Homo erectus (upright man): sometimes known as Homo ergaster and commonly referred to as Peking Man, lived some 1.9 million years ago. The first hominin to control fire. Teeth are larger than Homo Sapiens dentition but smaller than most other hominins. Prominent supraocular ridges are a characteristic feature.
30 AUGUST 2019 - 1 SEPTEMBER 2019

We’re going to Durban!
1200 delegates expected
Africa’s Premier Dental and Oral Health Event of the Year!

Proudly hosted by:
The South African Dental Association

• International speakers
• South African speakers
• Plenary and parallel programmes
• Hands-on sessions
• Social programmes

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Dear Readers,

This issue sees the last of our articles for the Theme: Oral manifestations of systemic disease.

Many thanks and much appreciation are extended to all contributors in this regard.

“The more things change, the more they stay the same” may ring very true and is ever more relevant to oral healthcare workers in the setting of HIV/AIDS. Infectious disease will always remain of clinical concern.

Critically, Sub-Saharan Africa has seen the greatest resurgence of many long-forgotten infections as HIV/AIDS continue to ravage the population. Anti-retroviral medications have been miraculous in extending the longevity of HIV-infected patients.

These patients, though, remain at risk for the development of infectious diseases and infectious neoplasms whilst also being increasingly susceptible to chronic lifestyle diseases including cardiovascular disease and diabetes.

Despite non-HIV-infected individuals living longer, they too have become susceptible to a range of infectious conditions as they age. This places an enormous burden on our healthcare system as well as affecting the quality of life for these patients.

The morbidity of infectious diseases has severely impacted the ability of many patients to remain economically active members of society.

The United Nations have developed several development goals in order to ensure a safer, sustainable future by increasing the quality of life for all.

Good health and wellbeing are crucial goals in this respect. The role of oral healthcare workers in the identification, diagnoses and management of diseases is thus an essential component in the realization of these goals.

We hope that this Educational Theme has assisted in raising such awareness and has inspired readers to contribute to better health and wellbeing for all patients under our care.

I would like to thank the South African Dental Association, the editor of the South African Dental Journal, as well as all of the contributors for the opportunity to encourage reflection on the burden of infectious diseases we currently face and to promote continued holistic patient care.

Regards,
Belinda.

B Bunn: BDS, Fellowship Oral Pathology (SA), MDent.
Head Clinical Unit, Oral Pathologist & Senior Lecturer,
Department of Oral Pathology and Oral Biology, School of Dentistry,
University of Pretoria. E-mail: belinda.bunn@up.ac.za
The capital city of South Africa, Pretoria, was adorned in a haze of purple from a plethora of Jacaranda trees whilst playing host to an amazing SADA Dental & Oral Health Congress and Exhibition 2018 on the 12th – 14th October 2018. The Sun-Times Square Arena and Conference Centre in the heart of Menlyn was the chosen venue for this event.

The Congress which was hosted under the title “From Start to Finish” was delivered excellently through our 31 local and overseas speakers. SADA was honoured to have delegates from neighbouring SADC countries converging for a magical 2,5 days of learning, networking and fun.

During this Congress, we hosted in excess of 1113 delegates. Our event included interesting topics such as:

- Long term complications of dental implant prosthetics: The Mayo Clinic experience
- Management of severe curvatures and complex anatomy with controlled memory EDM files: A new approach for predictable results
- Composite vs Ceramic: Adhesthetics protocols
- Magnification: Not just a nice to have anymore
- Importance of oral cancer screening by primary dental and medical providers
- Assessing your infection control protocols
- The importance of high torque (full smile) in the occlusion aesthetic of the smile and biology in complex cases
- Treatment of skeletal class II/III in adult patients and conclusions
- The practice manager’s role in creating a collaborative positive practice culture
- Effective tissue management and the use of endo-chemistrys
- Strategies to improve clinical color matching of restora-tive materials for the replacement of teeth and gingivitis
- The role of the dentist in sleep medicine
- Getting along in the dental team
- The oral microbiome - recent advances in science

just to mention a few of the offerings. The 2,5 days programme certainly had something for everyone and delegates were offered a myriad of Master and Hands-on Classes and Parallel Sessions to select from.

Attendance to the event allowed delegates to earn a maximum of 21 CPD points when attending the whole event. It was absolutely a sparkling event in a magnificent backdrop of the Pretoria plenary.

This was the first time that SADA extended an invitation to oral health students to experience a SADA event of this magnitude prior to their completion of studies and graduation. Sefako Makgatho Health Sciences University, University of the Witwatersrand and University of Pretoria between them ensured 40 oral health students attended the event, their attendance being sponsored by SADA.

On Saturday evening SADA hosted one of the best gala dinners held for the oral health graduands from the three universities in the area. This was a first to have SMU, Wits and UP students celebrate together. The feedback from graduands attending the Congress and those who attended the Gala Dinner were very positive, and SADA are considering hosting the local graduates from the three universities again in the future. SADA Gauteng South and Pretoria branches led by Dr Sanele Poswa and Dr Ian Erasmus were outstanding in looking after the students.

Our local and international speakers helped make this an event to remember and celebrate. There was also plenty of time for socialising and networking since most of the delegates and exhibitors were staying at or close to the venue. Many stayed past midnight catching up and sharing both dental practice challenges and possible solutions.

After such a successful event, we are working on hosting the next event on the 30th August – 1st September 2019 at the Nkosi Albert Luthuli International Convention Centre, Durban (Formerly Durban International Convention Centre). With an expected 1300 delegates, local and international exhibitors, more dental and oral health professional associations joining in with SADA this promises to be bigger, better and enlightening. Durban is a destination not to be missed. It is a coastal city in eastern South Africa's KwaZulu-Natal province. Durban is known for its African, Indian and colonial influences. It boasts a beautifully refurbished seaport promenade running from uShaka Marine World, a huge theme park with an aquarium, to the futuristic Moses Mabhida Stadium. The Durban Botanical Gardens showcases African plant species.

The SADA Scientific Advisory Committee (SADASAC), chaired by Prof Dale Howes is already working very hard to design a great clinical programme with the best speakers both locally and internationally who are experts in their field.

As Abraham Lincoln said, “The best way to predict the future is to create it." SADA is busy creating a new energy amongst the dental and oral health profession.

Yours in Dentistry,

KC.
· An excellent exhibition area
· Well attended workshops
· Phenomenal guest speakers
· Exciting social functions

A random display of the excited SADA Dental & Oral Health Congress and Exhibition that was held at the Sun Time Square Arena and Conference Centre in Pretoria from the 12th to the 14th of October 2018
Dental fraud in South Africa 2007 – 2015

SUMMARY

Healthcare fraud wastes money properly allocated to patient treatment, and the extent, never researched, is difficult to determine, especially in the South African two-tier healthcare system. A retrospective, record-based study aimed to determine these data between 2007 and 2015.

Data were sourced from the Health Professions Council of South Africa (HPCSA), from Discovery and from the Board of Health Care Funders (BHF). Cases against dentists and dental specialists at the HPCSA peaked in 2013 with 22 cases, while dental therapists faced 12 cases in both 2009 and 2014. While there is a gradual decrease in the number of fraud cases, the amounts involved are increasing.

Discovery revealed that fraudulent cases involving dentists have decreased from a high in 2007 with 179 cases to 63 in 2015, with total fraud just more than ZAR13.6 million. Cases involving dental therapists have increased from 1 in 2007 to 22 in 2015. The total for all dental professionals at Discovery was ZAR18.1 million. BHF estimated that ZAR 40 million was lost to dental fraud over the nine year period. Dental fraud appears to be on the increase. It seems that dental therapists have a higher incidence than amongst dentists or dental technicians.

INTRODUCTION

Fraud is not a new phenomenon and is not limited or confined to certain countries, governments or even specific industries. It is however more prevalent in some countries than in others, affecting developed as well as resource-poor countries.

Most countries aim to diminish crime, fraud included; yet there is a stoical awareness that it is unlikely that a crime-free society will ever be achieved. Policing can, however, try to make it as difficult as possible to commit crime, and in this case, to commit fraud or to indulge in fraudulent activities.

The healthcare industry is unique in that it has many different role-players; government, large private companies including hospital groups and insurance companies (called medical aid schemes in South Africa), private healthcare practitioners and consumers, and is both a service- and product-based industry.

This results in a very complex industry where vast amounts of money are spent while at the same time there are too many role-players vying for their own personal gain. Inevitably, some take advantage of the system and manipulate it to their benefit.

Very little research has been done on healthcare fraud and specifically dental fraud in South Africa. Even though medical aid spending on dentistry forms only a relatively small part of the healthcare industry in South Africa, millions of rands are at stake, offering a viable market for healthcare fraud.

With the general public complaining of the high cost of and steep inflation in healthcare, fraud detection and prevention in the industry should be of paramount importance for governments, politicians and policy makers. The purpose of the present study was to estimate the levels of dental fraud and the amounts involved, in South Africa. As with many other crimes, it is nearly impossible to determine the exact amount of dental fraud in the country.

An estimation can be calculated by using international standards and averages in relation to the total amount of healthcare expenditure. One method is to review the fraud cases documented by the Health Professions Council of South Africa (HPCSA) as well as examining fraud cases from individual medical aid schemes or administrators in South Africa. However, Gee et al. (2010) have shown that this will represent an under-estimate of the amount of fraud.¹

Detected cases of fraud do not of course represent the total number of fraud cases. According to the HPCSA, all cases of fraud in excess of R100 000 should be reported to the South African Police Service (SAPS), but it is not possible to obtain any data from the SAPS.²

Since there is a paucity of research in this area, a nine year retrospective study (2007-2015) on dental fraud was carried out. It was anticipated that the review would assist in the development of guidelines to help prevent dental fraud in the future.
The World Health Organisation (WHO) has re-iterated the fact that one can never eradicate the dishonest minority that commit healthcare fraud, but, by developing and implementing a strong anti-fraud culture, the number of the honest and ethical health practitioners can be increased.\(^9\)

Improving the character of a nation instead of writing and implementing more rules, may eventually result in a decrease in fraudulent crime.

**DEFINING FRAUD AND HEALTHCARE FRAUD**

Fraud can be defined as the wrongful deception, misrepresentation or concealment with the clear intention to deceive, resulting in personal or financial gain, or as intentional theft.\(^4,6\)

Vian (2008) described corruption as the misuse of power for personal or private gain, and also noted that definitions for corruption vary from country to country, and may even be different within areas of the same country.\(^7\)

Busch (2012) defined healthcare fraud as the deliberate practicing of a scheme or programme to defraud a healthcare scheme or attaining money or property by means of false undertakings, representations or deceptions.\(^8\)

Fraud is not the same as abuse. Hannigan (2006) reported that fraud implies an *intention* to be dishonest whereas abuse does not, and Busch (2012) defines healthcare abuse as substandard care. Hannigan (2006) goes further to state that although abuse does not imply intent, it is not excusable on the basis of ignorance.\(^9\)

It is clear that there is no perfect or uniform definition for fraud. The most difficult part when dealing with fraud is proving intent. Many fraudsters claim ignorance and often receive a lesser punishment for abuse, rather than being labelled as an intentional fraudster, which in most healthcare systems carries a far more severe punishment.

**HEALTHCARE FRAUD IN SOUTH AFRICA**

A survey conducted by KPMG in which several of the largest medical aid schemes in South Africa participated, reported 11 200 cases of fraud for the three year period 2007 – 2009.\(^11\) The rand value of these fraud cases exceeded ZAR221 million.

This was the third survey of its kind done by KPMG and even though the figures are very high, they have documented a downward trend in both the number of fraudulent cases per year as well as the value of these cases over nine years.\(^11\)

Discovery Health recovered more than ZAR250 million from fraudulent claims in that three year period.\(^12\) It must be noted that, with roughly 84% of the total population in South Africa not medically insured, a vast amount of healthcare fraud would not be included in these figures.

Statistics released by the Board of Healthcare Funders (BHF) recently reported healthcare fraud in South Africa to be approximately R22 billion annually, although when applying the international average of 7% of claims paid, it is estimated at between R3 billion and R15 billion annually.\(^12\)

Kahn (2014) recently reported estimates of between R8.22 billion and R42.2 billion.\(^13\) The Health Professions Council of South Africa (HPCSA) published figures for healthcare fraud estimates at between R4 billion and R15 billion, while the WHO at the same time reported healthcare fraud in South Africa to amount to between R4 billion and R8 billion annually.\(^2,3\)

The 2013-2014 annual budget for healthcare expenditure in South Africa was R133.6 billion.\(^14\) The estimate of R3 billion – R15 billion fraud would equate to 2.25% - 11.23% of that annual budget. This is in line with worldwide estimates when compared with annual healthcare budgets in the United States and Europe.

It does not, however, mean that this is an acceptable figure. The figures released by KPMG are much lower when compared with other estimates of the situation in South Africa, and this could be due to the fact that the KPMG report investigated fraud at the medical scheme level, and only around 16% of South Africans are medically insured.\(^11\)

As can be gleaned from the discussions above, the figures for fraudulent activities are all estimates with a wide range of values. The WHO confirmed the absence of accurate data in a recent report. A frightening statistic though, shows healthcare fraud to be on the increase in South Africa.\(^15\)

**DENTAL FRAUD IN SOUTH AFRICA**

No statistical data could be found specifically for dental fraud cases in South Africa, although Postma et al (2011) published data regarding complaints against oral health professionals in South Africa.\(^16\)

Fraud was one of the categories under which complaints were reviewed. Postma et al (2011) reported 30 fraudulent cases between 2004 and 2009, which added up to 29% of all complaints against dentists.\(^16\) For dental therapists 12 cases of fraud were reported between 2004 and 2009, resulting in 46% of the total number of complaints against dental therapists.\(^16\)

It was also noted that the fraud-related complaints generally arose due to the irregular accounts that were sent to patients and/or irregular submissions to medical aid fund administrators.\(^16\)

According to a report published by KPMG nearly 70% of all healthcare fraud consists of charging for services not rendered and code manipulation.\(^11\)

Unfortunately these figures do not provide a complete picture of the amount of dental fraud in South Africa. The cases were only those investigated by the HPCSA, and where practitioners were found guilty. Many fraud cases never reach the HPCSA and are either resolved at patient-practitioner level or at the medical aid administrator level.
COMMON HEALTHCARE FRAUD TYPES IN SOUTH AFRICA

Postma et al (2011) reported on the following types of fraud, which they found in the misconduct records of the HPCSA:

- over-servicing, over-charging, claiming for services rendered to non-members, changing service dates, discrepancies between clinical records and billing records, submitting claims whilst suspended from practicing, incorrect tariff codes, claiming for procedures not performed, split billing and claiming for non-claimable goods. Unfortunately, these HPCSA data do not reflect the exact amount of fraud in each type.10

The triennial KPMG survey found code manipulation to represent 39.81% of service provider fraud cases between 2007 and 2009.11 This was followed by: charging for services not rendered (25.32%).

These two alone made up nearly two thirds of the total number of healthcare fraud cases and showed an increasing trend in the number of code manipulation cases. Service provider fraud totalled ZAR151.9 million while member fraud came to ZAR67.3 million.

Ogunbanko et al (2014)15 compiled the following list of the types of healthcare fraud committed in South Africa:

- **Service provider fraud**
  - Pharmacies dispensing generic medication but claiming for expensive brand-name medication.
  - Pharmacies selling front-shop items but submitting claims for medication that is not dispensed.
  - Pharmacies selling high-cost devices in surplus of the needs of the member.
  - Claiming for services not rendered.
  - Service providers willingly treating non-scheme members but claiming as if treating a scheme member.
  - Dispensing doctors dispensing generic medication but claiming for expensive brand-name medication.
  - Fraudulent sick notes.
  - Providing cosmetic treatment but claiming for some other covered procedure.
  - Changing of diagnosis to access a specific benefit.
  - Claiming for excessive or additional material not used during treatment.
  - Dentists claiming for additional fillings or extractions that were not performed.
  - Dentists providing cosmetic gold inlays but charging for normal crowns.
  - Bio-kineticists acting as personal trainers to healthy members in gyms but claiming for rehabilitation services.

- **Member fraud**
  - Forging and submitting claims for procedures that were never rendered.
  - Claiming for high-cost equipment, receiving the money, but then failing to pay the supplier and not collecting the equipment.
  - In collusion with doctors and hospitals claiming for false hospital admissions.
  - Sharing of medical scheme membership card with non-scheme members.
  - Fraud by other individuals or syndicates
    - Submission of false membership applications and submitting claims for those false memberships.
    - Falsification of bank details to receive payment instead of members of service providers.
    - Admission of healthy members to hospitals in order to benefit from hospital cash-back insurance.
    - Syndicates colluding with employees of healthcare funders.
    - Brokers providing false information to avoid waiting periods and late joiner penalties.

Discovery reported on the following types of healthcare fraud in South Africa:

- Claiming for services not rendered
- Merchandising
- Claiming for non-covered benefits as a covered benefit
- Cash Loans (ATM scams)
- Card Farming
- Cosmetic Surgery
- Code Gaming or Manipulation
- Non-disclosures

It is clear from this list that perpetrators are always finding new and creative ways to commit fraud in the South African healthcare system.

Healthcare fraud wastes money that could be spent better in the management of patients. The exact amount of healthcare fraud is very difficult to determine, especially in a two-tier healthcare system like South Africa.

The amount and cost of dental fraud in South Africa has never been researched. If the amount and cost of fraud in a specific area can be determined, resources can be better used to combat healthcare fraud in the future.

The present study design was a retrospective, record-based study. The study protocol was submitted for ethical approval and approved by the University of the Western Cape Faculty and University Research Ethics Committee.

Confidentiality was maintained at all times. The number of dental fraud cases which had been recorded in South Africa over a nine year period was determined, and where possible, the value of the fraud in each case was calculated. The number of cases per dental professional was calculated to provide a picture of the overall level of fraud in the profession.

Data was accessed from the HPCSA website on which is published annually a list of completed cases against healthcare practitioners. Discovery provided data from a number of Medical Aids, after having sought the permission from the individual schemes. At no time was any personal information regarding patients, the practitioners or the individual Medical Aid discussed or recorded.

Contact with other Medical Aid schemes was attempted, but the enquiries were repeatedly referred to The Healthcare Forensic Management Unit (HFMU) of the Board of Healthcare Funders (BHF). That Unit responded to the request and provided relevant data without any personal information regarding patients or practitioners.
The collected data from the HPCSA, Discovery and the BHF were recorded and captured on a Microsoft Excel spreadsheet. The data varied considerably between institutions and were not readily comparable due to differences in the interpretation of fraud and fraud categories, and therefore no further statistical analysis was carried out on the data.

RESULTS

The lists of cases published annually by the HPCSA include procedures conducted against all healthcare professionals registered with the HPCSA and are not limited to fraudulent activity.

Dental professionals registered with the HPCSA include dentists and dental specialists, dental assistants, dental therapists and oral hygienists. Cases of abuse are included with the fraudulent cases reported by the HPCSA. Some practitioners had more than one case registered and in the present study each was counted separately.

The HPCSA data was available only from 2007 onwards. Discovery submitted data for all medical schemes that fall under their administration, which constituted just more than 1.2 million insured lives. The BHF were only able to obtain permission and data for 40% of the Medical Aid schemes registered with them.

These did however include the dental administrator DENIS as well as Medscheme. A lack of manpower at BHF prevented their providing data on the number of cases per practitioner. The actual number of cases might thus not give an accurate representation of the extent of fraud.

HPCSA finalised cases (2007-2015)

The reported HPCSA cases between 2007 and 2015 are shown in Table 1. Cases are shown for dentists and dental specialists together, as the HPCSA makes no differentiation between general dental practitioners and dental specialists.

Table 2 shows cases against dental therapists for the same period. There were no cases reported against oral hygienists or dental assistants and dental technicians are not registered with the HPCSA.

It should be noted that these Tables reflect only those cases where the HPCSA found the dental professional guilty, and are not an overall list of all the cases handled by the Council. Cases were categorised into fraud, clinically related, employment of unregistered person(s) or laboratory, poor record keeping, billing/price, incorrect advertising and unlicensed equipment.

Figures 1 and 2 are graphical representations of the fraud cases against dentists and dental therapists between 2007 and 2015 respectively.

Cases against dentists and dental specialists at the HPCSA reached a crescendo in 2013 with 22 cases. The majority of the fraud cases (19) in 2013 were committed by a single practitioner. The same practitioner was also responsible for 47 guilty charges of employing an unregistered laboratory/person. In 2014 a single practitioner was responsible for half of the fraud cases.

The data for dental therapists is very different from that of the dentists and dental specialists. Cases against dental therapists reached a maximum in both 2009 and 2014 with 12 cases. In between those years the cases dropped to zero in 2011.

The total number of dental fraud cases can be seen in Figure 3. The linear trend shows a gradual increase in dental fraud for all dental professionals in South Africa according to HPCSA data between 2007 and 2015. Due to the numbers being small and easily overshadowed by one or two practitioners with a large caseload against them, it is difficult to track the trends for fraud per dental professional.

RESPONSE RATE

The HPCSA data was available only from 2007 onwards. Discovery submitted data for all medical schemes that falls under their administration, which constituted just more than 1.2 million insured lives. The BHF were only able to obtain permission and data for 40% of the Medical Aid schemes registered with them.

These did however include the dental administrator DENIS as well as Medscheme. A lack of manpower at BHF prevented their providing data on the number of cases per practitioner. The actual number of cases might thus not give an accurate representation of the extent of fraud.
### Table 1. Finalised cases with the HPCSA for dentists and dental specialists: 2007 – 2015

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<td>13</td>
<td>8</td>
</tr>
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### Table 2. Finalised cases with the HPCSA for dental therapists: 2007 - 2015

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### Table 3. Number of fraud cases at Discovery (2007 – 2015)

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<td>3</td>
<td>37</td>
<td>108</td>
<td>29</td>
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### Table 4. Value of fraud cases at Discovery (2007 – 2015) in South African Rand (ZAR)

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<td>3180.00</td>
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<td>212 294.11</td>
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### Table 5. Number of fraud cases at BHF (2007 – 2015)

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<td>7</td>
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### Table 7. Total Value of Fraud Cases at BHF (2007 – 2015)

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<td>82 000.02</td>
<td>42165.99</td>
<td>1 218 383.55</td>
<td>8 666 631.95</td>
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Discovery cases (2007 – 2015)

The number of dental fraud cases per dental professional as found on all Medical Aid schemes under the administration of Discovery between 2007 and 2015 are shown in Table 3, and Table 4 shows the value in South African Rand of those cases. Data are shown for dentists and dental specialists together, as Discovery makes no differentiation between general dental practitioners and dental specialists. There were no cases reported against oral hygienists or dental assistants.

Figures 4, 5 and 6 show graphical representations of the number of fraud cases for dentists, dental therapists and dental technicians individually between 2007 and 2015. Figures 7, 8 and 9 show the value in South African Rand of those fraud cases.

The Discovery data show three distinct patterns for each dental profession. The overall picture for the dentists shows a gradual decrease in the number of fraud cases, although the actual ZAR values are still increasing. The number of fraudulent cases involving dentists as investigated by Discovery decreased from a high in 2007 with 179 cases to 2015 with 63 cases.

Fraud by dental therapists showed a remarkable increase in both the number as well as the value of cases. The incidence increased from 1 in 2007 to 22 in 2015. The number of registered dental therapists is just more than 10% of the number of registered dentists in South Africa on a yearly basis, yet the value of fraud for dental therapists is nearly 25% that of the dentists. The dental technicians showed very little fraud, except for a larger caseload in 2012 and 2013. Dental technician cases started at 1, climbed to a maximum of 134 in 2013, then decreased again to 2015 with 2 cases.

Data from Discovery reveals a total value of the fraudulent cases involving dentists from 2007 to 2015 to be just more than ZAR13.6 million, whilst the total for all dental professionals for the same time period was ZAR18.1 million.
Table 5 shows the number of dental fraud cases per dental profession as found in the BHF collated data between 2007 and 2015 and Table 6 shows the value in South African Rand of those dental fraud cases per dental profession.

Cases are shown for dentists and dental specialists together, as the BHF makes no differentiation between general practitioners and dental specialists. There were no cases reported against oral hygienists or dental assistants.

Figures 10 and 11 show graphical representations of the number of fraud cases for dentists and dental therapists respectively between 2007 and 2015, whilst Figures 12 and 13 show the value in South African Rand of those cases.

Between 2011 and 2013 the BHF underwent a change in the fraud detection system, resulting in very few cases being recorded during that period.

Unfortunately the BHF were unable to provide data for 60% of all the medical aid schemes that are members of BHF, and the number of fraud cases does not take into account more than one case per practitioner.

The actual number of fraud cases may therefore be grossly under-estimated. With the changes in the fraud detection systems, very little data exists between 2011 and 2013.

When the value of dental fraud from the BHF data was extrapolated for all their medical aid scheme members, the total was found to be ZAR 21.6 million over the nine year period.

It is clear from the results of the present study that it will always be impossible to accurately determine the actual amount of healthcare or dental fraud in any healthcare system. The total amount of fraud can be estimated, evidence of the amount of fraud identified or detected can be provided, but figures are often under-estimates. Many patients are victims to healthcare fraud without being aware of it.

It is of considerable concern that dental fraud over a nine year period in South Africa was estimated to be nearly ZAR 40 million. Even this figure is an under-estimate as it does not include the HPCSA data as the Council does not specify the value of the fraud. In addition, fraud appears to be on the increase in most cases.

A recent report by KPMG found that only 0.9% of healthcare fraud in South Africa was reported to the South African Police Service (SAPS) between 2001 and 2009. This is a very worrying statistic, especially considering the stance other countries are taking against fraud. As mentioned earlier, the Attorney General’s office in the USA listed healthcare fraud as one its most important priorities, second only to violent crime. Of course, with the high levels of violent crimes in South Africa it may be easy to argue why human resources are not being used to combat healthcare fraud but rather is being spent on fighting violent crimes.

There is no central database for reporting healthcare or dental fraud in South Africa, and very few cases are reported to the SAPS… or even the HPCSA. This makes tracking the total extent of fraud very difficult.
There is also a lack of co-operation and communication between the individual corporate bodies, necessary if they are to work together to decrease healthcare fraud. This situation is also prevalent in other countries.

Dental or Medical Councils do not report fraud by its members to the other international professional councils. This has been recently illustrated by a case in this country where a South African born dental practitioner was jailed in the UK for dental fraud, but returned to South Africa and has been permitted to continue to practice in the country. When a person is jailed for fraud and prevented from continuing his/her practice in a certain country, it is surely prudent that the authorities be informed.

Many Medical Aid schemes are unwilling to share their data to help combat fraud and this could also be why so little healthcare fraud is reported to the SAPS. Medical Aid schemes spend much time and effort recovering money which has been lost from members due to fraud and once that has been recovered, do not feel the need to report the perpetrators.

Fraud seems to be ever-increasing amongst all dental professionals, but more so with dental therapists. That discipline represents less than 10% of the registered dental professionals with the HPCSA, but fraud committed by therapists accounts for more than 30% of the total amount committed by dental professionals.

CONCLUDING REMARKS

One of the main hurdles in the fight against healthcare fraud, not only in South Africa, but also on a global scale, is the lack of communication and co-operation between the different role players. Very few Medical Aid Schemes or administrators share fraud data among themselves, with the HPCSA, and even less with the SAPS. If healthcare fraud is to be reduced, this situation has to change.

It should be a requirement that all healthcare fraud above a certain threshold should be reported to the SAPS as well as to the HPCSA. Practitioners proven to have committed fraud above a certain value should be taken off the Register and at least not be allowed to practice for a period of time. The FDI (World Dental Federation) may be that body which could institute an international register to list practitioners, and at least not be allowed to practice for a period of time.

Billions of dollars, euros, pounds and rand are lost annually to fraud with no clear light at the end of the tunnel. Prevention could save millions of rand that could be ploughed back into the delivery and provision of healthcare. To achieve this, it is important that all role-players in the South African healthcare milieu take a policy approach of zero tolerance and work together to combat and overcome medical and dental healthcare fraud.

It is important to remember that not all medical and dental professionals commit fraud; the profession is filled with ethical, honest and dedicated men and women. However, while it is only a small percentage of practitioners who commit fraud, the problem is in fact on the increase. Further studies are needed on a regular basis to track changes in dental fraud in South Africa.

References

Teeth are often used to assist in the identification of human bodies after death, especially in cases where the body is badly burned or decomposed. Age estimation can play a significant role in helping narrow down the spectrum of possible identities. Gustafson created a method of dental age estimation, using six age-related changes of teeth that occur after the eruption of the dentition. This age estimation method has been used on unidentified individuals at the Salt River and the Tygerberg Medico-Legal Laboratories. However, it may be questionable as to whether the method is accurate when applied to the population of the Western Cape.

The aim of this study was to test the accuracy of Gustafson's method on a sample of adult teeth from the Western Cape, of known chronological age.

Extracted mandibular central and lateral incisors and maxillary central incisors were used in this study. Two examiners independently used Gustafson's method to estimate the ages of the donors of the teeth.

This method was found to be inaccurate when applied to a sample of the adult population of the Western Cape.

Keywords
Forensic Dentistry, Age estimation, Teeth.

SUMMARY

Introduction

Teeth are often used to assist in the identification of human bodies after death, especially in cases where the body is badly burned or decomposed. Age estimation can play a significant role in helping narrow down the spectrum of possible identities. Gustafson created a method of dental age estimation, using six age-related changes of teeth that occur after the eruption of the dentition. This age estimation method has been used on unidentified individuals at the Salt River and the Tygerberg Medico-Legal Laboratories. However, it may be questionable as to whether the method is accurate when applied to the population of the Western Cape.

Aim

The aim of this study was to test the accuracy of Gustafson's method on a sample of adult teeth from the Western Cape, of known chronological age.

Methods

Extracted mandibular central and lateral incisors and maxillary central incisors were used in this study. Two examiners independently used Gustafson's method to estimate the ages of the donors of the teeth.

Conclusion

This method was found to be inaccurate when applied to a sample of the adult population of the Western Cape.

Keywords
Forensic Dentistry, Age estimation, Teeth.

INTRODUCTION

Age estimation is an important part of forensic dentistry. The use of teeth for this purpose can play a significant role in the process of identifying a human body after death, especially in cases where the body is badly burnt or decomposed, as teeth are usually preserved for a long period of time, even after most of the other tissues have disintegrated. Identification is usually undertaken by comparison of ante-mortem and postmortem dental records, when the identity of the deceased is suspected. In some cases, however, when the identity of the body is not known, age estimation can play a significant role in narrowing down possible identities which are culled from the Missing Persons database.1,2

Teeth continue to show several different age-related changes, even after the formation and development of the dentition is complete. These changes can be used to estimate the individual's chronological age. The tooth changes used in the Gustafson method (1950) include attrition, change of the level of the periodontal attachment, secondary dentine deposition, resorption of the root, apposition of cementum and translucency of the root.4-7

 REVIEW OF THE LITERATURE

In the past, several different techniques have been suggested for the estimation of a person's dental age, based on the facial and/or oral structures. These include morphological and radiological methods.

Some of the techniques can also be classified as invasive, which can mostly be used only in deceased individuals, and others as non-invasive, which can be used in both the living and the dead.6

Gustafson's method (1950) used the six age-related changes mentioned above.7 Gustafson then applied his method to teeth that had been extracted from persons of known age. He used ground sections of 1.0 mm thickness to determine the translucency of the root dentine and ground sections of 0.25 mm thickness to determine the remaining five factors.

Each of these changes in a specific tooth was then rated and given a value between 0 and 3. The sum of the values of each change of each tooth, combined with the age of
the individual (from whom the tooth had been extracted) was then used to create a regression line. From this graph, the age of an unknown individual was then determined with reasonable accuracy.$^{1,6,7}$

According to Gustafson, the average error in age estimation using this technique was about 3.63 years. He then also found that the estimation of an individual’s age is even more accurate if more than one tooth from that individual were examined. This study was however done only on Europeans from Sweden.$^{1,6,7}$

Over the years numerous studies have been conducted in order to either prove or disprove Gustafson’s method of age estimation, as several researchers and investigators were convinced that there was an error in this method.

Some of these researchers were of the opinion that Gustafson based his method on several assumptions that were most likely incorrect.$^{6,13}$ He assumed that these six criteria were all equally accurate and effective in the process of age estimation and that the rates at which the individual criteria change are equal, resulting in his method of just adding the data together.$^{8,10}$

Gustafson also assumed that the age information obtained from the six different criteria is statistically independent, which has been shown to be inaccurate.$^{8,10}$

The six criteria used in Gustafson’s method of age estimation are also influenced by several different factors (other than aging) and can even have an influence on each other, which Gustafson did not consider. These include the following:

Attrition can be influenced by bruxism, diet, morphology of the teeth and dental arches, the direction and force of masticatory movements and the number of teeth present in the mouth.$^{11,13}$

Secondary dentine can be influenced by attrition, abrasion, periodontal disease and mechanical injury or irritation caused by dental procedures and caries.$^{14}$

Periodontal attachment level can be influenced by periodontal disease.$^{13}$

Cementum apposition can be influenced by periapical periodontitis, root resorption and whether or not the tooth is in function.$^{13}$

Root resorption can be influenced by dental trauma, periapical periodontitis, excessive forces including mechanical forces applied by orthodontic appliances and occlusal forces, hormonal imbalances and pressure from impacted teeth or benign neoplasms that press on the roots of adjacent teeth.$^{13,14}$

Translucency can be influenced by periodontal infection and diseases of the pulp of the tooth.$^{15}$ As a result of these incorrect assumptions and additional influencing factors, most of the subsequent studies have proved the Gustafson method faulty, and have resulted in several modifications to the original method.$^{4,6,16-19}$

Many studies have been performed over the years in order to either prove or disprove this method of age estimation. These were undertaken in several different countries including Sweden (Gustafson, 1950),$^7$ Scandinavia (Bang and Ramm, 1970; Johanson, 1971),$^8,16$ France (Haertig et al., 1985),$^{21}$ Limpopo, South Africa (Nkhumeleni et al., 1989),$^{22}$ Australia (Richards and Millar, 1991),$^{23}$ Germany (Lampe and Roetzsch, 1994),$^{24}$ China (Li an Ji, 1995),$^{25}$ England (Lucy et al., 1995),$^{26}$ America (Pigno et al., 2001), Iran (Monzavi et al. 2003)$^{19}$ and India (Rai et al., 2006; Shrigirwar and Jadhav, 2013).$^2,28$ None were conducted in the Western Cape, in South Africa.

The Western Cape has a population constituting numerous ethnic groups and cultures. A wide diversity in socio-economic circumstances, eating habits, oral hygiene habits and smoking habits also exist, which can all play a role in the age-related changes of a tooth.

This leads to the question: Is Gustafson’s method of age estimation of teeth accurate when applied to the people of the Western Cape?

The aim of this study was to determine the accuracy of Gustafson’s method of age estimation of adult teeth when applied to a sample of the adult population of the Western Cape.

MATERIALS AND METHODS

Caries-free central and lateral mandibular incisors (FDI tooth numbers 32, 31, 41 and 42) and maxillary central incisors (tooth numbers 11 and 21) were collected from the University of the Western Cape Oral Health Centre at Tygerberg Hospital.

These teeth had been extracted as part of routine dental treatment, which resulted in only a limited number of teeth being available for the study. An additional source of teeth was from cadavers of known chronological age (after dissection by medical students), used in the Anatomy and Histology Department of the Faculty of Medicine and Health Sciences of the University of Stellenbosch.

Figure 1. Isomet circular saw used to cut the teeth.

Figure 2. Tooth section mounted on a glass microscope slide.
A total of 55 teeth was used to conduct the study. The teeth were from persons between the ages of 21 and 76 years. Carious teeth, restored teeth, endodontically treated teeth and teeth that presented with either crown or root fractures were excluded from the study.

Details regarding the date of birth, date of death, sex and ethnic group of each cadaver were obtained from the death certificates. The anatomy registration numbers were used to identify the teeth collected from the cadavers. Teeth collected from patients were given random numbers. The teeth were sectioned in the long axis of the tooth (from the labial surface to the lingual surface), using the Isomet circular saw (Figure 1). The thickness of each section was standardized at 100μm.

The tooth sections were subsequently mounted on glass microscope slides, embedded in DPX® (from Leica Microsystems) and covered with a glass cover slip (Figure 2).

Age estimation of each tooth was then undertaken independently by two examiners using the Gustafson’s method. Using this method, the degree of attrition, change of the level of periodontal attachment, the extent of secondary dentine deposition within the pulp, the apposition of cementum, the resorption of the root and the transparency or translucency of the root were given a value for each tooth.

The range of the values was between 0 and 3, with the value 0 meaning the change is not present, and 3 meaning the change is severe. The regression line compiled by Gustafson was then used to estimate the age of the individual from whom the tooth had been extracted. The examiners were blinded as to the chronological age of the individual from whom the tooth derived.

The chronological age was revealed only at this stage and was compared with the estimated age value. The results of the comparisons were statistically analyzed to determine the degree of accuracy of the Gustafson’s method of age estimation.

RESULTS

A total of 55 teeth were used to conduct the study. Of these, 52 had been harvested from cadavers and three teeth extracted from live patients. The age range of the tooth donors was between the ages of 21 and 76 years.

The mean age of the sample was 45 years. Sixteen (16) of the donors were female and 39 were male. Thirty-nine (39) of the donors were of the Coloured ethnic group of the Western Cape, 14 of the Black ethnic group and only two of European origin.

When the estimated ages as calculated by Examiner 1 (using Gustafson’s method of age estimation) were compared with the chronological (real) ages of the donors, none were entirely accurate: 25.5% of the cases showed a difference of less than 5 years between the estimated and real ages, 21.8% a difference of between 5 and 10 years, 38.2% a difference between 10 to 20 years and 14.5% showed a difference of more than 20 years (Figure 3). The mean of the differences (average error) between the real and estimated ages (for Examiner 1) was 13.7 years. The median was 13.0 years and the standard deviation was 9.38 years.

When the estimated ages as calculated by Examiner 2 (using Gustafson’s method of age estimation) were compared with the chronological (real) ages of the donors, none were entirely accurate: 25.5% of the cases showed a difference of less than 5 years between the estimated and real ages, 21.8% a difference of between 5 and 10 years, 38.2% a difference between 10 to 20 years and 14.5% showed a difference of more than 20 years (Figure 3). The mean of the differences (average error) between the real and estimated ages (for Examiner 2) was 11.6 years. The median was 10.5 years and the standard deviation was 8.52 years.

When examining the scatter plots (Figures 5 and 6) showing the difference between the real ages and the estimated ages on the one axis, and the real ages on the other axis, it becomes clear that the ages were over-estimated in most cases by both Examiners 1 and 2. The ages were over-estimated in all the cases where the donors were younger than 45 years of age. It is only in donors 45 years of age and older that both Examiners 1 and 2 under-estimated the ages in 10 cases.

Using the Student’s t-test and p-value to compare the mean differences between the estimated ages and real ages as found by Examiners 1 and 2, it was established that there was not a statistically significant difference between the results found by Examiners 1 and 2, the p-value being greater than 0.05.
Using the Student’s t-test and p-value\textsuperscript{30, 31} to compare the mean differences between the estimated ages and real ages as found by Examiners 1 and 2 with Gustafson, it was established that there was a statistically significant difference between the results found by Examiners 1 and 2 compared with Gustafson, as the p-value < 0.05.

The mean difference (average error) between the estimated ages and the chronological ages was 13.7 years for Examiner 1 and 11.6 years for Examiner 2, with standard deviations of 9.38 years and 8.52 years respectively. Gustafson claimed that the average error in age estimation using this technique was about 3.63 years.\textsuperscript{7}

There is clearly a statistically significant difference between the average error claimed by Gustafson and those found in this study (p-value < 0.05). The results found in this study are also different from those found in other similar studies that were recently done by Bajpai (2011), who found a mean difference of 4.86 years between the estimated and chronological ages and Shrigiriwar and Jadhav (2013) who found a mean difference of ± 4.43 year.\textsuperscript{28, 32}

This study found that the ages were over-estimated in most cases by both Examiner 1 and 2, especially when the donors were younger than 45 years of age. In donors 45 years of age and older, the ages were under-estimated in ten cases.

This is consistent with the results found by Solheim and Sundnes (1980) who found that over-estimation of ages occurred mostly in individuals younger than 40 years and under-estimation in individuals older than 50 years of age.\textsuperscript{33}

Mandibular central incisors usually erupt between the ages of 6-7 years. Mandibular lateral incisors and maxillary central incisors usually erupt between the ages of 7-8 years.\textsuperscript{34} These permanent teeth would have been present in the mouth for the longest period, and would presumably give the most accurate results when used in age estimation.

Other studies have, however, used several different teeth and have obtained much more accurate results than those found in this study. These include studies done by Maples (1978) who used second molars, Monzavi et al. (2003) who used first premolars and Bajpai (2011) who used canines, premolars and incisors (in order of preference).\textsuperscript{9, 18, 32}

Although the differences between the results found by Examiner 1 and Examiner 2 were not statistically significant (p > 0.05), a method that uses scientific devices to accurately measure the different criteria used in Gustafson’s method of age estimation needs to be developed, which may contribute to even more uniform results.

The limitations of this study included the small sample size and the relatively small demographic area in which the teeth were collected. The small sample size was however due to the very strict exclusion criteria.

The sample size (55 teeth) in this study was larger than the sample originally used by Gustafson (41 teeth) and that used recently by Bajpai (20 teeth).\textsuperscript{7, 32} Further studies are therefore necessary which will ideally consist of a larger sample size and a wider demographic area in which the teeth are collected.

The results found support the hypothesis and prove that Gustafson’s method of age estimation is not applicable for the adult population of the Western Cape.
CONCLUSION

Gustafson’s method of age estimation was derived more than 60 years ago and was based only on Europeans. The present study was undertaken to test the hypothesis that the age estimation of Gustafson was not applicable to the population of the Western Cape.

The results showed that Examiner 1 and Examiner 2 over-estimated the ages of 45 of the 55 individuals. All the cases where the ages were under-estimated were of individuals who were over 45 years of age.

The difference between the results found by Examiner 1 and Examiner 2 were not statistically significant (p > 0.05). The results of the two independent examiners were found to be consistently and uniformly inaccurate when attempting dental age estimation of the sample.

REFERENCES

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Isolated palatal injury due to a bicycle accident

ABSTRACT

Trauma to the soft palate due to impalement of a bicycle handlebar does not appear to have been reported in the literature. More commonly the trauma occurs in toddlers due to impalement with objects such as pencils. We report an atypical presentation of an 11 year old with a significantly lacerated soft palate suffered when he fell off a bicycle.

The child’s mouth must have been open when he fell, which allowed the handlebar to injure the soft palate without causing damage to his teeth. A clinical evaluation and advanced radiographic images revealed significant impairment of the musculature of the soft palate.

Treatment was performed under general anaesthesia and involved correct placement and suturing of these anatomical structures, which were ectopically displaced.

Good oral hygiene was prescribed, including the use of a chlorhexidine based oral rinse. The patient’s recovery was uneventful with almost complete healing within three weeks.

It is clear that bicycle safety needs more emphasis in South Africa. Apart from the use of safety devices such as helmets and mouth guards, there may be a need for legislation to be amended to enhance safe accommodation on the roads for cyclists.

INTRODUCTION

Injuries associated with the childhood achievement of bicycle riding can have devastating effects on both victims and parents. An estimated 900 deaths and 494,000 visits to emergency department occurred due to bicycle-related injuries in the USA, translating into an expenditure of over $2 billion on bicycle-related traumatic brain injuries (TBI).1

Brain and maxillofacial trauma, and dental injuries are common amongst bicycle riders.1 Bicycle accidents may also result in trauma to the abdominal organs including the liver, spleen and kidney.

ACRONYMS

BSI: Bicycle Spoke Injury
MIO: Maximal Interincisal Opening
MRI: Magnetic Resonance Image
TBI: Traumatic Brain Injuries

The thorax, genito-urinary organs and limbs are also at risk.2 Bicycle spoke injury (BSI) involves the foot, ankle or the lower leg as a result of entrapment in the bicycle spokes, while bicycle handlebar injury may involve abdominal trauma. TBI is the leading cause of morbidity and mortality after bicycle-related accidents.

Maxillofacial trauma resulting from bicycle injuries includes orbital, zygomatic, nasal and mandibular fractures.2,3 Dental injuries include crown or root fracture, luxation, intrusion and avulsion.3

Most injuries involve superficial trauma such as abrasions (“road rash”), contusions and lacerations. Abrasions can range from being superficial to those involving partial or full skin thickness. Treatment requires debridement and irrigation of embedded residue to prevent “traumatic tattooing”.4

Cyclists may suffer accidental falls as a result of inattention, poor road conditions, and road traffic accidents such as collisions with other cyclists or motor vehicles.3 To our knowledge, isolated injuries of the soft palate due to bicycle accidents have not been reported in the literature.

CASE REPORT

An 11 year-old boy was referred to the practice by a medical doctor a week after he had fallen from a bicycle. The initial consultation had been at a local district clinic where analgesics and a mouth rinse were prescribed.

A clinical assessment revealed trismus with a maximal interincisal opening (MIO) of 30mm. He complained of dysphagia and dysphonia while hypernasal speech was also noted.

The temporomandibular joints were not tender. Both the mandible and maxilla were intact and there was no neurological deficit of the face. An intraoral examination showed a laceration of approximately 2cm on the right side of the hard palate, compounded by a significant soft palatal defect.

There was also a previous wound on the posterior end of the right hard palate which extended to the midline, and posteriorly to the uvula. The entire right side of
the soft palate was detached anteriorly and medially (Figure 1a and 1b). The right tonsil appeared to be enlarged. A magnetic resonance image (MRI) was requested to determine the orientation of the residual soft palatal muscle.

The MRI revealed a soft palatal defect which measured 16.6mm in a craniocaudal dimension and 9mm in an anterior-posterior dimension. Asymmetric enlargement of the palatine tonsil was also observed. Diffuse high signal disturbances were noted in the right veli palatini muscles. Defects were also noted in both the palatopharyngeus and palatoglossus muscles. Intense inflammation was present around the defect.

The patient was taken to theatre for an examination under anaesthesia and reconstruction of the remaining soft tissues of the soft palate. Possible modification of the treatment plan included harvesting a right buccal mucomyocutaneous flap if there was inadequate viable tissue for reconstruction.

On examination, what had seemed like an enlarged right tonsil was found to actually be the soft palate, suspended into the oropharynx and healing in an ectopic position as it was still attached to the right lateral oropharynx. The treatment plan was modified to include mobilisation of the detached soft palate.

All wound margins were debrided and sutured in three layers, the nasal mucosa, muscle and oral mucosa. The patient recovered without any complications from the procedure and the general anaesthesia. He was discharged with strict instructions that included a soft diet and the use of an oral rinse with a 0.2% chlorhexidine gluconate solution.

A postoperative examination of the repaired tissues performed a day (Figure 2a) and one week after the procedure (Figure 2b) revealed intact operative sites with no significant complications.

Clinical examination at the final review was performed three weeks postoperatively, at which time the soft palate had almost completely healed (Figure 3). The patient also reported that his vocal ability was restored (no signs of hypernasal speech) and the pain and dysphagia had settled.

DISCUSSION

Maxillo-facial injuries related to bicycle falls are often quite severe. Junior et al described mandibular fractures as being the most common facial bone fracture followed by zygomatic complex fractures in both motorcycle and bicycle falls in Brazil. An Italian paper reported that facial and dental injuries related to bicycle accidents included crown fractures, periodontal injuries, maxillo-facial bone fractures as well as soft tissue lacerations and contusions. A relevant finding noted by these authors was that facial fractures more frequently involved the mid-face in younger individuals, whilst the mandible, maxilla and zygoma suffered in slightly older children.

This significant finding could be credited to a larger, more developed and therefore a more susceptible mandible in these children. Facial fractures were often compounded by dental injuries.

The goals of the managing this patient were to restore form and function. The hypernasal speech could be explained by a loss of oropharyngeal seal resulting in escape of the air via the right soft palate defect to the nose during speech while the dysphagia was due to the exposed sensory nerve fibres of the open wound as well as the intense inflammation around the defect.

We hypothesise that the patient fell forward over the bicycle with his mouth widely opened during the accident, resulting in tearing of the soft palate by impact with the handlebar.
Although the muscles of the soft palate were not dissected out during the repair, function and form were restored to the satisfaction of the parents and the patient. Assessment of the soft palate revealed a fully functional levator veli palatini muscle. Although rare, an injury of this nature should not be treated casually.

Caldwell described an incident of a young male who suffered an impalement injury to the soft palate. He then presented with neurological deficits and died within six days.

The cause was due to the formation of a post-traumatic intramural thrombus in the internal carotid artery (which lies in close proximity to the posterolateral oropharynx), which then resulted in a haemorrhagic stroke possibly due to embolization of the thrombus to the middle cerebral artery in the Circle of Willis.6

CONCLUSION

Bicycle riding is accompanied by a high risk of injury. Safety precautions such as helmet usage are of pivotal importance in terms of prevention. Helmets provide protection against severe TBI, reduces the possibility of facial fractures and saves lives even after the victim sustains an intracranial haemorrhage.7

Skull fractures and subdural haematomas are less prevalent in helmet wearers.8 Noakes found that in South Africa an estimated 85% of cycling fatalities due to head injury could have been prevented by helmet usage.9

He also stated that the frequency of wearing helmets amongst younger individuals was exceedingly low and therefore recommended that the drafting of appropriate legislature making helmet-wearing compulsory may be the only method of increasing usage.

Maxillo-facial injuries would still be prevalent even with helmet usage and it would therefore be sensible for bicycle users to embrace the wearing of mouth guards to provide some protection for dental and oral tissues. Bicycles equipped with retractable handlebars are also advantageous.

The wearing of reflective gear is strongly recommended, particularly for night cycling.9 Prevention should also ideally centre on education, supervision and the improvement of infrastructure. Learning programs at schools and community centres should focus on knowledge of road safety and cycling skills from a very young age.

The upgrading of rural roads and the designation of specific cycling lanes may seem to be an unrealistic short-term goal in South Africa but it is definitely an achievement to strive toward.

References:
Enamel demineralisation as an iatrogenic effect of Orthodontic treatment: a clinical review

SUMMARY
Small areas of demineralised enamel, commonly referred to as white spot lesions (WSLs), constitute an important clinical problem in Orthodontics following treatment with fixed appliances. They are the result of an imbalance between de- and remineralisation of enamel, caused by the interrelationship of several factors.

Clinicians and patients are usually so focussed on the alignment of teeth within the arches and the relationship of the jaws to each other, that the iatrogenic effects of demineralisation and WSLs are often overlooked.

While attempts should be, and are, made to prevent this clinical problem, it can pose a challenge to manage in high risk patients.

Benefit must supersede risk and it is thus of utmost importance that the clinician be aware of and educates the patient on means to prevent or to minimise and manage WSLs. This manuscript is intended to elucidate the presentation, aetiology and management of WSLs, in the hope of promoting more favourable clinical outcomes for both patient and practitioner.

DEFINITION
White spot lesions are the earliest clinical sign of dental caries. They can be defined as ‘subsurface enamel porosities’ caused by an imbalance between de- and remineralisation.

Enamel translucency relates to the degree of mineralisation and initial demineralisation presents as ‘milky-white opacities’ on the smooth surfaces of teeth.1 Such lesions can develop within a period of four weeks - the time between one Orthodontic appointment and the next.2

ACRONYMS

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<thead>
<tr>
<th>ACRONYMS</th>
<th>DESCRIPTION</th>
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<tr>
<td>CPP-ACFP</td>
<td>Casein Phosphopeptide-Amorphous Calcium Fluoride Phosphate</td>
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<td>CPP-ACP</td>
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<td>GI</td>
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<td>RMGI</td>
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<td>SBS</td>
<td>Shear Bond Strengths</td>
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<td>WSL</td>
<td>White Spot Lesion</td>
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INCIDENCE
The presence of WSLs is significantly greater in Orthodontic patients and Orthodontically-treated arches, compared with non-Orthodontic patients and untreated arches in Orthodontic patients.3, 4 The overall incidence varies greatly between 2% and 96%,5 with the prevalence of WSLs on at least one tooth found to be as high as 49.6% in Orthodontically-treated patients, compared with 24% in a control group of untreated patients.6

The large variation could be due to varying methods of assessment of demineralisation, whether developmental and other idiopathic enamel lesions or local environmental factors are taken into consideration, and whether any form of fluoride is being used during treatment.7

It has also been found that while the prevalence in male and female Orthodontic patients is the same, the severity of WSLs is greater in males, attributed to a lower standard of oral hygiene.8, 9

DISTRIBUTION
The teeth most frequently affected are molars, maxillary lateral incisors, and mandibular canines and premolars.6, 7 The surface most frequently affected is the vestibular surface, and the regions most frequently affected are the cervical and middle thirds of teeth.10 If tooth surfaces are divided into quadrants, larger lesions have been found to occur in the gingival quadrants, more specifically distogingival quadrants. With regard to the arches, maxillary anterior teeth are more severely affected than mandibular anterior teeth, with no significant difference between right and left sides of the respective arches.11
AETIOLOGY

Microbial factors

Streptococcus mutans have been found to be implicated in the initiation and progression of dental caries. These bacteria are able to synthesise extracellular glucans from dietary sucrose, which increases the cariogenicity of plaque. This causes an increase in plaque mass, promotes colonisation of Streptococcus mutans, and changes the diffusion properties of plaque matrix. Lactobacilli, which are found in large numbers in advanced carious lesions, are responsible for the further development of carious lesions but do not play a major role in the initiation of lesions. A fivefold increase in these bacteria has been found in patients undergoing active Orthodontic treatment.

Orthodontic appliances create retention sites that lead to an increased proliferation of Streptococcus mutans and Lactobacilli. The number of Orthodontic attachments, the length of treatment and the level of oral hygiene are factors influencing the problem.

Salivary factors

Apart from the co-existence of the four factors needed for demineralisation to occur (namely bacterial plaque, fermentable carbohydrates, a susceptible tooth surface, and time), salivary parameters such as pH, flow rate and buffering capacity are extremely important. They influence the degree of mineral loss after an acidic episode, the rate of progression of demineralisation, and the potential for repair.

Typically, an increase in salivary flow rate promotes the physical cleaning action, increases its buffering capacity and antibacterial activity, and accelerates clearance of substrates. Salivary pH and buffering capacity both aid in neutralising the plaque acid. The pH generally follows the rate of secretion. Unstimulated saliva may have a pH less than 6, which can rise to about 8 at high flow rates. A low pH favours aciduric bacterial colonies and a high pH maintains a high buffering capacity.

Environmental factors

Oral hygiene

Both oral hygiene methods and the ability of the tongue to remove food particles from the mouth become more difficult when Orthodontic attachments are present. This leads to plaque accumulation and the breakdown of retained carbohydrates.

Diet

The main dietary factor is frequency of carbohydrate consumption. When this increases, overlapping episodes of acid attack occur without any significant intervening recovery, which ultimately results in a net loss of minerals over time.

Fixed Orthodontic appliances

The appliance itself creates stagnation areas, resulting in an increase in bacteria and plaque and promoting demineralisation. However, resting salivary flow rate (and thus salivary pH and buffering capacity) also increases during fixed appliance therapy. This effect counteracts the tendency for demineralisation and explains why there is little demineralisation in some patients with high plaque indices. Figure 1 shows the interrelationship between the various aetiological factors, which may result in either de- or remineralisation.

Figure 1. De-/remineralisation: inter-relationship between aetiological factors (Adapted from Chang et al, 1997).
CLASSIFICATION OF WHITE ENAMEL DISCOLOURATIONS

Not all white enamel discolourations are of carious or demineralisation origin. Russell (1961) classified white tooth discolourations as:
1. Dental fluorosis
2. Non-fluoride opacities
3. White spot lesions

According to Russell’s criteria, the following are features of each of the above discolourations...

Dental fluorosis
- White/yellowish lesions, ill-defined, blends with normal enamel, symmetric distribution.

Non-fluoride opacities
- Localised, well-differentiated from surrounding enamel, randomly distributed and often found in the centre of the tooth crown.

White spot lesions
- Seen in Orthodontic patients, under loose bands, around edges of bracket bases, and in areas that are difficult to clean.¹⁷

METHODS OF EVALUATION

Evaluation of demineralised white spot lesions is important to both clinicians and researchers. While various methods have been used to study the characteristics of these lesions, certain criteria are mandatory. Namely:

1. Validity
   It should determine that the lesion was caused by mineral loss from enamel by acid demineralisation during Orthodontic treatment.

2. Reproducibility
   A reading taken by an assessor at a specific time point should be very similar to a reading taken by that same assessor at a later time point, and readings of the same lesion taken by different assessors should be very similar.

3. Ease of use
   It should easily be applied to clinical practice and be relatively inexpensive, and it should be able to assess two factors: whether demineralisation is present/absent, and measuring the severity of the lesion.¹⁸

There are macroscopic and microscopic methods for the detection and measurement of demineralised white spot lesions.

Macroscopic methods

These methods rely on the change in optical properties of demineralised enamel. Most photons are now backscattered within the lesion itself rather than by the dentine, and the backscatter is also greater. This gives the clinical appearance of the white spot.

When such a lesion is dried and the water filling its porosities is replaced by air, the refractive index decreases further, resulting in an even whiter appearance of the lesion,¹⁸ as is often manifested by the chalky appearance of etched enamel.

Macroscopic methods include:
1. Clinical examination
2. Photographic examination
3. Optical non-fluorescent methods/light scattering
4. Optical fluorescent methods

Clinical examination is used to evaluate lesions before, during or after Orthodontic treatment. While this method is simple, inexpensive and clinically valid, the main disadvantage associated with its validity is the difficulty in distinguishing those lesions caused by demineralisation from lesions due to other causes.

Photographic examination, routinely used in research studies, provides a permanent record, has the potential to eliminate bias and is quite versatile. The main disadvantages associated with this method is over-estimation of the incidence of lesions due to the reflection of the flash, and standardisation with regards to tooth wetness and lighting.

Light scattering can be measured by means of the Optical Caries Monitor. This method uses a light source and measures the backscatter with a densitometer. It is a convenient, non-destructive and clinically applicable method, but can also prove to be technique sensitive.

Optical fluorescent methods use the property of fluorescence, which is a function of light absorption. With demineralised white spot lesions there is more backscatter, less absorption of light, and thus a lower intensity of fluorescence. For this reason, lesions appear as dark areas. Typical examples of this method are: fluorescent dye uptake, the use of ultraviolet light, laser (argon laser/quantitative laser fluorescence), Diagnodent, or light (quantitative light-induced fluorescence).

Microscopic methods

Microscopic methods typically use one of two models:
1. The Caries Model
2. The in situ Caries Model

The Caries Model involves the placement of bands or brackets on teeth destined for eventual extraction but allowed to remain in the oral cavity for a period. The extracted tooth is then evaluated using a destructive method, such as microhardness testing, microradiography or polarised light microscopy.

The in situ Caries Model is more sophisticated and involves sections of enamel instead of whole teeth. The enamel sections are placed in a removable appliance, which is worn by a volunteer, after which the sections are removed and evaluated.¹⁸
PREVENTION DURING
ORTHODONTIC TREATMENT

Caries risk assessment
In order to implement effective preventative measures, it is important to identify those patients most at risk. This can be done using the Caries Risk Assessment tool. Factors noted during this assessment include:
1. Salivary flow rate
2. History of past enamel caries and caries incidence
3. Residence in fluoridated or non-fluoridated communities
4. Dietary patterns
5. Plaque scores
6. Microbial counts (Streptococcus mutans and Lactobacillus)

Patient education
This forms an essential part of the Orthodontic treatment plan. The Orthodontist, together with the Dental Hygienist, is responsible for this education.

Oral hygiene
A good oral hygiene regimen is often considered the first step in lesion prevention. This involves proper toothbrushing, with a fluoridated dentrifice. Dentrifices usually contain fluoride compounds such as sodium fluoride, monofluorophosphate, stannous fluoride, amine fluoride, or a combination of these. The fluoride concentration may vary, but should not fall below 0.1%. The cariostatic potential of these dentrifices is a direct result of two occurrences: its ability to improve oral hygiene, and fluoride ions being incorporated into the enamel surface of the tooth to form a fluorapatite crystal structure. This crystal structure has a lower solubility in the oral environment compared with the hydroxyapatite crystals of enamel, thus preventing dissolution and/or increasing remineralisation. However, in less compliant patients, regular use of a fluoridated dentrifice has been shown to be insufficient in prevention.

Fluoride
Supplemental sources of fluoride are recommended for non-compliant patients. This fluoride needs to be present in saliva and plaque fluid, in order to have maximum effect on enamel.

Salivary and thus plaque-fluid fluoride has been found to be significantly higher following two weeks of daily use of a sodium fluoride rinse. It has also been shown that daily rinses containing 0.05% sodium fluoride (NaF) or weekly rinses containing 1.2% acidulated phosphate fluoride significantly decrease the incidence of lesions during Orthodontic treatment. For this reason, the daily use of a fluoride rinse has been recommended, as was suggested 30 years ago.

In reality, however, Orthodontic patients who do not comply with proper oral hygiene procedures will most likely not comply with the daily use of fluoridated rinses. It has been shown that less than 15% of Orthodontic patients rinse daily as instructed. For these patients, topical fluoride, such as varnishes, solutions or gels, may be recommended.

The in-office application of high concentration fluoride varnish does not rely on patient compliance but may increase patient costs, increase chair time, and cause a temporary (yellowish) discoloration of teeth and gingival tissue. Nonetheless, both in vitro and in vivo studies have shown promising results. A particularly convincing in vivo study has shown that demineralised white spot lesions in Orthodontic patients may be reduced by up to 44.3% with twelve weekly applications of fluoride varnish.

Resin sealants
Resin sealants may be applied to tooth surfaces, before or after bonding, to seal susceptible areas. Unlike early generation sealants, newer sealants have proven to be more wear resistant (as they are highly filled), with effective sealing abilities and often additional protection is offered by the incorporation of fluoride.

While it is true that there is a significant decrease in the concentration of fluoride released by fluoride-releasing sealants with time, the sealant is able to be recharged with fluoride ions (using an acidulated phosphate fluoride solution). In vitro studies have shown that the application of Pro-seal (Reliance®) results in a significant decrease in lesions, more than an unfilled resin sealant or fluoride varnish. In vitro studies on the product found a 98% reduction in demineralised white spot lesions, and a 92% reduction in lesion depth. While these findings are impressive, in vivo studies to support this are yet to be published.

Adhesives
Glass ionomer (GI) cements were first introduced as bonding adhesives due to their chemical bonding and sustained fluoride release. However, because of their low bond strengths, resin particles were added to create resin modified glass ionomer (RMGI) bonding adhesives. These adhesives have relatively higher bond strengths, while still releasing fluoride. Furthermore, in vivo studies have shown no significant differences with regards to bracket failure rates or shear bond strengths (SBS) between RMGI bonding adhesives and composite (resin) bonding adhesives. These RMGI adhesives may play a greater role in Orthodontics in the near future.

Antimicrobials
Attempts have also been made to incorporate antimicrobials into bonding adhesives or primers, without negatively affecting bond strengths. It has been shown that no significant decrease in shear bond strength occurs when chlorhexidine is added to bonding primers or applied once all bonding procedures are complete, but does occur when added as a separate varnish layer during bonding. It has also been shown that 2.5% cetylpyridinium chloride added to bonding adhesives does not significantly affect tensile bond strength, and leads to an inhibition of bacterial growth for 196 days.
Interestingly, the combination of an antimicrobial self-etching primer and a fluoride-releasing adhesive has not been found to cause any significant reduction in shear bond strength, and has in fact been found to have a stronger shear bond strength than a conventional composite (resin) adhesive used with the usual acid-etch/primer procedure. 

Antimicrobials may also be applied as varnishes. An in vivo study found that the combination of a fluoride and chlorhexidine varnish or a fluoride varnish alone, results in a 30% reduction of lesions.

The combination of varnishes does not lead to significantly less lesions than fluoride varnish alone, however, the combination of varnishes has been found to be specifically associated with half as many lesions on maxillary incisors. For this reason, it is important to consider the use of products combining fluoride and antimicrobials in patients who cannot maintain optimal oral hygiene.

**NATURAL REGRESSION**

Under normal circumstances, dental enamel is in equilibrium with the oral environment, and saliva contains all the elements required for hydroxyapatite crystal growth. In this natural state, demineralisation and remineralisation occur continuously.

Insertion of fixed Orthodontic appliances disturbs this natural balance, but with the removal of the appliance, a favourable balance between demineralisation and remineralization is again restored and as a result, lesions may undergo natural repair. Natural repair will occur in the presence of adequate amounts of salivary and plaque calcium, phosphate and fluoride ions, and provided other etiological factors are favourable. However, it has been shown that this natural repair is limited.

**Time**

The most rapid reduction in lesion size without any treatment intervention has been found to occur during the first 12 weeks after fixed appliance removal, with little further reduction thereafter. Studies show that lesions reduce by one third after 12 weeks, and one half the size of the initial lesion after 26 weeks. Small lesions, however, have been found to show a rapid improvement six weeks after debonding, with little further improvement after six months.

**Lesion size**

The severity of the lesion is another aspect which affects regression. A study in which patients were re-examined six years after debonding showed that while 75% of the small demineralized white spot lesions regressed during the six year time period, 25% of the most severe demineralised white spot lesions did not regress and remained visible on the tooth surfaces.

It is important to note that clinical improvement in lesions is not just due to remineralisation, but also abrasion of enamel surfaces e.g. from toothbrushing. It has therefore been suggested that polishing or abrasion of dull and irregular enamel surfaces be done in order to expose more tightly packed enamel crystals which give a harder and glossier clinical appearance.

**MANAGEMENT AFTER FIXED APPLIANCE REMOVAL**

While prevention of demineralised white spot lesions is ideal, in reality, these lesions may still occur. In general, management of demineralised white spot lesions should always commence with the most conservative approach. If such an approach fails to resolve the problem, a more aggressive approach should be adopted.

**Conservative/non-invasive approaches**

These approaches aim at remineralisation of lesions. The remineralisation process depends on various factors, such as the status of the lesion, lesion depth and the remineralising potential of various products. Many products on the market claim to promote remineralisation.

**Fluoride**

Topical fluoride is well-known and was used before the invention of other remineralising products. It is available in many forms e.g. toothpastes, gels, rinses or varnishes. After debonding, a saliva-mediated remineralisation usually takes place and the application of topical fluoride helps with this. The absorbed fluoride attracts available salivary calcium and phosphate ions and together the three are able to form a new crystalline structure, fluorapatite or Ca$_{10}$(PO$_4$)$_3$F.

High concentrations of fluoride are important for prevention and seem to be beneficial in the management of lesions. However, the use of high concentrations on active lesions may cause surface hypermineralisation or plugging of diffusion pathways of enamel. The arrested lesion then remains the same size and often undergoes yellowish or dark brown staining. For this reason, many authorities warn against the use of high concentration fluorides.

Despite the large amount of in vitro and in vivo studies testing lesion regression with the use of fluoride, there still seems to be confusion when it comes to how large a role additional fluoride (other than conventional use of a fluoridated toothpaste) plays in remineralisation and the management of lesions after Orthodontic treatment.

**Casein phosphopeptide-amorphous calcium phosphate (CPP-ACP)**

The use of CPP-ACP, a milk derivate, is a novel remineralisation approach. The CPP-ACP complex can be found in a wide range of tooth creams, pastes, gels and mousses. CPP stabilizes high concentrations of calcium and phosphate ions, as nanoclusters of ACP, in supersaturated solutions at the tooth surface, which can then act in the subsurface part of enamel lesions and effectively promote remineralisation.

Most studies testing regression of lesions with the use of CPP-ACP containing products have shown that these
products are, indeed, effective at remineralisation.\textsuperscript{51-54} However, one of these studies have shown that the remineralisation is not superior to ‘natural regression’ with the daily use of a fluoride toothpaste.\textsuperscript{54}

**Casein phosphopeptide-amorphous calcium fluoride phosphate (CPP-ACFP)**

CPP-ACFP can produce an outcome similar to that of CPP-ACP, but is further enhanced by the inclusion of fluoride. CPP-ACP and fluoride work synergistically and result in the formation of CPP-stabilised amorphous calcium fluoride phosphate at the tooth surface\textsuperscript{55} which results in increased concentrations of bioavailable calcium, phosphate and fluoride ions in the correct ratio to form fluoroapatite.\textsuperscript{56} The above concept has been used in many products such as CPP-ACFP-containing chewing gums and tooth creams.

Studies testing lesion regression with CPP-ACFP containing products have shown that these products (like CPP-ACP containing products) cause remineralisation throughout the body of the lesion instead of just at the surface, and that they may be more effective than CPP-ACP containing products or fluoride.\textsuperscript{56, 57}

Many reasons could exist as to why some of the above remineralisation studies present with contrasting results. Duration of studies differ greatly, with some being as short as 10 days and others running for periods up to six months. The period and frequency of application of product also varies in some instances. While it was easy to stipulate the size of lesions (small or deep) in \textit{in vitro} studies, \textit{in vivo} studies sometimes fail to classify lesions on the basis of their size. And lastly, methods of assessment also vary from study to study based on the specific parameters investigated e.g. change in surface area, depth or microhardness of lesions.

**Micro-invasive or minimally-invasive approaches**

**Resin infiltration**

This newer treatment modality uses a micro-invasive infiltration technique to halt progression of lesions that are too advanced for remineralisation.

It involves steps of prophylaxis, etching, rinsing, drying, application of Icon-Dry®, application of Icon Resin-Infiltrant Smooth Surface® and curing. This product, Icon Resin-Infiltrant Smooth Surface® (DMG), was specifically developed for infiltration on the smooth surfaces of teeth (particularly demineralised white spot lesions following Orthodontic treatment).

Resin infiltration improves the appearance of demineralised white spot lesions as a result of the similar refractive index of infiltrated and sound adjacent enamel. Both short and long-term studies have shown that this approach is able to effectively camouflage demineralised white spot lesions.\textsuperscript{56, 59}

**Microabrasion**

Microabrasion has been used in the past for the removal of superficial, non-carious enamel defects, but more recently, its use has been suggested for the management of stabilised or long-standing demineralised white spot lesions caused during Orthodontic treatment.\textsuperscript{7} Steps involved in this approach include prophylaxis, isolation, abrasion with a slurry of 18% hydrochloric acid mixed in a fine pumice, followed by application of a fluoride varnish.\textsuperscript{60, 61}

It has been shown that microabrasion leads to a significant reduction (83%) of visible lesions and is thus effective in managing long-standing lesions.\textsuperscript{62}

**Invasive approaches**

**Restorations and veneers**

The most aggressive approach is composite restorations or porcelain veneers. With composite restorations, and even more so with veneers, a large amount of tooth structure must be removed and so local anaesthetic is required. These treatment modalities also cost more, compared with other less invasive approaches. For these reasons, such an approach should be reserved for the most severe of lesions which cannot be managed by any other approach.\textsuperscript{62}

**CONCLUSION**

Demineralised white spot lesions are an iatrogenic effect of Orthodontic treatment. Their prevalence in Orthodontic populations is particularly high, and since they affect teeth in the aesthetic zone, they prove to be a concern to both patient and clinician alike.

While prevention is ideal, high risk patients may still present with these lesions and their management becomes increasingly important. While there are many different treatment modalities to manage these lesions, prevention is optimal and should constitute the first line of defence.
References

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Do the CPD questionnaire on page 588

The Continuous Professional Development (CPD) section provides for twenty general questions and five ethics questions. The section provides members with a valuable source of CPD points whilst also achieving the objective of CPD, to assure continuing education. The importance of continuing professional development should not be underestimated, it is a career-long obligation for practicing professionals.

Online CPD in 6 Easy Steps

1. Go to the SADA website www.sada.co.za.
2. Log into the ‘member only’ section with your unique SADA username and password.
3. Select the CPD navigation tab.
4. Select the questionnaire that you wish to complete.
5. Enter your multiple choice answers. Please note that you have two attempts to obtain at least 70%.
6. View and print your CPD certificate.
Periodontal disease - risk factors and treatment options

SUMMARY

Periodontal disease (PD) encompasses both gingivitis and periodontitis. Both are initiated by plaque and are influenced by the immune and inflammatory responses of each individual. In addition, PD is modified by several risk factors including smoking, medications, alcohol, age, gender and systemic diseases.

Gingivitis affects 50–90% of adults worldwide and is reversible by simple, effective oral hygiene and lifestyle changes. Between 10–15% of the global adult population suffer from progressive periodontitis, which if left unattended, results in halitosis, pain and loss of teeth.

As dental plaque is the principal etiological factor in the pathogenesis of PD, effective oral hygiene and plaque removal is the most important strategy in the prevention of this disease. There is also evidence that PD has several modifiable risk factors in common with certain non-communicable chronic diseases like diabetes. Therefore, to prevent PD, the approach of controlling the common risk factors could be an effective strategy.

Potential risk-factor entry points are reduction of tobacco use, reduction in consumption of harmful levels of alcohol, a healthy diet and good nutrition and improvement of personal hygiene. Whilst PD is not contagious it can become extremely common and debilitating, given the ideal environment. This paper discusses the risk factors and identifies options by which PD can be prevented and reduced.

INTRODUCTION

Periodontal disease (PD) encompasses a cluster of diseases that result in inflammatory responses and chronic destruction of the tissues that surround and support the teeth, namely the gingiva, periodontal ligament, cementum and alveolar bone (collectively referred to as the “periodontium”). It therefore refers to both gingivitis and periodontitis.

Gingivitis is an inflammatory condition of the soft tissues (gingiva) surrounding the teeth whilst periodontitis involves the destruction of the supporting structures of the teeth and periodontium.

Clinical signs of a healthy periodontium include: maintenance of a functional periodontal attachment level, minimal or no recession with no loss of interproximal bone; and, where present, functional dental implants, all in the absence of inflammation.

Both gingivitis and periodontitis are initiated by plaque and are influenced by the immune and inflammatory responses of the individual. Both conditions are modified by several factors including smoking, medication, age and systemic diseases.

Gingivitis affects 50–90% of adults worldwide (Fig. 2) and is readily reversible by simple, effective good oral hygiene and lifestyle changes.

Gingivitis can be defined as the presence of gingival inflammation, whereby the gum can appear reddened, swollen, and may easily bleed, but without loss of connective tissue attachment.
Periodontitis can be defined as the presence of gingival inflammation at sites where there has been a pathological loss of attachment. This loss of attachment contributes to pocket formation. The rate of progression of periodontitis is neither predictable nor steady.

Calculated plaque (calculus) does not have a major role in the pathogenesis of periodontal disease, although it does act as a ‘retention web’ for bacteria and interferes with the ability to perform personal oral hygiene.

Studies have shown that microorganisms quickly colonize clean tooth surfaces after cessation of oral hygiene procedures and that within a few days microscopic and clinical signs of gingivitis can be observed. At this point these changes can be reversed, provided the individual resumes adequate tooth cleaning procedures.

Gingivitis is a result of the microorganisms within the plaque releasing products that induce tissue inflammation. Most individuals develop clinical signs of gingivitis after 10-20 days of plaque accumulation. It has been noted that not all patients will develop periodontitis following gingivitis and therefore those who do must have a unique response to microbial plaque.

The teeth specificity and predilection in periodontal disease probably localize to the sites of retention of plaque where oral hygiene is inadequate or in areas of calculus formation, restorative overhangs or poor margins of crowns. In normal situations, more than six months may pass before the lesion of gingivitis changes to periodontitis.

Periodontal disease is considered to have multiple risk factors. According to one author the term “risk factor” refers to an aspect of personal behaviour or lifestyle, an environmental exposure, or inherited characteristics, which on the basis of epidemiological evidence is known to be associated with a health related condition. Risk factors therefore are part of the causal chain for a particular disease or can lead to an exposure of an individual to a disease and therefore the presence of risk factors implies a direct increase in the probability of the disease occurring.

Destructive periodontal disease is a consequence of the interaction of genetic, environmental, host and microbial factors. Risk factors for periodontal disease include genetics, age, gender, smoking, socioeconomic factors and some systemic diseases.
Age

The prevalence of periodontal disease is seen to increase with age, while the extent and severity also increases with advancing age.\(^3\)

However, it is not clear whether becoming older is related to an increased susceptibility to periodontal disease or whether the cumulative effects of disease over a lifetime may explain the increased prevalence of disease in older people.\(^2\)

Some authors suggest that up until 70 years of age the rate of periodontal destruction is the same throughout adulthood; age per se is not a risk factor for people under 70.\(^10, 11\)

In South Africa life expectancy increased from 59 years in 2000 to 63.6 years in 2016.\(^12\) This increase in life expectancy is considered mainly due to the mass roll out of Antiretroviral (ARV) treatment for HIV and its positive consequences.

The increased life expectancy would result in an increased geriatric population requiring oral health treatment especially for periodontal diseases.

Socioeconomic status

A possible relationship between PD and socioeconomic status (SES) was found in several studies.\(^13-15\) Gingival condition is directly related to SES with evidence of poor gingival health and is more prevalent in persons from a low SES.

The relationship between SES and periodontitis is less direct. It can be certain that gingival health is better among individuals with higher education and with a more secure income.\(^16\) South Africa has a high unemployment rate of around 27% and coupled to this is a high disparity between the rich and the poor.\(^17, 18\)

These factors combined with poor education and low levels of knowledge all indicate that the prevalence of periodontal diseases will increase in the next few years, placing an added burden on the public oral health sector.

Race and gender

Destructive periodontitis is consistently more prevalent in males than females which could be due to lifestyle choices of males which include an increased alcohol and smoking consumption.\(^2\)

PD also has been reported to be more prevalent amongst blacks than whites with a Brazilian study reporting that groups of blacks have a three times higher risk of periodontal destruction compared with whites of the same age cohort.\(^14\)

This could be due to lifestyle choices and genetic factors and may be applicable to similar South African populations. The distribution of PD within countries also differs according to race or ethnic group regarding prevalence and severity.\(^19\)

South Africa comprises 81% Black African and just over 51% of all South Africans are females.\(^20\) These factors also impact on the prevalence of PDs and clinicians should be aware of these risk factors when examining and treating their patients.

Smoking

A consistent, positive association between smoking and loss of periodontal attachment has been reported and confirmed in many studies.\(^21, 22\) Smoking alone accounts for more than 50% of PD cases.

The prevalence of smoking is higher in the uneducated, poorer communities and low-income earners compared with their more affluent and educated counterparts.\(^9\) Other studies have reported that smokers were five times more likely to have a periodontal emergency compared with non-smokers.\(^23\)

The disease is more prevalent, extensive and severe among current smokers, and occurs least amongst those who have never smoked. It has been proven that those who smoke over many years have considerably higher periodontal disease rates, and that this occurs even with cannabis smoking, suggesting that it is the smoking, rather than any specific characteristic of tobacco, that is responsible.\(^24\)

Although South Africa has seen a decrease in the national tobacco consumption, tobacco use remains prevalent in certain underprivileged and marginalized communities.\(^25\)

Added to this, studies have reported an increase of electronic cigarette and water pipe smoking and long term research is required on the effects of these innovative habits on the oral cavity.\(^26\)

Genetics

There is a growing body of evidence from studies that genetic factors predispose individuals to periodontal disease. This is seen especially in the rare and more severe forms of periodontitis like early onset periodontitis, now classified as aggressive periodontitis, where family studies have provided good evidence for a prominent genetic role.\(^28, 29\) A gene mutation for pre-pubertal periodontitis has been identified.\(^30\)

Systemic disease

Systemic disease can adversely affect host defense systems and therefore can act as risk factors for PD.\(^2\) Among the associations observed between oral health status and chronic systemic diseases, the link between PD and diabetes mellitus is the most consistent.\(^31\)

Periodontal diseases are well established as a complication of diabetes, and have been considered the sixth most common complication of diabetes.\(^32, 33\)

In a study of Brazilian individuals with poorly controlled type 2 diabetes, significantly higher levels of periodontal pockets and loss of attachment were found compared with controls.\(^34\)
Although HIV disease has a relatively minor effect on the progression of chronic periodontitis compared with other pathogenic factors, patients who are HIV-positive and immunosuppressed can present with distinctive forms of necrotising gingivitis and periodontitis.

Diseases of the oral cavity strongly associated with HIV are: linear gingival erythema (LGE), necrotising ulcerative gingivitis (NUG) and necrotising ulcerative periodontitis (NUP). It has been proven that the presence of NUP and NUG may offer a significant diagnostic, as well as a prognostic, value.

Periodontitis and stress

It is well known that cardiovascular disease, diabetes, and other chronic diseases are related to psychosocial factors, but there is also evidence that stress is linked to periodontal disease.

Stressful life events, and marital problems are associated with PD, possibly through physiologic responses that increase susceptibility and reduce the immune response.

Periodontitis and pregnancy

Studies have demonstrated that PD have been shown to increase the risk of adverse pregnancy outcomes such as premature birth and low birth weight. Uterine contractions are stimulated by oxytocin, which is produced by the hypothalamus and by prostaglandins produced by the placenta. This process normally occurs in the third trimester and leads to birth. However, chronic infection can stimulate the inflammatory process, which leads to elevated amniotic levels of prostaglandins, TNF-α, Interleukin-1 and -6. These mediators then lead to premature rupture of membranes and pre-term labour. Other work has suggested that periodontal pathogens may travel from the gingival sulcus to the placenta and stimulate preterm birth.

Alcohol

High alcohol consumption increases the risk of a wide variety of conditions such as increased blood pressure, liver cirrhosis, cardiovascular disease, diabetes, and cancers of the mouth. Recent research also indicates that excessive alcohol consumption is associated with increased severity of periodontal disease.

Alcohol consumption, tobacco use, and unhealthy diet commonly go together. People who consume tobacco are more likely to drink alcohol and eat a diet high in fats and sugars but low in fiber and polyunsaturated fatty acids. Those who have a high consumption of tobacco and alcohol are thus more likely to be at a higher risk of severe periodontal disease and oral cancer.

Local risk factors

Any plaque retentive feature such as restoration overhangs or deficiencies, may contribute to the local risk of periodontal disease.

CONCLUSION

Risk factors work to change the susceptibility or resistance of individuals to the disease. Risk factors for periodontal disease can be both systemic and local, such as smoking; medical conditions, poorly controlled diabetes, possibly obesity and stress play a significant role in the initiation and progression of PD.

The modification of these risk factors plays a strategic role in the management of periodontal disease, accepting of course that some, such as race or genetics, cannot be changed. The identification of high-risk patients is therefore essential in the ultimate management and treatment of PDs.

As has been demonstrated, periodontal disease is highly linked to systemic diseases such as diabetes and HIV. There is also evidence that periodontal disease has several modifiable risk factors in common with certain non-communicable chronic diseases and therefore to prevent periodontal disease one can use the common risk factor approach.

It is therefore essential that clinicians adopt a holistic and systemic approach to identify high risk patients and to recommend behaviour and lifestyle changes to attain the common goal of preventing and managing PDs.

References

Maxillofacial Radiology 165

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CJ Nortjé

A 30-year old female presented with a swelling extending between 35 and 43, which had grown over the past ten months (Figs. A,B,C), with intermittent pain. Figures D,E,F show a similar case in a 15 year old male patient. Describe the radiological features and list your differential diagnosis.

INTERPRETATION

The cropped pantomograph (Fig.B) shows a multilocular radiolucency of an expansive tumour containing varying calcifications. Figures D&E show a well demarcated multilocular lesion in the body/ramus region of the left mandible, with two displaced molar teeth, resorption of the roots of 34 and 35 and tooth-like calcifications at the borders of the lesion. The coronal T2 MRI image (Fig.F) shows a multilocular hyper-intense lesion, with no discernible calcifications. Diagnosis: the rare tumour, odonto-ameloblastoma (OA), first reported by Kemper and Root (1944). Shafer et al (1983) described this as an odontogenic neoplasm of mixed tissue origin, consistent with both ameloblastoma and odontoma. Histologically, a typical ameloblastoma component whilst the odontoma element may be either compound or complex. Shafer et al (1983) emphasized that this is one neoplastic process with relatively highly undifferentiated tissues. Occurring most commonly in the second decade of life, it may vary in size. Smaller lesions occur between the teeth, confined to the alveolar bone between the crest of the ridge and the teeth apices (Figs. A,B&C). Buccal expansion of the cortex is common, even for smaller lesions. The odontoma component presents various stages of development, early lesions being radiolucent with radiopaque flecks. More mature lesions are better developed odontoma, resembling teeth or as a nonspecific complex odontoma mass. Importantly, this is an aggressive tumour, treated precisely as an ameloblastoma. The differential diagnosis should include: ameloblastic fibro-odontoma, Pinborg tumour, calcifying odontogenic cyst and adenomatoid odontogenic tumour.

References

Hamartomas in the opercula of four unerupted primary molars

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N Potgieter¹, L Robinson², I Middleton³

ABSTRACT

Odontogenic lesions in the opercula may result in delayed eruption of primary molars.

Case report

This case presents a rare occurrence of delayed eruption of four primary molars causing interference with normal function. The lesions were surgically excised to expose the underlying first deciduous molars. Histopathological analysis of the excised tissue revealed hamartomatous lesions. These lesions appeared histologically identical to those of an odontogenic giant cell fibroma, consisting of odontogenic epithelial islands with scattered giant cells and histiocytes in a surrounding dense fibrous connective tissue stroma. Occasional dyskeratotic cells were also noted with an intermixed mild, chronic inflammatory cell infiltrate.

Conclusion

Eruption cysts are traditionally left untreated to resolve spontaneously with the eruption of the underlying teeth. However, lesions that stay unresolved or interfere with function should be surgically excised and sent for histological analysis. The presence of hamartomas in the operculum should be considered as a differential diagnosis in persistent unerupted deciduous and permanent teeth.

Keywords

Eruption cyst, hamartoma, delayed eruption, odontogenic giant cell fibromatosis

INTRODUCTION

A dome shaped swelling in the mucosa overlying an unerupted tooth is commonly diagnosed as an eruption cyst (EC).¹ An EC is a dentigerous cyst that develops when the dental follicle separates from the crown of an erupting tooth that is still enfolded in the soft tissue overlying alveolar bone.¹,²

The lesions are usually associated with unerupted deciduous mandibular central incisors and permanent first molars, but rarely with deciduous molars.³

Eruption cysts are usually asymptomatic and are conservatively left to rupture spontaneously when the underlying tooth erupts.¹, ⁴, ⁵ However, surgical intervention should be considered if the EC's cause pain or discomfort, infection or interfere with normal function (i.e. breastfeeding/mastication).⁴,⁶

Persistent multiple EC's on all four first deciduous molars of one patient is a rare occurrence. This paper describes the surgical management and final diagnosis of such a case.

CASE REPORT

A healthy, 19-month-old boy presented with his mother. The main complaint was that swellings in the boy’s mouth were interfering with breastfeeding. According to the history, the swellings had been present and had increased gradually in size for approximately eight months. Initially, the referring dentist explained to the mother that the swellings would self-correct on eruption of the primary molars. After a year had lapsed without self-correction and the boy started experiencing functional problems, the dentist referred the patient for further investigation.

Upon clinical examination, dome-shaped lesions were noted in all four quadrants in the areas where the first deciduous molars were expected to erupt. The swellings were the same colour as surrounding mucosa with a slight bluish tint and were soft and tender on palpation (Figures 1A and B). The patient could not close the mouth completely due to the size and location of the lesions. No other clinical pathological findings were noted. Due to the patient’s age and cooperation it was decided not to expose the child to radiation for radiographic examination. A clinical diagnosis of multiple eruption cysts was made.

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The child was examined by the anaesthesiologist and cleared for general anaesthesia. All four cysts were surgically removed through elliptical incisions made with a surgical blade (Figure 2A).

Clear fluid was drained once the mucosa was cut. The incisions were made larger than normal to prevent tissue contact and reattachment. The excised tissues were sent for histopathological analysis. Bleeding was easily controlled without any sutures or haemostatic agents (Figure 2B). Post-operative instructions were given to the mother and follow-up visits were scheduled.

At the one-week clinical follow-up (Figure 3A), the tissues surrounding all four teeth were sound with slightly rolled borders and marginal inflammation. All four deciduous first molars were clearly visible. The mother reported an improvement in feeding and did not report pain or discomfort experienced by the boy. At the three-week follow-up (Figure 3B), all signs of gingival inflammation had cleared and the infant functioned and fed well.

Histological analysis confirmed the diagnosis of multiple eruption cysts with the presence of odontogenic giant cell fibromatosis in the overlying mucosa. The lesions showed the presence of odontogenic epithelial islands with scattered giant cells and histiocytes in a surrounding dense fibrous connective tissue stroma. Occasional dyskeratotic cells were seen, which were more abundant in one fragment, and a mild, chronic inflammatory cell infiltrate was present within the surrounding fibrous connective tissue (Figures 4A to D).
Figure 4:

(A) Eruption cyst: Low power magnification showing a mucosal fragment with an underlying cystic lesion lined by non-keratinizing stratified squamous epithelium (H & E, x4).
(B) Cyst wall: Higher magnification of the cyst wall shows the presence of fibrous connective tissue with scattered giant cells and odontogenic islands representing odontogenic giant cell fibromatosis in a background of a mixed, chronic inflammatory cell infiltrate (H & E, x20).
(C) Odontogenic giant cell fibromatosis: High magnification highlights the giant cells and odontogenic islands with background inflammatory cells (H & E, x40).
(D) Dyskeratotic cells: Focally the eruption cyst shows the presence of numerous dyskeratotic cells (H & E, x40).

DISCUSSION

Biopsies from the opercula overlying the impacted first deciduous molars showed increased odontogenic epithelial rests that varied in size and shape. These epithelial rests may obstruct normal tooth eruption.\(^7\)

These lesions have been described using terms such as “odontogenic giant cell fibromatosis (OGCF)” and “pericoronal hamartomatous lesions”.\(^8\) The term hamartoma can be defined as a “non-neoplastic, unfocal/ multifocal developmental malformation”.\(^9\) While the pathogenesis of hamartomas remain speculative, it is known that these tissues are most commonly derived from the mesoderm.\(^10\)

The hamartomas in our patient corresponded to OGCF, a term first coined by Philipsen et al.,\(^7\) to describe an odontogenic hamartomatous lesion present within the opercula of permanent first and second molars with delayed eruption. Histologically, OGCF presents as a non-encapsulated lesion consisting of dense fibrous connective tissue containing a large number of spindle-shaped or stellate cells and large multinucleated cells, proliferating strands and islands of odontogenic epithelium with occasional squamous cell metaplasia and no calcifications present.\(^8\)

These multinucleated giant cells and stellate cells, found in great number in these lesions, are histologically identical to those described in giant cell fibromas.\(^2\) The patient in question presented with a focal area of dyskeratotic cells in the wall of the cyst which is a rare phenomenon, but is well documented in odontogenic cysts. The strong association between OGCF and mandibular impacted molars suggests that the biology of these hamartomatous lesions differs from that of “classic” odontogenic tumors.\(^7\)

Both Philipsen et al.\(^7\) and Yonemochi et al.\(^11\) concluded that the presence of hamartomatous lesions in the pericoronal areas of teeth may cause delayed eruption which can interfere with the pathway of an erupting tooth, causing tissue derangement and remodelling.\(^7,8,11\)

The odontogenic hamartomas presenting in this case were successfully resolved following conservative surgical incision. Odontogenic hamartomas are non-neoplastic and once they have been resolved, there is little chance of recurrence.\(^12\)

CONCLUSION

In this case report, the delayed eruption of primary molars, which is uncommon, is described and is attributed to eruption cysts, characterised by OGCF hamartomatous lesions in the opercula. Failure to spontaneously resolve and a reduced ability to breastfeed resulted in a decision to treat the cysts surgically.

Conservative surgical intervention resulted in the successful eruption of all four primary molars, improved function and overall quality of life.

Disclosure

The authors declare no conflicts of interest related to the case reports depicted in this article.

References

Depression is among the most prevalent chronic diseases worldwide. It is a well-established and important risk factor for many systemic conditions, including obesity, sleep disturbance, and chronic diseases. In South Africa, depression rates reported from different studies and settings have ranged from 18% -35%. Oral diseases have also been associated with depression.

Most studies have drawn attention to the contribution of poor oral health status to depression, although there are also published studies whose results have suggested that there exists a bi-directional relationship between oral health and mental disorders.

In view of the relevance of depression to oral health, and the lack of consensus about the association between these conditions, a study that summarizes the literature is of great importance. Cademartori and colleagues (2018)1 reported on a systematic review that sought to investigate any association between depression and oral health.

This review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. An electronic search was conducted in PsycINFO, PubMed, Scielo, Scopus, and Web of Science, without language restrictions and with no initial date restriction until 20 April 2018.

Based on the inclusion and exclusion criteria, two reviewers independently screened titles and abstracts. In case of disagreement, a consensus was determined by discussion after a comparison of the lists of the included studies.

In the next step, the same two reviewers read the full texts of the articles, reviewing according to the inclusion and exclusion criteria. When articles were excluded reasons for the exclusion were recorded. The reviewers also performed a hand search in the reference lists of the included studies. Original cross-sectional and longitudinal prospective and retrospective observational studies were included.

To be included, studies should have investigated an association between major depression disorder and oral health conditions (dental caries and/or periodontal disease and/or tooth loss). In order to ensure adequate power to detect statistical significance and the representativeness of the sample, the study was expected to present details regarding sample selection, or the study should have clearly described the representativeness of the sample (e.g., representative subsample of a national survey).

Studies of specific sample, such as psychiatric populations, individuals with periodontitis, children or adolescents, populations comprising only pregnant women or people with learning disability were not included. Studies that described their sample as “convenience” were also excluded. Additionally, case-control studies, reviews, technical reports, case reports and series, abstracts from conferences, letters to the editor, animal studies, in vitro studies, and qualitative studies were not considered.

As criteria for a diagnosis of depression, studies with a primary diagnosis of depression, including clinical diagnoses or diagnostic criteria through validated tools, were selected.
When measures of lifetime and current diagnoses were presented, a preference was given for current diagnoses, again using validated tools. Other mental disorders and dental phobias were not included.

In relation to oral diseases, only those considered as public health problem with a global burden were included. Therefore, studies that presented dental caries, periodontal disease, or tooth loss as outcome or as exposure were considered. As inclusion criteria, oral health should have been evaluated by self-reported measures or clinical diagnoses. When these were presented, a preference was given for clinical diagnoses.

In addition, when more than one category of oral disease was presented; the most severe was chosen. Tooth loss and edentulism were considered separately. Studies that explored other oral diseases including erosion, temporomandibular dysfunction, or xerostomia were not included. Studies of qualitative measures related to dental outcomes such as poor oral health were also excluded.

Data related to study identification (first author's name and year of publication), location, sample characteristics, the study design, and information regarding exposure and outcomes variables were extracted. The statistical methods and effect measures used, adjustments performed, and confounding factors which had been considered were recorded. When necessary, authors were contacted and questions about the study were clarified.

The methodological quality assessment of the included studies was performed using the Critical Appraisal Checklist for observational studies referred by The Joanna Briggs Institute (JBI). The checklist is comprised of ten items, which are to be answered with “Yes,” “No,” or “Unclear” by reviewers. The sum of the number of “Yes” answers defined the overall score for each study, ranging from 0 to 10. Studies were classified as follows: low quality (0–3 scores); medium quality (4–6 scores); and high quality (7–10 scores). The data extraction and the quality assessment process were independently performed by the same two reviewers, who matched the information collected and discussed cases of disagreement.

For each oral disease, distinct meta-analyses were performed, taking into consideration the direction of association presented in the study. If a study presented two or more variables of interest, data were also analysed independently. If a study presented more than one category for the variable of interest, the most severe category was considered.

For meta-analysis, preferably adjusted results were included. When that was not possible, crude result estimates were considered or calculated. Odds ratio (OR) was used to measure effect size with 95% confidence interval (CI). Relative risk measures presented in studies were converted to ORs. Fixed- and random-effects models were used to estimate pooled OR. The random-effects model was chosen in the presence of heterogeneity. Heterogeneity was evaluated with the I² statistic and considered when I² was more than 50%. Sensitivity analyses were used to observe the effect of each study on the pooled results.

RESULTS

The searches performed in the electronic database presented 2504 potential articles, of which 1127 were duplicates. A first screening was performed for title and abstract in 1377 articles, and resulted in 44 studies. In the second screening, the studies received full-text reading, and 28 publications were excluded. Therefore, 16 studies were included in this systematic review and 15 studies were included in this meta-analysis.

Dental caries versus depression

Two studies assessing association between dental caries and depression were included in this systematic review. The studies considered association depression as an exposure variable and oral health as an outcome variable. The studies were published between 2012 and 2015, with sample sizes ranging from 390 to 4667 individuals with a high methodological quality. Both had a cross-sectional design and assessed depression as a current measure through validated scales, Beck’s Depression Inventory (BDI) and Geriatric Depression Scale (GDS).

According to a pooled estimate, depression was associated with dental caries (OR 1.27; 95% CI 1.13–1.44; p< 0.05).

Periodontal disease versus depression

Five studies testing the association between periodontal disease and depression were included in this systematic review. The sample sizes ranged from 701 to 63540 individuals. Depression was measured using validated scales, specifically the BDI, the GDS, and the World Health Organization Composite International Diagnostic Interview, auto version 2.1 (CIDI-Auto). In the meta-analysis, the pooled estimate does not show an association (OR 0.96; 95% CI 0.84–1.10; p>0.05) between depression and periodontal disease. Heterogeneity among studies was not found (0%).

Tooth loss versus depression

Five studies investigated the association between tooth loss and depression. The studies were published between 2012 and 2015, with sample sizes ranging from 1553 to 5,419,019 individuals.

In the meta-analysis, the pooled estimate revealed an association between depression and tooth loss (OR 1.31; 95% CI 1.24–1.37; p< 0.05). Individuals with depression presented 1.31 times higher odds of tooth loss. A prevalence of 13.4% of heterogeneity between studies was found, which is considered as low heterogeneity.

Edentulism versus depression

Seven studies had investigated the association between edentulism and depression. The studies were published between 2012 and 2017, with sample sizes ranging from 768 to 201953.

Two meta-analyses were performed according to the direction of association applied in analyses in the studies. A positive association was found on the pooled estimate for depression as an exposure variable for edentulism.
It was found that depressive individuals presented 1.17 times higher odds of edentulism (OR 1.17; 95% CI 1.02–1.34; p<0.05).

A second meta-analysis was performed for those studies in which edentulism were tested as an exposure variable for the development of depression. The pooled estimate revealed an association between edentulism and depression (OR 1.28; 95% CI 1.06–1.55; P<0.05).

CONCLUSION

The results of this systematic review and meta-analyses show a positive association between depression and oral diseases, specifically dental caries, tooth loss, and edentulism, in adults and elders.

More longitudinal studies are required to test causal and temporal relationship between depression and oral health status.

Implications for practice

Mental and oral health are among the main disabilities worldwide. This high quality systematic review provides evidence of the link between oral health and mental disorders, highlighting the importance for both clinicians and policy makers to consider the patient’s psychological status in the management of oral health conditions.

Reference


2. Does the use of CAD/CAM technology produce dentures with improved fit?


Removable complete dentures remain the least invasive and most cost-effective option for the prosthodontic rehabilitation of edentulous patients. A crucial factor determining the quality of removable dentures is the denture fit. Well-fitting dentures show a higher primary wearing comfort and reduce the occurrence of traumatic ulcers. Tissue-congruent denture fit is the most important key factor for good retention in removable complete dentures. Denture retention affects the masticatory performance and speaking ability and thereby has a strong impact on the patients’ quality of life. Therefore, achieving maximal tissue congruence should be one of the main goals in complete denture fabrication.

Before the introduction of CAD/CAM technology into removable prosthodontics, the congruence between denture base and denture-bearing tissues was always impeded by the resin’s polymerisation shrinkage. The shrinkage causes distortions of the denture base and therefore has a negative impact on fit and retention of removable complete dentures. In CAD/CAM fabrication, on the other hand, the manufacturing process is subtractive: The denture bases are milled from fully polymerised acrylic resin pucks and are therefore not subject to shrinkage or distortion phenomena anymore.

Steinmass and colleagues from Austria (2018) reported on an in vitro study that sought to investigate whether CAD/CAM fabricated denture bases have a higher congruence with the denture-bearing tissues than conventionally processed denture bases. Therefore, the null hypothesis for this study was that there is no difference in the precision of fit between CAD/CAM fabricated and conventional dentures.

MATERIALS AND METHODS

This in vitro study included maxillary study casts originating from ten edentulous patients, serving as master casts. Different anatomical situations were included: moderate to strong alveolar resorption with or without undercuts and high and shallow palates, as well as granular and smooth mucosal surfaces. Five dentures were fabricated from each of these ten master casts: four different CAD/CAM dentures and one conventional denture.

The four different CAD/CAM dentures per cast were provided by the four CAD/CAM denture manufacturers (Avadent Digital dentures [Global Dental Science Europe], Baltic Denture System [Merz Dental GmbH], Whole You Nexteeth [Whole You Inc], Wieland Digital Dentures [Wieland Dental + Technik GmbH & Co]). Each company produced one denture per master cast.

The anatomical information required for manufacturing the study dentures was obtained from master cast scans by AvaDent, Baltic Denture System and Wieland Digital Dentures. The Whole You Nexteeth system could not process master cast scans.

Therefore, impressions of the master casts had to be generated using Imprint 4 Super Quick Heavy and Light polyvinylsiloxane impression material (3 M) and DENTCA impression trays (Dentca INC). The impression scans could then be integrated into the Whole You Nexteeth digital workflow.

The ten conventionally manufactured dentures fabricated from each of the ten original master casts served as a control group. The conventional dentures were made in compressed mould technique. For the mould, class IV gypsum was processed according to the manufacturer’s instructions and then isolated with a plaster-against-resin separating fluid. The denture bases were made from heat polymerising resin in the recommended long-term heat polymerisation cycle (75 °C water bath for 8.5 h). All study specimens were finished only on the oral surfaces, while the mucosa-sided surfaces were left unfinished, as is customary in clinical usage. Before analysis, all dentures were stored in sealed beakers containing 200 ml of deionised water at 37.0 °C for 7 days in darkness.
Prior to the fabrication of the conventional dentures, which would involve the destruction of the casts, the master casts were scanned using a 7Series Dental Wings scanner, after applying a thin and homogenous layer of Shera scanspray. The generated digital data (3D meshes) was processed in STL-format. The same procedure was applied to the mucosal surfaces of each study denture.

After standardised cropping of the meshes, the mucosal denture-base surfaces were matched with the master cast surfaces. The measurement points were set at minimal distance, resulting in a 2-mm mesh. Unsigned absolute mismatch-values were used to avoid the neutralisation of positive and negative values. Besides calculating the overall mean mismatch, the master cast surface was also divided into five functionally relevant sections (posterior palatal seal, anterior and lateral border seal, alveolar ridge, tubera maxillaria and palate) to evaluate the region-specific mismatches.

Following these analyses, all specimens were submitted to a thermocycling protocol simulating six months of intraoral use. The dentures underwent 5000 cycles of alternating immersion in deionised water with 5 and 55 °C. After thermocycling, the scanning and matching procedures were repeated, following the aforementioned protocol.

RESULTS

Overall denture fit

The deviations between mucosal denture surfaces and the corresponding master cast surfaces were measured at an average of 650.2, SD = 86.1 measuring points per denture. There were no outliers in the data, and the deviation values were normally distributed. Conventionally fabricated dentures showed a mean deviation of 0.105 mm, SD = 0.019 from the master cast. All CAD/CAM fabricated dentures had lower mean denture base incongruences than did the conventionally fabricated dentures. AvaDent Digital Dentures showed the greatest congruence with the master cast surface with a mean deviation of 0.058 mm, SD = 0.005. Wieland Digital Dentures showed a mean deviation of 0.068 mm, SD = 0.005, Whole You Nexteeth prostheses showed a mean deviation of 0.074 mm, SD = 0.011 and Baltic Denture System prostheses showed a mean deviation of 0.086 mm, SD = 0.012.

The mean incongruence values indicated statistically highly significant differences among the manufacturers, F(1, 873, 16,855) = 28.878, p < 0.0005, partial η² = 0.762.

AvaDent Digital Dentures, Wieland Digital Dentures and Whole You Nexteeth prostheses showed a highly significantly better denture base congruence than did the conventional dentures. Baltic Denture System prostheses also showed a more precise fit than the conventionally fabricated dentures, but the mean value difference was not statistically significant.

Compared with the conventional dentures, AvaDent Digital Dentures had a difference of mean misfit of 0.047 mm (95% Cl [0.023, 0.071], p < 0.0005), Wieland Digital Dentures of 0.037 mm (95% Cl [0.016, 0.058], p = 0.001) and Whole You Nexteeth dentures of 0.031 mm (95% Cl [0.008, 0.054], p = 0.008). The fit of all three CAD/CAM systems was statistically highly significantly better than in the control group. The mean misfit of Baltic Denture System was 0.019 mm (95% CI [−0.007, 0.046]), smaller than in conventional dentures, but this difference, as previously mentioned, was not statistically significant (p = 0.258). Therefore, the null hypothesis had to be rejected.

Region-specific misfit

The functional regions with the most precise fit in conventional and in almost all the CAD/CAM dentures were the alveolar ridge and the palate. The greatest extent of misfit was found in the posterior palatal seal regions and the anterior and lateral seal regions. AvaDent dentures and Wieland Digital Dentures, which had the highest precision of fit, also showed rather low deviations in the posterior palatal seal regions (0.057 mm, SD = 0.005 and 0.071 mm, SD = 0.008) and the anterior and lateral seal regions (0.084 mm, SD = 0.017 and 0.088 mm, SD = 0.011, respectively). Whole You Nexteeth prostheses showed the highest values of deviation in the posterior palatal seal region (0.166 mm, SD = 0.044).

The differences in the denture base congruence between the various functional regions were statistically highly significant in conventional dentures and also in all CAD/CAM dentures (p < 0.01). The congruence in the palatal region was statistically significantly higher than in the anterior and lateral seal region, in all groups (p < 0.05). In Whole You Nexteeth prostheses, the posterior palatal seal region showed a statistically significantly higher misfit than all other regions (p < 0.05).

Post-thermocycling misfit

Thermocycling did not have a statistically significant impact on the precision of fit. Not only were the changes in fit within the imprecision of the scanning and matching processes, but there was also no reproducible trend towards increased or diminished precision of fit, neither for conventional, nor for CAD/CAM dentures.

CONCLUSION

Computer-aided design and manufacturing produced dentures with higher tissue congruence than with conventional denture fabrication. AvaDent Digital Dentures, Whole You Nexteeth prosthesis and Wieland Digital Dentures had a significantly higher precision of denture base fit than did the conventional dentures.

Implications for practice

The investigated CAD/CAM systems were able to reproduce the master cast surfaces very precisely with even more precision than the conventional manufacturing protocol. The findings of the present study explain the observed clinical excellence of CAD/CAM-fabricated dentures regarding retention.

Reference

Dealing with difficult patients

Interactions between dentists and their patients can sometimes be challenging. Most of us have had consultations and treatment sessions where the interaction and outcome could have gone better, and are often a matter of regret for either the patient or clinician, or both, occasionally ending with a formal complaint. Dr Alasdair Mckelvie, Head of Dental Services Africa at Dental Protection looks at some of the issues that can bring about difficult interactions with patients and gives tips on how best to deal with these situations.

A recent survey of dental members in South Africa undertaken by Dental Protection revealed that 50% of dentists are experiencing more demanding patients with higher expectations and more than 80% agreed or strongly agreed that patient expectations have increased in the last five years.

It is therefore unsurprising that an increasing number of complaints which have been laid before the Health Professions Council of South Africa (HPCSA) and the South African Dental Association (SADA) Mediator describe situations where the patient expected so much more than they actually experienced.

Anecdotal evidence from conversations with colleagues suggests that we may be inadvertently increasing the expectation gap, which in turn can cause an increase in complaints. To give an example of this, I was approached by a Dental Protection member at the recent SADA Congress who described a complaint he had recently received and wanted reassurance that he had done the right thing.

The complaint involved a 16 year old teenager who needed four permanent teeth extracted for orthodontic reasons. The extractions had been arranged without a referral letter from the orthodontist and without a pre-extraction consultation to save fees. The father had dropped the teenager off, gone away, and when he returned was unhappy that none of the planned extractions had been completed and three fillings were undertaken instead. The member was not overly worried about the complaint but concerned that the consent given by the teenager would be invalid.

In not insisting that an examination and discussion should take place before any treatment was provided, the dentist created an expectation that this part of the care of the patient was not that important.

Whilst he did explain why the treatment of caries and the re-evaluation of the treatment plan was needed he had already lost the opportunity to manage the father’s expectations.

Consent

Discussing a recognised complication after the event can often be an uncomfortable conversation, as the patient may say if it is a recognised complication then they should have been warned from the outset in the consent process. Whilst it may not have influenced the decision to press ahead with the treatment, discussions around risk during the consent process makes it much easier to manage the potential fall-out when a risk does materialise.

A common source of disappointment and tension can arise during an endodontic procedure where a fractured endodontic instrument complicates an otherwise straightforward treatment. Not infrequently, the services of an endodontist is required to recover the file and this comes at a cost to either the practitioner or the clinician, depending upon what was discussed in the consent process. Even when used correctly, files will still occasionally break in service.

This would be considered a non-negligent complication if the correct protocols were followed. However, the discussion with the patient becomes more problematic when you try and explain this to them after the event, and mention the additional costs the patient will need to meet.

Breakages are generally associated with carelessness by patients. When they are unaware of the risk and financial consequences, they may blame the clinician and won’t expect to be paying for the remedial treatment themselves.

Fees

Disputes about the predicted cost of treatment, and an unwillingness to acknowledge that a misunderstanding can exist when an unexpected co-payment is outstanding is often reported in complaints to the HPCSA.

This reflects our survey findings where more than 70% of dentists think that patients are more likely to complain. In many cases the unexpected co-payment arises from an underpayment made by the medical scheme. In nearly every case it was the patients’ expectation that the benefits covered all dental fees and the dentist’s expectation that they would be paid properly for the treatment rendered.
Expectation created around the obligations of third party funders, if not properly managed, can undermine the consent procedure.

Underpayment by medical schemes on behalf of their member should never happen when authorisation has been obtained in an ideal world. However they do occur and often lead to a difficult and unexpected conversation with a patient about an account they were not expecting.

In his presentation to the recent SADA Congress, the Dental Mediator, Dr Kobus Barnard, gave an overview of the cases he mediates. Just under half of the complaints he deals with relate to disputes around fees and billing. The solution to this problem generally always comes before the difficult conversation ever becomes necessary and involves informed financial consent.

A legal and ethical obligation places a duty on each practitioner to provide information about treatment costs. If the treatment includes laboratory items or the services of an anaesthetist then these costs need to be discussed as they have an influence on treatment choices. It would also be advisable to discuss with the patient whether there is any co-payment and the approach the practice will take in the event that there is a shortfall payment by the medical scheme.

Communication

In nearly all of the cases discussed, the difficult interaction could have been avoided through better communication in the consent process. On the basis that patients continue to measure a clinician’s technical ability more by their communication skills than the emergence profile and marginal fit of their restorations, we will all still have days and moments where we have to manage disappointment and unmet expectations in a difficult interaction.

When you find yourself at the start of, or in the middle of, a difficult interaction there are four recognised steps you can take towards achieving a satisfactory solution:

1. Acknowledge there is a problem and try to summarise and gain agreement as to what are the key issues
2. Maintain firm boundaries and avoid being drawn into point-winning arguments that are not focussed on solutions. Often it is the failure to discuss the elephant in the room and to find some common ground that leads to a further deterioration in the interaction
3. Show compassion and demonstrate you understand why the patient is unhappy
4. Keep your focus on the best outcome for both parties rather than winning the argument.

There are occasions when managing patient expectations becomes a far bigger challenge than the actual treatment itself. If you choose to ignore an unhappy patient you may lose control of the situation, and our experiences show that the patient will select the path of least resistance, often to the HPCSA or beyond to their own attorney. You can always look to Dental Protection for support and advice on how to approach a challenging situation.

In June 2018 DPS surveyed 173 dentists in South Africa
“Nebulous” is the adjective sometimes conjured when the concepts of Ethics are debated... and in recognising that insubstantiality, philosophers, teachers and practitioners have through the ages endeavoured, paradoxically, to define solidly concrete principles in ethical practice. It is as though we all know what is meant... but need the reassurance of having guidelines spelt out.

Carl Sandberg made these somewhat sardonic comments: “I have taken a course in Ethics. I read a thick textbook, heard the class discussions and came out of it saying I had not learnt a thing I did not know before about morals and what is right or wrong in human conduct.”

Be that as it may, the experience of the profession over these many years is conclusive... reinforcement of the principles of Ethics is essential to good practice. To that end the HPCSA has devoted much time and effort to condense otherwise rather tenuous beliefs into those desired guidelines. It is warranted to repeat the Core Ethical Values published by the HPCSA.

These represent all required of a professional to maintain good ethical practice. Juxtaposed below next to each Value is a quotation designed to stimulate pensive meditation on the concept. It may even be that as a result the practice of Good Ethics becomes routine!

1. **Respect for persons:** Healthcare practitioners should respect patients as persons, and acknowledge their intrinsic worth, dignity, and sense of value.
   
   *Ethics is knowing the difference between what you have a right to do and what is right to do.* - Potter Stewart

2. **Best interests or well-being:** Healthcare practitioners should not harm or act against the best interests of patients, even when the interests of the latter conflict with their own self-interest.

   *In law, a man is guilty when he violates the rights of others - in Ethics he is guilty if he only thinks of doing so.* - Immanuel Kant

3. **Best interest or well-being:** Beneficence: Healthcare practitioners should act in the best interests of patients even when the interests of the latter conflict with their own personal self-interest.

   *The first step in the evolution of Ethics is a sense of solidarity with other human beings.* - Albert Schweitzer

4. **Human rights:** Healthcare practitioners should recognise the human rights of all individuals.

   *Ethics is in origin the art of recommending to others the sacrifices required for cooperation with oneself.* - Bertrand Russell

5. **Autonomy:** Healthcare practitioners should honour the right of patients to self-determination or to make their own informed choices, and to live their lives by their own beliefs, values and preferences.

   *Ethics are more important than Law.* - Wynton Marsalis

6. **Integrity:** Healthcare practitioners should incorporate these core ethical values and standards as the foundation for their character and practice as responsible healthcare professionals.

   *While academic abilities remain integral it is work Ethics that form the soul of the business (or practice).* - Jamshyd Godret

7. **Truthfulness:** Healthcare practitioners should regard the truth and truthfulness as the basis of trust in their professional relationships with patients.

   *You do not teach morals and Ethics and empathy and kindness in the schools .. you teach them at home and children learn by example.* - Judy Sheindlin

8. **Confidentiality:** Healthcare practitioners should treat personal or private information as confidential in professional relationships with patients unless overriding reasons confer a moral or legal right to disclosure.

   *In civilised life, Law is on a sea of Ethics.* - Earl Warren

9. **Compassion:** Healthcare practitioners should be sensitive to, and empathise with the individual and social needs of their patients and seek to create mechanisms for providing comfort and support where appropriate and possible.

   *Non violence leads to the highest Ethics, which is the goal of all evolution. Until we stop harming all other living beings we are still savages.* - Thomas Edison

10. **Tolerance:** Healthcare practitioners should respect the rights of people to have different ethical beliefs as these may arise from deeply held personal, religious or cultural convictions.

    *It may not have the virtuous ring of the golden rule but the maxim “Never say Never” is one of the most important in Ethics.* - Julian Baggini
11. **Justice:** Healthcare practitioners should treat all individuals and groups in an impartial, fair and just manner.

*In just about every area of Society, there is nothing more important than Ethics.* - Henry Paulson

12. **Professional competence and self-improvement:** Healthcare practitioners should continually endeavour to attain the highest level of knowledge and skills required within their area of practice.

*Medicine rests on four pillars – philosophy, astronomy, alchemy… and Ethics.* - Paracelsus

13. **Community:** Healthcare practitioners should strive to contribute to the betterment of society in accordance with their professional abilities and standing in the community.

*There is no question that as science, knowledge and technology advance, we will attempt to make more significant things… and there is no question that we will always have to temper these things with Ethics.* - Benjamin Carson

After that pensive meditation, “Nebulous “ may not yet have retreated into thin air… but at least there may be a greater confidence in the guidelines for enhanced Ethical awareness.

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**Do the CPD questionnaire on page 588**

The Continuous Professional Development (CPD) section provides for twenty general questions and five ethics questions. The section provides members with a valuable source of CPD points whilst also achieving the objective of CPD, to assure continuing education. The importance of continuing professional development should not be underestimated, it is a career-long obligation for practicing professionals.

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**Online CPD in 6 Easy Steps**

1. Go to the SADA website www.sada.co.za.
2. Log into the ‘member only’ section with your unique SADA username and password.
3. Select the CPD navigation tab.
4. Select the questionnaire that you wish to complete.
5. Enter your multiple choice answers. Please note that you have two attempts to obtain at least 70%.
6. View and print your CPD certificate.
Continuous Professional Development questionnaire

This edition is accredited for a total of 3 CEUs: 1 ethical plus 2 general CEUs

GENERAL

Dental fraud in South Africa 2007 – 2015

1. According to current legislation every case of dental fraud in South Africa should be reported to the SAPS.
   a. True
   b. False

2. Fraudulent behaviour is an intentional action.
   a. True
   b. False

Identify the incorrect statement.

3. The following are examples of fraud committed in Dentistry:
   a. Claiming for excessive or additional material not used during treatment
   b. Inadvertently identifying an incorrect tooth as having a restoration placed
   c. Dentists claiming for additional fillings or extractions that were not performed
   d. Dentists providing cosmetic gold inlays but charging for normal crowns

4. HPCSA data between 2007 and 2015 shows a gradual decrease in dental fraud for all dental professionals in South Africa.
   a. True
   b. False

Enamel demineralisation as an iatrogenic effect of Orthodontic treatment

5. White spot lesions have been reported to occur in nearly half of all orthodontically-treated patients.
   a. True
   b. False

Identify the incorrect statement.

6. Resin sealants applied to the surface of teeth offer the following advantages in preventing white spot lesions:
   a. greater wear resistance as they are highly filled
   b. may incorporate fluoride which is released, enhancing protection
   c. effective sealing abilities
   d. fluoride content remains at effective levels

7. In severe cases beyond the possibility of remineralisation, resin infiltration may improve the appearance of demineralised white spot lesions as a result of the similar refractive index of infiltrated and sound adjacent enamel.
   a. True
   b. False

Testing Gustafson's dental age estimation method on a sample of Western Cape adults

8. Gustafson used four age related changes in his method of age estimation.
   a. True
   b. False

Identify the correct answer.

9. When the estimated ages as calculated by Examiner 1 (using Gustafson's method of age estimation) were compared with the chronological (real) ages of the donors, what percentage proved to be accurate?
   a. 0%
   b. 16.4%
   c. 23.6%
   d. 38.2%

Periodontal disease - risk factors and treatment options

Identify the correct answer.

10. Bacteria which cause periodontal disease have the ability to colonise because:
    a. they can remain free with no attachment to periodontal tissues
    b. they can multiply
    c. they can compete with other microbes
    d. they can resist the host defense mechanisms

11. Periodontal disease is considered the sixth most common complication of diabetes.
    a. True
    b. False
Isolated palatal injury due to a bicycle accident

Identify the correct answer.

12. Maxillo-facial trauma due to bicycle accidents may involve fractures of the following bones:
   a. orbital
   b. zygomatic
   c. nasal
   d. mandible
   e. all of the above

13. Prevention of bicycle accidents should focus on:
   a. education
   b. supervision
   c. improvement of infrastructure
   d. all of the above

Hamartomas in the opercula of four unerupted primary molars: a case report

14. The hamartomatous lesion identified as Odontogenic Giant Cell Fibromatosis (OGCF) seems to have a strong association with mandibular impacted molars suggesting it may be biologically different from “classic” odontogenic tumours.
   a. True
   b. False

15. In the management of hamartomas, surgical intervention may be indicated if there is pain or discomfort, infection or interference with normal function (i.e. breastfeeding/mastication).
   a. True
   b. False

Clinical Window

16. The results of the Cademartori et al review provide evidence that there was a significant association found between periodontal disease and depression.
   a. True
   b. False

17. The Cademartori et al study showed a positive association between periodontal disease and depression.
   a. True
   b. False

18. In the Steinmass et al study, the AvaDent Digital Dentures showed a significantly better denture base congruence with the soft tissues than did the conventional dentures.
   a. True
   b. False

Maxillofacial and oral radiology case report

19. The first report of an odonto-amelolastoma was made by Mervyn Shear.
   a. True
   b. False

20. The odonto-ameloblastoma is a very common tumour.
   a. True
   b. False

ETHICS

21. These values designated by the HPCSA represent all that is required of a professional to maintain good ethical practice.
   a. True
   b. False

22. Healthcare workers should respect their patients but should not allow the patient to take decisions which are potentially harmful to them.
   a. True
   b. False

23. Healthcare workers have no responsibility regarding the needs of the community in which they practice.
   a. True
   b. False

Identify the correct statement.

24. Practitioners should manage the personal and private details of their patients by:
   a. allowing free electronic access to facilitate research
   b. accepting that for legal and moral reasons, disclosure may be justified
   c. deleting the data after every appointment
   d. recording only the banking details

25. It is expected that all patients from all backgrounds should adhere to the ethical principles of the attending healthcare practitioner.
   a. True
   b. False
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