

Suction, No Suction or Passive Drainage for Pulmonary Oedema

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There are several causes of pulmonary oedema (PE) in the perioperative period and Intensive Care Unit. A combination of cardiogenic and non-cardiogenic mechanisms usually contributes to the pathogenesis of PE, including negative pressure PE, anaphylaxis, acute lung injury, fluid maldistribution and neurogenic and cardiogenic PE (1). All anaesthesiologists are familiar with the management of PE. The mainstay of treatment remains IPPV with positive end-expiratory pressure (PEEP), diuretics, morphine and treatment of the inciting cause. Mild cases respond well once treatment is initiated along these lines. However, occasionally severe cases, which involve frothing of copious secretions from the endotracheal (ET) tube, are difficult to manage.

The suction pressure recommended for ET suctioning is 11–16 kPa (80–120 mmHg) using a catheter of diameter no greater than half the internal diameter of the tracheal tube for a maximum suction time of 15 seconds (2). Although there is no absolute contraindication, it is generally accepted that ET suctioning for acute PE should not be frequently performed because it creates a negative alveolar pressure, harming already damaged capillaries and entraining more oedema fluid. In fact, Pang et al. reported a case wherein a negative intrathoracic pressure of 100 mmHg, generated for 3–4 seconds by direct suctioning at the ET tube adapter, in a young healthy male patient led to PE similar to that induced by laryngeal spasm during emergence from general anaesthesia (3).

In severe PE, secretions may find their way into the carbon dioxide (CO₂) sampling line and the anaesthesia breathing circuit, interfering with both monitoring and ventilation. Furthermore, alveolar flooding often greatly impedes gas exchange, resulting in severe hypoxaemia, compelling the anaesthesiologist to perform an ET suction.

The AARC Clinical Practice Guidelines on ET suction in mechanically ventilated patients recommend the following: i) ET suctioning should not be performed routinely but only when secretions are present, ii) pre-oxygenation should be considered in patients who experience clinically important reductions in oxygen saturation with suctioning, iii) shallow rather than deep suction should be used and iv) instillation of normal saline prior to ET suction should not be performed routinely. They also emphasise that, for patients requiring a high-inspired oxygen concentration (FiO₂) or a high PEEP and for patients at risk of lung

derecruitment, closed suction catheter systems should be used so that ventilator disconnection and loss of lung recruitment does not occur. Additionally, lung recruitment manoeuvres must be used if suctioning-induced lung derecruitment should occur (4).

The indications for ET suctioning include the need to maintain the patency and integrity of the artificial airway and removal of accumulated pulmonary secretions as evidenced by either visible secretions in the airway, a saw-tooth pattern on the flow-volume loop, the presence of coarse crackles over the trachea, an increased peak inspiratory pressure during volume-controlled mechanical ventilation or decreased tidal volume during pressure-controlled ventilation, deterioration in oxygen saturation and/or arterial blood gas values, patient's inability to generate an effective spontaneous cough, acute respiratory distress and suspected aspiration of gastric or upper-airway secretions and the need to collect a sputum specimen (4).

In patients with severe PE, specific complications include a further decrease in dynamic lung compliance and functional residual capacity, atelectasis, hypoxaemia, tissue trauma and capillary damage, bronchospasm, increased microbial colonisation of the lower airway and cardiovascular instability. Although loss of lung recruitment is the inevitable outcome of circuit disconnection, this may become necessary in order to maintain tube patency and prevent the patient from 'drowning in his secretions'. We believe that the risks associated with ET suctioning in these patients may be reduced by lowering the head end of the table by 10°–15°, performing a gentle chest wall compression and percussion to mobilise secretions, turning the patient's head to the side and then transiently disconnecting the circuit to facilitate passive drainage of oedema fluid without introducing a suction catheter with all the attendant risks of ET suctioning. We found this technique to be associated with a rapid improvement in oxygen saturation while avoiding risks of further damage to the alveolar capillary membrane, and it also prevented blockage of sampling lines and interruptions of CO₂ monitoring and ventilation. This technique can be used in operating rooms where closed suction catheters are usually not available. We believe that intermittent passive gravity-assisted drainage may be preferable to ET suctioning in patients with acute PE because the well-entrenched dictum of not suctioning the trachea of a patient with PE can result in the patient drowning in their secretions, and this technique seems to be a compromise between ET suctioning and no suctioning.

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