

Objectives

At the conclusion of this presentation the participant will be able to:

- Recognize hemorrhagic shock signs and symptoms
- Explain the importance of early control of hemorrhage in trauma patients
- Describe the management and ongoing evaluation of hemorrhagic shock
- List the components of damage control resuscitation



Hemorrhagic Shock



- Feared by all
- Respected by many
- Foreign to none





Every trauma center will see hemorrhaging patients with some frequency.

Trauma centers have made a large impact on decreasing hemorrhage deaths over the past 25 years.

Yet, unrecognized bleeding or delayed resuscitation remain causes of preventable death within the United States

Time to Trauma Death

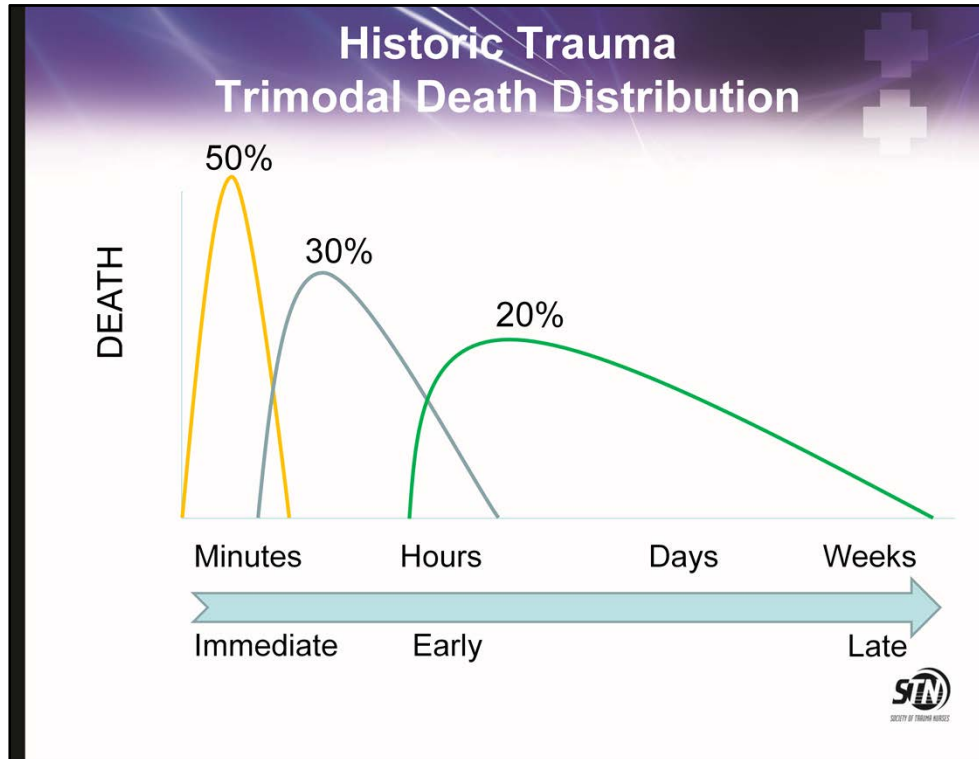
- 50% deaths occur at scene within minutes:
 - CNS injury 40-50%
 - Hemorrhage 30-40%
- 50% after hospital arrival:
 - 60% die within first 4 hrs
 - 84% die within first 12 hrs
 - 90% die within first 24 hrs
- Hemorrhage accounts for 50%
 - Deaths in the first 24 hours



90% of all trauma deaths occur within the first 24 hours with the majority occurring within the first hours after injury.

Immediate and early deaths account for the majority of trauma deaths, with <10% of deaths occurring late in the ICU.

Hemorrhage accounts for a significant portion of deaths at the scene and in the hospital.

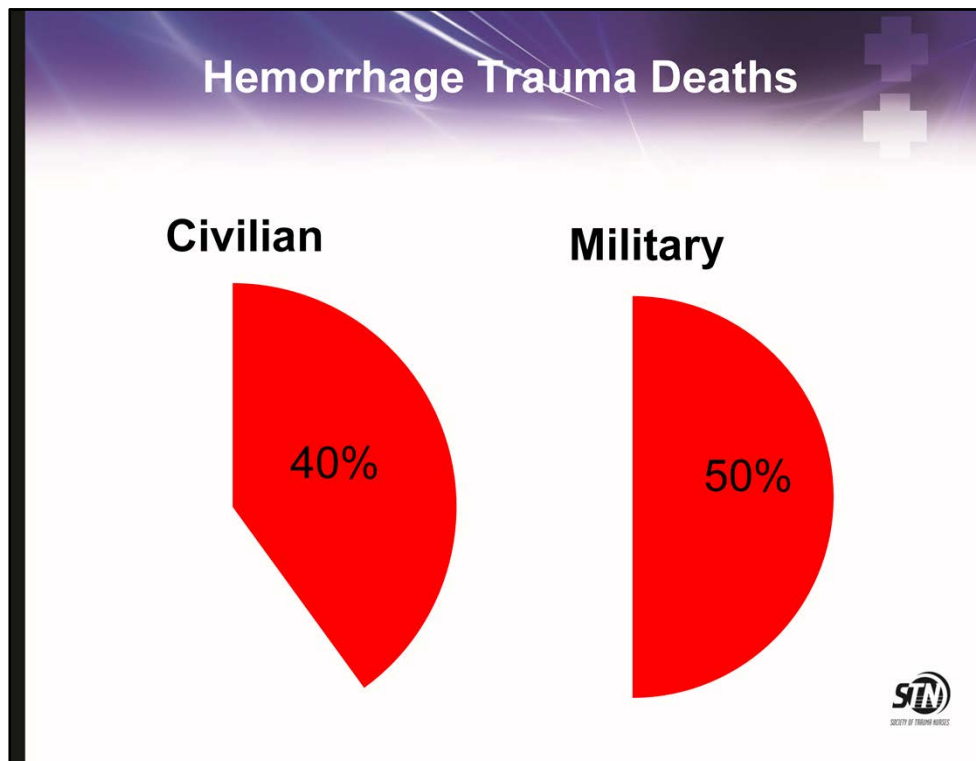


The trimodal death distribution of trauma was first described by Trunkey in 1983.

Immediate deaths occur within minutes and consist of major lacerations to the brain, brainstem, aorta, spinal cord, and heart. These are nonsurvivable injuries in which only injury prevention can affect outcome.

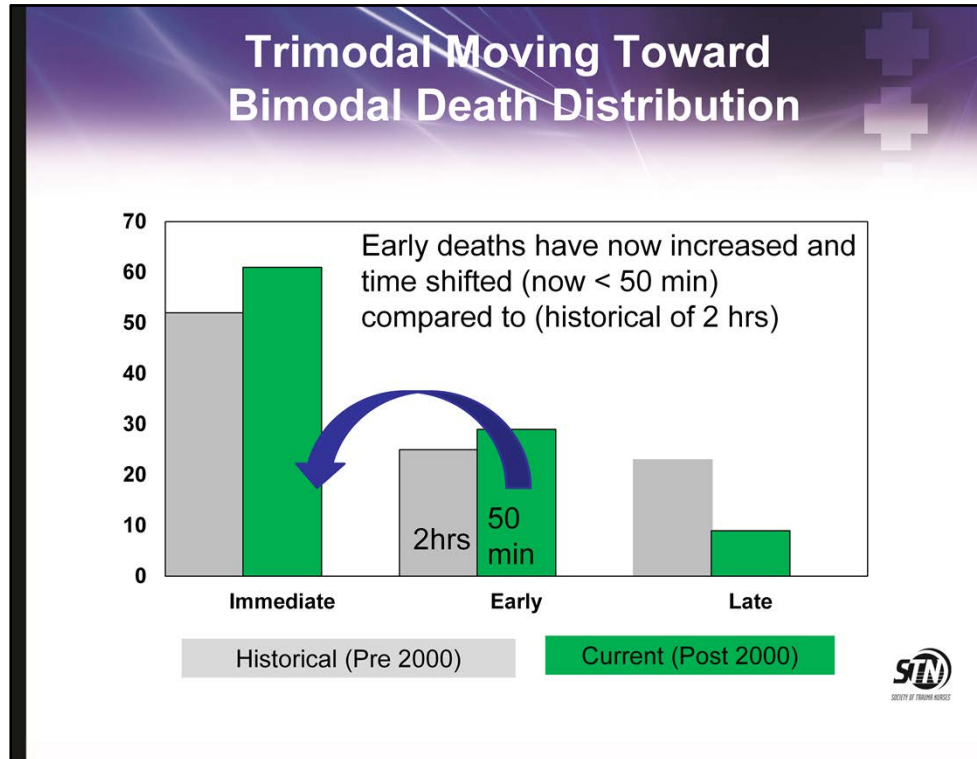
Early deaths occurred within the first few hours and consist of potentially survivable injuries such as epidurals, subdurals, hemopneumothorax, pelvic fractures, long bone fracture, and abdominal injuries. Access to trauma center care is important for this group.

Late deaths can occur up to weeks later in the intensive care unit and include deaths from sepsis and multiple organ failure. Improved resuscitation and critical care can improve these outcomes.



Hemorrhage is responsible for 40% of civilian trauma deaths, and 50% of military trauma deaths.

Up to half of the civilian deaths occur in the prehospital phase and 80-90% of combat deaths occur in far-forward environments.



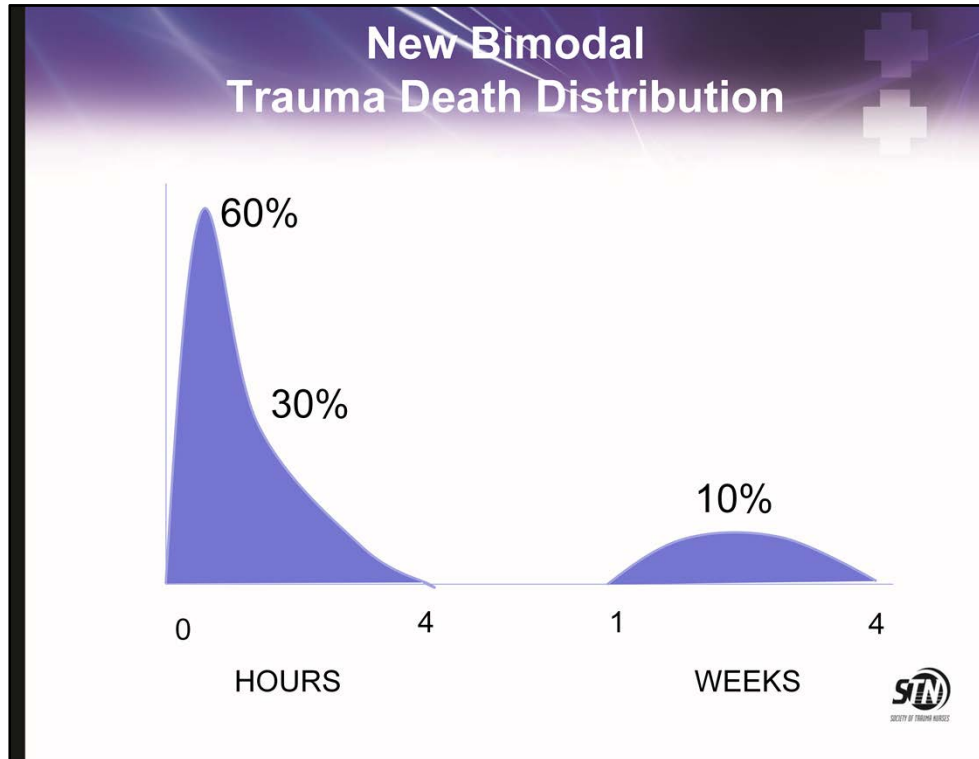
Compared with the historical controls as described by Trunkey in 1983, more recent trauma death distribution reveals:

- an increase in the percentage of immediate deaths
- small increase in early deaths
- decrease in late deaths

Most significantly, the early deaths are occurring much sooner, effectively shifting them closer to the immediate death group.

This suggests improvements in pre-hospital care has resulting in earlier arrival of severely injured patients at the hospital.

Importantly, <5% of deaths occur after the first 24 hours, resulting in a markedly diminished late peak, suggesting improvements in resuscitation, critical care, and prevention and treatment of complications.

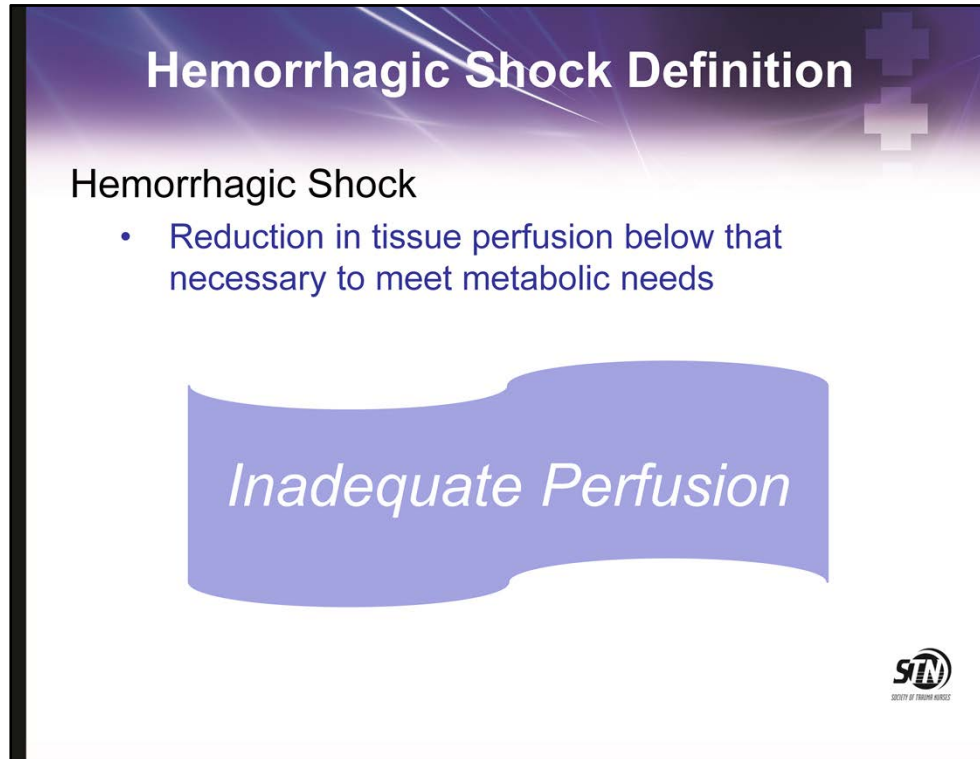


With advances in trauma systems, the deaths have now assumed a largely bimodal distribution, with a vast majority of deaths occurring within the first few hours.

Patients who survive beyond that are likely to live, with a dramatic decrease in the number of late deaths.

At the same time, large numbers of immediate and early deaths emphasize a persistent need for ongoing efforts in injury prevention and control.

The data shows a shift to the left, suggesting that patients who made it to the hospital were actually dying earlier than historical controls. This is believed to be the result of efficient prehospital providers bringing sicker patients to the hospital who previously died at the scene.



Hemorrhagic Shock Definition

Hemorrhagic Shock

- Reduction in tissue perfusion below that necessary to meet metabolic needs

Inadequate Perfusion

STN
SOCIETY OF TRANSFUSION NURSES

Shock is a physiologic state characterized by a systemic reduction in tissue perfusion below that necessary to meet the metabolic needs of tissues and organs.


Hypoperfusion results in oxygen debt, occurring as oxygen delivery becomes unable to meet metabolic requirements.

Hypoperfusion is a time-dependent emergency

Injuries Prone to Hemorrhage

Vascular	Solid Organ	Bones
Aorta Vena Cava	Spleen Liver	Pelvis Femur

Quickly Rule Out Blood Loss
Chest – CXR / FAST
Abdomen - FAST
Pelvis – Xray
Femur – exam / Xray



The 3 primary areas to look for blood loss are the chest, abdomen and pelvis.



Isolated head injuries are not the cause of hemorrhagic shock.

Fracture Associated Blood Loss

- Humerus 750 ml
- Tibia 750 ml
- Femur 1500 ml
- Pelvis > 3 L

Associated Soft Tissue Trauma
Release of Cytokines

- Increased permeability
- Magnify fluid loss



Femur and pelvic fractures are most often associated with large blood loss.

Pelvic fractures can run from stable with minimal blood loss to catastrophic hemorrhage of the entire blood supply.

Edema occurs in injured soft tissues. Tissue injury results in activation of systemic inflammatory response and production and release of multiple cytokines.

Cytokines have profound effects on the vascular endothelium, which increases permeability.

This shifts fluid primarily from the plasma into the extravascular space causing further depletion of the intravascular volume.

Confounding Factors In Response To Hemorrhage

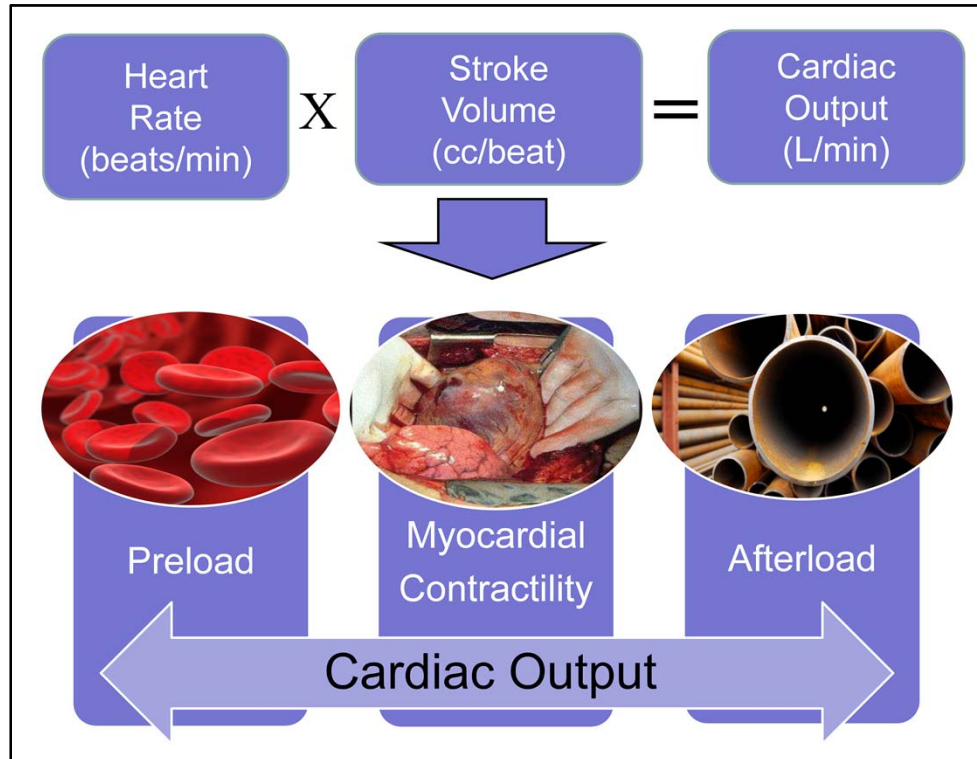
- Patients age
- Pre-existing disease / meds
- Severity of injury
- **Access to care**
- **Duration of shock**
- Amount prehospital fluid
- Presence of hypothermia



Of all of these confounders, **duration of shock** is the most important predictor of survival which of course is related to **access to care**.

Someone who is severely injured within minutes of a trauma center with prompt access to care has a much higher likelihood of survival than the person with a less severe injury but who has delayed access to care (rural, etc).





Cardiac output which is defined as the volume of blood pumped by the heart per minute, is determined by multiplying the heart rate by the stroke volume.

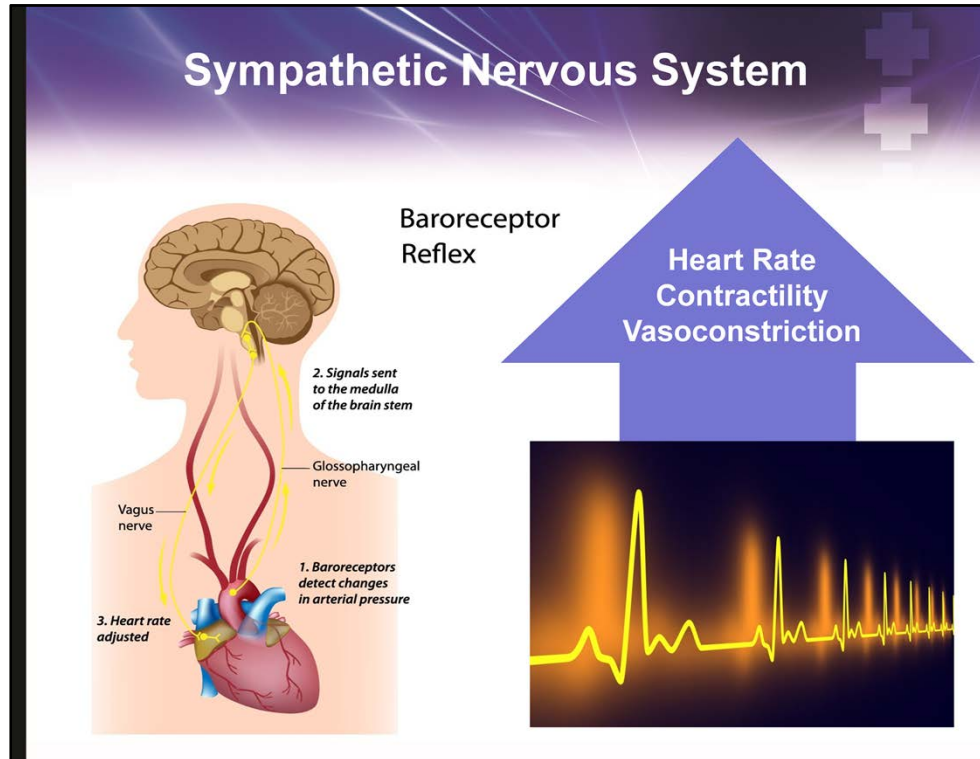
Stroke volume, the amount of blood pumped with each cardiac contraction, is classically determined by the following:

- Preload (volume of venous return to the heart)

- Myocardial contractility

- Afterload (systemic or peripheral vascular resistance) The resistance of forward flow of blood.

Hemorrhagic shock primarily affects the PRELOAD



Uncontrolled hemorrhagic shock initially leads to hypotension (decrease in mean arterial pressure [MAP]) due to an acute decrease in cardiac output.


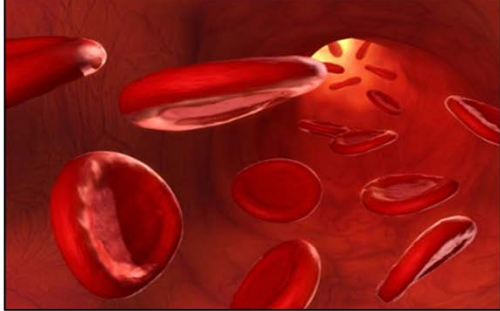
The intrinsic response to hemorrhagic shock is stimulation of the sympathetic nervous system via the baroreceptor reflex which results in an increase in heart rate in an attempt to preserve cardiac output.

In most cases, **tachycardia** is the **earliest measurable circulatory sign of shock**.

Sympathetic Nervous System

Progressive Vasoconstriction:

- Skin
- Muscle
- Splanchnic Bed



Sympathetic Nervous System stimulation as a result of response to blood loss are compensatory in nature.

They result in progressive vasoconstriction of cutaneous, muscle and visceral (splanchnic) circulation which preserves blood flow to the heart and brain.


This is why the skin is often cold and clammy in shock.

The skin, muscle and gut are sacrificed to preserve blood flow to the heart and brain.

Important Hormones in Shock


Catecholamines: Epinephrine & Norepinephrine

- Increased heart rate & contractility
- Vasoconstriction & narrowed pulse pressure



Renin-Angiotensin Axis: Aldosterone and ADH

- Water & sodium conservation & vasoconstriction
- Increase in blood volume and blood pressure
- Decreased urine output



Catecholamines:

Endogenous catecholamines (epinephrine and norepinephrine) are released from the adrenal gland which results in increased peripheral vascular resistance, which in turn increases diastolic blood pressure and reduces pulse pressure which you may observe in Class II shock.

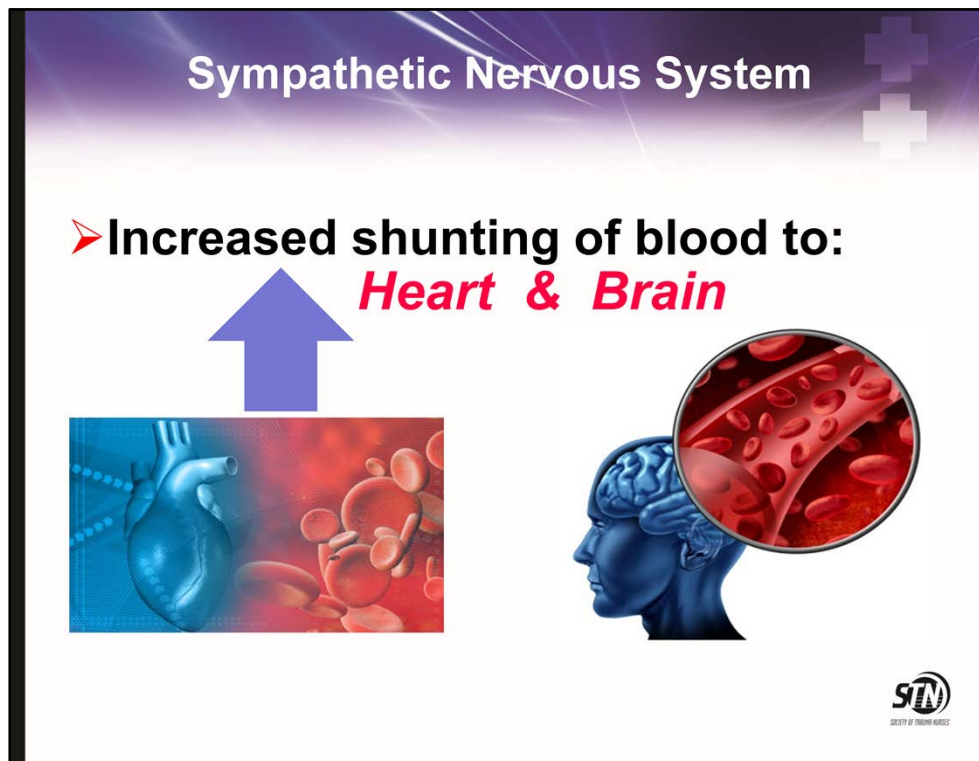
Renin-Angiotensin Axis

Renin is released from the juxtaglomerular apparatus (JGA) when there is decreased arterial blood flow to the kidney.

The renal medulla releases aldosterone one of the most potent vasoconstrictors in the body, as well as increasing reabsorption of sodium and water.

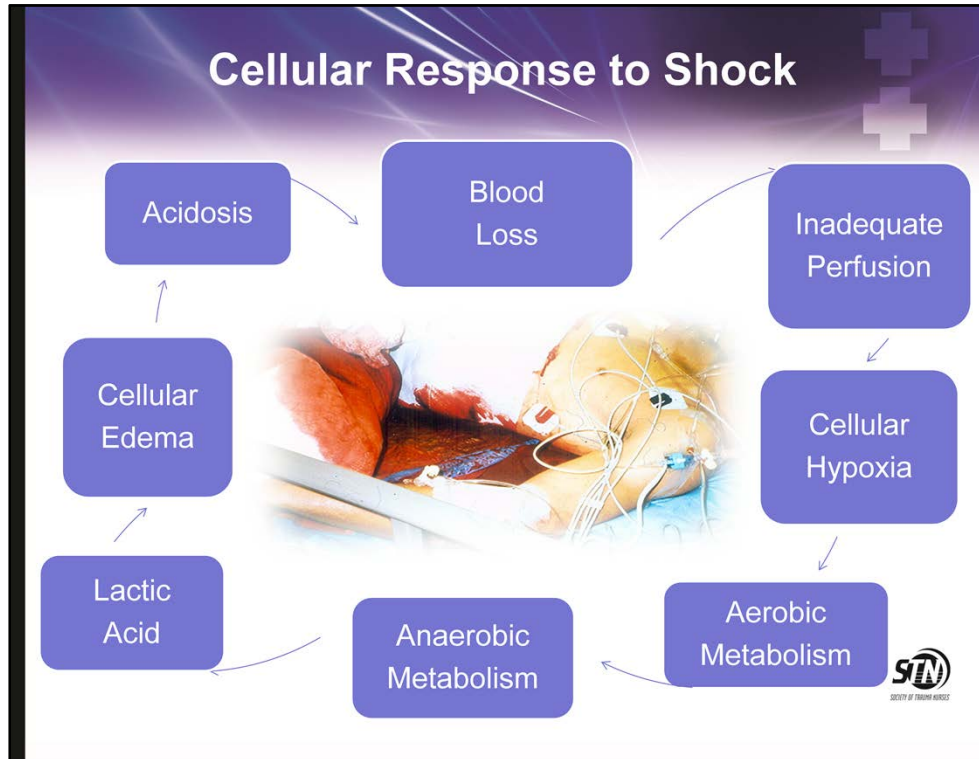
Osmoreceptors in the hypothalamus release antidiuretic hormone (ADH) which causes reabsorption of water.

The net effect of both is the resultant reduction in urine output.



Sympathetic nervous system stimulation results in increased blood flow to the heart and brain.

Everything is geared toward saving the heart and brain.



At the cellular level, inadequately perfused and oxygenated cells are deprived of the essential substrates for normal aerobic metabolism and energy production.

Initially compensation occurs by shifting to anaerobic metabolism, which results in the formation of lactic acid and the development of metabolic acidosis.

If shock is prolonged, the cell membrane loses its integrity, the normal electrical gradient is lost and swelling and damage occur and eventual cell death.

Arterial pH serves as a marker of metabolic derangement.





Classic Signs & Symptoms of Shock

- Changing mentation
- Tachycardia
- Cool, clammy, skin
- Prolonged capillary refill
- Narrowed pulse pressure
- Decreased urine output
- Hypotension




These are the classical signs and symptoms of shock. They can be difficult to appreciate early in shock and can be missed until the patient is in trouble.

Combining traditional assessment with labs and non invasive monitoring can increase the accuracy in identifying patients earlier in shock.



Normal
Vitals do not
r/o Occult Hypo
Perfusion



Early IV fluid and or physiologic compensation can lead a clinician to think that the patient is stable.

It is important to remember that the presence of normal vital signs does not rule out the presence of occult hypoperfusion.

Realize that occult hypoperfusion is not readily observed with vital signs or physical exam.

It often requires non invasive hemodynamic monitoring and or global parameters of shock such as base deficit and lactate to determine if the patient is compensated shock.

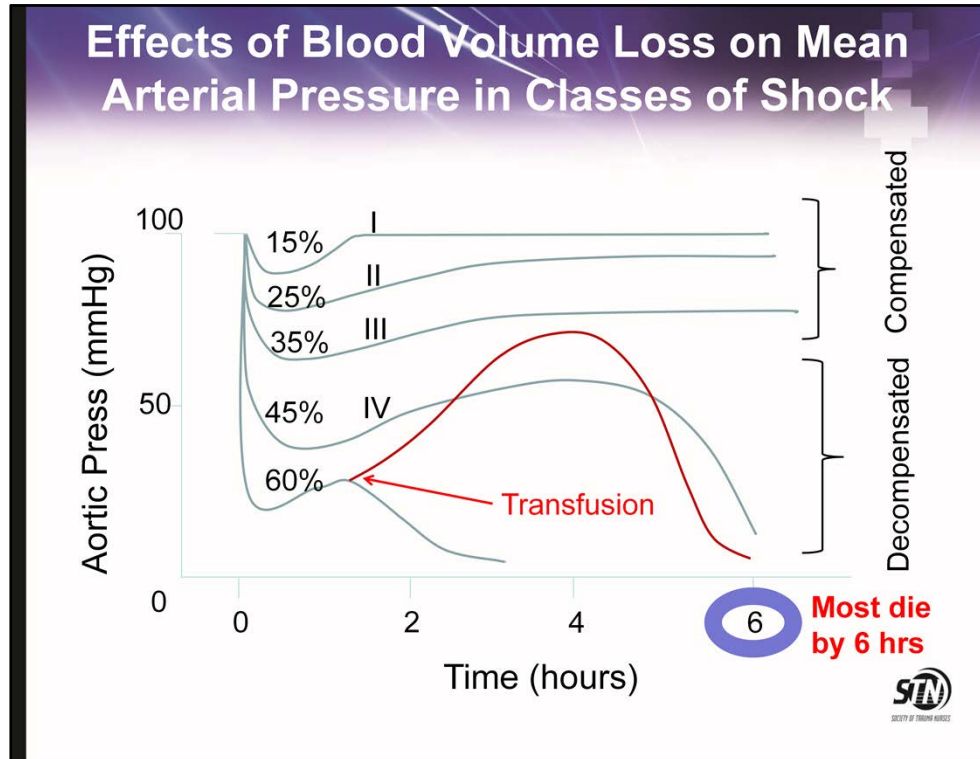
ATLS Classification of Hemorrhagic Shock

	CLASS I	CLASS II	CLASS III	CLASS IV
Blood Loss (ml) %	<750 15%	750-1500 15%-30%	1500-2000 30-40%	>2000 >40%
HR	<100	>100	>120	>140
BP	normal	normal	decrease	decrease
PP	normal	decrease	decrease	decrease
RR	14-20	20-30	30-40	>35
UOP	>30	20-30	5-15	negligible
CNS	slightly anxious	mildly anxious	anxious confused	confused lethargic



The classification of hemorrhage into 4 classes based on clinical signs is a useful tool for estimating the percentage of acute blood loss.

This classification system is also useful in emphasizing the early signs and pathophysiology of shock.



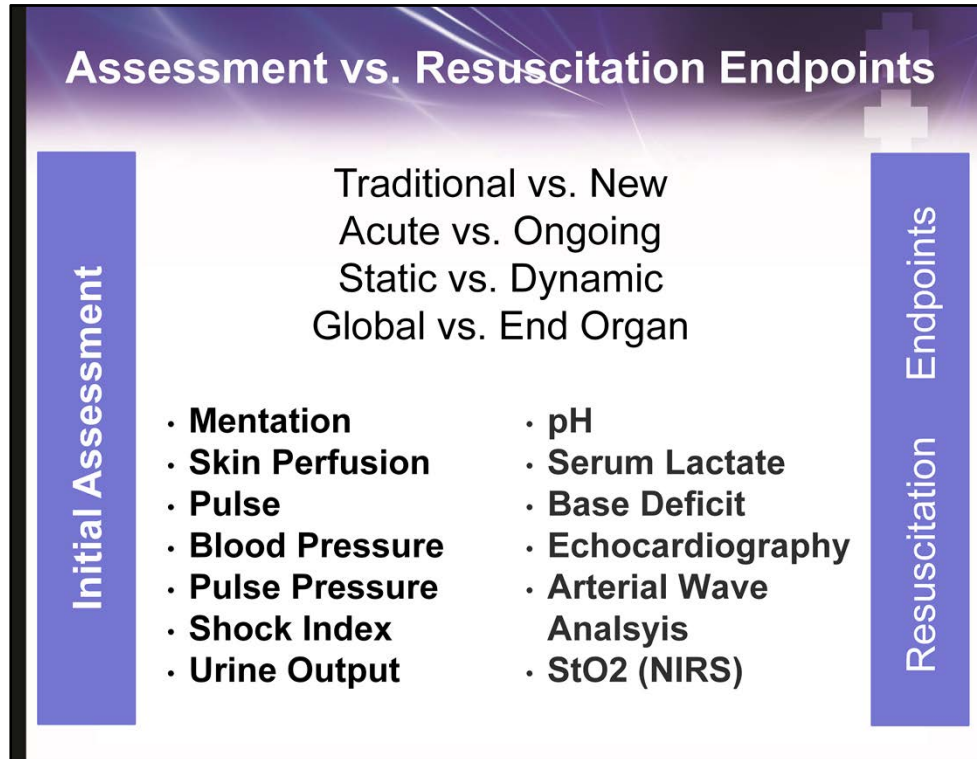
This graph shows the effects of blood volume loss in the 4 classes of hemorrhage over time.

Class I – III is compensated shock.

Class IV transitions to decompensated shock (body's inability to compensate for continued shock state).

Most patients who bleed to death will do so within about 6 hours.

Transfusion can temporize blood loss but will not fix it.



Initial assessment is usually performed with rudimentary physical exam and vital sign assessment on patient arrival.

Resuscitation Endpoints are more sophisticated and require ongoing assessment during the ED to OR to ICU time phases of care.

Resuscitation endpoints have been described in a variety of ways.


The ultimate end point of any resuscitation is an awake, functional, hemodynamically stable patient.

This target becomes obvious in the later stages of resuscitation, after successful surgical source control of hemorrhage.

At that point the clinician can strive for normality in vital signs, laboratory measures, and organ system function.

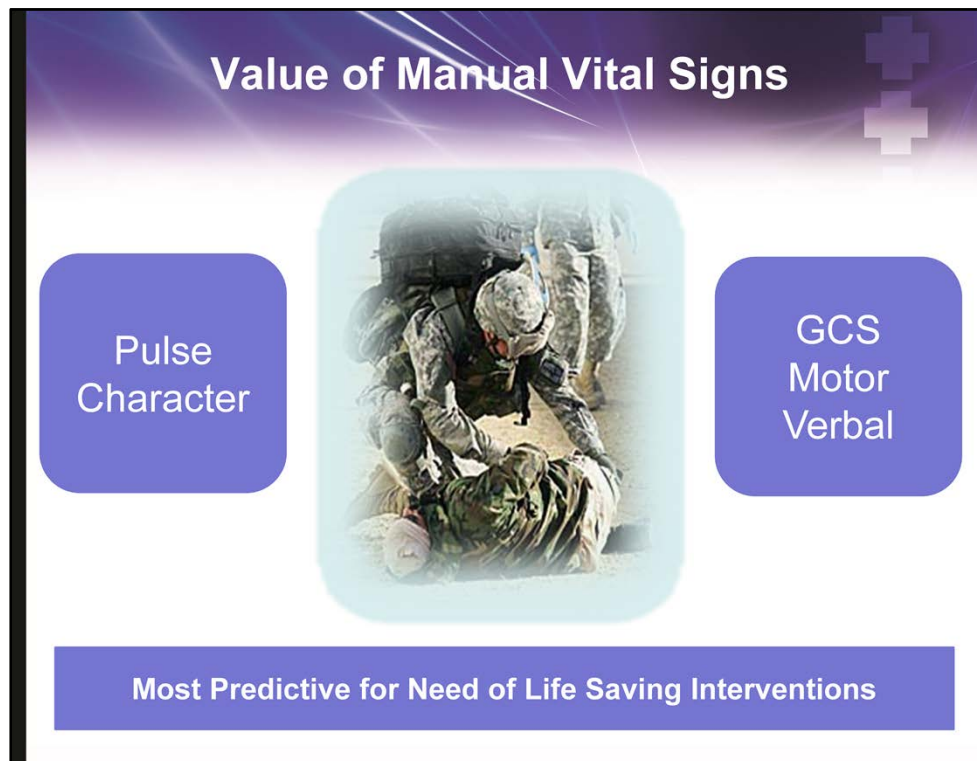
Each of these will be covered on the following slides

Response Fluid Resuscitation			
EVAL	Rapid Response	Transient Response	No Response
Vital Signs	Return to normal	Transient improvement	Remain abnormal
Estimated Blood Loss	Minimal (10-20%)	Moderate and ongoing (20-40%)	Severe (>40%)
Need for more IV fluid	Low	High	High
Need for Blood	Low T&C	Moderate Type Spec	Immediate O Pos/Neg
OR	Possibly	Likely	High



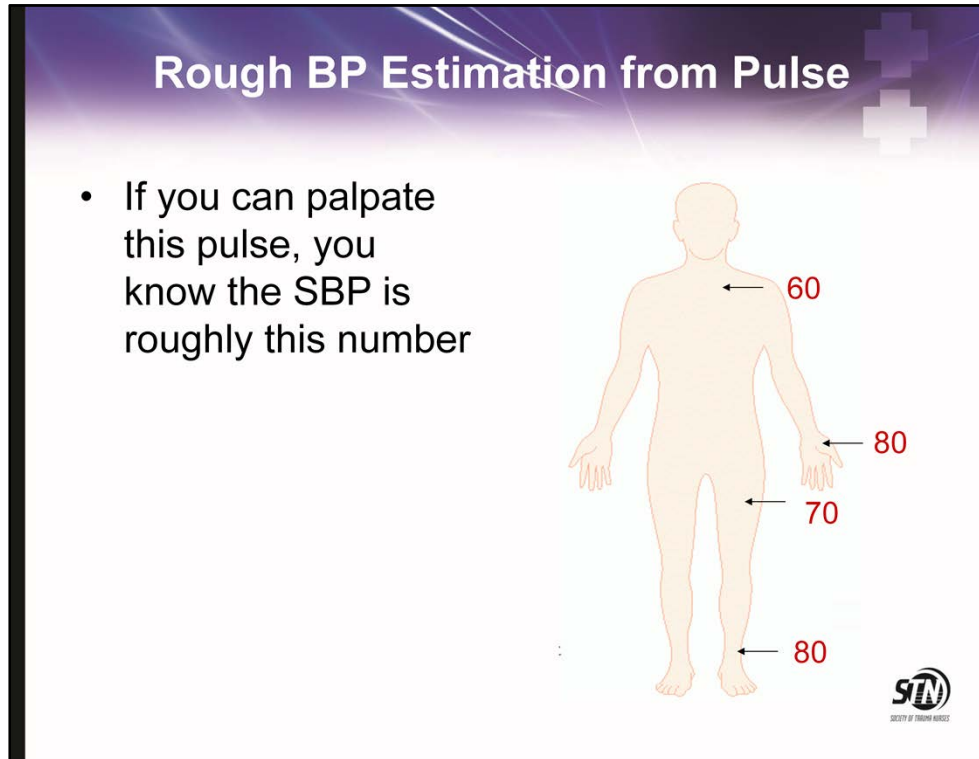
It is estimated that of bleeding patients 1/3 will be rapid responders, 1/3 will be transient, and 1/3 no response (in extremis)

The patient's response to initial fluid is the key to determining subsequent therapy.



KISS: Keep it simple stupid technique.

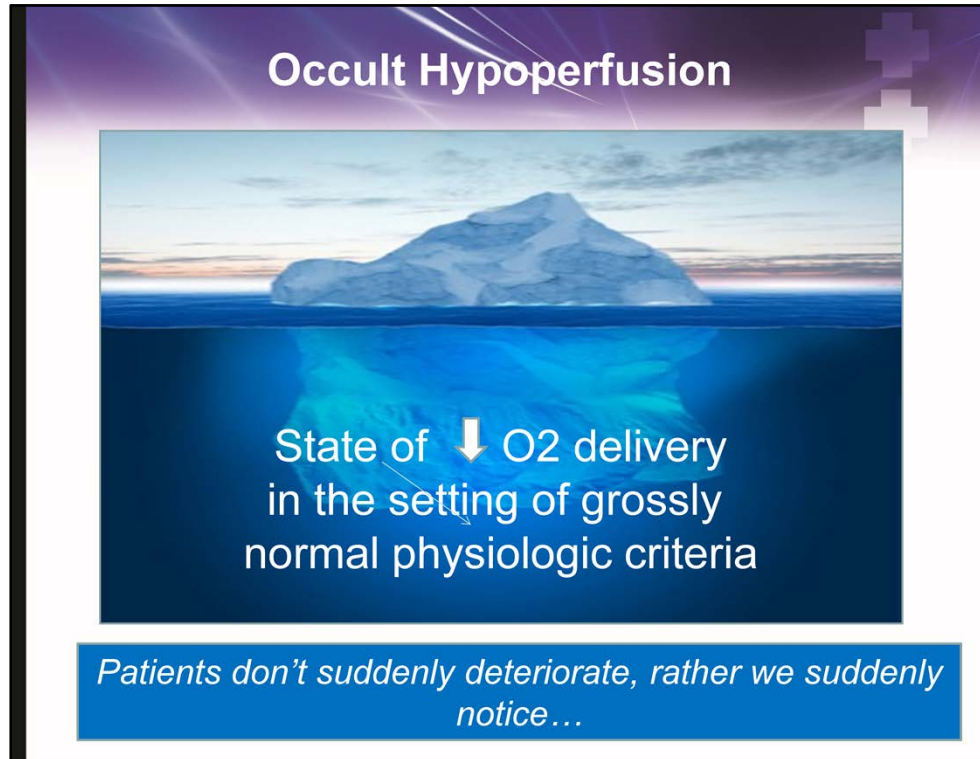
Military studies show that manual vital signs performed (without equipment) are the simplest and most predictive for need of life saving interventions.



Crude measures such as BP estimation from pulse and mentation from (can the patient talk) test, are useful for both simplicity and accuracy in trauma.

They have assisted in r/o shock especially in the military for use on the battlefield and for civilian use in disaster triage scenarios.

Now it is also used by prehospital personnel to ID which patients may require an IV and or any fluids.



The diagnosis and management of occult shock presents a unique challenge.



Using a variety of assessment parameters (resuscitation endpoints) will help.

No one parameter is 100% fool proof.

The combination of vital signs + physical assessment + lab values + non invasive/invasive hemodynamic parameters give you the best shot at identifying shock early.

Changing Mentation

- Indicator of perfusion
- Affected by drugs & alcohol
- Hypoxia/Head Injury
 - Until proven otherwise





Mentation change is one of the first signs of shock.

Although shunting of blood to the brain and heart is preserved in early shock, hypoxia to the brain progressively worsens with oxygen deficit accumulation.

Never assume that altered mentation is due to drugs or alcohol alone. Consider hypoxia from inadequate perfusion and or head injury until proven otherwise.

Skin Perfusion

- **Pale, cool, mottled**
 - Vasoconstriction
- **Most sensitive in pediatrics**
 - Starts distal extremities
 - Ascends towards trunk
- **Capillary Refill**
 - Unreliable to measure
 - Normal < 2 seconds



Skin perfusion changes in adults are signs of physiologic compensation. **Note the mottling of the legs in the picture**

Skin perfusion changes are early and sensitive in pediatric patients. It starts distally and ascends toward the trunk as the shock worsens

Capillary refill is a clinical sign popularized in the past 15 years.


Gained widespread acceptance without research

Intuitively it is a measure of peripheral perfusion which is likely to be a function of cardiac output and peripheral vascular resistance.

It is a semiquantitative test, with a value of < 2 seconds generally considered to be normal.

Blood Pressure

- **BP response to volume loss**
 - Non-linear due to compensatory mechanisms
 - Insensitive sign of early shock
- **NTDB study**
 - SBP did not decrease < 90
 - Until base deficit was > 20
- **Infrequently & or inadequately monitored**
 - First BP should always be manual
 - Automated BP overestimated by 10 mm Hg




Compensatory mechanisms allow significant reductions in central circulating blood volume, stroke volume, and cardiac output to occur well before changes in arterial blood pressure.

Such physiologic compensations can mask the true nature and severity of many traumatic injuries, leading to under appreciation of the severity of injury, under triage, and increased mortality.

Research has demonstrated that automated devices overestimate the true manual blood pressure by approximately 10 mm Hg

Blood Pressure

- Systolic BP drop a late sign
- Systolic BP does not fall until:
 - Adults 30% blood loss
 - Pediatrics 40-45% blood loss
- SBP \leq 90 mm Hg: mortality approaches 65%

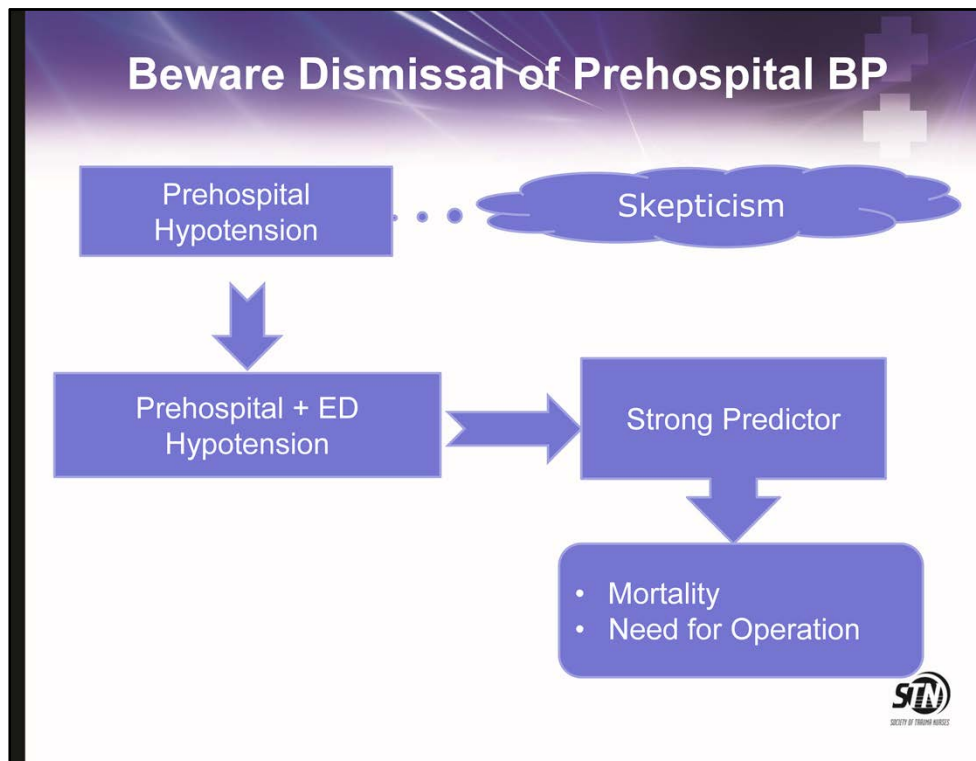


Measurements early in the course of shock are not well correlated with blood flow or cardiac output.

Significant hypoperfusion occurs in blunt and penetrating trauma patients despite normal, standard vital signs, **especially in young, healthy patients.**

Compensatory mechanisms allow significant reductions in central circulating blood volume, stroke volume, and cardiac output to occur well before changes in arterial blood pressure.

Such physiologic compensations can thus mask the true nature and severity of many traumatic injuries.




Prehospital hypotension is sometimes met with skepticism by clinicians.

However the presence of prehospital & ED hypotension is **HIGHLY** predictive of mortality and need for surgical intervention

Hypotension Redefined?


The New Hypotension: **SBP \leq 110**

- Associated with onset of physiologic changes



- Base Deficit
- ICU Days
- Ventilator Days
- Complications

- Use only to diagnose shock
- Not as resuscitation endpoint



Recent research suggest that SBP of 110 mm Hg after injury is associated with the onset of profound physiologic and outcome changes.

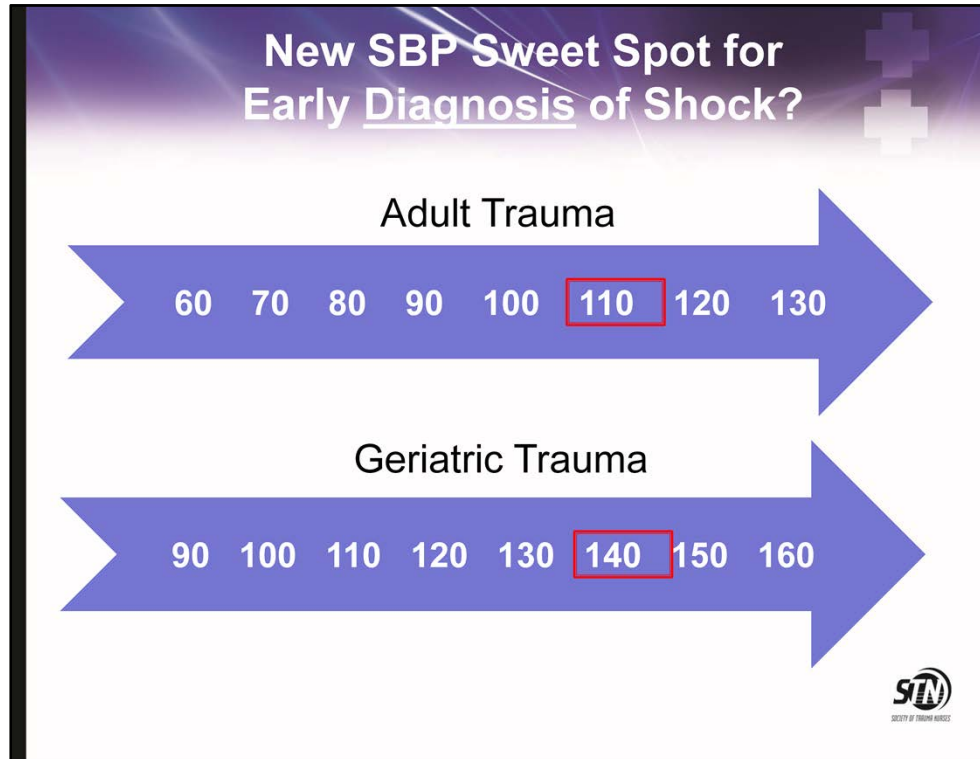
Interestingly an SBP of 115 mm Hg appears to be associated with the inflection point of base deficit and corresponds to the inflection point at which complications, ICU days and ventilator days increase.

These results are independent of both age and gender.

Research demonstrates that optimal SBP
for improved mortality in hemorrhagic
shock increases with age



Mounting evidence indicates that the optimal SBP for improved mortality in
hemorrhagic shock increases with increasing age



In adults, tissue hypoperfusion is apparent starting with SBP \geq 110 mm Hg after trauma. This provides an earlier awareness of risk for insidious evolution of shock.

You should not feel comfortable or let your guard down just because the patient's SBP is between 90 and 110 mm Hg.


Patients older than 70 years have a much higher mortality after trauma compared with similar injured younger patients. The cause is multifactorial:

- Elderly patients have lower cardiac output, decreased ability to compensate to hemodynamic stress, and an interrupted response to increased sympathetic tone.
- In addition, baseline blood pressure is higher in the elderly, so relatively decreased perfusion occurs at higher SBP compared with younger patients.
- Elderly patients also have multiple comorbidities and associated medications (ex: beta blockers) , which can mask normal physiological responses to injury.
- Elderly trauma patients (70 years or older) without major head injuries should be considered hypotensive for SBP values less than 140 mm Hg.
- Therapy for hemorrhagic shock may be indicated in the trauma bay for elderly patients with SBP less than 140 mm Hg and no evidence of head injury to prevent deaths in this population.

Pulse Pressure

- Narrowed pulse pressure suggests significant blood loss
- Result of increasing diastolic pressure from compensatory catecholamine release

100/66 100/74 100/77 100/84




The release of endogenous catecholamines increases peripheral vascular resistance, which in turn increases diastolic blood pressure and reduces pulse pressure but does little to increase organ perfusion.


Normal pulse pressure is around 40-50.

Pulse

- Lacks specificity alone
- Age dependent
- Affected by:
 - Emotion
 - Fever
 - Pain
 - Drugs
- Pulse **&** character together more reliable
- Trended over time *may?* have sensitivity
- When to be concerned?
80 90 100 **110 > 120**



Any patient who is cool & tachycardic is in shock until proven otherwise (ATLS)



Clinical studies have demonstrated a poor correlation between tachycardia and hypotension, calling into question the validity of using heart rate as a reliable sign of hemorrhagic shock.

A combination of pulse rate and quality, as advocated by ATLS, better correlates with shock.



In most cases, tachycardia is the earliest measurable circulatory sign of shock.

Heightened concern for patients presenting with tachycardia >120 bpm appears appropriate.

This is the cutoff for patients with increased need for emergent intervention or blood transfusion.

Relative Bradycardia (Paradoxical Bradycardia)

- Defined as Pulse < 90 with SBP < 90
- Occurs in up to 45% of all hypotensive trauma
- Cause remains unclear:
 - Sign of rapid & severe internal bleeding?
 - Increased vagal tone from blood in abd cavity?
 - Protective reflex designed to increase diastolic filing in the presence of severe hypovolemia?



Paradoxical Bradycardia is also known as Relative Bradycardia.

Cessation of tachycardic response historically considered a preterminal event.



In the late 1980's several reports appeared confirming the rare presence of relative bradycardia in the presence of hemorrhagic shock.

A more recent multicenter study found that it occurs in up to 45% of all hypotensive trauma.

The cause of RB remains unclear; however, multiple causes have been suggested, including a neural reflex mechanism, direct compression of the vagus nerve, and severe internal bleeding.

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?




Shock index (SI) is the ratio of heart rate in bpm to systolic blood pressure in mm Hg.


It is commonly used to assess the amount of blood loss and degree of hypovolemic shock.

The shock index is useful for raising early suspicion of hypovolemia even when the heart rate and blood pressure remain normal.

Urine Output



Adult	0.5 ml / kg / hour
Child	1.0 ml / kg / hour
Toddler	1.5 ml / kg / hour
Infant	2.0 ml / kg / hour





Urine Output (UOP) is best calculated by wt and time

Remember also that UOP is affected by: glycosuria, ETOH, diuretics. Interpret with caution in the presence of these.

Hemodynamic Monitoring



Central Venous Pressure	Pulmonary Artery Catheter
<ul style="list-style-type: none">• Not advocated for hemorrhagic shock• Poor relationship between CVP and blood volume• Unreliable for assessing response to fluid• Use:<ul style="list-style-type: none">• Acute air embolus• Acute PE• Rt Ventricular infarction• Acute lung injury	<ul style="list-style-type: none">• Not advocated for hemorrhagic shock• Dynamic response of the systems too slow to guide therapy• Use:<ul style="list-style-type: none">• May benefit geriatric trauma• Sepsis goal directed therapy



The “gold standard” methods, such as central venous pressure monitoring and pulmonary artery catheter (PAC) monitoring , have seen a decline in routine clinical use and have fallen out of favor for hemorrhagic shock.

Doppler Echocardiography (Transthoracic or Transesophageal)

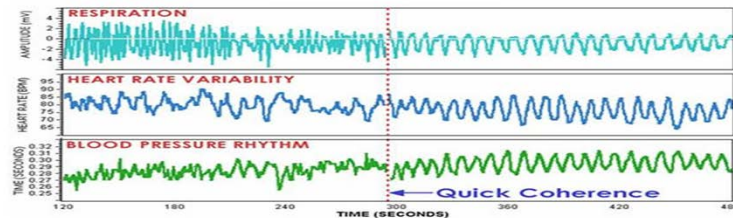
- Allows for physician bedside assessment:
 - Ventricular function
 - Volume status
 - Stroke volume
 - Cardiac output
- Dependent on:
 - Technology investment
 - Technical expertise
 - Intra-observer variability
- Excellent diagnostic tool
- Poor monitoring device



- Minimally invasive technologies which have been used recently to determine fluid volume status include echocardiography and transesophageal Doppler
- Basic parasternal, apical, and subxyphoid views can be obtained in the majority of patients to assess real-time hemodynamics.
- These skills can be acquired through specialized training under the guidance of an experienced echocardiographer or in a critical care training program with an emphasis on echocardiographic assessment. Without continued use, the skills deteriorate quickly.
- Caution: a problem of noninvasive monitors in that the algorithms have been developed and much of the clinical validation studies performed in relatively healthy and normal patients.
- Algorithms used to derive familiar metrics, such as cardiac output and stroke volume, may be flawed in patients with the extreme physiology of severe shock or exsanguinating hemorrhage.

Physiologic Variability as Predictors

- Subtle patterns of variation produced by healthy biological systems is normal



- Loss of this variability is seen in critical illness
- Early loss of HR variability predicts mortality in trauma



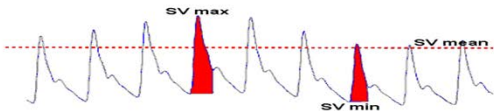
A normal heart rate for example constantly fluctuates by at least several beats per minute.

These patterns of variability reflect complex interactions between neuroendocrine systems, tissues and organ systems, autonomic responsiveness and hemodynamic and metabolic changes.

Emerging evidence reveals that heart rate variability and complexity in trauma are useful predictors of death.

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)



Determining where the patient lies on their individual Starling curve, during the resuscitation process, may be more important than the fluid type being administered.

Arterial pressure waveform systems function on the relationship between **pulse pressure and stroke volume (SV)**.

Systolic pressure variation, the difference between maximum and minimum systolic pressure during one mechanical breath, has been shown to predict fluid responsiveness to volume loading.

Concepts, such as pulse pressure variation and stroke volume variation in ventilated patients, have been extensively reviewed in the literature and found to be reliable predictors of volume responsiveness.

Limitations- may be affected by:

Vent settings
Chest wall compliance
Dysrhythmias

Examples of systems:

PiCCO (Phillips) | pulseCO (LiDCO, Ltd.) | FloTrac/Vigileo (Edwards)

Near Infrared Spectroscopy (NIRS)

Skeletal muscle StO₂

- Measures hemoglobin oxygen saturation in tissue
- Tracks systemic O₂ delivery
- Continuously and Noninvasively
- Comparable results to BD and Lactate
 - Predicts MSOF
 - Predicts Mortality
 - Research ongoing as resuscitation endpoint

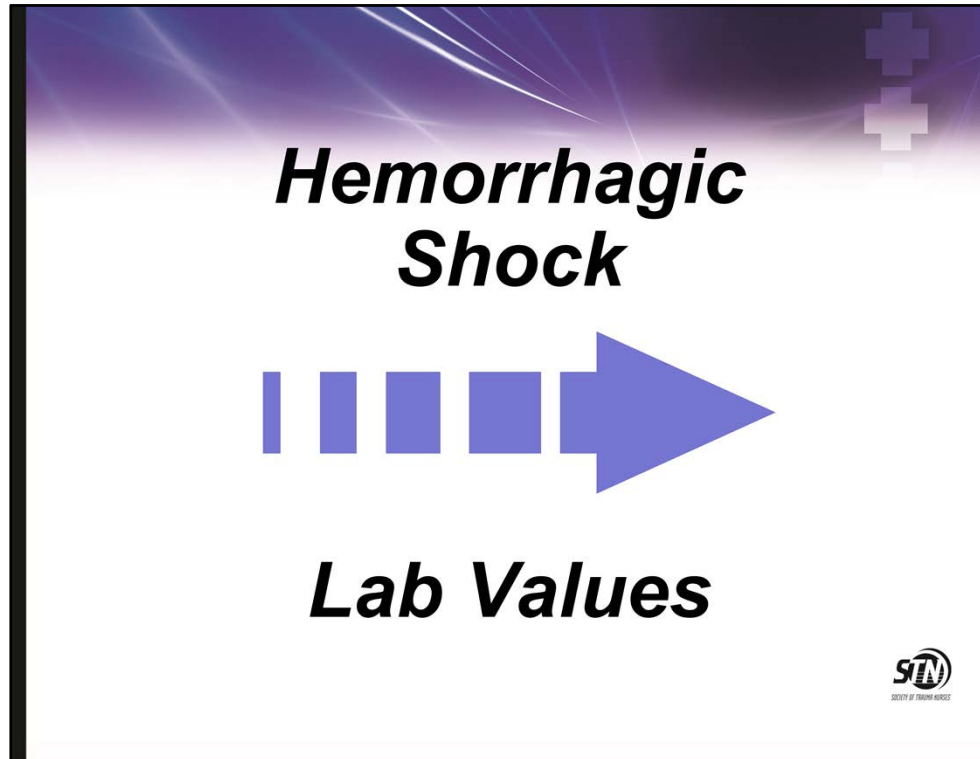


Skeletal muscle oxygenation at the periphery, is easily measured and has been suggested as an end point of resuscitation.

StO₂ and Base Deficit are comparable in predicting death and identify poor perfusion and predicting the development of multi system organ failure.



StO₂ has the added benefit of being measured noninvasively and continuously.

Transcutaneous probe is applied to the thenar eminence of the hand.



Hemoglobin / Hematocrit

- Unreliable estimation acute blood loss
- Lag time of several hours
- Baseline value for comparison only

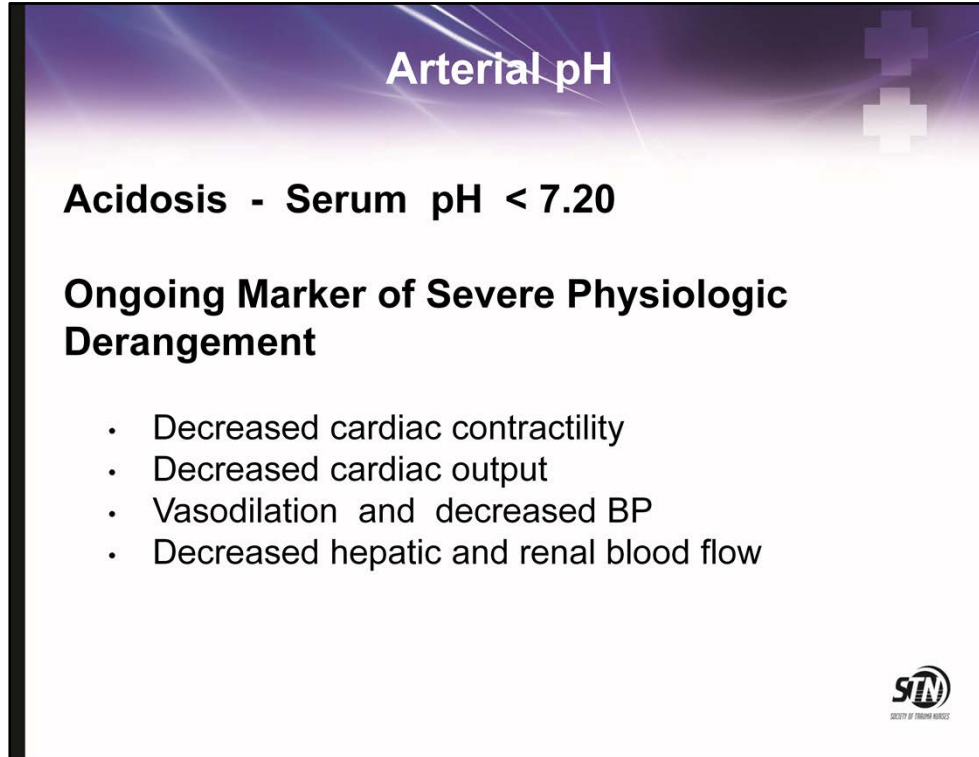


In real time, laboratory data lag behind the clinical situation.

The fastest measure of blood composition to turn around—the hemoglobin—is not a good indicator of the depth of hemorrhage, because it expresses a concentration of red cells, which will not change in a patient who is losing whole blood.

Use of the hematocrit to estimate acute blood loss is unreliable and inappropriate”. Changes in hematocrit show a poor correlation with blood volume deficits and red cell volume deficits in acute hemorrhage. In fact, loss of whole blood is not expected to change the hematocrit because the relative proportions of plasma and red cell volume are unchanged. **The decrease in hematocrit occurs when the kidney begins to conserve sodium (as described previously), which takes 8 to 12 hours to become evident.** Another factor that drops the hematocrit in acute hemorrhage is the administration of intravenous (asanguinous) fluids.

The administration of intravenous (asanguinous) fluids is expected to produce a dilutional decrease in the hematocrit, even in the absence of blood loss, and thus a decrease in the hematocrit during volume resuscitation is a dilutional effect, and it is not an indication of ongoing blood loss.




Arterial pH

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



If the pH is low (under 7.35) and the bicarbonate levels are decreased (<24 mmol/l), metabolic acidosis is diagnosed.

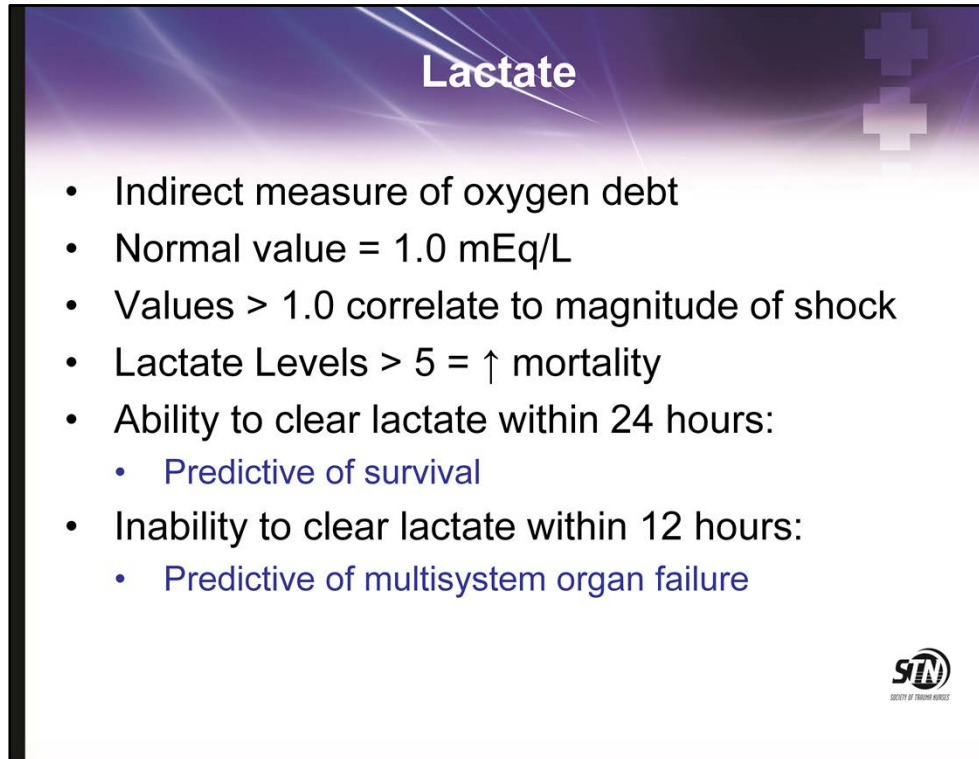
A pH < 7.20 is associated with generalized myocardial depression. Treatment with bicarbonate is rarely advocated, rather fixing the underlying injury is the recommended action.

Concerns regarding myocardial contractility with lactic acidosis are often cited as reasons for administration of sodium bicarbonate.

There have been several studies of the hemodynamic impact of sodium bicarbonate in human lactic acidosis.


Although sodium bicarbonate increased pH and serum bicarbonate concentrations, it did not improve hemodynamics or catecholamine responsiveness.

Specifically, the effects of bicarbonate were indistinguishable from saline with regard to heart rate, CVP, pulmonary artery pressure, mixed venous oxyhemoglobin saturation, systemic oxygen delivery, oxygen consumption, arterial blood pressure, pulmonary artery occlusion (wedge) pressure, and cardiac output.

A presentation slide titled "Lactate" with a purple gradient background and a white cross icon in the top right corner. The slide contains a bulleted list of points about lactate levels and their clinical significance. The STN logo is in the bottom right corner.

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



Lactate reflects an imbalance between glycolysis and glucose oxidation. Elevated levels are of concern, but the time to clearance of lactate back to normal levels is most predictive of outcome in hemorrhagic shock.


Values > 1.0 indicates magnitude of shock

Be aware that as under perfused tissue beds become reperfused, the accumulated lactate may be washed into the circulation.....causing a temporary elevation of serum lactate levels.

Ability to clear lactate to normal is the most important variable predicting survival after injury

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 is performed on blood gases and it is the amount of base required to titrate whole blood to a normal pH.

It is considered a reliable marker for shock and the need for transfusion.

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



The correlation of base deficit to mortality has been well studied.

It is a good test to run on critically unstable patients to follow until stable.

It is an easy to use clinical tool that is helpful in diagnosing shock.

Be aware that a moderate BD is at high risk for requiring blood.

International Normalized Ratio (INR)

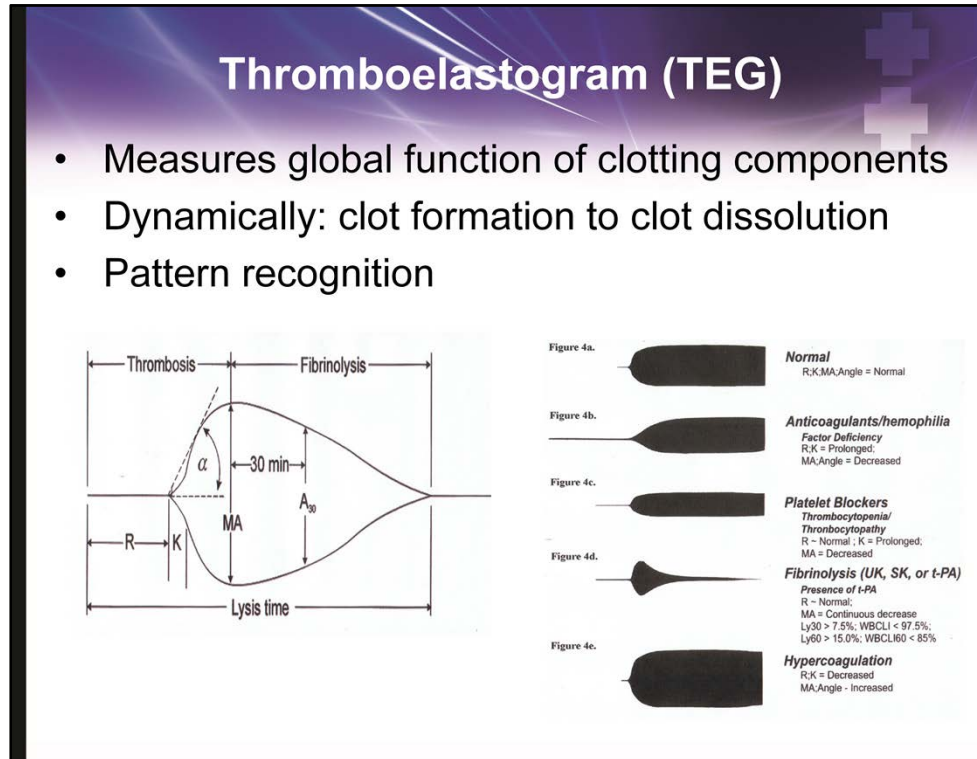
- Test of clotting (extrinsic pathway)
- Internationally accepted method of reporting prothrombin (PT) results worldwide

Population	Value
Normal	0.8 - 1.2
Anticoagulant Use	2.0 - 3.0
Trauma	> 1.5 = coagulopathy



What is the INR?

- Prothrombin time times vary across labs due to variations in the reagents used to perform the test.
- The INR was devised to standardize the results.
- INR is commonly used to monitor the effects of Warfarin.
- **It has applicability in trauma by it's ability to help identify : Trauma Induced Coagulopathy EARLY**



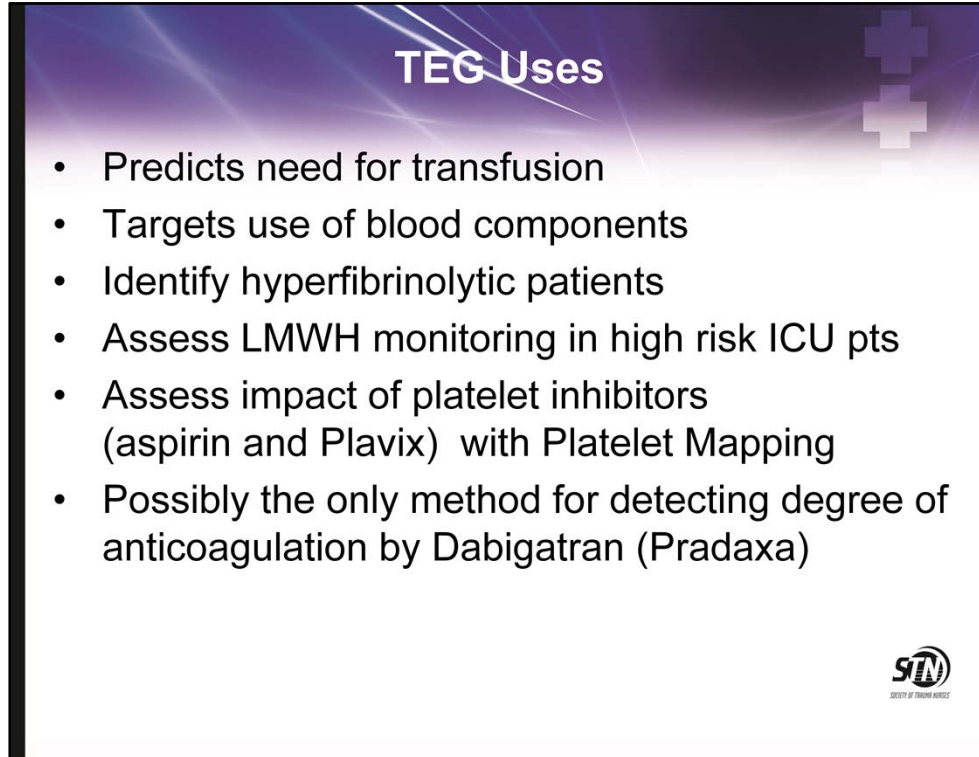
Thromboelastography (TEG) is a blood test which assesses global hemostatic and fibrinolytic function and has existed for more than 60 years.

Previously it has been used extensively in cardiac and transplant surgery. **The same test commonly used in Europe is called thrombelastometry (ROTEM).**

Improvements in TEG technology have led to increased reliability and speed which has increased its usage.


TEG monitors the whole **dynamic process** of hemostasis, from clot formation to clot dissolution, as opposed to the conventional endpoint tests, such as platelet count, protime, partial thromboplastin time, and fibrinogen.

The procedure allows for a global assessment of hemostatic function, beginning with the interaction of platelets with the protein coagulation cascade, from the time of initial platelet-fibrin interaction, through platelet aggregation, clot strengthening and fibrin cross linkage, to eventual clot lysis.



TEG Uses

- Predicts need for transfusion
- Targets use of blood components
- Identify hyperfibrinolytic patients
- Assess LMWH monitoring in high risk ICU pts
- Assess impact of platelet inhibitors (aspirin and Plavix) with Platelet Mapping
- Possibly the only method for detecting degree of anticoagulation by Dabigatran (Pradaxa)

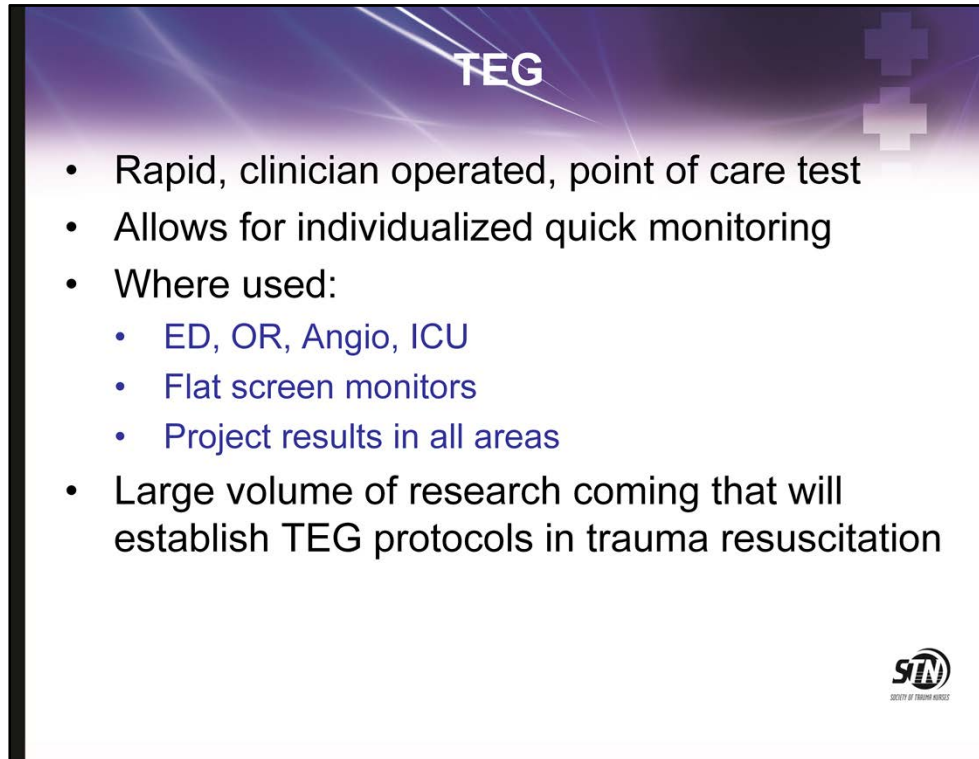

SOCIETY OF THROMBOSIS NURSES

TEG has use in both early resuscitation and in the ICU.

It allows for prediction for the need for transfusion, and the targeting of which blood component to use.

In the ICU, it can be used to identify the hyper-fibrinolytic patient.

It can also be used to assess low molecular weight heparin (LMWH) monitoring in high risk ICU patients.



The slide features a purple gradient background with the text 'TEG' in white at the top center. A list of bullet points is presented in black text, with some sub-bullets in blue. The STN logo is located in the bottom right corner of the slide.

- TEG
- Rapid, clinician operated, point of care test
- Allows for individualized quick monitoring
- Where used:
 - ED, OR, Angio, ICU
 - Flat screen monitors
 - Project results in all areas
- Large volume of research coming that will establish TEG protocols in trauma resuscitation

STN
SOCIETY OF TRAUMA NURSES

TEG has the advantage over traditional lab tests in that it can be done quickly at the bedside and interpreted by the clinician.

Full test results are known within 30-40 minutes. However, in most cases, the key data points (wave form) can be seen within 5-10 minutes.

Hospitals are embracing this technology and many have installed flat screen monitors on the walls of each area of resuscitation (ED, OR, ICU) where results can be displayed at all times.

It is estimated that in the future, TEG monitoring will largely replace traditional lab tests because of the speed needed during resuscitation and the fact that it provides individualized management of the patients care.



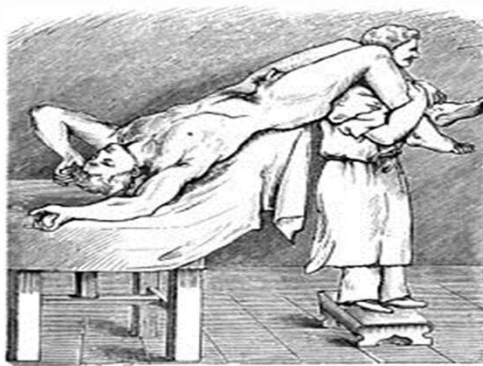
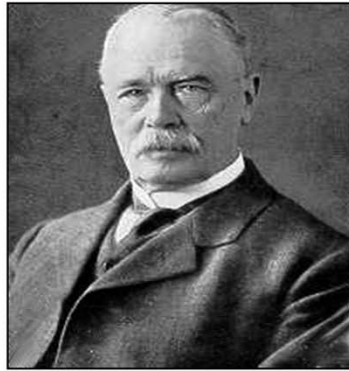


Airway and breathing should be taken care immediately

Rule out tension pneumothorax as the initial cause of the hypotension

Is There a Shock Position?

- Dr. Friedrich Trendelenburg 1800's
- To improve surgical exposure - pelvic organs



No Benefit in Shock




The Trendelenburg position was originally used to improve surgical exposure of the pelvic organs. It is credited to German surgeon Friedrich Trendelenburg (1844-1924).

After World War I, use of the Trendelenburg position became common practice in managing patients with shock. **There is no research to support its use in shock.**

Mechanical Means of Stopping Hemorrhage


Pelvic Binders

- Reduce pelvis volume
- Tamponade effect



Tourniquets

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



Pelvic fractures are a significant cause of mortality after blunt trauma. Early control of hemorrhage is associated with improved outcomes. Application of a binder or sheet significantly reduces the potential space for bleeding and provides a tamponade effect.


Tourniquets

Hemorrhage from arterial injuries involving the extremities have been difficult to control. Recent experience in the military have proven that tourniquets are safe and effective. Use of tourniquets has allowed patients to arrive to the hospital without massive bleeding. The use of tourniquets has not been associated with increased delayed amputation rates or the development of compartment syndrome.

Mechanical Means of Stopping Hemorrhage

Hemostatic Dressings

- Research advancing quickly
 - Made of volcanic rock, clay, shells
- Actions:
 - Direct compression
 - Activation of clotting
 - Adhesion
- Utility
 - Speed of application (under fire)
 - Pliable, Z Fold conformation



The images show three types of hemostatic dressings. The top image shows a box of Celox Gauze and a Z-fold dressing. The middle image shows a box of QuikClot Gauze. The bottom image shows a Z-fold dressing.

Hemostatic dressings are key to avoiding coagulopathy and controlling bleeding early.

Primarily used for nonextremity hemorrhage, but also useful in severely mangled limbs.



Currently they are used in the military but will eventually expand to civilian use.

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



PIV=peripheral intravenous
ExtJug=external jugular
IO= intraosseous

IV Access is dependent on:

- Magnitude of injury
- Anatomical locations available
- Skill and expertise of the provider

In general the rank order for attempted IV placement in shock are as follows:

1. Peripheral IV (antecubital)
2. Femoral Vein
3. Subclavian

External jugular is another option, but interferes with c collar placement.

Recommend: IV above & below diaphragm

Avoid IV Access

- Injured limb
- Distal to possible vascular wound
- Femoral access with injury below diaphragm





Avoid placing IV in locations where there is suspected injury in the proximal vasculature.

Avoid femoral access in major abdominal injury. At least one IV line should be placed in a tributary of the superior vena cava, as there may be vascular disruption of the inferior vena cava.

IV Access in Shock

- **Femoral Vein**
 - 8.5/9.0 French Introducer
 - Side port removed ↑ flow rate
 - Out of the way of intubation or chest procedures
- **Subclavian/Internal Jug**
 - Higher risk (pneumothorax)
 - Lower success rate
 - In chest injuries, place on side of injury



Percutaneous Femoral Vein


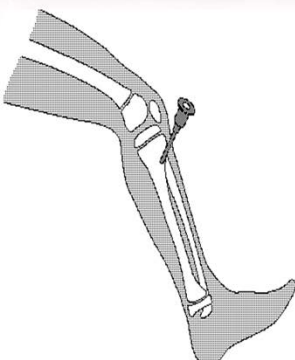
- Commonly used in major trauma resuscitations because it allows multiple providers to give care at the same time.
- It gets the provider out of the way of the people performing intubation and or chest procedures.
- When using 8.5 French PAC introducer **remove side port as this increases the resistance roughly four fold**

Subclavian / Internal Jugular

- Not routinely used in hypovolemic trauma patients
- Incidence of complications is higher
- Rate of success is lower due to venous collapse and it is operator skill dependent
- Insert on side of injury in patients with chest wounds, reducing the chances of collapse of the uninjured lung, especially if a chest tube is already in place

Intraosseous Devices

- Temporary access
- Children & adults
- Insert within 1 minute
- Manual or power drill
- Prox tibia/humerus/sternum
- Avoid fracture /injury sites
- Good for fluid/blood/meds
- Flow rates up to 125 mL/min w pressure bag
- Risk: extravasation → compartment syndrome



Three intraosseous devices have been approved by the Food and Drug Administration for use in adult trauma patients when intravenous access cannot be obtained.

Sites of insertion are the sternum (FAST1), proximal tibia and humerus (Big Injection Gun), and proximal and distal tibia and humerus (EZ-IO).

Insertion generally requires less than 1 minute, and flow rates up to 125 mL/min can be achieved.

The devices are used for emergency resuscitation and should be removed within 24 hours of insertion or as soon as practical.

Contraindications include fractures or other trauma at the insertion site, prosthetic joints near the site, previous attempts to insert an intraosseous device at the same site, osteoporosis or other bone abnormalities, infections at the proposed site, and inability to identify pertinent insertion landmarks.

Primary complications are extravasation of medications and fluids into the soft tissue, fractures caused by the insertion, and osteomyelitis. (Critical Care Nurse. 2011;31:76-90)

Pre Hospital IV Placement in Trauma? EAST 2009 Guideline

- **No evidence to support IV placement at scene**
- Enroute OK
- Limit 2 attempts → I.O.
- Saline lock/Keep open
- Avoid continuous IV
- Use small boluses (250cc)
- Titrate to palpable radial



There is no evidence of benefit placing an IV at the scene for either blunt or penetrating trauma. Multiple studies have shown that it delays scene time and may actually be harmful in trauma.

The use of intraosseous access is recommended in trauma patients requiring vascular access in which IV access is unobtainable or has failed 2 attempts, **but only if personnel are trained in its use.**

Despite the widely held belief that prehospital venous access placement and fluid resuscitation is standard of care, there is little data to support this practice. In fact, an increasing amount of data suggests that it may be quite harmful to a significant number of critically injured patients.


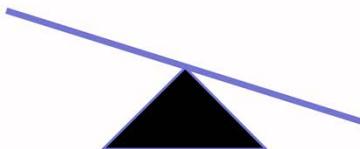
The EAST committee has found that placement of venous access at the scene delays transport and placement of access en route should be considered. In those patients in whom intravenous access has failed, intraosseous may be attempted if equipment and trained personnel are available. There is insufficient data to suggest that blunt or penetrating trauma patients benefit from prehospital fluid resuscitation.

Fluid Resuscitation



Fluid Administration Balance

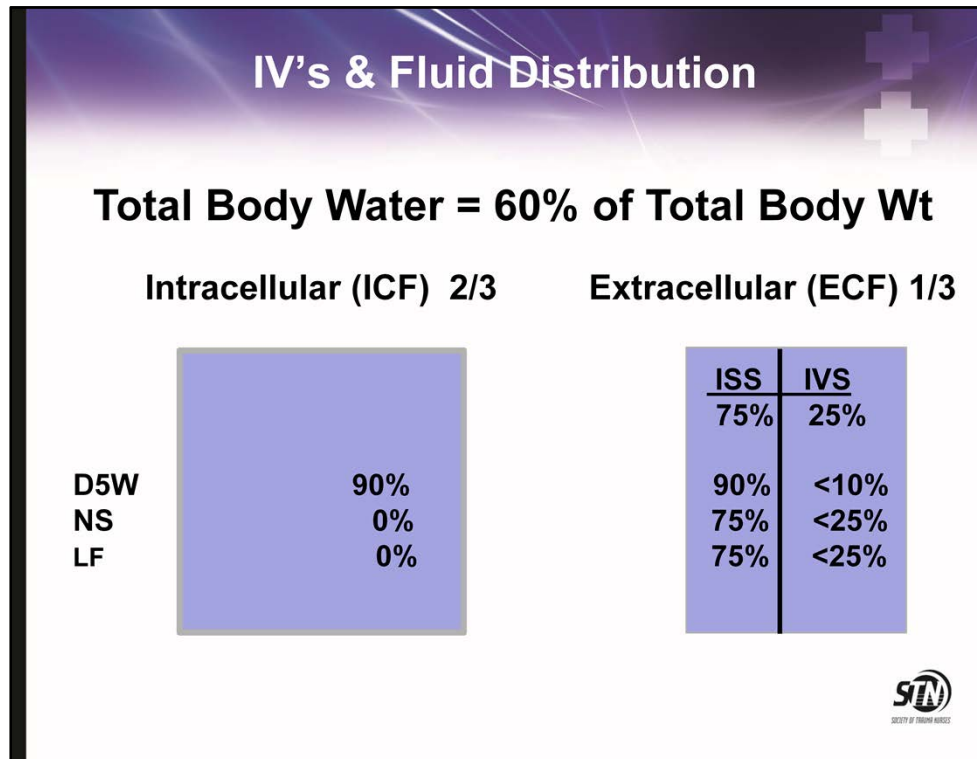
- **Too little...**
 - Ongoing shock
 - Continued acidosis
 - Coagulopathy
 - Myocardial dysfunction
 - Renal failure
 - Death
- **Too much...**
 - Increased bleeding
 - Clot disruption
 - Dilution coagulation factors
 - Compartment syndromes
 - Transfusion concerns
 - Inflammation
 - Immunosuppression
 - Transfusion Related Acute Lung Injury (TRALI)



The correct balance of fluid administration is narrow.

Many fear under resuscitating which can lead to death.

As a result, for the past 20 years, heavy fluid administration has resulted in severe complications such as clot disruption, further bleeding, compartment syndromes and TRALI with minimal improvement in outcomes.



Total body water = 60% of body weight. In a 70 kg man = $0.6 \times 70 = 42$ L

Distributes further into: Intracellular fluid (ICF) (425 ml/kg or 2/3) and Extracellular fluid (ECF) (175 ml/kg or 1/3) .



The extracellular fluid is further divided into the Interstitial Space (ISS) and the Intravascular space (IVS) which is where plasma volume expansion occurs.


Key Points:

- Note that of the total body water the vast majority (2/3's) resides inside of cells
- Note that of the extracellular water only 25% resides in the intravascular space (IVS)
- Note that of LR and NS only 25% remain in the intravascular space.

What you administer determines distribution!!

NS vs. LR

<u>Normal Saline</u>	<u>Lactated Ringers</u>
<ul style="list-style-type: none">• Na,Cl• Fluid of choice for blood• Con:<ul style="list-style-type: none">• Hyperchloremic acidosis	<ul style="list-style-type: none">• Na, Cl, K, Ca, Lactate• Fluid of choice per ATLS• Con:<ul style="list-style-type: none">• Immune modulation
	



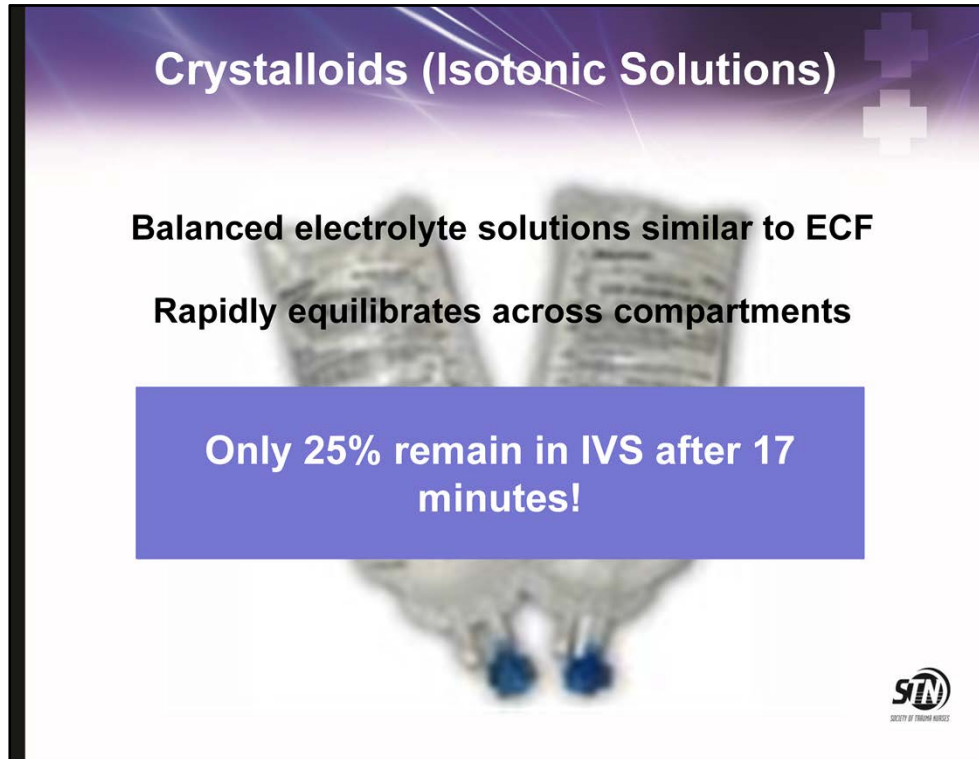
The lactate in LR acts as a buffering solution and is ideal for trauma patients in shock. Lactate is converted to bicarbonate in the liver which buffers acidosis. Even in low flow states and or cirrhosis lactate is converted.

Large volumes of normal saline may be deleterious because this fluid is actually slightly hypertonic and can predispose the patient to hyperchloremic metabolic acidosis.

LR has been implicated in activating neutrophils and potentially priming the immune system but is felt to be more a theoretical than practical concern.

It is generally believed that LR is better in trauma than NS because it provides a better buffer for metabolic acidosis.

In practical terms, it is advisable to have more than one IV and hang both NS and LR in trauma resuscitations.



Fluid resuscitation typically begins with isotonic crystalloid solutions (fluids similar to extracellular fluid (ECF), such as normal saline and lactated Ringer's solution).

These solutions have the advantage of being inexpensive, plentiful, and easy to administer.

For patients who are hypovolemic (rather than hemorrhaging), isotonic crystalloids may be all the fluid that is required.

These solutions do not linger long in the bloodstream, with rapid equilibration of administered volume across the interstitial and intravascular compartments and relatively rapid transit into the cells.

Patients with normal renal function can tolerate relatively large amounts of administered crystalloid solution without harm and will maintain normal blood chemistry.

Small Volume Resuscitation Paradigm Shift

- Using hypertonic/hyperosmotic fluid
- Remains in vascular space longer
- Restores vascular volume
- Without flooding patient
- Started by military → civilian trauma

Examples:


- Hetastarch (Hespan/Hextend)
- Hypertonic Saline (3% to 7.5%)



The military have extensive experience with 7.5% hypertonic saline or Hespan in 250 ml boluses. These are ideal as they can't logistically carry liters of fluid in the field.


Small Volume Resuscitation: Hetastarch/Hespan/Hextend

- Plasma volume expander
- 500cc hetastarch expands blood volume 800cc



The diagram consists of two blue ovals. The left oval contains the text '500ml Hetastarch'. The right oval contains the text '2-3 L LR'. A blue double-headed arrow connects the two ovals, with the word 'Equivalent' written in the center of the arrow.

- Safe and effective at 500cc bolus
- Cons:
 - May cause coagulopathy in large doses (>2L dose)
 - Renal tubular dysfunction concern



When crystalloid is infused, it undergoes an exponential departure from the intravascular space with a half-life of only 17 mins, yielding an eventual distribution of intravascular to interstitial fluid of between 1:3 and 1:10. This is why one must infuse a much larger volume of crystalloids than the perceived blood loss to stabilize MAP in operative hemorrhage.

Current thinking is to reduce infusion and perhaps administer as boluses rather than continuous flow.

The literature is changing quickly in this area.

500 cc of Hetastarch is equivalent of administering 2-3 Liters of LR


**Small Volume Resuscitation
Hypertonic Saline**

Type:
3.0% and 7.5% Sodium Chloride

Action:
Rapidly pulls fluid from tissues into bloodstream
Stabilizes BP & CO and controls ICP

250 ml ~ 1 Liter
NS or LR

Results:
Large RCT unable to show survival benefit

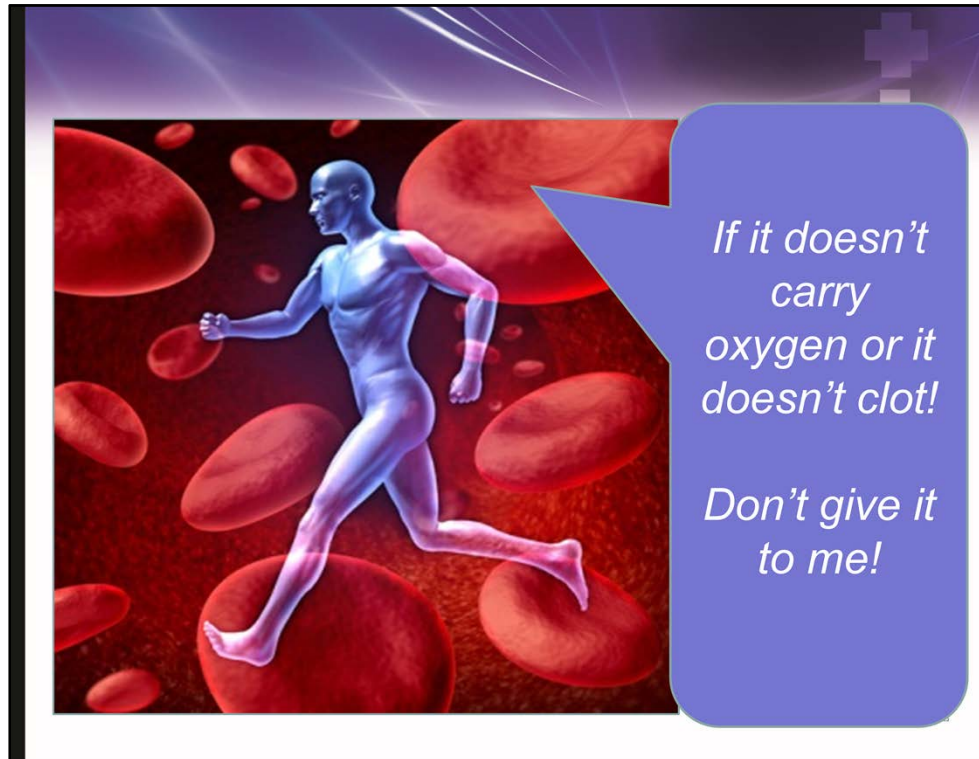


Hypertonic solutions (i.e., 3% or 7% NaCl) act like magnets, drawing fluid from tissues into the bloodstream, and thereby rapidly increasing circulating volume.

A 250 ml bolus is equivalent to the administration of 1 liter of NR or LR

Individual studies show encouraging results. However a 2009 Cochran meta analysis was unable to show a survival benefit (yet).

Continued research is ongoing.

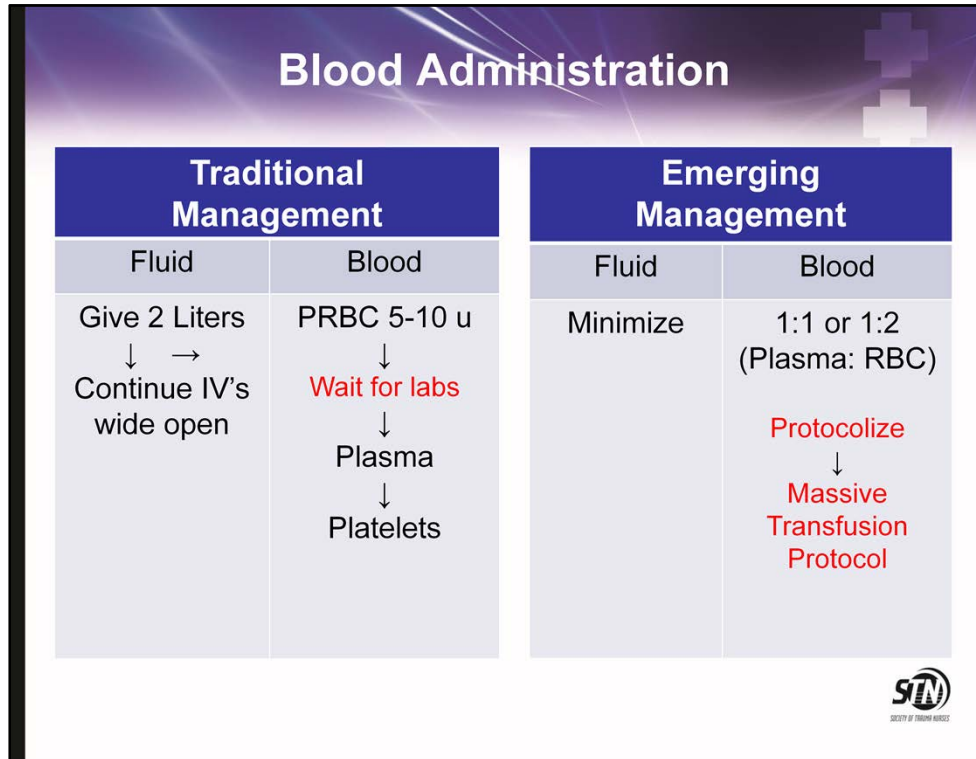


In the presence of hemorrhage, IV fluids without blood transfusion will only make things worse.

If blood is lost, then blood must be replaced with blood.

	Packed Red Blood Cells	Plasma	Platelets
Action	Carries Oxygen No clotting factors	Coagulation Factors	Aggregation
1 unit	~300 ml (Hct 55%)	~250 ml	~25 ml individual unit ~150 pooled unit
Dose	↑ Hgb by 1 g/dl ↑ Hct by 3 % In the non-bleeding pt	↑ coags by 2.5% (Need at least 4 u for significant change)	1 unit Apheresis (pooled) ↑ 25,000-50,000 per u
Storage	-4 C <u>Progression:</u> Emerg Uncrossmatched (immediate) Type Specific (20 min) Cross Matched (60 min)	<u>Non Trauma Center</u> <ul style="list-style-type: none"> Frozen thaw time 2 u in 30 minutes <u>Trauma Center</u> <ul style="list-style-type: none"> Room Temp Good for 5 days Monitor wastage 	Room temp Agitated

A review of blood components is helpful in understanding the differentiation of components that form whole blood.

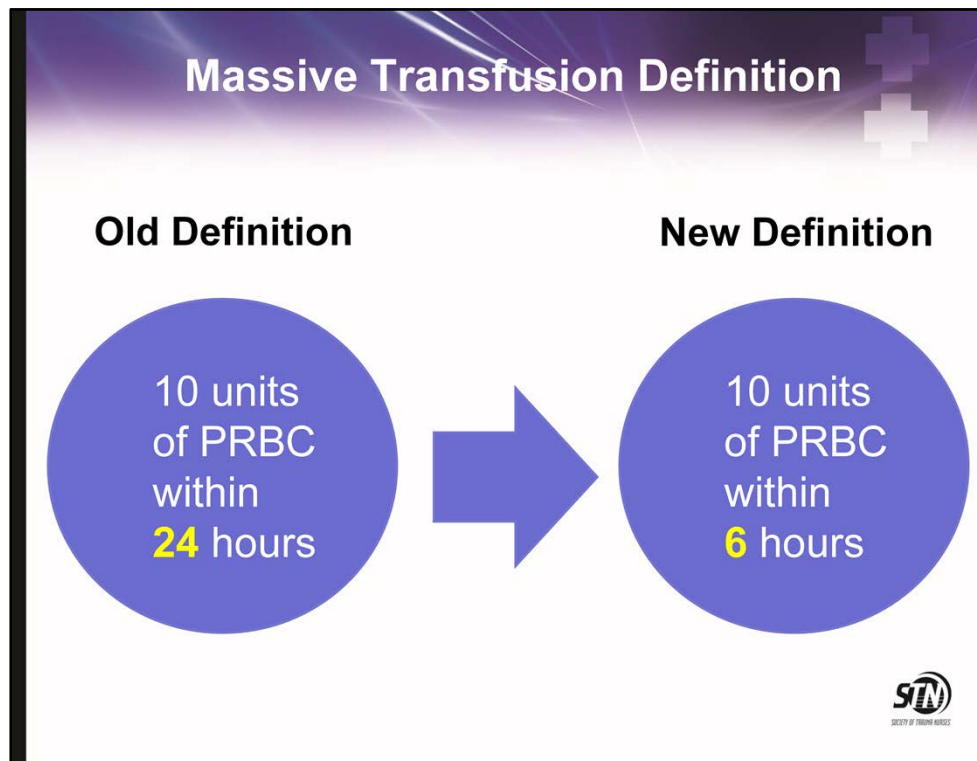


Traditional management included 2 Liters of crystalloid. If the patient had not stabilized then fluids were continued and packed red blood cells were started. Labs were drawn and they dictated blood component usage, but often lagged behind the patient's dynamic response to resuscitation.

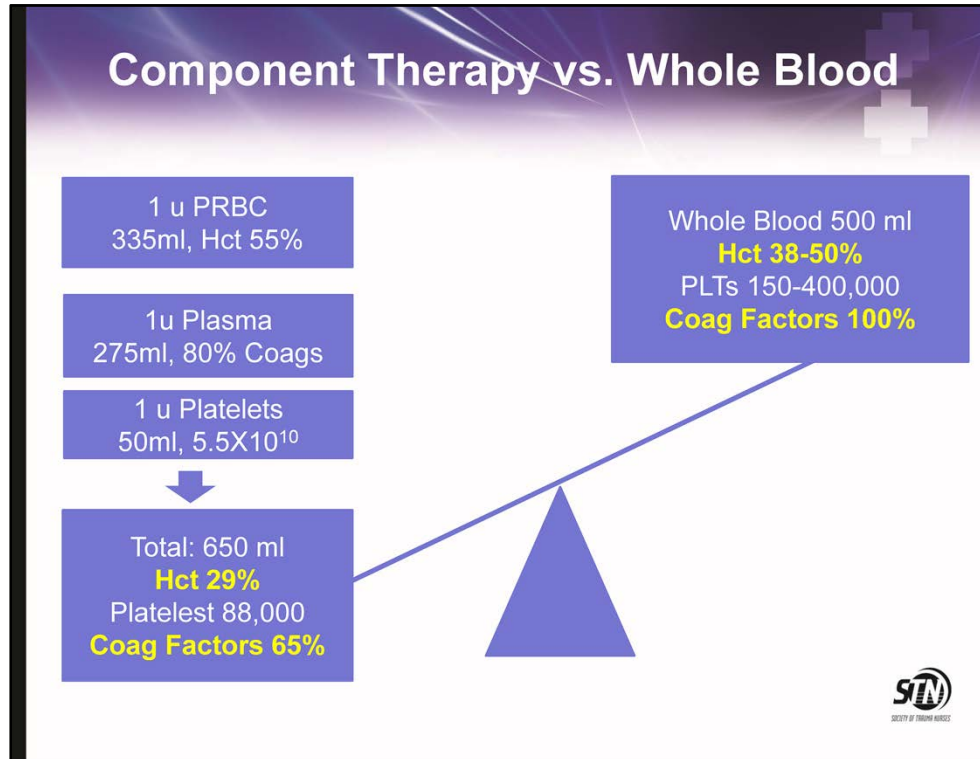
Plasma and Platelets were often not given until after 10 or more units of PRBC were given. Meanwhile excessive fluid was also given which diluted coagulation factors even further.

Emerging management learned from the military includes minimizing fluids while administering blood components in a configuration that mimics whole blood based on protocol rather than lab values.

The ideal ratio of Plasma to RBC is not yet known but is presumed to be around 1 Plasma for every 1-2 units of blood.



The traditional MT definition of 10 units of PRBC within 24 hours of hospital admission is no longer felt to be accurate. Several authors have suggested changing it to 10 units within 6 hours.

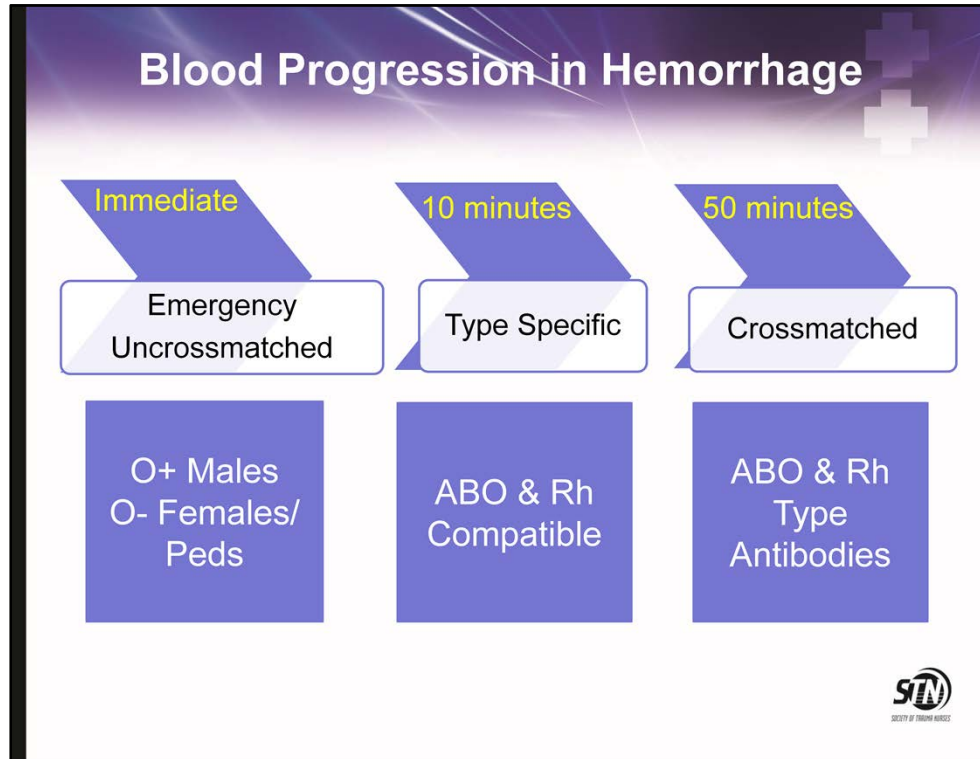


Note the difference in Hct and Coagulation factors between component therapy vs. whole blood.

Whole blood is far superior in trauma but unavailable for distribution in the US which developed a component based system in the 1970's.

The military however has ready access to a whole blood donor pool by having soldiers donate on a regular basis.

The improved results (data) from the military on the use of whole blood in hemorrhaging trauma has prompted civilian trauma centers to develop Massive Transfusion Protocols that mimic whole blood with 1:1:1 (Plasma:RBC: Platelets).



Emergency Uncrossmatched Blood:

Ideally in the resuscitation room prior to patient arrival

O Positive blood is reserved for male trauma. It is roughly 36% of the donor pool and is therefore more readily available.

O Negative blood is reserved for childbearing females and children. It is a more scarce blood supply (roughly only 8% of the donor pool)

Hurley Medical Center
Flint, Michigan
Trauma Service

Massive Transfusion Tracking Sheet
 Numerical Order of Transfusions

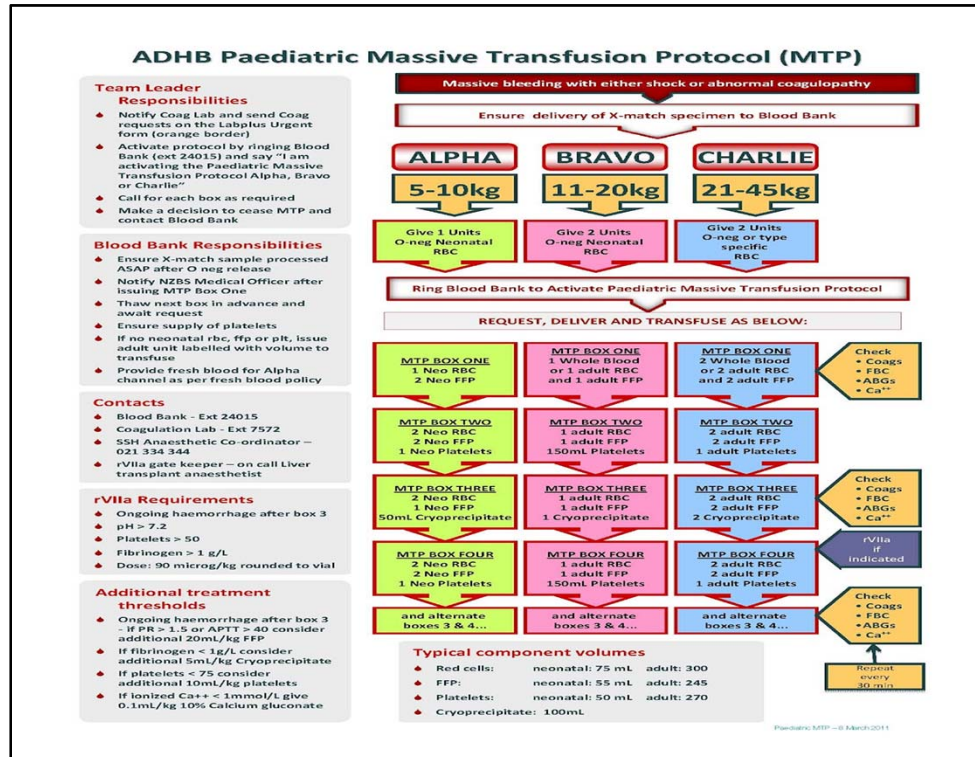
Suggested Use: Follow the numbers, cross off units as you give them & doc time given next to each #

Chest Shipment		RBC's	Thawed Plasma	PLT's 1 pooled Apheresis unit 1 pooled unit = 6-8 single units	CRYO 2 pooled units 1 pooled unit = 5 single units	rFVIIa Dose 6 mg dose for > 100 kg 5mg dose for 65-100 kg (Pediatric dosing per physician)
Class I Activation	Chest 1	1 3 5	2 4* →	1 Gm Bolus Tranexamic Acid (TXA) Followed by 1Gm drip over 8 hours		
	Chest 2	7 9 11	8 10 12			
	Chest 3	14 16 18	15 17 19	13		
Massive Transfusion	Chest 4	23 25 27	24 26 28		20, 21	22 Consider
	Chest 5	30 32 34	31 33 35	29		
	Chest 6	39 41 43	40 42 44		36, 37	38 Consider
	Chest 7	46 48 50	47 49 51	45		
	Chest 8	55 57 59	56 58 60		52, 53	54 Consider
	Chest 9	62 64 66	63 65 67	61		
	Chest 10	71 73 75	72 74 76		68, 69	70 Consider
	Continue as necessary					
	* Remember to deactivate the massive transfusion guideline over the trauma radio when finished					

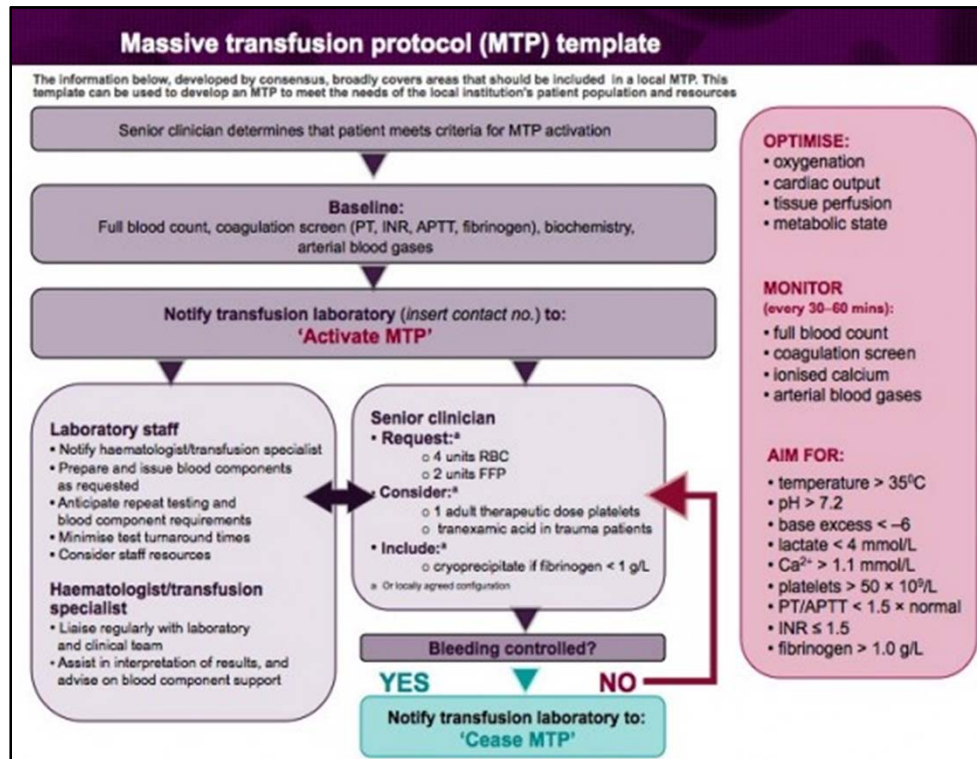
Judy Mikhail 2010

Here is an **example** of a Massive Transfusion Protocol.

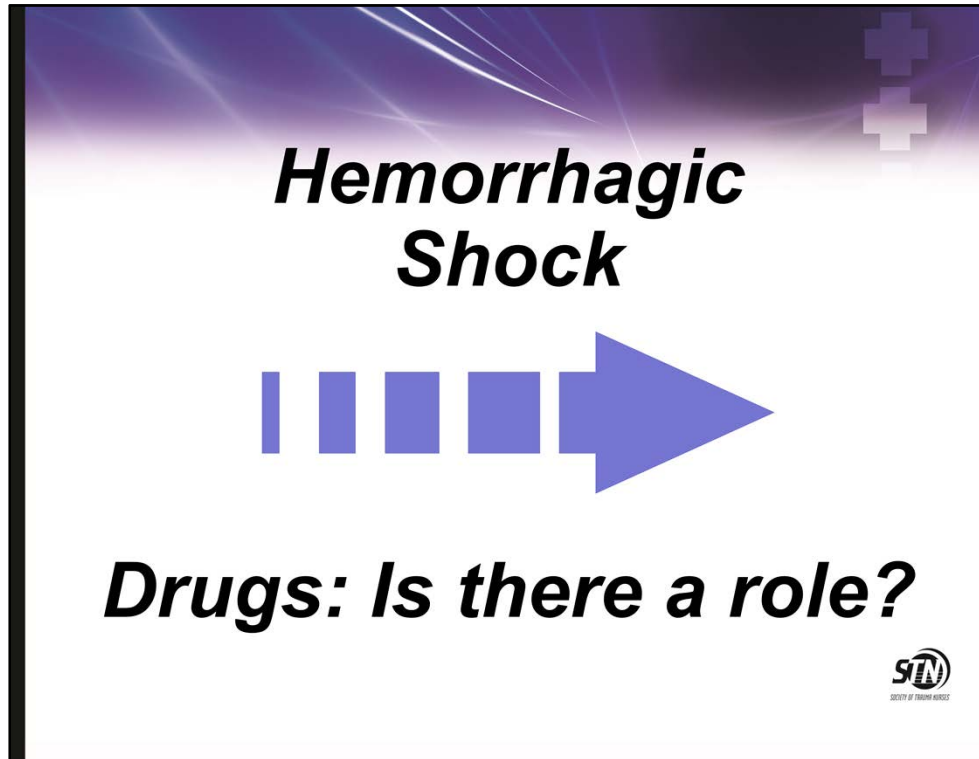
Note the ease of use for nurses in a resuscitation by just following the numbers.



Massive Transfusion Protocol example for pediatric trauma.



Yet another example of a massive transfusion protocol.



What is the role of drugs (if any) in hemorrhagic shock?

Two drugs have been identified as having roles in the treatment of hemorrhagic shock.


1. Factor VIIa
2. Tranexamic acid (TXA)

Recombinant Factor VIIa NovoSeven

- Refractory bleeding in trauma
- Activates Extrinsic Pathway
- Off label use in trauma
- Research Results in Trauma:
 - Numerous anecdotal reports
 - 1 RCT published trauma:
 - ↓ blood use
 - ↓ MSOF ↓ ARDS
 - Trend toward ↓ mortality
 - No ↑ thrombotic events

Correct before use:

- Hypofibrinogenemia:
 - Give Cryoprecipitate
- Thrombocytopenia
 - Give Platelets
- Hypothermia
 - Correct Temperature
- Acidosis
 - Consider Bicarbonate



The use of Factor VIIa remains a topic of considerable debate.

Numerous anecdotal reports reveal support of Factor VIIa in trauma.

One large randomized control trial (RCT) has been completed and showed decreased blood use, incidence of MSOF and ARDS, but not a significant change in mortality.


There is debate also about the timing of the drug and selection of patients to receive the drug.

The drug appears to be less effective in the setting of acidosis, but remains effecting in all but the most severely hypothermic settings.

Recent reports have noted a concern for increased thromboembolic events. This should be weighed against the need for the drug.

Factor VIIa

- Include in Massive Transfusion Protocol:
 - Do not use too early or too late
 - Administer between 8 - 20 PRBC's
 - Recommended dose: 100 mcg/kg
 - Expensive:
 - $100\text{mcg} \times 70\text{kg} = 7,000\text{mcg} = \$7,700$
 - Repeated at 1-2 hour intervals if required



Placement of Factor VIIa in the Massive Transfusion Protocol is under debate. It should not be placed too early or too excessively late.

Recommendations are to administer the drug somewhere between the 8-20 units of PRBC.

The drug is very expensive and should be used with deliberation.

Tranexamic acid (TXA)

- Derivative of AA Lysine - **inhibits fibrinolysis**
- Inexpensive (\$80/dose) and proven safety profile
- **Cochrane review (2007)** 53 RCT's Cardiac/Ortho
 - Sig reduction in bleeding without thrombotic complications
- **CRASH2 trial (2010)** Prospective RCT, > 20,000 pts
 - Stat sig 1.5% reduction in mortality (overall)
 - Subgroup analysis (Severe bleeding & early admin)
 - Reduced bleeding by 30% **IF** given within 1 hour
- **MATTERs trial (2011)** Camp Bastion in Afghanistan
 - Marked improvement in survival in most severely injured compared to those who did not receive it
- Soldiers to carry autoinjectors on battlefield



Tranexamic acid is a synthetic derivative of the amino acid lysine that exerts its antifibrinolytic effect through the reversible blockade of lysine binding sites on plasminogen molecules.

CRASH 2 Trial


- The CRASH-2 trial was an international randomized controlled trial of the early administration of tranexamic acid (TXA) to bleeding trauma patients.
- The trial recruited 20,211 patients from 274 hospitals in 40 countries.
- The results show that TXA reduces mortality in trauma patients with or at risk of bleeding, with no apparent increase in side effects.
- **If given within three hours of injury**, TXA reduces the risk of death due to bleeding by about a third.
- TXA administration has been shown to be highly cost-effective in high, middle or low income countries

MATTERS Trial:

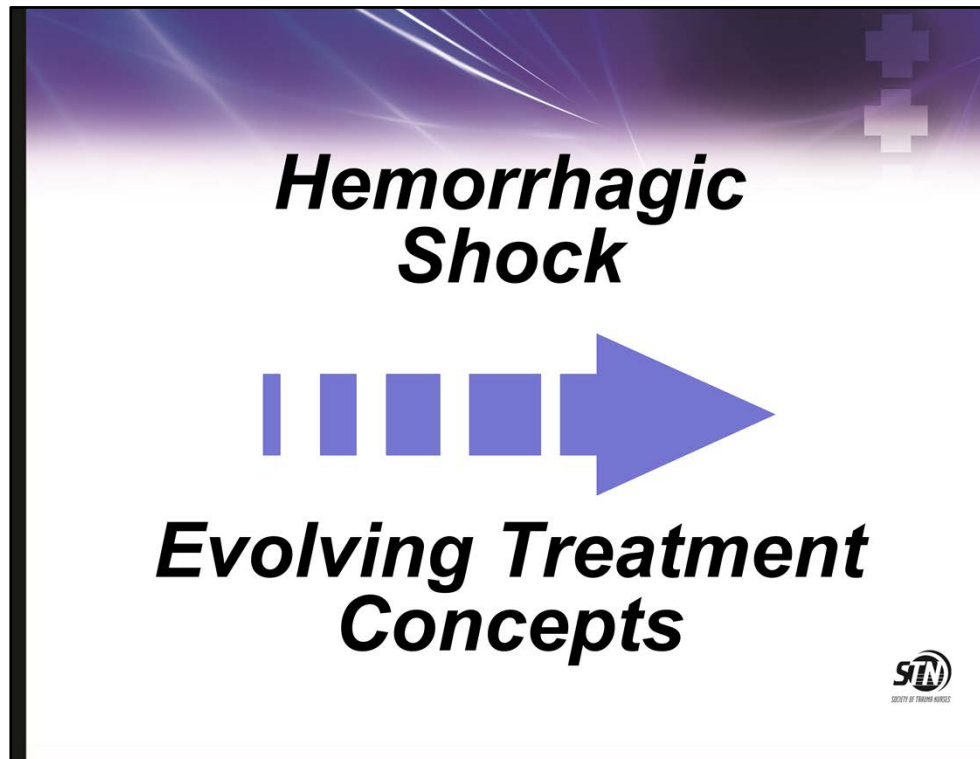
- The use of TXA with blood component based resuscitation following combat injury results in improved measures of coagulopathy and survival, a benefit that is most prominent in patients requiring massive transfusion.
- Treatment with TXA should be implemented into clinical practice as part of a resuscitation strategy following severe wartime injury and hemorrhage.

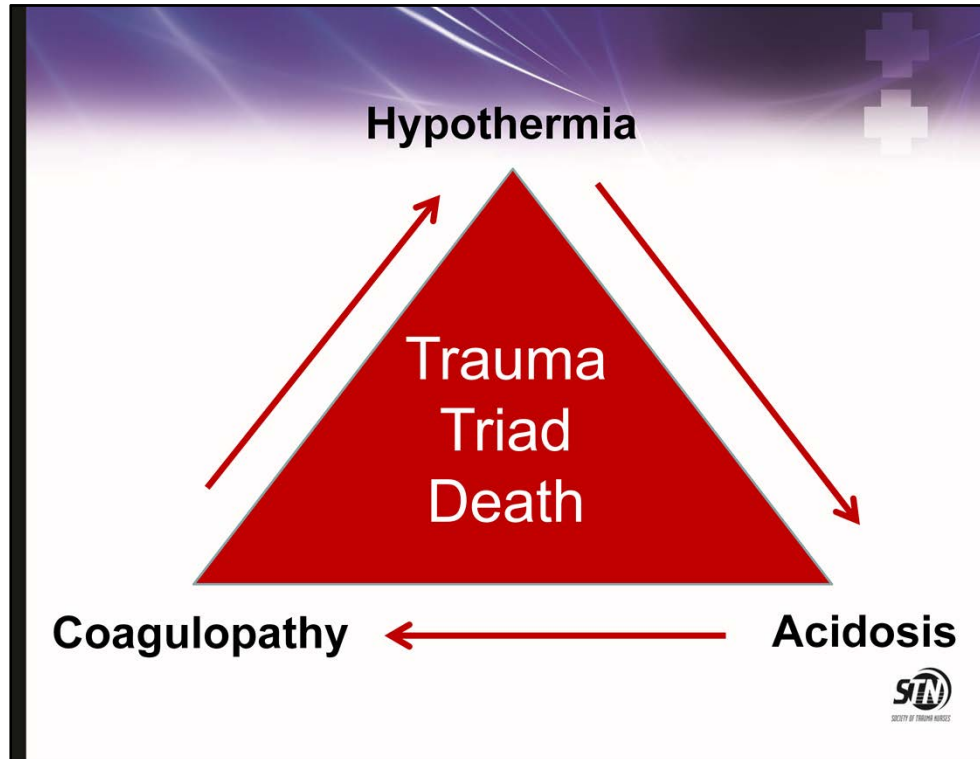
Tranexamic Acid (TXA) Example Protocols

<u>Military Protocol</u> <ul style="list-style-type: none">• Give within 1-3 hours of injury• 1 unit of blood• 1 Gm of Bolus of TXA• 1 Gm Infusion over 8 hrs	<u>Oregon Health & Science University Protocol</u> <ul style="list-style-type: none">• MTP activated• Pt has received > 4 units within 2 hours• Give 1 Gm bolus• Start 1 Gm drip over 8 hrs
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Examples of current protocols for use of Tranexamic Acid





There is a recognition that the onset of hypothermia, acidosis and coagulopathy in trauma place the patient at high risk for death.

The three conditions share a complex relationship; each factor can compound the others, resulting in high mortality if the cycle continues uninterrupted.

Severe hemorrhage in trauma diminishes oxygen delivery, causing hypothermia.

This in turn can halt the coagulation cascade, preventing blood from clotting.

In the absence of blood-bound oxygen and nutrients (hypoperfusion), the body's cells burn glucose anaerobically for energy (lactic acidosis), which in turn increases the blood's acidity causing metabolic acidosis.

Such an increase in acidity can reduce the efficiency of the heart muscles (myocardial performance), further reducing the oxygen delivery.

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

Moderate to Severe Hypothermia Occurs In <10% of Trauma



Hypothermia occurs when heat loss exceeds body's ability to generate heat. Coagulation activity decreases by 10% for every 10 degree temperature drop. But the greater negative action is the effect on platelet function.

There are many predisposing factors:

Environmental

- skin exposure, wet clothing, external temp, air movement

Extremes of age

- very young, elderly

Injuries

- Burns, open wounds, head injury, spinal cord injury

Meds

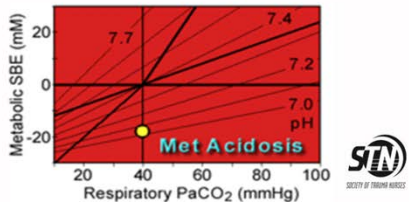
- Alcohol, Muscle relaxants, Sedative hypnotics, Anesthetic agents

Treatment related

- Body cavity exposure
- Fluid resuscitation
- Massive transfusions
- Immobilization

Acidosis

- **Effects:**
 - Altered hemostasis
 - Myocardial depression
- **Correlates with:**
 - Depth of shock
 - Degree of tissue injury
- **Assessed:**
 - pH
 - Base Deficit
 - Lactate
- **pH < 7.2**
- **Initial BD ≥ 6**
 - Predicts transfusion
 - Increased ICU days
 - Risk for MSOF
- **Initial BD ≥ 7.5**
 - \uparrow mortality



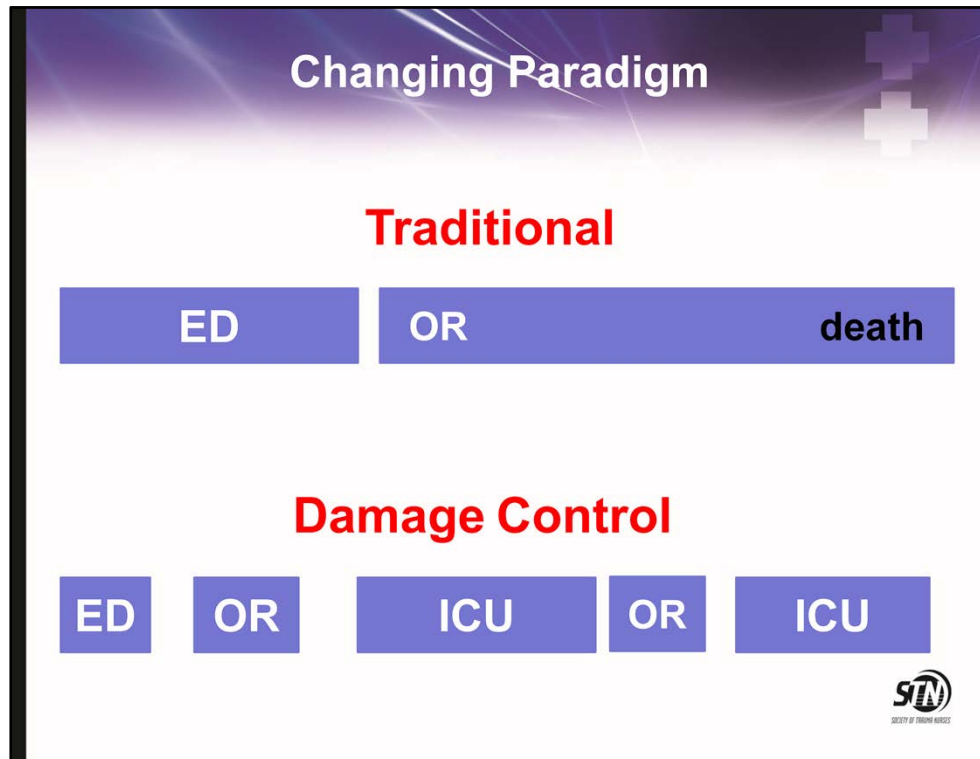
Refractory acidosis is a marker of physiologic derangement.

Arresting hemorrhage and correcting the underlying injury is the optimal treatment.

Acidosis is part of the triad that promotes coagulopathy. As pH drops below 7.2 there is a 20% prolongation of the PT and PTT

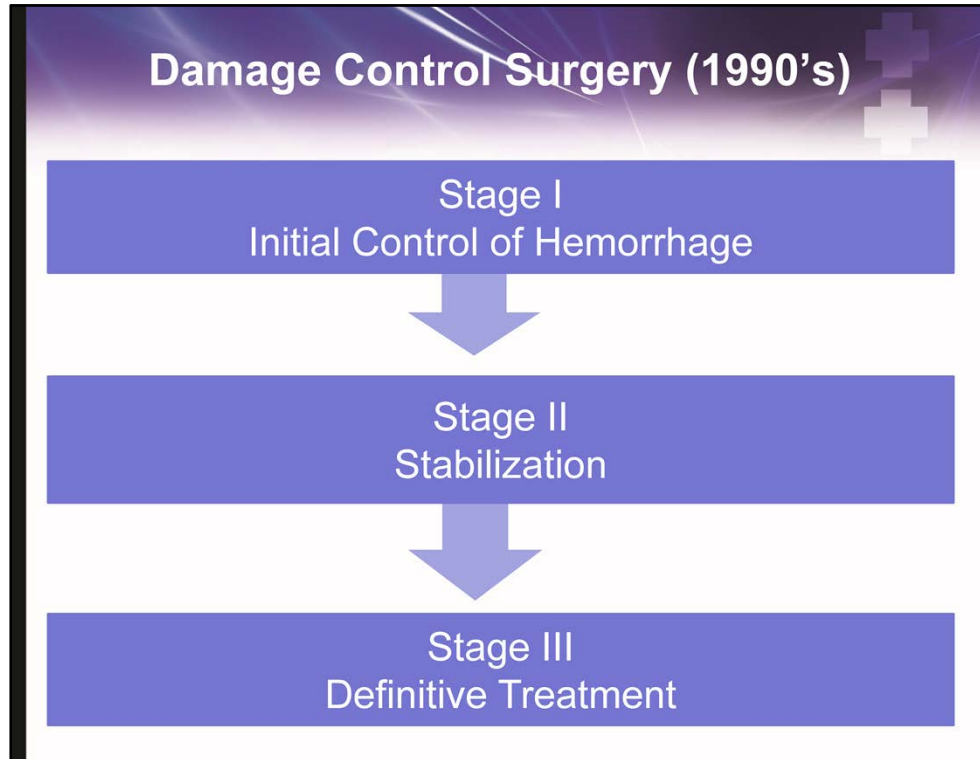
Acidosis correlates to the depth of shock and tissues injury.

It is most often assessed by ABG, BD and Lactate



The old way of doing things was to resuscitate first and then move to surgery.

Now the resuscitation and surgery are undertaken simultaneously.

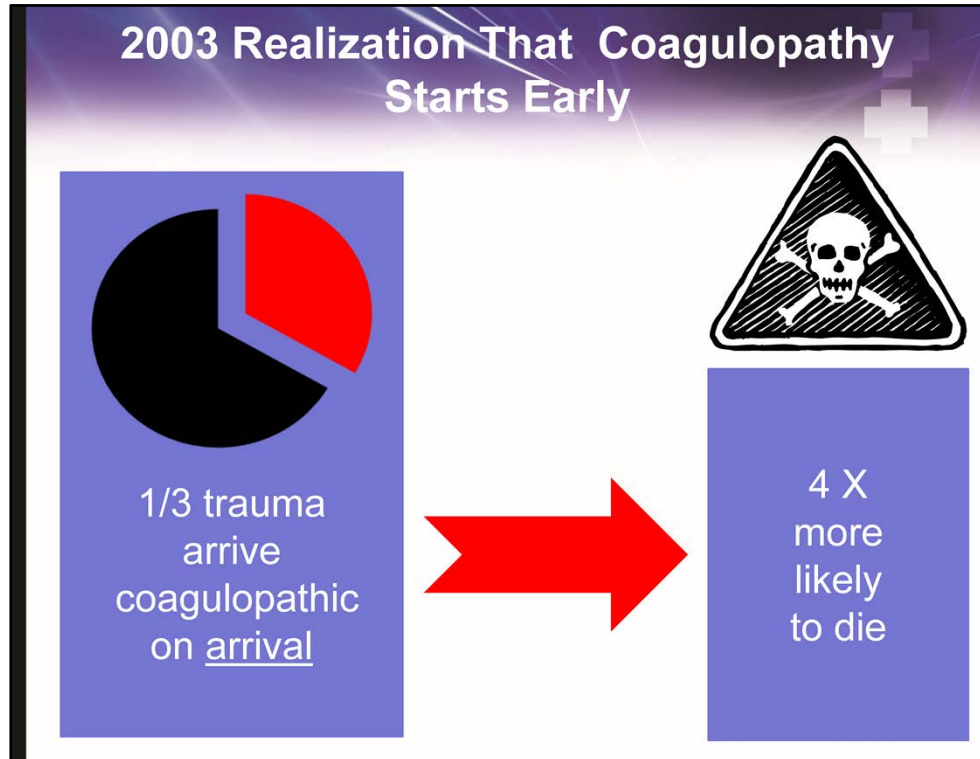


Damage Control Surgery was first popularized in the late 1990's resulting in subsequent improvement in hemorrhage outcomes.

Stage I: stop the bleeding, remove major contaminants, consider leaving open to avoid abdominal compartment syndrome, abort surgery early

Stage II: transfer to ICU, resuscitate further attempting to normalize BP, Temperature, and normalizing coagulation factors

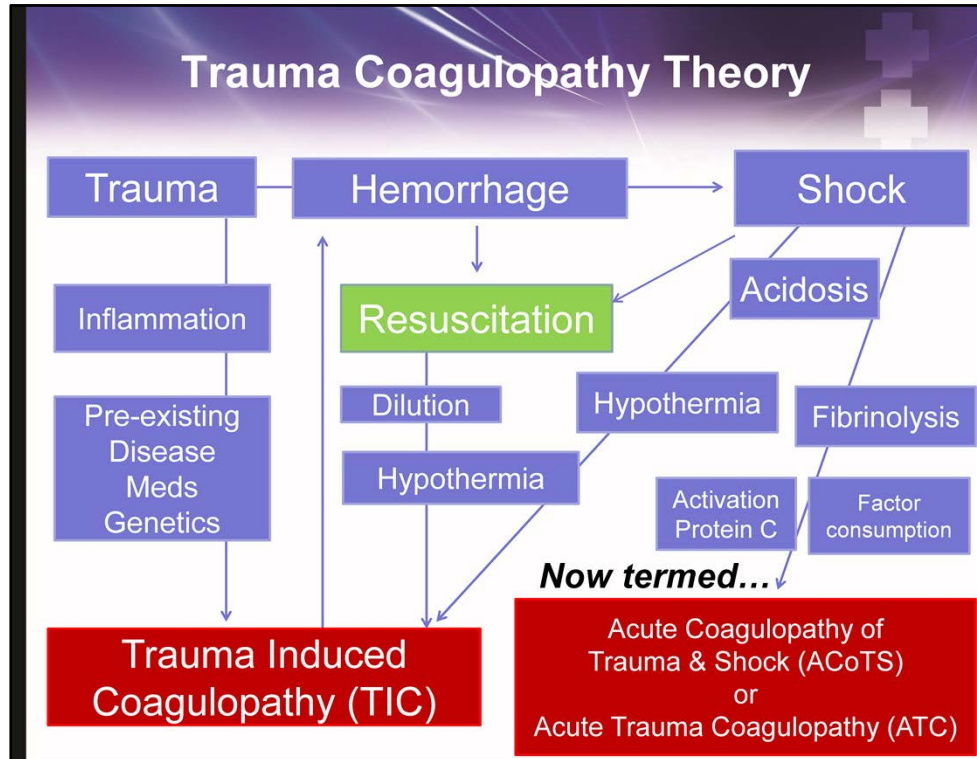
Stage III: return to OR within 12 – 48 hours for definitive surgery



Until 2003, it was thought that trauma patients became coagulopathic during the resuscitation process.

Brohi (2003) showed for the first time that coagulopathy was already present in up to 28% of trauma on arrival to the ED.

This group of patients are more likely to require massive transfusion, develop multisystem organ failure and are 4 times more likely to die.



The human coagulation system can be rapidly overwhelmed by severe injury.

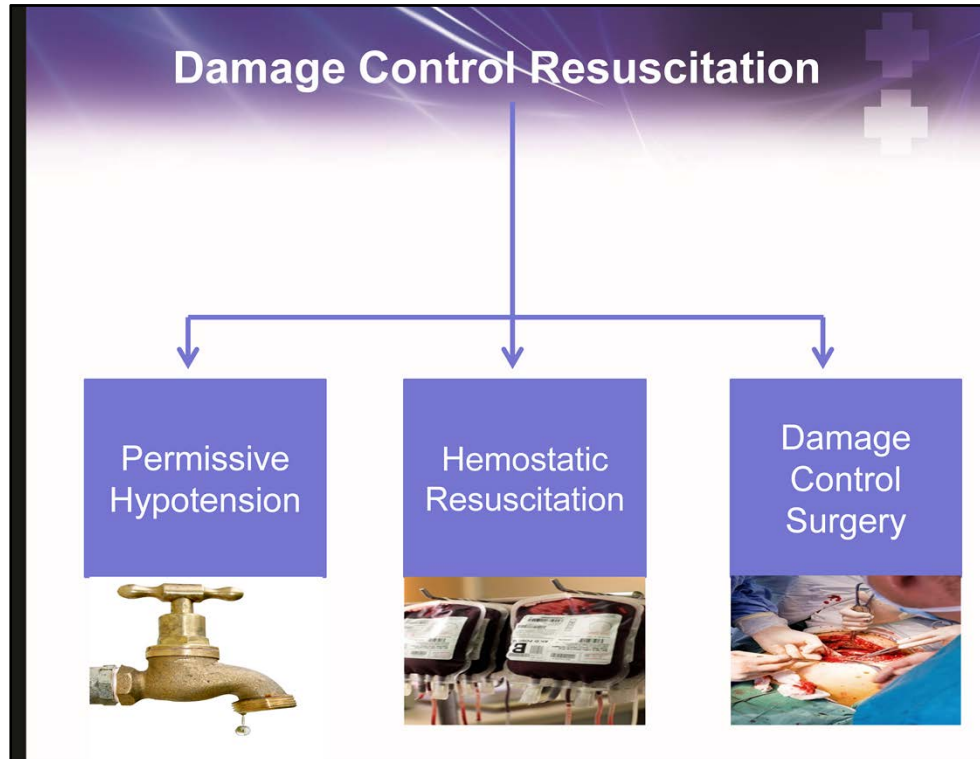
The cause or causes of **trauma-induced coagulopathy (TIC)** may be multi-factorial and still poorly understood.

It is clear that shock can lead to acidosis, hypothermia and inflammation, all of which can effect coagulation. None of this is new.

However, recent work has postulated a theory of tissue hypoperfusion leading to a primary coagulopathy termed **acute coagulopathy of trauma shock (ACoTS)** or **Acute Trauma Coagulopathy**.

Direct loss and consumption of coagulation factors, dilution, hypothermia, acidosis and fibrinolysis all diminish hemostasis.

Each of these mechanisms can occur independently, but all occur more frequently and severely with worsening degrees of injury, and their interaction can drive the coagulation system beyond functional limits.



Damage control resuscitation (DCR), a concept that has been popularized by the military, is being evaluated for its applicability in the civilian setting.

With DCR, systolic blood pressure is maintained around 90 mm Hg and isotonic crystalloid use is limited or nonexistent as blood products are transfused at a fixed ratio of one unit of fresh frozen plasma (FFP) for every unit of packed red blood cells.

DCR differs from current resuscitation approaches by achieving correction of coagulopathy through early hemostatic resuscitation, via set transfusion protocols based on physiologic variables in severely injured trauma patients.

The concept centers on the fact that coagulopathy can present early after injury and earlier diagnosis and interventions to correct it in the most severely injured patients.

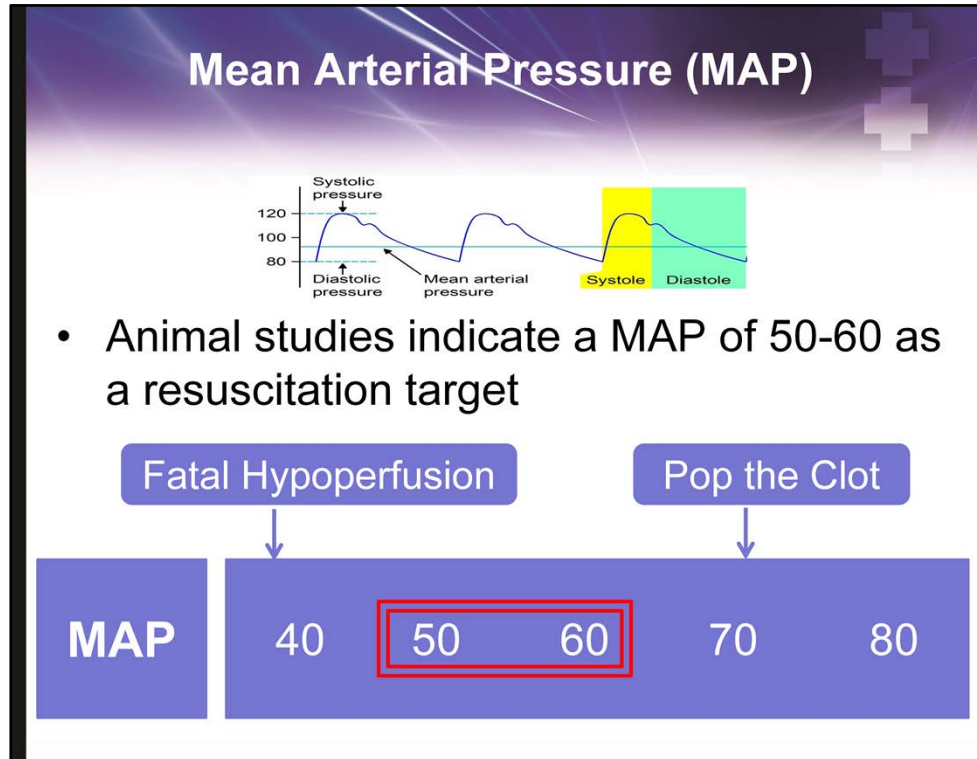
Permissive Hypotension

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



Permissive hypotension is allowing the SBP or MAP to remain at a minimum level that still allows for perfusion for up to 2 hours to prevent popping the clot.

Most often, the MAP is used to titrate to in the hospital setting.



The mean arterial pressure represents the average arterial pressure throughout the cardiac cycle, and is the force that drives blood through the vasculature.

MAP can be described in various ways but all mean the same thing:

$$\text{MAP} = 2/3 \text{ diastolic pressure} + 1/3 \text{ systolic pressure}$$

$$\text{MAP} = 2 \times \text{diastolic pressure} + \text{systolic pressure} / 3$$

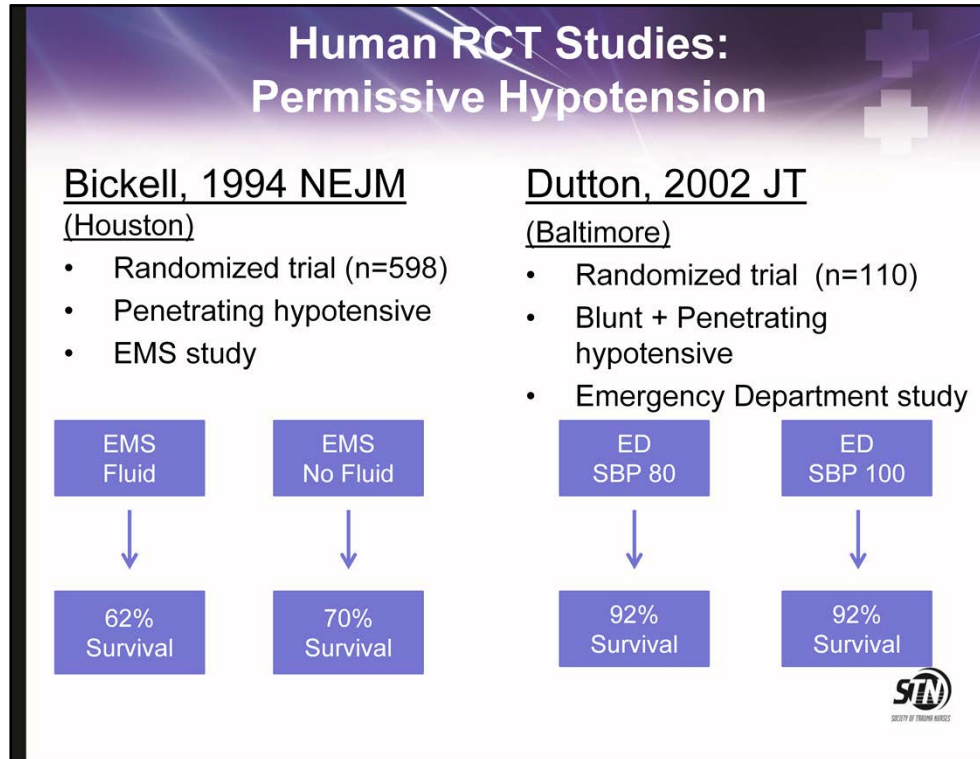
$$\text{MAP} = \text{diastolic pressure} + (1/3 \times \text{pulse pressure})$$

Animal data show that survival from uncontrolled hemorrhagic shock is enhanced by fluid resuscitation **limited to that required** to maintain MAP at 50-60 mmHg (70-80 % of normal).

Animal models precisely delineate the boundaries of fatal hypoperfusion (about 2 hours at a MAP of 40 mmHg) and fatal rebleeding (any MAP greater than 80 percent of normal)

Published studies almost universally demonstrate reduced total blood loss and improved survival when the hemorrhaging animal is maintained within these limits.

Fatal hypoperfusion



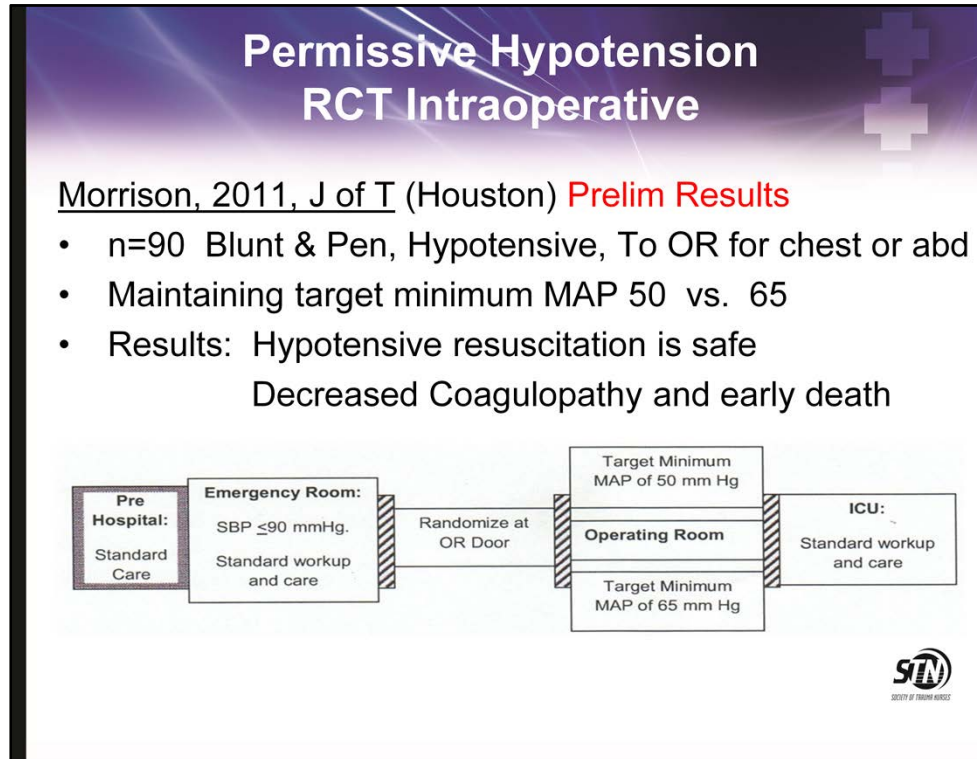
RCT = randomized control

The 2 studies are not equivalent:

They were performed a decade apart. You must take into consideration that improvement in care over time (especially damage control techniques) make it difficult to compare.

What Dutton's study shows is that keeping the ED SBP at 80 did no harm.

This further promotes the concept of permissive hypotension, in which further research is ongoing.



The most recent RCT:

Hypotensive Resuscitation Strategy Reduces Transfusion Requirements and Severe Postoperative Coagulopathy in Trauma Patients With Hemorrhagic Shock: Preliminary Results of a Randomized Controlled Trial

C. Anne Morrison, MD, MPH, Matthew M. Carrick, MD, Michael A. Norman, MD, Bradford G. Scott, MD, Francis J. Welsh, MD, Peter Tsai, MD, Kathleen R. Liscum, MD, Matthew J. Wall, Jr., MD, and Kenneth L. Mattox, MD, Journal of Trauma 2011: 70, 652-663

This study is the first randomized, prospective study of **intraoperative hypotensive resuscitation** for trauma in human subjects. The aim of this study is to assess patient outcomes after accrual of 90 patients to establish the safety of a hypotensive resuscitation strategy including its effects on intraoperative fluid administration, bleeding, postoperative complications, and mortality within the trauma population.

Patients undergoing laparotomy or thoracotomy for blunt and penetrating trauma who had at least one in-hospital documented systolic blood pressure (SBP) 90 mm Hg were randomized to one of the two treatment groups. Randomization occurred on arrival to the operating room (OR), and all patients were assigned to either an experimental group whose target minimum mean arterial pressure (MAP) for resuscitation was 50 mm Hg (LMAP) or to a control group whose target minimum MAP was 65 mm Hg (HMAP). Methods by which the target blood pressure goals were met were left to the discretion of the treating anesthesiologist. It should be noted that these target MAPs represent the minimum blood pressures at which further specific resuscitative interventions (e.g., fluids,

transfusions, or vasopressors) were administered. It was not our intent to ensure that patients remained at their minimum target MAP for the duration of the case; if patients were able to spontaneously maintain a MAP above their assigned target, the blood pressure was never intentionally lowered to reach this minimum target MAP.

BP Measurements			
Systolic	Diastolic	Pulse Pressure	MAP
120	80	40	93
115	75	40	88
110	75	35	87
105	70	35	82
100	70	30	80
95	65	30	75
90	60	30	70
85	55	30	65
80	50	30	60
75	50	25	58
70	45	25	53
65	40	25	48
60	35	25	43

**Normal
MAP
70-100**

**Coming
Soon?
New
Target
MAP
50-70**

This chart is helpful to show the relationship of Systolic, Diastolic to pulse pressure and MAP.

It appears that there is a survival benefit for keeping the MAP somewhere between 50-70.

The lowest MAP that is still safe in trauma is not yet known.

Some authors speculate it could be as low as 50-70.

What is known, is that keeping the SBP below 100 is optimal in all non head injured patients with hemorrhage.



Both Geriatric and TBI patients are known to be at greater risk from periods of hypotension, blood loss, and long resuscitations.

In the only study to include both hemorrhage and TBI is a [canine study](#) that examined resuscitation in a combined hemorrhage/TBI model, the best survival was achieved by early use of deliberative hypotension.

Novak 1999 J of Trauma 47:834-844.

No one knows the answer to this, yet.

Application of permissive hypotension is not recommended in these populations ---at this time.

Hemostatic Resuscitation

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



Hemostatic resuscitation has become a popular form of transfusion therapy.

The concept of giving plasma and platelets early along with red cells in an attempt to closely approximate whole blood.


Massive Transfusion Protocols help achieve this.

Blood Loss

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

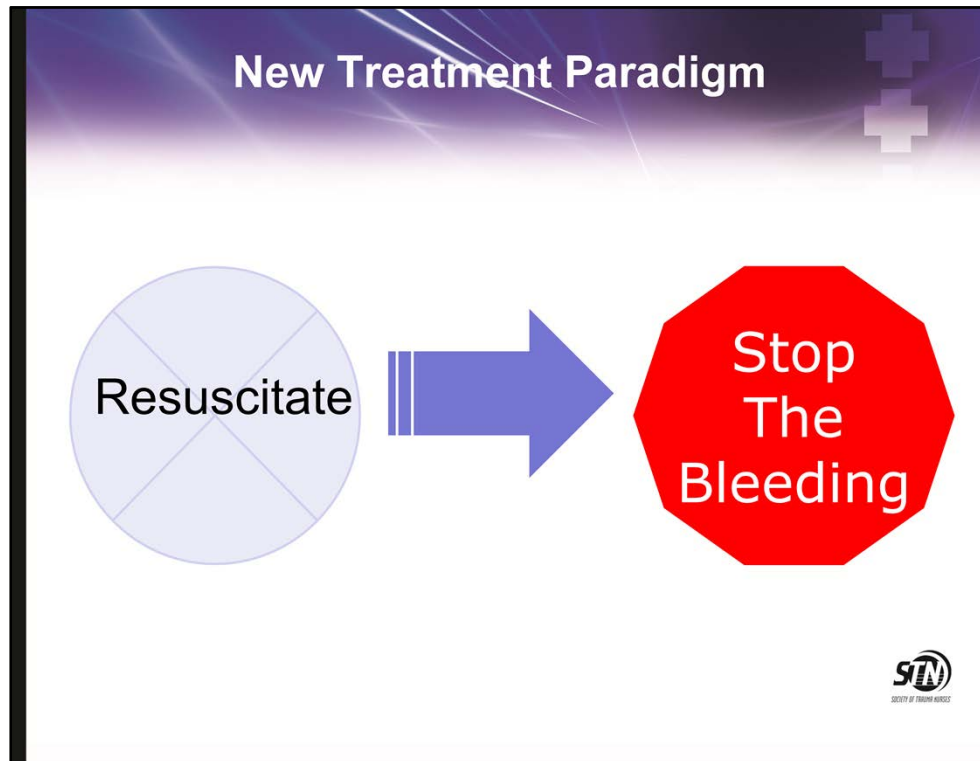
Current consensus:
Damage Control Resuscitation

- Permissive Hypotension
- Hemostatic Resuscitation
- Damage Control Surgery

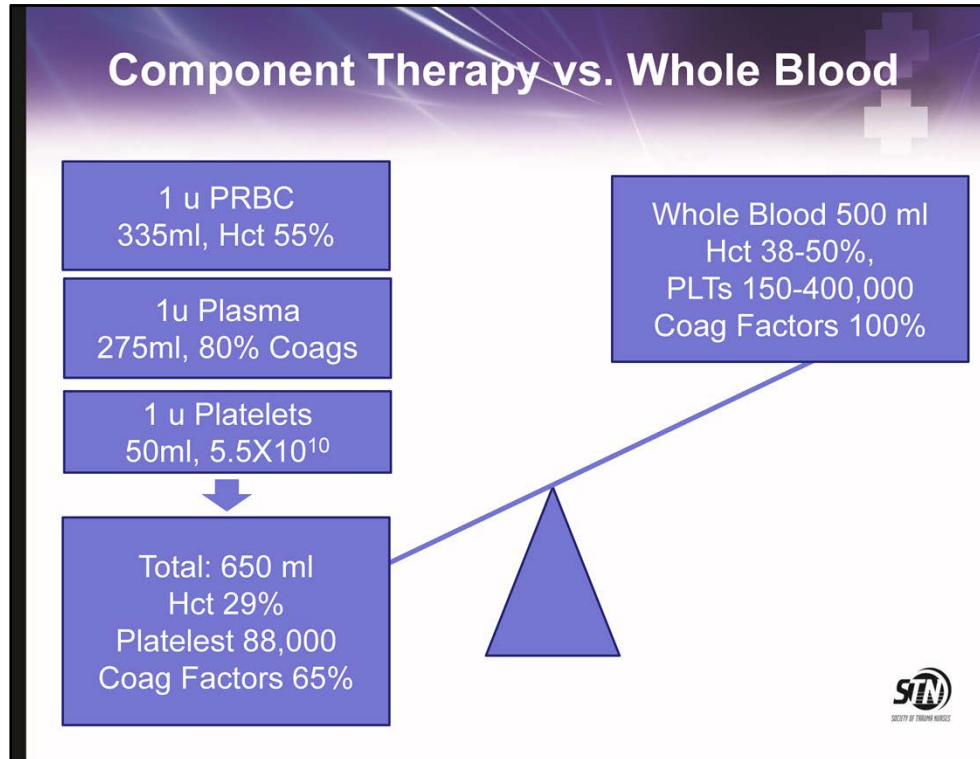


The old system of giving 2 Liters of fluid followed by more fluid and finally blood but no plasma or platelets has shown no increase in survival but rather has probably contributed to early death from hemorrhage.

There is a huge shift in thinking across the nation with a move toward Damage Control Resuscitation: permissive hypotension, hemostatic resuscitation and damage control surgery.



Too much emphasis in the past has been on resuscitating with fluids and not enough on stopping the bleeding.



This diagram is to show the way in which a donated unit of whole blood changes through the process of fractionation and then theoretical reconstitution at the point of care.

The resulting 'whole blood' fluid provides minimally acceptable levels of red blood cells (RBC), clotting factors and platelets, with scant margins to replace existing deficits.

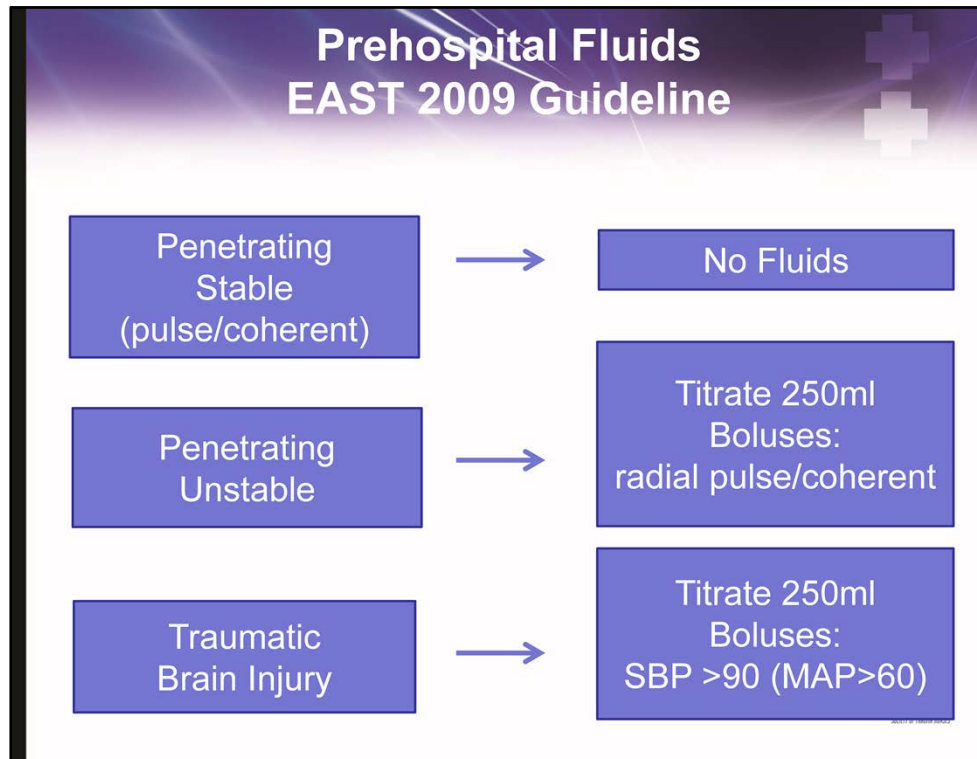
Banked blood products are relatively dilute and clotting factor poor.

This has promoted the movement toward of 1:1:1 transfusions of PRBC:Plasma:Platelets in trauma.



Hemorrhage research is fast moving.

Keep abreast of all research and amend your protocols as the research emerges.



In patients with penetrating injuries and short transport times (less than 30 minutes), fluids should be withheld in the prehospital setting in patients who are alert or have a palpable radial pulse.

Fluids (in the form of small boluses, i.e., 250 mL) should be given to return the patient to a coherent mental status or palpable radial pulse.

In the setting of traumatic brain injury, however, fluids should be titrated to maintain systolic blood pressure greater than 90 mm Hg (or mean pressure greater than 60 mm Hg).

Hypertonic saline boluses of 250 mL seem equivalent in efficacy to 1,000 mL boluses of standard solutions (lactated Ringer's, 0.9% sodium chloride).


There is insufficient evidence to show that injured patients with short transport times benefit from prehospital blood transfusions.

Identifying The Patient At Risk In ED

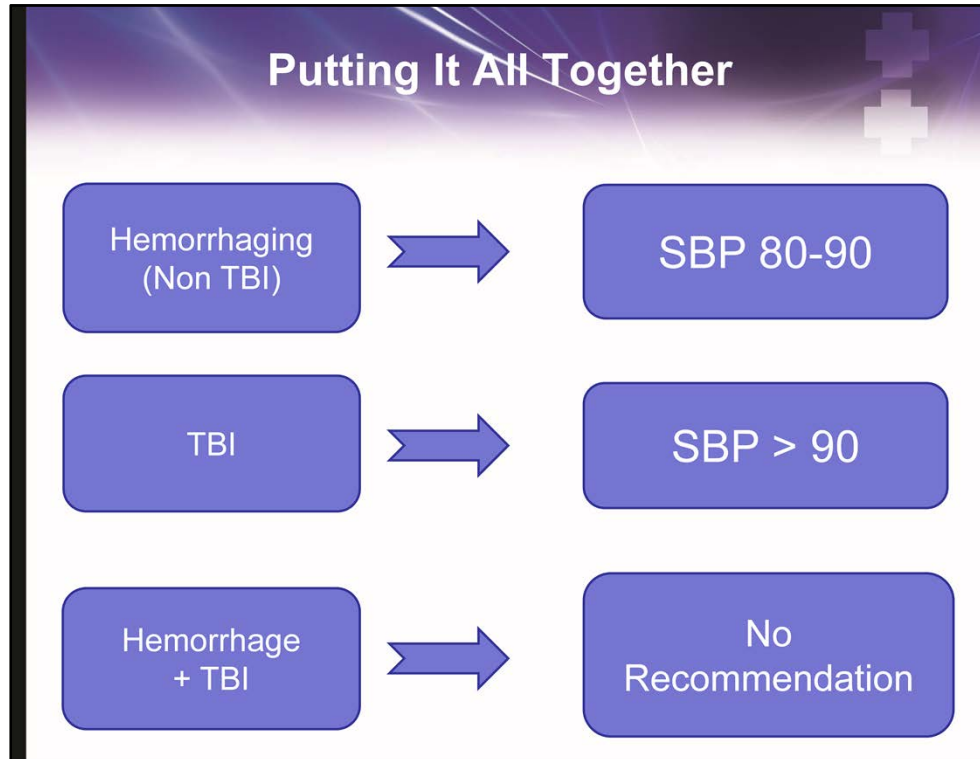
- High ISS
- SI > 0.9
- SBP < 90 mm Hg
- Acidosis Base Deficit ≥ 6
- Hypothermia T < 35C (95 F)
- INR ≥ 1.5
- Elevated Lactate

Required Labs
For Major
Activations:

- ABG
- BD
- Lactate
- INR



These parameters will help you identify the hemorrhaging patient who is at risk for Acute Coagulopathy of Trauma, who may need massive transfusion and ICU admission.



Current literature indicates the following situations.

The non TBI hemorrhaging trauma patient has a target SBP of 80-90.

TBI patients should target a SBP over 90.

There are no current recommendations for the hemorrhaging patient with concomitant TBI.

Research is ongoing in all areas.

Summary

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

