Background: Central sleep apnea (CSA) and exercise oscillatory ventilation (EOV) might be both originated from an instability in feedback peripheral and central chemoreflex control of ventilation. However, it is unknown the relation between CSA and EOV in patients with heart failure (HF).

Methods: In 27 patients with moderate to severe CSA determined by cardiorespiratory polygraphy (apnea/hypopnea index 34±11/h, central apnea index 22±10/h), cardiopulmonary exercise test was performed. EOV was defined as 3 or more regular oscillatory fluctuations in ventilation.

Results: EOV was observed in 70% of patients (mean cycle length of EOV: 81±26sec). Notably, the cycle length of EOV was similar to that of CSA (R=0.89, p<0.001, Figure). The patients with EOV had lower rest end-tidal carbon dioxide (ETCO2) level (4.4±0.6 vs. 5.2±0.6%, p<0.001), lower peak oxygen uptake (628±172 vs. 1087±382ml/min, p<0.05) and longer cycle length of CSA (80±25 vs. 61±11sec, p<0.05), compared to the patients without EOV. In patients with cycle length of CSA ≥ 78 sec, 100% of patients had EOV. In patients with cycle length of CSA < 78 sec, however; 61% of patients had EOV. Multivariate analyses revealed that ETCO2 level was the strongest independent predictor of EOV.

Conclusion: Presence of EOV is determined by resting pCO2 level and cycle length of CSA in HF patients with CSA.