ACVC
Association for
Acute CardioVascular Care

Edition 2025

CLINICAL DECISION MAKING TOOLKIT

Instant guidance for diagnosis, risk stratification and management

ESC
European Society
of Cardiology

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Acute CardioVascular Care

The Clinical Decision Making Toolkit

is produced by the Association for Acute CardioVascular Care (ACVC) of the European Society of Cardiology (ESC).

This toolkit is supported by Boston Scientific and Inari Medical in the form of an unrestricted financial support. The scientific programme has not been influenced in any way by its sponsor.

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The Association for Acute CardioVascular Care
Clinical Decision-Making TOOLKIT

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ISBN: 978-2-9537898-7-4

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SPECIFIC EMERGENT SITUATIONS

CHAPTER 9.1

MANAGEMENT OF DELIRIUM IN THE ICCU

G.Tavazzi & W.Akhtar

Delirium Definition

Delirium is an acute and **fluctuating disturbance** of attention and cognition, caused by an underlying medical condition.

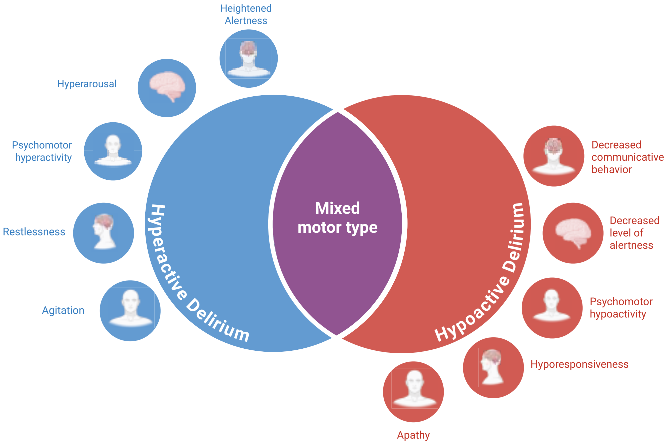
The pathophysiology of delirium that is associated with critical illness remains largely uncharacterized and may vary depending on the cause. The increased risk associated with the use of GABAa agonists and anticholinergic drugs led to the suggestion that the GABAergic and cholinergic neurotransmitter systems play a contributory role. In particular, central cholinergic deficiency may be a final common pathway. Alternative hypotheses include excess dopaminergic activity and direct neurotoxic effects of inflammatory cytokines. Currently, these hypotheses are un- proven, making pharmacologic management strategies largely empirical.

Hyperactive delirium may manifest as a combative patient who does not follow the rules of treatment, while hypoactive delirium may manifest as a somnolent patient who is disengaged and inattentive.

The epidemiology of delirium in cardiac intensive care units varied consistenly across differen studies rangin from 8% to > 50% in those with advanced age and it is assocated with increased mortality ICU mortality (27% versus 3%; P<0.001).

Delirium Definition

Delirium is a complex neuropsychiatric syndrome marked by acute, fluctuating disturbances in attention, awareness, and cognition (DSM-5).



Ref: Delirium in cardiovascular medicine, European Heart Journal, ehag088 - https://doi.org/10.1093/eurheartj/ehag088 - Figure 2

Aetiology and Risk Factors

D - Drugs and Medications
Polypharmacy, alcohol withdrawal, medications with psychoactive side effects (eg steroids)

E - Electrolyte Imbalance
Hypo/hypernatraemia, hypercalcaemia, dehydration, lack of nutrition

L - Lack of Sensory Input
Vision or hearing impairments

I - Infections
Sepsis from any source

R - Reduced Brain Reserve
Dementia, neurological disease

I - Intensive Care Environment
Sleep deprivation with noise and light, immobilisation, constipation, urinary retention, long admission

U - Underlying Medical Conditions
Liver failure, renal failure, heart failure

M - Major Surgery or Trauma
Surgical and anaesthesia related risks, Pain

Prevention

Expanded A to I bundle for intervention, early detection and prevention of Delirium	
Tools and therapies	**Environment**
A • **A**ssessment and management of pain **B** • **B**oth daily sedation hold & spontaneous breathing trials **C** • **C**hoice of analgesia and sedation **D** • **D**elirium detection CAM-ICU-4AT **E** • **E**arly mobilization	**F** • **F**amily engagement & empowerment **G** • **G**aining insights into patient's needs **H** • **H**olistic and personalized care (music, pleasant visual stimuli, white boards) **I** • **I**CU design redefinition (e.s. bigger "noise isolated" rooms with windows and relatives 24/7)

Ref: Crit Care Med 2019; 47:3-14

Assessment of pain

Critical Care Pain Observation Tool (CPOT)	
Variable	**Evaluation (points)**
Intubated	Yes/no
Facial Expression	Relaxed, neutral 0 Tense +1 Grimacing +2
Body movements	Absence 0 Protection +1 Restlessness +2
Muscle Tension	Relaxed 0 Tense/rigid +1 Very tense +2

Score interpretation:

Pain score: no (NRS = 0/CPOT = 0), mild (NRS = 1-3/CPOT = 1-2), moderate (NRS = 4-6/CPOT = 3-4), or severe (NRS = 7-10/CPOT = 5-8) pain.

Assessment of pain (Cont.)

Behavioral Pain Scale (BPS)	
Variable	**Evaluation (points)**
Intubated	Yes/no
Facial Expression	Relaxed +1 Partially tightened (brown lowering) +2 Partially tightened(eyelid closed) +3
Upper limb movements	No movement +1 Partially bent +2 Fully bent with finger flexion +3 Permanently retracted +4
Compliance with mechanical ventilation	Tolerating movement +1 Coughing but tolerating for most of the time +2 Fighting ventilator + 3 Unable to control ventilation +4

≤3 no pain; 4-5 indicate mild pain; 6-11 indicate an unacceptable amount of pain; ≥12 indicate maximum pain

Identification

CAM-ICU: For Sedated or Non-Verbal Patients

- **Step 1: Acute Change or Fluctuating Mental Status**
  Check for new or fluctuating changes in mental status. Positive if yes.
- **Step 2: Inattention**
  Perform «Squeeze on A» test: Patient squeezes hand only when «A» is heard during a letter sequence. Positive if >2 errors.
- **Step 3: Altered Level of Consciousness**
  Assess alertness. Positive if RASS score other than 0.
- **Step 4: Disorganized Thinking**
  Five Yes/No questions (e.g., «Will a stone float on water?»). Positive if >1 error.

Delirium is present if Steps 1 and 2 are positive, plus either Step 3 or Step 4

4At : for awake and verbal patients

Alertness (0-4): Is the patient abnormally drowsy or agitated?
AMT4 (0-2): Test memory with questions like «What is your age?»
Attention (0-2): Ask patient to recite months backward.
Acute Change or Fluctuation (0-4): Is there a sudden change in cognition?

Total score of **4 or above** suggests delirium.

SAT/SBT

Daily and progressivly longer Spontaneous Awakening Trials (SATs) and **spontaneaous breathing trials** (in intubated patients) are crucial to asses neurological status and to shorten the length of continous sedative infusion and mechanical ventilation.

In this setting the use of benzodiazepine, although not specifically indicated for clinical reasons (e.g. seizures), is discouraged as it does prolong the time to awakening without established benefit on the haemodynamic profile.

ICU treatment

1. Modifiable factors

- Modulate disruption of previous medication (e.g.anti-depressamt/anti-pshycotic drugs)
- Treat the withdrawal of abuse substances (e.g. alcool, drugs)
- Target the appropriate nutrition (nutrition score – e.g NUTRIC Score, NRS-2002, MST)
- Correct electrolyte imbalance
- Control the source of sepsis
- Control the metabolic/organ failure
- Optimize hemodynamic/perfusion
- Reduce discomfort (devices, orotracheal tube, etc..)

2. Unmodifiable factors

- Patient’s previous history predisposing to delirium
- Neurological diseases (e.g. dementia, mental impairment)
- Psychiatric disease
- Hypoxic brain injury

Sedation/Pain treatment

Drug	Contraindications/side effect	Dosages
Propofol	Hypersensitivity to egg/soy; hypotension and respiratory depression (at higher dosages and bolus)	ICU sedation infusion: start 5 mcg/kg/min (0.005 mg/kg/min) and titrate 5-50 mcg/kg/min (0.3-3 mg/kg/hr)
Dexmedetomidine	Bradycardia, advanced heart block, hypotension, significant heart failure; No bolus	Infusion: typically 0.2-0.8 mcg/kg/hr
Midazolam	Accumulation/toxicity with prolonged infusion (renal failure, metabolic acidosis). Delirium in advanced age	Infusion: usual initial 0.02-0.10 mg/kg/hr
Fentanyl	Accumulation/ toxicity (ileus)/addiction. Adjust in hepatic impairment.	IV infusion 0.5-3 mcg/kg/hr for continuous analgesic infusions
Remifentanyl	Thoracic rigidity/ toxicity (ileus)/addiction.	Maintenance dose: 0.008-0.25µg/kg/min

Delirium pharmacological Treatment

Drug	Contraindications/side effect	Dosages
Quetiapine	QTc prolongation, less extrapyramidal effects than haloperidol	100 to 200 mg orally per 2 in at 2 per day
Olanzapine	Bradycardia, arrhythmias	5-25 mg day (2-3 admin)
Dexmedetomidine	Bradycardia, advanced heart block, hypotension, significant heart failure; No bolus	0.2 to 0.5 g/kg per h maintenance dose
Haloperidol	QTC prolongation, large potential for extrapyramidal effects	Oral: 0.5 to 5 mg every 6 to 8 h IV (haloperidol lactate only): 0.5 to 10 mg q15 to 30 min until response achieved, then give 25% of last bolus dose Q6H
Ziprasidone	TC prolongation, less extrapyramidal effects than haloperidol. Not to be used in recent HF and AMI	10 mg Q2H or 20 mg Q4H IM (max 40 mg/ daily) for acute agitation
Aripriprazol	Bradycardia	5- 15 mg daily

Non-pharmacological Treatment

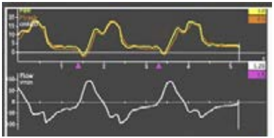
Orientation activities

Relatives presence

Vision, speaking and Hearing Aids

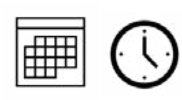
Respect circadian cycle

Patient-ventilator synchrony



Early mobilization/physiotherapy

Provide calendar and clock



Delirium in multidisciplinary round/Clinical sheets

Avoid/minimize restraints

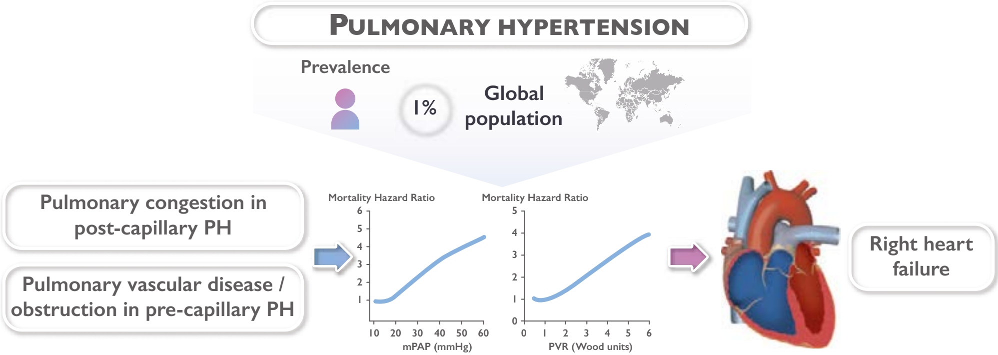
CHAPTER 9.2

EMERGENCIES IN PULMONARY HYPERTENSION

S.Price &, M.Ardissino

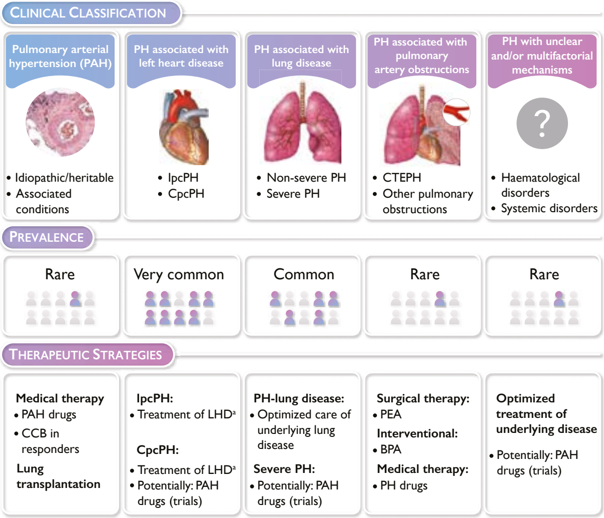
Epidemiology and classification of pulmonary hypertension

For teams managing suspected or confirmed PH decompensation;
complements the full 2022 ESC/ERS PH guideline



Ref: 2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension, European Heart Journal, Volume 43, Issue 38, 7 October 2022, Pages 3618–3731 Figure 1

Epidemiology and classification of pulmonary hypertension (Cont.)



Ref: 2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension, European Heart Journal, Volume 43, Issue 38, 7 October 2022, Pages 3618–3731 Figure 1

Diagnosis of pulmonary hypertension - clinical, ECG and radiographic features

Signs of PH

- Central, peripheral, or mixed cyanosis
- Accentuated pulmonary component of the second heart sound
- RV third heart sound
- Systolic murmur of tricuspid regurgitation
- Diastolic murmur of pulmonary regurgitation

Signs of RV backward failure

- Distended and pulsating jugular veins
- Abdominal distension
- Hepatomegaly
- Ascites
- Peripheral oedema

Signs pointing towards underlying cause of PH

- Digital clubbing: Cyanotic CHD, fibrotic lung disease, bronchiectasis, PVOD, or liver disease
- Differential clubbing/cyanosis: PDA/Eisenmenger's syndrome
- Auscultatory findings (crackles or wheezing, murmurs): lung or heart disease
- Sequelae of DVT, venous insufficiency: CTEPH
- Telangiectasia: HHT or SSc
- Sclerodactyly, Raynaud's phenomenon, digital ulceration, GORD: SSc

Signs of RV forward failure

- Peripheral cyanosis (blue lips and tips)
- Dizziness
- Pallor
- Cool extremities
- Prolonged capillary refill

Ref: 2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension, European Heart Journal, Volume 43, Issue 38, 7 October 2022, Pages 3618-3731 Figure 3

Diagnosis of pulmonary hypertension - clinical, ECG and radiographic features (Cont.)

Typical ECG abnormalities in pulmonary hypertension

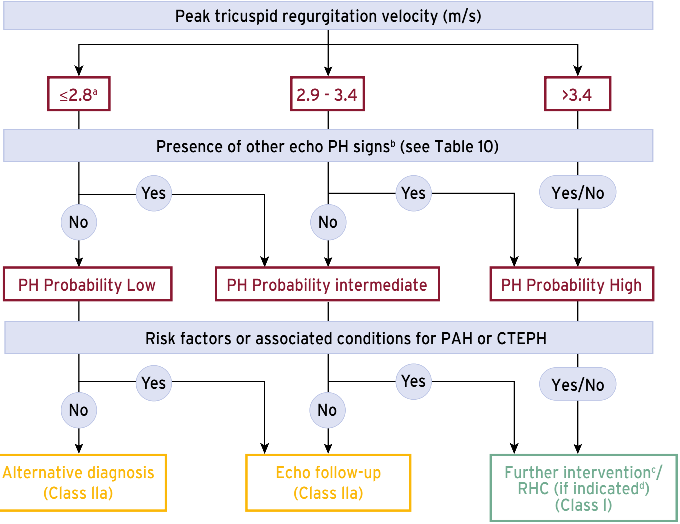
- P pulmonale (P >0.25 mV in lead II)
- Right or sagittal axis deviation (QRS axis >90° or indeterminable)
- RV hypertrophy (R/S >1, with R >0.5 mV in V1; R in V1+S in lead V5 >1 mV)
- Right bundle branch block—complete or incomplete (qR or rSR patterns in V1)
- RV strain pattern (ST depression/T-wave inversion in the right pre-cordial V1-4 and inferior II, III, aVF leads)
- Prolonged QTc interval (unspecific)

Typical chest x-ray abnormalities in pulmonary hypertension

Signs of PH and concomitant abnormalities	Signs of left heart disease/pulmonary congestion	Signs of lung disease
Right heart enlargement	Central air space opacification	Flattening of diaphragm (COPD/emphysema)
PA enlargement (including aneurysmal dilatation)	Interlobular septal thickening 'Kerley B' lines	Hyperlucency (COPD/emphysema)
Pruning of the peripheral vessels	Pleural effusions	Lung volume loss (fibrotic lung disease)
'Water-bottle' shape of cardiac silhouette	Left atrial enlargement (including splayed carina) Left ventricular dilation	Reticular opacification (fibrotic lung disease)

Ref: 2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension, European Heart Journal, Volume 43, Issue 38, 7 October 2022, Pages 3618-3731 table 8 & 9

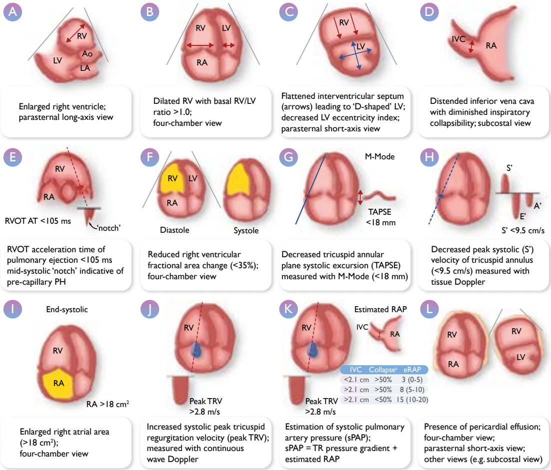
Diagnosis of pulmonary hypertension - Echocardiography



Ref: 2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension, European Heart Journal, Volume 43, Issue 38, 7 October 2022, Pages 3618-3731 Figure 5

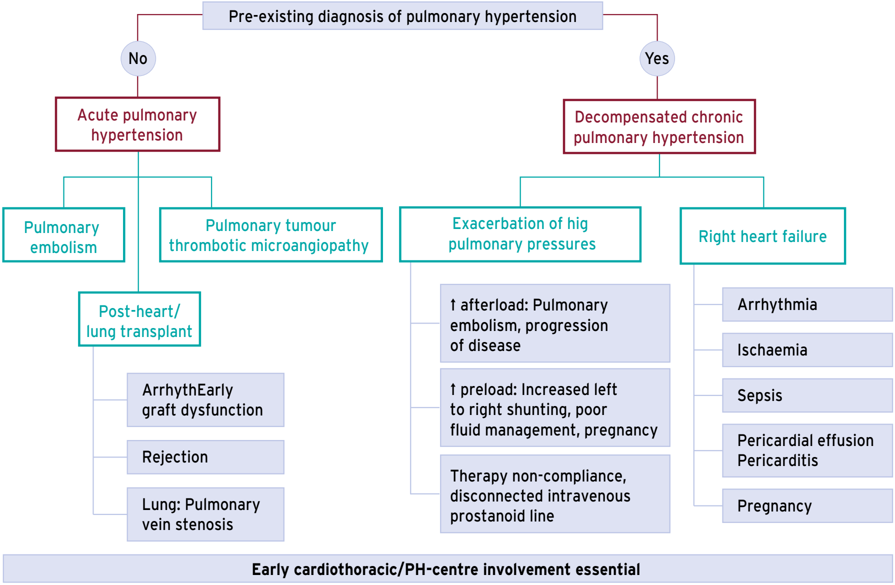
Diagnosis of pulmonary hypertension - Echocardiography (Cont.)

Estimated sPAP from TR gradient is not a diagnostic criterion by itself, and IVC-based RAP estimation has limitations - use the whole echo picture



Ref: 2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension, European Heart Journal, Volume 43, Issue 38, 7 October 2022, Pages 3618-3731 Figure 4

Classification of emergencies in pulmonary hypertension



Diagnostic workup

Initial critical care assessment

- Airway
- Breathing
- Circulation
- Disability
- Exposure

Evaluate exacerbating factors

- ECG for arrhythmia or ischaemia
- Temperature, chest x-ray and sepsis screen

Imaging assessment

- Echocardiography:
  - Pericardial effusion, RV (and LV) diastolic/systolic function, presence of shunts, valvular disease, fluid status (IVC)
  - Post-lung Tx transoesophageal to visualise pulmonary veins
- Radiology:
  - CTPA (/VQ) to look for embolism

Haemodynamic assessment

- Pulmonary artery catheter (PAC)
  - Right atrial pressure
  - Cardiac index
  - Central venous O2
  - Cardiac output measurement
- Arterial line
- Calculate PVR and record SvO_2/$ScvO_2$ trends as targets for response to therapy

Management: Key principles

Airway & breathing	Circulation
Hypercapnia and hyperinflation should be avoided as they increase pulmonary vascular resistance[1] Oxygen saturation should be kept at >90-92%[2] Intubation, mechanical ventilation and positive end expiratory pressure (PEEP) can precipitate haemodynamic deterioration – but may be needed in specific circumstances. Use the most experienced practitioner available[1,2]	Careful fluid management: both hypovolaemia and hypervolaemia are potentially detrimental[1] Inotropes to support CO if low; dobutamine and milrinone are the most frequently used[1] Pulmonary vasodilators to reduce afterload on a case-by-case basis[1] Mechanical circulatory support of the RV, most commonly with VA-ECMO but pRVAD is an option[1]

When to call the PH centre / retrieval team / ECMO on-call
· refractory hypotension · rising lactate · RV failure with low CI despite inotrope · deteriorating gas exchange

Management strategy: Airway and Breathing

Before intubation: making the decision		
High threshold in view of significant risk of deterioration	If required, decision should be made quickly & undertaken by an experienced practitioner	In a high-risk patient, consider whether ECMO team should be on standby during intubation

At the time of intubation: preventing circulatory collapse		
Induction of anaesthesia with sedatives/ muscle relaxants will suppress sympathetic drive	Consider inotropes and vasopressors as adjuncts to the induction of anesthesia	Consider use of the SAVIOR algorithm[1]

After intubation: optimise ventilation strategy		
Avoid hypercapnia, hypoxia and hyperinflation	Ensure ventilator synchrony	Close monitoring of haemodynamic response to change in ventilation strategy

[1]: Srour et al Journal of Cardiothoracic and Vascular Anesthesia 2020 34305-306

Adapted from Rajgopal et al, Evaluation and Management of Pulmonary Hypertension in Noncardiac Surgery: A Scientific Statement From the American Heart Association. Circulation. 2023;147:1317–1343

Proposed SAVIOR (Spontaneous, Awake Ventilation, Inotropes and Optimization of Respiratory physiology) Algorithm for the Physiologically Difficult Airway

Goals: Avoid paralytic, minimize effect of positive pressure ventilation on "lung pump", minimize dead space to tidal volume ratio and effective minute ventilation, maximize inotropic support while minimizing righ ventricle afterload and pulmonary vascular resistance

To be undertaken by a highly experienced practitioner

Management

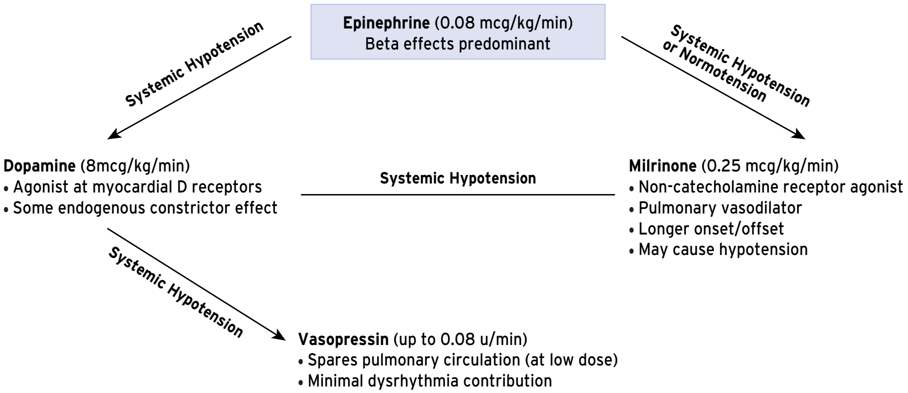
- Medications listed (drips, boluses, nebs.) in algorithm
- Bite block in place, scope introduction and lidocaine instillation
- Once coughing abates, enter cords, advance ETT and secure
- PS ventilator setting 0/0 with inhaled vasodilator in circuit, slowly increase PS and PEEP as tolerated
- Minimal FiO2 to minimize dead space

Airway equipment

- Nebulized Lidocaine
- Fiberoptic bronchoscope with ETT loaded
- Oxygen line at 15L/min for passive oxygenation

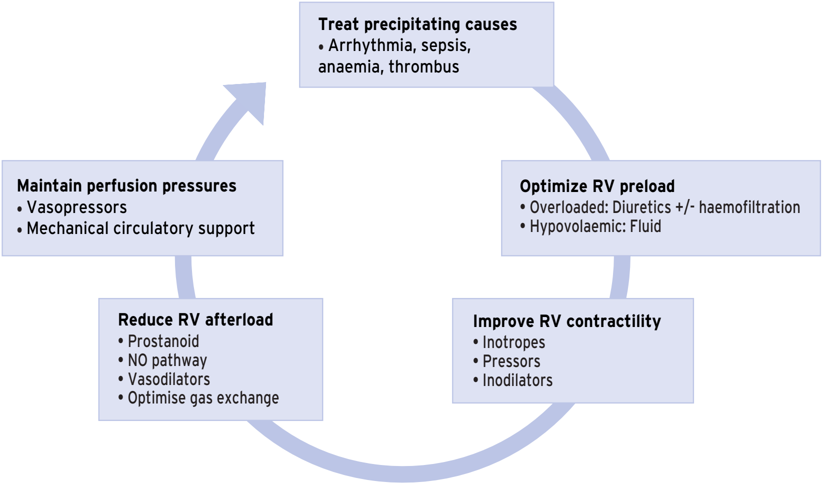
Adapted from: Srour et al Journal of Cardiothoracic and Vascular Anesthesia 2020 34305-306

Proposed SAVIOR (Spontaneous, Awake Ventilation, Inotropes and Optimization of Respiratory physiology) Algorithm for the Physiologically Difficult Airway



Adapted from: Srour et al Journal of Cardiothoracic and Vascular Anesthesia 2020 34305-306

Management strategy: Circulation



Parenteral anticoagulants

Class	Drug	Usual dose	Usual dose
Prostanoid	Epoprostenol	i.v.: 1–2 ng/kg/min, target 10 ng/kg/min after 48–72 h neb: 0.2–0.3 ml/min of 10–20 μg/ml	Reduction in PVR, may reduce SVR
	Iloprost	i.v.: 1–2 μg/h, target 6 ml/h after 48–72 h neb: 5 μg 7 times/day	Reduction in PVR, also reduce SVR
NO pathway	Nitric oxide	neb: 5–80 ppm	Reduction in PVR, rapid onset/offset (needs I&V)
	Sildenafil	i.v.: 10 mg tds	Reduction in PVR, may reduce SVR
Inotropes	Dobutamine	i.v.: 2.5–10 μg/kg/min (250 mg in 50 ml NS)	Tachycardia, increase CO
	Dopamine	i.v.: 0.5–10 μg/kg/min (200 mg in 50 ml NS)	Tachycardia, minimal increase in CO
Inodilators	Milrinone	i.v.: 50 μg/kg over 10 min, then 0.375–0.75 μg/kg/min neb: 0.2–0.3 ml/min	May cause profound hypotension
	Enoximone	i.v.: 0.5–1 mg/kg slow injection then 5–20 μg/kg/min	May cause profound hypotension
	Levosimendan	i.v.: loading 6–12 μg/kg/min over 10 min, then 0.1 μg/kg/min	May cause profound hypotension Slow onset/offset
Vasopressors	Norepinephrine	i.v.: 0.01–0.4 μg/kg/min (4 mg in 50 ml D5W)	May ↑PVR
	Phenylephrinine	i.v.: 40–100 μg bolus, 50–300 μg/min infusion	May ↑PVR, Bradycardia
	Metaraminol	i.v.: 1–5 mg/h (20 mg in 40 ml NS)	Peripheral or central administration
	Vasopressin	i.v.: 0.01–0.04 U/min (200 U in 250 ml D5W)	Bradycardia. Higher doses may be cardiodepressive and ↑PVR

Management strategy: Monitoring

Clinical signs

- Arterial pressure
- Diuresis
- Weight
- Arterial oxygen saturation
- Temperature
- Respiratory rate

Right cardiac catheterisation

- Right atrial pressure
- PA pressures
- Cardiac index
- Central/mixed venous oxygen saturation

Biomarkers

- BNP/pro-BNP
- Troponin
- Lactate
- Glomerular filtration rate
- Sodium levels
- Transaminases/bilirubin

Echocardiography

- Pericardial effusion
- Inferior vena cava dimensions (static & dynamic)
- Right atrial size
- Tricuspid regurgitation severity
- Parameters of RV function (TAPSE, Tei index)
- Parameters of LV function

Metrics for local quality monitoring

- Time to vasodilator
- Time to vasoactive support
- Time to PH-centre call
- Pre-/post-intervention mPAP/PVR/SvO_2

Monitor for signs of stabilisation

- Improving lactate and SvO_2/$ScvO_2$
- Rising urine output
- Down-trending RAP with stable MAP
- Improving TAPSE/TV annular S′

At-a-Glance Checklist

Team Activation

- Activate critical care team early
- Notify PH centre ASAP
- Assign clear team roles (airway, echo, drugs, communication)

Immediate Haemodynamic

Actions

- **Fluids:** Small cautious boluses only if needed
- **Inotropes**: Dobutamine or milrinone (support RV contractility)
- **Vasopressors:** Norepinephrine preferred (maintain perfusion)
- **Pulmonary vasodilators:** Inhaled NO or prostacyclin (consult PH centre)

ABCDE Priorities

- **A/B:** Avoid hypoxaemia and hypercapnia
  - High FiO_2, cautious ventilation (avoid acidosis/barotrauma)
  - Intubation with extreme caution, SAVIOUR algorithm
- **C:** Haemodynamic support
  - MAP > 65 mmHg; avoid RV overload
  - Cautious fluids: small boluses only if hypovolaemic
- **D/E:** Ongoing reassessment & decision-making, consider underlying cause

Echo Cues (POCUS/Focused TTE)

- RV dilation or septal flattening («D-sign»)
- Interventricular septum shift → LV underfilling
- IVC dilated and non-collapsing → elevated RAP
- Pericardial effusion? Consider tamponade

Don't Forget

- Early consultation with **specialist PH centre**
- Consider ECMO if:
  - Cardiac arrest or severe RV failure unresponsive to therapy
  - Bridge to recovery or transplant

At-a-Glance Checklist (Cont.)

Emergency Clue	Likely PH Group	Immediate Priorities	Specialist Consult
· Sudden decompensation in known PH patient	· **Group 1 (PAH)**	· Avoid hypoxia, cautious fluid, early inotrope, pulmonary vasodilators	· PH centre ± transplant/ECMO team
· RV failure with history of VTE or known CTEPH	· **Group 4 (CTEPH)**	· V/Q or CTPA, check anticoagulation, consider thrombolysis if PE	· Early referral to PEA/BPA centre
· Acute dyspnoea, hypoxia, leg swelling; no PH history	· **Group 4 (PE / undiagnosed CTEPH)**	· CTPA, initiate anticoagulation if confirmed/suspected PE	· Haematology or PH referral for follow-up
· Respiratory failure with chronic lung disease (e.g., COPD, ILD)	· **Group 3 (Lung disease)**	· Support oxygenation, avoid hypercapnia/ acidosis, cautious fluids	· Respiratory ± PH centre if unstable
· Hypoxaemia with known left heart disease (HFpEF/HFrEF)	· **Group 2 (LHD)**	· Diuresis, treat primary cardiac issue, avoid volume overload	· Cardiology; PH input if RV failure
· Young adult with syncope, loud P2, no obvious cause	· **Group 1 (Idiopathic PAH?)**	· Stabilise, avoid hypoxia/hypercapnia, early echo & referral	· Urgent PH centre discussion
· Chronic liver disease, new dyspnoea & hypoxia	· **Group 1 (Portopulmonary PH)**	· Oxygen, cautious fluids, evaluate for liver transplant referral	· Hepatology + PH centre

Red Flags - PVOD & CTEPH

PVOD

· Severe hypoxaemia, DLCO <40%
· CXR/CT: septal thickening, GGO
· Worsens with vasodilators

→ Urgent PH centre input

CTEPH

· Hx of VTE/unprovoked PE, dyspnoea on anticoagulation
· V/Q: mismatched defects
· CTPA: webs, ring stenoses

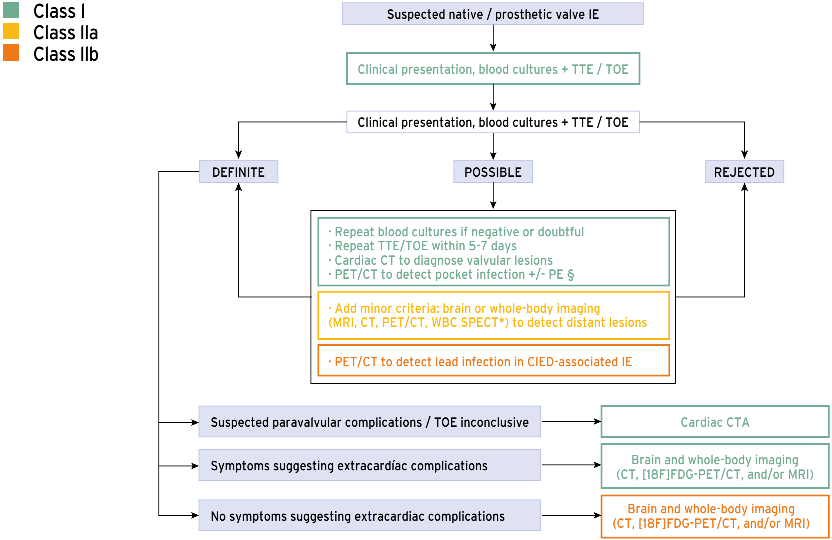
→ Refer early to PEA/BPA centre

CHAPTER 9.3

EMERGENCIES IN INFECTIVE ENDOCARDITIS

A.Sionis & S. Montero

Diagnostic Workflow of Infective Endocarditis



* First test in suspected prosthetic valve IE § Indicated in suspected CIED-associated IE

Diagnostic Criteria for Infective Endocarditis

Major criteria

(i) Blood cultures positive for IE:
(a) Typical microorganisms consistent with IE from two separate blood cultures:
Oral streptococci, Streptococcus gallolyticus (formerly S. bovis), HACEK group, S. aureus, E. faecalis
(b) Microorganisms consistent with IE from continuously positive blood cultures:

- ≥2 positive blood cultures of blood samples drawn >12 h apart.
- All of 3 or a majority of ≥4 separate cultures of blood (with first and last samples drawn ≥1 h apart).

(c) Single positive blood culture for C. burnetii or phase I IgG antibody titre >1:800.
(ii) Imaging positive for IE:
Valvular, perivalvular/periprosthetic and foreign material anatomic and metabolic lesions characteristic of IE detected by any of the following imaging techniques:

- Echocardiography (TTE and TOE).
- Cardiac CT.
- [18F]-FDG-PET/CT(A).
- WBC SPECT/CT.

Minor criteria

(i) Predisposing conditions (i.e. predisposing heart condition at high or intermediate risk of IE or PWIDs)a
(ii) Fever defined as temperature >38°C
(iii) Embolic vascular dissemination (including those asymptomatic detected by imaging only):

- Major systemic and pulmonary emboli/infarcts and abscesses.
- Haematogenous osteoarticular septic complications (i.e. spondylodiscitis).
- Mycotic aneurysms.
- Intracranial ischaemic/haemorrhagic lesions.
- Conjunctival haemorrhages.
- Janeway's lesions.

Diagnostic Criteria for Infective Endocarditis

Major criteria

(IV) Immunological phenomena:
- Glomerulonephritis.
- Osler nodes and Roth spots.
- Rheumatoid factor.

(V) Microbiological evidence:
- Positive blood culture but does not meet a major criterion as noted above.
- Serological evidence of active infection with organism consistent with

IE Classification (at admission and during follow-up)

Definite:
- 2 major criteria.
- 1 major criterion and at least 3 minor criteria.
- 5 minor criteria.

Possible:
- 1 major criterion and 1 or 2 minor criteria.
- 3-4 minor criteria.

Rejected:
- Does not meet criteria for definite or possible at admission with or without a firm alternative diagnosis.

Surgical Timing for Patients With Left-sided Infective Endocarditis

Indications for Emergency Surgery (<24 hours)	
Cardiogenic shock or pulmonary oedema	Class I
Indications for Urgent Surgery (within 3-5 days)	
Heart failure and poor haemodynamic tolerance	Class IIa
Local complications (abscess, false aneurysm, fistula, enlarging vegetation)	Class I
Persistent positive blood cultures	Class IIa
Resistant bacteria or fungi	Class I
PVE caused by S. aureus or non-HACEK Gram-negative bacilli	Class IIa
Vegetation >10 mm and emboli despite appropriate antibiotic therapy	Class I
Vegetation >10 mm and other reasons for surgery	Class I
Vegetation >10 mm and no evidence of embolus	Class IIb
Non-urgent Surgery (within hospital admission)	
Heart failure, uncontrolled infection or high risk of embolism or established embolism without any of the above conditions	Class I

Class I · Class IIa · Class IIb

CHAPTER 9.4

CARDIAC EMERGENCIES IN PREGNANCY

S.Gevaert & M. Bergami

Haemodynamic changes during pregnancy

Trimester	1st	2nd	3rd
Blood volume	↑	↑↑	↑↑↑
Cardiac output	↑	↑↑	↑↑↑
Stroke volume	↑	↑↑↑	↑-↓ or =
Heart Rate	↑	↑↑	↑↑-↑↑↑
Blood pressure	=	↓	=
Systemic vascular resistance	↓	↓↓↓	↓↓

In case of cardiac emergency during pregnancy:

Activate Pregnancy-Heart Team <15 min

- **Always: Obstetrician-Cardiologist-Intensivist-Anaesthetist-Neonatologist**
- **Upon indication: Interventional cardiologist, Cardiothoracic surgeon, Foetal imager, Nurse specialist(s)**

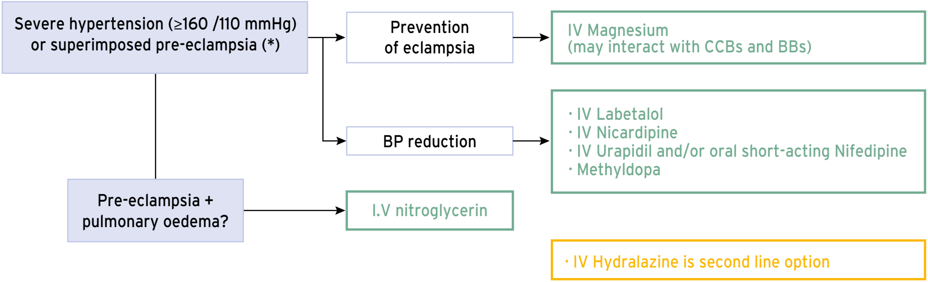
Diagnostic tools during pregnancy

	Indications	Comments
Careful history	Always	Known cardiac disease? Other symptoms?
Clinical exam	Always	Parameters, including pulse oximetry New murmur? Diastolic is always pathological.
BNP/NT proBNP	Suspicion of AHF	• Normal in healthy pregnant women • Can be elevated in (pre)eclampsia • Known heart disease: rise associated with worse outcome
High sensitive troponin	Suspicion of ACS	Normal in healthy pregnant women
D-dimer	Limited value	Usually elevated during normal pregnancy
ECG	HF/ACS/arrhythmia/ pulmonary embolism	Possible ECG changes during normal pregnancy • Leftward axis deviation • Transient ST depression/ flattened or inverted T waves • Q wave III
Echocardiography	Suspicion of HF/ACS/ Pulmonary embolism/amniotic fluid embolism, AAS	Changes during normal pregnancy: • Mild dilatation of chambers • Increased valve gradient
Lung ultrasound	Suspicion of HF	Preferred above Chest X-Ray

Diagnostic tools during pregnancy (Cont.)

	Indications	Comments
Chest X-ray	When clinically indicated	
Venous compression US	Suspicion of DVT or pulmonary embolism	
CT-angiography	High suspicion of pulmonary embolism or acute aortic syndrome AND other diagnostic tools are insufficient	Use low radiation CT: 0,01-0,66 mGy
Ionizing radiation	High suspicion pulmonary embolism and other diagnostic tools are insufficient: use perfusion scan	V/Q scan not advised
MRI	Limited indications	After 1st trimester, avoid gadolinium
Coronary angiography	STE-ACS or NSTE-ACS	Radial approach - ALARA (as low as reasonably achievable)
Electrophysiologocal exam	Refractory arrhythmia	Preference for non-fluoroscopic techniques

Hypertensive emergencies during pregnancy



SBP ≥ 160 mmHg or DBP ≥ 110 mmHg in a pregnant woman is an emergency and requires hospitalisation.

Women at high or moderate risk of pre-eclampsia should be advised to take 100-150 mg of aspirin daily from week 12 to weeks 36-37

(*) pre-eclampsia: gestational hypertension with significant proteinuria (>0.3 g/24 h or ACR >-30 mg/mmol) other maternal organ dysfunction (AKI, liver dysfunction, neurological complications, haematological complications) and uteroplacental dysfunction.

Medications for hypertensive emergencies during pregnancy

Medication	Safety in pregnancy	Dose adjustment	Foetal effects
ACE-is, ARBs, ARNIs aldosterone agonists	Contraindicated		Renal or tubular dysplasia, foetal renal failure, oligohydramnios, growth retardation, ossification skull disorders, lung hypoplasia, contractures, large joints, anaemia, and intrauterine foetal death
Labetalol	Considered safe	Yes, Increased renal clearance reduces half-life as pregnancy progresses	There is placental transfer Foetal hypoglycemia, bradycardia, hypotension, IUGR, respiratory depression
Hydralazine	Use with caution*		There is placental transfer. Foetal thrombocytopenia , tachyarrhythmia **Compared to other agents, increased risk of maternal or foetal complications** Teratogenic effects in animals
Nitroglycerine	Use with caution	i.v. infusion of 5µg/min, Gradually increase every 3-5 min to a maximum dose of 100 µg/min.	Unknown placental transfer
Nifedipine	Considered safe	Yes. Shorter dosing intervals due to accelerated liver metabolism	There is placental transfer **(foetal asphyxia caesarean delivery, prematurity, and intrauterine growth retardation)** Teratogenic and adverse effects in animals

*Can cause maternal lupus-like syndrome, reflex tachycardia

Medications for hypertensive emergencies during pregnancy (Cont.)

Medication	Safety in pregnancy	Dose adjustment	Foetal effects
Alfa- methyl dopa	Considered safe	No. Dose dependent hepatotoxicity in up to 1% of patients	There is placental transfer. Mild neonatal hypotension. Higher risk for pre-term birth Smaller head circumference in males
Nitroprusside	Use with caution	After 1st trimester, avoid gadolinium	There is placental transfer Few data in humans, risk for toxic levels of cyanide
Urapidil	Use with caution		Not enough data

Acute Heart Failure during pregnancy

Diagnostic clues to determine cause					
	Pre-existing cardiac disease	**Myocarditis**	**Peripartum CMP**	**ACS**	**(pre)-eclampsia-hypertensive HF**
History	Known/unknown cardiac disase Family History	Infection, fever, pericardial symptoms	De novo HF Other aetiologies exluded	Chest pain, nausea	Hypertension
Timing	-End of 2nd trimester -End of pregnancy -Postpartum		End of pregnancy or within months after	SCAD: end of pregnancy, early postpartum	End of pregnancy, can occur earlier
Biomarkers	Natriuretic peptides	Troponin Natriuretic peptides	Natriuretic peptides	Troponin ± Natriuretic peptides	Proteinuria Natriuretic peptides
ECG	Can reflect underlying disease: eg HCMP, RBBB, ...	Signs of pericarditis, ST-T changes	Non-specific	ST-T changes, can be normal	Usually normal
Echo	Structural abnormalities	Pericardial fluid Myocardial dysfunction	EF <45%, diffuse hypokinaesia	Regional wall motion abnormalities	Diastolic dysfunction LV function can be temporarily depressed

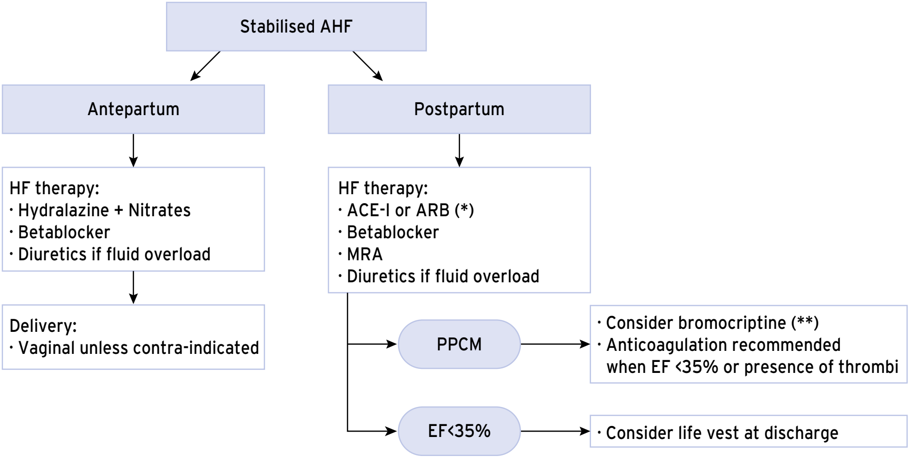
Treatment of Acute Heart Failure during pregnancy

Severe AHF-Shock during P	· Urgent transfer tertiary care centre · SHOCK team standby · Lung maturation if <35 weeks of gestation · URGENT C-section in case of shock
Congestion?	· IV diuretics: Furosemide preferred · Avoid overdiuresis
Systolic BP> 110mmHg?	· Vasodilators: Nitroglycerin preferred · Avoid hypotension
Hypoperfusion, multi-organ failure?	· Levosimendan preferred (*) · Alternative: Dobutamine or Milrinone
Refractory hypotension?	· Noradrenalin: MAP>65mmHg avoid adrenaline
PPCMP	· Consider bromocriptine (**) · Anticoagulation recommended when ejection fraction <35% or presence of thrombi or treatment with bromocriptine
Refractory shock-AHF?	Discuss MCS with Shock-team

(*) start low dose: 0.05-0.1 µg/kg/min for 24h

(**) • Severe: 2.5 mg BID for 2 weeks followed by 2.5mg OD for 6 weeks,
• Uncomplicated: 2.5 mg OD for 1 week
• Combine with anticoagulation

Treatment of Acute Heart Failure during pregnancy (Cont.)



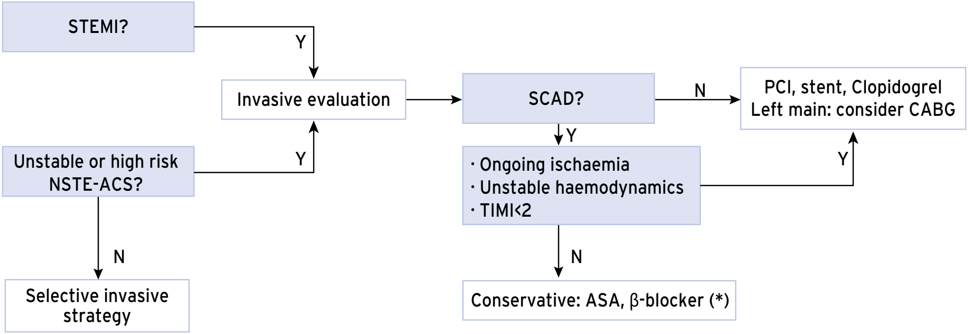
Contra-indicated during P:
- **SGLT2-I**
- **ACE-I and ARB**
- **Spironolactone and other MRA**

To avoid:
- **Atenolol, non-selective β-blockers**

(*) during lactation: enalapril, captopril safe

(**) • Severe: 2.5 mg BID for 2 weeks followed by 2.5mg OD for 6 weeks,
- Uncomplicated: 2.5 mg OD for 1 week
- Combine with anticoagulation
- Limited data on the use of bromocriptine

Management of ACS during pregnancy



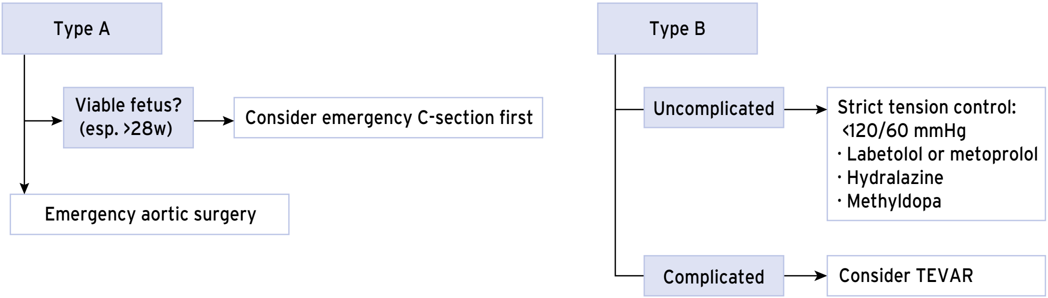
Contra-indicated during P:
- Thrombolysis in SCAD
- ACE-I, ARB and ARNI
- MRA
- SGLT2-I

To avoid:
- IIbIIIa antagonists
- Ticagrelor and Prasugrel
- DAPT in the absence of stent
- Atenolol

Case by case:
- Statins

Considered safe:
- (*)Beta-blocker (β1-slective: metoprolol preferred, avoid atenolol)
- Low dose Aspirin
- Nitrates
- Nifedipine

Acute aortic disease during pregnancy



Acute supraventricular arrhythmia during pregnancy

Haemodynamically unstable?

Y → Immediate DC cardioversion classic or antero-posterior pad position, foetal heart rate monitoring

N ↓

Atrial fibrillation/flutter

Cardioversion
· IV flecainide or ibutilide (*)
· DC cardioversion

Anticoagulation:
Follow guidelines as in non-pregnant, low threshold
· DOACS are contraindicated
· VKA in presence of mechanical valves or moderate to severe mitral stenosis
· If persistent/permanent AF at elevated thromboembolic risk: LMWH therapeutic dose

Rate control:
· IV betablocker e.g. Metoprolol or IV Verapamil
· Digoxin

Rhythmcontrol:
· Flecainide (*) plus betablocker
· Sotalol (**)

Supraventricular tachycardia

1. Vagal manoeuvres
2. IV adenosine

→ If unsuccesful: rate control with
1. IV metoprolol
2. IV verapamil

Pre-excitation

Re-entry:
1. Vagal manoeuvres
2. IV adenosine
3. Cardioversion

Atrial flutter/fibrillation:
· IV flecainide (*)
· IV ibutilide (*)

Contra-indicated during P:
• NOAC
•Amiodarone

Avoid during P:
• Atenolol

Use with caution during P:
• Diltiazem

(*) Control of pro-arrhythmic risk factors
(**) In the absence of structural heart disease

Acute ventricular arrhythmia during pregnancy

Haemodynamically unstable

Y

- Immediate DC cardioversion

If refractory or recurrent:

- IV Amiodarone

If idiopathic VF:

- IV isoproterenol
- PO Quinidine
- IV verapamil

N

Monomorphic VT

Unknown aetiology:
- IV beta-blocker

Structural heart disease:
1 · Routine IV betablocker
2 · IV procainamide

Idiopathic RVOT-VT:
- IV beta-blocker or IV adenosine

Fascicular VT:
- IV verapamil

Brugada syndrome:
- IV isoprotenerol
- PO Quinidine

Polymorphic VT

Long QT, Torsade de pointes:
- Remove precipitating factors
- IV betablocker (eg propranolol)
- IV Mg, K
- Overdrive pacing

CPVT:
- IV betablocker

Contra-indicated during P:

- Amiodarone (consider short term if refractory or recurrent ventrucular arrhythmia)

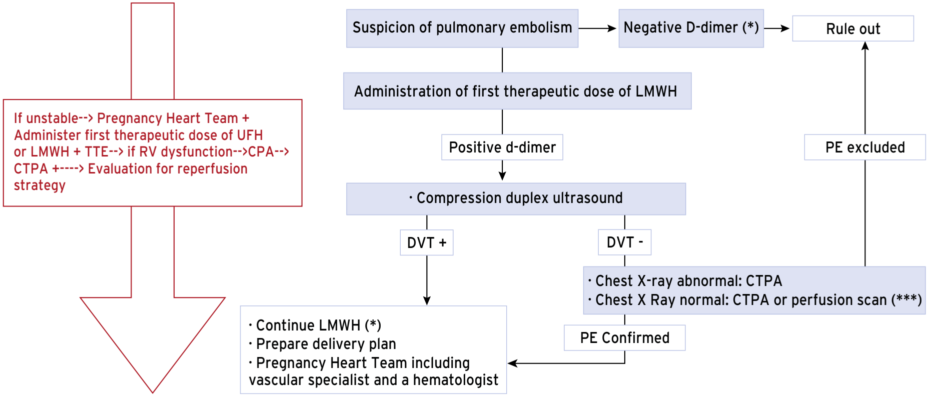
Use with caution during P:

- Diltiazem

Avoid during P:

- Atenolol

Pulmonary Embolism during pregnancy



(*) Usually elevated during pregnancy
(**) Either once or twice based on early pregnancy weight
- Consider antiXa dosing in high risk (extreme obesity, CKD, recurrent venous thrombo-embolism)
- Consider Fondaprinux as alternative for allergy or adverse events
- Stop at least 24h prior to delivery
- Convert to UFH 36h prior to delivery (stop UFH 4h pre deilvery)
- Duration: min three months from diagnosis and min 6 weeks after delivery

(***) Review by experienced radiologist, nuclear physician

Contra-indicated during P: NOAC

Considered safe during P and lactation
-LMWH
-UFH
-Low dose warfarin

Management of acute venous thromboembolism during pregnancy

Medication	Safety in pregnancy	Dosage
Unfractionated heparin*	Considered safe	Based on early pregnancy body weight
Low molecular weight heparin	Considered safe	

* Although not crossing the placenta, it is associated with higher risk of thrombocytopenia

Management of acute venous thromboembolism during pregnancy (Cont.)

Medication	Safety in pregnancy	Dosage
Fondaparinux	**Use with caution** Reasonable in case of Heparin-induced thrombocytopenia	Fondaparinux prophylactic dose: 2.5 mg daily; Therapeutic dose: up to 10 mg daily can be considered if there is an allergy or adverse response to LMWH
NOAC	**Contraindicated**	

In pregnant women with acute iliofemoral DVT, interventional thrombus removal should not routinely be performed.
Vena cava filters
Limited data. Their placement should be limited to recurrent VTE despite appropriate anticoagulation or contraindication to therapeutic-dose anticoagulation therapy

Thrombolysis
Thrombolytic or interventional treatment of PE is not recommended in the peripartum period and should only be considered in pregnant women with acute high-risk PE after consultation with a specialized multidisciplinary team

In pregnant or post-partum women with a diagnosis of acute high-risk PE, surgical thrombectomy may be considered as an alternative to a catheter-based approach or systemic thrombolysis

Amniotic fluid embolism

Diagnostic criteria: all must be present
- Sudden onset of cardiorespiratory arrest or hypotension
- DIC documented by low platelet count, increased INR and low fibrinogen level
- Onset during labor or within 30 minutes after placenta delivery
- Absence of fever

Other symptoms:
- Aura (chills, feeling unwell, change mental status,...)
- Bleeding
- Tonic-clonic seizures

Echocardiograpy:
- Rise in pulmonary pressure
- Followed by ventricular failure

Management:
- BLS/ALS
- Haemodynamic support with fluids, vasopressors
- Respiratory support, consider ECMO but high bleeding risk
- Management of DIC:
    - Platelet transfusion
    - FFP
    - If fibrinogen is <200 mg/dL: cryoprecipitate and FFP

Cardiac arrest during pregnancy

- Activate cardiac arrest team
- Document time of onset
- Place patient supine position, left lateral manual displacement of uterus or left lateral position of patient if >20 weeks
- Remove foetal monitors
- Start classic BLS/ALS algorithms
- Anterolateral defibrillator pad placement with the lateral pad placed under the breast
- IV access above the diaphragm
- If patients is on IV magnesium:
    - STOP magnesium immediately
    - ASAP: Give IV CaCL (10mL in 10% solution or Ca gluconate (30mL in 10% solution)

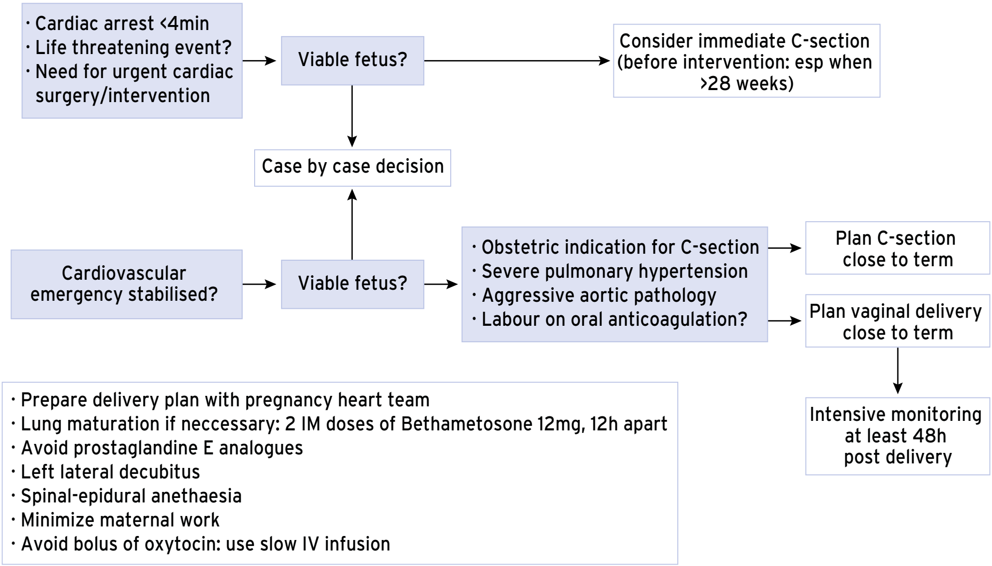
If not succesful <4 minutes and viable fetus:
Emergency C-section: aim at delivery <5 minutes from onset

Search and treat possible contributing factors: **BEAU-CHOPS** acronym

- **B**leeding/DIC
- **E**mbolism: coronary, pulmonary, amniotic
- **A**naesthetic complications
- **U**terine atony
- **C**ardiac disease (ACS, aortic dissection, cardiomyopathy)
- **H**ypertension: pre-eclampsia/eclampsia
- **O**ther: 4H and 4T
- **P**lacenta abruptio/previa
- **S**epsis

Based on AHA algorithm for cardiac arrest during pregnancy, 2010

Delivery

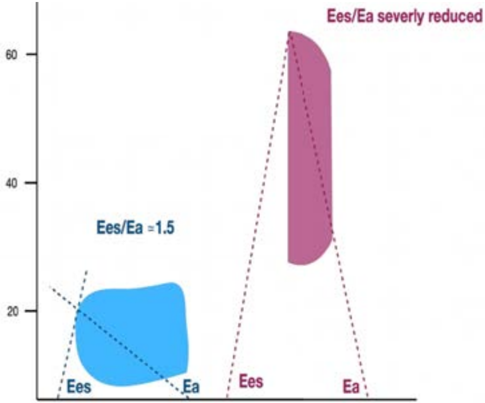

· Cardiac arrest <4min
· Life threatening event?
· Need for urgent cardiac surgery/intervention
Viable fetus?
Consider immediate C-section (before intervention: esp when >28 weeks)
Case by case decision
Cardiovascular emergency stabilised?
Viable fetus?
· Obstetric indication for C-section
· Severe pulmonary hypertension
· Aggressive aortic pathology
· Labour on oral anticoagulation?
Plan C-section close to term
Plan vaginal delivery close to term
Intensive monitoring at least 48h post delivery
· Prepare delivery plan with pregnancy heart team
· Lung maturation if neccessary: 2 IM doses of Bethametosone 12mg, 12h apart
· Avoid prostaglandine E analogues
· Left lateral decubitus
· Spinal-epidural anethaesia
· Minimize maternal work
· Avoid bolus of oxytocin: use slow IV infusion

CHAPTER 9.5

ACUTE RV FAILURE

G.Tavazzi & A. Zaher

RV Physiology and Pathophysiology



Physiology

RV ejects blood into the low-pressure, high-capacitance pulmonary circulatory system, able to accommodate large variation in venous return (preload) without significant rises in end-diastolic pressure and as afterload (Ea) is low during the whole cardiac cycle and fall throughout ejection.

Pathophysiology

Uncoupling occurs due to increase afterload (Ea) or ischemia (Ees) resulting in further right ventricular (RV) dilation and a drop in stroke volume, which may even cause a drop in pulmonary systolic pressures.

The right-upper shift of the PV loop requires a higher work (sum of stroke work and potential energy) to maintain the stroke volume.

- **Normal Ees/Ea 1.5-2**
- **Maximal transfer of potential energy form RV occurs with Ees/Ea =1**
- **Uncoupling Ees/Ea < 0.7-0.9**

Definition & cause

Acute Right Ventricular failure: Right ventricle (RV) inability to tolerate acute rise in pressure and volume loading conditions and generate enough stroke volume which will lead to systemic venous congestion, left ventricle underfilling and ultimately cardiogenic shock.

Reduced contractility

- Right ventricular infarction
- Inferior and posterior wall infarction
- Myocarditis
- Post – Cardiotomy syndrome
- Post Heart Transplant (Primary Graft Dysfunction)
- Left ventricular assisted device implantation
- Arrhythmias
- Sepsis

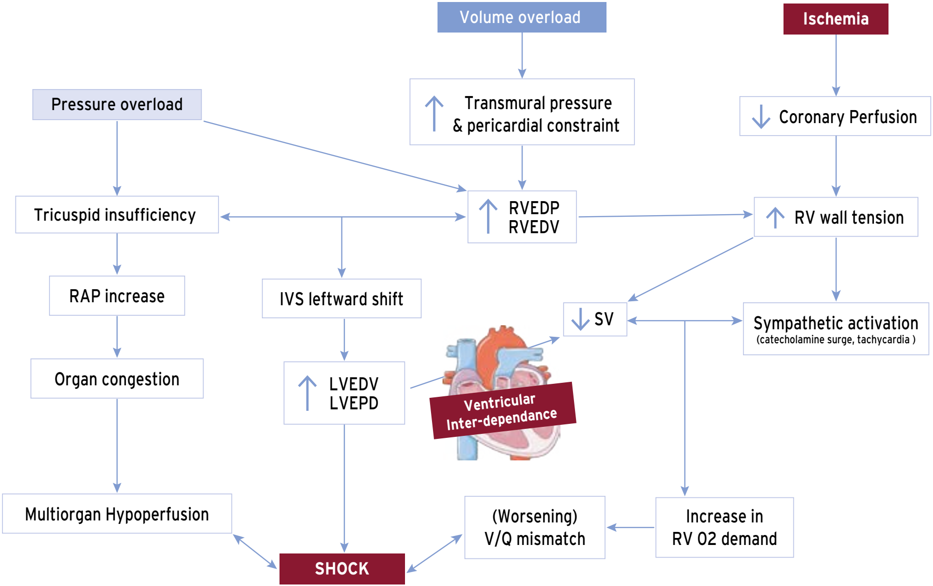
Volume overload

- Excessive fluid administration
- Transfusion associated cardiac overload
- Acute left ventricular failure
- Acute left to right shunting (VSD, ASD)

Pressure overload

- Acute respiratory distress syndrome (ARDS)
- Transfusion related acute lung injury
- Pulmonary contusions
- Severe Hypoxia/lung shunt
- Hypercarbia
- Acidosis
- Positive pressure ventilation
- Acute Pulmonary embolism
- Post Protamine in cardiac surgery

Pathophysiology



Clinical assessment framework from risk factors to presentation

Predisposing conditions	Familiar history
· Coronary artery disease, · LV failure or valvular disease, · CCOPD, · Thrombosis (PE) · Connective tissue disease, · Chronic infection or connective diseases, · Tobacco	· PAH, · LV failure · Arrythmogenic cardiomyopathy · Immune disorder
Symptoms	**Signs**
· Exertional dyspnoea · Orthopnoea · Fatigue · Dizziness · Syncope · Lower extremity edema · Exercise intolerance · Fatigue · Epigastric fullness · Right upper abdominal discomfort	· Jugular venous distension · Peripheral oedema · Ascites/anasarca · Hepatosplenomegaly · 3rd heart sound./loud P2 · Graham steel murmur · Ejection systolic murmur · Bradyarrhythmia, AV block · Ventricular arrythmias · Cardiac arrest

Biomarkers and Right heart waveforms

Troponin I or T (hs)-
Rule in/out ischemic aetiology and pulmonary embolism

D-dimer -
Reflect pro-thrombotic state and high in pulmonary embolism– no prognostic value

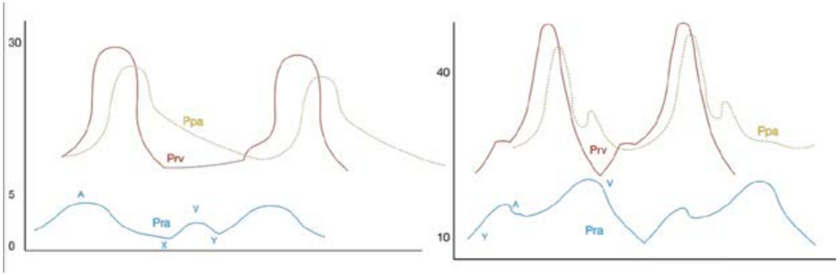
BNP or NT-proBNP-
marker of decompensation and worse prognosis but lack of sensitivity

Liver function
20/30% of severe liver injury in ARHF define as aminotransferase levels >20 times the upper limit of normal – Portal Doppler may be informative

Lactate -
Increase level due to sympathetic activation and hypoperfusion.
Liver and kidney dysfunction must be contextualized in case of no lactate clearance

Renal function
Renal dysfunction in up to 70% of ARHF secondary to primary congestion and/or hypoperfusion. Indicative of the cardio-renal syndrome

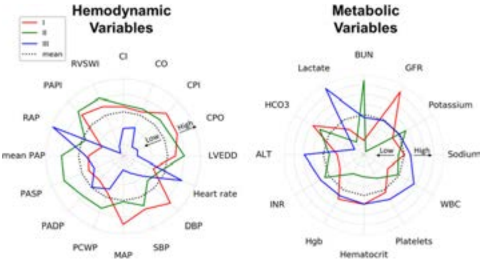
Biomarkers and Right heart waveforms



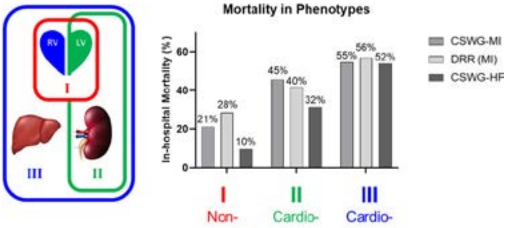
As **RV dysfunction** occurs:

diastolic waveform progressively changes from horizontal to an oblique aspect, followed by the appearance of a square root sign suggesting progressive loss in RV diastolic compliance. Concomitantly a progressive predominance of the V wave on the right atrial waveforms with a steep depression of the Y descendent.

Pulmonary Artery catheter & Hemodynamic Indices



Blues lines reflect trajectories of hemodynamic and metabolic variables in case of RV involvement.



Comprehensive evaluation of RV/ pulmonary hemodynamic may improve recourse allocation and outcome

RV involvement is associated with worse:

- organ dysfunction,
- metabolic profile
- prognosis

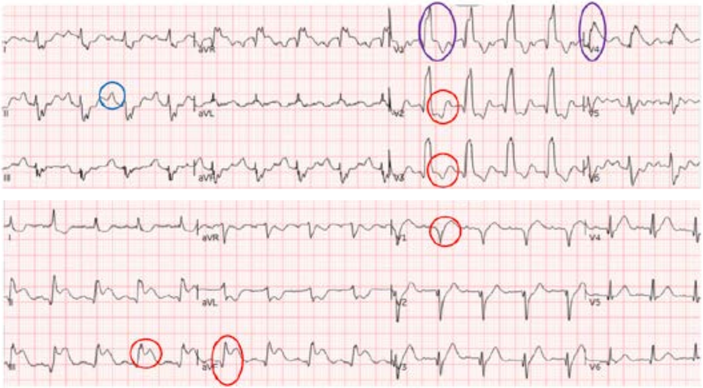
Ref: J Am Heart Assoc. 2021;10:e020085.

Hemodynamic of RV failure

Cardiac index	< 2.1 L/min/m²
RAP	12 or > 5 mmHg increase with fluid loading CVP waveform with flattering of X descendent, C-V fusion and tall V waves
RAP/PCWP	0.63(RVF after LVAD) > 0.86 (RA AMI)
PAPi (sPAP-dPAP)/RAP	< 1.85 (RVF after LVAD) < 1 RV-CS (not validated yet)
Pulmonary vascular resistances (PVR)	> 3 WU
Transpulmonary gradient (TGP) mPAP-PCWP	> 15 mmHg (in heart transplant) > 10 mmHg (RVF after LVAD)
Diastolic pulmonary gradient (DPG) dPAP - PCWP	> 7 mm Hg (RVF after LVAD)
RV stroke work (RVSV) (mPAP - RAP)/SV*0.0136	< 15 (RVF after LVAD) < 10 (RVF after acute MI)
RV stroke work index (RVSVi) (mPAP - RAP)/Sv index	< 0.3-0.6 (RVF after LVAD)
PA compliance SV/(sPAP-sPAP)	< 2.5 (RVF in chronic HF)

Ref: Circulation. 2017;136:314-326

ECG



RV strain

Qr and ST elevation in V1 ; V3/V4 reversal wave ; right bundle branch block

Ischemia

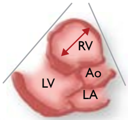
ST elevation in V1; ST depression in V2;ST elevation in III > II

Chronic RA enlargement

P pulmonale (P in II > 2,5 mm)

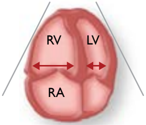
Echocardiography

A



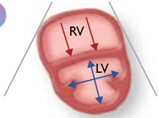
Enlarged right ventricle;
parasternal long-axis view

B



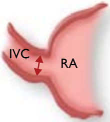
Dilated RV with basal RV/LV ratio >1.0;
four-chamber view

C



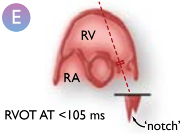
Flattened interventricular septum (arrows) leading to 'D-shaped' LV; decreased LV eccentricity index; parasternal short-axis view

D



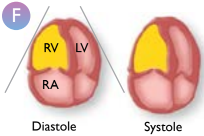
Distended inferior vena cava with diminished inspiratory collapsibility; subcostal view

E



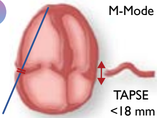
RVOT acceleration time of pulmonary ejection <105 ms mid-systolic 'notch' indicative of pre-capillary PH

F



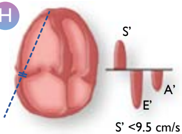
Reduced right ventricular fractional area change (<35%);
four-chamber view

G



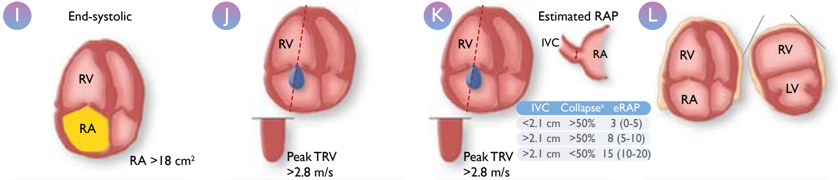
Decreased tricuspid annular plane systolic excursion (TAPSE) measured with M-Mode (<18 mm)

H



Decreased peak systolic (S') velocity of tricuspid annulus (<9.5 cm/s) measured with tissue Doppler

Echocardiography (Cont.)



Enlarged right atrial area (>18 cm^2); four-chamber view

Increased systolic peak tricuspid regurgitation velocity (peak TRV); measured with continuous wave Doppler

Estimation of systolic pulmonary artery pressure (sPAP); sPAP = TR pressure gradient + estimated RAP

Presence of pericardial effusion; four-chamber view; parasternal short-axis view; other views (e.g. subcostal view)

IN ACUTE SETTING

Most accurate/feasible

- **Longitudinal function** (TAPSE, S′ at TDI)
- **Size** (RA dilatation and RV/LV) ratio
- **Tricuspid regurgitation** and **Stroke volume**

- **Strain/speckle tracking/3D** may have limited application

- **Severe RV failure** – TR gradient underestimate true pulmonary pressure
- **Mechanical circulatory support** - evaluation must be contextualized with the level of support

Ref: 2022ESC/ERSGuidelinesforthediagnosisand treatment of pulmonary hypertension - European Heart Journal, Volume 43, Issue 38, 7 October 2022, Pages 3618–3731,Figure 4

Management

Etiological treatment

- AMI
- Pulmonary embolism
- Pressure overload (including ARDS)
- Obstructive shock

Volume optimization

- Aim for RAP 8-12
- Avoid fluid administration if signs of volume overload (RAP; RAP/PCWP, RV size, Jugular reflux/edema)
- Decongestive therapy (loop diuretics and thiazide for response augmentation – RRT)

PH treatment

- Epoprostenol (50 μg/kg/min i.h. – 0.5–2 μg/kg/min i.v.)
- Iloprost 2.5–5 μg/kg/min i.h.; 1 μg/hour i.v. (up-titration to 0.12 μg/kg/h)
- Treprostinil (3–30 ng/kg/min s.c.; 1.25–2.5 ng/kg/min i.v.)

RV inotropy

- Dobutamine (≤ 5 mcg/kg/min)
- PDE3i (milrinone ≤ 0.5 mcg/kg/min)
- Levosimendan (Once stabilization achieved – no bolus!) infusion 0.05 / 0.1 mcg/kg/min

Coronary / organ perfusion

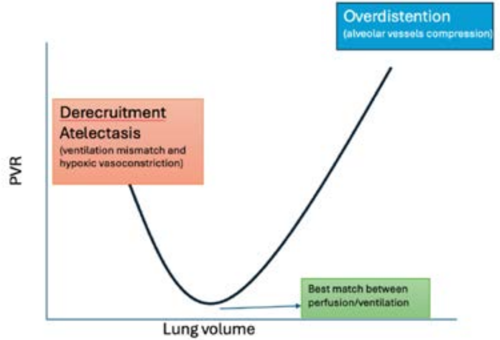
- Norepinephrine (0.1–0.25 mcg/kg/min)
- Vasopressin (0.01–0.04 UI/kg/min)

Heart rate and rhythm controln

- Anti-arrhythmic drugs
- Super selective short-acting β blockers (e.g. Landiolol)
- Pacemaker if implanted

Use always the lowest effective dose & for the shortest time

Respiratory management



***Avoid PPV if not mandatory**

If needed:

- **Consider awake intubation (call the expert!)**
- **If intubation required**

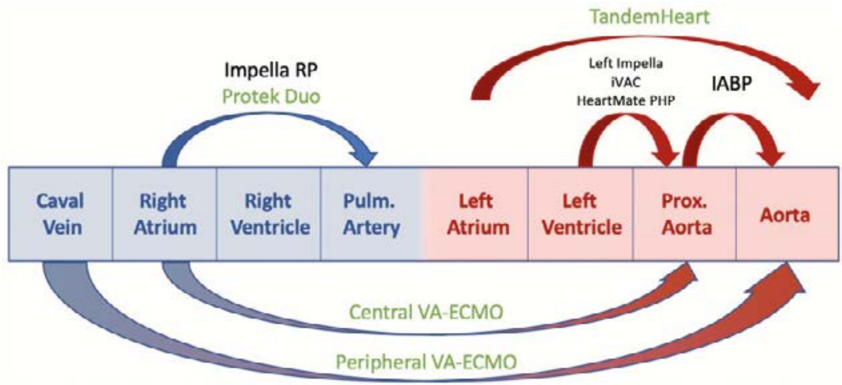
- Etomidate for induction may be preferred
- V-A ECMO stand-by on site

Initial settings iMV:

- PEEP < 5 cmH2O and Vt/Kg 6-8 ml/Kg
- Frequently alternate decubitus to avoid posterior consolidation (if hemodynamic allows)
- Pplat < 25 cmH2O & Driving pressure < 12 cmH2O
- PaO2/FiO2 ≥ 200 (avoid hyperoxia and hypercarbia)
- Wean ASAP !!!!!

PPV is positive pressure ventilation

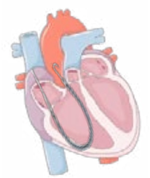
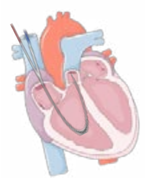
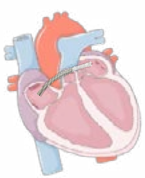
Mechanical circulatory support



Arrows indicate which part of the circulation is supported by the pVAD-modality

Ref: EHJ-ACVC, Volume 10, Issue 5, May 2021, Pages 570-583

Mechanical circulatory support

Impella RP/RP flex	Protek duo	Tandem Heart	ECMO
			

Effect	· Up to 4 L/Min · RV/RA Offloading	· Up to 4 L/Min · RV Offloading · Gas exchange	· Up to 4 L/Min · RA offloading	· Up to 4-6 L/Min · Biventricular support · Gas exchange
Drawback	↑ · PA pressure · LV preload · Challenging during weaning	↑ · PA pressure · LV preload · Larger bore access	↑ · PA pressure · LV preload · Limited availability in EU	↑ · LV afterload · Lung hypoperfusion

Ref: Circulation. 2017;136:314–326. DOI:10.1161/CIRCULATIONAHA.116.025290

Initial Diagnostic-therapeutic bundle

Multidisciplinary team consultation!!

Severity (SCAI) & Phenotype (SHARC) assessment

RV etiological & organ dysfunction management

Close SCAI re-assessment

Maintain/ upscale the treatment accordingly

Clinical evaluation

- Echo Focus/Echocardiography
- ECG
- Central vein, invasive arterial & pulmonary artery catheter monitoring
- Biomarkers
- Metabolic & organ function assessment

Establish and monitor therapeutic targets

Close Clinical Re-evaluation

- Echo Focus/Echocardiography
- ECG
- PAC
- Biomarkers
- Metabolic & organ function assessment

- Define if further diagnostic pathways are needed (e.g. CT angio/CMR is fesible)
- Define exit strategies and follow up pathways (including early cardiac rehabilitation process)
- Implement Guideline directed medical therapy

Preload

- RAP < 12mmHg
- RAP/PCWP < 0.65 mmHg

Hemodynamic

- MAP ≥ 60 mmHg
- Maintain SBP/sPAP ratio
- Maintain PVR/SVR ratio

Heart Rate

- Restore sinus rhythm (if possible)
- Treat (non compensatory) sinus tachycardia
- Avoid excessive bradycardia

Organ function

- PaO_2 and $PaCO_2$ close to normal
- Establish perfusion target (e.g lactates, SvO_2)
- Monitor and treat organ function (kidney/liver)
- Strict Infection control!!!!!

Abbreviations

APTT = Activated partial thromboplastin time
AB = Airway and breathing
ABG = Arterial blood gas
AADs = Antiarrhythmic drugs
AAS = Acute aortic syndrome
ACEI = Angiotensin converting enzyme inhibitor
ACLS = Advanced cardiovascular life support
ACS = Acute coronary syndrome
ACT = Activated clotting time
AD = Aortic Dissection
AED = Automated external defibrillator
AF = Atrial fibrillation
ANA = Antinuclear antibodies
Ao = Aortic
aPTT = Activated partial thromboplastin time
ARB = Angiotensin receptor blockers
AS = Aortic stenosis
AV = Atrioventricular
AVB = Atrioventricular conduction block
AVN = Atrioventricular node
AVNRT = Atrioventricular nodal re-entrant tachycardia
AVNT = Atrioventricular nodal tachycardia
BID = Twice a day
BBB = Bundle branch block
BLS = Basic life support
BNP = Brain natriuretic peptide
BP = Blood pressure
CABG = Coronary artery bypass grafting
CAD = Coronary artery disease
Cath Lab = Catheterisation laboratory
CCB = Calcium channel blockers
CCU = Coronary care unit
CHF = Congestive heart failure
CMR = Cardiovascular magnetic resonance
COPD = Chronic obstructive pulmonary disease
CPAP = Continuous positive airway pressure
CPR = Cardiopulmonary resuscitation
Cr = Creatinine blood level (mg/dL)
CrCl = Creatinine clearance
CRP = C-reactive protein
CS = Cardiogenic shock
CSM = Carotid sinus massage
CSNRT = Corrected sinus node recovery time

Abbreviations (Cont.)

CSS = Carotid sinus syndrome
CT = Computed tomography
CT-angio = Computed tomography angiography
cTn = Cardiac troponin
CUS = Compression venous ultrasound
CV = Cardiovascular
CVA = Cerebrovascular accident
CXR = Chest X-ray
DAPT = Dual antiplatelet therapy
DD = Dyastolic dysfunction
DM = Diabetes mellitus
dTT = Diluted thrombin time
DVT = Deep vein thrombosis
ECG = Electrocardiogram
Echo = Echocardiogram
ECMO = Extracorporeal membrane oxygenation
ECT = Ecarin clotting time
ED = Emergency department
EF = Ejection fraction
EG = Electrograms
eGFR = Estimated glomerular filtration rate (ml/min/1.73 m^2)
EMB = Endomyocardial biopsy
EMS = Emergency medical services
EPS = Electrophysiological study
ERC = European Resuscitation Council
ESR = Erythrocyte sedimentation rate
ETT = Endotracheal tube
FFP = Fresh frozen plasma
FiO_2 = Fraction of inspired oxygen
FMC = First medical contact
GER = Gastroesophageal reflux
GFR = Glomerular flow rate
GI = Gastrointestinal
GP = Glycoprotein
Hb = Haemoglobin
HF = Heart failure
HIT = Heparin-induced thrombocytopenia
HOCM = Hypertrophic obstructive cardiomyopathy
HTN = Hypertension
HR = Heart rate
hsTn = High-sensitive troponin
IABP = Intra-aortic balloon pump
ICC = Intensive cardiac care
ICCU = Intensive cardiac care unit

Abbreviations (Cont.)

ICD = Implantable cardioverter defibrillator
ICI = Immune checkpoint inhibitors
IHD = Ischemic heart disease
IMH = Intramural hematoma
IRF = Immediate-release formulation
ISFC = International Society and Federation of Cardiology
i.o. = Intraosseous
IV = Invasive ventilation
i.v. = Intravenous
KD = Kidney disease
LBBB = Left bundle branch block
LD = Loading dose
LGE = Late gadolinium enhancement
LMWH = Low-molecular weight heparin
LOC = Loss of consciousness
LV = Left ventricular
LVAD/Bi-AD = left ventricular, bi-ventricular assist device
LVD = Left ventricular dysfunction
LVEF = Left ventricular ejection fraction
LVH = Left ventricular hypertrophy
LVSD = Left ventricular systolic dysfunction
MCS = Mechanical circulatory support
MD = Maintenance dose
MDCT = Computed tomography with >4 elements
MI = Myocardial infarction
MRA = Mineralocorticoid receptor antagonist
MRI = Magnetic resonance imaging
Mvo = Microvascular obstruction
NIV = Non-invasive ventilation
NOAC = New oral anticoagulants
NSAID = Non-steroidal anti-inflammatory drugs
NSVT = Non-sustained ventricular tachycardia or recurrent
NSTE-ACS = Non ST-segment elevation acute coronary syndrome
NSTEMI = Non ST-segment elevation myocardial infarction
NTG = Nitroglycerin
NT-proBNP = N-terminal pro brain natriuretic peptide
NVAF = Non-valvular atrial fibrillation
NYHA = New York Heart Association

Abbreviations (Cont.)

OH = Orthostatic hypotension
PAP = Pulmonary arterial pressure
PAU = Penetrating aortic ulcer
PCI = Percutaneous coronary intervention
PCM = Physical counter-measures
PCP = Pulmonary capillary pressure
PE = Pulmonary embolism
PEA = Pulmonary endarterectomy
PEEP = Positive end expiratory pressure
PPC = Prothrombin complex concentrate
PR = Pulmonary regurgitation
PRECISE-DAPT = PREdicting bleeding Complications In patients undergoing Stent implantation and subsEquent Dual Anti Platelet Therapy
PRF = Prolonged-release formulation
ProCT = Procalcitonin
PRN = Pro re nata
PS = Pressure support
PS-PEEP = Pressure support-positive end-expiratory pressure
PSVT = Paroxysmal supraventricular tachycardia
QD = Once a day
QPM = Every evening
rFVIIa = Recombinant factor VIIa
rtPA = Recombinant tissue plasminogen activator
RV = Right ventricular
RVOT-VT = Right ventricular outflow tract ventricular tachycardia
SBP = Systemic blood pressure
s.c = Subcutaneous
SIRS = Systemic inflammatory response syndrome
SLE = Systemic lupus erythematosus
SMU = Syncope management units
STE-ACS = ST-segment elevation acute coronary syndrome
STEMI = ST-segment elevation myocardial infarction
SVT = Supraventricular tachycardia
Spo_2 = Oxygen saturation
TEE = Transesophageal echocardiography
TEVAR = Thoracic endovascular aortic repair
TIA = Transient ischemic attack
TID = Three times a day
TLOC = Transient loss of consciousness
TOE = Transoesopageal echocardiography

Abbreviations (Cont.)

TSH = Thyroid-stimulating hormone
TTE = Transthoracic echocardiography
UA = Unstable angina
UFH = Unfractionated heparin
ULN = Upper limit of normal
VBGA = Venous blood gas analysis
VF = Ventricular fibrillation
VR = Vascular resistance
VT = Ventricular tachycardia
VTE = Venous thromboembolism
VVS = Vasovagal syncope
WBC = White blood cell count
WHO = World health organization
WPW = Wolff-Parkinson-White

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Developed by the task force for the diagnosis and treatment of pulmonary hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society (ERS). Endorsed by the International Society for Heart and Lung Transplantation (ISHLT) and the European Reference Network on rare respiratory diseases (ERN-LUNG).

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Acknowledgements

We are indebted to all the authors for their commitment and for the strong effort to synthesise their wide scientific knowledge and clinical experience into simple algorithms and schemes using the aim to help clinicians in everyday clinical practice in the easiest possible manner as the main driver of their work.

The support of this initiative by the Association for Acute CardioVascular Care (ACVC) board members was essential to launch this initiative as was the hard work of the ESC staff to make this project move forward.

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