Editorial Comment

A Device for Nocturnal Hypertension with Sleep Apnea

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Researchers have discovered that blood pressure (BP) varies in a diurnal manner throughout a 24-h period, being higher during the day and lower at night. Most people have a dipping BP pattern characterized by a nighttime BP that is 10–20% lower than their daytime BP. Individuals who experience a less than 10% reduction in nighttime BP are described as having a non-dipper type BP pattern. The prognosis is poor in non-dipper patients who show little decrease of BP during the night or those with a morning surge of BP, and their incidence of cerebral infarction is increased (*1*, *2*). Thus, nocturnal BP is considered to be a better predictor of cardiovascular events than office BP, home BP, or average daytime BP (*3*). In patients with obstructive sleep apnea syndrome (OSAS) or upper airway resistance symptom (UARS), it has been reported that a non-dipper pattern is common (*4–6*).

Though its clinical application is reportedly difficult, assessment *via* muscle sympathetic nerve activity (MSNA) reveals an increase in sympathetic activity, such as that occurring during sleep apnea. Using monitoring and sphygmomanometry with a Finapres, transient elevation of BP is found when breathing starts again. This sympathetic activation persists in OSAS patients until the next day, and it is thought to be a cause of hypertension.

Nieto *et al*. (*7*) reported that the frequency of hypertension showed an increase with aggravation of sleep-related breathing disorders in the Sleep Heart Health study. In the studies of Tanigawa *et al*. (*8*) and Sasanabe *et al*. (*9*), BP also increased with aggravation of sleep-related breathing disorders and the frequency of hypertension showed an increase as well. It was mentioned in JNC VI that the presence of OSAS should be suspected in patients with drug-resistant hypertension. Furthermore, OSAS was listed first among the causes of hypertension in JNC 7 (*10*). Based on these results, great importance should be attached to OSAS in the diagnosis and treatment of hypertension. However, the Japanese guidelines (JSH 2004) (*11*) do not mention anything about OSAS.

Invasive recording of BP is generally difficult to perform. On the other hand, ambulatory BP monitoring (ABPM) can easily be done at night, but intermittent elevation of the BP at the time when breathing restarts cannot be measured precisely. According to the method of Shirasaki *et al*. (*12*), a decrease of arterial O_2 saturation is used as a trigger to start sphygmomanometry in OSAS patients, and a novel and important method is used to control elevated BP at the time of restarting breathing. If home BP monitors that incorporated this method became widely available, drug-resistant hypertension and the complicated clinical status of OSAS patients would be improved, and more appropriate treatment would be provided.

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