

Assessment of age-related bone loss in normal South African women by means of the Hologic QDR 1000 system

A. A. Kalla, A. B. Fataar, L. Bewerunge

The aim of this study was to evaluate age-related changes in cortical and trabecular bone mineral density (BMD) in South African subjects, and to develop a local reference database for dual energy X-ray absorptiometry with the QDR 1000 densitometer. A questionnaire was used to recruit volunteers. Age, years since menopause, use of medications and medical diseases were recorded. Men were excluded. Only whites and coloureds were studied. Three hundred and eleven women had single measurements over a 2-year period. Seven sets of subjects were defined according to age (18 - 44; 45 - 49; 50 - 54; 55 - 59; 60 - 64; 65 - 69; > 70 years) ($N = 163; 32; 35; 23; 25; 16; 17$ respectively). Height and weight did not change significantly with age. There were significant differences in lumbar and femoral BMD (Ward's) compared with those of American subjects in the different age categories. Both the lumbar and total femoral BMD began to fall significantly between the ages of 60 and 65 years ($P < 0,01$). Ward's triangle showed a significant fall in BMD between 45 and 50 years of age ($P < 0,01$). Ward's BMD predicted 36% of the variation in lumbar BMD at 45 years but 10% at 70 years. Ward's triangle is a useful predictor of femoral bone loss in later years. The fracture threshold at the lumbar vertebrae was $0,822 \text{ g/cm}^2$; at Ward's triangle it was $0,443 \text{ g/cm}^2$. This gave a 16% prevalence of osteopenia in the lumbar region and a prevalence of 24% at Ward's area.

S Afr Med J 1994; **84**: 398-404.

Osteoporosis is a major cause of morbidity and mortality among elderly women in the UK, USA and Europe.^{1,2} Numerous risk factors for osteoporotic fracture have been defined, but they have poor predictive value in assessing an individual's susceptibility to fracture.³ Bone mineral density (BMD) measurement is the only definitive way of predicting fracture risk.⁴ Studies in South Africa are lacking, but there is evidence that, among some groups of people, the

Rheumatic Diseases Unit, Department of Medicine and Department of Nuclear Medicine, Groote Schuur Hospital and University of Cape Town

A. A. Kalla, M.B. CH.B., F.C.P. (S.A.), M.D.

A. B. Fataar, M.B. CH.B., M.MED. (NUC. MED.)

L. Bewerunge, DIP. RAD.

prevalence rates are comparable to those of other countries.⁵⁻⁷

Dual-energy X-ray absorptiometry (DXA) is currently regarded as the most useful non-invasive method of measuring trabecular bone density.⁸⁻¹⁰ The equipment has only recently become available in South Africa. Different methods include the Hologic QDR 1000 system, the Lunar system and quantitative computed tomography (QCT). There are no BMD reference values for any of the machines in use that can define a group of South African individuals at risk. Current Hologic software is designed to evaluate subjects in respect of American normals. The software of the Hologic QDR 1000 densitometer uses American normative data based on healthy American women aged between 20 and 80 years. Those whose BMD is in the lowest quartile for young normal subjects are at greatest risk of fracture.^{11,12} It is generally recommended that appropriate reference values be established for geographically different populations.¹³ Patient differences may reflect a combination of genetic and environmental effects on BMD and strength.

This study was designed to ascertain differences from American data and to develop a range of values which could serve as reference data for normal South African women, in order that subjects at greater risk of fracture could be identified and studied prospectively for fracture risk and prevention. We also looked at relationships between cortical and trabecular bone within the femur.

Materials and methods

Female volunteers were recruited from the hospital staff as well as the general public of Cape Town. Members of the public were invited to participate through requests in local newspapers, as well as the provision of self-addressed postcards displayed at supermarkets and other strategic points. Subjects were screened by questionnaire for the presence of co-existent disease likely to interfere with BMD, such as early menopause, thyroid disease and severe arthritis. Current use of oestrogen preparations was recorded. Subjects taking any form of hormone replacement therapy (HRT) were excluded from the analysis; they were not asked about the previous use of HRT. Patients who had had a hysterectomy were excluded, even if the ovaries were purported to have been left *in situ*. Volunteers were also excluded if they were severely immobilised or using any medications likely to interfere with bone metabolism. There were 311 subjects suitable for study. Duration of menopause was not accurate and the subjects were stratified into groups as follows: 1 year (pre- and perimenopausal) = 1; 1 - 5 years = 2; 6 - 10 years = 3; and > 10 years = 4.

The number of black women who volunteered for this research was too small (4 subjects) to allow statistical comparison. This study was thus confined to whites and coloureds (as defined by the now abolished Population Registration Act). These 2 subgroups were compared in respect of all BMD measurements taken at the vertebrae and left hip. They were not age-matched, but the age range (18 - 59 years) was equalised for the 2 subgroups (see 'Statistical methods'). American data based on 650 normal

women in North America were obtained from the manufacturers' manual.

A single measurement was made with the Hologic QDR 1000 DXA absorptiometer, measuring several sites. BMD at the left hip and first 4 lumbar vertebrae (L1 - L4) was measured. At the hip, Ward's triangle, the neck, trochanter and inter-trochanter regions were measured. Total and mean values were also available for the hip and vertebrae. Height and weight were recorded at the same time. The lumbar BMD was expressed as a mean of the BMD of L1 - L4; collapsed vertebrae, if present, were excluded from the measurement.

The *in vitro* coefficient of variation of the technique at our institution was 0,5% in a phantom measured at regular intervals ($N = 577$) during the 2-year period (November 1989 - December 1991). *In vivo* precision is important in sequential studies of the same individuals over time, but is irrelevant in cross-sectional studies such as ours.

Statistical methods

Spearman correlation coefficients were used for the construction of a correlation matrix between different measurements and sites. A theoretical fracture threshold was determined based on the mean -2 SD of subjects aged 30 - 39 years, for lumbar and Ward's BMD. Step-wise multiple regression analysis was used to see if lumbar BMD could be predicted from femoral BMD. Ward's BMD was the dependent variable when lumbar BMD and BMD at other femoral regions were used in the prediction. Analysis of variance was used to determine the age at which changes in BMD became significant. The white women were significantly older than the coloured women, so the comparison excluded white subjects over 60 years of age to coincide with the age range of the coloured subjects. All statistical analyses were performed on the mainframe computer at the University of Cape Town with current versions of the BMDP and SAS statistical software packages.¹⁴⁻¹⁶ Statistical comparisons with American data were based on the mean and SD per decade, supplied by the manufacturers.

Results

Three hundred and eleven women were seen over the 2 years from November 1989 to December 1991. The mean age was 44 years (SD 15) with a range of 18 - 75 years. There were 163 under 45 years of age (young) and 148 over the age of 45 years (old). Backache was a symptom in 21% of the younger women and in 27% of the older group. All volunteers were independently mobile, and postmenopausal subjects were not receiving HRT at the time. We were not able to ascertain how many subjects had received HRT in the past. There were no significant differences between premenopausal females using contraceptives ($N = 48$) and those who did not ($N = 115$). Table I shows that there were no significant differences between whites and coloureds in respect of BMD, and all further studies were done on the group as a single unit.

A comparison with the American database showed that the lumbar BMD was significantly higher in our subjects, particularly in those aged over 45 years. Peak lumbar bone

density was, however, attained at the same age in South African and American subjects (Table II; Fig. 1). That of Ward's triangle, in contrast, was significantly lower in our cohort, especially in the elderly. Peak Ward's bone density was attained at a slightly older age than in American subjects (Table III; Fig. 2). We had too few subjects over 75 years of age to allow a meaningful comparison of that group.

Fig. 3 is a frequency histogram of the lumbar BMD stratified by 5-year intervals after the age of 45 years. The mean begins to fall around the age of 55 years.

Table I. Mean (\pm SD) of BMD at different sites for white and coloured subjects

Variable	White	Coloured	P-value
No.	184	69	
Age (yrs)	41 \pm 12	34 \pm 8	0,0001
Height (cm)	164 \pm 10	159 \pm 7	0,0004
Weight (kg)	61 \pm 12	61 \pm 12	0,84
Lumbar BMD (g/cm ²)	1,04 \pm 0,13	1,02 \pm 0,14	0,25
Ward's BMD (g/cm ²)	0,66 \pm 0,13	0,67 \pm 0,21	0,45
Total femoral BMD (g/cm ²)	1,09 \pm 0,13	1,1 \pm 0,16	0,52

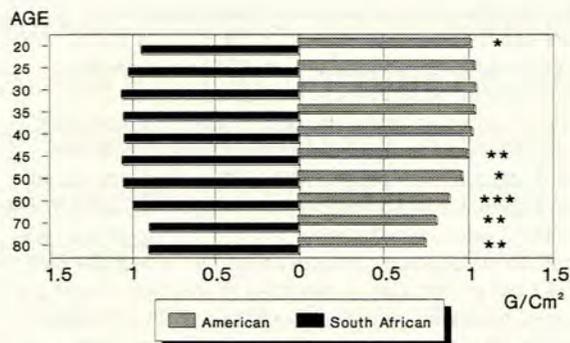
Whites over 60 years of age were excluded from this analysis.

Table II. Mean (\pm SD) of BMD (g/cm²) at the lumbar spine (L1 - L4); comparison of 650 American and 311 South African subjects aged 20 - 80 years, per half-decade

Cut-off age	American	South African	t-value	P-value
20	1,019 \pm 0,110	0,940 \pm 0,095	2,49	0,025
25	1,040 \pm 0,110	1,018 \pm 0,129	0,93	NS
30	1,047 \pm 0,110	1,062 \pm 0,109	0,79	NS
35	1,041 \pm 0,110	1,050 \pm 0,114	0,47	NS
40	1,024 \pm 0,110	1,045 \pm 0,134	0,80	NS
45	0,999 \pm 0,110	1,058 \pm 0,116	3,00	0,005
50	0,967 \pm 0,110	1,049 \pm 0,176	2,42	0,025
60	0,892 \pm 0,110	0,991 \pm 0,140	5,48	0,0001
70	0,815 \pm 0,110	0,891 \pm 0,132	3,55	0,005
80	0,752 \pm 0,110	0,895 \pm 0,213	2,66	0,01

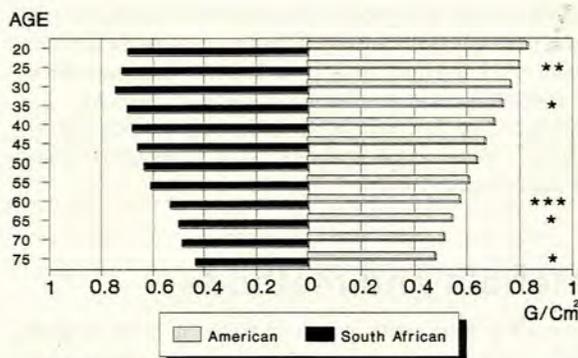
Table III. Mean (\pm SD) of BMD (g/cm²) at Ward's triangle of the left hip; comparison of 650 American and 311 South African subjects aged 20 - 80 years, per half-decade

Cut-off age	American	South African	t-value	P-value
20	0,832 \pm 0,120	0,696 \pm 0,141	0,35	NS
25	0,801 \pm 0,120	0,719 \pm 0,163	2,75	0,01
30	0,769 \pm 0,120	0,743 \pm 0,125	1,19	NS
35	0,737 \pm 0,120	0,666 \pm 0,250	1,81	0,05
40	0,706 \pm 0,120	0,678 \pm 0,118	1,21	NS
45	0,674 \pm 0,120	0,657 \pm 0,089	1,14	NS
50	0,642 \pm 0,120	0,632 \pm 0,117	0,44	NS
55	0,611 \pm 0,120	0,606 \pm 0,104	0,29	NS
60	0,579 \pm 0,120	0,528 \pm 0,081	3,08	0,005
65	0,547 \pm 0,120	0,495 \pm 0,103	2,47	0,025
70	0,516 \pm 0,120	0,481 \pm 0,112	1,17	NS
75	0,484 \pm 0,120	0,428 \pm 0,098	2,06	0,05
80	0,452 \pm 0,120	0,419 \pm 0,160	—	—



* p<0.05 ** p<0.01 *** p<0.0001

Fig. 1. Horizontal bar graph showing a comparison of American and South African lumbar (L1 - L4) BMD per half-decade.



* p<0.05 ** p<0.01 *** p<0.005

Fig. 2. Horizontal bar graph showing a comparison of American and South African Ward's BMD per half-decade.

When multiple range tests (Duncan) were done, the differences were statistically significant ($P < 0,01$) after the age of 60 years. The differences were statistically significant even after correction for multiple comparisons [overall $P < 0,0001$] (Bonferroni effect). The fracture threshold at the lumbar vertebra was 0,822 g/cm², giving a 16% prevalence of lumbar osteopenia in our cohort over 45 years of age. Of those older than 70 years, 29% had a BMD reading below 0,822 g/cm². The prevalence of lumbar osteopenia in the postmenopausal group was 9%. However, in the group who were more than 10 years postmenopausal, 30% of the subjects had a lumbar BMD below the fracture threshold.

The BMD of Ward's triangle is presented similarly in Fig. 4. It is evident that the mean BMD begins to fall at an earlier age than the mean lumbar BMD. Multiple range tests showed that this difference becomes statistically significant at age 50 years ($P < 0,05$). The fracture threshold of Ward's area was 0,443 g/cm², giving a 24% prevalence of femoral osteopenia in our cohort over 45 years of age. However, 71% of subjects older than 70 years had a BMD measurement below 0,443 g/cm². The prevalence of Ward's osteopenia in the menopause was 7%, but 37% of the group that was 10 years post-menopausal were at high risk of fracture at the femur.

LUMBAR BMD

Mispoints	< 45	46-50	51-55	56-60	61-65	66-70	> 70
1.520							
1.500							
1.450							
1.400							
1.350							
1.300							
1.250							
1.200							
1.150							
1.100							
1.050							
1.000							
0.950							
0.900							
0.850							
0.800							
0.750							
0.700							
0.650							
0.600							
0.550							
Mean	1.040	1.056	1.023	0.967	0.902	0.872	0.898
Std. Dev.	0.151	0.166	0.136	0.143	0.133	0.146	0.206
n	163	32	35	23	25	16	17

Group	Mean	n	<45	46-50	51-55	56-60	61-65	66-70	>70
<45	1.04	163							
46-50	1.056	32							
51-55	1.023	35							
56-60	0.967	23							
61-65	0.96	25							
66-70	0.87	16							
>70	0.90	17							

The M denotes that a value coincided with the mean. The numbers in column 1 indicate the total number of subjects with the corresponding BMD (g/cm²). The change was statistically significant ($P < 0.01$) after the age of 60 years. The accompanying table shows the probability that the differences were not due to chance.

Fig. 3. Frequency histogram of change in lumbar BMD with age.

WARD'S BMD

Mispoints	< 45	46-50	51-55	56-60	61-65	66-70	> 70
1.160							
1.120							
1.080							
1.040							
1.000							
0.960							
0.920							
0.880							
0.840							
0.800							
0.760							
0.720							
0.680							
0.640							
0.600							
0.560							
0.520							
0.480							
0.440							
0.400							
0.360							
0.320							
0.280							
0.240							
0.200							
0.160							
0.120							
0.080							
0.040							
0.000							
Mean	0.701	0.627	0.618	0.542	0.483	0.508	0.419
Std. Dev.	0.129	0.097	0.113	0.093	0.098	0.106	0.129
n	163	32	35	23	25	16	17

The M denotes that a value coincided with the mean. The numbers in column 1 indicate the total number of subjects with the corresponding BMD (g/cm²). The change was statistically significant ($P < 0.01$) after the age of 50 years. The accompanying table shows the probability that the differences were not due to chance.

Fig. 4. Frequency histogram of change in Ward's BMD with age.

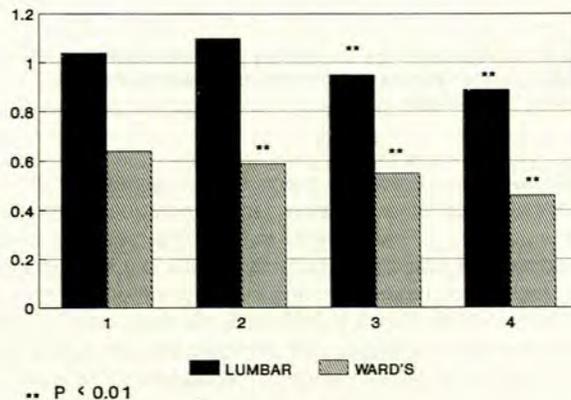
Table IV shows BMD stratified according to approximate time after menopause. In 6 subjects, duration of menopause was not recorded. There is a noticeable difference in the time that femoral bone loss occurs relative to loss at the lumbar vertebrae. These findings are similar to those described for the effects of age, and show that femoral loss precedes spinal loss in our cohort. Fig. 5 shows the mean spinal and femoral BMD according to menopausal stage. These findings do not show the expected marked losses early in the menopause followed by a period when bone loss tapers off. In fact, our cross-sectional data suggest that peak lumbar bone mass is attained early in menopause, with progressive loss after 6 - 10 years of oestrogen deficiency. Femoral bone loss, in contrast, is significant within the first 5 years of the menopause.

Table IV. BMD at different sites relative to stage of menopause

No.	Stage	Lumbar BMD	Femoral neck BMD	Total femoral BMD	Ward's
199	1	1,04	0,86	0,95	0,69
36	2	1,1	0,79*	0,92	0,59†
24	3	0,95†	0,75†	0,87*	0,55†
46	4	0,89†	0,67†	0,81†	0,46†

* $P < 0,05$.
† $P < 0,01$.

Stratification is based on the following scheme: 1 = pre- and peri-menopausal; 2 = 1 - 5 years; 3 = 6 - 10 years and 4 = > 10 years post-menopausal.



1 = pre- and peri-menopausal; 2 = 1 - 5; 3 = 6 - 10; 4 = > 10 years post-menopausal. Changes at the femur occur earlier than changes at the spine.

Fig. 5. Histogram of mean BMD at lumbar spine and Ward's area, stratified according to stage of menopause.

Fig. 6 is a correlation matrix between lumbar BMD and a series of different variables whose correlation coefficient is expressed as a histogram. These apply to 3 age groups of subjects. The negative effect of age over 50 years and the menopause is confirmed. It is evident that, while significant correlations are found, there are differences according to age. The prediction of lumbar BMD from femoral BMD is much more accurate in younger subjects than in their elderly counterparts. This may partly be explained by the inaccuracies of lumbar BMD due to aortic calcification and vertebral osteophytes in the elderly. Variation in lumbar BMD is significantly and consistently explained by variations in femoral BMD in subjects aged between 60 and 65 years. The results suggest that caution should be exercised in the making of inferences about one site from measurements at another site. No more than 64% (r^2) of the variation at the spine could be explained by the variation at the femur; this suggests that both sites need to be measured simultaneously in all subjects.

Multivariate regression analysis was used to test the relative effects of age and menopause at the relevant trabecular bone sites, viz. lumbar vertebrae and Ward's triangle. When lumbar BMD was the dependent variable in a step-wise multiple regression analysis which included age and menopause together with femoral measurements as the independent variables, menopause (2%) was a better predictor of lumbar BMD than age (0%). However, lumbar

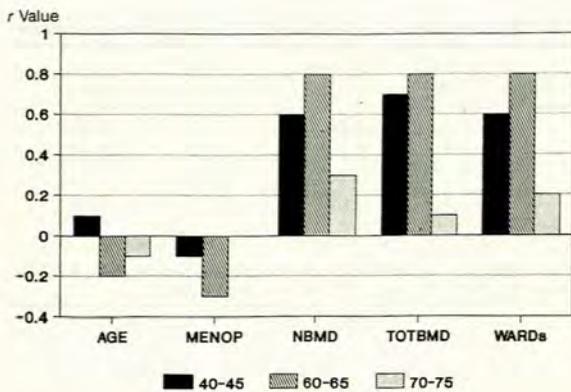
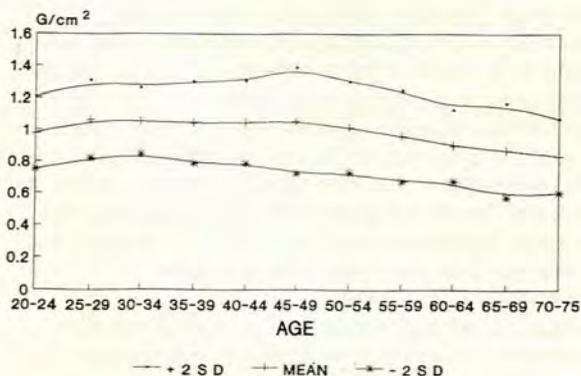


Fig. 6. Histogram of the correlation coefficient between lumbar BMD, age, menopause and femoral sites, according to 3 different age groups.

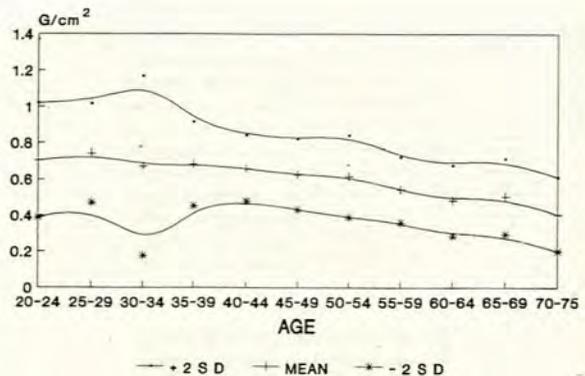
BMD (44%) was predicted mainly by trochanteric BMD, suggesting that other unknown factors were more important in the prediction. This confirms that femoral measurements could be useful predictors of lumbar BMD, but suggests that the trochanteric region contains the most trabecular bone at the femur. When Ward's triangle was the dependent variable in the regression analysis, age predicted the variation to a greater extent (3%) than stage of menopause (1%). Again, unknown factors seemed important, since neck BMD predicted 85% of the variation in Ward's BMD. Lumbar BMD did not feature as a significant predictor of Ward's BMD, irrespective of age or menopause. This renders questionable the wisdom of using Ward's BMD to predict the risk of femoral fracture.

Figs 7 and 8 represent the nomogram of lumbar and femoral trabecular BMD (Ward's area) respectively in South African subjects. The range encompasses the mean and 2 SD to either side of the mean per decade. Subjects are regarded as being at high risk for fracture if the BMD falls below 2 SD of the mean (or the lowest quartile) relative to young normals (aged 30 - 39 years) (*t*-score). Both sets of graphs confirm the relatively steep fall in BMD after



Values falling more than 2 SD below the mean for young subjects indicate risk of fracture (fracture threshold).

Fig. 7. Reference range of lumbar BMD encompassing the mean and 2 SD above and below, per 5-year groups after age 45 years.



Values falling more than 2 SD below the mean for young subjects represent risk of fracture (fracture threshold).

Fig. 8. Reference range of Ward's BMD encompassing the mean and 2 SD above and below, per 5-year groups after age 45 years.

menopause, which occurs at an average age of 52 years in Cape females. Tables II and III provide the mean and SD per half-decade groups by age at the 2 sites of interest. These values can be used in the Hologic software to generate a South African reference nomogram for interpretation of results in local subjects. The curves are smoother when results are grouped by decade.

Discussion

It is virtually impossible to eliminate bias entirely from a population study of this nature, particularly that associated with urbanisation, literacy, social stratum and race. Our finding of backache as a symptom in up to 20% of the subjects suggests that our sample was not biased in favour of those with skeletal abnormalities. This is borne out by the absence of a significant fall in height with age or menopause in our subjects.

Numerous reports have confirmed age-related bone loss and its acceleration in the menopausal female¹⁷⁻²⁰ and those with certain diseases.²¹ Solomon,⁵ using metacarpal indices, showed that South Africans follow the same trend of bone loss with age and menopause. Wagener and Hough⁶ found similar changes in their patients, but did not assess fracture prevalence. Our study confirms the tendency for trabecular bone density to fall with age. The mechanisms of age-related bone loss are poorly understood, but the pathogenesis is likely to be multifactorial. Physiological changes in the metabolism of parathormone may be important.²² In women, the most important effect is that related to oestrogen withdrawal which accompanies menopause. The high prevalence of osteopenia in our subjects over 70 years of age confirms their increased risk. The definition of the fracture threshold in this group is contentious.²³

Osteoporotic syndrome is characterised by structural bone failure, resulting in fractures at the wrist, hip and spine. While numerous clinical risk factors have been defined, none of these can adequately predict BMD or fracture.³ Measurement of BMD is the best predictor of future fracture risk,⁴ underlining the importance of accurate assessment of bone density. Non-invasive measurement of BMD has

evolved over the years. The relatively non-sensitive measures of cortical bone at the metacarpals (radiogrammetry)²⁴⁻²⁶ were superseded by single-photon absorptiometry at the wrist,²⁹⁻³² but a computer-assisted measure of metacarpal BMD using digitised radiogrammetry may be as good as single-photon absorptiometry.³³ However, dual-photon absorptiometry^{9,23,34} and, more recently, DXA are the most widely reported methods of BMD measurement, and the most sensitive and reproducible non-invasive techniques.¹⁰ Quantitative CT of the spine^{35,36} and broad-band ultrasound attenuation at the heel³⁷ are also available as useful measures of trabecular bone. These are widely used to screen for osteoporosis risk.

Several DXA machines (e.g. Hologic, Lunar, Norland) and quantitative CT scanners are currently in use at different centres around South Africa and all of these use American normative data provided by the suppliers. In order for these machines to be used effectively in defining local subjects at risk for fracture, reference data from a local cohort of healthy subjects need to be established. Such data would also be of value to researchers interested in studying pathological causes of bone loss which may compound the 'physiological' effects of age and the menopause. Our results confirm that American data are inappropriate for the screening of South African subjects.

Our results confirm that backache is a not infrequent symptom in women of all ages. It was generally a poor predictor of low BMD (osteopenia). The fall in femoral BMD started relatively early in the menopause (age 50 - 60 years). Riggs and Melton³⁸ have categorised 2 types of osteoporosis based on clinical observations. Our cross-sectional analysis suggests that femoral bone loss was a feature of oestrogen withdrawal, while lumbar bone loss was a feature of ageing. This apparent difference from the reported literature^{4,12,38} may be the result of a number of factors, including the cross-sectional nature of our study and other unknown (climatic/ dietary/genetic) factors. It is possible that longitudinal studies in the same patients over time may contradict our initial impression. The step-wise multiple regression analysis agreed with established concepts in that menopause was a predictor of lumbar BMD while age was a better predictor of Ward's BMD.

The use of BMD measurement at one site to predict fracture at another site is controversial. Fracture data suggest that the best predictor of fracture is BMD measurement at the site of interest.³⁹ While trochanteric BMD was a significant predictor of lumbar BMD in this model ($P < 0,01$), much of the variation was unexplained (54%). Lumbar BMD could not predict any of the variation in the BMD of Ward's triangle. The regression analysis suggests that trochanteric BMD is the best measure of trabecular bone in the femur. Yet, most studies regard Ward's area as the best predictor of femoral neck fracture.³³ On the other hand, the fact that neck BMD is the best predictor of Ward's BMD may imply that cortical bone predominates at the latter site. There seems little doubt that both cortical and trabecular bone loss may be important in the genesis of fracture at the different sites.⁴⁰ Prevalence rates of osteopenia at the 2 sites confirm differential rates of loss. The significantly high prevalence of Ward's osteopenia in subjects over 70 years of age may explain the increased incidence of femoral neck fractures in this group.

A number of studies^{4,12} have shown that fracture risk is highest among subjects whose BMD is in the lowest quartile of young normal subjects aged 30 - 39 years (*t-score*). Results from different geographical regions have shown the need to develop reference values for each region. While we did not study the relationship between BMD and fracture, our subjects showed changes in the femur at an earlier age (chronological and menopausal) than at the vertebrae. This is contrary to reports of fracture prevalence at these sites.³⁸ Our subjects show a similar tendency to lose BMD with age and menopause as those of other populations. Solomon,⁶ and others,^{41,42} have shown that fracture prevalence among South African women is also comparable to that of other populations. Age-related prevalence of fractures at different sites would help to place our findings in perspective.

Significant differences in cortical BMD at the metacarpals have been shown between different groups of people,⁷ but these bear no relationship to fracture prevalence/incidence. In the study by Solomon,⁶ some groups of people had a lower fracture prevalence in spite of their lower metacarpal BMD. Clearly, quality of bone is likely to be as important as quantity of bone in the pathogenesis of fracture. Unfortunately, non-invasive techniques for measurement of bone strength are not currently available. Other factors such as neurovascular instability and type of trauma may also be important.¹¹

Screening for osteopenia in the general population remains controversial.^{43,44} Cost and availability of DXA for routine studies should be compared with the cost of femoral neck fractures to the health system and the economy as a whole. These figures are not available in South Africa. Many patients with fractures have normal BMD values, which may be due to aortic calcification and/or osteophytes. Lateral spine DXA scans have been developed in an attempt to improve accuracy of the measurement,⁴⁵ but prospective studies showing a major advantage with the modified technique are awaited. Our subjects aged over 45 years showed a prevalence of lumbar and femoral osteopenia similar to that reported from overseas centres. Prospective studies are needed to address the socio-economic implications of screening South African subjects for osteopenia.

We hope that the nomograms provided herein may serve as reference ranges for South African subjects in whom measurements are made with the Hologic QDR 1000 system. However, comparisons with other centres within South Africa are required to confirm our results. The use of similar machines, standard phantoms and quality control are essential in the development of reference ranges and the calculation of fracture thresholds of local patients.

Conclusions

South African women show the same tendency to bone loss with age and menopause as their counterparts elsewhere in the world. However, absolute BMD values for age at the 2 sites differ from those of American subjects. Femoral bone loss occurs earlier than lumbar bone loss in South African subjects. Prospective, longitudinal studies need to be planned in order to assess the extent to which prophylactic measures are needed to avoid long-term complications of ongoing bone loss. Local reference BMD values for fracture risk assessment are now available.

We sincerely thank the radiographers of the Department of Nuclear Medicine who helped with the scanning of subjects. We are also deeply indebted to Smith-Kline Beecham Pharmaceuticals for allowing us to use the DXA machine for this project. In addition, our thanks go to all those who volunteered for measurements of BMD for this research, to Dr B. K. Adams for reading the manuscript and Dr S. Isaacs for his help with some of the statistical analysis.

REFERENCES

- Browner WS, Seeley DG, Vogt TM, Cummings SR. Non-trauma mortality in elderly women with low bone mineral density. *Lancet* 1991; **338**: 355-358.
- Heyse SP, Sartori L, Crepaldi G. Epidemiology of osteoporosis: a study of fracture mortality in Italy. *Calcif Tissue Int* 1990; **46**: 289-293.
- Cooper C, Shah S, Hand DJ, et al. The multicentre vertebral fracture study group. Screening for vertebral osteoporosis using individual risk factors. *Osteoporosis Int* 1991; **2**: 48-53.
- Cummings SR, Kelsey JL, Nevitt C, O'Dowd KJ. Epidemiology of osteoporosis and osteoporotic fractures. *Epidemiol Rev* 1985; **7**: 178-208.
- Solomon L. Bone density in aging Caucasian and African populations. *Lancet* 1979; **2**: 1326-1330.
- Wagener GW, Hough FS. Metacarpal bone mass in the white and coloured populations of the Cape. *S Afr Med J* 1987; **72**: 205-208.
- DeSimone DP, Stevens J, Edwards J, Shary J, Gordon L, Bell NH. Influence of body habitus and race on bone mineral density of the midradius, hip and spine in aging women. *J Bone Miner Res* 1989; **4**: 827-830.
- Lai KC, Goodstitt MM, Murano R, Chestnut CH III. A comparison of two dual energy x-ray absorptiometry systems for spinal bone mineral measurement. *Calcif Tissue Int* 1992; **50**: 203-208.
- Schaadt O, Bohr H. Bone mineral by dual photon absorptiometry. Accuracy-precision-sites of measurement. In: Dequeker J, Johnston CC jun, eds. *Non-Invasive Bone Measurements; Methodological Problems*. Oxford: IRL Press, 1982: 59-72.
- Sievanen H, Oja P, Vuori I. Precision of dual energy x-ray absorptiometry in determining bone mineral density and content of various skeletal sites. *J Nucl Med* 1992; **36**: 1137-1142.
- Cummings SR. Are patients with hip fractures more osteoporotic? Review of the evidence. *Am J Med* 1985; **78**: 487-494.
- Melton LJ III, Riggs LB. Epidemiology of age-related fractures. In: Avioli LV, ed. *The Osteoporotic Syndrome*. New York: Grune & Stratton, 1982: 45-70.
- Ross PD, Norimatsu H, Davis JW, et al. A comparison of hip fracture incidence among native Japanese, Japanese Americans, and American Caucasians. *Am J Epidemiol* 1991; **133**: 801-809.
- SAS Institute. *SAS User's Guide: Statistics, Version 5* Ed. Cary, NC: SAS Institute, 1985.
- SAS Institute. *SAS User's Guide: Basics, Version 5* Ed. Cary, NC: SAS Institute, 1985.
- Dixon WJ (chief ed.), Brown MB, Engelman J, et al. *BMDP Statistics Software*. Berkeley, Calif.: UCLA Press, 1985.
- Cohn SH, Aloia JF, Vaswani AN, Yuan K. Women at risk for developing osteoporosis: determination by total body neutron activation analysis and photon absorptiometry. *Calcif Tissue Int* 1986; **38**: 9-15.
- Exton-Smith AN, Millard PH, Payne PR, Wheeler EF. Pattern of development and loss of bone with age. *Lancet* 1969; **2**: 1155-1157.
- Horsman A, Simpson M, Kirby PA, Nordin BEC. Non-linear bone loss in oophorectomised women. *Br J Radiol* 1977; **50**: 504-507.
- Riggs BL, Wahner HW, Dunn WL, Mazess RB. Differential changes in bone mineral density of the appendicular and axial skeleton with aging. *J Clin Invest* 1981; **67**: 328-335.
- Saville PD, Kharmosh O. Osteoporosis of rheumatoid arthritis: influence of age, sex and corticosteroids. *Arthritis Rheum* 1967; **10**: 423-430.
- Parsons JA. Physiology of parathyroid hormone. In: De Groot LJ, Cahill GF, Odell WD, et al., eds. *Endocrinology*. Vol. 2. New York: Grune & Stratton, 1979: 621-629.
- Mazess RB. Bone density in diagnosis of osteoporosis: thresholds and break-points. *Calcif Tissue Int* 1987; **41**: 117-118.
- Dequeker J. Precision of the radiogrammetric evaluation of bone mass at the metacarpal bones. In: Dequeker J, Johnston CC jun, eds. *Non-Invasive Bone Measurements; Methodological Problems*. Oxford: IRL Press, 1982: 27-32.
- Exton-Smith AN, Millard PH, Payne PR, Wheeler EF. Method for measuring quantity of bone. *Lancet* 1969; **2**: 1153-1154.
- Barnett E, Nordin BEC. The radiological diagnosis of osteoporosis: a new approach. *Clin Radiol* 1960; **11**: 166-174.
- Dequeker J. Quantitative radiology: radiogrammetry of cortical bone. *Br J Radiol* 1976; **49**: 912-920.
- Dequeker J, Geusens P, Verstraeten A. Clinical relevance of the radiogrammetric measurement of peripheral skeletal mass. In: Dequeker J, Johnstone CC, jun, eds. *Non-Invasive Bone Measurements; Methodological Problems*. IRL Press, 1982: 155-168.
- Cameron JR, Mazess RB, Sorenson JA. Precision and accuracy of bone mineral determination by direct photon absorptiometry. *Inv Radiol* 1968; **3**: 141-150.
- Nicoll JJ, Smith MA, Reid D, Law E. Measurement of hand bone mineral content using single-photon absorptiometry. *Phys Med Biol* 1987; **32**: 697-706.
- Reid DM, Nicoll JJ, Brown N, Tohill P. Measurement of hand bone mass by single photon absorptiometry in rheumatoid arthritis and asthma: comparison with metacarpal indices (Abstract). *Clin Phys Physiol Meas* 1986; **2**: 184.
- Wahner HW, Riggs BL, Beabout JW. Diagnosis of osteoporosis: usefulness of photon absorptiometry at the radius. *J Nucl Med* 1977; **18**: 432-437.
- Kalla AA, Meyers OL, Parkyn ND, Kotze TjvW. Osteoporosis screening — radiogrammetry revisited. *Br J Rheumatol* 1989; **28**: 511-517.
- Mazess RB. Noninvasive methods for quantitating trabecular bone. In: Avioli LV, ed. *The Osteoporotic Syndrome*. New York: Grune & Stratton, 1982: 85-114.
- Genant HK, Cann CE, Faul DD. Quantitative computed tomography for assessing vertebral bone mineral. In: Dequeker J, Johnston CC jun, eds. *Non-Invasive Bone Measurements; Methodological Problems*. Oxford: IRL Press, 1982: 215-249.
- Genant HK. Quantitative CT in osteoporosis assessment. In: Riggs BL, Melton LJ III, eds. *Osteoporosis: Etiology, Diagnosis and Management*. New York: Raven Press, 1988: 221-250.
- Truscott JG, Simpson M, Stewart SP, et al. Bone ultrasonic attenuation in women: reproducibility, normal variation and comparison with photon absorptiometry. *Clin Phys Physiol Meas* 1992; **13**: 29-36.
- Riggs BL, Melton LJ. Evidence for 2 distinct syndromes of involutional osteoporosis. *Am J Med* 1983; **75**: 899-901.
- Need AG, Nordin BE. Which bone to measure? *Osteoporosis Int* 1990; **1**: 3-6.
- Mazess RB. Fracture risk: a role for compact bone. *Calcif Tissue Int* 1990; **47**: 191-193.
- Schnitzler CM, Solomon L. Osteomalacia in elderly white South African women with fractures of the femoral neck. *S Afr Med J* 1983; **64**: 527-530.
- Dent CE, Engelbrecht HE, Godfrey RCC. Osteoporosis in lumbar vertebrae and calcification of the abdominal aorta in women living in Durban. *BMJ* 1968; **4**: 76-79.
- Purdie DW. Screening for osteoporosis. *Br J Hosp Med* 1992; **47**: 605-608.
- Hall FM, Davis MA, Baran DT. Bone mineral screening for osteoporosis (Sounding Board). *N Engl J Med* 1987; **316**: 212-214.
- Reid IR, Evans MC, Ames R, Wattie DJ. The influence of osteophytes and aortic calcification on spinal mineral density in postmenopausal women. *J Clin Endocrinol Metab* 1991; **72**: 1372-1374.

Accepted 5 Nov 1993.

Corticosteroid therapy and bone mass — comparison of rheumatoid arthritis and systemic lupus erythematosus

A. A. Kalla, O. L. Meyers, T. J. v. W. Kotze, R. Laubscher

This study was designed to evaluate the effects of low-dose corticosteroid (CS) therapy for rheumatoid arthritis (RA) and of high-dose CS therapy for systemic lupus erythematosus (SLE) on metacarpal bone mass in young (premenopausal) subjects. Ninety-eight patients with RA, 63 patients with SLE and 85 healthy controls of comparable age, race, sex and nutritional status were studied. Metacarpal bone mass was measured by radiogrammetry using a digitiser. In the RA patients, mean bone mass of CS-treated subjects (27%) was 52,31 g/cm², while that of untreated subjects was 56,69 g/cm² ($P < 0,02$). In the SLE group, mean bone mass of CS-treated subjects (76%) was 61,47 g/cm² and that of untreated subjects 62,36 g/cm² ($P > 0,1$). Although patients with SLE required larger cumulative doses of CS for longer periods, their bone mass was higher than that of the RA subjects ($P < 0,01$). None of the patients had femoral neck or vertebral crush fractures. In RA, bone loss was probably a feature of severe disease rather than of CS therapy.

S Afr Med J 1994; **84**: 404-409.

Rheumatic Diseases Unit, Department of Medicine, University of Cape Town

A. A. Kalla, M.B. Ch.B., F.C.P. (S.A.), M.D.

O. L. Meyers, M.B. Ch.B., F.C.P. (S.A.), M.D.

Institute of Biostatistics of the South African Medical Research Council, Parowvallei, CP

T. J. v. W. Kotze, D.Sc.

O. L. Meyers, M.B. Ch.B., F.C.P. (S.A.), M.D.