BONE DENSITY AFTER RIGID PLATE FIXATION OF TIBIAL FRACTURES

A DUAL-ENERGY X-RAY ABSORPTIOMETRY STUDY

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We used dual-energy X-ray absorptiometry (DEXA) to measure the bone mineral content (BMC) of both tibiae in 13 patients who had been treated for a tibial fracture by rigid plate fixation. Within two weeks of plate removal the BMC was significantly greater in the bone that had been under the plate than at the same site in the control tibia. An unplated area of bone near the ankle showed a significant decrease in BMC at the time of plate removal with subsequent return to the level of the control tibia during the ensuing 18 months.

We conclude that osteoinductive influences outweigh the potential causes of osteopenia, such as stress shielding and disuse, and that, contrary to expectation, demineralisation is not a factor in the diminished strength of the tibia after plating for fracture.

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When diaphyseal fractures of the tibia are treated by open reduction and application of a metal plate, utilising the principle of dynamic compression, the implant diverts load away from the bone (stress shielding) and it is widely believed that the bone then becomes osteoporotic (Akeson et al 1976).

In animal experiments bone has been shown to have reduced strength characteristics after the removal of rigid internal fixation devices (Tonino et al 1976; Låftmann, Sigurdsson and Strömberg 1980; Låftmann et al 1989). The causes of this weakness are thought to be due to two

different mechanisms, cortical atrophy and the effect of the residual screw holes.

Uhthoff and Dubuc (1971) plated osteotomised canine femora for periods ranging from 2 to 30 weeks. They found that this resulted in cortical thinning, trabeculation of the cortex and persistence of disorganised woven bone at the osteotomy site. Most studies have shown that the previously plated segment of bone has thin cortices and an enlarged medullary cavity from endosteal resorption (Akeson et al 1976; Moyon et al 1978; Paavolainen et al 1978; Slätis et al 1978; Strömberg and Dalen 1978; Terjesen and Benum 1983; Låftmann et al 1989). Carter, Vasu and Harris (1981), however, in animal studies reported thickening of the cortical bone adjacent to the plate due to new bone formation.

Two hypotheses, which are not mutually exclusive, are proposed to explain the phenomenon of cortical atrophy observed in animal models. The first, and most widely held view, is that the rigid plate diverts compressive bending and torsional stresses away from the bone, which reacts by reducing its cortical thickness and bone mass in accordance with Wolff's law (Wolff 1892; Carter et al 1984; Perren 1991). The second hypothesis is that the rigid plate compromises the periosteal blood supply causing ischaemia and resorption of the underlying bone. This latter concept has led to the development of the 'limited contact - dynamic compression plate' (Perren 1991).

The increased porosity (trabeculation) of cortical bone has been investigated by microscopy, microradiography (Paavolainen et al 1978; Slätis et al 1978; Strömberg and Dalen 1978; Terjesen, Nordby and Arnulf 1985) cross-sectional point counting (Strömberg and Dalen 1978), single and dual photon absorptiometry (Terjesen and Benum 1983; Rosson, Petley and Shearer 1991), and by gamma ray absorption (Tonino et al 1976). All studies found an increase in the porosity of the cortical bone under the plate. Most of them, however, were performed on intact animal long bones. Låftmann et al (1989) combined an osteotomy with plating and reported an initial increase in bone mineralisation, as measured by ash weight, only to find that it soon returned to normal and subsequently became atrophic. Some studies have

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compared the effect of plates of different rigidity and found that rigid plates resulted in more stress shielding and greater subsequent bone resorption (Akeson et al 1976; Moyon et al 1978; Carter et al 1984).

Most animal studies have found a direct relationship between osteopenia and bone weakness. The reduction of strength of rabbit tibiae after 12 weeks of plating varied from 28% to 53% (Låftmann et al 1980; Terjesen and Benum 1983). There was a non-linear recovery curve, the period required to regain maximum strength being approximately half the duration of the plate fixation.

Only one study has shown plate-induced osteopenia in man. Terjesen et al (1985) used quantitative CT in previously fractured and plated femora and found an 11% reduction in bone mineralisation but no evidence of diminished cortical width. Rosson et al (1991), who investigated previously fractured and plated forearm bones using single photon absorptiometry, found atrophic bone changes only when plates were removed prematurely (less than 21 months). The presence of plate osteopenia therefore has not been conclusively demonstrated in fractured, plated, human, long bones after the fracture has united.

Tibial plates remain in place for 12 to 18 months on average. When they are removed there is a period of relative weakness of the bone which is said to be associated with an increased likelihood of refracture (Buckwalter and Cruess 1991). It has been assumed that there is then a gradual return to normal bone strength. The duration of this period of relative weakness is unknown.

Our study aimed to test the following hypotheses: 1) that a relative osteoporosis exists in tibiae after the application of rigid AO plates for the management of fractures of the tibial diaphysis; and (2) that this osteoporosis is reversed after removal of the plate.

Dual-energy X-ray absorptiometry (DEXA) is a minimally invasive technique for measuring bone density. X-rays of two energies are passed through a calibrator and subsequently through the subject, and a detector assesses their differential absorption by the tissues.

DEXA has several advantages over existing forms of densitometry. It is more precise than single or dual photon absorptiometry or quantified CT and its results are more reproducible (Cullum, Ell and Ryder 1989; Sartoris and Resnick 1989; Glüer et al 1990). Tiedeman et al (1990) showed a good correlation between densitometric evaluation and bone rigidity.

PATIENTS AND METHODS

We studied ten men and three women with an average age of 26.2 years (16.5 to 42.4) all of whom had sustained a fracture of the tibial diaphysis which had been plated with an AO Narrow Dynamic Compression Plate (AO International, Davos, Switzerland). The fractures had united clinically and radiologically at the time of plate

removal. The average duration of plate fixation was 14.8 months (10 to 24). No patient suffered from any medical disorder, or was taking any drugs, such as oral contraceptives, which could affect bone metabolism. All the plates were removed under general anaesthesia by one of the authors. The patients were advised to avoid contact sports and heavy manual work for a period of six months.

Bone mineral content was assessed using DEXA (Hologic QDR - 1000/W, Waltham, Massachusetts). We examined the area beneath the plate (the region of the fracture) and the tibia above the ankle (Fig. 1). The region beneath the plate was subdivided into the area under the proximal portion of the plate (P1), that under the middle of the plate (P2) and that under the distal end of the plate (P3). The supramalleolar region was subdivided into the mid-distal region (D1) and the ultra-distal region (D2), 6 mm proximal to the joint line, well above the subchondral bone plate. The DEXA results are expressed as grams of hydroxyapatite per centimetre of bone.

The first scan was performed within two weeks of the removal of the plate in all 13 patients. Five patients were subsequently lost to follow-up. The remaining eight were rescanned between 12 and 18 months later. Both tibiae were scanned on both occasions. To ensure that we used equivalent areas of the tibia, the distance of the fracture from the ankle was measured and the equivalent region on the contralateral tibia was scanned.

The data are expressed as the mean BMC at each level of the tibia $\pm$ the standard error. The same data are presented as a ratio of fractured to control limbs at the equivalent sites of the tibia (Table I) to show the differences between the limbs. It is not possible to compare different areas of the tibia because of the

Fig. 1

The areas scanned by DEXA after removal of the rigid plate.

Table I. BMC (g/cm hydroxyapatite crystal; mean $\pm$ SEM) of 13 fractured and control tibiae within two weeks of removal of the plate

	Fractured tibiae	Control tibiae	Difference (per cent)	Ratio
Region near fracture				
P1: proximal	3.94 $\pm$ 0.21	3.47 $\pm$ 0.13	+13.65*	1.14
P2: middle	4.09 $\pm$ 0.28	3.43 $\pm$ 0.15	+19.38*	1.19
P3: distal	3.67 $\pm$ 0.32	3.20 $\pm$ 0.16	+14.80*	1.15
Region near ankle				
D1: mid-distal	3.04 $\pm$ 0.21	2.94 $\pm$ 0.16	+3.42	1.03
D2: ultra-distal	3.76 $\pm$ 0.33	4.07 $\pm$ 0.22	-7.51†	0.92

* represents an increase in the fractured limb compared with the control, $p < 0.05$ † represents a decrease in the fractured limb compared with the control, $p < 0.05$ **Table II.** BMC (g/cm hydroxyapatite crystal; mean $\pm$ SEM) of eight fractured and control tibiae 12 to 18 months after removal of the plate

	Fractured tibiae	Control tibiae	Difference (per cent)	Ratio
Region near fracture				
P1: proximal	4.14 $\pm$ 0.24	3.30 $\pm$ 0.14	+25.45*	1.25
P2: middle	4.32 $\pm$ 0.37	3.27 $\pm$ 0.17	+32.11*	1.32
P3: distal	3.84 $\pm$ 0.44	3.12 $\pm$ 0.21	+23.07*	1.23
Region near ankle				
D1: mid-distal	3.12 $\pm$ 0.33	2.86 $\pm$ 0.21	+9.09*	1.09
D2: ultra-distal	3.74 $\pm$ 0.55	3.85 $\pm$ 0.29	-2.85	0.97

* represents an increase in the fractured limb compared with the control, $p < 0.05$

variations in width and ratio of cancellous to cortical bone.

Statistical analysis was performed by the Wilcoxon matched pairs signed-rank test.

RESULTS

BMC two weeks after plate removal. In 12 of the 13 patients the BMC was significantly greater ($p < 0.05$) at all sites under the plate (Table I); in the other it was slightly diminished. In the mid-distal region (D1) there was also a small but not statistically significant increase. By contrast, the ultra-distal region (D2) of the tibia showed a decrease in mineral content ($p < 0.05$).

The bone width at the fracture site, measured at the time of the first scan, was not significantly different from that of the control tibia.

BMC 18 months after plate removal. The BMC of eight of the 13 patients was measured again 12 to 18 months after removal of the plate. The increase in BMC in the area of the plate was found to have persisted and was significantly greater in all areas than immediately after plate removal (Table II). In the mid-distal region (D1) the increase was also statistically significant. In the ultra-distal (D2) region the relative demineralisation found initially had been reversed.

Changes in BMC over 18 months. The BMC of the control tibiae did not change significantly during the 18 months of the study. The variation was 1.0% or less in the central diaphysis ($p = 0.96$) and 1.4% or less at the distal and ultra-distal sites ($p = 0.38$). By contrast, the previously plated limbs showed a progressive increase in

Fig. 2

Comparison of the mean BMC (g/cm hydroxyapatite crystal) in fractured and control tibiae.

BMC which, when using matched pairs, was 8% in P2 ($p = 0.005$) and 10% in P1 ($p < 0.001$) (Fig. 2); the 6% increase at P3 was not significant. The site D1 showed a smaller but significant increase of 5.5% ($p < 0.01$). The area D2, which had initially shown a significant decrease in BMC, had a reversal of this trend with its value approaching that of the control tibia. The difference between the control and the fractured tibiae at D2 was not significant.

DISCUSSION

This is the first study to have determined the BMC in human fractured and plated tibiae and, as far as we are

aware, it is the first to have used DEXA in this context. DEXA is now established as the most accurate method of determining the degree of mineralisation of bone.

The results show that there was no osteopenia beneath the tibial plates at the time of their removal; contrary to expectation there was an increase in BMC at that site. There was, however, a reduction in BMC above the ankle probably caused by relative disuse. Our study did not determine the BMC *during* the healing phase of the fracture, but *after* union had occurred. The increase in bone density beneath the plate suggests that there is an osteogenic stimulus sufficient to overcome the effects of disuse and stress shielding. The most likely cause of the increased mineral content is the formation of new bone during fracture repair but we find it surprising that the influence extended along the entire area of bone under the plate.

The process of induction of bone continued for more than a year after the plate had been removed possibly

from resumption of normal activity and the cessation of the effects of stress shielding. The increase in BMC was greater in the area under the plate than in the area above the ankle.

The fact that the BMC is high after removal of the plate does not mean that the bone is strong. Strength depends not only upon mineral content but also upon the ultrastructural and biochemical organisation of bone, and localised defects such as screw holes are probably important. This study does no more than show that one of the possible occurrences of weakness after plate removal, namely demineralisation, is not a factor.

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