Hemodynamic Effects of Commercial Flights. Potential Implication on Patients

Hernan Cohen Arazi*, Silvina Waldman, Norberto Casso and Mauricio Abello
Department of Cardiology, CEMIC Saavedra, Argentina

Abstract

Background: The main cause of in-flight mortality is a cardiac event. Most of the available information arises from small retrospective studies. The aim of the study is to analyze the cardiovascular response during a commercial flight in healthy volunteers.

Methods: Twelve healthy volunteers were studied. Prior to the flight all subjects were studied with physical evaluation, electrocardiogram (EKG), capillary saturation monitoring, echocardiogram, stress test echocardiogram, 24 h EKG Holter, 24 h blood pressure monitoring, and blood tests which included pro brain natriuretic peptide (pro BNP) and C reactive protein (CRP). During the flight, all subjects had a 24 h EKG Holter, an echocardiogram was performed in cruise altitude, blood pressure and capillary saturation was measured every 15 mins. One hour after landing blood pro BNP and CRP was dosed.

Results: There was a significant difference in the saturation observed between base and at 120 mins (98.4% + 0.5 vs. 93.1% +1.7, p<0.001). Blood pressure decreased from baseline to 120 mins (119 mmHg + 11.6/ 76.6 + 11 mmHg and 110 mmHg+ 7/ 70.2 mmHg+ 5, respectively, p=0.03). No differences were observed in the measurements of heart rate, CRP and proBNP. During the flight, 2 subjects showed transient regional wall-motion abnormalities, one of whom showed apical ballooning pattern.

Conclusion: We observed important decrease in arterial oxygen saturation during the flight and the arterial blood pressure was significantly lower compared to baselines measures. Two healthy volunteers showed cardiovascular dysfunction during the commercial flight. Haemodynamic changes should be considered in patients with cardiac conditions, after thoracic surgery and cardiovascular treatment.

Introduction

Every year, nearly 40 million people flight around the world [1]. Moreover, the average passenger age is rising due to an increased life expectancy in western countries, estimating that by 2030, half of the passengers in commercial flights will be over 50 years of age [2]. This translates into a higher number of people with cardiac diseases or with history of cardiac surgery that travel in commercial flights. Due to this matter, the question if flying is safe or if special care is needed appears to be a frequent issue, not only in hospitals but also in outpatient settings [3].

Retrospective data shows that the higher percentage of in flight mortality is due to cardiac events [1]. Reflecting this concern, in the 2001 Federal Aviation Administration stated that any flight that weighted over 7,500 pounds (3,400 kg) has to provide an external defibrillator in the emergency plane kit [1,4]. Moreover, since 2004, basic pulmonary cardiac resuscitation training is mandatory for in-flight assistants [4].

Commercial flights maintain a relative cabin altitude between 5,000 and 8,000 feet during routine flight. At this altitude, the barometric pressure (BP) decreases from a normal sea level value of 760 mm Hg to around 560 mm Hg, with lower baseline arterial partial pressure of oxygen (a OP), higher oxygen consumption and higher sympathetic tone – which predispose to arrhythmia, and deep vein thrombosis secondary to blood stasis [5-7]. These conditions could be more important and frequent in people who had a cardiac surgery.

Most information comes from retrospective studies, and there is no study evaluating the effect of commercial flights in the cardiac physiology. There are no references of these changes after heart or thoracic surgery. The aim of this study is to evaluate changes in cardiac physiology in a group...
of healthy volunteers. It could be a basement to analyze the situation when a physician needs to consider the patient risk for air travel.

**Methods**

This is a prospective, observational study, in which a cohort of healthy volunteers served as self-control. The objective was to assess changes in cardiovascular physiology during air travel. Male or female subjects, between 18 and 40 years were selected if they had no history of cardiovascular disease, hypertension, diabetes mellitus, or dyslipidemia. Subjects with history of arrhythmias, acute coronary syndrome, cardiac surgery, blood pressure> 135/85 mmHg, resting hypoxemia (SaO2 <97% at rest at sea level, breathing room air) and history of panic attacks or other anxiety disorders were excluded. Also subjects with abnormal electrocardiogram (AV block, bundle branch block, of pre-excitation syndrome, atrial fibrillation, or signs of atrial or ventricular overload) and poor acoustic window for echocardiogram were excluded from the study.

All volunteers who met the inclusion criteria and had no exclusion criteria had a complete medical history, physical examination, 12-lead electrocardiogram and an exercise echocardiogram. Moreover, Doppler echocardiogram, 24 h ambulatory blood pressure monitoring, 24 h holter EKG, pulse oximetry and a blood test (including C Reactive Protein (CRP) and proBNP) was performed before the study and in-flight, to each participant.

The study included data derived from three different trips, including 4 volunteers in each one. One of the subjects was re-evaluated again with a second stress echocardiography with exercise stress testing, within two weeks of the flight, and no ventricular dysfunction was observed. However, this subject repeated severe ventricular dysfunction (equal to 80% of baseline LV ejection fraction), with an apical ballooning pattern in the second flight.

The cabin pressurization and weather conditions were similar in the 3 flights.

There were no significant differences in heart rate at different times of flight (HR baseline = 70.5 (61.5-73) beats/minute, takeoff=72 (69-80) beats/min, p=0.27, 71 (65-75.5 bpm) to 8,000 meters of altitude, p=0.56 (compared to baseline).

Significant differences were observed in the blood pressure values; the maximum SBP and DBP were observed at the time of takeoff (130.6±19 mmHg and 79.8±6.8 mmHg, respectively). The lower SBP and DBP, were recorded at cruise altitude (at 120 mins: 110±7 mmHg: 70.2±5 mmHg, respectively) compared to baseline (119±11.6 mmHg: p=0.049 and 76.6±11 mmHg, p=0.03, respectively) and landing (122.2±14 mmHg: p=0.018 and 72±2.5 mmHg, P=0.043, respectively) (Figure 1 and 2).

A decrease in arterial oxygen saturation level was observed during the flight when compared with baseline, with lowest saturation levels recorded at 120 minutes in cruise altitude (98.4 vs. 93.1±1.7+0.5, p<0.001).

The echocardiogram performed in flight, showed no significant differences with the baseline diameters of the left ventricle (LV) and right ventricular function. The LV ejection fraction at baseline was 66% (65-72) and at in flight was 62% (55-69), (p=0.29). However, a decrease in LV ejection fraction was observed in two subjects, (76-46% in one and from 68-35% in another), generating severe ventricular dysfunction. One of these subjects was re-evaluated again with a second stress echocardiography with exercise stress testing, within two weeks of the flight, and no ventricular dysfunction was observed. However, this subject repeated severe ventricular dysfunction (equal percentage drop in LV ejection fraction), with an apical ballooning pattern in the second flight.

**Statistics**

The data was analyzed using STATA statistical program 9.0. Values are expressed as mean±standard deviation and interquartile range or average, as appropriate.

A comparative analysis between the baseline results and the measurements obtained in-flight was made; Student test and chi² were used to assess statistical significance considering a p-value <0.05 as statistically significant.

**Results**

Twelve male individuals (35±4 years) with no history of cardiac disease or coronary risk factor were included.

The cabin pressurization and weather conditions were similar in the 3 flights.

There were no significant differences in heart rate at different times of flight (HR baseline = 70.5 (61.5-73) beats/minute, takeoff=72 (69-80) beats/min, p=0.27, 71 (65-75.5 bpm) to 8,000 meters of altitude, p=0.56 (compared to baseline).

Significant differences were observed in the blood pressure values; the maximum SBP and DBP were observed at the time of takeoff (130.6±19 mmHg and 79.8±6.8 mmHg, respectively). The lower SBP and DBP, were recorded at cruise altitude (at 120 mins: 110±7 mmHg: 70.2±5 mmHg, respectively) compared to baseline (119±11.6 mmHg: p=0.049 and 76.6±11 mmHg, p=0.03, respectively) and landing (122.2±14 mmHg: p=0.018 and 72±2.5 mmHg, P=0.043, respectively) (Figure 1 and 2).

A decrease in arterial oxygen saturation level was observed during the flight when compared with baseline, with lowest saturation levels recorded at 120 minutes in cruise altitude (98.4 vs. 93.1±1.7+0.5, p<0.001).

The echocardiogram performed in flight, showed no significant differences with the baseline diameters of the left ventricle (LV) and right ventricular function. The LV ejection fraction at baseline was 66% (65-72) and at in flight was 62% (55-69), (p=0.29). However, a decrease in LV ejection fraction was observed in two subjects, (76-46% in one and from 68-35% in another), generating severe ventricular dysfunction. One of these subjects was re-evaluated again with a second stress echocardiography with exercise stress testing, within two weeks of the flight, and no ventricular dysfunction was observed. However, this subject repeated severe ventricular dysfunction (equal percentage drop in LV ejection fraction), with an apical ballooning pattern in the second flight.
We were unable to accurately measure pulmonary systolic pressure in all subjects during flight consequently, it is not reported.

There were no differences in CRP (basal and post flight: 0.73±0.23, p=0.15), or BNP values (pg 60 before and after the flight).

We analyzed differences between the two individuals who had ventricular dysfunction and the other subjects. A lower maximum HR (103.5±123.9±2.1 vs. 11.5 beats/min, p=0.03) and a lower 120 minutes mean arterial pressure (DBP + 1/3 the differential) was observed in the two individuals with ventricular dysfunction (58.5±4.6 without LV dysfunction and 48.5±1.2 mmHg in subjects showed LV dysfunction, p=0.04) compared to the other subjects without LV dysfunction.

We attempted to determine whether the two patients who had ventricular dysfunction also presented pre-flight parameters significantly different from the rest of the population included. The basal TAS was lower in these two patients (107±2.8 mmHg) compared to the rest of the subjects (121.4±11.2 mmHg), p=0.06; and left ventricular diastolic diameter (LVDD) was higher (51.5 vs. 48.4±0.7 mm±1.7 mm, respectively, p=0.04) compared to the rest of the evaluated population.

The basal EKG holter recorded frequent supraventricular extrasystolia (2250 beats) in only one patient, which appeared to be significantly higher during the flight (4298 beats).

**Discussion**

There are few clinical studies related to changes in cardiovascular physiology during commercial flights, and most of the available information is derived from studies using simulation tests in specialized laboratories [3]. Although the aircraft cabins are pressurized, they do not provide identical environment to that of sea level. Besides it is assumed that the cabin pressurization is constant throughout the journey, variations may exist. The pressurization changes affect oxygen concentration. Changes in pressure generate lower arterial blood pressure of oxygen (aO2P). In healthy individuals at rest, PaO2 is about 98 mmHg at sea level, and may decrease to less than 60 mmHg at the height a routine commercial flight achieves [3]. Consequent hypoxia, may have effects on vascular hemodynamics causing vasodilation, increased heart rate and blood pressure [8], increased myocardial contractility [9] and cardiac index and elevated pulmonary artery pressure [10,11]. The postulated mechanism for the increased cardiac output caused by slight levels of hypoxemia is tachycardia, which reaches a peak in the first 5 minutes to descend to baseline levels within 20 minutes on land. These effects are not usually apparent until severe levels of hypoxia (aO2P<40 mmHg and arterial oxygen saturation <80%) occur [12].

The respiratory system responds to hypoxia increasing the respiratory rate and tidal volume [13]. Previous studies have demonstrated an increase in sympathetic activity at altitudes between 1,500 and 3,000 meters high [12]. The sympathetic tone and its association with hypoxemia, cause a rise in systemic and pulmonary arterial pressure, with a consequent augment in the left ventricle diastolic pressure. The increased cardiac output is associated with an incremental risk of myocardial ischemia, which could further elevate the left ventricular diastolic pressures. Myocardial ischemia and high ventricular filling pressure may trigger ventricular arrhythmias [8].

Our study was consistent with other publication that showed hypoxemia during flight [13]. However, the subjects did not suffer significant variations in heart rate and a decrease in BP was observed during flight except for the landing and takeoff periods.

The most relevant finding was that two subjects showed significant ventricular dysfunction on echocardiography coincidently with lower values of PC and TAM. Because of the characteristics of the study we could not determine whether these changes were the cause or the consequence of decreased LV Fdy. In one of the 2 subjects we found a comparable echocardiographic pattern to the one that is observed in takotsubo syndrome, an acute and transient cardiomyopathy, characterized by asymmetric impaired ventricular motility [14]. Ischemia by coronary microvascular dysfunction, reperfusion injury, microinfarction, fat metabolism disorders induced by catecholamines or stunning have been suggested to be involved in the pathophysiology of this syndrome. Catecholamine levels are two to three times higher in patients with Takotsubo compared to those who suffer myocardial infarction, describing a situation of severe emotional stress as the main precipitant [15]. Air travel might raise anxiety. Ten to 40% of the population refers fear of flying, situations that can lead to a significant adrenergic discharge [16]. In our study the two patients with left ventricular dysfunction had no ischemia in echocardiography stress tests and no evidence of excessive adrenergic state was detected (they had lower FC and TA and equal stress score compared to the other subjects). Importantly, the decline in LV ejection fraction during the flight was not associated with elevation of proBNP. Troponin was not determined. Another possible explanation could be the decrease in the microcirculation in the coronary bed, as it has been demonstrated a decrease in the sublingual microcirculatory flow in subjects ascending to a maximal altitude of 77950 m above the sea level [17].

The number of subjects included did not allow us to determine predictors of hemodynamic patterns. In individuals over 40 years old, a progressive decrease in PaO2 of 5 mm Hg occurs per every decade [18]. Patients with chronic lung disease, who present PaO2 at sea level of 60 mmHg and very depressed forced expiratory volume in spirometry, can achieve risky levels of aO2P even with the cabin pressurization [19]. Experimental studies in patients with documented coronary heart disease have shown a significant decrease in coronary flow reserve which is mainly observed above 2500 m of altitude [20].

A previous study in men aged between 50-64 years postulated that hypoxia secondary to high altitude produced atrial and ventricular ectopy caused alpha and beta adrenergic stimulation [21]. Our hypothesis was that air travelling could be associated with increased QTc dispersion and ventricular late potentials, predisposing to ventricular arrhythmias [22]. Subjects in our study had no ventricular arrhythmias during the flight.

After the thorax has been opened to perform cardiac surgery it is inevitable that some air will remain once the wound is closed. It takes 3 to 10 days for air to be reabsorbed. If any significant amount of air remains in the pericardial space or in the thoracic cavity it may expand by up to 60% during a flight. It may be painful or dangerous. In patients without any of these complications, flying should be safe after 10-14 days [3,23]. However the Canadian Cardiac Society Guidelines on Flying recommends that a hemoglobin level of 9 g/dl is a threshold value below which travelling is inadvisable for passengers who have undergone CABG [24].

The association between inflammation and flight status has not been studied. Our group suggests that flight stress, prolonged rest with blood stasis, and hemodynamic changes could be associated
with an increased inflammatory tone with high levels of C-reactive protein. We did not find any difference in CRP, probably because of the short period between the flight and the sampling or due to the lack of real time relationship.

**Limitations**

For economic and safety reasons, a small population of healthy individuals was assessed and does not represent the true risk in the population with cardiovascular disease. Travelling time was less than 4 h, so alterations that occur on longer flights can be assessed.

**Conclusion**

During the flight variations in HR, TA and oximetry are observed, in healthy and young individuals. Impaired left ventricular function on echocardiography was detected in two subjects. This finding can have greater significance in those patients with history of cardiovascular disease and in those who have undergone a heart surgery. Larger studies are needed to confirm this data.

**References**