Obstetric Shock

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OB Shock- Lecture Organization

- **Definition/Classification of Shock**
- **Pathophysiology of Shock**
- **Hemorrhagic Shock**
- **Sepsis (SIRS)**
- **Resuscitation**
- **Special Circumstances**

Shock - Statistics

- **One of the most common causes of death in the US today**
- **Shock and Respiratory Failure together account for majority of emergent ICU admissions**
- **Shock mortality is high**

(CDC, 1992; Rodriguez and Rosenthal, 1997)

Shock Statistics - Continued

- **Septic Shock Mortality - 40%-60% (non-pregnant)**
- **Septic Shock Mortality (Pregnancy) - LOWER (20%- late septic shock-relative lack of underlying diseases)**
  - **younger age**
  - **source/site**

  (NIH, 1992; Blanco, 1981; Porter, 1997)

Shock - Definition

- **Functionally, "Shock" represents a clinical condition in which intravascular volume (and/or perfusion) is below intravascular capacitance (and/or demand)**
- **Operationally, "Shock" is broadly divided into three types:**
  - **Hypovolemic**
  - **Cardiogenic**
  - **Neurogenic**

Shock - Obstetrics

- **Lecture will focus predominantly on two conditions that incite the pathophysiologic cascade of shock:**
  - **Hemorrhagic**
  - **Septic**

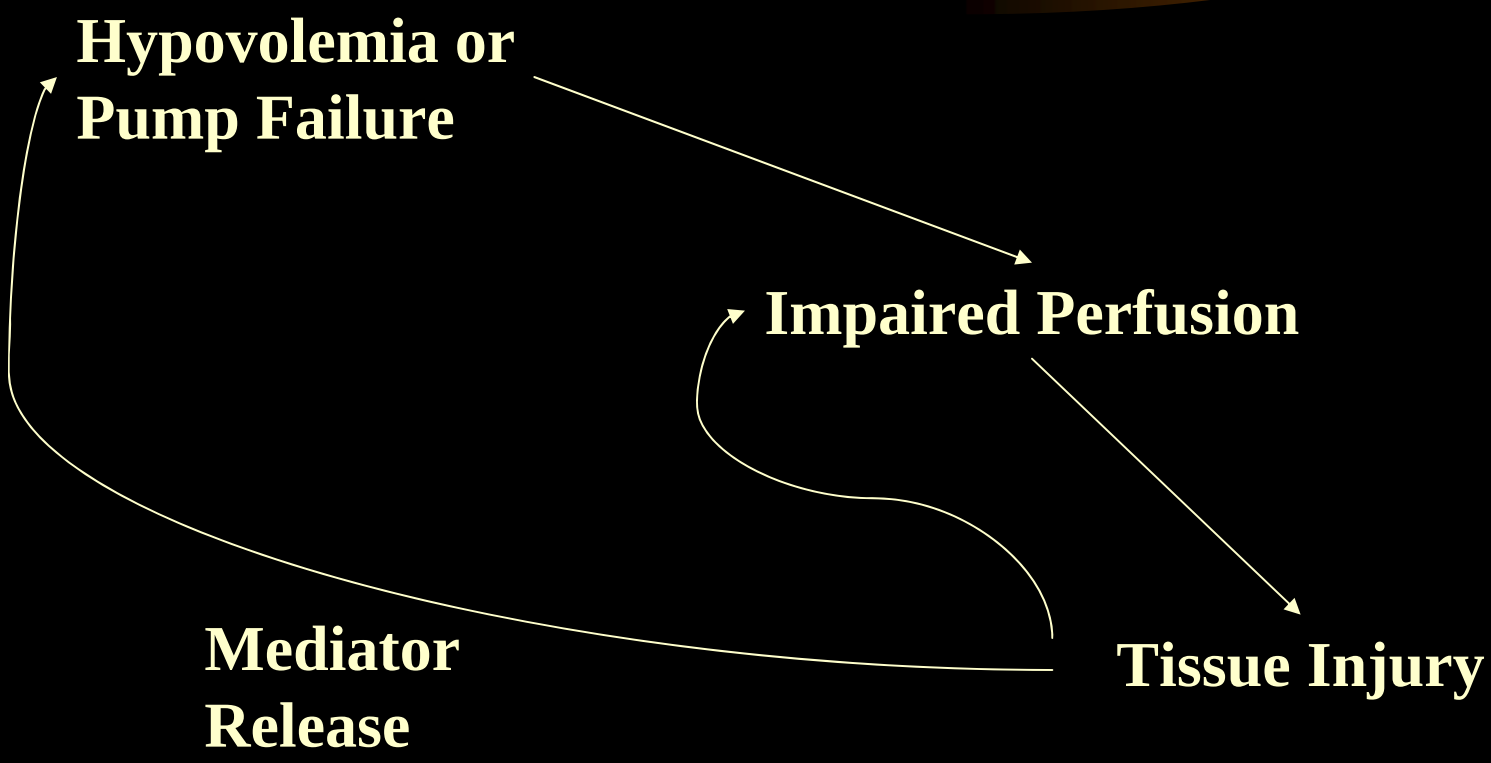
Shock - Pathophysiology

- **Primary pathophysiologic mechanism in shock is impaired oxygen utilization by tissue**
- **Impaired utilization encompasses a continuum**
- **Impaired utilization may be from:**
  - **reduced perfusion**
  - **deficient uptake**
  - **abnormal relative perfusion**

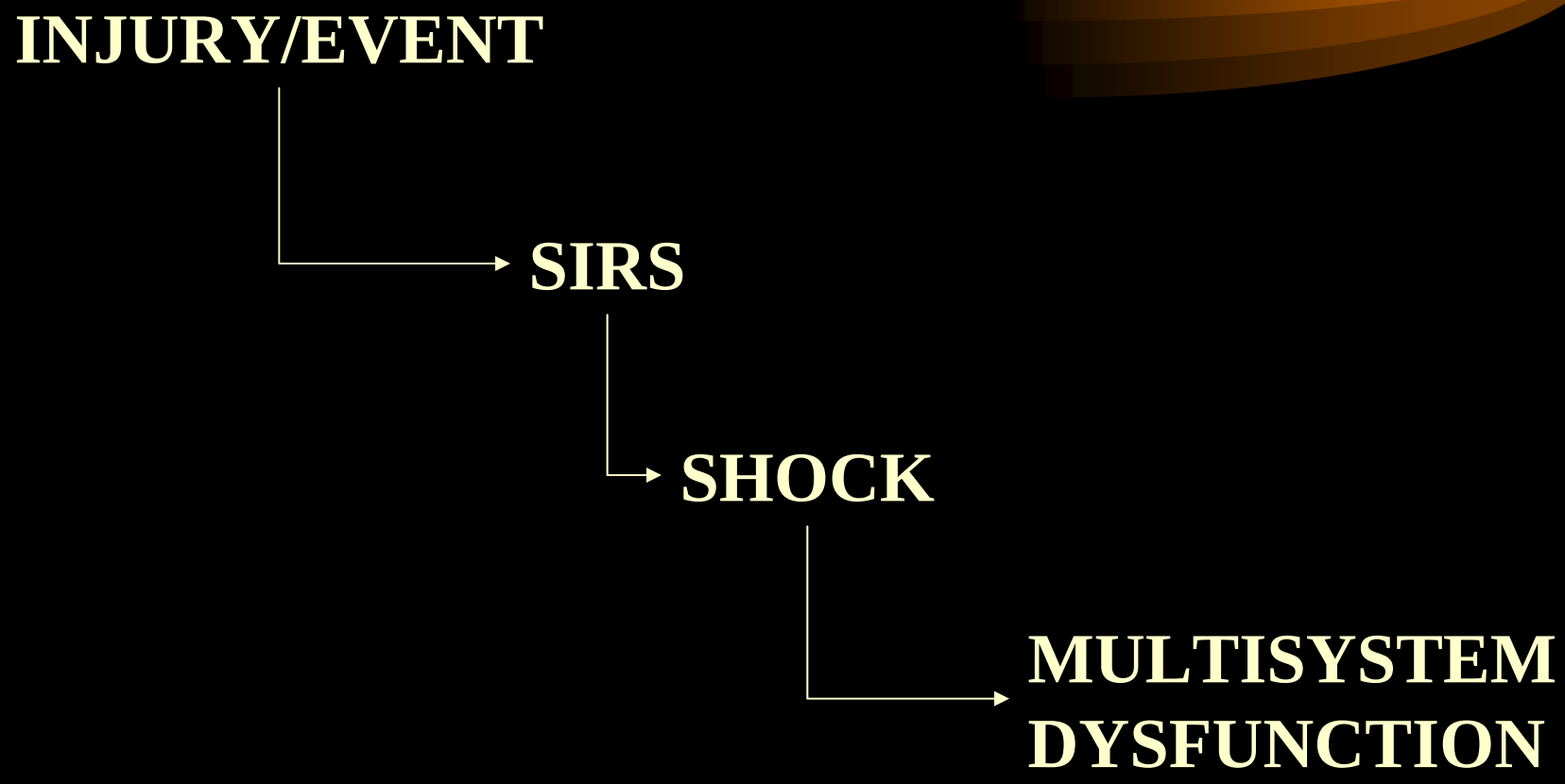
Shock - SIRS Continuum

- **Shock represents one extreme of a continuum of SYSTEMIC INFLAMMATORY RESPONSE SYNDROME (SIRS)**
- **SIRS characterized by (any 2):**
  - **Fever or hypothermia**
  - **Pulse > 90/ min**
  - **Tachypnea (> 20/min or PaCO2 < 32 torr**
  - **Leukocytosis (> 12K), Relative Leukopenia (<4K), or > 10% immature forms**

Shock - SIRS Continuum



Shock - SIRS Continuum



MEDIATORS OF INJURY

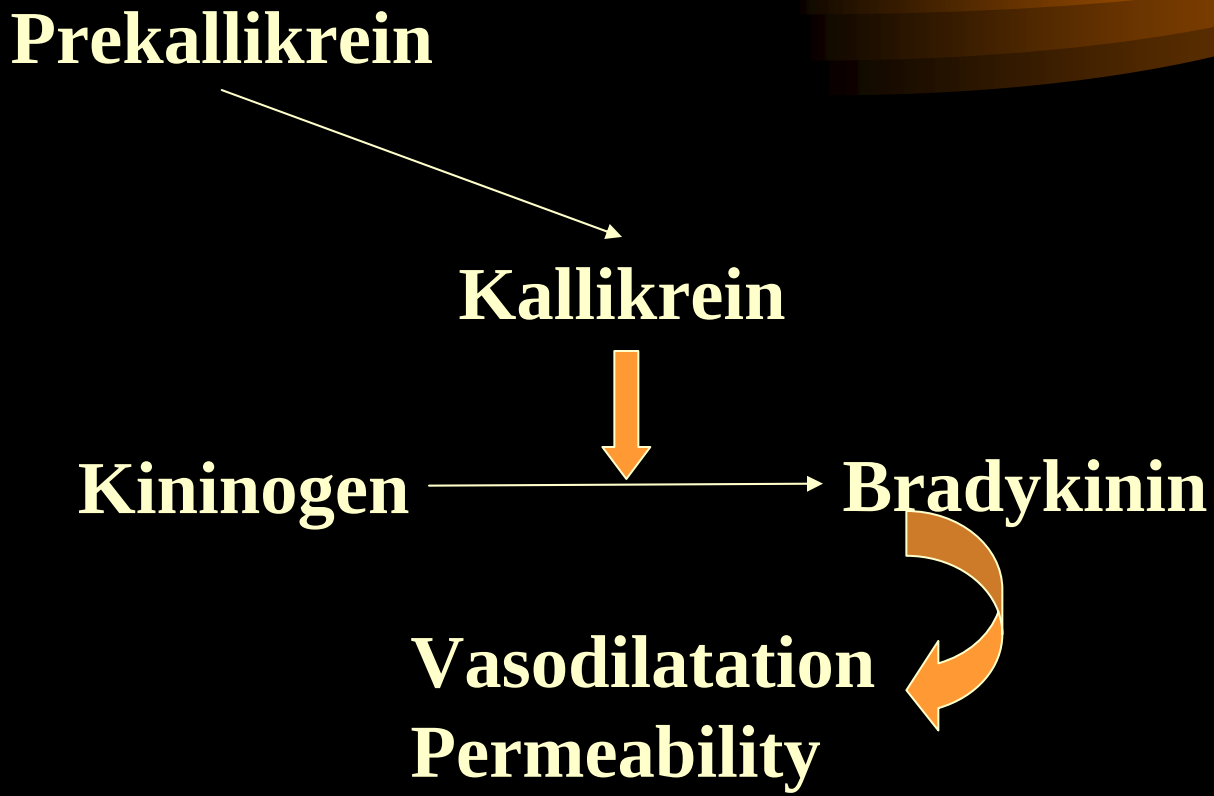
- **Complement/Leukocytes/Superoxides**
- **Kallikrein-Kinin**
- **Prostaglandins/Leukotrienes/PAF**
- **Nitric Oxide**
- **Cytokines**

Complement/Leukocytes Superoxides

- **Complement activation by classical pathway (Ag-Ab complexes) or alternative pathway (e.g. lipopolysaccharide)**
- **Complement pathway activates neutrophils**
- **Neutrophils release reactive oxygen species**
  - **Lipid peroxides**
  - **H2O2**
  - **Hydroxyl radicals**

(Goris et al, 1985 and others)

Kallikrein - Kinin



Prostaglandins/Leukotrienes PAF

- **All are elevated in SIRS/SEPSIS and Shock (and with ARDS)**
- **Animal studies with inhibitors are promising**
- **Human data from antagonist treatment not as encouraging (NSAIDS may improve outcome in hypothermic SEPSIS??)**

(Haupt et al, 1991; Bone et al, 1989; Dhainaut et al, 1994; Arons et al, 1999)

Cytokines

- **Cytokines are low MW proteins secreted by immune cells that exhibit autocrine, paracrine, and/or endocrine function**
- **Cytokines will induce hemodynamic effects of shock- Clinical trials with inhibitors with mixed results**
- **Examples of cytokines:**
  - **TNF alpha**
  - **Interleukin (IL-1, IL-6, IL-8)**

(Heard, 1997; Fisher, 1994)

Nitric Oxide (NO)

- **Ubiquitous free radical inorganic gas/mediator**
- **TNF-*a* induces NO synthesis**
- **NO metabolites increase in Shock/SIRS/Sepsis**
- **Albeit blockage of NO pathway improves BP in Shock (Sepsis), relative perfusion may suffer and apropriate neutrophil response may be impaired (issue is multi-modal and complex?)**

(Malawista, 1992 and others)

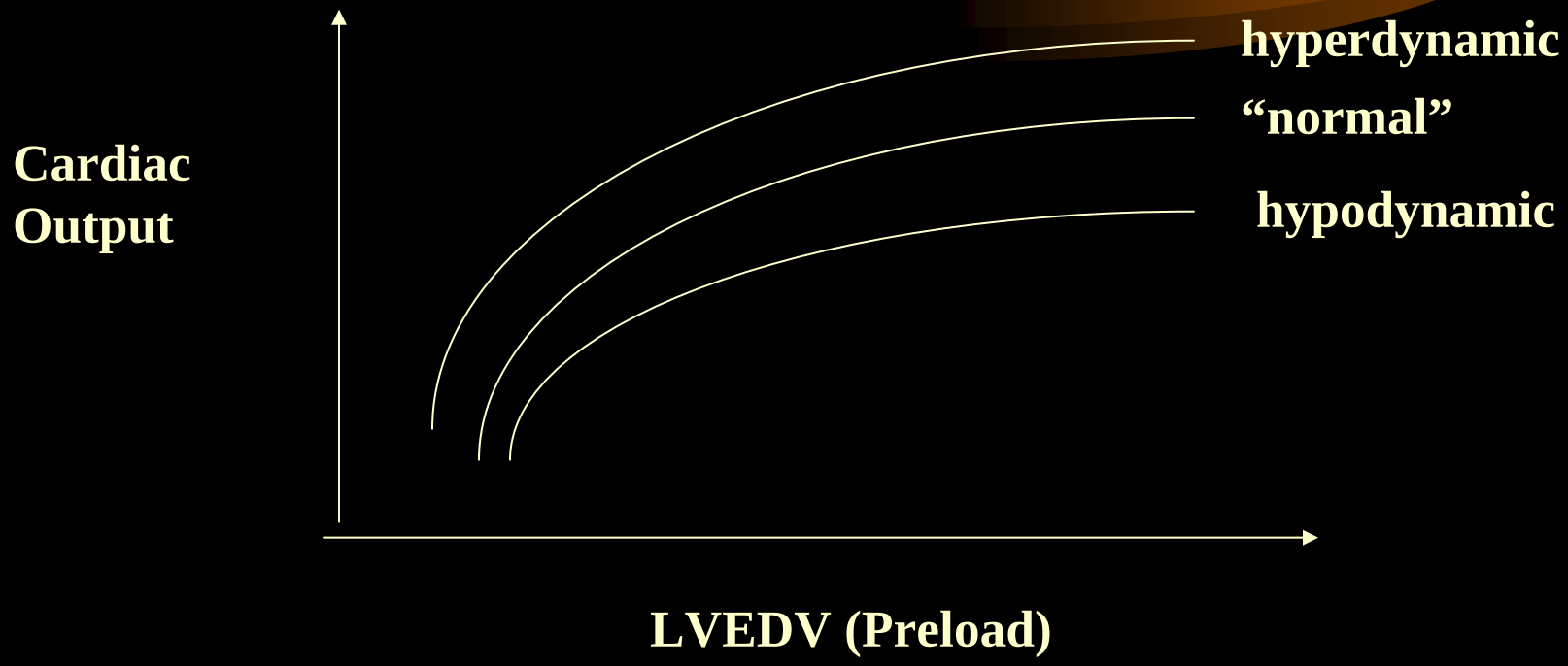
Conclusions- Shock/SIRS (Mediators)

- **Process is a continuum**
- **Cascade of events may be initiated by a variety of factors (with same final common pathway)**
- **Secondary tissue injury and progression of syndrome is due to un-modulated (or mis-modulated) immune response**
- **Mediator treatment promising, but not yet fully developed**

Hemodynamics of Shock

- **Shock can be classified hemodynamically-H**
  - **Hyperdynamic**
  - **Hypodynamic/Cardiogenic**
  - **Hypovolemic (“Normodynamic”)**
- **Hemodynamics may change during the natural progression of a particular etiology of shock**

Hemodynamics of Shock (2)



CO = HR x SV

MAP = CO x TPR

Hemodynamics of Shock (3)

- **Septic shock is initially hyperdynamic (normal filling pressure; enhanced contractility). BP drop is related to decrease in SVR**
- **Hemorrhagic shock is initially normodynamic (diminished filling pressure and CO; normal LV function). BP drop is related to low CO**
- **Late Shock is usually hypodynamic with increased SVR eventually progressing to total systemic collapse**

(Parker and Parillo, 1985; Lee, 1988; Porter, 1997)

Hemodynamics of Shock (4)

- **Since MAP is determined by CO and TPR, hypotension may be present with normal, elevated or decreased contractility (CO)**
- **TPR (SVR) is usually initially increased with hemorrhagic shock**
- **TPR (SVR) is usually decreased in early septic shock**
- **Late(irreversible) shock usually with low CO and increased TPR (SVR) eventually progressing to total systemic collapse as a terminal event**

Hemodynamics of Shock (5)

- **Acute lung injury in conjunction with SIRS or shock may be -**
  - **hydrostatic (elevated pressure)**
  - **oncotic (lowered COP)**
  - **capillary membrane (cell injury)**

Pulmonary edema may be an inevitable consequence of inappropriate *or* appropriate fluid therapy!

Hemodynamics of Shock - Conclusions

- **Hemodynamics may be bimodal or trimodal**
- **Late shock is usually with high SVR and diminished contractility**
- **Low filling pressures (low effective perfusion volume) is an early feature of all shock- the mechanisms are different, however**

OB Hemorrhagic Shock

- **Hemorrhagic = Hypovolemic**
- **Leading cause of Obstetric death**
- **Significant cause of morbidity during pregnancy and immediately postpartum**
- **May be poorly recognized due to physiologic changes of pregnancy**

(Berg, 1996; Clark, 1997)

Postpartum Hemorrhage

Traditional definition = > 500 ml blood loss

Normally seen blood losses:

- **Vaginal delivery - 50% > 500ml**
- **C/Section- 1000ml**
- **Elective C-hys - 1500ml**
- **Emergent C-hys - 3000ml**

Postpartum Hemorrhage (2)

Pregnancy is normally a state of hypervolemia and increased RBC mass
Blood volume normally increased by 30%-60% (1-2 L)
Pregnant patients are therefore able to tolerate some degree of blood loss
***Estimated* blood loss is usually about 1/2 of actual loss!**

Common Causes of OB Hemorrhage

- **Antepartum**
  - **Abruptio Placenta**
  - **Trauma**
  - **Placenta Previa**
- **Postpartum**
  - **Retained Placenta**
  - **Uterine Atony**
  - **Uterine Rupture**
  - **Lacerations**
  - **Coagulopathy**

Categorization of Acute Hemorrhage

	Class 1	**Class 2**	**Class 3**
Blood loss (% blood volume)	15%	15%-30%	30%-40%
Pulse rate	<100	>100	>120
Pulse pressure	Normal	Decreased	Decreased
Blood Pressure	Normal or increased	Decreased	Decreased

OB Hemorrhage - Treatment

- **First step in treatment is recognition**
- **Pregnant patients may have modified or attenuated response to moderate blood loss**
- **Blood loss may not be noted at vaginal delivery due to distraction**
- **Despite standards to the contrary, nursing staff may be multi-tasked during critical post partum period**

Treatment - Hemorrhagic Shock

- **Recognize and treat underlying condition!**
- **Restore intravascular volume**
  - **Blood**
  - **Volume**
  - **Access**
- **Monitor patient until resuscitation successful**
- **Prevent/manage hypothermia**
- **Treat coagulopathy**

Volume Therapy - Hemorrhagic Shock

- **In addition to volume loss from hemorrhage itself, vascular damage produces pronounced intravascular volume depletion**
- **First choice in treatment is crystalloid (Lactated Ringers or 0.9 NS??)**
- **NO compelling advantage for the use of colloid - outcome not different**
- **Volume = 3:1 - adjusted to clinical response**

Volume Therapy - Hemorrhagic Shock (2)

- **NO improved outcome from use of PA catheters or CVP- if present use them**
- **Restore volume as it was lost**
- **Warm fluids a MUST (OR "Cascade" warmer or (better) trauma infuser)**
- **Endpoints (Positive and Negative)**
  - **improved blood pressure**
  - **improved mental status**
  - **resumption of urine output**
  - **pulmonary edema!!**

Pulmonary Edema - Hemorrhagic Shock

- **May be consequence of appropriate resuscitation (Acute lung injury/ARDS continuum)**
- **Is easier to treat than oliguric ATN, myocardial ischemia or acute brain injury**
- **In resuscitated, warm patient- can be suspected by pulse oximetry changes**

(Van Hook et al, 1997; Van Hook, 1998)

Monitoring

- **Pulse oximetry - not accurate with hypothermia, low cardiac output state, or as indicator of ventilatory respiratory failure**
- **CVP - not generally indicated. If already present may or may not reflect filling pressure**
- **PA-catheter - not generally indicated for primary management. May be useful for evaluation of pulmonary edema or in patient with an additional indication for device**
- **Large-bore peripheral IV's will deliver as much or more volume as central lines do**
- **Consider continuous arterial blood pressure monitoring**
- **What is the patient's pulse?**

Blood Component Therapy - Hemorrhagic Shock

- **Packed RBC generally more available than whole blood**
- **Fresh frozen plasma (FFP) not indicated for volume replacement**
- **FFP not indicated for "prophylactic" transfusion after arbitrary number of packed RBC units**

(NIH consensus, 1985)

Component Therapy - Hemorrhage (2)

- **Thrombocytopenia more apt to be etiologic in massive transfusion bleeding**
- **Each unit donor platelets will raise platelet count 5-10,000/cm^3/M2- (*Easy way in normal size/weight patient = Each unit will raise platelet count by 10,000/cm^3/M2)***
- **Consider platelet transfusion with platelet count less than 50,000/M2**

Component Therapy - Hemorrhage (3)

- **FFP (*Easy Way)***
  - **replaces all clotting factors to degree found in normal unit volume of blood**
- **Cryoprecipitate (*Easy Way)***
  - **“best” choice for hypofibrinogenemia (*easy= each unit raises fibrinogen 10 mg% - “target” level often > 100mg%*)**
  - **used for Factor VIII, VWF, XIII, fibrinectin**

Component Therapy - Hemorrhage (4)

- **Transfusion Goal Hematocrit (HCT):**
  - **ISOVOLEMIA is more important than arbitrary HCT for acute management - may tolerate HCT as low as 18% *if not bleeding***
  - **some data suggest that increased DO2 may improve outcome in hemorrhagic shock - O2 content only marginally increased as HCT rises above 37%-30%**

(Morrison et al, 1993; Shoemaker et al, 1987, Cunningham et al, 1997)

Adjunct Therapies - Hemorrhagic Shock

- **Vasopressors - Not useful as *ab initio* therapy**
  - **use for "rescue" treatment**
  - **will diminish tissue perfusion**
- **Renal Protective Therapy (0.5-2*u*g/kg/min Dopamine) - questionably beneficial**
- **Inotropes (Oxygen delivery augmentation) - may be helpful after initial resuscitation based upon experience in trauma**

Oxygen Delivery (DO2)

DO2= O2 Content X Cardiac Output

(Goal = > 650 mL/min/M2)

Content increased by:

a. Hematocrit
b. O2 saturation

Output increased by:

a. Inotropic agents
b. Volume tx.

(Shoemaker, 1987; Clark et al, 1997 and others)

Septic Shock

- **SIRS (defined earlier) associated with documented infection is termed *SEPSIS***
- ***SEVERE SEPSIS* indicates the presence of organ dysfunction, hypoperfusion, and/or hypotension**
- ***SEPTIC SHOCK* consists of severe sepsis refractory to volume resuscitation**
- ***MULTISYSTEM DYSFUNCTION SYNDROME (MODS)* is the terminal phase of this sequence of events**

(Bone et al, 1992; Porter, 1997)

Septic Shock - Background

- **Progression from bacteremia into septic shock is poorly predictable**
- **Exaggerated inflammatory response predicts poorer outcome (APACHE II)**
- **Inflammatory mediators may mimic syndrome**

(Bone 1991; Bone, 1992; Rangel-Frausto, 1995)

Septic Shock - Obstetrics

- **Septic Shock uncommon in Obstetric patients**
- **Bacteremia rate (with infection) is approx. 8%-10%**
- **Up to 12% incidence of septic shock with bacteremia**

(Blanco, 1981; Duff, 1984; Balk, 1989; Porter, 1997)

Septic Shock - Obstetrics

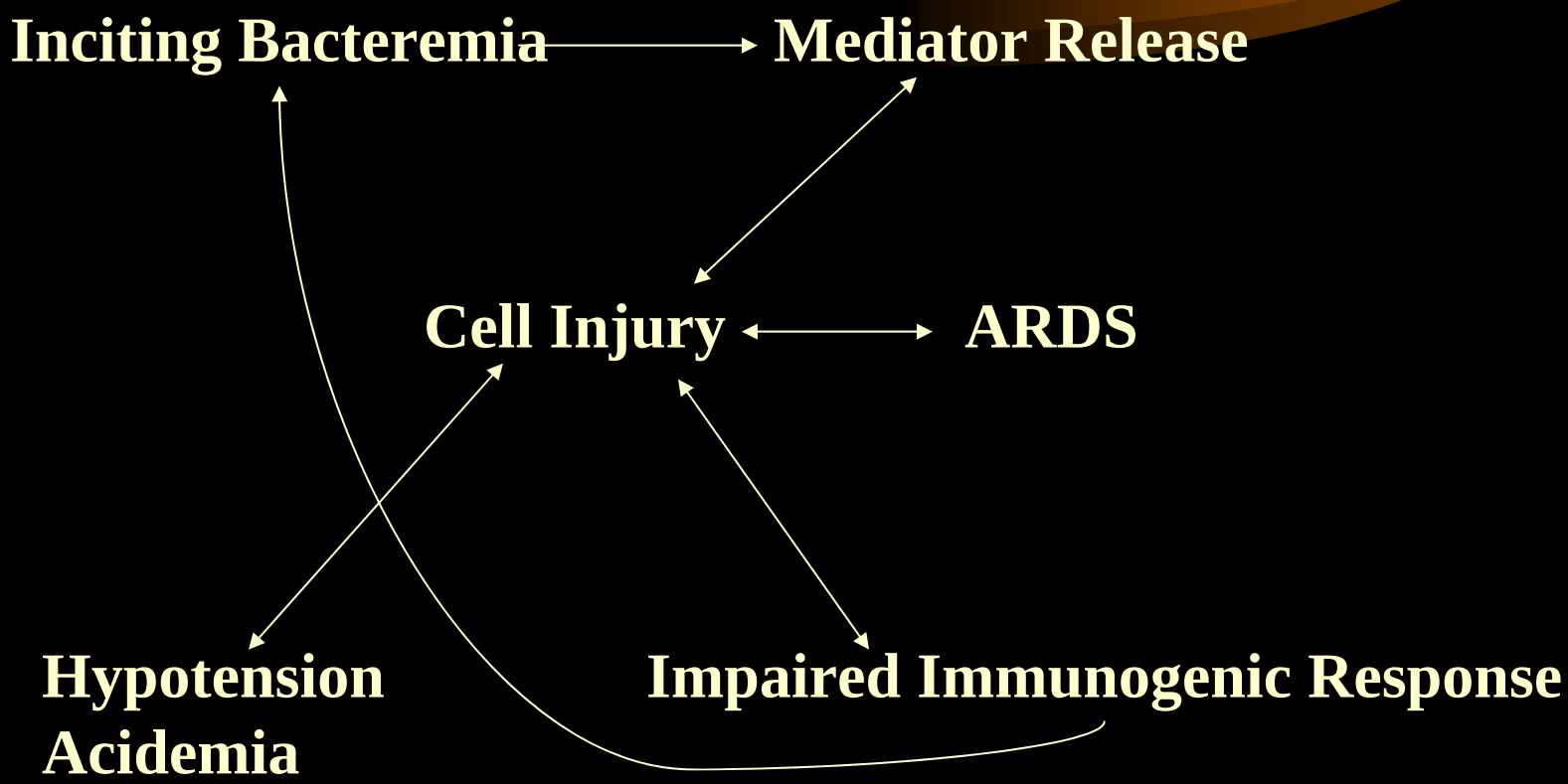
- **Infection Type:**
  - **Post C-section endomyometritis (0.5%-85%)**
  - **post vaginal delivery endomyometritis (< 10%)**
  - **UTI/Pyelonephritis (2%-4%)**
  - **Septic Abortion (2%)**
  - **Necrotizing Fasciitis (< 1%)**
  - **Toxic Shock Syndrome (< 1%)**

(Data as modified from Porter, 1997)

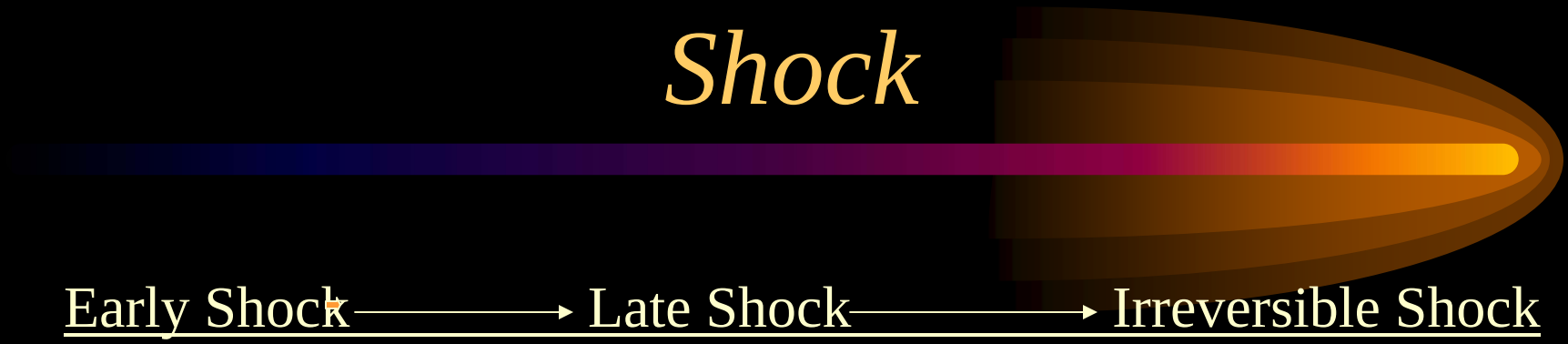
Septic Shock - Pathophysiology

- **(As delineated earlier) mechanism entails mediator release as response to inciting event**
- **Secondary tissue injury, if unabated, incites pathophysiologic cascade**
- **Originally described in response to G-negative organisms (can occur with all organisms and not in relationship to infections at all. EXAMPLE - Hemorrhagic shock)**

Septic Shock Cascade



Clinical Progression of Septic Shock



Early Shock → Late Shock → Irreversible Shock

Hypotension
Low SVR
Tachycardia
Elevated CO
Febrile
PAWP low

Hypotension
Cyanosis
Oliguria
Acidemia
Acute Lung Injury
PAWP ±
CO decreased
SVR variable

Obtundation
ARDS
Anuria/azotemia
Acidemia
DIC
MSDS
CO decreased
SVR low
PAWP high

Septic Shock - Continued

- **(Once again) - shock is a systemic disease!**
- **Myocardial dysfunction is a progressive feature of septic shock-**
  - **CO is initially increased (*but not enough to meet hypermetabolic demands)***
  - **Direct myocardial depression occurs as a late and progressive finding**
  - **(Initial) low cardiac filling pressure aggravates inadequate CO response**
- **Oxygen debt becomes the predominant hemodynamic feature of progressive shock**

(Porembka, 1993; Parrillo, 1985; Lee, 1988)

Treatment of Septic Shock

- **Antibiotics**
- **Volume**
- **Vasopressors**
- **Inotrope**
- **Mediator Therapy**
- **Corticosteroids**
- **Surgical**

Antibiotic Treatment

- **Specific recommendations beyond scope of this talk**
- **OB/GYN infections usually should be empirically treated by broad spectrum therapy**
- **Once patient with full blown septic shock, outcome not appreciably improved in era of antibiotics!**

Septic Shock - Treatment

- **Volume Therapy - (see previous slides)**
- **Vasopressors - (as with hemorrhagic shock) are only useful to "buy time" - may impair tissue perfusion**
- **Mediator Therapy - (previously discussed) presently disappointing (Corticosteroids?; NSAID?)**

Septic Shock Treatment - Inotrope Therapy

- **Augmentation of oxygen delivery (discussed earlier) is not as efficacious in treatment of sepsis-induced shock as it is in the treatment of post-trauma patients**
- **Balance between excess lung water and tissue perfusion often exists (most patients with full-blown shock manifest ARDS)**

(Shoemaker, 1987; NEJM, 1998 and others)

Lung Water vs. Perfusion (Shock)

Improved by:
volume
higher filling
hyperdynamics

PULMONARY EDEMA

Improved by:
diuresis
lower filling pressures
attenuation of hyperdynamics

ORGAN PERFUSION

Corticosteroids - Septic Shock

- **High dose treatment popularized in the 1980's ("attenuate inflammation")**
- **High dose treatment (30 mg/kg methylprednisilone) = DISMAL FAILURE**
- **Recent data - lower dose corticosteroids (300-450 mg/day hydrocortisone may be of benefit in some patients (adrenal "replacement" dosing)**

(Systemic Sepsis Cooperative Study Group, 1987; Crit Care Med, 1999)

"Best Approach" - Septic Shock

- **EARLY RECOGNITION!!**
- **Early Antibiotic Treatment (before cascade progresses)**
- **balance between perfusion and lung injury**
- **preservation of other organ systems (renal, CNS, nutrition)**
- **minimize secondary morbidity (EXPERT HELP)**
- **If able - control febrile morbidity**

Trauma - Related Maternal Adaptations to Pregnancy

Parameter	Change	Implications
Plasma volume	Increases by 45%-50%	Relative maternal resistance to limited blood loss
Red-cell mass	Increases by 30%	Dilutional anemia
Cardiac output	Increases by 30%-50% resistance to limited	Relative maternal blood loss
Uteroplacenta blood flow	20%-30% shunt	Uterine injury may predispose to increased blood loss Increased uterine vascularity

Trauma - Related Maternal Adaptations to Pregnancy

Parameter	Change	Implications
Uterine size	Dramatic increase	Increased incidence of uterine injury with abdominal trauma Change in position of abdominal contents
Minute ventilation	Increases by 25%-30%	Diminished $Paco_2$ Diminished buffering capacity
Functional residual volume	Decreased	Predisposition to atelectasis and hypoxemia
Gastric emptying	Delayed	Predisposition to aspiration