Polytrauma:
Pathophysiology and Management

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Polytrauma

pathophysiology, priorities, and management

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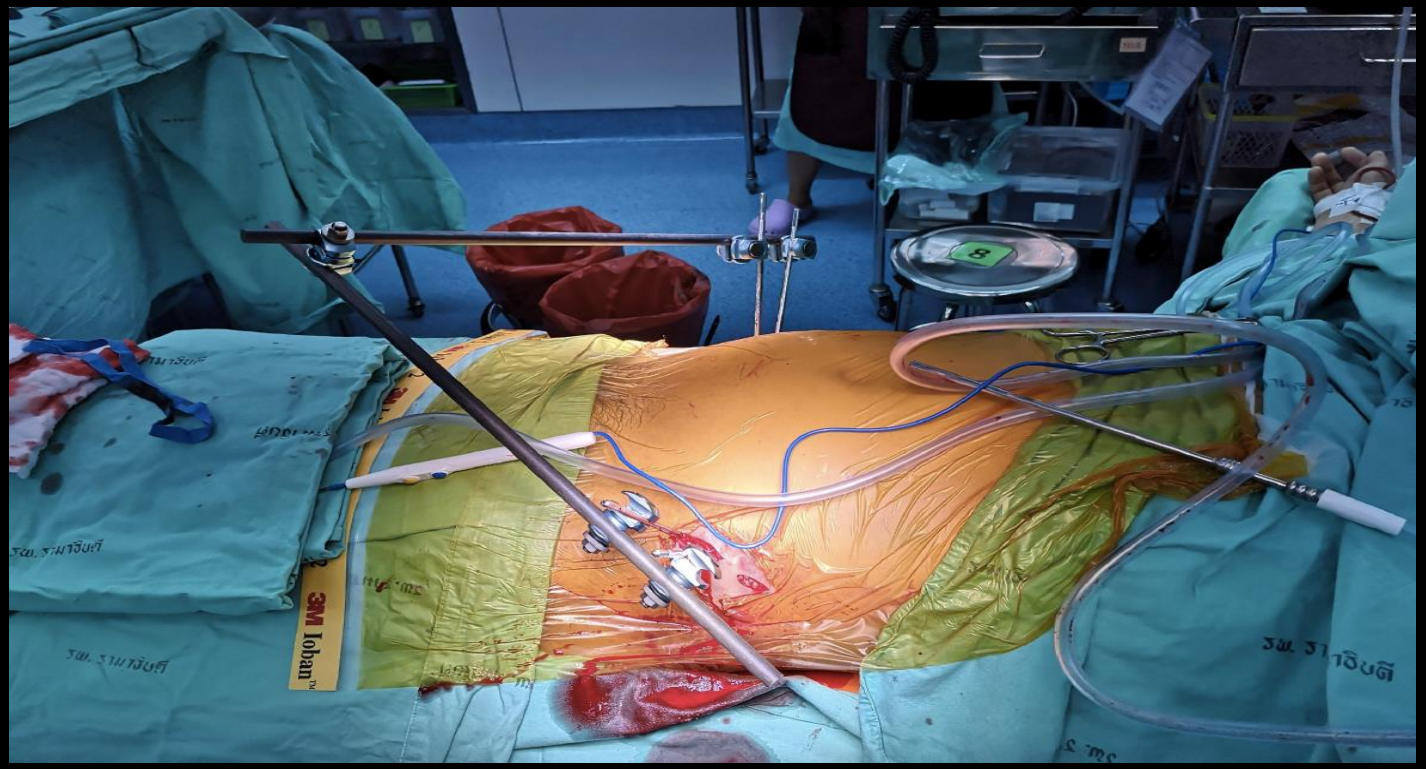


pathophysiology : Tharin Thampongsa M.D.

Definition

- Polytrauma is not a synonym of multiple fractures.
- Multiple fractures are purely orthopedic problem as there is involvement of skeletal system.
- While in polytrauma there is involvement of more than one system like associated head injury / chest injury / spinal injury / abdominal or pelvic injury

Polytrauma

Syndrome of multiple injuries with **sequential systemic reactions** that may lead to **dysfunction or failure** of remote organ and vital systems, which have not themselves been directly injured.

AIS >2 or ISS >=16

1

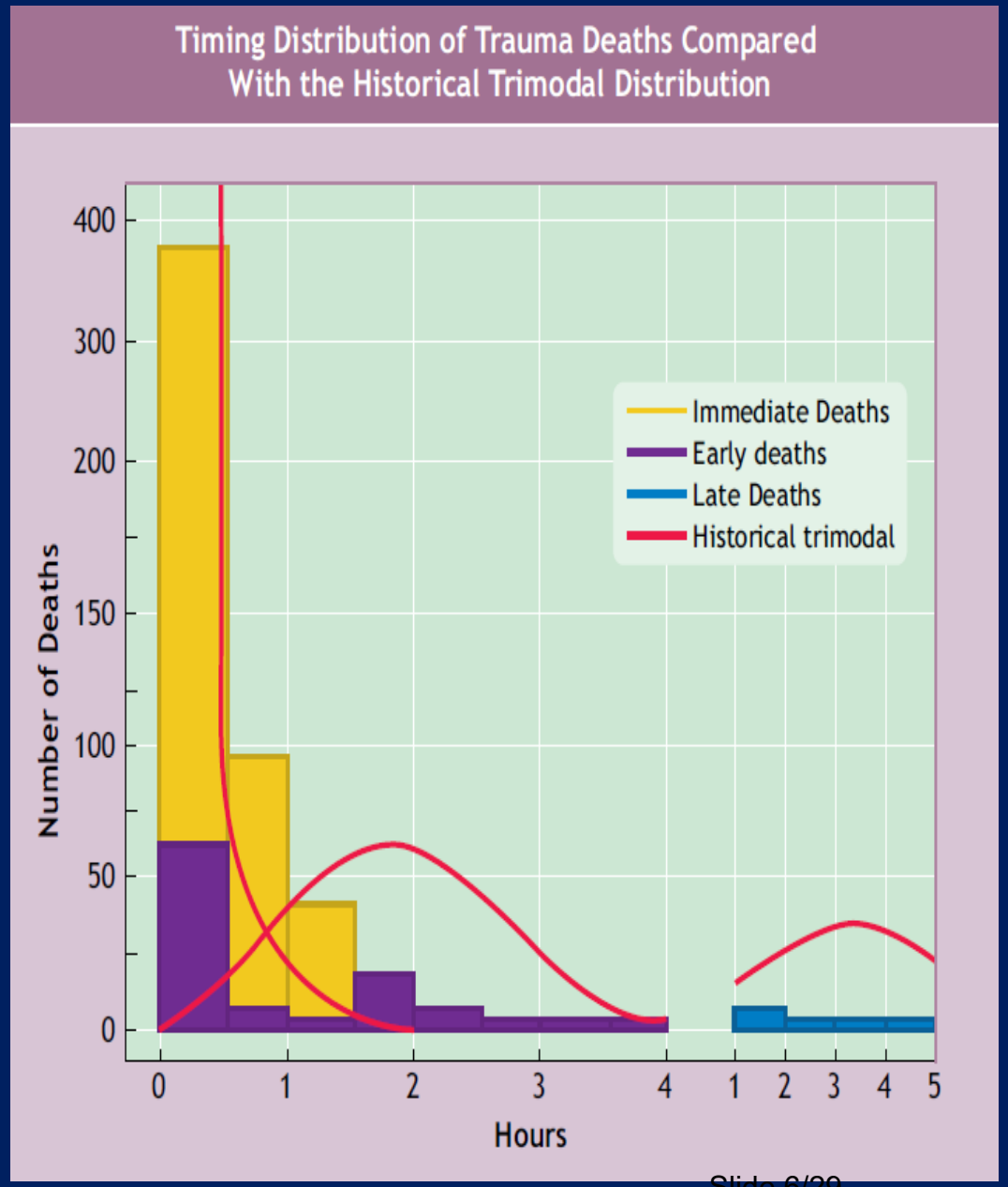
1.First peak -major neurological or vascular injury

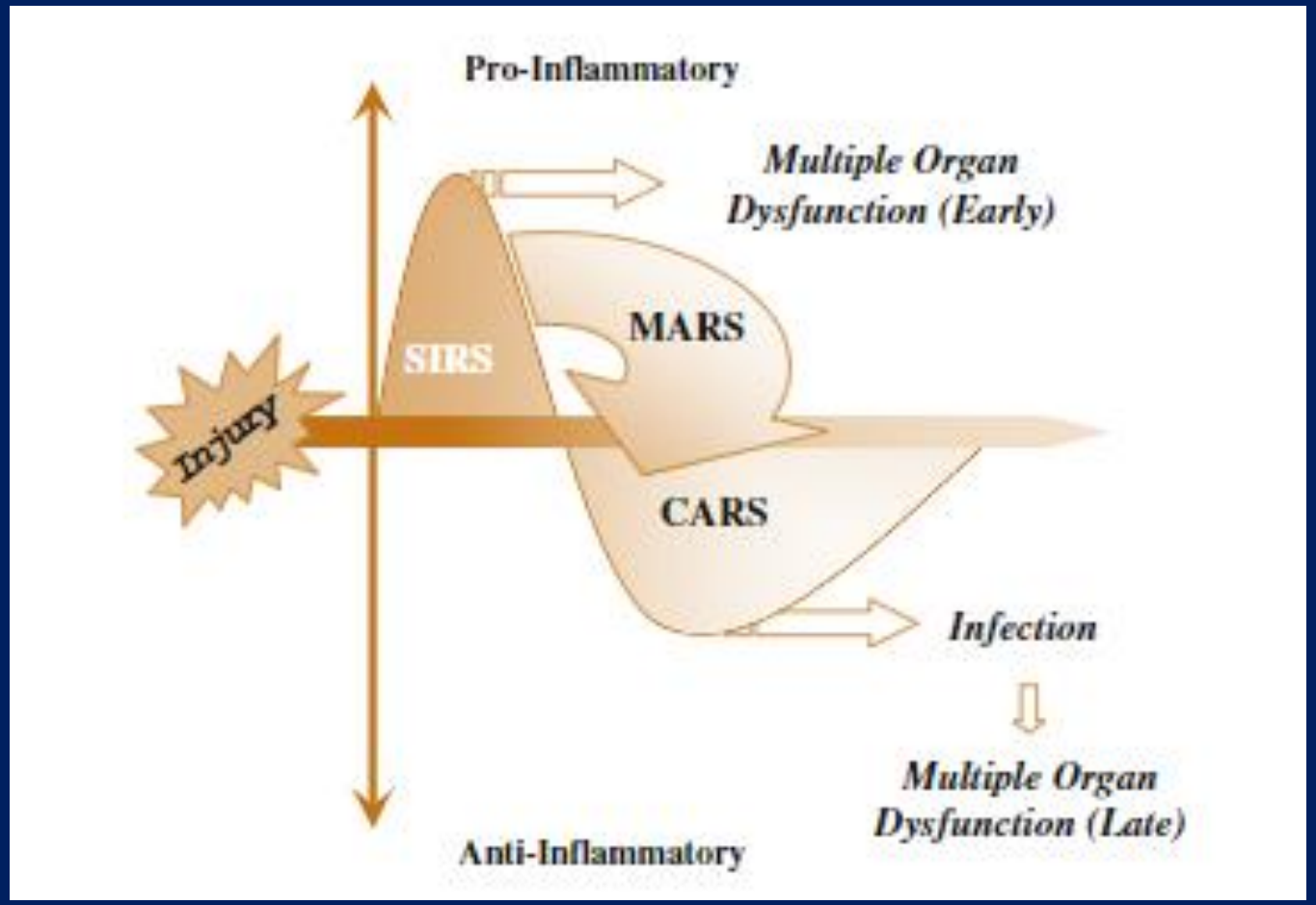
2

2.second peak– intracranial,major thoracic or abdominal/pelvic injury,multiple limb injury .*** The Golden hour ***

3

3.third peak- sepsis and multiple organ failure.





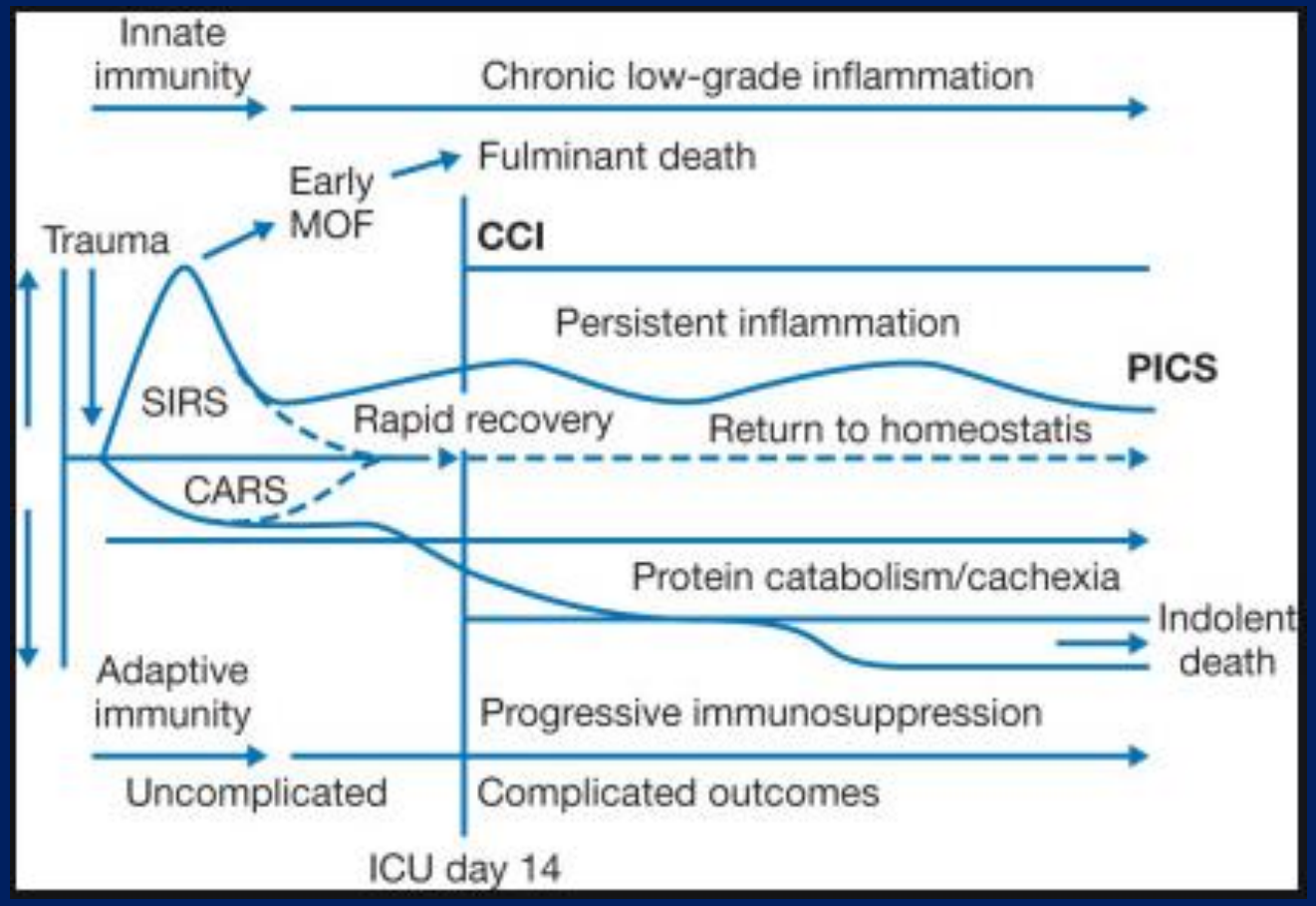
systemic inflammatory response syndrome (SIRS)

Compensatory antiinflammatory response syndrome (CARS)

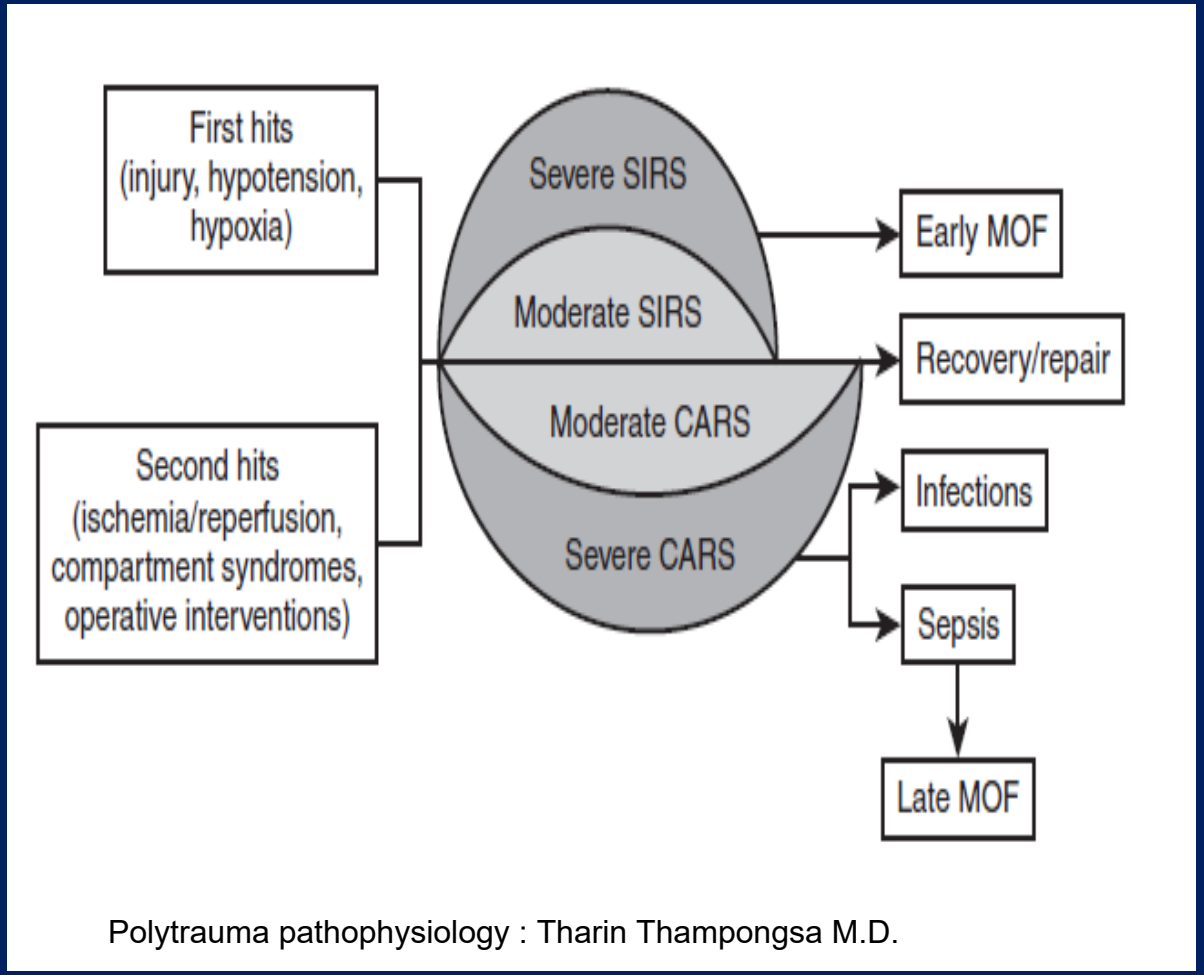
Mixed antagonistic response syndrome (MARS)

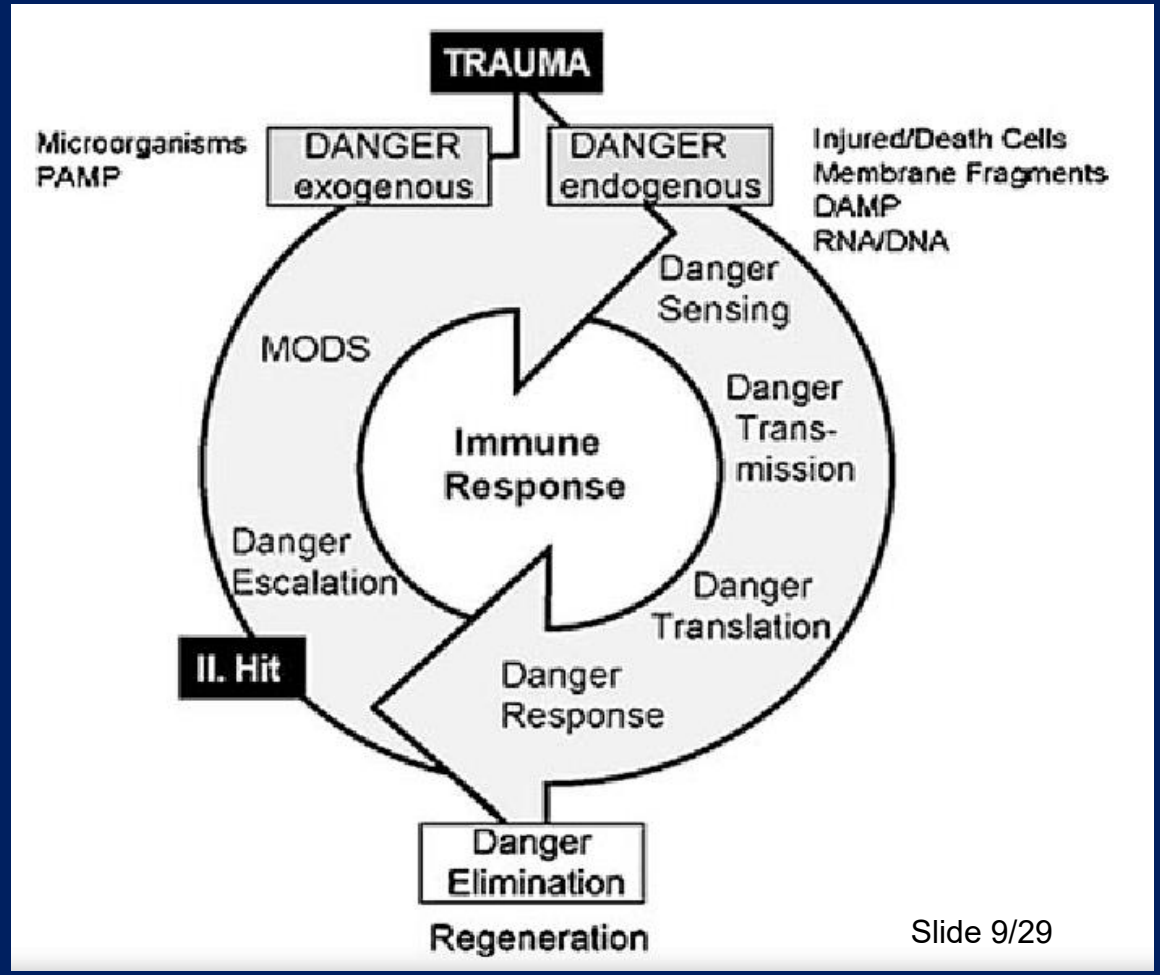
Trauma-induced intravascular coagulopathy (TIC)

Model of the inflammatory response in sepsis



Pathophysiological





Pathophysiological

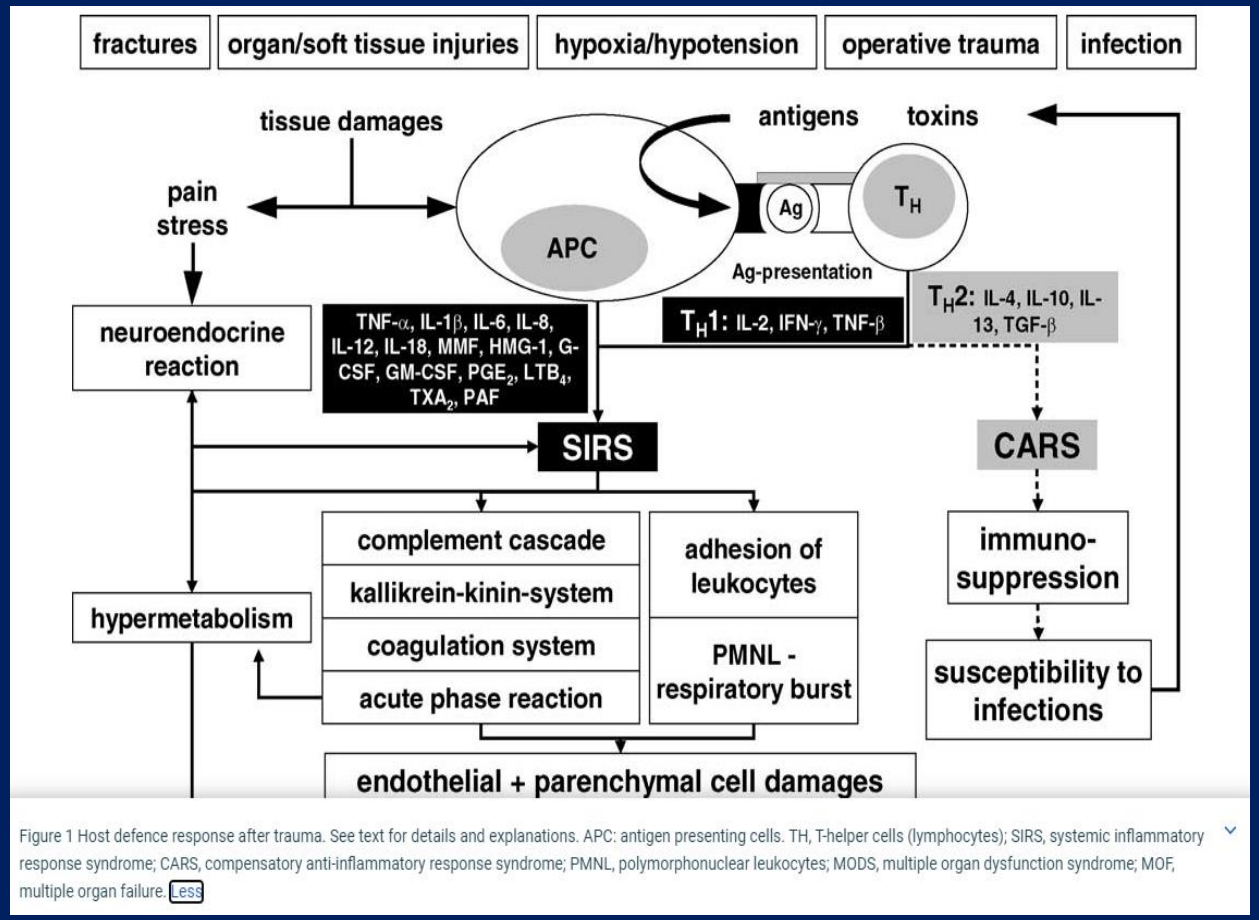


Figure 1 Host defence response after trauma. See text for details and explanations. APC: antigen presenting cells. TH, T-helper cells (lymphocytes); SIRS, systemic inflammatory response syndrome; CARS, compensatory anti-inflammatory response syndrome; PMNL, polymorphonuclear leukocytes; MODS, multiple organ dysfunction syndrome; MOF, multiple organ failure. Less

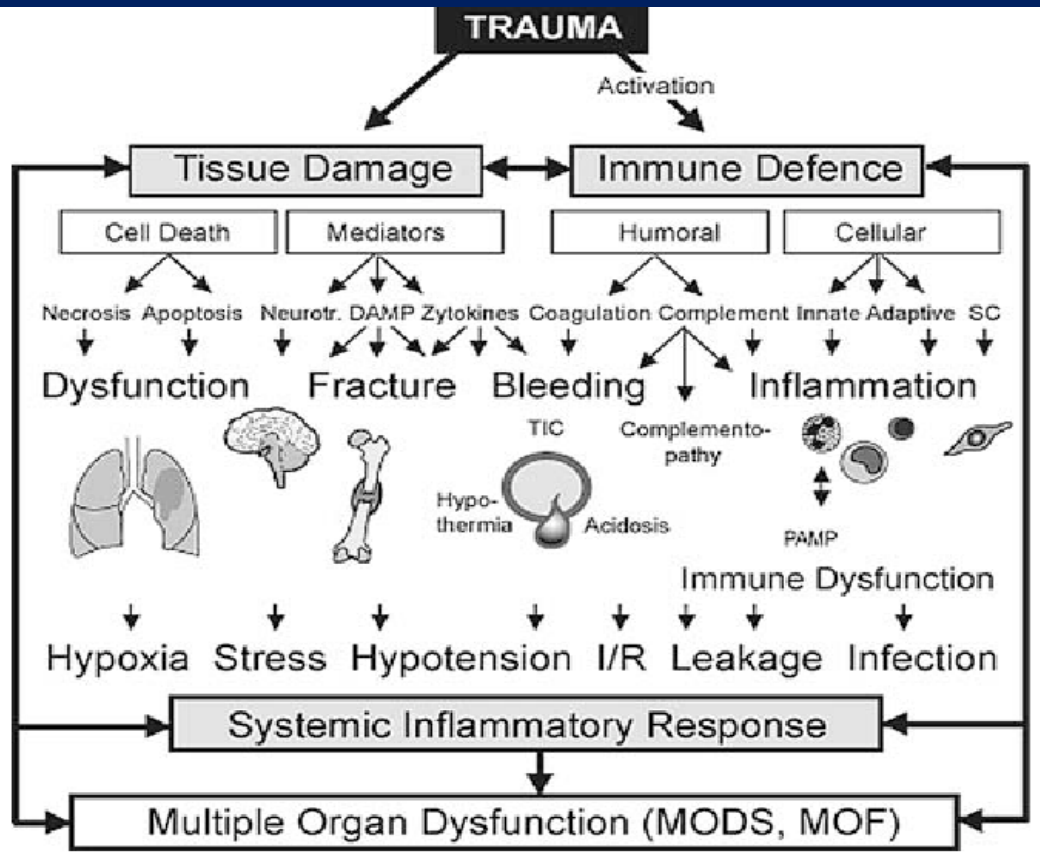
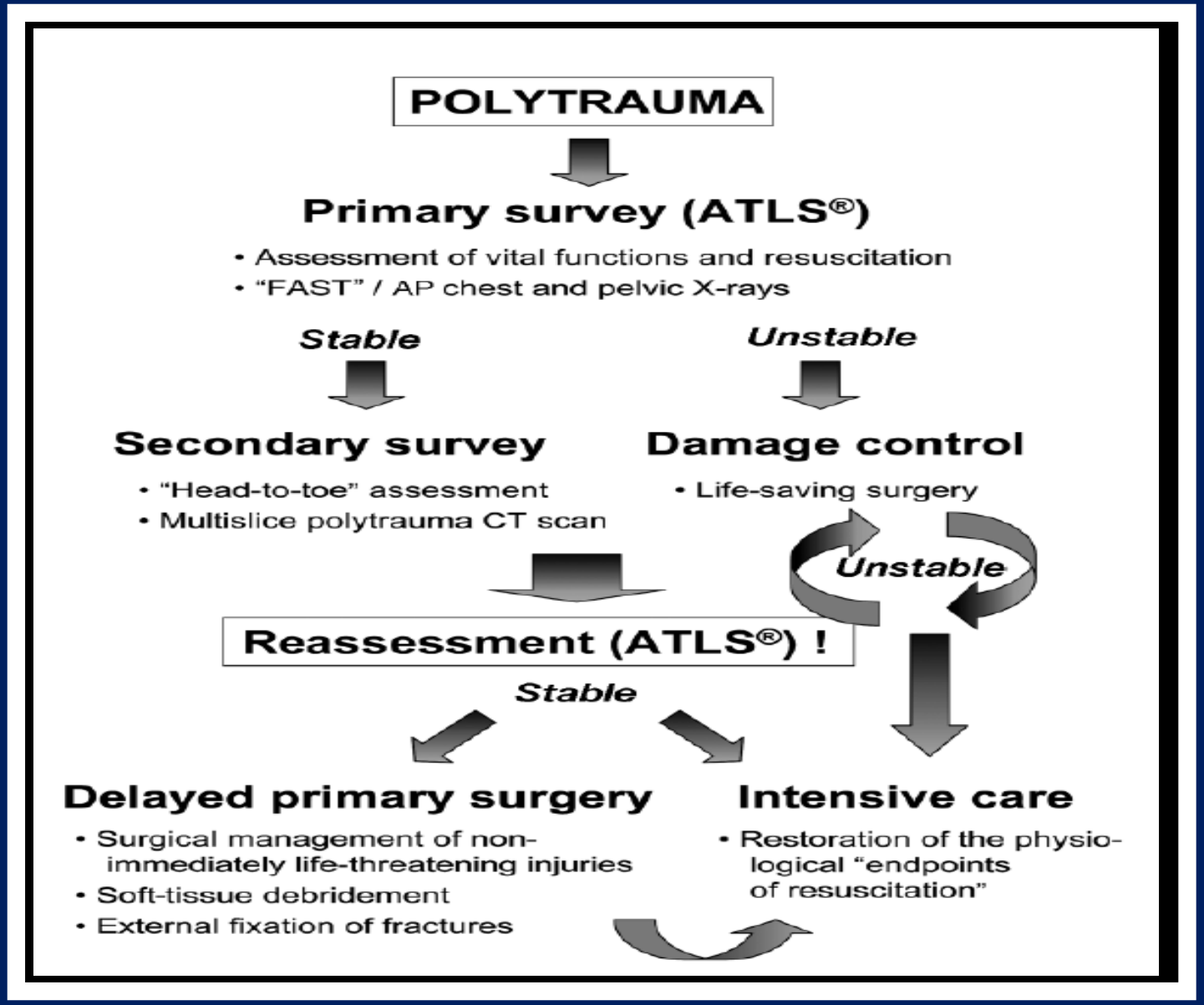


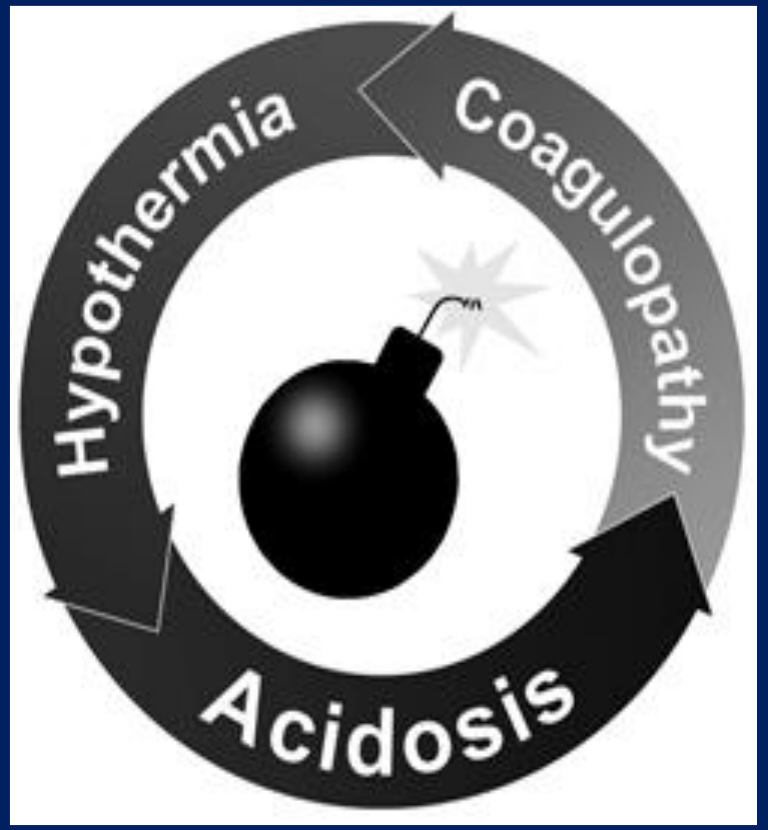
Fig. 2 Important pathophysiological changes after polytrauma.

Pathophysiology of polytrauma • M Keel, O Trentz Published 2005 Medicine Injury



Phase I

Life saving procedure



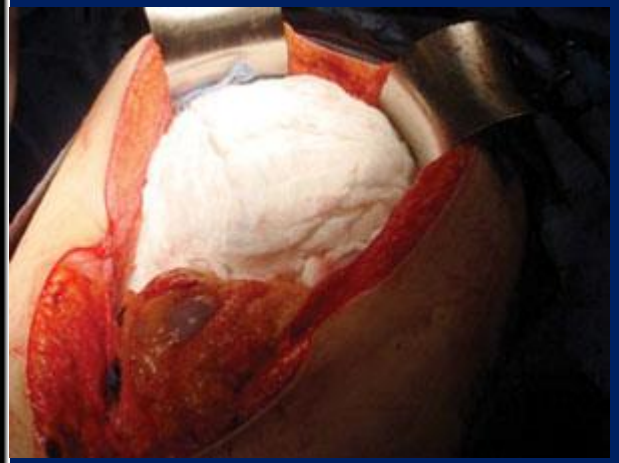
Current Concepts of Polytrauma Management Philip F. Stahel*, Christoph E. Heyde*, Wolfgang Ertel

Damage Control Surgery

Hemorrhage => Temporary clamp, shunt, ligate, pack

Hollow organ => Closed or resect without anastomosis

Musculoskeletal => Ext fixation



Holcomb JB. et al, J Trauma 2007

Phase II : Vulnerable
hyperinflammation

- Patient defense is Uncontrolled
- Minimize “ second hit “

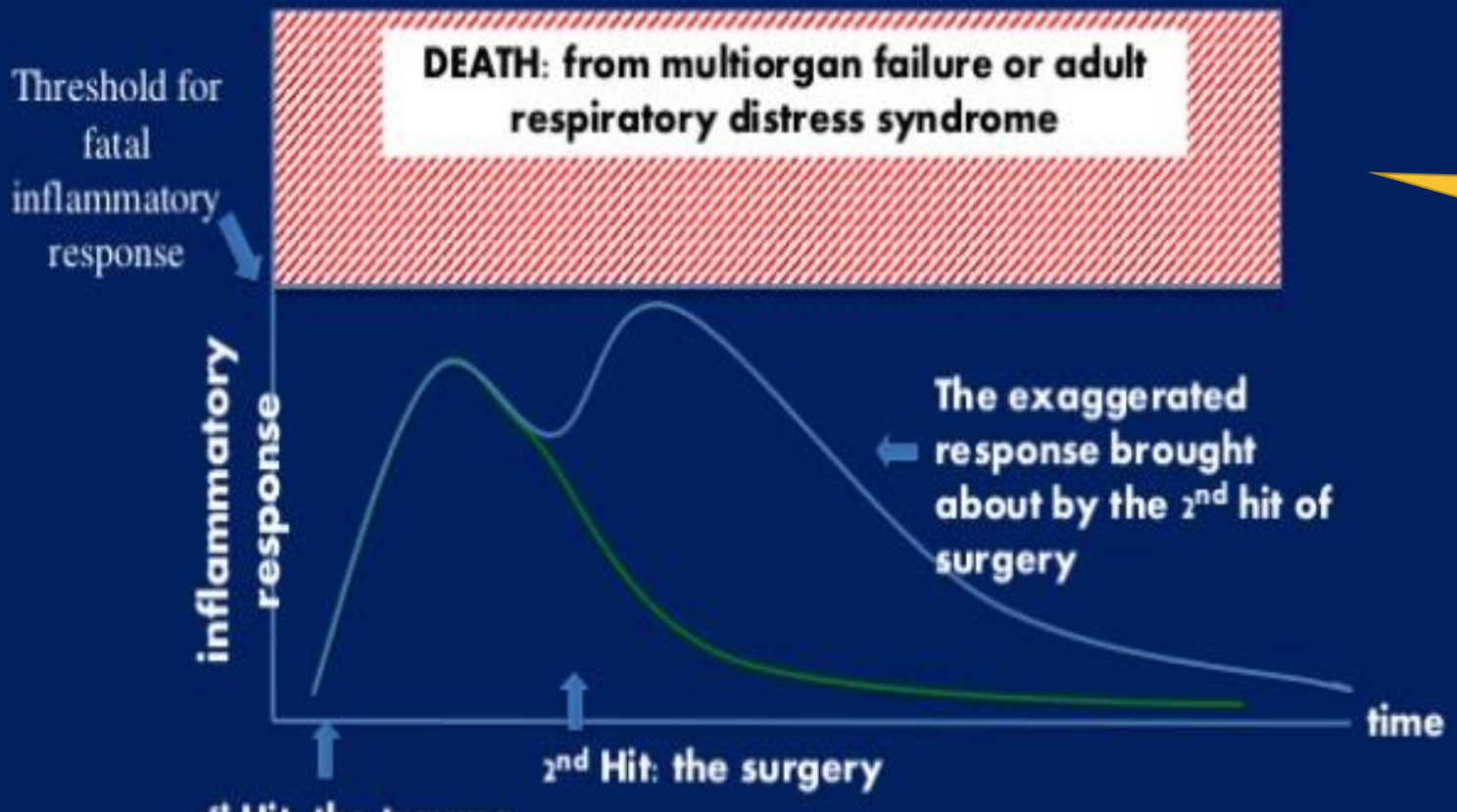
Phase III : Inertia
Window of opportunity

- 1 st Reconstructive surgery
  - Definitive surgery of
  - Long bone fractures-shaft-articular

Pathophysiological

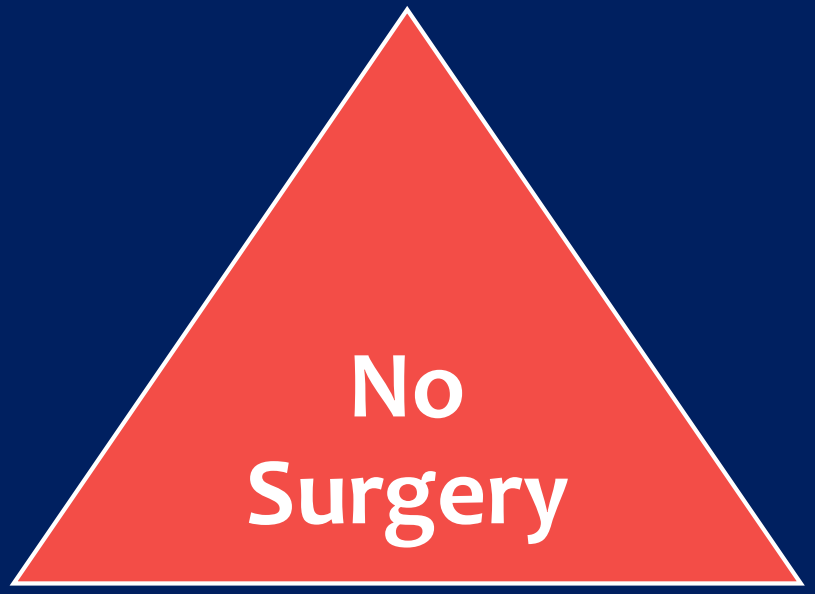
THE 'SECOND HIT' (2-5 DAYS)

Severe trauma can result in a life threatening inflammatory response (SIRS)



Second looks only

Phase IV : immunosuppression



Phase V : Recovery

- Secondary reconstructive surgery

Time to surgery

- Day 1 DCS
- Days2-5 Second Look
- Days5-10 window of opportunity
- Days12-21 Immunosuppression : NO surgery
- Week3+ Recovery : secondary reconstructive surgery

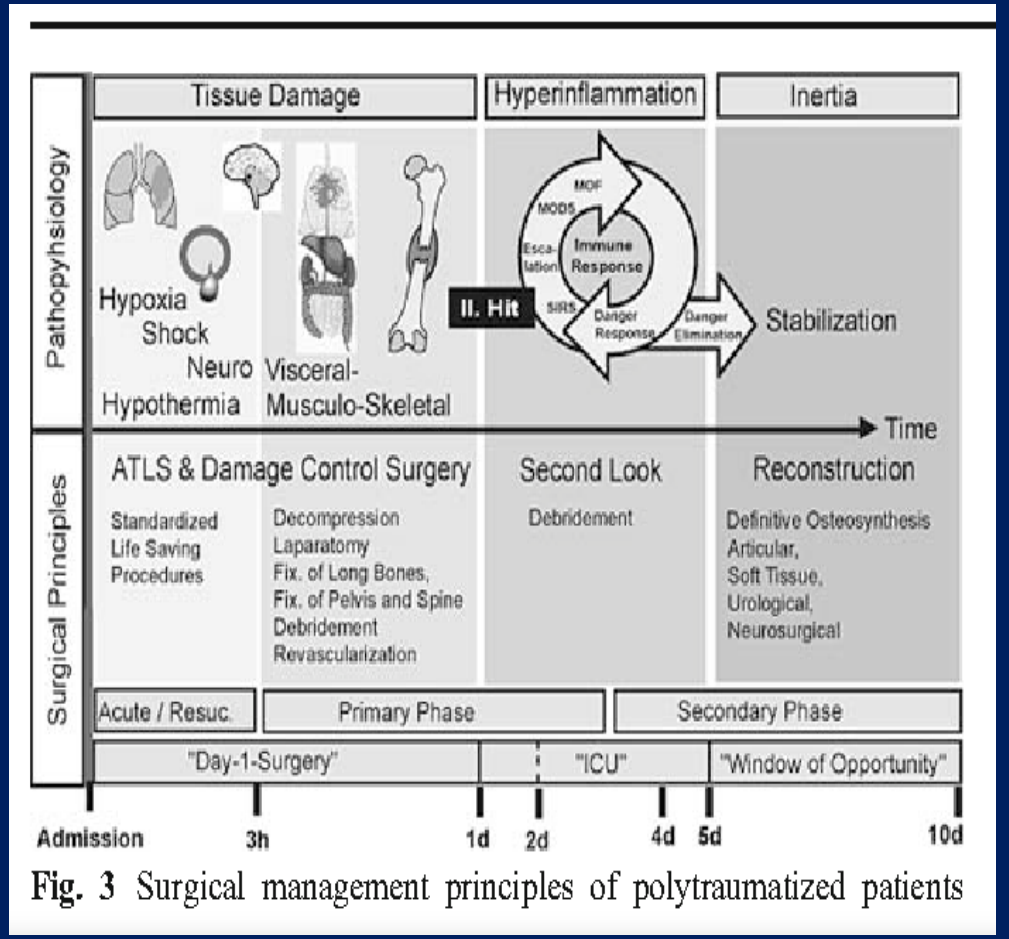


Fig. 3 Surgical management principles of polytraumatized patients

Window of opportunity

Indicators for possible surgical procedures during the secondary phase after multiple injury
>5 days after trauma
Stabilized hemodynamic (without vasoconstrictors)
Stabilized coagulation
Improvement of oxygenation (paO_2/ FiO_2>280)
Normothermia
Reduced capillary leakage
Controlled inflammation: falling CRP (<110 mg/l), procalcitonin, IL-6
Reduction of SOFA score
Serum lactat<2 mmol/l
Diuresis>1 ml/kg BW/h
Platelets>100,000/µl
Intracranial pressure<20 mm Hg

Table 2. Timing and priorities of operative interventions in polytrauma patients depending on the physiological status [24].

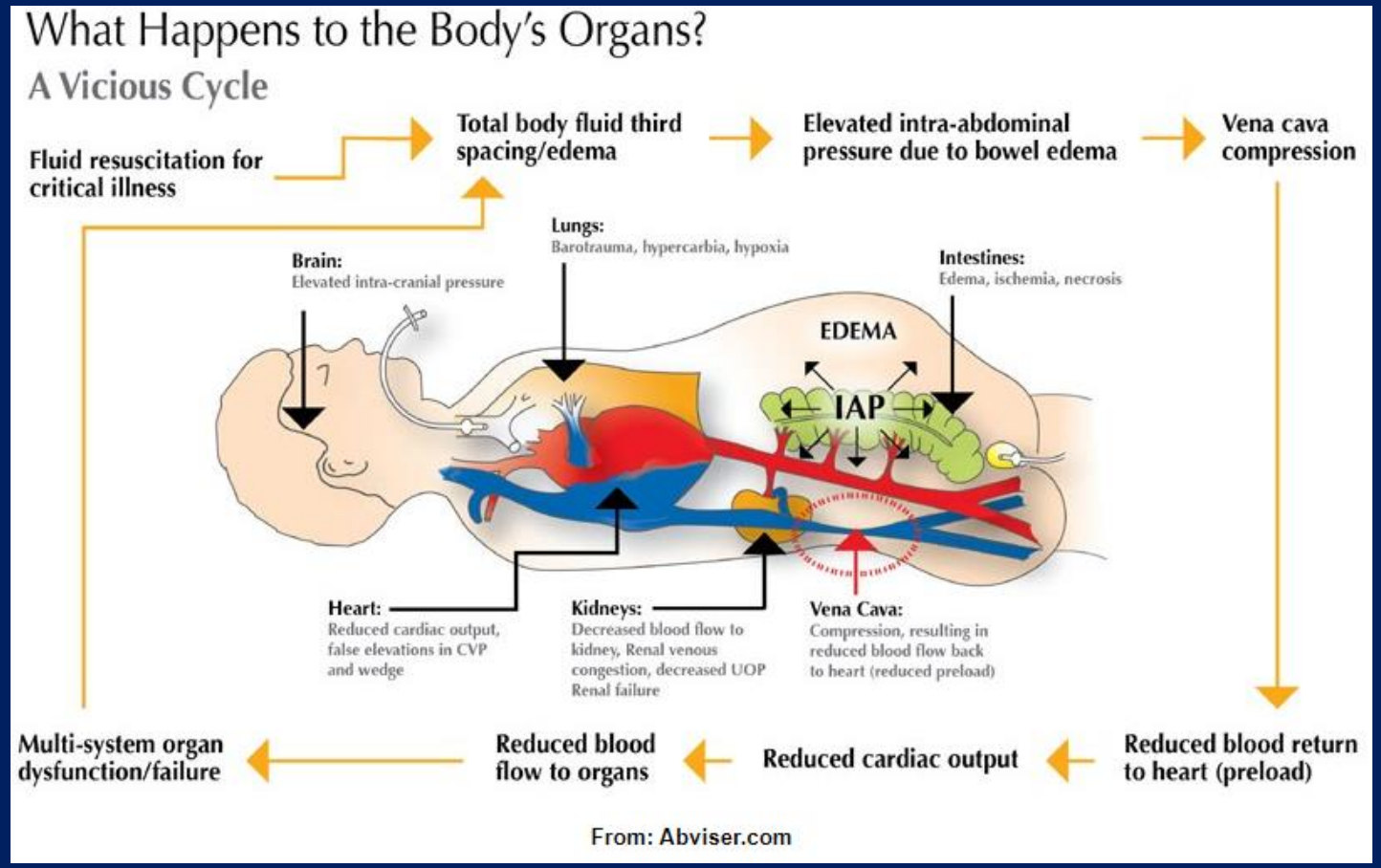
Physiological status		Operative procedures	Timing
Compromised vital functions	→	Life-saving surgery	
Stable vital functions	→	Delayed primary surgery	Day 1
Highly unstable/*in extremis*	→	Damage control surgery	
Hyperinflammation		"Second looks" only!	Day 2–4
"Window of opportunity"		Scheduled definitive surgery	Day 5–10
Immunosuppression		No surgery!	
Recovery		Secondary reconstructive surgery	After 3 weeks

Timing and priorities of surgery

Current Concepts of Polytrauma Management

Philip F. Stahel*, Christoph E. Heyde*, Wolfgang Ertel1

Abdominal Compartment Syndrome



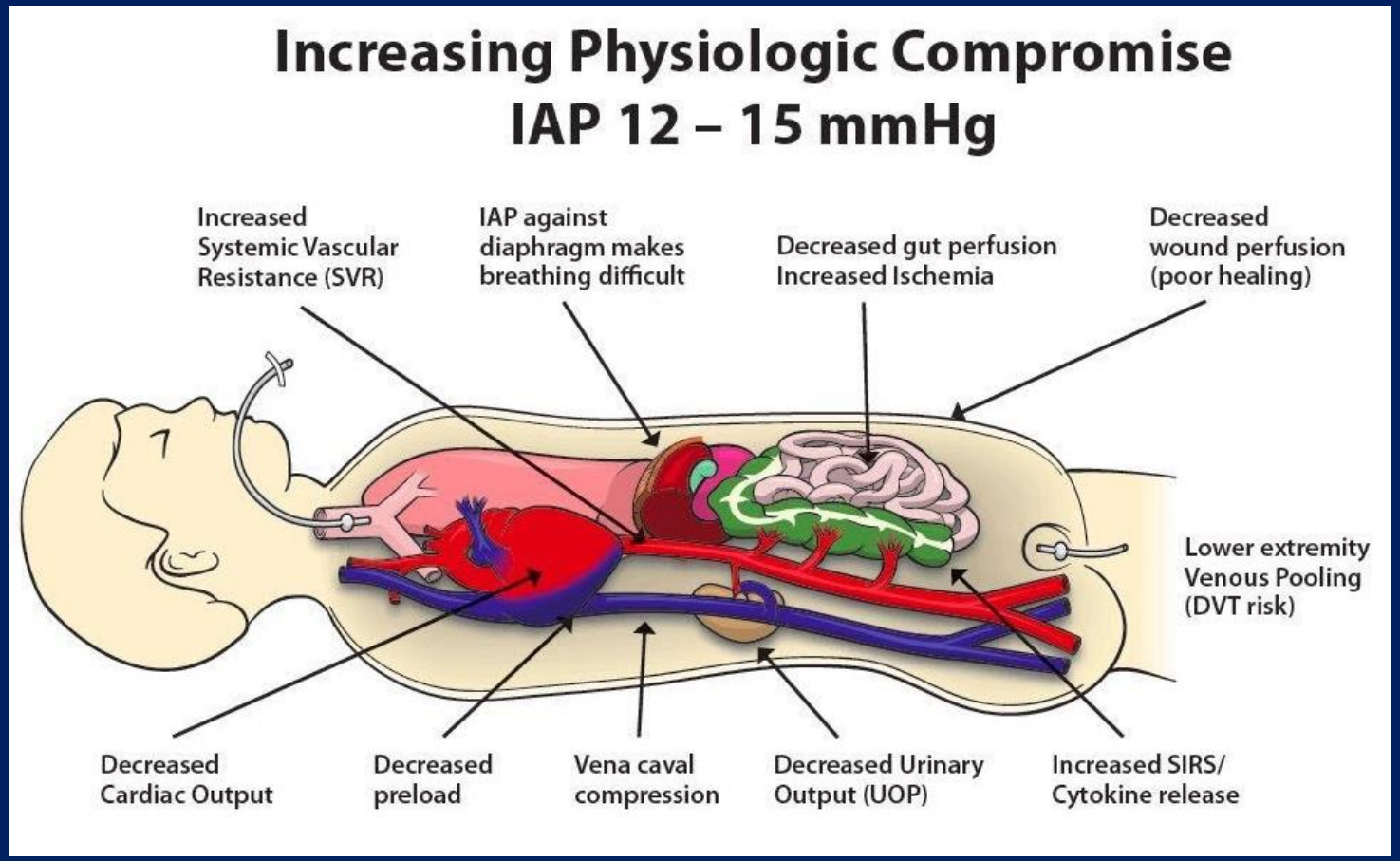
Grade I: IAP 12-15 mm Hg
Grade II: IAP 16-20 mm Hg
Grade III: IAP 21-25 mm Hg
Grade IV: IAP >25 mm Hg

https://sites.google.com/a/learnpicu.com/learnpicu/gi/abdominal-compartment-syndrome

Diagnosis

- Imaging not always helpful
- Measurement of IAP is key
- Measurement technique:
  - Bladder pressure:
    - 1) Drainage catheter of Foley is clamped.
    - 2) Sterile saline (1 cc/kg up to 25 cc) is instilled into the bladder via the aspiration port and the catheter also is filled with fluid
    - 3) 18 gauge needle attached to pressure transducer is inserted into the aspiration port (or via the same set up as an arterial line transducer)
    - 4) Pressure is measured at end expiration in the supine position after ensuring no abdominal muscle contractions are present
    - 5) Zeroed at level of mid-axillary line
  - May not be accurate if there are intraperitoneal adhesions, a neurogenic bladder, or pelvic fractures/hematomas

Grade I



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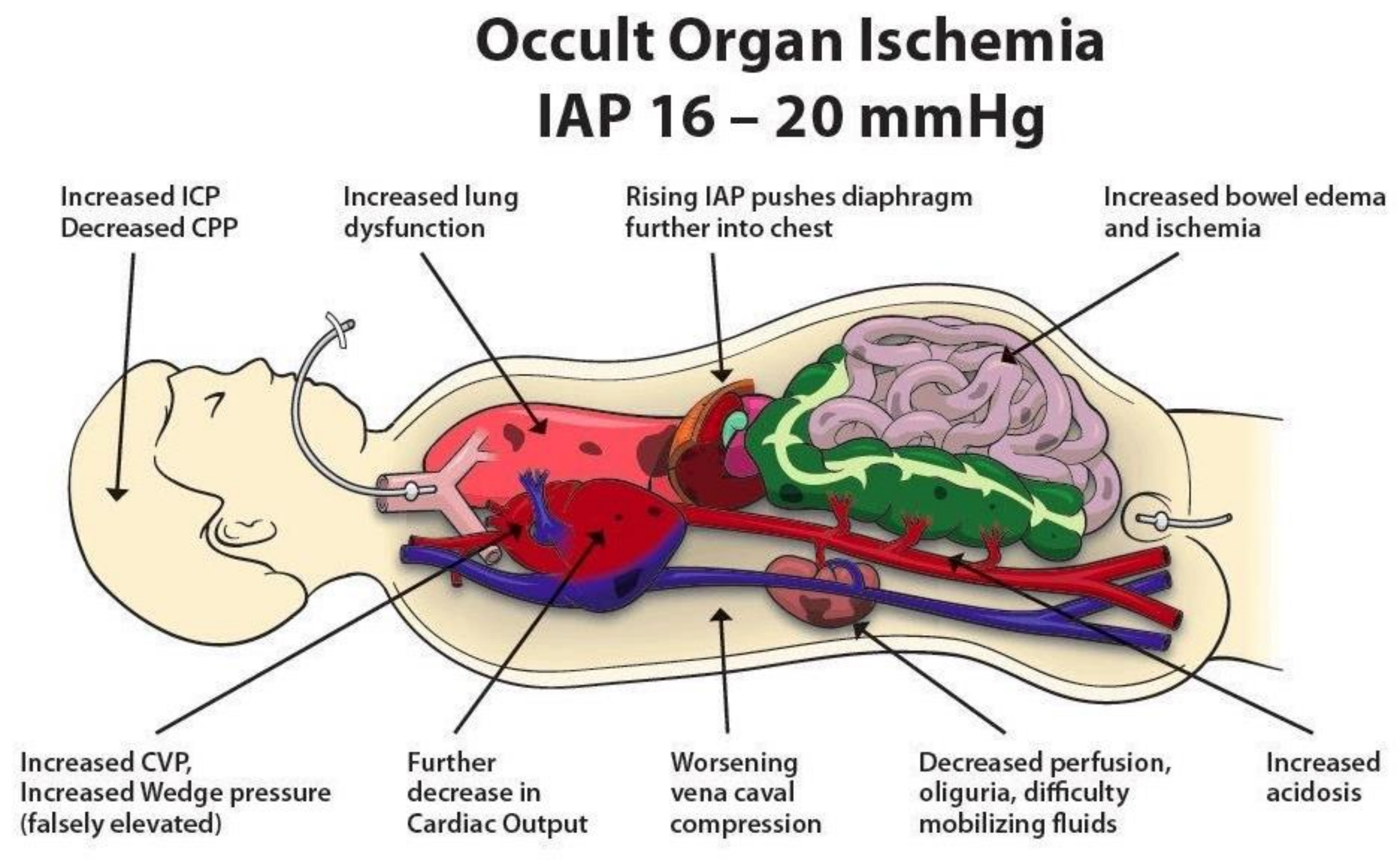
Grade II



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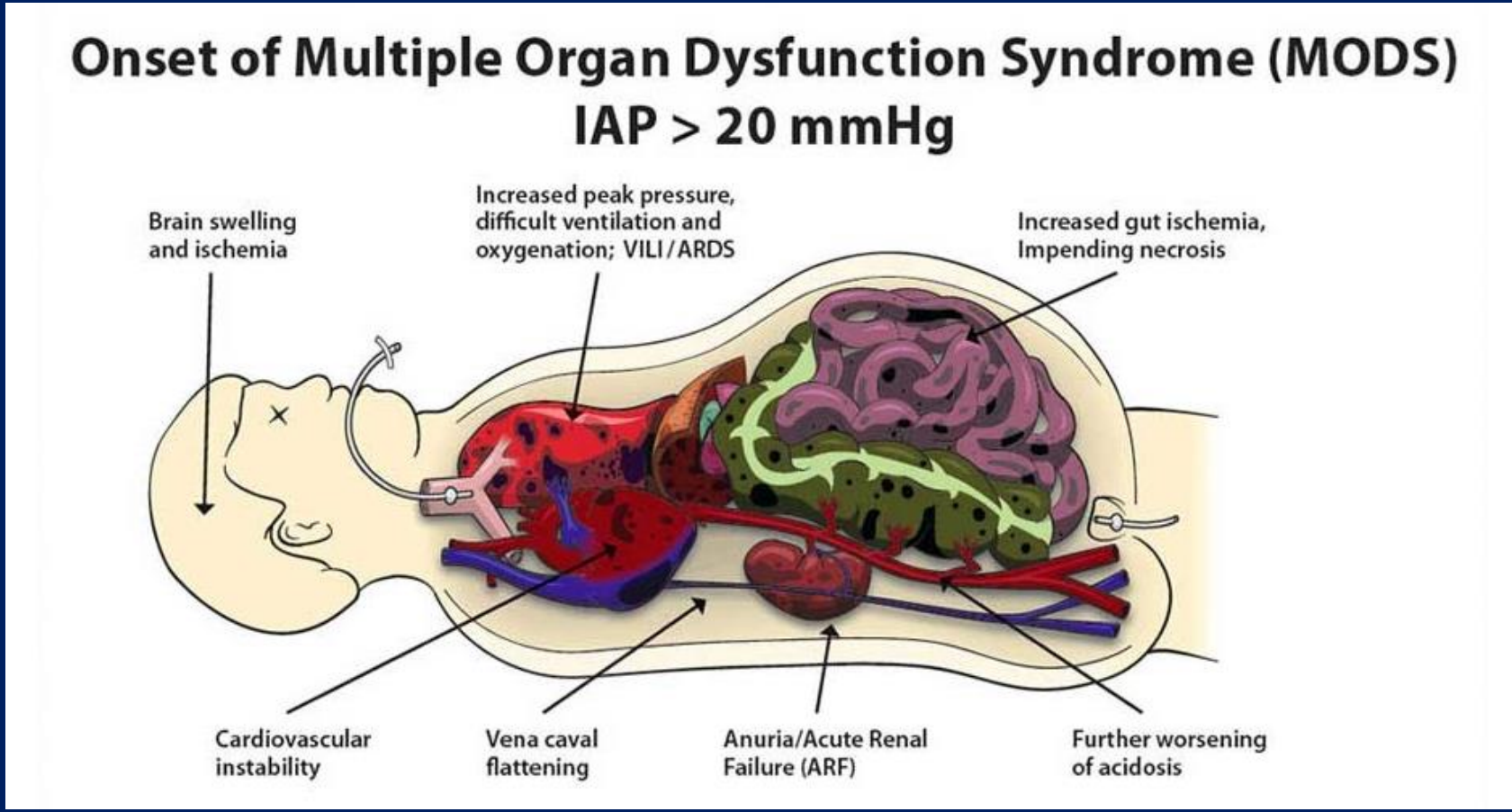
Grade III-IV



https://sites.google.com/a/learnpicu.com/learnpicu/gi/abdominal-compartment-syndrome

Treatment

Intensive Care Med (2013) 39:1190–1206
DOI 10.1007/s00134-013-2906-z

CONFERENCE REPORTS AND EXPERT PANEL

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Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome

- Supportive care including gastric and rectal decompression
- Percutaneous drainage of ascites or intraperitoneal hematomas
- Sedation and chemical paralysis to relax the abdominal musculature
- Ventilatory support as needed
- Hemodynamic support as needed
- **Surgical decompression** and usually maintenance of an open abdomen via a temporary abdominal wall closure is the definitive treatment

Update from the Abdominal Compartment Society (WSACS) on intra-abdominal hypertension and abdominal compartment syndrome: past, present, and future beyond Banff 2017

Authors: Andrew W. Kirkpatrick, Michael Sugrue, Jessica L. McKee, Bruno M. Pereira, Derek J. Roberts, Jan J. De Waele, Ari Leppaniemi, Janeth C. Ejike, Annika Reintam Blaser, Scott D'Amours, Bart De Keulenaer, Manu L.N.G. Malbrain

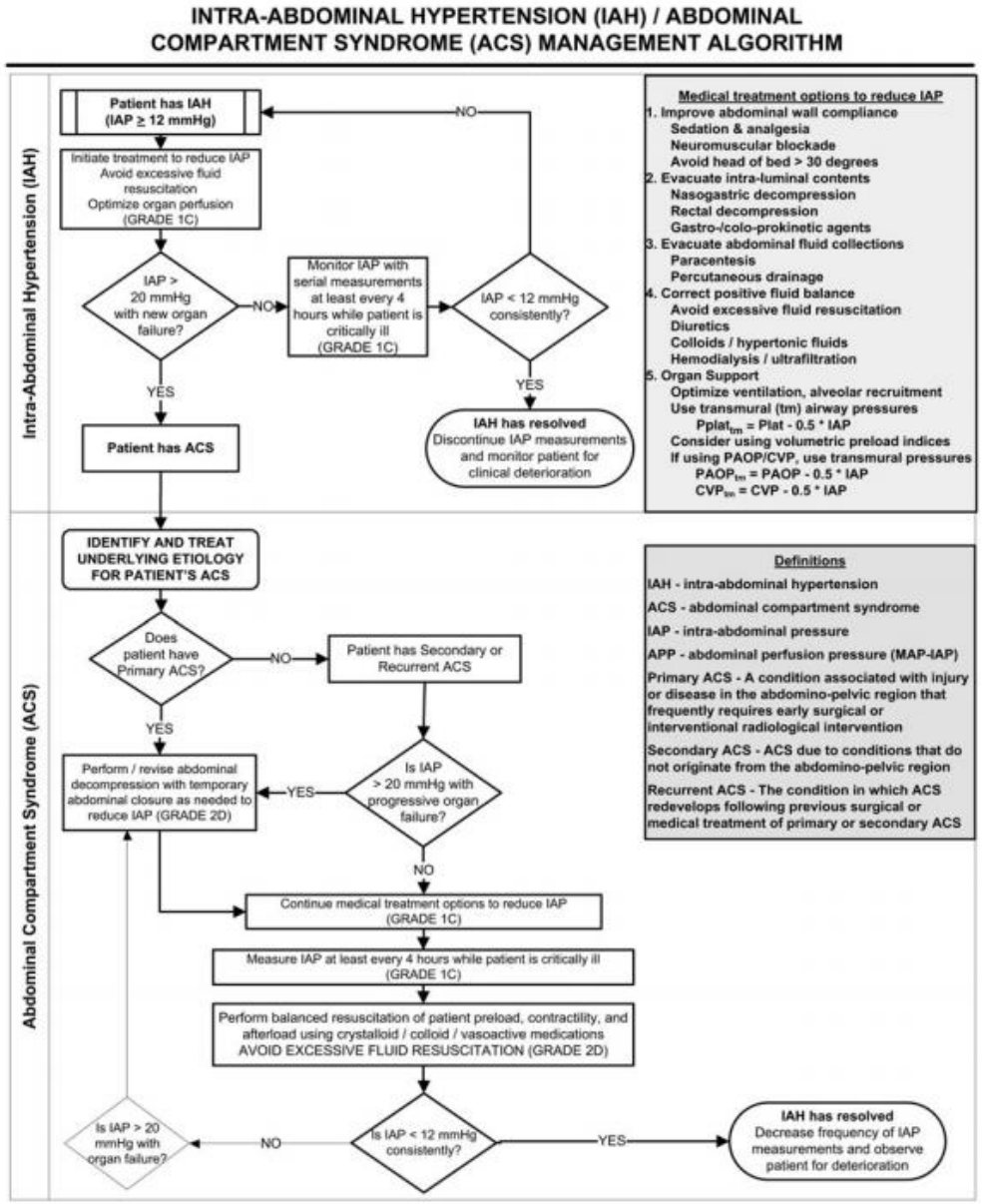


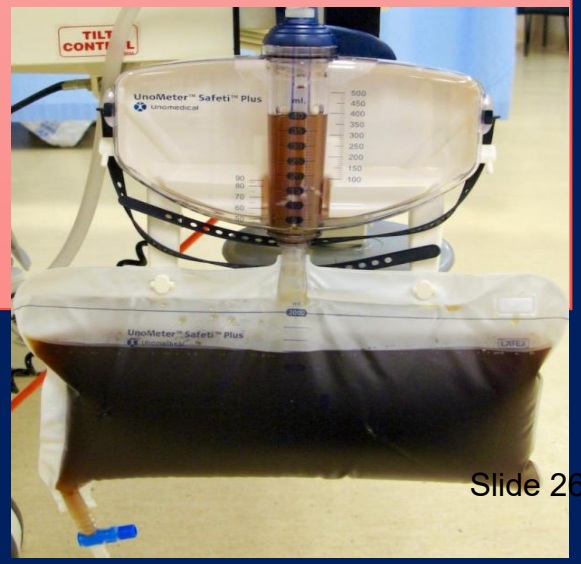
Fig. 1 Updated intra-abdominal hypertension (*IAH*)/abdominal compartment syndrome (*ACS*) management algorithm. *IAP* intra-abdominal pressure

Crush Syndrome

- Shock
- electrolyte problems
- renal dysfunction
- compartment syndrome

Rhabdomyolysis

- **Classic triad**
  - Myalgia
  - Generalized weakness
  - **Darkened** urine (red to brown)



Diagnosis and Management of Rhabdomyolysis in the Absence of Creatine Phosphokinase: A Medical Record Review

CPT Abhimanyu Chandel, MC USA; LT Kara Brusher, MC USAF†; LT Victoria Hall, MC USA†; Robin S. Howard, CIV‡; MAJ Paul A. Clark, MC USA§*

AST > 110 u/L****CPK >5000 u/L

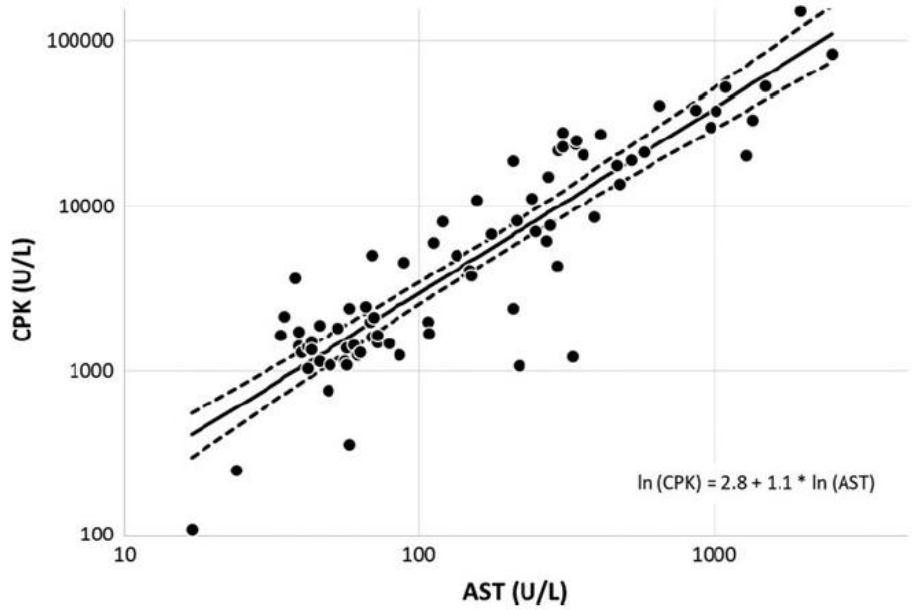
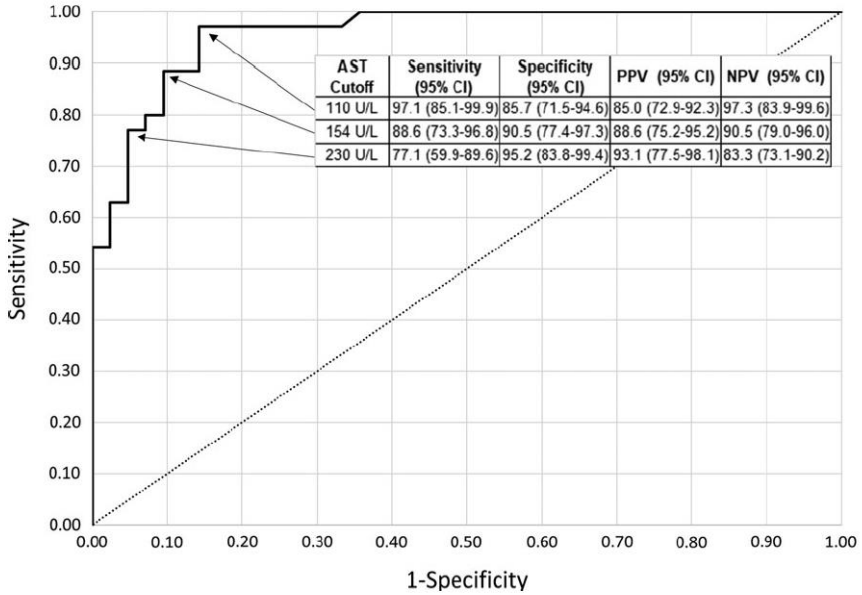


FIGURE 2. Day 1 creatine phosphokinase (CPK) vs. aspartate aminotransferase (AST) with linear regression model and associated formula.

Rhabdomyolysis in the Absence of Creatine Phosphokinase



AST Cutoff	Sensitivity (95% CI)	Specificity (95% CI)	PPV (95% CI)	NPV (95% CI)
110 U/L	97.1 (85.1-99.9)	85.7 (71.5-94.6)	85.0 (72.9-92.3)	97.3 (83.9-99.6)
154 U/L	88.6 (73.3-96.8)	90.5 (77.4-97.3)	88.6 (75.2-95.2)	90.5 (79.0-96.0)
230 U/L	77.1 (59.9-89.6)	95.2 (83.8-99.4)	93.1 (77.5-98.1)	83.3 (73.1-90.2)

FIGURE 3. Receiver operator characteristics curve for aspartate aminotransferase (AST) as predictor of day 1 creatine phosphokinase (CPK) ≥5,000 U/L. Also shown are sensitivity, specificity, and predictive values at defined AST cutoffs.

Take home message

1.Life salvage

2.Limb salvage

3.Salvage of total function if possible

“ To Restore the patient back to his pre-injury status ”

TEAM

THANK YOU