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The effect of high altitude commercial air travel on oxygen saturation

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Summary

Air travel has increased steadily over the last decade, and its effect on the health of passengers has been the subject of much debate. There is a paucity of evidence on the effects of air travel on oxygen saturation in general populations. The peripheral oxygen saturation and pulse rate of 84 passengers, aged 1–78 years, were measured by pulse oximetry at round level and altitude during air travel. There was a statistically significant reduction in oxygen saturation in all passengers travelling long haul and short haul flights ($p < 0.05$). The mean [range] (SD) SpO_2 for all flights at ground level was 97% [93–100] (1.33) and at cruising altitude 93% [85–98] (2.33). Fifty-four per cent of passengers had SpO_2 values of 94% or less at cruising altitude. This is a value which may prompt physicians to administer supplemental oxygen in hospital patients.

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Air travel has grown steadily over the last decade, with up to 1.5 billion passengers flying in commercial aircraft annually. These aircraft are flying at higher altitudes, up to 39 000 ft or above. The in-cabin pressure corresponds to an altitude of 5000–6000 ft, and to a fraction of inspired oxygen of 0.17–0.15 at ground level. Many flights are also of a longer duration than previously, up to 16 h non-stop [1, 2]. The reduction in the fraction of inspired oxygen at altitude leads to a reduction in oxygen saturation (S_pO_2) in both healthy passengers and in those with coexisting medical conditions during actual and simulated aeromedical flights [3–7].

The aim of this study was to determine, in a general population, the degree of hypoxaemia during short haul and long haul air travel.

Methods

Following Regional Ethics Committee approval, and with written informed consent, we measured the oxygen saturation and pulse rate, by pulse oximetry, of 84 passengers aged 1–78 years flying short and long haul in commercial aircraft. Passengers consisted of anaesthetists and their travelling companions and each passenger acted as their own control. No individual had severe cardio-respiratory problems, and no one required permission from their doctor to fly. We have no record of their smoking status. All flights lasted 1 h or more. The pulse oximeters used were three models of the commercially available Nellcor™ Oximeter (Pleasanton, CA). Each acted as its own control and these are reported to be accurate at maximum cabin altitude. Data were obtained from flights with more than 10 different airlines. Percentage oxygen saturation and pulse rate were recorded in all passengers at ground level before departure of the aircraft. On short haul flights these measurements were repeated at maximum altitude (that is, maximum cabin depressurisation) as defined by the cabin crew. On long haul flights the second set of measurements were

taken 2 h after maximum altitude had been reached. Other parameters recorded included the age of the passenger and the duration of the flight. No consent was sought from the airlines involved.

Statistical analysis

Paired and unpaired Student's *t*-tests, and one-way ANOVA were used to compare data, as appropriate, with the aid of the graphpad PRISM computer software package. A p-value of < 0.05 was deemed to be statistically significant.

Results

Eighty-four passengers were recruited to the study; 55 passengers took long haul flights (duration greater than 2 h) and 29 passengers took short haul flights (duration less than 2 h) (Figs 1 and 2).

Maximum altitude ranged from 37 000 to 41 000 ft above mean sea level with an estimated fraction of inspired oxygen concentration of 0.16–0.15.

As both long and short haul flights showed similar results, the data have been presented together. There was a significant reduction at cruising altitude of S_pO_2 in all passengers, during all flights, when compared to ground level ($p = 0.001$) (Fig. 2). The mean [range] (SD) S_pO_2 at ground level was 97% [93–100] (1.33) and at cruising altitude 93% [85–98] (2.33). There was no significant change in pulse rate, the mean [range] (SD) at ground level being 82 bpm [55–128] (16.7) and at cruising altitude 80 bpm [62–118] (13.1).

Using one-way analysis of variance the actual change in S_pO_2 as a function of each airline was greater for passengers on some airlines than for those on other airlines. The final value was seen to be a function of the starting value and was significant with respect to age; older people started low and ended low.

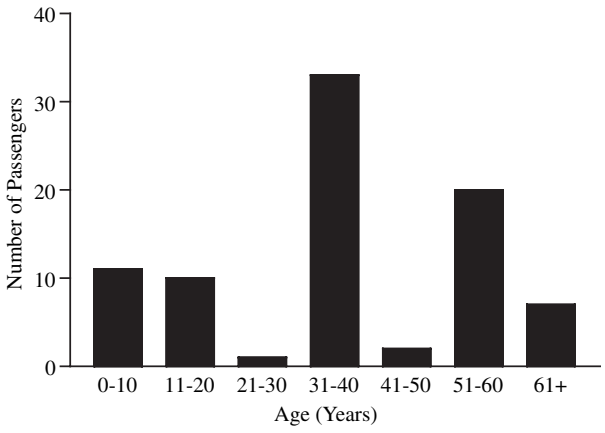

Figure 1 Bar chart illustrating the number of passengers in each age group for all flights ($n = 84$).

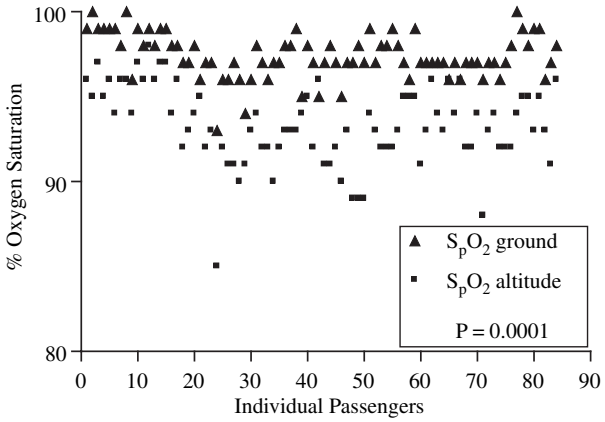

Figure 2 Scattergram illustrating percentage oxygen saturation at altitude and at ground level during all flights ($n = 84$).

Discussion

The House of Lords and Department of Transport have both acknowledged that more studies need to be carried out with respect to the effects of air travel on health, as there is little information on the physiological effects of flying on passengers currently available [7, 8].

There has been much media and public interest recently in the safety of air travel. This has largely focused on the apparent increase in deep venous thrombosis (DVT) and pulmonary emboli occurring in individuals who have recently flown in commercial aircraft [9]. To date, most of the research surrounding the safety of air travel in respect to deep venous thrombosis has concentrated on the general immobility and dehydration of passengers throughout the flight [10].

Three predisposing factors for DVT are reduced flow, increased coagulability and damaged vessel walls. Limb oedema is believed to be greater in aircraft than for equivalent periods of travel in cars or trains, and to be worse at lower cabin pressures. This may be due to local hypoxia, which causes vasodilation and increased capillary permeability. The ventilation system of aircraft is determined by the required air quality rather than the oxygen supply, for example to dilute human body odour and CO_2 to an acceptable level [11]. There is a paucity of data, however, on the quality of this air and on the effect of this quality on the S_pO_2 of the individual passengers. Pressurised aircraft do not maintain a sea level pressure during flight for practical reasons. During commercial flights most cabins are pressurised to an equivalent altitude of 5000–8000 ft, which equates to an inspired oxygen fraction of 0.17–0.15 at sea level [5]. Current US Federal Aviation Regulations specify that pressurised cabins must provide a cabin pressure altitude of not more than 8000 ft (2440 m) at the maximum operating altitude of the

airplane (FAA, 1996), in order to maintain the partial pressure of oxygen [12].

Actual cabin altitude pressure measurements by 28 airlines range from sea level to 8,915 ft (2717 m) with a mean altitude of 6214 ft (1894 m) and the flying altitude of the planes ranging from 10 000 ft (3053 m) to 60 000 ft (18 290 m). The newer aircraft fly higher than older aircraft, with greater altitude exposure to passengers and an increased risk of hypoxia [1]. In our study the maximum altitude of the aircraft was 27 000–37 000 ft, potentially reducing the partial pressure of oxygen, although we did not formally ascertain this value in our study.

Alveolar oxygen tension is known to decrease to 65 mmHg at 8000 ft, with a resultant reduction in arterial oxygen tension to 60 mmHg in healthy individuals [1]. Hypoxaemia augments the sympathetic nervous system and has many physiological effects. There is an increase in ventilatory effort in response to hypoxic conditions and the cardiovascular system responds by elevation of the heart rate, with subsequent reduction of stroke volume and an increased risk of angina and dysrhythmias in coronary patients [2, 13]. Acute hypobaric hypoxia is also known to induce a hypercoagulable state [14]. The cerebral effects are less clearly defined but include reduced night vision and a reduction in cognitive function [1, 2].

When considering S_pO_2 and its effects on partial pressure of oxygen, it is essential to understand the unique characteristics of the oxyhaemoglobin dissociation curve. Our study indicates that passengers flying long and short haul have a significant reduction in their S_pO_2. One passenger had an S_pO_2 of 94% at ground level and this decreased to 85% at altitude, Patients with chronic hypoxia may have a greater reduction in S_pO_2 when they reach high altitude than those with healthy lungs. Over one-third of respiratory physicians would prescribe supplementary oxygen for patients with an S_pO_2 of less than 94% [15]. Over half the passengers studied fell within this category. We analysed the long and short haul data separately but found no significant difference between the two, and so have combined the data. The passengers in our study had S_pO_2 values at cruising altitude (maximum depressurisation) that correspond to a pO_2 value on the shoulder of this curve so that a further small reduction in pO_2 (kPa) would result in a large fall in S_pO_2. This could have detrimental effects on all body systems.

In conclusion, this preliminary study is the first study to quantify the reduction of percentage oxygen saturation at high altitude during commercial air travel. It demonstrates that there is a reduction in S_pO_2 during both long and short haul flights in all age groups. While this study is purely observational in nature, we postulate that this hypoxia (along with other factors such as dehydration, immobility and low humidity) may be a factor in morbidity during and after air travel.

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