Sleep disordered breathing and post-cardiac surgery atrial fibrillation

Roop Kaw1,2, Reena Mehra3,4,5,6

1Department of Hospital Medicine, 2Department of Anesthesia Outcomes Research, 3Sleep Disorders Center, Neurologic Institute, 4Heart and Vascular Institute, 5Department of Molecular Cardiology, Lerner Research Institute, 6Respiratory Institute, Cleveland Clinic, Cleveland, Ohio, USA

Correspondence to: Roop Kaw. Cleveland Clinic, Cleveland, Ohio, USA. Email: kawr@ccf.org; Reena Mehra. Cleveland Clinic, Cleveland, Ohio, USA. Email: MEHRAR@ccf.org.

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We thank the editor for inviting an editorial on our recent publication in Chest. We also appreciate and thank Chan et al. for a very thoughtful editorial. We hereby take the liberty of making a few observations for the readership of this journal as well as the investigators in this particular area. Postoperative atrial fibrillation (AF) is a growing problem and most predictors appear to be non-modifiable. The relation between obstructive sleep apnea (OSA) and new onset postoperative AF is complex and requires further characterization, but is also important as it may be a salient modifiable factor with potential benefit realized from the treatment of OSA.

There are some challenges with predicting new onset post-cardiac surgery AF (PCSAF) in regards to the definition of this outcome of interest. Investigators are divided between excluding patients with any history of prior AF; no AF 30 days before surgery or just normal sinus rhythm at the time of surgery. Society for Thoracic Surgeons in their 2012 update allows to code for post-operative AF if a patient went to the operating room in normal sinus rhythm (or a rhythm other than AF) and then developed post-operative AF requiring treatment in the postoperative period. Other studies (1,2) have also used asymptomatic AF noted on continuous ECG monitoring as an outcome of interest. Furthermore, lack of institutional protocols and varying thresholds in the management of PCSAF can lead to significant misclassification of this postoperative outcome. Lack of standardization across different published studies also then poses a challenge in comparability of the studies and interpretation of consistencies in results.

Although obese individuals in general are at higher risk of developing AF; it is not known whether obesity also increases the risk of PCSAF. Our meta-analysis group has been able to show that obesity solely confers a marginal but definitive risk of PCSAF (3), although many of these studies were not specifically designed to address the specific question of obesity as a risk factor for PCSAF. Whether the presence of OSA in the obese is the common link that predisposes to the higher risk of PCSAF needs to be further investigated. Most studies till date, including ours, have been limited by sample size. Higher risk of (non-surgical) AF has been noted with increased atrial size (4) as well as the presence of moderate to severe OSA (5). However, there are no data yet that point to left atrial volume as a common link in the development of PCSAF after cardiac surgery. Severe OSA has been shown to be associated with refractory AF in the non-surgical population (6). Future studies also need to address any relationship between severity of OSA and PCSAF. Our study was not able to identify any threshold effects for severity of sleep disordered breathing leading to higher chances of PCSAF. The obesity modifier effect in our study was observed not only with severe OSA but also with frequency >3% and >4 % thresholds of oxygen desaturation. Larger studies are needed to study the potential hypoxic and non-hypoxic predilections for...
PCSAF in this population. Furthermore, in the non-obese population; the role if any, of central SDB in predisposing to PCSAF needs to be better defined.

Another question of interest is whether the risk of development of new onset AF after cardiac surgery among patients with OSA is specifically highest in the immediate postoperative period or when the follow-up is extended beyond 30 days subsequent to (1). The fact that most patients late in the post-operative stage (before or after) do not have continuous ECG monitoring makes it difficult to detect asymptomatic PCSAF or paroxysmal AF after surgery.

More importantly, whether prior use of nocturnal positive airway pressure (PAP) therapy can decrease rates of PCSAF as suggested by Wong et al. remains unclear (7). P wave characteristics (duration; P wave-index and dispersion) can be used as predictors of PCSAF and have recently shown to be modifiable by PAP therapy in patients with OSA (8). Most of the literature till date is limited in the documentation of use/adherence of PAP in the perioperative period.

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Footnote

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References


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