BONE LOSS AFTER TIBIAL FRACTURE

EVALUATED BY DUAL-ENERGY X-RAY ABSORPTIOMETRY

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We evaluated changes in bone mineral density (BMD) after tibial fractures, both at the site of fracture and at adjacent sites, using dual-energy X-ray absorptiometry. Five patients were studied prospectively for six months after fractures of the midshaft treated by intramedullary nailing or the application of an external fixator. We also reviewed 21 adults and 10 children who had sustained fractures of the tibia at least five years previously, comparing BMD in the injured limb with that in the non-injured limb and in a control group of 10 normal subjects.

We found a significant fall in BMD distal to a tibial fracture; this was evident at one month, fell to approximately 50% of normal at three months and persisted at six months. We found no significant improvement with weight-bearing.

Review at 5 to 11 years after adult midshaft fractures showed persistent bone loss in the distal tibia ($46.5 \pm 9.8\%$ of control values), but persisting sclerosis at old fracture sites ($172 \pm 38\%$ of control values). In contrast, we found no significant differences in BMD between the injured and control limb after fractures sustained in childhood either at the fracture site or in the distal segment.

We conclude that, in adults, tibial fractures are associated with definite and persistent post-traumatic loss of distal BMD.

J Bone Joint Surg [Br] 1995;77-B:473-8.

Received 8 March 1994; Accepted after revision 5 August 1994

One of the early responses of bone to trauma is an increase in the rate of mineral turnover (Bauer 1954) and many investigators have reported a significant reduction in bone mass after fracture of long bones, not only at the fracture but also at adjacent sites both proximal (Andersson and Nilsson 1979) and distal to it (Ulivieri et al 1990). This persists after union, but there is a wide range in both the amount of loss and the extent of recovery. These differences may be due, in part, to the variable accuracy of the techniques used for the measurement of bone mass.

We have reported the marked and sustained loss of bone in both the tibia and femur after osteotomy for limb lengthening (Eyres, Bell and Kanis 1993a,b), amounting to approximately 50% when measured by dual-energy X-ray absorptiometry (DEXA) of the segments distal to the distraction site. This effect was maximal during the phase of distraction, but the reduction persisted for at least two years after fixator removal. Since this degree of bone loss may increase the risk of fracture, we aimed to discover if there were comparable losses in the tibia after traumatic fracture.

PATIENTS AND METHODS

We made a prospective study of five patients with acute fractures of the tibia (Table I). Four were adults of mean age 38 years (26 to 55) and one was a child aged 12 years. All fractures were closed unilateral injuries of the midshaft which had been managed surgically by external fixation ($n = 3$) or by intramedullary nailing ($n = 2$). Measurements of bone mineral content (BMC) and bone mineral density (BMD) were made on both the injured and non-injured tibia by DEXA within one week of fracture, and repeated monthly for six consecutive months. Healing of the fracture was determined on plain radiographs by the presence of trabeculae crossing the site.

We also reviewed 31 adults who had sustained fractures of the tibial shaft at least five years previously. Of these, 11 men and 10 women were adult at the time of fracture and 10 were children (6 boys and 4 girls). They were identified from the operating records of the Royal Hallamshire and Sheffield Children's Hospitals and contacted by letter. Those who replied (31 of 46; 67%) attended for the measurement of BMD of both the injured and non-injured limb.

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0301-620X/95/3948 \$2.00

Table I. Details of the five patients studied prospectively after midshaft tibial fractures

Case	Age (yr)	Sex	Side	Treatment
1	12	M	L	External fixator
2	35	M	L	Intramedullary nail
3	36	M	R	External fixator
4	26	M	R	External fixator
5	55	F	L	Intramedullary nail

Most of the original clinical records and radiographs had been destroyed. The site and configuration of the fracture were therefore deduced from the DEXA image and the cause of injury was provided by the patient. Six of the women were postmenopausal, but none was taking oestrogens. None of the patients took medication or suffered from conditions likely to affect bone metabolism.

The causes given for the fractures were sport ($n = 5$), road-traffic accidents ($n = 14$) and falls ($n = 12$). Treatment had included manipulation and immobilisation in plaster ($n = 25$), manipulation and the incorporation of pins in plaster ($n = 3$), external fixation ($n = 2$) and internal fixation using a dynamic compression plate ($n = 1$). A concomitant fracture of the fibula was identified in 15 patients. All the fractures had united and there was no evidence or history of infection. The single plate had been removed at 18 months after surgery. All fractures were closed; 13 were left-sided and 18 right-sided. Most were in the midshaft, but four were in the distal tibia.

To assess any effect of lateral dominance on bone density of the leg, we performed DEXA studies on both the left and right tibia in a control group of five men and five women of mean age 41 and 47 years, respectively. Of these, 60% were right-footed as judged by the preferred side to kick a ball, 20% were left-footed and 20% gave no preference.

We measured the BMC and apparent BMD by DEXA (Hologic QDR 1000; Hologic Inc, Waltham, Massachusetts) as previously described (Eyres et al 1993a). The BMD of the distal tibia was measured using constant regions of interest of $4\text{ mm} \times 4\text{ mm}$ at the medial and lateral cortical regions, and in the medullary region at 40 mm proximal to the tibial plafond. We also recorded the total BMD across the width of the distal tibia at this level, using a $22\text{ mm} \times 7\text{ mm}$ region of interest (Fig. 1). We expressed the difference between the two sides as the percentage ratio between injured and non-injured sides.

To assess the reproducibility of this technique, two consecutive scans were performed in five patients after repositioning. The coefficient of variation (CV) depended on the site and size of the area examined. For $4\text{ mm} \times 4\text{ mm}$ regions of interest, the coefficients of variation were 1.2% and 4.4% for BMD measurements greater and lower than 0.2 g/cm^2 respectively. For the $22\text{ mm} \times 7\text{ mm}$ region of

Fig. 1

BMD measurements were made from $4\text{ mm} \times 4\text{ mm}$ regions of interest placed in the medial and lateral cortical and central medullary regions at 40 mm above the tibial plafond (left). The total BMD across the tibia at this level was measured from a $22\text{ mm} \times 7\text{ mm}$ region of interest to include all regions and to avoid the fibula (right).

interest the CV was 1% and 2.2%, respectively for the same range of density measurements.

Statistical analysis was by ANOVA, Student's *t*-test, and the Mann-Whitney U test as appropriate.

RESULTS

Control side and non-injured limbs. As expected, BMD values were consistently lower at the medullary zones than at the cortical zones (Table II). There were no significant differences in mean BMD values between children and adults, or between men and women.

In normal subjects there was no difference in BMD measurements between the left and right leg, or differences according to dominance. As expected, there was a close correlation between BMD values measured on the right and left sides for all the sites measured ($r = 0.97$ for total BMD; $r = 0.85$ for medial cortical sites; $r = 0.92$ for lateral cortical sites; $r = 0.86$ for medullary sites). There was also no significant difference between the mean BMD of the non-injured limb of the adult fracture group and either limb of the control group (Table II).

Sequential changes in BMD. In all five patients studied prospectively there was a significant decrease in BMD of the distal tibia within four weeks of fracture (Table III). After this there was a further reduction in BMD to a mean value of 57% of the non-injured limb at three months, and 47% at four months from the time of fracture. We found no further significant change in BMD after this time. These reductions appeared to be less marked in the two patients treated by intramedullary nails than in the three by external fixation.

Bone loss appeared to be unaffected by the start of weight-bearing at two to three weeks after intramedullary

Table II. Bone mineral density (BMD in g/cm^2 ; mean $\pm$ SEM) in the distal tibiae of non-injured limbs and control subjects

Group	Number	Mean age (yr)	BMD			
			Medial cortex	Medullary zone	Lateral cortex	Total
Children, non-injured limb						
All	10	9	0.93 ± 0.04	0.72 ± 0.04	0.99 ± 0.07	0.80 ± 0.03
Male	6	11	0.93 ± 0.04	0.73 ± 0.05	1.09 ± 0.08	0.85 ± 0.02
Female	4	9	0.93 ± 0.06	0.69 ± 0.05	0.83 ± 0.04	0.74 ± 0.04
Adult control group						
All	10	45	0.99 ± 0.07	0.57 ± 0.03*	0.83 ± 0.04	0.75 ± 0.03
Male	5	38	0.96 ± 0.08	0.57 ± 0.04	0.86 ± 0.06	0.78 ± 0.04
Female	5	42	0.94 ± 0.09	0.58 ± 0.05	0.79 ± 0.05	0.71 ± 0.04
Adults, non-injured limb						
All	21	43	1.04 ± 0.1	0.60 ± 0.03*	0.86 ± 0.05	0.77 ± 0.04
Male	11	40	1.03 ± 0.1	0.59 ± 0.04	0.89 ± 0.07	0.80 ± 0.05
Female	10	47	1.01 ± 0.1	0.57 ± 0.05	0.81 ± 0.05	0.74 ± 0.04

* significant difference ($p < 0.001$) between medullary and cortical BMD

nailing, or two to six weeks after the application of an external fixator. The time to union for the two methods of treatment was similar at 2.5 to 4.3 months for external fixation and two to four months for intramedullary nailing. There was significantly greater loss of bone from the medial than from the lateral cortical region of the distal tibia (Table III).

Long-term review. None of the patients had complaints or abnormalities on physical examination at 5 to 11 years after their tibial fracture. The BMD findings differed between patients who had sustained fractures as children and those injured as adults.

Adult injury. The mean ages at the time of assessment were 40 years for men and 47 years for women, not a statistically significant difference. There was also no significant differ-

ence in the mean BMD in the distal tibia of the injured limb between men and women ($0.80 \pm 0.02 \text{ g}/\text{cm}^2$ and $0.73 \pm 0.02 \text{ g}/\text{cm}^2$ respectively; $p = 0.32$).

At a mean time interval between fracture and assessment of 8 years (6 to 11), there was a significant difference in the BMD between the fractured and control limbs. For mid-shaft and distal fractures considered together, the mean BMD across the width of the distal tibia was $71 \pm 13\%$ of that of the control limb ($p < 0.001$). The magnitude of this difference, however, depended on the site of fracture.

For fractures of the distal tibia the scans traversed the old fracture site and the scan profile showed a consistently higher BMD than at the same site over the non-fractured leg (Fig. 2). In the four patients with distal fractures the local BMD was increased to $172 \pm 38\%$ of the control limb,

BMD measurements across the width of the tibia at the site of an old midshaft fracture and in the distal tibia in an adult and a child (case 4; Table I). In the adult (top left) there was sclerosis at the old fracture site with an increase in the transverse diameter in comparison with the non-injured limb. In the child, greater remodelling had left minimal sclerosis and similar dimensions (top right). In the distal tibia the adult showed persisting bone loss (bottom left), but in the child there was no significant difference between the injured and non-injured limb (bottom right).

Fig. 2

Table III. BMD in the distal tibia after fracture, expressed as a percentage of the measurement in the non-injured limb. The three right-hand columns show the distribution of bone loss at six months at each of three sites (see text)

Case	Time from fracture (mth)							Percentage loss at 6 months		
	0	1	2	3	4	5	6	Medial cortex	Medullary zone	Lateral cortex
1	100	78	56	30	35	40	42	20	60	67
2	100	85	80	75	68	60	65	55	55	70
3	100	74	60	55	41	48	44	40	42	58
4	100	87	70	65	60	50	47	41	38	55
5	100	80	70	62	60	64	69	58	48	67
Mean	100	81	67	57	53*	52*	53*	43†	49	63
SEM		2	4	7	6	4	5	4	4	3

* significantly different ($p < 0.001$) from values at time 0

† significantly different ($p < 0.001$) from lateral cortical values

with increases in both cortical and medullary regions (Table IV). After midshaft fractures of the tibia, the density profiles across the old fracture sites also showed increased BMD, but, in contrast, the BMD of the distal tibia was significantly reduced to 46.5% of control values. This effect was seen in both men and women (Table IV).

The lack of medical records made it impossible to assess the method or duration of treatment and its influence on bone mass, but we found no difference in the amount of bone loss in cases in which there was synostosis between the tibia and fibula.

Childhood injury. In ten patients with midtibial fractures in childhood the BMD was assessed at 6 to 9 years after the fracture. Scans of the distal tibiae showed no significant difference in BMD between the fractured and control limbs (Table IV; Fig. 3). At the sites of the old fracture, the BMD was higher than on the control side, by up to 130%, but even this difference was smaller than those after fractures in adult life (Fig. 2).

DISCUSSION

Our sequential study showed loss of bone to about 50% of the level in the control leg at four months. In adults, our retrospective study showed that this bone loss persisted for at least 11 years. Since we found no difference in BMD of the distal tibia between the non-injured limb and the tibiae in our control group, the finding supports the view that loss of bone density after trauma affects only the injured side (Nilsson 1966).

The persistence of bone loss after fracture or other trauma has been shown in many previous studies, but the reported degree of loss has varied. This probably results from the use of different techniques and the varied skeletal sites measured as well as their relationship to the fracture site (Table V). Reported long-term deficits in BMD range from 7% to 50%, but DEXA reveals much more marked losses (Ulivieri et al 1990; Eyres et al 1993a,b; Sarangi et al 1993). Single-energy techniques of measurement do not account for the uneven distribution of soft tissue and the errors in accuracy are likely to be high.

Fig. 3
BMD at the distal tibiae after unilateral fractures sustained during childhood. There was no significant difference between the fractured and the control limb. The diagonal line is that of identical results.

The loss of bone at the tibia does not appear to be related to the severity of trauma, the method of fixation or the duration of immobilisation (Ulivieri et al 1990). Our retrospectively studied patients had been recovered fully for many years, but bone loss was not related to the alteration in weight-bearing which could have been caused by a tibiofibular synostosis. Our few prospective cases showed a smaller bone loss after treatment by intramedullary nails. Bone loss is known to be greater in patients with algodystrophy (Bickerstaff, O'Doherty and Kanis 1991; Bickerstaff, Charlesworth and Kanis 1993; Sarangi et al 1993), but none of our patients gave a history of this disorder.

Persistent regional osteoporosis may increase the risk of further fractures. Finsen and Benum (1987) recorded 2744 earlier fractures in a series of 1659 acute fractures and found that femoral, patellar and tibial fractures were seen more often on the same side as previous fractures of the lower extremity. Their results show a fourfold increase (95% CI: 1.5 to 13.9) in the risk of refracture of the ipsilateral limb compared with the contralateral limb. Stress fractures of the distal tibia after the healing of an ipsilateral,

Table IV. BMD expressed as percentages of control values in the distal tibia ($\pm$ SEM) 5 to 11 years after fractures in adulthood and childhood

Group	Number	BMD			Total
		Medial cortex	Medullary zone	Lateral cortex	
Adults					
All	21	73.8 $\pm$ 8.2	70.1 $\pm$ 9.8	77.8 $\pm$ 10.2	71.4 $\pm$ 13.5
Midshaft	17	65.1 $\pm$ 5.5	50.8 $\pm$ 5.2*	60.3 $\pm$ 4.2	46.5 $\pm$ 9.8
Distal	4	137 $\pm$ 21.1	152 $\pm$ 6.2	156 $\pm$ 2.6	172 $\pm$ 38
Men					
All	11	68.4 $\pm$ 9.2	68.2 $\pm$ 14	66.2 $\pm$ 10.5	55.8 $\pm$ 14.6
Midshaft	9	57.4 $\pm$ 6.9	51.8 $\pm$ 8.1	53.1 $\pm$ 4.6	43.4 $\pm$ 4.4
Distal	2	110 $\pm$ 3.3	145 $\pm$ 13	134 $\pm$ 11*	124 $\pm$ 17
Women					
All	10	92.2 $\pm$ 15.2	71.9 $\pm$ 13.2	90.6 $\pm$ 17.6	85.9 $\pm$ 25.5
Midshaft	8	63.4 $\pm$ 8.4	52.5 $\pm$ 7.9	64.8 $\pm$ 7.1	53.1 $\pm$ 7.7
Distal	2	151 $\pm$ 37	148 $\pm$ 9.5	185 $\pm$ 32	217 $\pm$ 49
Children (all midshaft)					
All	10	102.1 $\pm$ 2.0	103.3 $\pm$ 2.6	100.1 $\pm$ 3.1	101.4 $\pm$ 2.6
Male	6	105.1 $\pm$ 2.5	107.2 $\pm$ 2.3	99.1 $\pm$ 3.1	99.1 $\pm$ 3.2
Female	4	95.5 $\pm$ 2.5	97.5 $\pm$ 1.5	102.9 $\pm$ 4.9	106.2 $\pm$ 1.2

* significant difference ($p < 0.001$) between medullary and cortical values**Table V.** Bone loss reported after limb injury

Author	Site of fracture of injury	Site of measurement	Time (yr)	Bone loss (%)	Technique*
Nilsson (1966)	Tibia	Distal femur	15	25	DPA
Bjork and Lempert (1967)	Below-knee amputation	Proximal tibia	—	50	CW
Nilsson and Westlin (1969)	Post-menisectomy	Distal femur	5	7	DPA
Westlin (1974)	Colles' fracture	Midshaft radius and ulna	1	18	SPA
Westlin (1974)	Colles' fracture	Distal radius	1	18	SPA
Nilsson and Westlin (1977)	Midshaft radius and ulna	Distal radius	5	15	SPA
Andersson and Nilsson (1979)	Tibia	Proximal tibia	1	25	DPA
Nilsson and Obrant (1983)	Tibia	Femoral cortex	10	10	CW
Finsen and Benum (1986b)	Colles' fracture	Distal radius	2.5	+36	SPA
Finsen and Haave (1987)	Tibia	Distal tibia	2.5	7	SPA
		Distal femur	2.5	8	SPA
Ahl, Sjoberg and Dalen (1988)	Ankle	Calcaneus	3	7	SPA
Finsen (1988)	Tibial osteotomy	Distal tibia	2	10	SPA
Finsen et al (1988)	Femur	Femur	2	12	DPA
Ulivieri et al (1990)	Tibia	Distal tibia	0.3	50	DPA
Bickerstaff et al (1993)	Colles' fracture	Metacarpals	1	25	SPA
Eyres et al (1993a,b)	Leg lengthening	Distal tibia	1.5	50	DEXA
Eyres and Kanis (1995)	Tibia	Distal tibia	5 to 11	50	DEXA

* DPA, dual-photon absorptiometry; SPA, single-photon absorptiometry; DEXA, dual-energy X-ray absorptiometry; CW, cortical width on radiographs

more proximal fracture have also been described (Schultz 1990). There is a well-known relationship between the risk of fracture and BMD (Cummings et al 1993), and the increased risk is likely to be related to persistent osteopenia. The degree of bone loss which we report is commensurate with the apparent increase in risk of fracture.

The causes of bone loss in the distal tibia after injury are uncertain, but are likely to be related to changes in bone remodelling secondary to changes in the local blood supply to the limb, to altered mechanics or to a stress-protection effect. Normal bone tissue undergoes continuous remodelling at specific loci on periosteal and endosteal surfaces and within the cortex; these leave behind new units of bone tissue (Parfitt 1983). At these sites, osteoclasts erode a

cavity which is subsequently filled by new bone synthesised by osteoblasts. The activities of osteoclasts and osteoblasts are therefore tightly coupled, but are sensitive to mechanical, electrical and hormonal stimuli (Lanyon 1990). After immobilisation, disuse or injury, there is good evidence that bone turnover is increased, but that the amount of new bone laid down in erosion cavities is less than normal (Minaire et al 1974). Both the increased turnover of bone and the imbalance between resorption and formation give rise to skeletal deficits (Kanis 1991).

In contrast to the reduced BMD distal to tibial fractures, we found a consistent increase in BMD at the site of fracture in adults, with a persisting increase in the width of the bone at the old fracture site. These persistent features in

response suggest that remodelling of callus is slow and incomplete in adults. Loss of distal bone may well predispose to later fractures, but the increased bone density at the old fracture site may be protective. This protective effect has been described for Colles' fractures (Finsen and Benum 1987) and for hip fractures (Finsen and Benum 1986a) in adults.

In contrast to our findings in adults, we found no residual deficits in BMD in children reviewed from six to nine years after injury, despite the marked loss which we identified in our prospective series, and have reported after osteotomy (Eyres et al 1993a). There was some persistent sclerosis at childhood fracture sites but this was much less than that after adult fractures. Our findings confirm that the potential for modelling and remodelling of bone is greater in children than in adults.

Conclusions. Tibial fractures appear to be associated with marked and sustained losses of bone at sites distal to the fracture. Such losses are recoverable during childhood, but in adult life they persist for many years, and perhaps indefinitely. The increased risk of refracture at a site distal to the first fracture makes it important to identify the causes of these losses in the hope that they can be modified by treatment.

We thank Ms D. Charlesworth for her technical assistance.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

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