# Difficult physiology of airway management: mind the interaction between hypoxia types

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Editor—The editorial by Sakles and colleagues<sup>1</sup> about hypoxaemic cardiac derangement during tracheal intubation of critically ill patients provides a concise overview on acute respiratory distress syndrome pathophysiology and raises awareness of the often neglected physiological challenges encountered during airway management. The suggestion to preserve spontaneous breathing as long as possible with the goal of optimising gas exchange is compelling. The respiratory pump may also protect from hypoxaemia by maintaining cardiac output, particularly in circulating bloodvolume-depleted patients.<sup>2</sup> It is important to recall Barcroft's<sup>3</sup> work suggesting that concomitant occurrence of different hypoxia types synergistically worsens tissue oxygenation, mechanisms that could conceivably contribute to haemodynamic failure during emergency airway management. When contemplating intubation in a hypoxaemic patient, the physician should ensure that other hypoxic contributions are unlikely in the form of reduced cardiac output and anaemia.

The acute and chronic hypoxaemia tolerance of a healthy human is impressive. During testing of pulse oxygen saturation devices, healthy persons withstood acute hypoxaemia of 50% haemoglobin saturation for 10 min.<sup>4</sup> Participants of the legendary Operation Everest studies<sup>5</sup> and a recent study at the German Aerospace Center (DLR)<sup>6</sup> tolerated sustained severe hypoxia lasting weeks. Elite mountaineers perform at extreme altitudes over weeks and some reported almost restful moments on the top of Mount Everest. Finally, aviators and astronauts withstood short periods of anoxia and vacuum.<sup>7</sup> Indeed, pioneering work from the 1940s showed that the altitude-acclimatised human, exposed to increasing hypoxia, defends an alveolar oxygen partial pressure of ~4.7 kPa.<sup>8</sup>

Why then can some patients not persevere when oxygen saturation drops? Possibly, different hypoxia categories occur simultaneously. Barcroft<sup>3</sup> differentiated anoxic hypoxia, stagnant hypoxia, and anaemic hypoxia. He realised that synchronous appearance of more than one of these hypoxia categories would exacerbate tissue hypoxia by limiting compensatory reserve. Furthermore, in frail patients, the hypoxia categories may exacerbate each other, leading to an unstoppable vicious anoxia circle. Barcroft's astute observations highlight the importance of intact physiological interplay between the lungs, heart, brain, blood, and kidneys in maintaining hypoxia tolerance, and suggest that impaired interactions can have grave consequences for patients. For example, healthy coronary flow reserve maintains cardiac oxygenation in the face of hypoxaemia. The same degree of hypoxaemia may elicit myocardial ischaemia in a patient with reduced blood haemoglobin content.

When performing tracheal intubation in severely hypoxaemic patients, particularly in those with co-morbidities, one should keep in mind Barcroft's<sup>3</sup> anoxia concept. Applying an emergency checklist (Table 1) reminds physicians to think about and optimise the physiological hypoxia prevention systems before starting an airway procedure. Emergency checklists have proven useful in aviation emergencies and may be just as helpful in the dynamic situation of the physiologically difficult airway.<sup>1</sup>

Table 1 Physiological pre-intubation emergency checklist to reduce hypoxic harm.

#### Consider and initiate context-sensitive countermeasures for

1. Anoxic hypoxia

2. Stagnant hypoxia

3. Anaemic hypoxia

4. Multiplying effects of hypoxia combinations

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### Ventilator settings and arterial blood gases during video-assisted thoracoscopic surgery including pneumonectomy with pressure support ventilation

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Editor-Lung cancer surgery is associated with a high rate of postoperative pulmonary complications including pneumonia, acute lung injury, and adult respiratory distress syndrome (ARDS).<sup>1,2</sup> It requires lung-protective ventilation strategies,<sup>3,4</sup> which include spontaneous ventilation videoassisted thoracoscopic surgery under general anaesthesia (SV-VATS).<sup>5,6</sup> During SV-VATS, patients in the lateral decubitus position breathe spontaneously with iatrogenic incomplete or subtotal lung collapse, and respiratory settings that allow for stable surgical access. Although advocated as protective for the lungs,<sup>5,6</sup> data on mechanical ventilator settings and resulting arterial blood gases during SV-VATS have not been reported. Such data constitute a prerequisite for evaluating the respiratory consequences of nonintubated spontaneous breathing patients during lung cancer surgery. Such data would also be necessary for the design of a prospective randomised trial comparing SV-VATS with standard management of thoracic surgical patients. Here we provide such data from anatomical lung resections including pneumonectomy in lung cancer patients.

During a 19 month period, 20 patients scheduled for videoassisted thoracoscopic surgery (VATS) for anatomical resection of lung cancer including left lower lobectomy (n=7), middle lobectomy (n=5), right lower lobectomy (n=5), lingula resection (n=1), lower bilobectomy (n=1), and pneumonectomy (n=1) were offered non-intubated pressure support ventilation (SV-VATS). All patients gave informed written consent in agreement with the ethical committee of the Bayerische Landesärztekammer (2020-1041) and Bavarian hospital law (BayKrG §27). Surgical and anaesthetic management followed expert consensus<sup>6</sup> and included EEG monitored target-controlled infusion of propofol and remifentanil, laryngeal mask airway, intrathoracic vagal blockade, and pressure support ventilation (Zeus and Perseus ventilator; Dräger, Lübeck, Germany). Settings of pressure support ventilation were chosen to guarantee lung protective tidal volumes with a maximum of 6-8 ml kg<sup>-1</sup> (predicted body weight), adequate oxygenation, and sufficient space for surgical access. Patients had 16 and 18 gauge peripheral venous access and an arterial blood pressure catheter. Treatment was