Sleepers Versus Nonsleepers: Another Twist to the Dipper/Nondipper Concept
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T
here is growing evidence that an elevated nocturnal
blood pressure is associated with an adverse cardio-
vascular outcome. In the Dublin Outcome Study, for
each 10-mm Hg increase in mean nighttime systolic blood
pressure, the mortality risk increased by 21%.1 At present,
ambulatory blood pressure measurement is the only technique
that permits close examination of the circadian profile and
identification of patterns that may be associated with risk.

Much happens to the cardiovascular system at nighttime,
especially in relation to blood pressure. The patterns of
nocturnal blood pressure–nocturnal hypertension, nocturnal
hypotension, dipping and nondipping, reverse dipping, and
autonomic failure–have been largely ignored in clinical
practice. Many studies evaluating morbidity and dipping
status have supported the concept that a diminished nocturnal
blood pressure fall is associated with a worse prognosis.2

However, despite compelling evidence that changes in night-
time blood pressure may hold many secrets that, if unlocked,
might benefit the clinical management of hypertension, there
has been reluctance to focus on nocturnal blood pressure both
clinically and in hypertension research.3

The nocturnal period of the 24-hour blood pressure profile,
which is surprisingly complex, can be divided into a number
of windows in which discrete phenomena may occur. These
windows are the retiring (or perhaps more aptly named vesperal)
window, the nighttime (or basal) window during which sleep is most likely, and the preawakening (or matinal)
window, which precedes rising. In the normal individual
there is a decline in blood pressure in the vesperal window
daytime levels of blood pressure to reach a plateau
during the basal window (the “dipping” pattern), with a
modest rise in the matinal window to regain daytime levels of
blood pressure.4 In hypertensive patients (or some normoten-
sive patients with cardiovascular disease), the decline in the
vesperal window may be absent (nondipping) so that blood pressures do not reach basal levels, or blood pressure may
even rise above daytime levels (reverse dipping).5 Alterna-
tively, there may be a marked fall in basal blood pressure
(extreme dipping). Patients with a marked nocturnal fall in
blood pressure are at risk for nonfatal ischemic stroke and
silent myocardial ischemia. This is particularly likely in
extreme dippers, who already have atherosclerotic disease
and in whom excessive blood pressure reduction is induced
by injudicious antihypertensive medication.5 This possibility
was originally enunciated by Floras6 as long ago as 1988,
when he postulated that patients with critical coronary steno-
ses or hypertrophied ventricles might have impaired coronary
vasodilator reserve and would, therefore, be at greatest risk of
myocardial ischemia or infarction if subendocardial perfusion
pressure fell below the lower threshold of blood flow auto-
regulation. This was most likely to occur during sleep, when
excessive antihypertensive treatment might cause unrecog-
nized nocturnal hypotension leading to coronary artery hypo-
perfusion, which might explain why treatment had not dimin-
ished the risk of myocardial infarction in patients with
hypertension.6 Also, in hypertensive patients, the matinal rise
in blood pressure may soar above the daytime average, the
preawakening or morning surge, which may be excessive in
extreme dippers leading to a high-risk of future stroke.5

I have applied the term “basal” to the window between the
vesperal and matinal windows in acknowledgement of the
seminal article written by Horace Smirk in 1964.7 In this
article, Smirk outlines the technique for obtaining basal blood
pressure under standardized conditions with sedation using
oral pentobarbitone, which he differentiates from casual
blood pressure as obtained in hospital or elsewhere without
special precautions; the term supplemental blood pressure
was applied to the difference between the casual and basal
blood pressures. Smirk7 observed that the basal blood pres-
sure was practically a physiological constant in normotensive
subjects and, although more variable in hypertensive patients,
was much less variable than the casual blood pressure.
Moreover, he observed that the casual blood pressure of an
individual was a “most unreliable” guide to the basal blood
pressure and that basal and supplemental pressures were
almost independent variables. The compelling conclusion
from Smirk’s analysis7 was that basal blood pressure was
superior to casual pressure in predicting outcome. I believe
that the growing evidence for the superiority of nocturnal
blood pressure measured with ambulatory blood pressure
monitors is that we are measuring blood pressure when it is
most stable or basal, in contrast to daytime measurements,
which are subject to so many influences, not the least of
which is activity. This reasoning is supported by evidence
that administering a mild sedative during ambulatory blood
pressure monitoring may help to identify the patients with a
high cardiovascular risk, namely, those patients who continue
to manifest a blunted nocturnal dip despite sedation.8

Valuable though the information derived from nocturnal
blood pressure may be, there are several methodologic
limitations to recording blood pressure at night. These include poor reproducibility, different criteria for defining dipping/ nondipping status, and the influence of daytime physical activity on the dipping phenomenon.9 Now Verdecchia et al10 have added a further caveat to the measurement of nocturnal blood pressure, namely, the influence of sleep disturbance. A review of the literature on the effect of the quality of sleep on blood pressure measurement at night is nonconclusive, with some (but not all) studies suggesting that nocturnal blood pressure may be affected by the noise and disturbance caused by repeated cuff inflations, but none have assessed the prognostic significance of such sleep disturbance.10 In the Italian study there were 356 major cardiovascular events and 184 all-cause deaths over a mean 7-year follow-up period in 2934 hypertensive subjects. Sleep quality was assessed by questionnaire according to the number of estimated hours of sleep deprivation. The interesting finding from the study is that nighttime blood pressure significantly rises and loses its prognostic significance in a sizeable minority (≤14% of untreated hypertensive subjects) who perceived sleep deprivation of ≥2 hours during overnight blood pressure monitoring. On the other hand, 86.4% of subjects did not report any significant disturbance of sleep in spite of having measurements every 15 minutes throughout the night. The number of cardiovascular events in the subset without significant perceived sleep disturbance compared with that with significant perceived sleep disturbance was 300 and 56, respectively, and the total number of deaths in the 2 groups was 157 and 27, respectively.

Although the results of this study are interesting and important in that they provide outcome data suggesting that sleep disturbance may affect the prognostic value of nocturnal blood pressure, they must be interpreted with some caution. Apart from the potential unreliability of questionnaires to assess sleep quality and the fact that the group with more severe sleep deprivation was smaller than the group without sleep deprivation, my main concern is that the methodology of nocturnal measurement may have influenced the results. Firstly, there is the issue of the frequency of measurement. I would argue that the potential disturbance to sleep induced by recording blood pressure every 15 rather than every 30 minutes throughout the day and night.11 Second, there is the issue of the amount of sleep disturbance caused by the ambulatory device used, and there is a clear message here for manufacturers of ambulatory devices. Interestingly, the older device used in the Italian study, the SpaceLabs 5200 model, was associated with more sleep deprivation and lesser day–night blood pressure reduction than the newer models, the SpaceLabs 90202 and 90207, indicating that the bigger size and louder noise of the pump during inflation in the early model might have induced more sleep disturbance. It behoves manufacturers, therefore, to look critically at the design of devices for ambulatory blood pressure measurement. Although both the size of monitors and the noise of pumps have improved in recent times, it is fair to say that manufacturers have shown little innovative flair over the past 30 years in providing a device for ambulatory measurement that is not dependent on a relatively heavy and cumbersome recorder being connected to an inflatable arm cuff.

Disclosures

None.

References