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The role of ambulatory blood pressure monitoring in chronic and end-stage renal disease

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In the past 30 years or so, the introduction of 24-h ambulatory blood pressure monitoring (ABPM) has enabled a more comprehensive estimate of a patient's true blood pressure (BP) and its changes. Although this tool has been used in the general population for the diagnosis of white coat hypertension, its role in the clinical management of patients with chronic and end-stage kidney disease is less well defined. In patients with kidney disease, routine clinic and dialysis center BP measurements may be poor indicators of BP control. Loss of the normal nocturnal decline in BP is also common. Moreover, there is increasing evidence that this loss, which ABPM alone can detect, is associated with poor renal and cardiovascular outcomes. To slow the progression of renal disease and lessen cardiovascular morbidity and mortality in patients with kidney disease, tight BP control is needed. However, the traditional methods of measuring BP intermittently in the medical setting may fail to provide an accurate picture of BP load. Ambulatory or some form of home BP monitoring should be more widely adopted in patients with chronic and end-stage renal disease.

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Hypertension and chronic kidney disease (CKD) are inextricably intertwined: the majority (70%) of individuals in the general population who have an elevated serum creatinine are hypertensive,[1] and hypertension is both a cause and a consequence of CKD. Most patients with CKD die of the same cardiovascular diseases that afflict hypertensives without CKD, and renal function is an independent predictor of cardiovascular events.[2,3] The importance of hypertension as a determinant of the progression of CKD has been recognized by official guidelines such as the Joint National Committee 7,[4] the 2003 European Society of Hypertension–European Society of Cardiology Hypertension Guidelines,[5] and the National Kidney Foundation Working Group Report on Hypertension and Diabetes.[6] Aggressive blood pressure (BP) control is advised with some guidelines recommending that BPs be targeted to less than 130/80 mmHg, or 10 mmHg lower than in most other hypertensive populations.[4–6] Yet, despite this recognition that treating hypertension can prevent many of its complications, BP remains poorly controlled in many patients with renal insufficiency.[7]

In the past 30 years or so, the introduction of 24-h ambulatory blood pressure monitoring (ABPM) has enabled a more comprehensive estimate of a patient's true BP and its changes. A substantial number of prospective studies have shown that ABPM predicts cardiovascular events better than clinic-based readings, and also correlates more closely with target organ damage. Whereas diurnal variation in BP, characterized by a nocturnal 'dip' and morning surge, has long been recognized, the advent of ABPM has also allowed greater investigation into its prognostic significance. Loss of the nocturnal decline in BP has been described in hypertensives, diabetics, African Americans, and in patients with sleep apnea and renal disease. In cross-sectional studies and in a growing number of prospective trials, this loss, which ABPM alone can detect, is associated with target organ damage and adverse cardiovascular events.

ABPM has also been used to better define the relationship between BP, target organ damage, and outcomes in patients with chronic and end-stage renal disease (ESRD). Hypertension in these patients shows some distinguishing features on ABPM. First, the prevalence of non-dipping (elevated nocturnal BP) is very high. Second, in patients on dialysis, changes in intravascular volume in the intra- and interdialytic

period may result in marked swings of BP. Third, as in patients without CKD, discrepancies between clinic and home BP readings are common. The traditional methods of measuring BP intermittently in the medical setting may thus fail to provide an accurate picture of BP load, and hence result in sub-optimal treatment. In this review, we will examine the relationship between office and ambulatory BP and their prognostic significance in patients with CKD and ESRD. We will also review recent insights gained into the prevalence, pathogenesis, and prognostic significance of abnormalities in the diurnal variation of BP in this population.

RELATIONSHIP BETWEEN OFFICE AND AMBULATORY MEASUREMENTS IN PATIENTS WITH CKD

It is well established in studies of patients without CKD that conventional clinic BP measurements may significantly under- or overestimate the true BP.[8–10] Although less well described, discrepancies between clinic and ambulatory BPs have also been reported in patients with CKD. Thus, in a study of 232, mostly male patients with CKD, approximately 30% had clinic BPs that were higher than ABP measurements, whereas 28% had clinic BPss that underestimated ABP.[11] White coat hypertension, defined by hypertension on clinic measurements and normotension on ABPM, has been reported in approximately 9% of diabetic patients with microalbuminuria or macroalbuminuria.[12] In pediatric patients with treated hypertension and CKD, white coat hypertension, defined by the 95th percentiles for clinic and ABP readings, was found in 17% of patients. Masked hypertension, where clinic BP underestimates the true BP, was reported in another 5%.[13]

PROGNOSTIC SIGNIFICANCE OF OUT OF OFFICE MEASUREMENTS

In numerous prospective studies, ABPM has proven to be superior to clinic readings in predicting cardiovascular outcomes. This superiority is due, in part, to ABPM's ability to identify subgroups of patients who are at higher and lower risk of cardiovascular events than would be predicted by their clinic measurements. ABPM is perhaps most widely used in clinical practice for the diagnosis of white coat hypertension. In comparison to normotensive patients, patients with white coat hypertension do not appear to be at greater risk of cardiovascular morbidity.[8] Similar to patients with white coat hypertension, treated hypertensives with elevated clinic pressures but controlled ambulatory pressures have a lower risk of adverse cardiovascular events than patients with true refractory hypertension.[14,15] In contrast, there is evidence that masked hypertension is associated with worse cardiovascular outcomes both in treated hypertensives and patients found to be normotensive on clinic measurements.[14]

Similar studies examining the prognostic significance of ABPM in patients with CKD are much needed. However, there is evidence that home BP monitoring is superior to clinic measurements in predicting outcomes in this population. In a prospective cohort study of veterans with CKD, home BP readings averaged over 1 week were superior to standardized and routine clinic BP measurements in predicting the composite end point of ESRD or death. During a median follow-up of 3.5 years, 22% of patients with masked hypertension (defined by routine measurements) developed ESRD, whereas no patient with white coat hypertension (defined by standardized or routine measurements) progressed to ESRD. Even after adjustment for the standardized clinic systolic BP and other risk factors, including proteinuria, age, and estimated GFR, home BP readings were found to provide additional prognostic information on the risk of progression to end-stage disease.[16]

ABPM AS A PREDICTOR OF TARGET ORGAN DAMAGE IN CKD

Additional support for ABPM's role in the management of CKD patients comes from small, cross-sectional studies showing that ambulatory BPs correlate more closely than clinic measurements with BP-related target organ damage. In these studies, ABPM has either shown a stronger correlation with left ventricular hypertrophy (LVH) than clinic-based readings or has predicted LVH when no association could be shown with clinic measurements. Twenty-four hours systolic BP but not clinic readings predicted left ventricular mass index (LVMI) in a study of 29 pediatric CKD patients.[17] In 26 normotensive subjects with polycystic kidney disease and relatively preserved renal function, 24-h systolic BP was the only variable predictive of LVMI on multiple regression analysis.[18] Finally, ABPM showed a stronger correlation with LVMI than single casual clinic measurements in a study of 85 CKD patients without a history of diabetes or vascular disease.[19] In addition, ABPs have been reported to correlate more closely with proteinuria. In a study of 232 veterans with CKD, Agarwal and Andersen[20] found that ABPM had a stronger correlation with proteinuria than home, standardized, or single routine clinic readings taken by nurses using an automated device. These studies, in which one or a few clinic readings were compared with ABPM, suggest that ABPM gives a better measure of BP control. However, whether or not ABPM correlates better with target organ damage depends to a large extent on how many clinic BP readings are used in the comparison,[21] and additional studies are needed in patients with CKD to determine the predictive advantage of ABPM in comparison to a larger number of clinic readings.

RELATIONSHIP BETWEEN DIALYSIS CENTER MEASUREMENTS AND ABPM

Hypertension's contribution to cardiovascular morbidity and mortality has been difficult to demonstrate in dialysis patients.[22] A paradoxical relationship between increased mortality and lower BPs has been described and can be attributed in part to an association between lower BP and impaired cardiac function.[23,24] However, dialysis center measurements, used in many studies to explore the relationship between hypertension and cardiovascular events, can be

poor predictors of interdialytic BP control. This failure of dialysis center measurements to accurately characterize BP has likely also made it more difficult to define the prognostic significance of hypertension in this population.

In hemodialysis patients, dialysis center measurements are often poor indicators of interdialytic BP control. Of 71 dialysis patients in whom 44-h interdialytic BP monitoring was compared with dialysis center measurements taken by a nurse, 43% of patients classified as hypertensive by predialysis systolic BP were normotensive on ABPM, while 25% of patients classified as normotensive by predialysis systolic BP were hypertensive.[25] Whereas predialysis BP measurements tend to overestimate BP load, the relationship between post-dialysis readings and BP control appears to be more variable.[26,27] In pediatric patients on hemodialysis or peritoneal dialysis, casual BP measurements have also been shown to be poor predictors of home BP levels.[28,29]

Because so many BP measurements are made during dialysis, studies have attempted to determine if one reading in particular or an average of readings can provide a more accurate assessment of interdialytic BP. Mitra *et al.*[30] compared measurements made on arrival, 10 min after resting in a quiet room, at the onset of dialysis, end of dialysis, and 20 min post-dialysis, with average 48-h ABP readings in a group of 40 stable dialysis patients and found that the 20 min post-dialysis BP reading was most representative of BP control in the interdialytic period. Conlon *et al.*[31] averaged dialysis center readings from multiple visits and showed that predialysis BPs averaged over 12 treatment sessions showed a strong correlation with ABPM. Based on the results of 48-h ABP recordings in 36 hemodialysis patients, Coomer *et al.*[27] developed a model to predict mean BP based on age, sex, race, and pre- and postdialysis BP. Finally, Agarwal and Lewis[26] compared ABPM with a 2-week average of dialysis center readings in 70 dialysis patients and found that a 2-week average cutoff predialysis BP of 150/80 mmHg or higher had 80% sensitivity and 67% specificity to detect interdialytic hypertension as defined by an average ABP of 135/85 mmHg or higher. Although these methods can be used to obtain a better estimate of interdialytic control, they cannot reliably determine BP in any individual patient. Home BP monitoring and standardized predialysis BP measurements, however, can aide in the assessment of BP control. In a prospective cross-sectional study, home BPs averaged over 1 week were shown to be superior to routine dialysis center measurements averaged over 2 weeks in predicting hypertension on 44-h ABPM (defined as an awake BP greater than or equal to 135/85 mmHg). Standardized predialysis BP averaged over 2 weeks, although time intensive for the dialysis staff, had similar predictive ability as home measurements.[32]

REASONS FOR POOR CORRELATION BETWEEN DIALYSIS AND AMBULATORY BPs

The poor correlation between dialysis and ABPM readings is explained in part by changes in BP that occur in the interdialytic period, which cannot be captured by measurements made in the dialysis center. Santos *et al.*[25] studied BP in the interdialytic period in 71 stable hemodialysis patients and found a statistically significant increase in average daytime and night time BPs from the first to second day of the interdialytic period. In a study of 20 mostly black hemodialysis patients, Agarwal[33] reported a similar pattern of BP changes and also observed a decrease in BP in the period after dialysis, a finding that has been described by others.[27,33,34] In addition to these likely volume-induced changes in BP, improper measurement technique and a white coat effect may also contribute to the poor correlation between dialysis center and ABP readings. In a cross-sectional study of 270 hemodialysis patients, routine dialysis center measurements were 14.3/7 mmHg higher than those taken by a nurse following standard American Heart Association guidelines for BP measurement, and in 53% of patients, routine measurements were more than 10 mmHg greater than standardized readings (Figure 1).[35] A marked difference in BP ($>20/10$ mmHg) between ABP readings taken in the 6 h predialysis and BP measured immediately after arrival was also found in 41% of patients in a study by Mitra *et al.*[30] This difference persisted in seven of 15 patients after repeat BP measurement after 10 min in a quiet room, illustrating both the importance of appropriate BP measurement technique and suggesting a possible white coat effect. Finally, the practice of withholding BP medications on the day of dialysis may also contribute to the poor correlation between dialysis-based readings and ABPM. Studies specifically addressing this issue, however, are currently lacking.

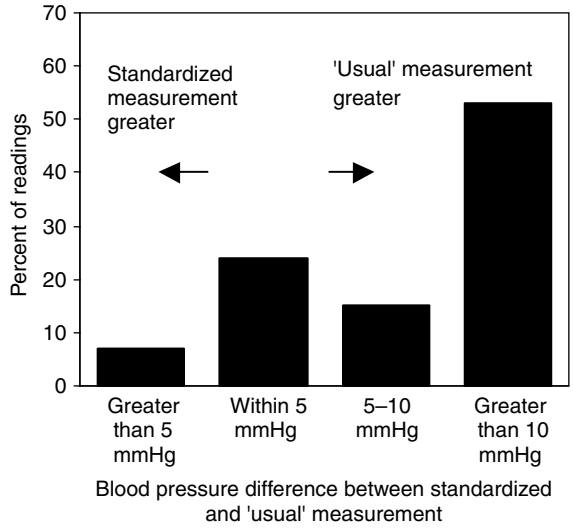

Figure 1 | Systolic BP difference between standardized and 'usual' dialysis center BP measurements in 270 hemodialysis patients. (Adapted from Rahman *et al. Am J Kidney Dis* 2002; **39**: 1226–1230.)

PROGNOSTIC SIGNIFICANCE OF ABPM IN ESRD

As in patients with CKD, there is evidence from cross-sectional studies that ABPM is superior to dialysis center

measurements in predicting target organ damage in patients with ESRD. In a small study of patients on peritoneal and hemodialysis, ABP but not office BP was associated with LVM.[36] LVMI and LV wall thickness were more strongly correlated with ABP than the average of two casual dialysis center measurements in a study of non-diabetic hemodialysis patients.[37] In perhaps the largest study to date, Agarwal *et al.*[38] reported that 44-h ABPM and home BP monitoring, although weak determinants of LVH, were superior to a 2-week average of standardized and routine dialysis center measurements in 140 chronic hemodialysis patients. However, the predictive advantage of ABPM for LVH was lost in two studies in which comparisons were made with a greater number of dialysis center readings. The correlation between LVH and BP was similar using ABPM and an average of 12 standardized predialysis measurements in a study of 35 stable hemodialysis patients.[31] In a second study of 64 non-diabetic hemodialysis patients without heart failure, including the results of 24-h ABPM to a model already containing the average of 12 routine predialysis measurements did not add significantly to the correlation with LVMI.[39]

In addition to these studies, three prognostic studies have also used ABPM to explore the relationship between BP and cardiovascular morbidity and mortality in hemodialysis patients. The three studies were small, and although all showed an association between ABPM and outcome, none reported a relationship between outcome and predialysis measurements.[40–42] The exact components of ABP that have been predictive of outcome have varied, although one observation has emerged consistently: loss of the normal nocturnal decline in BP carries a poor prognosis. These studies highlight the prognostic significance of disruptions in diurnal variation in patients with CKD and ESRD, the topic of the remainder of this review.

DIURNAL VARIATION IN BP AND ITS DISRUPTION IN KIDNEY DISEASE

In the general population, BP falls on average by 10–20% during sleep, a phenomenon referred to as 'dipping'. In about 25% of healthy subjects, and in certain disease states, however, a loss of diurnal variation in BP (Figure 2) has been reported (non-dipping). Non-dipping is particularly common in both children and adults with CKD, and an inverse relationship between GFR and the prevalence of non-dipping has been described (Figure 3).[17,43] Although the reported prevalence of non-dipping in adults with CKD varies, rates of 50% or higher have been observed at the earliest stages of disease, whereas rates of more than 80% have been observed in patients on dialysis.[43]

PATHOGENESIS OF THE DIMINISHED NOCTURNAL FALL IN CKD AND ESRD

Whether or not the diseased kidney itself directly causes abnormalities in diurnal variation is unknown and studies attempting to answer this question have, to date, produced somewhat mixed results. Portaluppi *et al.*[44] performed ABPM following the discontinuation of BP medications in 30 non-diabetic CKD patients and 30 controls matched by sex, age, and mean 24-h BP. Although controls exhibited an average nocturnal *decline* in systolic and diastolic BP, nocturnal systolic and diastolic BP *increased* in patients with CKD, suggesting a role for the kidney in the pathogenesis of this disorder. In a study by Baumgart *et al.*,[45] 61 patients with CKD, ESRD on dialysis, or post-renal transplant were matched by age, sex, systolic office BP, and use of antihypertensive medication to 61 controls with normal renal function. Compared with controls, the nocturnal decline in BP was diminished in all patient groups with renal disease. Taking a somewhat different approach, van de Borne *et al.*[46] attempted to isolate the role of the diseased kidney by performing ABPM in a small group of dialysis patients carefully selected to exclude many of the causes and complications of kidney disease known or hypothesized to affect diurnal variation. In this trial, dialysis patients with diabetes, hypertension, an inability to ambulate, heart failure, cardiac arrhythmias, psychiatric diseases, irregular sleep–wake schedule, transmeridian travel, past or present use of erythropoietin, and use of any medication other than calcium and vitamin D were excluded and hemodialysis patients were matched with controls by age, sex, and casual systolic

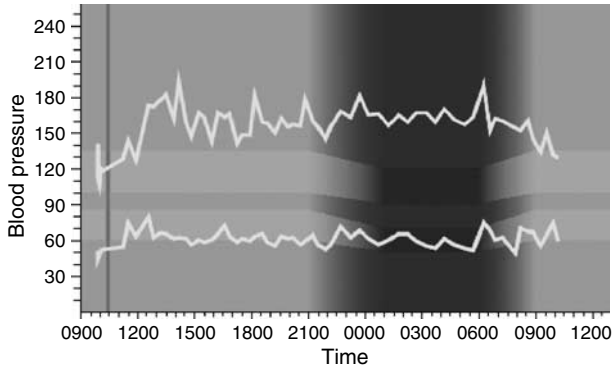

Figure 2 | A non-dipping pattern of BP is seen on ABPM in a CKD patient with masked hypertension.

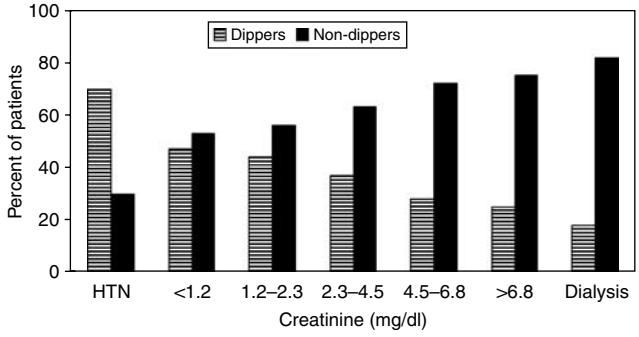

Figure 3 | Prevalence of non-dipping in essential hypertension and at various stages of renal dysfunction. (Adapted from Farmer *et al*. *Nephrol Dial Transplant* 1997; 12: 2301–2307.)

BP measurement. In contrast with the findings of others, van de Borne *et al.* showed that in this select group of ESRD patients, the nocturnal decline in BP was in fact preserved.

In non-CKD patients, an association between non-dipping and volume expansion has been described. In salt-sensitive subjects, who tend to be non-dippers, the nocturnal fall in BP is significantly increased by a low sodium diet.[47] Diuretics can also restore dipping status, as evidenced by an increase in the nocturnal fall in systolic and diastolic BP following thiazide diuretic administration in patients with essential hypertension.[48] In ESRD patients, a statistically significant increase in the night/day ratio[49] and a statistically significant decrease in the awake–sleep difference in BP[30] has been reported from the first to the second day after dialysis. In a study of 71 unselected hemodialysis patients, a trend toward an increased prevalence of non-dipping was also seen that did not reach statistical significance.[25] However, switching patients from thrice weekly to daily dialysis, although effective in reducing BP and extracellular body water, was not found to affect dipping status.[50] A high prevalence of non-dipping has also been reported in patients receiving long, slow home hemodialysis thrice weekly.[51] Hence, factors other than volume likely also play a role.

Abnormal sympathetic nervous system (SNS) activity has been reported to contribute to the non-dipping pattern of BP in non-CKD patients. In healthy subjects, SNS activity falls during sleep, as evidenced by a decrease in urinary and plasma catecholamine levels and in muscle sympathetic nerve activity (non-REM sleep).[52–55] In non-dippers without CKD, this nocturnal fall in urinary epinephrine and norepinephrine levels is diminished.[56] As increased SNS activity has been described in patients with kidney disease,[57] it is possible that loss of diurnal variation in this population is due, in part, to abnormal SNS activity as well.

In addition to these factors, many others, known to cause and or arise as a complication of kidney disease, may also play a role in this diminished nocturnal decline in BP seen in patients with renal disease. Diabetes and autonomic dysfunction, operating through increased volume, nocturnal SNS activity, or via other mechanisms, are associated with non-dipping and are prevalent in the CKD and ESRD populations. Low levels of physical activity during the daytime[58] and poor sleep quality[59] have been linked to a diminished night time fall in BP. Furthermore, sleep apnea, which has been reported in 15% of an unselected group of dialysis patients and at higher rates in dialysis patients complaining of sleep problems, is associated with nocturnal hypertension.[60,61] A smaller decline in nocturnal BP has also been described in treated versus untreated hypertensives, possibly owing to the waning effect of antihypertensive medications taken in the morning.[62]

PROGNOSTIC SIGNIFICANCE OF THE NON-DIPPING PATTERN IN CKD AND ESRD

A loss of diurnal variation in BP has been associated with a poor renal prognosis. In cross-sectional studies of patients with established kidney disease, a diminished sleep-to-wake ratio of BP has been associated with greater proteinuria and diminished renal function.[20,43,63] These observations have been confirmed in some longitudinal trials. In normotensive type I diabetics, the absence of a normal nocturnal fall in systolic blood predicted the later development of micro-albuminuria.[64] In a 3-year longitudinal study of 28 non-dippers and 20 dippers with hypertension and CKD, non-dipping at baseline was predictive of a faster decline in renal function and a greater increase in proteinuria.[65] In a second study of 95 patients with non-diabetic kidney disease, non-dipping at baseline was also associated with a faster rate of progression of renal disease during 3 years of follow-up.[66] However, in this study non-dipping was associated with a higher creatinine and greater proteinuria at baseline as well, making it hard to determine what role, if any, non-dipping played in the progression of renal disease.

Loss of the nocturnal decline in BP has also been linked to LVH, adverse cardiovascular outcomes, and all-cause mortality in patients with ESRD. In a study of 59 hemodialysis patients, a correlation was found between the day/night ratio and LVMI,[67] whereas in peritoneal dialysis patients an elevated night time BP load was the sole predictor of LVH.[68] As described, three prospective studies have reported an association between loss of the nocturnal decline in BP and adverse outcomes. In a cohort of 80 dialysis patients without a history of congestive heart failure or significant cardiovascular disease, non-dipping status was associated with an increased adjusted hazard ratio for cardiovascular morbidity and mortality.[41] A second study of 57 hypertensive ESRD patients without a history of systolic cardiac dysfunction or valvular disease found that after controlling for age, sex, and cardiovascular history, an elevated nocturnal blood systolic BP was associated with increased cardiovascular mortality.[40] Finally, in a study by Tripepi *et al.*,[42] 168 dialysis patients without a history of diabetes, cardiovascular disease, or clinical evidence of heart failure were followed for 38 months. In a multi-regression analysis model not including LVH, an association between the highest night/day BP tertile and increased cardiovascular and all-cause mortality was found. In contrast, the predialysis BP averaged over 1 month did not predict events.

WHY SHOULD NON-DIPPING INCREASE RISK?

Why this loss of nocturnal variation carries such a poor prognosis is unknown. In the study by Tripepi *et al.*,[42] 24-h systolic BP was also greater in subjects with higher night/day systolic ratios. Hence, some of the increased risk associated with elevated nocturnal pressures may simply be due to the greater 24-h BP load associated with this elevation. It is also possible that the absence of a nocturnal decline in BP is not itself a cause of adverse outcomes, but is instead just a marker of sicker patients. As described above, less daytime activity, poorer sleep quality, sleep apnea, autonomic dysfunction, volume overload, and the use of antihypertensive medications have all been associated with loss of diurnal variation in

BP. Finally, although dipping status may be an important predictor of outcome, it is interesting to note that dipping status itself is not very reproducible. In 21 hemodialysis patients in whom dry weight and BP medications were held constant, up to 43% changed their dipping status from the first to second interdialytic day, whereas up to 38% changed dipping status on repeat testing at least 4 weeks later.[69] In patients with polycystic kidney disease and mild renal impairment, only approximately 40% of patients maintained their initial dipping status on repeat testing, although dipping status in this study was divided into quartiles and BP medications changes that occurred during the course of the study may have confounded these results.[70] Such findings, coupled with the observation that the correlation between end-organ damage and non-dipping is strongest in patients with a reproducible non-dipping pattern,[71] suggesting that in trials and in clinical practice, dipping status should probably be defined by or confirmed with repeat testing.

RESTORING THE NOCTURNAL DIP IN BP

Just as BP can be lowered in patients with CKD, there is some evidence that dipping status can be restored. Changing the timing of administration of the calcium channel blocker, isradipine, from morning to night, improved the nocturnal decline in BP in patients with kidney disease.[72] An increased nocturnal decline in systolic BP was seen at 6-month follow-up in hypertensive children with kidney disease started on an angiotensin converting enzyme inhibition.[63] As noted above, diuretics and a low sodium diet selectively lower nocturnal BPs in non-CKD patients with salt-sensitive hypertension, an effect that may also occur in patients with CKD. There is also evidence that kidney transplantation restores the nocturnal decline in BP. In a cross-sectional study of 45 renal transplant patients, Gatzka *et al.*[73] found that with increasing time from transplant, the number of patients displaying a normal nocturnal decline in BP increased. The use of cyclosporine post-transplant, however, has been associated with worsening nocturnal pressures. In a cross-sectional study of 46 renal transplant patients randomly assigned to cyclosporine or azithioprine 1 year post-transplant, nocturnal pressures were significantly higher in cyclosporine-treated patients.[74] An improvement in overall BP control and nocturnal BPs was also reported in 18 renal transplant patients randomized to conversion from a cyclosporine to azathioprine-based immunosuppressive regimen.[75]

CONCLUSION

In the past 30 years or so, the introduction of ABPM has enabled a more comprehensive estimate of a patient's true BP and its changes. Although ABPM is a useful tool for studying the relationship between BP and outcomes in patients with kidney disease, it has been underutilized. The relationship between BP and mortality in patients on hemodialysis remains a major unresolved question. Studies using conventional BP measurements have produced conflicting results and future studies examining this relationship or assessing the impact of treating hypertension in patients on hemodialysis should use either ambulatory or home BP monitoring. The prognostic significance of ambulatory and home BP readings in patients with CKD has not been adequately studied, and their role in directing antihypertensive therapies also needs to be addressed. Finally, studies are needed to determine if restoration of the nocturnal decline in BP will translate into improved renal and cardiovascular outcomes above and beyond those obtained from reducing the overall BP load. In the meantime, ambulatory or some form of home BP monitoring should be used to obtain a more accurate picture of BP control in patients with chronic and ESRD. Clinic BPs frequently under- or overestimate the true BP in CKD patients and dialysis center BP measurements, although widely used to guide therapy, are poor indicators of interdialytic BPs. Tight BP control is needed to limit the progression of renal disease and lessen cardiovascular morbidity and mortality in patients with kidney disease. However, to achieve this goal, BP must be accurately measured. Ambulatory or some form of home BP monitoring should be more widely adopted in patients with chronic and ESRD.

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