Fitness to fly for passengers with cardiovascular disease

The report of a working group of the British Cardiovascular Society

Guidance at a glance

Condition	Functional status	Lay explanation	Restriction/guidance
Angina	CCS angina I–II	Chest pain on considerable exertion with no recent change in symptoms or medication	No restriction
	CCS angina III	Chest pain on minimal exertion with no recent change of symptoms or medication	Consider airport assistance and possible in-flight oxygen
	CCS angina IV	Chest pain at rest or a change in symptoms and/or medication	Defer travel until stable or travel with medical escort and in-flight oxygen available
Post-STEMI and NSTEMI	Low risk: age <65, first event, successful reperfusion, EF >45%, no complications, no planned investigations or interventions	If you have had a heart attack but you are aged <65 years, the blocked artery has been opened, the heart pump is not badly damaged and no further tests or treatment are planned	Fly after 3 days
	Medium risk: EF >40%, no symptoms of heart failure, no evidence of inducible ischaemia or arrhythmia, no planned investigations or interventions	If you have had a heart attack, your heart pump is quite good and you have no symptoms of breathlessness or chest pain and no other tests or treatments are planned	Fly after 10 days
	High risk: EF <40%, signs and symptoms of heart failure, those pending further investigation, revascularisation or device therapy	If you have had a heart attack, the heart pump is significantly damaged and you have symptoms of breathlessness or you are waiting for further tests or treatment	Defer travel until condition stable
Elective PCI uncomplicated		You have had the heart arteries treated with a balloon and stent and there are no complications	Fly after 2 days
Elective CABG uncomplicated	Allow for intrathoracic gas resorption. If complicated or symptomatic, see heart failure	You have had heart bypass surgery and time must be allowed for any air in the chest to be absorbed	Fly after 10 days if no complications. If symptomatic, follow guidance for specific symptoms
Acute heart failure		You have been in hospital or treated at home because of 'water in the lungs' which made you very breathless	Fly after 6 weeks if stabilised (see chronic heart failure)
Chronic heart failure	NYHA I and II	You get breathless on mild to moderate exercise but no recent (within 6 weeks) change of symptoms or medication	No restriction
	NYHA III	You get breathless on walking 20–100 yards/metres at your own pace but no recent (within 6 weeks) change of symptoms or medication	May require in-flight oxygen
	NYHA IV	You are breathless at rest and mainly bedbound	Advised not to fly without in-flight oxygen and medical assistance
Cyanotic congenital heart disease	NYHA I and II	You have congenital heart disease with blue blood and get breathless on mild to moderate exertion but no recent (within 6 weeks) change of symptoms or medication	May require in-flight oxygen*
	NYHA III	You have congenital heart disease with blue blood and get breathless on walking 20–100 yards/metres but no recent (within 6 weeks) change of symptoms or medication	Consider airport assistance and may require in flight oxygen advisable*
	NYHA IV	You have congenital heart disease with blue blood and are breathless at rest and mainly bedbound but no recent (within 6 weeks) change of symptoms or medication	Advised not to fly without in-flight oxygen and airport assistance available*
Valve disease (see heart failure)			
Following pacemaker inplantation		If you have had a temporary or permanent pacemaker there is a risk of the lung being punctured. If it has not been punctured, you can fly after 2 days. If it has, then you should wait until 2 weeks after it has fully healed	Fly after 2 days if no pneumothorax. In the event of a pneumothorax, flying should be deferred for 2 weeks following complete resolution
Following ICD implantation		If you have had a defibrillator, the same advice for pacemakers (above) applies but, in addition, you should not fly after the ICD has delivered a shock until your condition is considered stable	The same advice as for pacemakers above but, in addition, rhythm instability should be treated
Arrhythmia	Stable	If you get occasional palpitations that do not cause fainting and have not recently become more frequent or you have an irregular pulse which is treated and stable	No restriction

Continued

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Condition	Functional status	Lay explanation	Restriction/guidance
Ablation therapy		If you have had an ablation (burn) procedure to get rid of your palpitations you can fly after 2 days. If flying within 1 week of the procedure, you should consider yourself at high risk of forming blood clots and talk to your doctor	Fly after 2 days*

New York Heart Association (NYHA) grading of heart failure

NYHA	Symptom
I	No limitation of physical activity and no shortness of breath when walking or climbing stairs
II	Mild symptoms of shortness of breath and slight limitation during ordinary activity
III	Marked symptoms of shortness of breath during less than ordinary activity (eg, walking 20–100 yards). Comfortable only at rest
IV	Severe limitation of activity with symptoms at rest

Canadian Cardiovascular Society (CCS) grading of angina

CCS	Symptom
I	Angina only during strenuous or prolonged physical activity
II	Slight limitation, with angina only during vigorous physical activity
III	Symptoms with everyday living activities (ie, moderate limitation)
IV	Inability to perform any activity without angina or angina at rest (ie, severe limitation)

*Consider at high risk of DVT/VTE.
CABG, coronary artery bypass graft; CCS, Canadian Cardiac Society; DVT, deep vein thrombosis; EF, ejection fraction; ICD, implantable cardioverter defibrillator; NSTEMI, non-ST elevation myocardial infarction; NYHA, New York Heart Association; PCI, percutaneous coronary intervention; STEMI, ST elevation myocardial infarction; VTE, venous thromboembolism.

Guidance for the avoidance of deep vein thrombosis and venous thromboembolism

Blood clots (DVT and VTE)	Risk criteria	Risk reduction advice for passengers
Low risk	No history of DVT/VTE No recent surgery (4 weeks) No other known risk factor	Keep mobile. Drink plenty of non-alcoholic drinks. Do not smoke. Avoid caffeine and sedative drugs
Moderate risk	History of DVT/VTE Surgery lasting >30 min 4–8 weeks ago Known clotting tendency Pregnancy Obesity (BMI >30 kg/m^2)	As for 'low risk' with the addition of compression stockings
High risk	Previous DVT with known additional risk including known cancer Surgery lasting >30 min within the last 4 weeks	As for moderate risk but subcutaneous injections of enoxoparin 40 mg before the flight and on the following day

BMI, body mass index; DVT, deep vein thrombosis; VTE, venous thromboembolism.

Fitness to fly for passengers with cardiovascular disease

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SUMMARY

Following this review of evidence and after due consideration, it is clear that there are few cardiovascular conditions that warrant the denial of fitness to fly as a passenger. Given the right aircraft, on-board equipment and appropriately qualified and experienced escort personnel, aircraft can act as flying intensive care units and carry extremely ill passengers.[1]

For those with cardiovascular disease who are not critically ill but who wish to fly on commercial aircraft, the aircraft environment does not pose a significant threat to their health. It is only when their underlying condition is associated with a significant risk of acute deterioration that reasonable restrictions should apply. For those at the more severe end of the spectrum of their specific cardiovascular condition, services exist to help make the journey more easily and safely. Most carriers and airport authorities provide assistance on the ground and in the air. Oxygen is available on most major carriers, although this is sometimes subject to a charge and at least 7 days notice is normally required.[2]

Passengers are advised to plan their arrival at the airport in plenty of time to avoid having to rush and to warn the carrier and/or airport authority of any requirements for assistance, including requirement for in-flight oxygen, well in advance of the date of departure. They are strongly advised to ensure they have an appropriate supply of their medication, a clear list of the medications and doses they take and a letter of explanation from their doctor regarding their condition, drugs, allergies and devices (eg, pacemaker).

Physicians are advised to consider the stability of a passenger's condition and apply the guidance herein.

The authors have contributed to this document in good faith and consider it to be an honest conclusion of the review of evidence and assessment of the risks. It is guidance only and responsibility for declaring a patient fit to travel rests with the attending physician.

The airline carrier should always be informed if a sick passenger is intending to fly. It has a right to refuse the carriage of a passenger at its own discretion, even if they technically fulfil these guidelines recommendations shown overleaf.

1. INTRODUCTION

1.1 This Working Group of the British Cardiovascular Society was established to produce a report on Passenger Fitness to Fly in response to the House of Lords Science and Technology Committee report on Air Travel and Health[3] in which it is suggested that specialist cardiology guidance would be of assistance to general practitioners, passengers and passenger carrying organisations when determining the risks of passenger flight for those with cardiovascular disorders.

1.2 There are many existing guidelines on passenger fitness to fly,[4–7] most of which include some reference to certain cardiovascular disorders, but there is variation in the recommendations, particularly in the time required to elapse between an event or medical procedure and the flight.

1.3 There is a lack of clarity over the purpose of the current guidelines. The suggestion by the House of Lords Committee on Air Travel and Health that there be specialist cardiology guidance is vague about the overall goal of the guidance. There are widespread concerns among the public that air travel has the potential to be harmful, many of which are expressed by the House of Lords committee and are reflected in the following few paragraphs.

1.3.1 Although passenger flight is commonplace, the aircraft cabin provides what might be considered a relatively alien, restrictive and hostile environment. Passengers are strapped for long periods in an upright sitting position and subjected to continuous noise, low humidity, cosmic radiation and hypobaric hypoxia, any or all of which may have a deleterious effect on their health.

1.3.2 Passengers may already have a medical condition and exposure to the flying environment may precipitate an acute deterioration or potentially catastrophic event.

1.3.3 The aircraft is an isolated environment with limited facilities for acute health care and out of reach of assistance other than that which can be gained from radio communication. It is not an ideal place in which to fall ill.

1.3.4 There are now a very large number of passengers, and a high proportion of them are elderly. The number of passenger hours in the air is increasing and with it the chance of spontaneous events occurring in flight but not actually caused or precipitated by the flight.

1.3.5 Events in flight may cause disruption for staff and other passengers and, at worst, may lead to the aircraft being diverted to where appropriate medical care can be provided. Diversions are very costly and disruptive for the carrier and it is desirable that such events are kept to a minimum.
1.4 Guidelines may be directed at any of the above problems, but the British Cardiovascular Society and this Working Group have considered the House of Lords requirements and feel this guidance should be directed neither at limiting cost and disruption to airline carriers nor to avoiding potential dangers of the in-flight environment to healthy passengers, but solely to providing advice about the risks of flying for passengers with recognised cardiovascular disease.

2. TERMS OF REFERENCE

2.1 To review the evidence for safety and risk of air travel for people with cardiovascular conditions.
2.2 To agree appropriate advice and guidance for air travel which can be expected to be safe for patients with cardiovascular conditions.
2.3 To agree appropriate advice and guidance for healthcare requirements for people undertaking air travel with potentially unsafe cardiovascular conditions, or suspected cardiovascular conditions, including 'medical repatriation'.
2.4 To report and to make recommendations to the Board of Trustees of the British Cardiovascular Society.

3. MEMBERSHIP

Dr L D R Smith (Chairman), Dr C Aps, Dr N Boon (ex officio British Cardiovascular Society), Mrs E Clark, Dr N Dowling, Dr S Gibbs, Dr D Hackett (ex officio British Cardiovascular Society), Professor M Joy, Dr M Anderson, Dr W Toff, Dr R Johnston (co-opted) and Professor J Cleland (co-opted).

4. WHAT THIS DOCUMENT DOES NOT COVER

4.1 Cardiovascular disease is a term which may be used to include any pathological condition of the heart and its components, the great vessels and the peripheral vasculature. It could therefore include diabetes mellitus, the idiopathic arteritides, peripheral arteriosclerosis and abdominal aortic aneurysm. It could include stroke, multi-infarct dementia, carotid artery disease and cerebral arteriovenous malformations, phlebitis and varicose veins. The Working Group considered the scope of the report and agreed that it would not be appropriate to include all these conditions. For the purposes of this document, the term 'cardiovascular' will refer to conditions of the heart and the great vessels.
4.2 This guidance does not refer to cardiovascular fitness of individuals to take control of an aircraft, nor does it deal with the risks associated with flight in private aircraft, military aircraft, aircraft with non-pressurised cabins or other means of flight such as paragliders or balloons.

5. WHAT THIS DOCUMENT DOES COVER

5.1 This document is solely about the fitness to fly of air travel passengers who have cardiovascular conditions and covers all aspects of cardiovascular fitness to fly for commercial passengers. It deals with the common questions and problems that patients, their relatives and their doctors ask about the advisability of flying, and it reviews the existing evidence in the formulation of guidelines for passenger fitness to fly.
5.2 The overriding consideration for the Working Group was that the aim of the guidelines should be, wherever possible, to allow people to fly and not to be unnecessarily restrictive.
5.3 This document considers the effects of passenger flight and provides guidance accordingly for the conditions that come under the broad headings of ischaemic heart disease, heart failure, cyanotic congenital heart disease, abnormalities of cardiac rhythm and cardiac devices.
5.4 It is now widely accepted that guidelines published by professional societies, national and joint international committees follow a specific style that includes an indication of the level of importance of a particular guideline and the level of supporting evidence that justifies it. There is such a paucity of randomised controlled trials, meta-analyses and registries regarding the risks of commercial flight for passengers with cardiovascular disease that the Working Group felt unable to adopt this style. What evidence there is has been reviewed and used, but a significant element of the guidance within the document is based on professional judgement and an understanding of the interaction between the flight environment and the pathophysiology of the cardiovascular condition.
5.5 There was much discussion about the inclusion of venous thromboembolism (VTE) since this condition has so clearly been implicated in long-haul air travel. Much has been written on the subject of VTE already, but the group considered that a section on VTE should be included.

6. REVIEW OF EVIDENCE

There are very few direct clinical studies of the pathophysiological effects of commercial passenger flight on patients with existing cardiovascular diseases. Concerns that there may be deleterious effects are based on extrapolation from an understanding of the physics of altitude, cardiorespiratory physiology and studies that attempt to simulate the conditions of flight, either by studying patients adapting to living on land at altitude or by studying the effects of normobaric hypoxia in a laboratory setting.

We have considered the physics and physiology and formed a view on whether there is evidence to suggest that being a passenger in a commercial aircraft has any adverse effects that may increase the risk of a cardiac event.

We have also considered people who may be considered high risk by dint of a recent cardiac event or procedure and in whom restrictions on flying are designed to limit the chance of an in-flight event where facilities for managing medical emergencies are poor.

6.1 Physics of air travel

6.1.1 Dalton's law

6.1.1.1 The atmosphere is made up of a mixture of a number of gases, the main components being nitrogen, oxygen and water vapour. The atmosphere is not of uniform thickness, being greater at the poles than at the equator. The pressure it exerts varies accordingly and also with weather systems, but is considered to have a mean value at sea level of 100 kPa (760 mm Hg) which decreases with increasing altitude above sea level (figure 1).
6.1.1.2 Since Dalton's law states that the pressure exerted by a mixture of non-reacting gases is equal to the sum of the partial pressures of the component gases, the partial pressure of oxygen also falls with increasing altitude (figure 1). Oxygen forms 21% of dry air, nitrogen forming almost all the rest. At 3000 m (9842 ft) the partial pressure of inspired oxygen is 13.3 kPa and at 8900 m (29 200ft, the altitude of the top of Mount Everest) the partial pressure of oxygen falls to approximately 6.2 kPa.
6.1.1.3 Conventional commercial aircraft fly at altitudes from 6500 m (22 000 ft) to 13 500 m (44 000 ft), and without a pressurised aircraft cabin would be untenable for humans.
6.1.1.4 In the USA the cabins of commercial aircraft are required to be pressurised to a pressure equivalent to an altitude

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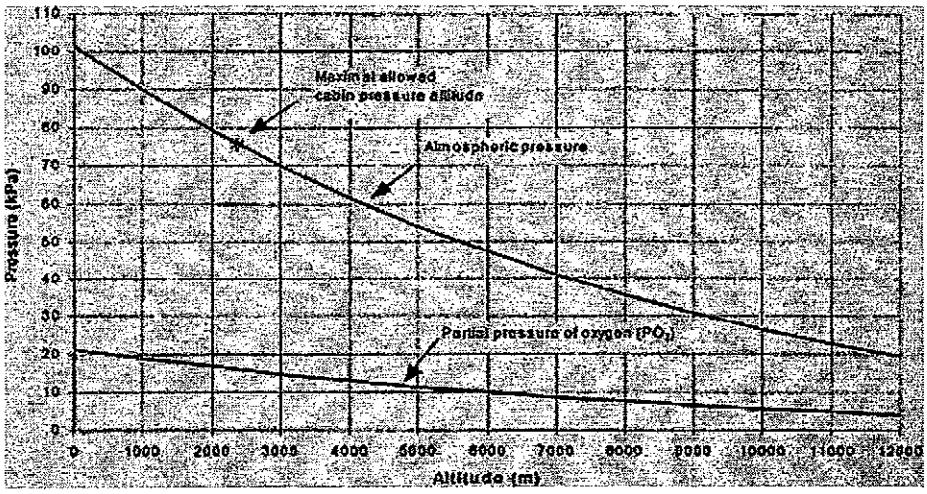

Figure 1 Variation in atmospheric pressure with altitude. Reproduced with permission from Committee on Air Quality in Passenger Cabins of Commercial Aircraft.[172]

('pressure altitude') of 2438 m (8000 ft), which should never be exceeded even at their highest attained flying altitude.[8] Similar regulations apply in the UK and Europe.[9] At this pressure altitude the partial pressure of oxygen is approximately 15 kPa.

6.1.1.5 It is assumed that the required cabin pressure is maintained at all times, but it may vary at different stages of the flight. There are suggestions that, although the aircraft systems are designed to maintain this pressure altitude requirement, there are still wide variations of cabin pressure during flight.[10] Other studies, however, have not supported these suggestions and, in modern aircraft, point to consistency of cabin pressures well above the minimum requirement.[11]

6.1.2 Boyle's law

6.1.2.1 Boyle's law states that, for a given amount of a gas at a constant temperature, the volume is inversely proportional to the pressure. This has consequences for those changing altitude as gas in body cavities expands on increasing altitude and falls again on descending. This has no effect on the heart and circulation but has implications for those who have had recent surgical procedures when air that has collected in the thorax or pericardium may expand.

6.1.2.2 The volume of a gas is also related to its temperature (Charles' law), but these effects will not be considered here since the cabin temperature is artificially controlled.

6.2 Physiology of air travel

6.2.1 Hypobaric hypoxia

6.2.1.1 The most significant physiological change that occurs during flight is hypobaric hypoxaemia. Because the cabin is pressurised, the partial pressure of oxygen (pO_2) does not fall below 15.7 kPa. Table 1 shows the relative arterial oxygen pressure (paO_2), ambient pO_2 and barometric pressure for healthy adults at different altitudes up to the maximum pressure altitude of commercial aircraft. It is important to note that Pao_2 falls with age despite alveolar pO_2 remaining constant. This is thought to be due to age-related disparity between ventilation and perfusion.[18]

6.2.1.2 Above the age of 40 years a constant progressive fall in pO_2 occurs that can be approximated to a fall of 5 mm Hg (0.67 kPa) per decade, although the linearity of this equation at extreme old age is not certain.

6.2.1.3 When the oxygen dissociation curve for oxyhaemoglobin (figure 2) is applied to table 1 it shows that, at a pressure altitude of 2438 m (8000 ft), the cabin oxygen partial pressure is 118 mm Hg (15.7 kPa), paO_2 is 62–67 mm Hg (8.2–9 kPa) and oxygen saturation is 90–93%.

6.2.1.4 This estimate of arterial oxygen saturations concurs with measurements using continuous reading pulse oximeters. Cottrell and colleagues[14] measured oxygen saturation in 38 pilots on 21 flights of about 4 h duration. Maximal and minimal oxygen saturations were 95–99% (mean 97%) and 80–93% (mean±SD 88.6±2.9%), respectively. Of the subjects, 53% developed an oxygen saturation of <90% at some time during the flight. With elderly passengers this value is likely to be lower.

Table 1 Relationship of barometric pressure, ambient oxygen partial pressure (pO_2) and arterial pO_2 with change in altitude in healthy adults at rest

Altitude		Barometric pressure		Ambient pO_2		Arterial pO_2	
Feet	metres	mm Hg	kPa	mm Hg	kPa	mm Hg	kPa
0	0	760	101.3	160	21.3	95–100	12.7–13.4
6000	1828	609	81.2	128	17.1	66–71	8.8–9.5
8000	2438	564	75.2	118	15.7	62–67	8.2–9

Reproduced with permission from Slonim and Hamilton.[12]

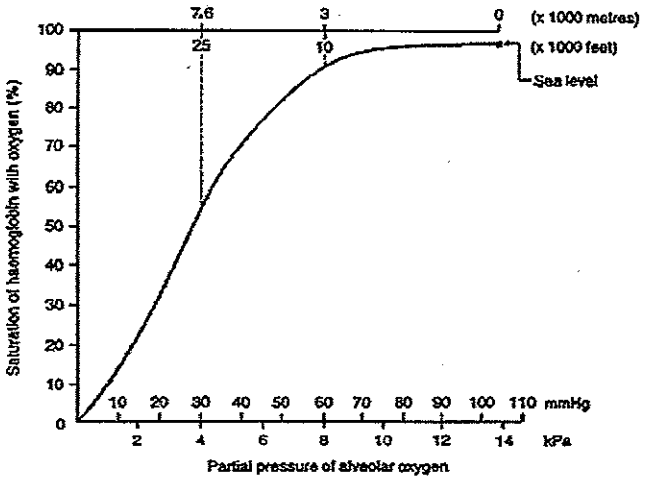

Figure 2 Oxygen dissociation curve from Aviation, Space and Environmental Medicine guidelines.[4]

6.2.2 Effects of hypoxia on the circulation and potential for myocardial ischaemia

6.2.2.1 Hypoxia is reported to have many effects on the circulation including local vasodilation of coronary and cerebral vascular beds, increase in heart rate, increase in systemic blood pressure,[15] increased myocardial contractility, increase in cardiac output[16 17] and increase in pulmonary artery pressure.[18 19] Responses to exercise are also altered,[20 21] and the effects of hypoxia change with the duration of exposure.[21]

6.2.2.2 Hyperventilation can occur which in turn activates pulmonary stretch receptors which override direct effects of hypoxia and produce tachycardia.[22 23]

6.2.2.3 Most of the studies from which the above data are derived are laboratory-based animal studies or have involved the study of active (walking, climbing or physical work) human subjects at moderate to extreme altitude for extended periods. Most of these reported effects do not become apparent until marked hypoxia with a paO_2 of ≤40 mm Hg occurs.[15 18 23]

6.2.2.4 Levels of paO_2 of 40 mm Hg would not normally be seen at modest altitude. Using the nomogram devised by Dillard and Ewald[24] for estimating the paO_2 at 2438 m (8000 ft) in patients with pulmonary disease, it can be estimated that to attain a paO_2 of 40 mm Hg would require the resting paO_2 at sea level to be 60 mm Hg and the forced expiratory volume in 1 s to be 29%—in other words, someone with very severe pulmonary disease.

6.2.2.5 There are few data on the effects of short-term isocapnic mild hypoxia at rest in the semi-recumbent position which is the state in which airline passengers find themselves.

6.2.2.6 A study by Phillips *et al*[25] measured the dose-response curve of isocapnic hypoxia and cardiac output in healthy men in a recumbent position and found an increase in cardiac output associated with relatively mild levels of hypoxaemia (table 2). The increase was entirely accounted for by increasing heart rate and after a 5 min peak tended to return to base level after 20 min. There was no increase in blood pressure or ventilation rate.

6.2.2.7 Another study[26] exposed normal subjects (age range 6–83 years) to an altitude of 2900 m, slightly above the maximum pressure altitude of an airline cabin, and analysed cardiovascular parameters at 1 h and over the following 24 h. Oxygen saturations fell significantly on arrival at altitude and there were significant increases in heart rate and systolic blood pressure across all age groups. All other parameters were normal and there was no correlation between the degree of hypoxia and the changes in heart rate and blood pressure.

6.2.2.8 Such an inconsistent relationship between blood pressure and degree of hypoxia was also shown by Thomson *et al*[27] when studying the circulatory response to isocapnic hyperoxia and hypoxia in normal healthy men. They exposed the subjects to each condition in a semi-recumbent position at normal room temperature for 1 h with a mean oxygen saturation during hypoxia of 82.6%. Hypoxia induced significant increases in heart rate and cardiac index and significant falls in systemic vascular resistance index and arterial stiffness.

Table 2 Haemodynamic and ventilatory response to hypoxaemia

Oxygen saturation (%)	97 (room air)	90.0	85.3	80.5
Cardiac output (l/min)	7.0	7.1	7.9*	8.4*
Heart rate (beats/min)	63	69.7	72	75*
Systolic BP (mm Hg)	125.6	127.6	127.1	128.2
Diastolic BP (mm Hg)	72.1	74.5	74.1	73.2
Respiratory rate (breaths/min)	17.1	17.3	16.6	16.8

*p<0.0005 for room air and 90% oxygen saturation.

6.2.2.9 In a paper even more relevant to this guidance, Wyss *et al*[28] took normal controls and a group of patients with angina (Canadian Cardiovascular Society functional class II–III) and angiographically documented coronary artery disease and exposed them to simulated altitudes of 4500 m and 2500 m, respectively. Haemodynamic parameters, ventilation rate, myocardial blood flow and coronary flow reserve using adenosine and bicycle ergometry were measured. There was a significant increase in rate pressure product at rest in both groups when exposed to their respective simulated altitudes and an increase in myocardial blood flow in both groups. However, there was a significant reduction in coronary flow reserve on exercise in the coronary artery disease group at the simulated altitude of 2500 m. There was no significant increase in ventilation in the coronary artery disease group at 2500 m.

6.2.2.10 It is reasonable to conclude from the above that there is no extreme change in circulatory parameters at rest with isocapnic hypoxia, even with oxygen saturations as low as 80%. Circulatory changes are confined to mild (probably transient) increases in heart rate, small decreases in systemic resistance which may result in increased cardiac output and some degree of increased coronary blood flow. Oxygen saturations of 80% would be unlikely to occur at pressure altitudes of 2500 m in commercial aircraft.

6.2.2.11 Conclusion

The mild hypobaric hypoxaemia induced by commercial airline flight may be expected to have little or no effect in precipitating myocardial ischaemia in people without critical ischaemia at sea level.

6.2.3 Effects of hypoxia in heart failure

6.2.3.1 It might be expected that hypoxia would have a deleterious effect on patients suffering with left ventricular dysfunction. However, pulmonary vasoconstriction may help protect the pulmonary microvasculature from increased pressure and falls in the systemic vascular resistance caused by hypoxia and thought to be mediated by nitric oxide[29] may limit rises in pulmonary venous and left ventricular filling pressures.

6.2.3.2 There is a paucity of literature on the effects of hypoxia on central circulatory variables in humans with heart failure, although work by Hobkirk *et al*[30] has shown that, in 21 patients with treated New York Heart Association (NYHA) grades III and IV heart failure with ejection fractions <40%, lying supine and breathing 15% oxygen caused a fall in arterial oxygen saturation to 86% but did not cause any significant symptoms. Blood pressure and pulmonary artery pressure rose significantly but heart rate did not (19 (90%) were on β blockers). Indirect measures of ventricular filling pressures suggested a fall.

6.2.3.3 In the Ideal Cabin Environment (ICE) Study,[31] 72 volunteers (mean age 54 years) with stable NYHA grade II were subjected to a simulated 7-h flight. The mean oxygen saturation of the group at 'sea level' was 94% and at 2438 m (8000 ft) cabin altitude mid 'flight' it fell to 91%, but there were no symptoms of dyspnoea or changes in blood pressure. The equivalent mean values for the normal volunteers (mean age 43 years) were 96% at 'sea level' and 93% at 2438 m (8000 ft).

6.2.3.4 The effect of hypoxia on workload in patients with heart failure has also been investigated. Agostoni *et al*[32] showed that patients with severe but stable heart failure (NYHA grades III and IV) were able to complete cardiopulmonary exercise without stopping even when subjected to hypoxia equivalent to an altitude of 3000 m (9842 ft).

6.2.3.5 Conclusion

The available evidence suggests that, for patients with stable heart failure including NYHA grades III and IV, short-term (up to 1 h) hypoxia at rest produces no significant deleterious effects. Periods of up to 7 h are tolerated by those with mild to moderate stable heart failure (NYHA grade II).

6.2.4 Effects of hypoxia on the ECG, arrhythmia and pacing thresholds

6.2.4.1 It might be anticipated that hypoxia and the associated increase in α- and β-adrenergic stimulation would increase the susceptibility to arrhythmia, particularly in the presence of myocardial ischaemia, but there are surprisingly few data on the subject. In a study of healthy men aged 50–64 years, increased atrial and ventricular ectopy was observed during acute ascent in a cable car to altitudes up to 2632 m (863 4ft) above sea level, the frequency of ectopy being proportional to the altitude.[33]

6.2.4.2 In another study healthy volunteers subjected to a simulated ascent of Mount Everest in a hypobaric chamber[34] showed transient changes in mean frontal QRS axis and voltages and increases in heart rate proportional to the degree of hypoxia. There were no ECG signs of ischaemia or arrhythmia even with an oxygen saturation of 49%.

6.2.4.3 It is not known whether hypoxia during air travel results in an increased predisposition to sustained ventricular arrhythmia and implantable cardioverter defibrillator (ICD) activation in susceptible individuals.

6.2.4.4 Profound hypoxaemia induced by inhalation of a gas mixture containing 10% oxygen has been shown to result in a significant and reversible increase in pacing threshold.[35] However, acute exposure of 13 patients with implanted pacemakers to an altitude equivalent of 4000 m, simulated in a hypobaric chamber, did not result in any alteration of stimulation thresholds after 30 min.[36]

6.2.4.5 Conclusion

Hypoxia is unlikely to produce an increase in susceptibility to arrhythmia or to have any adverse effect on pacing threshold at cabin altitudes likely to be encountered during air travel.

6.2.5 Effects of hypoxia on thrombogenicity (see also section on DVT/VTE)

6.2.5.1 Passengers with existing atheromatous coronary disease, whether diagnosed or not, may be at risk of acute coronary syndromes if being a passenger on a commercial flight has a prothrombotic effect which might predispose them to formation of occlusive coronary thrombus.

6.2.5.2 Hypoxia has been shown not to enhance exercise-induced activation of clotting factors in healthy athletes[37] and, although short-term and long-term periods at high altitude (>5000 m) have been shown to produce activation of clotting factors,[38 39] well-controlled studies of both normobaric[40] and hypobaric[41] isocapnic hypoxia simulating an altitude of 3600 m for 8 h revealed no significant effect.

6.2.5.3 Toff *et al*[42] simulated a long-haul flight by subjecting 73 low-risk 'passengers' to 8 h of either sedentary normoxia or hypoxia in a crossover trial using a hypobaric chamber. A small proportion of women taking oral contraceptives were included but subjects with factor V Leiden and prothrombin G20210 mutations were not. Measures of endothelial function, platelet activation and clotting showed no significant difference between the hypoxic and normoxic conditions suggesting that, in low-risk passengers, hypoxia has no prothrombotic effect over and above prolonged sitting.

6.2.5.4 In a study of predominantly high-risk 'passengers'[43] with a high proportion of participants taking oral contraceptives or bearing the factor V Leiden mutation, an 8-h flight was compared with other 8-h periods of sitting in a cinema or light ambulatory activity. There were increases in clotting factors (D-dimer, thrombin/antithrombin complex and prothrombin fragments 1 and 2). This suggests that, in high-risk individuals, mild hypoxia alone may have a prothrombotic effect.

6.2.5.5 Conclusion

Commercial airline passengers breathe air with a reduced oxygen content which results in low blood oxygen saturations. The levels of blood oxygen saturations attained appear to have little or no adverse circulatory effects which would make the passenger more liable to myocardial ischaemia, myocardial infarction, left ventricular failure or arrhythmia. Passengers already at high risk may suffer additional risk from hypoxia during flight. This conclusion is supported by the available evidence and applies to short or medium length flights. There is no adequate information to indicate whether extended flying (>12 h) might have adverse effects.

6.2.6 Effects of pre- and post-flight environment

Taking a flight with a commercial airline involves more than the time spent in the aircraft during flight, and the potential threats to the health of passengers with cardiac disease may occur before and after flight.

6.2.6.1 Anxiety: A significant proportion of the population (10–40%) report a fear of flying,[44] although this prevents few from completely avoiding it.[45] Fear and anxiety may be heightened at take-off and landing or if turbulence is experienced. The threat of terrorism adds to the fear of flying and can a have a major impact on passenger willingness to fly. Ito and Lee assessed the September 11 attacks in New York as having a major effect on reducing passenger numbers, an effect which continued at least until 2003.[46]

6.2.6.2 The security measures which are now in place mean passengers have to arrive well ahead of their flight time. Concern about missing the flight, uncertainties about where to check in and identifying the departure gate all add to the stress and mean passengers may be hurrying and anxious.

6.2.6.3 Frustration about delays has been shown to result in anger,[47] and anger is one of the more potent of the many mental stresses that may provoke myocardial ischaemia.[48]

6.2.6.4 Exercise: After arrival at the airport a considerable amount of physical activity is involved, much of it carrying, pushing or pulling bags, which may be well in excess of the passenger's normal exercise limits. Similar stresses apply at the destination airport and may also be exacerbated by the altitude or temperature of the destination. Many major destination cities are above 5000 ft, especially in North and South America, Africa and the East, and many have extremes of temperature. The potential for the airport stresses to induce a deterioration of a chronic condition is well recognised and most airports provide excellent services to assist the disabled passenger. These services can be found on arrival, but it is better to call ahead and make the arrangements beforehand.

6.2.7 Sleep deprivation and circadian rhythm disruption

6.2.7.1 Departure times in the early hours of the morning often mean that, with travel time to the airport and the delay before boarding, passengers may be awake much of the night before travel. This may result in sleep deprivation and disruption of

circadian rhythm that can also occur with flights that cross many time zones.

6.2.7.2 The circadian rhythm of the cardiovascular system reflects the changing sympathetic-vagal balance and is recognised to result in periods of cardiac vulnerability.[49] Studies of shift workers suggest that sleep deprivation and disruption of circadian rhythm may increase the risk of coronary events.[50] Heart rate variability as a marker of circadian variation may be altered in patients after infarction and in those with congestive heart failure, hypertension and diabetes[51] and can be modulated by β blockers.

6.2.7.3 Despite this, one study has shown that, in patients with heart failure, there is no relationship of circadian rhythm with sudden cardiac death[52] and there is no direct evidence of increased cardiac vulnerability resulting from occasional short-term disruptions such as those experienced by airline passengers.

6.2.7.4 Probably the most important effect of disturbed diurnal patterns for the passenger with cardiovascular disease is the ease with which the regularity of medications is disrupted. It is very difficult to adhere to a pattern of tablet-taking when the daily routine is altered, especially when confusion about the right time compounds the problem. For passengers with stable heart failure, angina or arrhythmia, it is very important that they maintain the regularity of their medication.

7. PASSENGER FLIGHT WITH CHRONIC CONDITIONS AND AFTER CARDIAC EVENTS OR PROCEDURES

Regardless of any direct deleterious effects that the aircraft cabin environment may have, it does not provide an ideal environment in which to manage medical problems and an acute in-flight event may result in diversion of the flight, poor medical outcomes or both. Cardiac events or procedures, diagnostic or therapeutic, may leave people at short-term high risk of a further event or complication. This acute risk normally reduces in the short to medium term until a stable situation is reached. If a cardiac event or procedure carries a significant risk of events or complications occurring, then consideration must be given to the time interval before the risk becomes acceptable and the cabin environment no longer a concern. It is generally the instability of the condition that predicts risk rather than the severity alone.

Although some considerations are relatively easy to define, such as the time it takes for post-thoracotomy intrathoracic air to be reabsorbed, most of the situations are complex. Patients have a multitude of factors that contribute to the risk of an event and consequently any guidance is necessarily a generalisation based on reasonable judgement and should not be considered absolute.

In the following section acute and chronic conditions and different cardiac procedures are considered and the appropriate time that should pass between the event and the flight suggested. The conditions and procedures considered are listed in table 3.

7.1 Acute coronary syndromes (ACS) including ST elevation myocardial infarction (STEMI) and non-ST elevation myocardial infarction (NSTEMI) and troponin-negative acute coronary syndromes

7.1.1 The observations of the limited effects of the cabin environment on cardiac physiology (see above) should be equally applicable to patients following an acute coronary syndrome, there being little or no deleterious effect.

However, following an acute coronary syndrome, patients may be at risk of events as a direct result of their condition. The early risk following an acute coronary syndrome is multifactorial and includes age, heart rate, ventricular function/Killip class, severity of the coronary disease and the degree of revascularisation.[53–58] In existing guidelines on fitness to fly, only acute myocardial infarction (AMI) is considered without distinction between different clinical presentations although it is sometimes separated into complicated and uncomplicated.

7.1.2 There is conflicting advice regarding the delay to flying following AMI, with varying guidance from professional bodies[4–7] and varying acceptance by passenger carriers. Data on which to base guidance are relatively sparse.

7.1.3 Retrospective analyses of patients following myocardial infarction have suggested that flying—both short and long haul—is safe at 2–4 weeks,[59 60] but in these studies the elapsed time between myocardial infarction and the flight was not an adjustable variable.

7.1.4 Another retrospective study[61] analysed 213 aeromedical repatriation patients after myocardial infarction and concluded that travel <2 weeks after AMI can be safely undertaken and that there is a low incidence of transfer-related complications. The patients were clinically heterogeneous, some having ST elevation myocardial infarction (STEMI) and some non-ST elevation myocardial infarction (NSTEMI), with varying numbers having undergone revascularisation. All the patients in this study were accompanied by an appropriately trained medical escort, and it is not clear whether this actively contributed to the low complication rate.

7.1.5 A small prospective trial[62] analysed the effects of flight 2 weeks after AMI in patients who were able to climb a flight of stairs and who were randomised to receive oxygen (2 l/min) or no oxygen during the flight. Holter monitoring was performed for ST shift and arrhythmia and oxygen saturation was measured throughout the flight. The authors noted some asymptomatic hypoxia (oxygen saturation <90%), which was also noted in a few patients in the paper by Thomas *et al*,[61] but only one episode of ST shift on the Holter tracing which was asymptomatic. There was no difference in outcome between patients with supplemental oxygen and those without. The authors concluded that, in this group of patients, commercial flight was safe and did not induce myocardial ischaemia, that supplemental oxygen as routine was not required and that medical attendants were unnecessary.

Table 3 Conditions, procedures and events considered in this section

Acute conditions	Chronic conditions	Diagnostic procedures	Therapeutic procedures
Acute coronary syndromes	Stable angina	Cardiac catheterisation	Emergency PCI
Acute left ventricular failure	Stable heart failure	Electrophysiological studies	Elective PCI
	Paroxysmal SVT		Insertion of pacemaker/AICD
	Persistent/permanent AF		CABG
	Cyanotic congenital heart disease		Surgical valve replacement
	Anaemia		Percutaneous valvotomy
			Ablation flutter, right-sided pathways, left-sided pathways, AF

AF, atrial fibrillation; AICD, implantable cardioverter defibrillator; CABG, coronary artery bypass grafting; PCI, percutaneous coronary intervention; SVT, supraventricular tachycardia.

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7.1.6 Longer distance medical transport is also reported in a retrospective study by Essebag *et al*[63] in which 51 patients were transported within 7 days of AMI, half of them high risk with Killip class II–IV. This study suggested that transfer was safe 3–7 days after admission.

7.1.7 These studies do not distinguish between high-risk and low-risk post-infarction patients. There is a spectrum of risk which will depend on the success of reperfusion, extent of myocardial dysfunction, predisposition to arrhythmia and presence of major complications.

7.1.8 STEMI

7.1.8.1 Figures from the Myocardial Infarction National Audit Project (MINAP) suggest overall mortality from STEMI is highest in the acute phase, falling to 2% after 10 days,[64] but these figures include all patients with STEMI and do not distinguish between high- and low-risk groups.

7.1.8.2 Successful revascularisation during primary angioplasty for STEMI confers a prognostic benefit in the short and medium term, especially in low-risk STEMI,[65] and in a cohort of 1791 patients treated by primary angioplasty de Luca *et al* devised a scoring system to identify the risk of 30-day mortality.[66] The authors' score card (see table 4) was able to identify patients at very low risk of early mortality or cardiac arrest (0.1% at 2 days and 0.2% at 2–10 days). They were able to demonstrate the stability of the score over the following year. Although patients with higher scores had higher early and late mortality in all groups, actuarial survival curves showed that relative stability of risk had been reached by 10 days. They concluded that a large proportion of patients following primary percutaneous coronary intervention (PCI) for STEMI may be identified as very low risk (score ≤3) with a stable risk from 2 days onwards. There was a strong correlation between risk score and ejection fraction (EF), patients with scores of 0–3 having mean±SD EF of 45±10% and those with scores of 4–6 having a mean±SD EF of 40±12%. This suggests that patients with low risk scores and EF >40% have a very low chance of events from day 3 onwards.

Table 4 Zwolle risk score for ST elevation myocardial infarction (STEMI)

Killip class	Points	Risk score	RR (95% CI) of death by 30 days
1	0	0–1	0.03 (0.008 to 0.13)
2	4		
3–4	9	2	0.09 (0.02 to 0.37)
TIMI flow post PCI			
3	0	3	1.04 (0.04 to 2.45)
2	1		
0–1	2	4	1.40 (0.5 to 3.98)
Age			
<60	0	5	2.48 (0.96 to 6.42)
≥60	2		
3 vessel disease		6	2.52 (0.75 to 8.46)
No	0		
Yes	1	7	5.99 (1.98 to 18.1)
Anterior infarct			
No	0	≥8	32.1 (18.6 to 55.8)
Yes	1		
Ischaemic time <4 h			
No	0		
Yes	1		
Total available score	16		

Reproduced with permission from de Luca *et al*.[66]
PCI, percutaneous coronary intervention; TIMI, thrombolysis in myocardial infarction.

7.1.8.3 The treatment of STEMI with primary angioplasty is increasingly widespread but still not the norm. In 2007, MINAP reported 4228 primary angioplasties in England and Wales out of a total of 27 199 STEMI (John Birkhead, MINAP, personal communication, April 2009). The British Cardiovascular Intervention Society (BCIS) audit returns[67] reported a slightly higher figure at 4858 primary angioplasties. Either way, it only represents about 15% of those with STEMI but it is rapidly increasing. The remainder received fibrinolytic agents or no reperfusion therapy. In these patients, successful reperfusion, extent of coronary disease and degree of left ventricular dysfunction are not known so quickly after hospitalisation and it is recommended that patients undergo further investigation and risk stratification using echocardiography within 48 h and coronary angiography during the same admission.[68] If there remain concerns about possible inducible ischaemia, stress testing may be undertaken in the following 4–6 weeks and appropriate revascularisation planned. Revascularisation with PCI or coronary artery bypass graft (CABG) almost halves the risk of subsequent unstable angina or death.[69]

7.1.8.4 Left ventricular EF is a marker of increased risk of ventricular arrhythmia and sudden cardiac death (SCD). Patients with an EF ≥40% and no ventricular tachycardia after day 2 are at low risk of SCD. Those with EF ≤30% and prolonged QRS duration may be considered for an implantable defibrillator (AICD).[70]

7.1.8.5 Bradyarrhythmia complications may require temporary or permanent insertion of a pacemaker. In either case there is a risk of pneumothorax (see section on pacemaker insertion).

7.1.9 NSTEMI and troponin-negative acute coronary syndromes

Patients with NSTEMI are at increased risk of death or further myocardial infarction and are recommended to undergo coronary angiography and appropriate revascularisation before discharge.[71]

7.1.10 Haemorrhage

Current management of all acute coronary syndromes involves the use of antithrombotic agents and anticoagulants as well as invasive strategies for revascularisation. There is a therefore a risk of haemorrhage related to the arterial access site as well as a risk of spontaneous bleeding from the gastrointestinal tract or, rarely, retroperitoneal haemorrhage.

7.1.10.1 Haemorrhagic complications are more likely with femoral artery than radial artery access,[72] but are unlikely to occur after 1 week. Subacute gastrointestinal haemorrhage is reported after both primary angioplasty[73] and fibrinolytic therapy[74] and may complicate the in-hospital course. Heparin and the small molecule IIbIIIa receptor antagonists have a short half life, but abciximab may still have effects at 7–10 days. Gastrointestinal haemorrhage from the use of the antiplatelet agents may also occur after discharge, but it is relatively rare at 0.07 per patient-year.[75]

7.1.10.2 If there are haemorrhagic complications during the in-hospital course after an acute coronary syndrome, it may alter the advisability of flying early after discharge.

7.1.11 Conclusion

Passengers who have suffered an acute coronary syndrome can be reasonably divided into those at very low risk who may safely fly as early as 3 days after the event, those at medium risk who

may fly from 10 days onwards and those at high risk or awaiting further investigation/treatment in whom flying should be deferred until a more stable situation is achieved.

Very low risk: age <65 years, first event, successful reperfusion, EF >45%, no complications and no planned investigations or interventions.

Low risk: EF >40%, no symptoms of heart failure, no evidence of inducible ischaemia or arrhythmia and no further investigations or interventions planned.

High risk EF: <40% with signs and symptoms of heart failure, those pending further investigation with a view to revascularisation or device therapy.

7.2 Heart failure

7.2.1 Acute left ventricular failure

The risk of hospitalisation for heart failure following AMI is highest in the few weeks after the infarction but stabilises after 45 days.[76] Episodes of acute left ventricular failure may be caused by an acute coronary syndrome (see above) or provoked by anaemia or infection on the background of chronic left ventricular dysfunction. Once any precipitant is identified and treated, most cases should be stabilised within 6 weeks and should be safe to fly.

7.2.2 Chronic heart failure

Passengers with stable chronic heart failure without recent changes in symptoms or medication are likely to be able to tolerate the mild hypoxia of the aircraft cabin environment even if they have severe heart failure.[30–32] It is advisable that they take the precautions of avoiding physical exertion at the airport and making sure that they take their regular medication. It is probably prudent for passengers who are severely limited with NYHA class IV symptoms not to fly without special consideration and the availability of in-flight oxygen.

7.2.3 Conclusion

Following an episode of acute heart failure, most patients should be stable within 6 weeks and be able to fly. With chronic heart failure there should be no restriction, although those with NYHA III and IV should consider airport assistance and request the availability of in-flight oxygen.

7.3 Stable angina

The available evidence suggests that it is unlikely that flying would precipitate acute myocardial ischaemia and no restriction should be applied. Passengers should be advised to allow plenty of time at either end of their journey to limit anxiety and haste. Passengers should be advised to seek assistance with personal transport at the airport if needed. They should also be advised to remember to take their medication in the usual way and at the usual times.

7.4 Arrhythmia

7.4.1 Paroxysmal supraventricular arrhythmia

Flight itself does not appear to induce paroxysmal supraventricular tachycardia, atrial fibrillation or atrial flutter, and providing the passengers are symptomatically stable with a low frequency of events, there should be no restriction to flying.

7.4.2 Passengers with permanent or persistent atrial fibrillation should be stable with appropriate rate control and anticoagulation.

7.4.3 Passengers with a history of ventricular arrhythmias should have these controlled before flying. Ventricular arrhythmias may be treated by implantable defibrillators (see section on DVT and VTE).

7.4.4 Patients with uncontrolled haemodynamically significant ventricular arrhythmias should not travel on commercial aircraft.

7.5 Cyanotic congenital heart disease

7.5.1 Patients with congenital cyanotic heart disease with Eisenmenger syndrome[77 78] are hypoxaemic with a consequent erythrocytosis and haemoglobin levels of 15–24 g/l. They might be thought to be at risk when flying owing to the effects of hypoxia and hyperviscosity.

7.5.2 The circulatory pathology, however, does not impair alveolar gas transfer and the arterial hypoxia does not result from low alveolar oxygen tension but from the central right-to-left shunting. As a result, small changes in the alveolar oxygen concentration have little effect on hypoxaemia. In addition, a chronic adaptation of a shift in the oxygen dissociation curve to the right has been suggested by Harinck *et al*[79] and subsequently confirmed in a study by Broberg *et al*.[80]

7.5.3 In the study by Broberg *et al*, 53 patients with Eisenmenger syndrome were compared with 48 acyanotic patients and a 10-year record of 1157 flights was studied. One or two of the patients reported symptoms of breathlessness during flight and were treated with in-flight oxygen therapy but the authors reported no serious adverse effects on account of the relative additional hypoxia experienced in-flight and suggested there was no justification for limiting air travel in these patients.

7.5.4 The erythrocytosis that occurs may increase the already existing risk of venous thromboembolic disease but with the additional possibility of a 'paradoxical' embolus. One patient in the study by Broberg *et al* was thought to have had a transient cerebral event.

7.5.5 Conclusion

Passengers with the Eisenmenger syndrome should not be restricted from flying although they are advised to request in-flight oxygen. They are also advised to maintain good levels of hydration, avoid alcohol and follow the guidance for VTE prophylaxis for high-risk groups (see section on DVT and VTE).

7.6 Anaemia

7.6.1 Reduced circulating haemoglobin restricts the oxygen-carrying capacity of a unit volume of blood and induces compensatory mechanisms in the circulation that result in increased cardiac output and ventricular stroke work.[81 82]

7.6.2 In the normal healthy heart, remarkably low haemoglobin levels can be tolerated[83] although chronic anaemia may precipitate angina in those with normal coronary arteries.[84] However, in an already compromised circulation, anaemia may risk pulmonary oedema or critical myocardial ischaemia.[85]

7.6.3 It is well recognised that anaemia commonly complicates chronic heart failure[86] and that treatment significantly improves the functional class.[87] The same applies to those with chronic angina[88] and following either PCI[89] or CABG.[90]

7.6.4 The Canadian Cardiac Society Guidelines on Flying[6] recommend that a haemoglobin level of 9 g/l is a threshold value below which travel is inadvisable in passengers who have undergone CABG.

7.6.5 Conclusion

When considering passengers with cardiovascular disease wishing to fly, physicians should consider the additional potential effect of anaemia on the underlying cardiovascular pathology.

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7.7 Cardiac catheterisation

Uneventful cardiac catheterisation should not restrict flying and patients may fly the next day. Bleeding from the access site may be a reason to defer a flight, as may other complications.

7.8 Elective PCI

Elective PCI varies in its complexity from simple single vessel PCI to complex three vessel treatments and the use of adjunctive devices and pharmacology. With aggressive anticoagulant and antiplatelet regimens, bleeding is a more likely event than with elective cardiac catheterisation and bigger contrast loads are more likely to cause renal impairment. In an uncomplicated case there is no reason to restrict flying for more than 2 days, but there should be discretion in the advice given by the physician and account taken of the degree of ventricular and renal dysfunction.

7.9 Pacemaker insertion including temporary systems, implantable cardioverter defibrillators (ICD) and biventricular devices

7.9.1 The insertion of any type of pacemaker including temporary wires, permanent pacemakers, biventricular pacemaker and implantable cardio-defibrillators (ICD) may involve subclavian vein puncture. Pneumothorax is a recognised complication. Before discharge an anteroposterior chest x-ray is routinely obtained to check the lead positions and to exclude pneumothorax, although it is possible that very small pneumothoraces may not be evident on the x-ray. In the absence of complications, patients are typically discharged from hospital within 24 h of implantation of the device.

7.9.2 Pneumothorax requiring intervention (aspiration or chest drain insertion) occurs in 0.6–0.8% of cases with less significant pneumothoraces (occupying <10% of the lung field) in a further 1%.[91 92]

7.9.3 Passengers should not fly with a pneumothorax because of the risk of gaseous expansion at altitude which may compromise respiratory function and the possible development of tension pneumothorax. If implantation has been complicated by pneumothorax, travel should be deferred for 2 weeks after full radiographic resolution.[93]

7.9.4 In the context of aeromedical repatriation with a medical escort, a stable patient with a persistent bronchopleural fistula may be able to fly safely with a chest drain attached to a one-way Heimlich valve.

7.9.5 In the absence of pneumothorax or other complications such as bleeding or electrode problems, there is no absolute contraindication to air travel within a 1–2 days of implantation. However, travel with a fresh surgical wound might be associated with discomfort and an increased risk of mechanical or infective complications. A small pneumothorax might not be evident or detected on the initial postoperative x-ray and a delay of a few days might allow an undetected pneumothorax to resolve. In the early weeks after pacemaker implantation, patients are generally advised to restrict arm movements on the ipsilateral side, to avoid raising the elbow above the shoulder and to avoid heavy lifting in order to minimise the risk of lead displacement. This may limit their ability to carry luggage and use the overhead lockers in the aircraft cabin.

7.10 Ablation

7.10.1 Electrophysiological studies with or without ablation therapy involve the often prolonged insertion of a number of catheters into the systemic veins. The clotting cascade is activated by the presence of the catheters and increases with the duration of the procedure.[94] Femoral vein thrombosis is a well-recognised complication and, although relatively rare as clinical complication, can be found subclinically much more frequently. In a study of 52 cases (68 femoral veins), non-occlusive thrombus was found in 17% but had disappeared after 1 week.[95]

7.10.2 Clinically evident thromboembolic complications including femoral vein thrombosis and pulmonary embolus have been reported in 0.14%,[96] 0.8%[97] and 1.04%[98] of right-sided procedures. This rate is sufficiently low and the benefit of aspirin as prophylaxis for systemic venous thrombosis insufficiently high[99] that use of routine aspirin after right-sided ablation is not a recommendation in the consensus document of the European Heart Rhythm Association.[100]

7.10.3 It is recommended that patients undergoing left heart electrophysiological studies and ablation involving transeptal puncture for atrial fibrillation or ventricular tachycardia should be given warfarin for at least 3 months and 1 month, respectively.[100]

7.10.4 Given the small additional risk of thromboembolism during or following flight, it is advisable for any passenger wishing to fly within 1 week after left- or right-sided ablation therapy for arrhythmia to be considered to be in a high-risk group and follow the advice for high-risk passengers with DVT/VTE (see below).

7.11 Open chest heart surgery including coronary artery bypass grafting and valve replacement

After the chest has been opened to perform cardiac surgery it is inevitable that some air will remain once the chest wound is closed. It takes 3–10 days for air to be reabsorbed. If any significant amounts of air remain in the pericardial space or in the thoracic cavity it may expand by up to 60% (see section on physics of flight) and this may be painful or dangerous.

Fast-track postoperative care and early extubation are leading to earlier discharge for patients following surgery[101] but without an impact on readmission to hospital.[102] Patients undergoing cardiac surgery which is without complication are mobile and at home in less than 10 days. Cardiac function should be improved following the surgery, but there may be complications of arrhythmia, pleural effusion, wound infection, anaemia, rhythm disturbances and left ventricular dysfunction. In patients without any of these complications, flying should be safe at 10 days. If complications do occur or there are symptoms of angina, heart failure or arrhythmia, patients should be advised about flying according to the guidance for those specific conditions (see appropriate section).

Patients may be in discomfort after cardiac surgery and should be advised not to carry heavy bags or hurry. If travelling between 10 days and 6 weeks after surgery, ground assistance may be required.

8. PACEMAKERS AND ICDS

8.1 Background

8.1.1 In the UK over 40 000 new patients receive an implanted pacemaker or ICD each year.[103] The majority of these devices are conventional pacemakers, but there are an increasing number of cardiac resynchronisation therapy (CRT) pacemakers and ICDs, with or without CRT capability.

8.1.2 It is estimated that there are currently 380 000 patients with pacemakers, 6000 patients with CRT pacemakers and 33 000 patients with ICDs (including 7000 with CRT capability) in the UK (D Cunningham, personal communication, 2009). The majority of pacemaker recipients are elderly (mean age at implant 75.5 years), but patients may receive a pacemaker at any age from infancy to adult life.

8.1.3 The main indications for pacing are atrioventricular block or sinus node disease, the predominant aetiologies being age-related fibrosis or ischaemic heart disease. Patients with pacemakers have a comparable survival to those without, and most return to a full and active life with little limitation of work and leisure activities in relation to the device.
8.1.4 Patients receiving CRT and ICDs tend to be younger (mean age at implant 69.9 and 62.5 years, respectively) and more often have significant underlying cardiac disease such as previous myocardial infarction, cardiomyopathy, impaired left ventricular function and heart failure. When assessing the patient's fitness to fly, the nature and stability of these underlying conditions require careful consideration and may be of greater significance than the presence of the device.
8.1.5 Although the majority of patients with an implanted device may travel safely by air, there are a few specific issues that should be considered before travel and a number of concerns for which the patient may require guidance or reassurance.

8.2 Electromagnetic interference (EMI)

8.2.1 Pacemakers and ICDs are inherently susceptible to electromagnetic interference (EMI) in the presence of strong electric or magnetic fields. Modern devices are well shielded against electromagnetic radiation and interference signals predominantly enter the devices via the lead which effectively acts as a loop aerial. Unipolar systems are more susceptible than bipolar systems owing to the larger electrode separation and greater effective area for inductive coupling.[104]
8.2.2 Most pacing systems are now implanted using bipolar leads which markedly reduce the risk of EMI. The devices incorporate specific design features to detect and reject EMI, and also a noise reversion mode (typically fixed-rate pacing) to which pacemakers will switch in the presence of EMI that is recognised as such by the device.
8.2.3 The possible effects of undetected EMI on pacemaker function include inappropriate inhibition of output (missed pulses or complete cessation of pacing) due to the input signal being falsely interpreted to be of cardiac origin and triggering, according to the operating mode. Alteration of the programmed settings may occasionally occur. The clinical consequences of EMI will be influenced by its duration and by the extent to which the patient is pacemaker-dependent. Fewer than one-fifth of patients have symptoms or fail to develop an adequate escape rhythm in response to abrupt inhibition.[105]
8.2.4 In an ICD, EMI may be falsely interpreted as tachyarrhythmia and trigger inappropriate therapies (antitachycardia pacing or shocks). Interference protection has improved substantially since the introduction in recent years of feed-through capacitors which are now incorporated into the majority of modern pacemakers and ICDs. These filters effectively attenuate most EMI in the frequency range from 30 MHz to 10 GHz.[106]

8.3 Airport security

8.3.1 Before boarding the aircraft passengers are normally required to pass through a metal detector gate. These gates typically incorporate one or more coils through which alternating current is passed to induce a primary magnetic flux. Movement of metallic objects within the coils results in a secondary magnetic flux which causes voltage changes in the coils that are used for detection. Studies of typical installations showed magnetic field frequencies between 0.1 kHz and 3.5 kHz with predominantly saw-toothed or pulsed waveforms and relatively high field strengths of up to 299 A/m (3741 mG) peak to peak.[107]
8.3.2 Nonetheless, clinical experience and experimental data suggest that clinically significant EMI is highly unlikely if patients walk briskly through the gate. In an early experimental study reported 20 years ago, 103 patients with implanted pacemakers were monitored as they walked through a typical airport metal detector gate.[108] No EMI or adverse effect on pacemaker function was detected. In a more recent study, 348 patients (200 with a pacemaker and 148 with an ICD) were monitored during passage through a security gate with no evident interference to device function.[109] Patients should be advised to walk through the gate at a normal pace if requested to do so and not to linger in the vicinity. They should, however, alert the security staff to the presence of the device as the metal casing of the device may activate the alarm.
8.3.3 In some instances, passengers may also be asked to submit to scanning using a hand-held metal detector. These typically operate at a higher frequency than the walk-through gates (89–133 kHz) with sinusoidal waveforms and substantially lower field strengths of up to 6 A/m (76 mG) peak to peak.[107] There has been at least one report of a spurious ICD shock triggered by a hand-held metal detector.[110] Consequently, some reviewers have suggested that patients with ICDs request a hand search as an alternative to use of the hand-held detector.[111] If a hand-held detector is used in a passenger with an implanted device, it should not be held close to the implant for longer than absolutely necessary and repeated sweeps to and fro should be avoided.
8.3.4 Notwithstanding the remoteness of the risk of clinically significant EMI, the US Transportation Security Administration and some other airport authorities permit or may even recommend that patients with an implanted device who have been so advised by their physician request a pat-down search rather than passing through the metal detector gate or being hand-wanded.[112 113] Bearing in mind the possibility that unadvertised changes in screening technology might be introduced, a reasonable approach might be to reassure patients that the risk of EMI from metal detectors is remote and suggest that they inform the security staff of the presence of an implanted device and be guided by them.

8.4 EMI during flight

8.4.1 The risk of EMI affecting pacemaker function in commercial aircraft has been comprehensively assessed in the context of pilots with pacemakers seeking recertification to fly.[114–117]
8.4.2 The aircraft environment has many sources of broadband radiation which may give rise to transients of relatively high field strength associated with activation of aircraft equipment. While these are a potential source of EMI, their brief duration makes it unlikely that they would cause clinically significant effects. Aircraft systems also include a wide variety of radio-frequency emitters in the HF, VHF and microwave bands which present a greater theoretical hazard. Several investigators have empirically assessed the effect of the environment in different types of aeroplane on the function of explanted pacemakers in a variety of test rigs. These either showed no effects or, at worst, one or two missed pacing pulses.[115 117–120]
8.4.3 Experimental studies in the 1980s showed that explanted unipolar pacemakers were susceptible to EMI at field strengths and frequencies that might be encountered in commercial aircraft.[114 115 117] Susceptibility was greatest in the HF and VHF bands and increased by modulation and pulsing of the radiated signal. However, exposure of similar implanted devices showed them to be unaffected at field strengths at which the explants

showed interference and failure. This provided support for the hypothesis that shielding by body tissues offers at least some protection against the effects of EMI. Explanted bipolar devices were also shown to be less susceptible to EMI.

8.4.4 Since these studies were conducted, there have been significant improvements in EMI protection of pacemakers and ICDs, notably the widespread use of feed-through capacitors which are most effective in the VHF and microwave bands.

8.4.5 The potential for portable electronic devices to interfere with aircraft systems is well recognised and the potential risks associated with the use of implanted devices in commercial aircraft have been considered by the aviation authorities. The risk of significant interference arising from implanted devices is likely to be negligible and both the US Federal Aviation Administration and the UK Civil Aviation Authority suggest that they should be exempted from the general restriction on the use of portable electronic devices, although policy is ultimately determined by the individual airlines.[121 122]

8.4.6 Conclusion

The accumulated data suggest that the risk of clinically significant EMI affecting pacemakers or ICDs in the aeroplane environment is minimal.

8.5 Cosmic radiation

8.5.1 It has been recognised for many years that cosmic radiation can disrupt the function of electronic devices, even at sea level.[123] The interaction of high-energy neutrons with the silicon nuclei in the static random access memory integrated circuit of an ICD can result in a 'single-event upset' that may affect the critical software control of the device.[124] The ICD architecture incorporates internal checks that routinely screen for such events and either correct them or, when a manufacturer-defined threshold is reached, trigger an automated software reset. These resets typically result in reversion from the programmed settings to the manufacturer's nominal settings for the device and they do not usually result in any significant hazard.

8.5.2 Events of this type are distinct from the very rare but more serious disruption of ICD function due to 'latch-up'. This causes battery depletion, transient loss of output and hardware reset which suspend tachycardia detection and therapy and has been reported in relation to the effects of cosmic radiation on a particular chip used in some specific older devices.[125]

8.5.3 The estimated overall frequency of ICD software resets induced by cosmic radiation is approximately 1 in 100 over 5–6 years (V Ivans, personal communication, 2009).

8.5.4 Experimental studies have shown that single-event upsets due to cosmic radiation are more frequent at higher altitude and it is likely that the risk will be increased several fold during air travel.[123] The radiation exposure at typical cruising altitude is about 100 times that on the ground.[126]

8.5.5 A single centre conducting ICD follow-up recently reported three cases of single-event upsets resulting in device electrical resets in three patients with an ICD during air travel.[127 128] In each case, time and date stamping of the event confirmed its occurrence during a flight.

8.5.6 In the absence of other reports, this is likely to be a relatively rare phenomenon but the observation of three events in a single follow-up clinic in as many years suggests that it may simply be unrecognised or under-reported. Software resets often trigger an audible patient alert, but this was overlooked by the patient in one of the three reported cases and the reset only came to light at a routine follow-up visit.

8.5.7 It may be prudent to alert patients with an ICD to the possible effects of cosmic radiation, particularly when a prolonged flight is planned. They may, however, be reassured that such interactions are unlikely to have a significant effect on the ability of the device to detect and treat life-threatening arrhythmia.[128]

8.6 Vibration

8.6.1 Activity-sensing rate-adaptive pacemakers and ICDs commonly incorporate a piezoelectric crystal or accelerometer to detect vibration from heel strike during walking or body motion, which is used to match the pacing rate to the user's perceived level of activity. These sensors may be sensitive to the effects of external vibration during vehicular transport of any sort. The extent and impact on pacemaker function of vibration during air travel have been extensively investigated in devices incorporating a piezoelectric crystal sensor.[117 129–131]

8.6.2 A more recent study has also assessed the function of two accelerometer-based rate-adaptive pacemakers in single-engine fixed-wing aircraft.[120] In fixed-wing aircraft, vibration levels are generally low and unlikely to cause significant rate rises except during take off, landing and turbulence when modest increases in pacing rate may occur.

8.6.3 In helicopters, however, vibration levels are high throughout the flight and sustained rises in pacing rate are seen which might be problematic for some patients.[117 129–131] The problem may be avoided by reprogramming the device to attenuate or disable the rate-response function prior to the flight. In the absence of a programmer, application of a magnet will usually disable the rate-adaptive function of a pacemaker and cause it to switch to asynchronous pacing at an increased rate.

8.6.4 Magnet application over an ICD will cause temporary suspension of tachyarrhythmia detection and therapy but does not usually affect bradycardia pacing functions.

9. DEEP VEIN THROMBOSIS (DVT) AND VENOUS THROMBOEMBOLISM (VTE)

9.1 World scheduled airlines carried some 2.1 billion passengers in 2006 on flights with an average duration of 2 h. This mean value has doubled over the last 30 years due to increasingly lengthy international sectors now spanning 12–14 h or more. Prolonged immobility, discomfort and dehydration may contribute to an increased risk of deep venous thrombosis (DVT) and its significant complication venous thromboembolism (VTE). These pathologies have a substantial morbidity and mortality. The Olmsted County Study[132] indicated an age- and sex-corrected annual incidence of 117 per 100 000 population while the Clinical Outcomes in the International Cooperative Pulmonary Embolism Registry[133] recorded a 3-month adjusted mortality of 15.3%.

9.2 In 1940 Simpson[134] recorded an increased incidence of pulmonary embolism in subjects confined in air raid shelters, but it was Homans[135] who first recorded a relationship between air travel and VTE. Symington and Stack later labelled this the 'economy class' syndrome.[136]

9.3 One and half centuries ago Virchow suggested that the triad—venous stasis, vessel wall trauma and altered coagulation—were implicated in venous thrombosis. Inherited increased susceptibility to venous thrombosis—the thrombophilic syndromes—include antithrombin III deficiency, protein S deficiency, protein C mutation, hyperhomocysteinaemia, factor V Leiden mutation and antiphospholipid antibodies. 'Idiopathic' venous thrombosis is associated with a 2–3-fold increased risk of neoplasm over the succeeding 12–24 months, affecting 13.0% of 854 patients followed for 8.1 years.[137]