

A tsunami is hitting oral implantology

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Peri-implantitis is characterised by inflammation and bone resorption. Reducing the inflammation is important; it is a prerequisite, the first step, but not the therapy of bone metabolism. If the bone metabolism is negative, this is reflected in bone resorption, which ultimately leads to implant loss.

It is not the inflammation that is the problem, but the negative bone metabolism. In peri-implantitis therapy, it is not enough to treat the inflammation—it is the bone metabolism that is causing the patient to lose the implant.

Bone resorption results from too many activated osteoclasts. There are no microorganisms that break down bone. Even if the bone is in the ground for 100 years, microorganisms will not break it down. These are different causes. Microorganisms cause inflammation, and too many activated osteoclasts cause bone resorption. Different causes require different treatments.

We need to learn how to treat bone metabolism. If more bone is broken down than built up, the bone metabolism is negative. Depending on how negative the bone metabolism balance is, the patient will lose the implant quickly or over many years.

The activity, quantity and function of osteoblasts/osteoclasts can be influenced therapeutically. For example, osteoclasts can be reversibly inactivated, regardless of how they were activated.

There are many options for the treatment of periodontal inflammation. Bone metabolism therapy, on the other hand, is less known and even less practised. Previous attempts to treat peri-implantitis have been based on reducing the inflammation. Patients with peri-implantitis resorb more bone than they rebuild. The obligatory balance in bone remodelling is shifted towards degradation. Bone is effectively type I collagen, and the collagen metabolism can be measured digitally

using the aMMP-8 test, with levels below 10 ng/ml considered normal and values up to 20 ng/ml representing the upper tolerance range. Any level above 20 ng/ml indicates excessive collagen degradation and the need for treatment. However, negative bone metabolism can also be diagnosed by careful observation of the patient.¹¹

Implants are foreign bodies. This means that a subliminal foreign body reaction will occur.⁵ Unlike teeth, implants are firmly anchored in the bone, have no intrinsic mobility and depend on a balanced bone metabolism.

There are many causes that can lead to the additional activation of osteoclasts and thus to increased bone loss. The ageing process begins after the age of 35. This is a fundamental problem for implant patients, who are usually older than 35. As the patient ages, everything becomes less: less hair, less smooth skin, less muscle, less new bone formation. The decrease in new bone formation only gives the appearance that bone resorption predominates. In fact, however, the formation of new bone decreases. People age, and just as everything slows down with age, new bone formation also slows down.

Bone quality deteriorates, and the bone loses stability due to insufficient osteoblast activity, and osteoclast activity appears to increase proportionally. In addition, there is a decrease in mineralisation, which has a major impact on bone stability. Masticatory forces result in rotational forces on the implant. The axis of rotation is located in the centre of the implant, and the maximum deflection and load is

in the marginal area, which shows peri-implant crestal bone loss, the beginning of pocket formation. The deepening pockets change the milieu, and thus the microbial composition, from supragingival, aerobic, regenerative to subgingival, anaerobic, pathogenic.

Where the implant emerges into the oral cavity, a tissue area is formed that is similar in structure to the corresponding area on the tooth.¹² Herman et al. give

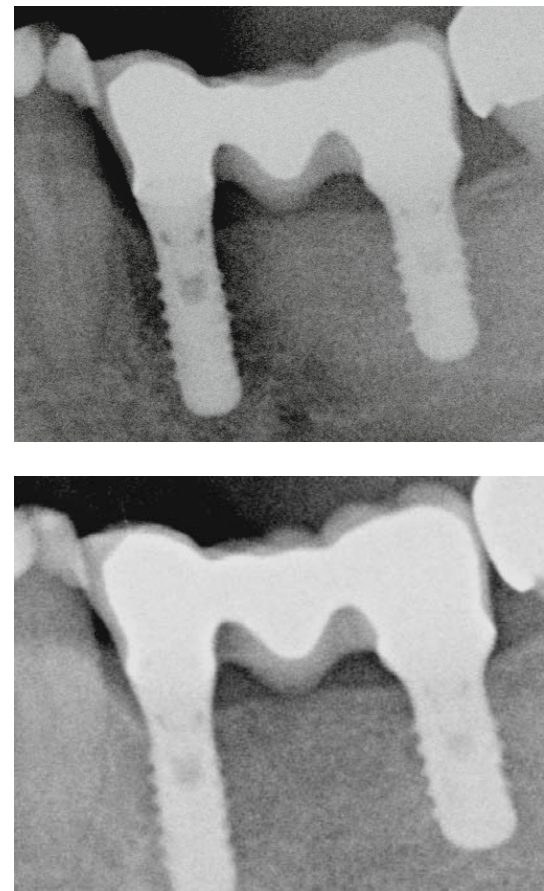


Fig. 1: Before starting therapy. **Fig. 2:** Completion of therapy after nine months.

average values of 3.0 mm for the biological width at the implant.⁴ Teeth are different from implants. The tooth is connected to the alveolar socket and neighbouring teeth by a fibrous apparatus through connective-tissue attachment structures.¹⁴ On the implant, on the other hand, there is only adhesion via hemidesmosomes.³ However, this connection osteogenesis would already be the maximum success; usually only contact osteogenesis is achieved.¹³

The gingival pocket is protected by the constant flow of sulcular fluid. The gingival sulcular fluid is a serum transudate and exudate. In a 5 mm pocket, it is replaced approximately 40 times per hour.⁷ The implant has no sulcular fluid flow. The saliva is stationary; it is not moved or replaced. In a vase of flowers, if the water has been standing too long, it becomes putrid. The same applies to implants. The implant sits in a stagnant, putrid liquid. Sulcular fluid is a reliable indicator for the diagnosis of peri-implantitis.^{1,2}

Prof. Antoine Béchamp (1816–1908) once said: “The microbe is nothing, the environment is everything.” In order to achieve lasting therapeutic success, we need to change the milieu, the environmental conditions for the microorganisms, and also induce the regenerative microorganisms to proliferate.

Peri-implant bone metabolism cannot be assessed by examining the oral cavity. Radiographs also show no evidence of negative bone metabolism in the early stages.

This is where the aMMP-8 test comes in. aMMP-8 is currently the only clinical parameter that indicates collagen degradation even before it has started. This means that we can start treatment even though there are no clinical findings. In this treatment phase, only bone remodelling therapy is required. What is being treated is a negative bone metabolism that has not yet occurred—*restitutio ad integrum*.

All those microbial tests do not help us. Only when bone resorption is already in full swing do changes in microbial composition occur, accompanied by horizontal bone loss. The therapeutic success when

Paradigm shift in oral implantology

- Inflammation is triggered by pathogens
- Bone resorption is triggered by osteoclasts

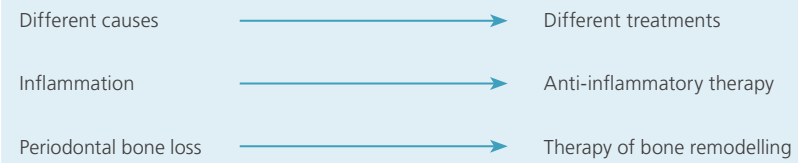


Fig. 3: Paradigm shift in oral implantology.

treating bone metabolism is now *restitutio cum defectu*.

There is only one bone metabolism, not one for the jaw, one for the spine, one for the knee, etc. So peri-implantitis is just the dental term for an overall negative bone metabolism. There are many causes of negative bone metabolism. The main causes are lack of exercise, oxygen and fluids, effective microorganisms, too many high carbs, deficiencies of vitamin D3, vitamin K2, vitamin A, calcium, iodine, magnesium, etc.—In short, a lack of physiological bone load, untrained lungs, fluid deficits and inadequate intake of vital substances.

It would be a dream to be able to change this, but it would require patients to make drastic lifestyle changes. Experience shows that patients need to have serious general signs of illness before they are willing to comply here. There will be some patients

who do comply, and it makes sense to get them on the right track.

The whole treatment will only work if the dentist has a good understanding of systemic bone metabolism. Half-truths and haphazardly selected measures will not bring the desired success. Bone metabolism is extremely important. Bone has more than just a holding and supporting function. Calcium metabolism is directly linked to bone metabolism and therefore influences almost all life processes, because every cell, every muscle, every brain cell needs calcium to function. Calcium is the most abundant mineral, and this is where and why many mistakes are made. In today's industrialised lifestyle, calcium intake is reduced and we struggle with the effects of the calcium paradox.

We have too much calcium where we do not need it (soft tissues, organs, blood

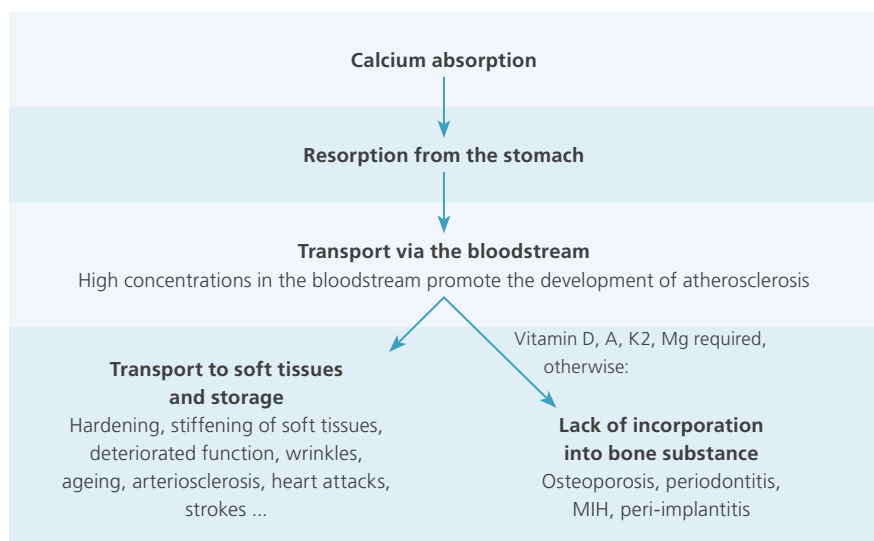


Fig. 4: Calcium intake: the calcium paradox.

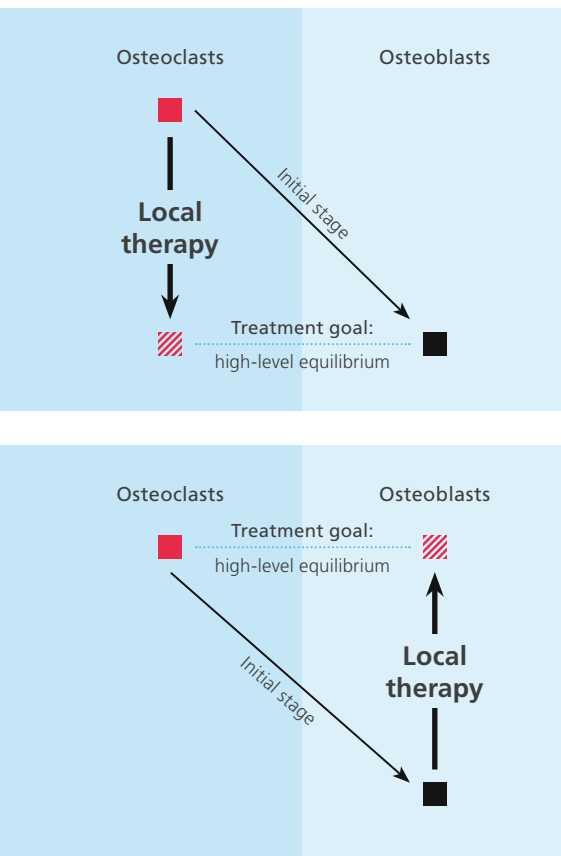


Fig. 5: Difference between the local and systemic therapy of bone metabolism.

vessels, brain) and too little where we do need it (teeth, bones = MIH, osteoporosis).

Bone also has other functions. Blood cells only live for 120 days and then have to be replaced by a new one. They are produced by the red bone marrow, as are immune cells, tumour killer cells and many others that depend on a functioning bone metabolism.

Since this knowledge is not part of the dentist's basic training, I recommend that interested colleagues attend special training courses, e.g. those offered by the Saxony State Dental Association.

Implants—defining the problem

1. Local therapy of bone metabolism—symptomatic

The excess activated osteoclasts are inactivated, and osteoclasts activity is slowed down to match the insufficient number of activated osteoblasts. Now the amount of new bone formation is equal to the

amount of bone resorption, albeit at a low level.

2. Systemic therapy of bone metabolism—cause-related

Osteoclasts are not the problem in negative bone metabolism. It is not that too much bone tissue is suddenly degraded. Rather, the structure and mineralisation of the bone do not meet the requirements, so it only appears as if excessive bone resorption were the cause. Systemic therapy activates osteoblasts, stimulates new bone formation and mineralisation, and creates a balance between bone resorption and replacement at a very high level.

Therapy of peri-implantitis

During therapy, the connective tissue collar around the implant is pulled very tightly, so that the pocket disappears completely and no putrid saliva lakes can form around the implant. In parallel, osteoclasts are reversibly inactivated, osteoblasts are activated, and calcium metabolism/transport/storage is activated. The maximum time for bone maturation is nine months. The therapy has been described in the *Dental Barometer*.⁸⁻¹⁰

Unfortunately, few dental practices are technically equipped to treat peri-implantitis when considering how to effectively manage biofilm on implants:

1. Using ultrasound or sonic systems to shake implants with reduced osseointegration and applying vertical and rotational forces?—Definitely NOT!
2. Hand instruments, perhaps with the screw thread exposed?—How would that work? (Koch)
3. Supragingival powder jets penetrate the pocket a maximum of 2 mm?—Insufficient.
4. Rotating instruments?—Unfavourable.

Summary

It is less important to explain a peri-implantitis treatment that has worked for almost 30 years while the underlying

therapeutic approach is unknown. The reduction of inflammation and bone resorption are different processes. At present, only inflammations are generally treated. These are triggered by microorganisms. There are no microorganisms that break down bone. The faster and more effective the inflammation treatment, the further bone resorption slips into the negative range, e.g. microorganisms are killed by antibiotics. The dead microorganisms are removed by the body's own scavenger cells. In order for the large phagocytes to get to the site, they push along high levels of aMMP-8 to break down collagen.

A healthy clinical situation emerges, but bone metabolism slips further into negative territory.

References



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