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## AIR TRAVEL FOR INFANTS WITH CONGENITAL HEART DISEASE

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**ABSTRACT: INTRODUCTION:** It is well documented that, healthy person can tolerate the cabin environment of a commercial airline which is pressurized to the level of 5000 ft. however this environment brings profound physiological changes in patients with cardiovascular disease. With rise in number of patients travelling internationally for treatment of cardiac problems especially infants travelling by air for congenital heart disease treatment has increased in the recent time and is evident by the reports of medical incidents involving infants with congenital heart disease onboard.<sup>(1)</sup> Though the IATA medical manual mentions that adult patients with Eisenmenger Syndrome should not undertake air travel.<sup>(2)</sup> This article examines the case for infants with congenital heart diseases as there have been no previous studies reported.

**KEYWORDS:** Air travel for congenital heart disease.

**INTRODUCTION: METHOD:** A retrospective analysis of data collected from airline Pertaining to the number of infants with congenital heart disease transported, nature of the congenital heart disease and the subsequent outcome. Data was collected from 5 major airline operating in India.

**PHYSIOLOGICAL ASPECTS OF AIR TRAVEL:** The primary difference between the aircraft environment and the ground environment relates to the atmosphere. Contrary to popular belief, modern aircraft are not pressurized to sea level equivalent. Instead, on most flights the cabin altitude will be between 5000 and 8000 ft. (1524 m and 2438 m). This results in reduced barometric pressure with a concomitant decrease in partial pressure of oxygen (PO<sub>2</sub>). While the barometric pressure is 760 mm Hg at sea level with a corresponding PaO<sub>2</sub> (arterial O<sub>2</sub> pressure) of 98 mm Hg, the barometric pressure at 8000 ft. will be 565 mm Hg with PaO<sub>2</sub> of about 55 mm Hg.<sup>(3)</sup> If these last data are plotted on the oxyhemoglobin dissociation curve, we obtain a blood oxygen saturation of 90% at. Although most healthy travellers can normally compensate for this amount of hypoxemia, this may not be true for coronary, pulmonary, cerebrovascular, and anaemic patients. Because these patients may already have a reduced PaO<sub>2</sub> on the ground, further reduction in aircraft cabin pressure will bring them to the steep part of the oxyhemoglobin dissociation curve with a resultant very low saturation, which could cause distress and/or exacerbation of their illness

### BREIF OF CONGENITAL HEART DISEASE:

**INCIDENCE:** Congenital heart disease occurs in approximately 8 of 1,000 live births. The incidence is higher among stillborns (2%), aborts (10}–25%), and premature infants (about 2%)

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**TABLE 1:**

<b>Lesions</b>	<b>% of All Lesions</b>
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Ventricular septal defect	25–30
Atrial septal defect (secundum)	6–8
Patent ductus arteriosus	6–8
Coarctation of aorta	5–7
Tetralogy of Fallot	5–7
Pulmonary valve stenosis	5–7
Aortic valve stenosis	4–7
D–Transposition of great arteries	3–5
Hypoplastic left ventricle	1–3
Hypoplastic right ventricle	1–3
Truncus arteriosus	1–2
Total anomalous pulmonary venous return	1–2
Tricuspid atresia	1–2
Single ventricle	2
Double–outlet right ventricle	1–2
Others	5–10
-	

\*Excluding patent ductus arteriosus in preterm neonate, bicuspid aortic valve, peripheral pulmonic stenosis, mitral valve prolapse.

**CLASSIFICATION:** First, congenital cardiac defects can be divided into two major groups based on the presence or absence of cyanosis,.

**CYANOTIC CONGENITAL HEART LESIONS:** This group of congenital heart lesions can also be further divided based on pathophysiology: whether pulmonary blood flow is decreased or increased

**CYANOTIC LESIONS WITH DECREASED PULMONARY BLOOD FLOW:** These lesions must include both an obstruction to pulmonary blood flow (at the tricuspid valve, right ventricular, or pulmonary valve level) and a pathway by which systemic venous blood can shunt right to left and enter the systemic circulation (via a patent foramen ovale, ASD, or VSD). Common lesions in this group include tricuspid atresia, tetralogy of Fallot, and various forms of single ventricle with pulmonary stenosis. In these lesions, the degree of cyanosis depends on the degree of obstruction to pulmonary blood flow.

**CYANOTIC LESIONS WITH INCREASED PULMONARY BLOOD FLOW:** In this group of lesions, there is no obstruction to pulmonary blood flow. Cyanosis is caused by either abnormal ventricular-arterial connections or by total mixing of systemic venous and pulmonary venous blood within the heart. Transposition of the great vessels (TGV) is the most common of the

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former group of lesions. In TGV, the aorta arises from the right ventricle and the pulmonary artery from the left ventricle. The total mixing lesions include those cardiac defects with a common atria or ventricle, total anomalous pulmonary venous return, and truncus arteriosus.

**ACYANOTIC CONGENITAL HEART LESIONS:** Acyanotic congenital heart lesions can be further classified according to the predominant physiologic load they place on the heart as the lesion producing increased volume load and lesion producing pressure load.

**LESIONS RESULTING IN INCREASED VOLUME LOAD:** The most common lesions in this group are those that cause left-to-right shunts: atrial septal defect (ASD), ventricular septal defect (VSD), atrioventricular septal defects (AVSD, AV canal), and patent ductus arteriosus (PDA).

The pathophysiologic common denominator in this group is a communication between the systemic and pulmonary sides of the circulation, resulting in the shunting of fully oxygenated blood back into the lungs. The direction and magnitude of the shunt across such a communication depends on the size of the defect and the relative pulmonary and systemic pressures and pulmonary and systemic vascular resistances.

**LESIONS RESULTING IN INCREASED PRESSURE LOAD:** The pathophysiologic common denominator of these lesions is an obstruction to normal blood flow. The most common are obstructions to ventricular outflow: valvar pulmonic stenosis, valvar aortic stenosis, and coarctation of the aorta. Unless the obstruction is severe, cardiac output will be maintained and symptoms of heart failure will be either subtle or absent.

**DISCUSSION:** Though the effects of cabin environment on congenital heart disease are obvious the reasons many of them travel are lack of awareness among the physicians, Anxiety of parents for quick treatment and non-availability of published data.

The effect of cabin environment will not only depend on the duration of the exposure but also severity of the disease.<sup>(4)</sup> These factors are dynamic and may change dramatically with: Pressure changes during the ascent or descent of the aircraft; physiological changes in a newborn.

The jet airliner can climb at over 5000 feet per minute, and the cruising altitude is usually attained within 15 minutes. For this reason, "altitude" has been divided into "cruising", "take-off", "landing", and "ground". The cabin pressure during various altitudes may vary between 5000 to 8000 ft.<sup>(5)</sup>

	Age	Nature of Congenital Heart disease	Duration of flight	In-Flight Oxygen supplementation	Outcome
1	51 days	PDA	Short	yes	Completed the journey safely
2	60 days	VSD, PAH, PDA	Short	yes	Completed the journey safely

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3	30 days	TGA, PDA, CFO, PPHN	short	yes	Completed the journey safely
4	2 days	Not known		No	Expired
5	-	TOF	long	-	Developed severe hypoxia with Spo2 50% had to be resuscitated with defibrillator completed the journey
6	Not known	Not known	Not known	Not known	Expired
7	Not known	Not known	Not known	Not known	Diverted

Table 2

The basic pathophysiology in congenital heart disease is the altered oxygen capacity either due to shunting of deoxygenated blood back in to the systemic circulation or the mixing. This is further affected by the fact that the cabin barometric pressures results in a decrease in the partial pressure of arterial oxygen from about 95 mm Hg to about 56 mm Hg in healthy passengers. This represents only a 4 percent reduction in the oxygen carried by the blood, The important point is that the partial pressure of oxygen of 56 mm Hg lies on the flat part of the oxyhemoglobin dissociation curve. However Arterial oxygen saturation in children with Cyanotic congenital heart disease is about 83.9%<sup>(6)</sup> and lies on the steep portion of the oxyhemoglobin dissociation curve already, any small changes in the PaO<sub>2</sub> are proportional to changes in the SaO<sub>2</sub>. At ordinary cabin pressures the oxygen saturation may fall dramatically and increase the risk of hypobaric hypoxia.

In case of cyanotic lesions with decreased pulmonary blood flow, the degree of cyanosis depends on the degree of obstruction to pulmonary blood flow. If the obstruction is mild, cyanosis may be absent at rest. However, these patients may develop hypercyanotic ("tet") spells during conditions of stress. These hyper cyanotic spells may be exacerbated by the cabin atmosphere and could be fatal.

In patients with cyanotic lesions with increased pulmonary blood flow. Cyanosis is caused by either abnormal ventricular-arterial connections or by total mixing of systemic venous and pulmonary venous blood within the heart. The total mixing, in this group, deoxygenated systemic venous blood and oxygenated pulmonary venous blood mix completely in the heart, resulting in equal oxygen saturations in the pulmonary artery and aorta. Thus exposure to hypoxia can cause steep fall in oxygen saturation.

Transposition of the great vessels (TGV) is the most common of the former group of lesions. Systemic venous blood returning to the right atrium is pumped directly back to the body, and oxygenated blood returning from the lungs to the left atrium is pumped back into the lungs. The persistence of fetal pathways (foramen ovale and ductus arteriosus) allows for a small degree of mixing in the immediate newborn period; however, when the ductus begins to close, these infants develop extreme cyanosis.

In case of acyanotic congenital heart lesions though not clinically obvious there is some amount of compromise in oxygen carrying capacity of the blood. For lesions resulting in increased volume load, the direction and magnitude of the shunt across such a communication depends on

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the size of the defect and the relative pulmonary and systemic pressures and pulmonary and systemic vascular resistances. And for lesions resulting in increased pressure load, Unless the obstruction is severe, cardiac output will be maintained and symptoms of heart failure will be either subtle or absent. Though the effect of exposure to cabin atmosphere on short routes is debatable in these cases, effects in case of long haul route are obvious.

**CONCLUSION:** Even though air travel for adults with cyanotic congenital heart has been reported to be safe due to acclimatization secondary to exposure to chronic hypoxia(7), this does not apply for infants with cyanotic congenital heart disease and exposure to hypoxia could lead to fatal cyanotic spells.

In case of acyanotic congenital heart disease the effect of hypoxia are debatable however exposure to hypoxia on long haul flights could lead to dangerous fall in oxygen saturation.

We recommend that infants with cyanotic congenital heart disease should not be exposed to cabin atmosphere without supplemental oxygen.

However retrospective analysis of larger data available with airlines could provide conclusive evidence as a prospective study of exposing an infant with congenital heart disease would be impractical.

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