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Clinical Applications of Adaptive Servoventilation Devices
Part 2

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Adaptive servoventilation (ASV) is an automated treatment modality used to treat many types of sleep-disordered breathing. Although default settings are available, clinician-specified settings determined in the sleep laboratory are preferred. Depending on the device, setting choices may include a fixed expiratory positive airway pressure (EPAP) level or a range for autotitrating EPAP; minimum and maximum inspiratory positive airway pressure or pressure support values; and type of backup rate algorithm or a selectable fixed backup rate. ASV was initially proposed for treatment of central sleep apnea and Hunter-Cheyne-Stokes breathing associated with congestive heart failure (CHF), and numerous observational studies have demonstrated value in this setting. Other studies have reported varying efficacy in patients with complex sleep apnea syndromes, including those with mixtures of obstructive and central sleep-disordered breathing associated with CHF, renal failure, or OSA with central apneas developing on conventional positive airway pressure therapy. Patients with opioid-induced sleep apnea, both obstructive and central, may also respond to ASV. The variability in response to ASV in a given patient along with the myriad choices of specific models and settings demand a high degree of expertise from the clinician. Finally, randomized controlled studies are needed to determine long-term clinical efficacy of these devices. CHEST 2014; 146(3):858-868

ABBREVIATIONS: AHI = apnea-hypopnea index; ASV = adaptive servoventilation; BNP = brain natriuretic peptide; CAI = central apnea index; CHF = congestive heart failure; CompSA = complex sleep apnea; CSA = central sleep apnea; EPAP = expiratory positive airway pressure; HCSB = Hunter-Cheyne-Stokes breathing; HFrEF = heart failure with reduced ejection fraction; IPS = inspiratory pressure support; LVEF = left ventricular ejection fraction; MV = minute ventilation; NYHA = New York Heart Association; PAP = positive airway pressure; PB = periodic breathing; PSG = polysomnography; SRBD = sleep-related breathing disorder; S/T = spontaneous/timed

In part 1 of this two-part series, we discussed the operation of three adaptive servoventilation (ASV) devices.[1] In the present article, we discuss some of the clinical applications for this technology. Potential applications include hybrid sleep-related breathing disorders (SRBDs) associated with conventional positive airway pressure (PAP) treatment of OSA and complex or central SRBDs in patients with congestive heart failure (CHF); cerebrovascular, neuromuscular, and neurologic

Manuscript received July 30, 2013; revision accepted June 10, 2014.

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DOI: 10.1378/chest.13-1778

disorders; and sojourn at high altitude. In most cases, however, prospective randomized controlled studies are needed to determine whether ASV devices will enhance the clinical outcomes of these disorders. We have successfully used these devices in three types of SRBDs, including CHF with predominant central sleep apnea (CSA)/Hunter-Cheyne-Stokes breathing (HCSB), sleep apnea induced by long-term opioid medication use, and persistent complex sleep apnea (CompSA) in which OSA coexists with one or more of the following: treatment-emergent CSA, CSA due to high-altitude periodic breathing (PB), CSA due to a medical disorder without HCSB, CSA with HCSB, or primary CSA[2-6] (for reviews[7-10]).

We emphasize the absence in most cases of randomized controlled trials with ASV devices and that in some of the aforementioned SRBDs, conventional PAP devices with or without an oxygen bleed (and in particular, bilevel devices with a backup rate) may be effective and could be used. However, differences exist between conventional PAP and ASV devices that could be anticipated to improve long-term outcome. In contrast to ASV technology, bilevel devices generally use fixed pressure support values and backup rates. Thus, minute ventilation (MV) cannot fall below a fixed value and, hence, could be excessive, augmenting hypocapnia and promoting rather than suppressing CSA. Furthermore, during crescendo episodes of HCSB, the patient may perceive excess ventilation as uncomfortable and thereby curtail adherence. In addition to the lack of variation in MV, bilevel devices with fixed inspiratory and expiratory pressures may increase intrathoracic pressure, and the effect on cardiac hemodynamics can be unpredictable,[11,12] particularly in patients with CHF. Depending on the existing operating point on the Starling curve, the reduction in preload and afterload could decrease cardiac output rather than augment it. As discussed in part 1, we suspect that ASV devices with algorithms designed to achieve the minimum effective inspiratory pressure support (IPS), backup rate, and expiratory PAP (EPAP) should, in the long run, prove most effective in terms of patient comfort and preservation of hemodynamics[1]; these advantages may, therefore, improve adherence and outcome. The results of the long-term randomized controlled trials currently in progress are awaited to clarify these issues.

Congestive Heart Failure

Patients with CHF frequently suffer from CompSA, predominantly CSA/HCSB and components of OSA. Moreover, the proportion of CSA/HCSB vs OSA contributing to the overall apnea-hypopnea index (AHI) may vary with body position, time during the night, and sleep state. In a large number of these patients, CSA is not suppressed with CPAP use, and we recommend ASV therapy. CPAP, however, is the treatment of choice in patients with heart failure and exclusively (or perhaps predominantly) OSA, although CSA[13,14] may emerge with the use of this therapeutic modality as we observed in an early study.[13] In a study comprising 192 patients,[14] the prevalence of CompSA was estimated at 15%. The patients demonstrating CompSA were found to exhibit heightened CO_2 chemosensitivity, which has been shown to predispose to PB by increasing loop gain.[15,16] In as many as 53% of patients with CHF and heart failure with reduced ejection fraction (HFrEF), CSA is not suppressed during the first night of CPAP titration,[13] and this finding usually persists, with prevalence declining only slightly to 43% at 3 months.[17] At present, it is not possible to accurately predict whether a given patient will exhibit CSA/HCSB on CPAP. One study found that a low $Paco_2$ may be a surrogate for high loop gain and CPAP nonresponsiveness, but this was not an invariable relationship.[13] Further prospective studies phenotyping patients with heart failure are needed to determine whether high loop gain and low $Paco_2$ reliably predict CPAP nonresponse and obviate the need for a trial of CPAP titration. At present, we recommend continued use of CPAP only in patients in whom CSA is suppressed during the initial titration.[13,17] We recommend ASV titration when CSA/HCSB persists during the initial CPAP titration because we believe that continued use could be detrimental.[7-11,17] To emphasize, even though a small proportion of patients with CHF will exhibit resolution of CSA/HCSB over time when central events are not initially suppressed by CPAP therapy, the high failure rate (43% at 3 months)[17] and the inability to predict long-term success suggest that it is not beneficial to recommend CPAP therapy if the AHI does not fall to $<$ 15/h on the first night of titration.

There are many short- and long-term observational studies of ASV devices used to treat sleep apnea in HFrEF[5,6,18-49] and heart failure with preserved ejection fraction.[50,51] The following discussion concentrates on the studies that make specific points of greatest importance to the clinician.

Teschler et al[19] were the first to report using an ASV device (the MV-targeted ResMed-AutoSet CS) to treat CSA/HCSB in patients with CHF. The patients