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Dynamic Interfragmentary Motion in Fractures During Routine Patient Activity

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Natural interfragmentary motion was measured in tibial fractures during normal patient activity, and the results were interpreted using correlations from the literature to examine the influence of natural motion on healing. Ten patients were selected with reduced, diaphyseal tibial fractures stabilized with Orthofix external fixators. Three-dimensional motion was monitored with an instrumented spatial linkage during walking, standing, and muscle activities at 2 and 4 weeks postfixation. Fracture motion arising from dorsal to plantar flexion while supine produced peak cyclic displacements of the same order of magnitude as that seen during weightbearing activity. Thus, therapeutic exercise may be used to provide a stimulus to osteogenic repair processes in patients who are unable to bear weight. In 3 patients, maximum amplitudes of axial motion during walking were 1 mm or greater. This implied regular gap closure and high tissue strains within the 1 mm ± 0.5 mm gaps. In 3 patients, axial motion was less than 0.25 mm. These 2 extremes may indicate a range of displacement relative to gap size that embraces inhibitive and stimulative influences on healing. Transverse shear displacements also varied greatly from between 0.6 and 0.75 mm in 3 patients to less than 0.2 mm in 5 patients.

The biologic process of long bone fracture healing is greatly influenced by the relative motion between the 2 interposing bone fragments at the fracture site.[1,8,13,14,16,18,21] It has been shown that the periodic mechanical application of cyclic interfragmentary motion (passive) influences the speed of early healing as the callus structure becomes established.[7,9,11,14,19,20] Too little activity during early healing has been shown to produce little callus, as seen with fractures that are fully immobilized by internal plating and are healing by the direct process.[1] However, excessive movement will create large peak strains in the intragap tissue in line with the external cortex, and this may increase tissue proliferation[11] externally, with the risk of a delayed or hypertrophic nonunion. Some evidence indicates that cyclic movement, controlled in direction and amplitude, may be beneficial

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to the healing process[7,9,20]; osteogenesis also has been stimulated in intact bone through the use of cyclic strains applied at physiologic rates and frequencies.[10] Passive compressive motion of 0.5 mm has been mechanically applied to stimulate early healing in sheep osteotomies with 3.0-mm gaps, whereas 2 mm of motion[9] inhibited healing. Although it has been shown that patients with plaster casted fractures eventually healed despite substantial shear movement,[12] healing may have been inhibited until movement became constrained by the callus. There also is some evidence that substantial applied shear at osteotomies in the rabbit tibia may cause nonunion,[21] but relatively small shear strains experienced at the cellular level may cause tissue proliferation.[2]

The movement that occurs during the everyday function of the patient creates the environment under which fractures heal in response to normal treatment conditions. Although it may be assumed that physiologic rates, frequencies, and durations of active movement are beneficial,[10] the amplitude and direction of active movements at the fracture are more random and sometimes may inhibit healing. Active movements depend on at least 4 characteristics, in addition to the stiffness of the material at the fracture: magnitude and direction of motion, frame flexibility, gap size, and bone support. The amplitudes of axial and shear movement are influenced by 2 of these that relate to the structure of the fracture: the degree of longitudinal support to tibial load offered by the interposing bone ends in contact across the fracture, and the size of the interfragmentary gap that may be deformed under load. Given the nature of the bone support and the gap, the degree of weightbearing used by the patient and the flexibility of the fixator frame under load also will influence the amplitude and direction of all orientations of movement (axial, angular, and shear).

It should be possible to investigate 1 of these 4 characteristics while holding the other 3 constant. Specifically, the range of the magnitude of movement in each direction may be investigated in a group of patients with variable weightbearing, while keeping constant and representative of common clinical conditions as far as possible, the 3 variables (frame flexibility, gap size, and bone support). The current study used this approach to characterize the amplitude and direction of active movement in tibial fractures and to discuss its potential influence on healing.

PATIENTS AND METHODS

Amplitudes of cyclic interfragmentary motion were measured during walking and standing. The influence of specific muscle activity on fracture movement also was investigated to establish whether predictable patterns of fracture movement can be created in patients unwilling or unable through injury to bear weight. Because weightbearing is a dominant influence on fracture movement, the scale that patients are willing to weightbear was found for the initial 4 weeks after fixing the fixate. In an attempt to keep frame flexibility, gap size, and bone support constant, the fractures chosen were fully reduced, well supported longitudinally through the bone ends in contact, and stabilized by the same fixator in a common geometric configuration. Although the fractures initially were reduced, some variation in the ultimate gap size arising from alignment and resorption or deformation at the fragment ends was inevitable.

Patient Selection and Treatment

Ten patients were selected who were male, aged 22 to 57 years, with diaphyseal tibial fractures, either transverse or oblique, either open or closed, and noncomminuted. Longitudinal stability was achieved at the initial reduction, and all were allowed to take tibial load longitudinally across the fracture. All patients in the study were fitted with Modulsystem Dynamic Axial Fixators (Orthofix, Verona, Italy) in a standardized arrangement, and these have been shown to be of approximately average stiffness when compared with a group of currently available fixators.[4] There were 3 bone screws of 6.0 mm diameter and 130.0 mm length either side of the fracture, and the fixator was positioned 10.0 mm from the end of the screws. The screws were inserted in the medial surface, in a plane at approximately 35° to the sagittal plane.

Patients were encouraged to gradually increase weightbearing (standing and walking) with the aid of crutches, from as early as 1 week after fixators were fixed and were not discharged from the hospital until 20.0 kg (approximately 200 N) was achieved. Radiographs of the fracture region were taken at 2 and 4 weeks in orthogonal planes.

The Measurement of Fracture Motion

The measurement of dynamic interfragmentary motion has been accomplished by developing an instrumented spatial linkage.[5] This is a computerized 3-dimensional measurement system, using magnetic field Hall Effect sensors to continuously monitor independently movement in 6° of freedom (3 linear orthogonal and 3 rotational directions). The device was fixed externally to the inner pair of bone screws across the fracture gap, parallel to the bone and within 5.0 mm of the external cortex (Fig 1A). Motion of the screws in response to tibial load during patient activity was translated to movement of the distal fragment relative to the proximal at the fracture center. This translation assumed that both the 5 mm length of bone screw from the linkage clamp to the bone and the undamaged region of bone, from screw insertion to fracture, were rigid.[5] Because the screw bone anchorage also is assumed to be rigid, data may be used only in which screws are found not to be loose. Fortunately, screw loosening is immediately apparent from such data because it has been found that there is little correlation between the pattern of fracture movement and the pattern of weightbearing. When screws are loose, this may be confirmed by releasing the screw clamps. A calibration jig with vernier scales was used to simulate fracture movement in the 6° of freedom, and correlation with the spatial linkage indicated that linkage measurement was accurate to within ±0.025 mm and ±0.025°.

The 3-dimensional fracture movements were converted to 4 specific directions: axial movement (along the longitudinal axis of the proximal tibial fragment), resultant angular movement (in the plane of maximum angulation), resultant transverse shear movement (perpendicular to the axis of the proximal tibial fragment), and torsional shear movement (rotating about the axis of the proximal tibial fragment), as shown in Figure 1B. Interfragmentary gap size was measured approximately by scaling from the pairs of radiographs, and measurements were confirmed by recording gap closure distances under load (closure was indicated by displacement remaining constant under increasing load).

Patient Test Protocol

Maximum Tibial Load Test

Patients were asked to stand on a wedge shaped heel block and to apply the maximum axial load to the fracture that discomfort would allow. None of the patients indicated that they experienced pain during weightbearing; thus, if loading was below full body weight, the constraint on maximum weightbearing was not considered to be physical. The peak interfragmentary motion corresponding to peak load also was measured.

Fracture Motion During Walking

The same 10 patients adopted their usual gait while walking over a load cell measuring the vertical ground reaction force. Test durations of 6.0 seconds were used, and the test procedure was performed 5 times to establish that peak cyclic movements were repeatable to within 10%. Peak load and the corresponding peak interfragmentary motion were measured.

Fracture Motion During Muscle Activity

Five patients were asked to dorsiflex and plantar flex their ankle while supine. The movement was continued cyclically at approximately 1 cycle each second (1.0 Hz) for 20 seconds while peak interfragmentary motion was measured.

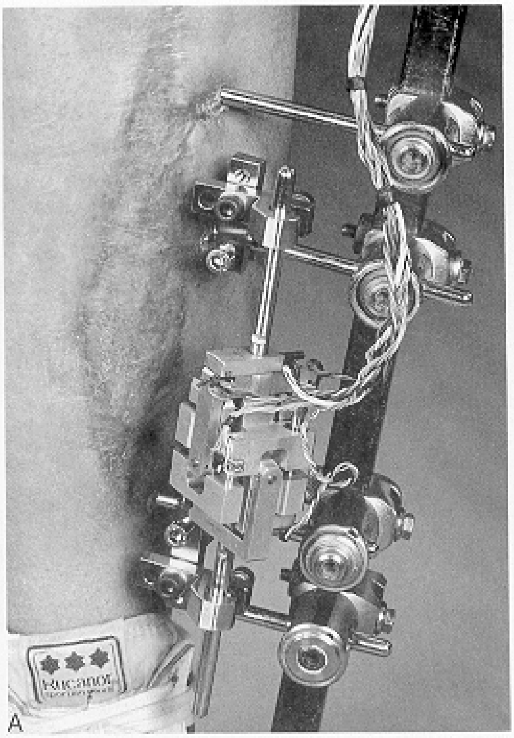
Patients were monitored at 2 and 4 weeks after fixation in these 3 activities.

RESULTS

No patients experienced pin loosening during the period of measurement, and all patients were found to have healed sufficiently to allow removal of the fixation device between 10 and 25 weeks.

Maximum Tibial Load and Axial Movement

This test provided an indication of how soon patients may be encouraged to weightbear on a well supported fractured tibia. Table 1 shows mean weightbearing in the standing position for the group of patients, and the



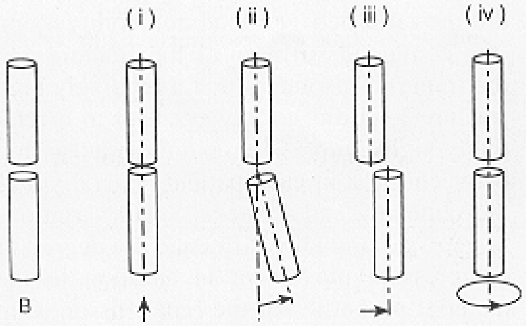


Fig 1A–B. (A) The instrumented spatial linkage measures the 3-dimensional motion of the distal fragment in relation to the proximal at the center of the fracture during patient activity. It consists of 6 magnetic field Hall Effect Devices that measure displacement in 6° of freedom: 3 linear and 3 rotational. (B) Interfragmentary motion at the fracture is expressed as relative movement in 4 orientations: (i) axial, (ii) angular, (iii) transverse shear, and (iv) torsional shear.

corresponding mean compressive movement at the fracture at 2 and 4 weeks. Patients with this type of fracture and fixation device are able to rapidly increase maximum static weightbearing, with apparently little discomfort, to greater than 50% of average body weight (of approximately 650 N). Thereafter, weightbearing only marginally increased to 4 weeks. The corresponding gap compression followed a similar trend, and at 2 weeks, the mean movement was greater than expected in view of the fractures all being fully reduced. Standard errors were large because of the variability of load and compression between patients and, to a lesser extent, because of the small sample number of patients. Figure 2 shows the individual static ground loads for the 10 patients at 4 weeks,

TABLE 1. Maximum Axial Load and Peak Elastic Fracture Compression in 10 Tibial Midshaft Fracture Patients (standing test)

Time	Axial Load (N)	Axial Compression (mm)
2 weeks		
Mean	374 ± 30	0.59 ± 0.11
Range	220–494	0.09–1.16
4 weeks		
Mean	434 ± 44	0.71 ± 0.15
Range	179–550	0.06–1.4

Group means are shown with standard errors.

with the corresponding compressive movements at the fracture site. Load and compression varied substantially among patients, as did the ratio between load and compression (the combined stiffness of the fracture and the frame). This means that a relatively high fracture load did not always lead to a relatively high compression, but the relationship between the 2 in each patient generally was the same at 2 and 4 weeks. Small variations in gap size probably influence the degree of compressive movement in addition to the material properties of the repair tissue. Gap sizes were observed to be within 1 ± 0.5 mm for all patients; the conservative error bounds were applied because of imprecision in defining the bone fracture interfaces from radiographs.

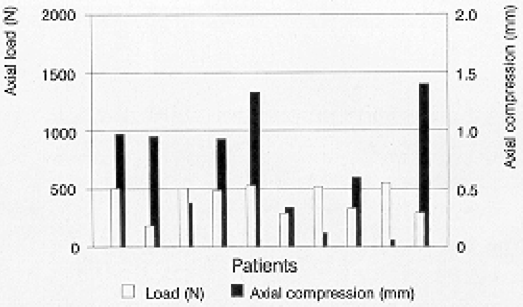


Fig 2. Maximum tibial weightbearing and the corresponding compression of the fracture gap for 10 patients during standing (averaged for the period 0 to 4 weeks after fixation).

Fracture Motion During Walking

These tests characterize peak dynamic movement at the fracture while patients are walking in a manner typical of their daily activity. Table 2 shows the mean ground loads and peak amplitudes of axial, angular, and shear movement for the 10 patients at 2 and 4 weeks.

Walking activity produced a mean maximum ground load for the group within 17% of the mean maximum ability to weightbear (standing). Again, patients reached maximum dynamic weightbearing and maximum compression after only 2 weeks, and mean axial compression for the group was 0.66 mm. Axial compression in 1 patient increased to 1.8 mm at 4 weeks, indicating gap closure and regular end bearing contact deforming or slightly opening the gap. In most patients there was no residual axial movement after the foot was removed from ground contact after each step; thus, the movement response may be described as elastic. The profile of the cyclic axial movement was observed to be approximately sinusoidal.

Figure 3A–C shows the peak amplitudes of cyclic displacements of the fractures in the axial (compressive), angular, and shear directions at 4 weeks. Figure 3A indicates that dynamic weightbearing followed the same pattern as static loading. Load, gap compression, and the

TABLE 2. Peak Ground Load and Peak Elastic Fracture Movement in 10 Tibial Midshaft Fracture Patients (walking test)

		Interfragmentary Motion			
Time	**Ground Load (N)**	*Axial (mm)*	*Angular (°)*	*Tranverse Shear (mm)*	*Torsional Shear (°)*
2 weeks					
Mean	337 ± 53	0.66 ± 0.13	0.60 ± 0.16	0.40 ± 0.10	0.17 ± 0.04
Range	144–576	0.13–1.20	0.11–1.41	0.13–1.00	0.0–0.35
4 weeks					
Mean	338 ± 48	0.65 ± 0.17	0.48 ± 0.10	0.27 ± 0.08	0.13 ± 0.04
Range	143–588	0.1–1.84	0.14–1.01	0.1–0.73	0.0–0.38

Group means are shown with standard errors.

ratio between the 2 all varied considerably among patients. Individually, dynamic load was less than static load, but the dynamic and static load to compression ratios were largely the same, indicating consistent fracture site stiffness properties for both activities.

Angular movement (Fig 3B) and transverse and torsional shear (Fig 3C) were as substantial as axial movement and varied considerably from patient to patient. Table 2 shows that mean angular and shear movements for the 10 patients decreased between the second and fourth week, whereas load remained constant. The transverse to torsional shear ratio was approximately 2 when both were present in a fracture (in 8 patients), implying the 2 are related.

The greatest peak amplitudes of movement observed in the patients with tibial fracture during the first 4-week period were 1.84 mm (axial), 1.41° (angular), and 1.00 mm (transverse shear). Peak axial movement was observed to correlate well with peak angular movement for each patient and almost as well with transverse shear (but not torsional). This is almost certainly related to the 1 sided support to the tibia provided by the unilateral frame; tibial load causes a bending moment about the fixator column, which creates axial compression and angular movement (in the plane of the pins) at the fracture site. Although the plane of the fixator remained common to all patients anatomically, no commonality was found for the anatomic direction of the plane of maximum angular motion.

Transverse shear also may arise parallel to the pins because the pivot of angulation between the fragments, caused by column bending, is shifted along the tibial axis from the fracture site. This correlation between the movements in 3 directions also was evident in the standing test for maximum axial loadbearing, where nonaxial tibial loads probably are minimal. Torsion remained less than 0.5° for all patients. In general, standard errors were large, illustrating great interpatient variability, despite similar geometries of the fractures and frames.

Fracture Motion Arising From Muscle Activity

Table 3 shows that maximum movements caused by dorsi flexion and plantar flexion were of the same order as those caused by

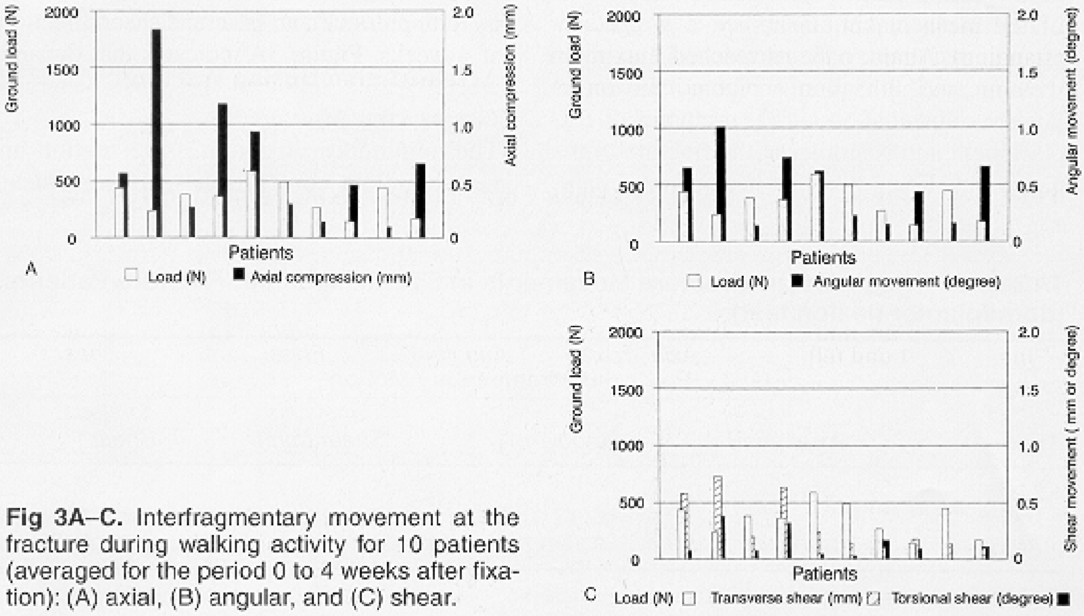


Fig 3A–C. Interfragmentary movement at the fracture during walking activity for 10 patients (averaged for the period 0 to 4 weeks after fixation): (A) axial, (B) angular, and (C) shear.

dynamic weightbearing during walking activity. The transverse to torsional shear ratio was approximately 1.5; again, this was not very different to that found with walking.

DISCUSSION

The Nature of Dynamic Interfragmentary Motion

Compressible gaps in the fractures may have originated from stress induced resorption of bone at the fragment ends, debriding of the surfaces in contact as a consequence of regular weightbearing, or simply through an incomplete reduction between the 2 irregular surfaces. The combination of compressible gaps and flexible fixator frames (operating in the locked mode) allowed elastic interfragmentary motion in response to weightbearing because interfragmentary displacement was fully recovered immediately on unloading the tibia. Because fracture material alone generally is considered to be viscoelastic (where recovery of movement is time dependent), the spring response of the loaded frame appears to be forcing the immediate elastic recovery of the gap. Thus, compressive movement is proportional to load until end bearing contact of the interposing bone ends restricts additional displacement. Therefore it is not surprising that the cyclic motion arising from walking and dorsal and plantar flexion is largely sinusoidal about the midpoint of displacement and is similar at the fracture to the passive cyclic motion that has been mechanically applied in other studies[7,9] to stimulate callus healing. However, although passive motion may be applied at an optimum amplitude and direction to stimulate healing, active motion during walking with amplitudes and directions of motion that are not strictly controlled may in some patients have a deleterious effect on healing.

Active Motion During Dorsal and Plantar Flexion

The greatest interfragmentary movements for the muscle activity group were substantial, giving 0.74 mm, 0.76°, and 1.08 mm for axial, angular and transverse shear movement, respectively. This is not surprising because it has been shown that muscle activity during weightbearing can produce movements momentarily as great as 5 times that of typical weightbearing movements.[5] The significance for treatment is that the fracture motion associated with callus healing can be provided by simple exercise routines, in which patients are unable to weightbear because of injury or because they are unwilling. Physiotherapy also may be used to stimulate callus healing in patients who are completely immobilized because applied ankle rotation with the patient passive caused motion similar to that provided by active dorsal and plantar flexion.

Active Motion During Walking

Compressive Motion

The amplitudes of compressive motion appear small in comparison with movements

TABLE 3. Peak Elastic Fracture Movements in 5 Tibial Midshaft Fracture Patients (dorsi/planter flexion test)

	Interfragmentary Motion			
Time	*Axial (mm)*	*Angular (°)*	*Transverse Shear (mm)*	*Torsional Shear (°)*
4 weeks				
Mean	0.42 ± 0.14	0.43 ± 0.15	0.48 ± 0.18	0.34 ± 0.19
Range	0.06–0.74	0.07–0.77	0.12–1.08	0.0–1.00

Group means are shown with standard errors.

with other forms of external fixation, such as plaster casting or bracing.[12] However, healing processes in fracture tissue probably respond to strain amplitude and only indirectly to displacement amplitude, and therefore gap size as well as gap compression is important. At the upper end of the range of movement, amplitudes were 1 mm or more in 3 patients. Because patient gap sizes were approximately 1 mm, gap closure occurred regularly during walking. This would have caused high strains in the intragap tissue and in any external callus immediately adjacent to the gap.[3] Not until external callus has been established adjacent to the gap will interfragmentary motion be reduced,[17] causing compressive strain within the intragap tissue to fall to tolerable levels to allow primitive soft fibrous tissue to be replaced by calcified tissue through endochondral ossification.[21] Thus, at least 30% of the patients may have encountered mechanical environments that initially were inhibitory to healing. At the lower end of the range of motion, the amplitudes for 3 patients were between 0.1 and 0.25 mm, indicating a wide range of motion in the study group. Very small amplitudes of 0.1 mm may have a nonstimulatory influence on callus healing similar to that of rigid fixation,[8] although the midrange amplitudes may be beneficial.[7] It is possible that midrange strains may allow early intragap bridging by fibrous cartilage and endochondral ossification with little external callus being needed, which may avoid the delay associated with establishing abundant peripheral callus to reduce strain.

Angular and Shear Motion

Contrary to general opinion, the measured interfragmentary motion on weightbearing was not predominantly axial but angular (1.41°), and transverse shear movements (1.0 mm) were at least as great as axial motion. This probably is typical of well supported fractures because the bone ends in contact constrain against axial movement but only partially resist transverse shear and do little to resist angular movement. The absence of any substantial constraint to movement in the angular direction during walking is potentially harmful in view of the tendency to larger angular movement observed in some fractures. Angular movement (when added to axial movement) in some patients could cause substantial compressive longitudinal strain in the plane of maximum angulation, where the risk of refracture is greatest. In support of this theory, lines of weakness located laterally through the thickening collar of callus have been commonly observed from radiographs during early healing (Fig 4); this may be symptomatic of refracture caused by angulation. Thus, continual refracture during walking may be the stimulus for additional external layers of callus to be formed. This will thicken the collar of callus, increase angular constraint, and reduce axial strains at the periphery to below yield to allow healing to progress beyond the callus formation stage. However, the production of abundant restraining callus may not be the most efficient course of healing. If angular movement was better constrained by the stabilizing device or by reduced weightbearing, a smaller volume of callus may be sufficient to induce an early commencement of the bone formation and remodeling stages after the cessation of high longitudinal strains.[6] Internal fixation probably reduces angular movement, as well as axial and shear, and this may be a contributory reason for the reduced amount of callus seen with this form of treatment.

Cyclic Interfragmentary Strain and Healing

Simply evaluating the relationship of motion with healing outcome for the 10 patients would be misleading. A much larger patient group would be needed to neutralize the influence of many other variables, such as periosteal condition, severity of injury, and age. However, it may be of interest to approximate healing outcome by correlating subject data with animal studies, for which many of the variables have been eliminated. Experi-

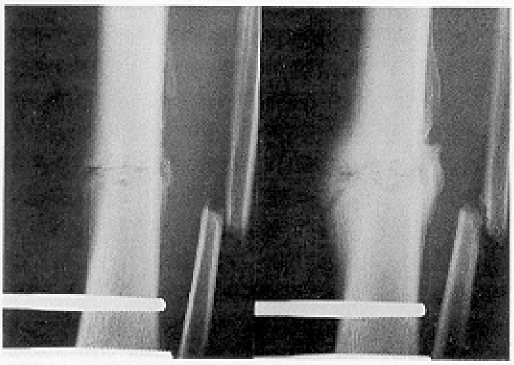


Fig 4. A transverse diaphyseal tibial fracture stabilized by an Orthofix unilateral external fixator. The radiographs show a fracture line extending from the interfragmentary gap to the peripheral callus tissue at the medial surface adjacent to the fixator, at (left) 8 weeks and at (right) 18 weeks.

mental fractures in sheep tibia[7] produce callus of similar morphology, scale, and shape to that seen with human fractures. Thus, similar strain distributions throughout the callus caused by equivalent gap compressions may enable first order approximations to be made of the influence of motion on healing for the group of patients.

For the 3 patients with amplitudes greater than 1.0 mm, interfragmentary strain (gap compression per gap size) will be between 67% and 100%, allowing for the error bounds associated with gap measurement. This may have been deleterious to healing because 2-mm amplitudes in 3-mm gaps (67%) were shown in animals to cause an inhibitive effect, whereas 0.5-mm amplitudes (17%) stimulated healing.[9] In addition, it has been shown that compressive strains of 67% may be expected to cause yield failure when applied to homogeneous repair tissue, such as fibrous cartilage, cartilage, and woven bone; although initial granulation tissue may remain elastic at strains as great as 100%.[15] The magnitudes of additional interfragmentary strain arising from the angular displacements can be illustrated in a simple approximation of gap closure at the compressive cortical surface. For a tibial diameter of 22 mm and a 1.0-mm gap, an angular movement of 1.4° implies additional gap closures of between 20% and 50% and double these values if the pivot point is at the cortical surface, rather than at the long axis of the bone. When gap closures arising from angular displacements are added to the interfragmentary strain generated by axial displacement, increased interfragmentary strains at the compressive cortical surface may have caused inhibitory strains in more of the patients. Also, the mechanical environment is probably not improved when interfragmentary shear strains of between 15% and 40% (arising from transverse motions of 0.6 mm to 0.75 mm in 3 of the patients) and compressive interfragmentary strains are applied to the fracture site.[21]

Limitations of the Study

Although amplitudes of interfragmentary motion are measured accurately, gap sizes are not. This has caused some uncertainty in defining proportional gap closures. However, the difficulty has been resolved by evaluating the range of closures possible arising from the bounds of gap error, rather than single values. Some uncertainty is caused by the assumption, implicit in the approach to estimating strain level, that the maximum strain experienced by the tissue is at least as great as interfragmentary strain. However, because tissue strains vary due to localized regions of high and low distortional and volumetric changes, they are likely to be both greater and smaller than interfragmentary strain. Thus, the anticipated influence on healing for patients with high interfragmentary strains also will apply when tissues are experiencing the full distribution of strain. Only intragap tissue and external callus tissue immediately adjacent to the gap has been discussed because these are present in some form during early healing and because the measurements of motion correspond to the initial callus formation stage. Only the initial fixation stage has been examined because many clinicians believe that early inducement and maturation of initial callus growth is a precursor for rapid union, and it has been demonstrated that motion may influence early callus growth.[11]

The range of active motion found in the fractures would seem to be capable of causing stimulatory and inhibitory influences on early healing; the path is determined mainly by gap size and the patient's instincts regarding weightbearing. Such inhibitory influences on healing could be a contributory cause of delayed union or nonunion in patients who are overactive or underactive weightbearers. The findings suggest that an effort should be made to suppress motion in patients with large gaps who may be too willing to weightbear and to encourage patients with well supported fractures who do not weightbear. It is possible for patients to commence stimulatory interfragmentary motion by therapeutic exercise immediately after surgery before mobility is restored and as soon as pain and discomfort allow.

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