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23 Third molar impaction in a cross section of adult orthodontic patients
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The teeth of the Dusky Shark (Carcharinus obscurus) are carried forward on a “conveyor belt” system, are used in function, then discarded over the edge of the jaw, to be replaced by the next row of teeth advancing along the conveyor belt.
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Turning denticles inside-out.

Two thoughts at the beginning of this new year… firstly a Handsel to you all, that is an expression of good wishes, a word dating from the 13th Century and in Scotland used to describe Handsel Monday - the first Monday of a New Year. and apparently that was the ONLY holiday of the year for the industrious Scots! But at least they received good wishes too.

So a sincere Handsel to start the year!

Secondly, an intriguing paradoxical enquiry on the evolution of teeth… the Inside-Out or Outside-In dilemma. Brought to mind by consideration of a paper in this issue which in part explores some of the extraordinarily complex dentition of shark teeth. The question is raised as to whether those shark teeth point towards the evolutionary path leading to mammalian dentition, including of course Homo sapiens.

The development of jaws and teeth made a major contribution to the evolutionary success of vertebrates, a fact well accepted and understood. However, considerable controversy perseveres regarding just how teeth actually evolved. The shark could perhaps offer a clue! Amongst its fearsome armaments, this predator has an external integument fully equipped with structures which tear the skins of prospective prey, inducing copious bleeding which then stimulates the shark to attack in earnest. Those integumental structures are denticles, named for their close resemblance to teeth—or is it that the teeth bear a close resemblance to denticles???

That is the essence of the Inside-Out/Outside-In controversy. Did teeth develop as a result of the migration of denticles, (or denticle like structures), INTO the mouth. or did teeth develop intraorally as new structures independently? There is certainly circumstantial evidence that teeth and denticles (or scales) share structure and organogenesis and that phylogenetically in vertebrates denticles appeared earlier than teeth. All support for the Outside-In theory. The Inside-Out theory has depended upon evidence that endoderm is required for dental development, that tooth -like structures evolved before denticles appeared and that teeth may have evolved on multiple occasions. Now there are opposing claims, for more recent work has shown that teeth may develop from dermal, endodermal or mixed epithelia. In addition, the pharyngeal “teeth” seen in some fish evolved separately from true teeth. Strong opinions are expressed that “odontodes” were expressed first in the dermal skeleton and that their distribution extended internally through the oral cavity… and nasal and pharyngeal orifices. In other words, the capacity to develop teeth was transferred from the external dermis to the internal epithelia. Discard the Inside-Out and embrace the Outside-In!! In the long run, the development of internal odontodes, or teeth, and the development of external odontodes or denticle scales, have probably become distinct and separate modules in modern vertebrates.

So while we may view with shuddering horror the bristling teeth in the jaws of the shark, we realise that in fact we are looking at evidence of how we have teeth… and of how our patients have teeth -without which we could not practice!

Exploring a little further the Inside-Out and Outside-In theories it is intriguing to know that explanations of evolution in Biology and in the Galaxy have invoked the theories. Did the eukaryotic cell enjoy an evolution of the nucleus and endomembrane system within the cytoplasm of a prokaryotic cell. OR, did the cell extrude membrane arms to encircle ectosymbiotic proto mitochondria and in this way enclose the future cell contents? That would fit the Inside-Out theory.

The growth of the galaxy is being further elucidated by the application of the Inside-Out theory in which bursts of star formation at the centre may be followed, billions of years later, by star birth at the outer edges.

Would it not be wondrous if there is indeed similarity in the manners in which minute cells develop and the limitless galaxy expands?
Evolution is a continuous process... and that continuity is reflected in the Journal for we are scheduled this year to evolve by expanding to some 60 pages of content. The front cover pictures will illustrate aspects of the development of the hominid dentition. The Journal will of course remain an essentially scientific publication but especially now with expanded pages we shall warmly welcome additional clinical content. Reports of clinical cases, observations of clinical presentations, summaries of lectures and seminars will all be considered for publication.

A Handsel is also a gift or token for good luck...and the Journal hopes to extend such a Handsel of stimulating reading to readers, and is confident that the Journal in turn will receive the Handsel of continued papers from authors.

References
The financial compensation of SA dentists just doesn’t compare.

As we begin the New Year of 2018, I am alert to one of the matters that have been raised by many dentists in my meetings across South Africa…the fact that dentists are expected to offer an increased quality of service …but within the ambit of an ever reducing remuneration-for-service regime. Many believe that dentists in South Africa earn exorbitant amounts of money, more than most other medical doctors and specialists. However, while the public may perceive this to be the case - largely because the allocations to dentistry made by the medical aid schemes leave members having to pay towards their oral health care out of their own pockets - it couldn’t be further from the truth.

The reality is that most dentists in South Africa, despite spending at least five years learning the basics of the discipline, earn less than other medical professionals and far less than their international counterparts.

According to the international online salary, benefits and compensation information company, Pay Scale, the average salary for a South African Dentist is R426,304 per year – about R35,500 per month. This is notably less than the average annual salaries of Orthopaedic Surgeons (R1,076,000), Radiologists (R589,581), General Surgeons (R550,000), Anaesthetists (R480,000), and General Practitioners (R474,574).

The Health Professions Council of South Africa (HPCSA) recognises 30 specialties and 18 subspecialties, in the health-care field. Within these specialties, it is only the dentists who are required to have all the latest, specialist equipment available at their individual practices, equipment which comes in at a massive cost. Within other medical fields comparable equipment needs are limited to the larger and more advanced facilities, usually hospitals. It is generally much more expensive to set up a private dental practice than for any other medical discipline.

While our dentistry professionals refuse to use inferior equipment, or to drop the standard of their services, the cost that goes into pur- chasing and maintaining this kind of technology is making it increasingly difficult for dental practices to make a reasonable profit. This is further compounded by the pressure put on the discipline by the lack of support from private medical aid companies in their annual allocations, and from the public sector in not investing adequately in educating the public on the high importance of oral healthcare in maintaining optimal overall health.

Career Junction’s list of the top paying positions in South Africa in 2017 showed that the highest earning jobs in the country are, in fact, in the engineering sector. This is in stark contrast to the global top 10 highest-paying jobs which, according to Investopedia, is dominated by healthcare occupations – including dentists (number five on the list). To the questionable imbalance of remuneration is added the realisation that certain sectors and professions within the national healthcare industry are being prioritised over others. How different are the priorities of our country to those of the rest of the world!

Should we not be placing more value on the health of our nation? More specifically, should we not be questioning why oral healthcare – a crucial element for our overall health – is being pushed to the bottom of the essential needs list?

This is compounded by the fact the provision of dental cover continues to be eroded despite increases in medical aid premiums well- above-inflation. Increases in these premiums were between 10% and 12% for 2017 - but, while premiums were increasing significantly, payments to dentists continued to decrease, reflecting the “low priority” medical aid funders allocate to dentistry. I noted at the SADA
Congress 2017 that the dental profession is severely under attack – one is expected to go through five years of dental school, set up a dental surgery with very onerous requirements, use expensive equipment and material, employ fully qualified (and registered) staff, offer quality treatment to patients and yet it is the third party funders who decide on the cost of such treatment, sometimes to the great disadvantage of the dental service provider and his/her patient.

This is despite the fact that oral health is essential to sound general health, and that dentists can often detect more serious health problems early. Many studies show an as-yet-unexplained association between gum disease and several serious health conditions, including diabetes and heart disease.

South African dentists received only 2.34% of the total funds paid out to health care providers in 2015. This was down from 2.42% in 2014. With the exception of dental procedures covered under Prescribed Minimum Benefit (PMB) limits, dentistry is considered low priority by funders. The average increases in dental tariffs year on year have been between 5% and 6%. From 2014 to 2015, the total funds paid out to dentists increased by 5.44%. This shows that there has been no growth in the benefit structures available for dentistry as this figure is very much aligned to the inflation rates. The dental professionals are under pressure to deliver high quality service in keeping with international standards whilst struggling to meet practice overhead expenses in the light of consistently declining medical aid benefits. This is an area on which the Association will be focussing in the next few years, with a view to correcting the disparities so that all stakeholders are not disadvantaged. The patients will remain our top priority.
Introductory remarks on cartilages, bones and on “Bone: formation by autoinduction”

U Ripamonti

PROLOGUE
Introductory remarks on cartilages, on bones and on: “Bone formation by autoinduction.”
U Ripamonti

A prologue is an explanatory and introductory discourse, which in this case should then commence by asking “Why did I become interested in sharks, sharks’ cartilages, sharks’ teeth and evolution?”

It all started many years ago when, soon after landing in January 1983 in Africa from the cold shores of Milano University in North Italy, I met Tracey at the University of the Witwatersrand, Johannesburg, and with her hastened to the hot, feverish Natal North Coast, spending time in Umhlanga Rocks. There I visited the Natal Shark Board and was reminded during my tour that whilst sharks have an endoskeleton of cartilage there are some bony appendages on the integument, known as placoids or denticles (Fig. 1A). These render the integument into a formidable abrasive instrument which lacerates victims, causing bleeding, and provoking a full shark attack.

Back at the University of the Witwatersrand, Johannesburg, I went to the library of the Medical School where, with difficulties, I started to manually sieve through the Index Books then printed and made available world-wide by the National Library of Medicine of the National Institutes of Health (NIH) in Bethesda, Maryland USA.

It was serendipitous that I was attracted by an interesting, perhaps alluring, title of a published paper on sharks: “How to swim with sharks: A primer”. I read with mounting enthusiasm and interest the essay by a then unknown author who signed himself as Voltaire Cousteau. As the Editor’s foot note stated “the essay will make fascinating reading material and sets fundamental rules and principles that, if followed, will make it possible to swim, albeit with difficulties, amid sharks, whilst becoming experts through practice”.

Cousteau makes the wry comment “Actually, nobody wants to swim with sharks” and, reflecting on those who may have wrongly assumed that waters were not shark infested, “has by now doubtless lost any interest in learning how to swim with sharks”.

The essay “How to swim with sharks: A primer” further sparked my interest in shark biology, leading to my reading classic papers on the cartilaginous fishes and discovering that shark genera and species are fully evolved animals with superb differentiating pathways that make the shark the most effective killer swimming in the waters of our planet. Sharks or Selachians are named Chondrichthyes, fishes with an endoskeleton of cartilage thus including the jaws and the chondrocrania (a cartilaginous box containing the spinal nervous tissue of the shark).

Further reading across several different scientific disciplines revealed the concept that the cartilage forming the endoskeletons of Chondrichthyan like Elasmobranchs is not a primitive condition. It had been thought that cartilage was the original structure of the endoskeleton of fishes and that “bone only appeared at a late date in the history of fishes to augment and replace the cartilage,” However, it had been reported at the beginning of last century that Elasmobranchs, including sharks, are placoderm descendants “which have degenerated in their skeletal structure from an ancestral condition in which bone was present.”

Romer concurred in 1963, stating “the purely cartilaginous condition (of Elasmobranchs) is not truly a primitive one” and the absence of bone “is not an ancestral character, but one due to the degeneration from bone-bearing ancestors.”

This has obviously changed our understanding of the evolutionary skeletogenesis in animals up the vertebrate mammals. It is worth nothing then as Romer stated “that instead of beginning with a purely cartilaginous skeleton, and later gradually acquiring bone, the early vertebrates had a considerable degree of ossification which was followed in a majority of cases by a slump toward a cartilaginous condition.” Romer further stated that “Bone is an ancient, rather than a relatively new, skeletal material in the history of vertebrates”.

That regression may explain how in modern vertebrates “internal skeletal structures are first formed in cartilage, and only as development proceeds does the transformation of these cartilages into bony elements take place.”

In mammals, the majority of the bones of the skeleton arise from a cartilage anlage that serves as a strut for the induction of bone formation, undergoing vascular invasion with capillary sprouting into the hypertrophic cartilage. This vascular invasion brings about chondrolysis, i.e. the death of the cartilage anlage, and the differentiation of the first waves of osteoblast-like cells. These dif-
Differentiated cells lay down the early bone matrix, later to be mineralized to form the long bones of the mammalian skeleton (Fig. 1).

**Figure 1.**

A: Consecutive denticles (light blue arrow) embedded within the superficial integument of the adolescent dusky shark Carcharhinus obscurus. The developmental organization of such denticles and/or placoids make the skin of the sharks a highly abrasive tool that is continuously used by the animals to “feel” and/or “perceive” the potential prey. The contact and/or touch usually draw blood which will precipitate the full shark attack.1

B: The induction of chondrogenesis (light blue arrow) by highly purified naturally-derived bone morphogenetic proteins (osteogenin) purified to homogeneity delivered by collagenous bone matrices as carrier and implanted in the subcutaneous space of the rat.9 Note the tight adhesion/connection of the newly formed cartilage with the carrier matrix (light blue arrow).

C: Vascular invasion of the newly formed cartilage (recapitulating the cartilage anlage as seen in embryonic development) brings about the death of the newly formed cartilage (chondrolysis) (light blue arrow) extending to the hypertrophic chondrocytes (dark blue arrow).

D: Sustained progression of angiogenesis and capillary sprouting invocate the transdifferentiation of perivascular/pericytic stem cells that detach from the endothelial/vascular compartment (light blue arrows) to differentiate into osteoblastic-like cells when migrating under differentiating morphogenetic gradients to the bone inductive compartment.

E: De novo induction of a large ossicle heterotopically constructed in the rectus abdominis of Papio ursinus by 125 µg recombinant human transforming growth factor-β3 (hTGF-β3) harvested 30 days after intramuscular implantation. Note the corticalized surface (dark blue arrow) harvested from the rectus abdominis muscle.

F: Morphological digital image of an undecalcified section prepared from E showing extensive osteoid deposition along the newly formed bone as yet to be mineralized (dark blue arrows). The osteoid matrix is populated by contiguous secreting osteoblasts surrounded by a highly vascular stroma.

**Figure 2.** Plasticity of the newly forming bone by cellular condensations as engineered by morphogenetic capillaries and osteogenetic vessels. Capillary sprouting and invasion, cellular trafficking, perivascular stem cell differentiation and the induction of the primate cortico-cancellous bone.

A: Differentiating morphogenetic gradients set into motion by capillary sprouting and the morphogenetic and osteogenetic vessels initiate the molecular and morphological induction of an inductive microenvironment that constructs the cortico-cancellous primate bone. Each morphogenetic vessel is surrounded by mesenchymal cellular condensations (light blue arrows) that molecularly cross-talk with the osteogenetic/morphogenetic central blood vessel (white arrows) that constructs the spatio/temporal plasticity of the newly formed mineralized bone covered by plumped osteoblastic-like cells in close relationship with mineralized bone (dark blue arrows) enveloping the invading morphogenetic central blood vessels.

B: Early bone formation in relation to blood vessels (white arrows).

C: Cortico-cellular bone formation.

D: Blood vessels with adjacent osteogenic activity.

E: Osteogenesis in angiogenesis.

The critical role of angiogenesis in osteogenesis was described in detail in the middle of last century by the classic studies of Trueta which grandly provided the first insights into the supramolecular assembly of the extracellular matrix of bone.8 In several research experiments conducted at the Bone Research Laboratory, using either naturally-derived bone morphogenetic proteins9-11 or coral-derived macroporous constructs,12,13 the critical role of angiogen-
esis in osteogenesis has been characterised by defining the induction of bone formation as “osteogenesis in angiogenesis” (Fig. 2E).10,11,14-16

Intriguingly, Aristotle (384-322BC) has been credited to have stated that forming blood vessels have a patterning function during organogenesis.16,17 This concept of “morphogenetic vessels” long predates the idea proposed by Trueta in 1963 of “osteogenetic vessels.”18 The invading “organogenetic blood vessels” shape, pattern and induce the multistep cascade of the induction of bone formation, which is “osteogenesis in angiogenesis” (Fig. 2). An overview of the critical role of the vessels in bone formation was published in Science-in-Africa.19

Recently, a team of scientists has identified a specific vessel subtype in bone that links angiogenesis to osteogenesis.19 These specialized capillary sub-types, morphogenetic and osteogenetic vessels, provide niche signals to perivascular osteoprogenitor cells, as previously postulated by the grand insights of Aristotle and Trueta.8,11 As in mammals and as briefly discussed above, the cartilage anlage serves to initiate endochondral bone formation, that is a molecular and cellular morphogenetic event that forms the basis for the induction of endochondral bone formation, i.e. via a cartilaginous phase (Fig. 1B-D).10,11,15

Different molecular and morphogenetic events initiate the induction of bone formation via the intramembranous ossification pathway without a cartilage anlage. Angiogenesis with capillary sprouting is the three-dimensional construct for the induction of bone formation. Osteogenic precursor and other committed mesenchymal cells aggregate and condense in selected areas of the craniofacial skeleton. The mesenchymal condensations are surfaced by multiple osteoblast-like cells and other committed precursors which embrace the invading capillaries. The blood vessels are morphogenetic since there is further differentiation of surrounding mesenchymal tissue condensations. Finally, the three-dimensional pattern of new vessels with capillary sprouting and invasion within the newly formed secreted matrix, engineers the osteonic primate cortico-cancellous bone, a process which is shown in the fascinating “morphogenetic” images presented in Figure 2. Further angiogenesis and development give rise to the majority of the cranial and craniofacial skeleton which forms via intramembranous ossification without the need for a cartilage anlage. The process is clearly both “morphogenetic” and “osteogenetic” as per the Aristotlean and Trueta8 views and may be described as “osteogenesis in angiogenesis.”11,16

We now know that the presence of a cartilaginous endoskeleton in sharks has replaced a bony skeleton in the early evolution of the Elasmobranchs. The question of course arises how indeed was degeneration of the bony skeleton possible? A recent paper in Nature5 provides important insights into the evolution of cartilaginous fishes. That molecular experimentation reports that Elasmobranchs lack a specific secretory calcium binding phosphoprotein (SCPP).5 SCPP genes, arising from the Sparc-like1 (Sparcl1) gene family, have a crucial role in the formation of bone.5 It has been proposed, therefore, that the absence of the SCPP genes in the Elephant shark Callorhinus milii will account for the absence of bone from the endoskeleton of the animal.5

The Feature Paper which follows describes our understanding of these evolutionary “de-differentiating” events from a bony to a cartilaginous endoskeleton invoking evolutionary speciation highly favorable to degenerate the bony endoskeleton thus blocking the induction of bone formation and skeletogenesis. These “de-differentiating” events in selachians returning to cartilaginous endoskeletons have set evolutionary specificity that resulted in more resilient animals, with higher capacities to float, more favorable to deep immersions and feeding into the oceans without breaking a bony endoskeleton.

As Moss clearly states in his essay on the “Skeletal Tissues in Sharks,”20 the “cartilaginous endoskeleton of fossil Elasmobranchs is derived from phylogenetically ancestral forms with osseous endoskeletal tissues. In spite of the total lack of a bony endoskeleton, it has been emphasised that “bone does exist at the base (or pedicle) of the teeth and dermal denticles.”20

Hence, and importantly, sharks may not lack osteogenic ability, or their differentiating chondroblasts may have an intrinsic or genomic ability to differentiate into functional osteoblasts.20 Moss concludes that the “chondral” – or cartilaginous – state of the Elasmobranchs “was once believed to prove that cartilage preceeded bone in vertebrate evolution”. The acquisition however of a cartilaginous endoskeleton “does not establish a fundamental alteration of the potential and intrinsic ability of the Elasmobranchs’ scleroblasts to modulate into osteoblasts.”20

De-differentiation from a bony to a cartilaginous, or predominantly cartilaginous, endoskeleton in Elasmobranchs might have occurred following natural evolutionary selection resulting in the specifying of selected gene clusters encoding powerful inhibitors of angiogenesis. Cartilaginous matrices contain powerful morphogenetic signals that inhibit angiogenesis and capillary sprouting.21,22 Shark cartilages contain a substance that strongly inhibits the growth of new vessels which may well explain the rarity of malignant tumours in Elasmobranchs,23,24 and potentially could be used to inhibit tumour angiogenesis in humans.

Evolutionary pressure for superior habitats for feeding in the deeper waters of the oceans might have set in motion genetic mutations leading to the expression of several powerful inhibitors of angiogenesis, thus blocking the induction of bone formation. The molecular and cellular cascades of osteogenesis via an endochondral pathway, as seen in mammals and in teleost fishes (those having an endoskeleton of bone) have been blocked in the shark by the inhibitors of angiogenesis which have been found in extant shark cartilage.

On consideration, there is an intriguing possibility that it may be possible to “reactivate” the osteogenic pathways in the shark. What biological mechanisms could be involved?

My research interest then was primarily focused on the induction of bone formation using morphogens, firstly defined in 1952 25 as “forms generating substances”, and subsequently named “osseogenin” or “bone morphogenetic proteins”.9,26-28 following the colossal experimental work of several pioneers headed by Marshall Urist, at the Bone Research Laboratory at the University of California Los Angeles (UCLA). Urist published his fundamental and crucial studies in Science in a report titled “Bone: formation by autoinduction.”22 Hari A Reddi, at the Bone Cell Biology Section, NIH, Bethesda, made seminal discoveries on the chaotropic dissociative extraction and reconstitution of the bone matrix components, published in the Proceedings of the National Academy of the Sciences, USA.30,31

“A chaotropic agent is a molecule in water solution that can disrupt the hydrogen bonding network between water molecules (i.e. exerts chaotropic activity).
In this context, the question was then formulated whether the bone morphogenetic proteins would have the capacity to “force” endochondral bone formation and/or direct intramembranous bone formation by inducing bone in heterotopic intramuscular sites of cartilaginous Selachian recipients.

The idea of the experiment was conceived at the famous Oyster Box Hotel in Umhlanga. The Director of the Natal Shark Board granted permission to use their fast boats which continuously check the off-shore shark nets along the white beaches of the North Coast. (Figure 3A.) The Animal Ethics Committee of the University approved the intention to fish sharks and to harvest chondrocrania and vertebrae to enable the extraction of whatever morphogenetic factors Selachians may have within their cartilaginous matrices. In three expeditions off the shores of Umhlanga several dusky sharks and a larger shark, Carcharinus taurus, were fished out the ocean and enough quantities of cartilages were then secured (Fig 3B.)

At the laboratories of the then Dental Research Institute in Johannesburg Laura Yeates and I undertook the attempt at the extraction process from cartilage taken from the large shark Carcharinus taurus, using techniques we had learnt when studying the purification of naturally-derived bone morphogenetic proteins from bovine bones. Laura, already attached to the embryonic and emerging Bone Research Laboratory within the Dental Research Institute of the University, was confronted by the ultra-viscous extracted material overly rich in high molecular weight mucopolysaccharides that characterized the cartilaginous extracts. Several attempts to extract and purify the shark proteins resulted in partially purified morphogenetic factors.

Several months elapsed in the year 1989 without further experimentation and in 1990 I decided to invite Laura to join me at the Bone Cell Biology Section of the NIH in Bethesda. In collaboration with Dr AH Reddi, the then Chief of the Bone Cell Biology Section, Laura Yeates and I purified, to homogeneity, osteogenin, a bone morphogenetic protein, from baboon bone matrices (Fig. 1 B). An account of our mutual interactions during that rewarding and exhilarating scientific period was published some years later in Science in Africa, highlighting not only the extraction and purification of bone morphogenetic proteins from baboon bone matrices but also the grand effect of geometry on the induction of bone formation by culturing in vitro osteoprogenitor cells on specific geometric configurations of coral-derived macroporous bioreactors. The work was also published in a classic paper in Biomaterials.

An account of our endeavours was published by Science in Africa in 2012. Together we had cracked the purification to homogeneity of osteogenin from baboon bone matrices and defined the critical role of geometry on the induction of bone formation. These experiments were described at length in two papers in 2012.

After our success with the baboon proteins, we thought to again try to extract and purify proteins from Selachian cartilages, but using different chromatographic procedures. The samples had been flown with Laura and carried in her hand luggage from Africa to Washington DC. The extracted morphogenetic factors were later implanted in the subcutaneous space of rodents and uniquely and provocatively also implanted into the muscle of adolescent dusky sharks Carcharinus obscurus in a series of in vivo experiments in the salty water ponds of the Oceanographic Research Institute, Marine Parade, Durban, under the blue skies of the subtropical African sun.

My primary dedication was then, as it is now, the induction of bone formation in non-human and human primates using bone morphogenetic proteins as well as exploring the induction of bone formation by the mammalian transforming growth factor-β (TGF-β) isoforms. The work has continued with unique research results on the substantial induction of bone formation by the hTGF-β1 morphogen (Figs. 1 E,F), later published in the Journal of Cellular Molecular Medicine.

Whilst working as a learning and developing scientist at the then Dental Research Institute of the University, I had become interested also in the unique dentition of shark species that showed superb evolutionary traits for survival and predation. Of particular intrigue was the polyphyodonty (several rows of multiple teeth) in Selachians, which is the genesis of continuously erupting teeth that migrate forward until, like rolling over the edges of the carti-
laginous jaws, they are shed, leaving space for the replacement row of the newly formed and forward-moving teeth (Figs. 4 A,B). Biologically, this is rendered possible by the presence of a continuously erupting dental lamina located in the mucous membrane behind the rear rank of the phalanx (Fig. 4 C).41

**Figure 4**

**A**: radiographic analysis of a slice of *Carcharinus obscurus* jaw depicting the mineralized cartilaginous strut of the jaw (**light blue** arrow) and the continuous erupting and forward movement of the teeth revolving around the edge of the cartilaginous strut with later shedding of the most forward teeth.

**B**: The Selachians' teeth revolving along the cartilage and the conveyor belt (**light blue** arrow) eventually move into a different morphogenetic gradient that initiates the induction of dentinoclastogenesis and the loss of the tooth at the end of the jaw (**dark blue** arrow).

**C**: The continuously erupting dental lamina (**light blue** arrows) continuously generate dental elements (**dark blue** arrow) that move forward along the conveyor belt as discussed in the text.

It is the intention of the following Feature Paper to describe the extraction and purification of *Selachian*’s cartilages and the implantation of the extracted morphogenetic factors both in rodents and in *Carcharinus obscurus* sharks at the Oceanographic Research Institute in Durban. The manuscript also details the attachment apparatus of the connective tissue matrix to dentine or dentine-like material of the multiple shark teeth and describes the forward movement and migration of the *Selachian*’s teeth by a mechanism of a conveyor belt of condensed mesenchymal cells packed with fine connective tissue fibres that by tractional forces between cells and secreted matrix must move the rows of teeth forward until their exfoliation.

The Feature Paper additionally highlights the fact that the loss of teeth is physiologic in sharks but pathologic in mammals, and that tooth shedding in *Selachian* fishes is the *conditio sine qua non* for the ancestral evolutionary predatory habit of the sharks amidst the richly populated waters of our oceans.

We show that the implantation of coral-derived macroporous bio-reactors induce the differentiation of chondroblastic tissue and the differentiation of cartilage within the macroporous spaces, indicating the lack of an overt osteogenetic programme within the DNA of the *Selachian* cartilage.

We further propose that the lack of the induction of bone formation in *Selachian* intramuscular sites is the result of deficient angiogenesis and vascular invasion in the *Selachian* cartilages because of the overtly rich anti-angiogenic factors within the cartilaginous matrices21-24 which block “osteogenesis in angiogenesis.”
Sharks belong to the superclass \textit{Gnathostomata} (jawed vertebrates), class \textit{Chondrichthyes} (cartilaginous fishes), and subclass \textit{Elasmobranchii} (sharks, skates and rays). Sharks are classified into eight orders of which \textit{Carchariniforms} are well spread throughout the oceans. A phylogenetic investigation of twenty-four species of \textit{Carchariniforms} showed that most of the existing lineages of \textit{Carchariniforms} originated in the late Eocene to early Oligocene period (28 to 37 MYBP). The oldest fossilized shark remains are placoid scales from the Harding sandstone (late Ordovician ~ 450 MYBP) of Colorado.

\textit{Elasmobranchs} possess physiological feature that make them unique amongst fishes: a cartilaginous endoskeleton, with no ribs, a cartilaginous jaw which is not connected to the skull (chondrocranium)\(^2\) (Figs. 5, 6), and a cartilaginous back bone or chondrocranium engineered of cartilage to protect the cerebral ganglia of the animal (Fig. 7D). The shark uses body undulations to propel its mass through water via the use of large muscles enveloping the endoskeleton and also possesses an “active skin”. The skin, covered by placoid scales (dermal denticles or odontodes) is connected to thrust-producing muscles through myosepta. (Figs. 1A, 8).

Histological examination of selected shark tissues with notes on the extraction and purification of shark cartilaginous protein extracts.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure5}
\caption{Shark' jaws, mineralized cartilages, the tesserae, and the conveyor belt moving the teeth forward along the jaw of Car-}
\end{figure}
Carcharinus obscurus specimens. Low power view of C. obscurus’ jaws with multiple teeth moving forward as set by the conveyor belt above the mineralized tesserae the jaw. A: A conveyor belt-like structure (dark blue arrows) sets the teeth forward as described in the text. B: High power view depicting the conveyor belt (dark blue arrows) between the roots of the Selachian’s teeth (left) and the mineralized cartilage (right, light blue arrow). Mineralized areas are named tesserae as magnified in Inset D (light blue arrow). C: Transversal section of C. obscurus cartilaginous jaw depicts several forward moving teeth along the conveyor belt (dark blue arrow). E: TGF-β3 immunolocalization (light blue arrows) within the mineralized tesserae of the Selachian’s cartilaginous jaw. Undecalcified sections, tolu dine blue stain.

Figure 6

Figure 7. Low power views of the shark vertebral area, enveloping muscle and chondrocrania (D). A,B: Vertebrae with mineralized areas (light blue arrows) across the cartilage body also showing the cartilaginous protected ganglia of the Selachian’s body (dark blue arrows). C: Transversal section of C. obscurus caudal area showing the large muscular mass (white arrow) which propels the rapid undulatory movements of the Selachian fish. D: Transversal section through the chondrocranium of C. obscurus illustrating the cartilaginous tissues surrounding the ganglia of the Selachian’s rudimentary nervous system. Undecalcified sections, A,D Goldner’s trichrome; B,C toluidine blue.

Figure 8

Figure 8. Low power views of Carcharinus obscurus integument depicting the skin denticles embedded within the subjacent connective tissue stroma of the shark’s skin (light blue arrows).

The entire endoskeleton of the shark is composed of cartilage which may be mineralized to varying degrees with hydroxyapatite calcium phosphate crystals. The cartilaginous endoskeleton appears to be of functional importance related to the need to main-
tain a stiff yet flexible endoskeleton allowing for rapid undulating forward movements deep in the oceans. Type II collagen, proteoglycans and water are the main components of the elasmobranch cartilages. Types I and II collagens are responsible for the strength and stiffness of the endoskeleton. Notably, proteoglycans do have an inhibitory effect on the calcification of cartilage. Degradation of the proteoglycans by lysosomal enzymes results in calcification. Shark cartilage is strengthened in the jaws and vertebrae by small calcified areas called “tesserae” (Figs. 5D, 6A). Tesserae in sharks may occur in the jaws, gill arches, chondrocrania (a cartilaginous box containing the neural ganglia of the animal), vertebral centra (Figs. 7A,B), and the supporting cartilages of the fins and claspers. Tesserae are mainly localized at the periphery of the cartilaginous jaws (Figs. 5D, 6A). Note that TGF-β immunolocalizes within the tesserae of the cartilaginous jaws (Figs. 5D, 6A). There are three types of cartilaginous mineralization: areolar calcification, a compactly calcified tissue which is found in the vertebral centra (Figs. 7A, B), and prismatic and globular calcifications which may both occur in the blocks of mineralized tissue of the tesserae.

Sharks are polyphyodont, replacing teeth continuously throughout life. Their teeth are not fused to the jaws and are shed at regular intervals with rows or sets of teeth being replaced from behind in a “conveyor belt”-like progression by newly formed teeth as the shark grows or the anterior teeth become worn, broken or lost in predatory acts. (Figs. 4-6).

Still unresolved are the molecular forces which create the forward movement of the teeth. This conveyor belt mechanism was first described by Grady. Tractional forces are possibly initiated by interactions between cell and cell and between cells and extracellular matrix, propelling forward the conveyor belt and with it, all the dentition which has erupted along the belt-like tissue. High power histological examination of undecalcified whole mount sections display the mechanism just below the embedded roots of the Selachians’ teeth and above the margin of cartilage matrix, with calcified tesserae along the jaws (Figs. 5A-D). These views show aligned mesenchymal cells packed along with connective tissue fibres, further connected by a tenuous yet marked fibrillary matrix that envelopes the conveyor belt in its forward mechanisms, carrying the teeth and their attachment apparatus forward along the mineralized cartilage tesserae (Fig. 9).

It is possible that the conveyor belt is packed with myofibroblasts of Gabbiani’s definition50-52 that provide tractional forces along the connective tissue fibres, moving the teeth forward (Figs. 6,9).
Of note, only half of the total number of teeth are functional at any one time, the others being concealed beneath the epithelial layer. Teeth originate in the dental lamina which is a fold of the oral epithelium extending posteriorly to the lamina propria of the oral mucosa. It is the continuously erupting multiple teeth from the dental lamina within the shark’s jaws that defines polyphydonty (Figs. 5, 10, 11A).
arrows) yielding sequentially newly formed teeth (magenta arrow) that move forward by tractional forces of the conveyor belt (white arrows). B-D: TGF-β₁ immunolocalization of the erupting dental lamina (light blue arrows) as well as in the follicle below the erupting lamina (white arrows B,C). Undecalcified section, toluidine blue stain.

The dental lamina immunolocalizes the TGF-β₁ isoform (Figs. 11B,C,D), a multifunctional pleiotropic morphogen active in embryonic development in several animal phyla. It is not only localized in the dental epithelium (Fig. 11D) but also in mesenchymal proliferating cell condensations resting below the dental epithelium (Figs 11B,C) indicating further roles of the TGF-β₁ isoform in tissue morphogenesis of the Selachian masticatory apparatus.

Together with the cartilaginous endoskeleton and the “active skin” allowing both sinuous and powerful muscular propulsion of the shark through the waters of our oceans, the polyphodonty in Selachian species is the result of a natural genetic evolutionary selection that has ensured that, with no further evolution, the Selachians are unbeatable predators in the waters of the planet. Research has shown that tooth replacement rates are dependent on water temperatures, faster rates being recorded for summer and slower rates for winter months.

Various hypotheses have been proposed to account for the rapid tooth succession and replacements in sharks. It has been suggested that the proliferation of the dental epithelium together with the intercellular tissue fluid tension cause the forward movement of the Selachian’s teeth.54 One opinion held that fluid pressure within vacuoles situated in the tissue layer below the tooth bands create the forces necessary for tooth movement.54 It has been suggested that the expansion of the jaw cartilage during growth may account for tooth replacement. However, the cartilage expands only during growth and development, whereas continuous tooth replacement occurs throughout the life of the Selachian fishes.

Shark teeth consist mainly of dentine, (variously termed enameloid, mesodermal enamel or durodentin) covered with a calcified layer of ectodermal enamel, which is derived from ameloblastic activity and is attached to the underlying epithelial tissues by means of basal calcified tissue.53 This enamelloid covering of the tooth crown corresponds to the mammalian tooth enamel.54 Miss Moss claims that bone is present at the base or pedicle of shark teeth and placoid scales.30

Researchers in 1979, conducting a histological study on the jaws of three shark species, observed that between the teeth and the tesseræ layer of the jaw there are two layers of dense connective tissue.48 The upper layer is the tooth bed which contains large collagen fibres that radiate into the calcified matrix of the jaws. These fibres are akin to the Sharpey’s fibres seen in the mammalian periodontium,14 and serve as anchorage for the tooth. The inner layer (or supra-tesseræal layer) of connective tissue is similar, also having Sharpey-like fibres but with anchorage into the outer layer (or cap) of the tesseræ. There are blood vessels between these two connective tissue layers but very few branches are directed toward the tesseræ.

The Selachian’s attachment apparatus, then, is composed of several fibres uniting the conveyor belt to the root surfaces in the tridimensional space embedded within the supra-belt connective tissue (Fig. 9D, E). More coronally, connective tissue fibres, resembling periodontal ligament fibres as seen in mammals, connect the root surfaces to the surrounding mesenchymal tissues of the Selachian’s root microenvironment (Figs. 9F,G).

A distinctive feature in sharks, as opposed to mammals, is that, more medially or anteriorly along the cartilaginous jaw, which is reinforced by mineralized tesseræ, there is connective tissue attachment loss together with dentinoclastogenesis that eventually results in the exfoliation of teeth. Lost teeth are replaced by new rows of teeth which have been transported along the conveyor belt (Fig. 9). We have envisaged mechanical tractional forces set into motion by the mechano-transduction activity of the myofibroblasts52–55 that effectively transport the Selachian’s dentition forward (Fig. 4) replacing rows upon rows of exfoliated teeth.

The mechano-transducer conveyor belt also plays a fundamental role in tooth replacement. Low power digital images of the Selachian’s jaws show that when the edge of the cartilaginous jaw is reached, the connective tissue belt moves further anteriorly to the profile of the jaw, together with the teeth and their tractional bundle of connective tissue (Figs. 9A,B,C). The more anterior region is thus anatomically different and occupies a different molecular microenvironment whereby root resorption is set in train after activation of dentinoclasts (Fig. 9C).

Vertebral centra (Fig. 7) were first used for assessing age in Elasmobranchs; growth bands of shark centra were visualized by fluorescent markers to assess age and growth of leopard sharks Triakis seminasciata. An examination of the vertebrae of seven elasmobranch species found that Carcharhinids had the strongest vertebral centra, and that Elasmobranch vertebral cartilage is comparable in ultimate strength to mammalian bone.46 Mineralization in the jaws is related to their individual mineralized sub-units, i.e. tesseræ along the border of the cartilaginous matrix.

Carcharhinus obscurus or dusky shark species are found in the Atlantic, Pacific and Indian Oceans and can measure up to four meters in length. Dusky sharks are long-lived (up to 40 years of age) reaching sexual maturity at approximately 20 years of age. They give viviparous birth to offspring that measure between 85 and 100 cm. The maxillary teeth of C. obscurus are triangular and slightly oblique; the mandibular teeth are erect with narrow cusps. Dermal denticles (placoid scales) cover the integument of C. obscurus and are large and closely imbricated (Fig. 8).

- Studies have reported signs of an osteogenic programme during tissue morphogenesis in selected Elasmobranch species,55,56 further supporting the concept of the bony origin of the exoskeletal tissues of denticles. The possibility of “forcing” and/or “reprogramming” the induction of bone formation in shark intramuscular heterotopic sites became feasible as a result of the simultaneous availability of several key factors:
  - The extraction and purification of vertebral and chondrocranial proteinaceous material.
  - Large doses of recombinant human osteogenic proteins-1 (hOP-1).
  - Highly purified naturally-derived bone morphogenetic protein fractions, then labelled as osteogenin.9
  - The contemporary use of macroporous self-inducing coral-derived constructs.12,13

In addition, in vivo bioassay studies were undertaken to find out whether shark cartilage extracts might have retained osteoinductive proteins which could induce heterotopic bone formation in both the rodent and shark.
Shark tissue harvest and preparation of cartilaginous vertebrae and chondrocrania for protein extraction and purification.

Overall, the study encompasses a period from 1989 to 1994. The last bioassay of the intramuscular heterotopic sites of captive C. obscurus was completed in February 1994 at the Oceanographic Research Institute, Marine Parade, Durban, where captive sharks were housed. Records of the prolonged experimentation, including the rationales, material, methods, procedures and results were kept in two NIH record books 7530-00-222-3525. Federal Supply Service dated 1989 and 1990. The work fluctuated from the South African shores at Umhlanga Rocks, to the Dental Research Institute and later at the Bone Research Laboratory, Johannesburg, to the US shores at the National Institutes of Health, Bethesda, Bone Cell Biology Section, back again to the Bone Research Laboratory and finally again to the Oceanographic Research Institute to harvest the last heterotopically implanted C. obscurus animals.35

From June 1989 up to May 1990, with the help of the Natal Shark Board Umhlanga, sixteen C. obscurus and one C. taurus were harvested off the Indian Ocean. Animals brought to the docks of the boats were euthanized with 7 to 12 ml sodium pentobarbital injected through the ampulla in the cephalic ventral area. Whilst we were still at sea and fishing for additional adolescent C. obscurus: euthanized animals were dissected so as to efficiently harvest vertebral and chondrocranial cartilage material; jaws were dissected free and used only for histology and morphological analyses after fixation in 70% ethanol. Cartilages were kept on ice below board.

The harvested vertebrae and chondrocrania were cleansed of all the attached connective tissue as best as possible. When in the laboratories, the harvested cartilage was cooled with liquid nitrogen and immediately wrapped in cotton cloth and placed on the laboratory benches. Cartilages were frantrumated (shattered) and partly pulverized using a rubber hammer. The samples were dehydrated in ethanol and ethyl ether yielding a total of 934.12 g of vertebrae cartilaginous material: the corresponding chondrocranial material amounted to 429.4 g. Batches of cartilage harvested from C. taurus were extracted and partially purified at the Dental Research Institute of the University whilst the bulk of the dehydrated vertebrae and chondrocrania underwent extraction and the resulting proteins partially purified at the Bone Cell Biology Section, NIH, Bethesda, USA, July/August 1990.35

Chaotropic extraction of cartilaginous vertebrae and chondrocrania

Batches of dehydrated cartilage of both vertebrae and chondrocrania were demineralized with 10 volumes of 0.1N hydrochloric acid (HCl) under continuous monitoring of the pH to measure available hydroxyapatite ions. Cartilages were brached and demineralized after the fourth wash showed a constant strongly acid pH. The demineralized cartilage was then washed with deionized water to restore neutrality.

Different batches of approximately 200/300 g each were extracted with 4l 1.2 M guanidinium hydrochloride (Gdn-HCl) with enzyme inhibitors (β-aminio caproic acid, benzamide, and phenil methyl sulphonyl fluoride (PMSF), performed overnight in the cold with 20 g Ultrol®Grade Chaps [3-(3-cholamidopropyl)-dimethylammonio-1-propanesulfate]. The supernatant was drained off and centrifuged for 3hrs at 3000 rpm to remove proteoglycans. Vertebræ and chondrocrania were re-extracted with 1.2 M Gdn-HCl 0.5M acetic acid, pH 3.0 with enzyme inhibitors as above to further slightly demineralize the re-extracted residue. Vertebræ (and chondrocrania in separate experiments) were re-homogenized with short bursts of a polytron homogenizer. Extracts, kept cold all the times, were concentrated using a Pharmacia Filtron Ultrafiltration Unit. Extracts were diluted with 6l 0.5M acetic acid passed through an Amicon YM-100 spiral ultrafiltration cartridge to remove proteins having molecular weights higher than 100kD. The recovered proteins, of less than 100kD, were concentrated using the Pharmacia Filtron ultrafiltration unit (8l > 500ml – add 3.5l Urea = 7 volumes) to a final concentration of 500 ml 6M Urea/50mM Tris, pH 7.4.

Purification of vertebral and chondrocranial extracts, Heparin-Sepharose and Hydroxyapatite -Ultrigel affinity and adsorption chromatography.

After concentration and exchange to 6M urea/50mM Tris, pH 7.4, the extracts were loaded onto hydroxyapatite Ultrogel adsorption chromatography, eluted with 100 mM Na/Phosphate. Concentrated and exchanged eluates were loaded onto a heparin-Sepharose affinity chromatography column with a modified affinity column for further interactions between extracts and the chromatography gel. A 500ml Heparin-Sepharose gel was added to a 2l container and washed twice with 500 ml 6M Urea/50mM NaCl, 50mM Tris, pH 7.4. Cartilage extracts were then added adjusting the salt concentration to 0.15M NaCl by adding dry NaCl. The gel was mixed overnight under continuous stirring in the cold. The Heparin-Sepharose gel and bound fractions were repacked into a Pharmacia 500ml column and left to settle for at least an hour. The column was then washed with two column volumes of 0.15M NaCl, 6M Urea, 50mM Tris to wash off the unbound proteins. The column was then eluted with 6M Urea/50mM Tris, 1M NaCl, pH 7.4.

Characterization of protein extracts, preparation of protein samples for bioassay in rodents.

The 1M NaCl 6M Urea/50mM Tris Eluates after affinity chromatography of vertebral and/or chondrocranial extracts were concentrated to 150ml and exchanged to 4M Gdn-HCl/50 mM Tris and to final concentrations of 7 to 21ml 4M Gdn-HCl/50 mM Tris, pH 7.4. Seven to twenty-one ml of eluate of protein concentrates after Heparin-Sepharose affinity chromatography from either vertebrae or chondrocrania were loaded onto tandem Sephacryl S-200 HR (high resolution, Pharmacia) gel filtration chromatography column, equilibrated and eluted as described.3,28 Two ml aliquots from tubes 21 to 31 were concentrated in centriconos to ~200µl: 100µl of concentrate sample was used for reconstitution and bioassay in heterotopic sites of Long-Evans rats. Protein concentrations ranged from 175 to 540µg (vertebral extracts) and from 107 to 266 µg (chondrocranial extracts). Proteins were added to 25mg of insoluble collagenous bone matrix of Long-Evans rat suitably prepared.3,28 Two lyophilized pellets were implanted subcutaneously bilaterally under the skin of the chest of each rat 9,28 for a total of 22 implants in 11 Long-Evans rats.

Implantation of cartilaginous extracts and bone morphogenetic proteins in vivo in heterotopic intramuscular sites of Carcharhinus obscurus.

A total of nine adolescent Carcharinus obscurus ± 120cm in length were captured on different occasions at Umhlanga Rocks, north of Durban, South Africa. Animals were rapidly brought to the Oceanographic Research Institute by the Natal Shark Board fast boats and housed in the facilities of the Research Institute, Marine Parade, Durban. In three pre-planned surgical proce-
dures – cleared by the Animal Ethics Committee of the University of the Witwatersrand, Johannesburg – general anaesthesia was induced with ketamine-HCl. Operating at the edge of the salty ponds of the Institute the anaesthetized animals were implanted with one to four implants per animals. Time constraints had been stipulated by the Animal Ethics Screening Committee of the University so that in some animals no more than one or two pellets of osteogenic material were implanted. After incisional wounds and blunt dissection of the muscular tissue, lyophilized implants were inserted within the muscle pouch.

Table I below reports the number of implants and the type of implanted material after reconstitution with either vertebral or chondrocranial extracts, naturally-derived highly purified bone morphogenetic proteins, as well as recombinant human osteogenic protein-1 (hOP-1) delivered by either the extracted cartilaginous residues, synthetic RG503 matrix or by coral-derived fully-converted hydroxyapatite constructs with and without BMPs or hOP-1 as control.

Proteins concentrations of the different extracts and level of purification varied considerably across the purification scheme, from 4.15 to 7.76 mg/ml in crude extracts from both vertebrae and chondrocrania, to 0.82 mg/ml in Hep 1M fractions down to 540 to 175 or 266 to 107 µg as evaluated in S-200 gel filtration fractions from both vertebral and chondrocranial protein fractions eluates.

Table 1. Number of implanted specimens and type of osteogenic inductive morphogens reconstituted with various delivery systems as carrier for osteoinduction

<table>
<thead>
<tr>
<th>Osteoinductive Morphogen(s)</th>
<th>Number of implants</th>
<th>Number of Carcharhinus obscurus Time of tissue harvest</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5 &amp; 2.5 mg hOP-1 in RG503 matrix</td>
<td>6</td>
<td>A total of 17 C. obscurus housed at the Oceanographic Research Institute were used to implant 27 different osteogenic preparations as discussed in text. Animals were implanted with one to four implants each</td>
</tr>
<tr>
<td>Naturally-derived osteogenin + shark residue</td>
<td>2</td>
<td>21 and 37 days</td>
</tr>
<tr>
<td>S-200 shark vertebrae extracts</td>
<td>3</td>
<td>21 days</td>
</tr>
<tr>
<td>S-200 shark chondrocrania extracts</td>
<td>4</td>
<td>21 days</td>
</tr>
<tr>
<td>shark residue control</td>
<td>4</td>
<td>21 and 37 days</td>
</tr>
<tr>
<td>Coral-derived construct control</td>
<td>3</td>
<td>21 and 37 days</td>
</tr>
<tr>
<td>Coral-derived constructs + hOP-1 in RG503</td>
<td>3</td>
<td>21 days</td>
</tr>
</tbody>
</table>

Key: hOP1: osteogenic protein-1; RG503 synthetic matrix as carrier for hOP-1; S-200: gel filtration fractions after Sephacryl S-200 gel filtration chromatography; coral-derived construct: coral-derived macroporous hydroxyapatite after full hydrothermal conversion of calcium carbonate into hydroxyapatite.

Histological evaluation of tissue induction after in vivo implantation of cartilaginous extracts and bone morphogenetic proteins in heterotopic intramuscular sites of rodents.

Various extracts and purified protein fractions from either chondrocranial or vertebral cartilaginous matrices purified by gel filtration chromatography were reconstituted with allogeneic rat insoluble collagenous bone matrix residue and implanted in the subcutaneous space of the chest bilaterally in Long-Evans rats.

Tissue sections prepared from specimens harvested on day 12 after heterotopic implantation showed the invasion of multiple cellular elements between the allogeneic collagenous matrix particles with several rounded nuclear cells together with multinucleated giant cells with overall limited vascular invasion and angiogenesis (not shown). No cartilage or bone induction was observed in any of the examined sections (not shown).
or chondrocrania of the Selachians and tissue induction by recombinant human osteogenic protein-1 (hOP-1) implanted intramuscularly with carrier matrices in C. obscurus. A,B: Morphogenetic inductive gradients induced by) 0.5 mg hOP-1 combined with the synthetic RG503 polymeric carrier. Tissue induction of a calcified matrix with lacunae surfaced by cellular elements highly reminiscent of lamellar/osteonic bone (light blue arrows). C,D: Induction of mineralized matrix (light blue arrows) by 2.5 mg hOP-1 with multi-cellular lacunae highly suggestive of osteocyte lacunae but lack of cellular elements surfacing the newly formed tissue within the selachian’s muscle. E,F: Reconstitution of 0.5 mg hOP-1 with synthetic RG503 polymeric carrier (dark blue arrows - color bold TY) induces cellular and matrix tissue formation surrounding the implanted carrier (light blue arrows) highly reminiscent of the induction of bone formation with however lack of osteoblastic-like cells differentiation.

Specimens of rat allogeneic bone matrix reconstituted with 3 µg recombinant human osteogenic protein-1 (hOP-1) as positive control, induced cartilage and bone differentiation in heterotopic subcutaneous sites in the rodent bioassay (not shown).

Morphological and histological evaluation of tissue induction after in vivo implantation of cartilaginous extracts and bone morphogenetic proteins in heterotopic intramuscular sites of Carcharinus obscurus sharks.

Several inductive preparations delivered by either cartilaginous shark residue or coral-derived macroporous bioreactors as well as synthetic matrices were implanted in heterotopic intramuscular pouches created by sharp and blunt dissections in several recipient C. obscurus adolescent sharks.

Figure 13 shows tissue induction upon delivery of 0.5 or 2.5 mg recombinant hOP-1 in RG503 synthetic matrix implanted intramuscularly in C. obscurus and harvested on day 21 after heterotopic implantation. Tissue induction resulted in deposition of mineralized matrix resembling bone matrix with osteonic structures covered by cellular elements secreting the matrices, possibly pseudo-osteoblast-like cells palisading the newly secreted matrices (Figs. 13A,B).

Additional specimens representing heterotopic sites implanted with the higher dose of hOP-1 (2.5 mg recombinant hOP-1 in RG503 synthetic matrix) also showed the induction of mineralized trabecular-like tissue but without evidence of cellular elements along the trabecular surfaces (Figs. 13C,D).

High power view of heterotopically implanted specimens of 0.5 mg hOP-1 in RG503 synthetic matrix showed the induction of extracellular deposition with cellular activities within the matrix surrounding the implanted synthetic RG503 matrix (Figs. 13E,F). RG503 matrix without hOP-1 showed fragmentation of the synthetic carrier within the muscular tissue (not shown).

Specimens of collagenous residues recombined with S-200 fractions from either chondrocrania and/or vertebrae extracts showed encapsulation without evidence of chondrogenesis and/or osteogenesis (not shown).

Some tissue specimens could not be identified, being either totally absorbed or damaged by the shark movements after implantation; only one relatively intact specimen of coral-derived macroporous bioreactor could be identified and thoroughly processed; other coral-derived constructs presented as crushed and, although histologically processed, tissue invasion and/or induction within the macroporous spaces could not be easily identified.

It is noteworthy that a coral-derived macroporous construct implanted as control without addition of naturally-purified osteogenin and/or recombinant hOP-1 showed a significant chondrogenic tissue induction within the macroporous spaces (Fig. 13).

Figure 14. Within the limit of the implantation constraints into the intramuscular sites of the Selachian’s tissues at the Oceanographic Research Institute, Marine Parade, Durban, we were able to implant a number of coral-derived macroporous calcium-carbonate/hydroxyapatite bioreactors in heterotopic sites of the muscle tissues of selected sharks. The bioreactors were pre-loaded with recombinant human osteogenic protein-1 (hOP-1), highly purified naturally-derived osteogenic protein fractions (osteogenin,9 and hOP-1 pre-combined with RG503 synthetic matrices. Macroporous constructs were also implanted solo as control. Because of the inherent fragility of the calcium/phosphate constructs several specimens could not be properly retrieved or the histology showed only the crumpled aspect of the implanted bioreactors. One control specimen was amenable to proper histological processing and sectioning with successful staining of the newly formed induced tissues developing within the macroporous spaces. A,B: Low power views of the implanted constructs (white arrows). C,D: Induction of tissue formation within the macroporous spaces (light blue arrows) of cartilage-like material. E: Induction of cartilage with secretion of cartilaginous matrices (light blue arrow). Note columns of progressively differentiating chon-
droblasts (dark blue arrow) patterning the newly formed cartilage as in mammalian embryonic development. We speculate that the micro-inductive micro-environment of the coral-derived macroporous bioreactors engineers cartilaginous columnar condensations as seen in the mammalian counterpart. Tissue induction however lacks the induction of bone differentiation. The absence of an inductive programme setting into motion the induction of bone formation is possibly due to the evolutionary lack of genes regulating the bone induction cascade; alternatively, we do believe that the lack of vascular invasion followed by chondrolysis may be responsible for the lack of bone formation after evolutionary expression and synthesis of powerful inhibitors of angiogenesis (see text for detail) that block osteogenesis in angiogenesis.50,11,15

Chondrogenesis developed within the macroporous spaces even without the exogenously application of osteogenic morphogens (Fig. 14). Differentiated chondrocytes showed the secretion of chondrogenic extracellular matrix within the macroporous spaces and were thus embedded within the secreted matrix (Fig. 14E).

DISCUSSION, COMMENTS AND PERSPECTIVES

The study explored several biochemical and tissue induction phenomena in different animal species under the vast and pleiotropic umbrella of tissue induction and tissue biology by an array of morphogenetic substances with a vast pleiotropic cascade of biological activities. Certainly, the most salient result of the reported multiple studies is the induction of chondrogenesis by the heterotopically implanted coral-derived macroporous bioreactor.

Selected points should be discussed as novel information of the biology of the Selachians. The identification of a clear cut conveyor belt via special stains that moves the newly formed set of teeth forward by the continuously erupting dental lamina is worthwhile, and hence our hypothesis that such forward movement is controlled by modified myofibroblasts of Gabbiani’s definition.50-52 This molecular tractional pathway needs further explorative research by selected immunohistochemistry. A recent molecular and morphological investigation did report the presence of specific genes that govern the development and continuous regeneration of teeth in sharks.50

The immunolocalization of the TGF–β1 protein within the tesserae of the jaws and in the proliferating epithelium is of great significance, and additionally indicates that Selachian tissues do retain morphogens that in primates are powerfully inducers of endochondral bone formation.36-40

The TGF–β1 immunolocalization in the continuously erupting dental lamina is also noteworthy and suggests that the pleiotropic morphogen is also biologically active in the induction of tissue morphogenesis in Selachian tissues. A recent contribution, appearing in Bone, has indicated that TGF–β1 signalling is essential for osteoblast migration and differentiation in the medaka fish.56 In situ hybridization has shown the importance of TGF–β2 expression during fracture healing of the medaka fish. However, this is a bony fish. In Selachians, there is no bone, yet immunolocalization has shown the expression of the TGF–β1 isofrom. This indicates – as often previously stated - the ancient signalling pathways shared by several genera and species across the induction of tissue morphogenesis, from the fruit fly Drosophila melanogaster, to the bony fish medaka, to the non-human primate Papio ursinus to the human primate Homo sapiens, back to the amphibian tongueless and toothless African clawed toad Xenopus laevis.

Finally, and perhaps most importantly, a coral-derived construct without any adsorption of exogenously applied morphogenetic inductive signals, has activated the induction and differentiation of cartilage tissue within the heterotopically implanted macroporous spaces. As we have recently reported in Biomaterials.32 “the unique connubium of the hydrothermally exchanged coral-derived bioreactors, Ca++ release, angiogenesis, stem cell differentiation with expression and secretion of both angiogenic and osteogenic soluble molecular signals sets into motion the construction of the morphogenetic gradient of the macroporous bioreactor. Spontaneously, the bioreactor thus initiates the induction of bone formation even without the exogenous application of the osteogenic soluble molecular signals of the TGF–β1 supergene family”.

We thus report in our final statement that in heterotopic intramuscular sites in Selachians, the coral derived bioreactor does not initiate the cascade of bone differentiation but rather the induction of chondrogenesis within the macroporous spaces. The DNA of the Selachians does not have the developmental memory of the osteoinduction programme though it retains the induction of chondrogenesis as a recapitulation of evolutionary differentiating and de-differentiating events, ultimately lacking the angiogenesis and capillary sprouting required for setting chondrolysis into motion. Thus it may not restore the extinct signalling pathways of the induction of bone formation.

ACKNOWLEDGMENTS

The experiments on shark cartilages, harvesting the Selachian fishes out of the green waters of the Indian Ocean, the harvesting of the Selachian cartilages on the rolling decks of the powerful boats of the Natal Shark Board, working at the Dental Research Institute and later at the Bone Cell Biology Section of the NIH in the USA, flying back to Africa to implant not only purified shark extracts but recombinant human bone morphogenetic proteins intramuscularly in sharks together with purifying baboon osteogenin to homogeneity at the NIH has excited us to no end and determined our scientific performance and our hard and multiple tasks for several years ahead. The NIH period was for all of us the beginning of a scientific intellectually fascinating ride across the vast phenomena of the induction of bone formation, the spontaneous inducivity, animal phyla, molecular evolution, speciation and de-differentiation. We thank Hari Reddi, presently distinguished Professor and Hellison Chair, Musculoskeletal Molecular Biology, Centre for Tissue Regeneration and Repair, the University of California, Davis, USA, who made available to us back in the nineties his mind and his laboratories, not only to purify baboon bone matrices but also shark cartilages. We wish to thank Frank Luyten, then associate researcher with AH Reddi at the Bone Cell Biology Section for his help and suggestions on the shark cartilage extraction and purification. A note of thanks to Shabnum Meer, Oral Pathology, University of the Witwatersrand, Johannesburg, for discussing and diagnosing cartilage induction as reported in Figure 14. Preparing a manuscript with significant information and digital iconography and remaining continuously in touch with co-authors and the Bone Research Unit at the University entails communication often difficult when overseas. For the use of the IT connection, paper, printers – colour and black and white – the senior author would like to thank Carlo Brambilla and his outstanding wireless connection in Carnate, Milano, Francesco of the Sangiorgio Calzature, Merate, Milano, and finally Alberto and his team of managers at the Spar Mountain Lake, Broederstroom, North West Province, South Africa. This critical help as well as interest have been fundamental for many papers conceived, prepared and published by the Bone Research Laboratory of the University.

DECLARATION: There is no conflict of interest.
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Third molar impaction in a cross section of adult orthodontic patients.

ABSTRACT
Introduction: Third molars (M3) show the greatest variability of the human dentition. Impacted third molars have been implicated in oral infections, neoplastic conditions and late onset dental crowding.
Aims and Objectives: To assess the prevalence of third molar impaction among a sample of adult orthodontic patients in relation to their antero-posterior and vertical craniofacial skeletal patterns.
Design: A descriptive cross-sectional study.
Methods: Orthopantomograms and lateral cephalograms of 62 adult orthodontic patients were assessed. Presence and type of M3 impaction according to Winter’s and the Pell and Gregory’s classifications were documented and the relationships of these data to the vertical and antero-posterior cranio-facial skeletal patterns were assessed. All analyses were performed using the SPSS package, version 22. Level of significance was set at p<0.05.
Results: Median age was 23.5 years. Disto-angular impaction was commonest in the maxilla while mesio-angular and horizontal impactions were limited solely to the mandible (p<0.001). There was a significant relationship between Winter’s classification of M3 impaction and the antero-posterior skeletal pattern (p=0.007). Pell & Gregory class 3 impactions were found almost entirely among patients with class II malocclusion, (p<0.001).
Conclusion: M3 impaction is prevalent among the sample, the more severe impactions occurring in skeletal pattern II subjects.
Keywords: Impaction, Cephalometry, Third Molar, Skeletal Pattern

INTRODUCTION
The third molars (M3s), also called the “wisdom teeth” are the most variable of the teeth in man in terms of development and eruption. M3s may fail to appear because they are congenitally absent (agenesis) or fail to erupt because there is an obstruction in their path (impaction). Many factors have been implicated in these conditions. A meta-analysis has reported that M3 impaction occurs in 24.4% of the world population with no gender difference. A more locally based study observed an M3 impaction occurs in 24.4% of the world population with no gender difference. A recent meta-analysis has asserted that mandibular impactions are more commonly seen in the general population, although other studies had indicated that maxillary M3 impactions were more frequent. With regards to the skeletal pattern, M3 impaction has been reported to be less common in individuals with class III malocclusion, but more frequent among people with retrognathic mandibles. as well as with a tendency to deep bite occlusal relationships, and the type categorized by the facial axis angle. A recent meta-analysis has asserted that mandibular impactions are more commonly seen in the general population, although other studies had indicated that maxillary M3 impactions were the more frequent. With regards to the skeletal pattern, M3 impaction has been reported to be less common in individuals with class III malocclusion, but more frequent among people with retrognathic mandibles. as well as with a tendency to deep bite occlusal relationships, and the type categorized by the facial axis angle.

RESULTS
The overall rate of mandibular third molar impaction was 58.76 per cent. Those with a facial axis angle >93 (brachyfacials) however, M3 impaction has been observed in instances of both adequate and indeed excessive retro-molar space. This connotes that there are other factors involved in the impaction processes. These may include late calcification of the teeth, the position of mandibular M3s relative to the external oblique ridge, the inclination of the M3, length of the mandible and the skeletal pattern of the individual.

A recent meta-analysis has asserted that mandibular impactions are more commonly seen in the general population, although other studies had indicated that maxillary M3 impactions were more frequent. With regards to the skeletal pattern, M3 impaction has been reported to be less common in individuals with class III malocclusion, but more frequent among people with retrognathic mandibles. as well as with a tendency to deep bite occlusal relationships, and the type categorized by the facial axis angle. Reduced retro-molar space distal to the second molars has been reported as the single most important factor in the aetiology of M3 impaction, the availability of this space being dependent on growth of the jaw, and the facial type categorized by the facial axis angle.

1 Joy U. Ifesanya, BDS, MPH, FMCDs, Lecturer and Honorary Consultant Orthodontist, Faculty of Dentistry, University of Ibadan and University College Hospital, Ibadan.
2 Timothy O. Aladelusi, BDS, MSc, FWACS. Lecturer and Honorary Consultant Oral and Maxillofacial Surgeon, Faculty of Dentistry, University of Ibadan and University College Hospital, Ibadan.

Acronyms
M3: third molar tooth
OPGs: Orthopantomograms
METHODOLOGY
Ethical approval was obtained from the Institutional Reviews Committee (Approval number: UI/EC/16/0177). Orthopantomograms (OPGs) and cephalograms of patients seen in the orthodontic clinic were examined. The records of patients who at the time of first clinical evaluation were 17years and above, had not previously undergone orthodontic treatment nor had had third molars extracted, were selected for the study. Demographic variables of age and gender of all subjects were documented. The presence of M3 impaction, affected jaw and side as seen on the OPGs, were recorded. The type of M3 impaction was documented according to Winter’s,16 as well as Pell and Gregory’s17 classifications. The Winter classification is based on the size of the angulation between the long axis of the impacted mandibular M3 and the long axis of the second molar. The Pell and Gregory classification assesses mandibular M3 impaction on the basis of depth relative to the occlusal plane of the second molar tooth (with categories A, B or C) and the mesio-distal width of the tooth relative to the ramus (with categories 1, 2 or 3). These two schemes are the most widely accepted classification systems for quantifying M3 impaction and form the basis for most other classification schemes, hence their selection for use in this study. A classification of maxillary M3 impaction was described by Archer18 and corresponds to Winter’s,16 as well as Pell and Gregory’s17 classifications. The radiographs were viewed on an X-ray film viewing box independently by both researchers and the result verified with the documented models of the classification. A 90% agreement between the researchers was achieved (p<0.05).

The skeletal pattern of the subjects was measured on cephalometric radiographs which had been taken using the Pan-Blue-Oris machine (Blue-X Imaging ASSAGO, ITALY). The heads of the subjects had been held in a cephalostat, with the Frankfort plane parallel to the floor. The cephalograms were traced manually in a darkened room using 0.003″ cellulose acetate tracing sheets and a sharpened 2H pencil. The Steiner’s analysis was used to evaluate the skeletal pattern. The following variables were obtained:

SNA angle: Sella turcica-nasion-subspinale (A point) angle: Measures the relative position of the maxilla in relation to the anterior cranial base. It is indicative of prognathic or retrognathic maxilla. (Reference value for the Nigerian population is 85.5± 3.5°).19

SNB angle: Sella turcica-nasion-supramentale (B point) angle: Expresses the horizontal position of the mandible in reference to the anterior cranial base. It is indicative of a prognathic or retrognathic mandible. (Reference value for the Nigerian population is 82.3± 3.2°).19

ANB angle: A point-nasion-B point angle: Relates the maxilla & mandible to the cranial base. It is indicative of the skeletal pattern. (Reference value for the Nigerian population is 2-4°).19

FMA: Frankfurt Mandibular plane angle: Used to assess the degree of vertical discrepancy according to Tweed’s analysis. (Reference value for the Nigerian population is 20.8±3.1°).19

SNMPA: SN-Mandibular plane angle: Used to assess vertical facial discrepancy according to Steiner’s analysis. (Reference value for the Nigerian population is 30.9a±6.0°).20

Statistical analysis was carried out using the IBM SPSS package, version 22 (Armonk, New York, USA). Measures of central tendency were calculated and the relationships between third molar impaction and the various skeletal parameters were assessed using the Pearson’s Chi-square. Findings are presented in Tables. Level of significance was set at p<0.05.

RESULTS
Sixty-two patient records were assessed. The median age was 23.5years (IQR: 20 – 28). Twenty-eight (45.2%) subjects were males, 34 (54.8%) were females. There was no significant difference between the mean ages of the two gender groups (p=0.24; f-test= 1.40) and hence the data was analyzed jointly. M3 impaction was present in 32 (51.6%) subjects. There was no significant variation in the prevalence of third molar impaction between the gender groups (p=0.78).

Fifteen patients (24.2%) had skeletal Class I, 31(50.0%) had skeletal Class II and 16 (25.9%) had skeletal Class III. There was no significant difference in the prevalence of M3 impaction based on antero-posterior skeletal type (p=0.98). There was also no significant difference in the prevalence of M3 impaction based on vertical skeletal classification whether the SNMP (p=0.14) or FMA (p=0.45) was the reference angle for assessment.

Of the 248 third molars expected to be present in this study, six (2.4%) were congenitally missing in 3(4.8%) patients leaving a total of 242 third molars which were further analyzed in this study. The developmental absence of a third molar was recorded in six cases, all females, and affecting the maxillary arch in four instances (66.7%). Of the 242 third molars, 81(33.5%) were impacted. Fifty-two (64.2%) of these were found in the mandible, while 29(35.8%) were in the maxilla. Based on Winter’s classification, vertical impaction was the most prevalent, seen presented by 26 (32.1%) third molars. Mesio-angular impaction was observed in 25(30.9%) third molars, disto-angular impaction in 22(27.2%) third molars, while horizontal impaction was the least prevalent and was seen in eight (9.9%) of the third molars. Disto-angular impaction was commonest in the maxilla while mesio-angular and horizontal impactions were limited to the mandible (p<0.001), shown in Table 1. There was significant relationship between Winter’s classification of M3 impaction and the antero-posterior skeletal Classes (p=0.007). There was however no significant relationship between Winter’s classification of M3 impaction and the vertical skeletal classification as presented in Table 2.

On the basis of the Pell and Gregory classification, position ‘A’ and Class ‘2’, lower molar impaction was most prevalent overall. Pell & Gregory category 3 impactions with the mandibular M3s most embedded in the mandibular ramus were seen almost entirely among patients with Class II malocclusion. Pell & Gregory category 2 impactions were more common in Class I malocclusion, a finding which was statistically significant (p<0.001). The other relationships between the categories of M3 impaction assessed by applying the Pell and Gregory classification and the Winters system are shown in Table 3.

DISCUSSION
This study observed that less than 5% of our study participants suffered agenesis of one or more third molars. This is similar to a previous finding by Al-Delaimi et al.21 although other studies have however reported higher prevalences of M3 agenesis among orthodontic patients.1,13 Where gender predilections have been observed, females are more likely to present with M3 agenesis12 and this was true in this group of orthodontic patients where all cases showing missing third molars were females. With respect to jaw affected by M3 agenesis, our findings also agree with the general report of higher frequency in the maxillary arch.2,22

With regards to impaction, this study of our hospital patients found a prevalence similar to those previously reported among dental patients, whilst being higher than the prevalence seen in the general population, as has been reported previous-
Orthodontic patients are more likely to have a higher prevalence of third molar impaction than the general population since space deficiency is an aetiological factor common to both malocclusion and impactions. Contrary to other reports, we found no significant differences in the occurrence of M3 impaction in relation to skeletal patterns although the condition appeared to be more prevalent among Class II subjects. Findings from this study also agreed with the meta-analysis that there is no gender predilection for M3 impaction, contrary to the predominance of prevalence in the female gender as has been reported in another study.

Winter’s vertical impaction was overall the most common type observed in this study, with a marginal edge over the generally more prevalent mesio-angular impaction category. The majority of vertical impactions occurred in the maxilla while the mesio-angular impaction was most prevalent in the mandible, an observation similar to other reports among Iranians and a previous Nigerian study.

Analysis of the data found a relationship between Winter’s classification and antero-posterior skeletal classification, but not with the vertical skeletal pattern. This implies that the antero-posterior length rather than the vertical height of the jaw plays a more significant role in M3 impaction. The Pell and Gregory classification also found a significant relationship between the antero-posterior skeletal pattern and the class 3 impaction in which the M3 is seated deepest in the mandibular ramus, a category seen almost exclusively in skeletal Class II. This is similar to the report by Sogra et al, who found a significant correlation between the Pell and Gregory classification of mandibular M3 impactions and the skeletal pattern. The results of the present study, however, differ from those of Sogra et al., in that while those authors reported a significant correlation between the Pell and Gregory ‘ABC’ sub-classification and the skeletal classification, our findings show a significant relation with the ‘123’ sub-classification that is restricted mainly to Class II malocclusions. This may be attributed to the fact that skeletal Class II jaws have relatively smaller mandibles which are likely to have more severe space deficiencies than other skeletal jaw types, and hence more severe M3 impactions.

CONCLUSION

In conclusion, M3 impaction has been shown to be prevalent among orthodontic patients in this locality. Although the prevalence of impaction is not significantly higher in any specific Class of malocclusion, more severe impactions were observed in Class II malocclusion subjects presenting in our clinic. The clinical importance of this is that comprehensive orthodontic treatment planning should prudently consider the management of the M3 especially in Class II cases associated with retrognathic mandibles.

References

Table 1: Relationship between Winter's classification of impaction and jaw affected by impaction

<table>
<thead>
<tr>
<th>Variable</th>
<th>Jaw affected</th>
<th>Maxilla</th>
<th>Mandible</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disto-angular</td>
<td>16(72.7)</td>
<td>6(27.3)</td>
<td>22(100.0)</td>
<td></td>
</tr>
<tr>
<td>Vertical</td>
<td>13(50.0)</td>
<td>13(50.0)</td>
<td>26(100.0)</td>
<td></td>
</tr>
<tr>
<td>Mesio-angular</td>
<td>0(0.0)</td>
<td>25(100.0)</td>
<td>25(100.0)</td>
<td></td>
</tr>
<tr>
<td>Horizontal</td>
<td>(0.0)</td>
<td>8(100.0)</td>
<td>8(100.0)</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>29(35.8)</td>
<td>52(64.2)</td>
<td>81(100.0)</td>
<td></td>
</tr>
</tbody>
</table>

Chi Square = 33.7

Table 2: Relationship between Winter's classification and skeletal classification

<table>
<thead>
<tr>
<th>Variables</th>
<th>Winter's classification of M3 impaction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Disto-angular</td>
</tr>
<tr>
<td>Class I</td>
<td>5(26.3)</td>
</tr>
<tr>
<td>Class II</td>
<td>16(35.6)</td>
</tr>
</tbody>
</table>
P<0.001*   | x^2=17.80    |
| Class III | 1(5.9)       | 12(70.6) | 4(23.5)      | 0(0.0)     | 17(100.0) |
| Vertical skeletal pattern SNP |
| Normofacial | 11(22.9)  | 17(35.4) | 15(31.3)     | 5(10.4)    | 48(100.0) |
P=0.20     | x^2=8.53    |
| Brachyfacial | 0(0.0)   | 4(57.1)  | 3(42.9)      | 0(0.0)     | 7(100.0)  |
| Dolichofacial | 11(42.3) | 5(19.2)  | 7(26.9)      | 3(11.5)    | 26(100.0) |
| Vertical skeletal pattern FMA |
| Normofacial | 8(40.0)   | 3(15.0)  | 7(35.0)      | 2(10.0)    | 20(100.0) |
P=0.13     | x^2=9.95    |
| Brachyfacial | 0(0.0)    | 6(60.0)  | 4(40.0)      | 0(0.0)     | 10(100.0) |
| Dolichofacial | 14(27.5) | 17(33.3) | 14(27.5)     | 6(11.8)    | 51(100.0) |
| Total      | 22(27.2)    | 26(32.1) | 25(30.9)     | 8(9.9)     | 81(100.0) |

Table 3: Relationship between Pell & Gregory classification and the skeletal classification number (percent).

<table>
<thead>
<tr>
<th>Variables</th>
<th>A-P skeletal pattern</th>
<th>Vertical skeletal pattern SNP</th>
<th>Vertical skeletal pattern FMA</th>
</tr>
</thead>
<tbody>
<tr>
<td>A-P skeletal pattern</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class I</td>
<td>2(14.3)</td>
<td>11(36.7)</td>
<td>11(36.7)</td>
</tr>
<tr>
<td>Class II</td>
<td>9(33.3)</td>
<td>8(29.6)</td>
<td>2(40.0)</td>
</tr>
<tr>
<td>Class III</td>
<td>9(31.8)</td>
<td>2(8.22)</td>
<td>8(47.1)</td>
</tr>
<tr>
<td>Total</td>
<td>20(38.5)</td>
<td>21(40.4)</td>
<td>X²=2.55</td>
</tr>
<tr>
<td>*P&lt;0.001</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vertical skeletal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pattern SNP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normofacial</td>
<td>1(36.7)</td>
<td>13(36.7)</td>
<td>8(26.6)</td>
</tr>
<tr>
<td>Brachyfacial</td>
<td>3(60.0)</td>
<td>2(40.0)</td>
<td>0(0.0)</td>
</tr>
<tr>
<td>Dolichofacial</td>
<td>6(35.3)</td>
<td>8(47.1)</td>
<td>3(17.6)</td>
</tr>
<tr>
<td>Total</td>
<td>20(38.5)</td>
<td>21(40.4)</td>
<td>X²=2.55</td>
</tr>
<tr>
<td>*P=0.064</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vertical skeletal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pattern FMA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normofacial</td>
<td>3(25.0)</td>
<td>5(41.7)</td>
<td>4(33.3)</td>
</tr>
<tr>
<td>Brachyfacial</td>
<td>2(33.3)</td>
<td>2(33.3)</td>
<td>6(100.0)</td>
</tr>
<tr>
<td>Dolichofacial</td>
<td>15(44.1)</td>
<td>14(41.2)</td>
<td>5(14.7)</td>
</tr>
<tr>
<td>Total</td>
<td>20(38.5)</td>
<td>21(40.4)</td>
<td>X²=2.90</td>
</tr>
</tbody>
</table>

*Statistically significant
Knowledge, attitude and practices of alcohol and smoking among undergraduate oral health students at a South African University.

ABSTRACT
Background: The prevalence of alcohol consumption and smoking among university students is high.
Aim: To determine the knowledge, attitudes and practices of alcohol consumption and smoking among undergraduate oral health students at a South African university.
Methods: A cross-sectional study was conducted among dental and oral hygiene students (n=344) who were registered at a South African University in 2015. A self-administered questionnaire was used to collect information on socio-demographic characteristics, knowledge, attitudes and practices regarding consumption of alcohol and smoking. Data analysis included frequencies and correlations using chi-square tests, at a level of significance of p<0.05.
Results: A total of 269 (78%) students agreed to participate. The mean age was 22 years and 74% were female. More than a third (41%) reported consuming alcohol regularly while 11% reported being current smokers. More males reported indulging in both habits as compared with females. More than half felt that alcohol was acceptable as a social drink. The majority of clinical students (86%) associated periodontal diseases with smoking compared with pre-clinical students (74%).
Conclusion: The overall knowledge on social and health implications of excessive consumption of alcohol and smoking was adequate. The majority associated alcohol consumption and smoking with social activity.
Keywords: Knowledge, attitude, practices, alcohol and smoking, undergraduate university students

INTRODUCTION
Alcohol consumption and smoking tends to peak between the ages of 18 and 25 years and university students in these age groups are at particular risk for increased alcohol consumption when compared with non-enrolled age-matched controls. It has been observed that the overall trend of alcohol use increases from high school into university and then plateaus off after graduation. Alcoholic drinks contain ethanol, commonly known as “alcohol” and includes wines, beers and spirits. Alcohol consumption has been common for thousands of years and the drinking of alcoholic beverages is frequently a feature of social gatherings globally.

The rate of consumption of alcohol among the general population and heavy episodic drinking of alcohol among young adults are on the rise in many countries. Alcoholic drinks contain ethanol, commonly known as “alcohol” and includes wines, beers and spirits. Alcohol consumption has been common for thousands of years and the drinking of alcoholic beverages is frequently a feature of social gatherings globally.

Alcohol consumption and tobacco use are strongly-related behaviours, and the association between these two substances has been found to be strengthened with the heavier use of either substance. A study reported that the urge to smoke increases rapidly following heavy drinking, even among light smokers. It appears that smoking as a habit increases from school years into university and that smoking patterns among university students are relatively high. The university years seem to be a time of increased exposure to the risk of starting to smoke and a progression into regular patterns of use. National studies in the U.S. have shown that approximately 30% of university students reported having smoked in the past 30 days and 40% reported having smoked in the past year. University smokers are more likely to be non-daily smokers but smoke more in social situations when compared with their non-university peers.

The prevalence of smoking among South African students varied between 37% and 43% among males in tertiary institutions. As regards oral health students, only one similar study, has been carried out, in the Western Cape in 2010. Those authors reported an prevalence of 23%; which was lower than reports in other international studies.
No such investigation has been conducted in the dental universities of Gauteng and the current study provides baseline data that can be used for future intervention and surveys. The results should also enable dental schools to assess the knowledge of students on alcohol and smoking and if necessary provide the basis to modify the current teaching module to improve the outcomes.

Oral health graduates are role models in their communities and their social practices will be an example for many professional and lay people with whom they interact. Many habits will have been developed during their formative years as students and it is relevant therefore to determine the knowledge and consumption patterns of smoking and alcohol amongst an undergraduate oral health population.

AIM
The aim was to determine the knowledge, attitudes and practices of alcohol intake and smoking among undergraduate oral health students registered at a South African university in 2015.

METHODS
Ethical approval was obtained from the University of Pretoria, Faculty of Health Sciences Ethics committee (Ref 346/2015) No personal details of the students were disclosed and all information was strictly confidential and anonymous.

A cross-sectional analytical study design was used. There was a total of 298 dental and 46 oral hygiene students (n=344) registered at the School of Dentistry in 2015 and all were invited to participate. A modified and validated, self-administered questionnaire was used to collect information on the socio-demographic characteristics, knowledge, attitude and practices of the students with regards to smoking and consumption of alcohol.

The students’ knowledge regarding alcohol and smoking was assessed by asking them to select from a list of health conditions those that they thought may possibly be associated with the consumption of these substances. Knowledge was regarded as adequate with a score of 50% or more.

Attitudes towards the habits were obtained by asking questions related to how the students perceived alcohol served as a social drink and whether the intake can lead to social vices.

The practices of the students regarding alcohol and smoking were determined by asking students to provide their consumption patterns and the frequency of their intake of alcohol and of smoking. Regarding alcohol consumption patterns, students were grouped into; never (no alcohol intake); rarely (once a week) and regular (more than three times a week).

Students were categorized into either clinical or preclinical groups, as has been done in other similar studies. The clinical group included all 3rd, 4th and 5th year dentistry and 2nd and 3rd year oral hygiene (OH) students. The preclinical group included the 1st and 2nd year dental and 1st year OH students. Data analysis was performed using SPSS version 22. Descriptive and analytical statistical tests were done and the level of confidence was set at 95%. The level of significance was set at p<0.05.

RESULTS
The response rate was 78% (n=269) with a mean age of 22 years (17-42; SD±3.25). The final sample included 225 (83%) dental and 44 (16%) OH students; 199 (74%) were female and 136 (51%) were preclinical students (Figure 1).

There were 107 (41%) participants who reported their consumption of alcohol as “regular” and almost half of all males (46%) acknowledged as being regular consumers of alcohol. Of those who responded to the question related to the habit of smoking (n=258), a significant number (89%) reported they did not smoke regularly (p=0.01). Of the 11% who reported being current smokers, more males (19% of male sample) compared with females (8% of female sample) were smokers (Table I).

Assessment of the knowledge of students regarding the ill effects of alcohol consumption was revealed by a majority (93%) correctly identifying liver disease, while less than half (39%) associated hypertension (46%), diabetes (39%) and cancer (39%) as outcomes of excessive alcohol intake. Significantly more clinical (46%) than pre-clinical (33%) students correctly identified cancer as an outcome of excessive alcohol consumption (p=0.03) but there were no significant differences in the levels of knowledge between gender and course of study (Table II).

Regarding attitudes towards alcohol consumption, 63% felt that alcohol was acceptable as a social drink and 70% felt it was acceptable for alcohol to be consumed when entertaining friends. However, it must be noted that almost half of the sample (47%) recognized the harm associated with excessive alcohol consumption.

When considering the attitude towards smoking, the majority of the participants (92%) felt that professionals have a role in giving advice to patients about cessation of any tobacco products, should routinely advise patients who smoke (89%); and should seek training on controlling tobacco use (84%). Of the five health conditions that participants could identify as associated with smoking, more clinical students (86%) associated periodontal diseases with smoking compared with the pre-clinical (74%) group (p=0.01). There were no significant differences between the genders or the course of study in relation to their knowledge regarding smoking (Table III).

DISCUSSION
The response rate for this study was 78% and this could be due to the delivering and collecting of the questionnaires during lectures as attendance is compulsory. Most of the students agreed to participate and completed the questionnaire.

The overall prevalence of smoking in the sample was 11% and this was a proportion similar to that reported in other studies in which the frequency varied between 3% and 13%. However, the figure was much lower compared with the Western Cape study which reported a prevalence of 23%. That survey was conducted more than five years ago and since then South Africa has implemented and extended its anti-tobacco legislation and policies throughout the country, probably resulting in the reduced prevalence. The relatively superior knowledge level associated with smoking seen in SA could be due to the inception of that legislation and the campaigns which have been implemented being robust and effective. In addition, health professionals are used as advocates in the prevention of smoking. The dental team is involved in efforts to reduce smoking, and the profession plays a critical role in tobacco control, ongoing treatment, counselling and prevention. This study is the only paper which has reported that students linked smoking with periodontal diseases. The majority of previous studies recorded student opinion as linking smoking with lung cancer and heart diseases. This may be due to the fact that the current study was conducted amongst oral health students, whilst most other reviewed studies have been conducted amongst medical students.

There were no significant differences between the genders, nor
between the courses of study (dentistry and oral hygiene) in relation to knowledge regarding smoking.

The widespread use of alcohol and the emphasis of education regarding the harms of excessive alcohol consumption may have led to the very high level of knowledge regarding the damaging effects on the liver. However, knowledge is inadequate with regards to hypertension, diabetes and especially cancer, which is a concern, although the results are similar to those found in other studies.21,20

Regarding the attitude towards alcohol consumption, 63% (dental) and 70% (oral hygiene) felt that alcohol was acceptable as a social drink and for entertaining with friends. This was considerably higher than the Nigerian study which reported that only 20% serve alcohol to friends as a social drink.21 This difference could be due to cultural and religious beliefs.

Alcohol intake is known to be the world’s third largest risk factor for the burden of disease.30 Excessive alcohol intake is known to be common among adolescents and young adults which is associated with intoxication and many negative social and health consequences including violence, child neglect and abuse, absenteeism from workplace and chronic diseases.31 This suggests that there is a need for intensive campaigns against alcohol use for this group of young individuals.

CONCLUSION

The overall knowledge of undergraduate students on social and health implications of excessive consumption of alcohol and smoking was adequate although it was lacking on alcohol with regards to certain diseases. The majority associated smoking and alcohol consumption with social activities. Alcohol and smoking practices were similar to other studies.

RECOMMENDATIONS

More emphasis on the ill effects of alcohol and smoking should be included in the curriculum to help improve students’ knowledge. Students should be allowed to visit oncology wards during their outreach visits to make them aware of the ill effects of alcohol and smoking as risk factors in the development of cancer.

Outreach activities should be used to allow students to deliver anti-smoking, drug and alcohol messages to learners at primary and high school settings to reinforce their own knowledge and to discuss the ill effects of smoking and alcohol with learners.

ACKNOWLEDGEMENTS

The authors would like to thank all the students for participating in this study and the management of the University for granting permission to conduct the study.

Declaration: No conflict of interest was declared.

References

24. Andrade APAd, Bernardo ACC, Viegas CAdA, Ferreira DBL, Gomes


### Table I: Alcohol and smoking consumption of the participants (n=264)

<table>
<thead>
<tr>
<th></th>
<th>Male (%)</th>
<th>Female (%)</th>
<th>Total (%)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Never consumed alcohol</td>
<td>19 (27)</td>
<td>49 (25)</td>
<td>68 (26)</td>
<td>0.08</td>
</tr>
<tr>
<td>Rarely consumed alcohol</td>
<td>19 (27)</td>
<td>70 (36)</td>
<td>89 (34)</td>
<td></td>
</tr>
<tr>
<td>Regularly consumed alcohol</td>
<td>32 (46)</td>
<td>75 (39)</td>
<td>107 (41)</td>
<td></td>
</tr>
<tr>
<td>Prevalence of smoking (n=258)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current smokers</td>
<td>13 (19)</td>
<td>16 (8)</td>
<td>29 (11)</td>
<td>0.01</td>
</tr>
<tr>
<td>Non-smokers</td>
<td>54 (81)</td>
<td>175 (92)</td>
<td>229 (89)</td>
<td></td>
</tr>
</tbody>
</table>

### Table II: The ability of students to positively link health conditions with excessive alcohol consumption.

<table>
<thead>
<tr>
<th></th>
<th>Liver disease (%)</th>
<th>p-value</th>
<th>Hypertension (%)</th>
<th>p-value</th>
<th>Diabetes (%)</th>
<th>p-value</th>
<th>Cancer (%)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>66 (93)</td>
<td>0.99</td>
<td>34 (48)</td>
<td>0.78</td>
<td>25 (35)</td>
<td>0.49</td>
<td>27 (38)</td>
<td>0.78</td>
</tr>
<tr>
<td>Female</td>
<td>184 (93)</td>
<td>0.25</td>
<td>63 (46)</td>
<td>0.96</td>
<td>46 (35)</td>
<td>0.25</td>
<td>45 (33)</td>
<td>0.03*</td>
</tr>
<tr>
<td>Preclinical</td>
<td>124 (91)</td>
<td>0.22</td>
<td>17 (39)</td>
<td>0.25</td>
<td>12 (27)</td>
<td>0.09</td>
<td>12 (27)</td>
<td>0.72</td>
</tr>
<tr>
<td>Clinical</td>
<td>126 (95)</td>
<td>0.22</td>
<td>108 (48)</td>
<td>0.25</td>
<td>92 (42)</td>
<td>0.25</td>
<td>94 (42)</td>
<td>0.72</td>
</tr>
<tr>
<td>Oral Hygiene</td>
<td>39 (89)</td>
<td>0.22</td>
<td>125 (46)</td>
<td>0.25</td>
<td>104 (39)</td>
<td>0.25</td>
<td>106 (46)</td>
<td>0.72</td>
</tr>
<tr>
<td>Dentistry</td>
<td>211 (94)</td>
<td>0.22</td>
<td>125 (46)</td>
<td>0.25</td>
<td>104 (39)</td>
<td>0.25</td>
<td>106 (46)</td>
<td>0.72</td>
</tr>
<tr>
<td>Total</td>
<td>250 (93)</td>
<td>0.22</td>
<td>125 (46)</td>
<td>0.25</td>
<td>104 (39)</td>
<td>0.25</td>
<td>106 (46)</td>
<td>0.72</td>
</tr>
</tbody>
</table>

*Statistically significant using Chi-Square test

### Table III: The ability of students to positively link health conditions with excessive smoking (n=269)

<table>
<thead>
<tr>
<th></th>
<th>Periodontal disease (%)</th>
<th>p-value</th>
<th>Hypertension (%)</th>
<th>p-value</th>
<th>Lung disease (%)</th>
<th>p-value</th>
<th>Cancer (%)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>53 (75%)</td>
<td>0.17</td>
<td>21 (50%)</td>
<td>0.52</td>
<td>64 (87%)</td>
<td>0.28</td>
<td>45 (64%)</td>
<td>0.11</td>
</tr>
<tr>
<td>Female</td>
<td>157 (83%)</td>
<td>0.01*</td>
<td>102 (46%)</td>
<td>0.96</td>
<td>175 (92%)</td>
<td>0.12</td>
<td>142 (74%)</td>
<td>0.51</td>
</tr>
<tr>
<td>Preclinical</td>
<td>98 (74%)</td>
<td>0.01*</td>
<td>60 (45%)</td>
<td>0.25</td>
<td>123 (93%)</td>
<td>0.99</td>
<td>97 (73%)</td>
<td>0.28</td>
</tr>
<tr>
<td>Clinical</td>
<td>112 (86%)</td>
<td>0.01*</td>
<td>63 (48%)</td>
<td>0.25</td>
<td>113 (88%)</td>
<td>0.99</td>
<td>90 (70%)</td>
<td>0.28</td>
</tr>
<tr>
<td>Oral Hygiene</td>
<td>36 (86%)</td>
<td>0.01*</td>
<td>21 (50%)</td>
<td>0.25</td>
<td>38 (90%)</td>
<td>0.99</td>
<td>33 (79%)</td>
<td>0.28</td>
</tr>
<tr>
<td>Dentistry</td>
<td>174 (79%)</td>
<td>0.01*</td>
<td>102 (46%)</td>
<td>0.25</td>
<td>198 (90%)</td>
<td>0.99</td>
<td>154 (70%)</td>
<td>0.28</td>
</tr>
<tr>
<td>Total</td>
<td>210 (78%)</td>
<td>0.01*</td>
<td>123 (46%)</td>
<td>0.25</td>
<td>239 (89%)</td>
<td>0.99</td>
<td>187 (69%)</td>
<td>0.28</td>
</tr>
</tbody>
</table>

*Statistically significant using Chi-Square test
Abstract: Introduction: Accurate tooth size prediction of permanent successor teeth is important for treatment planning during the mixed dentition stage.

Objectives: To compare and determine the accuracy of two methods used to predict mesiodistal widths of permanent canines, first and second premolars in a sample of Black South Africans.

Methods: Measurements of teeth were performed on study models of a sample of 100 children (50 males and 50 females) aged between 17 and 21 years. Digital calipers, accurate to 0.05mm, were used. Estimations of the widths of the permanent canines, first and second premolars (C+P1+P2) were performed for mandibular and maxillary arches using the Schirmer and Wiltshire and the Modified Tanaka-Johnston prediction methods. The estimated (C+P1+P2) values were then compared with measurements of the actual teeth on the study models.

Results: Males presented significantly larger mesiodistal tooth dimensions than females. The Modified Tanaka-Johnston equation is accurate for females but overestimates actual tooth measurements for males. The Schirmer and Wiltshire method is accurate for male maxillary and female mandibular tooth sizes but overestimates male mandibular and underestimate female maxillary tooth sizes.

Conclusion: Both methods have deficiencies; however the Modified Tanaka-Johnston equation proved preferable because it overestimates rather than underestimates actual tooth sizes.

Keywords: prediction; mesiodistal widths; Moyers method; Tanaka and Johnston equation

INTRODUCTION

Tooth size prediction and space analysis are an essential part of orthodontic treatment planning in the transitional dentition stage when it is critical to establish whether or not the available arch space is sufficient to accommodate all the permanent teeth. During the transitional dentition stage the permanent canine, first and second premolars are usually not clinically visible and accessible for measurement of their actual mesiodistal widths.1-3 It is hence necessary to estimate the mesiodistal dimensions of these teeth to enable treatment planning. The accuracy of prediction methods is critical in orthodontic management, since inaccurate estimation could compromise any treatment and potentially result in failed intervention or extended treatment time.4,5

LITERATURE REVIEW

Most orthodontists use the Moyer’s4,6,7 and the Tanaka and Johnston8,9 methods to estimate the widths of the canines, the first and second premolars. The Moyer’s prediction table method was the first used to predict widths of permanent teeth, hence its popularity and status as reference point. This method was derived from North American Caucasian subjects, which makes it most applicable and reliable in similar populations.10 The Moyers tables present two major shortcomings; first, this method has limited applicability among different ethnic groups. A recent meta-analysis found that the Moyer’s method cannot be “universally applied without question”, hence the need to develop population specific prediction tables which may counteract the overestimation errors possible when using Moyer’s tables.4,10 Secondly, the prediction tables are considered cumbersome and not easy to use, when compared with regression equations. Tanaka and Johnston developed a regression equation based on a sample of 506 Caucasian children.6 In predicting the dimensions of permanent (C+P1+P2) the equation requires that half the sum of the widths of the mandibular incisors is added to 11.0 for maxillary teeth and 10.5 for mandibular teeth. However, and in a manner similar to its predecessors, this prediction method is not applicable across all races and ethnic groups, especially non-Caucasian samples.4,5,11

Acronyms
MTJ : Modified Tanaka and Johnston
SaW : Schirmer and Wiltshire
Two studies undertaken among black South Africans showed that the Moyers and the Tanaka and Johnston methods were inadequate in their prediction and needed modifications.\textsuperscript{12,13} It was further found that these methods underestimated tooth dimensions in South African populations, and failed to account for gender differences. In addressing these problems, Khan, Seedat and Hlongwa\textsuperscript{12} applied the Tanaka and Johnston approach to develop a regression model more applicable to black South Africans. Their new equation for males calculated the maxillary \((C+P_1+P_2)\) per quadrant as \(8.31 \text{ mm} + 0.62x\) and the mandibular \((C+P_1+P_2)\) per quadrant as \(7.15 \text{ mm} + 0.67x\), where \(x\) is the sum of the widths of the mandibular incisors.

The Moyers tables and the Tanaka and Johnston equations have indeed undergone modification and adaptations worldwide to conform to the norms of local population groups. Specific models are available for Chinese,\textsuperscript{8} Jordanians,\textsuperscript{14} Italians,\textsuperscript{15} Africans,\textsuperscript{11,16} Thai,\textsuperscript{17} Malay\textsuperscript{18} and other groups. In South Africa, modifications to these methods were tested among black Africans by Schirmer and Wiltshire\textsuperscript{13}, and by Khan et al.\textsuperscript{12} under different settings and time. In the South African context, the methods used for blacks to replace Moyers tables and Tanaka and Johnston equations are Schirmer and Wiltshire and Modified Tanaka-Johnston by Khan et al., respectively.\textsuperscript{12,13}

The aim of this study was to apply the Schirmer and Wiltshire (SaW) and Modified Tanaka and Johnston (MTJ) methods to a sample drawn from the black South African group to determine which method yielded the more accurate prediction of mesiodistal widths of canine, and premolars.

**METHODS**

**Study design**

Cross-sectional survey of pre-treatment orthodontic study casts (maxillary and mandibular arches).

**Study Setting**

The research was undertaken in the Department of Orthodontics at the Sefako Makgatho Health Sciences University, Medunsa Oral Health Centre. Data used in this study was based on the archived study casts of patients attending the clinic.

**Study Population**

Orthodontic study casts of black South African patients were sourced from the Department of Orthodontics at the Sefako Makgatho Health Sciences University, Medunsa Oral Health Centre. A sample estimation of 100 was derived, using Epi\textsuperscript{TM} 7 at 95\% confidence level and 5\% confidence limits with the expected frequency of 8\%, from a total number of 813 black patients between the ages of 17-21 who consulted during the year 2011. The sample comprised equal groups of the models of 50 males and of 50 females, all having Class 1 molar relationships with all permanent teeth present up to the first permanent molar in all quadrants.

The inclusion criteria required that all permanent teeth were fully erupted and relatively well aligned, and the study models were of good quality with no fractures, voids, or abnormalities. Excluded from the sample were models of patients who were of a different race group, whose ages were outside the limits or had severe malocclusions such as deep bites, excessive crowding, hypoplastic teeth, multiple spacing and diastemas, occlusal wear, missing permanent teeth, retained deciduous teeth or evident tooth size discrepancies.

**Determination of mesiodistal measurements (widths)**

All measurements were carried out by the principal researcher, using an electronic digital Vernier caliper (Sylvac, Fowler). The caliper was calibrated in 0.5 millimetres and values recorded to two decimal places. A random sample (10\%) of orthodontic casts were selected and re-measured by the principal researcher and by a second researcher to determine reliability of data.

Mesiodistal widths were measured and recorded for the four mandibular permanent incisors, maxillary and mandibular permanent canines and premolars.

**Data analysis**

Data were entered into a computer and analysed using SAS for Windows version 9.2. An independent sample Student t-test was used to assess the differences in total widths \((C+P_1+P_2)\) between the actual and predicted values. These tests were stratified by gender. Pearson correlations values were calculated to measure the strength of association.

**Ethical Clearance**

Permission to undertake this research was granted by the Medunsa Research and Ethics Committee, (Project: MREC/D/47/2011).

**RESULTS**

The sample size of 100 was considered adequate based on the Central Limit theorem,\textsuperscript{19} similar studies\textsuperscript{12,13,20,21} and objectives of the study.

(i) Reliability test

The blinded repeated measurements of 10 casts revealed almost perfect correlation, and validation of the reproducibility of measurements. The intra-rater and inter-rater coefficients were extremely high, indicative of excellent correlation (r>0.90) in both cases.

(ii) Statistical tests of means

Descriptive analysis of the mesiodistal widths \((C+P_1+P_2)\) in both arches indicate a significant gender difference in this parameter. Males presented with significantly larger teeth than females in both the maxilla and the mandible (P values, 0.00017 and 0.0002) respectively. (Table 1).

The paired t-test in males revealed significant differences between predicted (MTJ) and the actual mesiodistal measurements, at P<0.000 for both the maxillary and mandibular arches. In both instances the MTJ method overestimated the mesiodistal widths of the permanent teeth. (Table 2) Analysis of the female data yielded insignificant differences in both arches, P=0.1848 and 0.3776 respectively for maxilla and mandible. (Table 2). These findings can be interpreted to mean that the Modified Tanaka-Johnston method fails to predict with precision the actual dimensions of permanent \((C+P_1+P_2)\) in males. In females however, this prediction method was shown to be comparatively accurate.

Comparison of the mesiodistal widths of teeth between the SaW predicted data and the actual values show mixed results for gender and arch. In the maxillary arch, there is a significant difference between the measured and predicted mesiodistal dimensions of permanent teeth of females (P=0.0114); for males the p-value of 0.1748 is indicative of a lack of statistical significance. Mandibular arch discrepancies are however significant for males (p=0.0198), and for females, although at a lower level (p=0.2990). (Table 3).

**DISCUSSION**

That there are racial and ethnic differences in tooth dimensions is well established in the literature, with blacks having larger teeth than most other population groups.\textsuperscript{14,18,20} A comparison of the mesiodistal widths of teeth in the study sample with those of other groups\textsuperscript{12,13,20} (previously derived from Tanaka and Johnson equations) was undertaken. The findings indicate that the study population presented with larger teeth for both genders and in all groups.
Significant sexual dimorphism is apparent in this study; males present with larger mesiodistal dental measurements than females (Table 1). The findings in this study are similar to several comparable studies, hence the recommendation that it would be prudent to develop gender-specific equations and tables for prediction.

When assessed statistically, the MTJ equation overestimated the mesiodistal widths of mandibular and maxillary teeth in males, but was precise in females (Table 2). These findings indicate that this method can be applied with greater confidence in predicting the dimensions of unerupted teeth for females than in males in similar groupings. A plethora of studies indicate that the Tanaka and Johnston equation overestimates tooth dimensions, and as a consequence better equations were developed to cater for specific racial groups and genders. Expressing a lonely contrary opinion, one study on Jordanian children observed that the Tanaka and Johnston equation underestimated tooth measurements.

### Table 1: Comparison of actual mean mesiodistal widths (C+P1+P2) between males and females

<table>
<thead>
<tr>
<th>Arch</th>
<th>Gender</th>
<th>Mean (SD)</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maxilla</td>
<td>Male</td>
<td>47.20 (2.65)</td>
<td>0.0002</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>45.60 (2.30)</td>
<td></td>
</tr>
<tr>
<td>Mandible</td>
<td>Male</td>
<td>47.28 (2.86)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>45.21 (2.44)</td>
<td>0.00017</td>
</tr>
</tbody>
</table>

### Table 2: Comparison of actual mean measurements (C+P1+P2) with Modified Tanaka-Johnston (MTJ) predictions in males and females

<table>
<thead>
<tr>
<th>Arch</th>
<th>Gender</th>
<th>Methods</th>
<th>Mean(SD)</th>
<th>p-value</th>
<th>Prediction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maxilla</td>
<td>Male</td>
<td>Actual measurement</td>
<td>47.20 (2.65)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>MTJ</td>
<td>48.37 (2.11)</td>
<td>0.0001</td>
<td>overestimate</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>Actual measurement</td>
<td>45.60 (2.30)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>MTJ</td>
<td>45.95 (1.32)</td>
<td>0.1848</td>
<td>accurate</td>
</tr>
<tr>
<td>Mandible</td>
<td>Male</td>
<td>Actual measurement</td>
<td>47.28 (2.86)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>MTJ</td>
<td>48.61 (2.28)</td>
<td>0.0001</td>
<td>overestimate</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>Actual measurement</td>
<td>45.21 (2.44)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>MTJ</td>
<td>44.95 (1.32)</td>
<td>0.3776</td>
<td>accurate</td>
</tr>
</tbody>
</table>

### Table 3: Comparison of actual measurements (C+P1+P2) with Schirmer and Wiltshire (SaW) in males and females

<table>
<thead>
<tr>
<th>Arch</th>
<th>Gender</th>
<th>Methods</th>
<th>Mean(SD)</th>
<th>p-value</th>
<th>Prediction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maxilla</td>
<td>Male</td>
<td>Actual measurement</td>
<td>47.20 (2.65)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>SaW</td>
<td>47.64 (0.88)</td>
<td>0.1748</td>
<td>accurate</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>Actual measurement</td>
<td>45.60 (2.30)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>SaW</td>
<td>44.92 (1.33)</td>
<td>0.0114</td>
<td>underestimate</td>
</tr>
<tr>
<td>Mandible</td>
<td>Male</td>
<td>Actual measurement</td>
<td>47.28 (2.86)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>SaW</td>
<td>48.12 (1.04)</td>
<td>0.0198</td>
<td>overestimate</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>Actual measurement</td>
<td>45.21 (2.44)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>SaW</td>
<td>44.89 (1.28)</td>
<td>0.2990</td>
<td>accurate</td>
</tr>
</tbody>
</table>
The SaW method in our study statistically underestimated the widths of maxillary teeth in females, which is contrary to the findings by Burhan et al. Data from similar odontometric studies concur with our findings that SaW tables, an adaptation of the Moyers method, overestimate the actual mesiodistal dimensions of male permanent teeth in the mandible. Evidence of no difference in the prediction/actual size comparison was reported by Buwembo.

Both methods demonstrated accurate prediction of actual tooth sizes, some of the time. The MTJ equation overestimated the dimensions in 50% of the cases, and the Saw method, in 25% of the cases. In determining which method is the more accurate and applicable, clinical significance should be considered. Underestimation of tooth size results in inadequate space, an inability to accommodate permanent teeth, and thus poor orthodontic outcomes (occlusion and aesthetics); overestimation may result in unnecessary extractions. In clinical practice, underestimation of tooth sizes will have a comparatively poorer prognosis than overestimation. Generally, a difference of more than 2mm is clinically significant and can impact on the prognosis in orthodontic treatment.

It would seem, given the findings and clinical consideration, that the MTJ is better than the SaW method for the black patients in this South African sample.

LIMITATIONS

The study did not include a size disparity evaluation, such as the Bolton’s analysis. It may then have been possible that some teeth with unusual sizes could have been included in the sample, despite the preliminary evaluation of the study models.

Studies with larger sample size will have sufficient power to develop accurate and applicable prediction equations and tables. Regular updates of prediction tools should use present day data, in order to avoid underestimation due to secular trends.

CONCLUSION

The Modified Tanaka and Johnston equation has proven to be more accurate than the Schirmer and Wiltshire method in the prediction of the mesio-distal dimensions of the permanent premolars and canines in a sample of black South Africans. We recommend new equations and tables of data be developed based on adequately powered studies with representative sampling.

References

Intraoral repair protocols for fractured metal-ceramic restorations - Literature review

ABSTRACT:
Metal ceramic restorations are still widely used for prosthodontic rehabilitation of compromised teeth and in general are durable and long lasting. However, post-fitting complications in metal-ceramic crowns and fixed partial dentures do occur. One of the most frequently encountered complications in metal-ceramic systems is the fracture of veneering porcelain, becoming a dental emergency, especially when located in the anterior region. Replacing the entire restoration may not be the most practical solution. Intraoral repair of the fractured porcelain offers an easy and cost-effective alternative. The exact protocol for repair varies with the type of fracture that has occurred. This paper provides an overview of various methods of repairing different types of porcelain fractures with an aim to help clinicians manage these dental emergencies in a more effective and conservative manner.

Key words: fracture, ceramics, intra-oral repair, veneering porcelain

1. INTRODUCTION
Metal ceramic restorations are still widely used for the prosthodontic rehabilitation of compromised teeth.1 A single metal-ceramic restoration simultaneously offers significant strength and optimal esthetics, owing, respectively, to the metal framework and the veneering porcelain.2 These restorations also demonstrate longer life and durability in clinical service as compared with all-ceramic and fibre-reinforced composite restorations.3-7 Survival rates of 98% after 5 years, 97% after 10 years and 85% after 15 years of intraoral service have been reported in the literature.8 Post-insertion problems of metal-ceramic crowns and fixed partial dentures do occur. Complications can either be biological, including secondary caries, pulp pathologies and periodontal problems, or technical, such as loss of retention, ceramic delamination or fractures.9 A retrospective study calculated a mean complication incidence of 27% in fixed dental prostheses between periods of five and 14 years of service,10 while a 25% incidence of problems in metal-ceramic prostheses serving for more than five years has been reported.11 The most frequently encountered issue with metal-ceramic restorations is the fracture of veneering porcelain.12 A systematic review13 calculated a 34% frequency of porcelain chipping in metal-ceramic fixed dental prostheses over a period of three years. In contrast, another study reported a mean chipping rate of only 2.9% after a five year observation period,14 whilst a prevalence of chipping of between 5% and 10% over 10 years of use has also been claimed.15 Despite these differences in the reported rates, it can be concluded that porcelain fractures in metal-ceramic restorations are problems which will face most dentists, usually in emergency situations.

Fracture of the veneering porcelain does not always mean a failure of the restoration.16 However, the misadventure becomes a dental emergency if located in the anterior region of the mouth, compromising aesthetics.17 In such a clinical scenario, replacing the entire restoration may not be the most practical solution.18 Not only is the replacement time consuming and costly, but there is also the risk of damaging the prepared abutment while attempting to remove the restoration.19 Repairing the fractured porcelain intraorally, on the other hand, is relatively easy and offers a cost- and time-effective alternative to the patient and the dentist, adequately restoring both function and esthetics.20 Composite resins are the recommended materials for repairing porcelain fractures.16,21 The exact protocol for repair, however, varies depending upon the type of fracture that has occurred. This paper aims to provide an overview of various methods of repairing different types of porcelain fractures, albeit possibly in a temporary manner. The knowledge will help clinicians manage such dental emergencies in a more effective and conservative manner.

Acronyms
MDP: 10-methacryloyloxy-decyl dihydrogen phosphate
MPS: 3-Methacryloxypropyltrimethoxysilane
SIE: selective infiltration etching

1. Ayesha Aslam BDS, M.Sc., CHPE Senior Registrar, Department of Prosthodontics, AMC / AFID, National University of Medical Sciences (NUMS), Islamabad, Pakistan.
2. Syed Hammad Hassan BDS, FCPS, M.Sc. Med Edu Assistant Professor, Department of Prosthodontics, AMC / AFID, National University of Medical Sciences (NUMS), Islamabad, Pakistan.
3. Maleeha Nayyer BDS, M.Phil. Senior Lecturer, Department of Dental Materials, AMC / AFID, National University of Medical Sciences (NUMS), Islamabad, Pakistan.
4. Bilal Ahmed FCPS, BDS, FFD FRCSI-II (IRE), CMT, PhD Res. Professor, Department of Prosthodontics, Abottabad International Medical Institute, Abottabad, Pakistan.

Ayesha Aslam Department of Prosthodontics, AMC / AFID, National University of Medical Sciences (NUMS), Islamabad, Pakistan. dr.ayesha.aslam@hotmail.com
2. TYPES OF PORCELAIN FRACTURES

The type of porcelain fracture as well as the material involved will determine the repair protocol. Porcelain fractures have been classified in a number of different ways. A system for porcelain fractures, proposed by Friedman in 1998, describes three types of fractures (Figure 1):

i. Static Fracture – where a segment of porcelain fractures but remains in place
ii. Cohesive Fracture – fracture occurring within the body of porcelain; also known as chipping fracture
iii. Adhesive fracture – failure of the bonding interface between veneering and core porcelain or between porcelain and metal substrate

Another system is based on treatment need according to severity of the situation. The Heintz and Rousson classification has three grades:

1. Grade 1: Fractures requiring polishing only
2. Grade 2: Fractures requiring repair
3. Grade 3: Fractures requiring replacement

Although this classification system is practical and simple, it does not elaborate the criteria used to determine the severity of the fracture. In 2012, four criteria were added to the system to determine the need for replacement of the crown rather than the repair of fractured porcelain i.e. Grade 3 fractures:

i. When fracture extends into a functional area and repair is not possible
ii. When a recontouring attempt will badly alter the anatomic form
iii. When recontouring poses significant risk of thermal damage to the pulp
iv. When repair will result in poor aesthetics

Another classification system was put forward specifically for fractures occurring in metal-ceramic restorations. The authors suggested two categories simple fractures that involve only porcelain or complex fractures that result in exposure of metal substrate.

In this article, a combination of the classification systems proposed by Heintz and Rousson and Friedmann will be used.

3. INTRAORAL REPAIR OF FRACTURED PORCELAIN

3.1 Isolation of the Tooth

Regardless of the type of repair being undertaken, it is recommended that the involved teeth be effectively isolated. The ideal means of achieving field isolation is the use of a rubber dam, either conventional or paint-on, ensuring isolation at the gingival margin. This not only helps control moisture but also protects the hard and soft tissues of the patient from undue damage. Moisture control is mandatory when procedures involving composite resins are undertaken.

3.2 Fractures Requiring Polishing (Static Fracture):

The easiest way to repair a static fracture of porcelain is to polish the fractured surface thoroughly. This is done to minimize surface flaws that might lead to future failure. Polishing also eliminates any probability of accumulation of microorganisms on the fractured surface. This method of repair can also be employed for small chippings of porcelain that do not affect aesthetics or function in the posterior region. In case of larger defects, more polishing time is required with subsequent generation of heat. Overheating may lead to plastic deformation of the porcelain, and overheating of the pulp...hence the use of air-water cooling is recommended to prevent further fracture of the ceramic mass.

A variety of polishing techniques for porcelain have been described in the literature. Polishing kits are available that consist of diamond burs, abrasive rubber cups, felt wheels and polishing pastes. However, the published literature fails to recommend any standard surface-finishing protocol for porcelain and the choice depends largely on the preference of the clinician.

3.3 Fractures Requiring Repair:

3.3.1 Cohesive Fracture:

Cohesive fracture, occurring within the body of porcelain, can be repaired intraorally by either recementing the broken porcelain fragment with a resin cement or by restoring the broken porcelain with composite resin. In both situations, the restoration substrate first needs to be surface treated, as described below, to facilitate bonding between porcelain and the repair material. Despite this enhancement, the bond is not infallible.

Surface Modification:

The fractured porcelain surface is modified and a long bevel is created to facilitate optimal bonding and to achieve esthetically merging margins of porcelain and the repair material. If the broken porcelain chip is to be reattached, then that fragment must also be slightly beveled.

Surface Roughening:

The porcelain is then prepared for micromechanical bonding with the resin cement. The surfaces are first roughened with diamond burs, air abrasion using an intraoral sandblaster or by etching with Hydrofluoric (HF) acid. Table I summarizes the recommended roughening
protocols for different types of ceramics. Etching with 2.5 – 10% HF acid for 60 seconds is the recommended method for the chairside surface preparation of fractured silicate ceramics. However, the use of HF acid demands extreme caution as any spills could be hazardous to the soft tissues. Acidulated phosphate fluoride (APF) in a concentration of 1.23% has also been used for surface etching. It is safe to the oral tissues but an etching time of at least 6 minutes on both sides is required. Alternatively, effective surface roughening can be achieved by using an intraoral sandblaster. Air abrasion with 50 micrometer aluminium oxide particles at an air pressure of 2-3 bars sufficiently roughens and activates the surface, improving its wettability. The major drawback of air abrasion is its potential to generate small surface flaws, which might lead to crack propagation in ceramics. For pure silicate ceramics, air abrasion should not be used as HF acid etching provides adequate surface roughening. For oxide based ceramics, however, acid etching does not produce sufficient surface roughening. Zirconia, in particular, is resistant to etching due to its dense polycrystalline structure and lack of a glass phase. This makes air particle abrasion the method of choice for surface roughening of these materials. Surface damage in such cases can be minimized by decreasing the air pressure to 0.5 bars without compromising the results.

More recently, lasers have been used as an alternative to HF acid etching and air abrasion to achieve a roughened ceramic surface. Lasers such as CO₂, erbium: yttrium-aluminium-garnet (Er: YAG) and neodymium: yttrium-aluminium-garnet (Nd: YAG) have been used on ceramic surfaces to promote micromechanical adhesion. Nd:YAG laser melts the ceramic surface which, upon solidification, results in a surface with blisters. CO₂ laser, in contrast, results in the formation of conchoidal tears in the ceramic surface that aid in mechanical retention. Published literature suggests that Er: YAG laser surface roughening does not yield a durable resin-ceramic bond and although CO₂ and Nd:YAG lasers show better results, the effect is inferior to that achieved with HF acid. The generation of heat also contradicts the use of some lasers.

Surface Treatment:
Irrespective of the method used for roughening, the surfaces must also be treated with a coupling agent that would promote chemical bonding between ceramic and resin (Table II). Usually silane coupling agents are employed. These are bifunctional molecules, one end binding to the silanol group present in the silicate material through a condensation reaction while the other end binds to the resin via an addition polymerization reaction. Silane also increases the wettability of the surface, thereby allowing enhanced surface penetration of the resin. The most widely used silane is 3-Methacryloxypropyltrimethoxysilane (MPS).

<table>
<thead>
<tr>
<th>Type of Ceramic</th>
<th>Diamond Burs</th>
<th>HF Acid Etching</th>
<th>Sand blasting</th>
<th>Tribochemical Silica Coating</th>
<th>Lasers</th>
<th>Recommended Method</th>
</tr>
</thead>
<tbody>
<tr>
<td>Feldspathic Porcelain e.g. IPS Classic (Ivoclar Vivadent, Inc., Amherst, New York), VITA Mark II (Vident, Brea, California)</td>
<td>Effective</td>
<td>Most effective</td>
<td>Effective</td>
<td>Long term low stability</td>
<td>Low bond strength</td>
<td>HF Acid Etching</td>
</tr>
<tr>
<td>Lithium Disilicate based Ceramic e.g. IPS e.max Press, Ivoclar Vivadent, Inc., Amherst, New York</td>
<td>Effective</td>
<td>Most effective</td>
<td>Effective</td>
<td>Reduces bond strength</td>
<td>n/a</td>
<td>Low bond strength</td>
</tr>
<tr>
<td>Leucite-Reinforced Glass Ceramic e.g. IPS Empress, Ivoclar Vivadent, Inc., Amherst, New York</td>
<td>Effective</td>
<td>Low bond strength</td>
<td>Effective</td>
<td>Effective</td>
<td>Low bond strengths</td>
<td>Sand blasting with alumina particles</td>
</tr>
<tr>
<td>Glass-infiltrated Aluminium oxide Ceramic e.g. In-Ceram Alumina; Vita Zahnfabrik, Bad Säckingen, Germany</td>
<td>Ineffective</td>
<td>Ineffective</td>
<td>Effective</td>
<td>Most effective</td>
<td>Low bond strengths</td>
<td>Tribochemical Silica Coating</td>
</tr>
<tr>
<td>Bad Säckingen, Germany</td>
<td>Ineffective</td>
<td>Ineffective</td>
<td>Effective</td>
<td>Most effective</td>
<td>Low bond strengths</td>
<td>Tribochemical Silica Coating</td>
</tr>
<tr>
<td>Densely Sintered Aluminium Oxide Ceramic e.g. Procera All-Ceram, Nobel Biocare, USA, Inc., Yorba Linda, California</td>
<td>Ineffective</td>
<td>Ineffective</td>
<td>Effective</td>
<td>Most effective</td>
<td>Low bond strengths</td>
<td>Tribochemical Silica Coating</td>
</tr>
<tr>
<td>Zirconia based Ceramics e.g. In-Ceram Zirconia (Vita Zahnfabrik, Bad Säckingen, Germany), Cercon (Dentsply, York, PA, USA), Lava (3M ESPE, St. Paul, Minnesota)</td>
<td>Ineffective</td>
<td>Ineffective</td>
<td>Effective</td>
<td>Most effective</td>
<td>Low bond strengths</td>
<td>Tribochemical Silica Coating</td>
</tr>
</tbody>
</table>
Oxide ceramic materials lack the silanol groups and are unable to bond with silane.12 This can be overcome by ‘silicatizing’ them beforehand. The procedure involved is called tribochemical coating,63 made possible by the development of a chairside system “CoJet silicate-ceramic surface treatment system, 3M ESPE”.64 It involves aluminium oxide particles 30 micrometer in size doped with silica. When the surface is bombarded with these particles, it not only helps in roughening the ceramic but also incorporates silica into the ceramic.64,65 This silicatized ceramic is then able to bond with silane.

Alternatively, primers may be used for oxide ceramics. These are bifunctional phosphate monomers capable of bonding to oxide ceramics on one side and to the resin on the other side.66 One such monomer is 10-methacyrloyloxy-decyl dicydrogenphosphate (MDP). Primers are often added to resin materials67 which are then referred to as modified resins.12 They eliminate the need for separate treatment with primers. Panavia68,69 is one such MDP-containing resin luting cement, widely used for cementation of indirect restorations. Certain manufacturers provide a combination of silane and primers appear to be the most effective, owing to the chemical interaction between the hydroxyl groups of the oxide ceramic surface and the MDP phosphate esters.60

Another (laboratory) method to condition the surface of zirconia ceramics is selective infiltration etching (SIE),32,70 In SIE, the zirconia surface is covered with a glass-containing conditioner and heated to a temperature above the glass transition temperature of the conditioner. Once cooled to room temperature, the glass is rinsed in an acid bath. The process results in the formation of a new retentive surface, which when combined with a silane coupling agent, yields significant improvement in resin-zirconia bond.71,72 However, studies reporting the clinical efficacy of SIE need to be carried out before any recommendations can be made.

**Repair:**
In rare cases where the patient is able to salvage and produce the broken porcelain fragment, the fragment may be recemented using a resin cement. This, however, requires careful evaluation of the fragment to assess whether it is suitable for reattachment. The fragment and the porcelain substrate should both first be surface treated as described above. Recementation can then be achieved with the help of a resin cement. If the broken porcelain fragment is large, recementation is not recommended as the repair resin might impair the correct positioning of the fragment.75

Small chipped off parts can be built up using composite resins. Porcelain is first roughened and surface treated. The lost part is restored using a nanohybrid composite restorative material. A layering technique may be used to achieve optimum aesthetics,73 reducing the undesirable effect of the underlying metal shining through.

The third option to repair a cohesive porcelain fracture is to bond a new ceramic veneer on to the existing restoration. To achieve this, the existing restoration will have to be modified. The entire porcelain is removed with the help of rotary instruments and preparation margins are created.74 Impressions are recorded and sent to a laboratory for the fabrication of a porcelain veneer.75 The patient can be provided with temporary restorations until the next appointment. When the final restorations are received, they can be cemented using a resin based adhesive.

Cerec (CAD/CAM) system can be used to fabricate porcelain veneers at the chairside by trimming a single block of porcelain. A study on 617 Cerec veneers reported that CAD/Cam veneers fabricated with the Cerec system demonstrated a high survival rate of 94% after nine years and favourable clinical results.76 A number of materials can be used by the Cerec system including VITA Mark II (Vident, Brea, California), ProCad (Ivoclar Vivadent, Inc., Amherst, New York), In-Ceram Alumina and Spinell (VITA Zahnfabrik, Bad Säckingen, Germany).

### 3.3.2 Adhesive Fracture:
Adhesive failure of porcelain is the failure of the bonding interface. The failure may occur between the veneer layer and the core porcelain, or between the porcelain and the metal substrate resulting in exposure of the underlying metal framework.15 Intraoral repair of such a damage can be quite challenging as it may prove difficult for the clinician to mask the opaque core or framework colour with a ceramic veneer, thereby restricting the aesthetic outcome.12,74 Adhesive failure between core and veneer porcelain is managed in the same way as cohesive porcelain fractures. Where the metal substrate is exposed, the repair protocol needs to be slightly modified as described below.

**Surface Roughening for Micromechanical Bonding:**
The metal surface should be roughened using air abrasion as described for oxide ceramics. Etching with an acid is not sufficient since no currently available acid is capable of breaking metallic bonds.43 Undercuts can also be created to promote mechanical retention. Lasers have also been used as a means of etching the alloy surface. In comparison with air abrasion, alloy treatment with XeCl lasers showed improved bond strengths to composite resins17 whereas treatment with the Er:YAG laser did not yield effective surface roughening sufficient to promote the metal-resin bond.41,78

**Surface Treatment for Chemical Bonding:**
Similar to oxide ceramics, metal needs to be sili-

---

**Table II: Recommended surface treatment for various dental ceramics**

<table>
<thead>
<tr>
<th>Type of Ceramic</th>
<th>Silane Coupling Agent only (e.g. MPS)</th>
<th>Tribochemical Silica coating + Silane</th>
<th>Primer (e.g. MDP)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Silicate Ceramics</td>
<td>Recommended</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Oxide Ceramics</td>
<td>Ineffective</td>
<td>Recommended</td>
<td>Effective</td>
</tr>
</tbody>
</table>
To promote chemical bonding between the silica-coated metal and the repair material, a silane coupling agent must be used. Higher bond strengths between metal and resin have been reported if the metal is pretreated with silica. Alternatively, alloy primers can create a direct chemical bond between metals and resin, without the need for any silicatization. These primers contain carboxylic or phosphoric acid functional monomers which react with oxides present on the metal surface. Products combining both silane and primers are recommended for enhancing bond strength in the intraoral repair of base metal alloys bonded to ceramic restorations where the metal has been exposed.

A newer method to enhance bonding between metal and resin is silica-lasing. It involves coating the metal with an opaque porcelain slurry and irradiating it with a laser such as Nd:YAG or Er:YAG. Madani et al. reported that laser treatment of alloys in conjunction with air-borne particle abrasion yields significantly better bond strengths than laser treatment alone. However, silica lasing is a newer method and no appreciable data exists regarding its clinical performance.

Repair with Composite Resin:
To repair an adhesive fracture with composite material, a more opaque shade is selected for the first layers to emulate the dentin and to mask the colour of underlying metal, whereas lighter and more translucent shades are then utilized for surface restoration. The use of fibre-reinforced composites has been recommended for the repair of metal-ceramic crowns and fixed partial dentures as they offer increased fatigue resistance, thereby increasing the longevity of the repair.

A laboratory-fabricated composite or ceramic veneer can also be bonded to the facial surface of the damaged prosthesis with the help of a resin based cement. This is a more feasible clinical option if complete porcelain delamination occurs.

4. LONG TERM SUCCESS OF INTRAORAL REPAIR
Studies reporting the long-term success of repaired restorations are rather scarce. Özcan and Niedermeier reported an 89% survival rate over a mean period of 36.4 months of metal-ceramic restorations repaired intraorally with composite resins. Another study reported a 97.6% survival rate for metal-ceramic restorations repaired with composite after 3.5 years of clinical service. There exists a need for more in vivo studies with adequate follow-ups to evaluate the long-term success of such repairs.

5. RECOMMENDATIONS:
See figure below.

6. CONCLUSION:
Fractures involving the veneering porcelain of metal-ceramic restorations are routinely encountered in dental practice. The decision to repair or replace such a restoration revolves around a number of different factors including time and cost. While replacing the failed restoration may be the ideal treatment, it is not always practical. Repair of fractured porcelain should be attempted whenever possible. Repair protocols further vary depending upon the type of porcelain fracture. To ensure clinical
success and longevity of the restorations, a clinician must be well-versed with the various surface conditioning and surface treatments required to promote bond formation between the resin and the porcelain. This will help in optimizing the performance of metal-ceramic restorations as well as in achieving better patient satisfaction.

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Masticatory, temporal and buccal space infection in an immunocompromised patient: a case report

ABSTRACT

‘AIDS’ (Acquired Immunity Deficiency Syndrome) is a term used to describe the various clinical syndromes, specific opportunistic infections or malignancies that can occur with HIV infection. Oral manifestations of these diseases are common in people with Human Immunodeficiency Virus (HIV) infection and may be due to a decline in immune function. Hence patients with AIDS are subjected to recurrent, life threatening opportunistic infection. This is a case report of a 70yr old female who presented with infection of the right buccal, masticator and submandibular spaces. A routine blood report revealed seropositivity for HIV infection. She was treated with antibiotics and underwent an incision and drainage following hospitalization.

Key words: Masticatory, Submandibular space infection, Opportunistic infection

INTRODUCTION

The AIDS epidemic officially began on the 5th of June 1981. On that day Gottlieb et al. published the first report, titled “Pneumocystis Pneumonia - Los Angeles”. The AIDS pandemic began in India in the mid 1980s. In 1986, 12 commercial sex workers tested HIV positive in Chennai. Drug users, male homosexuals/bisexual contacts, receipt of blood transmission or blood products, multiple sex partners, perinatal transmission, transmission in Health care settings are the common sources accountable for the spread of HIV infection. For the dental practitioner, the significance of intraoral manifestations associated with HIV disease cannot be overstated.

AIDS is associated with several immunological diseases like T-lymphocyte deficieny, B-lymphocyte defects, and macrophage and neutrophil dysfunctions. Individuals with AIDS may have decreased salivary lactoferrin and IGA production, which may account for the high incidence of oral infection. With the decline in immune function, individuals with AIDS are subjected to recurrent, life threatening, opportunistic infection.

CASE HISTORY

A 70-year old female patient visited the Department with the chief complaint of pain and swelling in the right side of the face, first observed about a month previously. Initially it had been a small swelling that started in the area of the right angle of the mandible, accompanied by mild pain and low grade fever. The swelling had increased in size over time and the patient had sought treatment from a district clinic where she had been prescribed a week's course of antibiotics. (Figure1 and Figure2).

Acronyms

AIDS : Acquired Immunity Deficiency Syndrome
HIV : Human Immunodeficiency Virus

Pyogenic orofacial infections may originate in an odontogenic location. The majority are confined to local lesions, while in some cases they spread from the affected tooth along the anatomic spaces and occasionally advance to a site far from the initial infection. Significant morbidity or even death may occur in the cases that advance into the retropharyngeal, mediastinal, intra-cranial or intra-orbital spaces.

This paper highlights a case of a HIV infected female who presented with simultaneous infection of the right buccal, the masticator, and the submandibular spaces.

Figure 1: Showing Right Buccal and Submandibular space infection.

1. Dr. Chaithra Kalkur, Senior lecturer, Department of Oral Medicine & Radiology, Phone no: 9448815455. Email Address: chaithra.kalkur@gmail.com
2. Dr. Atul P Sattur, Professor and HOD, Department of General Dentistry, S.D.M College of Dental Sciences & Hospital, Email Address: atulsattur@gmail.com
3. Dr Venkatesh G Naikmasur, Professor, Department of Oral Medicine & Radiology, S.D.M College of Dental Sciences & Hospital, Email Address: drvnaikmasur@gmail.com
4. Dr. S J Govindraj, Reader, Department of Oral Medicine & Radiology, Century International Institute of Dental Science & Research Centre, Email Address: govindraj@gmail.com
On examination, a large, diffuse swelling was seen over the right side of the face, commencing at the midline of the scalp and extending inferiorly to 3cm to 4cm below the lower border of mandible. The skin over the swelling appeared stretched. On palpation the swelling was soft tender and fluctuant and a local rise of temperature could be discerned. The submandibular and cervical lymph nodes were enlarged bilaterally and were tender on palpation.

An intraoral examination was hampered by of the presence of trismus but it could be seen that the teeth revealed generalised stains and the presence of calculus, due to the twenty-year habit of chewing pan. The lower incisors showed attrition and the associated gingival tissue was inflamed, bleeding readily on probing.

Teeth 47 and 48 showed grade III mobility with periodontal pockets. Pus discharge was seen in the gingival sulci of teeth 47 and 48. (Figure 3)  

Figure 3: Restricted mouth opening due to involvement of Masticatory space.

The patient was advised to submit to a routine blood examination, which revealed that she was seropositive for HIV infection. The provisional diagnosis was an acute periodontal abscess affecting 47 and 48 with infection of the masticatory, temporal and buccal spaces.

The patient did not attend appointments for further and follow up management procedures.

DISCUSSION

Oral lesions are common in patients infected by the HIV virus and may indicate an impairment in the patient’s general health status and consequently predicting a poor prognosis. In some cases, the oral lesions are the first signs of infection and many of these HIV-positive patients present manifestations involving the maxillofacial region in all stages of the disease.

A strong correlation has been shown between HIV infection and various oral lesions including oral candidiasis, hairy leukoplakia, Kaposi’s sarcoma, non Hodgkins lymphoma and specific forms of periodontal disease.

Studies indicate that the majority of pyogenic oro-facial infections are due to odontogenic infections which are usually due to dental caries, pericoronitis, periodontitis, trauma to the dentition and the supporting structures, or complications from dental procedures. Second and third permanent molars are the teeth most commonly associated with pyogenic odontogenic infection.

Periodontitis is a disease attributable to multiple infectious agents and interconnected cellular and humoral host immune responses. However, it has been difficult to unravel the precise role of various putative pathogens and host responses in the pathogenesis of periodontitis.

Periodontitis in HIV-infected patients may resemble that of periodontitis in non-HIV-infected individuals, or may more dramatically present with profuse gingival bleeding or necrotic gingival tissue.

Periodontal diseases that are most strongly associated with HIV infection include linear gingival erythema, necrotizing ulcerative gingivitis and necrotizing ulcerative periodontitis.

Srivanitchapoom et al. revealed that the submandibular space was the most frequently involved in non-HIV infected patients, whereas the superficial masticator space was the most frequently affected in HIV infected counterparts. They further observed that immunocompromised patients tended to develop multiple space infections.

The masticator space is a distinct deep facial space, bounded by the superficial layer of the deep cervical fascia. It contains the ramus and posterior body of the mandible, and the four muscles of mastication, including the medial and lateral pterygoid muscles, the temporal muscle and the masseter muscle. Contracture of medial and lateral pterygoid muscles in response to inflammation causes trismus and precipitates pain in the temporomandibular joint.

The temporal space is posterior and superior to the masseteric and pterygomandibular spaces. Bounded laterally by the temporalis fascia and medially by the skull, it is divided into two portions by the temporalis muscle. The patient demonstrated swelling over the temporal area, posterior from the lateral aspect of the lateral orbital rim. Trismus is always a feature of this infection, resulting from infectious involvement of the temporalis muscle.
The buccal space contains the buccal pad of fat, Stenson’s duct and the facial artery. Infection results in a clinically marked cheek swelling. Submandibular space infections are commonly seen because odontogenic infections readily spread from the root apices of the second and third molar teeth which extend inferior to the mylohyoid line of muscle attachment. 3

For dental practitioners, the medical evaluation of patients with HIV is three-tiered. They are,
• Complications that may arise during dental therapy secondary to a patient’s immunologic, haemostasis and pharmaco-therapeutic status.
• Medical conditions that may directly interfere with provision of dental procedures.
• Patient’s prognosis for survival.

Dental providers need to continue to render dental care to all patients, regardless of their social or religious background or sexual orientation. The provision of dental care for HIV –infected individuals is similar to that of non-infectious patients, with attention being paid to appropriate infection control measures.2

CONCLUSION:
Patients with HIV infection are at an increased risk for rapidly progressive severe periodontal disease. Dental management requires early identification of lesions and prevention of further periodontal deterioration. The treatment of patients infected with HIV is a demanding discipline within both medicine and dentistry.

References

Source of support: Nil.
Conflicts of Interests: None declared

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.

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5 Enter your multiple choice answers. Please note that you have two attempts to obtain at least 70%.
6 View and print your CPD certificate.
A 7-year old boy presented with a longstanding swelling in the region of the left angle and body of the mandible, (Fig.1) with associated tumour like nodules present within the oral cavity.

INTERPRETATION
The cropped pantomograph (Fig.2) showed numerous multilocular radiolucencies, bony expansion and excess tooth eruption. The coronal CT scan (Fig.3) confirmed the bony expansion of the lesion as well as the involvement of the pterygoid plates on the left side (red arrow) denoting an aggressive lesion which has spread beyond the mandible into the pterygoid muscle. On the MRI scans there was a high intensity signal in all sequences in both parapharyngeal fat spaces, indicative of slow venous flow in the severely congested pterygoid plexuses. Figure 4 shows the tortuous nature of the flow-voids (arrows) visible on the sagittal projection on one of the MRI scans, indicative of the vascular nature of the lesion. A final diagnosis of central haemangioma was made. The facial bones are a common site for this very rare lesion. According to Langlais et al the literature is inconsistent regarding the various types of vascular lesions. They believe there are two groups of lesions: haemangioma and AVM’s. Haemangioma may have been present since the first year of life, whereas arteriovenous malformations (AVM’s) may develop in a teenager, possibly after trauma. Facial asymmetry, especially over the mandible may result from swelling or hypertrophy. The skin or mucosa may be bluish, purplish, or reddish (Fig.5&6). Central haemangioma may yield blood readily on aspiration, whereas the pressure of the central AVM may drive the plunger out of the syringe. Lamberg et al (1979) discovered that 11 deaths occurred during treatment of central haemangioma. According to them extraction of teeth extending into a hematangiomatous region seems to be the most common cause of fatal bleeding, and the tooth was a molar in all cases. In 8 of the 11 cases the operator was aware of or suspected a haemangioma. They also stressed the importance of suspecting this lesion in young patients with gingival bleeding and mobile teeth. In conclusion a certain clinician made the following statement regarding the presence of a haemangioma “When you press on a tooth in the presence of haemangioma it feels like pressing on a soccer ball”.

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What’s new for the clinician
Summaries of and excerpts from recently published papers

1. Topical application of morphine for wound healing and analgesia in patients with oral lichen planus: is it effective?


Oral lichen planus (OLP) is a chronic inflammatory disorder of the oral mucosa. Prevalence figures are low and vary across different populations. However, more than 60% of patients are women between 30 and 60 years of age.\(^1\) Approximately 1-5% of patients with OLP present with cutaneous lesions. In contrast, 77% of patients with skin lesions also have oral lesions.\(^1\) Asymptomatic variants (reticular and plaque-like types) and symptomatic variants (erythematous, formerly atrophic; erosive; ulcerative; and, rarely, bullous types) may appear singly or in combinations.

Lichen planus is believed to result from an abnormal T-cell-mediated immune response in which basal epithelial cells are recognized as foreign because of changes in the antigenicity of their cell surface. The cause of this immune-mediated basal cell damage is unknown.

As OLP is a chronic, often persisting disorder of immunologic background, treatment is mainly focused on pain reduction and healing of lesions. Cause-related therapies are not available to this date. Topical application of corticosteroids (clobetasolpropionate, triamcinolone, prednisone, and dexamethasone) is the treatment of choice.\(^1\) However, in long-term use, these drugs may, even though rarely, result in systemic adverse endocrine effects (Cushing’s disease, buffalo hump, moon face), adverse metabolic effects (e.g., hyperglycemia, osteopathy), ocular manifestations (e.g., cataract, glaucoma), and electrolyte imbalance (e.g., oedema, hypocalcemia).\(^1\) In view of this, opioids have been considered mainly for topical application as their effectiveness has been demonstrated on wound healing and pain relief. Zaslansky and colleagues (2018)\(^1\) reported on a clinical trial that sought to test the topical application of morphine hydrochloride in patients with symptomatic erosive and/or ulcerative OLP and its effect on healing. The reduction of pain and safety were secondary outcomes.

**MATERIALS AND METHODS**

This was a single-centre, prospective, double-blind, placebo-controlled, randomized, three arm, phase II study. Patients were eligible if their diagnosis of erosive and/or ulcerative OLP was confirmed by a pathologist; if they were level I–II according to the American Society of Anaesthesiologists (ASA) classification (healthy or minimal systemic disease requiring no treatment), 18–75 years old, either sex, and deemed able to provide assessments of their pain and side effects. Patients were excluded if they had a condition of alcohol abuse or addiction (opioids and/or benzodiazepines), known hypersensitivity to morphine, major renal or hepatic dysfunction, pregnancy or lactation, sleep-apnea-syndrome, diabetes or had participated in other studies. Patients who were included in the trial were then taught how to use a pain diary and how to assess opioid-related side effects using an 11-point item numerical rating scale (NRS) which ranged from score 0 (no side effects) to 10 (worst possible side effects).

After clinical examination of the oral cavity of each of the included patients, the size of the lesion was measured, photographed and scored using the Thongprasom clinical criteria score. Severity of pain was assessed using the NRS. Patients were then assigned to one of three treatment groups using a random log randomization chart and were given a bottle with sufficient medication to last for the duration of the study. Bottles contained 20 ml of either 40 mg (0.2%) or 80 mg (0.4%) morphine hydrochloride dissolved in glycerol 85%. Placebo consisted of 10 mg caffeine dissolved in glycerol (85%). The initial dose of test substance was applied to the lesion(s) by the investigator. Patients were discharged an hour later after confirmation that they experienced no adverse effects.

At home (days 2-5), patients self-applied the test substance three times daily for 5 days. Patients were asked to leave the test substance in the mouth for one min and then spit it out (‘swish and spit’). They were requested to refrain from eating, drinking, or smoking for an hour after application of the test substance. In the event of unrelieved pain, patients were permitted to apply one cm of Volon A Haftsalbe® (=‘rescue medication’) onto the lesions, up to three times a day. As the triamcinolonaacetomid in Volon A Haftsalbe® was expected to have an effect on healing, patients taking this medication were excluded from the analysis regarding healing, though not regarding pain.

Monitoring the well-being of patients was carried out by a once daily telephone interview. Patients reported the intensity of pain during this call, whether they experienced side effects and if they had used the rescue medication.
After five days (day six), patients returned to the clinic; their lesions were examined clinically and photographed. Photographs were coded and assessed by a blinded examiner and participation in the study ended.

The primary outcome was the extent of healing of lesions assessed on the return visit.

Secondary outcomes were as follows: (1) pain scores (spontaneous) during the five days of treatment; (2) presence and severity of central (nausea, vomiting, sedation, constipation) and local (burning, dry mouth) side effects; (3) whether patients required ‘rescue medication’ for treatment of pain, the dose, and when it was taken; and (4) total intake of test substance.

RESULTS
Of the 123 OLP patients screened, only 45 fulfilled the inclusion criteria although there had been a recruitment drive of almost three months. Patients were randomized into the three study groups, n = 15 received 0.2% morphine; n = 16 received 0.4% morphine and n = 12 received placebo. Forty-three patients were included in the final analysis. Two patients were excluded because they discontinued intervention.

In the 0.2% morphine group (n=15), there were 12 females and 3 males with an mean age of 58 years, SD 10 years; the 0.4% morphine group (n=16) had 12 females and 4 males with the mean age of 60 years, SD 14 and the placebo group (n=12) had 10 females and 2 males (mean age 65 years, SD 8).

Clinical variants of OLP at the beginning of the study included the following: erosive (n = 23, 53.5%); ulcerative (n = 6, 14.0%), and erosive/ulcerative (n = 14; 32.6%). The most frequently affected location was the gingiva (55%) followed by buccal mucosa (32%) and dorsum of tongue (5%).

There were no differences between the groups with regards to extent of healing (P < 0.686). For the Pain scores, there were no differences between the groups (P < .530) at any time during the observation period. Scores were low (under 3/10) in all groups. None of the patients reported moderate or serious adverse events. None of the patients used the rescue medication.

There were no differences in use of the test substance according to amounts (g) remaining in the bottles at the end of the study (P = 0.679).

CONCLUSIONS
The results of this trial suggest that locally applied morphine hydrochloride in a solution of 0.2 and 0.4% used in a period of one week has no clinically relevant benefit compared to placebo treatment based on caffeine and glycerol in OLP.

Implications for practice: This trial provides evidence that morphine should not be considered as a treatment option for OLP. Corticosteroids are still the gold standard for providing symptomatic relief for patients with OLP.

Reference

2. A Comparison of root canal cleanliness using different irrigation activation systems.


The success of any root canal treatment depends on good biomechanical preparation. Irrigation is an essential part of root canal debridement to ensure cleaning in areas that were not touched by mechanical instrumentation. These irrigants must have direct contact with the entire canal wall for effective action. Different techniques and irrigant delivery devices have been proposed to increase the effect of chemical disinfection within the root canal system and to improve canal cleanliness following mechanical instrumentation. However, thorough cleaning of the entire root canal system is still challenging as mechanical instrumentation of the canal walls always generates a smear layer.

Recently, EDDY, a sonic powered irrigation activation system made of flexible polyamide with a size of 25.04, was introduced (VDW, GmbH, Munich). According to the manufacturer, it allows an efficient cleaning of complex root canal systems without the limitations of ultrasound-activated devices. EDDY is activated to a frequency range of between 5000 and 6000 Hz by an air-driven handpiece (Air Scaler). According to the manufacturer, the instrument is claimed to create a three-dimensional movement that triggers “cavitation” and “acoustic streaming”—two physical effects which up to now have only been caused by passive ultrasonic irrigation (PUI) and to which have been attributed the improved cleaning efficiency of PUI. EDDY is a non-cutting, sterile single-use instrument. Another sonic activation system available is the EndoActivator (EA) system, which is a cordless sonic handpiece that activates highly flexible polymer tips in the range of between 33–167 Hz. Hence, with regard to the frequencies, these sonically activated tips differ at least by factor 36 and it is reasonable to assume that this difference may have an impact on the clinical performance of these two sonic devices. However, there are no published data regarding the efficiency of the sonic activation system EDDY. Urban and colleagues (2017) reported on an in vitro study that sought to assess and compare the efficacy of EDDY with manual irrigation (MI), EndoActivator (EA), and passive ultrasonic irrigation (PUI) regarding removal of debris and smear layer in the coronal, middle, and apical thirds of straight root canals. The null hypothesis tested was that all activation systems perform equally regarding removal of debris and smear layer.

Acronyms
EA: EndoActivator
ISO: International Standardisation Organisation
MI: manual irrigation
PUI: passive ultrasonic irrigation
MATERIALS AND METHODS
A sample of 58 single-rooted mandibular premolar teeth extracted for periodontal and orthodontic reasons were used in the study. Two digital radiographs were taken in a bucco-lingual and mesio-distal direction to verify root canal anatomy presenting only one central root canal. Inclusion criteria were permanent teeth, intact apices, no previous root canal treatment, or extensive coronal restoration. Exclusion criteria were oval canals (long versus short diameter ≥1.5) root caries, cracks, and fracture lines.

The crowns were not removed in order to preserve the normal trajectory of irrigation instruments. Following access cavity preparation, patency was checked using a size 10 C-Pilot file (VDW GmbH, Munich). Simultaneously, root canal working length was visually determined using a stereomicroscope by subtracting one mm from the measurement taken when the file just passed the foramen major. Additionally, an apical gauging was performed to verify comparable canal diameters and foramen sizes of all samples. This was done using ISO tapered silver points with sizes 20, 25, and 30. Only teeth with an apical size of about size 25 were included in this study.

Prior to canal preparation, all apices were covered with wax in order to guarantee a closed system. Root canal preparation was performed by only one experienced operator with Reciproc R40 instruments using the VDW-Silver motor and the setting “RECIPROC ALL.” During instrumentation, all root canals were irrigated after each preparation cycle with 2.5 ml of 3% sodium hypochlorite with a 30-g open-ended needle inserted into the root canal without binding and not deeper than one ml short of the working length. One preparation cycle consisted of three pecks with an amplitude of not more than three mm. When the instrumentation was completed, activation was performed in the following activation procedures with a total volume of 12ml of NaOCl and three activation cycles (4ml of NaOCl per cycle) of 30s:

1. Manual irrigation (volume 12 ml; irrigation time 90 s)
2. EndoActivator (166 Hz, size 25.04)
3. PUI (Irri S size 25; VDW-Ultra device; VDW; setting 30% resulting in about 30 kHz)
4. EDDY (6000 Hz, size 25.04)

One group (n = 10) served as control
5. Negative control group (no irrigation)

The tips of all activation devices as well as the irrigation needle were placed one mm short of the working length without binding. Specimens were prepared for SEM evaluation. After screening of the entire canal wall, three photomicrographs of each specimen were taken to visualize the coronal, middle, and apical portions. Canal areas that were not instrumented were not assessed, and the apical third regions beyond the working length were excluded. For each portion of the canal, always the area showing the greatest amounts of debris and smear layer was selected. A total of 348 images (58 samples × 3 portions, apical, middle, and coronal) were analysed twice at an interval of 48 hours by two blinded and experienced observers who underwent a training process with reference to the scoring system of the SEM evaluations. The following scoring system was used:

- Score 1: clean canal wall, only very few debris particles
- Score 2: few small conglomerations; less than 25% of the canal wall covered
- Score 3: many conglomerations; 25% to 50% of the canal wall covered
- Score 4: 50% to 75% of the canal wall covered
- Score 5: complete or nearly complete (more than 75% of the canal wall) covering of the canal wall by debris

Scoring of debris was performed using a ×200 magnification. Scoring of smear layer was performed using a ×1000 magnification, and the scores were recorded:

- Score 1: no smear layer, orifices of dentinal tubules patent
- Score 2: small amount of smear layer, some open dentinal tubules
- Score 3: homogenous smear layer along almost the entire canal wall, only very few open dentinal tubules
- Score 4: the entire root canal wall covered with a homogenous smear layer, no open dentinal tubules
- Score 5: a thick, homogenous smear layer covering the entire root canal wall

RESULTS
None of the activation methods completely removed debris and smear layer. With regard to smear layer and debris removal, significant differences between the control group and all experimental groups were obtained (p < 0.001).

Canal cleanliness increased significantly from the apical to the coronal portion of the root canals (p < 0.01). Manual irrigation removed significantly less debris compared with all other groups (p < 0.001). Further significant differences between groups were not obtained (p > 0.05).

Significantly more smear layer was found in the apical portion compared with the middle and coronal thirds of the root canals, independent of the activation method (p < 0.05). PUI and EDDY removed significantly more smear layer than MI (p < 0.01).

Conclusions: Under the conditions of this study, all activation methods were superior compared with manual irrigation regarding debris removal. EDDY and PUI obtained significantly better smear layer scores compared with manual irrigation.

Implications for practice: This study has provided evidence that manual irrigation methods used during endodontic treatment can be considered as outdated when compared with the more modern and effective alternatives.

Reference

Professor V Yengopal, PhD. Community Dentistry, Head, Department of Community Dentistry, School of Oral Health Sciences, Faculty of Health Sciences, University of the Witwatersrand, Johannesburg. Email: Veerasamy.yengopal@wits.ac.za
To pen or to probe
Prescribing versus treating, how to decide

SA&DJ Feb 2018, Vol 73 no 1 p53
Leanne M Sykes, William G Evans, Glynn Buchanan, Nichola Warren, Nelson Fernandes

"The pen is mightier than the sword" is an adage coined by Edward Bulwer-Lytton in 1839, indicating that communication (particularly written language), is a more effective tool than direct violence. He was making a statement to members of his own society suggesting that the administrative powers or advocacy of an independent press could be a more effective communication tool than direct violence. This could be applied to Dentistry in 2017, where the pen refers to the writing of prescriptions and the violent sword is replaced by a (hopefully more gentle), probing clinician – both literally and figuratively. If that is so, then one needs to investigate the ethics of the current trend and laissez faire attitude with which many dentists write out prescriptions.

INTRODUCTION

The question of wellness was explored in Part 15 of this series, based on the definition of health proposed by the World Health Organization (WHO). However that description is now over 70 years old and does not fully address the more holistic approach to oral health, first proposed by Dolan in 1993, which defines oral health as "having a comfortable and functional dentition that allows individuals to continue their social life". Others have added that "It is the ability to chew and eat the full range of foods native to the diet, to speak clearly, to have a socially acceptable smile and dento-facial profile, to have a fresh breath and to be comfortable and free from pain". In 2016 the FDI proposed that "oral health is multi-faceted and includes the ability to speak, smile, smell, taste, touch, chew, swallow and convey a range of emotions through facial expressions with confidence and without pain or discomfort and disease of the craniofacial complex". For most clinicians and patients, this may have seemed like an unrealistic and unattainable ideal. It was thus later modified by adding the proviso that: "It (sic. Oral health) is influenced by the individual’s changing experiences, perceptions, expectations and ability to adapt to circumstances".

In the endeavour to secure oral health for a patient the dental practitioner may invoke any of the many ways of treating pain and disease, but a frequent choice is with medication – in particular antibiotics and analgesics. This may be prior to, in place of, in conjunction with, or after some form of physical intervention. A poignant question is: which is the best treatment for each situation, i.e. what to do and when?

WHY DO DENTISTS PRESCRIBE?

In 2001, Daily and Martin conducted research on antibiotic prescribing habits in an emergency dental clinic in the United Kingdom. They found that 74% of patients diagnosed with pulpitis were issued with a prescription for antibiotics without any form of active treatment being carried out. Similarly Tulip and Palmer (2008) noted that more than 50% of patients who presented to an emergency clinic with dental conditions were treated with the provision of antibiotics alone with no follow-up management to address the aetiology of their pain. In the UK, dentists prescribe 10% of all antibiotics dispensed from community pharmacies, and often do so in contradiction to clear clinical guidelines. In Cananda, dental prescribing increased by 62.2% from 1996 to 2013.

It is disturbing that a similar cross sectional survey conducted in the United Kingdom found the prevalence of antibiotic prescribing to remain alarmingly high despite the universal concern about the increase in antibiotic resistant organisms.

In over 70% of cases, clinicians offered no operative therapy in conjunction with the antibiotic prescription. Of greater concern is the report that a further 12.6% of dentists in this study, and almost 65% in an Indian survey, prescribed antibiotics for the treatment of diagnosed irreversible pulpitis where the patients had no systemic symptoms and where evidence suggests that preoperative antibiotic therapy does little to reduce the pain in these situations.

Investigations into prescribing habits have revealed that dentists prescribe antibiotics more often when under time pressure, where they have difficulty in making a definitive diagnosis, or if treatment needs to be delayed. Other reasons for high prescription rates could be personal, such as to avoid working late or going out to see an after-hours emergency patient. Practitioners often consider these times inconvenient because the emergency procedures may be complex, and there is the risk that the patient may not pay for services rendered. Providing a prescription may be a way to “get rid” of the patient quickly and easily. More alarming instances are where the dental practitioner succumbs to patient demands or expectations for medication; or where patients report to be in pain but state that they are unable to get to the consulting rooms. Prescriptions may be made out for friends, colleagues or family members based on verbal conversations without the practitioner ever seeing the person. This is tantamount to relying on an often-untrained third party to self-diagnose and dictate treatment.

Ethical considerations in deciding whether to treat family and friends are complicated. On the one hand, the patient’s right to choose the dental practitioner of his or her choice must be considered, respecting their right to autonomy. However, on the other hand the question of the potential impairment of a clinician’s objectivity in making sound clinical treatment decisions must be considered. Kling (2015) advised that one should: “refrain from treating family and friends, except in emergency situations and where no other doctors are immediately available”.

ETHICAL ISSUES AND IMPLICATIONS

Telephonic consultations do not allow for any form of physical examination of the patient, or the use of diagnostic aids. The diagnosis is based purely on the patient’s description and interpreta-
tion of their own symptoms. This is subjective, depends on their degree of pain, often over-exaggerated or distorted, and may be blatantly dishonest if the patient’s main motivation is to obtain medicines. Who will be accountable for an incorrect diagnosis, adverse side effect of medication or worse, complications resulting from the lack of intervention?

The dental community has been implicated in the over-prescription of antibiotics, a practice which has contributed to the universal problem of antimicrobial resistance (AMR). There are two ways in which AMR occurs. Firstly, microorganisms can adapt and change after being exposed to antimicrobial drugs (such as antibiotics, antifungals, antivirals, antimalarials, and anthelmintics). Secondly, bacteria of different taxa can work mutualistically to form a community that can be up to one thousand times more tolerant to environmental stress, including antibiotics, than individual colonies or cells to antimicrobials.

AMR occurs naturally over time through genetic changes; however, the misuse and overuse of antimicrobials accelerates this process. “These organisms are colloquially referred to as “superbugs”, due to their resistance to the medicines routinely used to treat them, and result in persistent infections, and increased risk of spread to others. This is a global public health issue that threatens future effective prevention and treatment of an ever-increasing range of infections. The implications are vast. It may compromise the success of major surgery and cancer chemotherapy, jeopardise organ transplantation and diabetes management, increase the cost of health care for patients by prolonging the duration of illness and hospital stay, and may necessitate additional tests, use of more expensive drugs and even intensive care. In addition, patients infected by drug-resistant bacteria tend to have worse clinical outcomes, consume more health-care resources and have a higher risk of death from their infections.”

Other examples of antimicrobial misuse are when they are prescribed for people with viral infections like colds and flu, and when given as growth promoters in animals and fish which later results in resistant organisms being found in people, animals, food, and the environment. Two main global concerns are the increase in multi-drug resistant TB. The WHO estimates that, in 2014, there were about 480 000 new cases of multidrug-resistant tuberculosis (MDR-TB), yet only about a quarter of these (123 000) were detected, reported and treated. This form of TB requires much longer treatment, which is often less effective than in non-resistant TB. The second is the widespread resistance to first-line drugs used to treat infections caused by *Staphylococcus aureus*, a common cause of severe infections in health facilities and the community. People with methicillin-resistant *Staphylococcus aureus* (MRSA) are estimated to be 64% more likely to die than people with a non-resistant form of the infection. In addition, the fact that the antibiotics of choice in dentistry are amoxicillin in combination with metronidazole, and erythromycin in cases of penicillin allergy, could have contributed to the widespread resistance to these drugs.

Local measures should always be the first line of treatment regarding any dental infection. Some clinical situations which require antibiotic cover include oral infections presenting with systemic spread such as lymphadenopathy and trismus, systemically elevated body temperature indicating pyrexia, persistent chronic sinusitis with purulent discharge, and facial cellulitis, which can have fatal consequences if left untreated.

Localized dental infections requiring systemic antibiotic administration, often in conjunction with localized treatment, include periodontal abscesses, necrotizing ulcerative gingivitis (NUG), and pericoronitis.

A useful table, adapted from “Drug prescribing for Dentistry: Dental clinical guidance”, is included below:

Prophylactic antibiotic coverage for the prevention of infective endocarditis (IE) is warranted in particular circumstances. This is true for patients who are at a high risk of developing IE, including: patients with a previous history of IE, patients with a prosthetic heart valve, those having undergone cardiac valve repair, and those with cyanotic congenital heart disease.

Penicillin still remains the drug of choice for treating infections of dental origin. Clindamycin has been shown to be effective in patients who are allergic to penicillin, whilst metronidazole is most effective against anaerobic infections.

If antibiotic treatment is necessary, the recommended dosages are as follows: 1g amoxicillin/clavulanic acid bd for 3-5 days, or 150mg clindamycin qid for 5 days if allergic to penicillin for dental abscesses; 200mg metronidazole tds for 3 days for NUG/peri-

Table 1
coronitis; and 500mg amoxicillin tds for 7 days, or 100mg doxycycline bd for 8 days for sinusitis.\textsuperscript{18,20}

CONCLUSION

The inappropriate prescription of medication, in particular antibiotics by dental professionals, may be contributing to the rapidly increasing universal problem of antibiotic resistance. In addition, the unnecessary use of medication is costly, potentially harmful and may even prolong the time of pain and infection. At worst, prescribing rather than treating may merely mask the problem, and could necessitate more extensive interventions at a later date. Alerting and re-educating practicing clinicians, as well as targeting undergraduate dental students and therapists who have prescribing privileges, is an important future step in minimising antibiotic consumption.\textsuperscript{21} Perhaps broader training at dental undergraduate level should be instituted, specifically with regards to the prescribing of medications.\textsuperscript{22}

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Dr. Moreniko Ukpeng, Nigeria
Dr. Anna Maria Vierrou, Greece

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**Sharks, shark cartilages and shark teeth:** A collaborative Africa-USA study to attempt to induce “Bone formation by autoinduction” in cartilaginous fishes.

1. The process by which blood vessels “shape, pattern and induce the multistep cascade of the induction of bone formation” is described as:
   a. Haematogenetic osteogenesis
   b. Osteomorphological angiogenesis
   c. Osteogenesis in angiogenesis
   d. Angiomorphological osteogenesis

2. The polyphyodont dentition of the shark is fused to the cartilaginous jaws and teeth are shed through a process of dentinoclastogenesis.
   a. True
   b. False

3. The dentine of a shark tooth (variably termed enameloid, mesodermal enamel or durodentin) is covered by a calcified layer of ectodermal enamel.
   a. True
   b. False

4. The authors suggest that the induction of chondrogenesis by the heterotopically implanted coral-derived macroporous bioreactor is the most relevant finding.
   a. True
   b. False

5. The research proved that the DNA of the Selachians retains the developmental memory of the osteoinduction programme.
   a. True
   b. False

**Third molar impaction in a cross section of adult orthodontic patients**

6. Third molar impactions are implicated in the following
   a) Odontogenic infections
   b) Neoplasms
   c) Early onset of dental crowding
   d) a and b only
   e) a, b and c

7. More severe mandibular third molar impactions are more likely to be observed in Class II malocclusions.
   a. True
   b. False

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**Knowledge, attitude and practices of alcohol and smoking among undergraduate oral health students at a South African University.**

8. The majority of the participants (92%) felt that professionals have a role in giving advice to patients about cessation of any tobacco habit.
   a. True
   b. False

9. The study showed that students accumulated sufficient knowledge of the ill effects of alcohol on cancer, hypertension and diabetes.
   a. True
   b. False

**Comparison of two methods of predicting mesiodistal widths of permanent canines and premolars in a sample of black South Africans**

10. The study failed to demonstrate any significant differences in the size of teeth between male and female data.
   a. True
   b. False

11. In clinical practice, underestimation of tooth sizes will have a comparatively poorer prognosis than overestimation.
   a. True
   b. False

**Intraoral repair protocols for fractured metal-ceramic restorations - Literature review**

12. The most effective method for surface treatment of zirconia based ceramics is:
   a. Diamond burs
   b. Acid etching
   c. Lasers
   d. Tribochemical silica coating

13. The major drawback associated with air abrasion of ceramics is:
   a. Heat generation
   b. Generation of surface flaws
   c. Hazardous to soft tissues
   d. Results in ineffective roughening

**Masticatory, temporal and buccal space infection in an immunocompromised patient: a case report**

14. The periodontal diseases most often concurrent with HIV infection include linear gingival erythema, necrotizing ulcerative gingivitis and necrotizing ulcerative periodontitis.
   a. True
   b. False
15. The root apices of the second and third molar teeth extend inferior to the mylohyoid line of muscle attachment, leading to the frequency of odontogenic infection spreading into the sublingual space.
   a. True
   b. False

Maxillo-facial and Oral Radiology

16. Haemangioma may present in the first year of life.
   a. True
   b. False

17. Can the extraction of teeth with roots that extend into a haematogenous region lead to fatal blood loss?
   a. True
   b. False

What’s new for the clinician – summaries of recently published papers

18. In the Zaslansky et al study, pain scores were significantly higher in the placebo group.
   a. True
   b. False

19. The Urban et al root canal study is an example of a laboratory based investigation.
   a. True
   b. False

20. In the Urban et al study, the EDDY method removed significantly less debris than PUI.
   a. True
   b. False

ETHICS February 2018.

21. The frequency in the United Kingdom of the prescribing of antibiotics without accompanying clinical treatment has dropped considerably in response to concern about the increase in antibiotic resistant organisms.
   a. True
   b. False

22. The only reason why dentists routinely prescribe antibiotics is because they are working under pressure and must save time.
   a. True
   b. False

23. The advice of seasoned experts is that practitioners should wherever possible refrain from treating family members.
   a. True
   b. False

24. The dental practitioner who routinely prescribes antibiotics may be contributing to the escalation of bacterial strains resistant to the drugs.
   a. True
   b. False

25. The prescribing of antibiotics over a telephone diagnosis is neither ethical nor sound practice.
   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.

Online CPD in 6 Easy Steps

1. Go to the SADA website www.sada.co.za.
2. Log into the ‘member only’ section with your unique SADA username and password.
3. Select the CPD navigation tab.
4. Select the questionnaire that you wish to complete.
5. Enter your multiple choice answers. Please note that you have two attempts to obtain at least 70%.
6. View and print your CPD certificate.
World Oral Health Day - 20 March 2018
Say Ahh – Think Mouth, Think Health

As defined by the FDI, ‘Oral health is multi-faceted and includes the ability to speak, smile, smell, taste, touch, chew, swallow and convey a range of emotions through facial expressions with confidence and without pain, discomfort and disease of the craniofacial complex’. Therefore good oral health is an essential component of general health and quality of life.

The global burden of oral disease remains a paradox, affecting 3.9 billion people worldwide. Oral conditions, such as tooth decay (dental caries), gum disease and oral cancer, are among the most common and widespread diseases of humankind. Dental caries in children is five times more common than asthma and seven times more common than hay fever.

Oral disease which is largely preventable remains a major public health problem, sharing common risk factors with non-communicable diseases (NCDs), which are the leading cause of death around the world. Yet, the response to the burden of oral disease remains slow, with untreated tooth decay alone impacting almost half (44%) of the world’s population. South African studies have shown that 60% of primary school children had dental caries, and 80% of the caries remains untreated. Left untreated dental caries results in pain, disability and a poor quality of life

Achieving optimal oral health thus constitutes a major public health challenge that requires strong advocates, who are committed to promoting oral health initiatives emphasizing disease prevention measures, and integrating oral health into general health and NCD policies.

WHAT IS WOHD?
Celebrated each year on the 20th of March, WOHD is the largest global awareness campaign on oral health. It was launched by FDI in 2007 and is the culmination of year-long activities dedicated to raising global awareness on the prevention and control of oral diseases. Each year, WOHD focuses on a specific theme and reaches out to the general public, oral health professionals and policymakers, who all have a role to play in helping reduce the disease burden.

WHY IS WOHD IMPORTANT?
As the largest global awareness campaign, WOHD brings to the fore the burden and consequence of oral disease, which is still not a priority issue in most countries and remains low on the global health and development agenda.

Moreover WOHD, emphasises the link between oral health and general health since poor oral health share common risk factors (tobacco use, harmful use of alcohol and unhealthy diets) and the same social determinants with most NCDs, such as diabetes, cardiovascular disease, respiratory disease, and gastrointestinal and pancreatic cancers. Therefore a comprehensive and integrated preventative and curative response would yield better results.

Despite the unacceptably high disease burden, oral health is still not considered a priority issue and has remained low on the global health and development agenda. With the recent adoption of the United Nations (UN) Sustainable Development Goals (SDGs), recognizing health as a major prerequisite for addressing economic, social and environmental development, we are entering into a new era for global health. Now, more than ever there is an urgent need for global commitments from countries to address and integrate oral health into broader general health and NCD policies.

WOHD is a key date in the calendar that can be leveraged to promote the oral health agenda. It’s an opportunity to put the spotlight on the immense burden caused by oral diseases and drive awareness among the general public, media and policymakers as well as call for integrated action. It is also a day to educate people to practice good oral hygiene habits and manage their risk factors, including adopting a healthy diet (one that is low in sugar), avoiding tobacco use and excessive alcohol consumption – all actions that will not only benefit oral health but help maintain general health and well-being.

The 2018 WOHD theme: ‘Say Ahh’. The 2018 campaign sub-theme ‘Think Mouth, Think Health’ introduces the link between oral health as being an indicator of general health and well-being. The campaign aims to educate people that keeping a healthy mouth is crucial to keeping it functioning correctly and for maintaining overall health and quality of life.

PREPARED
Dr Khanyi Makwakwa
FDI Liaison: South Africa

References
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Oral diseases may be linked to an increased risk of general health conditions and vice versa.

Preventive care is always the best option to protect your mouth and body. Seek early detection and treatment to fight oral diseases and associated health complications.

www.worldoralhealthday.org

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