Why Would Physiologic Support with Continuous Positive Airway Pressure Not Improve Outcomes in Patients with Atrial Fibrillation with Sleep Apnea?

To the Editor:

Traaen and colleagues examined the benefit of continuous positive airway pressure (CPAP) in the management of atrial fibrillation (AF) in patients with sleep apnea (SA) (1). However, the study's unexpectedly low statistical power and short treatment duration fail to strongly support the conclusion that CPAP is not beneficial in patients with AF, a benefit previously seen in observational studies.

The event rate used by Traaen and colleagues for power analysis was dependent on participants being in AF ∼34% of the time as in a previous study. However, in this study, the participants only spent ∼5% of their time in AF. Based on rough *post hoc* estimates, this unanticipated finding substantially decreased the power of the study to well below the 80% threshold for avoiding type-2 error. Although the authors did concede that their data may suffer from type-2 error, they did not commensurately temper their conclusions, which would have required a much larger sample size to have sufficient power to suggest a conclusion of CPAP having lack of efficacy in AF.

In contrast, many non–randomized control trials (non-RCTs) and observational studies have demonstrated a favorable relationship between reduction in sleep-disordered breathing and lower AF recurrence (2, 3). Although RCTs are considered superior, this RCT did not reach sufficient power to abrogate previous results demonstrating a benefit of CPAP in AF management. Importantly, consistent outcome measures are required when comparing results in AF trials, such as the ectopy measure of effectiveness used in demonstrating a positive effect of CPAP treatment in AF (4).

SA and AF are both degenerative conditions that become more prevalent and more severe with age. Floras summarized the cardiovascular risks induced by SA and its "threat to homeostatic cardiovascular rhythms" (5). The deterioration in cardiac conduction that leads to AF takes years to develop. Perhaps it is overly optimistic to expect CPAP to reverse the decline of AF in only 5 months given the length of time it took for AF to develop. However, if CPAP only reduces nocturnal cardiovascular stress sufficiently to halt further decline in arrhythmias, this in itself is a win. The lack of improvement in daytime somnolence in this study does call into question the adequacy of CPAP quality and duration achieved in these patients and whether other physiologic improvements could even be expected.

Thus, although this study may be demonstrating that therapy of limited efficacy and duration does not reduce the number of manifestations of AF, it may be confirming that even some CPAP halts further deterioration in AF frequency. In contrast, other studies do demonstrate decreased AF recurrence with CPAP treatment after ablation (6). Given the lack of statistical power and potential inadequacy of CPAP in the study population, it is hard to interpret the lack of benefit portrayed in Traaen and colleagues' Table E6. To conclude that CPAP has no place in the management of AF might be parallel to suggesting metformin has no place in the treatment of insulin resistance because it does not reverse diabetic nephropathy in a period of 5 months. In both situations, prevention of further deterioration is a beneficial outcome.

We recognize that the work of Traaen and colleagues is a valuable contribution to this conversation surrounding the benefits of CPAP. However, suggesting that current data demonstrate CPAP has no therapeutic efficacy would be the wrong conclusion to draw. Withdrawing CPAP in the management of patients living with AF and SA would be a dangerous path to follow unless the studies suggesting a benefit from CPAP were to be refuted by an RCT of sufficient size and duration. Overall, this study is too underpowered to support the conclusions being put forward. ■

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