Hypertension and the Treating Physician
Understanding and Reducing Therapeutic Inertia
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The tide of hypertension is rising across the globe, including the United States. Among the 65 million Americans with hypertension, inadequately treated patients contribute substantially to the overall magnitude of the problem. Based on data from the National Health and Nutrition Examination Survey, 68% of the total remains above the therapeutic guideline for blood pressure (BP) goals. Those not at goal divide into 3 groups: those unaware of their high BP (≈31%), those who are aware but are not being treated (≈11%), and those who are being treated but remain above the goal of 140/90 mm Hg (≈27% of all hypertensive persons, or 47% of those treated with medication).2

In this innovative article using data from a diverse set of medical practices in the Southeastern part of the United States, Okonofua and colleagues provide some insights into this latter group’s inadequate treatment. The focus of their research is therapeutic inertia (TI), defined as “the provider’s failure to increase therapy when the treatment goals are unmet.” Although their sample was not based on a regional or national probability sample, their findings from the Southeast may have wide applicability, given that the level of BP control in their study was 45% at the end of the 2003 study period, not too different than the national rate of control of 53% among persons treated for hypertension reported for 1999 to 2000. The principal manifestation of TI (or clinical inertia, a more common term) documented in this study is failure to increase the dose of antihypertensive medications or add an additional medication in clinical encounters where the BP is above goal level. For patients whose providers showed a high level of TI, the number of medications actually declined during the 1-year study period from an average of 1.6 to 1.4, while those whose providers showed the lowest level of TI had their medications increased from 1.4 to 1.8. Whereas the rate of BP control declined in patients of providers with the highest level of TI, control improved substantially in the patients of providers with the lowest level of TI, illustrating the importance of changes in medication dose or number in the control of hypertension.

Another important observation from the study is that lack of appropriate action was more frequent in high-risk patients defined by the presence of diabetes, heart failure, or other manifestations of cardiovascular disease, in which the absolute benefit of adequate treatment would be the greatest. These results emphasize the need to increase the willingness of clinicians to add additional medication to the antihypertensive regimen if 1 or 2 drugs provide inadequate control. Despite the scientific limitations of the current study, such as the use of nonstandardized single BP measurements, a definition of TI that did not consider the degree of elevation of BP above the therapeutic goal, or lack of use of a more statistical approach to define whether the true BP was above goal, there is little doubt that the overall conclusion of the study is true—that TI explains a large part of the failure to adequately control pressure in medical practice.

The article by Okonofua et al provides little information on the reasons for TI. Phillips proposed 3 primary reasons why clinicians fail to intensify treatment for chronic conditions such as hypertension: (1) overestimating how many of their patients are at therapeutic goal; (2) use of “soft” reasoning to avoid intensification of therapy (physician perception without patient input that the patient will not accept more medications); and (3) lack of education, training, and organizational practices aimed at achieving therapeutic goals.4 In addition, we suggest (consistent with Phillip’s third explanation for TI) that beliefs by some physicians regarding a low risk of adverse cardiovascular outcomes associated with modest elevation in systolic blood pressure (SBP) may shape their behavior. Without effective education they may not fully appreciate the magnitude of the increased risk from seemingly modest SBP elevations of 5 to 10 mm above 140 mm Hg. Evidence that supports this hypothesis comes from a 1996 to 1997 national survey of primary care physicians, in which 76% said they would not initiate drug therapy for patients age 70 and older with SBP of 140 to 159 mm Hg, and 33% said they would not intensify treatment in a patient with SBP of 158 mm Hg.5 This reluctance to intensify treatment is actually consistent with the lack of strongly conclusive evidence from randomized trials in hypertensive patients that treating to SBP levels <140 mm Hg would be beneficial, particularly in older patients. A recent review concluded that “treatment of systolic hypertension in older patients with SBP of at least 160 mm Hg is supported by strong evidence...”6 The behavior of the clinicians in this study may reflect this conclusion, because
the only baseline risk factor associated with less TI was stage 2 hypertension, which is defined in part by a SBP of 160 mm Hg or higher.

This lack of appreciation of the true risk may illustrate anew how long it takes for scientific knowledge to diffuse into clinical practice. The current focus on systolic hypertension in the range of 140 to 159 mm Hg arose from clinical trials in the 1980s and early 1990s. In 1993, the Fifth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure lowered the criterion for systolic hypertension to 140 mm Hg from 160 mm Hg. The situation regarding control to below this range is better in the United States than in many countries and is improving for men. Among treated men in the United States, BP control improved from 45% to 60% from 1988–1991 to 1999–2000, but stayed the same among women at 48%.2

The need to reduce TI is clear. Dr Okonofua and colleagues estimate that a 50% reduction in TI would reduce average SBP by more than 5 mm Hg and lead to attainment of control rates similar to that seen in many clinical trials (65% to 70%).9 A 5 mm reduction in SBP, if maintained for several years, reduces the risk of major cardiovascular events (ie, stroke, coronary artery disease, heart failure, or cardiovascular death) by approximately 15% to 20%.9,10

Several approaches have been studied to eliminate clinical inertia in the treatment of hypertension and other conditions. These include feedback to physicians and patients on the progress toward therapeutic goals, decision support for intensification of treatment, continuing training through academic detailing, and organizational changes.11 The body of evidence is incomplete, and because the positive impact of these interventions is modest and variable, studies often involve simultaneous use of more than one intervention.12 As a result there is need to develop more powerful practical interventions to reduce clinical inertia and learn better how to adapt effective interventions to different practice settings. Future research should use stronger research designs where feasible and describe more fully the characteristics of the interventions and the organizational settings.12 Scientific and practical challenges also remain in the definition of clinical inertia. Can we develop refinements in the definition of inertia that incorporate the cause of the lack of change in the therapy, reflect the magnitude of the risk to the patient, and more effectively separate out a true deviation from the goal to a deviation caused by random variation?11

As great as the problem with TI is today in the management of hypertension, it may get worse because of the increasing scientific evidence that the SBP goal should be lower than 140 mm Hg in higher risk patients.13 If additional high-quality clinical trials confirm the strong epidemiological evidence and the more limited clinical trial data that our therapeutic goal should be 120 mm Hg rather than the current consensus goal of 130 mm Hg in patients with renal disease or diabetes or 140 mm Hg goal for most other patients, then effective strategies to further reduce TI will become even more important. These strategies will not only be important for hypertension but could be applied to other common risk factors and diseases, such as LDL cholesterol levels, hemoglobin A1c levels in patients with diabetes, and other chronic diseases such as depression. In addition, for BP lowering we must continue to use our current knowledge on effective lifestyle strategies both at the population and clinical levels, because each millimeter of reduction of SBP reduces cardiovascular risk.

References

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