In this issue of Diabetes Care, Berlowitz et al. (1) report that diabetic subjects with hypertension are receiving less intensive antihypertensive therapy than patients without diabetes. This report comes from a group of investigators well known for their interest and publications in the area of proper blood pressure control (2). Using a threshold for hypertension control of $\leq 140/90$ mmHg, they found that 73% of diabetic hypertensive subjects did not reach this target blood pressure. Using validated study methods, the authors determined that one reason for this poor control is that clinicians are not appropriately increasing antihypertensive medications, despite knowing that the levels of blood pressure are still high and above published target goals. Focused concern with glycemic control in diabetes has always been given as one reason for ignoring good overall medical control in the diabetic patient. However, Berlowitz et al. (1) show that this is not the case for why clinicians do not increase antihypertensive medications in a given visit to reach target blood pressure.

There is also the question of why this therapeutic gap in blood pressure control in diabetic compared with nondiabetic individuals should exist, given the numerous studies showing that tight blood pressure control in diabetic subjects markedly reduces the risk for cardiovascular disease and stroke (3–6). Also, there are numerous recently published guidelines, including those of the sixth report of the Joint National Committee (7) and the World Health Organization/International Society for Hypertension (WHO/ISH) (8), that clearly define target blood pressure levels for diabetic patients as $\leq 130/85$ mmHg. Is it possible that there really is a lack of concern or awareness of published guidelines by clinicians who treat hypertension in diabetic subjects? As diabetic subjects were less likely to receive an increase in antihypertensive medications at a given visit than hypertensive subjects without diabetes, one could advance the thought that those clinicians could either be overestimating their level of care or that they may lack proper training on antihypertensive therapy in diabetes (2). An interesting interpretation for this behavior, as noted by the authors, is described by Phillips et al. (9) as “clinician inertia.” This term is especially disturbing to those involved in teaching medical concern and care toward patients with chronic and difficult medical diseases. It is hoped that this situation is not the only or major reason for poor control in chronic medical disease states such as hypertension and diabetes, and that other factors explain the treatment gap.

Hypertension in diabetic subjects is usually difficult to treat. The authors refer especially to a group of obese hypertensive diabetic patients whose hypertension may be resistant to therapy. Additionally, cuff size differences can lead to large errors in blood pressure measurement accuracy, and there was no indication in this study that cuff size was corrected for arm circumference. It is also known that diabetic subjects with hypertension have a large variation in minute-to-minute blood pressure levels, so that multiple readings at a visit or ambulatory blood pressure measurements are preferred methods to diagnose and treat hypertension. The investigators state that to correct for this possibility they undertook multiple measurements of blood pressure at each visit, but then went on to acknowledge that in 80% of visits, only one blood pressure reading could be recorded.

Another area of concern is the diabetic subject with nephropathy. It is surprising that this subgroup was not identified, as it can be detected easily by serum creatinine and urinary protein excretion. In most diabetic clinics, a substantial number of diabetic subjects of comparable age ($65.9 \pm 8.3$ years) will have a serum creatinine $\geq 1.5$ mg/dl. Hypertension in this group will be difficult to treat, and perhaps it is here where the clinician might defer from increasing medical treatment owing to the fact that in a non-nephrologic environment, an increase in serum creatinine may follow blood pressure control and can be perceived as a deleterious effect of the antihypertensive agent on kidney function. In the same setting, fears of hyperkalemia sometimes prevent the clinician from adding a needed agent, such as an angiotensin-converting enzyme inhibitor (ACEI) or an angiotensin II receptor blocker (ARB). One would also like to know if any lifestyle changes, such as weight loss and exercise, were recommended in the study group before and during the antihypertensive therapy. Lifestyle changes in diabetic subjects with hypertension are part of the guidelines on treatment of diabetic hypertension (7,8).

The retrospective design of this report may have limited any control over lifestyle therapy. In fact, this omission further supports the contention of Berlowitz et al. (1) of lack of knowledge or interest by the clinician in the application of a comprehensive medical approach to treatment of hypertension in diabetic patients.

A significant limitation of the study as stated by the authors is that they used data from 1990 to 1995 that presumably came from the study leading to their landmark publication in 1993 of poor control of hypertension in the general population. In this study, they used subjects from five Departments of Veterans Affairs hospitals in the New England area (2). Based on this limitation, it is noteworthy that almost all the new outcome studies on prevention of microvascular and macrovascular complications by tight control of hypertension in diabetes have been published since 1995 (3–6). In addition, guidelines for threshold blood pressure levels in hypertension in diabetes were not firmly established and disseminated until the sixth report of the Joint National Committee (7) and the WHO/ISH publications (8). These guidelines recommended a target blood pressure in hypertensive diabetic subjects of $\leq 130/85$ mmHg. Thus, it is possible that clinicians from the present study did not have sufficient knowledge at that time to direct them to more aggressive antihypertensive therapy to reach target blood pressure in diabetes. A study of similar design, yet using a more current study population, would have strengthened the impact of this report. One might
hope that the wealth of new information on hypertension in diabetes would have yielded better results in their end point of an increase in antihypertensive medications when goal blood pressure had not been attained. Thus, it is possible that today, clinicians are more aware of these studies and are more aggressive in their efforts to reach goal. However, if this presumption were found not to be the case and the newer clinician group also failed to properly increase antihypertensive medications, then Berlowitz et al. would be even more accurate in their conclusions.

MICHAEL L. TUCK, MD
From the Department of Endocrine and Metabolism, VA Medical Center, Sepulveda, of the VA Los Angeles Greater Health Care System, Los Angeles, California.
Address correspondence to Michael L. Tuck, MD, Chief, Endocrine and Metabolism, VA Medical Center, 16111 Plummer St., Sepulveda, CA 91343 of the VA Los Angeles Greater Health Care System, 11301 Wilshire Blvd., Los Angeles, CA 90073. E-mail: mtuck@ucla.edu.

References