placebo effect was not significant on ABPM data of the full population but significant on the ABPM data of the placebo responders in clinic: “Placebo decreased the clinic BP and reduced systolic and diastolic ambulatory BP.” The second publication showed significant reduction with placebo of both systolic and diastolic BP but the “placebo effect was not observed for pulse pressure and heart rate.” In both publications we insist on the fact that placebo presents a proper effect and may decrease BP both in clinic and ABPM in similar population.

Hope that this gives you satisfaction.

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doi:10.1016/j.amjhyper.2005.08.012

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To the Editor:
We read with interest the article by Hawkins and Houston invoking a relationship between use of thiazide diuretics and end stage renal disease (ESRD). We agree with the concerns expressed in the accompanying editorials about the inherent pitfalls in inferences the investigators drew from their ecologic study. Hawkins and Houston failed to address another major problem with their study, namely “indication bias.” In fact, diuretics have a role in the management of chronic kidney disease, especially the loop diuretics, which they suggested may comprise as much as 46% of all diuretics prescribed. Therefore individuals diagnosed with chronic kidney disease, an antecedent to ESRD, may be prescribed diuretics for clinical management, hence the indication bias. In addition, although the investigators correctly point out that estimated glomerular filtration rate (GFR) was lower in the thiazide group compared to amloidipine in the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT), this parameter may be difficult to interpret due to acute hemodynamic changes associated with initiation of amlodipine therapy. They fail to report that the incidence of ESRD in ALLHAT was not different between the angiotensin-converting enzyme (ACE) inhibitor and the diuretic groups (relative risk lisinopril versus chlorthalidone 1.12; 95% confidence interval [CI] .89–1.04, \( P = .33 \)) and the calcium channel blocker versus diuretic groups (relative risk amloidipine versus chlorthalidone 1.11, 95% CI 0.88–1.38, \( P = 0.38 \)). This was consistent when stratified by baseline GFR and the presence of diabetes at baseline. The composite end point of a 50% decline in GFR or ESRD was also not different between thiazide diuretic compared to amloidipine and lisinopril in patients with moderate/severe reduction in GFR. Renal outcomes were prespecified secondary end points in ALLHAT, and the data are robust with large numbers of patients, and a total of 448 ESRD events (total of 1049 renal end points including >=50% decline in GFR events), clearly sufficient to refute the notion that diuretic therapy is associated with increased risk of ESRD. The question studied by the investigators is of clinical interest but the question is better answered by treatment comparisons in randomized controlled trials such as ALLHAT.

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