# Shock

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T. B. AND RESPIRATORY DISEASES

# SHOCK

- **1st Part**
- **Defination**
- **Pathophysiology**
- **Stages**
- **Approach to patient with shock**
- **Hypovolaemic shock**

- **2** nd **Part**
- **Cardiogenic shock**
- **Septic shock**
- **Anaphylactic shock**
- **Obstructive shock**

# WHAT IS SHOCK?

**Inadequate Tissue Perfusion**

Shock is a physiologic state characterized by a systemic impairment in oxygen delivery as a result of reduced tissue perfusion, almost universally mediated by low blood pressure.

# Pathophysiology of Shock

- A mean arterial pressure (MAP) of 80 to 120 mmHg is needed for cells to receive the oxygen and nutrients needed to metabolize energy in amounts sufficient to sustain life.
- The body has compensatory mechanisms to assist in maintaining this MAP in response to changes in volume, pumping ability of the heart and changes in the vascular system.
- As long as these mechanisms are effective, the body can survive the changes. When these mechanisms fail, tissues are inadequately perfused and shock begins

# Compensatory Mechanisms

- **Baroreceptors (pressure receptors)** - located in the carotid sinus and aortic arch.
- Decrease in MAP causes decreased stretching of the baroreceptors (they lose their inhibitory effect on the vasomotor center)
  - Sympathetic efferent activity is stimulated. Brain sends impulse to the adrenal glands to release catecholamines (epinephrine and norepinephrine)
  - Catecholamines cause an increase in heart rate and vasocontriction
  - Parasympathetic activity is decreased at the same time

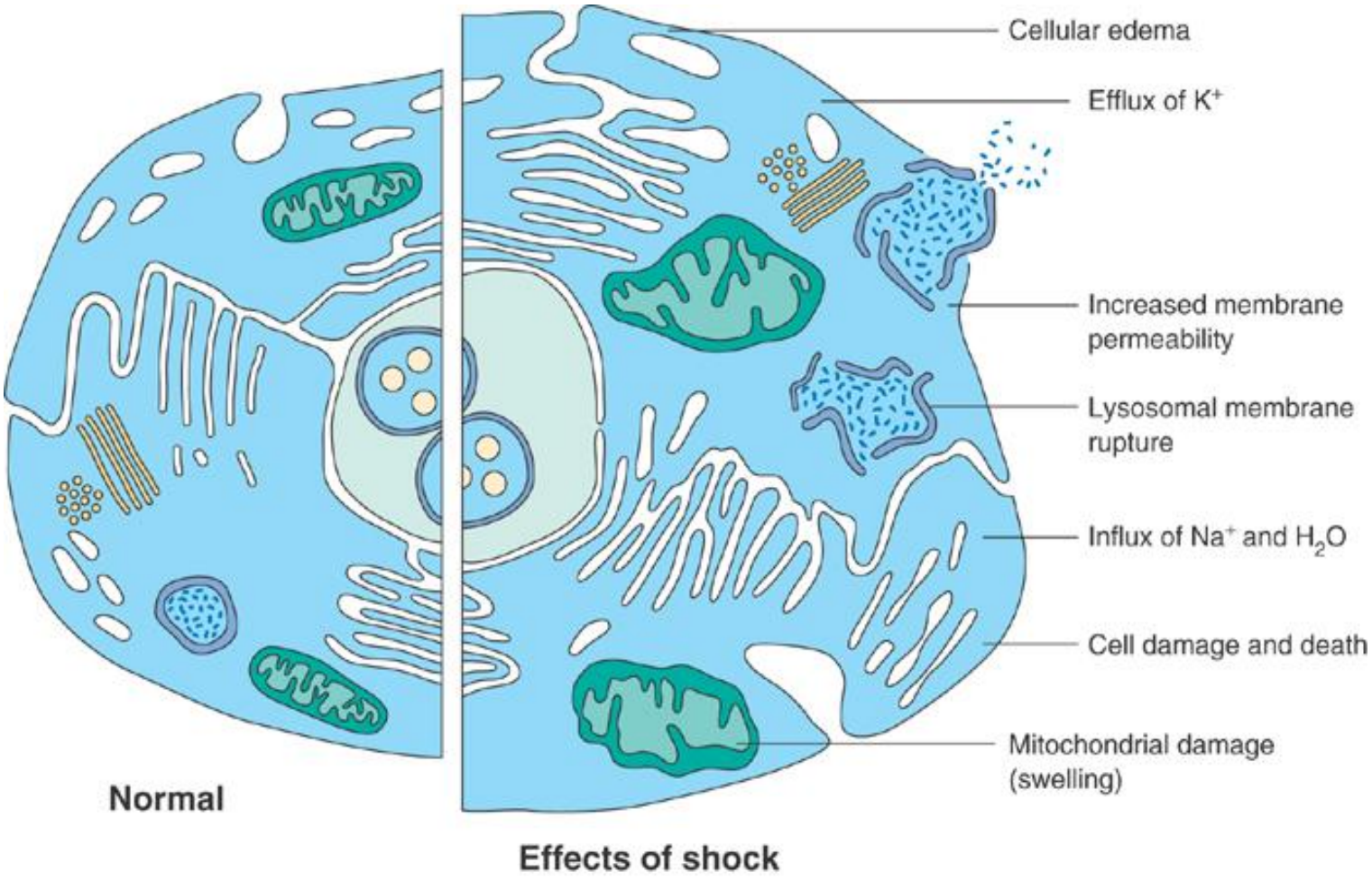
- **Chemoreceptors** - located in the aortic arch and carotid arteries
  - receptive to oxygen changes in the blood
  - regulates blood pressure and heart rate
- **Kidneys** - release renin which leads to the conversion of angiotensin I to angiotensin II, a potent vasoconstrictor
  - leads to release of aldosterone from the adrenal cortex which results in retention of sodium and water
  - increase in sodium triggers release of ADH (antidiuretic hormone)
  - ADH causes kidneys to retain water to raise blood volume and blood pressure

# Chain of Events

- Hopefully, with the drop in blood pressure and the decrease in oxygenated blood, the baroreceptors and chemoreceptors will be able to compensate.
- If the compensatory mechanisms cannot restore tissue perfusion the syndrome of shock begins
- Diminished tissue perfusion deprives cells of oxygen, nutrients, and therefore energy.
- In presence of adequate oxygen & nutrients cells make ATP (energy) and store it for later use.
- If cells have to do this in an oxygen poor environment, then inefficient anaerobic metabolism results in production of lactic acid.

- The increased acidity causes normal cell functions to cease.
- Cellular dysfunction is reversible at first but leads to organ damage if untreated
- The cell swells, the cell membrane becomes more permeable and fluid and electrolytes seep from and into the cell. Mitochondria and lysosomes are damaged and the cell dies.

# Cellular


Cellular edema
Efflux of $K^+$
Increased membrane permeability
Lysosomal membrane rupture
Influx of $Na^+$ and $H_2O$
Cell damage and death
Mitochondrial damage (swelling)
Normal
Effects of shock

- Platelets and white blood cells clump together and obstruct the microvasculature
- Major organs begin to malfunction as they are deprived of oxygen, as a result of hypoxemia and metabolic acidosis
- Goal is to maintain cerebral and cardiac perfusion(Vasoconstriction of splanchnic, musculoskeletal, and renal blood flow)
- Respiratory failure, renal failure, decreased cerebral perfusion, and disseminated intravascular coagulation (DIC) may also be seen
- The earlier that medical management and nursing interventions can be initiated, the greater the chance of survival for the patient

# Stages of Shock

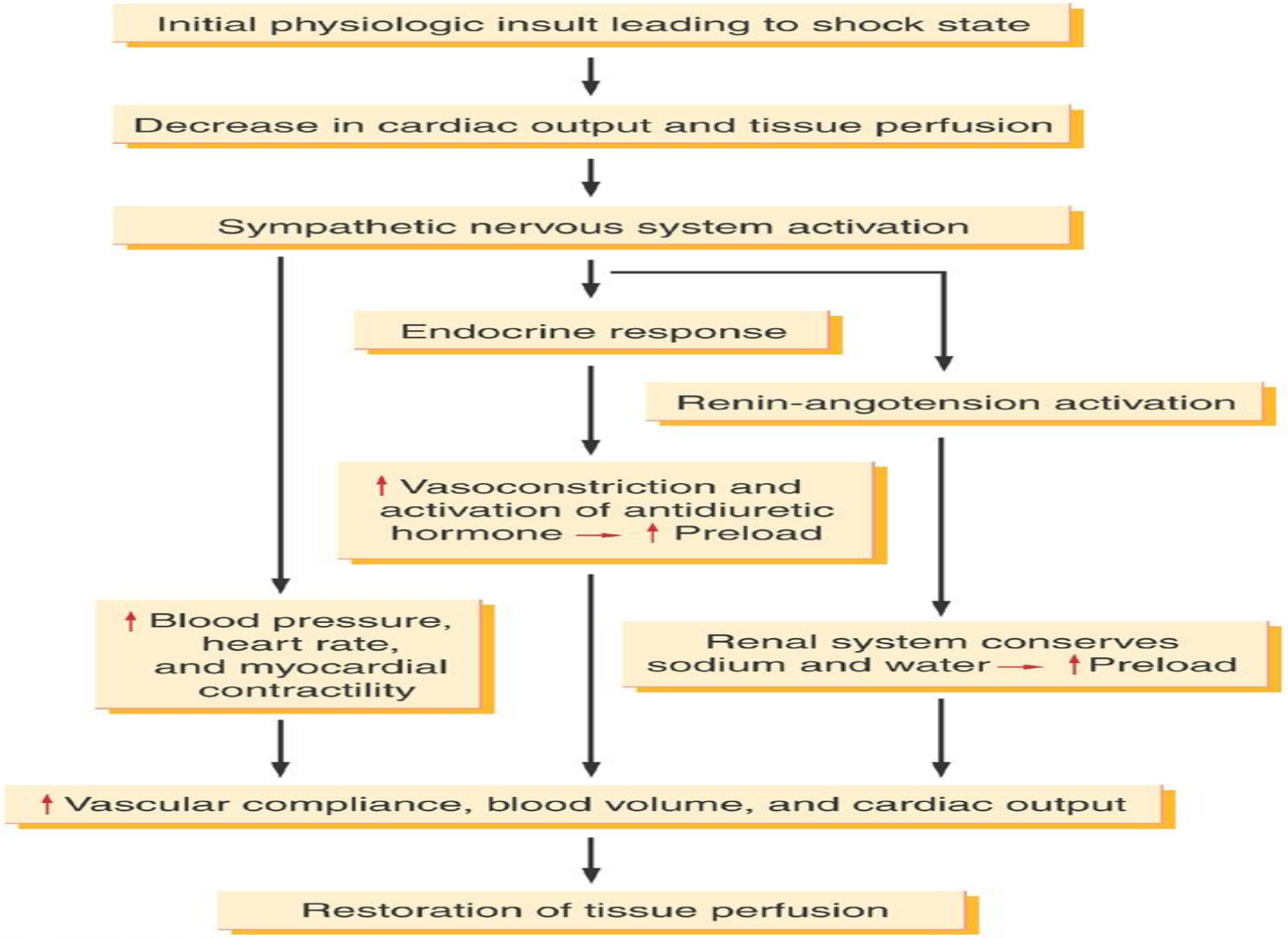
1. Compensatory stage
2. Progressive stage
3. Irreversible stage

# Stages of Shock - Compensatory

**If treated, prognosis is good**

- **the patient's blood pressure remains in normal limits**
- **vasoconstriction, increased heart rate and increased contractility maintain adequate cardiac output**
- **blood is shunted away from "nonessential" organs (skin, lungs, kidneys, GI tract)**
- **Assessment data:**
  - **cold, clammy skin**
  - **hypoactive bowel sounds**
  - **decreased UOP**
  - **confusion, combativeness (result of compensatory respiratory alkalosis)**

# Compensatory Mechanisms



# Progressive Shock

**BP drops (< 80-90 mmHg). Prognosis worsens. Even if the cause of the shock is reversed, the patient may not recover.**

- **Overworked heart becomes ischemic and can result in failure of the pumping ability of the heart**
- **Cellular membrane permeability increases causing cells to "leak" fluid into the interstitial spaces (3rd spacing) and decreasing fluid return to the heart**
- **Organ systems decompensate:**
  - **Lungs - ARDS develops leading to respiratory failure**
  - **Heart - dysrhythmias, HR > 150, chest pain, MI, elevated cardiac enzymes**

- **Brain - level of consciousness deteriorates, pupils may be dilated and sluggish to react to light**
- **Kidneys - acute renal failure can occur. BUN and Cr increase. UOP usually < 20 cc/hr**
- **Liver - less able to metabolize drugs and waste products (ammonia and lactic acid), more prone to infections, SGOT (AST), SGPT (ALT) and LDH are elevated, patient is jaundiced**
- **GI - stress ulcers, GI Bleed, mucosa can become necrotic and slough resulting in bloody diarrhea, toxins are released into the blood stream that cause cardiac depression and vasodilation**
- **Hematologic System - DIC, platelets and clotting factors are consumed, PT/PTT are prolonged**

# Irreversible Stage

**Organ damage is so severe that the patient does not respond to treatment and cannot survive**

- **BP remains low**
- **Complete renal and liver failure, releasing toxins, contributing to an overwhelming metabolic acidosis**
- **Anaerobic metabolism is creating more lactic acid also contributing to metabolic acidosis**
- **ATP reserves are used up**
- **The cells can no longer store ATP related to cell destruction**
- **Patient develops multi-organ failure**

# Signs/Symptoms of Shock

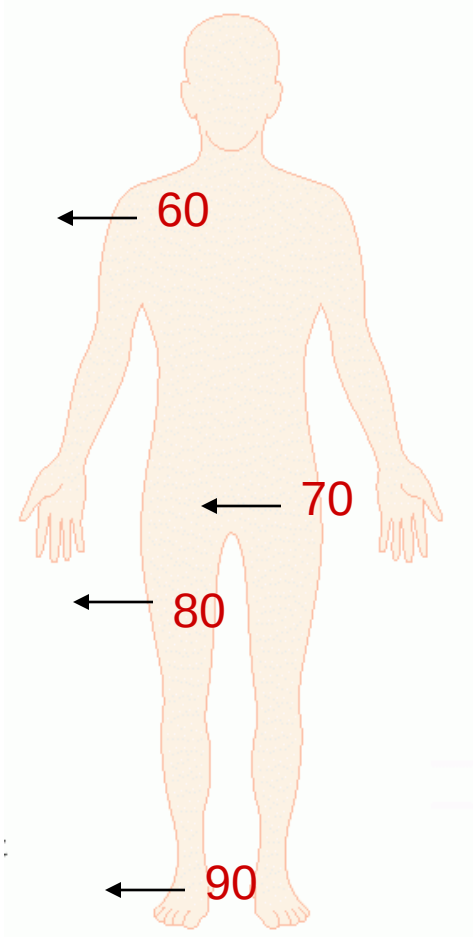
- Cardiovascular – Hypotension
- Nervous – Agitation → Delirium → Coma
- Pulmonary – Tachypnea; hypoxia
- Epidermal – Cool, clammy skin; peripheral cyanosis
- Kidneys – Oliguria; increased BUN/Cr ratio
- GI – Ileus, hemorrhage; hepatic dysfunction
- Hematologic – Coagulopathy → DIC
- Diffuse Cellular Injury – Lactic acidosis

# Diagnosis

- Physical exam (VS, mental status, skin color, temperature, pulse etc)
- Infectious source
- Labs:
  - CBC
  - Lactate
  - Coagulation studies
  - Cultures
  - ABG

# Shock

- Way to quickly estimate blood pressure by pulse
  - If you palpate a pulse, you know SBP is at least this number



# Further Evaluation

- CT of head/sinuses
- Lumbar puncture
- Wound cultures
- Abdominal/pelvic CT or US
- Cortisol level
- Fibrinogen, FDPs, D-dimer

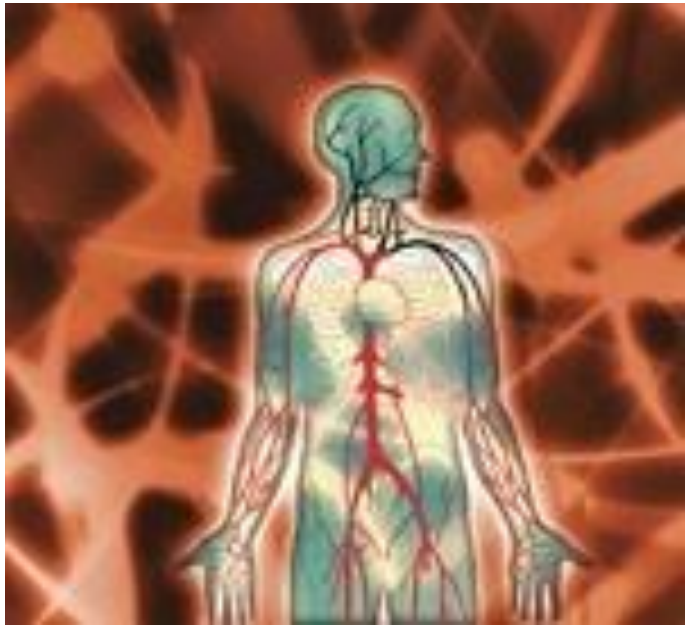
# Approach to the Patient in Shock

- History
  - Recent illness
  - Fever
  - Chest pain, SOB
  - Abdominal pain
  - Comorbidities
  - Medications
  - Toxins/Ingestions
  - Recent hospitalization or surgery
  - Baseline mental status
- Physical examination
  - Vital Signs
  - CNS – mental status
  - Skin – color, temp, rashes, sores
  - CV – JVD, heart sounds
  - Resp – lung sounds, RR, oxygen sat, ABG
  - GI – abd pain, rigidity, guarding, rebound
  - Renal – urine output

# Types of Shock

- Hypovolemic Shock
- Cardiogenic Shock
- Distributive Shock
  - Neurogenic shock
  - Septic shock
  - Anaphylactic shock

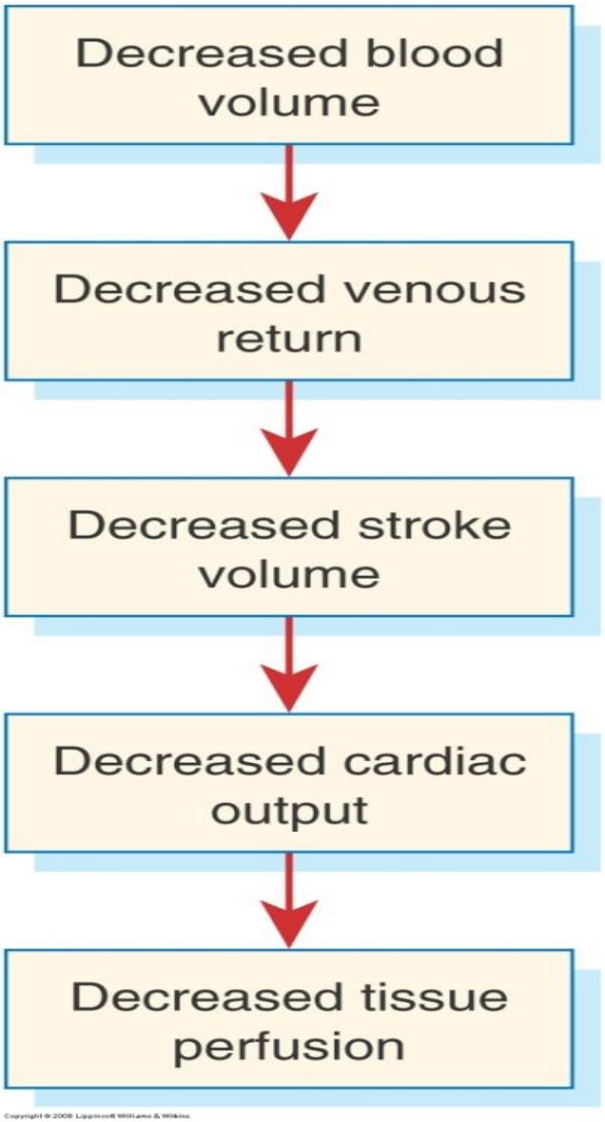
# Hypovolemic Shock



# Hypovolemic Shock

- Non-hemorrhagic
  - Vomiting
  - Diarrhea
  - Bowel obstruction, pancreatitis
  - Burns
  - Neglect, environmental (dehydration)
- Hemorrhagic
  - GI bleed
  - Trauma
  - Massive hemoptysis
  - AAA rupture
  - Ectopic pregnancy, post-partum bleeding

# Hypovolemic Shock



# Presentation of Hypovolemic Shock

- Hypotensive
- flat neck veins
- clear lungs
- cool, cyanotic extremities
- evidence of bleeding?
  - Anticoagulant use
  - trauma, bruising
- Oliguria
- tachycardia
- tachypnoea

# Categorized by severity as class I-II-III

| | Mild | Moderate | Severe |
|---|---|---|---|
| Heart rate | 100/min | 100-140/min | >140/min |
| Blood pressure | Normal or slightly reduced; postural drop present | Decreased;marked postural drop present | Markedly decreased; may be unrecordable |
| Respiratory rate | <20/min | 20-35/min | 35/min |
| Skin | Mild pallor | Increasing pallor,cold,clammy | Severe pallor,sweating, hypothermic |
| Urine output | <30ml/hr | <20ml/hr | <10ml/hr |
| Mental state | Anxious | Incresingly anxious; confused | Obtunded,may be comatosed |
| Arterial pH | Normal | Slightly reduced | ≤ 7.2 |
| Blood loss | ≤ 700ml | 700-1750 | >1750 |
| Fluid replacement | Crystalloids | Crystalloids+blood | Crystalloids+blood |

# Evaluation of Hypovolemic Shock

- CBC
- ABG/lactate
- Electrolytes
- BUN, Creatinine
- Coagulation studies
- Type and cross-match

- As indicated
  - CXR
  - Pelvic x-ray
  - Abd/pelvis CT
  - Chest CT
  - GI endoscopy
  - Bronchoscopy
  - Vascular radiology

# Goals of Treatment

- **ABCDE**
  - **A**irway
  - control work of **B**reathing
  - optimize **C**irculation
  - assure adequate oxygen **D**elivery
  - achieve **E**nd points of resuscitation

* Positioning
  - ☐ the recommended position  for the patient in shock is supine with legs elevated 45 degrees.

# *MANAGEMENT*

1. Fluid Resuscitation
2. Replacement of haemoglobin with blood transfusions
3. Determining the aetiology of hypovolemic shock and treatment of its cause

# *1. FLUID RESUSCITATION*

- PRINCIPLE

The ultimate objective is to restore and maintain oxygen uptake in vital organs of the body so as to preserve organ function.

CANNULATION SITE

In severe shock, it is mandatory to have access to two veins through two large bore (16 guage) intravenous catheters into peripheral veins.

- Central venous catheters donot permit rapid fluid replacement because of
- Long length
- The lumen of the usual central lines is too small to allow rapid fluid flow.

As the maximum infusion rate is dependant on the size(lumen) of the catheter, and not on the size of the vein to be cannulated.

e.g. infusion rate is 4 times greater in the 2” short peripheral catheter then in 12” long central venous catheter.

# *ESTIMATING VOLUME REQUIREMENTS*

ESTIMATE NORMAL VOLUME .In adults normal blood volume is estimated at 66ml/kg for males and 60ml/kg for females. For obese patients ,the blood volume is first estimated on the actual body weight and 10% is then subtrated from the value obtained initially.

## *ESTIMATE THE % OF VOLUME DEPLETED*

VO2 = Cardiac output x Hb x 13 x (SaO2 - SvO2 )

## *CALCULATION OF VOLUME DEFICIT*

This is done by multiplying the estimated normal blood volume and the % lost.this quantifies the volume to be replaced.

# Replacement Fluids

IV replacement fluids can be divided into two categories based on whether they do or do not have a tendency to stay intravascular:

Crystalloid – Normal saline, lactated Ringer's, D5W

Colloid – Fresh frozen plasma, albumin

# Fluid Replacement

**Crystalloids - electrolyte solutions that move freely between intravascular compartment and interstitial spaces**

- **Usually give isotonic solutions - same concentration of electrolytes as the extracellular fluid (avoids wide changes in plasma electrolytes)**
- **Most commonly used are Lactated Ringer's and Normal Saline**
- **Requires large amounts - for every part that remains in the intravascular system, 3 parts move to the interstitial spaces**
- **If a hypertonic solution is used (3% Saline), fluid moves from interstitial spaces to vascular system**

**Colloids - contain molecules too large to pass through capillary membranes.**

- **Pull fluid into intravascular space by means of oncotic pressure (like hypertonic solutions)**
- **Takes less volume and acts longer**
- **Most common used are 5% Albumin, 6% Hetastarch (Hespan) and 6% Dextran solution**
- **Caution must be used with Dextran because it interferes with platelet aggregation**
- **Anaphylactic reactions can occur with colloids**

- Although there are theoretical reasons to favor colloids over crystalloids for volume resuscitation in patients with shock, no data to date has shown any significant outcome difference.

- Therefore, crystalloid is almost always the preferred choice, given its decreased cost and decreased risk.

# RESUSCITATION WITH REFERNCE TO THE FLUIDS USED

- If whole blood is used, then volume to be replaced is 1x volume deficit.
- If colloids are used, then volume to be replaced is 1x volume deficit.
- If crystalloids are used, then volume that needs to be infused is 3x volume deficit.

# *Special conditions in which colloids are used*

i. In severe hypovolemic shock with well marked hypotension, colloids are used for quicker restoration of blood volume, a more rapid rise in cardiac output and blood pressure and a quicker improvement in tissue perfusion is observed.

ii. In fluid restoration for burns patients, intravenous albumin infusions are used after 24 hrs to minimize the formation of oedema which would result from further continued administration of crystalloids.

iii. After volume restoration with crystalloids,colloids may be used in patients who are water logged or who have well marked pitting edema.the increased plasma oncotic pressure mibilizes accumulated extravascular fluid into the vascular compartment,the extra fluid being then excreated via the kidneys.

# Complications of fluid therapy

- **Cardiovascular overload**
- **Pulmonary edema**

**Monitor patient for adequate UOP, changes in mental status, skin perfusion and vital signs.**

**Assess breath sounds frequently during fluid administration**

**Patients may have arterial lines, CVP or Swan-Ganz catheter**

**If CVP being monitored, should be between 4 and 12**

# *REPLACEMENT OF Hb IN SHOCK DUE TO BLOOD LOSS*

Blood compatible for ABO and Rh is given to patients who have lost >20% of blood volume, or those who still continue to bleed. If type specific blood is unavailable ,type O Rh negative blood may be given.

Blood transfusions are ordinarily given to maintain Hb of 10-11g/dl.

# *INDICATIONS FOR BLOOD TRANSFUSIONS IN NORMOVOLAEMIC PATIENTS*

a) VO2 below the normal range
b) Blood lactate levels >4mmol/l
c) Oxygen extraction ratio >0.5

# End Points of Resuscitation

- Goal of resuscitation is to maximize survival and minimize morbidity
- Use objective hemodynamic and physiologic values to guide therapy
- Goal directed approach
  - Urine output > 0.5 mL/kg/hr
  - CVP 8-12 mmHg
  - MAP 65 to 90 mmHg
  - Central venous oxygen concentration > 70%

# Persistent Hypotension

- Inadequate volume resuscitation
- Pneumothorax
- Cardiac tamponade
- Hidden bleeding
- Adrenal insufficiency
- Medication allergy

# Determining the aetiology and its treatment

## Identification of External Bleeding

- Arterial Bleed
  - Bright red
  - Spurting
- Venous Bleed
  - dark red
  - Steady flow
- Capillary Bleed
  - Dark red
  - Oozing

# Control of External Bleeding

- Direct Pressure
  - gloved hand
  - dressing/bandage
- Elevation
- Arterial pressure points

- Upper extremity: Brachial
- Lower extramity: Femoral

- Tourniquets
  - Final resort when all else fails
  - 3-4” wide
  - Do not loosen or remove until definitive care is available
  - Do not cover with sheets, blankets, etc.

# Internal Bleeding

- Can occur due to:
  - Trauma
  - Clotting disorders
  - Rupture of blood vessels
  - Fractures (injury to nearby vessels)
  - GI bleed

- Signs and Symptoms
  - Pain, tenderness, swelling, discoloration at injury site
  - Bleeding from any body orifice
  - Vomiting bright red blood or coffee ground material
  - Dark, tarry stools (melena)
  - Tender, rigid, or distended abdomen

- Management
  - Open airway
  - High concentration oxygen
  - Assist ventilations
  - Control external bleeding
  - Stabilize fractures
  - Transport rapidly to appropriate facility

# *GASTROINTESTINAL BLEED*

- First resuscitate and stablise the pt
- Important diagnostic techniques to detect GI bleed are endoscopy & imaging techniques like arteriography.
- Definitive therapy include surgical intervention,laser photocoagulation,injection of GI varices by sclerosing agents,selective embolisation Of bleeding vessels, or mechanical balloon tamponade of bleeding varices.

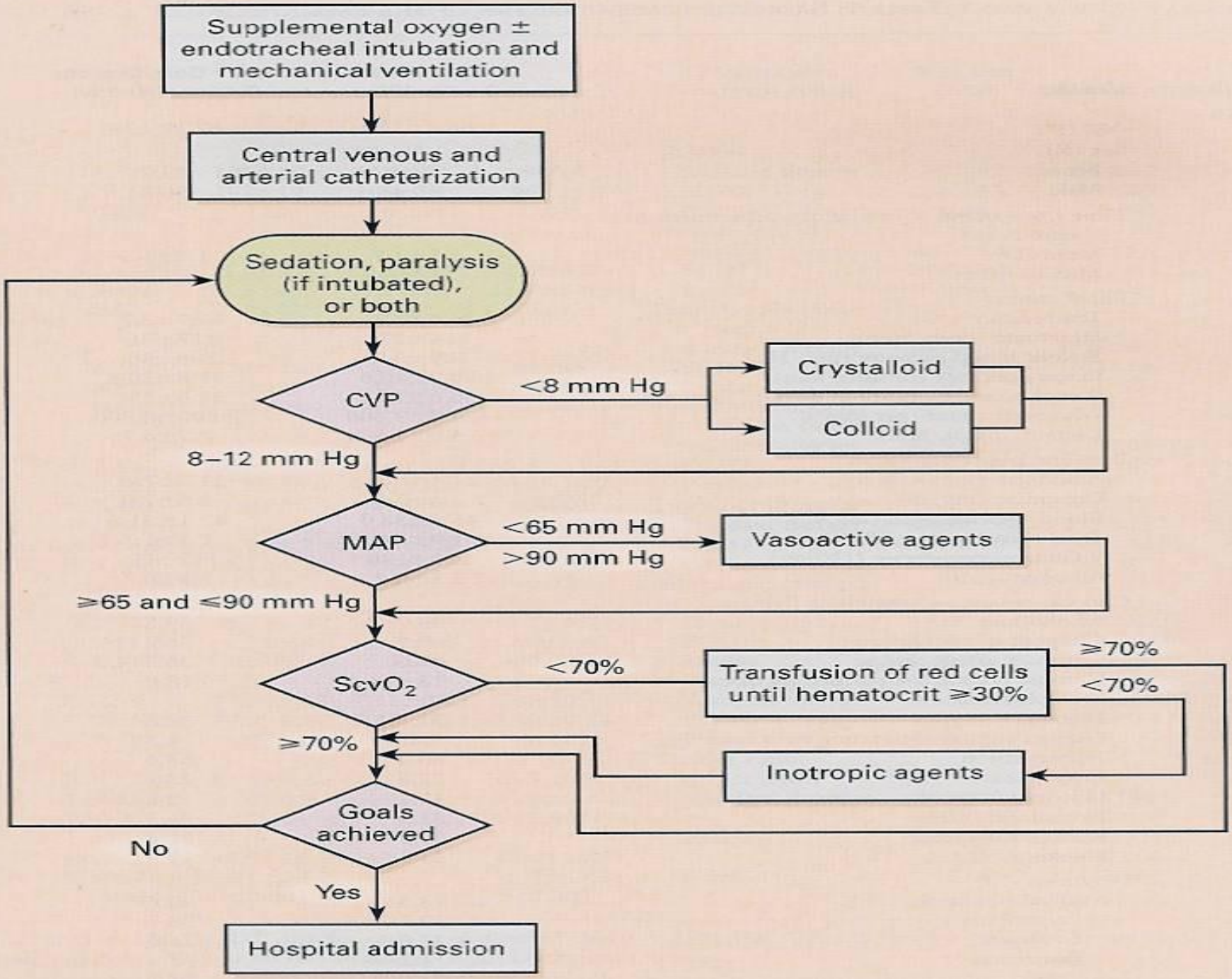
# Other causes of hypovolemic shock:-

- Adrenocortical insufficiency -intravenous hydrocortisone is mandatory and lifesaving in hypovolaemic shock of adrenocortical insufficiency.

- Hypovolaemic shock in diabetic ketoacidosis cannot be fully reversed without the use of insulin.

# Nutritional Support

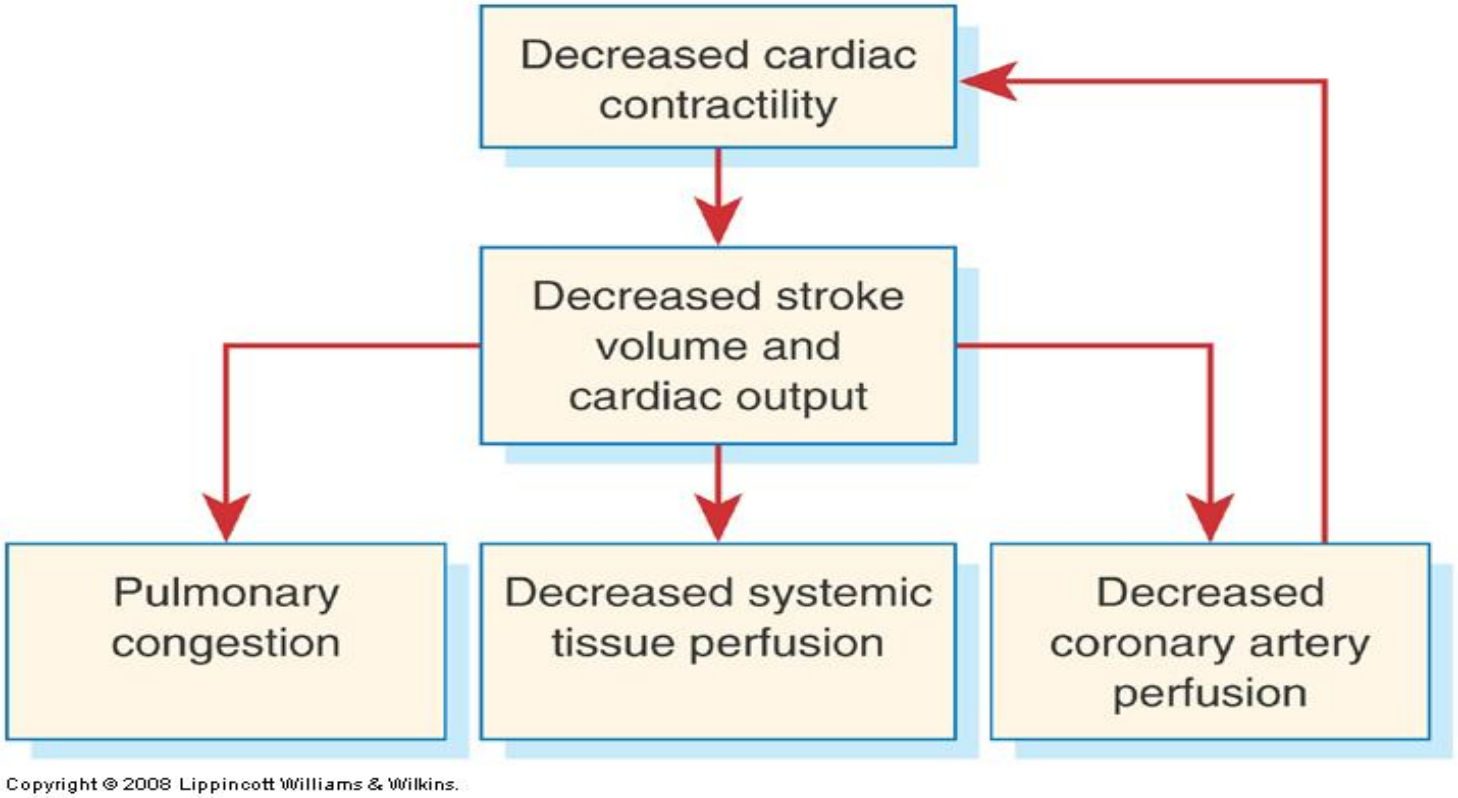
- Patients in shock can require over 3000 calories per day
- Release of catecholamines causes glycogen stores to be used up - can occur in 8-10 hours. This causes skeletal muscle to be broken down for energy.
- Start parenteral or enteral (NGT, J-Tube, Duodenal tube, Dobb-Hoff) within 3-4 days
- Usually will require H2 blockers (cimetidine, ranitidine) to prevent stress ulcers related to decrease perfusion to GI tract


Supplemental oxygen ± endotracheal intubation and mechanical ventilation
Central venous and arterial catheterization
Sedation, paralysis (if intubated), or both
CVP
<8 mm Hg
Crystalloid
Colloid
8–12 mm Hg
MAP
<65 mm Hg
>90 mm Hg
Vasoactive agents
≥65 and ≤90 mm Hg
ScvO2
<70%
Transfusion of red cells until hematocrit ≥30%
≥70%
<70%
≥70%
Inotropic agents
Goals achieved
No
Yes
Hospital admission

Thanks

# Cardiogenic Shock

# Cardiogenic Shock



# Pathophysiology of Cardiogenic Shock

- Often after ischemia, loss of LV function
  - Lose 40% of LV ⟶ clinical shock ensues
- CO reduction = lactic acidosis, hypoxia
- Stroke volume is reduced
  - Tachycardia develops as compensation
  - Ischemia and infarction worsens due to decreased coronary perfusion.

# ETIOLOGY

MI (usually 40% of LV is damaged)

Myocardial ischemia (left main artery disease, multivessel coronary artery disease)

Cardiomyopathy

Arrhythmias

Heart failure

Cardiac tamponade

Acute valvular dysfunction (acute mitral regurgitation, aortic insufficiency)

Papillary muscle rupture

Other severe forms of myocardial injury (trauma)

# Cardiogenic Shock

- Defined as:
  - SBP < 90 mmHg
  - CI < 2.2 L/m/$m^2$
  - PCWP > 18 mmHg
- Signs:
  - Cool, mottled skin
  - Tachypnea
  - Tachycardia
  - Weak pulse
  - Hypotension
  - Narrowed pulse pressure
  - Crepitations, murmur
  - JVD
  - Oliguria
  - Altered mental status

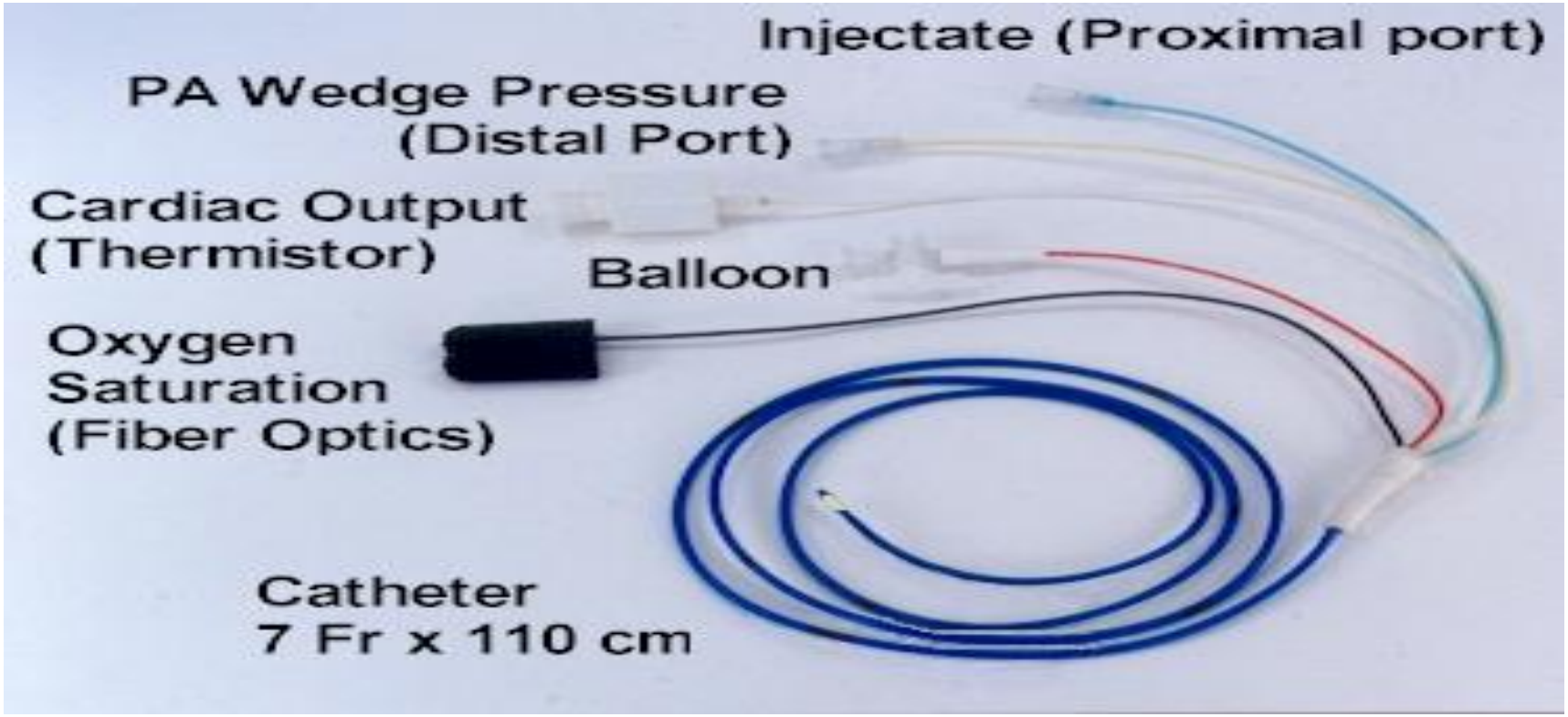
# *MONITORING PATIENTS IN CARDIOGENIC SHOCK*

- Half hourly HR,BP,RR
- Hourly skin& rectal temperature
- Hourly urine output
- ECG
- CXR
- Blood count,PCV,serum electrolytes,blood urea,serum creatinine,arterial ph,ABG , cardiac enzymes, coagulation studies
- Echocardiogram
- Haemodynamic monitoring
- ❖ CVP
- ❖ Pulmonary artery pressure
- ❖ Cardiac output,cardiac index,stroke volume,SVR

# IN ABSENCE OF INVASIVE MONITORING

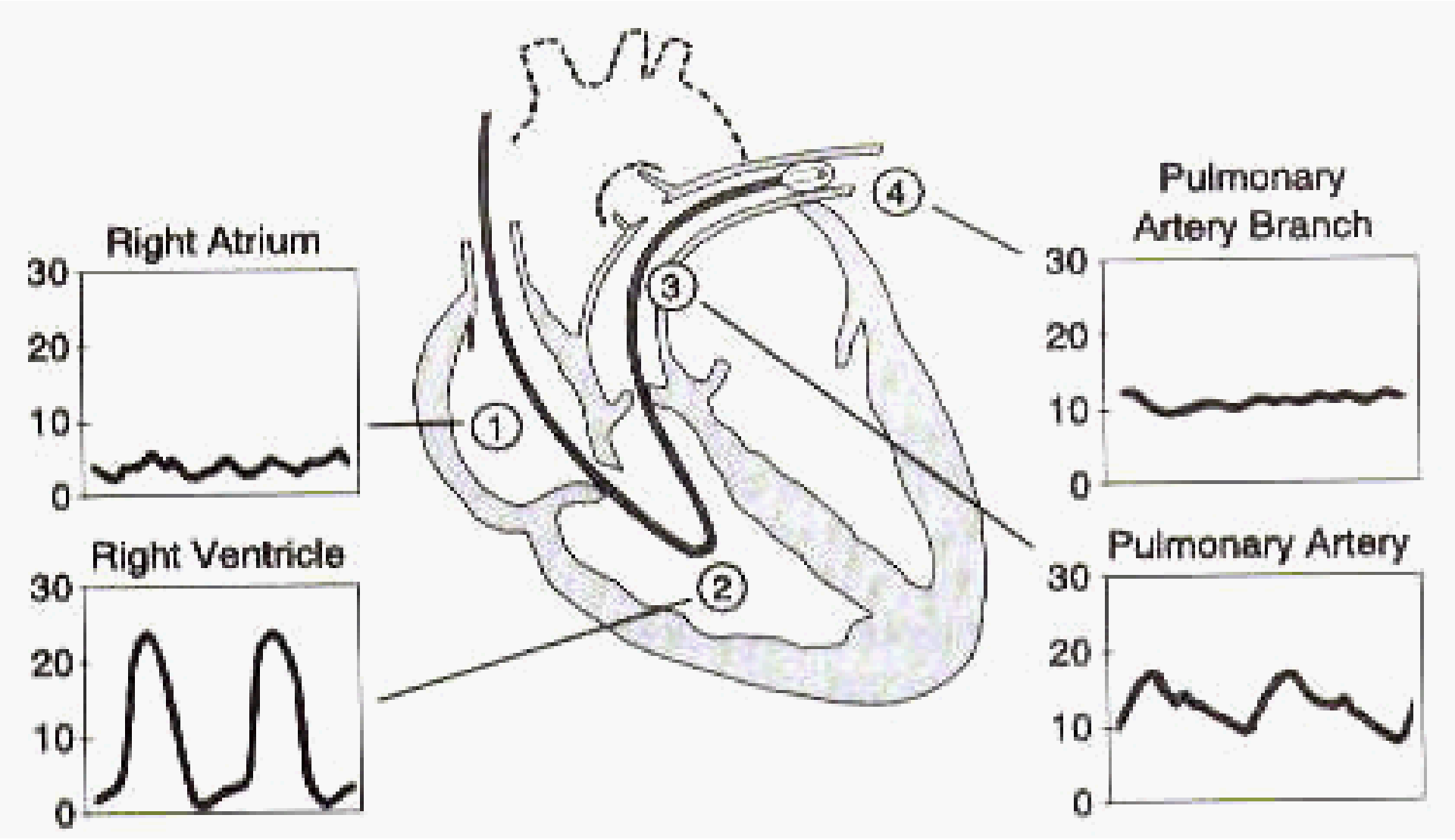
- Measure CVP via central line
- High PCWP evidenced by
  - ✓ CXR showing pulmonary edema
  - ✓ Clinically- crepitations over lung bases,left ventricular diastolic gallop(audible 3rd HS)
- Estimate CO & tissue perfusion clinically
  - ✓ blood pressure
  - ✓ skin temperature
  - ✓ volume of peripheral pulse
  - ✓ hourly urine output
  - ✓ general sensorium,absence/presence of lactic acidosis

# The Pulmonary Artery (PA) Catheter



# The PA Catheter

- As the catheter is “floated” from either the internal jugular or subclavian veins, and advanced from the RA to the RV, and from the RV to the PA, a number of specific pressure waveforms should be observed.


Right Atrium
30
20
10
0
Right Ventricle
30
20
10
0
1
2
3
4
Pulmonary Artery Branch
30
20
10
0
Pulmonary Artery
30
20
10
0

# The PA Catheter

- The PA catheter allows measurement of 3 types of data:
  1. Central venous, pulmonary artery, and pulmonary capillary occlusion (or “wedge”) pressures
  2. Cardiac output and vascular resistence
  3. Sampling of mixed venous blood

- Situations in which PA catheters are most helpful:
  1. Guiding the management of severe CHF
  2. Estimating fluid status in non-cardiogenic pulmonary edema
  3. Diagnosing pulmonary hypertension
  4. Diagnosing right heart infarction

# *MANAGEMENT*

a. Maintain oxygenation,administer oxygen at flow rate of 6-8l/min. If PaO2 still <60mmHg or if respiratory distress(rate >35/min),intubate &put on ventilator support.

b. Maximizing preload: volume challenge if PCWP is <15mmHg.Increase PCWP to 15-20mmHg provided there is no pulmonary edema.

c. Use of vasodilators:Priciple-reduce abnormally high preload to optimal levels(15-18mmHg):reduce increased afterload.

- IV nitroglycerin in patients with high PCWP & well marked pulmonary edema

- IV nitroprusside if marked increase in SVR,PCWP>20mmHg,poor tissue perfusion, systolic BP>90-100mmHg.

d. Increasing cardiac contractility:

- Dobutamine or dopamine by IV infusion or both
- Digoxin

e. IV furosemide if there is pulmonary edema.

f. Correction of electrolytes and acid- base abnormalities

g. Correction of factors aggravating cardiogenic shock.

h. Pain is relieved with morphine 2-4 mg IV,buprenorphine 0.3mg IM or IV or pethidine 25mg IV.

# Vasopressors and Inotropes

- Vasopressors – Act to increase SVR, and subsequently increase BP.
- Inotropes – Act to increase CO.  BP may either be increased or decreased.

Together, vasopressors and inotropes are colloquially known as “pressors”.

# Vasoactive Drugs

- Used when fluid alone cannot maintain adequate MAP
- Drug of choice is selected according to what correction is needed to increase CO:
  - increase contractility
  - cause vasoconstriction
  - regulate the heart rate
- Act on receptors of the sympathetic nervous system
  - Alpha - Vasoconstriction of Cardiorespiratory and GI systems, skin and kidneys
  - Beta1 - increase heart rate and contractility
  - Beta2 - vasodilatation of heart and skeletal muscles, relaxation of bronchioles

- A given drug may have an effect on multiple receptors, and this interaction may be dose dependent.
- Hypovolemia must be corrected prior to the institution of vasopressor therapy. Therefore, pressors are generally not helpful in hypovolemic shock.
- A given agent may affect systemic blood pressure through both direct actions, as well as indirect reflex actions.

- Monitor vital signs q15 min while vasoactive drugs are being used
- Administer through a central line
- Use an IV Pump
- Titrate drip rate according to patient parameters
- Do not stop drips abruptly - wean slowly while monitoring vital signs q15 min

# Vasopressors and Inotropes

| Drug | Alpha-1 | Beta-1 | Beta-2 | Dopa. | Effect on SVR | Effect on HR | Effect on contractility | Typical Dose |
|---|---|---|---|---|---|---|---|---|
| Phenylephrine | +++ | 0 | 0 | 0 | ↑↑ | ↔ / ↑ | ↔ | 20-200 µg/min |
| Vasopressin (mechanism of action poorly understood) | 0 | 0 | 0 | 0 | ↑↑ | ↔ | ↔ | 0.01-0.04 U/min |
| Norepinephrine | +++ | ++ | 0 | 0 | ↑↑ | ↑ | ↑ | 0.5-20 µg/min |
| Epinephrine | +++ | +++ | ++ | 0 | ↓ (low dose)<br>↔ / ↑ (high dose) | ↑ | ↑ | 2-10 µg/min |
| Dopamine 0.5 – 2<br>5 – 10<br>10 – 20<br>(µg/kg/min) | 0<br>+<br>++ | +<br>++<br>++ | 0<br>0<br>0 | ++<br>++<br>++ | ↔<br>↑<br>↑↑ | ↑<br>↑<br>↔ | ↑<br>↑<br>↔ | 1-20 µg/kg/min |
| Dobutamine | 0 / + | +++ | ++ | 0 | ↓ | ↑ | ↔ / ↑ | 2.5-20 µg/kg/min |
| Isoproternol | 0 | +++ | +++ | 0 | ↓ | ↑ | ↑ | 1-10 µg/min |
| Milrinone (acts as a phosphodiest-erase inhibitor) | 0 | 0 | 0 | 0 | ↓ | ↔ | ↑↑ | Load: 50 µg/kg over 10 min<br>Maintenance: 0.375 – 0.75 µg/kg/min |

# Vasopressors and Inotropes
## (Generalized Summary)

Phenylepherine

Norepinepherine

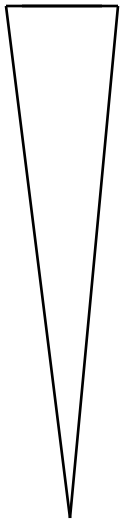
Epinepherine

Dopamine

Dobutamine

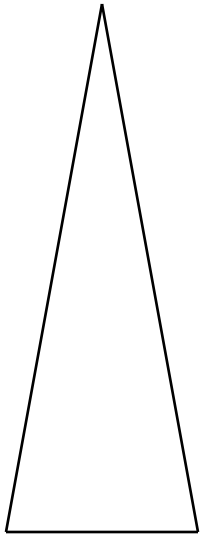
Milrinone

↑ SVR



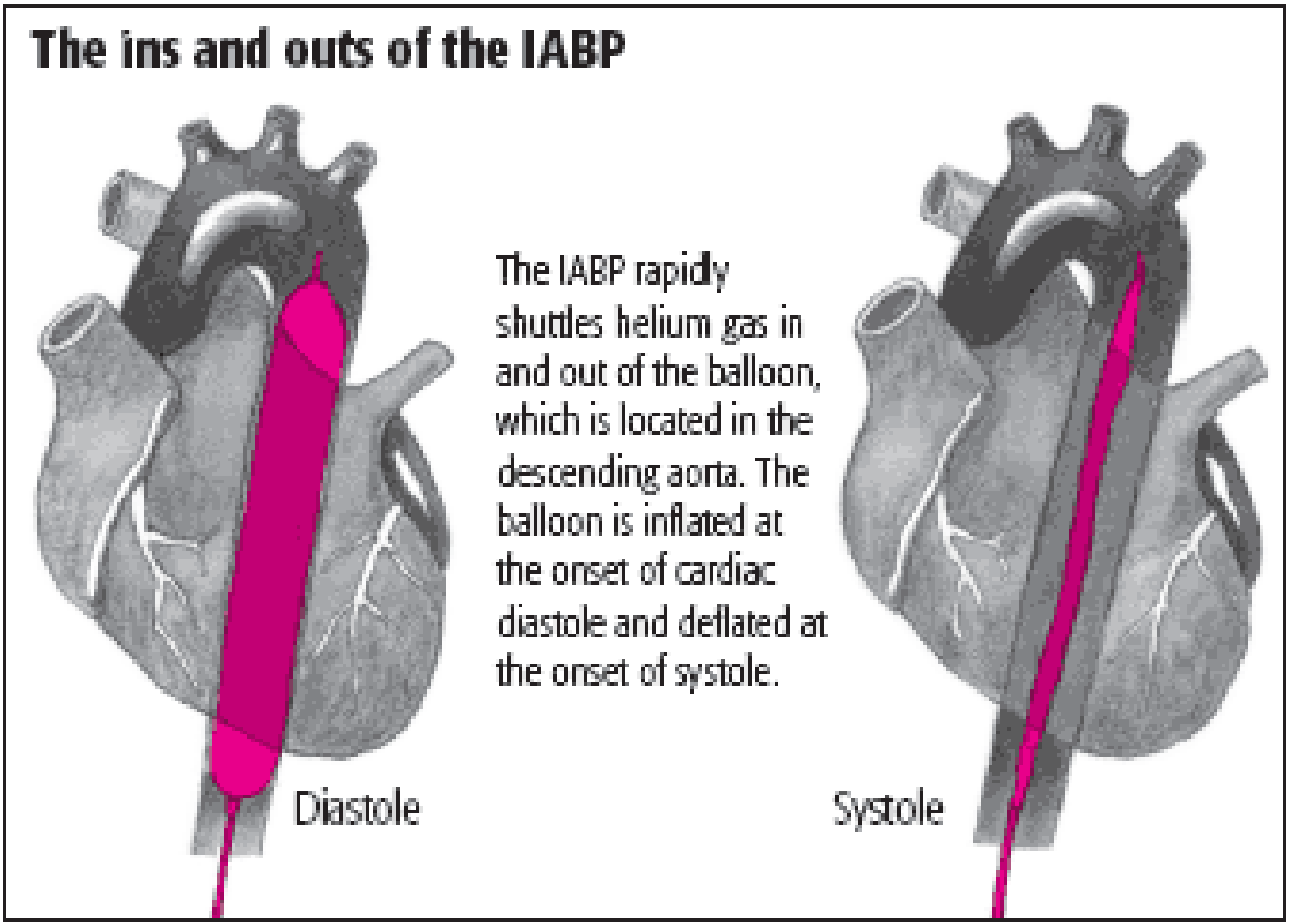
↓ SVR

No effect on contractility

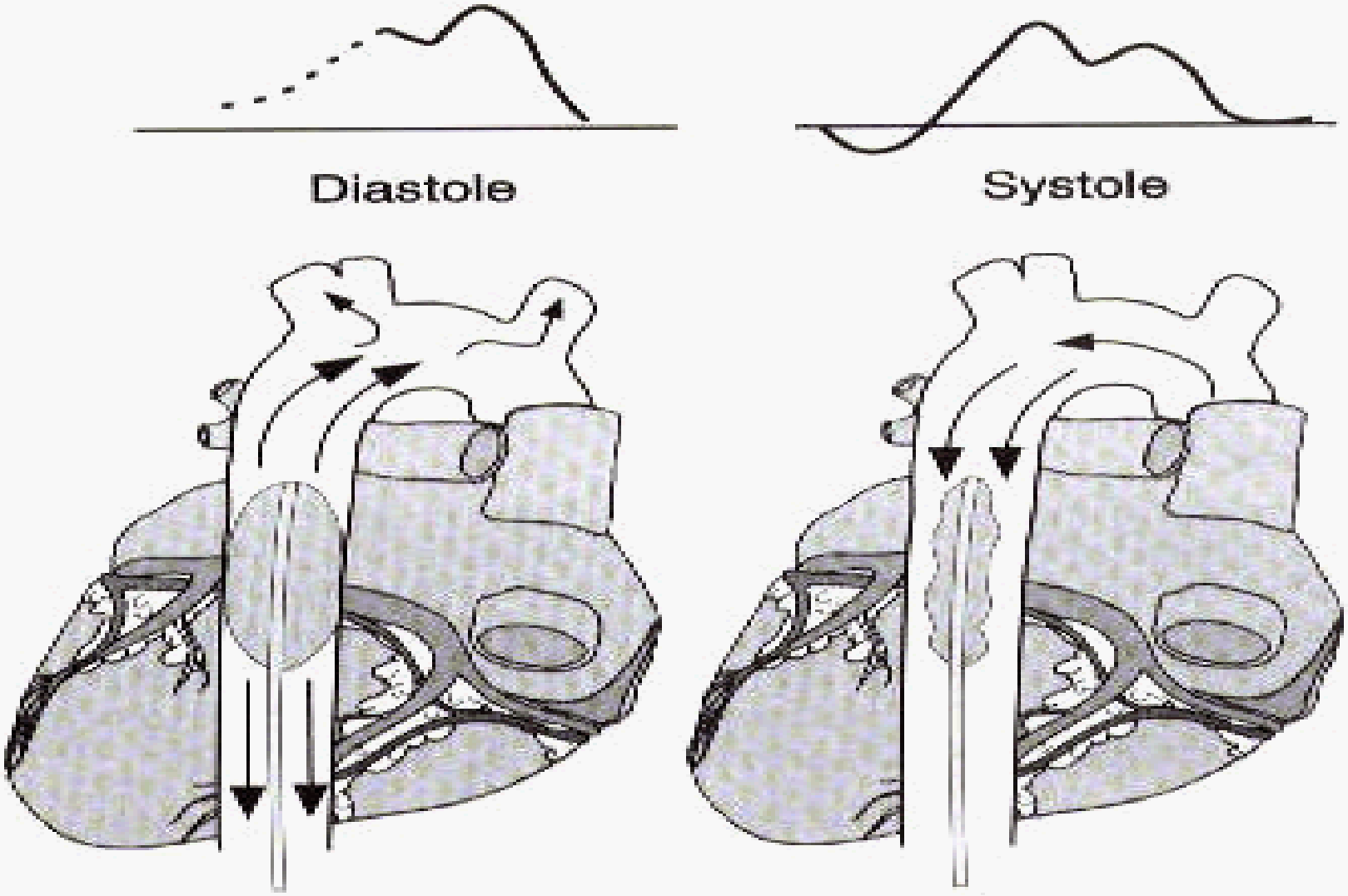


↑ Contractility

# Intra-aortic Balloon Counterpulsation



# Intra-Aortic Balloon Pump



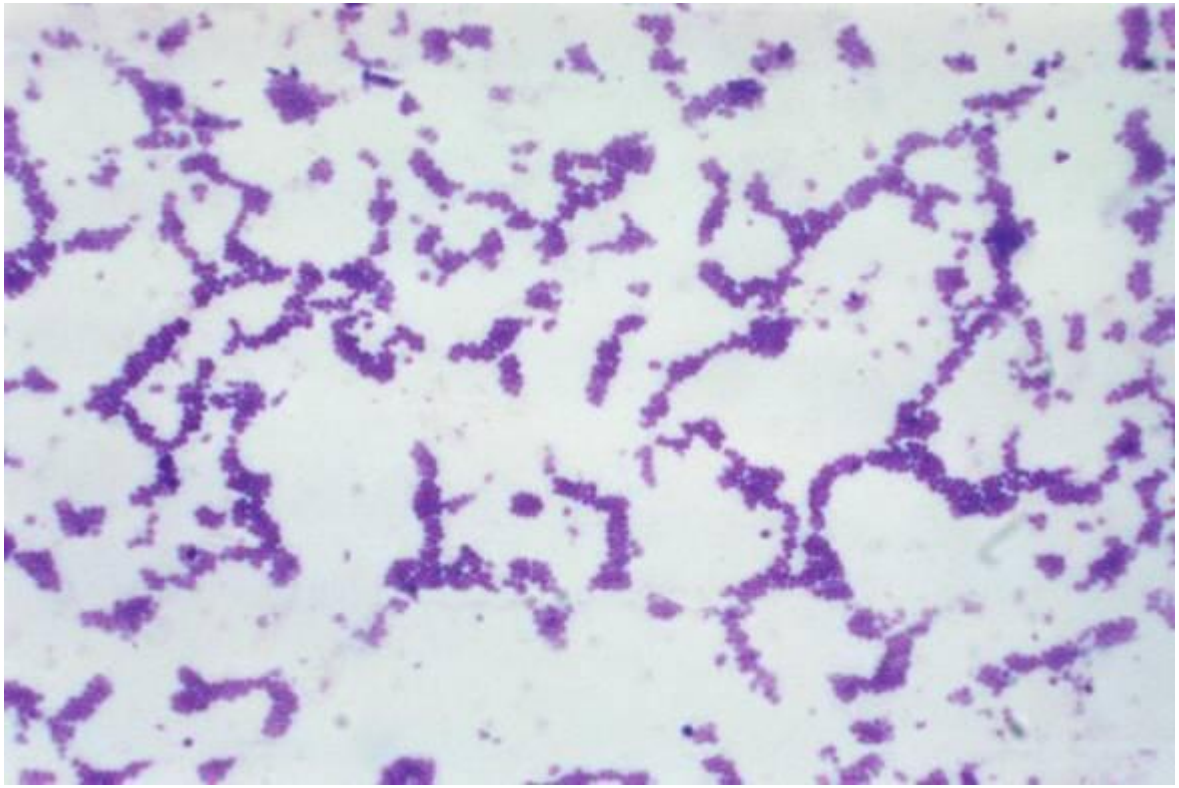
# Intra-aortic Balloon Counterpulsation

- The only thing that reduces afterload and augments diastolic perfusion pressure
- Beneficial effects occur without increase in oxygen demand
- No improvement in blood flow distal to critical coronary stenosis
- No improvement in survival when used alone
- May be essential support mechanism as a bridge to definitive therapy

- By medical measures & by support of the aortic balloon pump patient's haemodynamic profile is improved so that diagnostic intervention through angioplasty or surgery become feasible.
- Recent MI characterized by ST elevation should be promptly thrombolysed provided there is no absolute contraindications.

It is essential to distinguish between hypovolemic and cardiogenic shock because definitive therapy differs significantly. Both forms are associated with a reduced cardiac output and a compensatory sympathetic mediated response characterized by tachycardia and elevated systemic vascular resistance. However, the findings in cardiogenic shock of jugular venous distention, rales, and an S3 gallop distinguish it from hypovolemic shock and signify that ongoing volume expansion is undesirable.

# Septic Shock



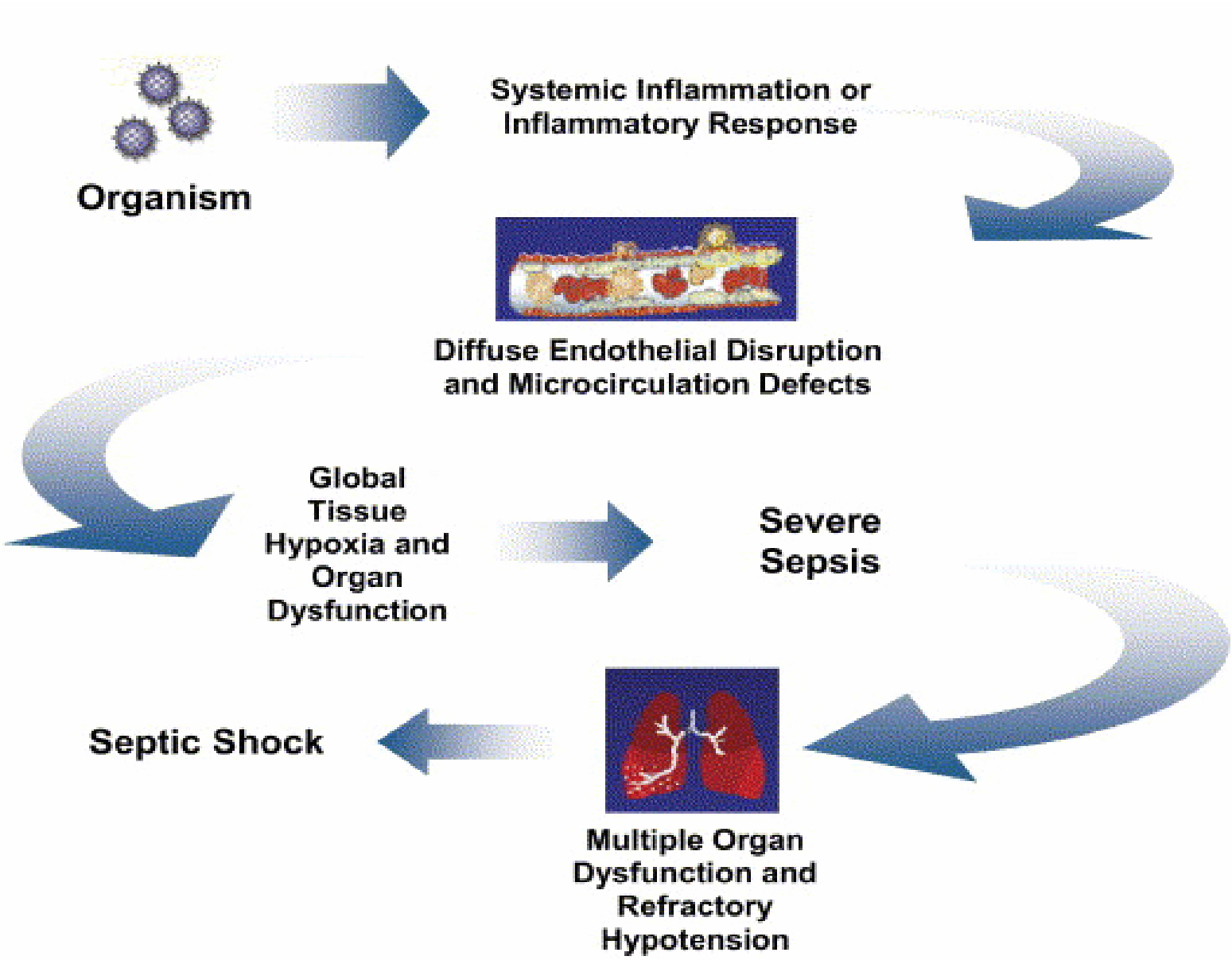
# Sepsis

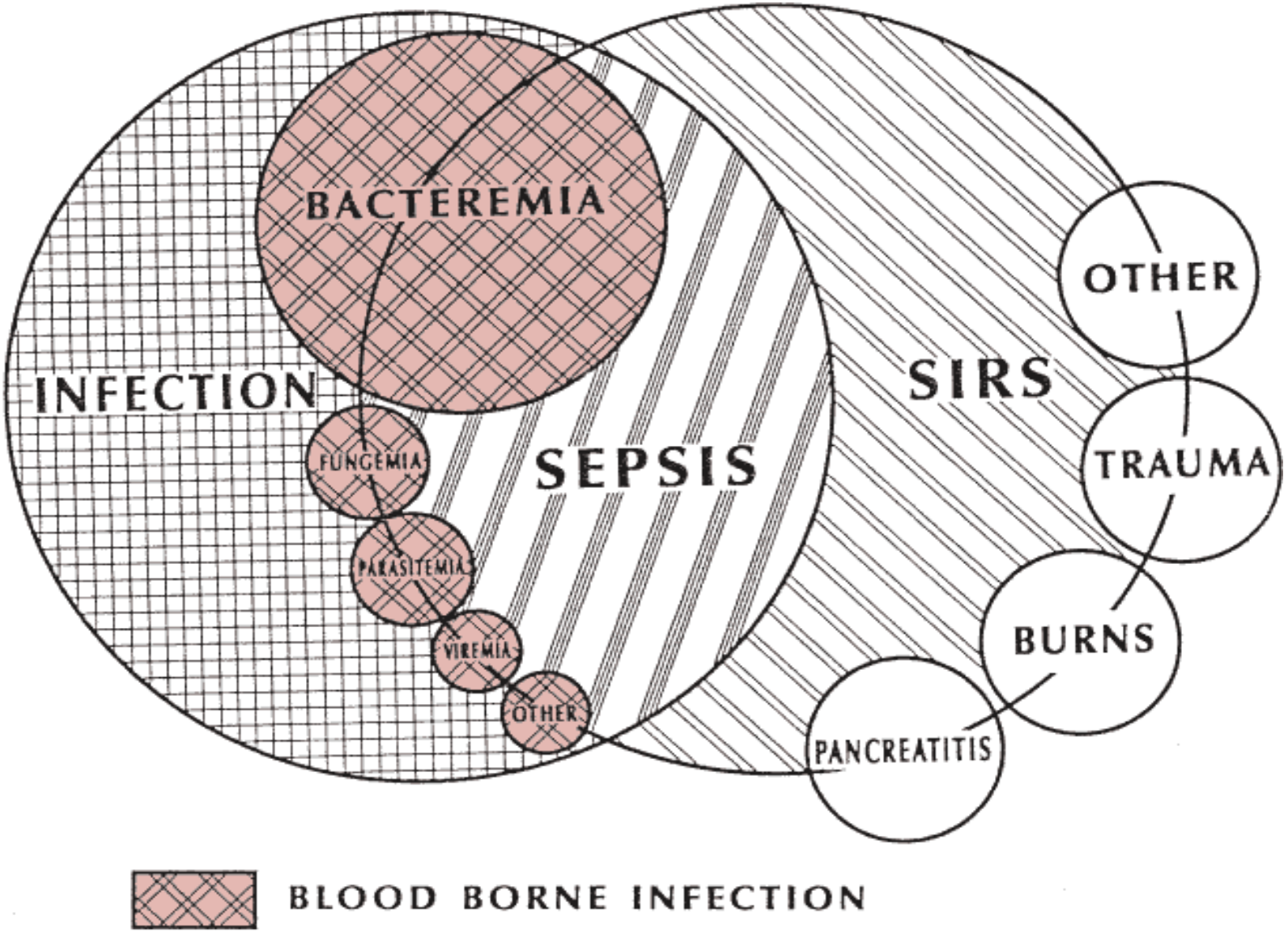
- Two or more of SIRS criteria
  - Temp > 38 or < 36 C
  - HR > 90
  - RR > 20
  - WBC > 12,000 or < 4,000 or >10% band forms
- Plus the presumed existence of infection
- Blood pressure can be normal!

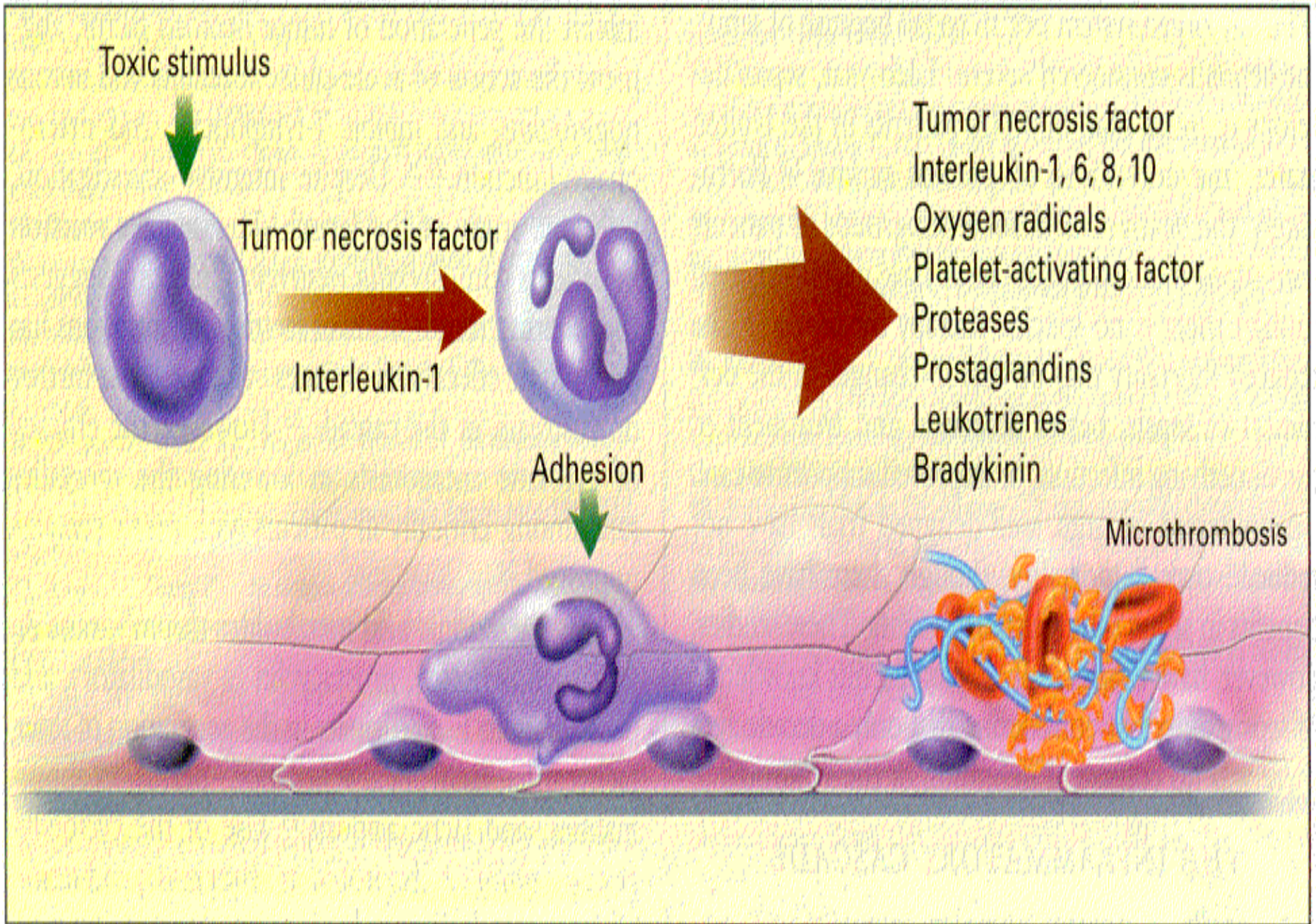
# Septic Shock

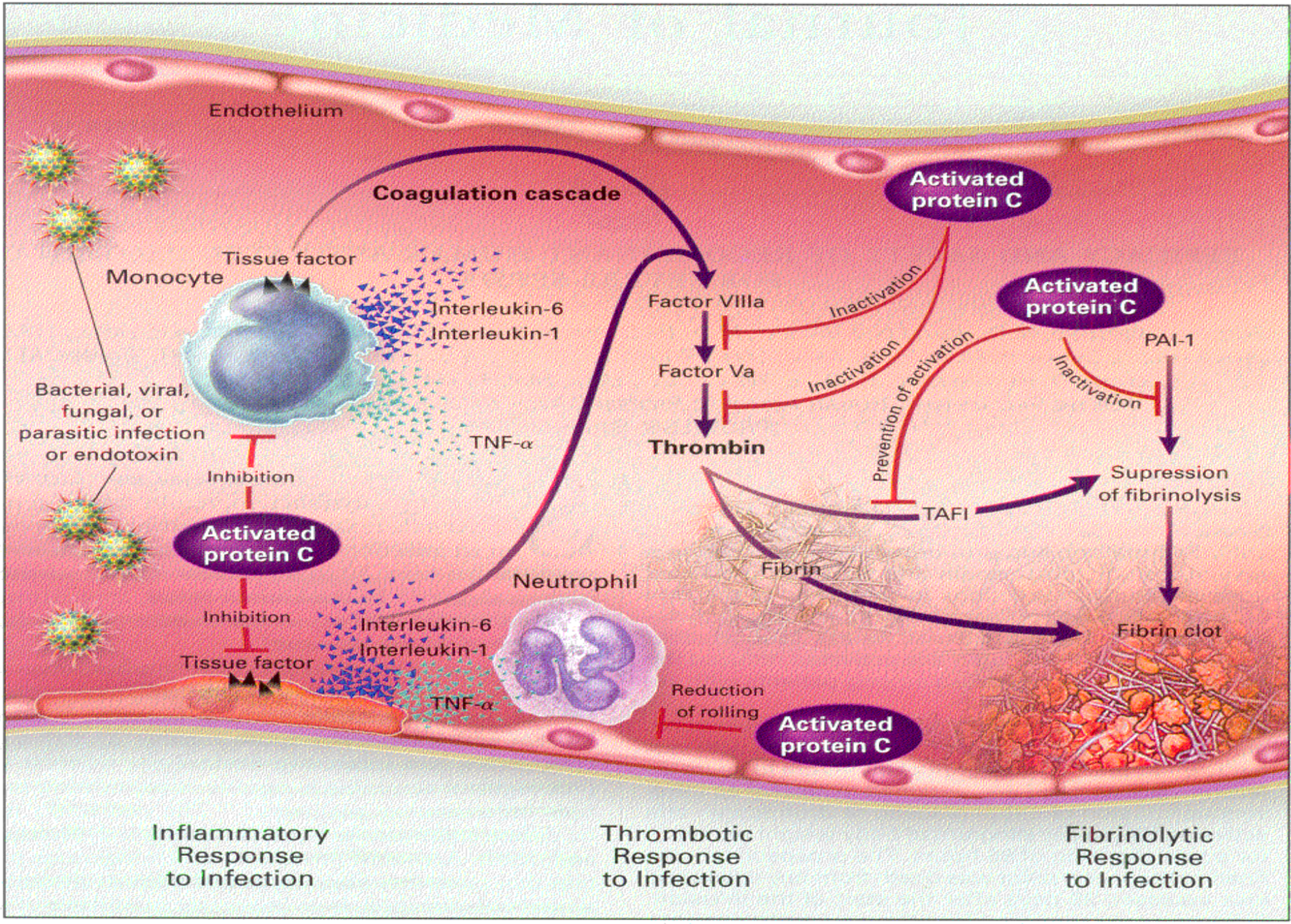
- Sepsis
- Plus refractory hypotension
  - After bolus of 20-40 mL/Kg patient still has one of the following:
    - SBP < 90 mm Hg
    - MAP < 65 mm Hg
    - Decrease of 40 mm Hg from baseline

# Pathogenesis of Sepsis






Toxic stimulus
Tumor necrosis factor
Interleukin-1
Adhesion
Tumor necrosis factor
Interleukin-1, 6, 8, 10
Oxygen radicals
Platelet-activating factor
Proteases
Prostaglandins
Leukotrienes
Bradykinin
Microthrombosis


Endothelium
Coagulation cascade
Activated protein C
Tissue factor
Monocyte
Interleukin-6
Interleukin-1
Factor VIIIa
Inactivation
Activated protein C
Factor Va
Inactivation
PAI-1
Bacterial, viral, fungal, or parasitic infection or endotoxin
TNF-α
Thrombin
Prevention of activation
Inactivation
Inhibition
TAFI
Supression of fibrinolysis
Activated protein C
Fibrin
Neutrophil
Inhibition
Interleukin-6
Interleukin-1
Fibrin clot
Tissue factor
TNF-α
Reduction of rolling
Activated protein C
Inflammatory Response to Infection
Thrombotic Response to Infection
Fibrinolytic Response to Infection

# Septic Shock

- Clinical signs:
  - Hyperthermia or hypothermia
  - Tachycardia
  - Wide pulse pressure
  - Low blood pressure (SBP<90)
  - Mental status changes
  - Fall in urine output( <25-30ml/hr)
- Beware of compensated shock!
  - Blood pressure may be “normal”

# Ancillary Studies

- Cardiac monitor
- Pulse oximetry
- CBC,coagulation profile, LFTs, lipase, Blood sugar,RFTs,C reactive protein
- ABG with lactate
- Blood culture x 2, urine culture
- CXR
- Foley catheter (urine output)

# *MANAGEMENT OF SEPTIC SHOCK*

I. **Reverse Shock**

a) Restore & maintain altered haemodynamic profile to normal

- Volume infusions to keep PCWP at 15-18mmHg
- Inotropic support & vasopressors to ensure adequate O2 transport

b) Objectives:MAP ≥ 65mmHg,SvO2 70%, urine output ≥ 1ml/kg,no base deficit

II. **Ventilator support to all critically ill**

## III. Eradicate infection

- Antibiotics- Survival correlates with how quickly the correct drug was given
- Cover gram positive and gram negative bacteria

  cefoperazone or ceftriaxone 1 gram IV ***or***

  - Imipenem 1 gram IV
- Add additional coverage as indicated
  - Pseudomonas- Gentamicin or Cefepime
  - MRSA- Vancomycin
  - Intra-abdominal or head/neck anaerobic infections- Clindamycin or Metronidazole
  - Asplenic- Ceftriaxone for *N. meningitidis, H. infuenzae*
  - Neutropenic – Cefepime or Imipenem

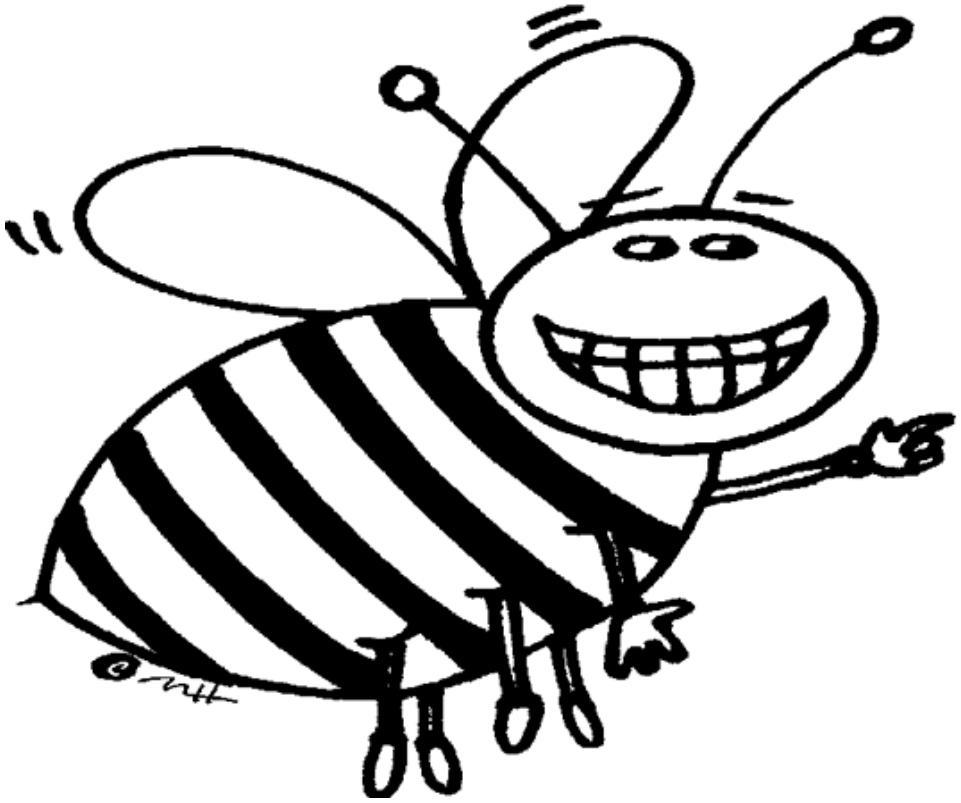
IV. Recombinant human activated protein C in severe cases as continuous infusion at 24ug/kg/hr for 4 days.

V. Provide nutritional,metabolic support

VI. Prophylaxis for deep vein thrombosis & stress ulcer

VII. Corticosteroids: Hydrocortisone as 50mg IV 8hrly for 5-10 days

# Anaphalactic Shock



# ETIOLOGY

1. Antibiotics
   - Pencillin & analogs
   - Sulfonamides
   - Tetracyclines
   - Streptomycin
2. Local anaesthetics
   - Lidocaine
3. Nonsteroidal anti-inflammatory drugs
4. Blood products& vaccines
   - RBC,WBC& Platelet transfusions
   - Gamma-globulin
   - Rabies
   - Tetanus
   - Diphtheria
   - Snake & spider antivenoms

6. Venoms
- Bees,wasps,spiders,jelly fish

7. Foods
- Eggs,milk &milk products, legumes,nuts,shellfish,citrus fruits

8. Hormones
- Insulin
- Hydrocortisone
- Pituitary extracts
- Vasopressin

9. Other drugs
- Protamine
- Parental iron
- Dextrans

# Common Features

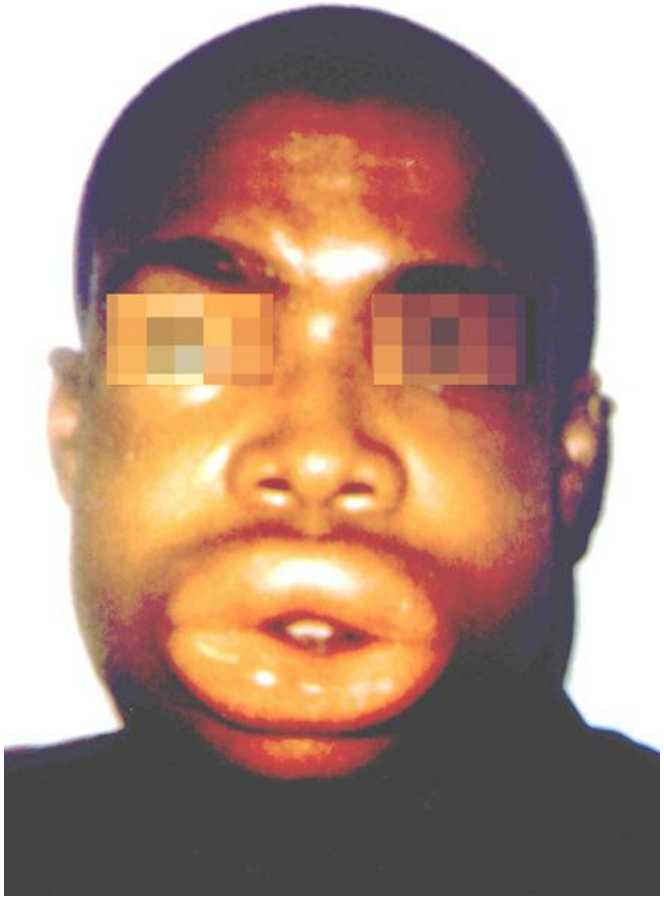
- Angio-oedema
- Bronchoconstriction
- Vasodilatation and hypotension
- Urticareal rash
- Laryngeal edema & obstruction
- Tachycardia, arrhythmias,syncope& seizures
- Diaphoresis,abdominal pain with cramps & diarrhoea  may occur

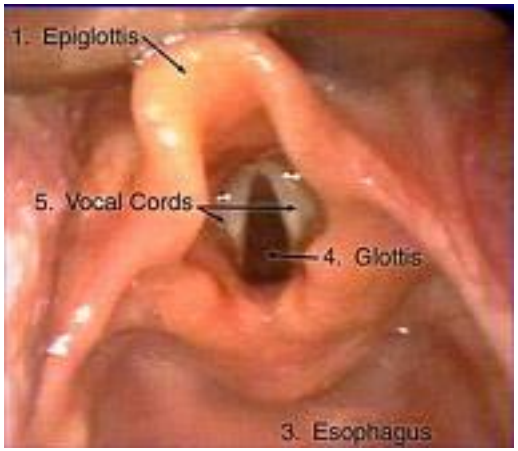
# Anaphylactic Shock

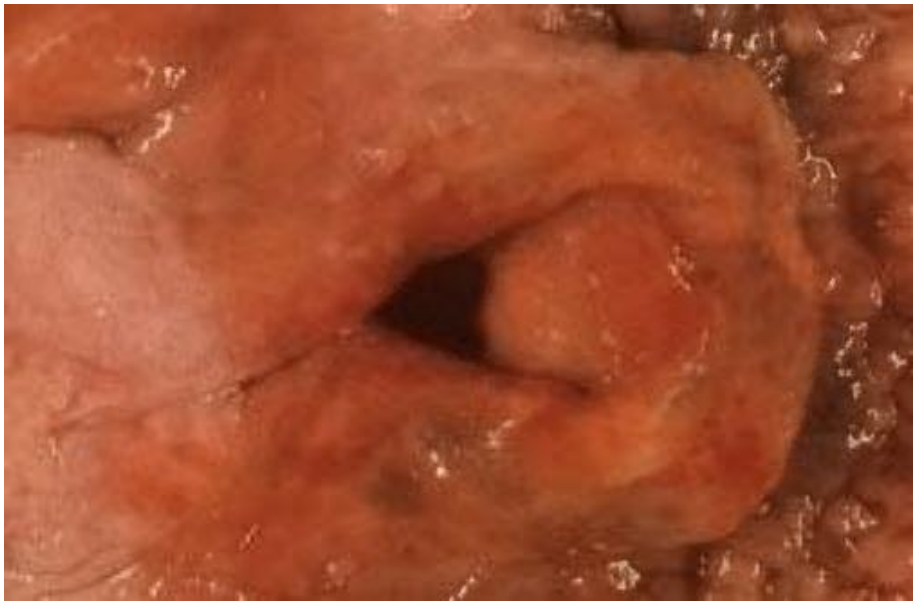
## Symptoms

- First- Pruritus, flushing, urticaria appear
- Next- Throat fullness, anxiety, chest tightness, shortness of breath and lightheadedness
- Finally- Altered mental status, respiratory distress and circulatory collapse

# Angio-oedema





# Anaphylactic Shock

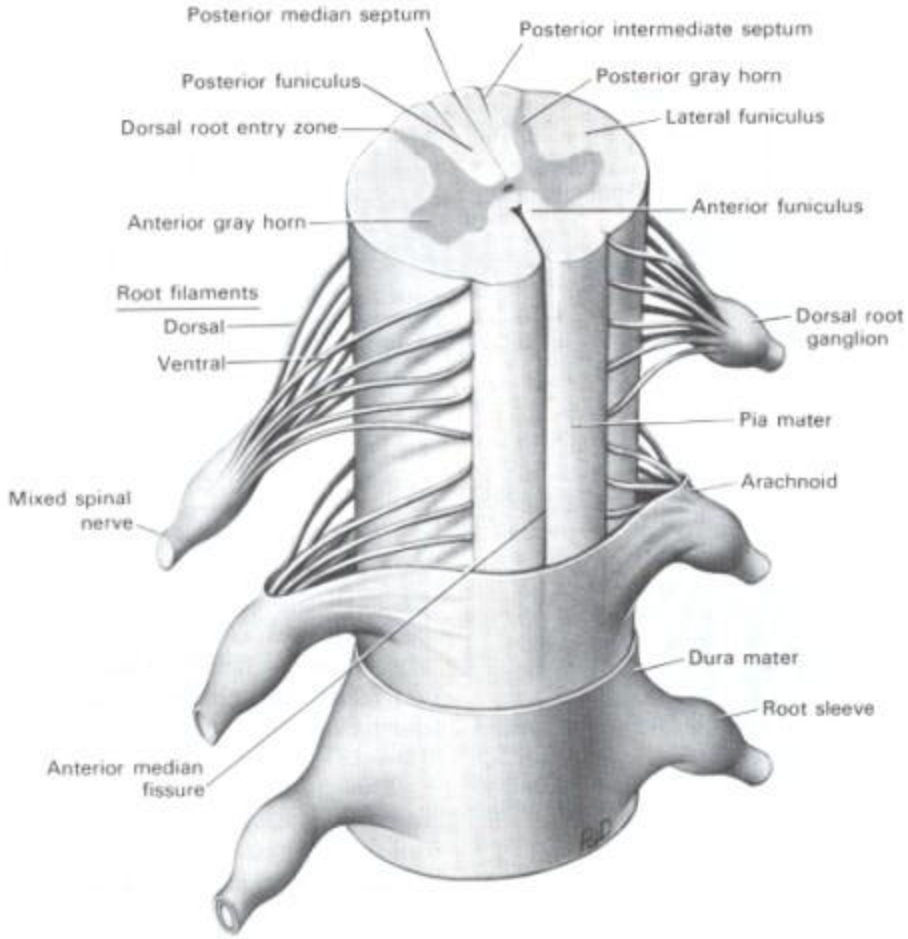
- Mild, localized urticaria can progress to full anaphylaxis
- Symptoms usually begin within 60 minutes of exposure
- Faster the onset of symptoms = more severe reaction
- Biphasic phenomenon occurs in up to 20% of patients
  - Symptoms return 3-4 hours after initial reaction has cleared
- A “lump in my throat” and “hoarseness” heralds life-threatening laryngeal edema

# *MANAGEMENT*

1. Intubate & administer oxygen at high flow rates. If intubation not possible, emergency tracheostomy or puncture of cricothyroid membrane.
2. Epinephrine 0.5-1ml of 1:1000 solution s/c; this can be repeated every 10 mins, & is followed by a maintenance infusion of epinephrine.
3. If no response to s/c epinephrine or if laryngospasm or CV collapse present, 5-10 ml of 1:10000 solution of epinephrine given IV.
4. If IV access unavailable,0.5-1ml of1;1000 solution of epinephrine given IM or 10ml of 1:10000 solution instilled through endotracheal tube.

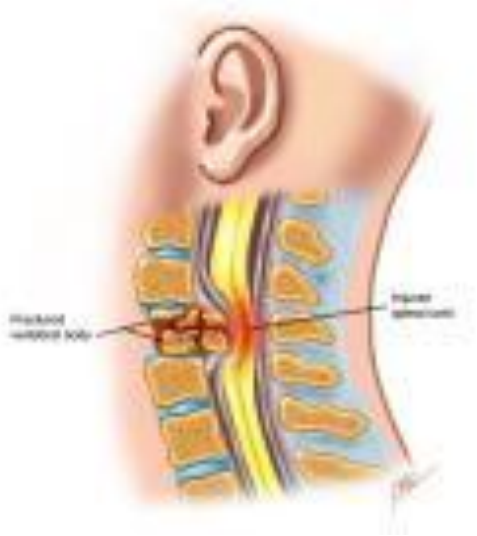
4. Administer volume load.
5. Use antihistaminic e.g. diphenhydramine 1 mg/kg IV & repeat 6 hrly.
6. Relief of bronchospasm by IV aminophylline 250 mg in 20ml dextrose
7. Hydrocortisone 300mg IV stat,followed by 100mg IV 6 hrly.
8. If hypotension persists despite epinephrine,volume load &antihistaminics, start dopamine infusion.If this doesnot raise BP,start epinephrine infusion.
9. Ventillatory support with high FiO2 may be required in critically ill patients.

# Neurogenic Shock



# Neurogenic Shock

- A form of distributive shock caused by the sudden loss of CNS signals to vascular smooth muscle following spinal cord injury
- Results in an immediate decrease in peripheral vascular resistance and hypotension
- May be associated with normal heart rate or even bradycardia



# Neurogenic Shock

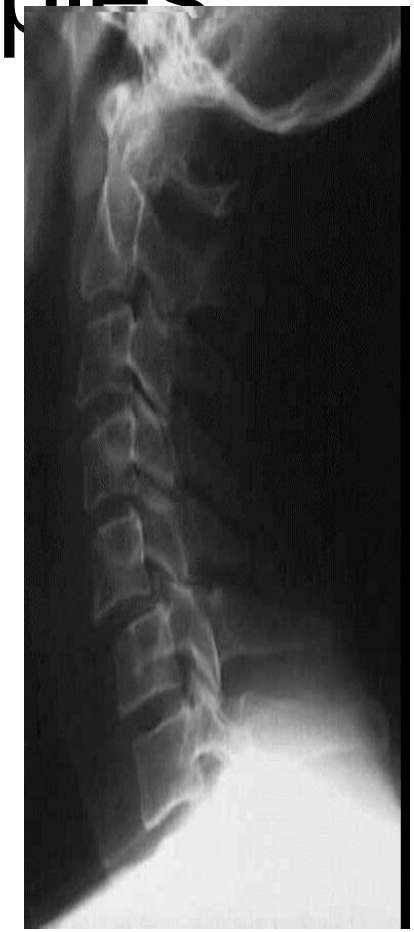
- Loss of sympathetic tone results in warm and dry skin
- Shock usually lasts from 1 to 3 weeks
- Any injury above T1 can disrupt the entire sympathetic system
  - Higher injuries = worse paralysis

# Neurogenic Shock
# Basic Management Principles

- Adequate oxygenation
- Spine immobilization
- Restore vasomotor tone after insuring adequate volume status
  - Alpha agonist pharmacologic agents
- Often a role for early use of flow directed pulmonary artery catheter
- For bradycardia

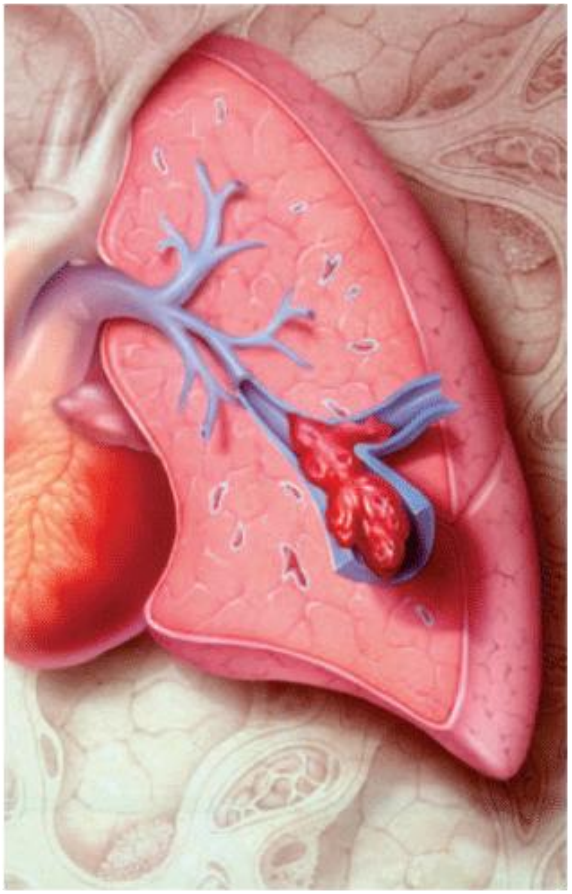
  Atropine

  Pacemaker



- Methylprednisolone
  - Used only for blunt spinal cord injury
  - High dose therapy for 23 hours
  - Must be started within 8 hours
  - Controversial- Risk for infection, GI bleed

# Obstructive Shock



# Obstructive Shock

- Tension pneumothorax
  - Air trapped in pleural space with 1 way valve, air/pressure builds up
  - Mediastinum shifted impeding venous return
  - Chest pain, SOB, decreased breath sounds
  - Rx: Needle decompression, chest tube

# Obstructive Shock

- Cardiac tamponade
  - Blood in pericardial sac prevents venous return to and contraction of heart
  - Related to trauma, pericarditis, MI
  - Beck's triad: hypotension, muffled heart sounds, JVD
  - Diagnosis: large heart CXR, echo
  - Rx: Pericardiocentisis

# Obstructive Shock

- Pulmonary embolism
  - Virchow triad: hypercoaguable, venous injury, venostasis
  - Signs: Tachypnea, tachycardia, hypoxia
  - Low risk: D-dimer
  - Higher risk: CT chest or VQ scan
  - Rx: Heparin, consider thrombolytics

# Obstructive Shock

- Aortic stenosis
  - Resistance to systolic ejection causes decreased cardiac function
  - Chest pain with syncope
  - Systolic ejection murmur
  - Diagnosed with echo
  - Rx: Valve surgery

# Multiorgan Dysfunction Syndrome (MODS)

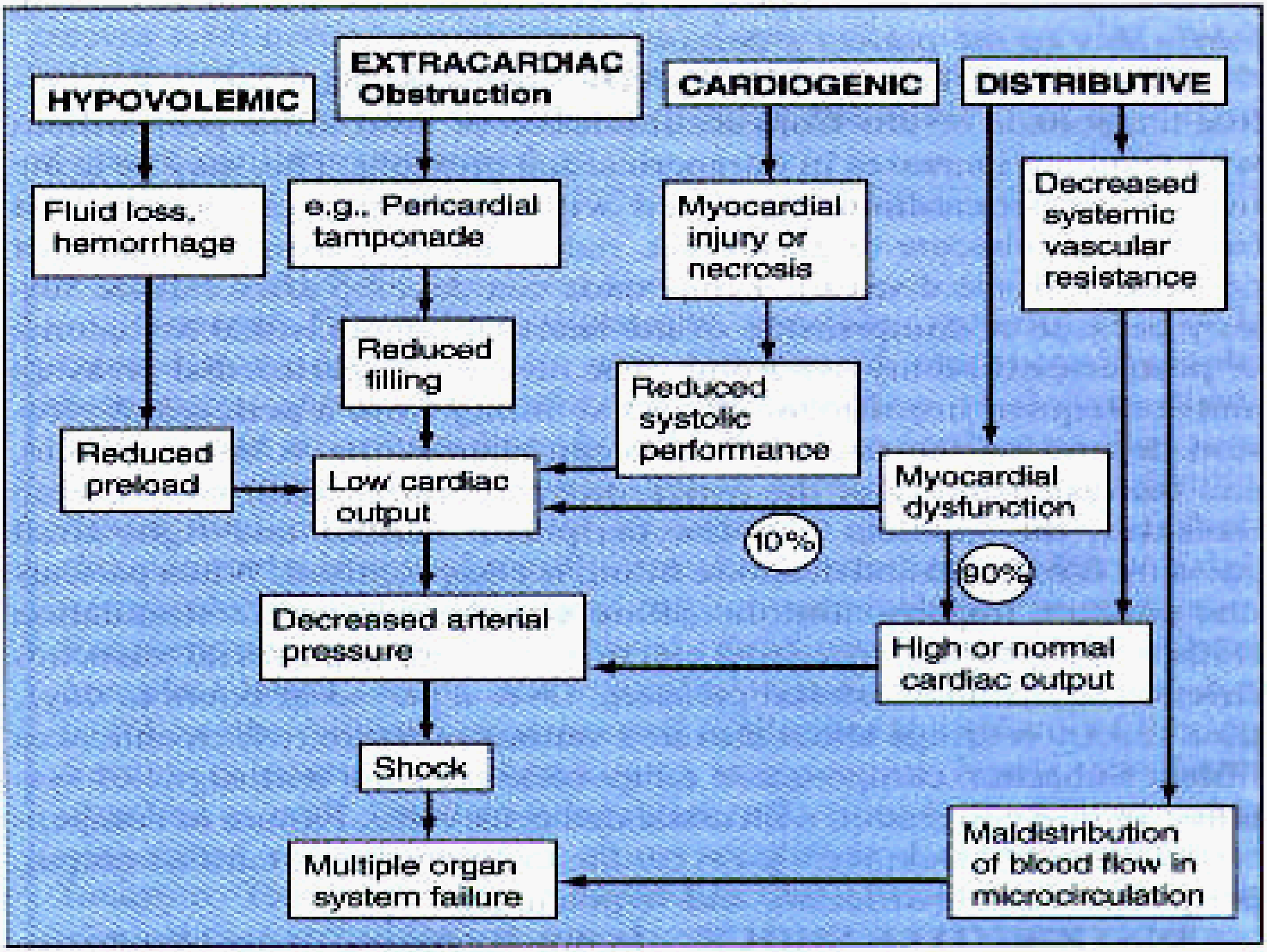
- Progression of physiologic effects as shock ensues
  - Cardiac depression
  - Respiratory distress
  - Renal failure
  - DIC
- Result is end organ failure

- Can occur as a complication of all forms of shock
- The exact mechanism that triggers it is unknown
- Usually begins with lungs and is followed by liver and kidneys
- 2 patterns of presentation:
  - Initial episode of hypotension which is treated and patient seemingly responds
  - If patient presents with a pulmonary insult and has respiratory failure, can rapidly develop MOF and patient only survives 2 to 4 days

- Other pattern occurs most often with septic shock:
  - progressive development over a month
  - patient experiences respiratory failure and often requires ventilator
  - despite apparent hemodynamic stability, patient exhibits a hypermetabolic state (hyperglycemia, hyperlactatemia, polyuria) - if can be reversed, mortality rate is 25-40%
  - infection is usually present and skin breakdown begins to occur

- severe loss of muscle mass (auto- catabolism) occurs
- if hypermetabolic phase can't be reversed, MOF progresses

- patient becomes jaundiced, has hyperbilirubinemia and renal failure - often requires dialysis
  - patient becomes hemodynamically unstable
  - Mortality rate increases to 40-60% during early stage of MOF and 90-100% in later stage - Patient usually dies in about 28 days


HYPOVOLEMIC
EXTRACARDIAC Obstruction
CARDIOGENIC
DISTRIBUTIVE
Fluid loss, hemorrhage
e.g., Pericardial tamponade
Myocardial injury or necrosis
Decreased systemic vascular resistance
Reduced filling
Reduced systolic performance
Reduced preload
Low cardiac output
Myocardial dysfunction
10%
90%
Decreased arterial pressure
High or normal cardiac output
Shock
Multiple organ system failure
Maldistribution of blood flow in microcirculation