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# A Failure of Adaptive Servo-Ventilation to Correct Central Apneas in Cheyne-Stokes Breathing

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CASE REPORT

An 80-year-old man with severe central sleep apnea due to Cheyne-Stokes breathing (AHI 41.2) caused by severe cardiac failure underwent a trial of adaptive servo-ventilation (ASV) by full face mask after failure of a fixed CPAP trial. Recommended procedure was closely followed and the ASV device activated normally during central apneas. Initial settings were EEP 5, PSmin 3, PSmax 15 on room air. The device did not capture the thorax or abdomen, as shown by lack of change in respiratory inductive plethysmography, despite expected mask pressure waveforms. Snoring was also detected during apneas with device activation. Desaturation continued, followed by arousals during hyperpnea. On the device, the patient clearly slept for 1-3 epochs during the central apneas only to awaken during hyperpnea. We hypothesize that the failure to capture may have resulted from "reverse" obstructive apnea, possibly due to glottic closure during ASV activation. We suggest that earlier manual adjustments to ASV in cases such as ours, prior to waiting for the recommended 20 to 40 min of sleep, may be appropriate in selected patients. We also consider additional interventions that may increase the likelihood of a successful trial.

**Keywords:** Central sleep apnea, Cheyne Stokes breathing, adaptive servo-ventilation, continuous positive airway pressure

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Central sleep apnea (CSA) or Cheyne-Stokes breathing (CSB) occurs in approximately 40% of patients with congestive heart failure.[1] These respiratory patterns produce severe sleep disruption with recurrent arousals and awakenings irrespective of systolic function[1,2] and are associated with decreased survival.[3] Common modalities employed to treat CSA/CSB include nasal oxygen,[4,6] continuous positive airway pressure (CPAP),[5,6] and bilevel positive airway pressure,[6] with success in some but failure in many.

Another approach to treatment of central sleep apnea consists of adaptive servo-ventilation (ASV), which can provide mandatory breaths during central apneas in addition to variable bilevel PAP. ASV has shown superiority to other modalities in decreasing the apnea-hypopnea index and increasing sleep quality in the form of N3 and REM sleep in patients with cardiac failure.[6]

We report a patient with CSB due to chronic systolic congestive heart failure and pulmonary hypertension whose central apneas did not correct with ASV. We observed failure to capture thoracic or abdominal motion by ASV despite careful adherence to the treatment protocol specified by the manufacturer.

## REPORT OF CASE

An 80-year-old Caucasian male was referred from pulmonary hypertension clinic to the sleep laboratory for complaints of increasing dyspnea on exertion and worsening pedal edema. Past medical history included myocardial infarctions requiring coronary stent placement twice, systolic congestive heart failure, mixed pulmonary hypertension, peripheral vascular disease, moderate chronic obstructive lung disease, and type 2 diabetes mellitus. His medications included warfarin, aspirin, clopidogrel, lisinopril, isosorbide, furosemide, carvedilol, bosentan, atorvastatin, and metformin. He did not regularly take narcotics but had a prescription for acetaminophen/propoxyphene for use as needed.

He has had multiple hospital admissions for congestive heart failure exacerbations. His ejection fraction was 15% by left heart catheterization. Right heart catheterization showed a pulmonary artery pressure of 80/33 mm Hg with both passive and non-passive components, and no response to nitric oxide inhalation. He began bosentan for his pulmonary hypertension and showed some improvement clinically. However, he developed atrial flutter which persisted but could not undergo cardioversion because of left atrial thrombus. He was worsening clinically; decompensated congestive heart failure led to the request for polysomnography. He had marked daytime sleepiness, with an Epworth Sleepiness Scale score of 21/24.

On examination, his vital signs were: blood pressure 156/66; heart rate 66; respiratory rate 18; temperature 96°F; and pulse oximetry saturation 98% on room air. His body mass index was 24 kg/m$^2$. He did not appear in respiratory distress at rest. On chest examination, he had bilateral decreased air entry with no adventitial sounds. Jugular venous distension was present. Heart sounds were normal and regular. There was no murmur heard. Extremities showed pitting lower extremity edema bilaterally up to his scrotum. Recent arterial blood gas showed pH 7.44, $pCO_2$ 27.8, and $pO_2$ 94 on room air.

Polysomnography (SomnoStar 9.1d) included monitoring of 6 standard EEG electrodes, 2 EOG electrodes, 3 chin EMG electrodes, right and left leg EMG, chest EKG in 2 leads, snore,

nasal pressure (BINAPS, Salter Labs, Arvin CA), oro-nasal airflow by thermistor (Thermisense Salter Labs, Arvin CA), and respiratory effort by respirator inductive plethysmography (Respiband adult, Palm Springs CA). **Table 1** shows polysomnography data. The initial diagnostic phase before CPAP showed central sleep apnea syndrome due to Cheyne-Stokes breathing (**Figure 1**). The apnea-hypopnea index AHI was 41.2 events/h, with 88% scored as central apneas. There was marked fragmentation of sleep, with an arousal plus awakening index of 53 per hour.

Split-study protocol was invoked, and the patient was titrated on fixed CPAP by nasal soft gel mask (Mirage, ResMed) with and without chin strap and full face mask (Comfort Gel, Respironics) from 6 to 10 mm Hg over 3 h 45 min. CPAP did not eliminate the central apneas, although saturation improved. He continued to have central apneas with arousals and awakenings.

Adaptive servo-ventilation (ASV, ResMed Corp, Poway CA) was performed within 2 months of the baseline split study using a ResMed Mirage Quattro large size full face mask with initial settings of end expiratory pressure (EEP) 5 cm $H_2O$, and minimum and maximum pressure support (minPS, maxPS) of 3 and 15 cm $H_2O$, respectively. We carefully followed the procedure recommended by the manufacturer.[7] Successful use of ASV was not achieved. We observed mask pressure waveforms (**Figure 2**) and frequency which varied somewhat during the study, consistent with activation of the device. We observed persistence of apneas, as identified by lack of motion in respiratory inductive plethysmograph tracings of abdomen and thorax despite pressure deflections observed in the mask pressure waveform (**Figure 2**). We also observed waveforms on the snore channel during device activation only (**Figure 2**). The patient typically had 1-3 epochs of N1 or N2 sleep during apneas followed by awakenings during hyperpnea. Technician observations confirmed the activation of ASV during residual apneas without capture of the chest or abdomen. Air leak was absent throughout the night with few exceptions. The baseline pulse oximetry saturation was improved on ASV.

Despite the failure to reach 20 to 40 minutes of sleep, minimum pressure support was manually increased to 4 cm $H_2O$, but there was no benefit. A repeat trial of ASV with further manual changes and sedation was recommended, but the patient declined.

**Table 1**—Polysomnographic data

| PSG Variable | Baseline | CPAP | ASV |
|---|---|---|---|
| Sleep efficiency (%) | 39 | 28 | 44 |
| Time in bed (min) | 172 | 224 | 396 |
| Stage N1 (% TST) | 53 | 71 | 57 |
| Stage N2 (% TST) | 44 | 25.8 | 36 |
| Stage N3 (% TST) | 0 | 0 | 7 |
| REM Sleep(% TST) | 3 | 3.2 | 0 |
| AHI (/h of sleep) | 41.2 | 38.7 | 36.2 |
| CAI (/h of sleep) | 36.9 | 35 | 33.9 |
| OAI (/h of sleep) | 4.6 | 3 | 2.3 |
| Min $O_2$ (any stage) | 69 | 75 | 88 |
| Min $O_2$ NREM sleep | 73 | 76 | 88 |

TST, total sleep time; AHI, apnea-hypopnea index.

**Figure 1**—Compressed view of a diagnostic polysomnogram showing ten 30-sec epochs

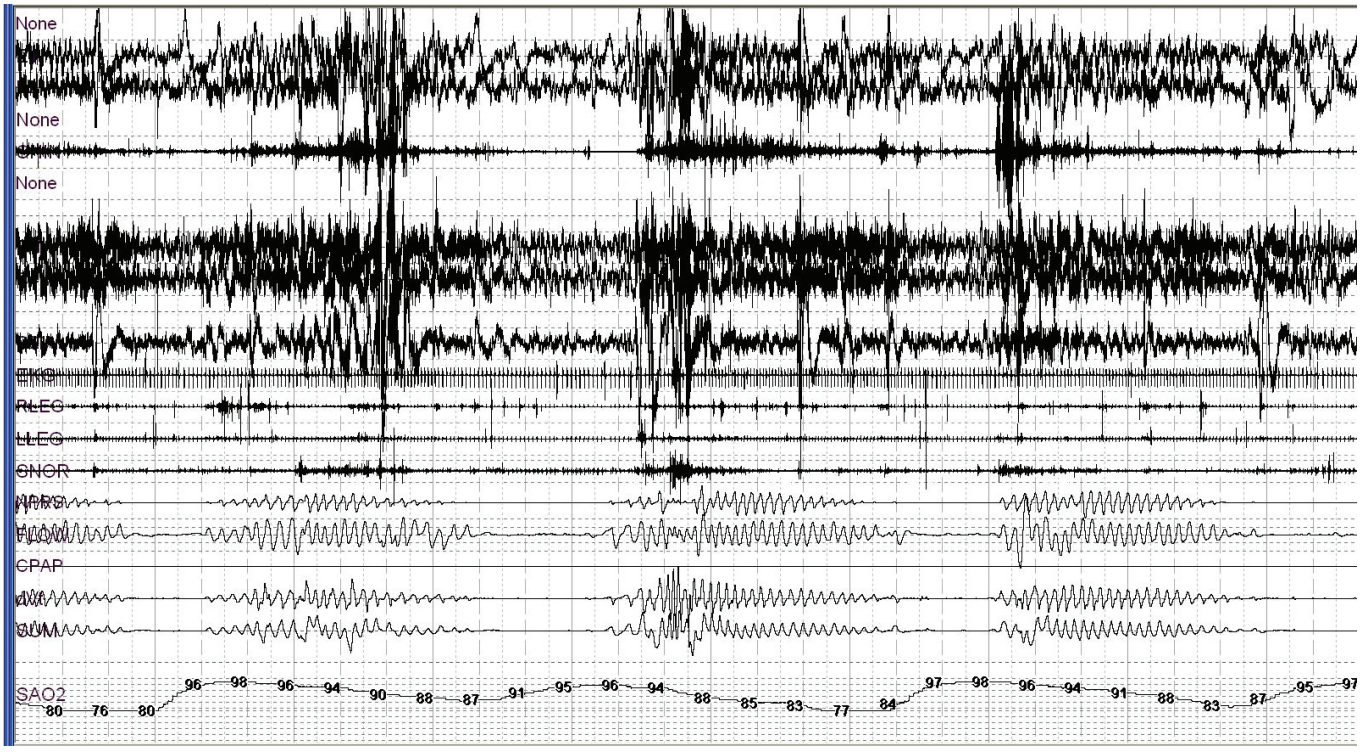
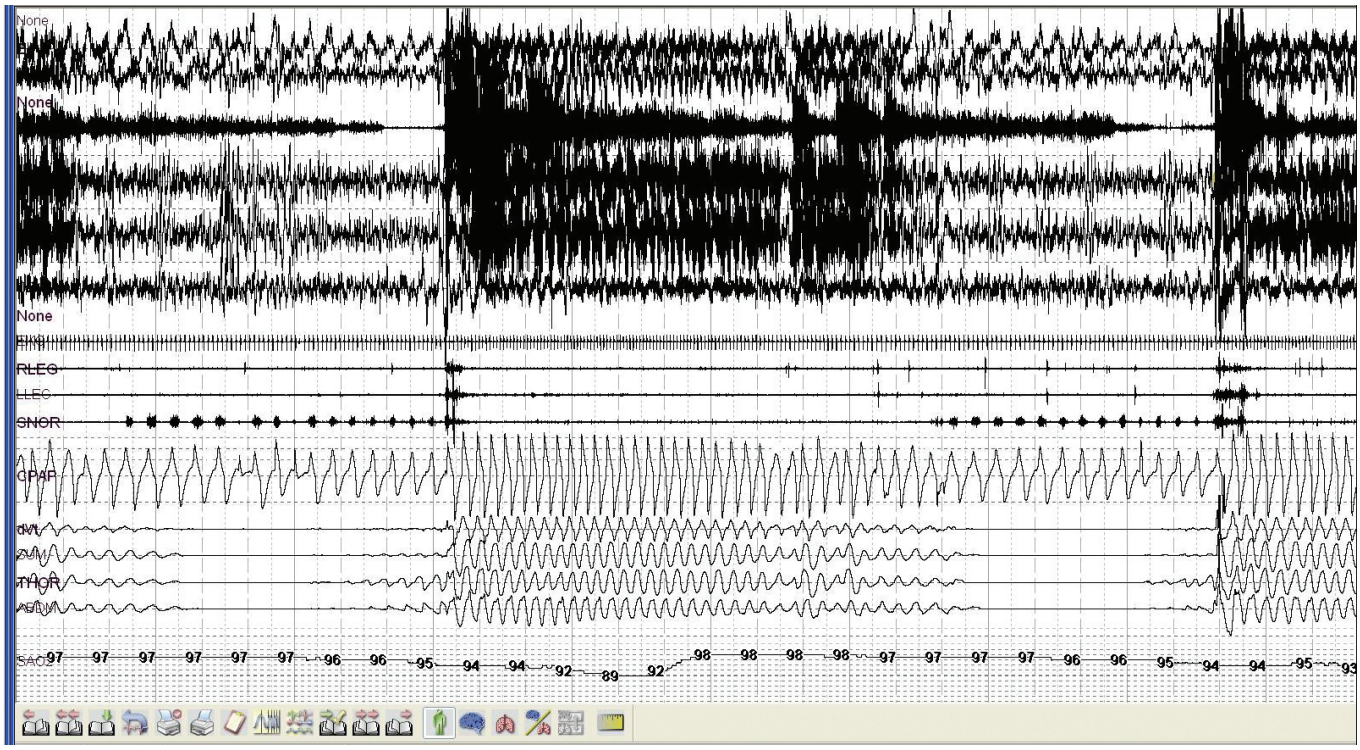



Figure shows 4 central apneas followed by hyperpneas with arousals. Desaturation is shown, also. CPAP channel is off. Nasal pressure and thermistor flow with central apneas are shown. Snore channel is silent during central apneas.

**Figure 2**—Compressed view of a treatment polysomnogram on ASV showing ten 30-sec epochs




CPAP channel is actually ASV showing mask pressure delivered by the device at the frequency shown. Note also signal on the snore channel during ASV activation.

## DISCUSSION

We followed the manufacturer's recommendation not to manually change settings before 20 to 40 minutes of sleep. Although the patient did sleep on ASV, this was only during the apneas for a few epochs. He continued to have awakenings due to hyperpnea following apneas. Later in the study, manually increasing the minimum pressure support level gave no benefit. Whether manual adjustment earlier in the study would have been beneficial was not determined but would be useful information.

In the present case, we hypothesize that glottic closure may have caused "reverse" obstructive apneas, caused by the device, resulting in the observed pattern of mask pressure waves with no chest or abdominal motion. The presence of "reverse" snoring during the device activation supports this hypothesis and also tends to exclude obstruction in the glossopharynx as the cause of failure to capture. Conceivably, full face mask application may have predisposed to reverse obstruction. In the study by Teschler and colleagues, nasal masks were used exclusively for ASV.[6] Other possibilities for failure to capture the chest and abdomen appear unlikely in our patient, who was not obese, and had only moderate obstruction with no restriction on pulmonary function testing.

We believe this is the first report of failure to capture chest wall and abdominal motion with ASV in peer-reviewed literature. The concept of reverse obstructive apneas also appears novel.

The purpose of this case report is to alert sleep physicians to other potential cases where ASV fails to capture. In such cases, we suggest manual intervention at an earlier time than 20 to 40 min of sleep, perhaps after 60 minutes without persistent sleep. Options for intervention include manually raising PSmin progressively, manually raising EEP above minimum, changing mask device, and providing sedative medication. Sedatives are routinely given at some sleep centers although our policy has generally been to prescribe when intolerance to positive airway pressure necessitates repeat trial of therapy.

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## DISCLOSURE STATEMENT

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