Crisis In Pregnancy and Managements

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Case Presentation

Case Summary

2 a.m.

- A 40 year old woman, 37 weeks pregnant with TWINS arrives at the hospital
- Cervix: 6 cm dilated. Patient is in severe pain. Labor is progressing rapidly
- Epidural block : 15 ml 0.125% bupivacaine + fentanyl 75 µg
- 15 minutes later - patient is still in severe pain
- 12 ml 0.25% bupivacaine given in two increments
- Patient is comfortable.

Case Summary

3 a.m.

- Obstetrician and anesthesiologist called “stand by ” to labor room
- Membranes ruptured spontaneously 10 min ago
- 3 min ago, the patient complained of difficulty breathing and lost consciousness
- Fetal heart rate: 90 beats/min
- Vaginal bleeding
- Patient cyanotic
- Maternal BP and Pulse not obtainable…

- ***What do you do ???***

Optimal Outcome

Immediate CPR ➔ ACLS

Early intubation

Left Uterine displacement

Start Cesarean by 4 min

Delivery by 5 min

IS THIS REALISTIC OUTSIDE THE OR?

- Standard ACLS protocol should be used.

- Perform chest compressions higher, slightly above the center of the sternum .
- Standard pharmacologic therapy should also be used without modification.

- In 5 cases where resuscitation was required after unintentional IV injection of Bupivacaine occurred:
  - The 3 patients who underwent immediate C-Section survived with no neurologic deficit.
  - Those 2 whom delivery was delayed suffered irreversible brain damage.

- Even if the fetus is nonviable, a C-Section may facilitate resuscitation by:
  - Relieving Aortocaval compression and restoring venous return
  - Decreasing metabolic demands
  - Emptying uterous allowing more effective chest compressions
- Early delivery (within 4-5 minutes of maternal cardiac arrest) maximizes the chances of both maternal and infant survival.

- If delivery does not facilitate successful maternal resuscitation, consider
  - Thoracotomy
  - Open-chest cardiac massage
  - Cardiopulmonary Bypass

- There are no specific AHA guidleines for CPR in the immediate postpartum period. Recommendations:
  - 1) Right hip displacement to relieve aortocaval compression.
  - 2) Firm surface under patient for best chest compressions.
  - 3) Open-Chest cardiac massage when standard closed-chest compressions fail .

- Cardiopulmonary Bypass (CPB)
  - bupivacaine-induced cardiac toxicity
  - pulmonary embolectomy in patients with massive pulmonary embolus(Amniotic emboli) .

Prevention - 'Be Careful'

- Evaluate the parturient's airway carefully
- Place epidural catheters with care and use appropriate test doses and local anesthetic doses
- Anticoagulate patients with previous thromboembolic events
- Manage parturients with cardiac disease carefully
  - Obtain a cardiology consult
  - Treat chronic arrhythmias
  - Consider placing invasive monitors during labor
- Careful administration of all medications to the parturients with a history of drug allergies.
- Use Beta mimetic tocolytic agents with caution

Management

- Assess that there is a cardiac arrest
  - Check peripheral pulses
  - Check for respiration
  - Check ECG
  - ***Loss of conciousness and No Breathing or No normal Breaths(Gasping)***
- If cardiac arrest is present
  - Inform the obstetrician and anesthesiologist and call for help
  - Start CPR
  - Follow management according to ACLS protocol
- Pregnant patients are ALWAYS considered to have full stomachs
  - Mask Ventilation should be performed with ***cricoid pressure***
  - The trachea should be intubated as quickly as possible

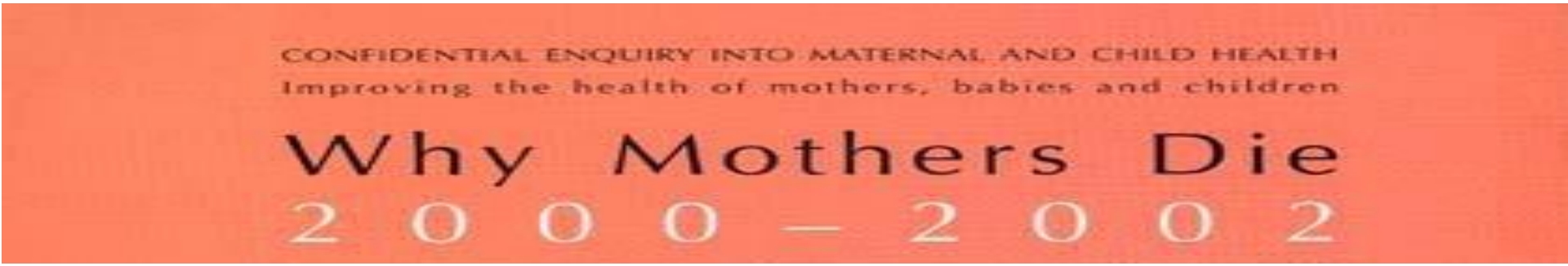
Management

- Maintain left uterine displacement during resuscitation
  - Minimize aortocaval compression
- If the fetus is viable and immediate resuscitative efforts are not successful, cesarean section should be performed quickly
  - To maximize the chances for maternal and fetal survival, this decision should be made within 5 minutes of arrest
  - The mother is easier to resuscitate after delivery of the infant because aortocaval compression is relieved

Consider: "Perimortem" Cesarean Section

- Start by 4 minutes, deliver by 5 minutes
- May help even if performed later
- Have "stand by " C/S kit available
- Perform operation in patient's room:
  Can move to OR after delivery
- Don't worry about sterility
- Vertical abdominal incision quickest
- Prepare for uterine hypotonia and bleeding

- Maternal complications that can occur when CPR is performed during pregnancy include
  - Laceration of the liver
  - Uterine rupture
  - Hemothorax
  - Hemopericardium


CONFIDENTIAL ENQUIRY INTO MATERNAL AND CHILD HEALTH
Improving the health of mothers, babies and children
Why Mothers Die
2000–2002

UK Confidential Enquiries 2000-2002 report

Cardiac deaths 11.25% of all maternal deaths

Or

2.2 / 100,000 maternities

Causes of Cardiac Arrest in Pregnancy (1)

Multiple, but includes:

Pre-eclampsia , eclampsia , HELLP

AFE syndrome

Obstetric anaesthesia complications :

Anaphylaxis

Toxicity

Total spinal

Causes of Cardiac Arrest in Pregnancy (2)

- Haemorrhage / hypovolaemia and shock.
- Septic shock
- Pulmonary embolus
- Trauma
- Congenital and acquired cardiac disease

Myocardial infarction(3-4times), cardiomyopathy, hypertensive disease, ***Dissection of aortic aneurysm .***

CEMACH Report 2002-2004

Acquired heart disease :

- Increasing maternal age
- Obesity
- Hypertension

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8 women died of myocardial ischaemia , Coronary artery dissection common cause (63%)

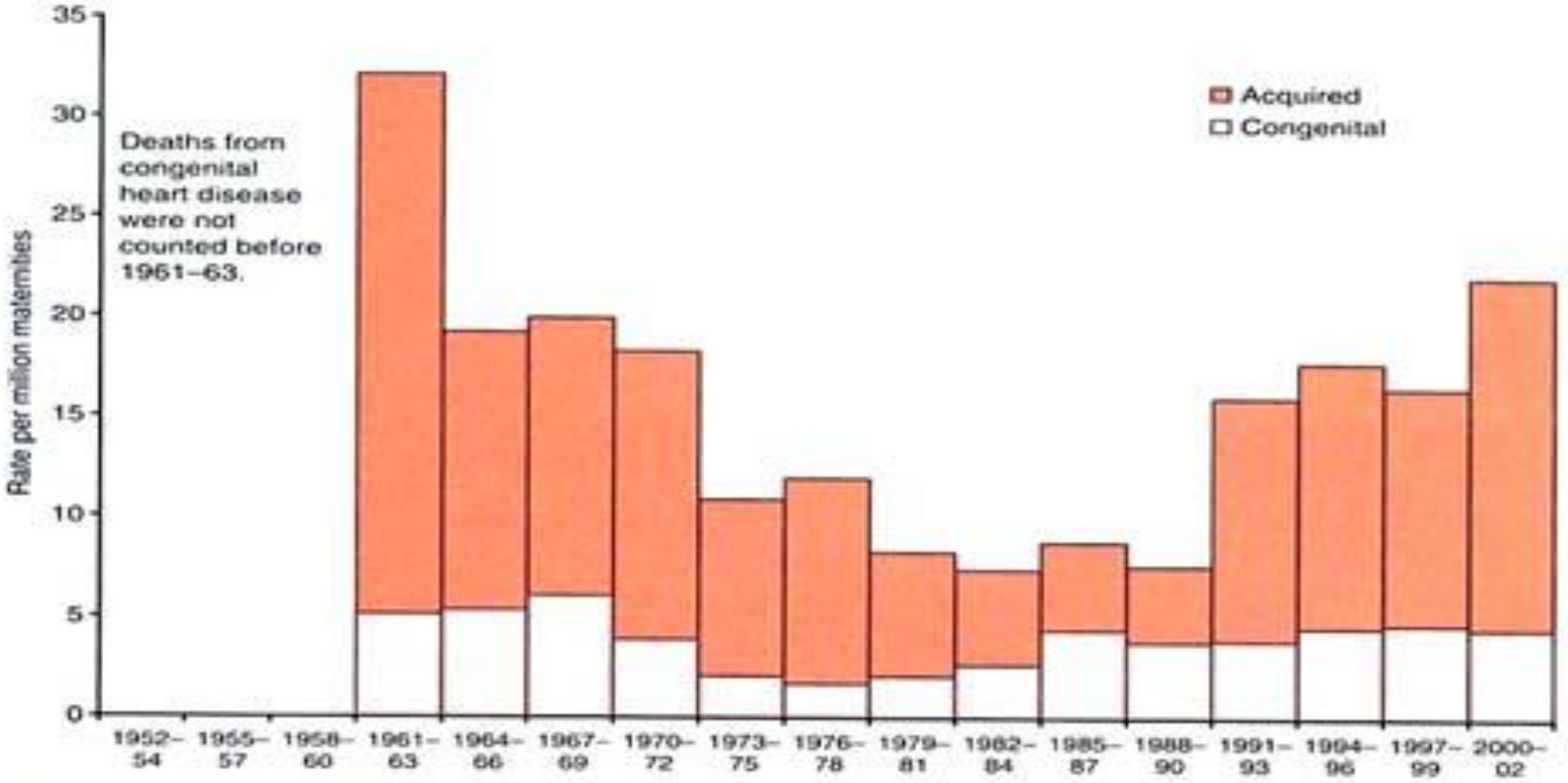


Figure 10.2 Maternal mortality rates for congenital and acquired cardiac disease; England and Wales 1961–1984, United Kingdom 1985–2002

CEMACH REPORT 2000-2002

INCREASING CARDIAC DEATHS DUE TO ACQUIRED CARDIAC DISEASE.

CONGENITAL CARDIAC DISEASE MORTALITY RATE IS ALMOST THE SAME AS 40 YEARS AGO.

Maternal Anatomical and Physiological Changes (1)

All maternal organ and systems change

Maternal Anatomical and Physiological Changes (2)

Cardio-vascular system

- Cardiac output rises to 150% of non pregnant state, (30% of CO to uterus vs 2%)
- Blood volume rises(50%)
- Heart rate rises
- Systemic and peripheral vascular resistances fall (except in PTE syndromes)
- Aorto-caval compression causes falls in CO and venous return

Maternal Anatomical and Physiological Changes (3)

Respiratory system

- Minute ventilation rises
- O2 consumption rises
- Functional residual capacity falls
- Compliance falls

Maternal Anatomical and Physiological Changes (4)

Gastro-intestinal system :

- Motility falls
- Lower oesophageal sphincter tone falls

(Increased risk of Aspiration in LOC)

Maternal Anatomical and Physiological Changes (5)

Blood Constituents

- There is a slight ***decrease*** in [Na+], [K+] and [Cl-]
- Albumin, globulins & total protein increase, **but their plasma concentrations decrease**

- Colloid oncotic pressure ↓ from 27→22 mm Hg (↑ risk of pulmonary edema).

Blood Constituents

•Thus, the preeclamptic patient, or those on tocolytic therapy are prone to the development of ***pulmonary oedema .***

• Changes in protein binding may lead to ***drug toxicity***, due to changes in the unbound fraction

Maternal Anatomical and Physiological Changes (6)

All these anatomical and physiological changes(Cardio-vascular &Respiratory) predispose to a **more rapid decrease in** arterial and venous O2 tension during periods of hypoxia of any cause.

Implications for Resuscitation (1)

- CPR produces (at maximum) 30% of normal cardiac output.
- Uterus takes 30% of cardiac output at term.

Implications for Resuscitation (2)

- CPR unlikely to sustain maternal and fetal life……

- If O2 supply and acid-base balance is maintained Utero-placental circulation has minimal resistance.

Implications for Resuscitation (3)

- Hypoxia / hypotension may sensitise utero-placental circulation to therapeutic doses of adrenaline causing further vasoconstriction.

Implications for Resuscitation (4)

Airway

Difficulty :

full dentition

hypertrophy of breasts

oedema of larynx / supra glottis

flared rib cage

raised diaphragm

Regurgitation / aspiration

Implications for Resuscitation (5)

<u>Breathing:</u>

- O2 consumption raised
- compliance reduced
- Functional Residual Capacity reduced

(Severe Hypoxemia during No Breaths)

Implications for Resuscitation (6)

General :

- cardiac compression difficult
- flared rib cage
- diaphragm raised
- obesity
- hypertrophy of breasts
- supine hypotension syndrom / aorto-caval compression

Peri-mortem Caesarean Section

CEMACH 2000-2002

- 8/19 (42%) infants born by ***peri mortem*** LSCS survived .
- No baby survived a ***post mortem*** LSCS.
- Peri-mortem LSCS important in the resuscitation of the mother

Peri-mortem Caesarean Section

***Important of C/S for maternal resuscitation* :**

a) relieves aorto-caval compression.

b) improves thoracic compliance.

c) increases blood volume (Autotransfusion from uterine).

Peri-mortem Caesarean Section (problems)

-The uterus should be emptied surgically within 5 minutes of cardiac arrest for effective resuscitation.

- Pregnant women become hypoxic more Quickly .

- CPR must be continued throughout C/S.

Results :

1 -Removal of aorto-caval compression is one of the major factors determining survival .

2 - Rapid development of maternal ***acidosis*** makes it more difficult to achieve resuscitation.

3 - Perimortem Caesarean section is important for resuscitation of the mother and fetus and should be

achieved within 5 minutes of cardiac arrest.

Obstetric Disorders

- Pre-eclampsia , Eclampsia
- Pulmonary edema
- Amniotic fluid embolism
-Venous thromboembolism
- Ovarian hyperstimulation syndrome
- HELLP
- Acute fatty liver of pregnancy

- Maternal mortality in the developed world, in patients admitted to intensive care units (ICUs), is rare.

- Unfortunately, mortality rates in the developing world are much higher.

- Risk of dying in pregnancy
is 1 in 65 in Asia and parts of Africa, whereas that same risk is 1 in 8700 in Switzerland.

- Most maternal deaths (up to 70%) occur antepartum, whereas 27% of mothers in the first 6 weeks postpartum.

- Obstetric disorders account for 55% to 80% of admissions to the ICUs in the obstetric population.

Pregnancy-induced hypertension (PIH) :

- ***Gestational hypertension (nonproteinuric hypertension),***
- ***Preeclampsia (proteinuric hypertension)***
- ***Eclampsia*** .

PRE-ECLAMPSIA

- PEC and eclampsia (6 – 8 - 10 % of all pregnancies)were the second most likely cause of maternal mortality in a study published in 1988 (At now 4th cause).

- Pre-eclampsia (PEC) is an idiopathic **multisystemic disorder** that is specific to human pregnancy and the puerperium (نفاس).

- It is essentially a **placental disorder** because complete molar pregnancies that contain no fetal tissue have been associated with PEC.

Definition

PEC is blood pressure of 140/90mmHg or more in a previously normotensive woman ***after 20 wks' gestation or in the early postpartum period and returning to NL within 3 months after delivery*** ***and proteinuria more than 300 mg/dL or 2+ or more on a urine dipstick with or without peripheral edema.***

PEC occurs in 8%to 10% of pregnancies, but in most cases it is mild to moderate.

Or :

HTN onset after 20 wks' gestation and at least one of the following:

1- proteinuria > 300 mg/24hr

2- Oliguria

3- Headaches with hyperreflexia, eclampcia, clonus or visual disturbances.

4- Increased liver enzymes or RUQ pain.

5- Thrombocytopenia, LDH ⬆, Hemolysis, DIC.

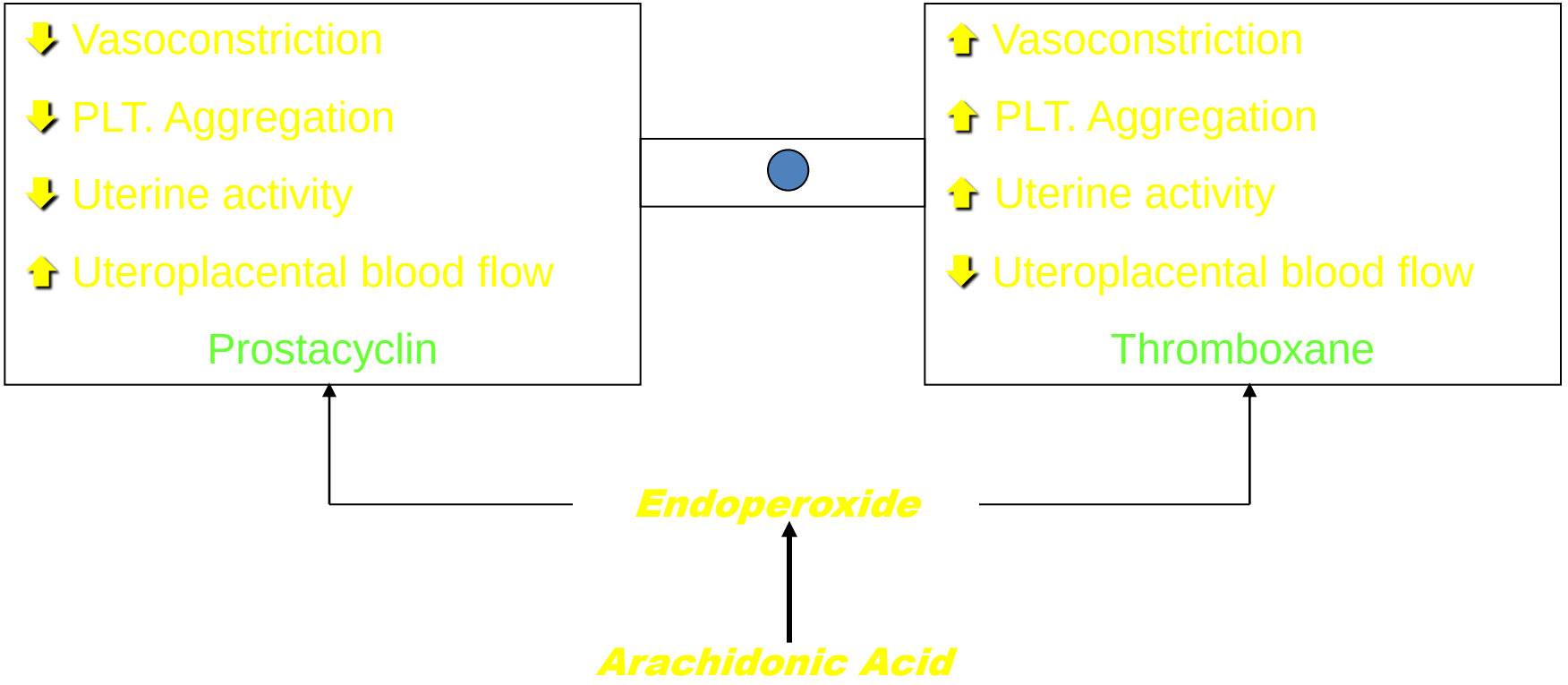
6- IUGR

Etiology :

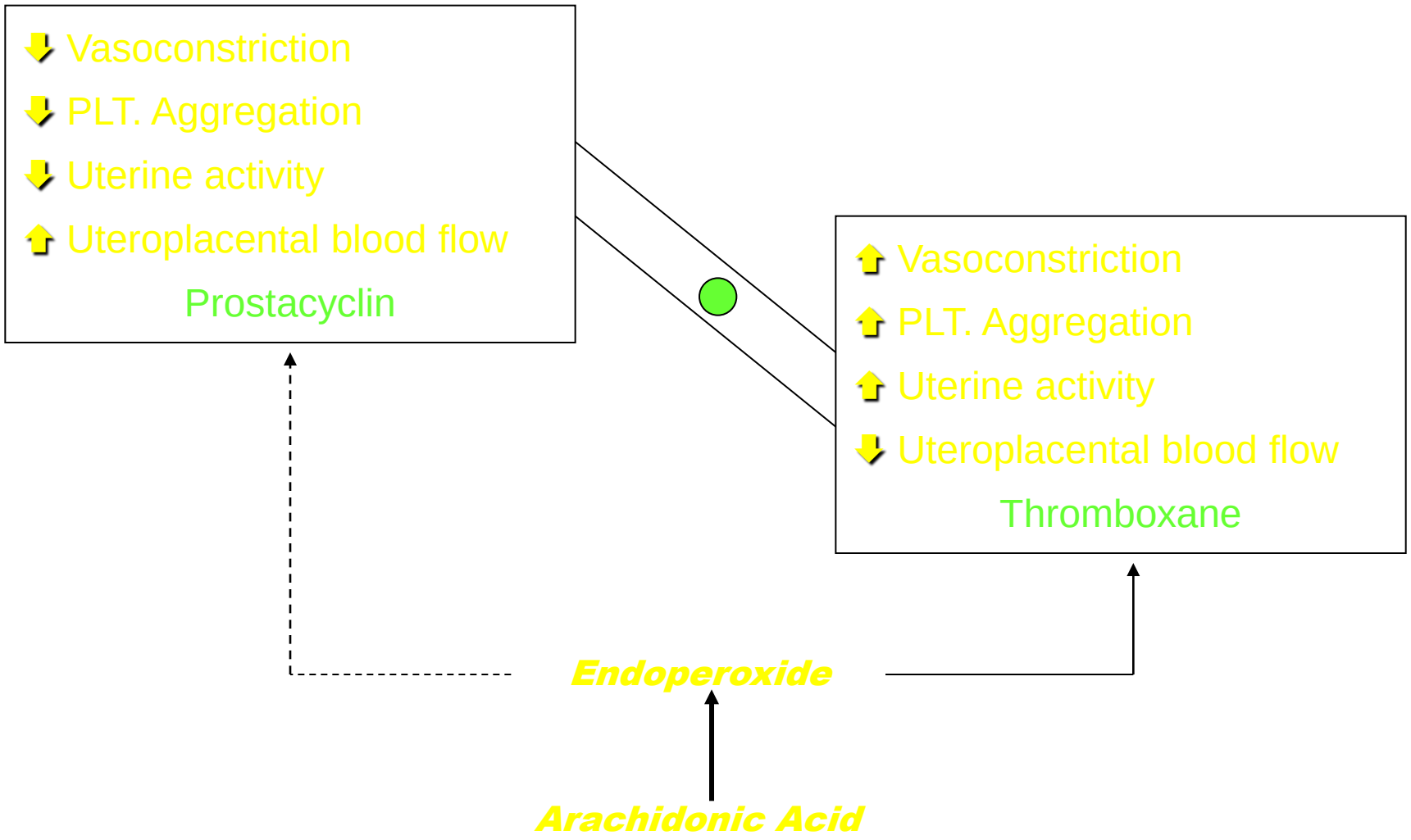
- ***Abnormal sensitivity of vascular smooth muscles to catecholamines,***
- ***Antigen-antibody reactions between fetal and maternal tissues (Immune Response),***
- ***Imbalance in the production of vasoactive prostaglandins (thromboxane A and prostacyclin).***

Pathophysiology :

An imbalance in placental postacyclin & thromboxane production:



(NL Pregnancy)



(Preeclampcia)

Clinical features :

1- Upper airway edema

2- Pulmonary edema

3- Cordiovascular effects

4- Renal effects

5- Hepatic impairment

Classification :

	Mild	Severe
SBP	<160mmHg	≥ 160 mmHg
DBP	<110mmHg	≥ 110 mmHg
Urinary prot.	<5g/24h	≥ 5 g/24h
	Dipstick + or 2^{+}	Dipstick 3 + or 4 +
U/0	> 500 ml/24h	≤ 500ml/24h
Headaches	No	yes
Visual dist.	No	Yes
Epigast. Pain	No	Yes
RUQ pain	No	Yes
Pul. Edema	No	Yes
Cyanosis	No	Yes
HELLP	No	Yes
Plt. Count	> 100/000/mm^3	<100/000/mm^3

Hypertensive disease is the fourth leading cause of deaths in mothers (New Studies).

ICH is the largest cause of deaths in preeclampcia / eclampcia, reflecting a failure of effective anti – hypertensive therapy.

Treatment :

1- Control of HTN

2- Prevention of seizure

3- Delivery of fetus

Eclampsia

Eclampsia is defined as seizures or coma in the setting of PEC without any evidence of other neurologic disorders.

The cause of the convulsions is thought to be related to cerebral vasospasm with local ischemia, hypertensive encephalopathy, vasogenic edema, or endothelial damage.

Seizure prophylaxis and control are best achieved with magnesium sulfate (MgSO4) infusions(patellar reflex monitoring and monitoring of respiratory rate and urine output for Mg Toxicity).

Benzodiazepines (respiratory depression in the newborn)
Phenytoin 3rd drug.

Eclampsia : (Treatment)

- ***Seizure superimposed on preeclampsia***
- ***Airway support, oxygenation, and immediate treatment to stop the seizure activity***
- ***Thiopental, a benzodiazepine (diazepam), or a bolus of magnesium sulfate***
- ***Magnesium therapy for prophylaxis against subsequent seizures***

Seizure Control:

Magnesium Sulfate is the agent of choice.

It reduces Seizures by more than 50%.

Mechanism of effect is debatable.

Mgso4 is a smooth muscle relaxant in relatively high concentration .

Dose: Loading : 4 gm/IV/over a 10 min.

Maint : 1gm/h

Therapeutic serum level is narrow (2-3.5 mmol/L).

Resp. depression & ⬇ or absent DTR are indicative of ***toxicity****.*

If toxicity is present: 10ml of 10% calcium gluconate given by slow I.V inj. counteracts its effects.

Magnesium Sulfate Toxicity

***Cardio vascular** : ECG interval changes (prolonged PR, QRS and QT intervals) at magnesium levels of 2.5–5 mmol/L to AV nodal conduction block, bradycardia, hypotension and cardiac arrest at levels of 6–10 mmol/L.*

***Neurological effects** ranging from loss of tendon reflexes, sedation, severe muscular weakness, and respiratory depression are seen at levels of 4–5 mmol/L*

Other *: Gastrointestinal symptoms (nausea and vomiting), skin changes (flushing), electrolyte/ fluid abnormalities (hypophosphatemia).*

Anticonvulsant therapy and antihypertensive therapy should continue at least 48 hours postpartum.

Hypertensive Emergency

Complications :

PEC may present with severe hypertension with

a ***potential for end-organ damage*** , retinal hemorrhage, papilledema, pulmonary edema, sever headache, and renal failure.

Acute cerebral complications (eg, intracranial hemorrhage, massive cerebral edema) are particularly worrisome because they account for more than 75% of maternal deaths secondary to PEC.

The goal of treatment is to prevent end-organ damage while still maintaining adequate uteroplacental perfusion.

Strategy of Treatment

Blood pressure of more than 180 mm Hg (systolic) or 110 mm Hg (diastolic) should be treated urgently in all cases.

Patients with evidence of end-organ damage benefit from treatment of blood pressure more than 160 mm Hg (systolic) or 100 mm Hg (diastolic).

Drugs

- Intravenous **labetalol** or intravenous **hydralazine**.

  Labetalol is better.

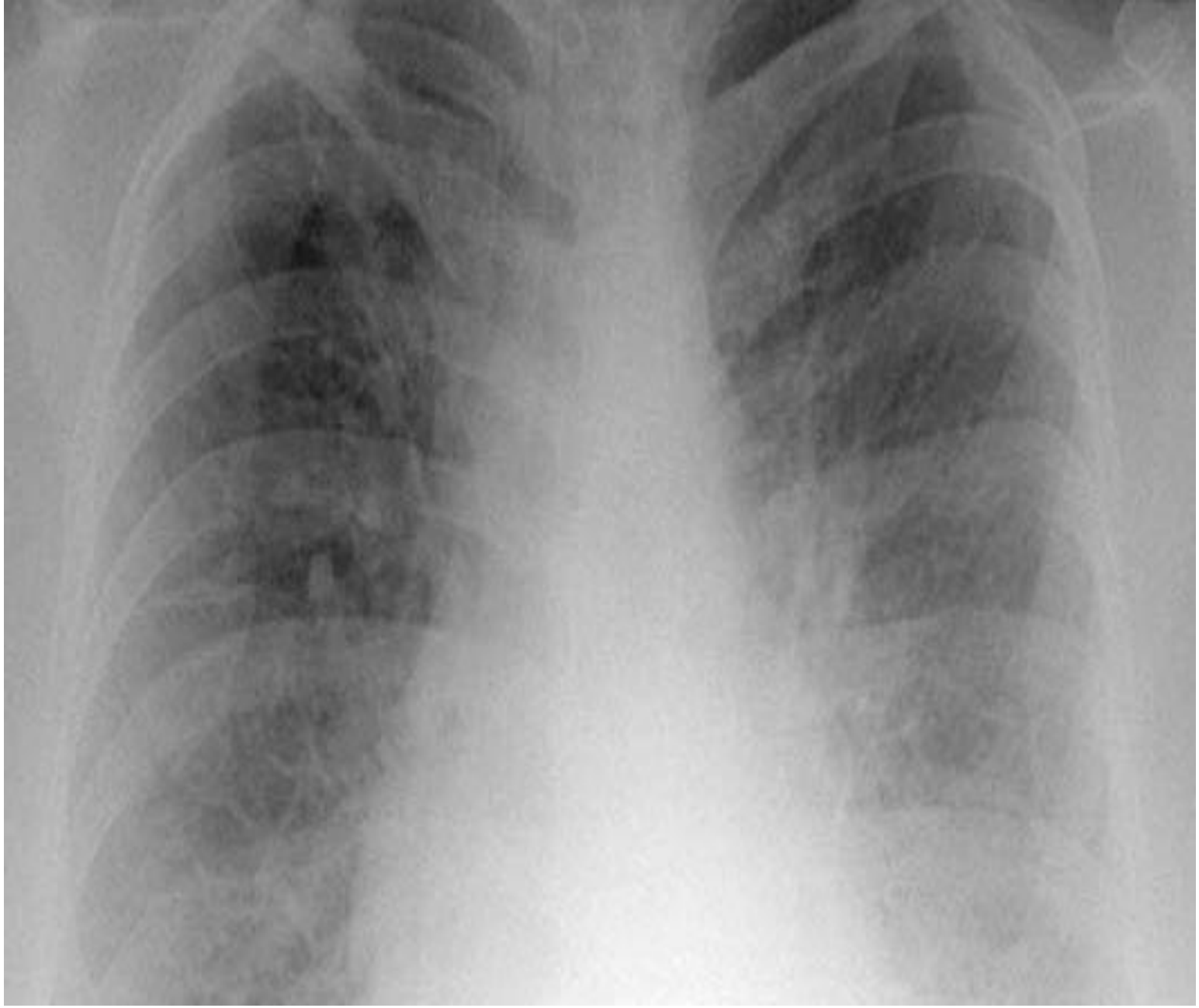
- Nifedipine orally, in 30 min.
- Esmolol
- Nitroglycerin.
- Nitroprusside (cyanide accumulation in the fetus).

Comparison of properties hydralazin & Labetalol

Drug	Hydralazine	Labetalol
Mode of action	Vasodilator	α & β blocker (1:3)
Onset	Gradual	Quick
Dose	5-10 mg/IV/Slowly	10-20 mg/ Iv/Slowly
Interval	Repeat after 20 min.	Titrate to effect
Infusion rate	2mg/hr up to 20mg/hr	20 mg/h Up to 160 mg/hr
Effect on heart	Compen . tachycardia	No effect

Pulmonary Edema

- Pulmonary edema complicates 0.05% of low-risk pregnancies and 2.9% of all cases of PEC.
- Pulmonary edema in patients with PEC can be either cardiogenic or noncardiogenic.
- Plasma colloid oncotic pressure falls.
- Pulmonary edema develops most commonly (70%–80% of cases) in the postpartum period.



44-year-old G1P0 with pre-eclampsia and cardiogenic pulmonary edema.

Treatment of pulmonary edema is depending on whether pulmonary edema is thought to be cardiogenic or noncardiogenic.

-Most patients respond to 10 mg of *furosemide* administered intravenously.

- Afterload reduction and blood pressure control may be achieved with administration of intravenous *hydralazine* or *labetolol*.

HEMOLYSIS, ELEVATED LIVER ENZYMES, AND LOW PLATELETS (HELLP)

- **HELLP** complicates 1 in 1000 pregnancies,

but is much more common in patients with severe PEC, occurring in up to 20% of patients.

- It is important to differentiate HELLP from thrombotic thrombocytopenic purpura/hemolytic uremic syndrome.

- ***Delivery*** is the ultimate treatment for HELLP syndrome.

HELLP Synd :

Hemolysis	**1. abnormal peripheral Blood smear (Schistocyte)** **2. BiL.⬆**
Liver enzymes	**⬆1. Aspartate aminotransferase ≥ 70 U/L(AST)** **2. LDH > 600 U/L**
PLT. ⬇	**PLT < 100 / 000 / mm3**

(Signs & symp. in HELLP synd. are not diagnostic.)
PLT Count usually returns to NL within 72 hrs of delivery.

ACUTE FATTY LIVER OF PREGNANCY

- Affects 1 in 13,000 pregnancies but is usually associated with a high mortality. Acute fatty liver of pregnancy is characterized by the deposition of microvesicular fat in the hepatocytes.

- Clinical presentation may include ***fulminant hepatic failure with coagulopathy, coma, and renal failure.***

- Transfer to a liver unit may be necessary in severe cases, and some patients may require liver transplantation.

AMNIOTIC FLUID EMBOLISM (AFE)

-Amniotic fluid embolism (AFE) is a rare but potentially catastrophic obstetric complication.

- The incidence of AFE varies significantly in the literature

from 1 in 8000 to 1 in 80,000 live births, mortality reports ranging from 26% to 86%.

- Complications (80%):Cardiovascular collapse , DIC(80%),

ARDS,neurologic complications related to a ***hypoxic*** injury.

- Risk factors : use of oxytocin, uterine tetany, male fetus, multiparity, and advanced maternal age.

- Manifestations : Hypoxia, Hypotension, Right ventricular failure ,

Left ventricular dysfunction, septic shock-like, noncardiogenic pulmonary edema.

Diagnosis of AFE is based on a high degree of suspicion and recognition.

Treatment of AFE after the initial resuscitative effort is supportive.
Goals of therapy should be early oxygenation, hemodynamic support, improving oliguria, and close monitoring for the development of coagulopathy

Hemodynamic Optimization (goals):

Early Goal-Directed Treatment.

- Central venous pressure of 8 to 12 mm Hg,

- MAP of 65 to 100 mm Hg,

- ScvO2 70%,

- hematocrit 30% or hemoglobin 8 g/dL,

- lactate <2 mmol/L,

- urine output 0.5-1 mL / kg /h

OVARIAN HYPERSTIMULATION SYNDROME (OHSS)

- Rare but potentially life-threatening.

- Associated with assisted reproductive technologies.

- OHSS presents at approximately ***3 to 8 weeks***' gestation with ascites, dyspnea, severely enlarged polycystic ovaries, electrolyte imbalance, hemoconcentration.

- Symptoms: nausea, vomiting, and diarrhea to hemodynamic instability, acute renal failure, and ARDS, and hypercoagulability.

Patients with OHSS should be monitored frequently for worsening severity with daily weights and periodic laboratory measurements of electrolytes, analysis of renal and hepatic function, complete blood counts, and physical examinations.

Treatments:

- *Intravenous fluids* are needed to expand the intravascular volume.
- Thromboprophylaxis with *anticoagulants* such as unfractionated Heparin or LMWH.
- Mechanical ventilation, invasive hemodynamic monitoring, and short-term hemodialysis are occasionally required.
- Dopamine for patients with severe oliguria.

--*Early termination of pregnancy in patients with critical complications of OHSS.*

PULMONARY EMBOLISM

- Incidence of VTE is 5 to 12 per 10,000 pregnancies in the antenatal period and 3 to 7 per 10,000 deliveries in the postpartum period.

- Hypercoagulability in pregnancy is a result of increased levels of procoagulant factors (increased factor V and VIII levels) and decreased fibrinolytic and anticoagulant activity

- Risk of VTE in pregnancy is increased in patients with additional risk factors, including prolonged bed rest, advanced maternal age, family history of thrombosis, multiparity, previous thrombosis, previous superficial phlebitis, pre-eclampsia, tobacco use, or operative delivery.

Diagnosis is based on:

Ph.Exam

Radiologic studies(CTPA , MRI , SONO)

D-dimer test

Treatments:

Anticoagulation therapy or fibrinolytic therapy by:

- Heparin or LMWH.(warfarin is teratogenic)
- SK , UK, Tpa(safe in pregnancy)

PERIPARTUM CARDIOMYOPATHY

- Peripartum cardiomyopathy is a dilated cardiomyopathy of uncertain cause.

- The true incidence is unknown, with reported rates ranging from 1 in 1500 to 1 in 15,000.

- Mortality from peripartum cardiomyopathy range from 25% to 50%.

Approximately 50% of women recover to baseline ventricular

function within 6 months of delivery.

The other 50% of women have varying degrees of persistent

dysfunction ranging from mild, compensated heart failure to deterioration and death, with most deaths occurring in the first 3 months postpartum.

Diagnostic criteria :

- Development of cardiac failure in the last month of pregnancy or within 5 months after delivery.
- The absence of a determinable cause of cardiac failure.
- The absence of demonstrable heart disease before the last month of pregnancy.
- Left ventricular dysfunction as demonstrated by *echocardiography*

Risk factors for peripartum cardiomyopathy include :

- advanced maternal age
- multiple gestation
- pre-eclampsia
- gestational hypertension.

Several possible causes have been proposed :

- myocarditis .
- abnormal immune response to pregnancy.
- prolonged tocolysis .
- familiar .

Treatments: As CHF:

- ***Sodium restriction***

- ***Diuretics(loop diuretic"safe "in pregnancy and breast feeding)***

- ***Vasodilator therapy:***

1 - ACE inhibitors are contraindicated in pregnancy because of teratogenicity

but some are compatible with breastfeeding.

2 -Hydralazine *and* ***nitrates*** *are safer alternatives in pregnancy.*

3 -Beta blockers *may be useful primarily in the postpartum period.*

4 -Anticoagulation *should be considered in patients with peripartum cardiomyopathy(EF<35%,AF, mural thrombus).*

Considerations

Anesthetic and Obstetric Considerations in PIH :

Severity of the condition

Associated features

Systemic imvolvement

Evaluation of the airway

Fluid status

BP Control

Lab. Tests: ***CBC***

renal Profiles

LFTs

If coagulopathy in clinically present, coagulation tests also should be performed.

Manage parturients with cardiac disease carefully:

1- Obtain a cardiology consult
2- Treat chronic arrhythmias
3- Consider placing invasive monitors during labor

In suspision to or Pul.Edema :

1 – Na , K ,Cl *(and In OHSS)*
2 – Albumin
3 – CXR
4 - Echocardiography

In gestational DM:

1- F.B.S or B.S

2 – Urine Glucose and Ketone in DKA

In VTE :

1 – PT ,PTT ,INR

2 – Platelet count

Monitoring Options in Critically ill Patint:

1. General intensive care monitoring

Arterial catheter

Oxygen saturation by pulse oximetry

Continuous ECG

CVP

ScvO2

Temperature (bladder, esophagus)

Urine output

Arterial blood gases

Serum lactate

Blood glucose, electrolytes, CBC, and general blood sampling

Chest radiograph

2. More advanced hemodynamic monitoring

Echocardiography

Cardiac output monitoring (either noninvasive or PA catheter)

3. Cerebral monitoring

EEG (on indication/continuously): early seizure detection and treatment

CT/MRI