Clinical Practice Guidelines:
Respiratory/Acute pulmonary oedema

Policy code	CPG_RE_ACO_0417
Date	April, 2017
Purpose	To ensure consistent management of acute pulmonary oedema
Scope	Applies to Queensland Ambulance Service (QAS) clinical staff.
Health care setting	Pre-hospital assessment and treatment.
Population	Applies to all ages unless stated otherwise.
Source of funding	Internal – 100%
Author	Clinical Quality & Patient Safety Unit, QAS
Review date	April, 2020
Information security	UNCLASSIFIED – Queensland Government Information Security Classification Framework.
URL	https://ambulance.qld.gov.au/clinical.html

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Acute pulmonary oedema

April, 2017

Acute pulmonary oedema (APO) refers to the rapid buildup of fluid in the alveoli and lung interstitium that has extravasated out of the pulmonary circulation. As the fluid accumulates, it impairs gas exchange and decreases lung compliance, producing dyspnoea and hypoxia.[1] The pathophysiological mechanisms are traditionally categorised into two primary causes:

Cardiogenic

Cardiogenic APO occurs when cardiac output drops despite an increased systemic resistance, so that blood returning to the left atrium exceeds that leaving the left ventricle (LV). As a result, pulmonary venous pressure increases, causing the capillary hydrostatic pressure in the lungs to exceed the oncotic pressure of the blood, leading to a net filtration of protein poor fluid out of the capillaries.[1,2]

Examples include:

- *Left ventricular failure (LVF):*
  - Acute Coronary Syndromes (ACS)
  - Arrhythmia
  - Pericarditis, myocarditis or endocarditis
  - Valve dysfunction (e.g. aortic stenosis, mitral regurgitation)
- *Increased intravascular volume:*
  - Fluid overload
  - Non-compliance with fluid restriction or diuretics
  - Renal failure
- *Pulmonary venous outflow obstruction:*
  - Mitral valve stenosis

Non-cardiogenic

Pathological processes acting either directly or indirectly on the pulmonary vascular permeability are thought to cause this form of APO. As a result, proteins leak from the capillaries, increasing the interstitial oncotic pressure, so that it exceeds that of the blood and fluid is subsequently drawn from the capillaries.[1,2]

Examples include:

- *High output states*
  - Septicaemia
  - Anaemia
  - Thyrotoxicosis
- *Systemic increase of vascular permeability*
  - Pancreatitis
  - Eclampsia
  - Disseminated Intravascular Coagulation (DIC)
  - Burns
- *Toxins/environmental*
  - Immersion/submersion
  - Toxic inhalation
  - High Altitude Pulmonary Oedema (HAPE) & decompression illness
- *Other*
  - Head Injury/intracranial haemorrhage
  - Drugs (e.g. NSAIDs, calcium channel blockers and naloxone)
  - Pulmonary embolus

Clinical features

- Sudden onset of extreme breathlessness, anxiety, and the feeling of drowning
- Profuse diaphoresis
- Crackles are usually heard at the bases first; as the condition worsens, they progress to the apices.
- Cough is a frequent complaint that suggests worsening pulmonary oedema in patients with chronic LV dysfunction.
- Pink, frothy sputum may be present in patients with severe disease.
- Tachypnoea and tachycardia
- Hypertension is often present because of the hyperadrenergic state.
- Hypotension indicates severe left ventricular and cardiogenic shock.
- Cyanosis (late sign)
- Raised jugular venous pressure

Risk assessment

- Regardless of the aetiologies between cardiogenic and non-cardiogic pulmonary oedema the presenting features of dyspnoea and tachycardia remain the same.[3]

Additional information

- Cardiogenic pulmonary oedema patients often have a history of cardiac hypertrophy/Acute Myocardial Infarction (AMI) and/or LVF.
- The primary goal in the treatment of cardiogenic pulmonary oedema is reduction in preload and afterload with nitrates.
- All patients with APO should be given supplemental oxygen as required to meet their physiological needs and reduce hypoxia.
- Patients with cardiogenic shock and concurrent respiratory failure from APO require CCP support where available. These patients may have a fluid deficit, therefore cautious fluid bolus (250–500 mL maximum) resuscitation should be titrated against haemodynamics and clinical effect. Inotropic support may be required to increase cardiac output.
- Non-cardiogenic APO requires respiratory support (with lung protection ventilation strategies) and treatment of the underlying cause.[3,4]

Note: *Clinicians are only to perform procedures for which they have received specific training and authorisation by the QAS.*

CPG: Clinician safety
CPG: Standard cares

Appropriate positioning

Determine cause of oedema

Cardiogenic?

Consider:

- Oxygen
- Aspirin
- GTN
- 12-Lead ECG
- IPPV
- PEEP
- CPAP

Manage as per appropriate CPG:

- CPG: Relevant dysrhythmia
- CPG: Acute coronary syndrome

Is the patient hypotensive?

Y

Manage as per:

CPG: Cardiogenic shock

N

Transport to hospital
Pre-notify as appropriate

Non-cardiogenic?

Consider:

- Oxygen
- 12-Lead ECG
- IPPV
- PEEP
- CPAP

Manage as per appropriate CPG:

- CPG: Burns
- CPG: Post Submersion
- CPG: Head injury
- CPG: Spinal injury
- CPG: Relevant toxicology/toxinology