Letter to the Editor

Does Sleep Apnea Change Over Time in Patients With Acute Myocardial Infarction?

To the Editor:

I read with great interest the article by Tsukamoto and Ohara who reported the temporal worsening of sleep-disordered breathing (SDB) in patients with acute coronary syndrome (ACS). The authors stated that SDB in the acute phase of myocardial infarction (MI) has not been well analyzed before and that there are no previous reports documenting changes in the severity of SDB during the acute phase of MI as analyzed by polysomnography (PSG). Additionally, I noticed that the authors did not exclude some of the conditions that may increase the severity of SDB in the acute phase of MI, such as being on sedation or narcotics or decreased level of consciousness, which may account for the temporary worsening in the acute phase of MI.

In a recently published paper, we have already reported the prevalence and time-course of SDB in 50 consecutive patients with ACS using full overnight PSG within 3 days of the acute event and 6 months later after excluding the above conditions that may affect SDB. The body mass index of our group was comparable to that of Tsukamoto and Ohara’s patients (ie, 26.9±0.8 kg/m²). Fifty-six percent of the studied group had an apnea/hypopnea index (AHI) >10/h, 44% had an AHI >20/h and 34% had an AHI >30/h. AHI was 23.1±3.6/h. AHI was divided into the obstructive apnea index (OAI) and central apnea index (CAI). OAI was 20.3±3.2 and CAI was 3.9±0.8. Cheyne-Stoke respiration (CSR) lasting more than 10% of total sleep time was documented in 6 patients. Ejection fraction was significantly lower in the group that had CSR compared with those who did not. Interestingly, AHI, OAI and the mean duration of obstructive apneas did not change significantly over the 6 months. On the other hand, CAI and central apnea duration were significantly lower in the second assessment.

A few possible explanations may clarify the differences between our results and those of Tsukamoto and Ohara. A possible confounder is the administration of drugs that may affect respiration, such as narcotics and hypnotics. Although we used a drug-free period of at least 48 h, Tsukamoto and Ohara did not address this point in their methodology. Additionally, studies such as ours may have a risk of “regression towards the mean” phenomenon, which means that if a subgroup with a higher AHI is tested a second time, it is likely that the AHI may be lower at the second investigation.

Obviously, more studies with larger number of patients and stringent control for possible confounders are required to clarify the relationship between ACS and SDB.

References

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Author’s Reply

Importance of Patient Selection for Analyzing the Effect of Acute Myocardial Infarction on Sleep-Disordered Breathing

Dr BaHammam raises several issues with regard to our investigation of sleep-disordered breathing (SDB) in the acute phase of acute myocardial infarction (AMI). In our study, a significant reduction in the apnea/hypopnea index (AHI) was observed during the acute phase of myocardial infarction, whereas in their study, they did not. Why were the results different between our study and theirs? They mentioned the possibility that the use of narcotics or hypnotics might affect the result of our study, but neither drug was used 24 h before polysomnography (PSG), so cannot explain the difference. We speculate that the difference in the results is mainly caused by the difference in the study population. We would like to emphasize that we paid much attention to determining the entry criteria in the present study. As reported previously, congestive heart failure (CHF) has a close relationship with the severity of SDB, especially with the severity of central sleep apnea (CSA). Therefore, to assess the effect of AMI on SDB, we carefully excluded patients with the complication of CHF, whereas their study included patients with relatively low ejection fraction (40.2±3.2), who may have been complicated with CHF. This might explain their result that only the central apnea index was significantly reduced in the chronic phase. Moreover, in their study, although 13 patients with AMI were included, they analyzed patients with AMI and those with unstable angina together. Therefore, the study population is quite different from ours. Because we also considered the possibility that residual ischemia may affect SDB, we carefully excluded such patients. In their study, they did not mention the administration of β-blockers, whereas in our study β-blockers were used in all patients. There is some possibility that in our study the use of β-blockers may have played a role in the reduction in the AHI. The difference in study protocol is another important point. They performed the second PSG only for patients with AHI >10 in the first PSG, whereas we performed the second PSG for all patients.

In our study, we revealed that the presumed mechanisms of CSA in CHF could not be applied to SDB in AMI; that is, the mechanism of temporal worsening of SDB in AMI has been left unsettled. Further study with a larger study population and analysis from different viewpoints are required to reveal the pathophysiology of temporal worsening of SDB in AMI.
References


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