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Methodology Article

Application of CPAP Telemonitoring as a "Health Monitor"

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ABSTRACT

We review the previous literature and our own cases to describe the conditions reflected by Cheyne-Stokes Breathing (CSB) % variability based on data downloaded from CPAP devices in patients with Obstructive Sleep Apnea (OSA) and the possibility of detecting abnormalities in these conditions by telemonitoring CSB. Telemonitoring of CSB can allow early clinical prediction of heart disease onset and exacerbation. In addition, incorporating changes in cycle length may improve the accuracy of anomaly detection, and CSB telemonitoring may also reflect dynamic changes in loop gain phenomena. Furthermore, it may be able to reflect pathologies other than heart disease. However, there are still challenges to be overcome for further clinical application of telemonitoring of CSB.

Keywords: Telemonitoring; Obstructive sleep apnea; Cheyne-Stokes Breathing (CSB); Body mass index; Acute heart failure

Abbrevations: AHF: Acute Heart Failure; AHI: Apnea-Hypopnea Index; Af: Atrial fibrillation; BMI: Body Mass Index; CHF: Chronic Heart Failure; CL: Cycle Length; CPAP: Continuous Positive Airway Pressure; CSA: Central Sleep Apnea; CSB: Cheyne-Stokes Breathing; Gc: Gain controller; Gp: Plant gain; LG: Loop Gain; LVEF: Left Ventricular Ejection Fraction; OSA: Obstructive Sleep Apnea; PB: Periodic Breathing

INTRODUCTION

Continuous Positive Airway Pressure (CPAP) devices have advanced to serve not only as therapeutic devices but also as devices for monitoring their usage and therapeutic effects [1]. In addition, it has been suggested that longitudinal monitoring of sleep indices such as Cheyne-Stokes Breathing (CSB) in individual patients can enable clinical prediction of heart failure [2-4]. In other words, these devices can be used as "Health Monitors" for early detection of cardiovascular pathological exacerbation of patients [5].

LITERATURE REVIEW

Overlapping mechanisms of OSA and CSB

The mutual interactions among the following four endotypes are thought to be involved in the pathogenesis of OSA: "Anatomic Traits of the Upper Airway," "Low Arousal Threshold," "Upper Airway Dilator Muscle Responsiveness," and "Respiratory Control Instability" [6]. Especially, for respiratory control instability in the non-REM phase, the theory of Loop Gain (LG), which is an engineering term, has been applied [7]. It is considered that there are three important factors affecting the LG in the respiratory system: Changes in the partial Pressure of Arterial Carbon Dioxide ($PACO_2$) due to changes in the ventilation volume (plant gain; Gp), circulation delay, and change in the ventilation volume in response to the stimulation of chemoreceptors by $PACO_2$ (controller gain; Gc). Sands, et al. [8], have reported that this LG allows prediction of immediate responses to CPAP. Orr, et al. [9], have reported that Central Sleep Apnea (CSA)-CSB and treatment emergent CSA can be almost always explained as the consequences of an increase in this LG and that there is sufficient evidence for the overlapping mechanisms of OSA and CSA. Naughton, et al. [10], have suggested that the LG can be an important index for the severity of apnea in addition to the standard indices such as the AHI. Thus, we analyzed data collected at our hospital and from previous reports with a focus on the LG in each disease and

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explored whether CSB monitoring with CPAP devices was clinically useful in patients with OSA.

CSB% as an indicator

In our previous study, the median CSB% was as low as 0.34% in stable long-term CPAP users. However, the CSB% was high in patients with comorbidities such as Af or CRBBB, and it is difficult to set a standard value because of individual differences even during the stable period. It is still important to examine the time-series variation in a single patient [5].

Clinical usefulness of CSB telemonitoring in heart disease

In heart disease, low cardiac output, highly activated sympathetic nervous system, and pulmonary congestion (i.e., elevated left atrial pressure) induce circulation delay and increase Gc and Gp. Moreover, these conditions mutually interact to cause the CSB pattern. In each disease, the LG appears to change depending on the severity of the disease, disease stage (e.g., the acute and chronic phases), and posture (e.g., recumbency) even in the same patient. It is complicated to consider the involvement and changes in these multiple factors [11]. However, changes in the CSB% often indicate an exacerbation during an increase and stable condition during a decrease in CSB in the real-world setting for individual patients.

Acute heart failure

Javaheri, et al. [12], have reported that CSB may be the first sign of mild heart failure even before the onset of symptoms of orthopnea or paroxysmal nocturnal dyspnea. We have also reported that the CSB% demonstrated an increase in most of the patients in the AHF group approximately 8–10 days before they visited the outpatient clinic with symptoms. Therefore, the most important mechanism for the onset of CSB appears to be an increase in the Gc due to increased sensitivity of chemoreceptors [3]. The increased sensitivity of chemoreceptors has been attributed to increased left atrial pressure. A rapid increase in left atrial pressure increases the sensitivity of chemoreceptors [13]. In addition, increased pulmonary capillary wedge pressure is common in CSB and is considered to correlate with the severity of CSB [14-16]. Yumino, et al. [17], CSB is also reported to be associated with the state of the body fluid, including the degree of overnight peritoneal fluid shift, which suggests that CSB may increase the Gp in the LG [17]. Thus, increased CSB in AHF may be associated with an increase in the Gc, mainly the Gp. Close monitoring of the CSB% allows detection of abnormalities at an early stage.

Chronic heart failure

In patients with a history of heart failure, including those with an originally high CSB%, monitoring of the CSB% is less sensitive for the detection of abnormalities than in patients with AHF [3]. Classically, CSB has been attributed to increased circulation delay from the lungs to chemoreceptors [16,18-20]. Although the circulation delay appears to be one of the causes, CSB does not often occur in CHF patients with low LVEF in whom the circulation time is presumably delayed [21]. There has been a report of a decrease in CSA after mitral clip placement [22]. In our previous study, we have also detected increased ratios of early mitral inflow velocity to early diastolic mitral annular velocity and a history of heart failure despite favorable ejection fractions on echocardiography in patients who did not experience CSB at CPAP initiation but subsequently developed CSB [5]. We assume that CSB occurs in patients with mild pulmonary congestion even during relatively stable hemodynamic conditions; thus, increased Gc and Gp may be factors for the onset of CSB. In other words, CSB% monitoring data also reflect the severity of pulmonary congestion and diastolic dysfunction.

Another index that should be noted is the Cycle Length (CL). The subanalysis of the SERVE-HF trial has shown that the baseline CL was significantly longer in patients with adverse events [23]. Our previous study has also reported that the cut-off CL was 68.9 s, which significantly distinguished between patients with and without exacerbation of heart failure. Since our previous study is a single-center retrospective study affected by biased selection of cases and small sample size, this cut-off CL cannot be a generalized reference value [3]. However, it is sufficiently plausible that monitoring of the CL in addition to the CSB% would contribute to further improvement in the sensitivity for detecting exacerbation and abnormalities and to stratification of the risks of patients with heart failure.

Atrial fibrillation

CSB telemonitoring gets confusing in the presence of Af. Among studies on the improvement in CSB after treatment of Af. Fox, et al. [24], reported that the incidence of central respiratory events decreased immediately after defibrillation in 116 patients with Af or atrial flutter. In the first place, the cumulative incidence of Af is higher in patients with OSA than in those without OSA. In this regard, Gami, et al. [25], have described the mechanisms of acute onset of Af and progression to chronic Af as follows: Mechanisms of acute onset of Af:

Presence of comorbidities such as hypertension, diastolic dysfunction, heart failure, and obesity

- Factors for sympathetic nervous system surge due to increased left atrial pressure and vagal-sympathetic disequilibrium
- Changes in the intrathoracic pressure and extension of the atria and pulmonary vessels

Mechanisms of progression to chronic Af

- Persistence of the comorbidities described above
- Increased activity of cardiac sympathetic nerves
- Atrial fibrosis, remodeling, and enlargement

The CSB% often increases with the onset of Af. Our previous study has also reported that the CSB% increases with the perpetuation of Af, in other words, the progression from transient to persistent Af [5]. Conversely, this suggests that perpetuation of Af could be predicted by monitoring the CSB% in these patients.

METHODOLOGY

Normal case

A 57-year-old man with a Body Mass Index (BMI) of 29.0 kg/m^2 at approximately 5 years after CPAP initiation.

Acute Heart Failure (AHF)

A 75-year-old woman with a BMI of 32.2 kg/m^2 at approximately 7 years after CPAP initiation. AHF was caused by acute mitral regurgitation due to tendon rupture.

Acute myocardial infarction complicated by AHF with a prior history of Af

A 64-year-old man with a BMI of 25.7 kg/m^2 at approximately 2 years after CPAP initiation. He had a history of persistent Atrial fibrillation (Af). Although he had remained in sinus rhythm for a while, his Af relapsed. Subsequently, he developed acute myocardial infarction, which was complicated by AHF.

Exacerbation of Chronic Heart Failure (CHF)

An 83-year-old man with a BMI of 24.0 kg/m^2 at approximately 13 years after CPAP initiation. He had poor adherence to medication because of cognitive decline. He repeatedly experienced acute exacerbation of paroxysmal Af and CHF over a short period of time.

Changes in the CSB% over 1 year in each case are presented in Figure 1.

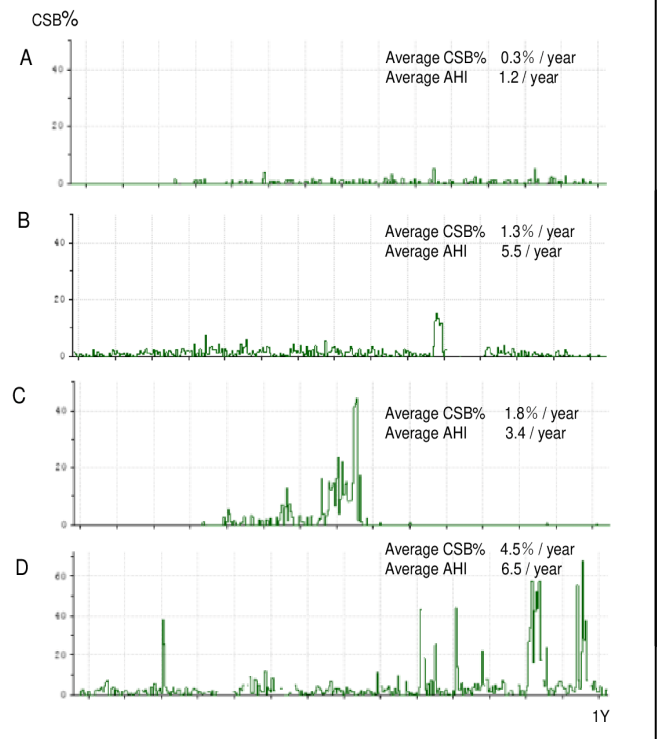

Figure 1: Fluctuations in CSB% over 1 year for each case. **Note:** CSB=Cheyne Stokes Breathing, AHI=Apnea Hypopnea Index.

RESULTS AND DISCUSSION

From another perspective, the mechanism of the onset of CSB may differ between transient and persistent Af. It appears that transient Af is mainly affected by pulmonary congestion due to rapidly increased left atrial pressure, in other words, increased Gc or Gp, whe reas persistent Af is largely affected by left atrial enlargement and circulation delay. Thus, monitoring of the CSB % may detect increases in the above-described factors, which are involved in the increase and progression of CSB. However, when the use of the CSB% is considered as a pathological index, the most important problem is that some patients with persistent Af show high CSB%, although their condition is relatively stable. The CSB% also demonstrates substantial fluctuation. Thus, the CSB% alone cannot be an index that facilitates a better understanding of pathological conditions.

Figure 2 shows Case F with recurrent heart failure and Case G without a history of heart failure. In both cases, the patients had persistent Af. The CL was clearly different between these cases. Furthermore, in Case F, the CL during exacerbation of heart failure (E-1) was longer than that during the stable phase (E-2). Although LVEF measured by echocardiography was ≥ 55% in both phases, the left atrial diameter was greatly different between Case E (82 mm) and Case F (49 mm). These findings suggest that the CL should be considered in patients with Af to determine whether CSB should be treated. In other words, in heart disease, the accuracy of abnormality detection may be improved by adding the mean value of CL and time-series variation of CL to the index.

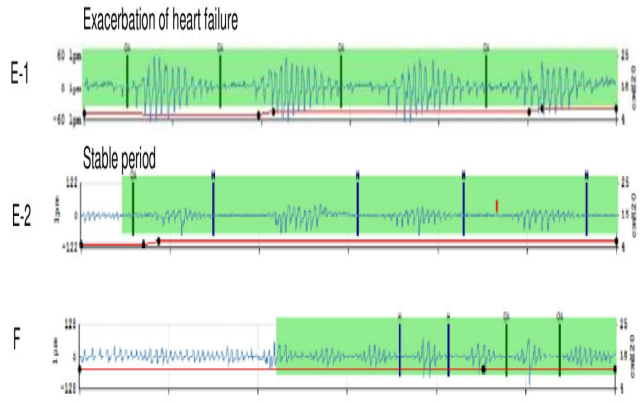

Figure 2: Comparison of CPAP telemonitoring raw waveforms in patients with permanent atrial fibrillation. **Note:** CPAP= Continuous Positive Airway Pressure.

Figure 3 shows the changes in CSB% over a one-year period in patients other than those with heart disease: Case H had spinocerebellar degeneration and was attending our outpatient clinic with progressive symptoms. Thus, CSB% may reflect patient conditions other than cardiac disease. There are some problems, such as the lack of uniformity of CSB calculation criteria for each CPAP device and the current situation where CL is not included in the downloadable items. Figure 4 shows the association between the pathophysiology-loop gain-endotypes and CSB.

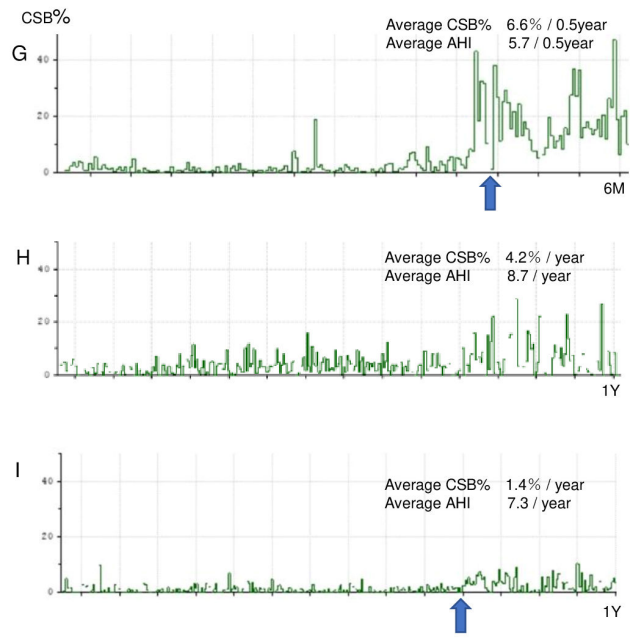

Figure 3: Shows the association between the pathophysiology-loop gain-endotypes and CSB. **Note:** Case G had a stroke in the right middle cerebral artery region and was admitted to a hospital on the date indicated by the arrow; Case I had depression and started taking antidepressants from the arrowed date.

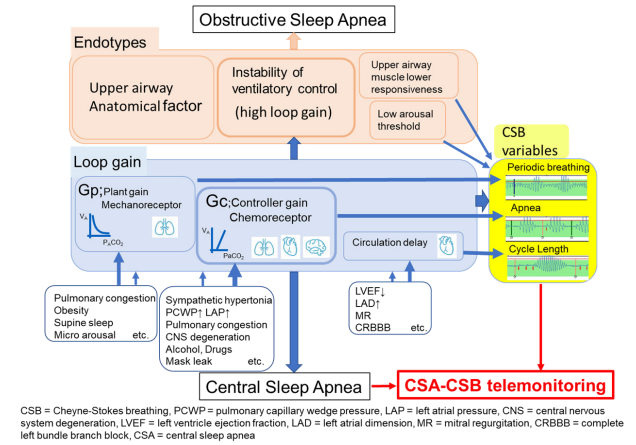

Figure 4: Imaginary diagram of pathophysiology-loop gain-endotype and CSB linkage.

CONCLUSION

Continuous positive airway pressure devices can be used as "Health Monitors" for early detection of cardiovascular pathological exacerbation of patients with obstructive sleep apnea. Telemonitoring of Cheyne-Stokes Breathing can allow early clinical prediction of heart disease onset and exacerbation. In addition, incorporating CL variation may improve the accuracy of anomaly detection. In addition to telemonitoring of Cheyne-Stokes Breathing reflecting the dynamic changes in the loop gain phenomenon. Furthermore, it may be able to reflect pathologies other than heart disease. However, there are still challenges to be overcome for further clinical application of telemonitoring of Cheyne-Stokes Breathing.

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CONFLICTS OF INTEREST

The author declare no conflicts of interest associated with this manuscript. Availability of data and material. The datasets used and/or analyzed during this manuscript are available from the corresponding author on direct request.

ETHICAL APPROVAL

All patients provided written informed consent after we provided information regarding the study procedure. Informed consent: Written informed consent was obtained from participants.

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