Does Blood Pressure Control Contribute to a More Successful Aging?

Athanase Benetos

In developed countries, life expectancy at birth dramatically increased from 47 years at the beginning of the 20th century to ≈80 years at the beginning of the 21st century. This astonishing change in survival patterns was primarily attributable to the very significant decrease in infant mortality and to the eradication of infectious diseases, both of which are strongly related to the improvement of life conditions and to the progress of modern medicine. At the same time, although to a lesser degree, the progress in diagnosis, treatment, and prevention, especially of cancer and cardiovascular diseases, has also contributed to increased life expectancy at more advanced ages. A significant increase in life expectancy leads to an important increase in the number of subjects who are frail, incapacitated, and have reduced autonomy.

The perspectives of a greater increase in life expectancy raises several biological, ethical, medical, and socioeconomic questions that go far beyond the scope of this article. Nevertheless, one of the major issues of modern medicine is how to increase disease-free and incapacity-free life expectancy. The progressive reduction in functional reserves and the deterioration of cognitive, cardiovascular, sensorial, and osteomuscular systems are the most frequent causes of frailty, incapacity, and loss of autonomy in the elderly.

Assessment of clinical and biological markers and factors associated with morbidity and mortality has become a major issue that has considerable medical, social, and economic consequences. This concept, which evolved primarily in the field of cardiovascular medicine, led to the creation of a large number of risk evaluation equations.1 The aging of the population in industrialized countries and the consequent increase in preventive medicine among the elderly attempt to answer the following pertinent questions: Which parameters should be considered to evaluate the risk of disease, incapacity, loss of autonomy, and death? Can factors associated with longevity be identified? Does correcting these factors lead to an increase in total and in disease-free life expectancy, thus contributing to a more successful aging?

The article by Oscar Franco et al answers some of these questions.2 The authors analyzed the life course of 3128 participants of the Framingham Study according to their blood pressure (BP) levels at age of 50 in terms of life expectancy and life expectancy without cardiovascular disease. This analysis found that normotensive subjects survived ≈5 years longer than hypertensives, whereas cardiovascular disease-free life expectancy was 7.2 years longer in normotensive than in hypertensive individuals.

During the past 30 years, a very large number of studies have shown that hypertension is a major risk factor for morbidity and mortality in young, middle-aged, and older populations of both genders.3 Hypertension is a risk factor for stroke, ischemic white matter lesions, coronary artery disease, and heart failure. Moreover, large trials have clearly shown that normalization of BP with treatment significantly reduced cardiovascular and renal morbidity and mortality.4 High BP has also been related to vascular dementia, as well as to Alzheimer’s disease.5 The association between hypertension and vascular dementia is not surprising because hypertension is the predominant factor for all types of cerebrovascular diseases that often lead to vascular dementia. What is more surprising is the observation that hypertension is also strongly associated with other types of dementia, notably with Alzheimer’s disease. Support for the role of hypertension in Alzheimer’s disease is also provided by some, but not all, clinical trials showing that antihypertensive treatment was able to reduce the incidence of cognitive impairment dementia and, more specifically, Alzheimer’s disease.6 Finally, hypertension increases the risk of several other age-related diseases such as high intraocular pressure and osteoporosis.7 Although noncardiovascular morbidity was not evaluated in the study by Oscar Franco et al,2 it is possible that life expectancy without age-related diseases such as dementia and osteoporosis was greater in normotensive than in hypertensive subjects in the Framingham Study.

What are the mechanisms of such associations? Essential hypertension is closely linked to the aging process in humans. Systolic BP (SBP) increases continuously with age, whereas diastolic BP (DBP) increases until the sixth decade and then tends to be stable or even decline in the elderly. These age-dependent changes in SBP and DBP, leading to a rise in pulse pressure (PP), are attributable to the stiffening of the large arteries, which reflects the aging of the arterial system. Individuals who have an excessive increase in aortic stiffness and PP with age are considered to have accelerated arterial aging and are at higher risk for cardiovascular morbidity and mortality.8 Interestingly, several studies have shown that the presence of high BP in middle age is associated with the manifestations of accelerated arterial aging later in life. In a longitudinal study, we found that subjects with high BP levels develop more pronounced stiffness of their aorta later in life.9 These results reinforce data from the Framingham cohort.
showing that people with high BP are more likely to present an excessive increase in SBP and a decrease in DBP later in life. Therefore, there is a vicious circle between elevated BP and arterial stiffening. Chronic increase in SBP and DBP in middle-aged individuals is responsible for vascular wall structural and functional modifications accelerating age-related arterial stiffening. The latter induces a further increase in SBP, with a concomitant decrease in DBP. We can suggest that hypertension-related accelerated vascular aging at the level of the small or large arteries leads to impaired perfusion of the different organs and therefore contributes not only to cardiovascular complications but also to the decline in the function of several organs and systems and therefore the facilitation of several age-related diseases.

Do these results allow us to draw the conclusion that controlling BP could prolong life expectancy and reduce diseases and incapacity? Although the design of the present study cannot answer this question, clinical trials in middle-aged and elderly populations have previously shown that treating hypertension clearly leads to a decrease in cardiovascular morbidity and mortality and probably to a partial prevention of dementia, a major cause of incapacity and loss of autonomy.

From the start of human history, suppression of diseases, extension of life span, and antiaging strategies are huge areas for myths, popular beliefs, and biological research as well as pseudoscience and cheating. From Serge Voronof and Frank Lydston, who performed testicular transplants in the early 20th century, to the modern hormonal therapies, merchants of eternal youth propose several treatments and ideas for stopping age-related diseases, increasing life expectancy, and obtaining “successful” aging. Although as scientists, we must be receptive to advances in biology and medicine, so far, most of these antiaging medicines are designed to benefit those who sell them. Control of cardiovascular risk factors, regular physical exercise, and a balanced diet, although they do not guarantee eternal youth, have clearly shown their efficacy in improving quality of life and reducing age-related decline of several functions. The study by Franco et al adds some solid evidence to the fact that prevention and effective control of high BP can prolong life expectancy and, even more important, can add some disease-free years to an elderly person’s life, a key element for a more successful aging.

References

Does Blood Pressure Control Contribute to a More Successful Aging?

Athanase Benetos

_Hypertension_. 2005;46:261-262; originally published online June 27, 2005;
doi: 10.1161/01.HYP.0000173425.50869.a9

_Hypertension_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2005 American Heart Association, Inc. All rights reserved.
Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://hyper.ahajournals.org/content/46/2/261

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Hypertension_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at: http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Hypertension_ is online at: http://hyper.ahajournals.org//subscriptions/