

# Understanding Fracture Healing Biomechanics Based on the “Strain” Concept and its Clinical Applications

Chápání biomechaniky hojení zlomenin postavené na koncepci „napětí“ a jeho klinické aplikace

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## PREAMBLE

Biomechanical conditions play an obvious role in respect to fracture healing. Fractures can heal solidly or not heal at all depending on a variety of biomechanical conditions. What is the critical biomechanical parameter that determines and guides the mode of healing?

As we compare the following cases it will become obvious that fracture mobility cannot be the main criterion determining the biomechanical conditions that lead to different occurrences or modes of healing. On the one hand, a highly mobile fracture bridged solidly as demonstrated by a mountain goat (Fig. 1). On the other hand, a fracture affected only slightly by mobility due to failed internal fixation did not heal in a young lady<sup>5</sup> (Fig. 2). We propose thinking in terms of tissue defor-

mation (strain) instead of considering only fracture mobility (“stability”). Strain is first and foremost a more appropriate way of understanding fracture healing and improving treatment. The clinical impact of strain will be discussed giving priority to biomechanical effects that, in our understanding, are the inductors whereby we will not address the closely related issue of the bio-

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<sup>5</sup> The two conditions differ in other respects but the main difference is biomechanical



Fig. 1. Solid spontaneous bone bridging of the fracture **in spite of high mobility**. The misalignment is not considered here (U. Geret).



Fig. 2. Young lady with tibia fracture treated in a way that **low mobility was not prevented** (lag screw within the fracture gap. Union at 8 months).

chemical sequence of events (4) that plays the role of the effector.

The term “stability” is widely used in internal fixation. Unfortunately “stability” is used in medical terminology for different, incompatible aspects. Some use it to expressing strength (load at failure), others use it to expressing stiffness (resistance to deformation). We use “stability” to mean stiffness but we will avoid using the term “stability” whenever possible. The term “biological fracture fixation” defines a mode of fracture fixation that comprises flexible fixation for induction of callus and minimal surgical trauma to keep soft tissues and bone healthy. The surgical trauma consists of the trauma to the soft tissues during the surgical approach and the reduction of the fracture as well as of the implant contact which compromises the blood supply to bone. Biological fracture fixation aims at improved reliability of healing, improved resistance to infection and minimized risk of refracture. The advantages are achieved at the cost of more demanding procedures and implants. Precision of reduction is weighed against biological damage and, whenever possible, the fracture should not be exposed for inspection or even “cleaning” of soft tissues.

## THE CONCEPT OF STRAIN

An important biomechanical condition for repair tissue formation and differentiation is cellular deformation. The repair cell does not “see” the amount of fragment movement nor the width of the gap but senses its own deformation, called strain (1, 3, 6, 9, 10). The influence of movement as well as the effects of gap size have been dealt with extensively (2, 7). Deformation of cells in a fracture gap depends upon gap width and amount of relative movement between the fracture surfaces. Such deformation is called strain (which, in its practical application to fracture fixation, equals the amount of fracture gap movement ( $\Delta L$ ) divided by the gap width ( $L$ ) or  $= \Delta L/L$ <sup>6</sup>. This means that depending on gap width very

different amounts of tissue deformation can occur for the same amount of movement: the wider the gap the less tissue deformation and vice versa. The problem is that if a gap is very small and is moved by a similarly small amount there may still be high strain conditions (e.g.  $100/100 = 1/1 = 0.01/0.01$ ). Let's consider a very small fracture gap of 10  $\mu$ m that is about a cell width. If the fracture surfaces displace only 10  $\mu$ m the cell size is deformed to 10  $\mu$ m + 10  $\mu$ m which is a 100% deformation (Fig. 3). The problem is that the small gap is invisible to the naked eye and, likewise, the displacement cannot be detected. Still, 100% is an extreme deformation that can be tolerated by a cell of granulation tissue but not by connective tissue or cartilage or bone. Such conditions are not visible but they must be understood, that is, the function of the surgeon's eye is replaced by the function of his brain.

When a tissue (or any other material) is deformed beyond a certain limit disruption occurs. It goes without argument that a tissue cannot be formed when the strain exceeds its limit of elongation at rupture, in other words, conditions that would disrupt a tissue do not allow its formation. The critical parameter is elongation at rupture, which is the upper limit of accepted strain or the limit of strain tolerance. Similarly, tissue repair or differentiation is not induced below a certain limit.

Successful fracture healing occurs within the bandwidth between strain induction and strain tolerance (ref. Perren in 6/2014)<sup>7</sup>. Fig. 4 through Fig. 6 – illustrate the mechanical properties of different tissues that play a role in secondary fracture healing (15).

<sup>6</sup> We simplify for easier understanding of the basics. In reality strain is three dimensional and depends not only on mobility but also on fluid flow as outlined early on by E. Cheal.

<sup>7</sup> Perren, S. M.: Fracture healing: Fracture healing understood as the result of a fascinating cascade of physical and biological interactions. Part I. An attempt to integrate observations from 30 years AO Research. Acta Chir. orthop. Traum. čech., 81: 355–364, 2014.

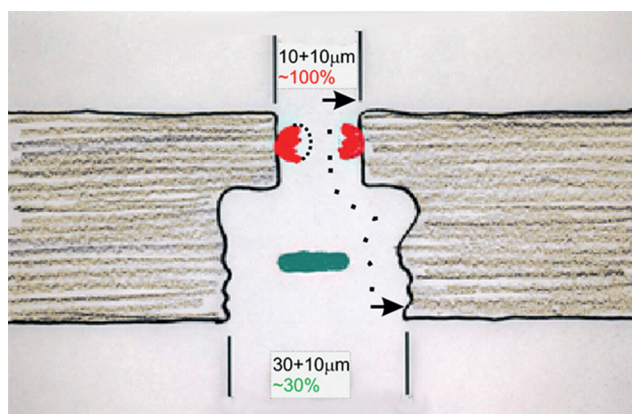


Fig. 3. **The effect of the width of the fracture gap on tissue deformation.** Assuming a relative movement of the two fracture surfaces of 10 µm, the cell in the smaller (10 µm) wide gap is disrupted (100% strain) while the cells in the larger (30 µm) wide gap are less deformed (~30%) and remain intact. The dotted line indicates the position of the fragment before displacement.

## STRAIN INDUCTION VS. STRAIN TOLERANCE

### Aspects of strain induction

Under conditions of spontaneous healing the limit of strain induction is often not an issue. In contrast, under conditions of flexible internal fixation maintaining strain above induction level is an issue. As an extreme example: when a large defect is spanned with two plates the large gap and the scarce mobility result in minimal strain that is below induction level. The clinical aspect is characterized by painless function ensured by the implants that do not help fracture healing but replace it. We call this “prosthetic osteosynthesis” where the internal fixation provides painless function but does not induce proper healing (12). Late bone formation filling the gap is then obviously not induced by biomechanical conditions such as strain. This is a type of bone formation that needs further attention.

### Aspect of strain tolerance

The upper limit of strain, i.e. tolerance, is elongation at rupture as mentioned above. The values for elongation at rupture for biological materials are listed in Yamada 1970. The value for the initial repair tissue, granulation tissue, is not reported there but the closest material to granulation tissue is parenchyma with a value of roughly 100%. The next stage of repair is bridging by connective tissue and, frequently, cartilage before final bridging by callus is rendered possible by reduction of strain due to widening of the gap, the generally increasing stiffness of the repair tissue and a change of dimensions due to the increasing diameter of the soft tissue cuff. An element that facilitates bridging is that the first bridges are arranged like a spring, thus reducing the strain within the spring element (Fig. 7). Furthermore, the stiffness of connective tissues, for example, dramatically increases with extension above a certain limit (Fig. 8). This contributes through increased stiffness to reduced mobility

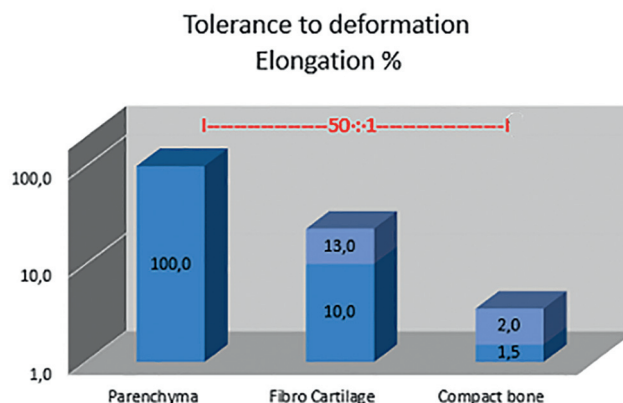


Fig. 4. **Tolerance to deformation for three selected repair tissues.** The data for parenchyma corresponds +/- to granulation tissue. From granulation tissue to bone the tolerated elongation at rupture is decreased by 50 times. The two different color shadings represent the different values. (data from Yamada).

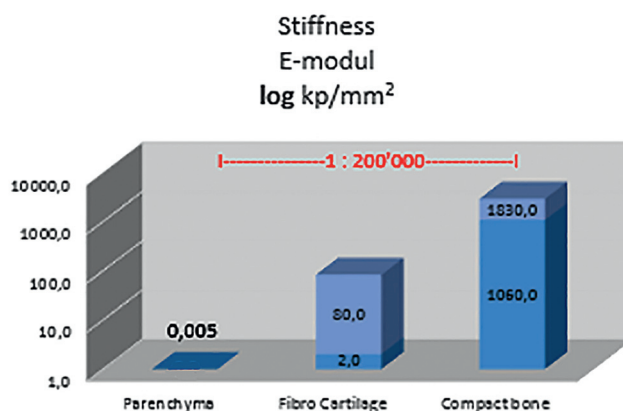


Fig. 5. **Stiffness for three selected repair tissues.** From granulation tissue to bone the stiffness increases by an enormous factor of  $2 \times 10^5$ . The data is plotted logarithmically. Increasing stiffness stabilizes the fracture. (data from Yamada).

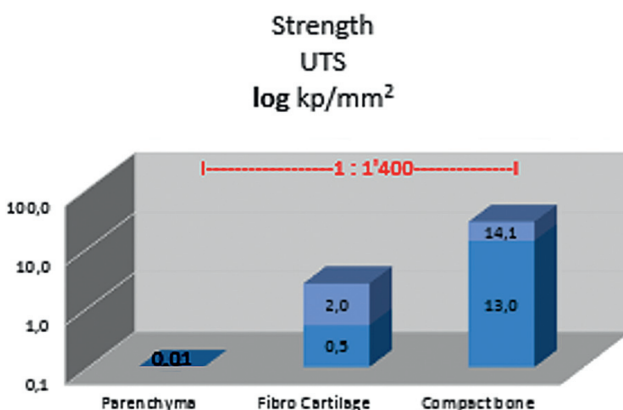


Fig. 6. **The strength for three selected repair tissues.** From granulation tissue to bone the stiffness increases by an enormous factor of 1400x. The data is plotted logarithmically. This strength plays a role in final fracture strength (data from Yamada)



allowing progression to the next step in differentiation. The tissues with higher stiffness are less tolerant to strain with cartilage at about 10% whereby it contributes by its moderate stiffness and strength. Finally, the bone

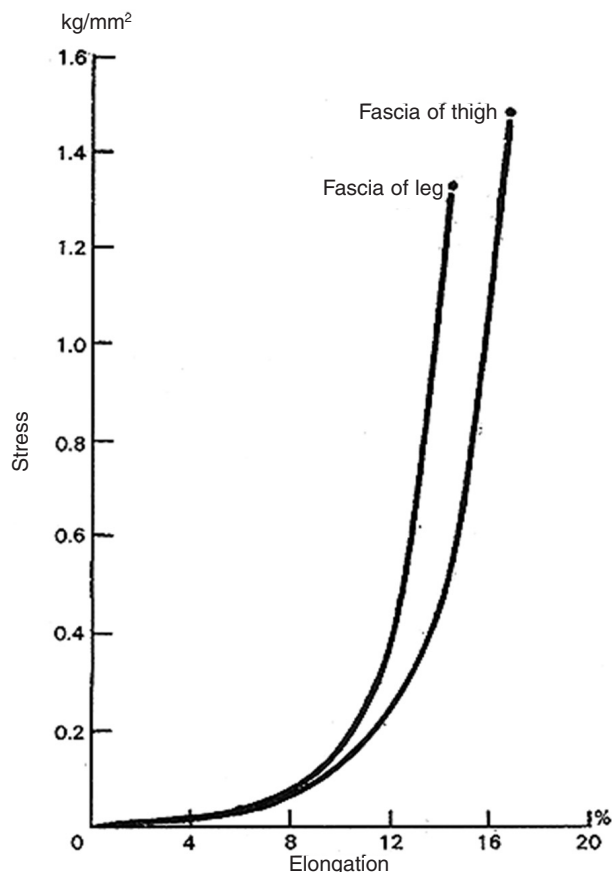


Fig. 7. **First tissue bridge crossing a moving gap.** The 3D arrangement of the bridge acts like a spring reducing the strain on the tissue element for a given overall strain (Perren JBJS 2002). The histological picture and a schematic of the spring are superimposed. The spring is able to take large overall deformation while its elements are subject to small deformation. This enables the woven bone (callus) to bridge gaps with a strain higher than the elongation at rupture of the repair tissue.

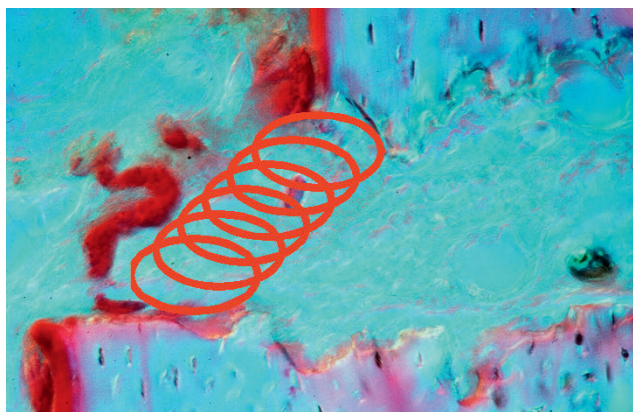


Fig. 8. **Nonlinear stress strain** behavior of some connective tissues. Here of a human fascia (Yamada). This allows the smooth transition of different repair tissues.

bridge has an extremely low elongation at rupture of 2% and provides high stiffness and strength. Spontaneous healing or healing under flexible fixation progresses from tissues that tolerate deformation well ("strain tolerant") but have low stiffness to strain intolerant brittle bone with high stiffness and strength.

Beside the changing material properties the dimensions play an important role. The increasing diameter of the callus increases the structural stiffness by the third power of the diameter so that doubling the diameter of the callus increases the structural stiffness by a factor of roughly eight times.

### REQUIRED STRAIN CONDITIONS FOR DIFFERENTIATION OF REPAIR TISSUE

Spontaneous healing starts with a condition of high mobility and a wide fracture gap. From granulation tissue to connective tissue to cartilage and bone the material stiffness increases, the diameter of callus increases with a marked increase of structural stiffness. Together with increasing gap width due to resorption strain decreases. The consequent reduction of strain allows differentiation in steps with final solid bone bridging. Initially, tolerance to deformation and later on increasing stiffness and strength are the critical mechanical parameters.

### THE CLINICAL OPTIONS FOR INSTALLING STRAIN CONDITIONS

To provide proper biomechanical conditions for healing strain must be within the bandwidth between the lower limit required for induction and the upper limit required for tolerance. As strain depends on the amount of movement and gap width several components can be adjusted by the surgeon. Let's start with movement. The amount of gap mobility depends on several elements: the most obvious is the amount of functional loading, the more load the more movement. Instructing the patient to control weight bearing may help but it is not a very reliable tool. The next element is the stiffness. The stiffer the splint, the less the gap moves. The critical parameter is the structural stiffness. For example, the structural stiffness of a plate bridging the gap depends on its dimensions, the material, and the free span between the innermost screws. The option of selecting a plate of a certain stiffness is elusive because of strength and shape requirements. The distance between the inner screws, that is, the free span determines the structural stiffness of the construct. This is an element that can be adjusted during the surgery. The final element in adjusting strain conditions is gap width which the surgeon can select during surgery.

### Adjusting strain during flexible internal fixation

How can the surgeon install proper strain conditions during the internal fixation procedure when the above mentioned mechanical elements prevail? He can make a fair guess at the amount of mobility to be expected:

weight, muscular condition and control thereof play a role. Adjusting the width of the fracture gap allows the surgeon to install strain within the required bandwidth: The greater the expected mobility, the larger the required gap. To adequately induce and allow fracture healing under conditions of flexible fixation for a given amount of expected mobility of the fracture, adjusting the gap size allows installation of proper strain but this is a demanding option and gap width should only be varied within a small range.

### Wrong simple rules regarding strain

The surgeon needs simple rules for his procedures. Attempts to achieve this have led to inadequate interpretation of strain. Keeping strain within the bandwidth discussed above requires adaptation of procedures. Therefore, it does not make sense to formulate a simple rule like “a small or a large gap is the goal”. Such statements are unfortunate and dangerous simplifications.

### INTERNAL FIXATION, BIOMECHANICS AND HEALING

The priority of internal fixation is to restore the function of bone, limb and patient. While the fracture produces a discontinuity of bone stiffness, internal fixation reinstates continuity that should allow early restoration of painless function. Restoration of function is a prerequisite for keeping the tissues healthy, for example, avoiding reflex dystrophy, which is a consequence of prolonged and extensive immobilization of a limb (ref. Perren in 1/2015)<sup>8</sup>

Internal fixation can install conditions where the fragments are kept in immobile compressed contact. Such contact does not allow relative displacement of the fracture fragments as would occur under conditions of usual functional load and strain is not an issue. Today our understanding is that the repair tissue does not react to the **presence** of a fully immobilized fracture (11). We have observed that osteons that cross fractured surfaces in close permanent contact do not as a rule change speed or shape or direction when crossing. The observation that bone necrosis due to implant contact with a damaged blood supply induces internal remodeling as kind of creeping substitution suggests that the internal remodeling of such an immobilized fracture is induced by bone necrosis as a consequence of trauma. The internal remodeling is not a response to the fracture but is induced by necrotic bone in the vicinity of a fracture. Therefore, we now consider primary healing as a mere side effect of the removal of necrotic bone tissue whereby the osteons plug the fracture like dowels and eventually, after some delay, provide strength (Fig. 9). This explains why fractures fixed with compression need to be protected for some time whereby the implants are generally not removed for two years. In contrast, Miclau et al. (8) and Tepic et al. (13) demonstrated solid reliable healing at 10 weeks when the locally elevated (undercut) plate permitted callus formation thus preventing an avascular gap end from acting as a notch immediately deep to the plate. When they

<sup>8</sup> Perren, S. M.: Fracture healing: Fracture healing understood as the result of a fascinating cascade of physical and biological interactions. Part II. Acta Chir. orthop. Traum. čech., 82: 13–21, 2015.

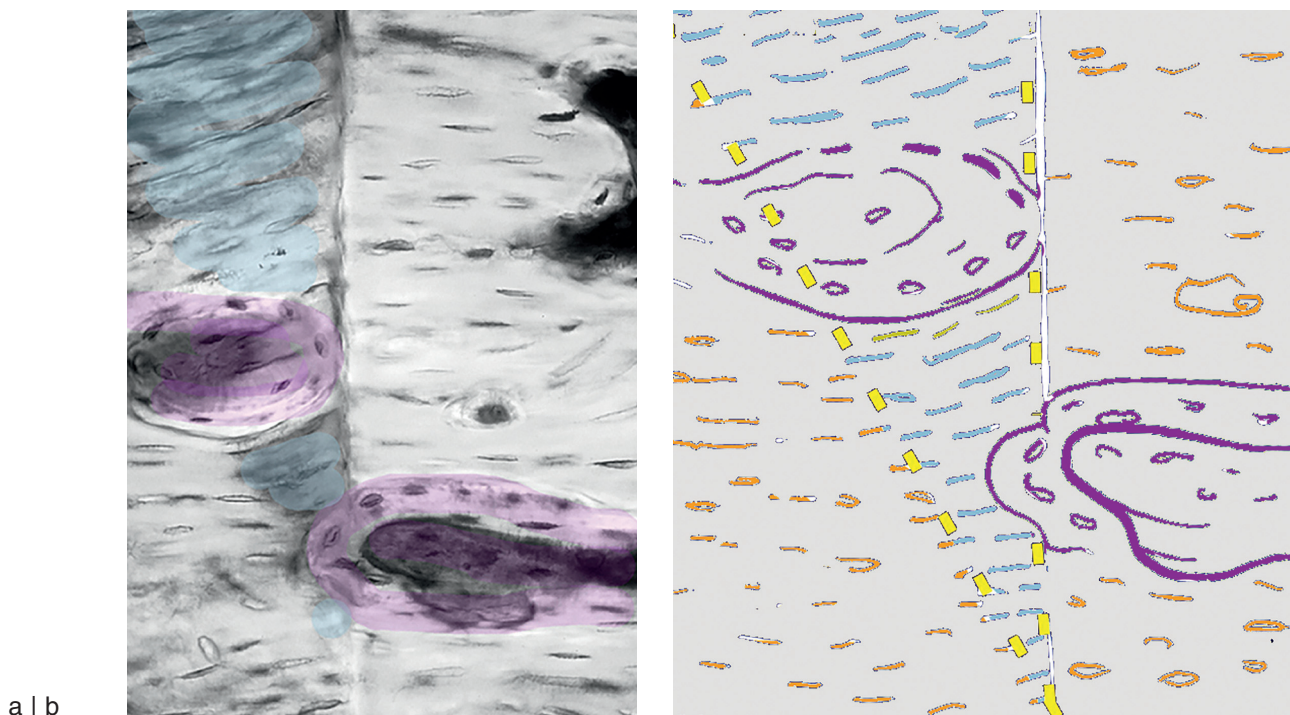


Fig. 9. **Osteons remodeling an area of stably fixed micro fractures.** The osteons remodel the multi-fracture area as if it were intact bone: A further hint that an immobilized fracture is not recognized in the absence of mobility. The internal remodeling removes dead bone which is produced by disruption of blood vessels. Fig. 9a LEFT: histology by B.A. Rahn, Fig. 9b RIGHT: schematic representation, the area within the dotted yellow lines contains the multiple micro fractures. Color coded histology.



subjected the fracture to bending testing after removal of the implants all failures were located within the fracture with the conventional DCP whereas all fractures withstood the load with the undercut LC-DCP and failure only occurred at the next screw hole. The latter condition is a prerequisite for implant removal.

#### Clinical aspects of strain – see also Fernandez (5)

1. The amount of strain within and around the fracture gap determines the amount of callus produced as is characteristic for spontaneous healing and healing under flexible fixation.
2. When strain between fragments exceeds the optimal bandwidth between stimulation and tolerance two distinct pattern of healing problems result:
3. Low strain conditions result in a pattern that is often mistaken for insufficient biological activity called “atrophic nonunion”. The observation that restoring proper strain conditions frequently results in prompt healing points to the biomechanical contribution to the problem.
4. High strain conditions result in abundant callus formation that cannot form a bridge and looks like hypertrophic nonunion where a reduction of fracture mobility and a simultaneous restoration of strain within optimal bandwidth results in prompt bridging (Fig. 10).
5. Dosage of optimal strain conditions depends on balancing fracture mobility and gap width. This is a task that is demanding in the individual case (Fig. 12).
6. The fact that fracture surfaces that do not exactly correspond<sup>9</sup> will experience different strain conditions which, consequently, increases tolerance to gap width adjustments.
7. Reduction of fracture mobility<sup>10</sup> needs to be weighed against additional surgical trauma to soft tissues and bone.
8. Compression fixation of a fracture prevents displacement of the fracture fragments in contact due to preloading<sup>11</sup> and/or friction. In such a situation, strain is absent in contact areas: no irritation and therefore no stimulation of healing.
9. Without biomechanical stimulation healing may proceed through plugging of the fracture surfaces by osteons acting like dowels.
10. Osteons with their cutter heads are most likely stimulated to remove dead bone in the vicinity of the fracture and will remove it, as has been observed, at the bone-to-implant interface<sup>12</sup> (Fig. 9) .
11. In areas adjacent to compressed contacts the strain is minimal resulting only from a small deformation of the contacting bone under intermittent functional load.

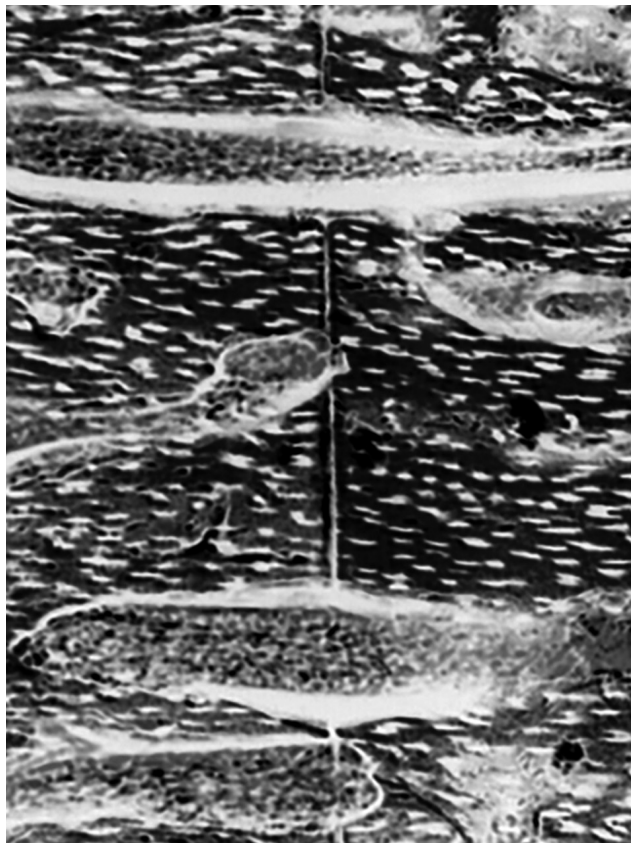


Fig. 10. *The osteons crossing an osteotomy that is held in closed contact do not appear to react to the mere presence of the fracture.*

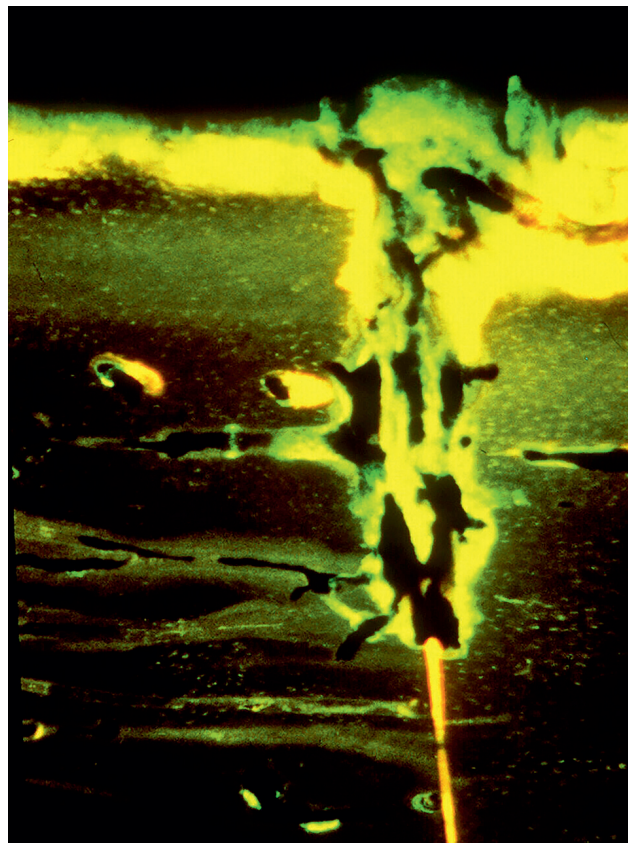


Fig. 11. *Tear drop phenomenon. At the outer end of the compressed gap under functional load intermittent gaping results. The reaction is widening of the gap by resorption and with it reducing strain. Reduction of strain then allows filling by callus.*



Fig. 12. **Optimal strain:** Flexible fixation of a spiral fracture. The width of the gap was properly adjusted. Solid secondary healing (C.Ryf).

## HOW DOES REPAIR TISSUE OVERCOME HIGH STRAIN CONDITIONS

The initial bridging of a fracture gap is usually based on callus. The English term of “woven” bone implies the understanding that callus is a 3D structure. In respect to strain such a structure acts like a spring. The spring is able to sustain large overall strain (overall deformation) while the elements of the spring sustain only very small deformation. The same can be observed for the first tiny bridges of bone crossing a gap (Fig. 7). The histological picture shows only the width of a histological cut but the elements shown here can easily be understood as being a cut through a spring. Callus is therefore able to bridge a gap that is under higher strain than the strain tolerance of the bone element, namely ~2% strain.

## ATROPHIC DELAYED OR NON-HEALING

Formerly, mainstream opinion was that a fracture would not heal if biology was not sufficient (atrophic pseudoarthroses). Today, based on the observation that atrophic non unions may heal after correction of the biomechanical conditions, the focus is more on installing the proper biomechanical conditions by reducing or increasing the stability of implants Fig. 13 through Fig. 15.

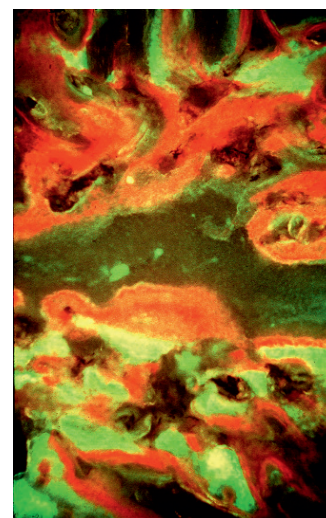
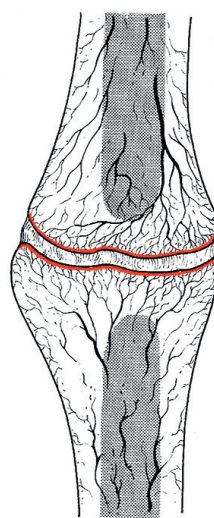
12. When compression applied to a fracture surface acts within a limited range the areas outside this range may undergo high strain conditions as a result of small displacement combined with very small gap widths or gaps intermittently closing. This results in the so-called tear drop phenomenon (“gocce di cere” Fig. 11).

<sup>9</sup> E.g. due to small displacement.

<sup>10</sup> Fracture mobility is relative movement of the opposing fracture surfaces in relation to each other.

<sup>11</sup> Preloading results from compressing the fracture surfaces against each other to prevent opening of the fracture gap when traction is applied as long as preload is greater than traction.

<sup>12</sup> Such biological activity by necessity starts in living bone adjacent to dead bone.



a b c d

Fig. 13. Hypertrophic non-union. Figs a–c from Weber and Cech (14). The treatment consists in reduction of mobility through splinting with a plate; a – abundant callus but no bridging; b – scintigraphy showing biological activity which has the potential but cannot bridge; c – elephant foot appearance; d – histology of a similar situation in sheep. From the fragment ends above and below callus advances decreasing the gap width and with it increasing strain.



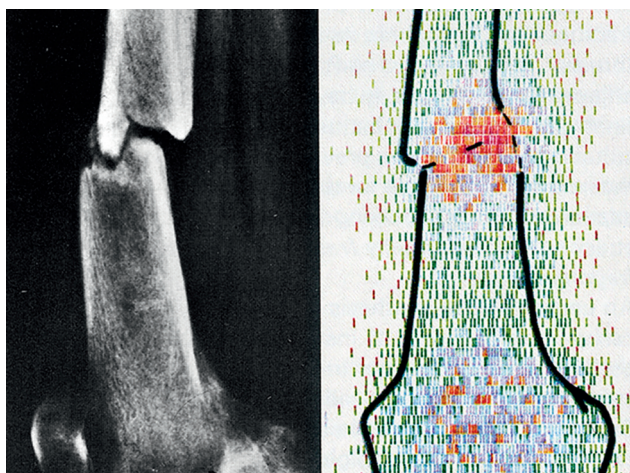


Fig. 14. Non-union that is biologically active but does not produce callus. Large gap, low strain without biomechanical induction. The treatment consists in application of axial compression that results in approximation and decrease of gap width increasing strain and enabling induction. Weber and Cech (14)

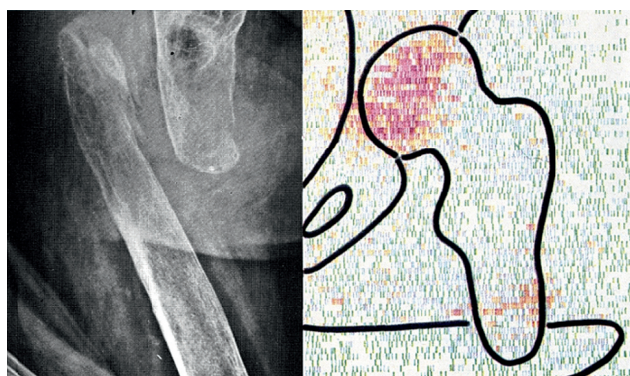


Fig. 15. Biologically inactive non-union, large gap width. Frustrated exhaustion and/or missing biomechanical induction? Weber and Cech (14).

## CANCELLOUS BONE AND CORTEX

The tolerance of bone to elongation applies similarly to cortical and cancellous bone as the elements of both bone structures are similar whereas resistance to load (stress) and resistance to deformation (stiffness) is very different for these two types of bone. The speed of reaction due to the accessibility of bone trabeculae is also an element to consider.

## MISUNDERSTANDING STRAIN

The way we express strain in respect to clinical application is deliberately as simple as possible. Declaring it as “oversimplified” misses the point that the basic change of thinking in terms of strain instead of stability is the essential and important issue. The argument that we show the deformation of a single cell in the fracture gap “where there are many cells” is the height of misunderstanding.

## CONCLUSIONS

Biomechanical conditions play an obvious role in respect to fracture healing. Fractures can solidly heal or not heal under a variety of biomechanical conditions. We propose that instead of fracture mobility consideration should rather be given to “tissue deformation” that is strain. Strain depends upon mobility and, more importantly, upon gap width and is first and foremost a more appropriate way of understanding the biomechanics of fracture healing and of improving treatment.

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