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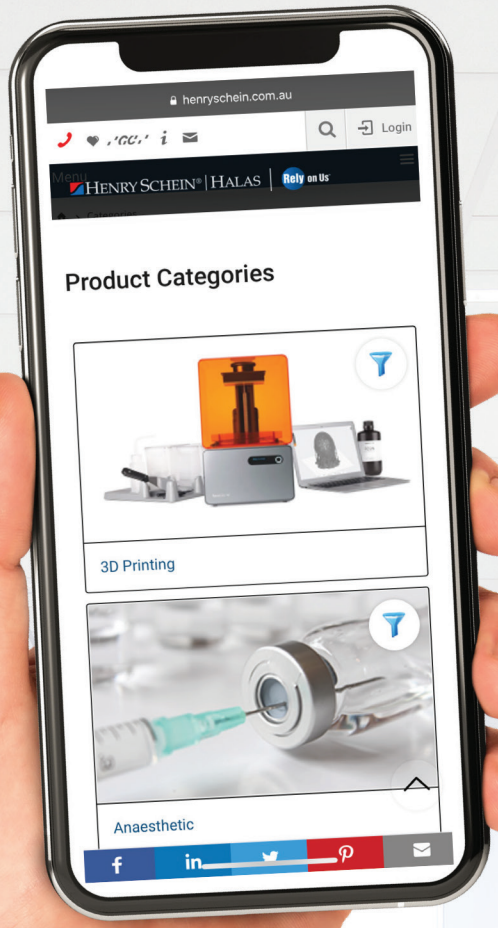

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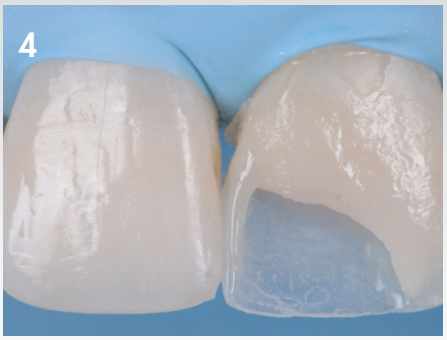
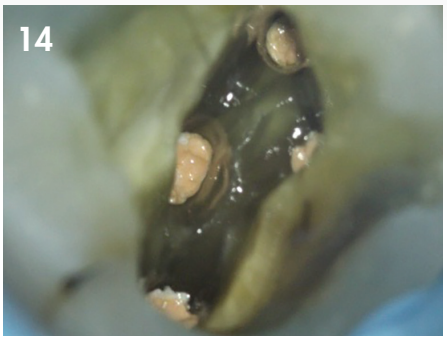
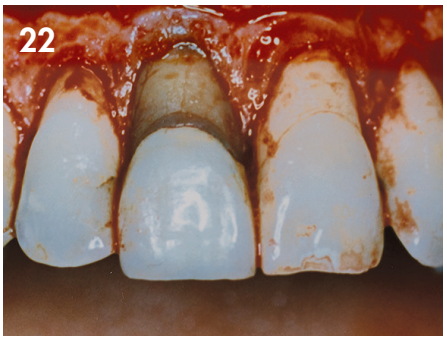
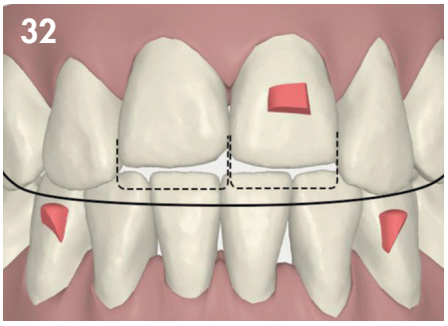


Contents

Volume 16 No. 1







Masking up thanks to Henry Schein

So whilst to many the end of 2020 and beginning of 2021 began to feel like groundhog day, the realisation set in that Covid was here to stay, for the time being at least. A mask order was mandated within certain states in Australia and the flow-on effect was that masks became harder to come by and those without access to disposable income were disadvantaged once again.

In response to this, throughout the month of January Henry Schein Cares donated 30,000 masks to those who may not otherwise been able to access this vital health resource. We continue to ensure that the most vulnerable in the community are supported in their health and wellbeing through the provision of face masks.

Some of Henry Schein's most valued community Organisations such as Our Big Kitchen, Will2Live, Canice's Kitchen and Georges River Life Centre, to name a few, were the recipients of much needed masks. This allowed the masks to be provided throughout the community networks, reaching as many people in need as possible.

Henry Schein has a history of philanthropic values and the belief that those who are often ostracised in society should not be forgotten and more so during a global pandemic. Our vulnerable community members are at much greater risk due to their often transient lifestyles and not having a safe haven to isolate within. Henry Schein will continue to ensure that these members of society are safe and not forgotten.

If you have any questions about Henry Schein Cares and other projects we are involved in, please email **scheincares@henryschein.com.au**

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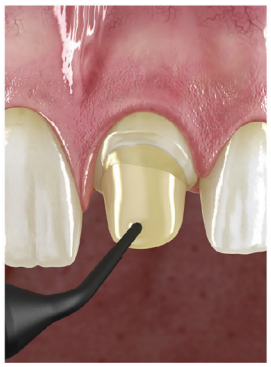

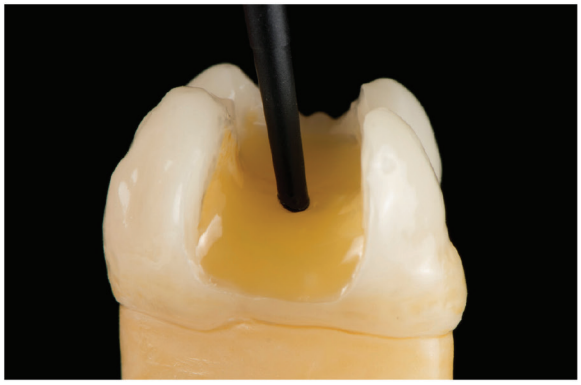

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A full digital workflow with 3D-printed temporary restorations

Anthony Mak[1] and Andrew Chio[2]

The evolution of digital technologies in dentistry has paved the way for the development of simplified and predictable protocols in field of restorative dentistry. Digital dental technologies have allowed the seamless delivery of complex treatments.

Proper treatment planning protocols are the foundation of any fixed restorations in the arch involving dental implants. The data or information from the CBCT scan and intraoral surface scans (IOS) combined with the use of CAD software allow the simplification of workflows including diagnostic facially driven mock-ups, restoration-driven implant treatment planning and the design and fabrication of surgical guides. The design of the temporary and permanent prosthesis and the design of the master die model can all be done on CAD software and then manufactured either with 3D printing or milling. The prosthetic design can be visualized, planned and even designed prior to the patient even attending for the surgical phase of treatment.

An accurate and predictable outcome of the implant surgery as well as the restorative rehabilitation are realised this way.

The following case study demonstrates a scenario where a complete digital workflow was utilised with two provisionalisation phases to rehabilitate the full upper arch.

[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

Diagnostic Record Collation and Treatment Planning Phase

A 79-year old patient presented with an unremarkable health history.

Chief complaint:

- mobile teeth
- occasional discomfort from the areas around his existing upper fixed partial denture

Examination (both clinical and radiographic) indicated the following (Fig. 1):

- moderate to advanced bone loss affecting many of his upper and lower teeth.

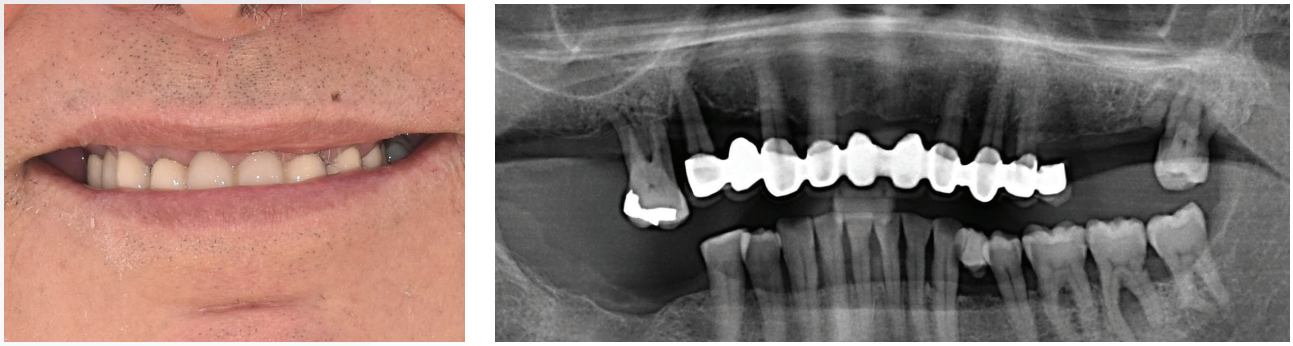

Figure 1: Pre-operative smile and orthopanthomogram.

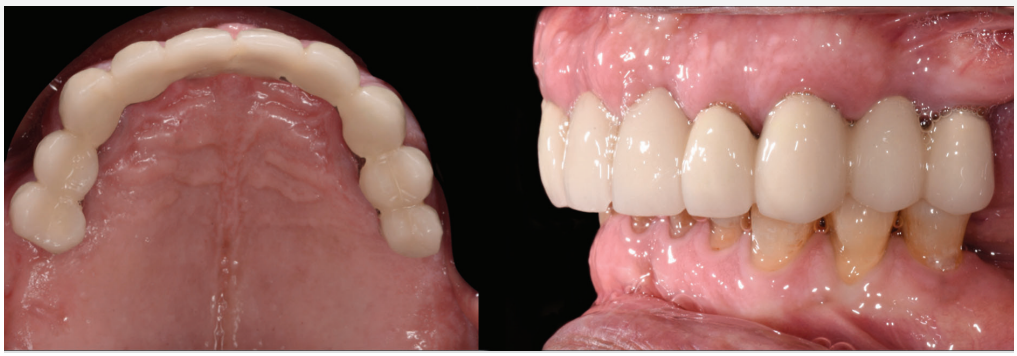

Figure 2: Occlusal and lateral view after periodontal treatment and extraction of tooth 16 and 28.

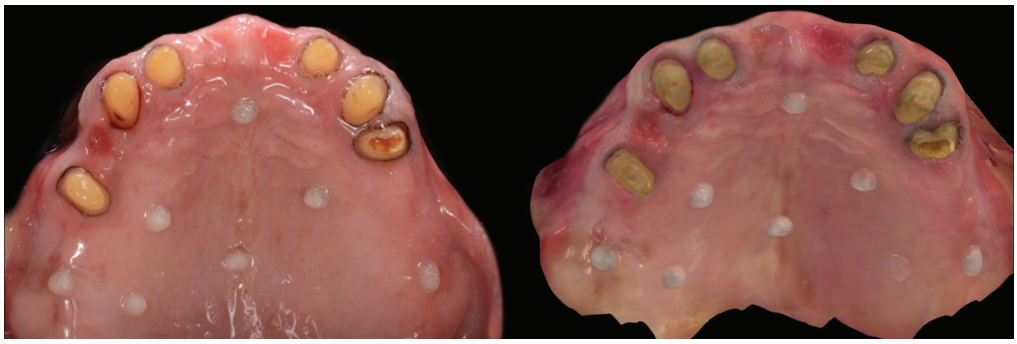

Figure 3: The accuracy of image registration between the CBCT and IOS scans can be improved with radiographic markers (composite blobs). Removing sources of radiographic scatter (in this case, the PFM bridge) also improves the accuracy.

- secondary decay was diagnosed on the abutments of his fixed dental prosthesis.
- Teeth 15, 16 and 28 had a poor prognosis and were planned for extraction.

The goal of the treatment was to rehabilitate the upper arch with a combination of crowns and implant retained restorations to provide the patient with a fixed solution.

In the initial treatment phase, teeth 16 and 28 were extracted and the remaining dentition was periodontally treated (Fig. 2).

After the initial clinical examination and treatment, further information was collated. This included:

- 3D CBCT scanning for the presurgical planning.
- Intra-oral scans (IOS): digital impressions before and after removal of the original PFM bridge were taken, as well as the patient's occlusion (bite). Rough preparation of the tooth abutments were also completed prior to the acquisition of the subsequent IOS scan.

Tip: the accuracy of image registration (superimposition of the IOS and CBCT data) can be enhanced by (Fig. 3):

- the use of radiographic reference markers: a composite such as G-ænial Universal Injectable with a radiopacity of 250% Al, does not result in radiographic scattering during CBCT scans.
- prior removal of the porcelain-fused-to-metal (PFM) bridge: reduction of radiographic scatter caused by the metallic components of the prosthesis

Treatment Plan

Following the collation of the information, the initial treatment plan was formulated and involved:

- Guided surgical placement of implant fixtures in the 16, 14, 11, 21 and 25 sites. A bone graft was also planned in the 11 site due to bony defects. A two-stage surgical protocol was chosen for proper integration of the implants in the 11 and 21 site.
- Immediate provisionalization with a 3D-printed temporary bridge (GC Temp PRINT) from 15 to 24. The existing shape and contours of the current failing bridge were copied from the pre-operative IOS to create the temporary bridge.

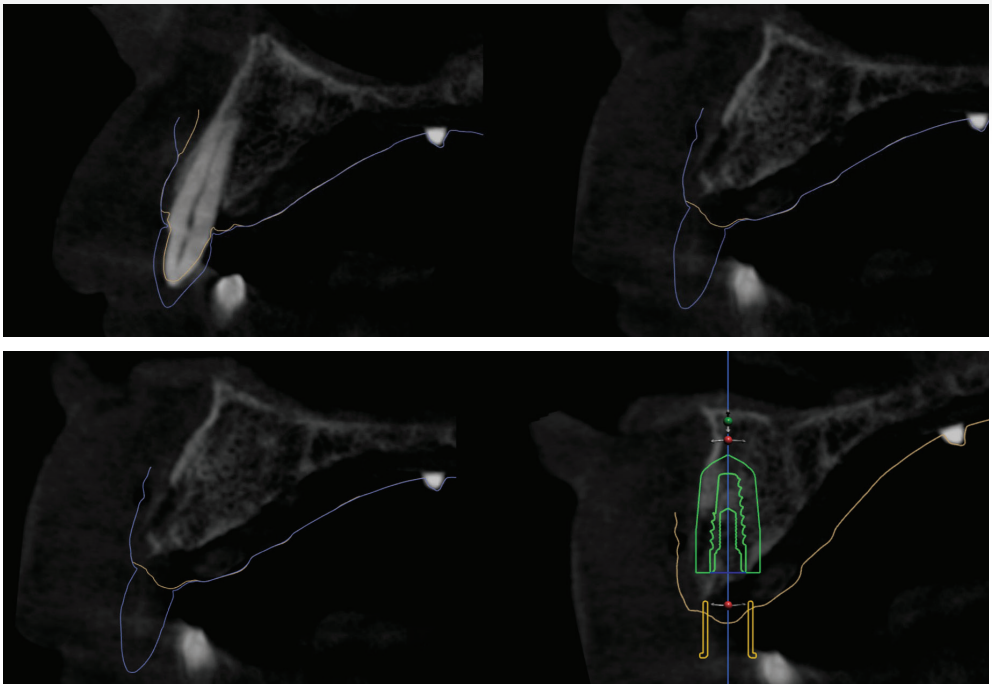

Figure 4: Intraoral surface scans (IOS) before and after removal of the original PFM bridge superimposed on the CBCT scan: this facilitates the planning of implant placement from a restorative perspective (restoration driven implant placement).

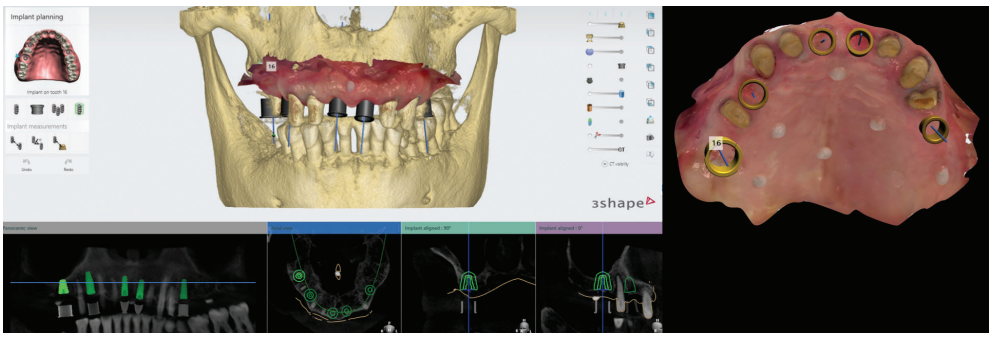

Figure 5: Planning of implant placement. A surgical guide is designed based on the desired implant position.

- After implant integration, a second phase of provisionalization was foreseen with individual temporary restorations (GC Temp PRINT) on the implants and natural teeth. This allowed:
  - Verification of aesthetics and occlusion
  - Soft tissue management
  - Extraction of tooth 15.
  - It was planned to use lithium disilicate and monolithic zirconia for the permanent restorations on both the natural teeth and implant abutments.

Digital Implant Planning and Surgical Guide Fabrication

Digital data from the three scans – the CBCT and the IOS before and after bridge removal - were accurately merged. This enabled virtual planning of the number, position, angulation and access position of the implant fixtures following a restoratively driven protocol (Fig. 4).

Based on the planned implant positioning (Fig. 5), a surgical guide was designed with the dedicated software. Master sleeves from the guided surgical system were placed

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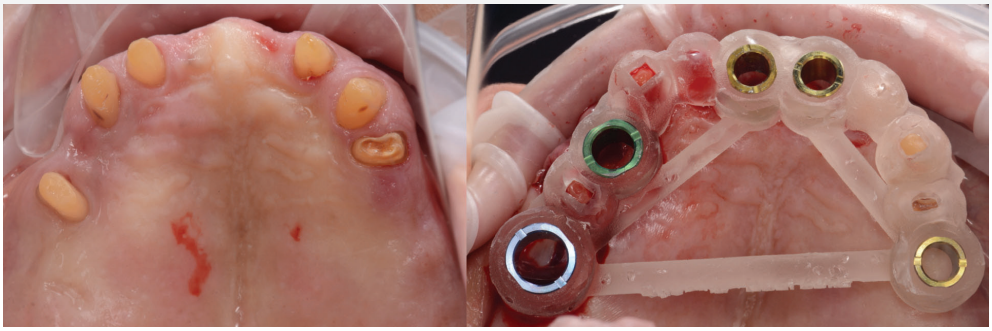


Figure 6: Five implant fixtures were placed using a fully guided surgical protocol.

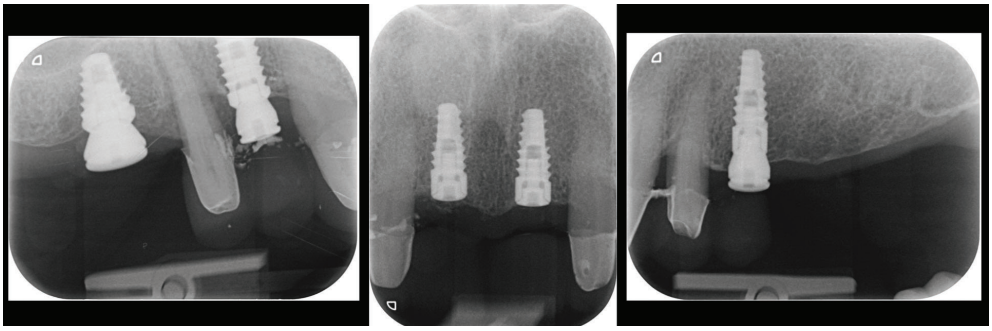


Figure 7: A flap was raised in the 11 region as buccal bone grafting was required due to a bony defect.

and fixed to the printed guide/framework.

The design of the previous PFM was also copied and replicated in the digital planning of the temporary bridge. It was then printed using the Asiga Max UV and GC Temp PRINT (medium shade) set at 50µm on the 3D printer.

Guided Implant Surgery and First Provisionalization Phase

The following clinical procedures were then completed on the day of implant surgery:

- All five implant fixtures were placed following a fully guided surgical protocol with the surgical guide (Fig. 6) and primary stability was confirmed.
- A flap was raised in the 11-21 region, a bone graft with bovine cancellous particulate was placed and covered with a porcine collagen membrane. Cover screws were placed and primary closure was established after a relieving incision and closed with PTFE sutures. At the other implant sites (16, 14 and 25), healing abutments were placed (Fig. 7).
- The 3D-printed temporary bridge was then cemented with GC Fuji TEMP LT on the remaining natural teeth (Fig. 8).

A healing period of 16 weeks allowed complete osseointegration of the implant fixtures. During this period, tooth 24 (upper left first premolar) developed signs and symptoms of pulpal necrosis. Hence, it was endodontically treated (Fig. 9).

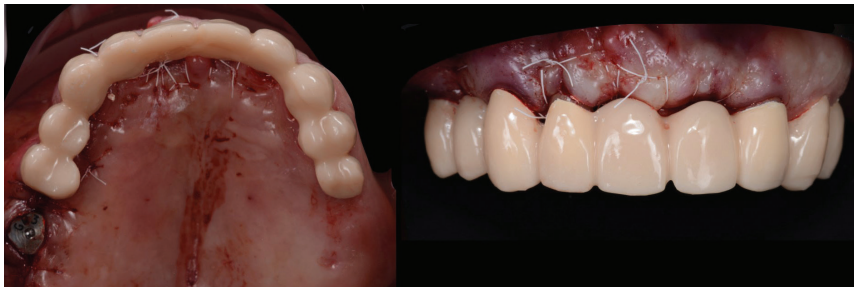


Figure 8: Immediate post-operative following guided implant surgery and temporary cementation of the provisional fixed bridge printed from GC Temp PRINT (medium shade).

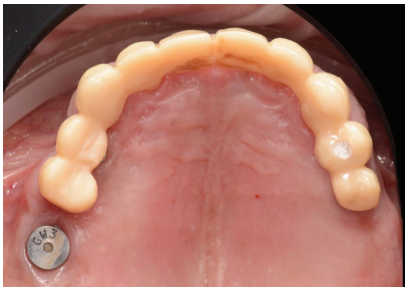


Figure 9: During the healing phase, tooth 24 developed pulpal necrosis and was endodontically treated.

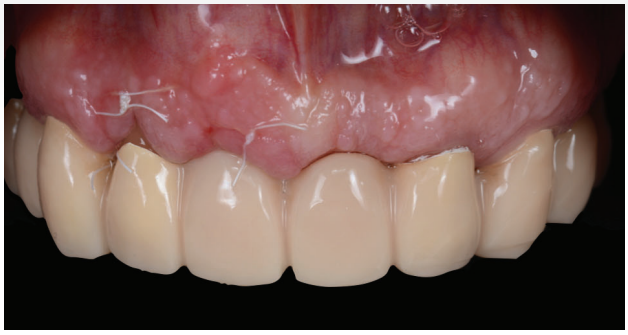


Figure 10: View at 10 days after implant surgery.

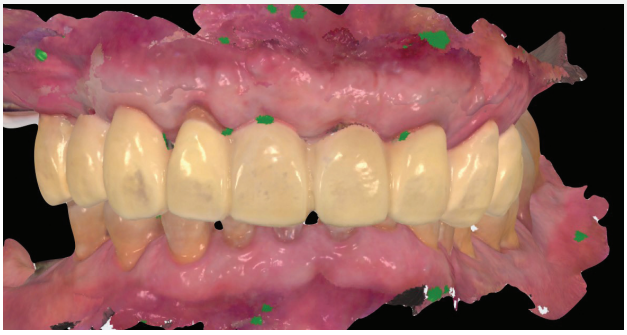


Figure 11: Pre-operative surface scan.

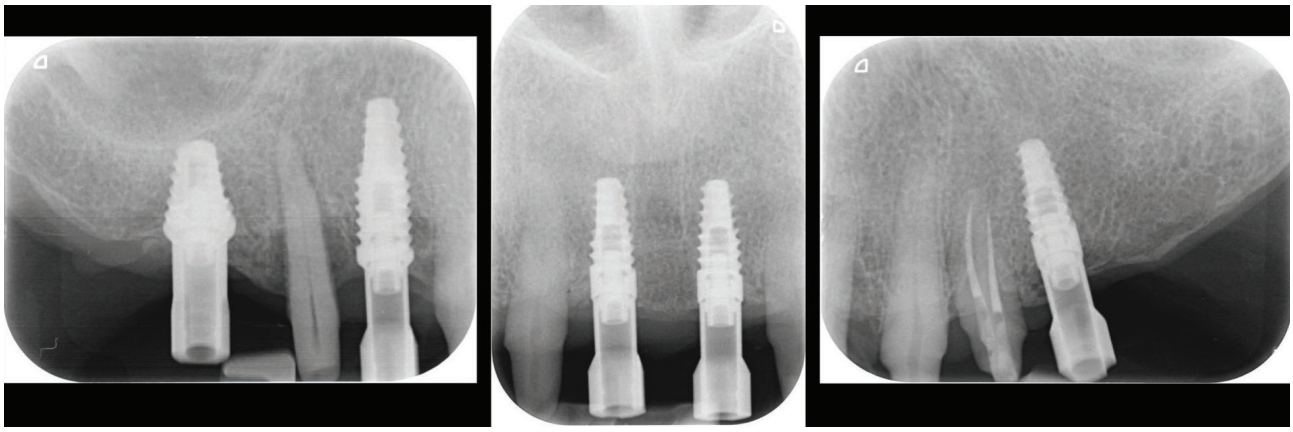


Figure 12: Periapical radiographs to verify the seat of the digital scan bodies.

Second Provisionalization Phase after Implant Integration.

Once the 16-week healing phase was completed and the fixtures were integrated, the restorative phase could be initiated. The patient confirmed that he was happy with the shape and occlusion of the initial temporary bridge (Fig. 10). The aesthetic and occlusal scheme could therefore be replicated in the second phase of provisionalization.

A pre-preparation IOS was taken with the healing abutment and temporary bridge in situ (Fig. 11).

The temporary bridge was then removed and preparation of the abutment teeth finalized and re-margined to the healed gingival tissue levels.

Stage 2 implant surgery on the 11 and 21 sites was completed using a soft tissue diode laser. The implants were exposed and cover screws removed.

An emergence profile scan was taken immediately after the healing abutments were removed to record gingival contours around the implant before any collapse of the tissues. Next, the full upper arch was scanned with digital scan bodies in place to capture the implant position accurately (Fig. 12).

All other prosthodontic records including the bite registration and the opposing arch were also captured with the intra-oral scanner before placing the temporary bridge back. All IOS were taken following the "Mak optimised scan strategy" (MOSS), allowing accurate stitching of IOS images. In soft tissue "pink" areas, the availability of landmarks is often limited; MOSS uses a specific scan path with or without markers for an enhanced scan accuracy and was especially designed for cases with few teeth to correlate to.

All the digital data was then sent to the ceramist for the fabrication the second set of provisional restorations. Provisional restorations were printed with GC Temp PRINT and characterised with OPTIGLAZE color (GC). Temporary abutment cylinders were utilised for the implant-retained restorations. The contours of the 11 and 21 implant-retained provisionals as well as the pontic of 15 were designed and fabricated to shape the soft tissues for optimal support and

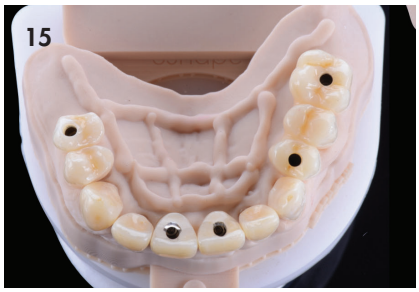
Figure 13: Second set of provisional restorations printed with GC Temp PRINT (medium shade) using the Asiga Max UV 3D printer.

Figure 14: Completed provisional crowns, implant retained crowns and bridge, characterised with OPTIGLAZE color (GC) – Dental technician: Brad Groblar, Oral Dynamics, New Zealand.

(Figs. 13-15).

Following removal of the temporary bridge, all the abutments were cleaned and the tooth 15 was extracted (Fig. 16). The provisional implant restorations, fabricated with direct screw access were torqued to the manufacturer's recommendation. All other temporary printed restorations were cemented with FujiTemp (GC) (Figs. 17-19).

The soft tissues were prosthetically shaped and allowed to



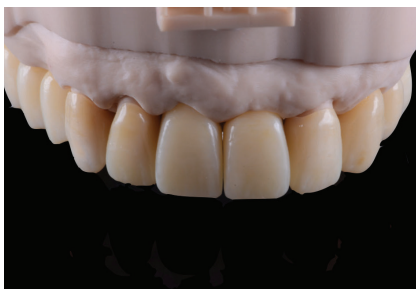
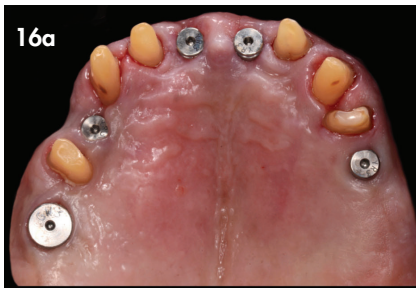


Figure 15: Completed provisionals fitted onto the printed models to allow the refinement of the contact points and occlusal contacts.



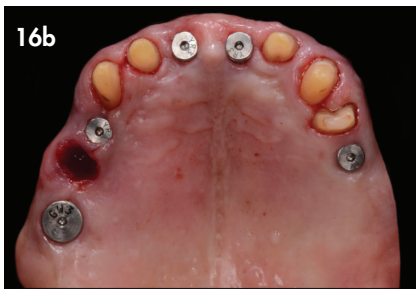
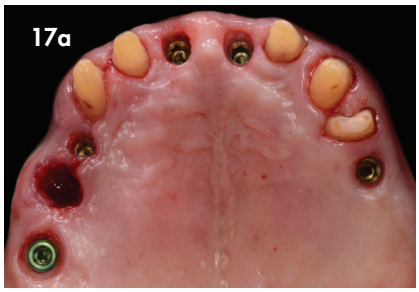


Figure 16: (a) After removal of the temporary bridge from the first provisionalization phase. (b) Tooth 15 was extracted.



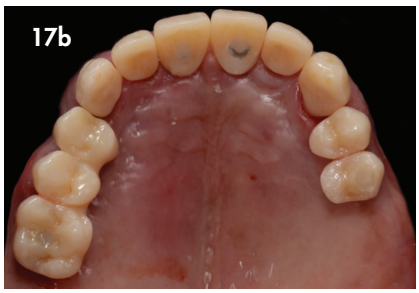


Figure 17: (a) Healing abutments were removed and (b) the second set of temporary restorations was placed.

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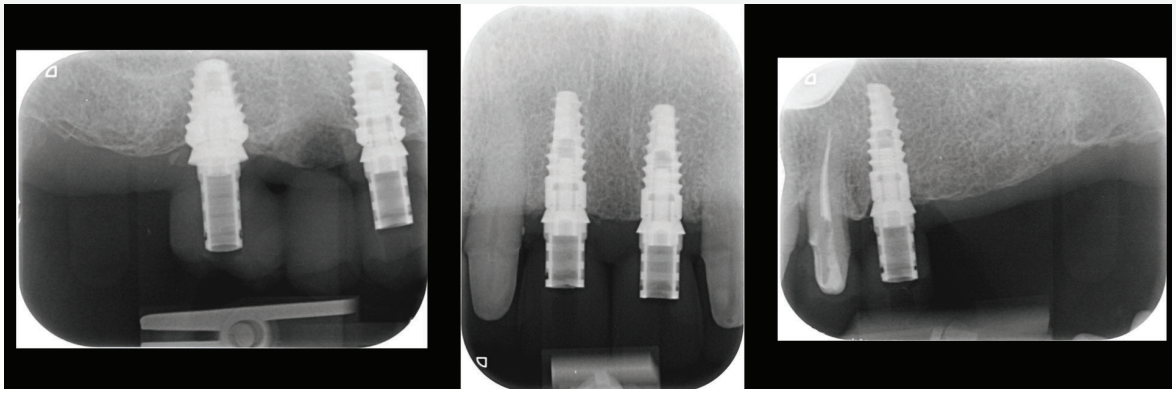

Figure 18: Periapical radiographs to verify the seat of the implant-retained provisional restorations.

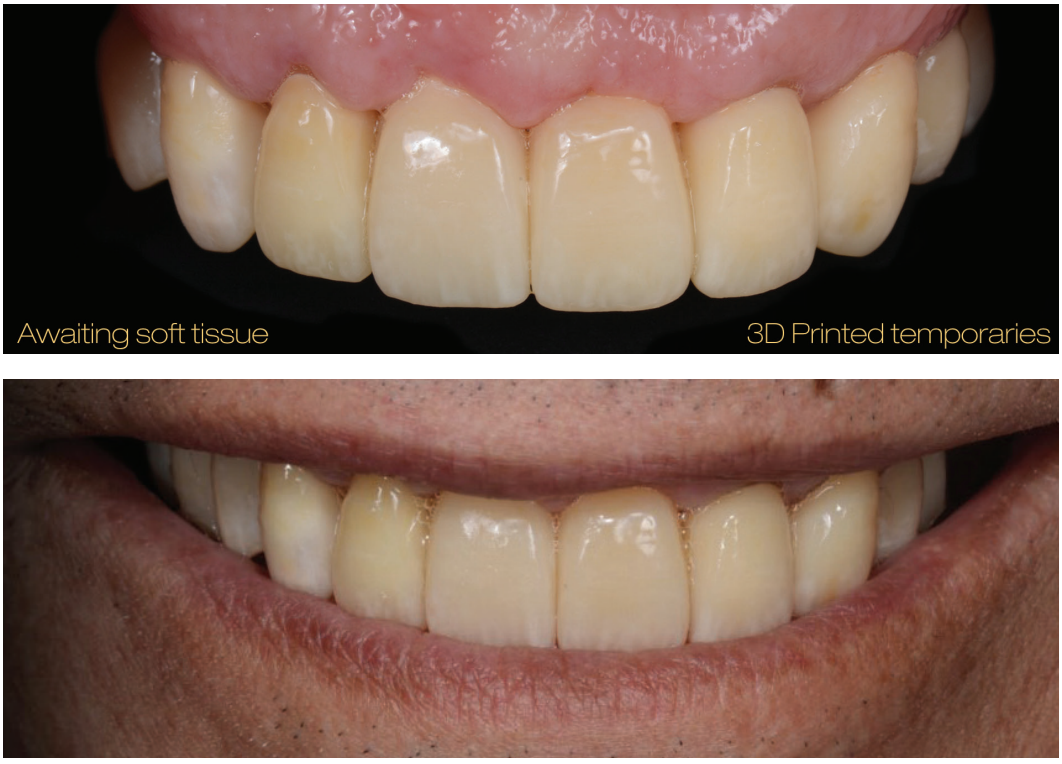

Figure 19: Immediate post-operative view of the inserted provisionals.

heal for a period of 3 months before the finalisation of the rehabilitation with the definitive restorations.

Conclusion

The case presented illustrates how advances in digital technologies can provide clinicians with the tools for diagnosis, treatment planning, the execution and provision of dental restorative procedures in a truly transformative way.

Simplification of clinical protocols, increased accuracy over conventional analogue techniques and improved patient comfort and outcomes are compelling reasons of the benefits of a full digital workflow in the field of restorative and implant dentistry.

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Management of the MB2

Kreena Patel[1]

The MB2 or 'fourth' canal has a quite the reputation for being challenging to locate and negotiate. Kreena Patel discusses facts about the MB2 and clinical tips for successfully managing it.

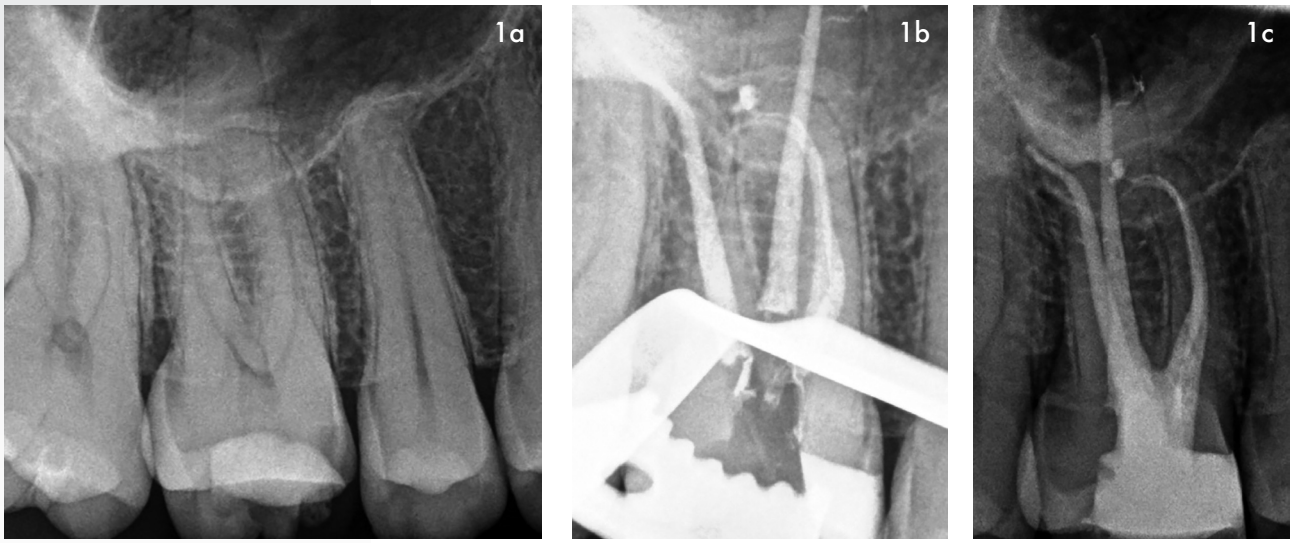
[1] Kreena Patel, BDS(Hons), MJDF RCS(Eng), MClinDent (Endodontology), MEndo RCS(Edin).
Specialist in Endodontics. Lecturer on the Endodontic specialist programme, King's College London
Private Practice, Surrey and Berkshire, UK.
@kreenaspecialistendodontics.

Importance

Just how important is this little canal? This question is often debated among clinicians as some feel root canal treatment can be carried out successfully without treating MB2.

Successful endodontic treatment relies on locating, disinfecting and obturating all the canals. Studies have shown the incidence of MB2 canals is roughly 90% in maxillary first molars and 60% in maxillary second molars. Therefore, the majority of maxillary molars contain four canals and we should start root canal treatment of these teeth with this in mind.

There are often multiple ports of communication between MB1 and MB2. The MB2 can join MB1 along its path or terminate via a separate apical foramen (Figure 1 and Figure 2). Rarely, there can be an MB3 canal present (Figure 3).



Figures 1a, 1b and 1c: MB1 and MB2 are two separate canals

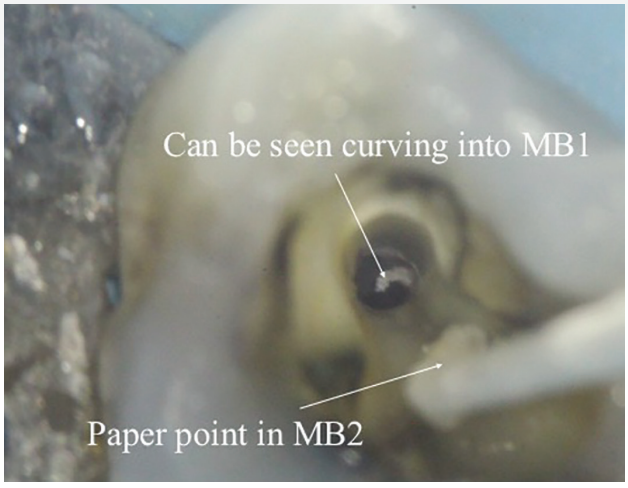


Figure 2: A paper point has been placed in the MB2, and can be seen appearing in MB1 where the canals join

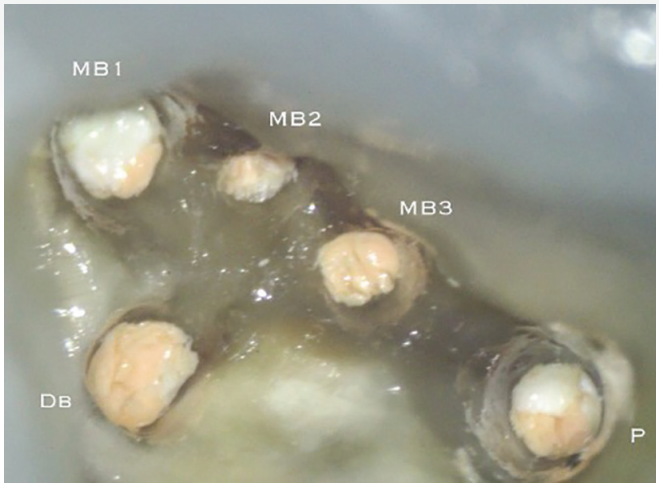


Figure 3: Upper second maxillary molar with MB1, MB2 and MB3

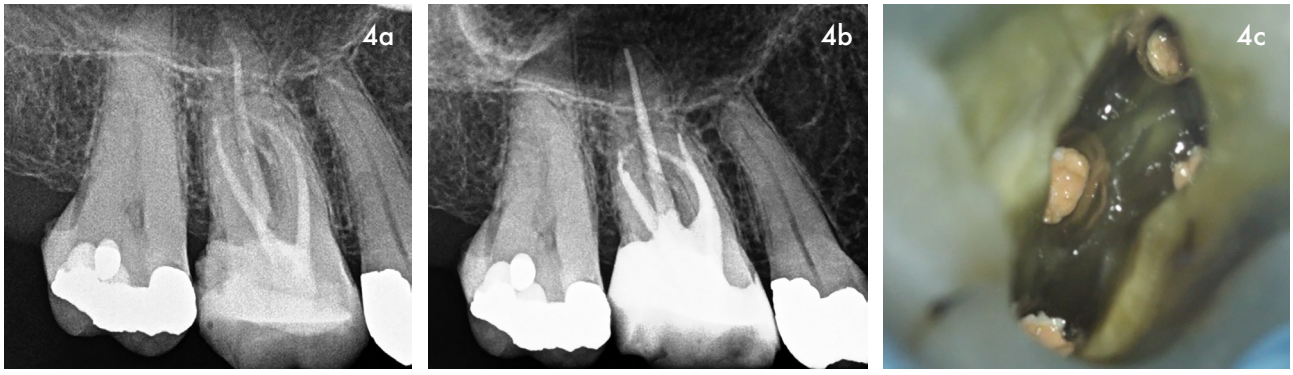
A missed MB2 canal is one of the main causes of endodontic failure in maxillary molars. In cases of irreversible pulpitis, it may be responsible for ongoing temperature sensitivity, and in necrotic cases, residual bacteria will increase the risk of infection (Figure 4).

CBCT

Cone beam CT has become a very useful tool in endodontics because it allows us to see root canal morphology is three dimensions. It is important to note there are large differences in scan quality obtained by various CBCT machines; a high-resolution small volume scan is necessary to visualise the fine details required for endodontics. CBCT has been shown to be a reliable tool for detecting MB2 canals and assessing their path up the root (Figure 5). However, studies have shown MB2 canals are still sometimes located clinically even when they are not seen on the scan (Blattner et al, 2010; Parker et al, 2017).

Location

Magnification and lighting make all the difference when trying to locate MB2. Studies have shown that the frequency of MB2 canal detection for microscope, dental loupes and no magnification were 71.1%, 62.5% and 17.2% respectively (Buhrley et al, 2002). The experience of the operator and time spent searching for the canal have also been shown to be important factors.



Figures 4a, 4b and 4c: Patient was referred for the root canal retreatment of UR6. The tooth had been treated privately with her general dentist three years ago; the root canal treatment had been carried out to a good standard (under rubber dam isolation, three canals cleaned, shaped, disinfected using sodium hypochlorite and obturated to length). The patient did not have significant pain but the tooth did not 'feel right' and was affecting her function. Root canal retreatment was carried out and an additional MB2 canal was located. The patient's symptoms settled immediately following treatment and she was advised to proceed with a cuspal coverage restoration

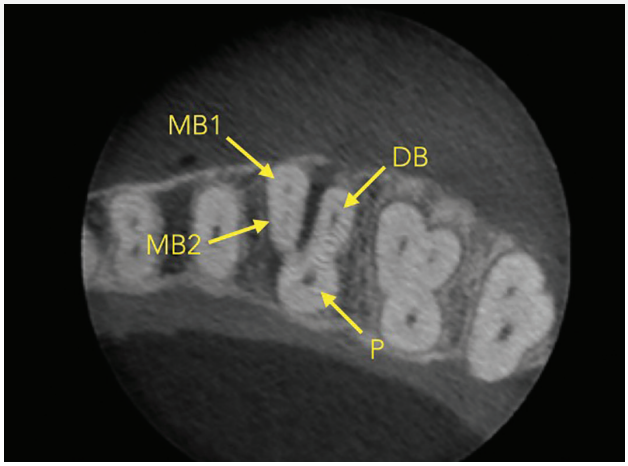
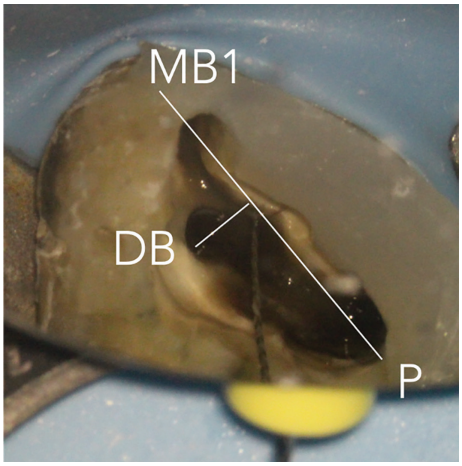


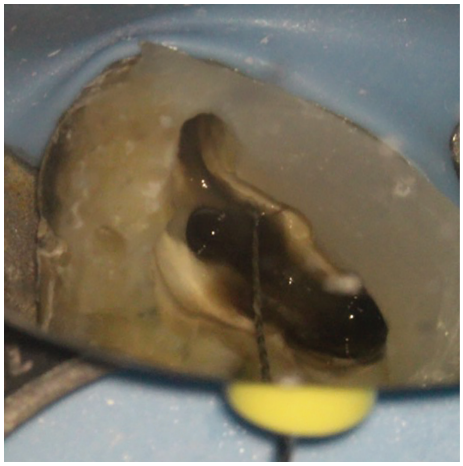
Figure 5: CBCT image (axial view) of a maxillary molar highlighting the presence of two canals in the MB root. Sometimes the MB2 canal is more calcified and cannot be seen clearly. If the MB root form is oval and the MB1 is asymmetrically positioned then there will still likely be an MB2 canal present

The pulp floor has developmental root fusion lines which are darker; these can provide a road map for locating canals because of where the orifices lie (Krasner and Rankow, 2004). The MB2 canal is commonly located within the developmental groove between MB1 and palatal orifices. Envisage a line joining the MB1 and palatal canal, and draw another line from DB to this line – in the majority of cases MB2 is located at this juncture, and a few millimetres away from the MB1 orifice (Figure 6). A sharp DG16 probe is essential for exploring the area and locating the orifice. Less frequently, the MB2 orifice can lie closer to the palatal orifice (Figure 7) or within the MB1 orifice itself (Figure 8).

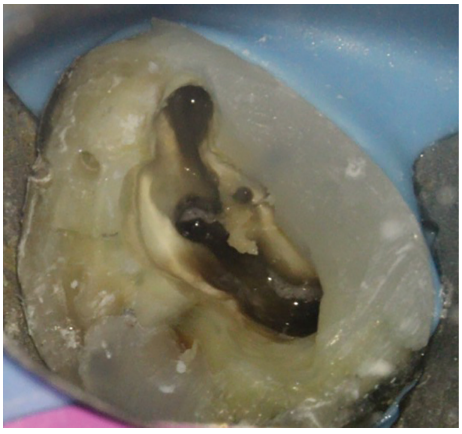
The orifice lies at the junction of the pulp floor and mesial wall, and is frequently covered by a mesial lip or 'shelf'



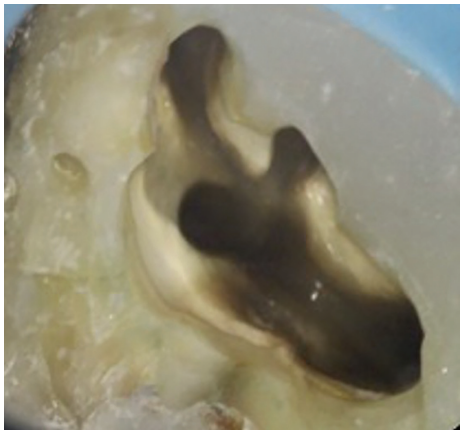
6a: The MB2 orifice normally lies at the juncture of these two lines



6b: MB2 located with a hand file



6c: Orifice enlargement and coronal flaring



6d: After cleaning and shaping

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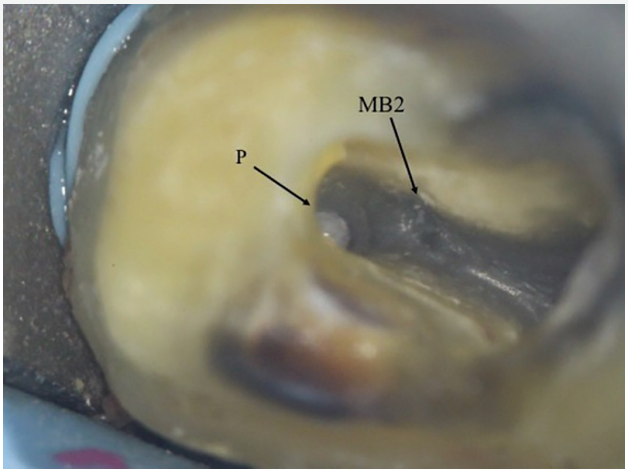
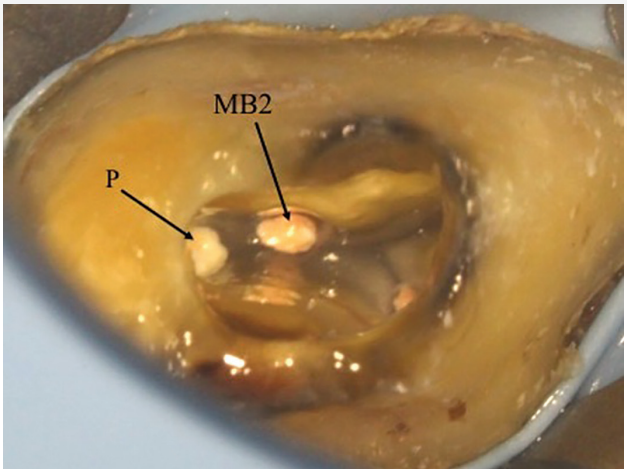
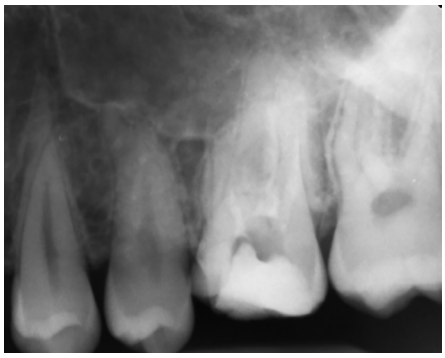


7a: MB2 orifice detected close to the palatal canal

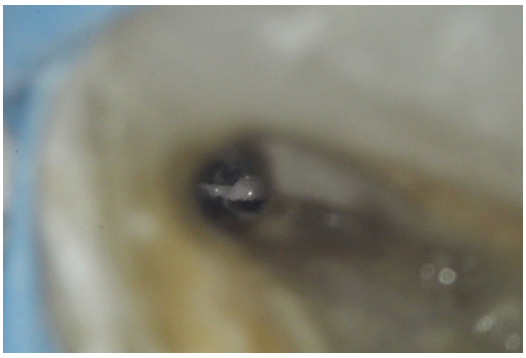


7b: After obturation

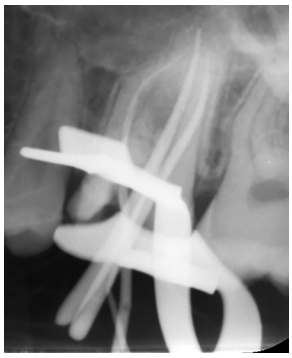
of dentine, particularly in older patients. Therefore, the access cavity will need to be extended to remove this. In calcified cases, the MB2 may be slightly deeper apically. Piezo-electric ultrasonic tips or long-neck burs used in a slow handpiece are very useful removing the dentine shelf and searching for MB2. They allow good vision during



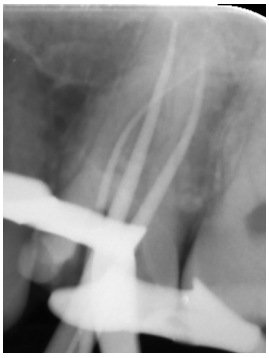
8a: Preoperative radiograph. The tooth had previously been accessed by the referral dentist.



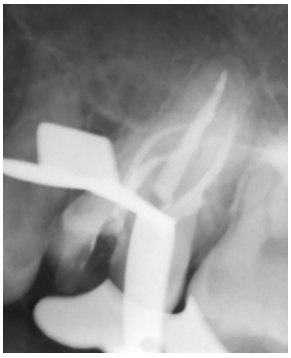
8b: MB2 was located within the MB1 orifice (mesial wall). The microscope photograph was taken after the MB1 and MB2 have been fully prepared



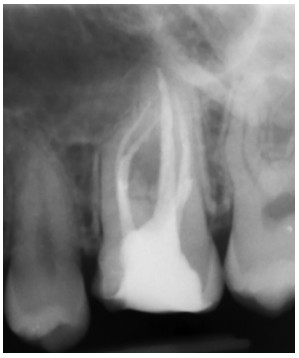
8c: Master cone radiographs taken with a GP cone in MB1

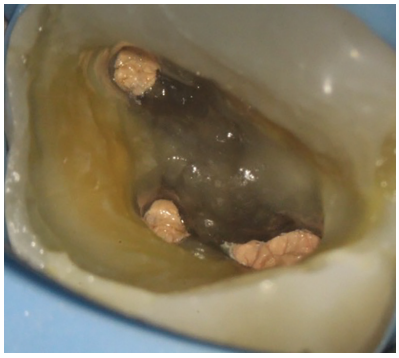


8d: Master cone radiograph taken with cone in MB2 d) Mid-fill radiograph shows MB1 and MB2 are separate canals

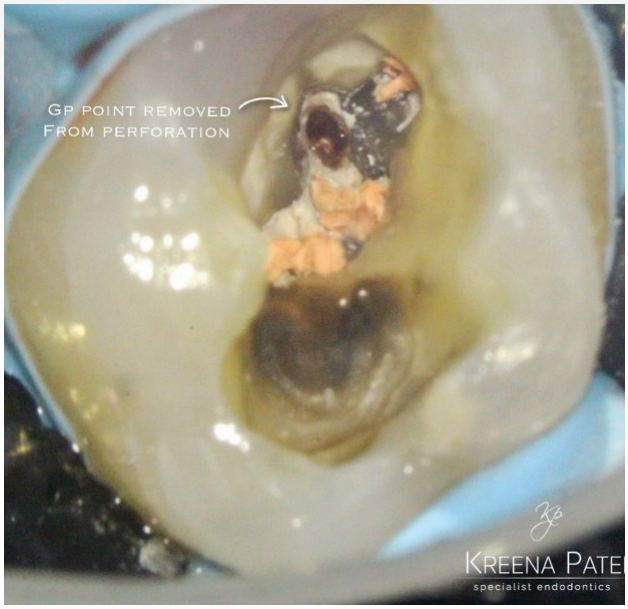


8e: Postoperative radiograph

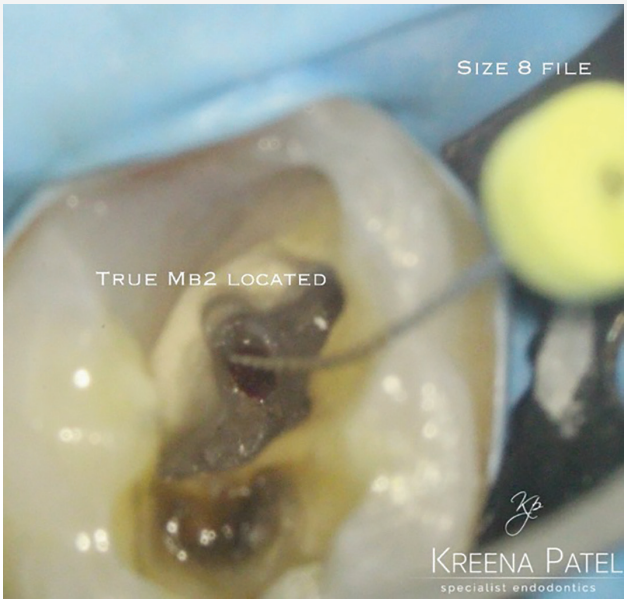




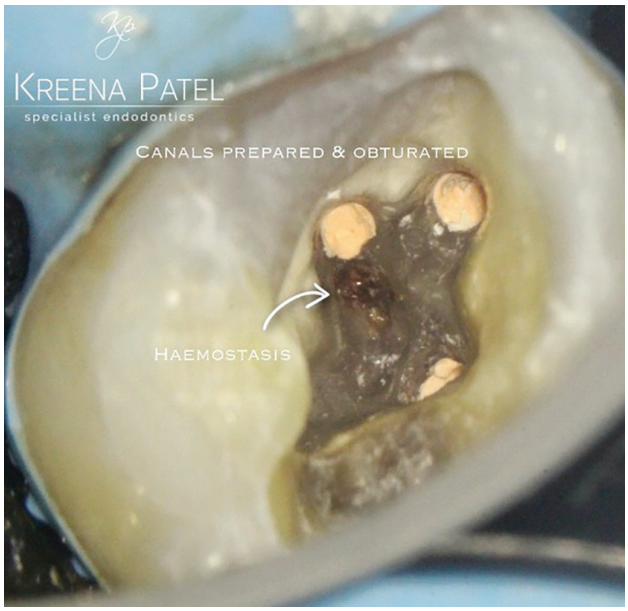
8f: Post-obturation photograph showing one MB orifice. A trough line can be seen where MB2 was searched for in its 'typical' location



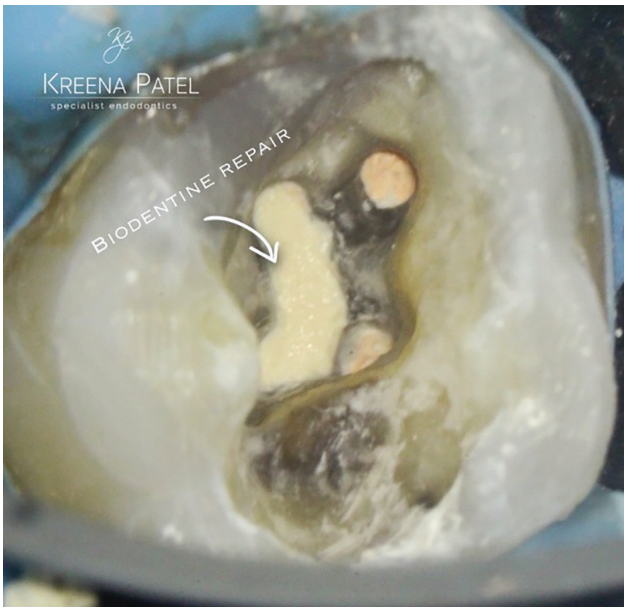
9a: Access regained: the GP point was removed to reveal the perforation on the pulp floor



9b: The true MB2 canal was located using a small hand file

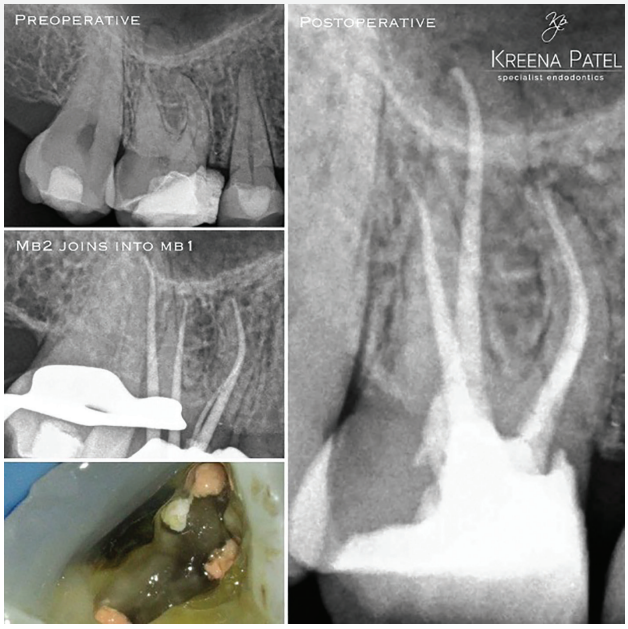


9c: Canals prepared and obturated

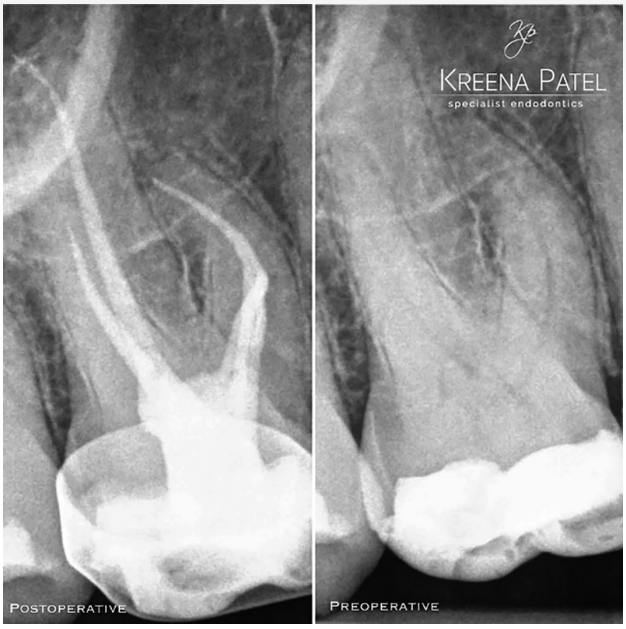


9d: Perforation repaired using Biodentine

preparation because the head of the handpiece does not obstruct your view. They also remove dentine and calcifications in a controlled manner so the preparation is more conservative. It is essential to preserve as much dentine as possible because it is quite easy to perforate the mesial wall or pulp floor when searching for MB2 (Figure 9).



10a: Master cone shows MB2 joining MB1



10b: MB2 joins MB1 at an acute curvature (white arrow)

Instrumentation

I strongly advise fully preparing the MB1 canal prior to locating and instrumenting MB2. The MB2 often joins MB1 at a sharp angle, and it makes negotiation much easier if the MB1 is already enlarged (Figure 10).

Instrumentation of MB2 can often be difficult. The canal often has an abrupt mesial curvature in the coronal 1-3mm making it challenging to negotiate initially. I find that spending time initially gaining correct access and coronal flaring to gain straight line access can prevent ledges, which once formed are very difficult to bypass. Rotary orifice openers used in a brushing motion away from the furcation are very useful for this.

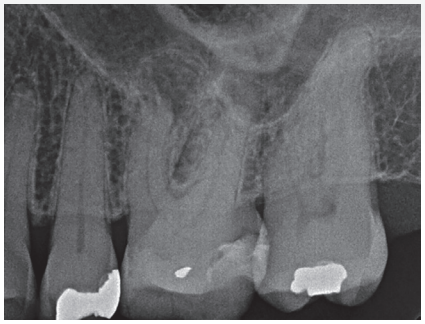
Small hand files (size 8-10) can then be taken further into the canal and a glide path formed prior to using rotary instruments. If at any point the hand file meets resistance the canal can be flared up to this point prior to taking pre-curved hand files back into the canal. The MB2 can be calcified in extensively restored teeth and older patients. Stiffer hand files and 17% EDTA solution can be very useful for these situations but care needs to be taken as it is also easy to ledge the canal using these.

The MB root is a small root and a more conservative preparation of the MB2 is necessary to prevent unnecessary weakening and fracture long-term. I would recommend using a slightly less tapered (6%) rotary file for the final preparation. Some clinicians advise preparing MB2 to the level it joins MB1 to conserve dentine. However, in my experience I find the MB2 frequently joins MB1 but then separates into its own canal apically

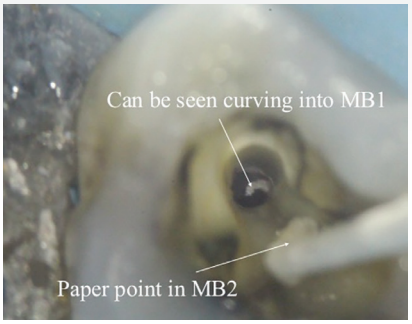
(2-1-2 morphology) (Figure 11). Therefore, I would recommend always preparing the MB2 to full length. Obturation of these complicated systems presents its own challenge and can only be done effectively using a warm technique.

Summary

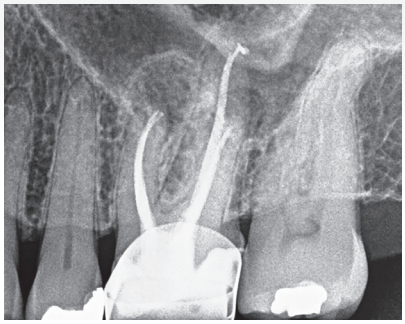
Successful root canal treatment involves treating all the canals present. The MB2 has been shown to be present in most maxillary molars and the clinician should approach treatment of these teeth with this in mind. The MB2 is notoriously challenging to locate and negotiate, but with the correct magnification, light, equipment, knowledge and experience it can be treated predictably.



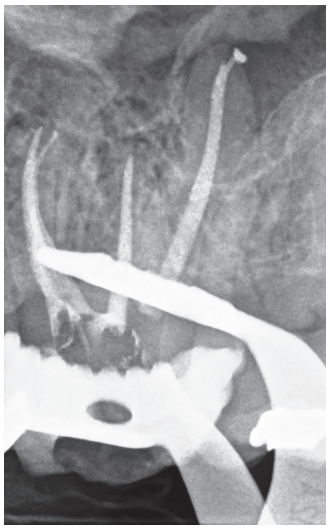
11a: Preoperative radiograph



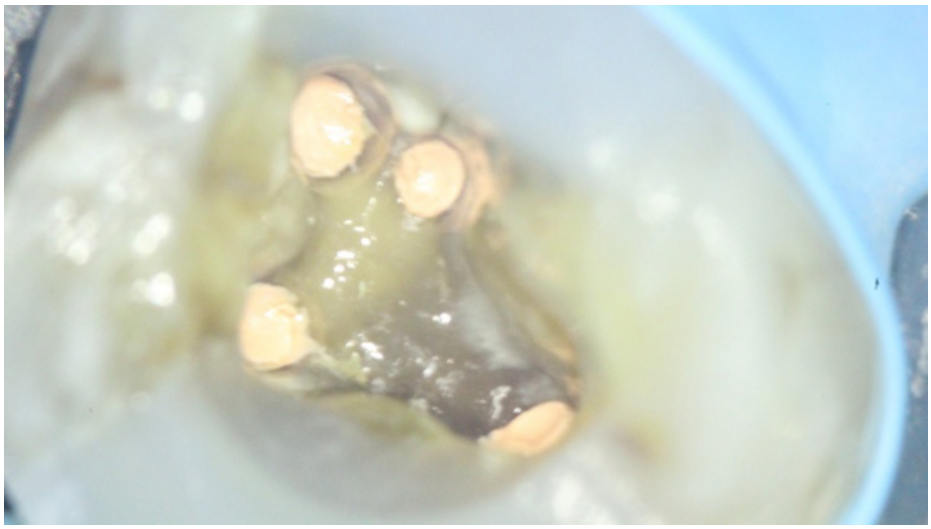
11b: A paper point has been placed in MB2, and can be seen appearing in MB1 where the canals join



11c: Mid-fill radiograph showing the MB2 has a separate apical foramen from MB1. The MB1 &MB2 have a 2-1-2 morphology (2 orifices-merging into 1 canal – separating into 2 canals). The MB2 should always be fully instrumented because this anatomy is difficult to predict



11d: Postoperative radiograph



11e: Microscope photograph

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Krasner P, Rankow HJ (2004) Anatomy of the pulp-chamber floor. J Endod 30(1):5-16 Parker J, Mol A, Rivera EM, Tawil P (2017) CBCT uses in clinical endodontics: the effect of CBCT on the ability to locate MB2 canals in maxillary molars. Int Endod J 50(12):1109–1115

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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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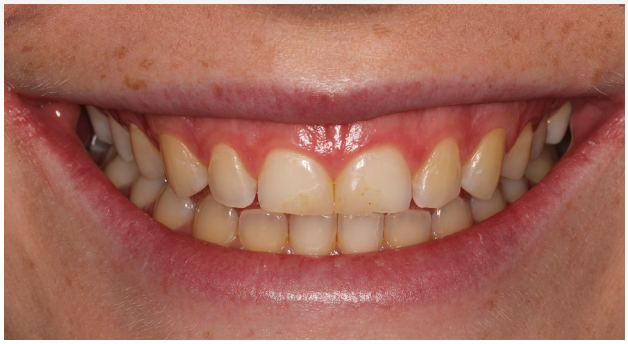

Figure 1: Gummy smile due to Altered Passive Eruption.

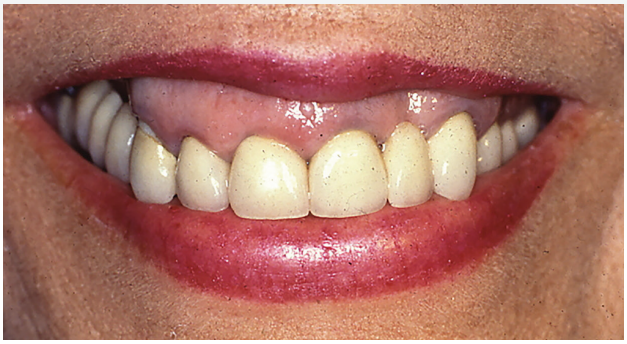

Figure 2: Asymmetric gingival contour.

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

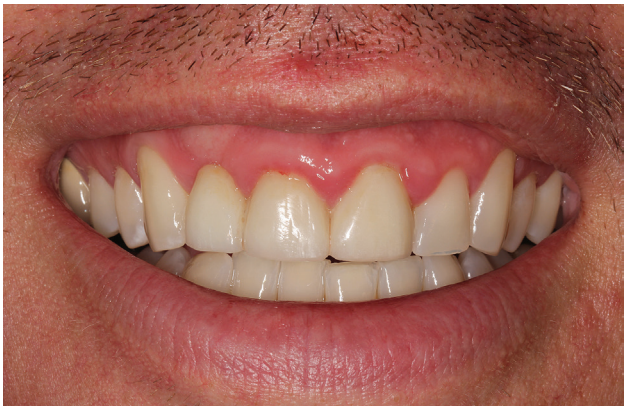

Figure 3: A gummy smile due to vertical maxillary excess where teeth are almost fully exposed.

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

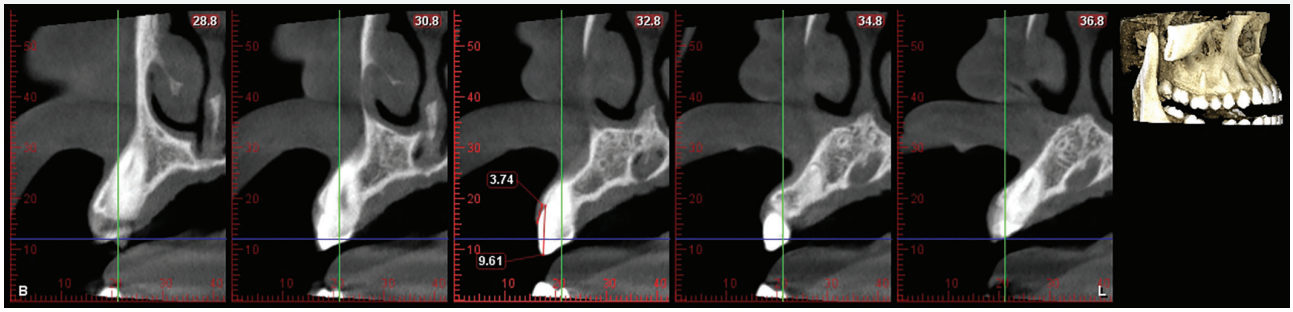

Figure 4: Soft tissue retraction allows for measurement of supra-crestal gingival dimensions.

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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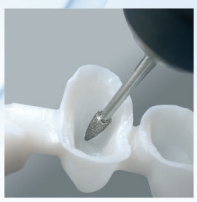

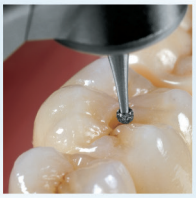

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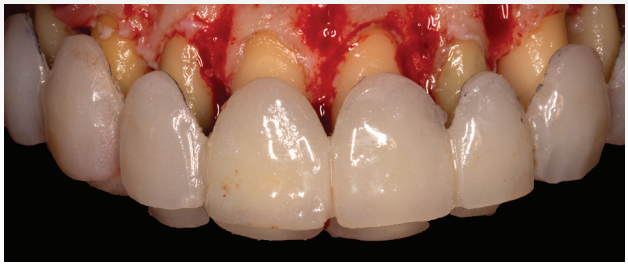

Figure 5: Surgical stent used to assess ostectomy levels.

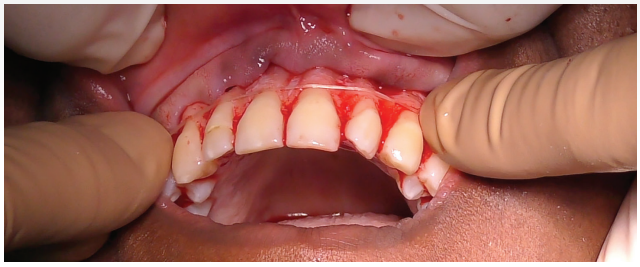

Figure 6: Floss can be cold sterilized and used intra-operative to assess symmetry in bone levels.

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):

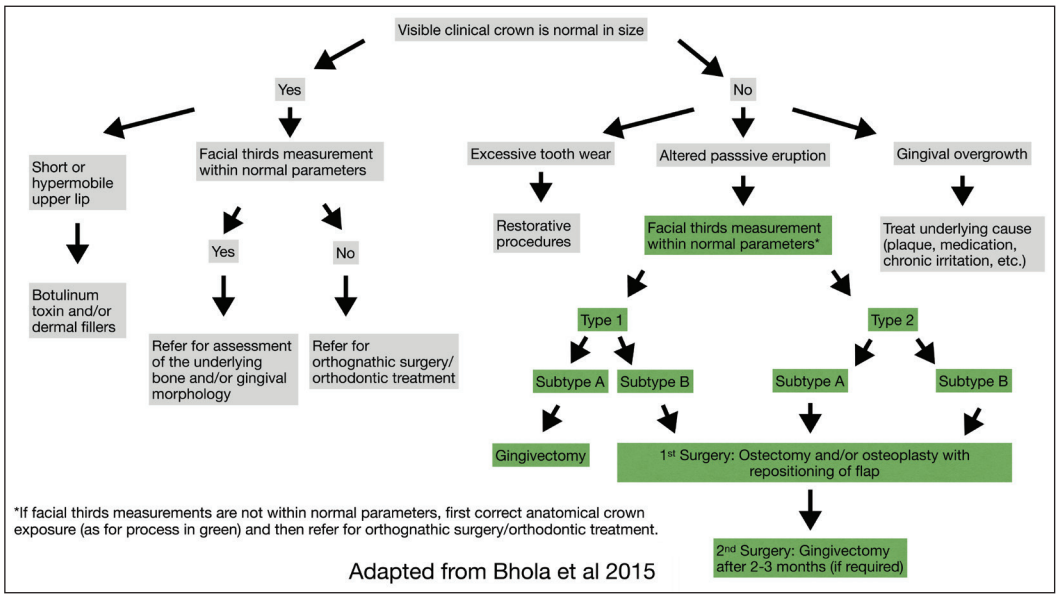

Figure 7: Decision tree for diagnosing excessive gingival display and selecting appropriate treatment.

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

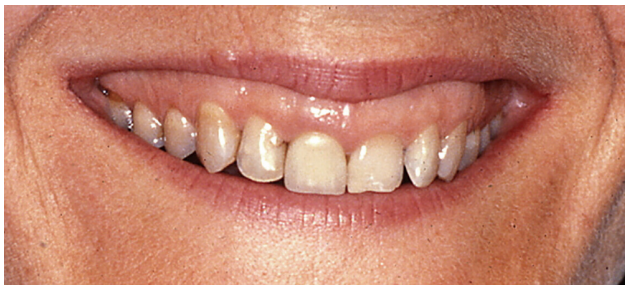

Figure 8: Altered passive eruption with a component of vertical maxillary excess, showing incomplete exposure of clinical crowns.

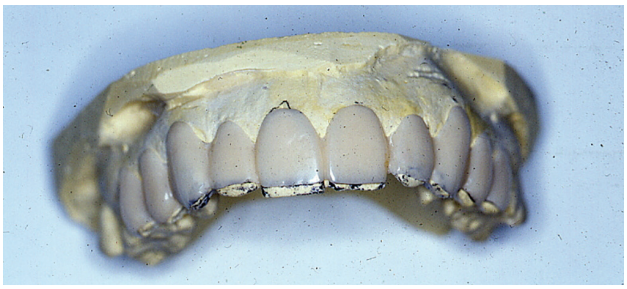

Figure 9: Surgical stent to indicate the new planned clinical crowns.

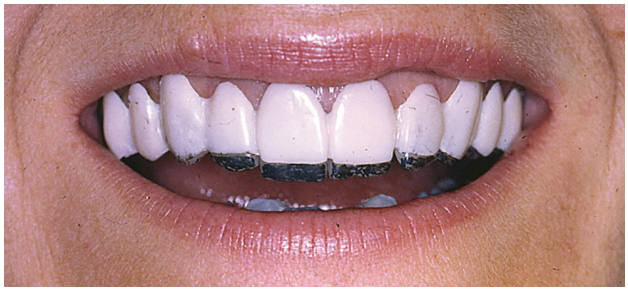

Figure 10: Surgical stent placed intra-orally with incisal edges blocked out using a black pencil.

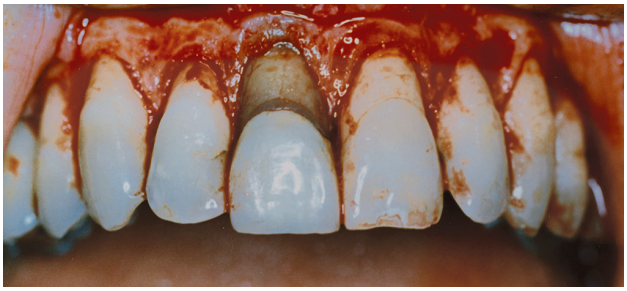

Figure 11: Full flap reflection with partial thickness interdentally, allowing access to buccal and interdental bone for ostectomy and osteoplasty.

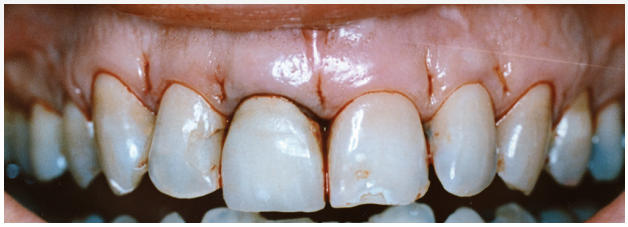

Figure 12: Closure of flap with vertical everting mattress sutures to allow for maximum soft tissue fill in embrasure spaces.

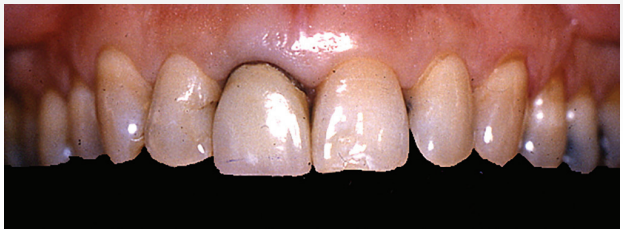

Figure 13: Healing after 3 months showing 2-3mm of recession in a symmetrical pattern, following the bone contour.

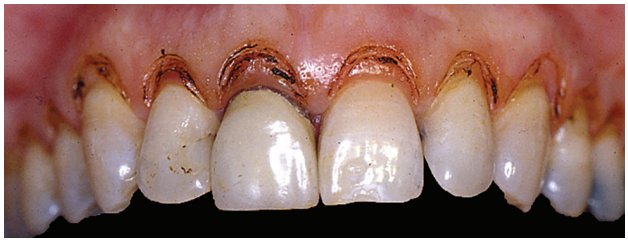

Figure 14: Second stage gingivectomy after 4 months.

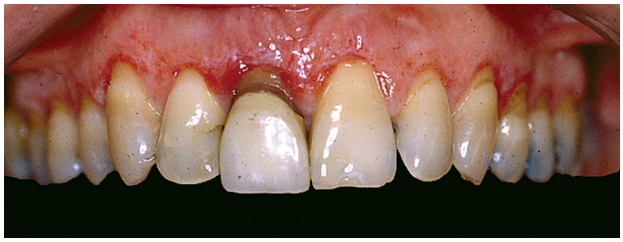

Figure 15: Two weeks after second stage surgery, showing the extent of lengthening.

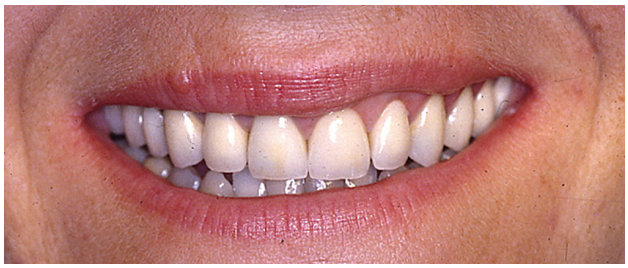

Figure 16: Six months after placing final crowns.

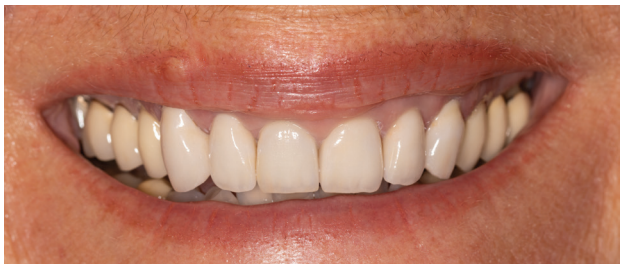

Figure 17: 25 Years after surgery, showing stable gingival margins.

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.

Acknowledgements

The authors would like to thank Dr. Callie Hamman, Prosthodontist, private practice, Durbanville, Cape Town for the prosthodontic concept, stent and crowns of the Case Report. as well as Dental Technician: Ian Robertson, Bellville, Cape Town

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Stratified layering of composite restorations after the use of orthodontic aligners

Linda Greenwall[1] and Robert Katz[2]

There is a trend towards minimally invasive aesthetic dentistry and ensuring that no healthy enamel is cut in the preparation of an enhanced smile.

This case illustrates the use of orthodontic aligners, whitening treatment in the aligners, and composite bonding using a stratified layering technique and the placement of glass ionomer restorations on the cervical erosion areas.

The use of aligners in orthodontics

The use of removable aligners has increased greatly over the last 18 years. In 1999, Align Technology addressed the demand for an aesthetic alternative to braces by developing an 'invisible' method of orthodontic treatment (Invisalign) that uses a series of computer-generated, clear, removable aligners to move the dentition (Kunicio et al, 2007). Align Technology reports that, since then, more than four million Invisalign cases have been undertaken worldwide.

Aligner popularity has increased in adult patients who do not want to wear fixed braces, as they find them more difficult to tolerate, due to their effect and impact on daily life (Bernabe et al, 2008). The simple idea that a clear aligner can be used to align and reposition teeth is appealing to adult patients.

Patients can remove the aligners for eating, brushing, flossing and important meetings, but can wear the aligners for most of the day (Joffe, 2003).

The aligners are usually comfortable and offer ease of use. They are made of polyurethane and are normally 0.75mm thick. Patients are asked to wear the aligners for two weeks and then change to the next number in the sequence of aligners.

Patient assessment

The orthodontist will normally undertake a full assessment of a new patient. Treatment options are enumerated and discussed.

While fixed orthodontic braces may move the teeth more predictably and quickly, many patients do not want to wear braces. They want the effects of the treatment without having fixed braces. It is often the preference of orthodontists to undertake fixed appliance therapy because it can be more predictable and, in some cases, the teeth can move quicker. However, patients are given all the treatment options to align their teeth and many choose to have clear aligners. There are now several aligner brands on the market that the orthodontist can choose.

A recent systematic review of Invisalign research by Lagravere and Flores-Mir (2005) found that no strong conclusions could be made regarding the treatment effects of Invisalign appliances. It is the personal selection of the orthodontist and their patient.

[1] Dr Linda Greenwall, Specialist - Prosthodontics and Restorative Dentistry, London, UK

[2] Dr Robert Katz, Specialist - Orthodontics, London UK

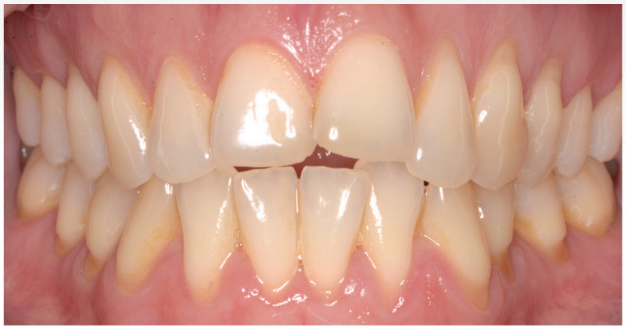


Figure 1: Retracted smile before treatment commenced. The patient was unhappy with the overlapping of the anterior teeth and wanted the teeth to be whiter.

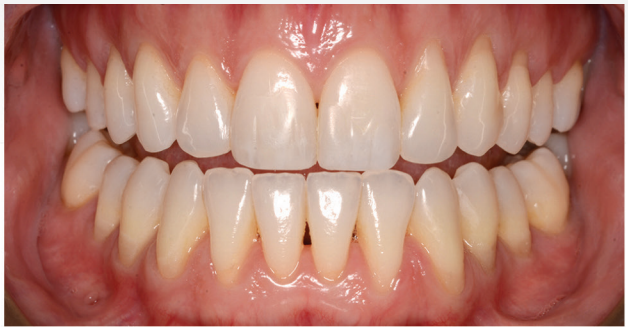


Figure 2: The results after Invisalign, whitening and restorative bondings.

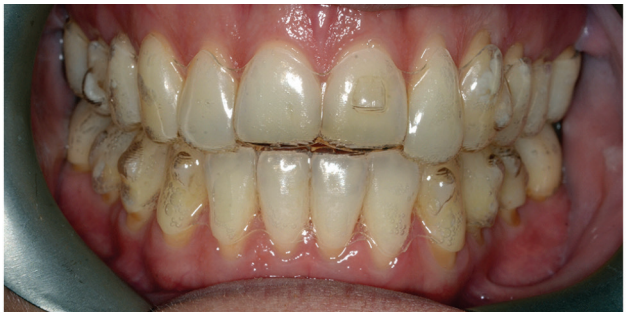


Figure 3: Patient commenced upper and lower Invisalign treatment with orthodontist Dr Katz to improve the positioning of the teeth.

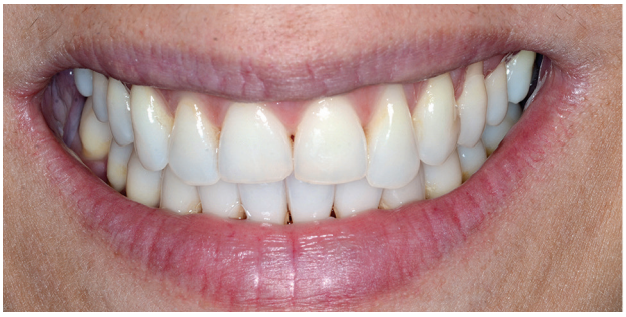


Figure 4: While wearing the Invisalign aligners, the patient bleached with 10% carbmide peroxide to improve the shade of her teeth. This photo illustrates the whitening gel has started to work and starts on the incisal edges first and moved up to the neck of the tooth.

The computerised Clincheck

With Invisalign treatment, the number of aligners needed is assessed with a computer scan called Clincheck (Align Technology).

Each aligner is programmed to produce a precise movement on a tooth of about 0.15- 0.25mm (Vlaskalic et al, 2001).

The stereolithographic technology is used to fabricate custom aligners from an impression or an intraoral digital image scanned in the dental practice.

Patient compliance is mandatory to achieve good results with Invisalign. It is important for patients to wear their aligners for 22 hours a day or more (Malik et al, 2013).

Once the Clincheck is undertaken, the number of aligners needed is calculated and the position and location of the attachments determined. The attachments are fabricated from clear composite resin and are transferred onto the teeth with an attachment template. The attachments are removed at the end of treatment. Where interproximal enamel reduction (IPR) is necessary, this is calculated in the Clincheck.

Studies have been undertaken to assess the accuracy of the computerised Clincheck assessment. In a study by Houle et al (2017), the mean accuracy of posterior expansion planned with Invisalign for the maxilla was 72.8% and 87.7% in the mandible.

There are limited data on the amount of discrepancy between predicted and actual achieved movements with Invisalign (Krieger et al, 2012). In a prospective clinical study by Kravitz et al (2009), the mean accuracy of tooth movement in the anterior region was found to be 41% with Invisalign.

An internal study from Align Technology found that one should expect about 80% of tooth movement seen on Clincheck (Tuncay and Orhan, 2006).

A multidisciplinary case – which treatment first?

This case involved multidisciplinary treatment including orthodontic treatment, restorative treatment and aesthetic

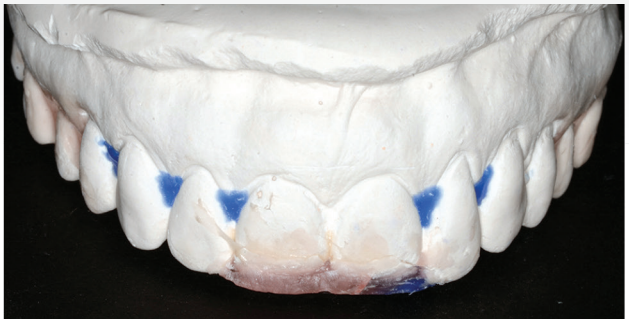


Figure 5: Study models used to make the retainer after Invisalign treatment was completed. The aligner had to be fitted immediately after treatment started and before undertaking the restorative bonding to lengthen the upper right anterior. The technician made a stent so we knew how much length we needed to add.

Figure 6: SDI Aura is applied onto the tooth to check the shade of the composite against the tooth after whitening. A test composite is placed onto the tooth at the very beginning of the bonding procedure prior to isolation so that the correct shade of composite is selected before the tooth dehydrates to a lighter shade. The translucent enamel shades are tested first. Here Aura E1 and E2 are being tested onto the transluscent incisal tip

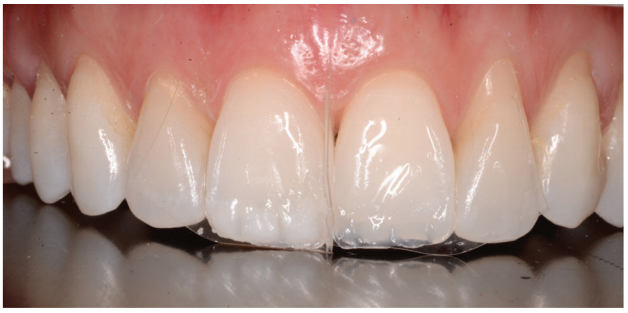


Figure 7: SDI Aura is built-up in layers to look like natural enamel, starting with the placement of the enamel layer on the incisal and palatal edge. Lobes are created to give the effect of the mamelons and the transluscency at the tip. The lobes also help to determine the secondary anatomy and correct form and shape. The composite is always over built and the restoration reshaped and polished afterwards.

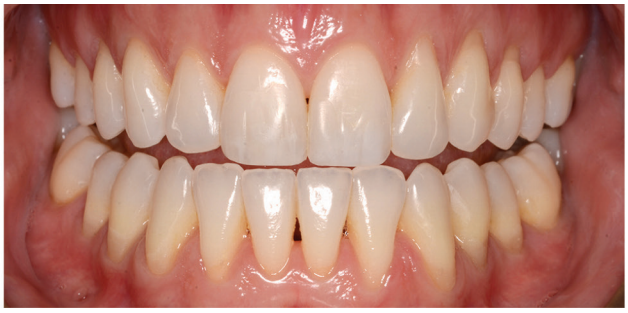


Figure 8: The results after Invisalign, whitening and restorative bondings with SDI Aura on the upper right and left centrals. The composite is shaped and polished with discs, rubber wheels and then final polish with felt wheels and SDI polishing paste for the enamel lustre. As the Aura has the enamel as a microfill it can be polished to a high gloss afterwards. The dentine layer is a nanohybrid.

home whitening treatment (Figures 1 and 2).

Invisalign treatment was commenced first (Figure 3). After the teeth had moved significantly, when the central incisors had straightened and towards the end of treatment, tooth whitening was undertaken in the upper and lower aligners (Figure 4).

Once whitening was satisfactorily completed, new retainers were made from new study casts (Figure 5). Composite bonding was undertaken (Figures 6-8) to repair the worn and shorter incisal edges of the upper central incisors. This was followed by glass ionomer treatment placed in the lower cervical areas to reduce sensitivity.

Normally, class V glass ionomer restorations are placed first prior to commencing any treatment as this helps to reduce sensitivity during whitening and also reduce sensitivity of the orthodontic tray rubbing against the cervical area of the tooth.

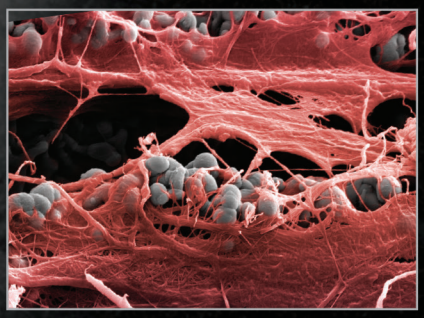
However, it was decided that the erosion of the cervical areas was not an area of concern and so Invisalign 'Each aligner is programmed to produce a precise movement on a tooth of about 0.15-0.25mm' treatment was started first.

Whitening treatment was next followed by composite bonding and class V restorations. The cervical areas of the lower premolars became extremely sensitive during whitening, so after whitening and waiting for the bond strength to improve, the areas were restored with a light-cured glass ionomer restoration.

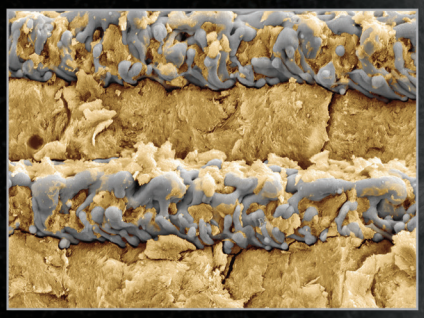
better science, better implants

is osseointegration enough?

Only the Laser-Lok surface has been shown using light microscopy, polarized light microscopy and scanning electron microscopy to also be effective for soft tissue attachment. [1,2]



Colorized SEM shows connective tissue physically attached to the Laser-Lok surface.[1]



Colorized SEM of Laser-Lok® microchannels shows superior osseointegration[3]

interact at laser-lok.com

1. Human Histologic Evidence of a Connective Tissue Attachment to a Dental Implant. M Nevins, ML Nevins, M Camelo, JL Boyesen, DM Kim. International Journal of Periodontics & Restorative Dentistry. Vol. 28, No. 2, 2008. 2. Histologic evidence of a connective tissue attachment to laser microgrooved abutments: a canine study. M Nevins, DDS, DM Kim, DDS, DMSc, SH Jun, DDS, MS, K Guze, DMD, P Schupbach, PhD, ML Nevins, DMD, MMSc. Accepted for publication: IJPRD, Vol 30, No. 3, 2010. 3. Maintaining inter-implant crestal bone height via a combined platform-switched, Laser-Lok® mplant/abutment system: A proof-of-principle canine study. M Nevins, ML Nevins, L Gobbato, HJ Lee, CW Wang, DM Kim. Int J Periodontics Restorative Dent. Volume 33, Number 3, 2013.

SPMP17297 REV A OCT 2017

Orthodontic treatment

In this case, the patient's main concerns where the uneven smile and the shortened upper central incisor teeth.

The characteristics of the malocclusion were as follows:

- Class I molar and canine occlusion
- 5mm overjet
- Proclination of the upper and lower incisor teeth
- Mild lower anterior crowding
- Upper incisor irregularity.

After a new patient consultation and a treatment planning discussion where all the options were discussed with the patient, she elected to have an orthodontic assessment to explore the options to move and align the teeth.

She was presented with two options, that of fixed braces or aligning treatment.

She requested that aligning treatment was undertaken. PVS impressions were sent to Align Technology for conversion into 3D study models using the company's software.

The Clincheck Pro software was used to modify the initial set-up (Figures 10 and 11). When finalised, 20 upper and lower aligners were prescribed, giving a treatment duration of about 10 months (Figures 12 and 13). IPR, totalling 1.2mm in the lower arch and 2.4mm in the upper arch, was prescribed. IPR was necessary to make space to correct the crowding the decrease the incisor protrusion.

Treatment proceeded as expected with no complications, or need for refinement. The patient was compliant and wore the aligners 22 hours per day.

When treatment was finished and the restorative treatment completed, fixed retainers were bonded upper (palatal) and lower (lingual) on the incisors and canines.

The retention phase

Normally, the final aligner acts as the retainer in most cases of Invisalign treatment.

Despite extensive research, the various elements leading to relapse of treated malocclusions are not completely understood, which makes retention one of the most challenging aspects of orthodontic treatment (Kuncio et al, 2007).

However, in this case, immediately after composite bonding, the upper retainer was cut in the incisal area to allow for the increased incisal length. New impressions were taken after completion of the restorative treatment and new retainers were made to ensure that the occlusion was well maintained. It is essential to stress that patients should wear their Invisalign retainers as prescribed by the orthodontist to ensure stability of the occlusion and correct alignment of the teeth.

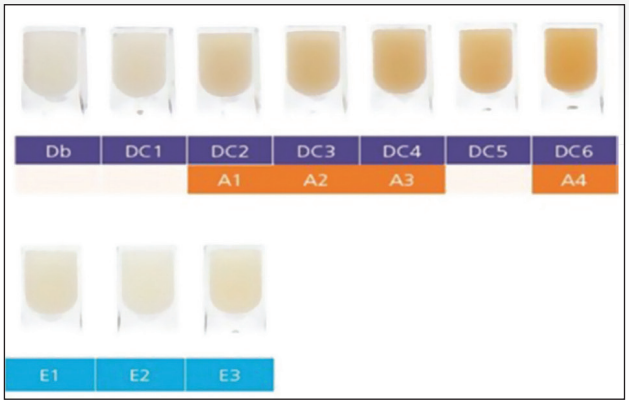


Figure 9: Shades of Aura composite.

In a comparative study of the retention of fixed braces versus Invisalign retainers, Kuncio et al (2007) found that, in many cases, aligner treatment can relapse more than fixed braces, and so patients are instructed to continue to wear their retainers for maintenance treatment.

However, the total number of patients in each group was 22, which is a very small number and further research is necessary.

Whitening in Invisalign aligners

Using the Invisalign aligners as whitening trays has become a viable treatment option for patients.

The recommendation is to wait for a month after the first aligners are placed and after the initial discomfort has settled down. The patient applies the whitening gel directly into the aligners.

A similar Van Haywood protocol can be adopted, namely to whiten the upper teeth first followed by the lower teeth.

The upper teeth whiten quicker and have fewer side effects and so the first stage of whitening is relatively simple. The lower whitening normally takes longer. It is thought that this is due to the wash-out effect of the salivary glands.

Due to the rigid nature of the aligner, it seems that whitening may occur quicker than a normal bleaching tray, but this has not been studied significantly.

Most aligners do not reach over the gingivae, so the gingivae may be less irritated during whitening.

There is no effect of whitening around the orthodontic attachments, and the whitening occurs in a multidirectional way and can whitening around the attachments.

Whitening and composite bonding

It is essential after completion of home whitening treatment

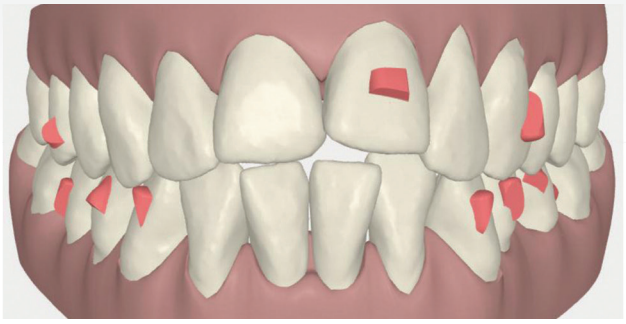


Figure 10: Initial centre view using Clincheck software.

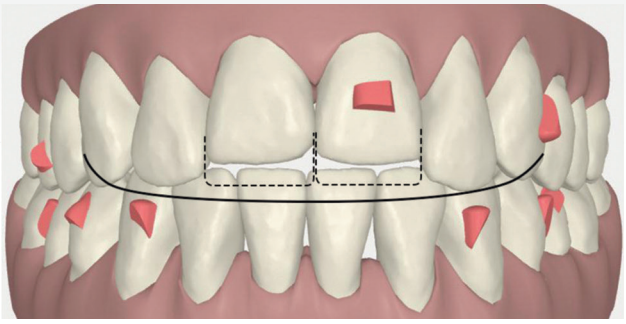


Figure 11: Final centre view.

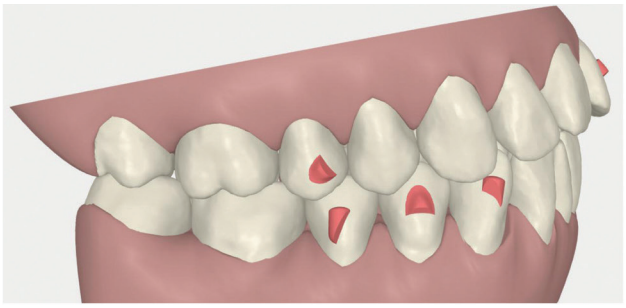


Figure 12: Initial right buccal view.

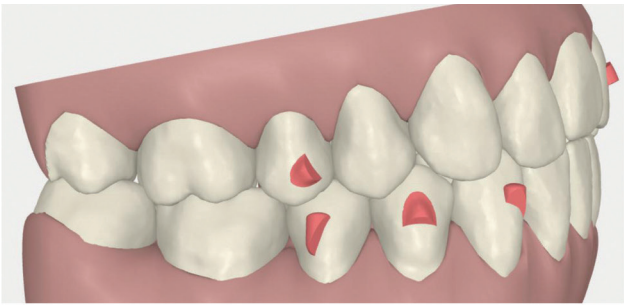


Figure 13: Final right buccal view.

to wait for a period of two weeks to allow all the oxygen to be dissipated from the tooth and for the shade to settle to the actual shade.

After tooth whitening, there is maximum saturation of oxygen inside the enamel. This causes a reduction in bond strength of 20%. It is thus essential to wait for two weeks for the enamel bond strength to recover back to the normal levels prior to commencing direct bonding onto the surface of the tooth.

Stratified layering technique

With the introduction of multiple component composite systems, it is now possible to create beautiful natural restorations using multiple layers of composite using their different optical and material properties. The Aura composite system is ideal as it contains both enamel and dentine shades (Figure 9).

The enamel shades are a microfill composite, which give the properties of a glass-like appearance of natural enamel (Figure 5). The dentine shades are a nanohybrid material (Figure 6), which gives extra strength for occlusal build-ups and they can be used as a bulk-fill material. There are separate bulk-fill syringes available for this purpose.

Shade selection of composite resin

According to Vanini (1996), it is essential to undertake a detailed evaluation of hue, chroma, opalescence, and fluorescence of the tooth in order to simplify the composite stratification technique.

This is done early in the clinical procedure to ensure that there is no dehydration of the tooth when the tooth is fully isolated (Figure 5).

Once the tooth is fully isolated, it dehydrates and lightens and this can result in the selection of a shade that is too light. Blends of composite colours are normally used and, after selection of the transluscent enamel shade (Dietchi, 2008), the dentine shade is used (Figure 6).

The hue is given by the dentine. The hue remains the same, although the greater thickness of the enamel interferes in its perception, giving it a less saturated aspect. Therefore, the hue of the tooth is given by the dentine and influenced by the enamel. The enamel does not change the hue, but only

confers a greater or lesser saturation or chroma according to its thickness (Franco et al, 2007).

This is applied from darkest to lightest to give the restoration a lifelike appearance. Different translucencies may be selected for the mesial corners as, often, these are more translucent than the distal corners (Figure 7).

Used with an understanding of tooth morphology, restorative material selection, colour options, and the physical properties of light, these layering techniques allow optimally aesthetic restorations to be predictably achieved (Terry, 2003).

Applying the composite resin

A test run is undertaken first using a variety of composite shades. A clinical photograph is taken after the first test to review how the shades appear on a digital photograph. A polarised light photograph can also be taken to understand the nuances of the existing anatomy of the tooth, which needs to be copied.

As the teeth have been bleached, the bleaching composite shades can be used. In Aura composite, the DB shade blends very well to the bleached enamel (Figure 8). The enamel shades are tested first and light-cured followed by the dentine shades.

Placing the layers

Normally, the tooth is built-up from the palatal part first. A clear matrix is adapted to help form the shape of the missing incisal edge.

A wax-up can be used and a silicone stent can be made for ease of placement and for patient and dentist visualisation of the final outcome. The clear matrix is curved and rolled in the operator's gloved hand to form a curve. This is placed on the missing incisal edge and bonded into place using a dentine bonding agent. This helps to keep the hands free so that the layering of the composite can commence.

The layers are placed into the area with a flat plastic tool and then sculpted into place and refined with a fine haired brush dipped into bonding liquid or a rubber sculpting instrument.

The tooth is built-up in layers and light-cured (Figures 6 and 7). Each layer is checked after placement and modifications made as the restoration takes shape. Once the layering is completed, the final form is created with finishing instruments.

Sof-lex polishing discs (3M Espe) are used first to remove any bulk excess and then fine flame finishing burs are used.

After the form is completed, the patient is asked to sit up so that the incisal edges can be checked from the front of the patient. When the dentist is in a working position behind the patient, there is tendency to build the incisal edge restorations too long. This final length of the incisal edge is checked when the patient is supine in the chair.

The occlusion is checked and the final polishing can commence with the use of rubber wheels and a felt tip rubber wheel and polishing paste. The final glossy layer is created with a special rotary rubber wheel and polishing paste.

Conclusion

A multidisciplinary case such as this requires essential communication between the specialists undertaking the treatment, and also between the dentist and the patient, so that the patient is fully aware of the risks and the benefits of the different treatment procedures, and what is involved in each treatment so that an ideal outcome can be achieved. The patient also needs to be fully aware of the retention phase needed to maintain the teeth in the same position, and to maintain a beautiful smile and when further whitening may be needed. In addition, any repairs to composite need to be detailed and occlusal checks need to be made regularly to maintain a beautiful smile. From time to time, during recall appointments, further polishing of the composite may be required, including retention recall evaluations.

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COVID-19 risk management in dental practice: The infection chain pathway of SARSCoV-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, spreading fast with an increasing number of infected patients world-wide. The current global statistics on March 22, 2021, show the total number of confirmed cases 123,868,982, total deaths 2,727,738 and total recovered /discharged 99,795,152 individuals. Current global active infected cases 21,346,092 (17,2 %) of which 21,255,883 (99,6%) have a mild condition and 90,209 (0,4%) are serious / critical.1 The COVID-19 statistics for Australia on March 22, 2021 showed a total number of 29,205 and New Zealand 2,462 cases; total deaths: Australia 909, New Zealand 26 and total recovered: Australia 26,243, New Zealand 2,373 cases. Current active COVID-19 cases on March 22, 2021 were: Australia 2,053, New Zealand 63 of which none 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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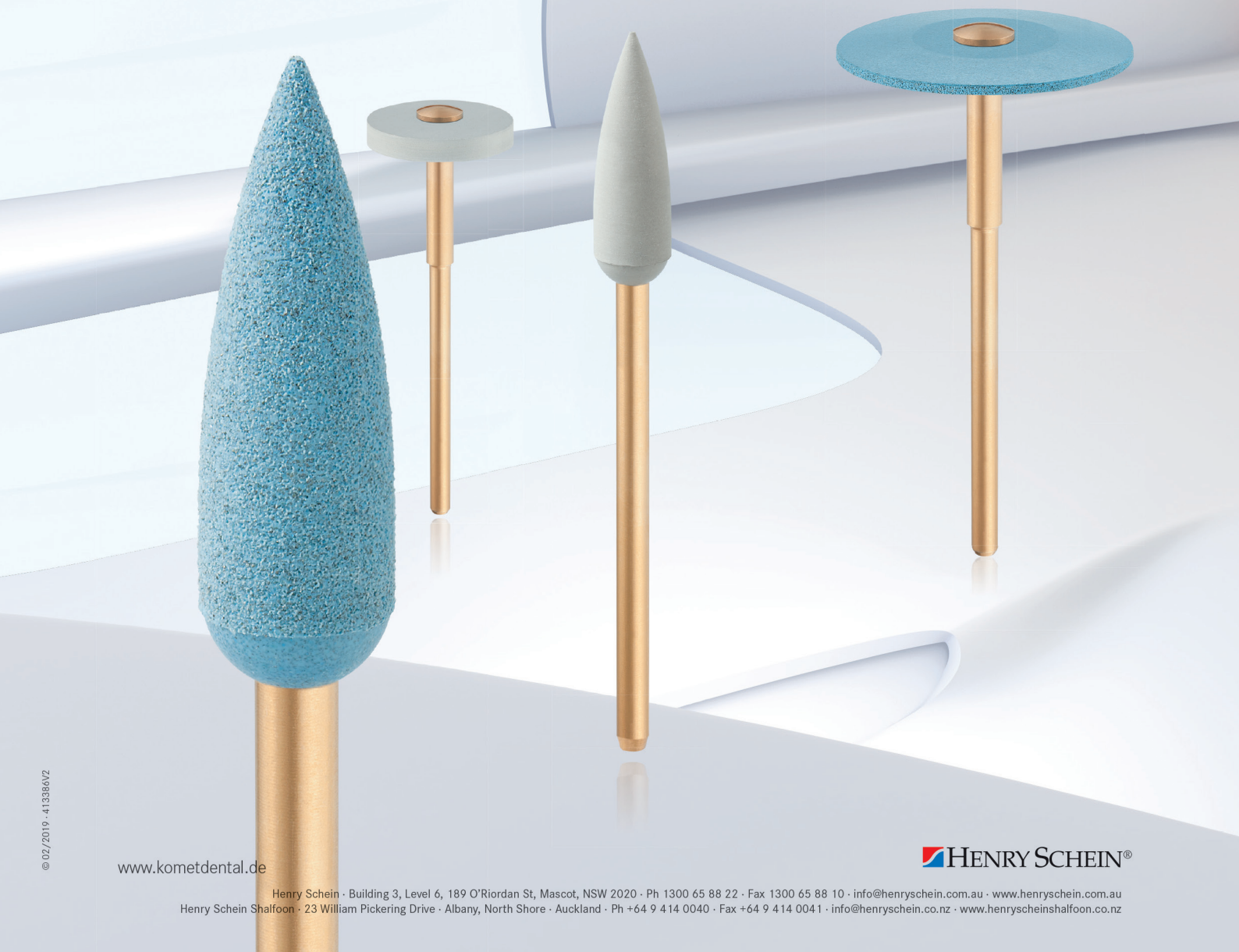

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Table 1 : The infection chain pathway from pathogen to host and appropriate infection control strategies to mitigate or contain transmission of the coronavirus between health care workers and patients in the dental practice setting

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

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

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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]

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- ***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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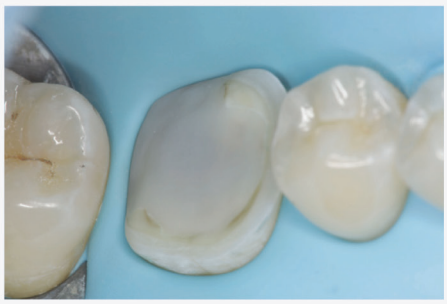
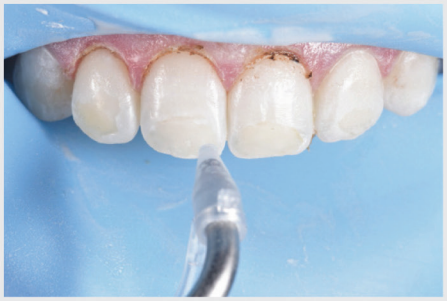
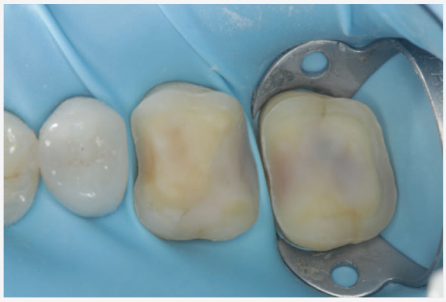


Air abrasion with AquaCare: 29µm aluminium oxide prior to onlay bonding.



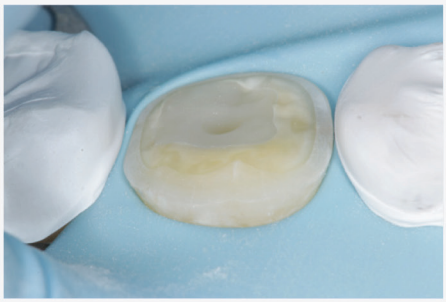
Removal of old composite bonding using 29µm alox.

Minimally invasive and respectful of natural tooth structure, allowing for a clean surface to rebond to free of existing restorative material.



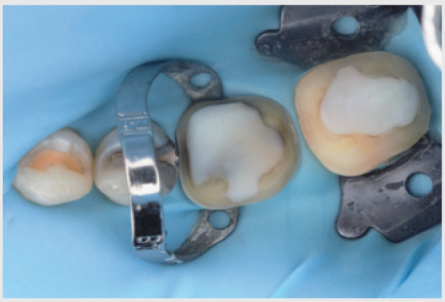
Air abrasion with AquaCare: 29µm aluminium oxide prior to onlay bonding.

Veneer Bonding facilitated by 29µm alox and Sylc surface preparation.



Air abrasion with AquaCare: 29µm aluminium oxide prior to onlay bonding for a root treated tooth.

Dr. Yo-Han Choi BDSc (Hons)



Onlay surface preparation: 29µm alox + Sylc

Dr. Bharat Agrawal

MSc Aesthetic Dentistry (King's London)
PGDip(AdvancedAesthetic&RestorativeDentistry)
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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.

References

References 1 - 157 are available on request at dentsa@iafrica.com or www.moderndentistrymedia.com

Posterior restoration with a universal adhesive and nano-hybrid composite

Tomislav Skrinjaric[1]

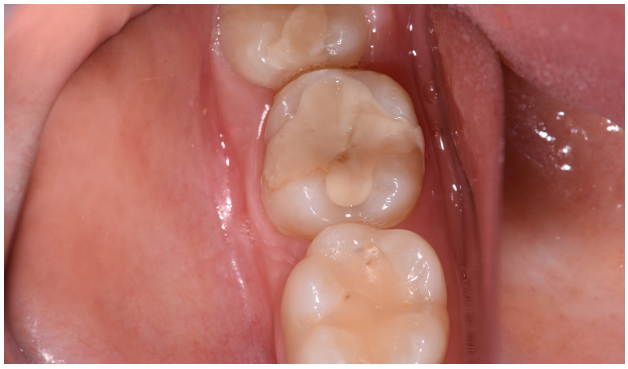

Figure 1: Initial situation: Insufficient composite filling, tooth 47.

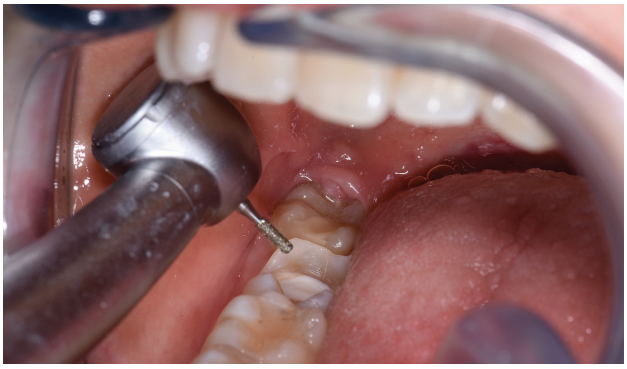

Figure 2: Removal of the defective composite filling.

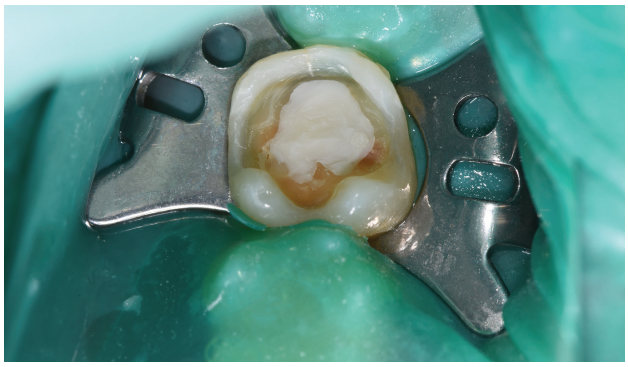

Figure 3: Cavity lining for pulp protection with Calcimol LC and Ionoseal (VOCO). For creating a dry working field is recommended the use of a rubber dam.

Figure 4: Hold the SingleDose blister between thumb and forefinger and, by pressing on the area marked "press here", so that the liquid contained in the blister flows into the mixing and dispensing chamber.

Figure 5: Put the enclosed Single Tim applicator in the centre of the coloured circle in order to pierce through the film of the mixing and dispensing chamber. By stirring thoroughly with the applicator, create a homogeneous, streak-free mixture of the two liquids.

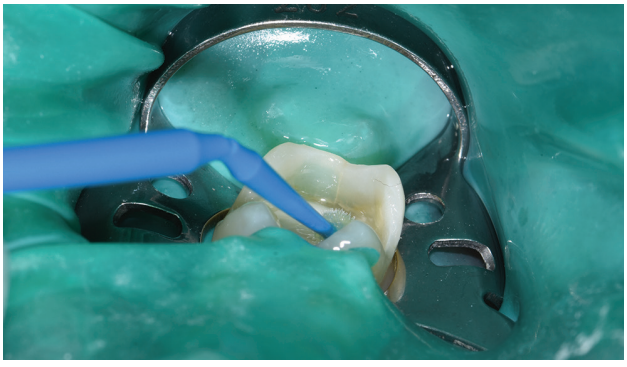

Figure 6: Apply the adhesive homogeneously to all cavity surfaces and rub in for 20 seconds using the SingleTim.

[1] Dr. Tomislav Skrinjaric, Croatia

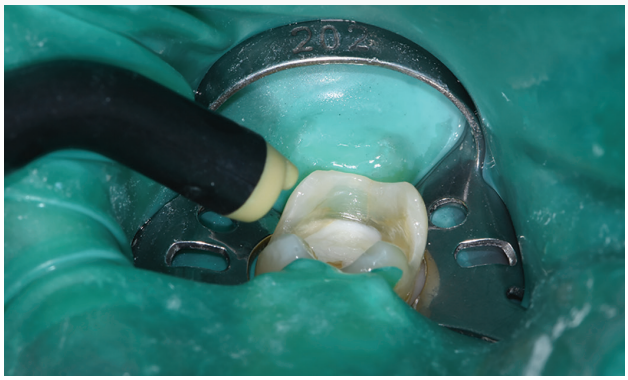

Figure 7: Dry off the adhesive layer with dry, oil-free air for at least 5 s in order to remove any solvents.

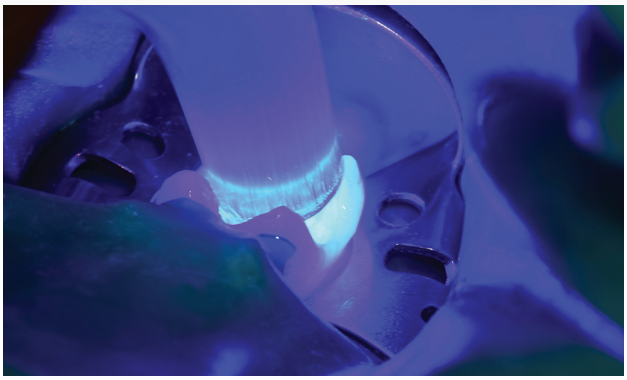

Figure 08: Cure the adhesive layer for 10 s using a commercially available polymerisation device (LED or halogen light with an output of > 500 mW/ cm²).

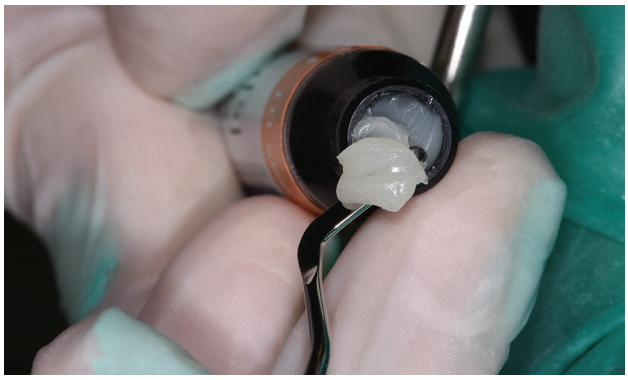

Figure 9: Taking the light-curing material for the posterior bulk filling (4mm) off the rotary syringe.

Figure 10: The light-curing posterior bulk-fill material (4mm) x-tra fil.

Figure 11: The cavity will be filled with x-tra fil (universal colour) by using the incremental technique.

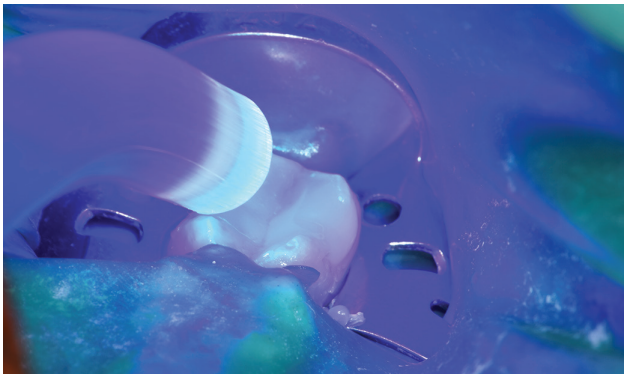

Figure 12: Polymerise each increment for 10 seconds.

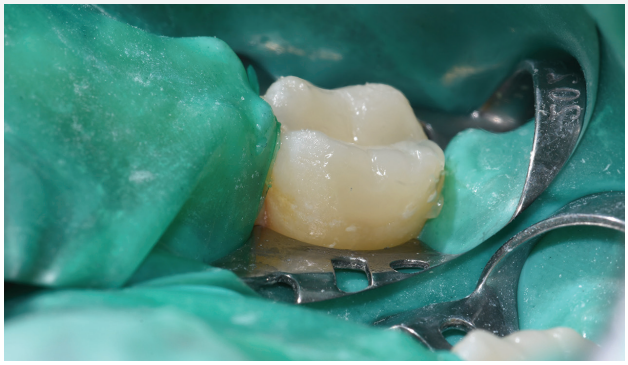

Figure 13: Filled cavity with x-tra fil before preparation.

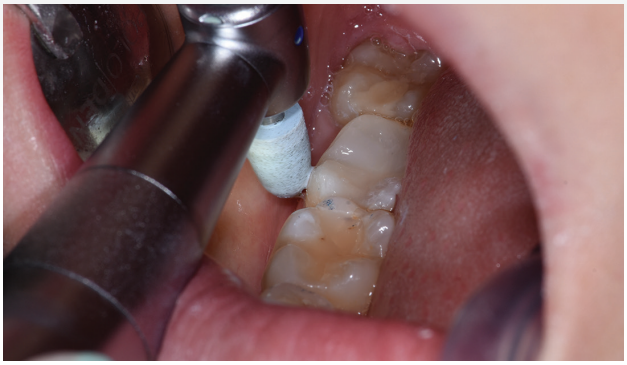

Figure 14: Finishing and polishing of the composite filling.

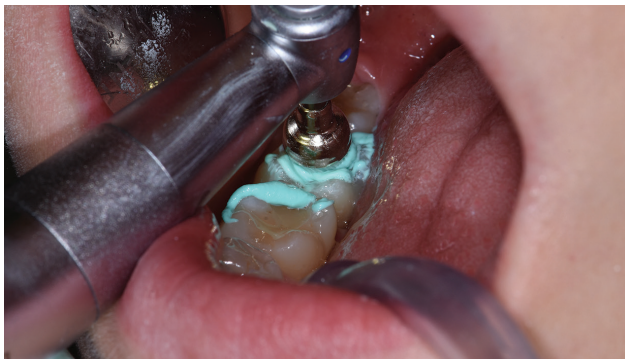

Figure 15: Finishing and polishing of the composite filling.

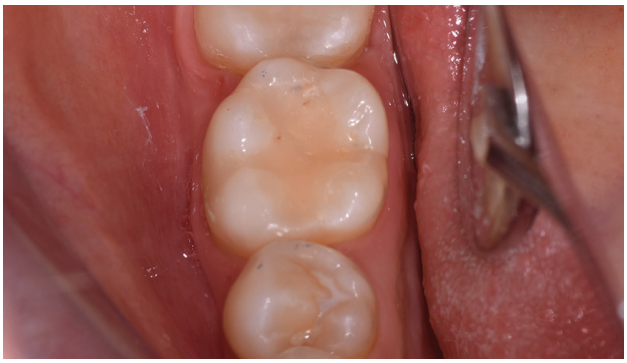

Figure 16: Final result: Finished restoration with x-tra fil, tooth 47.

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[1] GC R&D, Japan, 2020, Data on File.
[2] Yamanaka A et al. (2020), Improvement of dentin bonding effectiveness using the next generation 2-step system with a newly developed hydrophobic bonding agent, Adhes Dent, 38(3): 112
[3] Tichy A et al. (2020), A comprehensive evaluation of dentin bonding durability of a novel two-step self-etch adhesive, Adhes Dent, 38(3): 109.

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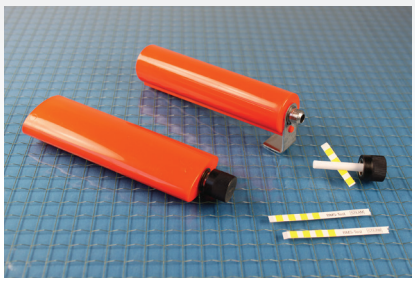

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SURFACE DISINFECTANT WIPES

Henry Schein Surface Disinfectant Wipes are a premium quality wipe soaked in a cleaning and broad-spectrum disinfection solution. Developed to reduce cross contamination in healthcare environments, they provide fast acting disinfection including activity against SARS-CoV-2 (Covid 19) in 2 mins. The wipes are absorbent, lint free and textured to aid cleaning. For use on hard, non-porous surfaces, not intended for use on medical devices or on skin.

KEYSTONE
KEYMODEL ULTRA™

KeyModel Ultra™ is a material designed for 3D printing of dental and orthodontic models. Ideal for thermoforming aligners.

Uses and Advantages

- Ultra-fast printing
- Thermoforming quick-release agent
- Flawless detail
- Multiple available colors
- Carve-able without chipping

All products available from: HENRY SCHEIN • Tel: 1300 65 88 22 • www.henryschein.com.au

iTERO
5D PLUS SCANNER

The iTero Element Plus Series offers all of iTero's digital capabilities in the power of one scan, with a plus.
A powerhouse of capabilities:*

- Integrated 3D intraoral1 camera - get more out of one scan
- 20% less waiting time for scan to process
- Enhanced intraoral camera giving detailed visualisation

State-of-the-art experience:

- 20+ years of engineering expertise
- Ergonomically designed to conform to your workspace

Designed based on your needs:

- More freedom of movement with an 18% smaller footprint
- Unlimited and secure cloud-based storage
- Integrated battery to allow 30 minutes of scanning while unplugged

*Unless otherwise specified, all comparisons are relative to previous generation iTero Element scanners.

HU-FRIEDY
HARMONY SCALERS & CURETTES

Available now Exclusively with Henry Schein

NEW Harmony Scalers & Curettes Designed with TrueFit™ Technology

Fit for you with over 2.8 Million data points measured, this handle was designed to easily adapt to your individual grasp

- Confidence in your hand
- Not too heavy or too light
- Secure and nimble grasp
- Reduces Pressure on tooth by 37%*
- Everedge Working Ends which are 72% Sharper than the next leading competitor*
- Reduces Pinch Force by up to 65%*
- 100% Recyclable Packaging

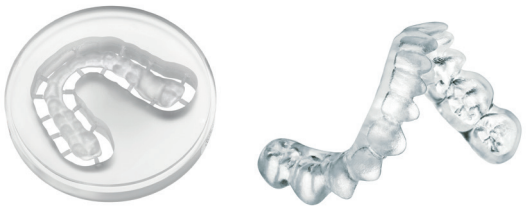
*Data available on file

FORMLABS
NEW DRAFT RESIN

Draft Resin is Formlabs fastest material ever, capable of printing a dental model in under 20 minutes. This highly accurate resin prints with a smooth surface finish, making Draft Resin the ideal material for and any orthodontic practice or lab looking to bring aligner and retainer production in house.

There's no need to sacrifice quality for faster print speed. Draft Resin, even printing at speed, produces highly accurate orthodontic models. Printed at 200 microns, 98% of surfaces are within 100 microns of actual scans.

SCHEU
DURASPLINT FLEX



MMA-free material for the fabrication of functional occlusal splints with thermoplastic memory effect.

The material is flexible at body temperature, leading to an increased wearing comfort. At the same time, the material is characterized by a high restoring torque at room temperature, high breaking resistance, durability and permanent transparency.

The unique manufacturing process guarantees permanent tastelessness of the finished splints. Available as milling blank in thicknesses of 16 and 20 mm and as cold-curing resin for use in the casting and pressing techniques.

All products available from: HENRY SCHEIN • Tel: 1300 65 88 22 • www.henryschein.com.au