REVIEW SERIES

Chronic obstructive pulmonary disease • 11: Fitness to fly with COPD

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For most passengers, even those with respiratory disease, air travel is safe and comfortable. Some patients with COPD may be at risk but, with screening, these patients can be identified and most can travel safely with supplemental oxygen.

A House of Lords Select Committee on Science and Technology published a report entitled "Air Travel and Health" in November 2000.[1] They concluded that air travel was safe for the vast majority of passengers but that a few were at risk, and the Government, regulators and the airline industry were open to criticism for failing to give "sufficient active attention to health". One of the key recommendations of the report was that more information should be available to intending air travellers so that they could make informed choices about the risks (even though these may be minor) of air travel. Another recommendation was that intending air travellers should be urged to consult their doctor if they had any doubt regarding their fitness to fly.

A recent survey of chest physicians providing advice to patients with respiratory disease who wished to travel by air concluded that "the risk of hypoxia at altitude is recognised by most respiratory physicians in England and Wales, but assessment methods and criteria for recommending oxygen vary widely".[2] The article suggested that evidence based guidelines were required.

The British Thoracic Society has recently produced guidelines on fitness to fly in patients with respiratory disease which concentrates on intending air travellers with chronic obstructive pulmonary disease (COPD).[3] It provides a concise summary of the field with an extensive literature list.

ALTITUDE AND HYPOXIA

Commercial aircraft are pressurised during flight because passengers could not survive exposure to the low atmospheric pressure at the usual cruising altitude (10 000–13 500 m). For reasons of aircraft weight and fuel economy, it is impractical to maintain cabin pressure at sea level pressure but international regulations do not allow cabin pressure to fall below 74 kPa (the equivalent of atmospheric pressure at 2450 m (8000 ft)) except in emergencies.[4] In practice, cabin altitude is normally maintained at about 1850 m but, according to one study, flight profiles in newer aircraft may produce cabin altitudes that are near or possibly exceed the stipulated maximum.[5] The concentration of oxygen in air is constant at 20.9% to very high altitudes (up to 90 km), so the lowest inspired oxygen pressure to which passengers should be exposed under normal flight circumstances is about 15 kPa (20.9% of the cabin pressure of 74 kPa).

The other main effect of altitude is that of Boyle's law which states that the volume of gas varies inversely with the surrounding pressure at constant temperature. One litre of gas at sea level will expand to about 1.4 litres at an altitude of 2450 m.[6] This is clearly only of medical relevance if gas is trapped in a confined space such as the pleural cavity, the middle ear, or in a body cavity after surgery.

EFFECTS ON PASSENGERS

In healthy passengers the hypobaric hypoxia of the aircraft cabin will result in only mild hypoxaemia; arterial oxygen tension (Pa_{O_2}) falls to about 8 kPa but the shape of the oxyhaemoglobin dissociation curve prevents any fall in oxygen saturation (Sa_{O_2}) below about 92%.[6]

However, some patients with chronic lung disease may not tolerate this mild degree of hypoxia and may become significantly hypoxaemic. Schwartz *et al*[7] studied 13 subjects with COPD exposed to an altitude of 2250 m in whom arterial blood gas tensions were measured at sea level and at altitude. Mean Pa_{O_2} fell from 9.0 kPa at sea level to 5.9 kPa, a level which most physicians would regard as undesirable although none of these subjects developed any symptoms. Pa_{CO_2} fell from 5.4 kPa to 4.8 kPa.

The effect of acute hypoxaemia on patients with stable COPD is not well studied and, subjectively, many patients appear to tolerate hypoxaemia well.[6 8 9] However, some patients may develop respiratory symptoms and arrhythmias even at rest.[10] The effect of hypoxaemia combined with exercise has not been studied and, in addition, many patients with COPD will have co-existing cardiac disease which may limit normal cardiovascular responses to hypoxaemia. While the effects of acute hypoxaemia during air travel have not been fully explored, most authors consider it reasonable to choose an arbitrary level of hypoxaemia (usually Pa_{O_2} of 6.6 kPa) below which it is considered unsafe to fly without supplemental oxygen therapy.[8] This approach seems to work in practice to ensure safe and comfortable air travel.[11]

By far the most common effect of altitude induced gas expansion is ear pain associated with poor ventilation of the middle ear. However, gas expansion is clearly of relevance to passengers with a pneumothorax which is regarded as one of the few absolute contraindications to air travel.

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Gas expansion within the pleural cavity can become life threatening, as illustrated in 1995 by the well publicised case of a woman with a pneumothorax which was treated over the Himalayas by doctors using a scalpel, knife and fork, a coat hanger and a urinary catheter.[12] Airlines recommend that a pneumothorax should have settled completely before air travel and that flying should be avoided for at least 6 weeks. However, there appears to be no evidence to support the 6 week recommendation, which takes no account of the type of treatment which may have been given.

The pressure changes during air travel will not precipitate a pneumothorax, but reduced access to health care on board an aircraft makes a recurrence of a pneumothorax undesirable. There is little evidence available on the time frame for recurrence of a pneumothorax but, in the absence of a pleurectomy or talc pleurodesis at thoracotomy,[13–15] the risk of recurrence remains high (possibly up to 50–70%), with the highest risk occurring in the first year and in patients with underlying COPD.[16–20] Potential passengers will need careful medical assessment and may wish to take recurrence rates into account when deciding on air travel plans.

EPIDEMIOLOGY OF IN-FLIGHT EMERGENCIES

Death during air travel is unusual. A recent study of in-flight medical emergencies examined the in-flight treatment and follow up diagnosis in 1132 incidents dealt with by MediAire—a company which provides ground based medical assistance to a number of US carriers.[21] The reported fatality rate of 0.107 per million passenger embarkations was less than other similar surveys. Qantas reported a fatality rate of 0.38,[22] and 42 IATA member airlines reported a rate of 0.31.[23] The majority of in-flight deaths appear to be due to cardiac causes, but there may be associated respiratory symptoms.[21]

Respiratory problems account for 2–10% of all in-flight medical emergencies and usually make up the second or third most common group of serious medical emergencies (with cardiac and neurological incidents).[21–28] However, in the CAMI/MediAire study oxygen was administered in 58% of the medical incidents and resulted in an improvement in the condition of the passenger in 81% of cases.

Thus, although respiratory incidents appear to account for a small proportion of all in-flight medical emergencies, respiratory problems make up a significant number of the more serious emergencies and unrecognised hypoxaemia may well account for a number of the other incidents such as "vasovagal" episodes or non-specific events which then improve with supplemental oxygen therapy.

The frequency and outcome of air travel in patients with COPD has not been well studied. One North American study reported on a cohort of 100 patients with COPD,[29] 44 of whom chose to travel by air over the 28 month period of the study. Those who did not travel had a lower mean forced expiratory volume in 1 second (FEV_1), which suggests that many patients with more severe COPD choose not to travel by air. Eight of the 44 developed transient symptoms during the flight. A similar British study reported similar findings: of 97 subjects with COPD, 34 had travelled by air within 24 months and nine developed transient respiratory symptoms during the flight but all arrived safely.[30]

PRE-FLIGHT ASSESSMENT

The aim of pre-flight assessment is to identify passengers likely to develop respiratory symptoms during air travel. Given the current state of knowledge, most guidelines suggest trying to identify those likely to develop significant hypoxaemia. Three methods of pre-flight assessment have been suggested.

The first method has been put forward by the airline industry and consists of asking passengers whether they are capable of walking 50 metres on the flat. If the answer is "yes", they are probably fit to fly.

The second method consists of applying one of a number of prediction equations which have been developed from studies in which subjects with COPD were exposed to hypobaric or normobaric hypoxia and arterial blood gas tensions were measured. Some equations only require measurement of Pao_2 at sea level and then allow calculation of a predicted Pao_2 at altitude. Examples include the formula produced by Henry *et al*[31]:

$$Pao_2alt = 20.38 - (3*cabinalt) + (0.67*Pao_2grnd)$$

and that produced by Gong *et al*[8]:

$$Pao_2alt = 22.8 - (2.74*cabinalt) + (0.68*\ Pao_2grnd)$$

where Pao_2alt is the predicted Pao_2 at altitude in mm Hg, cabinalt is the cabin altitude to be used in thousands of feet, and Pao_2grnd is the Pao_2 at sea level in mm Hg.

Other authors have used spirometric values in addition to sea level Pao_2 in an attempt to reduce variation. Examples here include the formula from Dillard *et al*[32]:

$$Pao_2alt = 0.453\ (Pao_2\ grnd) + 0.386\ (FEV_1\ \%pred) + 2.440$$

and another more complicated formula from Dillard *et al*[33]:

$$Ln(Pao_2alt/Pao_2grnd) = k*(Pio_2alt - Pio_2grnd)$$

Another approach is to use a purely mathematical model which allows calculation of a predicted altitude Pao_2 from sea level Pao_2 and $Paco_2$ by assuming that the arterial-to-alveolar oxygen ratio remains constant at altitude.[34]

The third method of pre-flight assessment involves measuring the subject's response to a simulated aircraft cabin environment. In this investigation—termed the hypoxic challenge test—the subject is usually exposed to an inspired oxygen tension of 15 kPa which is the expected worst case scenario for the aircraft passenger. This can be achieved at sea level by administering 15% oxygen in nitrogen using a face mask and non-rebreathing circuit,[8] or by filling a body plethysmograph with 15% oxygen.[35] A modification of this latter technique is to use a hood over the subject's head and shoulders which is ventilated with a constant supply of 15% oxygen. Alternatively, 15% oxygen can be administered simply by using a 40% Venturi mask with nitrogen as the driving gas. The Venturi dilutes the nitrogen with air so that 15% oxygen is produced.[36 37] During the hypoxic challenge the subject is monitored continuously with pulse oximetry to prevent severe hypoxaemia and blood gas tensions are measured before and at the end of the hypoxic exposure. Some authors recommend ECG monitoring to detect asymptomatic arrhythmias.[8] Usually a predicted Pao_2 of 6.6 kPa (which was derived from 50 mm Hg in the original study) is used as the cut off below which supplemental oxygen is recommended for air travel.[8]

The 50 metre walk test has the advantage of being an individualised test of cardiorespiratory function but, despite its widespread use by airline medical departments, does not appear to have any evidence base. In addition, potential passengers are usually simply asked if they can walk 50 metres and are not formally tested. A recent audit in our department suggests that neither patients nor healthcare staff are good at assessing distance.

Mean predicted altitude Pao_2 derived from predicted equations appears to compare well with mean predicted Pao_2 from both hypobaric and normobaric hypoxic challenge in groups of subjects with COPD.[38] However, it is not clear whether prediction equations provide information which is sufficiently robust to allow the decision of whether or not to recommend in-flight oxygen therapy. Prediction equations appear to be infrequently used by practising physicians in UK.[2]

Hypoxic challenge has the advantage of assessing an individual's response to hypoxia and affords the opportunity of monitoring symptoms and other responses to hypoxaemia such as hyperventilation, cardiac arrhythmias, or angina.[8 38] Cardiac disease often co-exists with COPD and yet is usually an exclusion criterion for studies of pre-flight assessment.

The results of pre-flight prediction of hypoxaemia have not been compared with actual hypoxaemia during air travel, nor with the development of respiratory or cardiac symptoms. The cut off level of Pa_{O_2} of 6.6 kPa appears to be empirical with no evidence base, but has been generally accepted as a reasonable compromise.

The above data suggest that the hypoxic challenge test is the gold standard for pre-flight assessment. However, the test is not widely available and increasing numbers of patients with COPD are considering air travel. Clearly, widespread use of the hypoxic challenge test is inappropriate. The BTS guidelines[3] suggest that saturation measured by pulse oximetry can be used to identify groups of potential passengers who will definitely require oxygen (sea level Sp_{O_2} <92% or already on long term oxygen therapy) or who are very unlikely to require oxygen (either sea level Sp_{O_2} >95% or sea level Sp_{O_2} >92% without any additional risk factors such as severe COPD, hypercapnia, recent exacerbation or relevant co-existing disease). These groups would not need testing but the intermediate group (Sp_{O_2} <95% and >92% with risk factors) would need to have a hypoxic challenge test.

PRESCRIBING IN-FLIGHT OXYGEN THERAPY

Berg *et al*[39] investigated the effects of oxygen supplementation in 18 patients with non-hypercapnic COPD in a hypobaric chamber. Oxygen was supplied by nasal cannula at 4 l/min, 24% Venturi mask, and 28% Venturi mask. The conclusion was that all forms of oxygen therapy improved Pa_{O_2}, but the Venturi masks did not fully correct Pa_{O_2} to the sea level baseline (Pa_{O_2} 0.7 kPa below baseline with the 28% mask and 1.6 kPa below baseline with the 24% mask) whereas the nasal cannula slightly overcorrected (Pa_{O_2} 1.3 kPa above baseline). All the oxygen devices raised Pa_{O_2} over 6.6 kPa and were therefore all felt to be appropriate for in-flight use; the choice of device may depend on the subject's propensity to carbon dioxide retention when given oxygen. If oxygenation is felt to be critical, an accurate assessment of the requirement can be made by titrating oxygen by mask or cannulae in a body box filled with 15% oxygen[35] or, ideally, in a hypobaric chamber if available.[39] In practice, most aircraft oxygen systems are only capable of providing 2 or 4 l/min; a flow rate of 2 l/min is suitable for most passengers, but those already on long term oxygen therapy may be advised to increase it by 2 l/min above their normal flow rate.

Most airlines are able to provide supplemental oxygen therapy although the charge for this varies widely.[40] Some UK based tour operators will not provide oxygen so it is important that potential passengers check with the airline before booking (personal communication). Other airlines provide restriction on the duration of oxygen that can be provided and all will want to know the flow rate required and whether the oxygen is for intermittent or continuous use. Oxygen is usually provided from cylinders so the duration of oxygen therapy is needed to calculate the number of cylinders required. Some airlines use the aircraft's mains oxygen system.[41] Many will provide nasal cannulae but some provide masks.[40]

The altitude of stopover airports and the final destination needs to be considered. Passengers who are identified as being hypoxaemic at the moderate altitude of the aircraft cabin may also be hypoxaemic at high altitude destinations such as Mexico City (2239 m). Most airlines will not provide oxygen for stopovers and direct flights are preferred.

Most airlines will indicate that 48 hours notice is sufficient to organise supplemental oxygen but this is seldom the case in practice. Passengers may not carry their own oxygen supply on the aircraft unless they wish to take an empty cylinder to be refilled for use at their destination.[40]

Box 1 Key points

- Increased awareness of the need for and methods of pre-flight assessment is required as the number of air travellers with chronic lung disease increases.
- Patients should seek advice regarding the advisability of air travel before booking their journey as not all airlines will provide oxygen and others have restrictions.
- Doctors need to be aware of the aircraft cabin environment and need to give consistent advice.
- If screening indicates the need for in-flight supplemental oxygen, the passenger should consider the cost of oxygen when comparing air ticket prices. It is best to give the airline plenty of notice and to check the day before travel that the necessary arrangements have been made.
- Communication with the airline is often through a MEDIF (medical information) form which will require details of the patient's diagnosis, the reason for supplemental oxygen, and the duration of oxygen therapy (intermittent or continuous) with a further section for information on the patient's mobility and need for special boarding or seating arrangements. Most airlines have a medical department which may be very helpful although getting through can be time consuming.[40]
- Passengers should carry any necessary medication in their hand luggage and may need to carry a prescription with them. A letter confirming the medical necessity of any electrical equipment such as nebuliser compressors, CPAP machines, or oxygen concentrators is essential with current tight airline security. Insurance can pose a problem but patients may receive helpful advice regarding insurers from medical charities.
- Passengers with lung disease should be advised to avoid smoking and alcohol before and during air travel. Like all passengers they should carry out regular leg exercises, but those passengers requiring oxygen should request a seat near the toilets to avoid long walks.

CONCLUSION

For most passengers, even those with respiratory disease, air travel is safe and comfortable. Some patients with COPD may be at risk but, with screening, these patients can be identified and most can travel safely with supplemental oxygen. There are large gaps in the evidence base for advising potential air travellers. More research is needed, especially on the effects of real air travel on passengers with lung disease rather than the effect of simulated hypoxia which can only reproduce part of the stress of air travel. A summary and general advice for potential air travellers with COPD is given in box 1.

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LUNG ALERT

Obstructive sleep apnoea with hypoxaemia leads to hepatic dysfunction

▲ Chin K, Nakamura T, Takahashi K, *et al*. Effects of obstructive sleep apnoea syndrome on serum aminotransferase levels in obese patients. *Am J Med* 2003;**114**:370–6

Abnormal aminotransferase levels in obese patients prompted this group to investigate changes in serum aminotransferase levels in 40 obese patients with obstructive sleep apnoea who, on polysomnography and clinical symptoms, were candidates for treatment with nasal CPAP (continuous positive airway pressure). Patients who had an excessive intake of alcohol, hepatitis B antigen, hepatitis C antibody, or autoimmune disease were excluded. Serum aminotransferase levels were measured in the afternoon and morning, before and after nasal CPAP. Before treatment with CPAP aminotransferase levels increased after sleep ($p=0.006$). After the first night of nasal CPAP overnight mean increases in aminotransferase levels were less marked ($p=0.0003$). 35% of patients had abnormal aminotransferase levels (>1.5 times upper limit of normal) initially. In these patients changes in aminotransferase levels were significantly greater ($p<0.0001$). Improvements in aminotransferase levels were maintained after 1 month ($p=0.0006$) and 6 months of CPAP.

Recurrent apnoea and hypoxaemia may aggravate hepatic dysfunction in these patients, as indicated by release of aspartate aminotransferase. CPAP may therefore prove to be a useful method of preventing some complications of obstructive sleep apnoea such as hepatic dysfunction and cirrhosis.

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