It was agreed that bone loss in women starts at or about the menopause and proceeds at about 1%/annum. In men it is slower and starts later. This loss of bone in women is associated with a steep rise in the wrist fracture rate (to about 50/10 000/annum) which does not occur in men. It was also agreed that this loss of bone is due to an increase in the rate of bone resorption rather than a fall in the rate of bone formation as Albright had originally postulated. However, there was some disagreement as to the way in which loss of estrogen causes bone resorption. Gordan thought that estrogens had an anti-catabolic effect on collagenous tissues in general and quoted reports of thin skin and reduced collagenous tissues in cases of osteoporosis. Nordin considered that bone was unduly sensitive to the action of parathyroid hormone in the absence of estrogens. Duursma thought there might be altered sensitivity of bone to growth hormone or thyroid hormone. Klotz’s view was that there might be a deficiency of calcitonin. Dequeker suggested that osteoporosis was associated with growth hormone deficiency and showed increased growth hormone levels in osteo-arthritic patients with increased bone mass.

**The Effects of Estrogen and Calcium on Osteoporosis**

Despite this disagreement on mechanisms, there was general agree-
ment that estrogen therapy delayed or prevented loss of bone in post-menopausal women, and might even increase it. Furuhjelm reported a comparison of 13 post-menopausal women given 4 mg of micronized estradiol daily who were studied by X-ray densitometry and compared with 13 untreated women. The treated cases gained bone and the untreated lost bone. Dequeker reported metacarpal cortical width measurements, both retrospective and prospective, which showed significantly less bone loss in treated than untreated patients. Gordan quoted the prospective trials of Aitken and Meema which showed the same estrogen effect, and reported a reduced fracture rate in his own estrogen-treated patients. Nordin reported a prospective controlled trial in which gamma-ray absorptiometry of the forearm was used. This showed significant bone loss in the control group of 15 cases but no significant loss over 2 years in the group of 15 cases treated with ethinyl estradiol.

There was some discussion about the role of calcium in the prevention of osteoporosis. Nordin reported that his prospective trial also included a group of women treated with calcium supplements, in which no significant bone loss occurred in 2 years. He also reported that oophorectomy had little effect on bone mass in rats unless combined with a low calcium diet, when a severe loss of bone could be produced. This suggested that estrogen deficiency impaired the organ’s ability to adapt to a low calcium intake. He thought that the beneficial effect of calcium supplements (and the reported effect of calcium infusions in osteoporosis) was due to parathyroid gland suppression and consequent reduced PTH-mediated bone resorption. However, Gordan considered that the effect of a calcium infusion on bone resorption was far too prolonged to be due simply to parathyroid switch-off.

In addition to the effects of estrogens and calcium, Dequeker reported that women treated for long periods with a progestagen had significantly more bone than untreated controls.

**Biochemical Effects of the Menopause**

Biochemical effects of the menopause were described by van Paassen and Duursma who studied pre- and post-menopausal women, the latter including treated and untreated cases. Their main finding was a rise in plasma calcium, phosphate and alkaline phosphatase after the menopause, which were to varying degrees reversible by estrogen