Medica Foundation, 1968:456–7.
5. The HSG, Steven MM, Van der Linden SM, Cats A. Evaluation of diagnostic criteria for ankylosing spondylitis: a comparison of the Rome, New York and modified New York Criteria in patients with a positive clinical history screening test for ankylosing spondylitis. *Br J Rheumatol* 1985;**24**:242–9.
6. Wright V, Moll JMH. *Seronegative polyarthritis*. Amsterdam: North Holland Publishing Co., 1974.
7. Moll JMH, Haslock I, Macrae IF, Wright V. Associations between ankylosing spondylitis, psoriatic arthritis, Reiter's disease, the intestinal arthropathies and Behçet's syndrome. *Medicine* 1974;**53:** 343–64.
8. Scott SG. *Adolescent spondylitis or ankylosing spondylitis. The early diagnosis and its treatment by wide-field X-ray irradiates*. Oxford: Oxford University Press, 1942.
9. Calin A. *Spondyloarthropathies*. Orlando: Grune and Stratton, 1989:1–8.
10. Moll JMH, Wright V. Normal range of spinal mobility—an objective clinical study. *Ann Rheum Dis* 1971;**30**:381–6.
11. Moll JMH, Wright V. Measurement of spinal movement. In: Jayson MIV, ed. *The lumbar spine and back pain*. 3rd ed. Edinburgh: Churchill Livingstone, 1987:215–34.
12. Pile KD, Laurent MR, Salmond CE, Best MJ, Pyle EA, Moloney RO. Clinical assessment of ankylosing spondylitis: a study of observer variation in spinal measurements. *Br J Rheumatol* 1991;**30:** 29–34.
13. Rigby AS. Review of the UK data for the rheumatic diseases—5. Ankylosing spondylitis. *Br J Rheumatol* 1991;**30**:50–3.
14. Ebringer A. The cross-tolerance hypothesis, HLA-B27 and ankylosing spondylitis. *Br J Rheumatol* 1983;**22:**(Suppl 2):53–66.
15. Geczy AF, Prendergast JK, Sullivan JS, *et al.* HLA-B27, molecular mimicry, and ankylosing spondylitis: popular misconceptions. *Ann Rheum Dis* 1987;**46**:171–12.
16. Schwimmbeck PL, Oldstone MBA. Molecular mimicry between human leukocyte antigen B27. *Am J Med* 1988;**85**:51–3.
17. Yu DTY, Choo SY, Schaak T. Molecular mimicry in HLA-B27-related arthritis. *Ann Intern Med* 1989;**111**:581–91.
18. Mielants H, Veys EM. The gut in the spondyloarthropathies. *J Rheumatol* 1990;**17**:7–10.
19. Khan MA, Khan MK. HLA B27 as an aid to diagnosis of ankylosing spondylitis. *Spine—State of the Art Reviews* 1990;**4**:617–25.
20. Bjorkman PJ, Saper MA, Samraoui B, *et al.* Structure of the human class 1 histocompatibility antigen, HLA-A2. *Nature* 1987;**329**:506–18.
21. Arnett FC. The seronegative spondyloarthropathies. *Curr Opin Rheum* 1989;**1:** 137–8.
22. Chamberlain MA. Socio-economic effects of ankylosing spondylitis. *Int Rehab Med* 1981;**3**:94–9.
23. Hordon LD, Bird HA. Joint laxity and ankylosing spondylitis. *Br J Rheumatol* 1988:**27**:241–2.

CAN EXERCISE PREVENT OSTEOPOROSIS?

PRIMARY osteoporosis is a bone disease of multifactorial pathogenesis. Well known causative determinants are nutrition, hormonal status and mechanical load. While nutritional or hormonal deficiencies can be compensated by supplements of calcium, vitamin D or oestrogens, the deficit of mechanical load in modern life theoretically could be compensated by physiotherapy or recreational exercises recommended for different reasons to those that prompt their use in conditions such as ankylosing spondylitis. Whether such exercises have a preventive or restorative power, has so far been studied in 11 prospective longitudinal controlled trials, mostly examining post- or perimenopausal women [1–12], but not addressing young women. And only one investigation included osteoporotics with some morphological changes in the lumbar vertebrae [12], while all the other studies followed healthy, elderly women.

Endpoint measures have been the bone mineral content (BMC) or bone mineral density (BMD) in all these studies on exercise and bone width in a few [4–6, 10]. Much more relevant endpoints, such as fracture rate, loss of function or quality of life, have not been addressed in any of these trials. They would need much larger sample sizes. The treatment modalities under examination varied from mild to vigorous.

Correspondence to Prof. N. J. Gerber.

Mild exercises for 30 min, three times a week [2], walking 2 miles or performing five aerobic dances four times a week [4], walking 7 miles a week [5, 6] or prone lying back extensions 10 times daily [9] seemed to have no [5,6, 9] or only minor positive effects on bone mineral content [2, 4].

Moderate forms of training, such as walking, running, lying exercises and ball games twice a week for 1 h [3] were followed by a significant increase of BMC in the lumbar spine (+3.5% versus −2.7% in controls) and a definite sparing of bone loss in the forearm (unchanged BMC in exercisers versus loss of 3.7% in controls) after 8 months of training [3]. Grip strength exercises, squeezing a tennis ball as hard as possible, six times daily [11] had a surprising radial bone gain (+3.4% BMC) parallel to an increase of grip strength after 6 weeks [11]. Six months after cessation, most of these gains were lost.

Not surprisingly, all trials evaluating the effects of vigorous exercises revealed unequivocal results: this was the case in Aloia's [1] pilot study on weekly three times 60 min conditioning training of increasing intensity, according to individual tolerance, leading after 1 year to a total bone mineral content increase of +2.6% in the exercise group, compared to a decrease of −2.4% in the controls. However, when normalized to total body potassium, i.e. lean body mass, there were

no significant changes in either group. A second trial on the effects of vigorous walking, jogging, rowing and stair climbing in 35 healthy postmenopausal women leading a sedentary life resulted after 9 months in a +5.2% increase of lumbar bone mineral content compared to a loss of −1.4% in the controls [7]. After 22 months the exercisers had a +6.1% increase versus a loss of −1.1% in the controls. Following cessation of the exercises, at the end of the detraining period, bone mass returned nearly to baseline levels. Whether such a time consuming and intensive programme could or would be adopted by many elderly women, is questionable. And whether walking and jogging alone would have had the same effect is also left open. A third extensive and impressive controlled longitudinal trial by Smith and co-workers [10] on vigorous aerobic dancing, walking and jogging, performed at an intensity of 70–85% of maximum heart rate, 45 min three times a week, revealed after 4 years that the control group BMC and BMC/bone width declined significantly in both arms, whereas the rate of decline in the exercise group was significantly less for 12 of the 18 bone variables. Lesser differences between groups were observed in the humerus. This study gives evidence that in middle-aged women vigorous exercises three times a week for 4 years may minimize or even reverse involutional bone loss of the upper extremities, regardless of the menopausal status. Again, whether such an intensive programme over such a long period is feasible for a majority of women must be questioned.

All the above mentioned trials investigated healthy women. Concerning their hormonal state, however, not all were comparable, as some were mixing pre- and postmenopausal women [3, 10], and several did not mention the number of years after menopause and some did allow taking of oestrogens as well [4].

In the only controlled longitudinal study investigating the effects of exercise in osteoporotic women [12], 14 exercisers, performing vigorous loading, pulling and twisting of the distal forearm for 15 min and whole body exercises for an additional 30–35 min three times a week for 5 months were compared with 26 comparable controls. The result was a decrease in bone mineral density by −1.9% in controls, whereas exercisers increased by +3.8%. BMC, however, did not change over the whole period, but low back pain complaints were reduced in number and severity in the exercise group. If this trial could be reproduced on a larger scale, this would prove a positive effect of loading exercises on trabecular bone in postmenopausal osteoporotics.

In conclusion, all four trials of vigorous exercise show a positive effect on bone mineral content [1, 7, 10, 12] and one of these trials even seems to reduce the incidence of low back pain. Exercises of moderate [3, 8, 11] or mild intensity [2, 4–6, 9] revealed less definite results. Further studies should try to avoid problems by observing some of the following crucial points: compliance, randomization procedure, sufficient sample size, ovarian or oestrogen status, muscle mass and strength. To start with *compliance*, this was recorded in a few trials only [3–6, 8]. A negative exercise result thus seems largely invalid when compliance has not been recorded. A proper pretrial *randomization procedure* for group allocation is the only means to avoid biases. Unfortunately, only three trials had used such a procedure [4–6, 9]. A sufficient *sample size* to overcome the relative imprecision of BMC measurements inherent in the method was given only in two of the 10 prevention trials [5, 6, 10]. *The ovarian or oestrogen status* should be recorded by indicating the exact number of years after menopause and exact dosage and duration of oral, transdermal or vaginal replacement, considering their major influence on bone quantity. *Muscle mass and strength,* probably the two most important prerequisites for the effect of physical exercise on bone mass, have so far only been recorded in few trials [5, 6, 11]. Other, and probably more relevant, endpoint parameters than BMC are needed in further exercise trials: quality of life, general fitness, well being, number of falls and fractures, bone architecture, etc. In the light of increasing costs of treating osteoporosis and of the cheapness and safety of exercises, such trials are needed.

N. J. Gerber, B. Rey
Department of Rheumatology, University Hospital, CH-3010, Berne, Switzerland

References

1. Aloia JF, Stanton HC, Ostuni JA, *et al.* Prevention of involutional bone loss by exercise. *Ann Intern Med* 1978;**89**:356–8.
2. Smith EL, Reddan W, Smith PE. Physical activity and calcium modalities for bone mineral increase in aged women. *Med + Sci Sport + Exercise* 1981;**13**:60–14.
3. Krolner B, Toft B, Nielsen SP, Tondevold E. Physical exercise as prophylaxis against involutional vertebral bone loss: a controlled study. *Clin Sci* 1983;**64**:541–6.
4. White MK, Martin RB, Yeater RA, Butcher RL, Radin EL. The effects of exercise on the bones of postmenopausal women. *Int Orthop (SICOT)* 1984;**7**:209–14.
5. Kriska AM, Bayles C, Cauley JA, LaPorte RE, Sandler RB. A randomized exercise trial in older women: increased activity over two years and the factors associated with compliance. *Med Sci Sports Exerc* 1986;**18**(5):557–62.
6. Sandler R, Cauley J, Hom D, *et al.* The effects of walking on the cross-sectional dimensions of the radius in postmenopausal women. *Calcif Tissue Int* 1987;**41**:65–9.
7. Dalsky GP, Stocke KS, Ehsani AA, *et al.* Weight-bearing exercise training and lumbar bone mineral content in postmenopausal women. *Ann Intern Med* 1988;**108**:824–8.
8. Cavanaugh DJ, Cann CE. Brisk walking does not stop bone loss in postmenopausal women. *Bone* 1989;**9**:210–14.
9. Sinaki M, Wahner HW, Offord KT, *et al.* Efficacy of non-loading exercises in prevention of vertebral bone loss in postmenopausal women: a controlled trial. *Mayo Clin Proc* 1989;**64**:762–9.
10. Smith EL, Gilligan C, McAdam M, Ensign CP, Smith PE. Deterring bone loss by exercise intervention in pre-menopausal and post-menopausal women. *Calcif Tissue Int* 1989;**44**:312–21.
11. Beverly MC, Rider TA, Evans MJ, Smith R. Local

bone mineral response to brief exercise that stresses the skeleton. *Br Med J* 1989;**299**:233–5.
12. Simkin A, Ayalon J, Leichter I. Increased trabecular bone density due to bone-loading exercises in postmenopausal osteoporotic women. *Calcif Tissue Int* 1987;**40**:59–63.

ANNOUNCEMENTS AND CALENDAR FOR 1991–92

1991		
April	12	BSR Spring Meeting and Heberden Round. OXFORD (Dr A. Mowat).
May	23–24	BSR Basic Rheumatology Course. BRISTOL (Dr J. Kirwan).
Sept	18–20	BSR Annual General Meeting. IMPERIAL COLLEGE, LONDON.
1992		
March	27	BSR Spring Meeting and Heberden Round. SOUTHAMPTON. (Dr M. Cawley).
July	22–25	7th EULAR Symposium and BSR Annual General Meeting. BARBICAN, LONDON.

EDUCATIONAL VISITS TO UK RHEUMATOLOGY CENTRES

As a trainee in Rheumatology (Registrar, Senior Registrar or equivalent), you may wish to broaden your experience by visiting other rheumatology units. Many centres around the UK are willing to host such visits. Details of these rheumatology units are available from the British Society for Rheumatology. The organization of the visit is then up to the visitor. Doctors from both the UK and overseas are welcome to contact the British Society for Rheumatology for further details:

Further information about these events from Ms L. Johnson, British Society for Rheumatology, 3 St Andrew's Place, Regent's Park, London NW1 4LE.