# Er:YAG-Laser—The key factor in the therapy of periimplant inflammations

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[PICTURE: ©2JENN]

## _Introduction

Today, dental implants are widely accepted by patients and are seen as a desired therapy for the restoration of partly or wholly edentulous jaws. As a result, this therapy option is applied more and more often in the daily practice. It follows that the number of periimplantitis cases, an infection of the periimplant tissue, increases. Thus, periimplant inflammations will become more and more important for dentists in the future.

The prerequisite for the development of products for the prevention and therapy of periimplant disease is of course a sound knowledge of its aetiology, pathogenesis and epidemiology. The majority of early clinical studies used to judge the quality of treatment results by survival rates, with the implants remaining physically in the oral cavity. In the beginning, authors saw mechanical incidences as the reason for implant loss rather than biological causes. Today, the health status of periimplant tissues has become a focal point for implant survival. Although implant treatment is perceived to be generally successful, periimplant infections occur frequently. These are called periimplant mucositis or periimplantitis. Much like periodontal diseases, periimplant diseases are of an infectious origin and can ultimately lead to the loss of the bone supporting the implant.

In periimplant mucositis, the inflammation is by definition restricted to the periimplant mucosa, while periimplantitis also includes the periimplant bone. For positive long-term results of implants as well as for the prevention and treatment of oral infections, these diseases must be monitored.

The available epidemiological data suggest that one in five patients will develop periimplantitis sooner or later, and that, in general, periimplant mucositis often occurs in implant patients. Currently, only limited data about the treatment of periimplant diseases are available. Most of the procedures are oriented towards periodontitis therapy. The most important therapy aim is infection control. This can include the adjustment of dentures, if their form impairs an adequate oral hygiene or the professional cleansing of the implant surface from biofilm and calcifications.

In advanced periimplantitis, a surgical procedure can be indicated in order to remove the biofilm. A regenerative treatment can be done in the course of those surgical procedures in order to replace lost bone. Therapy interventions in periimplantitis are still predominantly based on the clinical experience, as reliable clinical data have not yet been available. However, research activities in this field have been numerous and new data a generated constantly, which is why more distinct guidelines for the treatment of those diseases can be expected.[1] Early diagnosis by periodontal probing and the evaluation of the health status of periimplant tissues are essential for the prevention of periimplant mucositis and periimplantitis. Early diagnostic identification permits early intervention, which can be clinically effective. If early symptoms are misjudged, a complex therapy is necessary, but may produce results which are less predictable.

## _Aetiology and pathogenesis

The literature has proven that the presence of microorganisms is an essential prerequisite for the development of periimplant infections. We know today that glycoproteins from the saliva accumulate at the titanium surfaces of the implant or abutment which are exposed towards the oral cavity immediately after implantation. This glycoprotein layer is then colonised by microorganisms. A subgingival microflora forms within a short amount of time after implantation, which is dominated by *Peptostreptococcus micros*, *Fusobacterium nucleatum* and *Prevotella intermedia*. The majority of periimplant diseases are characterised by gram-negative, anaerobic microflora, which is found in a similar fashion in periodontitis. High concentrations of periodontal pathogens, such as *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis, Prevotella intermedia, Tannerella forsythia* and *Treponema denticola*, have been detected in periimplantitis cases. Moreover, studies suggest that the microflora often contains *Fusobacterium nucleatum, Actinomyces* as well as *Staphylococcus aureus* and *enterococci. Staphylococcus aureus* also colonise other foreign elements, which, for example, may lead to complications in hip transplants. Titanium seems to promote the adhesion of *S. aureus*, which is often found in dental implants.[1]

The implant's soft tissue collar consists of an epithelial and a connective-tissue attachment. The epithelial periimplant mucosa, which consists of oral gingiva epithelium, oral sulcus ephitelium and non-keratinised junctional epithelium corresponds largely with the epithelial tooth-mucosa contact. The connective-tissue attachment to the implant is achieved via fibre bundles which are inserted in the marginal bone. They arrange themselves closely to the implant, parallely and circularly to its surface. Other than the connective tissue surrounding the tooth, the supraalveolar connective tissue is deficient in cells as well as vessels. This leads to a reduction of the defense mechanisms against bacterial influences on the implant. Periimplant inflammations can thus spread faster than comparable inflammations of the periodontium. Missing desmodontal structures limit the defense capacities of the host organism to the vessel proliferation within the marginal soft-tissue collar, which leads to an increase in the manifestation of the clinical inflammation symptoms of the marginal soft tissue.

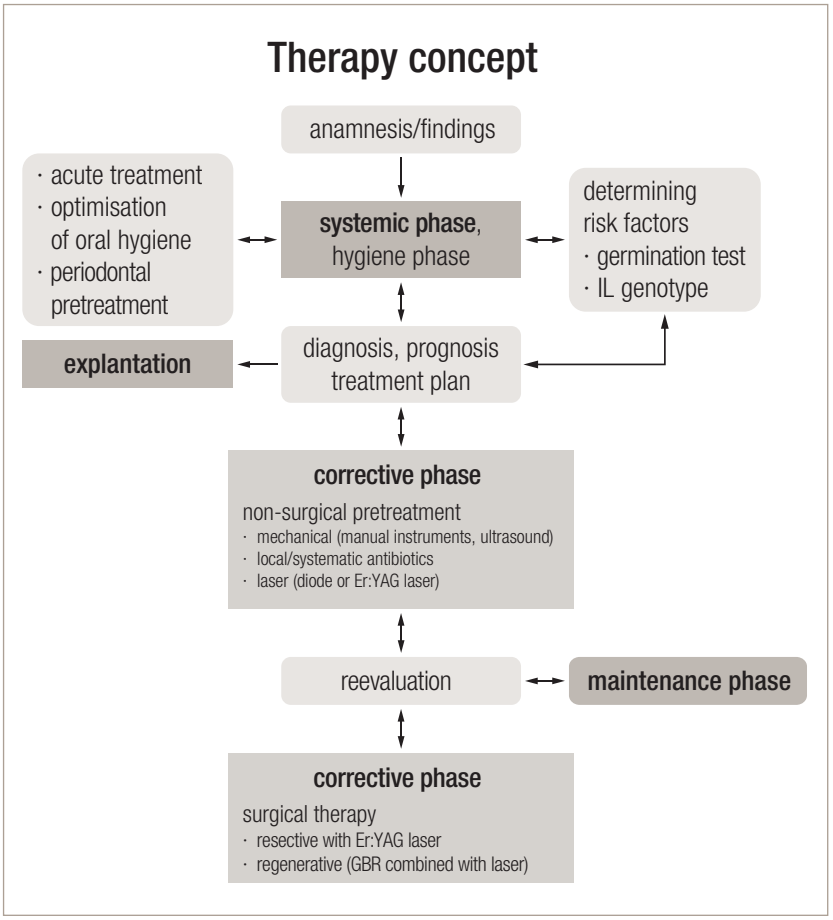


**Fig. 1**_Sequence of a systematic therapy of periimplant infections.

There probably is a connection between the microflora present in the oral cavity during implanta-

**Table 1**_Symptoms of periimplant infections.

| mucositis | periimplantitis |
|---|---|
| bleeding on probing | bleeding and/or pus on probing |
| reddening and swelling | reddening and swelling |
| surface inflammation | probing > 4 mm |
| no loss of bone | loss of bone |
| slight pocket formation | increased pocket formation |

**Fig. 2**_Fibre tips.
**Fig. 3**_Cylindrical fibre tip.
**Fig. 4**_Window hand piece.
**Figs. 5 & 6**_The patient presented with a loss of the implant-supported metal-ceramic bridge 35–37.

Fig. 2

Fig. 3

Fig. 4

Fig. 5

tion and the biofilm which develops on the implant. Periodontal pockets can therefore function as a reservoir for microorganisms for natural teeth in the partially edentulous. The microorganisms then settle at the newly-placed implant.

## _Periimplantitis: an inflammatory disease caused by infection (Tab. 1)

- Microorganismus colonise implants very shortly after insertion or uncovering of the implant in two-stage procedures.
- Implants are colonised by a microflora similar to that of natural teeth.
- Periodontally diseased teeth can function as a reservoir for pathogenic microorganisms.

Fig. 6

- It is imperative that periodontally diseased teeth are treated before implantation.
- Due to the possibility of the pathogenic microflora being transferred from the periodontal lesions to the newly-placed implant, an implantation is contraindicated in cases of an active periodontal disease.

The periimplant mucosa around titanium implants has many things in common with the gingival tissues of natural teeth. Like the gingiva, periimplant mucosa forms a collar-like barrier, which adheres to the surface of the titanium abutment. Periimplant mucosa is a keratinised oral epithelium, whose collagen fibres start at the crestal bone und run parallelly to the implant surface. Similarly to natural teeth, the accumulation of bacterial plaque causes an infection in the periimplant mucosa and increases the probing depth. After longer contact with dental plaque, the periimplant lesion extends apically without being encapsulated by the collagen fibres as in periodontitis cases. The inflammatory infiltrate can extend to the alveolar bone or even the marrow spaces in periimplantitis, while it is separated from the bone by ca. 1 mm of non-inflamed connective tissue in periodontitis. This might explain the varying degree and configuration of the bone defects in periimplant inflammations.

## _Diagnosis with dental probe and X-ray

Bleeding on probing as the clinical symptom which confirms mucositis occurs in up to 90 % of functioning implants. Unfortunately, the definition

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**Fig. 7**_Periimplant brightening in form of a significantly enlargened split in the course of the covering the complete implant surface between implant and surrounding alveolar bone.
**Fig. 8**_Careful uncovering of the alveolar process in region 36–38 under local anaesthesia, after forming the mucoperiosteal flap and exposing the bone defect in implant region 37.
**Fig. 9**_Granulation tissue is depicted in the cervical implant area.
**Figs. 10 & 11**_Settings for "implantitis" therapy (Fig. 10), which can be altered according to the experience and knowledge of the user (Fig. 11).

Fig. 7

Fig. 8

Fig. 9

Fig. 10

of periimplantitis varies and the term is used inconsistently in the literature. It was decided on a recent consensus conference that the definition of periimplantitis as an inflammatory lesion leading to bone loss was acceptable, but that the diagnostic criteria are anything but explicit. For example, it should be taken into account that bone remodelling occurs during implant healing, during which the most coronal periimplant bone can be lost. This physiological rearrangement can take up to one year and should not be seen as a pathological process. From the clinical point of view, the bone level at the moment of prosthetic restoration should be the defined as the reference value for future radiological changes of the bone height. Only in this moment should the reference X-ray be produced, which is then used for the assessment of the periimplant bone loss. It should be noted that measurement errors can occur even under ideal conditions: in cases of double measurement, a deviation of about 0.5 mm was documented. The diagnosis of periimplantitis is justified if there is a radiological bone loss of 2 mm compared to the initial values and combined with bleeding and/or pus on probing. In immediate loading, an X-ray after one year is recommended as a reference for future X-rays.[1]

## _Periimplant mucositis and periimplantitis: frequent complications in implant patients

Periimplant mucositis is described as a reversible inflammatory reaction of the periimplant mucosa without any symptoms of periimplant bone loss, comparable to gingivitis. Periimplantitis is described as the further progression of plaque accumulation and consequently the spreading of the bacterial infection to the periimplant bone, characterised by bone destruction due to the inflammation. It is seen as the pendant to periodontitis.

- Roughly four in five implant patients exhibit periimplant mucositis.
- After ten years, one in five patients develops periimplantitis.
- Periimplantitis is especially frequent in smokers, patients with insufficient oral hygiene and patients who have already had periodontitis.
- Implants with a rough surface accumulate more plaque when exposed towards the oral cavity than smooth implants.
- The prevalence of periimplantitis can be expected to rise in the future due the increasing replacement of teeth by implants and the use of moderately rough surfaces.

## _Our therapy concept

In principle, a procedure analogous to the systematic periodontal therapy, consisting of systemic phase, hygiene phase, corrective phase and supervision phase should be maintained in the therapy of periimplant infections. Figure 1 is the schematic representation of the systematic therapy of periimplant infections as performed in our clinic. Primarily, die pathogenic microflora must be reduced by a

Fig. 11

Fig. 12

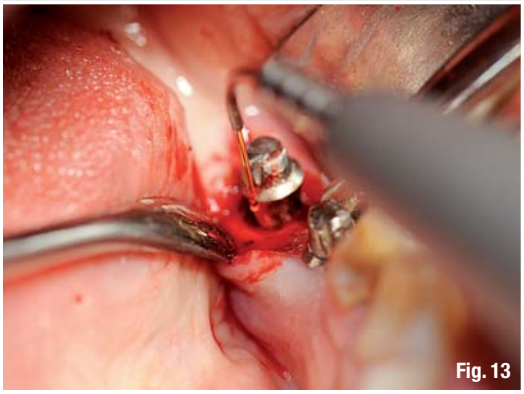
Fig. 13

Fig. 14

**Figs. 12 & 13**_Removal of the soft-granulation tissue from the split-shaped bone defect and the implant surface.
**Fig. 14**_Application of the fibre tip parallelly to the implant surface into the depth of the periimplant bone defect.

causal therapy in order to counteract a progression of the disease.[2] The removal of subgingival concrements and the bacterial biofilm of titanium implants is, however, hindered by various modifications of the implant surfaces.[3] Prosthetic options and superstructures often make the access to infected surfaces difficult. In this regard, decontamination or conditioning of the exposed implant surface is demanded in addition to the mechanical removal of the biofilm in order to optimise the removal of bacteria and their lipopolysaccharides from the microstructured implant surface. For this, a non-surgical therapy approach can be differentiated from a surgical one. The latter is obligatory in resective or regenerative procedures (guided bone regeneration, GBR). Contrarily, the removal of the biofilm as a preliminary to resective or regenerative procedure can be done surgically as well as after mobilisation of a mucoperiosteal flap under visual control.[4] It should, however, then be noted that critical probing depths have not yet been defined for the therapy of periimplant infections. These would help in deciding between non-surgical or surgical therapy approaches.[5] An adequate plaque control by the patient and a sufficient recall system are basic prerequisites for all therapy concepts.

## _Laser application in the therapy of periimplant inflammations

Laser applications have proven to be clinically effective in periodontology in our clinic. The high bactericidal potential of laser light in the gingival sulcus and the surrounding soft tissues is an advantage that has been described by authors such as Ben Hatit et al. 1996, Coffelt et al. 1997 and Moritz et al. 1997.[6-8]

It is imperative to note that the effects of different laser light wavelengths on implant surfaces vary. Thus, Nd:YAG laser must not be applied on titanium implant surfaces. This laser would destroy the implant surfaces, with a macroscopically visible welding effect. In contrast, Er:YAG lasers are suitable for the application in close proximity to titanium implants, especially for cleaning and decontaminating implant surfaces. Er:YAG lasers were introduced in 1974 by Zharikov et al. as solid state lasers with a wavelength of 2,940 nm in the near- to mid-infrared range.[9] The special quality of this wavelength is that it concurs with the maximum absorption in water and is even 15 times higher than that of the $CO_2$ laser. Depending on the physical laser parameters chosen by the user (laser power, focus-tissue distance, application time, pulse rate and energy density), different biological processes occur in live tissues. In thermo-mechanical ablation, the removal of biological tissue is based on the fact that that the proportion of water in the tissues undergoes a rapid transition from the liquid to the gaseous state when absorbing ultrashort laser light impulses. Accompanied by a fast expansion of the water, the pressure becomes high enough to blast off and thus remove hard and soft tissue material.[10]

**Fig. 15**_Uncovering the implant circularly, split-shaped bone loss.
**Fig. 16**_Filling the four-wall bone defect with xenogeneic bone substitute.
**Fig. 17**_Good success prospects after primary coverage of the defect.
**Fig. 18**_Postoperative check-up after laser decontamination and augmentation with Bio-Oss® granulate of a particle size of 0.25–1 mm und coverage via Bio-Gide® membrane.

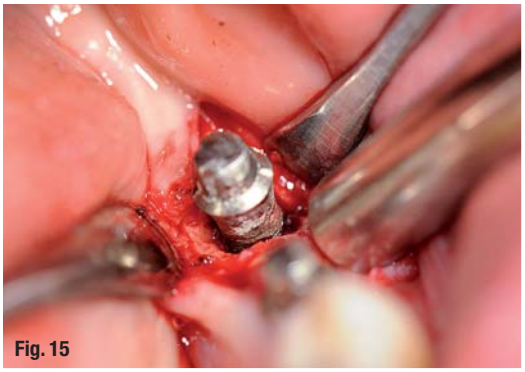
Fig. 15

Fig. 16

Fig. 17

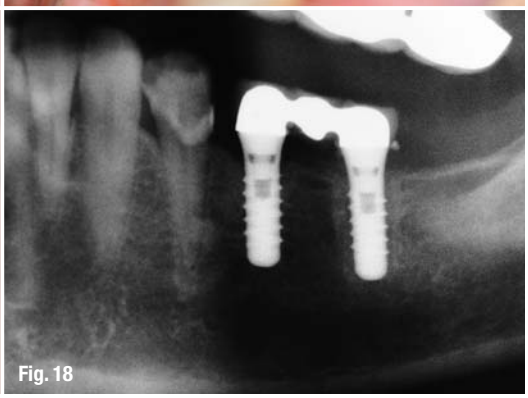
Fig. 18

Of course, the laser tip/laser fibre used must ensure that all decontaminated areas of the implant surface or the inflamed implant site in the alveolar bone can be reached precisely. In my practice, I use fibre tips (Fig. 2) as well as a cylindrical working end, which reflect the laser light via a bevel (phase) in an angle of 45° (Fig. 3), so that parts of the macroscopically present implant screw threads are treated three-dimensionally. In easily accessible or exposed implant surfaces or defect areas of the alveolar bone, I like to use the so-called window hand piece, which allows an extensive laser-light application with a high energy density without fibre or sapphire light wedge (Fig. 4).

## _Case presentation

In the following patient case, the resective and regenerative treatment sections of the complex therapy concept are discussed only exemplarily for didactic reasons.

*Anamnesis and findings*

Female patient, 56 years old, smoker, no general diseases, condition 14 years after implant insertion, regular dental check-ups until 20 months ago, afterwards neither prophylaxis or check-ups, treatment stop.

The patient presented with a loss of the implant-supported metal-ceramic bridge 35–37 (Figs. 5 & 6, lateral and occlusal view). Clinical examination showed: mild loosening of implant regio 37 (grade 1), minimal bleeding on probing, minor pus release region 37. Contrarily, there was no bleeding on probing or pus release in implant 35. However, all in all no redness of the gingiva, no inflammatory infiltration, swelling or loosening of implant 35, whose percussion sound was bright and clear, were detected.

Radiologically, a periimplant brightening in form of a significantly enlarged gap in the complete implant surface between implant and surrounding alveolar bone (Fig. 7) was noted.

After a modified application of our therapy concept (Fig. 1), we attempted a prompt surgical treatment of the periimplant infection in implant regio 37 due to the loss of the bridge. The patient was informed about the limited prospects of success with regard to implant preservation already at the beginning of the therapy. The alveolar process was exposed carefully under local anaesthesia after forming the mucoperiosteal flap in regio 36–38 and the bone defect was prepared in implant region 37 (Fig. 8). Granulation tissue is depicted in the cervical implant area in an overview of the exposed operational field (Fig. 9). The configuration of the bone defect made the application of various laser fibre tips necessary. We used an Er:YAG laser (KaVo KEY 3+ by KaVo GmbH, Germany). The programme selection already provides preconfigured settings for the therapy of "implantitis" (Fig. 10), which can be altered according to the experience and knowledge of the user (Fig. 11). Thus, the first therapy step of laser

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**Figs. 19 & 20**_Probing depth of 2 to 2.5 mm.
**Figs. 21 & 22**_Good agglomeration of the surrounding bone in implant regio 37.

Fig. 19

Fig. 20

Fig. 21

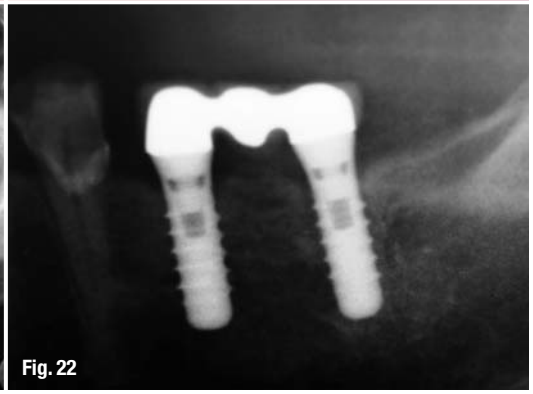
Fig. 22

applications consisted of using a thin fibre (Fig. 2), which can also be applied in other surgical situations in order to remove the soft granulation tissue from the slit-shaped bone defect and the implant surface (Figs. 12 & 13).

Afterwards, a special cylindrical sapphire tip was used (Fig. 3), which features a 45° bevel (phase), which helps the laser light reaching hardly accessible areas, for example the screw threads of the implants, by circular and lateral radiation in a 45° angle. For this, energy was increased to 350 mJ at a pulse rate of 15 Hz and 5.25 W. The figures show how the fibre tip is held parallelly in accordance with the implant surface into the depth of the peri-implant bone defect (Fig. 14). Fig. 15 already depicts a visible circular exposition of the implant with a split-shaped bone loss, which corresponds to class 4 according to Spiekermann 1993.[11]

After laser decontamination and cleansing of the implant surface via Er:YAG laser and physiological NaCl solution, all the macroscopically present granulation tissue and the infected surface of the alveolar bone facing the implant were removed. This was followed by filling the four-wall bone defect with xenogeneic bone substitute, which was accumulated up to the implant region for minimal vertical augmentation (Fig. 16). An implant plastic with removal of the rough surface as was previously described by other authors[1,4] was not applied in order to avoid introducing titanium particles to the surrounding bone, which can be seen later in the X-ray. The procedure was discussed extensively with the patient beforehand and according to our experience provides good prospects of success (Fig. 17). After covering the xenogeneic augmentation material by a collagen membrane and primary wound closure, the implants were stabilised first by a long-term temporary restoration in configuration with the former bridge. This long-term temporary solution, which was visibly reduced in its occlusal height, was used for temporary splintage for six weeks in order to stabilise the minimally loosened implant 37. Immediate postoperative control after laser decontamination and augmentation with Bio-Oss® granulate of a particle size of 0.25 to 1 mm and coverage via Bio-Gide® membrane was checked radiologically (Fig. 18).

After a clinically uneventful healing period of six weeks, the long-term temporary solution was exchanged with the original definite bridge restoration. Treatment success was checked after four and, later, six months. Professional prophylaxis was performed in addition.

Clinical check-up four years after laser therapy and augmentation in region 37 presented us with a patient who was, subjectively, without any pain and a clinically stabile implant abutment in region 37. Neither bleeding on probing or pus were recorded. Probing depth was and is 2–2.5 mm (Figs. 19 & 20). Radiological check-up (Figs. 21 & 22) showed a good agglomeration of the surrounding bone in implant regio 37.

The prospects of success in saving an implant by laser decontamination and combined GBR procedure, according to my experience, are high. If the implant surface as well as the bony implant site can be decontaminated, one can rely on the high regenerative quality of the alveolar bone. Additional application of xenogeneic augmentation materials suggests a significant improvement of the therapy success, as both guidance for the yet-to-be formed bone and primary mechanical stability of the implant are achieved immediately after insertion of the material.

Users of GBR techniques already know that this biological process demands space as well as stability and the longest possible regenerative period. Therefore, exposition to masticatory forces should be avoided during the four- to six-weeks healing period. For this reason, removing the complete supra structure of several implants and letting the wound heal after coverage is the safer method in general.

## _Summary

Today, the success rate of implant therapy is generally regarded to be high. However, infectious complications such as mucositis or periimplantitis are frequently documented and seen as usual complications in implants which have been in situ for five to ten years. Periimplant mucositis and periimplantitis have infectious origins. When not treated, they will lead to implant loss sooner or later. As soon as the inflammation has reached the periimplant bone, the implant surfaced should be cleansed and decontaminated by applying an Er:YAG laser in addition to an ablative treatment of the infected bone. The combined treatment of GBR procedures improves the clinical situation and favours biological regeneration._

_contact laser

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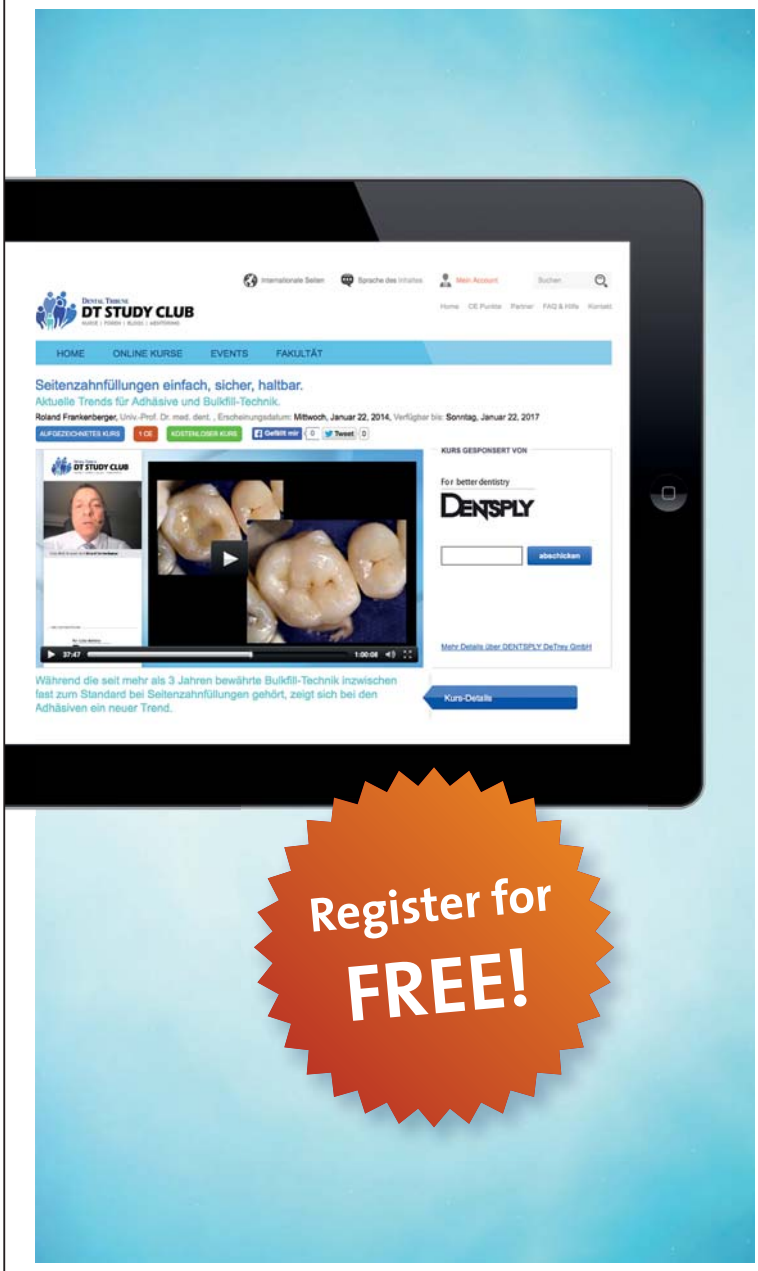


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