Shock & resuscitation

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What is Shock?

Condition of inadequate delivery of oxygen and nutrients necessary for normal tissue and cellular function

In other words, blood flow (pressure) and oxygen delivery to the body is too low

An Approach to Shock

$$BP = CO \times SVR$$

BP = blood pressure

CO = cardiac output

SVR = systemic vascular resistance

An Approach to Shock

If the blood pressure is low, then either the:

CO is low

or

the SVR is low

Low SVR

There are only a few causes of low SVR.

They ALL cause vasodilation:

- sepsis
- acute spinal cord injury (spinal, epidural)
- vasodilators (NTG, anesthetics)
- anaphylaxis

How do you assess SVR?

Look at and feel the patient!

Low SVR has the features:

- warm !!!
- pink (may also a rash)
- hyperdynamic heart (fast and pounding)

What if the SVR is high?

- patient will have cool or cold arms/legs
- patient will NOT look pink

Cause of shock or low BP is then:

low CO

What are factors of CO?

$$CO = HR \times SV$$

CO = cardiac output

HR = heart rate

SV = stroke volume

HR Problems

- HR problems are easy to diagnose

Low SV (stroke volume)

Most common cause of shock

but

Most difficult to diagnose and manage

Factors of SV

Preload: is the ventricle full?

Contractility: how well does the ventricle contract

Valve function: normal?

regurgitation?

stenosis?

Stroke Volume

Which factors can we influence?

- Preload and contractility

We cannot change valve function

Summary

Perfusion (blood pressure) depends on:

BP = CO x SVR

CO = HR x SV

SV = preload
contractility
valves

Types of Shock

- Hypovolemic
- Inflammatory
- Compressive
- Neurogenic
- Cardiogenic

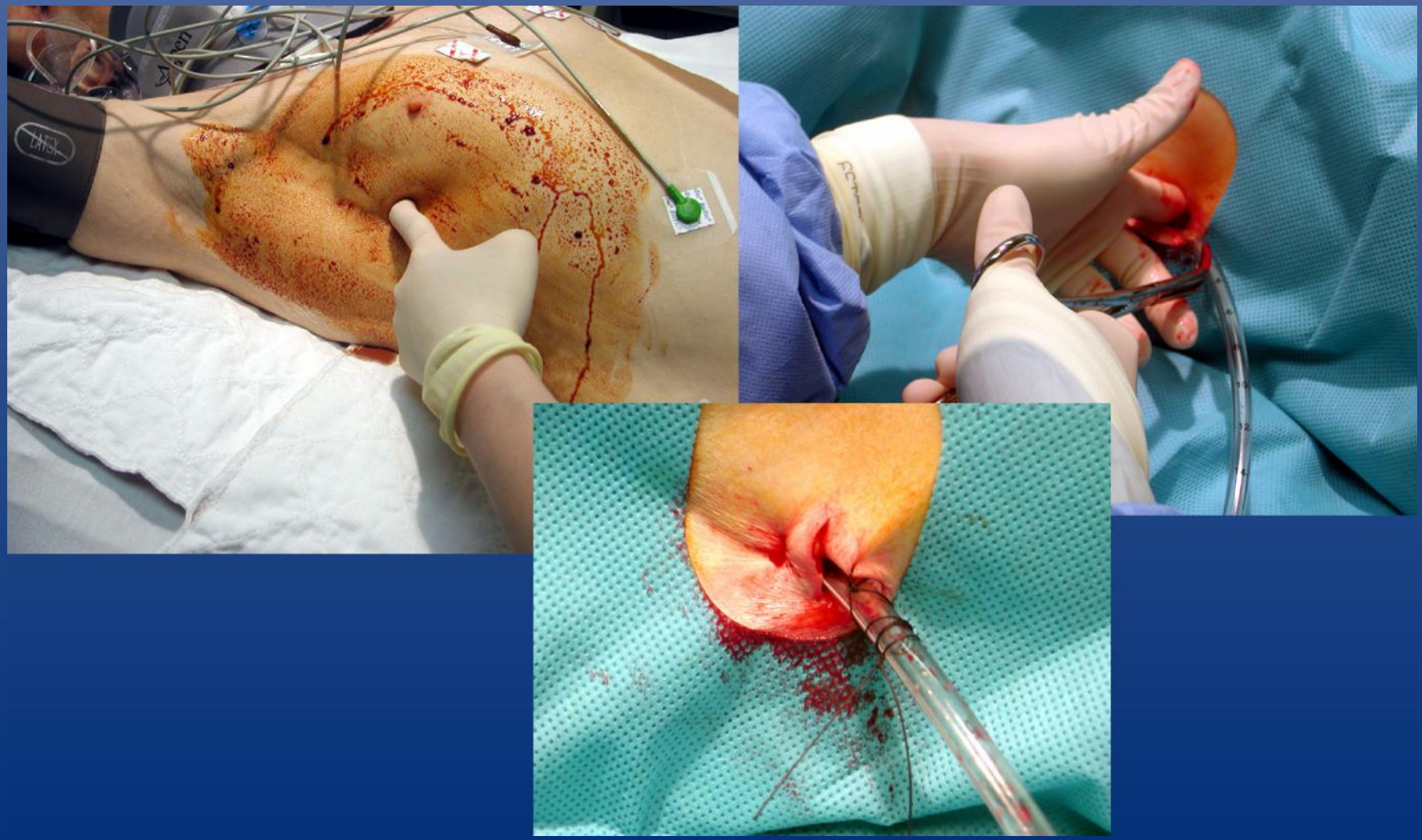
- 41M assaulted in street, stabbed in L side of chest.
- VS 90/60, HR 130, RR 40s, O2 Sat 85%
- Decrease BS on Left, Left JVD, tracheal shift toward right

Compressive Shock

- External forces compress the chambers of the heart or great veins (VC) resulting in decreased cardiac filling / cardiac output.

- Mechanism of Tension Pneumothorax?

Tension Pneumothorax

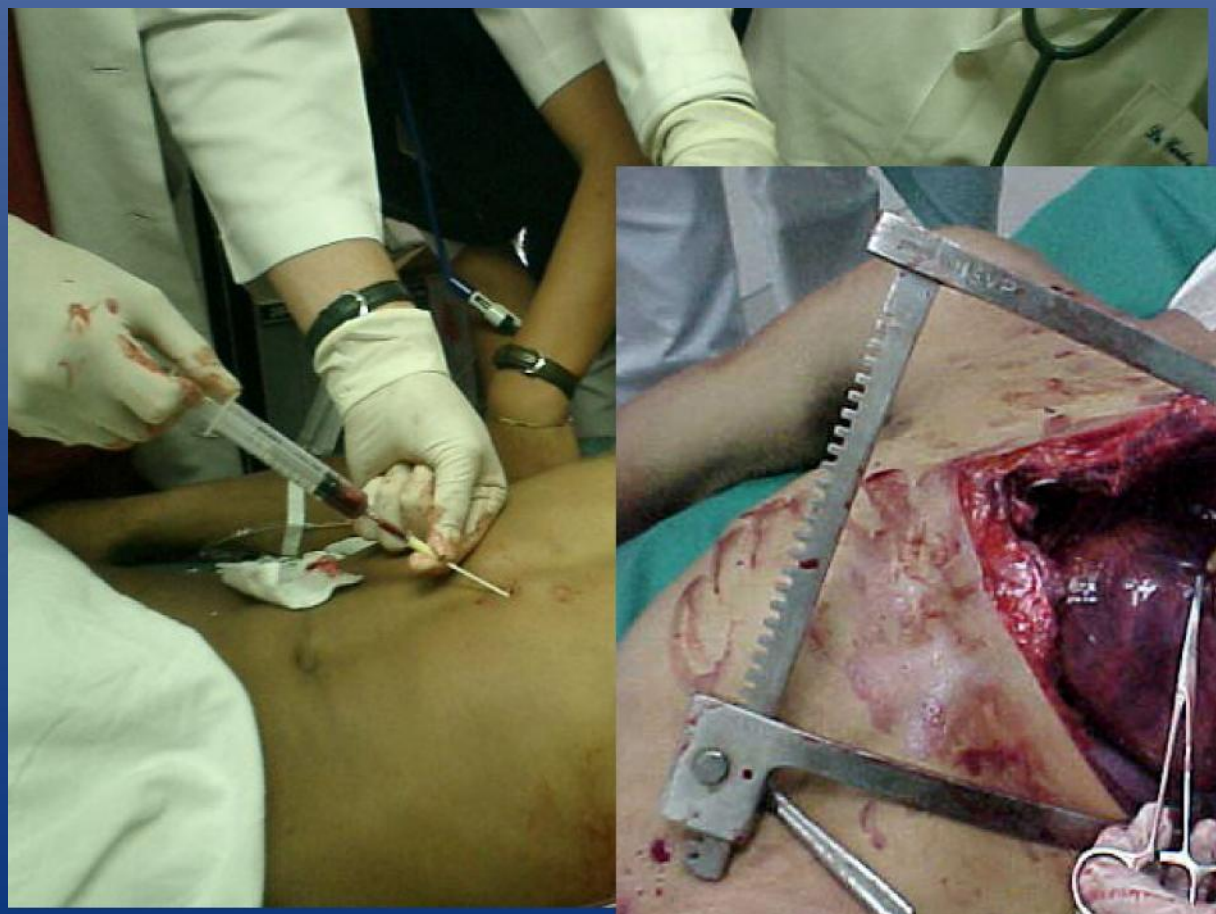


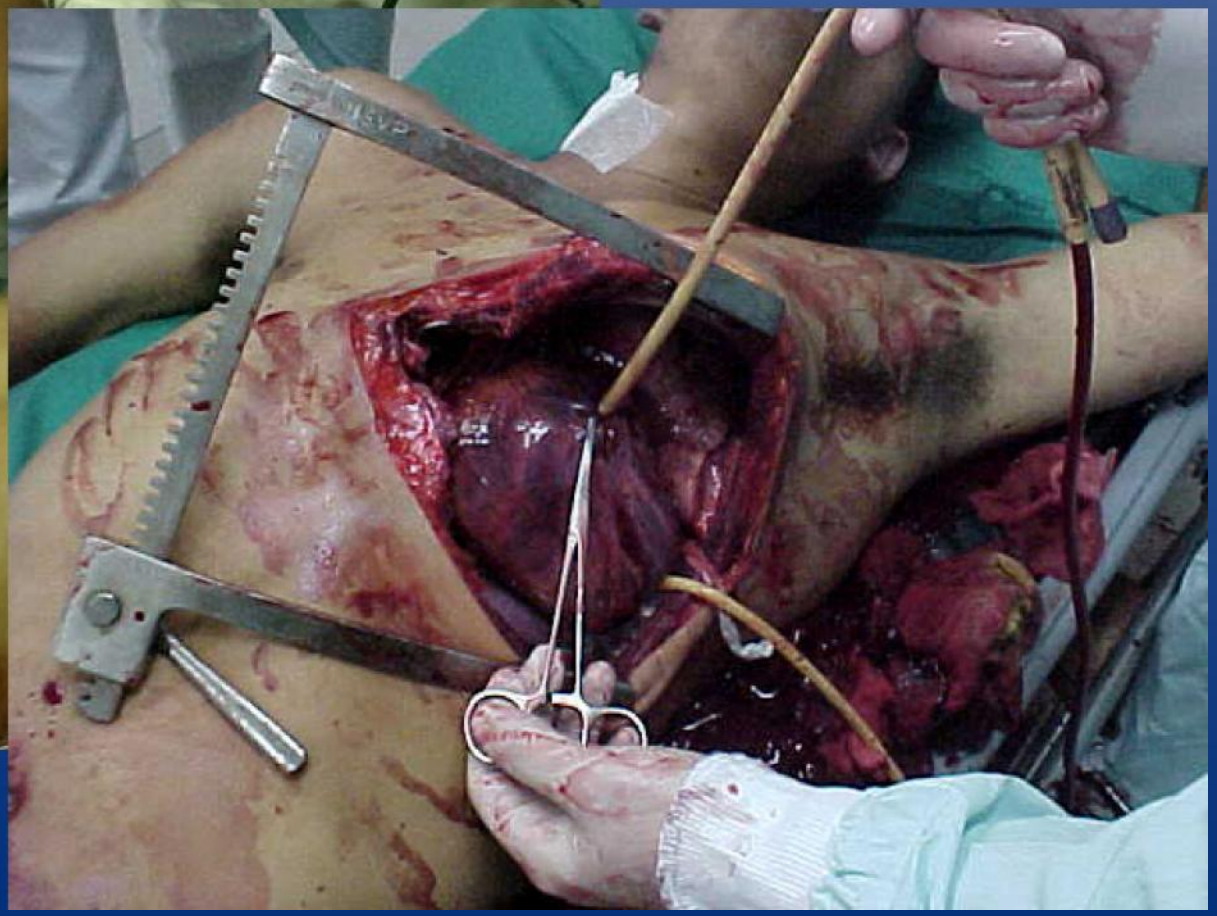
Other Method of Compressive Shock?

Pericardial Tamponade

- Compression by fluid in pericardial sac obstructs contraction by heart
- Sx: Beck's Triad

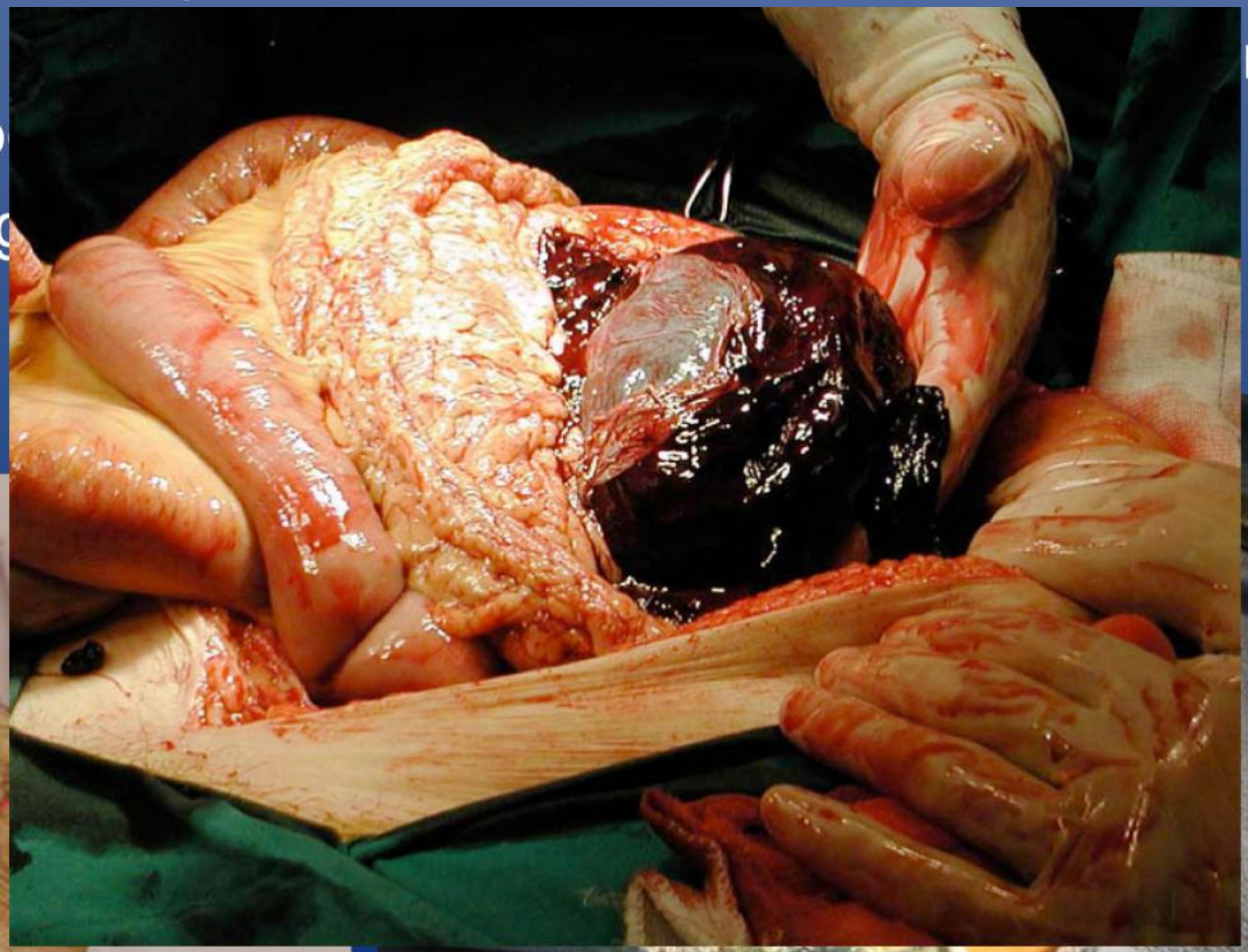
Pericardial Tamponade





- 22M s/p
  flipped re
- VS HR
- Unrespo
- Chest / A d,
  tender di

- Initial CXR negative
- Despite ment in hemo
- DPL - g



Hypovolemic shock

- Acute blood loss in trauma patient results in loss of circulatory volume
  - SNS stimulation, Release of epinephrine and norepinephrine
  - Vasopressin release
  - Activation of Renin angiotensin cascade
  - Vasosconstriction to maintain cerebral and coronary blood flow
- Shock in a trauma patient should be presumed to be due to hemorrhage until proven otherwise

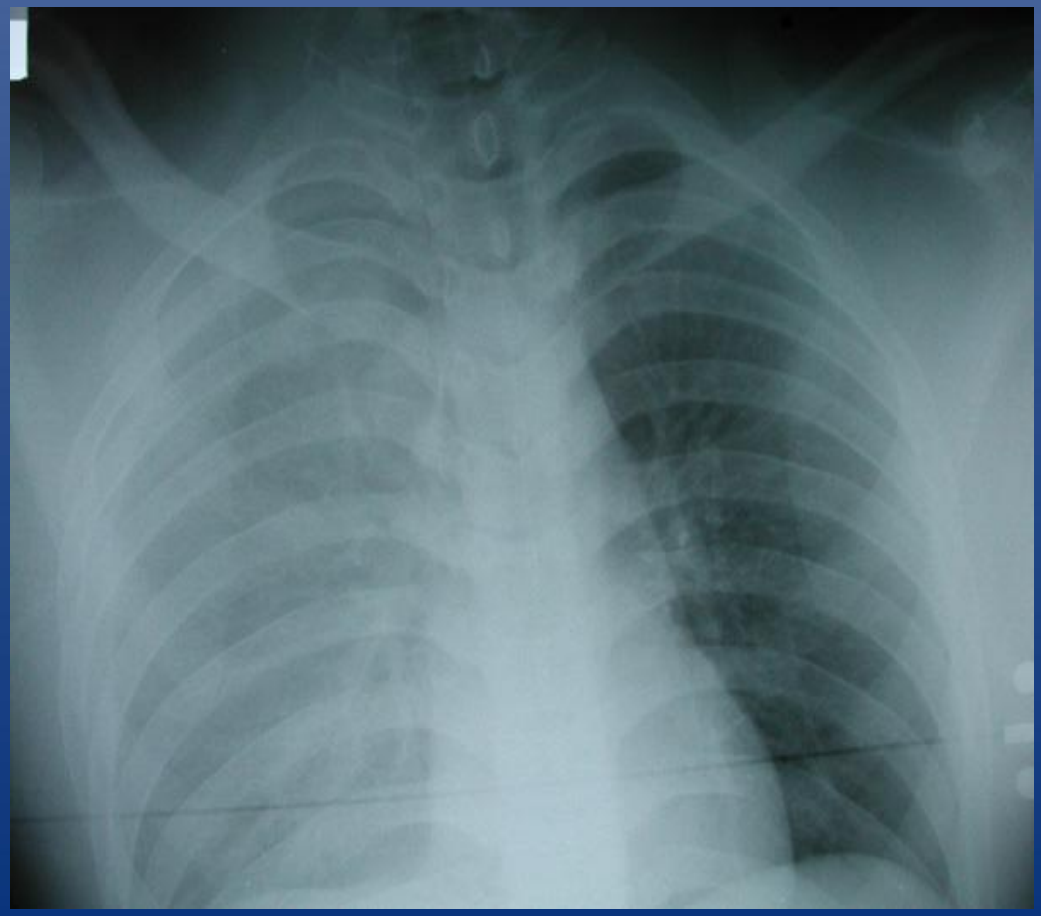
Table 3

Classification of Hemorrhagic Shock and Associated Physiologic Changes

	Class I	Class II	Class III	Class IV
Blood loss (cc)	Up to 750 cc	750-1500 cc	1500-2000 cc	>2000 cc
% Volume loss	Up to 15%	15%-30%	30%-40%	>40%
Pulse rate	<100	>100	>120	>140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate	14-20	20-30	30-40	>35
Urine output (cc/hr)	>30	20-30	5-15	Negligible
Mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic
Fluid replacement	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and blood

Sources of Bleeding

- External
- Thoracic - CXR
- Intraperitoneal
  - DPL
  - FAST
  - CT
- Retroperitoneal
  - Pelvic Fractures
  - CT, angiography



Initial Resuscitation in Hypovolemic Shock

extremities

Initial Fluid Resuscitation

- 1-2 L bolus Isotonic Fluid (Formula?)
  - Repeat x 2
- Normal Saline (.9% NaCl)
  - Isotonic
  - Hi chloride
  - Hyperchloremic acidosis
- Lactated Ringers
  - Lactate and acetate to buffer acidemia which occurs w/ shock state
- Need __? volume of resuscitation crystalloid to compensate for same volume of blood loss

Colloids

- Albumin or Starch
- Expand intravascular volume much more than crystalloid
- Theoretical advatange that would require less volume of resuscitation
- No clear evidence that is better than crystalloids
- Much less available

Hypertonic Saline

- 7.5% Saline Solution
- Pull intracellular water out into intravascular space
- Much better blood volume expansion
- Some evidence of favorable effects on inflammatory response to injury
- Less cellular edema associated w/ damage
- May be good for situations where large volume resuscitation is not available (ie mass casualties, combat)

Tranfusion

- Data suggests limiting transfusions in critically ill
  - Poorer overall outcome
  - Immunosuppresses?
  - May be true in euvolemic ICU patient
- But in SHOCK…..first priority is to restore intravascular volume

ACS guidelines

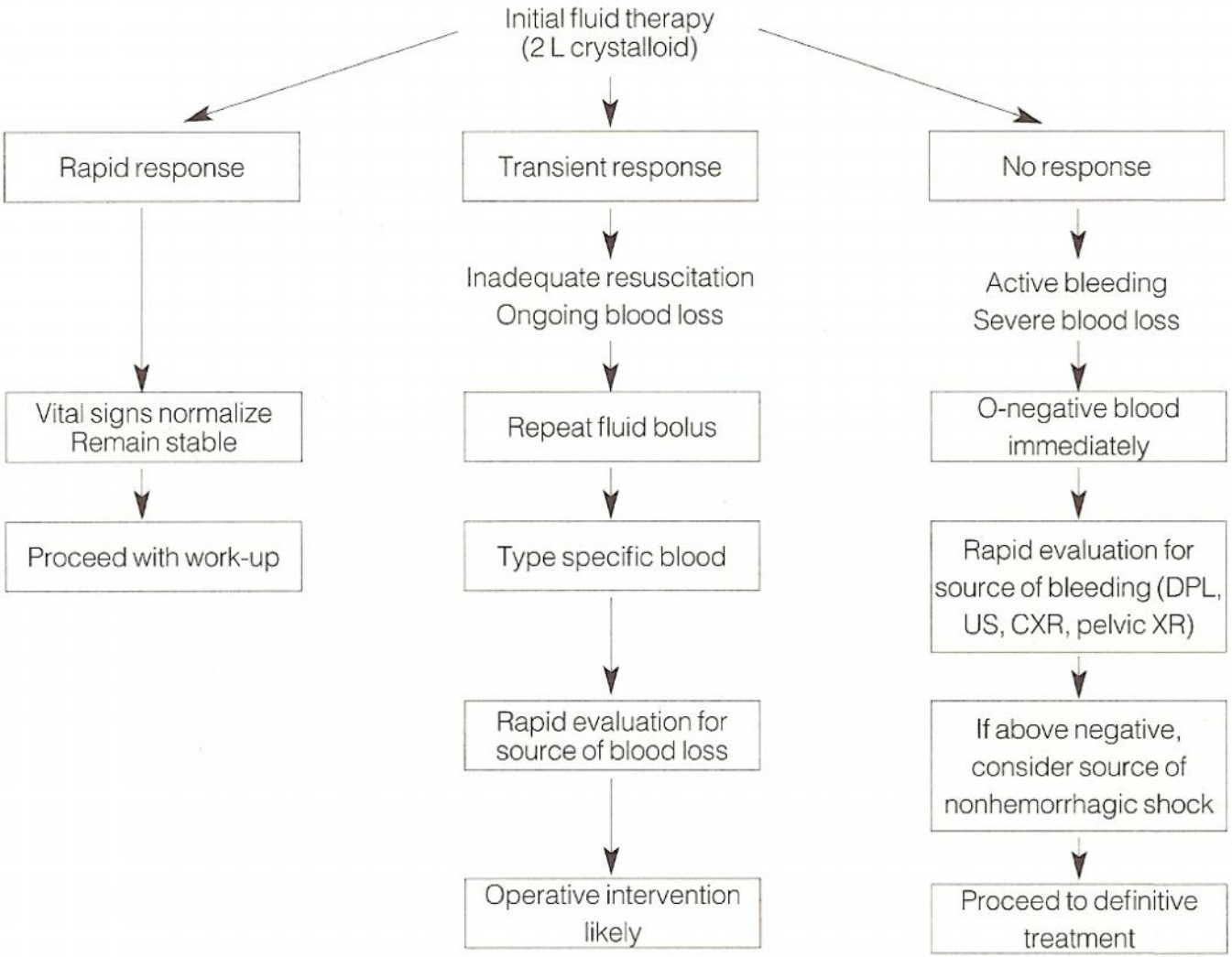
- **Hb 7 g/dl** is adequate in a young patient (No CAD, bleeding controlled)
- **Hb 8 g/dl** is adequate in a young patient who may be at slight risk for further bleeding.
- **Hb 9 g/dl** is required if the risk of bleeding is substantial.
- **Hb 10 g/dl** should be the goal if overt ischemia is present or there is a significant risk of occult ischemic disease (peripheral vascular disease, CAD)

Transfusion

- Emergency: O(-) blood
- Type Specific
- Type and Crossmatched
- Future of blood substitutes?

Figure 1

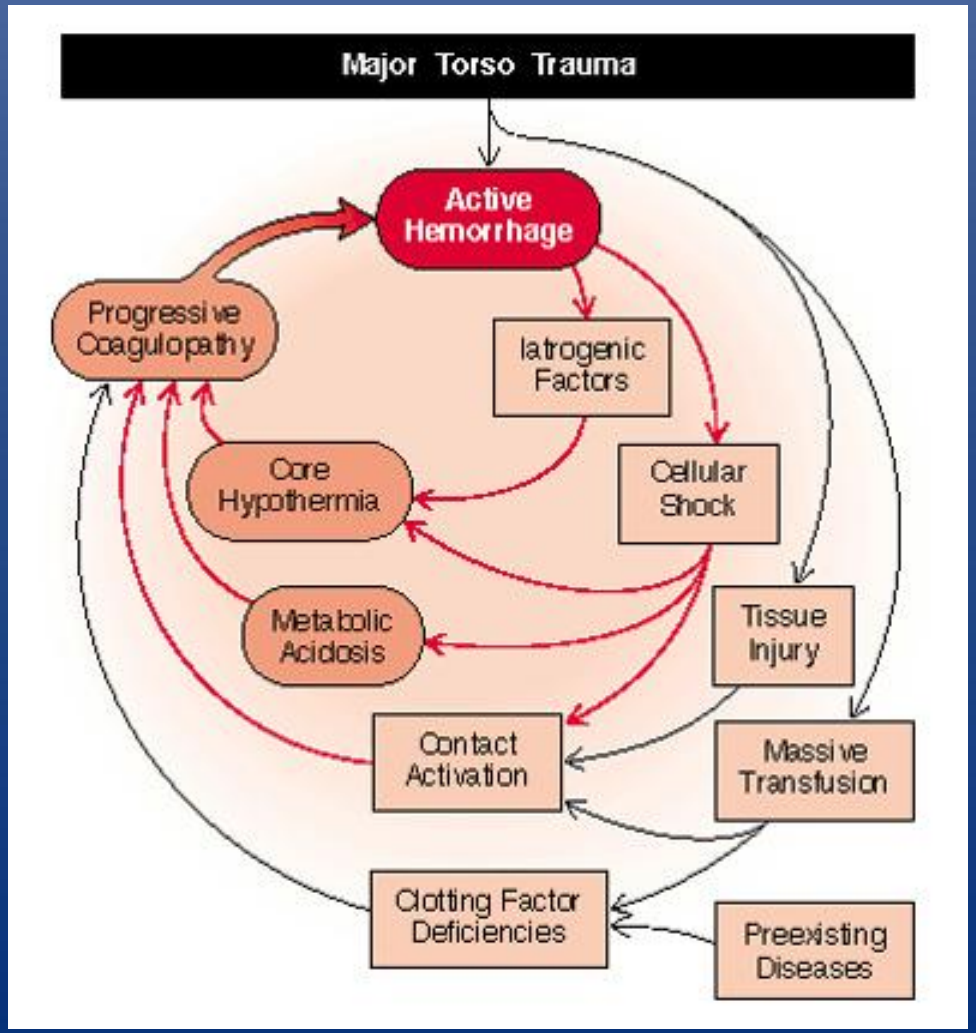
Algorithm for fluid resuscitation and management of shock



Irreversible shock

- Ongoing fluid / blood requirement despite control of hemorrhage
- Persistent hypotension despite restoration of intravascular volume

- Futile cycle of uncorrectable hypothermia, hypoperfusion, acidosis, coagulopathy
- Inevitably terminal



- 74F BIBA found passed out in Hot summer apartment, lethargic, febrile, delerious
- CVA tenderness, Dirty UA, Cloudy urine
- Fever 102, HR 110, BP 80/50, RR 20
- WBC 24

Septic Shock

By product of body's response to infection.

- In attempt to eradicate pathogens, reticuloendothelial system releases cytokines which modulate inflammatory cells fxn.
- Increase in microvascular flow enhances delivery of killing forces to areas of infection.
- Sepsis occurs when this occurs systemically – hemodynamic collapse.

Signs: **Early**– warm w/ vasodilation, often adequate urine output, febrile, tachypneic.
Late-- vasoconstriction, hypotension, oliguria, altered mental status.

- SVR is low – inflammatory mediators cause increased permeability in tissues

Treatment

- Antibiotics
- Resuscitation w/ crystalloid
- Operative drainage of infected collections
- Inotropes

- 19M diving into pool, hit head at bottom
- Grasping neck and head when rescued
- BIBA limp, sensory deficits
- VS HR 50, BP 80/40

Neurogenic Shock

- Diminished tissue perfusion d/t loss of vasomotor tone to peripheral arterial beds
- Cspine or Hi Tspine injuries disrupt sympathetic regulation of vascular tone
- No sympathetic imput to heart to increase HR and contractility, No catecholamine release

Initial Treatment

- Secure airway
- Fluid resuscitation
- Vasoconstrictors to improve vascular tone

Cardiogenic Shock

- Circulatory pump failure resulting in tissue hypoxia
  - Direct myocardial contusion
  - Valvular injury
  - Pre-existing cardiac disease + trauma

Decrease cardiac output in face of adequate intravascular volume

=> Heart Failure

Diagnosis

- Measure Cardiac Output by Swan Ganz Catheter
- Transesophageal echocardiography

- Hi CVP, Wedge Pressure, Low CO

Treatment

- Maintain intravascular volume, but do not overload (Heart failure, pulmonary edema)
- Inotrophic support
  - Dobutamine
  - Dopamine
  - Epinephrine
- Intra-aortic balloon pump

Hemodynamic profiles

	PCWP	CVP	CO/CI	SVR/I
Hypovolemic	Low	Low	Low	High
Cardiogenic	High	High	Low	High
Inflammatory	Low / N	Low/N	High	Low
Neurogenic	Low	Low	Low	Low

The End!