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Shock

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CONTENTS

INTRODUCTION

- Shock is a physiologic event with many different causes; but if untreated it has a single clinical outcome.
- Mortality rate - 20%.

DEFINITION

- Shock is a life threatening situation due to poor tissue perfusion with impaired cellular metabolism, manifested in turn by serious pathophysiological abnormalities. (Bailey and love)

- Shock is a term used to describe the clinical syndrome that develops when there is critical impairment of tissue perfusion due to some form of acute circulatory failure. (Davidson's)

- Shock may be defined as inadequate delivery of oxygen and nutrients to maintain normal tissue and cellular function.(Schwartz's)

- The state in which profound and widespread reduction of effective tissue perfusion leads first to reversible, and then if prolonged, to irreversible cellular injury. (Kumar and Parrillo ,1995)

- A life-threatening clinical syndrome of cardiovascular collapse characterized by :

  -An acute reduction of effective circulating blood volume (hypotension)

  -An inadequate perfusion of cells and tissues (hypoperfusion)
- If uncompensated, these mechanisms may lead to impaired cellular metabolism and death.
- The clinical manifestations of shock are the result of stimulation of the sympathetic and neuroendocrine stress responses, inadequate oxygen delivery, end-organ dysfunction.

inadequate tissue perfusion

cellular dysfunction

damage-associated molecular patterns (DAMPs or "danger signals") and inflammatory mediators

maldistribution of blood flow, further compromising cellular perfusion; causing multiple organ failure (MOF) and, if the process is not interrupted, leads to death

SHOCK

CLASSIFICATION 1

- Primary (INITIAL SHOCK)
- Secondary (TRUE SHOCK)
- Anaphylactic (Type I immunologic reaction)
- True shock- circulatory imbalance between oxygen supply and oxygen requirements at cellular level; hence name CIRCULATORY SHOCK.

- Initial shock - transient and usually benign vasovagal attack due to sudden reduction of venous return caused by neurogenic vasodilatation and consequent peripheral pooling of blood (immediately following trauma, severe pain, emotional over reaction etc.)

- In routine clinical practice, true shock is the form which occurs due to hemodynamic derangements with hypo perfusion - commonly referred to as shock.

ACCORDING TO ETIOLOGY

- HYPOVOLEMIC SHOCK
- CARDIOGENIC SHOCK
- SEPTIC SHOCK
- OTHER TYPES :
  - TRAUMATIC
  - NEUROGENIC
  - HYPOADRENAL

(Harsh Mohan 4th ed)

- Due to low flow(reduced stroke volume)
  - hypovolemic
  - cardiogenic
  - obstructive

- Due to low peripheral arteriolar resistance (vasodilatation)
  - septic
  - anaphylactic
  - neurogenic (Davidson's 21st ed)

- Vasovagal
- Psychogenic
- Neurogenic
- Hypovolemic
- Traumatic
- Burns
- Cardiogenic
- Septic (endotoxin) : → hyper dynamic /warm
  → hypovolemic hypo dynamic /cold
- Anaphylactic

(Bailey & Love's short practice of surgery)

Proposed by HINSHAW and COX (1972)

1. Hypovolemic shock
2. Cardiogenic shock
3. Extra cardiac obstructive shock
4. Distributive shock

    Septic shock

    Anaphylactic shock

    Neurogenic shock

- SHOCK DUE TO REDUCED BLOOD VOLUME (HYPOVOLEMIC SHOCK OR COLD SHOCK)

ü TRAUMATIC SHOCK
ü HEMORRHAGIC SHOCK
ü SURGICAL SHOCK
ü BURN SHOCK
ü DEHYDRATION SHOCK

- SHOCK DUE TO INCREASED VASCULAR CAPACITY(Blood volume normal; occurs because of inadequate blood supply to the tissues due to increased vascular capacity):

ü NEUROGENIC SHOCK

ü ANAPHYLACTIC SHOCK

ü SEPTIC SHOCK

- SHOCK DUE TO DISEASES OF THE HEART(CARDIOGENIC SHOCK)
- SHOCK DUE TO OBSTRUCTION OF BLOOD FLOW.

What is Shock_.flv

PATHOPHYSIOLOGY

HYPOVOLAEMIC SHOCK

- hemorrhage
  - trauma
  - surgery
  - burns
- dehydration

SEPTIC SHOCK

gram negative septicemia
gram positive septicemia

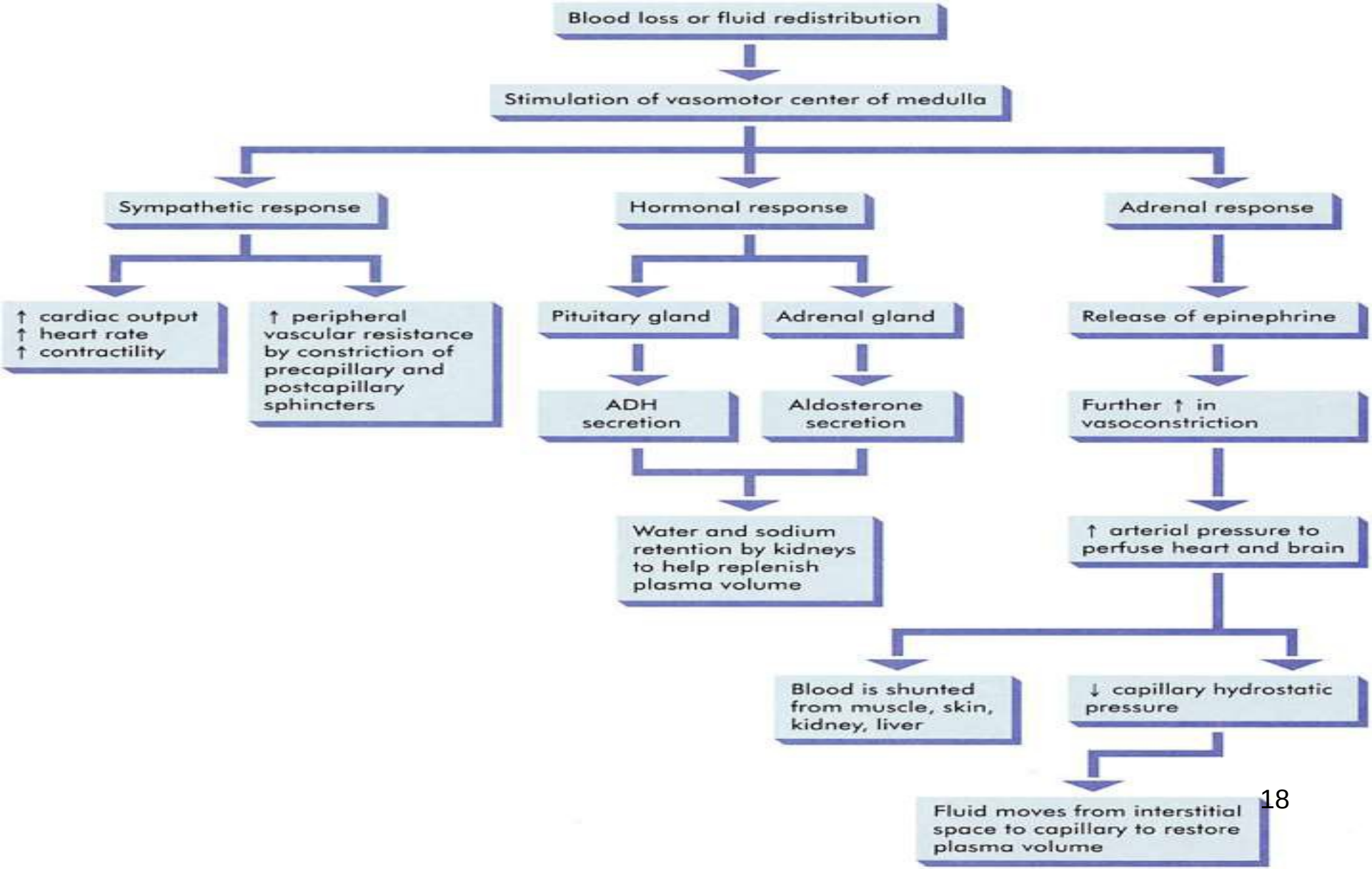
CARDIOGENIC SHOCK

STAGES OF SHOCK

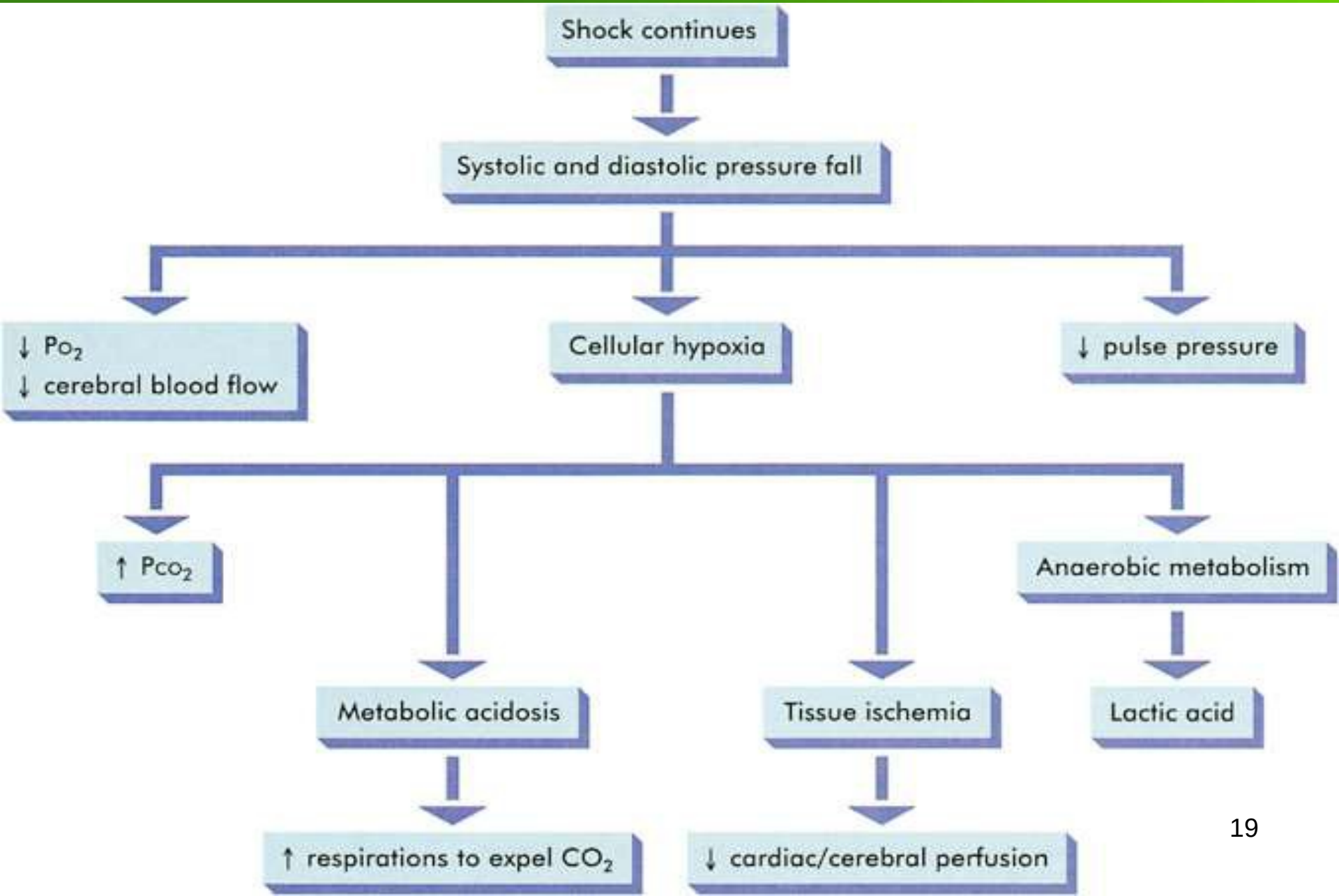
- Deterioration of circulation in shock is a progressive & continuous phenomenon & compensatory mechanisms become progressively less effective

1. NON-PROGRESSIVE (INITIAL, COMPENSATED REVERSIBLE) SHOCK
2. PROGRESSIVE DECOMPENSATED SHOCK
3. DECOMPENSATED (IRREVERSIBLE) SHOCK

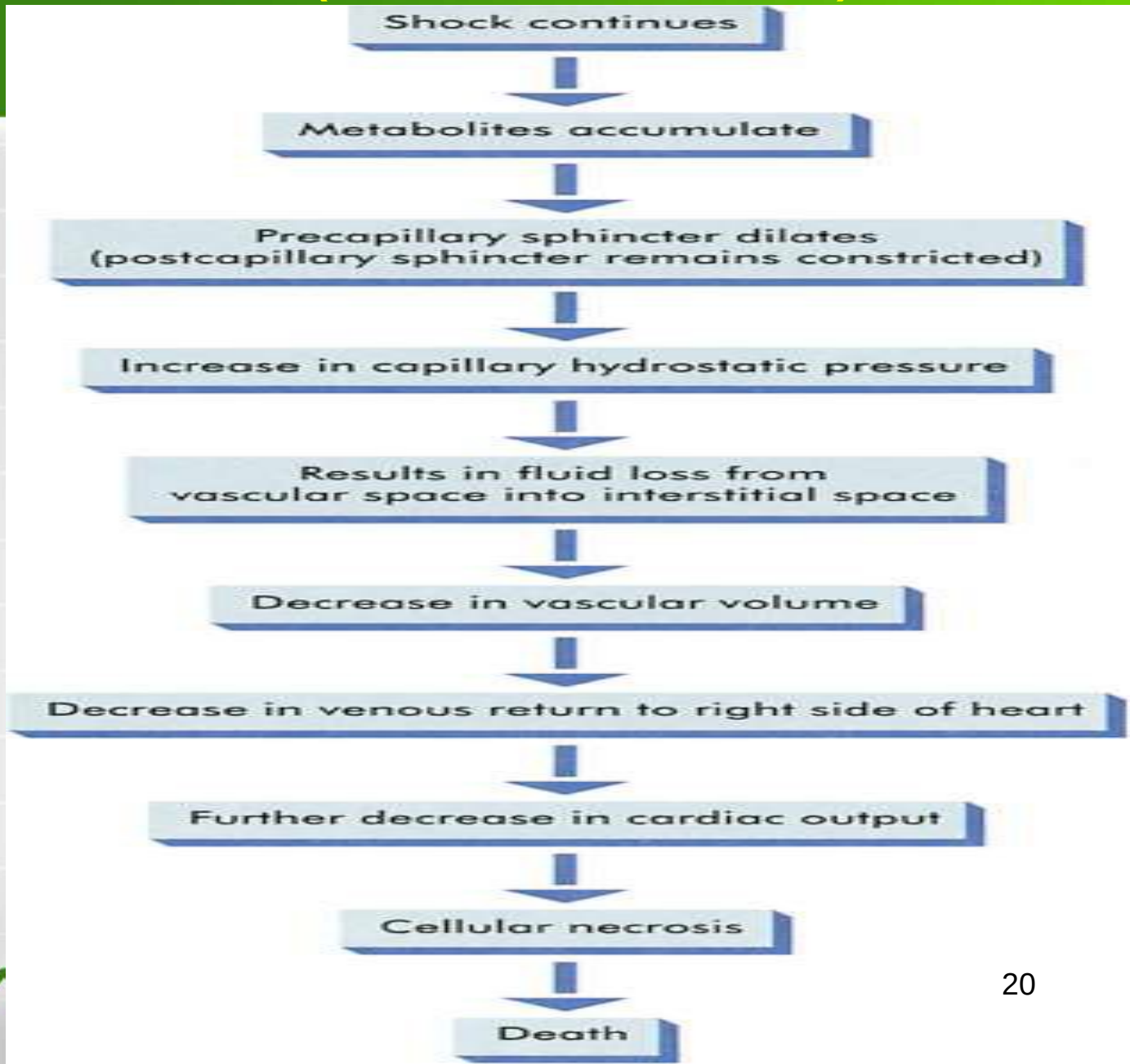
NON-PROGRESSIVE (INITIAL, COMPENSATED REVERSIBLE) SHOCK



PROGRESSIVE DECOMPENSATED



DECOMPENSATED (IRREVERSIBLE) SHOCK



STAGE	PATHOGENESIS	EFFECTS
INITIAL SHOCK	• Baroreceptor • Catecholamine release • Renin-angiotens in activation • ADH release • Sympathetic stimulation	• Peripheral vasoconstriction • Cool clammy skin • Tachycardia • Fluid conservation by kidney
PROGRESSIVE SHOCK	• Anaerobic glycolysis • Lactic acidosis • Lowered pH	• ↓ Cardiac output • DIC • Mental confusion • ↓ Urinary output
IRREVERSIBLE SHOCK	• Persistent vasoconstriction • Vasodilatation & ↑ vascular permeability • Myocardial ischaemia (MDF) • Cerebral ischaemia • VDM • TNF • Intestinal factor • Bacterial factor • Hypercoabulability	• Brain: death • Lungs: ARDS • Heart: focal myocardial necrosis • Kidney: ATN • Liver: necrosis • Spleen: hyperplasia • Stomach: ulcer • Intestine: necrosis • Splanchnic: vasodilatation

GENERAL CLINICAL FEATURES

- Hypotension (Systolic BP<100mmHg)
- Tachycardia (>100/min)
- Cold , Clammy Skin
- Rapid, Shallow Respiration
- Drowsiness, Confusion, Irritability
- Oliguria (Urine Output<30ml/hour)
- Elevated or Reduced central venous pressure
- Multi-Organ Failure

GENERAL PRINCIPLES IN MANAGEMENT

- Patients should be treated in ICUs preferably
- Continuous electrocardiographic monitoring
- Pulse oximetry
- A reduction of elevated serum lactate levels is one good indicator of successful resuscitation and is often used as a therapeutic goal

Initial Assessment - ABC

- Airway:

  Does patient have mental status to protect airway?

  GCS less than “eight” means “intubate” (E4 V5 M6)

  Airway is compromised in anaphylaxis

- Breathing:

  If patient is conversing, A & B arefine

  Place patient on oxygen

- Circulation:
  - Vitals (HR, BP)
  - IV, start fluids, put on continuous monitor

- In a trauma, perform ABCDE, not just ABC
- Deficit or Disability
    - Assess for obvious neurologic deficit
    - Movement of all four extremities? Pupils?
    - Glasgow Coma Scale (V5, M6, E4)
- Exposure
    - Loosening of clothing on trauma patients.

EFFECT OF SHOCK

CARDIOVASCULAR

RESPIRATORY

RENAL and ENDOCRINE

decreased urine output

stimulation of renin angiotensin and aldosterone axis

release of vasopressin from hypothalamus

resulting vasoconstriction and increase Na+ and water reabsorption.

MICROVASCULAR

Activation of immune and coagulation systems hypoxia and acidosis , activate complement and prime neutrophils oxygen free radicles and cytokine release damaged and endothelium fluids leak out and edema ensues.

CELLULAR

Cells switch from aerobic to anaerobic metabolism à Decreased ATP production à lactic acidosis à Glucose exhausts and aerobic respiration ceases à Na+/ K+ pump impaired à Lysosomes release autodigestive enzymes à mitochondria damage à cell death.

METABOLIC CHANGES IN SHOCK

CARBOHYDRATE METABOLISM

- Compensated shock : Hyperglycemia due to increased hepatic glycogenolysis.
- Decompensated shock : Hypoglycemia due to hepatic glycogen depletion & increased consumption of glucose by tissue.
- Anaerobic glycolysis occurs as assessed by high blood levels of lactate & pyruvate.

PROTEIN METABOLISM

- Increased intracellular protein catabolism
- Conversion of amino acids to urea.
- Increased blood non-nitrogen protein.

FAT METABOLISM

- Increased endogenous fat metabolism.
- Rise of fatty acid level in blood.

WATER & ELECTROLYTE DISTURBANCES

- Failure of sodium pump à potassium leaves the cell (hyponatremia) à causes cellular swelling.
- Shock due to loss of plasma only (in burns) à hemoconcentration

METABOLIC ACIDOSIS

- Hypoxia of kidney, renal function is impaired blood levels of acids like lactate, pyruvate, phosphate & sulfate rise causing metabolic acidosis.

MORPHOLOGIC COMPLICATIONS

- Morphologic changes in shock are due to Hypoxia. resulting in degeneration & necrosis in various organ.
- Organs affected are : Brain, Heart, Lungs, Kidneys, Adrenals and GIT.

HYPOXIC ENCEPHALOPATHY

Compensated shock results in cerebral ischemia which produce altered state of consciousness. However ,if blood pressure falls below 50 mmHg as in systemic hypotension in prolonged shock & cardiac arrest, Brain suffers from serious ischemic damage with loss of cortical functions, coma,& vegetative state.

HEART IN SHOCK

2 types of morphologic changes in Heart

1. Hemorrhage's & Necrosis : Located in subepicardial & subendocardial region.
2. Zonal Lesion: Opaque transverse contraction bands in a myocyte near an intercalated disc.

SHOCK LUNG

- Lungs have Dual blood supply & generally not affected by hypovolemic shock
- But in Septic shock à SHOCK LUNG seen as symptoms of ARDS including congestion , interstitial & alveolar edema, interstitial lymphocytic infiltrate, alveolar hyaline membrane.
- Thickening & fibrosis of alveolar septa, fibrin & platelet thrombi in pulmonary microvasculature.

SHOCK KIDNEY

- Irreversible renal injury à Important complication of Shock.
- Renal ischemia following systemic hypotension is considered responsible for renal changes in Shock à End result is generally anuria & death.

ADRENALS IN SHOCK

Adrenals show stress response in SHOCK. It includes

1.Release of aldosterone in response to hypoxic kidney.

2.Release of glucocorticoids from adrenal cortex & catecholamine like adrenaline from adrenal medulla.

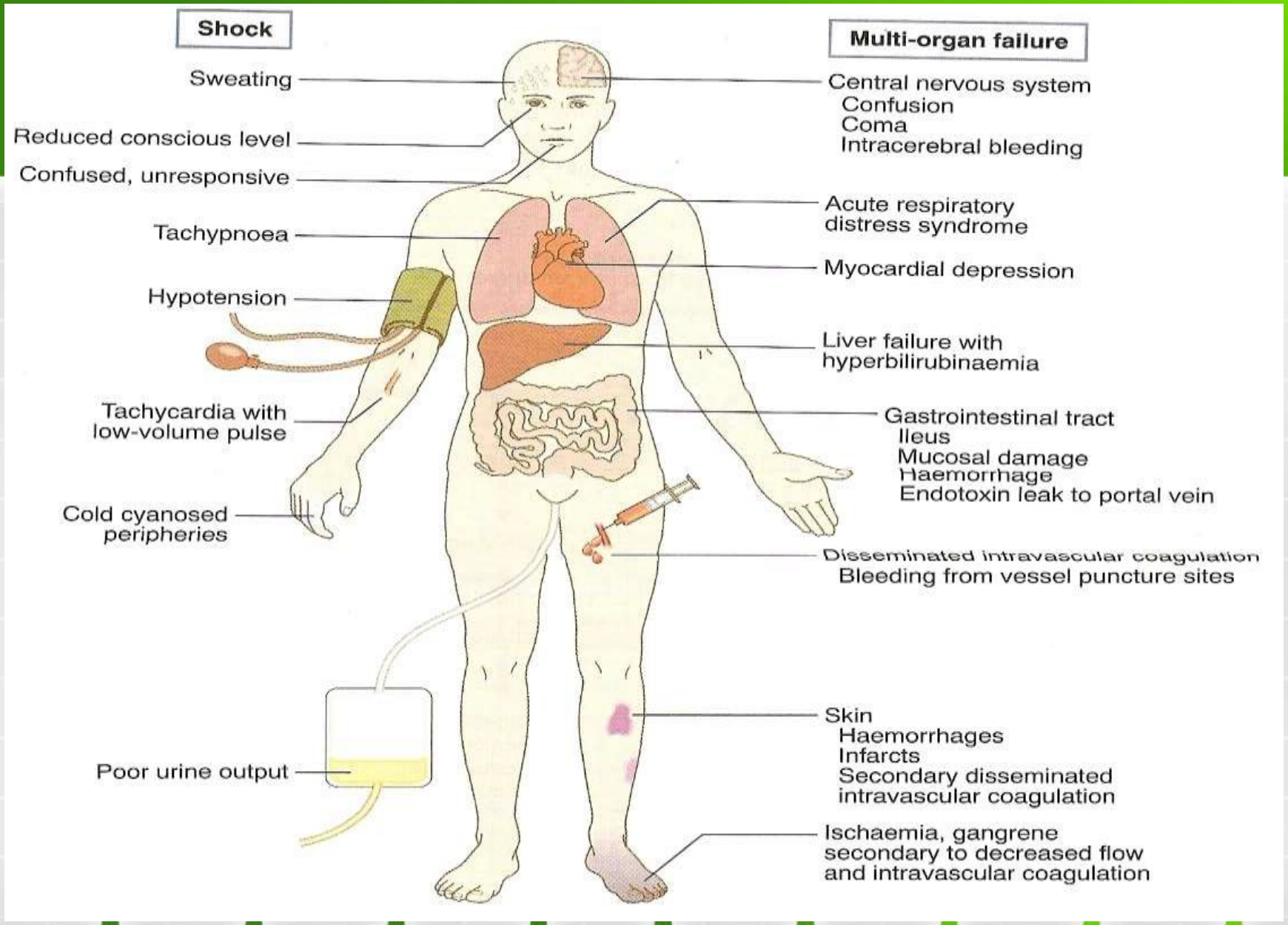
"SEVERE SHOCK RESULTS IN ADRENAL HAEMORRHAGES"

GIT

- Hypo perfusion of Alimentary tract à Mucosal & Mural infarction called "HAEMORRHAGIC GASTROENTEROPATHY "
- In Shock due to burns, acute stress à ulcers of stomach/duodenum à "CURLING'S ULCERS"

LIVER

- Hypoxia, VDM is released à Vasodilatation
- Others include focal necrosis, fatty change, impaired liver function.


Shock
Sweating
Reduced conscious level
Confused, unresponsive
Tachypnoea
Hypotension
Tachycardia with low-volume pulse
Cold cyanosed peripheries
Poor urine output
Multi-organ failure
Central nervous system
Confusion
Coma
Intracerebral bleeding
Acute respiratory distress syndrome
Myocardial depression
Liver failure with hyperbilirubinaemia
Gastrointestinal tract
Ileus
Mucosal damage
Haemorrhage
Endotoxin leak to portal vein
Disseminated intravascular coagulation
Bleeding from vessel puncture sites
Skin
Haemorrhages
Infarcts
Secondary disseminated intravascular coagulation
Ischaemia, gangrene secondary to decreased flow and intravascular coagulation

ISCHEMIC REPERFUSION SYNDROME

It is the injury that occurs once the normal circulation is restored to the tissues

Reasons:

-Acid and potassium load built up leads to myocardial depression, vascular dilatation and hypotension.

-Neutrophils are flushed back into the circulation; causes further injury to the endothelial cells of lungs and kidneys.

Results:

Acute lung and renal injury

Multiple organ failure

Death

HYPOVOLEMIC SHOCK

- Occurs from inadequate circulating blood volume
- Major effects are due to decreased cardiac output and low intra cardiac pressure
- Severity of clinical features depends on degree of blood volume lost

PATHOPHYSIOLOGY

Hemorrhage from small venules & veins (50%)

Decreased filling of right heart

Decreased filling of pulmonary vasculature

Decreased filling of left atrium & ventricle

Left ventricular stroke volume decreases (Frank Starling)

Drop in arterial blood pressure & tachycardia

Poor perfusion to pulmonary arteries

Cardiac depression & pump failure

CLASSSIFICATION OF HYPOVOLEMIC SHOCK

HEMORRHAGIC: TRAUMA

GASTROINTESTINAL BLEEDING

NON-HEMORRHAGIC:

EXTERNAL FLUID LOSS

DIARRHOEA

VOMITING

POLYUREA

FLUID REDISTRIBUTION

BURNS

ANAPHYLAXIS

CLASSIFICATION OF ACUTE BLOOD LOSS

- Class I : blood loss up to 15% à mild clinical symptoms (compensated)
- Class II: blood loss 15-30% (1000-1500ml) à mild tachycardia, tachypnea, weak peripheral pulses and anxiety (mild)
- Class III: blood loss 30-40% (1500-2000ml) à Hypotension, marked tachycardia [pulse >110 to 120 bpm], and confusion (moderate)
- Class IV: blood loss >40% (>2000ml) à significant depression in systolic BP, very narrow pulse pressure (severe)

	Class I	Class II	Class III	Class IV
Blood loss (mL)	Up to 750 mL	750 – 1500 mL	1500- 2000mL	>2000 mL
Pulse rate & pulse pressure	<100 normal or decreased	>100 decreased	>120 decreased	>140 Decreased
Blood pressure	Normal	Normal	Decreased	Decreased
Respiratory rate	14 – 20	20 -30	30 - 40	> 35
Urine output mL/hr	> 30	20 -30	5 -15	Negligible
Fluid replacement	Crystalloid	Crystalloid & blood	Crystalloid & blood	Crystalloid

Signs and symptoms

- Anxiety, restlessness, altered mental state
- Hypotension
- A rapid, weak, thready pulse
- Cool, clammy skin
- Rapid and shallow respirations
- Hypothermia
- Thirst and dry mouth
- Distracted look in the eyes

COMPENSATORY MECHANISMS

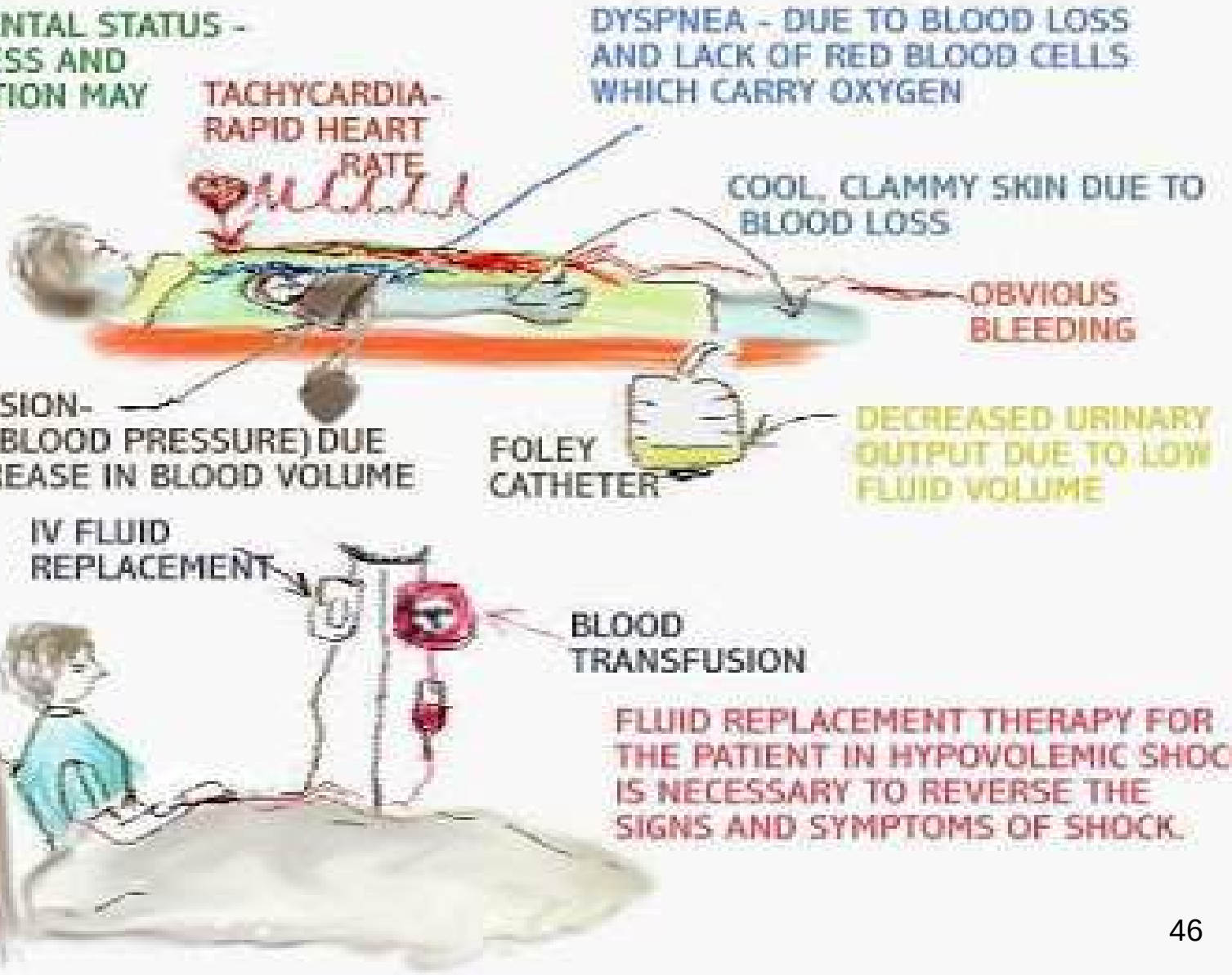
1. Adrenergic discharge
2. Hyperventilation
3. Vasoactive hormones

   Angiotensin ,Vasopressin, Epinephrine
4. Collapse
5. Re-absorption of fluid from interstitial tissue
6. Resorption of fluid from intracellular to extracellular space
7. Renal conservation of body water & electrolyte.

CLINICAL MONITORING

- Blood pressure
- Respiration
- Urine output
- Central venous pressure
- ECG
- Swan-Ganz catheter
  - * cardiac output
  - * mixed venous oxygen level
  - * vascular pressure
- Pulmonary artery wedge pressure

CLINICAL PICTURE OF A PATIENT IN HYPOVOLEMIC SHOCK



DIAGNOSIS

- In management of trauma patients, understanding the patterns of injury of the patient in shock will help direct the evaluation and management.
- Blood loss sufficient to cause shock is generally of a large volume (e.g. external, intrathoracic, intra-abdominal, retroperitoneal, and long bone fractures).
- Diagnostic and therapeutic tube thoracotomy may be indicated in unstable patients based on clinical findings and clinical suspicion.
- Chest radiographs, pelvic radiography, diagnostic ultrasound or diagnostic peritoneal lavage.

MANAGEMENT

OBJECTIVES

a. Increase Cardiac Output
b. Increase Tissue Perfusion

The plan of action should be based on

a. Primary problem
b. Adequate fluid replacement
c. Improving myocardial contractility
d. Correcting acid-base disturbances

- Resuscitation
- Immediate control of bleeding: Rest, Pressure Packing, Operative Methods
- Extracellular fluid replacement:

- Infusion of fluid is the fundamental treatment
- Crystalloids, for initial resuscitation for most forms of hypovolemic shock.
- After the initial resuscitation, with up to several liters of crystalloid fluid, use of colloids.

- Drugs
    1. Sedatives
    2. Chronotropic agents
    3. Inotropic agents

DISTRIBUTIVE SHOCK

- As in hypovolemic shock, there is an insufficient intravascular volume of blood
- This form of "relative" hypovolemia is the result of dilation of blood vessels which diminishes systemic vascular resistance
- Examples of this form of shock :

  Septic shock

  Anaphylactic shock

  Neurogenic shock

TRAUMATIC SHOCK

- Primarily due to hypovolemia from :

ü Bleeding externally eg: open wounds, fractures

ü Bleeding internally eg: ruptured liver, spleen

- Clinical features :

ü Presence of peripheral & pulmonary edema.

ü Infusion of large amount of fluid which is adequate in hypovolemic shock is inadequate here.

PATHOPHYSIOLOGY

Traumatic tissue activates the coagulation system

Release of micro-thrombi into circulation

Obstruction parts of pulmonary micro vasculature

Increased pulmonary vascular resistance

Increased right ventricular diastolic & right atrial pressure

Humoral products of thrombi induce increase in capillary permeability

Loss of plasma into interstitial tissue

Depletion of Vascular volume

MANAGEMENT

1. Resuscitation

2. Local treatment of trauma & control of bleeding , surgical debridement of ischemic & dead tissue & immobilization of fracture.

3. Fluid replacement with Ringers lactate, Ringers acetate, Normal saline.

4. Anticoagulation with one intravenous dose of 10,000 units of heparin

CARDIOGENIC SHOCK

- Primary dysfunction of one ventricle or the other

- Dysfunction may be due to
  - > Myocardial infarction
  - > Chronic congestive heart failure
  - > Cardiac arrhythmias
  - > Pulmonary embolism
  - > Systemic arterial hypertension

Dysfunction of right ventricle à right heart unable to pump blood in adequate amount into lungs, filling of left heart decreases , so left ventricular out put decreases.

Dysfunction of left ventricle à left ventricle unable to maintain adequate stroke volume , left ventricular output & systemic arterial blood pressure decreases ,there is engorgement of the pulmonary vasculature due to normal right ventricular output, but failure of left heart

Cardiogenic compressive shock:

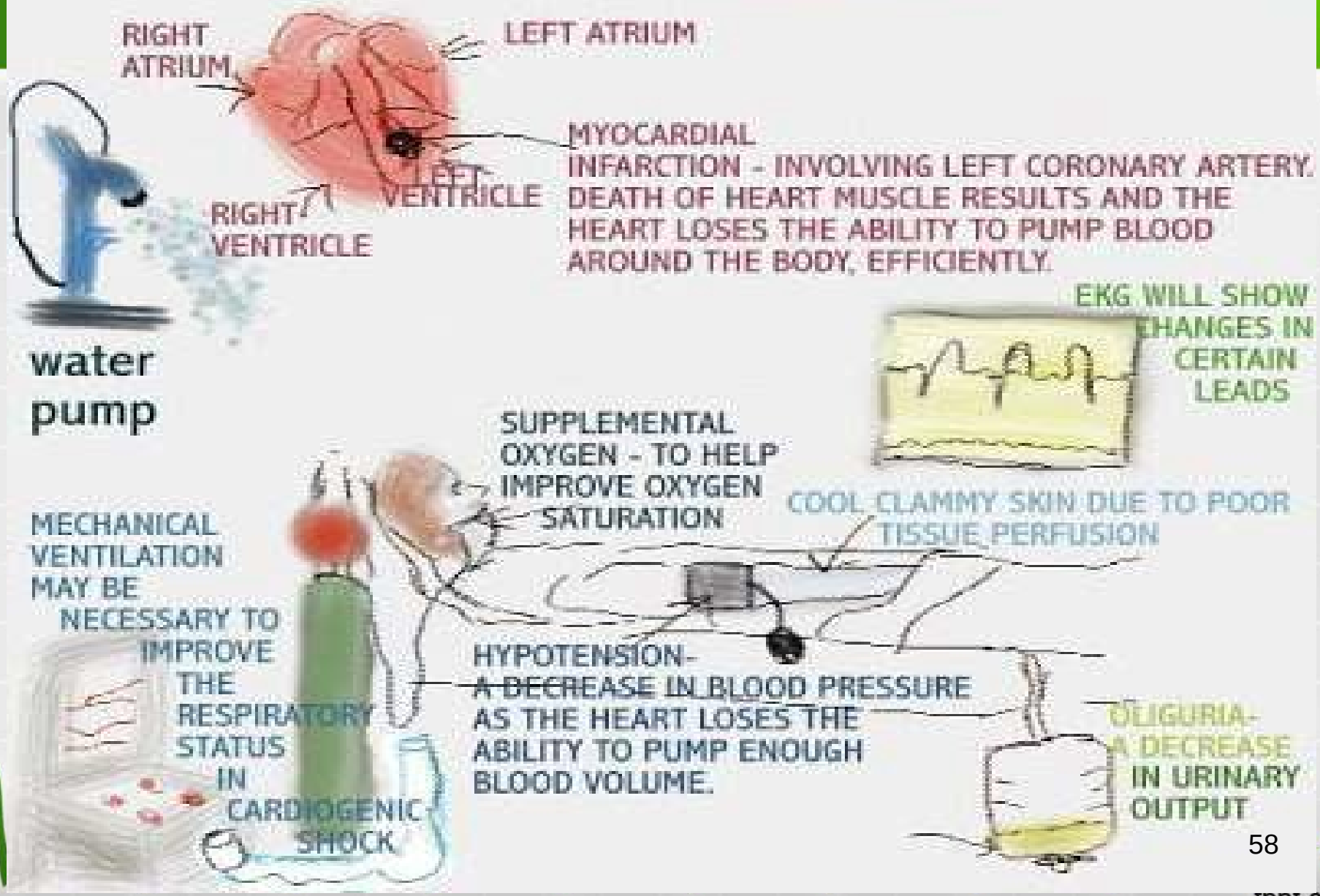
- Arises when heart is compressed from outside to decrease cardiac output , the cause may be
  * Tension pneumothorax
  * Pericardial tamponade
  * Diaphragmatic rupture with herniation of the bowel into the chest.

CLINICAL FEATURES

- Skin is pale & urine out put is low.
- Pulse becomes rapid & the systemic blood pressure is low.
- Right ventricular dysfunction, neck veins are distended & liver is enlarged.
- Left ventricular dysfunction , there are bronchial rales & third heart sound heard.
- Gradually, the heart also becomes enlarged.

SIMPLIFYING CARDIOGENIC SHOCK

THE CLINICAL PICTURE



MANAGEMENT

- Air way must be cleaned
- Initial measures include supplemental oxygen and, when systolic blood pressure permits, administration of i.v. nitroglycerin. Insertion of an intra-aortic balloon pump decreases ventricular after load, improving myocardial performance
- Revascularization with either angioplasty or bypass surgery have suggested improved survival
- Vasodilators

  Beta-Blockers

- Cardiogenic shock can also occur after prolonged cardiopulmonary bypass ; the stunned myocardium may require hrs or days to recover sufficiently to support circulation. Treatment consists of combination of inotropic agents
- In case of pulmonary embolus it should be treated with large doses of heparin, intravenously
- Pain ,if present should be controlled with sedatives like morphine
- Fulminant pulmonary edema should be controlled with diuretics.
- Drugs mainly employed are Inotrophic agents

EXTRACARDIAC OBSTRUCTIVE SHOCK

- Flow of blood is obstructed, which impedes circulation and can result in circulatory arrest
- Several conditions result in this form of shock

a. Cardiac tamponade

b. Constrictive pericarditis

c. Tension pneumothorax

d. Massive pulmonary embolism

Treatment

- Treatment of choice is pericardial drainage via surgery
- Pulmonary embolism is usually treated with systemic anticoagulation, but when massive pulmonary embolism causes right ventricular failure and shock, thrombolytic therapy should be strongly considered

NEUROGENIC SHOCK

- Primarily due to blockade of sympathetic nervous system à loss of arterial & venous tone with pooling of blood in the dilated peripheral venous system.
- The heart does not fill à the cardiac output falls.
- Neurogenic shock caused by: Paraplegia
  Quadriplegia.
  Trauma to Spinal cord.
  Spinal anesthesia.

CLINICAL FEATURES:

- Warm skin, pink & well perfused
- Heart rate is rapid
- Blood pressure is low
- Urine output may be normal

Pathophysiology

Dilatation of the systemic vasculature

Decreased systemic arterial pressure

Pooling of blood in systemic venules & small veins

The right heart filling & stroke volume decreases

Decreased pulmonary blood volume & left heart filling

Discharge of angiotensin & vasopressin though they fail to restore the cardiac output to normal

MANAGEMENT

1. Assuming Trendelenburg position—displaces blood from systemic venules into right heart & increases cardiac output.
2. Administration of fluids.
3. Vasoconstrictor drugs.

   Phenylephrine & Metaraminol

- Only type of shock safely treated with vasoconstrictor .Its prompt action saves patient from immediate damage to important organs like brain, heart & kidney.

VASOVAGAL / VASOGENIC SHOCK

- Part of neurogenic shock
- Pathophysiology : pooling of blood due to dilatation of peripheral vascular system particularly in the limb muscle & in splanchnic bed.
- This causes reduced venous return to the heart leading to low cardiac output & bradycardia, blood flow to brain is reduced causing cerebral hypoxia & unconsciousness.
- Management: Trendelenberg position-- increases cerebral flow & consciousness is restored

PSYCHOGENIC SHOCK

- Part of Neurogenic shock.
- Occurs following sudden fright from unexpected bad news or at the sight of horrible accident.
- Effect may vary in intensity from temporary unconsciousness to even sudden death.

SEPTIC SHOCK

- Most often due to gram-negative & gram-positive septicemia.
- It occurs in cases of,
  - -Severe septicemia
  - -Cholangitis
  - -Peritonitis
  - -Meningitis etc.
- The common organisms that are concerned with septic shock are E.coli, klebsiella, aerobactor, proteus, pseudomonas, bacteroides, etc

GRAM POSITIVE SEPSIS AND SHOCK

- It is usually caused by dissemination of a potent exotoxin liberated from gram positive bacteria without evidence of bacteremia.
- It is usually seen in Clostridium Tetany or Clostridium Perfringes infection.
- It is basically caused due to massive fluid losses.
- Arterial resistance falls but there is no fall in cardiac output.
- Urine output usually remains normal.

GRAM NEGATIVE SEPSIS AND SHOCK

- The most common cause of this infection is genito-urinary infection.
- Persons who have had operations of the genito-urinary tract are also susceptible.
- It may also be seen in patients who have undergone tracheostomy or those with gasterointestinal system infections.
- The severity may vary from mild hypotension to fulminating septic shock which has a poor prognosis.
- The prognosis is more favorable when the infection is accessible to surgical drainage.

- The clinical manifestations of septic shock may be fulminating and rapidly fatal. It is recognized initially by the development of chills & fever of over 100 degrees.
- Two types are clearly defined

  -Early warm shock.

  -Late cold shock.

EARLY WARM SHOCK

- In this type there is cutaneous vasodilatation.
- Toxins increase the body temperature. To bring this down vasculature of the skin dilates. This increases the systemic vascular resistance.
- Arterial blood pressure falls but the cardiac output increases, because the left ventricle has minimal resistance to pump against.
- Adrenergic discharge further Increases the cardiac output. The skin remains pink, warm & well perfused.
- The pulse is high & the blood pressure low.
- There are intermittent spikes of fever with bouts of chills.

LATE COLD SHOCK

- There is increased vascular resistance due to release of toxic products.
- This leads to hypovolemia with decrease in right heart filling.
- There is decreased flow to pulmonary vasculature so the left heart filling & the cardiac output decreases.
- The knowledge of existence of a septic focus is the only factor that differentiates septic shock from traumatic & hypovolemic shock.

Treatment:

- The only way to reduce mortality in septic shock is by prompt diagnosis & treatment.
- It can be divided into two groups.

ü Treatment of the infection.

ü Treatment of the shock.

- Therapy of septic shock has 3 main components
- 1st, the nidus of infection must be identified and eliminated
- 2nd, adequate organ system perfusion and function must be maintained, guided by cardiovascular monitoring.
- Maintenance of blood Hb level, O_2 saturation are imp therapeutic guidelines.
- 3rd therapeutic goal is to interrupt the pathogenic sequence leading to septic shock, achieved by inhibiting toxic mediators such as endotoxin, TNF, and IL-1.

- It consists of:

ü Fluid replacement.

ü Debridement & drainage of the infection.

ü Administration of the antibiotics.

ü Mechanical ventilation.

ü Steroids.

ü Vasoactive drugs.

ü Specific gamma globulins to bind the endotoxins.

ü The antibiotic polymixin E also absorbs some of the endotoxin.

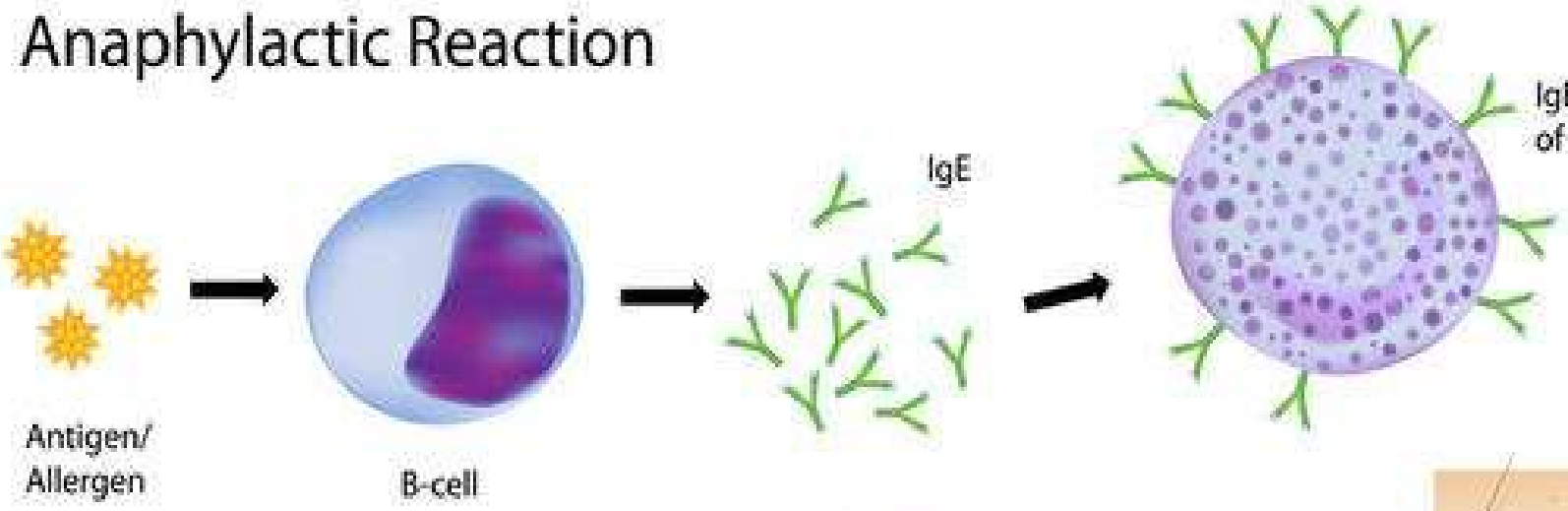
ANAPHYLACTIC SHOCK

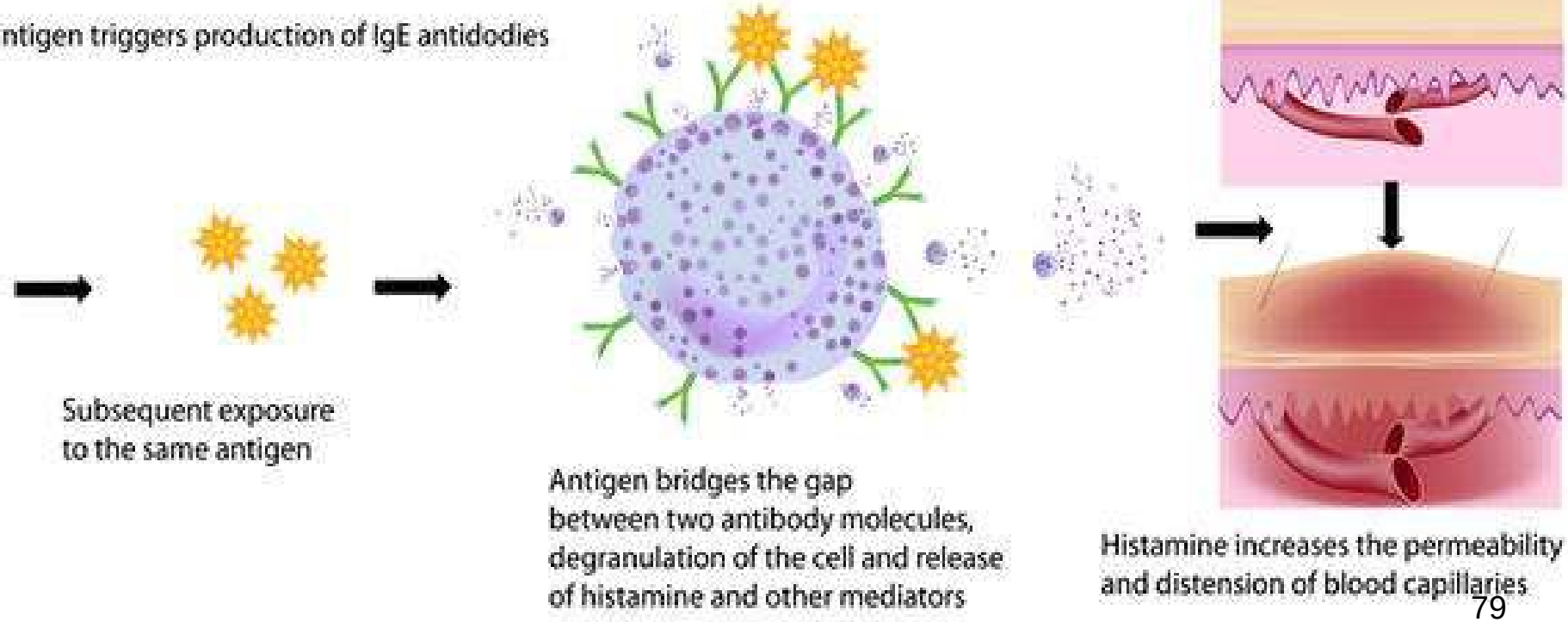
- The most common cause of anaphylaxis is the administration of penicillin.
- The other causes include anesthesia, dextrans, serum injections, stings, consumption of shell fish.

- The antigen combines with Ig E on the mast cell & basophils releasing large amounts of histamine and slow releasing substances of anaphylaxis.

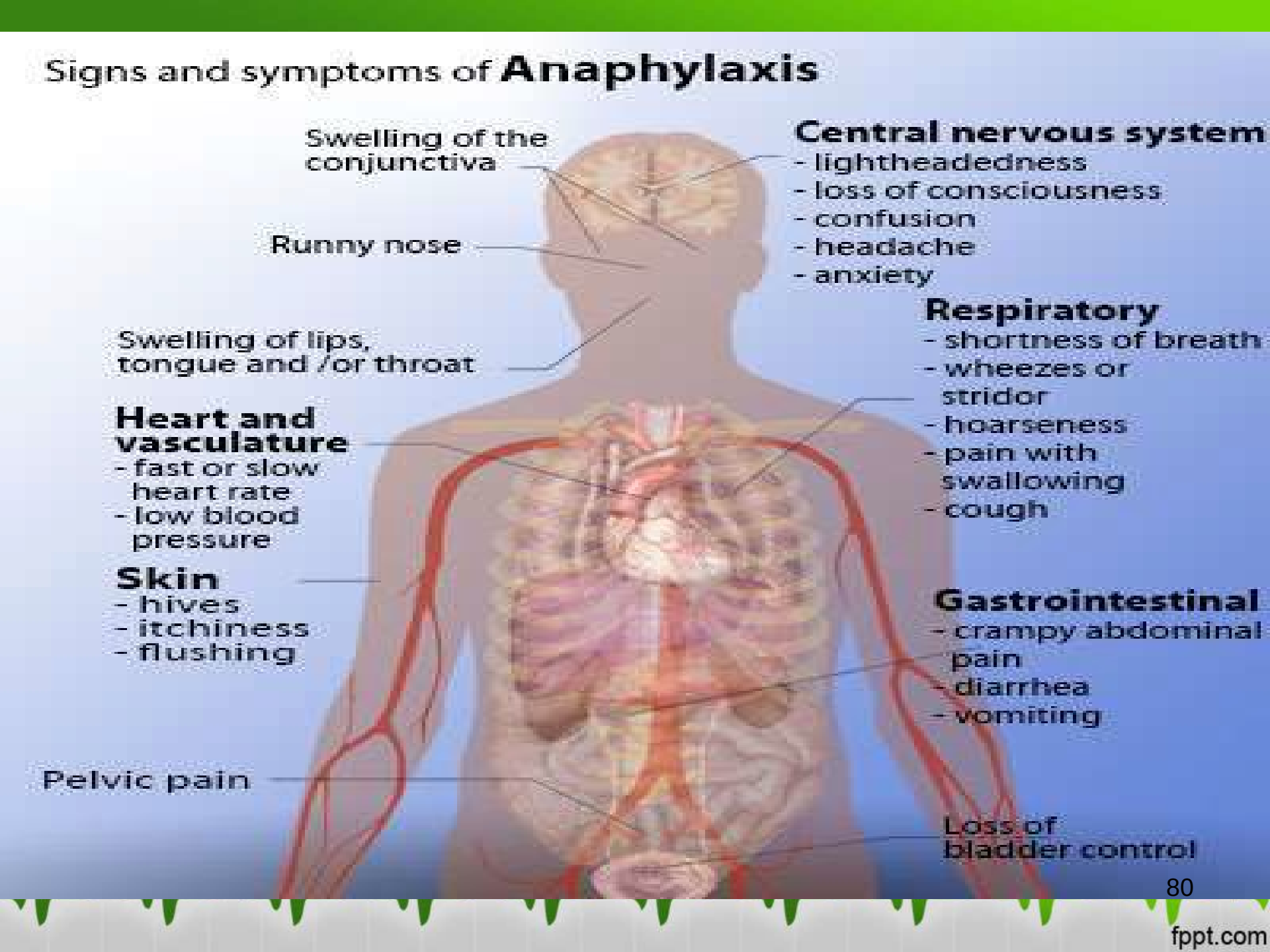
- It manifests as bronchospasm, laryngeal edema, respiratory distress, hypoxia, massive vasodilatation, hypotension and shock.
- The mortality rate is 10%.
- In the dental office this reaction can occur during or immediately following the administration of penicillin or LA to a previously sensitized patient.

Anaphylactic Reaction





Signs and symptoms of **Anaphylaxis**



Management

- Immediate & aggressive management is imperative if the patient is to survive.

  Position the patient

  Place the patient in a supine position with the legs slightly elevated.

  A-B-C

  Open the airway by tilting the head. Breathing & circulation should be established carrying BLS as needed.

  Definitive care

  As soon as a systemic allergy is suspected emergency medical help is sought.

(A) **Administration of epinephrine subcutaneously**

- **0.3ml of 1:1000 for adults, 0.15 for children,0.075ml for infants.**
- **With decreased perfusion the absorption of epinephrine will be delayed.**
- **In such situations it can be administered sublingually or intralingually.**
- **If the respiratory or cardiovascular regions fail to improve within 5 minutes of administration, a 2nd dose should be given.**
- **Subsequent doses can be given away 5-10 minutes as needed provided the patient is properly monitored.**

(B) **Administration of oxygen**

- **Deliver oxygen at a flow of 5-6 liters per minute by nasal hood or full face mask at any time during the episode.**

(C) **Monitoring of vital signs**

- **Monitoring the patients cardiovascular & respiratory status continuously.**
- **Record blood pressure & carotid heart rate at least every 5minutes & start closed chest compression if cardiac arrest occurs.**

(D) Additional drug therapy

- **After the administration of epinephrine, the other drugs to be administered are : Antihistamines, Corticosteroids.**
- **These drugs are administered only after clinical improvement is noted & are not be given during the acute phase as they are too slow in onset.**

PROGNOSIS OF SHOCK

- The prognosis varies with the origin of shock and its duration.
- 80%-90% of young patients survive hypovolemic shock with appropriate management.
- Cardiogenic shock associated with extensive myocardial infraction : (mortality rate up to 75%)
- Septic shock : (mortality rate up to 75%)

- Hypovolemic, anaphylactic and neurogenic shock are readily treatable and respond well to medical therapy.
- Perfusion of the brain may be the greatest danger during shock.
- Therefore urgent treatment is essential for a good prognosis

DENTAL CONSIDERATION IN SHOCK

1. Through diagnosis and treatment plan

- Allergies
- Systemic review of the patient

2.Local anesthesia used during dental treatment

3. Pain and anxiety.

4.Anxiety by the vision and perception of bigger and larger instrument in periodontology, oral surgery etc.

5. Shock (or) Syncope due to longer duration of treatment.

MANAGEMENT IN DENTAL OFFICE

MANAGEMENT OF DELAYED ONSET, ALLERGIC SKIN

↓

TERMINATE DENTAL PROCEDURE

↓

POSITION THE PATIENT

↓

BASIC LIFE SUPPORT AS INDICATED

↓

DEFINITIVE MANAGEMENT

OBSERVE PATIENT

ADMINISTER ORAL ANTIHIATAMINES

ADMINISTER I.M.+ORAL ANTIHISTAMINES E EVERY 4-6 HOURS

↓

MEDICAL CONSULTATION

NO SIGNS AND SYMPTOMS OF ALLERGY

↓

TERMINATE DENTAL PROCEDURE

↓

POSITION THE PATIENT(SUPINE WITH LEGS ELEVATED)

↓

BASIC LIFE SUPPORT AS INDICATED

↓

SUMMON MEDICAL ASSISTANCE

↓

ADMINISTER OXYGEN

↓

MONITOR VITAL SIGNS

↓

DEFINITIVE MANAGEMENT

MANAGEMENT OF GENERALISED ANAPHYLAXIS (SIGNS AND SYMPTOMS OF ALLERGY)

↓

TERMINATE DENTAL PROCEDURE

↓

POSITION THE PATIENT (SUPINE WITH LEGS ELEVATED)

↓

BASIC LIFE SUPPORT AS INDICATED

↓

SUMMON MEDICAL ASSISTANCE

↓

ADMINISTER EPINEPHRINE (SC, IM, IV)

↓

ADMINISTER OXYGEN

↓

MONITOR VITAL SIGNS

↓

ADDITIONAL DRUGS; ANTI HISTAMINES, CORTICOSTEROIDS

CONCLUSION

- Shock can present as a consequence of multiple causes & affect the body at cellular, visceral & systemic levels.
- Regardless of source, the fundamental primary treatment of shock remains recognition & prompt fluid replacement.
- The search for the underlying cause of the shock is only initiated after stabilization.

Literature

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- Gonzalez, K. W., Desai, A. A., Dalton, B. G., & Juang, D. (2015). . (Journal of pediatric intensive care.)
- Suh, G. J. (2018). . Singapore: Springer Singapore, Imprint: Springer.

Questions for the next lecture

physiology of detoxification systems?

general laws of pharmacodynamics and pharmacokinetics of substances?

the concept of medicine and poison?