INTRODUCTION

Pulmonary oedema is a condition that results from the accumulation of fluid in the lungs. Fluid congestion decreases gas exchange across the alveoli, resulting in decreased oxygenation of the blood and, in some cases, accumulation of carbon dioxide (CO_2).

The pathophysiology of pulmonary oedema can be thought of in terms of three factors:

1. flow
2. fluid
3. filter.

Flow

The ability of the heart to eject the blood delivered to it depends on three factors:

1. the amount of blood returning to the heart (preload)
2. the co-ordinated contraction of the myocardium
3. the resistance against which it pumps (afterload).

Pre-load may be increased by over-infusion of IV fluid or fluid retention associated with renal failure. Co-ordinated contraction fails following heart muscle damage (myocardial infarction (MI), heart failure) or due to arrhythmias. After-load increases with hypertension, atherosclerosis, aortic valve stenosis or peripheral vasoconstriction.

Fluid

The blood passing through the lungs must have enough 'oncotic' pressure to 'hold on' to the fluid portion as it passes through the pulmonary capillaries. As albumin is a key determinant of oncotic pressure, low albumin states lead to pulmonary oedema, e.g. burns, liver failure, nephrotic syndrome.

Filter

The capillaries through which the fluid passes may increase in permeability, e.g., acute lung injury (as in smoke inhalation), pneumonia or drowning.

The commonest cause of pulmonary oedema presenting to UK Ambulance Services is secondary to acute heart failure.[1]

The overall prevalence of heart failure varies between 1-2%, varying with age. 80% of these people will be diagnosed following acute admission to hospital.[1] Approximately 30% will be re-admitted to hospital each year.[2]

The signs and symptoms of pulmonary oedema can be difficult to differentiate from other causes of breathlessness, such as exacerbation of chronic obstructive pulmonary disease (COPD), pulmonary embolism or pneumonia. Therefore, a thorough history and physical examination are needed. Accuracy of Paramedic assessment of acute left ventricular failure (LVF) varies between 77% and 89%[3-5] when compared to physician in-hospital diagnosis.

HISTORY

See dyspnoea guidelines for evidence-based differential diagnoses.

Symptoms:	dyspnoea worsening cough (productive of white sputum) pink frothy sputum waking at night gasping for breath breathlessness on lying down (sleeping on more pillows recently?) anxious / restless.
Symptoms of MI may be associated with:	ankle oedema chest pain worsening of angina.
Previous history:	admissions for 'heart failure', 'fluid on legs/lungs' previous MI / angina / angioplasty /coronary artery bypass grafting diabetes hypertension.
Current medication:	home oxygen ACE inhibitors beta-blockers diuretics anti-arrhythmic drugs.
Other risk factors for heart disease:	smoking family history high cholesterol diabetes.

ASSESSMENT

Primary Survey: Assess ABCD

Monitoring and baseline observations:

- respiratory rate
- pulse
- blood pressure (BP)
- Initial 3-lead ECG followed by 12-lead ECG.

Specifically assess:

- excessive sweatiness or clamminess
- carotid pulse (rate, rhythm) – tachycardia common
- BP may be high (>170/100), or low in extremis
- raised jugular venous pressure
- central cyanosis.

Chest

- respiratory rate and effort
- fine inspiratory crackling heard over the bases
- wheeze may indicate either asthma or pulmonary oedema
- pitting oedema to the ankles often associated.

ECG changes

ECG may show signs of:

- acute MI
- arrhythmia
- heart strain
- hypertrophy.

Evaluate TIME CRITICAL factors:

- extreme breathing difficulty
- central cyanosis
- hypoxia i.e. oxygen (O_2) saturation levels (SpO_2) <95% or not responding to high concentration O_2 (***refer to dyspnoea guideline***)
- exhaustion
- decreased level of consciousness
- systolic blood pressure (BP) <90mmHg, or tachycardia in beats per minute numerically exceeds systolic BP mmHg measure.

If any of these features are present, start correcting **A** and **B** problems, give high concentration **O_2, LOAD and GO** to **nearest suitable receiving hospital** and treat en route. Provide the hospital with **an alert message / Information call.**

MANAGEMENT

NOTE: Remember that in a significant proportion of patients, the underlying cause will be acute MI. If suspicious, follow the acute coronary syndrome guideline.

Follow **medical emergencies guidelines** remembering to:

start correcting:

- **A**IRWAY
- **B**REATHING
- **C**IRCULATION
- **D**ISABILITY (mini neurological examination).

Administer high concentration oxygen (O_2) via a non-re-breathing mask, using the stoma in laryngectomee and other neck breathing patients, to ensure an oxygen saturation (SpO_2) of >95%, except in patients with chronic obstructive pulmonary disease (COPD) (***refer to COPD guideline***).

Consider assisted ventilation at a rate of 12–20 breaths per minute if:

- oxygen saturation (SpO_2) is <90% on high concentration O_2
- respiratory rate is <10 or >30
- expansion is inadequate.

Sit the patient upright / prop the trolley up.

Prepare equipment for respiratory or cardiac arrest.

Administer **glyceryl trinitrate** (GTN) (***refer to the GTN drug protocol for dosages and information***), assess for response.

Gain IV access where possible en-route to hospital.

Administer furosemide (***refer to the* furosemide *drug protocol for dosages and information)***.

Apply continuous positive airway pressure (CPAP) if equipment and training allow, if respiratory distress continues after 10 minutes, as evidenced by:

- persisting tachypnoea (>24 breaths per minute)
- persisting hypoxia (central cyanosis or saturations <90%).

Reassess the patient and reconsider the diagnosis.

If wheeze is a predominant feature, administer salbutamol by nebuliser (***refer to the salbutamol drug protocol for dosages and information***).

Continuous Positive Airway Pressure (CPAP)

CPAP is a single level of positive pressure applied throughout the whole respiratory cycle. Its use requires specialist equipment and training.[6]

- By providing constant pressure, the alveoli are 'splinted open' and gas exchange is promoted throughout the lungs
- Three prospective randomised controlled trials have looked into the use of CPAP in emergency department patients.[7-9] These and others conclude that it is a feasible intervention, which improved survival to hospital discharge, decreased intubation rates and resulted in fewer complications. Importantly, the average age of trial participants was comparable to the population likely to be encountered by Paramedics
- Three pre-hospital studies exist suggesting CPAP is feasible in this setting, and may reduce severity of acute LVF and increase SpO_2 levels.[10-12] Expert opinion has recommended CPAP for use in the pre-hospital environment.[13-15]
- Exclude contra-indications:
  - high likelihood of alternative diagnosis
  - hypotensive (systolic <90mmHg)
  - patients <V on AVPU scale
  - suspected MI
  - renal patients requiring dialysis
  - vomiting
  - unable to tolerate the tight-fitting face mask
  - use an initial starting pressure of 10cmH_2O

Glyceryl Trinitrate (GTN)

The use of nitrates in pulmonary oedema is associated with improved survival to hospital discharge in retrospective cohort studies.[16]

Buccal nitrates produce an immediate reduction in pre-load, comparable with IV GTN.

Nitrates have some benefit as the first line treatment in acute pulmonary oedema.[17]

Furosemide

There is little high-level evidence for or against the use of **furosemide** (***refer to furosemide protocol for dosage and information***) in the treatment of acute pulmonary oedema, but it has been standard treatment for many years.

There is some evidence that furosemide can have a transient adverse vasoconstrictor effect; it is unclear whether this is beneficial or harmful.[18-20]

The acute vasodilator effect of furosemide is inhibited by aspirin.

Pre-hospital trials comparing repeated furosemide vs. repeated nitrates favour the use of nitrates.[21]

Furosemide should only be given after nitrates (which act on both pre-load and after-load).

Other Treatments

The effectiveness of salbutamol in the treatment of pulmonary oedema presenting in the acute setting is unclear. However, owing to the diagnostic uncertainty and possibility for misdiagnosis,[5] it forms part of the management algorithm; this may avoid depriving COPD/asthma patients of vital bronchodilators.

Morphine and diamorphine are commonly used in the in-hospital emergency management of pulmonary oedema. The drugs act by reducing pre-load (venodilation) and also serve to decrease anxiety. Despite their widespread use, there is no conclusive trial evidence showing symptomatic improvement or mortality benefit. There is some concern over their safety for the pre-hospital management of pulmonary oedema[5] and Paramedics currently only have legal authority to administer morphine in order to provide analgesia (***refer to morphine drug protocol for dosage and information***).

Key Points – Pulmonary oedema

- Pulmonary oedema can be difficult to differentiate from other causes of breathlessness, such as exacerbation of COPD, pulmonary embolism or pneumonia; therefore, a thorough history and physical examination are needed
- Symptoms include dyspnoea, worsening cough, pink frothy sputum, waking at night gasping for breath, breathlessness on lying down (sleeping on more pillows recently?), and anxiousness/restlessness
- Prepare equipment for respiratory or cardiac arrest
- Early oxygen and nitrate administration are the key to early treatment
- Sit the patient upright.

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METHODOLOGY

Refer to methodology section.