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Editorial

Oxygen: both friend and foe

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I enjoy reading books about expeditions and climbing. One recent favourite was *Into Thin Air* by the journalist, Jon Krakauer.[1] Krakauer was briefed by *Outside* magazine to join a large expedition to the summit of Everest (8848m) amidst concerns of increasing commercialisation. His was one of 2 large parties (and several smaller) attempting to summit on the same day in May 1996.

Unfortunately, a major blizzard came in suddenly, catching about 37 guides, clients and Sherpas on the South face, close to the summit. Eight people died – the highest single day mortality on the mountain until 2015 when an earthquake caused an avalanche that struck base camp and killed 22.

Krakauer's book is controversial because it examines factors that may have contributed to the disaster and to an extent, attributes blame without giving those accused a right of reply. Some of those who died had exhibited mild symptoms of high altitude pulmonary oedema or cerebral oedema (HAPE and HACE in American argot) before the summit attempt and arguably weren't in the best shape to survive an unexpected storm.

Severe HAPE requires prompt descent and evacuation to a clinic at 4240m at Pheriche, Nepal where treatment with hyperbaric oxygen and acetazolamide can be given. Nifedipine and sildenafil can be used to decrease pulmonary vasoconstriction associated with hypoxia. HACE presents with vomiting, headache, lethargy and confusion – sometimes causing climbers to just "give up" or "forget" to look after basic safety. HACE can be more rapidly fatal than HAPE and also requires prompt descent, acetazolamide or steroids. Sometimes climbers take prophylactic acetazolamide to prevent symptoms but many climbers at the highest altitudes probably suffer sub clinically from both.

On the day of the final ascent to the summit, one of the experienced guides chose to make a "personal best" without supplementary oxygen and Krakauer felt that in this hypoxic, zombified state, he was not able to look after the clients to the best of his abilities. On his return to Camp 4 (7951m), the guide donned oxygen, ascended and single-handedly went on to rescue 3 stranded climbers – needless to say, he was not best pleased with Krakauer's book.

The physiology on Everest is extreme. At the summit, total barometric pressure is 33.7 kPa. Ambient PO_2 is 7 kPa - the alveolar pressure of oxygen is somewhat lower at 5.7 kPa due to water vapour occupying space in the lungs. [2]

At this low partial pressure, oxygen can only slowly diffuse into the pulmonary capillaries – the transit time of blood through the capillaries becomes critically important and any increase in cardiac output with exertion lowers the transit time further. In addition, nearly everyone at this altitude has some degree of subclinical high altitude pulmonary oedema so that when the arterial PO_2 was recorded in 4 climbers after 20 minutes of breathing ambient air at 8400m, arterial PO_2 was only 3.3kPa at rest! [3] Surprisingly, there is little evidence for anaerobic respiration as lactate levels were not increased in these subjects. [3]

Hyperventilation, stimulated by low PO_2 at high altitude induces metabolic alkalosis which shifts the oxygen dissociation curve to the left increasing oxygen capture but it also lowers arterial PCO_2 inducing cerebral vasoconstriction, decreasing cerebral blood flow and impairing concentration further. Hyperventilation also carries warm moist air away from the body which can hasten the onset of hypothermia and dehydration. [2]

Elite climber, Ed Viesturs, who has scaled all 14 of the World's 8000m + summits without oxygen describes the experience thus "I'd be literally having to force myself after that 15th breath to take the next step." [4]

The oxygen delivery systems used by climbers deliver 4L O_2 per minute and are often used in Camp 4 on the night before an ascent – this lessens the risk of another hazard of climbing – high altitude sleep apnoea/periodic breathing which is associated with poor sleeping, headache and fatigue.

The survival statistics for ascent without oxygen are stark - 1 in 12 climbers descending from the summit without oxygen die versus 1 in 34 of those using supplementary oxygen [3]

With oxygen, its estimated that more reasonable arterial PO2 levels of around 6.7 kPa are achieved which lessens hyperventilation and in turn protects against cerebral vasoconstriction, dehydration and heat loss.[3]

Krakauer observed that a large proportion of the clients on Everest were doctors and dentists – he felt that both professions contain many chronic over-achievers. I suspect we can all think of colleagues who, when advised to go and relax by taking a gentle walk up the Cavehill, will be attempting to summit Everest 2 years later!

Its Monday morning and I am reading through some GP referral letters to cardiology clinic:

"Please see this 90-year-old patient with dyspnoea. N-terminal BNP is mildly elevated and during a CT abdomen, the Radiologist commented that the inferior vena cava and hepatic veins appeared enlarged possibly reflecting heart failure".

The right-sided venous congestion is almost certainly due to raised right heart pressure secondary to pulmonary hypertension in this age group. What has caused the pulmonary hypertension? A rough calculation estimates that this patient has passed about 50 million litres of one of the most toxic oxidising agents known over their alveoli – not surprising there has been some long-term damage. Diuretics and maybe even pulmonary vasodilators might help – just like the climbers!

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