**Appendix 1 (as submitted by the author):** Physiological mechanisms linking a raised $FiO_2$ to hypercapnia[1]

(a) Reversal of regional hypoxic pulmonary vasoconstriction in the face of a high $FiO_2$ - diversion of blood from high to low ventilation units creates more wasted ventilation (dead space), and thus less alveolar ventilation, leading to an increase in alveolar ($PACO_2$) and ultimately in arterial $PaCO_2$;

(b) Reduced respiratory drive (for some patients) and a reduction in minute ventilation in response to a high $FiO_2$;

(c) displacement of $CO_2$ from red cells exposed to higher arterial oxygen, (the Haldane effect).

1. Petersson J, Glenny RW. Gas exchange and ventilation−perfusion relationships in the lung. *Eur Respir J* 2014;44:1023-41. PMID: 25063240.