European Respiratory Society and European Sleep Research Society statement on the treatment of central sleep apnoea with adaptive servo-ventilation

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Shareable abstract (@ERSpublications)
Adaptive servo-ventilation has no negative cardiovascular impact, but positive effects on patient-reported outcomes in optimally treated heart failure with preserved and reduced (30–45%) ejection fraction https://bit.ly/4kNCm70

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Abstract

Adaptive servo-ventilation (ASV) has been considered effective in controlling various forms of central sleep apnoea (CSA) and also any additional obstructive sleep apnoea (OSA) component. However, after the publication of the SERVE-HF study, its use was restricted in patients with systolic heart failure (HF) and prevalent CSA, and was withheld from many patients with symptomatic CSA. In the meantime, the devices have been further developed and the algorithms adapted, and there is new evidence from randomised controlled trials and observational studies that makes it necessary to re-evaluate some societies' statements on the use of ASV, especially in patients with HF and CSA and with the current ASV devices. This short statement is based on a review of the effect of ASV on hard cardiovascular end-points, echocardiographic parameters and exercise capacity as well as on sleep architecture and sleep quality, symptoms and quality of life (QoL) in patients with congestive HF. The expert group concludes that ASV has positive effects on CSA and QoL in various forms of CSA, that current ASV devices have no negative effect on hard cardiovascular end-points, and that ASV has positive effects on patient-reported outcomes. Moreover, it is used by Task Force members after optimal treatment of the underlying disease and after an unsuccessful continuous positive airway pressure trial in patients with HF with preserved ejection fraction, but also in patients with left ventricular ejection fraction 30–45%. In the latter group, however, initiation is performed in expert centres only. In severe systolic HF, ASV is sometimes evaluated in a palliative therapy concept for severely symptomatic patients with CSA.

Introduction

The presence of heart failure (HF) poses a significant public health burden [1]. Up to 60% of patients with HF experience sleep disordered breathing (SDB), including obstructive sleep apnoea (OSA) and central

sleep apnoea (CSA), coexisting OSA and CSA, and Cheyne–Stokes respiration (CSR) [2–6]. Adaptive servo-ventilation (ASV) has proven to be the most effective treatment for suppressing CSA and CSR [7–9]. It is superior to medical therapy of underlying diseases alone, specific pharmaceutical therapies (*e.g.* acetazolamide), non-invasive ventilation, neural stimulation therapy and nocturnal oxygen therapy [7–11]. Nevertheless, ASV indications and usage in patients with predominant CSA and left ventricular ejection fraction (LVEF) ⩽45% have been restricted since the publication of the SERVE-HF (Treatment of Sleep-Disordered Breathing with Predominant Central Sleep Apnea by Adaptive Servo-Ventilation in Patients with Heart Failure) study in 2015 [12–16]. The restrictive statements or information of manufacturers, scientific societies and health administrations [13–15, 17] were valid only for this clearly defined group representing <10% of ASV indications in sleep clinics [18–20]. ASV therapy has not only proven to be effective in controlling CSA/CSR in HF, but is also an established and effective treatment modality for other forms of CSA, particularly treatment-emergent CSA and opiate/opioid-induced CSA, as well as post-stroke CSA or idiopathic CSA [21, 22]. In these patients, ASV could continue to be used in symptomatic patients without restriction after SERVE-HF if continuous positive airway pressure (CPAP) is not working.

Numerous scientific publications have thoroughly examined the strengths and weaknesses of the SERVE-HF study, regarding device algorithms and settings, adherence, and the populations investigated [23–26]. Moreover, since the publication of SERVE-HF further research priorities were defined [27, 28], and several prospective cohorts, randomised controlled trials (RCTs) and real-life data in HF have been made available in the field [22, 29–44]. Most recently, the ADVENT-HF (Adaptive Servo-Ventilation for Sleep-Disordered Breathing in Patients with Heart Failure with Reduced Ejection Fraction) trial, another multinational, multicentre, prospective RCT on ASV in HF with reduced ejection fraction (HFrEF), was published [28].

Data from RCTs are the mainstay for therapeutic algorithms. However, conclusions derived from long-term RCTs are limited due to strict patient selection and the exclusion of highly symptomatic patients for ethical reasons. Therefore, data from well-performed large cohorts and registries provide indispensable and complementary contributions to the scientific evidence [45]. There are many new data from recent studies, including ADVENT-HF, on cardiovascular outcomes, mortality and hospitalisation rates in these patients [33, 35, 38, 44, 46, 47] which question the concerns of harm. Specific phenotypes of good responders to ASV have been consistently highlighted [4, 5, 33, 46, 47]. In addition, HF phenotypes with reduced ejection fraction (HFrEF), mildly reduced ejection fraction (HFmrEF) and preserved ejection fraction (HFpEF) differ in prevalence of CSA and outcomes under ASV treatment [4, 5, 33, 46, 47].

This novel evidence requires re-consideration of the previous statements on ASV in CSA. While it is obvious that any treatment may only be used if it does no harm (*primum non nocere*), decisions on treatment should consider both major outcome parameters and patient-reported outcome measures (PROMs).

A re-consideration is also of crucial relevance because of major changes in technologies. The early studies, including SERVE-HF, used devices that applied a minimum pressure support of 3 mbar and a fixed expiratory positive airway pressure (EPAP). These devices have not been used for almost a decade and subsequent studies in patients with HF and sleep apnoea were performed using algorithms with variable EPAP and without minimal pressure support. It is unclear whether these or other differences are causative for any new findings.

Methods

The European Respiratory Society (ERS) and European Sleep Research Society (ESRS) established a group of European experts in the fields of respiratory sleep medicine and cardiology focusing on the evaluation and interpretation of available studies with a special focus on the currently available ASV algorithms. The panel reviewed the evidence for benefits and potential harms of ASV with respect to major non-cardiac and cardiac events, cardiac surrogate parameters, quality of life (QoL), and patient-reported outcomes in patients with CSA with and without HF. It agreed on the following PICO (Population, Intervention, Comparison, Outcome) question:

> *Is there evidence of improvement or harm regarding major cardiovascular outcomes and/or breathing disturbances, sleep quality, patient-reported or functional outcomes (outcome) in adult patients with CSA (including CSR) (population) under currently available ASV devices (intervention) compared with optimal treatment of the underlying disease or CPAP (comparison)?*

Group members carried out a literature search in MEDLINE using the following search strategy: (((central sleep[Title/Abstract] OR CSA[Title/Abstract] OR central breat*[Title/Abstract] OR Cheyne[Title/Abstract])) OR ((“Sleep Apnea, Central”[Mesh]) OR “Cheyne-Stokes Respiration”[Mesh])) AND ((“adaptive serv*”[Title/Abstract] OR ASV[Title/Abstract] OR servo-vent*[Title/Abstract] OR servovent*[Title/Abstract])).

RCTs, meta-analyses, cohort and observational studies on patients older than 18 years published in English language between 1995 (invention of ASV) and October 2024 were considered for further evaluation. Case reports, reviews, studies with less than 10 patients and studies without description of ASV type were excluded. After abstract and full-text screening, the Task Force members additionally checked the references of the included studies to identify if there were additional studies that should be included. The results of the search are presented in the PRISMA (Preferred Reporting Items for Systematic reviews and Meta-Analyses) flow diagram in figure 1.

Abstract and full-text screening to identify eligible articles on the effect of ASV on one of the outcomes defined in the PICO was performed by the panel, and two authors focused on a specific group of outcomes to extract data and summarise key findings.

These key findings, methodologies and conclusions from the relevant studies were extracted and a summary shared with the expert panel for discussion. The statements were developed in a multistage process that included literature review, data synthesis and expert discussion of the summarised evidence between the panel members. To reach final agreement on the statements and a shared conclusion, the expert panel convened for a final virtual meeting during which the final statements were drafted. Panellists were asked to evaluate the draft and provide final feedback on the accuracy, clarity and relevance of each statement to ensure that the final statements reflect the collective evaluation and interpretation of the expert panel. A patient representative who uses ASV was part of the process from the start and the assignment was agreed with the European Lung Foundation.

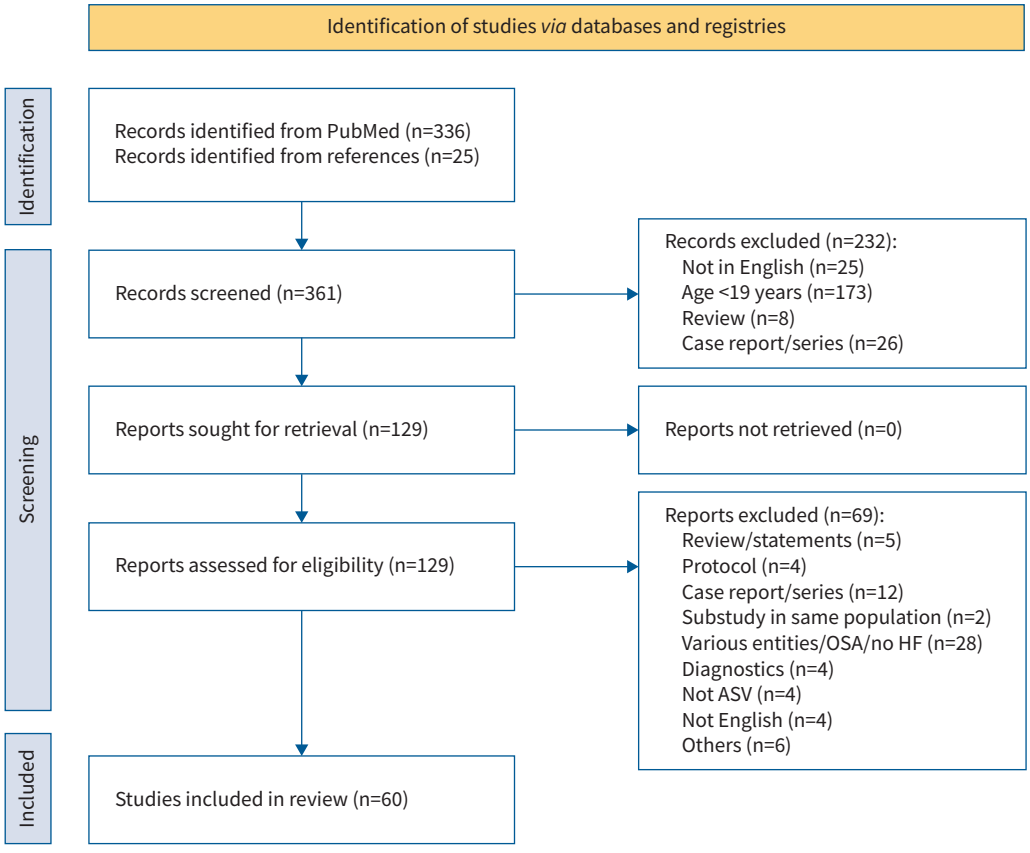

FIGURE 1 PRISMA flow diagram. OSA: obstructive sleep apnoea; HF: heart failure; ASV: adaptive servo-ventilation.

Description of the evidence

Does ASV influence major adverse cardiac events?

Five RCTs and an important post-hoc analysis [12, 24, 33, 35, 48, 49], 13 observational studies [37, 38, 44, 46, 50–58] and four meta-analyses [47, 59–61] on the effect of ASV in HF on major adverse cardiac events (MACEs) were included (supplementary table S1). One study included only HFpEF [57], the two analyses of the FACE study [46, 58] included both HFrEF and HFpEF, and all other studies included only patients with reduced systolic function (HFrEF).

Based on the evidence from RCTs and prospective observational studies following the publication of the SERVE-HF trial [12], ASV has no effect on the occurrence of MACEs in a heterogeneous unselected group of patients with HF and SDB [33, 35, 37, 46]. After SERVE-HF, all studies were either neutral or even showed a benefit with regard to mortality and MACE risk. There was no evidence or signal of increased risk of mortality or MACEs in either observational studies or RCTs, although the findings of ADVENT-HF were underpowered for final conclusions to assess mortality [35]. A meta-analysis of eight studies (more than 2000 patients, three RCTs), also including trials from before 2015, found no significant effect of ASV on mortality, but a reduction in MACEs under ASV compared to usual care at LVEF >33% [47]. A Cochrane review based on RCTs only including patients with HFrEF found no significant effect of ASV on mortality [59]. Based on the RCT data, the panel concludes that there is no effect of ASV on MACEs or mortality, not only on the basis of more recent studies with current devices, but also considering all pooled evidence from RCTs.

Observational studies indicate improved event-free survival (death, cardiovascular death and hospitalisation for worsening HF) under ASV in the subgroups of patients with more pronounced nocturnal hypoxaemia, with longer nightly ASV usage, with HFpEF and with predominant OSA [38, 46]. However, despite efforts to correct for MACE-relevant confounders, the observational studies may not have considered all potential confounders. RCTs in the population with HFpEF are missing.

Based on pooled data from RCTs, there is no evidence that ASV increases mortality or the risk of MACEs, neither in patients with HFrEF nor in HFpEF, and particularly not with the current ASV settings

What are the effects of ASV treatment on other outcome measures, especially cardiac surrogate parameters, or exercise tolerance?

Five RCTs including one secondary analysis [32, 34, 62–64], 10 observational studies [41, 56, 65–72] and two meta-analyses [9, 73] on the effect of ASV on exercise, echocardiographic parameters and N-terminal pro-brain natriuretic peptide (NT-proBNP) were evaluated (supplementary table S2).

In addition to mortality and major cardiac events, effects on cardiac surrogate parameters may influence therapeutic decisions. These include arrhythmias and echocardiographic measures, as well as functional findings such as exercise tolerance.

Starting with short-term trials of 1 h, ASV showed favourable haemodynamic effects of ASV in HF patients with CSR as revealed by increased stroke volume indices compared to healthy volunteers [72]. ASV was compared to other SDB treatments such as oxygen therapy, bilevel positive airway pressure (BiPAP) and CPAP. Compared to oxygen therapy, in a “single night” RCT, ASV reduced cardiac overload and attenuated sympathetic nervous activity and ongoing myocardial damage effectively in 42 HFrEF patients with SDB [64]. ASV was superior to oxygen treatment in ameliorating brain and atrial natriuretic peptide levels. An RCT including 37 HFrEF patients with New York Heart Association Functional Class (NYHA-FC) II and III compared BiPAP *versus* ASV over 6 weeks. Besides better evidence of improvement in LVEF in BiPAP users, there were no differences in terms of blood pressure and echocardiographic parameters such as end-diastolic diameter and shortening fraction between the ventilation modalities [62]. In contrast, ASV was superior to CPAP after 3 months of follow-up in terms of echocardiographic parameters and 6-min walk test (6MWT). The ASV mode showed significant reductions in plasma NT-proBNP and an increase in 6MWT distance [63, 68]. Left ventricular end-systolic diameter and LVEF increased, while mitral regurgitation area decreased in the ASV mode group compared to the CPAP mode group [63]. ASV significantly reduced urinary norepinephrine excretion and increased 6MWT distance, whereas CPAP did not [68].

Compared to cardiovascular standard care alone, ASV ameliorated LVEF and NYHA-FC in prospective studies on CSR-CSA [56, 65, 70]. In cardiopulmonary exercise testing, workload increased by 5.4 W and peak oxygen uptake ($\dot{V}_{O_2}$) improved compared to patients without ASV [65, 70]. Moreover, in association with suppression of CSA in HF patients, ASV also improved muscle sympathetic nerve activity and LVEF

after 3 months [67]. While ASV and standard care both reversed left ventricular remodelling, only ASV treatment was able to reduce left atrial volume [34]. Toyama *et al.* [32] also showed ameliorations in cardiac sympathetic nerve activity, cardiac function, exercise capacity and symptoms in HF patients with CSR after 6 months of therapy. A study by Oldenburg *et al.* [71] demonstrated that ASV increased the workload during cardiopulmonary exercise testing and $\dot{V}_{O_2}$ at the anaerobic threshold and predicted peak $\dot{V}_{O_2}$. LVEF and NT-proBNP levels also improved in response to ASV treatment.

Regarding arrhythmias, Nagasaka *et al.* [69] retrospectively analysed 141 consecutive hospitalised patients with HF due to ischaemic heart disease. 75 patients started ASV during the hospitalisation and were compared to 66 controls with a follow-up period of 1 year. At the 1-year follow-up, the ASV group presented fewer arrhythmia events, including paroxysmal atrial fibrillation or ventricular tachycardia, compared to the non-ASV group, and showed improvement in estimated glomerular filtration rate after adjusting for demographic and cardiovascular disease risk factors [69]. Piccini *et al.* [41] confirmed the role of ASV in reducing atrial fibrillation burden in 35 HF patients compared to standard care.

A follow-up of almost 1 year showed that ASV therapy *versus* no treatment of CSA in HFpEF patients led to a significant increase in peak $\dot{V}_{O_2}$, predicted peak $\dot{V}_{O_2}$, anaerobic threshold, oxygen pulse and minute ventilation to carbon dioxide output ratio ($\dot{V}_E/\dot{V}_{CO_2}$). ASV also improved echocardiographic parameters of left ventricular function (*e.g.* peak early/atrial Doppler mitral inflow velocity (*E/A*), *A*, early diastolic lengthening velocity (*e′*) and *E/e′*) [66, 73].

Two meta-analyses studied the effects of ASV on cardiac function and exercise parameters. Sharma *et al.* [9] found that the weighted mean difference in apnoea–hypopnoea index (AHI) and LVEF both significantly favoured ASV. In addition, ASV improved 6MWT distance, but had no influence on peak $\dot{V}_{O_2}$, $\dot{V}_E/\dot{V}_{CO_2}$ slope or QoL. These results were confirmed by Wu *et al.* [73] in their meta-analysis on seven studies involving 301 patients. The weighted mean difference in AHI, LVEF, urinary noradrenaline and exercise capacity measured by 6MWT distance improved under ASV compared to control. There were no differences in left ventricular end-diastolic diameter and Epworth Sleepiness Scale (ESS) score [73]. Overall, ASV was more effective than control conditions in improving SDB, cardiac function and exercise capacity [74].

Does ASV influence sleep parameters?

Three RCTs [39, 63, 75], two clinical trials [76, 77], one comparative study [31] and one research support study [78] were included (supplementary table S3).

Data on changes of sleep macro-structure (sleep stages) and micro-structure (arousals) under ASV are scarce and controversial. This may be due to substantial methodological differences related to study design, heterogeneity of populations included (*e.g.* HFpEF, HFrEF and treatment-emergent CSA), different pathophysiological mechanisms, comorbidities, sleep pattern at baseline or follow-up period.

A pre-planned substudy of SERVE-HF showed that the respiratory arousal index (RAI) was significantly lower and the number of periodic leg movements during sleep (PLMS) and PLMS-related arousal index were significantly higher under ASV [39]. Compared to control, ASV showed some improvement in sleep efficiency, sleep latency and RAI, mainly after 12 months of treatment, but differences were not considered clinically relevant. Moreover, the PLMS index, a predictor of all-cause and cardiac mortality in HF patients, increased markedly in the first 3 months after initiation of ASV and was significantly higher in the ASV *versus* control group at 12 months [39]. Similarly, Xie *et al.* [78] and Bradley *et al.* [35] observed an increased PLMS index and PLMS arousal index in HF patients under ASV.

However, early studies showed that patients with chronic HF with CSA had a significant improvement in sleep parameters after switching from CPAP treatment to ASV [63, 79]. The same results came from a study of Teschler *et al.* [75] showing that sleep quality improved significantly under ASV compared to other treatment modalities in patients with HF and CSA/CSR. Roder *et al.* [77] performed a retrospective polysomnographic analysis of 30 ASV-treated patients with stable HFrEF and moderate-to-severe CSA. The results showed a reduction in non-rapid eye movement (NREM) N1 and N3 sleep stages and an increase of N2 and REM sleep. The authors discussed that the changes in sleep structure could be explained by the autonomic imbalance with higher sympathetic tone in REM sleep and reduced vagal predominance in N3 sleep under ASV. In addition, ASV was associated with a significant reduction in respiratory-related arousals without differences in the PLMS arousal index. However, the study was limited by its retrospective design and the small sample size, and several comorbidities may have influenced the

results [77]. Most recently, the ADVENT-HF trial showed that ASV significantly improved the total number of arousals and the number of respiratory arousals, N3 and REM sleep duration [35].

The results of Heider *et al*. [80] in a cohort of 114 HFpEF patients are in line with these improvements in sleep micro- and macro-structure. They retrospectively showed an increase in both N3 and REM sleep during ASV treatment. A marked increase in N3 with an increased alertness level, as quantified by the Maintenance of Wakefulness Test, has also been reported in a recent paper by Karimi *et al*. [76].

All these results highlight the need for a better understanding of the origin of sleep impairment in HF patients with CSA. The diagnosis of impaired sleep at baseline and under treatment, including arousals, PLMS and disturbed sleep, can play a pivotal role in better defining treatment indication and selecting therapeutic options.

What are the effects of ASV treatment in patient-reported outcomes?

16 RCTs [8, 12, 32, 33, 35, 62, 63, 68, 74, 81–87] and 10 observational studies (six on HFrEF and four on HFpEF) [22, 25, 37, 46, 50, 76, 88–91] on the effect of ASV in HF on QoL and symptoms were identified.

The major long-term RCTs on ASV had no or modest effects on symptoms and QoL [12, 35]. Short-term studies in patients with symptomatic CSA ASV exhibited greater effects on sleepiness and/or measure of QoL [33, 85], while RCTs of ASV in non-sleepy patients showed no significant effects on sleepiness and QoL [81].

In SERVE-HF, ASV did not improve general (assessed by the EuroQol Group 5-Dimension Self-Report Questionnaire (EQ-5D)) or HF-specific measures of QoL (Minnesota Living with Heart Failure Questionnaire (MLHFQ)), or NYHA-FC [12]. However, ASV showed a modest improvement in ESS scores compared to control.

The ADVENT-HF study demonstrated improved sleepiness and HF-specific QoL (MLHFQ) under ASV [35]. While the improvements were modest, these persisted over the course of the 5-year trial, and were associated with improvements in NYHA-FC and objective sleep structure [35, 92].

In a small crossover study including sleepy HFrEF patients with CSA, ASV significantly improved objective daytime sleepiness (Osler Maintenance of Wakefulness Test) [85].

Following the SERVE-HF study, the randomised controlled CAT-HF (Cardiovascular Improvements with Minute Ventilation ASV Therapy in Heart Failure) trial enrolled hospitalised patients with HFrEF, HFmrEF and HFpEF, and used an ASV device for treatment of CSA and OSA [33]. It revealed no significant benefits of ASV on functional capacity, as indicated by 6MWT distance, NYHA-FC, ESS and general QoL measures (Duke Activity Status Index, EQ-5D, Patient Health Questionnaire-9 and Pittsburgh Sleep Quality Index). However, patients treated with the ASV device had numerical improvements in HF-specific QoL (Kansas City Cardiomyopathy Questionnaire) compared to the control group.

Smaller RCTs investigating the effect of ASV on cardiac symptoms, such as angina pectoris [74], dyspnoea and HF-related QoL, did not always show consistent results [8, 32, 33, 62, 68, 81, 82, 84–87] (supplementary table S4).

RCTs are complemented by observational studies [22, 25, 37, 46, 50, 76, 88–91] with more representative study populations. Two of those were large multicentre observational studies [22, 46]. Recent findings from the READ-ASV (Treatment of Central and Complex Sleep-Disordered Breathing with Adaptive Servo-Ventilation) registry [5, 22], including consecutive ASV users (sleep clinic population, 23% with HF), indicated that a 12-month treatment with ASV for CSA, with or without coexisting OSA, led to significant improvements in disease-specific QoL (Functional Outcomes Sleep Questionnaire) and reduced daytime sleepiness (ESS). These improvements were particularly notable in individuals who had symptoms of SDB prior to the initiation of therapy (62% of the population) [5, 22], whereas the results of the observational FACE study, which included non-sleepy HF patients, indicated that 2 years of ASV therapy did not result in any clinical improvement in MLHFQ and ESS scores in patients with chronic HFrEF, HFmrEF or HFpEF and SDB [46].

Most recently published data (after the current statement was finalised) from the French registry confirm these findings in a huge variety of CSA entities [93].

Statements

Based on the results of the literature search and a discussion and consensus process, the panel agreed on the following statements. These statements reflect the reassessed practice of the experts and are not intended as a general recommendation (figure 2).

1) ASV has clear positive effects in improving breathing disturbances and QoL in CSA/periodic breathing in HF, treatment-emergent CSA, drug-induced CSA, CSA in neurological or other medical disorders and idiopathic CSA. Improvements in sleep parameters, exercise performance and cardiac surrogate parameters are less consistent. Recent data on the use of ASV devices with the currently available techniques do not report any increase in mortality or MACEs.
2) In patients with CSA/periodic breathing due to a cause other than HF, there is no evidence for a negative impact of ASV on major outcome parameters. The members of the panel treat symptomatic patients with ASV if treatment of any underlying disease and a CPAP trial have failed. This includes patients with idiopathic CSA, drug-induced CSA, treatment-emergent CSA and CSA in neurological diseases or non-cardiac medical disorders.
3) There is growing evidence of positive effects of ASV not only on polysomnographic and functional parameters, but also on PROMs. There is evidence from observational studies that ASV has a positive effect on MACEs in subgroups or specific phenotypes (longer nightly ASV usage; HFpEF; pre-dominant OSA) of HF patients. In their clinical practice, the members of the panel decide on treatment based on a comprehensive patient evaluation including functional parameters (sleep quality; exercise tolerance) and patient-reported outcomes after optimisation of any underlying disorder or condition. As an initial step, they give preference to CPAP due to availability, feasibility and cost-effectiveness.
4) In HF patients with predominant OSA, there is no evidence of a negative impact of ASV on major outcome parameters. The members of the panel treat symptomatic patients with ASV if treatment of any underlying disease and a CPAP trial have failed. In HF patients with an LVEF ⩽45% and predominant OSA, treatment is initiated in expert centres.
5) In HF patients with predominant CSA and an LVEF between 30% and 45%, there is no evidence for a negative impact of ASV with current devices and settings on major outcome parameters. The members of the panel treat symptomatic patients with ASV if treatment of any underlying disease and a CPAP trial have failed and initiate treatment in expert centres.

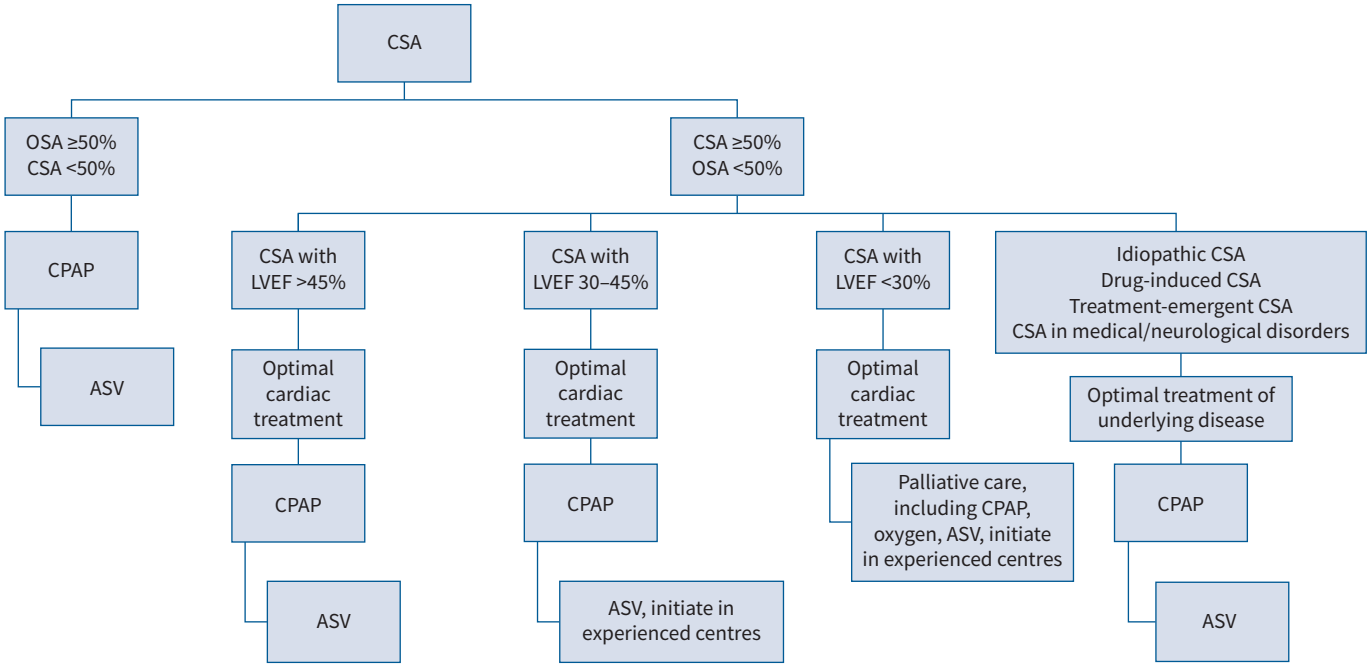

FIGURE 2 The current practice of how the members of the Task Force treat patients with central sleep apnoea (CSA). The figure is not intended as a general recommendation. OSA: obstructive sleep apnoea; LVEF: left ventricular ejection fraction; ASV: adaptive servo-ventilation; CPAP: continuous positive airway pressure.

6) HF patients with predominant CSA with an LVEF <30% have a very high mortality in general (comparable to advanced lung cancer). In a palliative care setting, despite lacking scientific evidence, the members of the panel sometimes use ASV as a palliative care option in highly symptomatic patients if there is no other option to reduce symptom burden, and initiate treatment in expert centres.

Discussion

The panel reviewed the available literature and reassessed its current practice in the use of ASV. The resulting approach is described in the differentiated statements on various entities of CSA. The main focus is on CSA in HF patients, while there are no changes in our current practice for other entities. This statement considers data on efficacy in controlling CSA and improving its symptoms and markers of cardiac function, as well as safety and hard cardiovascular end-points. Data from prospective observational studies are discussed in addition to evidence from RCTs. RCTs focus on selected populations in RCTs and do not adequately represent different subgroups of patients with HF and sleep apnoea, which *per se* have different prognoses and require separate assessment (see specific statements), as well as groups that are under-represented in RCTs, such as HFpEF and sleepy patients.

In summary, the current evidence from RCTs and prospective observational studies suggests that ASV is effective in controlling various forms of CSA and is superior to CPAP in stabilising breathing and suppressing central events, particularly in CSA/CSR, treatment-emergent CSA and opiate-induced CSA. Although ASV generally improves sleep quality and other patient-reported outcomes, and although today's common ASV algorithms and settings are ideally adapted to the pathophysiology of CSA with periodic breathing and can optimally control CSA, its use has been limited in the last 10 years, at least in HFrEF, due to safety concerns. This is now being reconsidered on the basis of new evidence. The previous practice was mainly driven by the SERVE-HF study published a decade ago, which has been challenged by more recent evidence, including the ADVENT-HF study. Both were prospective, multinational, parallel group RCTs in patients with symptomatic HFrEF (LVEF ⩽45%, AHI >15 events·h^{-1}). SERVE-HF randomised 1325 patients with predominant central apnoeas [12]. 1161 patients completed the study and were treated with optimal cardiac therapy alone or additional ASV. According to the current technical standard at the time of the study initiation, the ASV device applied a minimal pressure support of 3 mbar and a fixed expiratory pressure (EPAP). The study documented 232 MACEs in 666 ASV group patients and 193 in 659 controls. While there was no difference in the primary combined end-point (neutral results), all-cause mortality and cardiovascular mortality were significantly higher under ASV. A multistate modelling analysis showed that the mortality risk was increased in patients with LVEF ⩽30% and in those without previous hospital admission [24].

ADVENT-HF included not only HF patients with predominant CSA, but also non-sleepy HF patients with predominant OSA [28]. The ADVENT-HF population included less patients in NYHA-FC III and IV, but the mean LVEF was similarly and severely reduced in both studies. All sleep studies of ADVENT-HF were evaluated in a core laboratory to avoid misdiagnoses and heterogeneity of scoring. As the study was discontinued prematurely due to the Philips device recall and the COVID-19 pandemic, with 731 subjects having been randomised, it was statistically underpowered, in particular to definitely address safety issues. There was no significant difference in the predefined end-points, although the hazard ratio (HR) for mortality was in favour of ASV, at the level as pre-calculated (HR 0.78). In contrast to SERVE-HF, ADVENT-HF did not provide any evidence that ASV may cause harm, but the early termination did not allow a robust safety analysis. ASV improved the ESS score and sleep parameters in comparison to standard care [35]. The reason for the increased mortality in SERVE-HF is not clear. It is possible that differences in the study populations between SERVE-HF and ADVENT-HF may have influenced the different findings. As mentioned earlier, the ASV algorithm and settings used in SERVE-HF differed from the more recent studies, including ADVENT-HF. While the historical algorithms applied unavoidable pressure support and invariable EPAP, the current technology allows for zero pressure support during hyperventilation and varies the EPAP allowing for minimal pressure application. However, the relevance of this technical difference is unclear.

In addition, we have learned from prospective observational studies with a broader, less selected patient population that phenotyping is crucial for long-term outcome independent of sleep apnoea therapy and that there are indeed phenotypes of HF patients with sleep apnoea that may even benefit in terms of MACEs [46]. However, these subgroups have not been included in the large RCTs and there are no RCTs specifically in HFpEF. Adherence to ASV, which is different in different subgroups, must also be taken into account for the treatment effect and is often higher in observational studies than in RCTs. Thus, a differentiated view of different groups with CSA is indicated, as reflected in the statements.

Recognising the potential risks of any intervention in certain subgroups, particularly those with severe systolic HF and poor autonomic regulation, the panel uses ASV, like any other treatment for SDB in potentially vulnerable subgroups, only in experienced centres with good monitoring (*e.g.* attended in-laboratory sleep study). This includes the need to consider both long-term outcome measures and PROMs when making treatment decisions, as they contribute significantly to the assessment of treatment efficacy.

Previous studies on ASV were not designed to study the effects or true effect size on PROMs, since the vast majority of patients are asymptomatic or oligosymptomatic [12, 35]. Sleepiness and impaired sleep apnoea-related QoL have been identified as important predictors for clinically relevant effects of ASV on sleepiness and disease-specific QoL [22]. Although the available studies did not consistently show improvements in PROMs, the potential positive effects on symptoms and QoL are promising, and symptoms and treatment goals should be considered in relation to PROMs. It is therefore important for the future that cardiologists treating patients with HF refer patients with symptomatic sleep apnoea to experienced sleep centres for an assessment of the type of sleep apnoea and phenotyping, and for a treatment recommendation that considers all the listed outcomes.

Conclusions

The available evidence suggests that ASV improves breathing disturbances and QoL in the various entities of CSA without any increase in mortality or MACEs. It may have a positive effect on MACEs in subgroups or specific phenotypes of HF patients. Therefore, if treatment of any underlying disease and a CPAP trial have failed, the members of the panel use ASV for the treatment of symptomatic patients with idiopathic CSA, drug-induced CSA, treatment-emergent CSA, CSA in neurological diseases, CSA in non-cardiac medical disorders and in HF patients with predominant OSA. In HF with predominant CSA with LVEF $\geqslant$30%, the panel initiates ASV treatment in expert centres. Close cooperation between the cardiologists treating patients with HF and the respiratory sleep specialists as ASV experts is of crucial importance. The literature appraisal performed for this statement suggests the need and feasibility of formal evidence syntheses to support a clinical practice guideline on the topic.

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