شوک

تعریف ،ارزیابی و درمان

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متخصص بیهوشی و مراقبت های ویژه

استادیار دانشگاه علوم پزشکی دزفول

پاییز ۱۴۰۱

تعریف شوک :

کاهش پرفیوژن بافتی پایین تر از مقداری که بتواند نیازهای بافتی را برطرف کند . پس در یک کلام شوک یعنی :

INADEQUATE PERFUSION

CAUSES OF SHOCK:

- **LV failure ("cardiogenic" shock):**
  - LV systolic failure (e.g. MI, myocarditis, beta-blocker overdose)
  - Acute aortic or mitral valve regurgitation (e.g. endocarditis, papillary muscle rupture, aortic dissection)
- **RV failure:**
  - Pulmonary embolism
  - Decompensated chronic pulmonary hypertension
  - Right ventricular myocardial infarction

CAUSES OF SHOCK:

- **Arrhythmic shock:**
  - Tachyarrhythmia (usually >> 150 b/m)
  - Bradyarrhythmia (usually < 45 b/m)
- **Hypovolemic shock:**
  - Hemorrhage (external, GI bleed, retroperitoneal bleed, intraperitoneal bleed, hemothorax, post-partum).
  - Hypovolemic (e.g., vomiting, diarrhea, over-diuresis, post-ATN or postobstructive polyuria).

CAUSES OF SHOCK:

- **Obstructive:**
  - Tension pneumothorax
  - Tamponade
  - Abdominal compartment syndrome
  - AutoPEEP or high mean airway pressures

CAUSES OF SHOCK:

- **Vasodilatory shock ("distributive" shock):**
  - Sepsis
  - Severe systemic inflammation (e.g. pancreatitis, post-cardiac arrest, post-MI)
  - Anaphylaxis
  - Adrenal crisis, thyroid storm
  - Neurogenic shock (severe CNS/spinal trauma, spinal anesthesia)
  - Liver failure
  - Excess vasodilatory drugs

CELLULAR RESPONSE TO SHOCK:

Blood Loss

Inadequate Perfusion

Cellular Hypoxia

Aerobic Metabolism

Anaerobic Metabolism

Lactic Acid

Cellular Edema

Acidosis

NORMAL

VITALS DO NOT

R/O OCCULT HYPO

PERFUSION

bedside approach to undifferentiated shock/hypotension

	IVC or Jugular vein size	Lung POCUS	Significant pericardial effusion	RV dilation	LVEF	Mitral or aortic regurg?	Cardiac Output
Distributive shock - Septic shock - Anaphylaxis* - Adrenal crisis, Thyroid storm - Post-cardiac arrest SIRS - Pancreatitis, Hepatic failure - Neurogenic (trauma, spinal anesth) - Vasodilatory medications	↓↓ or normal	Normal (or focal abnormality from pneumonia)	–	–	nl / ↑	–	**High output:** - Warm extremities. - Low diastolic Bp. - Wide pulse pressure. - May be febrile. - May look toxic. - Capillary refill is variable.
Hypovolemic shock - Vomiting, diarrhea, overdiuresis - Hemorrhage (GI, peritoneal, RP) **Abdominal compartm. syndrome**	↓↓↓	Normal	–	–	nl / ↑	–	**Low output** - Cool extremities. - Diastolic Bp may be normal. - Narrow pulse pressure. - Delayed capillary refill.
RV failure - PE, - Decompensated chronic PH - RV myocardial infarction	↑↑	Normal	–	+	nl / ↑	–	
Tamponade	↑	Normal	+	–	nl / ↑	–	
Tension pneumothorax	↑	No slide on affected hemithorax	–	–	nl / ↑	–	
AutoPEEP / high airway pressure - Asthma > COPD > ARDS - Exacerbated by hypovolemia - Dx based on history, vent waves	nl/↑	Normal	–	+/–	nl / ↑	–	
LV failure - MI - Myocarditis, postpartum CM - Takotsubo cardiomyopathy - Beta-blocker overdose	nl/↑	B-lines everywhere	–	+/–	↓↓	+/–	
Valve dysfunction - Endocarditis - Post-MI papillary muscle rupture - Prosthetic valve dysfunction **LV outflow obstruction (LVOTO)** - Elderly with chronic HTN - HOCM or Takotsubo CM	nl/↑	B-lines everywhere	–	–	nl / ↑	+	

SHOCK IS EXTRAORDINARILY IMPORTANT BECAUSE IT IS GENERALLY A FINAL COMMON PATHWAY BEFORE DEATH.

HOWEVER, SHOCK IS OFTEN REVERSIBLE.

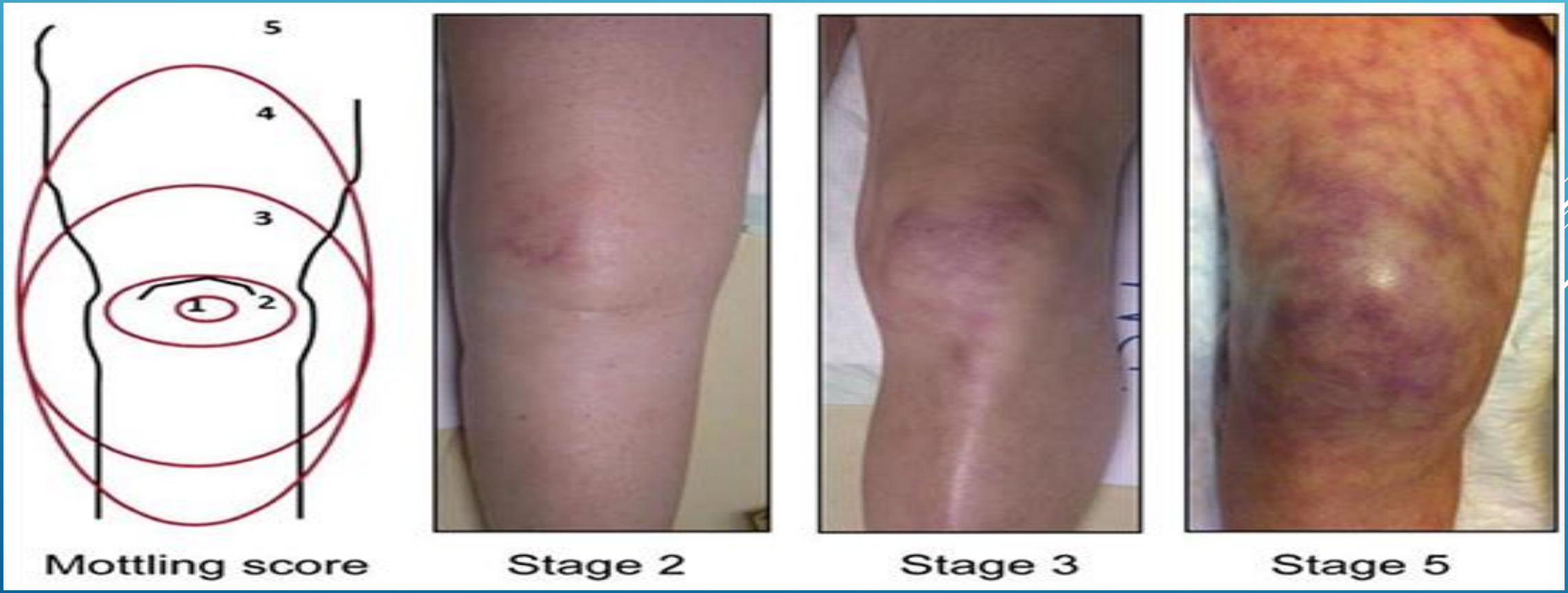
CLASSIC SIGNS & SYMPTOMS OF SHOCK:

- Changing mentation:
- New-onset delirium can be a sign of shock. However, this is neither very sensitive nor specific. Most new-onset delirium isn't due to shock. Furthermore, patients with cardiogenic shock often maintain normal mentation (delirium tends to be a feature of *septic shock* rather than of cardiogenic shock).
- Tachycardia
- Cool, clammy skin:
- are an early sign of vasoconstriction with reduced cardiac output. Normal people may have cool hands, but if *all* extremities are cool that's more specific for hypoperfusion

CLASSIC SIGNS & SYMPTOMS OF SHOCK:

- Prolonged capillary refill
- Narrowed pulse pressure
- Decreased urine output:
- urine output below 0.5 cc/kg/hr is worrisome for renal malperfusion. Immediately following Foley catheter placement the urine output won't be known – in this situation scanty and dark urine is worrisome
- Hypotension:
- MAP<65 and/or significant drop from baseline

- **Mottling** is less sensitive, but more specific for hypoperfusion and elevated mortality (figure below). Mottling suggests active endogenous vasoconstriction, implying that the patient would benefit from an increase in cardiac output (e.g. an inotroph) – not additional exogenous vasoconstrictors.



Approach to undifferentiated shock/hypotension

Investigations to consider

Data review
- Vital sign & lab trends.
- Recent procedures or medication changes?
- Relevant history (e.g. heart failure, adrenal dysfunx).

Bedside exam including:
- General appearance & skin perfusion.
- POCUS of heart & lungs.
- POCUS scan for DVT if PE is a concern.
- Abdominal exam (tender? tense? Consider FAST).

Labs, potentially including:
- Basic labs (chemistries, CBC, coags, liver function tests).
- Lactate.
- If infection suspected: cultures, urinalysis.
- If infected suspected & ABX started: procalcitonin.
- If hemorrhage possible: type & cross-match blood.
- If adrenal insufficiency possible: cortisol level.
- If thyroid storm suspected: TSH & free T4.

Radiologic studies
- EKG.
- Chest X-ray (? Evidence of heart failure or PNA).
- Formal echocardiogram, if POCUS is equivocal.
- CT scan depending on scenario, for example:
  - CT angiogram if PE suspected.
  - CT abdomen/pelvis to look for septic focus.
  - CT angiogram abdomen to look for hemorrhage.

Assessment vs. Resuscitation Endpoints

Traditional vs. New
Acute vs. Ongoing
Static vs. Dynamic
Global vs. End Organ

Initial Assessment

- Mentation
- Skin Perfusion
- Pulse
- Blood Pressure
- Pulse Pressure
- Shock Index
- Urine Output

Resuscitation Endpoints

pH
Serum Lactate
Base Deficit
Echocardiography
Arterial Wave Analsyis
StO2 (NIRS)

SHOCK INDEX (SI)

- SI = HR / SBP
- Elevated early in shock
- Normal 0.5 - 0.7
- SI > 0.9 predicts:
  - Acute hypovolemia in presence of normal HR & BP
  - Marker of injury severity & mortality
- Caution in Geriatrics
  - May underestimate shock due to higher baseline SBP
- Uses
  - Prehospital use → triage
  - Predict risk for mass transfusion?

BASE DEFICIT:

- Sensitive measure of inadequate perfusion
- Normal range -3 to +3
- Run on blood gases
- Admission BD correlates to blood loss
- Worsening BD:
  - Ongoing bleeding
  - Inadequate volume replacement

BASE DEFICIT CLASSIFICATION

Category	Base Deficit	Mortality
Mild	< 5	11%
Moderate	6-9	23
Severe	10-15	44%
	16-20	53%
	>20	70%

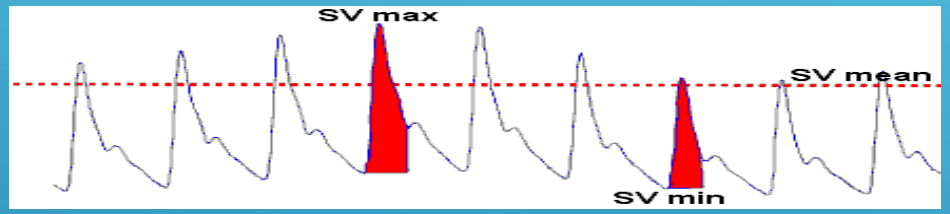
DOPPLER ECHOCARDIOGRAPHY (TRANSTHORACIC OR TRANSESOPHAGEAL)

- Allows for physician bedside assessment:
  - Ventricular function
  - Volume status
  - Stroke volume
  - Cardiac output

- Dependent on:
  - Technology investment
  - Technical expertise
  - Intra-observer variability
- Excellent diagnostic tool
- Poor monitoring device

ARTERIAL PRESSURE WAVEFORM SYSTEMS

- Measures pulse pressure & stroke volume *variation*

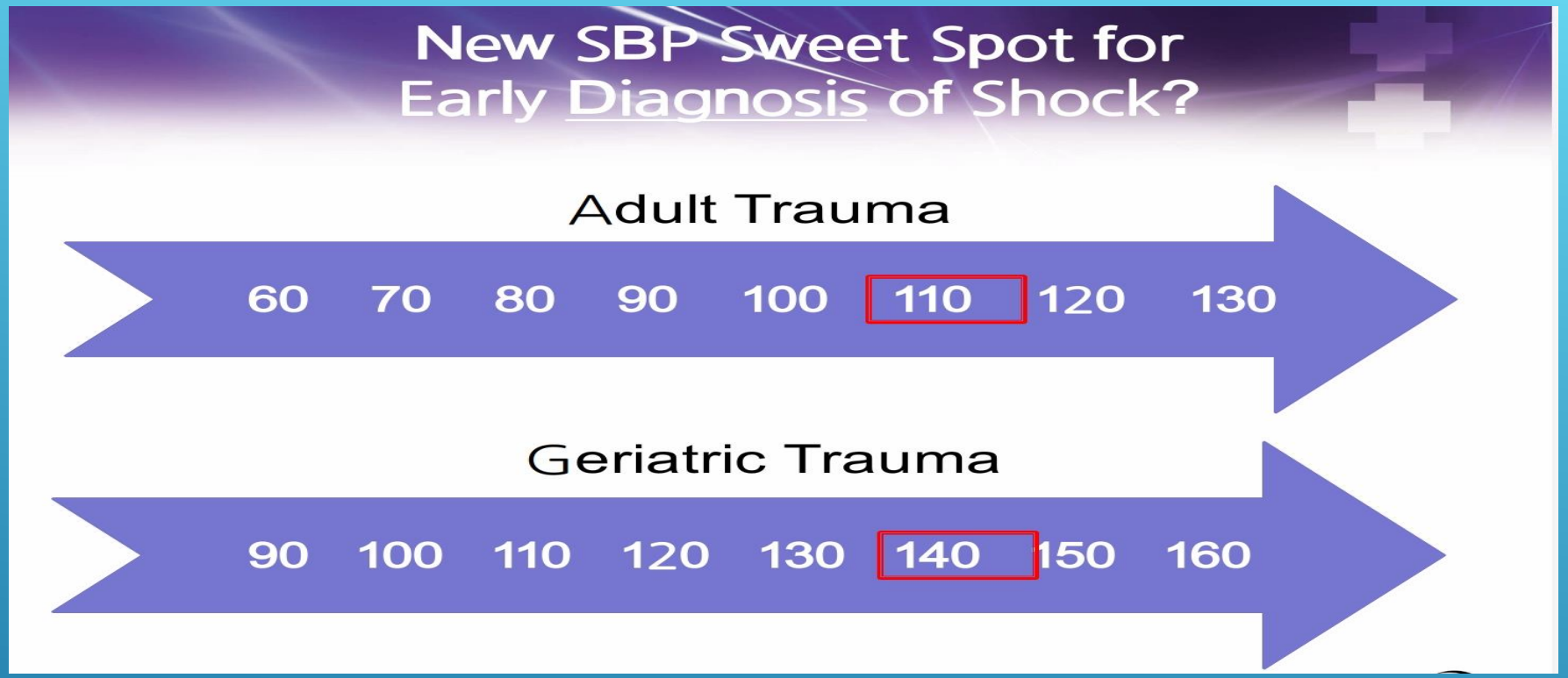


- Reliable predictors of volume responsiveness
- Determines where the patient lies on their own individual Starling curve

Examples of systems:
PiCCO (Phillips)
pulseCO (LiDCO,Ltd.)
FloTrac/Vigileo (Edwards)

نکات مهم در ارزیابی بیماران با شوک :

- هوشیاری بیمار یک معرف مقدار پرفیوزن مغزی است که می تواند با مصرف الکل و داروها تحت تاثیر قرار بگیرد .
- تروما به سر و هیپوکسی نیز میتواند این نمایه را تحت تاثیر قرار دهد .
- افت فشار یک علامت تاخیری است و دیر رخ می دهد .(در بالغین با 30 درصد از دست دادن خون در شوک هموراژیک)
- فشار سیستولیک کمتر از 90 میلی متر جیوه یعنی 65% مرگ ومیر
- فشار سیستولیک زیر 110 یعنی شروع تغییرات فیزیولوزیک
- فشار پالس باریکتر یعنی افزایش فعالیت کاتکولامینها



نکته: با افزایش سن در بزرگسالان و افراد مسن فشار سیستولیک مناسب جهت کاهش مرتالیتی افزایش مییابد .

Stabilization

پایدار کردن

Stabilization must start immediately,

often before the cause of shock is known.

STABILIZATION:

▸volume resuscitation:

- Often advisable, with the following exceptions:
  - Patients with **left ventricular function failure and pulmonary edema**.
  - . **The total amount of fluid administered should generally be limited to <1-2 liters** in the absence of a history suggesting substantial total-body volume depletion (e.g., severe gastroenteritis with a colostomy).
- **Fluid administration can be diagnostic and therapeutic in confusing situations where hypovolemia is suspected:**
  - If fluid resuscitation alone resolves shock, this supports a diagnosis of hypovolemia.
  - If fluid resuscitation fails, this suggests an alternative diagnosis. This is especially true if fluid resuscitation results in adequate filling pressures (e.g., full IVC) *without* resolving the shock.

STABILIZATION:

- **vasopressor administration:**

- should be started immediately if the blood pressure is inadequate (e.g. MAP<65 mm).
- Pressors may be administered via peripheral vein.
  - Norepinephrine may be given peripherally with careful monitoring of the IV site for limited periods of time.

STABILIZATION:

- Antibiotics:

  If sepsis is possible, cultures should be performed and empiric antibiotics should be started without delay.

- .

STABILIZATION:

Steroids:

- Indicated for patients whom you suspect have adrenal crisis, for example:
- Patients with known adrenal insufficiency
- Patients taking chronic steroids who recently missed doses

عوارض شوک هموراژیک بر ارگانهای مختلف بدن :

- دربیشترمطالعات اثرکاهش دمای مرکزی بدن،کاهشpH یاافزایش deficit base اختلالات انعقادی وترومبوسیتوپنی درافزایش مرگ ومیرتأئیدشده است.دراین میان،سه گانهٔمرگبار یا چرخهٔمعیوب ومهلک اسیدوز،هیپوترمی واختلالات انعقادی هستندکه اگر باهم رخ دهند،بیشترین میزان مرگ ومیر را درپی خواهند داشت . ازاین رو به منظورافزایش بقاء بیماران وبهترساختن پیامد،راهکارهای ادارهٔبیماران باید درراستای اجتناب یا کاهش این عوارض باشد.از همین رو است که اندازه گیری زودرس ومکررمتغیرهای دما،وضعیت اسید،باز،کلسیم یونیزه شده،هموگلوبین،شمارش پلاکتها،PT/INR،aPTTوسطح فیبرینوژن توصیه می شود .
- (برگرفته از دستورالعمل انتقال خون ایران)

- مقادیر ذیل نشانهٔ اختلالات وخیم دربیماران باخونریزی حجیم هستند:

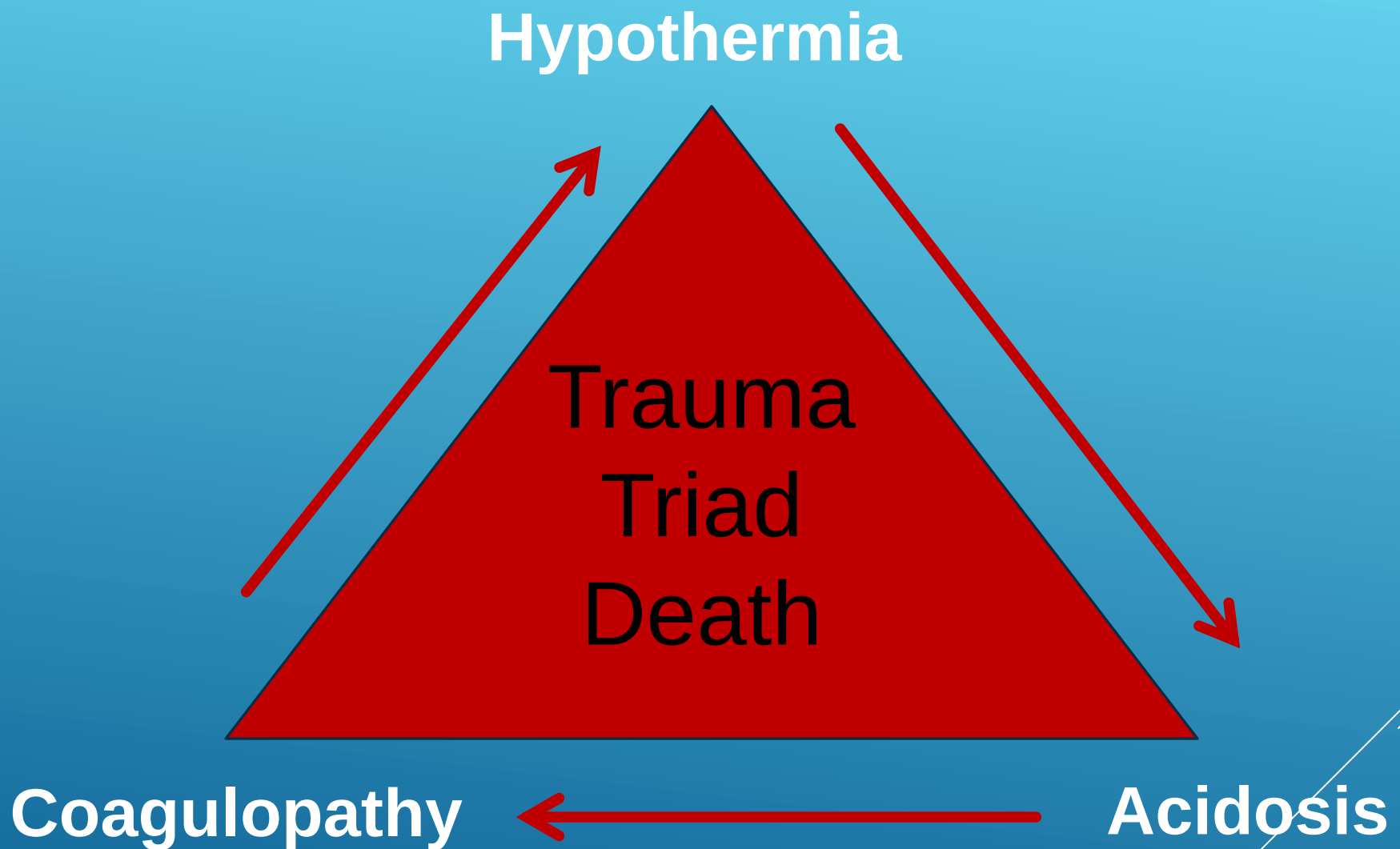
- temperature < 35°C
- pH <7.2, base excess>-6, lactate> 4 mmol/L
- ionised calcium< 1.1 mmol/L
- platelet count< 50 × 109/L
- PT > 1.5 × normal
- INR > 1.5
- APTT > 1.5 × normal
- fibrinogen level < 1.0 g/ L

- **Acidosis - Serum pH < 7.20**

- **Ongoing Marker of Severe Physiologic Derangement**

  - Decreased cardiac contractility
  - Decreased cardiac output
  - Vasodilation and decreased BP
  - Decreased hepatic and renal blood flow

نقش اسیدوز در شوک


Hypothermia
Trauma
Triad
Death
Coagulopathy
Acidosis

HYPOTHERMIA

Defined:

- Core Temp < 35C (95F)

Action:

- ↓ coagulation factors
- ↑ platelet dysfunction

Classification:

- Mod 32-34 C (90-93 F)
- Severe <32 C (< 90 F)
  T < 32C = 100% mortality

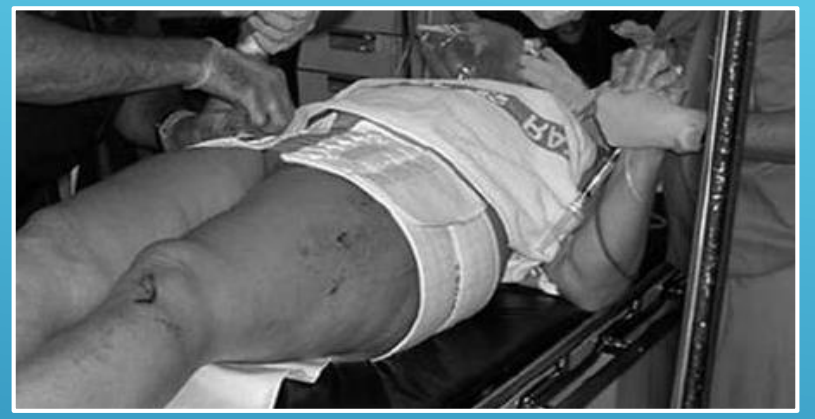
LACTATE

- Indirect measure of oxygen debt
- Normal value = 1.0 mEq/L
- Values > 1.0 correlate to magnitude of shock
- Lactate Levels > 5 = ↑ mortality
- Ability to clear lactate within 24 hours:
  - Predictive of survival
- Inability to clear lactate within 12 hours:
  - Predictive of multisystem organ failure

MECHANICAL MEANS OF STOPPING HEMORRHAGE

Pelvic Binders

- Reduce pelvis volume
- Tamponade effect



Tourniquets

- Studied extensively in war
- Good outcomes
- Safe and effective

IV Access Principles in Shock

- Fastest, simplest route best (antecubital)
- Large bore, short length (14-16 gauge, 2inch length)
- Flow limited by IV gauge & length ***not size of vein***

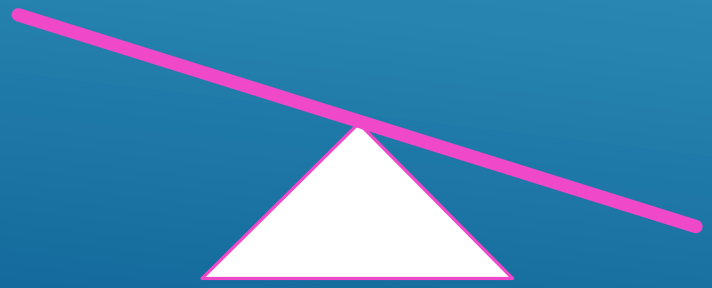
Optimally

- Two people attempting simultaneously
- Two different sites (above & below diaphragm)
- Two to three sites required per major trauma
- Progression [PIV → Femoral → Subclavian]
- Consider Intraosseous (IO) ***early*** as rescue device

Fluid Resuscitation

FLUID ADMINISTRATION BALANCE

- **Too little…**
  - Ongoing shock
  - Continued acidosis
  - Coagulopathy
  - Myocardial dysfunction
  - Renal failure
  - Death



- **Too much…**
  - Increased bleeding
  - Clot disruption
  - Dilution coagulation factors
  - Compartment syndromes
  - Transfusion concerns
    - Inflammation
    - Immunosuppression
    - Transfusion Related Acute Lung Injury (TRALI)

NS VS. LR

Normal Saline

- Na,Cl
- Fluid of choice for blood
- Con:
  - Hyperchloremic acidosis

Lactated Ringers

- Na, Cl, K, Ca, Lactate
- Fluid of choice per ATLS
- Con:
  - Immune modulation

BLOOD ADMINISTRATION

Traditional Management	
Fluid	Blood
Give 2 Liters ↓ → Continue IV's wide open	PRBC 5-10 u ↓ Wait for labs ↓ Plasma ↓ Platelets

Emerging Management	
Fluid	Blood
Minimize	1:1 or 1:2 (Plasma: RBC) Protocolize ↓ Massive Transfusion Protocol

Massive transfusion protocol (MTP) template

The information below, developed by consensus, broadly covers areas that should be included in a local MTP. This template can be used to develop an MTP to meet the needs of the local institution's patient population and resources

Senior clinician determines that patient meets criteria for MTP activation

Baseline:
Full blood count, coagulation screen (PT, INR, APTT, fibrinogen), biochemistry, arterial blood gases

Notify transfusion laboratory (*insert contact no.*) **to:**
'Activate MTP'

Laboratory staff
- Notify haematologist/transfusion specialist
- Prepare and issue blood components as requested
- Anticipate repeat testing and blood component requirements
- Minimise test turnaround times
- Consider staff resources

Haematologist/transfusion specialist
- Liaise regularly with laboratory and clinical team
- Assist in interpretation of results, and advise on blood component support

Senior clinician
- **Request:**[a]
  - 4 units RBC
  - 2 units FFP
- **Consider:**[a]
  - 1 adult therapeutic dose platelets
  - tranexamic acid in trauma patients
- **Include:**[a]
  - cryoprecipitate if fibrinogen < 1 g/L

a Or locally agreed configuration

Bleeding controlled?

YES NO

Notify transfusion laboratory to:
'Cease MTP'

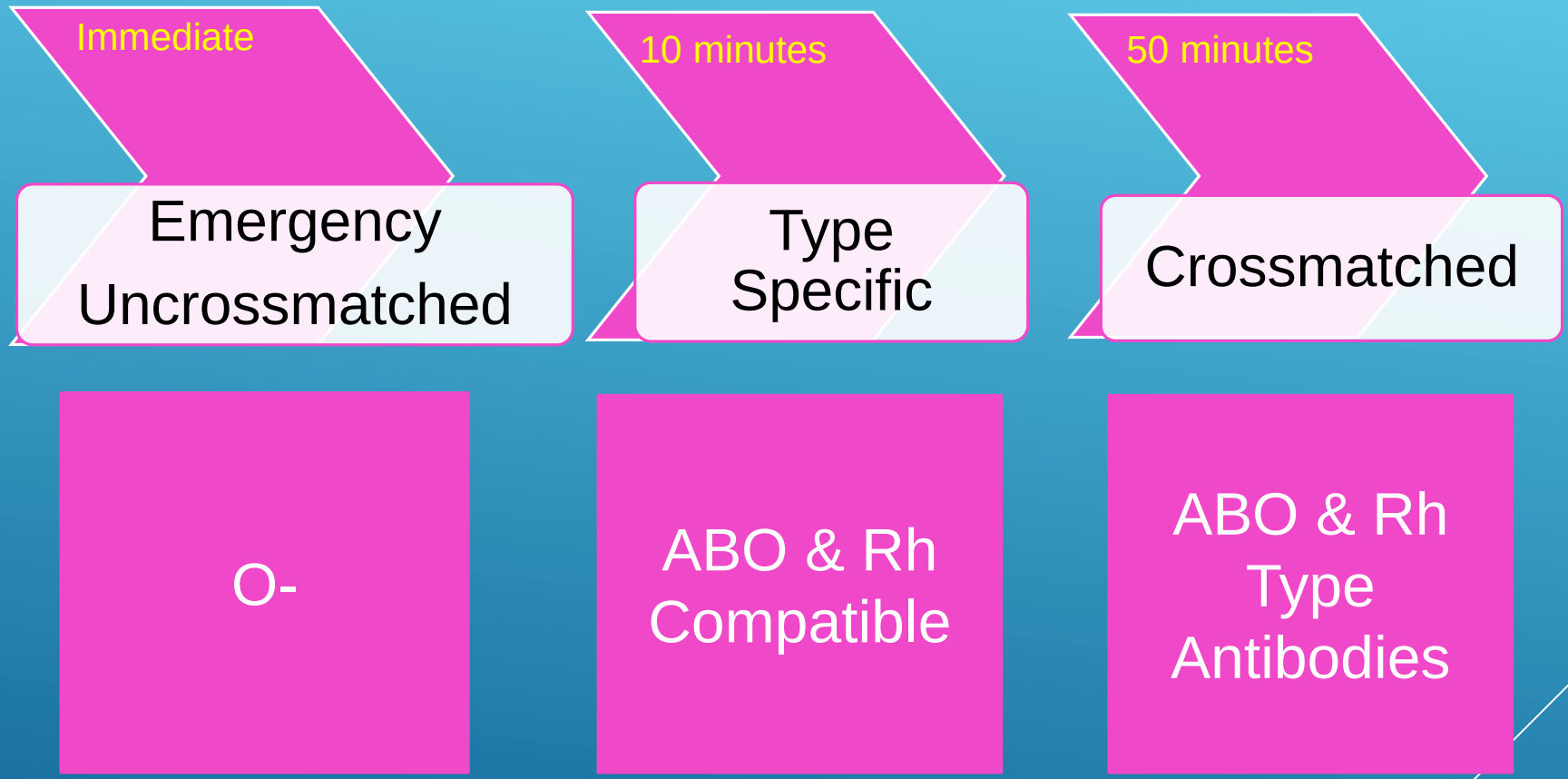
OPTIMISE:
- oxygenation
- cardiac output
- tissue perfusion
- metabolic state

MONITOR
(every 30–60 mins):
- full blood count
- coagulation screen
- ionised calcium
- arterial blood gases

AIM FOR:
- temperature > 35°C
- pH > 7.2
- base excess < –6*
- lactate < 4 mmol/L
- Ca^{2+} > 1.1 mmol/L
- platelets > 50 × 10^9/L
- PT/APTT < 1.5 × normal
- INR ≤ 1.5
- fibrinogen > 1.0 g/L

*The numerical representation of base excess can be shown differently in varying texts. Please be aware that for the purposes of this template, a base excess of <-6 refers to a base excess of -5, -4, -3 and so forth. A base excess of -7, -8, -9 and so on is associated with a worsening prognosis. The normal range for base excess is -2 - +2.

BLOOD PROGRESSION IN HEMORRHAGE



TRANEXAMIC ACID (TXA) EXAMPLE PROTOCOLS

Military Protocol

- Give within 1-3 hours of injury
- 1 unit of blood
- 1 Gm of Bolus of TXA IN 10 MINUTE
- 1 Gm Infusion over 8 hrs

CHANGING PARADIGM

Traditional

Damage Control

DAMAGE CONTROL SURGERY (1990'S)

Stage I
Initial Control of Hemorrhage

Stage II
Stabilization

Stage III
Definitive Treatment

Damage Control Resuscitation

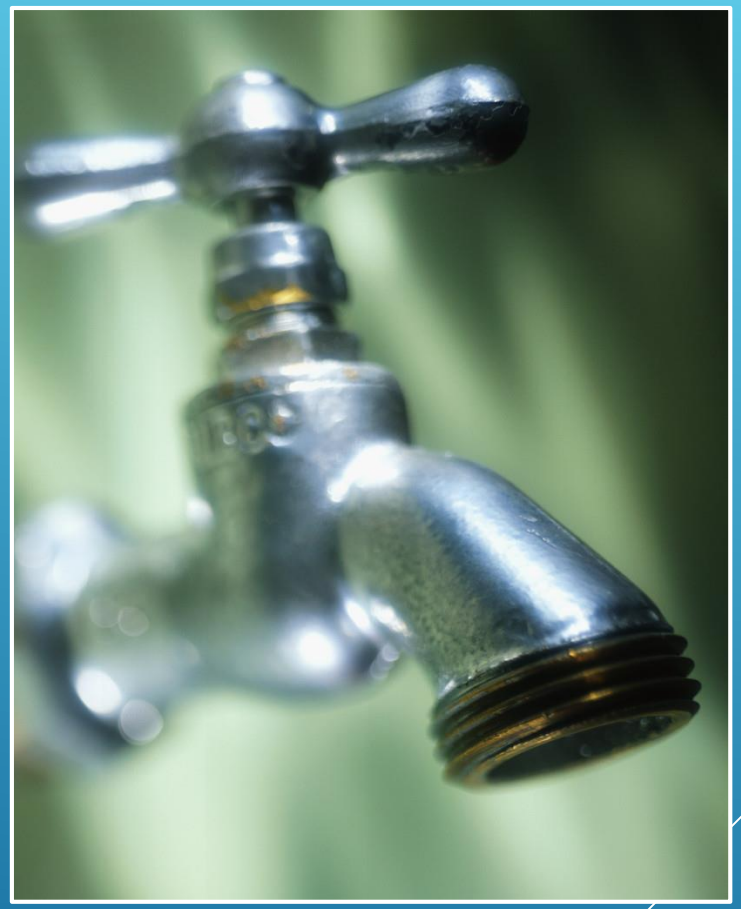
- Permissive Hypotension

- Hemostatic Resuscitation

- Damage Control Surgery

PERMISSIVE HYPOTENSION

- Restricted fluid administration
- Avoid "popping the clot"
- Accepting limited period (< 2 hours) of suboptimum end organ perfusion
- Titrate to Mean Arterial Pressure (MAP)



HEMOSTATIC RESUSCITATION

- Early diagnosis in ED
- 1:1 ratio (PRBC to FFP)
- Early frequent:
  - Cryoprecipitate
  - Platelets
- Minimal crystalloids
- Stop the bleeding

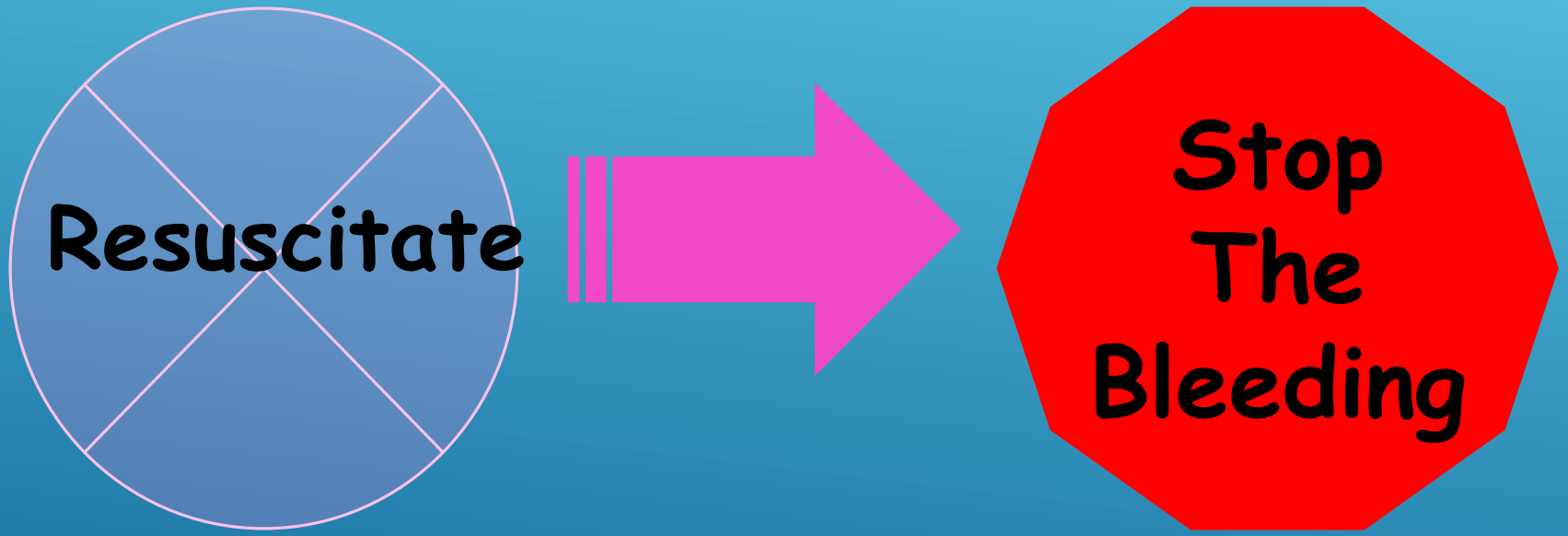
BLOOD LOSS

OLD ATLS:
After 20 years of high volume fluid resuscitation
Chasing tachycardia
Using Crystalloid > Blood
Little evidence of improved survival

Current consensus:
Damage Control Resuscitation

- Permissive Hypotension
- Hemostatic Resuscitation
- Damage Control Surgery

NEW TREATMENT PARADIGM



SUMMARY :

- Assess for coagulopathy early
- LR is fluid of choice in trauma
- Utilize Massive Transfusion Protocol
- Small volume resuscitation techniques
- Consider Tranexamic acid
- Correct acidosis and hypothermia
- STOP THE BLEEDING!