Detection of non-dipper hypertensives in clinical practice: does it really matter?

Sir,

In their recent Editorial Comment on night-time blood pressure (BP) in diabetic patients, Schernthaner et al. [1] argue that 'ambulatory BP measurement is indispensable for optimizing antihypertensive treatment in diabetic patients with nephropathy' and that 'the evaluation of night-time BP permits a much more focused antihypertensive treatment' in these patients. We feel that at present there is absolutely no evidence to support the usefulness of the assessment of the diurnal BP profile in the management of any subgroup of hypertensive patients, and more importantly, there are several good reasons to avoid it in general practice.

First, there are several difficulties in the correct classification of hypertensive individuals as dippers and non-dippers. In the analysis of ambulatory BP data, fixed day and night periods are routinely applied, while actual patient reported sleeping hours are used only occasionally [2–4]. It has been shown however, that analysis of ambulatory BP profiles using arbitrary day–night-time schedules underestimate the nocturnal BP drop, resulting thereby in an overestimation of the prevalence of non-dippers [3,4]. Until ambulatory equipment manufacturers improve application software to allow for individualized analysis of ambulatory BP profiles, the interpretation of ambulatory BP data using individual patients reported sleeping hours is not feasible because it requires additional effort and time [4].

Another important issue is the poor reproducibility of the nocturnal BP fall. Several studies have shown that the pattern of diurnal BP profile is less reproducible than mean ambulatory BP values [5–7]. Mansoor et al. [5] studied 27 untreated hypertensives with ambulatory BP monitoring on two occasions, separated by 1.5–18 months. Among subjects classified as dippers in the initial assessment, 20% changed to a non-dipper profile in the second assessment and half of non-dippers in the initial assessment switched to dippers in the second assessment. We also studied 78 untreated hypertensives with ambulatory BP monitoring on two occasions, 2 weeks apart [8] and found disagreement between the initial and the second assessment in the dippers–non-dippers classification in 38 and 22% of cases, for systolic and diastolic BP dip respectively (unpublished data). These findings indicate that the dipper–non-dipper BP profile may change in individual patients over time and therefore, a single 24-h ambulatory BP monitoring cannot characterize the diurnal BP profile of the individual patient [6]. Moreover, the lack of bimodality in the distribution of the nocturnal BP fall suggests that the dippers–non-dippers classification, by using the 10% cut-off difference, is arbitrary [4,6]. It is noteworthy that recent guidelines for the management of hypertension [9,10] while confirming the importance of ambulatory BP monitoring as a research tool for the investigation of the clinical relevance of phenomena, such as BP variability and nocturnal fall, propose its use in clinical practice almost exclusively in patients with suspected white-coat hypertension and in those with apparent drug resistance.

An additional reason to argue against the opinion expressed in the Editorial Comment is the fact that so far no controlled prospective study has addressed the issue whether non-dippers should be treated differently, i.e. at an earlier stage or preferably with drugs, such as \( \beta \)-blockers, angiotensin converting enzyme inhibitors or calcium entry blockers, known to accentuate night dipping [11]. Medicine should be based on evidence rather than hypotheses, and hard evidence is now available. In the two recently published outcome studies [12,13] that established the importance of the aggressive control of BP in diabetic patients with hypertension, the decision to treat hypertension was exclusively based on clinic BP measurements, as was the assessment of the effects of treatment. This was also the case in the MDRD study [14] that established the treatment target-BP in patients with renal disease. It should be emphasized that in both Europe and the USA, the control of hypertension is poor, with only 15–27% of the hypertensives having BP levels equal or below 140/90 mmHg [15]. In order to achieve a clear mortality benefit, physicians should draw their attention to the aggressive control of clinic BP and more aggressively so in hypertensives with diabetes and/or renal disease. Until more information on the optimal management of non-dipper hypertensives is available, indiscriminate use of ambulatory BP monitoring in clinical practice will inevitably lead to deviation from this main goal and will add considerably to both confusion and cost.

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2. Pickering TG. How should the diurnal changes of blood pressure be expressed? Am J Hypertens 1995; 8: 681–682
rhabdomyolysis

Cocaine-induced acute renal failure has been reported in association with rhabdomyolysis [1,2]. However, only one Letter which is not seen infrequently in the nephropathic diabetic microvascular complications in type 2 diabetes: UKPDS 38. UK common, or whether even a paradoxical increase occurs 6. Tight blood pressure control and risk of macrovascular and night-time decrease in blood pressure fails to occur, as is 1998; 351: 1755–1762 are to be expected, given the fact that an attenuated fall of 14. Peterson JC, Adler S, Burkart JM et al. Blood pressure control, factors? Fluctuations in the dipper status Rudzki H. Nephropathy of type 2 diabetes: evidence for heredit-

Sir, We thank Drs Stergiou and Mountokalakis for their interest in our recent Editorial Comment [1]. We certainly agree that in the age of evidence based medicine one should try to obtain evidence whenever this is possible, but certainly—in the absence of controlled evidence—it does not hurt to use clinical common sense as well. We presume that the authors do not hesitate to perform renal biopsy in a proteinuric patient with microhaematuria although there is no controlled evidence that knowing the result affects outcome; nevertheless renal biopsy definitely remains good clinical practice.

We are certainly aware of the difficulties of the correct classification of the dipper status, and the artefact resulting from differences in the time when subjects are asleep. The latter is absolutely elementary and was well taken care of in past studies of our own [2,3] and this caveat should be known to the readers of NDT.

We are uncertain whether the variable reproducibility of the dipper status in patients with essential hypertension is really relevant for the diabetic patient with nephropathy in whom such reproducibility has not been studied systematic-

ally in a similar fashion. Fluctuations in the dipper status are to be expected, given the fact that an attenuated fall of blood pressure during night-time is to a large extent explained by changes in volume [4] which is notoriously variable in the nephropathic diabetic patient.

It would be decidedly wrong to abstain from ambulatory blood pressure measurements because of uncertainties about categorization, since the main point is to find out whether a night-time decrease in blood pressure fails to occur, as is common, or whether even a paradoxical increase occurs which is not seen infrequently in the nephropathic diabetic patient.

We are somewhat surprised by the argument that the studies documenting the importance of aggressive blood pressure control in diabetic patients with hypertension used clinic blood pressure [5]. For logistic reasons these studies were indeed based on clinic blood pressure; but what evidence is available that outcome might not have even be better if ambulatory blood pressure measurement had been used?

Undoubtedly, controversy is the pepper and salt of scientific progress, but we feel that progress comes not from polemics but from careful studies, and such a study is currently in the planning stage. We shall be happy to share our experience with Dr Stergiou once it is available.

3. Strojek K, Greszczak W, Morawin E, Adamski M, Lacka B, Rudzki H. Nephropathy of type 2 diabetes; evidence for heredit-

ary factors? Kidney Int 1997; 51: 1602–1607