Trauma, Critical Care and Anesthesia

Bill Howie, DNP, CRNA, CCRN.
Care of Patients with Complex Needs: The Basics.
2015

Objectives

- Discuss the concept of mechanism of injury and how it helps guide management of care.
- Describe why it is important to have a high index of suspicion when considering the care of a trauma patient.
- Provide strategies to minimize or prevent primary injury.
- Discuss the concept of secondary injury, and how to minimize this type of injury.

Objectives

- Discuss appropriate lab values necessary to provide optimal patient care (follow trends).
- Formulate a dynamic plan of care for the critically ill/injured patient.
- List 5 components of the primary survey as they relate to assessment of the patient.
- Define shock.
- Delineate the six major causes or types of shock.

Objectives

- Define damage control surgery.
- Define and discuss damage control resuscitation
- Describe the three stages of damage control surgery.
- Name at least 5 basic rules of trauma.
- Discuss the definitive management a trauma patient's airway. Include a brief description of the necessary steps and how many people are required to safely accomplish them.

Introduction

- Trauma, whether caused by unintentional or intentional injury, is the leading cause of death in the USA from birth to 44 years of age (CDC 2006).
- 160,000 deaths/year, 340,000 permanently disabled, $107 billion/year in medical fees, hospitalization, and lost wages.
- Assaults/GSW's cost on average $40,000 per pt. (70% not insured) at the WFSTC.

TRAUMA DEFINED

- A structural alteration or physical imbalance brought about by acute exposure to mechanical, thermal, electrical or chemical energy. At times, the trauma can be caused by and lack of heat, food , water , or O2.
- In cases of blunt and penetrating trauma, a transfer of energy leads to the ultimate damage produced to the victim.
- The precipitating cause of the traumatic injury is typically referred to as the mechanism of injury.

Incidence/Demographics

- Each year the STC admits about 8000 pts.
- 72% are male, 28% are female.
- Statistics:
  - 42% MVC (about 34% did not use a seatbelt);
  - 5% MCC (90% did not wear a helmet);
  - 6% Pedestrian struck;
  - 17% Fall;
  - 8% GSW;
  - 8% Stabbed;
  - 84% Blunt Trauma;
  - 16% Penetrating Trauma

10 Leading Causes of Death, United States

2010, All Races, Both Sexes

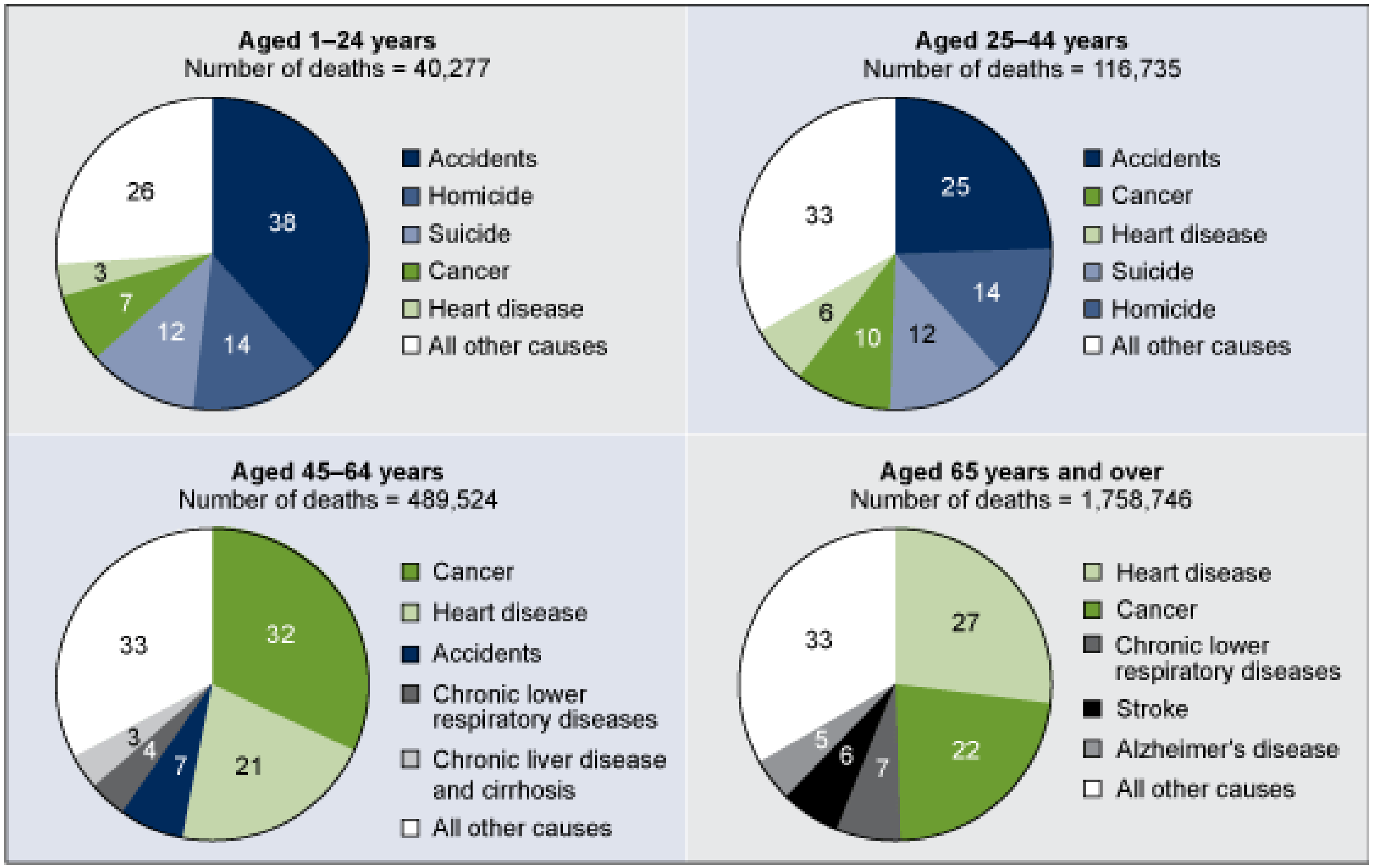
Rank	Age Groups											
Rank	<1	1-4	5-9	10-14	15-19	20-24	25-34	35-44	45-54	55-64	65+	All Ages
1	Congenital Anomalies 5,107	Unintentional Injury 1,394	Unintentional Injury 758	Unintentional Injury 885	Unintentional Injury 4,537	Unintentional Injury 7,804	Unintentional Injury 14,573	Unintentional Injury 14,792	Malignant Neoplasms 50,211	Malignant Neoplasms 109,501	Heart Disease 477,338	Heart Disease 597,689
2	Short Gestation 4,148	Congenital Anomalies 507	Malignant Neoplasms 439	Malignant Neoplasms 477	Homicide 1,832	Suicide 2,941	Suicide 5,735	Malignant Neoplasms 11,809	Heart Disease 36,729	Heart Disease 68,077	Malignant Neoplasms 396,670	Malignant Neoplasms 574,743
3	SIDS 2,063	Homicide 385	Congenital Anomalies 163	Suicide 267	Suicide 1,659	Homicide 2,846	Homicide 4,258	Heart Disease 10,594	Unintentional Injury 19,667	Chronic Low. Respiratory Disease 14,242	Chronic Low. Respiratory Disease 118,031	Chronic Low. Respiratory Disease 138,080
4	Maternal Pregnancy Comp. 1,561	Malignant Neoplasms 346	Homicide 111	Homicide 150	Malignant Neoplasms 601	Malignant Neoplasms 1,003	Malignant Neoplasms 3,619	Suicide 6,571	Suicide 8,799	Unintentional Injury 14,023	Cerebro-vascular 109,990	Cerebro-vascular 129,476
5	Unintentional Injury 1,110	Heart Disease 159	Heart Disease 68	Congenital Anomalies 135	Heart Disease 342	Heart Disease 686	Heart Disease 3,222	Homicide 2,473	Liver Disease 8,651	Diabetes Mellitus 11,677	Alzheimer's Disease 82,616	Unintentional Injury 120,859
6	Placenta Cord Membranes 1,030	Influenza & Pneumonia 91	Chronic Low. Respiratory Disease 60	Heart Disease 117	Congenital Anomalies 202	Congenital Anomalies 210	HIV 741	Liver Disease 2,423	Cerebro-vascular 5,910	Cerebro-vascular 10,693	Diabetes Mellitus 49,191	Alzheimer's Disease 83,494
7	Bacterial Sepsis 583	Septicemia 62	Cerebro-vascular 47	Chronic Low. Respiratory Disease 73	Cerebro-vascular 86	Influenza & Pneumonia 133	Diabetes Mellitus 606	Cerebro-vascular 1,904	Diabetes Mellitus 5,610	Liver Disease 9,764	Influenza & Pneumonia 42,846	Diabetes Mellitus 69,071
8	Respiratory Distress 514	Benign Neoplasms 59	Benign Neoplasms 37	Benign Neoplasms 45	Chronic Low. Respiratory Disease 60	HIV 125	Cerebro-vascular 517	HIV 1,898	Chronic Low. Respiratory Disease 4,452	Suicide 6,384	Nephritis 41,994	Nephritis 50,476
9	Circulatory System Disease 507	Perinatal Period 52	Influenza & Pneumonia 37	Cerebro-vascular 43	Influenza & Pneumonia 48	Complicated Pregnancy 121	Liver Disease 487	Diabetes Mellitus 1,789	HIV 3,123	Nephritis 5,082	Unintentional Injury 41,300	Influenza & Pneumonia 50,097
10	Necrotizing Enterocolitis 472	Chronic Low. Respiratory Disease 51	Septicemia 32	Septicemia 35	Benign Neoplasms 46	Diabetes Mellitus 120	Congenital Anomalies 397	Influenza & Pneumonia 773	Viral Hepatitis 2,376	Septicemia 4,604	Septicemia 26,310	Suicide 38,364

WISQARS™ Produced By: Office of Statistics and Programming, National Center for Injury Prevention and Control, Centers for Disease Control and Prevention

Data Source: National Center for Health Statistics (NCHS), National Vital Statistics System

Five leading Causes of Death

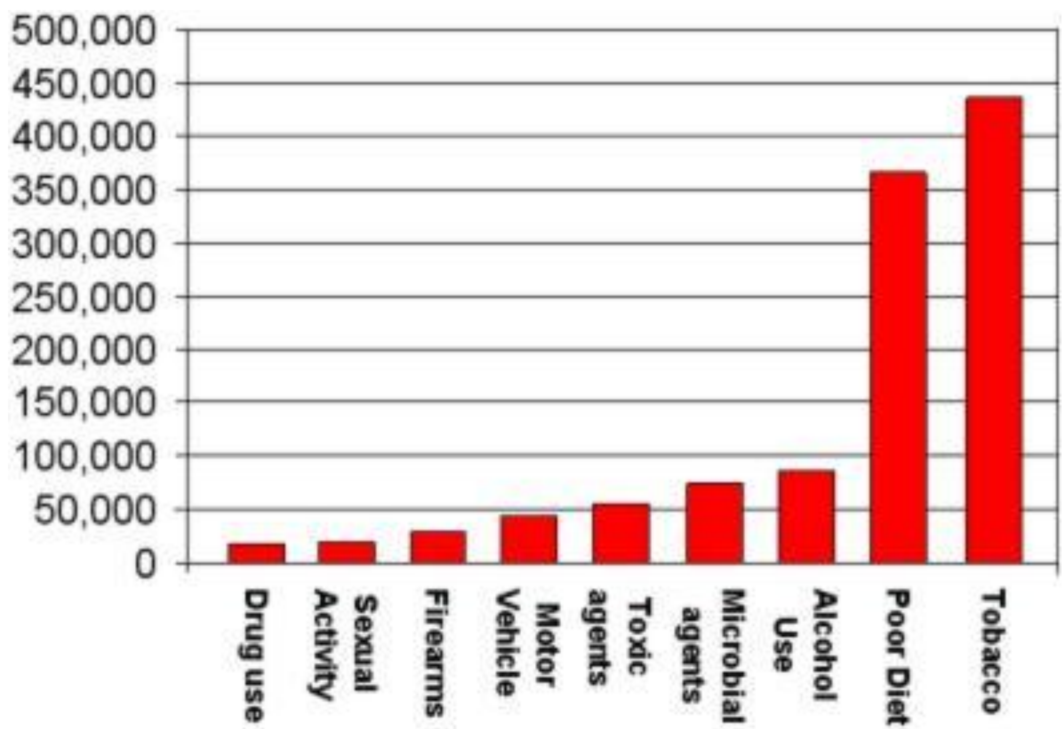
Figure 5. Percent distribution of five leading causes of death, by age group: United States, preliminary 2009



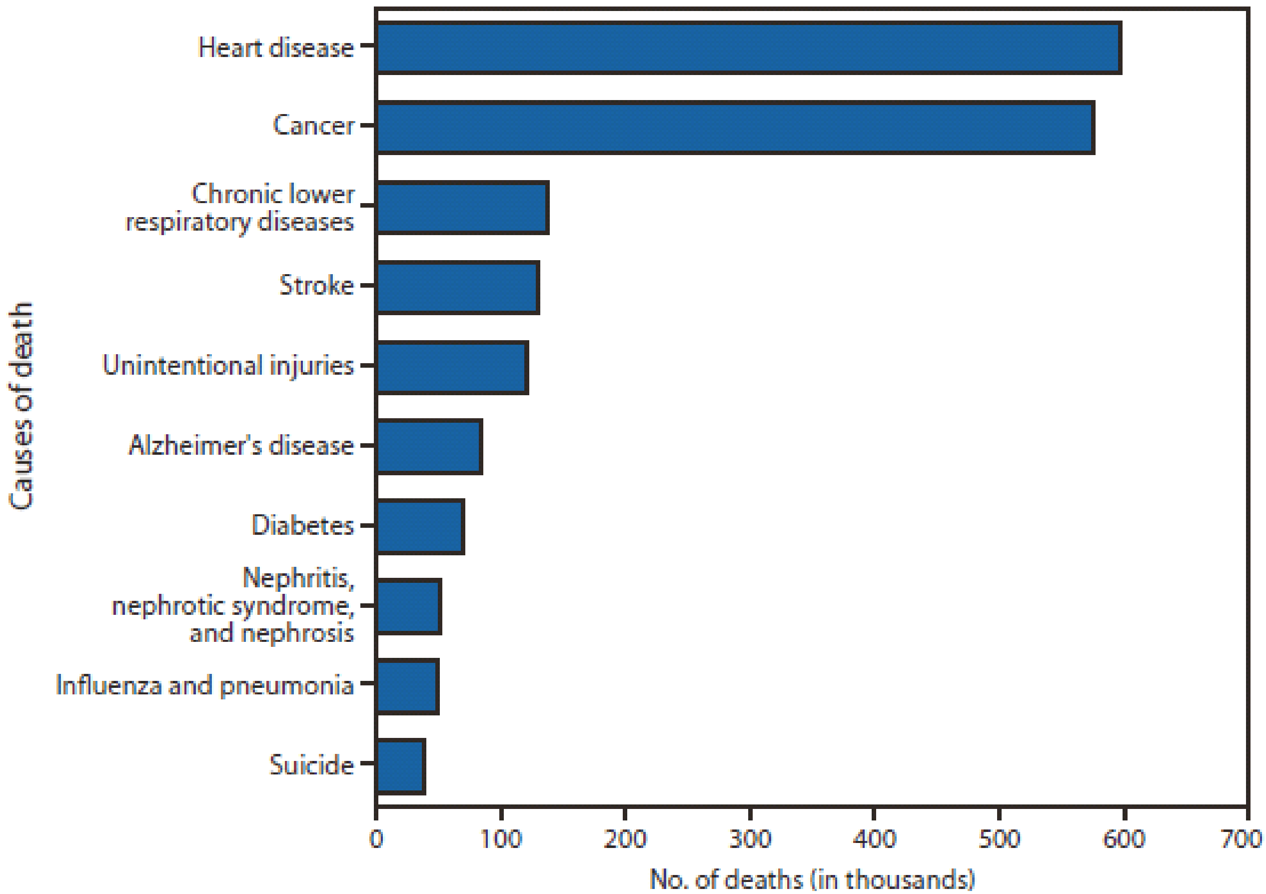
SOURCE: CDC/NCHS, National Vital Statistics System, Mortality.

Causes of Preventable Deaths in U.S.

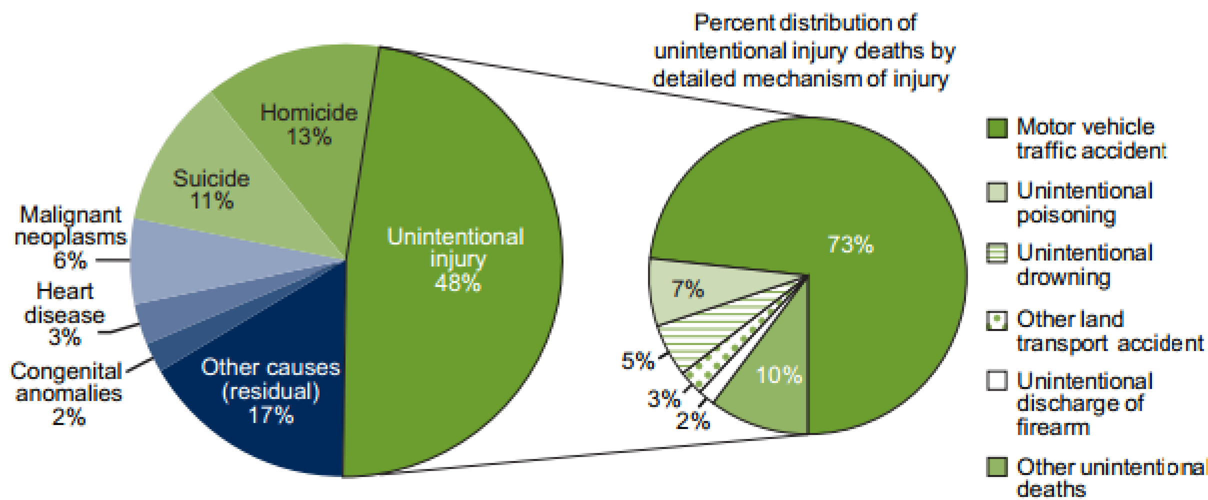
Ali H. Mokdad,.,et.al.JAMA, March 10, 2004—Vol 291, No. 10. p 1238-1245.



Causes of Death in the USA 2013

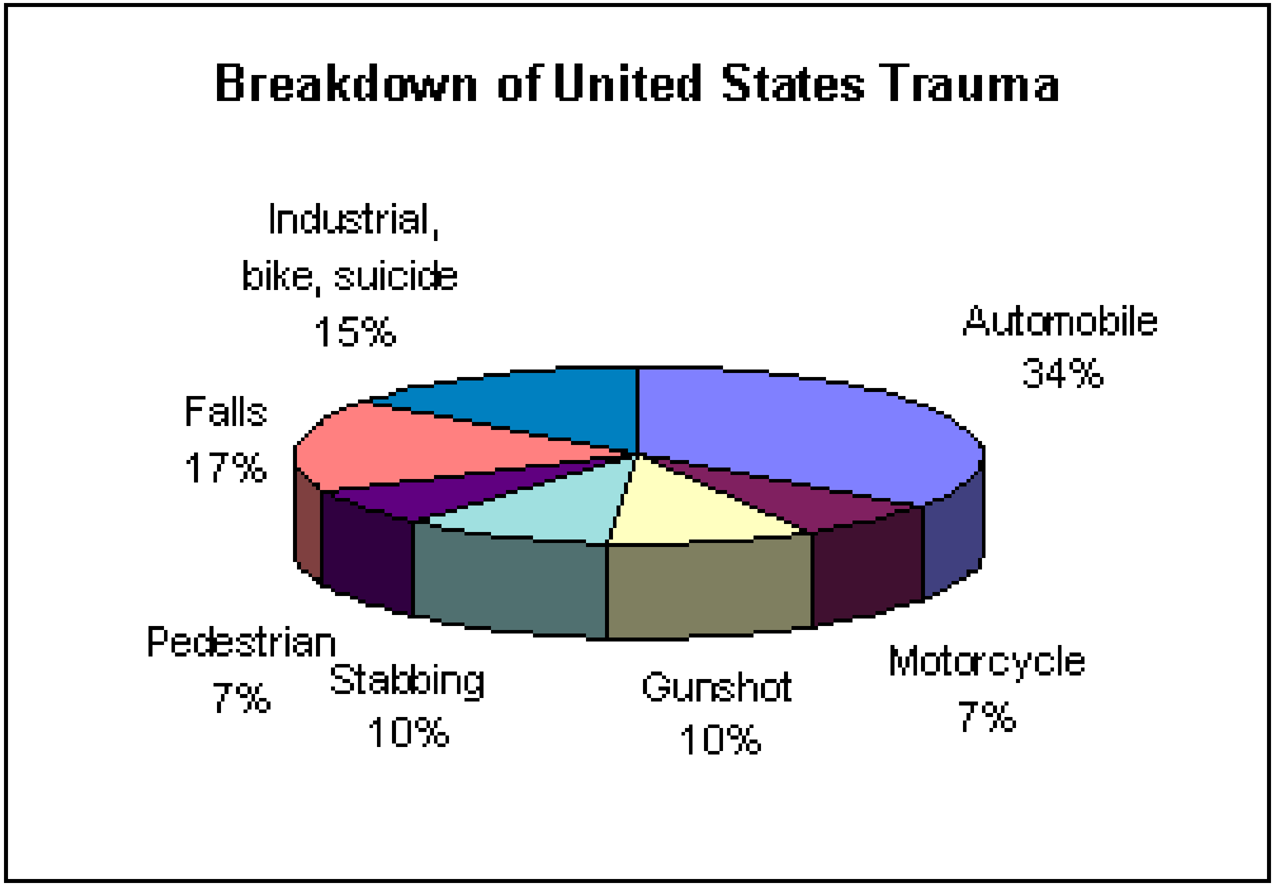


National Vital Statistics 2010

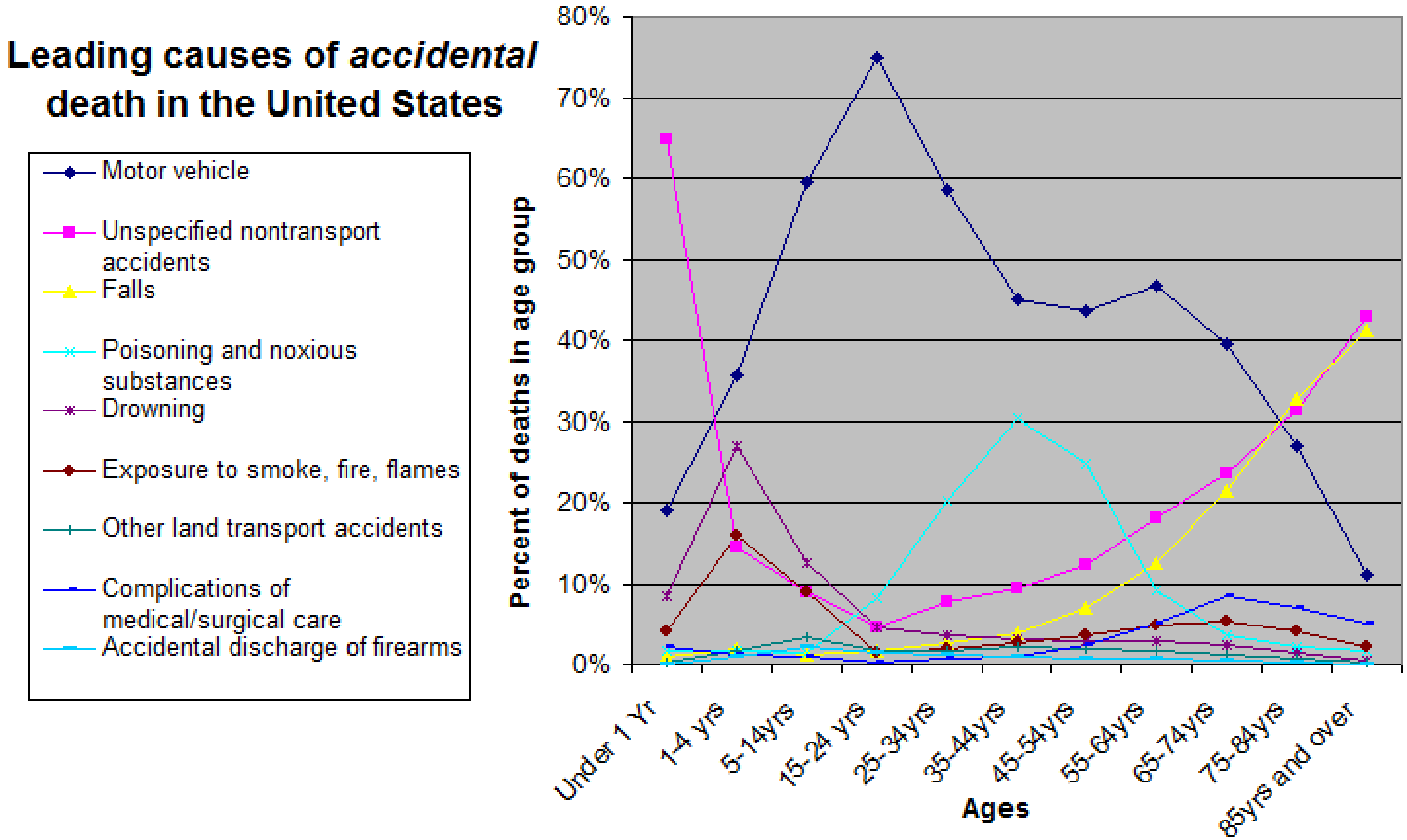


Source: National Vital Statistics System, 2010

Types of Trauma



Causes of Death by Mechanism of Injury in the USA (National Vital Statistics)



Mechanism of Injury

- What is a mechanism of injury?
- Why is it important to consider how a patient is injured?
- What are the most common mechanisms of injury seen at the STC and in the US?
- Does the mechanism of injury have an impact on how the patient is triaged and sent to a given facility for further work-up?

Mechanism of Injury

Table 1. INDICATIONS OF MAJOR BLUNT TRAUMA AND HIGH-IMPACT ΔV

Two or more long bone fractures
Unstable pelvis
Flail chest
Sternal, clavicular, scapular, or upper rib fractures
Falls of 4.5 m or more (adult) or 3.6 m or more (child)
Delta V of greater than 32 km/h without restraints, 40 km/h with restraints
Rearward displacement of vehicle by 6 m
Rearward displacement of front axle
Engine intrusion into passenger compartment
Frame intrusion into passenger compartment
37.5 cm on patient side of car
50 cm on opposite side of car
Ejection of a passenger
Rollover
Death of a passenger
Pedestrian struck at 32 km/h or more
"Spiderweb" in windshield
Prolonged extrication

Adapted from Stene JK, Grande CM: Trauma Anesthesia. Baltimore, Williams & Wilkins, 1991, p 51; with permission.

Health Worker Safety?

Table 2

Patient and Provider differences in the use of Standard Precautions

(Observational period 1)

	Wearing Gloves Induction	Wearing Gloves Emergence	Using Eye Protection Induction	Using Eye Protection Emergence	Gloves to Tape ETT	Gloves to Tape IV
Level of Training	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
sRNA (junior)	47 (96)	44 (98)	47 (6)	44 (7)	11 (82)	5 (40)
CA2 (junior)	37 (97)	35 (100)	37 (30)	35 (29)	9 (100)	0 (0)
CA3 (junior)	29 (90)	26 (100)	29 (41)	26 (35)	†13 (38)	15 (60)
Fellow (senior)	*47 (51)	*44 (70)	*47 (64)	*44 (61)	†21 (24)	10 (10)
CRNA (senior)	*33(42)	*32 (50)	*32 (50)	*32 (50)	†19 (16)	12 (17)
Race						
Caucasian	99 (78)	91 (88)	98 (37)	91 (38)	39 (41)	24 (33)
Black	32 (53)	29 (66)	32 (44)	29 (34)	12 (25)	8 (25)
Hispanic	33 (94)	33 (94)	33 (36)	33 (30)	11 (73)	5 (60)
Asian	7 (100)	7 (100)	7 (29)	7 (43)	2 (0)	2 (0)
Other	22 (59)	21 (67)	22 (36)	21 (33)	9 (44)	3 (33)
Age Group						
Infant	34 (76)	33 (82)	34 (44)	33 (39)	14 (43)	7 (29)
Child	140 (77)	131 (85)	139 (35)	131 (34)	50 (42)	32 (31)
Adolescent	19 (58)	17 (71)	19 (47)	17 (47)	9 (44)	3 (67)
Gender						
Male	131 (76)	122 (83)	130 (33)	122 (32)	47 (47)	23 (35)
Female	62 (74)	59 (85)	62 (47)	59 (44)	26 (35)	19 (32)

* $p < 0.05$ CRNA, Fellow vs sRNA, CA2, CA3
† $P < 0.05$ sRNA, CA2 vs CA3, Fellow, CRNA

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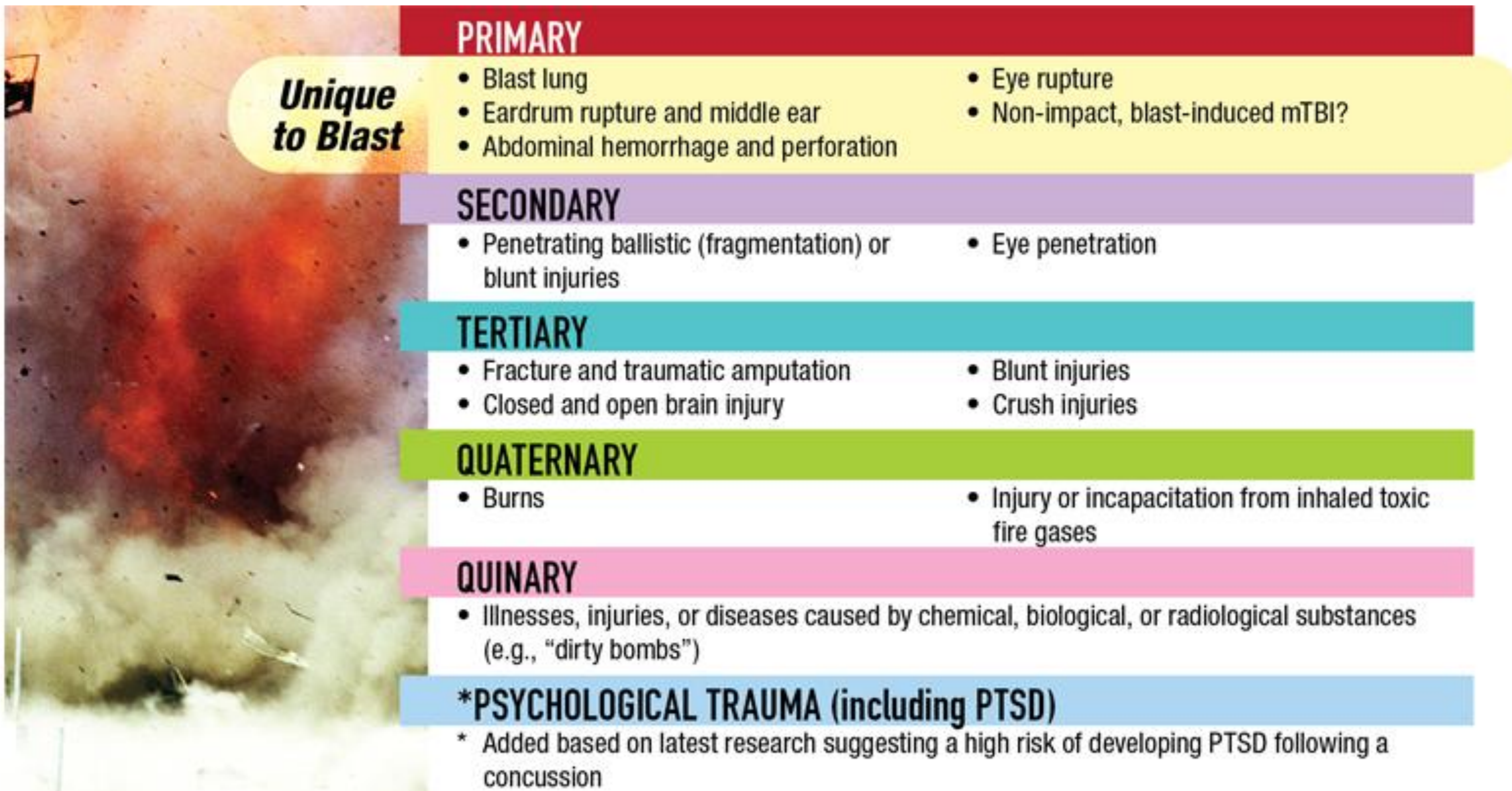
A new concern of Civilian Trauma in the US?



Basic Principles

- ABC (**D**isability **E**xposure) (Reassess) This process is dynamic, not static (the primary survey).
- Primary Survey/ Secondary Survey.
- Work-up =standard tests (History, X-ray exams, Lab work, Physical exam…etc.).
- Index of suspicion must be high, and based on presentation and mechanism. Why?

Airway Management

- All patients should receive:
  - 100% 02 3 to 5 minutes with an effective mask fit.
  - Suction immediately available.
  - Cricoid pressure (for suspected full stomach).
  - In line stabilization (for suspected C-spine injury).
  - All appropriate airway equipment and pharmacologic agents available.
  - End tidal C02 confirmation.
  - How many people are required to safely manage the airway?


3
2
1
4

Airway

- Commitment to a “definitive” airway
- Your first try is usually your best try
- RSI is usually the rule
- If it isn’t within 3 feet of you, it might as well be on the moon
- If we can bag, we’re going to go, if we can’t bag, we’re going to go (push the relaxant)
- The surgeon is your friend

Definitive Airway Cpmtrol

Rapid Sequence Induction, Inline w/ cricoid

Check etCO2, cricoid pressure

Rapid DL no neck extension

Airway Management

- The skillful and appropriate placement of an endotracheal tube is the best way to:
  - Ensure delivery of adequate oxygenation.
  - Ensure optimal ventilation.
  - Protect the lungs from aspiration.
  - Facilitate optimal resuscitation of the patient.

Direct laryngoscopy with orotracheal intubation is the principle method used in acute trauma patients because of speed and technical advantages. (Wilson, Grande, & Hoyt Trauma 2007).

Emergency Airway Management

- In the hands of experienced anesthesia, rapid sequence intubation followed by direct laryngoscopy is a remarkably effective approach to emergency airway management. An algorithm designed around this approach can achieve very high levels of success.
- During the first hour after admission, 6088 patients required intubation, of whom 21 (0.3%) received a surgical airway. During the first 24 h, 10 more patients, for a total of 31, received a surgical airway, during approximately 32,000 attempts (0.1%).
  - Stephens CT, Kahntroff S, Dutton RP. (2009, 866-72).

Airway Management

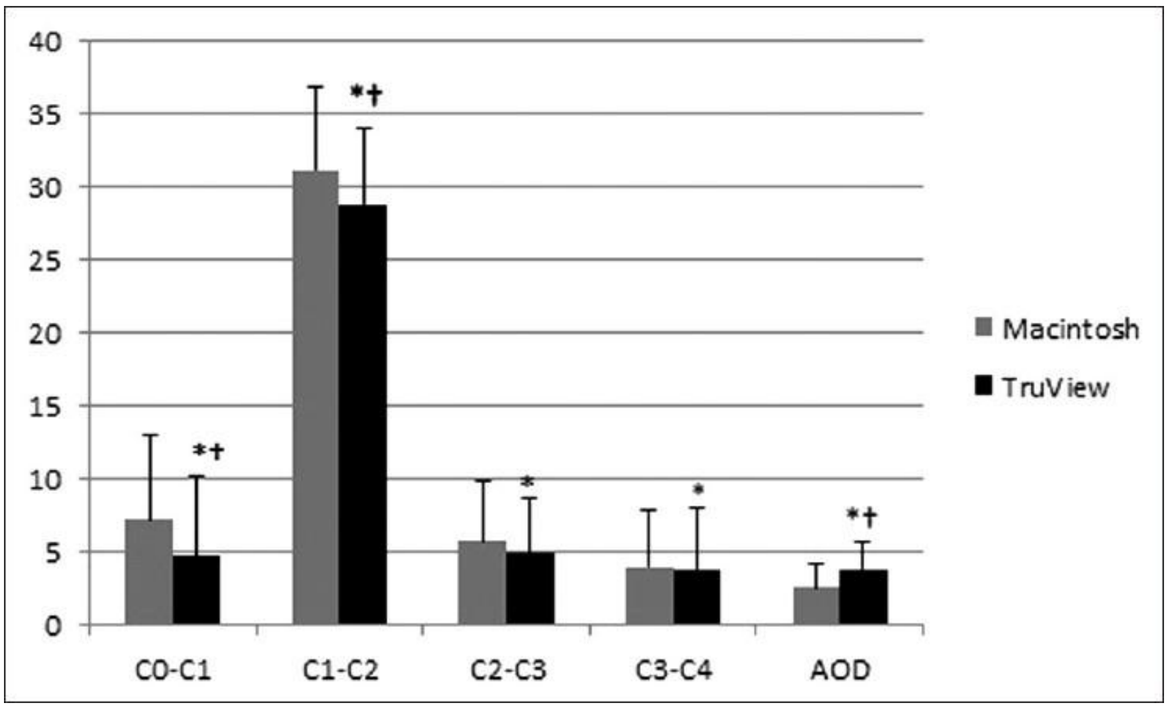
- Rapid sequence induction(RSI) with in line cervical spine stabilization is considered the gold standard in airway management in the acutely injured trauma patient.
- Assisted ventilation (modified RSI) is frequently justified because trauma patients are often hypoxic and hypercapnic.
- Following intubation, the ETT position must be confirmed with measurement of ETc02.

Airway Management

- Inability to ventilate or intubate constitutes an emergency condition. Airway adjuncts such as the Glidescope and bougie are useful backup strategies to the standard direct laryngoscopy.
- Adjuncts such as the LMA and the esophageal combitube are useful backup options to temporize until a definitive airway can be obtained.
- A surgical airway (cricothyrotomy or tracheostomy must be considered when endotracheal intubation is deemed “impossible”).

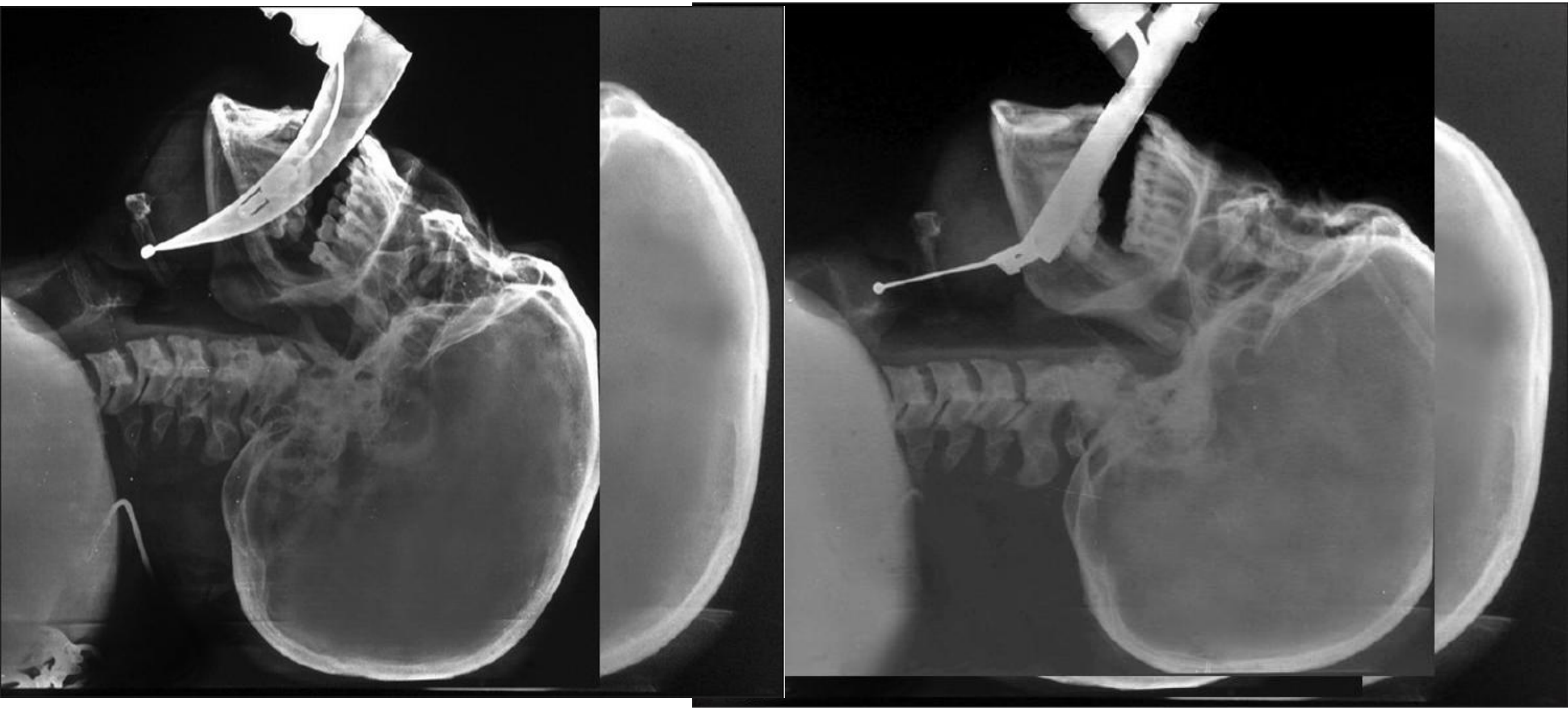
The C spine status is often unknown if immediate airway control is needed.

- Treat all trauma airways as an unstable c-spine and full stomach until proven otherwise



C spine movement with laryngoscopy

MAC versus Tru-view

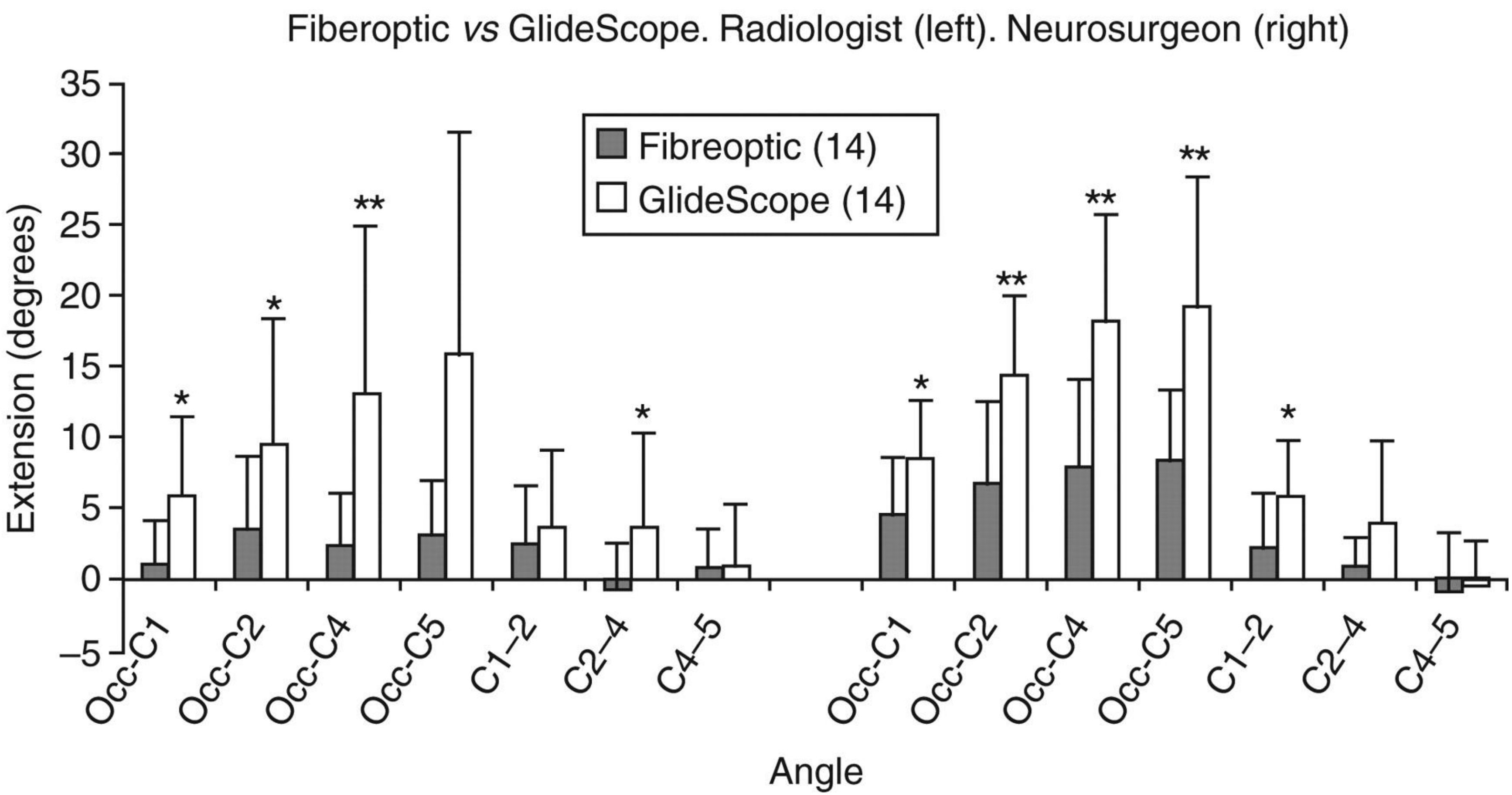


C spine Movement with Airway Manipulation

- Various studies have noted less movement with the GlideScope and fiberoptic airway management techniques
- Mean movement approx 11° with the Glidescope, and 14° with a standard Macintosh intubation. (a max of 19° vs 29°)
- The light wand or trach light produces even less movement
- Turkstra et al. (2005) Anesth Analg: 101(4) pp 910-915.

Kill, et al. (2013) Videolaryngoscopy with glidescope reduces C spine movement. J Emerg Med: 44(4) pp 750-756.

Wong et al. (2009) Br. J Anesth: C spine movement during flexible bronchoscopy compared with lo-pro glidescope: 102(3) pp 424-430.



A better use for the MAC blade?


TRU
For Emergency
ONLY
TRU INTUBATION BOX
DO NOT USE AFTER
34
BD 30ml Syringe
Luer-Lok Tip
BD 10ml Syringe
NELLCOR
7.5
8.0

Basic Pathophysiology

- Traumatic Shock is a state triggered by failure to deliver adequate O_2 to the cells in the body.
- Clinical indicators of shock include: ↓LOC , ↓BP , ↓UO, lactate and ↑CRT
- ↑HR, ↑acidosis, ↑O_2 demand coupled with decreased 02 delivery produces ↑lactate
- Cyanosis, pallor, and low or absent pulse ox.

Definition of Shock

A failure of adequate oxygen delivery or utilization at the cellular level, perpetuated by cellular and humoral responses.

Prolonged shock results in a cumulative “oxygen debt” and in disruption of end-organ integrity and homeostasis. (Dutton, 2006)

Types/Causes of Shock

- Cardiogenic: Pump failure
- Hemorrhagic: Decreased fluid
- Anemic: Decreased O2 carrying
- Neurogenic: Loss of vascular tone
- Toxic/Endotoxic/Anaphylaxis/Sepsis
- Traumatic Shock: Acute accident/injury. Can be a combination of factors

Causes of Cellular Ischemia

- Decreased O2 uptake by the lungs
- Decreased O2 carrying capacity
- Decreased vascular volume and tone
- Decreased cardiac function (Causes can include: tension pneumo, tamponade, ischemia or contusion, decreased inotropic state, drugs, CNS injury and failure of cellular metabolism(sepsis).

Clinical Characteristics of Shock

- Diminished blood pressure
- Decreased mentation
- Cyanosis, pallor, diaphoresis
- Hypothermia/Hyperthermia
- Decreased urine output
- Absent pulse oximetry signal

Novel Indices to Detect Shock

- **Shock Index**
  - HR / SBP (>0.9)
  - Better indicator than hypotension alone
- “**ABC**”
  - 4 parameters: **penetrating mechanism, positive FAST, SBP < 90, HR > 120**
  - 2 or more: 75% sensitive 86% specific for predicting massive transfusion

Cocci MN, et al. Emerg Med Clin North Am 2007; 25: 623-642.

Nunez TC, et al. J Trauma 2009; 66: 346-352.

Prognostic value of Peripheral Perfusion=capillary refill time (CRT)

- Using a Sequential Organ Failure Assessment (SOFA) score and serum lactate levels 50 ICU patients were studied. It was concluded that:
  - An unfavorable outcome (worsening SOFA score) was 7 times more likely in a patient with abnormal peripheral perfusion (cool extremities)than in one with normal peripheral perfusion (CRT)
  - A patient with abnormal CRT was 5 times more likely to have hyperlactatemia than a patient with normal CRT

SOFA Score

SOFA score	0	1	2	3	4
Respirationa PaO_2/FIO_2 (mm Hg) SaO_2/FIO_2	>400	<400 221–301	<300 142–220	<200 67–141	<100 <67
Coagulation Platelets $10^3/mm^3$	>150	<150	<100	<50	<20
Liver Bilirubin (mg/dL)	<1.2	1.2–1.9	2.0–5.9	6.0–11.9	>12.0
Cardiovascular[b] Hypotension	No hypotension	MAP <70	Dopamine </=5 or dobutamine (any)	Dopamine >5 or norepinephrine </=0.1	Dopamine >15 or norepinephrine >0.1
CNS Glasgow Coma Score	15	13–14	10–12	6–9	<6
Renal Creatinine (mg/dL) or urine output (mL/d)	<1.2	1.2–1.9	2.0–3.4	3.5–4.9 or <500	>5.0 or <200

Lima et al (2009) The prognostic value of the subjective assessment of peripheral perfusion in critically ill patients. Crit Care Med: 37(3) pp 934-938.

Recognition of Shock State

- Tachycardia.
- Vasoconstriction.
- ↓Cardiac output.
- Narrow pulse pressure.
- ↓MAP.
- ↓Blood flow.
- Mounting metabolic debt leading to apoptosis (cellular death) and ultimately patient death.

TRAUMATIC SHOCK STAGES (After Blood Loss per ATLS)

- I) Up to 15% of blood volume= anxiety.
- II) Up to 30%= increased HR, normal BP, Increased RR,decreased UO, anxiety.
- III) Up to 40%= Increased HR,RR, anxiety, confusion,decreased BP, oliguria.
- IV) Greater than 40%= Increased HR,RR, anuric, confusion leading to lethargy.

Pathophysiology of Shock

Compensated Traumatic Shock (not an issue if homeostasis returned)
Decompensated Traumatic Shock (Lack of perfusion builds a Debt of local cell damage that yields a toxic effect when perfusion restored)
Sub-Acute Irreversible Shock=MSOF
Acute Irreversible Shock=PPBBBOL

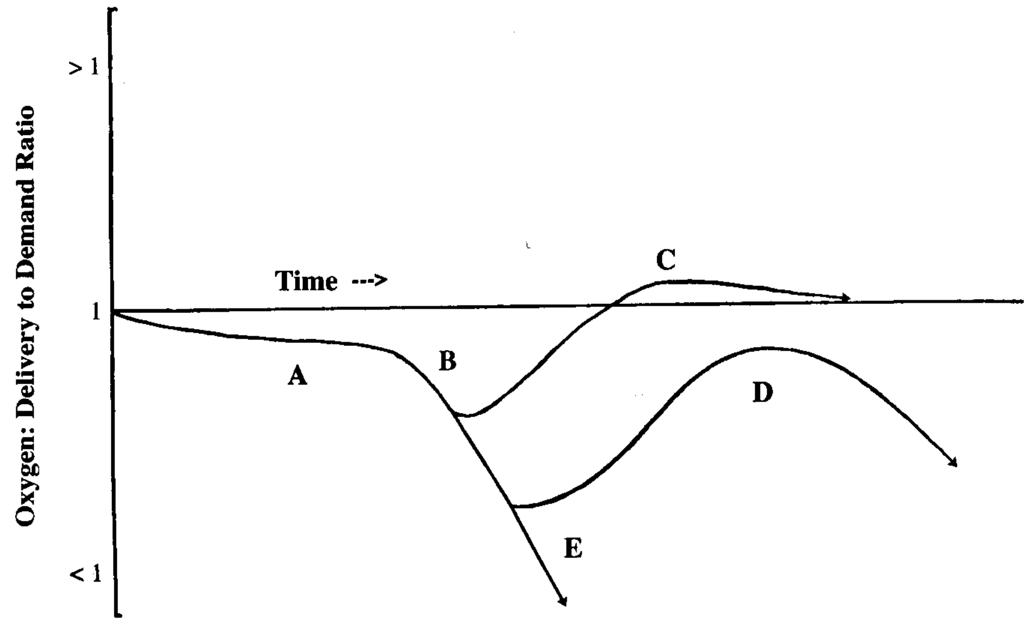


Figure 1. Traumatic shock, and the potential outcomes. In early shock (A), there is only a small drop in oxygen delivery, owing to compensation by the cardiovascular system. Decompensated shock (B) is characterized by an accelerating defect in oxygen delivery. Recovery from decompensated shock (C) includes a hyperdynamic period as the body's oxygen debt is repaid. In subacute irreversible shock (D) the macrocirculation is restored and bleeding stopped, but hypoperfusion has been severe enough that oxygen debt cannot be repaid. Lethal multiple organ system failure develops. Acute irreversible shock (E) occurs when hemodynamic control is never regained. The patient exsanguinates and dies in cardiovascular collapse.

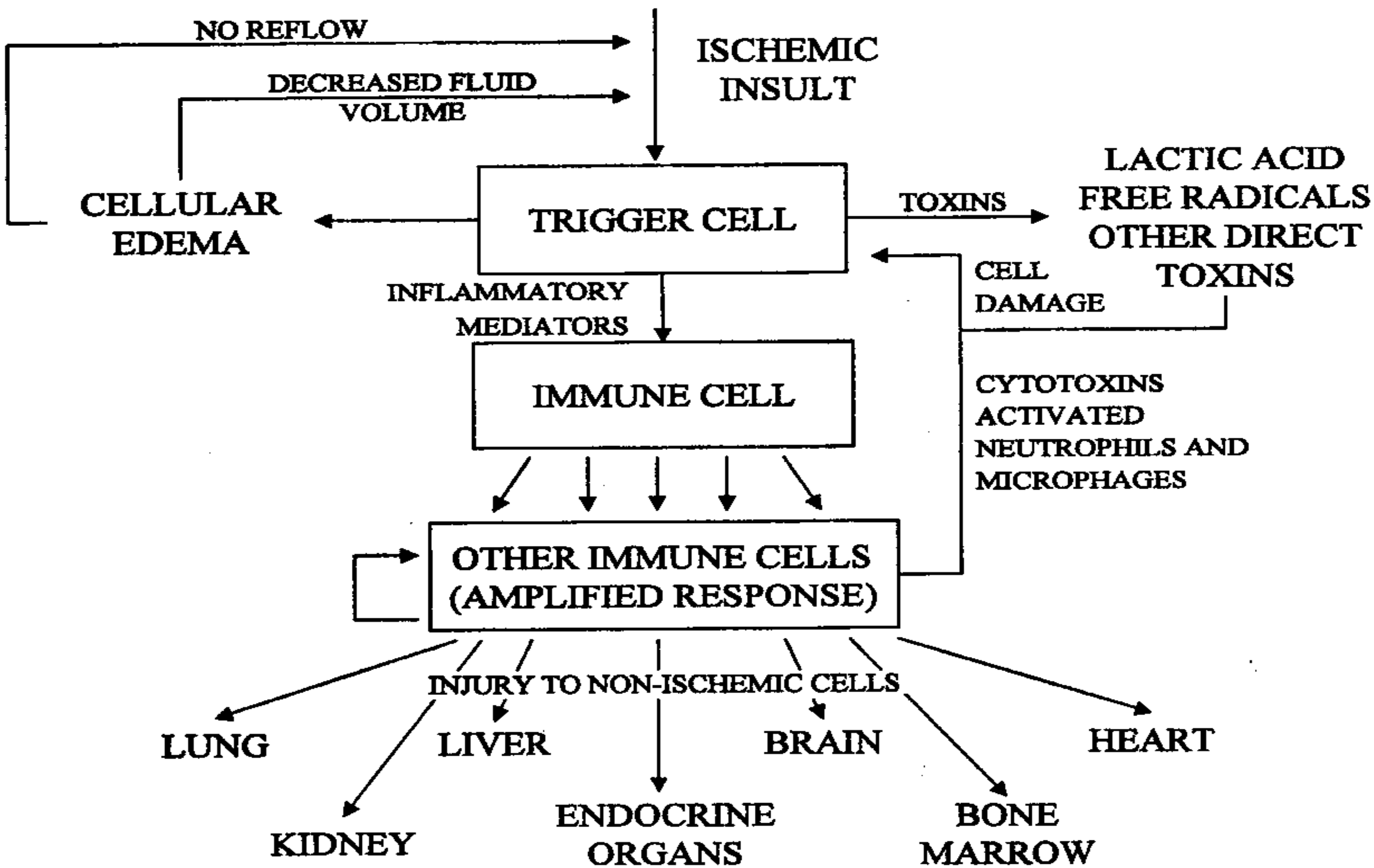


Figure 2. The inflammatory cascade. Ischemia at the cellular level produces a chain reaction of events that ultimately leads to cell damage and death. Dozens of mediators and cytokines are known to participate in this process following traumatic injury and during and after a period of shock.

Wilson (2007, 217)

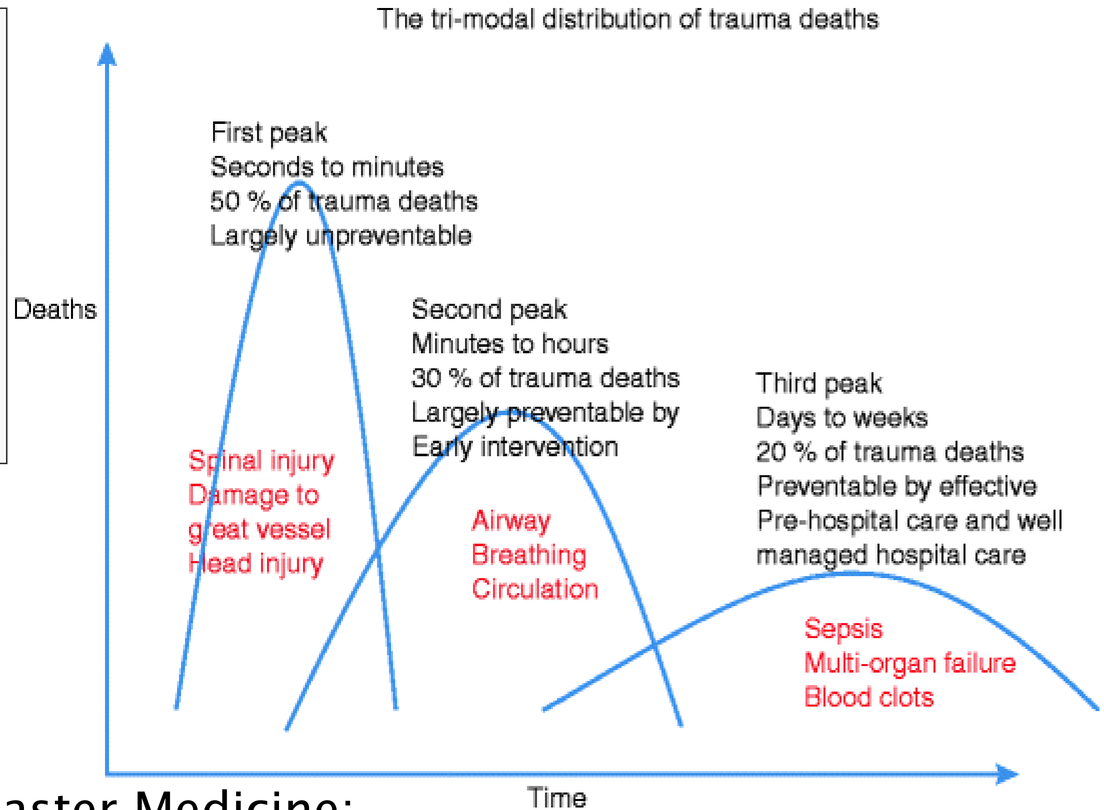
Trimodal Distribution of Trauma Deaths

Table 1

Trunkey's 1983 classification of immediate, early, and late trauma deaths*

Deaths	Timing†	Location	Cause	Interventions‡
Immediate	Minutes	Scene	Nonsurvivable injuries	Injury prevention
Early	Hours	Hospital	Severe injuries, potentially survivable with optimal care	Improved access to trauma care
Late	Weeks	Hospital	Multiple organ failure, sepsis	Improved resuscitation/critical care

*From Trunkey, 1983 (3).
†Timing of death after sustaining injuries.
‡Primary interventions with the most potential to reduce trauma deaths.



MacGarty & Nott (2013) Disaster Medicine:
A Case Based Approach: Springer-Verlag London.

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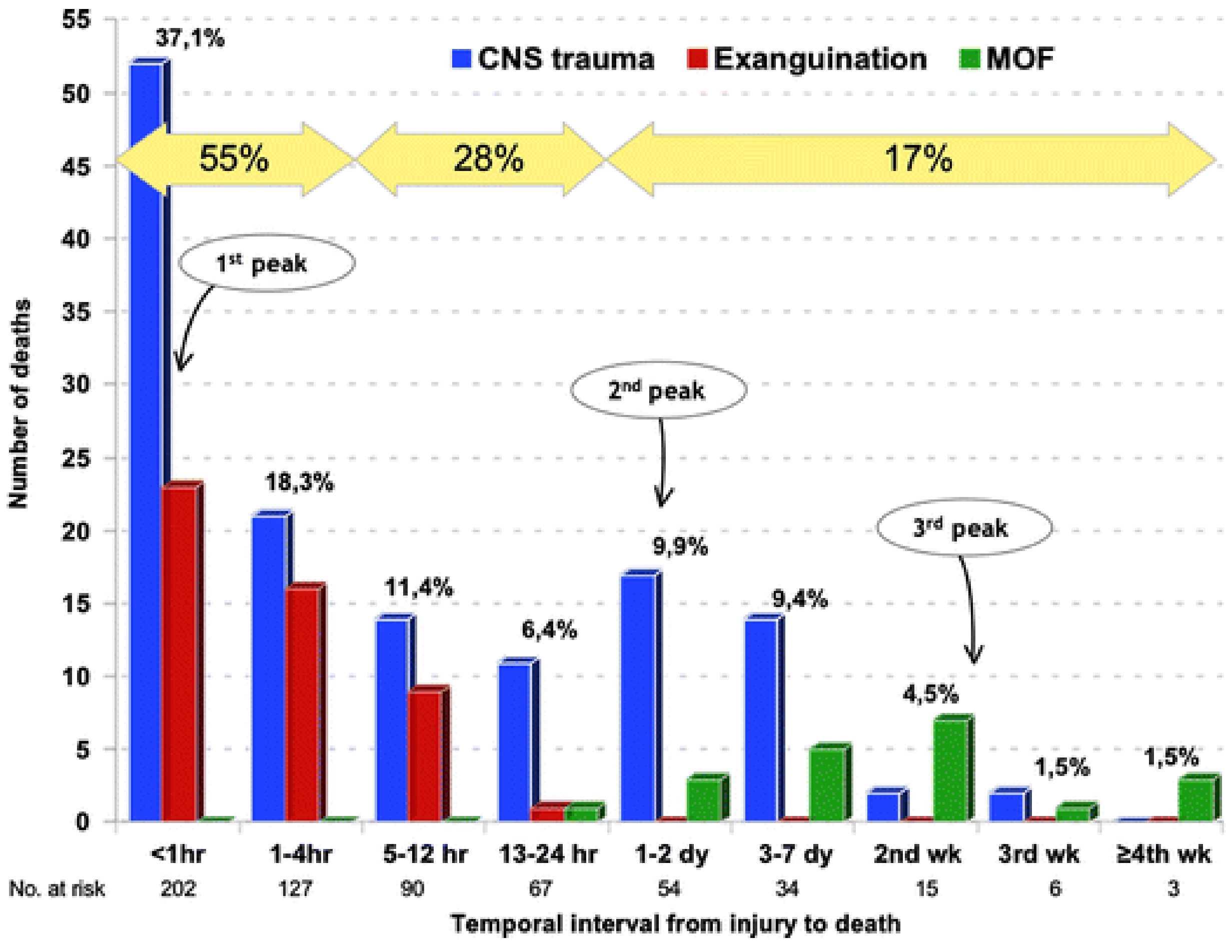
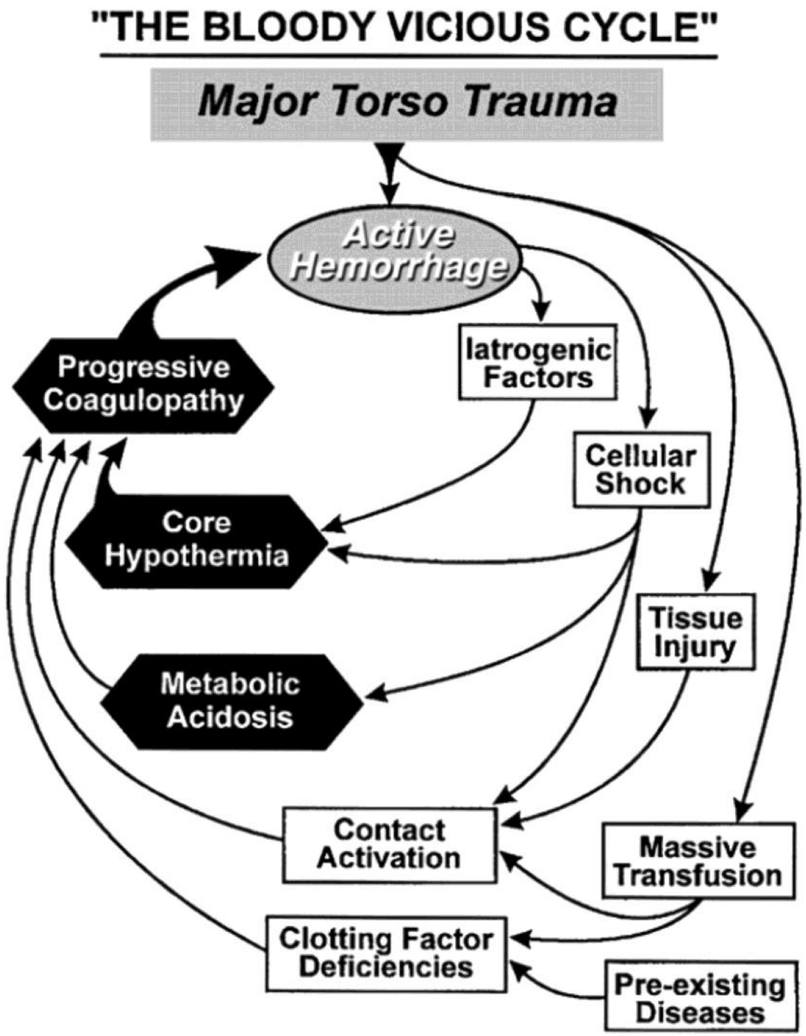
From: Epidemiology and Contemporary Patterns of Trauma Deaths(2007) World Journal of Surgery Vol 31(11) 2092-2103



The pathogenesis of the bloody vicious cycle following major torso trauma is multifactorial, but usually manifests as a triad of refractory coagulopathy, progressive hypothermia, and persistent metabolic acidosis.



Sihler K C , Napolitano L M
Chest 2009;136:1654-1667

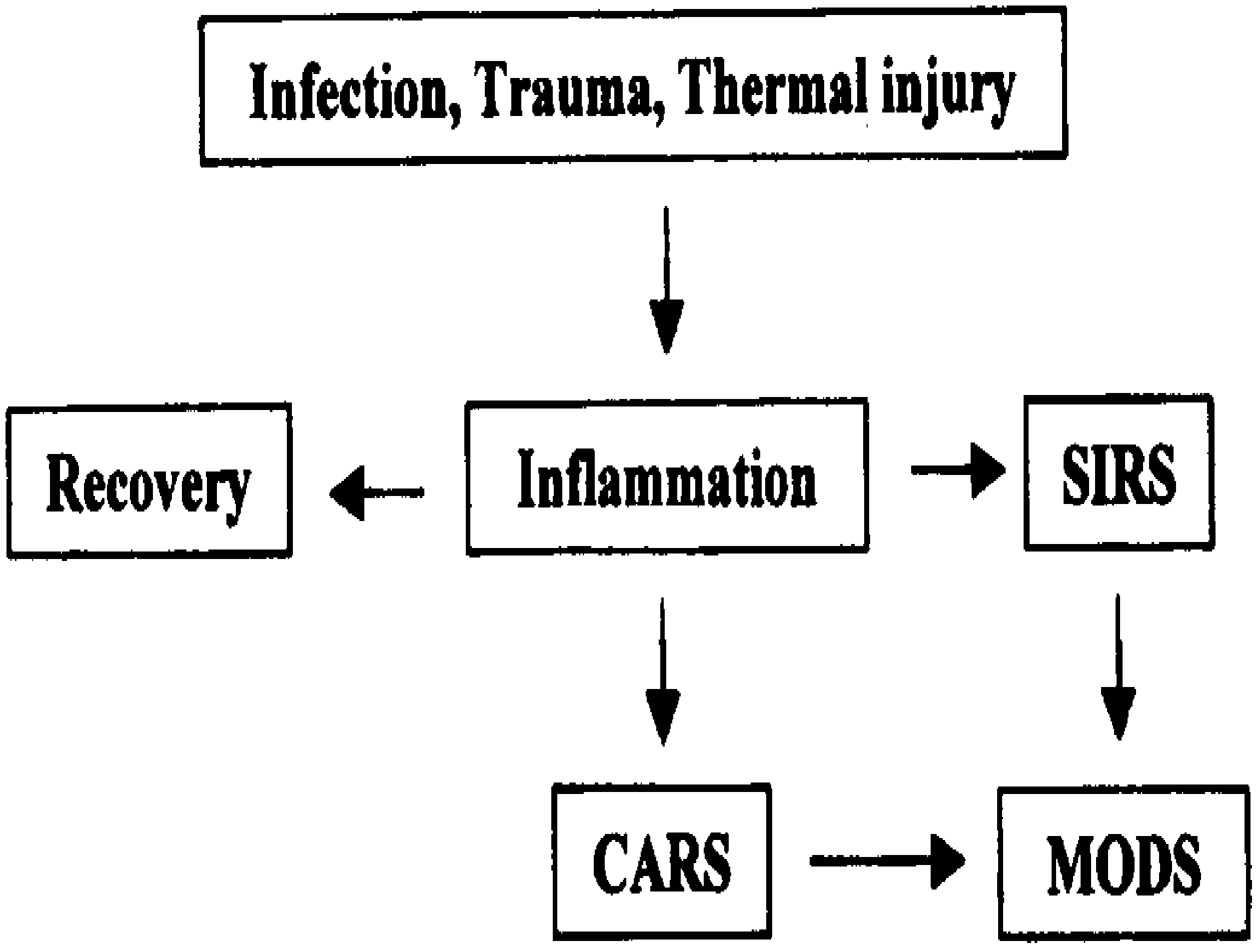


FIG. 1. Inflammatory and antiinflammatory responses to injury. SIRS = systemic inflammatory response syndrome; CARS = counter antiinflammatory response syndrome; MODS = multiple organ dysfunction syndrome.

ORGAN SYSTEM RESPONSE TO TRAUMATIC SHOCK

- The stages of traumatic shock are closely related to the body's response to hemorrhage or inadequate perfusion.
- Macrocirculatory response is mediated by the neuroendocrine system.
- Microcirculatory responses will determine the overall patient outcome.

CENTRAL NERVOUS SYSTEM

- Prime trigger of neuroendocrine response to shock. The tendency is to maintain perfusion to the heart and brain, at the expense of other tissues.
- Kidneys and adrenal glands produce renin, angiotensin , aldosterone, cortisol, erythro-poientin, and catecholamines.

HEART AND LUNGS

- Initially, well maintained in early shock. Lactate, free radicals and other factors are released from ischemic cell beds to act as negative inotropes.
- The lungs tend to be a filter for the inflammatory by-products. This can unfortunately lead to ARDS and MSOF.

GI /LIVER/SKELETAL MUSCLES/IMMUNE SYSTEM

- The gut is one of the earliest organ systems altered by poor perfusion.
- Glucose, nutritional status, and coagulation function can result from liver insult.
- Skeletal muscles are a prime source of lactate and free radicals.
- Sepsis is a result of the bodies altered immune system.

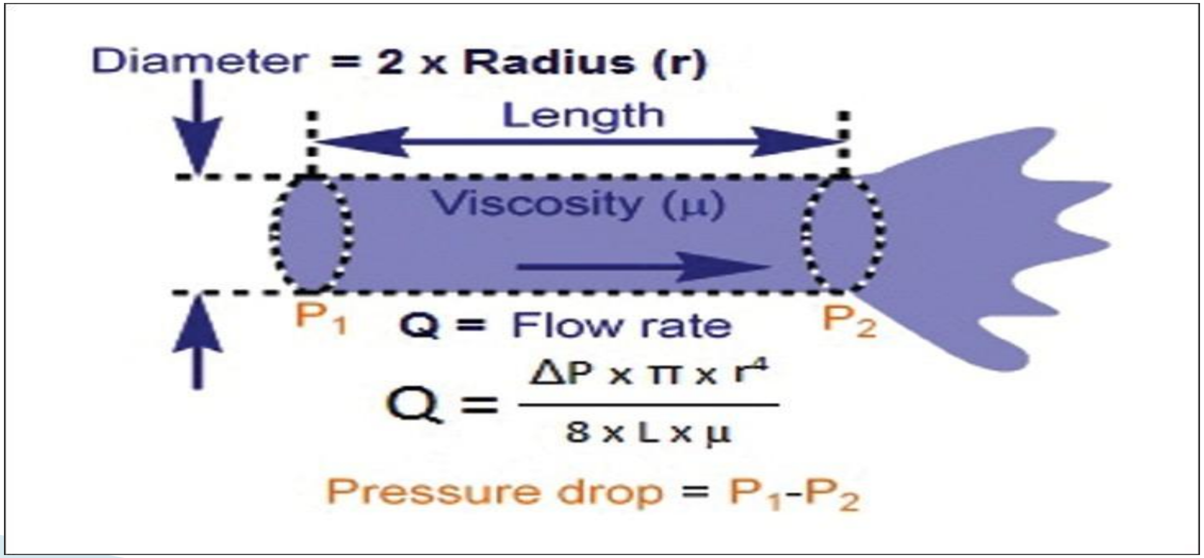
Mainstays of Therapy

- Volume resuscitation and blood/blood product transfusion is the mainstay of treatment for hemorrhagic shock.
- Large bore IV access and rapid infusion systems are invaluable adjuncts in the early resuscitation stages.
- Hypothermia worsens acid-base disorders, coagulopathies and myocardial function. Use fluid warmers, convection forced-air blankets, increased OR room temperature, and the like to combat this preventable problem.

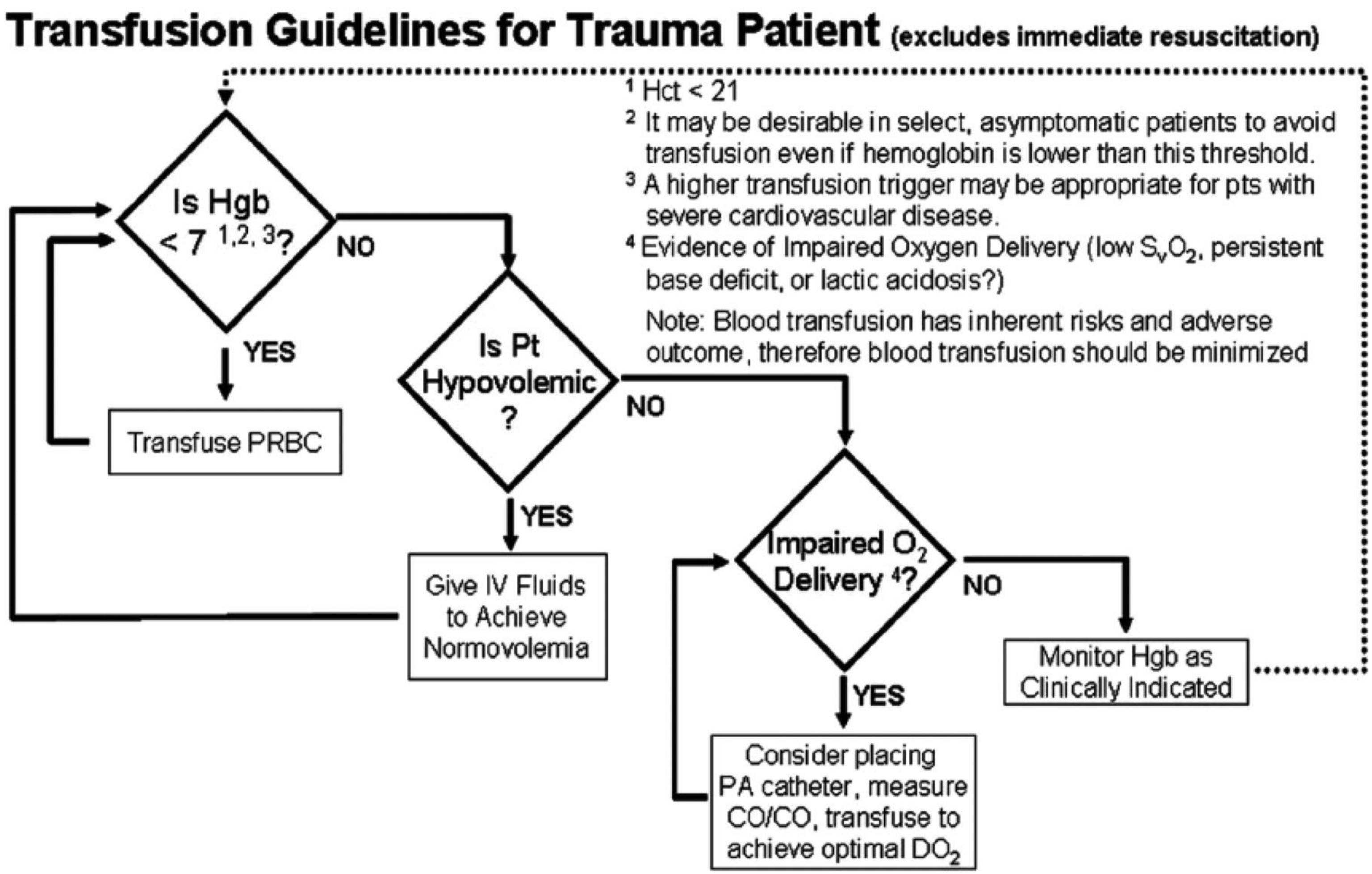
Green et al. (2012) International Journal of Critical Illness & Injury Science: Volume 2(3): pp 135-142.

Gauge	Published flow rate (ml/min)*	Flow rate gravity (ml/min)[2]	Flow rate level 1 (ml/min)[3]	Flow rate RIS (ml/min)[3]	10 cc syringe pump (ml/min)[4]
4 Fr	Not published	286	450	516	Not published
5 Fr	Not published	380	533	667	Not published
6 Fr	Not published	480	548	702	Not published
7 Fr	Not published	Not published	564	772	Not published
8.5 Fr	Not published	674	596	857	Not published
14	330	Not published	488	584	Not published
16	220	Not published	368	412	Not published
18	105	92	209	205	Not published
20	60	51	140	144	Not published
22	35	37	Not published	Not published	106
24	17	Not published	Not published	Not published	78

*All values are for crystalloid solutions, published flow rate from BD packaged IV catheters



Guidelines for transfusion in the trauma patient to guide PRBC transfusion therapy for critically ill patients after the immediate resuscitation phase



Sihler K C , Napolitano L M Chest 2009;136:1654-1667

Massive Hemorrhage

Definition of massive hemorrhage

- Loss of one blood volume over 24 hours.
- Loss of 50% blood volume over 3 hours.
- Loss of 150 mL per minute.

Massive Hemorrhage

Total blood volume for 70 kg adult

.07 x 70 kg = 4.9 liters.

50% of total blood volume for 70 kg adult

.50 x 4.9 liters = 2.45 liters.

40% of total blood volume for 70 kg adult

.40 x 4.9 liters = 1.96 liters.

Note: An acute loss of 40% blood volume is immediately life threatening!

The Problem

- Trauma mortality
  - 40% related to failed hemorrhage control
    - Sauaia A, et al: J Trauma 1995; 38:185-93.
- Massive hemorrhage
  - 2nd cause of pre-hospital death
    - Mackenzie EJ, et al: J Trauma 2000, pp. 21-39.

Potentially Preventable Injury Mortality

- Of the injured who will die, 50% die before they reach a hospital.
- Head injury is the major cause of death in the field and in the hospital.
- Uncontrolled hemorrhage or MOF-related shock is the cause of death in about 40% of deaths.
- About 20% of hemorrhagic deaths appear to have been potentially preventable.

Sources of Life-Threatening Hemorrhage

- Chest.
- Abdomen.
- Retroperitoneum.
- Soft Tissue Compartment.
- External (“The Street”).

Laboratory Evidence of Shock

- Metabolic acidosis / base deficit
- Elevated lactate
- Anemia
- Elevated osmolarity
- {Coagulopathy}

Mortality of Massive Transfusion

Mortality versus units of blood transfusion.

- Average mortality 47% if more than 10 units.
- Mortality 28% if received 10 to 19 units.
- Mortality 65% if received 20 to 39 units.
- Mortality 83% if received 40 or more units.

Wilson WK, et al. Am Surg. 1987; 53:310-317.

Mortality of Massive Transfusion

The Shock Trauma Experience

- Mortality 22%   1 - 10 units.
- Mortality 30% 11 - 20 units.
- Mortality 50% 21 - 40 units.
- Mortality 59% greater than 40 units*.

*One patient received 126 units RBC's
and was discharged to home.

Overview: Complications of Massive Transfusions

- The goal of treatment is the rapid and effective return of an adequate blood volume. Adequate return implies that body hemostasis, oxygen carrying capability, and oncotic pressure are returned to pre-insult levels and are maintained through-out the hospitalization period.
- Metabolic demands must be met.

DEFINITION of
MASSIVE TRANSFUSION EVENT (MTE):

Clinical Presentation:

Hypovolemic shock and coagulopathy can lead to widespread cellular dysfunction and organ damage. The MTE will provide a guide for efficient and effective delivery of blood products for Trauma and other patients who meet criteria for massive transfusion.

Criteria for activation of MTE

1. Loss of Blood volume in the first hour of activation:
   - Adult patients requiring >4 units of RBCs
   - Pediatric Patients requiring >20 ml/kg of RBCs

2. Loss of RBC units in the first 12 hours of resuscitation:
   - Adults using more than 10 units of RBCs
   - Pediatric patients using greater than 0.1 units/kg of RBCs

R Adams Cowley Shock Trauma Center
University of Maryland

Final 2014

Declaration of a Massive Transfusion Event (MTE)

1. To assure availability of needed blood products in appropriate ratios for massive transfusion
2. To assure appropriate and timely notification of the Blood Bank to allow them to prepare

1 X SBP <90 within 10 minutes of admission
OR
2 X SBP <100 within 10 minutes of admission
OR
Unable to obtain blood pressure

AND

Penetrating torso trauma
OR
Positive FAST
OR
Obvious ongoing external blood loss
OR
Plans to take patient directly to OR

EXCLUSION: Cardiac Arrest with no ROSC

More than 2 units of uncrossmatched PRBC are being given

Note: The first 2 units of uncrossed PRBCs are often given to "catch up" with blood loss that may not be ongoing. Thawed group AB universal donor plasma is typically not needed in this situation unless there is obvious coagulopathy. Group AB plasma should be given with the next 2U of PRBC and in an equal ratio to PRBC thereafter if concern for ongoing hemorrhage

Physician discretion

TRU Tech/Charge RN or OR RN calls the Blood Bank at 8-5630.
Should state:
1. We are declaring a Massive Transfusion Event
2. Patient's MRN and STC number and name, if known.
3. Patient's sex and approximate age
4. Name of Physician in Charge (Trauma or Anesthesia Attending)

Cross-match sample should be immediately "tubed" to the Blood Bank

Once the sample is received and tested, the Blood Bank will immediately prepare:

6 units of type-specific or compatible PRBC
6 units of type-specific or compatible plasma
1 bag of apheresis platelets (6 equivalent units)

The TRU (OR) should send a Tech/Volunteer to the Blood Bank
Type specific or compatible RBC's and plasma should be ready within 30-45 minutes.
DO NOT wait for all products if there is a delay in dispense

The Blood Bank will continue to prepare:

6 units of type-specific or compatible PRBC
6 units of type-specific or compatible plasma
1 bag of apheresis platelets (6 equivalent units)
until informed that the MTE is terminated

Uncrossed matched group O PRBC and group AB plasma should be given until type-specific or compatible products are available.

The completed "cards" should be sent to the Blood Bank as products are used.

Please also call the Blood Bank with ANY use of O Rh-neg PRBC so it can be replaced

Once the need for massive transfusion is over, the TRU or OR calls the Blood Bank at 8-5630.
Should state:
1. We are ending the Massive Transfusion Event
2. Patient's MRN and STC number and name, if known.

Goals for Resuscitation -- Early

- Expedite control of hemorrhage.
- Limited crystalloid infusion.
- BP 80 - 100 systolic.
- Blood products early and often: 1:1:1.
- Frequent lab studies (lactate, ionized Ca).

Intra operative Management

1. Minimize agent – effects of Volatile Anesthetics on CV system.
2. Higher narcotic approach – why?

We often have no idea how volume contracted a patient is CO= HR x SV. Volatile Agents effect SV and afterload. Narcotics primarily effect HR only. If we control for HR and BP drops then we can assume volume depletion.

Early Management: Anesthesia

1. Supportive fluid resuscitation:
   - Avoid acute exsanguination.
   - Minimize risk of organ system failure.
   - Facilitate hemostasis.

2. Preservation of homeostasis:
   - Temperature.
   - Pulmonary function.

Goals for Resuscitation -- Late

- BP > 100 systolic
- Cardiac output normal or high
- Adequate Perfusion!
  - Lactate, base deficit.
  - Mixed venous oximetry.
  - New technology.

Adjuncts to Hemostasis

- Damage control
  - Failure of standard methods.
  - Hypothermia and coagulopathy.
  - Acidosis and metabolic abnormalities.
- Principles of damage control
  - Obtain surgical hemostasis.
  - Temporize hollow viscus injury.
  - Pack coagulopathic bleeding.
  - Resuscitate and correct hypothermia.

Pre-hospital fluids (too ~~much~~ of a good thing?)

time of operative intervention in hypotensive patients with penetrating injuries to the torso.

Methods. We conducted a prospective trial comparing immediate and delayed fluid resuscitation in 598 adults with penetrating torso injuries who presented with a prehospital systolic blood pressure ≤90 mm Hg. The study setting was a city with a single centralized system of prehospital emergency care and a single receiving facility for patients with major trauma. Patients assigned to the immediate-resuscitation group received standard fluid resuscitation before they reached the hospital and in the trauma center, and those assigned to the delayed-resuscitation group received intravenous cannulation but no fluid resuscitation until they reached the operating room.

fluid resuscitation ($P = 0.04$). The mean estimated intraoperative blood loss was similar in the two groups. Among the 238 patients in the delayed-resuscitation group who survived to the postoperative period, 55 (23 percent) had one or more complications (adult respiratory distress syndrome, sepsis syndrome, acute renal failure, coagulopathy, wound infection, and pneumonia), as compared with 69 of the 227 patients (30 percent) in the immediate-resuscitation group ($P = 0.08$). The duration of hospitalization was shorter in the delayed-resuscitation group.

Conclusions. For hypotensive patients with penetrating torso injuries, delay of aggressive fluid resuscitation until operative intervention improves the outcome. (N Engl J Med 1994;331:1105-9.)

FOR the past two decades the preoperative approach to hypotensive patients with trauma in North America has included prompt intravenous infusion of isotonic fluids.[1-3] The rationale for this treatment has been to sustain tissue perfusion and vital

orrhage is surgically controlled.[7-13] More recent studies have demonstrated that in uncontrolled hemorrhage, aggressive administration of fluids may disrupt the formation of thrombus, increase bleeding, and decrease survival.[14-18]

Prehospital Fluids

loss was found in the immediate-resuscitation group, but the volume of blood loss could not be measured in the more applicable prehospital and trauma-center phases of management, when control of internal hemorrhage was not attempted.

Nonetheless, other clinical findings are consistent with the hypotheses derived from experiments in animals. The initial mean systolic blood pressure measured at the emergency center was significantly higher in the immediate-resuscitation group. Also, despite the relatively small amount of crystalloid infused in

Table 3. Systemic Arterial Blood Pressure, Heart Rate, and Laboratory Findings at the Time of Initial Operative Intervention in Patients with Penetrating Torso Injuries, According to Treatment Group.*

Variable	Immediate Resuscitation (N = 268)	Delayed Resuscitation (N = 260)	P Value
Systolic blood pressure (mm Hg)	112±33	113±30	0.98
Diastolic blood pressure (mm Hg)	57±22	60±21	0.10
Heart rate (beats/min)	102±25	104±23	0.25
Hemoglobin (g/dl)	10.7±5.8	11.5±2.6	<0.001
Platelet count ($\times 10^{-3}/mm^3$)	195±97	198±105	0.99
Systemic arterial pH	7.27±0.16	7.28±0.15	0.75

Exposure

- 34° C was the critical point at which enzyme activity slowed significantly, and at which significant alteration in platelet activity was seen. Fibrinolysis was not significantly affected at any of the measured temperatures
  - Watts, Dorraine Day, et al. "Hypothermic coagulopathy in trauma: effect of varying levels of hypothermia on enzyme speed, platelet function, and fibrinolytic activity." *The Journal of Trauma and Acute Care Surgery* 44.5 (1998): 846-854.

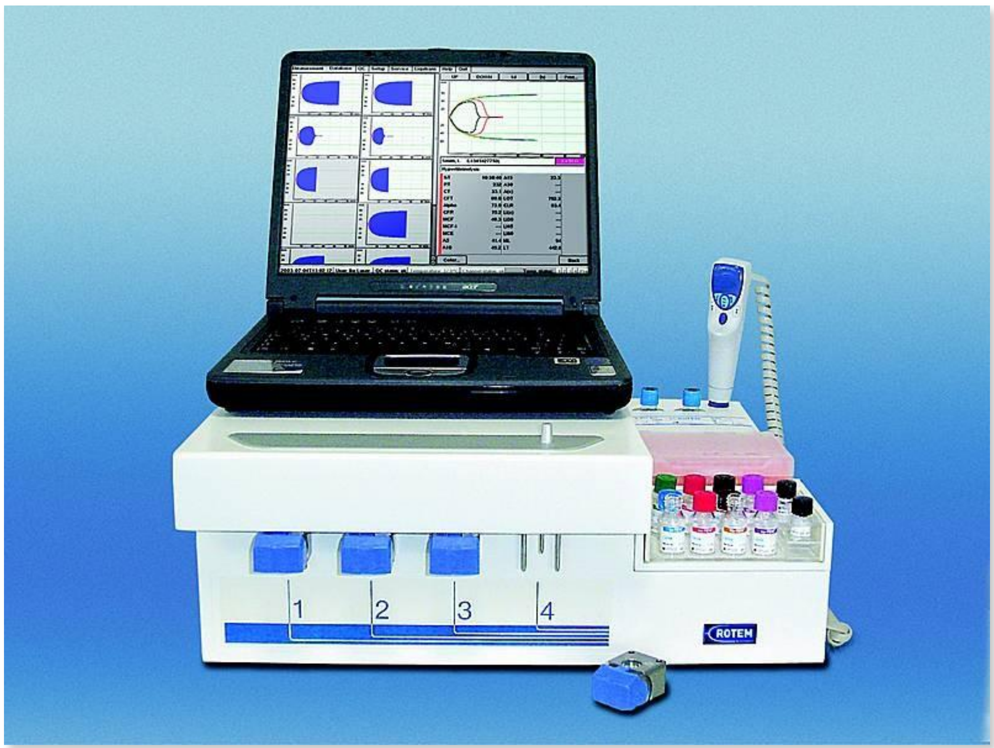
- Keeping pt warm
  - Warm blood products
  - Bair hugger type devices
  - Warm operating room

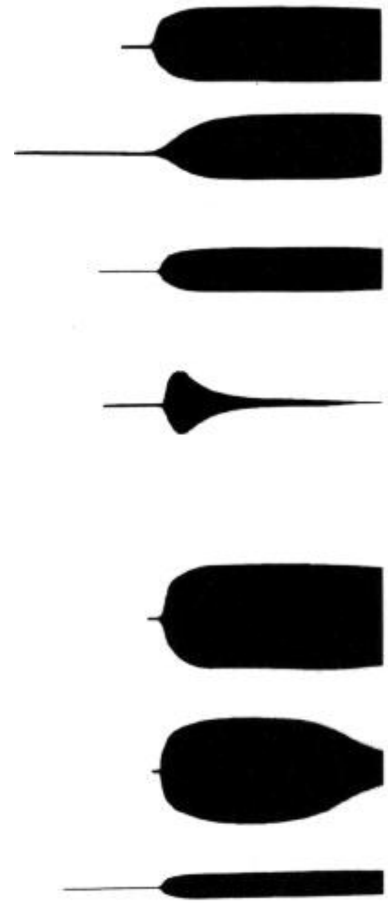
Monitoring

- Basic
- Advanced
  - A line
  - CVP?
  - PPV- FloTrac
  - TEE

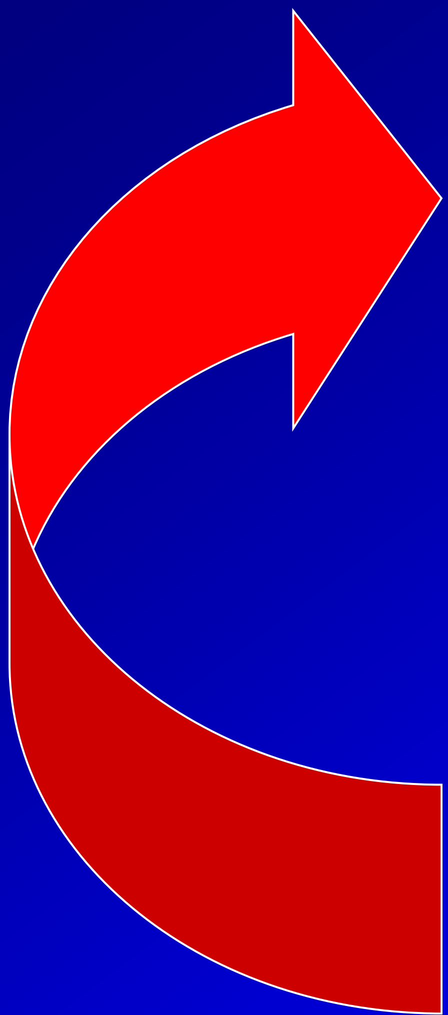
- Labs- CBC, coags, lytes, ABGs
- POC
  - Hemoque- Hgb
  - iStat- lytes/gases
  - ROTEM- coagulation

Clotting Dynamics





Trauma Associated Coagulopathy



Hemorrhage

↓

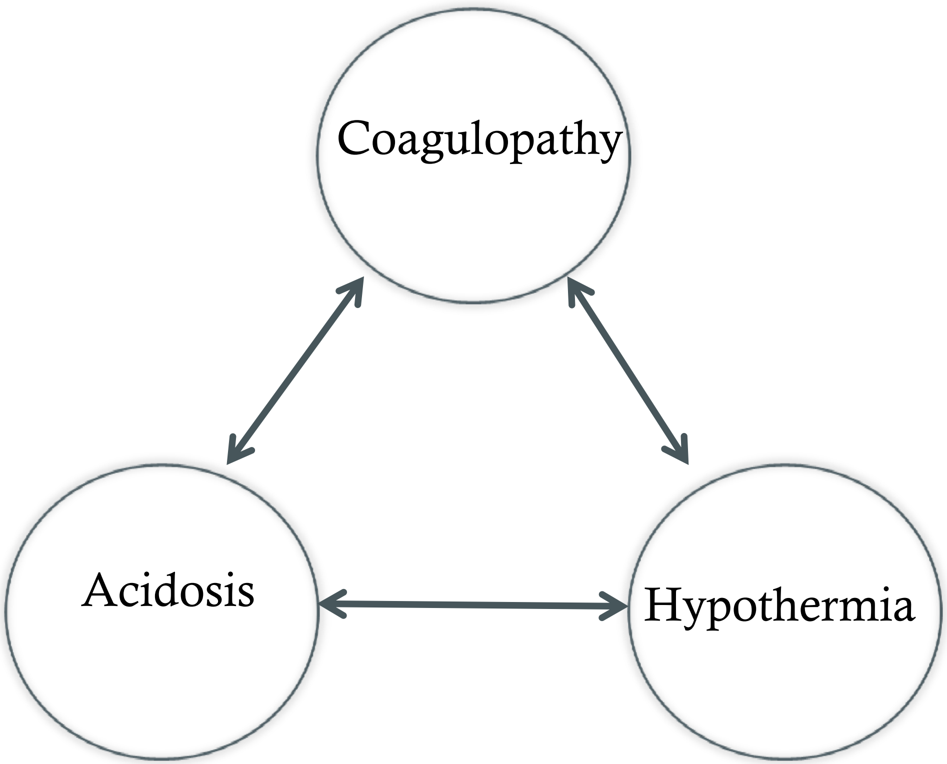
Massive transfusion

↓

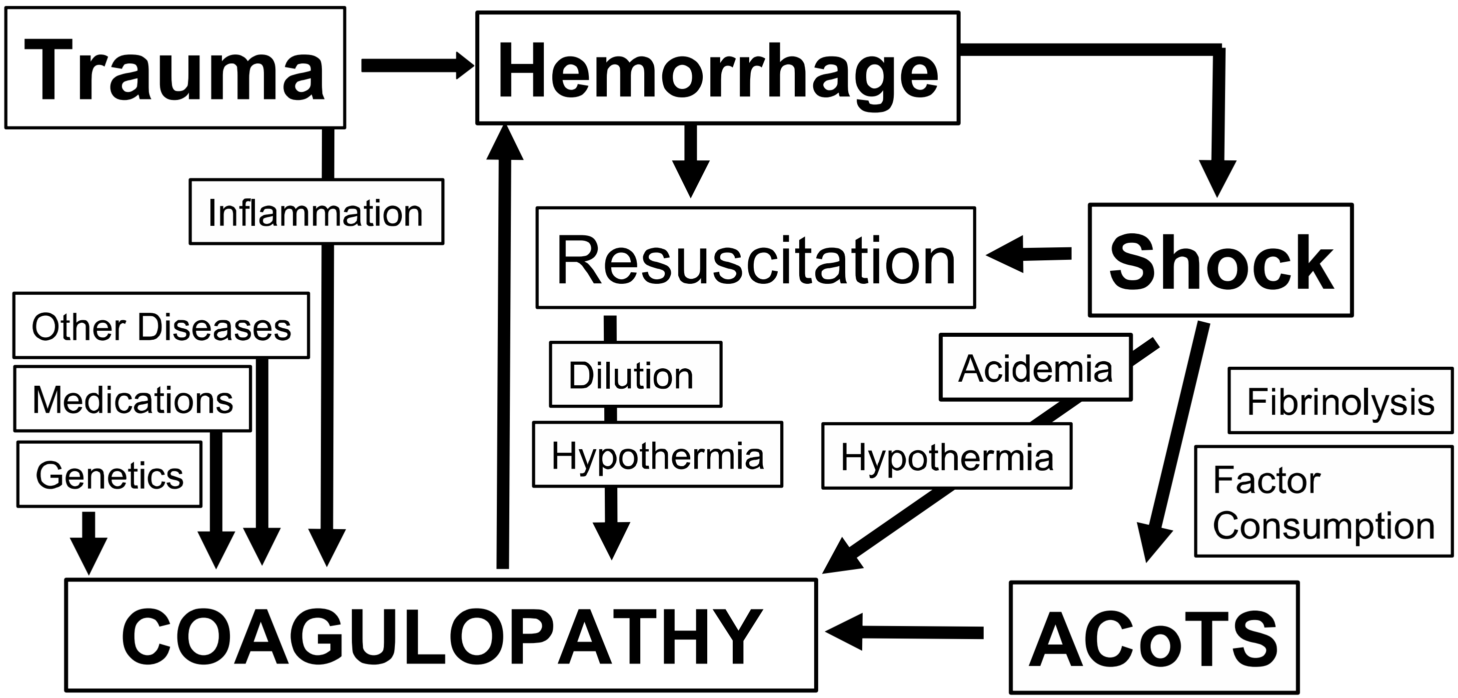
Hypothermia

Acidosis Δ **Coagulopathy**

Lethal Triad



More than the Lethal Trial



Hess JR, Brohi K, Dutton RP; et al. The coagulopathy of trauma: a review of mechanisms, J Trauma 2008 654 748-754.

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Adjuncts to Hemostasis

- Damage control
  - Failure of standard methods
  - Hypothermia and coagulopathy
  - Acidosis and metabolic abnormalities
- Principles of damage control
  - Obtain surgical hemostasis
  - Temporize hollow viscus injury
  - Pack coagulopathic bleeding
  - Resuscitate and correct hypothermia

Damage Control Surgery (DCS)

- To minimize the occurrence of: Bleeding begets volume resuscitation begets more bleeding begets “lethal triad” begets death; Damage Control has emerged
- Acidosis◄►Hypothermia◄►Coagulopathy
  - DCS avoids extensive procedures on unstable patients, stabilizes potentially lethal problems at initial operation, and applies staged surgery after successful initial resuscitation

(Cirocchi R., et al. (2010) Damage control surgery for abdominal trauma. Cochrane database of systematic reviews . Issue 1

Dutton, RP. Damage Control Anesthesia. *Trauma Care*. 2005;15:197-201.

Table 1. Goals for Damage Control in the Severely Injured Patient

- Stable airway and oxygenation
- Hemostasis—control of life-threatening hemorrhage
  - Exploratory laparotomy or thoracotomy
  - Rapid, wide exposure
  - Excision over repair of “expendable” organs
  - Focus on hemostatic procedures only
    - -Vessel ligation or repair (avoid grafting if possible)
    - -Packing for diffuse bleeding
  - Temporary closure
  - Angiographic embolization in selected cases
- Effective analgesia and sedation
- Appropriate blood composition:
  - Oxygen-carrying capacity (red blood cells)
  - Clotting potential (platelets, clotting factors)
  - Chemistry (especially calcium, glucose, potassium, chloride)
- Stabilization/reversal of tissue acidosis
- Normothermia

Table 2. The Essentials of Damage Control Anesthesia

- Airway and ventilator management
  - Rapid sequence intubation
  - Titration of ventilation
- Control of bleeding
  - Deliberate hypotensive resuscitation
  - Maintenance of blood composition
- Preservation of homeostasis
  - Normothermia
  - Restored and sustained end-organ perfusion
- Analgesia and sedation

Table 3. Resuscitation Goals *During* Damage Control Surgery*

- Systolic blood pressure 90 mm Hg
- Heart rate <120 beats per minute
- Pulse oximeter functioning, SaO_2 >95%
- Urine output present
- $PaCO_2$ <50 torr
- pH >7.25
- Hematocrit >25%
- Lactate stable or decreasing
- Ionized calcium >1.0
- International normalized ratio <1.6
- Platelets >50,000
- Normothermia
- Deep anesthesia

*Lower blood pressure may be tolerated as long as acidosis is not worsening.

Damage Control Resuscitation

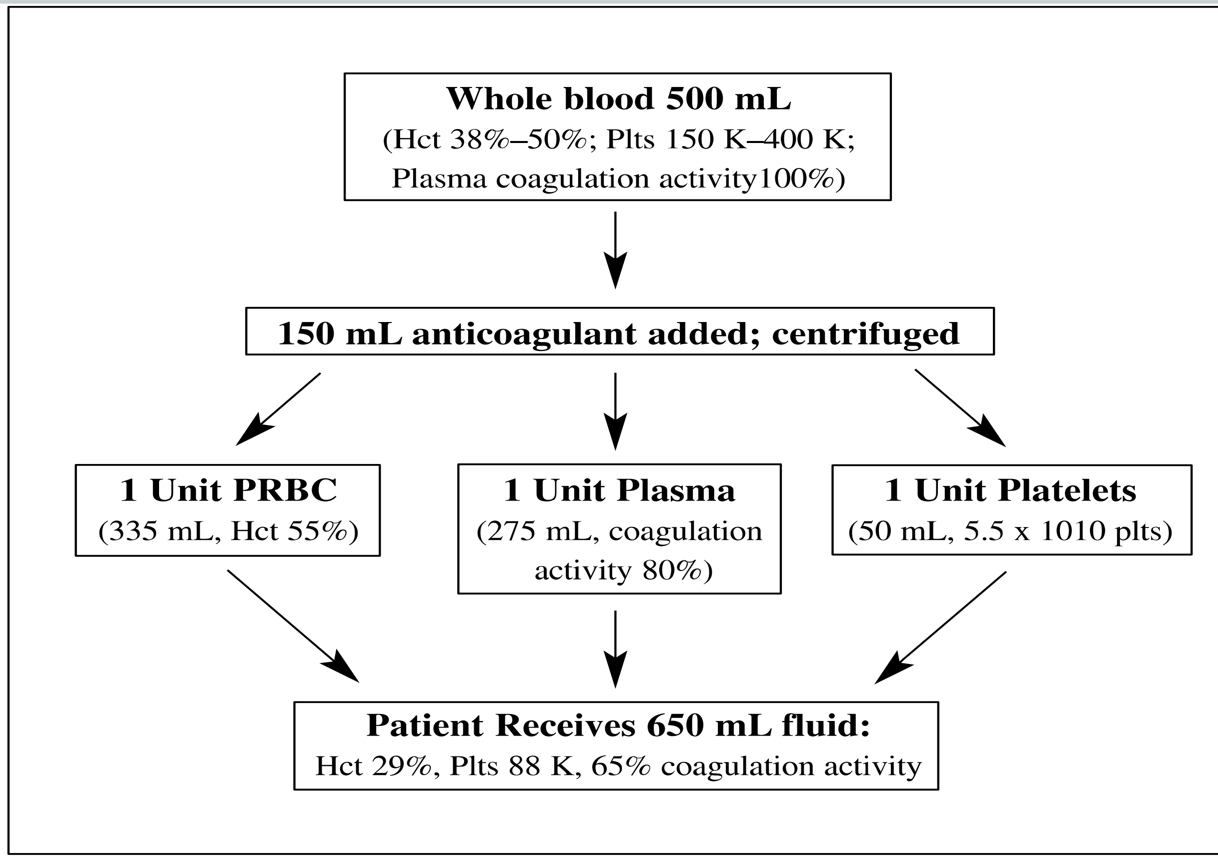
- In severely injured casualties DCR consists of 2 parts and is initiated within minutes of facility arrival
  - Resuscitate to keep SBP approx 90 mm Hg (to prevent rebleeding from any recently clotted vessels)
  - Intravascular volume restoration using a 1:1 or 1:2 ratio of FFP to PRBCs (minimize dilutional coagulopathy)
  - It is critical that major sources of blleding are addressed visa a vie Damage Control Surgery concepts

Holcomb JB., et al. (2007) Damage control resuscitation: Directly addressing the early coagulopathy of trauma. J Trauma. 62. 307-310.

Resuscitation Strategies

- Ratio based resuscitation
  - RBC:FFP:PLTs (currently 1:1:1) Ongoing studies will help guide this concept
- Laboratory based resuscitation
  - Lab delays
  - Lost samples
- Point of Care
  - Coagulation con
  - centrates
  - ROTEM

Component Therapy



Dutton, R. P. (2012), Resuscitative strategies to maintain homeostasis during damage control surgery. Br J Surg, 99: 21–28. doi: 10.1002/bjs.7731

Dilution Is Inevitable When Giving Blood Components

- Whole blood 500 mL
  - Hct 38%–50%
  - Plts 150 K–400 K
  - Plasma coagulation factors = 100%
- Components
  - 1 U PRBC = 335 mL with Hct 55%
  - 1 U Plts = 50 mL with 5.5 x 10^{10} Plts
  - 1 U plasma = 275 mL with 80% coagulation activity

Thus: 1 U PRBC + 1 U Plts + 1 U FFP = 660 mL with Hct 29%, Plts 88 K/µL, and coagulation activity 65%.

FFP, fresh frozen plasma; Hct, hematocrit; PRBC, packed red blood cells.

IMPLICATIONS

- Avoid the 5 H's
  - hypoxia,
  - hyoventilation,
  - hypovolemia,
  - hypotension,
  - hypothermia.
- Always practice universal precautions.
- Intubate/protect the airway early with CHI, airway trauma(blunt/penetrating, burns).
- Remain suspicious(was anything missed?)
- Do not forget the family/significant others (keep them informed and help them get support).

Implications

- Always use the largest and shortest bore IV catheter possible(secure it well however, under pressure and poorly secured it might work its way out).One line is rarely enough.
- A urinary catheter is a standard monitor for volume resuscitation (hematuria can be one of the most confirming signs of a transfusion reaction under general anesthesia).

Implications

- Alert the Blood Bank early if a massive resuscitation is expected.
- An extra pair or two of hands makes the resuscitation process much easier.
- Consider cell salvage (if appropriate) and don’t forget to request a perfusionist and a Rapid infusion system (RIS) or an FMS 2000 or a level 1 system (you must warm the blood products).

Implications

- Track patient perfusion by sending ABG's and periodic Lactate levels. Track homeostasis by monitoring CBC's, coags (TEG) and electrolytes. Track ionized calcium levels. Urine output can aid in assessment. Note for respiratory variation in pulse ox and arterial waveforms.
- If arterial pressure monitoring is employed, make sure the connection is tight and the pressure bag for flush has adequate flow.

Implications

- Don’t loose track of fluids administered. Stack bags on the floor, and fill out those blood product slips (after the case is over is better than not at all).
- Universal precautions for all-(gloves, mask, and eye protection are mandatory).
- Keep track of blood loss-watch for phantom suction. Cell save as much as possible.

Implications

- Combat hypothermia. Utilize Forced Air Warming Devices, Fluid Warmers, and warm the environment as much as possible. Cover all portions of body possible during surgery).
- Try not to fall too far behind with your resuscitation efforts. The surgeons can help by quickly repairing bleeders, and if all else fails pack the wound or clamp vessels and get ready to go off to Angio (damage control surgery).

Thrombocytosis in Trauma Patient: What is the significance?

- A retrospective study looked at 3,286 trauma patient and found that:
  - Reactive thrombocytosis is a common finding in trauma patient in the ICU
  - It was associated with more severally injured patients and produced a higher rate of DVTs/ PEs
  - However, patients with higher platelet counts had significantly lower mortality rates and longer ICU and hospital stays than patients who did not have thrombocytosis

Salim A., et al. (2009) What is the significance of thrombocytosis in patients with trauma? J Trauma. 66(5). 1349-1354.

Basic Rules of Trauma

- The C-spine is never initially clear.
- The stomach is always full.
- Altered LOC is caused by a head injury.
- The patient is always hypovolemic.
- Partial airway obstruction may progress to complete obstruction.
- The patient may already be premedicated.

References

- Ali H. Mokdad,et.al. (2004). Causes of preventable deaths in the United States. *Journal of the American Medical Association, 291*(10), 1238-1245.
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- Wilson, W.C., Grande, C.M., & Hoyt, D.B. (2007). *Trauma emergency resuscitation perioperative anesthesia surgical management .* New York: Informa Healthcare.

Shock is defined physiologically as inadequate delivery of substrates and oxygen to meet the metabolic needs of the tissues. As cells are starved of oxygen and substrate, they can no longer sustain efficient aerobic oxygen production. Aerobic metabolism generates 36 ATP molecules per glucose molecule. As oxygen delivery (DO_2) is impaired, the cell must switch to the much less efficient anaerobic metabolic pathway, which generates only 2 ATP molecules per molecule of glucose, with resulting production and accumulation of lactic acid. (**Adam J Schwarz, MD Emedicine 2009)**