*J. Biomechanics*, Vol. 3, pp. 263–270. Pergamon Press, 1970. Printed in Great Britain

# MECHANICAL AND MORPHOLOGICAL ASPECTS OF EXPERIMENTAL OVERLOAD AND FATIGUE IN BONE*

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**Abstract** – The author reports the results of experiments in which long bone fatigue is produced in 30 pairs of dog ulnas by applying opposing forces at both extremities thereby causing a strain. On a force-deformation curve the force axis indicates a zone of load, an intermediate zone of fatigue and a zone of overload; the deformation axis shows an elastic zone and a plastic zone. These zones are defined in the text.

The fatigue test in the load zone does not indicate either weakening of the bone or histological signs of fatigue. The fatigue test in the intermediate zone is accompanied by a progressive bone deformation, a diminution of resistance and microscopic lesions of fatigue. The fatigue test in the load zone with brief applications of overload produces the same histological lesions. These lesions, which are slip lines and microfissures striating the compressed cortex, are the cause of progressive bone weakness and of stress fractures.

## 1. INTRODUCTION

THE FIRST evidence of the effects of stress in living bone was given by Müller (1922) who used dogs. He removed a segment of one radial diaphysis and allowed the dog to walk on its front paw, which was weakened by two-thirds. An overload on the ulna resulted and consequently a fatigue fracture appeared. Rutishauser and Majno (1949, 1950), after having used this technique, described the histological aspects of stress. In 1967 Tschantz and Rutishauser, using this same technique, analysed in great detail the stress lesions and the successive phases of bone transformations. These authors showed that dogs, after a few hours of walking on their ulna, developed stress lesions that finally caused a fatigue fracture. They gave as evidence that in the compressed cortex oblique prefailure lines appeared similar to those which developed in minerals (Rinne, 1928) and polymerised crystals (Williams, 1969), as well as in metals subjected to fatigue or deformed in the plastic phase.

More recently Tschantz *et al.* (1968) showed that in man such lesions occur in walking fractures. A description of these oblique lines is a prerequisite to the understanding of stress fractures (Fig. 1). These strongly hematoxylinophilic lines appear very distinctly on the eosinophilic base of the bone cortex under overload. They appear only in the compressed area. In the sagittal section these lines mainly consist of innumerable crossing fissures which are obliquely oriented and form an angle of about 28–35° with the long axis of the diaphysis. One is able to distinguish both narrow and wide lines. They often begin as narrow lines and become progressively larger, terminating as fissures. These prefailure lines are of variable length, some crossing the cortex from one end to the other, independent of the lamellar structure. They often begin under the periostium and end in a large osteone or they may cross small osteones without changing planes or direction. One also finds them in the large trabeculae of spongy bone. These oblique lines can be considered to result from shearing induced by loading when the stress produced plastic deformation of the sample. This is also the opinion of Dempster and Liddicoat (1952),

*Received 7 October 1969.

who were able to obtain similar fissures in cubes of compact bone which had been compressed longitudinally up to fracture. The same picture of bone under compression was demonstrated by Frost (1960, 1965) and Frost *et al.* (1961).

Ascenzi and Bonucci (1968), in demonstrating the mechanical qualities of the osteones, described the appearance of microscopic fissures induced by compression during the plastic deformation phase. These fissures developed independently of the bone structures, forming an angle between 30 and 35° with the osteone axis. Under the electron microscope the edge of the fissure showed a disrupted crystal pattern structure and ruptured collagenous fibres.

Very little information about the mechanical conditions necessary for the appearance of oblique lines and microfissures can be found in the literature. In order to verify the hypothesis that they are the initial morphological expression of overload and bone fatigue, we have attempted to recreate experimentally *in vitro* on the dog ulna the mechanical conditions leading to the appearance of the oblique slip lines, duplicating as closely as possible the mechanical conditions found in Müller's *in vivo* experiment (1922).

These mechanical conditions should lead not only to slip lines, but also to a bone fatigue characterised by a lessening of bone resistance and eventually a fracture.

Before discussing the experiment, it is worthwhile to examine the mechanical properties of bone. Numerous publications explain the mechanical qualities of bone tissue subjected to forces of compression, tension, and torsion (Rauber, 1876; Hülsen, 1896; Calabrisi and Smith, 1951; Dempster and Liddicoat, 1952; Evans 1961; Frost *et al.*, 1961; Frost, 1964; Currey, 1962 a, b, 1964).

When the lengthening or the shortening of a bone sample under progressive tension or compression is measured, a graphic representation of the deformation as a function of the forces is obtained. We have chosen this type of graph to illustrate our biomechanical demonstration.

In 1952 Dempster and Liddicoat published a stress–strain curve for compact bone in compression. The curve shows that the bone specimen passes through an elastic deformation phase that is reversible and represents practically one-half of the total deformation. The elastic deformation phase is followed by a plastic phase which is partially reversible. More recently Ascenzi and Bonucci (1967, 1968) measured the mechanical properties in tension and compression of specifically isolated osteones. They demonstrated that these specific properties varied according to the direction of the collagen bundles in the osteone, and concluded that the osteone is the mechanical unit of compact bone.

## 2. MATERIALS AND METHODS

Our work is based on 30 pairs of adult dog ulnas. The bones were stripped of all muscle and stored temporarily in a deep freezer, the mechanical properties remaining unchanged. These fresh, moist bones were deformed in an automatic press (Fig. 2) which allowed simultaneous measurement of the applied force, the resulting deformation and the speed of deformation of the object.

By means of the first two parameters a force-deformation curve can be easily recorded up to the time of the fracture. Also, the press can be operated in two directions; increasing and decreasing forces can be applied to produce diagrams of loading and unloading at the same time. During the experiment the different points were registered by successive photographs. The ulnas, grouped in pairs from the same animal, are deformed by two opposing forces applied at each end. These forces tend to bend the natural arc of the ulna causing a strain. Deformation is characterised by an increase of the arc and diminution of the chord length.

In our experiment we have chosen the reduction of the chord length as proportional to the deformation. With one ulna we estab-

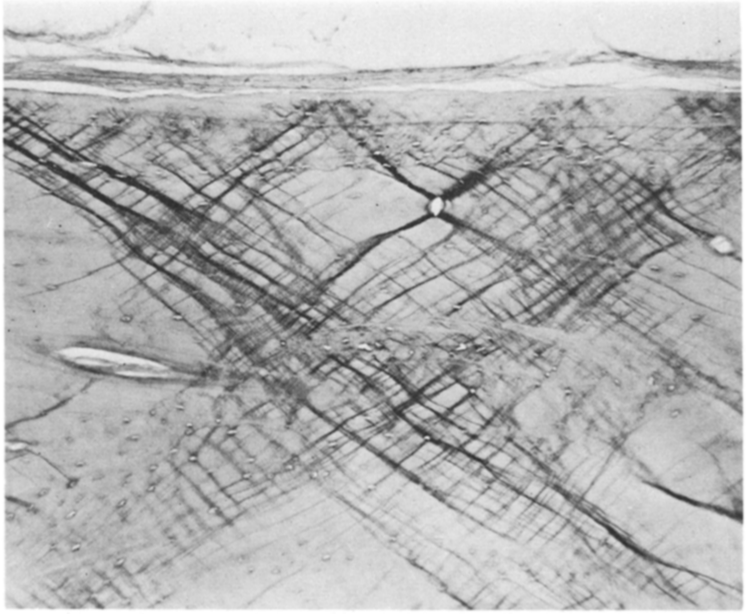

Fig. 1. Zone of plastic deformation in a compressed cortex. Prefailure slip lines and microcracks are obliquely oriented, forming an angle of about 30° with the long axis of the diaphysis hematoxyline eosine stain-88 × sagittal section.

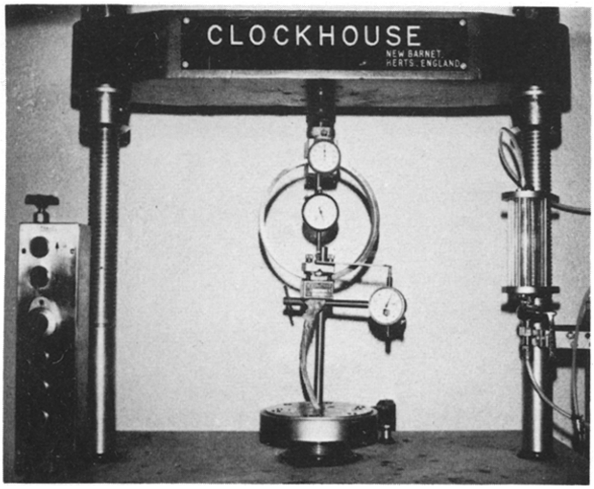


Fig. 2. The press used to test the ulnas.

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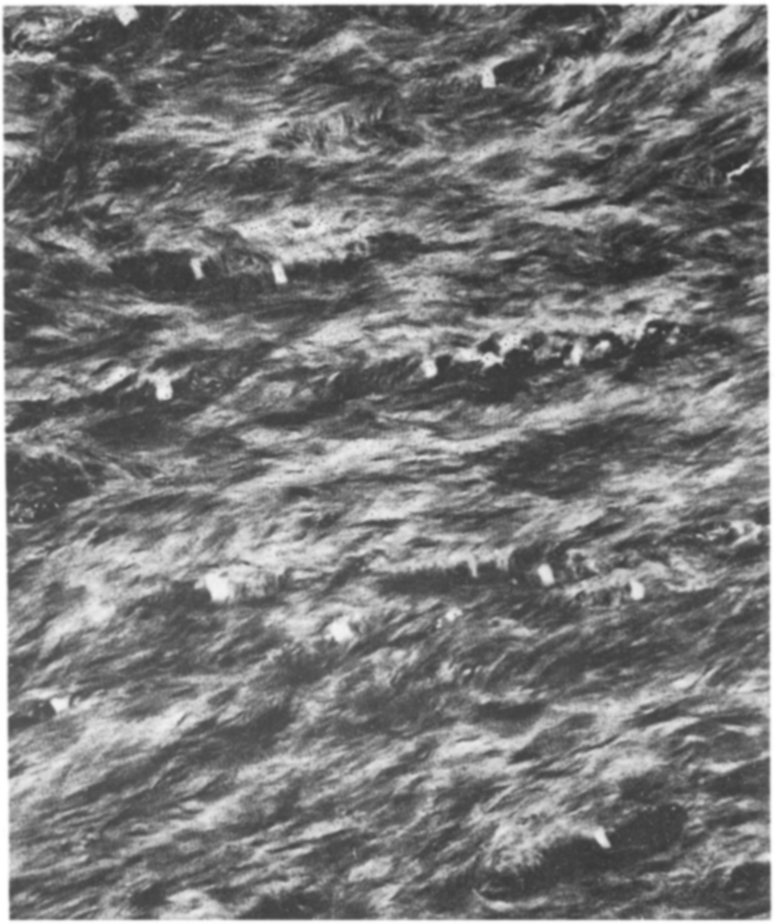

Fig. 7. Slip lines as seen under the electron microscope. The prefailure zones appear darker and perforated with many holes. The collagen bundles are bent in a *Z* fashion in the prefailure zone making it appear like an incomplete shear of the bone. The holes are the cross sections of the canaliculi (6·200 ×).

Fig. 8. In these prefailure zones that appear clearer in the photomicrograph, one sees some free apatite crystals in the optically empty parts. Some collagen fibres are ruptured while others are bent. Some interconnecting osteocyte canaliculi appear enlarged and deformed (12500×). *Note*: The photomicrographs of Figs. 7 and 8 are of *in vivo* experiments.

lished a force-deformation curve going from the resting position to a fracture. This curve served as a reference and enabled us to choose the appropriate values for a fatigue test on the other ulna. The fatigue test consisted of applying rhythmically opposing loads to each extremity of the ulna. Histological observations were made regularly during all phases of the force-deformation curve and after each fatigue test to determine the conditions under which the slip lines appeared.

The bone samples that were kept in 5 per cent formalin were decalcified, embedded in paraffin, cut to $10\mu$ and stained with hematoxyline eosine.

*Technics for electron microscope* (Philips EM 75)

The samples were fixed in phosphate buffered glutaraldehyde, embedded in Epon and cut thin in order to show the microcrack zones. These zones were isolated, stained in $KMnO_4$ according to Tahmission's method (Pease, 1964) and reembedded in Epon before being examined.

Knowing exactly the mechanical characteristics of the tested bone sample enabled us to verify our original hypothesis that the slip lines represent the morphological substratum of an overload or fatigue lesion. This study also demonstrated certain biomechanical aspects of the fatigue fracture.

### 3. ANALYSIS OF FORCE-DEFORMATION DIAGRAMS

A force-deformation diagram is a graphic description of the ulna strain produced by two opposing loads. These forces increase the natural arc of the bone. The strain, which in other words is the decreasing chord length, is plotted on the abscissa, and the force applied is shown on the ordinate axis. The curve serves as a reference for choosing the loads applicable for the fatigue test on the opposite ulna. Such a reference curve was determined for each fatigue test.

First, we analysed the deformation changes in a cross section perpendicular to the long axis of the diaphysis. The changes measured from the surface of the diaphysis are both maximum and in opposing directions (Evans, 1957). The compressive forces decrease linearly from the concave surface to the point $O$ situated on the neutral axis. From point $O$ the forces become negative up to their maximum on the convex surface. The concave cortex is in compression and the convex cortex is in tension. As the arc intensifies, the neutral axis is displaced from the surface of the convex cortex towards the concave cortex.

The first part of the force-deformation curve is practically a straight line whose slope varies in each case, and represents the modulus of elasticity ($E$) which is inversely proportional to the deformation. The first part of the curve closely resembles Hooke's law on elastic bodies. We shall call this zone of force, in which the bone reacts like an elastic body, the *load zone*.

The load zone in our diagram (Fig. 3) is limited by point $O$. At point $O$ the curve starts to incline and no longer follows Hooke's law. With force still applied, the curve passes through its maximum at point $P$, where resistance quickly weakens and the rupture appears at a force distinctly inferior to the force supported by the object at the critical point $O$.

Point $P$ is situated in the overload zone. The

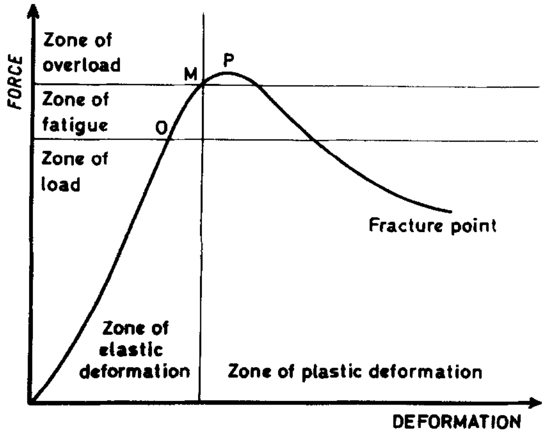


Fig. 3. Force-deformation diagram of a dog ulna.

bone subjected to overload is characterised by the presence of prefailure slip lines in the compressed cortex (Fig. 1).

Between the load and the overload zone there is an intermediate zone characterised by an inclination of the curve. The zone starts at point *O* and ends at point *M* where the first slip lines appear, as seen in the overload zone. We shall call this the *fatigue zone*. The effect of loading-unloading at a frequency of 1–5/sec, as seen in walking or running, produces a progressive bone deformation. This deformation first causes the appearance of slip lines, and if the mechanical stress continues, the appearance of the so-called fatigue fracture. The fracture is always situated in the middle of the territory crossed by the slip lines. On the deformation axis point *M* marks the limit between the elastic and plastic phases. The bone deformed in its elastic phase does not contain slip lines; these appear only in the plastic zone.

*Remarks*

Many experiments have been conducted on bone samples of children, adults and aged persons. These tests show that the plastic phase is short for dense cortical bone and that rupture takes place near point *P*.

In children the plastic phase is much longer, rupture becoming evident after a greater degree of deformation. The mechanical bone properties involved depend upon the degree of mineralisation (Ascenzi and Bonucci, 1967).

*Repeated strain in the load zone* (Fig. 4)

In this case one observes, after a period of adaptation varying from 2–5 min (according to the extreme load values chosen), a stable and closed hysteresis loop. This in no way is influenced by the deformation speed employed or by the number and frequency of the cycles to which the sample is subjected. No fracture can be obtained in this case. Histological controls (observation) do not show the presence of slip lines.

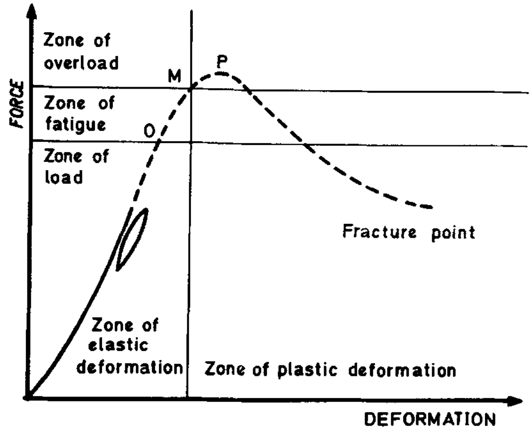


Fig. 4. Repeated strain in the load zone. The stable and closed hysteresis loop is situated on the right of the reference curve.

*Repeated strain in the fatigue zone* (Fig. 5)

A slow bone deformation in the plastic phase is observed under the influence of repetitive mechanical stimulations. In fact, the hysteresis loops are not exactly superimposable as in the preceding case; rather, they remain slightly open from cycle to cycle as the bone deformation is accentuated by finally ending in the plastic phase with the characteristic bone lesions. The number of cycles necessary to obtain a slow fracture depends greatly on the

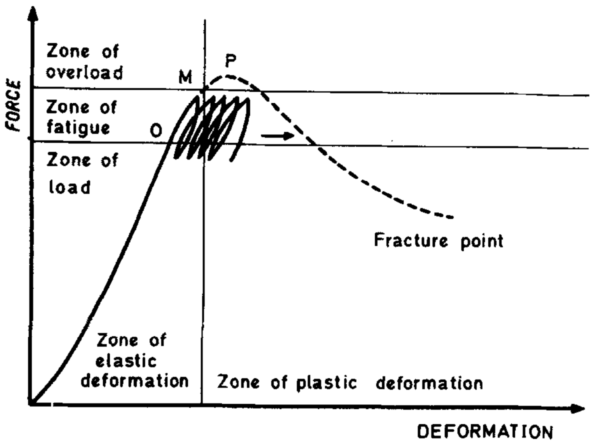


Fig. 5. Repeated strain in the fatigue zone. In this case a slow bone deformation is observed progressing to the plastic phase under the influence of repetitive mechanical stimulations. The hysteresis loops are not exactly superimposable as they remain open.

maximal hysteresis tensions; with higher tensions a smaller number of cycles is necessary to fatigue the bone until a fracture is evident. The cycle frequency and the deformation speed are also determining elements. When the frequency is elevated, deformation occurs more rapidly.

As soon as the deformation of the fatigued ulna is such that it corresponds to a plastic deformation on the reference curve, one observes slip lines in the compressed cortex at the maximum deformation level.

*Repeated strain in the load zone with overload during short periods* (Fig. 6)

This concerns the biomechanical mechanism which is most frequent in early stress fractures. Here the hysteresis loops are closed and stable since they are situated in the load zone, but the short periods of overload cause an added increase of the strain each time. The increment of the deformation (due to the overloading) is not resolved completely since the repetitive mechanical pressure continues immediately afterwards.

The relaxation time between each load is not sufficient to permit reduction of the deformation which tends to be accentuated at each overload. As long as the deformation stays in the elastic zone, the hysteresis cycles are closed and the bone keeps all its elasticity. Histology does not reveal any slip lines.

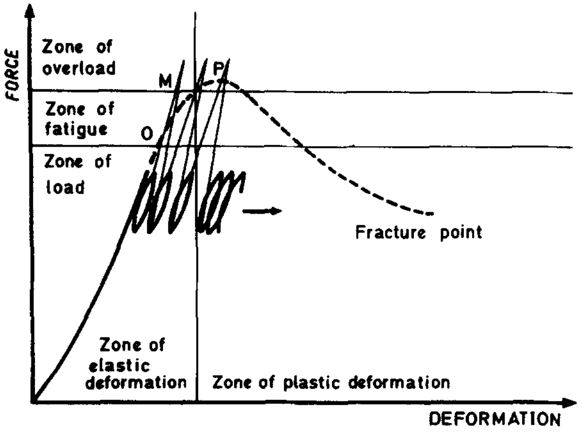


Fig. 6. Repeated strain in the load zone with overload during short periods. The hysteresis loops are closed and stable. The short periods of overload cause an added increase of the strain which becomes plastic after point *M*.

When the deformation reaches the end of the elastic zone, slip lines appear in the compressed cortex at the maximum deformation level. The appearance of slip lines coincides with the opening of the hysteresis loops and with the diminution of the bone resistance. Consequently, deformation occurs during plastic phase. The application of a normal load is then sufficient to create a fracture.

Application of a very short overload past point *M* does not involve an extensive deformation since the bone takes time to be deformed due to a certain internal viscosity.

The bone cushions the shock by absorbing the energy applied to it. It is possible, however, that at the same moment submicroscopic lesions are created which eventually would lead to the plastic lesions previously recorded.

Under the electron microscope these lines appear like parallel zones in which the regular bone architecture is disrupted. The fibre bundles, which are aligned and fixed together, are 'wrinkled' and separated from each other. The double curvature of these bundles on the slip line makes it appear like an incomplete shear of the bone, because not all the bundles are torn but are only bent (Figs. 7 and 8). In the wrinkled zones with collagenous fragmentation the canaliculi often appear optically empty or of a grey homogeneous color which is the sign of degeneration or rupture of the broken extensions of the osteocytes. These lesions represent the first physical manifestation of fatigue in a compressed cortex. No plastic deformation line is observed in the cortex under tension. Rupture occurs perpendicularly to the tension lines without any apparent prefracture lesions. On the other hand, in the compressed cortex the fracture is parallel to the slip lines.

## 4. DISCUSSION

The experiments *in vitro* on fresh dog ulnas reproduced the two mechanical conditions leading to overload lesions, as observed *in* *vivo* by Tschantz and Rutishauser (1967). They resected a segment from the radial diaphysis and let the dog walk on its ulna. The dogs rapidly developed slip lines and microfissures in the compressed cortex of the ulna followed by a slow fracture.

The lesions triggered a bone transformation which was simultaneously osteoclastic in eliminating the necrotic regions and osteogenic in being responsible for the bone consolidation and adaptation hypertrophy. The repairing process lasts over nine months (Tschantz and Rutishauser, 1967). The premature stress lesions, which are identical to the ones we described in our mechanically fatigued bones, shall be called plastic deformation lines. We cannot yet explain why the prefailure lines are absent in the cortex under tension.

Currey (1962b) studied mechanical bone properties and explained them as a function of their inhomogeneous and discontinuous structure. He showed that the multiple internal discontinuities in bone, such as the osteocyte canaliculi, lacunae, blood-channels and the Haversian canals, served as stress concentrators. As such, they reduced the resistance, particularly the tensile resistance. He also showed that fissures and intracortical microfractures which occurred during important stress might stop in one of these lacunae. In this way a lacuna would permit a better distribution of the stress by limiting the spread of the fissures and slip lines.

Returning to the discussion of our graphs, when a dog walks, rhythmically mechanical and azial impulses are produced. These impulses are transcribed on the force-deformation diagram as hysteresis loops.

*Repeated strain in the load zone* (Fig. 4)

When the load is adapted to bone resistance, the strain is not accentuated and the hysteresis loops remain stable as only a purely elastic deformation is involved which does not produce a lesion. These are the normal mechanical conditions usually found in the body. The bone adapts itself by a reinforcement proportional to the stress to which it is subjected (Frost, 1964 a, b; Evans, 1961).

*Repeated strain in the fatigue zone* (Fig. 5)

After a certain number of mechanical stimulations with an extra-heavy load or on a weak bone, a fatigue fracture occurs. The hysteresis cycles in the fatigue zone are not superimposable; the hysteresis loop remains open from cycle to cycle the strain is accentuated, eventually producing a fracture. Microlesions accumulate in the compressed cortex with each cycle, progressively weakening the bone. This is the most frequent phenomenon found in the Tschantz and Rutishauser experiment.

*Repeated strain deformation in the load zone with overload during short periods* (Fig. 6)

When a dog starts to jump or run, it intermittently overloads its ulna, which does not have time to resume its initial form before a new force is applied to it. One observes here the summation of the overloading strain which tends to deform the bone in the plastic phase and to accumulate slip lines. These lesions weaken the bone, therefore, making it susceptible to a fracture with a comparatively lower load.

We did not want to make a quantitative study, since such a study on dog ulnas is only of limited interest. The general shape of the force-deformation curve may vary a little according to the bone sample tested, but we always find the same type of curve with the different zones described on the graphs. Point *P* in the majority of the cases corresponds to a load varying between 100 and 150 per cent of the dog's weight. The applied forces necessary to obtain a fatigue effect correspond to the dog's weight ±20 per cent. The mechanically deformed bone takes a certain amount of time to resume its initial shape. This time characterises the internal shock absorption, which is a physical property that depends on the internal friction. This is demonstrated by heat formation as in the fatigue phenomenon studied in metal.

Förster and Köster showed that internal absorption can be precisely determined on metal samples by resonance (Bogroff *et al.*, 1947). The same type of measurements can be used with bone samples. Although we did not measure this internal absorption exactly, we were able to establish that it varied with the quality of the bone. For instance, the absorption is less for a mineralised bone than it is for a partially demineralised one. Internal absorption is an important factor in explaining bone fatigue. In fact, with repetitive mechanical stimulations the bone does not have time to resume its initial form between each load, but tends only to be more deformed and to accumulate plastic lesions. In studying bone fatigue one has to take into account not only the mechanical characteristics and their frequency but also the bone's ability to absorb them.

Several experimental models have been collected to show the biomechanical mechanism of the overload fracture.

## 5. CONCLUSIONS

These experiments have shown that

(1) The application of one or more stresses in the load zone does not produce microscopic lesions.

(2) A single stress in the intermediate zone (between load and overload) does not produce lesions except during a fatigue test where such lesions do appear.

(3) An overload produces such lesions when the strain overshoots the elastic phase. We see no microscopic lesions when the overload is applied for a short period in the elastic phase.

We can, therefore, say that in the compressed bone cortex all irreversible microscopic or submicroscopic slip lines are a manifestation of plastic bone deformation. The transformations that occur *in vivo* in the compressed bone are due to the necrosis induced by these plastic deformation lines (Tschantz and Rutishauser, 1967).

*Acknowledgements*—This report was done under the supervision of Professor Taillard and in collaboration with the Institute of Morphology (Professor Baud), the Institute of Pathology (Professor Majno) of the University of Geneva and the Technical High School of Geneva (Director, L. Huguenin). Also assisting with the project were Mrs. R. M. Jordi (electron microscopy), Mrs. E. Neumann and E. Halter (histology), H. Masson (technician), E. Denkinger (photography), and C. Ryser and M. A. Tschumi (draughtsmen). We thank them for their collaboration.

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