Commentary

Aging Well and Aging Poorly: Primary and Secondary Low Blood Pressure

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The gerontological literature has been haunted over the past 20 years by cyclical rediscoveries of the inverse associations of blood pressure, weight, and cholesterol with mortality in the very old and frail. Unfortunately, the articles defining each cycle of discovery and rebuttal have not lead to a clearer understanding of this phenomenon. Dr. Goodwin’s article, “Embracing Complexity: A Consideration of Hypertension in the Very Old,” (1) resurrects the debate for blood pressure.

The study by Hajjar and colleagues showed that many physicians are reluctant to treat high blood pressure in the very old (2), and an accompanying editorial suggested that advanced chronological age should not be a contraindication to treatment (3). Dr. Goodwin disagrees, but his argument is based on flawed studies. These studies do not adequately account for the fact that the physiology of blood pressure differs between young and old primarily in those with normal or low blood pressure, not in those with hypertension. This difference, among those considered normal, turns out to be critical in estimating risks due to high blood pressure in old age.

Unlike younger populations, where a lower blood pressure is a sign of healthy contractile arteries, lower blood pressure in very old age often indicates pump failure secondary to hypertension or ischemic disease or reflects weight loss from poor health (4). This is clear in the studies that Dr. Goodwin cites. In the Tampere study, for instance, low blood pressure was associated with living in an institution or being in a hospital at the time of the study, with dementia or being older even among the oldest-old, being thinner, and having a lower glucose, cholesterol, and lower packed-cell volume. Those with low diastolic pressure had a higher prevalence of heart failure as well (5). This association of poor health with low blood pressure is also true in other studies (6) and the association was likely underestimated since studies did not include subclinical cardiovascular indicators or blood pressure history, which might also indicate poor health.

At any age, people with low blood pressure in a population are a mix of those with truly “healthy” low blood pressure (primary low blood pressure) and those with low blood pressure secondary to other causes, as identified above (secondary low blood pressure). Longitudinal studies of blood pressure show only a small proportion of those with low blood pressure in old age will have had lifelong low blood pressure. Most people in developed countries increase blood pressure with increasing age secondary to stiffening of the arterial wall, resulting in fewer and fewer people with primary low blood pressure with age. However, the proportion of people with low blood pressure remains stable, because factors associated with older age also cause secondary low blood pressure, so people “drop in” to the low blood pressure group with age.

A fundamental contribution of gerontology to clinical medicine is the concept of “biologic age,” defined as the interaction of genes, environmental exposures, health habits, pertinent social and psychological resources, health conditions, and treatments to produce a level of apparent health and physiologic reserve in old age. Biologic aging results in heterogeneity in the older population, with multiple
subgroups that differ in predisposition to, and recovery from, stressors. These subgroups range from those who are physiologically and functionally much younger than their chronologic age to those who are the frailest among a larger frail group. Biologic age provides the context to evaluate risk factors measured in old age, whether in population studies or in a clinical setting. Certainly the very old with higher systolic or diastolic pressure die at higher rates than even younger persons with these characteristics. In younger populations, those with primary low blood pressure predominate so the risk associated with low blood pressure is low. In old age, those with secondary low blood pressure predominate and risk associated with low blood pressure is high, as might be expected in a group where biologic age is poor. As the change from primary to secondary low blood pressure occurs over the lifetime, low blood pressure is transformed into a marker for poor, rather than good, health. Thus, when high blood pressure is compared with low blood pressure risk in old age, it will appear that the risk for high blood pressure is less. Similar arguments pertain to weight and other weight-related risk factors such as cholesterol.

This does not specifically address the question of why risks for morbidity and mortality might differ in the very old. There are at least 3 factors contributing to this. First, morbidity studies are often of disease incidence, so prevalent cases are excluded, thereby eliminating many with secondary low blood pressure. The comparison group for these incidence studies will have more cases of primary, lifelong low pressure. Second, death is associated with catastrophic stresses on the aged physiology causing failure of a critical organ. In this context, even primary low blood pressure may increase risk of death because reserve capacity may be low. Third, those treated for hypertension may have had sustained damage to vessels, which, in turn, lowered cardiac reserve. Lowering blood pressure may preserve function in daily life and lower risk of complications of hypertension, but it may not protect these individuals during periods of sustained physiologic stress, which may include noncardiac stress such as a hip fracture or other surgical procedure, infection, or depressive episode.

In younger populations, the risks for morbidity and mortality are similar, so interventions for one outcome tend to have parallel benefits for the other outcome. In older age, risk of morbidity and risk of mortality sometimes differ, such that morbidity risk factors are consistent in the young and old while mortality risk factors are opposite in direction. However, even if these conflicting risks cannot be explained, there is the question of whether prolonged life or quality of life is valued more by the old-old. For hypertension, where there is general agreement that treatment of hypertension, even in the very old, decreases risk of stroke and congestive failure (7) and may also prevent onset and worsening of cognitive impairment as well (8), attempting to preserve function seems like the most reasonable course.

No doubt blood pressure control with age requires more investigation. The efficacy of treatment of diastolic blood pressure was established first because this was the more common condition in younger people. Many older people derived beneficial treatment for hypertension because they had combined diastolic/systolic hypertension. However, it required specific trials for isolated systolic hypertension before the medical community began to treat this condition, and hypertension in the very old is still understudied. Development of hypertension is a slow process associated with physiologic changes throughout the cardiovascular system. Change of blood pressure in what is currently considered the “normal” range may ultimately be shown to carry as much risk as sustained high blood pressure, as was recently highlighted by the new designation of “preclinical hypertension.” The effect of chronic treatment for hypertension for 40 or 50 years is as yet unknown. As new indicators of subclinical physiologic reserve become available, the questions that can be addressed will identify new areas for prevention. Future generations will have been better treated and hopefully better controlled and may come into old age with greater reserve capacity. Preserving that capacity will represent a challenge to the next generation of geriatricians.

Fundamental principles of geriatric medicine still need to be disseminated to the community of physicians who care for older patients. Chronologic age as a basis for treatment of older persons has no place in modern medicine, since age captures only a small part of the relevant physiologic information necessary to evaluate that individual’s condition. “Biologic age” is very important because comorbidity and frailty are real issues in treating very old patients. Few trials have included this type of patient and, in the absence of data, the integration of information about health over the lifetime may have to guide treatment. Current blood pressure should be assessed in the context of biologic age as either primary or secondary, no matter what the level. As with most geriatric conditions, the emphasis should be on enhancement of functional ability with minimizing side effects. Certainly it is appropriate to fault physicians who put their patients at risk simply on the basis of the patient’s chronologic age.

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