

'Help, I want to have a fresh breath' – how periodontists can help patients tackle the taboo of halitosis

Halitosis remains a big taboo in modern society, even though recent research has shown that it affects nearly a third of the global population. Although not all cases of halitosis are related to periodontitis, there is a clear relationship between the two diseases. Kavitha Seerangaiyan and Edwin Winkel, members of the International Association for Halitosis Research, explain the issues and show how periodontists can help their patients with halitosis.

Halitosis is generally described as an offensive or unpleasant odour from expired air. Halitosis is coined from Latin *halitus* meaning "breath" and the Greek *osis* meaning "abnormal" or "diseased". Oral malodour, *fetor ex ore*, and *fetor oris* are other terms for halitosis. The long history of halitosis dates back to ancient writing of Chinese, Greek, Islamic, and early Christians. In recent years, the scientific knowledge on the source and cause of halitosis has improved considerably, but halitosis remains one of the biggest taboos of modern society. In a survey involving about 50,000 individuals in the Netherlands, nearly 70% of participants believed that halitosis is a taboo topic to talk about. A recent systematic review by Silva et al. (2018) estimated a worldwide halitosis prevalence of 31.8% and suggested a trend towards increase in prevalence. There are both oral and non-oral sources of halitosis (see figure 2). Nearly 80-90 per cent of halitosis cases originate in the oral cavity and are called intra-oral halitosis (IOH). This involves bacterial degradation of sulphur containing amino acids (methionine, cysteine, and cystine) resulting in volatile sulphur gases such as hydrogen sulphide (a smell like rotten eggs), methyl mercaptan (a pungent smell that resembles that of rotten cabbage), and dimethyl sulphide (unpleasantly sweet). Between 10 and 20 per cent of halitosis cases are of non-oral origin and are called extra-oral halitosis (EOH); they may some-



Figure 1: Organoleptic mouth-breath evaluation, the 'gold standard' of halitosis diagnosis.

times be a manifestation of serious disease. The source of EOH includes infection from the upper and lower respiratory tract, the gastrointestinal tract, and certain metabolic diseases in the kidney and the liver. Recent research has identified a genetic element (a mutation in SELENBP1) among the causes of EOH. In order to attain a proper diagnosis and the correct therapy, it is of utmost importance to differentiate between the oral and non-oral origin of halitosis.

Classification of halitosis

Both IOH and EOH are forms of what is termed "genuine halitosis" and IOH is further categorised as either physiologic halitosis (no apparent disease or pathologic condition) or pathologic halitosis (occurs as a consequence of an infection of the oral tissues). As well as genuine halitosis, there are also three other classifications related to halitosis: temporary halitosis, pseudo-halitosis, and halitophobia.

- **Temporary halitosis:** caused by certain food substances, such as onion and garlic. "Morning breath" is a form of temporary halitosis that disappears after activities such as breakfast and tooth-brushing.
- **Pseudo-halitosis:** although no oral malodour is present, the patient stubbornly believes in its presence.
- **Halitophobia:** patients persist in the belief of the presence of halitosis even after therapy for genuine halitosis or pseudo-halitosis.

Intra-oral halitosis or oral malodour

IOH has a negative impact on quality of life and interpersonal relationships. Assessing IOH is often subjective and estimating its prevalence is also hindered by a lack of standard methodology and sociocultural differences. IOH is mainly attributed to volatile sulphur compounds (VSC), namely hydrogen sulphide and methyl mercaptan (which together ac-

count for 90% of all VSC content) and, to a lesser extent, dimethyl sulphide. The main aetiologies for IOH are tongue coating (a white or yellow deposit on the tongue's dorsal surface), periodontal disease, or the combination of the two. These aetiologies were well demonstrated in a study involving 2,000 patients that showed tongue coating as the cause of IOH in 51% of cases, gingivitis/periodontitis was responsible for 13%, and the combination for a further 22%. The other aetiologies were stress and xerostomia. In periodontally healthy patients as well as in those with periodontal disease, the tongue coating plays an essential role in the production of volatile sulphur gases. Despite the severity of periodontal diseases, the study of Bosy et al. (1994) emphasised tongue coating as the major source of VSC. The components of tongue coating – such as desquamated epithelial cells, blood cells, and bacteria – provide a suitable environment for putrefaction and the production of volatile sulphur compounds. The anatomy of the tongue – with a surface area of approximately 25cm², numerous fissures, crypts, and papillae – promotes the growth and colonisation of bacteria that cause bad breath. The tongue dorsum, posterior to the circumvallate papillae, has been reported to carry higher loads of bacteria compared to other regions of the tongue. The anatomy of the tongue itself is a barrier to the cleansing action of saliva

1-4	Tackling the taboo of halitosis	5-6	Perio & Cardio campaign	7	Symposium on EFP clinical practice guideline	8	Latest JCP research
-----	---------------------------------	-----	-------------------------	---	--	---	---------------------

and, over time, an anaerobic environment with an increase in the thickness of the tongue coating is formed. The formation of tongue coating is multifactorial, but the degree of oral hygiene is the strongest influential factor. In a recent study, the metabolic profile of IOH tongue coating showed that branched-chain fatty acids from bacteria might underlie the formation of tongue coating. The tongue is normally pink with the presence of a thin white coating, which does not in itself imply the presence of IOH, although the amount of tongue coating has been reported as increasing in patients with bad breath. The greater the amount of tongue coating, the higher the production of the volatile sulphur gas. Patients with a history of periodontitis had tongue coating six times greater than those with no history of periodontitis and produced four times more VSC. A significant amount of tongue coating was also reported in patients with bad breath and a healthy periodontium compared to subjects with non-halitosis and a healthy periodontium.

Halitosis and periodontal disease

The relationship between periodontal disease and IOH was clear from 1967 when an association was established between the inflamed periodontal pocket and the production of hydrogen sulphide. Various research studies have correlated VSC concentration to the number and depth of periodontal pockets. VSC concentration tends to increase with the severity of the periodontal disease. A study by Yaegaki and Sanada (1992) observed a higher concentration of methyl mercaptan and an increased methyl mercaptan/hydrogen sulphide ratio in subjects with pocket probing depth greater than or equal to 4mm. This higher concentration of methyl mercaptan can be dimerized into dimethyl sulphide and hence traces of dimethyl sulphide could be present in intra-oral halitosis. Also, VSC concentration increases proportionally with the gingival bleeding index, which indicates that the blood components might accelerate VSC production. However, the role of the periodontal pocket as a major source of VSC is a point of debate, because the pocket is considered to be a "sealed" environment with a smaller surface area, so not much gas escapes, and

it is therefore unlikely that the gases that do escape contribute extensively to intra-oral halitosis, with the exception of necrotising periodontal disease with ulcers on the surface of the gums. Methyl mercaptan is produced abundantly in periodontal disease, but the role of VSC in causing periodontitis is unclear. VSCs have the potential to alter the permeability of gingival tissues inducing an inflammatory response and modulating the function of gingival fibroblasts. They alter the permeability of intact mucosa and thus stimulate IL-1 cytokines which act synergistically with lipopolysaccharide of bacterial antigens to induce secretion of prostaglandin E2 and collagenase, which are important mediators of inflammation and tissue destruction. This concept of mucosal permeability might play a role in the transition from gingivitis to periodontitis.

Microbiology of IOH and VSC

Gram-negative, proteolytic, and Gram-positive bacteria have a role

in VSC production. The tongue is an important source of VSC and the tongue's indigenous flora is different from periodontal flora. One other factor is that the tongue acts as a carrier of periodontopathic bacteria in both periodontally healthy and diseased states. The composition of the IOH tongue coating in subjects with periodontal disease is different from that in those without periodontal disease. In subjects with both IOH and periodontal disease, the three important species isolated were *Treponema denticola*, *Porphyromonas gingivalis*, and *Tannerella forsythia*. A test called BANA (benzoyl-DL-arginine-2 naphthylamide) detects these species. Persson et al. (1989) isolated a list of periodontal bacterial species that produce hydrogen sulphide and methyl mercaptan *in-vitro*. In periodontally healthy subjects with IOH, the composition of the tongue coating was similar to that in those without IOH. However, differences were found in the individual levels of the bacteria. Therefore, an increase in bacterial numbers and their metabolites

is considered to play a role in the causation of IOH.

Diagnosis of intra-oral halitosis

The first step in diagnosing IOH is an inspection of the oral cavity, which involves examination of hard and soft tissue along with a thorough periodontal inspection. To assess bad breath, inspecting the coating of the tongue is an essential procedure. There are various visual indexes to evaluate the quantity of coating. The Winkel Tongue Coating Index (WTCI) is a widely accepted index in which the tongue is divided into six sections, three anterior and three posterior. The presence and absence of coating is scored on a scale from zero to two (0 = no coating, 1 = presence of coating, 2 = thick coating), so the total score from the six sections is a maximum of 12. There are no strict protocols for diagnosing halitosis. Based on scientific studies, the three main methods to diagnose halitosis are organoleptic measurements, sulphide monitoring, and gas chromatography (see figure 2).

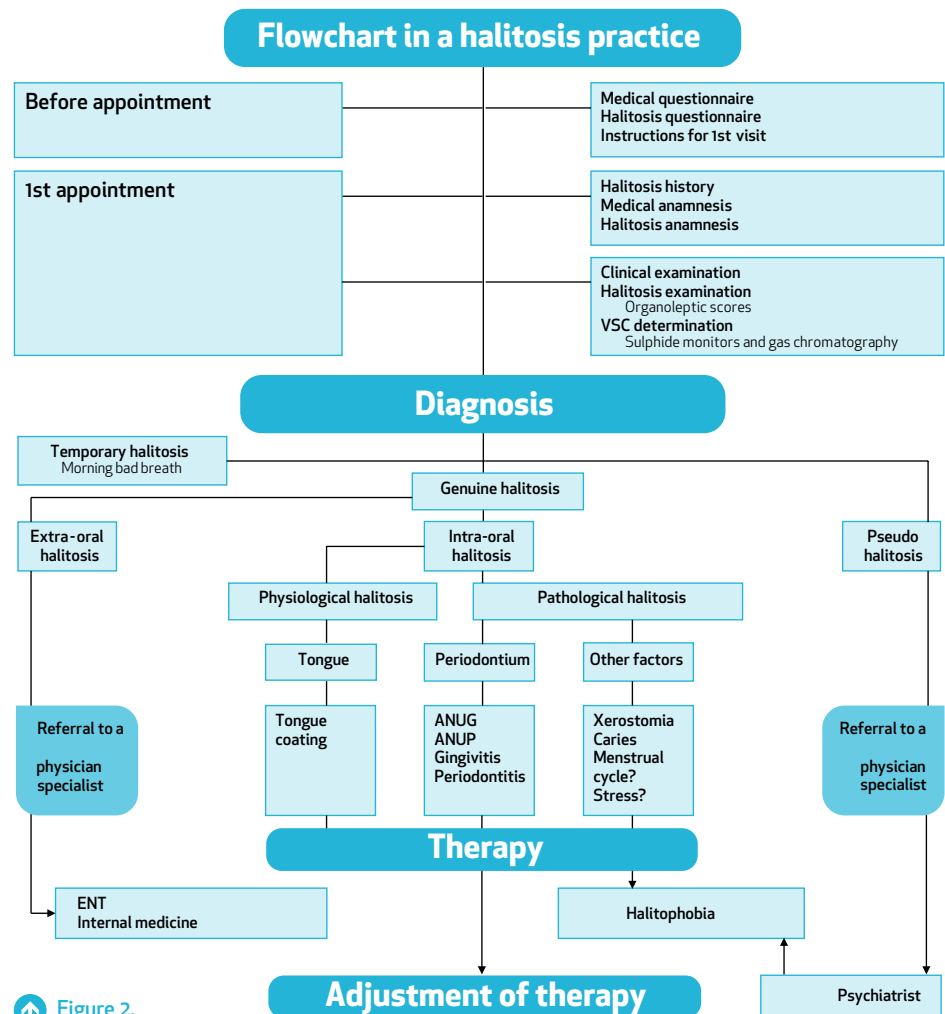


Figure 2.

a. Organoleptic assessment:

This is a subjective method, yet a “gold standard” in halitosis practice. This method is carried out by an examiner who sniffs the air exhaled from the patient’s nose and mouth. The comparison of nose and mouth breath helps to differentiate intra- and extra-oral halitosis. Organoleptic mouth-breath evaluation (figure 1) is done by instructing the patient to close their mouth for one minute and asking them to exhale slowly about 10cm from the nose of the examiner. Similarly, organoleptic nose breath is evaluated by asking the patient to exhale slowly from the nose. The odour assessment is based on a scale from zero to five.

b. Sulphide-monitor assessment:

Sulphide monitors such as Breathron, iSenLab, HaliSens, and the Halimeter assess the sulphide of the breath. For example, the Halimeter measures the total VSC content of the breath and is highly sensitive to hydrogen sulphide. However, it lacks sensitivity to methyl mercaptan, which is an important contributor to halitosis in patients with periodontal disease. And it cannot measure dimethyl sulphide, so it is not suitable for diagnosing extra-oral halitosis. Before every measurement, the Halimeter needs to be calibrated to zero based on ambient air. A disposable plastic straw is placed at the entrance of the patient’s nose. When the patient slowly exhales from the nose, the peak value of VSCs is recorded. In order to measure VSC from the mouth, the patient is asked to close the mouth for

one minute and a disposable straw is placed on the posterior dorsal surface of the tongue with the tongue extended, and the straw is held until the peak VSC is recorded. The Halimeter records the VSC level in parts per billion (see figure 3).

c. Gas chromatography: Gas chromatography measures different volatile components of bad breath. OralChroma, a form of gas chromatography, is specifically designed to measure the three important VSC molecules of bad breath. The great advantage of this method is that it differentiates between intra- and extra-oral halitosis.

A 1ml plastic syringe is placed halfway inside the patient’s mouth and is held tightly by the lips for one minute while the plunger is completely into the syringe. After one minute, the plunger is pulled until 1ml of mouth air is collected. After removing from the oral cavity directly a needle is attached to the syringe. Then the plunger is pushed in until 0.5ml is left in the syringe. This 0.5ml is injected into the OralChroma device. The result is displayed digitally within minutes (figure 4).

Therapy for intra-oral halitosis

The therapy for intra-oral halitosis includes mechanical cleaning of the tongue coating and chemical reduction of the bacterial load. Mechanical cleansing is recommended in patients with tongue coating and can be carried out either using a tongue scraper or a toothbrush. The toothbrush is less effective in removing

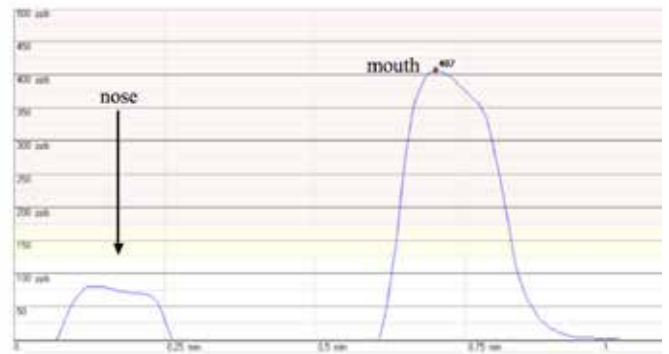


Figure 3: VSC reading from Halimeter in parts per billion.

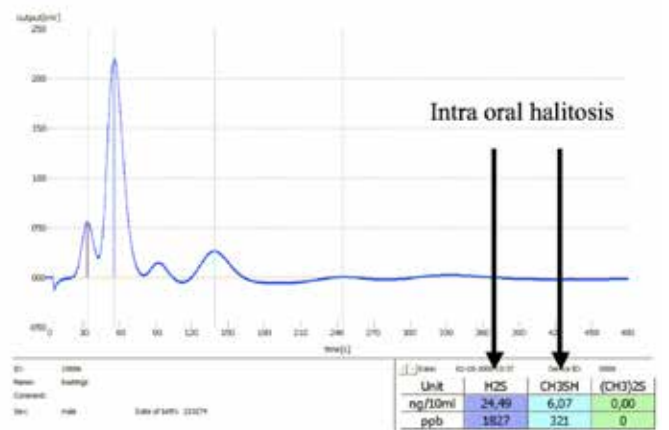


Figure 4: Reading from OralChroma in parts per billion.

the coating because of its height which creates more gag reflex while cleaning. As the coating is often deposited on the posterior tongue surface, cleaning the back of the tongue as far as possible is essential. The recommended regime for tongue scraping involves five gentle strokes from posterior to anterior until the coating is removed completely. Utmost care needs to be taken to avoid any

damage to soft tissues and especially to the circumvallate papillae. Tongue scraping has been shown to reduce the substrate for putrefaction rather than the bacterial load and to reduce the presence of volatile sulphur compounds by up to 75%. In addition, tongue scraping improves taste sensation, particularly the salt taste. These findings together suggest that tongue cleaning as an important procedure in maintaining proper oral hygiene (figure 5). In addition to tongue scraping, mouth rinses are prescribed whose active ingredients include antimicrobial agents such as Cetylpyridinium chloride, chlorhexidine, essential oils, chlorine dioxide, metal ions (such as zinc lactate/citrate and zinc chloride), triclosan, and hydrogen peroxide. The efficacy of these products on real intra-oral halitosis patients is not



Figure 5: Tongue coating (left) and tongue scraping (right).

always clear. Moreover, the long-term usage of mouth rinses results in taste loss and discoloration (teeth and tongue). For long-term usage, it is preferable that a mouth rinse does not contain alcohol. The recommended protocol for mouth rinse in cases of IOH is gargling (not rinsing) twice a day for one minute with the tongue out, so that the active ingredients can reach the pos-

terior dorsal surface. Intra-oral halitosis with the presence of periodontitis can be treated with scaling and root planing, and periodontal surgery, along with the use of a mouth rinse. IOH treated with periodontal therapy alone has been shown to result in the minimal suppression of VSC whereas the combined effect of periodontal therapy and mouth rinses

has proven to be much more effective in treating IOH.

More research needed

Intra-oral halitosis is manageable. Successful treatment requires proper diagnosis. Since intra-oral halitosis originates in the oral cavity, oral-healthcare professionals must be knowledgeable about the diagnosis and ther-

apy of intra-oral halitosis. The increased prevalence of halitosis demands more research and the International Association for Halitosis Research (www.iafhr.org) is working towards raising public awareness, enhancing information, and providing treatment advice to professionals in both the dental and medical fields.

Bibliography

Bosy A, Kulkarni GV, Rosenberg M, McCulloch CA (1994). Relationship of oral malodour to periodontitis: evidence of independence in discrete subpopulations *J Periodontol* 65:37-46.

De Geest S, Laleman I, Teughels W, Christel Dekeyser, Quirynen M (2016). Periodontal diseases as a source of halitosis: a review of the evidence and treatment approaches for dentists and dental hygienists. *Periodontol* 2000, 71:213-227.

Persson S, Claesson R, Carlsson J (1989). The capacity of subgingival microbiota to produce volatile sulfur compounds in human serum. *Oral Microbial Immunol* 4:169-172.

Pol A, et al. (2018) Mutations in SELENBP1, encoding a novel human methanethiol oxidase, cause extraoral halitosis. *Nat. Genet.* 50:120-129.

Quirynen M, Avontroodt P, Soers C, et al. (2004) Impact of tongue cleansers on microbial load and taste *J Clin Periodontol* 31:506-510.

Quirynen M, Dadamio J, Van den Velde S, De Smit M, Dekeyser C, Van Tornout M and Vandekerckhove B (2009). Characteristics of 2000 patients who visited halitosis clinic *J. Clin. Periodontol.* 36:970-975.

Ratcliff PA and Johnson PW (1999) The relationship between oral malodour, gingivitis and periodontitis, a review *J. Periodontol* 70:485-489.

Seeman R, Conceicao MD, Filippi A, Greenman J, Lenton P, Nachnani S,

Quirynen M, Roldán S, Schulze H, Sterer N, Tangerman A, Winkel EG, Yaegaki K and Rosenberg M (2014). Halitosis management by the general dental practitioner—results of an international consensus workshop. *J. Breath Res.* 8 017101.

Seerangaiyan K, Jüch F, Atefeh F, Winkel EG (2018). Tongue cleaning increases the perceived intensity of salty taste. *J Nutr Health Aging* 22:802-804.

Seerangaiyan K, van Winkelhoff AJ, Harmsen HJM, Rossen JWA and Winkel EG (2017). The tongue microbiome in healthy subjects and patients with intra-oral halitosis. *J. Breath Res* 11 36010.

Silva MF, Leite FRM, Ferreira LB, Pola NM, Scannapieco FA, Demarco FF, Nascimento GG 2018 Estimated prevalence of halitosis: a systematic review and meta-regression analysis *Clin Oral Investig* 22:47-55.

Tangerman A (2002) Halitosis in medicine: a review. *Int Dent J* 52:201-206.

Van Tornout M, Dedamio J, Coucke W, Quirynen M (2013). Tongue coating: related factors *J Clin Periodontol* 40:180-185.

Winkel E.G. (2008) "Halitosis Control" in Jan Lindhe, Thorkild Karring, Niklaus P. Lang (editors) *Clinical Periodontology and Implant Dentistry*, 5th Edition, Blackwell Munksgaard. Chapter 60, 1325-1340.

Yaegaki K and Sanada K (1992) Biochemical and clinical factors influencing oral malodour in periodontal patients. *J Periodontol* 63:783-789.



Kavitha Seerangaiyan practises as a dentist in Tamil Nadu, India. She received her degree in dentistry from M.G.R Medical University, India, in 2008, and her PhD on "tongue coating: its impact on intra-oral halitosis and taste" from the University of Groningen, the Netherlands, in 2018 under the guidance of Prof. Edwin Winkel. In her

thesis work, she pioneered the multi-omics (next-generation sequencing) technique to identify potential tongue microbiome- and microbial-derived compounds in the formation of tongue coating in subjects with intra-oral halitosis. After completing her PhD, Kavitha worked as a postdoctoral scientist at the University of Miami, USA, on microbial cross-talk between the gut and the oral cavity. She is a member of International Association for Halitosis Research and delivered a scientific talk at the EuroPerio 9 congress in Amsterdam in 2018.

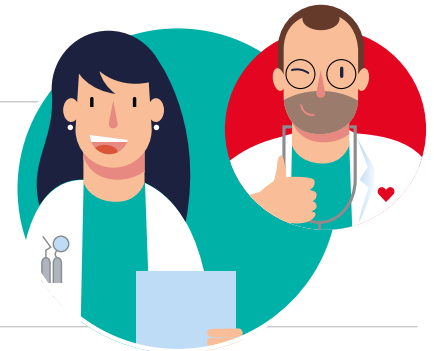


Edwin Winkel was a professor of periodontology at the University of Groningen, the Netherlands, until 2019. From 1982 till 2002 he worked in the Department of Periodontology at the Academic Centre for Dentistry Amsterdam and was involved in a longitudinal epidemiological study (15-year follow-up) among Indonesians with no regular dental care (the Java Study).

A specialist in periodontology and implants, he also started the first halitosis clinic in the Netherlands in 1997. He has published widely on topics including the development of clinical microbiology, the effects of systemic antimicrobial therapies on periodontitis, peri-implantitis patients, and the diagnosis and therapy of halitosis. He is a former chair of the Dutch Society of Periodontology and served on the EFP's executive committee as treasurer and chair of the external affairs committee. In 2016, he received the EFP's Distinguished Service Award.



'Periodontists and cardiologists should work together'



The Perio & Cardio campaign was launched by the EFP and the World Heart Federation (WHF) in September. The initiative, sponsored by Dentaid, offers clear advice, based on the latest scientific evidence, about the significance of the associations between periodontal and cardiovascular diseases and about the steps that should be taken in terms of prevention and treatment. Spanish cardiologist Álvaro Marco del Castillo talks about how periodontists and cardiologists should work together to improve outcomes for their patients.

How would you sum up the latest research on the associations between periodontal and cardiovascular diseases?

There is clear scientific evidence of associations between periodontitis and cardiovascular disease (CVD), including increased risk of acute myocardial infarction, heart failure, and stroke. In patients with CVD, periodontal treatment and good oral-hygiene habits may reduce the incidence of acute CVD events and patients with CVD should certainly receive a thorough oral-health examination. There are common lifestyle factors – such as smoking, stress, obesity, diabetes, and an unhealthy diet – which aggravate both periodontitis and CVD. Finally, the early diagnosis, prevention, and co-management by dentists and physicians of both cardiovascular disease and periodontitis is of the utmost importance.

How do cardiologists view the idea that dentists want to be more involved in the

systemic management of their patients, given the volume of evidence on the links between periodontal and systemic diseases?

We have studies that show how treating systemic inflammation can improve the overall health of these patients and reduce the burden of cardiovascular events during follow-up. So, if we reduce systemic inflammation by treating periodontitis, we can probably help these patients because it has been shown that periodontitis generates this kind of systemic inflammation.

I think we need to look closer at this and start collaborating between each other. I see a lot of interest from the perio side, but I don't see the same level of interest from our side, because we are so focused on other things that we are not paying attention to it. Over the last 20 to 25 years, medicine has been all about subspecialisation and we probably need to take a few steps back and look at holistic health and how different organs relate to each other.

As cardiologists, we have been extremely focused on just one organ and we have omitted the rest of the body. Cardiologists don't routinely check for systemic inflammation before treating cardiovascular diseases, and that's an important mistake. We treat cardiovascular diseases and forget about everything else. Sometimes we don't even follow up some risk factors that we should check.

How can periodontists help their cardiologist colleagues?

You can help us learn about periodontitis – how can I assess it in my clinic? Should I regularly check the gums of my patients or should I send them to you? And another thing that would be good for both of us would be to establish protocols of collaboration: when patients need to see dentists and when patients should see the cardiologist – when should each one be referred.

One key issue for periodontists when treating patients with cardiovascular disease concerns whether anti-platelet or anti-coagulation therapy needs to be suspended temporarily ahead of surgery to prevent the risk of bleeding. Current guidelines – such as those of the UK's National Institute for Health and Care Excellence (NICE) – suggest not suspending this treatment. What's your opinion?

Whenever they are on two medications, I would really encourage you to be careful because the risk of bleeding increases exponentially. They should be referred to the cardiologist just to make sure

they need them, because most of the time they only need one of them.

Does periodontal surgery expose cardiovascular patients to an increased risk of cardiovascular events?

The problem is spikes of systemic inflammation, which are extremely dangerous, especially if we know that there are some unstable atherosclerosis plaques going on – for example, a recent myocardial infarction. If you want to treat a patient for periodontitis in the 12 months after the myocardial infarction, he's going to be on two antiplatelet therapies and – even though he can undergo treatment – you don't really want that spike of acute inflammation.

When performing periodontal surgery on patients with cardiovascular disease, clinicians should be aware of signs of discomfort and stress, because being nervous increases the heart rate and raises the blood pressure. Those two factors combined in a patient with a cardiovascular disease can sometimes lead to fatal consequences. In longer surgical procedures, periodontists might check the patient's blood pressure every 30 minutes and give a mild sedative if it rises above 160-180.

There are some questions that a periodontist could ask patients with cardiovascular disease ahead of surgery, such as whether they feel pain in their chest or legs, if they have trouble breathing, or if they have palpitations and an irregular pulse. There is a question that anaesthesiologists do in



↑ Periodontists and cardiologists at the Perio-Cardio Workshop.

their clinics which is: can you go up one flight of stairs? If someone can go up a flight of stairs without any chest pain or any extreme exhaustion it means their cardiovascular fitness should tolerate almost any intervention.

The Perio & Cardio campaign was launched in September and features

recommendations for dentists, doctors, and patients as well as infographics and a video animation. How should dentists and cardiologists use this material?

From our side, we cardiologists have to study more – how to recognise the early signs of periodontitis so we can refer

patients to periodontists. It is also important to use all that knowledge to start co-operating – maybe establishing a liaison between cardiac and dental clinics to create early referral transfers or protocols. Adding periodontal care to what we call cardiac rehabilitation, along with instructions on how to eat and how to exercise, would be useful.

How many lives could all this save?

I don't know how many lives we would save by improving gum health in patients with cardiovascular disease. Imagine we save only five lives, is it less important? No. They need to take care of their mouth anyway. So, let's just go with it – if it's five, good; if it's 2,000 or 10,000 even better.

This article is based on the EFP Perio Talks conversation between Álvaro Marco del Castillo and Filippo Graziani (chair of the EFP's European project committee and former federation president) which took place on 14 September. EFP Perio Talks is a series of monthly live educational sessions on the EFP's Instagram page (@perioeurope) in which leading periodontists discuss key topics in periodontal science and clinical practice.



Dr Álvaro Marco del Castillo studied Medicine at the Complutense Universidad of Madrid, where he graduated in 2013. He started his career in cardiology with a residency at the University Hospital Ramon y Cajal in Madrid and completed his training in Calgary, Canada. He currently works as an attending physician at University Hospital 12 de Octubre, one of the most important health centres in Madrid, where he shares his time between the areas of cardiac arrhythmias and clinical research. Dr. Marco del Castillo has authored more than 30 book chapters in leading publications of his field, and has also published several scientific papers, focused on acute cardiac care and valve heart diseases.

Latest EFP campaign focuses on healthy gums for a healthy heart

The **Perio & Cardio campaign**, launched by the EFP and the World Heart Federation (WHF) in September, is based on the latest scientific evidence about the significance of the associations between the two diseases and about the steps that should be taken in terms of prevention and treatment.



The campaign, made possible thanks to an unrestricted grant from Dentaid, includes recommendation documents designed specifically for three groups of people: oral-healthcare professionals, medical professionals and pharmacists, and patients and the public.

It also includes infographics and a video animation, as well as the consensus report of the Perio-Cardio Workshop, upon which all the materials are based. At this workshop – held in Madrid in February 2019 and organised jointly by the EFP and the WHF, with sponsorship from Dentaid – 20 experts reviewed the latest scientific evidence on the links between periodontitis and cardiovascular diseases.

The campaign has translated the findings of the workshop into accessible and lively formats. It was created by the EFP's communications team working with expert advisers Filippo Graziani (chair of the EFP European Projects Committee) and Henrik Dommisch, Hady Haririan, and Paula Matasanz (the committee's junior officers).

The key messages of the Perio & Cardio campaign are:

- There is scientific evidence of associations between periodontitis and cardiovascular disease (CVD), including increased risk of acute myocardial infarction, heart failure, and stroke.
- In patients with CVD, periodontal treatment and good oral-hygiene habits may reduce the incidence of acute CVD events. Patients with CVD should receive a thorough oral-health examination.
- Common lifestyle factors – such as smoking, stress, obesity, diabetes, and an unhealthy diet – aggravate both periodontitis and CVD.
- The early diagnosis, prevention, and co-management (by dentists and physicians) of both cardiovascular disease and periodontitis is of the utmost importance.

The *Recommendations for the oral-healthcare team* emphasise the need to compile detailed patient histories to assess CVD risk factors, inform them of any CVD risk, and tell them to consult their doctors if any of these factors is not controlled.

Dentists and other oral-healthcare professionals are advised to inform patients with periodontitis of their higher risk of suffering CVD, provide them with a tailored oral-hygiene regime, and encourage them to address lifestyle factors that increase the risk of both diseases.

Patients with CVD should be given a thorough oral examination and placed on a preventive care regime if no periodontitis is diagnosed or a treatment regime if periodontitis is found.

The *Recommendations for medical professionals and pharmacists* state that patients with CVD should be advised that periodontitis may worsen their disease and increase the risk of CVD events, and that periodontal therapy may have a positive impact on their cardiovascular health.

Doctors should also ask patients with CVD about signs and symptoms of periodontitis (such as bleeding gums and loose teeth) and, where appropriate, recommended a periodontal evaluation. If patients have been diagnosed with periodontitis, doctors should investigate if appropriate periodontal care and maintenance are being provided.

The *Recommendations for patients and the public* emphasise good oral hygiene at home and there are also clear instructions on how to recognise gum disease and the lifestyle risk factors for both periodontitis and cardiovascular disease.

Student symposium on EFP Guideline

The EFP's *Treatment of Stage I-III Periodontitis: The EFP S3-level Clinical Practice Guideline* was the focus on an online symposium on September 17 involving the 15 universities that teach the federation's accredited postgraduate programmes in periodontology and implant dentistry. Students gave presentations explaining the guideline's recommendations and how they are being applied in their faculties, with presentations organized around its four steps of therapy.

Step 1

Rebecca Kirana (ACTA, Amsterdam, Netherlands) outlined the first step of therapy, which aims to provide periodontitis patients with tools to facilitate compliance and ensure adequate outcomes. This step focuses on patient and professional supragingival plaque control and risk-factor control.

Step 2

Juan Khoury (Rambam Health Care Campus, Haifa, Israel) outlined the professional control of subgingival biofilm and calculus using subgingival instrumentation, seeking to achieve no periodontal pockets greater than 4mm and an absence of bleeding on probing. Eric Schmid (University of Bern, Switzerland) discussed the adjunctive use of lasers and antimicrobial photodynamic therapy, noting the Guideline's recommendation that dentists are suggested not to use these approaches. Guilhem Jolivet (University of Strasbourg, France) focused on adjunctive host-modulating agents such as probiotics, statins, and sub-antimicrobial-dose doxycycline whose use is also not endorsed by the Guideline. David Naughton (Dublin Dental University, Ireland) addressed chemotherapeutic adjunctive rinses (home care), professional locally administered subgingival antiseptics and antibiotics. Manoetjer Siawasch (Catholic University of Leuven, Belgium) put the spotlight

on systemic antimicrobials, discussing how they provide a significant clinical benefit beyond reductions in periodontal probing depth and improvements in clinical attachment levels and can reduce the need for periodontal surgery. But antimicrobial resistance and adverse events make the use of systemic antimicrobials "difficult, controversial, and still unclear."

Step 3

Giacomo Baima (University of Turin, Italy) focused on general recommendations for surgical procedures, noting that the Guideline states that surgical treatment is effective but frequently complex and should be performed by specialists or dentists with additional training, and that efforts should be made to improve patient access to this level of care. He discussed the use of patient stratification, involving a tailored approach to treatment. Mario Romandini (Complutense University of Madrid, Spain) explored three options for step 3: (1) repeat step 2 (subgingival instrumentation), (2) various access-flap procedures, (3) resective flap procedures. Factors to take into consideration include whether there is an intrabony defect, class II or III furcation involvement, aesthetic factors, or a need to modify soft or hard tissues. Oded Heyman (Hebrew University-Hadassah Medical Centre, Jerusalem, Israel) looked at regenerative



One of the student presentations at the symposium.

surgery versus access-flap surgery in treating intrabony periodontal defects, discussing when to use different regenerative materials such as enamel matrix derivative and the various types of barrier membrane and bone-fill materials. Camilo Torres (International University of Catalonia, Barcelona, Spain) assessed mandibular furcation, discussing the benefits of resective surgery for the treatment of furcation involvement and explaining that tooth retention after complex periodontal therapy of teeth with furcation involvement presents reasonable survival rates and is economically more cost-effective than extraction and replacement with an implant-supported prosthesis. Samantha R. Uy (University of Hong Kong) discussed maxillary furcations, exploring non-surgical furcation therapy, tunnelling, surgical resective therapy, guided tissue regeneration, and grafting.

Step 4

Yaman Altaep (University of Liège, Belgium) focused on supportive periodontal care (SPC) and the key question of how often to schedule periodontal-maintenance visits. He discussed the importance of an SPC programme customised to patient needs and addressing behavioural chang-

es. Mohammed Alqarzaee (Eastman Dental Institute, London, UK) discussed home care and recommendations for repeated individually tailored instructions in mechanical oral hygiene, considering patients' needs and preferences, and interdental brushes rather than flossing as the first choice for interdental cleaning. Eirini Chatzopoulou (University of Paris, France) summarised adjunctive therapies for gingival inflammation, which the Guideline says may be considered in specific cases as part of a personalised treatment approach. Deniz Findik Balci (Yeditepe University, Istanbul, Turkey) explored professional interventions, discussing supragingival dental biofilm control including the use of lasers, adjunctive methods such as sub-antimicrobial-dose doxycycline, photodynamic therapy. In addition, the importance of risk factor control during SPC was evaluated. The symposium concluded with a lively question-and-answer session in which students' queries were addressed by Moshe Goldstein, (chair of the EFP postgraduate committee), David Herrera (chair, workshop committee) and Mariano Sanz (co-chair, workshop committee).



David Herrera and Moshe Goldstein during the symposium Q&A session.

Latest research

Journal of Clinical Periodontology

Self-reported bleeding on brushing as a predictor of bleeding on probing: Early observations from the deployment of an internet-of-things network of intelligent power-driven toothbrushes in a supportive periodontal care population

Bleeding on brushing (BoB) is an important sign of gingival inflammation. Intelligent toothbrushes and oral-health applications have shown potential to improve oral and periodontal health. This study sought to audit the adoption and retention of this new technology and to assess the feasibility of gathering data on BoB and associate them with clinical periodontal parameters.

A hundred subjects with different periodontal case diagnoses participating in supportive periodontal care (SPC) were provided with and instructed on using an intelligent power-driven toothbrush connected to an app (I-Brush). Brushing sessions and occurrence of BoB were recorded through the app.

Compared with baseline, subjects' oral hygiene, bleeding on probing, and prevalence of residual pockets improved gradually while using the I-Brush. The number of BoB episodes in the two weeks leading to the SPC appointment and the number of residual pockets predicted bleeding on probing ($p < .001$) detected during the examination. App use in the previous two weeks was associated with lower plaque scores at SPC.

These preliminary observations indicate good adoption and retention of a mobile health system built around an intelligent power toothbrush in a SPC population. Deployment of mobile health applications seems feasible in dental practice and may bring significant oral-health benefits. More investigations are needed in this area.

Authors: Maurizio S. Tonetti, Ke Deng, Allis Christiansen, Katja Bogetti, Chiara Nicora, Susanne Thurnay, Pierpaolo Cortellini

Full article: <https://doi.org/10.1111/jcpe.13351>

Tunnel technique with connective tissue graft versus coronally advanced flap with enamel matrix derivate for root coverage: Two-year results of an RCT using 3D digital measuring for volumetric comparison of gingival dimensions

The aim of this randomised clinical trial was to compare clinical and volumetric outcomes of the tunnel technique (TUN) with a subepithelial connective tissue graft (CTG) versus coronally advanced flap (CAF) with enamel matrix derivate (EMD) two years after treatment for gingival recession.

Twenty-three patients contributed 45 Miller class I or II gingival recessions. At baseline and follow-up examinations, study models were collected. Their three-dimensional scans allowed precise computer-assisted measurement of recession depth (REC), complete root coverage (CRC), percentage of root coverage

(RC), pointwise (pTHK) and mean areal (aTHK) marginal soft-tissue thickness. Clinical examination delivered probing depths and height of keratinized tissue.

Two years after surgery, digitally evaluated CRC was present in 60% of the TUN + CTG and 0% of the CAF + EMD-treated sites ($p < .0001$), meaning a certain relapse of the gingival margin with both approaches. RC amounted to 94.0% (TUN + CTG) and 57.3% (CAF + EMD), respectively ($p < .0001$). REC reduction (RECCred) was significantly higher for TUN + CTG, as were pTHK and aTHK values. Statistical analysis detected positive correlations between THK and both RC and RECCred.

Two years post-operatively, CTG showed better clinical and volumetric outcomes than EMD. Increased THK values were associated with improved outcomes regarding RC and RECCred.

Authors: Otto Zuhr, Stephan F. Rebele, Kirstin Vach, Hari Petsos, Markus B. Hürzeler, on behalf of the Research Group for Oral Soft Tissue Biology & Wound Healing

Full article: <https://doi.org/10.1111/jcpe.13328>

Three periodontitis phenotypes: Bone loss patterns, antibiotic-surgical treatment, and the new classification

This study compared three periodontitis clusters (A, B, and C) for alveolar bone loss (ABL) patterns, antibiotic prescriptions, and surgeries and related them to the new classification of periodontitis.

ABL patterns, prescription of systemic antibiotics, and the number of surgeries were retrieved for all patients ($n = 353$) in the clusters. Comparisons and possible predictors for antibiotics were assessed and results also evaluated in relation to the new classification.

Cluster A is characterised by angular defects often affecting the first molars and localised stage III/IV grade C periodontitis. Cluster B contains mainly localised or generalised stage III/IV, grade C patients. Cluster C contains mainly patients with generalised stage III/IV grade C periodontitis. Patients in cluster A received significantly more antibiotics compared to those in clusters B and C (78% vs. 23% and 17%). The predictors for antibiotic prescription were young age and localised ABL. No differences in numbers of periodontal surgeries were observed between clusters.

Within stage III/IV, grade C periodontitis, three clusters of patients were detected. The distinct localized ABL pattern and younger age in cluster A presumably prompted clinicians to prescribe antibiotics.




Authors: Chryssa Delatola, Bruno G. Loos, Marja L. Laine

Full article: <https://doi.org/10.1111/jcpe.13356>







EFP full-member societies

-  **Austria** Österreichische Gesellschaft für Parodontologie
-  **Belgium** Société Belge de Parodontologie / Belgische Vereniging voor Parodontologie
-  **Croatia** Hrvatsko Parodontološko Društvo
-  **Czech Republic** Česká Parodontologická Společnost
-  **Denmark** Dansk Parodontologisk Selskab
-  **Finland** Suomen Hammaslääkärisseura Apollonia
-  **France** Société Française de Parodontologie et d'Implantologie Orale
-  **Germany** Deutsche Gesellschaft für Parodontologie
-  **Greece** Ελληνική Πериδοοντολογική Εταιρεία
-  **Hungary** Magyar Parodontológiai Társaság
-  **Ireland** Irish Society of Periodontology
-  **Israel** Israeli Society of Periodontology and Osseointegration
-  **Italy** Società Italiana di Parodontologia e Implantologia
-  **Lithuania** Lietuvos Periodontolog Draugija
-  **Netherlands** Nederlandse Vereniging voor Parodontologie
-  **Norway** Norsk periodontist forening
-  **Poland** Polskie Towarzystwo Periodontologiczne
-  **Portugal** Sociedade Portuguesa de Periodontologia e Implantantes
-  **Romania** Societatea de Parodontologie din Romania
-  **Serbia** Udruzenje Parodontologa Srbije
-  **Slovenia** Združenje za ustne bolezni, parodontologijo in stomatološko implantologijo
-  **Spain** Sociedad Española de Periodoncia y Osteointegración
-  **Sweden** Svensk förening för Parodontologi och Implantologi
-  **Switzerland** Société Suisse de Parodontologie / Schweizerisch Gesellschaft für Parodontologie / Società Svizzera di Parodontologia
-  **Turkey** Türk Periodontoloji Derneği
-  **United Kingdom** British Society of Periodontology

EFP associate-member societies

-  **Azerbaijan** Azərbaycan Parodontologiya Cəmiyyəti
-  **Georgia** Georgian Association of Periodontology
-  **Morocco** Société Marocaine de Parodontologie et d'Implantologie
-  **Russia** Российской Пародонтологической Ассоциации
-  **Ukraine** Асоціація лікарів-пародонтологів України

EFP international associate members

-  **Argentina** Sociedad Argentina de Periodontología
-  **Australia** Australian Society of Periodontology
-  **Brazil** Sociedade Brasileira de Periodontologia
-  **Lebanon** Lebanese Society of Periodontology
-  **Mexico** Asociación Mexicana de Periodontología
-  **Taiwan** Taiwan Academy of Periodontology