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Osteoporosis of the Aged Spine

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Osteoporosis is a disease of middle and later life in which the composition of the bone is normal, but there is too little of it. Histologically, the cortical surfaces are thinned and the trabecular structure narrowed, so that the marrow spaces are enlarged. The bone structure is weakened and can be easily fractured by trauma, or sometimes without trauma from the stress of weight-bearing alone. Persons with this condition have increased liability to fractures, and osteoporosis plays a large role in the increased frequency of fractures of the lower end of the radius, the spine and the neck of the femur in older people, particularly women. The existence of osteoporosis must be taken into consideration in the treatment of such fractures, particularly when there is a need for internal metallic fixation of bone fragments with nails, screws and plates, because the bony structure may not be strong enough to retain the metallic appliances used for fixation.

Since osteoporosis is particularly common in the aged, it must influence to a large extent the treatment of fractures in patients with this condition. It is necessary to curtail bed rest and immobilization, because disuse increases the loss of calcium, and in general one should strive to get patients out of bed quickly. Complicating conditions are frequently present in the aged patient, such as cardiac, renal, mental and gastrointestinal disturbances.[22] Because aged people eat poorly, frequently they are ill-nourished; and they do not tolerate sedation well. Fractures in such patients may prove to be a starting point of a chain of events, resulting in organ breakdown, such as cardiac failure, because of pain and salt retention, or cystitis, because of dehydration or of infection and pulmonary complications. Thus the aged osteoporotic patients are much poorer risks than the patients with relatively strong bones. For this reason they must be treated with understanding of the total problems inherent in both osteoporosis and senescence, so that possible complications, can be avoided.

Even relatively healthy people with severe osteoporosis and compression fractures of the spine will frequently show rapid deterioration when because of pain they cannot be gotten out of bed. Severe pain and muscle spasm, inability to use the bedpan and loss of urinary control will sometimes produce a triggerlike effect in an aged person who has done well all his life until fracture of the spine has occurred. Such a patient may never really recover his previous well-being because of complications induced by the combination of osteoporosis, fracture and aging.

The frequency of osteoporosis in our aged population has made it the subject of a great deal of research today. Better understanding of bone metabolism is needed if we are to find the means of decreasing its prevalence.

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ETIOLOGY

The pioneer work of Albright and Reifenstein[2] seemed to prove that osteoporosis was due to a defect in matrix production, associated with relatively less anabolism due to aging and leading to decreased bone formation. This theory held sway until the last decade, when other factors in the production of osteoporosis were proposed. Consideration of the possible causes must include the various cellular and other organic components of bone, as well as disturbances of mineral metabolism. Failure of bone formation or an alteration in the rate of resorption may be due to disorders in cellular function, or of matrix production, or of the various factors regulating the mineral phase of bone. Investigation of these various factors has tended to reduce the emphasis formerly placed on the Albright-Reifenstein theories to the category of an ancillary cause of osteoporosis. Nordin[25] and Whedon[14] have expressed the opinion that a high calcium diet may help in the treatment of osteoporosis, and Nordin has stressed the possibility of a dietary deficiency in the intake of calcium as the cause of osteoporosis, basing his opinion on the effect of varying calcium diets in animals. Still another etiologic concept, which is gaining favor, has been expressed by Ray,[26] Jowsey,[17] and by Nicholas and Bronner[6]; based on evidence from balance and kinetic studies this concept holds that osteoporosis is due neither to a defect in matrix production nor to low calcium intake but to an accelerating ratio of resorption to deposition of bone with aging. Saville[28] has shown by sampling bone from the iliac crest in cadavers that there is a rapid rise of mineral components of bone in youth to adolescence, and that the level of calcium content is maintained in women until the mean age of 49, which corresponds to the mean age of the menopause, after which there is a fairly rapid decrease. In men the level seems to be maintained up to the age of the late 60's or early 70's.

From what has been said, one can readily understand that in any attempt to relate the decrease in the amount of bone in the aging human being to the etiologic factors, one must consider the various components involved in bone metabolism. At present, knowledge of the role of these factors is still fragmentary. The metabolism of calcium is one entity which can be studied from ingestion through transport to excretion; yet phosphorus metabolism and the controlling influence of parathormone, as well as the part played by various enzymes (such as alkaline phosphatase), vitamin D and proteins, are factors which also will affect the state of the calcium economy and the role of bone formation and destruction.

In the last few years there has been a shift of research toward molecular biology, and from these studies has come the knowledge of calcium crystallization on collagen fibers and its structural affinity for crystallization on nodal points of these fibers. More and more knowledge is accumulating from the use of tissue culture, microradiographic and histochemical technics regarding the basic processes of life and death of cells, all of which play a part in any consideration of osteoporosis. The effect of aging in itself involves still other considerations in relation to osteoporosis, such as fibroblastic proliferation, degeneration and avascularity as they affect bone. Inactivity, with loss of physical stresses, which are necessary to guide bone formation in consideration of Wolff's law, must play a part in the production of osteoporosis, and this factor at present is not given enough consideration. Therefore, osteoporosis appears as a different process to the clinician, the radiologist, the histologist, the biochemist, the microhistologist and the molecular biologist. There is a great tendency for the men working in each of these disciplines to study osteoporosis from his own separate point of view without giving heed to the whole. For this reason the clinician is unable to give a comprehensive synthesis of the cause of osteoporosis and

must speak in general terms, such as increasing bone resorption or decreasing bone formation.

The etiology of osteoporosis remains an enigma because fundamentally it deals with the formation or resorption of living tissue, and thus it becomes a complex problem. When the clinician has to deal with osteoporosis, he must consider the role of the components of bone physiology and how the aberrations of each may play a role in the individual case. He must give consideration to the organic, the inorganic and the cellular components of bone, and the various controls exerted upon them by the mechanisms of the body, realizing that a defect in any one of these segments of the bone physiology will be capable of producing too little bone.

Another way of looking at this problem is simply that, with the advance of age, resorption of bone outstrips the processes of repair and bone formation, so that bone gradually rarefies. This rarefication does not necessarily mean that the resorption of bone is abnormal with increasing age, but one must try to find the various elements in each case that are responsible. In some cases it may mean that there is a decrease in the availability of mineral in areas of the skeleton and that the bone has become more and more inorganic and less exchangeable, something that is known to occur in old age.

BONE DEPOSITION AND BONE RESORPTION

The process of aging has been shown to be accompanied by calcification of some tissues, such as arteries and hyaline cartilage. There seems to be a direct linear relationship between this process and age as demonstrated by Lansing[18] in the case of arteries and Hass[13] in the case of cartilage. A proportional depletion of chondroitin sulfate in articular cartilage with increase of age has also been reported, as well as a progressive increase in the structural protein-ground substance ratio.[16] What effect this change may have on the formation of matrix is not known.

The effects of increased or decreased oxygen tension in the tissues on bone metabolism must also be considered. Such states can be related to the integrity of the blood supply of bone. A high concentration of oxygen in tissue culture was found by Goldhaber[12] to induce an initial rapid and progressive resorption in 2-day-old mouse calvaria. This was due to increased cellular activity rather than to a degenerative process. In fact, in the late stages of resorption, when macrophages and osteoclasts developed, bone resorption tended in such cultures to become minimal. He concluded that bone resorption resulted from marked cellular activity and suggested that increased oxygen tension modified the metabolic pathways in such a way as to enhance resorption. Gaillard[10] also has shown that in tissue culture parathyroid extract was capable of initiating resorption.

There is very little information concerning factors which might inhibit resorption. Tonna[30] has shown in rats that with increasing age the osteogenic layer of the periosteum contains fewer cells and that there is a decrease in the number of osteoblasts. There is also a decline in the number of mitochondria, and this is associated with a drop in the respiratory activity of the cells. On the other hand, Frost[9] has recently reported that he has found an increasing number of osteocytes in aging human bone.

As mentioned previously, dietary factors may be involved in osteoporosis. Liu and McCay[20] in experiments on old dogs experienced difficulty in maintaining a positive calcium balance unless the diet was very rich in calcium. Some individuals have inferred from these studies that with aging individual humans require a larger amount of calcium and phosphorus than in youth.

The role of hormones in the processes of bone formation and bone resorption has been studied for many years. Balance data in studies by Albright and Reifenstein re-

vealed the effect of sex hormones many years ago. Their results have been confirmed by other investigators. Ackerman[1] and Bogdonoff[5] reported that nitrogen retention increased in patients with negative nitrogen balance when they were given estrogens and testosterone, but when the patients were in positive balance, there was no such increase. Most of this work was based on balance studies, which showed that estrogens increase calcium deposition. These views have won a high level of clinical acceptance. But it should be pointed out that there are few long-term studies available which can point conclusively to long-term gains in calcium storage on sex hormone treatment.[15]

The effects of adrenal hormone in causing demineralization and increased calcium output have been demonstrated in patients suffering from Cushing's disease and in others who have been treated with cortisone.[29] The exact mechanism of this action has not been shown, although it has been suggested that it is related to the profound negative nitrogen balance which is induced and a depletion of matrix available for bone formation. Cortisone-treated rats excreted more labeled amino acid N/5 glycine fluids than controls. Carlsson interpreted this result as meaning a less effective utilization of amino acids. Carlsson and Bauer[4] showed that in the rat adrenalectomy produced a marked change in the skeletal distribution of calcium 45, and thought that accretion and resorption of bone salt were enhanced in the same proportion to bone.

Studies by Comar,[8] Amprino,[3] LeBlond[19] and Carlsson[7] with P-32 and calcium 45, both by in-vivo and in-vitro experiments, demonstrated that when isotopes were incorporated in bone at the time of its formation they remained in the bone throughout its biologic life, and that the calcium exchange reactions were confined largely to the areas of newly formed well-hydrated bone. Robinson[27] showed that with aging the hydration of bone decreased. This decrease reduced the amount of exchange reaction. Neuman and Neuman[21] found that the calcium crystals became effectively isolated in the body fluid without relation to their potential surface exchangeability.

Isotopic studies have made it quite clear that when calcium enters the body, it distributes throughout the entire calcium pool, and that excretory mechanism and bone formation and resorption to a large extent, as well as intake and absorption, regulate the size of the pool. However, it is obvious that there are many cellular mechanisms and factors which have been alluded to, whose effects on such a pool at this time are not known. Current research stresses the factors which regulate the pool size and behavior.

POSTURAL CONSIDERATIONS

From a clinical standpoint, the occurrence and the development of osteoporosis in an individual is strongly influenced by postural considerations. According to Wolff's law, mineral is deposited in bone in response to the stresses of weight-bearing. Postural deformities of the spine of long standing determine to a considerable extent the lines along which the bone will be deposited in the vertebrae, and also the areas where atrophy or absorption will take place. The postural deformities, which can have a bearing on the aged osteoporotic spine, include structural scoliosis of greater or lesser degree, round-back deformity resulting from multiple epiphysitis in adolescents or Scheurman's disease, and old trauma or infection of certain of the spinal segments. Vertebral wedging develops and becomes fixed, and with aging the intervertebral disks at the points of maximum deformity become thinned, with resulting increase in each of the curves. The saying that structural scoliosis does not increase after the skeleton has matured does not hold true in the case of some of the conditions of aging, especially when associated with osteoporosis. Many

individuals with scoliosis lose height with increase of the deformity.

Deformity in such instances develops in the planes of stress and pull from the center of gravity of the body. For example, in a round back the curvature on the concave side becomes greater with increase of the round-back deformity, as well as of the lordosis. There is also a concomitant increase in the cervical lordosis and production of the so-called "widow's hump." This produces abnormal wear on the intervertebral disks, which become narrowed, and leads to considerable fixation of the joints of the cervical spine in extension.

In scoliosis, also, the curves may increase, first as a result of intervertebral disk degeneration, and then with osteoporosis there is increased wedging of the vertebral bodies. The ribs migrate further down on the concave side of the curve, so that a large segmental contact of bone with the rim of the pelvis results, and rigidity increases. The anteroposterior diameter of the chest often becomes increased. This enlargement is frequently associated with the development of emphysema in older life, and the vital capacity of many of these patients is diminished. The sternum bulges prominently and becomes sensitive, and frequently the costal joints become calcified.

The patient often presents an appearance of round back, short stature and protuberant abdomen. This condition is frequently associated with degenerative disease of the posterior facets of the lumbar spine. There is narrowing of the interlaminal spaces, and the spinous processes in the lumbar area lie in close contact with each other, a condition known as "kissing spines," which frequently produces pain in the lumbar area.

In the dorsal area the direction of the deformity is opposite, with increased round back. In such cases there is wedging of the anterior portion of the vertebral bodies, and there is stretching of the interspinous ligaments and of the fascial planes overlying the rib. It is quite common to find thickened and gritty nodular bands developing around the scapulae at the points of attachment of muscles, and the scapular margins are frequently tender. Because of the rounded curve of the upper thorax in these individuals, the plane of motion of the scapula is diminished, and these people will have limitation of movement of the shoulders, particularly in elevation and external rotation, and they will often complain of pain in the region of the shoulder.

It is important that these postural deformities be considered in formulating any therapeutic program. Frequently, bracing of the patient or good corseting will do as much to increase comfort by overcoming some of the postural problems as would diet or drugs. Also, one should not overlook the fact that exercises to develop stretched muscles that are needed for better spinal support, as well as improved respiratory excursion, can help in these cases. Although the patients may be elderly, and there may be difficulty in obtaining cooperation, nevertheless the effort should be made, and many times it proves to be rewarding.

SYMPTOMATOLOGY

The presenting symptoms of osteoporosis are well known. The type and the character of the pain varies and cannot be regarded as specific. On the other hand, in many cases each vertebral collapse is associated with sudden onset of acute pain, usually radiating around through the flank into various areas of the abdomen, depending on the level of fracture. Such pain is generally aggravated by movement or coughing. Rarely does the pain radiate to the leg. Frequently, it is described as coming in spasms, during which the patient must remain immobile, and any movement is apt to be agonizing. Other patients have persistent, dull, deep-seated and ill-defined pain in the back, generally worse when they are erect or walking. There are variable patterns of pain, and it is difficult to state that any type of pain is characteristic, except the girdling

pain that is associated with acute collapse. However, acute episodes associated with fresh compression of vertebrae and accompanied by spasm are well known, and it is an acute attack like this that is generally responsible for admission to the hospital of older patients with osteoporosis. Ileus and constipation are frequently present with such attacks, partly perhaps as a result of retroperitoneal hemorrhage, but more often the result of inability of the patient to make any muscular effort to aid in bowel evacuation. Appetite is diminished, urinary output frequently decreases, and there is the need for strong sedation to relieve pain.

Many other patients do not have acute pain but complain of postural backache with attacks of discomfort lasting generally a few days and then subsiding. Multiple bone tenderness is found frequently, and this may be over the xyphoid, in the sternocostal area, over the posterior portion of the rib cage, beneath the scapulae and over the spinous processes of the vertebrae themselves. Lumbosacral and lower lumbar tenderness are almost always present.

The response of osteoporotic patients to bed rest is variable. Many patients with acute onset will note that the pain disappears rather rapidly after a week or 10 days, but it generally recurs with return to activity. Still others improve rapidly with rest, only to relapse frequently.

Loss of stature in most of these patients is common, and it is always useful to keep a record of height and weight. While loss of height with aging is almost universal, such losses usually amount to less than an inch, whereas in the osteoporotic patients the loss may be several inches. Patients are often inaccurate about their previous height. Some patients, who are aware of their height from previous military service, may lose more than an inch without being aware of it. A great deal of height loss is caused by postural slumping, that is, increase of existing curves, which is a common occurrence in the osteoporotic.

ROENTGEN FINDINGS

Upon roentgenologic examination, the osteoporotic spine commonly shows, even before structural changes occur, a general washed-out appearance of diminished bone density. The cortical bone is thinned in the vertebral bodies, and there is a loss of the trabecular markings, often associated with a fine striated vertical pattern. At this stage the patient usually is not complaining of painful symptoms referable to the spine, but the clinician will say that the patient is developing osteoporosis. Unfortunately, not too much reliance can be placed on the diagnosis at this stage because so many variables are introduced in making an x-ray examination. These variables include the time of exposure, the distance at which the tube is placed, the amount of power used in the examination, the positioning of the patient and even the technic of development of the films. Other variables include the habitus of the patient, whether fat or thin, whether muscular or obese—in other words, the amount of tissue to be penetrated and the density of that tissue.

Many efforts and experiments have been made by the roentgenologists to determine the mineral density of bone by standard types of examination, but the results of these efforts have been disappointing and unsatisfactory. It simply is not possible to exclude variance in the factors involved in the examination. Some investigators have endeavored to determine bone density by reference to marking materials, which are included on the films, such as ivory or aluminum, but this attempt has not proved to be successful. It is possible that improved technics may be developed, and this development is greatly to be hoped for, because at the present stage we can recognize osteoporosis only when it is fully established and probably in an irreversible condition. Nor can serial roentgen-ray examinations of certain areas of the spine at different dates be used for comparison to show whether or

not improvement has occurred as a result of treatment. In fact, one of the great points of weakness about treatment, particularly with reference to the use of estrogens or other hormones, is that it has never been possible to show by roentgen-ray examination that improvement in bone deposition has been obtained.

One may be right in concluding from inspection of an x-ray film that the spine is osteoporotic even before structural changes have developed; but to be certain in making the diagnosis one must always look for confirmation to the secondary changes that are brought about by structural collapse of the bone. Also, in studying from time to time the progress of the disease in a patient, one must look for additional structural changes brought about by the pathologic condition, to be able to say that the patient is worse.

The most typical early change seen in the x-ray films is ballooning of the intervertebral disks. This may involve only a few vertebrae in the beginning; but in the later stage, when many vertebrae are involved, the spine may show a condition that is called "fishtailing" or "codfish vertebrae." The ballooning and fishtailing are caused by the weakness of the subchondral plates of the vertebrae under the hydrostatic bulging pressure of the nucleus pulposus. The next change to occur is actual vertebral collapse, with wedging as a result of loss of the anterior vertical height of the vertebral body. In the extreme case there are multiple compression fractures, both in the dorsal and the lumbar areas, and generally this state is followed by collapse of the intervertebral disk, due to extrusion of a nucleus into the soft structure of the vertebral body. In the course of time there is osteophytic reaction about the margins of such collapsed disks.

In the case of a single vertebral collapse, particularly following slight trauma, one looks to find in the lateral view irregularity of the anterior cortex. When there are multiple compressions of long standing, the anterior cortical irregularity is overshadowed by productive changes and osteophytic lipping at the anterior margins of the vertebral body. When making a comparison of the progress of the disease from time to time, one should count the number of collapsed vertebrae; and it is helpful in doing the counting to number each vertebra in the dorsal and the lumbar areas on the films for easier comparison.

DIFFERENTIAL DIAGNOSIS

A number of conditions will produce pathologic fractures of the spine in the aged besides osteoporosis, and these must always be considered before arriving at a diagnosis. By far the most common of these are metastases from malignant tumors and multiple myeloma. Multiple compression fractures are common in all of these conditions. A primary tumor arising in a vertebra, such as an aneurysmal bone cyst or a hemangioma, usually involves only a single vertebra; therefore, the collapse is localized. Among other conditions which may result in vertebral compression and therefore simulate osteoporosis are hyperparathyroidism, polycythemia vera, hemolytic anemia and ankylosing spondylitis. Vertebral compression fractures have also been found in osteomalacia, tuberculosis, Paget's disease and infiltrative lesions, such as Gaucher's disease. The diagnosis of osteoporosis can be made only by the exclusion of the other conditions which may produce vertebral compression.

Although it is true that osteoporosis is the most common explanation of vertebral compression in the aged spine, and that the frequency of this condition is such that most of the patients in a nursing home for the aged were reported to have shown it,[11] still it is incumbent on the physician to make sure that he is not overlooking some other condition as the cause of compression. In patients under the age of 55 a diagnostic work-up is particularly required, because it has been shown that the chance of a vertebral compression's being due to osteoporosis

at that time is only about 1 in 10.[24] On the other hand, if the patient is over the age of 65, the chances of the condition's being due to osteoporosis rather than any other condition are about 7 out of 10. If the number of fractures counted on the roentgenologic films is less than 3, the likelihood of the condition's being due to a malignant disease is very great until proved to be otherwise. In osteoporosis there are usually multiple vertebral compression fractures, and not just 1, or 2, as in early malignant disease. Multiple myeloma, which frequently occurs in older life, often produces destructive and lytic areas of bone throughout the spine, and there may be many fractures.

It is often helpful to note whether or not there is evidence of sclerosis in the vertebra or any other evidence of repair of fractures. Such a finding indicates that malignancy is unlikely. Extreme triangular compression with soap-bubble appearance or the presence of lytic areas suggests tumor more than osteoporosis, as does pedicle destruction.

The diagnosis of osteoporosis cannot be made through any single diagnostic test. As mentioned before, it is a diagnosis arrived at only by exclusion. The proper work-up of a patient with compression fractures includes a number of diagnostic tests which are shown in the following outline:

COMPRESSION FRACTURES

Medical History

- Activity versus bed rest
- Duration of symptoms, onset and type of precipitating trauma
- Menstrual history, pregnancy, breast feeding, hysterectomy
- Decrease in height and appearance of slouch
- Gynecologic, gastrointestinal, or genitourinary disease
- Food intake, especially calcium in various forms
- Medication, especially corticosteroids

Physical Examination

- Posture
- Multiple bone tenderness, specific localization in the spine
- Motion
- Height, weight
- Motor or sensory changes
- Evidence of systemic disease, especially in the gastrointestinal tract

Laboratory Tests

- Complete blood count, erythrocyte sedimentation rate and urine
- Serum proteins:
  - Albumin-globulin ratio
  - Electrophoretic pattern
- Serum calcium, phosphorus and alkaline phosphatase
  - 24-hour urine calcium excretion on varying intake
- Other special tests where indicated (stool fat content, renal function)
- Radioisotopes, absorption loads

Roentgenograms

- Chest (all patients)
- Spine, skull, hands or other indicated areas
- Special studies, where indicated:
  - Intravenous pyelogram
  - Gastrointestinal

Biopsy

- Marrow, sternal or iliac
- Direct vertebral or other

We feel that all patients with painful symptoms, associated with vertebral compression fractures, should have a sternal or iliac marrow biopsy performed. This can be done with little or no discomfort, and the results are usually rewarding. Direct vertebral body biopsy is sometimes required when the marrow biopsy is negative, particularly when the sedimentation rate is elevated or the alkaline phosphatase is higher than normal, or when there is an

increased level of calcium excretion. Infection such as tuberculosis or low-grade osteomyelitis should not be overlooked as possible causes of the condition.

A number of reports in the literature show how vertebral biopsy has helped when the previous diagnosis was doubtful. In the lumbar area such a biopsy may be made of the spinous process, and, if necessary, the vertebral body can be readily reached retroperitoneally through a kidney-type incision. In the dorsal spine it is possible to approach the vertebral body by performing a costal transversectomy. Direct surgical approach of these areas for purposes of biopsy may appear to be somewhat heroic, and many surgeons prefer to make an aspiration needle biopsy, following the technic of Craig.

In patients in whom malignant disease with metastases is strongly suspected, a roentgen survey of the skeleton should be carried out, and particularly the skull should be examined for the presence of lytic areas. The finding of multiple radiolucent areas in other bones is indicative of myeloma or metastatic disease.

In the presence of a normal sedimentation rate and blood count and no abnormal chemical findings in the blood or the urine, and with only mild symptoms and no evidence of systemic disease, and when the patient is over the age of 65, one is probably justified in treating the patient expectantly with the diagnosis of osteoporotic compression fracture. However, such patients should be rechecked over intervals of several months to be sure that no change which might indicate the possibility of malignant disease has occurred in the pattern.

TREATMENT OF PATIENTS WITH OSTEOPOROSIS

The therapeutic problem in patients with osteoporosis may be summarized as the treatment of bone weakness and spinal fractures in an elderly person who frequently has other health problems. Unfortunately, it is generally true that by the time the doctor is able to make the diagnosis the disease is already well advanced and irreversible. We must admit that at the present time there is no cure for osteoporosis. No evidence has been produced to show increasing mineralization or bone production as a result of treatment. On the other hand, the bone still possesses its reparative power, and fractures in elderly persons will heal almost as well as in younger people. Therefore, we must treat the fracture according to well-established principles of rest and support. We must also treat the osteoporosis in the best manner that we know, and we must treat the patient as a whole from the standpoint of existing complications and along the lines which are best calculated to improve health.

During the acute phase, which is generally of sudden onset and attended by severe pain, rest on a hard bed with fracture boards and limited use of pillows is the best treatment. It is true that recumbency and bed rest give rise to calcium loss from the skeleton, but this is on a small scale and is unimportant in comparison with the relief of pain usually obtained. In a case of recent compression the bed is the best splint that can be used. On the other hand, we feel that to treat the patient purely as a fracture is a mistake, and that we should not aim to obtain hyperextension or correction of deformity. Attempts to fix these spines in plaster of paris jackets cause too much discomfort and are harmful to the patient's general condition. More than half of the patients treated by the authors required from 2 to 4 weeks of strict bed rest and from 4 to 6 weeks of hospitalization before being made ambulatory.[23] When adequate nursing care can be provided at home, there is no need for hospitalization.

The patient's position in bed is not of great importance, and change of position at frequent intervals is desirable. Generally, the patient experiences the greatest comfort when lying in the supine position with a

pillow under the knees to relax muscular pull and leverage on the pelvis. We try to avoid the semiflexed position; and if the patient's head and trunk must be raised, the elevation should be as little as possible, and pillows should be restricted to 1 or at most 2.

A hospital bed, in which the upper body portion and the knee section can be elevated, is of much help, and the modern electrically operated bed, which the patient can control, is a great comfort. The use of an overhead fracture frame with trapeze for hand pull is also considerably helpful. Little advantage has been obtained from the use of traction.

Care of the bowels in patients with osteoporosis is generally a problem, because the use of a bedpan is a frightening and painful experience. Under such circumstances the patients are unable to use the extrinsic musculature to aid in evacuation. In our experience, getting the patient out of bed and using a commode chair, placed conveniently at its side, is easier than subjecting the patient to the ordeal of trying to use the bedpan. Care is necessary to avoid fecal impaction, and laxatives, together with enemas, must be used at regular intervals.

The length of recumbent treatment must be determined by subsidence of acute symptoms. With time and rest the patient finds generally that movement in bed becomes easier, and it is possible to proceed from this state to experiments in getting the patient out of bed for short periods to see how well the vertical position and movement are tolerated. The height of the bed from the floor should be lowered so as to make it easier to get in and out. A straight-back chair with arms should be used, with a pillow at the back, as indicated. The patient should be instructed in deep-breathing exercises, and if these can be tolerated and the patient is cooperative, they are of considerable help. Usually the patients complain of acute pain on first standing; but as they move about, this pain becomes less. Getting up from the bed or the chair are the things that provoke the most pain. Sometimes the use of a walker or crutches or cane can be of value. The duration of periods of ambulation and activity should be very limited in the beginning; gradually, as tolerance increases, the periods can be extended. It is generally a mistake to let the patients sit for more than 2 hours at a time in the early period without an interval of bed rest.

The principle of spinal bracing should be applied insofar as the patient tolerates it. On theoretic grounds a rigid spinal brace of the Taylor type would be the best aid that could be employed. However, many of the patients are thin, with bony prominences, and do not tolerate such rigid supports. Also, one must admit that rigid immobilization, sufficient to eliminate movement and pain, cannot be obtained with any brace. On the other hand, good types of orthopaedic corsets do give considerable support of the lumbar spine and the abdomen, and most of all they help in improving spinal posture. These are particularly helpful in semiobese patients. For the thin person we have more often employed light lumbar spinal braces. In the case of a patient who is developing severe round-back deformity with considerable loss of height, long braces of the Taylor type, or with anterior chest-wall compression of the 3-point type, are usually required; but even these are powerless to prevent gradual increase of deformity in some cases. Many patients state that they feel better when they are wearing a support of one type or another. Some patients claim that they can stay up for longer periods of time with them than without them. As patients improve, they are apt to want to get rid of their braces, and we have allowed them to do so. We do not feel that the use of braces will prevent further fractures; on the other hand, the greater activity permitted by brace removal may bring about improvement in health.

It is difficult to estimate the value of hor-

mone therapy in osteoporosis. Its use dates from the work of Albright and Reifenstein, but recent work in laboratories, particularly with the use of radioactive calcium and strontium, have not completely substantiated this earlier work. However, we must remember that it is difficult to evaluate laboratory data. On the whole, clinicians still feel that hormones are valuable, even though it has not been possible to show increased bone deposition as a result of their use.

Estrogens have been shown to increase calcium balance in the studies available today. When estrogen is used, it should be in small quantity; and under such circumstances it is usually well tolerated, will do no harm and may be of considerable benefit. When it is used in excess, vaginal bleeding may develop, even in aged patients, and if the patient is not properly briefed in advance, considerable emotional reaction may result. Swelling of the breasts and occasional discharge from the nipples are other complications from its administration. Then it is necessary to stop the therapy for a time, and if it is to be resumed, androgens should be administered with the estrogen in an effort to counteract this side-effect. Androgenic therapy has not been shown to have any effect on calcium balance but is much more potent in reversing negative nitrogen balance. On the other hand, estrogens have little effect on increasing nitrogen balance. Whether or not increased nitrogen balance has any influence in favoring matrix formation is not known, but experience with hormone therapy has led to the adoption of a program of treatment with mixed hormones that is very prevalent today. Most patients are able to tolerate a small dose of estrogen and androgen. Many preparations with a combination of these hormones are available today, and those most favored are depot preparations in oil given by intramuscular injection at monthly intervals. Their use ensures that the patient is getting the recommended treatment and renders easier the evaluation of the patient's response to this treatment.

The question is frequently asked: How long should hormone therapy be continued? If one commits oneself to the desirability of hormone therapy, then one must adopt the conclusion that it should be used as long as possible, and possibly, indeed, throughout life. The authors have had patients on hormone therapy as long as 9 years without apparent harm. Although there was no demonstrable remineralization, it must be remembered that the only criteria employed in making this statement was evaluation by roentgenogram and the study of additional structural failure in the spine. Estrogen does not have the capacity to increase endo-osteal bone formation in humans in the same way as it does in birds and rats. If it can be shown that it decreases bone resorption—in which case it may be claimed that it prevents increase of the disease, and in which case the skeleton would remain on the same level of porosity as it was at the time the diagnosis was first made—then it can be claimed that hormones may act as a brake on further loss of mineral. Although in most instances the use of hormones will produce a drop in the urinary calcium, this does not necessarily prove that there is increased deposition of calcium in the skeleton. Therefore, until it can be shown definitely that the hormonal changes brought on by the climacteric result in loss of minerals from the skeleton, no definite answer can be given as to the effect of hormonal therapy in counteracting osteoporosis. That hormones may be of value in improving the psyche of the patients and by increasing the appetite and the sense of well-being seems to be evident in clinical practice. We agree in recommending that hormones be used in stubborn cases when well tolerated. The dosage must be determined by experiment, and whatever dose is best tolerated is probably the dose of choice.

The role of diet in the treatment of osteoporosis is not clear. It is certainly logical

to try to increase the calcium intake in patients who on the basis of their history are taking inadequate amounts. From our interviews with patients a third were on a low calcium intake not reaching 500 mg. a day. In older patients it is common to find a history of inability to drink milk and of apparent gastric intolerance. In such cases a trial of skimmed milk should be recommended, and a level of 2 glasses a day will provide an adequate calcium intake. If skimmed milk cannot be tolerated, then the use of wafers containing calcium should be advised, although so many wafers are necessary for an adequate intake that many patients will not cooperate. Some investigators have claimed that the calcium requirements in older life are greater than those in younger people, and if this claim be true, then the intake of calcium will have to be increased to as much as 2 to 4 grams daily. At the present moment we feel that the need is met by taking 2 to 3 glasses of milk daily, about 500–750 milligrams of calcium.

The role of proteins in the diet of patients with osteoporosis has not been stressed of late as much as it was formerly, since it is now felt that as long as the patient is not in a negative nitrogen balance, increasing the positive balance will not augment mineral deposition. A good level of nutrition should be sought in thin patients, and decreased caloric intake and weight loss should be advised in others whose weight is above average.

The use of drugs known to induce a negative calcium balance is to be avoided, and this directive applies particularly to cortisone or its derivatives. When the use of such drugs is indicated, as in ulcerative colitis or other conditions, it may be advisable to administer mixed hormones of estrogen and androgen, as these have been reported to inhibit the calcium excretion produced by corticosteroids.

Physical therapy can be of considerable importance in most patients with osteoporosis. This should be used chiefly in the form of muscle re-education and exercise to increase muscle tone and maintain function in the various articulations. We have made little use of massage. Of course, heat in one form or another, the oldest form of physical therapy, is always comforting when applied to the spine, either as dry heat or moist hot packs. We do not feel that there is any particular benefit to be obtained from the use of diathermy or microwave treatments. Deep-breathing exercises, abdominal and gluteal contractions, as well as hip-flexion exercises, are helpful when tolerated. The greatest problem is to obtain cooperation in these elderly patients, chiefly because they have been inactive for years and simply do not understand the principles of exercise. On the other hand, it has been remarkable to observe the progress made by some patients who responded to the instructions of the physical therapist and cooperated in carrying them out several times daily.

Exercises to correct postural deformity and obtain better standing position are probably of greater importance than anything else in the prevention of further deformation. However, to obtain success, it is necessary to stimulate the motivation of the patients who are not accustomed to exercises, and the doctor can do a great deal to reinforce the efforts of the physical therapist in obtaining cooperation. It is unfortunate that the use of such exercises cannot be begun at a much earlier period, when probably much could be accomplished in the prevention of later deformity.

In treating osteoporosis in older patients, it is important to avoid overlooking other conditions that should also be treated. Many of the patients will be found to have severe osteoarthritic deformities involving the joints of the lower extremities or the shoulders or the hands. Limitation of movement in these joints or pain on motion may make it much more difficult to make patients ambulatory. Diseased joints of this type should not be allowed to stiffen from disuse during the necessary period of bed rest and

recumbent treatment, and in general the best treatment for these joints is exercise done every day.

Surgical Measures

Occasionally, patients are encountered with severe radiating pain, chiefly down the hip and the leg. Such a condition may be brought about by irritation of one of the lumbar nerve roots from bony or osteophytic compression. In such cases decompression of a nerve root by laminectomy and appropriate surgical measures may obtain dramatic improvement of the radiating pain. It will not bring about any relief of back pain due to osteoporosis, and in a few instances, which the authors have reviewed, such pain has continued to be a problem. Rhizotomy or root section is another measure that may be employed in stubborn cases. The authors recall one patient in her late 70's, with old scoliosis and osteoporosis and aggravated pain radiating into the buttock and the leg, who required cordotomy after previous rhizotomy had failed. This patient was completely relieved by this operation. We also recall one patient who was in good general health, except for recurring attacks of compression of single vertebrae in the dorsal area, in whom spinal fusion was performed with benefit. In general, we would advise conservative approach to problems of this kind, and the examples are cited simply to show that exceptional cases are seen from time to time which require exceptional measures. The authors have had no experience with the operation that has been reported by some authors for relieving pain caused by impingement of the spinous processes against each other—the so-called "kissing spines"—by resection of the spinous processes.

Local injection of lidocaine or other analgesics and cortisone has been found to be of some benefit in the interspinous areas when there has been acute tenderness in the area, and it was felt that the symptoms of which the patient complained were not due to the compression fractures, shown by roentgenogram. Similar injection treatment may be indicated in cases of localized tenderness and limitation of movement that are thought to be caused by tendonitis.

Roentgen-radiation therapy has been reported as useful in the treatment of some of these patients, but this has not been advocated in recent years, and the authors would recommend against it, because there is no evidence that it can have any specific effect on the condition.

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Summario in Interlingua

Osteoporosis del Vetule Rhachide

Osteoporosis es un importante morbo proque illo es associate con le population de avantiate etates con tendentias profractural e un frequente co-existentia de altere conditiones pathologic. In tal casos, fracturas es importante, per consequente, proque illos pote initiar un catena de evenimentos per complicationes resultante ultimemente in severissime morbos medical.

Un revista del etiologia de osteoporosis revela que le causa non es cognoscite, sed il existe certe indicationes que factores hormonal e un declino in le utilisation de calcium ha un certe rolo in le production de iste condition. Altere factores que es importante sin dubita es le factor invetulatori del ossos mesme e le metabolismo de calcium e su transporto.

Osteoporosis in nostre dies non es un morbo curabile. Tamen, adequate mesuras

therapeutic pote prevenir su pejoration. Pro tractar osteoporosis adequatemente il es importantissime que on pote diagnosticar le morbo in stadios precoce a base de evidentia clinic e radiologic. In le area del observationes clinic, abassamento del statura, deformitate ronde del dorso, e dorsalgia precede frequentemente fracturas de compression. Alterationes secundari de osteoporosis del rhachide produce considerabile grados de disconforto e de alteration in le musculatura abdominal e inferior e etiam in le rhachide lumbar que tende a devenir plus lordotic. Dolores in osteoporosis es capace de grande variabilitate, e il non es facile in certe casos relationar le sito del dolor can le loco del fractura in le rhachide. Osteoporosis in altere ossos, particularmente le cervices femoral e le humeros produce morbo degeneratori como illo lo face in le rhachide ubi morbo degeneratori de discos es frequentemente aggravate per osteoporosis.

Le diagnose differential de fracturas de compression in vetulos debe considerar—a parte osteoporosis—multiple conditiones. Malignitate metastatic, myeloma, anemias hemolytic, spondylitis ankylosante, osteomyelitis, e infectiones pote omnes esser confundite con osteoporosis. Spontanee fracturas de compression in patientes de etates de minus que 55 annos es rarmente causate per osteoporosis. Sed con le avantiamento del etate, iste possibilitate deveni marcatemente plus forte. Le diagnose de osteoporosis debe esser establite per le exclusion de omne le altere possibilitates. In le processo diagnostic on debe utilisar biopsias, examines de medulla ossee, e etiam le usual mesuras laboratorial.

Le tractamento de osteoporosis a iste tempore non es supportate per un demonstration scientific del possibilitate de remineralisar osso. Tamen, le uso de hormones sexual, de adequate ingestion de calcium e proteina in le dieta, e de vitaminas es elementos standard in le tractamento de osteoporosis depost multe annos. Exercitio, activitate, e supportos passive insimul con le prevention de deformitates additional es aspectos importante ben que ancillari del tractamento. Tamen, le plano therapeutic debe esser individualisate a casa del varietate de problemas medical que existe in multes de iste patientes.