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Utility of home sleep apnea testing devices in patients with cardiac conditions—critical manual interpretation of the raw data is important

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A recent study [1] published in this journal utilized the ApneaLink (ResMed) home sleep apnea testing (HSAT) device with a single channel (nasal airflow) to screen adult patients with stable chronic heart failure for sleep apnea. By design, the authors did not aim to distinguish between central sleep apnea (CSA) and obstructive sleep apnea (OSA). The data from the device was autoscored. HSAT using the above device and polysomnography (PSG) was performed simultaneously on the same night. The authors reported a "strong and significant correlation" between the apnea hypopnea index (AHI) determined by ApneaLink and the AHI determined by PSG. Based on the above results, the authors suggest that ApneaLink could be used to identify heart failure patients with sleep apnea and thus in their opinion, relegate the need for a PSG in such patients, which they feel will provide a more cost-effective and timely diagnosis.

The premise and conclusions of this article are noteworthy. However, several things need to be noted. As is well known, patients with heart failure (and other cardiac conditions such as atrial fibrillation) are at risk for both CSA and OSA [2, 3]. HSAT devices can detect CSA [4] but are not validated for the diagnosis of this [5]. The management of CSA can be very different and more complicated compared to OSA. Thus, appropriately diagnosing the type of sleep disordered breathing that a patient may have is very important for adequately managing them.

The majority of the prior studies on HSAT devices have described their ability to primarily detect OSA. I am aware of only one prior study which compared a type 3 HSAT device (recording oxygen saturation, respiratory effort, and nasal air flow) against PSG for the detection of both CSA and OSA [6]. The HSAT device utilized during this study was the ApneaLink Plus (ResMed) and the patients included in this study were those hospitalized with decompensated heart failure. The authors of this study found a strong concordance between the central and obstructive AHI (all scoring performed manually) determined by this device and that determined by PSG. However, it is unclear whether these findings determined using the ApneaLink Plus HSAT device can be extrapolated to other HSAT devices. For example, in our sleep laboratory, we utilize the Alice NightOne (Philips Respironics) HSAT device. Like the ApneaLink Plus, it has an airflow sensor, a single effort belt which goes around the chest, and a pulse oximeter. In my experience, while this device does allow for identification of apneas, it can at times be hard to distinctly say whether these are obstructive or central in nature as the signal from the effort belt can often be hard to clearly interpret and this generally seems to be more of a problem in patients who have mild–moderate sleep apnea and generally seems to be less of a problem in patients who have severe sleep apnea. The reason behind this is unclear although it must be noted that in the Alice NightOne, both the ends of the effort belt feed directly into the device unit which also sits on the chest. It is possible that this arrangement may somehow through an unknown mechanism contribute to the degradation in the signal quality from the effort belt. Previously, we used to utilize the Alice PDx (Philips Respironics) HSAT device which in addition to the above noted sensors also has an effort belt around the abdomen. Utilization of this device, for the most part, allows one to clearly distinguish between obstructive and central apneas. The signal from the additional effort belt may contribute to this difference. Unfortunately, our sleep laboratory

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discontinued its use as it was bulkier compared to the device that we currently use. Interestingly enough, the effort belts in the Alice PDx, create a complete end-to-end loop around the chest and abdomen and connect indirectly to the device unit. Similar to this, the single effort belt in the ApneaLink Plus also creates an end-to-end loop around the chest and connects indirectly to the device unit.

Use of HSAT devices can certainly save time and money [7–10]. However, critical manual interpretation of the raw data is important especially so in patients with cardiac conditions who are at risk for CSA. This should include looking closely at the signal from the effort belts as well as the oxygen saturation trend which can show a very characteristic pattern of intermittent desaturation in patients with CSA which is different from that seen in patients with straightforward OSA.

Insurance factors which are a unique problem in the United States can be confounding. For example, in some of my patients with underlying cardiac conditions (such as atrial fibrillation, heart failure etc.) who have undergone a HSAT, the results have clearly shown the presence of sleep apnea though the distinction between CSA and OSA has been ambiguous. Under such circumstances, one should ideally obtain either a PSG or a split night study which would allow for accurate diagnosis of the patients baseline respiratory physiology during sleep. However, insurance companies often do not cover this and may allow only for a titration in the sleep laboratory. Under this scenario, if one does see a pathological number of central apneas during the titration, one is left wondering whether these are truly a part of the patients underlying baseline respiratory physiology or whether these are treatment emergent in nature. Some of the patients that I have mentioned about above, choose not to come to the sleep laboratory and preferred to simply go ahead with a trial of auto CPAP despite the ambiguous results on their HSAT. In such patients, at follow-up, if the download shows an elevation in the AHI, one should consider underling CSA as a possible etiology. However, at follow-up, if the download shows an AHI which is at goal, one is still left with some doubt as CPAP devices are known to under detect central events [11]. Either way, the presence of frequent central apneas on a HSAT or an ambiguous picture on a HSAT where one cannot clearly distinguish the underlying respiratory physiology, i.e., CSA vs OSA or a negative HSAT in a patient suspected to have sleep apnea [12, 13], should warrant further evaluation in the sleep laboratory. Patients who are more likely to have CSA (such as those with significantly depressed ejection fractions) should ideally speaking, from the very beginning, be preferably studied in the sleep laboratory. On the other hand, I have also been pleasantly surprised by the findings in other heart failure patients where the use of a HSAT device by itself was helpful. For example, I recall having a couple of young patients with severe heart failure, who for varied reasons could not be studied in the sleep laboratory. A HSAT in both these patients showed the presence of clear cut OSA and allowed for the initiation of CPAP which given the severity of their heart failure was started at an empiric low fixed pressure of 8–10 cm and gradually adjusted depending on the appearance of their CPAP download.

Summarizing, I do think that while HSAT can certainly be performed in patients with cardiac conditions such as heart failure, appropriate patient selection and critical interpretation of the results is necessary. In certain such patients, the use of HSAT may be more cost-effective and time saving though this may not necessarily hold true in every patient, some of whom may require in-laboratory sleep studies for further evaluation.

Compliance with ethical standards

Off label and investigational use Not applicable.

Conflict of interest The author declares no conflict of interest.

Disclosures None.

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