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# Impact of antiseptics on onestage, full-mouth disinfection

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### Letter to the editor

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#### Dear Editor,

We read with great interest the ongoing debate on optimization of the infection control phase of periodontal therapy (Apatzidou & Kinane 2004, Kinane 2005, Koshy et al. 2005, Wennström et al. 2005).

Periodontal breakdown primarily develops when the subgingival microbial load overrules the local and systemic host defence mechanisms. Several studies also indicated that the presence of periopathogens (persisting or reestablished after treatment) is associated with a sub-optimal clinical outcome of periodontal therapy (Renvert et al. 1996, Haffajee et al. 1997, Renvert et al. 1998, Socransky et al. 1998, Cugini et al. 2000). Most of these periopathogens are found throughout the oropharynx: the saliva, the mucosae, the tongue dorsum and the tonsils (Danser et al. 1994, 1996, Petit et al. 1994, von Troil-Linden et al. 1996, Beikler et al. 2004).

After mechanical debridement, the subgingival microbial load drops to 0.1% (Goodson et al. 1991, Maiden et al. 1991). However, already 1 week later, the periodontal pocket is re-colonized by approximately the same number of bacteria, fortunately with a less pathogenic composition (Harper & Robinson 1987, Wade et al. 1992). The origin of these bacteria is still a matter of debate. The multiplication of bacteria remaining within the pocket (Petersilka et al. 2002), within the junctional or pocket epithelium (Lamont & Yilmaz 2002) and/or the dentinal tubules (Adriaens et al. 1988, Giuliana et al. 1997), has often been considered as the major cause for this subgingival recolonization. The impact of the "supragingival" area on this early subgingival re-colonization was considered negligible. Hence, traditional approaches have focused on optimal subgingival instrumentation, besides proper supragingival plaque control.

Our group felt that more attention should be given to the re-colonization by bacteria from other areas. Translocation from untreated pockets during traditional, quadrant-based, therapy as well as from other intra-oral reservoirs of periodontal pathogens was proposed as a possible mechanism. A new treatment strategy based on a one-stage, fullmouth disinfection was introduced 10 years ago (Quirynen et al. 1995). It aimed to eradicate, or at least suppress. all periopathogens in a very short time span, from all their oro-pharyngeal habitats (mucous membranes, tongue, tonsils, saliva). As such, the re-colonization of the treated pockets by bacteria from untreated sites, called cross-contamination or intra-oral translocation, could indeed be delayed until a better healing of the pockets was achieved. A series of prospective studies confirmed this hypothesis (Quirynen et al. 1995, 1999, 2000, Bollen et al. 1996, Vandekerckhove et al. 1996, Bollen et al. 1998, Mongardini et al. 1999, De Soete et al. 2001).

The data of these studies seem to be in contrast (Kinane 2005) with three recently published papers on full-mouth approaches in the initial treatment of periodontitis (Apatzidou & Kinane 2004, Koshy et al. 2005, Wennstrom et al. 2005). This discrepancy can be explained by major differences in treatment protocol, especially from the perspective of a risk for microbial cross-contamination (Table 1). Indeed, differences in the use of chlorhexidine (mouth rinsing and/or disinfection of all niches), the time interval before completion of all quadrants, the absence of oral hygiene instructions prior to/during therapy, the recall frequency, the severity of the disease, and the amount of calculus before therapy all have an impact on the chances for cross-contamination in the respective studies.

Besides the Leuven group, only one other study used a entire mouth disinfection (Koshy et al. 2005) in order to reduce cross-contamination by periopathogens from the other intra-oral niches. However, compared with our studies a weaker antiseptic (0.05% chlorhexidine) was used and for a shorter period (4 weeks). The other two papers did not use any antiseptic at all.

The timing of oral hygiene instructions also varied considerably. In two studies (Koshy et al. 2005; Wennström et al. 2005), for example, all subjects received one to two oral hygiene instruction sessions (including the use of inter-dental cleaning aids), prior to the start of the study. This probably reduces the chances for cross-contamination. In the Leuven studies, designed as "proof of principle", the patients did not exert plaque control at the untreated sites, until after their subgingival debridement. For the control group, this means that inter-dental cleaning in the last quadrant was postponed until 6 weeks after the debridement of the first quadrant. This of course increased the chances for cross-contamination in the control group.

The 0.2% chlorhexidine rinsing during the healing phase after debridement

Table 1. Systematic overview of the experimental design of recent	v of the experimental desig		studies on the one-stage, full-mouth disinfection.	infection.		
Authors	Treatment	V	Antiseptics in Fm group(s)	\$)	Oral hygiene before or	FM versus standard therapy
	strategy	chairside	de	home	ummg sund	# III outcours at cura study
		Subgingival irrigation	# other oral niches			
Quirynen et al. (1995)	OSFM 2d vs. Q 2w	CHX 1%	CHX 0.2%	CHX 0.2% 2 m	No OH instructions before treatment pi s lower in	$\Delta$ PPD, $\Delta$ CAL and $\Delta$ BOP all s better for OSFM group
Mongardini et al. (1999)	OSFM <sub>2d</sub> vs. Q <sub>2w</sub>	CHX 1%	CHX 0.2%	CHX 0.2% 2m	OSFIM during inst 2 m No OH instructions before treatment pi s lower in	$\Delta$ PPD, $\Delta$ CAL and $\Delta$ BOP all s better for OSFM group
Apatzidou & Kinane 2004	Fm-SRP 1 d vs. Q 2 w	None	None	None	OSFM during first 2 m No OH instructions before treatment pi = for both	$\Delta$ PPD = , $\Delta$ BOP = , $\Delta$ RAL s more in Fm for deep
Wennstrom et al. (2005)	Fm-Ud 1h vs. Q 1w	None	None	None	groups OH instructions 3 w before treatment pi = for both	pockets $\Delta$ PPD = , $\Delta$ RAL = , $\Delta$ BOP = , treatment time: s
Koshy et al. 2005	Fm-Ud <sub>2h</sub> vs. Q <sub>1w</sub>	povidone iodine 1%	¢.	CHX 0.05% 1 month	groups OH instructions 3 w before treatment $pi = for both$ groups	shorter in Fm Δ PPD = , Δ CAL = , reduction percentage sites ≥5 mm: s more in Fm Δ BOD <sup>*</sup> s more in morvionment
Quirynen et al. 2005 (unpublished data)	OSFM 2d vs. Q 2w	CHX 1%	CHX 0.2%	CHX 0.2%, AmF-SnF, 2–8 m	No OH instructions before treatment pi s lower in OSFM during first 2 m	for Fm, treatment time: s shorter in Fm $\Delta$ PPD, $\Delta$ CAL and $\Delta$ BOP all s better for OSFM group with CHX as antiseptic
BOP, bleeding on probing; CHX, chlorhexidine; CAL, clinical attachment level; d; days; FW scaling and root planing; s, statistically significant; Ud, ultrasonic debridement; w, weeks.	X, chlorhexidine; CAL, clir atistically significant; Ud, u	ical attachment level; d; days ltrasonic debridement; w, we	s; FM, full-mouth; m, mon seks.	ths; OSFM, one-stage, full-me	BOP, bleeding on probing; CHX, chlorhexidine; CAL, clinical attachment level; d; days; FM, full-mouth; m, months; OSFM, one-stage, full-mouth disinfection; PPD, probing pocket depth; pi, plaque index; SRP, scaling and root planing; s, statistically significant; Ud, ultrasonic debridement; w, weeks.	ket depth; pi, plaque index; SRP,

probably contributed to the significantly better plaque scores for the one-stage, full-mouth disinfection groups in our studies when compared with the control groups. The higher plaque scores in the control group can, at least partially, be explained by the remaining plaque, calculus or infection in the untreated sites, acting as reservoir for the bacteria colonizing the root-planed teeth (Dahan et al. 2004, Rowshani et al. 2004). It is known that the microbial load in the saliva of periodontitis patients indeed drops significantly after completion of the debridement, and as such reduces the rate of the de novo, supragingival plaque formation (Dahan et al. 2004; Rowshani et al. 2004). This by itself implies a delayed subgingival re-colonization (Sekino et al. 2004). The significant impact of optimal plaque control or repeated supragingival professional cleaning on the outcome of periodontal therapy has been illustrated in several studies (Axelsson et al. 1991, Dahlen et al. 1992, al Yahfoufi et al. 1995, Hellstrom et al. 1996. Westfelt et al. 1998. Ximenez-Fyvie et al. 2000, Checchi et al. 2002). Thus, at least in patients with severe periodontitis, a one-stage, fullmouth approach will result in an immediate reduction of the microbial load and as such in a delayed de novo plaque formation.

The time schedule of the full-mouth debridement (Table 1) as proposed in Leuven was not respected in the abovementioned papers (Apatzidou & Kinane 2004, Koshy et al. 2005, Wennström et al. 2005). The root planing of the four quadrants in these studies had been completed within the same day, often within some hours, whereas for the Leuven studies, the interval between the first and second half of the mouth was always 24 h. The time interval of 24 h between the first and second bacteraemia (because of subgingival instrumentation) might be responsible for an enhanced immune response (eventually a Schwartzman reaction). This was proposed as a potential explanation for the success with this one-stage, full-mouth disinfection approach (Mongardini et al. 1999, Quirynen et al. 2000). This is solely a speculation as it has not been proven in studies designed to examine this aspect.

Although often forgotten (Kinane 2005), one should realize that two of these three studies (Apatzidou & Kinane 2004, Koshy et al. 2005), even demonstrated limited, but statistically signifi-

cant, advantages for the full-mouth approach. Koshy et al. (2005) reported a significant additional reduction in sites with pockets  $\geq 5$  mm, as well as a more dramatic reduction in bleeding upon probing for the full-mouth approach. The level of significance might partially be explained by differences (although not statistically significant) at intake. Apatzidou and Kinane (2004) observed, at the end of their study, a significant additional gain in attachment of nearly 1 mm in deep pockets of patients treated with the full-mouth protocol, but they warned for the fact that only a low number of sites were involved.

The one-stage, full-mouth approach may probably be especially useful in patients with higher risks for cross-contamination, such as patients with nonoptimal plaque control, severe periodontitis and especially with high amounts of plaque and calculus in the untreated areas. The use of an antiseptic seems essential in this aspect.

Taking all these aspects into consideration and knowing the economic benefits of a one-stage, full-mouth disinfection approach, (two instead of four appointments, thus less transportation, and a better understanding of the therapy by the patient (all other infectious diseases that the patient had so far had been treated in one global approach)), and realizing the fact that patients do appreciate a full-mouth approach, we still feel that this approach should be the treatment of choice.

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