Osteoporosis is defined as a decrease in bone mass or an increase in bone fragility such that a fracture is likely to result following even minimal trauma (i.e., bending over to pick up a newspaper, opening a drawer, etc.) The most common fracture sites are the wrist, spine, and hip. The clinical diagnosis often is not made until a fracture occurs. However, since the definition of osteoporosis is based upon the probability of a fracture, not the presence of one, it should be possible to make the diagnosis based upon measurements of bone mineral content, which usually is closely correlated with bone strength.

Assessment of bone quality and quantity has been difficult. Although bone is metabolically a very active tissue, bone turnover is slow, and prospective studies take a long time. Until recently, precise repeated measurements of bone mineral content were not possible. Standard X-rays have been available to clinicians since the 1920's, but X-rays are insensitive, and one-third of bone mineral must be lost before the bone loss becomes apparent. Precise X-ray techniques to examine the hand, wrist and forearm have been developed, but they do not necessarily reflect the mineral content of the spine and hip. Bone biopsies permit visualization of bone cells and their activity at a given location. However, a biopsy is an invasive procedure and cannot be performed repeatedly at the same site in the skeleton to follow changes over time. Most bone mineral is calcium, and total body calcium can be measured by neutron activation analysis. This procedure is very expensive and is only available in a few research laboratories. In the past decade, computed tomography, dual photon absorptiometry, and, most recently, X-ray absorptiometry, have made available more precise, repeatable measurements of bone mineral content (1,2). The result has been a marked increase in studies concerning sequential changes in bone.

The skeleton is expected to provide support for a lifetime, and throughout life normal bone must undergo cycles of remodeling and repair (3). Discrete areas of bone known as bone forming units first undergo an activation phase under the influence of local biochemical mediators. This is followed by a resorption phase lasting several weeks during which bone cells known as osteoclasts remove an area of bone. Subsequently, under the influence of other bone cells known as osteoblasts, there is a formation phase lasting several months. During this time new bone is formed to replace the bone that previously was removed. If bone resorption and bone formation occur at different sites, bone is remodeled.

If bone resorption exceeds bone formation, there is net loss of bone mass. This occurs to some extent in everyone during the aging process. When net bone loss is great enough, osteoporosis results.

The adult skeleton is composed of two major types of bone, cortical bone and trabecular bone, which undergo remodeling and repair at different rates. Cortical bone forms the surfaces of all bones and forms most of the shaft of the long bones of the arms and legs. Cortical bone is dense, highly mineralized, and turns over very slowly (approximately 3 percent/year). Cortical bone increases in diameter but becomes thinner and weaker with age. Trabecular bone, which turns over more rapidly (approximately 30 percent/year), is composed of interconnecting bony plates (trabeculae) and is found primarily in the spine and pelvis and at the ends of the long bones. Trabecular bone contains more soft tissue per unit volume, and thus is less mineralized. With aging, trabecular plates become thinner, but the more serious loss of trabecular bone occurs when bone resorption results in perforation and disappearance of the cross bridges between trabeculae. Once this happens, repair is not possible.

Trabecular bone mass peaks in the third decade; cortical bone mass reaches its peak early in the fourth decade. Thereafter bone mass decreases. Initially, this occurs slowly. In women, bone loss accelerates during the decade after menopause and then slows again. There is no comparable acceleration of bone loss in normal males (4). It seems logical that efforts to maximize bone mass should be made during the first three decades when bone mass is accumulating, but this has never been proven. Peak bone mass is greater in men than in women and is greater in blacks than in whites and Orientals. Although all racial groups lose bone with age, white women (especially those of northern European extraction) and Oriental women are at greater risk for developing osteoporotic fractures.

Fractures of the wrist and spine occur primarily in women, usually between the ages of 55-70 (in the U.S., the Caucasian female: male ratio = 6.8:1). Fractures of the hip occur in both sexes, usually in persons over the age of 70 (in the U.S., the Caucasian female: male ratio = 2:1). To account for these sex and age-related differences, it has been proposed that osteoporosis be classified into two types (5). Type I osteoporosis is thought to be characterized primarily by loss of trabecular bone of the hip and spine due to loss of sex hormone (estrogen) following menopause. In normal males there is no abrupt loss of sex hormone (testosterone) comparable to the loss of estrogen, although circulating testosterone concentrations do decrease somewhat with age. In males who do have a marked loss of testosterone (due to trauma or disease), bone fragility and osteoporotic fractures
are increased. Type II osteoporosis is thought to be the result of loss of both trabecular and cortical bone due to decreased absorption of calcium in older individuals. Hip fractures in both sexes are believed to be the consequence of type II osteoporosis and of an increased propensity to fall with aging. Whether the different types of osteoporotic fractures actually are caused by different pathologic mechanisms is still to be determined.

In adults the ability to remodel bone is very limited, and emphasis must be placed upon prevention of bone loss rather than on treatment once bone loss has occurred. Efforts to prevent bone loss have been focused upon: 1) replacement of sex hormones; 2) maintenance of bone mineral content using nutritional strategies to maximize the availability of calcium; 3) exercise regimens to maintain bone mass; and 4) lifestyle changes to diminish fracture risk.

There is no question that estrogen replacement therapy prevents the accelerated loss of bone associated with menopause (6). Retrospective and prospective studies carried out for up to 15 years show stabilization of bone mineral content associated with estrogen use, and a corresponding decrease in osteoporotic fractures of the wrist, spine and hip. Although estrogen receptors have been found in bone and estrogen decreases the sensitivity of bone to parathyroid hormone (a hormone involved in the activation and resorption phases of bone and remodeling) the exact mechanism by which estrogen prevents bone loss is unknown. Estrogen replacement therapy results primarily in stabilization rather than formation of new bone, and thus estrogen is most effective if begun soon after menopause, before the accelerated phase of bone loss has occurred. Accelerated bone loss resumes when estrogen replacement is discontinued. It is unclear whether estrogen therapy is effective in preventing fractures if it is begun so long after menopause that the period of accelerated bone loss is already complete.

Estrogen stimulates the endometrial lining of the uterus, and the incidence of endometrial cancer is increased after estrogen use. This can be decreased to below the level expected in women on no hormone replacement by the addition of another hormone, a progestin, for at least 10 days per month (7). Some progestins may have adverse effects on circulating cholesterol concentrations, however, and the optimal dose of progestin is uncertain. In men who are testosterone deficient, sex hormone replacement therapy should stabilize bone mass, just as it does in postmenopausal women. However, long term prospective studies following testosterone replacement have yet to be completed.

Ninety percent of total body calcium is stored in bone, and the decrease in total body calcium associated with aging is reflected in a decrease in bone mineral content. Numerous studies of the role of dietary calcium in the stabilization of bone mass and prevention of osteoporotic fractures have been performed. Population studies have not shown good correlation between dietary calcium intake and the prevalence of osteoporotic fractures. Genetic differences and variations in the type and quantity of exercise are some of the confounding factors. The most frequently cited study in support of the beneficial effects of dietary calcium was conducted in two Yugoslavian villages in which ethnic background and level of exercise were comparable but calcium intake (dairy products) differed markedly. Women in the dairying community who ingested more calcium had a higher bone mass at all ages and had a lower incidence of hip, but not wrist fractures (8). This study suggested that if the difference in hip fracture rate was due to the difference in calcium intake, it was the ingestion early in life resulting in a higher peak bone mass that was important.

Is there evidence that increasing calcium intake later in life is beneficial? In the United States the average dietary calcium intake of adult women is low (~500-600 mg/day) and is below the recommended daily allowance of 800 mg/day. Calcium absorption decreases in both men and women with age. In women the calcium requirement to stay in calcium balance (intake = excretion) is affected by estrogen. One study has shown that premenopausal women and postmenopausal women taking estrogen required 1000 mg of calcium/day to stay in calcium balance, while postmenopausal women not taking estrogen required 1500 mg calcium/day (9). Although studies have shown that women taking calcium supplements have less net calcium loss than women taking no added calcium, increased calcium intake alone cannot fully prevent loss of bone mineral. However, in one study of fracture rates, as opposed to bone mineral content, calcium supplements alone were associated with a decrease in vertebral fractures (10). Calcium supplements also may decrease the amount of estrogen required to maintain bone mass (11). Supplemental calcium may be more effective in preventing loss of highly mineralized cortical bone than loss of the less mineralized trabecular bone (12). If so, calcium supplements may be more beneficial for the hip than for the spine. More studies are needed, particularly in persons over the age of 70.

Calcium is being added to foods in which it does not occur naturally (i.e., orange juice and cereals), and a large number of calcium supplements are available. The percentage of calcium in these products varies considerably. Calcium carbonate contains 40 percent calcium, while calcium lactate contains 10 percent. Individual tolerance for different supplements and their absorption efficiency also are highly variable and influenced by diet and disease states.

Vitamin D increases calcium absorption but first must be converted to its active form by enzymatic steps occurring in the liver and then in the kidney. The enzyme activity responsible for the last step decreases with age. However, adding pharmacologic doses of vitamin D, even in its most active form, has not resulted in significant improvement in bone mass or fracture rate (13). Furthermore, daily doses of vitamin D in excess of the physiologic range of 400-800 units may be dangerous. Since vitamin D is a fat soluble vitamin, a large excess may be stored, leading to very high blood and urine calcium concentrations.

Studies also have been done using calcium plus calcitonin, a hormone which inhibits calcium resorption from bone. The use of calcitonin resulted in a very small increase or stabilization of bone mass compared with use of calcium alone (14). Prolonged studies to examine the effect of calcitonin on osteoporotic fracture rates are not yet available. Calcitonin is expensive and cannot be given orally; a snuff preparation is now being tested.

Calcium is only one component of bone, and other nutrients obviously affect bone tissue. Excess phosphate, protein, fiber, phytate, acid loads, and caffeine have been reported to have adverse effects upon calcium balance and presumably upon bone mass. However most of these observations have been derived from animal studies, epidemiologic studies, or studies which have been carried out with pharmacologic doses in a form not usually found in the diet, rather than in normal mixed meals. It is important that these conclusions be confirmed under more physiologic conditions before they are incorporated into dietary recommendations for the general public.
Normal bone growth and remodeling are dependent upon alterations in mechanical stress, and bone mass is lost when gravitational forces are not present or during prolonged immobilization. Controlled studies have shown that weight bearing exercise may result in stabilization or increase in bone mass at all ages, even in the very elderly (15, 16). However, the exercise must be directed toward specific areas of the skeleton. Walking is beneficial for the hips and spine but does nothing to increase bone mass in the wrist. Individuals who already have lost so much bone mass that they are at high risk for osteoporotic fractures should undertake exercise with caution under medical supervision. Excessive exercise also may be harmful even in those with normal bone mass. For example, excessive exercise in premenopausal women results in decreased estrogen production due to suppression of the hypothalamic-pituitary-gonadal axis. This in turn results in a decrease in bone mass.

The hormonal and nutritional strategies considered above may stabilize bone mass, but they have not been shown to result in a significant increase in bone mass in those who have already lost bone mass. Efforts are being made to find regimens which will restore bone in depleted areas of the skeleton where fractures have not yet occurred.

Fluoride has been shown to increase trabecular bone mass and decrease fractures of the spine after one to two years in some individuals (17). However, the high doses required to achieve adequate bone concentrations of fluoride within two years may also have severe side effects. Since total body calcium has not been shown to increase after fluoride administration, there is concern that fluoride may result in shifting calcium from cortical bone to trabecular bone. Thus fluoride may protect the spine at the expense of the hip. Fluoride treatment for osteoporosis has not yet been approved by the Food and Drug Administration, but controlled studies using fluoride are now in progress.

As indicated above, bone remodeling and repair require cycles composed of an activation-resorption-formation sequence. Since bone resorption and formation are coupled, stopping resorption completely would also halt bone formation. However, it has been proposed that if many bone forming units could be induced to undergo activation and resorption simultaneously, but the resorption phase were terminated early before removal of the usual quantity of bone, bone formation would proceed normally. The sequence could then be repeated and, in time, a net gain in bone mass would result. This concept is known as coherence therapy (since multiple bone forming units would be in phase with each other) (3). Many compounds are known to initiate the activation phase in vitro and agents which inhibit bone resorption also are known. Investigators are attempting to find a combination of effectors and timing which will adequately test this hypothesis. Preliminary reports are available, but results are mixed. Newly discovered growth factors and new mechanisms of action for those already known provide intriguing possibilities (18). In the future it may be possible to selectively enhance the bone formation phase.

Although methods for examining bone mineral content are more precise and more available than before, it is not possible to predict from one or even two examinations who will develop osteoporotic fractures (19). For example, postmenopausal women who initially have a small bone mass may not develop fractures if their subsequent rate of bone loss is relatively slow. Concern about developing osteoporosis should be influenced by the number and type of risk factors present (2). Age and genetically determined risk factors such as sex and race, and previous family history of osteoporosis cannot be altered. On the other hand, risk factors such as smoking, sedentary life style, poor nutrition, high alcohol ingestion, and some medications and disease states certainly can be modified. So can living conditions (loose throw rugs, slippery floors, no railings, etc.) which increase the propensity to fall. It is most important that anyone at potential risk for osteoporosis develop an individualized prevention plan—and the sooner the better!

References

Introduction

Since the arrival of human beings on this planet and the discovery of fire, people have used fire to warm their shelters, cook their meals, and take advantage of their neighbors. Fire became not only a tool but, unfortunately, a weapon. Through the ages, humans have used fire to help defend their homes and, in war, to drive enemies out of their homes. Today, persons continue to use this primitive weapon against their fellow human beings (1).

Arson

Arson is a crime in most of the civilized world. In Minnesota it is a felony to use fire to destroy property (Minn. Stat. Section 609.561). It makes no difference if the property is that of another or if it is your own. This is a serious crime, with a 20-year prison term for setting fire to a dwelling or any building where someone may be residing. Only murder carries a more severe penalty.

What is arson? The statutes define it as the deliberate and intentional destruction of property by fire or explosion (2). While an investigator can determine that a fire is of incendiary cause, only a judge and jury can make a determination of "arson."

The major question for a fire investigator is whether the fire was of other than accidental cause. All other reasonable, potential accidental causes for the fire must be eliminated. If the investigator can not eliminate all reasonable accidental causes, the investigator cannot declare the fire to be incendiary.

Fires are categorized as accidental, incendiary, or uninvestigable. Accidental fires—caused by careless use of smoking materials, improper electrical equipment, careless use of flammable and combustible liquids, and acts of God such as lightning—are familiar. Uninvestigable fires are those where all traces of the structure are destroyed by the fire leaving nothing but ashes in the wake. These are usually unwitnessed fires with no fire department response. It is usually impossible to determine origin and cause in these cases. That leaves the incendiary fire. What motivates the firesetter to take such drastic action?

Motives for Arson

The motives for arson fit into a number of commonly recognized categories (3,4). In order of frequency they are:

1. Juvenile firesetting
2. Spite/Revenge
3. Fraud
4. Pyromania
5. Hero (Image seeking)
6. Crime Concealment
7. Civil Unrest/Disobedience