vasopressin and Shock

- **Animal models of septic shock**
- **Septic versus cardiogenic shock**

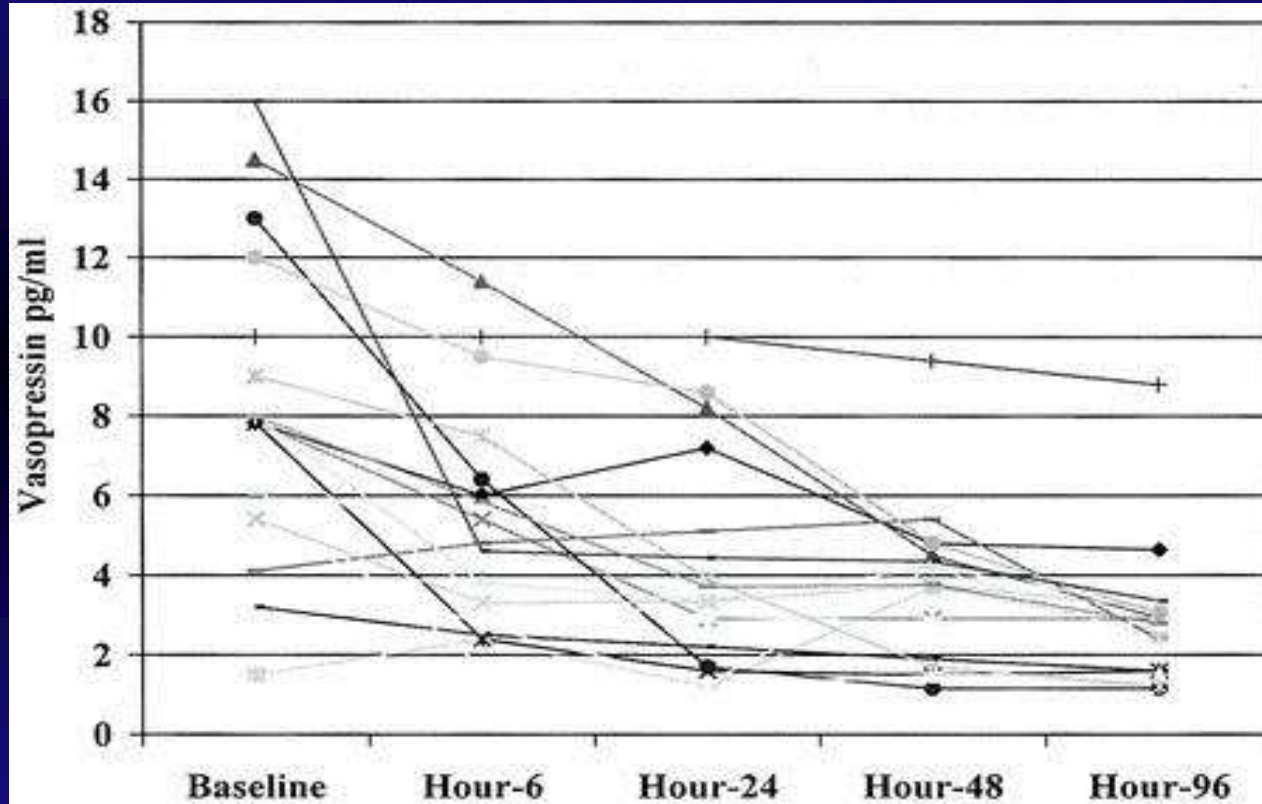


# Effects of Dopamine, Norepinephrine, and Epinephrine on the Splanchnic Circulation in Septic Shock: Which is Best?



De Backer D, et al. *Crit Care Med* 2003; 31:1659-1667

# Circulating Vasopressin Levels in Septic Shock



Sharshar T, et al. *Crit Care Med* 2003; 31:1752-1758

# Vasopressin and Septic Shock

- Human septic shock studies with low rates of infusion (.01-.04 units minute) decrease traditional vasopressor requirements.

## So where do we stand with vasopressin therapy?

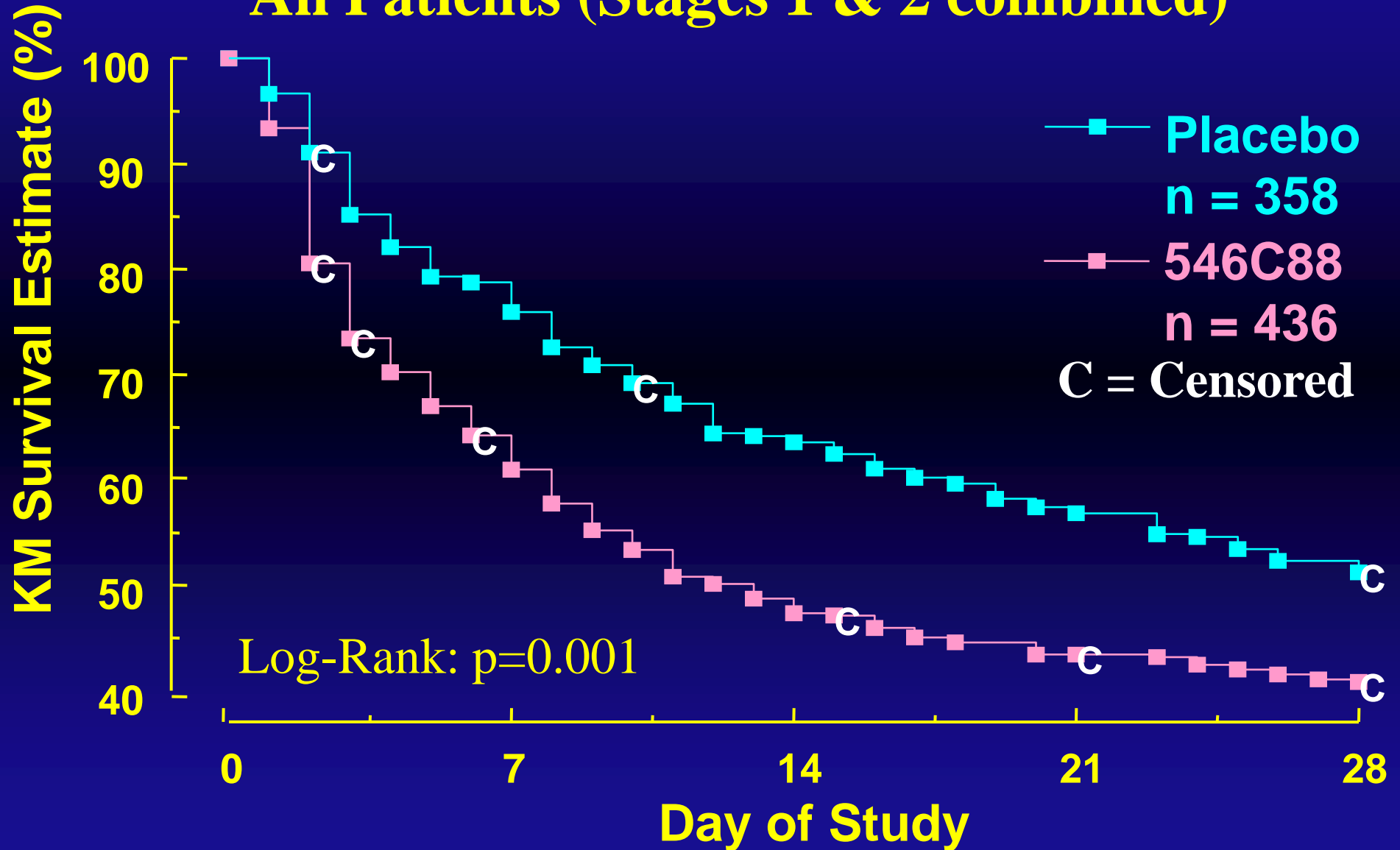
- Low dose vasopressin is very effective in decreasing or eliminating requirements of other vasopressors in septic shock

## Concerns with routine use of vasopressin

- Effect on splanchnic circulation
- Effect on stroke volume and cardiac output

# LMAB3001 - Kaplan-Meier Survival Curves

## All Patients (Stages 1 & 2 combined)



# Discussed in SSC Guidelines Presentation

- **Early goal directed resuscitation**
- **Steroid therapy**
- **Recombinant Activated Protein C**



## Reasonable Therapeutic Goals in Severe Sepsis with Hypoperfusion

- Mean arterial pressure of 60–65 mm Hg
- Urine output of  $\geq 0.5$  ml/kg/hr
- ScvO<sub>2</sub>  $\geq 70\%$
- Reversal of lactic acidosis
- If cardiac output measured,  $\geq 3.0$