Ventricular ectopy\(^1\) and central sleep apnea\(^2\) are independent negative predictors of excess mortality in patients with heart failure. Recent studies have established an association between central sleep apnea and ventricular ectopy.\(^3\) Because the 2 conditions share many common risk factors, however, a causal relationship remains unclear. Here, we present images of a nocturnal polygraphy that provide evidence for a causal relationship between central sleep apnea and ventricular ectopic beats.

A 79-year-old man (body mass index, 24.8 kg/m\(^2\); height, 1.74 m) with known ischemic heart failure (ejection fraction, 30\%) was admitted with suspected cardiac syncopes. The 24-hour ECG revealed frequent monomorphic ventricular ectopic beats with intermittent episodes of a bigeminus. Because of a suspected pulse deficiency and coexistent daytime tiredness, a polygraphy was performed, which confirmed a pulse deficiency (Figure 1) with an effective peripheral bradycardia during the episodes of bigeminus and revealed a severe central sleep apnea syndrome (apnea hypopnea index, 47.3 per hour; mean \(\text{O}_2\) saturation, 90\%). Between 4 and 6 AM, the episodes of ventricular bigeminus were triggered by the hyperpneic phases (Figures 2 through 4). As a consequence, the peripheral pulse plethysmography showed a recurrent bradycardia occurring with a rhythm simultaneous to the central hyperpneic phases.

After adjustment of an adaptive servo ventilation therapy, the polygraphy continued to show ventricular ectopic beats, but no further episodes of bigeminus occurred during the

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**Figure 1.** Paroxysmal ventricular bigeminus in the ECG. The complete pulse deficiency of the ventricular ectopic beats is well documented by the peripheral plethysmography.
night. The patient reported no further syncopes and an improvement in his daytime sleepiness.

Patients with heart failure frequently suffer from sleep-disordered breathing, and central sleep apnea is a common condition in such patients. The underlying Cheyne-Stokes respiration appears to be induced by the heart failure itself and has been identified as an independent predictor of excess mortality. Because central sleep apnea increases sympathetic activity, induction of ventricular arrhythmia has been suggested as a potential explanation. Yet, only a few studies have shown an association between ventricular ectopic beats and central sleep apnea in heart failure patients. In a cumulative analysis of 23 patients, Leung et al have found a 40% increased rate of ventricular ectopy during the hyperpneic phase.

The images of our polygraphy presented here demonstrate for the first time a direct link between central sleep apnea syndrome and ventricular arrhythmia, suggesting a causal relationship between the 2 conditions. Therefore, increased attention should be paid to this sleep disorder in patients with heart failure.

Figure 2. The respiratory (breathing effort of thorax and abdomen, nasal flow, snoring) and cardiovascular (ECG, peripheral plethysmography, peripheral oxygen saturation [SAO2], pulse rate, peripheral plethysmography [plethysmogr.]) parameters of the polygraphy are shown simultaneously. The polygraphy shows recurrent central sleep apneas (nasal flow, thorax, abdomen) with severe oxygen desaturation (SAO2). This image shows over a period of 2 minutes that the episodes of ventricular bigeminus were exactly triggered by the hyperpneic phases of the central sleep apnea. As a consequence, the peripheral pulse rate shows a recurrent bradycardia occurring with a simultaneous rhythm as the central hyperpneic phases. Also see Figures 3 and 4.
Disclosures

None.

References


Figure 3. Polygraphy over a period of 5 minutes. For further details, see Figure 2.
Figure 4. Polygraphy over a period of 15 minutes. For further details, see Figure 2.
Central Sleep Apnea Induces Ventricular Bigeminus: Conclusions From a Single Polygraphy
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