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RESEARCH ARTICLE

Occlusal Trauma has a Primary Role in the Etiology of Site-Specific Periodontitis/ Perimplantitis

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ABSTRACT

An in media preponderant school of thought considers the onset from the very beginning of the periodontal/periimplant disease due to an infection. In other words the disease begins when pathogenic bacteria infect a tissue which is wholly healthy thanks to saprophyte bacteria. We will consider occlusal trauma as well as cause of the disease. The aim of the work is to demonstrate how the long-lasting mechanical stress over gingival-tooth/implant apparatus can induce the change of bacterial flora from saprophyte to opportunistic pathogenic one capable of causing periodontal/perimplant infection. Two cases are presented referring to two different patients, one affected by periodontitis, the other by perimplantitis, in which periodontitis/perimplantitis occurred only on a single tooth/implant, both in evident occlusal trauma, sparing all contiguous elements.

Since periodontal/perimplant disease is a multifactorial syndrome with hypothetical causes from infectious to genetic ones, or related to internal medicine and biomechanics,this research proposes to understand the causes of the periodontal/perimplant disease moving away from conventional schemas based on the insufficient hygiene with consequent build-up of dental plaque which is considered "the only "cause of the disease. In fact we must consider both teeth and implants not as static elements but as a dynamic complex which during its function can undergo a very heavy load according to precise biological laws. In addition to all the best-known risk factors the causes that displace mandible and therefore modify occlusion must deserve specific attention. Considerable overloading on teeth and/or implants involved can contribute to the pathogenesis of the tooth/implant and its supporting tissue too. The two clinical cases of the work present site-specific periodontitis and perimplantitis. Both cases show that occlusal trauma with its consequent chronic mechanical stress is the initial cause of the disease. Later the subsequent inflamed tissue becomes an easy road to the periodontal-perimplant infection as final stage. Following the evolution of the two cases it becomes apparent that it is important to know the diseases due to occlusal trauma. Only through that knowledge we can prevent the diseases described above.

Introduction

In periodontology, bacterial plaque is considered the fundamental clinical element in which periodontal disease is identified, as it contains what is considered the unique and absolute pathogenic noxa: bacteria.

Every diagnostic and therapeutic protocol is oriented towards bacterial plaque and the absolute preventive value (ex juvantibus) of oral hygiene is emphasized.[1-7].

Periodontal disease, however, should not be interpreted as an infectious disease ab initio, especially if diagnostic criteria directed at occlusal problems are adopted.[8-10]. We emphasise that the oral cavity also represents an ecosystem regulated according to microbiological and microbiotic balances, like any other district of the organism in relation to the micro- and macroenvironment.

Most of the bacterial species considered to be opportunistic pathogens are, in fact, already normally present in the periodontally healthy individual, albeit in peculiar proportions, but what is most significant is that endless attempts to identify the aetiological agent in the healthy chorion have never yielded clear results.[11-14].

This could mean that unless a continuous solution is established through the mucogingival seal, saprophytes would not be able to invade the underlying tissues as they are unable to perforate a healthy seal in any way.[15-17].

The role of chronic occlusal trauma in causing tissue suffering has been demonstrated. This favourable terrain may favour subsequent periodontal infection. This condition would justify the transformation of the bacterial flora from saprophytic to opportunistic pathogenic [18-20] with the consequent interpretation of the site-specificity of the periodontal lesion.[21-22].

The effects of functional mechanical stimulation on bone tissue, both in terms of remodelling and in terms of dystrophy and atrophy, are well known. Compressive and diplacing forces, if biomechanically inappropriate and replicated over time, can lead to dystrophic pictures with dystrophy and atrophy of the periodontal organ.[23-25].

Understanding the pathogenetic mechanism of periodontal disease is, according to our research, the parallel investigation of the bacterial profile, static-dynamic occlusion and all biomechanical dysfunctional sources. Occlusal rebalancing by means of conservative cuspal remodelling favours the improvement of the dento-parodontal site in terms of microcirculation, with improving consequences on tissue trophism and bacterial profile [26,27] as well as on the monitoring of classical periodontal parameters (probing, bleeding, mobility). [28, 29].

In other words, it is the morphological quality of the bacterial profile that indicates the trophic or dystrophic conditions of the periodontal niche and of the tooth-gingiva-parodontal ecosystem by determining bacterial morphologies (rods, filamentous, fusiform and spiral-shaped) with anaerobic metabolism to the detriment of the predominantly aerobic cocci. [30, 31].

When we observe the damage, even minimal, caused by occlusal trauma, we know that we are always faced with a dislocated mandible that occludes in procoresis on a substitutive static, with the condyles displaced mesialy or mesio-laterally with respect to the position of centric relation. By repositioning the condyles from this pathological relationship, in the most physiological position of their primitive centric relationship and guiding the mandible to occlude, we will make it rest on those precontacts that we must eliminate to regain occlusal harmony.

The ability to perform this manoeuvre correctly allows us to immediately highlight the presence of a precontact and consequently the presence of an occlusal imbalance.

This is very important because it immediately makes it possible to ascertain that the lesions of the masticatory apparatus detected are attributable to occlusal trauma.
Repositioning of the condyles can be achieved using individually or alternatively the following operative techniques:

- manual
- instrumental (bite-plane, Lucia's jig, Pasqualini's stopper)
- bioelectric (myo-monitor, myo-pulsar, centric occlusal detector)
- psychosomatic.

THE PSYCHOSOMATIC CONDYLAR REPOSITIONING

The 'smile technique' is based on certain psychosomatic tricks that create a special operator-patient relationship that facilitates the manoeuvres of condylar repositioning and

precontact signalling. The 'smile technique' makes it possible to localise the centric precontacts by exploiting with manual manoeuvres and appropriate terms certain spontaneous qualities common to everyone. These spontaneous qualities are identified in four perceptive stages:

- smile
- touch
- clench
- detach

This manoeuvre practically brings the mandible into the physiological free-way-space position, without muscle tension, and the condyles will be correctly positioned in the glenoid fossa in centric relation. At this point it is sufficient to gently guide the teeth to the occlusal contact, the pure rotation movement will prevent the condyle from changing its position in the glenoid fossa and allow the presence of any centric precontact to be detected.[32].

Clinical Case 1

A 32-year-old Caucasian woman, seen in 1989 with the right upper incisor, vital, mobile, affected by periodontal disease, presenting a bone atrophy which was progressively worsening despite the continuous specialist therapies practised and still in progress. He reports numerous sessions of professional hygiene, scaling, root planing, and prophylaxis with courses of antibiotic therapy, which, however, did not stop the aggravation of the atrophy. Furthermore, to limit its mobility, the tooth had been splinted with the central incisor contiguous. The clinical and radiographic examination showed that the infection only affected the right incisor and did not involve the adjacent teeth (Figs. 1, 2). Suspecting the presence of an altered occlusion, a definite diagnosis is required.

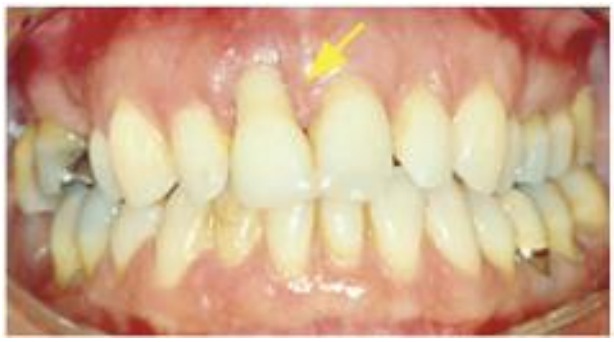

Fig. 1 Right upper central incisor severely compromised by site-specific periodontal disease (1989).

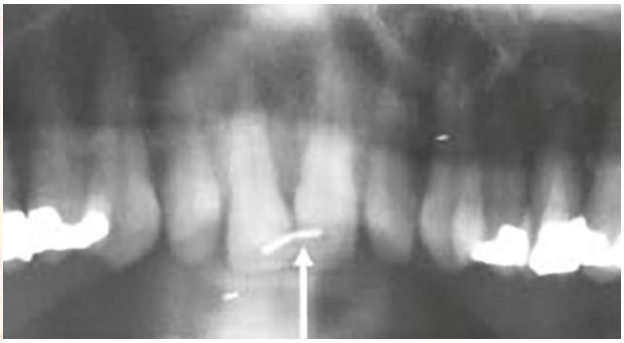

Fig. 2 - The radiograph and splint with the left upper incisor

In the presence of an occlusal imbalance, we are always faced with a dislocated mandible that occludes in procoresis on substitutive statics, with the condyles displaced mesially or mesio-laterally with respect to the position of centric relation. By repositioning the condyles from this pathological relationship, in the more physiological position of their primitive centric relationship and guiding the mandible to occlude, we will make it rest on any precontacts: the presence of which will confirm the diagnostic suspicion. In fact, the manoeuvre described above shows, in our patient, the presence of a precontact on the mesio palatal marginal ridge of tooth 17, which is extruded due to the absence of the antagonist. The precontrast can be defined as centric because the mandible has been repositioned in a centric relationship and the presence of the centric precontrast indicates that the patient's habitual occlusion is realised thanks to the dislocation of the mandible on a substitutive static that, having lost the centric relationship, is no longer physiological, but pathological (fig. 3).

To better understand the behaviour of the mandible in the presence of a centric precontact, let us recall some concepts of physiology and occlusal pathophysiology.

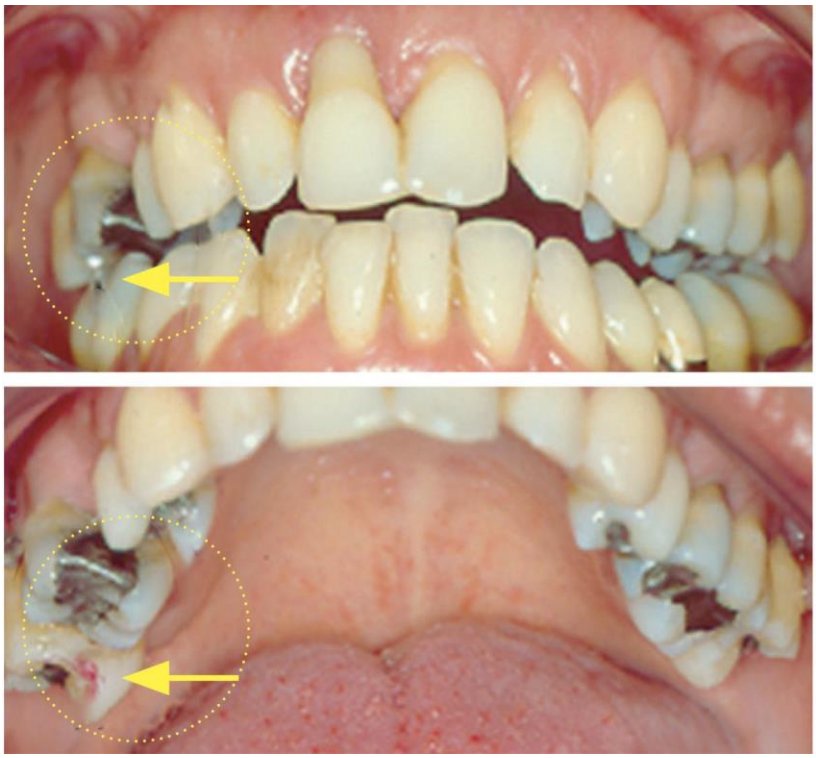

Fig. 3 - The repositioning of the condyles in centric relation shows a centric precontact on the mesial marginal crest of 17 (extruded due to the absence of the antagonist) precontact marked in red by the articulation chart (circle).

Occlusion can be defined as 'stable centric' when, without any bodily displacement of the centres of rotation of the condyles, with maximum muscular economy and without any tension of the articular ligaments, the mandible, in the closing movement rotating in the hinge axis, stops on numerous and simultaneous static contacts that maintain the intercuspation in a centric relationship. The pressures exerted on the teeth are physiological and accompany without consequences the thousands of daily swallowings (3500 swallowings according to Mountcastle). [33]. This is because the pressures are evenly distributed along the central axis of the roots of premolars and molars, and keep the structures designed to bear them within the limits of resistance.

Whereas, on the other hand, the "front teeth", central incisors, lateral incisors and canines, due to their natural inclination, develop transversal forces in the contact, and this is the reason why they must not have contact in the physiological static occlusion, but must only, at most, touch each other because otherwise they would be damaged by the transversal forces that develop during swallowing (clench or over-occlusion). [34,35].

If, rotating in the hinge axis, the occlusal plane of the teeth in the closing movement encounters premature contacts, equally centred, but incapable of giving the jaw a stable static support adequate for swallowing and chewing (precontacts), the conditions of an unstable occlusion are realised.

The occlusal instability caused by precontact alters the harmony of the neuromuscular system compromising the normal automatisms of chewing and swallowing. For this reason, the CNS, which cannot tolerate the instability, plans the search for new statics in an attempt to re-establish the stability of the occlusion itself.
Unable to oscillate to balance the pre-contact, the mandible dislocates forward and/or to the side (procoresis), accompanied by a bodily displacement of the centre of rotation of the condyles, in search of an increase in the dental fit that allows it to stabilise with the best possible balance. In this way, a replacement statics is achieved, with acentric anterior contacts that are no longer physiological, which, although pathological, allow chewing and swallowing with normal automatism with the pathological extension of the contact to the front teeth (Fig. 4).

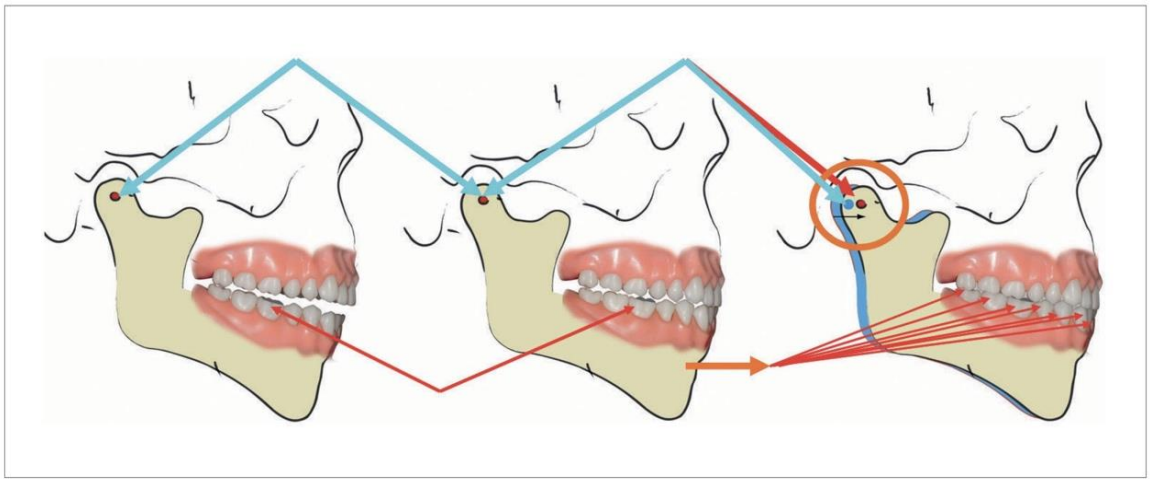

Fig. 4 - The dislocation of the mandible that from the occlusal instability caused by the precontraction (central drawing) shifts to a substitutive statics with pathological but more stable acentric contacts that may involve the frontal teeth.

The jaw, however, in the closing movement on the replacement static loses its centric relationship. Therefore, to maintain the new equilibrium, tensions develop in the musculature and articular ligaments and these tensions, during the clench of swallowing, increase the action of the occlusal forces that are unloaded on the imbalances of the substitutive support plane, favouring a mechanics that can exceed the physiological resistance limits of the structures involved. This creates the conditions for the development of pathologies affecting the teeth, implants, mucous membranes, TMJ and/or the supporting tissue, caused by static occlusal trauma. [36] (Fig. 5).

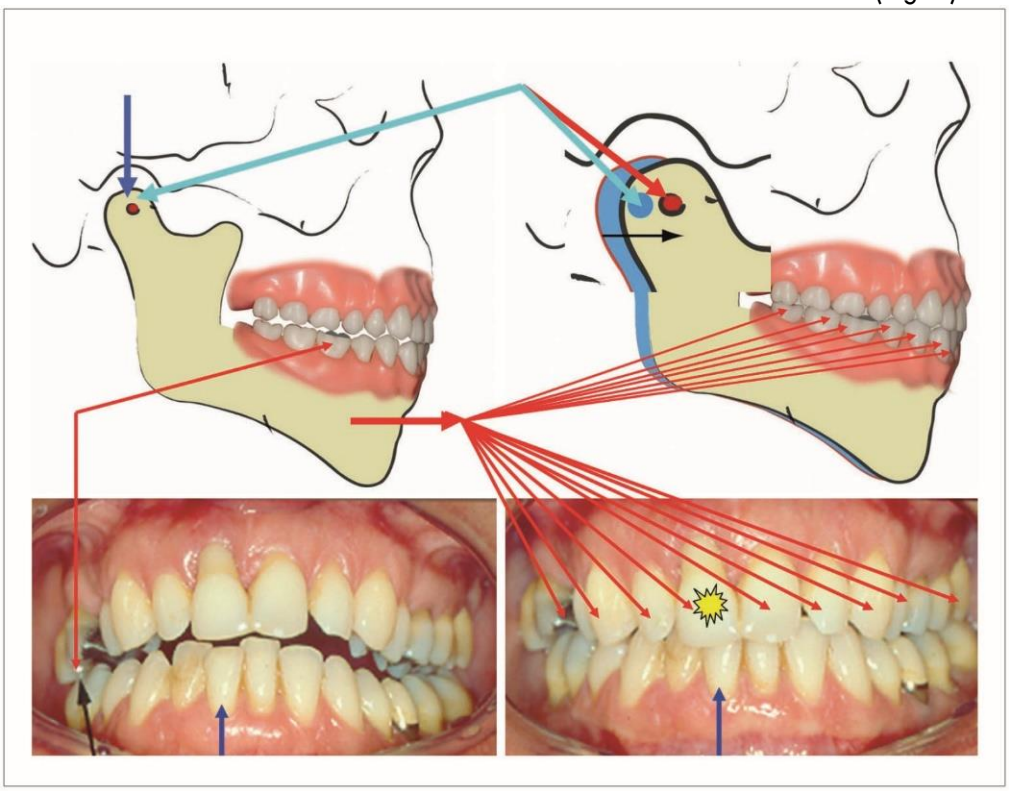

Fig. 5 - In the upper part of the figure the drawing shows the displacement of the mandible from the centric precontraction to the pathological replacement statics and at the same time in the lower part of the figure the dislocation of the patient's mandible from the centric precontraction to the usual pathological replacement statics is shown by analogy.

In summary, a centric precontact becomes responsible for:

- occlusal instability that alters the harmony of the neuromuscular system;
- altered mandibular posture;
- a series of modified static occlusal contacts involving the frontal teeth;
- a consequent modification of dynamic occlusal contacts;
- a different position of the condyles in the glenoid fossa with possible temporomandibular disorders (TMD);
- an altered muscular activity due to the loss of the centric relationship;
- onset of parafunctions that accelerate the progression of the damage.

Returning to our case and for a better understanding of how the frontal teeth involved in the pathological contact are compared, after the dislocation of the mandible on the patient's habitual replacement statics, let us compare the photos of the mouth with the previously proposed schematic drawing.
As can be clearly seen in figure 5, the lower right central incisor, indicated by the blue arrow, is more vestibularised than the line of the other teeth and, in the replacement statics, involves the upper incisor affected by site-specific periodontitis, its natural antagonist, first in the contact, traumatising it continuously during the clench of swallowing.
The importance of occlusal trauma as an aetiological factor is evident, it explains the site-specificity of the lesion and the reason why the infection - because it is an infection - remains localised to the affected incisor and does not extend to adjacent teeth.

It also explains the progressive worsening of the disease and the lack of positive results although the patient was always kept under specialist supervision and received the usual therapies according to the periodontal disease protocol. The incisor, now too compromised, was spontaneously lost a few days after the first visit.

Clinical Case 2

It refers to a young 19-year-old Caucasian woman with congenital agenesis of the upper lateral incisors, treated by her orthodontist with distalisation of the canines to create space for two replacement implants for the missing incisors. After the flap-less insertion of two bi-corticised Italian school screws (1986), two immediately loaded provisional crowns were immediately cemented in place, with careful control of the absence of static and dynamic precontacts. When the soft tissue of the paradentium healed, the provisional resin crowns were replaced by two single porcelain crowns, respecting the physiological occlusion (Figure 6).

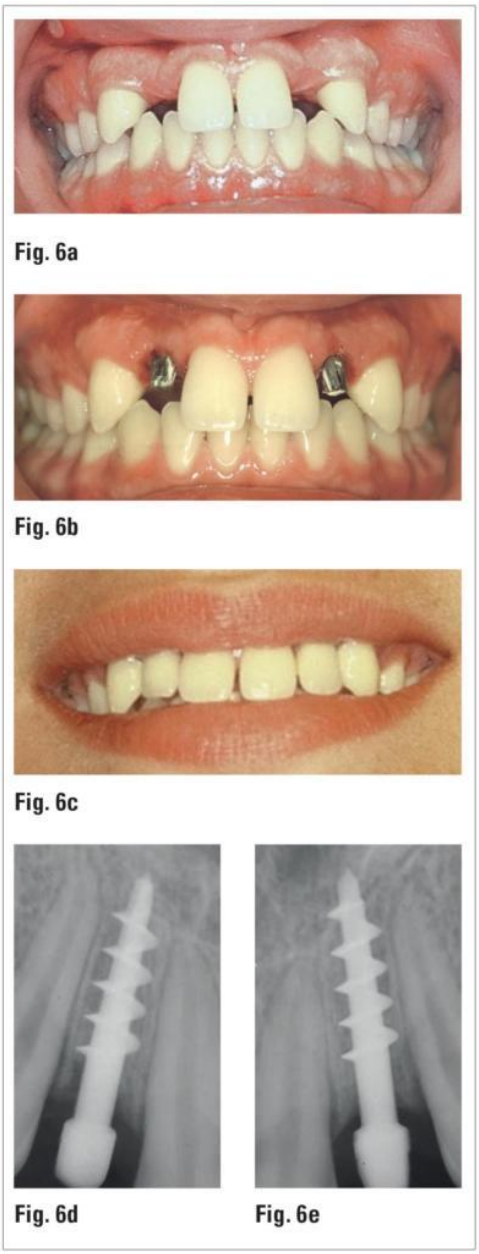

Fig. 6 - Agenesis of the lateral incisors in a young patient (1986). The two one-piece monoimplants performed using the flap-less technique. The radiograph and the two porcelain-gold crowns (6c).

She was not seen again until three years later because she was concerned about the considerable gingival inflammation that had developed at the crown of the left lateral incisor (figure 7).

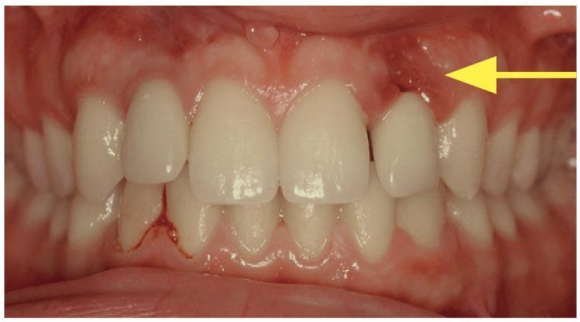

Fig. 7a

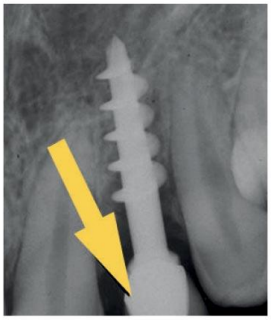

Fig. 7b

Fig. 7 - After 3 years the left mono-implant appears in distress (7a), on radiographic control a discrete peri-implant bone resorption is noted (7b).

The X-ray showed initial bone resorption involving the implant, so peri-implantitis was diagnosed. Also notable is the difference in the external appearance of the mucous membranes surrounding the crowns of the two implants. The anamnesis seems to provide no data on the possible causes of the peri-implantitis; only the filling of a molar performed by another colleague about a year earlier is reported, and therefore with no apparent relation to the current problems. The repositioning of the condyles in centric relation did, however, reveal the presence of a single precontact, marked by the articulating chart with the help of Pasqualini's stopper, precisely on the amalgam filling.

The resulting dislocation of the mandible shifted forward on the replacement static involved the front teeth in the contact, traumatising in particular the crown of the implant in position 22 during the clench of swallowing, opening the door to pathogenic bacteria and thus inducing the site-specific peri-implant lesion (figure 8).

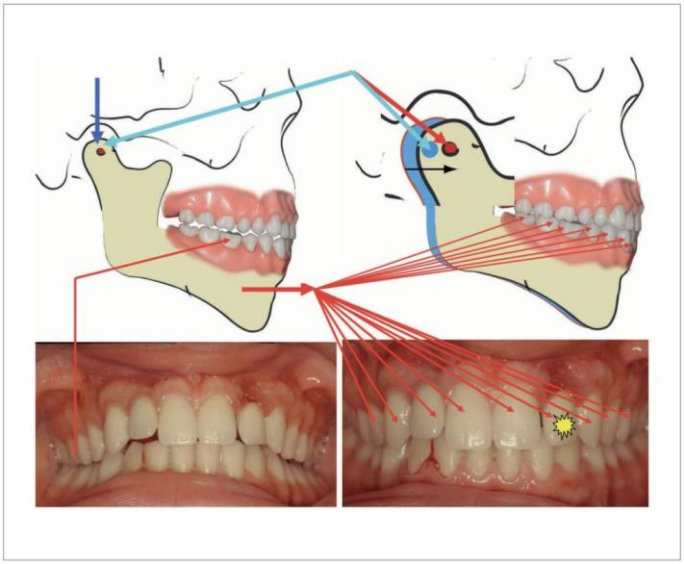

Fig. 8 - Lower part: The repositioning of the condyles in centric relation allows the identification of a centric precontact on 16 indicated by the red arrow in the left photo. The dislocation of the mandible on the replacement static, shifting the contact on the front teeth, traumatised the crown of the implant in position 22 more violently inducing the peri-implant site-specific lesion (right photo). Upper part: As in the previous case, we used the schematic drawing associated with the patient's photos to make the dynamics of the mandibular dislocation more understandable.

Removal of the precontact restored the mandible to centric relation and eliminated the indirect occlusal trauma on the crown of the 22 (figure 9).

The patient, seen again after one month, was completely healed and the prosthetic crown could be replaced.

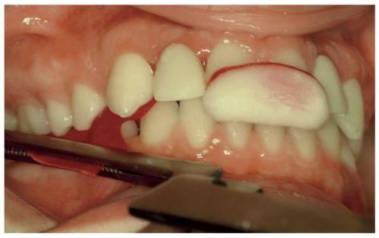

Fig. 9a

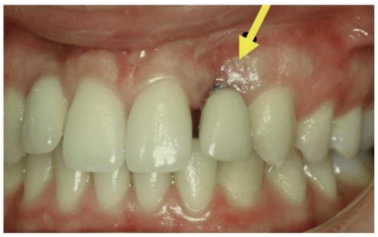

Fig. 9c

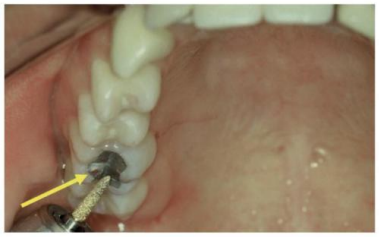

Fig. 9b

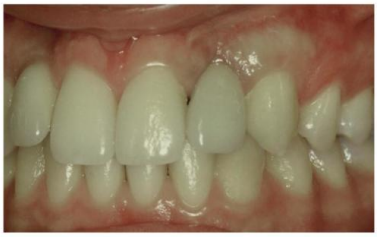

Fig. 9d

Fig. 9 - The exact and punctiform marking of the pre-contact on the occlusal plane of the amalgam, which, when removed, returns the occlusion to its correct statics (9a, 9b). The health of the peri-implant mucosa healed spontaneously after only occlusal rebalancing and porcelain crown replacement (9c, 9d).

It is very interesting to compare the radiograph taken at the time of the implant suffering with the one taken after the crown replacement, with the surprising restoration of the peri-implant bone tissue (1989) (figure 10).

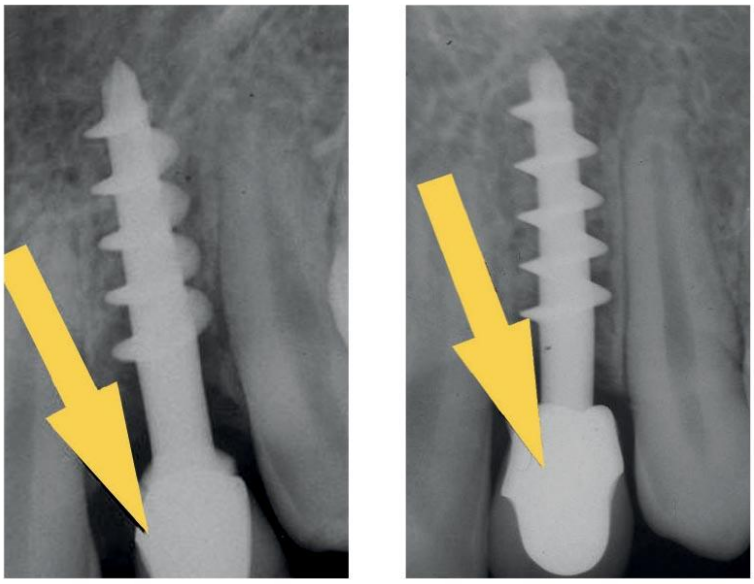

Fig. 10 - Radiographic control. In the image on the right, the amazing healing of the peri-implant bone tissue is evident.

The amalgam filling, although skilfully modelled, but without occlusal contact control, presented a centric precontact which, by displacing the mandible on the replacement static, involved the front teeth in the contact, traumatising precisely the crown of the incisor in position 22 and provoking the peri-implantitis site specific to the implant inserted three years earlier. Compared to the previous clinical case, it was possible to intervene in good time before the injuries became irreversible. In 1986 there were no specific treatments for peri-implantitis, so none of the therapies currently in use were practiced.[37-39]. Healing of the bone tissue occurred spontaneously after removal of the precontact which returned the mandible to its physiological centric position restoring occlusal harmony. This shows how the aetiology of this site-specific peri-implant lesion is related to the trauma of occlusion (cause-effect). [40-42].

Conclusions

In view of the results obtained, and above all of the current knowledge of the nature of the aetiopathogenetic bases, periodontal disease should be considered, rather than infectious ab initio and dependent solely on the bacterial component present in the dento-gingival plaque, a true chronic involutional dystrophic-atrophic syndrome on a biomechanical-dysfunctional basis.
Periodontal damage is the result of environmental influence by chronic occlusal-dysfunctional overload, which induces a reaction of the supporting apparatus with adaptive phlogosis. The bone tissue, subjected to persistent dysfunctional actions, reacts according to genetic and epigenetic response modes with a phlogistic evolution that progressively moves away from physiological remodelling with an increase in osteoclastic phenomena to the detriment of osteoblastic ones and consequent progressive atrophy. [43-46].

The authors suggest a critical analysis in the interpretative attitude following the clinical approach to periodontal disease, which should be considered as the response to complex and multifactorial environmental pressures (biochemical, biomechanical, metabolic, immune, microvascular, psychoemotional, bacterial). On this principle, a secondary overlapping evolution in a phlogistic-sectic-bacterial sense called periodontitis and/or peri-implantitis is possible. It follows that the prevention and/or treatment of periodontal disease and its recurrences should result from a multifactorial analysis through an interdisciplinary therapeutic approach.

Conflicts of Interest Statement

"The authors have no conflicts of interest to declare".

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