

Clinical Reversal of Occlusal Pit and Fissure Carious Lesions (OPFCLs)

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Abstract: Ozone has been shown to clinically reverse carious lesions in previous studies. This study aimed to assess a single 20-second dose of ozone to manage primary occlusal pit and fissure carious lesions in a general dental practice over a 24-month time period. Treatment of the carious lesion consisted of thorough prophylaxis of the occlusal surface, treatment with ozone or compressed air for 20 seconds, and the application to all treated teeth of a remineralising wash. All lesions and teeth were exposed to remineralising products from a standard dentifrice and brush throughout the 24-month study period.

Objective: To assess the effect of an ozone delivery system¹, combined with the daily use of a remineralising patient kit², on the clinical severity of non-cavitated primary occlusal pit and fissure caries carious lesions (OPFCLs), in a young population group.

Design: A total of 106 subjects, (age range 20 – 29, mean \pm SD, 24.3 \pm 3.7 years), each with at least two OPFCLs, were recruited. Carious lesions were evaluated with a validated caries severity index (the CSI^{3,4}) and lesions in the CSI3 index were selected and were clinically assessed with the DIAGNOdent⁵. The lesions in each subject were randomly assigned for treatment with ozone or no ozone (compressed air), in a double blind study design, in a general dental practice. Subjects were recalled 3, 6, 12, 18 and 24 months. Lesions were clinically re-assessed with the DIAGNOdent and re-evaluated with the CSI at each visit.

Results: There were no observed adverse events in the ozone-treated group. In the control group, one subject was dropped from the study at 18 months as the control lesion had become worse, and the subject presented with pain (this tooth was treated with ozone, cleaned and conventionally filled). Immediately after treatment, the DIAGNOdent readings were recorded. In the control lesion group,

there was no change in the DvT0a values. In the lesions that were treated with ozone – ie the test lesions, there was an immediate decrease in the DvT0a values, with 86% (91 lesions) changing from 20-24 Dv group, to the 10-19 Dv group, and 14% (15 lesions) showing no change – ie they remained in the 20-24 Dv group.

After **3 months**, in the ozone treated group, 91 OPFCLs (86%) had reversed from CSI3 to CSI2&1 and none had deteriorated, whilst in the control group, 6 OPFCLs (6%) had become worse (CSI4) ($p < 0.01$), and 2 (2%) had reversed (CSI1&2). At the **6-month** recall, in the ozone-treated group, 17 OPFCLs (16%) remained static at CSI3, the remaining 88 (84%) OPFCLs remained reversed at CSI1&2, whilst in the control group, 9 OPFCLs had become worse (9%) and one had arrested and reversed ($p < 0.01$). At **12 months**, in the ozone treated group 79 (79%) remained in CSI1&2, 20 (20%) were recorded as CSI3, and 1 lesion (1%) was recorded as CSI4. In the control group 17 (17%) of the OPFCLs had progressed from CSI3 to CSI4, i.e. became worse, 83 OPFCLs (83%) remained in CSI3 ($p < 0.01$). At the **18-month** recall, 69 (73%) of ozone treated OPFCLs remained in CSI2&1, 23 (24%) in CSI3, and 3 (3%) had become worse (CSI4), whilst in the control group, 23 lesions (24%) of the OPFCLs had worsened from CSI3 to CSI4 ($p < 0.01$), 72 (76%) OPFCLs remained in CSI3 ($p < 0.01$). After **24 months**, and at the study end, 43 (58%) of ozone treated OPFCLs remained in CSI2&1, 26 (35%) in CSI3, and 5 (7%) had become worse (CSI4); in the control group, 38 lesions (51%) of the OPFCLs had worsened from CSI3 to CSI4 ($p < 0.01$), 36 (49%) OPFCLs remained in CSI3 ($p < 0.01$).

Conclusions: Non-cavitated OPFCLs of CSI3 or less can be managed non-operatively with ozone and remineralising products. This study, when viewed in terms of the Holmes J 2003 Root Caries study⁶, illustrates that a single dose of ozone is not an effective treatment for all carious lesion types, and that an alternative treatment protocol may be to re-treat with ozone at regular intervals. However, ozone treatment is an effective alternative to conventional "drilling and filling".

¹: HealOzone, KaVo GmbH Germany; ²: Remineralising Patient Kit, CurOzone Inc, Canada; ³: CSI – Clinical Severity Index, Lynch E and Holmes J, Arresting Occlusal Fissure Caries Using Ozone. AADR Abstract no.678; 2003; ⁴: Holmes J. Clinical Reversal of Occlusal Pit and Fissure Caries Using Ozone. The First Pan-European Festival of Oral Sciences, Cardiff, Wales, UK. Abstract no 431; 2002 and J Dent Res 82: C-535; 2003; ⁵: DIAGNOdent, KaVo GmbH Germany; ⁶: Holmes J. Clinical Reversal of Root Caries Using Ozone, double-blind, randomised, controlled 18-month Trial. Gerodontology 2003:

20 (2): 106-114.

Key words: occlusal pit and fissure caries, reversal, arrest, ozone, toothpaste, mouth-rinse, spray.

Introduction

In 1881, at Wiesbaden, in Germany, at a meeting of the American Dental Society of Europe, a young American dentist, Dr. W. D. Miller, presented the results of his experiments conducted in the laboratory of bacteriologist Dr. Robert Koch. Miller's studies¹ found that acid produced by microorganisms in the mouth caused caries of the enamel, and caries in dentin resulted from acidic decalcification. These bacterial acids led to the demineralisation of enamel and dentin. His research found that bacteria did not need to be present in enamel or dentin to initiate demineralisation. He also succeeded in producing artificial caries that was not different from the natural carious lesions. Yet it took nearly 100 years before it was accepted that plaque was the layer from which the cariogenic processes came from.

As early as the 1950's, it was already known that germ-free rats did not develop caries, compared to 'normal' animals who had a normal bacterial spectrum present in their mouths and gut systems, despite a cariogenic diet. Writing in 2002, Young's literature review² of published data over the past decade suggested that dental caries should be treated as a curable and preventable infectious disease.

In 2003, ten Cate³ reviewed the current consensus of dental caries⁹, its pathogenesis, aetiology and prevention, and compared it to the concepts in the 1950's (ten Cate JM. 2003). ten Cate argued that the debate has moved away from a chemo-parasitic pathogenesis versus the proteolytic theory of caries and that it is now accepted that bacteria and acids formed in plaque are the cause of dental caries.

Ten Cate also noted that as the rate of progress of carious lesions is reported to have slowed, so there is at last an opportunity for a directed interventional and preventive approach, rather than a restorative therapeutic approach.

Research in the last 25 years show that cariogenic bacteria are transmissible. The transmission, especially of *S. mutans*, was reviewed by Berkowitz⁴ (Berkowitz RJ 2003).

Practical dentistry world-wide is characterised by the treatment of the effects of caries, ie drill 'n fill, rather than treating the disease itself. This pharmaceutical approach to treating dental disease is the next evolution for the dental profession and requires caries risk assessment. The current restorative-driven disease management has been shown to have no measurable effect on the cariogenic bacterial loading in the remainder of the mouth⁵ (Wright JT et al, 1992). This is supported by a more recent study⁶ (Featherstone et al 2002). This study followed a group of adult subjects who were restored according to the current "standard of care." Levels of mutans streptococci and lactobacilli were measured at the study start and after restoration. The result showed that there was no statistical difference between before and after restorative care in the oral bacterial loading. The importance of these studies is to illustrate that without pharmaceutical management as part of a treatment protocol, a restored mouth will continue to be prone to the effects of dental infection. This has obvious implications for caries high-risk individuals, as well as the cost of centrally-funded dental care. The profession needs new diagnostic technology to detect infection long before restorative care is required, feeding into risk-assessment to guide the necessary management of the individual.

A mature active carious lesion contains in excess of 450 different bacterial species. A great deal is known about the initiation and progression of dental caries. Caries is multifactorial, and that the presence of bacteria with food substrates is the key to its understanding. The loss of minerals from the tooth surface under acidic conditions, be it enamel or dentine, is known as demineralisation. It is also known that teeth are able in 'normal' conditions to take up minerals. This is the process of remineralisation. This mineral outflow and uptake are normally in balance, and this balance is influenced by the pH of the oral cavity, food types and saliva flow.

Saliva contains a number of important minerals such as phosphate, calcium and fluoride that may aid lesion remineralisation in the right environment, and saliva acts as an important buffer for oral acids (Silwood et al 1999⁷, Silwood et al 2002⁸, Silwood et al 2002⁹). It is only when the rate of demineralisation exceeds remineralisation will the overall loss of minerals lead to the formation of a cavity.

Research has shown that as a carious lesion begins, there is a zone of intact tooth surface with the zone of demineralisation spreading out gradually below this. As this zone of mineral loss extends deeper into the tooth tissue, the surface breaks down or cavitates. This cavitation allows the normal mix and balance of bacterial types – acid and alkali-producing bacteria - to set up a micro-niche environment within this roughened area. Routine oral hygiene measures are no longer able to remove bacteria and oral debris that accumulates into this roughen area, and the protein pellicle protects the developing bacterial community from the effects of mineral rinses and anti-microbial agents, such as phosphate, calcium, fluoride and Triclosan.

Over time, this bacterial community evolves from one where there is an average pH neutrality, to one where the acidophilic and aciduric bacterial types predominate. As the lesion becomes more acidic, two processes start to happen: First, the lesion will only support acid-loving bacterial types, as oxygen is now excluded from the niche environment. Secondly, the increase in the local acidity of the lesion lowers the cavity pH. As the lesion moves to being more acidic, the process of mineral loss dominates. The whole process becomes self-fuelling, and lesion progression through the tooth tissue accelerates. Once the enamel dentine junction (EDJ) is reached and penetrated, the lesion mushrooms out due to the dentinal tubular structure and morphology. The advancing waves of organic acids are implicated in dental pain, and the denatured dentine goes through various stages of mineral loss and destruction, from hard, to leathery, finally to soft. This progression has been classified in buccal carious lesions by Beighton & Lynch¹⁰ and Holmes & Lynch published the CSI¹¹ that allows lesion classification in a general dental practice.

A Pharmacological Approach to Caries Management.

Halting the lesion before cavitation is the ideal goal of the dental profession. If the acidic nature of the lesion is changed towards neutral or alkaline and the acid biomolecules are removed from the lesion, remineralisation will occur. The remineralised area or 'healed' area is harder and more resistant to subsequent acid attack. This is because the lesion is hyper-mineralised. It may stain in this process, and will not recover the pre-lesion optical properties due to the mineral infiltration.

Further studies have linked salivary and plaque conditions (Fure 1998¹², Beighton & Lynch 1995¹⁰). It is now recognised that the most advantageous treatment for root caries, for example, is lesion arrest and remineralisation (Allen *et al* 1999¹³, Holmes J 2003¹⁴). The use of pharmaceutical agents (Lynch 1996¹⁵, Baysan & Lynch 2001¹⁶) and fluoride-containing dentifrices may provide some protection for high caries risk patients. Various pharmaceutical agents have been used, such as fluoride (Lynch *et al* 2000¹⁷, Lynch & Baysan 2001¹⁸, Baysan *et al* 2001¹⁹, Duckworth 1993²⁰), and chlorhexidine or chlorhexidine in combination with thymol (Lynch *et al* 1995²¹).

The attempt to deal conservatively with dental caries seems to have picked up momentum in the last 10 years of the 20th century with the advent of new materials and tools. The most common practice today is still the complete removal of all soft decayed tissue regardless of the level of infection and histological destruction. There have been calls for changing the paradigm from “drilling and filling” to managing the disease as an infection. (Rethman J. Trends in preventive care: Caries risk assessment and indications for sealants. *J Am Dent Assoc* 2000; 131 (Suppl):8S-12S.²² Kidd E. Caries management. *Dent Clin N Am* 1999;43:743-64.²³ Featherstone JDB The caries balance: the basis for caries management by risk assessment. *Oral Health Prev Dent.* 2004;2 Suppl 1:259-64.²⁴ Featherstone JDB The caries balance: contributing factors and early detection. *J Calif Dent Assoc.* 2003 Feb;31(2):129-33.²⁵

However, there have been few studies that evaluated the outcomes of such an approach; moreover, some dental schools do not teach or practice this model. The dental care systems in the UK, USA and other countries do not reimburse or promote such an approach. Instead, the routine provision of “prophylaxis” and professional fluoride application has been the main form of preventive care provided by dentists. Ozone is ideally suited as a pharmacological agent to assess the management and treatment of decay as a disease and infective process.

Diagnosis and Prevention.

The initial diagnosis of caries can be difficult in general dental practice, made more difficult due to differing levels of investment in equipment from practice to practice. Whilst calls for a change from a

“drill-n-fill” of an infectious disease, to a pharmaceutical management strategy have been made by numerous authors ¹⁴.Holmes J 2003 ¹⁶ Baysan & Lynch 2001, ¹⁷ Lynch *et al* 2000 ¹⁸. Lynch & Baysan 2001 ¹⁹. Baysan *et al* 2001) the call has largely been ignored for a variety of reasons; the lack of accurate diagnostic tools; inadequate operator skills; the cost of investment into the new technologies; inertia by both the dental profession and Government Health Departments. There is also a failure to communicate effectively to both the public and the majority of dental practitioners the benefits of a change in management and treatment of a preventable disease.

In the United Kingdom, for example, advanced electronic diagnosis in the form of the DIAGNOdent is not available in every practice. Likewise, the age of equipment varies between the older practice and practitioner, in comparison to some of the newly equipped dental centres and younger dental practitioners.

A review by Lussi A *et al* in 1999 ²⁶ illustrated that depending on the systems and technology used to make a diagnosis of early caries, the agreement amongst a group of dental professionals can be very different. Likewise, in the treatment of that lesion, there are various protocols that have been shown to be more effective than others are, depending on the operator’s knowledge, skill and manual dexterity.

Where there is an obvious frank cavitation, the diagnosis is very easy to make, and there is little room for disagreement. However, where the lesion is non-cavitated and not visible to the examiner, the diagnosis of the presence or absence of a carious lesion becomes more problematic.

Primary fissure caries lesion accounts for up to 70% of all new lesions seen in dental practice ²⁷ (The Implications of Using Ozone in General Dental Practice, in ‘Ozone – The Revolution in Dentistry. Lynch E, Pub Quintessence London 2004, ISBN 1-85097-088-2 p71.).

It is now believed that the carious process in the occlusal pits and fissures of molar teeth starts before full eruption into the oral cavity. A study in 1992 by Carvalho *et al* ²⁸(Carvalho *et al* 1992) showed statistically the majority of molar teeth erupt into the oral cavity with an occlusal fissure carious lesion already established. This is explained by the long eruption time of 5 to 45 months ²⁹ (Ekstrand KR *et al*, 2003), from the point of rupture through the oral mucosa of the molar cusps to full mucosal break

down and exposure of the occlusal surface. At the point of first rupture into the oral cavity, the oral flora gain access to the hitherto sterile and sound enamel fissures. A bacterial niche is quickly established. As the partially buried occlusal surface cannot be cleaned, and the mucosal surface is often sore and painful, this niche is rapidly exploited and colonised. Over time, the bacterial niche changes to predominantly acidic, leading to mineral loss within the fissures and from the tooth surface. The eruption pocket is regularly filled with oral debris that the bacterial colonies use as their substrate. Inevitably an acidic niche becomes established, that will lead to the development of an area of demineralisation. Over time, this infection will spread into the sub-surface layers. In pre-molar teeth, the shorter eruption time of 3-6 months would seem to explain why these teeth show fewer cavities. The variation in molar eruption duration highlights the importance of individualising caries preventive strategies for children and incorporating a correct risk assessment.

Some studies have concentrated on how to limit the establishment of this pre-eruption carious lesion, and a recent paper from Australia would suggest that the use of a mineral-releasing glass ionomer such as FujiVII, placed below the operculum and onto the un-erupted molar surface can prevent this type of lesion developing. The paper 'The Fissure Seal Time-Bomb' by Geof Knight, Australia, ³⁰ (Geoff Knight G, 2002) points a way forward. Once the tooth is fully erupted, the glass ionomer FujiVII is removed, and once the occlusal surface of the molar tooth has fully erupted into the oral environment, the exposed glass ionomer can be removed. A conventional fissure sealant can be placed to allow normal occlusion to develop. Routine oral hygiene measures can now be instituted, such as toothbrushes and their mechanical versions. The fissures are also exposed to the effects of fluorides and mineral washes. Depending on the chemistry of the mineral wash and the pH of the fissures, these may be more or less effective.

However, this does not help the dental profession make the all-important diagnosis of a carious lesion in the fissure pattern. Occlusal fissures show a bottle-like structure. The fissure entrance walls are often in close approximation, and sealed with a permeable organic plug. Areas of demineralisation may start in the deeper walls or at the base of the fissure, and is effectively hidden from conventional detection. This 'hidden caries' was first described in 1868 ³¹ (Knapp J 1868). Studies by Lussi et al

(Lussi *et al*²⁶) and others have shown that decay needs to be 2-3 mm inside the dentine – ie extending through the enamel, through the edj and into the dentine structure of the tooth - before it will even show on an x-ray.

In the late 70's, the concept of prevention started to make headway into the dental profession. It was postulated that if the disease process could be prevented, slowed or halted, then the common end expression of caries – ie cavities – could be avoided.

As early as the 1950's, it was already known that germ-free rats did not develop caries, compared to 'normal' animals who had a normal bacterial spectrum present in their mouths and gut systems, despite a cariogenic diet. Cariogenic bacteria were known to be part of the gram-positive oral flora group. Lastly, when fed through a tube – i.e. food was placed directly into the stomachs of laboratory animals, those animals with a 'normal' bacterial spectrum did not develop caries. By the late 1950's, the work of Fitzgerald and Keyes³² (Fitzgerald, R.J., Keyes, P.H. 1960) illustrated that caries can be transmitted between individuals, and should be regarded as an infection.

In 1975 the Specific Plaque Hypothesis was put forward by Loesche³³ (Loesche WJ 1976). Loesche argued that out of the total 'normal' bacterial flora found in an individual, only a small number were actually involved in the carious process. Systems to identify subjects at risk were based on identifying mutans streptococci and lactobacilli, and have been reviewed in the dental literature – for example by Krasse in 1990 (Bader JD 1990³⁴) and Powell in 1998³⁵ (Powell LV 1998). Anderson, in his 1993 medical treatment model of dental decay³⁶ (Anderson MH *et al* 1993), proposes that treatment should start with the elimination of the potential sites for infection – the fissures, pits, grooves and potential plaque retentive areas - by altering the morphology of these areas to prevent bacterial ingress and infection. Following this step to reduce the potential bacterial niche sites, the bacterial profile of the individual is modified by dietary and antimicrobial treatment. Lastly, the individual is placed on an individualised recall programme, not the ubiquitous 6-month recall that has little scientific credibility or foundation. This dental treatment model is in essence that used in Swedish University dental departments since the 1970's³⁷ (Krasse B. 1985).

Later studies by Becker *et al* in 2002³⁸ (Becker RM *et al* 2002) suggest that the main causative

bacterial species are *Streptococcus mutans*, *Lactobacillus*, and *Bifidobacterium*. Workers involved in the production of an inoculation against caries have created a genetically modified strain of *S. mutans*, and the results of the initial trials look very interesting. However, with the total number of species in a mature lesion exceeding 450, isolating the main causative bacterial species in such colonies where each species shows interdependency, yet retains the capability of evolving nutritional systems independently, illustrates how complex these colonies of bacteria are. They are far from being simple bacteria with loose associations of interdependency as they are often presented to be. This should not be surprising, as bacteria have evolved over millions of years, whereas their hosts – man and animals - have had a shorter existence and evolutionary time span.

The concept of fissure sealing was introduced to the dental profession 1970's. The preventative sealants have a placement protocol: The tooth surface is cleaned with an abrasive powder and rotary bristle brush. Various weak acids are used to strip off the protein coat of the tooth surface, and expose the prism enamel structure, so that an organic resin can be bonded to this surface. The preventative argument is that if the bacteria are denied entry, the carious process cannot initiate. It was also argued that even if there is incomplete removal of bacterial colonies, the sealing process would deny these bacteria food substrate, and the carious process will therefore halt.

However, there are a number of flaws in this protocol. First, the abrasive pumice powder and bristle system cannot penetrate into the fissures. The bristle size is large in comparison to the fissure orifice that it is unlikely that the organic plug is ever removed. Zones of demineralisation in the fissure surface were simply unknown at this point of time by the average dental practitioner. Moreover, deeper hidden lesions were simply not recorded by the general dental practitioner, as there was no system to allow their diagnosis.

Secondly, effects of the acid-etchant can be variable. Dentists are not known for their adherence to instructions. They will 'gestimate' times, so etchants may be left on either for too little time – giving poor enamel structure exposure and poor bonding; or too long a time, weakening the structure and leaving a poorly bonded polymer on the tooth surface.

Thirdly, the bacterial colonies below sealants do not die – they either became static, or enter an almost dormant life cycle. In addition, the bacterial bio-molecules – the biochemical effluents that each level of the bacterial 'tree' involved in the developing and mature lesion – are left in situ. The dental

profession knows a great deal about these bio-molecules now. A mature lesion will re-establish within 6-8 weeks if only the bacteria are removed. However, if you remove the bacteria *and* the bio-molecules, then the lesion takes 16 weeks or longer to re-establish. This is the essential difference between just bacterial-removal, and sterilisation of the infected lesion.

Lastly, the resins used to 'seal' the fissures were prone to problems of polymerisation and the establishment of a true 'seal' can almost impossible. The first sealants were of a twin-mix variety, where a base and catalyst were mixed, then applied to the etched and dried enamel surface. Dispensing exactly the same amounts of base and catalyst is difficult from two pots of resins. With the modern dispensing twin-syringes and light-cured resins, this has become less of a problem. After the introduction of polymerisation lights, the process of setting these sealants became easier. The modern fissure sealant has evolved a great deal since the first auto-polymerisation products of the 1970's.

The net result of these historical materials and systems were to leave areas of imperfect seal at the edges of the organic plug or at the base of structural grooves, allowing food substrates to leak below the sealants. The 'quiescent' and 'in-active' bacterial colonies became active and the establishment over time of a deep carious lesion and an almost 'hollow' tooth has been seen by many dental practitioners in every country where these preventive systems were used. Examples of the leakage sites are the palatal end of the palatal groove in upper molars, and the gingival extent of the buccal groove on lower molar teeth. For those practitioners who have been in practice long enough, they will have seen patients for whom they placed sealants in the late 70's and 80's, only to see these patients returning with 'bombed-out' molar teeth in later years and 'unexplained' onset of dental pain.

The historical use of mirror and probe is very unreliable in terms of its diagnostic capability, especially in identifying enamel demineralisation and fissure caries. In the fissure caries scenario, the probe tip is not small enough to penetrate through into the fissure due to the close approximation of the fissure walls. The research has shown that caries can develop at the base and in the walls of these fissures. It is impossible to probe into these areas of the fissure pattern without making an access cavity. Diagnosis of caries at the base of the fissure becomes even harder and fraught with problems of access and visibility. Even magnifying the tooth surface with loops, intra-oral cameras, and microscopes will not help, due to the narrow opening to the fissure, and the presence of the organic

plug. As a dental student in the 1980's, I remember being told to maintain a sharpened probe tip. Then we were told that the sharp tip might actually cause a lesion, so they should be blunt. Lastly, we were then told to re-sharpen the probe tips, as blunt tips could not 'diagnose' lesions. Little wonder that the dental profession at large has made little change, after so many mixed messages from the academics.

Diagnosis is further complicated by requirements from the dental profession's mal-practice insurers. In setting the minimal acceptable diagnosis standard as the taking of x-rays means that the majority of patients may show extensive tissue destruction and infection, before remedial operative care is started. Put in different terms, with this model substantial numbers of lesions may go undiagnosed, leaving the patient in a compromised state and requiring traditional restorative care. Often these lesions are not visible on the surface. In addition, the effect of the use of mineral washes and fluoride has been to make the tooth denser and impermeable to x-rays, making the diagnosis from x-rays more difficult.

For many years, before some of the more sophisticated technologies for caries diagnosis were available, fissurotomy burs and air abrasion were used to open up occlusal fissures. The use of fissurotomy burs undoubtedly lead to the destruction of sound tooth tissue, albeit in reduced volume when compared to routine diamond and tungsten carbide dental burs. The use of the dental handpiece, and the use of dental burs has been the dentist's usual tool for cavity preparation; the majority of dental practitioners quote the tactile feedback as the main reason for the continued use of this item, quite apart from relatively low cost, speed of preparation, and good visual field of sight. Air abrasion allows a far finer preparation, but the lack of tactile feed back is the main reason for the small number of dentists who use this system. Other objections to the use of air abrasion are the dust caused by the system leading to poor visibility, and the slow preparation time.

Air abrasion is useful to remove the organic plug and open the fissure orifice. Abrasion units that allow the adjustment of the air pressure and powder flow are more useful than the simpler on-or-off systems. Inevitably, abrasion will remove a small amount of tooth tissue. Air abrasion technologies were introduced in the late 1970's. The particle size of 27 microns was chosen, as this sized particle is not implicated in lung disease. Smaller particle size has been implicated as the source of the development of mesotheliomas.

What was clearly needed was a system that can measure the density of the tooth structure, or 'read' the bacterial contamination of the tooth, so that the dental profession could diagnose at a much earlier stage in the development of a carious lesion. Ideally, this would be at the development stage where the demineralisation was confined to enamel and long before the lesion had reached or penetrated through the edj into the dentine.

Every dental student is taught the classical signs of areas of decay; The tactile response to a sharp explorer and lesion colour. There are a number of validated indices used in caries research – combining the visual appearance of carious lesion and the tactile feedback from a sharp dental explorer. The index most often used is the Ekstrand Index³⁹. (Ekstrand KR *et al* 1998)

The Ekstrand Index has seven groups as follows;

0	No or slight change in enamel translucency after prolonged air drying (>5s)
1	Opacity (white) hardly visible on the wet surface, but distinctly visible after air drying
1a	Opacity (brown) hardly visible on the wet surface, but distinctly visible after air drying
2	Opacity (white) distinctly visible with out air-drying.
2a	Opacity (brown) distinctly visible with out air-drying.
3	Localised enamel breakdown in opaque or discoloured enamel and or greyish discolouration from the underlying dentine.
4	Cavitation in opaque or discoloured enamel exposing the dentine beneath

Table 1; The Ekstrand Index

In earlier studies published as IADR Abstracts, the Ekstrand Index was modified to allow easy and fast indexing of lesions in a general dental practice environment. This new index, called the Clinical Severity Index (CSI, Table 1b), was developed by Professor Edward Lynch (Queens University, Belfast) & Dr Julian Holmes (Queens University, Belfast & UKSmiles Wokingham) (Holmes J 2002⁴⁰. Holmes J 2003⁴¹. Holmes J, Lynch E. 2003⁴². Holmes J 2003⁴³. J. Holmes and E. Lynch 2004⁴⁴). The CSI marries the Ekstrand Index to suggested ozone treatment times. This varies from 0 seconds for

the lowest (0) CSI score to 40 seconds with the highest (5) CSI score. The CSI was used in this and the earlier studies referenced above.

Index	Clinical Severity Index (CSI)	Tx (sec)
5	<p>SEVERITY INDEX 5 Lesion deemed to require drilling and filling.</p> <p>Definition; presence of infected dentine extending one or more mms into Dentine</p>	40 seconds O ₃
4	<p>SEVERITY INDEX 4 Lesion deemed to require drilling and filling.</p> <p>Definition; presence of infected dentine extending less than one mm into Dentine</p>	30 seconds O ₃
3	<p>SEVERITY INDEX 3 Lesion deemed to require drilling and filling with a preventive resin restoration.</p> <p>Definition; presence of enamel caries extending to the enamel dentine junction.</p>	20 seconds O ₃
2	<p>SEVERITY INDEX 2 Lesion deemed to require fissure sealing.</p> <p>Definition; presence of enamel caries confined to enamel and not extending to the enamel dentine junction</p>	10 seconds O ₃
1	<p>SEVERITY INDEX 1 Lesion deemed to be reversing.</p> <p>Definition; presence of infected demineralised dentine or carious enamel, which is reversing. This scenario is where clinical remineralisation of the underlying dentine is considered to be in the process of remineralising the demineralised dentine but is not yet complete. The frosted enamel in the fissure (visible after drying) will be reducing</p>	10 seconds O ₃
0	<p>SEVERITY INDEX 0 Lesion arrested.</p> <p>Definition; Infected dentine which reversed and where clinical remineralisation of the underlying dentine is considered to be complete, with no infection remaining in the dentine as well as remineralised enamel with no frosting on drying.</p>	No treatment required except preventative

Table 2 Clinical Severity Index (CSI)

In all these studies, the CSI was tested against the DIAGNOdent. The DIAGNOdent (KaVo GmbH, Germany) has been shown by various studies to offer a fast, reliable and a scientific way to evaluate carious lesions in dental research and dental practice. Various studies have shown that caries diagnosis is difficult especially where the lesion is at an early stage of development, within the enamel surface. At this stage, visual inspection and the use of mirror and probe have a poor inter-examiner correlation ⁴⁵ (Lussi *et al*, 2001). Enamel demineralisation cannot be seen on x-ray, and studies comparing the histological appearance of a lesion and an x-ray of that lesion suggest that once a lesion is visible on an x-ray, the lesion is at least 2mm through the EDJ into the dentine⁴⁵ (Lussi *et al* 2001).

The use of transillumination, such as the DIFOTI system, has not become a common diagnostic tool in general dental practice for unknown reasons. Trans-illumination offers little diagnostic value for occlusally originating carious lesions, due to the bulk of the tooth in a lingual-buccal orientation. However, it is very useful for the identification and evaluation of inter-dental lesions. It is therefore outside the scope of this paper.

The use of the Electric Caries Meter (ECM Lode, Holland) which provides the gold standard in research is too expensive and takes too long in time, for use in general practice. The ECM works by passing an electric current through a tooth surface, and measuring the impedance or resistance to the current. The measurement provides an indication of the tooth's density and by definition, mineral loss or uptake if values are compared. However, when the average examination in general dental practice is estimated to take just 8 minutes, and a lesion analysis with the ECM up to 20 minutes, it is not the sort of tool that will interest the routine general dental practitioner who just wants to pick up his trading instruments of mirror, probe and turbine hand piece.

In a review of caries diagnosis, Lussi *et al*⁴⁶ (Lussi *et al* 2004) published a guide to the use of the DIAGNOdent in caries diagnosis. This has now been incorporated into a modified CSI as a guide to the time of treatment required for a carious lesion according to the lesion's severity.

a basic guide to the DIAGNOdent values			
DIAGNOdent Values		extent of carious lesion	
Appears as white spot -> DV	10~19	confined to enamel	CSI 2
Appears as a stain -> DV	20~24	at the edj	CSI 3
Just visible on X-rays -> DV	25~29	1-2mm into dentine	CSI 4
Visible on X-rays -> DV	> 30	3-4+ mm into dentine	CSI 5

Table 3 The Basic Guide to DIAGNOdent Values (Dv)

Incorporating data from Lussi et al2004⁴⁶

Recent publications conclude that ozone should be considered as an alternative pharmaceutical management strategy (Baysan 2002⁴⁷, Baysan *et al* 2001¹⁹, Baysan *et al* 2000⁴⁸, Baysan & Lynch 2004⁴⁹, Holmes 2003¹⁴, Lynch 2003⁵⁰, Lynch 2003⁵¹) rather than the traditional drill and fill approach. Ozone (a pale blue-coloured gas, chemical formulae O₃) plays an important role as a natural constituent in the higher layer of the Earth's atmosphere. It has been used for many years in medicine, and within recent years in dentistry. A device that has a CE mark, know as the HealOzone (CurOzone, USA and KaVo GmbH & Co, Germany) has been available commercially in Europe for more than 2 years. Ozone is a very powerful antimicrobial agent. Recently, Baysan *et al.* (Baysan *et al* 2000⁴⁸) and Baysan & Lynch 2004⁴⁹ reported that ozone application either for 10 or 20 seconds was effective to kill the great majority of microorganisms in PRCL's (>99% microbial killing after 10 seconds Ozone application). Ozone has been shown to oxidise the niche environment biomolecules, to acetate acid and carbon dioxide. The importance of this is that

1. The bacterial colonies are oxidised and removed from the lesion within a very short time period. The short bacterial elimination time means that in reality, bacteria will never evolve a resistance to ozone treatment.
2. The carious micro-flora biomolecules are oxidised. The former acidic pH of the niche environment is destroyed, and it becomes more alkaline.

3. The alkaline pH niche has a 14 – 18 week time window of opportunity to take up minerals either from the saliva, or from surface applied mineral sources, or both. The carious microflora has to evolve over an extended period of time to re-establish a mature bio-environment. If the biomolecules were not removed by the action of ozone, previous research suggests that the carious lesion microflora would re-establish within 4-5 weeks.
4. It is speculated that the destruction of the biomolecules deep in the dentinal tissues at the advancing edge of the carious lesion results in a reduction of sensitivity and pain.
5. As an additional feature, ozone opens the tubular structure, and raises the possibility of direct bonding to the treated lesion surface without the need to stabilise and create the 'hybrid' layer from the etched dentinal tubular surface.

O₃ is naturally produced by the photo-dissociation of molecular O₂ into activated oxygen atoms, which then react with further oxygen molecules. This transient radical anion rapidly becomes protonated, generating HO₃[•], which, in turn, decomposes to hydroxyl radical. Further reactions convert O₃ to an even more powerful oxidant, the hydroxyl radical (OH[•])

In view of its powerful oxidising properties, O₃ can attack many biomolecules such as the cysteine, methionine and the histidine residues of proteins. The effects of ozone on cell structures, metabolism and microorganisms are well documented in published papers (Baysan 2002⁴⁷, Bocci 1992⁵², Bocci *et al* 1993⁵³, Bocci 1996⁵⁴, Bocci 1996⁵⁵, Bocci 1999⁵⁶) in both dentistry and medicine. Research has shown that ozone disrupts the cell walls of micro-organisms within seconds, leading to immediate functional cessation. This effect within a very short time is of great clinical significance, as the potential for microbial resistance to this treatment modality is insignificant. Baysan *et al* (Baysan 2002⁴⁷, Baysan *et al* 2000⁴⁸) have published reductions from Log₁₀ 6.0 to Log₁₀ 0.46 colony forming units after just 20 seconds of ozone. Studies have shown that just 10 seconds of ozone treatment is sufficient to produce reversal of PRCL's (Baysan 2002⁴⁷, Baysan *et al* 2001¹⁹, Baysan *et al* 2000⁴⁸, Baysan & Lynch 2004⁴⁹, Lynch 2003⁵⁰, Lynch 2003⁵¹).

The data in this study, 'Clinical Reversal of Occlusal Pit and Fissure Carious Lesions (OPFCLs) using Ozone in General Dental Practice', was originally published as an abstract for the Cardiff Pan European Festival of Oral Sciences 2002 meeting⁴⁰ (Holmes J 2002). This study reported the results of a study to assess the effects of the use of ozone on occlusal pit and fissure caries in a general dental practice. The data presented here is in part drawn from this larger pilot study that followed 376 subjects with carious lesions in 1170 teeth. In this published study, after randomisation, lesions were assigned to either receiving no treatment or ozone treatment. Ozone was applied to each test lesion for 10, 20, 30 or 40 seconds depending on the (CSI) clinical severity index. The DIAGNOdent was employed to objectively quantify each carious lesion. After 3 months, subjects were recalled and clinically re-assessed. The DIAGNOdent was again employed to objectively quantify each lesion. In this pilot study, there were no observed adverse events in either the treated or control group. 99% of the ozone treated carious lesions (978 teeth) had clinically reversed based on the DIAGNOdent readings ($P < 0.001$) at 3 months after ozone treatment. This pilot study also showed that large carious lesions showed reversal over a longer time (3-6 months) interval, requiring multiple applications of ozone. The control carious lesions, which had not received any ozone treatment, did not significantly change in the 3-month study period, or became worse. The conclusion drawn from this study was that ozone should be considered an effective alternative to conventional "drilling and filling" for carious lesions in general dental practice.

This study follows a group of 20 – 29 year old subjects over a period of 24 months. The age group was chosen to simplify subject consent for entry into the study, and this age group has been identified as one where the incidence of caries may be increasing.

Fluoride has been shown to reduce the incidence of caries in the younger population. The reality of the situation is that many commentators now view this not in terms of prevention, but in terms of delaying the age at which carious lesions seem to form. This trend is believed to be linked to the events of leaving home to take up gainful employment outside the closely guarded family environment – where the parents arrange the oral hygiene and dental examinations, the appointments for general health care, supply a well balanced diet, and insist on communal exercise in the forms of walking, etc. Once these individuals leave this guarded environment, financial constraints, poor diet and lack of

exercise, and failure to take up responsibility for their own health conspire to allow bacterial communities to establish, and the development of carious lesions soon follow.

The interest in ozone as a treatment modality in its own right is growing among dental researchers and the public. Recent reports in worldwide magazines and international press coverage on dental and medical ozone treatment has led to an interest in and a demand for this 'homeopathic-type' treatment by the public. Studies in the early 21st century by [Baysan et al](#)⁴⁸ ([Baysan et al](#) 2000) have shown that just 10 seconds of exposure to ozone (O₃) can reduce the number of colony-forming bacteria from log₁₀ 7 to log₁₀ 4.35 after 10 seconds, and if 20 seconds of O₃ application are used, the number of colony-forming bacteria is reduced to log₁₀0.46. Recent clinical studies by [Holmes J](#)¹⁴ ([Holmes J, 2003](#)) and [others](#) have shown that the predictability of a positive outcome – the elimination of the carious niche, arrest and reversal – can be improved with a small modification in the treatment protocol, and caries reversal can be achieved with ozone treatment with limited tissue amputation and intervention.

Aim:

The aim of this study was to assess the effect of an ozone delivery system, combined with the daily use of a remineralising patient kit, on the clinical severity of non-cavitated primary occlusal pit and fissure caries carious lesions (OPFCLs), in a young population group.

Design:

A total of 106 subjects, (age range 20 – 29, mean \pm SD, 24.3 \pm 3.7 years), each with at least two OPFCLs, were recruited. Carious lesions were evaluated with a validated caries severity index (the CSI) and lesions in the CSI3 index were selected and were clinically assessed with the DIAGNOdent. The lesions in each subject were randomly assigned for treatment with ozone or no ozone (compressed air), in a double blind study design, in a general dental practice. Subjects were recalled 3, 6, 12, 18 and 24 months. Lesions were clinically re-assessed with the DIAGNOdent and re-evaluated with the CSI at each visit.

Results:

There were no observed adverse events using the HealOzone unit.

Immediately after treatment, the DIAGNOdent readings were recorded. In the control lesion group, there was no change in the DvT0a (DIAGNOdent value Time zero after ozone treatment) values. In the lesions that were treated with ozone – i.e. the test lesions, there was an immediate decrease in the DvT0a values, with 86% (91 lesions) changing from 20-24 Dv group, to the 10-19 Dv group, and 14% (15 lesions) showing no change – i.e. they remained in the 20-24 Dv group.

After **3 months**, all subjects were successfully recalled. In the ozone-treated group, 91 OPFCLs (86%) had reversed from CSI3 to CSI2&1. Not a single lesion treated with ozone had deteriorated. In the control group, 6 OPFCLs (6%) had become worse (CSI4) ($p < 0.01$), and 2 (2%) had spontaneously reversed (CSI1&2).

At the **6-month** recall, one subject was dropped from this study. This subject presented with a control lesion that had become painful. This tooth and the area of caries were treated conventionally. In the ozone-treated group, 17 OPFCLs (16%) remained static at CSI3, the remaining 88 (84%) OPFCLs remained reversed at CSI1&2. In the control group, 9 OPFCLs had become worse (9%) and one had arrested and reversed ($p < 0.01$). An additional 3% of the control lesions had become worse – ie showed signs that the carious lesions were progressing.

At **12 months**, 6 subjects failed to attend their recall request, and were unobtainable. In the ozone treated group 79 (79%) remained in CSI1&2, 20 (20%) were recorded as CSI3, and 1 lesion (1%) was recorded as CSI4. One year after a single dose of ozone, the results show a single lesion that has become worse from the start of the study. 5% of the ozone-treated lesions have moved from CSI 1&2 to CSI3, and 1% of the lesions in CSI3 have moved to CSI4. In the control group 17 (17%) of the OPFCLs had progressed from CSI3 to CSI4, i.e. became worse, 83 OPFCLs (83%) remained in CSI3 ($p < 0.01$).

At the **18-month** recall, a total of 11 subjects failed to attend their recall requests. 69 (73%) of ozone treated OPFCLs remained in CSI2&1, 23 (24%) in CSI3, and 3 (3%) had become worse (CSI4). In the control group, 23 lesions (24%) of the OPFCLs had worsened from CSI3 to CSI4 ($p < 0.01$), 72 (76%) OPFCLs remained in CSI3 ($p < 0.01$).

After **24 months**, 32 subjects of the original study number of 106 failed to respond to their recall reminders, illustrating the difficulty of research in a mobile employment region.

The positive result of this study is that over 90% of the ozone-treated group show continued lesion arrest and reversal. 43 (58%) of ozone treated OPFCLs remained in CSI2&1 and 26 (35%) in CSI3 showing continued stability. Only 5 (7%) lesions had become worse, moving from CSI1&2, through to CSI3 and then showing lesion re-activation to CSI4.

This positive outcome is in contrast to the control group, where over 50% (38 lesions or 51%) of the OPFCLs had worsened from CSI3 to CSI4 ($p < 0.01$), 36 (49%) OPFCLs remained in CSI3 ($p < 0.01$).

The study was terminated at this 24-month recall and review.

Occlusal Pit and Fissure Carious Lesions

	T0		T0a		T3/12		T6/12		T12/12		T18/12		T24/12	
	Number	%	Number	%	Number	%	Number	%	Number	%	Number	%	Number	%
O³ Treated Lesions	106	100	106	100	106	100	105	100	100	100	95	100	74	100
Dv - 10-19	0	0	91	86	91	86	88	84	79	79	69	73	43	58
Dv - 20-24	106	100	15	14	15	14	17	16	20	20	23	24	26	35
Dv - 25-29	0	0	0	0	0	0	0	0	1	1	3	3	5	7
Control Lesions	106	100	106	100	106	100	105	100	100	100	95	100	74	100
Dv - 10-19	0	0	0	0	2	2	1	1	0	0	0	0	0	0
Dv - 20-24	106	100	106	100	98	92	95	90	83	83	72	76	36	49
Dv - 25-29	0	0	0	0	6	6	9	9	17	17	23	24	38	51

T0	Surface Prophylaxis, DIAGNOdent reading		106 subjects	
T0a	Treatment, DIAGNOdent reading			
T3/12	Review, Surface Prophylaxis, DIAGNOdent reading	at 3 months post Treatment start	106 subjects	
T6/12	Review, Surface Prophylaxis, DIAGNOdent reading	at 6 months post Treatment start	105 subjects reviewed, 1 subject dropped out	at 6 months
T12/12	Review, Surface Prophylaxis, DIAGNOdent reading	at 12 months post Treatment start	100 subjects reviewed, 6 subjects dropped out	at 12 months
T18/12	Review, Surface Prophylaxis, DIAGNOdent reading	at 18 months post Treatment start	95 subjects reviewed, 11 subjects dropped out	at 18 months
T24/12	Review, Surface Prophylaxis, DIAGNOdent reading	at 24 months post Treatment start	74 subjects reviewed, 32 subjects dropped out	at 24 months

Discussion

The gradual increase in the number of subjects that failed to attend their recall requests is typical for the general dental practice that is situated in an area with a high turnover of management employees. The dental practice that this study was conducted in draws patients from the Thames Valley area in Berkshire, UK. The Thames Valley is also known as the UK's Silicon Valley, as there is a high percentage of the workforce involved in computing and allied businesses. These employees are mobile in terms of their place of work, and in common with modern employment trends, may have to move location periodically. It is noteworthy to mention that members of the medical profession (excluding dental practitioners) tend to be more mobile in terms of employment opportunities, whereas members of the dental profession, once they have established a practice and the associated high cost of this investment tend to remain static for the remainder of their practicing lifetime.

The immediate drop in the Dv in lesion treated with ozone, and immediately re-assessed with the DIAGNOdent cannot be explained in terms of lesion arrest and reversal, as insufficient time has passed for lesion remineralisation. The observed drop in the Dv value has to be explained in terms of a decrease in the colour chromaticity of the treated lesion. It is known that the DIAGNOdent as part of the evaluation process of a lesion, measures colour. Indeed, amongst some general dental practitioners, (personal communications and feed back from the UK HealOzone User Groups), there has been concern expressed that after remineralisation, there is an observed increase in the Dv, but the lesion has arrested and fully reversed. This tends to be a feature of the larger lesions that have a greater dentine involvement than lesions confined to enamel.

This observation must be seen in terms of the effect of ozone, in whitening or bleaching the enamel and/or the removal of bacterial products. It is postulated those lesions that initially showed a higher Dv (23 or 24) within the 20-24 Dv group may have also been subjected to this effect, but it was not enough to lower the Dv to 19 or below and drop these lesions into a lower group. This feature of the DIAGNOdent was also commented on by Lussi and Francescut in Chapter 2.1, 'Use of the DIAGNOdent in Detecting and Monitoring Caries lesions and Residual Caries for Ozone Treatment' in 'Ozone – The Revolution in Dentistry' Quintessence' 2004²⁷.

In this and every study that has used the HealOzone as the source generator for ozone gas, there has never been any adverse reaction or negative event recorded. The HealOzone's system design, where a vacuum has to be generated around the treatment area *before* ozone is released, is the safest system in the dental market. Whilst there is a plethora of manufacturers of ozone generators, some being actively marketed to the dental profession in various countries around the world, not one of these alternatives has any safety data to show they are safe to use in the treatment of oral disease.

After 3 months, 2 lesions (2%) had spontaneously reversed (CSI1&2). The observed reversal of these two control lesions can be explained in terms of improved oral hygiene, and the use of remineralising pastes and rinses.

At the 6-month recall, 2% of the ozone-treated CSI 1&2 lesions showed signs of re-activated caries despite the use of mineral washes and pastes. The control lesions showed caries progression. It is notable that one of the previously reversed lesions in the control group moved from being arrested, to becoming active. This shows the dynamic nature of caries in lesions where oral hygiene is difficult, and the inability to remove cariogenic material from the fissures. This can be compared to previous studies on root carious lesions where once a lesion has reversed, the lesion then remained stable. Root surfaces are easier to access for oral hygiene measures and this may explain this observation.

At 12 months the data sets show that a single dose of ozone is insufficient to maintain lesion arrest. The control lesions show progressive caries severity. Given that fissures are impossible to clean beyond the surface, it is not an unexpected result.

At the 18-month recall, arrested and stabilised carious lesions in the ozone-treated group show progressive caries severity. In the control group, the rate of lesion degradation is accelerating at a faster rate. This difference between the two lesion groups illustrates the effect of ozone in the complete sterilisation of the lesion and the removal of the bacterial colonies, compared to the control lesion.

After 24 months the study shows the positive result of ozone treatment; 93% of the ozone-treated group show continued lesion arrest and reversal (58% remained in CSI2&1 and 35% in CSI3 showing continued stability). Only 5 (7%) lesions had become worse, moving from CSI1&2, through to CSI3 and then showing lesion re-activation to CSI4. This positive outcome is in contrast to the control

group, where over 51% of the OPFCLs had worsened from CSI3 to CSI4 ($p < 0.01$). From this data alone, it can be seen that carious lesions, if left untreated, will progress and become worse.

Caries reversal is associated with several factors including the level of microbial reduction and the oxidant effects of ozone on POFCLs. The dramatic reduction in microbial flora will have eradicated the ecological niche of the acidogenic and aciduric microorganisms. This shifting of microbial flora to normal oral commensals would predominantly allow remineralisation to occur within the carious process. Previous studies have shown that ozone reduces bacterial by-products and metabolites. The by-products such as pyruvic acid cannot cause further demineralisation of the tooth. In addition, the removal of some metabolites might deny other bacterial types in the lesion their important nutrients. In this way, ozone has a dual effect when used to control caries.

Other studies have shown an oxidant (sodium hypochlorite) can improve the remineralisation potential of demineralised dentine. Inaba^{57, 58} *et al.*, (1995) found that the use of an oxidant (10% sodium hypochlorite) on demineralised root dentine lesions improved their potential to remineralise since sodium hypochlorite is a non-specific proteolytic agent and was effective in removing organic components in the lesions. Further published research in this field by Inaba^(3, 4) *et al.*, (1996), has shown that when root dentine samples were treated with this oxidant for 2 minutes, the permeability of the lesion to fluoride ions increased. The conclusion of this study was that removal of organic materials from dentine lesions was an acceptable approach to enhance remineralisation.

In this light, part of the dramatic remineralisation results shown after ozone application in this and other studies can be accounted for, as it is known that ozone is one of the most powerful oxidants available. It may also indicate that ozone has the ability to remove proteins in carious lesions, and to enable calcium, phosphate and fluoride ions to diffuse through the lesions, a phenomenon resulting in arrest and remineralisation of the majority of POFCLs after ozone application in this study.

The Clinical Severity Index was the primary outcome variable. The DIAGNOdent is very sensitive to stains, which is why the cleaning protocol prior to DIAGNOdent assessment is so important. The

results acquired from this ozone study mirror those from other clinical research trials in other research centres.

Prevention.

Health care models all over the developed world began emphasising prevention as early as the 1960's. For some reason, and in stark contrast to the Medical Profession, this concept has been the most difficult to embrace for the dental profession for several reasons.

In a pharmacological management strategy, there are variety of antibacterial agents available. Xylitol, is not fermented by cariogenic bacteria, inhibits attachment and transmission of bacteria. It can be delivered through chewing gum or lozenges as an effective anticaries program. Xylitol gum chewed by mothers during the first two years of their children's lives led to reduced levels of caries in these children (Lynch H *et al* 2003).

Sodium bicarbonate (baking soda) has antibacterial properties and neutralises bacterial acids. It can be delivered via toothpaste or in a solution in hyposalivatory cases.

Chlorhexidine gluconate is a broad-spectrum antibacterial. It is administered as a 0.12 percent mouthrinse, and it is effective against the mutans streptococci but not lactobacilli. Anderson's 2003 study used chlorhexidine mouthrinse, 10 ml once daily for a two- week period every two to three months. (Anderson MH 2003). In high-risk individuals this therapy may need to be extended over 12 months, with bacterial assessment. It must be administered by the individual or home caregiver, it affects taste, and compliance is often poor (Bird WF 2003).

Iodine in the form of povidone iodine (10 percent povidone iodine, 1 percent available iodine) has been used for high-risk children when applied once every two months but has not been thoroughly proven. (Den Besten PK *et al* 2003). Both chlorhexidine mouthrinse and povidone iodine are effective agents for at-home application.

The dental profession has by its inherent conservatism remained practicing and teaching amputation therapies. This treatment modality is based on sound Victorian tenants that any building or engineering project must be placed on sound foundations. To this end, an area of infection can only be treated by its removal to leave a sound base on which reconstruction can take place. The problem with this approach is that it is often impossible to decide when enough material is sufficient to remove the infection, and when insufficient allows the lesion to redevelop below the new restorative reconstruction. To avoid failure, the dental profession has erred on the side of excessive tissue removal, or 'extension for prevention'. Cohen (Cohen HB 2002) argues that GV. Black's principle of "extension for prevention" has been miss-interpreted. Cohen argues that Black intended that the most experienced operators should choose when to extend, and when not to. For the less experienced majority, the 'universal principle' should be followed to reduce the risk of failure. However, Black was not the first to put forward this principle. In 1883, Webb presented a concept of "prevention of extension of decay". This concept advocated the removal of natural tooth contacts in proximal areas to allow 'preparation of the margins, along with proper restoration contours, to promote natural cleansing of the embrasures with saliva and fluids in the diet' (Osborne JW *et al*, 1998).

Extension for prevention has been an integral part of dentistry for over 100 years. GV Black's 1891 idea of "extension for prevention" was to provide extension of the preparation to the facial and lingual line angles in order to bring about "self-cleansing" margins via food excursion. Black's concept also included extending preparations through fissures to allow cavosurface margins to be on non-fissured enamel. By the 1950's, narrower, more conservative preparations were seen by a few as being more effective in preserving teeth. By the mid-1960's and early 1970's a more conservative approach to amalgam preparation was advocated and was being taught in some dental schools. Today, a standardised outline form is rarely taught or used as a principle of cavity preparation. In areas where fissure caries has necessitated a preparation extending into dentin, a composite resin or dental amalgam restoration should be placed, and a fissure sealant should be used to protect remaining susceptible fissures from carious attack (Osborne JW *et al*, 1998). This current form of the concept of extension for prevention, which is supported by clinical research, preserves sound tooth structure that, using outdated concepts, would have been cut away. Preventing unnecessary extension and allowing sounder tooth structure to remain is one important aspect of helping patients to maintain their teeth for their lifetimes.

However, dental academics are still split over the issue of cleaning or sterilising a cavity prior to restoration. Writing in 2004, Kidd argues that there is little evidence that infected dentine must be removed prior to sealing the tooth. Leaving infected dentine does not seem to result in caries progression. (Kidd EA 2004). Previous studies have shown that caries begins in dental plaque³ (ten Cate JM 2003). If the metabolic activity in dental plaque is the driving force behind any loss of mineral from the tooth or cavity surface, Kidd states that modification by altering the biofilm may be all that is required. This can be achieved by most conveniently by disturbing it by brushing with fluoride-containing toothpaste. The role of operative dentistry in caries management is to restore the integrity of the tooth surface so that the patient can clean. Thus, Kidd argues that the question, 'how clean must a cavity be before restoration?' and the removal of the acid niche environment may be irrelevant. However, this argument stands at odds with the research that shows remineralisation will not occur in an acid-based niche, and there is no medical parallel to leave an area of infection in situ.

The question of how much infected tissue needs to be removed has never been satisfactorily quantified. In part, this is impossible as every infection has a different presentation. Different approaches to provide an answer for this dilemma are possible.

The first approach is to show the dental practitioner the extent of the carious infection. A great deal is known about how a bacterial infection and the demineralisation effects of bio-molecules move through tooth tissue – from the first stage demineralisation of the enamel, to the demineralisation and break up of the collagen matrix in dentine. This de-natured dentine has altered physical properties, one being the uptake of vegetable dyes. Numerous dyes have been marketed to detect denatured dentine. Danville Caries Detector is just one example.

A different approach is to limit the removal of dental tissue by the use of special chemicals or drills. Decalcified dentine becomes leathery then soft then loose debris, as one moves further away from the base of the lesion to the outer oral surface. One possible use of this observation is to design a system that selectively removes the soft and middle leathery layer, leaving the mineralised dentine intact. Two such systems are available for general dental practitioners; To provide tactile feedback, set of hand instruments and a chemical gel that softened denatured dentine only was developed, known as the Carisolv System. When combined with caries indicator dyes, it is a very accurate caries removal system. The disadvantage for the average general dental practitioner was that it takes time to prepare

a cavity, and as time in general practice has a cost attached to it, this type of treatment protocol was expensive. To speed up this process, the hand instrumentation stage was mechanised.

An alternative approach was taken by the introduction of a polymer dental burr. A special plastic was developed that could only cut soft denatured dentine. If these polymers are used to cut into enamel or sound dentine, the cutting flutes abrade and break off (Freedman G *et al* 2003). This alternative approach to conserving tooth tissue allows the dentist to buy into a low-cost conservative system.

The concepts of minimally invasive dentistry were reviewed by Creugers in 2003 (Creugers NH 2003), and Ericson in 2004 (Ericson D 2004).

Creugers describes the mutual relations between three important dental concepts; 'Minimal invasive dentistry', 'adhesive dentistry' and the 'dynamic treatment concept', concepts that have changed restorative dentistry substantially during the last decade. The ultimate goal of restorative dental care - , is the maintenance of a healthy and functional dentition for life - remains unchanged. However, Creugers states that the need for monitoring and aftercare of restorative work is increasing as invasiveness of interventions is decreasing. Ericson writing in 2004 has help define the evolution in these concepts. Ericson defines 'Minimally Invasive Dentistry' as the application of "a systematic respect for the original tissue", the common delineator being tissue preservation, by preference preventing disease from occurring and intercepting its progress. If the disease process is already established, the aim of minimally invasive dentistry is to remove as little tissue as possible. It does not suggest that the dental profession should make small fillings to restore incipient lesions as routine procedures.

The introduction of predictable adhesive technologies and innovative materials has led to move towards minimally invasive dentistry in dental practices who are involved in the change from tissue amputation, to those where tissue preservation is the goal. The evidence-base for survival of restorations clearly indicates that restoring teeth is a temporary palliative measure that is doomed to fail if the disease that caused the condition is not addressed properly. The question raised by Kidd perhaps is retrospective, given that the dental profession has the means to quickly sterilise a lesion, without the need to remove infected tooth tissue.

Today, the means, motives and opportunities for minimally invasive dentistry are at hand; sadly, the incentive, financial or otherwise, for a dental practitioner is definitely lacking. The paymasters for the majority of dental professionals are either centralised government departments, like the United Kingdom National Health Service & Department of Health or insurance companies, such as Denplan in the UK, Medicare in South Africa, Delta in the USA and Blue Cross in Canada for example. It is easier for these bodies to pay on work that can be seen to have been performed, than for work that involves a concept of education and home care. The United Kingdom National Health Service, (UK NHS) for example has struggled with the UK Dental Profession to change the way socialised dental services are remunerated. For many years, payment was made on the actual work carried out, much of it destructive – the cavity drilled and filled, the tooth extracted, the x-ray taken. This Item of Service payment system is largely historical, and its cost to the taxpayer huge. Recent attempts to change this in favour of a preventative approach by establishing oral health care clinics, and the teaching of prevention have not been met with universal acceptance, as it seems that a lower value is placed on this service by the funding authorities. Patients and third parties seem to be convinced that the only things that count are replacements. Namely, they are prepared to pay for a filling but not for a procedure that can help avoid having one. Also, the public have a real problem in accepting responsibility for their own health. Many would prefer to abrogate their responsibility to the dental or medical profession, in the misconception that the professions can fix any health-related problem.

Despite the body of evidence to suggest that the overall cost of 'drilling and filling' treatment is greater than prevention, there is little encouragement from within the profession to move to a preventative role in preference to an amputation role. The dental profession is torn between preventive oral health systems versus the traditional surgical or amputational treatment for oral disease.

The dental professional has been taught from a very early stage in undergraduate training, that the only way to treat decay is to cut it out, until a sound base has been achieved. This 'Victorian' amputation approach to micro engineering has been taught at dental teaching facilities around the world for decades. Only then can traditional materials, such as amalgam, glass ionomer and composite materials have any measure of long-term success. Throughout this time, the design of cavity has changed and evolved as more conservative treatment protocols and materials that are more modern

have been made available to the dental profession. Yet in a recent abstract, the results of a study that combined Atraumatic Restorative Treatment (ART) with ozone treatment showed the following results; even where a soft dentine base was left, after ozone treatment acid-etched resin bonded composite fillings are stable and functional at 6 months post-placement (Holmes J 2004). This flies in the face of current teaching, and points to the requirement of further research to show if this observation can be reproduced. If it can, what are the processes that allow it to be successful? The necessity to amputate large volumes of tooth material would then disappear, to the patients benefit.

Unpalatable as it may be sections of the population, mass medication in the form of water fluoridation has had a huge impact into the prevalence of dental disease, and has been one of the major contributions to improve oral health in the UK, USA and Europe in certain age groups. This success in the 1960s and 1970s of water fluoridation in reducing the prevalence of dental caries was mirrored in the late 1970s and 1980s by the introduction of fluoridated dentifrices and other fluoride products such as mouth rinses.

Fluoride inhibits demineralisation and enhances remineralisation. At certain concentrations, it can inhibit bacterial activity. Topical application of fluoride is more effective than a systemic source and include drinking water; toothpastes and gels and over-the-counter fluoride rinses. Professionally applied fluoride can be in the form of office topical varnishes, foams, gels, acidulated phosphate fluoride, and stannous fluoride. High-concentration fluorides should be used with great care in children due to the risk of ingestion and fluorosis. As they some sources are self-administered, compliance is a major problem. Patients must be persuaded as to the need to use these products, and that parental supervision is critical for a successful outcome.

The dental profession is aware that preventative regimes can reverse caries in some cases, but that these are not predictable. If the results of previous European studies were to be extrapolated to this study, and the comments by Kidd (Kidd EA 2004) that brushing and fluoride are all that is required, it would be expected that a greater number of lesions in the control group would arrest and reverse, just with improved oral hygiene and care. In tandem with this, there should be only a few lesions in the ozone-treated group becoming progressively worse with time. In this study, this was not observed, and there are two potential reasons why not; First, in the real world, in every dental practice across the world, people do not always follow instructions (Bird WF 2003). They often are in a stressful situation,

so the oral care message is often partially heard, if at all. Secondly, the unique morphology of the occlusal pits and fissure make dealing with these surfaces unique challenge, so the lessons learnt with buccal caries cannot be simply extrapolated to carious lesions in different tooth surfaces.

Ozone is one of nature's most powerful oxidants, which accounts for its ability to kill bacteria, spores and viruses. Unfortunately, many studies concerning the clinical evaluation of ozone have been based on assessments of its harmful effects rather than demonstrating any therapeutic benefits it may offer. It has been used for many years for the treatment and purification of water sources and it has been used in various aspects of medicine. This powerful antibacterial feature of ozone is very promising and published research has shown the potential application for caries control.

The young patient's attitude to future dental care is often highly influenced by traumatic dental care in the early years. With increasing age and anxiety about dental treatment, dental care is often postponed until more radical treatment is required due to experiences. These situations can be overcome using early intervention strategies. In this respect, the use of ozone should be considered especially for the young, medically compromised patients, those who are in long-term care, domiciliary care patients and homebound people ¹⁹ (Baysan A *et al.*, 2001). There is no injection or tissue amputation involved in ozone treatment and the ozone delivery system is portable. Research by Domingo *et al* (Domingo *et al* 2001) has already shown that this treatment modality is very acceptable to the public.

Ozone has been demonstrated to be an alternative treatment methodology to traditional 'drilling & filling'. It is safe, predictable and cost effective. Research has show that ozone treatment has the potential to have a major role to play in the treatment of carious lesions. This may have considerable cost implications in developing countries with limited resources.

This study also showed an important feature of carrying out research in a highly mobile population group. The Thames Valley is an area that serves the London-based employment market. The area is mainly blue-collar company employees, involved in managerial jobs. They are very mobile, in terms of their location, and relocation as career and change of employment opportunities arise. The dental practice that this data was collected in is in the middle of this area, and the number of subjects that

dropped out over the 24-month study period is a reflection on this employment-related migration from one area to another. The loss of 32 subjects that accounted for 30% of the initial study group has obviously had an effect on the results. However, this gradual depletion of the subject numbers does not diminish the study value, and it should be seen as a pilot study for a larger group, or several groups from around the country with collated data, to show the effects of this treatment modality in different social areas of the United Kingdom.

Writing in 2002, Young ²(Young DA 2002) commented that the emphasis should be placed on early detection. The DIAGNOdent is one of the new detection technologies commercially available to help detect carious lesions early. What is now required is a clear strategy on how to merge these new technologies in a way consistent with today's changing paradigm of caries management. There is a real danger that these early detection technologies may be used over aggressively to justify tissue destruction or "drill and fill" using Black's "extension for prevention" preparations. Use of the CSI, Lussi's guide to the DIAGNOdent, air abrasion and the HealOzone are synergistic technologies that fulfil the requirements of not just detection & assessment, and minimal preparation. The integration of these technologies, with proper training and education leads the way to micro-preparation and intervention for the future management of caries in the general population. Concurrent with these technologies, the fear and anxiety of dental intervention (Holmes Smith etc) can be addressed with a positive message being sent to the public; that dental care for the 21st century has evolved at last towards enabling homeopathic healing to take place, replacing the traditional 'drill-n-fill'.

This study has shown that ozone can be used to manage occlusal pit and fissure caries. In comparison with earlier studies, the management of occlusal caries requires a different management strategy, due to the morphology and access to the carious lesion. Buccal caries can be managed without the need to place a sealant or filling, as access to the lesion's surface is very easy. Pit and fissures present with a different morphology. The lesions are within areas that are difficult to access. The size disparity between the fissure orifice and the bristle tip of toothbrushes designed for home-use, make the cleansing of surface impacted debris and the removal of the organic plug impossible. The placement of a sealant for these surfaces is therefore of paramount importance to maintain the sterility ozone-treated lesion. The choice of sealant will determine the potential for remineralisation.

Various flowable composite resin based systems are available to the general dental practitioner. It is questionable if resin-based materials are able to release sufficient bio-available minerals for the remineralisation of ozone-treated carious lesions in the initial stages of 'healing'.

Biomaterials are available for minimally invasive cavity treatment. Fluoride and mineral-containing restorative materials, including glass ionomer products, help prevent further decay at the site of placement. Glass ionomer materials have a long history of use as a source of minerals for remineralisation. With the advent of FujiVII (GC Japan) and Tooth Moose, that incorporates Recaldent™ (GC Japan and Cadbury-Schweppes), the choice of material use becomes clearer. These new and innovative materials promote remineralisation in the acidic and basic-phases of salivary flow. Recaldent is a water based, sugar free dental topical crème containing Recaldent™ CPP-ACP (Casein Phosphopeptide - Amorphous Calcium Phosphate). It is derived from the milk protein, casein. For many years, it has been known that milk and its derivatives have a tooth protective effect. Recently, research has shown that this activity is due to a part of the casein protein called Casein Phosphopeptide (or CPP), which carries calcium and phosphate ions 'stuck' to it, in the form of Amorphous Calcium Phosphate (or ACP). This complex of CPP-ACP (Recaldent™) is an ideal delivery system for bio-available calcium and phosphate ions.

The first product for professional use that contains Recaldent™ technology is GC Tooth Mousse. GC Tooth Mousse incorporating Recaldent™ promises to make an important contribution to protecting the oral environment in a wide range of situations where a mineral imbalance may arise.

FujiVII (known as Fuji Triage in the USA) is a pink-coloured glass ionomer cement that has been advocated as a primary sealant for pit and fissures, as well as buccal cavities. It contains a greater concentration of bio-available fluoride than other glass ionomers. The pink colour has created a unique polarisation of dental practitioners who either like it – or refuse to use it. This reaction to the colour is the result of a failure to understand the reasoning behind the colour. It is a transitional type of material that can be used for a period to allow remineralisation before the definitive filling material is placed, or to show the patient as well as different dental reviewers that something special has been applied to the treated surface. In the case of post-ozone treatment and where caries extended into dentine, the lesion should undergo a period of remineralisation prior to the placement of any long-term

sealant or restorative material. However, various studies where FujiVII has been used for the transitional restoration of the occlusal and buccal surfaces have shown at 6 months or longer that whilst there are signs of surface wear of the material, the retention rate of FujiVII is very good. Moreover, patients themselves rarely have an issue with this type of material, when the reason for its' choice is carefully explained.

In Holmes J and Lynch E.⁴⁴ (Holmes J and Lynch E 2004) ozone has been shown to clinically reverse early pit and fissure carious lesions. This study assessed the hardness of ozone-treated dentinal caries 3 months after sealing. 38 Subjects were recruited in a general dental practice, each with two non-cavitated occlusal carious lesions with radiographic radiolucencies extending 2-4mm into dentine, In each Subject, lesions were randomly assigned to either; Group 1; where air abrasion was used to remove the unsupported enamel over the lesion and remove soft dentinal caries until the hardness was scored as leathery after probing. Approximately 1 mm of leathery caries at the advancing front of lesions was not removed. Ozone was then delivered for 40 seconds, a mineral wash applied and each lesion was sealed with GC FujiVII, a mineral-releasing glass ionomer. After 3 months, the glass ionomer was carefully dissected and the floor of the cavity again probed, and then restored with a posterior composite. At 3 months all Ozone-treated dentine caries was hard and required no additional removal. The conclusion of this study was that treatment using air abrasion, ozone and sealing was associated with reversal of caries, more conservation of tooth structure and less sensitivity pain than conventional drilling and filling.

The concept of 'Technology Aware and Abuse Syndrome' (TAAS) was introduced by Holmes in 2004²⁷ (Ozone – The Revolution in Dentistry. Edited Lynch E, Published Quintessence London, ISBN 1-85097-088-2) Dental practitioners need to be aware of this potential, and stress the continuance of 'at-home' oral care, with conventional use of dentifrices and dental brushes. This abuse of modern technology has already been seen in medicine. For example, chronic alcoholics in terminal liver failure have had liver transplants, only then to continue their alcohol abuse. Likewise, chronic smokers in heart or lung failure often fail to curb their habit after organ transplant, and go on smoking with their new donor organ. Whilst some may consider the comparison unfair, the outcome is essentially the abuse of modern technology allowing the individual to continue with a habit that has a negative health-related outcome, at the expense of a centrally funded health care system.

Fortunately, the numbers of individuals involved in TAAS tends to be small in number. In the case of treatment with the Healozone, where the technology is cheap, easy to apply, access to it is via the practitioner in primary care, and the technology is proven, I suspect that dental practitioners will see a growing number of individuals who will abuse the technology.

The profession needs to tackle the problem of cariogenic bacterial transmission from mother to child (Berkowitz RJ 2003). By preventing primary infection by mutans streptococci, the risk for future dental caries is reduced (Featherstone JD *et al* 2002). Young suggested that dental caries should be treated as a curable and preventable infectious disease² (Young DA 2002).

To reduce the risk to children, parents, especially mothers, with high caries profiles should receive aggressive dental care. Therapy should eliminate all active caries lesions, provide dietary counselling and use topical antimicrobial agents. Education of mothers about the transmissibility of caries-causing bacteria, how dental decay occurs, and how it can be prevented should be included both pre- and post-natal counselling.

Caries Risk Assessment should be part of a daily management tool for all patients. Risk assessment starts by a thorough and comprehensive examination, not only of the oral tissues, but encompassing diet, habits such as smoking, frequency of brushing, bacterial sampling, and so on, until a full picture of the individual is built, and a targeted treatment protocol can be formulated, drawing together oral care, home care, and restorative care. Computer generated risk assessment is possibly the easiest, as research can be used to weigh positive or negative factors to arrive at an assessment. The O H Manager, DOCexpert Group, Germany collates information from the examination, known risk factors, periodontal screening and the DIAGNOdent. It then presents the dental team with a risk analysis for that patient, and from this analysis flow various treatment recommendations and plan options.

Conclusion.

Ozone has been shown to play an important role in the management of caries. Studies of buccal lesions showed that a single treatment session with ozone reversed nearly 90% of these lesions⁴⁷ (Baysan A 2002), with another showing 100% arrest and reversal when a periodic ozone treatment protocol was instituted¹⁴ (Holmes J 2003). Buccal caries can be managed without the need

to place a sealant or filling. In the case of occlusal pit and fissure caries, a different management protocol is required, due to the morphology and access to the lesion. Due to the different morphology of pits and fissures, the lesions are difficult to access and maintain. The placement of a sealant for these surfaces is therefore of paramount importance to maintain the sterility of the ozone-treated lesion. Freedman G *et al*, comment that "Watch and Wait" is no longer an acceptable treatment. (Freedman G *et al* 2000). UltraConservative dentistry represents a great step forward for the dentist, the profession, and particularly for the patient. It involves the early detection and complete elimination of all accessible and non-accessible carious material from the tooth. Untreated carious lesions, though they may be small, can be extremely and very rapidly destructive. The earliest interception of decay maintains total dental health, and increases the likelihood of the restored teeth lasting a lifetime.

In terms of costing such a treatment protocol, In Domingo H *et al* (Domingo H *et al* 2004), Data from 20 randomised, controlled, double-blind, clinical trials, showing the effective reversal of dental caries after ozone treatment were compared. The results were used to extrapolate the potential savings to centrally-funded dental healthcare. The conclusion of this study was that ozone treatment significantly reversed decay in every study with reversal range of 83%-99%. In 2 of the 20 studies, when ozone treatment was combined with the daily use of a remineralising toothpaste, mouthrinse and spray, the reversal of caries was 99% The success rates were lower in the populations with a higher caries incidence and prevalence than in populations with a lower caries rate. Ozone treatment was most effective for early pit and fissure caries as well as non-cavitated primary root caries.

Ten Cate has commented that as the rate of progress of carious lesions is reported to have slowed, so there is at last an opportunity for a directed interventional and preventive approach, rather than a restorative approach.

This study shows that a single ozone treatment is not sufficient to maintain a caries-free occlusal surface for more than 12 months where no sealant was placed after ozone application. A different treatment protocol is required for occlusal decay when compared to studies involving ozone and buccal lesions; this should include the placement of a fissure sealant of some description to prevent fissure debris and bacterial ingress, and/or the re-application of ozone on a regular basis.

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