IDentistry

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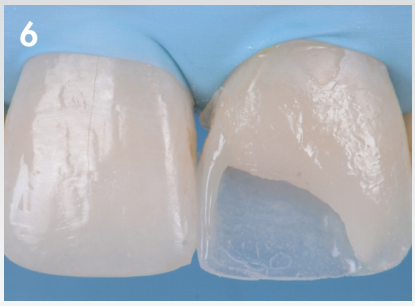

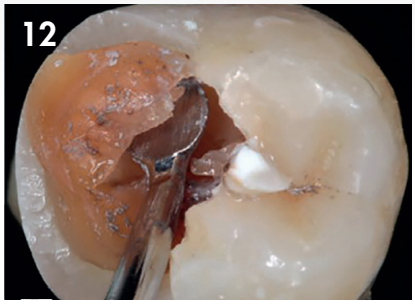

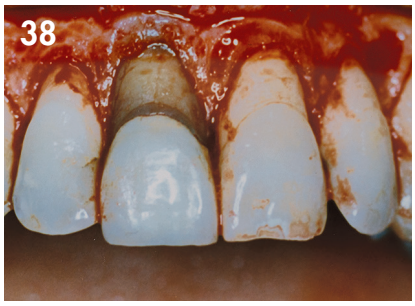

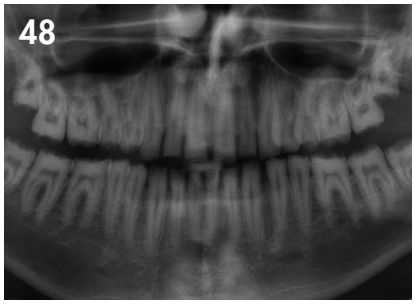

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COVID 19 SAFE

Scientific studies show that SDF can arrest caries and is more effective than NaF *Lynch et al, Nature 2019*

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Invitation to authors

Professor Andre W van Zyl
MChD (Oral Medicine & Periodontics)

Through all the years that I trained young people there was one constant thread. All expressed a desire to become involved in lecturing or to achieve just a little more than the mere extra qualification.

However, not all are suited for the lecturing circuit, be it from an inherent shyness, not having a voice for lecturing or just never getting an opportunity to be invited.

Covid has changed all of that. Congresses have been cancelled, even local gatherings of study clubs are not acceptable. This has created a level playing field and one that should be grabbed with both hands by those with forward thinking.

Now is the time to prepare for when Covid will be under control and meetings/congresses will be presented once more. The time is now to use the printed media to build your curriculum vitae, to put your name in the hat for when new speakers/leaders are needed or just to build the profile of your practice. Nothing instils confidence amongst patients like a clinician who publishes in professional journals. I want to invite or challenge each and every one of the young people who aspire to become a leader, speaker or just to feel good about yourself, to submit articles to us for publication. Clinical cases, novel treatment approaches or a case series of certain treatment options are all acceptable. What does it take to write an acceptable article? It needs to be backed up by some evidence that it has worked or can work or that it is justified to try it due to previous publications. Evidence used to be the exclusive domain of universities with access to expensive journals which private practitioners just could not afford. Not anymore. Open access journals are now accessible to all and you can google almost any topic and find free access to articles. Any good lecture can be turned into a good clinical article.

In addition you need to master intra-oral photography if you wish to publish clinical material. This is again not that difficult. You need a camera with a macro lens and some ring-flash or similar. These you will be able to pick up for under R30 000 at reputable dealers. It is tax deductible.

Do yourself a favour and scan previous editions (available online) to get an idea of what is acceptable. Some of the leading clinicians in South Africa regularly publish in this very journal. This invitation is for general practitioners and specialists alike. An article that reaches every reader is more valuable than one reaching a small subsection of a particular specialty. Another constant that I heard from experienced clinicians at congresses through the years was the old "In my hands this works every time". So here is another challenge to our more experienced colleagues, even if you are nearing or already retired. Share that knowledge with the younger generation. To transfer the skills and knowledge built up through a lifetime in private practice is a very rewarding exercise and a worthy one. Think of all the top private clinicians that performed part-time lecturing duties when we were students. This is just another way of teaching the young of today, and you can do it from any location in the world.

We are here to help you with constructive criticism or advice, so give it a try. You may just change the way you see your profession in the years to come.

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Class IV restoration with direct composite resin: A case study utilising the layering-stratification technique with a novel composite system

Anthony Mak[1] and Andrew Chio[2]

The concept of layering or stratification of direct composite restorations utilises the combination of optical properties from the different resin layers with the aim of emulating the natural colour, characteristics and translucency of the natural dentition. Progressive improvements in composite resin technologies have led to the simplification of this treatment procedure that is commonly perceived as complex. However, difficulties exist in mimicking the remaining tooth structure when restoring teeth in the anterior segment of the dentition because of the variety of shades, chroma, and translucency levels of many current composite resin systems.

The G-ænial A'CHORD represents the evolution of the highly successful G-ænial system that has been utilised in dental practices throughout the world for the past 10 years. Compared to its predecessor, the G-ænial A'CHORD system provides an upgrade from the original G-ænial system in the following aspects:

- Beautiful and harmonious under any light with a natural fluorescence.
- Optimal handling properties allowing for the material to be easily sculpted with conventional composite manipulation instruments or brushed with restorative brushes.
- The Full-Coverage Silane Coating (FSC) technology that covers the nano-fillers with silane coupling agent leads to high polish and gloss with only a few steps.
- The incorporation of additional opaque and enamel shades allows an infinite range of opacity and value possibilities.
- Simplification with 5 CORE shades which covers all 16 Vita shades.

VITA® Classic	A1	A2	A3	A3.5	A4	B1	B2	B3	B4	C1	C2	C3	C4	D2	D3	D4
Opaque	AO1	AO2	AO3	AO3	AO3	AO1	AO2	AO3	AO3	AO2	AO3	AO3	AO3	AO1	AO2	AO3
CORE	A1	A2	A3	A3.5	A4	A1	A2	A3	A3.5	A2	A3	A3	A3.5	A1	A2	A3
Enamel	JE	JE	AE	AE	AE	JE	JE	AE	AE	JE	AE	AE	AE	JE	JE	AE

Multi-layering technique correspondence

[1] Dr Anthony Mak, BDS (USyd), Grad Dip Clin Dent (Oral Implants) (USyd), Private Practice, Sydney, Australia, focusing on comprehensive and implant dentistry.

[2] Dr. Andrew Chio, BDSc. (Melb.) Private Practice, Melbourne, Australia

Case Report

The following case study demonstrates the use of the G-ænial A'CHORD (GC Europe) direct composite system in the restoration of a complex class IV in a 22-year old female patient. The patient presented to the practice relaying her dissatisfaction of an existing restoration on her upper left central incisor (FDI tooth 21). She requested its replacement with a new restoration that was conservative and "invisible" when she smiled or engaged in normal conversation. She also relayed that the existing class IV restoration had been done 4 times by her previous dentist without an outcome or result that was satisfactory to her.

Clinical examination revealed a high smile-line with a symmetrical and aesthetic gingival architecture. The existing composite restoration on the tooth 21, while clinically acceptable, did not integrate with the shade of the tooth and to the other teeth in her dentition. The discolouration and minor ledging on the disto-labial aspect of the existing restoration also indicated the likelihood of marginal leakage in the region.

The pre-operative colour assessment showed that the upper left central incisor (21) was slightly more chromatic than the adjacent upper right central incisor (11). The upper left central incisor (21) also exhibited a very slight labial displacement in its alignment compared to the adjacent right central incisor (11).

The patient's health history was unremarkable.

Radiographic and periodontal examination showed that the tooth 21 demonstrated no pathology or issues requiring intervention prior to the commencement of the restoration. The 21 exhibited a normal response when the vitality was thermally tested.

The treatment options were discussed with the patient and the advantages and disadvantages of each of the options were carefully identified. The options presented were:

1) A single reductive ceramic veneer on tooth 21.
2) A full surface composite veneer on tooth 21. The patient was advised that due to the slight labial displacement of tooth 21, a very small labial reduction would be required to allow the space to mask the chromatic dentine.
3) A conservative complex class IV on the tooth 21 to be completed additively to minimise any preparation and reduction of the natural tooth structure.

She preferred the conservative approach to her treatment involving an additive protocol (option 3). She relayed that she would be happy with a harmonious composite restoration on tooth 21 and did not feel that the slightly chromatic upper left central (21) would be an aesthetic concern for her.

From the clinician's perspective, final plan and goal of the treatment was to restore the tooth 21 with a durable and long-lasting conservative direct composite restoration with a final result that is biomimetic with optimal aesthetic and morphological integration with her existing natural dentition.

Step by step

Prior to the commencement of the restorative process, diagnostic images and the selection of the estimated shade was completed. Diagnostic impressions were also taken to allow the fabrication of silicone palatal stent or matrix that would facilitate the three-dimensional blueprint for the layering of the composite increments.

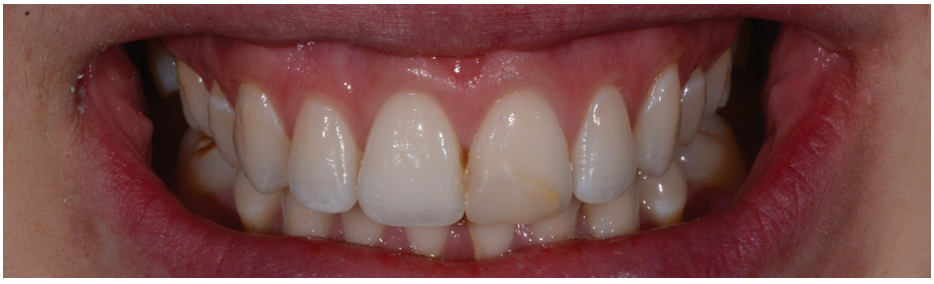

Figure 1: Pre-Operative unretracted view illustrating the unaesthetic and failing direct composite restoration on the upper left central incisor (tooth 21).

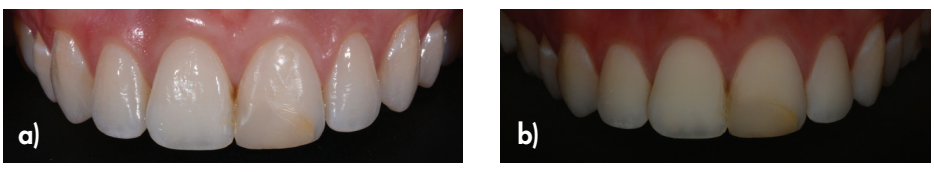

Figure 2: Pre-Operative Retracted a) with regular flash b) with Polarized filter.

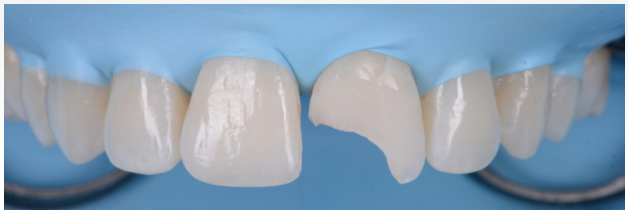

Figure 3 : The working field was isolated with the use of the rubber dam. The existing restoration and caries was removed and a 2 mm bevel prepared on the labial margin of the preparation to facilitate the aesthetic and functional integration of the restoration to the remaining natural tooth structure.

Figure 4: The bevel was prepared and finished with a tapered diamond bur (Komet 6862.314.012 and 8862.314.012). All the transition angles of the cavity were rounded with an oval or egg-shaped polishing diamond bur (Komet 8379.314.023). The burs form part of the "Dr Anthony Mak Custom C&B Selection" Kit from Komet Dental.

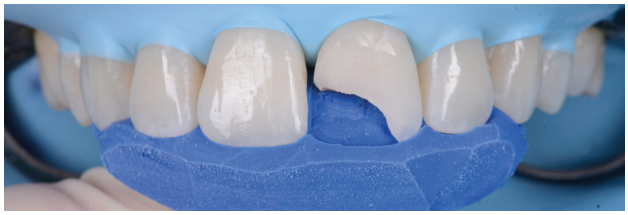

Figure 5: The palatal stent was trimmed and tried-in to verify the fit of the silicone matrix and to ensure the absence of any interferences to its seating from the rubber dam and clamps.

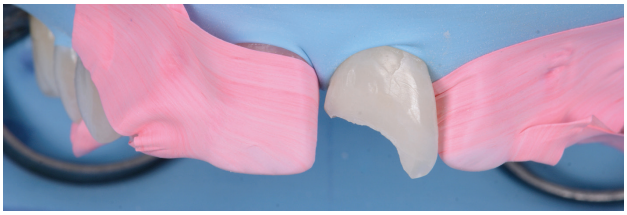

Figure 6: The cavity was lightly air abraded with a 29-micron aluminium oxide powder AquaCare (Velopex) prior to the adhesive procedure and Teflon (PTFE) tape was utilised to prevent the inadvertent bonding to the adjacent teeth.

Figure 7: The adhesive procedure commenced with the with the cavity selectively etched with 37% phosphoric acid gel Ultra-Etch (Ultradent). The etching gel was rinsed away and the adhesive protocol was completed by the application of a universal bonding agent, G-Premio BOND (GC Europe). The universal bonding agent was then air dried for 5 seconds with maximum air pressure and light-cured for 10 seconds according to the manufacturer's instructions.

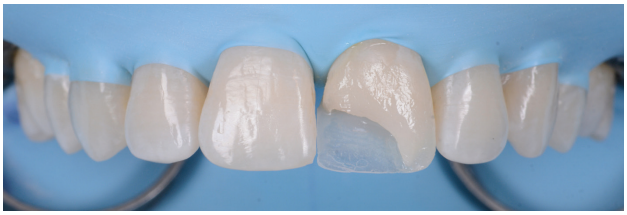

Figure 8: Following the adhesive protocol, a thin layer of semi-translucent enamel, G-ænial A'CHORD shade JE (GC Europe), was used to create the palatal shell.

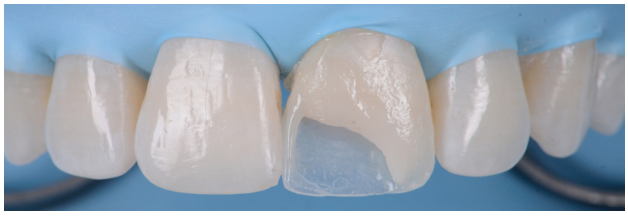

Figure 9: The interproximal wall was then completed utilising the same semi-translucent enamel shade, G-ænial A'CHORD shade JE (GC Europe). The interproximal wall was formed with the use of a plastic myeloid strip and pull through technique to help developing an anatomical contour.

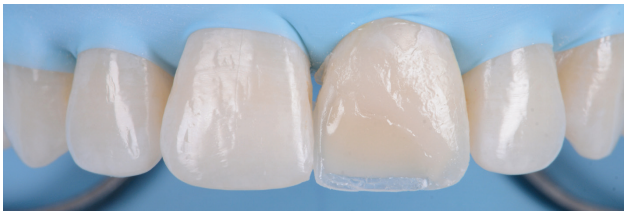

Figure 10: The dentine layer was then completed by the application of an opaque shade, G-ænial A'CHORD shade AO2 (GC Europe). This increment was shaped to emulate the extensions of natural dentine core morphology and was extended just slightly short the prepared bevel. The dentine or opaque shade provides the correct opacity to the final restoration.

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* L. P. Samaranayake, J. Reid, N. Evans; ASDC Dent. Child. 56, 442-444 (1989); The efficacy of rubber dam isolation in reducing atmospheric bacterial contamination (only abstract available)

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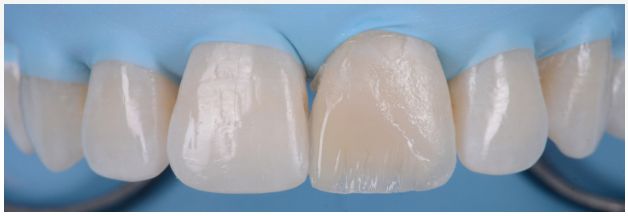


Figure 11: A chromatic body shade, G-ænial A'CHORD shade A2 (GC Europe) was then applied and extended beyond the bevel to mask the transition line. Internal anatomy (i.e. mamelons) in the incisal third was also sculped and formed in this increment of composite resin.

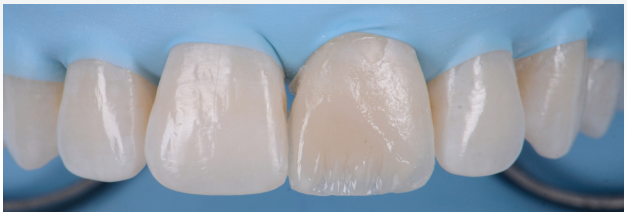


Figure 12: White tints, Essentia White Modifier (WM) (GC Europe) was utilised to accentuate the mamelons and to replicate the similar characteristics and features present in the adjacent right central incisor (tooth 11). Comparisons to the polarised diagnostic images taken prior to commencement of the restoration provided a reference for the incorporation of these internal features.

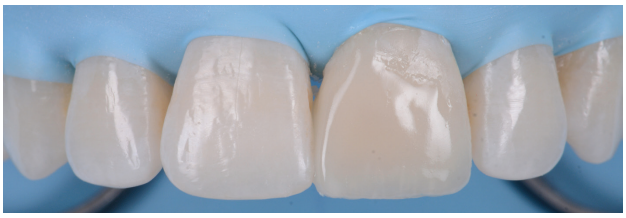
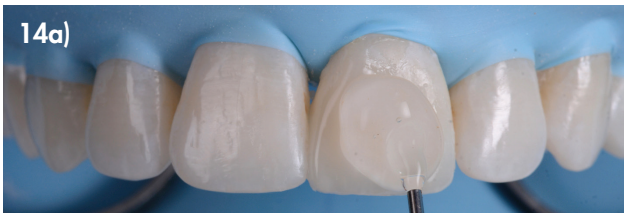


Figure 13: A final translucent shade of G-ænial A'CHORD shade JE (GC Europe) was then layered to bring the anatomy to full contour and to achieve a natural optical blending effect.



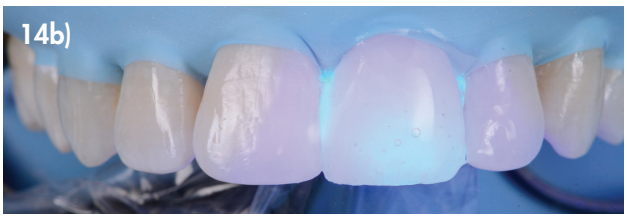


Figure 14 a, b: Glycerine gel was then applied over the buccal surface of the restoration and light-cured to maximise the polymerisation of the layered direct composite restoration due to the absence of the oxygen-inhibition layer.

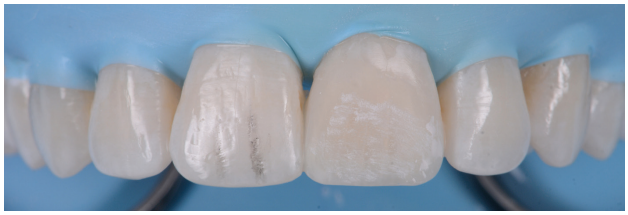


Figure 15: The restoration was then polished and finished to incorporate the primary, secondary and tertiary anatomy with the aim to produce a life-like restoration that mirrored the adjacent right central incisor (tooth 11).

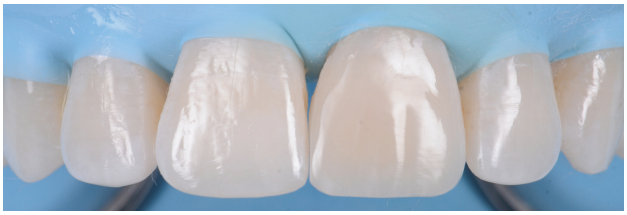


Figure 16: The polishing and finishing protocol employed the use of abrasive discs (Soflex; 3M-ESPE), polishing diamond burs (Komet), followed a graded sequence of silicone polishers and finishers (Astropol; Ivoclar-Vivadent). The restoration was then completed using a Diapolisher paste (GC Europe) on a felt-buff (Flexi-Buff; Cosmedent Inc) to recreate the gloss of natural enamel.

Conclusion

While developments in single shaded universal composite systems for the anterior dentition continue to improve and advance layering techniques for the placement of a truly aesthetic anterior direct composite restoration will always be necessary in the contemporary aesthetic dental practice. This is due to the intrinsic anatomy of the natural tooth where the emulation of the optical and morphological properties

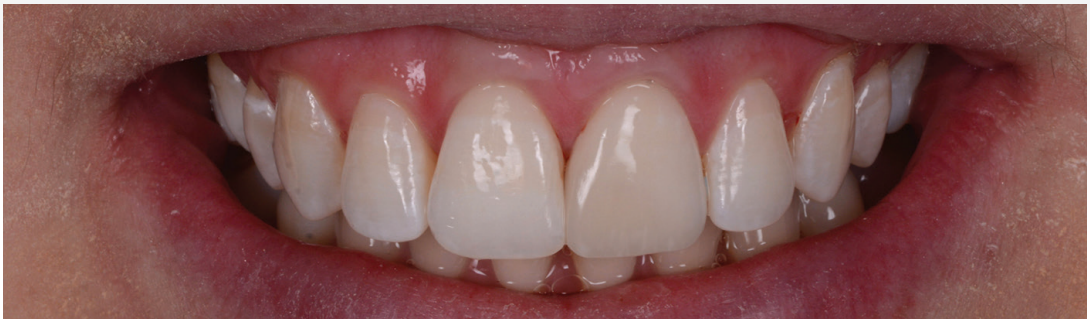


Figure 17: Immediate post-operative (Unretracted). The finished and polished G-ænial A'CHORD (GC Europe) restoration demonstrates the morphological and optical aesthetic integration of the completed restoration to the existing natural dentition.

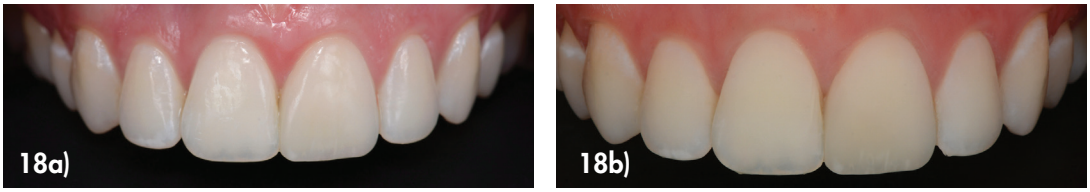


Figure 18: Immediate post-operative (Retracted) a) regular flash b) polarised filter

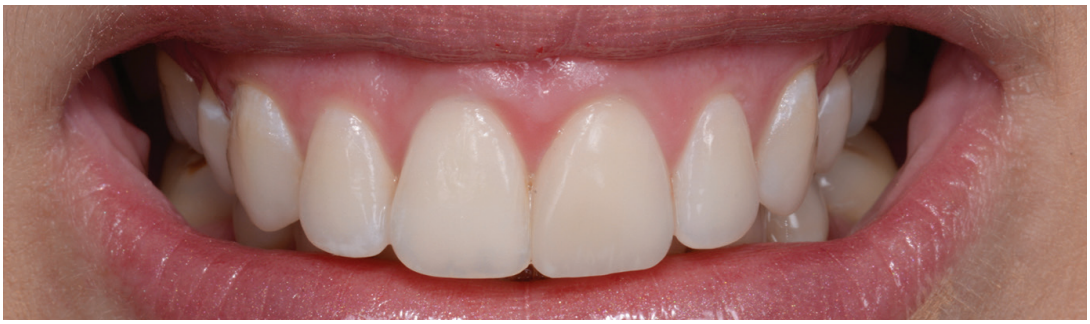


Figure 19: 2-week review demonstrating the complete optical and functional G-ænial A'CHORD restoration on the tooth 21.

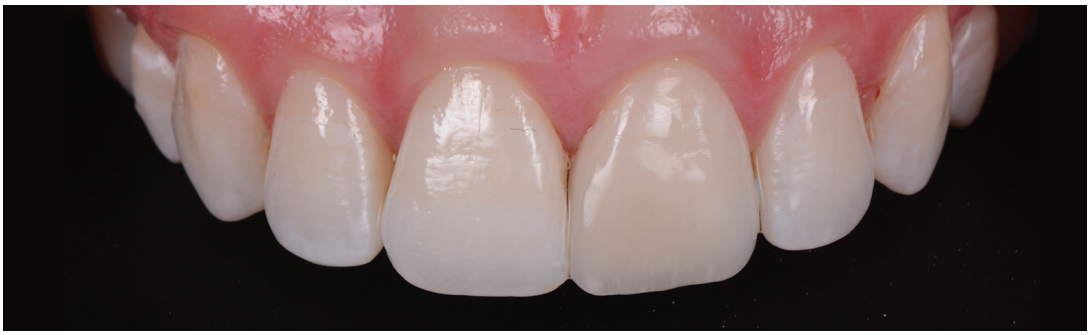


Figure 20: 2-week review demonstrating the complete optical and functional G-ænial A'CHORD on the tooth 21.

cannot be achieved by a single mass of restorative material. The G-ænial A'CHORD (GC Europe) composite system has a simplified approach to the shading/layering process while providing a final result that is truly biomimetic, aesthetic and long-lasting.

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Silver diamine fluoride: a practical guide

Louis Mackenzie[1]

The COVID-19 pandemic and subsequent limitations on the use of aerosol generating procedures has renewed interest in the use of silver diamine fluoride (SDF) as a simple, minimally invasive method for stabilising and arresting carious lesions.[1,2,3]

Silver diamine fluoride (SDF)

SDF is a colourless alkaline (pH 9-10) solution containing silver (~25%) and fluoride (~5%) stabilised in ammonia. It has been used internationally for caries management for decades but is currently only licenced as a desensitising agent in the UK, with one CE marked product: RIVA STAR (SDI. Australia) – Figure 1.

Silver has been used as an antimicrobial agent worldwide for over a century, as it has been demonstrated to be capable of destroying bacterial cell walls, inhibiting bacterial metabolism and enzyme activity and reducing biofilm formation. In SDF, this combines with the remineralising properties of fluoride to offer a range of clinical indications and reported advantages, listed in Table 1.[1,2,3]

The principal disadvantage of SDF is a potential for black/brown discolouration of carious dentine, caused by precipitation of metallic silver and silver oxide, which limits its acceptance aesthetically. Staining may be reduced with the use of potassium iodide solution.

Contraindications for SDF are listed in Table 2 and other cautions include the following:

- Unaesthetic staining of restorative margins
- Staining, irritation or burns of mucosa and skin
- Damage to worktops and clothing
- Slight metallic taste/ammonia odour (but generally well tolerated)
- May reduce bond strengths of glass ionomer and resin composite materials (but may stabilise hybrid layers)

Clinical procedure for SDF caries treatment

Silver diamine fluoride is available in a range of concentrations eg 12-40%, with 38% considered to be the most effective.

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Figure 1. RIVA STAR (SDF) delivery systems.

The following step-by-step stages have been recommended for treatment of carious lesions[1,2,3] and key clinical stages are simulated on an extracted tooth (Figure 2A)

- Cover worktops and protect the patient's clothing with a plastic bib
- Apply a suitable barrier material (eg petroleum jelly) to protect the patient's lips
- Remove visible plaque
- Excavate soft, necrotic, infected dentine eg using a hand instrument (Figures 2B,C)
- Isolate the treatment area eg rubber dam, liquid dam (provided in kit) or cotton wool rolls

Table 1: Clinical indications and advantages of SDF

Immediate relief of dentine hypersensitivity (Silver iodide blocks dentine tubules and has low solubility) eg cervical abrasion cavities
Relief of symptoms from carious cavities (especially occlusal lesions)
Promotes minimally invasive cavity preparation (eliminating the need for extensive caries excavation)
Minimises the need for aerosol generating procedures
Inexpensive and obviates the need for local anaesthesia
Quick and easy application makes it useful for patients who are: vulnerable, uncooperative, have behavioural problems and those with limited access to conventional dental treatment
Stabilisation/arrest of caries into dentine in deciduous teeth eg may avoid the need for hospital admission (GA/Sedation)
Stabilisation of deciduous teeth soon to be exfoliated
Stabilisation/arrest of caries in elderly patients eg root caries
Acclimatisation to dental treatment and stabilisation of multiple lesions
Remineralisation of incipient carious lesions (2-3 times more fluoride concentration than sodium fluoride varnish)
Endodontic irrigation and inter-appointment medicament

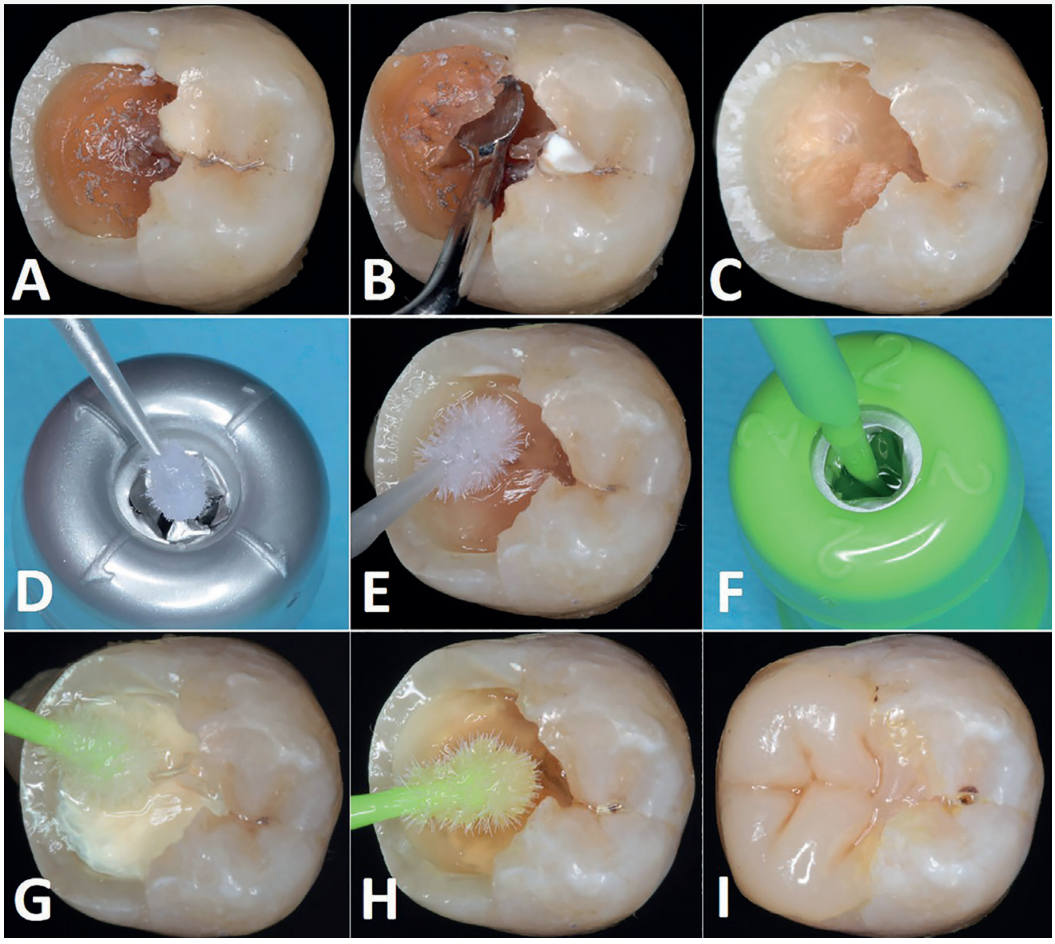

Figure 2. Practising the use of silver diamine fluoride (RIVA STAR, SDI) for caries management on an extracted natural mandibular molar.

- Pierce the SDF capsule (silver) with a microbrush and thoroughly wet the tip (no mixing is required) – Figure 2D
- A glass Dappens dish may also be used as a dispenser (the SDF capsule contains sufficient material to treat approximately five carious lesions)
- Apply to SDF to dentine caries (with care to avoid soft tissue contact) as in Figure 2E
- Blot excess with cotton wool or gauze
- Working time is approximately five minutes and once placed leave for 1-3 minutes
- Pierce the Potassium Iodide (KI) capsule (green) with a new (colour coded) microbrush (Figure 2F)
- Continuously apply KI to the SDF until the creamy white precipitate (Figure 2G) turns clear (Figure 2H)
- Repeat blotting of excess material
- Leave to dry completely
- Restore tooth as required eg resin composite or glass ionomer. It is recommended to rinse enamel with water first (Figure 2I)
- Remove isolating materials
- Safely dispose of single-use capsules and used microbrushes
- Review patient for signs of arrest in around 2-4 weeks

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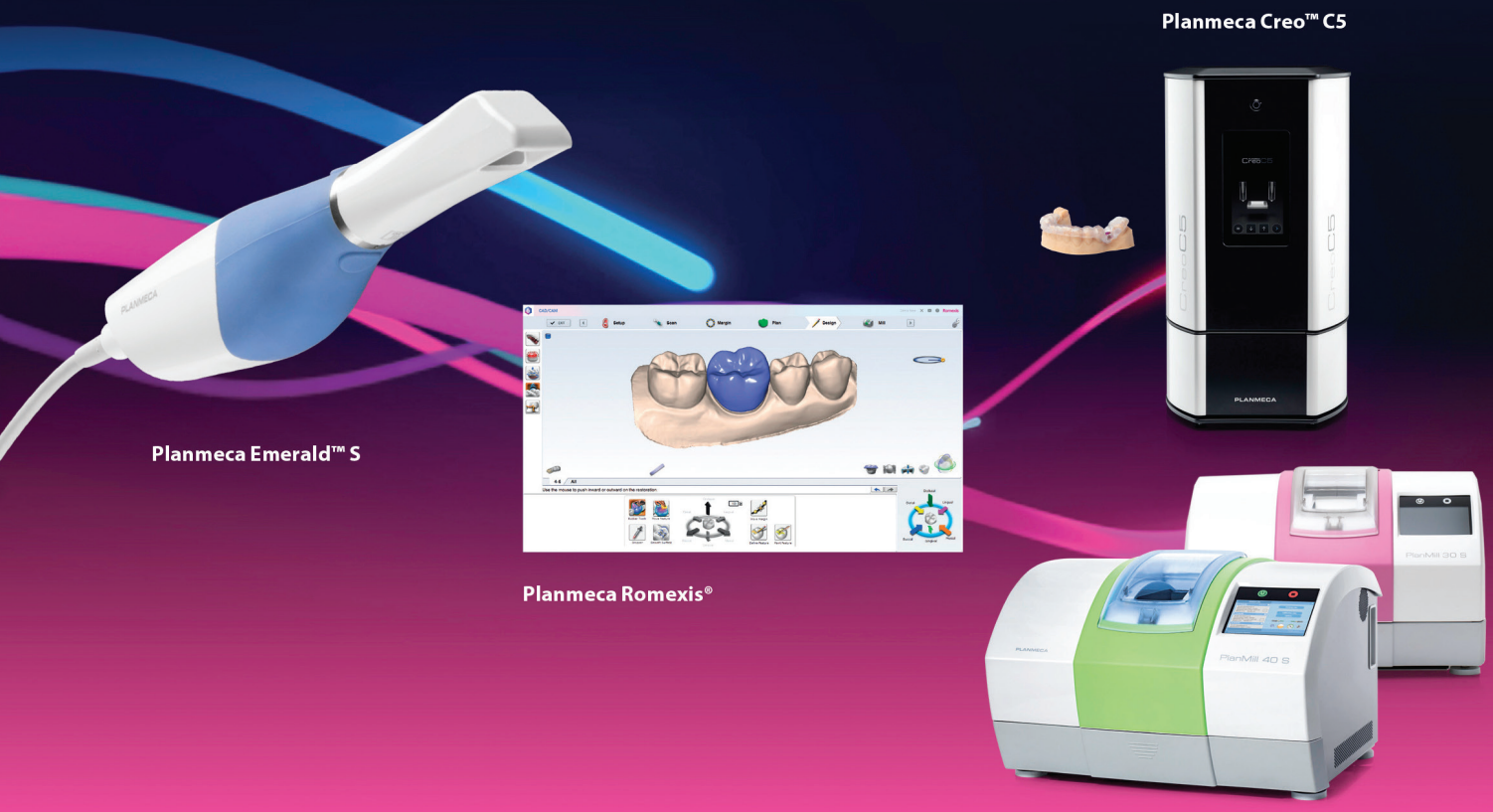

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Table 2: Contraindications for SDF.[1]

Silver/heavy metal allergy
Pregnancy and breastfeeding (risk of overloading developing thyroid with iodide)
Lesions close to the pulp/possible pulpal involvement (avoids silver ions in pulp chamber)
Signs and symptoms of periapical pathology (clinical and/or radiographic)
Ulceration, mucositis or stomatitis
Restoration of permanent anterior teeth

(large cavities may need a second SDF application)

- Six monthly re-application is recommended for SDF treated lesions that are not definitively restored
- Successful use of SDF will be significantly enhanced as part of a comprehensive prevention/caries management programme

Prescribing SDF

SDF is licenced for use in the UK as a desensitising agent (and cavity cleanser). Its use beyond the approved indications is considered to be off-licence prescribing. If used for caries management, the Medicines and Healthcare Regulatory Agency (MHRA) guidelines must be followed regarding prescribing in the patient's best interest, based on the best available evidence and when there is no suitable alternative treatment.[3]

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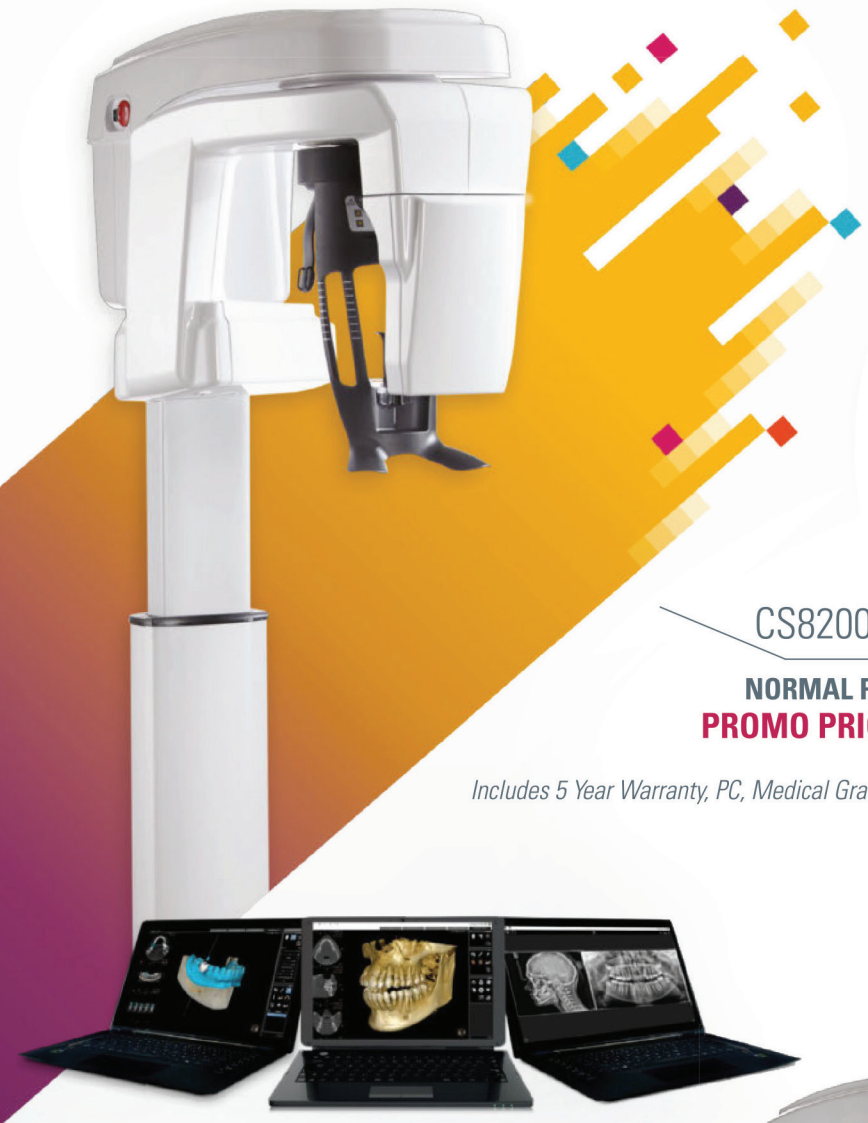

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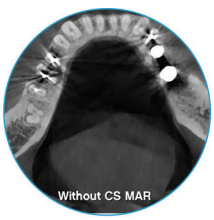

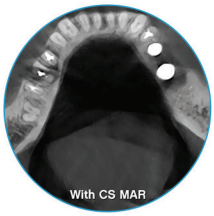

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COVID-19 risk management in dental practice Part 2: The infection chain pathway of SARS-CoV-2

Johan Hartshorne[1] and Andre van Zyl[2]

Keywords: aerobiology, aerosols, airborne, droplets, coronavirus, co-morbidities, COVID-19, SARS-CoV-2, dentistry, risk management, reservoir, transmission, infection chain, susceptible host

Executive Summary

Rationale

Understanding the coronavirus (SARS-CoV-2) and its pathways from its reservoir to host can help us understand how to fight the virus, and is critical in developing effective and sustainable infection prevention and control measures.

Key points

The pathogen – SARS-CoV-2

- SARS-CoV-2 is the most contagious of all the respiratory viral infections.
- The main sources of SARS-CoV-2 are asymptomatic, pre-symptomatic, symptomatic COVID-19 individuals in the population.
- SARS-CoV-2 transmission from asymptomatic and pre-symptomatic hosts are a fundamental fault- line in the spread of COVID-19 because we do not know who they are.
- The average incubation period (infectious period) is 6.4 days (range 2-24 days)
- SARS-CoV-2 is very stable at room temperature, wide range of pH and on smooth surfaces (including glass, plastic, and stainless steel).
- SARS-CoV-2 is stable on stainless steel and plastic for up to 9 days.
- Detectable levels of infectious virus is still present on the outer layer of a surgical mask at 7 days.
- SARS-CoV-2 is very susceptible to standard disinfection materials, including 70% ethanol, household bleach and 7.5% povidone-iodine.

Reservoir

- SARS-CoV-2 infectious cycle can start in the lungs, naso-pharynx, oral mucosa, tongue and salivary glands.
- During the first 10 days of incubation the virus mainly accumulates in the pharyngeal, oral and nasal areas.
- SARS-CoV-2 is consistently detected in saliva.

Portal of exit (leaving the host reservoir)

- Individuals with infection produce respiratory droplets or aerosol particles from breathing, talking, singing, coughing and sneezing

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- Common exit portals for SARS-CoV-2 are the mouth, nose, respiratory tract and faecal route.

Mode of transmission

- Transmission of SARS-CoV-2 can occur from asymptomatic and symptomatic individuals.
- SARS-CoV-2 is mainly transmitted through close physical contact and respiratory droplets.
- Airborne transmission is possible during aerosol generating procedures.
- Large droplets (>5 μm) settle faster due to gravity, thus contaminating surrounding surfaces.
- Smaller droplets (<5 μm) evaporate faster forming droplet nuclei that can stay airborne for hours.
- Aerosolized viral droplet nuclei particles can travel great distances and remain airborne and viable for up to 3 hours and can infect dental health care workers, patients and contaminate surfaces.

Portal of entry and replication

- Portal of entry is through mouth, nose, respiratory tract.
- SARS-CoV-2 S-spike protein invades host target cells by using ACE2 as its receptor.

Susceptible host and disease pathogenicity

- Infected hosts can present with clinically inapparent (asymptomatic) or mild (80%), moderate severe (14%) or critical illness requiring hospitalization (6%).
- Individuals with co-morbidities presented with increased COVID-19 severity and higher case fatality rates.
- Hypertension and hyperlipidaemia were the most frequent co-morbidities.
- Elderly and immune-compromised individuals are most vulnerable
- Individuals with periodontitis may be linked to more severe COVID-19

Practice implications

- The disturbing reality is that we have no idea who among us is spreading the disease.
- Early recognition of an infected person (source of infection) and cutting off the route of transmission are key points to control COVID-19.
- A decrease in the oral viral load would diminish the amount of virus expelled and reduce the risk of transmission.
- Pre-procedural mouth rinse is a critical prophylactic measure for reducing oral viral load and risk of spreading SARS-CoV-2.
- The weight of combined evidence supports airborne precautions for occupational health and safety of health workers treating asymptomatic or suspected patients with COVID-19.
- Ventilation is of critical importance to control airborne transmission of SARS-CoV-2

Introduction

The global outbreak of coronavirus disease (COVID-19) is caused by the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). It is the most contagious of all the viral respiratory pandemics (Part 1 – Table 1) and spreading fast with an increasing number of infected patients world-wide. The current global statistics on February 22, 2021 (11:39 GMT), show the total number of confirmed cases 112,026,236, total deaths 2,479,303 and total recovered /discharged 87,392,155 individuals. Current global active infected cases 22,154,178 (19,8%) of which 22,060,880 (99,6%) have a mild condition and 93,898 (0.4%) are serious / critical.[1] The COVID-19 statistics for South Africa on February 22, 2021 showed a total number of 1,503,796 cases, total deaths 49,053 and total recovered 1,412,015 cases. Current active COVID-19 cases in South Africa on February 23, 2021 were 41,872 of which 546 (1.3%) cases are serious/critical.

Outbreaks of newly emerging infectious viral diseases present unique challenges and threats to health care providers due to lack of immunity, absence of specific, effective, and safe antiviral drugs, and a limited understanding of the emerging threat and reliance on infection prevention and control measures.

A series of events has to happen to enable a pathogen such as SARS-CoV-2 to cause an infection (COVID-19). This series of events is referred to as the 'chain of infection'. The links of the infection chain consist of : (i) the pathogen (infectious agent), (ii) reservoir or source, (iii) portal of exit, (iv) mode of transmission, (v) portal of entry, and (vi) a susceptible host.[2,3] The chain of infection model holds that infectious diseases result when an agent (pathogen) leaves its reservoir or host through a portal of exit, is conveyed by some mode of transmission, and enters through an appropriate portal of entry to infect a susceptible host.

The spread of infection can be mitigated by breaking the infection chain at any of its links. Therefore understanding the setting and characteristics of each link of the infection chain and how SARS-CoV-2 spreads to a susceptible host is critical in developing effective and sustainable infection prevention and control measures in the absence of a vaccine and anti-viral drugs.

Methodology and Purpose

The literature search methodology used for the data assimilation and knowledge synthesis in this series is described in Part 1. The epidemiological 'infection chain' model was also used to enhance data assimilation and knowledge synthesis. (Table 1)

Part 2 of this series will focus on the key parameters of the infection chain that impact directly on risk management in the dental practice (Table 1)[2,3] In addition, Part 2 also provides a review of the current knowledge and understanding of aerobiology and flow physics implicated in the generation, expulsion, evolution and transmission of virus-laden droplets and aerosols generated during expiratory activities such as breathing, talking, coughing and sneezing and during aerosol generating procedures.

This knowledge will enhance dental practitioners understanding the what, the why, and the how, underpinning SARS-CoV-2 infection control and prevention.

Pathways of the infection chain – What is our knowledge and understanding of SARS-CoV-2 (virus) and susceptible host characteristics?

1. The pathogen – How virulent and infectious is SARS-CoV-2?

• *The origin and identification of the coronavirus SARS-CoV-2*

In December 2019, a cluster of fatal pneumonia outbreaks originated in Wuhan City, China.[4] All patients had been associated with the Wuhan Wholefood Market, where seafood and live animals are sold. The disease spread rapidly to most provinces in China and subsequently the rest of the world.[5,6]

Chinese researchers quickly isolated a new virus from a patient and sequenced its genome (29,903 nucleotides).[7] The infectious agent of this viral pneumonia that originated in Wuhan was finally identified as a novel coronavirus (2019-nCoV), the seventh member in a family of coronaviruses that affect humans.[8] After analysis of respiratory samples, the Peoples Republic of China Centers for Disease Control declared that the pneumonia was caused by a novel coronavirus now referred to as SARS-CoV-2.[9]

Coronaviruses (CoV) are respiratory pathogens. They belong to the Coronaviridae family. Currently there are four genera of coronaviruses: α-CoV, β-CoV, λ-CoV, and δ-CoV.[10] SARS-CoV-2 is an enveloped single stranded RNA virus. Six corona viruses were previously known to cause disease in humans, SARS-CoV-2 is the seventh member of the coronavirus family that infects humans after SARS-CoV, MER-CoV.[4] and the Middle East respiratory syndrome coronavirus (MERS-CoV)[12] that occurred in 2002-2003 and in 2012 respectively. SARS-CoV, MERS-CoV and SARS-CoV-2 belong to β-CoV.7.[13]

• *The morphologic and genetic structure, replication and pathogenic mechanisms*

The morphologic structure of SARS-CoV-2 is similar to SARS-CoV, with virion size ranging from (60-140 nm) (0.06-0.14μm).[14] The virus has distinctive spikes of 9-12 nm that give the appearance of "coronas" around the sun. Spike, membrane and envelope surface viral proteins of the coronavirus are embedded in its host-derived lipid bi-layer membrane encapsulating the helical nucleocapsid comprising viral RNA.[15]

SARS-CoV-2 presents with two notable genomic features: (i) it is optimized for binding to human receptor angiotensin-converting enzyme 2 (ACE) and, (ii) the receptor binding domain in the spike S-protein has a site at the S1-S2 boundaries (the two subunits of the spike) through which insertion of nucleotides takes place. The cleavage site on the S-protein allows effective cleavage by furin (protease) and other proteases that allows nucleotides of SARS-CoV-2 to enter host cells through human cell receptor ACE2, to allow viral entry, duplication, infectivity and host range.[16-18]

ACE2 is an important receptor for SARS-CoV-2 [19] and found in many target cells including type II alveolar cells of lung[20-22], epithelial cells of the oesophagus, adsorptive enterocytes from the ileum and colon[22], cholangiocytes[23], myocardial cells, kidney proximal tubule cells, bladder and urothelial cells[20], salivary gland epithelial cells[24,25] and oral mucosa.[26]

Viral entry and cell infection trigger the host's immune response, and the inflammatory cascade is initiated by antigen presenting cells.[27] It is suggested that the severity of the virus infection is closely related to the maturity and binding capacity of ACE2.[28] Gao and co-workers[29] have suggested that a lower level of ACE2 and weaker binding could be a major contributing factor that leads to the absence of any clinical manifestations in asymptomatic cases.

• *What is known about the incubation period of SARS-CoV-19?*

Incubation period is the time from moment of exposure to the corona virus until signs and symptoms appear. The best current estimates of the incubation of SARS-CoV-2 range from 2-14 days with an average of 6.4 days.[27,30,31]

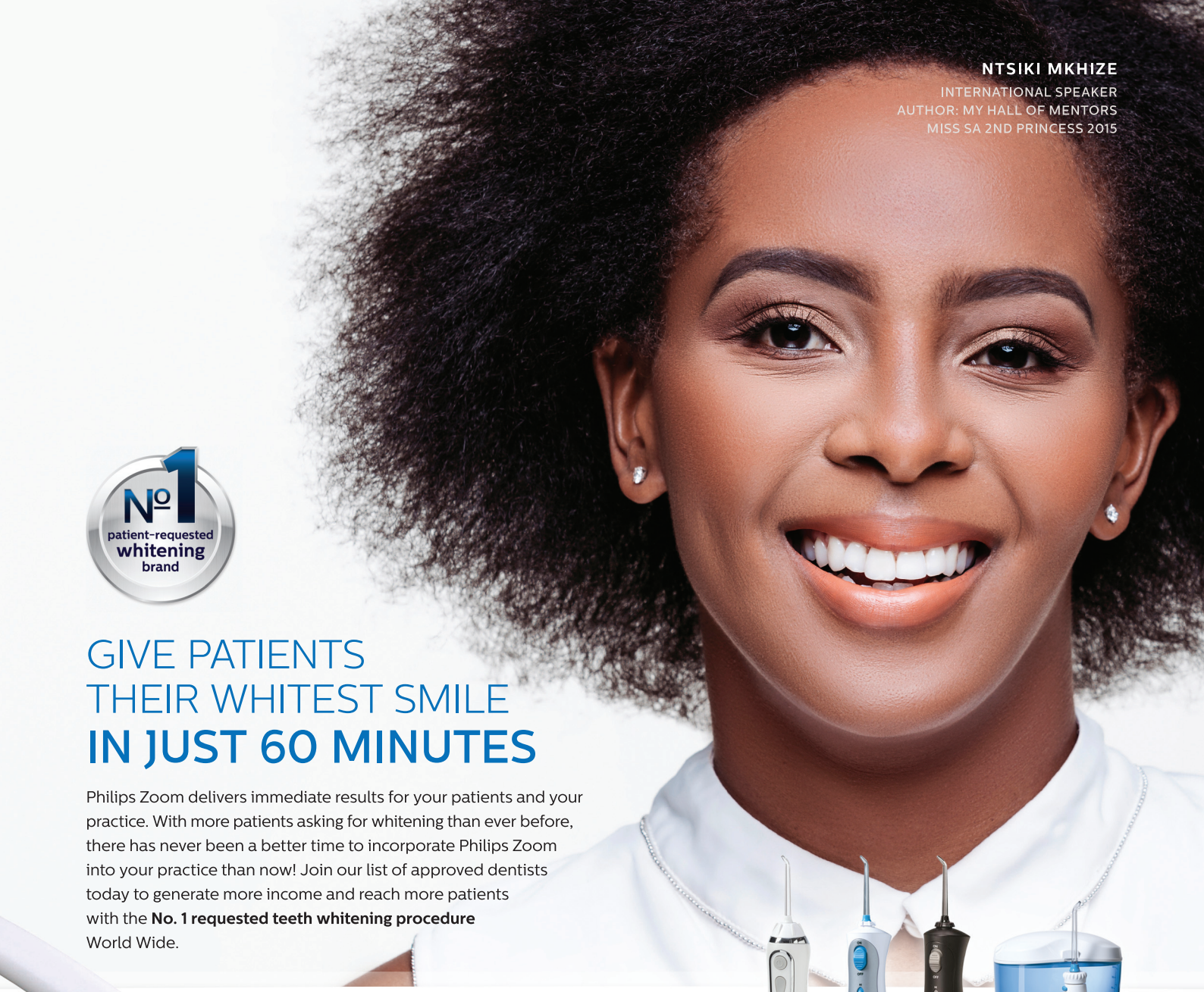

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Table 1 : The infection chain pathway from pathogen to host and appropriate infection control strategies to mitigate or contain transmission of the coronavirus between health care workers and patients in the dental practice setting

Infection chain pathway and definition	Infection chain characteristics	Infection control strategy
Pathogen of sufficient virulence and adequate number (load) to cause disease	• Contagion: Coronavirus (SARS-CoV-2) • Single stranded RNA • Enveloped – lipid bi-layer membrane • Diameter 60-140nm (0.06-0.14µm) • Spike protein 9-12nm - viral entry key • Very susceptible to standard disinfection methods • Very contagious	• Hand sanitizing – removes virus • Surface disinfection – Kills virus • Universal masking – evade virus • Social distancing – evade virus • Isolation • HEPA filters (virus scavenging) & UV light sterilization • HOCL fogging - airborne disinfection • Anti-viral drugs • Vaccine – antibody immune resistance
Reservoir or source (carrier) (A place that allows the pathogen to survive or multiply)	• Incubation (infectious period) 6.4 days • Human COVID-19 pre-symptomatic • Human COVID-19 asymptomatic • Respiratory tract (naso-pharynx and lungs • Oral cavity (Oral mucosal epithelial cells, salivary glands, tongue and periodontium) • Gastro-intestinal tract (intestinal epithelium) • Possible environmental reservoirs: Biofilm in waterlines and Ventilation systems	• Patient and staff screening for symptoms • Universal masking (evade) and hand sanitation (remove virus) • Maintain good hygiene and sanitation • Pre-procedural mouth rinse – kills virus and reduces viral load • Asepsis / sterilization • Waterline Disinfection • HEPA filters & UV sterilization
Portal of exit (Ways in which the virus leaves the reservoir)	• Mouth (talking) aerosols • Mouth - Contaminated saliva (aerosols) • Mouth - Respiratory secretions (air droplets (coughing or talking) • Nose - Respiratory secretions (air droplets) -sneezing • Faecal	• Pre-procedural rinse & gargle • Rubber dam isolation • High volume evacuation • PPE (Masks and gloves) • Hand sanitation
Mode of transmission from source to host (Ways in which the virus spreads from reservoir to the susceptible host)	• Direct contact (touch) with contagion (pathogen) in saliva or surfaces • Indirect contact with contaminated surface/objects (fomites) • Contact with conjunctiva, nasal or oral mucosa with contaminated droplets (coughing, sneezing and talking) • Inhalation of airborne microorganisms suspended in air • Aerosol generating procedures • Faecal-oral route • Superspreading events	• Hand sanitizing • Pre-procedural mouth rinse • Isolation – use of rubber dam • High volume evacuation • Appropriate PPE (Masks, Gloves, Gowns, Shields) • Appropriate surface disinfection • Ventilation, HEPA filters & UV sterilization • Prevent & control Superspreading events
Portal of entry (Ways through which the pathogen can enter a susceptible host)	• Deposition • Attachment (ACE2 receptors) & Entry • Replication and release • Respiratory tract • Nose (nasal mucosa) • Mouth (oral mucosal)	• Preprocedural mouth rinse • Surgical masks (FFP2) or N95 respirators (FFP3) • Goggles and/or face shields • Appropriate disinfection
Susceptible host Is an individual who is not immune Susceptible individuals may have co-morbidities that affect their susceptibility to, and severity of COVID-19	• Healthy individual • Immune compromised individual • Elderly • Co-morbidities • Smoking • Patients receiving ACE2-increasing drugs • Disease: COVID-19 • Asymptomatic • Pre-symptomatic • Symptomatic	• Pre-screening& risk identification • Isolation • Diagnostic testing • Universal masking • Hand sanitize • Preprocedural mouth rinse • Maintain good hygiene • Special precautions for individuals at high risk & co-morbidities • Enhance the immune system • Vitamin D supplementation • Healthy nutrition, reduce stress, adequate sleep • Eliminate smoking • Social distancing • Mucus modification • Anti-viral drugs • Designer antibodies & Vaccination

The maximum incubation period observed is as high as 24 days which suggests that this may increase the risk of virus transmission. Studies also suggest that elderly people have shorter incubation periods; thus,faster disease progression.[32] Transmission of SARS-CoV-2 can occur in the pre-symptomatic and symptomatic period.[33] Recent studies have revealed important transmission features of SARS-CoV-2, including infectiousness of asymptomatic[34-38] and pre-symptomatic cases.[39-41]

- ***How long do individuals shed infectious SARS-CoV-2 RNA after infection?***

Although a precise estimate of residual risk of SARS-CoV-2 transmission after recovery from COVID-19 cannot be generated at this time, it is likely substantially less than the risk during illness when most person to person transmissions occurs.[42] It is impossible to say with 100% certainty that all recovered individuals are no longer infectious. Persons who are immunocompromised may have prolonged viral shedding.[42] COVID-19 testing is not always possible and/or accurate to make a determination whether a patient is infectious or not. The viral burden in saliva usually declines after onset of illness.[42]

The CDC recommends that isolation be maintained for at least 10 days after illness onset (Illness onset is defined as the date symptoms began), and least 3 days after recovery. Recovery is defined as resolution of fever without use of fever-reducing medication or resolution of other symptoms.[42] Duration of infectious period for COVID-19 is approximately 10 days after the incubation period.[43]

- ***What is the stability of the virus in different environmental conditions?***

The virus is highly stable at 4°C, but sensitive to heat. Infectious virus could be recovered from printing or tissue paper after 3 hours whereas no virus could be detected from wood and cloth on day 2. By contrast SARS-CoV-2 was more stable on smooth surfaces on day 4 (glass and banknote) or day 7 (stainless steel and plastic.) Strikingly, a detectable level of infectious virus could still be present on the outer layer of a surgical mask on day 7.[44]

No infectious virus could be detected after a 5-minute incubation with various disinfectants (household bleach, 70% ethanol, 7.5% povidone-iodine, 0.5% chlorhexidine and 0.1% Benzalkonium chloride) at room temperature, and therefore very susceptible to standard disinfection methods.[44] SARS-CoV-2 is extremely stable in a wide range of pH values (pH 3-10) at room temperature.

- ***Transmission kinetics of SARS-CoV-2***

The efficiency of transmission for any respiratory virus has important implications for containment and mitigation strategies. (Infection prevention and control strategies) Studies suggest an estimated reproduction number (R0) of 2.2, which means that on average, each infected person will spread the infection to an additional two individuals. Until the number falls below 1.0, it is likely that the outbreak will continue to spread.[45]

Serial interval of COVID-19 is defined as the time duration between a primary case (infector) developing symptoms and the secondary case (infectee) developing symptoms.[46,47]

The basic reproduction number, which has been widely used and misused to characterize the transmissibility of the virus, hides the fact that transmission is stochastic, is dominated by a small number of individuals, and is driven by super-spreading events (SSE's).[48]

2. Reservoirs (source or target organ): SARS-CoV-2 infectious cycle

The reservoir of an infectious agent is the habitat in which the agent or pathogen normally starts its infectious cycle, lives, grows, and replicates. Reservoirs include animals, humans, and the environment. The reservoir may or may not be the source from which the pathogen is transferred to the host.

- ***Zoonosis and animal reservoirs***

Similar to other viruses, SARS-CoV-2 has many potential natural- intermediate- and final hosts. This poses great challenges to prevention and treatment of virus infections.

Humans are also subject to diseases that have animal reservoirs. Many of these diseases are transmitted from animal to animal, with human as incidental hosts. The term zoonosis refers to an infectious disease that is transmissible under natural conditions from vertebrate animals to humans. Genomic characterization of SARS-CoV-2 has shown that it is of zoonotic origin. Scientists agree that the coronavirus SARS-CoV-2 very likely originated in bats (natural source)[8] whilst pangolins and snakes may be intermediate hosts.[18]

- ***Human reservoirs***

Many common respiratory infectious diseases have human reservoirs. Diseases that are transmitted from person to person without intermediaries. Human reservoirs may or may not show the effects of illness. Asymptomatic or passive carriers are those who do not experience symptoms despite being infected. Incubatory carriers are those who can

transmit the pathogen (virion) during the incubation period (pre-symptomatic) before clinical illness begins.[3] Researchers have shown the role of the oral mucosa and salivary gland epithelial cells with high expression in ACE2 in SARS-CoV-2 infection.[24,26]

Current evidence suggests that SARS-CoV-2 transmitted by asymptomatic infected individuals may originate from infected saliva.[25] Asymptomatic carriers commonly transmit disease because they do not realize they are infected, and consequently take no special precautions to prevent transmission. Symptomatic persons who are aware of their illness, on the other hand, may be less likely to transmit infection because they are too sick to be out and about, take precautions to reduce transmission, or receive treatment that limits the disease.[3]

At present it is considered that the main source of SARS-CoV-2 are pre-symptomatic, symptomatic and asymptomatic COVID-19 individuals in the population.[18,49]

Reservoirs are places where SARS-CoV-2 infectious cycle starts, where it can replicate and survive i.e., lungs, nasopharynx, oral cavity (oral mucosa, salivary glands, tongue and possible the periodontium), and gastro-intestinal tract. Viruses are obligate intracellular parasites. They cannot produce outside of a cell. The sum total of all the events that take place in a virus infected cell or reservoir is called the infectious cycle, or viral replication. Once inside the cell, the virus hijacks the cellular machinery forcing it to produce more viruses.[50] These events consist of: (i) attachment, (ii) entry of the virion, (ii) uncoating and translation of mRNA into protein, (iii) genome replication, (iv) assembly of new particles, (v) and release of new particle (virions) from the host cell.[51] SARS-CoV-2 has been identified in both upper and lower respiratory tract samples from patients.[52] Higher viral loads have been detected in nasal passages and the upper respiratory tract of individuals infected with SARS-CoV-2, which means that coughs and sneezes may contain higher viral loads. One factor that is contributing to the rapid growth of COVID-19 infections is the higher viral load of the SARS-CoV-2 virus in the upper respiratory tract of asymptomatic hosts who shed virus-laden droplets during normal activities such as talking and breathing.[34]

Oral viral load of SARS-CoV-2 has been associated with severity of COVID-19, and thus, a reduction in the oral viral load could be associated with a decrease in the severity of the condition.[53] A decrease in the oral viral load would diminish the amount of virus expelled and reduce the risk of transmission.[53]

SARS-CoV-2 is primarily thought to infect lungs with transmission via the respiratory route. However clinical evidence suggest that the oral cavity,[26] salivary gland epithelial cells[24] and intestine[54] may present as viral target organs or potential reservoirs for SARS-CoV-2.

ACE2 is an important receptor for SARS-CoV-2 19 and highly expressed in salivary gland epithelial cells.[24] and the oral mucosa.[26] It is suggested that there may be an increased dental risk due to SARS-CoV-2 transmitted by asymptomatic infection that may originate from saliva especially during aerosol generating procedures.[25] It is also hypothesized that periodontal pockets may be a plausible reservoir for SARS-CoV-2.[55]

The SARS-CoV-2 receptor ACE2 is also highly expressed on differentiated enterocytes.[56]

- ***Environmental reservoirs***

The environment such as ventilation systems, sanitation facilities, waterlines and biofilms may also be reservoirs for SARS-C0V-2.[57-59] However, no studies have reported or suggested the possibility of ventilation systems, sanitation systems and waterlines being possible reservoirs or source of infection.

3. Portal of exit – How does the coronavirus leave the host reservoir?

Portal of exit is the path by which a pathogen leaves its host, corresponding to the site where the pathogen is localized.[3] During the infectious period, every individual emits potentially infectious aerosols all the time, not just when sneezing or coughing.[60]

Common portals of exit for SARS-CoV-2 include the mouth (breathing, talking, coughing, singing, aerosol generating procedures), nose (sneezing), respiratory tract (oro-pharynx and nasopharynx) (sputum production), and now added, the faecal route.[59]

Production of infectious respiratory droplets or particles are dependent on the type and frequency of respiratory activity, type and site of infection and viral load. Furthermore, relative humidity, particle aggregation, and mucous properties influence expelled particle size and subsequent transmission.[61]

- ***Respiratory droplets and aerosols***

Individuals with infections produce particles between 0,05 and 500μm from breathing, talking, coughing and sneezing.[61] This indicates that expelled particles carrying pathogens do not exclusively disperse by droplet or airborne transmission but avail of both methods simultaneously and current infection control precautions should be updated to

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include both methods of aerosolized transmission.[61]

Respiratory droplets are formed from the fluid lining of the respiratory tract (oro- and naso-pharyngeal complexes).[62,63] The mechanisms of formation are usually associated with distinct locations in the respiratory tract and both the characteristics of the respiratory tract as well as the viral load carried by the lining are functions of the location.[63,64] One key mechanism for the generation of respiratory droplets is the instability and eventual fragmentation of the mucous lining due to shear stress induced by the airflow.[65] The Rayleigh-Taylor instability (the instability between two fluids when the lighter fluid is pushing the heavier one) is particularly important in spasmodic events such as coughing and sneezing.[66,67]

The second mechanism for droplet formation is associated with the rupture of the fluid lining during the opening of a closed respiratory passage.[68]

These submillimetre-sized passages collapse during exhalation, and the subsequent reopening during inhalation ruptures the mucus meniscus, resulting in the generation of micron sized droplets.[63,64] A similar mechanism probably occurs in the larynx during activities such as talking and coughing, which involve the opening and closing of the vocal folds.[69] Finally, movement and contact of the tongue and lips, particularly during violent events such as sneezing, generate salivary droplets.[70] Higher viral loads have been detected in nasal passages and the upper respiratory tract of individuals infected with SARS-CoV-2, which means that coughs and sneezes may contain higher viral loads.[34]

- ***Saliva – oral droplets and aerosol generating procedures***

Several studies have confirmed that the viral load in human saliva is very high and that pre-operative mouth rinses can reduce this but cannot eliminate it.[71,72] In terms of coronavirus, Wang and co-workers examined the oral cavity of SARS patients and found large amount of SARS-CoV-2 RNA in their saliva (7.08×103 to 6.38×108 copies/mL).[73] This suggests a strong possibility of coronavirus transmission through oral droplets. According to Chowell and co-workers, evidence shows that the majority of SARS-CoV and MERS-CoV cases are associated with nosocomial transmission in hospitals, partly from aerosol-generating procedures.[74]

Recent reports of high viral load in the oropharynx early in the course of the disease aroused concern about increased infectivity during the period of minimal symptoms.[43,75] The potential for individuals infected with SARS-CoV-2 to shed and transmit the virus while asymptomatic is greater, and those in the latent stages of the diseases often shed the virus at a higher rate.[34]

- ***Gastrointestinal system – faecal route a potential portal of exit for SARS-CoV-2***

Evidence suggests that SARS-CoV-2 can infect and be shed from the gastrointestinal tract (faecal-oral route).[56,59] In addition, researchers have also detected SARS-CoV-2 in stool samples, gastrointestinal tract, saliva and urine.[18] There is evidence of ingestion, penetration of enterocytes and excretion of live SARS-Co-V-2 through the faecal route.

4. Mode of transmission

Human-to-human transmission of SARS-CoV-2 from its reservoir to a susceptible host occurs primarily via four routes: (i) large droplets from infected respiratory or saliva secretions that are expelled with sufficient momentum (i.e., coughing, sneezing, talking, singing) so as to directly impact the host recipients' mouth, nose or conjunctiva (droplet transmission)[76] (ii) physical contact with infected droplets deposited on a surface (fomite transmission) and subsequent transfer to the recipients' respiratory mucosa, conjunctiva or oral mucosa (contact transmission)[76,77,78] (iii) inhalation by the recipient of aerosolized droplet nuclei that are delivered by ambient air currents (airborne transmission)[70-81] and (iv) faecal-oral route of transmission.[59]

According to current evidence, SARS-CoV-2 is primarily transmitted between people through respiratory droplets and contact routes.[9,18,30,82-85] However recent evidence suggest that the airborne transmission route may be highly virulent and dominant for the spread of SARS-CoV-2.80 SARS-CoV-2 is mainly transmitted through close physical contact and respiratory droplets, while airborne transmission is possible during aerosol generating procedures.[78,86]

(i) Droplet and aerosol transmission

Transmission of SARS-CoV-2 is primarily via virus-laden fluid particles, namely droplets (>5 μm) and aerosols (<5 μm) (also referred as droplet nuclei) that are formed in the respiratory tract of an infected person and expelled from the mouth and nose during breathing, talking, coughing and sneezing or during aerosol generating procedures.[60,72,87] Viral transmission can occur when viral particles are aerosolized by a cough, sneeze or during dental procedures. According to Froum and Strange, particles can travel up to a distance of 6m from an infected person and have the potential to incite secondary infections.[88]

- ***Respiratory droplets and aerosols***

Asymptomatic and pre-symptomatic individuals, by definition do not cough or sneeze to any appreciable extent. This leaves direct or indirect contact modes and aerosol (airborne) transmission as the main possible modes of transmission. Both breathing and talking emit large quantities of aerosol particles, typically about 1 µm in diameter and are large enough to carry viruses such as SARS-CoV-2 to be readily inhaled deep into the respiratory tract of another individual.[60]

Ordinary speech aerosolizes significant quantities of respiratory particles. Studies suggest that speech emits more aerosol particles than breathing[89] and the louder one speaks, the more aerosol particles are produced.[90] It is plausible that a face-to-face conversation with an asymptomatic infected individual, even if both individuals take care not to touch or to maintain social distancing, might be adequate to transmit SARS-CoV-2.

Respiratory droplet transmission (droplet particle size >5-10 microns) occurs when a person is in close contact (within 1 m) with someone who has respiratory symptoms e.g., coughing or sneezing and is therefore at risk of having his/her mucosae (mouth or nose) or conjunctiva (eyes) exposed to infective droplets.[86] It is conceivable that infectious particles sized less than 10 µm have more serious health implications as they are able to penetrate into the lower respiratory tract to establish infection.

- ***Aerosol generating procedures***

Aerosol generating procedures (AGP) are defined as any dental and medical care procedure that results in the production of airborne particles (aerosols). AGP's can produce particles <5 µm in size which can remain suspended in the air and travel over a distance, causing infection when inhaled. AGP create the potential for airborne transmission of infections that may otherwise be transmitted by droplet route.

Aerosols and droplets are produced during many dental procedures (i.e., use of air turbines during restorative procedures, surgical handpieces, air abrasion, use of a 3-in-1 syringe, ultrasonic or sonic scalers, air polishing devices and use of ErYAG laser with water coolant function. Splatter droplets are much larger than aerosol particles (<50 micron). The size of the coronavirus-shaped spherical particle is estimated to be about 0.125 microns (125 nm) (range: 0.06 microns to 0.14 microns).[4] It is therefore plausible that both aerosol particles and splatter droplets can contain SARS-CoV-2 and therefore a potential hazard for health care workers, including dentists.

- ***Aerobiology and physics of aerosolization:***

Determining the fate of droplets and aerosols and transmission rates

Size, velocity, inertia, gravity and evaporation are key determinants of the fate of droplets, pathogen carriage, aerosolization, and transmission.[61]

- ***Temperature, humidity and evaporation***

Higher temperatures and lower relative humidity lead to larger evaporation rates that increase the critical droplet size.[91,92] Wells' simple but elegant analysis predicted that the critical size that differentiates large from small droplets is approximately 100µm.[91] Subsequent analysis has shown that typical temperature and humidity variations expand the critical size range from approximately 50 to 150µm.[92]

Droplet evaporation plays a significant role in the eventual fate of a droplet.[91] Large droplets settle faster than they evaporate, and so contaminate surrounding surfaces. Smaller droplets evaporate faster, so forming droplet nuclei that can stay airborne for hours and may be transported over long distances.[70]

Dependence of evaporation rates on ambient temperature and humidity has implications for the very important, and as yet unresolved, questions regarding seasonal and geographic variations in transmission rates.[93,94] as well as airborne transmission in various indoor environments.[95,96]

- ***Velocity***

The number, density, velocity and size distributions of droplets ejected by expiratory events have important implications for aerosolization, pathogen carriage and transmission of respiratory infectious disease.[61,70] A single sneeze can generate 40,000 or more droplets, with velocities upwards of 20 ms-1.[97] Coughing generates approximately 3,000 droplets, with velocities of approximately 10ms-1, but even talking can generate approximately 50 particles per second.[90] Breathing and talking generate jet velocities that seldom exceed 5ms-1 and mostly expel small droplets.[98] Recent studies have noted that, while breathing and talking generates droplets at much lower rate, it probably accounts for more expired bioaerosols over the course of a day than intermittent events such as coughing and sneezing.[99,100]

Droplet characteristics (number, density, size distribution and velocity) continues to be elusive due to the multifactorial nature of the phenomena as well as difficulty of making such measurements.[89,97,101]

- ***Turbulence and cloud dynamics***

It has also been shown that the respiratory jet transforms into a turbulent cloud or puff.[102] While large droplets are mostly not affected by the cloud dynamics, small and

medium-sized droplets can be suspended in the turbulent cloud for a longer time by its circulatory flow, thereby extending the air travel distance significantly.[102] This also has important implications for transmission via indirect contact with contaminated surfaces, since SARS-CoV-2 is able to survive on many types of surfaces for hours to days.[103] In addition, the turbulent cloud also moves upwards due to buoyancy[102] thereby enabling small droplets and droplet nuclei to reach heights where they can enter the ventilation system and accelerate airborne transmissions.[70] The notion of critical droplet size that was introduced by Wells[91] might need to be re-examined in view of our rapidly evolving knowledge about these expiratory events.[92,102]

- ***Diffusion***

Diffusion mainly occurs through coughing, sneezing, talking, singing and saliva aerosols. For the droplet transmission route, an important consideration is the horizontal distance travelled by large droplets. The 3-6 feet social distancing guidelines probably originate from Wells' original work.[70] However, studies indicate that while this distance might be adequate for droplets expelled during breathing and coughing,[92,104-106] large droplets expelled from sneezes may travel 20 feet or more.[92,102] Studies also suggest that social distancing in indoor environments could be complicated by ventilation-system-induced air currents.[107]

(ii) Direct and indirect contact transmission (Fomite transmission)

Direct or indirect contact modes require a susceptible individual to physically touch themselves i.e., oral, nasal, and eye mucous membranes with, for example, a virus-contaminated hand.[72] "Direct" indicates that person-to-person contact transfers the virus between infected and susceptible host (such as by hand shake), while "indirect implies transmission via a "fomite" which is an object like a light handle or x-ray tube that has been contaminated with infectious virus.[60] SARS-CoV-2 can also be transmitted to fomites aerosol generating procedures. SARS-CoV-2 may also be transmitted directly to surfaces, handles or equipment (fomites) due to poor hand sanitation.

Transmission may also occur through fomites in the immediate environment around the infected person.[108] Currently there is no evidence linking transmission of SARS-CoV-2 conclusively to contaminated environmental surfaces.[109]

(iii) Airborne transmission

Studies suggest that coronavirus has been detected on particles of dust or polluted air thus enabling coronavirus to be carried over longer distances air borne, potentially increasing the risk of infection.[110-113]

The airborne transmission route is associated with small droplets that are suspended and transported in air currents over longer distances. Under certain humidity and temperature environments, airborne droplets (aerosols) can remain in flight for hours.[27] Smaller droplets evaporate faster than they settle, forming droplet nuclei that can stay airborne for hours and may be transported over long distances.[70] Most of these droplets evaporate within a few seconds[92] to form droplet nuclei. The nuclei consist of virions and solid residue[114] but water may never be completely removed.[115]

Droplet nuclei are sub-micrometer to approximately 10µm in size, and remain suspended in the air for hours.[70] Each droplet nucleus could contain multiples virions, and, given the approximately one hour viability half-life of the SARS-CoV-2 virus,[103] and the fact that SARS-type infections in a host may potentially be caused by a single virus,[116] droplet nuclei play a singularly important role in the transmission of SARS-CoV-2.[60] The transport of droplet nuclei over larger distances is primarily driven by ambient airflows. Indoor environments such as homes, offices, hospitals, malls, aircraft and public transport vehicles pose a particular challenge to disease transmission. The importance of ventilation in controlling airborne transmission of infections is well known.[95,96]

In the context of dental practice, airborne transmission may be possible where aerosol generating procedures are performed; (e.g., ultrasonic scalers, use of air turbines, 3-in-1 syringes).[86] However, there have been no evidence-based reports on aerosol generated transmission to date. Studies are needed to determine whether viable SARS-CoV-2 is found in air samples in dental rooms where non-aerosol and aerosol generating procedures are performed. Current available evidence suggests that long-range aerosol-based transmission is not the dominant mode of SARS-CoV-2 transmission.[117]

Once infected droplets have landed on surfaces, their survivability on those surfaces determines if contact transmission is possible. Based on the current evidence, SARS-CoV-2 can remain infective, from 2 hours up to 9 days on inanimate surfaces, with increased survival in colder or dryer environments.[118-120] A study of people with Influenza found that 39% of people exhaled infectious aerosols.[121] If SARS-CoV-2 is transmitted in aerosols, then it is possible that virus particles can be transmitted over greater distances. Yan and co-workers also suggested that infected aerosols are

also produced during breathing and talking.[121] Therefore, it is suggested that when an air space is being shared, such as in a dental practice, breathing in infected air by airborne transmission is possible.[121]

(iv) Faecal-Oral route transmission

Many pathogens that cause gastroenteritis follow the so-called "faecal-oral" route because they exit the source host in faeces, are carried on inadequately washed hands to a vehicle such as food, water, or utensil, and enter a new host through the mouth.[3] SARS-CoV-2 has been detected in the faeces of some patients.

Thus taken together with fomite transmission, there is a potential possibility that SARS-CoV-2 could transmit via the faecal-oral route. The faecal-oral route describes a route of transmission where the virus particles can pass from one person to the mouth of another. Main causes included lack of adequate hand sanitation and poor hygiene and sanitation practices.[56]

5. Portal of entry and life cycle of SARS-CoV-2

The portal of entry refers to the manner in which a pathogen enters a susceptible host to initiate its lifecycle and pathogenicity. The portal of entry must provide access to tissues in which the pathogen can replicate. Often infectious agents use the same portal to enter a new host that they used to exit the source host.[3]

Viruses are basically molecular nanomachines that take over the host cell after entry and force it to produce numerous copies of themselves.[122] The life cycle of a coronavirus consists of the following stages: (i) deposition, (ii) attachment and entry, (iii) transcription and replication, and (iv) assembly and maturation, and (v) release.[51]

• *Deposition of droplets and aerosols (droplet nuclei)*

Infection entry points are through the mouth (oral mucosa), nose (nasal mucosa) and eyes (conjunctiva).[72] Inhalation or direct contact of virus-laden droplets and aerosols (droplet nuclei) and the deposition of the virus in the respiratory mucosa, oral mucosa, nasal mucosa, or conjunctiva of the host is the final stage of droplet or airborne transmission.[70] The nose typically filters air particles above 10μm. Therefore, if a particle is less than 10 μm, it can enter the respiratory system. Fine aerosol particle (<2.5μm) can enter the alveoli. Ultrafine aerosol particles (<0.1μm) such as SARS-CoV-2 can enter the bloodstream and target organs such as the brain and heart.

There are six mechanisms that determine the deposition location: impaction, sedimentation, interception, diffusion, electrostatic precipitation and convection.[124] The relative importance of these mechanisms depends on the particle size and the region of the airway where deposition occurs. For small droplet nuclei-sized particles, sedimentation will drive significant deposition in the upper respiratory tract of the host[125] and relies completely on turbulent diffusion, whereas deposition of larger droplets are driven by impaction, sedimentation and interception[126] and rely mostly on deposition velocity. Large droplets, despite a higher deposition velocity, probably deposit in the upper respiratory tract, and could be deactivated by the first defensive layer of the mucosa.[127] On the other hand, small droplet nuclei, despite their smaller deposition velocity, will penetrate deeper into the respiratory system, and this could affect the progression and intensity of infection.

Deposition of virus-bearing droplets in the respiratory tract does not always result in infection, since the mucus layer provides some level of protection against virus invasion and subsequent infection.[128]

• *Attachment and entry*

The S-protein of the virus interacts and binds to ACE2 in the first stage of virus replication called "attachment".[26,49] The specificity of this binding or "attachment" determines which cell type a virus can infect, a phenomenon called cell tropism.[51] ACE2 plays an important role in cellular entry,[29] thus ACE2-expressing cells are target cells and are susceptible to SARS-CoV-2 infection.[26,129] High ACE2 expression was identified in type II alveolar cells of lung,[20,21,22] epithelial cells of the oesophagus, adsorptive enterocytes from the ileum and colon,[22] cholangiocytes,[23] myocardial cells, kidney proximal tubule cells, bladder and urothelial cells.[20] Cells with high ACE2-expression should be considered as potential high risk for SARS-CoV-2 infection.[26] A recent study demonstrated that the ACE2 is expressed on the epithelial cells of the oral mucosa.[26] Interestingly, the ACE2 receptor was also highly expressed on the cells of the tongue. These findings support the plausible evidence that the oral cavity is potentially high risk for SARS-CoV-2 infection susceptibility.[26] Following receptor binding the virus enters the host cell cytoplasm.[51]

• *Transcription and replication*

Direct translation of the RNA-genome leads to the synthesis of structural and non-structural proteins (S, E, and M proteins)[51,123]

- ***Assembly and maturation release***

Following replication and sub-genomic RNA synthesis, the S, E, M proteins are translated and inserted into the endoplasmic reticulum where the viral genomes are encapsulated by a membrane via budding and resulting in the formation of mature virions.[51,123]

- ***Release of virions and initiation of pathogenicity***

Mature virions then travel to the cell surface inside vesicles and exit the cell by exocytosis to proceed with its pathogenic journey within the host.[51,123,130]

6. Susceptible host, co-morbidities and COVID-19

The final link in the chain of infection is the susceptible host. Susceptibility of a host depends on genetic factors, specific and non-specific immunity status, and factors that affect an individual's ability to resist infection such as age, immunodeficiencies, co-morbidities, stress, and nutritional deficiencies.[49]

- ***Susceptible host and risk factors***

An individual's genetic makeup or inborn errors of immunity may influence the immune response to infection thus either increasing or decreasing susceptibility and severity of developing the infectious disease COVID-19.[131,132] However, the role of human genetics in determining clinical response to the virus remains unclear.[132]

All groups are susceptible to COVID-19 regardless of age or gender. Patients aged 30-79 accounted for 86,6% of all cases.[30] Elderly male citizens are more susceptible to COVID-19 and studies showed a median age of death was 75. Most elderly affected had underlying comorbidities (e.g., diabetes, hypertension, heart disease etc)[133] or a history of surgery before admission.[32]

Factors that may increase susceptibility to infection by disrupting host immune defences include age (elderly), malnutrition, vitamin D deficiency, alcoholism, smoking, stress, obesity in males, hypertension, and therapies (e.g. cancer therapy, immune suppressors, ACE2 modulators) that may impair the non-specific or specific immune response.[134] Specific immunity refers to protective antibodies that are directed against a specific agent. Because this is a novel virus, individuals have no protective antibodies nor is there a vaccine available at this point in time (October 2020). Non-specific immunity that defend the host against infection include the skin, mucous membranes, the cough reflex, and non-specific immune responses.

With what we know about the pathogenesis of the SARS-CoV-2 virus, it seems reasonable to assume that those with higher levels of expression of ACE-2 receptors may be at greatest risk.[27]

- ***Diagnosis of COVID-19***

The detection of SARS-CoV-2 viral nucleic acid (RNA) by reverse transcriptase polymerase chain reaction (TR-PCR) serological test is the standard for non-invasive diagnosis of COVID-19.[29] However, the possibility of false negatives and the relative long testing time and availability of serological tests and resources for testing is a big problem.[18] Tthe radiographic features of coronavirus are similar to that found in community acquired pneumonia caused by other organisms. Chest CT-Scan is important to diagnose this pneumonia.[135]

- ***What are the clinical manifestations of COVID-19?***

Covid-19 is an acute viral infection with a mean incubation period of 6.4 days from onset of infection.[30,31] The most common clinical symptoms of COVID-19 observed in patients admitted to hospital in Wuhan, China were fever (89.9%), cough (67,7%), fatigue (38,1%), whereas diarrhoea (3.7%) and vomiting (5%) were rare.[133] In comparison symptoms commonly observed at hospital admission in Italy were fever (75%), dyspnoea (71%), cough (40%) and diarrhoea (6%).[136] A recent systematic review and meta-analysis showed that COVID-19 is characterized by the following most prevalent symptoms: fever [91.3% (95%CI: 86%-96%)], cough [67.7% (95%CI: 59-76%)], fatigue [51%, (95%CI: 34%-68%)], and dyspnoea, [34% (95%CI: 21%-40%)].[137] The typical clinical manifestations of patients who suffered from the novel viral pneumonia were fever, cough, and myalgia or fatigue with abnormal chest CT.[9,138,139] COVID-19 is now classified in 4 levels based on the severity of the symptoms: Mild (mild symptoms and no radiographic features); Moderate (fever, respiratory symptoms, radiographic features); Severe (one of the following : dyspnoea- (RR>30times /min); Oxygen saturation (<93; PaO2/F1O2 , 300mmHg); Critical (one of the following: respiratory failure, septic shock or multiple organ failure).[30]

Laboratory examinations revealed the following findings: lymphopenia (82.1%), thrombocytopenia (36.2%), elevated level of C-reactive protein (CRP), elevated levels of lactate dehydrogenase (LDH) and creatine kinase (CK).[9] Lymphocytopenia and cytokine storms are not exclusive to COVID-19 severity. Both are hallmarks of many other types of severe respiratory infections.[140] Increased ferritin levels and relatively low procalcitonin levels were commonly found

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in individuals with severe COVID-19 compared to those with moderate disease. Hypertension and hyperlipidaemia were the most frequent comorbidities. Individuals with severe Covid-19 had underlying pulmonary disease and the majority of individuals with severe COVID-19 presented with moderate to severe Acute Respiratory Distress Syndrome and hospital mortality was 25% within this group.[141] The presence of bacterial co-infection was also a common finding in individuals with severe COVID-19.[141] The potential role of periodontitis in bacterial co-infection or as a co-morbidity remains unclear and should be further investigated.[142]

A recent study has demonstrated that broad innate and adaptive leukocyte perturbations may be the cause of a dysregulated host immune response resulting in severe COVID-19 infection.[141] The general immune response landscape and their perturbations in severe COVID-19 presented with (i) elevated white blood cells and polymorphonuclear leukocytes, and (ii) lower frequencies of dendritic cells, CD8+ cells, innate lymphoid cells and natural killer cells.[141]

The neutrophil-to-lymphocyte ratio (NLR) has been proposed to be an independent risk factor for severe COVID-19. Both NLR as well as the neutrophil : T-cell ratio (NTR) were high in individuals with severe COVID-19, emphasizing and suggesting both as potential biomarkers of COVID-disease severity.[141] The data also indicate an exacerbated plasmablast response in severe COVID-19 cases. According to Kuri-Cervantes and co-workers, the top parameters driving the clustering of severe COVID-19 were associated with T-cell activation in the CD4+ and CD8+ T-cell memory subsets, frequency of plasmablasts and neutrophils.[141] According to the latter authors, the abovementioned immune dysregulation may necessitate targeted strategies to effectively manage clinical care.[141]

Currently there is no underpinning evidence to indicate what viral and/or human factors underpin whether a person with COVID-19 will develop a severe infection.

People infected with this highly contagious virus can present with clinically inapparent (asymptomatic), mild, moderate severe or critical illness requiring hospitalization.[143] Estimates show that about 80% of people with COVID have mild or asymptomatic disease, 14% severe disease, and 6% become critically ill.[6,144] Although the true case fatality rate is yet unknown, current model-based estimates ranged from 0.3% to 1.4% for countries outside China.[145]

Efforts to understand the pathogenesis and define the risk factors of severe COVID-19 has been hampered by our inability or unavailability of resources to identify all infected individuals, irrespective of clinical symptoms.[146]

There is increasing evidence that many infections of COVID-19 are asymptomatic, but they can transmit the virus to others.[29]

- ***Asymptomatic infections***

Asymptomatic infections are defined as positive detection of nucleic acid of SARS-CoV-2 in patient samples by reverse transcriptase polymerase chain reaction (TR-PCR) serological test, with no clinical symptoms or signs, and no apparent abnormalities in diagnostic images, including lung computed tomography.[29] The incidence of asymptomatic infections with COVID-19 in six different studies reported in a recent systematic review, ranged between 1.6% and 56.5%.[29] New evidence has emerged from China that 78% of new infections identified were asymptomatic.[147] In general, asymptomatic cases cannot be recognised if they are not confirmed by RT-PCT or other laboratory testing, and symptomatic cases may not be detected if they do not seek medical attention.[36] Nishiura and co-workers estimated asymptomatic ratio amongst 565 Japanese evacuees was 30.8% (95%CI:7.7%- 53.8%)[36,148] This approximates the percentage of asymptomatic case ratio (33.3%) reported from a study done in South Korea.[149]

Studies have shown that asymptomatic infections are more common in populations of young and middle-aged individuals with functional performance status without underlying diseases and comorbidities.[29] Asymptomatic cases have the same infectivity as symptomatic COVID-19 cases.[29, 151] Asymptomatic cases may play a key role in the transmission and therefore pose a significant challenge to infection control. It is also reported in the literature that the incidence of asymptomatic infections in children is lower than that of the whole population and might be related to the immune response and ACE2 levels in children.[29]

Transmission of SARS-CoV-2 from infected but still asymptomatic individuals has been increasingly reported.[34,38,150] Asymptomatic carriers during the incubation period can be a potential infection source of COVID-19.[34,38] Infection transmission by asymptomatic patients can make infection control and prevention very challenging. Viral loads peak within the first few days of symptoms, but asymptomatic patients can have a similarly high viral load.[43]

Early recognition of an infected person and cutting off the route of transmission is critical to controlling COVID-19. In addition most asymptomatic cases do not seek medical care which contributes to rapid spread of COVID-19.[29]

• ***Co-morbidities and increased risk of COVID-19 severity***

Individuals who are at higher risk of severe illness include people older than 65 years, people at any age that have severe medical conditions, including asthma, cardiovascular conditions, hypertension, haemoglobin disorders, liver disease, severe obesity, people in nursing homes and long-term care facilities[152] and individuals with immune compromised conditions such as diabetics, HIV and TB.[153] The most prevalent co-morbidities associated with COVID-19 are: hypertension [21% (95%CI: 13.0%-27.2%)], diabetes [9.7% (95%CI: 7.2%-12.2%)], cardiovascular disease [8.4% (95%CI: 3.8%-13.8%)], and respiratory disease [1.5% (95%CI: 0.9% - 2.1%)] [137] The major finding that hypertension is a host factor for severe COVID-19 may underscore the involvement of the renin-angiotensin system (RAS) in the pathogenesis of COVID-19.[154] Other co-morbidities associated with COVID-19 severity included malignancy (1%), chronic liver diseases (4.5%) and chronic renal disease (1.4%)[154] It is also suggested that patients with cardiac diseases, hypertension or diabetes, who are treated with ACE2-increasing drugs, are at higher risk for severe COVID-19 infection.[129,155] It is now also suggested that periodontitis may be linked to COVID-19 severity.[142,156,157]

Individuals with comorbidities presented with increased COVID-19 severity and higher case fatality rates compared to those individuals without comorbidities.[30,136]

Conclusion

The disturbing reality is that we have no idea who among us is spreading the disease. This extreme evasiveness of SARS-CoV-2 makes it harder to control.

Understanding the characteristics of the infection chain pathway is critical in the adoption of appropriate infection prevention and control strategies in the dental practice setting. Breathing, talking, sneezing, coughing and aerosol generating procedures are all implicated in the generation, expulsion, evolution, and transmission of virus-laden droplets and aerosols.

The infection chain can be blocked at various levels by applying infection control and prevention strategies, thus mitigating the risk of spreading infection. An effective risk mitigation strategy for dental practices has to be based on a combined approach of breaking the links of the infection chain and should include (i) screening and isolation of high risk patients as well as oral health care workers to reduce the risk of exposure, (ii) universal masking and hand sanitation remains the basic foundation of infection disease prevention and control strategy, (iii) pre-procedural mouth rinse to reduce the oral and naso-pharyngeal viral load remains an important but neglected strategy, (iv) use of appropriate personal protective equipment, (v) use of rubber dam and high volume suction (evacuation) to reduce exposure to contaminated aerosols and respiratory droplets and splatter, (vi) cleaning and surface disinfection, (vii) ventilation and airborne disinfection (HEPA- filters and UV lights, foggers), (viii) immune boosting, designer antibodies to neutralize the viral spike protein and use of a vaccine.

The current understanding and available evidence-based knowledge of the how and why of these infection prevention and control measures in the dental practice clinical setting will be discussed in Part 3 of the series.

Fundamental questions that remain unanswered include: (i) How does SARS-CoV-2 primarily spread in a dental clinical setting?; (ii) What is the viral titre in the respiratory fluid and the emitted aerosol particles during breathing, speech, coughing and sneezing and AGP (iii) What is the SARS-CoV-2 viral load in the saliva and pharyngeal mucus of asymptomatic and symptomatic salivary samples?; (iv) What is the infectious dose and length of exposure that will give an individual a significant chance of being infected? (v) What percentage of patients are asymptomatic and how do their infectiousness compare to those of symptomatic patients?; (vi) Who are the infectors and how does an infected individual's age and co-morbidities affect the risk of transmitting infection to others?; (vii) Is viable SARS-CoV-2 present in air samples in dental rooms where non-aerosol and aerosol generating procedures are performed? (ix) How effective are fogging devices at disinfecting airborne virus particles?

SARS-CoV-2 transmission from asymptomatic and pre-symptomatic hosts makes it more critical than ever that methods of rapid diagnosis are developed that provide better and faster prediction of COVID-19 infection and infectiousness. One of our greatest challenges globally is prophylactic prevention and control of transmission of SARS-CoV-2 from asymptomatic patients.

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ADVERTORIAL

Colgate laboratory tests show toothpaste and mouthwash neutralize 99.9% of the virus that causes COVID-19

Clinical research program underway to validate potential of oral health products to slow spread of the virus

Laboratory studies show that toothpastes containing zinc or stannous and mouthwash formulas with cetylpyridinium chloride (CPC) neutralize the virus that causes COVID-19 by 99.9 percent.The studies are part of a Colgate research program that includes clinical studies among infected people to assess the efficacy of oral care products in reducing the amount of the virus in the mouth, potentially slowing the transmission of the COVID-19 virus.

In the laboratory studies – the first to include toothpaste – Colgate Total and Meridol toothpastes neutralized 99.9% of the virus after two minutes of contact. Colgate Plax and Colgate Total mouthwashes were similarly effective after 30 seconds. The studies, completed in October, were conducted in partnership with Rutgers New Jersey Medical School's (NJMS) Public Health Research Institute and Regional Biosafety Laboratories.

The results suggest that some toothpastes and mouthwashes may help reduce the spread of SARS-CoV-2, the virus that causes COVID-19, by temporarily reducing the amount of virus in the mouth. The virus spreads through respiratory droplets or small particles produced when an infected person coughs, sneezes, sings, talks, or breathes, according to the U.S. Centers for Disease Control and Prevention.

"We're at the early stages of our clinical investigations, but our preliminary laboratory and clinical results are very promising," said Dr. Maria Ryan, Colgate's Chief Clinical Officer. "While brushing and rinsing are not a treatment or a way to fully protect an individual from infection, they may help to reduce transmission and slow the spread of the virus, supplementing the benefit we get from wearing masks, social distancing and frequent hand washing."

Said Dr. David Alland, Chief of Infectious Diseases and Director of the Center for COVID-19 Response and Pandemic Preparedness, who led the Rutgers NJMS study along with colleagues Drs. Pradeep Kumar and Riccardo Russo: " Given that saliva can contain amounts of virus that are comparable to that found in the nose and throat, it seems likely that SARS-CoV-2 virus originating in the mouth contributes to disease transmission, especially in persons with asymptomatic COVID-19, who are not coughing. T his suggests that reducing virus in the mouth could help prevent transmission during the time that oral care products are active. "

Concurrent to the laboratory study, Colgate sponsored a clinical study involving some 50 hospitalized subjects with COVID-19. This study demonstrated the ability of Colgate Total (with CPC and zinc), Colgate Peroxyl, and Colgate PerioGard mouthwashes to substantially reduce the amount of the virus in the mouth temporarily. The researchers plan to share their findings in December. Additional Colgate-supported clinical research studies on toothpaste and mouthwashes are in early stages at Rutgers, the Albert Einstein Institute in Sao Paulo, Brazil, and at the University of North Carolina at Chapel Hill Adams School of Dentistry, with some 260 people with COVID-19 participating in these studies.

"Colgate is collaborating with numerous investigators throughout the globe to conduct clinical research to explore the potential of oral care products to reduce oral viral loads as a risk reduction strategy," Dr. Ryan said. "We think oral care has a role to play in fighting the global pandemic, alongside other preventive measures."

Said Dr. Mark Wolff, Morton Amsterdam Dean of Penn Dental Medicine at the University of Pennsylvania: "With this pandemic, the more we understand about the virus, the more effective we can be in fighting it, so I am excited to see the impressive research program Colgate has undertaken. We need to continue to take the precautions recommended by health authorities, and with these studies we may demonstrate an additional way to address the transmission of disease among people in close contact, particularly in dental practice. That would be an important advance."

As the world's #1 trusted dental expert, Colgate is committed to leading in science and to ensuring that its products address health challenges and meet consumers' needs. For more information about the effects of oral hygiene on overall health and additional insights on mask mouth and other topics, **visit www.colgate.com.**

The company behind 9 million smiles and counting

Align Technology is a global, listed medical device company that is changing lives through better smiles. We reimagine and reinvent the way orthodontic and restorative treatment is presented and delivered to millions of people around the world.

We believe a better smile has the power to create a better future, so we create digital technology and experiences to help people move forward in life.

Our history

Align Technology has always set the bar for leadership and change in our industry by constantly innovating to bring new and better solutions to doctors and their patients.

In 1997, five employees in a small duplex in Redwood City, California, founded Align Technology with a simple concept in mind - how to leverage technology to straighten teeth. Just a few years later in 1999, Align Technology pioneered the invisible orthodontics market with the introduction of the Invisalign system and soon launched a large U.S national advertising campaign. By 2001, Align had manufactured one million unique clear aligners, helped treat hundreds of patients and trained over ten thousand doctors.

By combining digital treatment planning and mass-customization, with shape-engineering based on biomechanical principles, we have revolutionized the orthodontic industry. The Invisalign system has continued to evolve over the last 23 years. Building upon our experience, a data base of over 9.0 million cases, continuous innovations in aligner materials, software algorithms, 3D force systems, the Invisalign system can be used to treat a wide range of cases, from teens to adults.

In 2011, we acquired the iTero intraoral scanner. Digital scanning technology was a natural extension to our digital treatment processes of the Invisalign system, enabling enhanced patient scans and an improved patient experience. With more than 6.0 million restorative crown, bridge, and custom implant cases enabled by the iTero scanner to date, Align Technology continues to increase its investment in digital restorative workflows. Today's practices require a range of digitally enabled solutions – from chairside visualization, prosthetic CAD capabilities, in-office milling options, and laboratory workflows for restorative dentistry, to tools and applications exclusively designed for Invisalign® treatment, which only iTero Element can provide.

Today, we've helped treat over 9.0 million patients with the Invisalign system and are driving the evolution in digital dentistry with the iTero intraoral scanner – helping to modernize today's practices by enabling enhanced digital orthodontic and restorative workflows to help improve patient outcomes and practice efficiencies.

The Invisalign system

The Invisalign system is the most advanced clear aligner system in the world. Backed by two decades of innovation, the Invisalign system has helped bring smiles to over 9.0 million people including over 2.3 million teens.

SmartTrack material is Align Technology's own patented aligner material that provides gentle, more constant forces to improve control of tooth movements. After testing 260 different materials, our material on average enables faster treatment.

SmartStaging technology. Programming each tooth movement in a certain sequence, at the right time to achieve optimal outcomes and greater predictability.

SmartForce features. Attachments and features designed into the aligners deliver the precise forces when needed to achieve more predictable tooth movements. The Invisalign system is the only system in the world with features such as Bite Ramps, Precision Cuts, Power Ridge to help address a wide range of clinical situations.

invisalign

invisalign
9M smiles

The Invisalign system is the most advanced clear aligner system in the world, trusted by more than 9.0 million people worldwide to improve their smiles.

Align Technology is dedicated to transforming lives by improving the journey to a healthy, beautiful smile. Our goal is to become an indispensable partner to dental professionals worldwide while building a great company.

iTero®

The iTero Element Family of Intraoral Scanners propels today's dental practice into the future by enhancing the patient experience and elevating clinical precision.

iTero intraoral scanners

iTero intraoral scanners are designed to deliver speed, reliability, intuitive operations, and outstanding visualization capabilities for general practitioners or orthodontists. iTero scans have been used in more than 6.0 million restorative crowns, bridges, and custom implant abutment cases and more than 27.4 million iTero orthodontic scans, for a total of 33.4 million scans.

The iTero Element intraoral scanner propels today's dental practice into the future by enhancing the patient experience and elevating clinical precision. Its parallel confocal imaging technology uses optical and laser scanning to achieve accurate scans in color. iTero Element empowers practices in both orthodontic and restorative dentistry with its innovative technology. With over 27.4 million orthodontic and 6.0 million restorative scans worldwide, iTero scanners have a proven track record of success. iTero Element scans are engineered to work with the Invisalign® System, the exclusive Invisalign® Outcome Simulator and Progress Assessment tool.

Our vision is to bring clear aligner orthodontic treatment to the masses.

A better future with a better smile

Aesthetic two stage crown lengthening for altered passive eruption: A 25-year case report and review

André W van Zyl[1] and Inus Snyman[2]

Introduction

Mucogingival abnormalities may involve lack of gingiva, excess gingiva, recession, shallow vestibule, abnormal colour and aberrant frenula (AAP Consensus 1999).[1,2] Periodontal plastic surgery was a term introduced by Preston Miller in the early nineties to describe mucogingival or periodontal procedures dealing with aesthetics.[3] It is of the utmost importance to understand that none of the conditions or procedures described in this article can be correctly diagnosed or treated until such time as all infection and inflammation have been resolved. There should be no gingivitis or periodontitis present.

Periodontists are often requested to correct an excessive gingival display or correct excessive exposure of teeth due to recession. Periodontal aesthetic surgery around natural teeth therefore involves either the removal of excess tissue in gummy smiles or the repair of lost tissue such as in recession. The former involves the procedure of crown lengthening or gingivectomy and the latter the treatment of marginal gingival recession by grafting. Crown lengthening is carried out either for aesthetic or functional purposes. Indications for crown lengthening include subgingival caries, crown or root fractures, altered passive eruption, short teeth, excessive gingival display, uneven gingival contours, cervical root resorption and short clinical abutment.[4] This article will cover excessive gingival display (Altered Passive Eruption, APE) and the procedure of two stage crown lengthening only.

Crown lengthening (CL) may be achieved by either gingivectomy (removal of excess gingiva) or by removal of bone (ostectomy) and gingiva. It is essential to determine whether bone needs to be removed for lengthening. In the past this was done by doing bone sounding under local anaesthesia, which is a fairly invasive procedure. CBCT is now an alternative and not only can bone be assessed by this, but the soft tissue too, provided a soft tissue CBCT approach is used.[5]

CL is aimed at exposing more of the clinical crown of the tooth. There are various reasons why this may be desirable but for the purpose of this article we will focus on two of the main aesthetic reasons caused by APE:

1. A gummy smile where the teeth are not fully exposed and partly covered by gingiva (Figure 1)
2. An asymmetric gingival contour which is not aesthetic (Figure 2)

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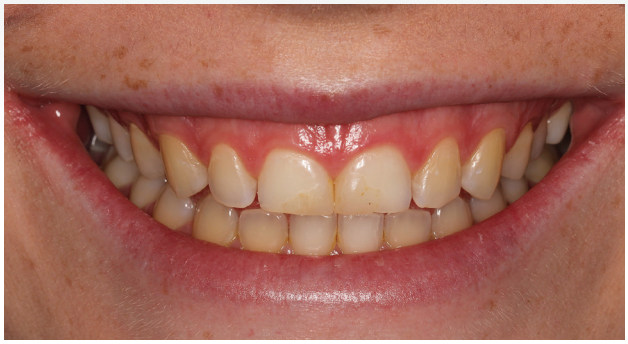

Figure 1: Gummy smile due to Altered Passive Eruption.

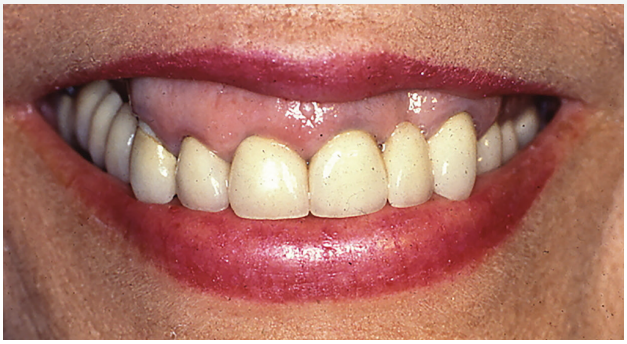

Figure 2: Asymmetric gingival contour.

It is important to be able to diagnose the underlying problem correctly before embarking on the procedure.

Altered Passive Eruption (APE)

It is important to note that excessive gingival display is not always due to APE, but may also be due to vertical maxillary excess, sometimes in combination with a high lip line (Figure 3).[6,7]

In a South African study it was found that Delayed Passive Eruption had a prevalence of 12%.[8] Excessive gingival display is most often diagnosed as APE, but it may also be seen in drug induced enlargement and in plaque induced swelling.[6] Passive eruption is the process that occurs after the tooth erupts into the oral cavity (the active eruption phase) and is the process where the gingiva slowly migrates apically to expose the anatomical crown.[6] APE occurs when the gingiva does not reach its correct position and covers part of the clinical crown, giving a gummy appearance.[6] It is not clear exactly when the physiological movement of passive eruption ends and thus, at what age a diagnosis of APE can be made.[6] Coslett et al. reported that by the age of 18 -20 years, the majority of individuals have a mature dentogingival relationship.[9]

Multiple factors have been proposed as possible causes for APE. These include occlusal interference by soft tissue during the eruption phase, the presence of thick fibrotic gingiva, genetics, the presence of thick bone, orthodontic trauma and endocrine conditions.[10]

APE can be classified into two types, based on the position of the mucogingival junction in relation to the cemento-enamel junction.[9] Type 1 APE is characterised by the mucogingival junction being apical to the alveolar bone crest, usually with a wide band of attached gingiva.[6] This band of attached gingiva is usually wider that the generally

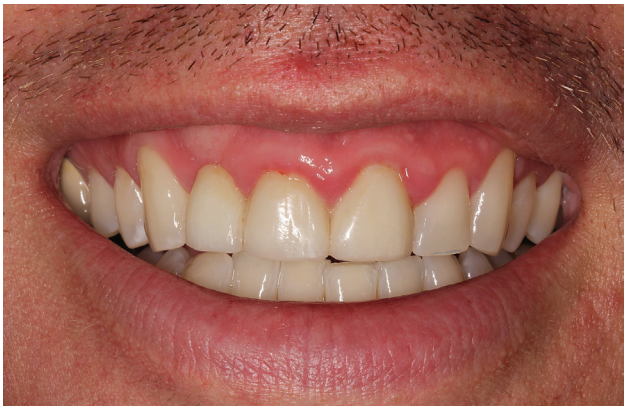

Figure 3: A gummy smile due to vertical maxillary excess where teeth are almost fully exposed.

accepted mean width of 3,0 - 4,2 mm in the maxilla and 2,5 - 2,6 mm in the mandible.[6] In contrast, Type 2 APE is defined by the presence of a band of attached gingiva which falls within the normal mean width.[6] In type 2 APE, the mucogingival junction is located at the level of the cemento-enamel junction, with the whole band of attached gingiva located on the anatomic crown.[6] Both type 1 and type 2 APE, can further be subclassified into subgroup A or subgroup B.[6] In subgroup A, the alveolar bone crest is located at the normal position, 1 – 3 mm apical to the cemento-enamel junction.[6] Subgroup B refers to those cases where the alveolar bone crest is located at or coronal to the level of the cemento-enamel junction.[6] Correct classification and diagnosis of each case is of critical importance before treatment commences. APE type 1 subgroup A may be treated with gingivectomy alone, whereas the authors recommend a two-stage crown lengthening approach for all other classifications.[6] Whether the second stage surgery (gingivectomy) in a two-stage crown lengthening approach

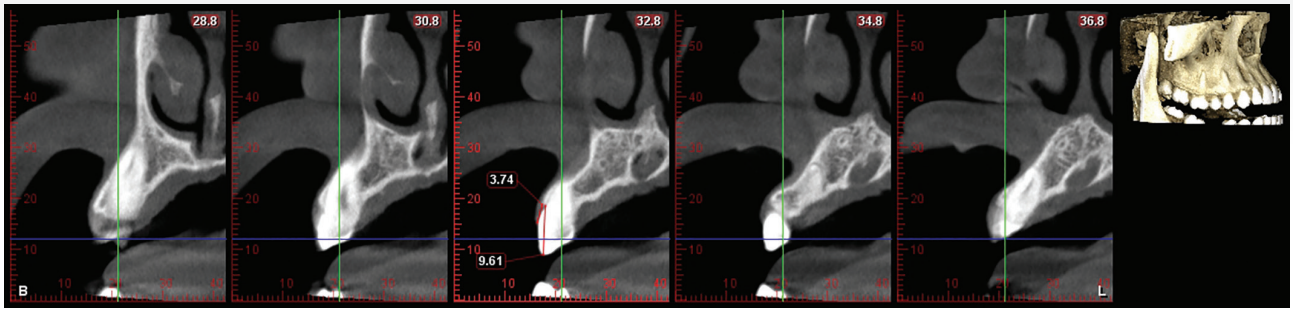

Figure 4: Soft tissue retraction allows for measurement of supra-crestal gingival dimensions.

is required, will be determined by the outcome after healing following the first stage surgery. It is the author's experience that a second stage gingivectomy is often not required due to adequate recession after ostectomy. All patients are given the choice of the second stage and very few opt to have a second stage. Should the planning however involve crowning of the anterior maxillary teeth, it is for the restorative clinician to decided whether optimal lengthening has been reached.

Supracrestal attached tissues (biologic width) and dento-gingival complex

The term biologic width was recently replaced with the term supracrestal attached tissues.[2,11] The physiological function of the supracrestal attached tissues is that of a protective barrier for the periodontal ligament and supporting alveolar bone.[12] The supracrestal attached tissues include the junctional epithelium and connective tissue attachment, the average dimensions which were measured to be 0,97 mm and 1,07 mm respectively, yielding an average dimension of 2.04 mm for the supracrestal attached tissues.[13] A more recent systematic review found similar mean values of the supracrestal attached tissues, reported as 2.15 mm - 2.30 mm.[12] It is however extremely difficult, if not impossible, to clinically measure the dimension of the supracrestal attached tissues accurately. For this reason, we should rather rely on the dimension of the dento-gingival complex, which can be measured clinically or by soft tissue CBCT (Figure 4). The dento-gingival complex includes the sulcus depth, in addition to the junctional epithelium and connective tissue attachment. A study examining the supraosseous gingiva dimension (dento-gingival complex), found the mean dimension of the maxillary facial dento-gingival complex to rage between 3,71 ± 0.51 mm and 4,03 ± 0,41 mm.[14]

Disagreement still exists among authors with regards to the amount of ostectomy needed during crown lengthening procedures.[6] The suggested distance between bone crest and cemento-enamel junction range between 1 - 3 mm and the suggested distance from bone crest to planned gingival margin is ≥ 3 mm.[7,15-23] Therefore, it is reasonable to perform presurgical measurement of the dento-gingival complex in each patient, to determine the extent of bone removal during a crown lengthening procedure.

It has been shown that significant alterations can occur in the marginal periodontal tissue level from the day of surgery up to 12-months following healing.[24] The greatest changes occur during the first 3 months after surgery.[25] The coronal shift of the soft tissue margin during healing, also referred to as soft tissue rebound, is more pronounced in thick periodontal phenotypes, compared to thin phenotypes.[24] For this reason, planning the extent of ostectomy should also take into consideration the patient's periodontal phenotype.[4] The term periodontal biotype was recently replaced with the term periodontal phenotype.[2] Periodontal phenotype describes both the gingival phenotype (gingival thickness and keratinized tissue width) and the thickness of the buccal bone plate.[11] Biotype refers to a group of organs which have the same genotype, whereas phenotype refers to the appearance of an organ based on a multifactorial combination of genetic traits and environmental factors.[11] The phenotype, unlike genotype, can change over time or can be modified by means of clinical intervention.[11]

To further complicate treatment planning, the mere action of elevating a full thickness flap during crown lengthening, may cause marginal bone loss. Two clinical studies reported a mean crestal bone loss of 0,6 mm and 0,47 mm respectively, after full thickness flap elevation.[26,27] If the surgeon did not plan for this additional bone loss, treatment may lead to unsatisfactory results such as exposed root surfaces or crown margins. It is thus clear that meticulous treatment planning should be performed before treatment commences.

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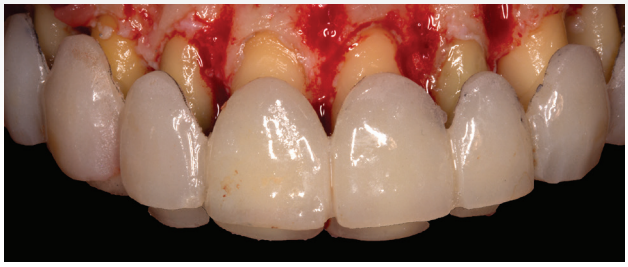

Figure 5: Surgical stent used to assess ostectomy levels.

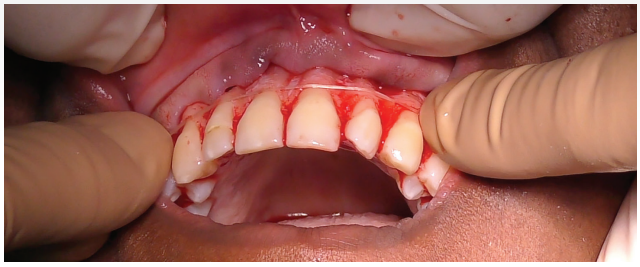

Figure 6: Floss can be cold sterilized and used intra-operative to assess symmetry in bone levels.

Two Stage Crown lengthening

The technique of performing a CL in two stages, with ostectomy and osteoplasty done in stage 1 and a gingivectomy, if indicated, in stage 2, is a predictable procedure with a low trauma impact to the patient. The alveolar bone is removed and shaped in the first procedure without any soft tissue removal and after a few months of healing, a second procedure of gingivectomy may be needed if there has not been sufficient gingival recession.

The classic procedure of crown lengthening is a single procedure, involving a simultaneous soft tissue contouring (excision) and bone removal (ostectomy). David Garber introduced the two stage crown lengthening in the early 1990's, describing a procedure where the bone contouring is done in the first procedure and the gingival contouring in a subsequent procedure after a suitable period of healing.[28] Removing gingiva (and bone) in one procedure in a perfect aesthetic symmetry is difficult and will harm the patient by reducing vital attached gingiva needed for long-term stability. In the authors' experience, very few if any patients have enough gingiva to undergo excision during a crown lengthening. It may also prove difficult to achieve a thin feather edge to the marginal gingiva, which gives the most ideal aesthetics, when excising gingival tissue in a one stage procedure.

Bone contouring by itself, is a more controlled slow process, where different diamond burs are used to sculpt the bone and finish it in a thin feather edge- which in turn will induce a thin marginal gingival edge. Achieving perfect symmetry with this process is also easier due to the slow controlled removal of tissue whilst allowing measurements using either a periodontal probe, a surgical stent (Figure 5) and floss to do a quick check intra-operatively (Figure 6).

Before any periodontal aesthetic surgery for excessive gingival display can be planned, a simple decision-making tree can be utilized to determine the diagnosis and following from that the correct procedure can be selected (Figure 7):

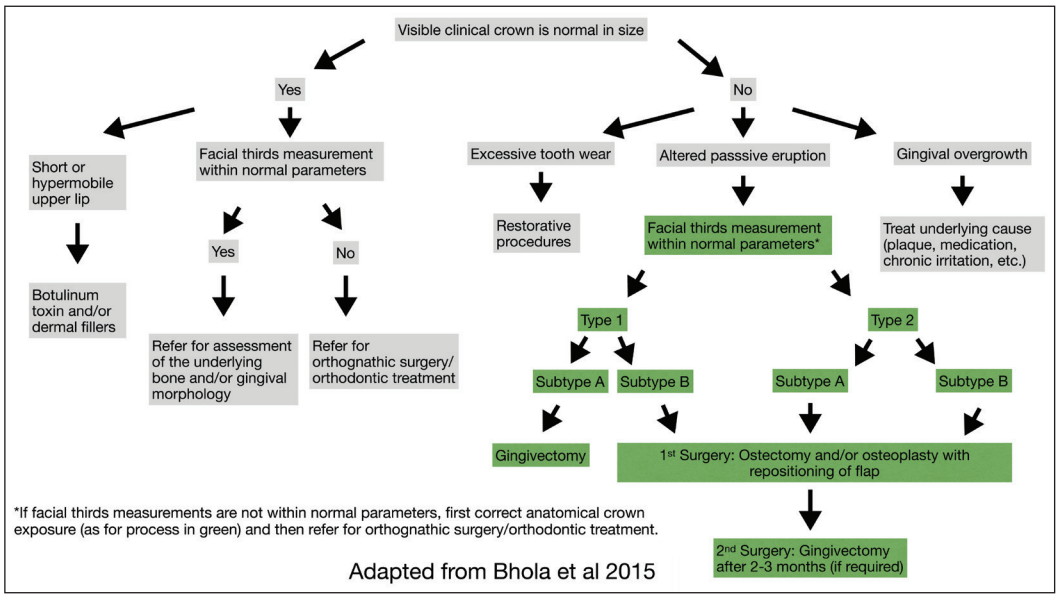

Figure 7: Decision tree for diagnosing excessive gingival display and selecting appropriate treatment.

Tooth measurements

The visible crowns of the anterior six maxillary teeth are important in the smile. Before any crown lengthening can be contemplated, the tooth sizes should be measured and documented in the file. Central incisor teeth are approximately the same size as the canines and in the range of 10-13mm, whereas lateral incisors are slightly smaller and in the range of 9-11mm.[29]

Case report: Two stage crown lengthening - a 25-year follow-up

A 34-year-old patient presented with a gummy smile (Figure 8) which was classified as altered passive eruption type 1B. The patient needed a full rehabilitation of the occlusion due to a deep bite, attrition on the palatal aspects of teeth 13-23 and loss of posterior occlusal stability. The patient had excellent plaque control, no periodontal disease and a non-vital 11 due to trauma a few years prior to consultation.

Radiographic examination revealed no alveolar bone loss and clinical probing depths varied from 1-3 mm.

The anterior maxillary teeth had over-erupted due to the attrition and lack of occlusal stability.

It was decided to perform a two-stage crown lengthening as it required extensive lengthening and the patient's aesthetic expectations were high. After a wax-up, a surgical crown lengthening guide was manufactured to fit over the teeth (Figure 9) to give an indication of what would be required to restore the smile surgically as well as prosthetically. The stent was fitted in the patient's mouth and a black pencil was used to block-out the incisal edges to simulate the final incisal edges and size of the teeth (Figure 10). This allowed a clear estimation of how much lengthening would be required. Photographs were taken and it was decided to do an ostectomy first, followed by a second stage of gingivectomy after 3-4 months.

Following administration of local anaesthesia, the ostectomy and osteoplasty was performed after elevating a full thickness buccal flap with no palatal flap elevation (Figure 11). The surgical guide served as a reference for the planned crown margins, to ensure adequate amount of ostectomy and to prevent future violation of the space to be occupied by the supra-crestal attached tissues. Deep interdental split thickness flap design allowed full access to the interdental bone for contouring. No interdental crestal reduction was done, mainly because it was not indicated

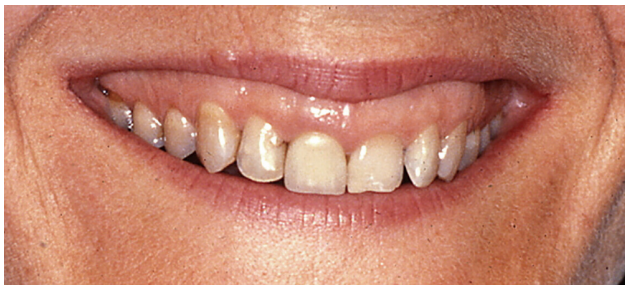

Figure 8: Altered passive eruption with a component of vertical maxillary excess, showing incomplete exposure of clinical crowns.

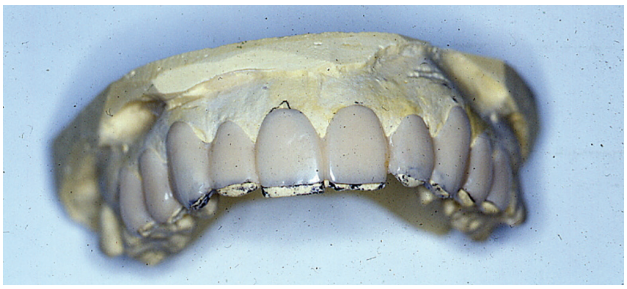

Figure 9: Surgical stent to indicate the new planned clinical crowns.

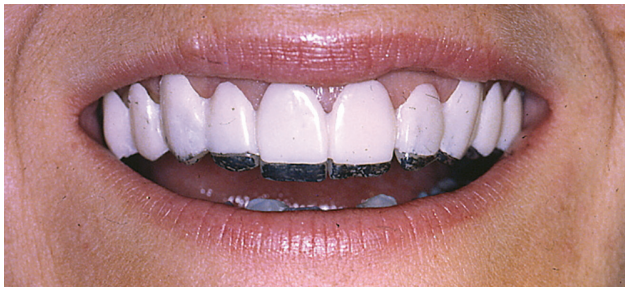

Figure 10: Surgical stent placed intra-orally with incisal edges blocked out using a black pencil.

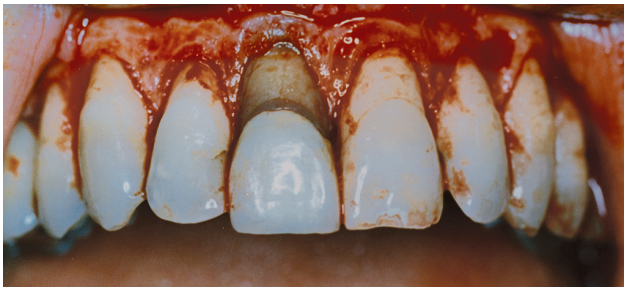

Figure 11: Full flap reflection with partial thickness interdentally, allowing access to buccal and interdental bone for ostectomy and osteoplasty.

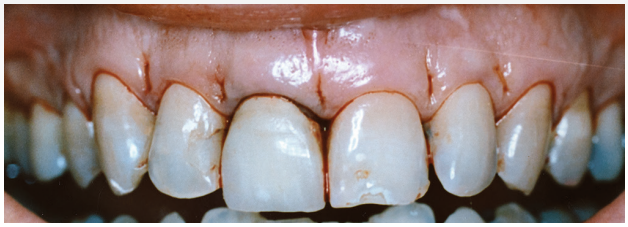

Figure 12: Closure of flap with vertical everting mattress sutures to allow for maximum soft tissue fill in embrasure spaces.

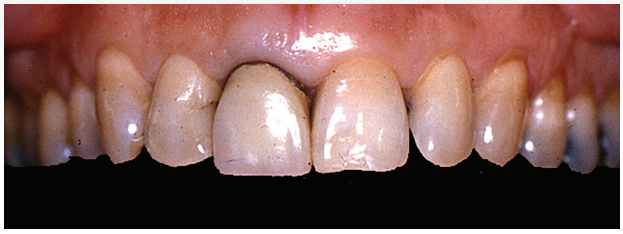

Figure 13: Healing after 3 months showing 2-3mm of recession in a symmetrical pattern, following the bone contour.

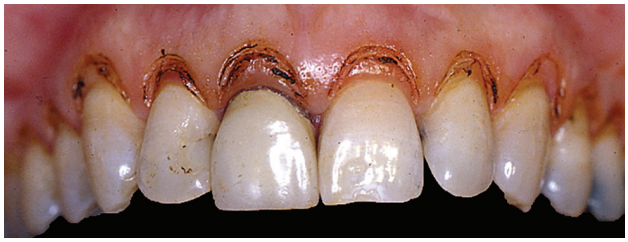

Figure 14: Second stage gingivectomy after 4 months.

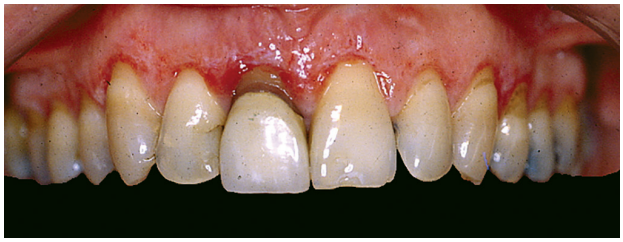

Figure 15: Two weeks after second stage surgery, showing the extent of lengthening.

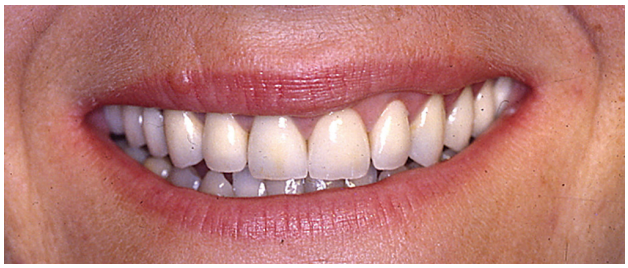

Figure 16: Six months after placing final crowns.

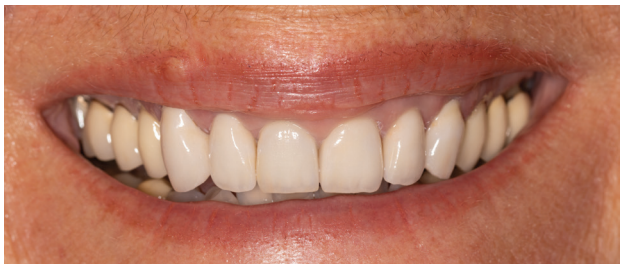

Figure 17: 25 Years after surgery, showing stable gingival margins.

in this case, but also to prevent inadequate papillae fill in the gingival embrasure spaces after healing (Figure 11). Vertical everting mattress 6/0 braided sutures were used to close the flap with maximum embrasure filling with soft tissue (Figure 12). Although monofilament sutures such as nylon have less bacterial contamination potential, softer braided sutures are much more comfortable to the patient. Healing was uneventful and some lengthening was obtained with the recession that took place after ostectomy (Figure 13).

After 4 months, gingivectomy was done with scalpel, cauterization and thinning of the tissue by course diamond drills (Figure 14). This was possible due to the presence of a wide band of attached keratinized mucosa (altered passive eruption type 1). Two weeks later, healing shows extensive lengthening (Figure 15)

VITA In-Ceram® (VITA Zahnfabriek, Germany) all-porcelain crowns were placed after a further 4 months of healing and tissue maturation, with a good aesthetic outcome (Figure 16).

The patient was followed up at regular intervals and after 15 years the anterior 6 crowns (13-23) were replaced due to marginal fractures of the In-Ceram® crowns on the palatal aspects.

The periodontal tissues are stable at 25 years (Figure 17). This case demonstrates a stable long-term result of performing extensive aesthetic crown lengthening utilizing a two-stage surgical protocol with a predictable step-by-step treatment. This allowed full control and minimal loss of attached gingiva, due to at least 2-3 mm of lengthening obtained from the process of recession.

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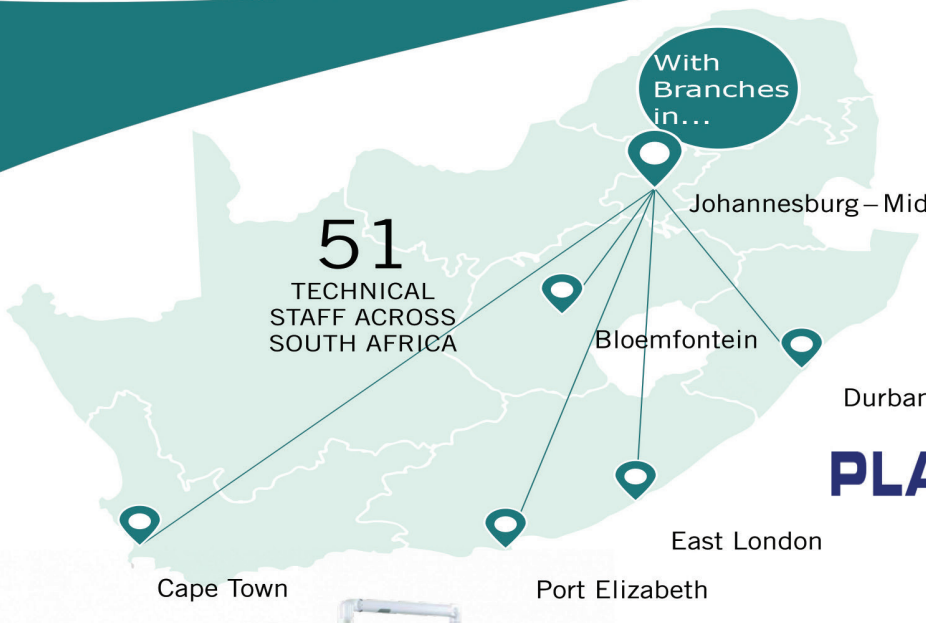

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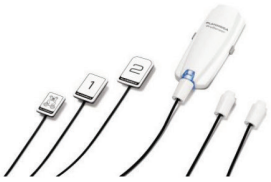

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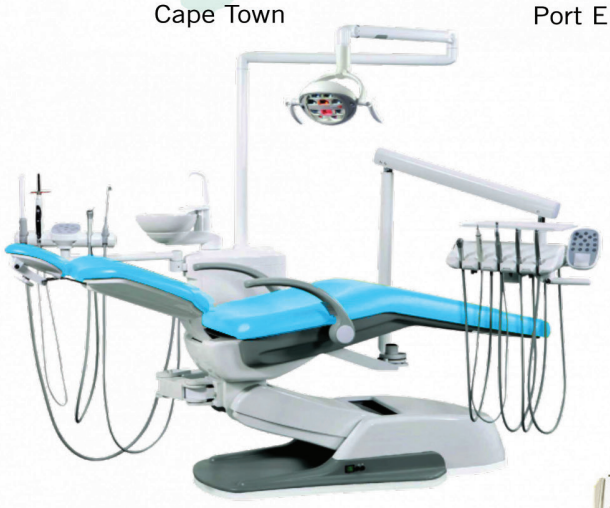

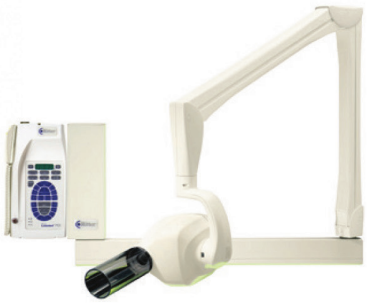

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All-ceramic single-tooth restorations for treating damaged dental enamel: Long-term results in patients with and without amelogenesis imperfecta

Andrea Klink, Fabian Hüttig and Martin Groten

Introduction

Teeth that have been affected by extensive wear or a genetic disorder can be successfully restored with adhesively bonded all-ceramic restorations. The following article summarizes the outcomes of a series of complex clinical cases. Two different groups of patients require the rehabilitation of impaired full dentitions:

1. Patients whose teeth show extensive erosion, abrasion or attrition caused by their diet (e.g. consumption of energy drinks) and/or overuse (e.g. grinding)
2. Patients who have a genetic disorder that affects the tooth structure and the composition of the tooth enamel (e.g. amelogenesis imperfecta, AI) (Figures 1 to 3)

Patients in the first group usually start experiencing problems (e.g. pain and compromised esthetics) in their forties and fifties. The teeth become shorter and look yellowish; parts of the existing tooth structure become fragile and a loss of vertical dimension occurs. Patients with a genetic enamel defect generally require treatment when they are in their teens. Today's advanced all-ceramic and adhesive systems allow minimally invasive tooth preparation and they produce durable, functional and esthetic restorations.

Before treating patients with dental erosion, it is important to obtain their full medical history (e.g. bulimia nervosa or reflux disease) and find out about their eating habits. If possible their general practitioner should also be involved in the treatment. If the patient is experiencing functional problems, these should be addressed in a pre-treatment phase. In patients with a congenital defect of the tooth structure, a thorough clinical and radiological examination is of utmost importance, since in addition to the enamel defects (Figures 4 and 5), these individuals may also suffer from follicular cysts, abnormal tooth eruption, retained or impacted teeth, an open bite or dental pulp calcification (Figures 6 to 8). The disorder is furthermore associated with gingival and periodontal disease. This must be taken into consideration in the preliminary treatment of hypomineralized and hypocalcified enamel. Depending on the severity of the impairment of the enamel formation, an adhesive bond may be much weaker than it would be on healthy enamel. As a result, the adhesive bond must be generated in the dentin in most cases (Figure. 9).

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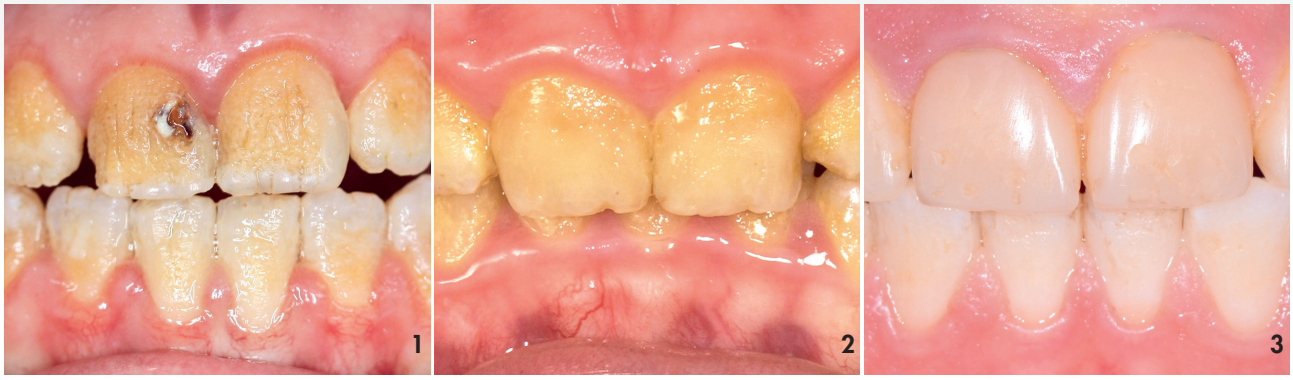
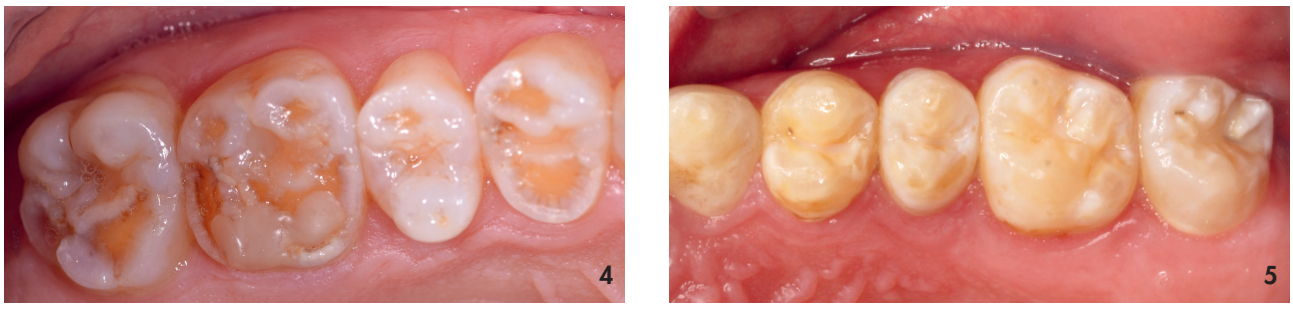


Figures 1 - 3: Three phenotypes of the autosomal dominant inherited amelogenesis imperfecta in the anterior teeth of three sisters



Figures 4 & 5: The posterior teeth of AI patients also show different types of enamel defects. The enamel layer may be missing entirely.

At present, no guidelines or scientific reports of an evidence-based protocol are available for the treatment of patients with AI. In a long-term study conducted at the Tübingen University Hospital, we identified the type of complications which could arise with adhesively placed all-ceramic single-tooth restorations in the mentioned patient groups.

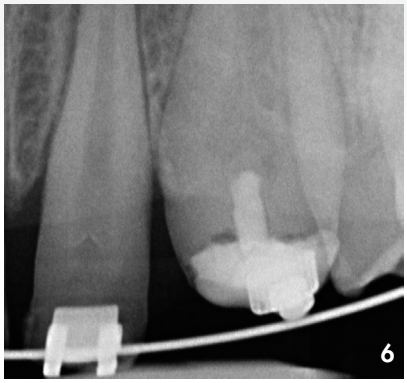
Data base and examinations

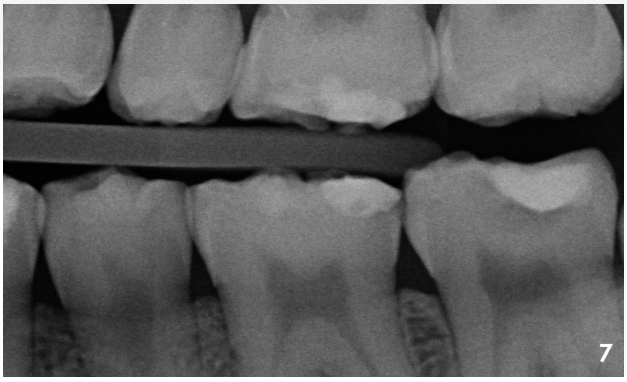
For our study, we selected patients who regularly attended the recall appointments and who had been treated as follows:

Single-tooth restorations (crowns, partial crowns) made of silicate (Si) or lithium disilicate ceramic (LiDi) had been placed with an adhesive composite. Table 1 contains a list

Table 1 — Overview of materials used

Group	Name	Manufacturer	Other characteristics
Silicate ceramic	Celay (feldspathic ceramic) IPS Empress 1	Vita Zahnfabrik, Bad Säckingen, Germany Ivoclar Vivadent, Schaan, Liechtenstein	Copy milling, predecessor product of Vitablocs Mark II Leucite-reinforced silicate ceramic
Lithium disilicate ceramic	IPS Empress 2 IPS e.max Press	Ivoclar Vivadent, Schaan, Liechtenstein	Lithium disilicate ceramic of the 1st generation Lithium disilicate ceramic of the 2nd generation: Only the vestibular aspect of anterior restorations was veneered (IPS e.max Ceram – Ivoclar Vivadent): cut-back technique
Veneered zirconium oxide frameworks	Ceramill Zolid Vitablocs YZ Cercon	Amann Girrbach, Pforzheim, Germany Vita Zahnfabrik, Bad Säckingen, Germany DeguDent, Hanau, Germany	Zirconium oxide frameworks veneered with IPS e.max Ceram (Ivoclar Vivadent) Zirconium oxide frameworks veneered with Cercon Ceram Kiss (Degudent) Zirconium oxide frameworks veneered with Cercon Ceram Kiss (Degudent)





Figures 6 & 7: The entire crown of tooth 23 is missing (Fig. 6). The bitewing radiograph (Fig. 7) shows normally formed dentin: parts of it are bare, while other parts are covered with only a thin layer of enamel.

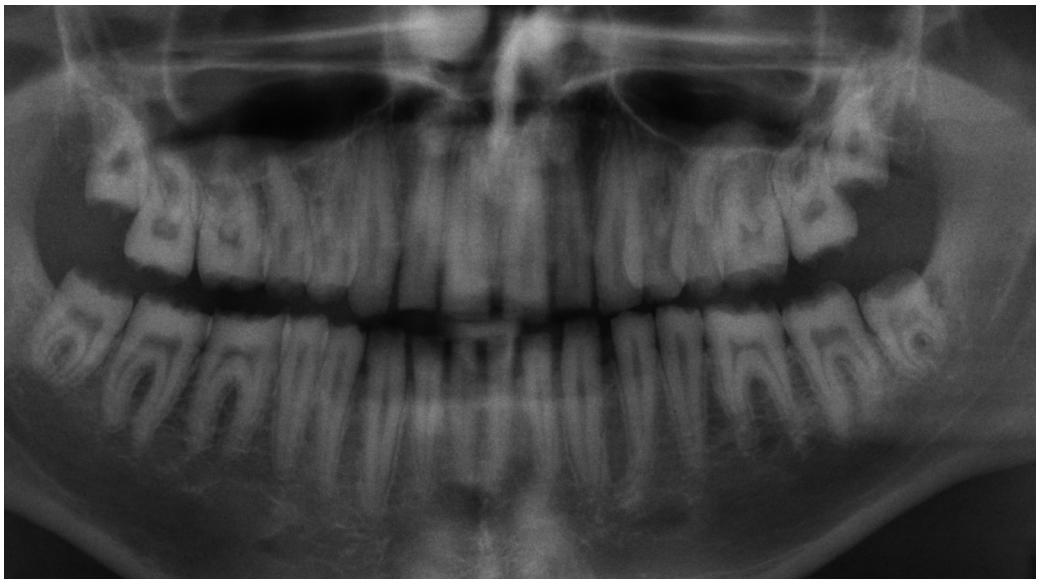


Figure 8: The panoramic radiograph shows a severe form of AI. None of the teeth show any enamel coverage.

of the materials used.

The patients had lost a maximum of four teeth. The missing teeth were replaced with not more than a three-unit all-ceramic bridge or a single-tooth implant with an all-ceramic crown. The patients whose vertical dimension of occlusal had to be opened by more than 4 mm were given an occlusal appliance which they had to wear 24/7 for at least four months before the treatment. The rapid developments in the field of all-ceramic materials continue to open up new treatment modalities. Initially, however, feldspathic and leucite-reinforced silicate ceramics were copy-milled to produce esthetic single crowns, which could be used in posterior teeth and were placed with the adhesive technique.

A clinical check-up program was initiated in order to monitor the quality of the treatment results. Therefore, the tooth status, periodontal probing depth and papillary bleeding index as well as the quality of all the restorations were annually assessed and classified according to the Ryge criteria. Irreparable fractures, tooth loss and deep probing depths were classified as "absolute failures". If the quality of a restoration was deemed to be compromised, but a new restoration was not justified, it was classified as a "relative failure" (e.g. chipping, cracking of the ceramic).

Patients, observations and results

The study involved 17 patients between the ages of 12 and 69 (at the time of the restoration placement) and a total of

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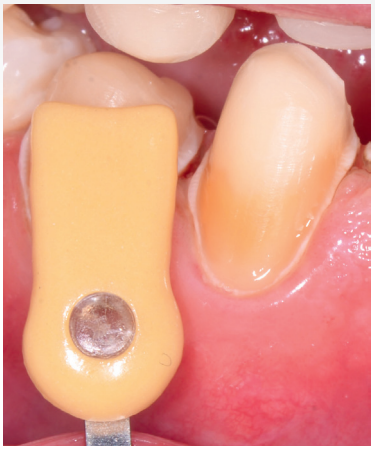
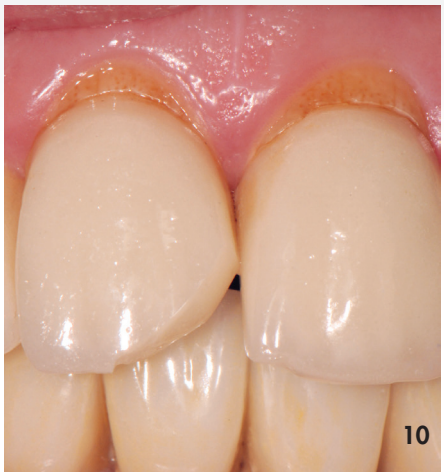


Figure 9: In many cases, the dentin colour deviates considerably from the norm. It is therefore important to select the correct tooth shade for an esthetic outcome of the monolithic crowns.



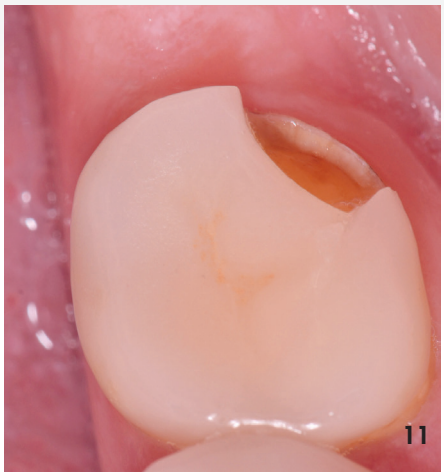


Figure 10: Grade 3 chipping of an IPS Empress II anterior crown after 16 years. The restoration was repaired by adhesively bonding the fragment in place again.
Figure 11: Classical fracture of a molar crown (in this case Celay silicate ceramic, Vita Zahnfabrik) after 14 years. The crown was completely removed and a new restoration was placed after minimal preparation.

450 restorations (Table 1 – QR code). Nine of the patients suffered from some form of AI. The patients were observed over a period of maximum 17 years. During this time, the following complications occurred in 44 restorations (10 %) in 11 patients (65 %):

- Eleven of the 44 restorations were classified as "absolute failures", which translates to an overall survival rate of 99.8 % after three years and 91.4 % after ten years. No significant differences were noted between patients with or without AI.
- Thirty-three of the "relative failures" were caused by chipping (25). It is interesting to note that 11 of the "chip-offs" were recorded in one patient alone. Despite the observed complications, the success rate was 95.7 % after three years and 81.4 % after ten years. A statistically relevant difference was noted between patients with and without AI. The success rate of patients with AI was significantly higher.

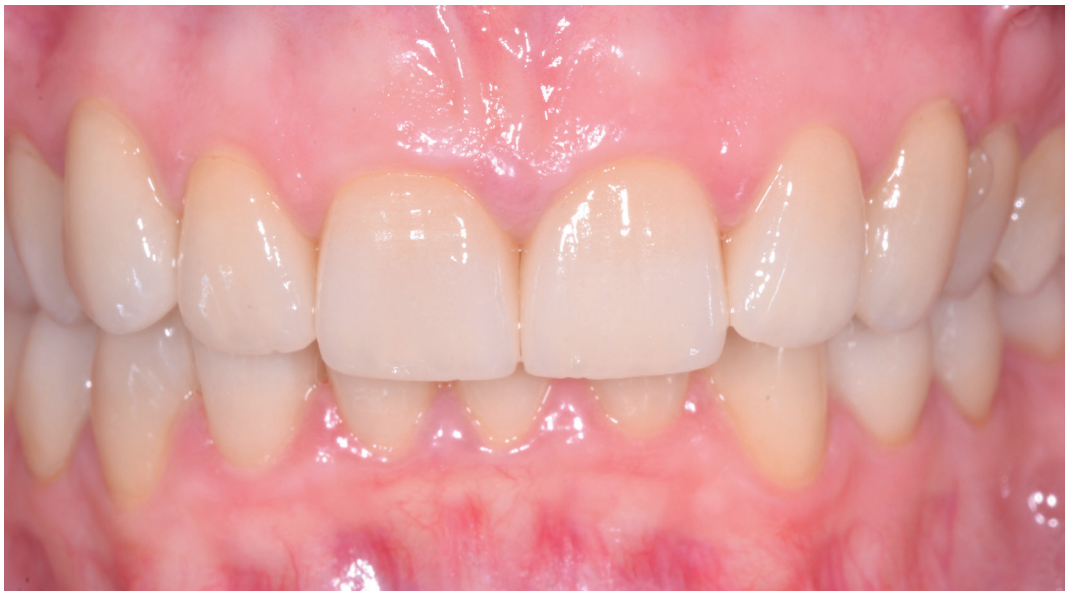


Figure 12: Photo of the restorations placed in the patient shown in Figure 2 after five years of service.

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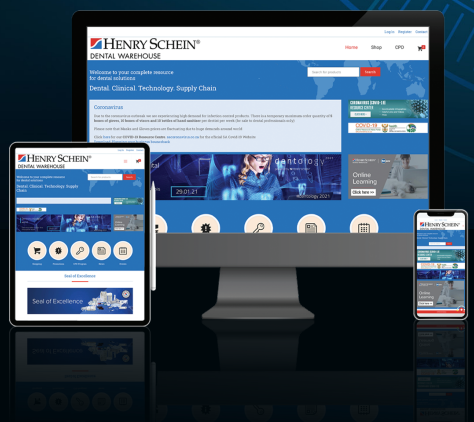

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Table 2 — Overview of all restorations placed, their observation period and material groups, assigned to the respective patient.

Pat#(AI?)	Age at the time of treatment	Observation period (months)	Crowns Lithium disilicate ceramic	Silicate ceramics	Partial crowns Lithium disilicate ceramic	Bridges	Total number of restorations
1 (AI)	12-13	171	6	22	0	0	28
2	41	182	0	27	0	0	27
3	60	88	0	22	0	1	23
4 (AI)	17	101	28	0	0	0	28
5 (AI)	39	98	28	0	0	0	28
6	31	26	28	0	0	0	28
7 (AI)	18	81	24	0	0	0	24
8	69	72	17	9	11	0	28
9 (AI)	18	65	27	0	0	0	27
10	65	61	20	0	4	1	25
11 (AI)	48	59	24	0	0	0	24
12 (AI)	23	51	27	0	0	0	27
13	43	50	27	0	0	0	27
14	45	43	20	0	0	2	22
15	26	41	28	0	0	0	28
16 (AI)	20	36	28	0	0	0	28
17 (AI)	17	23	27+1*	0	0	0	28
N= 8 (without AI)	Ø 47.5 y.	Ø 70 months	141	49	15	4	208
N=9 (without AI)	Ø 23.6 y.	Ø 76 months	219	22	0	0	242

*: Single-tooth crown (lithium disilicate ceramic), adhesively cemented on an implant abutment
AI: Amelogenesis Imperfecta

Discussion

The long-term performance of the different materials shown in Table 1 and 2 (QR code on page 7) was assessed on the basis of adhesively bonded single-tooth restorations. During an observation period of ten years, we found that fractures of single-tooth silicate ceramic restorations were spread over the entire length of time (Figures 10 and 11), whereas lithium disilicate crown failures tended to occur towards the end of it. These results are comparable to those in the literature. Nevertheless, it must be noted that even though the number of restorations involved in our study was high, the number of patients was relatively low. Consequently, our findings are limited in terms of their validity. However, they do allow us to identify certain trends.

Molar restorations fractured most frequently: after about five years of service and primarily in patients whose vertical dimension had not been opened. The type of material used may have been responsible for the fractures or the restorations may have been too thin. We were surprised by the fact that the patients with AI had fewer complications

than the patients without AI. We had assumed that their restorations would not perform as well due to the "unfavourable" conditions for the adhesive bond. Age may have played a role in this finding, since the patients with AI were about 24 years younger on average compared with the other subjects. In addition, the results of the AI patients showed that adhesive all-ceramic restorations do not cause any endodontic problems. Therefore, they can be classified as a long-term treatment option. Nonetheless, the teeth of patients with AI require circumferential preparation to prevent any weak areas such as cement lines and to restore the function and anatomy of the teeth.

Summary

Adhesively cemented all-ceramic single-tooth restorations achieve excellent clinical results irrespective of the initial situation (Figure. 12). However, patients with a history of functional problems are expected to have a higher rate of technical complications. Overall, it is likely that about two in one hundred crowns every year will show some sort of complication after five to ten years of service and that primarily restorations in molars will fracture. Therefore, we recommend at least one check-up per year in order to treat any complications as early as possible and therefore minimize the risk of failure.

Annotation

The present results were previously published in the following article: Klink A, Groten M, Huettig F; Complete rehabilitation of compromised full dentitions with adhesively bonded all-ceramic single-tooth restorations: Long-term outcome in patients with and without amelogenesis imperfecta. J Dent. 2018 Mar;70:51-58. doi: 10.1016/j.jdent.2017.12.011. Epub 2017 Dec 21.

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Stress, burnout, substance abuse and impairment amongst members of the dental profession

Elizabeth Meyer[1]

"Stress is the physiological, emotional and behavioural response of a person seeking to adapt and adjust to internal and external pressures or demands. It is basically a physical survival response leading to a fight or flight reaction".[1]

Dentistry is recognised as a very stressful occupation.[2] Dentists experience daily stress and have to deal with difficult situations on a daily basis. As a highly regarded, skilled professional, the expectations of patients and society places a dentist in a challenging and vulnerable position. Forrest [3, 4] hypothesized that the practice of dentistry is a rewarding but demanding profession. He claimed that dentists need to identify factors that cause stress and must take measures to eliminate or at least reduce the harmful effects of stress on their health and emotions. He claimed that the health of dentists might depend on how successfully they keep the rewards and demands of their profession in proper perspective.

Stress Inducing Factors

1) Work-related stressors are: [5]

- a high patient load;
- lack of sufficient control, especially over resources for effective service delivery, especially in community-based dentistry;
- lack of recognition and appropriate reward;
- lack of social support;
- quality of working life;
- occupational hazards: exposure to HIV, TB, HBV and now Covid 19, ocular problems, eye injuries, latex allergy and musculoskeletal pain.

2) Dentist - Patient Interaction

Attention-seeking behaviour, the discussion of personal problems, manipulative behaviour, non-compliance and chronic pain are examples of dentist-patient interactions that may be very stressful to the dentist.[6]

3) Personality traits of the clinician

The definition of personality[7] refers to individual differences in characteristic patterns of thinking, feeling and behaving. The study of personality focuses on two broad areas: One is understanding individual differences in particular personality characteristics, such as sociability or irritability. The other is understanding how the various parts of a person come together as a whole. Stress may thus be individualised. What is stressful for one dentist may not necessarily be stressful for the next. Denollet[8] alleges that people with Type D were associated with higher levels of perceived stress and increased levels of burnout.

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4) Perception of stress

Although the sources of stress are varied, the important factor is the individual's perception and reaction to the stressor. Most importantly, an individual must alter his perception of a stressor and improve the relevant coping skills. The source of a stressor may be internal or external frustrations, conflicting needs or goals and pressures. Identification of the stressor could enhance coping skills.[6] According to Moore and Brodsgard[9] dentists perceive the following as intense stressors: keeping to schedule; causing pain; workload too heavy; late and anxious patients.

How much stress a person can tolerate comfortably varies not only with the accumulative effect of the stressors, but also with such factors as personal health, level of energy or fatigue, family situations and age.[6] In South Africa[10] dentists' stress derives furthermore from rising costs, problems with medical schemes, external interference by government and insurance companies, repetitive nature of the work, feelings of isolation, risk of infection and litigation. The most satisfied and least stressed dentists are older, report higher income, attend more continued education and employ more dental auxiliaries than those who are less satisfied.

Professional Burnout

One of the possible consequences of chronic occupational stress is professional burnout.[11] Burnout is defined by three co-existing characteristics: Firstly, the person is exhausted, mentally and emotionally. Secondly, he develops a negative, indifferent or cynical attitude towards patients, co-workers – so called de-personalisation or dehumanisation. Finally, there is a tendency for persons to feel dissatisfied with their accomplishments and to evaluate themselves negatively. The effects of burnout, although work-related, will often have a negative impact on a person's personal relationships and well-being.[6, 12]

It is interesting to note that health professionals who burnout relatively early in their careers were more likely to stay in their chosen careers and adopt a more flexible approach to their work routines. This suggests that burnout does not necessarily have to result in far-reaching negative consequences.[13] Researchers who looked at three types of clinicians found that general dentists and oral surgeons had the highest levels and that orthodontists had the lowest levels of burnout.[14, 15]

Substance abuse

Although substance abuse may be divided into licit (i.e. alcohol, tobacco, cannabis and prescription drugs) and illicit substances, the intention of this article is to concentrate on alcohol and prescription drugs as substances of abuse.

Alcohol

Anecdotally, dentists have been singled out as the healthcare professionals most likely to be subjected to severe stress, burnout, failed marriages, depression, substance abuse and suicide.[16]

Stress and health problems among dentists were determined by Randkin and Harris.[4, 17] They reported that dentists are vulnerable to health problems due to the stress associated with their profession. Unfortunately, most of the literature on the stress that dentists experience is based on opinions rather than systematic research. However, they reported that most dentists use alcohol or drugs in moderation. Males are more likely to consume alcohol and both male and female dentists use alcohol more frequently than other drugs. Meyers[18] conducted an anonymous survey amongst dentists in the UK to assess overall stress, work stress and health. They found that alcohol use is associated with work-stress amongst dentists.[4]

It is estimated that approximately 10-15% of all healthcare professionals will abuse a substance at some time during their career, a rate in fact similar to that of the general population,[19, 20] although there are some indications that the profession could have an even higher prevalence.[20, 21] Alcohol may be the most commonly abused substance amongst dentists,[20,22,23] but other researchers have not been able to find evidence confirming higher alcohol consumption amongst medical students compared with students in other fields of study.[20, 24] Curtis reported that while 10-12 percent of the general American population becomes addicted to alcohol or drugs at some point during their lives, the prevalence for dentists and physicians is probably 12-19 percent.[21]

The factors underlying substance abuse amongst dentists are complex including work stress, personal vulnerability encompassing temperament, motivation, trait disposition, genetic disposition and 'coping mechanisms.' What remains unclear are the relative contributions of stress and personal vulnerabilities as mediators of Alcohol Use Disorder (AUD).[25] The results of the Winwood study[25] indicated that South Australian dentists suffered high levels of stress/burnout and hazardous levels of alcohol consumption (two to four times higher than the normative South Australian population) were reported, particularly amongst males and rural practitioners.

Of significance, Olivier[4] found that the majority of dentists

polled in his study, believe in the existence of the so-called "conspiracy of silence" where colleagues, friends and next-of-kin are reluctant to report dentists who have dependency problem. This "conspiracy of silence" leads to denial and enables the dentist to continue abusing alcohol.

Clarno[26] focussed on the gravity of the consequences of alcoholism and/or drug dependence within the dental profession. He was of the opinion that dentists suffering from these illnesses can be identified through a pattern of abnormal behaviour and personal, vocational, and social consequences that are progressive and potentially fatal. When denial by colleagues, family, friends, professionals, and office personnel is overcome, enabling no longer perpetuates the illness and help is forthcoming. The sophistication of today's alcoholism treatment provides us with the tools to initiate recovery. The dentist and his or her family has an excellent chance for recovery and everyone gains – the victim, the family, the dental profession, the dental patient, and society. Colleagues and next of kin must recognise their denial and enabling and accept the responsibility to help suffering colleagues. Dentists are just as susceptible to disease as other humans, and the tragedy of a wasted life because of alcoholism is inexcusable with our level of knowledge of alcoholism programs today.

Prescription drugs

According to the American Dental Association's Dentist Well-Being Committee (Dentist Health and Wellness),[21] alcohol is the drug of choice for 37 percent of dentists with substance abuse problems, while prescription drugs (particularly opiates and anti-anxiety agents such as the benzodiazepines) are used by 31 percent, nitrous oxide by 5 percent and street drugs (including cocaine) by 10 percent.

Dental students as well as organised dentistry should be made aware of the dangers of abuse of not only prescription drugs, but also of prescribing outside the dental scope of practice, and especially of self – prescribing.[20]

Many clinicians hold the view that the ability to prescribe drugs for themselves, friends or family is a convenient aspect of the job. They argue it can often save time, and perhaps even resources, to make a quick self-diagnosis and write up a prescription without the need to take time away from work to consult an independent GP or dentist.[27]

Although this practice is technically not illegal, it does raise serious ethical concerns and could ultimately result in a complaint to the HPCSA. The regulators worldwide advise against treating and diagnosing yourself or those close to you.

The HPCSA[28] has handled a number of cases where practitioners have been subject to fitness to practise proceedings for either self-prescribing or for prescribing to a family member or friend. Some more serious cases have also been referred to investigators over allegations of defrauding medical aids in relation to prescription charges. In one case, a doctor faced fitness to practise proceedings before the HPCSA after it was found he wrote out prescriptions in his patient's names for drugs that were for his own personal use.[29]

"Other than in emergencies," the HPCSA[30] says, "you should not prescribe drugs for yourself or for anyone with whom you have a close personal or emotional relationship."

This advice is echoed by the GMC[31] in ***Good Practice in Prescribing Medicines*** which emphasises the importance of objectivity in providing good care, saying: "independent medical care should be sought whenever you or someone with whom you have a close personal relationship requires prescription medicines." It advises doctors not to prescribe a controlled drug for themselves or anyone close to them except in emergency circumstances where a delay "would put the patient's life or health at risk".

There are many reasons for such tight controls on self-prescribing and prescribing to family/friends, most of which are connected to the loss of objectivity. The GDC[32] cautions: "Everyone needs objective clinical advice and treatment. Dentists who prescribe drugs for themselves or those close to them may not be able to remain objective and risk overlooking serious problems, encouraging or tolerating addiction, or interfering with care or treatment provided by other healthcare professionals."

Causing or fuelling addiction is a major factor in self-prescribing, as the GMC warns: "Controlled drugs can present particular problems, occasionally resulting in a loss of objectivity, leading to drug misuse and misconduct." The guidance adds that doctors who do prescribe these drugs "must be able to justify your actions and must record your relationship and the emergency circumstances that necessitated your prescribing a controlled drug for yourself or someone close to you."

A loss of objectivity leaves clinicians unable to provide optimal care which can result in serious problems being overlooked, missed/ diagnosis delayed or misdiagnosed.

While most clinicians should recognise that prescribing opiates or powerful painkillers is entirely unacceptable, it appears many still believe it is acceptable to diagnose and treat themselves or loved-ones for low-level illnesses such as

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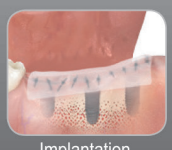
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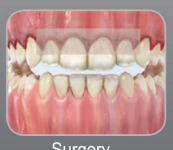
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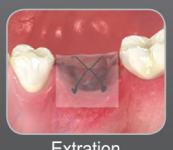
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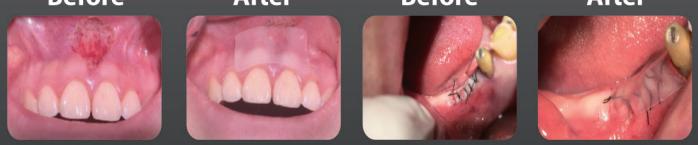

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It appears that the GMC and GDC take a very strict approach to clinicians who prescribe for themselves or those close to them. Recent GMC fitness to practise proceedings[33] have been raised against doctors for prescribing themselves or loved-ones with drugs such as benzodiazepines and opiates as well as with antibiotics and non-benzodiazepine hypnotics.

It is unfortunate that in South Africa, in spite of similar ethical guidelines and rules set by the HPCSA and the law, i.e. the three relevant Acts (Health Professions Act 56 of 1974, the Medicines and Related Substances Control Act 101 of 1965 and the National Health Act 61 of 2003) the law is not applied effectively to protect both the professional and the public.[20]

Impairment

An impaired dentist is unable to deliver optimal care to a patient. Colleagues who become aware of a dentist's dependency have a professional and ethical responsibility to intervene in a constructive manner.[35] Such interventions can involve discussing the issue with the afflicted colleague, calling upon family, friends or other support systems, offering help and finally reporting the dentist to the HPCSA Health Committee, established in terms of the Health Professions Act No. 56 of 1974 section 15(5)(F).

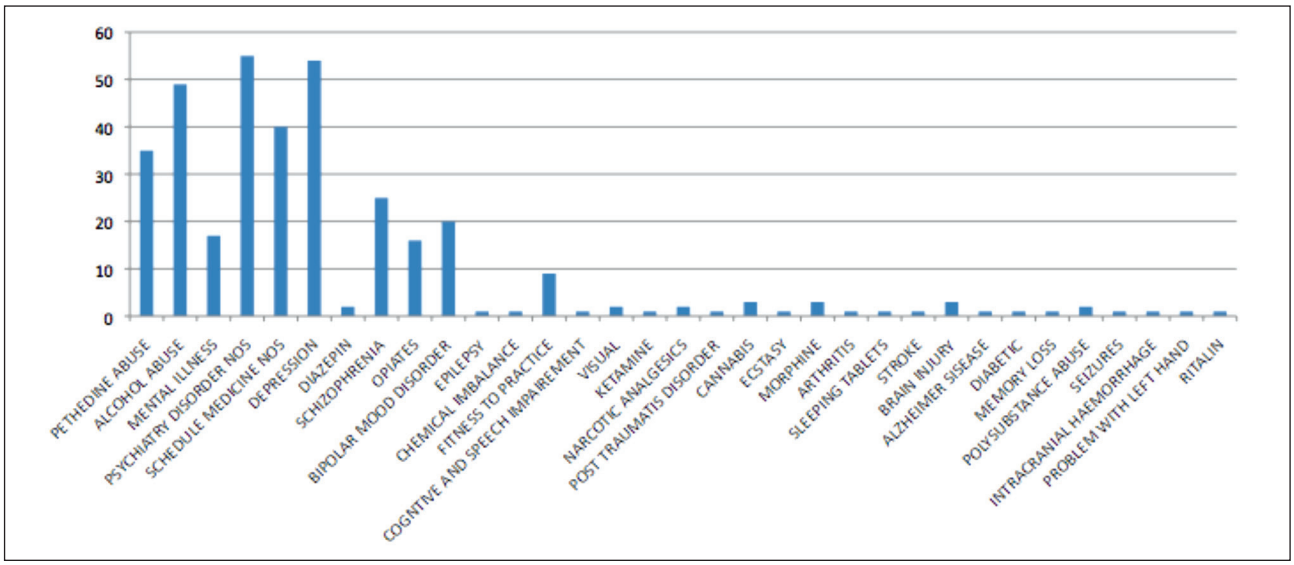
The committee regulates/advises impaired practitioners suffering from mental or physical conditions or the abuse of or dependence on chemical substances, affecting the competence, attitude, judgement or performance of a student or a practitioner.

The committee was established to manage compliance of the practitioners while protecting the public. The committee is non-punitive, meaning that its advice is provided to assist and not to punish.[34]

Conclusion

The dental profession is now, because of the Covid-19 crisis under more stress than ever. There are no indications that the additional financial and emotional pressures of the pandemic will be relieved soon. This will place vulnerable professionals at even a greater risk of stress related impairments. It is important for all dental personnel to be made aware of the risk factors leading to drug and alcohol addiction. It is important that co-workers, colleagues and next-of-kin recognise and understand when a dentist is under stress. It is the imperative that dentists identify support systems and it is perhaps opportune that the professional associations concentrate on creating support structures for the respective professions to help prevent substance abuse and to provide the necessary support for effective rehabilitation. As previous authors[51] have plead: "A national strategy between professional bodies and academic institutions in this regard is perhaps overdue and should be attended to as a matter of urgency."

List of stress-related impairments suffered by health professionals – April 2015 to March 2016 [34]



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CPD QUESTIONNAIRE 11.1.1

Article: COVID-19 risk management in dental practice. Part 2: The infection chain pathway of SARS-CoV-2. Hartshorne, van Zyl, page 20

1. *Which of the following statements relating to SARS-CoV-2 is TRUE?*
a Is a single stranded DNA virus b Non-enveloped
c Diameter 0.06 –0.14 nm
d Very susceptible to standard disinfection methods
e It invades the host target cells by using the sialic receptor for viral entry

2. *Which of the following statements relating to the stability of the SARS-CoV-2 in different environmental conditions is TRUE?*
a Not sensitive to heat
b Is unstable in a wide range of pH values (pH 3-10)
c Is stable on stainless steel for 2 days
d. Is stable on glass for 10 days
e Still present on the outer layer of a surgical mask after 7 days

3. *Which of the following statements relating to SARS-CoV-2 reservoirs is TRUE?*
a *A place where the infectious cycle starts, replicate and survive*
b *They can produce outside a cell*
c Lower viral loads are detected in nasal passages
d There are no viruses in the upper respiratory tract of asymptomatic hosts
e Salivary glands do not support SARS-CoV-2 replication

4. *Which of the following statements relating to how SARS-CoV-2 leaves the host reservoir is TRUE?*
a Cannot be transmitted through faecal route
b Can only exit through the nose
c Can exit the mouth during breathing
d Cannot exit the mouth in aerosols when performing aerosol generating procedures
e Cannot exit the mouth in sputum when coughing

5. *Which of the following statements relating to transmission of SARS-CoV-2 through respiratory droplets and aerosols is TRUE?*
a Expelled particles carrying pathogens exclusively disperse by either droplet or airborne transmission
b Respiratory droplets are formed from the fluid lining of the respiratory tract
c Lower viral loads have been detected in the nasal passages
d Coronavirus cannot transmit through oral droplets (sputum)

Article: COVID-19 risk management in dental practice. Part 2: The infection chain pathway of SARS-CoV-2. Hartshorne, van Zyl, page 20

6. *SARS-CoV-2 is primarily transmitted through which one of the following:*
a Respiratory droplets b Airborne (aerosols)
c Physical contact with infected droplets d All of above

7. *Which of the following statements relating to droplet and aerosol of transmission of SARS-CoV-2 is TRUE?*
a Viral particles can be aerosolized during dental procedures
b Breathing and talking does not emit aerosol particles
c Aerosol particles are >5 µm
d Aerosol particles cannot be inhaled into the respiratory tract

8. *Which of the following statements relating to the portal of entry and life cycle of SARS-CoV-2 is TRUE?*
a Droplet and aerosol entry points are only through the nose and eyes
b The S-protein of the virus allows it to attach to the ACE2 receptor on the host cell
c Recent studies show that ACE2 receptors are not expressed on the oral mucosa
d Mature virion exit the cell through the process of endocytosis

9. *Which of the following statements relating to susceptible host and risk factors for COVID-19 is TRUE?*
a The role of human genetics in determining clinical response is very specific and clearly defined
b Elderly female individuals are more susceptible to COVID-19
c Individuals with underlying hypertension are not at risk for COVID-19
d Most individuals admitted to hospital with COVID-19 had underlying co-morbidities

10. *Which of the following statements relating to asymptomatic infections is TRUE?*
a Asymptomatic infections are more common in elderly populations
b Asymptomatic infections are more common in individuals with co-morbidities
c Asymptomatic cases have the same infectivity as symptomatic COVID-19 cases
d Asymptomatic cases do not pose any challenges to infection control

ETHICS 2020 SUPPLEMENT

In response to our readers' requests, International Dentistry - African Edition is pleased to annouce the publication of our Ethics 2020 Supplement.

Co-edited by Prof Andre van Zyl and Dr Johan Hartshorne, the supplement has six excellent articles dealing with Ethics in dentistry, authored by Dr Johan Hartshorne, Dr Elizabeth Meyer and Prof Andre van Zyl.

Our sincere thanks go to the Editors and Authors and to our sponsors, **GSK** and **PPS,** who made this Supplement possible.

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CPD QUESTIONNAIRE 11.1.2

Article: Aesthetic two stage crown lengthening for altered passive eruption: A 25-year case report and review.
Van Zyl & Snyman, page 38

11. What is the reported prevalence of delayed passive eruption in South Africa?
a 19%
b 21%
c 12%
d 9.5%

12. What is the proposed treatment for Altered Passive Eruption type 1A?
a Osteoplasty followed by gingivectomy
b Gingivoplasty
c Gingivectomy
d Osteoplasty only

13. Can gingival phenotype be modified by clinical intervention?
a No, it is determined by genetics only
b Yes, but only in identical twins
c Yes, it can
d Not- it is impossible

14. What is the suggested distance between bone crest and planned gingival margin after crown lengthening?
a 5-6 mm
b 2 mm in more than 90% of cases
c ≥ 3mm
d Between 2-3mm

15. With regards to Altered Passive Eruption subgroup A, where is the alveolar bone crest located?
a 1-3mm coronal to cemento-enamel junction
b 1-3mm apical to cemento-enamel junction
c At the cemento-enamel junction
d Can be above or below the cemento-enamel junction

Article: Stress, burnout, substance abuse and impairment amongst members of the dental profession.
Meyer, page 56

16. Which of the following statements characterizing the dental professions are TRUE ?
a Patients and society place a dentist in a challenging and vulnerable position
b Dentistry is a rewarding but demanding profession
c Dentists have to deal with difficult situations on a daily basis
d All of the above

17. Patients with a Type A personality trait are claimed to have higher levels of perceived stress and increased levels of burnout (TRUE or FALSE?)
a True b False

18. Which of the following statement regarding professional burnout is TRUE?
a Orthodontists have the highest level of burnout
b General dentists and oral surgeons have the lowest level of burnout
c Most dentists with burnout are mentally and emotionally exhausted
d Tendency by dentists to feel satisfied with their accomplishments

19. Which of the following statements relating to substance and alcohol abuse is FALSE?
a Cannabis is a licit substance
b Smoking may be the most commonly abused substance among dentists
c Alcohol may be associated with work stress amongst dentists
d It is reported that the prevalence of addiction to alcohol or drugs for dentists is probably 12-19%

20. Which of the following statements regarding prescription drug is TRUE?
a A study has suggested that 31% of dentists use opiate and anti-anxiety prescription drugs
b The ability of dentists to prescribe drugs for themselves is technically illegal
c Relevant legislation is applied effectively in South Africa to protect both the profession and public against self-prescribing by dentists
d All of the above

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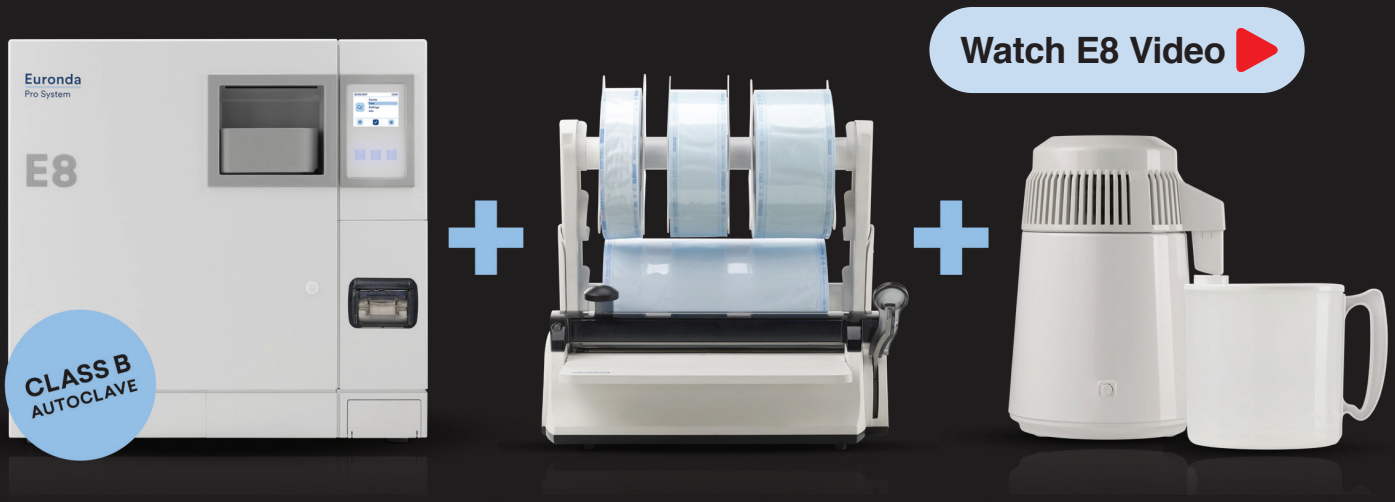

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