negative, and we empirically started antibiotic treatment with tazobactam/piperacillin. A chest X-ray performed on day 4after admission revealed slight improvement of the abscess (Fig. 1B), but hemoptysis developed 1 week later. Contrast-enhanced chest computed tomography (CT) revealed an enhanced nodule inside the abscess (Fig. 1C), suggesting PAP. The next day, because of continued hemoptysis and progressive respiratory failure, embolization of the pulmonary artery at the right A5 portion was performed with 2Interlock® detachable coils (Boston Scientific Corp), 4Hilal® embolization coils, and 1Tornado® embolization coil (Cook Medical). On day 2after embolization, contrast-enhanced chest CT revealed the disappearance of the enhanced nodule. On day 3after embolization, a chest X-ray revealed a marked decrease in the size of the abscess (Fig. 1D). One month after embolization, a chest X-ray showed almost complete resolution of the abscess (Fig. 1E). However, the patient suddenly died on day 39 after admission. The cause of death was unknown, because his family refused an autopsy.

The diameter of the lung abscess was greater than 6 cm. Therefore, surgical intervention, including chest tube drainage or surgical resection, in addition to antibiotics could have been chosen to treat this abscess.[1,8] Regarding surgical treatment, chest tube drainage might have been preferred because the patient's general condition did not allow surgical resection and the abscess was contiguous to the pleura. However, after admission, he complained of hemoptysis and was diagnosed with PAP, and embolization of the pulmonary artery showed remarkable improvement on chest imaging. This clinical course suggested a risk of bleeding, a life-threatening complication, with chest tube drainage. This report may have an impact on the choice of treatment for large lung abscesses.

The patient died suddenly, and because his family refused an autopsy the cause of death remains unknown. However, on the day of death, he had fever. Endovascular coil embolization is associated with a risk of developing infectious complications.[9] Therefore, it is possible that the patient died from infectious complications associated with the foreign endovascular material.

Following this case, we have reviewed the management of large pulmonary abscesses, whose standard treatment may not be applicable to patients of more advanced age and morbidity. The presence of a pseudoaneurysm must be taken into consideration in any interventional strategies in order to avoid complications.

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## Adaptive Servoventilation Device Software in the Assessment of Residual Respiratory Events in Patients with Central or Complex Apnoeas☆

## Software *de los dispositivos de servoventilación adaptativa para la evaluación de los episodios respiratorios residuales de pacientes con apneas centrales o complejas*

*To the Editor,*

The accuracy of auto-CPAP devices in determining residual apnea–hypopnea index (AHI) has been evaluated in several studies[1–7] but has not been confirmed in adaptive servo-ventilation (ASV) equipment. However, these data inform treatment and can significantly affect whether respiratory events are being treated optimally with the prescribed pressure setting.

We assessed the accuracy of respiratory event detection by ASV devices in 7 patients with central apneas/Cheyne Stokes respiration (CSA/CSR) and 9 with complex sleep apnea syndrome (CompSAS), diagnosed with respiratory polygraphy (RP) or conventional PSG. CSA was defined as an AHI >15 with predominant (>50%) central apneas according to AASM 2007 criteria.[8] CompSAS was defined as the appearance of central apneas (CAI of >15/h), during CPAP titration[9] in patients with obstructive sleep apnea at baseline, which persisted at follow-up using CPAP.

All patients were offered ASV treatment. The device brand depended on the supplier used by the Catalan Health Service, who provided treatment free of charge. The AutoSet CS, which did not allow for automatic expiratory pressure (EPAP) adjustment, was set at EPAP 6 cm $H_2O$, minimum pressure support (PS) 3 cm $H_2O$, maximum pressure (P max) 25 cm $H_2O$. The settings for the BiPAP autoSV Advanced were: EPAP min 4 cm $H_2O$, EPAP max 10 cm $H_2O$, PS min 0 cm $H_2O$ and PS max 25 cm $H_2O$.

A PSG with the patient's ASV device was performed 3 months later, and the AHI obtained from the device's software analysis (ASV-AHI) was compared to the AHI manually scored from PSG (PSG-AHI) over total sleep time (PSG-AHI-TST) and recording time (PSG-AHI-RT). Leaks obtained from the ASV smart card were recorded for analysis.

The agreement between PSG-AHI and ASV-AHI was studied with a Bland and Altman plot.[10] A Friedman correlation was used to assess any association between mask leakage and difference between PSG-AHI and ASV-AHI.

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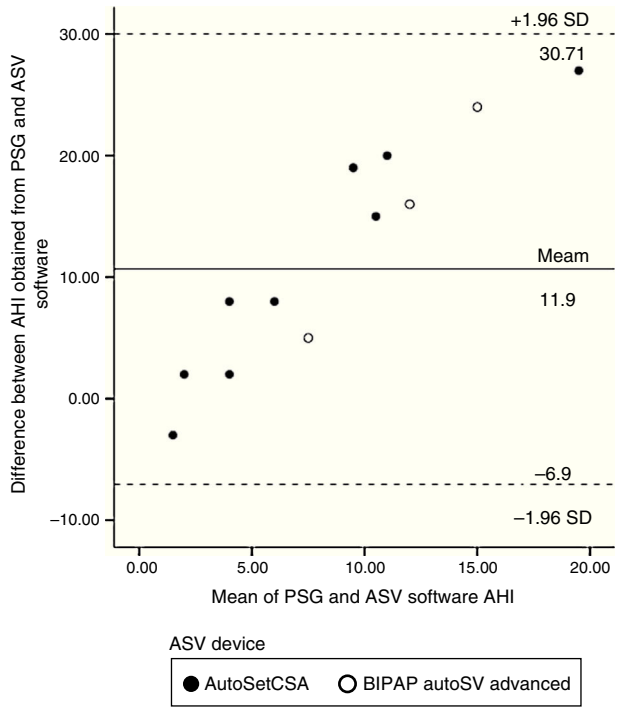


**Fig. 1.** Bland and Altman plot of the difference between AHI obtained from PSG and AHI obtained from ASV software, against the mean of PSG and ASV software AHI (mean±95% CI). PSG, polysomnography; AHI, apnea–hypopnea index; ASV, adaptive servo-ventilation.

Values were expressed as median (25th–75th percentile), or mean±SD.

Fifteen patients were men (93.7%), with no significant differences in baseline parameters between subjects with CSA or CompSAS in terms of age (67 [63–79] years vs. 73 [63–78] years), body mass index (28 [25–32] kg/m$^2$), Epworth score (4 [3–6] vs. 6 [4–13]) or AHI (50 [48–81] vs. 46 [41.5–60.5]).

Among the 16 patients, 2 declined ASV treatment. Comp SAS patients, who had been previously treated with CPAP for 284 days (34–902), were treated with a BiPAP autoSV Advanced ($n$=2) or AutoSet CS ($n$=6), and patients with CSA/CSR with a BiPAP autoSV Advanced ($n$=2) or AutoSet CS ($n$=4).

PSG at 3 months with the patient's device showed resolution of CA. In 2 patients, downloading from the ASV device was not possible (1 BiPAP autoSV Advanced, 1 AutoSet CS).

ASV-AHI in the 12 patients (3.3 [1.4–4.3]) was significantly lower than AHI-TST or AHI-RT (10.6 [4.8–20.2], $P$=.005 and 7.5 [3.8–15.7], $P$=.008, respectively). HI derived from the ASV device was also significantly lower than PSG HI-TST (2.8 [1.2–3.9] vs. 9.5 [3–20], $P$=.005) and PSG HI-RT (7.4 [2.5–15.5], $P$=.010).

Fig. 1 shows the Bland and Altman plot of the difference between PSG-AHI and ASV-AHI against the mean of both measurements, with a mean difference of 11.9±9.6 (95% limits of agreement −6.90, 30.71), which was greater because the residual AHI was higher.

The PSG-AHI was lower than 10 events/h in 6 of the 14 patients (42.8%) and lower than 15 in 8 of the 14 patients (57.1%), whereas according to ASV-AHI, all patients showed an AHI lower than 10.

No significant correlation was observed between mask leaks and the mean difference between PSG-AHI and smart card ASV-AHI ($r$=−0.423, $P$=.256) in patients using the AutoSet CS device. No high leaks were observed in patients using BiPAP autoSV Advanced.

The accuracy of internal software in estimating residual AHI has been addressed in a small number of studies with auto-CPAP devices[1–7] but, to our knowledge, there is no published information on ASV devices. While the ASV device adequately treated respiratory events in all patients according to the device software, treatment was sub-optimal in a substantial number of patients, according to PSG scoring. Thus, the ASV software underestimated the AHI, at the expense of underscoring hypopneas. Inter-observer variability in PSG manual scoring, especially in identifying hypopneas and the different criteria used to score hypopneas, have been suggested as possible explanations for the variance in results obtained by different studies analyzing auto-CPAP devices.[1,7] In our study, the same physician manually scored respiratory events on ASV treatment. Differences in hypopnea scoring criteria between human and machine detection of residual events are a more likely explanation for this finding. Manual hypopnea scoring criteria was a 50% airflow reduction followed by at least 3% desaturation and/or an arousal,[8] whereas the ASV device software does not take into account oxygen saturation or arousals from sleep when detecting respiratory events. Differences between PSG sleep time and recording time could suggest another explanation. Nonetheless, ASV-AHI was significantly lower than PSG-AHI whether calculated according to the sleep efficiency or based on recording time. Finally, the underscoring of residual events by the ASV software could also suggest the presence of excessive leaks, although we did not find any significant correlation between mask leaks during PSG as obtained by the ASV device and the difference between PSH-AHI and smart card ASV_AHI.

Although we studied 2 different populations of patients with CA and 2 different ASV devices, the baseline characteristics of both groups did not differ, and the effect of both ASV devices on CA was identified in both CSA and CompSAS subjects. However, due to the small number of patients using each device, we were unable to compare both devices in terms of analysis software accuracy.

In summary, device software-derived AHI significantly underestimated manually scored AHI at PSG, with a mean bias of 11, at the expense of hypopneas, and to a greater extent at a higher residual AHI. The clinical significance of residual AHI in these patients, many of whom suffer from heart failure, is still unknown. Further studies are needed to confirm these results and whether they are true of any ASV device.

## Author's Contribution

María Guadalupe Silveira: data analysis, manuscript writing Gabriel Sampol: study design, manuscript review Roser Cambrodi: data collection Àlex Ferre: data collection Patricia Lloberes: study design, manuscript writing, supervision.

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## Hoarseness for Two Years: Did It Start in the Lung? A Case Report☆

### *Caso clínico de ronquera de dos años de duración: ¿comenzó en el pulmón?*

*To the Editor,*

Laryngeal tuberculosis (TB) is the most common manifestation of ear–nose–throat tuberculosis[1] and the most common granulomatous disease of the larynx.[2] However, it is an uncommon site of TB, and represents about 1% of all cases of extra pulmonary TB.[2,3]

In the beginning of the last century, about half of all patients with advanced pulmonary TB presented concomitant laryngeal TB due to direct spread of the infection from the lung.[4]

Currently, due to earlier and more appropriate treatment of pulmonary TB, the incidence of laryngeal TB has steadily decreased in developed countries. However, it still is on the rise in developing countries, in association with the Acquired Immune Deficiency Syndrome (AIDS) epidemic, low socioeconomic conditions and lack of adequate TB chemotherapy, among other factors.[5–7]

We report the case of a 57-year-old man, smoker of 30 pack-years, presenting with a two-year history of hoarseness, weight loss of 20 kg, with no anorexia. He had no dysphagia, odynophagia, cough, fever or night sweats. The patient worked as a carpenter, and had had a history of heavy alcohol consumption until ten years previously. He reported a history of recurrent otitis and had undergone bilateral ear surgery about 10 years previously. He had facial palsy of unknown cause since he was13 years old, and had no personal history of TB or contact with patients with TB. Physical examination, apart from left hemifacial palsy, was unremarkable.

The patient was evaluated by the otorhinolaryngologist and underwent laryngoscopy (Fig. 1) that revealed mucosal thickening, irregularities and leukoplasia lesions of the left side of the epiglottis and of the left vestibular fold. Vocal cords were normal. Laryngeal CT showed no lymphadenopathies or other abnormalities.

The patient then underwent a suspension microlaryngoscopy during which the epiglottic lesion was biopsied and sent for histological and mycobacterial direct stain and culture study. Anatomopathological study showed no signs of malignancy or granulomas. Mycobacterial cultural exam was positive for *Mycobacterium Tuberculosis Complex*, susceptible to all first line anti-TB drugs.

The patient was then referred to the tuberculosis outpatient clinic. Occupational health services were immediately activated in order to initiate screening of exposed health professionals. HIV infection was excluded and a chest X-ray (CXR) was performed, showing a cavitary lesion at the left lung apex and an infiltrate on the left lower lobe. Sputum samples were collected and anti-TB medication with isoniazid, rifampin, pirazinamid and ethambutol was started. Mycobacterium direct test of sputum was negative and culture was positive for *Mycobacterium tuberculosis complex*.

The patient's symptoms improved and videolaringoscopy performed after 6 months of treatment showed resolution of the epiglotic lesions.

In the last three decades, a change in the pathophysiology of laryngeal TB has been observed. Although concomitant pulmonary involvement is common, more cases of primary laryngeal TB, without pulmonary involvement, have been described.[6,8–11]

Clinical presentation has also changed. Ling et al.[8] compared a group of patients diagnosed with laryngeal tuberculosis before 1990 and another after 1998 and found that odynophagia was the most frequent symptom among the former group and hoarseness among the latter. Moreover, in the group of patients from before 1990, constitutional symptoms were more frequently reported.

We believe that, in our case, epiglottic TB originated from dissemination of lung infection. This was subsequently demonstrated by the presence of cavitary lung disease and positive mycobacterial culture of the sputum. However, the patient had no pulmonary symptoms suggestive of TB, so lung infection was not

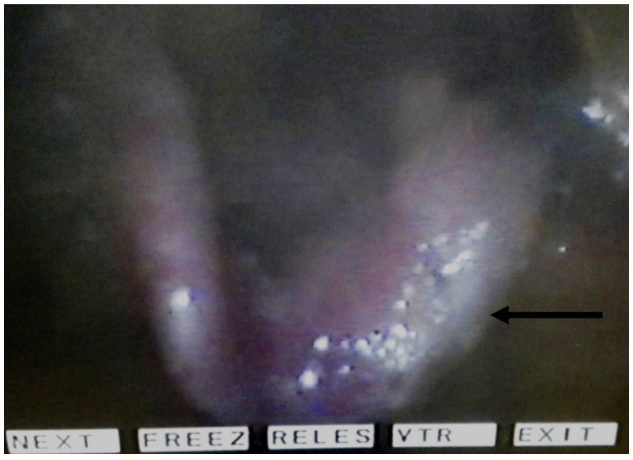


**Fig. 1.** Videolaryngoscopy prior to treatment of TB.

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