Editorial

Life-style modification at the edge

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We read with great interest a paper recently published by Kaw *et al*. in Chest investigating the association between sleep disordered breathing and post-operative atrial fibrillation (1). This is an important topic since approximately 1 in 3 patients will have atrial fibrillation post-cardiac surgery. Post-cardiac surgery atrial fibrillation (PCSAF) is associated with longer hospital stay, higher rates of stroke, and increased mortality (2,3). Post cardiac surgery atrial fibrillation is also the most common cause of rehospitalization after surgery and patients are five times more likely to develop atrial fibrillation in the future (4).

Many mechanisms have been proposed to explain the high rates of PCSAF including pre-existing structural changes to the heart, acute inflammation, oxidative stress from cardiopulmonary bypass, and increased excitability and automaticity of the atrial tissues peri-operatively (5). In recent years, there has been significant evidence to suggest a relationship between sleep disordered breathing (SDB) and atrial fibrillation (AF). Mehra *et al.* found that patients with sleep disordered breathing were four times more likely to develop atrial fibrillation even when adjusted for age, sex, body mass index, and coronary heart disease (6). Patients with more severe obstructive sleep apnea (OSA) also have higher rates of AF (7). The prevalence of SDB may be as high as 75% in the cardiac surgery population and many of these patients are undiagnosed (8). This has led to many studies investigating the relative contribution of SDB in the development of PCSAF.

Kaw *et al.* recently conducted a single centre cohort study which sought to address this question (1). They identified 190 patients with no previous history of AF, who had a polysomnography and subsequent cardiac surgery (coronary bypass graft surgery, valvular heart surgery, or both). Unlike many previous studies in this area, every patient included in their study had a formal polysomnography. Standard definitions of hypopnea and apneas were used and respiratory events were scored using the American Academy of Sleep Medicine task force deliberations (9).

In their study population, 93% of patients had and apnea-hypopnea index (AHI) ≥5 which is in keeping with sleep disordered breathing. The median apnea hypopnea index was 25.1. For every 5-unit increase in AHI, there was a statistically significant 6% increase in the odds of post-operative AF. However, when the analysis was repeated, adjusting for covariates of age, sex, race, body mass index (BMI), and hypertension, there was no statistically significant difference. The authors then proceeded to look at the effect modification of weight by analyzing the effect of SDB on PCSAF in two groups: BMI >32 kg/m^2 and BMI <32 kg/m^2. When a logistical model to adjust for age, sex, race, hypertension was applied, there was a statistically significant 15% increase PCSAF per 5-unit increase in AHI in patients with a BMI >32 kg/m^2. No statistical difference was seen in the group with BMI <32 kg/m^2.

Previous studies that have studied the impact of SDB on PCSAF have produced discrepant results. Studies which found no association between SDB and PCSAF may have had been underpowered to detect an effect due to smaller study sizes (10,11). Many previous studies which have shown a positive association between SDB and PCSAF have

relied on questionnaires, previous documentation of sleep apnea, or use of positive pressure ventilation (12,13). The prevalence of SDB may be as high as 75% in the cardiac surgery population, however, Wong *et al.* had a preoperative OSA prevalence of only 13% (12). van Oosten *et al.* similarly found 13% of patients had confirmed OSA and another 35% were high risk for OSA but had never had a formal diagnosis (13). Without polysomnography, it is likely that many patients in these two studies had undiagnosed sleep apnea and were analyzed in the wrong group.

This paper by Kaw *et al.* adds to the research in this field by being one of the few studies in which every patient underwent polysomnography and had a formal SDB. The authors were also able to accurately determine the severity of each patient's SDB and determine if the patient had central versus OSA. Since the AHI was known for every patient, the authors could analyze this as a continuous variable. They found a 6% increased odds of PCSAF per 5 unit increase in the AHI. However, this was no longer statistically significant when adjusted for cofounders. Their finding suggests that SDB may not be a major contributor to PCSAF. However, it is possible that since only 13 (7%) individuals with AHI of <5 were included, there may have been an underestimation of the effect of SDB contributing to PCSAF. Medications are also a major confounder. Unfortunately, the authors did not include medications in the perioperative period when reporting the patient characteristics. Disparate rates of antiarrhythmic use between the AHI <15 *vs.* AHI ≥15 groups could exaggerate or blunt the prevalence of AF in the post-operative period. Furthermore, exposure to sedation and narcotics is common in the post-operative period. These medications are known to alter a patient's sleep mechanics (14,15). A patient who was categorized as having no SDB or mild sleep disordered breathing may have a higher AHI or more desaturations in the setting of these medications.

The most interesting observation made by Kaw *et al.* was the effect modulation of body mass index. Obesity has been previously identified a risk factor for AF. In the post-cardiac surgery population, obesity is associated with an odds ratio of 1.1–1.2 (16,17). Kaw *et al.* found that an association between SDB and PCSAF was seen in patients with a BMI >32 kg/m^2, but not in those with a BMI <32 kg/m^2. A 15% increased odds of PCSAF per 5-unit increase in AHI was observed. This finding identifies obese patients with SDB as a higher-risk group for developing PCSAF. It also raises the question of whether sleep disordered breathing secondary to obesity portends a different risk for AF than SDB in normal weight individuals in which sleep apnea may be mechanistically different (related to otorhinolaryngological malformations).

We commend Kaw *et al.* for a methodologically vigorous study. Their analysis adjusted for important confounders which are often seen in patients with SDB and highlighted that obese patients with SDB are at higher risk for developing PCSAF. Therefore, patients with a BMI >32 kg/m^2 and SDB may benefit from targeted pharmacologic prophylactic treatment and should be monitored more extensively for the development of AF. The future research question in this field will need to focus on whether interventions and therapies for SDB will be effective in altering the rates of AF and its associated morbidity. It is also important to note that these patients with SDB also have higher rates of atrial fibrillation long term following their cardiac surgery (10). We must continue to be vigilant in looking for SDB and monitoring for AF in this population.

Acknowledgements

None.

Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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