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Editorial

By Dr David Muscat

Dear colleagues,

At the time of writing this article the following events are envisaged.

This month’s front cover features a picture of a toothless eel, taken off the Great Barrier Reef of Australia, kindly supplied by Dr Dan Keir.

We would like to take this opportunity to wish all our readers a very happy Christmas and a prosperous New Year!

Best regards,

David

Dr David Muscat B.D.S. (I.ON)
Editor, Vice President and P.R.O. DAM

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THE DAM SANOFI AVENTIS

THE DAM SANOFI AVENTIS

RECENT/PLANNED EVENTS

1 NOVEMBER
‘Fossils’ lecture by Dr Charles Galea Bonavia at MFPB with dinner at Café Jubilee sponsored by Abbott.

2 NOVEMBER
‘Simpler Implants’ Cherubino event at Cavalieri Hotel

9 NOVEMBER
‘Smile For Health’ at Le Meridien Hotel.

20 NOVEMBER
11am mass in Madliena followed by wine tasting at Linos.

22 NOVEMBER
‘Odontogenic And Non-Odontogenic Toothache’ by Dr Dan Keir at Rogantinos in Landrijet sponsored by Novartis (Catafast).

1 DECEMBER
‘Advantages In Using 3D Diagnostics For Implant Treatment Planning’ by Simplant/Bart Enterprises at Radisson.

12 DECEMBER
DAM Christmas Party at Palazzo DePiro Mdina

THE DAM SANOFI AVENTIS QUIZ AT LO SQUERO

1. What are the top 5 places in the world, besides Rio de Janeiro to experience the Lenten Carnival?
   NADUR in Gozo
   Korner in Croatia
   Victoria in the Seychelles
   Madeira (Portugal)
   Oruro, Bolivia

2. What exactly does Rodogy1 contain?
   Metronidazole and Spiramycin

3. In the middle ages, in England and France it was decided that 14th February was to be Valentine’s Day. There was a reason for this pertaining to nature. What was it?
   Birds pair off around 14th February

4. What exactly does Solpadol contain?
   Paracetamol and Dihydrocodeine

Winner Dr Matthew Cachia

BIAL DAM QUIZ

Bial is represented by Associated Drug Co. in Malta and has kindly sponsored the event.

1. Who designed St. Pauls Cathedral in Mdina?
   Lorenzo Gafa

2. Who was the first bishop of Malta?
   St Publius

3. This first bishop then went on to become the bishop of another city in another country. Which city was this?
   Athens

4. In St. Pauls Cathedral, which famous artist painted ‘The Conversion Of Saint Paul’?
   Mattia Preti

5. How long can a snail hibernate for?
   3 years

6. How many festi are celebrated in Malta and Gozo each year?
   90

7. In which town do hunters traditionally shoot a gun salute as the statue is carried out of the parish church?
   St Julians

8. The word ‘Imnarja’ is derived from which word?
   Luminara

9. How many Auberges are there in Valletta?
   Six

10. How many tons does the Rinella Gun weigh?
    100 tons
THE COUNCIL OF THE EUROPEAN UNION:

Having regard to the Treaty on the Functioning of the European Union;


Having regard to the proposal from the European Commission;

WHEREAS:

(1) The use of hydrogen peroxide is already subject to restrictions and conditions laid down in Annex III, Part 1 to Directive 76/768/EEC.

(2) The Scientific Committee on Consumer Products, which has been replaced by the Scientific Committee on Consumer Safety (hereinafter SCCS) pursuant to Commission Decision 2008/721/EC of 5 August 2008 setting up an advisory structure of Scientific Committees and experts in the field of consumer safety, public health and the environment and repealing Decision 2004/210/EC, has confirmed that a maximum concentration of 0.1% of hydrogen peroxide present in oral products or released from other compounds or mixtures in those products is safe.

(3) The SCCS considers that the use of tooth whitening or bleaching products containing more than 0.1% and up to 6% of hydrogen peroxide present or released from other compounds or mixtures in these products may be safe if the following conditions are satisfied. An appropriate clinical examination is necessary to ensure the absence of risk factors or other oral pathology of concern and the exposure to these products should be limited in a manner that ensures that the products are used only as intended in terms of frequency and duration of application. These conditions should be fulfilled in order to avoid reasonably foreseeable misuse.

(4) Therefore, those products should be regulated in a way that ensures that they are not directly available to the consumer. For each cycle of use of those products, the first use should be limited to dental practitioners as defined under Directive 2005/36/EC of the European Parliament and of the Council of 7 September 2005 on the recognition of professional qualifications or under their direct supervision if an equivalent level of safety is ensured. Dental practitioners should then provide access to those products for the rest of the cycle of use.

(5) An appropriate labelling regarding the concentration in hydrogen peroxide of the tooth whitening or bleaching products containing more than 0.1% of this substance should be provided for in order to ensure the appropriate use of these products. For this purpose, the exact concentration in percentage of hydrogen peroxide present or released from other compounds and mixtures in those products should be clearly indicated on the label.

(6) Directive 76/768/EEC should therefore be amended accordingly.

(7) The Standing Committee on Cosmetic Products has not delivered an opinion within the time-limit laid down by its Chairman.
HAS ADOPTED THIS DIRECTIVE:

ARTICLE 1
Annex III to Directive 76/768/EEC is amended in accordance with the Annex to this Directive.

ARTICLE 2
Transposition

1. Member States shall adopt and publish, by [Day, Month, Year = 12 months after the publication of the Directive] at the latest, the laws, regulations and administrative provisions necessary to comply with this Directive. They shall forthwith communicate to the Commission the text of those provisions. They shall apply those provisions from [Day, Month, Year = 12 months + 1 day after the publication of the Directive]. When Member States adopt those provisions, they shall contain a reference to this Directive or be accompanied by such a reference on the occasion of their official publication. Member States shall determine how such reference is to be made.

2. Member States shall communicate to the Commission the text of the main provisions of national law which they adopt in the field covered by this Directive.

ARTICLE 3
This Directive shall enter into force on the twentieth day following that of its publication in the Official Journal of the European Union.

ARTICLE 4
This Directive is addressed to the Member States.

Done at Brussels,
For the Council
The President

For further details kindly email iro@dam.com.mt

4. Date to be included.
5. Date to be included.

BDA pleased to see useful contribution to amalgam debate

11 October 2011

The British Dental Association (BDA) is pleased to see the publication of a World Health Organization (WHO) report on the use of different materials in dental fillings. The report, Future Use of Materials for Dental Restorations, reflects a November 2009 meeting at WHO’s Geneva headquarters which considered environmental and health factors arising from the use of different filling materials.

The report says that it may be prudent to consider a phasing-down, rather than a phasing-out, of the use of dental amalgam and calls for a multi-pronged strategy with short-, medium- and long-term elements. It also contends that the quality of alternatives to amalgam must be further improved for use in public dental care, arguing that a progressive move away from amalgam would be dependent on that quality improvement being achieved.

Stuart Johnston, Chair of the BDA’s Representative Body and a member of the FDI World Dental Federation Dental Amalgam Task Team, said: “This is a thorough report that provides a balanced view of the use of different filling materials in dentistry and will make a useful contribution to the ongoing debate in this area.

“Dentists find amalgam to be a stable material for fillings, with good handling properties. Expert toxicologists and medics have reported no evidence that it causes harm to patients. The alternative materials that are available are not so well proven and have their own disadvantages.

“The environmental risks around amalgam use are taken extremely seriously and modern disposal processes are very sophisticated in preventing mercury emissions. In the long term, of course, the aim should be that preventive care advances sufficiently so that the need for fillings is diminished.

“In the meantime, it is important that the potential problems with, and likely impact of, any change in policy are fully considered by the experts and competent authorities who make decisions about the use of dental amalgam and other fillings materials.”

Further details are available at www.bda.org/amalgam.

The British Dental Association (BDA) is the professional association for dentists in the UK. It represents 23,000 dentists working in general practice, in community and hospital settings, in academia and research, and in the armed forces. It also includes dental students.

For further information, please contact the BDA’s media team on 0207 563 4145/46 or visit http://www.bda.org/news-centre/. You can also follow news from the BDA on Twitter: http://twitter.com/#!/TheBDA.
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For years, desensitising toothpastes have only treated dentine hypersensitivity. Now the debate is moving on: how can we go further than just treating the pain, to give patients continuous repair and substantive daily protection?

Sensodyne Repair & Protect: going beyond pain treatment in dentine hypersensitivity

Sensodyne has responded with the development of Sensodyne Repair & Protect. This new arrival brings you the unique potential of NovaMin®, advanced calcium phosphate technology in a daily fluoride toothpaste.

The difference is in the layer

NovaMin® is progressive science because it helps builds a reparative hydroxyapatite-like layer over exposed dentine and within the tubules.2-7

This layer formed by Sensodyne Repair & Protect starts to form from the first use,2,3,6,8 and can withstand daily oral challenges such as toothbrushing and acidic food and drinks.2,5,6,9

In this way, it can help provide your patients with continual protection from the pain of dentine hypersensitivity with twice-daily brushing.10-12

Welcome to the new science of Sensodyne Repair & Protect

Sensodyne Repair & Protect

Specialist in dentine hypersensitivity management

References:

CPT approval Z-10-176.
Royal College of Surgeons of England awards Honorary Fellowship to Prof. George Camilleri

On Friday 28th October 2011 Miss Kathryn Harley, Dean of Faculty of Dental Surgery, Royal College of Surgeons of England awarded Prof George Camilleri an Honorary Fellowship in Dental Surgery, Royal College of Surgeons of England.

Prof. Camilleri was also invited to deliver the Bradlaw Oration Lecture titled “Bradlaw and Dentistry in the British Commonwealth”. During the award ceremony Prof Angus Walls read a citation in honour of Prof Camilleri and reproduced below is a part of his citation

“George Camilleri combines in depth knowledge and humility. Scores of dental graduates have benefited from these qualities. Always supportive of his juniors, allowing them to develop in their own way and encouraging them to develop their talents, George brings out the best in most people. Dedicated to his present and former students, George Camilleri is to Maltese dentistry not a passing phase but an era.”

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References
6. Qin (Dong Kweck) 1703 1-8, 1585.
Consensus statement on antimicrobial treatment of odontogenic bacterial infections

Published in Oral Medicine and Pathology 2004;9:363-76. Accepted for publication: July 2005.

INTRODUCTION

Although there is very little data regarding the incidence of infections of the oral cavity, no one doubts their relevance. Of these types of infections, odontogenic infections (infections that involve tooth and periodontal tissues) are the most frequent. It is the most frequent reason for seeking odontological consultation and intervention and it affects the entire population from childhood (especially cavities) throughout a person's entire lifespan (periodontitis, implant complications, etc.), which entails a considerable impact both on public health in general, as well as the economic resources destined to maintain public health. It has been estimated that odontogenic infections in Spain represent approximately 10% of all antibiotic prescriptions(1,2).

Scientific evidence has revealed a relationship between some serious oral infections and specific systemic cardiovascular(3), lung and endocrine (diabetes mellitus) diseases, as well as with alterations during pregnancy(4,5).

Because of this association between infection and other systemic diseases, it is essential that odontogenic infections be avoided as much as possible or failing that, that they be identified and treated promptly and appropriately.

On occasion, an odontogenic infection can spread and provoke polymicrobial infections in other locations, such as the paranasal sinuses (odontogenic maxillary sinusitis), cervicofacial subaponeurotic spaces, palate, central nervous system (cerebral abscess), endocardium (endocarditis), etc.(6).

However, and despite the frequency and importance of odontogenic infections, when undertaking a review of the literature, the dispersion of criteria in key aspects such as terminology, classification, treatment recommendations, etc. is surprising, as is the paucity of papers in prestigious publications, making it impossible to establish an appropriate level of scientific evidence.

Hence, this document is the result of bibliographic review, but, above all, it represents the fruit of the experience accumulated over many years of the participating specialists and of the group discussions held for the purpose of drafting it. The main objective of this document, which has been elaborated by specialists representing 10 public universities in Spain in collaboration with specialists in the microbiology of these kinds of infections, is none other than to establish recommendations that will be of use for all those involved in the daily clinical management of patients suffering from these diseases.

CLASSIFICATION OF ODONTOGENIC INFECTIONS OF THE ORAL CAVITY

Infections of mixed aetiology affecting the oral cavity can be classified into two main groups on the basis of origin:

a) Odontogenic cavities, pulpitis, periapical abscess, gingivitis, periodontitis, periconoritis, ostitis, and infection of the subaponeurotic spaces; and

b) Non-odontogenic infections of the oral mucosa, infections of the salivary glands, etc.(7).

In 1999, the American Academy of Periodontology organized an international task force to create a classification of periodontal diseases and conditions (8) in response to the criticisms of previous classifications (9) (obscure diagnostic criteria, overlapping of disease-related groups, too much importance given to the patient's age, onset of illness and rate of progression, which are often difficult to determine). The odontogenic infections that present most frequently would be those that result from dental cavities, dental periodontal infections (infections of the pulpa and periapical abscesses), gingivitis (including necrotising ulcerative gingivitis), periodontitis (including periconoritis and the periimplantitis), infections of the sub-aponeurotic spaces, ostitis, and osteomyelitis.

WHAT ARE THE MOST IMPORTANT MICROORGANISMS IN ODONTOGENIC INFECTION?

The oral cavity is a complex ecosystem made up of more than 500 bacterial species (10). Overall, the Streptococcus, Peptostreptococcus, Veillonella, Lactobacillus, Corynebacterium and Actinomyces genera represent more than 80% of all cultivable flora (11). In the aetiology of periodontal disease, a whole series of species such as Actinobacillus actinomycetemcomitans, Porphyromonas gingivalis, Prevotella intermedia and Tannerella forsythensis can be especially highlighted due to their frequency and the importance of the complications that may arise from them.

Facultative gram-negative bacilli are uncommon in healthy adults and are seen almost exclusively in elderly, hospitalised patients with serious medical diseases (12). The polymicrobial nature of odontogenic infection has been demonstrated in many papers. For example, in a study conducted by Brook et al.(13) in 32 patients with periapical abscesses, 78 bacterial isolates were obtained (55 anaerobic and 23 aerobic), with a mean of 2.4 isolates per sample. Only anaerobic bacteria were found to be present in 16 patients (50%), only aerobic in 2 (6%) and mixed aerobic and anaerobic flora, in 14 (44%). The main isolates consisted of bacteria belonging to the Peptostreptococcus, Prevotella and Porphyromonas genera. Of the facultative anaerobic bacteria, oral streptococci are the most frequent. Table 1 shows the most commonly found bacteria in each oral condition.

Continues on the next page.
Consensus statement on antimicrobial treatment of odontogenic bacterial infections

Continues from the previous page.

WHEN IS COMPLEMENTARY DIAGNOSTIC TESTING INDICATED?
The diagnosis of odontogenic infection is based on anamnesis, observation and examination that allows symptoms and signs to be recorded. Information regarding the patient’s history of the following conditions is essential, as it will necessarily influence treatment and prophylaxis: endocarditis, implants, diabetes, immunodepression, etc. Radiological diagnosis is fundamental in determining the location, extension and possible complications of these lesions.

The role of the laboratory in diagnosing odontogenic infections in routine practice in dentists’ offices is controversial. Non-specific analytical data (leucocytes, complement, lymphocytes, immunoglobulins, glycaemia, etc.) must be requested when dealing with repeated or unusual infections or infections that are suspicious of any underlying disease that can have repercussions in the oral cavity.

Better still, the internist’s report should be requested before undertaking any kind of action. The patient can be spared serious medical complications and the professional can avoid legal complications. Bear in mind conditions such as endocarditis, diabetes, AIDS, hepatitis, etc. Insofar as microbiological studies are concerned, pathology samples will be taken prior to commencing with antibiotic treatment and will be sent to the laboratory following proper standards. The rapid techniques currently on the market can be a great diagnostic aid. The microbiological diagnosis seeks to rule out a specific aetiology, identify the aetiology of the condition and obtain overall information that is currently lacking, as well as to determine sensitivity to antimicrobial agents. These data will be useful in deciding on the treatment to be administered, whether to effect a change in the event that the empirical treatment fails and to establish general empirical therapies.

<p>| TABLE 1: MICROORGANISMS INVOLVED IN MIXED BACTERIAL INFECTIONS OF THE ORAL CAVITY (62) |</p>
<table>
<thead>
<tr>
<th>Infection process</th>
<th>Predominant bacteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Caries</td>
<td>Streptococcus mutans</td>
</tr>
<tr>
<td></td>
<td>Actinomyces spp</td>
</tr>
<tr>
<td>gingivitis</td>
<td>Campylobacter rectus</td>
</tr>
<tr>
<td></td>
<td>Actinomyces spp</td>
</tr>
<tr>
<td>Endodontitis</td>
<td>Porphyromonas gingivalis</td>
</tr>
<tr>
<td></td>
<td>Bacteroides forsythus</td>
</tr>
<tr>
<td>periapical abcess</td>
<td>Porphyromonas gingivalis</td>
</tr>
<tr>
<td>endodontitis</td>
<td>Actinobacillus actinomycetemcomitans</td>
</tr>
<tr>
<td>endodontitis</td>
<td>Prevotella intermedia</td>
</tr>
<tr>
<td>endodontitis</td>
<td>Fusobacterium nuc leatum</td>
</tr>
</tbody>
</table>

**THERAPEUTIC MANAGEMENT OF ODONTOGENIC INFECTION**

The issue of odontogenic infection must be approached from three, mutually complementary treatment areas. Aetiological odontological treatment, which often includes surgical interventions of varying magnitude and requiring different levels of professional expertise; systemic support treatment, which covers a broad spectrum ranging from symptomatic pain management and controlling the inflammation, all the way to physical measures, hydration, fever control, glycaemic control, etc.

Finally, antimicrobial treatment should only be applied on rare occasions and on the basis of rational, efficiency criteria. In general, antimicrobial treatment must be initiated whenever the condition presents clear clinical manifestations of infection.

Antimicrobial treatment of odontogenic infections aims to prevent local spread and spread to neighbouring areas, to decrease the bacterial inoculum in the infectious focus and to prevent complications derived from dissemination via the circulatory system (14,15). Antimicrobial treatment is not the only treatment option for odontogenic infection, since antibiotic administration alone is often not sufficient to eradicate the infection. Depending on the infection and the patient’s characteristics, the optimum treatment for a given infection may require systemic or local antimicrobial agents, odontological treatment or surgery, or a combination of the above (6,16,17).

**IN WHAT SITUATIONS IS ANTIMICROBIAL TREATMENT WARRANTED?**

Not all odontogenic infections require antimicrobial treatment. In some cases, surgical treatment is also necessary and in others, the best course of treatment is debridement, irrigation and drainage. Endodontic Infections Arising from the Pulpa In some situations, acute endodontic treatment can be complemented with systemic antibiotics, as well as with analgesics and/or anti-inflammatory drugs (18). Antibiotics are also indicated in cases in which the patient is immunodepressed and requires prophylaxis.

**Chronic Gingivitis and Necrotising Ulcerative Gingivitis (NUG)**

Generally speaking, the treatment of mild gingivitis does not include systemic antibiotic administration. It requires local treatment to eliminate dental plaque and to disinfect the gingival grooves. Useful measures include rinsing with chlorhexidine, brushing with a mixture of sodium bicarbonate and hydrogen peroxide, and/or frequent rinsing with saltwater. One exception is NUG, in which systemic antibiotic use is recommended. The same is true of streptococcal gingivitis, caused by group A beta haemolytic streptococcus (Streptococcus pyogenes) that presents as a complication of acute streptococcal pharyngitis/tonsillitis, in which active antibiotics should be used against this microorganism (19). In the case of NUG, in addition to antibiotic treatment, debridement with ample irrigation is recommended (16). Topical application of mouthwash containing chlorhexidine.
or saline solution is effective in controlling the pain and ulceration that accompanies this condition.

**Periapical Abscess**
This comprises a clear indication for debridement and surgical drainage complemented with systemic antibiotics.

**Periodontal Abscess**
Treatment consists of debridement and draining the purulent pocket. Antibiotic treatment is reserved for those situations where there is local or systemic dissemination.

**Periodontitis**
Debridement, elimination of the calculus and root planning to remove subgingival plaque deposits constitutes the first line of treatment. Subgingival irrigation should also be performed using ultrasound tartar removal equipment to disinfect the gingival sulcus. Other useful measures consist of rinsing the mouth with chlorhexidine or brushing with a mixture of sodium bicarbonate and hydrogen peroxide. Systemic antibiotics are indicated especially for the treatment of aggressive periodontitis (16).

**Pericoronitis**
Systemic antibiotics are almost always necessary to keep the infection from spreading. Local treatment consisting of debridement, irrigation and drainage of the affected areas, or even tooth extraction can also be performed.

**Periimplantitis**
Systemic antibiotic therapy in certain cases may be accompanied by mechanical debridement. Rinsing the mouth with chlorhexidine for 30 seconds after brushing teeth may also be useful as coadjuvant treatment (20).

**Severe Infections of the Fascia and Deep Head and Neck Tissues**
The treatment of infections located in the cervicofacial aponeurotic spaces include the following measures: 1) aetiological treatment, 2) incision, debridement and drainage of purulent accumulations and 3) antibiotic therapy. Odontogenic infections are caused by a highly predictable group of bacteria, so the first choice of antibiotic is made empirically. However, if evolution is unfavourable, the antibiotic chosen can be substituted by another one or more than one after identifying the causal microorganisms by means of culture and antibiogram typing. 4) Finally, complementary systemic care is also required (hydration, nutritional support, analgesics, antipyretics and anti-inflammatory drugs). Attention must be paid at all times to aetiological treatment criteria that indicate the need to transfer the patient to a hospital, possibly on an emergency basis (Table 2).

**WHAT CHARACTERISTICS MUST THE IDEAL ANTIBIOTIC HAVE FOR THE TREATMENT OF ODONTOGENIC INFECTION?**
The ideal antibiotic for treating an infection must have a series of characteristics such as: a) it must be active against the microorganisms involved in the infection; b) it must meet appropriate pharmacokinetic parameters (good penetration and diffusion at the site of infection); c) it must be well tolerated and have a low incidence of side effects (21), and d) it must allow for a dosing schedule that facilitates treatment compliance. The ploymicrobial component of odontogenic infection advises the use of antibiotics that are active against both aerobic and anaerobic bacteria are recommended, requires that the proper antibiotic be used for treatment. It is often necessary to administer combinations of antibiotics that can achieve a spectrum of activity and are more appropriate to the type of infection.

**HOW SENSITIVE ARE THE PATHOGENS INVOLVED IN ODONTOGENIC INFECTION TO THE MOST COMMONLY USED ANTIMICROBIAL AGENTS?**
The increased prevalence of bacterial resistance means that antibiotics that have been useful in the past are currently no longer as effective as they once were, as is the case with certain dose levels. In this regard, in the last 10-15 years the number of resistant microorganisms in the oral cavity has doubled (22). We can cite the following example: studies have revealed the presence of beta-lactamase producing species in 74-88% of patients with periodontitis (23-24). Likewise, over the course of recent years as seen with other pathogens such as Streptococcus pneumoniae, the levels of resistance to macrolides, beta-lacta-mase and clindamycin of several viridans group streptococid species have increased notably (25-28). Whereas an increase in the macrolide doses does not lead to increased coverage against the resistant strains, in the case of beta-lactam, higher doses can lead to better coverage (29). Table 3 shows the activity of several antimicrobial agents against the most important microorganisms that cause periodontal disease.

**WHICH ANTIBIOTICS AND WHAT DOSES ARE ADEQUATE FOR TREATING ODONTOGENIC INFECTION?**
Treatment duration with antibiotics depends on the type of infection, the extension of the condition and on the antibiotic chosen. Overall, treatment duration will vary between 5 and 10 days; in other words, treatment should continue for 3 or 4 days after clinical manifestations have disappeared (30).

Amoxicillin, amoxicillin/ clavulanate, cephalosporins, doxycycline, metronidazole, clindamycin and macrolides, such as erythromycin, clarithromycin and azithromycin, all stand out amongst the large variety of systemic antimicrobials used to treat odontogenic infection. Tables 4 and 5 present antimicrobial agents and the dosing schedules recommended for each indication. Penicillins, Penicillin, ampicillin and amoxicillin are bacteicidies that are useful in treating the acute phase of odontogenic infection, in addition to preventing associated complications (7). Due to their effectiveness against facultative aerobic and anaerobic pathogens, they are considered to be the antibiotics of choice in the treatment of infections of mixed aetiology in the oral cavity (31). However, there are more and more beta-lactamase producing bacteria, enzymes that are capable of hydrolysing penicillins and, therefore, leading to treatment failure (32-34) particularly when strains of the Prevotella,

Continues on the next page.
Porphyromonas and Fusobacterium genera are present (35-37). In fact, penicillin administration has been linked to the appearance of beta-lactamase producing bacilli in the oropharynx (38,39). Amoxicillin and ampicillin increase penicillin’s spectrum to cover enteric gram-negative bacilli. Amoxicillin is better than ampicillin because of its superior enteric absorption (60-80% versus 30-55%) (40, 41).

Given the increased prevalence of beta-lactamase producing microorganisms, the association of a penicillin with a beta-lactamase inhibitor such as amoxicillin/clavulanic acid has become the treatment of choice in many of these conditions (42,43). The increased resistance of some species of oral streptococci indicates that high doses of amoxicillin be used to treat infections in which these pathogens might be involved.

In this regard, a new pharmacokinetically enhanced formulation of amoxicillin/clavulanate has been developed (amoxicillin/clavulanate, 1000/62.5 mg) that, in addition to lowering the number of daily doses to two, also eradicates strains considered to be resistant to conventional formulations (44-46).

Furthermore, this new formulation, when administered along with high doses of Amoxicillin, can delay or decrease the risk of increasing the prevalence rate of oral streptococci resistance, as seen in children with Streptococcus pneumoniae and a high dosis, short course of treatment with amoxicillin (5-7 days) (47,48). Cephalosporins Cephalosporins are classified in generations, based on their antibacterial spectra, regardless of when they were synthesised.

In general, as we move further along the generations, activity against gram-negative germs improves, while effectiveness against grampositive germs decreases (49). They present the disadvantage of having very poor activity against anaerobic bacteria, with the exception of cephapenic (cefoxitin, cefminox and cefotetan) for which there are no oral formulations (21). Tetracyclines Tetracyclines have classically been the standard antibiotic of use in treating odontogenic infection, although at present, they exert limited activity as a result of increased resistance, particularly in countries such as Spain where there is a high level of antimicrobial use (50).

Because of their high affinity for bone and dental tissue, its use is not recommended during pregnancy, while nursing or in children less than eight years of age, since when deposited on teeth and bones during development they can produce alterations such as dental hypoplasia, bone deformities and abnormal tooth colour (51).

### TABLE 3.- ACTIVITY OF DIFFERENT ANTIBACTERIAL AGENTS AGAINST PERIODONTOPATHOGENS (21).

<table>
<thead>
<tr>
<th>Antibiotic/CL</th>
<th>As. Actinomyces/Actinomycetemcomitans</th>
<th>Peptostreptococci spp</th>
<th>Prevotella spp</th>
<th>Porphyromonas spp</th>
<th>PropionIBacterium spp</th>
<th>Oral streptococci</th>
</tr>
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<tbody>
<tr>
<td>Penicillin G</td>
<td>≥</td>
<td>+</td>
<td>+</td>
<td>≥</td>
<td>+</td>
<td>+</td>
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<td>Amoxicillin</td>
<td>+</td>
<td>+</td>
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<tr>
<td>Amoxicillin/</td>
<td>+</td>
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<td>+</td>
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<td>Ar. Clavulanate</td>
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<td>+</td>
</tr>
<tr>
<td>Doxycycline</td>
<td>+</td>
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<td>+</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>O</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>O</td>
</tr>
<tr>
<td>Metronidazole</td>
<td>O</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>O</td>
</tr>
<tr>
<td>Macrolides</td>
<td>≥</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

* More than 80% of sensitive strains / O Less than 30% of sensitive strains / ± Between 30-50% of sensitive strains

### TABLE 4.- ANTIBIOTICS AND DESITOPSICS OF USE IN ODONTOGENIC INFECTIONS

<table>
<thead>
<tr>
<th>Odontogenic Infection</th>
<th>Drug of choice (oral and/or topical)</th>
<th>Alternative (oral and/or topical)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marginal gingivitis</td>
<td>Chlorhexidine</td>
<td>Clindamycin + chlorhexidine</td>
</tr>
<tr>
<td>Necrotising ulcerative gingivitis</td>
<td>Amoxicillin/clavulanate or amoxicillin + metronidazole + chlorhexidine</td>
<td>Clindamycin + doxycycline + chlorhexidine</td>
</tr>
<tr>
<td>Chronic periodontitis</td>
<td>Amoxicillin/clavulanate or metronidazole + chlorhexidine</td>
<td>Clindamycin + doxycycline + chlorhexidine</td>
</tr>
<tr>
<td>Aggressive periodontitis</td>
<td>Amoxicillin/clavulanate or metronidazole or oral doxycycline + chlorhexidine</td>
<td>Clindamycin or azithromycin or clarithromycin</td>
</tr>
<tr>
<td>Acute pulpitis</td>
<td>Amoxicillin/clavulanate</td>
<td>Clindamycin or azithromycin or clarithromycin</td>
</tr>
<tr>
<td>Periodical abscesses</td>
<td>Amoxicillin/clavulanate</td>
<td>Clindamycin or azithromycin or clarithromycin</td>
</tr>
<tr>
<td>Periodontal abscesses</td>
<td>Amoxicillin/clavulanate</td>
<td>Clindamycin or azithromycin or clarithromycin</td>
</tr>
<tr>
<td>Pericoronitis</td>
<td>Amoxicillin/clavulanate</td>
<td>Clindamycin or azithromycin or clarithromycin</td>
</tr>
<tr>
<td>Periapical abscesses</td>
<td>Amoxicillin/clavulanate</td>
<td>Clindamycin or azithromycin or clarithromycin</td>
</tr>
<tr>
<td>Ulcidulitis</td>
<td>Amoxicillin/clavulanate</td>
<td>Clindamycin or azithromycin or clarithromycin</td>
</tr>
</tbody>
</table>

* This table is indicative of the antibiotics used, which is not to say that they are limited in all cases.

### TABLE 5.- DOPING SCHEDULES OF DIFFERENT ANTIBIOTICS

<table>
<thead>
<tr>
<th>Antibiotic</th>
<th>Adult Doses</th>
<th>Pediatric Doses</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amoxicillin</td>
<td>1000 mg/8-12 hours</td>
<td>50 mg/Kg/day in 3 doses</td>
<td>In children, try another antimicrobial</td>
</tr>
<tr>
<td>Amoxicillin + Clavulanate</td>
<td>2000 mg + 125 mg/12 h</td>
<td>40-80 mg/Kg/day in 3 doses</td>
<td></td>
</tr>
<tr>
<td>Clindamycin</td>
<td>150-450 mg/6 hours</td>
<td>25 mg/Kg/day in 3-4 doses</td>
<td></td>
</tr>
<tr>
<td>Doxycycline</td>
<td>500-12 mg/12 hours</td>
<td>7.5-15 mg/Kg/day in 12 hours</td>
<td></td>
</tr>
<tr>
<td>Doxycycline</td>
<td>100 mg/12 hours</td>
<td>2 mg/Kg/day in 12 hours</td>
<td></td>
</tr>
<tr>
<td>Enrofloxacin</td>
<td>500-1000 mg/6 hours</td>
<td>50 mg/Kg/day in 3 doses</td>
<td></td>
</tr>
<tr>
<td>Metronidazole</td>
<td>500-750 mg/8-12 hours</td>
<td>45 mg/Kg/day in 3 doses</td>
<td></td>
</tr>
<tr>
<td>Azithromycin</td>
<td>500 mg/day for 3 consecutive days</td>
<td>10 mg/Kg/day for 3 consecutive days</td>
<td></td>
</tr>
</tbody>
</table>

* Continues from page 30.
THE APPROPRIATE SOLUTION FOR EACH SITUATION

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- Preventive agent in the treatment of gingivitis
- Oral care before and after surgery

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<table>
<thead>
<tr>
<th>mm</th>
<th>0.4</th>
<th>0.45</th>
<th>0.5</th>
<th>0.6</th>
<th>0.7</th>
<th>0.8</th>
<th>1.1</th>
<th>1.3</th>
</tr>
</thead>
<tbody>
<tr>
<td>ISO</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
</tr>
</tbody>
</table>

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The nasal cavity infection spreads posteriorly causing a post nasal rhinitis often causing pain on swallowing. Infected producing a sore throat the pharyngeal mucosa becomes causing some conjunctivitis. Also the infection spreads up the lachrymal ducts into the eyes nasal stuffiness and blockage. Watery fluid changing within a few hours to thick mucus which causes itching in the nose and sneezing. This condition is the formation of grape like watery structures arising from the nasal mucosa. A common cause is chronic allergic rhinitis secondary to multiple attacks of acute allergic rhinitis. Sometimes there is a family history of nasal polyps. Pre-existing chronic sinusitis and bronchial asthma are also predisposing causes.

The mast cells of the nasal mucosa already contain antibodies to the offending allergen. When the patient receives yet another dose of allergen, there is an inflammatory response in the nasal mucosa mast cells, consisting of the release of inflammatory mediators, such as histamine and leukotrienes.

These mediators stimulate the nerves, blood vessels and secretory glands of the lining of the nasal cavity to produce copious watery secretions, itching in the nose and sneezing. A second wave of mediators is released causing an influx of white cells, particularly eosinophils. The condition very quickly spreads up the lachrymal ducts to produce intense itching of the conjunctiva of the eyes. The itching is directly due to the histamine release. A post nasal drip into the pharynx often occurs, causing a desire to keep ‘clearing the throat’. The inflammatory response may well spread into the lining of the Eustachian tubes causing intermittent blockage, manifested by intermittent deafness which is relieved by yawning. Allergic rhinitis may become chronic if the allergen is not identified.

Unresolved chronic sinusitis may also be seen as the prodromal manifestation of childhood viral infections, such as measles.

Allergic Rhinitis
The patient suffers either repeated attacks of non infective rhinitis due to allergens, such as pollen, hay, and spores [known as seasonal allergic rhinitis] or permanent rhinitis from dust and pollutant allergens [known as perennial allergic rhinitis]. Certain foods, particularly of dairy origin, can cause an allergic rhinitis within minutes of ingestion.

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Chronic Non-Allergic Rhinitis
In this condition there is no allergic response in the mucosa. Usually the only symptom is the dripping of watery fluid from the nose from nasal congestion. Other patients complain of constant ‘throat clearing’ or ‘catarrh’. Itching eyes and nose are not present. Stress, hypothyroidism and chemical irritants have been associated with this condition.

Rhinitis Medicamentosa
There is no allergic response in the mucosa. Usually the only symptoms are permanent nasal stuffiness and obstruction due usually to mucosal swelling induced by the frequent and over enthusiastic use of medical nasal sprays. The long term use of certain medications, such as antihypertensives, beta blockers, oral contraceptives and antidepressants may cause this condition also.

Nasal Polyps
This condition is the formation of grape like watery structures arising from the nasal mucosa. A common cause is chronic allergic rhinitis secondary to multiple attacks of acute allergic rhinitis. Sometimes there is a family history of nasal polyps. Pre-existing chronic sinusitis and bronchial asthma are also predisposing causes.

Continues on page 16.
The polyps become so numerous and large that the nose is blocked and so the patient mouth breathes, and frequently complains of a ‘cold lasting for a year’ with constant dripping of watery fluid from the nose.

**Chronic Sinusitis Producing Polyps in the Sinuses and Nasal Cavity**

Unresolved acute infective sinusitis may well progress into chronic sinusitis where the mucosa of the walls of the sinuses becomes thickened with similar polyp formation.

This condition is associated with face pain, [often simulating dental pain], and a copious nasal mucous discharge. Spread of the polyps into the nasal cavity may occur, causing nose; stuffiness or obstruction. Occasionally, a polyp migrates from a maxillary antrum through an enlarged ostium into the nasal cavity to cause similar symptoms. This is known as an ‘antrochoanal polyp’ [1] caused by either antral infection or allergy.

**Enlarged and Swollen Inferior Turbinates**

The inferior turbinate bony structures in the nasal cavity are lined by a moist mucosa, vital for a correctly functioning sense of smell and pre-heating of cold air as it passes into the lungs.

Allergy and sinus infection are the two main causes of turbinate swelling. A third cause is due to a deviated nasal septum [of developmental or traumatic origin] which causes the inferior turbinate bone on the wide side to increase in size. All these causes lead to nasal obstruction with fluid dripping from the nose or post nasally.

**Deviated Nasal Septum**

This may be congenital or traumatic in origin leading to one sided nasal obstruction [on the narrowed of the nasal cavity] often blocking the sinus ostia on that side resulting in a unilateral obstructive sinusitis. [A tumour of the nasal cavity causing the same features needs to be excluded]. Face pain from the sinusitis is frequent.

**REACHING A DIAGNOSIS CLINICALLY**

The patient often presents at the stage where the disease process may have spread into an adjacent interconnecting structure which specifically produces its own set of symptoms.

Thus aetiological confusion occurs. The answer to this diagnostic dilemma is to take a full history of the onset of the initial symptoms. This should identify which structure was the site of the beginning of the disease process.

Certain symptoms may be disease specific. The common cold should easily be eliminated clinically-a very sore throat is diagnostic. Much itchiness around the eyes indicates allergy-not seen in other causes. Periodicity of symptoms suggests seasonal allergic rhinitis. The use of certain sprays and drugs medically suggests chronic non-allergic rhinitis or rhinitis medicamentosa.

A permanent ‘cold’ or blocked nose suggests the presence of nasal polyps or enlarged swollen turbinates.

Confusingly, the symptoms of some of these diseases are of face pain which may be wrongly identified as a dental problem. The normal invagination of the upper teeth and their sockets into the lower part of the maxillary antra enhances this mis-diagnosis. So, the patient with apparent dental symptoms should be checked for the alternative diagnosis of rhinitis in its many manifestations.

If medical treatment fails to work, or the cause cannot be identified clearly, then referral for an ENT opinion is advisable. This specialist may well ask for CT and perform FESS, both of which should be diagnostic.
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* For instant relief massage a small quantity directly on the sensitive tooth for one minute.
ODONTOGENIC AND NONODONTOGENIC TOOTHACHE

Daniel M. Keir, DDS Diplomate, American Board of Endodontics

Odontogenic and Nonodontogenic Toothache

Daniel M. Keir, DDS
Diplomate, American Board of Endodontics
Dental Association of Malta, 22 November 2011

Odontogenic Pain

• Pulpal Pain
• Periodontal Pain

Pain Referral

• Site versus Source

Site versus Source

• Site of pain is the location in which the patient feels the pain
• Source of pain is that area of the body from which the pain originates
• Primary Pain is when the site and source are in the same location
• Referred Pain is when the site and source are in different locations

Central Nervous System Sensitization

Myofascial Toothache

• Pain is nonpulsatile, constantly aching
• Lack of dental pathology to explain pain
• Pain not increased with stimulation of the tooth
• Pain increases with function of the involved muscle
• Local anesthesia of tooth does not relieve the pain

Neurovascular Toothache

• Pain is spontaneous, variable, and throbbing, similar to pulp pain
• Periods of remission and exacerbations. May be episodic, occur same time of day/week/month
• Lack of reasonable dental cause for pain
• If pain is posterior, may induce autonomic effects such as nasal congestion, lacrimation, edema of the eyelids and face
• Effect of local anesthesia is unpredictable
• Over time, the complaint spreads to involve wider areas of the face, neck, shoulder and may evoke muscle pain and restricted movement

Episodic Neuropathic Toothache

• Pain is unilateral, severe, lancinating, shock-like (paroxysmal)
• Generally has a trigger zone
• Local anesthesia of trigger zone prevents attacks
• Generally a lack of reasonable dental pathology to explain the pain

Continuous Neuropathic Toothache

• Pain felt in a tooth or a tooth site (max canine and premolar most frequent area)
• Pain is continuous ache or burning
• Pain has persisted for more than 4 months
• No reasonable dental condition to explain pain
• Local anesthesia is equivocal
• Known as phantom toothache, atypical odontalgia and atypical facial pain
**Pulpal Pain**
- Dull, aching, throbbing and occasionally sharp
- Rarely stays the same over time; gets worse or better
- Identifiable condition generally explains the symptoms
- Local anesthesia of affected tooth eliminates the pain

**Periodontal Pain**
- Dull, aching, or throbbing
- Identifiable periodontal condition
- Discomfort often felt with pressure. May feel sore or elongated
- Local anesthesia of affected periodontal tissues eliminates the pain

**Central Nervous System Sensitization**
- Primary pain produces nociceptive input that is transmitted to second order neurons in the trigeminal spinal tract nucleus and then onto the sensory cortex
- Protracted or intense input can centrally excite adjacent converging neurons in the trigeminal spinal tract nucleus and then relay additional nociception to the sensory cortex
- Referred pain therefore originates from deep pain arising from any structure that provides sensory convergence on the trigeminal spinal tract nucleus.
- Sesse and others in 1986 described the Convergence Theory of Pain Referral

**Types of Nonodontogenic Toothaches**
- Myofascial
- Neurovascular
- Cardiac
- Neuropathic
- Maxillary sinus/Nasal mucosa
- Psychogenic

**Toothache of Cardiac Origin**
- Aching pain in jaw or tooth is cyclic
- Toothache increased with physical activity
- Toothache associated with chest, arm or neck pain
- Toothache decreased with nitroglycerin tablets
- Stimulation of the tooth does not alter the pain

**Neuropathic Toothache**
- **Episodic**—Trigeminal Neuralgia
- **Continuous**—Phantom Toothache, Atypical odontalgia, Atypical facial pain

**Toothache of Psychogenic Origin**
- Reports of multiple teeth being painful with frequent change in character and location
- General departure from normal or physiologic pain patterns
- Lack of response to reasonable dental treatment or an unusual or unexpected response to therapy
- No identifiable pathology to explain the toothache
- Presents with chronic pain behavior.
ADULT ORTHODONTICS

WHAT'S THE ISSUES?

Form an ever increasing proportion of orthodontic patient cohort- approx. 44% of patient load in private practice in 2009.

Issues:
- Interdisciplinary care
- Restored dentition
- Periodontal disease
- Temporomandibular joint dysfunction
- Enamel wear

1

TM Dysfunction

- Adults are more likely to suffer from TMD
- Orthodontics for treatment of TMD cannot be guaranteed of success
- TMD symptoms often subside during treatment
- Following completion of treatment, TMD symptoms may return

2

Periodontal Disease

- Chronic periodontal disease far more prevalent in adults
- Adolescents have significantly more plaque and greater pocket depths than adults during treatment (Brown, 1992)

5

Extraction pattern

- Heavily restored teeth as well as teeth with a poor prognosis may have to be extracted instead of more favourable teeth
- This may increase treatment time and may complicate treatment mechanics
- Loss of bone in old extraction spaces hinders tooth movement as well as eruption of opposing teeth

6

Anchorage control

- Adults require greater anchorage control
- Headgear ruled out in most cases
  Alternatives include:
  Palatal arches, class II intermaxillary elastics and TAD's

8

Ceramic fixed appliances

- Give high bond strength and acceptable aesthetics
- Higher friction therefore increased treatment time
- Clear elastic modules may stain

9

Lingual appliances

- Becoming increasingly popular and demanded by patients as totally "invisible" appliance
- More problems with discomfort and speech at least in initial period
- Adjustments more technically demanding and difficult
- May increase anchorage requirements

11

10
**ODONTICS: DIFFERENCE?**

By Kevin Mulligan

---

**Growth**

- This is negligible in adults - use of functionals for class II correction ruled out and overbite reduction more difficult
- Only mild skeletal base discrepancies can be camouflaged

---

**Compromised dentition**

- Inadequate root treatments and non-vital teeth may complicate treatment
- Large restorations will affect extraction pattern or may cause problems with bonding of brackets
- Porcelain crowns and veneers would need etching with hydrofluoric acid and use of porcelain primer for bonding

---

**Appliance choice**

- Priorities for adults differ to those for adolescents
- In adults: aesthetics more important, cost less so
- Removable appliances are not easily tolerated
- Vacuum formed splints/aligners show better tolerance

---

**Aesthetic appliance systems**

- Invisalign® good to treat some more minor malocclusions
- Not good for extraction cases or more complex malocclusions
- Requires patient cooperation with wear

---

**Lingual appliances**

- STB system uses individual brackets for different teeth with varying bonding cement thicknesses to compensate for different tooth thicknesses
- Incognito® appliance is totally custom-made for patient and includes custom-made archwires
- Increased cost
- Use indirect bonding with set up of teeth

---

**Conclusion**

- Adult orthodontics is becoming increasingly mainstream
- Most will require increased periods with retention - may be indefinite
- Interdisciplinary care more often required
- Dentist cannot adopt authoritarian approach with adults
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Specialist in dentine hypersensitivity management

---

Efficiency of SOREDEX DIGORA® Optime UV disinfection system

Ari-Pekka J. Huovila, PhD Principal investigator
Institute of Medical Technology
University of Tampere and Tampere University Hospital

OBJECTIVES
The aim of this study was to evaluate the efficiency of ultraviolet (UV-C) disinfection of harmful microbes on the surfaces of the imaging plate carrier of the SOREDEX DIGORA® Optime intraoral imaging plate system.

METHODS
A literature research was done to evaluate the sensitivity of the pathogenic microbes to ultraviolet light at UV-C (λ < 280 nm) wavelengths emitted by the UV-disinfection system inside the DIGORA® Optime imaging plate reader. The technical characteristics of the UV-C system were provided by SOREDEX.

RESULTS
The calculated germicidal efficiency of UV-C radiation, equivalent to UV light emitted by the UV light source within the DIGORA® Optime imaging plate reader, is at least 99.9 % for the pathogenic microbes of primary interest.

CONCLUSIONS
UV radiation used in the DIGORA® Optime UV-disinfection system is 99.9% effective in reducing the number of harmful pathogens of primary interest. The elimination of pathogenic microbes efficiently reduces the theoretical risk of cross-contamination via the imaging plate reader and imaging plates. Together with other Opticlean™ features the SOREDEX DIGORA® Optime offers a comprehensive means of ensuring uniquely high level of hygiene for practical workflow in clinical dentistry.

INTRODUCTION
Ultraviolet-C light (UV-C, λ < 280 nm) is known to be effective in the disinfection of suspensions and surfaces of the area irradiated with an appropriate radiation dose11. UV-C irradiation eliminates the infectivity by disrupting the genomic DNA/RNA, thus rendering the cells and viruses unable to grow and reproduce. This review summarizes the experimental studies investigating the kinetics of the UV-C inactivation of known pathogens, with particular emphasis on clinical dentistry.

Microbes of primary interest, (Corynebacterium diphtheriae, Mycobacterium tuberculosis, Hepatitis viruses A, B, and C, Herpes simplex virus I, and Human immunodeficiency virus) are a significant hygiene concern in dental practices. Specific safety measures must be carried out when patients with known infections are being treated. Importantly, precautionary actions are also needed as patients may be unaware or unwilling to inform about any infection they may have.

These actions include the disinfection of all visible surfaces in the dental office. The internal parts of the imaging plate readers often remain unattended due to access difficulties.

The unique SOREDEX DIGORA® Optime UV disinfection system has been developed to minimize cross-contamination even in the event of inadequate hygiene precautions during the handling of the imaging plates. The Table 1 lists the UV-C inactivation data for pathogens of concern in clinical dentistry, and shows that the irradiation doses used in the DIGORA® Optime, efficiently eliminate the pathogens of primary as well as secondary interest.

SOREDEX Opticlean™ concept
The Opticlean™ concept focuses on ensuring a uniquely high level of hygiene in the use of the DIGORA® Optime imaging plate system in all environments. The Opticover™ imaging plate protective covers and the Optibag™ imaging plate hygiene bags provide an effortless end-to-end hygiene workflow in dental offices.

The Opticlean™ concept also includes touchless operation of the imaging plate reader to reduce the risk of cross contamination via the outer parts of the system as well as a UV-C system that irradiates the internal parts of the reader which are in contact with the imaging plate (the imaging plate carrier).

The frequency of the UV-C treatment can be easily set by the user according to the risk level. Each treatment consists of a 250s UV-C exposure period on the plate carrier surfaces in contact with the imaging plate.

The UV-C lamp with a peak wave length of 253.7 nm and output power of 160 mW is positioned 31mm away from the imaging plate carrier to provide the most efficient level of irradiation. The measured dose at the imaging plate carrier is at least 450 μW/cm² (i.e., 112 mJ/cm²).

Continues on page 25.
Hydrorise is the first hyperhydrophilic A-Silicone for any impression technique and for all types of clinical situations. Hyperhydrophilic technology has been exclusively developed by Zhermack research, making hydrorise extremely flowable like water.

With a contact angle less than 10° and thanks to the effects of the AMDA System (Advanced Moisture Displacement Action), Hydrorise is able to move fluids from the gingival sulcus and reproduce impression margins with maximised precision, like no other material on the market. The perfect synergy between its physical and mechanical properties ensure extremely accurate and reliable results, even in particularly difficult clinical situations.
Efficiency of SOREDEX DIGORA® Optime UV disinfection system

RESULTS AND DISCUSSION
This study is based on information that SOREDEX has disclosed about its UV-C disinfection system and on published scientific results. Table 1 summarizes the UV-C inactivation kinetics of the selected pathogens with references to the original research. Under the disclosed conditions and based on the published and well established research, the UV-C radiation dose used by the Opticlean™ system of the DIGORA® Optime is able to eliminate at least 99.9% of the following pathogens:

- Human Immunodeficiency Virus (HIV)
- Hepatitis viruses A, B, and C
- Mycobacterium tuberculosis
- Corynebacterium diphtheriae
- Herpes simplex virus-1 (HSV-1)

All other microbes of secondary interest in clinical dentistry, listed in the Table 1 are inactivated by at least 99%. The Opticlean™ concept of the DIGORA® Optime imaging plate system provides a comprehensive set of solutions to address the concerns of cross contamination in the use of imaging plates and provides a uniquely high level of hygiene for practical workflow in clinical dentistry.

REFERENCES
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- Misdiagnosis – a failure to diagnose the cause of your dental problems, or carrying out dental work based on an incorrect diagnosis of your condition
- Medication administration errors which lead to side-effects – including incorrect dosage of anesthetic
- Careless treatment – resulting in an injury or requirement for corrective treatment or surgery
- Swallowed instruments
- Other consequences following any dental care

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GEL 8ml, SPRAY 15ml & MOUTHWASH 60ml
Continues from page 23.

### Table 1. Summary of the germicidal UV-C doses.

<table>
<thead>
<tr>
<th>Microbe</th>
<th>Pathology, comments</th>
<th>UV dose (mJ/cm²) needed for MIC (log₁₀)</th>
<th>Notes</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1 (90%)</td>
<td>2 (99%)</td>
<td>3 (99.9%)</td>
</tr>
<tr>
<td>Bacteria</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bacillus anthracis</td>
<td>Anthrax</td>
<td>4.52</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bacillus subtilis</td>
<td>A commonly used reference/model organism in, e.g. germicide studies. Not considered as a human pathogen.</td>
<td>56</td>
<td>111</td>
<td>167</td>
</tr>
<tr>
<td>Campylobacter jejuni</td>
<td>Campylobacteriosis: inflammatory diarrhea, peridontitis or dysentery syndrome associated with fever, and severe cramps and pain. C. jejuni infection may cause a latent autoimmune neuropathy. Infections normally due to contaminated food or drink.</td>
<td>3</td>
<td>7</td>
<td>10</td>
</tr>
<tr>
<td>Clostridium perfringens</td>
<td>Diarrhea. Clostridial myonecrosis, a.k.a. gas gangrene, is a very serious medical emergency.</td>
<td>45</td>
<td>95</td>
<td>145</td>
</tr>
<tr>
<td>Clostridium tetani</td>
<td>Tetanus.</td>
<td>4.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Corynebacterium diphtheriae</td>
<td>Diphtheria.</td>
<td>3.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Enterococcus faecalis</td>
<td>Can cause endocarditis, bladder, prostate, and epididymal infections; rarely nervous system infections.</td>
<td>9</td>
<td>16</td>
<td>23</td>
</tr>
<tr>
<td>Escherichia coli</td>
<td>Virulent strains can cause gastrointestinal or urinary tract infections.</td>
<td>5</td>
<td>9</td>
<td>14</td>
</tr>
<tr>
<td>Legionella pneumophila</td>
<td>Legionnaire's disease and milder Pontiac fever, eg. pneumonia, may be dangerous especially to elderly. Infections most commonly via aerosols.</td>
<td>8</td>
<td>15</td>
<td>23</td>
</tr>
<tr>
<td>Mycobacterium tuberculosis</td>
<td>Tuberculosis.</td>
<td>10</td>
<td>20³¹⁴</td>
<td>29³¹⁴</td>
</tr>
<tr>
<td></td>
<td></td>
<td>7</td>
<td>14³¹⁴</td>
<td>21³¹⁴</td>
</tr>
<tr>
<td>Pseudomonas aeruginosa</td>
<td>Normally not a pathogen in healthy humans. An opportunistic pathogen in immunocompromised or in patients with respiratory illnesses.</td>
<td>6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Salmonella enteridis</td>
<td>Fever, cramps, diarrhea. Infections through contaminated food. The cause of &quot;egg-associated salmonellosis&quot;.</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Salmonella typhi</td>
<td>Enteric (typhoid) fever; a sustained systemic fever with headache and nausea. Other symptoms include constipation or diarrhea, dilatation of the spleen, possibly meningitis, and/or general malaise. Infections through contaminated food.</td>
<td>6</td>
<td>12</td>
<td>17</td>
</tr>
<tr>
<td>Salmonella typhimurium</td>
<td>Enteric (typhoid) fever ('mouse typhoid fever'). Infections through contaminated food.</td>
<td>8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shigella dysenteriae</td>
<td>The causative agent of the most severe shigellosis: severe dysentery - fever, diarrhea, vomiting, and cramps, along with ulceration, rectal bleeding, and drastic dehydration. Infections through contaminated food or drink.</td>
<td>3</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>Shigella sonnei</td>
<td>Shigellosis - see S. dysenteriae.</td>
<td>6</td>
<td>13</td>
<td>19</td>
</tr>
<tr>
<td>Staphylococcus aureus</td>
<td>A common inhabitant on skin and in the nose. may cause illnesses ranging from mild skin infections (e.g., pimples) to life-threatening pneumonia, meningitis, osteomyelitis, endocarditis, toxic-shock syndrome, &amp;c. Amongst the commonest cause of nosocomial (&quot;hospital&quot;) infections.</td>
<td>18</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Streptococcus faecalis</td>
<td>Enterococcus faecalis (see above).</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Continues on page 36.
Consensus statement on antimicrobial treatment of odontogenic bacterial infections

Continues from page 12.

It presents a good level of activity against anaerobic bacteria, although more and more resistant strains are emerging (33,54). More than 25% of the viridans group streptococci present a high degree of resistance (55) that cannot be overcome with high doses of this antibiotic, nor is it active against some gram-negative bacilli, such as A. actinomycetemcomitans, Eikenella corrodens and Capnocytophaga spp (56-58).

Macrolides Macrolides are bacteriostatic antibiotics with a spectrum of activity that covers gram-positive bacteria, some gram-negative bacilli, bacteria growing intercellularly and several anaerobic bacteria, including Porphyromonas and Prevotella. Bacteroides spp and Fusobacterium spp tend to be resistant to these antibiotics (59).

Like other streptococci species (S. pneumoniae, Streptococcus pyogenes) (60), the prevalence of resistance to oral streptococci has increased significantly, with rates of more than 50% in many areas of our country (55,61). Amongst representatives of this drug family, clarithromycin show the greatest in vitro activity against anaerobic gram-positive bacilli and azithromycin, against anaerobic gram-negative bacilli.

CONCLUSIONS
1. There are a host of microorganisms in the oral cavity whose taxonomy is difficult to ascertain and it is not always easy to determine how they relate to clinical presentations.
2. Microbial- and host-related factors play a role in oral and facial infections, which means that the response obtained in vivo may differ from what occurs in vitro.
3. Many oral bacteria produce beta-lactamases, which can sometimes complicate antibiotic therapy.
4. There are some individuals who are especially susceptible and in whom microorganisms produce more severe clinical symptoms and are more resistant to certain treatments.
5. Certain factors alter patients susceptibility to different microorganisms (age, blood dyscrasias, drug treatment, hospitalisation, avitaminosis and others).
6. Antibiotic efficacy is multifactorial and success depends on different parameters being met, such as dosing schedule, time, etc.
7. Amoxicillin/clavulanate, metronidazole and clindamycin are active against most of the microorganisms that are responsible for odontogenic infections. Other alternatives, such as clarithromycin and azithromycin, complete the therapeutic arsenal.

BIBLIOGRAFÍA/REFERENCES


55. Rodríguez-Avil J, Rodríguez-Avil C, Cularberas C. Distribution of mef(A) and erm(B) genes in macrolide-resistant blood isolates of viridans group streptococci. J Antimicrob Chemother 2001;47:727-8.


SIMPLER IMPLANTS

THE CHERUBINO EVENT AT CAVALIERI HOTEL PRESENTED BY DR HAROLD BERGMAN

Summarised by Dr David Muscat

The main contraindications for implants are uncontrolled diabetes and IV bisphosphonates. The latter upset the process of bone remodelling.

LOADS
With a denture a patient has a force of 25psi. With normal teeth a patient has a force of 250-950 psi. Implants handle compressive forces well but do not like lateral or torsional loads. One must try to keep loads as vertical and as central as possible.

OPPOSING DENTITION
Loading an implant against a denture is good but could be a problem against natural teeth.

AREA OF THE MOUTH
In posterior area there is twice as much load as it is closer to the TMJ.

NUMBER OF TEETH MISSING

SUPPORT
Nature of implant/bone interface. Surface area of implants. Circumference. Always go for a wider diameter and a longer length. The greater the surface area the better.

IMPLANT SURFACE
Not machined but grit blasted and acid etched and coated with hydroxyapatite.

NUMBER OF IMPLANTS
Minis minimum of 4 in the lower and 6-8 in upper with immediate loading. The more the better.

DEGREE OF HEALING
Immediate, first 3 months woven bone, remodelling, 24 months.

IMPORTANT
30ncms is the minimum force required with immediate loading. Try for 40-60 Ncems for tightness.

If you overload during the remodelling process you will lose bone. Medical health software package available by Dr Bergman where series of questions asked so as to assess patient suitability.

QUALITY OF BONE
Alveolar, basal, grafted. (grafted not good durability)

BONE
Cortical bone is very dense while cancellous bone is a latticework.

BONE DENSITY
OAK-a resorbed symphysis SPRUCE-mandible BALSA- maxilla (soft) FOAM-tuberosity (very little support)

To place a screw into oak you must always drill a hole first as the screw may break.

OTHER MEANS OF SUPPORT
Involve another tooth
Use other areas and cut down failures

Soft tissue load Teeth
Always splint in the upper jaw. No need to splint in the lower jaw.

TOOTH/BAR MATERIAL
With porcelain there is not much shock absorbing. This produces more loading. Always try to decrease to load so as to increase support.

PROCEDURE
1. PLAN ON OPG
2. How to place. Use technique with a 'slit bat guide'
3. Make pilot holes using 1.5mm twist drill
4. Insert implant into driver and thread implant into osteotomy site
5. Use ratchet driver for final tuning
6. Bottom of hex flush with mucosa
7. Reline denture over implants. Use a soft liner for 4 weeks
8. Impression o rings
9. Process o rings into the denture
10. Remove acrylic

The Physiodispenser is used. This delivers high torque, low RPMS and utilises a reduction handpiece.

FLAPLESS SURGERY
Extraction site/cookie cutter/slits
Dr Bergman who is enthusiastic about them. You are working in an area of the body that is very forgiving. If you go through lower border of mandible just use a shorter screw. It will heal. Use a 15 mm long drill.

The cookie cutter-tissue punch (4mm in diameter), flat ridge, atraumatic, wide ridge, circular cut. Slit-drill through soft tissue. Mini implants are 3mm or less.

They are one piece and can be immediately loaded. Simpler implants have square thread and this affords an increase in 60% more surface area And so more support for loading. You need 30ncms or more. The Implants are also HA coated.

ADVANTAGES TO PATIENT
Cost effective, non-invasive, immediate gratification, successful, benefit/risk ratio good.

ADVANTAGES TO DENTIST
cost effective, easy, minimal training, flapless.

In the USA 10% of patients are edentulous. 100% have lower dentures that do not function well.

There is an enormous market out there are patients are enthusiastic about them. You will improve quality of life.
QUESTIONS & ANSWER # 1

I hear so much about “Immediate Load,” “Four on the Floor”, “Mini Implants” in which the implants are loaded immediately. Can I load implants immediately?

ANSWER # 1

You can load any implant immediately. HOWEVER, the success rates are lower with immediate loading versus delayed loading especially in the upper jaw.

Whenever you are loading any implant at any time, you must always think in terms of LOAD and SUPPORT. All parts of your body respond best to using it within physiological limits. If you overuse and abuse it, you lose it and if you underuse it (Couch Potato), you lose it by wasting away. The trick is to use it within physiological limits. If you broke your arm, the doctor puts your arm in a cast to Immobilize and Support the broken parts. He instructs you not to overuse (Load) the arm until the bone has healed sufficiently (“knits”). Once the cast is removed, you will slowly increase its load through graduated exercises (within physiological limits).

An implant is no different, the bone must knit to the implant (osseointegration = knit). When you first place the implant, it must be tight in the bone (immobilized). Initially, you MUST keep loads to a minimum by using soft liners in the denture, try for a denture in the opposing arch versus natural teeth, soft diets, remove denture at night if patient grinds their teeth, etc.

You MUST also increase Support as much as possible by ensuring the bone is dense, increase the number and size of the implants, use Hydroxyapatite (HA) coatings on the implants, use the soft tissue or other teeth, if present for additional support, etc. One might consider construction of a metal or acrylic bar to splint the implants together to provide additional support, especially in the maxilla.

ANSWER # 2

There are two reasons. The topmost part of most implants have parallel walls whereas some systems have a flared top. Should you get acrylic under the flare during a chairside pickup, it will prevent removal of the denture from the mouth.

One solution is to block out under an attachment, the other is not to use an implant with a flared top. Secondly, if the implants and attached abutments are not parallel with each, there will be divergence of the abutments resulting in undercuts on some abutments.

QUESTION # 2

Why is it necessary to block out under an attachment such as an O ring or Locator when I do a chairside pickup with acrylic in a soft tissue overdenture in the mouth?

Continues on page 35.
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Should you get acrylic into the undercut it will prevent removal of the denture from the mouth.

One solution is to not use an abutment whose walls extend into the mouth such as a Locator abutment. Another solution is to ensure that whoever places the implants, places them parallel with each other.

There are a number of ways the operator can ensure parallelism (Figure 1, 2) including using a system neutral Paralleling Guide (Figure 3) when making a surgical stent or intra-orally at time of surgery.

**QUESTION #3**
I have a patient who has a soft tissue supported overdenture that is rocking. What is the problem and how do I correct it?

**ANSWER #3**
Soft tissue supported overdenture (STSOD) means exactly what it says. All the support for vertical loading should be taken by the soft tissue, NOT the implants.

The implants are there only to prevent lateral movement and vertical displacement. If a STSOD is rocking, it means the implant are being vertically loaded. Most STSODs are being held in place by as few as 2 implants.

That few implants are not designed to take loads of this magnitude and are at risk of failure. Currently, some attachments called “Locators” are very popular with STSODs amongst denturists and their use has replaced rubber O rings in many cases.

In my experience, most STSODs supported by Locators “rock” and the compressibility of the material is not as resilient as rubber O rings.

From a design, cost effectiveness, ease of use and physiological basis, I prefer using rubber O rings in a STSOD. If the STSOD is rocking, the attachment is either not seated deeply enough in the denture, the denture needs relining or there is too much acrylic immediately above the top of the O ring abutment.

The solution is to immediately remove the acrylic above the O ring abutment (Figure 4), or remove & replace the attachment or either redo the pickup or reline the STSOD.

There is a new design available now called the “Toadstool” abutment and/or Mini implant. The Toadstool is designed to virtually eliminate loading on the implants with STSODs.
<table>
<thead>
<tr>
<th>Virus</th>
<th>Acute Hepatitis (acute viral hepatitis)</th>
<th>Complete inactivation at 110 μm^2</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatitis B virus</td>
<td>Acute or chronic hepatitis (hepatitis B), may lead to liver cirrhosis; hepatocellular carcinoma. Infections through blood or body fluids.</td>
<td>≤16.4μl</td>
<td>16, 17</td>
</tr>
<tr>
<td>Hepatitis C virus</td>
<td>Acute hepatitis, may lead to liver cirrhosis. Infections normally through blood or mucosal contact.</td>
<td>≤33.6μl</td>
<td>16, 17</td>
</tr>
<tr>
<td>Herpes simplex virus 1</td>
<td>Oral (orofacial) and genital herpes are the commonest illnesses caused by the herpesviruses, characterized with painful inflammation. HSV1 causes also infections elsewhere in the skin, in the eyes, and in the neuronal system.</td>
<td>&lt;16μl</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&lt;48μl</td>
<td>21</td>
</tr>
<tr>
<td>Human immunodeficiency virus</td>
<td>Acquired immunodeficiency syndrome</td>
<td>Complete inactivation at 66 μm^2</td>
<td>8, 10</td>
</tr>
<tr>
<td>Influenza A virus</td>
<td>Influenza.</td>
<td>Complete inactivation at 66 μm^2</td>
<td>14, 27</td>
</tr>
<tr>
<td>Poliovirus type 1</td>
<td>Poliomyelitis; different types of neuronal symptoms, most often infections in motor neurons associated with muscle weakness paralysis.</td>
<td>28, 56μl, 84μl</td>
<td>1, 4, 12, 17</td>
</tr>
<tr>
<td>Rotavirus SA-11</td>
<td>Mild to severe gastroenteritis. The most common cause (along with adenoviruses) of severe diarrhea in infants and young children.</td>
<td>10, 20, 29</td>
<td>11, 23</td>
</tr>
<tr>
<td>Giardia lambia (G. duodenalis)</td>
<td>Giardiasis can be associated with a wide range of clinical symptoms. Acute gastrointestinal giardiasis causes severe diarrhea, abdominal cramps and vomiting, and fever.</td>
<td>2, 5, 11</td>
<td>11, 23</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3</td>
<td>18</td>
</tr>
</tbody>
</table>

**NOTES:**

1. MIC (μg/mL): microbial inactivation credit 1-23, 1 log means 90% inactivation, 2 log 99%, 3 log 99.9%, and 4 log 99.99% inactivation at the given dose (fluence).
2. No detectable infectivity after 8.7 μm^2 for B. anthracis, 15 μm^2 for C. diphtheriae, 15 μm^2 for E. coli, 0 μm^2 for P. aeruginosa, 24, 76 μm^2 for S. enteritidis, and 225 μm^2 for adenovirus 4113.
3. For S. mutans, the absolute UV fluence value is not available; the inactivating UV dose (1 and 3 log MIC) has been shown to be smaller than for P. aeruginosa, B. thermosphilus, and C. albicans. For P. aeruginosa, the dose (11 μm^2) has been reported.
4. Estimated from the Fig. 2B.
5. Complete inactivation of HIV was reported after 0 min exposure to 0.223 μm/μm^2, shorter treatments were not reported. Inactivating dosage 30 μm^2 has been reported on the website http://lightbulbsn100.com/germicidal-light.html (no reference to the original data).
6. Formerly inappropriately called "protozoa".
7. Predicted dose based on data from ref. 16.
8. Dose calculated according to the "Chick's law" of inactivation (N/NO) = e^−kt, esp. ref. 29, based on the assumption of "quasi" first order inactivation kinetics (ie, linear correspondence of log inactivation to the UV dose). The first order kinetics is a general assumption in the literature; several studies support this assumption at relevant virus/bacterial concentrations 5,10,28, 12.

**References:**

- [1] MIC (μg/mL): microbial inactivation credit 1-23, 1 log means 90% inactivation, 2 log 99%, 3 log 99.9%, and 4 log 99.99% inactivation at the given dose (fluence).
- [2] No detectable infectivity after 8.7 μm^2 for B. anthracis, 15 μm^2 for C. diphtheriae, 15 μm^2 for E. coli, 0 μm^2 for P. aeruginosa, 24, 76 μm^2 for S. enteritidis, and 225 μm^2 for adenovirus 4113.
- [3] For S. mutans, the absolute UV fluence value is not available; the inactivating UV dose (1 and 3 log MIC) has been shown to be smaller than for P. aeruginosa, B. thermosphilus, and C. albicans. For P. aeruginosa, the dose (11 μm^2) has been reported.
- [4] Estimated from the Fig. 2B.
- [5] Complete inactivation of HIV was reported after 0 min exposure to 0.223 μm/μm^2, shorter treatments were not reported. Inactivating dosage 30 μm^2 has been reported on the website http://lightbulbsn100.com/germicidal-light.html (no reference to the original data).
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- [8] Values calculated according to the "Chick's law" of inactivation (N/NO) = e^−kt, esp. ref. 29, based on the assumption of "quasi" first order inactivation kinetics (ie, linear correspondence of log inactivation to the UV dose). The first order kinetics is a general assumption in the literature; several studies support this assumption at relevant virus/bacterial concentrations 5,10,28, 12.
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Pen pictures of 19th century dentists in Malta

Arthur Crofton Sleigh

By George. E. Camilleri

Arthur Crofton Sleigh is the only British person who obtained the Warrant to practise Dentistry in Malta by the Malta Government. He was born in Mile End, London in about 1864. He joined the British Army, probably in the Army Medical Corps and was serving in Egypt in 1885. During this period the Marriage Question was a troublesome item in Maltese politics. All marriages involving Catholics were under the jurisdiction of the local church whilst Anglicans and non-Catholics required permission from the British Authorities and certificates supporting their civil status had to be presented. One such certificate issued by Sleigh gives us some details of him.

"I, Arthur Crofton Sleigh, a native of London, late of the Army Hospital Corps, now a 1st Class Sergeant of Police in the island of Malta, make oath and say as follows. In 1885 I was serving in the Army Hospital Corps in Egypt and became acquainted with Richard Chester Kirby Laffan, Surgeon, Medical Staff Corps and Miss Mary Clementine Jarrard, an Army Nursing Sister, both of whom were Protestants, Sgd ACS 6th December 1893". A Malta Passport was issued in 1894 to Sleigh to travel to Egypt giving his occupation as 1st Class Sergeant, Malta Police. Unfortunately, no records on Sleigh were found in the Malta Police Archives. In 1895 he is listed in the Voters' List which is an indication of some status as the list was very restrictive.

He married a Maltese girl, Regina Germani, which may explain why he settled in Malta, but I do not have details of his marriage. In 1897 he petitioned for the warrant of Phlebotomist which petition seems to have led to some discussion between the Government and the University. The Government appears to have been pushing the University to organise courses or examinations for Dentist and Phlebotomists. Salvatore L. Pisani, the Chief Government Medical Officer informed the Chief Secretary that he was ready to meet the Director of Education at his convenience. This meeting led to a note from Napoleon Tagliaferro, the Rector of the University to the Chief Secretary stating "In an interview between the CGMO and the undersigned it was agreed to recommend that the certificate of studies for admission to the Examination before the Medical Board for the Warrant of Dentist or Phlebotomist should be granted either by the University or by any other School authorized by Government. Napoleon Tagliaferro, 17-12-97". Sleigh’s petition for a warrant to practise as a phlebotomist was granted presumably on the strength of his previous Army job. Phlebotomy did not seem to have been enough to support his young growing family and Sleigh must have set his eyes on dentistry. In 1899 he petitioned for the warrant of Dentistry stating in his application that he had obtained, after a strict examination, a Diploma for Dental Surgery from the University of Naples.

He failed the examination of the Medical Board and was asked to reappear in six months time when he was successful. He now appears in the Directory of the Military Station Hospital in Valletta but in 1901 he advertised in the Malta Chronicle that "A Crofton Sleigh, Surgeon Dentist. Removed from Station Hospital to no 8 Strada Zecca near the Admiralty House, Strada Mezzodi".

Tragically his success in running a dental practice was short lived as he died of Malta Fever on the 17th August 1904. The obituary notice fully recognised his heroic efforts to establish himself. "Mr. Crofton Sleigh, Surgeon Dentist died at his residence in Strada Zecca yesterday morning. He was ill for only a short illness. Mr Crofton Sleigh, besides being recognised for an able dentist, was a man of real perseverance and a happily combative nature.

He made a career for himself in spite of initial difficulties such as would have deterred men of less resolute and hopeful mind. Death has however carried him in only the middle as we should judge of the course that stretched promisingly before him. And he leaves several children, all young, to mourn his irreparable loss. We offer our sincerest condolences to his affected widow and bereaved children". He is buried at the Addolorata Cemetery in an unmarked grave in the Germani family section.

He had five children, Arthur, Alexander, Charles, Richard, and Alice. Alexander graduated LDS Edinburgh in 1914, practised in England, and is registered as a member of the Malta Dental Association in 1955. Arthur became a priest and taught at St Edmund’s College, Old Hall, Ware. All the children seem to have emigrated from Malta and I could not find records of any Sleigh members in Malta.
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