Fluid therapy in critically ill patients; poison or Panacea

Dr.A.Saeed,Ped.intensivist


ANSWERS
SIMPLE BUT WRONG
COMPLEX BUT RIGHT
GOCOMICS.COM/NONSEQUITUR

ifa international fluid academy

- fluids are not innocent bags of water,

- ***Fluids are "Drugs"***

THE GOOD

THE BAD

THE UGLY

THE LIFE-SAVING

?

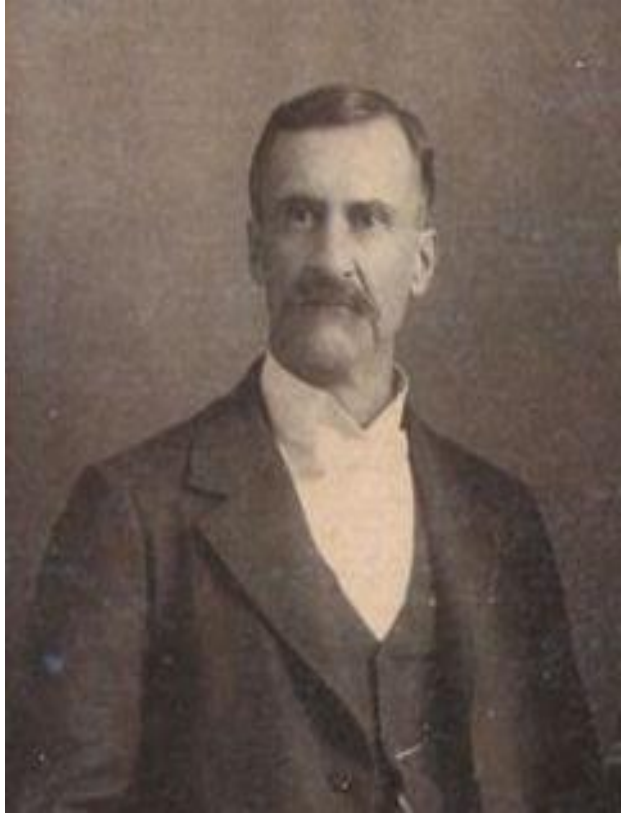
THE DEADLY

Study the past, if you would divine the future.

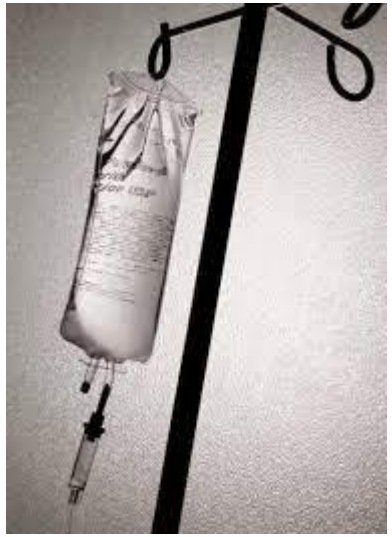
Confucius

- In 19th century;R. Hermann proposed injecting water;then *Jaehnichen* subsequently injected a cholera patient intravenously with 6 oz. of water, which resulted in a notable improvement in the
- patient's pulse; but …

The history of fluid therapy



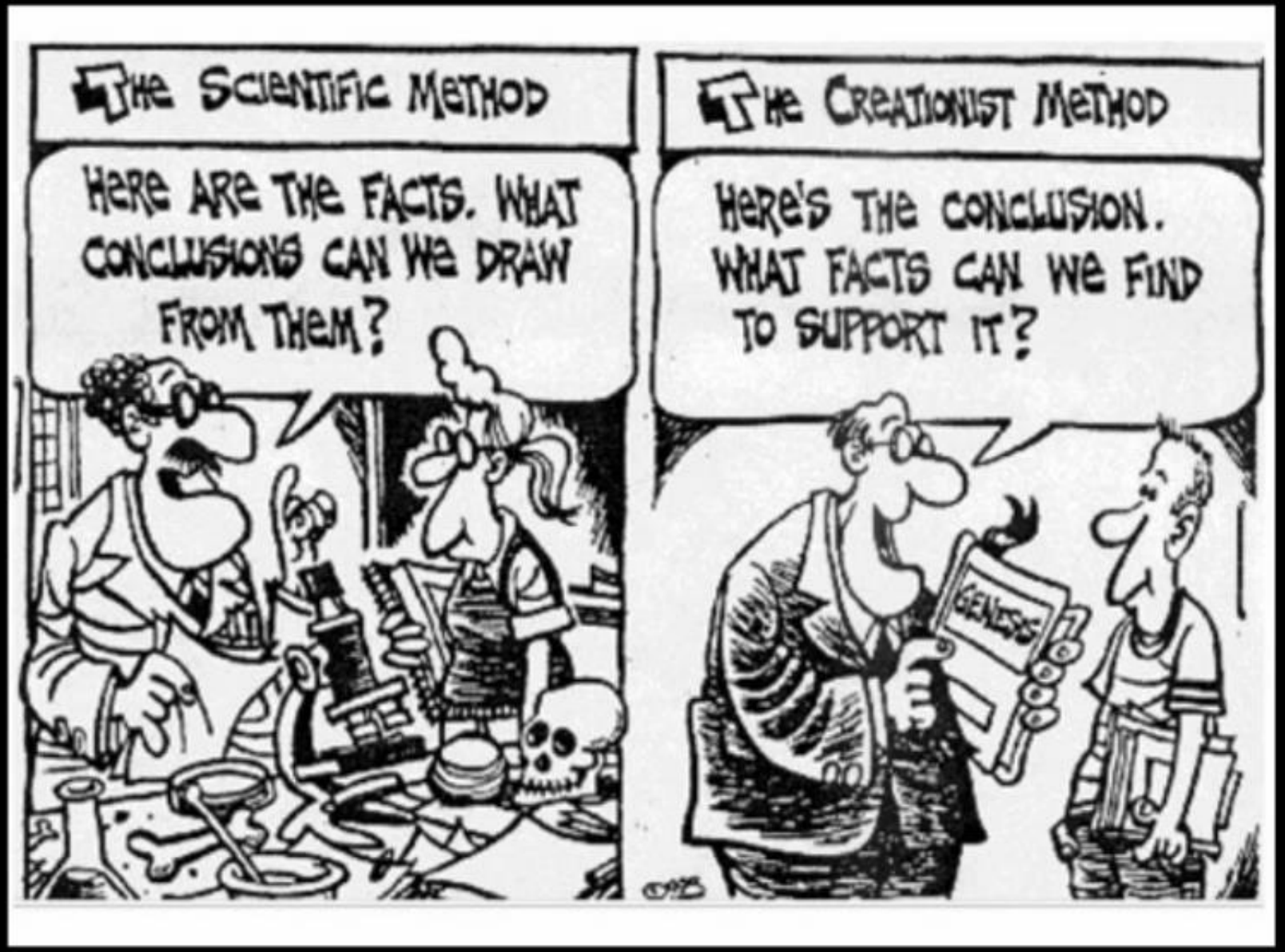
- Thomas Aitchison Latta (1796 – 19 October 1833) invented the intravenous saline infusion in 1832.



fluid. NB Latta's solution is very hypotonic compared with blood

	Latta's saline solution (1 L)	Modern day normal saline (1 L)
Sodium chloride	2.3–3.5 g	9 g
Sodium bicarbonate	0.8 g	Nil
Sodium concentration	49.6–69.8 mEq/L	154 mEq/L
Chloride concentration	40.5–60.7 mEq/L	154 mEq/L

What is knowledge?



BP = CO x SVR

↓

CO = SV x HR

↓

Stroke Volume

↙ ↓ ↘

Preload | **Afterload** | **Contractility**

↓ | ↓ | ↓

Volume of blood at end-diastole

Resistance the heart must overcome to eject blood

The heart **Strength** with pumping and its **Force**

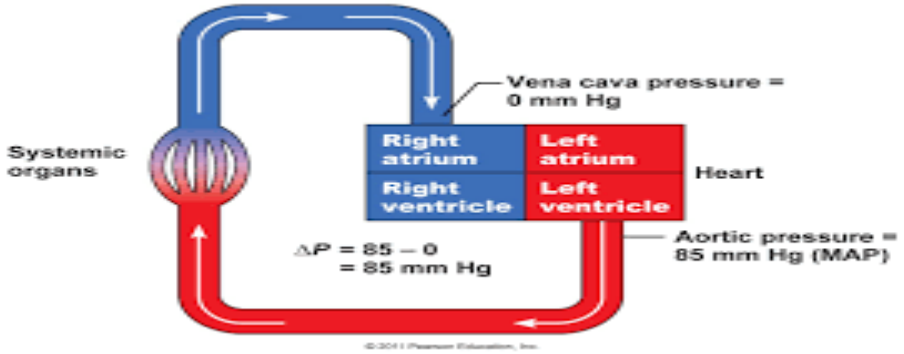
- Ernest Starling recognized at the turn of the last century that the heart can only pump out what comes back to it.
- At the end of the 19th century, Frank found that ventricular contractility was increased if the ventricle was stretched prior to contraction
- Starling and colleagues found that increasing venous return increased stroke volume.
- The Starling Principle states that fluid movements between blood and the tissue are determined by differences in hydrostatic and colloid osmotic pressures between plasma inside the microvessels and fluid outside them. (revised starling principle in 2012…)

This concept was later developed further by Arthur Guyton and Solbert Permutt;

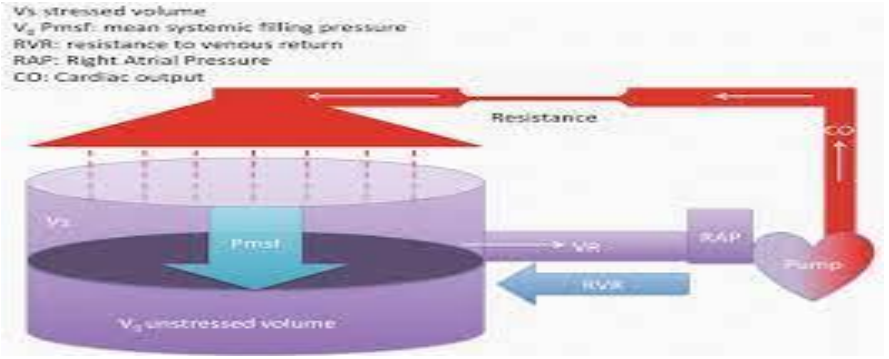
compliance, capacitance, stressed volume, and resistance to flow;

A central axiom in the circulation is that vascular volume is constant under steady state conditions. This volume stretches the elastic walls of the vasculature structure and creates an elastic recoil force that is present even when there is no flow but is also a key determinant of flow

The Guyton model suggests that the flow of blood returning to the heart is mainly driven by this mean systemic filling pressure. This is the pressure blood flows from, and CVP is the pressure it flows to, overcoming venous vascular resistance in the process. Cardiac output, in this model, plays no role in determining the pressure in the venous circulation.



- In 1955 *Dr. Arthur Guyton*; the factors that influence venous return physiology:three variables all of which independently affect venous return. These factors include: the right atrial pressure, the mean systemic pressure (Pms) and the vascular resistance.
- The Pms is the driving pressure competing against right atrial pressure to create a gradient that promotes forward flow. It is essentially the pressure measured in the vascular system if all blood flow were to cease
- The Pms is determined by the total volume of blood present in the venous system, and the intrinsic compliance of the vascular bed

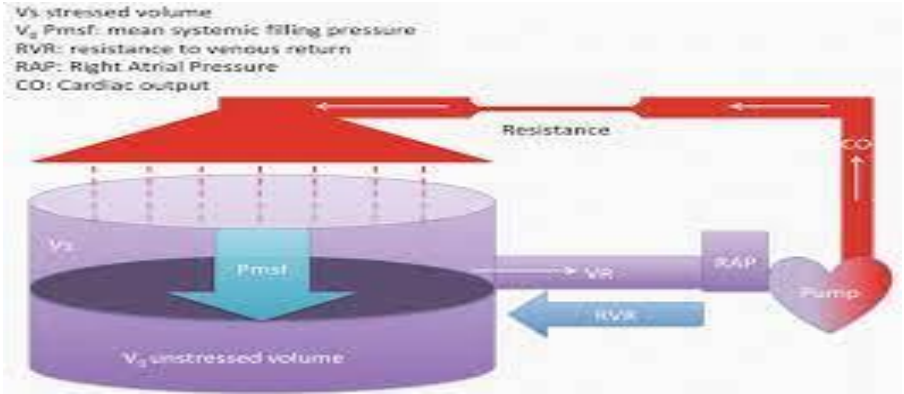


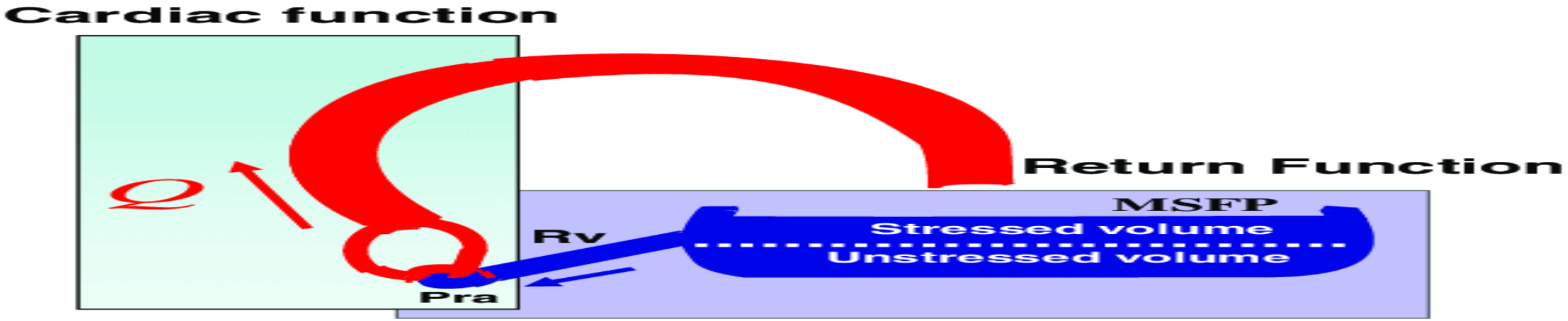
- The potential energy of this elastic recoil becomes evident when there is no flow in the circulation and large veins are opened to atmospheric pressure. Vascular volume empties from the veins even without cardiac contractions. The heart adds a pulsatile component to this static potential energy which redistributes the volume according to the compliances and resistances entering and draining each elastic compartment of the circulation.
- As already discussed, when the vasculature is filled with a normal blood volume but there is no flow, the vasculature still has a pressure and this pressure is the same in all compartments of the circulation. It is called mean circulatory filling pressure (MCFP) and is determined by the total stressed volume in the circulation and the sum of the compliances of all regions, including the pulmonary and cardiac compartments

- Mean circulatory filling pressure (MCFP) is the pressure that would be measured at all points in the entire circulatory system if the heart were stopped suddenly and the blood were redistributed instantaneously in such a manner that all pressures were equal.
- Mean systemic filling pressure (MSFP) is the pressure in only the systemic circuit, i.e. ignoring the heart and pulmonary circulation, also in the absence of flow. this is the pressure which is thought to push blood towards the right atrium along a pressure gradient.

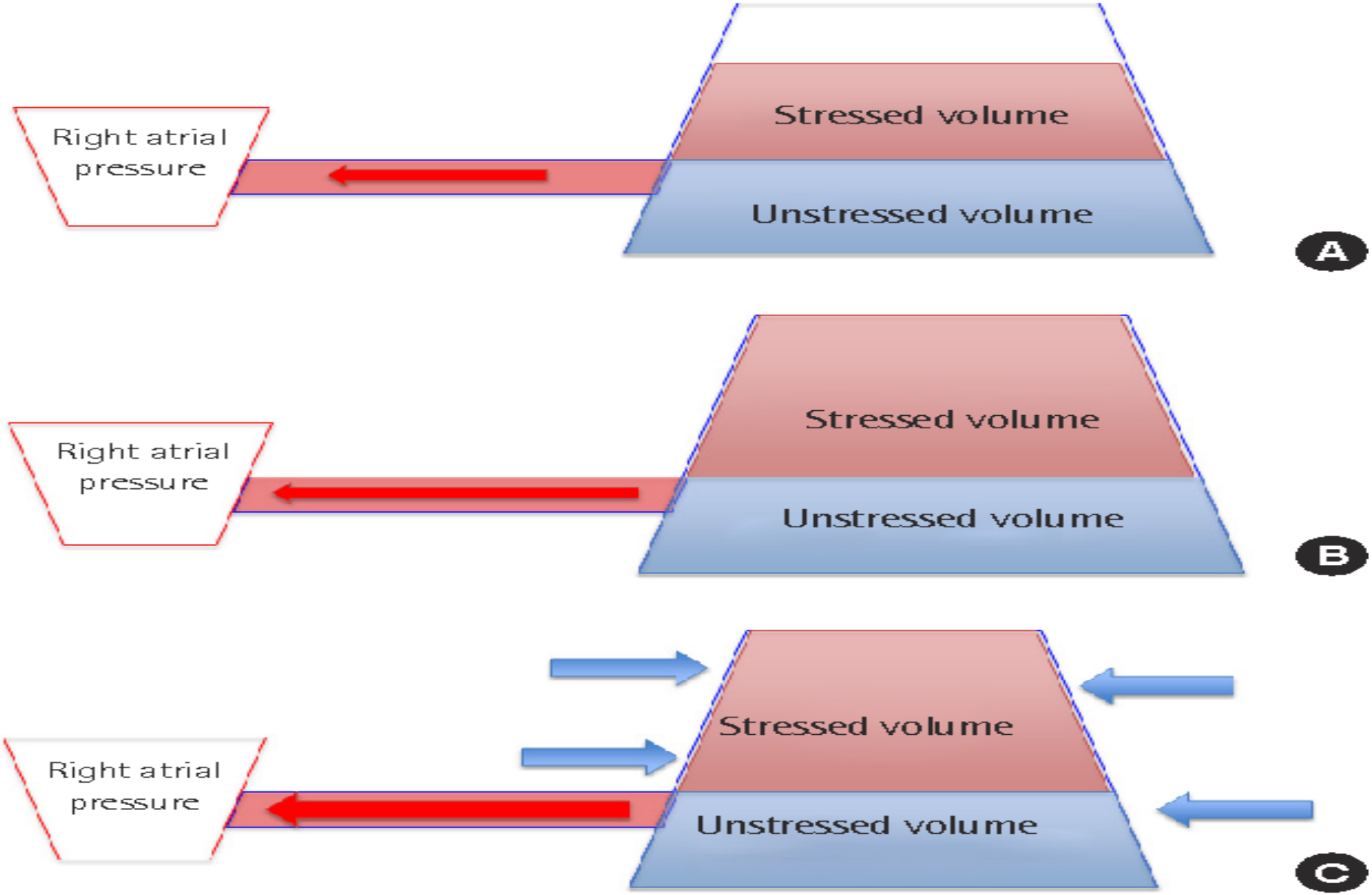
• Mean cardiopulmonary filling pressure (MCPFP) is the mean pressure in the motionless cardiac chambers and the pulmonary circulation. It is usually about 3 mmHg higher. It is usually about 3 mmHg higher than the MSFP, mainly because of the higher elastance of the cardiac chambers.

• The main determinants of MCFP and MSFP are total blood volume and venous resistance





- ***The "unstressed" volume*** is said to be a volume of fluid (presumably, blood) in the circulatory system which does not produce any "stress" on the walls, i.e. where measuring the MSFP would yield a pressure of 0 mmHg. According to Young (2010) and Magder (2016), "unstressed volume" describes about 85% of the total venous blood volume
- ***stressed volume***" is therefore "the volume of blood that must be removed from the vasculature to decrease the transmural pressure of the vessels from the existing value to zero
- in a circulation with minimal sympathetic tone, only about 15% of the total blood volume is contributing to generating the MSFP.
- It is about ***4 cc/Kg*** or ***300 cc*** in NL sized human or 6% total circulation volume


Right atrial pressure
Stressed volume
Unstressed volume
A
Right atrial pressure
Stressed volume
Unstressed volume
B
Right atrial pressure
Stressed volume
Unstressed volume
C

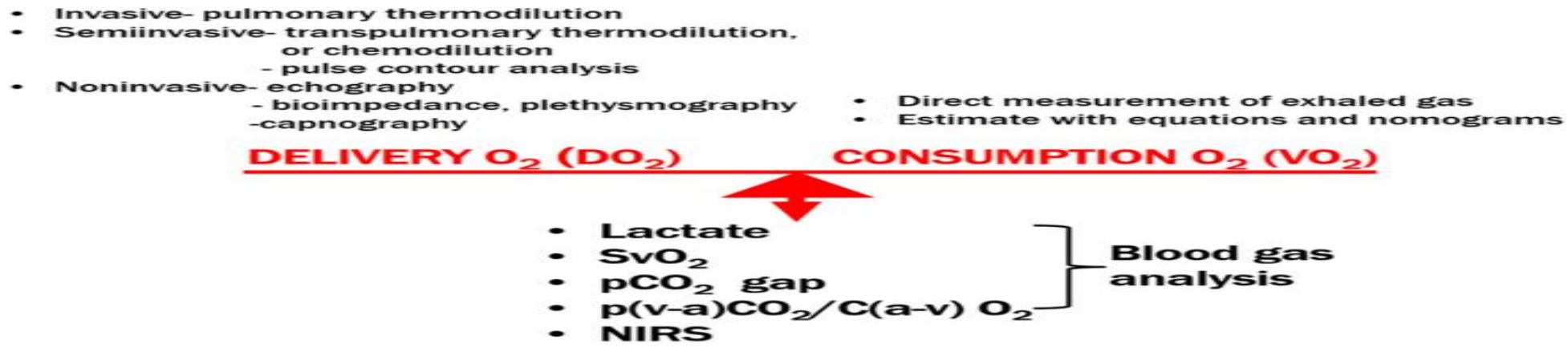
- Let us examine this model in a clinical setting such as septic shock. The hypotension observed in ***sepsis*** is typically a distributive process. Essentially the vascular bed has vasodilated causing a relative hypovolemia. The total volume status is unchanged, but the vasodilation has caused an increase in the vascular compliance. This shifts a portion of the stressed volume to an unstressed state(mal-distribution), leading to a decrease in the Pms, and in turn the venous return.
- In the hopes of correcting the physiologic perturbations induced by the septic state, it is not uncommon to attempt to implement changes by manipulating the stressed volume. Typically this is done in two fashions. **First**, one can add to the total volume of the system (in the form of a fluid bolus), which will increase both the stressed volume and the total volume. **Second**, one can promote a reduction in the vessel wall compliance (with the addition of vasopressor agents), causing a change in the ratio of volume in the stressed and unstressed states. In this case the total volume would stay constant, while the unstressed volume decreases and the stressed volume increases.

- Now instead let us examine the effects of hemorrhagic shock on the stressed and unstressed volume. In acute blood loss the total volume will be reduced, leading to a decrease in the stressed volume.
- In the initial phases of compensated shock the body attempts to adapt for this loss with a catecholamine-induced venoconstriction. This compensatory measure decreases the compliance of the venous system shifting blood from the unstressed to the stressed volume, increasing the Pms and temporarily maintaining the venous return. If bleeding is not controlled, blood loss will outpace these compensatory venoconstrictive efforts. At this point further attempts to augment preload through the shifting of unstressed to stressed volume will not improve venous return. Volume replacement is now required. The replacement of lost blood with blood products, is an attempt to restore both the total volume and stressed volume to a more physiologic state.
- Early use of low dose vasopressors (norepinephrine at 5 mcg/min) will result in a venoconstrictive effect, decreasing venous compliance, and shifting fluids from the unstressed venous beds to the useable stressed volume.
- Applying Guyton's approach, to increase venous return means decreasing central venous pressure (CVP, the backward pressure) or increasing Pmsf.

Oliguria ;and increased lactate as tissue hypoperfusion

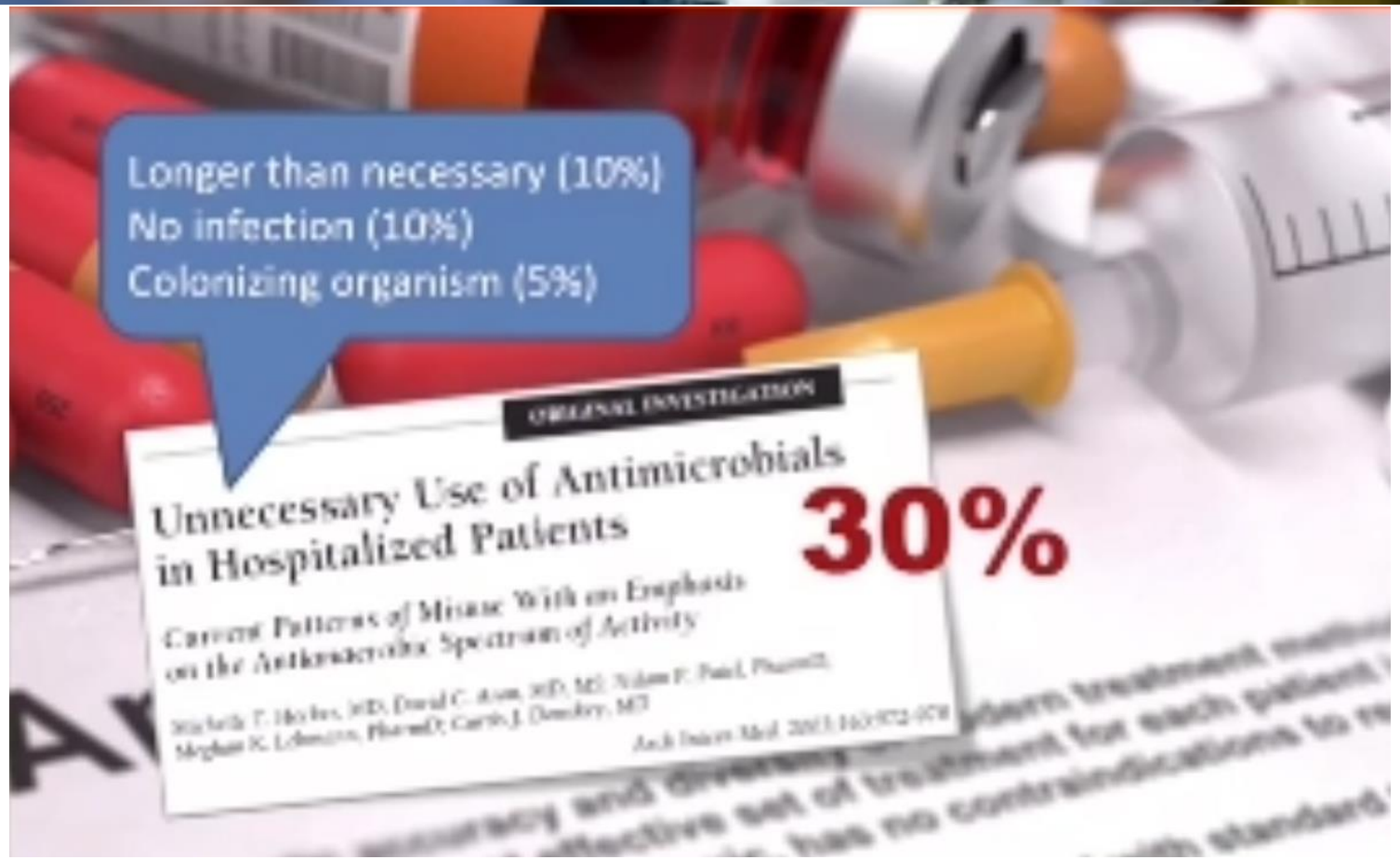
- Fluid resuscitation in septic shock is an effective intervention to increase venous return, and thus cardiac output (CO) and oxygen transport
- As only 50–60% of the patients in the early phase of septic shock are fluid responders, and 25% may already be fluid unresponsive after an initial fluid resuscitation

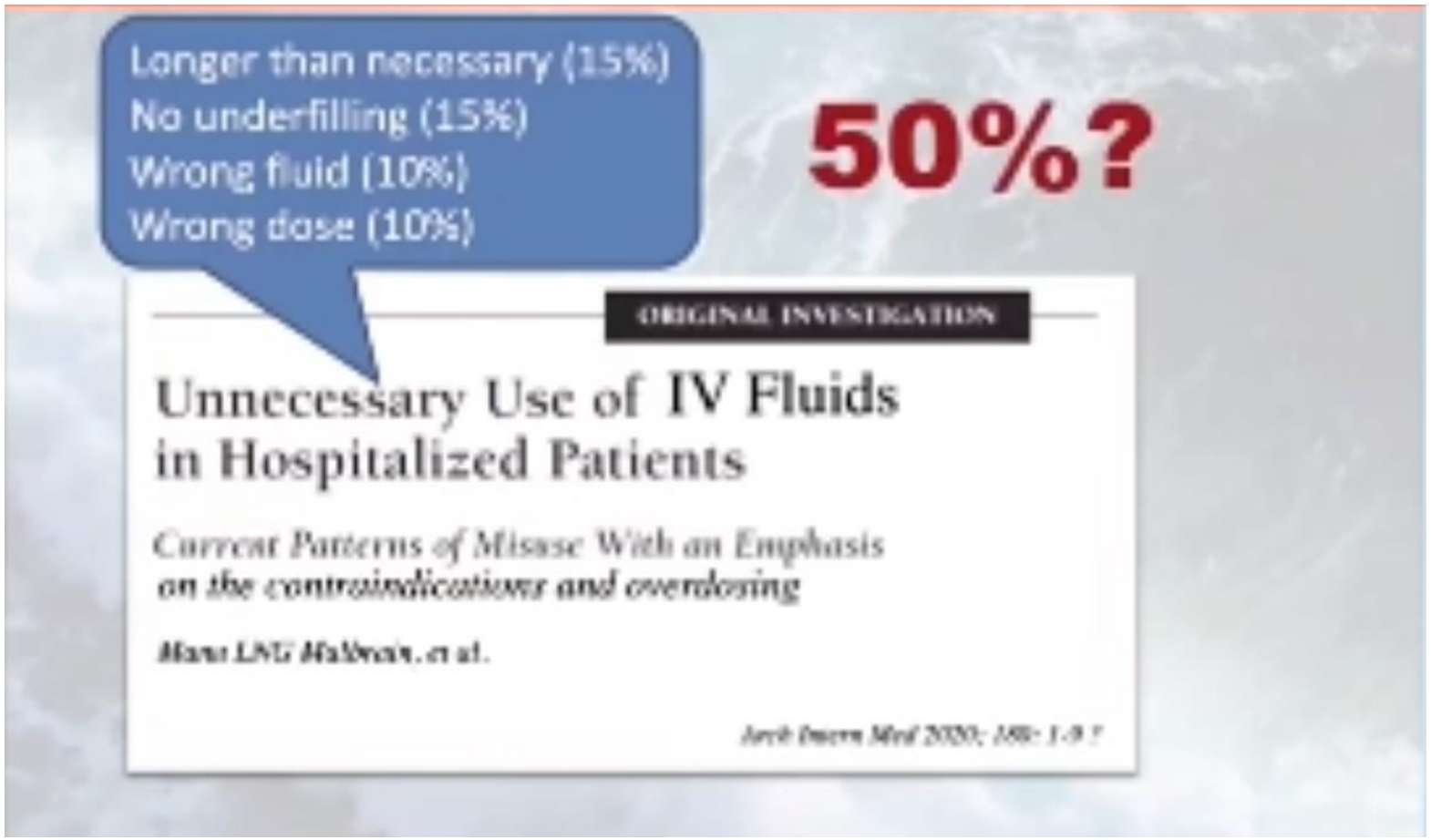
in an ovine model of hyperdynamic septic shock, oliguria occurred despite dramatic increases in both cardiac output and renal artery blood flow . These observations strongly suggest that oliguria is not a function of decreased renal perfusion during sepsis.




“ENOUGH
WITH THE
PLEASANTRIES,
LET’S MAKE
A PLAN”


ANTIBIOTIC
STEWARDSHIP


Longer than necessary (10%)
No infection (10%)
Colonizing organism (5%)
ORIGINAL INVESTIGATION
Unnecessary Use of Antimicrobials in Hospitalized Patients
30%


Longer than necessary (15%)
No underfilling (15%)
Wrong fluid (10%)
Wrong dose (10%)
50%?
ORIGINAL INVESTIGATION
Unnecessary Use of IV Fluids in Hospitalized Patients
Current Patterns of Misuse With an Emphasis on the Contraindications and Overdosing

Fluid Stewardship

Dr. Marcia McDougall

How could you not do this ?

Why ? Unwarranted Variations = Waste & Suboptimal Outcomes

→ Standardising Assessment and Prescribing

→ Reducing the number of fluids we use

→ Good fluid balance charting

Simplifying our systems

A WHOLE HOSPITAL APPOACH

The **right amount** of the **right fluid** at the **right time**

Why?
- maintenance
- replacement
- resuscitation

the way forward

Education

Good for patients, staff and saves resources/money

The 7 Ds of Fluid Therapy

D1: Definitions
D2: Diagnosis
D3: Drug
D4: Dose
D5: Duration
D6: De-escalation
D7: Discharge

Fluid stewardship

Everything you need to know about Fluid therapy (the 7D's)

Manu Malbrain

Personalised Fluid Therapy

Fluid Team

D1 Definitions

- Fluid Overload
- Hypervolemia
- Edema

D2 Diagnosis

Hypovolemia?

Monitoring

- Clinical
  - Organ Function
  - 4 Compartments
- Laboratory
  - Radiology
  - BIA Body Composition

D3 Drug

(Contra)Indications

4 Questions

- Start IVF
- Stop IVF
- Start FR
- Stop FR

4 Indications

- Resuscitation
- Maintenance
- Replacement
- Nutrition

Adverse effects

- (In)appropriate therapy
- Combination therapy
- Timing

D4 Dose

- Pharmaco-Kinetics
- Pharmaco-dynamics
- Toxicity

D5 Duration

- Appropriate duration
- Treat to response

D6 De-escalation

ROSE

4 Phases

- Resuscitation
- Optimization
- Stabilization
- Evacuation

- Clinical
  - Organ Function
- Laboratory
  - Radiological

4 HITS

- 1st Insult
- IR
- GIPS
- Hypovol

D7 Discharge

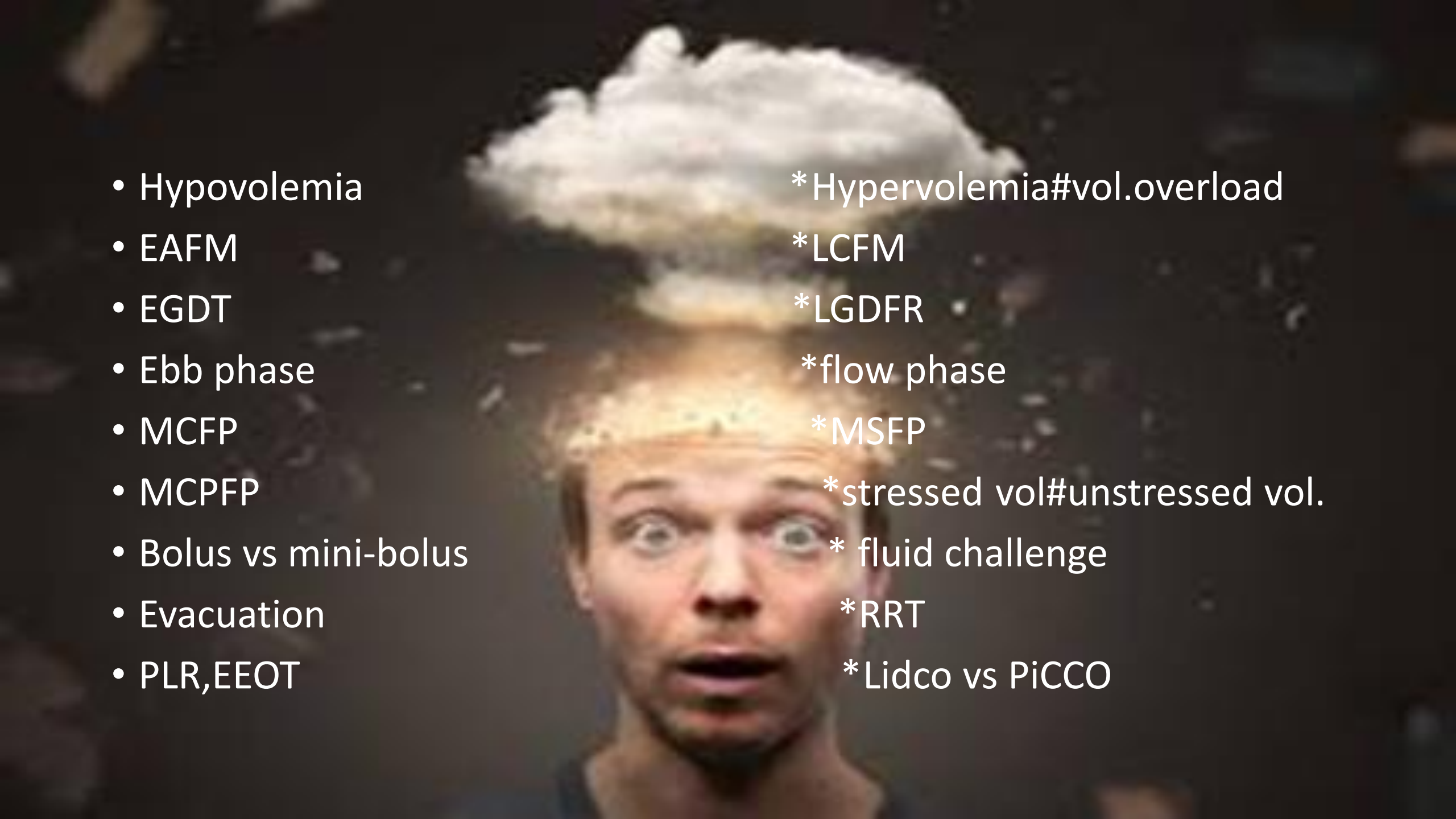
Fluid Stewardship

- Snapshot
- Survey
- Audit
- KPI
- PDCA

fluids ≠ Innocent

$D^7 = SF$ (Fluid Stewardship)

D1:



- Hypovolemia
- EAFM
- EGDT
- Ebb phase
- MCFP
- MCPFP
- Bolus vs mini-bolus
- Evacuation
- PLR,EEOT

*Hypervolemia#vol.overload
*LCFM
*LGDFR
*flow phase
*MSFP
*stressed vol#unstressed vol.
* fluid challenge
*RRT
*Lidco vs PiCCO

Definitions of Fluid Therapy

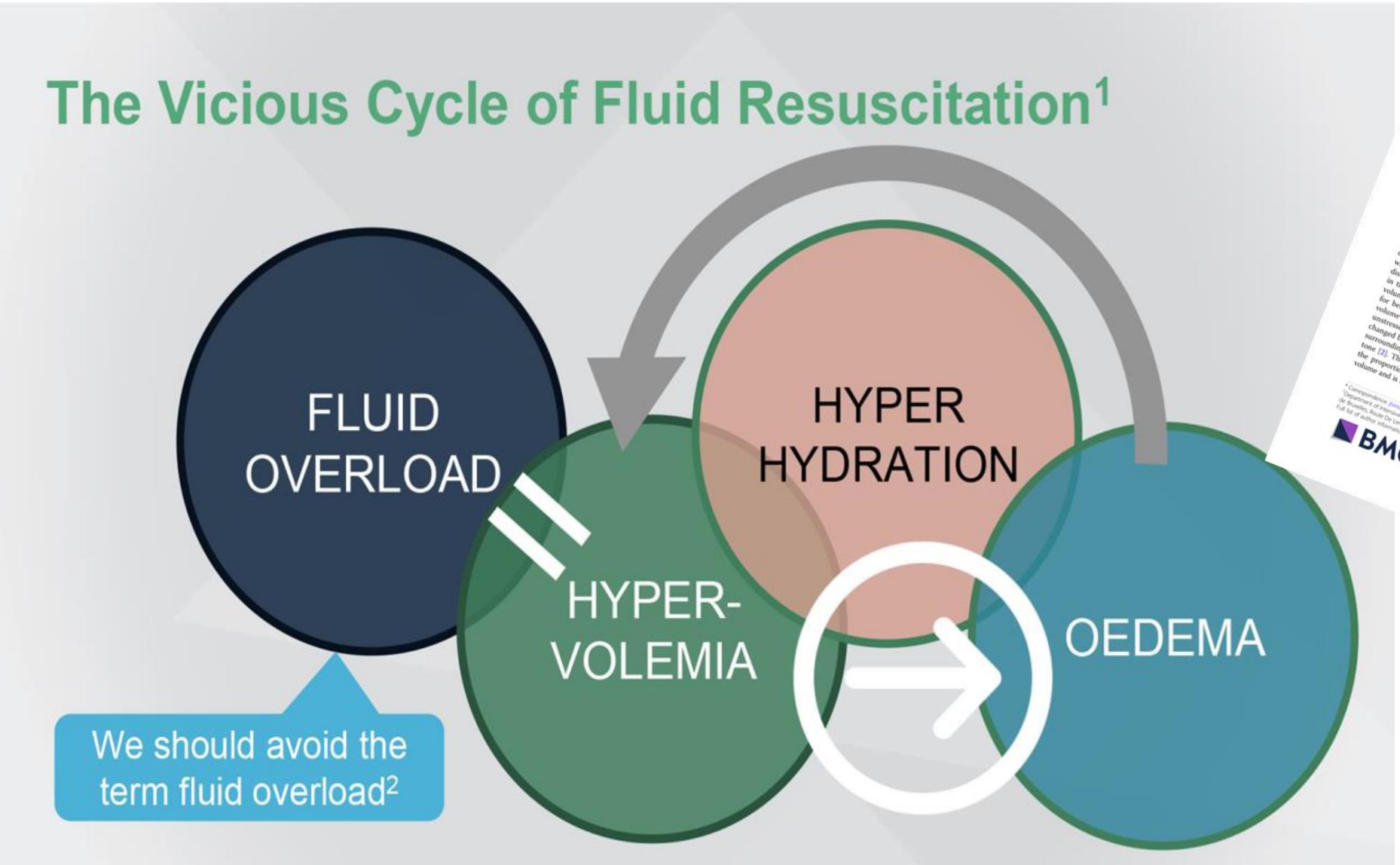
- Fluid Balance: Daily fluid balance is the daily sum of all intakes and outputs, and the cumulative fluid balance is the sum total of fluid accumulation over a set period of time
- **Fluid Overload:** Dividing the cumulative fluid balance in litres by patient's baseline body weight and multiplying by 100% defines the percentage of fluid accumulation
  - Fluid overload is defined by a **cut-off value of 10%** of fluid accumulation, as this is associated with worse outcomes
- Early Adequate Fluid Management (EAFM):
  - Goal-directed treatment
  - On average, 30 mL/kg within first 1-3 hours (SSCG)
- Late Conservative Fluid Management (LCFM):
  - 2 consecutive days of negative fluid balance within the first week of ICU stay
- **Late Goal-Directed Fluid Removal (LGFR):**
  - Active fluid removal by means of diuretics or renal replacement therapy with net ultrafiltration
  - Referred to as **de-escalation** or **de-resuscitation**

- ***EAFM:*** early adequate fluid management, defined as fluid
- intake > 50 mL/kg/first 12–24 h of ICU stay.
- ***ECFM:*** early conservative fluid management, defined as fluid intake < 25 mL/kg/first 12–24 h of ICU stay.

- **Fluid overload**
- As often described in pediatric populations, the percentage
- of fluid accumulation is calculated by dividing
- the cumulative fluid balance in liters by the patient’s
- baseline body weight and multiplying by 100%. Fluid
- overload at any stage is defined by a cut-off value of
- 10% of fluid accumulation, as this is associated with
- worse outcomes

- ***Late goal-directed fluid removal*** involves aggressive and active fluid removal using diuretics and renal replacement therapy with net ultrafiltration.
- ***Late conservative fluid management*** describes a moderate fluid management strategy following the initial treatment in order to avoid (or reverse) fluid overload.
- ***LLFM: late liberal fluid management***, defined as the absence of 2 consecutive negative daily fluid balances within first week of ICU stay

FLUID OVERLOAD vs HYpervolemia



EDITORIAL

We should avoid the term "fluid overload"

Critical Care

Open Access

Jean-Louis Vincent* and Michael R. Pinsky

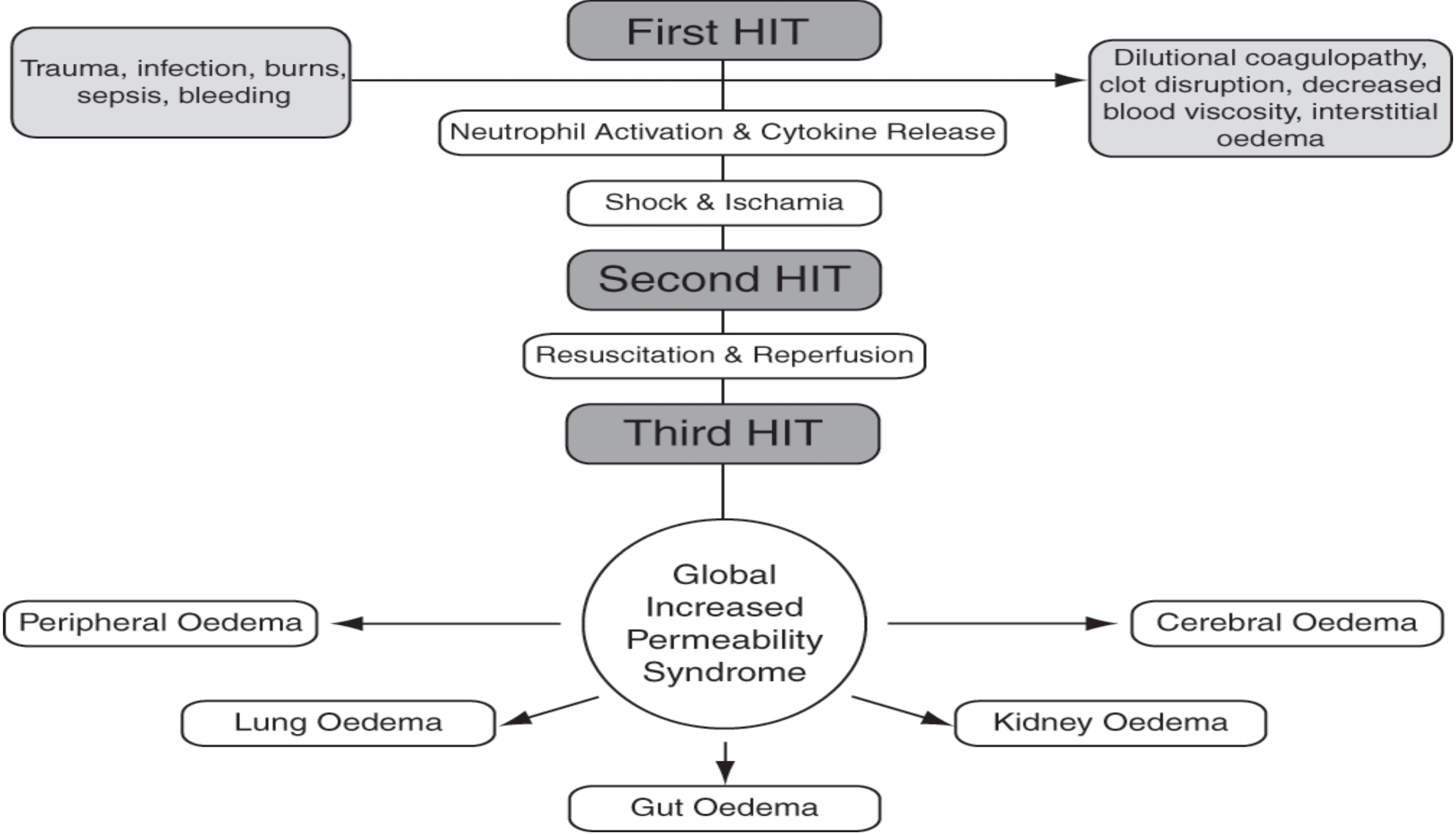
Keywords: Hypervolemia, Blood volume, Edema, Fluid administration, Shock

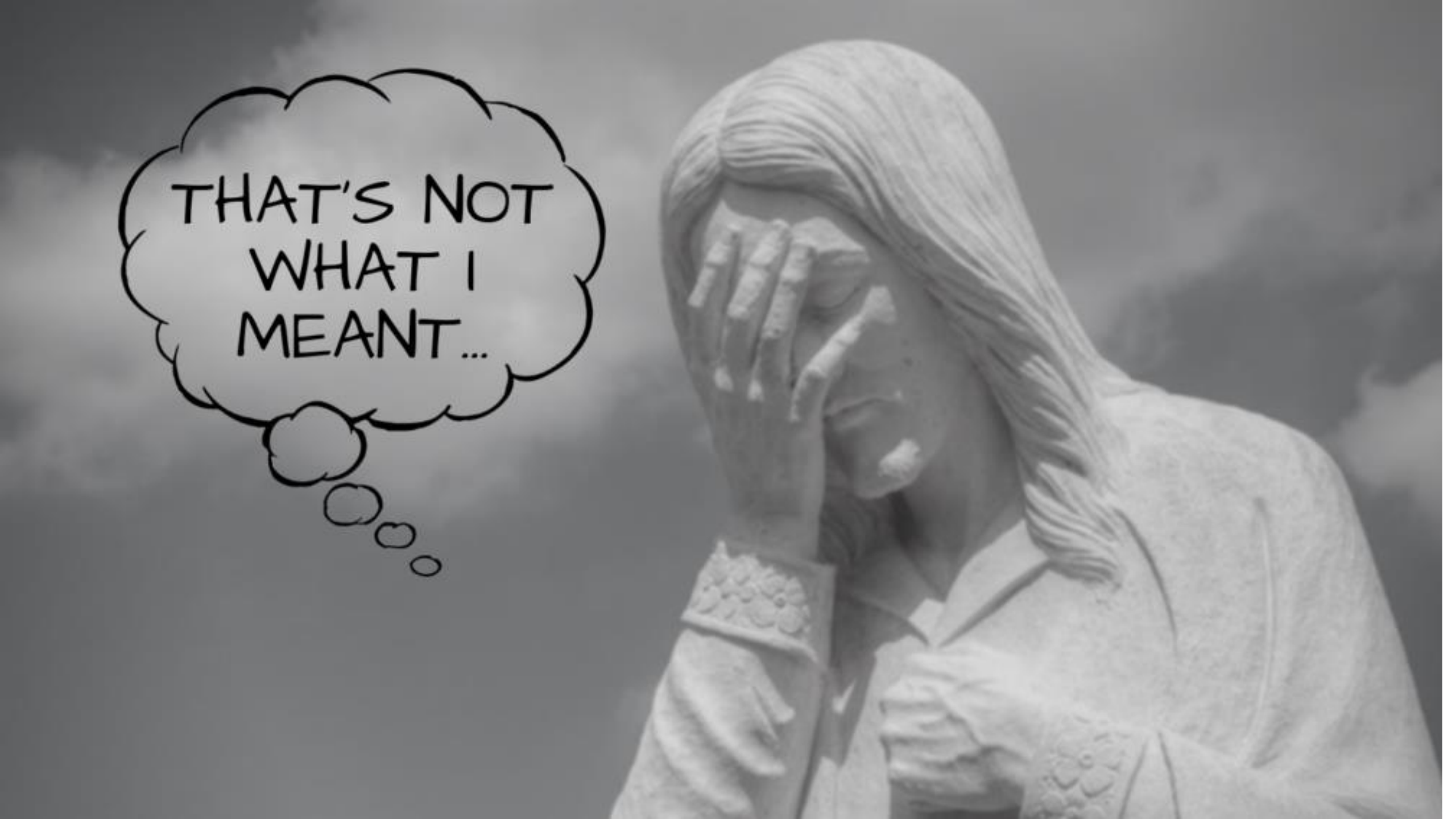
BMC

WHAT: Definitions

- *Global Increased Pemeability Syndrome (GIPS)*:
  - Combination of persistent positive cumulative fluid balance
  - Together with new onset organ failure
  - It is characterised by the absence of transgression from Ebb to Flow phase of shock
- *Acute Intestinal Distress Syndrome (AIDS):*
  - Confusing term – may refer to HIV
  - Re-classified as Acute Gastro-intesinal Injury (AGI)

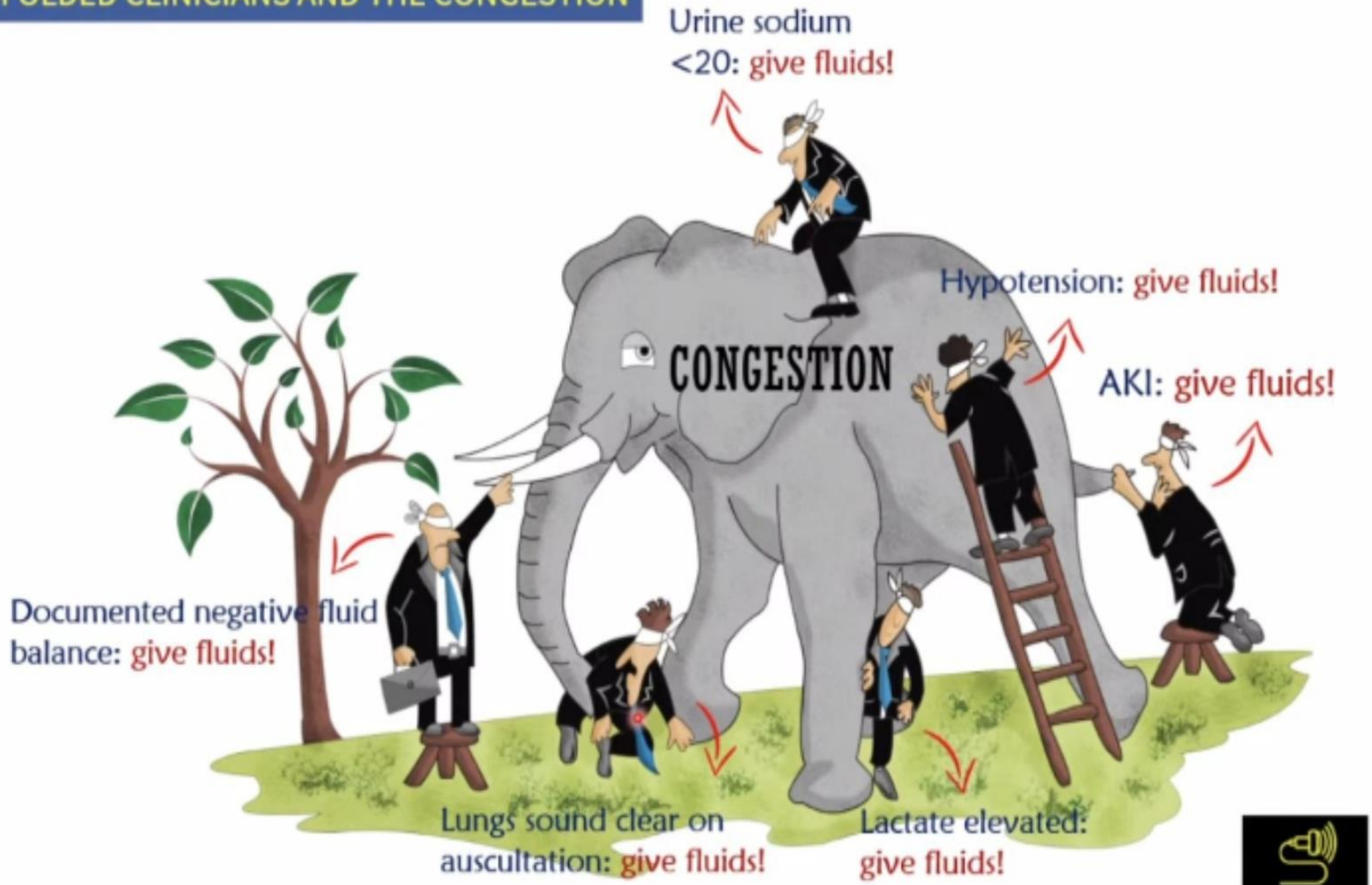
GIPS


First HIT
Trauma, infection, burns, sepsis, bleeding
Dilutional coagulopathy, clot disruption, decreased blood viscosity, interstitial oedema
Neutrophil Activation & Cytokine Release
Shock & Ischamia
Second HIT
Resuscitation & Reperfusion
Third HIT
Global Increased Permeability Syndrome
Peripheral Oedema
Cerebral Oedema
Lung Oedema
Kidney Oedema
Gut Oedema

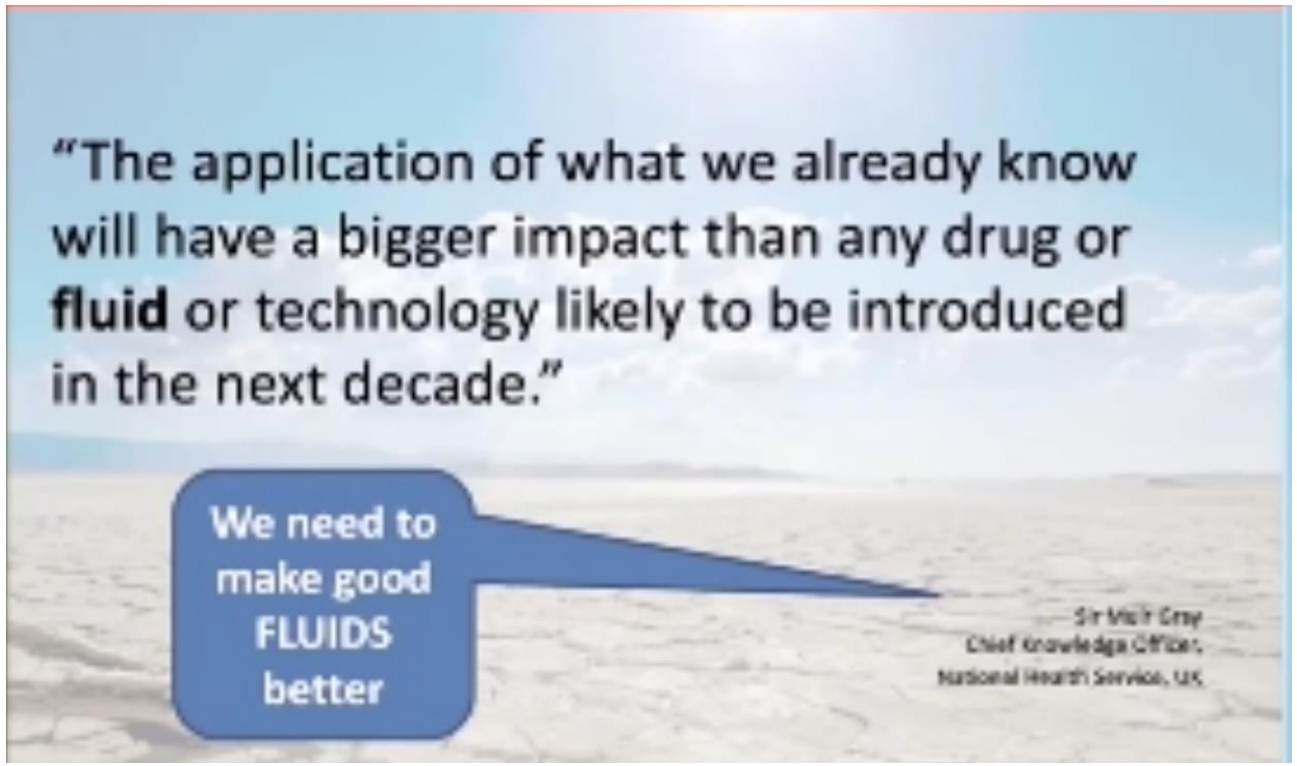

THAT'S NOT WHAT I MEANT...

D2:

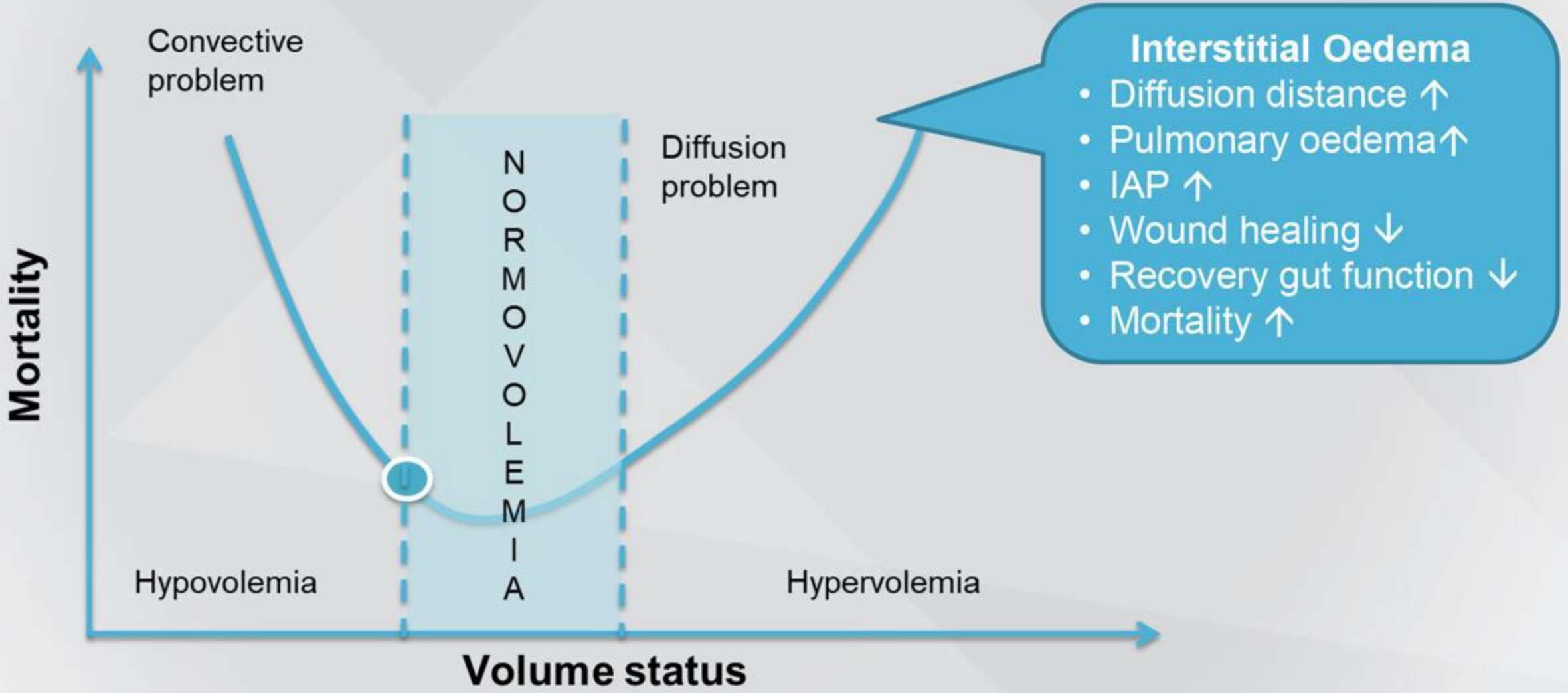
BLINDFOLDED CLINICIANS AND THE CONGESTION



NephroPOCUS


"The application of what we already know will have a bigger impact than any drug or **fluid** or technology likely to be introduced in the next decade."
We need to make good FLUIDS better
Chief Knowledge Officer,
National Health Service, UK

Hypovolemia Is Bad, But Hypervolemia Is Even Worse



FLUID MANAGEMENT IN AKI

Fluid therapy: Volume

Myths of Fluid Therapy

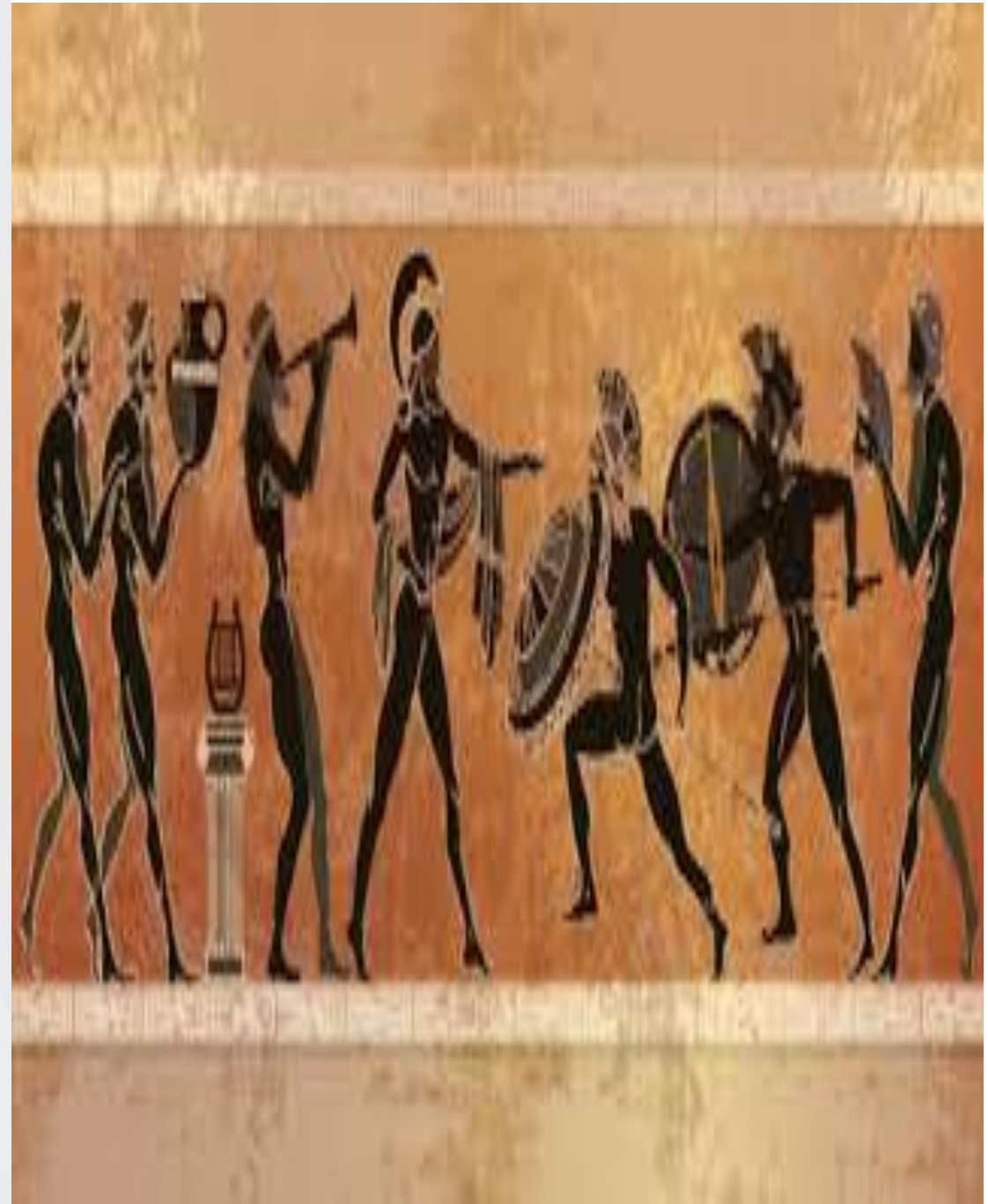
Septic patients ≠ Hypovolemic

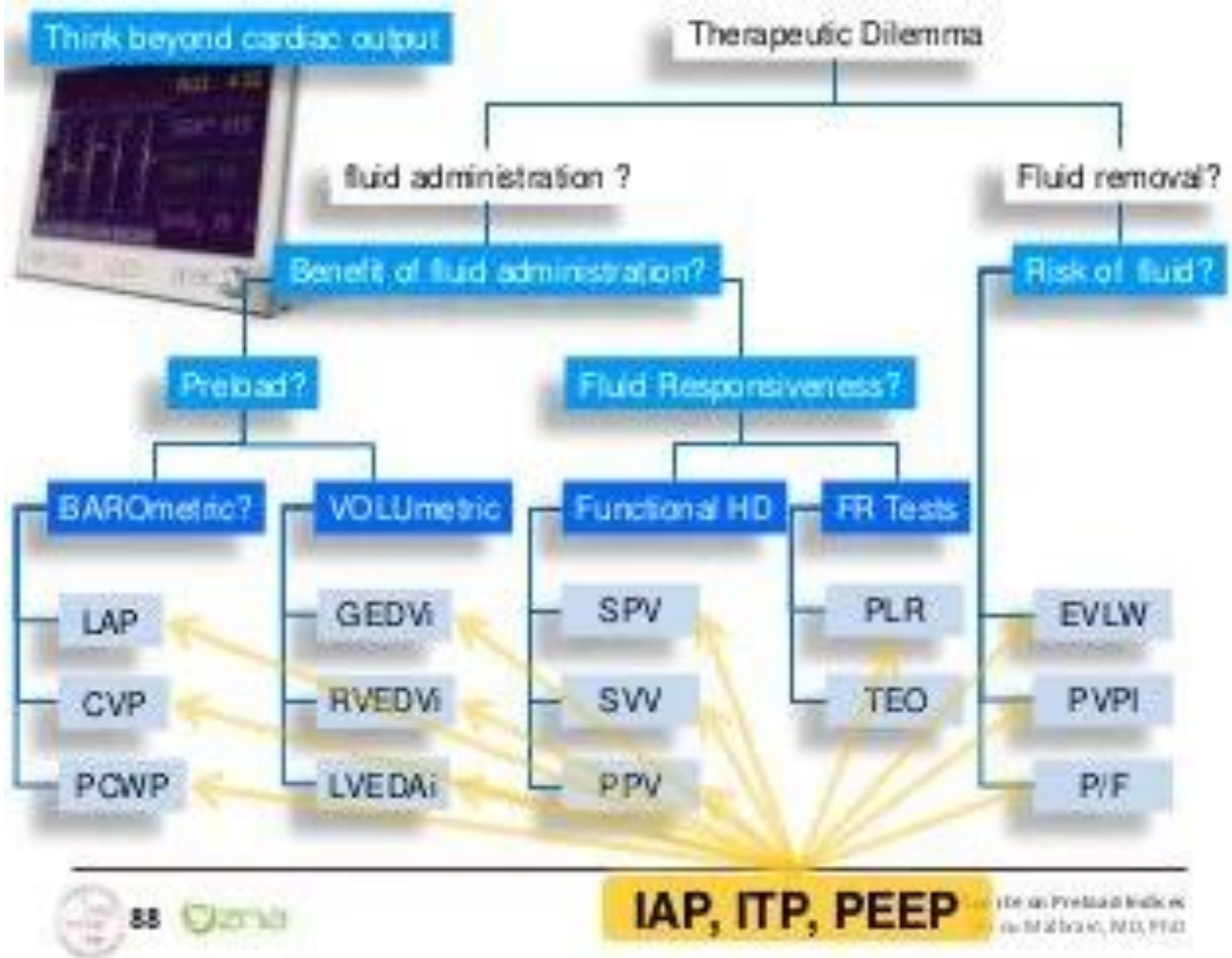
Surgical patients ≠ Hypovolemic

Low preload ≠ Give fluids

Sepsis ≠ Insensible loss

Oedema ≠ Give diuretics




Think beyond cardiac output
Therapeutic Dilemma
fluid administration ?
Fluid removal?
Benefit of fluid administration?
Risk of fluid?
Preload?
Fluid Responsiveness?
BAROmetric?
VOLUmetric
Functional HD
FR Tests
LAP
CVP
PCWP
GEDVi
RVEDVi
LVEDAi
SPV
SVV
PPV
PLR
TEO
EVLW
PVPI
P/F
IAP, ITP, PEEP

Clinical

- Body weight
- Fluid balance
- Cumulative FB
- Pitting edema
- 2nd and 3rd space fluid accumulation (orthopnee)
- JVP and HJR
- Capillary refill

Biochemical

- AKI, urinanalysis
- Urine albumin/creat ratio
- Dilutional anemia
- Low albumin, protein
- Infection, inflammation
- Increased CLI (CRP/alb)
- Low osmol, COP
- BNP and NT-pro-BNP

Imaging

- Cardiomegaly, congestive hili, Kerley B-lines, pleural effusions, lung edema
- Ascites
- E/e', LVOT VTI
- IVVCI (IVC > 2.5cm)
- LA volume >34ml/m2
- B-lines, comet-tail

Hemodynamic

- MAP, HR
- High CVP, PAOP
- High GEF/GEDVI
- High RVEF/RVEDVI
- Absence of FR
- Low PPV, SVV
- Negative PLR
- Negative EEO

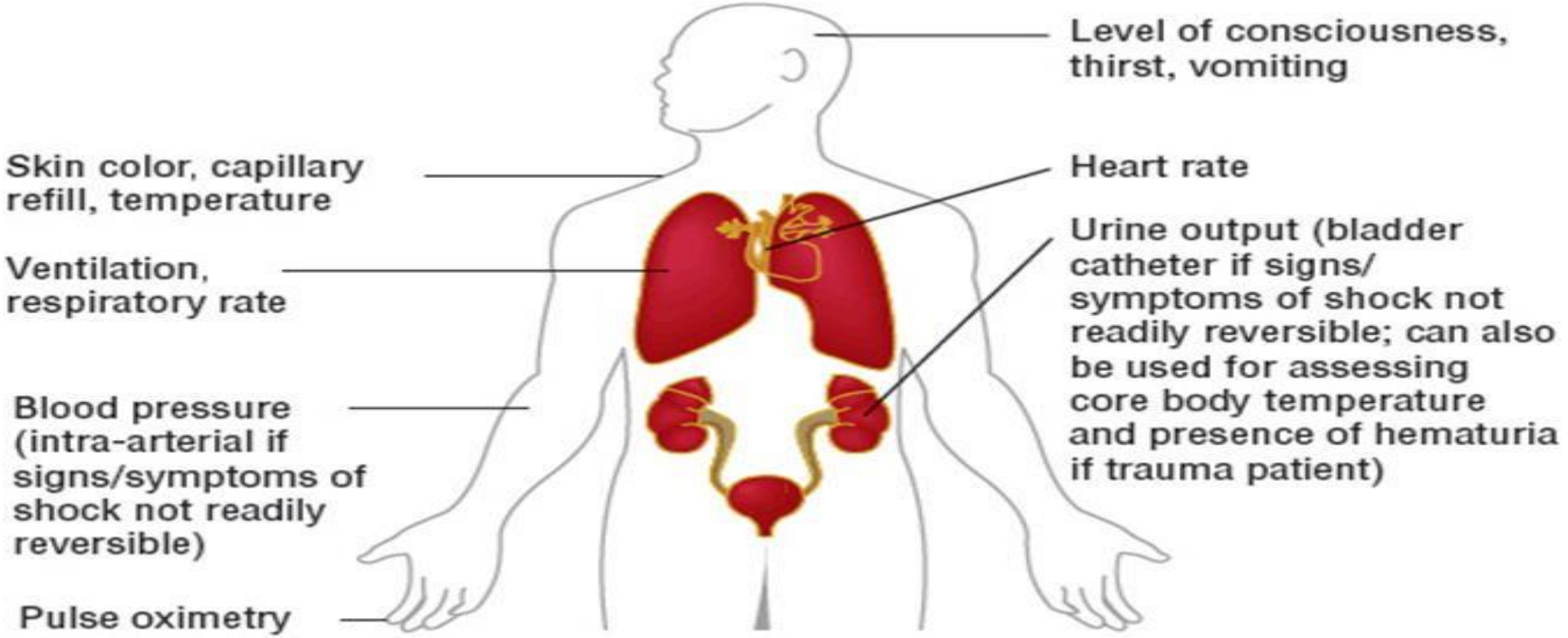
Organ Function

- EVLWI/PVPI
- P/F ratio
- ORI
- GIPS
- ICP, CPP (=MAP-ICP)
- IAP, APP (=MAP-IAP)
- AKI: biomarkers (N-GAL, cystatin C), CARS

Other (BIA, EIT)

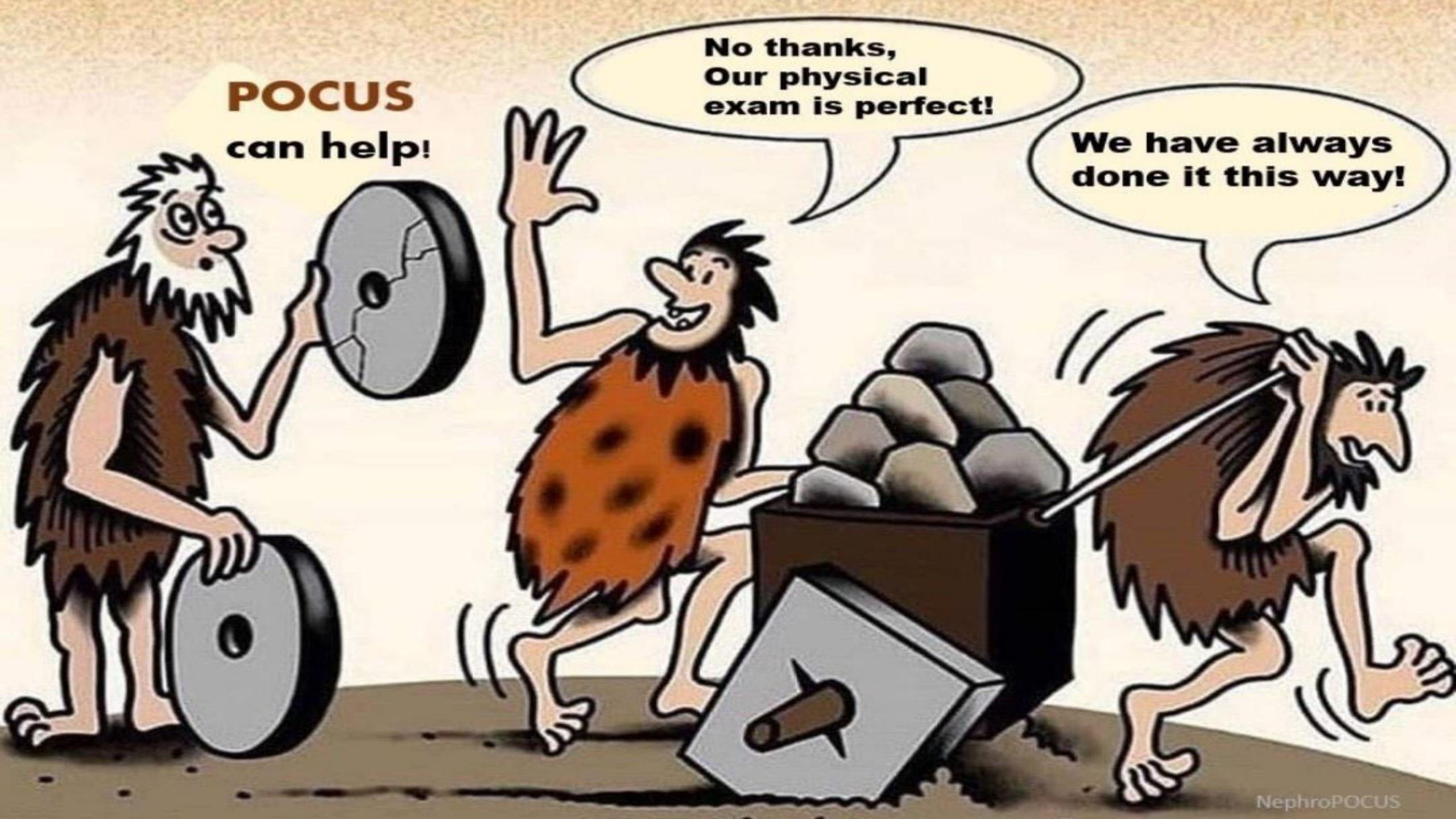
- V/Q match
- TBW
- ECW/ICW ratio
- Volume excess
- IVF vs EVF
- Total CBV (technetium albumin)
- BVI

clinical signs of hypovolemia



Laboratory signs of hypovolemia

- ↓SCVO2,↑Lactate
- ↑Albumin leak index(urine albumin/urine cr. Ratio)
- ↑Hct and Hb
- ↑serum Na
- ↑total pr. And Alb.
- ↑serum osmolality,and COP
- ↓BNP and Pro-BNP
- In urine:↑osm. ↓Na


POCUS
can help!
No thanks,
Our physical
exam is perfect!
We have always
done it this way!
NephroPOCUS

Does the Central Venous Pressure Predict Fluid Responsiveness? An Updated Meta-Analysis and a Plea for Some Common Sense*

Paul E. Marik, MD, FCCM[1]; Rodrigo Cavallazzi, MD[2]

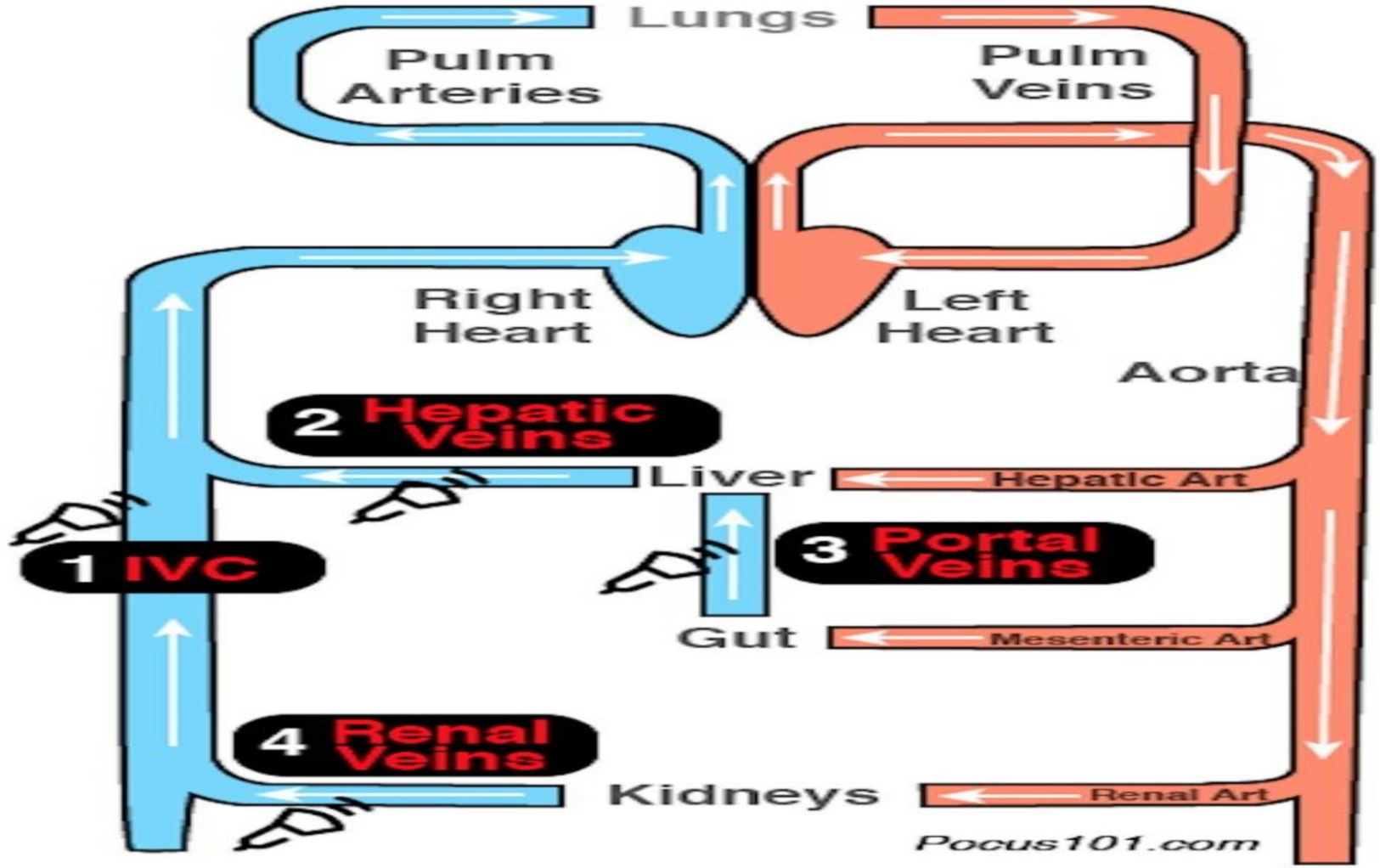
Crit Care Med 2013; 41:1774-81

43 articles 1802 patients

AUC = 0.56

There are no data to support the widespread practice of using central venous pressure to guide fluid therapy. This approach to fluid resuscitation should be abandoned.

Venous Excess Ultrasound
VExUS



Invasive monitorings

"It's OK, this is a teaching hospital. Some people just have to learn that the hard way."

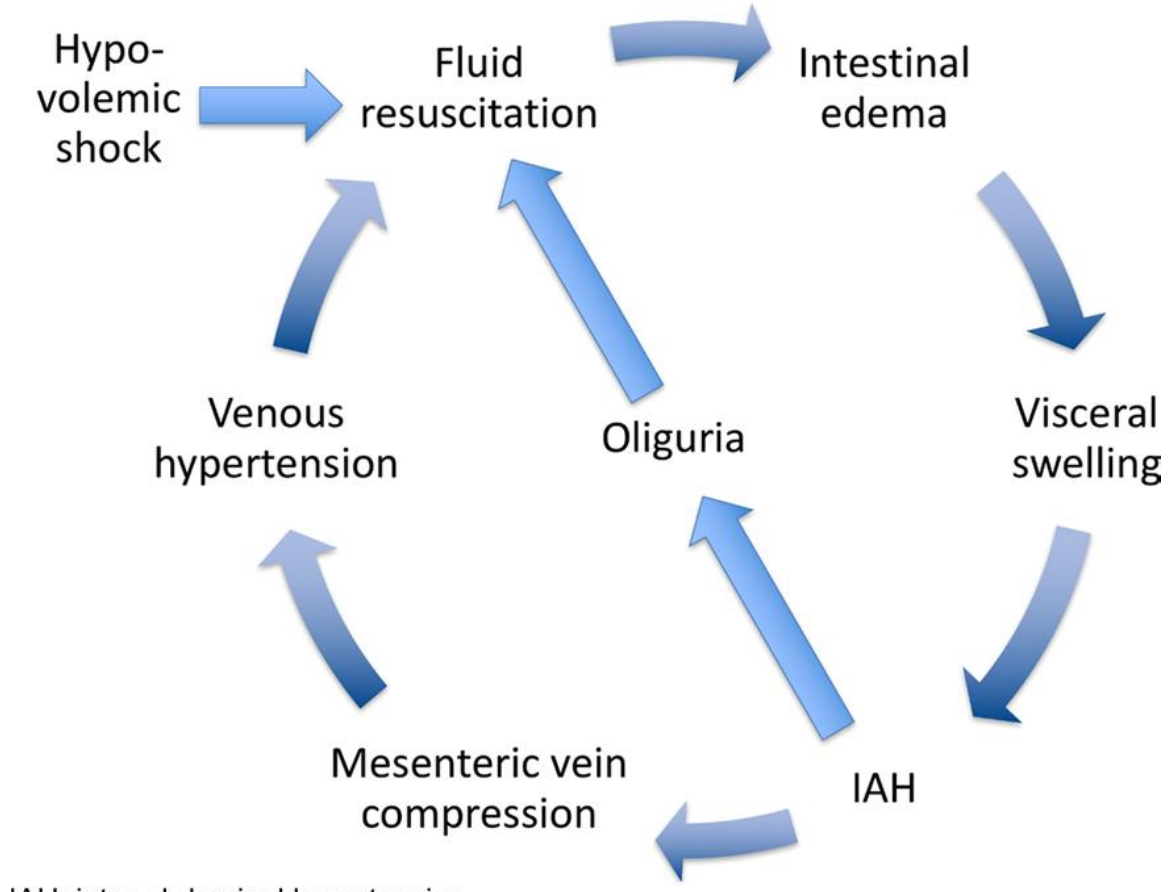
Why Patients with Sepsis Die?

Cellular Bioenergetic (Metabolic) Failure

~~Tissue Hypoxia~~

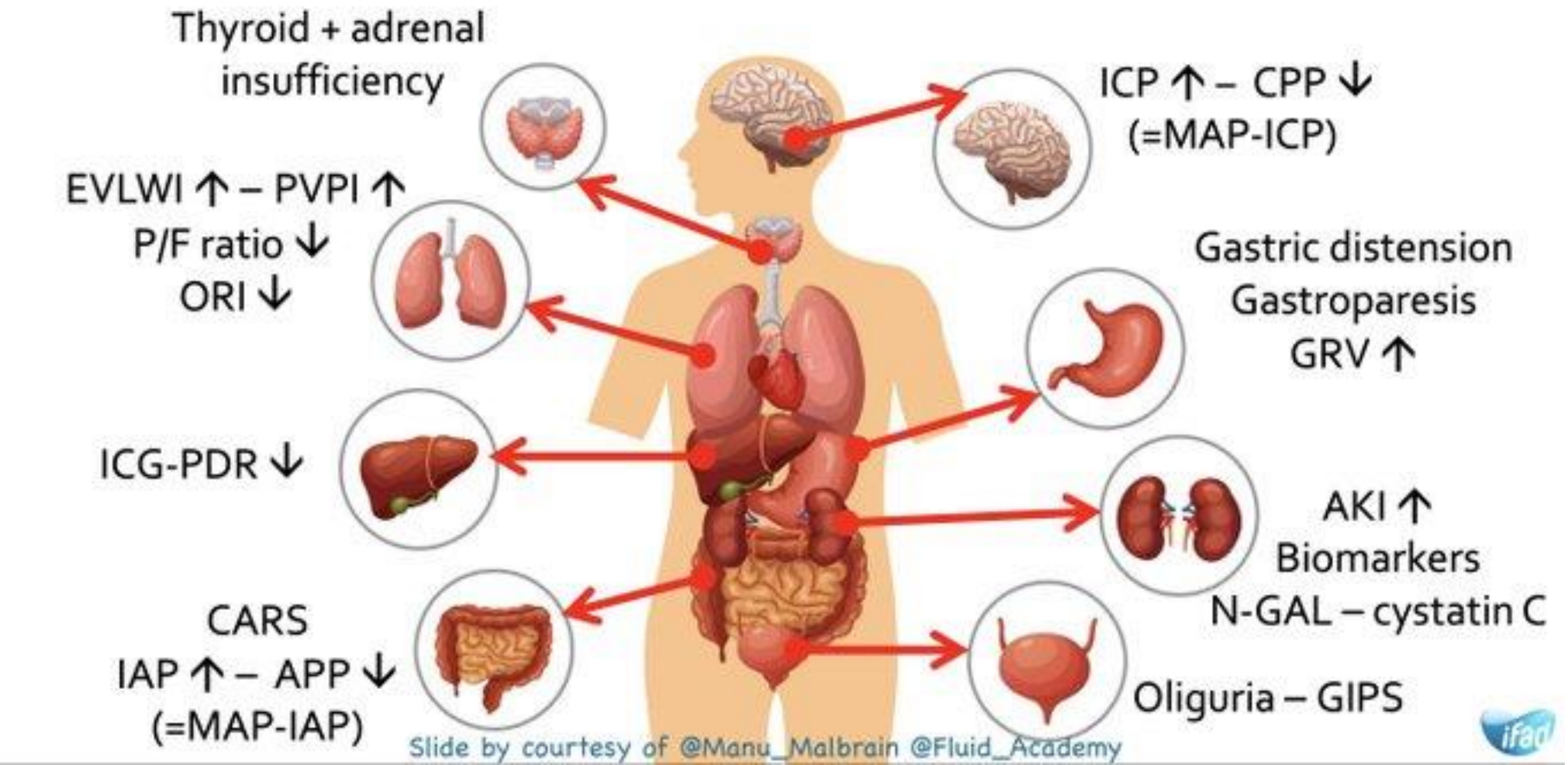

AVOID
FLUID
OVERLOAD

The vicious cycle of septic shock resuscitation



IAH: intra-abdominal hypertension

Organ Function Assessment Fluid Overload



Tests to assess Response to IV fluid

- ***Static surrogates***: parameters are often used to titrate fluid therapy(pre-load)

-central venous pressure (CVP :8-12 mmHg)

-Mean arterial pressure (MAP)

-Urine output(>0.5-1 cc/KG/Hr)

-Volumetric pre-load(GEDVI,RVEDVI,LVEDAI)

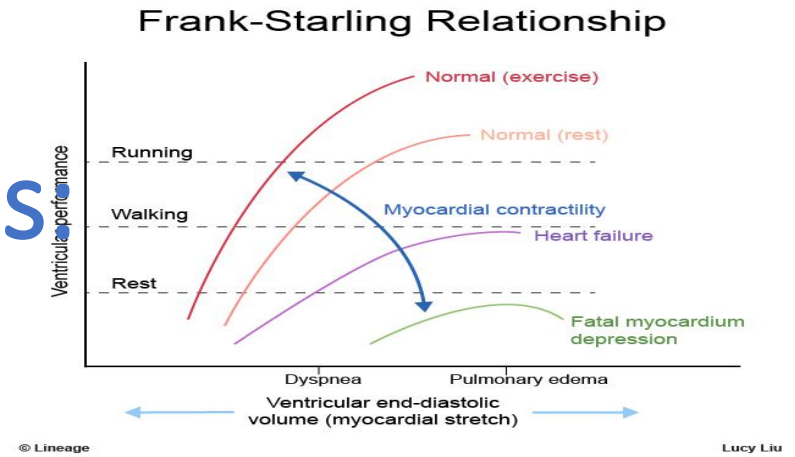
Dynamic functional hemodynamic parameters

-PPV(or SVV)

Dynamic tests to predict response to IVF

-PLR or EXOT

1. Fluid responsiveness



- The foundation of fluid resuscitation
- In patients with shock, there is tissue hypoperfusion and decreased cardiac output;Fluid administration can be beneficial if it increases stroke volume, and therefore, cardiac output.
- Increased stroke volume leads to increased end-diastolic volume and mean circulating filling pressure, thereby increasing preload and causing volume expansion.
- The goal in shock is to steer patients onto the ascending limb of the Frank Starling curve.
- When fluid administration does not increase stroke volume, there are potential harms.
- Excess fluid administration can lead to tissue edema and ultimately, tissue hypoxia and organ dysfunction.

- **Fluid responsiveness** indicates a condition in which a patient will respond to fluid administration by a significant increase in stroke volume and/or cardiac output or their surrogates. A threshold of 15% is most often used for this definition, as it is the least significant change of measurements of the techniques that are often used to estimate cardiac output
- only ***one half*** of patients in ICUs with circulatory failure respond to an increase in cardiac output

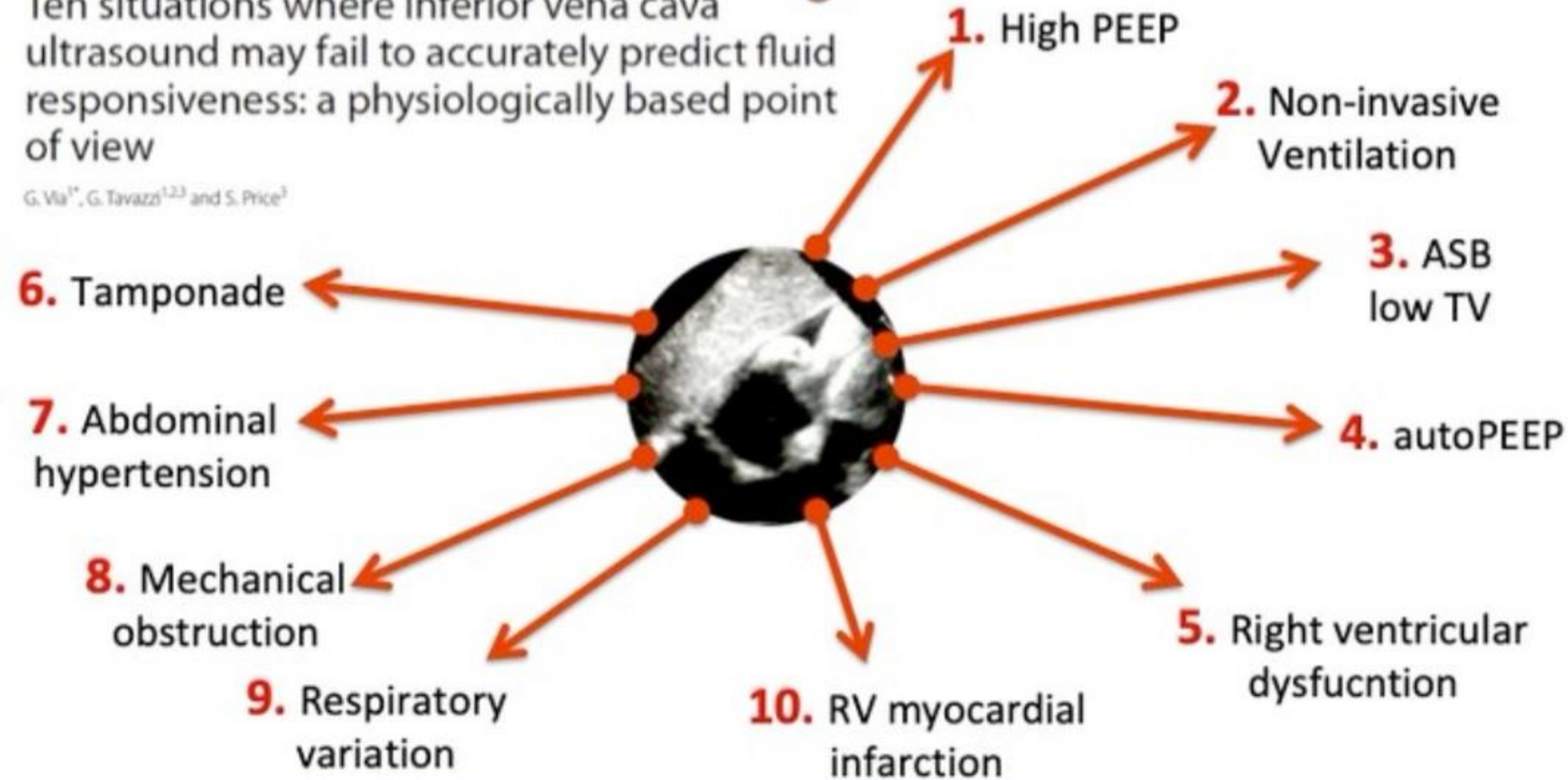
- Many septic patients have preceding heart failure, about 54% with diastolic dysfunction and 23% with systolic dysfunction, which can be worsened by fluid administration (Landesberg 2012). Over-stretching of the left ventricle (LV) causes impaired LV dilation, leading to pulmonary edema, pulmonary hypertension, and right ventricular dysfunction. In fact, diastolic dysfunction was associated with worse outcomes than systolic dysfunction (Landesberg 2012).
- A fluid challenge is necessary to determine whether fluid administration will benefit a patient.
- A patient is considered to be fluid responsive if their stroke volume increases by at least 10% after fluid administration (usually 500cc of crystalloids) as quickly as possible (usually over 10 minutes).
- Only patients who are fluid responsive should receive additional fluids.
- Patients who have decreased systolic or diastolic function (on the descending limb of the Frank Starling curve) will not respond to a fluid challenge, even if they are intravascularly depleted.

2. Clinical signs, the chest radiograph, the CVP, and ultrasonography cannot be used to determine fluid responsiveness

- Clinical signs can indicate tissue hypoperfusion, but not fluid responsiveness.
- Traditional indicators, such as CVP monitoring and mean arterial pressures (MAP), are not accurate enough.
- Ultrasound is a potential new tool that can be used to assess volume status and fluid responsiveness.
- For volume status, there have been studies looking at IVC caliber in ventilated patients, but this has yet to be validated in spontaneously breathing patients. Lee presented an evidence-informed algorithm based on the available literature to guide fluid resuscitation decisions (Lee 2016).
- For fluid responsiveness, transthoracic measurements of LV outflow tract velocities for the estimation of stroke volume require considerable expertise and not easily or rapidly obtainable.
- Marik suggests alternative methods, such as pulse pressure change measured by radial a-line, LVOT velocity time integral (VTI) on echo, carotid Doppler flow on transesophageal echo, pulse contour analysis, thoracic bioimpedance, b

Ten situations where inferior vena cava ultrasound may fail to accurately predict fluid responsiveness: a physiologically based point of view

G. Via[1*], G. Tavazzi[1,2,3] and S. Price[3]



3. The PLR Maneuver or a fluid challenge coupled with real-time SV monitoring is the only accurate method to date for determining fluid responsiveness

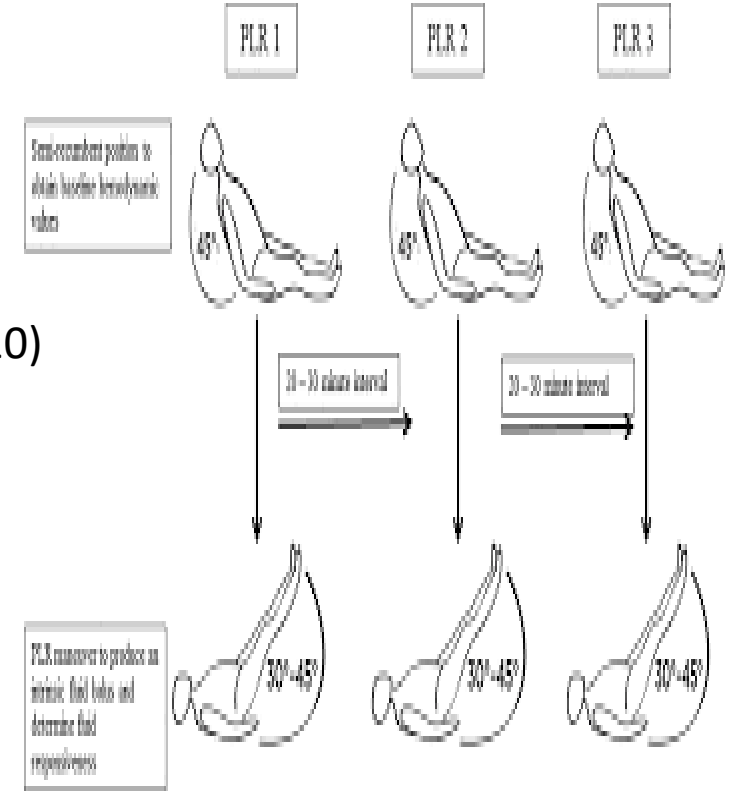
- Passive leg raise (PLR) and fluid challenge are two ways to determine fluid responsiveness, especially when used with real-time cardiac output monitors.
- PLR causes a shift in venous blood from the lower extremities to the thoracic compartment; it is essentially an auto-fluid bolus.

• Advantages of PLR:

- Reversible
- Non-invasive
- Amount of fluid mobilized is proportional to body size
- Can be used regardless of ventilation mode and cardiac rhythm (Cavallaro 2010)

• *Disadvantages of PLR:*

- Positional changes may be contraindicated
- Less useful in patients with elevated intra-abdominal pressures
- Need to stop other interventions during this maneuver



4. The hemodynamic response to a fluid challenge is usually small and short lived

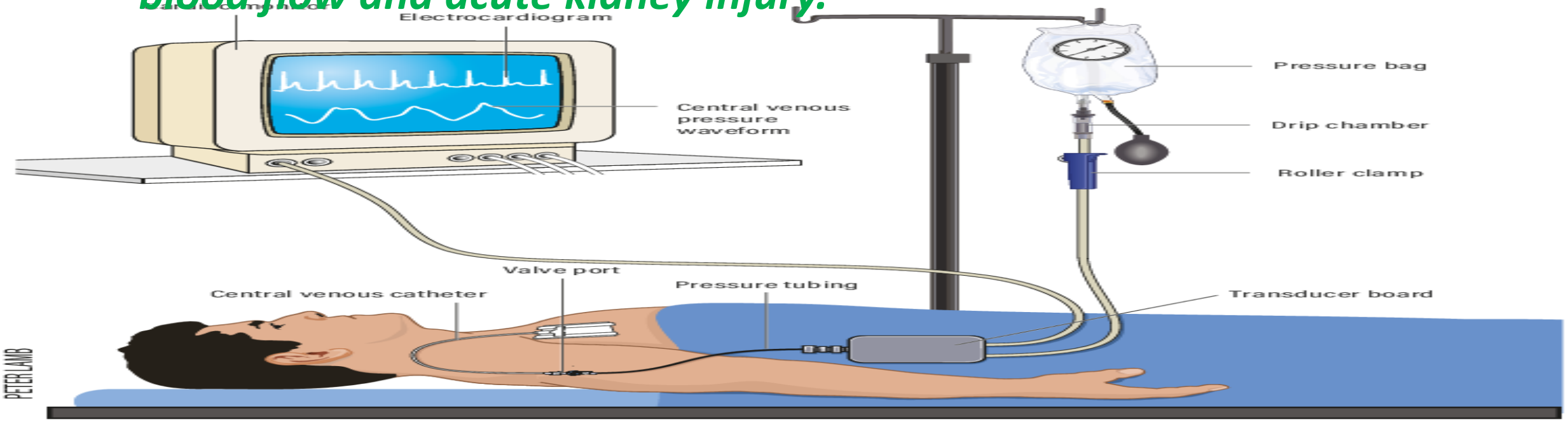
- The response to a fluid challenge is usually short lived; within 30 minutes, the cardiac index is usually back to baseline.
- The increase in MAP following a fluid challenge is minimal; fluid boluses should not be given if they do not increase stroke volume and cardiac output.

5. Fluid responsiveness does not equate to the need for fluid boluses

- Patients should only receive fluid boluses if the hemodynamic benefits are likely to outweigh the risks of becoming overloaded.
- As stated previously, the effects of fluid boluses are short-lived and minimal, and continuous fluid resuscitation in these patients will cause fluid overload.
- Instead, vasopressors and inotropes should be used early, which will increase organ perfusion while limiting tissue edema.

6. A high CVP is a major factor compromising organ perfusion

- ***An organ's blood flow is determined by the MAP and CVP. Our current guidelines recommend targeting a CVP greater than 8mm Hg.***
- ***When the CVP is greater than 8mm Hg, certain organs, like the kidney, develop increased renal pressure leading to decreased renal blood flow and acute kidney injury.***




JUST BECAUSE YOU ARE FLUID
RESPONSIVE DOESN'T MEAN YOU
NEED FLUIDS

D3:

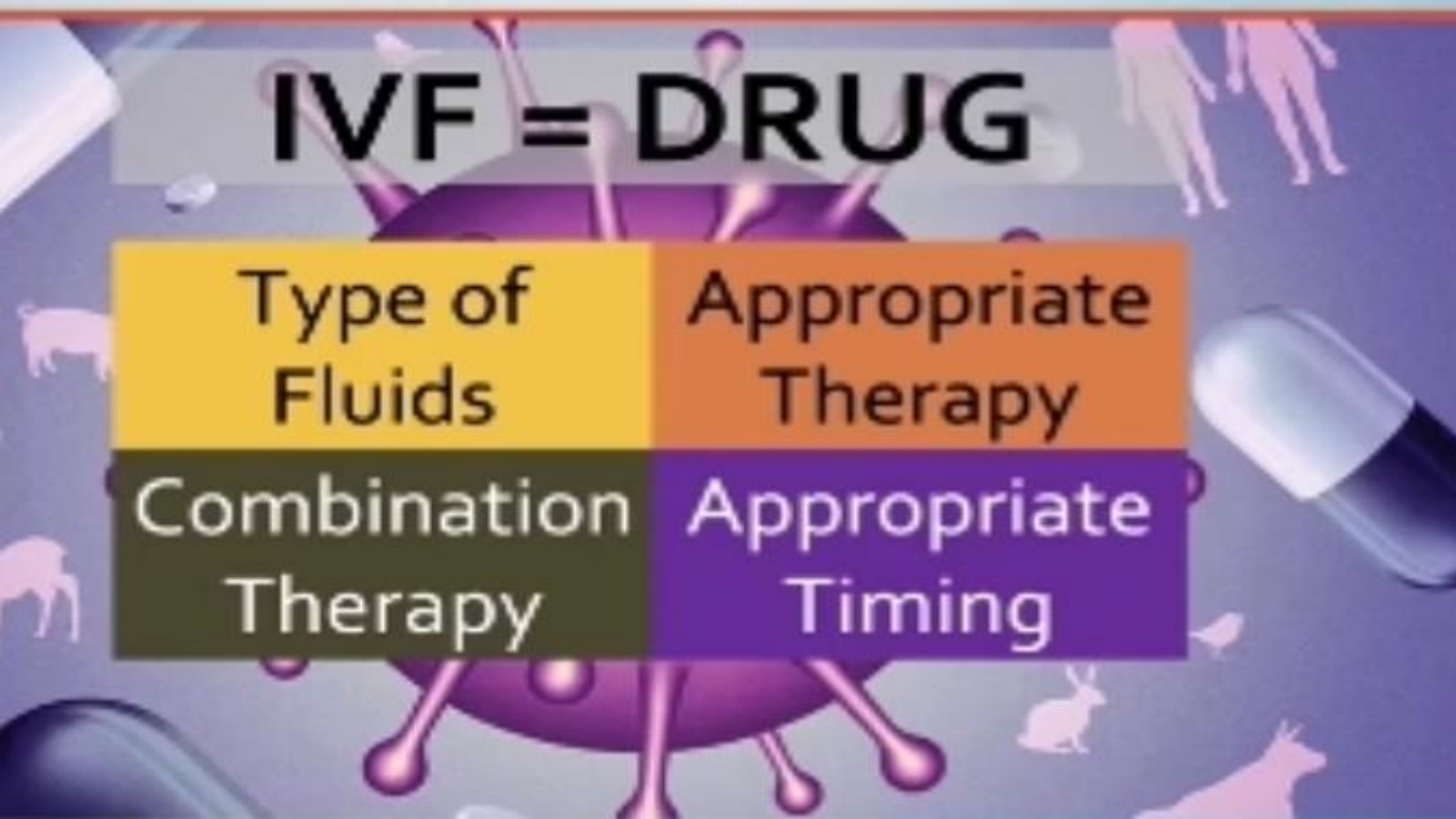
- IV Fluids are not innocent bags of water;
    - They should consider as: “DRUGS”

And

edema is akin to a drug overdose

Treating Fluids as Drugs: Four Questions About Fluid Therapy

1. When to start fluids?
2. When to stop fluids?
3. When to start fluid removal?
4. When to stop fluid removal?


IVF = DRUG
Type of Fluids
Appropriate Therapy
Combination Therapy
Appropriate Timing

Solution	Osmolarity	pH	Na^+	Cl^-	K^+	Ca^{2+}	Glucose	Other
NS	308	6.0	154	154	-	-	-	-
½ NS	154	6.0	77	77	-	-	-	-
3% NS	1026	5.0	513	513	-	-	-	-
LR	273	6.5	130	109	4	3	-	lactate 28
Plasmalyte	294	7.4	140	98	5	3	-	acetate 27 gluconate 23
D_5W	253	4.5	-	-	-	-	50	-
D_5W ½ NS	432	4.0	77	77	-	-	50	-
D_5W LR	525	5.0	130	109	4	3	50	lactate 28
7.5% $NaHCO_3$	1786	8.0	893	-	-	-	-	HCO_3 893
Albumin 5%	330	7.4	~145	-	≤2	-	-	albumin 50
Albumin 25%	330	7.4	~145	-	≤2	-	-	albumin 250
10% Dextran 40 in NS	308	4.0	154	154	-	-	-	dextran 100
Hetastarch 6% in NS	308	5.9	154	154	-	-	-	hetastarch 60

Hypotonic

Isotonic

Hypertonic

Osmolarity = mOsm/L

Electrolytes = mEq/L

Glucose, albumin = g/L

*Though sometimes used interchangeably, molarity and molality are not the same. The units for molarity are moles/liters versus the units for osmolality are moles/kilogram.

(Ab)Normal saline

Figure 5. How much sodium is in...

a) <u>1 L of normal saline</u>

0.9% sodium chloride contains 154 meq of sodium per liter.

1 L normal saline x (154 meq/L) x (23 mg Na^+/meq) = **3,542 mg Na^+**

A 1 oz. bag of Lay's® Classic potato chips contains 180 mg of sodium. Thus, 1 L of normal saline has about as much sodium as 20 bags of chips.

b) <u>1 kg of edema fluid</u>

If the serum [Na^+] is 140 meq/L, and 1 meq Na^+ = 23 mg Na^+, then...

1 L edema fluid x (140 meq/L) x (23 mg Na^+/meq) = **3,220 mg Na^+**

A single Oscar Mayer™ wiener contains 461 mg of sodium. Thus, 1 kg (or 1 L) of edema fluid has as much sodium as 7 hot dogs.

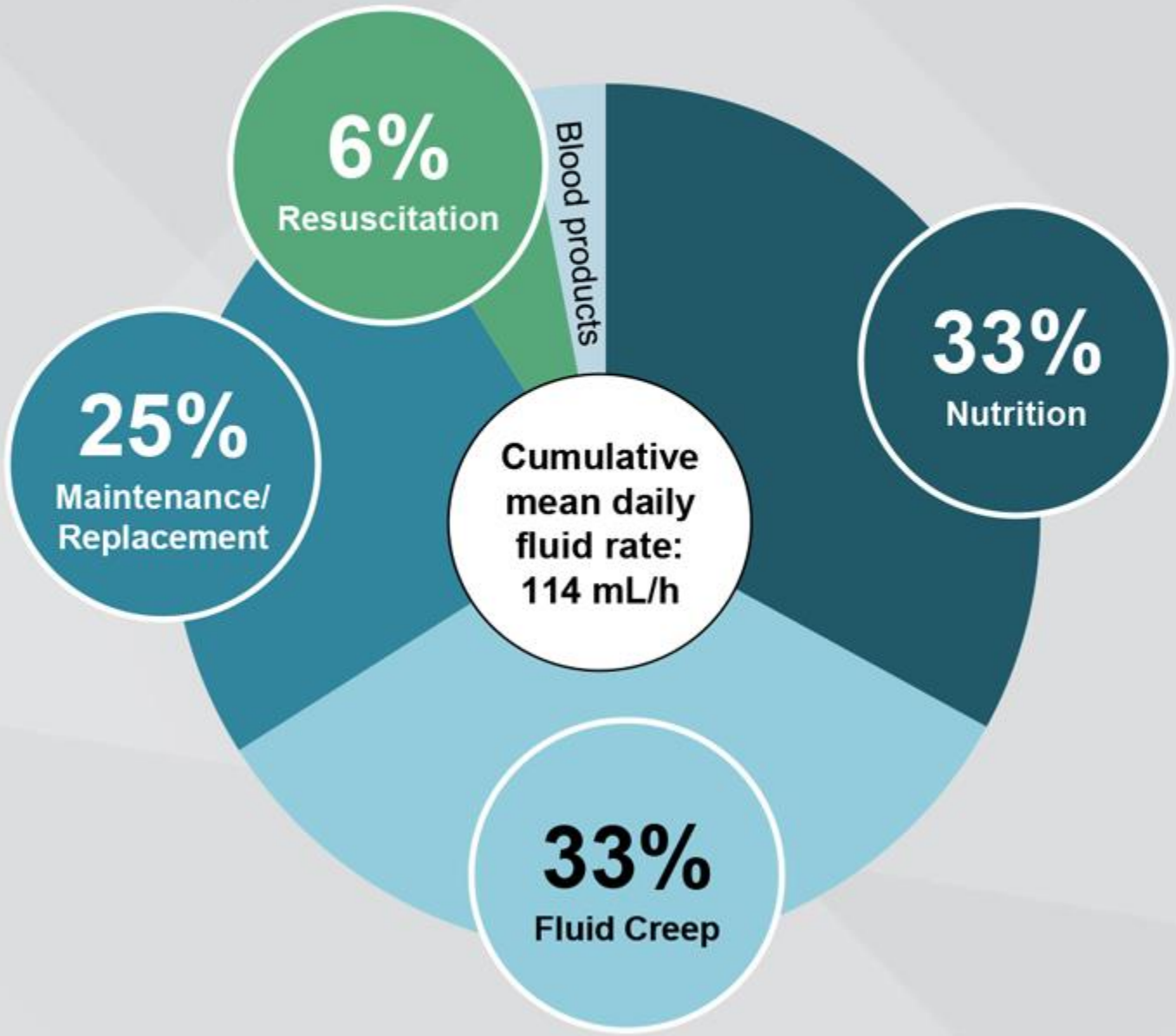
Indications for fluid administration

- 1.resuscitation
- 2.Maintenance
- 3.replacement
- 4.nutrition
- or a combination

Types of fluid therapy

- **Resuscitation fluids(save life):** are used to correct an intravascular volume deficit or acute hypovolemia
- **replacement solutions:** are prescribed to correct existing or developing deficits that cannot be compensated by oral intake alone
- **maintenance solutions:** are indicated in hemodynamically stable patients that are not able/allowed to drink water in order to cover their daily requirements of water and electrolytes
- **Nutrition fluids:** cover daily caloric needs
- **fluid creep:** fluids administered as drug diluents and to guarantee catheter patency

Different Fluid Types in the ICU



Intensive Care Med
https://doi.org/10.1007/s00134-018-5147-3

ORIGINAL

Maintenance fluid therapy and fluid creep impose more significant fluid, sodium, and chloride burdens than resuscitation fluids in critically ill patients: a retrospective study in a tertiary mixed ICU population

Niels Van Regenmortel[1,2*], Walter Verbrugghe[1], Ella Roelant[3], Tim Van den Wyngaert[4,5] and Philippe G. Jorens[1,5]

Abstract

Purpose: Research on intravenous fluid therapy and its side effects, volume, sodium, and chloride overload, has focused almost exclusively on the resuscitation setting. We aimed to quantify all fluid sources in the ICU and assess fluid creep, the hidden and unintentional volume administered as a vehicle for medication or electrolytes.

Methods: We precisely recorded the volume, sodium, and chloride burdens imposed by every fluid source administered to 14,654 patients during the cumulative 103,098 days they resided in our 45-bed tertiary ICU and simulated the impact of important strategic fluid choices on patients' chloride burdens. In septic patients, we assessed the impact of the different fluid sources on cumulative fluid balance, an established marker of morbidity.

Results: Maintenance and replacement fluids accounted for 24.7% of the mean daily total fluid volume, thereby far exceeding resuscitation fluids (6.5%) and were the most important sources of sodium and chloride. Fluid creep represented a striking 32.6% of the mean daily total fluid volume [median 645 mL (IQR 308–1039 mL)]. Chloride levels can be more effectively reduced by adopting a hypotonic maintenance strategy [a daily difference in chloride burden of 30.8 mmol (95% CI 30.5–31.1)] than a balanced resuscitation strategy [daily difference 3.0 mmol (95% CI 2.9–3.1)]. In septic patients, non-resuscitation fluids had a larger absolute impact on cumulative fluid balance than did resuscitation fluids.

Conclusions: Inadvertent daily volume, sodium, and chloride loading should be avoided when prescribing maintenance fluids in view of the vast amounts of fluid creep. This is especially important when adopting an isotonic maintenance strategy.

Keywords: Fluid therapy, Maintenance fluids, Fluid overload, Fluid creep, Chloride, Sodium, Hyperchloremia

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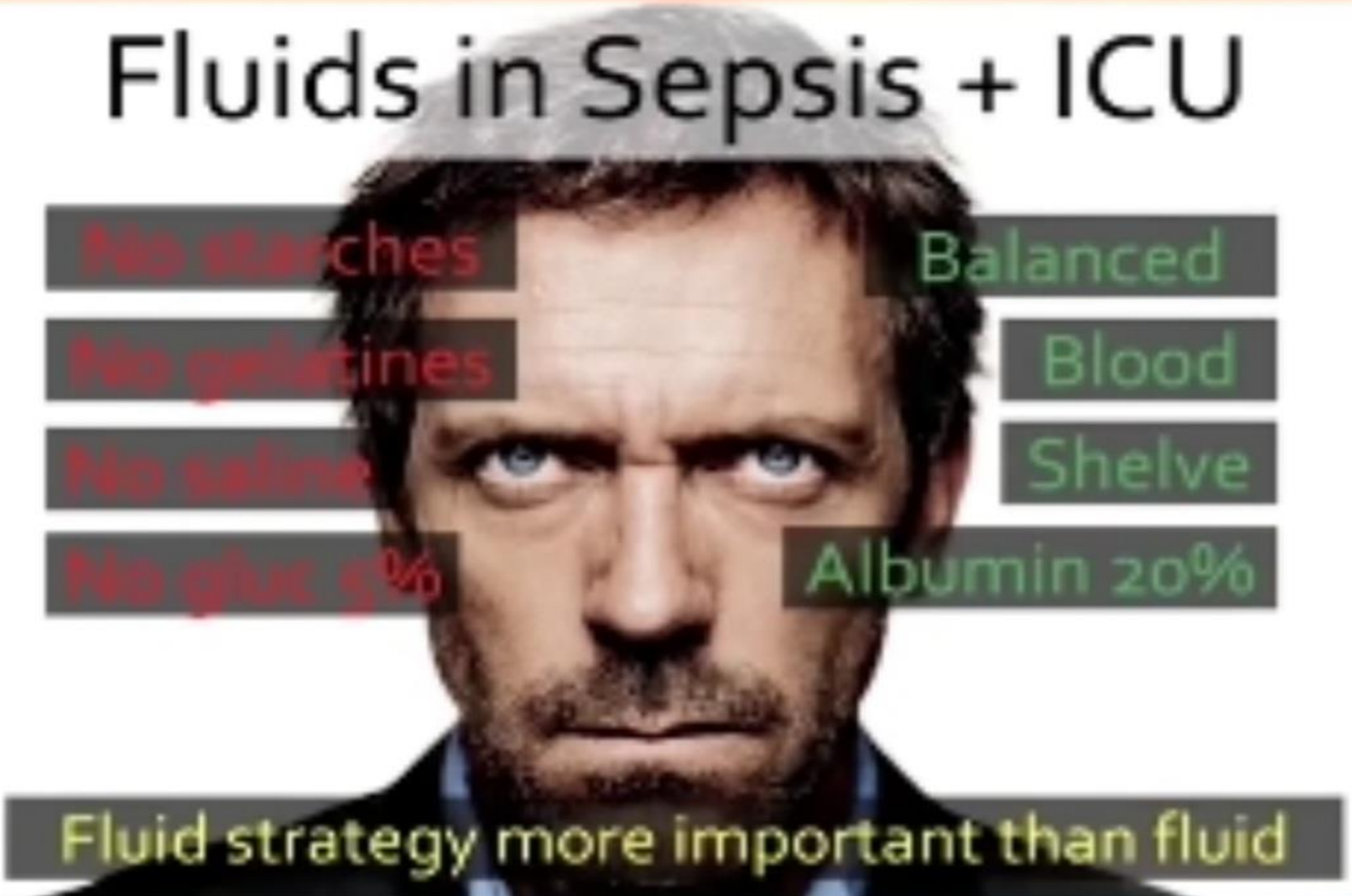
Springer

Choosing the best type of fluid

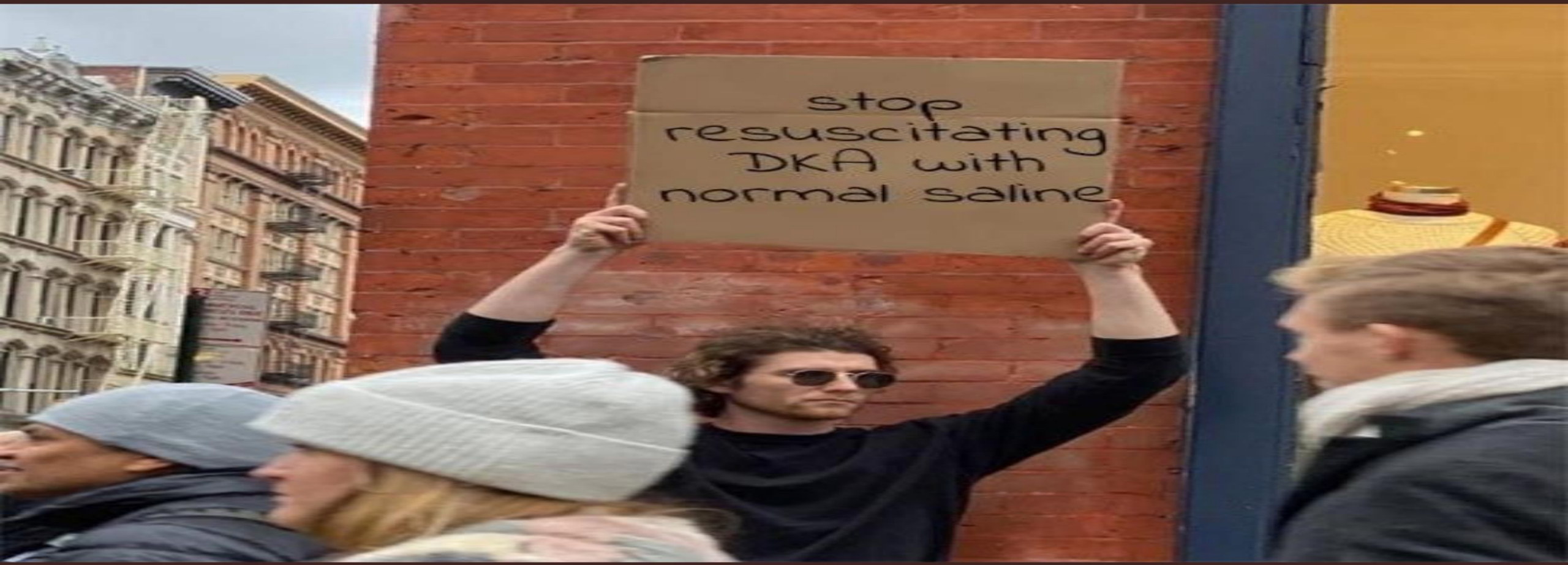
Drug

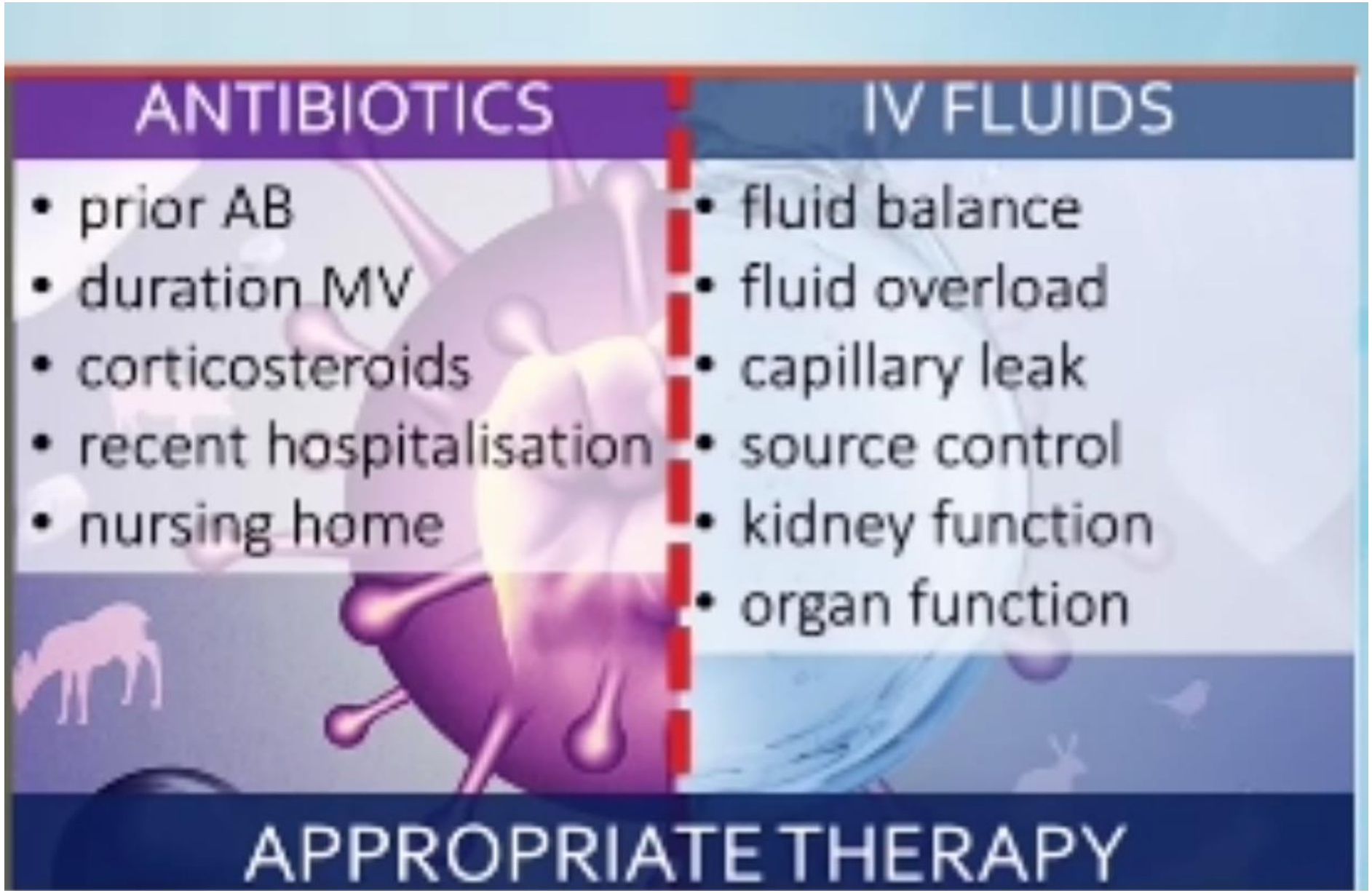
- Crystalloids versus colloids
- synthetic versus blood derived
- Balanced versus unbalanced
- intravenous versus oral
- osmolality, tonicity, pH, electrolyte composition (chloride,
- sodium, potassium, etc.) and levels of other metabolically
- active compounds (lactate, acetate, malate, etc.) are all equally important

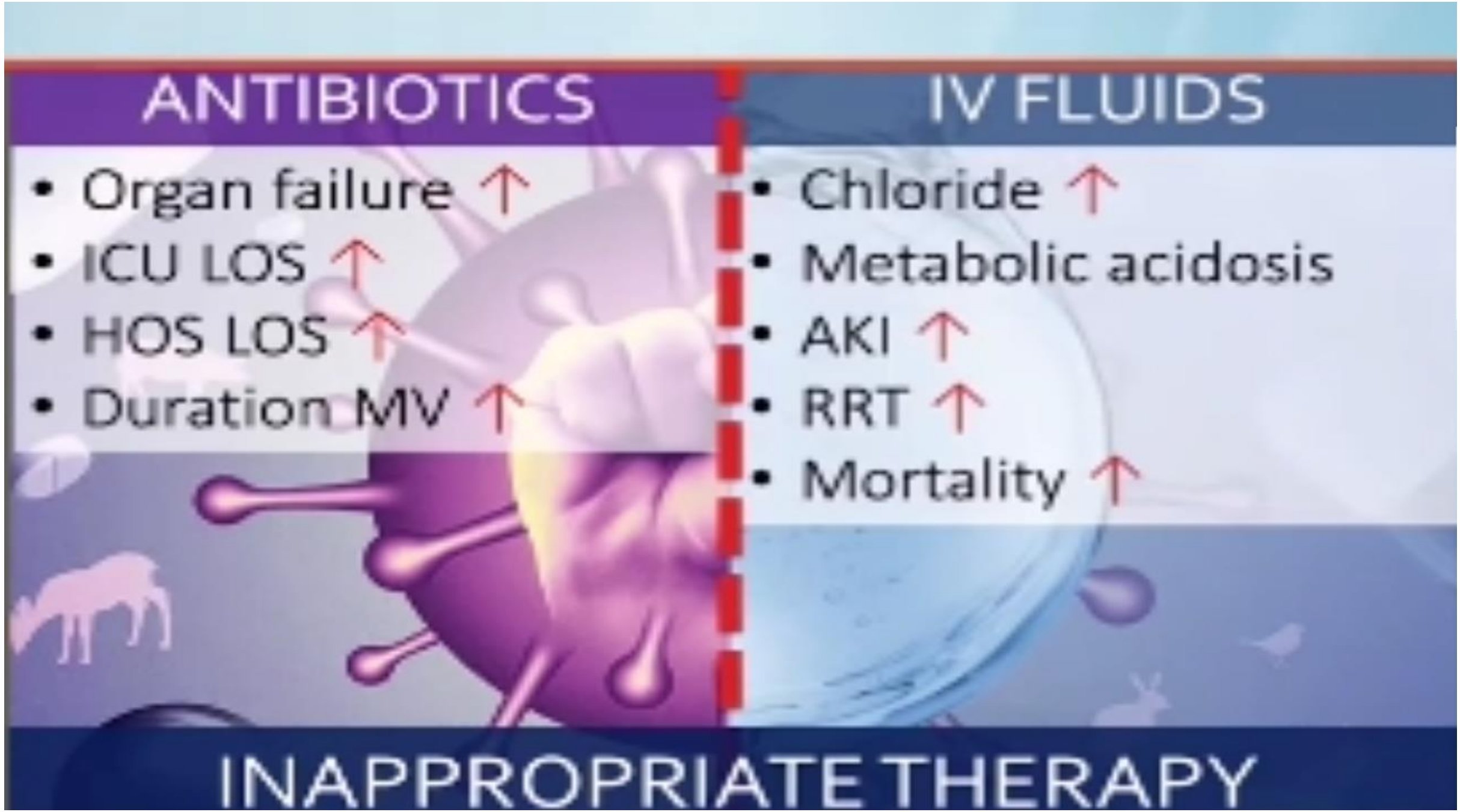
Fluids in Sepsis + ICU

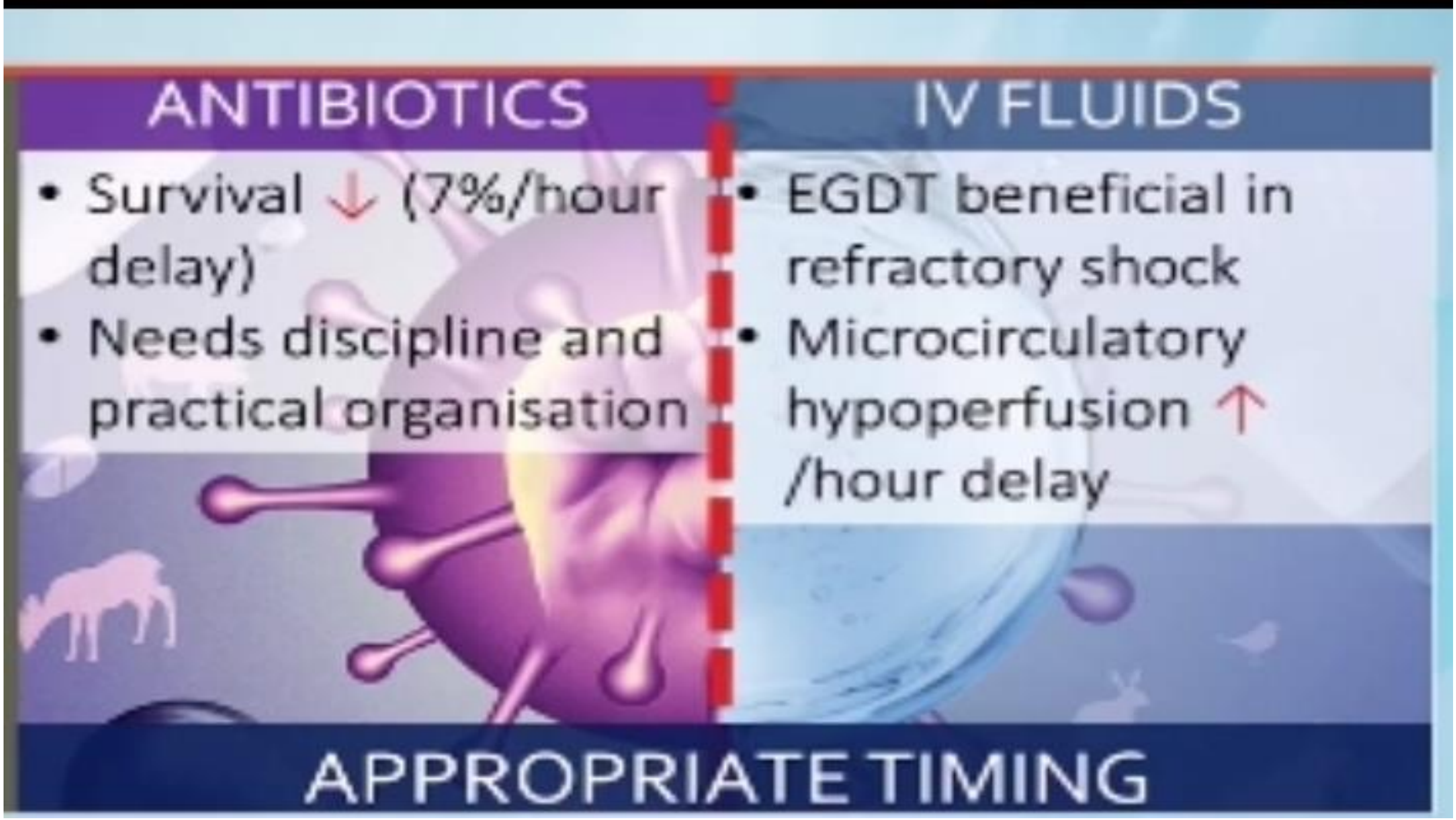


Fluids in Surgery + Trauma


stop
resuscitating
DKA with
normal saline


ANTIBIOTICS
• prior AB
• duration MV
• corticosteroids
• recent hospitalisation
• nursing home
IV FLUIDS
• fluid balance
• fluid overload
• capillary leak
• source control
• kidney function
• organ function
APPROPRIATE THERAPY


ANTIBIOTICS
• Organ failure ↑
• ICU LOS ↑
• HOS LOS ↑
• Duration MV ↑
IV FLUIDS
• Chloride ↑
• Metabolic acidosis
• AKI ↑
• RRT ↑
• Mortality ↑
INAPPROPRIATE THERAPY


ANTIBIOTICS
IV FLUIDS
• Survival ↓ (7%/hour delay)
• Needs discipline and practical organisation
• EGDT beneficial in refractory shock
• Microcirculatory hypoperfusion ↑ /hour delay
APPROPRIATE TIMING

D4:Dose

"All things are **poisons**,

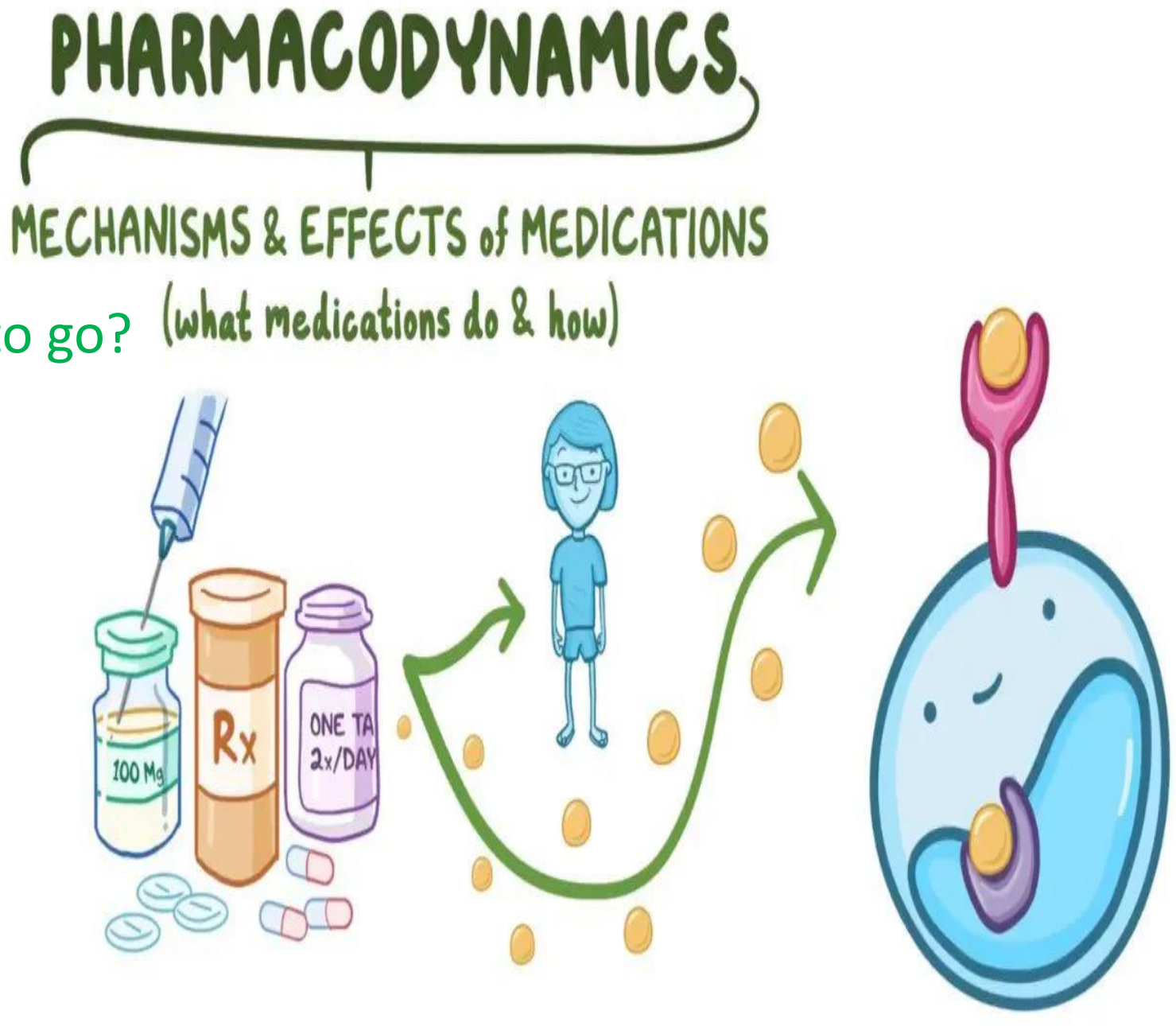
for there is nothing without poisonous qualities.

It is only the ***dose*** which makes a thing poison."

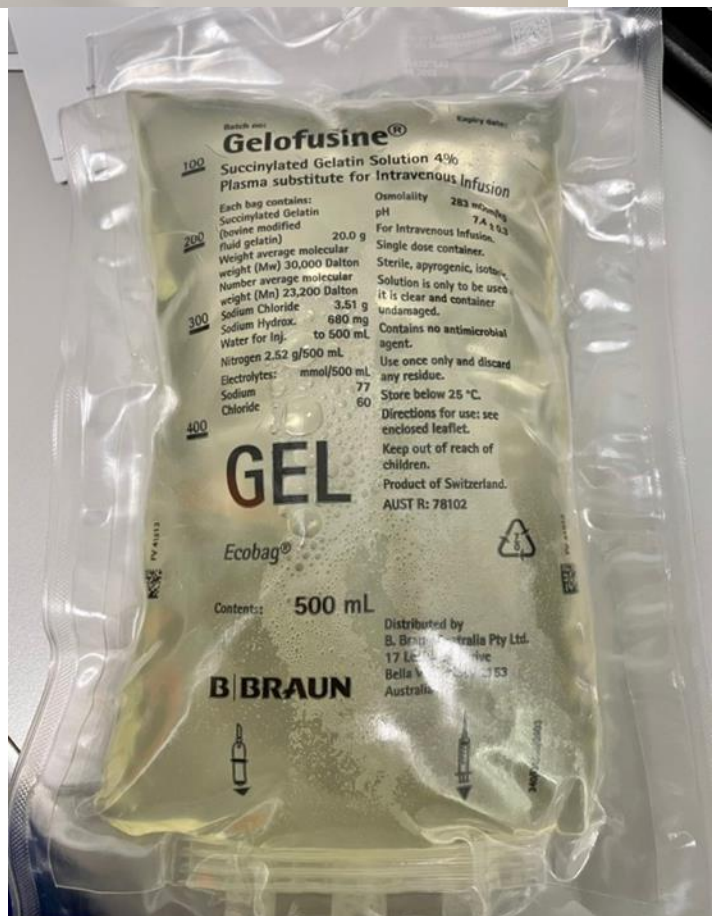
pharmacokinetic

- distribution and/or elimination and excretion will be ***slowed*** in case of :
- shock
- hypotension(20%)
- sedation
- or general Anesthesia

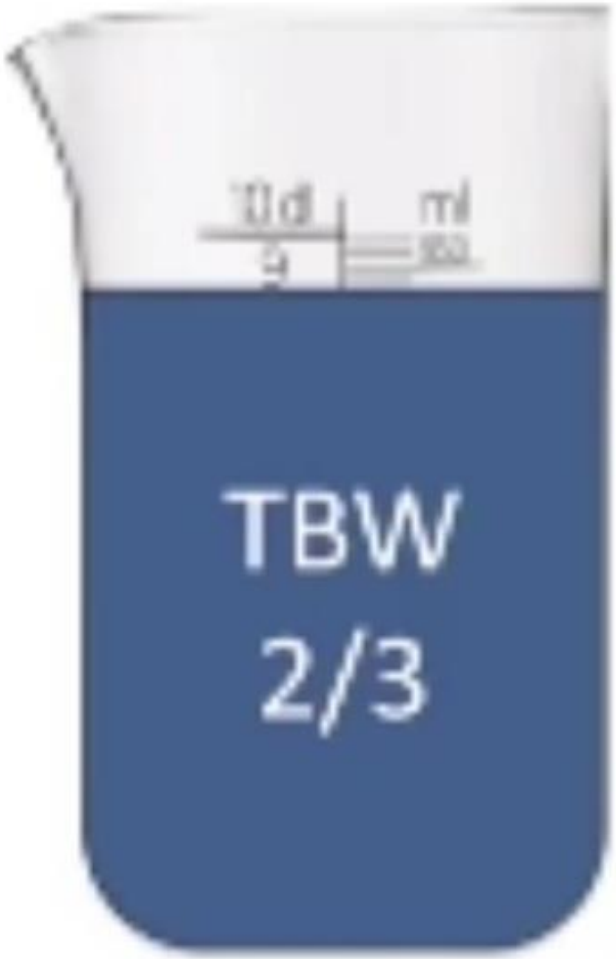
- Type of fluids
- Where you want to fluid to go?
- IV(resuscitation)
- IS
- IC(cellular dehydration)

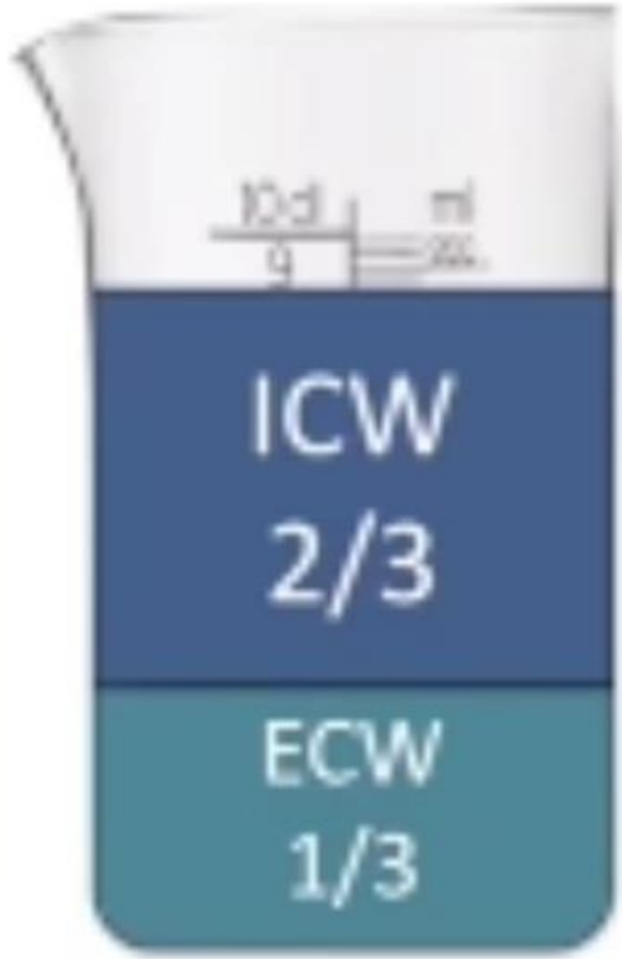


Clinical factors (underlying conditions, kidney or liver failure, presence of capillary leak, acid–base equilibrium, albumin levels, fluid balance,
etc.) must all be taken into account when choosing
the type and amount of fluid for a given patient at a given time.



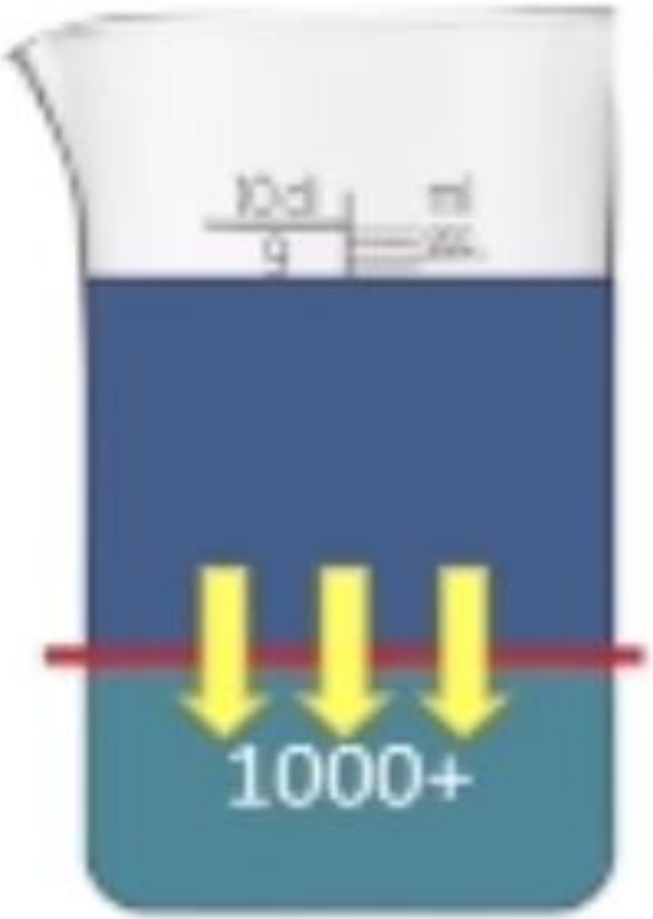
TBW = ECW + ICW





1L glucose 5% – hypotonic

1L saline (3%) – hypertonic



1L colloid – isotonic ?

If everyone is thinking alike, then no one is thinking.

~ Benjamin Franklin

Lets consider some examples:

- 1. a 7 yr/o boy presented with lethargy,fever and found hypotensive in ER,he was hydrated with …. Over ….
- 2. an 11 yr/o following acute GI bleeding presented with ↓LOC , and found hypotensive in ER,he was hydrated with …. Over ….
- 3. a 2 yr/o boy,a case of HLH,presented with edema,oliguria ,and found hypotensive in ER,he was hydrated with …. Over ….
- 4. a 16 yr/o girl with the history of COVID-19 in her parents,presented with tachycardia, and found hypotensive,tachyarrythmia in ER,he was hydrated with …. Over ….
- 5. a 8 months/o boy following watery diarrhea……..
- 6. ,a 9 yr/o boy,a case of CKD,presented with fever and lethargy, and found hypotensive in ER, so he was hydrated with …. Over ….

“Dose”

- Maintenance : 25 cc/Kg/day or 1 cc/Kg/hr
- Resuscitation :
- -EGDT
- -Fluid bolus

"Dose"

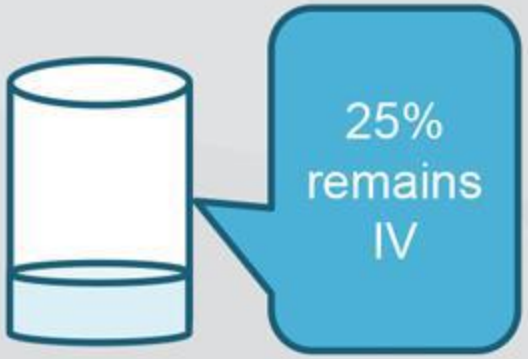
Resuscitation Fluids

Resuscitation fluids should save lives: Give them in appropriate dose and timing

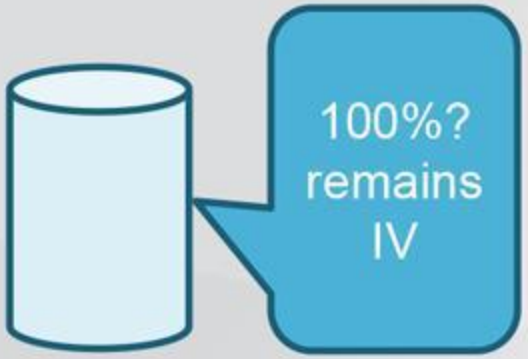
- 4 mL/kg/10 minutes and repeat
- Re-assess fluid responsiveness before and after fluid bolus administration (eg, with passive leg raising test)
- Set your targets appropriately to reach your goals
- Targets and goals are dynamic and can change during different phases

5% Glucose

Crystalloid



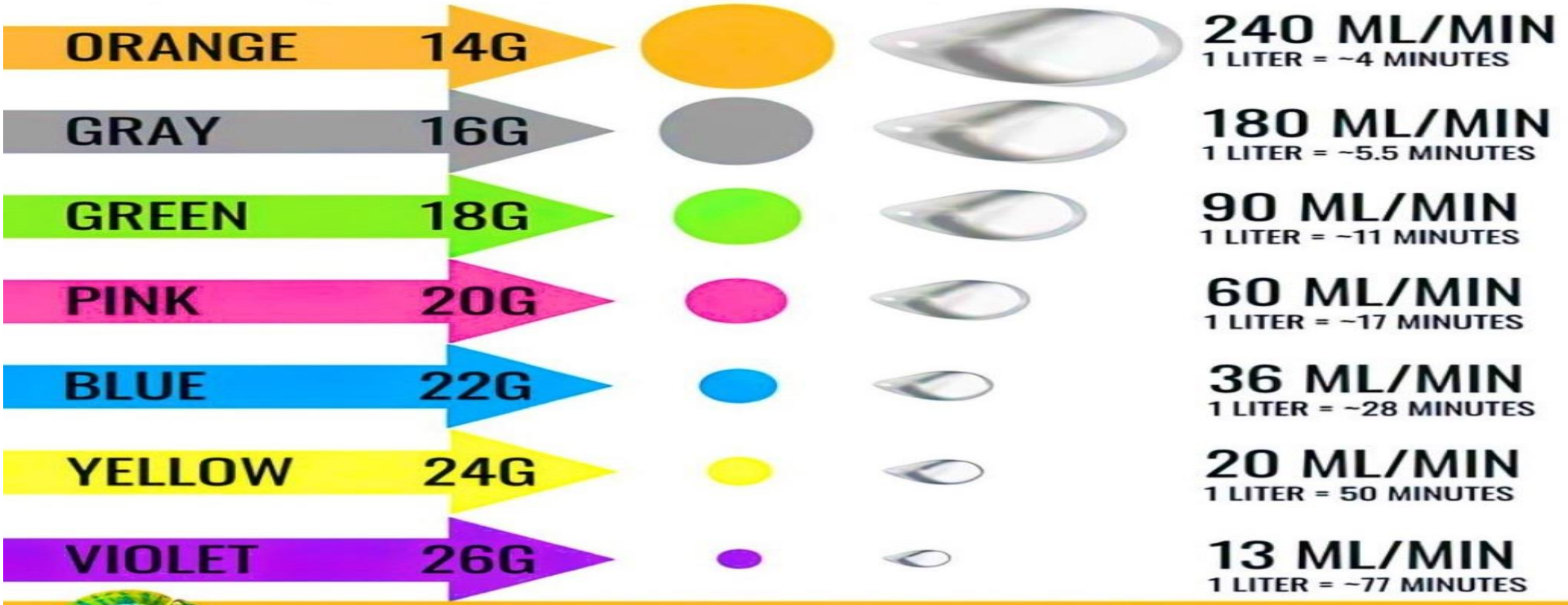
Colloid



INTERNATIONAL EMERGENCY MEDICINE EDUCATION PROJECT

Procedure Pearls

IV CATHETER SIZES AND FLOW RATES



- A fluid bolus is the rapid infusion of fluids over a short period of time. In clinical practice, a fluid bolus is usually given to correct hypovolemia, hypotension,inadequate blood flow or impaired microcirculatory perfusion. The volume of fluid bolus is heterogeneous among clinicians , typically 500–1000 mL

WHAT IS A FLUID CHALLENGE?

Give 500 mL

is NOT a ‘fluid challenge’

Give 500 mL in 30 min

is NOT a ‘fluid challenge’

Give 200 mL in 10 min

is NOT a ‘fluid challenge’

Give 200 mL in 10 min
and assess the patient’s response

is a ‘fluid challenge’

Fluid and Vasoactive-Inotrope Management Algorithm For Children

Surviving Sepsis Campaign

Healthcare Systems WITH Intensive Care

SEPTIC SHOCK

Healthcare Systems WITHOUT Intensive Care

Abnormal Perfusion with or without Hypotension

- If signs of fluid overload are absent, administer fluid bolus, 10-20 mL/kg.
- Repeat assessment of hemodynamic response to fluid and consider fluid boluses, 10-20 mL/kg, until shock resolves or signs of fluid overload develop.
- Assess cardiac function.
- Consider epinephrine if there is myocardial dysfunction or epinephrine/ norepinephrine if shock persists after 40-60 mL/ kg (or sooner if signs of fluid overload develop).

Abnormal perfusion WITHOUT hypotension

- Do NOT give fluid bolus unless there are signs of dehydration with ongoing fluid losses (eg, diarrhea).
- Start maintenance fluids.
- Monitor hemodynamics closely.
- Consider vasoactive-inotropic support (if available).

Abnormal perfusion WITH hypotension*

- If signs of fluid overload are absent, administer fluid bolus, 10-20 mL/kg.
- Assess hemodynamic response to fluid and repeat fluid boluses, 10-20 mL/kg, until hypotension resolves or signs of fluid overload develop.
- Assess cardiac function (if available)
- Consider epinephrine/ norepinephrine if hypotension persists after 40 mL/kg or sooner if signs of fluid overload develop.

Fluid in mL/kg should be dosed as ideal body weight.

Shock resolved, perfusion improved

- Do not give more fluid boluses.
- Consider maintenance fluids.
- Monitor for signs/symptoms of recurrent shock.

*Hypotension in healthcare systems WITHOUT intensive care is defined as either:

- SBP < 50 mm Hg in children aged < 12 months
- SBP < 60 mm Hg in children aged 1 to 5 years
- SBP < 70 mm Hg in children aged > 5 years

OR

Presence of all 3 World Health Organization criteria: cold extremities, prolonged capillary refill > 3 seconds, weak/fast pulse

www.sccm.org/SurvivingSepsisCampaign/Guidelines/Pediatric-Patients

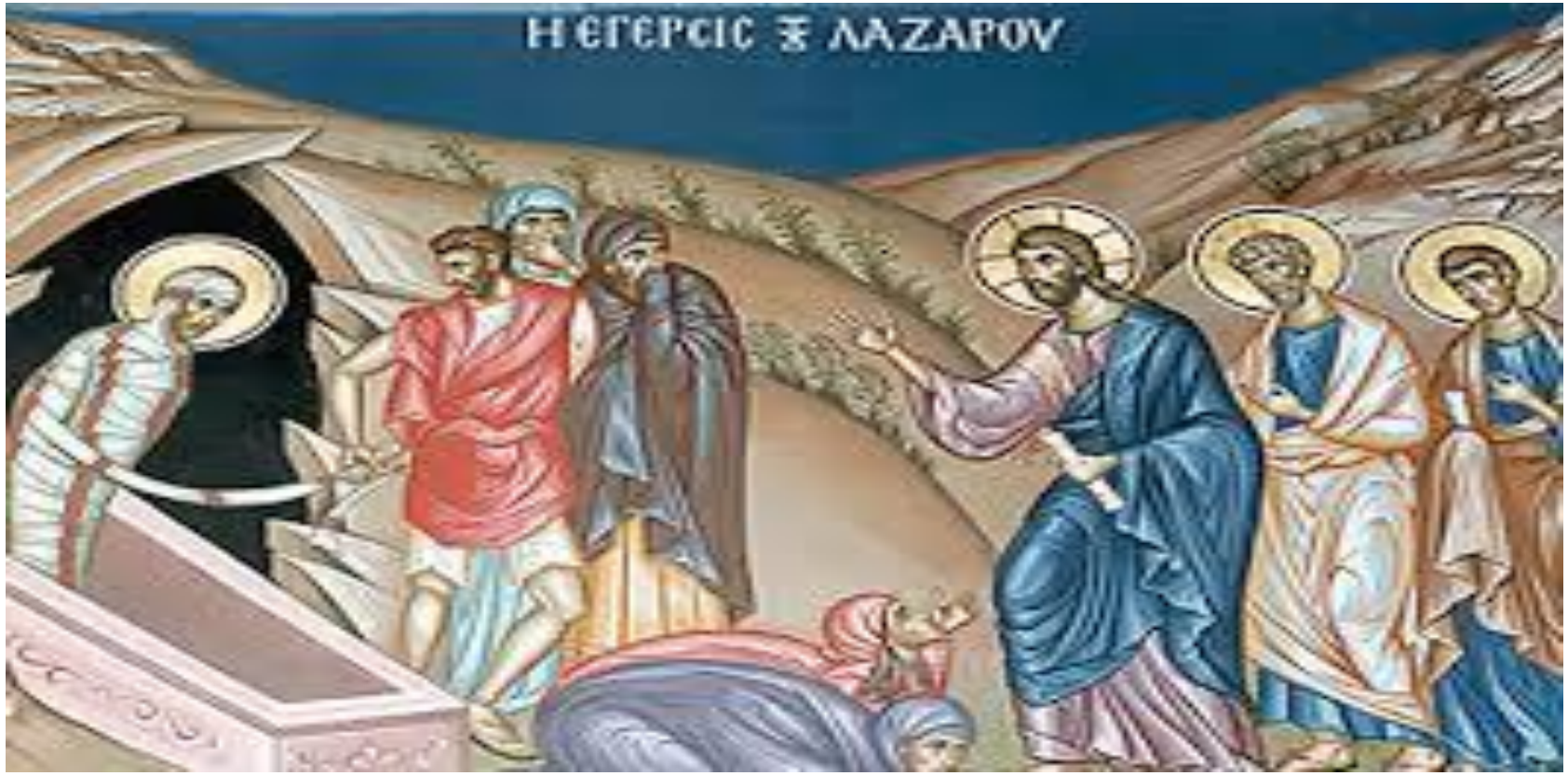
Society of Critical Care Medicine

ESICM

TABLE 2. Implications of the Strength of Recommendation

Category	Strength	Quality of Evidence	Implications to Patients	Implications to Clinicians	Implications to Policymakers
Strong recommendation	Strong	Usually high or moderate	Most individuals in this situation would want the recommended course of action, and only a small proportion would not	Most individuals should receive the recommended course of action. Formal decision aids are not likely to be needed to help individuals make decisions consistent with their values and preferences	Can be adapted as policy in most situations, including for use as performance indicators
Weak recommendation	Weak	Any	The majority of individuals in this situation would want the suggested course of action, but many would not	Different choices are likely to be appropriate for different patients, and therapy should be tailored to the individual patient's circumstances, such as patients' or family's values and preferences	Policies will likely be variable
Best practice statement	Strong	Ungraded	Same as strong recommendation	Same as strong recommendation	Same as strong recommendation
In our practice statement	Not a recommen-dation	NA	NA	NA	NA

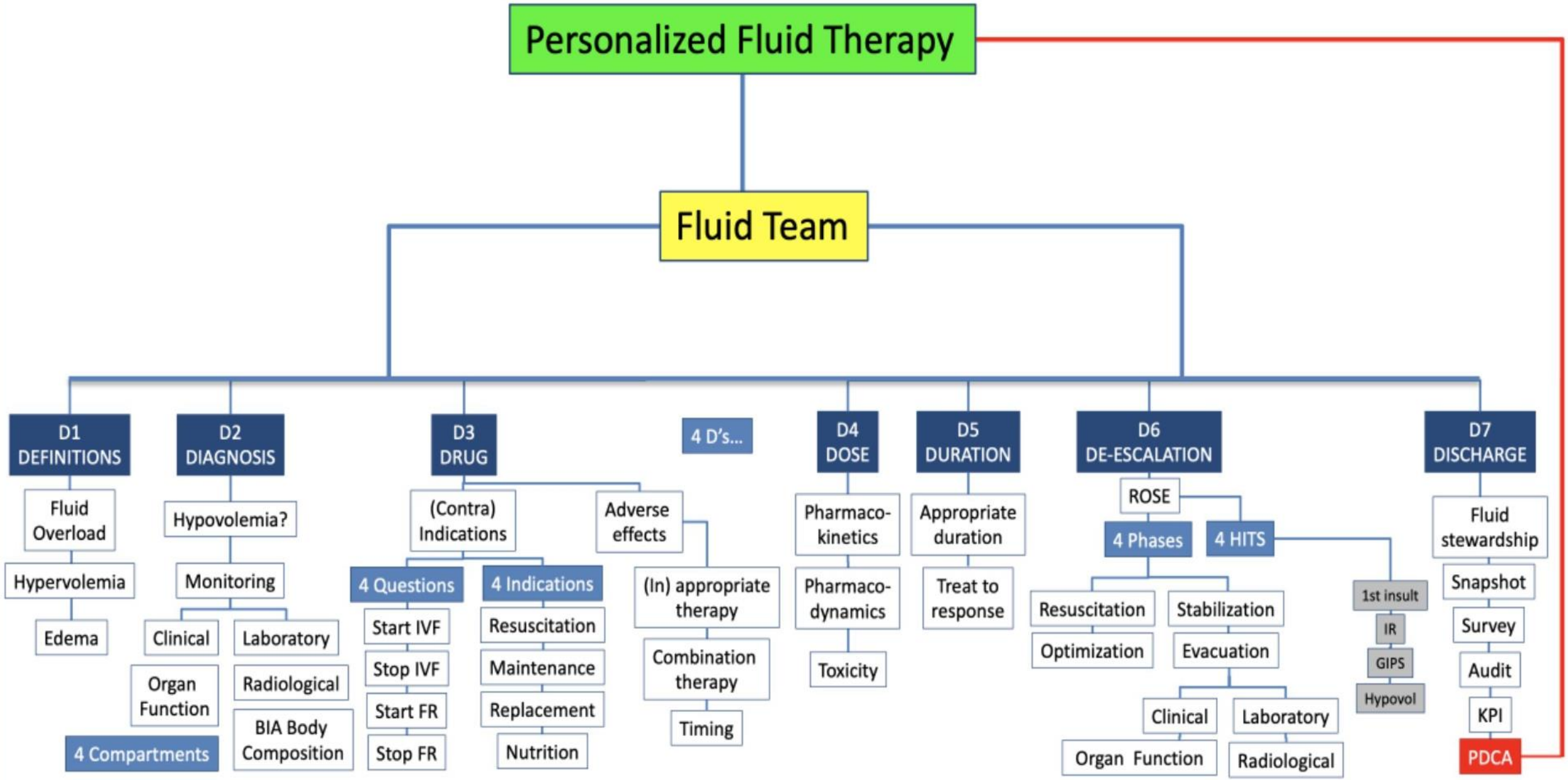
Resuscitation or resurrection



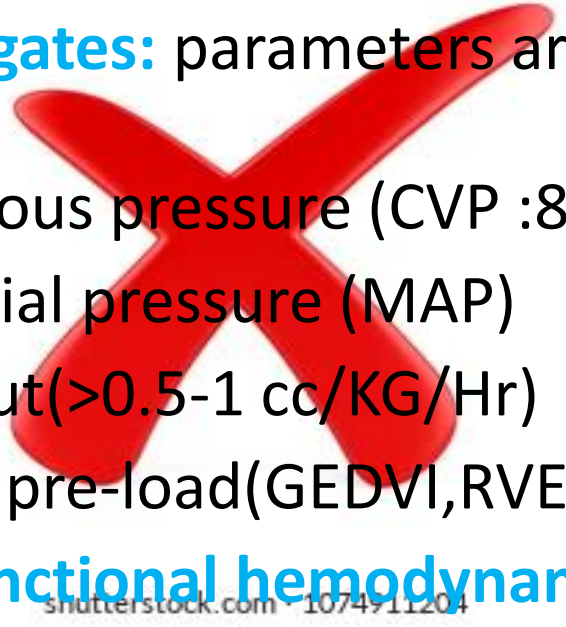
- A recent study employing a combined Bayesian and frequentist methodological approach to evaluate 12 randomized trials and 31 observational studies found that EGDT was potentially harmful in the patients with the highest disease severity
- In fact, rather than infusing a ***pre-defined*** given amount of fluid, the goal should be ***individualized*** for every patient, based on the evaluation of the need for fluids and on the patient's premorbid conditions


ONE-SIZE FITS ALL
STORE
SALE

Individualized management

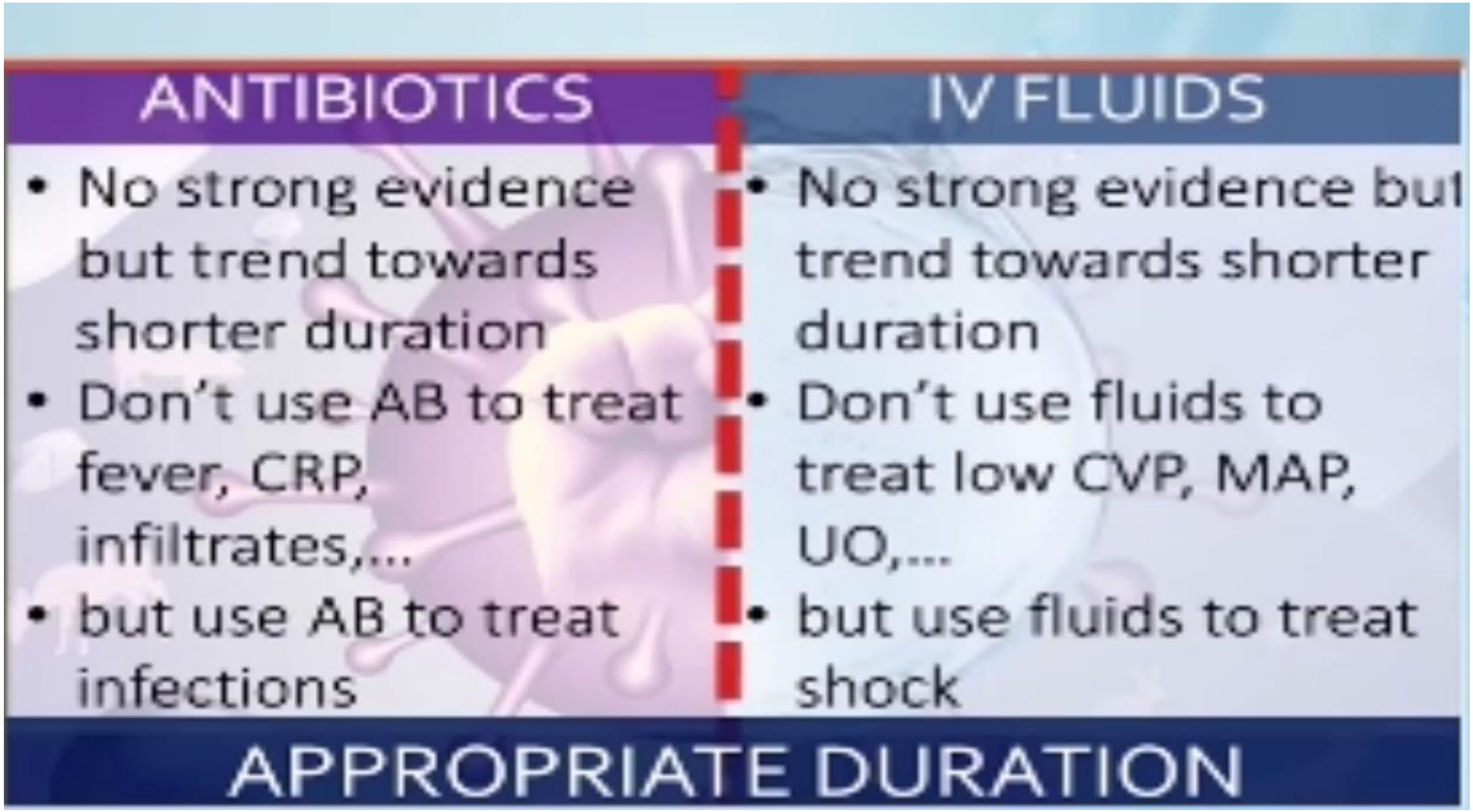

Personalized Fluid Therapy
Fluid Team
D1 DEFINITIONS
Fluid Overload
Hypervolemia
Edema
D2 DIAGNOSIS
Hypovolemia?
Monitoring
Clinical
Laboratory
Organ Function
Radiological
4 Compartments
BIA Body Composition
D3 DRUG
(Contra) Indications
4 Questions
Start IVF
Stop IVF
Start FR
Stop FR
4 Indications
Resuscitation
Maintenance
Replacement
Nutrition
Adverse effects
(In) appropriate therapy
Combination therapy
Timing
4 D's...
D4 DOSE
Pharmaco-kinetics
Pharmaco-dynamics
Toxicity
D5 DURATION
Appropriate duration
Treat to response
D6 DE-ESCALATION
ROSE
4 Phases
Resuscitation
Optimization
Stabilization
Evacuation
Clinical
Organ Function
Laboratory
Radiological
4 HITS
1st insult
IR
GIPS
Hypovol
D7 DISCHARGE
Fluid stewardship
Snapshot
Survey
Audit
KPI
PDCA

When to stop IV fluids



- **Static surrogates:** parameters are often used to titrate fluid therapy(pre-load)
- -central venous pressure (CVP :8-12 mmHg)
- -Mean arterial pressure (MAP)
- -Urine output(>0.5-1 cc/KG/Hr)
- -Volumetric pre-load(GEDVI,RVEDVI,LVEDAI)
- **Dynamic functional hemodynamic parameters**
- -PPV(or SVV)
- Dynamic tests to predict response to IVF
- -PLR or EXOT

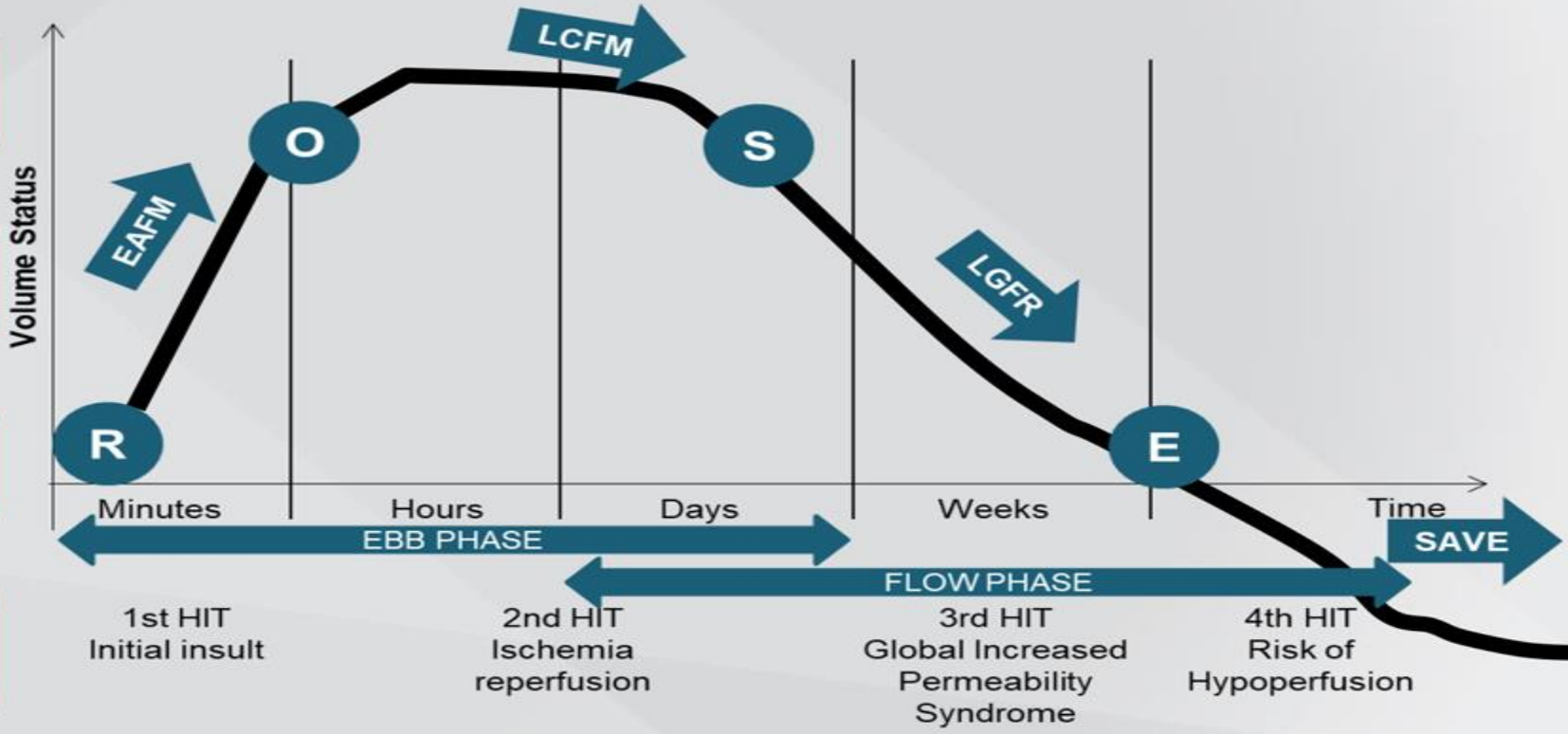
D5:


ANTIBIOTICS
IV FLUIDS
• No strong evidence but trend towards shorter duration
• Don't use AB to treat fever, CRP, infiltrates,...
• but use AB to treat infections
• No strong evidence but trend towards shorter duration
• Don't use fluids to treat low CVP, MAP, UO,...
• but use fluids to treat shock
APPROPRIATE DURATION

D6:

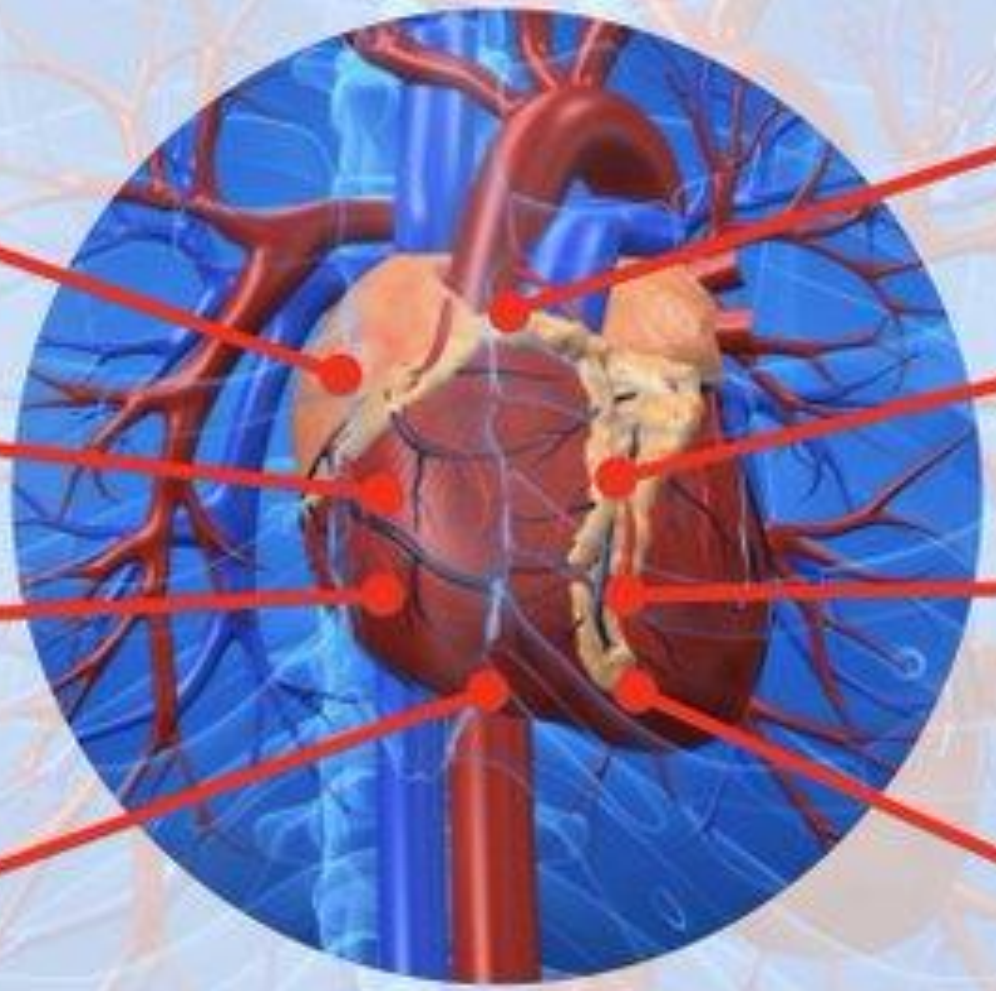
De-Escalation: The R.O.S.E. Model

- **R** Resuscitation
- **O** Optimisation
- **S** Stabilisation
- **E** Evacuation



- In this de-resuscitation phase, we try to find an answer to the third and fourth question: “When to start fluid removal?” and “When to stop fluid removal?” To answer these questions, testing ***preload***
- ***responsiveness*** may still be useful.

Hemodynamic Assessment Fluid Overload



Absence FR

Low PPV, SVV

Negative PLR

Negative EEO

MAP, HR ↑

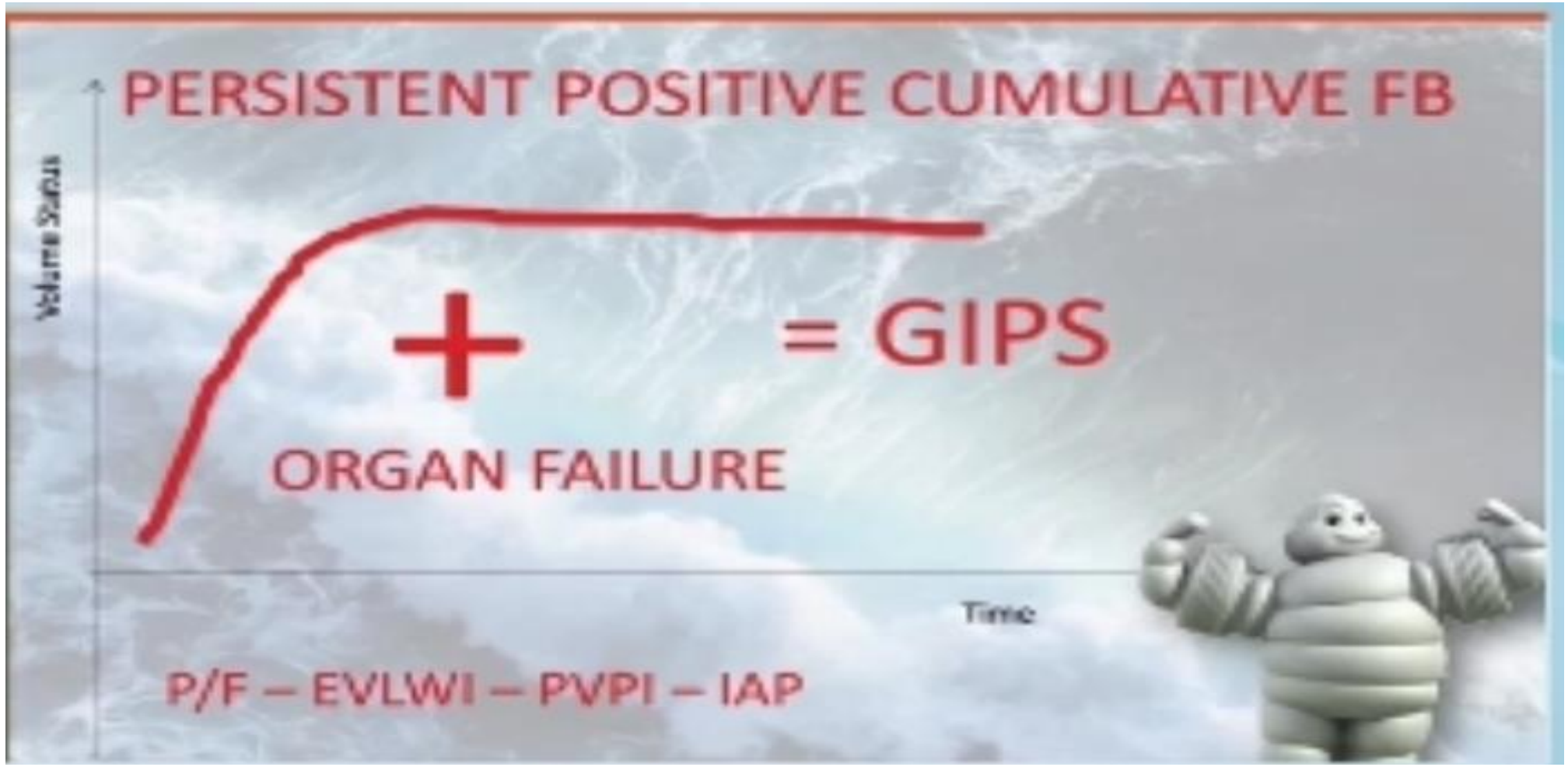
CVP ↑
PAOP ↑

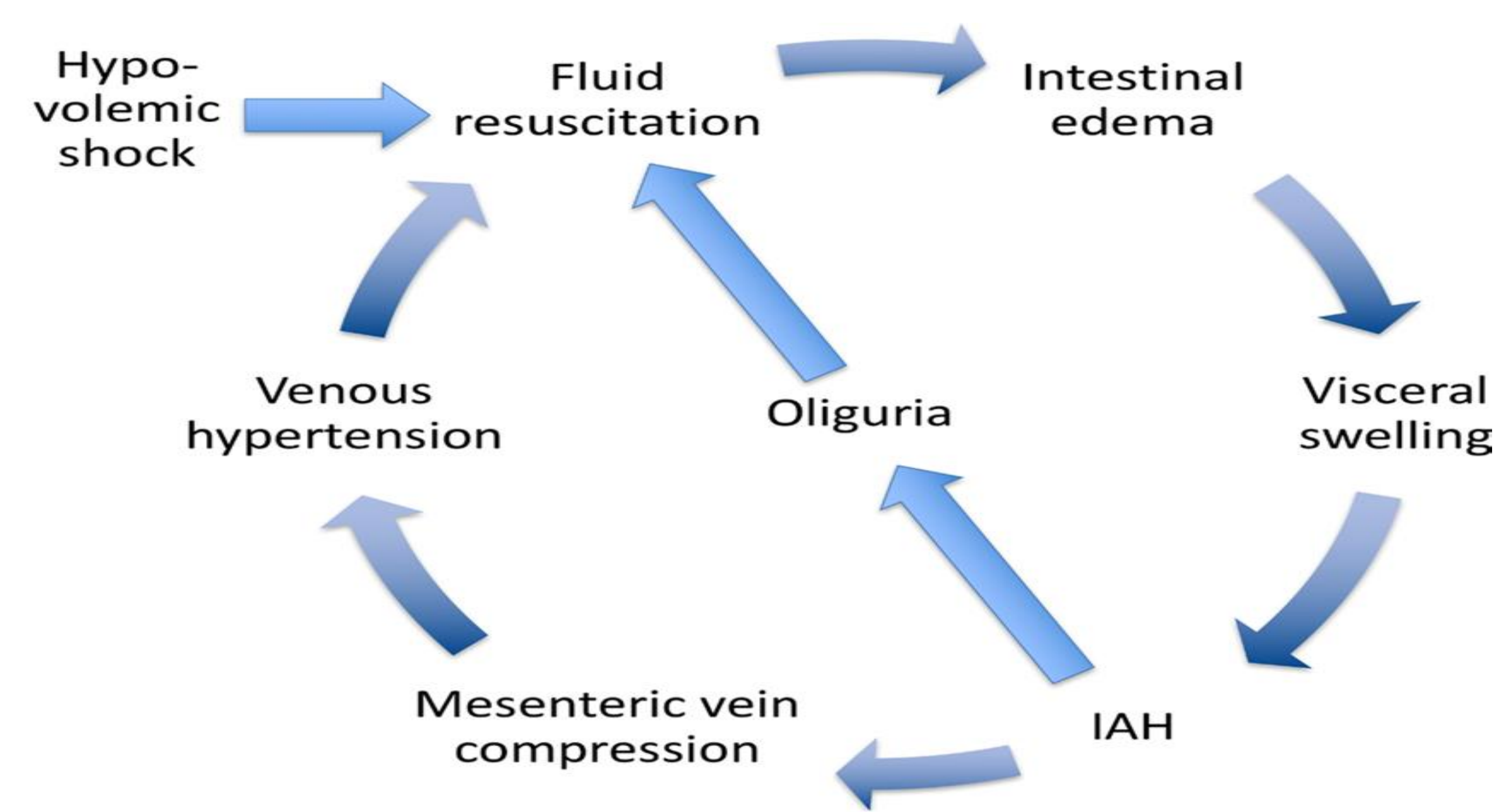
GEF/GEDVI ↑

REF/RVEDVI ↑

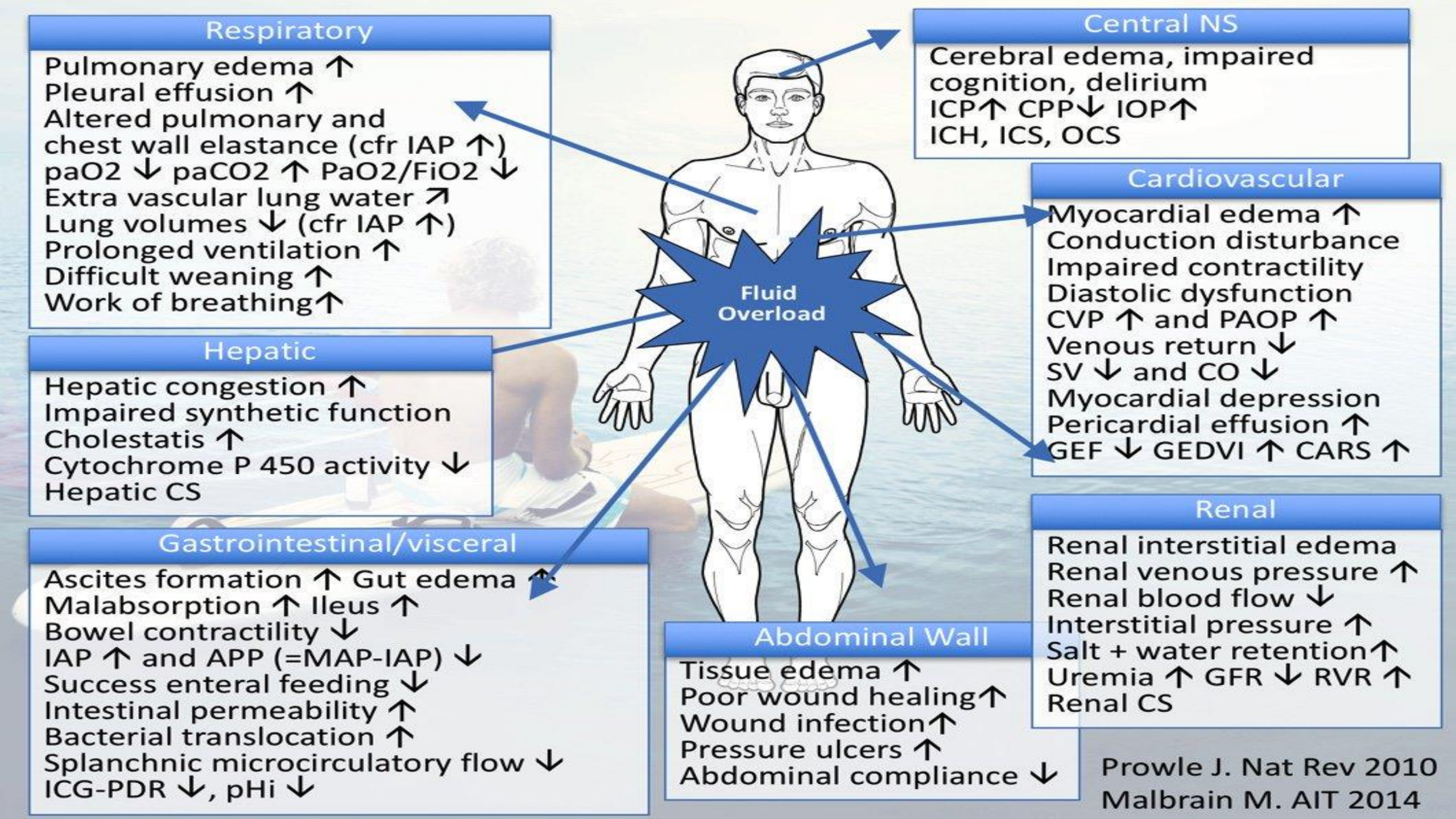
Slide by courtesy of @Manu_Malbrain @Fluid_Academy

When to start the 6th D?



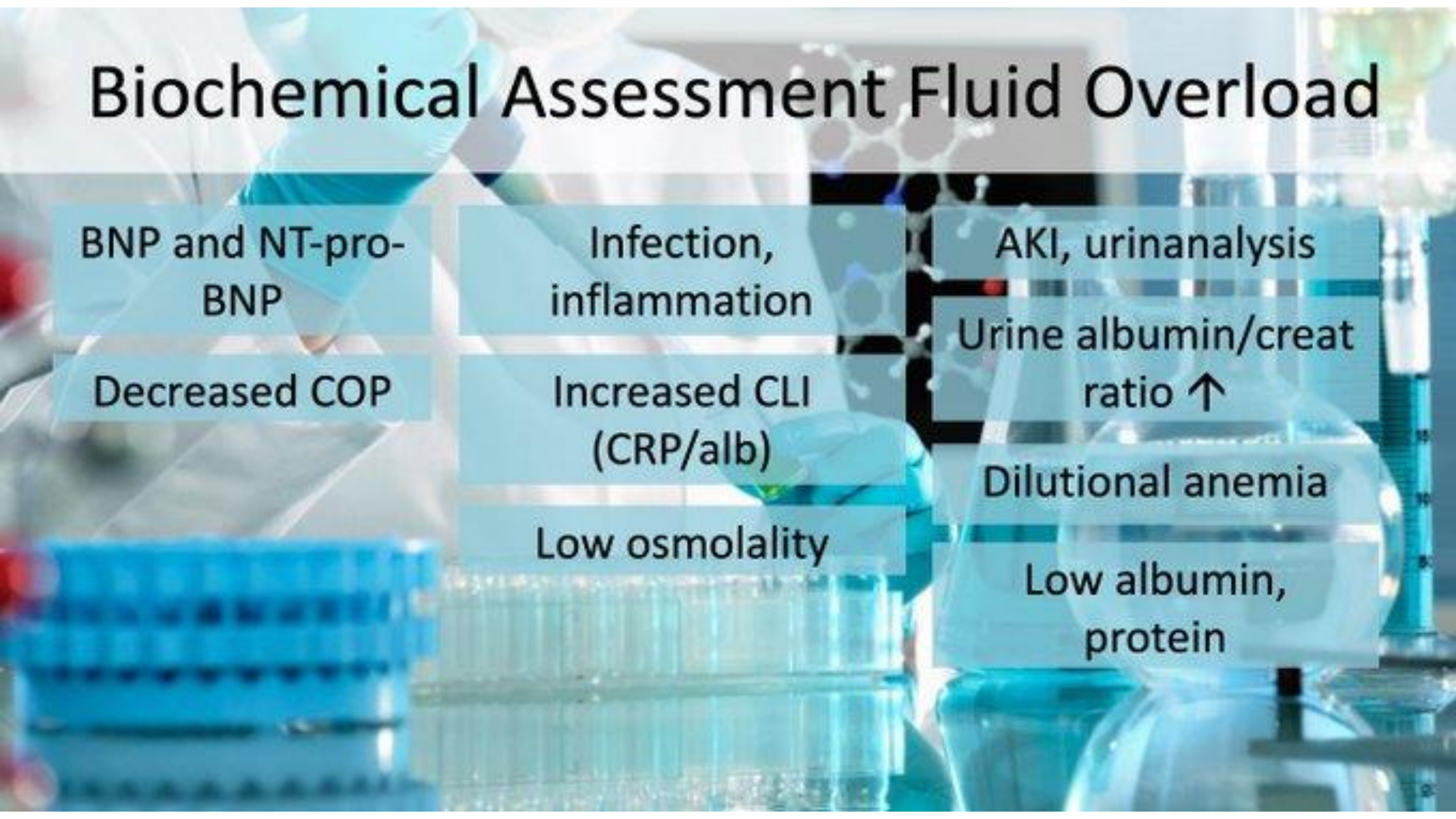


IAH: intra-abdominal hypertension

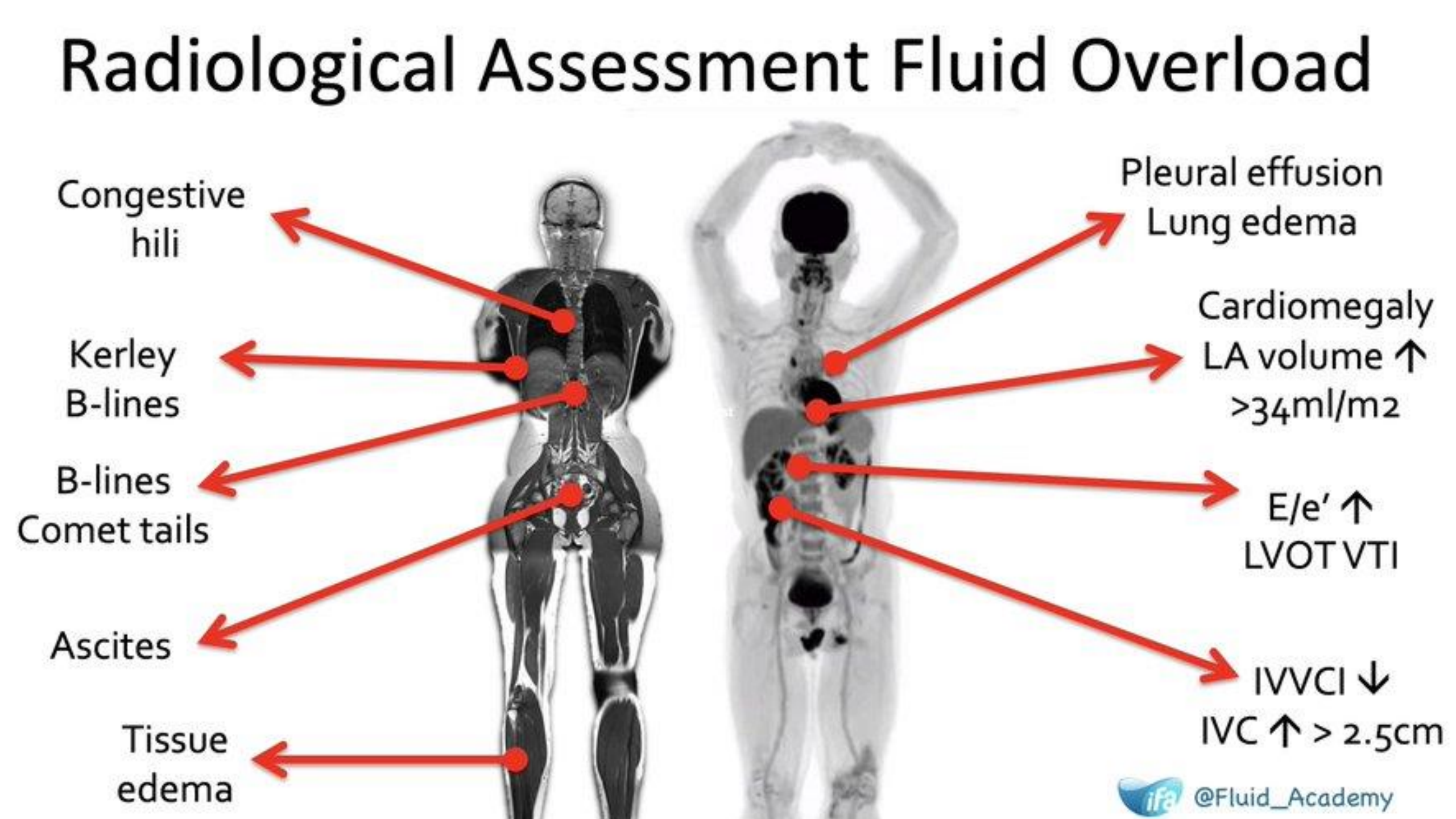


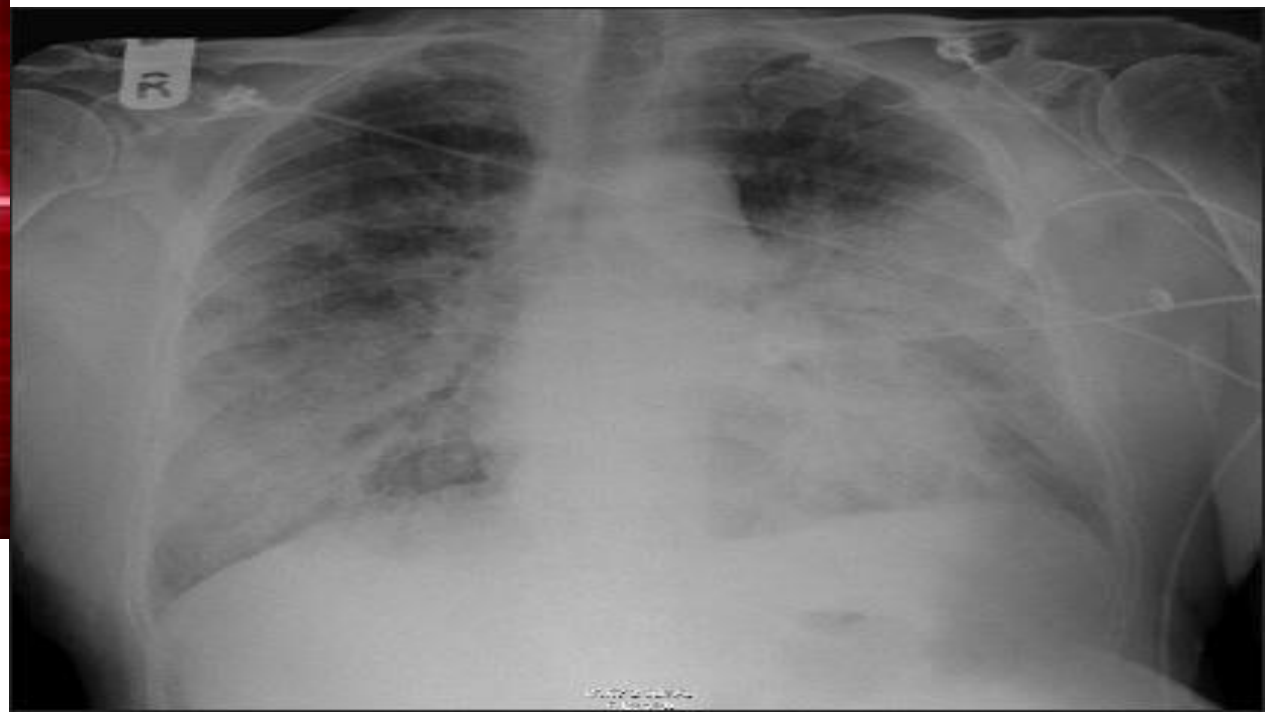
Prowle J. Nat Rev 2010
Malbrain M. AIT 2014

Biochemical Assessment Fluid Overload



Radiological Assessment Fluid Overload

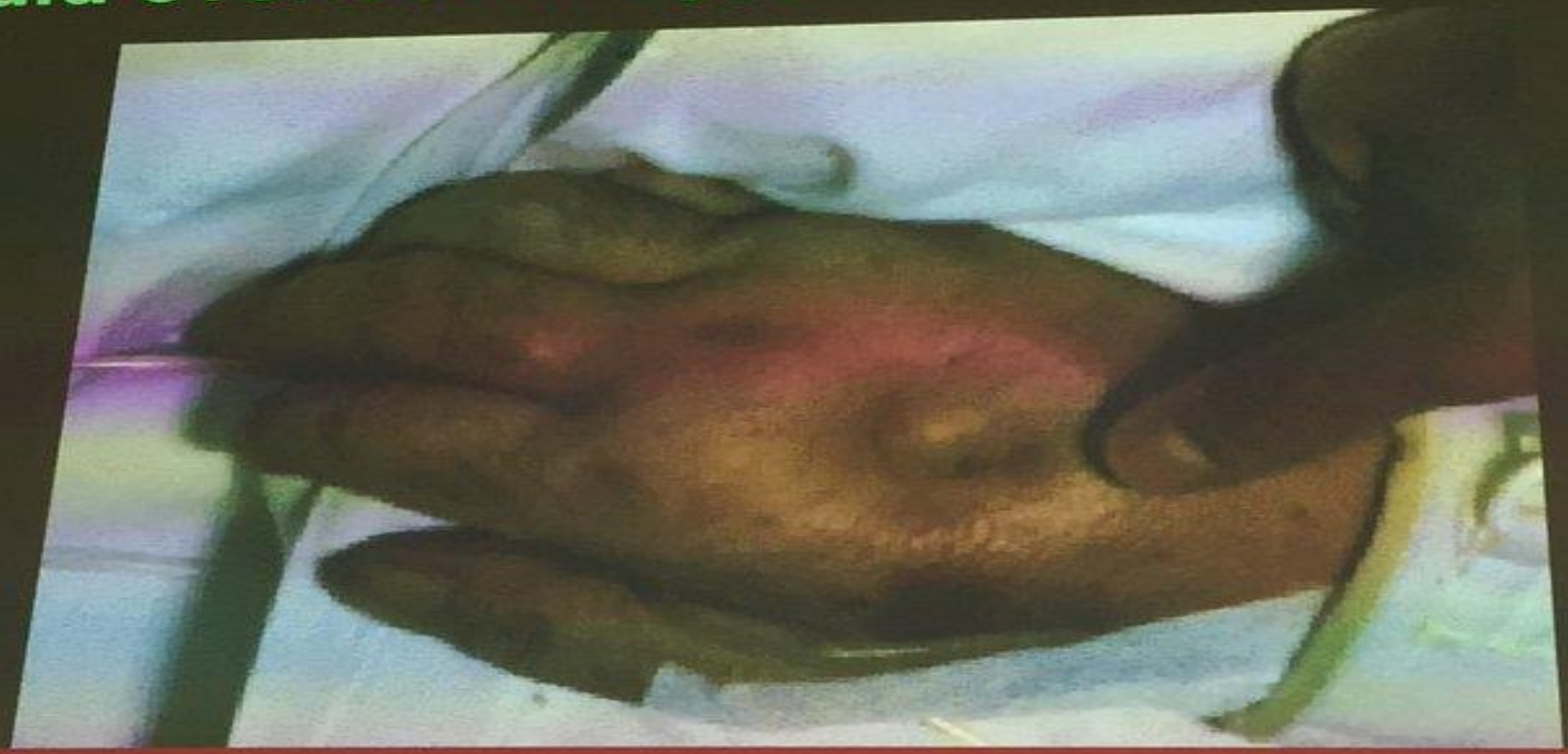



R


DIURETIC
DIURETIC
DIURETIC
DIURETIC

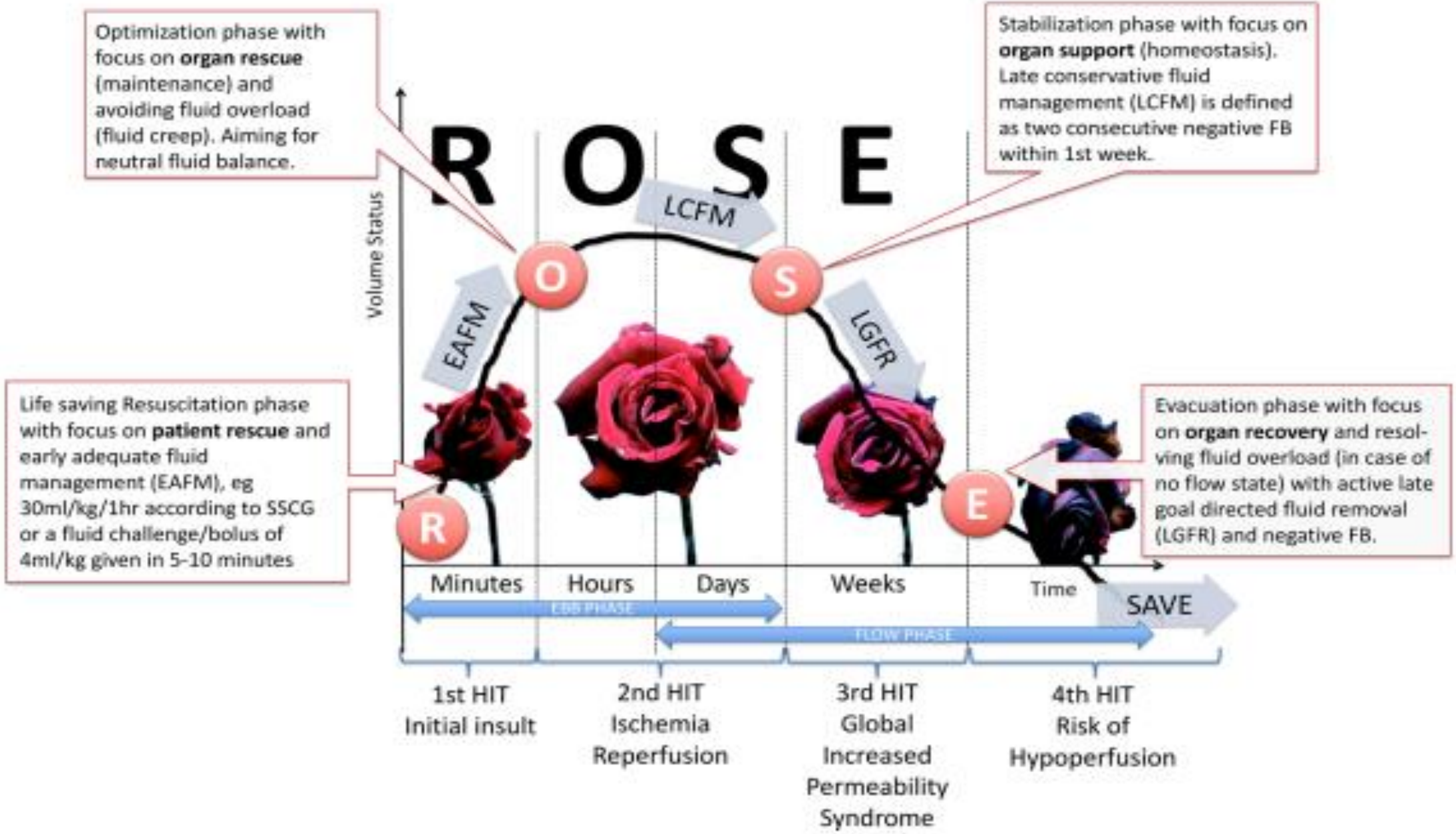
How do you call it?

Fluid overload? Hypervolemia? Edema



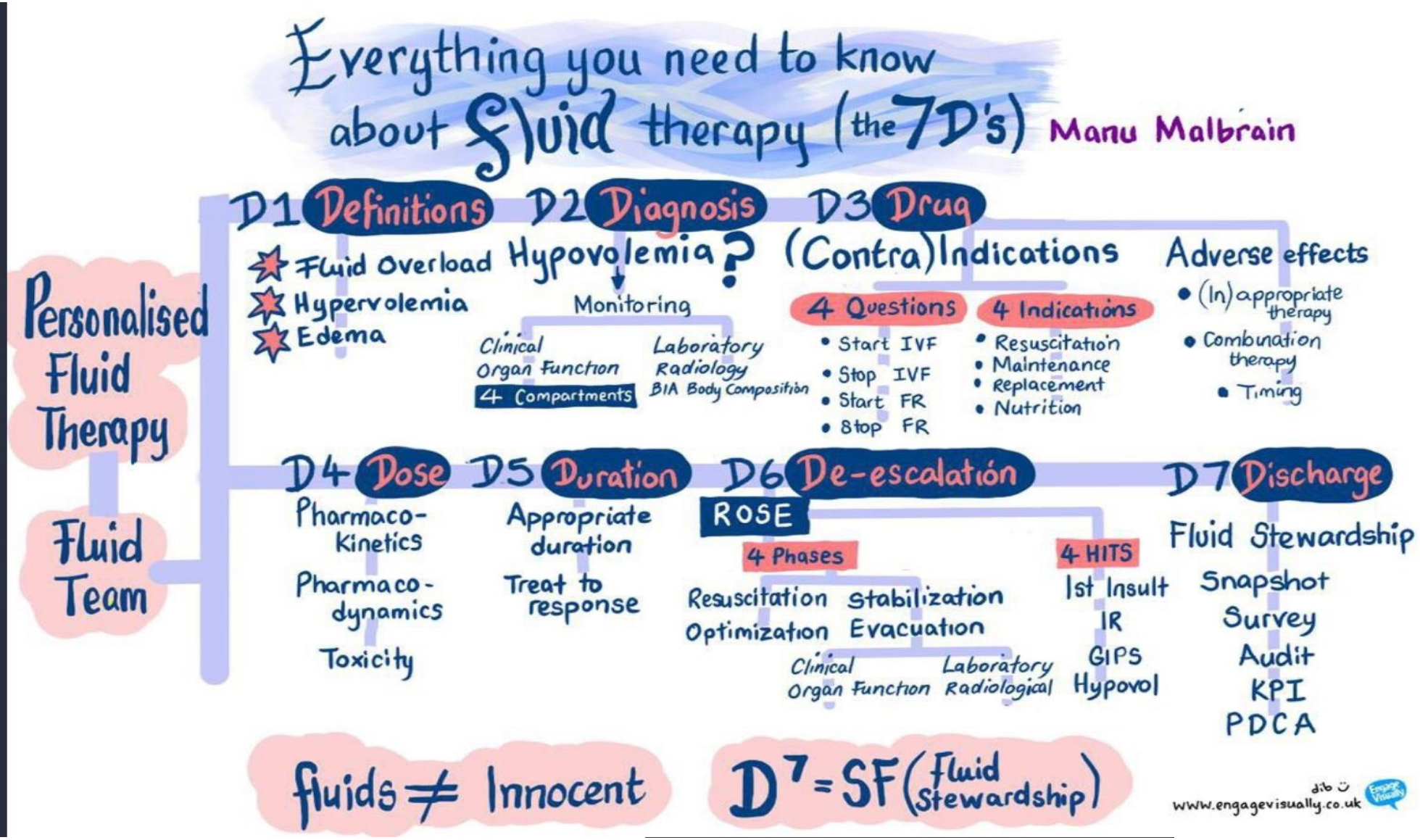
Edema does not exclude hypovolemia!

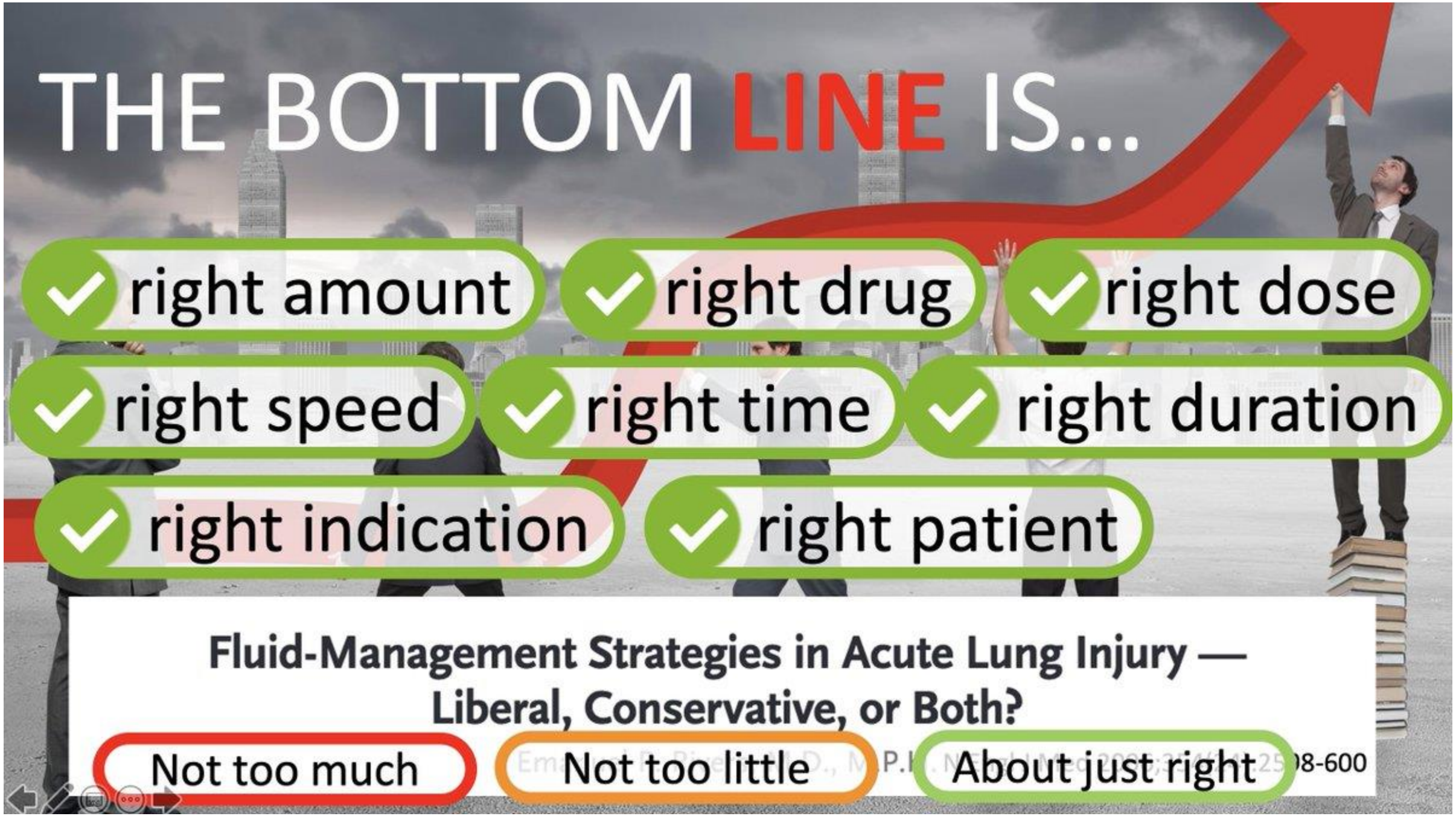
Panel A

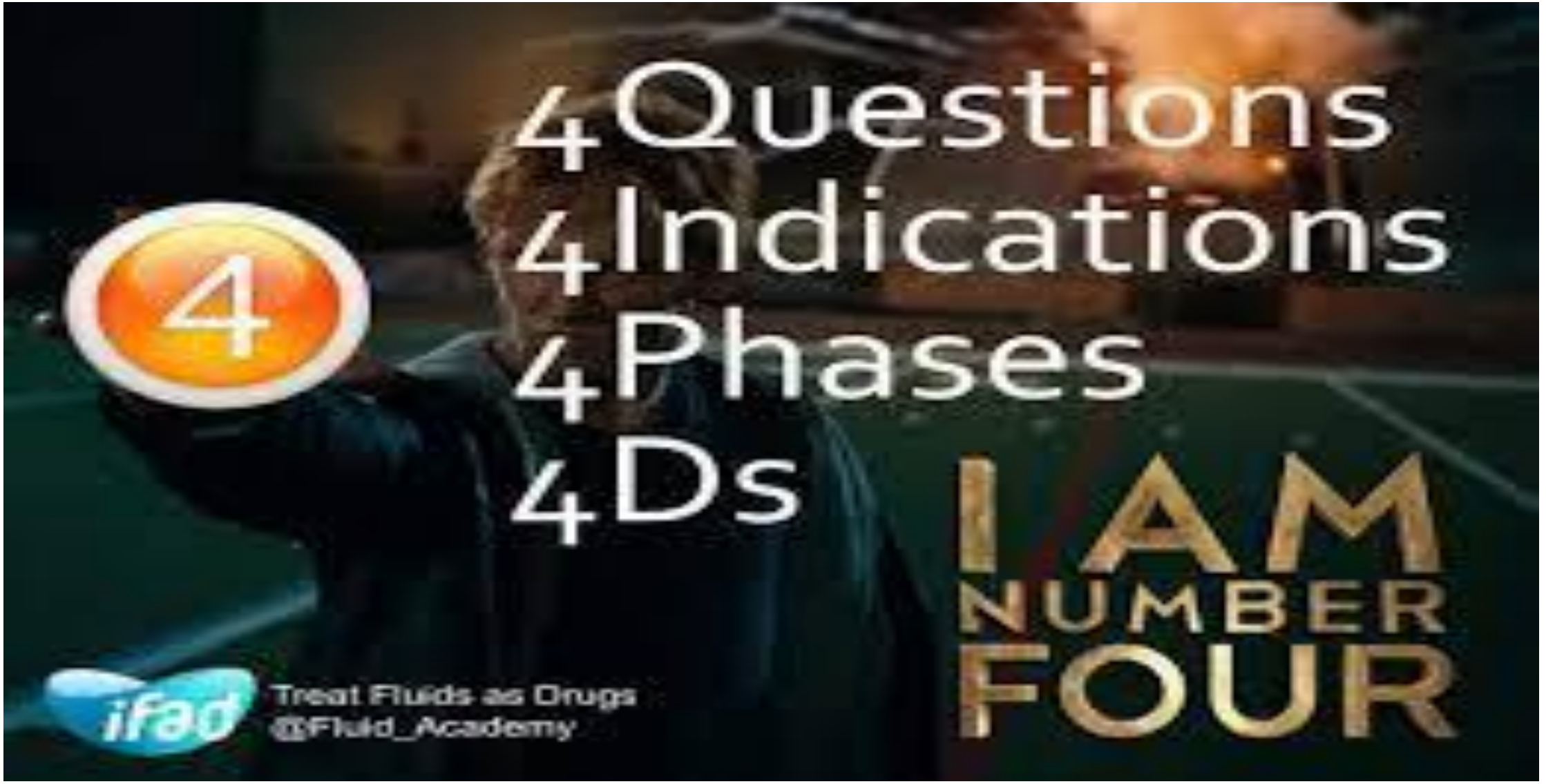

Optimization phase with focus on **organ rescue** (maintenance) and avoiding fluid overload (fluid creep). Aiming for neutral fluid balance.
Stabilization phase with focus on **organ support** (homeostasis). Late conservative fluid management (LCFM) is defined as two consecutive negative FB within 1st week.
R O S E
LCFM
EAFM
LGFR
Volume Status
Life saving Resuscitation phase with focus on **patient rescue** and early adequate fluid management (EAFM), eg 30ml/kg/1hr according to SSCG or a fluid challenge/bolus of 4ml/kg given in 5-10 minutes
Evacuation phase with focus on **organ recovery** and resolving fluid overload (in case of no flow state) with active late goal directed fluid removal (LGFR) and negative FB.
Minutes
Hours
Days
Weeks
Time
SAVE
EDS PHASE
FLOW PHASE
1st HIT
Initial insult
2nd HIT
Ischemia
Reperfusion
3rd HIT
Global
Increased
Permeability
Syndrome
4th HIT
Risk of
Hypoperfusion

D7:Discharge

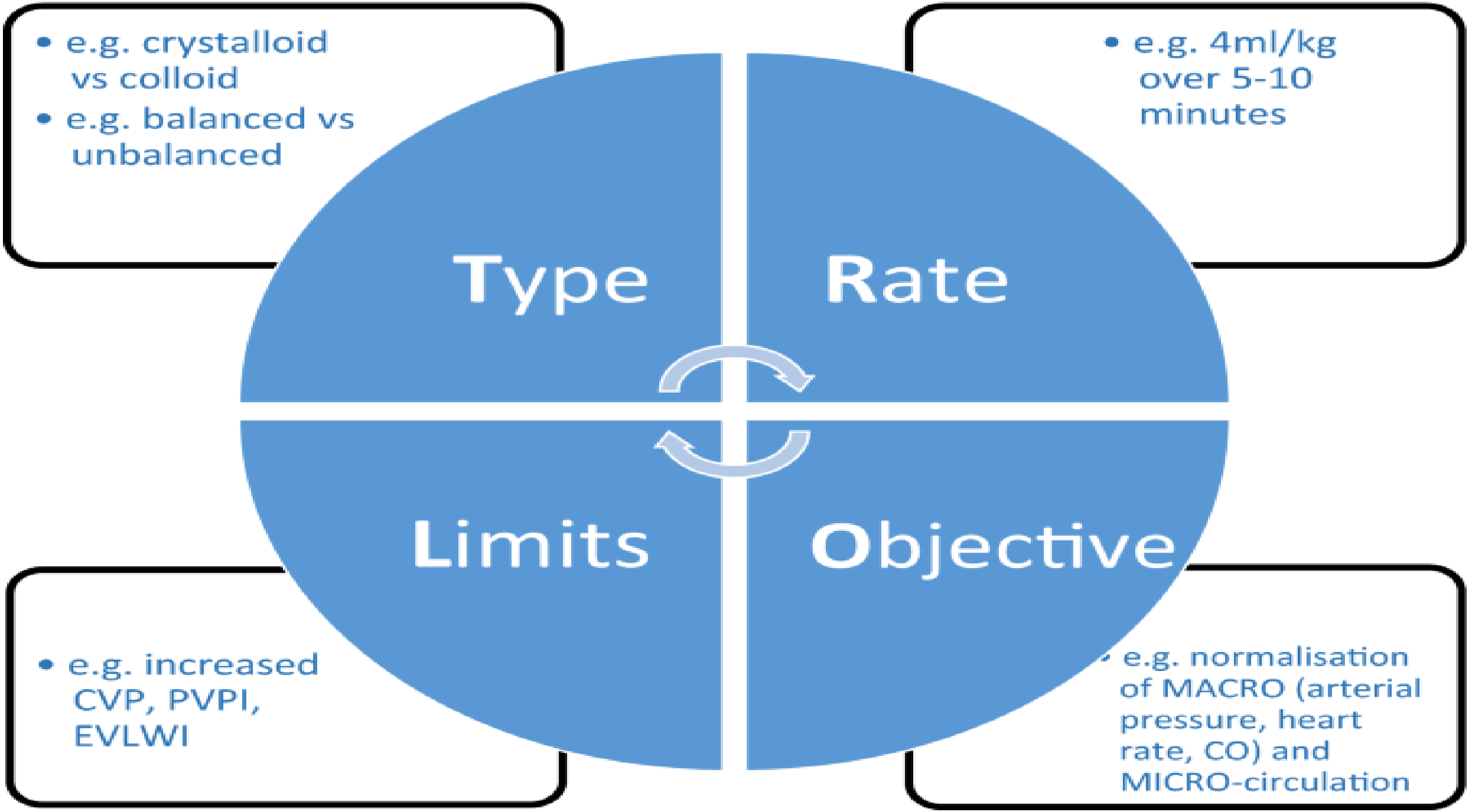

KEY
POINTS


Everything you need to know about Fluid therapy (the 7D's)
Manu Malbrain
Personalised Fluid Therapy
Fluid Team
D1 Definitions
Fluid Overload
Hypervolemia
Edema
D2 Diagnosis
Hypovolemia?
Monitoring
Clinical
Organ Function
4 Compartments
Laboratory
Radiology
BIA Body Composition
D3 Drug
(Contra)Indications
4 Questions
Start IVF
Stop IVF
Start FR
Stop FR
4 Indications
Resuscitation
Maintenance
Replacement
Nutrition
Adverse effects
(In)appropriate therapy
Combination therapy
Timing
D4 Dose
Pharmaco-Kinetics
Pharmaco-dynamics
Toxicity
D5 Duration
Appropriate duration
Treat to response
D6 De-escalation
ROSE
4 Phases
Resuscitation
Stabilization
Optimization
Evacuation
Clinical
Organ Function
Laboratory
Radiological
4 HITS
1st Insult
IR
GIPS
Hypovol
D7 Discharge
Fluid Stewardship
Snapshot
Survey
Audit
KPI
PDCA
fluids ≠ Innocent
D7 = SF (Fluid Stewardship)
www.engagevisually.co.uk

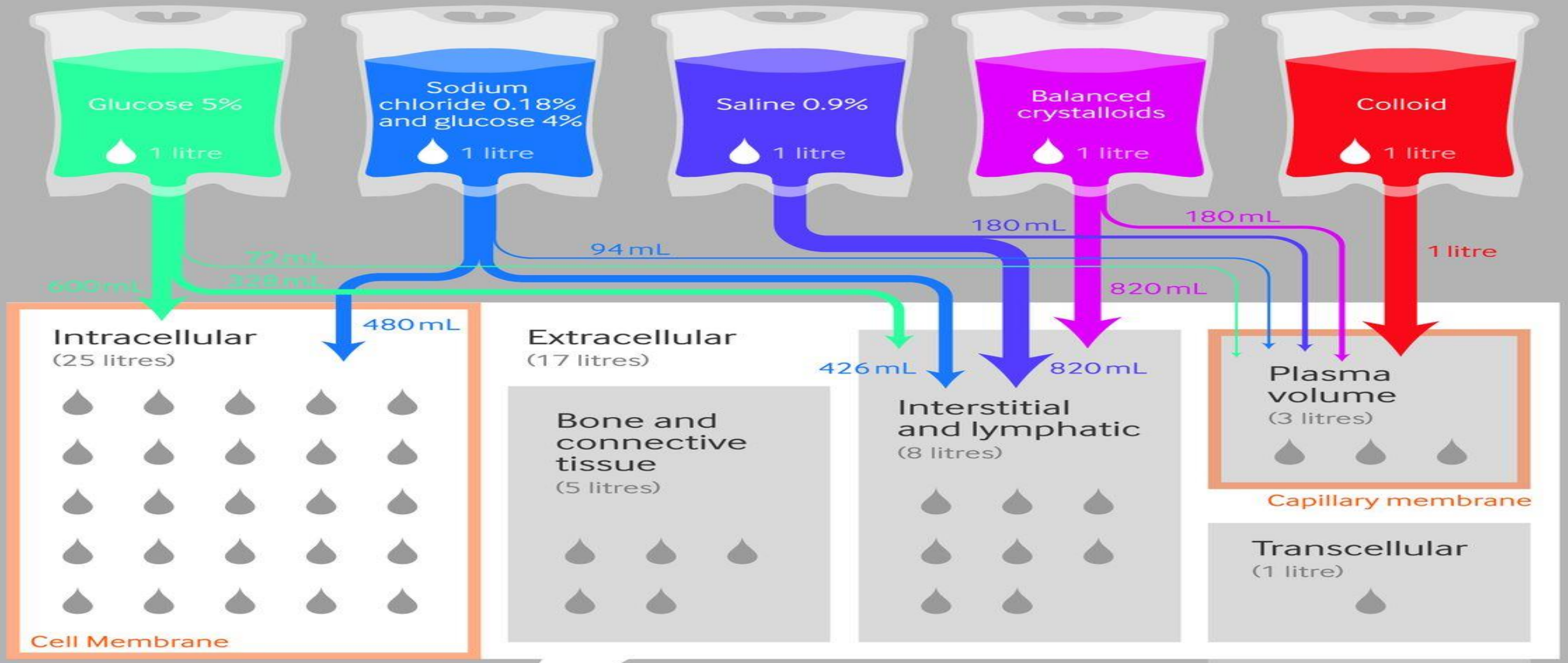

THE BOTTOM LINE IS...
right amount
right drug
right dose
right speed
right time
right duration
right indication
right patient
Fluid-Management Strategies in Acute Lung Injury — Liberal, Conservative, or Both?
Not too much
Not too little
About just right


4Questions
4Indications
4Phases
4Ds
I AM
NUMBER
FOUR
ifad
Treat Fluids as Drugs
@Fluid_Academy


Why And
WHAT
Drug
Dose
HOW MUCH
HOW LONG
Duration
De-escalation
WHEN


• e.g. crystalloid vs colloid
• e.g. balanced vs unbalanced
Type
• e.g. 4ml/kg over 5-10 minutes
Rate
Limits
• e.g. increased CVP, PVPI, EVLWI
Objective
• e.g. normalisation of MACRO (arterial pressure, heart rate, CO) and MICRO-circulation

Theoretical distribution of intravenous fluids on infusion

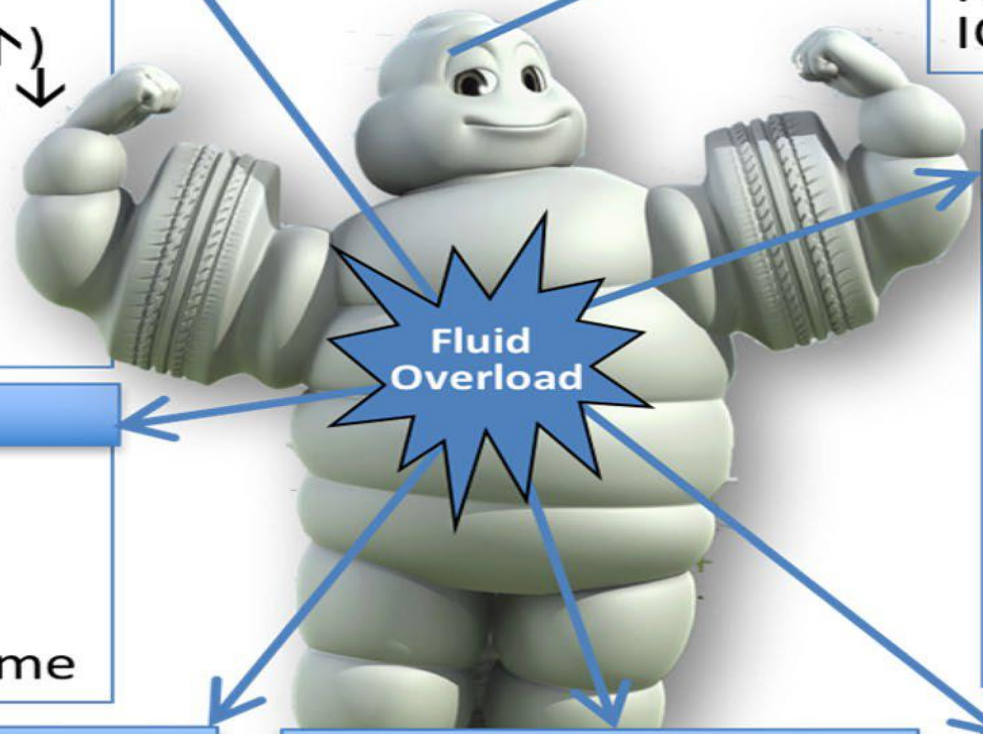


70 kg man
42 litres total body water

💧 = 1 litre

Electrolyte composition of some transcellular fluids (mmol/L)

Fluid	Na^+	K^+	Cl^-	HCO^{3-}
Sweat	65	8	39	16
Gastric	20—100	5—10	120—160	0
Bile	150	5—10	40—80	20—40
Ileal	140	5	105	40



Respiratory

Pulmonary edema ↑
Pleural effusion ↑
Altered pulmonary and chest wall elastance (cfr IAP ↑)
paO2 ↓ paCO2 ↑ PaO2/FiO2 ↓
Extra vascular lung water ↗
Lung volumes ↓ (cfr IAP ↑)
Prolonged ventilation ↑
Difficult weaning ↑
Work of breathing ↑

Central nervous system

Cerebral edema, impaired cognition, delirium
ICP ↑ CPP ↓ IOP ↑
ICH, ICS, OCS

Cardiovascular

Myocardial edema ↑
Conduction disturbance
Impaired contractility
Diastolic dysfunction
CVP ↑ and PAOP ↑
Venous return ↓
SV ↓ and CO ↓
Myocardial depression
Pericardial effusion ↑
GEF ↓ GEDVI ↑ CARS ↑

Hepatic

Hepatic congestion ↑
Impaired synthetic function
Cholestatis ↑
Cytochrome P 450 activity ↓
Hepatic compartment syndrome

Gastrointestinal/visceral

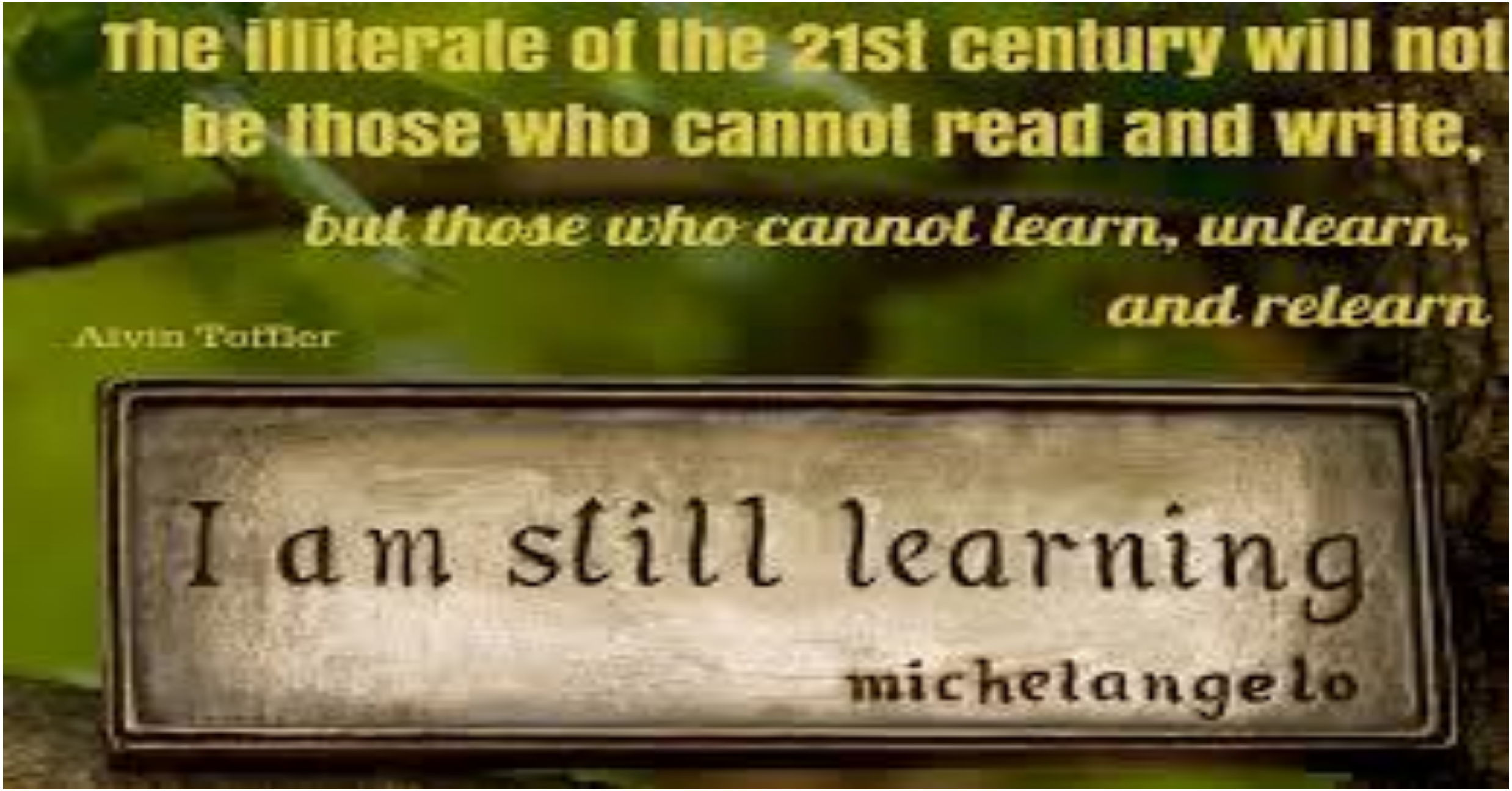
Ascites formation ↑ Gut edema ↑
Malabsorption ↑ Ileus ↑
Bowel contractility ↓
IAP ↑ and APP (=MAP-IAP) ↓
Success enteral feeding ↓
Intestinal permeability ↑
Bacterial translocation ↑
Splanchnic microcirculatory flow ↓
ICG-PDR ↓, pHi ↓

Abdominal Wall

Tissue edema ↑
Poor wound healing ↑
Wound infection ↑
Pressure ulcers ↑
Abdominal compliance ↓

Renal

Renal interstitial edema
Renal venous pressure ↑
Renal blood flow ↓
Interstitial pressure ↑
Salt + water retention ↑
Uremia ↑ GFR ↓ RVR ↑
Renal CS


The illiterate of the 21st century will not be those who cannot read and write,
but those who cannot learn, unlearn,
and relearn
Alvin Toffler
I am still learning
michelangelo