The Conscious – The Unconscious and Bone Structure

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Becoming aware of and understanding a phenomenon has to do with observation. This is the first step of the scientific process.

Aristotle

He who does not understand, and says so, is he who most shows intelligence, because he knows that he has not understood and that is the most difficult thing of all to grasp. Let us thank him, because he gives a gift to all of those around him who thought, wrongly, that they had understood.

Albert Jacquard

Abstract

The aim of this memo is to define a specific biological condition that would have its own psychological component, specific physical support and a scientific basis

We would like to propound the following assumption: in an older patient who suffers from bone structure complications due to osteoporotic disease, we think that there are two types of fear which persist after the bone appears to have healed. One is a conscious fear, which we will call the "scare-fear." The second is unconscious; we will call it the "reflex-fear."

If this assumption is correct, we should treat both of these factors at the same time: the fracture, which is the consequence of the osteoporotic disease, and the bone mass loss, which is the cause. This would reinforce the bone structure and prevent the patient's fear-reflex, which is caused by bone weakness. If the scare-fear persists, it should be treated by a psychiatrist.

Introduction

Everyone knows that a fracture is painful. The reasons for this have been identified by scientific research. The first explanation is that pain is a sensation that is experienced subjectively as unpleasant. The second depends on an objective scientific process (in that it has as its object to find the causes of the pain).

The factors of consolidation of the broken bone can be analyzed scientifically. They are observable facts. The subject is conscious of this consolidation. Once the bone is solid, it is no longer painful and can once again take on its physiological role.

One can ask oneself whether all patients are conscious of when their fracture is consolidated.

a) Pain is not a good criterion. On the one hand it is subjective, and on the other hand immediate immobilization of the broken bone is enough to make the site of the fracture painless even when it has just occurred and cannot yet have healed.

b) A return to normal function is not a good criterion either. Indeed, two phases in the evolution of the bone callus have been observed while the bone is not yet sufficiently solid to allow it to be used normally. However, some patients – but not all – say they feel something different around the sixth week after the break occurs.

Observation of older patients who have broken their hip indicates that even though the break is consolidated, some don't dare leave their homes out of "fear" of breaking the bone again if they fall.

The question this memo asks is the following: is there a simple explanation – purely subjective – to this "conscious fear" of breaking a bone, or is there another attendant fear that is hidden? In this case, what is this unconscious "other thing"? Is this unconscious "other thing" scientifically observable?

We would like to propound the following hypothesis. In an older patient with bone complications resulting from osteoporotic disease, we believe that such a behavior exists. Two types of fears would arise after the apparent healing of these complications:

- One is conscious, it is the "**scare-fear**." It is linked to the painful memory of the break or breaks. It is one of the factors which makes older people spend their time seated, in a restful environment, remembering good and bad times of their lives, sometimes with some nostalgia. They do not want to suffer once again, or to go through the same ordeal. We presuppose that they have recovered their pre-fracture autonomy.

- The other is unconscious, not perceptible, and therefore not analyzable by the subject. This is the "**reflex-fear**." This new way of living – somewhat apart from the world – is also the fear of falling again because of the fragility of the bones, which have not recovered all of their sturdiness.

To the conscious memory of pain caused by the fracture is added the unconscious fear of the risk of incurring another fracture because fragility of the bone. Can we demonstrate this scientifically?

DEMONSTRABLE SCIENTIFIC ELEMENTS OF BONE BIOLOGY

I – The four phases of bone consolidation

While it is true that "the consolidation of a fracture involves a succession of complex phenomena that remains shrouded in mystery", Mac Kibbin has shed some light on it by analyzing the four steps of bone consolidation (1).

II- The ductile and solid phases
Christel and Coll have noted an increase in the strength of the callus, which moves from the ductile phase to the rigid phase, around the fifth or sixth week (2).

III – The remineralization – reconstruction of the broken bone
The remineralization – reconstruction phase can last from 3 to 5 years. This process is identical to the process in a normal bone. The woven bone becomes a lamellar bone, or a haversian-type cortical bone. At the level of the cortex, because of mechanical constraints, the osteons are arranged along the main axis of the bone. We have noticed an alignment in columns of the grains of calcium carbonate during the cortical reconstruction of a broken femur affected by atrophic non-union. This phase occurs in the course of the third year (3). At the end of this local intracortical phase of the bone, bone reconstruction has finished. It coexists with the physiological remodeling.

Fig 1: At the level of the cortex, the spheres f biomaterials align themselves in parallel columns along the main axis of the bone. This arrangement is identical to that of the Haversian system (3).

IV – Bone remineralization
It will take place by changes inside the bone, where phases of destruction and phases of construction will repeatedly succeed each other, according to Frost's theory of the B.M.U. (4). This alternation, which allows the bone to maintain homeostasis, can be found in the remineralization process of people who have been subjected to weightlessness. The demineralization linked to weightlessness is nonetheless different from this permanent remodeling. The remineralization in a weightless environment can last from three to five years. (5)

THE SCIENTIFIC PHYSIOLOGICAL UNCONSCIOUS

a) Subliminal experiences

Below a certain threshold, a stimulus is not perceived even while it is felt and registered by our senses. Examples of this include subliminal images and sounds. One could call this the scientific physiological unconscious in that it relies on internal processes and circuits that have a physical basis (vision or hearing) that can be analyzed scientifically but which the conscious mind does not perceive. We can trace the evolution of our knowledge of these "little perceptions" from their postulation on a philosophical level to their demonstration on a scientific level.

b) Physiological osteopenia and pathological osteoporosis

Baldet has demonstrated the progressive loss of bone mass with age. This physiological osteopenia affects all subjects (6). Osteoporosis is defined as an abnormally high level of bone loss, both in quality and quantity. This pathological weakening of the skeleton would be linked to a decrease in the number and/or quality of osteoblasts while the

activity of the osteoclasts would not be changed. This weakening is not consciously perceived.

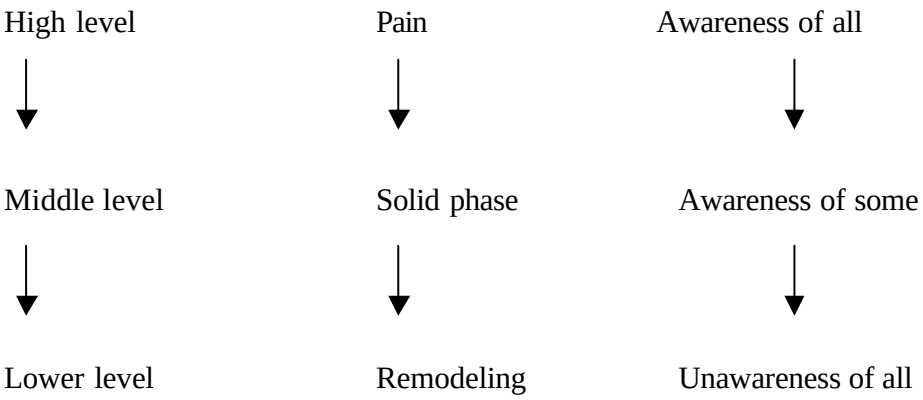
c) Osteoblasts and mechanical perception

There are indications of mechanical perception by the osteoblasts located at the surface of the trabecular bone. They are equipped with baroreceptors. The primary response to the variations in constraints involves cellular mechanoreceptors. This mechanoperception is not felt by most people.

CONSCIOUS AND UNCONSCIOUS BONE CONSOLIDATION

"Certain patients think they perceive a sudden improvement around the 5th or 6th week of orthopedic treatment." This corresponds to the above-mentioned solid phase (2). Not all patients perceive this improvement. It is therefore unknown to most people. Nonetheless, it does exist.

Isn't there between the fracture, the bone consolidation and the bone-remodeling phase a continuum of perception at different levels? One would therefore go from the awareness of the pain of the fracture, which is perceived by all, at the highest level; to the consciousness of the bone consolidation, which is perceived by only a few and unknown to most people, at a lower level; to the remodeling phase, which is unrecognized by all patients, at the lowest level.



BONE FRACTURE CONSCIOUSNESS AND UNCONSCIOUSNESS

I-The conscious "scare-fear" is the memory of the painful events experienced by patients at the moment of the fall. This remembered fear can become an obstacle to a return to autonomy, or at least to the same level of autonomy as before the trauma. Patients' behavior changes. Older patients no longer dare to leave their home for fear of breaking a bone again. This fear is common to all.

II- Calcium loss. The skeleton of older people who have broken a bone loses calcium not only at the site of the fracture but also throughout the entire skeleton:

a) Generalized physiological bone loss or osteopenia increases at the time of menopause. Most subjects are unaware of it.
b) Osteoporosis is directly responsible for the fracture. This bone fragility is unknown to people who are affected by it.
c) The bed rest that follows a break increases natural generalized bone loss. This phenomenon is identical to the global demineralization of the skeleton that can be observed following space travel. Both astronauts and people who are taking bed rest are unaware of this bone fragility.
d) At the level of the broken bone, bone calcium loss increases even more because of the minerals required for the construction of the bone callus. Most patients with broken bones are unaware of this additional bone loss.
e) Once the bone has consolidated, because of remodeling, remineralisation takes place slowly before the bone returns to its previous level of mineralization. All bones are subject to this reality. All subjects, whatever their age, are unaware of this modification of the demineralization.

When all of these losses are added up, the weakening of the bone is seen to be the result of five local and/or general factors: osteopenia, the fracture, osteoporosis, immobilization, and lastly remodeling.

fracture ----------	unconscious generalized demineralization
physical osteopenia ----------	unconscious generalized demineralization
osteoporosis----------	unconscious generalized demineralization
building of the callus----------	unconscious local demineralization
remodeling of the bone----------	unconscious general remineralisation

BIOPHYSIOLOGY OF THE BONE

A <u>Bone and central nervous system</u>

In his *rapport de maîtrise,* Corentin Barbu broadly examines the relationship between the central nervous system and bone homeostasis (7).

I- Pathways of nervous conduction that control bone homeostasy

After a long period of research made difficult by the structure of the bone itself, it was observed that the bone is rich in nervous fibers that are sensitive fibers and that come from the sympathetic system. Innervation is essentially located in the zones of bone remodeling – such as the bone callus – but also exists in long bone metaphysis. The osteoblasts and osteoclasts continuously secrete molecules, which would guide the growth of nervous fibers.

a) The sensory pathways responsible for proprioception and nociception are present in the bone. They appear to be important for the regulation of homeostasis of the bone (19).
b) The vegetative pathway – whether it is the orthosympathetic pathway or the parasympathetic pathway – can interact with the bone. All of the

pathways, afferent as well as efferent, pass through the spinal cord at different levels.

II- Chemical mediators of bone homeostasis

The sensory nervous system, the motor nervous system and the sympathetic nervous system are involved in the regulation of bone homeostasis through the mechanism of many chemical mediators.

a) The perception of pressure and more generally of mechanical constraint would appear to be dependant on the sensitory system (8-9-10-11-12). Serotonin appears to be particularly involved in the perception of pressure (13).
b) The sexual hormones secreted by the hypothalamus play a large role in the regulation of bone mass as is demonstrated by the relation between menopause and bone mineral density. The end of the secretion of estrogen leads to a sudden fall in bone mineralization.
c) Leptin is involved in bone homeostasis through the central nervous system. The hypothalamus plays a determining role in this mechanism. The antiosteogenic signal is transferred to the skeleton by the sympathetic system. (14).
d) Adrenaline and noradrenaline, the principal mediators of the sympathetic system, have an antiosteogenic effect. Neuropeptides that are present in the bone act at the level of the receptors located at the surface of bone cells. Catecholaminic endings are observed near the bone cells (15). Noradrenaline leads to an increase in osteogenic activity because of its effect on the increase in osteoblasts. A complete picture of the action of catecholamines is still evolving (16). However it is accepted that their action is globally antiosteogenic.
e) Chemical (17-18) or mechanical sympathectomies all provoke a resorption of bone mass. These experiments are to be compared with numerous clinical observations in man such as the section of sciatic nerves or of the spinal cord. Muscular traction models the morphology of the bone. The bone of a person suffering from polio is thinner, the faces of the diaphysis are smooth and circumferential, lacking in the protuberances, crests or lines that are usually found on an adult bone. This might explain why the consequences of these denervations are somewhat contradictory depending on the authors. However, most of the studies favor the hypothesis of the increase of resorption.
f) Glutamate is present both in the osteoblasts and in the nerve endings where they connect to the bone cells. It is found near the bone marrow fibers, blood vessels and bone cells. Some of the endings are in contact with the hematopoietic cells and the osteoclasts. The osteoblasts also contain varying quantities of glutamate. Glutamate seems to have a positive effect on bone mineralization. These phenomena are generally unknown to the subject.

Antiosteogenic mediators	Osteogenic mediators
Leptin	Glutamate
Catecholamine	Estrogen

III Regulation of bone homeostasis

The regulation of bone mass is dependent on the central nervous system (hypophyso-hypothalamic) and the sympathetic system. These are the ascending pathways of deep unconscious sensitivity.

a) *The bone system* plays a role in the "in situ" behavior of its component cells. Deep unconscious sensitivity would begin at the level of the bone, by mechanisms that are still poorly understood. This regulation would take place through the mediation of mechanoreceptors stimulated by mechanical factors (pressure, stretching, etc…) acting on the bone cells.
b) *The spinal cord*: Through the neurons of the sympathetic and parasympathetic vegetative systems, information would reach the spinal cord by going through the cells of the spinal ganglia and would reach the posterior cords by traveling through the sensitive roots of the rachidian nerves. They then reach Clarke's column along the medial part of the posterior horn of the spinal cord.
c) *The metencephalon*: From there, they go directly to the cerebellum (the center of balance and coordination of movement) by means of Flechsig's fasciculus.
d) *The diencephalon or intermediary brain*: After a passage in the olivary nucleus, they intertwine as they reach the thalamus. They contribute to the formation of Wernekink's commissure.
e) *The telencephalus*: They may be continued by thalamocortical neurons.

Most of the pathways of conduction go through the *cerebellum*, and end in the mid-brain. Except in the case of a lesion to the cerebellum, we have no perception of a problem of balance, if all else is normal.

HYPOTHESIS

One can conceive and admit that the "conscious fear" of breaking a bone again, to which is added the memory of the pain experienced at the time of the fall can cause the change in the relational behavior that follows the accident. Indeed, many older people no longer dare leave their homes or only do so when it is absolutely necessary (for example to go grocery shopping). They often ask someone to accompany them. Some use a crutch or a cane.

One can nonetheless wonder whether this conscious and subjective state, this new behavior, is the result of only this factor. We have emphasised the great number of mechanical and chemical factors which result in a decrease in bone mass both globally and locally. We have noted that this demineralization is particularly great in a fractured osteoporotic bone. Fear or pain lead to a sudden secretion of adrenalin. As we have seen,

the stimulation of adrenalin has a globally antiosteogenic action. All stress-inducing situations can – at least in theory – have an additional effect on the bone mass (in the sense of decreasing it).

The consequence of this depletion of the bone mass is an increased global weakening of the skeleton. When we look at a crystal glass, we are aware of the fragility of the object. When we want to sit on a Louis XV chair, we are aware of the fragility of the piece of furniture. And it is because we are afraid to break them that we are careful in what we do. We are subjectively conscious of their fragility when we want to use them.

However, *we are no more conscious*, subjectively or objectively (aside from specialists) of the demineralization of our bones, be they intact or broken, *than we are conscious* of their fragility (subjectively or objectively).

We put forth the hypothesis that the "conscious subjective fear" does not alone explain the behavioral changes in older people once their fracture has consolidated.

We put forth the hypothesis that there is what we will call an unconscious "reflex fear," that is related to the mechanical fragility of all of the bones that compose the skeleton.

This "mechanical reflex fear" would have as its origin bone cells whose mechanical stimuli would follow the pathways of deep unconscious sensitivity and whose fibers would end in the cerebellum or the thalamus. This perception would therefore not go beyond the diencephalus. It would be unconscious because not perceived.

As the principal hormone of our (conscious) emotions, adrenalin could also play a role in maintaining this pathological situation because of its predominant demineralizing action (unconscious because not perceivable).

INDICATIONS AND THERAPEUTIC PROPOSALS

Our intention is not to discuss here the indications or the role of current medical therapy in the prevention of osteoporosis.

The logical consequence of this analysis - if it is correct and proves true - is that we should not neglect either of these two factors in the treatment before and at the time of a fracture. The mechanical structure, which was *unbalanced* before the break, and remains so long after it has healed must be reinforced. One must not, therefore, focus only on the neurotic aspect that may appear after the apparent consolidation of the bone. What solutions can we consider, knowing they must be simple, have no side effects, be easy to implement and take effect quickly?

1. Medication that reduces the secretion of adrenalin could be used. It could – in theory at least – play a role by diminishing or inhibiting the demineralizing effect of the neuropeptide. However, it is important to know the secondary effects linked

to the inhibition of the entrance of adrenalin into the sympathetic fiber, or to the decrease of the sympathetic tone.
Here we enter the realm of therapeutic risk.

2. Therapies that reduce the activity of osteoclasts, or recently discovered therapies that would act on the osteoblasts "in situ," raise more questions than they answer.
    a) Indeed, in the course of the aging of the bone we note an adipose metaplasia of the bone marrow and a disappearance of metaphyseal vascularization. Do the afore-mentioned medications act at the level of the adipocytes, and how?
    b) The second question is a pragmatic one: How are these medications carried to the heart of the devascularized bone when the mineral support has disappeared?
    c) Finally, and secondarily, how can a medication act at the same time on structures that are subject to mechanical compression on one side of the bone while the other side of the bone is subject to tension?
    We are here in the realm of supposition and uncertainty. We are not even in the realm of hypothesis and theory, since these questions have not been dealt with to date – at least to our knowledge.

3. Surgical insertion "in situ" of mineral material in the form of bone substitute such as the calcium carbonate contained in natural coral (over 98%) contributes to the strengthening of the bone, which can be verified by bone densitometry. It only takes an increase of 20% above the fracture threshold to protect the bone from a break linked to a simple fall. This technique is simple, it has no side effects (20), and it is technically easy and effective quickly.
We are at the heart of concrete fact, of the target matter and of biocompatibility.

4. The last consequence is that, if a "neurotic conscious fear" persists after the remineralization of the bone has been proven, it will be the role of a psychiatrist or psychologist to do the necessary work to treat the residual pathological symptom which has been identified since it is conscious.
We are now in the realm of the spirit-concept and uncertain therapy.

CONCLUSION

This "unconscious reflex fear" would be similar to the "little perceptions." It would highlight and strengthen the close links between spirit and matter. It would remain hidden at the level of the bone structure without our being conscious of it, carried by the deep sensitive nervous system to the primitive brain.

This pathological "reflex fear" would find its origin in the unconscious but not in disturbances linked to sex. More precisely, it would be built on a profound imbalance of the weakened mechanical support of our body.

This reflex fear should disappear as soon as the bone recovers its mechanical properties of sturdiness while walking and resistance to shock. It is the aim of the bone substitute inserted by direct surgery to contribute to the decrease, even the elimination of bone fragility as well as of the fear reflex (20).

Unlike an irrational conviction, this unconscious "reflex fear" can be explained because it has a scientific basis. This unconscious "reflex fear" originates in an analyzable structure. It changes and becomes clearer with each new discovery, which is in accordance with Popper's definition. It could contribute to maintaining the patient in an unconscious pathological state.

For the older person, what does time mean? Outside of the past which he or she remembers, what is the length of his or her future? The present does not count, if it has ever existed – in fact it does not exist. One must therefore take care of the future of the person who is almost at the end of his or her life – as quickly as possible – in order to give to erect man the ability to remain erect. Time is of the essence. Let us hurry.

Paris 8 February 2007.

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