Infection Control in Dentistry and Drug-Resistant Infectious Agents: A Burning Issue. Part 1

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Abstract

Using molecular biological methods and retrospective investigations, some outbreaks in dental settings have been proven to be caused by mainly blood-borne viruses and water-borne bacteria. Nowadays, drug-resistant bacteria seem further hazards taking into account the worldwide overuse of antibiotics in dentistry, the limited awareness on infection prevention guidelines, and the lapses and errors during infection prevention (reported in more detail in Part 2). We chose MRSA and VRE as markers since they are considered prioritized bacteria according antibiotic resistance threats. Antibiotic-resistant bacterial infections inside of dental settings are relevant, and we argue about some hazards in dentistry, including dedicated surgeries. MRSA has a key role for its colonization in patients and dental workers, presence on gloves, resistance (days-months on dry inanimate surfaces), the contamination of different clinical contact surfaces in dental settings, the ability of some strains to produce biofilm, and finally its estimated low infective dose. For better dental patient and healthcare personnel safety, we need evidence-based guidelines to improve education and training initiatives in surgery.

Keywords: dentistry, surgery, guidelines, infection control, antibiotic resistance, biofilm

1. Introduction

Dentistry seems to provide safe procedures for oral health care taking into account all adverse events (AEs). Nevertheless, death, injury, and malfunctions due to dental devices (DDs) increased from the MAUDE report in 2000–2012, and the endosseous implants were at the top of the DDs involved in AEs [1]. In the same period, the number of malpractice payments in dentistry increased by 12%, while those in other health professions fell [2]. Dental AEs, complaints, and claims seem to be relatively common in different countries [3]. About 4–17% of AEs are due to infection [4, 5]. The iatrogenic infectious risk in dentistry has not been quantified closely yet [6, 7], but recently, some outbreaks caused by infective agents, mainly blood-borne viruses and water-borne bacteria, have been documented in dental settings based on molecular biological assays and/or retrospective investigations [8–11].

Some evidence exists around the hazard due of antibiotic-resistant infectious agents (ARIAs) in dentistry. Fatal adverse events (FAEs) had been reported within
90 days after different instances of dental care [7]. In the last 50 years, FAEs caused by an infection have (a) increased while respiratory complications and bleeding are steady, and those caused by cardiovascular or related to anesthesia have decreased, (b) significant (12%), (c) mainly associated to dental surgery (implant surgery/placement, extractions (>6 erupted teeth or impacted tooth/teeth), surgical extractions, osseous surgery, sinus lift surgery, bone biopsy, orthognathic surgery), and (d) associated with much longer times until death compared with other causes of death [7]. A study on dental malpractice analyzed 4149 legal claims (both in and out of court) from the years of 2000 to 2010 in Spain [12]. About 2.7% of all AEs resulted in death, and 45% of them were caused by infection. In the absence of specific information reported in both papers [7, 12], we do not exclude the possible involvement or nonrecognition of ARIAs or the failure of proper drug treatment in those FAEs. Recently, in an interview, Davies stated that in 20 years time even minor surgeries could be fatal because of infections [13].

We consider that the following two reviews are important and indicative of the limitedness of data published up to 2011–2012 in dentistry [14, 15]. The first review on methicillin-resistant Staphylococcus aureus (MRSA) infection concluded that (1) transmission was ascertained during surgical interventions, particularly in surgical units and among head and neck cancer patients; (2) carriage rates among dental healthcare personnel (DHCP) were lower than those among other healthcare workers (HCWs); (3) carriage rates among adult patients were low, whereas among pedodontic and special care patients rates were higher than those found in the general population; and (4) MRSA had been detected in the environment of emergency, surgical units, and in dental hospitals [14]. In the second review, multi-resistant bacteria infections had been included among the main healthcare viral and bacterial infections in dentistry [15], but the transmission of Enterobacteriaceae and/or their resistant strains did not exist yet. The interest on Enterobacteriaceae is warranted since they are susceptible to only a few (if any) antibacterial drugs.

Here, we think it is important to update these conclusions in the light of the global, wide, and long-term abuse and misuse of antibiotics in dentistry and selective pressure on opportunistic bacteria by favoring potentially pathogenic strains [16, 17]. In addition, the limited awareness on infection prevention guidelines and lapses and errors during infection prevention according to Centers for Disease Control and Prevention (CDC) dental guidelines [6, 8–11] sustain the evidence of possible reservoirs of ARIAs in humans (patient, dental staff) and in the environment (clinical contact surfaces (CCSs), dental instruments, and dental unit water lines (DUWLs)) and possible hazards in surgical dental setting. Our approach is in line with the CDC recommendation, in which it states that “Preventing infections negates the need for antibiotic use in the first place, and scientific evidence shows that reducing antibiotic use in a single facility can reduce resistance in that facility” [18–20].

In addition, the cluster of above problems is important for risk management, since it is rationally “harmful” that opportunistic species and/or ARIAs were involved in implant failures [21, 22], periodontitis [23, 24], endodontic failures [25] and oral mucosal and deep infections [26].

Here, we discuss briefly main recent evidence and controversy on infections in dedicated dental and mainly in implant surgery, taking into account that many other aspects (i.e. surgery technique, geometry, materials, and surface of dental implants) have already been reviewed extensively [27–30]. Dental implant (DI) complications are a burning issue, since the current demand of DIs is high (20 DIs in Italy and 4 in the USA per year per million of inhabitants), mainly applied in private offices, and the global DI market size was estimated at 3.77 billion USD in 2016 growing at a compound annual growth rate (CAGR) of 7.7% over 2024 [31]. It is important to underline that the incidence of esthetic, technical and infective complications is still high in implantology, and the 5-year infective complication increased from 7.4 to
9.4% [32]. In general, the expected implant-associated infections and the outbreaks from opportunistic pathogens (Staphylococci, Enterococci, Pseudomonas, etc.) will always be more important. In addition, we have to take into account other factors linked to risk management such as the impact on reputation and finances, the loss of protection of insurance coverings and reimbursements, and shocking advertising rapidly spreading through social networks in the case of outbreaks [8–11, 33–35].

Here (Part 1), we focus on the insufficient compliance with infection control (IC) recommendations in oral healthcare and the difficulties and problems of standard precaution implementation also in ambulatory surgical centers [6, 36–42]. In general, dental surgery and implantology are predominantly done in general dental practice under local anesthesia or sedation [43, 44]. This is a very important aspect since the cross-infection is widespread and more difficult to control compared to surgical rooms. We have divided problems and difficulties for infection prevention into different areas concerning the innovative molecular biology techniques; antibiotic misuse and overuse in dentistry; opportunistic pathogens and antibiotic resistance in dental patients and dental healthcare workers; and surgical infection prevention in dentistry. While in the chapter (Part 2), we have reported infection control implementation, not compliance, lapses and errors during infection prevention according to CDC dental guidelines. We focused on hand hygiene, gloves, environment decontamination, and instrument reconditioning in more detail [6, 43–47].

2. Approach

The electronic literature search was conducted via the PubMed and Google Scholar databases (from January 2010 up to and including April 2018) using various combinations of the following key indexing terms: (a) patient safety; (b) infection control; (c) implant; (d) endodontia; (e) sterilization; (f) reconditioning; (g) critical items; (h) semicritical items; (i) hand hygiene; (j) DUWL; (k) sharps safety; (l) personal protective equipment (PPE); (m) disinfection; (n) MRSA; (o) VRE; (p) ARIAs; (q) guidelines; and (r) cross-infection. In addition, manual searches were carried out in INTECH books. Then, bibliographic material from the papers has been used in order to find other or older appropriate sources. A total of 179 papers and links were found suitable for inclusion in this chapter (Part 1). Only few papers do not have a DOI or PubMed classification, but the available link by Internet and accessed date have been added.

3. Focus on molecular biology techniques

Expanded Human Oral Microbiome Database (eHOMD) provides the scientific community with broad and up-to-date information on the bacterial species present in the human aero-digestive tract, including the oral cavity. Genomes for 482 taxa (63% of all taxa, 89% of cultivated taxa) are currently available on eHOMD [48]. Fast and very sensitive molecular biological techniques, classified into nucleic acid-based methods [quantitative real-time polymerase chain reaction (PCR), multiplex PCR, microarray, next-generation sequencing technologies, etc.], are available for the screening, detection, and functional activities of pathogens and antibiotic-resistant bacteria, even those not cultivable by classical microbiological methods and by using both patient biological fluids and samples from inanimate objects (surface, air, DUWL colonization, DDs, and instruments) [49–52]. This is possible because DNA molecules can survive for long time and can be amplified. Current microbiological laboratory approaches based on high-throughput real-time
PCR allow quick, easy, and cheap detection of the oral microbiome and the anti-
bacterial resistome, throughout 300 antibiotic resistance genes [53] as far as the rapid
diagnosis of virulent slime-producing strains associated with dental caries [54]. The
specificity of MRSA plus MSSA carriage detected with Xpert MRSA is better than
standard culturing techniques, being 37.9 vs. 23.6%, respectively [55]. Concerning
microbiological features of peri-implantitis cases, culture methods were able to
detect 81.4% of the targeted species of the cases, whereas “checkerboard DNA–
DNA hybridization” method 99.3%. In relation to the limited association between
the bacterial contamination and the severity of the peri-implantitis [56], it is
decisive the sampling procedure, around DIs and during swabs on dental items, and
the use of the proper primer sequence for specific genes in different strains (i.e. ica
genes for *S. epidermidis* and *S. aureus*) [57]. PCR is more effective in detecting *E.
faecalis* than other analytical tools, such as culturing. *E. faecalis* has been found in
root-filled teeth associated with periradicular lesions in a range of 0–70% by culture
and 0–90% by PCR [58].

It is important to note that microbiological analysis (by culture or DNA-based
methods) is rarely used in dentistry mainly because of the difficulties to delay the
antibiotic treatment and for the plethora of infective agents involved in inflam-
matory diseases in dentistry. In addition, specific sampling procedures are needed
since the virulence features of microorganisms and problems to sample deep
periodontal and peri-implant pocket and abscesses. Sequencing methods that
evaluate the entire microbiome are needed to improve identification of microorgan-
isms (pathogen, opportunistic, noncultivable, drug-resistant ones) associated to
peri-implant infective diseases and to develop suitable countermeasures with the
expertise of clinical oral microbiologists [59]. In addition, emerging approach based
on optical nanoprobes, biosensors, and protein biomarkers suitable for peri-implant
crevicular fluids has been proposed to identify the severity and progression of the
disease and the response to therapy [60, 61].

4. The broad antibiotic misuse or overuse in dentistry

Globally, antibiotic prescription in dental care has continuously increased over
the last 17 years, and a lot of evidence has been published on wide antibiotic misuse
or overuse, in industrialized, low- and middle-income countries [62–70]. Dental
prescriptions make up 5–11% of all antibiotic prescription among patients in some
European countries, Canada, and the USA [19, 20, 65, 71, 72]. The rate of prescrip-
tion increased the most among dental patients of 60 years or above.

It is important to underline that antibiotic prescription is placed without a
microbiological analysis and has mainly prophylactic aim in dentistry. Recently, the
prescription of antibiotics in dentistry was reviewed by Holmstrup and Klausen,
while the use of antibiotics in odontogenic infections, in addition to the removal of
the source of infection, by Martins [73, 74]. A significant percentage (19–37.5%) of
microorganisms collected from their patients were penicillin resistant; neverthe-
less, the relationship between the clinical outcomes and microbial resistance with
penicillin is not clear [74].

Recently, to overcome the misuse and abuse of antibiotics in dentistry, different
institutions and associations recommended a more restrictive antibiotic policy to
improve treatment efficacy and decrease bacterial resistance. Specific guidelines
have been published for implantology [17], endodontia [75], oral surgery [17], third
molar extraction [76], and medically compromised patients [77] and to prevent
infective endocarditis [78, 79] or prosthetic joint infections [80].
5. Focus on opportunistic pathogens and antibiotic resistance in dental patients and dental healthcare workers

Here, based on recent and current knowledge, we focus on two well-known bacterial strains, *S. aureus* and *Enterococcus*, and their resistant strains. It is known that *S. aureus* and *Enterococcus faecalis* have been implicated in implant-associated infections [21, 23, 24, 81], endodontic infections [22, 25, 82], and recently in an outbreak of *Enterococcus* endocarditis [11]. We focus on these Gram-positive bacteria for the high innate resistance or ability to become resistant to most antibiotics along with some other virulence factors (hydrophobicity, adherence to abiotic surfaces (including dental implant materials), biofilm formation, ability to growth also in anaerobic conditions) [83]. These features are important in the exploration of standard precaution failures since bacterial adherence on dental implants, collagen-based biomaterials, or many other inanimate objects is known to be linked with the presence of surface components with nonpolar/hydrophobic vs. polar/hydrophilic characteristics. In addition, we focus on Staphylococci and Enterobacteriaceae as markers since they are considered prioritized bacteria according to antibiotic resistance threats, and better knowledge is available on their virulence factors and for dental settings (i.e. contamination on hands and environments, etc.) [6, 43, 45–47].

5.1 *Staphylococcus aureus* and MRSA

Single dose of prophylactic antibiotics in healthy volunteers induces a significant selection of resistant strains among the dynamic and complex community of resident oral and gastrointestinal bacterial microflora and causes a large disturbance of oral niches [84, 85]. Approximately, one third of participants gained resistant viridans *Streptococci* against amoxicillin, clindamycin, and penicillin-V, while in *Prevotella* spp., there was approximately a 28% gain in resistance to all antibiotics tested. The disturbance could reduce host colonization resistance, select new pathogens, and lead to an overgrowth of resistant bacteria [86].

*S. aureus* lives as a commensal primarily in the anterior nares and/or throat of 20–70% of adults [87, 88]. Some of the strains develop multidrug resistance and are well known to be involved in hospital-acquired (HA) infections [89]. The following two reviews are important and indicative of the limitedness of data published up to 2011–2012 in dentistry [14, 15]. *S. aureus* was normally absent or its colonization was very low in oral biofilm and ecological oral niches as reported in older evidence or not considered as a topic [14, 43, 90]. More recent data show that the presence of *S. aureus* in the oral cavity is more frequent and, nowadays, is to be considered a member of the oral microbiota (Table 1) [15, 84, 91–105]. Recently, metaproteomic analysis of human salivary supernatant from healthy persons was able to identify peptides from 124 microbial species including *Staphylococcus* [85]. The majority of *S. aureus* strains, isolated from the oral cavity of Tunisian patients, were biofilm/slime producers and exhibited some important genes (i.e. *ica, fnb, cna*) associated to adhesion and virulence factors [106, 107]. *S. pneumoniae* and *S. aureus* are common commensals of the upper respiratory tract in children and adolescents [14, 100, 108]. This fact is relevant since orthodontic patients are mainly children and adolescents and the high genotypic expression of peculiar genes (*icaA/icaD*) is important for *S. aureus* in the colonization of orthodontic appliances [109]. Recently, RNA-Seq data permit the analysis of active transcripts, assigned to antibiotics and toxic compounds, of the supragingival dental plaque biofilm in healthy subjects [110]. The transcripts assigned to Acriflavin resistance complex (*AcrA* and *AcrB* genes) were prevalent,
| Study                  | References | Study population | Number of subjects | Study carried out (years): | Country | Sampling, specimen, assay                                                                                                                                                                                                 | S. aureus carriage (%) | MSSA carriage (%) | MRSA carriage (%) |
|-----------------------|------------|------------------|--------------------|---------------------------|---------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------|------------------|-------------------|
| Roberts et al. (2011) | [91]       | Dental students  | 61                 | #                         | USA     | Swab, anterior nose; §                                                                                                                                                                                                     | #                      | 21               | 21.3              |
| Martínez-Ruiz et al. (2014) | [92]       | Dental students  | 100                | #                         | Mexico  | Paired nasal and throat swabs; §                                                                                                                                                                                             | #                      | #                | 20                |
| Petti et al. (2015)   | [93]       | Dental students  | 157                | #                         | Italy   | Dry cotton swabs from the mouth, nose, and skin between fingers of the nondominant hand; §                                                                                                                                  | 15.3 (9.7–20.9), any site | #                | 0, any site       |
| Baek et al. (2016)    | [94]       | Dental students  | 159                | #                         | Korea   | Nasal samples; §                                                                                                                                                                                                          | #                      | #                | 3.1               |
| Hema et al. (2017)    | [95]       | Dental students  | 200                | #                         | India   | Swab, anterior nares; §                                                                                                                                                                                                     | #                      | #                | 24.5              |
| Zimmerli et al. (2009) | [96]      | 500 dental patients >18 years | 500    | 2006                      | Switzerland | Swab, anterior nares; §                                                                                                                                                                                                     | 42                     | 41.6             | 0.4               |
| McCormack et al. (2015) | [97]     | 10-year retrospective analysis of laboratory data | 1429 | 1998–2007  | UK | Perioral clinical specimens (no. 1986); §                                                                                                                                                                                   | 90                     | 10               |                   |
| Kabanova et al. (2017) | [98]     | Patients from 5 maxillofacial departments | 2920 | 2014                     | Belarus | Swabbing the area after the incision (no. 162); §                                                                                                                                                                          | 15–70                  | #                | 5.6–278           |
| Dulon et al. (2014), review | [99]   | HCW in non-outbreak settings | 21,289 subjects from 31 studies | # | # |                                                                                                         | #                      | #                | 1.1–5.4 from high quality studies |
| Study                        | References  | Study population                      | Number of subjects | Study carried out (years) | Country | Sampling, specimen, assay                                      | S. aureus carriage (%) | MSSA carriage (%) | MRSA carriage (%) |
|------------------------------|-------------|---------------------------------------|--------------------|--------------------------|---------|---------------------------------------------------------------|------------------------|-------------------|-------------------|
| Esposito et al. (2015)       | [100]       | Healthy subjects aged 6–17 years      | 497                | 2013                     | Italy   | Oropharyngeal and nasal swabs; multiplex real-time PCR       | #                      | 49.7 (6–9 years); 54.9 (10–14 years); 52.9 (15–17 years) | 3.5 (15–17 years) |
| Koukos et al. (2015)         | [101]       | Healthy patients                      | 154                | 2010–2014                | Greece  | Subgingival samples and PCR assay                             | 10                     | #                 | 0                 |
| Kharialla et al. (2017)      | [102]       | Patient and DHCP                      | #                  | 2013                     | Egypt   | Swab, anterior nares, (no. 1300); culture plus molecular typing | 8.6                    | #                 | 11.1 patients; 6.7 nurses; 9.3 dentists |
| Yoo et al. (2018)            | [103]       | DHCP                                  | 139                |                          | Korea   | Swab, anterior nares; §                                      | #                      | #                 | 2.9               |

$: using microbiological culture methods; PCR: polymerase chain reaction; #: not indicated.

Table 1.
Staphylococcus aureus, methicillin-sensitive Staphylococcus aureus (MSSA), and methicillin-resistant Staphylococcus aureus (MRSA) carriage rates among dental students, dental patients, dental healthcare personnel (DHCP), and healthcare workers (HCWs).
while those encoding for putative macrolide-specific efflux system or proteins involved in acid stress and bacteriocins are less represented. High percentages of *Staphylococcus* species, MRSA, *P. aeruginosa*, and *C. albicans* were detected in the mouths of elderly patients [111, 112]. By PCR, a notable occurrence of MRSA, vancomycin-resistant *S. aureus* (VRSA), and VSSA have been observed in the oral cavity of patients with dental caries [113]. Chronic periodontitis showed extensive antibiotic-resistant subgingival periodontal pathogens in cultivable microbiota, associated with red and orange complex species, and also to Gram-negative enteric rods/Pseudomonads, *E. faecalis*, and *S. aureus* [21, 23, 24, 114].

Here, we report updated data on *S. aureus* and MRSA carriage rates among dental students, dental patients, HCWs, and dental healthcare personnel (DHCP) in Table 1 [91–103]. Despite the many differences between studies, nowadays there is a probable occupational exposure, from carriage rates, among DHCP and HCWs. This is higher in dental students (Table 1), but would seem evident in the last years [14, 91–95]. Nasal MRSA colonization, confirmed by the presence of the *mecA* gene that encodes a low-affinity penicillin-binding protein, occurs in dental students (3.1%), especially those who have clinical experience [94]. MRSA hand and nasal carriage rates in patients, nurses, and dentist are significant in dental settings (Table 1) [102]. The majority of MRSA isolates were multidrug resistant, and full resistance was generally higher for personnel than for the environmental isolates.

5.1.1 Community- and hospital-acquired MRSA infections and dentistry

Taking into account MRSA carriage in dental patients and DHCP, the effectiveness of MRSA decolonization, and the violation of IC precautions (see below and in Part 2), MRSA in the oral cavity could potentially be disseminated by carriers (patient and DHCP) to the environment [115]. It is well known that community-acquired MRSA (CA-MRSA) infections often occur in young and healthy individuals, whereas HA-MRSA infections occur predominantly in elder or immunocompromised patients in healthcare settings and vary considerably between different countries [116, 117].

HA-MRSA and CA-MRSA have opposite features concerning competitive fitness, virulence, and antimicrobial resistance [118]. Only rarely HA-MRSAs cause infections in healthy subjects, but at least two CA-MRSAs (USA300 and ST30) cause HA infections. It is not known if these strains acquire multiple resistant genes from HA-MRSA or if they increase bacterial fitness and survival despite the antibiotic resistance. Taking into account that their extracellular proteome seems to be differently involved, we think that this epidemiological change is not soothing for future dental epidemiology. In fact, from a 10-year retrospective analysis of laboratory data, obtained from oral and perioral clinical specimens, most of the MRSA isolates were epidemic MRSA-15 (EMRSA-15) or EMRSA-16 lineage, known to cause both very dangerous HA-MRSA infections [97]. No MRSA isolates belonging to community-acquired recognized lineages were identified. An alarming genetic similarity has been shown between seven MRSA isolated in dental clinic and the EMRSA-15 clone [102]. In addition, *S. aureus*, MSSA, and EMRSA-15 harbored differently on dentures of in- and outpatients [119].

5.2 *Enterococcus faecalis*

It is well known that antibiotic administration causes intestinal overgrowth of *Enterococci* and their translocation across a histologically normal intestinal epithelium; then, they can reach and avidly bind other soft tissues and endocardial tissue matrix components, causing infections, abscess, and endocarditis. There are some
reasons to consider Enterococci important for our topic. E. faecalis occurs in transient opportunistic infections involving the oral cavity and has been found in common dental diseases (i.e. caries, endodontic infections, periodontitis) and peri-implant infective disease, and its strains are peculiar in comparison to food ones [120]. Recently, public health officials reported an incidence rate of enterococcal endocarditis among the total patient population at the oral surgery practice, more than 200 times the expected rate among general population [11].

In addition, E. faecalis is so invasive that it is used to test dental materials (composite fillings, endodontic sealers, etc.) and the connection between DI and the abutment [121]. Since it is highly adhesive, has many virulence factors (resistance to extreme conditions (oxygen tension, pH, salts), collagen-binding proteins, gelatinase E, surface proteins), and the ability to form biofilm, E. faecalis can reside widely in and around tooth root canals, in the surrounding bone trabeculae, and in heavily infected subgingival sites [122, 123]. It is known that E. faecalis resistance to antibiotics has been increasing over time. Then, the oral cavity can constitute a reservoir for virulent E. faecalis strains possessing antibiotic resistance traits, able to transfer vanA resistance genes to MRSA [102] and with biofilm formation capabilities. The latter facilitates the exchange of genetic material (via horizontal gene transfer) important for resistance acquisition [120]. Tetracycline, erythromycin, clindamycin, and metronidazole revealed poor levels of in vitro activity against human subgingival E. faecalis clinical isolates [122].

Nowadays, Enterobacteriaceae and some resistant strains are present in oral cavity of dental patients, and recently, the transmission in dental practice has been proven [11, 120–124]. For dentistry of the future, whole-genome sequencing seems promising to study Enterobacteriaceae antimicrobial resistance based on genotype alone [125] and the role in dental implant-associated infections.

6. Surgical infection prevention in dentistry: from gold standard to reality

It is well known that the best choices for dental and implant surgery are a specialized and well-trained dental staff (surgeon, clean nurse, second nurse, anesthetist, etc.) and a specific designed surgical room with proper isolation, clean air system ventilation, instruments for automatic surface decontamination and ISO standards (UNI EN ISO 14644-ISO 5) that allow a very low environmental contamination, and proper antiseptic procedures (including hand washing, wearing, safe instruments passages). Unfortunately, this setting up is used in the case of maxillofacial surgery, and it is commonly present and economically sustainable in hospital surgical dental department. In ambulatory dental offices, there is no isolation and a full separation of the environments used for general dentistry and those used for implant surgery or dental extractions. Only rarely is present a clean air ventilation system according to ISO standards. This difference is very important since in general dental practices the cross-infection is widespread, and the infection prevention is more difficult or less controllable (i.e. absence of the second nurse, environmental contamination) compared to hospital surgical rooms. There are few controls legislated over the operating environment in ambulatory and private dental offices.

Bearing in mind the higher risk of contamination of ambulatory surgical areas, above all during long surgeries (sinus lift, several implant placing, guided bone regeneration (GBR)) and in medically compromised patients, we cannot exclude that a part of implant failures is the result of a chain of personnel latent errors, including some improper antiseptic measures (not surgical hand hygiene, unsterile
gloves, improper use of mask, contamination of operating surface or room air, unsterile barrier covering, lack of surgical guide disinfection and mouth rinses, suture contamination by perioral skin bacteria, among others), as far as untrained professional practice [17, 41, 42, 44, 126].

Maintaining sterile conditions during the surgical procedure is of utmost importance. Saliva, perioral skin, unsterile instruments, contaminated gloves, operating room air, or air expired by the patient, all interfere in the surgical procedure leading to contamination of the implant site [43, 45–47]. It has been reported that the prevalence rate of MRSA was the highest in samples from dental surgery compared to other dental environments [102]. MRSA’s involvement in surgical infections is in line with the estimated infective dose, which is very low (4 CFU), and surface contamination (<10 CFU/cm²) [127, 128]. In ambulatory surgical centers, the main infection control lapses identified were hand hygiene and use of PPE, injection safety and medication handling, equipment reprocessing, and environmental cleaning [41, 42, 129].

The majority of DIs are predominantly placed in general dental practice under local anesthesia. Concerning local anesthesia, hand contact is the main source of the wide contamination reported on anesthetic syringes and anesthetic tubes used in dentistry [130]. Then, DHCP has to follow scrupulously key recommendations for safe injection reported in CDC guidelines [6]. Taking into account the recent outbreaks, the violations seem very hazardous in dentistry [8, 11]. In addition, it is absolutely forbidden and highly risky in the reuse of whatsoever single use sterile medical devices (i.e. irrigation sets) and the use of the water from DUWLs during implant and piezoelectric surgery, etc. [6]. The use of sterile devices and instruments is a need during surgical cares, but even after reconditioning, the contamination of surgical dental instruments and drills is significant even in hospital settings [131–134]. Many other specific failures concerning dental instrument reconditioning will be discussed in Part 2. The importance of hand hygiene, sterile gloves, mask, and eye protection during surgery is well known. Violations are frequent and often surgical videos in dentistry show the surgical mask under the nose, that is risky taking into account MRSA nose colonization in dentists and dental nurses. We underline that it is a hazard to touch the barrier membranes during GBR with gloved hands: this is a frequent slip observed in untrained surgeons.

6.1 Infections associated to craniofacial skeleton

The most relevant infections are lateral and apical periodontitis, osteomyelitis, peri-implantitis, and their complications, such as facial cellulitis and other infections involving deep spaces of face and neck [135]. Microbiota associated with infections of the craniofacial skeleton, particularly maxilla and mandible, are polymicrobial in nature and a mix of aerobic-anaerobic genera. In head and neck space odontogenic infections, the most common bacteria isolated were Gram-positive cocci (Viridans streptococci, Prevotella, Staphylococci, and Peptostreptococcus), and discordant data have been reported on antibiotic resistance of Viridans streptococci, while very few isolates of Staphylococcus are now susceptible to penicillin [136, 137].

Taking into account the increasing life expectancy, it is important to underline that older patients, even without systemic diseases, are more prone to development of oral pathology infections because of often lower immunological response [138]. Concerning systemic and local odontogenic infection complications requiring hospital care, an analysis showed that medically compromised patients appear more susceptible to systemic rather than local infection complications with a need for significantly longer hospital stay and with an increased risk for fatal complications [139].
The main causative agents of maxillofacial inflammatory diseases are *S. aureus*, *S. epidermidis*, *Streptococcus* spp., *Escherichia coli*, and *Proteus* spp. [85]. Concerning the risk of maxillofacial surgeries, 4% of their patients showed odontogenic infections, and about 2–20% required intensive medical therapy after surgery [140, 141]. These compliances are expected to worsen taking into account the current oral carriage of *S. aureus* and MRSA (Table 1) and the presence of epidemic MRSA-15 (EMRSA-15) or EMRSA-16 lineage in dental settings.

Results have been conflicting concerning the occurrence of bacteremia after dental procedures; antimicrobial prophylaxis before an invasive dental procedure does not prevent bacteremia, although it can decrease both its magnitude and its persistence [142]. Delayed-onset infections (DOI) after mandibular third molar extractions are rare complications and usually occur about 30 days after the extraction, but they may also develop much later on [143]. The bacteria identified in DOI are *Fusobacterium*, *Prevotella*, *Bacteroides*, and *Peptostreptococcus*. A recent review reported in detail several oral and maxillofacial fungal infections, including mucormycosis, candidiasis, aspergillosis, blastomycosis, histoplasmosis, cryptococcosis, and coccidioidomycosis [144].

### 6.2 Infective agents in dental implantology

In general, dental implant procedures are considered clean-contaminated surgeries (graded as class II surgical procedures), since micro-organisms living in the oral mucosa and in saliva contaminate the surgical wound facilitating the infection, with local infection rates of 10–15% and an incidence of infection to 1% or less by the use of both prophylactic antibiotics and proper surgical technique [71]. Despite the statements reported between 1980 and 1990, even in the case of the use of prophylactic antibiotics, the reported prevalence of postoperative infection after implant installation ranges from 0 up to 11.5% and the prevalence of peri-implantitis varied from 4.2 to 47% of all implants [21, 56, 69, 71, 84, 114, 145–149]. These data are higher than the annual infection rate for cardiovascular implants and orthopedic implants, that is, 7.4 and 4.3% respectively, in USA hospital settings. Unfortunately, data are not available on the concurrent nasal/throat colonization of MRSA as possible patient-implant related factors and DI failure.

Clinical recommendations for avoiding and managing surgical complications associated with implant dentistry have been recently published [150, 151]. However, despite careful planning, infection is one of the early and late implant complications and iatrogenic actions are regarded as accidents during surgical procedures, complications, or failures caused by a deficient praxis of the professional. Infection is the most common explanation for complications such as swelling, suppuration, fistulas, and early/late mucosal dehiscence that may point to implant failure.

Many papers have reported improvements (mainly on the topography and surface features; antimicrobial dental implant functionalization strategies) of DIs and surgery techniques to get better osteointegration and to reduce the infective complications and then to improve long-term success (longevity and function of implants and uploaded prosthesis) [27–30, 32].

Peri-implantitis is a nonspecific, polymicrobial, and heterogeneous diseases of endogenous (caused by commensal oral strains) and iatrogenic nature, with an increased level of pathogenic bacteria from the orange and red complexes and towards a flora with a greater proportion of Gram-negative, motile, anaerobic bacteria [29, 152]. Compared to periodontal disease, the microbial biofilm harbored in peri-implant infective diseases is generally changeable and composed of opportunistic and Gram-negative species. Implant failure can occur at any time during the implant treatment by bacterial infection, but early healing period is quite important due to impaired wound healing.
These microorganisms have been found differently associated to implant infections: Porphyromonas gingivalis; endodontalis and spp.; Tanneraella forsythia and socransky; Prevotella nigrescens, oris, and intermedia; Fusobacterium spp. and nucleatum; Synergistes spp. HO T—360; Pseudoramibacter alactolyticus; Eubacterium spp.; Veillonella spp.; Enterobacteriaceae; Candida spp.; Filisfactor alocis; Dialister invisus; Mitsuokella spp. HOT 131; Peptococcus spp. HO T-168; Clostridia [F-1] [G-1] spp. HO T-093; Catonella morbid; Chloroflexi spp.; Tenericutes spp.; Aggregatibacter actinomycetemcomitans; Staphylococcus aureus, anaerobius, and intermedius; Streptococcus mitis; spirochete including Treponema denticola, with some differences associated to the type of DI and bacterial infiltration in the internal screw threads of implants [29, 153–155]. Moreover, implants with a peri-implant lesion had a higher frequency of superinfecting bacteria, mainly Klebsiella pneumonias and Burkholderia cepacia, which are considered environmental and multidrug-resistant bacteria. Significantly higher bacterial counts (Porphyromonas gingivalis, Tannerella forsythia, Treponema denticola, Prevotella intermedia, and Fusobacterium nucleatum) were found for periodontal pathogenic bacteria within the implant-abutment interface of implants in patients with peri-implantitis compared to those implants surrounded by healthy peri-implant tissues [156]. Using next-generation sequencing methods, recent results indicate that peri-implantitis and periodontitis are both polymicrobial infections with different causative pathogens, and the severity of the peri-implantitis was species-associated, including with Eubacterium minutum and an uncultured Treponema sp. [157, 158]. Opportunistic microorganisms (enteric rods and S. aureus) were found differently in peri-implantitis sites [21, 145].

We underline that some of them (Enterobacteriaceae, Candida, Staphylococcus, and Streptococcus) have been indicated as prioritized bacteria in CDC recommendation [18]. Some authors reported that antibiotics do not seem to reduce the incidence of postoperative infections and 2/3 of the infected implants failed before prosthetic loading [21, 146–149]. The majority of bacterial pathogens isolated from peri-implantitis were resistant in vitro to one or more of the tested antibiotics (clindamycin, amoxicillin, doxycycline, or metronidazol) [21].

Nevertheless, microbial investigations seem not contributory to clinician decisions or to be easily applicable nowadays in private practice; the standard procedures (probing, bleeding on probing, probing depth, radiographic assessment, implant mobility) and the visual evaluation of the hyperplastic soft tissues, color changes of the marginal peri-implant tissues, and suppuration are widely used to evaluate the consequences of implant-associated complications [158].

6.2.1 Why does the debridement in dentistry?

Here, we think important to underline some cellular events in relation to implant failures and surgical infections in dentistry. Osseointegration is completed within 3–6 months after implant placement into the dental alveolus, and infection may develop in the early operative period (early infection) or after the process of implant integration (late infection).

At the cellular level, implant-associated infections are the result of two critical phases in the first 6 h post implantation; firstly, the bacterial adhesion to a biomaterial surface by weak and unspecific forces within 1–2 h after implantation, and approximately 2–3 h later, a stronger adhesion with the formation of microcolonies and biofilm, which precedes clinical infection [63]. It is important that Staphylococcus species, isolated in dental settings, show high affinity to titanium and good biofilm production [102, 159], which are concurrent detrimental factors for osteogenesis [160, 161]. In addition, during the stationary phase, at least 1% of bacterial cells in biofilms become tolerant to antibiotics [162]. Moreover, the extracellular matrix should provide a
stable physical environment for cell to-cell contact, which allows the dissemination of antibiotic resistance by horizontal gene transfer among *S. aureus* [163].

In is well known that smoking is associated with DI failures [159] and that some infective agents (i.e. *Porphyromonas gingivalis*, SA, etc.) showed increased colonization in smokers. Cigarette smoking induces Staphylococcal biofilm formation in an oxidant-dependent manner and enhancement of fibronectin, an important extracellular matrix protein, binding in *S. aureus* [164]. This is relevant for adherence, invasion, and colonization since Staphylococci, in particular *S. aureus*, are the main causes of bone infections [165]. In addition, by molecular mechanisms, *Staphylococci* are able to invade *in vivo* host bone cells (osteoblasts and osteocytes), endothelial cells, and the canaliculi of live cortical bone leading to biofilm formation in osteocyte lacunae [166]. *Staphylococci*, as facultative intracellular pathogens, are shielded from immune response and antibiotics and are expected to induce a highly programmed and regulated cell death of osteogenic cells and then to impair bone formation. *E. faecalis* too is capable of surviving in a vegetative state in healed bone and of reactivation upon DI placement [22].

Then, it is not surprising that a nightmare and a difficult problem are to eradicate implant infections in present dental practice [149]. For the success of the DI surgery, it seems important a careful debridement of the alveolus from infective agents, frequently drug resistants, above all in the case of immediate DI loading after dental extraction and to defer DI placement after a dental extraction [27, 167].

### 6.3 Focus on orthodontia-associated surgery

Infections complications in orthognathic surgery are lower only to those caused by nerve injury [168]. The incidence of surgical site infections was limited to 1% of patients after bimaxillary orthognathic, osseous genioplasty, and intranasal surgery and under antibiotic treatment [162]. No attention is given to ARIAs in orthodontia and orthognathic surgery. To date, there is no gold standard for the treatment of postoperative infections in orthodontic surgery and the use of prophylactic antibiotics before some orthodontic procedures (orthodontic band placement, separator placement, or screw insertion) in patients with a medical history that reveals the presence of diseases affecting the host defense system (aging, patient on corticosteroids or bisphosphonates or anticoagulants, diabetes mellitus, HIV/AIDS) since they are at high risk of developing oral infection [37, 169]. Endocarditic prophylaxis is indicated only during the initial placement of orthodontic bands (not brackets).

We previously reviewed the problems related to task-specific evidence-based guidelines for cross-infection control when placing different temporary orthodontic anchorage devices [37]. Infection occurred in 17.3% of the installed miniplates and was caused by predominantly anaerobic, mainly Gram-negative bacteria and associated to immune aging [37, 170, 171]. The failure rate of mini-implants is about threefold to fivefold higher than that of dental implants and mini-plates; nevertheless, the mechanism that leads to mobility and then to their clinical failure is still unknown and more tricky to understand [172]. Recently, interest is arising on the use of antibiotics/antiseptics for some potential beneficial effects on tooth stability after orthodontic treatment, but the advantages should be very carefully balanced in accordance with the risk of antibiotic resistance [173].

### 7. Conclusion

Human infectious diseases will be never-ending [174]. After limitation of dental benefits, there was an increase in the volume and severity of odontogenic infections,
surgical cares increased 100%, and the related healthcare cost skyrockets [175]. The reported data show that opportunistic species and/or ARIA infections are nearby and expected to increase in dental setting [21–26, 29, 81, 82, 85, 91–99, 101–105, 109–114, 120–124, 136–141, 145–149, 153–155, 159, 160, 165] due to the overuse of antibiotics in dentistry and the limited awareness on infection prevention guidelines and the lapses and errors during infection prevention [176]. Moreover, it is considered alarming the genetic connection or similarity between MRSA isolates in dental clinics and on dentures and the EMRSA-15 or EMRSA-16 clone [97, 102, 119]. In addition, Enterobacteriaceae and some resistant strains are present in oral cavity of dental patients, and recently, the transmission in dental practice has been proven [11, 120–124]. The incidence rate of enterococcal endocarditis among the total patient population at the oral surgery practice has been reported to be more than 200 times the expected rate among general population [11].

Then, dental teams have to face occupational and clinical hazards due to ARIA infections in dental facilities. In the absence of or limited new effective antibiotic discovery, the sustainable use of antibiotics is essential but have delayed significant effects [177] based on many collective actions (people information, professional dental-care providers, policy-maker and regulators, industry stakeholders). On the contrary, the prevention of cross infection by adopting guidelines is easily applicable and has had early significant effects on infection prevention and cost-saving [178, 179]. Moreover, it is basic to safeguard dental team reputation, insurance coverings, and reimbursements [8–11, 33–42, 176] and to limit the nightmares to get rid of current dental implant infections [149].

Conflict of interest

L.B. had a service agreement with KerrHawe and is a consultant for Dental Trey Il Blog (http://blog.dentaltrey.it/), neither of which gave any input or financial support to the writing of this article. There are no other conflicts of interest to report.

Abbreviations

| Abbreviation | Definition |
|--------------|------------|
| AE           | adverse event |
| ARIA         | antibiotic-resistant infectious agents |
| CAGR         | compound annual growth rate |
| CA-MRSA      | community-acquired MRSA |
| CDC          | Centers for Disease Control and Prevention |
| CCSs         | clinical contact surfaces |
| cna          | collagen |
| DD           | dental device |
| DHCP         | dental healthcare personnel |
| DI           | dental implant |
| DOI          | delayed-onset infections |
| DUWL         | dental unit water line |
| eHOMD        | expanded human oral microbiome database |
| EMRSA        | epidemic MRSA |
| EPS          | extracellular polysaccharides |
| FAE          | fatal adverse event |
| fnb          | fibronectin |
| GBR          | guided bone regeneration |
| HA           | hospital-acquired |
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HA-MRSA hospital-acquired MRSA
HCW healthcare workers
HPC heterotrophic plate count
ica intercellular adhesion
HSV herpes simplex virus
IFU instruction for use
MAUDE manufacturer and user facility device experience database
MRSA methicillin-resistant Staphylococcus aureus
PCR polymerase chain reaction
VRE vancomycin-resistant Enterococcus

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References

[1] Heballi NB, Ramoni R, Kalenderian E, Delattre VF, Stewart DCL, Kent K, et al. The danger of dental devices as reported in the FDA MAUDE database. The Journal of the American Dental Association. 2015;142(2):102-110. DOI: 10.1016/j.adaj.2014.11.015

[2] Nalliah RP. Trends in US malpractice payments in dentistry compared to other health professions—Dentistry payments increase, others fall. British Dental Journal. 2017;222:36-40. DOI: 10.1038/sj.bdj.2017.34

[3] Hiivala N. Patient safety incidents, their contributing, and mitigating factors in dentistry [thesis]. Universitatis Helsinkiensis; 2016

[4] Ramoni R, Walji M. Creating a dental patient safety initiative. In: Proceedings of the Organization for Safety, Asepsis and Prevention Symposium (OSAP 2015); 28-30 May 2015; Baltimore. Available from: https://www.osap.org. [Accessed: Jun 10, 2015]

[5] Kalenderian E, Obadan-Udoh E, Maramaldi P, Etolue J, Yansane A, Stewart D, et al. Classifying adverse events in the dental office. Journal of Patient Safety. 2017;00:00-00. DOI: 10.1097/PTS.0000000000000407

[6] Summary of Infection Prevention Practices in Dental Settings. USA: Centers for Disease Control and Prevention; 2016. Available from: www.cdc.gov/oralhealth/infectioncontrol/pdf/safe-care2.pdf [Accessed: Jun 12, 2018]

[7] Reuter NG, Westgate PM, Ingram M, Miller CS. Death related to dental treatment: A systematic review. Oral Surgery Oral Medicine Oral Pathology Oral Radiology. 2016;123(2):194-204. DOI: 10.1016/j.oooo.2016.10.015

[8] Cleveland JL, Gray SK, Harte JA, Robison VA, Moorman AC, Gooch BF. Transmission of blood-borne pathogens in us dental health care settings. 2016 Update. The Journal of the American Dental Association. 2016;147(9):729-738. DOI: 10.1016/j.adaj.2016.03.02

[9] Arduino M, Miller J, Shannon M. Safe water, safe dentistry, safe kids. Organization for Safety, Asepsis and Prevention. Webinar. Available from: https://www.osap.org/page/LecturesWebinarsConf. [Accessed: May 27, 2017]

[10] Ricci ML, Ricci ML, Fontana S, Pinci F, Fiumana E, Pedna MF, et al. Pneumonia associated with a dental unit water line. The Lancet. 2012;379(9816):684. DOI: 10.1016/S0140-6736(12)60074-9

[11] Ross KM, Mehr JS, Greeley RD, Montoya LA, Kulkarni PTA, Frontin S, et al. Outbreak of bacterial endocarditis associated with an oral surgery practice. The Journal of the American Dental Association. 2018;149(3):191-201. DOI: 10.1016/j.adaj.2017.10.002

[12] Perea-Perez B, Labajo-Gonzalez E, Acosta-Gio AE, Yamalik N. Eleven basic procedures/practices for dental patient safety. Journal of Patient Safety. 2015;00:00-00. DOI: 10.1097/PTS.0000000000000234

[13] Davies DS. Dental news. British Dental Journal. 2013;214(7):329

[14] Petti S, Polimeni A. Risk of methicillin-resistant Staphylococcus aureus transmission in the dental healthcare setting: A narrative review. Infection Control and Hospital Epidemiology. 2011;32(11):1109-1115. DOI: 10.1086/662184

[15] Laheij AMGA, Kistler JO, Belibasakis GN, Välimaa H, de Soet JJ. Healthcare-associated viral and bacterial infections in dentistry. Journal
Infection Control in Dentistry and Drug-Resistant Infectious Agents: A Burning Issue. Part 1
DOI: http://dx.doi.org/10.5772/intechopen.80961

[17] Merlos A, Vinuesa T, Jane-Salas E, Lopez-Lopez J, Vinas M. Antimicrobial prophylaxis in dentistry. Journal of Global Antimicrobial Resistance. 2014;2:232-238. DOI: 10.1016/j.jgar.2014.05.007 2213-7165

[18] Centers for Disease Control and Prevention, Office of Infectious Disease. Antibiotic Resistance Threats in the United States, 2013. April 2013. Available from: http://www.cdc.gov/drugresistance/threat-report-2013 [Accessed: Jul 24, 2018]

[19] Ventola CL. The antibiotic resistance crisis. Part 1: Causes and threats. Pharmacy and Therapeutics. 2015;40(4):277-283

[20] Ventola CL. The antibiotic resistance crisis. Part 2: Management strategies and new agents. Pharmacy and Therapeutics. 2015;40(5):344-352

[21] Rams TE, Degener JE, van Winkelhoff AJ. Antibiotic resistance in human peri-implantitis microbiota. Clinical Oral Implants Research. 2014;25:82-90. DOI: 10.1111/clr.12160

[22] Flanagan D. Enterococcus faecalis and dental implants. Journal of Oral Implantology. 2017;153(1):8-11. DOI: 10.1563/aaid-joi-D-16-00069

[23] Rams TE, Degener JE, van Winkelhoff AJ. Antibiotic resistance in human chronic periodontitis microbiota. Journal of Periodontology. 2014;85:160-169. DOI: 10.1902/jop.2013.130142

[24] Ardila CM, Granada MI, Guzmán IC. Antibiotic resistance of subgingival species in chronic periodontitis patients. Journal of Periodontal Research. 2010;45:557-563

[25] Sun J, Song X, Kristiansen BE, Kjrøe A, Willems RJL, Eriksen HM, et al. Occurrence, population structure, and antimicrobial resistance of Enterococci in marginal and apical periodontitis. Journal of Clinical Microbiology. 2009;47(7):2218-2225. DOI: 10.1128/JCM.00388-09

[26] Dahlén G, Blomquist S, Carlén A. A retrospective study on the microbiology in patients with oral complaints and oral mucosal lesions. Oral Diseases. 2009;15:265-272. DOI: 10.1111/j.1601-0825.2009.01520.x

[27] Elias CN, Meirelles L. Improving osseointegration of dental implants. Expert Review of Medical Devices. 2010;7(2):241-256. DOI: 10.1586/ERD.09.74

[28] Duraccio D, Mussano F, Faga MG. Biomaterials for dental implants: Current and future trends. Journal of Materials Science. 2015;50:4779-4812. DOI: 10.1007/s10853-015-9056-3

[29] Pokrowiecki R, Mielczarek A, Zaręba T, Tyski S. Oral microbiome and peri-implant diseases: Where are we now? Therapeutics and Clinical Risk Management. 2017;13:1529-1542. DOI: 10.2147/TCRM.S139795

[30] Rasouli R, Barhoum A, Uludag H. A review of nanostructured surfaces and materials for dental implants: Surface coating, patterning and functionalization for improved performance. Biomaterials Science. 2018;6:1312-1338. DOI: 10.1039/c8bm00021b

[31] Dental Implants Market Size, Share & Trends Analysis Report By Product (Titanium Implants, Zirconium
Implants), By Region (North America, Europe, Asia Pacific, Latin America, MEA), and Segment Forecasts, 2011-2024. [Internet]. Available from: https://www.grandviewresearch.com/industry-analysis/dental-implants-market [Accessed: Apr 15, 2018]

[32] Pjetursson BE, Asgeirsson AG, Zwahlen M, Sailer I. Improvements in implant dentistry over the last decade: Comparison of survival and complication rates in older and newer publications. International Journal of Oral and Maxillofacial Implants. 2014;29(8 Suppl):308-324. DOI: 10.11607/jomi.2014suppl.g5.2

[33] Chang W-J, Chang Y-H. Patient satisfaction analysis: Identifying key drivers and enhancing service quality of dental care. Journal of Dental Sciences. 2013;8:239-247. DOI: 10.1016/j.jds.2012.10.006

[34] Clayton JL, Miller KJ. Professional and regulatory infection control guidelines: Collaboration to promote patient safety. AORN Journal. 2017;106:201-210. DOI: 10.1016/j.aorn.2017.07.005

[35] Collins FM. The significance of the US Food and Drug Administration for dental professionals and safe patient care. The Journal of the American Dental Association. 2014;217(2):858-861. DOI: 10.1016/j.adaj.2017.08.026

[36] Oosthuysen J, Potgieter E, Fossey A. Compliance with infection prevention and control in oral health-care facilities: A global perspective. International Dental Journal. 2014;64(6):297-311. DOI: 10.1111/idj.12134

[37] Barenghi L, Barenghi A, Di Blasio A. Implementation of recent infection prevention procedures published by centers for disease control and prevention: Difficulties and problems in orthodontic offices. Iranian Journal of Orthodontics. 2018;13(1):e10201. DOI: 10.5812/ijo.10201

[38] Barenghi L. Clean, disinfect and cover: Top activities for clinical contact surfaces in dentistry [Internet]. 2015. Available from: www.kerrdental.com/resource-center/clean-disinfect-and-cover-%E2%80%93top-activities-clinical-contactsurfaces-dentistry-dr [Accessed: Jun 12, 2018]

[39] Barenghi L. The Daily Fight to Limit Cross-infection in a Dental Office [Internet]. 2017. Webinar. Available from: http://blog.kavo.com/en/webinar-daily-fight-limit-cross-infection-dental-office [Accessed: Jun 12, 2018]

[40] Jakubovics N, Greenwood M, Meechan JG. General medicine and surgery for dental practitioners: Part 4. Infections and infection control. British Dental Journal. 2014;217(2):73-77. DOI: 10.1038/sj.bdj.2014.593

[41] Monarca S, Grottolo M, Renzi D, Paganelli C, Sapelli P, Zerbini I, et al. Evaluation of environmental bacterial contamination and procedures to control cross infection in a sample of Italian dental surgeries. Occupational and Environmental Medicine. 2000;57:721-726. DOI: 10.1136/oem.5711.721

[42] Schaefer MK, Michael J, Marilyn Dahl M, et al. Infection control assessment of ambulatory surgical centers. Journal of the American Medical Association. 2010;303(22):2273-2279. DOI: 10.1001/jama.2010.744

[43] Ehrlich T, Dietz B. Chapter 30. In: Modern Dental Assisting. 5th ed. USA: W.B. Sounders Company; 1995. ISBN: 0-7216-5053-8

[44] Miller CH, Palenik CJ. Chapters 14, 17. In: Infection Control and Management of Hazardous Materials for the Dental Team. 4th ed. Evolve. USA: Mosby Elsevier; 2010. ISBN: 978-0-323-05631-1

[45] Pankhurst CL, Coulter WA. Chapters 2, 4-9. In: Basic Guide to
Infection Prevention and Control in Dentistry. 2nd ed. UK: Wiley Blackwell; 2017. ISBN: 9781119164982

[46] Rutala WA, Weber DJ. The Healthcare Infection Control Practices Advisory Committee (HICPAC). Guidelines for Infection Control in Dental Health-Care Settings 2003–MMWR 2003521-61. Available from: www.cdc.gov/mmwr/preview/mmwrhtml/rr5217al.htm [Accessed: Jun 12, 2018]

[47] Rutala WA, Weber DJ, The Healthcare Infection Control Practices Advisory Committee (HICPAC). Guideline for Disinfection and Sterilization in Healthcare Facilities. 2008. Available from: www.cdc.gov/infectioncontrol/guidelines/disinfection [Accessed: Feb 15, 2017]

[48] Expanded Human Oral Microbiome Database (eHOMD). Available from: www.homd.org/index.php [Accessed: May 18, 2018]

[49] Siqueira JF, Fouad AF, Rocas IN. Pyrosequencing as a tool for better understanding of human microbiomes. Journal of Oral Microbiology. 2012;4:10743. DOI: 10.3402/jom.v4i0.10743

[50] Tsunemine H, Yoshioka Y, Nagao M, Tomaru Y, Saitoh T, Adachi S, et al. Multiplex polymerase chain reaction assay for early diagnosis of viral infection. In: Samadikuchaksaraeai A, editor. Polymerase Chain Reaction for Biomedical Applications. UK: InTech; 2016. pp. 69-82. DOI: 10.5772/65771

[51] Rozman U, Turk SS. PCR technique for the microbial analysis of inanimate hospital environment. In: Samadikuchaksaraeai A, editor. Polymerase Chain Reaction for Biomedical Applications. UK: InTech; 2016. pp. 119-134. DOI: 10.5772/65742

[52] Valeriani F, Protano C, Gianfranceschi G, Cozza P, Campanella V, Liguori G, et al. Infection control in healthcare settings: Perspectives for mfDNA analysis in monitoring sanitation procedures. BMC Infectious Diseases. 2016;16:394. DOI: 10.1186/s12879-016-1714-9

[53] Lamas A, Franco CM, Regal P, Miranda JM, Vázquez B, Cepeda A. High-throughput platforms in real-time PCR and applications. In: Samadikuchaksaraeai A, editor. Polymerase Chain Reaction for Biomedical Applications. UK: InTech; 2016. pp. 15-38. DOI: 10.5772/65760

[54] Koidhi B, Zmantar T, Mahdouani K, Hentati H, Bakhrouf A. Antibiotic resistance and adhesion properties of oral Enterococci associated to dental caries. BMC Microbiology. 2011;11:155. DOI: 10.1186/1471-2180-11-155

[55] Tsang STJ, McHugh MP, Guerendiain D, Gwynne PJ, Boyd J, Simpson AHRW, et al. Underestimation of Staphylococcus aureus (MRSA and MSSA) carriage associated with standard culturing techniques. Bone & Joint Research. 2018;7:79-84. DOI: 10.1302/2046-3758.71.BJR2017-0175.R1

[56] Charalampakis G, Leonhardt A, Rabe P, Dahlen G. Clinical and microbiological characteristics of peri-implantitis cases: A retrospective multicentre study. Clinical Oral Implants Research. 2012;23:1045-1054. DOI: 10.1111/j.1600-0501.2011.02258.x

[57] Korkut E, Uncu AT, Sener Y. Biofilm formation by Staphylococcus aureus isolates from a dental clinic in Konya, Turkey. Journal of Infection and Public Health. 2017;10:809-813. DOI: 10.1016/j.jiph.2017.01.004

[58] Siqueira JF, Rocas IN. Polymerase chain reaction-based analysis of microorganisms associated with failed endodontic treatment. Oral Surgery Oral Medicine Oral Pathology Oral
[59] Pankhurst C, Rautemaa-Richardson R, Seoudi N, Smith A, Wilson M. Antibiotics and consultant oral microbiologist posts. British Dental Journal. 2016;220(1):2-3. DOI: 10.1038/sj.bdj.2016.5

[60] Emecen-Huja P, Hasan I, Miller CS. Biologic markers of failing implants. Dental Clinics. 2015;59(1):179-194. DOI: 10.1016/j.cden.2014.08.007

[61] Hoyos-Nogués M, Brosel-Oliu S, Abramova N, Muñoz F-X, Bratov A, Mas-Moruno C, et al. Impedimetric antimicrobial peptide-based sensor for the early detection of periodontopathogenic bacteria. Biosensors and Bioelectronics. 2016;15(86):377-385. DOI: 10.1016/j.bios.2016.06.066

[62] Salako NO, Rotimib VO, Adibb SM, Al-Mutawac S. Pattern of antibiotic prescription in the management of oral diseases among dentists in Kuwait. Journal of Dentistry. 2004;32:503-509. DOI: 10.1016/j.jdent.2004.04.001

[63] Monteiro Lisboa S, Parreiras M, de Castilho LS, de Souza e Silva ME, Nogueira Guimarães AM. Prescribing errors in antibiotic prophylaxis by dentists in a large Brazilian city. American Journal of Infection Control. 2015;43:767-768. DOI: 10.1016/j.ajic.2015.03.028

[64] Marra F, George D, Chong M, Sutherland S, Patrick DM. Antibiotic prescribing by dentists has increased. Why? The Journal of the American Dental Association. 2016;147(5):320-327. DOI: 10.1016/j.adaj.2015.12.014

[65] Haliti N, Krasnqi S, Begzati A, Gllareva B, Krasnqi L, Shabani N, et al. Antibiotic prescription patterns in primary dental health care in Kosovo. Family Medicine & Primary Care Review. 2017;19, 2:128-133. DOI: 10.5114/fmpcr.2017.67866

[66] Löffler C, Bohmer F. The effect of interventions aiming to optimize the prescription of antibiotics in dental care—A systematic review. PLoS One. 2017;12(11):e0188061. DOI: 10.1371/journal.pone.0188061

[67] Buttar R, Aleksejuniene J, Shen Y, Coil J. Antibiotic and opioid analgesic prescribing patterns of dentists in Vancouver and endodontic specialists in British Columbia. Journal of the Canadian Dental Association. 2017;83:h8. PMID: 29513210

[68] Koyuncuoğlu CZ, Aydin M, Kirmizi NI, Aydin V, Aksoy M, Islı F, et al. Rational use of medicine in dentistry: Do dentists prescribe antibiotics in appropriate indications? European Journal of Clinical Pharmacology. 2017;73:1027-1032. DOI: 10.1007/s00228-017-2258-7

[69] Prasad S, Rajesvari R. Antibiotic prescribing practice among general dental practitioners. Journal of Oral Medicine, Oral Surgery, Oral Pathology and Oral Radiology. 2017;3(1):14-16. DOI: 10.18231/2395-6194.2017.0004

[70] Oteri G, Panzarella V, Marciano A, Di Fede O, Maniscalco L, Peditto M, et al. Appropriateness in dentistry: A survey discovers improper procedures in oral medicine and surgery. International Journal of Dentistry. 2018;00:10 Article ID 3245324. DOI: 10.1155/2018/3245324

[71] Khalil D, Lund B, Hultin M. Antibiotics in implant dentistry. In: Mazen Ahmad M, Almasri JA, editors. Dental Implantology and Biomaterial. UK: InTech; 2016. pp. 19-38. DOI: 10.5772/62681

[72] Swedres-Svarm. Consumption of antibiotics and occurrence of resistance in Sweden. Solna/Uppsala. 2016. ISSN: 1650-6332. Available from: www.
folkhalsomyndigheten.se/contentassets/d118ac95c12dc41b3e61d34ee6d2332/swedres-svarm-2016-16124.pdf [Accessed: Jun 16, 2018]

[73] Holmstrup P, Klausen B. The growing problem of antimicrobial resistance. Oral Diseases. 2018;24:291-295. DOI: 10.1111/odi.12610

[74] Martins JR, Chagas OL, Velasques BD, Niemczewski Bobrowski A, Britto Correa M, Torriani MA. The use of antibiotics in odontogenic infections: What is the best choice? A systematic review. Journal of Oral and Maxillofacial Surgery. 2017;75:2606.e1-2606.e11. DOI: 10.1016/j.joms.2017.08.017

[75] Segura-Egea JJ, Gould K, Hakan Sen B, Jonasson P, Cotti E, Mazzone A, et al. European Society of Endodontology position statement: The use of antibiotics in endodontics. International Endodontic Journal. 2018;51:20-25. DOI: 10.1111/iej.12781

[76] Ramos E, Santamaría J, Santamaría G, Barbier L, Arteagoitia I. Do systemic antibiotics prevent dry socket and infection after third molar extraction? A systematic review and meta-analysis. Oral Surgery Oral Medicine Oral Pathology Oral Radiology. 2016;122:403-425. DOI: 10.1016/j.oooo.2016.04.016

[77] Bailey E, Tickle M, Campbell S, O'Malley L. Systematic review of patient safety interventions in dentistry. BMC Oral Health. 2015;15:152. DOI: 10.1186/s12903-015-0136-1

[78] Cloitre A, Duval X, Hoen B, Alla F, MD LP. A nationwide survey of French dentists’ knowledge and implementation of current guidelines for antibiotic prophylaxis of infective endocarditis in patients with predisposing cardiac conditions. Oral Surgery Oral Medicine Oral Pathology Oral Radiology. 2018;125:295-303. DOI: 10.1016/j.oooo.2017.10.002

[79] Dayer M, Thornhill M. Is antibiotic prophylaxis to prevent infective endocarditis worthwhile? Journal of Infection and Chemotherapy. 2018;24. DOI: 18e24. DOI: 10.1016/j.jiac.2017.10.006 1341-321X

[80] ADA Expert Panel. American Dental Association guidance for utilizing appropriate use criteria in the management of the care of patients with orthopedic implants undergoing dental procedures. 2017;148(2):57-59. DOI: 10.1016/j.adaj.2016.12.002

[81] Hetrick EM, Schoenfisch MH. Reducing implant-related infections: Active release strategies. Chemical Society Reviews. 2006;35:780-789. DOI: 10.1039/b515219b

[82] Miranda-Rius J, Lahor-Soler E, Brunet-Llobet L, de Dios D, Gil FX. Treatments to optimize dental implant surface topography and enhance cell bioactivity. In: Almasri MA, editor. Dental Implantology and Biomaterial. UK: InTech; 2016. pp. 110-127. DOI: 10.5772/62682

[83] Toledo-Arana A, Valle J, Solano C, Arrizubieta M, Cucarella C, Lamata M, et al. The enterococcal surface protein, Esp, is involved in Enterococcus faecalis biofilm formation. Applied and Environmental Microbiology. 2001;67(10):4538-4545. DOI: 10.1128/AEM.67.10.4538-4545.2001

[84] Khalil D. The use of antibiotic prophylaxis in implant dentistry. A microbiological and clinical perspective [thesis]. Stockholm, Sweden: Karolinska Institutet; 2017

[85] Duran-Pinedo AE, Frias-Lopez J. Beyond microbial community composition: Functional activities of the oral microbiome in human health and disease. Microbes and Infection. 2015;17(7):505-516. DOI: 10.1016/j.micinf.2015.03.014
[86] van der Waaij D, Nord CE. Development and persistence of multi-resistance to antibiotics in bacteria: An analysis and a new approach to this urgent problem. International Journal of Antimicrobial Agents. 2000;16(3):191-197

[87] Gordon RJ, Lowy FD. Pathogenesis of methicillin-resistant Staphylococcus aureus infection. Clinical Infectious Diseases. 2008;46(Suppl 5):S350-S359. DOI: 10.1086/533591

[88] Mertz D, Frei R, Jaussi B, Tietz A, Stebler C, Fluckiger U, et al. Throat swabs are necessary to reliably detect carriers of Staphylococcus aureus. Clinical Infectious Diseases. 2007;45(4):475-477. DOI: 10.1086/520016

[89] Abreu AC, Tavares RR, Borges A, Mergulhão F, Simões M. Current and emergent strategies for disinfection of hospital environments. Journal of Antimicrobial Chemotherapy. 2013;68:2718-2732. DOI: 10.1093/jac/dkt281

[90] Costalonga M, Herzberg MC. The oral microbiome and the immunobiology of periodontal disease and caries. Immunology Letters. 2014;162(200):22-38. DOI: 10.1016/j.imlet.2014.08.017

[91] Roberts MC, Soge OO, Horst JA, Ly KA, Milgrom P. Methicillin-resistant Staphylococcus aureus from dental school clinic surfaces and students. American Journal of Infection Control. 2011;39:628-632. DOI: 10.1016/j.ajic.2010.11.007

[92] Martinez-Ruiz FJ, Carrillo-Espindola TY, Bustos-Martinez J, Hamdan-Partida A, Sanchez-Perez L, Acosta-Gio AE. Higher prevalence of methicillin-resistant Staphylococcus aureus among dental students. Journal of Hospital Infection. 2014;86(3):216-218. DOI: 10.1016/j.jhin.2013.12.00

[93] Petti S, Kakisina N, Volgenant CMC, Messano GA, Barbato E, Passariello C, et al. Low methicillin-resistant Staphylococcus aureus carriage rate among Italian dental students. American Journal of Infection Control. 2015;43:e89-e91. DOI: org/10.1016/j.ajic.2015.08.00

[94] Baek YS, Baek S-H, Yoo Y-J. Higher nasal carriage rate of methicillin-resistant Staphylococcus aureus among dental students who have clinical experience. Journal of the American Dental Association. 2016;147(5):348-353. DOI: 10.1016/j.adaj.2015.12.004

[95] Hema N, Raj NS, Chaithanya ED, Chincholi R, Iswariya M, Hema KN. Prevalence of nasal carriers of methicillin-resistant Staphylococcus aureus among dental students: An in vivo study. Journal of Oral and Maxillofacial Pathology. 2017;21(3):356-359. DOI: 10.4103/jomfp.JOMFP_212_17

[96] Zimmerli M, Widmer AF, Dangel M, Filippi A, Frei R, Meyer J. Methicillin-resistant Staphylococcus aureus (MRSA) among dental patients: A problem for infection control in dentistry? Clinical Oral Investigations. 2009;13:369-373. DOI: 10.1007/s00784-008-0244-2

[97] McCormack MG, Smith AJ, Akram AN, Jackson M, Robertson D, Edwards MB. Staphylococcus aureus and the oral cavity: An overlooked source of carriage and infection? American Journal of Infection Control. 2015;43(1):35-37. DOI: 10.1016/j.ajic.2014.09.015

[98] Kabanova A. Bacterial spectrum of orofacial infections and their antibiotic resistance in Belarus. Medical Research Journal. 2017;2(4):152-156. DOI: 10.5603/MRJ.2017.0021

[99] Dulon M, Peters C, Schablon A, Nienhaus A. MRSA carriage among healthcare workers in non-outbreak settings in Europe and the United
States: A systematic review. BMC Infectious Diseases. 2014;14:363. DOI: 10.1186/1471-2334-14-363

[100] Esposito S, Terranova L, Ruggiero L, Ascolese B, Montinaro V, Peves Rios W, et al. Streptococcus pneumoniae and Staphylococcus aureus carriage in healthy school-age children and adolescents. Journal of Medical Microbiology. 2015;64:427-431. DOI: 10.1099/jmm.0.000029

[101] Koukos G, Sakellari D, Arsenakis M, Tsalikis L, Slini T, Konstantinidis A. Prevalence of Staphylococcus aureus and methicillin resistant Staphylococcus aureus (MRSA) in the oral cavity. Archives of Oral Biology. 2015;60:1410-1415. DOI: 10.1016/j.archoralbio.2015.06.009

[102] Khariilla AS, Wasfi R, Ashour HM. Carriage frequency, phenotypic and genomic characteristics of methicillin-resistant Staphylococcus aureus isolated from dental health care personnel, patients and environment. Scientific Reports. 2017;7:7390. DOI: 10.1038/s41598-017-07713-8

[103] Yoo Y-J, Kwak E-J, Jeong KM, Baek S-H, Baek YS. Knowledge, attitudes and practices regarding methicillin-resistant Staphylococcus aureus (MRSA) infection control and nasal MRSA carriage rate among dental health-care professionals. International Dental Journal. 2018;00:1-8. DOI: 10.1111/idj.12388

[104] Didilescu AC, Skaug N, Marica C, Didilescu C. Respiratory pathogens in dental plaque of hospitalized patients with chronic lung diseases. Clinical Oral Investigations. 2005;9:141-147

[105] Smith AJ, Robertson D, Tang MK, Jackson MS, MacKenzie D, Bagg J. Staphylococcus aureus in the oral cavity: A three year retrospective analysis of clinical laboratory data. British Dental Journal. 2003;195(12):701-703. DOI: 10.1038/sj.bdj.4810832

[106] Merghni A, Nejma MB, Hentati H, Mahjoub A, Mastouri M. Adhesive properties and extracellular enzymatic activity of Staphylococcus aureus strains isolated from oral cavity. Microbial Pathogenesis. 2014;73:7-12. DOI: 10.1016/j.micpath.2014.05.002 0882-4010

[107] Merghni A, Nejma MB, Helali I, Hentati H, Bongiovanni A, Lafont F, et al. Assessment of adhesion, invasion and cytotoxicity potential of oral Staphylococcus aureus strains. Microbial Pathogenesis. 2015;86:1-9. DOI: 10.1016/j.micpath.2015.05.010 0882-4010

[108] Suzuki J, Yoshimura G, Kadomoto N, Kuramoto M, Kozai K. Long-term periodical isolation of Staphylococcus aureus and methicillin-resistant Staphylococcus aureus (MRSA) from Japanese children's oral cavities. Pediatric Dental Journal. 2007;17(2):127-130. DOI: 10.11411/pdj.17.127

[109] Merghni A, Nejma MB, Dallel I, Tobji S, Amor AB, Janel S, et al. High potential of adhesion to biotic and abiotic surfaces by opportunistic Staphylococcus aureus strains isolated from orthodontic appliances. Microbial Pathogenesis. 2016;91:61-67. DOI: 10.1016/j.micpath.2015.11.009 0882-4010

[110] Peterson SN, Meissner T, Su AI, Snesrud E, Ong AC, Schork NJ, et al. Functional expression of dental plaque microbiota. Frontiers in Cellular and Infection Microbiology. 2014;4(108):1-13. DOI: 10.3389/fcimb.2014.00108

[111] Abe S, Ishihara K, Okuda K. Prevalence of potential respiratory pathogens in the mouths of elderly patients and effects of professional oral care. Archives of Gerontology and Geriatrics. 2001;32:45-55. DOI: 10.1016/S0167-4943(00)00091-1
[112] El-Solh AA, Pietrantoni C, Bhat A, Okada M, Zambon J, Aquilina A, et al. Colonization of dental plaques: A reservoir of respiratory pathogens for hospital-acquired pneumonia in institutionalized elders. Chest Journal. 2004;126(5):1575-1582. DOI: 10.1016/S0012-3692(15)31374-X

[113] Vellappally S, Divakar DD, Al Kheraif AA, Ramakrishnaiah R, Alqahtani A, Dalati MHN, et al. Occurrence of vancomycin-resistant Staphylococcus aureus in the oral cavity of patients with dental caries. Acta Microbiologica et Immunologica Hungarica. 2017;00:1-9. DOI: 10.1556/030.64.2017.033

[114] Veloo AC, Seme K, Raangs E, Rurenga P, Singadji Z, Wekema-Mulder G, et al. Antibiotic susceptibility profiles of oral pathogens. International Journal of Antimicrobial Agents. 2012;40:450-454. DOI: 10.1016/j.ijantimicag.2012.07.004

[115] Sai N, Laurent C, Strale H, Denis O, Byl B. Efficacy of the decolonization of methicillin-resistant Staphylococcus aureus carriers in clinical practice. Antimicrobial Resistance and Infection Control. 2015;4:56. DOI: 10.1186/s13756-015-0096-x

[116] Otter JA, French GL. Molecular epidemiology of community-associated meticillin-resistant Staphylococcus aureus in Europe. The Lancet Infectious Diseases. 2010;10(4):227-239. DOI: 10.1016/S1473-3099(10)70053-0

[117] National and State Healthcare Associated Infections. CDC report is based on 2014 data. 2016. Available from: www.cdc.gov/HAI/pdfs/progress-report/hai-progress-report.pdf [Accessed: Jun 16, 2018]

[118] Figueiredo AM. What is behind the epidemiological difference between community-acquired and healthcare associated meticillin-resistant Staphylococcus aureus. Virulence. 2017;8(6):640-642. DOI: 10.1080/21505594.2017.1335847

[119] Lewis N, Parmar N, Hussain Z, Baker G, Green I, Howlett J, et al. Colonisation of dentures by Staphylococcus aureus and MRSA in out-patient and in-patient populations. European Journal of Clinical Microbiology & Infectious Diseases. 2015;34:1823-1826. DOI: 10.1007/s10096-015-2418-6

[120] Anderson C, Jonas D, Huber I, Karygianni L, Wölber J, Hellwig E, et al. Enterococcus faecalis from food, clinical specimens, and oral sites: Prevalence of virulence factors in association with biofilm formation. Frontiers in Microbiology. 2016;6:1534. DOI: 10.3389/fmicb.2015.01534

[121] Komiyama EY, Lepesqueur LSS, Yassuda CG, Samaranayake LP, Parahitiyawa NB, Balducci I, et al. Enterococcus species in the oral cavity: Prevalence, virulence factors and antimicrobial susceptibility. PLoS One. 2016;11(9):e0163001. DOI: 10.1371/journal.pone.0163001

[122] Rams TE, Feik D, Mortensen JE, Degener JE, van Winkelhoff AJ. Antibiotic Susceptibility of Periodontal Enterococcus faecalis. Journal of Periodontology. 2013;84(7):1026-1033. DOI: 10.1902/jop.2012.120050

[123] O’Driscoll T, Crank CW. Vancomycin-resistant enterococcal infections: Epidemiology, clinical manifestations, and optimal management. Infection and Drug Resistance. 2015;8:217-230. DOI: 10.2147/IDR.S54125

[124] Magiorakos AP, Burns K, Baño JR, M Borg M, Daikos G, Dumpis U, et al. Infection prevention and control measures and tools for the prevention of entry of carbapenem-resistant...
Enterobacteriaceae into healthcare settings: Guidance from the European Centre for Disease Prevention and Control. Antimicrobial Resistance and Infection Control. 2017;6:113. DOI: 10.1186/s13756-017-0259-z

[125] Tyson GH, Sabo JL, Rice-Trujillo C, Hernandez J, McDermott PF. Whole-genome sequencing based characterization of antimicrobial resistance in Enterococcus. Pathogens and Disease. 2018;76:fty018. DOI: 10.1093/femspd/fty018

[126] Abu-Ta’a M, Quirynen M, Teughels W, van Steenberghe D. Asepsis during periodontal surgery involving oral implants and the usefulness of peri-operative antibiotics: A prospective, randomized, controlled clinical trial. Journal of Clinical Periodontology. 2008;35:58-63. DOI: 10.1111/j.1600-051X.2007.01162.x

[127] Dancer SJ. Controlling hospital-acquired infection: Focus on the role of the environment and new technologies for decontamination. Clinical Microbiology Reviews. 2014;27(4):665-690. DOI: 10.1128/CMR.00020-14

[128] Petti S, Polimeni A, Dancer SJ. Effect of disposable barriers, disinfection, and cleaning on controlling methicillin-resistant Staphylococcus aureus environmental contamination. American Journal of Infection Control. 2013;41(9):836-840. DOI: 10.1016/j.ajic.2012.09.0

[129] Cheng VC-CC, Wong SC-Y, Sridhar S, Chan JF-W, Lai-Ming M, Lau SK-P, et al. Management of an incident of failed sterilization of surgical instruments in a dental clinic in Hong Kong. Journal of the Formosan Medical Association. 2013;112:666-675. DOI: 10.1016/j.jfma.2013.07.020

[130] Neves JK, de Araujo Martins MG, Germinio JES, de Andrade MC, de Oliveira SR. Effectiveness of disinfection of anesthetics tubes in oral surgery-an in vitro study. Journal of Pharmacy and Pharmacology. 2017;7:424-429. DOI: 10.17265/2328-2150/2017.01.005

[131] Hogg NJV, Morrison AD. Resterilization of instruments used in a hospital-based oral and maxillofacial surgery clinic. Journal of Canadian Dental Association. 2005;71:179-182. ISSN: 1488-2159. Available from: www.cda-acd.ca/jcda/vol-71/issue-3/179.html [Accessed: Jul 29, 2017]

[132] Wu G, Yu X. Influence of usage history, instrument complexity, and different cleaning procedures on the cleanliness of blood-contaminated dental surgical instruments. Infection Control and Hospital Epidemiology. 2009;30(7):702-704. DOI: 10.1086/598241

[133] Takamoto M, Takechi M, Ohta K, Ninomiya Y, Ono S, Shigeishi H, et al. Risk of bacterial contamination of bone harvesting devices used for autogenous bone graft in implant surgery. Head & Face Medicine. 2013;9(3):1-5. DOI: 10.1186/1746-160X-9-3

[134] Vassey M, Budge C, Poolman T, Jones P, Perrett D, Nayuni N, et al. A quantitative assessment of residual protein levels on dental instruments reprocessed by manual, ultrasonic and automated cleaning methods. British Dental Journal. 2011;210(9):E14. DOI: 10.1038/sj.bdj.2011.144

[135] Gaetti-Jardim E, Landucci LF, de Oliveira KL, Costa I, Ranieri RV, Okamoto AC, et al. Microbiota associated with infections of the jaws. International Journal of Dentistry. 2012;00:8. Article ID: 369751. DOI: 10.1155/2012/369751

[136] Rega AJ, Aziz SR, Ziccardi VB. Microbiology and antibiotic sensitivities of head and neck space infections of odontogenic origin. Journal of Oral and
Maxillofacial Surgery. 2006;64:1377-1380. DOI: 10.1016/j.joms.2006.05.023

[137] Sánchez R, Miranda E, Arias J, Paño JR, Burgueño M. Severe odontogenic infections: Epidemiological, microbiological and therapeutic factors. Medicina Oral Patologia Oral y Cirugia Bucal. 2011;16(5):e670-e676. DOI: 10.4317/medoral.16995

[138] Zawadzki BJ, Perkowski K, Padzik M, Mierzwińska-Nastalska E, Szafluk JP, Conn DB, et al. Examination of oral microbiota diversity in adults and older adults as an approach to prevent spread of risk factors for human infections. BioMed Research International. 2017;00:7. Article ID: 8106491. DOI: 10.1155/2017/8106491

[139] Seppänen L, Lauhio A, Lindqvist C, Suuronen R, Rautemaa R. Analysis of systemic and local odontogenic infection complications requiring hospital care. Journal of Infection. 2008;57(2):116-122. DOI: 10.1016/j.jinf.2008.06.002

[140] Opitz D, Camerer C, Camerer D-M, Raguse J-D, Menneking H, Hoffmeister B, et al. Incidence and management of severe odontogenic infections—A retrospective analysis from 2004 to 2011. Journal of Cranio-Maxillofacial Surgery. 2015;43(2):285-289. DOI: 10.1016/j.jcms.2014.12.002

[141] Ylijoki S, Suuronen R, Jousimies-Somer H, Meurman JH, Lindqvist C. Differences between patients with or without the need for intensive care due to severe odontogenic infections. Journal of Oral and Maxillofacial Surgery. 2001;59(8):867-872. DOI: 10.1053/joms.2001.25017

[142] Navarro BG, Salas EJ, Devesa AE, López JL. Bacteremia associated with oral surgery: A review. Journal of Evidence Based Dental Practice. 2017;17(3):190-204. DOI: 10.1016/j.jebdp.2016.12.001

[143] Brunello G, De Biagi M, Crepaldi G, Izaura Rodrigues F, Sivolella S. An observational cohort study on delayed-onset infections after mandibular third-molar extractions. International Journal of Dentistry. 2017;00:5. Article ID: 1435348. DOI: 10.1155/2017/1435348

[144] Telles DR, Karki N, Marshall MW. Oral fungal infections diagnosis and management. Dental Clinics of North America. 2017;61(2):319-349. DOI: 10.1016/j.cden.2016.12.004

[145] Lafaurie GI, Sabogal MA, Castillo DM, Rincón MV, Gómez LA, Lesmes YA, et al. Microbiome and microbial biomarkers of peri-implantitis: A systematic review. Journal of Periodontology. 2017;88(10):1066-1089. DOI: 10.1902/jop.2017.170123

[146] Camps-Font O, Figueiredo R, Valmaseda-Castellón E, Gay-Escoda C. Postoperative infections after dental implant placement: Prevalence, clinical features, and treatment. Implant Dentistry. 2015;24(6):713-719. DOI: 10.1097/ID.0000000000000325

[147] Esposito M, Grusovin MG, Worthington HV. Interventions for replacing missing teeth: Antibiotics at dental implant placement to prevent complications. The Cochrane Database of Systematic Reviews. 2013;31(7):CD004152. DOI: 10.1002/14651858.CD004152.pub4

[148] Ata-Ali J, Ata-Ali F, Ata-Ali F. Do antibiotics decrease implant failure and postoperative infections? A systematic review and meta-analysis. International Journal of Oral and Maxillofacial Surgery. 2014;43(1):68-74. DOI: 10.1016/j.ijoms.2013.05.019

[149] Sánchez FR, Andrés CR, Arteagoitia I. Which antibiotic regimen prevents implant failure or infection after dental implant surgery? A systematic review and meta-analysis. Journal of Cranio-Maxillofacial Surgery. 2018;46(4):722-736. DOI: 10.1016/j.joms.2018.02.004
[150] Garcés SMA, Escoda-Francolí J, Gay-Escoda C. Implant complications. In: Turkyilmaz I, editor. Implant Dentistry—The Most Promising Discipline of Dentistry. UK: InTech; 2011. pp. 369-396. DOI: 10.5772/19706

[151] Ucer C, Wright S, Scher E, West N, Retzepi M, Simpson S, et al. ADI Guidelines on Peri-implant Monitoring and Maintenance. 2013. Available from: www.adi.org.uk/resources/guidelines_and_papers/peri-implant/ [Accessed: Jul 24, 2018]

[152] Nandakumar V, Chittaranjan S, Kurian VM, Doble M. Characteristics of bacterial biofilm associated with implant material in clinical practice. Polymer Journal. 2013;45:137-152. DOI: 10.1038/pj.2012.130

[153] Pye AD, Lockhart DEA, Dawson MP, Murray CA, Smith AJ. A review of dental implants and infection. Journal of Hospital Infection. 2009;72:104e110. DOI: 10.1016/j.jhin.2009.02.010

[154] Do Nascimento C, de Albuquerque RF. Bacterial leakage along the implant-abutment interface. In: Implant Dentistry—The Most Promising Discipline of Dentistry. UK: InTech; 2011. DOI: 10.5772/20109

[155] Charalampakis G, Rabe P, Leonhardt A, Dahlen G. A follow-up study of periimplantitis cases after treatment. Journal of Clinical Periodontology. 2011;00:1-8. DOI: 10.1111/j.1600-051X.2011.01759.x

[156] Tallarico M, Canullo L, Caneva M, Özcan M. Microbial colonization at the implant-abutment interface and its possible influence on periimplantitis: A systematic review and meta-analysis. Journal of Prosthodontics. 2017;61(3):233-241. DOI: 10.1016/j.jpor.2017.03.001

[157] Zheng H, Xu L, Wang Z, Li L, Zhang J, Zhang Q, et al. Subgingival microbiome in patients with healthy and ailing dental implants. Scientific Reports. 2015;5(10948):1-11. DOI: 10.1038/srep10948

[158] Maruyama N, Maruyama F, Takeuchi Y, Aikawa C, Izumi Y, Nakagawa I. Intraindividual variation in core microbiota in peri-implantitis and periodontitis. Scientific Reports. 2014;4(6602):1-10. DOI: 10.1038/srep06602

[159] Smeets R, Henningsen A, Jung O, Heiland M, Hammacher C, Stein JM. Definition, etiology, prevention and treatment of peri-implantitis—a review. Head & Face Medicine. 2014;10:34. DOI: 10.1186/1746-160X-10-34

[160] Flemming HC, Wingender J, Szewzyk U, Steinberg P, Rice SA, Kjelleberg S. Biofilms: An emergent form of bacterial life. Nature Reviews Microbiology. 2016;14:563-575. DOI: 10.1038/nrmicro.2016.94

[161] Maisonneuve E, Gerdes K. Molecular mechanisms underlying bacterial persisters. Cell. 2014;157(3):539-548. DOI: 10.1016/j.cell.2014.02.050

[162] Posnick JC, Choi E, Chavda A. Surgical site infections following bimaxillary orthognathic, osseous genioplasty, and intranasal surgery: A retrospective Cohort Study. Journal of Oral and Maxillofacial Surgery. 2017;75(3):584-595. DOI: 10.1016/j.joms.2016.09.018

[163] Savage VJ, Chopra I, O’Neill AJ. Staphylococcus aureus biofilms promote horizontal transfer of antibiotic resistance. Antimicrobial Agents and Chemotherapy. 2013;57(4):1968-1970. DOI: 10.1128/AAC.02008-12

[164] Kulkarni R, Antala S, Wang A, Amaral FE, Rampersaud R, LaRussa SJ, et al. Cigarette smoke increases
Staphylococcus aureus biofilm formation via oxidative stress. Infection and Immunity. 2012;80:3804-3811. DOI: 10.1128/IAI.00689-12

[165] Wright JA, Nair SP. Interaction of staphylococci with bone. International Journal of Medical Microbiology. 2010;300:193-204. DOI: 10.1016/j.ijmm.2009.10.003

[166] de Mesy Bentley KL, Trombetta R, Nishitani K, Bello-Irizarry SN, Ninomiya M, Zhang L, et al. Evidence of Staphylococcus aureus deformation, proliferation, and migration in canaliculi of live cortical bone in murine models of osteomyelitis. Journal of Bone and Mineral Research. 2017;32(5):985-990. DOI: 10.1002/jbmr.3055

[167] de Oliveira-Neto OB, Timbó Barbosa FT, de Sousa-Rodrigues CF, de Lima JC. Quality assessment of systematic reviews regarding immediate placement of dental implants into infected sites: An overview. The Journal of Prosthetic Dentistry. 2017;117:601-605. DOI: 10.1016/j.prosdent.2016.09.007

[168] Friscia M, Sbordone C, Petrocelli M, Vaira LA, Attanasi F, Cassandro FM, et al. Complications after orthognathic surgery: Our experience on 423 cases. Oral and Maxillofacial Surgery. 2017;21:171-177. DOI: 10.1007/s10006-017-0614-5

[169] Almadih A, Al-Zayera M, Dabela S, Alhammadi MS, Mostafa YA. Success rates and factors associated with failure of temporary anchorage devices: A prospective clinical trial. Journal of Investigative and Clinical Dentistry. 2018;00:00-00. DOI: 10.1111/jicd.12331

[170] Aly SA, Alyan D, Fayed MS, Alhammadi MS, Mostafa YA. Success rates and factors associated with failure of temporary anchorage devices: A prospective clinical trial. Journal of Investigative and Clinical Dentistry. 2018;00:00-00. DOI: 10.1111/jicd.12331

[171] Smith KF, Goldberg M, Rosenthal S, Carlson L, Chen J, Chen C, et al. Global rise in human infectious disease outbreaks. Journal of the Royal Society Interface. 2014;11:20140950. DOI: 10.1098/rsif.2014.0950

[172] Salomon D, Heidel RE, Kolokythas A, Miloro M, Schlieve T. Does restriction of public health care dental benefits affect the volume, severity, or cost of dental-related hospital visits? Journal of Oral Maxillofacial Surgery. 2017;75:467-474. DOI: 10.1016/j.joms.2016.10.019

[173] Barenghi L, Barenghi A, Di Blasio A. Infection Control in Dentistry and Drug Resistant Infectious Agents: A Burning Issue. Part 2. UK: InTech; 2018

[174] Barenghi L, Barenghi A, Di Blasio A. Infection Control in Dentistry and Drug Resistant Infectious Agents: A Burning Issue. Part 2. UK: InTech; 2018

[175] Degeling C, Johnson J, Iredell J, et al. Assessing the public acceptability of proposed policy interventions to reduce the misuse of antibiotics in Australia: A report on two community juries. Health Expectations. 2018;21:90-99. DOI: 10.1111/hex.12589
Infection Control in Dentistry and Drug-Resistant Infectious Agents: A Burning Issue. Part 1
DOI: http://dx.doi.org/10.5772/intechopen.80961

[178] Rennert-May E, Conly J, Lea J, Smith S, Manns B. Economic evaluations and their use in infection prevention and control: A narrative review. Antimicrobial Resistance and Infection Control. 2018;7(31):1-6. DOI: 10.1186/s13756-018-0327-z

[179] Gao Q, Sui W. The function of nursing management for stomatology clinic infection. Journal of Nursing and Health Studies. 2017;2(1):1-4. DOI: 10.21767/2574-2825.100008