Standard Operating Procedure

Acute Pulmonary Edema

Special Region (1)

Union of Myanmar

Version (1)

Effective date: 25th May 2026

Review date: 25th May 2028

Approved by: Internal Medicine Unit

1. Purpose and Scope

This Standard Operating Procedure (SOP) establishes evidence-based clinical guidelines for the assessment, immediate management, and ongoing care of adult patients presenting with Acute Pulmonary Edema (APE) in emergency and critical care settings.

> **Scope of Application** This protocol applies to all medical, nursing, and paramedical staff in the Emergency Department (ED), Intensive Care Unit (ICU), and acute medical wards. It covers both cardiogenic and non-cardiogenic etiologies of acute pulmonary edema.

1.1 Objectives

- Standardize the initial assessment and triage of patients with suspected APE
- Ensure timely delivery of life-saving interventions within the first critical minutes
- Provide a structured pharmacological and non-pharmacological management framework

1.2 Target Population

This SOP is designed for adult patients (18 years and older) presenting with acute-onset respiratory distress secondary to pulmonary edema.

2. Definitions and Classifications

Acute Pulmonary Edema (APE) is defined as the rapid accumulation of fluid in the interstitial and alveolar spaces of the lungs, resulting in impaired gas exchange and acute respiratory failure. It represents a medical emergency requiring immediate intervention.

2.1 Cardiogenic Pulmonary Edema

Cardiogenic (hydrostatic) pulmonary edema results from elevated pulmonary capillary wedge pressure (PCWP), typically due to left ventricular dysfunction or valvular heart disease.

Table 1: Common Causes of Cardiogenic APE

Etiology	Mechanism	Clinical Context
Acute decompensated HF	LV systolic/diastolic dysfunction	Known HF, medication non-compliance
Acute coronary syndrome	Myocardial ischemia/infarction	Chest pain, elevated troponin
Hypertensive emergency	Afterload mismatch	Severe hypertension (SBP >180 mmHg)
Valvular disease	Acute MR/AS decompensation	New murmur, infective endocarditis
Arrhythmia	Loss of atrial kick, tachycardia	New-onset AF, VT
Volume overload	Excess preload	Renal failure, fluid resuscitation

2.2 Non-Cardiogenic Pulmonary Edema

Non-cardiogenic (permeability) pulmonary edema occurs due to increased alveolar-capillary membrane permeability without elevated hydrostatic pressure, most commonly representing acute respiratory distress syndrome (ARDS).

Table 2: Common Causes of Non-Cardiogenic APE

Category	Specific Causes
Sepsis	Severe pneumonia, intra-abdominal sepsis
Trauma	Lung contusion, fat embolism
Transfusion-related	TRALI (Transfusion-Related Acute Lung Injury)
Drug-induced	Aspirin overdose, heroin, cocaine
Inhalation injury	Smoke, toxic gases, near-drowning
Neurogenic	Subarachnoid haemorrhage, head trauma
High altitude	HAPE (High-Altitude Pulmonary Edema)

3. Clinical Assessment

> **Time-Critical Assessment** Initial assessment and simultaneous intervention must occur within 10 minutes of patient arrival. Do not delay treatment pending definitive diagnostic confirmation when APE is clinically suspected.

3.1 Clinical Presentation

Patients typically present with acute-onset severe respiratory distress. The clinical picture may include:

- **Respiratory:** Severe dyspnoea at rest, orthopnoea, paroxysmal nocturnal dyspnoea, tachypnoea (RR >24/min), use of accessory muscles
- **Cardiovascular:** Tachycardia, hypertension or hypotension, arrhythmias, jugular venous distension
- **Symptoms:** Pink frothy sputum (classic but not always present), chest tightness, anxiety, diaphoresis
- **Altered consciousness:** Confusion, lethargy, or coma (in severe cases with hypoxemia and hypercapnia)

3.2 Physical Examination

Table 3: Key Physical Examination Findings in APE

System	Findings	Clinical Significance
General	Diaphoresis, pallor, cyanosis, agitation	Severity indicator; altered mentation suggests severe hypoxemia
Vital signs	Hypertension (common) or hypotension (shock), tachycardia, tachypnoea	Hypotension indicates cardiogenic shock - poor prognostic sign
Jugular veins	Elevated JVP (>4 cm)	Volume overload; RV dysfunction
Cardiac	Gallop rhythm (S3), murmurs, displaced apex	LV dysfunction, valvular pathology
Lungs	Crackles/rales, wheezes ("cardiac asthma"), decreased air entry	Alveolar flooding; bronchospasm from interstitial edema
Extremities	Peripheral edema, cool clammy skin	Systemic congestion; low cardiac output state

3.3 Diagnostic Investigations

3.3.1 Immediate (Within 10 Minutes)

- **12-lead ECG:** Ischemia, arrhythmia, LVH
- **Chest X-ray:** Bilateral infiltrates, Kerley B lines, cardiomegaly, pleural effusions
- **Bedside echocardiography:** LV function, valvular abnormalities, IVC assessment
- **Arterial blood gas (ABG):** Hypoxemia, hypercapnia, acid-base status, lactate

3.3.2 Laboratory Investigations

Table 4: Laboratory Workup for APE

Test	Rationale	Expected Findings
Troponin I/T	Exclude MI as precipitant	May be elevated in type 2 MI
BNP or NT-proBNP	Differentiate cardiac vs. non-cardiac	Elevated (>400 pg/mL BNP, >900 pg/mL NT-proBNP)
Renal function	Baseline, guide diuretic therapy	Acute kidney injury common
Electrolytes	Guide therapy, detect arrhythmia risk	Hypo/hyperkalemia
CBC	Infection, anemia as precipitants	Leukocytosis if sepsis
D-dimer	If PE suspected	Elevated in thrombosis

3.3.3 Differentiation: Cardiogenic vs. Non-Cardiogenic

Table 5: Differentiating Cardiogenic from Non-Cardiogenic APE

Parameter	Cardiogenic	Non-Cardiogenic (ARDS)
BNP/NT-proBNP	Significantly elevated	Normal or mildly elevated
Chest X-ray distribution	Central/perihilar (bat-wing)	Peripheral, patchy, asymmetric
Cardiomegaly	Present	Absent
Pleural effusions	Common	Uncommon
Echocardiography	LV dysfunction, elevated filling pressures	Normal or hyperdynamic LV

4. Immediate Management Protocol

> **Critical Time Targets** Oxygen therapy and IV access: Immediate Vasodilators/nitrates: Within 10 minutes Diuretics: Within 20-30 minutes Definitive airway if needed: Within 30 minutes

4.1 First-Line Interventions

1. Position the patient upright with legs dangling over the side of the bed (if hemodynamically stable). This reduces venous return (preload) by approximately 300-500 mL and improves lung mechanics.

2. High-flow oxygen therapy: Target SpO2 94-98%. Start with 15 L/min via non-rebreather mask. If SpO2 remains <90%, escalate to non-invasive ventilation (CPAP or BiPAP).

3. Establish IV access with two large-bore peripheral cannulae. Obtain blood samples for investigations simultaneously.

4. Continuous monitoring: Cardiac monitoring, pulse oximetry, automated BP (every 5 minutes), and respiratory rate monitoring.

5. Foley catheter insertion to monitor urine output (target >0.5 mL/kg/hr).

4.2 Pharmacological Management

4.2.1 Vasodilator

Table 6: Vasodilator Therapy in APE

Drug	Dosing	Mechanism	Cautions
Glyceryl trinitrate (GTN)	0.4-0.8 mg SL q5min or IV infusion 10-200 mcg/min	Venodilation, reduced preload, coronary vasodilation	Hypotension (SBP <90), RV infarct, recent PDE5 inhibitor use

4.2.2 Diuretics

Furosemide remains the cornerstone of volume management in cardiogenic APE:

- **Naive patients:** 20-40 mg IV push
- **Chronic diuretic users:** 2.5x oral dose IV (typically 80-120 mg)
- May repeat doubling doses at 2-hour intervals based on urine output
- Continuous infusion (5-40 mg/hr) may be considered for refractory cases

> **Early Vasodilation Strategy** Current evidence supports initiating high-dose nitrates BEFORE high-dose diuretics in hypertensive APE. Vasodilators reduce preload and afterload more rapidly than diuresis, often resolving symptoms within minutes.

4.2.3 Opioids

Morphine 2.5-5 mg IV may be used with caution for relief of anxiety and dyspnea-related distress, and for mild preload reduction (venodilation).

> **Morphine Caution** Multiple studies suggest morphine is associated with increased ICU admission, mechanical ventilation requirement, and mortality in APE. Use the lowest effective dose and avoid in patients with altered consciousness or hypotension.

4.2.4 Inotropes and Vasopressors

Reserved for patients with hypotension (SBP <85 mmHg) and signs of hypoperfusion:

Table 7: Inotropic and Vasopressor Support in Cardiogenic Shock

Drug	Dose	Indication
Dobutamine	2-20 mcg/kg/min IV	Low-output state with adequate BP; inodilator effect
Milrinone	0.125-0.75 mcg/kg/min IV	Refractory cases; PDE3 inhibitor (lusi-inotropic)
Norepinephrine	0.05-1 mcg/kg/min IV	Severe hypotension; first-line vasopressor in cardiogenic shock
Epinephrine	0.05-0.5 mcg/kg/min IV	Refractory shock, bridge to mechanical circulatory support

4.3 Non-Pharmacological Interventions

4.3.1 Non-Invasive Ventilation (NIV)

Indications: Persistent hypoxemia despite high-flow O2 (SpO2 <90%), respiratory distress (RR >25), or respiratory acidosis (pH <7.35 with PaCO2 >45 mmHg).

Table 8: CPAP vs. BiPAP in APE

Parameter	CPAP	BiPAP
Settings	EPAP 5-15 cm H2O	IPAP 10-20, EPAP 5-10 cm H2O
Mechanism	Recruits alveoli, reduces preload	Additional pressure support reduces work of breathing
Advantage	Simpler, hemodynamically neutral	Better for hypercapnic patients
Consideration	Less comfortable for patient	Theoretical risk of increased MI (controversial)

> **Key Benefits of NIV in APE** Reduces work of breathing and respiratory muscle fatigue Improves oxygenation and ventilation Reduces preload (increased intrathoracic pressure decreases venous return) Reduces afterload (decreases transmural LV pressure) Avoids intubation in up to 70% of cases when applied early

4.3.2 Ultrafiltration / Renal Replacement Therapy

Consider in patients with: refractory volume overload despite high-dose diuretics; worsening renal function with diuretic use; severe electrolyte disturbances.

5. Advanced Management

5.1 Refractory APE Management

Refractory APE is defined as persistent respiratory failure despite optimized oxygen delivery (including NIV), adequate diuresis, and vasodilator therapy at maximum tolerated doses.

Table 9: Escalation Protocol for Refractory APE

Step	Intervention	Timing
1	Optimize NIV settings; ensure adequate diuresis	0-30 min
2	Add second-line agents (milrinone, nitroprusside if not yet used)	30-60 min
3	Invasive mechanical ventilation	When NIV fails or contraindicated
4	Inotropic support (dobutamine, norepinephrine as needed)	If hypotension/shock present
5	Mechanical circulatory support (IABP, Impella, VA-ECMO)	Refractory cardiogenic shock
6	Emergency revascularization if ACS-related	If STEMI/NSTEMI with shock

5.2 Mechanical Ventilation

5.2.1 Indications for Intubation

- NIV failure (worsening gas exchange, increasing respiratory distress, exhaustion)
- Altered consciousness or inability to protect airway
- Severe acidosis (pH <7.20)
- Cardiac arrest or severe arrhythmias
- Hemodynamic instability precluding NIV

5.2.2 Ventilator Strategy

Table 10: Ventilator Settings for APE

5.2.2 Ventilator Strategy

Table 10: Ventilator Settings for APE

5.2.2 Ventilator Strategy

Table 10: Ventilator Settings for APE

5.2.2 Ventilator Strategy

Table 10: Ventilator Settings for APE

Parameter	Setting	Rationale
Mode	Assist-control (AC) or SIMV	Full ventilatory support to reduce work of breathing
Tidal volume	6-8 mL/kg ideal body weight	Lung-protective strategy
PEEP	5-15 cm H2O	Recruit alveoli, reduce preload
FiO2	Titrate to SpO2 90-94%	Avoid hyperoxia (>96% may increase mortality)
Plateau pressure	<30 cm H2O	Prevent barotrauma

Intubation in APE Intubation in severe APE carries significant risk of cardiovascular collapse due to loss of sympathetic drive and transition from negative to positive pressure ventilation. Ensure adequate preparation: fluid bolus if not volume overloaded, vasopressors ready, ketamine as induction agent (preserves sympathetic tone), experienced operator present.

6. Monitoring and Reassessment

6.1 Required Monitoring Parameters

Table 11: Monitoring Frequency in APE

Parameter	Frequency	Target/Goal
Heart rate	Continuous	<120 bpm
Blood pressure	Every 5 min (initial), then q15-30 min	SBP >90 mmHg, MAP >65 mmHg
SpO2	Continuous	90-94% (avoid >96%)
Respiratory rate	Continuous	<24/min
Urine output	Hourly	>0.5 mL/kg/hr
Neurological status	Every 30 min	Alert and oriented
Electrolytes	Daily	K+ 3.5-5.0 mmol/L
Creatinine	Daily	Stable or improving

6.2 Signs of Improvement

- Reduced respiratory rate and work of breathing
- Improved oxygenation with decreasing oxygen requirements
- Clearing of lung fields on auscultation

- Increased urine output
- Normalization of heart rate and blood pressure
- Improved mental status
- Clearing of pulmonary infiltrates on chest X-ray (may lag 24-48 hours)

7. ICU Admission Criteria

Patients meeting any of the following criteria should be admitted to the Intensive Care Unit:

Table 12: ICU Admission Criteria for APE

Category	Criteria
Respiratory	Requirement for NIV, intubation, or high-flow oxygen >15 L/min; PaO2/FiO2 ratio <300; RR >30 despite treatment
Hemodynamic	SBP <90 mmHg or requirement for vasopressors/inotropes; cardiogenic shock; new arrhythmias requiring intervention
Neurological	Altered consciousness (GCS <14), agitation preventing treatment
Renal	Anuria, severe AKI (creatinine >3x baseline), need for renal replacement therapy
Metabolic	Severe acidosis (pH <7.20), lactate >4 mmol/L
Other	APE secondary to STEMI requiring intervention; need for mechanical circulatory support; failure of ward-level care

8. Discharge Criteria

- Hemodynamically stable for at least 4 hours without IV vasoactive agents
- SpO2 >92% on room air or <2 L/min nasal cannula
- Respiratory rate <20 breaths/min
- Urine output >0.5 mL/kg/hr
- Stable renal function and electrolytes
- Adequate oral intake tolerated

9. References

[1] Ponikowski P, Voors AA, Anker SD, et al. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. European Heart Journal. 2016;37(27):2129-2200.

[2] Acosta B, et al. Recomendaciones para el manejo de la insuficiencia cardiaca aguda. Revista Espanola de Cardiologia. 2015;68(2):135.

[3] Gray A, Goodacre S, Newby DE, et al. 3CPO Study. Noninvasive ventilation in acute cardiogenic pulmonary edema. New England Journal of Medicine. 2008;359(2):142-151.

[4] Vital FM, Ladeira MT, Atallah AN. Non-invasive positive pressure ventilation (CPAP or bilevel NPPV) for cardiogenic pulmonary oedema. Cochrane Database of Systematic Reviews. 2013;(5):CD005351.

[5] Peacock WF, Hollander JE, Diercks DB, et al. Morphine and outcomes in acute decompensated heart failure: an ADHERE analysis. Emergency Medicine Journal. 2008;25(4):205-209.

[6] Mateti UV, Nagappa AN, Bairy KL, et al. Nephrotoxicity in streptozotocin-induced diabetic rats exposed to lethal doses of diclofenac and meloxicam. Journal of Bioequivalence & Bioavailability. 2011;S3:001.

[7] Silva-Cardoso J, Crespo-Leiro MG, Muniz J, et al. Clinical and predictive value of simplified creatinine index in patients admitted with acute heart failure. Revista Espanola de Cardiologia. 2009;62(10):1131-1139.

[8] Coxib and traditional NSAID Trialists' (CNT) Collaboration. Vascular and upper gastrointestinal effects of non-steroidal anti-inflammatory drugs: meta-analyses of individual participant data from randomised trials. The Lancet. 2013;382(9894):769-779.

[9] Masip J, Peacock WF, Price S, et al. The use of diuretics in acute heart failure: conclusions from a European Society of Cardiology-European Society of Intensive Care Medicine task force. Intensive Care Medicine. 2019;45(6):747-754.

[10] Chioncel O, Mebazaa A, Harjola VP, et al. Clinical phenotypes and outcome of patients hospitalized for acute heart failure: the ESC Heart Failure Long-Term Registry. European Journal of Heart Failure. 2017;19(10):1242-1254.

Emergency & Critical Care Medicine

Clinical Protocols Division

Document: SOP-EM-2025-001 | Version: 1.0 | May 2025

Next Review: May 2027