In contrast, SpO_2% indicated significant respiratory depression in only 4/10 patients, with small absolute changes in SpO_2% from 96.5 (95.1 to 99.2)% at baseline to 96.2 (95.2 to 97.0%) at 30 min. A non-significant decline in NRD from baseline (109.5 (69.5 to 185.1) a.u.) to 30 min post IOT 84.3 (59.2 to 118.1) a.u., p = 0.12) was also observed. Baseline NRD and opioid-induced drop in SpO_2% were inversely related (r = -0.67, p = 0.04).

Conclusion Significant acute respiratory depression is commonly induced by opioid drugs prescribed to treat opioid addiction. Hypoventilation is reliably detected by capnography, but not by SpO_2% alone. Chronic suppression of NRD in the presence of underlying lung disease may be a risk factor for acute opioid-induced respiratory depression.

S51 ARTERIAL OXYGEN CONTENT REFLECTS HAEMOGLOBIN MORE THAN OXYGENATION INDICES IN 440 PATIENTS WITH PULMONARY ARTERIOVENOUS MALFORMATIONS

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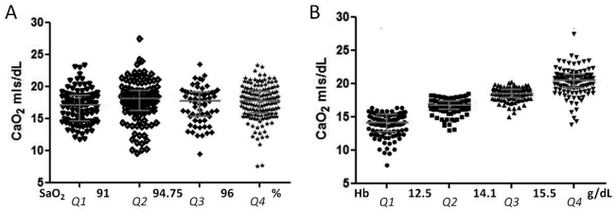
Introduction and objectives Our goal was to use a long term model of human hypoxaemia to evaluate factors that reduce arterial oxygen content (CaO_2) and therefore demand higher cardiac outputs to maintain tissue oxygen delivery. This is important for clinical practice; for clinical trials that use cardiac index as a primary outcome measure; and particularly relevant for patients with pulmonary and systemic arteriovenous malformations (AVMs) due to hereditary haemorrhagic telangiectasia (HHT).

Methods Presentation data were evaluated on 497 consecutive patients with pulmonary AVMs due to HHT, reviewed between 1999 and 2013. SaO_2 was measured by pulse oximetry in the supine and erect postures, and the mean SaO_2 calculated after 7, 8, 9 and 10 min standing. Same-day haemoglobin was measured in venous blood samples in 440 patients. Presentation CaO_2 was calculated by the equation *oxygen saturation (SaO_2, %) x haemoglobin (gram/dL) x 1.34/100*.

Results There was a four-fold difference in CaO_2 across the 440 patients (range 7.6–27.5, median 17.6) mls of oxygen per decilitre (dL) of arterial blood. SaO_2 ranged from 59–100% (median 94.8%), but CaO_2 did not change appreciably across the SaO_2 quartiles (median CaO_2 17.1; 18.1; 17.7; 17.8 mls/dL; p = 0.34, Figure 1A). In contrast, CaO_2 was primarily determined by haemoglobin which ranged from 5.9–21.8 g/dL (median 14.1 g/dL). The median CaO_2 across quartiles of haemoglobin were 14.1; 16.7, 18.5; and 20.5 mls/dL (p < 0.0001, Figure 1B). For each 1 g/dL rise in haemoglobin, there was a 10% increase in mls of oxygen per unit blood volume.



Abstract S51 Figure 1 Distribution of arterial oxygen content (CaO_2) across the quartiles of A) oxygen saturation (SaO_2), and B) haemoglobin (Hb) in 440 patients with pulmonary AVMs

Conclusions Currently, in long term conditions, more attention is paid to modest differences in SaO_2 than to haemoglobin.[1] It has been shown that patients with PAVMs maintain CaO_2, and deliver the same amount of oxygen per heart beat (oxygen pulse) before and after correction of hypoxaemia by PAVM embolisation.[2,3] For patients where higher cardiac outputs may be detrimental, further attention should be given to minor incremental falls in haemoglobin that substantially reduce arterial oxygen content.

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S52 THE EFFECT OF AGE ON ARTERIAL OXYGEN CONTENT IN PATIENTS WITH PULMONARY ARTERIOVENOUS MALFORMATIONS (PAVMS)

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Introduction and objectives It is recognised that age-associated changes in the chest wall and lung parenchyma lead to decreased efficiency of ventilation and gas exchange, resulting in reduced arterial partial pressure of oxygen (PaO_2) and haemoglobin saturation (SaO_2). The total oxygen content of arterial blood (CaO_2) depends upon SaO_2, as well as haemoglobin concentration. Our goal was to examine serial changes in arterial oxygen content with age in a cohort with hypoxaemia due to pulmonary arteriovenous malformations (PAVMs).

Methods Retrospective longitudinal follow-up data was collected for 100 consecutive PAVM patients presenting to a tertiary care institutional clinic between 1984 and 2001, and reviewed until 2015. Subjects provided up to 30 (median 9) separate annual datasets. SaO_2 was measured by pulse oximetry in the supine and erect postures, and the mean SaO_2 was calculated after 7, 8, 9 and 10 min standing. Haematological and biochemical blood indices evaluated haemoglobin, haematinics, and iron indices. CaO_2 in mls of oxygen per dL (ml/dL) of blood was calculated using the equation: *[SaO_2 (%) x haemoglobin (g/dL) X 1.34]/100*. Data were analysed using STATA IC v13.1.

Results Age and PAVM-treatment associated changes in SaO_2 were mostly accompanied by opposing changes in haemoglobin levels that maintained the CaO_2. Two major patterns were observed. The first was the expected increase in haemoglobin with lower SaO_2, due to secondary erthyrocytosis and polycythaemia. The second, less well recognised, was an increase in SaO_2 when haemoglobin fell, most commonly when subjects developed iron deficiency and anaemia. Nevertheless, excluding participants with iron deficiency, CaO_2 decreased with age (Figure 1, r^2 = -0.0654; p < 0.001, Figure 1).