Shock, DIC, HIT, & Septic Shock

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Shock

Definition – Inadequate tissue perfusion
- Widespread oxygen, nutrients and cellular function inadequate

Shock is life-threatening

Progression of shock is neither linear nor predictable

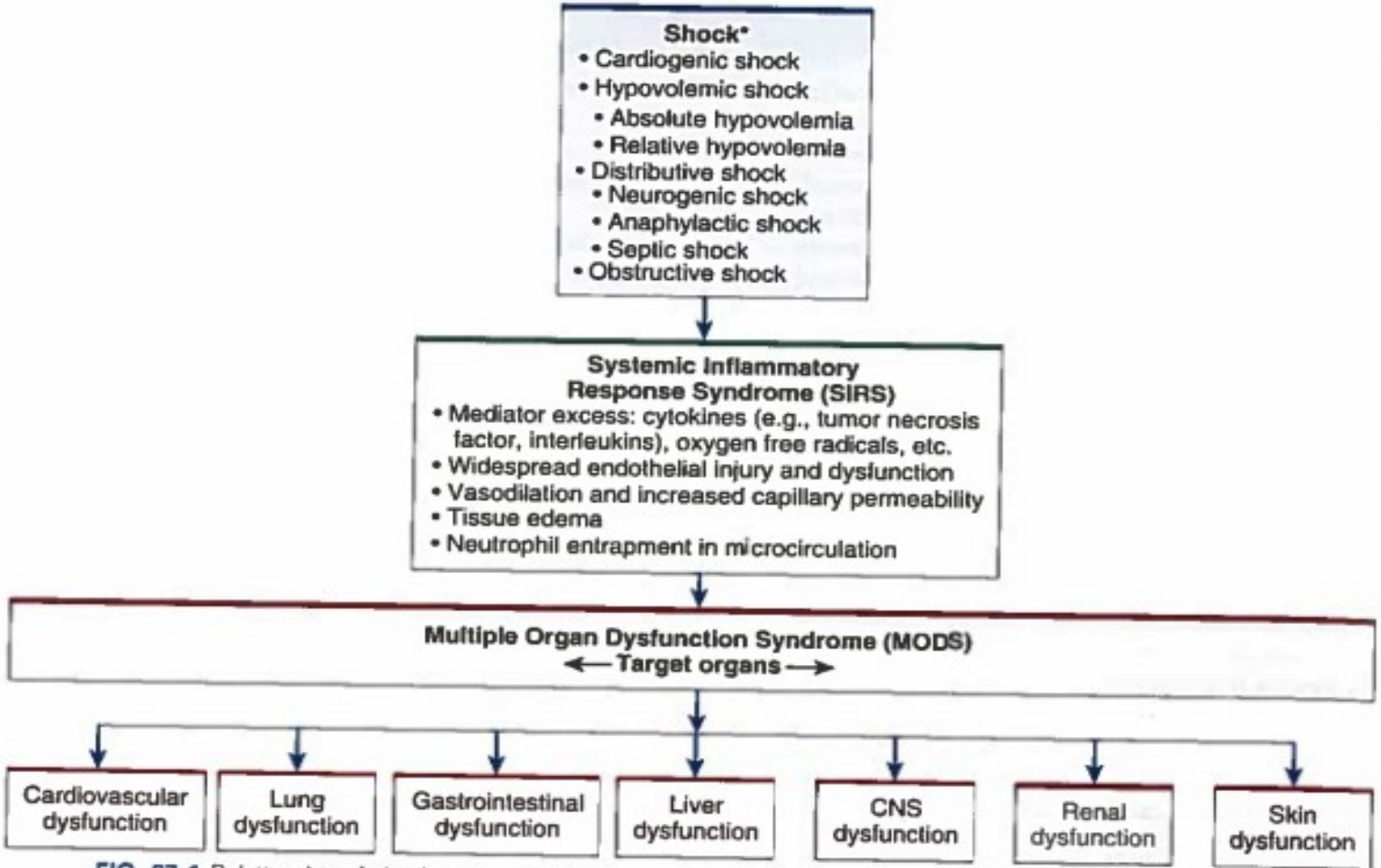


FIG. 67-1 Relationship of shock, systemic inflammatory response syndrome, and multiple organ dysfunction syndrome. *CNS*, Central nervous system. (See Table 67-1 for causes of shock states.)

Stages of Shock

Initial

Clinical findings

- No visible changes only cellular changes
- Production lactic acid

Stages of Shock

Compensatory stage

Clinical findings

- Blood pressure – begins to drop
- Heart rate – greater than 100 beats per minute
- Respiratory status – greater than 20 breaths per minute
- PaCo2 – less than 32 mm Hg
- Skin – Cold, clammy
- Urinary output – decreased
- Mentation – Confusion
- Acid-base balance – Respiratory alkalosis

Stages of Shock

Progressive

Clinical findings

- Blood pressure – Systolic less than 80 – 90 mm Hg
- Heart rate – greater than – 150 beats per minute
- Respirations – Rapid, shallow, crackles
- PaO2 – less than 80 mm Hg, PaCO2 greater than 45 mm Hg
- Skin – Mottled, petechiae
- Urinary output – 0.5 mL/kg/h
- Mentation – Lethargy
- Acid-base balance – Metabolic acidosis
- Hematologic dysfunction – risk of DIC

Stages of Shock

Refractory (Irreversible)

Clinical Findings

- Blood pressure – requires support
- Heart rate – erratic or asystole
- Respiratory status – requires support
- Urinary output – anuric
- Mentation – unconscious – cerebral ischemia
- Acid-base balance – profound acidosis

Calculating Mean Arterial Pressure

MAP = SBP + 2(DBP)/3

Example: Blood pressure 83 mmHg/50 mmHg

MAP = SBP + 2(DBP)/3

= 83 + 2(50)/3

= 83 + 100/3

= 183/3

MAP = 61 mmHg

SIGNS OF SHOCK

↓ In MAP (Mean Arterial Pressure)

EARLY SIGNS

Map is ↓ 10mm Hg from Baseline

Effective Compensation

O_2 → Vital Organs

Little ↑ Heart Rate

COMPENSATORY SIGNS
(NON PROGRESSIVE)

A ↓ in the MAP by 10-15mm Hg from Baseline

↑ Renin ↑ ADH

Vasoconstriction

↓ Pulse Pressure

↑ Heart Rate

↓ pH

Restless

Apprehensive

↑ K^+

PROGRESSIVE SIGNS
(INTERMEDIATE)

A sustained ↓ in the MAP that is > than 20mm Hg from the Baseline

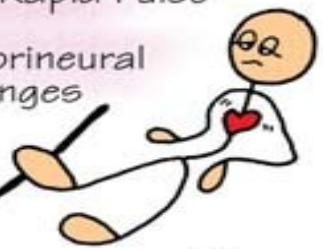
Tissue / Organ Hypoxia

↓ Urine (Oliguria)

Weak Rapid Pulse

↓ pH

Sensorineural Changes



REFRACTORY SIGNS
(IRREVERSIBLE)

Excessive Cell/Organ Damage

Multi System Organ Failure

↓ pH

CJMILLER

Concepts and Challenges

Monitoring tissue perfusion

Reducing anxiety

Promoting safety

Preventing complications

Promoting rest and comfort

Supporting family members

Management Strategies in Shock

Oxygen

Fluid replacement

Vasoactive medication therapy

Nutritional support

Nutritional Support

Meet increased metabolic energy requirements

Support with parenteral or enteral nutrition

GI system should be used to support its integrity

Administration of H2 blockers or proton-pump inhibitors

Fluid Replacement

Normal Saline – crystalloid (isotonic)

5%Dextrose – crystalloid (hypotonic)

Albumin – natural colloid

Dextran – artificial colloid

Classification of Shock

Hypovolemic shock

Cardiogenic shock

Distributive (Circulatory) shock
- Neurogenic shock
- Septic shock
- Anaphylactic shock

Obstructive shock

Hemodynamics in Shock

Physiologic variable	Preload (R)	Preload (L)	Pump function	Afterload	Tissue perfusion
Clinical measurement	**RAP/CVP**	**PCWP/LVEDP**	**Cardiac output/ index**	**SVR/TPR**	**MvO_2**
Hypovolemic · Hemorrhagic · Burns · Pancreatitis (3rd spacing)	↓	↓↓	↓	↑	↓
Distributive · Sepsis · Anaphylaxis · Addisonian crisis	↓	↓	↑	↓	↑
Cardiogenic					
LV Dysfunction · MI (LAD) · Acute myocarditis	↑	↑	↓	↑	↓
RVMI · RCA occlusion · Inferior and RV MI · Isolated RV dysfunction	↑	↓	↓	↑	↓
Obstructive					
Pulmonary Vascular · PE · Severe PH	↑	↓	↓	↑	↓
Mechanical · Pericardial tamponade · Tension pneumothorax · Constrictive pericarditis · Restrictive cardiomyopathy	↑	↑	↓	↑	↓

Hypovolemic Shock

Fluid and Blood replacement

Redistribution of fluid

Pharmacologic therapy

Administering blood and fluids safely

Implementing other measures – may need arterial line or pulmonary artery catheter to monitor pressures.

Cardiogenic Shock

First line treatment

- Oxygenation
- Pain control
- Monitoring
- Laboratory marker monitoring
- Pharmacologic therapy

Preventing cardiogenic shock

Medications and intravenous fluids

Distributive (Circulatory) Shock

ANAPHYLACTIC SHOCK RISK FACTORS

Penicillin sensitivity

Transfusion reaction

Bee sting allergy

Latex sensitivity

Severe allergy to some foods or medications

Distributive (Circulatory) Shock

NEUROGENIC SHOCK RISK FACTORS

Spinal cord injury

Spinal anesthesia

Depressant action of medications

Glucose deficiency

Distributive (Circulatory) Shock

SEPTIC SHOCK RISK FACTORS

Inflammatory response to microorganism

Gram negative bacteria most common cause

Long term IV or urinary catheter use

Non-healing wounds

Extremes of age

Obstructive Shock

RISK FACTORS

Cardiac Tamponade

Tension pneumothorax

Pulmonary Embolism

TABLE 67-1 CLASSIFICATION OF SHOCK STATES

Types and Causes	Examples
Cardiogenic Shock	
• Systolic dysfunction: inability of the heart to pump blood forward	Myocardial infarction, cardiomyopathy, blunt cardiac injury, severe systemic or pulmonary hypertension, myocardial depression from metabolic problems
• Diastolic dysfunction: inability of the heart to fill	Cardiac tamponade, ventricular hypertrophy, cardiomyopathy
• Dysrhythmias	Bradydysrhythmias, tachydysrhythmias
• Structural factors	Valvular stenosis or regurgitation, ventricular septal rupture, tension pneumothorax
Hypovolemic Shock	
Absolute Hypovolemia	
• External loss of whole blood	Hemorrhage from trauma, surgery, GI bleeding
• Loss of other body fluids	Vomiting, diarrhea, excessive diuresis, diabetes insipidus, diabetes mellitus
Relative Hypovolemia	
• Pooling of blood or fluids	Bowel obstruction
• Fluid shifts	Burn injuries, ascites
• Internal bleeding	Fracture of long bones, ruptured spleen, hemothorax, severe pancreatitis
• Massive vasodilation	Sepsis
Distributive Shock	
Neurogenic Shock	
• Hemodynamic consequence of spinal cord injury and/or disease at or above T5	Severe pain, drugs, hypoglycemia, injury
• Spinal anesthesia	
• Vasomotor center depression	
Anaphylactic Shock	
• Hypersensitivity (allergic) reaction to a sensitizing substance	Contrast media, blood or blood products, drugs, insect bites, anesthetic agents, food or food additives, vaccines, environmental agents, latex
Septic Shock	
• Infection	Pneumonia, peritonitis, urinary tract, invasive procedures, indwelling lines and catheters
• At-risk patients	Older adults, patients with chronic diseases (e.g., diabetes mellitus, chronic kidney disease, heart failure), patients receiving immunosuppressive therapy or who are malnourished or debilitated
Obstructive Shock	
• Physical obstruction impeding the filling or outflow of blood resulting in reduced cardiac output	Cardiac tamponade, tension pneumothorax, superior vena cava syndrome, abdominal compartment syndrome, pulmonary embolism

TABLE 67-3 CLINICAL PRESENTATION OF TYPES OF SHOCK

Cardiogenic Shock	Hypovolemic Shock	Distributive Shock: Neurogenic Shock	Distributive Shock: Anaphylactic Shock	Distributive Shock: Septic Shock	Obstructive Shock
Cardiovascular System					
Tachycardia ↓ BP ↓ Capillary refill Chest pain may or may not be present	↓ Preload ↓ Stroke volume ↓ Capillary refill	↓ BP ↓/↑ Temperature Bradycardia	Chest pain Third spacing of fluid	↓/↑ Temperature Myocardial dysfunction Biventricular dilation ↓ Ejection fraction	↓ BP ↓ Preload
Pulmonary System					
Tachypnea Crackles Cyanosis Rhonchi	Tachypnea → bradypnea (late)	Dysfunction related to level of injury	Shortness of breath Edema of larynx and epiglottis Wheezing Stridor Rhinitis	Hyperventilation Crackles Respiratory alkalosis → respiratory acidosis Hypoxemia Respiratory failure ARDS Pulmonary hypertension	Tachypnea → bradypnea (late) Shortness of breath
Renal System					
↑ Na^+ and H_2O retention ↓ Renal blood flow ↓ Urine output	↓ Urine output	Bladder dysfunction	Incontinence	↓ Urine output	↓ Urine output
Skin					
Pallor Cool, clammy	Pallor Cool, clammy	↓ Skin perfusion Cool or warm Dry	Flushing Pruritus Urticaria Angioedema	Warm and flushed → cool and mottled (late)	Pallor Cool, clammy
Neurologic System					
↓ Cerebral perfusion: • Anxiety • Confusion • Agitation	↓ Cerebral perfusion: • Anxiety • Confusion • Agitation	Flaccid paralysis below the level of the lesion Loss of reflex activity	Anxiety Feeling of impending doom Confusion ↓ LOC Metallic taste	Alteration in mental status (e.g., confusion) Agitation Coma (late)	↓ Cerebral perfusion: • Anxiety • Confusion • Agitation
Gastrointestinal System					
↓ Bowel sounds Nausea, vomiting	Absent bowel sounds	Bowel dysfunction	Cramping Abdominal pain Nausea Vomiting Diarrhea	GI bleeding Paralytic ileus	↓ to absent bowel sounds
Diagnostic Findings*					
↑ Cardiac markers ↑ b-Type natriuretic peptide (BNP) ↑ Blood glucose ↑ BUN ECG (e.g., dysrhythmias) Echocardiogram (e.g., left ventricular dysfunction) Chest x-ray (e.g., pulmonary infiltrates)	↓ Hematocrit ↓ Hemoglobin ↑ Lactate ↑ Urine specific gravity Changes in electrolytes		Sudden onset History of allergies Exposure to contrast media	↑/↓ WBC ↓ Platelets ↑ Lactate ↑ Blood glucose ↑ Urine specific gravity ↓ Urine Na^+ Positive blood cultures	Specific to cause of obstruction

*Also see Table 67-2.
ARDS, Acute respiratory distress syndrome; *BUN*, blood urea nitrogen; *LOC*, level of consciousness;

Intervention Goals

Control of underlying cause

Rapid correction of hypoperfusion

Halt oxygen debt accumulation

Repay oxygen debt

Careful monitoring of organ function

Summary

Resuscitative efforts must focus on stopping development of cellular dysfunction resulting in the downward spiral to death.

Disseminated Intravascular Coagulopathy (DIC)

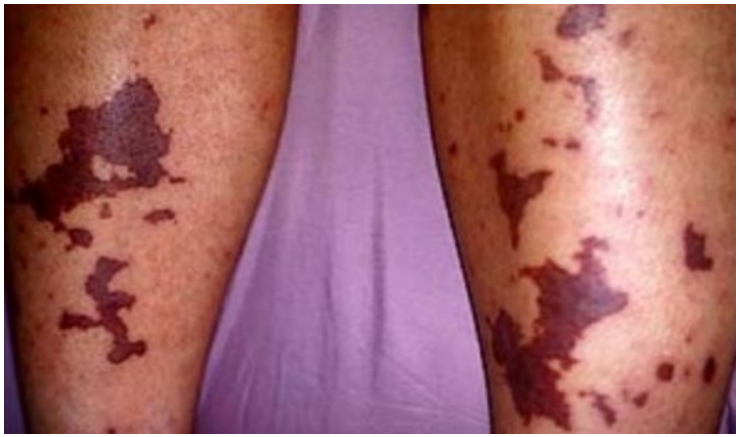
CONSUMPTIVE COAGULOPATHY

Disseminated Intravascular Coagulopathy(DIC)

Over stimulation of bleeding & clotting

Secondary to underlying disease/condition such as:

- Shock
- Major trauma
- Tissue damage: crush injuries, burns, severe head injury, snakebites
- Malignancy
- OB complications
- *Sepsis*



Normal Clotting Process

Endothelial Injury

↓

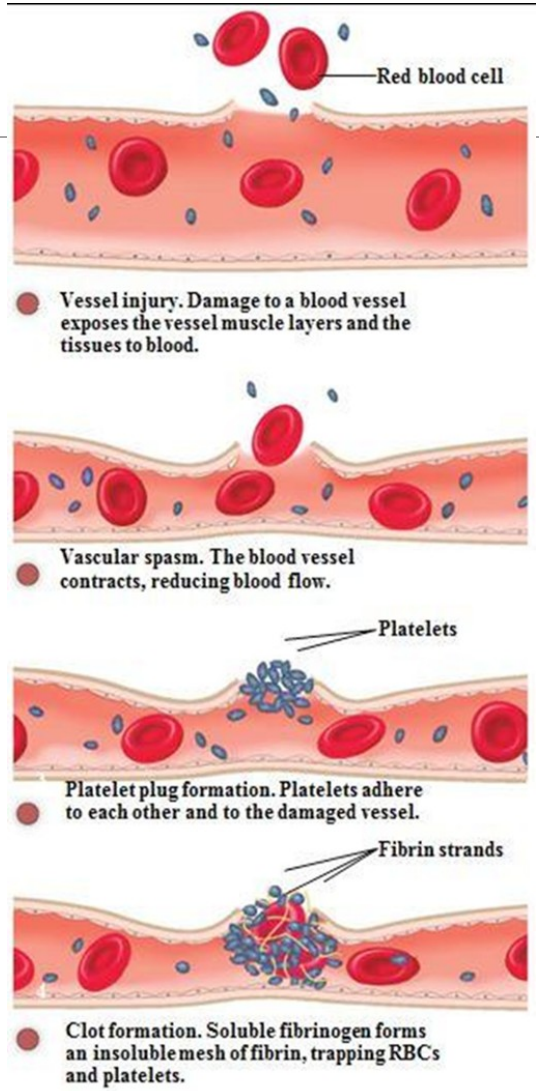
Platelet Plug

↓

Blood clot (Fibrin)

↓

Fibrinolysis & tissue remodeling



DIC Pathophysiology

Tissue damage

Clotting stimulated

Microvascular thrombi (everywhere in the body)

Fibrinolytic mediators are released

Chaos occurs

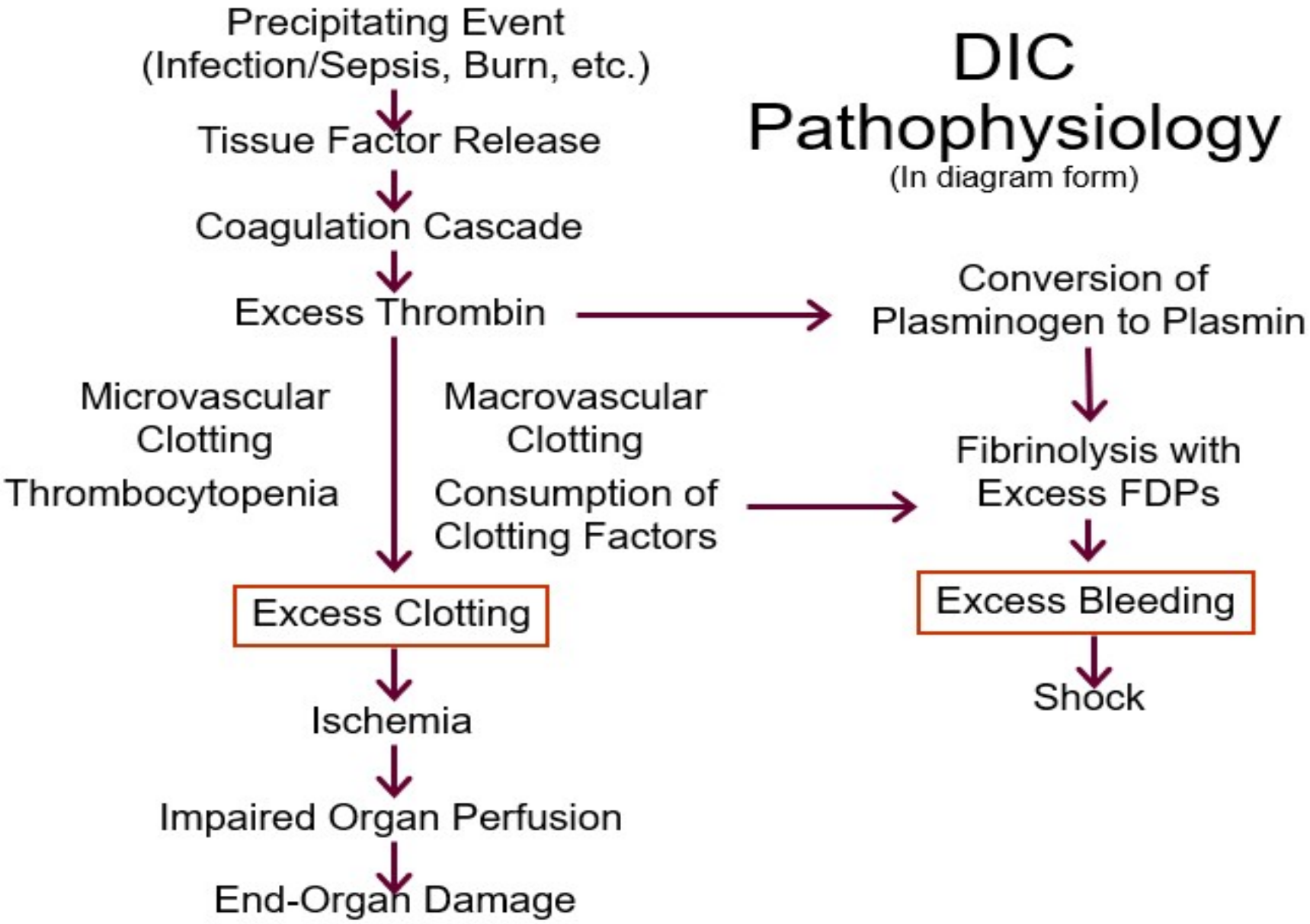
Lysis of clots, **consumption** of clotting factors

Ability to clot is lost

Bleeding occurs

DIC Pathophysiology

(In diagram form)



DIC Signs/Symptoms

Excessive Bleeding:

- Bleeding from various sites
- Hematuria
- Petechial rashes
- Oozing from IV sites
- GI bleeding
- Gingival oozing

Box 65-2 pg 1228 of NCLEX Book

Excessive Clotting:

- Thrombosis
- Gangrene
- Altered LOC, CVA
- SOB, PE
- Bowel ischemia/ infarction
- Acute renal failure

DIC: Labs

Massive Intravascular Clotting & 2ndary Depletion of Essential Clotting Factors

Platelet Count	Decreased
Fibrinogen Level	Decreased
Prothrombin Time (PT)	Prolonged
Partial Thromboplastin Time (PTT)	Shortened
	Prolonged
Protein C Level	Decreased

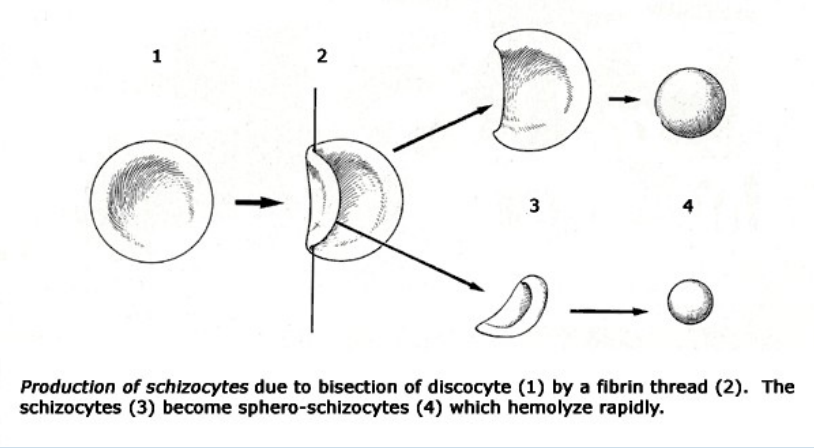
DIC: Labs

Excessive / Accelerated Fibrinolysis:

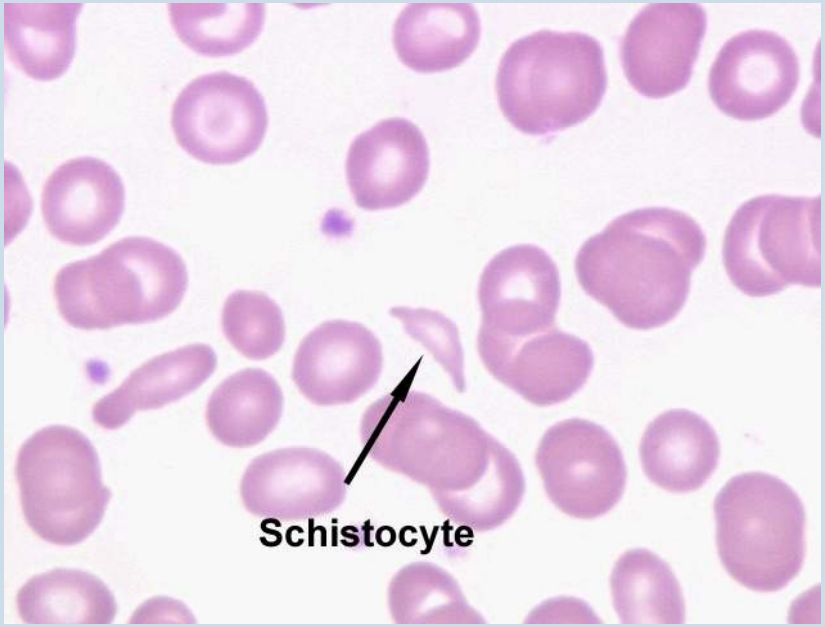
Fibrin Degradation or Split Products (FDPs)	Increased
D-dimer Assays	Increased
Antithrombin III Level	Decreased

DIC: Labs Continued

Schistocytes present in peripheral smear



Production of schizocytes due to bisection of discocyte (1) by a fibrin thread (2). The schizocytes (3) become sphero-schizocytes (4) which hemolyze rapidly.



Assessment

Early detection of bleeding
- Petechiae
- Oozing at IV or injection sites
- Increased heart rate
- Changes in mental status
- Increased abdominal girth
- Pain

Early detection of clotting
- Decreased renal output
- Decreased blood flow to extremities
- Cyanotic nail beds
- Pain

*General Assessment:
Monitor I&O, watch for renal failure (clotted arteries), pulmonary embolism, cerebrovascular accident, and acute respiratory distress syndrome

DIC Treatment

Early recognition

Treat underlying cause

Optimal oxygen delivery

Replacement of clotting factors

Prevention of further complications

- Such as hypovolemic shock, cardiac arrest, organ damage, limb loss

DIC Treatment continued

In Severe Bleeding:

- Cryoprecipitate
- Fresh frozen plasma
- Platelet transfusions
- RBC transfusion

Heparin Infusion:

- Interrupts thrombosis process
- Controversial

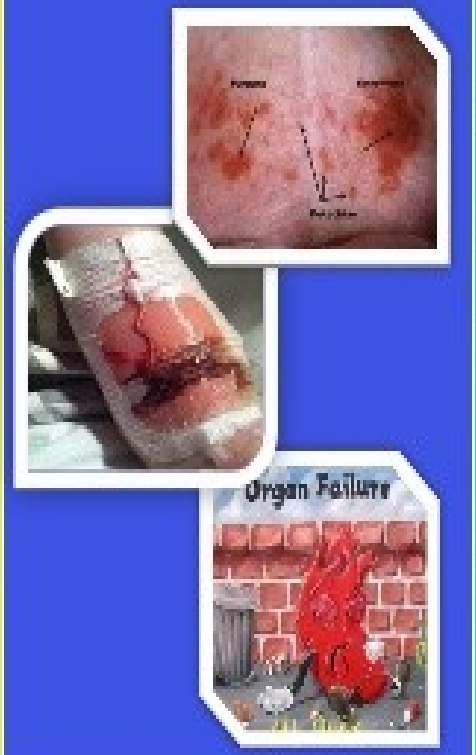
Disseminated intravascular coagulation (DIC)

Pathophysiology

- Hyper-activated coagulation system.
- Hyper-activated fibrin-lytic system, or both simultaneously.
- Coagulation factors and plts consumed as soon as they are made.
- Secondary to an underlying disease or condition. Ex; sepsis, placenta abruption, snake bites, toxin, trauma, graft vs. host disease, and burns.

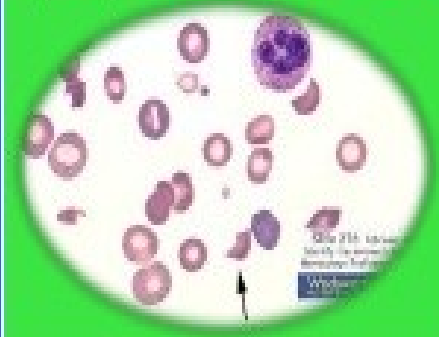
Clinical Finding

- Patients are at risk of bleeding and thrombosis.



Laboratory Finding

- Thrombocytopenia
- Prolonged PT, APTT, thrombin time.
- Decreased fibrinogen.
- Elevated D-dimers.
- Schistocytes on the peripheral blood smear.



Treatment of DIC

- Treatment of the underlying disorder .
- Transfusion support of Red Blood Cells or Fresh Frozen Plasma (FFP) to replace coagulation factors.

Heparin Induced Thrombocytopenia (HIT)

"ALLERGY TO HEPARIN"

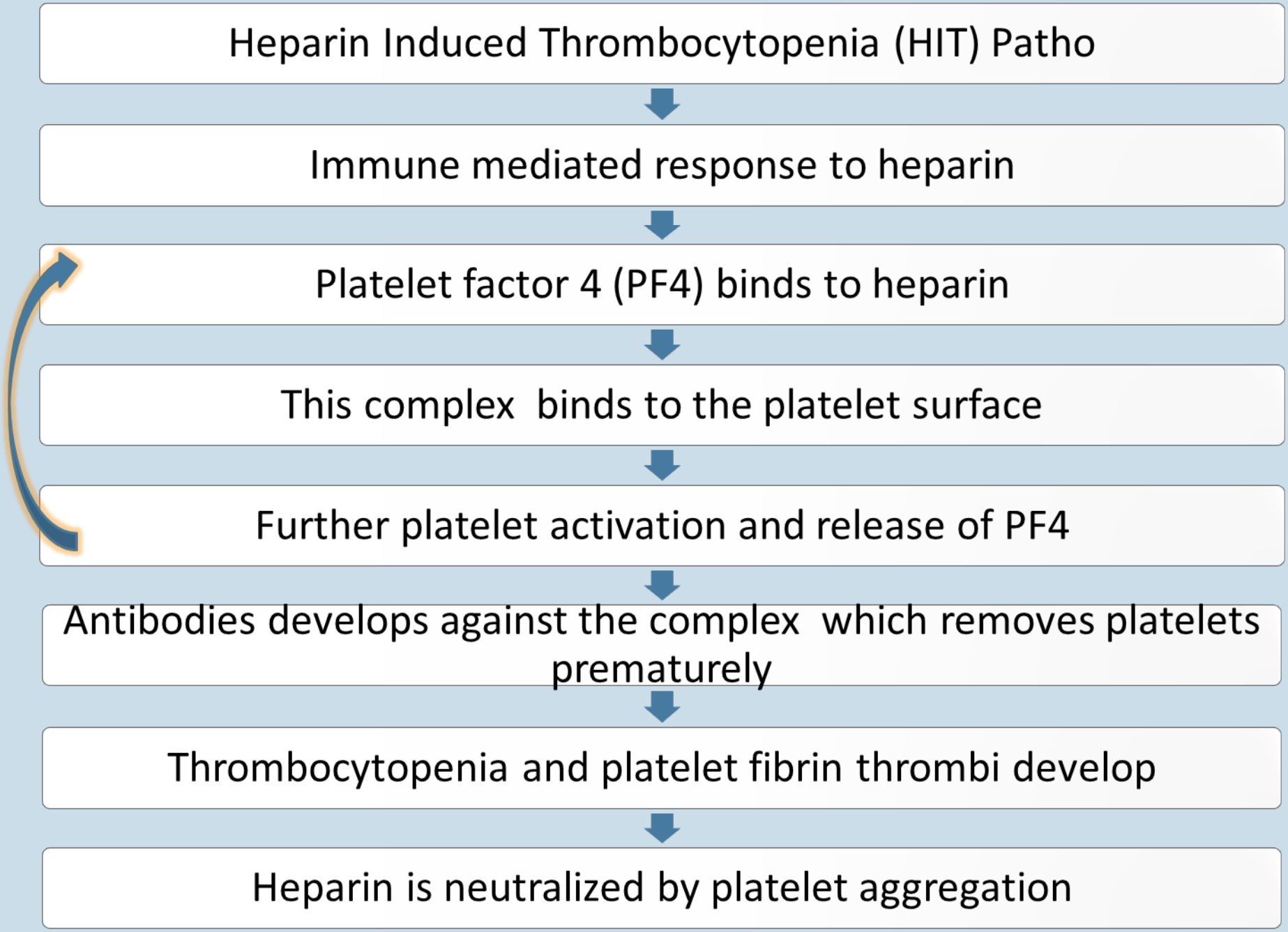
Heparin Induced Thrombocytopenia (HIT)

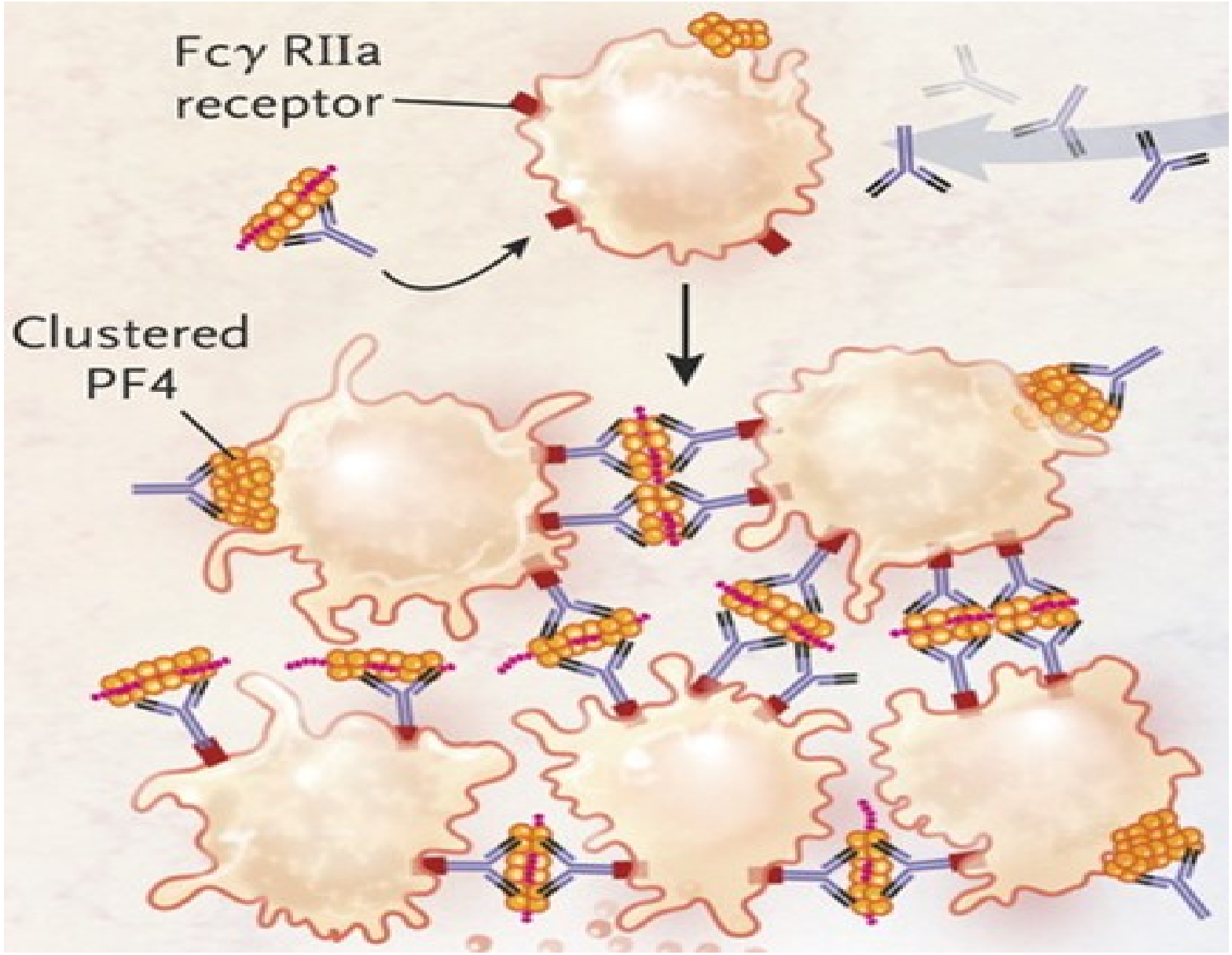
Develops 3 - 12 days after the onset of heparin therapy

Suspected - platelet count less than 100,000 or drops greater than 50%

Symptoms of bleeding *rare* – platelet count usually does not drop below 60,000

Major issue – venous and /or arterial thrombosis


Heparin Induced Thrombocytopenia (HIT) Patho
Immune mediated response to heparin
Platelet factor 4 (PF4) binds to heparin
This complex binds to the platelet surface
Further platelet activation and release of PF4
Antibodies develops against the complex which removes platelets prematurely
Thrombocytopenia and platelet fibrin thrombi develop
Heparin is neutralized by platelet aggregation


Fcγ RIIa
receptor
Clustered
PF4

Heparin Induced thrombocytopenia (HIT)

Clinical Manifestations

Petechiae

Bleeding post routine procedures

Vascular thrombosis

Diagnostic Studies

Medical history

Platelet count < 100,000

HIT Antibody test (PF4)

PT, PTT usually normal

Fibrin, FDP normal

Bone marrow aspiration

Heparin Induced Thrombocytopenia (HIT) Treatment

Avoid use of heparin

- ⊘ Heparin drips (IV)
- ⊘ Lovenox (SQ)
- ⊘ Heparin flushes

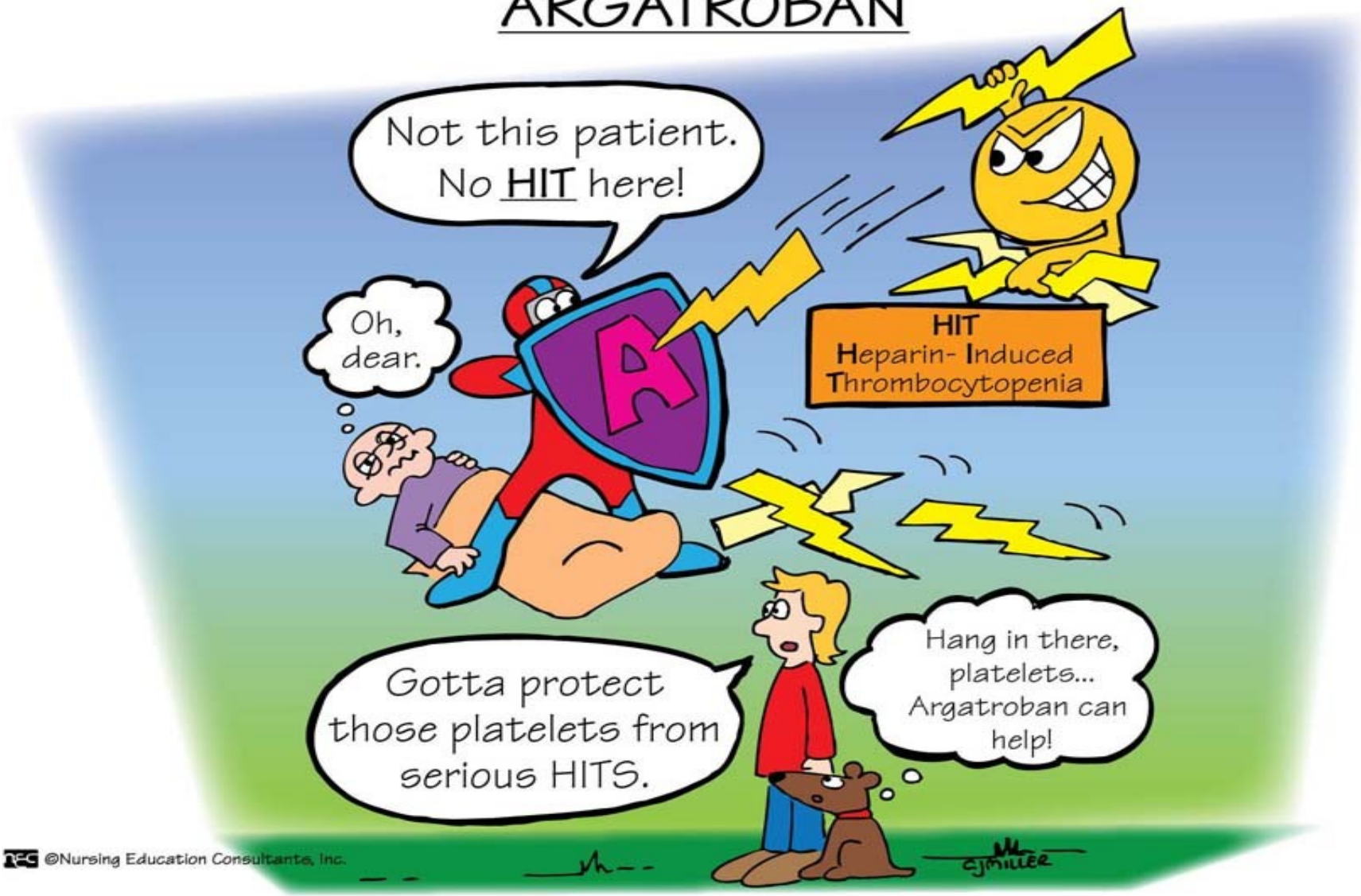
Anticoagulants

- Non heparin anticoagulant
- Argatroban (Acova):
  - A direct thrombin inhibitor

Monitor:

- Signs of Thrombosis
- Platelet Count
- Administer platelets ONLY if absolutely needed

ARGATROBAN


Not this patient. No HIT here!
Oh, dear.
A
HIT
Heparin- Induced Thrombocytopenia
Gotta protect those platelets from serious HITS.
Hang in there, platelets... Argatroban can help!
©Nursing Education Consultants, Inc.

Sepsis Pathophysiology

Bacteria. Fungi. Virus

↓

Inflammatory Response.
Release of chemical mediators.

↓ ↓

Endothelial damage

↑ Vascular permeability

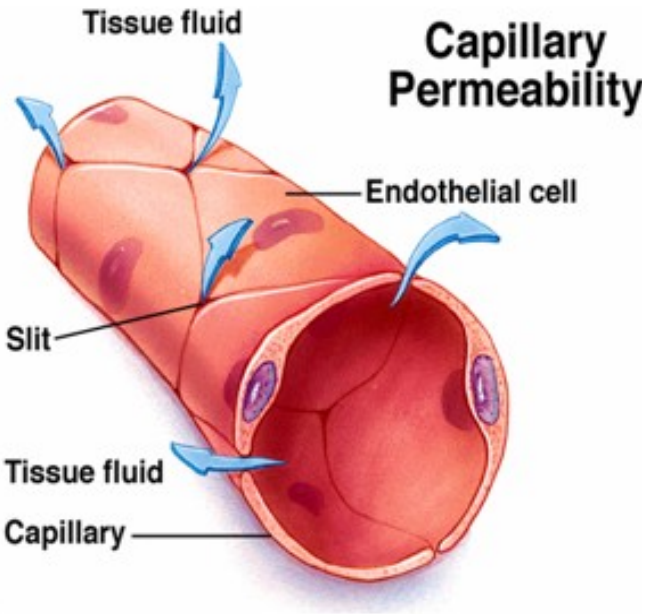
↓ ↓

Stimulation of coagulation. DIC.

Vasodilation.
Peripheral edema.
Hypotension.

↓ ↓

Tissue damage.
Organ failure. Decreased immune function.



<u>Clinical Manifestations:</u>
Tachypnea. Tachycardia. Fever. Leukocytosis, followed by leukopenia.

Example of SEPSIS Screening & Treatment in an ED

EXCLUSIONS
- Patient expires or is placed on comfort measures only within 3 hours of triage for severe sepsis
- Patient expires or is placed on comfort measures only within 6 hours of triage for septic shock
- Patient or surrogate refuses blood draws, fluids, or antibiotics
- Patient is a transfer from another hospital or ambulatory surgery center

A

Does the patient have an infection or suspected infection?

Screen for Sepsis

YES

Does the patient meet 2 or more SIRS criteria?
- Temp > 38.3˚C or < 36.0˚ C
- Heart Rate > 90
- Respiratory Rate > 20
- WBC > 12K or < 4K or > 10% Bands

YES

Patient has SEPSIS

Screen for Severe Sepsis

B

Is there at least 1 sign of acute organ dysfunction?
- SBP < 90 or MAP < 65
- A decrease in SBP > 40 points
- Creatinine > 2.0
- Urine output < 0.5 mL/kg/hr for 2 hrs
- Bilirubin > 2
- Platelet count < 100K
- INR > 1.5 or aPTT > 60 sec
- Lactate > 2

YES

Date/Time of triage (Time Zero)

Patient has SEVERE SEPSIS

3-HOUR and 6-HOUR TIMERS START FROM TIME OF TRIAGE

COMPLETE WITHIN 3 HOURS OF TRIAGE

Date/Time lactate drawn
Lactate level
Date/Time blood cultures drawn

- ❑ Administer crystalloid fluid bolus
- ❑ Draw lactate
- ❑ Draw blood cultures

Date/Time antibiotic administered
Antibiotic selection

- ❑ Administer appropriate broad-spectrum IV antibiotic (see list)

C

Is the patient still hypotensive despite initial fluid bolus?
OR
Is the lactate > 4?

YES

Patient has SEVERE SEPSIS WITH SEPTIC SHOCK

Date/Time fluid bolus initiated

- ❑ Administer 30 cc/kg crystalloid fluid bolus

COMPLETE WITHIN 6 HOURS OF TRIAGE

1

- ❑ Assess BP within 1 hr of completion of bolus

Is patient still hypotensive?

YES

Date/Time vasopressor initiated

- ❑ Start vasopressor (see list)

2

Date/Time of EACH of the items below

Re-assess volume status and tissue perfusion

MD/APN/PA evaluate all of the following
- Vital signs
- Cardiopulmonary status
- Capillary refill
- Peripheral pulses
- Skin

OR

Complete any 2 of the following:
- CVP measurement
- SvO2 measurement
- Bedside cardiovascular ultrasound
- Passive leg raise by MD/APN/PA or fluid challenge

3

Date/Time repeat lactate drawn

Repeat lactate level if initial lactate was > 2.0

SEPSIS + Septic Shock

The Plague Doctor
ILLUSTRATIONS FOR MEDICAL EDUCATION
OFFICIAL BLOG: THEPLAGUEDOCTOR.BLOG
FACEBOOK.COM/THEPLAGUEMD
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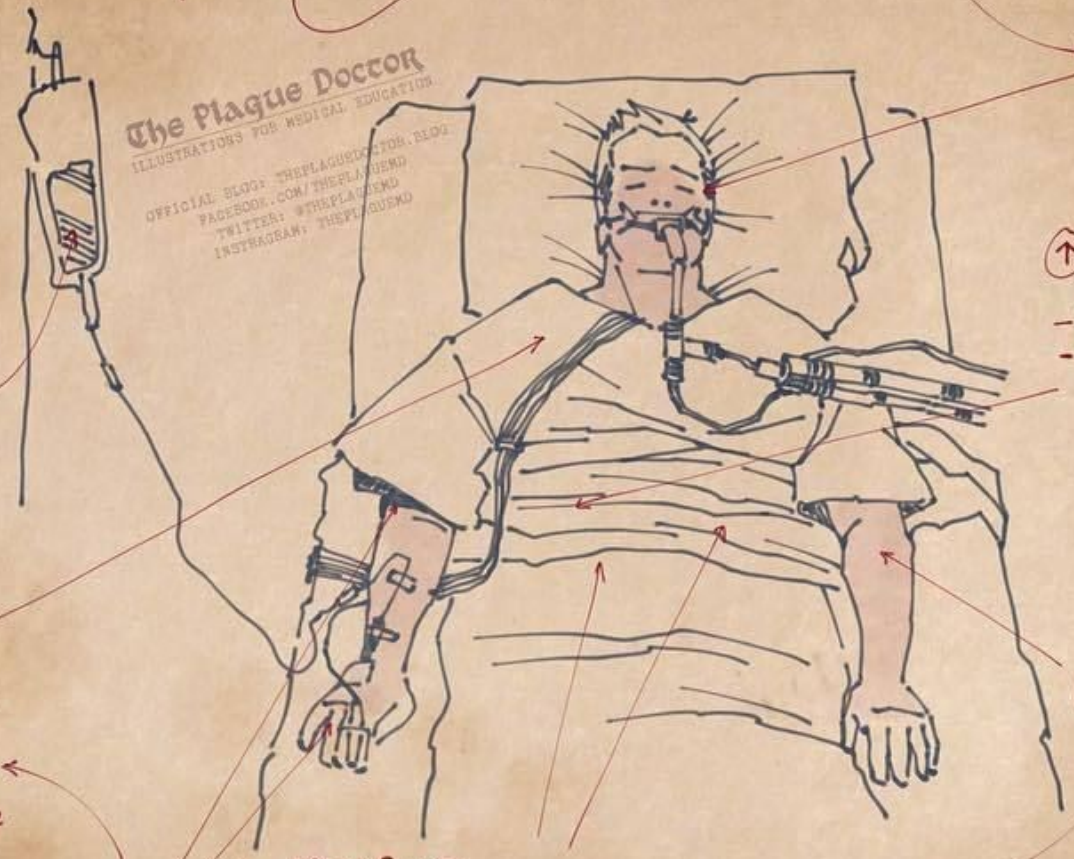
INSTRAGRAM: THEPLAGUEMD

Bacteria { exotoxins, endotoxins } > Bloodstream { "Bacteremia" } → Immune Response → humoral / celular { Interleukins, TNFα, Prostaglandin E2 } { Systemic Inflammation }



MANAGEMENT

1. ABCDE { life support }
2. fluids
3. vasopressors { ↑ BP }
4. Antibiotics
5. Corticosteroids (?)
6. Organ function Telemetry.

Hypothalamus
↓
Change in basal temperature

- ↑ temperature production
  - Tremors,
  - Protein synthesis
    - C-Reactive P.
    - Fibrinogen
- ↓ temperature loss.
  - Peripheral vasoconstriction (pallor, coldness).

→ FEVER!

↑ NITRIC OXIDE → VASODILATION
↓
↓ Blood Pressure
↙ Tachycardia (compensation) → Hypotension
↘

VASODILATION.
transudate to extravascular space
↙ Hypovolemia
↘ Edema

ORGAN FAILURE
- Heart failure – Pulm. edema.
- Resp. failure – ARDS
- Renal failure – ↑ Creat. Urea
- Altered mental Status.

↓ tissue perfusion (tissue hypoxia)
↳ Ischemia Inflammation

- Hypovolemia
- Hypotension

Tissue Hypoxia
↑ Pirvate
↳ ↑ Lactate
↓
ACIDOSIS
↓pH ↓HCO_3 ↑pCO_2

https://med.stanford.edu/septris/