

CASE REPORT

CPAP suppression of awake right-to-left shunting through patent foramen ovale in a patient with obstructive sleep apnoea

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Thorax 2005;60:880–881. doi: 10.1136/thx.2004.027508

The prevalence of an echocardiographically visible patent foramen ovale (PFO) is higher in patients with obstructive sleep apnoea syndrome (OSAS) than in normal controls. We report a patient who presented with OSAS and right-to-left shunting (RLS) through the PFO in whom the RLS disappeared after treatment for 1 week with nocturnal continuous positive airway pressure (CPAP). This case shows the role of OSA in generating an awake RLS through a PFO and its possible reversibility by CPAP. The mechanism of reversible awake RLS through PFO in OSAS is discussed.

Obstructive sleep apnoea (OSA) can provoke right-to-left shunting (RLS) through a patent foramen ovale (PFO) during sleep.¹ In the awake state the prevalence of an echocardiographically visible PFO is higher in patients with the obstructive sleep apnoea syndrome (OSAS) than in normal controls.^{2–3} We observed a patient with OSAS and RLS through a PFO when awake whose RLS disappeared after 1 week of treatment with nocturnal continuous positive airway pressure (CPAP).

CASE REPORT

A 63 year old woman was investigated for suspicion of OSAS. She complained of major daytime somnolence, morning headache, nocturnal snoring, and dyspnoea at rest. She had no previous medical or surgical history and was not taking any medication. Her body mass index was 32.7 kg/m² and the apnoea/hypopnoea index (AHI) obtained by night polysomnography was 82/hour. She had a normal chest radiograph, spirometric parameters and carbon monoxide transfer factor, but her daytime arterial blood gas tensions (seated, breathing room air) showed hypoxaemia (PaO₂ 7.7 kPa) with normal PaCO₂ (5.6 kPa) and pH (7.41). RLS, as estimated from the 100% oxygen breathing test, was 19% (table 1). A transcranial Doppler ultrasound study with gaseous contrast test showed a large passage of microbubbles in the middle cerebral artery. A PFO with RLS was finally diagnosed by transoesophageal Doppler echocardiography with gaseous contrast test. Pulmonary arterial pressure was not measurable but the right ventricle was moderately dilated.

Nocturnal treatment with CPAP was started and the AHI fell to 3/hour. She continued CPAP treatment at home and was re-evaluated 1 week later. Room air PaO₂ obtained under the same conditions was 9.6 kPa, PaCO₂ was 5.5 kPa, and pH 7.42, and the RLS (estimated by 100% oxygen breathing test) was 6% (table 1). The transthoracic Doppler echocardiograph was normal and the right ventricle was not dilated. A transcranial Doppler ultrasound study with gaseous contrast did not show any passage of microbubbles while the patient was breathing tidally. A large passage of microbubbles was observed during a Valsalva manoeuvre. Arterial oxygen

saturation (SaO₂) was recorded using a finger pulse oximeter and the patient was asked to breathe through an inspiratory (–50 cm H₂O) and expiratory resistance (+50 cm H₂O). Before the resistance SaO₂ was 97%. After breathing for 1 minute through the resistance the SaO₂ fell to 92% but returned to 97% after suppression of the resistance.

DISCUSSION

An echocardiographically visible PFO was detected in 69%¹ and 27%² of awake patients with documented OSAS, compared with 17% and 15% respectively in non-OSAS control subjects. A link between OSAS and PFO can thus be suspected. Indeed, it has been shown that obstructive apnoea can induce RLS through PFO.³ But how can OSAS provoke a permanent RLS through PFO when the patient does not sleep? In our patient CPAP treatment of OSAS suppressed the RLS occurring through the PFO when she was awake. This suggests that CPAP reversed some non-fixed mechanism(s) of OSAS induced RLS. Two mechanisms can be hypothesised: (1) a large swing in pleural pressure and (2) pulmonary hypertension.

The Valsalva manoeuvre (large positive pleural pressure) is commonly used for detecting PFO⁴ and in our patient it did cause RLS. The Mueller manoeuvre (large negative pleural pressure) is known to increase venous flow to the heart, a condition facilitating RLS through PFO.⁵ Obstructive apnoeas may be regarded as a succession of Valsalva and Mueller manoeuvres as the patient expires and inspires against an occluded airway. Many patients with OSAS also have increased upper airway resistance when awake.⁶ Several factors could explain this increased upper airway resistance. Firstly, patients with OSAS have interstitial pharyngeal oedema with a mild inflammatory reaction.⁷ This is probably a minor but reversible component. Secondly, they have a diminution of pharyngeal diameter because of their anatomy.⁸ This is the main but partially reversible component. We hypothesise that our patient had increased upper airway resistance and that breathing against such a resistive airway (a situation comparable, although less pronounced, to that of a succession of Valsalva and Muller manoeuvres) results in RLS. After treatment with CPAP the upper airway resistance decreased and RLS disappeared. The decrease in SaO₂ (suggesting the reappearance of RLS) which we observed when she was asked to breathe through a 50 cm H₂O external resistance supports this hypothesis. The role of increased airway resistance in generating RLS through PFO is also supported by the increased prevalence of PFO observed in patients with severe chronic obstructive pulmonary disease (COPD).⁹

Abbreviations: AHI, apnoea/hypopnoea index; CPAP, continuous positive airway pressure; OSAS, obstructive sleep apnoea syndrome; PFO, patent foramen ovale; RLS, right-to-left shunting

Table 1 Summary of parameters in study patient before and after CPAP treatment

| | Before CPAP | After CPAP |
|-------------------------|--------------------|-------------|
| AHI (events/h) | 82 | 3 |
| PaO ₂ (kPa) | 7.7 | 9.6 |
| PaCO ₂ (kPa) | 5.6 | 5.5 |
| pH | 7.41 | 7.42 |
| RLS (%) | 19 | 6 |
| Right ventricle | Moderately dilated | Not dilated |

About 15–20% of patients with OSAS have resting awake pulmonary hypertension.¹⁰ Non-fixed pulmonary hypertension may be an alternative explanation for our observation. It is possible that our patient developed RLS as a result of OSAS induced pulmonary hypertension. Indeed, inversion of the right-to-left atrial pressure gradient can cause reopening of a previously closed foramen ovale and may explain, in part, the higher prevalence of PFO in patients with OSAS or COPD. By reversing OSAS induced pulmonary hypertension, CPAP could restore the normal left-to-right atrial pressure gradient and thus suppress RLS. However, when present, OSAS induced pulmonary hypertension is generally mild (in the range of 20–35 mm Hg) and its expected effect on the right-to-left atrial pressure gradient should be small. Moreover, in patients with OSAS, CPAP treatment resulted in a decrease in OSAS induced pulmonary hypertension after 4–12 months.^{11 12} In our patient awake RLS was abolished after only 1 week of treatment.

Increased pleural pressure swings due to increased upper airway resistance and OSAS induced pulmonary hypertension are two possible mechanisms which are not mutually exclusive. They can even reinforce each other if pressure induced RLS causes severe hypoxaemia and aggravates pulmonary hypertension. These two mechanisms could be associated with a possible “plasticity” of the foramen ovale which could stay patent during the day if repeatedly opened during sleep. This plasticity could be diminished by the prolonged enclosure of the foramen ovale after several nights of treatment with CPAP.

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Conflicts of interest: none declared.

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Received 30 April 2004

Accepted 26 June 2004

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