We now have a wide variety of non-pharmacological therapies that aim to either relieve symptoms or improve the prognosis of severe heart failure patients. Nocturnal oxygen therapy and positive airway pressure are included in this category. Nocturnal oxygen therapy was formally approved by the Japanese Ministry of Health and Labor in 2004 as a treatment for heart failure with central sleep apnea after rigorous studies in which symptomatic and functional improvement was confirmed. However, as nocturnal home oxygen therapy could not prove a prognostic improvement or a decrease in the hospital admission rate, this therapy has been used more as a last resort for patients in the last stage of heart failure after all the other possible therapies have been utilized. Non-invasive positive pressure ventilation (NIPPV), including continuous positive airway pressure (CPAP) or other types of pressure support, such as bi-level PAP or adaptive servo-ventilation (ASV), has been increasingly utilized as a first-line therapy for acute severe congestive heart failure and has been recognized as a powerful tool for treating chronic severe heart failure in recent years. The mechanisms by which NIPPV therapy exerts favorably in heart failure patients are considered to be as follows. Firstly, the positive pressure that NIPPV machine produces in the thorax is considered itself to have several favorable mechanical effects, which include a decrease in preload by blocking venous return leading to the immediate alleviation of pulmonary congestion, a decrease in the left ventricular afterload by pressurizing the left ventricle from outside, a decrease in breathing work and even a decrease in mitral regurgitation. Secondly, NIPPV, especially that with pressure support, prevents central sleep apnea or unstable breathing, which is believed to have an adverse effect on patients with heart failure, and this normalization of breathing might help to reduce the enhanced sympathetic nerve activity in such patients.

Cumulative experience suggests that, in the treatment of chronic heart failure patients with central sleep apnea, especially those with Cheyne Stokes respiration, ASV may be more effective in improving cardiac function and the patient’s prognosis than oxygen therapy. As several randomized clinical trials to confirm the prognostic improvement with ASV therapy are being currently performed, we have to wait for a definitive answer about the effect of ASV treatment. As for the short-term effect of ASV, a previous study by Teschler et al presented favorable effects of ASV over oxygen therapy, CPAP, or bi-level PAP in improving sleep-disordered breathing. In this issue of the Journal, Yoshihisa et al nicely compare the effects of a single night of ASV or oxygen treatment in patients with heart failure and central sleep apnea. They found a clear advantage of ASV therapy over oxygen in terms of cardiac functional improvement. That is, only ASV but not oxygen decreased the levels of atrial and B-type natriuretic peptides after only one night of therapy. This result clearly indicated that the mechanical assistance by ASV using positive pressure on the thorax itself has a big effect in assisting the failing heart and in controlling any breathing abnormality in heart failure patients, and has strong synergic effects in such patients. Which one of these 2 factors exerts the most influence probably depends on the condition of each patient. But the advantage of ASV might be that this machine can work in either one way or both automatically according to the individual patient’s condition. The study of Yoshihisa et al clearly confirmed that this favorable dual action can achieve a good result in a very short period. This study has established a solid foundation for proving the mechanisms of the favorable action of ASV.

Yoshihisa et al, as well as Teschler et al, used similar pressure settings, which are the specific default setting for the ASV machine. This setting seems to be effective and safe in many heart failure patients because no major negative effect derived from this setting has been reported so far. We do not know, however, whether this pressure setting is the best for most heart failure patients and, if so, why. If a patient with heart failure has obstructive sleep apnea with an airway closing pressure of 12 cmH₂O, should we raise the end-expiratory pressure >12 cmH₂O, when a decrease in cardiac output because of blocked venous return may ensue and trigger or worsen the central sleep apnea? At this moment in time, many cardiologists seem to be using this predetermined set of values for ASV treatment. We have to rigorously seek a more individualized set of pressures for each patient using a simple and reliable method in a daily clinical practice to increase the usage of this effective device in an appropriate fashion and thus provide the best benefit to more heart failure patients.

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