Principles and Pathophysiologic Essentials of Kidney involvement in Critical Care

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The critical ill patient may have a single or multiple disease processes. For example: in the context of DM and COPD, the patient may have peritonitis lead to septic shock and then AKI and later may need HD.

Objectives:

- Identify organ dysfunction.
- Key principles of management. *The VIP rule ?*
- Individualized therapy and management.

Sepsis, a syndrome of physiologic, pathologic, and biochemical abnormalities induced by infection.

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection. (a state of acute circulatory Failure).

Accounting for more than $20 billion (5.2%) of total US hospital costs in 2011.

SIRS (Systemic Inflammatory Response Syndrome)(reflect an appropriate host response)

2 or more of:

Temp>38° C or < 36° C

HR>90/min

RR>20/min or $Paco_2$ <32mmhg

WBC > 12000/mm^3 or <4000/mm^3 or > 10% immature bands

The 3rd International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3)
Mervyn Singer, Clifford S. Deutschman, et al
JAMA. 2016; 315(8): 801-810.

- ***Septic shock:***

  patients need vasopressor to maintain MAP ≥ 65 mm Hg, and
  serum lactate level >2 mmol/L (18mg/DL) in the absence of hypovolemia.

  Hypotension should be denoted as a MAP < 65mmHg.

  Mortality rate > 40%.

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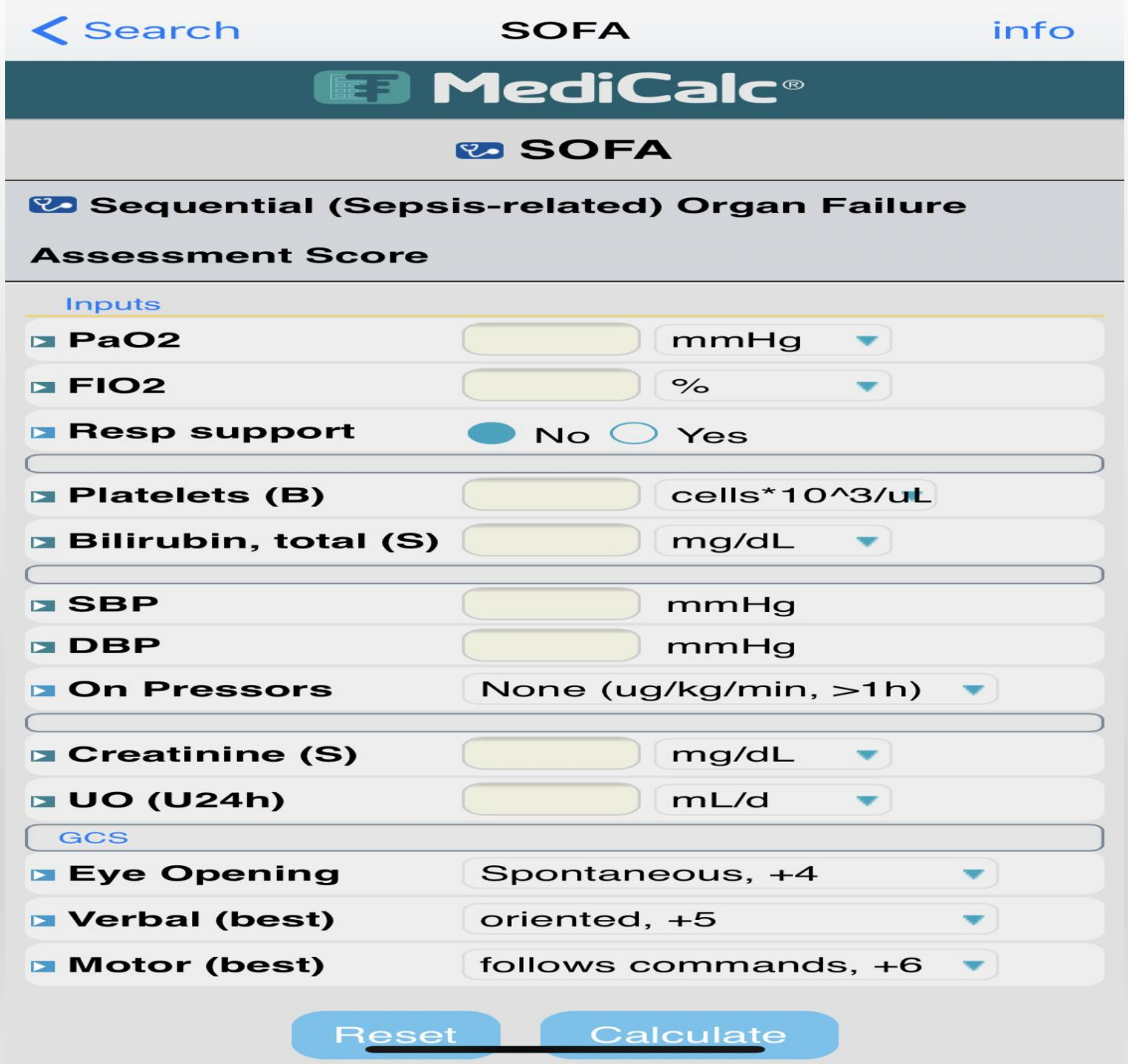
The Sequential Organ Failure Assessment (SOFA) Score[2]

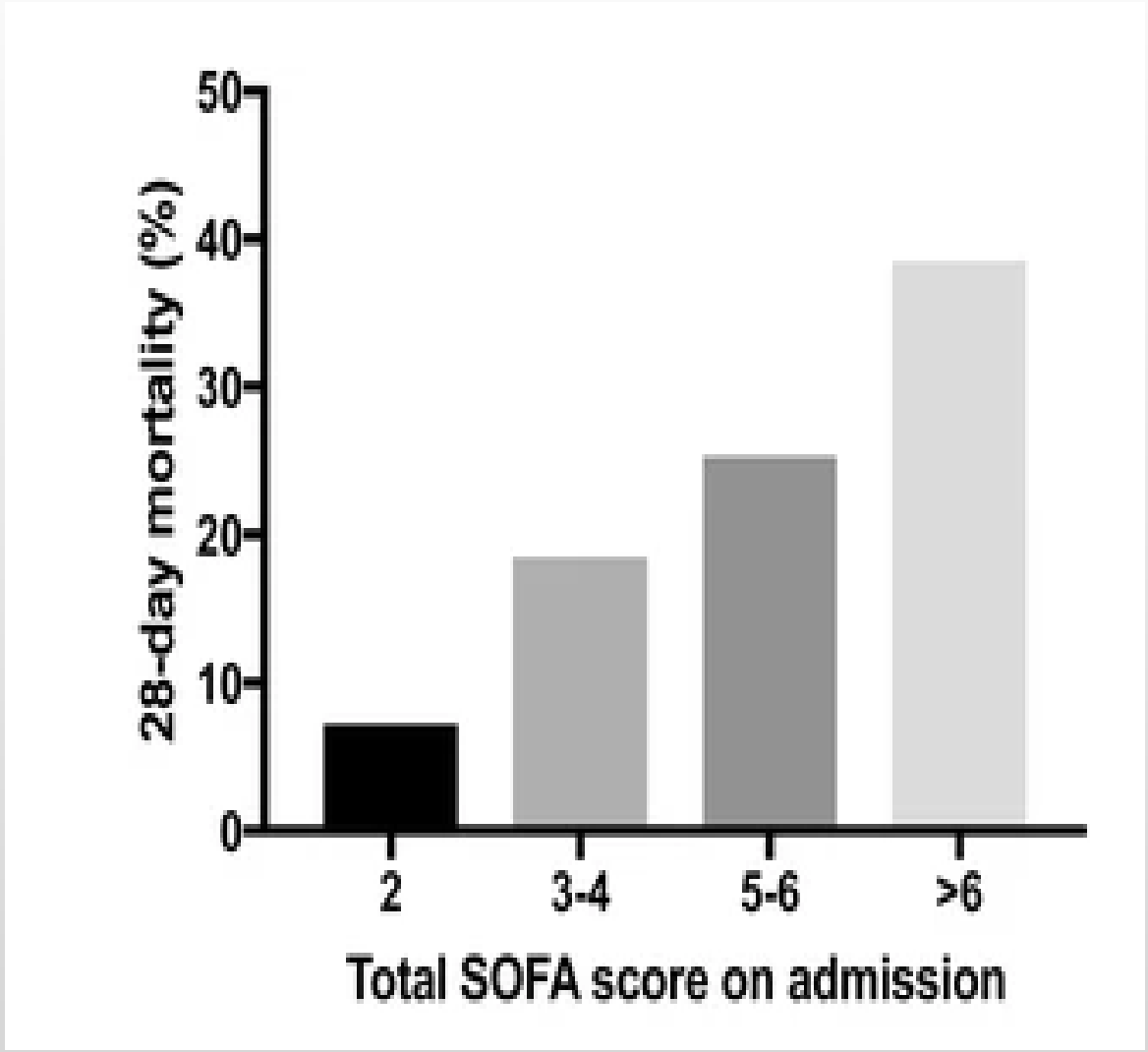
> 2 points is associated with an in-hospital mortality > 10%.

SCORE	0	1	2	3	4
Respiration					
PaO_2/FiO_2, mm Hg	>400	≤400	≤300	≤200 With respiratory support	≤100
Coagulation					
Platelets × $10^3/mm^3$	>150	≤150	≤100	≤50	≤20
Liver					
Bilirubin, mg/dL (Mmol/L)	<1.2 (<20)	1.2–1.9 (20–32)	2.0–5.9 (33–101)	6.0–11.9 (102–204)	>12.0 (>204)
Cardiovascular					
Hypotension	No hypotension	MAP < 70 mmHg	Dopamine ≤ 5 or dobutamine (any dose)*	Dopamine > 5 or epinephrine ≤ 0.1 or norepinephrine ≤ 0.1*	Dopamine > 15 or epinephrine > 0.1 or norepinephrine > 0.1*
Central Nervous System					
Glasgow coma score	15	13–14	10–12	6–9	<6
Renal					
Creatinine, mg/dL (Mmol/L)	<1.2 (<110)	1.2–1.9 (110– 170)	2.0–3.4 (171–299)	3.5–4.9 (300–440)	>5.0 (>440)
OR urine output				<500 mL/d	<200 mL/d

Quick SOFA (qSOFA) score: RR ≥ 22/min, altered mentation, SBP ≤ 100 mm Hg

*Adrenergic agents administered for at least 1 hour (doses given are in mcg/kg/min).


Search
SOFA
info
MediCalc®
SOFA
Sequential (Sepsis-related) Organ Failure Assessment Score
Inputs
PaO2
mmHg
FIO2
%
Resp support
No
Yes
Platelets (B)
cells*10^3/uL
Bilirubin, total (S)
mg/dL
SBP
mmHg
DBP
mmHg
On Pressors
None (ug/kg/min, >1h)
Creatinine (S)
mg/dL
UO (U24h)
mL/d
GCS
Eye Opening
Spontaneous, +4
Verbal (best)
oriented, +5
Motor (best)
follows commands, +6
Reset
Calculate



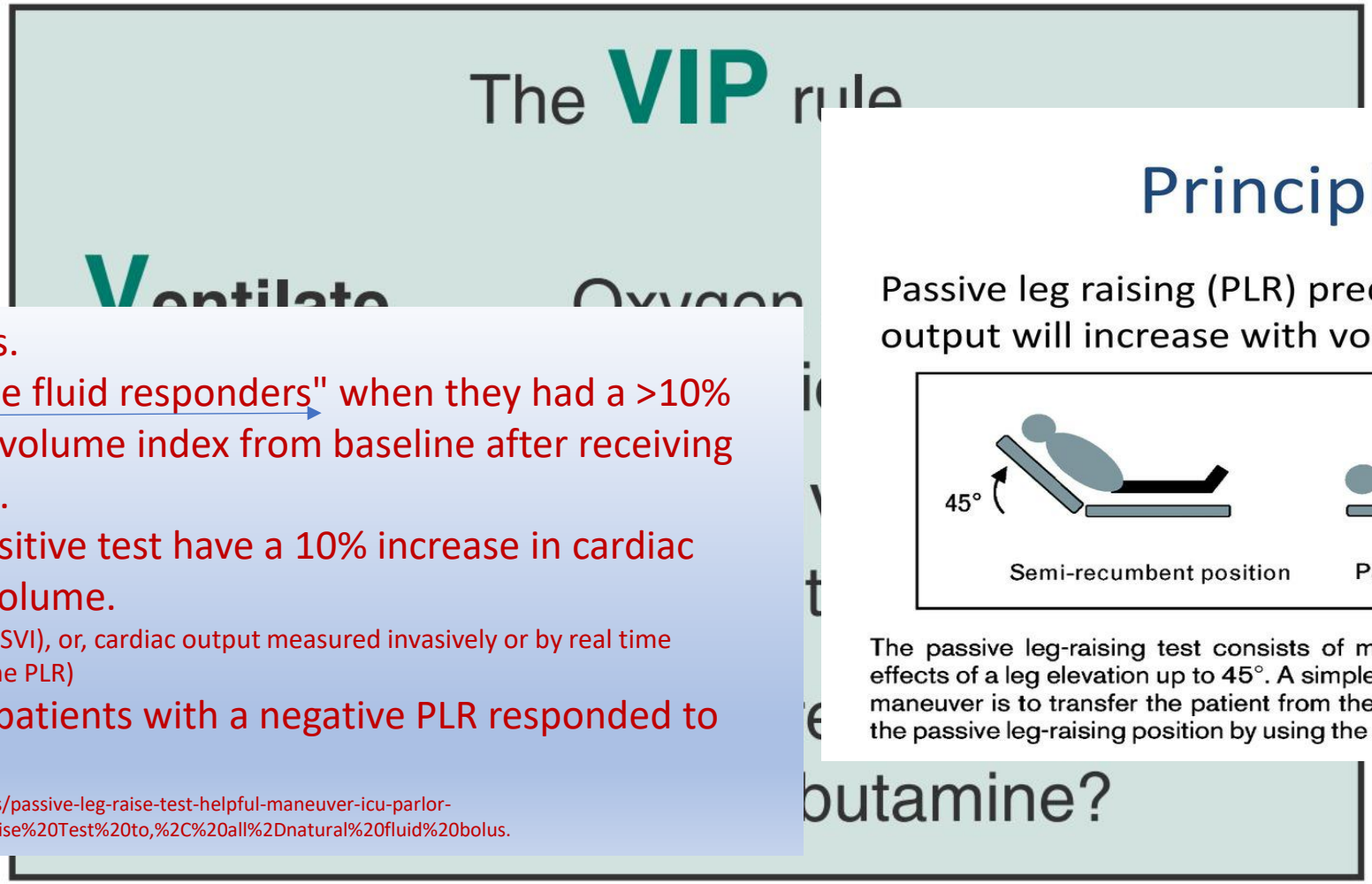
Maximum SOFA Score	Mortality
0 to 6	< 10%
7 to 9	15 - 20%
10 to 12	40 - 50%
13 to 14	50 - 60%
15	> 80%
15 to 24	> 90%

https://clincalc.com/IcuMortality/SOFA.aspx

Utility of SOFA score, management and outcomes of sepsis in Southeast Asia: a multinational multicenter prospective observational study

Journal of Intensive Care **volume 6**, Article number: 9 (2018)

Resuscitation



PLR 30-90 seconds.

Patients were "true fluid responders" when they had a >10% increase in stroke volume index from baseline after receiving 500 mL crystalloid.

Patients with a positive test have a 10% increase in cardiac output or stroke volume.

(with stroke volume index (SVI), or, cardiac output measured invasively or by real time echocardiography during the PLR)

Only about 1 in 8 patients with a negative PLR responded to fluids.

https://pulmccm.org/review-articles/passive-leg-raise-test-helpful-maneuver-icu-parlor-trick/#:~:text=Passive%20Leg%20Raise%20Test%20to,%2C%20all%2Dnatural%20fluid%20bolus.

Principle 3

Passive leg raising (PLR) predicts whether cardiac output will increase with volume expansion

The passive leg-raising test consists of measuring the hemodynamic effects of a leg elevation up to 45°. A simple way to perform the postural maneuver is to transfer the patient from the semi-recumbent posture to the passive leg-raising position by using the automatic motion of the bed.

http://crashingpatient.com/resuscitation/predicting-fluid-responsiveness.htm

FIGURE 1.2 The VIP (ventilation, infusion, pump) rule proposed by Max H. Weil and colleagues.[3] *IV,* Intravenous.

BOX 2.1

Insights Into Cardiovascular Signs of Insufficiency

- Tachycardia
  - Nonspecific stress
- Arterial hypotension
  - Loss of vital homeostasis causing loss of blood flow regulation
- No normal cardiac output
  - Cardiac output is adaptive to changing metabolic demands
- Central venous pressure rises only in disease
  - Normally it is close to zero as to not limit venous return

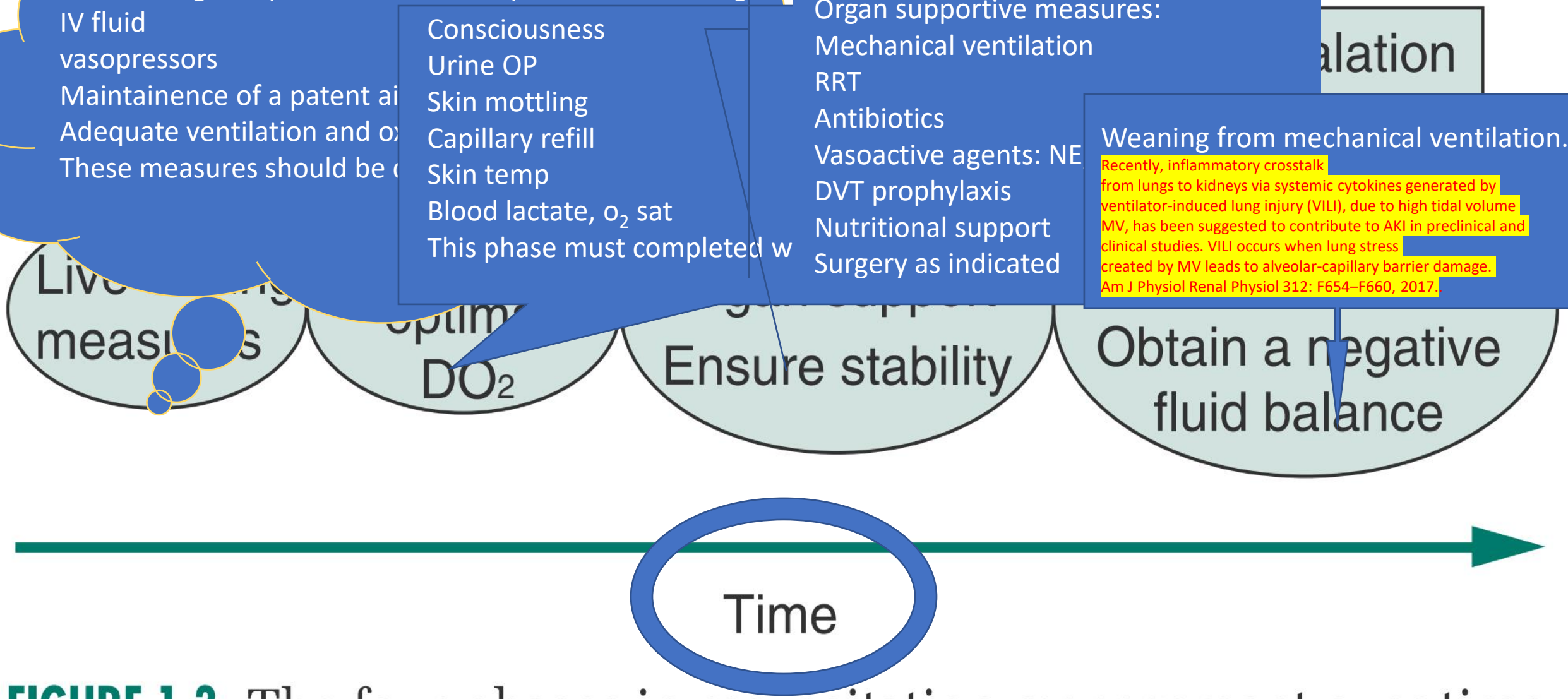
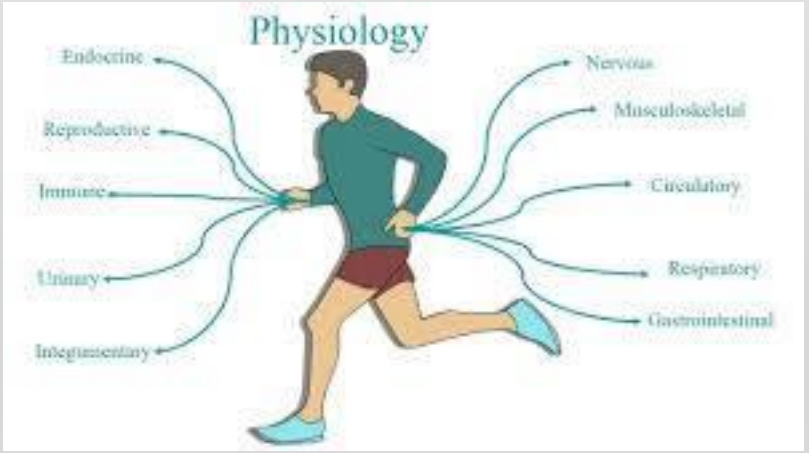
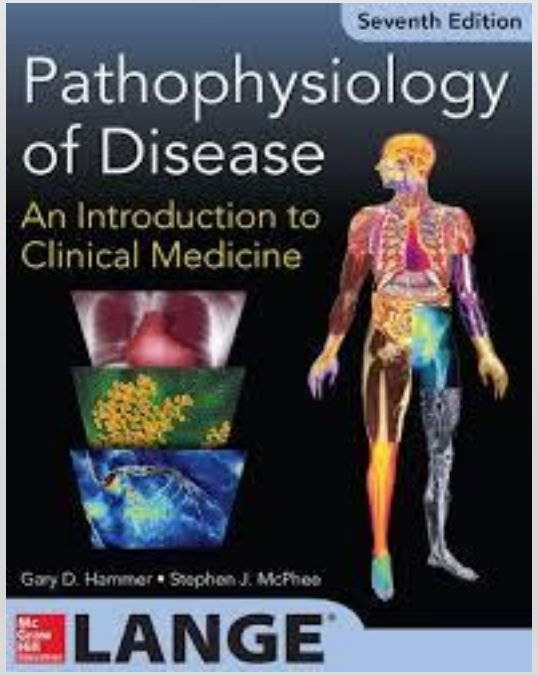


FIGURE 1.3 The four phases in resuscitation management over time. *DO_2*, Oxygen delivery. Modified from Vincent and De Backer[1].

Organ failure reflects failure of host defense homeostasis.

To manage the critically ill patient in the ICU, it is necessary to have a broad knowledge base in:





Global blood flow value targeted to organ perfusion metrics:

- Blood lactate
- Venous O_2 saturation
- Venoarterial CO_2 gradient

Ventricular filling pressures assessment:

- Right atrial pressure ⟹ CVP
- Left atrial pressure ⟹ Pulmonary artery occlusion pressure

Cryptic Shock

Global tissue Hypoxia

Elevated Lactate level

in the absence of hypotension.

Aggressive fluid management in the critically ill: Pro
Katsura Hayakawa
Hayakawa Journal of Intensive Care (2019) 7:9
https://doi.org/10.1186/s40560-019-0361-9

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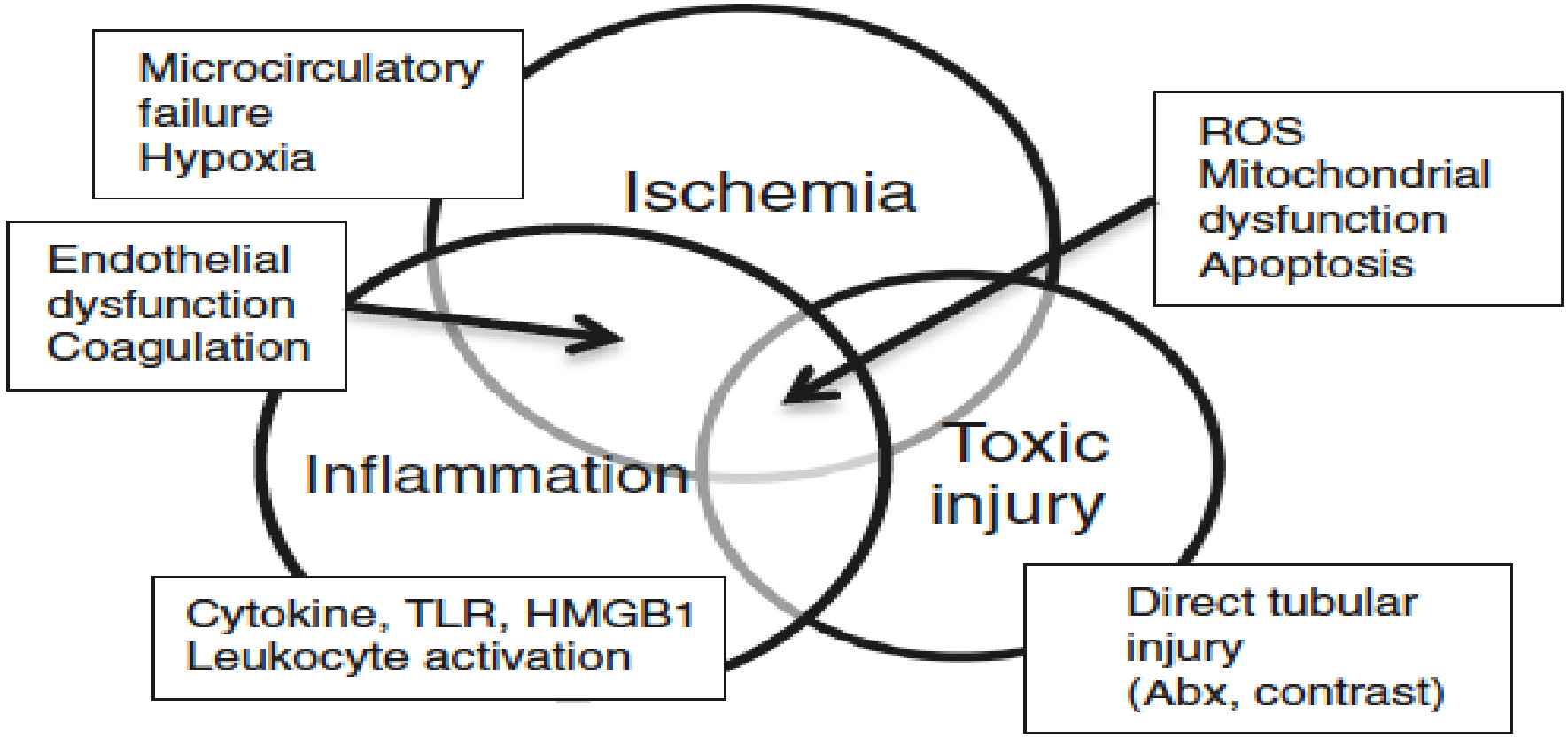

Fig. 1 Pathophysiology of AKI. Three major areas of ischemia, inflammation, and direct toxic injury to the kidney contribute to the pathogenesis of AKI with significant overlap. Each mechanistic pathway identified by basic studies will be categorized into one of these major areas; however, some will lie simultaneously in two or three areas. Details are described in other review articles [71–73]. *ROS* reactive oxygen species, *TLR* toll-like receptor, *HMGB1* high mobility group box 1, *ABx* antibiotics

Role of kidney injury in sepsis
Kent Doi
Journal of Intensive Care (2016) 4:17
DOI 10.1186/s40560-016-0146-3