Shock
Types, recognition and therapy

Dr . Moloud Balafar

Assistant Professor of Emergency Medicine
Medical University of Tabriz

SHOCK SYNDROME

Shock is a condition in which the cardiovascular system fails to perfuse tissues adequately

• An impaired cardiac pump, circulatory system, and/or volume can lead to compromised blood flow to tissues

• Inadequate tissue perfusion can result in:

– generalized cellular hypoxia (starvation)

– widespread impairment of cellular metabolism

– tissue damage organ failure

– death

PATHOPHYSIOLOGY

Cells switch from aerobic to anaerobic metabolism
lactic acid production
Cell function ceases & swells
membrane becomes more permeable
electrolytes & fluids seep in & out of cell
Na+/K+ pump impaired
mitochondria damage
cell death

COMPENSATORY MECHANISMS: Sympathetic Nervous System (SNS)-Adrenal Response

SNS - Neurohormonal response
Stimulated by baroreceptors
- ☐Increased heart rate
- ☐Increased contractility
- ☐Vasoconstriction (SVR-Afterload)
- ☐Increased Preload

COMPENSATORY MECHANISMS: Sympathetic Nervous System (SNS)-Adrenal Response

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Sympathetic Nervous System (SNS)-Adrenal Response

- SNS - Hormonal: Renin-angiotension system

☐Decrease renal perfusion
☐Releases renin angiotension I
☐angiotension II potent vasoconstriction &
☐releases aldosterone adrenal cortex
☐sodium & water retentio

COMPENSATORY MECHANISMS: Sympathetic Nervous System (SNS)-Adrenal Response

- SNS - Hormonal: Antidiuretic Hormone

☐Osmoreceptors in hypothalamus stimulated

☐ ADH released by Posterior pituitary gland

☐Vasopressor effect to increase BP

☐Acts on renal tubules to retain water

Failure of Compensatory Response

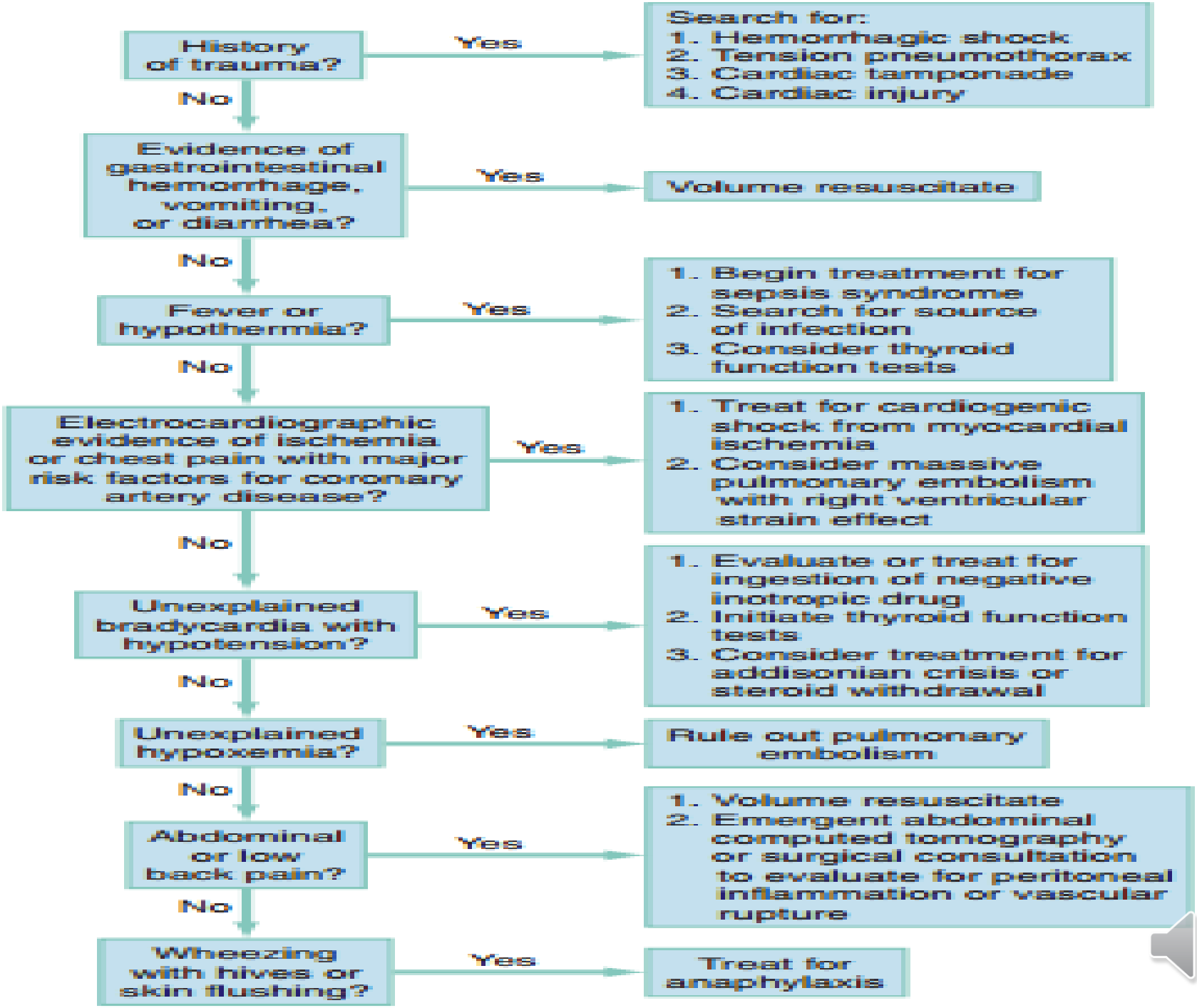
- **Decreased blood flow to the tissues causes cellular hypoxia**
- **Anaerobic metabolism begins**
- **Cell swelling, mitochondrial disruption, and eventual cell death**
- **If Low Perfusion States persists: IRREVERSIBLE DEATH IMMINENT!!**

Pathophysiology Systemic Level

- Net results of cellular shock:
  - **systemic lactic acidosis**
  - **decreased myocardial contractility**
  - **decreased vascular tone**
  - **decrease blood pressure, preload, and cardiac output**

Shock Syndromes

Hypovolemic Shock
–blood VOLUME problem
• **Cardiogenic Shock**
–**blood PUMP problem**
• **Distributive Shock**
[septic;anaphylactic;neurogenic]
–**blood VESSEL problem**


History of trauma?
Yes
Search for:
1. Hemorrhagic shock
2. Tension pneumothorax
3. Cardiac tamponade
4. Cardiac injury
No
Evidence of gastrointestinal hemorrhage, vomiting, or diarrhea?
Yes
Volume resuscitate
No
Fever or hypothermia?
Yes
1. Begin treatment for sepsis syndrome
2. Search for source of infection
3. Consider thyroid function tests
No
Electrocardiographic evidence of ischemia or chest pain with major risk factors for coronary artery disease?
Yes
1. Treat for cardiogenic shock from myocardial ischemia
2. Consider massive pulmonary embolism with right ventricular strain effect
No
Unexplained bradycardia with hypotension?
Yes
1. Evaluate or treat for ingestion of negative inotropic drug
2. Initiate thyroid function tests
3. Consider treatment for addisonian crisis or steroid withdrawal
No
Unexplained hypoxemia?
Yes
Rule out pulmonary embolism
No
Abdominal or low back pain?
Yes
1. Volume resuscitate
2. Emergent abdominal computed tomography or surgical consultation to evaluate for peritoneal inflammation or vascular rupture
No
Wheezing with hives or skin flushing?
Yes
Treat for anaphylaxis

Hypovolemic Sh

Loss of circulating volume "Empty tank " decrease tissue perfusion general shock response

- **ETIOLOGY:**

–Internal or External fluid loss

– Intracellular and extracellular compartments

- **Most common causes:**

☐**Hemmorhage**

☐**Dehydration**

Clinical Presentation Hypovolemic Shock

Tachycardia and tachypnea

- Weak, thready pulses
- Hypotension
- Skin cool & clammy
- Mental status changes
- Decreased urine output: dark & concentrated

Assessment & Management

S/S vary depending on severity of fluid loss:

- **15%[750ml]-** compensatory mechanism maintains CO
- **15-30%[750-1500ml**- Hypoxemia, decreased BP & UOP
- **30-40%[1500-2000ml]** -Impaired compensation &
profound shock along with severe acidosis
- **40-50%**- refactory stage:

loss of volume= death

Classification of shock

% blood volume lost	pulse	BP	UO / cons. level	therapy
0 – 15% 0-750	+	120/80	Normal/ agitated	nil
15 – 30% 750-1500	+++	110/90	agitated	i.v. fluids
30-40% 1500-2000	+++++	90/75	agitated	Fluids + blood
➢40% ➢> 2000	+++++++	palpable	obtund	Surgery + blood

Therapy of hypovolaemic shock

- Airway / breathing / C/spine control
- Stop all obvious haemorrhage
- Insert I.v. lines, take blood for X-match
- Give rapid bolus of fluid, then assess response
- Decide on need for surgery vs. decision to investigate

Resuscitation Endpoints

volume resuscitation:

1. CVP = 8-12 mm Hg
2. Wedge pressure = 10 to 12 mmHg
3. Cardiac index > 3 L/min/m2
4. Blood lactate < 4 mmol/L
5. Base deficit -2 to +2 mmol/L

Cardiogenic shock

Syndrome of inadequate tissue perfusion associated with normal circulating BV, and low cardiac output

- Symptoms: dyspnoea, poor exercise tolerance, confusion, sweating, PND
- Signs: tachycardia, cold skin, high JVP, added heart sounds, engorged liver, peripheral oedema

Cardiogenic Shoc

- **Assess for:**
  - **Blunt trauma to the chest**
  - **Cardiac tamponade**
  - **Cardiac dysrhythmias**

Core Skills Treat for Shock 28

- **Myocardial infarction**
- **Cardiac contusion**
- **Tachycardia**
- **Muffled heart sounds**
- **Engorged neck veins with hypotension**
- **Dyspnea**
- **Edema in feet and ankles**

Clinical Presentation Cardiogenic Shock

Pericardial tamponade
– muffled heart tones, elevated neck veins
• Tension pneumothorax
– JVD, tracheal deviation, decreased or absent unilateral breath sounds, and chest hyperresonance on affected side

Management Cardiogenic Shock

OPTIMIZING PUMP FUNCTION:

– **Pulmonary artery monitoring is a necessity !!**

– Aggressive airway management: Mechanical Ventilation

– Judicious fluid management

– Vasoactive agents

- **Dobutamine**
- **Dopamine**

Dopamine

Dopamine receptor activation at low doses-”splanchnic dilation” (2-5 mcg/kg/min)

- Beta receptor activation-increase cardiac output (5-10 mcg/kg/min)
- Alpha receptor activation-vasoconstriction (>10 mcg/kg/min)

Dobutamine

primarily a □1-receptor agonist (cardiac stimulation), but it also has mild □-2 effects (vasodilation)

- causes a dose-dependent increase in stroke volume
- decrease in cardiac filling pressures
- an alkaline pH inactivates catecholamines such as dobutamine
- Dose 2-20 mcg/kg/min

Dobutamine

dobutamine is the preferred inotropic agent for the acute management of low output states due to systolic heart failure. Because dobutamine does not usually raise the arterial blood pressure, it is **not indicated as monotherapy in patients with cardiogenic shock**

Norepinephrine

□-receptor agonist that promotes widespread vasoconstriction

- administration of any vasoconstrictor agent carries a risk of hypoperfusion and ischemia involving any tissue bed or vital organ
- Dose 2-20 mcg/min

Distributive Shock

Inadequate perfusion of tissues through maldistribution of blood flow

- Intravascular volume is maldistributed because of alterations in blood vessels
- Cardiac pump & blood volume are normal but blood is not reaching the tissues

Etiologies

- **Septic Shock (Most Common)**
- **Anaphylactic Shock**
- **Neurogenic Shock**

Anaphylactic Shock

A type of distributive shock that results from widespread systemic allergic reaction to an antigen

- This hypersensitive reaction is **LIFE THREATENING**

Pathophysiology Anaphylactic Shock

Antigen exposure
- body stimulated to produce IgE antibodies specific to antigen
  - **drugs, bites, contrast, blood, foods, vaccines**
- Reexposure to antigen
  - **IgE binds to mast cells and basophils**
- Anaphylactic response

Anaphylactic Response

Vasodilatation
- Increased vascular permeability
- Bronchoconstriction
- Increased mucus production
- Increased inflammatory mediators

recruitment to sites of antigen interaction

Clinical Presentation Anaphylactic Shock

Almost immediate response to inciting antigen

- Cutaneous manifestations
  - urticaria, erythema, pruritis, angioedema
- Respiratory compromise
  - stridor, wheezing, bronchorrhea, resp. distress
- Circulatory collapse
  - tachycardia, vasodilation, hypotension

Management Anaphylactic Shock
• Early Recognition, treat aggressively

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•AIRWAY SUPPORT
• IV EPINEPHRINE (open airways)
• Antihistamines, diphenhydramine 50 mg IV
• Corticosteroids
• IMMEDIATE WITHDRAWAL OF ANTIGEN IF POSSIBLE
• PREVENTION

Assessment, Diagnosis and Management of Neurogenic Shock

PATIENT ASSESSMENT

- **Hypotension**
- **Bradycardia**
- **Hypothermia**

MEDICAL MANAGEMENT

- **Goals of Therapy are to treat • Warm, dry skin or remove the cause**
- **RAP □**
- **PAWP □**
- **CO □**
- **Flaccid paralysis below level of the spinal lesion & prevent cardiovascular instability, & promote optimal tissue perfusion**

Management Neurogenic Shock

Alpha agonist to augment tone if perfusion still inadequate

- **dopamine at alpha doses (> 10 mcg/kg per min)**
- **ephedrine (12.5-25 mg IV every 3-4 hour)**

– Treat bradycardia with atropine 0.5-1 mg doses to maximum 3 mg

- **may need transcutaneous or transvenous pacing temporarily**

Septic shock

Syndrome of profound hypotension due to release of endotoxins / TNF / vasoactive peptides following bacterial destruction

- Usually associated with normal blood volume, high / low CO, and low SVR
- Re-distribution of blood to splanchnic vessels, with resultant poor skin perfusion

BOX 6.3

Definitions and Criteria for Septic, Hemorrhagic, and Cardiogenic Shock

SEPTIC SHOCK

Systemic Inflammatory Response Syndrome (SIRS)

Two or more of the following:

1. Temperature > 38°C or < 36°C
2. Heart rate > 90 beats/min
3. Respiratory rate > 20 breaths/min or $Paco_2$ < 32 mm Hg
4. White blood cell count > 12,000/mm^3, < 4,000/mm^3, or > 10% band neutrophilia

Severe Sepsis

SIRS with suspected or confirmed infection and associated with organ dysfunction or hypotension; organ dysfunction may include presence of lactic acidosis, oliguria, and/or altered mental status.

Septic Shock

SIRS with suspected or confirmed infection with hypotension despite adequate fluid resuscitation requiring vasopressor support; septic shock should still be diagnosed if vasopressor therapy has normalized blood pressure.

HEMORRHAGIC SHOCK

Simple Hemorrhage

Suspected bleeding with pulse rate < 100 beats/min, normal respiratory rate, normal blood pressure, and normal base deficit

Hemorrhage with Hypoperfusion

Suspected bleeding with base deficit < −4 mEq/L or persistent pulse rate > 100 beats/min

Hemorrhagic Shock

Suspected bleeding, with at least four criteria listed in Box 6.2

CARDIOGENIC SHOCK

Cardiac Failure

Clinical evidence of impaired forward flow of the heart, including presence of dyspnea, tachycardia, pulmonary edema, peripheral edema, and/or cyanosis

Cardiogenic Shock

Cardiac failure plus four criteria listed in Box 6.2

Clinical Presentation Septic Shock

Twophases:

– **“Warm” shock - early phase**

• **hyperdynamic response, VASODILATION**

– **“Cold” shock - late phase**

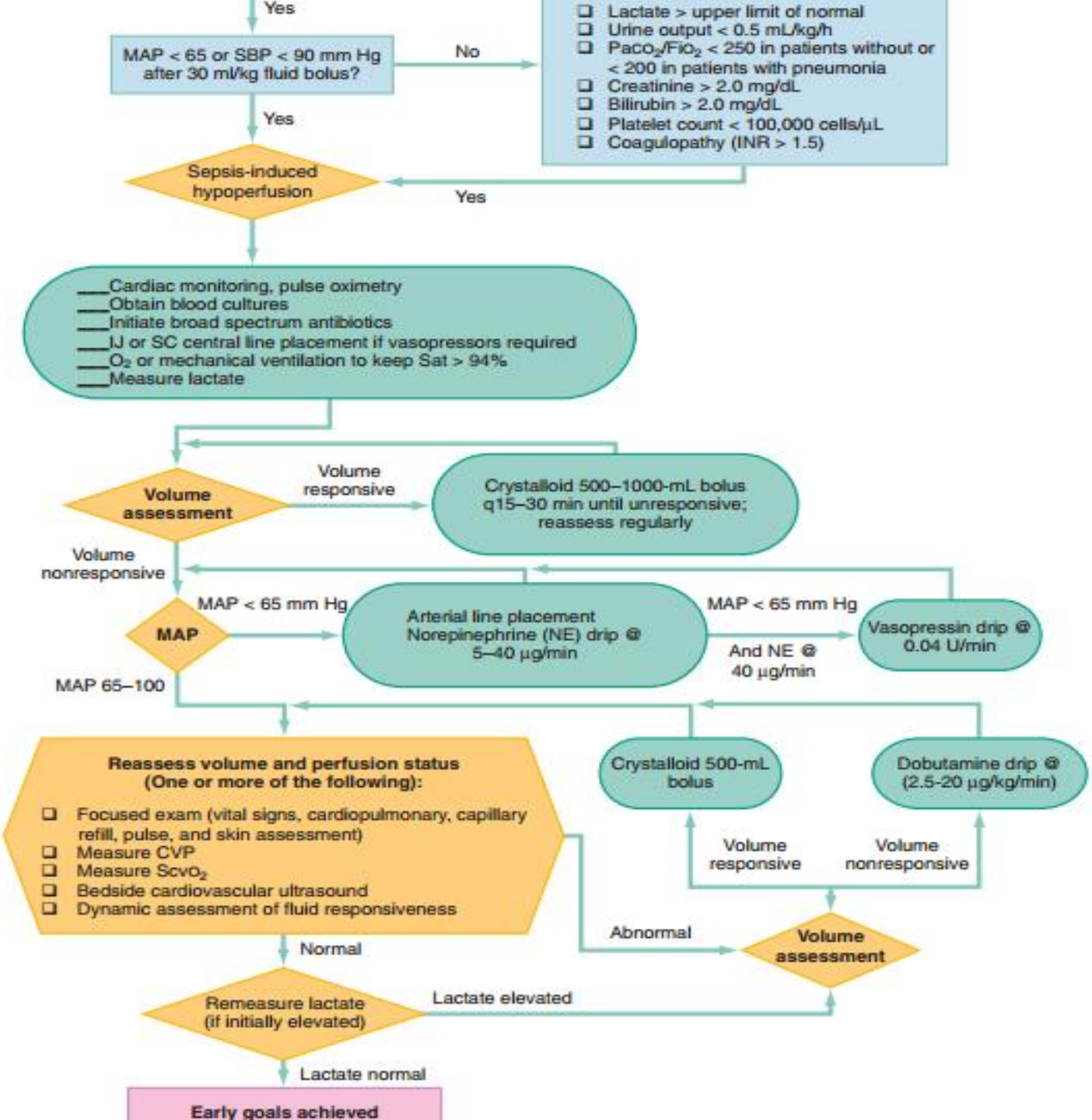
• **hypodynamic response**

• **DECOMPENSATED STATE**

Initial management of septic shock

Administer pure oxygen

- Start I.v. line, and take bloods for culture
- Give 20ml/kg boluses of colloid
- Observe rise in BP, CVP line if possible
- If > 60ml/kg (4200mL) consider ICU referral
- Broad spectrum antibiotics urgently


Yes
MAP < 65 or SBP < 90 mm Hg after 30 ml/kg fluid bolus?
No
❑ Lactate > upper limit of normal
❑ Urine output < 0.5 mL/kg/h
❑ PaCO2/FiO2 < 250 in patients without or < 200 in patients with pneumonia
❑ Creatinine > 2.0 mg/dL
❑ Bilirubin > 2.0 mg/dL
❑ Platelet count < 100,000 cells/μL
❑ Coagulopathy (INR > 1.5)
Yes
Sepsis-induced hypoperfusion
Yes
___Cardiac monitoring, pulse oximetry
___Obtain blood cultures
___Initiate broad spectrum antibiotics
___IJ or SC central line placement if vasopressors required
___O2 or mechanical ventilation to keep Sat > 94%
___Measure lactate
Volume assessment
Volume responsive
Crystalloid 500–1000-mL bolus q15–30 min until unresponsive; reassess regularly
Volume nonresponsive
MAP
MAP < 65 mm Hg
Arterial line placement Norepinephrine (NE) drip @ 5–40 μg/min
MAP < 65 mm Hg
And NE @ 40 μg/min
Vasopressin drip @ 0.04 U/min
MAP 65–100
Reassess volume and perfusion status (One or more of the following):
❑ Focused exam (vital signs, cardiopulmonary, capillary refill, pulse, and skin assessment)
❑ Measure CVP
❑ Measure ScvO2
❑ Bedside cardiovascular ultrasound
❑ Dynamic assessment of fluid responsiveness
Crystalloid 500-mL bolus
Dobutamine drip @ (2.5-20 μg/kg/min)
Volume responsive
Volume nonresponsive
Abnormal
Volume assessment
Normal
Remeasure lactate (if initially elevated)
Lactate elevated
Lactate normal
Early goals achieved