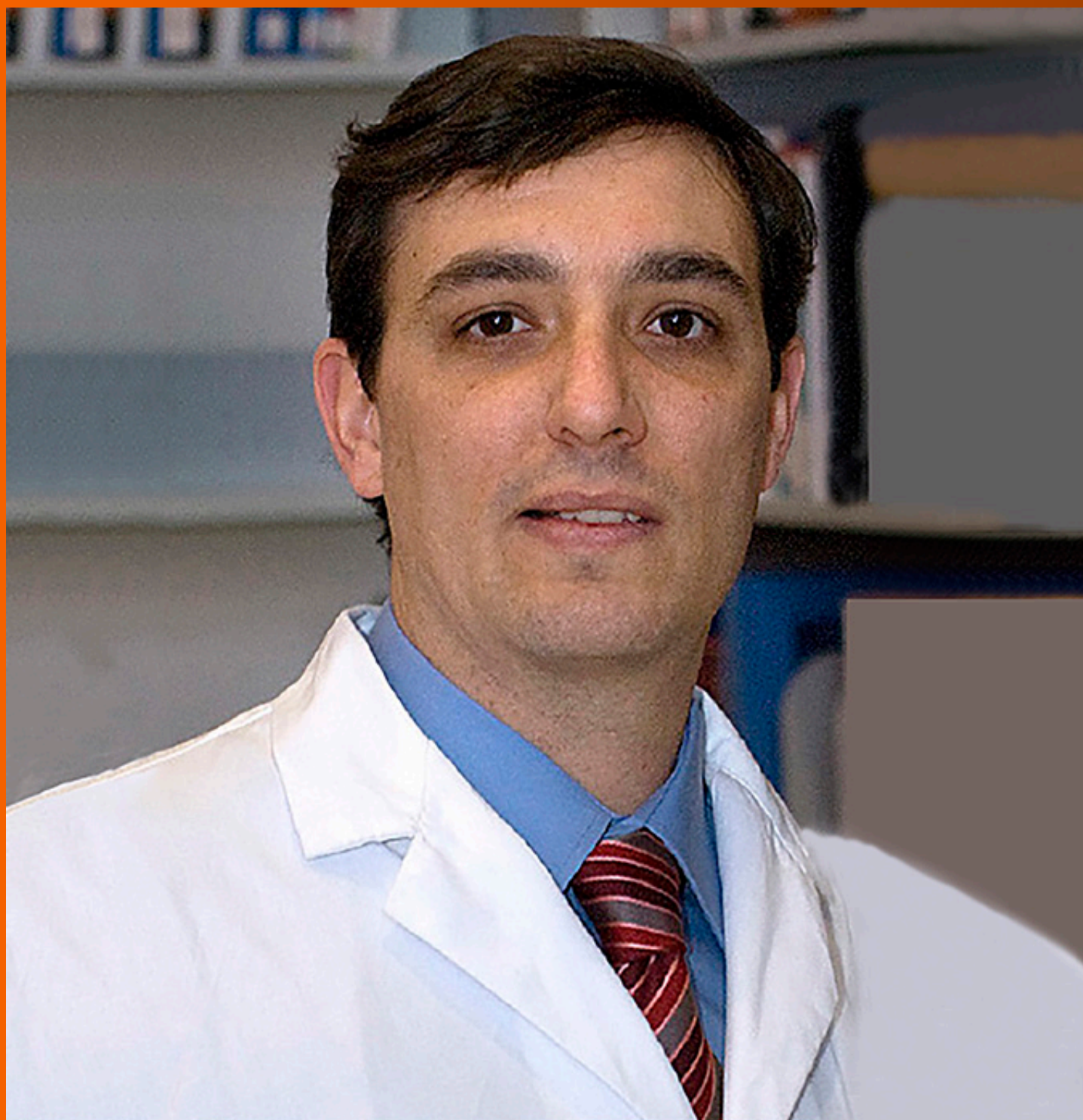


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- 97 Cardiovascular surgery in Turner syndrome - early outcome and long-term follow-up  
*Fuchs MM, Attenhofer Jost CH, Said SM, Hagler DJ, Connolly HM, Dearani JA, Egbe AC*

**LETTER TO THE EDITOR**

- 107 Demystifying airline syncope  
*Kingsley T, Kirchoff R, Newman JS, Chaudhary R*

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## Demystifying airline syncope

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### Abstract

Syncope forms a major part of medical in-flight emergencies contributing one-in-four in-flight medical events accounting to 70% of flight diversions. In such patients, it is important to elucidate the pathophysiology of syncope prior to diversion. Postural hypotension is the most common etiology of in-flight syncopal events. However, individuals without any underlying autonomic dysfunction can still experience syncope from hypoxia also known as airline syncope. Initial steps in managing such patients include positioning followed by the airway, breathing and circulation of resuscitation. These interventions need to be in close coordination with ground control to determine decision for flight diversion. Interventions which have been tried for prevention include mental challenge and increased salt and fluid intake. The current paper enhances the understanding of airline syncope by summarizing the associated pathophysiologic mechanisms and the management medical personnel can initiate with limited resources.

**Key words:** Syncope; Airline syncope; Aviation; Pathophysiology; Hypoxic syncope; In-flight emergency

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**Core tip:** Airline syncope is a major cause of in-flight emergencies. Understanding the pathophysiologic mechanism behind the event is a key in stabilizing the patient and determining if flight diversion is required.

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## TO THE EDITOR

Medical emergencies in-flight have been reported to be about 1 in every 604 flights. Syncope forms a major part of these emergencies contributing 25% to 37.4% of all in-flight medical events and leads to 70% of flight diversions<sup>[1]</sup>. Among healthy individuals, pathophysiology and its prevention remain unclear although several mechanisms have been hypothesized. The primary consideration in evaluating in-flight syncope is to assess the underlying mechanism prior to proceeding with diversion.

The most common pathophysiology underlying in-flight syncope events remains orthostatic vasovagal hypotension also known as postural hypotension. It is especially pronounced with underlying autonomic dysfunction like in diabetes, alcoholism, and in the setting of beta-blocker and benzodiazepine usage. The underlying mechanism is postural change from sitting to sudden standing which leads to venous blood pooling in legs, and a subsequent transient reduction in intracranial blood supply causing syncope. However, a significant number of individuals without any underlying autonomic dysfunction still experience syncope known as hypoxic syncope or airline syncope. Airplane cabins are routinely pressurized at 5000 to 8000 feet leading to an ambient environment of hypobaric hypoxia<sup>[2]</sup>. Different individuals have varying hypoxia tolerance depending on factors like physical fitness, fatigue, sleep disorder, history of smoking or any active or recent illnesses. Mechanistically, this exposure increases the minute ventilation rate due to stimulation of peripheral receptors, leading to a reduction in partial pressure of carbon dioxide. The reduced partial pressure can, in turn, inhibit ventilation thus promoting hypoxia. Hypobaric hypoxia also causes vasodilation which leads to reflex tachycardia and activation of carotid baroreflex resulting in increased sympathetic activity<sup>[3]</sup>. In healthy individuals, this increased sympathetic activity is associated with parasympathetic withdrawal. However, in airline syncope, there is an abnormal parasympathetic activation that results in activation of Bezold-Jarisch reflex leading to bradycardia and hypotension<sup>[4,5]</sup>. These mechanisms are further compounded by hypocapnic cerebral vasoconstriction and reduced cerebral blood flow. Other factors loosely associated with promoting hypoxia include immobility, drowsiness, gastrointestinal distention, and a high cabin temperature.

For management of syncope mid-air, the person experiencing it should be moved to the aisle and breathing/pulse should be confirmed. They should be placed supine position and legs raised to increase venous return. Supplemental oxygen initiated with measurement of vital signs and blood sugar. Majority of individuals recover within a few minutes with the aforementioned interventions. Upon recovery they should be rehydrated with oral fluids. However, in case the individual does not recover within a few minutes, intravenous fluids should be administered, and automated external defibrillator connected to check basic heart rhythm for any obvious arrhythmias or ST elevations. Close coordination with ground control regarding decision of flight diversion should be maintained.

Some proposed interventions to reduce the incidence of airline syncope include - evaluation of the effects of mental challenge (such as solving arithmetic problems) to maintain sympathetic activity in the brain on orthostatic tolerance. Although their pilot study had only 5 patients, it observed that mental challenges significantly improved the orthostatic tolerance of these individuals<sup>[6]</sup>. Another case report suggested prophylactic precautions with increased salt and fluid intake before the flight<sup>[7]</sup>. Much remains to be understood behind the underlying pathophysiology of airline syncope; however, improved education with aforementioned interventions may likely help reduce the incidence of syncope and the burden of cost of flight diversions.

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