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The Moray Eel is part of the family Muraenidae, renowned for their rather vicious bite. They possess pharyngeal teeth, virtually a second set. Unlike other fish having such additional dentition, the Moray can move the pharyngeal set and indeed does so during swallowing, accomplished in the eel by bringing the set forward to grip the prey and then retracting to progressively shift the prey down the gullet. With that dental armamentarium it is no surprise that the Moray bites and never lets go!
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Imbued with the Olympic Spirit

The television screen is set to hold irresistible attraction during this Olympic time. In a vicarious feast of sport we shall live with the triumphs and the disappointments, the victors and the vanquished, the medallists and the also rans. What a spectacle is the Olympics, and what exertions have been required by all.. to be prepared to compete, to have the stadia ready, to ensure the stopwatches are accurate! It will be a close race but be sure Brazil will do this in style!

Brazil has high expectations.. not least in the field of Dentistry. Already regarded as having one of the highest standards of the profession, the country is well endowed with Dental Schools and with dental acumen. A recent count indicated a total of 232 courses in Dentistry, with some 10,000 graduates each year. The national ratio of dentist to population was given in 2011 as 1:745 although some Brazilian states recorded poorer stats, 1: 2050 and 1:1763. There are some 240,000 dentists in Brazil, according to Euromonitor International. The profession supports seven Dental Journals, which in 2012 published 675 scientific articles. Indeed there is an expectation that very soon, Brazil will overtake the United States as the most productive nation in terms of overall world scientific publications.

Perhaps the enthusiasm is in part related to the Spirit of the Olympics. In Ancient Greece the Games were charged with the Concept of Olympic Spirit which included:

Areti: Virtue • Amilia: Noble Competition • Timi: Honour • Elefteria: Freedom • Irini: Peace

That is a pretty unbeatable combination. Athletes were, and still are, expected to win with honour and to suffer defeat in peace. A graceful acceptance in both circumstances.

The writing and publication of a scientific journal article carries some of the same considerations. In the first place the researcher has the freedom to explore and experiment, knowing that he/she has entered a noble competition where there may well be honour, provided there has been virtue in the research. Secondly, and finally, the author should have peace whether the paper is accepted or not, knowing that the paper has been dealt with honourably.

The Journal this month publishes papers of diverse emphases, but with a focus on Oral Medicine and Restorative materials. All our authors have successfully survived the stringent referee processing and it is with honour that we include their papers. Last year we published just over sixty scientific papers and perhaps that is by comparison a reasonable record from our dental population of some 4000 dental practitioners. That would not have been possible were it not for the referees who so consistently and willingly devoted time and energy to their tasks. They always had the freedom to decline a request but took on the responsibilities with an empathy for the aspirant authors who had entered the noble competition, publish or perish!!

To continue the Olympic analogy, papers receive their starting orders on arrival at Head Office and enter the first heat with their submission to referees. Some will trip at this stage and be obliged to withdraw, others may face a restart for despite all the training and preparation, there are yet flaws in the technique. These will have a further opportunity to race with a revision of the paper. Those who perform well are eligible for the final, and are published! Medals are possible! Now comes a dilemma, for there are times when the final is delayed, and authors have to wait until their own final is announced. To those who have had wait, the sincere appreciation of the Journal.

Publishing is not easy and the Journal could not survive without those who enter the race. The Olympic Spirit is alive and well in all those who support the Journal, a winner’s crown to you all.

References
The South African Dental Association has been part of the World Federation for many years. It is customary that we have a representative attend the World FDI Conference and our Association has maintained close ties with the parent body. At this time of the year when South Africa honours Women, recalling the historic 1956 march to the Union Buildings, it is apposite to find that the FDI Conference is to have a special section of the 2016 programme dedicated to “Women In Dentistry”. The Welcome letter by the President and the programme outline are reprinted below, with acknowledgements to the FDI, and the Association is happy to include these announcements in this month’s Communique.

The FDI Annual World Dental Congress is unique in the opportunities it provides for learning and debate with dental colleagues from around the world.

The FDI Annual World Dental Congress is, in fact, two interconnected but distinct events: on the one hand, there is an international congress with a first-rate scientific programme delivered by top international, regional and national speakers. FDI is proud to bring together in one place some of the recognized ‘heavyweights’ of the international dental community, all of them specialists in key areas of modern dentistry. We are also very proud of some of the new sessions, first introduced in 2012. They include ‘meet the expert’ sessions, a true innovation, where participants have the opportunity of interacting with major figures in international dentistry. A recent innovation has been the World Oral Health Forum, usually in two separate sessions, which focuses on areas on public policy and oral health. The aim is to widen the debate, with speakers from other health or policy sectors.

On the other hand, the FDI congress is the once-yearly get-together of the World Dental Parliament, a decades-long part of FDI. It is a mixture of formal and informal sessions: central is the General Assembly, our federation’s supreme legislative and governing body, where the project leaders from among FDI membership report on progress over the past twelve months in key areas where FDI and its partners are incrementally moving towards FDI’s ultimate goal of ‘Leading the World to Optimal Oral Health’. This is supplemented by the three Open Forums, where policy debate takes place in as less formal atmosphere.

Poland is an excellent choice because, with a resilient economy, demand for dental services is growing rapidly, with growth in the private dental sector estimated between 20 and 30% per year. So it makes sense to hold an international event there at this time.

Geographically, Poland is at a crossroads and we believe ideally placed to attract participants from throughout Europe. We also believe that it has the potential to attract participants from around the world. In addition, we know that we have a top quality local team so we really expect a large turn-out for the FDI Annual World Dental Congress in Poznan and count on it being the landmark international event in dentistry in 2016.

**WOMEN’S LEADERSHIP IN GLOBAL HEALTH**

1. **Global Health Through Oral Health: Issues & Challenges** *(Dr Lois Cohen)*

   - To understand global health problems as these intersect with oral health/diseases
   - To focus on roles for the oral health workforce in the context of these problems.
   - To consider gender concerns in relationship to the global health issues and potential solutions.

   Differential definitions of global health will be presented with their similarities and differences regarding target populations, the services needed, underlying assumptions regarding social equity, scope of skills needed to address the extent problems, mix of interventions and health care delivery models. Contextual settings in which infectious diseases and the non-communicable diseases interact and the infrastructure that varies across the globe present unique opportunities for new workforce strategies to deal with prevention, diagnosis and care. How gender plays a role in health and disease in many cultural settings is also becoming important for development assistance programs as they continue to grasp how people live, work and relate to each other at all levels. Delivering services, hoping to help, in multidisciplinary collaborative teams presents a challenge for oral health professionals but one in which women’s leadership is uniquely suited.

2. **Women Leading Change** *(Dr Jeanne Craig Sinkford)*

   - To learn global strategies from four International Women’s Leadership Conferences: France, Sweden, Canada, and Brazil.
   - To identify critical factors in leadership training for the advancement of women.
   - To understand an emerging paradigm for women’s health across a lifespan that includes interprofessional education and team-based care.
Gender in the leadership pipeline is an imperative for global health. The value of gender focused leadership training will be presented using a diagnostic prescriptive model for mid-level career development. Mentoring, career laddering, and interventions will be discussed. The concept of a new paradigm for women’s health will be presented for its potential use in curriculum changes and interprofessional education.

**ANESTHESIA AND SEDATION IN THE DENTAL OFFICE**

1. **What if something goes wrong?**
   *(Dr. Zeljka Martinovic)*
   - Fobic patients - Quality of life
   - The frequency of the emergencies in dental office
   - Patient selection - how to identify risk patient
   - Protocols and procedures in specific situations

   Sedation can be method of choice for the treatment of phobic patients and for the long lasting dental procedures. Good selection of patient is very important as well as good knowledge of the procedures and pharmacology of sedative agents. Emergencies in dental offices are more often than expected. Dental office team must be prepared to recognize and trained to deal with this. The ideal sedative agent would be the one with rapid onset, easy titration, high clearance and good safety profile. The development of new modes of administration would improve the quality of sedation.

2. **Sedation in dental office: pro et contra**
   *(Dr. Daniela Bandić Pavlović)*
   - Different types of sedation
   - The right type of sedation for the right patient
   - Minimizing the exposure of the dental office team

   Different types of sedation enable dentist to resolve all fobic patients. Whether we use inhalation, intravenous or oral sedation depends on patients characteristics (comorbidities) and preference of the anesthesiologist. Comparison of different technique, pro and contra effects explains how to choose the right type of sedation for the right patient. Last but not least, we will discuss exposure of dental office team to inhalation sedative (nitrous oxide).

3. **How to minimize anesthesia to the patients: mini- invasive surgery** *(Dr. Maria Gabriella Grusovin)*

   To learn the up-to-date mini invasive surgical approach in periodontology and implantology. Nowadays many alternatives for surgery are proposed and both patients and clinicians are looking for the most predictable, simplest and less painful treatment option, which could lessen the use of anesthesia. The presentation will focus on the different aspects of surgical clinical choices such as flapless surgery as opposed to flap elevation, short implants as an alternative to bone augmentation, post extractive implants versus delayed one, describing advantages and complications both from a clinical and a scientific point of view. Clinical use of anaesthesia technique will be described.

The Congress Organisers.

A comprehensive and intriguing programme.

To all our women members, the very best wishes of the Association, and sincere appreciation for all your contributions and commitments.

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**OBITUARY**

Professor Arthur Lewin

6 February 1926 - 28 July 2016

Arthur Lewin passed away in July. He had reached the venerable age of 90 at the beginning of the year. He was recognized internationally for his important contributions to Dentistry.

Professor Bertus van Rensburg worked closely with Arthur for some 46 years and wrote this tribute to an icon.

**Tribute**

Prof. Arthur Lewin can be regarded as the doyen of fixed prosthodontics in South Africa. He had an extremely perceptive mind together with the ability to summarize any situation rapidly and could apply whatever he had learned.

He was born in Oudtshoorn. His father was educated in Germany as an industrial chemist, whilst his mother came from Swellendam. Arthur the youngest child in the family was full of life and sports and often in trouble through his pranks. He was eventually sent to a Marist Brother’s in Uitenhage so that the Brothers could teach him how to behave and to learn all about discipline.

Arthur enrolled in the Faculty of Dentistry of the University of the Witwatersrand after he matriculated. His peers do not remember him for his academic prowess but rather as the cheerleader of the Wits students at inter-varsity meetings against Tukkies for two different years. Prof. Phillip Tobias told him when he joined the Senate of the University of the Witwatersrand, Johannesburg, that he would never have thought a cheerleader would ever have a seat in the Senate!
Arthur met a lovely, highly intelligent young lady from Klerksdorp in his final year of study. She was Joy Owsianic. They were married in December 1949. This marriage lasted for more than 60 years, ended only when she passed away after a long illness.

Arthur joined a dental practice in Klerksdorp after he qualified. This gave him the chance to become a real “wet-fingered” dentist. The practice had branches throughout the Northwest province and Arthur used to visit the outlying areas when there were cattle auctions in the towns. Treatment provided at these branch practices consisted mainly of extractions and dentures. Impressions and bites were taken the same day. The dentures were completed in Klerksdorp and sent off by post. A Joseph Rodger’s penknife was placed in the box with the dentures. The idea of the penknife was to provide the proud owner of the new dentures with a tool to ease the denture where it was hurting!

Dr Lewin became an avid golfer. This hobby was curtailed because of a knee operation and was replaced by his becoming a radio-ham. He established “comms” with many people but one special contact was a famous dentist named Stan Vogel in Los Angeles. When Stan attended courses over the weekends, he used to record the lectures and called Arthur on the following Monday evening, SA time. He told Arthur where he was and transmitted the lecture over the airwaves to Klerksdorp. The mutual interests led to Stan urging him to come to Los Angeles to meet Peter K. Thomas and others so that he could improve his knowledge of occlusion and gnathology by attending the courses on offer. Arthur did meet with Peter Thomas, Charles Stuart, Harvey Stallard and B B McCullum and became one of their followers. He came back full of knowledge and ideas ……plus a bag of articulators on his back, …..as Prof. Shepherd from Wits once said.

Dr. Lewin urged his colleagues to obtain comprehensive mouth records by using a face-bow, check bites, hinge-axis registrations and especially the pantograph. Arthur trained his technicians, notably Cliff Prew, in the skills of gnathology. He left Klerksdorp a few years after his return from Los Angeles and relocated to Johannesburg. The reason for this move was that many of his patients were from Johannesburg and they convinced him to move. He established a very successful practice in Lancet Hall and for many years worked closely with Cliff Prew. He and Cliff subsequently moved to the Rosebank Clinic … together with the patients.

Arthur became friendly with Prof. C J Dreyer who was Dean of the Faculty at Wits. Jan Dreyer introduced him to dental research, which he found he enjoyed. There was no Head of the Department of Restorative Dentistry at that time and Prof. Dreyer invited him to assume that position. He accepted the offer during 1969 but retained the right to limited private practice. He tutored the first graduate student in 1969 and started with the next group in 1970. He stressed the importance of gnathology and insisted that the graduate students themselves had to plan, design and do the wax-ups of the cases they were treating.

In his continued interest as a radio-ham he made contact again with a hobbyist called Jasper, with whom he had spoken in the past. Jasper told him he was doing research at the then “Jan Smuts” airport to determine how the earth and tarmac deform when a large plane lands. These data helped the engineers to design and calculate the strength required for the tarmac. Arthur asked him how it had been done. The answer was by means of strain gauges. Arthur’s reply was if you can do that, then I can use the same type of instruments but just much smaller to calculate how a tooth deforms during mastication. Many hours went into this research. Tests were also done when a tooth was dehydrated and when water was added to it. The idea was to compare the distortion of a neurologically vital tooth to that of a non-vital one.

His next project led to the invention of the Electro-gnathograph. This instrument uses Hall-effect transducers to sense movements of the jaw, recorded as alterations in the strength of the magnetic field of a small magnet attached to the mid-incisor point of the mandible. Arthur built the prototype of this device himself, followed by another design with the help of Engineer Nicol from Siemens.

This instrument was eventually produced and distributed by Siemens AG and more recently by Bio Research in Milwaukee. All graduate students under Professor Lewin used this device for the recording of unconstrained jaw movements (or jaw movements with as little constraint as possible), on the patients they were treating.

Professor Lewin retired, as Head of the Department of Restorative Dentistry when he turned 65, in accord with the rules of the University but he remained involved in an advisory capacity with the recording of jaw movements. His friend, Jim Booth from Montana, has carried on clinical research work, which has also now been extended to include Electro-myography.

Arthur had a further 15 years as a Professorial Research Fellowship at the Dental School. The students, especially the post-graduates, at Wits unfortunately lost out on his expertise and the zest for research and the guidance that he offered.

Prof. Lewin published numerous papers and lectured throughout the world. He was recognized as a pioneer contributor to the analysis of jaw movement and masticatory physiology and received honours from many institutions.

I hope that I was able to demonstrate in this short tribute that Professor Arthur Lewin can be regarded as the doyen or a great explorer in the field of Prosthodontics in South Africa. I salute Arthur Lewin for his achievements. I regard myself extremely fortunate to have been closely associated with him for more than 46 years.

Bertus van Rensburg
Cancrum Oris (noma) in an HIV-positive adult: a case report and literature review

**ABSTRACT**

**Background:** Noma refers to an overwhelming invasion of micro-organisms from the oral cavity into the face leading to gangrene, sepsis and, possibly, death. Figures available for 2006 indicate an estimated incidence of 100 000 to 140 000 new cases each year in sub-Saharan Africa with a mortality rate of 70% to 90%.

**Introduction:** The classic clinical picture of a noma patient is severe facial tissue destruction associated with oral ulcerations and, in some cases, acute necrotising gingivitis. The following case report concerns a 35-year-old female patient who was treated at the Department of Maxillo-Facial and Oral Surgery at the Tygerberg Oral Health Centre.

**Case report:** A 35-year old female was referred to the department with a gaping defect in her right cheek accompanied by necrosis of her mandible. Her medical history indicated that she was HIV+ and had previously been diagnosed with Multi-Drug Resistant TB (MDR). She had defaulted on treatment for both these diseases. Extra-oral examination revealed a gaping defect as a result of the loss of soft tissue of the right cheek. The patient was admitted so that her medical treatment regime could be optimized under supervision. A biopsy of the exposed mandibular bone and soft tissue was done under local anaesthetic as the patient was considered an anaesthetic risk. The results indicated acanthosis with diffuse epidermal hyperplasia.

**Discussion:** Globally it appears that HIV infection is not a strong risk factor for noma. In South Africa, HIV infection may play a substantial role in the pathogenesis of noma. In this particular case there was severe facial tissue destruction associated with oral ulceration and acute necrotising gingivitis. An increase in the incidence of noma shows that it cannot be dismissed as a scourge of previous centuries, but remains a public health issue in the poorest communities of the world, still claiming thousands of victims annually. Hence the attempt to reduce the incidence of noma has become a priority for the World Health Organization’s (WHO) five-point strategy.

The goal of treatment during the acute stage aims to keep the patient alive by administering antibiotics and specific treatment for co-existing diseases. Once the initial stages have been overcome and a good nutritional status has been achieved, patients can be assessed for reconstructive surgical treatment.

**Conclusion:** The clinical features of this case were consistent with classical features reported in the literature. It emphasizes how this condition contributes towards serious facial destruction and debilitation. This case also highlights a potential association between noma and HIV/AIDS.

**BACKGROUND**

The word noma is an ancient Greek word that means ‘meadow’, ‘grazing’, and, in a metaphorical sense, ‘a quickly spreading sore’. In medicine, noma refers to an overwhelming invasion of micro-organisms from the oral cavity into the face leading to gangrene, sepsis and in many cases, death.

Classical medical authors referred to the condition as Galenus and Celsus. In 1595, Carolus Battus, who settled in the Netherlands, wrote a chapter on ‘watercancker’ in his Surgeons’ Handbook. In 1680 Cornelius van de Voorde introduced the term ‘noma’ for oro-facial gangrene in children. He was convinced that this condition was not due to a cancerous growth but rather to infection.

The end of the eighteenth century marks the first successful attempt to reconstruct faces ravaged by noma, while by the mid-nineteenth century medical literature included accounts of extensive facial reconstruction in patients who had survived the disease.
Noma occurs predominantly among deprived African children living in an impoverished state with low hygiene and inadequate sanitation services. In 1998, the WHO estimated the annual global incidence of noma at 140,000 and the associated mortality at 70 to 80 percent among patients not treated immediately after diagnosis. A more recent report estimates the annual incidence at 25,000 in developing countries bordering the Sahara.6 Figures available for 2006 indicate an estimated incidence of 100,000 to 140,000 new cases each year in sub-Saharan Africa with a mortality rate of 70 to 90%, i.e. an estimated 75,000–100,000 deaths. These figures may be grossly underestimated since less than 15% of acute cases receive medical care.6

Other reasons for uncertain epidemiological data for noma include systematic errors in longitudinal studies, misreporting of cases due to the high mortality rate as well as the stigmatization associated with the disease causing affected children to be shunned by the community and sent to remote locations for medical treatment.5–7

The following are considered host risk factors (etiological factors) associated with noma:

- Malnutrition
- Viral theory (e.g. measles)
- Bacterial theory (e.g. malaria)
- Debilitating diseases (e.g. HIV-AIDS; Tuberculosis (TB); Pneumonia)

**Malnutrition**

Oral mucosa and the gingiva are characterised by high tissue turnover and can be severely affected by food deficiency, leading to an increased permeability of the tissue membranes. This can facilitate and increase the entry of oral pathogens due to protein and vitamin deficiencies. In addition, the inflammatory response further weakens the tissue membranes. Periodontal tissues and the oral flora are also directly affected by local and systemic consequences of malnutrition. Raised levels of anaerobic flora, gram-negative rods and spirochaetes are present in malnourished children.5,8

Inappropriate weaning practices of various ethnic groups may lead to malnutrition. In some cases breastfeeding is stopped abruptly with no gradual transition to a solid diet. This sudden change in nutrition may lead to deficient levels of vitamins, immunoglobulins, essential aminoacids and minerals. It is at this age that children usually develop kwashiorkor and may also suffer from malaria, measles, other childhood-related diseases and acute noma.5

**Viral theory**

The viral theory of the aetiology of noma suggests that infection with the herpes virus could lower local immunity, therefore facilitating the development of pathogenic bacterial flora. This hypothesis was originally concerned with the aetiology of acute necrotizing ulcerative gingivitis (ANUG) but has later been extended to include noma.5 The global increase in ANUG associated with HIV/AIDS3 can also contribute to the increased prevalence of noma. However, the percentage of ANUG or other oral ulcers that transform into noma, is generally very low.3

Tempest believed that measles was an important precursor to noma.9 Measles may lead to lower energy levels and immobilisation of hepatic Vitamin A. If the condition transforms to kwashiorkor or marasmus, it can be fatal. Children also often present with oral ulceration after measles, referred to as noma-like post-measles ulcerations. In addition, tissues impaired by vitamin deficiency are susceptible to the occurrence of noma.10

**Micro-organism theory**

In less developed countries, the contribution of malaria to noma has been debated. Eckstein postulated that malaria may lead to a decrease in immunity and consequently to noma.21 A malodour arising from the necrosis occurring in the lesions of noma is found in affected patients and has been associated with bacterial infection as an aetiological factor. After overcoming challenging obstacles complicating the investigation of various phases of infection, improved histological techniques have allowed scientists to examine and identify the micro-organisms in the lesions. The main objective of the microbiological analysis is to compare normal and diseased normal flora with a considered emphasis on technique sensitivity.3 *Prevotella melaninogenica, Corynebacterium pyogenes, Fusobacterium nucleatum, Bacteroides fragilis, Bacillus cereus, Prevotella intermedia* and *Fusobacterium necrophorum* have been identified as attendant to noma.

**Debilitating diseases**

Any debilitating disorder may facilitate the progression of buccal lesions towards noma. Other infectious diseases that have been considered as predisposing factors are chickenpox, smallpox, typhus, typhoid fever, diphtheria, visceral leishmaniasis (kala-azar), pneumonia, TB and, more recently, AIDS.3

The following are social/environmental aetiological factors:

- Poverty
- Poor oral hygiene
- Delay in seeking medical treatment
- Limited access to or inadequate health care facilities

Poverty is an important risk factor, especially in Africa where a lack of resources may lead to poor sanitation, inferior oral and general hygiene and chronic malnutrition. There is also a higher probability of people contracting noma in areas where infective agents are prevalent and communicable diseases are common.3,8,12

In fact, the incidence of noma corresponds to the worldwide geographical distribution of poverty in less developed countries.3 In those countries and definitely in many regions in Africa, the majority of the population do not own a toothbrush. For instance, only a fifth of the children in Nigeria use a toothbrush. Poor oral health can lead to the development of ANUG and research in Nigeria suggested that ANUG can be associated with noma as well as with various other infections.3

**INTRODUCTION**

The classic clinical picture of a noma patient is severe facial tissue destruction associated with oral ulcerations and, in some cases, acute necrotising gingivitis.3,10,13

Given the low incidence of patients presenting in the initial phase, the clinical features of the onset of noma are not as clear. Some of the initial symptoms are fever, malaise,
cervical lymphadenopathy, gingival bleeding, oral mucosal lesions, facial oedema and severe halitosis.\textsuperscript{14}

When the oral mucosa and/or overlying skin ulcerate, the destruction can lead to the exposure of underlying bone. It has been suggested that ANUG is a precursor to noma and, hence, the presence of ANUG should raise concern.\textsuperscript{15} The initial phase of the gingivitis commences at the tips of the interdental papillae and marginal gingivae. Blood flow to the infected tissues is inadequate, eventually resulting in an ischaemic area and localised necrosis.\textsuperscript{16,17} When the necrosis progresses beyond the mucogingival junction, it affects the alveolar, labial, palatal, buccal and lingual mucosa. The full thickness of the buccal or labial muscle layer is now involved and at this point the condition is known as necrotizing stomatitis.\textsuperscript{18} Further progression will lead to perforation of the facial tissues and skin. In many cases this may eventuate in a matter of days. Generally, the external tissue loss is not closely commensurate with the more extensive intraoral destruction.\textsuperscript{5,16,17} During the separation of the soft tissue slough, sequestration of the exposed teeth and bone occurs spontaneously. In severe cases, larger tissue destruction occurs with the nose, upper lip, infraorbital margin and premaxilla also being affected.\textsuperscript{5}

Possible complications are displacement of teeth associated with tissue destruction and joint displacement, extreme facial deformities, intense scarring, trismus, nasal regurgitation, fusion between mandible and maxilla and defective speech. Noma survivors have great difficulty coping with the disfigurement and functional impairment.\textsuperscript{3,19}

As the disease progresses, its systemic effect becomes debilitating. Patients are usually febrile, dehydrated and experience pain.\textsuperscript{2} Clinical symptoms such as tachycardia and tachypnoea and recurrent diarrhoea may be experienced. It is also common to find a low haemoglobin and white cell count and hypo-albuminaemia. Parasitic infections such as malaria and viruses can be evident.\textsuperscript{3} Orofacial lesions are characterised by dense scar tissue at the margins of the facial defect with extensive fibrosis.\textsuperscript{19} These usually occur unilaterally but may also present bilaterally.

The following case report concerns a patient who was treated at the Department of Maxillo-Facial and Oral Surgery at the Tygerberg Oral Health Centre.

\section*{CASE REPORT}

A 35-year-old female was referred to the Department with a gaping defect in her right cheek accompanied by necrosis of her mandible (Figures 1 and 2). Her medical history indicated that she was HIV+ and had previously been diagnosed with Multi-Drug Resistant TB (MDR). She had defaulted on treatment for both these diseases.

On general examination she appeared cachectic with signs of anaemia, jaundice and she had difficulty walking. Extra-oral examination revealed a gaping defect as a result of the loss of soft tissue of the right cheek, as shown in Figures 1 and 2. The margins of the defect were fibrotic and the exposed mandibular bone appeared necrotic. Intra-oral examination was limited by restriction of opening due to trismus caused by the chronic infection and subsequent fibrosis.

The orthopantomographic radiograph (OPG) (Figure 3) revealed the following:

- bone loss around the 18 and 17 teeth suggestive of periodontitis
- caries and periradicular radiolucencies in the 36 area suggestive of osteomyelitis
visible extraction sockets (Figure 3) of the 44, 45, 47, 48 teeth with radio-opacity of the bone suggestive of sclerosing osteitis.

The CT scan (Figures 4 and 5) confirmed the signs of osteomyelitis seen on OPG. Further, the scan showed a large defect (20mm wide) overlying the right maxilla (Figure 5) as well as in the lateral wall of the right maxillary sinus. The sinus was opacified by circumferential mucosal thickening. Inflammatory stranding was noted in the pre-maxilla fat space as well as within the masseteric space.

Special investigation confirmed the HIV+ status and revealed a low CD4 count (15cells/mm³). All liver function indicators (i.e. total bilirubin, conjugated bilirubin, aspartate aminotransferase (AST), alkaline phosphatase (ALP) and gamma glutamyl transpeptidase (GGT) were elevated which explained the clinical jaundice. The urea, creatinine and electrolyte test indicated low urea 1.2mmol/L and creatinine 18µmol/L levels which could be attributed to the malnutrition and liver dysfunction. The full blood count confirmed the clinical anaemia with haemoglobin of 6.8g/dL. The chest x-ray revealed mild signs of post-primary pulmonary TB. The abdominal ultra-sound revealed the following: bilateral hyperechoic kidneys; hyperechoic liver and moderate ascites.

The patient was admitted to optimize her medical treatment regime. She was started on empiric intra-venous amoxicillin and clavulanic acid 1.2g and metronidazole 500mg 8-hourly, commenced XDR TB treatment and received supplementary magnesium-sulphate and calcium.

A biopsy of the exposed mandibular bone and soft tissue was done under local anaesthetic as the patient was considered an anaesthetic risk. This tissue was sent for histology, gene-analysis and fungal culture. The histological results indicated acanthosis with diffuse epidermal hyperplasia. No TB was cultured.

Formal sequestrectomies and free vascularised tissue transfers were planned. Regrettably the patient passed away before treatment could be delivered.

DISCUSSION

The epidemiology of noma in the South African population is unknown, and the clinico-pathological features are poorly characterised. Globally it appears that HIV infection is not a strong risk factor for noma. However, in South Africa, HIV infection may play a substantial role in the pathogenesis of noma.\(^{18}\)

In this particular case there was severe facial tissue destruction associated with oral ulceration and acute necrotising gingivitis. This is consistent with the classical features of noma (cancrum oris) found in other reported cases.\(^{3,5}\)

WHO strategy against noma

An increase in the incidence of noma shows that it cannot be dismissed as a scourge of previous centuries, but remains a public health issue in the poorest communities of the world, still claiming thousands of victims annually. Hence the attempt to reduce the incidence has become a priority for the five-point strategy defined by World Health Organization.\(^{1,5}\)

1. Epidemiology and Surveillance

Most noma cases are reported in developing countries in Africa, Asia and South-America. It is estimated that there are about 100 000 new cases of noma yearly, with fatality rates of 80% in the absence of treatment. Due to the challenge of collecting reliable data, the WHO has developed tools for epidemiological studies on referred cases and national retrospective surveys of orofacial mutilations and noma. The WHO also recommends including noma in existing epidemiological surveillance systems.

2. Aetiological research

Research on the aetiology of noma was improved in the 1990’s (Figure 6). Poverty, inadequate sanitation, malnutrition, poor oral hygiene, lack of general hygiene and predisposing infectious disease such as HIV/AIDS, concomitant TB and measles are all considered aetiological factors.

3. Prevention

In its strategy to contain noma, the WHO has made prevention a high priority. Efforts include incorporating an awareness of noma and its symptoms into existing health education and promotional activities together with a call to urgent action to contain the disease. Furthermore, training of primary health care workers in a recognition of the condition to ensure early detection and timeous management could also play a pivotal role in curbing the devastation that noma wreaks in the life of a victim.

4. Primary health care

A leaflet describing the progress of noma from necrotizing gingivitis to the loss of tissue was published by the WHO in 1994. The WHO encourages integration of noma detection and management into existing health services, especially at a primary health care level, and recommends that all district personnel be trained in its recognition, management and referral.

5. Surgical rehabilitation

Survivors of noma who are left with deformities need plastic and reconstructive surgery. In 2001, the WHO recommended that the Noma Children’s Hospital in Nigeria should receive additional resources and designated the Regional Referral Centre for the management of noma cases and the training of personnel from other countries in the region.
TREATMENT OPTIONS AND TIMING OF RECONSTRUCTIVE SURGERY

Differentiation between the acute and late stages of noma is imperative in the determination of the specific treatment option. In the late stage the patient has survived gangrene, sloughing, sequestration, the formation of granulation tissue, wound contracture and re-epithelialization. The goal of treatment during the acute stage aims to keep the patient alive by administering antibiotics and specific treatment for co-existing diseases. Once the initial stages have been overcome and a good nutritional status has been achieved, patients can be assessed for reconstructive surgical treatment.

CONCLUSION

Cancrum oris remains rare in South Africa, but this case provides evidence that it is still present and that it continues to be a challenge to manage. The clinical features of this case were consistent with classical features reported in the literature. It emphasizes how this condition potentially leads to serious facial destruction and debilitation. This case also highlights a possible association between noma and HIV/AIDS.

Conflict of interest: None declared.

Ethical approval: Informed consent was obtained from the patient.

References

Oral diseases associated with human herpes viruses: aetiology, clinical features, diagnosis and management

ABSTRACT
Human herpesviruses (HHVs) are very prevalent DNA viruses that can cause a variety of orofacial diseases. Typically they are highly infectious, are contracted early in life, and following primary infection, usually persist in a latent form. Primary oral infections are often subclinical, but may be symptomatic as in the case of herpes simplex virus-induced primary herpetic gingivostomatitis. Reactivation of the latent forms may result in various conditions: herpes simplex virus (HSV) can cause recurrent herpetic orolabial lesions; varicella zoster virus (VZV) can cause herpes zoster; Epstein-Barr virus (EBV) can cause oral hairy leukoplaikia; and reactivation of HHV-8 can cause Kaposi sarcoma. In immunocompromised subjects, infections with human herpesviruses are more extensive and severe than in immunocompetent subjects. HSV and VZV infections are treated with nucleoside analogues aciclovir, valaciclovir, famciclovir and penciclovir. These agents have few side effects and are effective when started early in the course of the disease. This article highlights the diagnosis, clinical features and management of HHV-associated oral diseases, particularly of those most likely to be encountered by the general dental practitioner.

ACRONYMS
EM: erythema multiforme
HHV: human herpes virus
PCR: polymerase chain reaction
HSV, HHV-1: herpes simplex virus
VZV, HHV-3: varicella-zoster virus
EBV, HHV-4: Epstein-Barr virus
CMV, HHV-5: cytomegalovirus

Key words: herpes simplex virus, human herpes virus-8, varicella zoster virus, Epstein-Barr virus, recurrent herpes labialis, recurrent intraoral herpetic ulcers, treatment, valaciclovir, aciclovir, famciclovir.

INTRODUCTION
The human herpesvirus (HHV) family comprises a diverse group of DNA viruses that, following a primary infection, have the capacity to persist life-long in a latent form. The latent HHVs can undergo reactivation, particularly in subjects with immunosuppressive or debilitating conditions, causing subclinical infection with a low-rate of viral replication or causing frank clinical infection.1,3

The HHV family members comprise the herpes simplex virus (HSV) type 1 (HSV-1) also termed human herpes virus type 1 (HHV-1) and the herpes simplex virus type 2 (HSV-2, HHV-2); the varicella-zoster virus also termed human herpes virus type 3 (VZV, HHV-3); the Epstein-Barr virus (EBV, HHV-4); the cytomegalovirus (CMV, HHV-5); the more recently characterized HHV-6, HHV-7; and HHV-8.1,2,4-7

Any HHV can cause asymptomatic subclinical infection, or clinical infection with or without oral manifestations (Table 1). During active productive infection, depending on the site of replication of the virus, viral particles are shed in saliva, genital secretions, urine, tears or respiratory secretions making HHVs readily transmissible and infectious.6,7

Following primary oral infection with HSV or VZV the viruses ascend in sensory neurons from peripheral
### Table 1: Oral mucosal diseases caused by HUMAN HERPES VIRUSES (HHVs).

<table>
<thead>
<tr>
<th>Human Herpes Viruses</th>
<th>Disease</th>
<th>Oral Clinical Features</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>HHV-1 (HSV-1) HHV-2 (HSV-2)</td>
<td>Primary herpetic gingivostomatitis</td>
<td>Usually affects children under the age of five. Fever, malaise, eruption of multiple vesicles which coalesce and rupture forming ulcers and erosions anywhere on the oral mucosa. The vermilion borders are often affected. There is pain, bleeding and acute gingivitis.</td>
<td>Most primary HSV infections are subclinical. Self-limiting, resolves within 2 weeks, supportive therapy Usually the diagnosis is made on history and on clinical evidence. HHV-2 primarily affects the genital region.</td>
</tr>
<tr>
<td></td>
<td>Recurrent herpetic orolabial lesions</td>
<td>Prodromal symptoms of burning or pruritus, followed by the eruption of small vesicles surrounded by inflammatory erythema; the vesicles rupture readily with subsequent crusting.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Recurrent herpes labialis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Recurrent herpetic oral ulcers</td>
<td>In the mouth, masticatory mucosa of the palate and attached gingiva are most commonly affected. Typically, the vesicles may coalesce and on rupturing, form erosions.</td>
<td></td>
</tr>
<tr>
<td>HHV-3 (VZV)</td>
<td>Varicella (chicken pox)</td>
<td>Oral lesions are inconspicuous, but comprise a few vesicles which rupture rapidly resulting in non-specific erosions.</td>
<td>A common childhood disease with intensely pruritic vesicles and ulcers on the face and trunk. Self-limiting Recovery within 10-14 days.</td>
</tr>
<tr>
<td></td>
<td>Oral Zoster</td>
<td>Strictly unilateral: may affect any oral mucosal site in the sensory distribution of the trigeminal nerve.</td>
<td>Reactivation of VZ virus; adults and elderly; painful unilateral vesicles and ulcers distributed along any sensory nerve course. Self-limiting Complications: - post herpetic neuralgia - corneal involvement and scarring - facial paralysis because of extension to involve the geniculate ganglion</td>
</tr>
<tr>
<td>HHV-4 (EBV)</td>
<td>Hairy leukoplakia</td>
<td>A benign, asymptomatic, white hyperkeratotic lesion affecting primarily the lateral border of the tongue, unilaterally or bilaterally. Affects immunocompromised adults, particularly HIV-seropositive subjects. It is usually asymptomatic and does not have malignant potential.</td>
<td>Starts as glandular fever; fatigue. Associated with nasopha-ryngeal carcinoma</td>
</tr>
<tr>
<td></td>
<td>Ulcers</td>
<td>Very uncommon; non-specific; young adults affected</td>
<td></td>
</tr>
<tr>
<td>HHV-5 (CMV)</td>
<td>Chronic ulcers</td>
<td>Non-specific</td>
<td>Starts as glandular fever Particularly in immunosuppressed subjects</td>
</tr>
<tr>
<td>HHV-6 closely related HHV-7 genetically</td>
<td>Non-specific rash in children.</td>
<td>May or may not be maculopapular eruptions on an erythematous base.</td>
<td>Primary infection usually by 5 years; asymptomatic; may manifest as a febrile illness Reactivation mainly in the immunocompromised, potential to cause pneumonitis, encephalitis, hepatitis and bone marrow suppression.</td>
</tr>
<tr>
<td>HHV-8</td>
<td>Kaposi Sarcoma (KS)</td>
<td>Oral KS most commonly affects hard palate, gingiva; dorsum of the tongue; manifesting as macules, papules, nodules and exophytic masses, ranging from pink to red to blush purple to deep brown. Lesions may be single or multiple varying from a few millimetres to several centimetres. Exophytic lesions interfering with mastication may cause discomfort.</td>
<td>Primary infection maybe subclinical; clinical, non-specific including lymphadenopathy, splenomegaly, fever and arthralgia. Four epidemiological variants: - Classic - Iatrogenic - Endemic - HHV-associated</td>
</tr>
</tbody>
</table>
nerve endings adjacent to the infected epithelium to the trigeminal ganglia where they become latent. Upon reactivation the viruses descend within sensory neurons from the trigeminal ganglia and are released at the nerve endings whence they migrate into the oral epithelium and replicate, causing productive infection that often develops into viral lesions.2,4,9

Epstein-Barr virus (EBV) infects B lymphocytes and oropharyngeal epithelial cells where, following primary infection, they persist in latent form.5,10 CMV is believed to infect and establish latent infection in salivary gland cells, endothelial cells, macrophages, lymphocytes, and possibly in haematopoietic progenitor cells.1,3-6 The closely related HHV-6 and HHV-7 infect and establish latency in CD4+T lymphocytes. HHV-8 infects B lymphocytes where subsequently it persists in latent form and upon reactivation replicates in B lymphocytes and endothelial cells.5,11,13

The aim of this paper is to provide the general dental practitioner with a succinct review of the clinical features and diagnosis of the more common HHV-induced oral diseases and to discuss their management and pharmacotherapeutic treatment.

ORAL MUCOSAL DISEASES CAUSED BY HHVs

Herpes simplex viruses cause primary herpetic gingivostomatitis, recurrent herpes labialis and, less commonly, recurrent intraoral ulcers, together termed recurrent herpetic orolabial lesions.13 It appears that when primary infection with HSV-1 occurs in childhood, it manifests as herpetic gingivostomatitis; but later it may present as oro-pharyngotonsillitis.9 However, most HSV-1 primary infections are subclinical and are acknowledged only when recurrent orolabial herpetic infection occurs without a history of earlier primary herpetic gingivostomatitis14 (Table 1). Primary herpetic gingivostomatitis should be differentiated from other conditions with oral vesicles or ulcers, including erythema multiforme, herpes zoster, hand foot and mouth disease, aphthous stomatitis, recurrent herpes labialis and, less commonly, recurrent herpetic gingivostomatitis.14 (Table 1). Primary herpetic gingivostomatitis is characterized by the presence of sheets and fascicles of spindle cells forming nodular aggregates at the base of the lesions.15 As a rule, the extent and severity of primary and recurrent HHV infections are greater in immunocompromised than in immunocompetent subjects.13,20

There is some supportive evidence for the role of HHVs in the pathogenesis of periodontal disease as DNA particles and gene products can be detected in lesions of periodontal diseases. It is likely that productive subclinical HHV infection of the periodontium can dysregulate local periodontal immune responses and interfere with normal healing and tissue remodeling.21-23

Diagnosis of HSV and VZV-induced oral diseases is usually based on the clinical presentation and on the medical history. Histopathological examination of a biopsy specimen, if deemed necessary, would show ballooning degeneration of infected epithelial cells, inclusion bodies, fusion of cells to form syncytia of multinucleated giant epithelial cells, as well as acantholysis with the formation of Tzanck cells. The most common and cost-effective laboratory method of confirming the clinical diagnosis, however, is by microscopic examination for the presence of multinucleated giant cells or of large cells with ballooned nuclei in a smear preparation from the base of a lesion.6,24 These features are similar in both HSV and VZV induced lesions. If necessary, further differentiation between HSV and VZV can be done by immunofluorescent studies, and typing of the viruses present in the lesion can be determined by immunohistochemistry, in situ hybridization or polymerase chain reaction (PCR).4,6,7,13,24

Diagnosis of EBV-induced oral hairy leukoplakia or of HHV-8-induced oral Kaposi sarcoma is also based largely on the clinical features of the lesions, on the medical history and on microscopic examination of a biopsy specimen.

The characteristic microscopic features of hairy leukoplakia are epithelial hyperplasia, acanthosis, hyperkeratosis, presence of koilocyte-like cells, but with little or no inflammatory cell infiltrate in the underlying lamina propria.25

Kaposi sarcoma exhibits atypical vascular channels, sheets and fascicles of spindle cells forming nodular tumour like masses, extravasated red blood cells and an inflammatory cell infiltrate. HHV-8 DNA can be demonstrated in oral Kaposi sarcoma cells by PCR or by immunohistochemical methods.1,10,20,25

VZV causes chicken pox and zoster (shingles); EBV causes oral hairy leukoplakia, non-specific oral ulcers and is associated with nasopharyngeal carcinoma and a subset of lymphomas; and CMV is associated with salivary gland dysfunction and non-specific oral ulcers. The effect of HHV-6 and HHV-7 infections on the oral mucosa is unknown, but HHV-8 is a critical factor in the pathogenesis of Kaposi sarcoma (Table 1).7,11 As a rule, the extent and severity of primary and recurrent HHV infections are greater in immunocompromised than in immunocompetent subjects.13,20

The prevalence of HSV-1 and HSV-2 infections are the same. The prevalence of HSV-1 and HSV-2 infections is higher in developing than in developed countries.6,8,18,19

HHV-8 infects B lymphocytes where subsequently it persists in latent form and upon reactivation replicates in B lymphocytes and endothelial cells.5,11,13

HHV infection of the periodontium can dysregulate local periodontal immune responses and interfere with normal healing and tissue remodeling.21-23

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TREATMENT

Most HHV diseases are self-limiting and resolve within 7-10 days; but in order to alleviate the symptoms of primary herpetic gingivostomatitis, recurrent orolabial HSV infection, primary oral VZV infection, and oral herpes zoster, all are generally treated non-specifically and supportively with antimicrobial mouthwashes (chlorhexidine by choice), analgesic, soft diet and adequate intake of fluids.6,7,24

In immunocompromised or debilitated subjects, or in immunocompetent subjects with persistent, unusually painful, or frequently recurrent HHV infections, systemic antivirals are necessary (Tables 2 and 3).6,7,24,26,27 Drug treatment will not only shorten the course of the disease, but will also reduce the duration of virus shedding and will promote healing.1 In these subjects, recurrent orolabial herpetic lesions are usually larger and run a more severe clinical course than in immunocompetent subjects. In subjects with AIDS, the larger herpetic ulcers mimic major aphthous ulcers or necrotizing stomatitis so histopathological or cytological examination may be necessary for diagnosis and PCR or in-situ hybridization may reveal the presence of several different HHVs.6

The most commonly used antiviral agents in the treatment of oral HSV or VZV infections are the nucleoside analogues, aciclovir, valaciclovir, famciclovir and penciclovir (Table 3). These agents inhibit viral DNA polymerization and disrupt the viral DNA synthesis and replication, while having only a minimal effect on the host-cellular DNA synthesis.1,4,18,24,26 Long-term use of antivirals does not seem to cause the emergence of drug resistant viruses. Valaciclovir and famciclovir have a greater bioavailability when taken orally and consequently are administered less frequently per day than aciclovir. However, as the effectiveness of these drugs when taken according to the prescribed dosing schedule is similar, and as aciclovir is substantially less expensive, it is the most frequently prescribed first-line antiviral agent.20-32

As a rule, treatment with systemic or topical antiviral agents should be started as early as possible in the course of symptomatic HSV and VZV infections, as this may minimize or prevent the development of new lesions, accelerate resolution and significantly shorten the period of viral shedding.5,7,15

Factors to be considered in deciding whether or not to use antiviral agents for the treatment of symptomatic HSV and VZV oral infections, which are usually self-limiting, include the severity and extent of the disease, the duration of the condition since the onset (according to the patient), the age of the patient, the fitness of the immune system as deduced from the history and the potential side effects of the drugs.15,33 Those who will benefit most from the use of antiviral agents are immunocompromised subjects, or immunocompetent subjects with a history of frequent, painful and persistent episodes of recurrent orolabial HSV infection or of VZV infections. In such cases, systemic agents are more effective than topical agents in reducing the duration of signs and symptoms and in promoting healing.15

Antiviral drugs are best started in the prodromal or early stage of viral infection, so any patient with a history of frequent recurrent orolabial herpes infection should be provided with a small stock of the selected antiviral drug so that treatment can be started promptly at the onset of recurrence, avoiding the delay of professional consultation and prescription. However, self-diagnosis, and the potential adverse effects of self-medication are issues of some concern.15

PRIMARY HERPETIC GINGIVOSTOMATITIS

Active herpetic lesions are highly infectious so anyone with primary herpetic gingivostomatitis should avoid close contact with others to prevent transmission and spread of the virus; and should be aware of the hazards of auto-inoculation.6

In those cases of primary herpetic gingivostomatitis when pharmacotherapeutic agents are necessary, the recommended drug regimens are as in Tables 2 and 3. Treatment is best if started within the first three days of onset of the disease.1,6,7,34

RECURRENT HERPETIC OROLABIAL LESIONS

Recurrent herpes labialis, also termed ‘cold sore’ or ‘fever blister’, affects the vermilion border and the immediately surrounding skin and is the most common clinical manifestation of recurrent HSV-1 infection. Up to 50% of subjects will experience at least one episode of recurrent herpes labialis.1,18

Aciclovir 5% cream or penciclovir 1% cream when applied topically, if possible eight times a day from the start of the prodrome reduces morbidity and promotes healing.6-8,35 For management of severe recurrent herpes labialis, see Tables 2 and 3.6,36

When recurrent herpes labialis is triggered by exposure to sunlight during skiing or sunbathing, prophylactic use of aciclovir 400mg twice per day, valaciclovir 1g per day or famciclovir 250mg twice per day, together with the use of sunscreen for the duration of the vacation is generally effective.6,4 When episodes of apparently spontaneous recurrent infection are frequent and debilitating, occurring more than six times a year, long-term prophylactic treatment with antiviral agents, such as aciclovir 400mg twice per day or valaciclovir 500mg once daily for 4-12 months should be considered. Side-effects of long-term use of these agents are minimal (Table 2).

Sometimes, for unknown reasons, herpetic lesions occur following a local anaesthetic injection in the hard palate. When there is a history of this having occurred, then 2g valaciclovir taken twice on the day of the anticipated injection in the palate, and 1g taken twice on the next day has been reported to prevent or limit the herpetic eruption15,18,37 (Table 2).

HSV-ASSOCIATED AND IDIOPATHIC ORAL ERYTHEMA MULTIFORME

HSV-associated recurrent erythema multiforme (EM) is indirectly induced by an immune response to subclinical or clinical HSV infection, and in some cases, putatively idiopathic EM may also be provoked by HSV, as might be demonstrated by appropriate molecular investigations. In general, prophylactic treatment against HSV infection for 6-12 months may be beneficial for subjects who have
Table 2: Pharmacotherapeutic treatment regimens for oral mucosal diseases caused by HUMAN HERPES VIRUSES (HHVs).

<table>
<thead>
<tr>
<th>Human Herpes Viruses</th>
<th>Disease</th>
<th>Treatment Regimen: for practical purposes any of the following can be prescribed, and there is no clear evidence that any particular regimen is superior.</th>
</tr>
</thead>
<tbody>
<tr>
<td>HHV-1 (HSV-1)</td>
<td>Systemic:</td>
<td>Aciclovir 100-200mg x5/d for 7-10 days/or until resolution of symptoms; or Aciclovir oral suspension 200mg/5ml x5/d for 7-10 days/or until resolution of symptoms; 7 for children, 15mg/kg x5/d for 7 days (keep in the mouth for 2-5 minutes, then swallow); Valaciclovir 1000mg x2/d for 7-10 days/or until resolution of symptoms 26</td>
</tr>
<tr>
<td>HHV-2 (HSV-2)</td>
<td>Recurrent herpetic orolabial lesions</td>
<td>Topical: Penciclovir cream (1%) 8/d (2-hourly intervals, while awake), start at the beginning of the prodromal symptoms and continue for 4 days; or Aciclovir cream (5%) 8/d (2 hourly intervals, while awake), start at the beginning of the prodromal symptoms and continue for 4 days 7</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Systemic: Aciclovir 400mg x5/d for 7 days 27; or Famciclovir 500mg X2-3/d for 1 day 27; or Valaciclovir 2000mg x2/d for 1 day, 2g as soon as the prodrome is identified and 2 g 12 hours later 27,42</td>
</tr>
<tr>
<td></td>
<td>Prophylaxis for recurrent herpetic orolabial lesions</td>
<td>Systemic: Aciclovir 400mg x2-3/d 27; or Valaciclovir 1g/day 8; or Famciclovir 250mg X2/day 8 Start just before exposure to a known trigger and continue during the precipitating event For frequent and debilitating episodes occurring spontaneously more than six times per annum Valaciclovir 2g taken twice on the day of the procedure and 1g taken twice in the following day 8,10,27</td>
</tr>
<tr>
<td>HHV-3 (VZV)</td>
<td>Varicella (chicken pox)</td>
<td>Systemic: Aciclovir 100-200mg X5/d for 7 days; or Aciclovir oral suspension 200mg/5ml X5/d for 7 days 7</td>
</tr>
<tr>
<td></td>
<td>Oral zoster</td>
<td>Systemic: Aciclovir 400-800mg X5/d for 7 days; or Aciclovir oral Suspension 200mg/5ml (4 times a day for 7 days); or Valaciclovir 1g x3/d for 7 days 17,27; or Famciclovir 500mg x3/d for 7 days 17,27</td>
</tr>
<tr>
<td>HHV-4 (EBV)</td>
<td>Hairy leukoplakia</td>
<td>Antiviral agents are not effective</td>
</tr>
<tr>
<td>HHV-8</td>
<td>Kaposi's sarcoma (KS)</td>
<td>Antiviral agents are not effective</td>
</tr>
</tbody>
</table>

Table 3: Selected antiviral preparations available in South Africa

<table>
<thead>
<tr>
<th>Active Agent</th>
<th>Systemic</th>
<th>Topical</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aciclovir</td>
<td>200mg tablets (*Cyclivex®; *Aciclovir Sandoz 200®; *Adco-Acyclovir®) 400mg tablets (*Aciclovir Sandoz 400®; *Cyclivex®) 200mg/5ml suspension (<em>Zovirax suspension</em>)</td>
<td>50mg/g (5%) cream (*Acitop®; *Zovirax®; *Activir Pump Pack®; *Actvir®; *Lovire®; *Adco-Acyclovir®) 30mg/g (3%) ointment (*Zovirax®)</td>
</tr>
<tr>
<td>Valaciclovir</td>
<td>500mg tablets (*Hevitrex®; *Vorix®; *Zelitrex 500®; *Mylan Valaciclovir®; *Anviro 500®; *Vavirex 500®; *Zelivire 500®) 1000mg tablets (Actiprez 1000®)</td>
<td>Not available</td>
</tr>
<tr>
<td>Penciclovir</td>
<td>125mg tablets (*Farnvir®; *Ciplafam 125®) 250mg tablets (*Farnvir®; *Ciplafam 250®)</td>
<td>10mg/g (1%) cream (*Fenivir®)</td>
</tr>
</tbody>
</table>

a Aspen Pharmacare, Woodmead, South Africa; b Sandoz SA, Pinetown, South Africa; c Adcock Ingram, Bryanston, South Africa; d GlaxoSmithKline South Africa, Bryanston, South Africa; e Cipla SA, Bellville, South Africa; f Ranbaxy (Sun Pharma), Centurion, South Africa; g Bliss Pharmaceuticals, Johannesburg, South Africa; h Mylan South Africa, Modderfontein, South Africa; i Zydus Healthcare SA, Potchefstroom, South Africa; j Arrow Pharma, Germiston, South Africa; k Novartis, Johannesburg, South Africa.
experienced several episodes of recurrent HSV-associated or idiopathic EM per year. Suppression of HSV replication by aciclovir 400mg, or valaciclovir 500mg or famciclovir 250mg twice daily will prevent or reduce the frequency of the EM. However, treatment with antiviral agents has no effect on the clinical course of overt EM.

ORAL HERPES ZOSTER
Antiviral agents started within 72 hours of the development of the first oral zoster vesicles may reduce both the severity and the duration of the infection and minimise the risk of post herpetic neuralgia.\textsuperscript{17,37} Recommended drug regimens are as in Table 2.

THE RISK OF HSV TRANSMISSION AND ITS IMPLICATIONS FOR ORAL HEALTH-CARE PERSONNEL
As discussed earlier, reactivation of latent HSV in the trigeminal sensory ganglion with consequent replication of the virus in the oral or perioral epithelium may manifest clinically as recurrent orolabial herpetic lesions; but HSV infection usually occurs asymptomatically and subclinically. In both clinical and subclinical infections there is shedding of the virus from the oral epithelium into the oral fluids.\textsuperscript{6,8,38} Clinical and virological research have shown that about 6% of subjects free of clinical oral HSV infection nevertheless shed HSV-1 into their oral fluids on any given day and that about 70% shed the virus at least once a month.\textsuperscript{38} In healthy immunocompetent subjects each episode of HSV-1 shedding lasts for one to three days and is increased by local trauma or inflammation. In immunocompromised subjects the frequency, duration and quantity of oral shedding of HSV is increased. The load of HSV-1 in the infected oral fluids, even in the case of subclinical infections is sufficient to make the saliva a vehicle of potential viral transmission.\textsuperscript{38} The viruses in oral fluids may be dispersed in aerosols generated by normal breathing, coughing or spitting and by kissing or food sharing.\textsuperscript{39} However, although HSV is highly transmissible, most recipients of the transmitted virus will not develop HSV disease.\textsuperscript{6,7}

Recurrent orolabial herpetic infection is a potential occupational hazard for oral health care workers. Their risk of infection is particularly high when aerosols are generated by high-speed rotary and ultrasonic instruments even if the patient has subclinical HSV infection.\textsuperscript{39} Therefore, the importance of universal infection control measures cannot be overemphasized, and elective dental and oral treatment of patients with overt clinical orolabial herpetic infection should be deferred.\textsuperscript{40}

Herpetic whitlow, that is a primary or recurrent HSV infection of the finger, may result from self-inoculation in subjects with primary or recurrent orolabial or genital HSV infection; or in the case of oral health care workers from treating patients with active herpetic infection. Indeed, before the introduction of universal precautions against infection, dentists had a higher frequency of herpetic whitlow than the general population.\textsuperscript{6,40}

CONCLUDING REMARKS
HSV and VZV infections of oral and perioral tissues are usually self-limiting requiring only supportive treatment. However, susceptible subjects can have frequent, persistent and disabling episodes of these oral infections, and prophylactic treatment with systemic antiviral agents are usually helpful.

Conflict of interest: None declared.

References


Knowledge and beliefs about oral pseudomembranous candidiasis among traditional health practitioners in Limpopo Province, South Africa.

ABSTRACT

Introduction: Oral pseudomembranous candidiasis (OPC) is commonly associated with immunosuppression caused by HIV/AIDS and TB infections. The knowledge and beliefs about OPC among traditional health practitioners in South Africa are not well explored.

Purpose: To investigate this concern.

Methods: A cross-sectional descriptive survey was conducted in the rural Vhembe district of the Limpopo Province. Data were collected from 427 traditional health practitioners who were to attend training workshops on HIV/AIDS and TB diseases. An open-ended semi-structured questionnaire with an A4 colour picture of oral pseudomembranous candidiasis was presented to assess their previous exposure to, and their knowledge and beliefs about, the etiology and management of OPC.

Findings and conclusion: Only thirteen percent correctly identified the lesion. More than 64% were uncertain on etiology, and 24% blamed witchcraft and supernatural powers. Almost two thirds (60%) were confused about the relationship between HIV/AIDS and OPC lesions.

The belief that witchcraft and ancestors could cause OPC could increase the risk of HIV infections and result in delays in seeking treatment. The outcomes of this study should be incorporated in the training of the traditional health practitioners on oral signs and symptoms of HIV/AIDS diseases.

Keywords: Oral pseudomembranous candidiasis, knowledge and beliefs, traditional health practitioners (THPs).

INTRODUCTION

Oral pseudomembranous candidiasis (OPC), commonly called oral thrush, is an oral infection caused by yeast of the genus Candida and particularly Candida albicans. It is characterized by white patches on the surface of the buccal mucosa, tongue, and the soft palate. This condition has been observed over the years, and is typically associated with elderly patients, infants and medically compromised individuals.

Candida albicans are among the many organisms which live in the human mouth and gastrointestinal tract. Up to 75% of healthy individuals carry the yeast Candida as part of their normal commensal oral microflora. Since the 1980s there has been a surge of interest and associated research into OPC. This has largely been due to an increased incidence of OPC among immunocompromised patients and its association in patients infected by the human immunodeficiency virus (HIV). Oral pseudomembranous candidiasis occurred in up to 90% of HIV positive patients at some point during the course of the disease. More recently, with the advent of anti-retro-viral (ARV) therapy, this frequency has become very low. Manifestation is associated with high viral loads, low CD4+ T cell count (<200 cells/mm³), increased morbidity and has been shown to negatively affect the quality of life of HIV/AIDS patients.

The prolonged course of HIV infection predisposes these patients to recurrent episodes of OPC that can increase in frequency and severity with progressive HIV disease.

In most African countries, including South Africa (SA), traditional health practitioners, formerly referred to as witchdoctors and/or Sangomas, play a crucial role in providing primary health delivery including taking care of people living with emerging diseases such as HIV/AIDS. Published reports indicate that between 60-90% of the population in SA consult traditional healers.

Oral pseudomembranous candidiasis was present prior to the discovery of HIV/AIDS. It is highly likely that traditional
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health practitioners were, and still are, treating and taking care of patients presenting with OPC. These patients would be managed according to traditional beliefs and understanding of the source of the disease. The introduction of the Traditional Health Practitioners Act (No 22 of 2007), was aimed at legitimizing their traditional beliefs on supernatural powers and ancestors, and integrating the profession into the main health system in SA.23

In 2013, there were approximately 6.4 million persons infected with HIV/AIDS in SA.23 As the awareness among the traditional health practitioners of the association of OPC with HIV/AIDS is relatively low,24 these practitioners are likely to be managing some of the HIV patients presenting with OPC.

The purpose of this study was to determine the knowledge and belief amongst traditional healers, regarding oral pseudomembranous candidiasis, and its associated with HIV and AIDS. This is particularly important in sub-Saharan Africa, where many people are unaware of their HIV/AIDS status, as the recognition of OPC by traditional health practitioners may result in earlier detection of AIDS and consequent referral to the allopathic health facilities for further management.

METHODS

Study setting
The study was conducted in Vhembe District, one of 52 districts of South Africa. The district is located in the Limpopo Province, and shares its borders with Zimbabwe, Botswana and Mozambique. Vhembe district has four local municipalities categorized as rural. Based on the 2015 mid-year population estimates, the district has approximately 1.6 million inhabitants, with an average life expectancy of 58.2 years.25 More than 85% of the inhabitants in this district live in tribal areas, where traditional practices, beliefs and consultation of traditional health practitioners form part of their culture.23-25

Study design and sample size
A cross-sectional descriptive survey was conducted among traditional health practitioners attending training workshops on the signs and symptoms of HIV/AIDS and TB diseases. Delegates to the training workshops were selected by their respective organisations and recommended by the Vhembe traditional Health Council and traditional leaders. Eight HIV/AIDS training workshops were conducted between January 20 and August 30, 2014, in four local municipalities under the Vhembe district. A total of 437 traditional health practitioners attending the training workshops.

Data collection tools
The data were collected using a semi-structured questionnaire, prepared first in English and then translated into Tshivenda and Xitsonga. To maintain the consistency of the tool it was back-translated to English by two different holders of traditional sciences, ancestors, during the ritual ceremony (muphaso). The questionnaire was pre-tested on ten traditional health practitioners who were attending a meeting on ritual killings in Vhembe district. The questionnaire was consequently amended and adjusted to accommodate the desired data for the study.

An open-ended semi-structured questionnaire including an A4 colour picture of OPC [Figure 1] on the palate, was used to assess the pre-training knowledge and beliefs on OPC and the socio-demographic information. Five open ended questions included: 1) What is this condition? 2) What is the cause of it? 3) Have you treated such a condition before? 4) How did/ would you manage the patient? 5) Is it associated with HIV/AIDS? The responses were captured by a research assistant who had attended the necessary training on the objective of the study, data collection techniques, procedures and instruction on how to complete the questionnaires.

Ethical consideration
Ethical clearance was obtained from the Research and Ethics Committee of the University of Pretoria (REC 399-2013). Access permissions were granted by the Limpopo Provincial Health Research Council and Traditional Health Practitioners Council. A special permission to enter the traditional healers’ “sciences and world” was sought and granted from Wali (Supreme Being) and the holders of traditional sciences, ancestors, during the ritual ceremony (muphaso). Participation by the THPs was voluntary. All participants were informed that they could withdraw from the study at any time. Literate participants gave their consent through signing. Those who were illiterate agreed to participate in front of a representative of the traditional health practitioners.

Statistical analysis
The data were entered into Excel and exported to Stata 11 (Stata Cooperation). The descriptive findings were depicted in tables and graphs. For the response to the five open-ended questions, the “general inductive approach” was used to analyse the qualitative data. A coding scheme was established based on a preliminary analysis of a representative sample of the material, without any references to an existing theory or framework.26 Repetitive reading of the responses to the open ended questions was done, aimed at providing an understanding of the data in context. Twenty percent of the responses were read and coded by the researcher and research assistant together. Emerging responses were determined and coded accordingly. The remaining data was coded using the determined coding.

RESULTS

Demographic characteristics of traditional health practitioners (THP)
A total of 437 THPs were interviewed. The largest numbers of traditional health practitioners were diviners (23%) and family practitioners (51%). These are traditional healers who perform divination (throwing of bones or ancestral channeling), or who were family protectors (u vheka mu) and birth attendants. There were 36 (8.2%) respondents who used their knowledge of plants, herbs and animal products to cure illnesses. Spiritualists, including the faith healers, prophets and fortune tellers (u femba), accounted for 77 (17.6%) of the sample. There
With regard to gender, almost two-thirds were female. Half of the traditional health practitioners were above 60 years of age, 32.9% were between 41 and 60 years old; whilst 10 (2.3%) were minors (below 18 years of age). Almost thirty-five percent (34.8%) of the sample reported that they had no formal education, 178 (40.7%) reported that their highest level of education was primary school. Only 43 (9.8%) traditional health practitioners had achieved senior certificate level and 14 (3.2%) had a tertiary qualification.

Their years of experience as traditional health practitioners varied from less than 5 years (18.5%), between 6-10 years (22.2%), 11-20 years (31.8%) and 21 years and more years (27.5%). Almost all participants (93.4%) reported that this was their first attendance of a HIV/AIDS and TB training workshop. Almost 90% of traditional health practitioners reported not knowing their HIV status.

The response of the respondents with regard to the questions on OPC is summarised below.

Knowledge on the OPC and the causes of it
Thirteen percent of the participants were able to identify the lesion as OPC. The majority of those who correctly identified the lesions were diviners, followed by herbalists and faith-based healers. Their description of the lesion in indigenous terminology—“mahada” (white snow covering the surface) and “Vhudaadaa” (distress of speech associated with pain), was similar to what an allopathic health practitioner would call an OPC. A larger percentage (71.8%) indicated that they were unsure about this condition.

The knowledge regarding the aetiology of OPC varied. More than 64.3% were not sure about the causes of this lesion. Almost a quarter (24.4%) of the respondents thought that OPC was caused by witchcraft and supernatural powers such as evil spells and ancestors. One THP stated that…

OPC was caused by witchcraft and supernatural powers. Almost a quarter (24.4%) of the respondents thought that OPC was caused by witchcraft and supernatural powers. One THP stated that…

Previous exposure to OPC and its management
Almost half (46%) of the respondents did not recall seeing patients with such lesions. Among those who had seen patients presenting with OPC, a small number had referred their patients (38%) to other THPs, whilst others (57%) managed the patients using herbal medicine.

Association of OPC with HIV/AIDS
The knowledge on the association of OPC with HIV/AIDS infections was assessed. A small percentage of THPs (15.3%) reported that OPC may be as a result of HIV/AIDS infections. Almost double that percent (24.6%) said that there was no relationship with HIV/AIDS whilst sixty percent (60.1%) were unsure about any relationship.

DISCUSSION
Characteristics of traditional health practitioners
The profession of the traditional health practitioner has been practiced for centuries in Africa, long before western medicine became the dominant health system. Their knowledge and practice of the medicinal use of herbs, roots, and other items has been passed down through generations. It is acquired during their ancestral calling to become a traditional health practitioner (thwasana). A call to become a traditional health practitioner could start at any age and may present in different ways including unexplained illness, constant headaches, dreams and nightmares associated with ancestors and, loss of weight.

Unlike in the Western medical schools, becoming a traditional health practitioner does not follow selection criteria such as grades achieved at high schools, entry level examination, prescribed age limit etc. The majority of THPs were women between 41 and 60 years with no formal education (34.8%). This compared well with Vhembe District Profile, which reported that over 53% of the district’s population were female, with little or no formal education. In 2002, Berea found that a much higher percentage (59%) of her participants were not educated. This may influence the findings as lower levels of education have been found to correlate with lower level of knowledge of transmission, treatment and prevention of HIV/AIDS and TB. It has also been demonstrated that black people generally tend to have poorer HIV/AIDS knowledge due to low literacy levels associated with a lack of exposure to formal education.

The fact that more than two-thirds of the respondents (75%) had limited basic literacy suggests that extra measures and cautions should be considered in the planning, developing and alignment of the training materials to ensure appropriate relevance.

The wealth of experience and depth of knowledge of traditional medicine and patient management is derived from ancestral spirits incarnated during their initiation process (u wisiswa). The knowledge and practice of herbs, roots, and other medicine has passed down through generations. Compared with allopathic health professionals who are required to attend continuous training and updating on the latest medical developments, the traditional practitioner is expected to comply with the ancestral spirit that called them to become THPs. In a Western paradigm these fields and practices are considered backwards, unscientific, erroneous and potentially harmful. Lack of collaboration and inadequate formal referral between the two health systems could result in patients having drug overdoses, over treatment or drug interactions.

Knowledge and beliefs on the causes of OPC
It was noted that most of the THPs were not able to identify OPC. This was not surprising, as an intra-oral examination on “healthy” patients presenting with no oral health complaints is probably not a common practice among THPs. Having said that, OPC is one of the most common oral lesions associated with HIV/AIDS patients. Unless the patient has secondary infections such as gingivitis and periodontitis, OPC is generally not painful, and the patients may not be aware that s/he has a lesion. Recent findings by Ramphoma and Ndikoo revealed that even the oral health care workers working in well-resourced facilities were not vigilant enough.
in checking for oral lesions. Some of them were unable to correctly identify oral lesions associated with HIV/AIDS as they lacked adequate knowledge of these lesions.

This reported low knowledge level of OPC is comparable to a study conducted by Rudolph et al. They found that only 22.4% of 63 THPs knew about OPC. Ensuring the ability of THPs to recognise and identify that these lesions are associated with HIV/AIDS infections provides a golden opportunity to engage them in the fight against HIV/AIDS.

A question on “what is the cause of these lesions” revealed their beliefs in supernatural powers and the depth of traditional medicine. These responses were most probably the reflection of the past and present prevailing beliefs among THPs, especially amongst diviners and prophets. A quarter of the respondents (24.4%) believed that OPCs was caused by evil forces and ancestors. The explanation offered on what is thought to be the cause of OPC appeared to link with the indigenous name for it: vhudaadaa, which has been described above as “distress of speech associated with pain”. When literally translated this Venda word means that “one is not sure”. When one is unsure of the cause- it becomes vhudaadaa. This group of THPs could not explain the cause and origin of the lesion.

The other meaning, “distress of speech associated with pain”, could be associated with the fact that OPC was seen among the immunocompromised patients and in infants, who were unable to vocalise at all. The appearance of OPC in a six-month old child may not, however, herald a serious condition. It could be as a result of a transition period when- in the child is developing his/her own immunity and teeth are erupting in the mouth. However, the underlying cause should always be determined to rule out the possibility of a serious commensurate condition such as diabetes or immunosuppression caused by HIV/AIDS and other serious infections, of which both the patient and THP may not be aware. Any training course on HIV/AIDS should take into account the relevance of linking HIV/AIDS infections and OPC and the importance of contextualising the relevance of indigenous names and their meaning in the past and now.

**Management and the relationship of OPC to HIV/ AIDS infections**

The management of illness and diseases among THPs has usually been in line with an understanding of the cause of the disease. It was therefore not surprising to note that most of the THPs chose to refer their patients to AHPs (78.3%). This may be a positive result in the sense that THPs understand their role and recognise that some conditions are outside their expertise/ healing methods. It is also positive in that such a referral may address the underlying medical conditions such as HIV/AIDS, diabetes and other immunocompromising diseases.

Only 13.0% preferred to use herbal medicine to manage their patients. If in doubt, patients would be referred to other THPs (8.7%) for treatment or for a second opinion, and possibly to specialist traditional health practitioners. No further questions were posed on the questionnaire to explore why OPC patients were referred to AHPs and how it was being done, whether it was self-referral, or accompanying patients. A referral to an AHP appears to be a matter of great concern for health authorities as OPC has been known to occur in up to 90% of HIV positive patients.2 This has changed in the past few years with the introduction of ARVs.

Assessing the knowledge and management of these lesions by THPs could assist HIV/AIDS and TB trainers to understand traditional beliefs and practices related to OPC. As both the diagnosed and un-diagnosed HIV patients consult THPs at various stages of the diseases, the training of the THPs on OPC could alert them to refer their patients for confirming laboratory tests. Greater awareness may also encourage patients to check their HIV status and to take the necessary precautions to prevent spreading the disease.

The training and the involvement of THPs in the management of HIV/AIDS patients has been a thorny issue.15-17 Flint and Payne argued that the training of THPs in SA on HIV/ AIDS will assist in the fight against HIV/AIDS infections.2 Homsky and others suggested that trained THPs are key to scaling up comprehensive care for HIV/AIDS.27 Despite the calls made by WHO for integration, the facts that AHP are university trained and use evidence as determined by Western medicine whilst THP are selected on a calling and practice traditional healing, may make it a challenge for these healers to co-exist.30-35 However, taking into account that the THPs may be the first contact for patients seeking care, it is incumbent on the AHP to understand THP, their knowledge, practices and beliefs. This understanding should inform the development of the relevant educational interventions that, combined, would promote the health of the population which is a responsibility of the health (and other) sectors.31-33

Certain traditional practices could increase THPs risk of contracting HIV. It is common practice for THPs to use bare hands (ungloved) to apply topical medicine on open wounds and sores such as Herpes Zoster, thought to be caused by ancestors. Many also utilize their mouths to suck blood from their patient’s body as part of disease management.54 Lack of knowledge of infection control and no access to hand gloves may result in THPs handling HIV/AIDS-related lesions directly.

The researchers believe that engagement with THPs on best alternative practices and the sharing of knowledge through collaboration could help prevent further spread of HIV and TB infections. Collaboration with THPs may require a change of attitudes and mind sets of the AHPs, and a recognition that THPs also have a role to play in the delivery of health care in SA. It is through exposure and sharing of information that attitudes may change.

**CONCLUSION AND RECOMMENDATIONS**

This study has shown that despite the low knowledge level and etiology of OPC amongst THP’s, they are nevertheless managing OPC without knowing that it may be a result of HIV/AIDS.

Noting that the majority of patients consult THPs on a daily basis, and the fact that South Africa is experiencing an
HIV/AIDS and TB pandemic, urgent focus should be given to conducting further training workshops for THPs on the causes and symptoms of HIV/AIDS and TB diseases.

The existing beliefs that witchcraft and ancestors were the causes of OPC, could well delay patients from seeking allopathic treatment early.

Some patients were referred to AHPs, clearly demonstrating the need for constructive engagement and training workshops to exchange knowledge and information in both directions.

The researchers recommend that HIV/AIDS and TB training workshops be initiated to target the beliefs of THPs on the cause of OPC and to instruct on management practices. AHPs also need exposure to traditional medicine. The starting point may be the inclusion of a traditional medicine module in the curriculum of AHP students and engaging with THPs during training.

LIMITATIONS

Traditional medicine is mostly based on an acceptance of supernatural powers of the ancestors. The diagnosis and treatment modalities are directed and inspired by ancestors in the context of HIV/AIDS and the potential for middle ground between the traditional and biomedical healthcare settings in South Africa. Forum Develop. Stud. 2013; 40:47-68.


Management of an inflamed dentigerous cyst in a patient with an anterior cross-bite, using a modified obturator

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SUMMARY
Dentigerous cysts, amongst the most common odontogenic cysts occurring in children, are caused by the accumulation of fluid between the reduced enamel epithelium and the crown of the tooth. Both developmental and inflammatory aetiologies have been postulated. Enucleation and marsupialisation have remained the mainstay treatment modalities of such cysts although the former is a more radical option—especially in young children with unerupted and developing permanent teeth. A case of an inflammatory dentigerous cyst (IDC) in an 8-year old patient with an anterior dental cross-bite is presented. This was managed by marsupialisation of the cyst and concurrent correction of the anterior cross-bite (within 3 months) using a modified obturator. This treatment modality had the advantage of reducing the treatment time as well as obviating the need for a second appliance. There was no recurrence of either dentigerous cyst or cross-bite at a one-year follow up.

INTRODUCTION
The dentigerous cyst is one of the most common odontogenic cysts and develops due to obstruction of venous drainage by pressure of the erupting tooth on the follicle and consequently an accumulation of fluid between the reduced enamel epithelium and the crown of the tooth.¹,² The cysts are most frequently associated with the mandibular third molar, maxillary canine, mandibular premolars and maxillary third molar teeth, in decreasing order of frequency.³ The mandibular second premolar is the most commonly involved tooth in the 0-9 year old age-group and the permanent maxillary canine has this distinction in the 10-19 year old age-group.¹,⁴ A diagnostic feature is the absence of the affected tooth in the oral cavity.⁵ The cyst is usually asymptomatic and thus may be diagnosed incidentally on routine radiographs. A cyst may attain a large size, causing resorption of the roots of nearby teeth before it manifests clinically or becomes evident radiographically. Although most dentigerous cysts are considered to be of developmental origin, some seem to have an inflammatory pathogenesis. It is impossible to determine histopathologically whether the inflammatory component is primary or secondary in nature.⁷ An unusual case of IDC in

ACRONYMS
IDC: inflammatory dentigerous cyst

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Figure 1a: Pre-treatment photographs - Frontal photograph demonstrating cross bite of 12, 21 and 22

Figure 1b: Pre-treatment photographs - Maxillary occlusal photograph demonstrating retroclined 12, 21 and 22.

Figure 1c: Pre-treatment photographs - Mandibular occlusal photograph showing grossly carious 85 and the location of the swelling.
the right mandibular premolar area, together with an anterior cross-bite involving 12, 21 and 22, is presented in which both cyst and cross-bite were managed conservatively using a simple appliance (modified obturator).7

**CASE REPORT**

An 8-year-old male patient reported to the Unit of Paediatric Dentistry, Oral Health Sciences Centre Post-graduate Institute of Medical Education & Research, Chandigarh, with a chief complaint of a swelling in the mouth on the right side, first noticed two months previously. The patient gave a history of intermittent pain in that area for the past 15 days. The pain was moderate in nature and subsided on the taking of analgesics. Physically, the patient was healthy and there were no signs of any systemic disorder. Extra-orally, no swelling was visible in the right mandibular area; however on palpation a bony hard swelling was felt which was slightly tender and non-fluctuant. The sub-mandibular and sub-mental lymph nodes were not palpable and temporomandibular joint movements were found to be smooth and well coordinated. Intra-orally, the patient was in the early mixed dentition stage of dental development with active caries on 55, 65, 26, 36, 75, 85 and 46 (Figure 1, a-c). The dentition exhibited generalized fluorotic stains. Teeth 84 and 85 were mobile and the bony hard, slightly tender, swelling was felt in the buccal vestibule. There was no sign of a draining sinus associated with the swelling. Teeth 12, 21 and 22 were in a cross-bite relationship with the lowers. The vulnerable labial muco-gingival tissues showed inflammatory enlargement, but there was Class I gingival recession associated with the labial displacement of tooth 31 (Figure 1, a-c). Testing this tooth for fremitus was negative with no mobility. An orthopantomogram was taken which showed a unilocular radiolucency extending from distal surface of 42 to mesial surface of 46. There was displacement of the unerupted 43 and 44 and the roots of 84 and 85 showed premature resorption (Figure 2a).

Based on the clinical signs and symptoms, a provisional diagnosis of dentigerous cyst was reached. Considering the age of the patient and the underlying unerupted and displaced 43 and 44, a conservative treatment plan was adopted which consisted of marsupialisation of the lesion by creating a window through the extraction sockets of 84 and 85 and the provision of an acrylic obturator to maintain its patency.

Impressions of the patient’s upper and lower arches were taken. The plaster renditions of teeth 84 and 85 were ground off the poured cast. The bony window was reproduced in the cast and an acrylic extension into the cavity was made to serve as the obturator (Figure 3a). It was decided to modify the obturator by incorporating bilateral posterior acrylic bite blocks (Figure 3b) to dis-occlude the anterior teeth, allowing for possible self-correction of the anterior cross-bite. At surgery, the 84 and 85 were extracted, the cyst was marsupialised (Figure 3c) and its lining sent for histopathological examination. The obturator was fitted immediately post-surgery.

Histopathologic examination (Figure 4) revealed a keratinized stratified squamous epithelial lining of variable thickness. A space can be seen at the epithelial-connective tissue interface due to weak junction. The connective tissue wall shows loose fibrous stroma with chronic inflammation comprising chiefly of lymphocytes. A. H & E staining (10X) b. H & E staining (40X)

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**Figure 2a:** Orthopantomographs - Pre-treatment.

**Figure 2b:** Orthopantomographs - 6-months post-treatment. Considerable improvement in eruption of 44 and 45.

**Figure 2c:** Orthopantomographs - 1-year post-treatment. Crowding of the lower right segment and impaction of 43 remain a problem.

**Figure 3a:** Modified obturator - Acrylic extension to obturate surgical window.

**Figure 3b:** Modified obturator - posterior bite blocks, shorter on the right side to avoid biting pressure over the operative site.

**Figure 3c:** Modified obturator - Underlying 45 visible after the creation of a bony window.

**Figure 2b:** Orthopantomographs - 6-months post-treatment. Considerable improvement in eruption of 44 and 45.

**Figure 4:** Photomicrographs showing keratinised stratified squamous epithelial lining of variable thickness. A space can be seen at the epithelial-connective tissue interface due to weak junction. The connective tissue wall shows loose fibrous stroma with chronic inflammation comprising chiefly of lymphocytes. A. H & E staining (10X) b. H & E staining (40X)
space could be seen between epithelium and the connective tissue due to a weak junction. The connective tissue wall showed loose fibrous stroma with chronic inflammation comprised chiefly of lymphocytes. Based on the histopathologic findings a definitive diagnosis of IDC was made.

The patient was recalled daily for seven days postoperatively for irrigation of the socket with Betadine and saline. The parents of the patient were taught how to irrigate the socket with these solutions and how to ensure the maintenance of good oral hygiene. Thereafter the patient was recalled fortnightly and periodic trimming of the acrylic extension was done to facilitate eruption of 44 and 45. After three months (Figure 5, a-c), anterior crossbite correction was observed along with progressive eruption of 44 and 45. The 43 appeared to be improving in its path of eruption. The patient was then advised to discontinue the obturator. Follow-up after one year showed complete eruption of 44 and 45, and no relapse of the crossbites or recurrence of the dentigerous cyst (Figure 2c and Figure 6, a-c).

**DISCUSSION**

The inflammatory aetiology for dentigerous cysts was first mentioned as early as 1928 by Bloch-Jorgensen who suggested that all follicular cysts originated due to the overlying necrotic deciduous teeth. The suggested pathogenesis of such cysts was the spread of periapical inflammation from the roots of the deciduous teeth to involve the follicle of the unerupted permanent successor, producing inflammatory exudates which resulted in the formation of the dentigerous cyst. The role of persistent and prolonged inflammation irritating the dental sac of an unerupted tooth thereby causing a dentigerous cyst has also been suggested by other authors. Benn and Altini indicated that at least two types of dentigerous cysts occur. The first type is purely developmental in origin and occurs in mature teeth usually as a result of impaction. The second type is inflammatory in origin and occurs in immature teeth as a result of periapical inflammation from a preceding non-vital deciduous tooth, or other source, spreading to involve the tooth follicle. Benn and Altini considered three possible mechanisms in the histogenesis of inflammatory dentigerous cysts:

1. **Intra-follicular developmental cysts** formed around the crowns of permanent teeth that become secondarily inflamed, as a result of periapical inflammation spreading from non-vital deciduous predecessors.
2. **Radicular cysts** at apices of nonvital deciduous teeth that fuse with the follicles of unerupted permanent successors. “Eruption” of successor teeth into the cystic cavity results in the formation of the extra-follicular dentigerous cyst.
3. **Periapical inflammation** from any source, but usually from nonvital deciduous teeth, spreading to involve follicles of unerupted permanent successors.

Inflammatory dentigerous cysts are only found in the mixed dentition and involve the permanent teeth. Radiographically, they may appear as round or ovoid, well demarcated unilocular radiolucencies with a sclerotic border within the mandible. IDCs are usually associated with the roots of a non-vital or necrotic deciduous tooth and the crown of an unerupted permanent tooth.

Histologically, dentigerous cysts of typical non-inflammatory origin are lined by thin non-keratinized stratified squamous epithelium. However, many variations in the thickness of the lining epithelium may be noted depending on type and severity of any subsequent inflammation present.

The choice of treatment should be based on the size and location of the cyst, age of the patient, affected dentition, and the relationship with surrounding vital structures. These factors along with operator skill and preference, determine whether enucleation or marsupialisation of such cysts may be performed. Enucleation is the process mostly indicated for small cysts in which the cyst is completely removed without rupturing. However, for larger cysts this procedure may result in fracture of the mandible, devitalization of the associated teeth, or the loss of indispensable impacted teeth associated with the lesion. Thus, enucleation should be performed when there is no
likelihood of damaging anatomic structures such as the apices of vital teeth, maxillary sinus, neuro-vascular bundles and developing permanent teeth.

The treatment of IDCs includes extraction of the non-vital primary tooth and marsupialisation of the cyst, as illustrated in this case. Marsupialisation consists in creating a surgical cavity through the wall of the cyst, emptying its content, and maintaining continuity between the cyst lining and the oral cavity, maxillary sinus, or nasal cavity, whichever is more closely related. This technique is indicated for large cysts or cysts associated with unerupted teeth in geriatric and pediatric patients or in patients with systemic diseases. This allows healing of the cystic cavity and eruption of the permanent tooth in paediatric patients, provided that these procedures are performed at the normal time of eruption. The main advantage of this procedure over enucleation is its simplicity and a more conservative approach, inflicting less surgical trauma. Since a major disadvantage of marsupialisation is that pathologic tissue is left in situ, a thorough histologic examination is essential.

An obturator is also required to decompress the cystic lesion, maintain patency of the surgical opening and prevent entry of food debris into the cystic cavity. Although this technique is found to be quite successful, full compliance with recommended postoperative oral hygiene measures is critical.

In this case, posterior bite blocks were incorporated in the obturator bilaterally to dis-occlude the anterior teeth and assist in self-correction of cross bite of the lingually locked maxillary incisors. The main advantage of this modification is that it obviated the need for a second appliance to correct the cross bite. The appliance was also easily constructed although speech and mastication difficulties were of concern to the patient for the first few days, after which he became accustomed to manipulating the device. The results of the modified obturator were encouraging and the one year follow-up did not show any sign of recurrence of the cyst or relapse of the cross bite. The lower right first molar has drifted mesially and further orthodontic treatment remains a requirement to deal with crowding, overjet, midline discrepancy and impaction of the lower right canine.

Conflict of interests: None declared

References
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dentin hypersensitivity from baseline after 8 weeks†

20%* reduction in plaque build-up after 24 weeks compared to regular fluoride toothpaste*5

Helps control dental plaque*4,5

29%* improvement in gingival inflammation after 24 weeks compared to regular fluoride toothpaste*5

Supports good gingival health*4,5

For any product safety issues, contact GSK on +27 745 6001 or 0800 118 274

* With twice-daily brushing. † Parkinson C et al., 2013 reported a 33% reduction from baseline in Schiff sensitivity score at Week 8 for a stannous fluoride toothpaste. 2 Sensodyne® Complete Protection combines active ingredient 0.454% stannous fluoride with 5% sodium tripolyphosphate to help prevent extrinsic tooth stain historically associated with stannous fluoride-containing toothpastes.7,8
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Are fissure sealants still relevant as a caries preventive measure?

INTRODUCTION
The groundwork for fissure sealants was completed in 1955 and lead to the introduction in 1971 of the Nuva-Seal Fissure Sealant by L.D Caulk. Since caries predominantly affects the pits and fissures of the teeth of children, these sealants have been shown to be a valuable preventive procedure. FS are cost effective and provide an ideal preventive measure for children who have restricted access to dental services.

However the advent of dental lasers, caries detection systems, fluoride varnishes and novel adhesive systems may have displaced FS as a preventive measure of choice. The aim of this investigation was to establish whether FS remain a relevant preventive measure for the anatomically vulnerable fissure system.

METHODOLOGY
A sample of twenty extracted third molar teeth, which had been erupted and exposed to the oral environment, were selected after visual inspection had confirmed on each tooth the presence of a fissure system that was anatomically vulnerable and suitable for the placement of a FS. The fissure systems of both the control group (n=10) and the test group (n=10) were cleaned with a moist bristle brush to remove any pellicle or debris. Fissure sealants were placed on the teeth of the test group according to the manufacturer's instructions (Clinpro fissure sealant, 3M ESPE).

Thermocycling of the FS group and the control group (with no FS) was performed in a 2% methylene blue solution (MB) for 200 cycles with a dwell time of 75 seconds in 8°C and 50°C MB. This methodology was in accordance with the ISO guideline for “Polymer-based pit and fissure sealants ISO 6874(2005) E”. The teeth were then sectioned in a bucco-lingual direction into slices of a thickness of 200µm. The sections were viewed under 20X stereomicroscope magnification.

DISCUSSION
It has been common practice that anatomically vulnerable fissures receive a FS as a preventive procedure. But do FS still have a place in a modern dental practice when dental lasers, caries detection systems, various fluoride varnishes and novel adhesive systems are available?

All the teeth in the control group (n=10) showed some degree of MB penetration into the porous enamel fissure surface (Figure A). Even fissures that appeared on visual inspection to have no decalcification had MB penetration into the fissure system. In some cases the penetration extended to the dentinal enamel junction, with clearly identifiable enamel prism destruction (Figure B).

The group of teeth that had received FS protection demonstrated that MB penetration into all the fissure systems had been prevented. It became clear that fissure systems, although decalcified and even with an area of debris at the base of the narrow fissure, received adequate protection with FS application against the penetration of the MB solution during thermocycling (Figure C).

CLINICAL SIGNIFICANCE
Fissure sealants can therefore be considered an appropriate preventive procedure for the fissure system. The proven long term retention of up to 48 months of resin sealants in the fissure system of permanent molars provides the much needed resistance to fissure caries for children and adolescents.

Fissure visualisation is essential and therefore a very fine explorer probe (FT10 probe) or pigtail probe should be used during the assessment of the fissure system to enhance the diagnostic skills of the clinician and to assist in the decision to place FS (Figure D).

Resin fissure sealants provide a simple yet efficacious preventive method and should continue to play an important role in the protection of anatomically vulnerable fissures of the teeth of young patients. The retained FS will prevent the progression of fissure demineralization to fissure caries. A recent systematic review indicated that irrespective of the FS material utilised (resin or glass ionomer) the preventive effect was similar and no material was identified as superior. FS as a preventive measure in sound occlusal fissures resulted in an approximate 70-80% reduction in the incidence of occlusal caries versus non treated occlusal fissures.
were shown to be more effective in arresting non-cavitated pit and fissure caries compared with no treatment intervention or topical fluoride varnish application. Dental lasers, caries detection systems, fluoride varnishes and novel adhesive systems all contribute to the endeavour to preserve the dentition, however, based on the available literature, FS remain an important, and, as shown in this study, an effective, part of preventive dentistry.

**Conflict of interests:** None declared

**References**


**Figure A:** MB staining of the fissure system of an anatomically vulnerable tooth with early enamel decalcification.

**Figure B:** The fissure system stained with MB of an anatomically vulnerable tooth with extensive enamel decalcification at the DEJ.

**Figure C:** Fissure system protected by sealant and showing no MB penetration, although debris was present in the narrow fissure, which could not be probed.

**Figure D:** Section demonstrating the importance of using a fine explorer or pig tail probe to carefully explore the fissure system.
INTRODUCTION

A letter was recently sent to members of a research committee which read as follows: “Dear Members. We have 27 protocols to review and will divide them between all members. Each protocol will be evaluated by two people, thus you will all have to evaluate ±9 protocols.”

The response from the resident statistician read: “Hello. I would like to correct this common statement highlighted above. Although it is a colloquial statement, it should be corrected among members. It is preferred to state that “each will evaluate between 7-11 protocols or 9±2 (7-11 protocols).”

This amusing, yet technically correct, anecdote brings home the realization that many researchers, supervisors, reviewers and clinicians do not fully understand many research concepts and statistical terms, nor the significance (non-statistically speaking) behind them. This is the first of a planned series of papers which aim to explain, clarify, and simplify a number of these apparently esoteric principles. With that objective, the series could help future researchers improve their study designs, as well as empower their readers with the knowledge needed to critically evaluate any ensuing literature. The series will begin with definitions and explanations of statistical terms, and then will deal with experimental designs and levels of evidence.

The information and layout of Paper One is based on notes from the University of Barotseland and on the work of Schoeman. However, we recognise that the human mind responds better to stories and illustrations than to numbers and statistics. For this reason the paper has been interspersed with many “Quotes and anecdotes to engage and amuse the reader, and help promote their memory”, referenced by name where possible (Steven Pinker).

Scientific research refers to the “systematic technique for the advancement of knowledge and consists of developing a theory that may or may not be proven true when subject to empirical methods.” It should have an appropriate experimental design that produces objective data and valid results. These should be accurately analyzed and reported, so that they cannot be erroneously or ambiguously interpreted. This of course is in direct contrast to the satirical remark of Evan Esar who defined statistics as “The science of producing unreliable facts from reliable figures.” Classic research presupposes that a specific question can be answered, and then endeavours to do so by using a proper experimental design and following a step-wise approach of defining the problem (usually based on some observation), formulating a hypothesis (an educated guess to try to explain the problem / phenomenon), and then collecting and analyzing the data to prove or disprove the hypothesis.

1. DATA

This refers to any facts, observations, and information that come from investigations, and is the foundation upon which new knowledge is built. To paraphrase Author Conan Doyle “A theory remains a theory until it is backed up by data.” Data can be either quantitative or qualitative.

1.1 Quantitative data is information about quantities that can be measured and written down in numbers (e.g. test score, weight).

1.2 Qualitative data is also called categorical or frequency data, and cannot be expressed in terms of numbers. Items are grouped according to some common property, after which the number of members per group are recorded (e.g. males/females, vehicle type).

2. SAMPLE

In research, the target population includes all of those entities from which the researcher wishes to draw conclusions. However, it is impractical to try to conduct research on an entire population and for this reason only a small portion of the population is studied, i.e. a sample. The inclusion and exclusion criteria will help define and narrow down the target population (in human research). Sampling
refers to the process of selecting research subjects from the population of interest in such a way that they are representative of the whole population.

2.1 The sample population is that small selection from the whole who are included in the research. Inferential statistics seek to make predictions about a population based on the results observed in a sample of that population.

2.2 Sample size refers to the number of patients / test specimens that finish the study and not the number that entered it. When determining sample size, most researchers would want to keep this number as low as possible for reasons of practicality, material costs, time, and availability of facilities and patients. However, the lower limit will also depend on the estimated variation between subjects. Where there is great variation, a larger sample number will be needed. Statistical analysis always takes into consideration the sample size. As Joseph Stalin put it, “A single death is a tragedy; a million deaths is a statistic.”

2.3 Non-responders refers to those persons who refuse to take part in the study, who do not comply with study protocol, or who do not complete the entire study. Their non-participation could result in an element of bias, and can only be ignored if their reasons for refusal will not affect the interpretation of the findings.

2.4 Sampling methods are divided into nonprobability and probability sampling. In the former, not every member of the population has a chance of being selected, while in the latter, they all do have an equal chance.

2.4.1 Nonprobability

a) Convenience sampling refers to taking persons as they arrive on the scene and is continued until the full desired sample number has been obtained. It is NOT representative of the population.

b) Quota sampling is similar to convenience sampling except that those sampled are selected in the same ratio as they are found in the general population.

2.4.2 Probability

a) Random sampling is when the study subjects are chosen completely by chance. At each draw, every member of the population has the same chance of being selected as any other person. Tables of random digits are available to ensure true randomness.

b) Stratified random samples are constructed by first dividing a heterogeneous population into strata and then taking random samples from within each stratum. Strata may be chosen to reflect only one or more aspects of that population (e.g. gender, age, ethnicity).

c) Systematic sampling involves having the population in a predetermined sequence e.g. names in alphabetical order. A starting point is then picked randomly and the person whose name falls in that position is taken as the first to be sampled.

d) Cluster sampling is when the population is first divided into natural subgroups, often based on their being geographically close to each other e.g. houses in a street, staff in one hospital. A number of clusters are then randomly sampled.

2.5 Generalization is an attempt to extend the results of a sample to a population and can only be done when the sample is truly representative of the entire population. Generalizing the results obtained from a sample to the broad population must take into account sample variation. Even if the sample selected is completely random, there is still a degree of variance within the population that will require your results from within a sample to include a margin of error. The greater the sample size, the more representative it tends to be of a population as a whole. Thus the margin of error falls and the confidence level rises.

2.6 Bias is a threat to a sample’s validity, and prevents impartial consideration. It can come in many forms and can stem from many sources such as the researcher, the participants, study design or sample. The most common bias is due to the selection of subjects. For example, if subjects self-select into a sample group, then the results are no longer externally valid, as the type of person who wants to be in a study is not necessarily similar to the population that one is seeking to draw inferences about. Examples of bias could be: Cognitive bias, which refers to human factors, such as decisions being made on perceptions rather than evidence; Sample bias, where the sample is skewed so that certain specimens or persons are unrepresented, or have been specifically selected in order to prove a hypothesis.

2.7 Prevalence refers to the proportion of cases present in a population at a specified point in time, hence it explains how widespread is the disease. (Memory Point – remember all the P’s).

2.8 Incidence is the number of new cases that occurred over a specific time, and gives an indication about the risk of contracting a disease.

3. EXPERIMENTAL DESIGN

Design relates to the manner in which the data will be obtained and analyzed. For this reason, consultation with a statistician is crucial during the preparation phases of any research. Prior to embarking on the study one must already have determined the target population, sampling methods, sample size, data collection methods, and statistical tests that will be used to analyze the findings. Many studies fail or produce invalid results because this crucial step was neglected during the planning stages. As William James commented “We must be careful not to confuse data with the abstractions we use to analyze them”. Light et al were more blunt in stating “You can’t fix by analysis what you bungled by design.”

3.1 Descriptive statistics are used for studies that explore observed data. In descriptive statistics, it often helpful to divide data into equal-sized subsets. For example, dividing a list of individuals sorted by height into two parts — the tallest and the shortest, results in two quantiles, with the median height value as the dividing line. Quartiles separate data set into four equal-sized groups, deciles into 10 groups etc.

3.2 Inferential statistics are used when you don’t have access to the whole population or it is not feasible to
measure all the data. Smaller samples are then taken and inferential statistics are used to make generalizations about the whole group from which the sample was drawn e.g. “Receiving your college degree increases your lifetime earnings by 50%” is an inferential statistic. A word of caution, one has to be very clear of the meaning and interpretation of results presented as percentages. Consider the issue of percentages versus percentage points — they are not the same thing. For example, “if 40 out of 100 homes in a distressed suburb have mortgages, the rate is 40%. If a new law allows 10 homeowners to refinance, now only 30 mortgages are troubled. The new rate is 30%, a drop of 10 percentage points (40 – 30 = 10). This is not 10% less than the old rate, in fact, the decrease is 25% (10 / 40 = 0.25 = 25%).” Another classic example of mis-representation of data was a recent survey on smoking habits of final year medical students. There was only one Indian student in the class who also happened to be a smoker. The resulting report declared that “100% of Indian students smoke”. In the words of Henry Clay, one must still bear in mind that “Statistics are no substitute for judgement”.

3.3 Error
In all research, a certain amount of variability will occur when humans are measuring objects or observing phenomena. This will depend on the accuracy of the measuring tool, and the manner in which it is used by the operator on each successive occasion. Thus, error does not mean a mistake, but rather it describes the variability in measurement in the study. The amount of error must be recognized, delineated, and taken into account in order to give true meaning to the data. When humans are involved, the amount of error can be defined as inter-operator (differences between different operators), or intra-operator (differences when performed by the same operator at different times). To overcome this, a certain number of objects are measured many times and by different people to detect the variation. This will then set the limits as to how accurate the results will be.

3.4 Accuracy, Precision, Reliability and Validity
a) Accuracy is a measure of how close measurements are to the true value.
b) Precision is the degree to which repeated measurements will produce the same results (or how close the measures are to each other).
c) Reliability is the degree to which a method produces the same results (consistency of the results) when it is used at different times, under different circumstances, by either the same or multiple observers. It can be tested by conducting inter-observer or intra-observer studies to determine error rates. Low inter-observer variation (or error) indicates high reliability.

The research must test what is it supposed to test, and how correctly the results are interpreted and subsequently used. A note on sensitivity and specificity. Sensitivity and specificity are used as statistical measures to determine the effectiveness of a medical diagnostics. Sensitivity is a measure of the number of true positives and is calculated from the formula [true positive/true positive + false negative], while specificity is a measure of the amount of true negatives and is calculated by [true negative/true negative + false positive].

4. VARIABLE
This is the property of an object or event that can take on different values. For example, college major is a variable that takes on values like mathematics, computer science, English, psychology.

4.1 Discrete Variable has a limited number of values e.g. gender (male or female)

4.2 Continuous Variable can take on many different values anywhere between the lowest and highest points on the measurement scale.

4.3 Dependent Variable is that variable in which the researcher is interested, but is not under his/her control. It is observed and measured in response to the independent variable.

4.4 Independent Variable is a variable that is manipulated, measured, or selected by the researcher as an antecedent (precursor) condition to an observed behaviour. In a hypothesized cause-and-effect relationship, the independent variable is the cause and the dependent variable is the outcome or effect.

5. MEASURES OF CENTRE
Plotting data in a frequency distribution shows the general shape of the distribution and gives a general sense of how the numbers are bunched. Several statistics can be used to represent the “centre” of the distribution. These statistics are commonly referred to as measures of central tendency.
5.1 Mean (average) - is the most common measure of central tendency and refers to the average value of a group of numbers. Add up all the figures, divide by the number of values, and that is the average or mean. It is calculated from the formula \( \bar{x} = \frac{\sum x}{n} \). The sum all the scores in the distribution (\( \sum X \)) divided by the total number of scores (\( N \)). If you subtract each value in the distribution from the mean and then sum all of these deviation scores, the result will be zero (* see below). As one comic put it “Whenever I read statistical reports, I try to imagine the unfortunate Mr Average Person who has 0.66 children, 0.032 cars and 0.046 TVs”.

5.2 Median - is the score that divides the distribution into halves; half of the scores are above the median and half are below it when the data are arranged in numerical order. It is the central value, and can be useful if there is an extremely high or low value in a collection of values. The median is also referred to as the score at the 50th percentile in the distribution. The median location of \( N \) numbers can be found by the formula \( (N + 1) / 2 \). When \( N \) is an odd number, the formula yields an integer that represents the value in a numerically ordered distribution corresponding to the median location. (For example, in the distribution of numbers (3 5 8 9 9) the median location is (7 + 1) / 2 = 4. When applied to the ordered distribution (1 3 4 5 8 9 9), the value 5 is the median, three scores are above 5 and three are below 5. If there were only 6 values (1 3 4 5 8 9), the median location is (6 + 1) / 2 = 3.5. In this case the median is half-way between the 3rd and 4th scores (4 and 5) or 4.5.

5.3 Mode - is the most frequent or common score in the distribution, and is the point or value of \( X \) that corresponds to the highest point on the distribution. If the highest frequency is shared by more than one value, the distribution is said to be multimodal, and will be reflected by peaks at two different points in the distribution.

6. MEASURES OF SPREAD

Although the average value gives information about how scores are centred in the distribution, the mean, median, and mode do not help much when interpreting those statistics. Measures of variability provide information about the degree to which individual scores are clustered about, or deviate from the average value in a distribution.

6.1 Range is the difference between the highest and lowest score in a distribution. It is not often used as the sole measure of variability because it is based solely on the most extreme scores in the distribution and does not reflect the pattern of variation within a distribution.

a) Interquartile Range (IQR) provides a measure of the spread of the middle 50% of the scores. The IQR is defined as the 75th percentile - the 25th percentile. The interquartile range plays an important role in the graphical method known as the boxplot. The advantage of using the IQR is that it is easy to compute and extreme scores in the distribution have much less impact. However, it suffers as a measure of variability because it discards too much data. Nevertheless, researchers want to study variability while eliminating scores that are likely to be accidents. The boxplot allows for this for this distinction and is an important tool for exploring data.

6.2 Variance is a measure based on the deviations of individual scores from the mean. As noted in the definition of the mean (5.1 above), simply summing the deviations will result in a value of 0. To get around this problem the variance is based on squared deviations of scores about the mean. When the deviations are squared, the rank order and relative distance of scores in the distribution is preserved while negative values are eliminated. Then to control for the number of subjects in the distribution, the sum of the squared deviations is divided by \( n \) (population) or by \( n - 1 \) (sample). The formula for variance is thus \( s^2 = \frac{\sum (x - \bar{x})^2}{n-1} \). The result is the average of the sum of the squared deviations and it is called the variance.

6.3 Standard deviation provides insight into how much variation there is within a group of values. It measures the deviation (difference) from the group’s mean (average). The standard deviation (\( s \) or \( \sigma \)) is the positive square root of the variance. The variance is a measure in squared units and has little meaning with respect to the data. Thus, the standard deviation is a measure of variability expressed in the same units as the data. The standard deviation is very much like a mean or an “average” of these deviations. In a normal (symmetric and mound-shaped) distribution, about two-thirds of the scores fall between +1 and -1 standard deviations from the mean and the standard deviation is approximately 1/4 of the range in small samples (\( N < 30 \)) and 1/6 to 1/5 of the range in large samples (\( N > 100 \)). Standard deviation and variance are both measures of variability. The variance describes how much each value in the data set deviates from the mean (i.e. the spread of the responses), and is a squared value. The standard deviation also describes variability and is defined as the square root of the variance. This allows for a description of the variability in the same units as the data. A low SD will mean that the points of data are close to the mean, and a high SD indicates that the data is spread over a wide range of values. The SD is also used to describe the margin of error in the statistical analysis. This is usually twice the SD, typically described by the 95% confidence level. Confidence intervals consist of a range of values (interval) that act as good estimates of the unknown population parameter. After a sample is taken, the population parameter is either in the interval or not. The desired level of confidence is set by the researcher beforehand, for example 90%, 95%, 99%. If a corresponding hypothesis test is performed, the confidence level is the complement of the level of significance, i.e. a 95% confidence interval reflects a significance level of 0.05. Greater levels of variance yield larger confidence intervals, and hence less precise estimates of the parameter. Certain factors may affect the confidence interval size including size of sample, level of confidence, and population variability. A larger sample size normally will lead to a better estimate of the population parameter.

7. MEASURES OF SHAPE

For distributions summarizing data from continuous measurement scales, statistics can be used to describe how the distribution rises and drops.
7.1 **Symmetric** refers to distributions that have the same shape on both sides of the centre are called symmetric. A symmetric distribution with only one peak is referred to as a normal distribution.

7.2 **Skewness** refers to the degree of asymmetry in a distribution. Asymmetry often reflects extreme scores in a distribution.

   a) **Positively skewed** is when the distribution has a tail extending out to the right (larger numbers). In this case, the mean is greater than the median reflecting the fact that the mean is sensitive to each score in the distribution and is subject to large shifts when the sample is small and contains extreme scores.

   b) **Negatively skewed** is when the distribution has an extended tail pointing to the left (smaller numbers) and reflects bunching of numbers in the upper part of the distribution with fewer scores at the lower end of the measurement scale.

7.3 **Kurtosis** has a specific mathematical definition, but generally, it refers to how scores are concentrated in the centre of the distribution, the upper and lower tails (ends), and the shoulders (between the centre and tails) of a distribution.

8. **THE HYPOTHESIS**

A hypothesis is an assumption about an unknown fact. Donald Rumsfeld may have been trying to explain this when he said “We know there are known knowns; these are things we know we know. We also know there are known unknowns; that is to say we know there are some things we do not know. But there are also unknown unknowns – the ones we don’t know we don’t know.” Most studies explore the relationship between two variables, for example, that prenatal exposure to pesticides is associated with lower birth weight. This is called the alternative hypothesis. The null hypothesis (Ho) is the opposite of the stated hypothesis (i.e. there is no relationship in the data, or the treatment did not have any effect). Well-designed studies seek to disprove the Ho, in this case, that prenatal pesticide exposure is not associated with lower birth weight.

Tests of the results determine the probability of seeing such results if the Ho were true. The p-value indicates how unlikely this would be, or helps determine the amount of evidence needed to demonstrate that the results more than likely did not occur by chance. It describes the probability of observing results if the null hypothesis is true. A p-value of 0.05 indicates a 5% chance of obtaining that same result if there was no real effect of the experiment (a 1% chance that the null hypothesis is true). If the Ho can be rejected, then the test will be statistically ‘significant’ NB. Significant is a statistical term and does not mean important!

9.1 **Positive correlation** means that as one variable rises or falls, the other does as well (e.g. caloric intake and weight).

9.2 **Negative correlation** indicates that two variables move in opposite directions (e.g. vehicle speed and travel time).

9.3 **Causation** must not be confused with correlation. Causation is when a change in one variable alters another, but causation flows in only ONE direction. It is also known as cause and effect. E.g. Sunrise causes an increase in air temperature, in addition sunlight is positively correlated with increased temperature. However, the reverse is not true – increased temperature does not cause sunrise.

a) Regression analysis is a way to determine if there is or is not a correlation between two (or more) variables and how strong any correlation may be. It usually involves plotting data points on an X/Y axis, then looking for the average causal effect. This means looking at how the graph’s dots are distributed and establishing a trend line. Again, correlation is not necessarily causation. While causation is sometimes easy to prove, frequently it can often be difficult because of confounding variables (unknown factors that affect the two variables being studied). Again, once causation has been established, the factor that drives change (in the above example, sunlight) is the independent variable. The variable that is driven is the dependent variable (see point 4 above).

9. **CORRELATION**

This refers to the association between variables, particularly where they move together.

**CONCLUSIONS**

Understanding commonly used statistical terms should help clinicians decipher and understand research data analysis, and equip them with the knowledge needed to analyze results more critically. Perhaps then, the old adage of “All readers can read, but not all who can read are readers” will no longer be true of those reading the SADJ.

**References**

The police were summoned to investigate the foul smell coming from a bachelor flat in a high rise building in Braamfontein, Johannesburg. After forcing their way into the flat, they found the body of a severely decomposed male. It was assumed that the deceased was the owner of the flat, but due to the extensive decomposition a visual identification was not possible. The deceased’s children were all living aboard and were advised of the situation. From the state of the dentition, it was evident that the deceased had not visited a dentist for some time.

As no dental records could be found, other methods of identification had to be used. By chance a family member advised that she had a photo of her father. Generally “happy snaps” do not give sufficient detail to make a positive identification.

The following dental features were evident on examination of the dental status of the corpse (Figures 1 & 2):
1. Large diastema between the 11 and 21
2. 11, 12 and 13 present in the first quadrant
3. 21, 22 and 23 present in the second quadrant
4. 34 and 35 missing in the third quadrant
5. 34, 25 and 26 missing in second quadrant
6. Fractured amalgam filling in the 36

Noted the accumulation of maggots in the soft tissue.

Features visible in Figure 3 included:
1. Teeth 34 and 35 missing in the third quadrant
2. Filling visible in the 36

Figure 4 showed:
1. Large diastema present between front teeth
2. Teeth 21, 22 and 23 present
3. Teeth 24 and 25 missing

**DISCUSSION**

It is generally not possible to get a positive identification from social “happy snaps”. This case is unique in that not only do we see recognisable dental features in the front teeth, but there is a filled 36 which is visible in the photos provided. The presence of the diastema and the missing teeth allow us to match the general features present in the dentition of the victim with the general pattern present on the photo. The quality of the photos is such that small chips and minimal rotations would not be visible or provide sufficient detail to make a positive identification, but the general pattern association was sufficient for a positive match.

**CONCLUSION**

This case highlights the need to use whatever ante-mortem material is available to assist in the identification of otherwise unidentifiable corpses.

All names and places have been changed to protect the next of kin. Ethical approval has been granted by the Ethics Committee of the University of Pretoria to publish this case.

**References**


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The motivation to be ethical

Dentistry is indubitably regarded as an ethical profession, and one in which society has confidence will perform and behave in an ethical manner. Dental Ethics has been defined as:

• “The moral duty and obligation of a dentist towards his/her patients, professional colleagues and society.”

The FDI have adopted the International Principles of Ethics for the Dental Profession:

• The professional dentist will practice according to the art and science of dentistry and the principle of humanity.
• Will safeguard the oral health of patients irrespective of individual status.

The definitions are evocative and compelling but perhaps lack a little in precision, relying on broad concepts of “morality” and “humanity”. Indeed it was probably because the definition always was a mite ethereal that a dentist, himself impeccably ethical, was heard to remark. “Ethics is simply imbibed in your mother’s milk.” Could that be true or is there merit in the teaching of Ethics at our Dental Schools?

Medical and Dental ethics have embraced many of the concepts originally formulated by Immanuel Kant, the German philosopher who lived from 1724 to 1804. Kant held that it is a duty that will enable people to have the will to act in a morally correct manner. It is the motive for the action, not the consequences of the action, which determines whether the action is right or wrong. Hence it may be argued that good consequences may flow coincidentally from an action actually intended to cause hurt. Equally, bad results may be incurred by an otherwise well intended action. Kant then suggests that it is a good will that is the fundamental good. and that is in fact only good when an action is chosen because it is accepted as a duty. Such respect for duty surely provides a foundation for all ethics and indeed there is a term recognising this interpretation of Ethics, Deontological Ethics. Kant did not suggest the term himself for it was first used in 1930 by CD Broad in his book Five types of Ethical Theory. “Deon” comes from the Greek word ἀξίω meaning obligation or duty. The meaning of the term has been best retained in French, code de déontologie, ethical code, especially applied to professional ethics.

The application is readily discernible, professionals have a duty to behave morally towards their patients. Kant suggested that the ability to reason is the basis of morality and therefore himself reasoned that all persons, being rational beings, have the right to common dignity and respect. These are fundamental tenets in our modern Ethical Code. Kant expressed the philosophy in more abstruse terms: “Act only according to that maxim by which you can at the same time will that it should become a universal law.” For example, a lecturer who used students in a research project without their knowledge must then be prepared to allow ALL researchers the same latitude, an obviously undesirable situation.

Kant went further in his exploration of philosophical questions, and considered the concept of autonomy. In fact many believe that the basis of Kant’s theories on morality is the idea of autonomy. The dictionary definition of autonomy may be “independent, self governing”, but Kant understood that this is not freedom per se, that is, being restrained by no laws, but rather a freedom bounded by laws that are largely of one’s own making. So the autonomy originates in the freedom to make those laws and actions express the will of the individual, not the will of someone else.

In Dentistry one of our essential requirements is to ensure that the patient is informed and that Consent to Treatment is based on proper disclosure. But if we apply that unreservedly, we are delegating to the patient the treatment decision. It is a situation of reductio ad absurdum, how can a professional allow the management of the case be determined by an untrained patient?

Also recognised is the dilemma of when is it permissible to steal, perhaps when the alternative is starvation? Under what circumstances may it be considered right to kill? When you are defending yourself from attack? Kant’s Categorical Imperative had three major formulations, which may conveniently be summarised in a most simplistic interpretation: Do unto others as you would wish them to do unto you.

Kant insisted on the requirement that to be morally good, the laws of behaviour had to be applicable universally. To act in a morally right manner, actions must be determined on the basis of duty. Does that help in our applying autonomy in treatment decisions? Certainly, because although patient and dentist may be coming from different directions, it will be duty that will decide, for the patient, a duty to seek the best possible treatment under the circumstances; for the dentist, as set out by the American Dental Association, his or her moral obligations and duties must include involving patients in treatment decisions in a meaningful way, with due consideration being given to the patient’s needs, desires and abilities and safeguarding the privacy of the patient. (Of course the dentist retains the autonomy to ensure that he or she will continue to make a living!)

Ethics will always raise questions, ethics may on occasion be controversial, but there are clear principles underlying the ethos. While not all agree with his concepts, nevertheless Kant went a long way to guide the principles of professional ethics. For all these reasons Dental Ethics must retain an important role in Dental Curricula. Bebeau and Thoma undertook an exploration on how a course in Ethics affected the moral reasoning of a sample (n=720) of dental students. The conclusion was that not only did the students benefit from the course but also valued it.

Mother’s milk it may be.. it is certainly food for thought!
Maxillo-facial radiology case 142

A 6-year old boy presented to the hospital with a five-month history of a persistent, draining sore (Figs.1 & 2)) in the mandible on the left side. It all started when a carious 36 was extracted. Figs. 3, 4, 5 & 6 are images of other patients with the same condition. What is your diagnosis?

INTERPRETATION

The cropped pantomograph (Fig 2) shows a ‘blurred/ fussy” appearance of the trabeculae due to loss of density. Signs of cortical destruction are also present. A provisional diagnosis of acute osteomyelitis was made which was confirmed histologically. Osteomyelitis is an inflammation of bone cortex and marrow that develops in the jaw usually after a chronic infection. No detectable radiographic features of the infection are discernible in the first 8-10 days (radiological principle: 30-60% decalcification before any changes are observed). About three weeks after the start of the infection irregular radiolucencies with enlargement of trabecular spaces are noticed (Fig 3). In the chronic stages, extensive areas of the bone may be destroyed and sequestra are formed showing the classic moth-eaten appearance and occasionally a pathological fracture may also be present (Fig. 4). Note the sequestration of bone in the anterior mandible on the coronal CT scan (Fig. 5) of another patient with chronic supplicative osteomyelitis The coronal T2 W. MRI scan (Fig. 6) shows the sequestrated bone (no signal black) is surrounded by zones of high signal intensity (white signal) representing the regions of active inflammation. Most cases of osteomyelitis of the jaw result from odontogenic infections, infections from a fracture site; or, rarely, haematogenous spread from a distant site, which is more common in the long bones. The cause of infection is most frequently Staphylococcus aureus, but haemolytic streptococci and Bacteroides are also encountered. The mandible is by far the most frequently affected; the adult maxilla is very seldom the site of osteomyelitis. The acute phase of suppurative osteomyelitis is sudden in onset and rapid in course. The patient experiences severe pain, fever, regional lymphadenopathy, and leucocytosis. Teeth in the region become loose and tender to percussion. When the mandible is involved there may be paraesthesia of the lower lip on the affected side. As soon as drainage is established, the pain eases, the temperature drops, the patient becomes more comfortable. Without treatment the infection may progress into a protracted chronic supplicative osteomyelitis. Differential diagnoses include malignancies and osteoradionecrosis.

Reference

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Several clinical studies have established the relationship between diabetes and periodontitis. This relationship appears to be bidirectional, with diabetes being a risk factor for periodontitis whilst the severity of periodontitis is a factor influencing glycemic control and the development of complications in diabetic patients. In addition, periodontal treatment may have a positive effect on glycemic control in diabetic patients.

The clinical benefit of nonsurgical periodontal treatment is well documented. There is evidence that the use of antibiotics with nonsurgical periodontal therapy provides some benefit to systemically healthy patients, but their use is generally recommended only in specific clinical situations. Diabetes mellitus (DM) is recognized as a major risk factor for periodontal diseases, as patients with DM present increased prevalence and severity of periodontal destruction compared with those without DM.

There is good evidence indicating that the clinical benefits observed in systemically healthy subjects with chronic and aggressive periodontitis who are treated with adjunctive metronidazole (MTZ) and amoxicillin (AMX) are accompanied by a beneficial change in the composition of the subgingival biofilm. However, no studies to date have comprehensively evaluated the changes occurring in the subgingival microbial profile in subjects with DM receiving MTZ, AMX, and undergoing scaling and root planing (SRP).

Tamashiro and colleagues (2016) reported on a trial that sought to assess the changes occurring in the levels and proportions of oral bacteria in subjects with periodontitis and type 2 DM treated by means of SRP only or combined with systemic MTZ and AMX. A secondary aim was to compare the clinical efficacy of these two treatment protocols two years later.

**MATERIALS AND METHODS**

Adult patients with type 2 diabetes and generalized chronic periodontitis who met the following inclusion criteria were invited to participate in this trial: aged ≥35 y, diagnosis of type 2 DM during at least the past five years, glycated hemoglobin levels ≥6.5% to ≤11%, ≥15 teeth, >30% of the sites with pocket depth (PD) and clinical attachment level (CAL) ≥4 mm, and ≥6 teeth with at least one site with PD and CAL ≥5 mm and bleeding on probing (BoP). Exclusion criteria were as follows: pregnancy, lactation, smoking, SRP in the previous 12 months, systemic antibiotic treatment in the previous six months, need of antibiotic prophylaxis, systemic conditions (except DM) that could affect the progression of periodontitis, long-term use of anti-inflammatory or immunosuppressive medications, and allergy to MTZ and/or AMX. Subjects were informed of the nature, potential risks, and benefits of the study and signed a form of informed consent.

In this double-blinded, parallel-design, placebo-controlled randomized clinical trial (RCT), patients were randomly allocated subjects into one of the following groups: SRP + placebo (control; n = 29) or SRP + MTZ (400 mg thrice a day (tds) for 14 days) + AMX (500 mg tds for 14 days) (test; n = 29). Allocation concealment was ensured by means of sequentially numbered drug
containers of identical appearance. Subjects in the control group received two placebo pills tds for 14 days. Antibiotic/placebo administration started at the day of the first SRP session.

Initially, all subjects received supragingival plaque control and oral hygiene instructions. Two trained periodontists performed SRP in four to six appointments lasting approximately one hour each, using manual curettes and an ultrasonic device. An overall full-mouth SRP was performed during the first treatment visit to disrupt the subgingival biofilm and maximize the antibiotic effect from the beginning. Subsequently, one quadrant or sextant was treated per SRP session, depending on the number of deep pockets. Periodontal therapy was completed in 14 days. The clinicians and all participants were blinded to treatment assignment. All subjects received microbiological and clinical monitoring at baseline and three months, one, and two years post-therapy. Clinical measurements were also performed at six months. Periodontal maintenance was conducted at three, six, and nine months and one year and two years post-therapy and included oral hygiene instructions and supragingival/subgingival biofilm/calculus removal, as necessary.

An assistant monitored the compliance with antibiotic/placebo intake by calling the patients three times a week during the 14 days of medication. The subjects were asked to bring the empty bottles back at the end of each week, and these were checked for any possible remaining pills of antibiotics/placebos. On the fourteenth day, subjects answered a questionnaire about any self-perceived side effects of the medications.

A single calibrated examiner performed all clinical examinations. Presence or absence of plaque, marginal bleeding, BoP, suppuration, and PD and CAL measurements were assessed at six sites per tooth excluding third molars using the manual periodontal probe (North Carolina–Hu-Friedy). The examiner was blinded to the treatment allocation of the subjects.

After supragingival plaque removal, the subgingival biofilm samples were collected with individual sterile mini-Gracey curettes (#11–12) from six noncontiguous interproximal sites, two at each of the following baseline PD categories: shallow, PD ≤3 mm; intermediate, PD = 4 to 6 mm; and deep, PD ≥7 mm. These were evaluated for 40 bacterial species.

The clinical and microbiological data were evaluated using intention-to-treat analysis with last observation carried forward, and the level of significance was set at 5%.

RESULTS
Fifty-eight subjects were randomly assigned to receive SRP only (n = 29) or with MTZ (400 mg tds) and AMX (500 mg tds) (n = 29) for 14 days. Six subgingival plaque samples/subject were analyzed by checkerboard DNA–DNA hybridization for 40 bacterial species at baseline and three months, one year, and two years post-therapy. Ten patients in the control and 13 in the test groups were lost to follow-up between year one and year two.

Both treatments led to a significant reduction in the proportion of the red complex pathogens at three months (SRP: from 16.3% to 7.6%; SRP + MTZ + AMX: from 17.8% to 5.3%) (P < 0.05). The proportions of red complex pathogens were maintained up to two years in the antibiotic-treated group (5.5%) but increased to 9.8% at one year and to 12.1% at two years in the control group. The difference between groups for the proportions of this complex at two years was statistically significant (primary outcome).

Subjects with SRP-only treatment showed a significant reduction in the mean levels of Tanneraella forsythia and Porphyromonas gingivalis (P < 0.05), while the levels of nine species were altered in the test group, including a reduction in the three red complex pathogens (T. forsythia, P. gingivalis, and Treponema denticola). The reduction in the levels of T. forsythia and P. gingivalis from baseline to two years posttreatment was greater in the test than in the control group (P < 0.05).

No statistically significant differences were observed between groups for the demographic, glycemic, and clinical parameters at baseline or for the number of adverse events reported.

The percentage of sites with BoP and suppuration and full-mouth mean PD were significantly lower in the test group at one year and two years (P < 0.05). At one year, the antibiotic group had significantly fewer sites with PD ≥5 mm (primary outcome variable) than the control group, and this benefit was maintained up to two years (SRP = 14.7 ± 13.1, SRP + MTZ + AMX = 3.5 ± 3.4, P < 0.05); 75.8% of the subjects treated by SRP + MTZ + AMX and 22.3% who had SRP-only treatment were at low risk at two years. The antibiotic-treated group showed a greater reduction in mean PD and gain in mean clinical attachment at initially moderate and deep sites (P < 0.05) than the control group at one and two years posttreatment.

Stepwise forward logistic regression analysis showed that, of all predictor variables included in the model, the treatment with MTZ + AMX was the only variable that significantly increased the probability of a subject reaching the low risk profile for future disease progression and not having any site with PD ≥6 mm (odds ratio [OR], 14.3; P = 0.0000) at the two-year posttreatment (OR, 20.9; P = 0.0000).

CONCLUSION
The researchers concluded that the adjunctive use of MTZ + AMX in the active phase of periodontal treatment improved the microbiological and clinical outcomes of SRP in subjects with generalized chronic periodontitis and type 2 DM, up to two years post-treatment.

IMPLICATIONS FOR PRACTICE
Type 2 diabetes is a major public health problem in South Africa. This trial has conclusively shown the additional benefits of metronidazole and amoxicillin as adjuncts in the non-surgical management of patients with type 2 diabetes.

References
Dental implant therapy is widely accepted by patients and dentists as a reliable method for oral rehabilitation. When bone volume is not sufficient for a standard implant installation, different solutions are available to augment bone volume: these include onlay and inlay bone grafts, maxillary sinus elevation, guided bone regeneration, edentulous ridge expansion, or distraction osteogenesis, all of which involve prolonged healing time, higher morbidity, and higher costs. Alternatively shorter implants have been introduced for use, especially in cases with limited vertical bone dimension.

The use of short implants, however, may implicate the risk of increased load on the peri-implant bone, potentially resulting in enhanced loss of marginal bone or even in premature implant loss. However, whether a higher crown-to-implant ratio may lead to a higher degree of occlusal load, resulting in a negative influence on successfully osseointegrated implants, remains controversial.

A considerable number of clinical studies assessed implant survival rates as well as marginal bone-level changes for short implants when loaded with single crowns but these are based on data over short time periods only. Sahrmann and colleagues (2016) reported on a randomized controlled clinical two-centre trial that sought to assess survival and marginal bone loss of 6-mm and 10-mm implants supporting single crowns in the posterior jaws. The null hypothesis was that implants of both lengths would perform similarly with regard to survival and change in marginal bone level.

MATERIALS AND METHODS

This RCT considered systemically healthy patients who met the following inclusion criteria: patients had to present with a single-tooth gap in the premolar or molar region of the upper or lower jaw and an existing antagonist (tooth or implant-borne reconstruction). The missing tooth had to have been extracted at least six months prior to implant placement. No periodontal probing depths (PPDs) exceeding 5mm in the residual dentition were accepted. A minimum of 2mm of keratinized mucosa had to be present at the prospective implant site. Regarding bone dimensions, a minimal vertical bone height of 10mm in the lower jaw (alveolar crest to the mandibular canal) and 6mm of bone height in the maxilla (alveolar crest to the sinus floor) was required. Internal sinus floor augmentation (modified Summer’s technique) but no lateral guided bone augmentation procedures were allowed when placing the implants.

Exclusion criteria comprised general contraindications against surgical interventions and smoking of more than 19 cigarettes per day. The need for a preceding lateral bone augmentation with radio-opaque filler materials, prior therapeutic radiation of the jaw, severe bruxism or clenching habits, and any mucosal disease except sporadic localized gingivitis were further exclusion criteria. Insufficient oral hygiene and inadequate compliance were additional reasons for exclusion.

Implant placement was performed at two clinics by calibrated surgeons who were well trained with the implant system. The randomization of the patients to either the test (6-mm implant) or control group (10-mm implant) was determined using a computer-generated randomization list. After administration of a local anesthetic, sulcular incisions at the adjacent teeth and a midcrestal incision were performed, allowing a full-thickness flap to be raised. At this stage, the randomization concealment was broken and the surgical site was prepared according to the manufacturer’s instructions (SLActive standard plus soft tissue level implants; Straumann). The minimum primary stability had to reach 20 Ncm. All implants were covered with a healing cap. Flaps were closed with nonresorbable sutures, leaving the implants for transmucosal healing. Patients had to refrain from brushing at the surgical site and instead had to rinse with a 0.2% chlorhexidine solution for one minute twice a day until suture removal. Analgesics were provided for optional intake during the first postoperative days. After a healing period of six to ten days, sutures were removed. Three weeks later, oral hygiene was monitored, instructions for site-specific hygiene were repeated, and supragingival tooth cleansing was performed. Eight weeks after implant placement, impressions were taken using a standardized tray and a polyether impression material. The impression of the opposite jaw was taken with alginate. No provisional restorations were inserted. Screw-retained porcelain fused to metal (FFM) crowns were incorporated with a torque of 35 Ncm. After insertion of the reconstruction, a clinical examination (baseline) was performed measuring peri-implant and periodontal probing pocket depths, presence or absence of plaque, and bleeding on probing at six sites per implant and the neighbouring teeth. In addition, a standardized x-ray film was taken.

After six months, oral hygiene was controlled and reinstructed if needed. Thereafter, patients were recalled at regular intervals between six and 12 months for dental hygiene treatments according to their individual needs. At one year of loading and once every year thereafter, patients underwent a clinical examination of the study implant and the neighbouring teeth. These appointments were conducted by one examiner per clinic and included measurements of peri-implant and periodontal probing pocket depths, presence or absence of plaque, and bleeding on probing at six sites per implant and at the adjacent teeth. At these follow-up appointments, technical failures such as chippings or loosening of abutment
screws were recorded. In addition, intraoral photographs were taken as well as a standardised x-ray film positioned when applying the parallel technique.

History of periodontitis was determined as general attachment loss exceeding 5mm at more than 30% of the periodontal sites or tooth loss due to periodontitis.

Digitalized x-ray images of all implants were magnified 10-fold and size-calibrated by their known length, width, and interthread distance. Mesial and distal bone levels as well as the crown lengths were determined. Clinical lengths of crowns and implants were calculated by adding the supra-osseous part of the implant (composed of 1.8mm of machined implant neck and potential bone-level changes from the nominal bone level at the margin of rough and machined implant neck of the standard plus implant type) to the measured (technical) crown length and subtracting that distance from the length of the whole implant.

All measurements were performed by two independent examiners who had previously been calibrated. Statistical analyses were performed with the average values of the measurements recorded by both examiners'.

RESULTS
Initially, 96 patients could be included in the study. Two patients of the control group, however, did not receive the complete treatment according to the study, thus were excluded from further assessment. At three years of loading, 81 patients could be reassessed, while 13 patients did not show up for the appointments, skipped their recall due to personal reasons, or had moved abroad in the meantime. Of the remaining patients, 78 had x-ray films which could be analyzed.

All patients were in good general health at the follow-up appointments. One implant from the test group became mobile during the second year of loading without any radiographically detectable marginal bone-level change and had to be removed. All implants from the control group were still in place at the three year follow-up. This resulted in an overall survival rate of 98% for test and 100% for control implants. This difference was not statistically significant. No implant displayed peri-implantitis in terms of pocket depths >5 mm in combination with suppuration and/or progressive marginal bone loss. The mean crown-to-implant ratio in the test group (1.48 ± 0.33) was significantly higher (P < 0.001) than in the control (0.86 ± 0.18).

Over three years, the marginal bone-level changed by −0.19 ± 0.62 mm (test) and −0.33 ± 0.71 mm (control). These values for the bone levels at baseline and at three years showed no statistically significant difference for each group. No significant intergroup difference was found at three years.

A significantly higher number of implants with PPD of ≥5 mm was found in the test group (P = 0.023). These probing depths, however, had already been observed during the baseline examination and showed neither progression nor suppuration at any later time point. Regression analysis of the changes of the marginal bone level at the three year follow-up showed a nonsignificant effect of implant length (estimated effect 0.38 for more bone loss for the long implants with P = 0.152) when adjusting for the set of potential confounders (smoking, history of periodontitis, bone level at baseline, crown-to-implant ratio). With decreased initial bone level at baseline, regression analysis showed a distinct effect on future bone loss. No chipping of the veneering ceramic occurred and loosening of the abutment screw happened in three cases.

CONCLUSION
The researchers concluded that this randomized controlled trial found no difference between test and control implants supporting single crowns in the posterior jaw at three years with regard to the primary outcome parameters of survival and change in the marginal bone level. Technical complication rate was low, measuring 3.8%, whereas no biological complications were observed.

IMPLICATIONS FOR PRACTICE
The trial supports the use of shorter implants (6mm) for use for single tooth restoration which has the added benefit of reducing the invasiveness of implant surgery. Additionally, these could mean decreased patient morbidity, shorter surgical treatment time, and a minimized risk of damaging neighboring anatomical structures.

Reference
CPD Questionnaire

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

GENERAL

Cancrum Oris (noma) in an HIV-positive adult. (p 248)
1. In medical usage, the term ‘noma’ refers to a “quickly spreading sore”.
   a. True
   b. False

2. Malnutrition can affect the oral mucosa and the gingiva, leading to an increased permeability of the tissue membranes and susceptibility to infection.
   a. True
   b. False

3. Necrotizing stomatitis can rapidly lead to perforation of the facial tissues and skin, exposing teeth and bone.
   a. True
   b. False

Oral diseases associated with human herpes viruses: aetiology, clinical features, diagnosis and management (p 253)
4. Identify the incorrect statement. Herpes simplex viruses cause:
   a. primary herpetic gingivostomatitis
   b. recurrent herpes labialis
   c. Vincents necrotising stomatitis
   d. recurrent intraoral ulcers

5. The most frequently prescribed first-line antiviral agent is acyclovir.
   a. True
   b. False

6. Identify the incorrect statement. The decision to prescribe antiviral drugs is determined by:
   a. the severity, extent and duration of the disease
   b. the occurrence of spontaneous epistaxis
   c. the age of the patient
   d. the fitness of the immune system as deduced from the history
   e. the potential side effects of the drugs

Knowledge and beliefs about oral pseudomembranous candidiasis among traditional health practitioners in Limpopo Province, South Africa. (p 260)
7. Prior to the introduction of antiretroviral therapy, oral pseudomembranous candidiasis occurred in up to 90% of HIV positive patients at some point during the course of the disease.
   a. True
   b. False

8. Forty percent of the traditional Health Practitioners were confident they knew the cause of oral pseudomembranous candidiasis.
   a. True
   b. False

Management of an inflamed dentigerous cyst in a patient with an anterior cross bite, using a modified bite plane (p 266)
9. The tooth most commonly affected by dentigerous cyst development is:
   a. Maxillary third molar
   b. Mandibular third molar
   c. Maxillary canine
   d. Mandibular premolar

10. The most common diagnostic sign of dentigerous cyst is:
    a. Unerupted tooth
    b. Pus in the gingiva
    c. Pain in the tooth
    d. Swelling in the jaw

11. Dentigerous cyst can be treated by
    a. Marsupialisation
    b. Enucleation
    c. Both of the above
    d. Radical resection

Are fissure sealants still relevant as a caries preventive measure? (p 272)
12. Fissure sealants have been an effective tool in aiding the prevention of fissure caries.
    a. True
    b. False

13. Fissure sealants can be retained for up to 48 months.
    a. True
    b. False

The meaning of the MEAN, and other statistical terms commonly used in medical research (p 274)
14. Prevalence and Incidence are interchangeable terms in statistics.
    a. True
    b. False

15. In calculating Variance the deviations of scores about the mean are squared to eliminate negative values.
    a. True
    b. False
16. It is possible to make a positive forensic match on the evidence of the general patterns of a dentition.
   a. True
   b. False

Clinical Windows (p 282)

17. In the Tamashiro et al trial, both treatments led to a significant reduction in the proportion of the red complex pathogens at 3 months.
   a. True
   b. False

18. In the Tamashiro et al trial, the antibiotic-treated group showed no reduction in mean PD and gain in mean clinical attachment.
   a. True
   b. False

19. In the Sahrmann et al trial, the implants were immediately loaded upon insertion.
   a. True
   b. False

20. In the Sahrmann et al trial, the mean crown-to-implant ratio in the test group was significantly higher than in the control.
   a. True
   b. False

ETHICAL

The motivation to be ethical (p 280)

21. Dental Ethics may be defined as "the moral obligations and duties of a dentist towards the profession."
   a. True
   b. False

22. The sense in which Kant uses the word ‘will’ is the power of conscious, deliberate action or choice.
   a. True
   b. False

23. Deontological Ethics is based on:
   a. a concept of odontological behaviour
   b. purely religious concepts
   c. conforming to morally accepted duty
   d. a determination of practicality

24. Autonomy in the view of Kant is characterised not by freedom per se but by the freedom to formulate moral laws which could be of universal applicability.
   a. True
   b. False

25. The dentist has a moral and ethical duty to ensure the patient has sufficient information to decide on the Consent to Treatment.
   a. True
   b. False

Readers will note that we have reduced the number of General Questions to twenty whilst retaining five Ethics based questions. Our allocation of CPD points remains unchanged. There is optimism that this section will continue to provide members with a valuable source of CPD points whilst also achieving the objective of CPD, to assure Continuing Education. Please note that SADA is no longer offering the ‘CPD via SMS’ service.

Contact Ann Bayman at SADA, Tel: 011 484 5288, for any enquiries and assistance.

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- All smalls advertisements are charged at R25.00 per word, with a minimum charge per placement of R250.00 for Public Domain advertising irrespective of SADA Membership standing.

**Members ONLY Platform**
Smalls advertising available to registered Dental Healthcare Practitioners

- SADA Members have free placement of smalls advertisements meeting Advertising Rule criteria within the Members ONLY Platform.

**Smalls Advertising Rules**

- All smalls advertisements are restricted to a maximum 100 words per advertisement.
- All advertisement requests are required in writing with full contact details of the advertiser which should include:
  - the wording of the advertisement as you require it to be published;
  - the members professional number; (will not be published);
  - the members contact details (will not be published).
- Advertisement lifespan is two weeks from the date of upload.
- Advertisements to be repeated follow the same process as the original placement request.
- All advertisements which exceed a word count of 100 words will be forwarded to our publishers E-Doc for further processing as a potential advertisement to be placed in the SADJ electronically or as website advertising. E-Doc will contact you thereafter regarding your requirements.
- Advertisement must be paid in full prior to uploading on the web platform for Public Domain advertising.
- Invoice may be settled telephonically with the use of a credit card to prevent delay of placement.
- Telephonically processed payments will result in uploading of advertisement within 24 hours of settlement.
- Advertiser remains liable for placement costs should payment be dishonoured and invoice remains unpaid.

**Contact details:**
Ann Bayman
South African Dental Association
Tel: 011 484 5288
Fax to email: 086 683 0392
e-mail: ABayman@sada.co.za
or via fax to 086 683 0392

**SADA Contact Numbers:**

**MEMBERSHIP**
Adjustment / Application / Contact detail change / General enquiry / Renewal

All membership enquiries should be channelled through your allocated MRO (Member Relations Officer) who will direct your enquiry accordingly should they not be able to assist you.

**Branch:** Algoa Midlands
**MRO:** Nelisa Makubalo
**Email:** NMakubalo@sada.co.za
**Fax:** 086 758 9889

**Branch:** Border Kei
**MRO:** Nelisa Makubalo
**Email:** NMakubalo@sada.co.za
**Fax:** 086 758 9889

**Branch:** Free State
**MRO:** Joseph Moalusi
**Email:** JMoalusi@sada.co.za
**Fax:** 086 743 1309

**Branch:** Gauteng South
**MRO:** Sylinda Bayman
**Email:** Sylinda@sada.co.za
**Fax:** 086 688 5799

**Branch:** KwaZulu Natal
**MRO:** Nelisa Makubalo
**Email:** NMakubalo@sada.co.za
**Fax:** 086 758 9889

**Branch:** Limpopo
**MRO:** Anna Tsumane
**Email:** ATsumane@sada.co.za
**Fax:** 086 644 2411

**Branch:** Mpumalanga
**MRO:** Anna Tsumane
**Email:** ATsumane@sada.co.za
**Fax:** 086 644 2411

**Branch:** Pretoria
**MRO:** Anna Tsumane
**Email:** ATsumane@sada.co.za
**Fax:** 086 644 2411

**Branch:** North West
**MRO:** Anna Tsumane
**Email:** ATsumane@sada.co.za
**Fax:** 086 644 2411

**Branch:** Northern Cape
**MRO:** Joseph Moalusi
**Email:** JMoalusi@sada.co.za
**Fax:** 086 743 1309

**Branch:** Western Cape
**MRO:** Joseph Moalusi
**Email:** JMoalusi@sada.co.za
**Fax:** 086 743 1309

**Branch:** DPL Only Member
**MRO:** Nelisa Makubalo
**Email:** NMakubalo@sada.co.za
**Fax:** 086 758 9889

**Branch:** Affiliate (Non Branch Member)
**MRO:** Anna Tsumane
**Email:** ATsumane@sada.co.za
**Fax:** 086 644 2411

If you are not currently a member of SADA/DPL and would like to apply for SADA membership please speak to the MRO relevant to your provincial area.

**Continuing Professional Development**
If your enquiry is related to a CPD Accreditation Application or CPD Event, please forward your enquiry to CPD@sada.co.za
Join the fight against human trafficking and help MCSA to bring children home safely.

SMS MCSA to 41006 to donate R25 today!*  

OTHER WAYS TO GET INVOLVED:

ADOPT A SALARY  DIRECT DEPOSIT  MYVILLAGE CARD  VOLUNTEER OR DONATE

Office: 021 950 1546 • E-mail: info@missingchildren.org.za
Emergency: 072 647 7464 (072 MISSING) • www.missingchildren.org.za

Missing Children SA (Page)  @072missing

SWIPE OR GET YOUR FREE MYSHOOL CARD AND HELP RAISE FUNDS FOR MISSING CHILDREN SOUTH AFRICA (MCSA) TO HELP FIGHT AGAINST HUMAN TRAFFICKING, AND IT WON’T COST YOU A CENT. ADD MCSA AS 1 OF YOUR 3 BENEFICIARIES OR CHANGE 1 OF YOUR 3 BENEFICIARIES TO MCSA.

www.myschool.co.za | www.missingchildren.org.za/page/myschool-card

* A premium rate of R25 applies and free sms’s do not apply. This is a donation to Missing Children South Africa which is a non-profit organisation (NPO 067 095)