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Osteoporosis as a Community Health Problem: Lessons Learned From Studying Hypertension

Michael Kleerekoper, MD,* and Dorothy A. Nelson, PhD*

Campaigns to increase the medical and lay communities' awareness and understanding of the problems of unrecognized and untreated hypertension have led to a progressive decline in morbidity and mortality from hypertension-related diseases. While osteoporosis is also a community health problem, educational and awareness campaigns are still in their infancy and decades may pass before these result in a declining morbidity and mortality from osteoporosis. We identify areas where concepts learned from years of hypertension research might be applicable to the study of osteoporosis as a community health problem and thereby lessen the time needed to effect a declining morbidity and mortality from osteoporosis. We discuss the importance of a more specific diagnostic classification and the implications of these concepts for clinical trial design. (Henry Ford Hosp Med J 1988; 36:113-6)

A major triumph in community medicine during the past several decades has been the campaign to increase public awareness of the dangers of unrecognized and untreated hypertension—the so-called “silent epidemic.” Recent health care statistics show a progressive decline in the death rate from myocardial infarction and stroke (1).

Osteoporosis is a community health problem of lesser magnitude than hypertension and its sequelae but is nonetheless of significant proportions. Available data on the incidence and prevalence of osteoporosis indicate that even by a most conservative estimate 5% to 10% of all white women in the United States will have sustained at least one osteoporotic vertebral compression fracture by age 70 (2,3) and that the health care cost of osteoporotic fractures exceeded $6 billion in 1986 (4).

Increasing physician and public awareness of osteoporosis is a phenomenon of the 1980s that parallels the public awareness campaign for hypertension. We will explore some of the similarities between these two disorders to determine whether any of the lessons learned from decades of hypertension community action programs can guide us in our approach to the problem of osteoporosis. In particular, we discuss the importance of more specific diagnostic criteria, implications for prevention and therapy, and suggestions for effective clinical trial design.

Measurement

By convention the term blood pressure (BP) implies measurement of systolic and diastolic blood pressure in the brachial artery using a standard, calibrated sphygmomanometer. When BP is measured at a different site or by a different technique, a modifying comment is always appended.

No convention yet exists concerning the site and method of measuring bone mineral density (BMD). Even a preferred nomenclature does not exist, with terms such as bone mass, bone density, bone mineral content, and bone mineral density used interchangeably despite subtle but important differences in their meaning.

As BP has systolic and diastolic components, BMD has cortical and trabecular components. While these components of BP or BMD are related in apparently healthy subjects, this is not necessarily true, and information about one often cannot be inferred from knowledge of the other. Similarly, disease states exist in which the measurement of one component of BP or BMD is normal and the other is abnormal. Both systolic and diastolic BP are measured at the same site using the same methodology, but this is not yet possible for BMD measurements with current technology and may never be possible. With the

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exception of quantitative computed tomography which specifically selects a region of interest prior to measurement, current methods of BMD measurement cannot distinguish between cortical and trabecular bone.

Abnormal Measurement

Both BP and BMD change normally with normal aging, albeit in opposite directions. This age-related change in normal values is not necessarily trivial since those diseases resulting from prolonged abnormalities in BP or BMD become increasingly more prevalent with advancing age. Nonetheless, before assigning a disease process to an apparent abnormality detected in a measurement it is essential that the correct reference interval be employed. One important difference between the reference intervals for BP and BMD, in addition to being age-dependent, is that the reference interval for BMD is also gender- and race-related, with normal males having a higher BMD than normal females and normal blacks having a higher BMD than normal whites (5,6).

When the measured BP is above the appropriate reference interval, the subject in whom the measurement was made is given the diagnostic label “hypertension.” In most cases hypertension is asymptomatic and in the strictest sense of the word is not really a disease until complicated by a morbid event such as a stroke or myocardial infarction. However, the simple act of attaching the disease label “hypertension” to an asymptomatic person increases absenteeism from the workplace by as much as 80% (7-9) even though the reason for the absenteeism is seemingly unrelated to the elevated BP.

When the measured BMD is below the reference interval, most physicians append the diagnostic label “osteoporosis,” making no distinction between this asymptomatic condition and the clinical disease in which the low BMD is complicated by nontraumatic fractures. Unfortunately, no suitable skeletal equivalent of “hypertension” exists, ie, a descriptor of the condition of having a low BMD. “Osteopenia” is gaining some acceptance in this regard, but it is unlikely that widespread use of this term will penetrate most of the medical or lay communities. The rapid deployment of “osteoporosis screening centers” throughout the United States (4,10-12) all but ensures the continued use of the term osteoporosis to include both the asymptomatic subject with an abnormal measurement and the patient who is terminally ill from complications of an osteoporotic proximal femur fracture.

Despite the limitations of current terminology, it is essential that we learn from the experience with the diagnosis of hypertension as a cause of morbidity and absenteeism and not perpetuate this error in the case of a low BMD. As more women enter and remain in the work force past the age where a low BMD becomes more prevalent, this situation might worsen if patients with mechanical lower back pain and a subnormal BMD were labeled as having “osteoporosis” even though fractures had not yet occurred. It is therefore imperative to develop either an acceptable skeletal equivalent to the term hypertension or the routine use of modifiers to distinguish the asymptomatic premorbid “osteoporosis without fractures” from the morbid condition of “osteoporosis with fractures (spine, hip, forearm, etc).”

Preventing an Elevated BP or Low BMD

Although the precise etiology of essential hypertension is unknown, several diet and life-style factors appear to be important in maintaining a normal BP. Epidemiologic studies suggest, at least in susceptible individuals and animal models, that a high sodium intake predisposes to hypertension (13). Similar observations have been made recently in people and experimental animals concerning a low dietary calcium intake and the development of hypertension (14,15). Further studies are needed to identify, if possible, persons with a susceptibility to develop an elevated BP if exposed during their formative years to a diet that is too high in sodium, too low in calcium, etc. Similar comments could be made concerning obesity and the development of hypertension. A major factor in the pathogenesis of hypertension, genetic predisposition, is not yet susceptible to change.

Much more is known about the pathogenesis of the most common form of osteoporosis, postmenopausal osteoporosis, than about the pathogenesis of essential hypertension. From a community health point of view, the problem of osteoporosis is largely confined to white females. (While hypertension is more prevalent in blacks than in whites, the prevalence in each of these subgroups is sufficiently great to render hypertension a community health problem for both sexes and races.) The reason for the greater prevalence of osteoporosis in this white female population later in life is already apparent by age 25 or 30 when peak adult BMD is achieved (16,17). By this age males already demonstrate a higher BMD than females, and blacks have a higher BMD than whites at all ages (16). By optimizing their dietary calcium intake during childhood and adolescence and maintaining an optimum program of load-bearing physical activity during the same period, young women can optimize peak adult BMD and minimize the risk of subsequent osteoporosis (18). Little is known about factors that govern BMD between age 25 and the menopause, but some of the pieces of the puzzle are beginning to fall into place. Nulliparity is associated with a greater likelihood of osteoporosis in later life (19,20), as is a pattern of irregular menses during the reproductive years. Conversely, multiparity and use of oral contraceptives appear to provide some advantage with respect to maintaining an optimum BMD (19,21). The single most important factor in maintaining a normal BMD throughout life for most women is the avoidance of hypogonadism. Practically, this means adequate hormonal replacement therapy at the time of the menopause, a program that has been conclusively shown to retard postmenopausal bone loss and to reduce the likelihood of osteoporotic fractures (2,22). As is the case with hypertension, any genetic predisposition toward osteoporosis is not amenable to change.

Treating Acute Complications

No curative therapy exists for an acute hypertensive cerebrovascular accident (hemorrhagic stroke) but much can and should be done to minimize the affected area of the brain. This therapy includes cautious lowering of the BP if elevated at the
time of the stroke, although many of the more potent antihypertensive agents are relatively contraindicated in patients with a completed stroke. Once the patient is stable after the cerebrovascular accident, the mainstay of therapy is rehabilitation to restore and maintain an optimal functional state. Rehabilitation is also the mainstay of therapy following an acute myocardial infarction that complicates chronic uncontrolled hypertension, although recent advances in angioplasty and thrombolytic therapy offer a more aggressive and potentially more rewarding approach to therapy.

The only recognized complication of a chronically low BMD is a fracture that occurs in the absence of trauma or in response to only trivial trauma. Although therapy is dependent on the site of fracture, rehabilitation also plays a major role. The distal forearm (Colles) osteoporotic fracture is a relatively trivial event treated with closed reduction for a short time followed by physical therapy until the expected full restoration of prefracture function. With the proximal femur (hip) osteoporotic fracture, open reduction followed by early ambulation and an aggressive physical therapy/rehabilitation program is the treatment of choice. When the patient is too frail to undergo surgery, the rehabilitation program is also limited and the prognosis is poor. Overall, despite significant improvements in surgical techniques and physical therapy, there is still a 20% mortality during the first three months following an osteoporotic hip fracture, and at the end of one year only one third of the patients are restored to their prefracture functional state (23,24). For the most common osteoporotic fracture, vertebral compression fractures, no surgical approach is possible and the treatment program must revolve around an active rehabilitation program. With the possible exception of the Colles fracture that occurs most frequently in the first decade after the menopause, estrogen has a very limited documented role in the management of the patient with fractures complicating a low BMD.

This is not meant to imply that attempts should not be made to lower the BP in patients who have had a stroke or a heart attack or to increase the BMD in patients who have sustained an osteoporotic fracture. However, often this approach is too little too late: drugs and changes in life-style that are of critical importance in preventing the end-organ damage have a lesser role once that complication has occurred, and rehabilitation becomes crucial to the maximum recovery and maintenance of function following a stroke or an osteoporotic fracture.

Lessons to be Learned

In one important respect, osteoporosis has much to teach hypertension. The segment of society at greatest risk for developing osteoporosis (postmenopausal white females) is clearly identifiable. Furthermore, we already know how to prevent the premorbid asymptomatic state of having a low BMD (adequate dietary calcium and physical activity during the developing years and adequate gonadal hormone replacement in the immediate postmenopausal years). For many individuals an elevated BP can be avoided by careful attention to dietary sodium intake and avoidance of obesity, but it is not yet possible to identify those people who will benefit from this, nor is it known at what age this prophylactic program should begin.

Many more lessons that have already been learned from the hypertension/stroke aspect should now be applied to the osteopenia/osteoporosis field. Most importantly, it is time to focus attention with respect to drug therapy on those patients who have already been identified as having a low BMD (analogous to the treatment of patients with a high BP). Numerous clinical trials are currently under way to determine if a particular drug regimen can significantly reduce the fracture rate in patients who already have one or more fractures as an entry criterion for the trial. We believe that it is even more important to do trials in which the entry criterion is a reduction in BMD and the criterion for success is an increase or stabilization in BMD. Classes of drugs capable of increasing BMD should be developed and subjected to vigorous clinical trials. Many classes of antihypertensive medications have been developed and successfully introduced into clinical medicine even though the precise pathogenesis of essential hypertension remains enigmatic. Occasionally the pharmacologic action created by these drugs is only fully elucidated by studies completed after antihypertensive efficacy has been demonstrated. Therefore, there is no inherent need to await the complete unravelling of the pathogenesis of bone loss before newer drugs aimed at increasing BMD are developed and investigated. Patients entered into these trials ideally should be followed long enough to permit quantitation of morbidity endpoints (fractures), but this should not be the primary end-point of such trials.

No single drug therapy for hypertension exists, and even in the individual patient more than one drug is often required. Of the several different etiologies for hypertension that have been postulated over the years, many of these theories have been successfully translated into new BP-lowering drugs or classes of drugs. The generation of new information about the biology of bone and the mechanisms of bone loss and bone formation should also result in newer drug therapies. To a certain extent this is starting to happen as is the case with the ADFR (activate-depress-free-repeat) approach (25) to the treatment of osteoporosis. In the long term this particular theory may prove to be incorrect or incomplete, but for now it is clearly a step in the right direction.

We all must acknowledge for practical purposes of investigating and understanding osteoporosis that it is a recently recognized disorder being studied with very new technology. We must learn the capabilities and limitations of each new advance and avoid the temptation to expect all the answers to come tumbling out with every new piece of the puzzle as it unfolds. We must increase public awareness about osteoporosis yet be careful to avoid inducing unnecessary fear in healthy people or false optimism in those already afflicted. This can happen only if the medical community remains precise in our use of bone mass measuring techniques and particularly in the terminology with which aspects of this disease are discussed among ourselves and with our patients. The reasoned approach to hypertension is just now beginning to pay off for the community. With time the same will be true for osteoporosis.

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