CT scanning and osteoporosis prevention

Computerized tomography (CT scanning), according to researchers, promises to revolutionize the management of postmenopausal osteoporosis — spinal fractures caused by the loss of bone mineral content following menopause and that afflict one out of every four postmenopausal women. It would do so by providing routine assessment of bone mineral content in postmenopausal women — something not now available — and thus identifying women with dangerously low bone mineral content. Those women could then receive preventive medicine before fractures occur.

The new use of CT scans was discussed last week at an international symposium on clinical disorders of bone and mineral metabolism in Dearborn, Mich. The meeting was sponsored by the Henry Ford Hospital in Detroit, a leading bone research center.

The mineral content of the body's peripheral skeleton has been measurable for a number of years with two techniques, radiogrammetry and photon absorptiometry. Neither technique, however, is capable of measuring the mineral content of the spine, which is where postmenopausal bone loss and fractures usually occur. On the other hand, there is a new technique available at a few centers, called dual photon absorptiometry, that *can* measure the mineral content of the spine. Its disadvantage, though, is that it measures the mineral content of both kinds of bone present in the spine — trabecular and cortical — and it is trabecular mineral content which is lost postmenopausally and which leads to spinal fractures.

Thus Harry K. Genant, a radiologist with the University of California Medical School in San Francisco, and colleagues developed a CT scanning technique that can measure the mineral content of spinal trabecular bone only. Fifty medical centers in the United States and Europe are now using the technique to measure spinal trabecular bone in osteoporotic patients, while scientists at other centers are developing similar methods. But the most important thing, Genant stressed at the symposium, is that some 2,000 medical centers or hospitals throughout the world already have CT scanners and, with only slight and relatively inexpensive modification of their machines, would be capable of assessing the mineral content of spinal trabecular bone.

What's more, as scientists use CT scanners to measure spinal trabecular bone in osteoporotic patients they are learning what constitutes normal and abnormal postmenopausal bone loss and how much bone loss is necessary for fractures to occur. For instance, Borje E. C. Nordin of the Royal Adelaide Hospital in Adelaide, Australia, and co-workers used the CT scanning method developed by Genant and his team to study the spinal trabecular mineral content of 35 postmenopausal women with spinal fractures and of 50 age-matched control subjects. They found not only that there was less mineral content in the bone of patients than in the bone of controls, but that below a certain content level fractures occur. This cutoff point was not known before, Nordin said. Genant and his colleagues, on the other hand, used CT scanning to examine the mineral content of spinal trabecular bone in women who had experienced an artificial menopause by having both ovaries removed. As they expected, the women had lost considerable bone. Maximilian A. Dambacher of the University Clinic of Zurich and co-workers used CT scanning to determine the amount of trabecular bone in premenopausal women, healthy postmenopausal women and osteoporotic postmenopausal women. The premenopausal women showed no bone loss, the healthy postmenopausal women 0.9 percent bone loss per year, and the osteoporotic patients a loss of 2.7 percent per year.

All these developments, the researchers involved agree, are likely to revolutionize the management of osteoporosis. Specifically, the decline in estrogen production following menopause is known to somehow contribute to postmenopausal bone loss, and estrogen replacement therapy has been well documented as being capable of preventing postmenopausal bone loss. Yet currently few physicians are placing all their postmenopausal patients on estrogens because estrogens increase the risk of uterine cancer (SN: 1/3/76, p. 9). On the other hand, if physicians could routinely determine which postmenopausal women are losing dangerous amounts of spinal trabecular bone, they could then put only those women on estrogens and thus keep them from developing spinal fractures. —*J. A. Treichel*

Findings shed light on how ECT works

Electroconvulsive therapy, or ECT: in the imperfect science of psychiatry, it is perhaps the most mysterious of tools. Psychiatrist Frank M. Mondimore says, simply, "We don't know how it works." But Mondimore, of the Phipps Psychiatric Service at the Johns Hopkins Hospital in Baltimore, and a number of other researchers are among the first to uncover clues about the chemical actions of ECT in the brain and body.

More widely known as electroshock, ECT has been used for decades in treating severely depressed persons. While results have been mixed, it is apparent that some patients seem to improve, at least temporarily, following the administration of electric current to one or both temples to induce a seizure.

Just how or why such apparent improvement takes place remains unclear. But psychiatrists at the University of Arizona and elsewhere have recently linked ECT's antidepressant action to an increase of the patient's levels of beta endorphin, one of the "natural opiates" produced in the brain to combat pain and stress. Although the increase — measured in the blood serum of seven hospital patients — may be simply a reaction to the stress of being shocked, U. of Arizona researcher John Misiaszek says he thinks it represents "more than that. [Beta endorphin] acts in some way to help patients feel better after ECT," Misiaszek said in an interview at the recent meeting of the American Psychiatric Association in New York, where he presented his group's findings. The scientists found that the elevated blood levels present 20 minutes after shock administration dropped to their pre-ECT levels within 48 hours.

Misiaszek suggests that the plasma endorphin increase may "mirror a greater process that goes on centrally [in] the pituitary gland." It is possible, he says, that the brain may be getting even larger increases of beta endorphin than the blood levels would indicate. And while the full implications of this work have yet to be explored, Misiaszek says that more knowledge about the range of biochemical and physiological changes induced by electroconvulsive therapy might make it possible "eventually to substitute neurochemical [treatments] for ECT in the appropriate types of patients."

In their research, also presented at the APA meeting, Mondimore and his colleagues studied the blood serum of 20 patients to examine a negative consequence of ECT: the temporary confusion that often occurs in the 30 minutes to one hour following shock administration. Their findings suggest that electroconvulsive therapy somehow triggers a drug, injected prior to ECT to minimize risk of heart arrhythmia, to induce confusion. Atropine, one of a family of "anticholinergic" drugs, is given routinely to block receptors of the chemical acetylcholine in the vagus nerve of the heart. While they protect the heart during shock, such drugs are also believed to block acetylcholine receptors in the brain as well — an effect that could explain the post-ECT confusion.

Mondimore found that those patients with the highest anticholinergic levels also tested out as the most confused. Simply administering anticholinergic drugs without ECT triggers no such confusion, he says. The findings could lead to more appropriate dosages of pre-ECT drugs, he says, or to use of drugs such as physostigmines, which combat anticholinergic toxicity. —*J. Greenberg*