Night-time blood pressure patterns and target organ damage: A review

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BACKGROUND: Individuals who do not have a 10% to 20% reduction in blood pressure (BP) during the night are known as 'nondippers'. The cause of this nondipping phenomenon is not fully understood; however, there is a growing body of evidence linking a nondipping BP pattern with target organ damage.

OBJECTIVE: To review the literature and present an overview of the target organ damage found to be associated with a nondipping BP pattern.

METHODS: PubMed, CINAHL and Medscape searches of all available English language articles from 1986 to 2005 were performed. Search terms included 'BP nondipping', 'BP dipping' and 'target organ damage'.

RESULTS: There is evidence to suggest that individuals with hypertension who exhibit a nondipping BP profile are at higher risk of cardiac and extracardiac morbidity and mortality. In particular, nondippers with essential hypertension have been found to have more advanced left ventricular hypertrophy, left ventricular mass and left ventricular mass index, carotid artery wall thickness, carotid artery atherosclerotic plaques, silent cerebral infarct, stroke, cognitive impairment and microalbuminuria.

CONCLUSION: A better understanding of the importance of the circadian variations of BP may help to identify those at higher risk of cardiovascular morbidity and mortality, as well as lay the foundation for interventions to prevent/treat alterations in night-time BP patterns.

Key Words: Blood pressure dipping; Blood pressure nondipping; Dippers; Nondippers; Target organ damage

Amblulatory blood pressure monitoring (ABPM) is a noninvasive, automated portable technology that measures and records blood pressure (BP) over a 24 h period (1). This technique has allowed researchers and clinicians to measure the percentage of elevated BP readings, as well as 24 h BP loads and BP patterns (2). Consequently, it has been discovered that BP varies over 24 h in a diurnal manner, being higher during the day and lower at night (3). The normal variation in BP is characterized by a 10% to 20% reduction in BP from day to night (2). Individuals with this decline in night-time BP are known as 'dippers', and those who experience a blunted decline in night-time BP are known as 'nondippers' (4). Researchers have determined individuals to be nondippers on the basis of a less than 10% reduction in night-time systolic BP (SBP) (5-11), diastolic BP (DBP) (5) or both SBP and DBP measurements (12-31).

Rythme nyctéméral de la tension artérielle et atteinte des organes cibles : Survol

HISTORIQUE : Les personnes qui ne présentent pas une baisse de 10 % à 20 % de leur tension artérielle (TA) la nuit sont appelées *nondippers*. La cause de ce phénomène n'est pas entièrement élucidée. Par contre, les preuves s'accumulent quant à l'existence d'un lien possible entre l'absence de fluctuation à la baisse de la TA (ou *nondipping*) et l'atteinte des organes cibles.

OBJECTIF : Passer en revue la littérature et présenter une vue d'ensemble de l'atteinte des organes cibles observée en lien avec l'absence de fluctuation à la baisse de la TA nocturne.

MÉTHODES : Les auteurs ont interrogé les réseaux PubMed, CINAHL et Medscape pour recenser tous les articles de langue anglaise accessibles publiés entre 1986 et 2005. Les mots-clés de l'interrogation étaient « BP nondipping », « BP dipping » et « target organ damage ».

RÉSULTATS : Selon les preuves recueillies, les hypertendus dont la TA ne diminue pas la nuit sont exposés à un risque plus élevé de morbidité et de mortalité cardiaques et extracardiaques. Parmi les *nondippers* atteints d'hypertension essentielle, on a particulièrement noté la présence plus marquée des manifestations suivantes : hypertrophie ventriculaire gauche, augmentation de la masse ventriculaire gauche ou de l'indice de masse ventriculaire gauche et de l'épaisseur de la paroi artérielle carotidienne, plaques athéroscléreuses carotidiennes, infarctus cérébraux silencieux, AVC, atteinte cognitive et microalbuminurie.

CONCLUSION : Une meilleure compréhension de l'importance des variations nyctémérales de la TA pourrait contribuer à l'identification des sujets exposés à un risque plus élevé de morbidité et de mortalité cardiovasculaires et constituer la base de nouvelles interventions axées sur la prévention et le traitement du phénomène de *nondipping*.

The exact mechanisms of action responsible for nondipping are not fully understood. However, data suggest that nondippers have an increase in sympathetic nervous system activity (32,33) and a decrease in parasympathetic nervous system activity throughout the night (32,34). Nondipping is more commonly found among individuals with renal diseases, autonomic neuropathies, diabetes, sleep apnea, autonomic dysfunction, malignant hypertension and secondary hypertension (35,36). In addition, approximately 25% of individuals with essential hypertension have a nondipping BP profile (37).

There is evidence to suggest that individuals who exhibit a nondipping BP profile are at higher risk of cardiac and extracardiac morbidity (38). Verdecchia et al (39) used data from the Progetto Ipertensione Umbria Monitoraggio Ambulatoriale (PIUMA) study to examine the morbidity and mortality rates of 715 essential hypertensive patients over a one- to 13-year

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period. They found a significantly higher rate of cardiovascular events (myocardial infarction, unstable angina, stroke, transient cerebral ischemia, symptomatic aortoiliac occlusive disease, congestive heart failure and cardiac death) among hypertensive individuals with a nondipping BP pattern. In this study, the nondipping BP profile was determined to be an independent predictor of cardiovascular events (39). Other researchers have also reported greater cardiovascular events among hypertensive nondippers (31,40) especially in the female population (31,41).

Several studies involving subjects with uncomplicated essential hypertension have revealed an association between a nondipper BP profile and an increased risk of target organ damage at the cardiac, renal, vascular and cerebrovascular levels (6,9,10,15,18,27,28,42-45). Target organ damage has also been found to be linked with two additional abnormal BP patterns: reverse dipping (nocturnal rise in night-time BP) and extreme dipping (nocturnal fall in night-time BP of greater than 20%) (46). Although reverse dipping and extreme dipping BP patterns have been associated with greater risk of intracranial hemorrhage and fatal stroke (46), and silent cerebral infarct (6,46) and cerebral ischemia (46), respectively, for the purposes of the present paper, we will focus on the nondipping BP pattern. In the present article, we will review the nondipping BP pattern and its association with cardiac structural and functional alterations, hemodynamic variations, carotid artery abnormalities, cerebrovascular disease and renal damage among individuals with essential hypertension. The findings of the review are presented in Table 1.

CARDIAC STRUCTURAL ALTERATIONS

Left ventricular hypertrophy

Left ventricular hypertrophy (LVH) is a strong and independent predictor of cardiovascular complications and death among individuals with essential hypertension (47). Cuspidi et al (18,43) studied young and middle-aged, never-treated hypertensive patients with a less than one-year diagnosis of essential hypertension. On the basis of two ABPM sessions, individuals were classified as nondippers or dippers. LVH was found significantly more often among nondipping, hypertensive individuals. Individuals classified as dippers or nondippers did not differ with regard to age, sex, body mass index, smoking habit and known duration of hypertension. Differences were observed between the two groups with regard to mean SBPs and DBPs, with significantly higher values noted during the day in dippers and during the night in nondippers. Cuspidi et al (18,43) also examined subjects with a reproducible dipping pattern, a reproducible nondipping pattern and a variable dipping pattern. The prevalence of LVH was found to be highest among individuals with a reproducible nondipping pattern. In addition, individuals with a variable dipping pattern had a higher prevalence of LVH than individuals with a reproducible dipping pattern (43).

Previously, researchers have argued that attenuation of nocturnal BP fall is potentially harmful because it exposes individuals to a higher hemodynamic load throughout a 24 h period (48). However, in Cuspidi et al's study (43), both dippers and nondippers had similar average 48 h SBP and DBP. These findings suggest that cardiac structural changes among nondippers are due to the absence of night-time BP dipping (18), and are not the result of a higher overall BP load (18,43). Several other researchers have reached similar conclusions supporting a

TABLE 1
Target organ damage associated with night-time blood pressure patterns among individuals with hypertension

Target organ damage	Dipping status (reference)
Future cardiovascular events*	> in nondippers (31 [†] ,39,40,41 [†])
Left ventricular hypertrophy	> in nondippers (6,10,18,43)
Left ventricular mass and left ventricular mass index	> in nondippers (12,15,18,20,24 [†] ,28,29-31 [†] ,32,42 [‡] ,43,49,50)
Left atrial dimension	> in nondippers (20,43,49)
Atrial filling fractions	> in nondippers (20)
End-diastolic diameter	> in nondippers (11,20)
Interventricular septum thickness	> in nondippers (43)
Aortic root diameter	> in nondippers (43)
Left atrial maximum/minimum volumes	> in nondippers (52)
Left atrium passive emptying volume and fraction	< in nondippers (52)
Left atrium active emptying volume and fraction	> in nondippers (52)
Relative wall thickness	> in nondippers (24 [†])
Posterior wall thickness	> in nondippers (24 [†] ,31)
Atrial/brain natriuretic peptide	> in nondippers (58)
Left ventricular ejection fraction	↓ in nondippers (49,58)
Supraventricular/ventricular ectopies	> in nondippers (49,59)
QT dispersion/QTc interval	> in nondippers (60,61)
Systemic vascular resistance	↑ at night in nondippers (62) ↓ at night in dippers (62)
Stroke index	↑ at night in nondippers (63) ↓ at night in dippers (63)
Cardiac index	less of a ↓ at night in nondippers (63) ↓ at night in dippers (63)
Cardiac output	No difference between nondippers and dippers (24,62)
Pulse pressure	less of a ↓ at night in nondippers (12,67) ↓ at night in dippers (12,67)
Carotid artery intima-media thickness	> in nondippers (9,14 [§] ,15,17 [§] ,18 [§] ,44)
Carotid artery plaques	> in nondippers (9,14 [§] ,17 [§] ,43 [§])
Stroke	> in nondippers (46,73-75)
Lacunae	> in nondippers (6,10)
Periventricular hyperintensities	> in nondippers (6,10)
White matter lesions	> in nondippers (76)
Previous cerebrovascular event	More nondipping in those with previous events (71)
Cognitive impairment	> in nondippers (79)
Dementia	> in nondippers (78,81)
Memory	↓ in nondippers (82)
Sensory motor speed	↓ in nondippers (82)
Microalbuminuria	> in nondippers (6,27,84,85)

*Cardiovascular events: the Verdecchia et al (31) study included myocardial infarction, sudden cardiac death, stroke, transient ischemic attack, new-onset angina, new-onset congestive heart failure, aortoiliac occlusive disease, thrombotic occlusion of retinal artery and renal failure requiring dialysis. The Verdecchia et al (39) study included myocardial infarction, unstable angina with ischemic changes, stroke, transient cerebral ischemia, symptomatic aortoiliac occlusive disease and congestive heart failure. The Zwieler et al (40) study included transient ischemic attack, myocardial infarction and apoplexy. The Verdecchia et al (41) study included myocardial infarction, congestive heart failure, sudden death, stroke, transient ischemic attack, aortoiliac occlusive disease and retinal artery occlusion; [†]Among nondipping women only; [‡]Isolated systolic hypertensive subjects; [§]Nonsignificant trend. ↓ Decrease; ↑ Increase; > Higher; < Lower

higher prevalence of LVH among individuals who exhibit a nondipping BP profile (6,10).

LV mass and the LV mass index

Cuspidi et al (15,18,43) and Ijiri et al (49) studied nocturnal BP and target organ damage among untreated hypertensive individuals. Demographic characteristics and mean 48 h SBPs and DBPs did not differ between the dipper and nondipper groups. However, individuals with a nondipping BP status demonstrated significantly larger LV masses (LVMs) and LVM indexes (LVMI) than their dipping counterparts. Comparable findings have been demonstrated by other investigators studying cardiac abnormalities among individuals whose BP did not dip at night and who had essential hypertension (12,20,28,50,86), isolated systolic hypertension (42) and normotension (51).

In addition, several researchers have discovered sex-specific cardiovascular adaptation due to diurnal BP variations among persons with essential hypertension. In studies examining dipping status and hypertensive target organ damage, researchers found that LVM increased with ambulatory BP in women and men. However, this increase in LVM and LVMI was linked to the absence of a normal nocturnal BP fall in women only (24,29-31). The findings remained statistically significant after adjustment for average 24 h BP values, thereby suggesting that cardiac abnormalities may be more closely related to an altered diurnal BP rhythm rather than a higher average BP over 24 h. These data may be particularly significant, given that a nondipping BP profile has been found to be more common in hypertensive women with future cardiovascular complications (41).

Cardiac geometry

Abnormal cardiac geometry is often found among hypertensive individuals; left atrium enlargement is a common finding (52). Hypertensive individuals with LV concentric hypertrophy have been found to have more advanced target organ damage (53,54) and have the greatest likelihood of having a cardiovascular event (29,55) compared with individuals with other LV geometric patterns.

Several researchers have found an increased prevalence of altered patterns of cardiac structure and geometry among individuals with a nondipping BP status. Ferrara et al (20) discovered that nondippers had significantly larger left atriums, greater atrial filling fractions and end-diastolic diameter indexes than normotensive controls. However, the nondipping group also had higher clinic and 24 h BP values than the dipping group (20). These findings imply that elevated 24 h BP levels among individuals with mild hypertension could contribute to early cardiac structural and functional changes. In addition, these early abnormalities may occur before development of LVH or systolic dysfunction. The data also suggest that structural cardiovascular abnormalities may develop faster among nondippers with high BP levels (20).

Changes in cardiovascular structure have also been observed among nondippers with BP averages comparable with those of dippers. In studies of individuals with untreated essential hypertension and similar 24 h BPs, a nondipping BP pattern was associated with significantly larger atrial dimensions (43,49), interventricular septum thickness and aortic root diameter (43), as well as left atrium passive and active emptying volumes and fractions (52). In addition, normotensive

individuals classified as nondippers displayed a higher prevalence of relative wall thickness and concentric hypertrophy independent of 24 h BP levels than their dipping counterparts (51). Results suggest that even among normotensives, the absence of a nocturnal BP decrease may occur before a BP increase and be linked with a poorer cardiovascular prognosis.

There is also evidence to support the association between female sex, BP variation and cardiac abnormalities. Schmieder et al (24) found that female hypertensive nondippers had significantly greater relative wall thickness, posterior wall thickness and concentric LVH than female hypertensive dippers and male hypertensive dippers and nondippers. Even after controlling for age, body weight and 24 h BP, the differences were maintained (24).

There has been controversy over the proposed relationship between blunted nocturnal BP decline and the prevalence of cardiac target organ damage. Many researchers have evaluated cardiac damage in untreated individuals with essential hypertension and found no significant differences between dippers and nondippers with respect to LV diameter (14,21), LV systolic and diastolic function (21), interventricular septum thickness, posterior wall thickness, relative wall thickness (14,45,56), LV concentric remodelling (45,56), LVH, LVM and LVMI (14,21,45,56), aortic distensibility (21) and atrial dimensions (56). However, in these studies, the classification of dipping status was based on a single ABPM session. Several studies have found dipping status to be reproducible approximately 60% of the time (43,57). Consequently, Cuspidi et al (43) examined nocturnal BP and cardiac target organ damage in untreated hypertensive patients with three different nighttime BP patterns (reproducible dipping, reproducible nondipping and variable dipping status). They found that individuals who had a variable dipping BP profile had consistently lower echocardiographic values (LV diameter, interventricular septum thickness, posterior wall thickness, relative wall thickness, aortic root diameter, left atrium diameter, LVM, LVMI and LVH) than reproducible nondippers, but consistently higher values than reproducible dippers. These results may partially explain the conflicting results obtained in previous studies in which patients were classified as dippers or nondippers on the basis of a single ABPM session (43).

CARDIAC FUNCTIONAL ALTERATIONS

Researchers have also investigated the potential relationship between individuals who have an attenuated dip in nocturnal BP and cardiac functional alterations. There is evidence to suggest that nondippers are at increased risk of cardiac dysfunction. Nondippers have been found to have increased levels of cardiac natriuretic hormones (51,58), lower LV ejection fraction (49,58), more ectopic beats (49,59), prolonged QT dispersion (60) and a prolonged QTc interval (61).

HEMODYNAMIC VARIATIONS

Vascular resistance

A few researchers have investigated the underlying hemodynamics of individuals with dipping and nondipping BP profiles. Schmieder et al (24) used 24 h ABPM to classify individuals with essential hypertension as dippers or nondippers. They observed no significant differences in systemic vascular resistance (SVR) between dippers and nondippers while at complete rest during a 1 h morning evaluation. Cavelaars et al (61) used continuous intra-arterial 24 h BP monitoring

and acceleration sensors to measure hemodynamic changes, physical activity and diurnal variation while individuals performed regular daily activities. Similarly, the authors observed no change in SVR in response to daytime supine rest between nondippers and dippers. However, researchers observed individuals displaying a nondipping BP pattern experienced an increase in SVR during the night, whereas the SVR in dippers did not change significantly from day to night. In a similar study, Takakuwa et al (62) also found that the SVR index increased at night among individuals with essential hypertension regardless of dipping status.

With the use of venous occlusion plethysmography, Pierdomenico et al (44) and Rizzoni et al (63) assessed forearm minimal vascular resistance in subjects with essential hypertension. Both authors found that vascular resistance was significantly higher among nondippers than dippers. These data suggest that nondipping may be associated with vascular structural changes.

Stroke volume and cardiac output

Takakuwa et al (62) studied hemodynamics and BP variations, and found that dippers and nondippers with essential hypertension had significantly different stroke index values. Dippers had a decrease in stroke index during the night, whereas nondippers exhibited an increase in stroke index during the night. Among the persons in the nondipping group, the cardiac index decreased significantly less than in the dipping group. Furthermore, Cavalaars et al (61) and Schmieder et al (24) discovered that nocturnal cardiac output was not significantly different between dippers and nondippers.

Pulse pressure

Pulse pressure is highly correlated with SBP (64). Considering that a nondipping BP profile is associated with a blunted fall in night-time BP, it is not unexpected for researchers to find higher pulse pressures independently associated with a nondipping BP profile among individuals with hypertension (65). Iida et al (66) observed a blunted reduction of night-time pulse pressure among individuals older than 60 years of age with essential hypertension and a nondipping BP pattern. They also found that the degree of night-time pulse pressure reduction had the strongest inverse correlation with LVMI among this cohort. This finding suggests that a blunted fall in nocturnal pulse pressure may be a risk factor for LVH, and it may be partially responsible for cardiovascular complications, especially among elderly patients with essential hypertension and a nondipping profile. Other investigators have discovered similar results among middle-aged, nondipping overweight men with essential hypertension (12).

CAROTID ARTERY ABNORMALITIES

It has been reported that hypertension is linked to carotid artery hypertrophy and atherosclerotic complications (67). The intima-media thickness of the common carotid artery and the presence of carotid plaques are considered to be markers of subclinical atherosclerosis (68) and independent predictors of future cardiovascular events (69). Inquiry into the development of target organ damage among individuals with essential hypertension and a nondipping BP pattern has led researchers to examine structural carotid alterations.

Intima-media wall thickness and atherosclerotic plaques

Studies have shown that individuals with essential hypertension and a blunted nocturnal decline in BP have a significantly greater prevalence of common carotid artery intima-media thickness (9,15,44), internal carotid intima-media thickness, carotid bifurcation intima-media thickness and average intima-media thickness (9) than their dipping counterparts. They also found a significant increase in the prevalence of atherosclerotic plaques in the carotid arteries of nondippers (9). Findings were independently associated with 24 h (9) and 48 h (15) BP averages, as well as age (9,15).

Not all researchers have found significant differences in carotid artery structure. However, they have observed higher nonsignificant trends for increases in common carotid artery intima-media thickness (18), common carotid artery diameter, carotid bifurcation intima-media thickness (14) and common carotid artery plaques (14,44). Cuspidi et al (15) found significant differences in carotid structure among reproducible nondippers. Although reproducible nondippers did have significantly greater cardiac abnormalities, there was only a trend for increased carotid structural abnormalities in the Cuspidi et al (43) study. However, differences may reflect the notion that cardiac damage occurs before extracardiac damage.

CEREBROVASCULAR DISEASE

Silent cerebral infarct

Hypertension is considered to be strongly associated with cerebrovascular damage (70). It is believed to contribute to the development of cerebrovascular small artery diseases such as lacunae, white matter lesions and periventricular hyperintensities (71). Additionally, researchers have found hypertensive nondippers to be at increased risk of stroke (46,72-74).

With the use of magnetic resonance imaging and 24 h ABPM, Kario et al (6) and Shimada et al (10) studied asymptomatic elderly hypertensive and healthy normotensive individuals. In both studies, researchers discovered that hypertensive nondippers had a significantly higher number of lacunae and advanced periventricular hyperintensities than hypertensive dippers (6,10). Kario et al (6) found that nondippers had a significantly higher average 24 h SBP, whereas Shimada et al (10) found that nondippers had a significantly higher average 24 h SBP and DBP. These findings can be interpreted in two ways. They suggest that over time, average BPs influence hypertensive target organ damage. However, they also suggest that advanced cerebrovascular disease may prevent BP from falling at night in effort to maintain cerebral perfusion pressure (10).

In addition, Shimada et al (10) observed no differences in cerebral abnormalities between the normotensive group and the dipping hypertensive group. Interestingly, hypertensive dippers had higher ambulatory BPs than normotensive individuals, and exhibited a larger decrease in BP from day to night than both the normotensive and hypertensive nondipping groups. Consequently, this finding suggests that a dipping profile may inhibit the development of cerebrovascular abnormalities (10).

Sander et al (75) evaluated the relationship between circadian BP variation and the extent of white matter lesions. The independent risk factors for white matter lesions were found to be a blunted fall in nocturnal SBP and age. However, in hypertensive and normotensive individuals, the extent of white matter lesions was most closely related to a lack of dipping in

nocturnal SBP, whereas the absolute level of BP was not significantly associated with the extent of white matter lesions. This finding emphasizes the importance of night-time BP levels and supports the hypotheses that white matter lesions are not the cause of an altered circadian BP pattern, given that only the systolic parameter of BP was associated with white matter lesions.

Arterial hypertension is associated with lacunar infarction (76). Yamamoto et al (71,77,78) prospectively studied cohorts of individuals with symptomatic lacunar infarcts using magnetic resonance imaging and ABPM. The authors (71) discovered that individuals who also had a nondipper BP profile had a poorer long-term prognosis, including a greater risk of developing further silent ischemic lesions and symptomatic ischemic strokes. However, nondippers also had higher average 24 h BPs.

As of yet, researchers have not been able to determine whether nondipping is the cause or consequence of cerebrovascular disease. Yamamoto et al (70) hypothesized that among individuals with cerebrovascular disease, attenuation of nocturnal BP dipping was related to the affected region of the brain rather than the extent of organ damage. They found that compared with controls, individuals with cerebrovascular disease (multiple lacunar infarcts, thalamic hemorrhage, pontine tegmentum infarction and pontine hemorrhage) had less of a nocturnal fall in BP. They also found that the degree of nondipping increased among individuals who had a second stroke or multiple lacunae (70). This finding suggests that nondipping may be distantly associated with the degree of hypertension-related vascular disease, but more closely related to cerebral lesion location, resulting in a functional impairment of the central autonomic nervous system (79). Thereby, this supports the view that nondipping is a consequence, as opposed to a cause, of cerebrovascular disease (70).

Cognitive impairment

Yamamoto et al (77,78) examined the cognitive impairment of individuals with symptomatic lacunar infarcts using the Clinical Dementia Rating Scale. After multivariate analysis, nondipping status remained significantly and independently associated with cognitive impairment disorder (78) and dementia (77), whereas high 24 h SBP was independently associated with vascular events (77). Other researchers have also examined the potential relationship between cognitive impairment and nondipping status. Tohgi et al (80) found that persons with Binswanger- or lacunar-type dementia also had a blunted decrease in nocturnal BPs compared with individuals with no evidence of vascular lesions. In addition, van Boxtel et al (81) evaluated the potential effects of dipping status on cognitive performance. After adjusting for age, sex and level of education in a sample of community dwelling residents aged 28 to 82 years, researchers found that mean daytime and night-time BP values were not related to cognitive scores. However, they did discover that a nondipping BP pattern was associated with lower scores on memory and sensory motor speed tests.

RENAL DAMAGE

The kidney is another organ often susceptible to vascular injury among individuals with hypertension. Several researchers have found microalbuminuria to be a strong and independent predictor of cardiovascular morbidity and mortality in nondiabetic hypertensive patients (82).

Microalbuminuria

Tsioufis et al (27) examined the relationship between microalbuminuria and the circadian BP variation in untreated persons newly diagnosed with essential hypertension. Nondippers were found to have higher rates of microalbuminuria than dippers. In addition, individuals with a nondipping profile also had higher 24 h BP averages. Other investigators have documented similar results in their cross-sectional studies (83,84,86).

There is evidence to support the hypothesis that people with hypertension who have an attenuated nocturnal fall in BP do not have higher rates of microalbuminuria. In a study of target organ damage among untreated essential hypertensives, Cuspidi et al (43) found similar results of microalbuminuria among individuals with a reproducible dipping pattern, a reproducible nondipping pattern or a variable dipping pattern. In addition, the three groups did not differ with regard to age, sex, body mass index, clinic BP, 48 h BP average and heart rate. Similar findings were discovered by other researchers examining individuals with essential hypertension (6,15,18) or normotension (51).

There are three possible explanations for the discrepancies in these findings. First, researchers that have discovered an association between nondipping and higher levels of microalbuminuria also have reported higher 24 h BP among the nondippers. Therefore, early renal vascular injury may be the result of increased pressure load on the blood vessels (27). Second, renal and cardiac injuries may not occur at the same time during the initial phase of hypertension (85). Last, microalbuminuria may simply be a less sensitive marker for target organ damage than other markers, such as echocardiographic LVH (15,85). Consequently, the association between nondipping and renal damage remains somewhat controversial.

CONCLUSION

ABPM has revealed that BP varies in a diurnal manner. The normal variation in BP is a dipping pattern characterized by a 10% to 20% dip in BP from day to night (3,42). Individuals with a less than 10% reduction in BP during sleep are described as having a nondipping BP pattern (2,24,38). There is a growing body of evidence suggesting that nondipping is associated with a greater risk of target organ damage and a worse cardiovascular prognosis (38). A better understanding of the importance of the circadian variations of BP may help to identify those at higher risk of cardiovascular morbidity and mortality, and may lay the foundation for interventions to prevent or treat alterations in diurnal BP patterns.

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