Successful peri-implantitis prophylaxis

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During the last decades, implantology emerged as one of the most innovative enrichments in the field of dentistry. Considerable increase is expected in the future. Compared to earlier pre-prosthetic methods, endosseous implantology is a simple treatment that usually is not very stressful for the patients and offers many advantages, eg the physiological transfer of chewing forces into the bone, which - under certain conditions - even generates renewed bone growth.

Against this background and since implantology with all its prosthetic treatments varieties is considered an established method.

One of the most common and most feared complications occurring in implantology is peri-implantitis (Fig 1), which usually leads to implant loss in case it remains untreated.

Introduction
Initially, the peri-implant tissue disease manifests itself as mucositis with progressive bone loss at the implant area, as described by ALBREKTSSON et al. The reasons for this disease pattern are complex, and various hypotheses about the development of peri-implantitis were proposed, amongst them insufficient oral hygiene, lack of fixed gingiva, and/or overstressed implants. These putative triggering factors contradict the statements of well-known implantologists. An absence or insufficient width of keratinised gingiva is not aetiologically linked to the development of gingivitis and peri-implantitis or the functional strain placed on an implant cannot be solely held responsible for progressive bone loss. That means that additional pathologic influences, which trigger and sustain the process of disease, must exist next to these osensible causes.

Therapies reach from improved basic hygiene to antibiotics and disinfectant inserts into peri-implantal pockets up to ultrasound treatments and laser curettage of inflamed tissues. The main attention, however, should not be placed on therapy, but rather onto an efficient prevention of peri-implantitis.

Reflecting on gaps and hollow spaces of assembled implants It’s a fact assembled implants contain hollow spaces, which can be minimised but not prevented even at the most meticulous production. Because also threads hold gaps, the contamination of implant interiors with germs originating from the oral cavity is inevitable (Fig 2).

The re-infection from an implant cannot be ruled out. On almost every assembled implant we noticed a putrid smell of its content, which was extracted with a cotton tip. In 1996 we initiated the examinations after that confirmed the assumption that gaps and hollow spaces in the interior implants were contaminated with germs, which matched the germ spectrum of an interdental smear. Implant interiors in their dimensions, position and size are easily recognised by construction drawings, cross-sectional shapes and X-rays, and so it becomes clear that hardly any assembled implant is actually excluded from those facts.

Of course, these considera-
tions apply to screwed superstructures as well. Cemented superstructures seem to be sealed at first by the fastening cement, but everyone knows the smell that emerges when cement is drilled from crown and bridge work and gives evidence of germs permeating here as well.

The access paths of germs into the implant interior are easily comprehensible, and we were able to provide evidence by taking light- and electron microscopic exposures of a used implant (Fig 5).

The paper of BINON et al. Implant Component Compatibility, confirms this matter quite impressively. The results showed that the macroscopically good fit revealed severe flaws under electron microscopic examination.

Furthermore, the capillary forces and micro motions between the implant and the abutment in addition promote the exchange of infectious material, wherein the saliva is a good vehicle.

Figure 4 shows the proportion of the gap located between implant and abutment compared to an erythrocyte. In order to make the dimensions even more clearly, the randomly chosen germs shown are also matched to an erythrocyte exact to scale.

Development of peri-implantitis through re-infection from an implant
The implant gets contaminated with germs from the oral cavity as soon as it is opened for placement of the insertion tool. Germ growth starts immediately after fastening the locking screw, unless the implant interiors were previously treated with a material to seal and combat germs.

The breeding conditions - warmth, humidity and supply - enable bacterial growth and fungal colonisation in an ideal manner, so that a re-infection of peri-implant tissues via the outward leading gaps is given. Whatever treatment of this important area around the implant is applied, it will always remain short-lived.

Development and efficacy of Gap-Seal®
In order to counteract these re-infections we developed a material based on a highly viscous silicone matrix that seals the implant and protects it from bacterial or fungal penetration effectively.

Any antibiotic would not be sufficiently intensive and effective in such low doses, and would moreover contribute to sensitisation and the development of resistance. Afterwards we employed the so-called split-mouth technique to test the material against white Vaseline, and determined the required admixture of disinfectant.

The bactericidal and fungicidal properties and efficacy against viruses owes the sealing due to the principle: Where already something is, there nothing else can enter. If the medium does not offer a breeding ground, then nothing can grow.

The material met its purpose as gap and interior sealant more than satisfactorily and was subsequently named Gap-Seal® (Fig 5).

For the split-mouth studies GapSeal® was applied to the right sides of the implants, and Vaseline to the left sides. During this clinical comparability the Vaseline turned out to be thoroughly contaminated, while GapSeal® treated implants usually provided no evidence of germ growth. This is clearly proven by the follow-up examinations, which were conducted each six months afterwards.

The number of germs (CFU = colony forming unit) at each pertaining implant was determined through serial dilution, followed by counting the CFUs on the incubation plates. This process enabled a definite determination of germs contained in each interior implant smear. We were able to prove the material's efficacy by conducting follow-up examinations between 1996 and 2000 and do not want to abstain from using GapSeal® ever since (Fig 6). These studies finally showed a statistically significant reduction in peri-implantitis by more than a third of implants sealed with GapSeal®.

Application
It provides an opportunity to seal implant interiors with GapSeal® immediately after inserting and removing the insertion tool thereby eliminating the prospective peri-implantitis inducing the re-infection factor.

For this purpose the carbules are inserted into the applicator at first, and the closing cap needs to be removed. It is recommended to bend the cannula slightly around the applicator shaft according to the filling situation. Excess material gushing from the implant when the closure cap is screwed in indicates a good filling situation (Fig 7).

The material will be delivered in sterile blister packs; the applicator is autoclavable to warrant sterility. In case the implant is treated with GapSeal® at a later point, a thorough cleansing of the interior spaces with alcohol is recommended. Furthermore it is advised to fill the hollow spaces of screwed superstructures with Gap-Seal® too. During implant re-entry at recalls it is advisable to renew old material, which may be rinsed out with xylocaine. GapSeals® very stable, retains its qualities in case of cemented works over years, and requires neither exchange nor replenishment.

Results and discussion
Peri-implantitis is the most feared complication occurring in implantology, especially once the implant therapy with its appropriate prosthetics is completed. Suggestions regarding the treatment exist in ample variations and are put into practice as well.

However, it seems to be more reasonable to prevent the causes for peri-implantitis, which certainly originate to a large percentage from re-infection out of implant gaps and hollow spaces. The possibility of germ colonization on implant interiors exists and should be taken seriously. Attempts to combat re-infection are described in specialized literature since years.

Now GapSeal® with its 16 years of clinical experience offers a truly effective prevention against peri-implantitis.

About the author