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Charlatans in Dentistry: Ethics of the NICO Wars

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Robert E. McMahon, DDS

Ignaz Philipp Semmelweis would have understood. He was a Vienna-based Hungarian physician who in the 1840s, before the germ theory of infection existed, established through a case-control investigation that the simple act of washing hands between patients, and especially between the performance of an autopsy and a patient examination, dramatically reduced the mortality rate in his maternity ward.¹ At the time, postpartum deaths from septicemia were occurring at an alarmingly high rate throughout Europe. In a hospital ward supervised by Semmelweis one in five young mothers died after giving birth. With hand washing this rate fell to a negligible number. When hand washing was discontinued the rate rose again. Results were published in the *Vienna Medical Society Journal*, one of the most widely read journals of its time, and confirmed by U.S. and British reports.

This convincing experimental evidence should have persuaded all physicians, and especially obstetricians, to take up the habit in earnest. However, while his advice was followed by many progressive physicians within his own country, in many other quarters his work was treated with ridicule and abuse. Semmelweis was thought to lack proper respect for the knowledge and authority of his elders. Physicians felt that more concern should be given to a doctor's dignity than to the needs of females. Hide-bound tradition prevented many from altering practices which had remained unchanged since the Dark Ages.

NICO, the Disease

The authors of the present essay have had the privilege and the headaches of becoming personally involved in a similar Semmelweis scenario. This scenario requires a clinician to become knowledgeable about relatively common but obscure disease processes not previously well-understood: ischemic bone disease and low-grade infection of the maxillofacial bones. These processes for several reasons are challenging for the clinician and disheartening for the patient.

Firstly, the associated pain is often very severe, neuralgic and intractable. Secondly, since these are marrow diseases there is little alteration of the surface mucosa (edema, erythema, etc.) or the radiograph, even in severe cases. Thirdly, clinicians are tempted and trained to use a facial neuralgia diagnosis for

idiopathic pain once tooth- and sinus-related causes are ruled out. Therefore, the clinician has difficulty including the disease in a differential diagnosis, appropriate marrow evaluation is not done, and the patient must suffer excruciating, unbearable pain as well as doctors who repeatedly suggest that the pain is psychological or psychosomatic.

This disease process is now called NICO (neuralgia-inducing cavitational osteonecrosis), a name emphasizing its two most unique features: the neuralgia-like character of associated pain and the very unique ability to desiccate and hollow out large marrow spaces. Pain-free cases also occur, usually referred to as silent NICO or maxillofacial osteonecrosis.

Our profession's involvement with this disease process began early, coinciding with the birth of modern, organized dentistry. Initially called "bone caries" because it was considered to be an infection without pus (like tooth caries today), it was not an uncommon problem in the nineteenth century world of heavy metal pollutants and malnutrition (Ferguson, 1868; Noel, 1868). The disease essentially disappeared from the twentieth century dental literature until a flurry of research activity toward the end of the century dramatically increased our understanding of its pathophysiology and our ability to appropriately diagnose it.

It is now understood that NICO is the jawbone version of a skeletal disorder, termed ischemic osteonecrosis, avascular necrosis, aseptic osteomyelitis or bone marrow edema, with a long and active research history in the orthopedic surgery literature (Bouquot & McMahon, 2000; Neville, Damm, Allen, & Bouquot, 2002; Urbaniak & Jones, 1997). This disease is not so much a disease in its own right as it is an end-stage result of poor marrow blood flow and stagnation, usually associated with one or more inherited hypercoagulation states and often with a superimposed low-grade bacterial infection (Bouquot & McMahon, 2000; Gruppo, Glueck, McMahon, et al, 1996).

Potent neurotoxins are produced in the diseased marrow and are capable of damaging the myelin sheath of trigeminal nerves, sometimes with the adverse development of blood-born autoantibodies against myelin throughout the body (Bouquot & McMahon, 2000; McMahon, Bouquot, Mahan, & Saxen, 1998). The prevalence rate for biopsy-proven NICO in adult females, 1 per 2,000, is considerably greater than the adult male rate of 1 per 20,000 (Bouquot & McMahon, 2000). Technetium radioisotope scans, quantitative ultrasound and diagnostic anesthesia testing are more appropriate diagnostic tools than radiology for this disease (Bouquot & McMahon, 2000; McMahon, Griep, Marfurt et al, 1995; Neville, Damm, Allen, & Bouquot, 2002; Urbaniak & Jones, 1997). And a series of follow-up investigations has determined that at least 72% of affected patients experience dramatic pain relief after surgical curettage of affected bone (Bouquot & Christian, 1995; Bouquot & McMahon, 2000).]

The histopathology, etiologies, clinical features, and therapies for NICO are similar to those of other affected anatomic sites. Regardless of the site involved, however, the disabling pain, the well-known diagnostic subtleties and the demanding nature of the therapies make ischemic bone disease one of the more problematic disorders in humans.

The Controversy

So what does all this have to do with Semmelweis? In one sense, many clinicians would like to “wash their hands” of this disease. It is too difficult, too time consuming, and treatment is too often unsuccessful. This has led many clinicians to simply avoid the diagnosis. However, an additional and potentially greater problem emerged in the 1980s, when the disease became intimately associated with idiopathic facial neuralgias.

At that time NICO, under several diagnostic names, became one of the great controversies of dentistry, termed by some the “NICO wars”. While a reasoned progression of peer-reviewed publications has been adding to the literature, outside the literature the dialogue has more typically been characterized by personal opinions and belief systems, and the tools of “debate” have too often been ridicule and abuse. The healthy and necessary scientific debate of new concepts and disease characterization is almost completely lacking in this arena. The debate seems, rather, to be some sort of religious argument where scientific facts need not be applied.

Enter the charlatan: “a pretender of knowledge and skills that one does not possess, also called a quack” (Anderson, 2000). Usually those referred to as quacks or charlatans are individuals using practices *without* a corroborating literature base. In the NICO wars these disparaging names are certainly bantered about with remarkable frequency, but the most intriguing feature to us is the fact that the charge of quackery has been turned on its head. The clinicians being labeled as quacks are those *actually using* as source material a published literature of more than one hundred and sixty peer-reviewed papers, abstracts and book discussions pertaining to head and neck lesions (www.maxillofacialcenter.com/osteonecrosis, accessed 9-28-03). Moreover, *this* literature uses as *its* base more than two thousand publications in the orthopedic surgery, rheumatology and laboratory medicine literature.

The NICO literature is filled with microscopic and clinical photos and descriptions, published by a variety of dental specialists, and a very extensive review was published in 2000. To date not a single research paper has been published in a peer-reviewed setting which refutes in any way the data provided by the NICO literature. Yet, it is not unusual for a dentist to dismiss ischemic bone disease out of hand by

simply stating that NICO does not exist, and then proceed to belittle or threaten legal action against those clinicians willing to help NICO patients (Doddie & ??, 1996; ?? & Epstein, 2002; Baratz, 2001).

The reason for this completely escapes us, even though the irony has not. Moreover, the vehemence of the “anti-NICO” attacks has been so severe and so anti-intellectual that it lends wonderful credence to the old adage that those who know the least about a subject have the strongest opinions. We think that Semmelweis would have understood.

Attacks in the litigious climate of modern America often take the form of nuisance lawsuits and threats from state licensing boards. Within our own states, where we are well known, we have had no legal problems relative to this disease, but attacks have occurred with some regularity from individuals who do not know us and apparently do not read our published work. One of us (JEB), for example, has been accused in legal suits of actually *inventing* the disease in order to enhance his income (Baratz, 1995). He has been legally charged with fraud, conspiracy, malpractice, even assault and loss of conjugal relations.

Thus far legal attacks have had little or no consequence for us, other than making us more visible and thereby helping to grow our practices. It is clear, however, that much of this activity has been encouraged by the quintessential and now largely discredited, self-appointed vigilante group, the National Council Against Health Fraud. This group, which seems to believe that the worst sin in dentistry or medicine is to be unconventional, has for years suggested that any patient receiving a diagnosis of NICO should report it immediately to their state dental board and consider filing fraud and RICO (Racketeer Influenced and Corrupt Organizations Act) conspiracy charges against any “quack” who would be foolish enough to diagnose ischemic bone disease in the jaws (www.quackwatch.com). They have long reported that dentists in multiple states have lost their license because of “Dr. Bouquot’s diagnosis...or tax fraud.” Technically, the latter charge is true; many states must have dentists who lose their license because of tax fraud. But the former statement is *completely* untrue and, in fact, represents the opposite of truth. We have, in fact, been instrumental in helping to *keep the licenses* of NICO surgeons being attacked by their boards.

After a few initial successes more than a decade ago, prior to the development of a substantial body of peer-reviewed literature, this Council has not fared well in their attacks against NICO doctors and alternative health care providers in the courts. Their lead expert witnesses have made so many unsubstantiated claims in hearings and court proceedings that they no longer have credibility and some have even had contempt of court charges filed against them for falsifying their qualifications (State of Florida v. Phillips, 2002). A recent California suit was summarily dismissed because the “quackwatchers” expected the court, according to the judge, to merely accept their opinions without a shred of substantiating

evidence (<http://www.humanticsfoundation.com/quackwatchwatch.htm>). Their “experts” have admitted to earning considerably more from testifying than from clinical practice...perhaps a reason for them to continue to fuel the controversy? The “organization” seems actually to be in disorganization and will thankfully soon fade into the past as just another bad idea in American dental politics.

Attacks from state licensing boards have also occurred, sometimes with a rather personal touch. At least one contributor to the biopsy service of one of us (JEB) was told by her board that her license was in jeopardy unless she quit treating a disease which did not exist (State of Washington v. Hopkins, 2002). She was also told that she could not take continuing education courses from Dr. Bouquot and all biopsies sent out of state must receive a second opinion from the state university lab. This board so overstepped its role, and had so little to base its actions on, that the attorney general's office of that state has promised a written apology to Dr. Bouquot.

Other board activity has been worrisome, to say the least. One board took the surgical privileges (not the license) away from a general dentist treating NICO patients without a hearing or an unbiased investigation, largely based on the word of a single dentist (State of Iowa v. Allender, 1995). Several boards have refused to accept our histopathologic diagnoses, and sought a diagnosis more to their liking (i.e. anti-NICO), even after two, three or four nationally recognized oral pathologists confirmed marrow disease from review of the original microscopic slides (State of Arizona v. Lee, 1997; State of Washington v. Hopkins, 2002). Other boards simply refused to accept as credible *any* expert witness who used the dental literature as the basis of his or her opinion, while accepting, seemingly without question, comments made by quackwatch-associated individuals who could not review the literature and admit to no experience with the disease (State of Florida v. Phillips, 2001).

Certain boards have shown such a bias against NICO that they would only behave honorably when exposed to regional and national TV coverage (State of Arizona v. Lee, 1997). Other boards arranged for publication in statewide newspapers of fraud charges against NICO surgeons *before* a hearing was even scheduled, or were so confident of success that they let the local state dental journal publish an announcement of license revocation *before* the defending dentist, who was exonerated, had his hearing (State of Iowa v. Allender, 1995; State of Arizona v. Lee, 1997).

Sorting It Out

By now the controversy has greatly diminished, although there are a few who “remain proudly standing in the middle of their own preconceptions with the stolid air of one who is quite prepared to wait until

continental drift budes him from his position" (Pratchett, 1998). It seems worthwhile, then, toward the end of the battle, to offer a few comments and reflections stemming from our own experience in the "wars". On a personal level, work in such an arena of controversy has been filled with dichotomies. On the one hand, we have been well received during the delivery of more than 100 research presentations and invited lectures at universities and national or international scientific meetings. On the other hand we have at other national meetings, and not in our presence, been literally called quacks. While we have been visiting with most of the world's authorities on the histopathology and treatment of osteonecrosis of the jaws, hip and knees, learning much from the exchange of cases and concepts, we have been publically accused of not knowing the basic differences between normal bone and ischemic bone disease by persons with no obvious experience with stromal marrow disorders. While receiving an award for outstanding research, having a paper featured as the cover story of a highly respected medical journal, and being recipients of a major research grant (Residual Infection in Bone project of Indiana University Medical Center), we have been publically accused of publishing worthless research. While knowing first-hand the tremendous improvement experienced by a large proportion of our treated patients, and receiving microscopic confirmation of hundreds of cases, we have been vilified for treating a disease which some claim to be nonexistent.

It has been quite a journey for us, to be sure, and one which we would gladly undertake again. While we have emphasized the negative in the present essay, we could not conclude without also mentioning the tremendous dedication, compassion, innovation and intelligence which has characterized, in our opinion, the growing group of professionals interested in this fascinating disease process and the patients afflicted by it. Their willingness to continue in the face of inexplicable resistance and harassment is truly remarkable, and we wish to thank them for accepting their role on the front line of a new human endeavor. We think Semmelweis would have understood.

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Understanding Jawbone Cavitations and Their Relationship to Disease

by
Susan Stockton, MA

In 1993 two critically important books in the field of remedial dentistry were released, these are: "It's All in Your Head," by Hal Huggins, DDS and, "Root Canal Cover-Up," by George Meinig, DDS.

For many, an awareness of the problems posed by the use of mercury-containing dental amalgam, the so-called "silver" filling, as well as root canal procedures began with the information presented within these books. We learned that these dental practices could be the root cause of many seemingly unrelated systemic or whole body problems.

Less than a decade later we're beginning to discover that iatrogenic or physician-induced illness in the dental arena is not limited to mercury and root canal treatments. The public is beginning to become aware that systemic illnesses resulting from the toxic output of "focal infections" in the upper and

lower alveolar or tooth-socket containing bones of the mouth can exist even in the absence of amalgam and root canal fillings.

A "focus" is a enclosed volume of necrotic or dead and/or infected tissue that produces concentrated toxins. Such an alveolar focal infection site is often silent in the sense that it produces no local symptoms, but its toxic output can affect any organ in the body once those toxins gain systemic access.

Although over 70 factors contributing to focal development have been identified, the chief initiating factor in such infection appears to be trauma to the jawbone, with such trauma being physical, bacterial and/or toxic in nature. The most common initiating physical trauma appears to be tooth extraction as it is commonly performed.

Most of us lost our wisdom teeth, our 3rd molars,

early in life either because either they were causing problems or considered likely to do so. Ironically, the conventional extraction of a wisdom tooth, or any other tooth for that matter, to alleviate or prevent problems can finish up causing deep, long-standing problems if inadequately done.

When extracting a tooth, most dentists and oral surgeons do not take the time to ensure that the remaining periodontal ligament that once bonded the now extracted tooth into its bony socket is completely removed. Nor do they remove a thin layer of the harder, cortical or surface bone, lining the vacant socket and to which the extant periodontal ligament remains bound. Such removal or debridement of the ligament is needed to allow the softer, marrow-supporting cancellous bone within the outer, cortical bone to bleed into the surgical site.

Left in-situ, the remaining portions of the no longer functional periodontal ligament form a barrier to healing by interfering with critically needed blood flow into the area.

Although the extraction site might well *appear* to have healed properly, it is usual for holes or pockets to form beneath the thin "roof" of cortical bone that will eventually grow across the top of the socket.

When "oxygen breathing" or aerobic bacteria

are trapped in such an oxygen-free or anaerobic environment they change form to become continuous producers of *extremely potent* toxins reckoned in some cases to be ten times as neurotoxic as the mustard gas used in WW I.

The pocket or cavitation in the jawbone has thus become an invisible incubation chamber for microbes whose tremendously toxic and continuous output of waste products weaken the entire body.

When our bodies are young and vital the immune system is usually strong enough to seal off the output from toxin producing cavitation sites from the general circulation. As we age however, and in the form of injury and illness accumulate more stress to the body, our immunity tends to decline and the silent infection in the jawbone now begins to spread toxins into the body via the blood and lymph systems.

Once circulating, these toxins tend to settle into the most highly stressed, and therefore most weakened organs. Any disorder in the body can therefore originate in the jawbone even though there may be no pain or discomfort there. As well, and worse yet, the teeth around a cavitation site are eventually affected for they tend to die a slow death due to insufficient blood supply.

Knowledge of focal infection has been available in medical circles for a long time but has been buried, obscured by prevailing medical teachings that do not recognize the unimpeachable realities of focal infection.

In recent years a steadily growing number of progressive dentists and doctors have become aware of the damage caused by the conventional and entirely inadequate tooth extraction procedure as well as other routine dental procedures.

These pioneers have sought to both avoid and correct such damage by using proper extraction technique, avoiding root canal procedures and treating existing jawbone cavitations surgically.

Surgical removal of necrotic bone is an essential component in eradication of the oral focus and a necessary pre-condition for healing to occur.

Traditionally, the challenge for the dentist treating jawbone cavitation sites is to first locate them and second, to gain accurate information about their physical size and depth, etc. None of the diagnostic tools traditionally used in medicine and dentistry, including in particular the X-ray, was designed for this purpose and none therefore is particularly useful for such diagnostic investigation. There has been a

clear and pressing need for an effective and safe imaging system capable of giving more detailed and precise information about jawbone cavitations.

Through the pioneering efforts of Bob Jones of Aurora, Colorado, this longstanding need is now being met. Bob took an avid interest in dental problems in 1992, when he discovered that his own life-threatening neurological problems were the result of mercury toxicity, root canal treatment and jawbone cavitations. Confined to a wheelchair and given only six months to live, Bob's long standing and severe symptoms greatly subsided following mercury amalgam removal, extraction of root canal filled teeth, extensive cavitation surgery and ongoing detoxification to rid his body of accumulated poisons and toxic mercury.

Even before his recovery, he set out to develop an instrument designed to detect jawbone cavitations and as a design engineer with a background in sonar technology, Bob was convinced from the outset that such an imaging device could be developed using through-transmission ultrasonography or high frequency sound waves transmitted from one side of a bony structure and through the bone to then be "picked up" on the other side.

He and his son and numerous field researchers both local and far flung worked diligently over a period

of eight years to make the vision of a perfected CAVITAT the reality it has now become.

The CAVITAT's computer-driven imaging system and ultrasonic transmitting and receiving devices are engineered to reveal only bony structures, not soft tissue. This is the opposite of what other ultrasound devices used in the medical field do in that they show only soft tissue, not bone.

Sound waves travel readily through bone that has a good blood supply and is healthy. The result of ultrasound transmission through such bone is displayed on the CAVITAT's computer screen as a three dimensionally color-coded image that is green in the case of healthy bone.

Where a cavitation is present, the sound waves are unable to penetrate the soft-mush filled space as easily and in such cases the image displayed on the screen is red.

When only marginal blood supply is present, and the bone is therefore dying, the image displayed will be yellow or orange.

The 32 3-D images displayed on the computer screen, one for each tooth, can be rotated and viewed from any angle giving the dentist detailed information about the condition of the jawbone that is an invaluable

guide to a successful surgery.

I have seen firsthand the results of this technological breakthrough as my own long-standing health problems improved immensely after successful jaw surgery made possible by the CAVITAT.

I started writing about jawbone cavitations in 1998, and my book, "Beyond Amalgam: The Hidden Health Hazard Posed by Jawbone Cavitations," was released in 1999, but until the CAVITAT came onto the market in early 2001 I had very poor bone healing from previous surgeries because my surgeons were unable to determine the locations and extents of all the necrotic volumes of jawbone, upper and lower, and were therefore working on a "cut and try" basis; unable to find and remove them all.

The advent of ultrasonic bone sonography offers real hope both for dental patients and those chronically ill individuals whose conditions might have their roots in a silent jawbone condition.

Based on my experience, and given the prevalence of jawbone cavitations, I believe a CAVITAT scan should be a standard part of every routine dental exam or every annual physical exam due to the systemic problems that can stem from these cavitations.

Maxillofacial Osteonecrosis Chronic Ischemic Bone Disease

Multicystic discolored "mush" of posterior mandible in a patient with atypical facial neuralgia/pain. Photo: Journal of Oral Pathology & Medicine 1999: 28:423

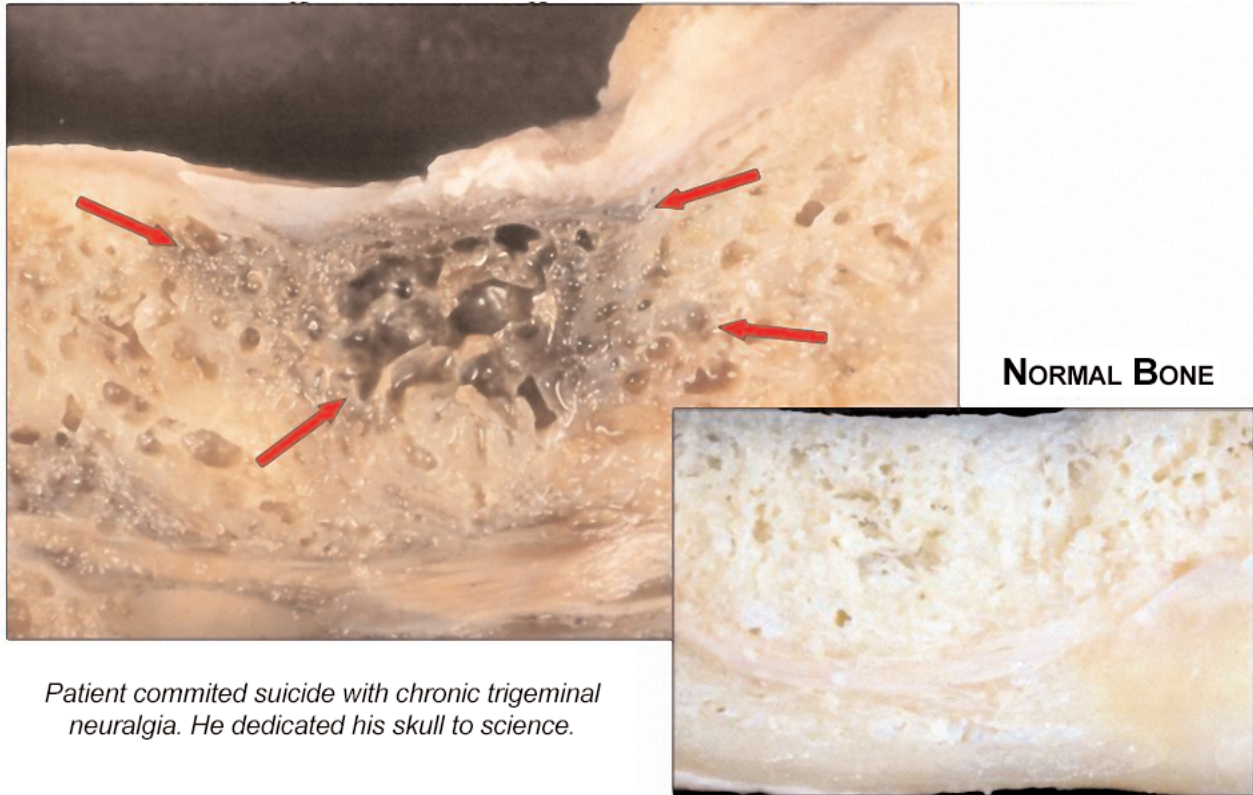


Fig. 1. Shown above is a section from a cadaver mandible that clearly shows the nature and extent of a cavitation infection. Noting how far reaching is the discoloration in the bone, one can easily surmise the difficulties involved in completely removing such an infection

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Jawbone Cavitations: Infarction, Infection & Systemic Disease

Susan Stockton

In the mid-90s I made the eye-opening discovery that chronic health problems which had eluded resolution for many years had their origins in, of all places, my jawbone. I would never have deduced this had it not been for a fortuitous finding: the complete disappearance of bladder problems of a year's duration within days of having an abscessed tooth removed. When I reported the "coincidence" to my dentist, he was surprised and incredulous. He shouldn't have been for he considered himself a "holistic" practitioner. Later however, and despite his lack of understanding of focal illness, that same dentist unknowingly helped me learn more about it by creating the conditions that triggered the full expression of a long silent jawbone disease -- osteomyelitis (a.k.a cavitations, and a dozen or so other names). Although infrequently diagnosed, this disease is in fact very common and is perhaps the most common source of focal conditions in the body. A "focus" is a walled-off area of concentrated toxins and necrotic (dead) and/or infected tissue.

A jawbone cavitation is a recess or pocket in the bone. A cavitation is not readily visible to the eye, and although it can be the hidden cause of facial pain syndromes such Neuralgia Inducing Cavitational names, NICO, it generally causes no local discomfort. There are many possible causes, chief among which is trauma to the jaw, especially the trauma of tooth extraction.

I first encountered the word, "cavitation" about 1995 in the writings of Dr. Hulda Clark. She described it in her books as "a bone infection resulting from an incompletely extracted tooth" -- i.e., an extraction where bits of infected bone were left behind. That description didn't resonate with me then despite the fact that it was exactly what had been silently going on in my jawbone for many years. I guess I thought if I had an infection in my jaw, I'd know it: surely there would be pain, inflammation, tenderness -- and my dentist would find the problem in the course of my routine check-ups. Well . . . wrong !!

Osteomyelitis of the jawbone is not characterized by the usual signs of infection and is most often a silent condition. And it's one that dentists are not trained in school to recognize. In fact, they're not even taught that the condition exists. This is a somewhat perturbing state of affairs, for the jawbone cavitation is not a new disease. It was described in 1848 by Thomas Bond in the first oral pathology book. He wrote about a jawbone necrosis that existed independently of abscessed teeth and gums. In 1915, Dr. G.V. Black, the father of modern dentistry described the condition as "chronic osteitis."

Jawbone cavitations are exquisitely described in an eye-opening book entitled, "Death and Dentistry" written in 1940 by Martin H. Fischer, medical doctor and professor of physiology at the University of Cincinnati. Citing the research of Frank Billings and E.C. Rosenow (early 1900s), Dr. Fischer speaks of "infarctions induced of micro-organismal emboli" that have broken into the general circulation from a peripheral focal

point in the jaw or tonsils. This "metastasis" of microorganisms is the cause of a surprising number of conditions. According to Fischer (p.8,9): "Embolitic infection that has struck the heart valves will be endocarditis; the heart muscle, myocarditis; the pericardium, pericarditis; if all are struck, it is pancarditis. Involving the skeletal muscles, the same pathological background will give rise to myositis; when the tendinous junctions are struck, fibrositis; and when the synovial bursae are affected, bursitis or tenosynovitis. The process in the joints is arthritis; and in the nerves and nerve ganglia, neuritis. In the brain, this is cerebritis, and in its coverings, meningitis." An impressive catalogue of debility . . .

Fischer goes on to explain the role of metastatic infection in gastric and duodenal ulcers, cholecystitis, cystitis, pneumonia, bronchitis, rheumatism, asthma, pleuritis, nephritis, thyroid disease, herpes, iritis, poliomyelitis, multiple sclerosis, certain skin disorders, diabetes, migraines, hypertension and more. He gives case histories and much clinical and laboratory evidence, including impressive photographs of cross-sections of infected teeth and photomicrographic slides.

While a jawbone cavitation might well be, and most often is, infected; in its early stages it can be aseptic. As long as it remains so, no systemic harm is done by it. Even after anerobic infection has developed in a cavitation site the problem remains localized and most likely silent until the bacterial toxins gain systemic access. Symptoms develop when the body burden of toxins increases to the point that nutritional reserves are depleted and the system is no longer able to confine the toxins to their point of origin. At such a point of depletion toxins then begin to travel via blood and lymph channels to other areas of the body.

Toxins create an extremely acidic environment. As long as the body's alkaline reserves (primarily calcium and sodium) remain intact, pH is kept within acceptable limits, homeostasis remains intact and the body functions normally. Once alkaline reserves are depleted however the balance is disrupted. It is not only acid-forming foods like the grains and meat so prevalent in the standard American diet that deplete the alkaline reserves but also the bacterial toxins generated at the site of jawbone cavitations. These toxins destroy critical enzyme systems in the body, including enzymes essential for energy production. Such inactivated enzymes are no longer able to fulfill their role as mineral chaperones. The net result is that even though present in the system, key minerals become biologically inactive because the enzymes needed to activate them have been destroyed by the potent bacterial toxins emanating from cavitation sites.

It is important to understand that such a "mineral deficiency" is unrelated to mineral intake and can exist in spite of ample high quality intake, though insufficient intake certainly compounds the problem. The toxins responsible for mineral deactivation and breakdown of homeostasis are carried throughout the system via blood and lymph vessels and tend to settle in areas of inherent or acquired weakness. This means, for instance, that my jawbone cavitation(s) can result in a symptom picture entirely different from yours.

The over-acid conditions that result once alkaline reserves are depleted have many deleterious systemic effects. When the pH of the blood becomes overly acidic, its viscosity increases -- that is to say it becomes thicker. Consequently, it does not flow as smoothly through the vessels as it once did and clotting anomalies result. A tendency to excessive clotting is very common in chronic cavitation patients. It is also known that excessive homocysteine levels are a factor that predispose one to such clotting anomalies. High levels of

this amino acid are positively correlated with increased risk of coronary infarction (heart attack) and stroke; a fact that is also well known today. Nowhere nearly so well known are the relationships among these facts and the jawbone "infarctions" spoken of by Dr. Fischer in the 1940s.

Although the word "infarct" has come to be associated with heart attack, the condition is by no means confined to the heart. Webster defines an "infarct" as: "An area of necrosis in a tissue or organ resulting from obstruction of the local circulation by a thrombus or embolus." Jawbone necrosis does indeed result from impeded circulation, commonly stemming from trauma to the jawbone. Such trauma is largely iatrogenic, the result of standard dental treatment. Any large fillings especially where toxic metals were used, i.e. crowns and bridges (which of course include the once healthy teeth used as abutments for the bridge) root canals, periodontal scaling and tooth extractions, etc. seriously reduce the blood supply to the consequently injured jaw. When blood supply is compromised nutrients can't get in and toxins can't get out.

Once toxins gain systemic access, alkaline reserves almost always depleted, the blood becomes hyperviscous, homocysteine levels go unchecked and infarction can occur. Such infarction tends to occur initially in the small vessels associated with traumatized bone tissue in the jaw. These infarctions of the microcirculation are, it would appear, a major factor in the development and spread of jawbone cavitations. Fischer understood this years ago when he wrote of, ". . . infarctions induced of microorganismal emboli."

Our current understanding of the role played by elevated homocysteine levels in coronary infarction helps us to better understand the mechanism by which cavitations occur. Engineer and cavitation patient, Bob Jones added a critical piece to the puzzle in 1998 when he observed in the majority of cavitation patients the mutation of genes C-677 and L-677, genes that control production of methylene tetrahydro folate reductase, an enzyme that regulates homocysteine levels. It would appear that while unstable homocysteine levels can ultimately lead to heart attack or stroke, they first effect a traumatized jaw creating infarctions there in the form of cavitations. Jones estimates that some 65 to 85% of the population is affected by this genetic mutation and are therefore predisposed to not only coronary infarction but to jawbone cavitations as well. Given such a genetic backdrop, all we need to add to the equation is dental trauma and voila, we have osteonecrosis/osteomyelitis of the jawbone.

The dental trauma most often associated with a cavitation is the standard tooth extraction, particularly if it involves a third molar (wisdom tooth) site. It is not standard procedure for the surgeon removing these or any other teeth to excavate the periodontal ligament that attached the tooth to the bone. With the tooth removed this ligament serves no purpose and if any part of it is permitted to remain in the socket it will serve as a barrier to healing by impeding all-important blood flow. While the extraction site will almost invariably "heal shut," any such "healing" is deceptively incomplete for below the seemingly "healed-over" surface, a pocket or hole has formed, which pocket is almost inevitably a breeding ground for anaerobic microorganisms whose toxic wastes and by-products are among the most toxic in all of toxicology. It is these microorganisms and their toxins that militate to form the infarction-inducing emboli Fischer so lucidly described many, many years ago.

Whether a cavitation forms following the standard extraction of a tooth depends largely upon how much of the periodontal ligament happened to have been removed along with the tooth and the type of microorganisms which are present at the site. More damaging than the microorganisms themselves are the extremely potent toxins they produce. Once these bacterial toxins gain systemic access, they can do a great deal of harm through inhibition of enzymes and mineral availability, as described above. The necrosis they produce is actually a gangrenous condition. Detoxification is a significant challenge at this point and is an absolute impossibility in the face of the continual toxic exudes from any focally infected teeth and/or jawbone.

The treatment of choice for jawbone cavitations is surgical removal of all necrotic and infected bone for in the presence of such bone the conditions that created the infection remain and blood supply continues to be impaired. When done in conjunction with a new extraction such surgical procedure is straightforward in most cases but where old, "healed over" and infected extraction sites are concerned the task is far, far more difficult and is complicated by the fact that up until very recently there has been no way to clearly image and gain information about a potential site's actual location, size and other distinguishing features short of surgically opening it to "look around," and even then the site cannot be viewed from all angles. To the trained eye the panoramic X-ray can reveal indications of the presence of a cavitation, but not always. Even when it does, details are not often clearly discernible and the surgeon is left operating "in the dark" to one degree or another as the 2-dimensional X-ray image cannot adequately reveal the structure of the 3-dimensional jawbone. In some severe instances however, cavitations can be depicted on X-ray; however, as much as 50% of the bone must be deleteriously effected before a cavitation's presence is unequivocally apparent.

While cavitations can be imaged by CAT scans and through MRI, these methods are neither practical nor cost-effective for use in the dental profession as they expose the patient to the adverse effects of radiation and require the interpretive services of a radiologist. The aware dentist has long been in need of a reliable instrument for clearly and safely imaging jawbone cavitations; ideally an instrument that could be used "in-house." Due to the unflagging efforts of Bob Jones, the engineer/patient responsible for uncovering the genetic mutation associated with elevated homocysteine levels, such an instrument is now available. Jones' story is interesting and as told in my book, *Beyond Amalgam: The Health Hazard Posed by Jawbone Cavitations*," even more dramatic than my own, so it's worth telling here.

A decade ago, Bob Jones was a picture of perfect health; or so it seemed. He was employed full-time as a commercial airline pilot and worked part-time as a ski instructor. This avid outdoorsman was slim, trim and fit. All that changed in 1987 when he was stricken with chronic debilitating fatigue, muscle atrophy and a neurological condition that baffled specialists. By 1992, he had become completely disabled, was wheelchair bound, had lost use of his arms and gained an excessive amount of weight. While the medicos couldn't come to agreement as to the exact nature of the problem, finally settling upon a speculative diagnosis of ALS, they were in agreement about one thing: Jones' condition was terminal. He was working through what he'd been told would be the last six months of his life when he stumbled upon an understanding of the source of his problem, and a way to turn it around. His search for solutions led him to the realization that potent toxins, by-products of standard dental treatments, were essentially poisoning his

system. Bob's symptoms subsided, and his condition dramatically improved once his diseased jawbone marrow and "silver," i.e. mercury, fillings were removed. Today he is completely mobile, moderately active and much of the excess weight has been lost. However, Bob is quick to point out that his recovery has not been 100%. While cavitation patients can indeed expect real and often substantial improvements, the fact remains that owing to the duration and the severity of their chronic conditions a complete cure is an uncertain outcome..

Even before his recovery, Bob set out to develop an instrument designed to detect jawbone cavitations. Since these lesions routinely elude detection through standard diagnostic procedures, the need for an improved imaging device was apparent. As a design engineer with a background in sonar technology, Bob was convinced from the onset that such an imaging device could be developed using sonography. Six months after commencing the arduous task of "cleaning out" his jawbone, Bob had developed the first working prototype of the CAVITAT™. There would be many design revisions and obstacles in his paths in the years to follow, but he worked diligently to make his vision of a perfected CAVITAT™ the reality it has now become.

The proprietary CAVITAT™ analog-to-digital circuitry has been awarded a patent substantiating 19 claims, with an additional 22 claims pending on the flexible transceiver circuits and their advanced cross-channel noise suppression technique. The device is unique in the sonography market in that it is engineered to image only bone, not soft tissue. All other ultrasound devices do just the opposite: they show tissue but not bone, and the image they display is 2-dimensional while the CAVITAT™ displays a relevantly color coded 3-dimensional image. Green, yellow and red colors reflect the degree of bone loss and necrosis while the 3-D images can be rotated in the purpose-written WinCav software for a view from any angle. One image is generated for each of the 32 tooth sites and all can be displayed on the computer screen simultaneously. This allows the operator to see an overall picture and how one affected site can influence an adjacent one. Each of the 32 images is generated from the very rapidly multiplexed outputs of the 64 ultrasound sensors that comprise the ultrasound receiver array, handheld within the mouth and against a tooth in question by the operator/dentist. This high speed multiplexing results in images that change in real-time with the operator's movements of the receiver probe thereby making possible the fine adjustments needed to acquire the most accurate image possible.

The new Generation 4 CAVITAT™ differs from its prototype precursor in many important respects. The resolution has been increased 800%, making for much clearer images and enabling detection of smaller cavitations. The Generation 4 is capable of detecting jawbone defects down to .030 inches in diameter.

By the end of 1999 Jones had introduced a limited number of the Generation 3 CAVITAT™ to a select number of biological dental practices. These were prototype models used for field evaluation and feedback from the dentists using the instrument provided the data necessary to make developmental improvements. The software was totally rewritten and the result is a user-friendly, state-of-the-art precision instrument. It is this version of the CAVITAT™ that is now being made available to doctors and dentists to assist them in diagnosis of jawbone cavitations and other bony defects of the jaw. The instrument can be used in real-time conjunction with a cavitation surgery, thereby enabling the surgeon to monitor progress being made in the removal of diseased and/or necrotic tissue.

The significance of this technological breakthrough cannot be overemphasized as the success of cavitation

surgery is dependent upon many variables; a major one being the extent to which necrotic tissue is removed. Before the advent of the CAVITAT™ dentists were operating very much in the dark, unable to see the full extent of the necrosis and therefore unable to remove all necrotic tissues. The result for many patients was poor bone healing, unchecked spreading of osteomyelitic lesions and consequent need for repeat surgeries. While excision of all diseased bone will not necessarily assure full recovery, it certainly does improve the odds. Most patients have had jawbone cavitations for a number of years before they are discovered. Consequently by the time treatment is initiated, a great deal of damage has been done. Dr. Fischer had stated in, "Death and Dentistry," "It is only in the earliest stages of oral disease that arrest of progressive infection seems possible." With the development of the CAVITAT™, early detection is finally possible. It may be our only hope of reining in on this silent, insidious condition that appears to have taken on epidemic proportions.

While thorough excision of osteomyelitic/osteonecrotic lesions is necessary in the treatment of cavitations, surgery alone is often insufficient for the chronic cavitation patient due to the effects of the chronic toxic assault these people have endured. An extensive detoxification protocol tailored to the individual's eliminative capabilities, overall condition and basic constitutional fortitude is required. Effective nutritional support is essential to rebuild bone, improve circulation, combat infection and chelate heavy metals.

While surgical treatment of cavitations falls within the domain of the dental profession, the metastatic infection seeded by these lesions has systemic consequences that should be of interest to all physicians. It is therefore imperative that every patient's history include questions about dental treatment. Remember that any trauma to the jaw can be the beginning of cavitations.

The high-speed drill routinely used by dentists cracks enamel thus allowing bacterial toxins to penetrate the dentine. There is evidence that such drills cause actual pulp damage. Drilling done then in preparation of a tooth for routine fillings, crowns and bridges can be damaging to the jawbone. Root canals will unquestionably cause cavitations sooner or later, as will routine extractions (where the socket is not properly cleaned out i.e. all necrotic/infected bone is removed). The eclectic physician will not only want to question his patients about these procedures, s/he will also want to be in a position to diagnose jawbone cavitations or to refer patients to a dentist who is able to make such a diagnosis. Once the diagnosis is made it is desirable that the dentist and doctor work together in initiating a treatment plan and following up with the patient.

In working with the chronic cavitation patient it is imperative that the entire jawbone be considered and examined, not just the site(s) of extractions. A mistake that is frequently made is to clean out (debride) new extraction sites while ignoring old ones. If all necrosis is not removed, it will spread and it will ultimately re-infect a new extraction site, even one that was properly debrided. Taking things a step further, it is important to be aware that the spreading of jawbone cavitations is not confined to edentulous areas. When the bone beneath an apparently "vital" tooth becomes affected/infected blood supply to that tooth is greatly reduced and it begins to die. Neither oral exam, nor X-ray evaluation is likely to reveal a problem with such a tooth. ElectroDermal Screening and muscle-testing might also miss the problem. The patient however frequently has a sense of something being "not quite right" with the tooth. If he or she insists

upon its extraction (usually against the advice of the dentist) and manages to talk his/her dentist into removing it, that dentist is counseled to carefully examine the extracted tooth. Chances are very good that upon drilling into the pulp chamber s/he will find evidence of the death process. This evidence will likely take the form of dryness as the blood supply has been severely hampered. I say all of this from personal experience as my last 3 extractions were done at my insistence and against the initial protestations of my dentist, who fortunately was open-minded and curious enough to drill open the pulp chambers of the extracted teeth.

Dentists are taught to save the tooth at all costs. Frequently however, the price paid is the systemic health of the patient. Dead and dying teeth can not remain in the jaw, even if they are not causing apparent distress to the patient. If CAVITAT™ scan of the jawbone shows pronounced necrosis under a "vital" tooth, please entertain the possibility that the tooth only appears to be vital and is in fact dying. Healthy teeth do not grow within necrotic bone.

For the chronic cavitation patient, extraction can be both the beginning and end of his or her health problems. An improperly done extraction, usually that of a wisdom tooth, is frequently the beginning of problems that might go undetected for decades and finally resolved only by the proper extraction of most or all of the remaining teeth along with removal of necrotic bone from edentulous areas and powerful yet gentle systemic detoxification. Prevention and early detection are the keys to avoiding this outcome. The CAVITAT™ imaging abilities provide us an accurate metric essential to early and effective intervention. Even so, the first step in any resolution cannot be other than an awareness of the problem's existence.

You have taken that step and are urged to take the next one.

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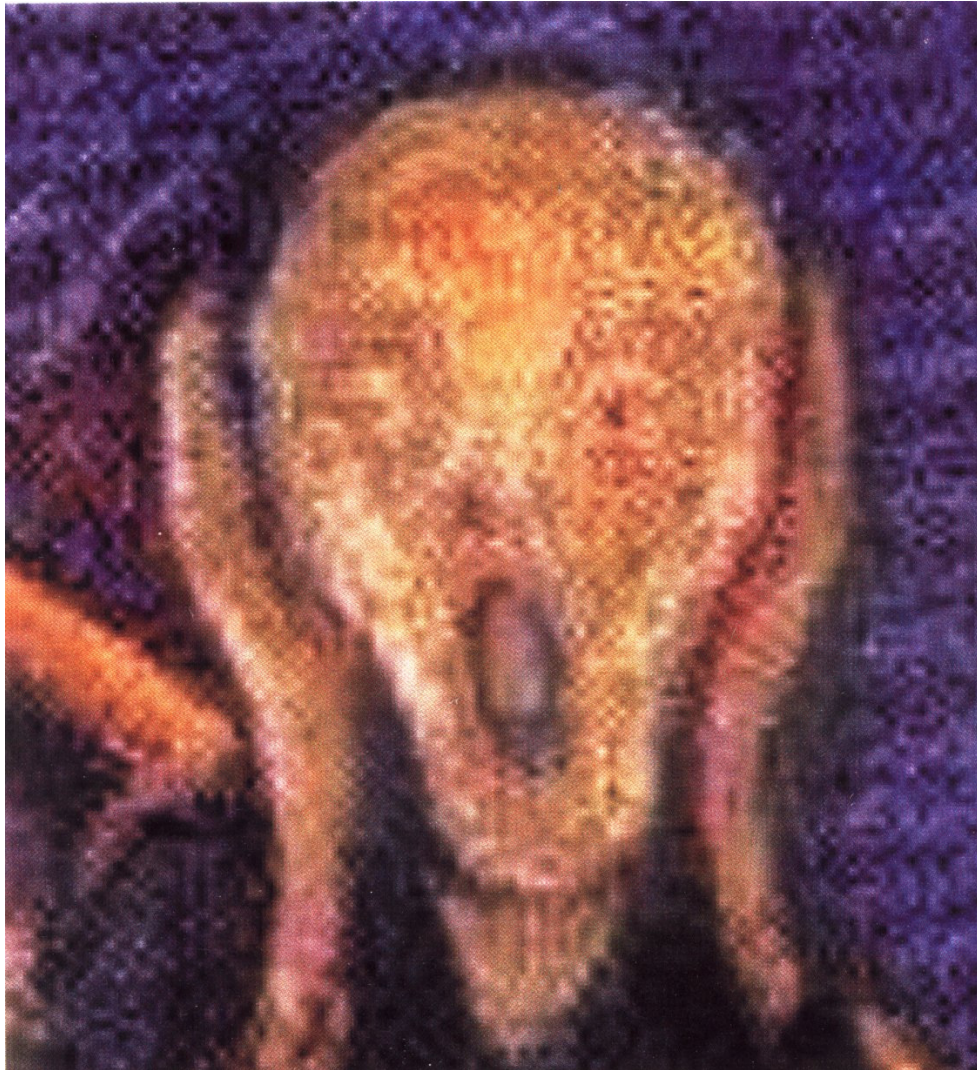
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Susan Stockton, MA, is a recognized writer and researcher in the field of natural health and medicine. She is author of a number of books and articles, including, "Beyond Amalgam" and the recently released, "The Terrain is Everything" (available through Power of One Publishing, 303-755-2605). Susan recently relocated from Winter Haven, Florida to Aurora, Colorado to work for CAVITAT Medical Technologies, Inc.

TMJ & Facial Pain Center

Wesley E. Shankland, II, D.D.S., Ph.D., Inc.

Jaw Bone Cavitations



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Dr. Shankland's Biography

Dr. Wesley Shankland is a graduate of The Ohio State University, earning a Bachelor of Science degree in both biochemistry and zoology. In 1978, he graduated from the College of Dentistry, The Ohio State University. Dr. Shankland returned to graduate school in 1989 and earned a Master of Science (1993) degree and a Doctor of Philosophy Degree, both in anatomy (1997).

The director of the TMJ & Facial Pain Center in Columbus, Ohio since 1983, Dr. Shankland's practice is limited to the diagnosis and treatment of craniofacial pain and temporomandibular disorders. The author of over 100 scientific papers and chapters in three textbooks, Dr. Shankland has also written three best selling books (*TMJ: Its Many Faces*, which was featured in *US News & World Report* Magazine and *Face The Pain*). His latest book, *Bioterrorism: You Can Survive*, was released in June 2002.

Dr. Shankland has lectured throughout North America, Mexico, the Caribbean, England, Germany, Australia, South Africa, Hong Kong and Norway on such subjects as anatomy, the differential diagnosis of headaches, the diagnosis of craniofacial pain disorders, the diagnosis and treatment of osteocavitations, and temporomandibular disorders.

The Past President of the American Academy of Craniofacial Pain (formerly, the American Academy of Head, Neck & Facial Pain), Dr. Shankland is also on the editorial board of four journals and is the Associate Editor of the *Journal of Craniomandibular Practice*.

Dr. Shankland has served on the faculty of the College of Dentistry and College of Medicine, both at The Ohio State University.

For hobbies, Dr. Shankland is a martial artist, a writer of fiction and a model railroader. He and his wife of 33 years, Cathy, are both from Lima, Ohio. They have three adult children and attend The Church Next Door (formerly, Lincoln Baptist Church).

Although Dr. Shankland's chief desire is to help both with education and treatment, he can't provide you with a phone or Internet consultation without first performing a physical examination. Without such an exam, it wouldn't be ethical or proper for him to give you a diagnosis. This brochure is intended for informational purposes only and is not meant to be, and should not be relied upon, for recommendations regarding diagnosis and treatment of any individual case. It is not meant to be a substitute for proper medical care by your doctor. You need to consult your doctor for diagnosis and treatment.

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Jaw Bone Cavitations

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I. Cavitation Overview

As recently as 1979, a newly described pain syndrome was reported by two separate oral surgeons, Ratner and Roberts.^{i,ii} This disorder was real new, for as early as 1915, Dr. G.V. Black, the father of modern dentistry, described these lesions in his pathology textbook,ⁱⁱⁱ calling the condition *chronic osteitis*.

Dr. Black felt this bony inflammatory process had the unique ability to produce extensive bone destruction without producing redness or swelling of the overlying tissues, without causing an increase in the patient's body temperature, and often producing no pain. He used the word *cavity* to describe these lesions within the bone marrow cavities. He recommended surgical curettage as the only effective treatment. Later this condition became known as *osteonecrosis* of the bone and was often a direct result of osteomyelitis (chronic osteitis), or bone inflammation of the bone marrow.

In 1992, a paper written by oral pathologist Dr. Jerry Bouquot^{iv} was instrumental in changing the name to NICO (Neuralgia-Inducing Cavitational Osteonecrosis). In other words, pain due to dead bone which *mimics* trigeminal neuralgia. However, not all cavitations produce pain. Many are quiescent, lying dormant, like smoldering embers, often for decades.

The concept of jawbone cavitations is not without controversy. For some strange reason, many in the dental profession refuse to believe that these bony lesions even exist. Yet, in the specialties of orthopedic and neurosurgery, internal medicine, and podiatry, such lesions are a major problem to doctor and patient alike. At least 20% of all hip replacements in the United States are due to osteonecrosis, so wouldn't it be reasonable that the same disease could also affect the jaws?

Why many in the dental profession deny the existence of areas of dead bone in the jaws remains a mystery. Perhaps your eyes only see what your brain knows (or wants to know). After all, and like the hip, the mandible (lower jaw) and maxilla (upper jaw), are medullary bones, meaning they contain bone marrow and produce blood cells.

Osteomyelitis of the jaws was a very serious and life-threatening disease until the advent of antibiotics. Since then, such bony lesions have been treated with limited success with antibiotics. However, unless the dead and dying bone tissues are removed surgically, regardless of how much antibiotic is given, the problem remains.

We now know that many of those who develop jawbone cavitations may actually have a genetic disorder, especially those who require multiple surgeries.

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II. Causes of Cavitations

The cause of cavitations is similar to the development of dead bony areas in the head of the femur (the hip): blockages in the tiny blood vessels either by swelling of those vessels or by the formation of blood clots due to a malfunction of one or several of the many steps in the normal blood clotting reaction. Both of these conditions prevent blood flow, thus robbing oxygen from areas of bone, causing bone death. This process is similar to a stroke, blocking tiny blood vessels in the brain, or a heart attack, robbing the heart tissues of oxygen, causing death around the blockage (termed an *infarction*). In the jaws, we think this process of infarction plus the effects of chronic bone inflammation from a dead or abscessed tooth, poor healing after surgery, or trauma to the jaws are some of the causes of bone death and thus, cavitation formation.

Table 1 lists some of the suggested causes of jaw bone ischemia and infarction taken ***directly from the scientific, medical literature***, all of which may eventually produce cavitations and if painful, NICO lesions.

Table 1: Factors suspected of causing cavitations in jaw bones.

| Local Factors | Systemic Factors |
|----------------------------------|------------------------------|
| Trauma | Steroid therapy |
| Chronic tooth infection | Hypofibrinolysis |
| Inadequate root canal treatment | Birth control pills |
| Bony infections/inflammation | Sickle cell diseases |
| Tooth extractions | Estrogen therapy |
| Swelling after oral surgery | Heavy smoking |
| Implants | Thrombophilia |
| Cortisone Injections | Systemic cortisone |
| Epinephrine in local anesthetics | Saline abortion |
| Restriction of blood flow | Systemic lupus erythematosus |

To date, we've isolated *at least 74 causes or risk factors* which cause cavitations to develop. These factors apply to all bones, including the jawbones. Trauma and infection are the primary triggering events for cavitation development, and no other bones in the body come close to the amount of trauma and infection experienced by the jaws from dental and periodontal infections, tooth extractions, root canals, effects of local anesthetics, and from oral surgical procedures.

Although we've isolated many probable causes of cavitations, the most common seen at the TMJ & Facial Pain Center are:

- \$ **Trauma**, which may be from mild or severe direct blows to the jaws.
- \$ **Persistent tooth infections.** These may be as simple as a chronically sensitive tooth after a dental restoration or a severe tooth infection, which requires root canal therapy and oftentimes, subsequent extraction of the tooth.
- \$ **Surgical trauma.** Next to a history of chronic tooth pain, we find bone trauma from the simple removal of a tooth, periodontal or endodontic surgery, or even major orthognathic surgery are very common with cavitation patients.
- \$ **Thromophilia and hypofibrinolysis.** Thrombophilia (increased tendency to develop blood clots [termed *thrombi*]) and hypofibrinolysis (reduced ability to destroy blood clots) appear to contribute greatly to cavitation formation and osteonecrosis (bone death) throughout the body. These genetic blood coagulation disorders probably predispose certain people to form blood clots, thereby preventing blood flow, which starves the bone of oxygen.

We've found that approximately 75% of our surgical failures are due to one or both of these genetic disorders. Such high numbers partially explain why osteonecrosis is such a difficult problem to treat, regardless of where it may occur.

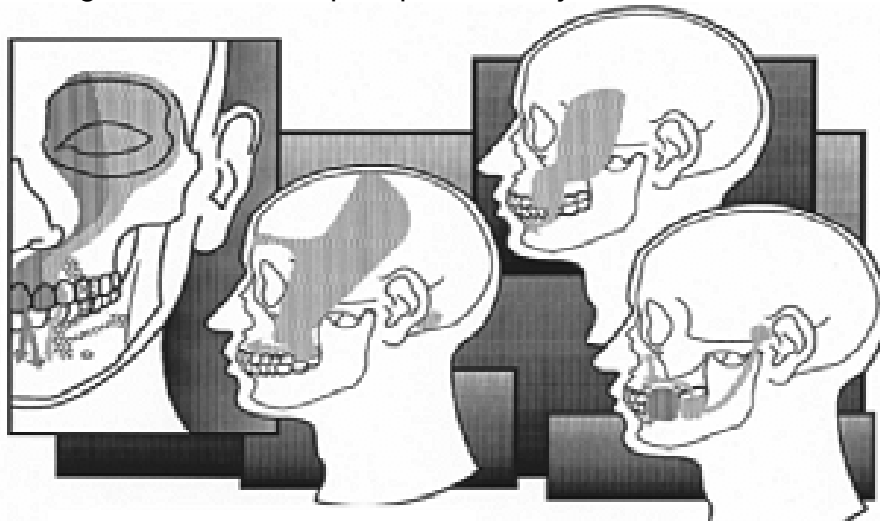
- \$ **Antiphospholipid syndrome.** Adding further confusion to the development and diagnosis of cavitations is the disease termed *antiphospholipid syndrome* (APS). With this immune system disease, blood clots develop within small arteries and veins because the body actually makes antibodies *against* itself. Normally, our bodies manufacture antibodies to destroy bacteria which invade us from the outside. However, with APS, antibodies are manufactured and act against the body, often producing blood clots and cavitations if the clots occur within a bone.

III. Symptoms of Cavitations

Cavitation lesions may produce *no symptoms at all*, especially if there is no redness over the area or signs of drainage. However, these lesions can also produce intense, trigeminal neuralgia-like symptoms,^{v,vi} which cause suffering to such an extent it is a wonder that patients can stand the pain and suffering.

There are established, characteristic referred pain patterns (Figure 1), which we find very consistent in most symptomatic cavitation cases. In those patients with pain, usually they have an underlying, constant dull aching. Along with this gnawing, deep pain, *often there is a sharp, shooting pain which*, understandably convinces doctors that the diagnosis is trigeminal neuralgia.

Figure 1: Referred pain patterns of jawbone cavitations.



A very common symptom we find is a *sour, persistent drainage* from the cavitation directly into the mouth. This foul taste makes many patients and doctors alike consider a diagnosis of sinusitis. Unfortunately, all the sinus surgery in the world will not correct the problem if the sour fluid is draining from a area of dead bone: a jawbone cavitation.

To confuse matters more, many patients report systemic symptoms like arm or leg pain and generalized fatigue. We've seen several patients with chronic fatigue syndrome which, once the cavitation lesion or lesions are removed, the chronic fatigue and systemic symptoms are improved or even stopped.

The typical cavitation patient B and this is horrible! B has had pain for approximately 6 years! Can you imagine the amount of money, suffering, lost work, damaged family relationships these people have experienced?

Are both the upper (maxilla) and lower (mandible) jaw affected with cavitations? Dr. Bouquot conducted a study where he analyzed biopsy material from 2,301 cavitation lesions. The maxilla was slightly more involved (51.5%) than the mandible (48.5%). In a study of 500 consecutive surgical patients^{vii} in our office, we found similar results: 54.2% in the maxilla and 45.8% of cavitations in the mandible.

Needless to say, there is a problem, and these studies are based upon microscopic examination of biopsy materials. Regardless of what one may believe, cavitations are real and devastating.

Some of the more common symptoms of cavitation which we see are:

- g **Deep bone pain**, which is constant, but varies in intensity.
- g **A sour, bitter taste**, which often causes gagging and bad breath.
- g **Sharp, shooting pain** from the jaws which eludes doctors' diagnostic attempts.
- g **Chronic maxillary sinusitis**, congestion and pain.
- g **A history of a large filling** being placed (or replaced), followed by pain, root canal therapy, and ultimately, removal of the tooth.
- " **Multiple root canals**.
- g **Endodontic surgery** (apicoectomy).
- g **Tooth extraction**, including wisdom teeth, several years earlier.
- g **Failed attempts to treat trigeminal neuralgia**.

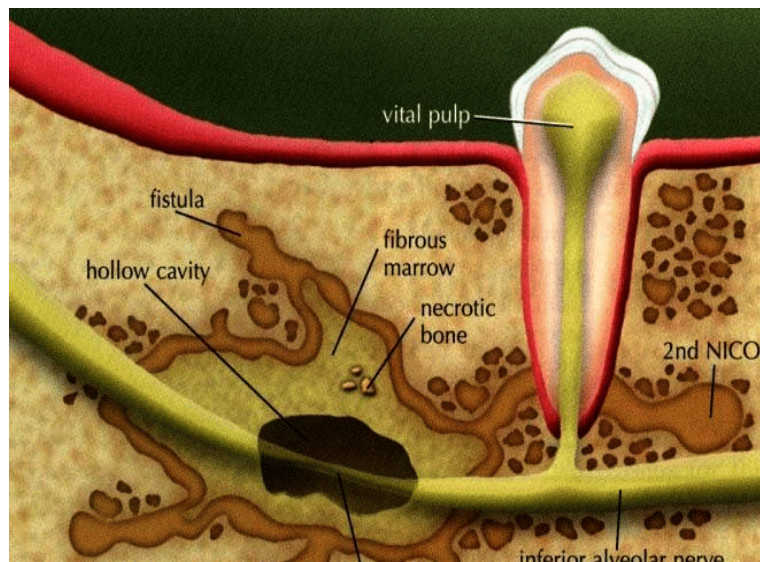
The most common scenario we see usually starts with a simple dental restoration. The family dentist replaces an old restoration (filling) and the tooth becomes sensitive, especially to cold temperatures. The doctor may replace the filling again or several more times, but the sensitivity never improves. Then, in most cases, the tooth is treated with a root canal. But guess what? The pain continues.

Another doctor is consulted, only to have the tooth re-treated with root canal therapy, but the pain persists, but is generally worse than in the beginning. Finally, out of sheer desperation (by both patient and doctor), the tooth is extracted, only to have the pain continue and intensify even more.

In this scenario, the finest dentistry was performed, but something went wrong. It wasn't negligence by the dentist, but damage to the tiny vessels in the jaw around and beneath the injured tooth. Assaulted by constant inflammation and swelling, an infarction occurs in one or more of the tiny vessels, producing ischemia and ultimately, bone death and cavitation formation (Figure 2).

Remember, cavitations can be completely painless, and this is not unique to the jawbones. In other bones, such as the femur, often there is no pain even when the bone destruction is extreme.

Figure 2: Diagram of cavitation lesions in the mandible.



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IV. Diagnosis of Cavitation Lesions

The diagnosis of cavitation lesions is complicated by the fact that x-ray examination of the jawbones often appears normal . . . to the untrained eye. Considerable diagnostic experience is required because changes in the bone are subtle and can mimic a number of other entities, including variations of normal anatomy.

Why is this so? Osteonecrosis is a disease of the marrow spaces of bone and 30% to 50% of such bone must be destroyed *before* changes can be seen on x-rays. So, if your dentist or oral surgeon takes an x-ray and pronounces the film normal in spite of your symptoms, do not necessarily believe it. X-rays may be interpreted as normal unless (1) there is a significant amount of bony destruction or (2) the doctor is experienced in reading x-rays specifically for cavitations.

Although MRI (magnetic resonance imaging) is the imaging technique of choice for long bones, flat bones of the face are not imaged well with MRI scans. CT scans are also ineffective in locating most cavitations in the jawbones.

Bone scans using a radioactive isotope are somewhat helpful in locating cavitations, but very difficult to interpret. Also, radiologists, not expecting these lesions in jawbones, often note the lesions in their radiology reports, but interpret the results as normal.

The best, most effective method to locate cavitations is the Cavitat bone scanning device.^{viii} This computer-based ultrasound imaging system was designed to aid the medical community with a detailed profile of the interior of bones. The Cavitat computer generates digitized two and three dimensional images of the interior of the jawbones from sound waves passed through the bones.

Because liquid is a near perfect conductor of sound waves, when these waves enter into voids or porosities in bone (areas that have compromised bone flow; i.e., cavitations), the sound waves slow down considerably, which produces images of the interior of the bony area being scanned. We have found the Cavitat results to be very accurate, especially when compared with patients' panoramic X-rays. Our diagnostic results have improved dramatically. Most importantly, our surgical successes have soared since we began using this technology.

For patients experiencing pain, diagnosis is further improved through anesthetic confirmation or anesthetic blocking. By giving a local anesthetic injection (similar to having your dentist numb the jaw before he or she performs a dental procedure), cavitations in the jaws can be selectively turned-off, meaning the sense of feeling of pain can be chemically and temporarily eliminated. If the pain goes away after the injection, then we can be reasonably certain that there is a problem in the anesthetized area, generating pain.

V. Treatment of Cavitations

A few different modes of therapy are used today to treat cavitations:

- **Antibiotics.** Prescribing antibiotics may *temporarily* reduce cavitation pain, but due to a compromised blood supply to the region, not much of the antibiotic can actually get into the bony lesion to be effective.
- **Injection of homeopathic remedies.** This has become very popular recently and some doctors inject specific homeopathic solutions directly into cavitations. This will never work permanently because the dead bone in the cavitation has no blood supply, which is required for normal healing and removal of toxins.
- **Anticoagulant therapy.** When a patient has a systemic clotting problem and other risk factors, we often use specific anti-clotting medications (e.g., Coumadin and low weight heparin) to prevent any further clotting within the blood vessels in the jawbones.
- **Hyperbaric therapy.** At times, hyperbaric therapy (placing a patient within a chamber and increasing the atmospheric pressure, driving oxygen into the tissues) may be used for treatment of osteonecrosis.
- **Surgery.** The only effective way to treat moderate to severe (in size) cavitations today is surgical curettage of the lesion itself, removing the dead bone and bone marrow. This creates a good healing environment, especially when used in conjunction with other therapies such as nutritional supplements, boosting the immune system, and anti-clotting procedures. All materials removed during surgery must be biopsied. The only true way to diagnose cavitational lesions is by evaluating this material through microscopic examination. Many bone disorders produce similar x-ray appearances to cavitations, so a biopsy is needed to confirm or deny the actual diagnosis.

What about surgical failures? We're sorry to report that osteonecrosis has a strong tendency to recur and/or develop additional lesions about 30% to 40% of the time. Also, maybe 5% to 10% have no or little reduction of pain and about 15% have moderate reduction of pain. Sadly, nearly 3% have an increase in pain after surgery.

Patients who don't respond to surgery often have multiple problems (for example, a concurrent TMJ problem), may have to see other types of doctors, or, may never totally respond regardless of what treatment modalities are performed. Sometimes we have to see patients multiple times with several weeks between each surgery.

If you're being treated by a doctor for a jawbone cavitation, make sure any material removed during surgery is biopsied, including teeth. This is very important in order to (1) establish the correct diagnosis and (2) to rule-out any other abnormalities which may be in the bone.

What Can You Do?

If you or a loved one suspects a cavitational problem, what can you do? First, allow no one, regardless of his or her number of degrees, to operate without first proving the pain can be stopped with a local anesthetic injection. If the pain can't be erased for a short time with a local anesthetic injection, chances are there's another problem causing the pain.

Second, find a dentist who is both trained and experienced in the diagnosis and treatment of cavitational lesions. Sadly, there are few such doctors worldwide today, but we're making efforts to train more as soon as possible.

Third, request an ultrasound (Cavitat) examination. After using the Cavitat for several years, I can't imagine treating patients without this remarkable and accurate device. There are many fine doctors who treat cavitations who don't use the Cavitat, but for me, I need the information I obtain from the scans. I credit this technology more than any other procedure for the vast improvement in our surgical successes.

When finding a doctor, ask the receptionist the following questions when making your consultation appointment:

1. What type of training does the doctor have in treating cavitations?
2. How long has the doctor been treating cavitations?
3. What's his or her success rate? If you're told more than 90%, find another doctor, or at least get another opinion.
4. Who would the doctor suggest as a second opinion? If a doctor won't provide the name or names of other doctors for a patient to visit for a second opinion, then don't make an appointment. If the diagnosis is correct in one office, it will be correct in other offices, too.
5. How does the doctor diagnose cavitations? Does he or she use a ultrasound (i.e., a Cavitat)? Anesthetic blocking? Panoramic radiographs?

VI. Possible Post-Operative Complications

As with all surgical procedures, there are possible complications and risks. Fortunately, with proper preparation, risks can be minimized. Concerning cavitation surgery, some of the following are possible post-operative complications:

- Pain;
- Bruising;
- Infection;
- Swelling;
- Partial improvement;
- No improvement;
- Worsening of the condition;
- Damage to nerves (usually the inferior alveolar and mental nerves) which may produce loss of feeling sensations temporarily or even permanently through the distribution of the nerve;
- Entrance into the maxillary sinus;
- Injury to the temporomandibular joint;
- The need to consult additional doctors;
- Loss of one or more teeth;
- Mutilation or damage to bony areas that may need to be treated with ridge augmentation prior to any restorative procedures; and
- The need to have the surgical procedure repeated.

Fortunately, rarely do any of these complications occur, but one considering cavitation surgery is entitled to know possible complications so that he or she may make an informed decision concerning surgical intervention.

Due to the anatomical complexities of the mandible and maxilla, these possible post-operative complications must be weighted against the benefits of surgery. If your

doctor doesn't mention or won't talk about such possible complications, find a new doctor.

VII. An Appointment With Dr. Shankland

If you'd like to be evaluated in Columbus by Dr. Shankland and his trained and expert staff, please call our office (614-794-0033) during normal working hours Monday through Wednesday and Thursday until 1:00 P.M. Eastern Time.

If you're traveling more than three hours or coming from a distant state or foreign country, please plan to stay in Columbus for at least three days. Our office has made arrangements with several fine hotels (with various room rates) for our patients. A couple of the hotels even provide shuttle service to and from the airport, to and from our office, and to and from stores and attractions in the Columbus area.

Generally, the first day's appointment lasts approximately two hours and its sole purpose is to determine an accurate diagnosis and plan for any definitive treatment. Please bring the following with you at the first appointment:

- The completed personal history form mailed to you with this booklet;
- Good copies of any x-rays, especially panoramic x-rays
- A list of all your allergies and other medical problems (if any);
- A list of all current medications which you're taking; and
- An expectant attitude that you, too, can be helped!

The second day we operate if necessary. All post-operative items (except prescription medications) will be supplied by our office. Usually the surgical procedures take 2 to 3 hours and are performed under local anesthesia and oral sedation (if necessary).

The third day is used for re-evaluation of your symptoms and post-operative inspection of the surgical site. This appointment takes but a few minutes and you may even plan to leave for home immediately after the third appointment, whether by car or plane.

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"Beloved, I pray that you prosper and be in good health, just as your soul prospers" (3 John 2)

Osteocavitation Lesions

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History and Overview

Cavitations or NICO lesions are hollow places in jaw bones. These hollow areas may never cause pain or a problem. However, cavitations can produce trigeminal pain, headaches, and facial pain. Cavitations are common in all bones that have bone marrow. Many cavitations linger for years without producing facial pain.

Most people know what we mean when we say cavity, but the word *cavitation* is confusing. Both of these words come from the same root word meaning hole. A cavity is a hole in the tooth, whereas a cavitation is a hole in bone. Unlike most tooth cavities, bone cavitations can't be detected by simply looking at the bone, and even using x-rays, many cavitations are missed. The term *cavitation* was coined in 1930 by an orthopedic researcher to describe a disease process in which a lack of blood flow into the area produced a hole in the jawbone and other bones in the body. Dr. G.V. Black, the father of modern dentistry, described this cavitation process as early as 1915 where he described a progressive disease process in the jawbone, which killed bone cells and produced a large cavitation area or areas within the jawbones. He was intrigued by the unique ability of this disease to produce extensive jawbone destruction without causing redness in the gingiva (gums), jaw swelling, or an elevation in the patient's body temperature. Essentially, this disease process, which produces osteonecrosis (dead bone) is actually a progressive impairment which produces small blockages (infarctions) of the tiny blood vessels in the jawbones, thus resulting in osteonecrosis, or areas of dead bone. These dead, cavitational areas, which produce pain, are now called NICO (Neuralgia Inducing Osteonecrosis) lesions (Figure 1). In his book on oral pathology, Dr. Black suggested surgical removal of these dead bone areas.

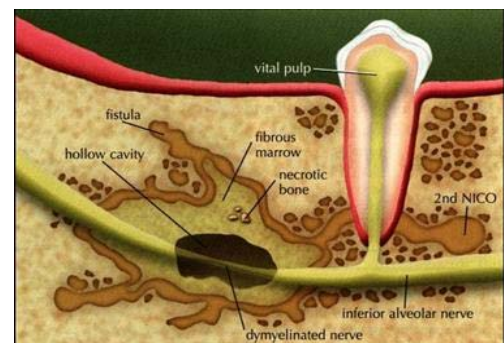


Figure 1: Diagram of cavitation lesions in the mandible.

Symptoms of Cavitations

Cavitations may produce no symptoms at all, especially if we find no redness over the area or signs of drainage. However, these lesions may also produce intense, trigeminal neuralgia-like symptoms, which cause suffering to such an extent that it's a wonder patients can stand the pain and suffering.

There are established, characteristic referred pain patterns (Figure 1), which we find very consistent in most symptomatic cavitation cases. Patients with pain usually have an underlying, constant dull aching. Along with this gnawing, deep pain, often there's a sharp, shooting pain, which, understandably, convinces doctors that the diagnosis is trigeminal neuralgia.

A very common symptom we find is a **sour, persistent drainage** from the cavitation directly into the mouth. This foul taste makes many patients and doctors alike consider a diagnosis of sinusitis. Unfortunately, all the sinus surgery in the world will not correct the problem if the sour fluid is draining from areas of dead bone, namely, a jawbone cavitation.

Some of the more common symptoms of cavitations are:

- **Deep bone pain and pressure**, which may be constant but vary in intensity
- **A sour, bitter taste**, which often causes gagging and bad breath
- **Sharp, shooting pain** from the jaws, which eludes doctor's diagnostic attempts
- **Chronic maxillary sinusitis**, congestion and pain
- **A history of large dental fillings** followed by pain, root canal therapy, and ultimately, removal of the tooth
- **Multiple root canals**
- **Endodontic surgery** (apicoectomy)
- Difficult **tooth extraction**, including wisdom teeth, several years earlier
- Post-operative complications, especially the development of a dry socket
- **Failed attempts to treat trigeminal neuralgia**

To confuse matters more, many patients report systemic symptoms like arm or leg pain and generalized fatigue. We've seen these systemic symptoms improve, or completely resolve, once the cavitation (or cavitations) is removed. The same has been seen in some chronic fatigue cases.

The most common scenario we see usually starts with a simple dental restoration. The family dentist replaces an old restoration (filling) and the tooth becomes sensitive, especially to cold temperatures. The doctor may replace the filling again or several more times, but the sensitivity never decreases. Then, in most cases, the tooth is treated with root canal therapy. But guess what? The pain continues. Another doctor is consulted, only to have the tooth re-treated with root canal therapy, but the pain persists . . . generally worse than in the beginning. Finally, out of sheer desperation (of both patient and doctor), the tooth is extracted, only to have the pain continue and intensify.

In this scenario, the finest dentistry was performed, but something went wrong. It wasn't neglect by the dentist but damage to the tiny vessels in the jaw around and beneath the injured tooth. Due to the constant inflammation and swelling, an infarction occurs in one or more of the tiny vessels, producing ischemia and, ultimately, bone death and cavitation formation (Figure 2).

Remember, cavitations may be completely painless. This is not unique to the jawbones. In other bones, such as the femur, often there is no pain even when the bone destruction is extreme.

Location of Cavitations

| Alveolar location | Maxilla | Mandible | Total |
|----------------------|---------|----------|---------|
| Central incisor area | 2.5 % | 0.2 % | 2.7 % |
| Lateral incisor area | 3.6 | 0.2 | 3.8 |
| Cuspid area | 5.0 | 2.9 | 7.9 |
| First bicuspid area | 5.2 | 1.1 | 6.3 |
| Second bicuspid area | 4.8 | 3.4 | 8.2 |
| First molar area | 6.8 | 12.6 | 19.4 |
| Second molar area | 2.6 | 5.1 | 7.7 |
| Third molar area * | 20.0 | 24.9 | 44.9 |
| Total: | 51.5 % | 48.5 % | 100.0 % |

* Includes tuberosity and retromolar areas

Table 1: Common locations of NICO lesions.

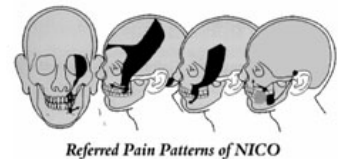


Figure 2: Referred pain patterns of NICO lesions.

In the last several years, the term cavitation has been used to describe various bone lesions which appear both as empty holes in the jawbones and holes filled with dead bone and bone marrow. In Table 1, common locations of NICO lesions are listed. Note that the most common locations overall are areas of wisdom teeth (third molars).

Often, these NICO lesions take years to develop, usually producing few if any symptoms . . . for a while. Then, generally for unknown reasons, pain in the jaws, face, head and neck may develop. There are characteristic referred pain patterns, which generally confuse patients and doctors alike (Figure 2).

Current Research

The results of recent research of Dr. Boyd Haley (former Chairman, Department of Chemistry, University of Kentucky) show that **ALL** cavitation tissue samples he's tested contain toxins, which significantly inhibit one or more of the five basic body enzyme systems necessary in the production of energy. These toxins, which are most likely metabolic waste products of anaerobic bacteria (bacteria which don't live in oxygen), may produce significant systemic effects, as well as play an important role in localized disease processes, which negatively affect the blood supply in the jawbone. There are indications that when these toxins combine with certain chemicals or heavy metals (for example, mercury), much more potent toxins may form.

Systemic Problems Associated With NICO Lesions

Researchers early in the 20th century and now recently have been concerned with systemic diseases caused by a primary problem (a focus of infection). The focal theory of infection fell out of favor with medical and dental doctors after the advent of antibiotics, but many researchers today believe that in spite of antibiotics, the focal theory of infection is alive and well. Ask a veterinarian doctor, and he or she will immediately agree that the focal theory of infection is a great concern of theirs.

Many researchers today believe that NICO lesions are the focus of various infections which may spread throughout the body. In the last few years, some of the most surprising medical news has been the discovery that bacteria from the mouth appear to be very influential in causing various heart, liver and kidney problems. If you have a joint implant or mitral valve prolapse, your dentist must prescribe an antibiotic before any dental treatment. Why? Because bacteria from the mouth can spread through the blood to cause serious problems elsewhere in the body. Could the toxins from NICO lesions do the same?

Initiating, Predisposing, and Risk Factors for NICO

There are many initiating, predisposing, and risk factors associated with cavitational lesions. It's likely that a combination of these factors present in a someone may influence the occurrence, type, size, progression and growth patterns of a cavitational bone lesion.

Initiating Factors: Probably the major initiating factors are dental trauma, which produce physical, bacterial, and toxic components, as described below.

Table 2: Dental traumas (initiating factors) associated with cavitational bone lesion development.

| Physical Trauma | Bacterial Trauma | Toxic Trauma |
|---|--|-----------------------------|
| Tooth Extractions | Periodontal Disease | Dental Materials |
| Dental Injections | Cysts | Root Canal Toxins |
| Periodontal Surgery | Abscesses | Anesthetic by-Products |
| Root Canal Procedures | Root Canal Bacteria | Anesthetic Vasoconstrictors |
| Grinding and Clenching | Non-vital (dead) Teeth | Chemical Toxins |
| Electrical Trauma from Dissimilar Metallic Restorations | Improper Removal of Periodontal Ligament after Tooth Extraction? | Bacterial Toxins |
| Heat from High Speed Drilling | Infected Wisdom Teeth | Other Toxins |

Predisposing Factors: There are many predisposing factors and no doubt, many more will be discovered. Most of the known predisposing factors include: blood clotting disorders such as thromophilia, hypofibrinolysis, or others; age -- evidence suggests that as many as 11% of older persons may have major or complete blockage of arteries feeding the jaws or of the smaller arterioles within the jaws themselves; radiation or chemotherapy for cancer; rheumatoid arthritis; lymphoma or bone dysplasia; changes in atmospheric pressures in occupations; osteoporosis; systemic lupus erythematosus; sickle cell anemia; homocystinemia; Gaucher's disease; hyperlipidemia; hemodialysis; gout; antiphospholipid antibody syndrome; physical inactivity (bedridden); and deficiencies of thyroid or growth hormones.

Risk Factors: There are many risk factors which greatly increase the probability of the development of cavitational lesions, especially in the occlusion or blockage of tiny blood vessels within the jawbones. The most common risk factors are: heavy smoking; high and long-term cortisone usage; pregnancy; estrogen use; alcoholism; and pancreatitis. Undoubtedly, there are many other risk factors.

Wisdom Teeth Sites: Research findings indicate that 45% to 94% of all cavitational lesions are found at wisdom teeth extraction sites. These areas are anatomically predisposed to develop these bony lesions because they contain numerous tiny blood vessels which are apparently, easily damaged from trauma (oral surgery in these areas) and osteonecrosis can easily develop. Also, many local anesthetic injections are given in the wisdom tooth areas and many of the local anesthetic solutions contain vasoconstrictors (especially epinephrine) which is used to intentionally close or shut-down the blood supply to the bone, teeth and gingiva to prolong the effects of the anesthetic and reduce bleeding. The actions of closing down the blood supply to these wisdom tooth areas may be a major cause for NICO development.

[Top](#)

The Appearance of NICO Lesions

Figure 3: Gross appearance of NICO lesions. Note that at least 4 lesions are visible. IAN: inferior alveolar nerve.



Cavitation lesions are difficult to discover. On most x-rays, unless the doctor is specifically trained, these bony lesions are usually missed.

Gross examination of NICO lesions are shown in Figure 2. Note the large nerve, the inferior alveolar, as it travels through and between NICO lesions.

Dental students and residents spend a lot of time learning to properly read x-rays of all types. A very useful x-ray view in dentistry is the panoramic radiograph.

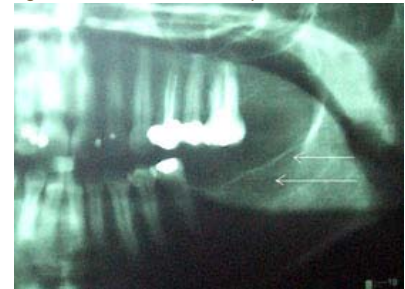
Unfortunately, all of us were trained to read certain irregularities as normal! We now know that these irregularities on panoramic x-rays are quite often cavitational lesions.

Figure 4: A normal panoramic x-ray?



See the panoramic x-ray above (Figure 4)? Most dentists, oral surgeons, radiologists, and other doctors would read this x-ray as normal.

Figure 5: NICO lesions in left posterior mandible.



Now, look again at the same x-ray, but with lines drawn to the NICO lesions (Figure 5).

This 44 year old lady had left lower jaw pain for a couple of years, after the last two molars were treated with large fillings, then root canals, and then removed. She also had a slow drainage into her mouth which produced a sore throat. Unfortunately, this lady saw at least 8 doctors (a dentist, 2 oral surgeons, a periodontist, an endodontist, 2 ENT physicians, and a family physician) and all could find nothing and even suggested she consult a psychologist!

Here's another interesting case. This is a 47 year-old business woman who has had extensive and good dental treatment. Her wisdom teeth were taken out when she was 14 years old and the surgery was difficult. When she was first married at age 18, she took birth control pills for only a few weeks because she developed phlebitis in the deep veins of her legs. When she was 45 or 46, she began experiencing deep aching pain in her lower right jaw. There was no swelling, but she complained of a terrible, sour taste.



Figure 6: An apparently normal panoramic x-ray.

Look at Figure 6. This is her panoramic x-ray. From the looks of this x-ray, there appears to be nothing wrong, yet she had continual deep aching pain in the right lower jaw, a sour taste and no teeth which seemed painful or sensitive.

Figure 7 is a copy of this lady's Cavitat or ultrasonic scan of her lower right jaw. Tooth #28 was removed years earlier for orthodontic reasons.

Note the red, yellow and brown colors in the areas of teeth numbers 29 through 32. These colors indicate areas in the bone of reduced blood flow or dryness, or in other words, a cavitation or cavitations. When this lady's lower jaw was numbed with a local anesthetic, all her pain subsided, but her sour taste persisted. This, along with her symptoms and Cavitat scan, indicated that she had a cavitation in the areas of teeth numbers 29 through 32.

Figure 8 is a picture during surgery of this area. Note the large, void area in the jaw bone. This cavitation area was present within the bone and not created by Dr. Shankland. The two last molars were removed, but the cavitation is lateral to the teeth. Her surgery was difficult and she had minor nerve damage for a few weeks. But today, more than four years after the surgery, she's pain-free and no longer has a sour taste in her mouth.

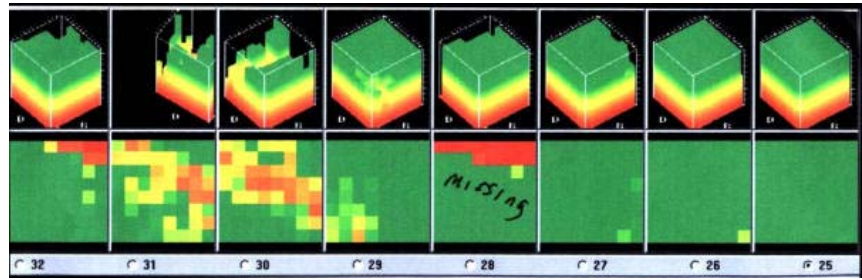


Figure 7: Ultrasound (Cavitat) scan of lower right jaw.

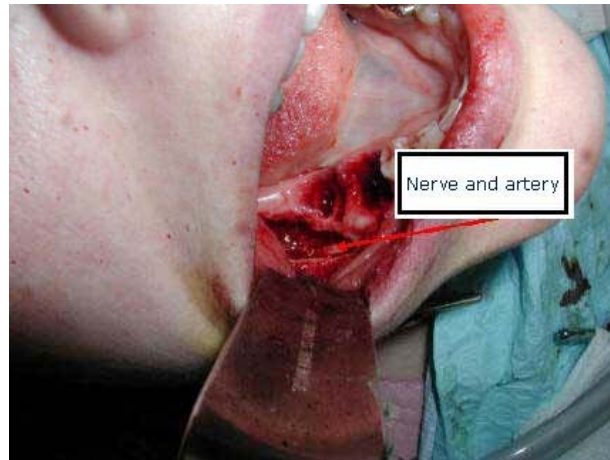


Figure 8: Cavitation in lower right jaw at surgery.

Not to belabor the point, look at Figure 10, which shows yet another case. Is there any doubt that there's a hole in this lady's upper jaw? At this point, only the gum tissue was lifted up; no bone was removed. This lady had constant upper jaw pain, pressure and a sour taste in her mouth. She was diagnosed with trigeminal neuralgia and scheduled for brain surgery. This bone lesion went clear through her jaw into her palate and up into the floor of her nose.



Figure 10: Cavitation in upper jaw of 58 year-old woman.

In one last case, this lady had undiagnosed right facial pain for years. She complained of a sour taste at times and when the sour taste wasn't present, she'd have intense pressure in her right jaw. Figure 11 shows her ultrasound or Cavitat scan. Note the red area in the area of #31 and as it extends into the area

of #30. Again, this shows an area of ischemia, or reduced blood flow, which is actually a jaw bone cavitation.

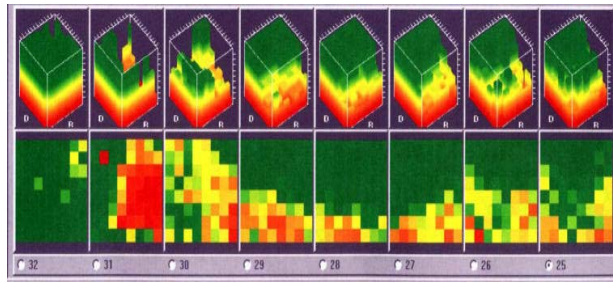


Figure 11: Cavitat scan of lower right jaw.

Figure 12 shows a panoramic x-ray of this same patient. If you look closely at the x-ray, it will look normal and you'll not be able to see any abnormality. Yet, look at Figure 13, a picture taken after an incision was made and nothing else done at the surgical site. Look at the large cavitation at a former extraction site.



Figure 12: Panoramic x-ray of the same patient shown in Figure 11.



Figure 13: A cavitation visible after an incision and the gingival tissue is retracted. This is a former extraction site.



Figure 14: The same cavitation with the bony roof removed..

Now, look at Figure 14, a picture of the same patient with the roof of the cavitation removed for access to surgically repair the area. Isn't that amazing? Several fine doctors couldn't diagnose this lady's problem and most thought she was crazy! Fortunately, she's doing fine with no further pain, sour taste and pressure and no nerve damage after the surgery.

Diagnosis of Cavitations

The diagnosis of cavitation lesions is complicated by the fact that x-ray examination of the jawbones often appears normal . . . to the untrained eye. Considerable diagnostic experience is required to detect disorders that mimic cavitations, including variations of normal anatomy.

Why is this so? Osteonecrosis is a disease of the marrow spaces of bone and 40% to 50% of such bone must be destroyed before changes can be seen on x-rays. So, if your dentist or oral surgeon takes an x-ray and pronounces the film normal in spite of your symptoms, don't necessarily believe it. X-rays may be interpreted as normal unless (1) there's a significant amount of bony destruction or (2) the doctor is experienced in reading x-rays specifically for cavitations.

Although MRI (magnetic resonance imaging) is the imaging technique of choice for long bones, flat bones of the face are not imaged well with regular MRI scans. CT scans are also ineffective in locating most cavitations in the jawbones.

However, we've discovered that using the technique of MRI STIR imaging (Figure 15) is very effective and accurate in locating areas of bone marrow edema (swelling) and ischemia (areas of reduced oxygen). Both of these conditions can and do lead to the formation of cavitations.

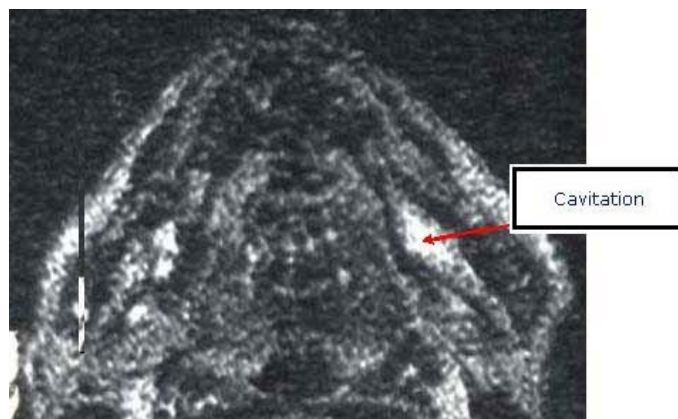


Figure 15: MRI STIR image. The cavitation is the larger white area on the right side of the picture.

Bone scans using a radioactive isotope are somewhat helpful in locating cavitations but very difficult to interpret. Also, radiologists, not expecting these lesions in jawbones, often note the lesions in their radiology reports but interpret the results as normal.



Figure 16: Bone scan using tech 99 radioisotope. The cavitation is the darker area in the lower right front.

The best, most effective method to locate cavitations is the Cavitat bone scanning device (Figure 17). This computer-based sonar imaging system was designed to aid the medical community with a detailed profile of the interior of bones. The Cavitat computer generates digitized two and three dimensional images of the interior of the jawbones from sound waves passed through the bones.

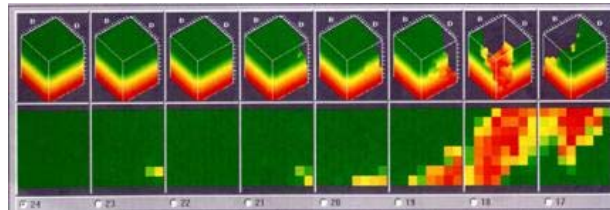


Figure 17: Cavitat scan.

Because liquid is a near perfect conductor of sound waves, when these waves enter into voids or porosities in bone (areas that have compromised bone flow; i.e., cavitations), the sound waves slow down considerably, which produces images of the interior of the bony area being scanned. We've found the Cavitat results to be very accurate, especially when compared with patients' panoramic x-rays. Our diagnostic results have improved dramatically. Most importantly, our surgical successes have soared since we began using this revolutionary device.

Therefore, since both MRI STIR imaging and ultrasound imaging (Cavitat) are so effective and accurate (Figure 18), since November of 2003 we're been using both imaging techniques with most patients. Using both of these diagnostic tests have helped improve our diagnostic abilities and better yet, have improved our overall success rate in treating cavitations of the jaws.

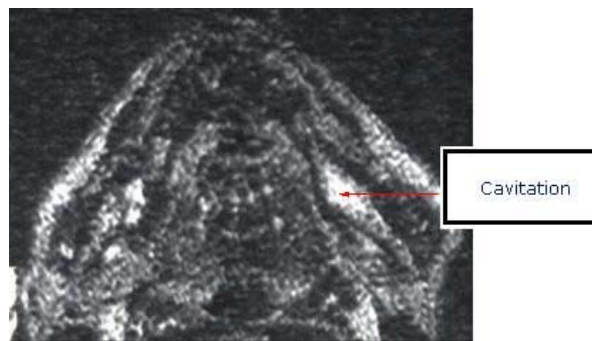


Figure 18: MRI STIR image and Cavitat scan of the same area, both demonstrating a cavitation in the same area.

For patients experiencing pain, diagnosis is further improved through anesthetic confirmation or anesthetic blocking. By giving a local anesthetic injection (similar to having your dentist numb the jaw before he or she performs a dental procedure), pain in the jaws can be selectively turned-off, meaning the sense or feeling of pain can be chemically and temporarily eliminated. If the pain goes away after the injection, then we can be reasonably certain that there's a problem in the anesthetized area, generating pain.

Recommended Treatment of Cavitational Lesions of the Jaws

The only treatment available at this time to remove cavitational lesions is surgical removal. Some have attempted to inject homeopathic remedies or ozone into these areas of dead bone, but unfortunately, there's no blood circulation within cavitational lesions, so any medications, drugs, or remedies can't get into and permeate these lesions, let alone allow toxins and metabolic products to be removed. Homeopathic remedies certainly have their place in NICO treatment, especially in healing after surgical removal of the lesions themselves.

The surgery basically consists of making an incision, exposing the bony defects, and scraping them clean

(termed *debridement*) to remove all unhealthy bone and other pathological problems like abscesses and cysts. It's not sufficient to simply punch a hole in the bone and rinse the area out, like some doctors recommend. In fact, treating these expanding bony lesions in such a conservative fashion often makes the lesion and subsequent pain much worse.

After removing the dead bone and other pathological products, the goal in healing is bone regeneration. But first, if possible, we remove all predisposing and risk factors.

What Can A Patient Do?

If you think you might have a NICO lesion, what can you do? First, find a doctor who understands this disease process; one who is trained in effectively diagnosing and treating these bony problems. Unfortunately, there are precious few such doctors in the world and very few in North America at this time.

If you're experiencing pain, don't allow anyone to operate without first proving where your pain originates. This is done most effectively by closely evaluating x-rays and using diagnostic anesthetic injections to actually turn-off the suspected NICO areas to see if the pain is turned-off. There are characteristic referred pain patterns of NICO lesions and there are also characteristic responses to local anesthetic testing. Find a doctor who knows about these characteristic patterns and realize that most doctors who treat orofacial and TMJ pain know nothing about NICO lesions.

Be certain that the doctor obtains Cavitat scans, MRI STIR imaging, or both in the process of diagnosing your problem. Both of these imaging tests give us a view of the size and extent of cavitations and can also indicate if surgery is truly needed or not.

Keep watching this site as we have many new and exciting things soon to come out about NICO lesions. For more information about NICO lesions and other orofacial pain problems which are often misdiagnosed, see Dr. Shankland's latest book, [Face The Pain](#).

Dr. Shankland consults and treats NICO, orofacial, and TMJ patients. However, to be in compliance with and cooperate with the Ohio State Dental Board, potential patients must understand that currently, the diagnosis and treatment of osteocavitations is considered experimental and alternative. Further, if you would like to consult with Dr. Shankland concerning this disorder, you first must have a referral from a physician in order to be seen by Dr. Shankland.

If you have any questions, please call Dr. Shankland's office (614-794-0033) and ask for the NICO Information Packet to be sent to you free of charge. You can also [consult with Dr. Shankland](#). If you'd like to know more about Dr. Shankland, click on [Biography](#).

OSTEOCAVITATIONS OF THE JAWS

by Wesley E. Shankland, II, DDS, PhD

Introduction

Chronic undiagnosed pain of the face and head frustrates both patient and doctor. Each of us has a 24.3% risk of developing chronic facial pain by age 50 and a 33.8% risk by age 70,¹ yet only 46% seek treatment.² In 1995, the cost of chronic orofacial pain was estimated to be more than \$32 million per year.³ Undoubtedly, the cost is much higher today.

One of the most devastating and incapacitating pain disorders of the face is trigeminal neuralgia, or classically known as *tic douloureux*.

Unfortunately, dentists and physicians are trained to diagnose facial pain as trigeminal neuralgia if the patient reports sharp, shooting, electrical pain or even deep aching, constant pain of an unknown origin. Sadly, doctors, regardless of degree, do not distinguish between typical and atypical trigeminal neuralgia and such an error may very well destin a patient to inappropriate and unnecessary invasive procedures.⁴ There are several theories concerning the cause of such severe facial pain. One theory, the *peripheral theory*, states that a low-grade, chronic, intraosseous infection near a branch of the trigeminal nerve may produce neuronal degeneration or demyelination producing inappropriate nociceptive signals to the central nervous system.^{4,5,6} These bony infections have come to be known as *cavitations* in the general population. To the medical community, these bony lesions are a result of ischemic bone disease, which includes osteoporosis, osteomyelitis, osteonecrosis and other bone disorders. Pure and simple, these cavitations are cavities within the jaw bones (Figure 1), although they occur in every bone in the body that contains bone marrow. Ischemic bone disease is not an actual disease in and of itself. Rather, it is a result of many local and systemic events or disorders that ultimately lead to ischemia and infarction of the bone marrow.

Dental surgeons in the United States accepted the idea of cavitations causing trigeminal neuralgia in the 19th century.^{7,8} In 1915, G.V. Black, the father of modern dentistry, wrote an entire section in his eloquent pathology textbook⁹ on this subject and termed this condition *chronic osteitis*. It was revisited later in this century by British neurosurgeon Sir Wilfred Harris,^{10,11,12} in America by Thoma,¹³ a well-known and very respected oral and maxillofacial surgeon and by Box,¹⁴ a Canadian oral pathologist. Recently, this theory has been gaining acceptance in the

Dental surgeons in the United States accepted the idea of cavitations causing trigeminal neuralgia in the 19th century.^{7,8}

United States (again),^{5,6,15,16,17} Germany,¹⁸ Russia,¹⁹ China^{20,21,22} and throughout most of the world. The concept of cavitations is supported voluminously by the orthopedic literature, including an entire book devoted completely to the subject of osteonecrosis.²³ Therefore, most physicians, although they may not understand the intricacies of ischemic bone disorders, accept the concept without hesitation. Dentistry, on the other hand and for some unknown reason, has

There is a growing controversy in dentistry today concerning endodontic therapy (i.e., root canals) of dead teeth.

arrogantly rejected even the thought of the existence of jaw bone cavitations. Perhaps such intellectual suicide permeates the dental profession because so many cavitations develop in conjunction with teeth treated with root canal therapy, yet this is just one cause of these disorders.

These jawbone cavities or lesions have been described in the literature as jawbone cavities,^{2,3,17,24,25,26} osteocavitation lesions,²⁷ pathologic bone cavities,²⁸ odontogenic trigeminal neuralgias,¹⁶ alveolar cavitation osteopathosis,²⁹ trigger-point bone cavities,²⁵ Ratner bone cavities,²⁷ Roberts bone cavities,²⁷ intrabony mandibular pathosis,³⁰ and cavitation bone defect.³¹ However, Bouquot et al³²

proposed the term *neuralgia-inducing cavitation osteonecrosis* (namely, *NICO*) as a more descriptive name for cavitations that produce jaw pain.

Etiology of Cavitations

Cavitations of the jaws are caused by a variety of predisposing local and systemic factors as listed in Table 1. To date, there are at least seventy-four (74) known risk factors for ischemic bone disease and each one may influence the development of hip osteonecrosis, jaw cavitations or osteonecrosis in virtually any bone. Should one be surprised that such inflamed or necrotic areas of the bone marrow of the mandible or maxilla develop when the oral cavity is so

potentially pathologic and dental surgeons perform invasive procedures, only to dismiss the patient from the office with a gaping, open wound which exposes the bone marrow of the jaw to the microbes and debris of the mouth? No other type of surgeon would even consider such post-operative care.

At the very least, the jaws are unique in that they possess small structures which may die, develop abscesses and provide a direct route to the underlying and surrounding bone marrow: teeth. Osteonecrosis of the jaws occurs in approximately the same frequency as it does in the hip, but unlike the jaws, the hips do not have the disadvantage of teeth embedding in them.

There is a growing controversy in dentistry today concerning endodontic therapy (i.e., root canals) of dead teeth. Many feel that such dental treatment has saved millions of teeth and has maintained the ability of many to chew effectively, and they are correct. However, what other discipline in medicine purposely leaves dead objects in the body and expects no morbidity? In addition, is it coincidental that so many develop jaw bone cavitations in the same areas where endodontic therapy has failed?

“CAVITATIONS” CONTINUED...

Regardless of the local or systemic etiologies of cavitations, the overall problem is intravascular coagulation within the small vessels in bone marrow producing bone marrow edema. This compromised medullary blood flow and subsequent edema produce intraosseous pressure, causing vessel collapse, stagnation of blood flow, thrombus formation with the eventual development of ischemia. **Unresolved ischemic conditions ultimately produce the most devastating of the ischemic bone disorders: osteonecrosis** (Figure 2).

The most common areas of cavitation formation are the molar areas and specifically, the third molar areas. These are areas furthest from the heart with turgid and slowed blood flow. The same is true for osteonecrosis of the femur or tibia: the areas further from the heart are the more common regions for ischemia.

There is also a high percentage of cavitation formation below and around root canal treated teeth and especially areas of teeth that have undergone failed root canal therapy and continual pain after oral surgery, especially if alveolar osteitis (i.e., dry socket) develops after tooth extraction. Most teeth treated with endodontic therapy fail because of the inability to adequately cleanse all microorganisms from the root canal system.^{33,34,35} Such chronically inflamed areas around the roots of these teeth produce ischemic conditions, oftentimes with subsequent cavitation development.³⁴

Symptoms

The symptoms of jaw bone cavitations are: (1) a history of undiagnosed facial and cervical (neck) pain, often diagnosed as trigeminal neuralgia; (2) a history of tooth extraction; (3) the presence of trigger areas; (4) the history of alveolar osteitis (*dry socket*)³⁶; (5) the history of endodontic therapy and, (6) normal radiographic findings. Patients suffering jaw bone cavities will have a history of tooth extraction perhaps years before their pain complaints begin. The pain radiates in specific patterns and may even radiate into the shoulders, neck and arms.³⁷

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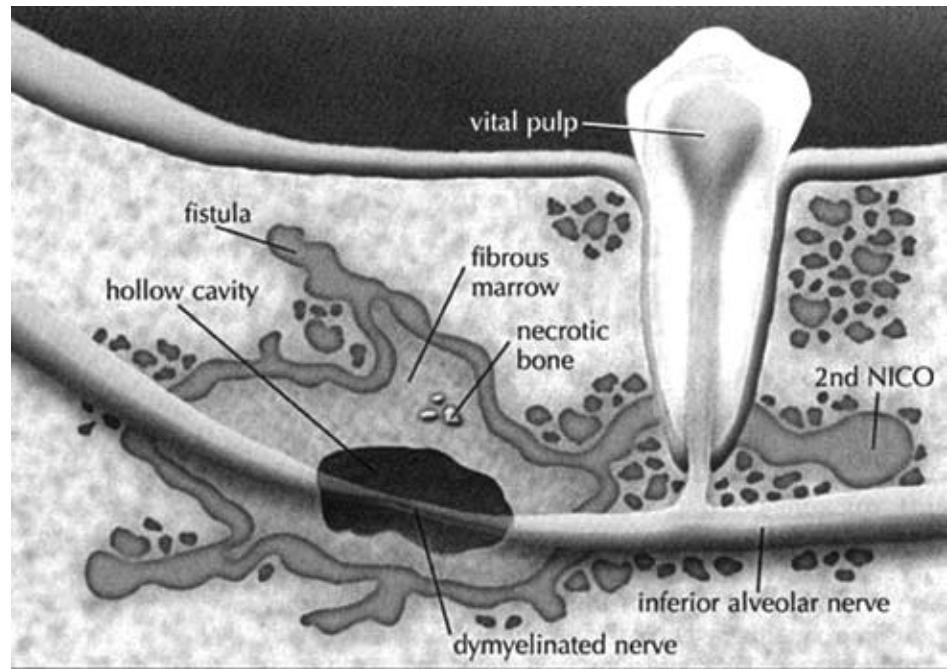


Figure 1:

Diagram of jaw bone cavitations in the mandible. Note the fistula and the secondary cavitation (NICO). From: Shankland, WE: “Common Causes of Non-Dental Facial Pain.” *Gen Dent* 1997;45(3):246-252. Used with permission.

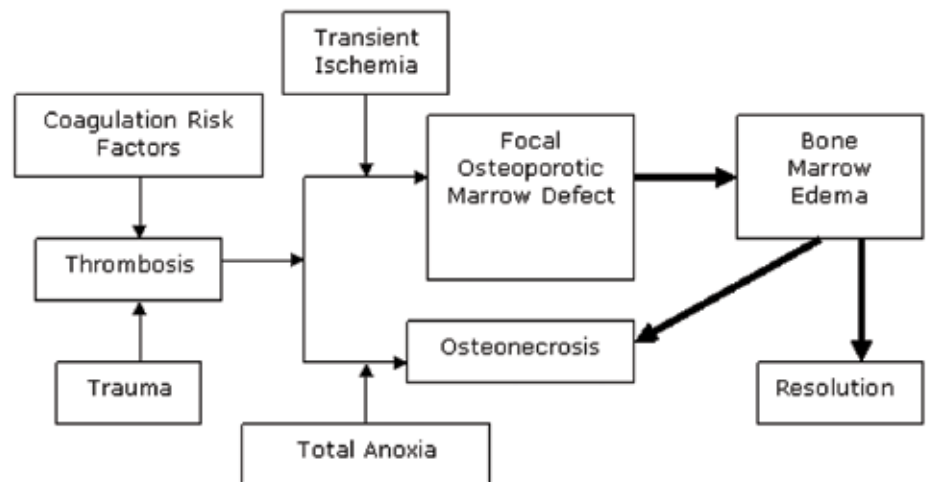


Figure 2:

Simplified diagram suggesting that persistent transient ischemia in bone marrow may be the etiological factor in the development of bone marrow edema, which may resolve, or the development of osteonecrosis (cavitation). From: Shankland, WE; Bouquot, JE: “Focal Osteoporotic Marrow Defect: Report of 100 New Cases With Ultrasonography Scans.” *J Craniomand Pract* 2004; 22:314-319. Used with permission.

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Trigger areas are found intraorally and when compressed with finger pressure, may reproduce specific pain patterns. Unfortunately, this orofacial pain is most often misdiagnosed as trigeminal neuralgia.^{4,17,25-32}

Another common complaint of those suffering with osteocavitations of the jaws is a distinct sour or putrid taste. This is simply necrotizing bone marrow escaping through a drainage tract into the mouth. Such drainage often produces a chronic sour throat, gastritis, irritable bowel symptoms and diarrhea.

Probably the most striking and confusing symptom of the presence of osteocavitation lesions is *normal radiographic findings*. Bender and Seltzer^{38,39} and Schwartz and Foster⁴⁰ have demonstrated that lesions may be present in bone, both mandibular and maxillary, which are not visible on radiographs until there is at least 45% to 50% loss of bone. Initially, clinicians have difficulty visualizing subtle loss of bone density, which may well indicate a cavitation, on panoramic radiographs. This is perhaps the chief reason why most dentists reject the idea of jaw cavitations, because they can not see these lesions on radiographs, therefore, to them, they do not exist.

Diagnosis

According to Bouquot and LeMarche, cavitations are infamous for their lack of obvious radiographic changes until extensive destruction has occurred.⁴¹ Plain MRI scans are not fully revealing, but MRI STIR imaging and thermography show great promise in identifying these lesions. In addition, bone scans with radioisotopes such as technetium show radioisotope uptake or “hot spots” at the sight of cavitations, but facial bone scans are difficult to interpret and these positive findings are often misdiagnosed as periodontal disease or sites of recent extractions.

The most accurate imaging device

available to dentistry today to aid in the diagnosis of cavitations is a specialized ultrasound device, the Cavitat. In February 2002, this type of bone sonography, when used in conjunction with a panoramic radiograph, was cleared by the U.S. Food and Drug Administration for the express purpose of evaluating the wetness (i.e., blood perfusion) and density of the medullary bone of the jaws. The Cavitat instrument uses a through transmission technology rather than the reflected

Most teeth treated with endodontic therapy fail because of the inability to adequately cleanse all microorganisms from the root canal system.^{33,34,35}

signals of other ultrasound instruments used in medicine. Normally hydrated bone and water conduct sound waves in a similar fashion. Sound waves traveling through regions of diminished bone hydration, reduced density or intramedullary cavitations (i.e., an air space void of hydration) are reduced in intensity, thus registering on the receiver with less intensity than waves traveling through normal bone.⁴² Sound impulses are converted into digitized graphs, generated as 2-dimensional or 3-dimensional images.

There is no question that tissues removed from cavitation lesions are abnormal.³²

This unique instrument generates 27,000 sound pulses per microsecond. Teeth have the same characteristics as bone; therefore, unlike on radiographs, the teeth and bone are not visible as separate structures. Each 3-dimensional color-coded image represents a 1:1 cm² area exposed to the ultrasonic signal. Up to 32 individual exposures can be made of the alveolar bone: eight in each quadrant, each representing a specific tooth site.

Each 3-dimensional image shows 64 colored columns or data points, each corresponding to 1/64th of the intraoral receiver. Good sound transmission shows as green, with attenuated transmissions appearing in the following order: yellow, brown, orange and red. Based upon Cavitat images, cavitations have been divided into five (5) grades, 0 through 4, with grade 4 being the most ischemic.

The clinical diagnosis is most accurately confirmed by histological examination of the tissue samples removed from the cavitation sites at the time of surgery. There is no question that tissues removed from cavitation lesions are abnormal.³² Generally, histological evaluation of the samples reveals bone marrow fibrosis, lymphocytic infiltration, and/or necrotic bone chips. Usually, there is very little evidence of osteoclastic activity, new bone formation, or bone repair. Unlike osteomyelitis, neutrophils are almost never associated with cavitation. Perhaps the most unique histological aspect of cavitations is the absence of new bone formation or healing;

this is not the case with osteomyelitis.⁴³ Also, osteomyelitis usually contains foci of isolated suppuration and exhibits a clinical course of acute exacerbations followed by periods of quiescence. The painful type of cavitations, NICO, lacks these painless periods; NICO is intensely painful without suppuration.

Treatment

The only treatment for later staged cavitations is surgery and therein lies a clinical problem. The earliest lesions, grades 0 through 2, may be treated conservatively with supplements, antibiotics, injections of ozone and homeopathic remedies or infrared reported as being efficacious. The problem is this: lesions of grades 0 through 1 are rarely painful and therefore, rarely detected and subsequently not treated in these early stages. Once a lesion reaches the grade of a late 2 or higher, the ischemic conditions (and therefore, lack of blood flow through the lesion) negate any benefits from such conservative therapies.

The most effective conservative therapy this author has witnessed is the use of supplements that either decrease platelet aggregation or increase the luminal size of the small vessels in the bone marrow, or both. Table 2 lists several of the more effective supplements for these purposes.

For most symptomatic and later staged

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Table 1:

Some of the more common risks factors of ischemic bone diseases.

Local factors

- Infection
- Trauma
- Alveolar osteitis (dry socket)
- Local injection of cortisone
- Bone infection and/or inflammation
- Failed root canal therapy
- Use of vasoconstrictors in local anesthetics

Systemic factors

- Pregnancy
- Birth control pills
- Estrogen therapy
- Corticosteroid therapy
- Hypofibrinolysis
- Thrombophilia
- Antiphospholipid syndrome
- Heavy smoking
- Autoimmune diseases (e.g. systemic lupus erythematosus)
- Malnutrition (starvation, anorexia)
- Anemia
- Alcoholism
- Frequent hyperbaric changes
- Radiation and chemotherapy
- Metastatic cancer
- Bisphosphonates
- Disseminated intravascular coagulation
- Hypothyroidism
- Hereditary clotting disorders
- History of deep vein thrombosis or phlebitis

Table 2:

Supplements which are effective in treating the early stages of ischemic bone diseases.

| <u>Supplement</u> | <u>Suggested Dosage per day*</u> |
|--------------------------|---|
| ▪ Ginkgo biloba | 80 to 100 mg |
| ▪ Vitamin B complex* | 1 tablet every 12 hours |
| ▪ Garlic | 900 mg |
| ▪ Ginseng | 100 to 200 mg |
| ▪ St. John's Wort | 900 to 1,800 mg |
| ▪ Rhubarb | 100 mg |
| ▪ Vitamin E | 800 to 1,200 IU |
| ▪ Coenzyme Q-10 | 50 to 1,000 mg |
| ▪ Omega 3 fatty acids | 5 grams |
| ▪ Flax seed oil | 3 grams, twice daily |
| ▪ Green tea | No established dose |
| ▪ Feverfew | 50 to 150 mg |
| ▪ White willow | 100 to 250 mg |
| ▪ Ginger | 1 to 4 mg |
| ▪ L-Arginine | 1,000 mg every 6 hours |
| ▪ Nattokinase | 100 mg two to three times |
| ▪ Baby aspirin | 1 daily |

**Doses will vary for each person. These are approximate values. Contact your health care professional for your specific needs regarding any supplements.*

Table 3:

Microbes isolated from osteocavitation lesions.

- Cladosporium species
- Candida albicans
- Candida dubliniensis
- Exophiala species
- Staphylococcus
- Pseudomonas aeruginosa
- Actinomyces odontolyticus
- Actinomyces israelii
- Moraxella catarrhalis
- Klebsiella pneumoniae
- Beta hemolytic Streptococcus B
- Saprophytic Cladosporium species
- Mixed skin flora
- Bacteroides fragilis
- Prevotella
- Porphyromonas
- Streptococcus fecalis

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lesions, the only choice of treatment is surgery. Approximately one-third of the lesions are hollow or cavities partially filled with blood. The shape is usually irregular and the bone is generally discolored. The size ranges from a few millimeters to several millimeters. The lesion or lesions must be totally removed by meticulous curettage of the contents of the lesion saving the material removed for biopsy. As many as 59% have multiple sites,⁴⁴ many of which are in different regions of the jaws. It is wise that a culture be performed in order to identify any microbes or yeast which are sometimes discovered in these bony lesions (Table 3), many of which are not endemic to the jaws. Based upon the culture results, accurate prescribing of an antibiotic, if necessary, can then be recommended.

Post-operative treatment is generally palliative in nature: anti-inflammatory medications, a soft diet for several days, rest and contrary to all other types of bone surgery, *no application of ice*. Although the use of ice greatly decreases post-operative swelling, the cold temperature also slows the flow of blood and therefore, may theoretically, enhance conditions for pre-mature blood clotting and in the environment of a surgical site that is in the process of healing.

What is the success of cavitation surgery? The answer depends upon what one considers success. In eleven separate studies^{5,6,15,16,19,22,26,45,46,47,48} which included a total of 2001 patients who had undergone cavitation surgery, the median post-operative pain reduction ranged from 50% (26 patients)¹⁶ to 100% (184 patients).^{5,19,26,49} Unfortunately, when considering all these studies together, nearly 40% needed to be re-operated, probably due to the difficulty in reestablishing adequate blood flow during healing. In a separate single long-term study of 103 patients treated surgically,⁵⁰ after an average of 4.6 years, 59.2% were pain-free and an additional 13.6% reported that they were considerably improved, meaning that they were taking no prescription medications and described their pain as “almost gone.” Twenty-two percent of these 103 patients required an additional surgery.

Conclusion

Osteocavitations of the jaws are frequent causes of undiagnosed facial pain. This painful condition oftentimes is misdiagnosed as trigeminal neuralgia and may be treated with medications by a neurologist or neurosurgery by a neurosurgeon. As with all other bones containing bone marrow, areas of infarctions within the small vascular supply of the jaws, if unresolved, may produce ischemic areas which ultimately develop osteonecrosis. If these ischemic regions produce pain, then the condition is termed *NICO*.

There are multiple causes of cavitations, both local and systemic. The ultimate etiology is compromised medullary blood flow and subsequent edema producing intraosseous pressure, causing vessel collapse, stagnation of blood flow, thrombus formation with the eventual development of osteonecrosis.

If detected very early, these bony lesions may be treated conservatively with ozone injections, injections of homeopathic remedies, supplements and infrared. If cavitations are detected later in their development (as is generally the case) when they become symptomatic, the only viable treatment currently is aggressive surgical curettage of the bony lesions. 📖

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Recognized internationally as an authority on the diagnosis and treatment of orofacial pain, Dr. Shankland has written over 100 scientific articles, has written several chapters in textbooks, and has written four books. He has lectured throughout North America, Hawaii, Mexico, the Caribbean, England, Germany, Australia, Norway, South

Africa, Singapore, Malaysia, India and Hong Kong. He's a past-president of the American Academy of Craniofacial Pain.

While in graduate school, Dr. Shankland discovered four (4) structures in the human body that were previously not described in the scientific literature. One structure, the zygomandibularis muscle, has been shown to be a major source of orofacial pain when its tendon is injured.

[Editors Note: For more information on this subject see *Beyond Amalgam*, by Susan Stockton. Available from PPNF, see order page.]

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Focal Osteoporotic Marrow Defect: Report of 100 New Cases with Ultrasonography Scans

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ABSTRACT: Focal osteoporotic marrow defect (FOMD) may be the earliest detectable form of the ischemic marrow disorders. The exact cause is unknown, but three theories have been proposed in the literature. A fourth is presented in this paper. In this study, 100 biopsies were examined histologically and were diagnosed as FOMD, based upon consistent histological characteristics. Until recently, the only diagnostic criteria were radiographic evaluation and incisional biopsy. In February 2002, a through-transmission alveolar ultrasonic test (Cavitat 4000, Cavitat Medical Technologies, Inc., Aurora, CO) was approved by the US Food and Drug Administration and by Health Canada for detection of low bone density and bone desiccation, both features of FOMD and chronic ischemic bone disease. Within this article, the diagnostic criteria and pathological findings of FOMD will be presented. The three current theories concerning its etiology will be briefly presented and a fourth theory will be proposed.

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Focal osteoporotic marrow defect (FOMD) may be the earliest detectable form of the ischemic marrow disorders.¹ First described in the dental literature by Cahn,² these bone marrow lesions present as poorly demarcated radiolucencies. Most lesions occur in the mandibular posterior regions,³⁻⁶ usually at former extraction sites.⁷ Approximately 75% of all cases reported in the literature occur in adult females. Most studies prior to this article have reported that these localized, radiolucent defects are of little consequence and are generally asymptomatic.^{3,8,9} However, Makek and Lello,¹⁰ Lipani, et al.,¹¹ Bouquot, et al.¹ and the results of this study disagree.

Radiographic appearances of FOMD vary greatly from localized radiolucencies with intermittent borders to sharply defined radiolucencies with obvious sclerotic borders. Often, fine trabeculations traverse the radiolucency, and lesions range in size from a few millimeters to several centimeters in diameter.³

The purpose of this study was three-fold: first, to present 100 new cases of FOMD, most of which were symptomatic alveolar radiolucencies diagnosed histologically as FOMD; secondly, to evaluate through-transmission alveolar ultrasonograph (TAU) scan results for FOMD lesions; and thirdly, to propose a new theory concerning the development of this disorder. This series represents the third largest series of FOMD lesions in the literature.

Materials and Methods

One hundred consecutive tissue samples from alveolar sites of localized radiolucency were biopsied and microscopically diagnosed as FOMD. Specimens were harvested from January 1999 through August 2003. All bone samples were decalcified with research grade formic acid and stained with hematoxylin and eosin.

All lesions were discovered by history, oral examination, panoramic radiographic examination, and after discovery, all were subjected to through-transmission alveolar ultrasonography (TAU) imaging using the Cavitat 4000.

The following features were assessed for each case: age, sex, location of the lesion, size of the lesion at surgery, presence or absence of teeth at the lesion site, type of bone marrow seen in the sample, pain upon palpation of the alveolus at the lesion site, source of orofacial pain, and TAU grading using the 5-point scale reported by Bouquot, et al.¹²

Those lesions reported by the patient to be painful without palpation or provocation were blocked with 3% Mepivacaine (without vasoconstrictor), as per the diagnostic anesthesia test described by McMahon, et al.¹³

Results

The mean average age was 50.15 years (± 9.15 years). Thirty-five (35%) of the lesions were in the maxilla; and 65% of the lesions were located in the mandible. Forty-five (45%) were on the right side of the mouth and 55 (55%) were on the left. Seventy-seven (77%) were females (mean average age: 50.80 years ± 9.04) and 23% were males (mean average age: 48.11 years ± 7.99). All of the subjects were Caucasian.

Sixty-eight (68%) of the 100 lesions were located in edentulous alveolar sites (old extraction sites). Fifty-seven (57%) of the lesions were tender with digital palpation (50 female and 7 male), 65 (65%) were apparent generators of pain (56 female and 9 male), and all painful sites were relieved of pain after anesthetic blockade.

The mean average TAU grade of the 100 lesions was 3.01 (**Figure 1**). At surgery, the mean average length of the lesions was 1.62 cm (range: 0.5–3.5 cm) and the mean average width was 1.12 cm (range: 0.2–2.0 cm).

The histological examination of the bone marrow in all cases was normal. In all cases, viable bone was visible, but bony trabeculations often appeared inactive with osteocytes missing. Also, there were two definite categories of bone marrow: fatty or yellow alone (79), or a mixture of fatty and red marrow (21). There were no samples of red marrow alone.

Fifty-five (55%) of the specimens showed evidence of mild inflammation with the presence of lymphocytes. Fifty-one (51%) showed small localized evidence of degeneration, ischemia or necrosis. One hundred (100%) of the samples contained viable bony trabeculae.

Chi square analysis demonstrated that the gender differences for tenderness and pain were significant ($\chi^2=0.0653$; $df=1$; $p<0.01$).

Discussion

All bone marrow in newborns is red. Yellow or fatty marrow begins to appear in long bones and in the mandible from the fifth to the seventh years, and by age 18, almost all long bone marrow is yellow.¹⁴ In the adult jaws, approximately 75% of the marrow is yellow or fatty and 20% is red.¹⁵ Fatty marrow is well vascularized, but almost all vessels are dormant much of the time. Red or hematopoietic marrow, on the other hand, has a rich and active blood supply and is usually located in the maxillary tuberosities and the maxillary and mandibular molar and premolar areas.^{3,16} Red marrow does not seem to occur in the adult in the anterior regions of the jaws.^{3,17} The relatively poor vascularization of fatty marrow makes the bone marrow in the adult jaw vulnerable to trauma, bacterial invasion, vascular congestion, and reduction of blood flow, potentially leading to a myriad of ischemic and inflammatory problems.

Bone marrow defects have been reported in the literature for well over two centuries. Any medullary bone can undergo a variety of marrow changes, some transient and others permanent. The maxilla and mandible are no exception and in fact, it has recently been reported that the prevalence of jaw osteonecrosis is similar to that of the hip.¹⁸ Up to a certain point, it appears that fatty marrow has the ability to be transformed back into red marrow when there is an unusual demand for increased blood cell production.¹⁹ Some authorities⁴ believe the development of this hyperplastic red marrow is seen as reduced bone density or osteoporosis on radiographs. However, these and other authorities assert that the radiolucency produced by FOMD does not represent a pathological disease process.^{4,20} This assertion does not conform with the results of the present study.

In the dental literature, bone marrow defects have been discussed throughout the twentieth century and especially within the last four decades. However, while several papers have discussed the clinical presentation of FOMD, the exact cause remains unknown. Three etiologic theories have been proposed in the literature:^{6,17,21}

1. Marrow hyperplasia following an increased demand for blood cells resulting in bone resorption. Although

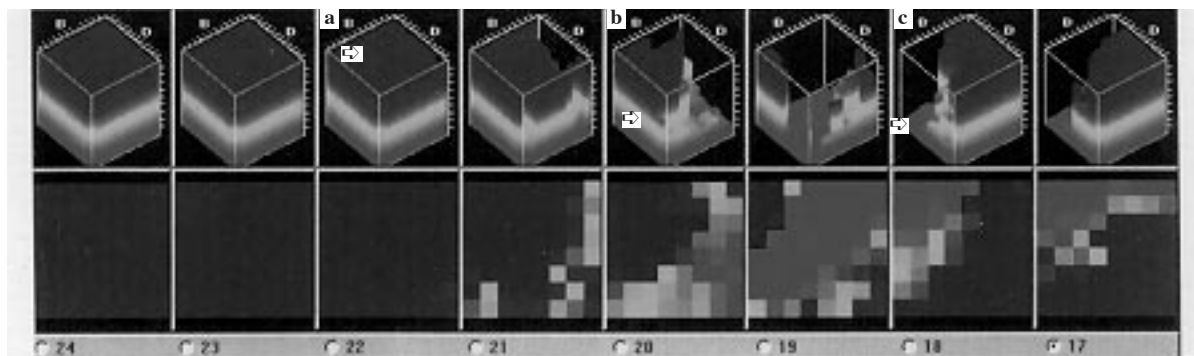


Figure 1

Through-transmission alveolar ultrasound (Cavitat) scan. The computer generates a digitized perspective 3-D image from analog signals received and converted to digital. The image being viewed is not a negative type as viewed on an x-ray but a computerized image rendition from sound waves received on the Cavitat array. The image is color-coded to distinguish the intensity of destruction (desiccation, dryness or ischemic regions), with normal bone represented by dark green (a) mild to moderately damaged bone represented by yellow, yellow-orange, and brown (b), and severely damaged bone represented by red (c). This is the sonogram of the same patient as seen in **Figure 3**. In alveolar areas 17 and 18, the lesion is Grade 2 to 3; in the alveolar area of 19, the lesion is Grade 4; in the area 20, Grade 3; and Grade 1 in the area 21.

hyperplasia may occur in the marrow as a response to demands for increased hematopoietic tissue creation, only a handful of the reported FOMD cases have been associated with systemic anemias.

Local pathological processes, on the other hand, may often produce an osteopenia, or localized osteoporotic defect, but unless 30-50% of the medullary bone is destroyed, no radiographic changes are visible.²² Rather than a normal process, might radiographic changes of FOMD be evidence of bone marrow edema^{23,24} or regional ischemic osteoporosis or early ischemic osteonecrosis?²⁷

2. Residual hematopoietic marrow persisting into adulthood. This may be true, but it is not known to be a pathological problem. In the adult, in addition to the mandible and maxillary posterior regions, hematopoietic marrow persists in other skull bones, the clavicles, vertebrae, ribs, sternum and pelvis. There is no evidence whatsoever that these areas engage in any function other than hematopoiesis. During times of marrow hyperplasia, these areas do not produce abnormal radiographic images, as do ischemic processes (e.g., bone marrow edema, ischemic osteoporosis, ischemic osteonecrosis) or other pathoses (e.g., aneurysmal bone cyst, central giant cell granuloma, traumatic bone cyst, or metastatic carcinoma).

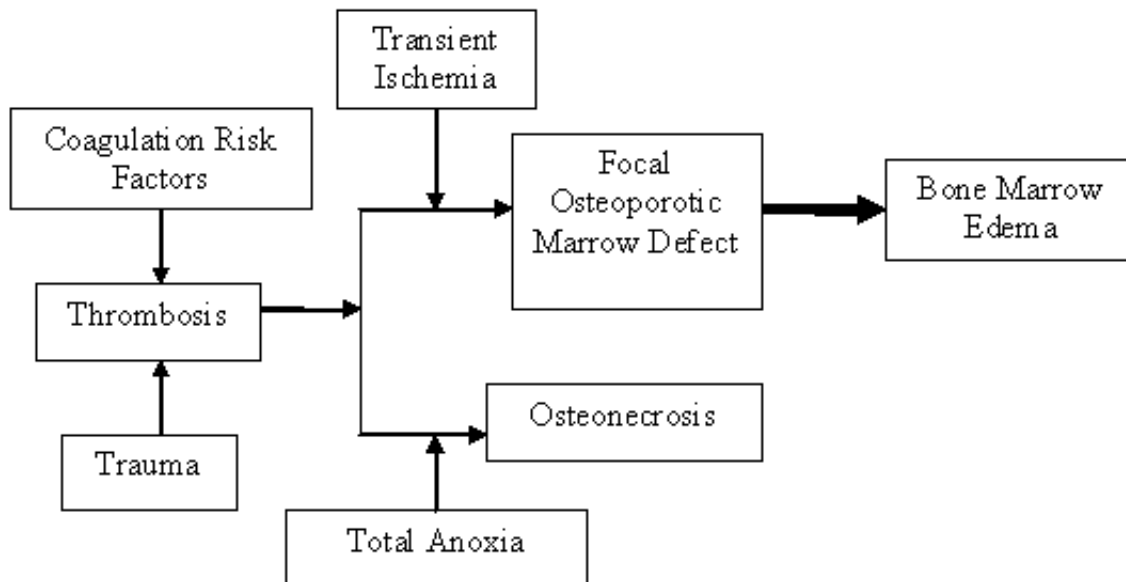
In the case of regional ischemic osteoporosis, radiographs reveal loss of bone density, blurring or loss of trabeculations, and poorly demarcated borders,²⁵ all of which are also characteristics of FOMD. Again, could FOMD be a stage of bone marrow edema and an early form of regional ischemic osteoporosis as Bouquot, et al.¹ suggested?

3. Defective regeneration of bony trabeculae in an area of previous trauma, local inflammation, or surgery.

This may also be true, but what caused the defective regeneration? It is well established that poor bone healing after peripheral infection produces either fibrous scar tissue or localized areas of bone sclerosis, or condensing osteitis. Osteoporotic bone has been a suggested result. However, ischemic bone diseases, such as NICO (neuralgia inducing cavitation osteonecrosis), routinely produce radiolucent areas on radiographs that are ill-defined, in many cases showing irregular remnants of old lamina dura and scattered flecks of sclerotic bone.²⁵⁻²⁷

There may be a better theory to explain the etiology and clinical presentation of FOMD. Could it be an early form of ischemic osteoporosis secondary to a malfunction of blood flow within the marrow? There are multiple reasons for the development of back-up pressure, capillary leakage and an accumulation of fluid in the extracellular marrow spaces (edema) (**Figure 2**), but it is telling that the majority of our FOMD cases showed localized microscopic evidence of ischemic change or damage.

Jones²⁸ asserts that bone marrow edema and osteonecrosis share a common pathway: an acute ischemic event (i.e., a sudden disruption of normal blood flow due to thrombus formation). He proposed that duration of ischemia and deficiency in blood flow are the key factors determining if bone marrow edema or more severe ischemic osteonecrosis will develop. Many researchers believe that bone marrow edema represents an early, possibly reversible, subtype of osteonecrosis.^{25,29,30} Koo, et al.³¹ have postulated that bone marrow edema is a secondary phenomenon that follows an ischemic attack. According to the histological criteria established by Ficat,³² areas of osteonecrosis show hematopoietic and fat cell necrosis in the marrow and osteocytic death. In our

**Figure 2**

Simplified diagram suggesting that persistent transient ischemia in bone marrow may be the etiological factor in the development of focal osteoporotic marrow defect.

study, 51 (51%) of the biopsies demonstrated the presence of ischemia, bony degeneration, or necrosis. In addition, 79% contained a mixture of fatty and hematopoietic marrow. Yet, in all samples, the trabecular bone was still viable. Based upon these findings, it appears, at least in this study, that FOMD may be an intermediary step between an ischemic event with the subsequent development of bone marrow edema and the subsequent possible development of ischemic osteonecrosis. FOMD may correspond to Stage II of the classification, according to Ficat and Arlet.^{33,34} In Stage II of this classification system, radiographs show definite abnormalities consistent with osteonecrosis: areas of radiolucency, sclerosis, and generalized osteopenia, all of which are seen radiographically in FOMD.

Earlier reports have alleged that those with FOMD do not report pain,^{4,7,21} but according to this study (57% of the areas were tender to palpation and 65% were generators of pain) and others,^{10,17,27} pain occurs and in a high percentage of those afflicted.

The frequency of painful FOMD is difficult to determine. Shankland²⁷ reported that in a study of medullary jaw diseases in facial pain patients, 4% (20 in 500) represented FOMD. However, in a recent and yet unpublished study, the number of FOMD cases was 14.6% (135 in 932). Obviously, the incidence of FOMD has yet to be accurately established, but most clinicians, pathologists

and epidemiologists would consider 4% to nearly 15% significant.

Why all subjects in this study were Caucasian is open to speculation. In an earlier study of 500 bony lesions,²⁷ 2.4% of the cohort were African-American, but none were diagnosed with FOMD. There may be racial differences, but as of yet, these differences have not been reported in the literature specifically for FOMD.

Gender differences were quite notable. Of the 122 tender areas and/or areas that were identified as pain generators, 106 (88.9%) were female. These differences were statistically significant and although unexplainable, conform to other studies published in the literature.³⁵⁻³⁷

The radiographic appearance of FOMD is quite variable, but the lesions are generally visible radiographically. In fact, some authors have suggested that these lesions are often discovered after routine radiographic examination.^{17,21,27} FOMD radiographically varies from sharply defined radiolucencies with distinct borders (**Figure 3**) to borders that are quite ill-defined and irregular. Further, these subtle lesions are considered to be variations of normal by some,¹⁷ but as demonstrated in this study, 100% were not normal.

In February 2002, bone sonography, when used in conjunction with a panoramic radiograph, was cleared by the US Food and Drug Administration for the express purpose of evaluating the wetness (i.e., blood perfusion) and

density of the medullary bone of the jaws. Health Canada, Canada's counterpart to the US Food and Drug Administration, also approved the Cavitat (Cavitat Medical Technologies, Inc., Aurora, CO) in May 2002 for the same express purpose. The Cavitat instrument uses a through-transmission technology rather than the reflected signals of other ultrasound instruments used in medicine. Normally hydrated bone and water conduct sound waves in a similar fashion. Sound waves traveling through regions of diminished bone hydration, reduced density or intramedullary cavitations (i.e., an air space void of hydration) are reduced in intensity, thus registering on the receiver with less intensity than waves traveling through normal bone.¹² Sound impulses are converted into digitized graphs, generated as 2-dimensional or 3-dimensional images (**Figure 1**).

This unique instrument generates 27,000 sound pulses per microsecond. Teeth have the same characteristics as bone; therefore, unlike on radiographs, the teeth and bones are not visible as separate structures. Each 3-dimensional color coded image represents a 1:1 cm² area exposed to the ultrasonic signal. Up to 32 individual exposures can be made of the alveolar bone—eight in each quadrant, each representing a specific tooth site.

Each 3-dimensional image shows 64 colored columns or data points, each corresponding to 1/64th of the intraoral receiver. Good sound transmission shows as green, with attenuated transmissions appearing in the following order: yellow, brown, orange and red.¹²

Cavitat scans are graded on a 5-point scale, Grade 0 represents normal (green from scanned alveolar area); Grade 4 represents the most desiccated or dry areas (i.e., some stage of ischemia) and is seen as red.¹²

Most FOMD lesions have been reported as occurring in the posterior mandible of middle-aged females,^{4,5,7,10,15,19,25} and the present data agree with this. In addition, most studies report, and the present study substantiates, that most sites of FOMD are former extraction sites.

Differential diagnoses of FOMD, based upon radiographic findings, are traumatic bone cysts,^{4,38} residual dental infections,⁴ idiopathic bone cavities,³⁸ central giant cell granuloma, incomplete healing of extraction sites,³⁹ alveolar osteitis,⁴⁰ neoplasm of the jaw, or metastatic carcinoma.⁴¹⁻⁴³

Since FOMD cannot be diagnosed with certainty based upon radiographic and clinical findings, surgical exploration and histopathological examination of the tissue from suspected lesions are indicated.^{1,3,6,17,21,25,27,37-44}

Conclusions

The following conclusions were derived from this study:

1. Focal osteoporotic marrow defect is perhaps the earliest detectable form of bone marrow edema;
2. Contrary to former reports, FOMD is not generally asymptomatic;

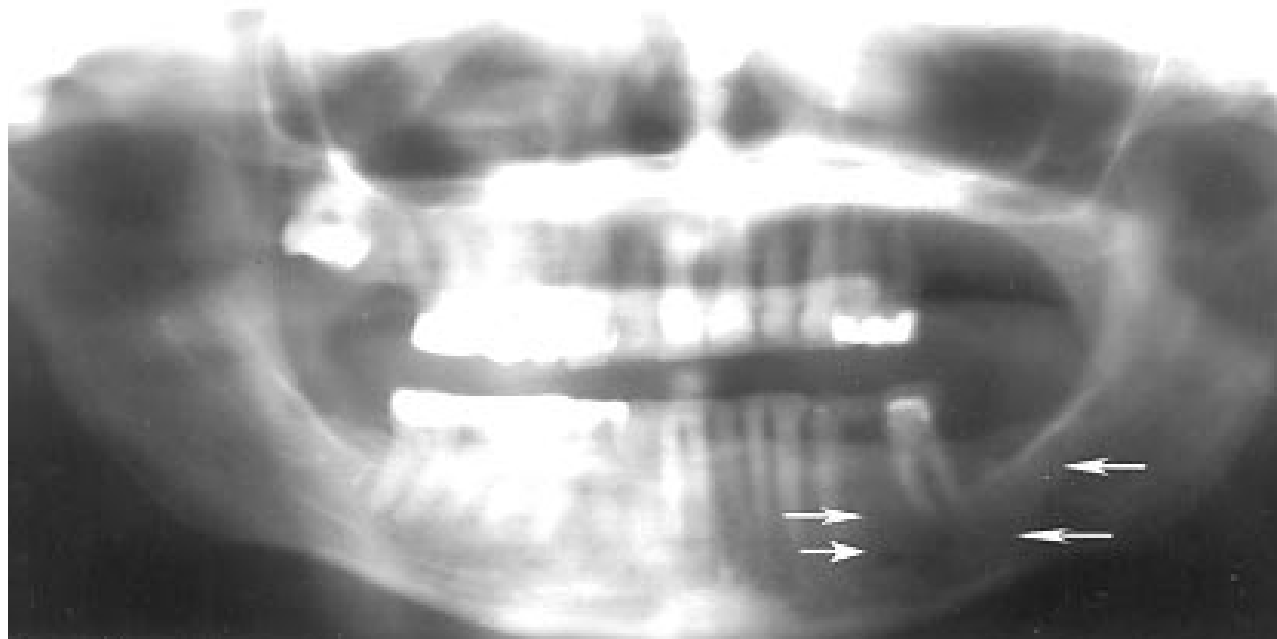


Figure 3
FOMD also may exhibit borders that are quite ill-defined and irregular (outlined by white arrows). Note that the localized loss of bone density is very subtle.

3. FOMD may be another common source of orofacial pain;
4. Through-transmission alveolar ultrasonography, when used in conjunction with panoramic radiography, is an accurate method of evaluating the condition of bone marrow of the jaws;
5. Because FOMD cannot be diagnosed with certainty based upon imaging and clinical findings, surgical exploration and histopathological examination of the tissue from the suspected lesions are indicated.

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Medullary and Odontogenic Disease in the Painful Jaw: Clinicopathologic Review of 500 Consecutive Lesions

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ABSTRACT: Ischemic jawbone lesions were first discussed in the dental literature more than a century ago, but then seemingly forgotten. In recent years, there has been considerable resurgence in interest in this unique pathological condition. Controversy surrounds the subject. Some proclaim these lesions to be mere fabrications of the imaginations of non-traditional or alternative dental surgeons. Others attribute all human maladies to these maxillofacial lesions. Aside from these philosophical and metaphysical arguments, are there common diagnoses of jawbone pathologies that produce pain? This present investigation reviews the clinicopathologic features of 500 consecutive jawbone surgeries with pathological confirmation in patients with *idiopathic* facial pain. Four hundred seventy-six (476) of the 500 lesions (95.2%) were directly attributed to impaired blood flow in the jawbone, tooth, or both, according to histopathological analysis and confirming Cavitat (bone ultrasound) examination. Statistical data concerning the location, frequency, and pathological diagnoses of these bony lesions are presented, as are brief methods of diagnosis, and treatment is also discussed.

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Few issues in dentistry, and specifically, oral surgery, have produced more controversy in recent years than the idea that inflammatory and ischemic lesions within the jawbones, which require surgical removal, are a common cause of facial pain. These bony disorders, when accompanied with orofacial pain, are more common than originally thought and have been termed neuralgia-inducing cavitation osteonecrosis (NICO).¹ Fortunately or unfortunately, depending upon how one surveys this subject, the general public has become aware and even educated about these lesions. Patients are well read, armed with refereed, nonrefereed, anecdotal, and Internet literature. In this, as well as in so many other health-related topics, the public seems to be forcing changes in traditional medicine, and dentistry is not excluded from those changes.

Every dentist, regardless of his or her specialty, has been rigorously educated in the diagnosis of acute, pus-associated osteomyelitis of the jaws. Most oral pathology textbooks state that true progressive osteomyelitis, due to odontogenic and other microbial infections, is not usually found in developed countries. However, osteonecrosis, a fairly common disease of medullary bone, is only casually mentioned in dental pathology textbooks.² This type of osteonecrosis is medullary bone damage and death from poor or interrupted blood flow within the bone marrow.

Ischemic marrow disease in bones other than the mandible and maxilla are not only common, they are widely researched and reported. Avascular necrosis (a form of ischemic osteonecrosis) develops most often in the femur and is seldom associated with infection. Other bones are also affected by both osteomyelitis³⁻⁹ and osteonecrosis.¹⁰⁻¹⁶ In fact, any and all medullary bones can be affected by both of these disorders. Why not the mandible and maxilla? Physicians readily agree that such lesions can and do occur in the jaws. So, why is there such controversy over this subject in dentistry?

The controversy is especially confusing from the historical perspective. Osteomyelitis, osteonecrosis, and similar medullary diseases are not new to the scientific literature concerning the jaws. In 1915 G.V. Black¹⁷ created an entire section in his classic textbook about alveolar osteitis, as he termed it. For some reason, Black's alveolar osteitis was forgotten or overlooked by the dental profession until the late 1970s when several reports were published describing an unusual chronic nonsuppurative osteomyelitis of the jaws in patients suffering with facial pain.^{2,18-23,25-35,41,42}

In addition to Black's observations, more contemporary writers have discussed marrow tissue changes and found inflammatory or ischemic change in the great majority of biopsy samples.^{1,19-42}

The purpose of this paper is to present data derived from 500 consecutive jawbone surgeries in patients with idiopathic, chronic facial pain and to briefly discuss the clinical presentations as well as diagnostic and treatment procedures.

Materials and Methods

The patients in this study were obtained from an orofacial pain center. Patients suffering from the effects of carious lesions, periodontal disease, soft tissue lesions, and exposed pulpal tissue were not included in this study. Five hundred consecutive surgical patients, presenting with a history of undiagnosed orofacial pain, were analyzed for this study. All lesions prior to surgery were confirmed by panoramic radiographic examination and since early in 2001, bone ultrasound (Cavitat 4000, Cavitat Medical Technologies, Aurora, Colorado). Most lesions were symptomatic at the time of the initial examination; some produced transient painful symptoms; others were constant generators of jaw and orofacial pain. The location of painful lesions was further confirmed with diagnostic anesthetic blocking using 3% mepivacaine (with no vasoconstrictor) as to the specific alveolar site or dental arch.

Of the 500 surgical sites, 413 (82.6%) were in dentate

regions and 87 (17.4%) were located in edentulous areas, so specific alveolar sites could not be accurately assigned to these 87 specimens. However, these edentulous areas were located in areas that appeared to be either the third molar areas or the maxillary tuberosity regions. For consistency in this study, the same pathological laboratory examined all 500 specimens.

For discussion purposes, the term NICO will be used in this paper when referring to painful lesions caused by nonsuppurative, low-grade bone marrow infections and/or ischemic marrow disease. By definition, NICO cases are associated with facial pain,^{2-4,35,45,48} whereas not all medullary lesions are painful.

Results

In this patient cohort, 149 participants (29.8%) were male and 351 (70.2%) were female. Ages ranged from 27 to 87 years of age, with the mean average being 50.4 years of age (standard deviation = 11.24 years). Racially, 97.6% (488) were Caucasian and 2.4% were African-American (10 females and two males).

Two hundred seventy-one (54.2%) of the lesions were located in the maxilla; 229 (45.8%) were of mandibular origin (**Figure 1**). Two hundred sixty nine (53.8%) were on the right side of the mouth and 231 (46.2%) on the left side (**Figure 2**).

Lesion distribution per quadrant was as follows: 1. maxillary right, 126 (25.2%); 2. maxillary left, 145 (29%); 3. mandibular right, 143 (28.6%); and 4. mandibular left, 86 (17.2%) (**Figure 3**).

The most common specific sites for jawbone lesions (n=413) were the third molar regions, the maxillary being most frequent (72 or 14.4% of lesions). Collectively, all four third molar sites (n=114 of 413) comprised 22.8% of the specific alveolar site lesions. However, 71.8% (n=359) of all lesions were located in the molar, retromandibular and maxillary tuberosity regions.

The two most frequent pathological diagnoses were 1. chronic nonsuppurative osteomyelitis (n=167, or 33.4%) and 2. ischemic osteonecrosis (n=160, or 32%) (**Table 1**). A total of 14 separate diagnoses were discovered in this study. A high number of diagnoses (n=476, or 95.2%) can be directly attributed to conditions of bone marrow ischemia.

Discussion

The concept of jaw osteomyelitis and osteonecrosis is not novel to the recent American scientific literature. Dental surgeons in England,²⁴ Canada,²⁵ Germany,²⁶ Russia,²⁷ China,²⁸⁻³⁰ and Jordan,³¹ have also written and

Distribution of Lesions (n = 413)

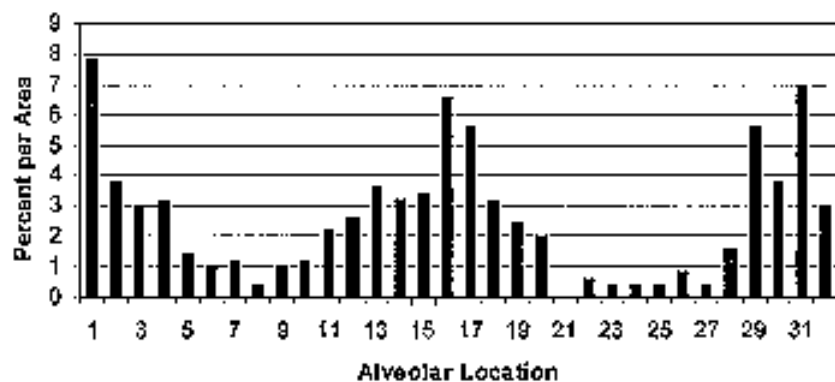


Figure 1

Anatomic distribution of lesions in specific alveolar sites. In this study, 87 lesions were located in nonspecific edentulous sites (see *Results* section).

Lesions per Side (n=500)

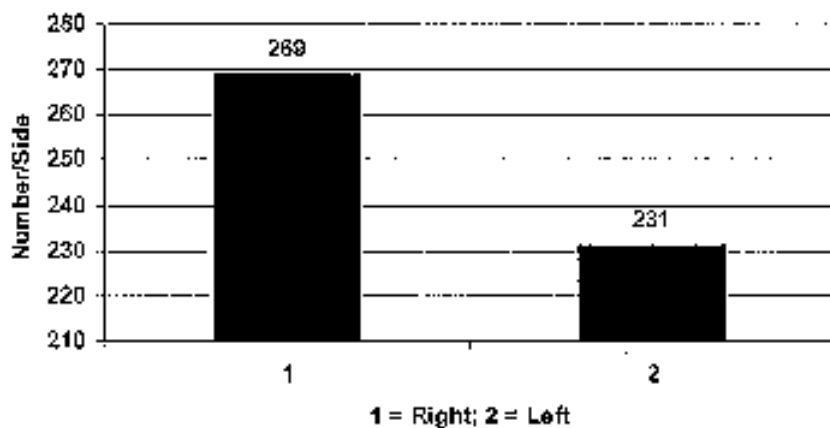


Figure 2

Anatomic distribution of lesions per side of mouth.

Distribution of Cavitations per Quadrant (n = 500)

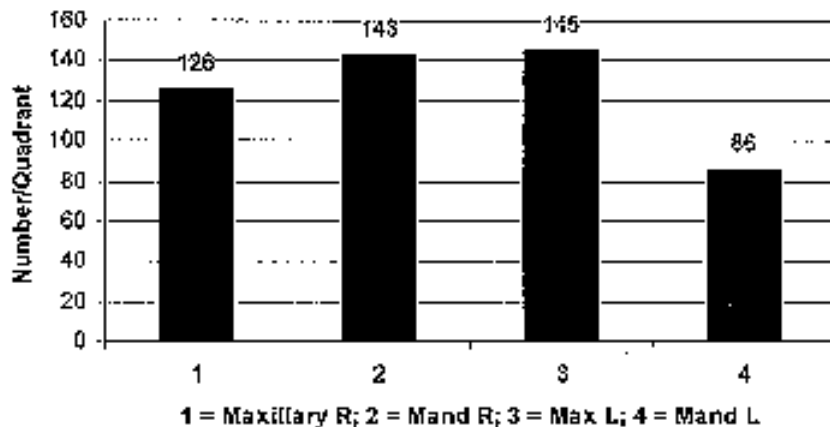


Figure 3

Distribution of lesions per quadrant.

Table 1
Relative Frequency of Specific Diagnoses
Ranked By Frequency

| Diagnosis | No. | % of 500 | ICD No. |
|--------------------------------------|------------|-------------|---------|
| Chronic nonsuppurative osteomyelitis | 167 | 33.4 | 526.4 |
| Ischemic osteonecrosis | 160 | 32.0 | 733.49 |
| Regional osteoporosis | 65 | 13.0 | 733.02 |
| Periapical granuloma | 35 | 7.0 | 522.6 |
| Periapical cyst | 20 | 4.0 | 522.8 |
| Focal osteoporotic marrow defect | 20 | 4.0 | 289.9 |
| Normal bone marrow | 12 | 2.4 | 526.9 |
| Foreign body | 9 | 1.8 | 528.9 |
| Residual cyst | 4 | 0.8 | 526.2 |
| Keratocyst | 3 | 0.6 | 526.0 |
| Periapical scar | 2 | 0.4 | 522.9 |
| Chronic sinusitis | 1 | 0.2 | 473.0 |
| Myxoma | 1 | 0.2 | 213.0 |
| Chronic mucositis | 1 | 0.2 | 528.0 |
| Totals | 500 | 100% | |

researched this subject extensively. Even Thoma, one of the 20th century's most respected American oral surgeons, discussed these disorders extensively in his classic, encyclopedic textbook.³²

In this study, the sexual ratios were consistent with studies by Byron³³ and Bouquot and McMahon.³⁴ The mean average age was similar to those of other studies as well.^{26,27,34,35}

As demonstrated by these data, there are many different types of jawbone lesions, most of which in our cohort produced pain symptoms. However, what is revealing is that the majority of our lesions were directly or indirectly attributed to impaired blood flow to localized areas of bone, according to the histopathologic diagnoses. In addition, many of the periapical lesions could also be attributed to a disruption in blood flow within the pulp of the tooth.³⁶ So, based on these numbers alone, approximately 476 of the 500 lesions (95.2%) can be directly attributed to impaired blood flow in the jawbone, tooth, or both, according to histopathological analysis.

In this study, disruption of blood flow was especially evidenced by the large number of lesions diagnosed as either osteomyelitis or osteonecrosis. Clinically, regional osteonecrosis secondary to vascular insufficiency, can be secondary to osteomyelitis or other factors (**Table 2**),^{37,38}

can be aseptic, and can provide a fruitful field for subsequent infection.

Acute osteomyelitis can arise directly from an endodontic infection.³⁶ Microbes exit the apical foramen, multiply, and spread through the marrow spaces, leading to bone necrosis, primarily due to vascular insufficiency.³⁹ Microbial invasion may also cause osteonecrosis of the jaws through recurrent maxillary sinusitis. Damage to the sinus walls and floor by inflammatory toxins and microbes infiltrating the alveolar bone can produce so much vascular insufficiency that osteomyelitis and/or osteonecrosis subsequently occur.

However, microbial invasion of the bone marrow is not the only known cause of osteonecrosis of the jaws. There are a myriad of other triggering events, trauma and infections being the primary ones. No other bones in the body are subjected to the level of trauma and infection experienced by the marrow of the jawbones from dental and periodontal infections, tooth extractions, periodontal and endodontic procedures and surgeries, and at times, trauma from occlusion.

In addition, many other causes of vascular marrow insufficiency have been reported (**Table 2**). This might explain why so many cases of NICO have been diagnosed in patients without extractions, without endodontic therapy, without trauma from occlusion, and without a history of maxillary sinusitis.

The concept of impaired blood flow causing these lesions should be especially shocking to those who practice dentistry. All students are taught to use local anesthetic solutions with vasoconstrictors for prolonged anesthesia and for hemostasis. Kim, et al.⁴⁰ reported that an injection such as 2% lidocaine with epinephrine 1:100,000 is capable of significantly restricting pulpal blood flow, decreasing oxygenation of the pulp. Could this possibly produce irreparable injury to pulp, bone marrow, or both? In the attempt to improve dental care, dentistry may itself be causing many jawbone lesions by impeding blood flow, thus creating an environment of ischemia stasis and possible thrombus formation.

The locations of lesions in this study were similar to those in larger studies by Byron³³ and Bouquot and McMahon³⁴ (**Figures 4 and 5**). Although the numbers are somewhat different, the trends of the graphs demonstrate a comparable frequency of alveolar site location of bony lesions. These data clearly demonstrate that the most common sites for bony lesions are: 1. the third molar/retromolar regions; and 2. the first molar areas in both arches.

Most NICO lesions have been present for a number of years, often totally asymptomatic until some precipitating factor (**Table 2**) begins the generation of severe and chronic, often undiagnosed pain.

Table 2
Etiologies Associated with Ischemic
Bone Disease^{47*}

Local factors

Trauma
Use of vasoconstrictors in local anesthetics
Radiation therapy
Intraosseous inflammation/infection of odontogenic origin
Failed endodontic therapy
Over-heating of bone during surgery
Corticosteroid injection of bone
Intramedullary hypertension
Trauma from occlusion

Systemic factors

Corticosteroid therapy
Atmospheric pressure changes in occupation
Sickle cell anemia
Pregnancy
Homocystinemia
Osteoporosis
Hypofibrinolysis
Antiphospholipid antibody syndrome
Hyperlipidemia
Chemotherapy
Blood coagulation disorders
Hormonal therapy
Systemic lupus erythematosus
Heavy smoking
Alcoholism

*Table modified and used with permission.

Osteonecrosis and osteomyelitis specifically pertaining to NICO lesions rarely produce clinical changes within the overlying gingival tissues, although some patients have reported episodes of edema and erythema. However, involved sites are typically tender and have an elevated mucosal temperature. Moreover, microscopic examination of the overlying oral mucosa routinely demonstrates mild to moderate subepithelial infiltrates of chronic inflammatory cells.⁴¹

NICO lesions are often not seen on panoramic radiographs inspected routinely. However, when the film is of good quality, studied with good lighting and even magnification, these apparent invisible lesions often become much more apparent (**Figure 6**). NICO lesions usually present as poorly demarcated radiolucencies, often with irregular vertical remnants of lamina dura (termed *laminar rain*) visible in old extraction sites

(**Table 3**). Contrary to what is taught, these irregular, ragged areas are not normal. They represent a poor attempt to heal the extraction site and extensions of ischemic or inflammatory changes beyond the socket. Under ischemic conditions, intraosseous fibrous scar tissue (marrow fibrosis, reticular fatty degeneration, ischemic myelofibrosis) is created in place of new, healthy bone because osteoclasts and osteoblasts require good nutrition and abundant oxygen, whereas fibroblasts do not.^{42,43}

Isolated extraction sites are not the only common locations for NICO lesions. In one study, more than 84% of subpontic bone demonstrate abnormal radiographic changes.⁴² Another common area of lesion formation is in and around the apex of endodontically treated teeth, many of which are totally asymptomatic.

Diagnosticians have difficulty localizing NICO lesions. This should not be surprising because 30-50% of the medullary bone must be destroyed before radiographic changes are visible,⁴⁴ and localization of alveolar pain is often problematic. Practitioners who have attempted to isolate a specific offending tooth in a painful dental arch will certainly understand and appreciate this fact.

To add to the diagnostic difficulties associated with radiographic interpretation, NICO lesions are also rarely seen with MRIs or CT scans. Also, radioisotope scans, often ordered by physicians have a high rate of false negative tests due to slow cell turnover, poor bone healing and poor circulation in bony areas containing these lesions and the presence of inflammation due to periodontal or periapical disease. Sadly, when the radiologist examines the films of a bone scan, a normal diagnosis is often given when anesthetic blockade and histopathological examination confirm the contrary.

A better diagnostic imaging technology is obviously needed for this marrow disease. In 2002, the Food and Drug Administration approved a new through-transmission ultrasonic device termed the Cavitat 4000. Using this device, osteoporotic, dry or hollow medullary bone of either the mandible or maxilla attenuates the transmitted sound, resulting in a weakened signal when it hits the receptor on the opposite side of the alveolus. The ultrasonic images (one per tooth site) allow the doctor to see the size, extent and position of ischemia areas in the bone in both two and three dimensions (**Figure 7**). This FDA approved device tests for low bone density and bone desiccation, both features of chronic ischemic bone disease. This valuable information, in conjunction with the patient's history, the patient's panoramic radiograph, and diagnostic anesthetic confirmation (if necessary), has added much more precision and accuracy to the localization of ischemic and inflammatory bone lesions.

Location of Maxillary Osteocavitation Lesions

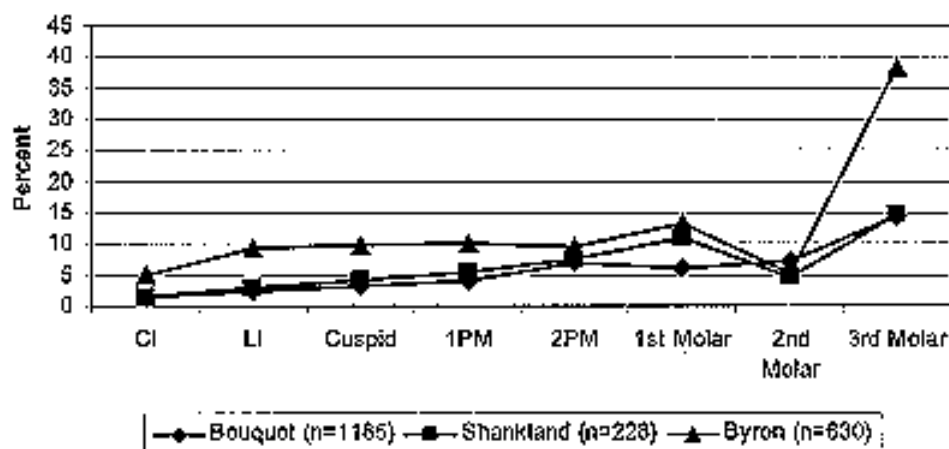


Figure 4
Location of maxillary medullary lesions in three separate studies.

Anesthetic blocking must be used to isolate painful suspected areas. NICO lesions exhibit deep aching bone pain, often producing referred pain, which is frequently misdiagnosed as trigeminal neuralgia or atypical facial neuralgia.^{20-23,26,28,34,35,45,48} Such anesthetic confirmation should be conducted without vasoconstrictor (e.g., 3% mepivacaine), for reasons discussed above.

The term *cavity* has seemingly been coined by alternative medicine advocates, but actually, the term cavity was first used to describe, in general terms, the effects of blood flow problems instead of infection in bones in the *Journal of Bone Surgery* in 1930⁴⁶ and Black¹⁷ for alveolar bony lesions. Where the term originated does not matter since cavity or cavitation accurately describes what is seen at the time of surgery.

As with all suspicious lesions, biopsy is required in order to make an accurate histological diagnosis. Although controversial in some circles, surgical exploration is recommended in order to rule out more ominous lesions.^{19-30,33,34,41,45,47-50}

At surgery, well over 75% of NICO lesions (in this author's experience) are completely hollow or filled with a soft, grayish-brown and mushy tissue. Characteristically seen is an oily material that oozes or rushes from the marrow spaces, displaying yellowish spheres of liquid fat (termed *oil cysts*) from fatty necrosis of the bone marrow.

After total surgical curettage of the cavitational areas, smaller satellite lesions are often found around the original lesion, often connected by interconnecting fistulae, which have been termed *worm holes* and are probably

Location of Mandibular Osteocavitation Lesions

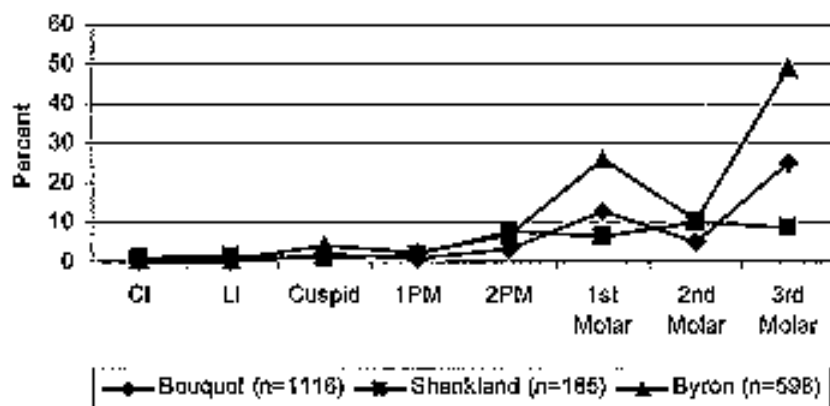


Figure 5
Location of mandibular medullary lesions in three separate studies.

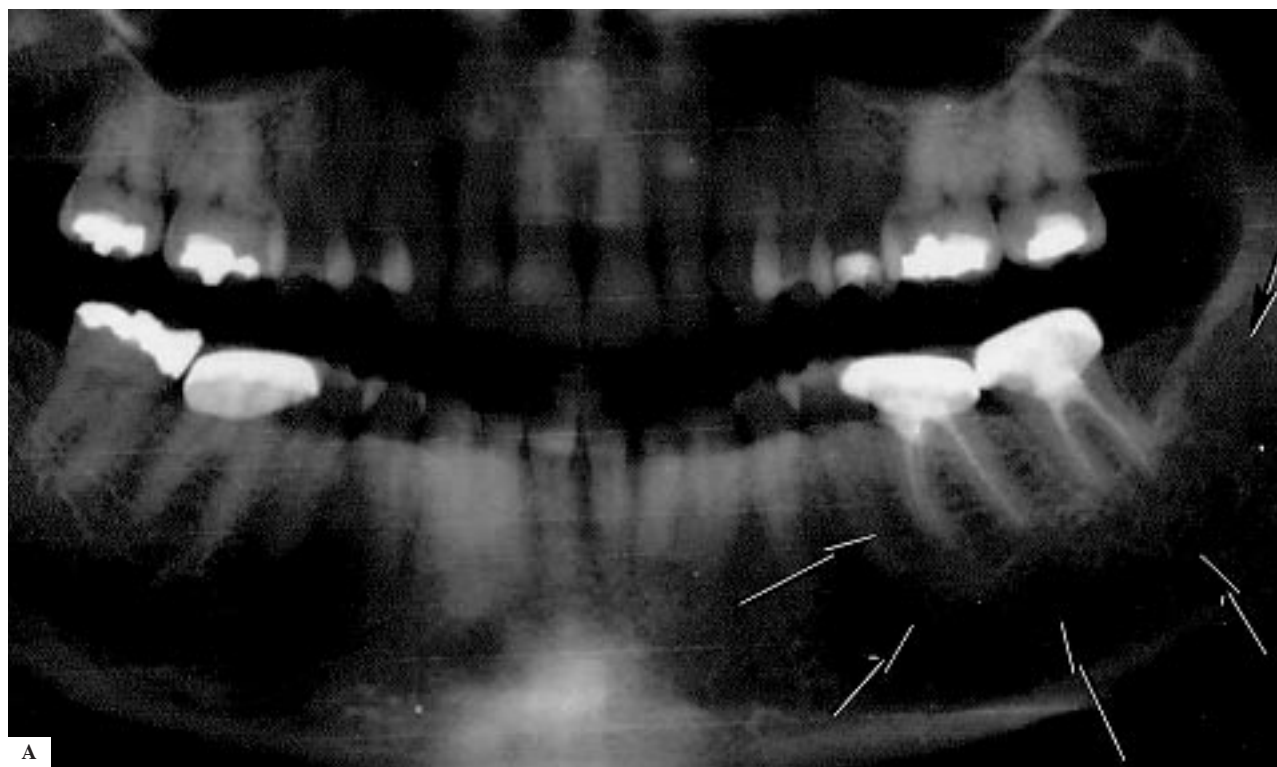


Figure 6
Identification of mandibular lesion using a panoramic radiograph and Cavitat scans. **A (above):** Panoramic radiograph; **B (below):** Lesion outlined (with arrows).

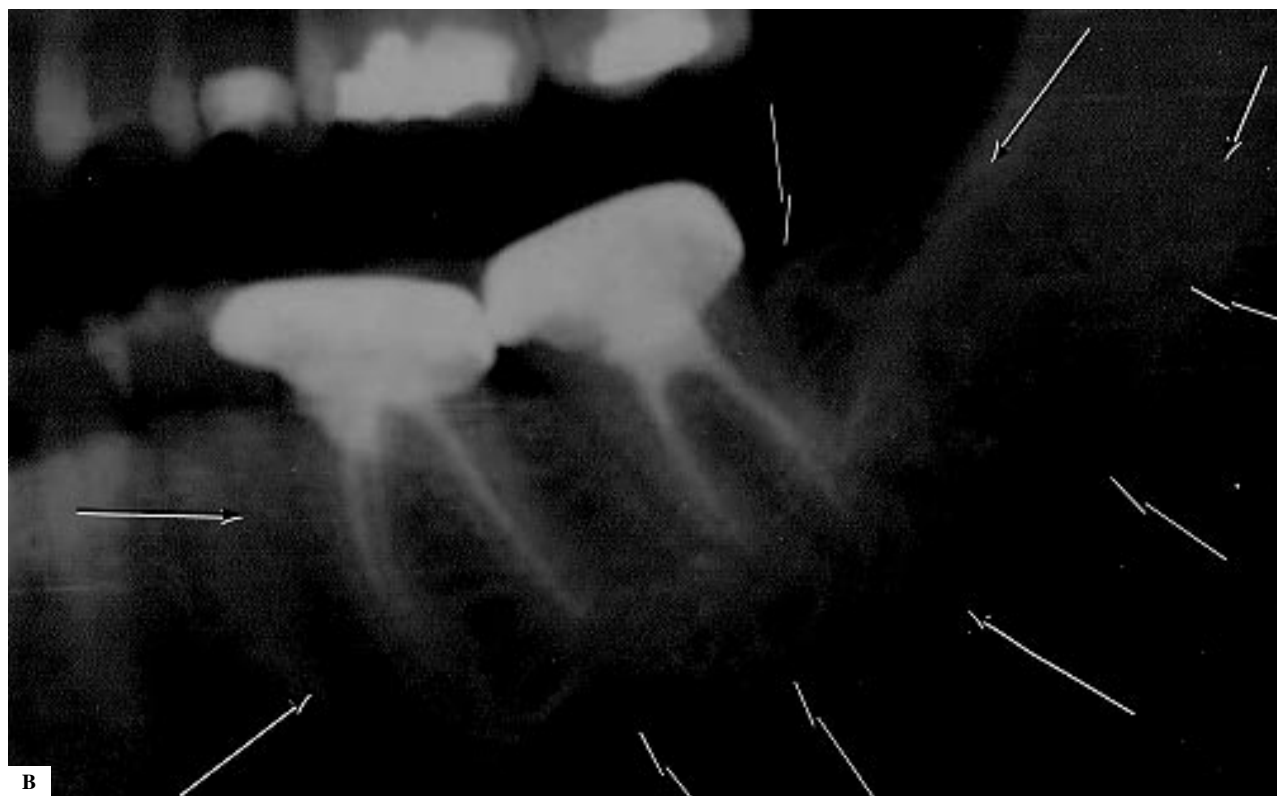


Table 3

Radiographic Features, Listed in Order of Frequency, Demonstrated in NICO Lesions^{47*}

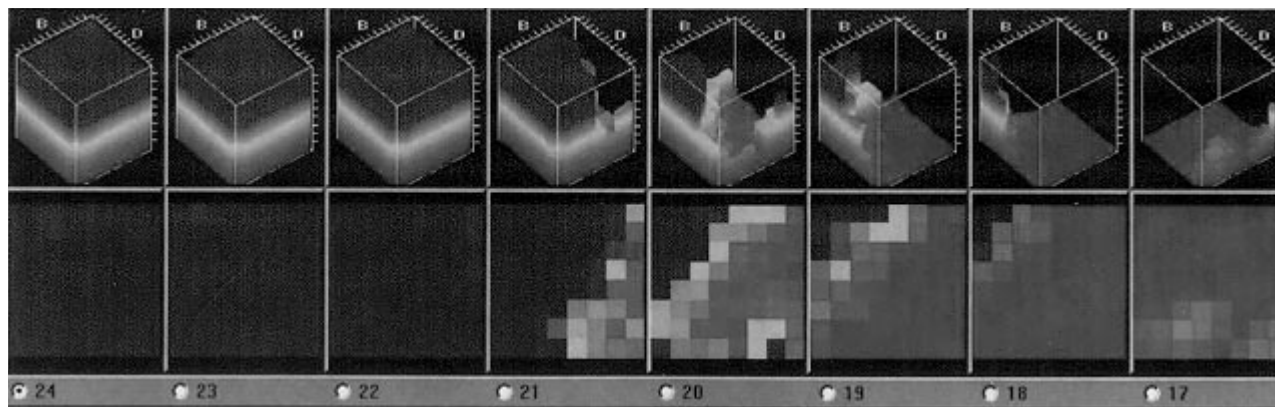
1. Poorly-demarcated radiolucency
2. Moth-eaten radiolucency (regional osteoporosis)
3. Irregular vertical trabeculae in edentulous area (*laminar rain* or *laminar lightening*)
4. Mild ground-glass radiopacity (*ghost marrow*)
5. Radiopaque flecks and streaks radiating outward from a central, poorly defined radiolucency (*eagle's nest*)
6. Focal destruction of bony wall of inferior alveolar canal
7. Soap bubble radiolucency
8. Horizontal trabeculae in edentulous areas
9. Focal destruction of wall of maxillary sinus
10. Focal destruction of cortical alveolar bone
11. Radiopaque flecks
12. Cotton-wool radiopacities

*Modified and used with permission.

Summary

In this study, 500 consecutive surgical specimens of painful jawbone lesions were biopsied. An overwhelming number of diagnoses (95.2%) were directly related to conditions of ischemia of the bone marrow. Various causes of ischemia have been presented. While reviewing patients' health histories, it is important to identify factors which may contribute to or cause ischemic conditions as listed in **Table 2**. In addition, patients presenting with undiagnosed orofacial pain need to be thoroughly evaluated for such jawbone lesions. This includes the use of anesthetic blockade, careful examination of panoramic radiographs, and the use of Cavitat technology. The latter two procedures are now considered by the FDA, when used together, to be the standard protocol when investigating these types of medullary lesions.

Lastly, to accurately establish a diagnosis, excisional biopsy should be considered as the treatment of choice of painful jaw bone lesions.

**Figure 7**

Cavitat scan of left mandible. This is the same left mandible shown in **Figure 6 A & B**. The areas of red indicate dry or hollow medullary bone. This continuous area corresponds to the lesion on the radiograph. The top row is a three-dimensional representation of each alveolar site; the bottom row is a two-dimensional representation of the same areas.

remnants of thrombosed vascular channels. These areas, too, must be curetted.

If a bony lesion is discovered in the process of diagnosis, the appropriate treatment of painful lesions is surgical intervention in order to establish a definite diagnosis, especially when the outline of lesion on the radiograph is not well demarcated.⁴⁹⁻⁵³

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Routine Dental Extractions Routinely Produce Cavitations

**Thomas E. Levy, MD, FACC, and
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ABSTRACT: Cavitations (CVs) are persistent holes found at the extraction sites of permanent teeth after apparent healing has taken place. Current dental literature considers this common phenomenon to be rare. In the scientific literature, CVs have a plethora of synonyms. They have been variably labeled as Ratner, Roberts, or trigger point bone cavities, interference fields, neuralgia-inducing cavitational osteonecrosis (NICO), and alveolar cavitational osteopathosis. Evidence suggests that the incidence of CVs is presently grossly underestimated. Therefore, we reviewed the charts of 112 randomly chosen patients treated at the Huggins Diagnostic Center (HDC) from 1991 through 1995 to determine the incidence of CVs in old extraction sites. We believe this problem to be important to the general health of patients who are being treated for a wide range of diseases where such a dental condition may be the ultimate cause or a significant contributing factor.

This manuscript is clearly one written for dental surgeons. It describes, however, a little known phenomenon that is extremely important in the general ill health of many patients with a number of different diseases that are not usually associated with dental problems. Since there is more and more need for health professionals to collaborate in their disciplines, the work reported here should be valuable to physicians.

—*Editor*

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Introduction

Black (1), an influential force in the earlier days of dentistry; described the pathology of CVs in 1915. Bone necrosis, resulting in persistently hollowed-out areas at the sites of old extractions, was described as typical of the lesion. The bone was usually softened initially by the progressive cellular death of cancellous bone, until an actual hole resulted. These holes often contained small particles of necrotic bone and other non-viable cellular debris. Black termed the disease process "chronic osteitis," although he was puzzled that extensive bone destruction could occur in the jawbone without overlying erythema or edema and without affecting systemic body temperature. The absence of such factors contradicted the concept of active inflammation, which typically produces both local and remote symptomatology. He even went on to describe the appropriate way to treat such lesions, which was essentially a surgical debridement. He indicated that it was easy to break through the relatively thin cap of bone at the top of the old extraction site, followed by removal of all particles of softened bone until margins of solid bone were once again reached. The gross morphology of such CVs can be most impressive (Figure 1).

Black's findings were either ignored, forgotten, or not given proper credence. Certainly, they were never assimilated into dentistry and dental teaching. In the 1970s, however, CVs were "re-discovered" and correlations with previously termed idiopathic pain syndromes were suggested. Patients with atypical facial neuralgia and trigeminal neuralgia of unknown cause were often found to have CVs at the sites of previous extractions, and the pathology was as Black had already described. Such cavities would be subjected to curettage and, upon complete healing with new bone, the pain syndromes would frequently resolve (2,3).

Materials and Methods

Periodontal Ligament Removal Procedure After the Extraction of Any Permanent Tooth and Cavitation Removal After Old Healing

Routine Extraction: After a tooth was removed, a #10 long-shanked, surgical round burr was used to remove one millimeter of bone as thoroughly as possible from the entire bony socket area, excluding the apex. This was done by the tactile sensations of the dental operator, usually involving the transition of going from a mushy or spongy feel to the firm resistance of uninvolved

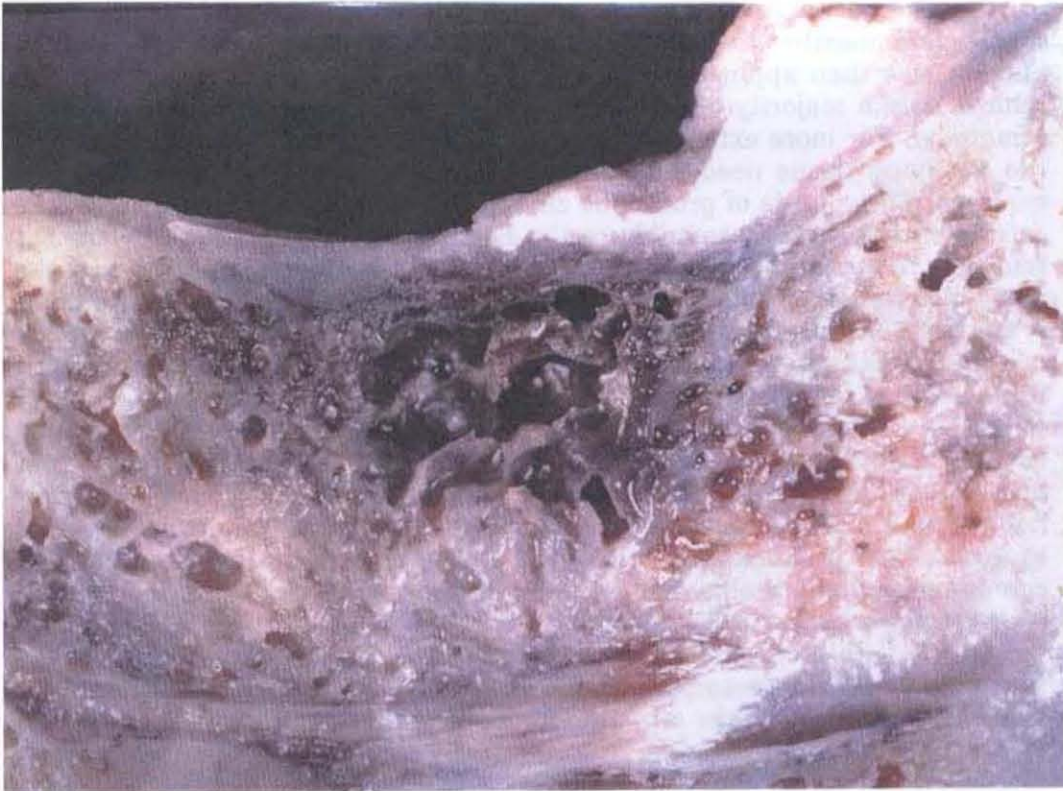


FIGURE 1

Autopsy mandible specimen sectioned longitudinally, revealing the gross morphology of cavitational osteonecrosis. The brownish discoloration is indicative of combined old hemorrhage and old necrotic fat. Photograph courtesy of J. E. Bouquot, DDS, MS.

bone. Taking one millimeter of good bone insures removal of the periodontal ligament. The remainder of the protocol is as for primary cavitation revision and is described below.

Cavitation Cleaning: A low speed handpiece was utilized with the #10 burr after an incision in the gingiva, if required. Usually only a few millimeters of necrotic bone were penetrated to reach the healthy tissue. During the drilling with the burr, the socket was irrigated with sterile saline, using a Monojet 412, 12 cc syringe. In some cases, several syringes of this type were used. The flushing removed all contaminated bone fragments as the bone was cut, so as not to allow any bone fragments to become trapped during the healing process. This also required diligent suction to avoid the patient risking swallowing of any contaminated material.

After the socket was properly routed, it was flushed with a non-vasoconstrictive anesthetic which was allowed to remain for about thirty seconds. Suction was then applied gently to the area for just an instant in order to remove only a majority of the anesthetic, leaving the socket coated with the remainder. For more extensive areas of old extraction, full thickness flaps of the overlying tissue needed to be reflected away from the bony area to be explored. Three units of protamine zinc insulin were injected adjacent to the extraction site, greatly to enhance the healing process. Antibiotics were not routinely recommended. Clinically, antibiotic administration appears to make its own contribution to a greater chance of a dry socket with an eventual CV in spite of performing an otherwise proper procedure. It is theorized that the use of antibiotics may convert the osteoblasts back into osteocytes, impeding a full healing of bone in the socket area.

Taking at least one millimeter of good bone insures removal of both periodontal ligament and most of the bone directly bathed with the toxins produced by the mutant streptococcus in the dentin tubules. It is very important not to use a high speed handpiece. Using the low speed handpiece prevents excessive heat production, avoiding the undesirable cauterization effect that can denature the proteins present and impede complete healing. The cutting process serves to "perturb" the adjacent bone, allowing a more effective stimulation of osteoblast formation from monocytes, which results in the needed new bone growth necessary to fill the cavity. This bone perturbation also appears to be superior to the stimulus for healing that occurs with manual curettage. The anesthetic also serves to perturb the bone and stimulate greater osteoblastic activity.

Simple manual curettage is discouraged, for the scraping required in the process can "push" much of the toxic products into the adjacent, good, cancellous bone, resulting in a greater chance of persistent or recurrent CVs, or simply a lack of primary healing after a tooth extraction. Flu-like syndromes, persisting for days, have been observed after the cleaning of CVs. While this protocol is certainly not the only way to remove a periodontal ligament or clean out a CV, it has realized clinical success and minimized the formation of dry sockets or recurrent CVs.

Several CV procedures at HDC included the injection of a small amount of radio-opaque medium into the freshly opened CV sites. The lower third molar sites pictured were only opened and not debrided prior to the introduction of the contrast medium. Before and after X-rays provide additional evidence as to the difficulty in detecting CV on X-ray alone (Figure 2).

Following this, the contrast agent was promptly flushed out and suctioned, with the remainder of the protocol for CV treatment then being followed. It should also be noted that a circular, routing motion with the dental burr is never performed until the operator feels the burr drop into a pre-existing CV. The CVs are never created by the dental burr, but they may be slightly expanded initially in the course of debridement in order to ensure that good bone is reached which is capable of healing.

Over the past year, selected patients have undergone 24-hour urinary porphyrin testing at HDC. This has been initiated in the hopes of developing a clinical test that could be specific for different forms of chronic toxicity that induce subtle but distinct clinical syndromes. Porphyrrias, disorders in which



FIGURE 2A
Mandible Enhanced



FIGURE 2B
Mandible Unenhanced



FIGURE 2C
Mandible Enhanced



FIGURE 2D
Mandible Unenhanced

FIGURE 2

Radiographic visualizations of two mandibular cavitations in the same patient, using a standard radio-opaque contrast medium. Note the difficulty in precisely localizing these cavitations on the unenhanced X-rays. The patient had no clinical symptoms specifically referable to these cavitations. The mottled black area over the unenhanced mandibular X-ray of the smaller cavitation is a film artifact.

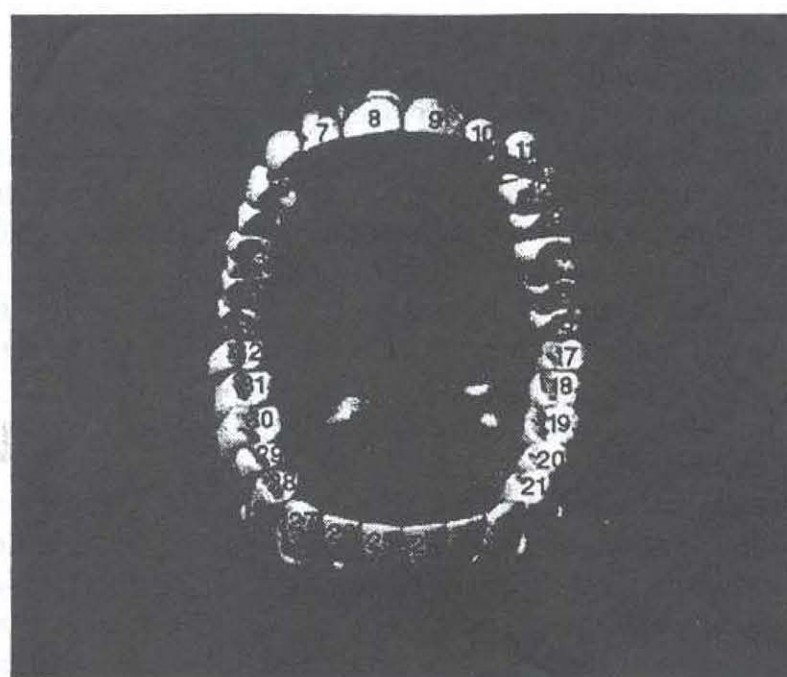
large amounts of urinary porphyrins are excreted, result from specific enzyme defects in the heme biosynthetic pathway. The porphyrin excreted is typically the substrate of the defective enzyme

Results

A review of the CV incidence on 112 randomly selected patients, who underwent total dental revisions at HDC from 1991 through 1995, was compiled. All old extraction sites were routinely explored for CVs, regardless of radiological appearance. Practical considerations, such as noting little residual space between adjacent teeth or anticipating a likelihood of sinus penetration due to atypical closeness to the extraction site, may have precluded exploration. Cavitations were routinely found at more than 75% of all extraction sites. When not found, the line of drill exploration healed very rapidly and did not present a clinical problem. An analysis of 112 randomly selected patient charts was done, with patient age ranging from 19 to 83 years among 40 males and 72 females.

The most commonly extracted teeth, the third molars ("wisdom teeth"), produced CVs that were found by clinical exploration in 313 out of 354 extraction sites (88%). Cavitations were found in 35 of 50 second molar extraction sites (70%), and for first molars, 60 of 73 extraction sites showed CVs (82%). They were found in 441 of the total number of 517 molar extraction sites explored (85%). For the maxillary non-molars, CVs were found in 72 of 123 extraction sites (58%), and for mandibular non-molars, 23 of 51 extraction sites were affected (45%). For all non-molars, the CV rate was 55%, representing 95 of 174 extraction sites. The overall CV rate, regardless of site, was 77% (536 out of 691 extraction sites) (Figure 3). Smaller CVs could have been missed, making these percentages conservative.

It should be emphasized that this incidence of CVs probably underrepresents the true figures. A number of practical considerations allow this conclusion. Since CVs are usually radiologically invisible, the exploratory drilling by the operator is a blind procedure and a greater success rate will be achieved with increased experience. An incorrect angle of attack can miss a large CV and small CVs can be missed even by an experienced operator. They can range in size from minuscule to greater than a cubic centimeter. Additionally, adjacent extraction sites will sometimes form an area CV, a small version of the channel CV described below, and it may result in being counted only as one if it is not completely clear that it arises from both sites. Fi-



R Cavitation Incidence L
By Tooth Number

[No. of cavitations/No. of extractions]

| | | | | | |
|-----|-------|-----|-----|-------|-----|
| 1. | 79/91 | 87% | 17. | 75/86 | 87% |
| 2. | 2/10 | 20% | 18. | 14/17 | 82% |
| 3. | 9/14 | 64% | 19. | 20/22 | 91% |
| 4. | 14/16 | 88% | 20. | 8/10 | 80% |
| 5. | 7/18 | 39% | 21. | 5/10 | 50% |
| 6. | 3/6 | 50% | 22. | 0/2 | 0% |
| 7. | 8/16 | 50% | 23. | 0/1 | 0% |
| 8. | 6/9 | 67% | 24. | 0/2 | 0% |
| 9. | 4/8 | 50% | 25. | 0/1 | 0% |
| 10. | 5/11 | 45% | 26. | 2/3 | 67% |
| 11. | 4/7 | 57% | 27. | 1/3 | 33% |
| 12. | 11/20 | 55% | 28. | 1/9 | 11% |
| 13. | 10/12 | 83% | 29. | 6/10 | 60% |
| 14. | 9/11 | 82% | 30. | 22/26 | 85% |
| 15. | 12/15 | 80% | 31. | 7/8 | 88% |
| 16. | 85/95 | 89% | 32. | 74/82 | 90% |

FIGURE 3

Incidence of cavitation formation by tooth number upon exploration at old extraction sites.

nally, the most obvious reason for not finding a CV is not looking for it and such a consideration is included in the data as "CVs not found." Small anterior tooth extraction sites, as noted earlier, may simply not be explored if the remaining spaces between surviving teeth appear adequately minimized. Similarly, when the first bicuspid had been removed to make room for orthodontic reconfiguration of teeth alignment with braces, enough adjacent tooth migration usually occurred such that the areas for potential CV were obliterated or severely downsized. It has to be a practical determination of the dental operator whether to pursue empirical exploration for tiny CVs that might persist in these anterior extraction sites, especially when braces follow the extractions.

Additional situations, although rare, in which CVs occur that may merit consideration for exploratory debridement, include undeveloped tooth sites and sites of early incomplete, extra tooth development. Also, patients may have had rare supernumerary molars removed in addition to their third molars, and these sites can also cavitate. In one patient who denied ever having her third molars removed, CVs were found in all 4 sites where the teeth were shown to be absent radiologically. While a given patient can forget having had extraction procedures, it appears that the vestigial remnants of undeveloped teeth can also cavitate. They may also be found at potential supernumerary molar sites, perhaps representing the vestigial remnants of those teeth, in the absence of any history of extraction of such teeth.

Clinical appearances of the detritus found in these holes at HDC varied greatly. Common colors were green, yellow-green, and even a dark, tarry appearance. Consistencies ranged from a thick "cottage cheese" type to a loose, runny type. It would occasionally even be serious and clear in appearance. Sometimes putrid odors, usually noticed much more readily by the patient than the dental operator, would also be noted. Bouquot (4) described additional lesion morphologies, which included accumulations characterized as blood soaked sawdust, chocolate ice cream, spongy bone, gritty powdered dust, and even green fatty globules. In patients who either were edentulous or just had large extents of missing teeth, it was typical that the CVs would interconnect and this was termed "channel cavitation." These channel CVs could be quite sizable, sometimes enough to accommodate a small pencil. Although no other references to such channel CV was found in the literature, they can be expected to be found in most edentulous patients. Bouquot had also noted that individual CVs were capable of burrowing several centimeters from the primary

extraction site and this would be consistent with the development of channel CVs when all teeth have been extracted without removal of the periodontal ligaments.

When enough chronically infected teeth have been removed in the course of preparing for permanent full dentures, clinical improvement may be anticipated in a wide variety of conditions. When, however, the CVs that result outweigh the degree of toxicity removed, clinical decline can be noted at the 3 to 4 month post extraction point, when the jawbone appears to have healed. This illusion of healing, however, masks the multitude of non-healing bony CVs now present, harboring the previously friendly aerobic mouth flora in an anaerobic environment. Although edentulous patients generally consider themselves to be spared any further such infectious toxicity, the toxicity has only become less apparent, cloaked in the guise of cavitational osteonecrosis.

Porphyrin Testing

In only one patient, two different types of urinary porphyrin were found in a specimen obtained immediately before cleaning out two maxillary CVs at the third molar sites. Uroporphyrin was 134 micrograms/24 hours, and coproporphyrin was 232 micrograms/24 hours. The follow up specimen, obtained 2 weeks later, had no detectable concentration of either porphyrin. The detection limit is 1 microgram (Figure 4). No other dental work was performed at that time. Amalgam replacement with biocompatible composite material had already been accomplished 6 months earlier. While far from being conclusive on the basis of one patient, we believe that this is worth further research.

We conclude from this that CVs occur very commonly after teeth are extracted in the standard manner utilized in dentistry today. The pathological characteristics of these lesions may prove to impact the health of many people severely, since most people have had their third molars removed. Edentulous patients might have the greatest risk of any potential toxicity.

Clinical Aspects

While a CV can result from any permanent tooth extraction, the molars appear to be the most frequent sites of these defects. The anterior teeth, including the first bicuspid removed for orthodontic reasons,

Pre vs Post Dental Urinary Porphyrins on a Patient with Cavitations Only.

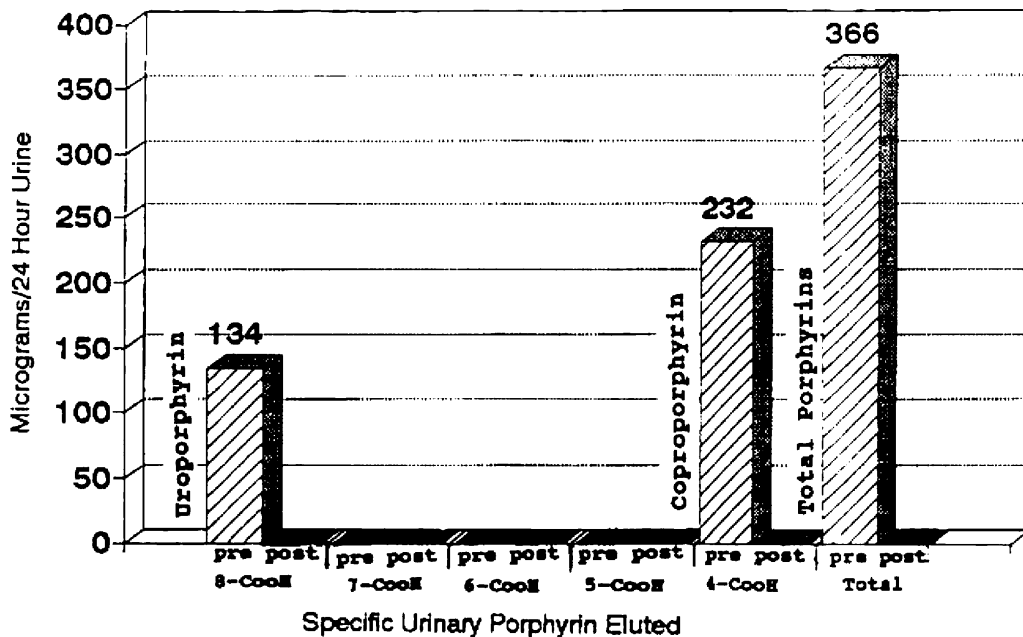


FIGURE 4

Urinary porphyrins detected before and after debridement of two cavitations in a patient. Note that substantial levels of coproporphyrin and uroporphyrin detected initially fall below measurable levels after cavitation debridement.

will cavitate less frequently. Braces exert a physical pressure that remodels bony structure, allowing the effective migration of teeth over small distances. This effectively obliterates any potential CV from a tooth extraction performed to make room for the eventual re-alignment of the teeth. When the first bicuspids are removed without the subsequent placement of braces for non-orthodontic reasons, their incidence of CV subsequently increases. Also, the smaller the tooth, the less likely CV will result, as noted with the anterior lower teeth extractions.

Routine dental extractions involve just the withdrawal of a tooth, intact or in pieces. As long as all of the bony tooth is removed, the extraction is considered to be complete. A most critical factor, how-

ever, in socket healing is not addressed by this standard approach. The periodontal ligament, which is the connective tissue attaching the tooth to the alveolar bone, is seldom removed as part of the extraction procedure. Such removal would typically be inadvertent, resulting when the ligament uncharacteristically preferred adherence to the tooth than to the surrounding bone. This ligament consists of collagenous bundles between which are loose connective tissue, blood vessels, lymph vessels and nerves (4). Also called the periodontal or periodontal membrane, its continued presence in the extraction site effectively prevents the adjacent bone from biologically recognizing that the tooth has been extracted. Bone cells are not going to proliferate spontaneously and migrate through a membrane intended by nature to define their growth limits. As long as the periodontal ligament remains intact, or largely intact, the underlying bone cells consider the tooth to be present, and no biological signal for bone growth is triggered. At the upper portion of the extraction site, however, where there is no periodontal ligament, osteoblastic bone activity does initiate, and a thin cortex of bone will heal across the hole. This cap of bone is rarely more than several millimeters thick.

As already indicated, bacteria are found in the CV and the obvious source is the oral cavity which is a teeming milieu of aerobic strains. Microbiological studies have indicated anaerobic flora to be present as well in the walls of the CVs (5). At the moment the healing cap of bone is complete over the extraction site, the CV has been officially formed and it can never be sterile. Instead, it now has aerobic bacteria trapped in an anaerobic environment. This entire situation is strongly analogous to *Clostridium botulinum*, an anaerobic but aerotolerant organism that characteristically produces extremely potent exotoxins when placed in a significantly oxygen deprived environment. Virtually harmless to man in the presence of oxygen, it becomes deadly when most of that oxygen is removed. Similarly, the mouth flora undergo metabolic transformations when oxygen deprived, and exotoxin production can be anticipated.

Discussion

Characteristics of Cavitations

A specific property of such bone cavities is that they are not readily discernible on radiographic examination, and when they are detectable by X-ray, the changes are typically very subtle. The CV, because

of this difficulty in its visualization, has even been branded as "invisible osteomyelitis." Most dental surgeons who are aware that a CV can occur at an old extraction site persist in thinking that its presence is ruled out when not apparent on X-ray. The vast majority of CVs will be missed by such a reliance, even some of the largest ones. The jawbone is already heavily trabeculated with many tiny holes due to the porous nature of healthy bone and a larger hole, regardless of its pathology, does not readily stand out. Additionally, the typical CV is not routinely visualized on magnetic resonance imaging (MRI), computed tomography (CT), or with radioisotope bone scans, except technetium-99 scans (6).

Although it has been likened to osteomyelitis, the pathology and natural history of CVs are really quite different from osteomyelitis. Strong similarities, with almost identical microscopic patterns, are seen in aseptic necrosis of bones elsewhere in the body. Pathologically, specimens from CV debridement typically show ischemic osteonecrosis. The CV appears to develop primarily as an avascular process rather than an infectious one. Indeed, bacteria are present in CVs, but they are not numerous enough to typify a primarily infectious process. It is possible that these bacteria may play a significant role in the overall toxicity of CVs by their production of toxic metabolic substances, resulting when previously aerobic strains from the oral cavity are permanently subjected to anaerobic conditions. Typical infection, that is, bacterial proliferation with large numbers of inflammatory cells, is definitely *not* seen. Neutrophils, the primary cells in abscesses or smaller pus-filled lesions, are conspicuous by their absence (6). Minimal numbers of streptococci, along with atypical, multinucleated monocytes with up to 4 nuclei, may be seen. The monocytes probably represent the body's limited ability to scavenge the breakdown products in the necrotic CV, but due to the avascular nature of the necrosis, such immune cells cannot access the core of the CV in any great number. Lymphocytes are also seen, but sparsely, with a relative absence of other inflammatory cells (7). Necrosis involves more than the mere death of cells or loss of their blood supply. It refers to those structural changes that occur in cells subsequent to their death while still within a living host. After coronary artery occlusion, necrotic changes will not be seen upon microscopic examination of heart tissue that had acutely lost its blood supply. Necrosis only occurs when the catabolic intra- and extracellular enzymes of the surviving host exert their effects on those tissues (8).

The standard, best, and really the only successful treatment for

necrotic tissue is debridement. A healed-over hole with necrotic debris in the jawbone, however, does not allow the body to "autoamputate" as it might with a gangrenous fingertip. When any gangrenous portion of the body cannot be spontaneously eliminated and surgical debridement is not performed, death or some form of chronic disease is the inevitable result because of the continual production, however slow, of anaerobic bacterial necrotoxins. One could avoid this slow toxicity if the gangrene was of the relatively rare, non-infected, dry variety which can result in a mummification of the affected tissue. A typically gangrenous limb that is not surgically separated from the body will usually result in death of the patient. More focal areas of gangrene, especially when sequestered in almost completely avascular pockets of bone, will not be expected to kill the organism, but they can contribute to a wide variety of clinical illnesses. Pathologically, CVs are focal pockets of gangrene in the jawbone, since gangrene is defined as necrosis due to obstruction of blood supply which may be localized or widespread, as in an entire extremity. These bony pockets invariably have some bacterial content, so a "dry gangrene" can never be expected to form. Any continued bacterial presence will result in ongoing production of bacterial metabolites and other waste products, even if they are minimal.

Potential Public Health Impact

It must be made clear that cavitation osteonecrosis is *not rare*. In fact, it is exceptionally common. Anyone who has had wisdom teeth removed can expect to have CVs present, even if the extractions took place decades earlier. While cases of neuralgia-inducing cavitation osteonecrosis are now well documented, it is not necessary that overt clinical disease of any kind be present for CVs to exist. Even current dental literature will refer to the existence of post-extraction CVs as being a rare event. Such statements are usually made because it is still not widely appreciated in dentistry that CVs are usually not detectable on X-ray. A CV, obvious on radiological examination of the jaw, is definitely the exception rather than the rule.

What, then, would be the significance for the average patient who underwent wisdom teeth removal? The data presented would indicate that a minimum of three CVs would be present, and many individuals would have four. The impact of only a few CVs on a given individual's health, by themselves, still needs to be determined. Clinical and labo-

ratory changes in many of the patients at HDC have indicated that CVs clearly play a strong contributory role in negatively impacting the immune system, along with any other dental toxicity present. Alone, in an otherwise pristine mouth, they are still separately their own negative influence on the immune system. Other negative forces working against the immune system, such as the heavy metals used in dentistry, may often appear to be the primary reasons for an immune system collapse or compromise. But like a felled boxer, the immune system only recovers optimally when totally unburdened. Except when present in massive numbers, CVs tend more to impede immune system recovery than to be a primary cause of its collapse. This, then, makes the effect of CVs on the public health especially pernicious and insidious, as many people simply accept the onset of different chronic diseases in middle age as being inevitable. Were CVs routinely revised, immune system function could rebound much more effectively from any of the many insults inflicted upon it, and the onset of truly chronic degenerative disease might be postponed for years. Of course, using the proper extraction technique and avoiding the formation of CVs would be optimal.

It was empirically observed several years ago at HDC, before CV exploration and cleaning was a routine part of the total dental revision, that most patients showed some clear clinical and laboratory improvements after their dental treatments. Many of the neurologically diseased patients such as those with multiple sclerosis, Alzheimer's disease, amyotrophic lateral sclerosis (ALS) and Parkinson's disease, showed further dramatic leaps in their clinical status when CV revision was added to the treatment protocol. In particular, both Parkinson's disease and ALS patients had been uniformly unresponsive to dental treatment that included amalgam removal and root canal filled tooth extraction. When CV revision was routinely undertaken, a great majority of such patients showed clearly discernible improvement clinically and/or in laboratory testing. Sometimes this would take the form of a less rapid rate of disease progression, especially with ALS, which is an "improvement" that most ALS patients would gladly accept.

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SECOND EDITION



ELSEVIER
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Cranial Manipulation Theory and Practice

OSSEOUS AND
SOFT TISSUE APPROACHES

Leon Chaitow

Foreword by
John E Upledger

With contributions by
Zachary Comeaux
John M McPartland
John D Laughlin III
with John D Laughlin IV
Frank O Pederick
Evelyn Skinner



Chapter 11

Cranial therapy and dentistry

John D Laughlin III with John D Laughlin IV

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DENTISTRY AND CRANIAL THERAPY: THE LINKS

Dentists, along with most doctors, are taught to analyze body systems and body parts separately. We are taught to change one area at a time, but such approaches frequently ignore the interconnectedness of the systems in the human body. If it is accepted that normal cranial motion and structure are necessary for the optimal functioning of the individual (Page 2003, Stockton 1998, Zeines 2000), it should be possible to acknowledge that dental procedures can potentially have debilitating, possibly long-term effects on a person's health, when those procedures interfere with the optimal functioning of the cranial complex (Fischer 1940, Hodgson & Hansen 2000, Morgan et al 1982, Simon 2001).

Similarly, cranial treatment may be less effective if inappropriate dental procedures produce changes that interfere with normal function (Frymann 1998).

In contrast, it is suggested that dental therapy that considers the whole body can result in major benefits, especially when integrated with suitable cranial therapies.

BACKGROUND

The cranium is a compact container with many structurally and functionally interrelating parts and tissues. Dysfunction of a single part can affect

the entire interrelated system (Upledger 1997). Because of this, an integrated 'whole-body' dental approach needs to take into account more than just the achievement of 'straight' teeth (Gelb 1971, 1977, Upledger 1987).

Dental education, for the most part, fails to take into consideration areas of the body beyond dentition, the status of the maxillae and mandible and their occlusion (Breiner 1999, Zeines 2000). Similarly, many practitioners who treat temporomandibular dysfunction symptoms (TMJ dysfunction or TMD) do not look beyond the interrelationships of the maxillae, mandible and TMJ (Hruby 1985, Page 2003). Issues surrounding hard tissue correction seem to be given more weight than other cranial interrelationship issues (Simon 2001). It is not surprising therefore that much of the discussion that follows is neither understood, nor accepted, by mainstream dentistry (Breiner 1999, Carter 1993, Zeines 2000).

In this chapter we define the goal of *whole-person dentistry* as:

- healthy tooth structure
- optimal occlusion
- mandibular/maxillary relationship, with correct structural relationship between the maxilla, the sphenoid and all other cranial bones (Breiner 1999, Gelb 1971).

DENTAL TREATMENT CAN ENHANCE OR INHIBIT CRANIAL TREATMENT

FUNCTIONAL JAW ORTHOPEDIC ORTHODONTICS (also known as functional jaw orthopedics or FJO)

Functional jaw orthopedic orthodontics: 'The use of orthopedic orthodontic appliances to influence the teeth and bone in such a way as to stimulate remodeling or alteration of growth patterns of the jawbones and associated neuromuscular tissues' (Zeines 2000). A longer definition would be:

The use of tooth and tissue anchored appliances, designed to create change during function, toward the eventual goal of cranial symmetry through orthopedic movement and soft tissue balance, while at the same time emphasizing correct TMJ

mechanics, cranial suture, cranial bone and sacral motion. (Hockel 1983, Hruby 1985, Wiebrecht 1966, 1969)

FJO can have positive effects in transforming a person's life. The author has regularly, in clinical practice, observed marked positive physical, mental and emotional changes, as the face, skull and body are reorganized following appropriate dental care (Magoun 1979, Page 2003, Stack 2004, Stockton 1999).

Depending on the individual's belief system, these beneficial changes might be ascribed as deriving from changes in CSF movement, cranial sutural mobility and/or membranous and facial stress reduction (Gelb 1977).

Some clinically documented examples of these transformations include: increased self-esteem, marked improvements in school grades, enhanced ease of learning, reduced ADD or ADHD symptoms, improved social skills, ease of breathing, bedwetting elimination, desire to change abusive relationships and increased energy.

FJO can make cranial treatment more efficient and effective and have longer lasting benefits by encouraging the correction of underlying structural problems (Hockel 1983).

FJO analysis and treatment from a dental relationship point of view

Class I Dental relationships

This refers to a fairly normal relationship of upper to lower teeth. However, this classification does not address the health of the TMJ nor the possible malposition of the maxillae relative to the cranial base.

For example, a patient may present having had several teeth extracted, a severe TMJ dysfunction, as well as cranial and esthetic disturbances and despite these problems may still have a classification of a Class I occlusion, merely because the teeth fit together well.

Figure 11.1A represents a post-treatment case with a normal face form and Figure 11.1B demonstrates an intraoral view of a normal overbite (vertical overlap) and overjet (horizontal overlap of the upper jaw compared to the lower jaw). In this Class I case the TMJ, tooth alignment and jaw relationship are ideal.

Box 11.1 Understanding what another health professional may be saying: different classifications (systems) for the same problem

In order to be able to establish good communication with a dentist it is vital that there is an understanding of the background of his/her diagnosis. There are five main types of classifications used to describe the interrelationship of the teeth, jaws and skull (Bowbeer 2003, Gelb 1977).

- I Dental
- II Functional
- III Esthetic
- IV Radiographic
- V Cranial.

Each can be used to describe the same case in a different way, which means that to communicate it is necessary to establish a common language. A broad overview of these classifications follows.

Dental relationships Classes I–III relate to how the upper and lower teeth fit together. This classification utilizes the first molars and the cuspids (eye teeth) as reference points to describe and diagnose a case. This classification is also known as describing occlusion or the interdigitation of the upper and lower teeth. It does not however describe or diagnose the health or function of the TMJ (Enlow 1975, Spahl & Witzig 1991).

Functional relationships The health of the TMJ and neuromuscular system can be identified by grading the degree of joint degeneration from 1 to 4. Neuromuscular dysfunction can be graded according to the degree of over- or undercontraction through the use of electromyography.

Esthetic relationships This classification is a diagnosis of how the face appears from the front and side.

The facial balance involves one-third upper, one-third middle and one-third lower face dimensions. This classification can overlap with the dental classification in that how the teeth fit together can also be related to how the face appears from the profile view. For example:

- Class I demonstrates a normal/ideal profile
- Class II Division I: the lower jaw is recessive (posteriorly positioned)
- Class II Division II: has a bite in which the dentition of the maxilla hangs too far over the dentition of the lower – an overclosed vertical or deep bite
- Class III: the lower jaw is too far forward and generally the upper jaw is deficient anteriorly (Celic & Jerolimov 2002, Enlow 1975).

Radiographic relationships The most commonly used radiographic analysis involves the use of cephalometric X-rays (lateral head and neck), transcranial X-rays of the TMJ, CT or MRI of the TMJ/skull and panoramic X-ray of the lower half of the skull.

Cranial relationships There are a number of classifications which seek to explain malformations and/or malalignments of the cranial structure. A professional's ability to classify such characteristics would depend on training and background, for example in cranial osteopathy or craniosacral therapy, Howell's neurocranial restructuring, orthobionomy, sacro-occipital technique (SOT) and chirodantics. Each area of study has its own classification methods and philosophy, sometimes making it difficult to hold a meaningful cross-educational-background discussion.

Class II Division I dental relationships

Division I refers to the occasions when the upper teeth are abnormally in front of the lower teeth, commonly known as 'buck teeth'. This condition involves having a recessive lower jaw, with or without crowded teeth. This is often related to headaches, ear problems (otitis media), TMJ clicking and a narrow cranial structure (extension pattern) (Morgan et al 1982, Price 1945).

Figure 11.2A represents the facial profile of a classic Class II Division I malocclusion. Figure

11.2B shows the same malocclusion from an intra-oral view. Note the horizontal protrusion of the upper teeth (overjet). The reality is that the lower jaw is extremely retruded (recessive). In cases such as this it would be a mistake for a dentist/orthodontist to extract upper bicuspids in order to move the upper teeth backward to match the lower jaw (Carlson 2004).

Appropriate dental treatment of this problem involves widening the upper jaw with a flexible appliance, such as the Advanced Lightwire



A



B

Figure 11.1 **A** A post-treatment case with a normal face form. **B** An intraoral view of a normal overbite (vertical overlap) and overjet (horizontal overlap of the upper jaw compared to the lower jaw). In this Class I case the TMJ, tooth alignment and jaw relationship are ideal.

Functional appliance (ALF – Fig. 11.3), combined with cranial treatment to balance the mechanism. Both methods, dental and cranial, can be utilized to encourage forward repositioning of the mandible. A twin block, or Bionator, is used to further advance



A



B

Figure 11.2 **A** The facial profile of a classic Class II Division I malocclusion. **B** The same malocclusion from an intraoral view. Note the horizontal protrusion of the upper teeth (overjet). The reality is that the lower jaw is extremely retruded (recessive). In cases such as this it would be a mistake for a dentist/orthodontist to extract upper bicusps in order to move the upper teeth backward to match the lower jaw.



Figure 11.3 Advanced Lightwire Functional appliance.

the lower jaw and decompress the TMJ. Treatment completion may possibly involve use of fixed orthodontic appliances to bring the back teeth together, providing the TMJ with better support through proper occlusion (Nordstrom 2003, Spahl & Witzig 1991).

Class II Division II Dental relationships

This is the diagnosis that represents a deep bite where the upper teeth are both forward of the lower and severely overlap them vertically. The result is an outward facial appearance of a large lower lip that in turn causes a cleft between the lip and chin.

This condition is classified by Jecman (1998) and others as a sphenobasilar symphysis (SBS) lesion, in which the SBS junction is in a 'hyper-flexed' position (invaginated superiorly). This position tips the posterior aspect of the maxillae up and posteriorly, causing the anterior maxillae (the premaxilla) to rotate inferiorly so that the tips of the front teeth incline posteriorly (see Fig. 11.4A,B). The result of this condition is that the mandible is trapped in a posterior position, with the temporal bones in external rotation (Magoun 1976).

Dental treatment First, the premaxilla must be released into a more anterior position. The teeth are tilted so that the tips are not retroinclined. In other words, a type of buck tooth position is created (with the upper front teeth ahead of the lower front teeth) before the mandible and temporal bones can change position. Appliances such as the Twinblock,

Box 11.2 Controlled arch development and non-surgical mandibular advancement

Cranial orthopedics is a term that helps to explain how the lower jaw is able to move forward in a patient who has a posteriorly displaced mandible. The mandible cannot make drastic changes in position without involving simultaneous changes in the position, function and mobility of the temporal and other cranial bones (Baker 1971, Jecmen 1995, Magoun 1979, Morgan et al 1982).

A wide variety of appliance designs have been used for many years to advance the mandible. These include the Andresson activator, Frankel Witzig orthopedic corrector, Mew orthotropics, Sved, Katsev's K-Flex, Clark's Twinblock, Nordstrom's ALF and others. All these appliances are dependent on affecting major cranial changes to accomplish their goals of mandibular advancement. Most dentists using these appliances do not fully realize the dental-cranial connection and therefore do not understand the positive impact a cranial therapist can have on assisting their progress (Zeines 2000). For example, in not adequately preparing for maxillary development, both anteriorly and laterally, the forward mandibular movement will be considerably impeded, because the maxilla is the matrix for mandibular position (Gelb 1994). If birth, general trauma or improper nutrition has restricted the normal maxillary growth and position, then the temporals, and thus the mandible, will be negatively affected (Gelb 1977, Nordstrom 2003, Price 1945).

The advantages of this form of treatment are the achievement of better face form, wider maxillae and zygomatic processes (allowing the cranial mechanism greater freedom of movement) and better forward positioning of the lower jaw (Diamond 1979a). Mechanically, this treatment also provides improved TMJ function, enhanced drainage of the sinuses and Eustachian tubes, as well as improved inner ear function, cranial motion and amplitude (Hockel 1983).

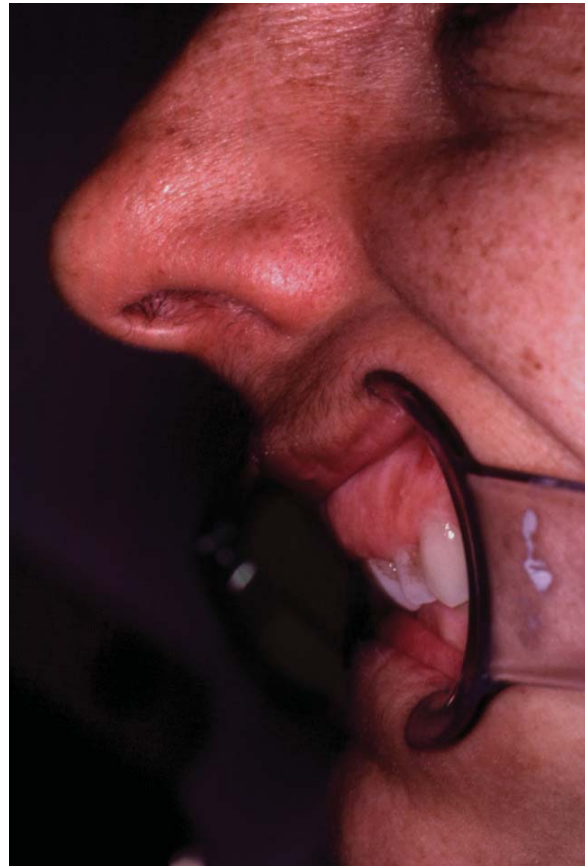
ALF twin block, Bionator, etc. are then used to further correct the mandibular position (Gelb 1977, 1994).

Results obtained

- Correction of the SBS restriction
- Improved position of the externally rotated temporals



A



B

Figure 11.4 A,B The anterior maxillae (the premaxilla) are rotated inferiorly so that the tips of the front teeth incline posteriorly.

- Reduced overbite and overjet, improving face length and esthetics
- Lessened stress of the TMJ complex
- Positive changes in positioning of the neck, back and neuromuscular system (Jecmen 1998).

Any technique which mobilizes the craniosacral mechanism can assist this transformation (Smith 2000b).

Class III Dental relationships

In this diagnosis the lower jaw is seen to be in front of the upper jaw (commonly known as an underbite) – see Figures 11.5A and C. A cranial description might include a maxilla that is positioned posteriorly (recessive/pushed back), laterally constricted and anteriorly underdeveloped. Internal rotation of the temporal bones is also often seen in a Class III diagnosis (Magoun 1976).

The author provided dental treatment utilizing upper and lower ALF appliances with elastics

hooked from the upper posterior to the lower anterior teeth. The elastics can encourage anterior development of the maxillae and provide a general widening effect of the upper arch (Nordstrom 2003). As shown in Figures 11.5B and 5D, the results obtained by such therapies can be excellent.

Low tongue posture (almost always present) should be addressed with special tongue retraining (myofunctional therapy) (Gelb 1977, 1994). The treatment generally includes stimulating the maxillae to become wider and positioned anteriorly. This creates more room for the tongue in the roof of the mouth.

The temporals can be further balanced with cranial therapy and improved vertical development (this adds to TMJ support) which is encouraged with the use of elastics (Spahl & Witzig 1991).

Improved TMJ function and improved esthetics of facial features can be achieved, usually without surgical intervention. When treatment is started before age 15–16 (the younger the better; 4–6 years

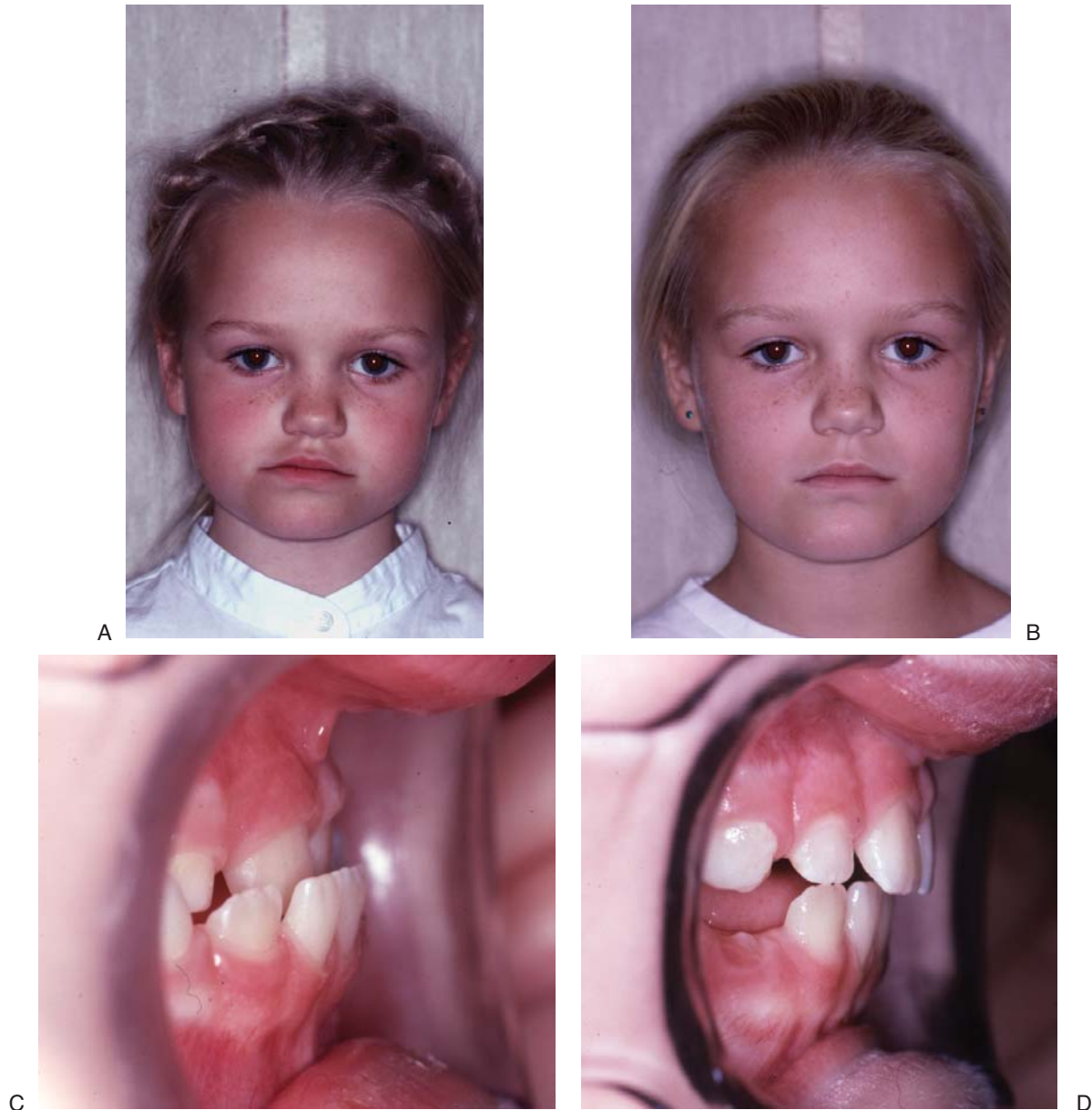


Figure 11.5 A–D Class III dental relationship. The lower jaw is in front of the upper jaw (commonly known as an underbite).

is best) the possibility of resorting to surgical or extraction therapy is greatly reduced (Page 2003, Simon 2001).

Functional jaw orthopedic FJO analysis and treatment from a cranial relationship point of view

Many varied cranial conditions are described in the literature, including sideflexions, lateral strains, vertical strains and torsions. Such dysfunctional patterns are commonly present in combinations

(Nordstrom 2003). In order to demonstrate how dental orthopedics can assist in the correction of these patterns, a sidebend/vertical strain combination has been chosen.

A sidebend (sideflexion) dysfunction pattern is a cranial classification of an imbalanced cranial form. In this classification the face, when viewed from the front, appears to have one side which is wider and compressed vertically (involving external rotation of the temporal bone) with the other side appearing narrower and longer (internal rotation of the temporal bone).

Figures 11.5A and 5C show an example of this dysfunction; the right side of this patient can be seen to be compressed and the lower jaw shifted to the right.

It has been reported by numerous experts that ear problems can often be found on the internally rotated side (Frymann 1998, Fushima et al 1999, Magoun 1976). Headaches (sometimes severe) are commonly also seen in these cases, as is unilateral chewing on the internal temporal side, which creates further cranial imbalance. Tinnitus is possible in later life and its symptoms are generally located on the side of the internally rotated temporal (Magoun 1976).

Dental treatment

The upper and lower ALF appliance is used, with elastics positioned on the upper outside of the externally rotated (wide) side, down to the lower inside on the same side.

This action helps to hold the wide side by 'putting the brakes on' the maxilla, with elastics on the ALF or Crozat appliance. This causes the opposite side of the maxilla to receive lateral stimulation from the expanding appliance transfixed to it.

The internally rotated side therefore balances out and the mandible is then encouraged to correct its position by moving and rotating toward the internally rotated side (Jecmen 1995).

Cranial therapy helps to facilitate the change through mobilization of the internally rotated side, as well as normalizing functional behavior of the sphenoid, occiput and temporals (Smith 1992, Upledger 1987).

The author suggests that when changes are only made dentally (as described above), without accompanying cranial support, the rest of the cranosacral system can be left in a state of sub-clinical or clinical distress.

In the case of the patient seen in Figure 11.5, an immediate vertical dimension restoration was accomplished by building up the vertical support over the lower back teeth. Use of an ALF appliance widened the maxilla and elastics helped develop the maxilla anteriorly. A K-wire was anchored to the upper cuspids (eyeteeth) to further encourage widening of the maxillary arch (Katsev 2003).

By using functional jaw orthopedics to correct this type of cranial lesion, combinations of the following benefits have been reported (Diamond 1979a, Page 2003).

- Restoration of normal facial symmetry (Fig. 11.5B,D)
- Bilateral chewing
- Normalized cranial motion and CSF flow
- Improved TMJ function
- Improvement or elimination of a variety of other symptoms (physical, mental, emotional).

Examples of a sidebend pattern of dysfunction may be present in a Class I, II or III malocclusion. All lesion and strain patterns may be present in combinations, often overlaying one another. It is for this reason that cookbook, analytical and reductionist methods of diagnosis, although necessary, may lead to incomplete and limited success (Cathie 1952, Zeines 2000).

STRUCTURAL/FUNCTIONAL ASPECTS OF THE CRANIAL SYSTEM

Implications of vertical dimension

Within dentistry, vertical dimension refers to the distance between the alveolar process of the mandible and the maxilla; in other words the height of the bones and teeth from the nose to the chin. If a person has all their teeth removed, with no dentures in place, their nose would nearly touch their chin. Missing one or more back teeth or wearing dentures with inadequate vertical dimension will to a lesser degree have the effect of reducing vertical support, but less dramatically (see Fig. 11.6A).

Unless dental orthopedics, fixed or removable dentures, crowns or bridges are included in the treatment plan, reduced vertical dimension can cause disruption of neuromusculoskeletal balance. This will often result in dysfunctions that are resistant to cranial therapy.

Symptoms of vertical dimension inadequacy

Symptoms that are seen in such cases may include inner ear problems involving hearing loss, tinnitus and infections; trigeminal neuralgia; bone/tooth

pain; sleep apnea; severe headaches and sinus infection (which may lead to tooth death and bone infections) (Fischer 1940, Morgan et al 1982).

It has been suggested (Morgan et al 1982, SOTO 2001) that when there is inadequate support for the TMJ, patients may exhibit irresolvable, unresponsive, structurally related TMJ pain, jugular foramen impingement with vagal nerve compression, equilibrium problems (Ménière's syndrome, Costen's syndrome), temporal artery compression and compression of occipitomastoid suture and nerves.

Results of a restored vertical dimension

When a patient's vertical dimension is restored (either temporarily with appliances or more permanently with prosthetics or by means of FJO) the TMJ is restored to proper form and function, thus reducing TMJ-related pain and condylar and/or disk displacement (Gelb 1994).

Restoration of the vertical dimension can also alleviate many underlying problems that would otherwise inhibit the effectiveness of cranial treatment.

Take as an example yawning, which involves translation of the mandibular condyles to the eminence of the glenoid fossae and a maximum opening of at least 42 mm. Structural corrections allow for this motion of yawning (as well as chewing) to improve (Jecmen 1998, Magoun 1976).

Once TMJ form and function are restored, the muscles and ligaments of the area (such as stylomandibular, stylohyoid, stylomastoid, internal pterygoid, tensor veli palatini, tensor tympani) are likely to assume normal length and tension. This reduces impingement on the vessels (lymphatic, blood and cerebrospinal), muscles and nerves in the region. An example is the vagus glossopharyngeal, its accessory nerves and internal jugular vein as they pass near and through the jugular foramen, just medial and posterior to the TMJ complex (Feeley 1988, Magoun 1976).

Once vertical dimension is restored and the condyle–fossa relationship is balanced, the temporal bones appear to experience greater freedom to resume normal internal and external rotation. Equilibrium difficulties such as Ménière's and Costen's syndromes have been reported to

be related to an excessive internal rotation of the temporal bone (Magoun 1976). Restoring the tensions of the medial TMJ region, the tensor veli palatini and the tensor tympani, accompanied by relaxation of the Eustachian tube, can contribute to greater ease in the manipulation of the temporo-occipital region, as well as to greater stability once correction is achieved (Magoun 1976, 1978, Morgan et al 1982).

Figure 11.6A demonstrates a loss of vertical support due to bone resorption under the dentures. The patient's dentures were 25 years old and needed temporary relining, followed by new upper and lower dentures. Once treatment was completed the patient's facial profile was improved (see Fig. 11.6B). Headaches were eliminated for this patient following the change, in conjunction with chiropractic care and cranial therapy.

Implications of sinus function as it relates to vertical dimension

Neuromuscular imbalance, brought about by the many structural effects of an inadequate vertical dimension, results in reduced fluid flow through the sinus cavities. The resultant congestion in the sinus cavities can inhibit the beneficial effects of cranial treatment. Restoration of improved vertical dimension to the mouth, restoring proper neuromuscular balance and function to the muscles of mastication, improves the flow of fluids through the sinus cavity and allows increased freedom of sutural motion, thus increasing the effectiveness of cranial therapy.

Inappropriate (shortened) muscle length, caused by an inadequate vertical dimension, reduces the effectiveness of the masticating muscles. Proper function of those muscles appears to be largely responsible for powering the action and drainage of the sinuses (SOTO 2001).

A suggested sequence might involve the following.

Proper function

- Correct occlusion in combination with efficient mastication causes external rotation of the maxillae during chewing and swallowing.
- This creates a pumping motion that aids fluid flow in the sinus cavities.

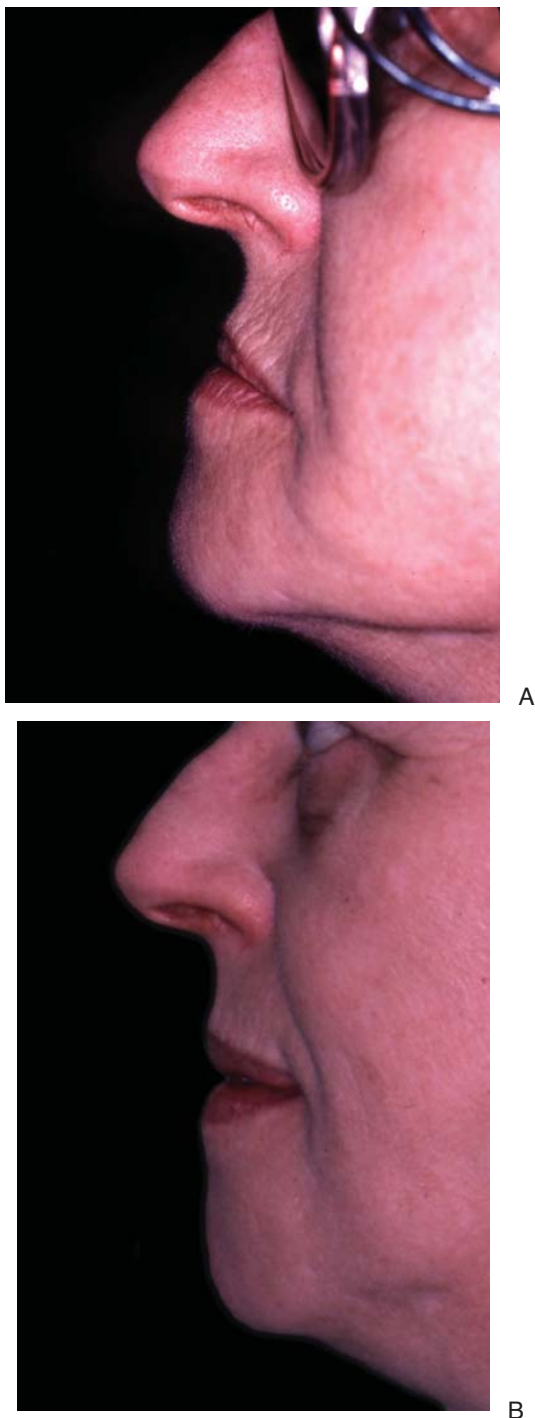


Figure 11.6 A Loss of vertical support due to bone resorption under the dentures. B The patient's facial profile was improved by the treatment.

- Decreased efficiency of these muscles results in diminished fluid flow through the sinuses.
- This reduced fluid flow may lead to increased pressures which inhibit the freedom of sutural movement, possibly limiting the effectiveness of cranial treatment.

Reduced motion of the maxillary/mandibular complex may produce a domino effect, resulting in the disruption of neuromuscular balance which in turn impacts on sinus function.

Improper function

- The resultant reduction in external rotation of the maxillae reduces function of the maxillary division of the trigeminal nerve.
- It also reduces stimuli to the cilia, resulting in less ciliary motion (Gelb 1977, Lundberg & Weitzberg 1999).
- Diminished ciliary motion moves the fluids through the sinuses less efficiently.

Normalization of bilateral chewing improves function of the maxillary sinuses, as well as other sinuses such as the nasal, frontal and sphenoidal (Fonder 1977, Gelb 1994, Page 2003, Upledger 1987).

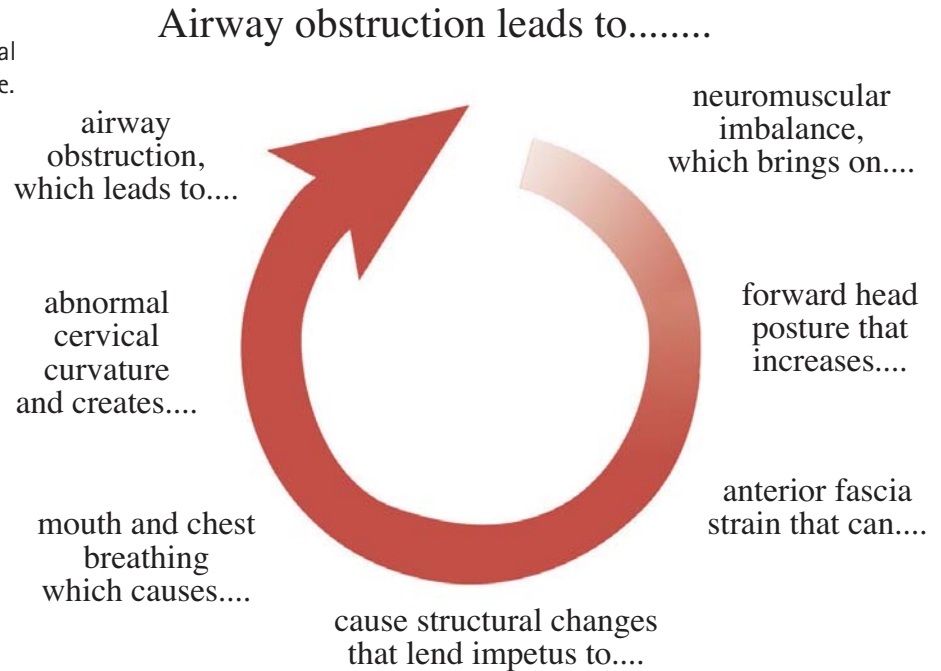
When correction of function is accomplished in a growing patient, reduced pressures allow the maxillary tuberosity to achieve greater growth, allowing for a larger, more efficient sinus cavity (Enlow 1975, Gelb 1994).

The importance of correct airway function

Normal nasopharyngeal airway function is essential to appropriate cranial growth, TMJ health and correct mandibular positioning. Restriction of the airway, anywhere between the nose and the alveoli of the lungs, can create a number of signs and symptoms (Fig. 11.7). Long-term dysfunction of the nasopharyngeal airway can, in some cases, result in postural changes, which can further inhibit correct cranial function (Gelb 1977, 1994).

A discussion of proper airway function, its effects and its presentation is not only appropriate when considering vertical support (as discussed above), but also in relation to a person's overall health. Poor dietary control (consuming foods that trigger allergic reactions), other allergies, cavitation-induced sinus infections (see later in this chapter), chronic or acute immune system disturbances (from any source) and many other causative factors can induce acute – possibly leading to chronic – nasopharyngeal airway dysfunction.

Figure 11.7 The acute/chronic nasopharyngeal airway dysfunction–postural correction–body adaptation cycle of change.



This dysfunction, as discussed below, can lead to serious inhibitions of cranial function (Page 2003, Rubin 2003, Stockton 1999).

Acute signs and symptoms of a dysfunctional nasopharyngeal airway (Diamond 1979b, Fonder 1990, Stockton 1999)

- Sore throat with infections
- Shoulder pain
- Middle and low back pain
- Sleep disorders
- Moodiness
- Allergies (both as cause and effect)
- Swallowing difficulties
- Ear problems
- Vertigo
- Reduced fluid flow to and from the head.

Chronic signs and symptoms of a dysfunctional nasopharyngeal airway (Char 1980, Gelb 1994, Hockel 1983, Rubin 2003)

- Increased decay of teeth and bone
- Adverse cervical curvature
- Facial deformity
- TMJ dysfunction
- ADD
- ADHD

- Bedwetting
- Crowded teeth.

Effects of functional nasal breathing

Nasal breathing may contribute to the ionization of cerebrospinal fluid through the olfactory bulb and the cribriform plate of the frontal bone (Chia & Chia 1993).

A side benefit to nasal breathing involves the relatively automatic superior and anterior positioning of the tongue. It has been suggested that this assists the flexion and extension of the sphenoid and occiput at the SBS. This may occur by way of lateral pressures at the maxillae, affecting the temporals (Gelb 1977).

Normal nasal breathing also stimulates nitric oxide (NO) production which is believed to play a vital role in regulation of blood flow (through endothelial relaxing factor – ERF), platelet function, immunity and neurotransmission. Nitric oxide seems to be produced in the paranasal sinuses, suggesting that the natural production of NO may be enhanced by improved functioning of the cranial/sinus system. If this hypothesis is correct it may explain why patients feel better when the maxillae are widened/developed and nasal breathing becomes easier (Lundberg & Weitzberg 1999).

TWO CAUSES OF DENTAL RELATED DISTRESS AND DISEASE

Cavitations: an underrated source of distress and disease in the human body

Definitions according to Dorland's illustrated medical dictionary (24th edn)

- Cavitations: the formation of caries.
- Caries: the molecular decay or death of a bone, in which it becomes softened, discolored and porous. This decay produces chronic inflammation and forms a cold abscess filled with a cheesy, fetid, pus-like liquid, which generally burrows through the soft parts until it opens externally by a sinus or fistula.
- **Fistula: an abnormal passage. In effect, these definitions mean that we have necrotic material that is hidden away from sight, mainly walled off, but eventually leaking its gangrenous materials into the rest of the body (Neville et al 2002, Newman 1996).**

Clinical results have demonstrated a consistent link between the existence of cavitations and the presence of many treatment-resistant diseases (Herzberg & Weyer 1998, Mattila 1993, Newman 1996, Nord & Heimdahl 1990) (see Fig. 11.8A).

Diagnosing cavitations

The presence of cavitations may be linked to a range of symptoms and the presence of recurrent and unremitting health problems suggests the possibility of cavitations. Dental assessment, including use of radiographs, electrodermal screening, applied kinesiology, CT and most accurately (and most recently) the Cavitat ultrasound three-dimensional imaging device, can diagnose the presence of cavitations. Treatment of these cavitations can often result in the alleviation of substantial and seemingly unrelated pain and suffering (Stockton 1998).

Symptoms which have been associated with cavitations include the following (Cutler 1999, Fischer 1940, Huggins & Levy 1999, Stockton 1998).

- Amyelotrophic lateral sclerosis (ALS)
- Migraine
- Multiple sclerosis

- Angina
- Arthritis
- Asthma
- Bacterial endocarditis
- Bronchitis
- Eczema
- Epilepsy
- Gangrene
- Gout
- Herpes
- Iritis
- Nephritis
- Neuritis
- Pain with or without referral
- Parotiditis
- Pneumonia
- Sinusitis
- Sore throat
- Trigeminal neuralgia
- Tonsillitis
- Ulcer

Diagnosis of cavitational lesions should be performed by a qualified dentist/dental surgeon using methods including the following.

- Radiographs. In this medium a cavitation is very difficult to discern. Most dentists have been trained to misdiagnose areas which we now know to be cavitations (by way of other diagnostic methods) as normal bone formation. Mainly this confusion is due to the fact that in most cases where actual cavitations are present, the cortical plate has not been compromised. Doctors seeing such X-rays only 'see' the healthy cortical plate which leads them to report an incomplete or faulty analysis of the condition. Unless the practitioner is adequately trained in this identification process the cortical plate is likely to conceal the presence of the vast majority of cavitational lesions. Figure 11.8B shows the highlighted presence of a cavitation that was identified using a combination of X-ray and Cavitat analysis.
- Electrodermal screening. Originally known as EAV (Electro-Acupuncture according to Voll), developed in the early 1950s, this technique measures electromagnetic field disturbances in the body. In the hands of a well-trained technician this type of screening can provide specific analysis of the body's health. An experienced and knowledgeable practitioner can use this device to uncover the possibility of cavitations and their location, often to the accuracy of a quadrant or tooth site (Fetzer 1989, Voll 1978).
- Applied kinesiology. This method of 'asking' the body to diagnose itself was originally developed by Goodheart (Walther 1988). Despite a paucity

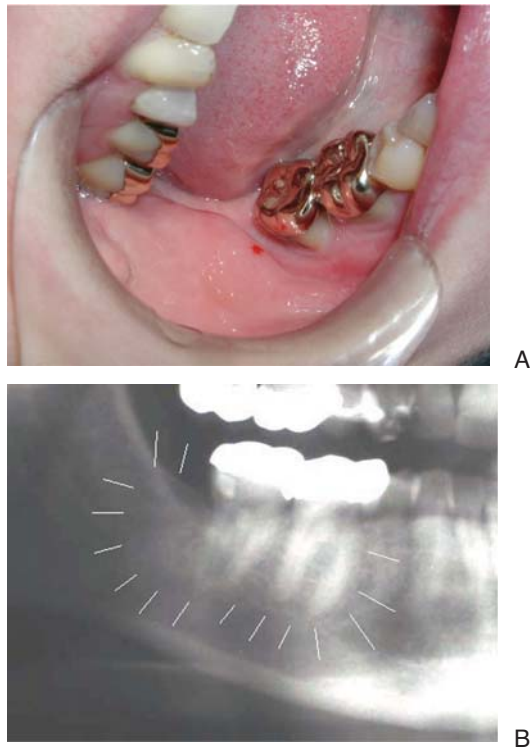


Figure 11.8 The first image (A) gives the appearance of a healthy jaw condition and X-ray analysis on its own could leave one with an inconclusive or incorrect diagnosis (B). Once diagnosed and surgical treatment is begun, the presence of cavitations becomes obvious.

of research validation, practitioners who are familiar with AK claim clinical success in identifying the effects of cavitations and may use that knowledge to more accurately assess the body's responses (Gelb 1977; see Ch. 5).

- Computed tomography (CT). This X-ray based diagnostic tool provides computerized axial tomography of the skull. The higher resolution and ability to see cross-sections inside the bone make it very precise in its diagnostic abilities.
- Cavitat ultrasonograph three-dimensional imaging device. This device can provide an accurate three-dimensional image representing density changes within the alveolar process (jawbone). These density changes have been determined to accurately represent the health of the bone (Stockton 2002, Walker 2000, Zeines 2000).

Cavitations, when diagnosed with the Cavitat, may be graded on a scale of 0–4, with a 0 rating indicating normal healthy bone. A rating of 1 is

the diagnosis for bone that has reduced blood/fluid flow in the examined area. 2 indicates that there is an ischemic area of bone present, which means that while the bone is probably still technically 'living' the lack of blood flow to the area is endangering its health and viability. 3 and 4 are indicative of the presence of necrotic material and the necessity for surgical intervention (Fischer 1940, Stockton 1998).

When the health of a section of bone begins to degrade and degenerate (rating of 2–3) the body identifies a growing source of toxicity and begins to defend itself by creating a hard bony layer around the toxic area. This walling off of the cavitation is what makes it difficult to identify cavitations through X-ray examination. While the toxic cavity is walled off the patient can go for extended periods of time without any indication that there is poisonous, gangrenous material in their jawbone. The growing cavitation will eventually begin leaking necrotic material into the rest of the body with potentially serious consequences (Cohen & Burns 2002, Herzberg & Weyer 1998, Neville et al 2002, Newman 1996, Price 1945).

Development of cavitations

Cavitations generally develop as a result of trauma, bacterial infection, reduced vascular activity or toxicity (Stockton 1998).

Cavitations are usually the result of one of the following (Shankland et al 2001, Stockton 1998):

- an infection the body has walled off to protect itself
- reduced blood flow resulting in dead or dying bone
- physical trauma, when the jaw is unable to heal itself.

Disease-related results of cavitations in the body

Dorland's illustrated medical dictionary (24th edn) provides a basis for a discussion of the implication of cavitations in the body.

- Metastasis. The transfer of disease from one organ or part to another not directly connected with it. It may be due either to the transfer of pathogenic micro-organisms or to transfer of cells, as in malignant tumors.

Box 11.3 Types of trauma that can lead to cavitations**Physical trauma**

Tooth extractions
 Root canal procedures
 Dental injections
 Periodontal surgery
 Grinding and clenching
 Electrical trauma from
 dissimilar metallic restorations
 Heat from high speed drilling

Bacterial trauma

Avital (dead) teeth
 Infected wisdom teeth
 Root canal bacteria
 Abscesses
 Cysts
 Improper removal of periodontal
 ligament after tooth extraction
 Periodontal disease

Toxic trauma

Root canal toxins
 Bacterial toxins
 Anesthetic vasoconstrictors
 Dental materials
 Chemical toxins
 Anesthetic by-products
 Other toxins

- **Metastases.** A growth of pathogenic micro-organisms or of abnormal cells distant from the site primarily involved by the morbid process.
- **Metastasize.** To form new foci of disease in a distant part by metastasis.

The ideas behind focal infection have been with modern medicine since 1877 with Carl Weigert's observations of a 'dissemination of "tuberculosis poison"' (Fischer 1940). Since then there have been many studies analyzing distant effects of focal infections as it has been shown that oral pathogens can infect other parts of the body (Herzberg & Weyer 1998, JADA 2002, Mattila 1993, Neville et al 2002, Newman 1996, Nord & Heimdahl 1990, Shankland et al 2001).

Treatment of cavitations

Treatment of cavitations can be accomplished by surgical removal of the cavitation lesion or non-surgical therapies designed to help the body heal itself. Though the non-surgical avenues of treatment are generally only appropriate when the lesion has not yet reached the 'necrotic' stage and is more ischemic in nature, those types of treatment can often be incorporated with surgical intervention to increase the chances of success.

Figure 11.8A gives the appearance of a healthy jaw condition and X-ray analysis on its own could leave one with an inconclusive or incorrect diagnosis (Fig. 11.8B). Once diagnosed and surgical treatment is begun, the presence of cavitations becomes obvious.

Figure 11.8C shows a first molar extraction and the beginnings of exposure of a deep cavitation at

the previous third molar extraction site. Once the oral surgeon began cleaning out the cavitation it became apparent how extensive the necrosis was (Fig. 11.8D).

Non-surgical treatment of less serious cavitation lesions is an area with less documentation, less consistent results and many differing avenues of treatment (Hodgson & Hansen 2000, Tuner & Hode 1999). Some success has been achieved with treatment protocols that involve infrared pads, low-level lasers and nutritional guidelines (including enzyme therapy). The internet is a good resource for identifying alternatives in this area.

Cranial effects of cavitations

In the author's clinical experience, cavitation lesions cause reduction in the amplitude of cerebrospinal fluid fluctuation and in the overall vitality of the individual. Elimination of the cavitations should theoretically have a positive effect on neuromuscular balance and on the effectiveness of cranial therapy.

Cranial implications of intraoral metals

Evidence suggests that the presence of mercury (silver amalgam) fillings and other metals in the mouth interferes with the proper function of the nervous system (Carpi 1998, EPA 1997). Additional electromagnetic fields, produced by the presence of different metals, may lead to irritation of the nervous system. Both these factors are reported to inhibit the effectiveness of cranial therapy (Huggins & Levy 1999, Walker 2000).



Figure 11.8 C shows a first molar extraction and the beginnings of exposure of a deep cavitation at the previous third molar extraction site. Once the oral surgeon began cleaning out the cavitation it became apparent how extensive the necrosis was (D).

Summary

Mercury is a powerful neurotoxin (EPA 1997, Simon 2001, WHO 1991).

- Mercury vapor constantly leaks from amalgam fillings, even after having been in the mouth for 20 years (Leistevuo 2001, Sellars & Sellars 1996, Zeines 2000).
- Research shows that there are three definitive genome types which determine how the body will handle the assimilation of mercury. This may explain why some people react strongly to small amounts of mercury (Cutler 1999, Ziff & Ziff 2001).
- Two or more dissimilar metals, in contact, cause a current, for example in the mouth of the patient featured in Figure 11.9A–C.
- In the mouth, restorations of differing metals (or even silver amalgam fillings done at different times) such as non-precious metal crowns, gold, stainless steel, etc., combined with saliva (an electrolyte), creates electrical currents that are far greater than those involved in normal neurological activity (Cutler 1999, Stortebecker 1985, Vimy 1999, Walker 2000, Ziff & Ziff 2001). These currents can negatively affect neurological function (Marino & Ray 1986, Neutra 2001, Thomas et al 1987).

Clinical example

The patient featured in Figure 11.9A was experiencing severe memory loss. The author's electrical tests showed very high readings between teeth, crowns, fillings and root areas. Figures 11.9B and 9C view the various metals present which, with the saliva acting as an electrolyte, were creating a flow of electrons similar to a battery (Becker & Selden 1985, Raue 1980, Stortebecker 1985). Improvement of her mnemonic abilities followed shortly after removal of the amalgam fillings and metallic crowns.

INAPPROPRIATE ORAL SURGICAL PROCEDURES

Alteration of the jaw form, structure and position without proper consideration given to the cranial mechanism can cause harmful long-term effects.

Bicuspid extraction

It is not uncommon for a dentist or orthodontist to diagnose a patient as having a tooth/jaw discrepancy (generally meaning that the jaw is not sufficiently large to accommodate the teeth that are present or erupting) (Simon 2001). Some



A



B



C

Figure 11.9 A–C Patient who experienced severe memory loss. The author's electrical tests showed very high readings between teeth, crowns, fillings and root areas. Figures 11.9B and 11.9C show the various metals present which, with the saliva acting as an electrolyte, were creating a flow of electrons similar to a battery.

orthodontic philosophies believe that jaws stop growing at a certain age, usually 11–15 years old, and after that age the only way to make room for the teeth, and to 'straighten' them, is to remove other teeth (Mahoney et al 2003, Mew 1999, Zeines 2000). In such cases referral may be made to an oral surgeon for bicuspid teeth to be removed.

Such removal leads to elimination of the normal forces on the jaw to continue its natural growth (Enlow 1975). This process often leads to the creation of more space than is actually necessary to 'straighten' the patient's teeth. Tight muscles and fascia whose forces may originally have contributed to the underdevelopment of the jaw continue to exert their force (Enlow 1975).

With no opposition, these forces may, through their constant constrictive action, force the arch to shrink to a size more appropriate for the remaining teeth (see Fig. 11.10A) (Gelb 1994, Mahoney et al 2003, Mew 1999).

This pressure is commonly increased with the orthodontic practitioner's use of braces and headgear, resulting in a posterior movement of the lower half of the face (see Fig. 11.10B) (Mew 1986, Page 2003, Spahl & Witzig 1991).

This posterior movement or 'distilization' of the maxilla (and as a result, the mandible as well) creates compression of various structures (nerves, vessels, dura, muscles, bones and fascia) between the upper front teeth and the occiput (Baker 1971, Jecmen 1998). It has been suggested that the effects of this procedure can lead to:

- depression (Hockel 1983)
- snoring (Katsev 2003)
- sleep apnea (Frymann 1998, Katsev 2003)
- vision problems (Page 2003)
- hearing difficulties (Gelb 1977)
- vocal cord nodules (Solberg & Clark 1980)
- swallowing problems (Jecmen 1995)
- TMJ dysfunction (Jecmen 1998)
- mid and low back pain (Page 2003)
- headaches (Solberg & Clark 1980)
- reduced self-esteem (Frymann 1998)
- birth/conception difficulties (Ziff & Ziff 1987)
- endocrine/growth disturbances.

The patient in Figure 11.10 is a good example of the conditions described in this section. Many of the symptoms listed here are issues she has faced.

Figure 11.10A shows the retarded growth of the upper and lower jaws, which has basically made the lower half to one-third of the face set back 8–10 mm. Functionally this has also resulted in an airway problem; esthetically the patient appears very nearly chinless.

Figure 11.10B shows a picture of two upper ALF appliances. The small one is that of the patient in Figure 11.10A, the large one belongs to her 8-year-old son. In both appliances, the 'cribs' (circles of metal that fit over the same teeth – first permanent molars in both mother and son) show an astonishing size difference. Consider the pressures that having the maxilla and mandible placed in such a posterior position must bring to bear on the rest of the cranial mechanism (Jecmen 1998). It also follows that the more teeth that are removed, the less support the TMJ receives, as vertical support is reduced.

Muscular imbalance

Loss of tooth mass produces neuromuscular imbalance (Page 2003, Smith 1986). Normal muscle length will now be inappropriate since the vertical distance from the upper skull (and jaw joint) to the lower jaw will have been reduced by the loss of teeth. The muscles and fascia anterior to the upper cervical spine would be altered, with potential changes in the neck curvature and occipital position. Vagal nerve compression and distress to the areas innervated by the vagus nerve can result (Gelb 1994).

Compressive effects

Some oral surgical procedures such as maxillary resection and bicuspid extraction can have compressive effects on the maxillary sinuses as well. As mentioned previously, tooth extraction often has the negative effect of reducing jaw growth. The resultant smaller skull size manifests in compressed vertical face height (Frymann 1998, Mahoney et al 2003, Mew 1999, Upledger 1987) which, when combined with scar tissue formation, creates compromised sinus size and function (Burr Saxton 1972, Voll 1978).

Though there is no research that the author is aware of substantiating reduced nasal function as a direct result of bicuspid extraction, it has



A



B

Figure 11.10 **A** This shows the retarded upper and lower jaws, which have in effect made the lower half to one-third of the face set back 8–10 mm. Functionally this has also resulted in an airway problem and esthetically the patient appears very nearly chinless. **B** Two upper ALF appliances. The small one is that of the patient in Figure 11.10A, the large one belongs to her 8-year-old son. In both appliances the 'cribs' (circles of metal that fit over the teeth – lower part of the appliance) fit onto the first molars. The size difference is astonishing. Consider the pressures that having the maxilla and mandible placed in such a posterior position must bring to bear on the rest of the cranial mechanism (Jecmen 1998). It also follows that the more teeth that are removed, the less support the TMJ receives, as vertical support is reduced.

been noted in clinical practice that one frequently accompanies the other. Possibly the reduced nasal capacity precedes the bicuspid extraction and contributes to the condition that eventually is diagnosed as requiring extractions. On the other hand, it could be the extractions that result in, or contribute to, the reduced nasal function (Hockel 1983, Page 2003).

There are some instances where removal of teeth is indicated; however, appropriate FJO and cranial treatment can reduce subsequent dysfunction to a minimum.

Dental Surgery

There are a variety of situations in which surgery may be appropriately or inappropriately suggested. For example, where there is upper to lower jaw size discrepancy, incorrect positioning of jaw or jaws, improper face form, clicking of the TMJ, acute trauma, severe joint degeneration, chronic infection or reconstruction following cancer or repair of congenital anomalies (Morgan et al 1982, Neville et al 2002, Solberg & Clark 1980).

Some of these conditions may be better served by a more conservative, non-surgical technique. Each case should be evaluated individually.

The specific surgical procedure proposed by the oral surgeon depends on the diagnosis and philosophy of the surgeon. When cranial/jaw surgery takes place, the new muscle orientation is resisted by those muscles which seek to return to their previous state. This reorientation places stress on the neuromuscular system and on the cranial mechanism. A relapse rate of 40–70% has been reported in the literature (Morgan et al 1982).

Oral surgery is seldom accompanied by follow-up treatment to help adjust the cranial mechanism (Smith 1986). Appropriate or inappropriate as the surgery may be, to not relieve the stresses created by such surgery on the neuromuscular system may cause undiagnosed effects to the structure and function of the cranium and its sutures.

Jaw surgery, though sometimes indicated, can also affect the somatognathic system by creating neuromuscular disturbances, often without improving the underlying cause of the dysfunction, which may very well have been neuromuscular or craniosacral in origin. An example of this situation can be seen in Figure 11.11 (Huggins & Levy 1999). It is imperative that cranial and other neuromuscular therapy accompanies surgery of the jaw (Frymann 1998).

Figure 11.11 shows a panoramic X-ray of a surgical procedure to close an anterior open bite secondary to TMJ treatment. Note the metallic parts relative to surgical realignment. The patient in this case experienced no relief from TMJ symptoms after the surgical procedure. In fact, her overall level of health declined considerably following the procedure. Her symptoms included: severe lymphatic congestion; suicidal thoughts with need for psychiatric care and antidepressants; reduced cognitive and speech abilities; partial loss of

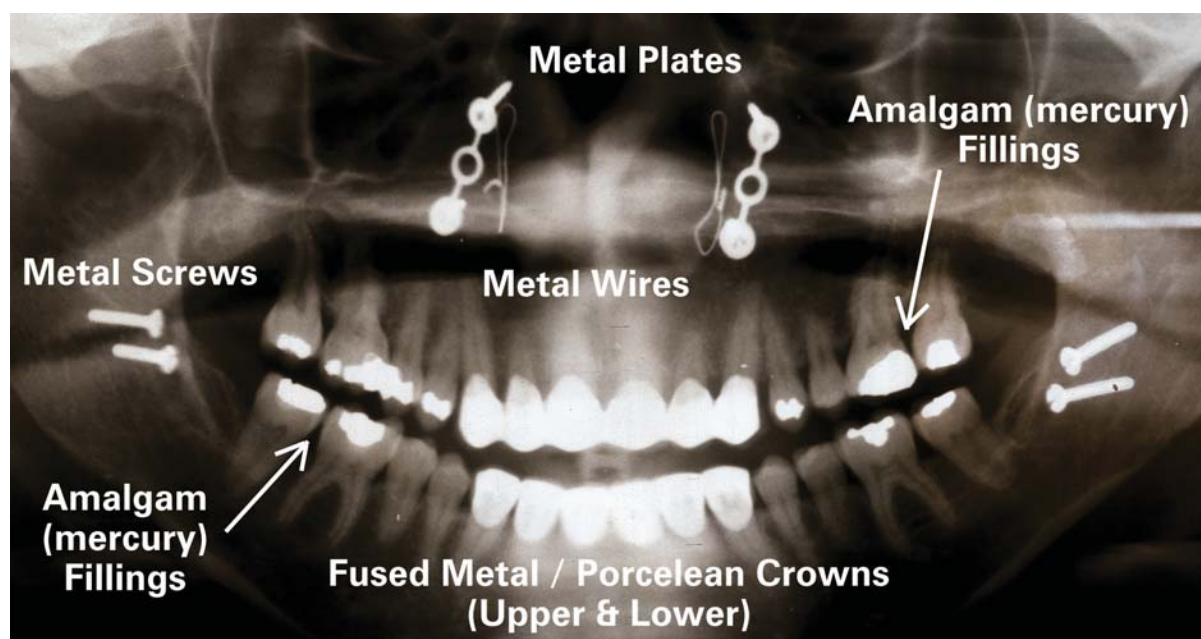


Figure 11.11 Jaw surgery can also affect the somatognathic system by creating neuromuscular disturbances, often without improving the underlying cause of the dysfunction, which may well have been neuromuscular or craniosacral in origin.

memory; and inability to smile. All symptoms and general condition improved following appropriate dental rehabilitation. Within 3 weeks this patient was off all antidepressant medication with the approval and recommendation of her psychiatrist.

Braces, bridges, dentures and other dental therapies

These can all have negative side-effects on cranial function when constrictive treatment modalities cross (fix) sutures in the maxillae or mandible. Though the number of functional sutures in the jaw bones is debated, the existence of four is generally acknowledged.

The three sutures found in the maxilla are the maxillary/midsagittal suture (the midline suture found between the two front teeth) and two premaxillary sutures (just medial to the cuspids – eye teeth).

In the mandibular area, the most widely recognized suture is the symphysis menti (also located between the two front teeth at the midline) (Gehin 1985, Magoun 1976, Simon 2001).

Though fixation, eliminating the freedom of movement, at any of these sites can have negative effects, it is most vital that the maxillary/midsagittal suture retains freedom of motion. Fixation of this suture can lock the front of the head and reduce overall cranial motion. In some patients this may not noticeably impact on the individual's daily life but in others the effects can be serious (Smith 2000a, Laughlin 2002a, b).

If cranial motion is reduced by mechanical means, the cranial therapist may be unable to influence the resulting symptoms. Release of the fixation will, in nearly every case, instantly improve cranial function and provide the patient with instant relief of seemingly unrelated symptoms (Laughlin 2002a, b).

Bridges, braces, dentures, some appliances (e.g. rapid palatal expander) and other therapies (e.g. headgear) can all have this 'fixating' effect to some extent. While all of these therapies are esthetically and functionally important, their use in some cases can be harmful (Frymann 1998, Huggins & Levy 1999).

In the author's clinical experience the most disturbing of all these therapies is the placement of

a bridge that crosses the midline maxillary suture. Some of the symptoms that this can contribute to are:

- depression (sometimes clinically)
- headaches
- feelings of claustrophobia
- irritability
- impaired reaction time
- sternocleidomastoid dysfunction
- sinusitis.

Inappropriate suture constriction caused by fixed bridges

Fixed bridges are prosthetic devices which are bonded onto two teeth in order to replace one or more teeth in between. Teeth are normally independent units, not bonded or fused together (see Fig. 11.12A,B). When a bridge is constructed and cemented in place it essentially fuses or locks a span of teeth together. This is especially restrictive when done in the front part of the mouth (crossing the midline) and inhibiting the action between the right and left maxillae.

In cases where the bridge has already been cemented and is found to be restrictive, the author's clinical experience has shown that cutting the bridge between the two front teeth provides immediate relief to the patient in the majority of cases.

In Figures 11.12A and B we see the crowns all splinted solidly together in one unit in both the maxillary anterior and the mandibular anterior, similar to what would be seen in a bridge.

Figure 11.12B also shows where the cuts in the splinted crowns were planned. These planned cuts correspond to the maxillary/midsagittal suture and two premaxillary sutures (it does not show where the cut was made for the mandibular/symphysis menti).

Figure 11.12C shows an exterior view of the splinted crowns after the three cuts were made at the locations of the sutures. A thin diamond disk was utilized to sever these sections. The patient noticed immediate relief of cranial tension and smiled more easily. She also soon experienced a 50–70% reduction in the swelling of her hands and feet. She was referred because she had been on medical leave from her forklift job, due to an

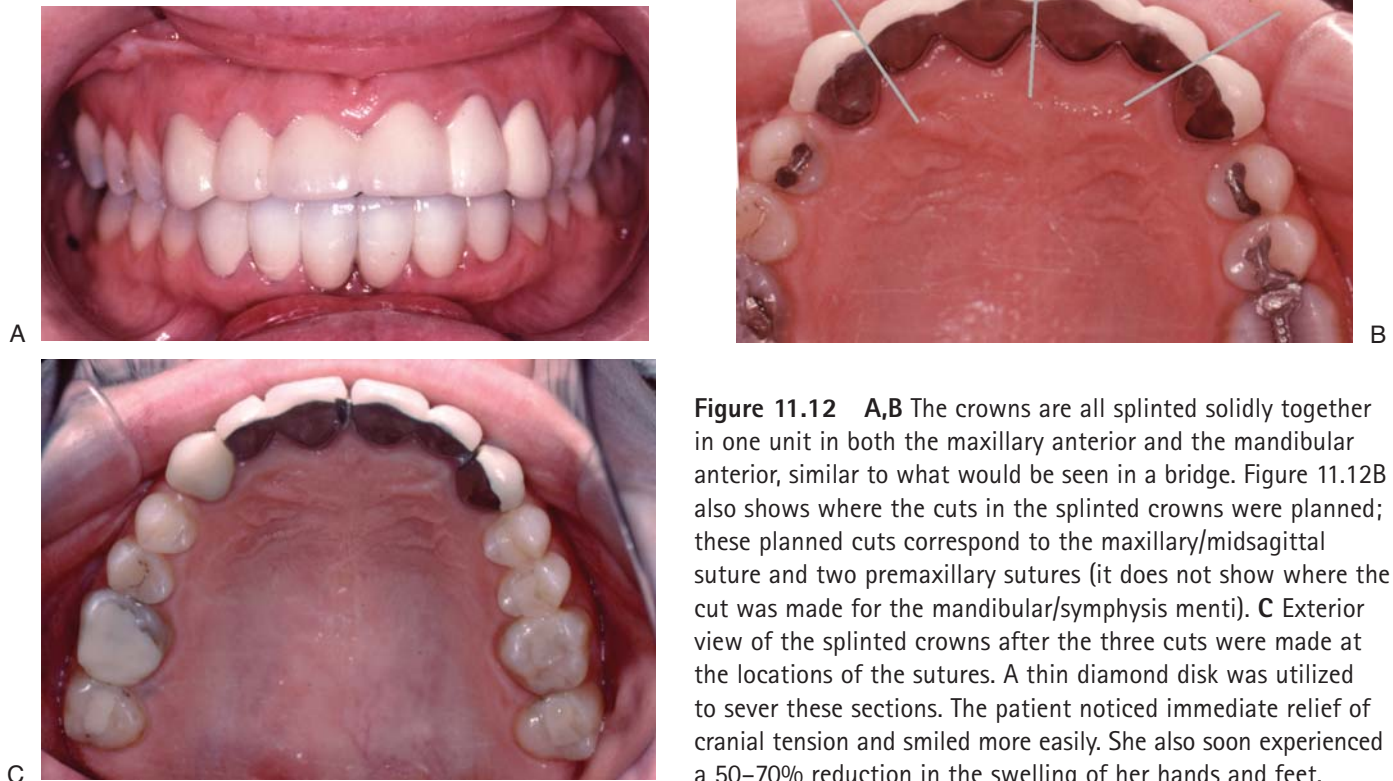


Figure 11.12 A,B The crowns are all splinted solidly together in one unit in both the maxillary anterior and the mandibular anterior, similar to what would be seen in a bridge. Figure 11.12B also shows where the cuts in the splinted crowns were planned; these planned cuts correspond to the maxillary/midsagittal suture and two premaxillary sutures (it does not show where the cut was made for the mandibular/symphysis menti). C Exterior view of the splinted crowns after the three cuts were made at the locations of the sutures. A thin diamond disk was utilized to sever these sections. The patient noticed immediate relief of cranial tension and smiled more easily. She also soon experienced a 50–70% reduction in the swelling of her hands and feet.

inability to close her hands due to the extreme swelling.

It is important to note that cutting the bridge in the mouth is like severing a bridge that crosses a river. When cut, the structural integrity of that bridge is compromised. Because of the probable benefits to the health of compromised patients, the author, in his clinical experience, will provide an option that the bridge be cut even though it could compromise the stability of the prosthesis.

Future options for the patient include the following.

- Replace the missing tooth with a removable non-metallic partial.
- Leave the 'cut' bridge in place (recementing if it dislodges).
- Insert a new (ideally non-metallic) bridge with a 'stress break' that allows sutural movement. A special attachment (called a CMA) has been developed that can be incorporated by the dental lab into the fixed bridge construction, allowing

the sagittal suture of the maxilla to retain its freedom of motion (Smith 1986, 2000a). This is one alternative that may solve the problem of cranial restriction for the patient.

Sutural restriction caused by fixed orthodontic braces

Fixed orthodontic braces can also restrict critical sutural motion by essentially creating a complete fixation of all upper and/or lower teeth (Frymann 1998, Magoun 1976, SOTO 2001). In some patients this will not impact their daily lives but in others, the effects can be debilitating. Young adults who are faced with this problem will commonly have trouble putting words to their difficulties. For this reason it is important to evaluate the overall well-being of the patient when use of this therapy is incorporated into a treatment plan.

The greater the arch-wire thickness used for braces, the greater the restriction of motion. Because braces are sometimes necessary, if the patient is

experiencing difficulty with their use, one option would be to ask the orthodontist to consider the cranial sutures in his/her treatment and to possibly reduce the period of time braces are used. Because of differences in training and philosophy, many orthodontic practitioners may be unconvinced regarding this concern. Using the ALF appliance before and during use of braces can also help to reduce the time braces are necessary and reduce the cranial restrictions.

Restricted motion of the maxilla, temporals and sphenoid can also occur with overly tight partials or dentures (Upledger 1987). Splint therapy using a rigid upper appliance (e.g. rapid palatal expander) can have similar negative effects due to its restrictive nature. In cases where these therapies could be the cause of health-compromising symptoms, it is suggested that the offending prosthetic devices be replaced with ones more conducive to sutural movement.

THE POTENTIAL BENEFICIAL INFLUENCES OF CRANIAL THERAPY

The information contained in this section is based on years of clinical practice in the field of whole-person dentistry. The author and his peers in the fields of functional orthopedics/orthodontics, biological dentistry and holistic dentistry have shared clinical experience with one another and come to the conclusions represented in this chapter. Distinct advantages can be gained when incorporating cranial techniques, such as the occipital, pterygoid and SBS release, into therapeutic dental programs.

Practically speaking, the author has accomplished orthodontic techniques with and without the benefit of cranial therapy.

The advantages of using (or referring) for cranial therapy may include improved:

- pain reduction following appliance adjustments
- amplitude and symmetry of cranial motion
- overall attitude of the patient and improved co-operation with the treatment.

Adult patients, following an orthodontic, TMJ or general dental appointment, commonly express gratitude after receipt of bilateral medial pterygoid,

SBS and suboccipital release (see below), which the author performs and believes to assist in rebalancing chronic TMJ/cranial issues and some acute (iatrogenic) dental trauma following their appointment.

OCCIPITAL RELEASE

Freedom of motion and relaxation of the suboccipital triangle appears to have broad-ranging effects. Throughout the author's 28-year career in whole-person dentistry, it has been frequently demonstrated that relaxation in this region greatly enhances the positive cranial changes which occur during the use of dental cranial orthopedics/TMJ therapy (Frymann 1998, SOTO 2001).

Clinical experience shows that the cranial release which occurs with treatment of the occiput, C1, C2 region facilitates the patient's recovery following dental appointments. Venous drainage is positively affected by this release (Frymann 1998). The author has had subjective responses from his patients reporting clearance of nasal and maxillary sinuses, ease of breathing and drainage into the throat with these procedures (Hammer 2003). It is theorized that all sinuses are positively affected, including the superior sagittal and straight sinuses. In the author's clinical practice, the person who experiences the stress of chronic TMJ/TMD, long dental appointments or dental orthopedic treatment can benefit greatly from these therapies (see Exercises 7.3 and 7.29, Ch. 7).

LATERAL AND MEDIAL PTERYGOID RELEASE

The author not only uses the internal pterygoid release following almost 90% of all dental procedures, but will often perform it before procedures are begun. The effects are marked. In this clinical setting two measurement criteria are used to determine the effectiveness of the therapy. The first is the maximum distance the mouth can open. Using this criterion, the release routinely demonstrates an average increase in jaw opening of 3–6 mm. The second criterion used to measure effectiveness is responsiveness to commands.

While in the dental chair it is common to see delays in the patient's ability to process information in the form of commands and their response to the command. Responses after the internal pterygoid release are faster and more accurate.

The lateral and medial pterygoid muscles are extremely important in TMJ and cranial dysfunction. The lateral pterygoid muscle is important in its relationship to the mandibular positioning as well as the temporal mandibular disk or meniscus positioning (Chaitow 1999, Chaitow & DeLany 2000, McCatty 1988).

The medial or internal pterygoid has its origin at the pterygoid process of the sphenoid bone and can directly affect not only the sphenoid but also the temporal, the occiput and the maxillae (Magoun 1976). The wide range of influences this muscle has on the patient's health and well-being requires it to be in a relaxed state when it is not in use. In clinical practice, the author has never found a patient with TMJ dysfunction that did not have problems with neuromuscular imbalance of either or both the pterygoid muscles. In the author's opinion it is essential that normal tone is restored to these muscles before any progress can be made in treating the orthopedic orthodontic, or TMJ, needs of patients (Laughlin 2002a, b). (See pp 282–283 for treatment methods for the pterygoids.)

In the author's opinion cranial therapists could benefit all dental patients if they were seen soon after dental treatment. This is especially true following a long operative (dental restorative) or surgical procedure(s), when the patient's musculature has been subjected to strain. Home therapy to release internal pterygoid tension prior to dental appointments is also possible.

SPHENOBASILAR SYNCHONDROSIS (SBS)

The author's clinical experience strongly supports the importance of 'balanced membranous tension' throughout the cranial structure. Marked clinical changes have been noted following techniques which are directed toward membranous/energetic/osseous mobility of the SBS. Enhanced responses to dental therapy and functional jaw orthopedics have frequently been observed following appropriate SBS treatment (Frymann 1998, Gelb 1977,

SOTO 2001). The author believes that compression of this region of the cranium may relate to dental fixation between the right and left maxillae (caused by bridges, braces, etc.). It is hypothesized that releasing those fixations changes the mobilization at the SBS (Jecmen 1998).

ADDRESSING CRANIAL ORTHOPEDICS EARLY IN LIFE

Early attention brought to dysfunctional tendencies can eliminate their development later (see Fig. 11.13) (Zeines 2000).

PREVENTATIVE TREATMENT SAVES PATIENTS PAIN AND SUFFERING

A variety of symptoms and problems can be avoided later in life with early treatment consideration, including the following.

TMJ dysfunction: what it is and how it may be avoided

Some of the symptoms associated with TMJ problems in adult life (i.e. late adolescent and through adulthood) include earache, clicking/pain of the jaw, headaches, unbalanced face form, tonsillitis, pain while chewing, sinusitis, tinnitus, crooked teeth and swallowing difficulty resulting in face, neck and/or lip contortions during swallowing (Gelb 1977). It is suggested that had the patient shown in Figure 11.13 continued to develop uncorrected, she would probably have exhibited many of these signs and symptoms (Page 1949, Simon 2001).

The development of such symptoms in adult/late adolescent life can be avoided if the tendency is diagnosed and treated early. Early treatment of such tendencies, such as arch widening, jaw repositioning (through orthopedic orthodontic appliances) and/or cranial therapy, can provide positive results. The most beneficial treatment may usefully involve a combination of neuromuscular, cranial and dental orthopedic therapies. In normal clinical practice, dental orthopedics will not usually be initiated prior to the age of 4–6 though when

future tendencies toward malformation are seen, 4–6 is an ideal age to begin treatment (Page 1949).

Improper growth (which can lead to considerable difficulties as an adult) is often responsive to orthopedic orthodontic techniques, especially if addressed early in life (Page 1949).

Although surgery may be unavoidable, in the author's opinion it should not be the first option considered as it may cause permanence of the skull/cranio/cervical malrelationship with the rest of the body. Scar tissue is a secondary adverse effect of surgery that may cause disruption of the functioning of the neuromuscular system (Becker & Selden 1985).

Early treatment with functional jaw orthopedics as well as identifying and eliminating the causes of the abnormal growth is paramount to the prevention of the malformation (Fig. 11.13). It is significantly more advisable to develop the jaw size at 4 years of age rather than have baby teeth extracted. There are some instances when surgery is not only important but necessary but it should be the last, not the first option explored. Seeking the alternative becomes an uphill battle where insurance reimbursement is concerned (Carter 1993). This may be another reason why dental practitioners are slow to make the shift to incorporating alternative therapies into their practices.

The five images in Figure 11.13 tell a complete story of dysfunctions corrected by FJO. No braces (fixed orthodontics) were used in this case.

Growth disturbances had been developing since birth in this case. The author began treatment very soon after the patient's first visit at the age of 7. A combination of direct bite build-ups (using composite resin, non-metallic filling material to build up the vertical of her teeth) and ALF appliances with elastics was used to produce these results by the time she was 8 years old.

Cranial therapy was used to great benefit and, in the author's opinion, enabled the results to be obtained in a shorter period of time. This patient is a prime example of a child whose situation, speaking

from clinical experience, would have probably led to TMJ problems as well as many other symptoms without the treatment as described.

Posture and airway obstruction

What follows is a scenario that has been seen regularly in the author's clinical experience.

- Poor posture contributes to forward head positioning during eating, sitting, studying, working or sleeping.
- This in turn leads to airway obstruction which causes mouth breathing and resultant low tongue posture.
- If the tongue is in a 'low posture' position, it is not properly positioned up against the roof of the mouth, unable to stimulate forward and lateral growth of the maxillae.
- This lack of stimulation inevitably leads to insufficient growth and development.
- The resultant poor growth and development of the jaws will result in crowding of the teeth.
- At this point, very often the parents will be advised to remedy the situation with four bicuspid extractions and fixed orthodontics.
- This only exacerbates the underlying problems and in the end can lead to body-wide hormonal changes and neuromusculoskeletal problems (Jecmen 1998).

Treatment from a dental orthopedic viewpoint involves proper maxillary, nasal, sinus and airway development. Such cranial development assists proper tongue and head position which then translates into correct head, neck, jaw and thorax relationship (Gelb 1977). When these regions are in balance and nasal breathing is habitual, then the tongue is in correct location in the mouth (up with the tip behind the front teeth) to provide positive encouragement toward stimulation and then stabilization of the arch form (Gelb 1994). This broad maxillary form then provides a sound base for the lower jaw to function within a healthy downward and forward direction (Mew 1986). The TMJ complex appreciates this posture and mandibular position – less compression, improved circulation and less crowding of the cranial system (Gelb 1994).



A



D



B



E



C



Figure 11.13A–E These images tell a complete story of dysfunctions corrected by functional jaw orthopedics. No braces (fixed orthodontics) were used in this case. Growth disturbances had been developing since birth in this case. The author began treatment very soon after the patient's first visit at the age of 7. A combination of direct bite build-ups (using composite resin, non-metallic filling material to build up the vertical of her teeth) and ALF appliances with elastics was used to produce these results by the time she was 8 years old. Cranial therapy was used to great benefit and in the author's opinion enabled the results to be obtained in a shorter period of time.

CORRECTING CRANIAL DYSFUNCTION DENTALLY

There are numerous classifications of cranial lesions which relate to the reciprocal tension membranes, motion of the cranial bones, flow of the CSF and lymph, as well as energetic blockages (Frymann 1998, Gelb 1977, Jecmen 1998).

Symptoms which are caused by cranial lesions may include headaches, ear problems (vertigo, tinnitus, otitis media), nasal congestion, sinusitis, maxillary and mandibular growth disturbance, endocrine disturbance, eye problems, swallowing problems and neck problems (Feeley 1988, Frymann 1998, Phillips 2001, Stockton 1999).

Methods of intervention using dental orthopedic techniques include: improved upper and lower jaw development; proper positioning of the maxillae and mandible relative to the sphenoid, occipital and other cranial bones as well as to each other (Jecmen 1998); establishment of proper reciprocal membrane tension within the TMJ/cranial complex (Jecmen 1998). In the author's opinion, these objectives are best accomplished through the combined therapy of functional jaw orthopedics and cranial therapy.

Example: ear problems

Correct TMJ support and correct length of the lateral pterygoid and temporal muscles are critical to free functioning and normal drainage of the Eustachian tube (Gelb 1977, Morgan et al 1982, Simon 2001).

Children may present with a variety of signs and symptoms which alert to potential ear-related problems. These include: runny nose; frequent colds; head congestion (indicative of thickened fluids and probably allergies); mouth breathing; dry lips; mandible shifting to one side, too far forward or too far backward; or a strong habit of thumb, finger or pacifier sucking. Once these issues have been identified a multidisciplinary analysis can determine the possible consequences and an ideal treatment plan (Morgan et al 1982).

Fonder (1977, 1990) accomplished some of the research in the 1960s and 1970s regarding ear problems and vertical support. He would build

up the back teeth with filling material to relieve otitis media and hearing loss. The author has found that releasing the internal pterygoid and improving vertical support are two of the most important techniques to employ. Figures 11.14A and B display a mouth-breathing patient with a developing cranial, cervical scoliosis. When treatment began she presented with extremely underdeveloped maxillary and mandibular arches. The resultant treatment brought about marked changes in posture, jaw form and jaw position (Figs 11.14C,D). Note the profile change and the ease with which the lips are able to close (facilitation of nasal breathing). Whereas before, the patient often experienced earaches and other ear problems, after the completion of treatment these problems were greatly diminished or eliminated.

EARLY OBSERVATION AND CROSS-REFERRAL

Early childhood observation and treatment, when appropriate, can eliminate or alleviate long-term symptoms and problems (Page 2003). Parental and professional attention to the criteria outlined below is critical to a timely identification and proper treatment, in order to avoid the possible development of malformations and their associated dysfunctions (Gelb 1994, Mahoney et al 2003, Mew 1999).

A list of what is required from a cranial therapist, in terms of observation, is provided below, with the suggestion that when problems are identified it may be time to consult a cranially educated dentist for further diagnosis.

Prenatal history

- Evaluate the nutrition and health of the parents. A healthy sperm and egg are the first steps toward a healthy fetus (Price 1945).
- Neuromuscular balance for the mother will help to lead to a normally functioning pelvis and birth canal (Phillips 2001).
- Magnesium consumption should ideally increase for pregnant and nursing mothers (Huggins 1981, Pierce 1994).

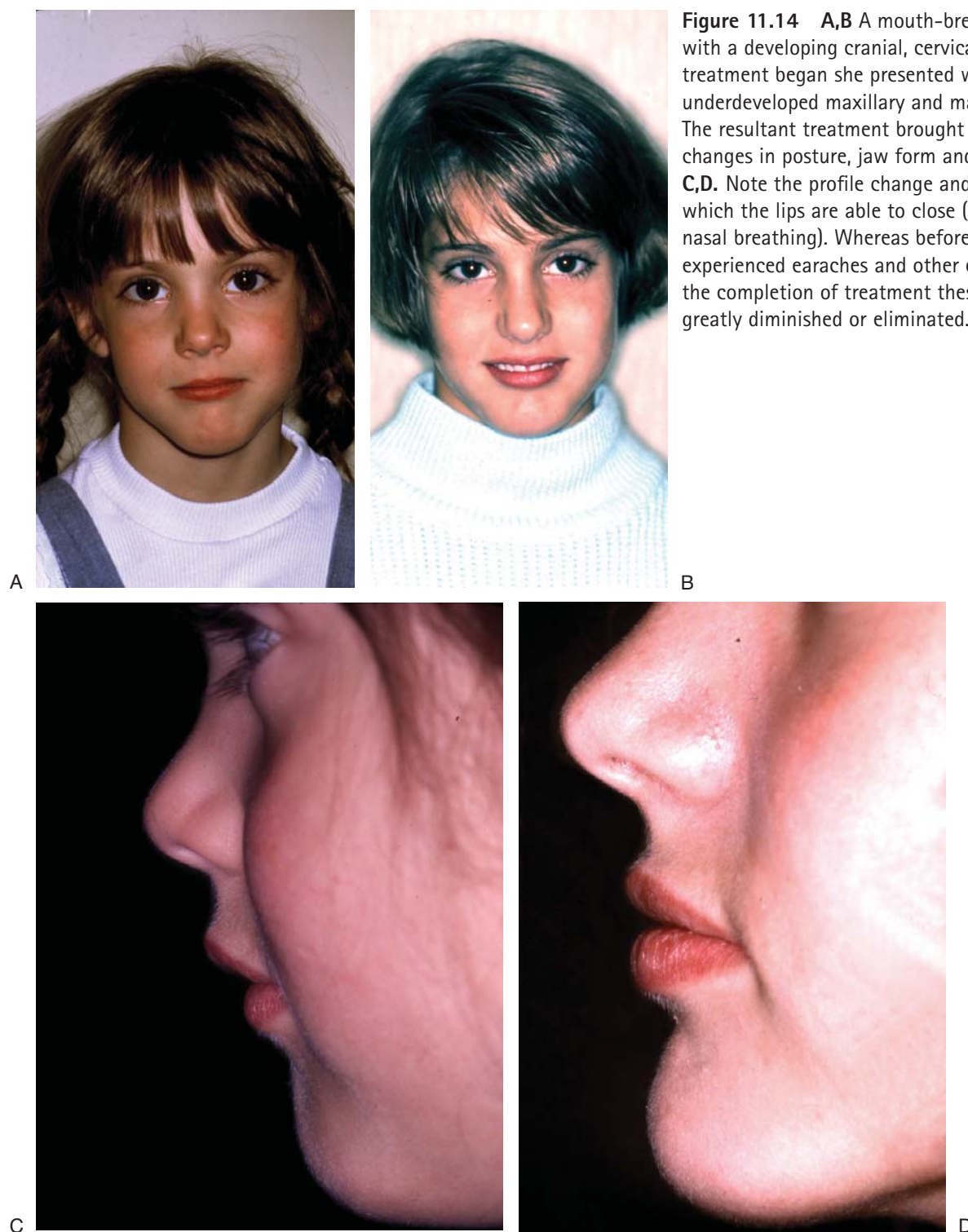


Figure 11.14 A,B A mouth-breathing patient with a developing cranial, cervical scoliosis. When treatment began she presented with extremely underdeveloped maxillary and mandibular arches. The resultant treatment brought about marked changes in posture, jaw form and jaw position C,D. Note the profile change and the ease with which the lips are able to close (facilitation of nasal breathing). Whereas before, the patient often experienced earaches and other ear problems, after the completion of treatment these problems were greatly diminished or eliminated.

Birth

- Birthing posture. Encourage ideal birth posture for the mother, avoiding being supine with stirrups, unless surgery is necessary (Northrup 1998, Phillips 2001).
- Note specifics of birth: ease, trauma (mechanical assists such as suction or forceps), length of labor, force of delivery, surgery, Apgar score (reflection of trauma) (Arbuckle 1954, Frymann 1976, 1998).

- Skull shape and form at birth (often reflective of trauma) if undiagnosed, unnoticed and untreated can have developmental repercussions in the future. These repercussions can affect the esthetic and functional presentation of the mouth (Arbuckle 1948, Frymann 1976).
- For example, consider a forceps extraction delivery of the newborn. The temporal bones, as well as the maxillae, can be driven into an internal rotation. Left untreated, this would probably lead to mouth breathing, a high palate and a recessive mandible. Early cranial treatment can reduce or eliminate the future manifestation of these conditions (Arbuckle 1948, Frymann 1976, Phillips 2001).

Age 0–4

Observe (Page 2003):

- mouth versus nasal breathing
- inability to latch on/nurse easily
- irritability and pain from gas (colic)
- inability or uneasy yawning
- earaches
- swallowing difficulty
- chewing problems.

Tongue freedom There may be a need for a frenectomy for a newborn or as soon as the restricted tongue movement is diagnosed. The 'tongue tied' condition is best addressed soon after birth but in any case, the sooner the better. If the tongue is 'tied' or 'tethered' too tightly to the floor of the mouth, low tongue posture will ensue (Gelb 1977). Low tongue posture inhibits maxillary growth both laterally and anteriorly, which reduces maxillary and cranial sinus size. This can also limit development of the nasal airway and pharyngeal airway which can sequentially lead to mouth breathing; more low tongue posture; excessive lower jaw growth; TMJ compression; and TMJ dysfunction (Hockel 1983). The use of the tongue then goes between the back teeth to act as a cushion (splint) to help take pressure off the TMJ and assist neuromuscular balance to the jaws, as well as the rest of the cranial mechanism (Hockel 1983).

This illustrates a cycle that has begun and will continue until corrective measures are taken (Gelb

1977). A simple frenectomy procedure can correct the 'tongue tied' condition and reduce future difficulties (Gelb 1994). This is best accomplished by a laser technique which reduces scar tissue formation and is nearly bloodless. The healing is further enhanced by the use of a low-level laser following the surgery (Tuner & Hode 1999). The author recommends 2×/day for 7–10 days for further healing and scar reduction.

Age 2–5

An examination by a cranially astute dentist trained in functional jaw orthopedics should be scheduled to evaluate posture, nasal breathing, arch form and jaw position (Gelb 1994).

Permanent teeth begin erupting between 5 and 6 years old so if the jaws are too small, the teeth will not have room to straighten. When small jaws are present it is not unusual to see one permanent tooth, in the lower front part of the mouth, displace two baby teeth as it forces its way into the jaw (Page 2003). The ideal arrangement in the 3–5 year old is to have 1–2 mm of space between all the front baby teeth because the permanent teeth are larger so they need more room than the baby teeth.

Age 5–12

Observing overall facial structure can provide a therapist with important clues as to historical growth patterns and possible undiagnosed dysfunctions (Zeines 2000).

- A 'long' face is one that seems too long and narrow, almost stretched out. This appearance may have been caused by allergies, mouth breathing, birth trauma, flaccid muscles of mastication, trauma or incorrect height of restorative treatments which have increased the vertical dimension by too great a degree (Enlow 1975, Gelb 1994, Mahoney et al 2003, Mew 1999).
- A 'short' face is noticed by an overall appearance of compressed facial features. This can be caused by clenching or grinding of the teeth secondary to trauma or emotional/psychological stress. Sacroiliac joint instability can also cause a TMJ clenching reflex to occur in an effort to stabilize the sacroiliac

joint (Enlow 1975, Feeley 1988, Frymann 1998, Gelb 1977).

IMPORTANCE OF MULTIDISCIPLINARY APPROACH

Multidisciplinary care can enhance the quality, efficiency, speed and effectiveness of cranial/TMJ/TMD treatment (Gelb 1971). Though more time and effort are required, the patient, therapist and doctor all benefit from the communication and joint treatment plans which result. In the words of Gelb (1977), a foremost author and proponent of these approaches, 'There is no place for intellectual isolationism in the holistic approach to the diagnosis and treatment of this clinical entity (TMJ dysfunction)'.

CONCLUSION

The strength of the cranial/dental connection cannot be overstressed. Without one or the other,

the best planned treatment can fail. It is this author's opinion that cranial therapy can have immediate positive effects on the general health and well-being of any individual. This especially includes those who are medically compromised. The author believes that cranial therapy is beneficial for all dental patients and should be included in most – if not all – dental regimens. That being said, he also believes that dentistry has a powerful effect (positive and negative) on the cranial mechanism and thus, can enhance or thwart the best efforts of the cranial therapist. The body needs to be viewed as an entire structure and the dental professional (dentist, orthodontist or oral surgeon) must be encouraged to understand and consider this interrelatedness. Only in that way can he or she truly appreciate the long-term impact their choice of treatment will have on their patient's overall health and welfare. It is this author's hope that through increased education and awareness, the health professions will make a concerted effort to utilize the information in this book.

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NOTE

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Incidence Levels and Chronic Health Effects Related to Cavitations



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What are Cavitations?

A cavitation is a hole in the bone, often where a tooth has been removed and the bone has not filled in properly. In the last several years, the term cavitation has been used to describe various bone lesions which appear both as empty holes in the jawbones and holes filled with dead bone and bone marrow(5). Dead, cavitational areas, which produce pain, are now called NICO (Neuralgia Inducing Osteonecrosis) lesions(6). Cavitations are often a result of either ischemic osteonecrosis, due to poor blood flow in the marrow, or a traumatic bone cyst. In his book on oral pathology, Dr. G.V. Black, one of the early experts on cavitations, suggested surgical removal of these dead bone areas. Other less traumatic measures are now first used and surgery with curetting is used primarily where the patient has significant health effects not resolved by other means. When a tooth is being extracted, in what has been normal dental procedure, the surrounding periodontal membrane is usually left behind. Theoretically, when a tooth has been pulled, the body will eventually fill in the space in the bone where the tooth once was. But when the membrane is left behind, an incomplete healing commonly takes place which leaves a hole or a spongy place inside the jaw bone. Experts speculate that perhaps this is because the bone cells on either side sense the presence of the periodontal membrane and "think" that the tooth is still there. This appears to be one common cause of cavitations. (1,3-5,16-22,29-32,45)

Odontodontic cysts are also commonly occurring usually in the gums at the tip of a tooth, that have pockets of bacterial infection that can cause inflammation and pain in some cases similar to cavitations (48,49,33). Bacterial infections are also known to have systemic effects.

A cavitation can form in any bone in the body, not just in the jaw bones. There are also other reasons that cavitations form, some of which are localized traumas, poor circulation to the area, clotting disorders, and the use of steroids.

On X-ray of an extracted tooth site, this membrane can form an image that appears to be a shadow of a tooth. Almost always, this is indicative of a cavitation. Most dentists are aware of this phantom tooth image, but they do not recognize it as a site of potential problems. Other means of locating or identifying cavitational areas include sonic imaging(CAVITAT)(3,68), local anesthesia, Spect Scan(65), pressure to determine trigger points, Computer Electro Dermal System(67), etc. While positive Spect Scans were found in 19 of 20 patients with jaw pain, several control patients with no pain also had positive scans- often finding previous jaw pathoses. Thus the Spect Scan was not sensitive at differentiating painful from non-painful conditions. Some of the other methods had more success at such differentiation.

What's hiding inside?

Inside a cavitation, bacteria flourish and deviant cells multiply. Cavitations act as a breeding ground for bacteria and their toxins. Research has shown these bacterial waste products to be extremely potent(7,8). Cavitations can also cause blockages on the body's energy meridians and can exert far-reaching impact on the overall system. Investigation has revealed that some cavitations are reservoirs of huge amounts of mercury and other toxic substances. Cavitations may be a source of low level or high level stress on the entire body. (1-73)

How toxic are cavitations and what type of effects are caused by cavitations?

The results of recent research of Dr. Boyd Haley (former Chairman, Department of Chemistry, University of Kentucky) show that ALL cavitation tissue samples he's tested contain toxins, which significantly inhibit one or more of the five basic body enzyme systems necessary in the production of energy(7,8). These toxins, which are most commonly likely to be metabolic waste products of anaerobic bacteria (bacteria which don't live in oxygen), may produce significant systemic effects, as well as play an important role in localized disease processes, which negatively affect the blood supply in the jawbone.

There are indications that other types of toxins also accumulate in cavitations, and when these toxins combine with certain chemicals or heavy metals (for example, mercury), much more potent toxins may form(5-15).

High levels of mercury are commonly found in some cavitations and in general in the jawbone of those with mercury amalgam fillings and to have significant local and systemic effects (79). Mercury is known to be extremely toxic and to commonly cause chronic adverse local and systemic health effects (70). Yeast and fungi have also been found to accumulate in cavitations, and to have significant systemic effects (10-14).

Accurate tests for cavitation related bacterial toxins have been developed by the Affinity Laboratory in Kentucky, based on research by Chemists from the Univ. of Kentucky Dept. of Chemistry (7,8). The

toxins released by anaerobic bacteria in cavitations have been found to be extremely toxic, and to have major effects on necessary body enzymes and the immune system.

Cavitations are Very Common

One study(1,20) of cavitation incidence involved an analysis of 112 randomly selected dental patient charts who had been tested for cavitations, with patient age ranging from 19 to 83 years among 40 \ males and 72 females. The cavitations were tested for using exploratory drilling. Cavitations were found at approximately 75% of all extraction sites examined.

The most commonly extracted teeth, the third molars ("wisdom teeth"), produced CVs that were found by clinical exploration in 313 out of 354 extraction sites (88%). Cavitations were found in 35 of 50 second molar extraction sites (70%), and for first molars, 60 of 73 extraction sites showed CVs (82%). They were found in 441 of the total number of 517 molar extraction sites explored (85%). For the maxillary non-molars, CVs were found in 72 of 123 extraction sites (58%), and for mandibular non-molars, 23 of 51 extraction sites were affected (45%). For all non-molars, the CV rate was 55%, representing 95 of 174 extraction sites. Note that the cavitations found were not all related to pain or known chronic conditions, and dental patients who had been tested for cavitations is not the same as the general population, so the general population likely has a somewhat lower cavitation incidence.

Bob Jones is the inventor of the CAVITAT -an ultrasound instrument designed to detect and image cavitations that has been approved for testing for cavitations by the FDA after undergoing FDA clinical trials (2b). He found cavitations of various sizes and severity in approximately 94% of several thousand wisdom teeth sites scanned(2a). He also found cavitations under or located near over 90% of root canal teeth scanned in both males and females of various ages from several different geographic areas of the United States. Note again that the population being tested for cavitations in these trials is not the same as the general population, which might have a somewhat lower incidence of cavitations. But its clear that the occurrence is very common.

Confirmation of cavitation necrosis and toxicity is commonly by 2 or the leading labs in the U.S. with technology for performing such tests, the Maxifillial Center in West Virginia and the Affinity Laboratory in Kentucky.(5-8). Analysis typically finds clear evidence of chronic intraosseous inflammation- often with dense marrow fibrosis or nonresorbing necrotic bone flakes with very little healing or new bond formation(6). It has also been found that these lesions often spread to other areas to initiate further cavities.

Root Canals and Cavitations

Research has demonstrated that virtually all root canals result in residual infection due to the imperfect seal that allows bacteria to penetrate. The most commonly used material in root canals is gutta percha, which is soaked with chloroform and heated. But when the chloroform evaporates and the gutta percha cools, there is significant shrinkage in all such root canal fillings, which allows entrance of bacteria(18-22,50). A condition that commonly occurs with root-canaled teeth is a radicular or periapical cyst or apical periodontitis, which is a pocket of bacterial inflammation that often forms in the gums at the tip of root-canaled teeth(48,49,52,53) due to bacteria inhabiting the tooth. These are the most common type of cysts that form in the gums and can also be a factor in formation of cavitations in the neighboring jawbone. Once established, nonmutans streptococci, enterococci and lactobacilli appear to survive commonly following endodontic root-canal treatment of teeth with clinical and radiographical signs of apical periodontitis (51). Large scale tests found cavitations under or located near approx. 90% of root canal teeth scanned in both males and females of various ages from several different geographic areas of the United States(2). The general population could be somewhat different from this sample as the sample was not a random sample. In tests of 745 randomly chosen root-canaled teeth at a dental school, done at least 1 year prior to test, 33% were found to have apical periodontitis(53).

The toxins given off by these bacteria are often even more toxic than mercury(7-10). The bacterial toxins from root-canaled teeth and associated cavitations can cause systemic diseases of the heart, kidney, uterus, immune, nervous and endocrine systems.

A useful and commonly used test to assess the cause of toxic related chronic health conditions is the urinary fractionated porphyrin test, which measures the degree that toxic exposures have blocked digestive enzymatic processes necessary to the function of the body, by looking at the level of various waste porphyrins in the urine caused by these blockages. The level of such toxic related porphyrins in the urine of people with chronic conditions including Parkinson's have been found to decline in some patients after cavitation treatment(or amalgam removal). (20). This is also been found for many cases of Lupus and MS(78,38). Lupus symptoms are often associated with blockage and resulting high levels in urine of Uroporphyrin, while MS is more commonly associated with high Coproporphyrin.

Cavitation Treatment usually results in significant pain improvement

Cavitations commonly cause adverse health effects, and many thousands of cavitations have been treated. They are commonly tested or biopsed by labs having the expertise to provide these services, and virtually all that have been tested or biopsed were found to be associated with dead, necrotic tissue and extreme toxicity(3,5-9). The types of conditions that cavitations have been most commonly related to are atypical facial neuralgia, trigeminal neuralgia, chronic sinusitis, phantom toothache pain, and headaches including migraines.

Dr. Briener, DDS, and others recommend two primary methods of treatment for their patients (40,54,33,etc.). First is a procedure where special homeopathic medications called Sanum remedies are injected into the cavitation site, and then a modified form of infrared light or low level laser light therapy is applied to the area. In some cases the light therapy alone has been sufficient to resolve the problem(54). This is often successful in cases related to smaller cavitations with primarily poor blood flow or bacterial toxin effects . Cavitations have also been treated successfully using oxygen/ozone therapy(74). Although cavitations are very common, they should only be treated surgically if there is

indication of a relation to pain or chronic health effects not resolved by other means. There are various ways to assess this.

If this method is not successful, the alternative is to surgically open the area and clean the remaining ligament and resultant debris from the bone. Every biopsy of bone material he has collected from cavitation surgeries has shown osteonecrosis, or dead bone material. In all studies reviewed, the majority of those undergoing surgery for NICO pain had significant pain relief after surgery(3-Table1,40,42-45,55-63,70,71,etc.). Clinical experience indicates that delays in treatment can lead to further infections(44), and the majority of patients have long term pain relief(45). However as much as 30% may have reoccurrence or new cavitations that lead to reoccurrence of pain. Prior to bone marrow biopsy the average NICO patient has been in pain for 6 years (up to 32 years), usually diagnosed as atypical facial neuralgia/pain, but also diagnosed as trigeminal neuralgia, chronic sinusitis, phantom toothache/pain, and various headaches, including migraine headache(3). However treatment has also been successful at eliminating rheumatoid arthritic pain(43,18,26,27).

French and German oral surgeons have developed an alternative method of minimally invasive cavitation surgery (41).

Due to the nature of the mechanisms related to cavitation formation, it is not uncommon for cavitation sites that are treated to become reinfected or to accumulate other toxins that can cause a relapse of symptoms. Such cases may require retreatment using either surgery or other options.

Chronic health conditions other than pain related to cavitations and oral bacteria levels

Many researchers today believe that NICO lesions, like periodontal disease, is the focus of various infections which may spread throughout the body and have systemic effects. In the last few years, some of the most surprising medical news has been the discovery that bacteria from the mouth appear to be very influential in causing various heart, liver, kidney, and immune problems(68).

Researchers from New York University found that certain bacteria from the mouth may be related to preterm delivery and low birth weight according to a study in the Journal of Periodontology (JOP). (68b) The presence of specific bacteria and combinations of bacteria in periodontal pockets also appears to be responsible for the relationship between periodontal disease and acute coronary syndrome (ACS), according to a new study published in the Journal of Periodontology (68c).

Dr. Weston Price was a prominent dental researcher leading a medical research team on the relation between root-canal teeth and chronic health conditions(70). Through a long series of well documented clinical cases and experiments his team found that root-canals accumulate bacteria that give off extreme toxins sufficient to cause serious health conditions, including cancer(22,25,28,29,36,37,38,46,47,70,etc.), cardiovascular conditions(19-22,29,36,38,70,72,73), arthritis (22,27,29,36,38,43,70,etc.), neurological conditions (3,5,42,56,70-72,etc.) kidney conditions, etc. Dr. Meinig, one of the founders of the endodontic association has reviewed the research of Dr. Price and others and is in agreement with their findings(18,20,26).

Many doctors and dentists through their experience with patients have reached similar conclusions (18-26,28,29,32-39,45,54-65,71,73). They have had large numbers of patients who have had such health conditions significantly improve after treatment of root canals or cavitations along with other detoxification measures. A collaborative study by the North Carolina Institute of technology using advanced tests developed by Affinity Laboratory has demonstrated the mechanisms by which cavitations can cause cancer(47).

Modern experiences also support this theory. Dr. Issels, a German physician, recommends extraction of root canal teeth as part of his protocol for terminal cancer patients. Over the last 40 years with 16,000 patients, he has observed a 24% total remission rate(46,25).

Dr. Florian Kubitzek, a physician and dentist in Munich, Germany, uses the CT scan to study the teeth and jaw. His scanning technique has been invaluable in diagnosing jaw abscesses below the teeth that have been inadequately treated by standard dentistry. Conventional dental X-rays have entirely missed the jaw abscesses known as cavitations. Kubitzek treats many cancer patients who have dental cavitations as a collaborative approach in the overall treatment of metastatic and primary cancer(37).

Dr. John Diamond(MD) says that all patients with breast cancer that he has tested had root canals on the tooth related to the breast area on the associated energy meridian." (25)

Other clinics that treat cancer have similarly found that most of their patients with cancer have root-canaled teeth or cavitations and that treating these is an important part in success at treating cancer (38,39)

Research and clinical cases have found cavitations to be related to many chronic health conditions which have improved after cavitation treatment, including cancer, congestive heart failure and other cardiovascular problems, lupus, rheumatoid arthritis, and autoimmune conditions- perhaps related to cavitations major effects on the immune system.

If you have a joint implant or mitral valve prolapse, your dentist must prescribe an antibiotic before any dental treatment. Why? Because bacteria from the mouth can spread through the blood to cause serious problems elsewhere in the body. There is growing evidence that the toxins from NICO lesions do the same.

Trigeminal Neuralgia, one of the most severe painful conditions which occurs to man and women, many other face and jaw neuralgic illnesses, have for the most part, had unknown causes until recently. The discovery of jaw bone infections (cavitations lesion) is proving an important reason for the occurrence of high percentage of these diseases. Their correction has achieved an impressive cure rate. In view of the subject matter of the Root Canal Cover-up, the fact of Root filled teeth being cause of these cavitations infections should be of particular interest

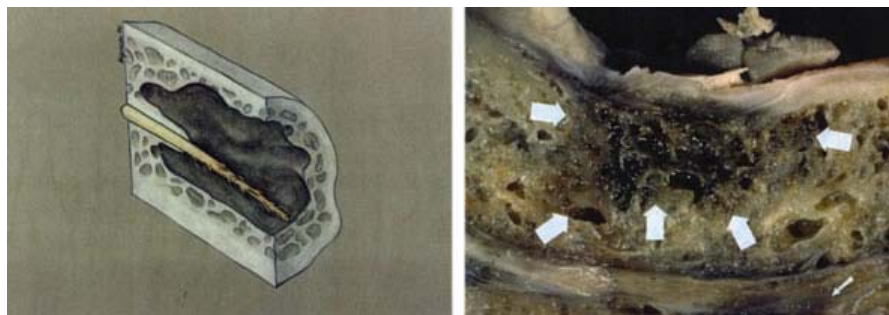


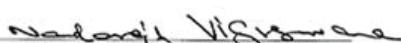
Photo from J.E. Bouquot D.D.S figure top drawing of atypical cavitating NICO lesion of the mandible shows an irregular outline, darkened walls, destruction of the inferior alveolar canal, and a frayed rope appearance to the exposed neurovascular bundle . Figure bottom . A large NICO lesions is seen in the mandibular molar region (large arrows) . The small arrows point to a less severely involved second site of osteonecrosis just above the inferior border and involving the inferior alveolar nerve.

A cavitation refers to a toxin-containing hole in the jawbone, often at the site of a previously extracted tooth. Cavitations have many scientific names such as ischemic osteonecrosis, chronic non-suppurative osteomyelitis, and neuralgia inducing cavitation osteonecrosis (NICO). This is not so much an infection in the bone, as a necrosis or gangrene (dead tissue) in the bone marrow, as a result of impaired blood flow (ischemia). A cavitation often develops because of incomplete healing after routine extraction. The contents of cavitations are always necrotic, dead or dying material. The microscopic picture looks the same as gangrene! If a gangrenous extremity is not amputated, the rest of the body will sicken and die, due to a high concentration of anaerobic bacterial toxins.

Incomplete healing of a cavitation is promoted by a number of factors, including the following:

- Failure to completely remove the periodontal ligament lining the tooth socket that holds the tooth to the bone
- Physically large surgical excavations, such as with impacted wisdom teeth, where the resulting holes can be expected to be larger than usual and more new bone is required to fill the holes
- Failure to clean out the infected adjacent bone and periodontal ligaments seen in the extraction of root canal treated and abscessed teeth
- Failure to remove condensing osteitis, the reactive bone formation that attempts to wall off infection, usually involving the periodontal ligament as well
- Poor systemic healing support from a compromised immune system
- Poor nutrition and a weak thyroid gland.
- Failure to allow the formation of a complete blood clot at the excavation site; too early dislodgment of a clot after extraction; also bleeding
- Smoking
- Antibiotic therapy
- Chronic osteoporosis of the jawbone
- Systemic and adjacent toxicity from other dental toxins and other sources
- Pre-existing periodontal disease, in addition to any other factor that would also promote periodontal disease

One of the primary factors in cavitation formation seems to be that the initial extraction does not include the thorough removal of the periodontal ligament from the socket after the tooth is removed. Unfortunately, this inadequate socket cleaning is the routine procedure with most extractions. Cavitation formation after tooth extraction is the rule and not the exception; yet, the condition is still largely unknown to most of dentistry, and underestimated by those who are aware of it. A cavitation can be expected to form when the socket lining separating the tooth from the bone is not thoroughly removed. A thorough removal of the ligament requires that a portion of the bony socket be removed as well.

| | |
|---|--|
| Oral & Maxillofacial Diagnostics <i>A Biopsy Service of the University of Texas Dental Branch at Houston</i> Department of Diagnostic Sciences, Room 3.094f, 6516 M.D. Anderson Blvd., Houston, TX 77030 Phone: 713-500-4406; Fax: 713-500-4416; Director: Jerry.Bouquet@uth.tmc.edu | |
| BIOPSY REPORT #UTDB 2006 - 1586 | |
| Surgery date: 04-07-06 Date received: 04-12-06 Date completed: 05-03-06 | |
| SURGEON: Corinne Vizcarra, D.D.S. 4453 Benfield Ct. San Diego, CA 92113 | PATIENT: LOWELL, ROB 6385 Janton Way Cumming, GA 30040 |
| AGE (47Yrs.):/ GENDER: M DATE OF BIRTH: 11-09-1958 | |
| SOURCE OF SPECIMEN (location): Area #1, #32 CLINICAL DIAGNOSIS/DESCRIPTION: Residual osteitis | |
| GROSS DESCRIPTION OF TISSUE RECEIVED: The specimen was received in a container of formalin and consists of multiple, small, irregularly shaped, tan to white fragments of soft and hard tissue measuring in aggregate 0.9 x 0.5 x 0.3 cm. The specimen is submitted in total pending decalcification. | |
| MICROSCOPIC DESCRIPTION OF TISSUE: Sections show very thin spicules of partially viable and focally non-viable bony trabeculae with minimal amounts of fatty marrow attached. The fatty marrow shows hemorrhage and a mild degree of ischemic myelofibrosis, plasmotosis and vascular congestion. | |
| MICROSCOPIC DIAGNOSIS: Osteoporotic marrow defect with localized ischemic marrow damage, posterior maxilla and mandible. | |
| PATHOLOGIST:  J.E. Bouquet DDS, MSD; N. Vigneswaran DDS, DMD; C. Flaitz DDS, MS | |
| NV:zh 305.311 526.2 | |

One purpose of this ligament is to give a certain amount of natural shock absorption to the tooth. Without it, chewing would be much like riding on rims rather than tires. When the periodontal ligament is not thoroughly removed from the socket after the extraction, the surrounding bone receives no notification that the tooth is gone. The continued presence of any portion of the ligament gives the biological message to the surrounding jawbone that all is well, and no new bone growth is needed. Bone cells are not going to start new growth and then migrate through a barrier naturally designed to limit such growth. The jawbone determines that if the ligament is still there, the tooth must be there as well.

Since the periodontal ligament does not extend to the upper edge of the extraction site, new bone growth activity will not be inhibited at the top of the socket, and a characteristic thin cap of bone will eventually extend over the extraction hole. Larger cavitations often have only a cap of gingival, or gum tissue, over them. Even the thin overlying cap of bone does not form in these cases. In routine dental extraction, portions of the periodontal ligament will sometimes remain more strongly attached to the tooth than the bone and be removed along with the tooth. When partially removed in this fashion, the spotty absence of the ligament will permit equally spotty growth of bone, resulting in the wide variety of cavitation shapes and sizes.

Even when large wisdom teeth are removed surgically from impacted sites with extensive excavation, cavitations are nearly always present. When the excavated hole is large enough, cavitation formation can be anticipated, even if most, or all, of the periodontal ligament is removed, since so much more new bone growth is needed to completely fill the hole. Condensing osteitis must be completely removed to give the opportunity for complete healing. Solid, healthy bone must be reached to allow the normal regeneration of bone. When infection or necrosis remain throughout the socket and adjacent bone, with or without condensing osteitis, healing will rarely ever be completed.

Blood clot formation, with its gradual retraction over time as the surrounding tissue heals in, is nature's way of promoting proper healing throughout the body, not just in the mouth. When a nicely formed blood clot fills the socket, healing gets a good start. But when it is dislodged early, or adjacent periodontal disease or smoking causes too rapid a retraction, a dry socket is the result, and cavitation formation can then be anticipated.

Preexisting diseases, such as osteoporosis with poor bone structure in the jawbone before the extraction, can clearly promote the formation of cavitations. Bleeding disorders, which would directly impair the formation of the important blood clot, can also be promoting factors. Periodontal disease can also serve to more easily infect the freshly extracted sites and bathe them in the toxins produced by the anaerobic bacteria trapped in the diseased gums. The presence of local and systemic toxins will also impair the healing process anywhere in the body. The presence of toxins such as heavy metals will chronically disrupt the

calcium/phosphorus balance in the body, promoting the continuous mobilization of calcium from the bone into the tissues and into the urine. Any healing bone needs more bioavailable calcium, not its removal.

The procedure to clean a cavitation has often involved use of a blind approach, due to a lack of reliable diagnostic tools. Consequently, dentists have missed smaller and unusually located cavitations. Even a larger cavitation could be missed by the wrong angle of attack by failing to explore the one cusp site that had cavitated. Cavitations will also interconnect and form channels in the jawbone.

An explored channel might be counted as only one cavitation when it actually developed from more than one unhealed extraction site. Many smaller cavitations will never be found because the operator may not opt to explore a smaller area between the teeth on either side of the old extraction site. Larger cavitations can extend below the mandibular nerve. Such large cavitations allow the toxins more access to the rest of the body by utilizing the mandibular and other nerve channels. Most dentists seem to convince themselves that if a cavitation cannot be seen on x-ray, it must not be there. However, a cavitation characteristically cannot be seen on x-ray. While some cavitations can be clearly visualized on a panoramic x-ray, by those who are trained to identify them, the vast majority of cavitations, even large ones, will be completely missed on a careful examination of the x-rays.

Only a cavitation that has formed with additional calcification around a well-defined border will be visualized on X-ray. The cavitation officially forms when healing at the top is complete. The healing over on top allows the rapid development of an oxygen-deprived, or anaerobic, state in the hole. Native mouth bacteria produce highly toxic metabolic by-products when deprived of oxygen. Bacteria that are normally harmless to man when oxygen is present form a deadly toxin when oxygen is removed. An example of deadly toxins forming when oxygen is absent is the botulism toxin, resulting from vacuum-packed foods, sealed with bacteria present. Some toxins found at cavitation sites are up to 1,000 times more toxic than botulism in their effects on enzymes systems.

Antibiotics will not help the individual poisoned with botulism, nor will they help the cavitation patients, as the problem stems primarily from bacterial toxins, not bacteria. Only a rapid neutralization of a large toxin dose will save the patient. Toxins in both cavitations and root canal filled teeth rapidly kill vital human enzymes at the lowest imaginable concentrations. Cavitation toxicity tends to be cumulative. The more cavitations you have, the more toxicity is present. Cavitations also may make other diseases worse. They will make it more difficult for a compromised immune system to ever completely recover. The immune system characteristically tolerates all stresses fairly well until it collapses relatively suddenly. It will compensate as long as possible, then very suddenly its defenses will no longer be effective. Cavitations might not be the cause of an illness, but they can easily be the factor that prevents recovery from it.

Sonic Imaging

There is a new technology that has been developed that can image the jawbone using unfocused ultrasound that provides a color-coded three dimensional representation of the density of the bone and shows loss of bone or ischemic dying bone tissue that is usually not seen on x-ray imaging. This technology, called a Cavitat, has been approved by the FDA and is now available. It has an incredibly high accuracy record in the findings of a correctly performed scan. This can be done chair-side at a dental facility so equipped. Check out the Cavitat website at www.dentalhelp.org. They have references for dentists who have cavitat devices, as well as other educational materials, etc.

Treatment

Surgery is often necessary to properly and thoroughly clean out a cavitation site, for there is no other way to remove dead bone. The key to bone healing and regeneration is through removal of necrosis and support of the thyroid gland, especially by using detoxified iodine. Neither injections into the bone with homeopathic remedies or laser light treatments will work to stop the progressive necrosis. If necrotic tissue is not thoroughly removed, the necrosis will spread and cause more destruction to the bone, nerves and blood vessels. This kills teeth in the process, for they are cut off from their blood supply. After cleaning out the necrotic bone, healing can then take place and new bone cells will fill in the cavitations. Unfortunately, there are only a handful of dental surgeons today who do a proper job of thoroughly cleaning out the diseased bone tissue.

Dr. Wesley Shankland has written a very comprehensive book for individuals who suffer with TMJ and related disorders. TMJ: Its Many Faces, 2nd Edition, published by Anadem Publishing, has drawn on Dr. Shankland's experience of over 23 years of treating patients from all over North America and around the world. These patients have seen, on average, six other health professionals prior to seeing Dr. Shankland. While many of these patients have been told that their TMJ problem is a result of stress, Dr. Shankland has found that nearly 100% of these patients do have physiological and/or anatomical reasons for their pain. This book discusses how the patient can become actively involved in his or her treatment. Included are complete instructions and diagrams of exercises. Lifestyle changes are outlined and a chart of dietary modifications are contained within the book. An entire chapter is devoted to choosing and evaluating a TMJ doctor.

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Cavitations & Root Fillings.

A cavitation as described here is not generally recognised by conventional medicine. However they exist. They were first described by the father of modern dentistry, Dr G V Black, over 100 years ago. Uncomfortable facts take time to be accepted.

Cavitations or NICO's occur when bone is deprived of its blood supply and dies. When the bone dies a hole in the bone develops, literally a cavity and into this hole migrate anaerobic bacteria. These bacteria live without oxygen, indeed oxygen is poisonous to them. Bacteria organise themselves into colonies which can be visualised as cities. Cities require food to come in (you the patient are the food!) and generate waste material. The waste material made by these bacteria is toxic in the extreme and in cavitations this toxic material is constantly being released into the body.

If Mustard Gas, used in WW1, is taken as a yard stick, then most of the bacterial waste products are 10 or more times as toxic than Mustard Gas.

Cavitations can occur in any bone in the body usually after infection or trauma of some sort. Naturally dental extraction can easily be the cause of a cavitation, this is especially true of wisdom teeth extractions. Most extractions are due to infection, they involve stretching and sometimes fracture of the bone, all predisposing factors for cavitation formation. Added to this is the fact that if the membrane that holds the tooth in place, the periodontal membrane, is not removed at the same time as the tooth comes out, this leads to cavitation formation. It is not common for the dentist removing the tooth to also remove the membrane at the same time, an unfortunate fact.

Cavitations can be symptomless in situ causing problems at other places in the body. They can also be intensely painful causing severe crippling pain similar to a very bad unremitting toothache or neuralgic pain. When this happens the cavitation is called a NICO or Neuralgia Inducing Cavitational Osteitis.

Not all extractions lead to cavitation formation. Just because you have a cavitation does not mean it must be treated. The decision as to whether to treat a cavitation depends on the size of the cavitation and the symptoms displayed by the patient. This must be assessed on an individual basis.

Diagnosis:

To find out if you have cavitations is not easy. Cavitations cannot be accurately diagnosed by normal X-rays. Normal X-rays show only an indication if a cavitation is present, however a CAT Scan does show cavitations but only does this accurately if all the metals in the mouth are removed before the CAT Scan is done. However, this diagnostic method requires high dose radiation exposure, specialist facilities and is fairly expensive.

Cavitat is an ultra sound device designed for detecting chronic bone infections like cavitation infections. It is highly accurate in skilled hands and needs to be read in conjunction with a good OPG x-ray. The Cavitat can show in 3D the position and size of Cavitation infections.

Our experience over the years has shown the Cavitat to be extremely accurate. It has never given a false positive reading. Occasionally it can miss a cavitation but local factors can come into play here. We are very pleased with the results of our Cavitat Machine.

A simple but not so reliable method of detecting cavitations is to place a finger and thumb on either side of the suspected cavitation and squeeze firmly. If pain is experienced then most likely this is a cavitation. As all cavitations are fluid filled, squeezing the bone increases the hydrostatic pressure inside the cavitation causing pain.

Neural therapy can often give an indication of the presence of a cavitation infection. A tiny single drop of local anaesthetic without adrenaline is placed over the suspected cavitation site. The symptoms associated with the cavitation can then subside for a short while. If the symptoms persist, a further drop of anaesthetic is injected usually starting from back of the mouth to the front until the site is found. This method, whilst good, is not 100% diagnostically correct. With suspected NICOs it is extremely accurate. The key is to use a tiny drop near the tooth apex. Once again experience is the key.

When local anaesthetics do not work, a reason is a cavitation infection near the injection site which can neutralise the local anaesthetic

NICO's:

These are Neuralgia Inducing Cavitational Osteitis lesions.

They are the cause of acute disabling mystery facial pain. Quite often mistaken for Trigeminal Neuralgia, they seem to defy traditional logic. The patient is sure that a tooth is to blame but when the tooth is extracted, the pain moves to

another tooth or area. The patient can then lose many healthy teeth as the dentist searches for the cause of the pain. The pain is often referred to another part of the mouth some distance from the NICO site which further confusing the situation. NICO's arise commonly following a "routine extractions" or trauma such as whiplash. The conventional treatment is antidepressants which do partially suppress the symptoms but at a cost. They are no long term solution. The best diagnostic tools are the Cavitat and Neural Therapy. See below for a case history.

Treatment:

Cavitations do not respond to antibiotics. The only effective treatment is to open them up and clean them out thoroughly. This means an operation.

We treat cavitations in a variety of ways, an old fashioned way is exactly as infections were handled before the advent of antibiotics. This is by opening and cleaning the cavitation then keeping the hole open with a dressing and changing the dressing daily. This has two purposes. The first is to allow oxygen into the cavitation to kill the bacteria and secondly if the wound is sewn together then there is a blood filled cavity ideal for re colonisation by bacteria. This allows a new cavitation to start. Keeping the wound open stops this and allows healing from the bottom up stopping new cavitation formation. However, this does lead to a loss of bone after healing sometimes creating a dip in the bone. Another disadvantage is the possible retention of the dressing in the surgical site. If all the dressings are not counted in and counted out, some may remain. This can happen when the patient is at home dressing the site alone or with the help of a family member. The gum grows over the dressing if it is left behind eventually requiring more extensive surgery to remove all the fibres.

The dressing should be sterile surgical cotton soaked in a mixture of Iodine, Oil of Cloves and Hyaluronic Acid. Iodine is fatal to bacteria and bacteria cannot become resistant to it unlike antibiotics. This is due to the unique biochemistry of Iodine. Oil of Cloves produces instant soothing in the operation site. Hyaluronic acid is the substance found in a baby's skin that makes it soft and supple. When applied to bone it encourages new blood vessels to grow through and over the bone in a matter of days. when the bone is covered with new blood vessels, it resembles the velvet on the new horns of a deer, infection cannot re-establish itself.

Our preferred method today is to clean and wash the site as above but with the addition of magnesium chloride. Magnesium Chloride 5% solution has proved to be of enormous significance in accelerating post operative healing. Magnesium Chloride was first used by the French Army in the first world war as wound irrigant. Subsequent research showed that it stimulated the immune system, was anti bacterial, anti fungal and anti viral as well. So now after cleaning the cavitation site as before, we irrigate the bone with magnesium chloride many times. The magnesium chloride solution is ozonated by bubbling ozone through the solution for 30 minutes before use. In addition we flood the whole area with ozone gas. Ozone is in a league of its own in killing the anaerobic bacteria that cause the cavitation infection. The hole is then filled with an ozone gel and sewn up. This method has proven to be remarkably effective with added advantages to the patient. These are no dressing changes which can be painful and time consuming. Bleeding is better controlled with the ozone and the loss of bone is markedly reduced. However, some cavitations are best treated the old fashioned way with dressings. which method we choose is down to our considerable experience in treating cavitations.

The bone removed from a cavitation can be tested to see how toxic it is but the individual toxins cannot be identified as yet.

This can be read about in more detail on Bioscience.COM.

Case Histories: All patients treated at the Munro-Hall Clinic

Resolution of Chronic Facial Pain

Case History and Discussion.

This describes a case of the alleviation of chronic facial pain of over 18 year's duration.

This is new technology revisiting the past, of ultrasound Cavitat Scans showing focal infections in alveolar bone and how it was successfully dealt with.

The patient was a 55 year old female with a history of chronic facial pain emanating from the upper left edentulous maxilla. The pain was severe and constant and centred in the maxillary arch. It radiated to the left eye socket and over the left temporal region.

The patient had gone from dentist to doctor to oral surgeon eventually ending up at a specialised dental post graduate centre

X-rays and a battery of blood and other tests had all been negative. She was given no positive diagnosis other than the pain was probably of psychological origin. The treatment offered was tranquilisers and amitriptyline antidepressants to be taken daily.

The medications reduced the severity of the pain but did not eliminate it.

When we saw her, she had been on this regime for 18 years. The side effects of the medications were now quite prominent. They included weight gain due to fluid retention, pain in joints and a totally disrupted bowel function. In addition she had psoriasis over the back and legs rendering any supine position extremely uncomfortable for the last 6 years. None of these symptoms had responded to drug treatment.

Examination and history taking showed routine dental treatment including a root filling on the right side and a number of amalgam fillings. The upper left maxilla had no teeth behind the second premolar. These teeth had been removed some 25+ years previously for unknown reasons. There was no sign of inflammation, the perio condition was good, there were mild muscle and joint symptoms associated with a minor TMJ dysfunction but no pain or limitation of movement of the mandible. The extracted teeth had never been replaced.

Pressure by thumb and forefinger over the ridge around the second molar area elicited some pain. A panorex radiograph showed a radiolucent area in the same region. This is unusual but illustrative which is why we are using this particular case. More often than not the radiographic result is read as normal (however, experienced eyes see things differently).

An ultra sound scan was done of the whole mouth using the Cavitat scanner. This showed in 2D and 3D that there was a cavitation space in the area of the pain and radiolucency. Administration of a small amount of local anaesthetic without adrenaline in the area eliminated, albeit temporarily, the pain.

These results confirmed the diagnosis of a bony cavitation infection in the pain producing area.

This is a NICO infection or a Neuralgia Inducing Cavitational Osteitis or a Ratner bony defect.

Cavitation infections are unresponsive to antibiotics. Only surgical debridement can bring about resolution of these infections. The causal organisms are anaerobic and the best consistent results in our experience are to open, thoroughly clean the bone and flood the site with oxygen.

The patient being assessed as suitable for outpatient surgery a decision was made to surgically clean out the cavitation area.

A flap was drawn back exposing the bone under local anaesthetic of reduced adrenaline content. On all cavitations infections there is a fibrous knot of tissue directly above it. This makes retraction of the flap more difficult especially in thin friable areas but does pinpoint the entry point to the cavitation quite precisely.

When the flap was retracted the hole in the bone was immediately seen at the second molar region. At no point throughout the procedure were burs or any rotary instruments used. All procedures were performed with hand instruments only.

Using a Hemmingway spoon curettes, soft bone was removed from the ridge opening the cavitation from the wisdom tooth area to just behind the second premolar. Probing showed a hole in the bone about 1cm deep and the entire width of the ridge. All the inner bony surfaces of the cavitation were soft as is usual. Slow and thorough hand debridement followed until no softness was left. The contents of a cavitation are of a slimy jelly nature with a characteristic stale Smokey smell and dark almost black in colour. In a large cavitation such as this, the centre is hollow and the contents adhere to the bony walls. Painstaking thoroughness is required in this type of surgery. Frequent and copious irrigation around the bone follows. This is with a 3 to 5% magnesium chloride aqueous solution ozonated prior to use for 30 minutes with 67ppm ozone. We use 10ml syringes and at least 6 syringes are used. Ozone gas, 20 ml also at 67ppm ozone was also put into the operation site using strict measures to prevent inhalation. The base of the cavitation was filled with ozonated olive oil and the entry to the cavitation filled with a thick sticky erythromycin gel. The site was firmly and tightly closed using resorbable sutures.

The erythromycin gel is made for us by a compounding pharmacist. Its use is not only a topical antibiotic but it "plugs" the hole. Ozone gel, an excellent bactericidal, becomes runny at body temperature and the erythromycin gel prevents this ozone gel running out. Ozone gel tastes particularly disgusting. Blood flows into this mixture nicely and a simple pressure pack controls bleeding.

The patient underwent intravenous infusions of 35g Vitamin C during the operation and for another 3 consecutive days afterwards to control infection and accelerate healing.

The Vitamin C was diluted in Lactated Ringers 1:4, and infused over three hours. The amount of Vitamin C used is dependant upon the weight of the patient.

The healing result was excellent as seen by the photograph of the operation site 7 days later.

The operation was performed in April this year. The chronic pain diminished substantially directly after the operation. Within three weeks the patient had reduced the medication by half, and was off all medication within eight weeks. She did this on her own against our advice of seeking medical supervision.

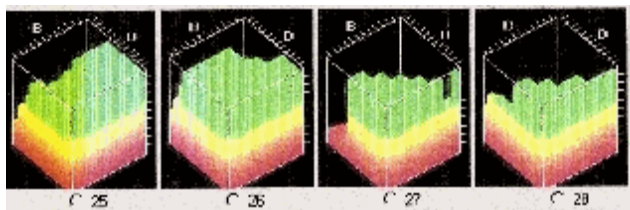
The chronic facial pain of 18 years duration had entirely resolved within two months. The pain in the joints has also gone and the bowels are now functioning normally. The psoriasis has been reduced markedly and the patient is no longer taking any prescription medication. A rescan some six months later show new bone growth at the operation

site.

No antibiotics were needed or taken orally before or after the operation.

In her words "I have not felt so well for years".

She has now stopped all medication and is without facial pain or discomfort.



Cavitat Scan of maxillary upper left area. Green is normal tissue, black areas show the cavitation

Discussion:

The theory of focal infection, out of fashion now in dental circles but still accepted by veterinarians, held sway in the profession before the advent of readily available antibiotics. In our opinion the invention of the Cavitat Scanner, an FDA approved device, is the most significant development in dentistry since the advent of the high speed turbine. It shows with unerring accuracy in experienced hands, the location and size of these chronic infections.

The terms cavity and cavitation were used by an orthopaedic researcher in the 1930s, the former referred to holes in teeth and the latter to holes in bone. Cavitations are brought about by infarctions of the supplying blood vessels to the bone. They are seen most often in extracted wisdom teeth areas.

The father of modern dentistry, Dr G V Black described cavitations in his textbook on Oral Surgery in 1915 where he recommended surgical removal of these dead and infected areas.

They have been accurately described by a medical Professor, Dr Fischer.

This case was a classic NICO. Some of the time, cavitation infections are painless. Some patients have a shooting pain that can be confused with Trigeminal Neuralgia, others have a constant foul sour taste from drainage into the mouth from a cavitation. Cavitations are infected with anaerobic bacteria which release exotoxins. Research from Prof. Boyd Haley has shown that these toxins are very potent at inhibiting various enzyme systems. Not only this, when these toxins combine with heavy metals, e.g. mercury, even more potent toxins are produced. The control for toxin potency is Mustard Gas. These combined toxins are frequently more than ten times toxic than Mustard Gas. These bacterial products have been indicated in blood vessel dysfunction such as occluded coronary arteries and strokes.

This is entering a contentious area but it would explain the focal infection theory and is born out in clinical practice where we see a variety of medical conditions and symptoms resolve after successful cavitation cleaning.

The bacteria produce a slime which insulates the bacteria from the immune system and any blood born antibiotics and oxygen. The aim of surgery is to physically remove as much of the infective material as possible by curettage instruments and allow oxygen in. We prefer not to use rotary instruments if possible as this may lead to microscopic fractures in the bone that bacteria can access.

As cavitations are caused by interruptions of blood supply, infarctions, it would naturally follow that cavitations are seen more frequently in the older age groups. However, we have seen 20 year olds with cavitations. Other predisposing factors are smoking, long term cortisone use, oestrogen replacement therapy and reduced thyroid function.

Initiating factors are always trauma to bone of various descriptions such as extractions, ID injections, root canals, bruxism, periodontal infections, tooth apical infections or orthodontic treatment. Injuries from falling off bikes and horses, whiplash and even falling drunk onto a taxi door twelve years previously have also caused cavitations we have treated.

Ozonated magnesium chloride, 3% solution, is used as a rinse. Prof. Pierre Delbet used Magnesium Chloride in treating wounds during World War 1. He found increased leucocyte activity and phagocytosis reduced the incidence of post surgical infection using this rinse. We have added ozone to it as an extra measure against anaerobic bacteria.

The bacteria are so well established that every reasonable measure must be employed to defeat them, hence the use of intravenous vitamin c, ozonated olive oil and the erythromycin gel. Experience over the years has found this rinse to be superior to any others we have tried in post operative infection control and trouble free healing.

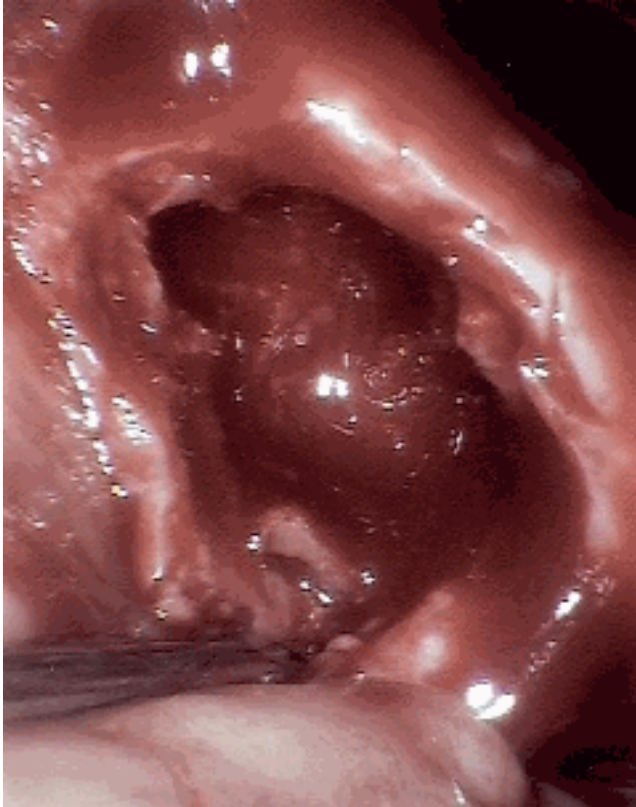
We use systemic oral antibiotics if post operative soft tissue infection occurs and occasionally if a "dry socket" type of infection establishes itself in the bone. Both are extremely rare occurrences.

In the beginning we routinely used systemic antibiotics after surgery but ran into problems with sensitive patients reacting to the antibiotics. Discontinuing the prophylactic use of antibiotics has not resulted in an increase in infection. Intravenous Vitamin C is an effective measure against all types of infection. We have performed close to 10,000

Vitamin C infusions over the years and found that this particular technique to be both safe and effective. Other diagnostic techniques are MRI and CT scans. MRI scans can be hard to interpret and CT scans can be inaccurate if metal restorations are present as the x-rays are scattered by the metals making the computer analysis suspect. Pre Cavitat Scanner, CT scans were our diagnostic measure of choice but the potential inaccuracies plus the amount of radiation used means we no longer use this form of scan. Success is measured by symptom relief by the patient and new bone growth in the operation site seen by a Cavitat Scan some 6+ months later. The re-occurrence of a cavitation infection after surgery has traditionally been around 30%, using the techniques described above, we have managed to reduce this to just over 10%.

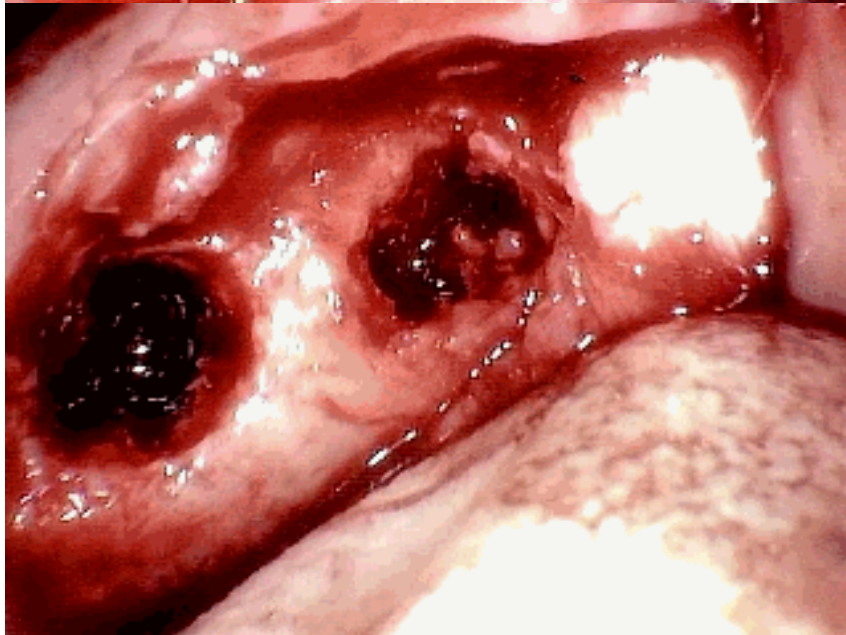
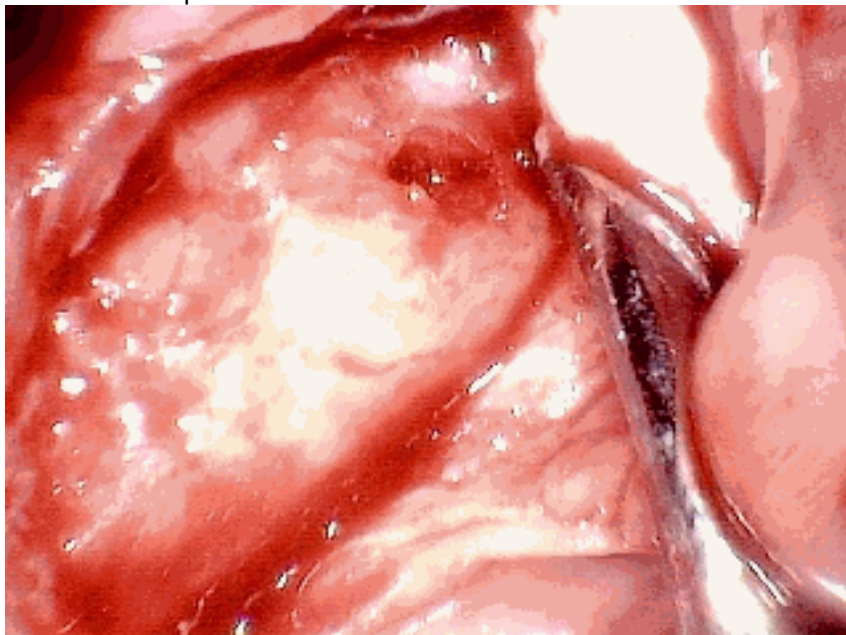


OPG. Note dark distinct area upper left. Such a picture is unusual in these cases.

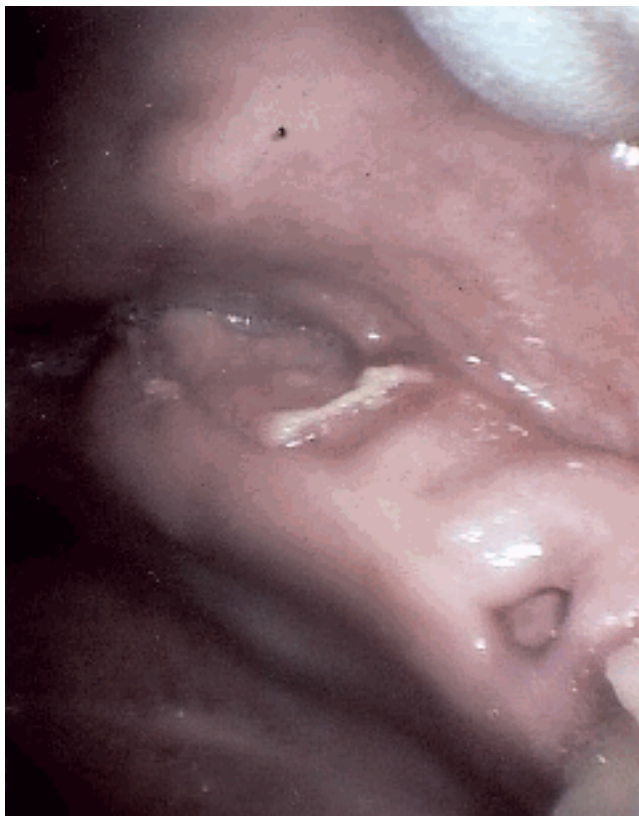


Fully opened cavitation. Only hand instruments used. No drills were needed, the bone was soft and mushy and was spooned out

Soft bone on top of maxilla removed



Bone exposed on flap retraction. Note the open defect in the bone some 20+ years after the extractions. The defect was directly under the fibrous attachment.



Area after 7 days. Note accelerating healing response typical of patients undergoing Hall V-Tox Therapy.

Root Fillings

If the nerve inside a tooth has died then the tooth may need a Root Filling.

A root filling is simply material put into a tooth where the nerve used to be. Traditionally a rubber like substance called Gutta Percha is used. This can contain mercury. Other materials include Formaldehyde, Cadmium, Steroids or even concentrated Sulphuric Acid.

The problem with root canals is both the toxic nature of the materials used and the fact that they do not fill fully the open spaces inside the tooth. Each tooth contained miles, and I mean miles, of little canals branching off the main canal. The main canal can be filled but traditional root filling materials cannot fill the tiny lateral canals. It is inside these lateral canals that anaerobic bacteria live and multiply. They too have waste products and it is the release of these toxic waste products, as in cavitations, that cause the problems.

Not all root fillings done conventionally are toxic. Just 90% of conventional root fillings tested at the clinic.

Diagnosis

A simple reliable test is available to show if a root filled tooth is toxic or not. This is the TOPAS test. The Topas test can be done immediately and the result known. A more precise test involving sending a paper point soaked in the fluid found around the neck of the tooth to Kentucky for testing. This takes longer and is more expensive.

In a TOPAS test a paper point is placed at the neck of the tooth for one minute. The point is removed and put into a solution that changes colour according to the degree of toxicity present. The solution is placed into a Colourimeter device made by Bioscience which accurately measures the change in colour and gives it a number. The process is repeated with a different solution to test for bacterial proteins. The gives a precise reading of both the severity of the toxins and the activity of the bacteria.

Treatment

If a root filling tooth is toxic it cannot be refilled. Its toxicity can be moderated using iodine and a TENS machine. How long the teeth remain toxin free is yet unknown. Our results show a maximum length of 3 weeks in a severely toxic tooth.

Toxic teeth are best removed using an atraumatic extraction technique and the wound treated as if it was a cavitation.

This prevents later cavitations forming. The technique we use is a modified Ogura technique as this does not crack the bone. It is gentle but does take a long time. The socket is treated exactly as we would treat a cavitation site, that is with cleaning, washing with ozonated magnesium chloride, ozone gas, ozone gel etc.

Biocallex or Endocal as it has been renamed, is a root filling material that is toxin free. Not only that it expands on setting and penetrates the tiny lateral canals pushing out the bacteria and not allowing re infection of the tooth canals. It does require a different technique than conventional root filling as the scraping of the canal side in conventional treatment blocks the entry to the lateral canals thereby virtually ensuring failure.

It is the only material that is safe to use at present, even then it is not 100% effective every time. Biocallex or Endocal is only faintly visible on X-rays, a fact which all patients with Biocallex/Endocal filled teeth must tell any future dentist they consult about this or it will be assumed that the tooth is incorrectly filled. Conventional root fillings are radio-opaque and show up white on x-ray film, Biocallex/Endocal does not unless Yterium is added to the mix.

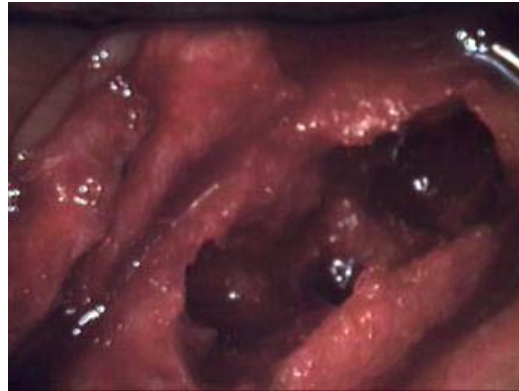
The most frequently used sign of success for a root filling is when it is painless and not sensitive to pressure. This is no guarantee that the tooth is toxic free. An x-ray taken of a "successfully" root filled tooth will often show changes at the root tip. These are interpreted as inflammation healing. These changes are not a positive sign but in reality show encapsulated infection.

Dead teeth behave in the same way as root filled teeth. They are darker than living teeth. A blow or extensive drilling on a tooth is usually a good way of killing a nerve inside the tooth. A "Blue" tooth is a dead tooth. These teeth eventually blow up into acute infections needing extraction or root filling.

Osteonecrosis of the jaw

Jacques Imbeau, D.M.D., NZDREX

This is the original article as I have written it on the 6 October 2006. The article was selected on the Medicine Portal on 12 November 2006 as one on Wikipedia's best articles related to Medicine as well as receiving a general good article rating on the 23 October 2006. Subsequently numerous other editors have altered the article and it was delisted from the good article rating on 9 June 2009.



Osteonecrosis of the jaw: Upper left jaw in a patient diagnosed with venous insufficiency.

Osteonecrosis of the jaws (ONj) is a severe bone disease that affects the jaws, including the maxilla and the mandible. Jaw bone (*osteo-*) damage and death (*-necrosis*) occurs as a result of reduced local blood supply (ischaemia). The condition is thus included in the general category of ischaemic or avascular osteonecrosis (literally "dead bone from poor blood flow.").

Various forms of ONj have been described over the last 160 years, and a number of causes have been suggested in the literature. In recent years, an increased incidence of ONj has been associated with the use of high dosages of bisphosphonates, required by some cancer treatment regimens, especially when the patient undergoes subsequent dental procedures. The possible risk from lower oral doses of bisphosphonates, taken by patients to prevent or treat osteoporosis, remains uncertain.^[1]

Various treatment options have been explored, however severe cases of ONj still require surgical removal of the affected bone.^[2] Careful anamnesis (patient history) and assesement of pre-existing systemic problems and possible sites of dental infection are required to help prevent the condition, especially if bisphosphonate therapy is considered.^[1]

Aetiology

Histopathological alterations

It may have either necrotic bone or bone marrow that has been slowly strangulated or nutrient-starved. Bone with chronically poor blood flow develops either a fibrous marrow since fibres can more easily live in nutrient starved areas, a greasy, dead fatty marrow (wet rot), a very dry, sometimes leathery marrow (dry rot), or a completely hollow marrow space (osteocavitation), also typical of ONj. The blood flow impairment occurs following a bone infarct, a blood clot forming inside the smaller blood vessels of cancellous bone tissue.

Under ischaemic conditions numerous pathological changes in the bone marrow and trabeculae of oral cancellous bone have been documented. Microscopically, areas of *"apparent fatty degeneration and/or necrosis, often with pooled fat from destroyed adipose cells (oil cysts) and with marrow fibrosis (reticular fatty degeneration)"* are seen. These changes are present even if *"most bony trabeculae appear at first glance viable, mature and otherwise normal, but closer inspection demonstrates focal loss of osteocytes and variable micro cracking (splitting along natural cleavage planes)*. The microscopic features are similar to those of *ischaemic or aseptic osteonecrosis of long bones, corticosteroid-induced osteonecrosis, and the osteomyelitis of caisson (deep-sea*

diver's) disease".^[3]

In the cancellous portion of femoral head it is not uncommon to find trabeculae with apparently intact osteocytes which seem to be "alive" but are no longer synthesizing collagen. This appears to be consistent with the findings in alveolar cancellous bone.^[4]

Osteonecrosis can affect any bone, but the hips, knees and jaws are most often involved. Pain can often be severe, especially if teeth and/or a branch of the trigeminal nerve is involved, but many patients do not experience pain, at least in the earlier stages. When severe facial pain is involved, the term NICO, for Neuralgia-Inducing Cavitational Osteonecrosis, is frequently used.

ONj, even in its mild or minor forms, creates a marrow environment that is conducive to bacterial growth. Since many individuals have low-grade infections of the teeth and gums, this probably is one of the major mechanisms by which the marrow blood flow problem can worsen; any local infection / inflammation will cause increased pressures and clotting in the area involved. No other bones have this mechanism as a major risk factor for osteonecrosis. A wide variety of bacteria have been cultured from ONj lesions. Typically, they are the same microorganisms as those found in periodontitis or devitalized teeth. However, according to special staining of biopsied tissues, bacterial elements are rarely found in large numbers. So while ONj is not primarily an infection, many cases have a secondary, very low-level of bacterial infection and chronic non-suppurative osteomyelitis can be associated with ONj. Fungal infections in the involved bone do not seem to be a problem, but viral infections have not been studied. Some viruses, such as the smallpox virus (no longer existent) can produce osteonecrosis.

The effects of persistent ischaemia on bone cells

Cortical bone is well vascularized by the surrounding soft tissues thus less susceptible to ischaemic damage. Cancellous bone, with its mesh like structure and spaces filled with marrow tissue is more prone to damage by bone infarcts, leading to anoxia and premature cell apoptosis.^{[5][6][7][8]} The mean life-span of osteocytes has been estimated to be 15 years in cancellous bone,^[9] and 25 years in cortical bone.^[10] while the average lifespan of human osteoclasts is about 2 to 6 weeks and the average lifespan of osteoblasts is approximately 3 months.^[11] In healthy bone these cells are constantly replaced by differentiation of bone marrow mesenchymal stem cells (MSC).^[12] However in both non-traumatic osteonecrosis and alcohol-induced osteonecrosis of the femoral head, a decrease in the differentiation ability of mesenchymal stem into bone cells has been demonstrated,^{[13][14]} and altered osteoblastic function plays a role in ON of the femoral head.^[15] If these results are extrapolated to ONj the altered differentiation potential of bone marrow mesenchymal stem cells (MSC) combined with the altered osteoblastic activity and premature death of existing bone cells would explain the failed attempts at repair seen in ischaemic-damaged cancellous bone tissue in ONj.

The rapidity with which premature cell death can occur depends on the cell type and the degree and duration of the anoxia. haematopoietic cells, in bone marrow, are sensitive to anoxia and are the first to die after reduction or removal of the blood supply. In anoxic conditions they usually die within 12 hours. Experimental evidence suggests that bone cells composed of osteocytes, osteoclasts, and osteoblasts die within 12-48 hours, and marrow fat cells die within 120 hours.^[16] The death of bone does not alter its radiographic opacity nor its mineral density. Necrotic bone does not undergo resorption; therefore, it appears relatively more opaque.

Attempts at repair of ischaemic-damaged bone will usually occur in 2 phases. First, when dead bone abuts live marrow, capillaries and undifferentiated mesenchymal cells grow into the dead marrow spaces, while macrophages degrade dead cellular and fat debris. Second, mesenchymal cells differentiate into osteoblasts or fibroblasts. Under favorable conditions, layers of new bone form on the surface of dead spongy trabeculae. If sufficiently thickened, these layers may increase the radiopacity of the bone; therefore, the first radiographic evidence of previous osteonecrosis may be patchy sclerosis resulting from repair. Under unfavorable conditions repeated attempts at repair in ischaemic conditions can be seen histologically and are characterized by extensive delamination or microcracking along cement lines as well as the formation of excessive cement lines.^[17] Ultimate failure of repair mechanisms due to persistent and repeated ischaemic events is manifested as trabecular fractures that occur in the dead bone under functional load. Later followed by cracks and fissures leading to structural collapse of the area involved (osteocavitation).^[16]

Other contributing factors

Other factors such as toxicants can adversely impact bone cells. Infections, chronic or acute, can affect blood flow by inducing

platelet activation and aggregation, contributing to a localized state of excess coagulability (hypercoagulability) that may contribute to clot formation (thrombosis), a known cause of bone infarct and ischaemia. Exogenous estrogens, also called hormonal disruptors, have also been linked with an increased tendency to clot (thrombophilia) and impaired bone healing.^[18]

Heavy metals such as lead and cadmium have been implicated in osteoporosis. Cadmium and lead also promotes the synthesis of plasminogen activator inhibitor-1 (PAI-1) which is the major inhibitor of fibrinolysis (the mechanism by which the body breaks down clots) and shown to be a cause of hypofibrinolysis.^[19] Persistent blot clots can lead to congestive blood flow (hyperemia) in bone marrow, impaired blood flow and ischaemia in bone tissue resulting in lack of oxygen (hypoxia), bone cell damage and eventual cell death (apoptosis). Of significance is the fact that the average concentration of cadmium in human bones in the 20th century has increased to about 10 times above the pre-industrial level.^[20]

Ethanol both from exogenous and endogenous sources and, its more toxic metabolite, acetaldehyde, have also been implicated in both osteoporosis and osteonecrosis. Acetaldehyde, a highly toxic metabolite of ethanol, can play a role in hypoxia and inhibit the osteoblastogenic potential of the bone marrow.^[21] Ethanol has been shown to alter the epithelial barrier through ethanol oxidation into acetaldehyde by the colonic microflora and downstream mast cell activation. Such alterations that remain for longer periods could result in excessive endotoxin passage into the vascular network.^[22] Intracolonic acetaldehyde may also be an important determinant of the blood acetaldehyde level and a possible hepatotoxin.^[23] High serum antibody titers against acetaldehyde-protein adducts have been found not only in alcoholics but also in patients with nonalcoholic liver disease, suggesting a contribution of acetaldehyde derived from sources other than exogenous ethanol.^[24] In a study on rats the role of intestinal bacterial overgrowth on the production and metabolism of ethanol, rats with a jejunal self-filling diverticulum (blind-loop) were compared to controls with a self-emptying diverticulum. Both endogenous ethanol and acetaldehyde were found in the blind-loop contents. Intragastric administration of sucrose produced a marked increase in acetaldehyde and acetate in the portal venous blood, with only a modest elevation of ethanol. It was concluded that the resulting high concentrations of acetaldehyde, both in the intestinal lumen and the portal blood, may have deleterious effects on the gastrointestinal (GI) mucosa and the liver.^[25] Another experimental in-vitro study showed the potential of certain bacteria representing normal human colonic flora to produce acetaldehyde under various atmospheric conditions that may prevail in different parts of the GI tract. This bacterial adaptation may be an essential feature of the bacteriocolonial pathway to produce toxic and carcinogenic acetaldehyde from either endogenous or exogenous ethanol.^[26] Many species of gut bacteria, yeast and fungal organisms such as *Candida albicans* found in the human GI tract and involved in gut dysbiosis, an imbalance in the microbial flora, have been shown to significantly increase blood ethanol levels, post-mortem, in individuals who had not consumed any alcohol before death.^{[27][28]}

The effects of chronic gut dybiosis and long term exposure to low levels of endogenous acetaldehyde on bone tissue and hepatic function is not yet fully understood. However Cordts et al suggested in 2001 that gut dysbiosis (as indicated by stool yeast) and hepatic detoxification challenge pathway exhaustion may lead to subclinical, systemic inflammation and chronic venous insufficiency (CVI). CVI is a pathological condition caused either by the congenital absence of or damage to venous valves in the superficial and communicating systems. Venous incompetence due to thrombi and formation of thrombi favoured by the Virchow triad (venous stasis, hypercoagulability, endothelial trauma) also can cause CVI.^[29]

Bisphosphonates may alter the disease process

In the past few years, thousands of cases of ONj in patients on bisphosphonate therapy have been diagnosed usually following lack of healing after a dental extraction but also in cases of spontaneous exposure of the cortical bone tissue through the gingiva and mucosa.^{[30][31]}

The recent increase of such cases has been linked with a major emphasis on the therapeutic use of bisphosphonates for osteoporosis, especially since hormone replacement therapy has been shown to increase the risk of breast cancer, clots and cardiovascular disease in women following the 2003 findings of the U.S. Women's Health Initiative study.^[32] Two classes of bisphosphonates are presently prescribed:

- Non-nitrogen containing bisphosphonates such as etidronate (Didronel®, Procter & Gamble Pharmaceuticals)
- Nitrogen containing such as alendronate (Fosamax®, Merck), pamidronate (Aredia®, Novartis), zoledronate (Zometa®, Novartis), risedronate (Actonel®, Procter & Gamble) and ibandronate (Boniva®, Roche Laboratories).

The nitrogen containing bisphosphonates are the most potent inhibitors and no case of ONj associated with etidronate has been reported yet. The main pharmacological action of bisphosphonates is inhibition of the osteoclast driven bone resorption. This is achieved by shortening osteoclast lifespan via apoptosis and by inhibiting osteoclast activity and recruitment on the bone surface (61). When a bisphosphonate binds to bone mineral, osteoclast resorb both bone and the bound bisphosphonate. During bone formation, if any, bisphosphonate remaining on the surface of the bone is covered and remains there until future osteoclastic bone resorption at the site. This explains why inhibition of bone resorption continues long after bisphosphonate treatment has been discontinued. [33]

This form of therapy has been shown to prevent loss of bone mineral density (BMD) as a result of a reduction in bone turnover. However bone health is a lot more than BMD.

In healthy bone tissue there is an homeostasis between bone resorption and bone apposition. Diseased or damaged bone is resorbed through the osteoclasts mediated process while osteoblasts form new bone to replace it, thus maintaining healthy bone density. A process commonly called remodelling.

However osteoporosis is essentially the result of a lack of new bone formation in combination with bone resorption in reactive hyperemia, related to various etiological and contributing factors, and bisphosphonates do not address these factors at all.

An individual who is already having problems with osteoporosis/ osteonecrosis of the jaws due to the effects of these etiological factors will be more susceptible to the adverse effects of bisphosphonates. In theory, by suppressing osteoclastic activity and bone resorption, any ischaemic-damaged bone will be left in situ instead of being resorbed. The damaged bone will not be repaired either if the factors already inhibiting osteoblastic activity are still present. Therefore the amount of osteonecrotic tissue should be expected to increase until it reaches a level when any trauma or insult to this necrotic bone will result in extremely poor healing, exposed necrotic bone to the oral environment, development of pain, and increased risks of microbial infection, as effectively seen in bisphosphonates associated cases of ONj.

In a systematic review of cases of bisphosphonates associated ONj up to 2006, it was concluded that the mandible is more commonly affected than the maxilla (2:1 ratio), and 60% of cases are preceded by a dental surgical procedure. According to Woo, Hellstein and Kalmar, oversuppression of bone turnover is probably the primary mechanism for the development of this form of ONj, although there may be contributing co-morbid factors (as discussed elsewhere in this article). It is recommended that all sites of potential jaw infection should be eliminated before bisphosphonate therapy is initiated in these patients to reduce the necessity of subsequent dentoalveolar surgery. The degree of risk for osteonecrosis in patients taking oral bisphosphonates, such as alendronate (Fosamax®), for osteoporosis is uncertain and warrants careful monitoring. [1]

History in dental medicine

ONj is not a new disease, around 1850 various forms of "chemical osteomyelitis" resulting from environmental pollutants, such as lead and the [white phosphorus]] used in safety matches (Phossy jaw), as well as from popular medications containing mercury, arsenic or bismuth, were reported in the literature. [34][35][36][37][38][39][40] This disease apparently did not often occur in individuals with good gingival health, and usually targeted the mandible first. [35] It was associated with localized or generalized deep ache or pain, often of multiple jawbone sites. The teeth often appeared sound and suppuration was not present. Even so, the dentist often began extracting one tooth after another in the region of pain, often with temporary relief but usually to no real effect. [36]

Today a growing body of scientific evidence indicate that this disease process, in the cancellous bone and bone marrow, is caused by bone infarcts mediated by a range of local and systemic factors. Bone infarcts as well as damage to the deeper portion of the cancellous bone is an insidious process. It is certainly not visible clinically and routine imaging techniques such as radiographs are not effective for that sort of damage. *"An important and often incompletely understood principle of radiography is the amount of bone destruction that goes undetected by routine x-rays procedures; this has been demonstrated by numerous investigators. Destruction confined to the cancellous portion of the bone cannot be detected radiographically, ad radiolucencies appear only when there is internal or external erosion or destruction of the bone cortex."* [41] In fact no radiographic findings are specific for bone infarction / osteonecrosis. A variety of pathologies may mimic bone infarction, including stress fractures, infections, inflammations, and metabolic and neoplastic processes. The limitations apply to all imaging modalities, including plain radiography, radionuclide studies, CT scans, and magnetic resonance imaging (MRI). Through-transmission alveolar ultrasound, based on quantitative ultrasound (QUS) in combination with panoramic dental radiography (orthopantomography) is

helpful in assessing changes in jawbone density.^{[42][43]} When practitioners have an up to date understanding of the disease process and a good anamnesis is combined with detailed clinical findings and course of events, the diagnosis, with the help of various imaging modality, can be achieved earlier, in most patients.

In the modern dental profession, it is only recently when severe cases associated with bisphosphonates came to light, that the issue of ONj has been brought to the attention of a majority of dentists. At present, the focus is mostly on bisphosphonates associated cases, and is sometimes referred to colloquially as "phossy jaw", a similar, earlier occupational disease.^{[44][45]} However, the pharmaceutical manufacturers of bisphosphonates drugs such as Merck and Novartis have stated that ONj in patients on this class of drug, can be related to a pre-existing condition, coagulopathy, anemia, infection, use of corticosteroids, alcoholism and other conditions already known to be associated with ONj in absence of bisphosphonate therapy. The implication is that bisphosphonates may not be the initiating cause of ONj and that other pre-existing or concurrent systemic and/or local dental factors are involved.^{[46][47]}

Since ONj has been diagnosed in many patients who did not take bisphosphonates, it is thus logical to assume that bisphosphonates are not the only factor in ONj. While the oversuppression of bone turnover seems to play a major role in aggravating the disease process, other factors can and do initiate the pathophysiological mechanisms responsible for ONj. In non-bisphosphonate cases of ONj, it is mainly the cancellous portion of the bone and it's marrow content that are involved in the disease process. The first stage is an oedema of the bone marrow initiated by a bone infarct, which is itself modulated by numerous etiological factors, leading to myelofibrosis as a result of hypoxia and gradual loss of mineral bone density characteristic of ischaemic osteoporosis. Further deterioration can be triggered by additional bone infarcts leading to anoxia and a localized areas of osteonecrosis within the osteoporotic cancellous bone. Secondary events such as dental infection, injection of local anaesthetics with vasoconstrictors, such as epinephrine, and trauma can add further complications to the disease process and chronic non-pus forming bone infection osteomyelitis can also be associated with ONj.^{[48][49][50]}

However, in patients on bisphosphonates, the cortical bone is also frequently involved as well. Spontaneous exposure of necrotic bone tissue through the oral soft tissues or following non-healing bone exposure after routine dental surgery, characteristics of this form of ONj, may be the result of late diagnosis of a disease process that has been masked by the oversuppression of osteoclastic activity, allowing pre-existing etiological factors to further aggravate bone damage.

Treatment

The treatment should be tailored to the individual patient according to the etiological factors involved and the severity of the disease process. With oral osteoporosis the emphasis should be on good nutrient absorption and metabolic wastes elimination through a healthy gastro-intestinal function, effective hepatic metabolism of toxicants such as exogenous estrogens, endogenous acetaldehyde and heavy metals, a balanced diet, healthy lifestyle, assessment of factors related to potential coagulopathies, and treatment of periodontal diseases and other oral and dental infections.

In cases of advanced oral ischaemic osteoporosis and/or ONj that are not bisphosphonates related, clinical evidence has shown that surgically removing the damaged marrow, usually by curettage and decortication, will eliminate the problem (and the pain) in 74% of patients with jaw involvement.^[2] Repeat surgeries, usually smaller procedures than the first, may be required, and almost a third of jawbone patients will need surgery in one or more other parts of the jaws because the disease so frequently present multiple lesions, i.e. multiple sites in the same or similar bones, with normal marrow in between. In the hip, at least half of all patients will get the disease in the opposite hip over time; this pattern occurs in the jaws as well. Recently, it has been found that some osteonecrosis patients respond to anticoagulation therapies alone. The earlier the diagnosis the better the prognosis. Research is ongoing on other non-surgical therapeutic modalities that could alone or in combination with surgery further improve the prognosis and reduce the morbidity of ONj. A greater emphasis on minimizing or correcting known etiological factors is necessary while further research is conducted on chronic ischaemic bone diseases such as oral osteoporosis and ONj.

In patients with bisphosphonates-associated ONj, the response to surgical treatment is usually poor.^[51] Conservative debridement of necrotic bone, pain control, infection management, use of antimicrobial oral rinses, and withdrawal of bisphosphonates are preferable to aggressive surgical measures for treating this form of ONj.^[52] Although an effective treatment for bisphosphonate-associated bone lesions has not yet been established,^[53] and this is unlikely to occur until this form of ONj is better understood, there as been clinical reports of some improvement after 6 months or more of complete cessation of bisphosphonate therapy.^[54]

Footnotes

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Mouth and Body Connection

Osteoporosis and

Osteonecrosis of the Jaws:

an underestimated problem with

multiple ramifications

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ABSTRACT

In the past 12 months a growing number of cases osteonecrosis of the jaws (ONj) in patients taking bisphosphonates have been reported worldwide in both the media and the scientific literature. ONj and its relationship with bisphosphonates, a class of pharmaceutical drugs used in the treatment of osteoporosis and hypercalcemia in cancer patients, are still debated in legal and medical circles.

In this article we will review both disease processes and the possible impact of bisphosphonates. A better understanding of the pathophysiological mechanisms involved could improve diagnosis and treatment of chronic ischaemic bone diseases (CIBD) such as oral osteoporosis and ONj. Bisphosphonates are not the only etiological factor involved in ONj, so a greater emphasis on other etiological factors such as heavy metals, gut toxicants, GI tract and hepatic function, as discussed in this article, is recommended as part of a more comprehensive approach in the management of CIBD such as oral osteoporosis and ONj.

KEY WORDS: Acetaldehyde, anoxia, bisphosphonates, bone infarcts, cadmium, chronic ischaemic bone diseases (CIBD), exogenous estrogens, metallo-estrogens, gut dysbiosis, hepatotoxicity, hyperemia, hypoxia, ischaemia, metallo-estrogens, osteonecrosis of the jaws (ONj), osteocavitations, osteoporosis, periodontitis, venous insufficiency.

INTRODUCTION

Chronic ischaemic bone diseases (CIBD) are characterized by gradual degenerative cancellous bone tissue damage related to altered blood flow. The degree of damage and the ability of bone tissue to repair itself is directly related to the severity and duration of the impaired perfusion with effects ranging from hyperemia, transient ischaemia, persistent ischaemia with hypoxia to complete infarction and anoxia. Numerous factors can be involved and will be discussed as we review the pathophysiological mechanisms involved in osteoporosis and osteonecrosis.

In dental medicine, oral osteoporosis and osteonecrosis of the jaws (ONj) are CIBD that can significantly impact on oral and systemic health and the U.S. National Institute of Health (NIH) considers osteoporosis as a devastating disorder with significant physiological, psychological and financial consequences. While the impact in craniofacial bones is acknowledged, there is a lack of reliable prevalence rate so the NIH recommends that more attention should be paid to skeletal health, especially in persons with conditions known to be associated with secondary osteoporosis. Of significance is the uniqueness of the maxilla and mandible. They are the only bones with teeth in them and numerous pathological processes related to the teeth and multiple therapeutic dental interventions may occur over a lifetime, including but not limited to, implantation of various foreign materials and repetitive injections of local anaesthetics with vasoconstrictors. Teeth are entirely dependant on healthy jaws both for their function and support. Finally the association between oral osteoporosis, systemic osteoporosis, periodontal diseases and cardiovascular diseases has been proven and recent complications with bisphosphonates treatment of systemic osteoporosis leading to complex cases of ONj have highlighted the pertinence of the NIH recommendation.

OSTEOPOROSIS DEFINED

Osteoporosis is a bone disease characterized by low bone mass and structural deterioration of bone tissue, leading to bone fragility and an increased susceptibility to fracture, especially of the hip, spine and wrist. It is generally characterized as primary (idiopathic) or secondary, depending on the presence or absence of associated medical conditions, surgical procedures or medications known to be associated with accelerated bone loss¹. Secondary osteoporosis is sometimes a purely local phenomenon, confined to particular bones or parts of them².

Osteoporosis results from abnormal organic matrix formation rather than abnormal bone calcification, as ♦

in osteomalacia. In general, in osteoporosis, the osteoblastic activity is less than normal, and consequently the rate of new bone formation is depressed. Living bone becomes osteoporotic as a result of osteoclastic bone resorption secondary to reactive hyperemia⁽³⁾ such as can occur in venous outflow problems initiated by a venous thrombotic process or venous insufficiency.

WHAT ARE THE CAUSES OF OSTEOPOROSIS ?

Multiple factors can be involved including, but not limited to, poor nutrient absorption to the extent that sufficient protein matrix cannot be formed, deficiency in vitamin C (which is apparently necessary for the secretion of intercellular substances by all cells, including osteoblasts)⁽⁴⁾, hormonal imbalances and exogenous oestrogens^(5,6). The role of heavy metals as exogenous oestrogens has been raised by recent reports of the ability of certain metal ions to also bind to oestrogen receptors and to give rise to oestrogen agonist responses in vitro and in vivo. This has resulted in the realisation that environmental oestrogens can also be inorganic and such xeno-oestrogens have been termed metallo-oestrogens. These metallo-oestrogens include aluminium, antimony, arsenite, barium, cadmium, chromium (Cr(II)), cobalt, copper, lead, mercury, nickel, selenite, tin and vanadate⁽⁷⁾. The effects of combinations of various metals can also be a factor as it has been demonstrated that a particular combination could be synergistic, antagonistic, or additive, depending on the relative doses employed. Generally, a combination was synergistic when the most toxic metal was present at or near its LD1 dose (dose that will kill one cell in a 100) in the presence of the much less toxic metal; the same combination was protective when the least toxic metal was present at or near its LD1 dose⁽⁸⁾. In studies on invertebrates development, it was demonstrated that Hg is three times more toxic than Cu, 20-30 times more than Cd, and 700-1000 times more toxic than Cr^(9,10). Studies on rats, sheep and monkeys implanted with dental amalgam have demonstrated a significant mercury burden in the, kidneys, liver, gut and jawbones of these animals^(11,12,13). So, if the experimental data is similar in humans, in theory a person with oral osteoporosis and mercury and cadmium in her jawbones, the effects of cadmium would be potentiated by mercury if mercury levels are close to a LD1 dose. Of course this is only an assumption and further research is needed before a definite conclusion can be reached in humans.

A strong association between cadmium, lead and osteoporosis has also been established. Low level exposure to cadmium is associated with an increased loss of bone mineral density readily in both genders, leading to osteoporosis and increased risk of fractures, especially in elderly and in females^(14,15). Animal studies have shown cadmium to stimulate the formation and activity of osteoclasts-mediated bone resorption⁽¹⁶⁾. Recent data demonstrate mild effects of cadmium on both kidney and bone with present environmental

exposure levels. Women may be at greater risk than men, because of increased gastrointestinal uptake of cadmium at low iron stores, which is common in women of childbearing age. The same data shows that about 90% of body lead is localised to bone and that there is a significant release of bone lead after the menopause, in association with the acceleration of bone resorption. Thus, postmenopausal women may be at increased risk of adverse effects of lead⁽¹⁷⁾.

Cadmium and lead also promotes the synthesis of plasminogen activator inhibitor-1 (PAI-1) which is the major inhibitor of fibrinolysis⁽¹⁸⁾. Persistent blot clots can lead to congestive hyperemia in bone marrow, impaired blood flow and ischaemia in bone tissue resulting in hypoxia, bone cell damage and eventual cell death. Of significance is the fact that the average concentration of cadmium in human bones in the 20th century has increased to about 10 times above the pre-industrial level⁽¹⁹⁾.

Other recent studies have highlighted the potential for adverse effect on bone tissue from certain gut toxins resulting from dysbiosis of the gastro-intestinal (GI) tract, a state of imbalance of the intestinal flora which may lead to excessive bacterial fermentation in the gut and auto-intoxication by microbial toxins, a particular problem in inflammatory bowel diseases⁽²⁰⁾. Acetaldehyde, a highly toxic metabolite of ethanol, can play a role in hypoxia and inhibit the osteoblastogenic potential of the bone marrow⁽²¹⁾. Ethanol itself has been shown to alter the epithelial barrier through ethanol oxidation into acetaldehyde by the colonic microflora and downstream mast cell activation. Such alterations that remain for longer periods could result in excessive endotoxin passage into the vascular network⁽²²⁾. Intracolonic acetaldehyde may also be an important determinant of the blood acetaldehyde level and a possible hepatotoxin⁽²³⁾. High serum antibody titers against acetaldehyde-protein compounds have been found not only in alcoholics but also in patients with nonalcoholic liver disease, suggesting a contribution of acetaldehyde derived from sources other than exogenous ethanol⁽²⁴⁾. In a study on rats the role of intestinal bacterial overgrowth on the production and metabolism of ethanol, rats with a jejunal self-filling diverticulum (blind-loop) were compared to controls with a self-emptying diverticulum. Both endogenous ethanol and acetaldehyde were found in the blind-loop contents. Intra-gastric administration of sucrose produced a marked increase in acetaldehyde and acetate in the portal venous blood, with only a modest elevation of ethanol. It was concluded that the resulting high concentrations of acetaldehyde, both in the intestinal lumen and the portal blood, may have deleterious effects on the gastrointestinal mucosa and the liver⁽²⁵⁾. Another experimental in-vitro study showed the potential of certain bacteria representing normal human colonic flora to produce acetaldehyde under various atmospheric conditions that may

prevail in different parts of the GI tract. This bacterial adaptation may be an essential feature of the bacteriocolonization pathway to produce toxic and carcinogenic acetaldehyde from either endogenous or exogenous ethanol⁽²⁶⁾. Many species of gut bacteria, yeast and fungal organisms such as *Candida Albicans* found in the human GI tract and involved in gut dysbiosis have been shown to significantly increase blood ethanol levels, post-mortem, in individuals who had not consumed any alcohol before death^(27,28). The effects of chronic gut dysbiosis and long term exposure to low levels of endogenous acetaldehyde on bone tissue and hepatic function is not yet fully understood. However Cordts et al suggested in 2001 that gut dysbiosis (as indicated by stool yeast) and hepatic detoxification challenge pathway exhaustion may lead to subclinical, systemic inflammation and chronic venous insufficiency (CVI). CVI is a pathological condition caused either by the congenital absence of or damage to venous valves in the superficial and communicating systems. Venous incompetence due to thrombi and formation of thrombi favoured by the Virchow triad (venous stasis, hypercoagulability, endothelial trauma) also can cause CVI⁽²⁹⁾.

Since reactive bone marrow hyperemia has been implicated in osteoporosis and hyperemia has been associated with a venous outflow problem, the relationship between gut dysbiosis, hepatic dysfunction and endogenous acetaldehyde production in CIBD warrants further attention.

THE MOUTH AND BODY CONNECTION

Further investigations have demonstrated a significant association between bone mineral density (BMD) of the mandible and the peripheral skeleton in postmenopausal women. Some studies also have linked low BMD of the mandible and the peripheral skeleton with alveolar bone loss of the mandible and tooth loss.

In a review of the literature in 1997, Hildebolt concluded that an association between osteoporosis and oral bone loss existed while recommending additional longitudinal studies. He suggested that inexpensive methods must be developed for sensitive and specific measures of oral bone loss⁽³⁰⁾.

More recent studies have cited osteoporosis as a risk factor for periodontal disease even while their association is still not well understood⁽³¹⁾. There is evidence that patients with systemic osteoporosis are likely to have decreased oral bone density, which may affect treatment decisions. Further, patients with decreased bone mineral density, indicative of osteoporosis, may be at a higher risk for periodontitis. Therefore, osteoporosis, could be considered a risk factor for periodontitis⁽³²⁾.

Numerous researchers have also established an association between periodontal diseases and various systemic diseases including cardio-vascular diseases. For example DeStefano et al. focused on the contribution of periodontitis and analyzed coronary heart disease and mortality outcomes in nearly 10,000 subjects followed for 14 years longitudinally in the

NHANES I study. Periodontitis for this cohort study was assessed with the periodontal index. Overall, subjects with periodontitis had a 25% increased risk for coronary heart disease relative to those with minimal periodontal disease.

This association occurred after adjustments for potential confounders like age, sex, race, education, marital state, systolic blood pressure, total cholesterol levels, body mass index, diabetes, physical activity, alcohol consumption, poverty and cigarette smoking. For males younger than 50 years, periodontitis more strongly affected the incidence of coronary heart disease with a relative risk of 1.72⁽³³⁾.

Lately, data regarding the periodontal microbial challenge support the biological plausibility of the associations seen in human population studies. Hertzberg et al. have reported that two oral microbes, *Streptococcus sanguis* and *P. gingivalis*, express a collagen-like platelet aggregation-associated protein that can stimulate thrombotic events^(34,35). Genco et al. presented preliminary data that suggest an odds ratio of 2.8 for subjects harboring *P. gingivalis* in periodontal pockets and exhibiting a myocardial infarction⁽³⁶⁾. In addition, Zambon et al. recently isolated DNA sequences specific for periodontal pathogens like *P. gingivalis* and *A. actinomycetemcomitans* from human atheroma specimens using polymerase chain reaction (PCR) techniques⁽³⁷⁾. Other non periodontal infectious agents like *Chlamydia pneumoniae*, *Helicobacter pylori*, herpes simplex and cytomegalovirus have been previously detected in atheromatous lesions using similar methods, and further support an infectious aetiology for cardiovascular disease⁽³⁸⁾.

The available evidence points to adverse impact on bone nutrition and impaired elimination of metabolic waste and xeno-toxicants because of altered blood flow to the affected bone areas.

In spite of the above scientific evidence, osteoporosis, including oral osteoporosis, is underdiagnosed⁽³⁹⁾ because diagnosis of osteoporosis is complicated by the fact that osteoporotic cancellous bone cannot be easily detected by routine clinical examination, even with the help of routine blood tests or x-rays⁽²⁾. Special investigations using dual x-ray absorptiometry (DEXA) and Quantitative Ultrasounds (QUS) are necessary⁽⁴⁰⁾.

OSTEONECROSIS:

Osteonecrosis is a severe bone disease that is also caused by ischaemia. It means literally, "dead bone from poor blood flow." It may have either necrotic bone or bone marrow that has been slowly strangulated or nutrient-starved. Bone with chronically poor blood flow develops either a fibrous marrow since fibres can more easily live in nutrient starved areas, a greasy, dead fatty marrow (wet rot), a very dry, sometimes leathery marrow (dry rot), or a completely hollow marrow space (osteocavitation), also typical of ONj. The blood flow impairment occurs following a bone infarct, a blood clot

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forming inside the smaller blood vessels of cancellous bone tissue.

Under ischaemic conditions numerous pathological changes in the bone marrow and trabeculae of oral cancellous bone have been documented. Microscopically, areas of “apparent fatty degeneration and/or necrosis, often with pooled fat from destroyed adipose cells (oil cysts) and with marrow fibrosis (reticular fatty degeneration)” are seen. These changes are present even if “most bony trabeculae appear at first glance viable, mature and otherwise normal, but closer inspection demonstrates focal loss of osteocytes and variable micro cracking (splitting along natural cleavage planes). The microscopic features are similar to those of ischaemic or aseptic osteonecrosis of long bones, corticosteroid-induced osteonecrosis, and the osteomyelitis of caisson (deep-sea diver’s) disease”⁽⁴¹⁾.

In the cancellous portion of femoral head it is not uncommon to find trabeculae with apparently intact osteocytes which seem to be “alive” but are no longer synthesizing collagen⁽⁴²⁾. This appears to be consistent with the findings in alveolar cancellous bone.

Any bone can be affected, but the hips, knees and jaws are most often involved. Pain can often be severe, especially if teeth and/or a branch of the trigeminal nerve is involved, but many patients do not experience pain, at least in the earlier stages. When severe facial pain is involved, the term NICO, for Neuralgia-Inducing Cavitational Osteonecrosis, is frequently used.

ONj, even in its mild or minor forms, creates a marrow environment that is conducive to bacterial growth. Since many individuals have low-grade infections of the teeth and gums, this probably is one of the major mechanisms by which the marrow blood flow problem can worsen (any local infection / inflammation will cause increased pressures and clotting in the area involved). No other bones have this mechanism as a major risk factor for osteonecrosis. A wide variety of bacteria have been cultured from ONj lesions. Typically, they are the same microorganisms as those found in periodontitis or devitalized teeth. However, according to special staining of biopsied tissues, bacterial elements are rarely found in large numbers. So while ONj is not primarily an infection, many cases have a secondary, very low-level of bacterial infection and chronic non-suppurative osteomyelitis can be associated with ONj. Fungal infections in the involved bone do not seem to be a problem, but viral infections have not been studied. Some viruses, such as the smallpox virus (no longer existent) can produce osteonecrosis in at least 5% of infected persons, usually in the leg bones⁽⁴³⁾.

THE EFFECTS OF PERSISTENT ISCHAEMIA ON BONE CELLS

Cortical bone is well vascularized by the surrounding soft tissues thus less susceptible to ischaemic damage. Cancellous

bone, with its mesh like structure and spaces filled with marrow tissue is more prone to damage by bone infarcts, leading to anoxia and premature cell apoptosis⁽⁴⁴⁻⁴⁷⁾. The mean life-span of osteocytes has been estimated to be 15 years in cancellous bone⁽⁴⁸⁾ and 25 years in cortical bone⁽⁴⁹⁾ while the average lifespan of human osteoclasts is about 2 to 6 weeks and the average lifespan of osteoblasts is approximately 3 months⁽⁵⁰⁾. In healthy bone these cells are constantly replaced by differentiation of bone marrow mesenchymal stem cells (MSCs)⁽⁵¹⁾. However in both nontraumatic osteonecrosis and alcohol-induced osteonecrosis of the femoral head, a decrease in the differentiation ability of mesenchymal stem into bone cells has been demonstrated^(52,53) and altered osteoblastic function plays a role in ON of the femoral head⁽⁵⁴⁾. If these results are extrapolated to ONj the altered differentiation potential of bone marrow MSCs combined with the altered osteoblastic activity and premature death of existing bone cells would explain the failed attempts at repair seen in ischaemically damaged cancellous bone tissue in ONj.

The rapidity with which premature cell death can occur depends on the cell type and the degree and duration of the anoxia. Hematopoietic cells, in bone marrow, are sensitive to anoxia and are the first to die after reduction or removal of the blood supply. In anoxic conditions they usually die within 12 hours. Experimental evidence suggests that bone cells composed of osteocytes, osteoclasts, and osteoblasts die within 12-48 hours, and marrow fat cells die within 120 hours⁽⁵⁵⁾. The death of bone does not alter its radiographic opacity nor its mineral density. Necrotic bone cannot undergo resorption; therefore, it appears relatively more opaque.

Attempts at repair of ischaemically damaged bone will usually occur in 2 phases. First, when dead bone abuts live marrow, capillaries and undifferentiated mesenchymal cells grow into the dead marrow spaces, while macrophages degrade dead cellular and fat debris. Second, mesenchymal cells differentiate into osteoblasts or fibroblasts. Under favorable conditions, layers of new bone form on the surface of dead spongy trabeculae. If sufficiently thickened, these layers may increase the radiopacity of the bone; therefore, the first radiographic evidence of previous osteonecrosis may be patchy sclerosis resulting from repair. Under unfavorable conditions repeated attempts at repair in ischaemic conditions can be seen histologically and are characterized by extensive delamination or microcracking along cement lines as well as the formation of excessive cement lines⁽⁵⁶⁾. Ultimate failure of repair mechanisms due to persistent and repeated ischaemic events is manifested as trabecular fractures that occur in the dead bone under functional load. Later followed by cracks and fissures leading to structural collapse of the area involved⁽⁵⁵⁾ (osteocavitation). ■

To be continued in the next issue.

Osteoporosis and Osteonecrosis of the jaws:

an underestimated problem with multiple ramifications

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AS mentioned in Part I, other factors such as toxicants can adversely impact bone cells. Infections, chronic or acute, can affect blood flow by inducing platelet activation and aggregation, contributing to a localized state of hypercoagulability that may contribute to thrombosis, a known cause of bone infarct and ischaemia. Exogenous estrogens have also been linked with thrombophilia and impaired bone healing⁽⁵⁷⁾. Heavy metals such as lead and cadmium have been implicated in osteoporosis and

shown to be a cause of hypofibrinolysis. Ethanol both from exogenous and endogenous sources and, its more toxic metabolite, acetaldehyde, have also been implicated in both osteoporosis and osteonecrosis.

BISPHOSPHONATES

In the past few years, thousands of cases of ONj in patients on bisphosphonate therapy have been diagnosed usually following lack of healing after a dental extraction but also in cases of spontaneous exposure of the cortical bone tissue through the gingiva and mucosa^(58,59).

TABLE 1 Initiating / predisposing risk factors for ONj other than genetically inherited hypercoagulability.

| PHYSICAL TRAUMA | MICROBIAL TRAUMA | TOXIC TRAUMA |
|----------------------|----------------------------|-------------------------------|
| Extractions | Periodontal disease | Cadmium, lead |
| Trauma from drilling | Cyst - granuloma | Root canal toxins |
| Oral surgery | Abscesses | Anaesthetic by-products |
| Endodontic therapy | Root canal bacteria/fungi | Vasoconstrictors (epinephrin) |
| Bruxism | Residual infections | Chemical toxins |
| Galvanism | Infected impacted teeth | Bacterial/fungal toxins |
| Radiation therapy | Infected implants | Metallic toxins |
| Accidents | Infected tooth buds | Thimerosal from vaccines |
| Deep sea diving | Infected dental alveola | Cigarette smoking |
| | Improperly cleaned alveola | Chemotherapy |
| | | Alcoholism (ethanol) |
| | | Bisphosphonates drugs |

NB: Individual factors may not be enough to initiate cancellous bone damage but a combination of many of the above factors is likely, especially if hypercoagulability is involved. The severity of the damage will vary according to individual susceptibility and the intensity of the above factors.

TABLE 2 – Syndrome, diseases & conditions associated with osteonecrosis

| | | |
|--|--|---------------------------------|
| Chronic fatigue syndrome | Irritable bowel syndrome | Myofascial dysfunction syndrome |
| TMJ syndrome | Micromercurialism | Fibromyalgia |
| Trigeminal neuralgia | Atypical facial neuralgia / Idiopathic facial neuralgia. | |
| Migraine headaches | Lupus erythematosus | Hypercortisolism |
| Hypercoagulation disorders – systemic and/or local (thrombophilia, hypofibrinolysisetc...) | | |
| Intra-osseous inflammation (related to microbial infections, trauma or autoimmunity) | | |
| Oestrogen replacement therapy (HRT) and xeno-oestrogens | | |
| Cardiovascular diseases and homocysteinemia | | |
| Hyperlipidemia & embolic fat | Hypertension | Arthritis |
| Chronic osteomyelitis | Osteoporosis | Arteriosclerosis |
| Cirrhosis, pancreatitis, fatty liver | Sickle cell anemia | |
| Cancers (Cancer induced hypercoagulation, lymphoma, carcinoma, radiotherapy induced osteonecrosis) | | |

The recent increase of such cases has been linked with a major emphasis on the therapeutic use of bisphosphonates for osteoporosis, especially since hormone replacement therapy (HRT) has been shown to increase the risk of breast cancer, clots and cardiovascular disease in women following the 2003 findings of the U.S. Women's Health Initiative study⁽⁶⁾. Two classes of bisphosphonates are presently prescribed:

- Non nitrogen containing bisphosphonates such as Etidronate (Didronel)
- Nitrogen containing such as Alendronate (Fosamax), Pamidronate (Aredia), Zoledronate (Zometa), Risedronate (Actonel) and Ibandronate (Boniva).

The nitrogen containing bisphosphonates are the most potent inhibitors and no case of ONj associated with Etidronate has been reported yet.

The main pharmacological action of bisphosphonates is inhibition of the osteoclast driven bone resorption. This is achieved by shortening osteoclast lifespan by killing them and by inhibiting osteoclast activity and recruitment on the bone surface.⁽⁶⁰⁾ When a bisphosphonate binds to bone mineral, osteoclast resorb both bone and the bound bisphosphonate. During bone formation, if any, bisphosphonate remaining on the surface of the bone is covered and remains there until future osteoclastic bone resorption at the site. This explains why inhibition of bone resorption continues long after bisphosphonate treatment has been discontinued⁽⁶¹⁾.

This form of therapy has been shown to prevent loss of BMD as a result of a reduction in bone turnover. However bone health is a lot more than BMD.

In healthy bone tissue there is an homeostasis between bone resorption and bone apposition. Diseased or damaged bone is resorbed through the osteoclasts mediated process while osteoblasts form new bone to replace it, thus maintaining healthy bone density. A process commonly called remodelling.

However osteoporosis is essentially the result of a lack of new bone formation in combination with bone resorption in reactive hyperemia , related to the etiological factors mentioned earlier, and bisphosphonates do not address these etiological factors at all.

An individual who is already having problems with osteoporosis/ osteonecrosis of the jaws due to the effects of these etiological factors will be more susceptible to the adverse effects of bisphosphonates. In theory, by suppressing osteoclastic activity and bone resorption, any ischaemically damaged bone will be left in situ instead of being resorbed. The damaged bone will not be repaired either if the factors already inhibiting osteoblastic activity are still present. Therefore the amount of osteonecrotic tissue should be expected to increase until it reaches a level when any trauma or insult to this necrotic bone will result in extremely poor healing , exposed necrotic bone to the oral environment, development of pain, and increased risks of microbial infection, as effectively seen in bisphosphonates associated cases of ONj.

In a systematic review of cases of bisphosphonates associated ONj up to 2006, it was concluded that the mandible is more commonly affected than the maxilla (2:1 ratio), and 60% of cases are preceded by a dental surgical procedure. According to Woo, Hellstein and Kalmar, oversuppression of bone turnover is probably the primary mechanism for the development of this form of ONj, although there may be contributing co-morbid factors (as discussed elsewhere in this article). It is recommended that all sites of potential jaw infection should be eliminated before bisphosphonate therapy is initiated in these patients to reduce the necessity of subsequent dentoalveolar surgery. The degree of risk for osteonecrosis in patients taking oral bisphosphonates, such as alendronate (Fosamax – Merck), for osteo-

porosis is uncertain and warrants careful monitoring.⁽⁶²⁾

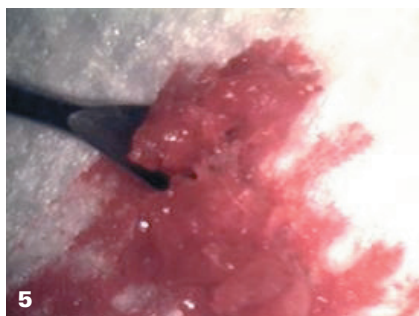
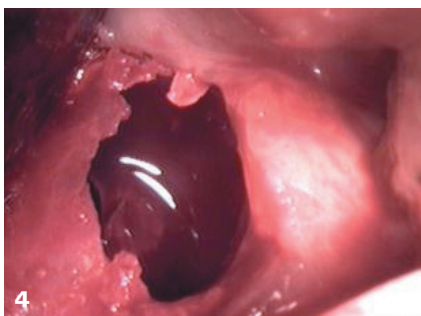
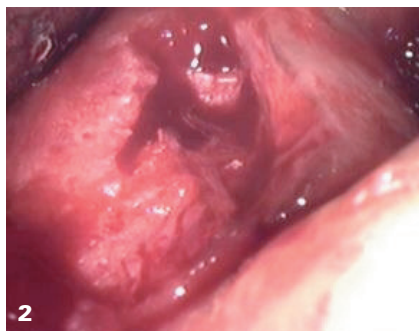
IS OSTEONECROSIS OF THE JAWS A CONTROVERSIAL ISSUE IN DENTISTRY?

Yes. In our opinion, the controversy is more the result of a lack of understanding of the disease than the lack of prevalence. ONj is not a new disease, around 1850 various forms of "chemical osteomyelitis" resulting from environmental pollutants, such as lead and the white phosphorus used in safety matches, as well as from popular medications containing mercury, arsenic or bismuth, were reported in the literature.⁽⁶³⁻⁶⁹⁾ This disease apparently did not often occur in individuals with good gingival health, and usually targeted the mandible first.⁽⁶⁴⁾ It was associated with localized or generalized deep ache or pain, often of multiple jawbone sites. The teeth often appeared sound and suppuration was not present. Even so, the dentist often began extracting one tooth after another in the region of pain, often with temporary relief but usually to no real effect.⁽⁶⁵⁾

Today a growing body of scientific evidence indicates that this disease process, in the cancellous bone and bone marrow, is caused by bone infarcts mediated by a range of local and systemic factors. And bone infarcts as well as damage to the deeper portion of the cancellous bone is an insidious process. It is certainly not visible clinically and routine imaging techniques such as radiographs are not effective for that sort of damage. *"An important and often incompletely understood principle of radiography is the amount of bone destruction that goes undetected by routine x-rays procedures. This has been demonstrated by numerous investigators... Destruction confined to the cancellous portion of the bone cannot be detected radiographically. Radiolucencies appear only when there is internal or external erosion or destruction of the bone cortex."*⁽⁷⁰⁾ In fact no radiographic findings are specific for bone infarction / osteonecrosis. A variety of pathologies may mimic bone infarction, including stress fractures, infections, inflammations, and metabolic and neoplastic processes. The limitations apply to all imaging modalities, including plain radiography, radionuclide studies, CT, and MRI. Through-transmission alveolar ultrasound (Cavitat CAV 4000 – Cavitat Medical Technologies, Emory, Texas) based on the principles of quantitative ultrasound (QUS) in combination with dental panoramic radiography is more helpful in assessing changes in jawbone density.^(71,72) When health practitioners have an up to date understanding of the disease process and a good anamnesis is combined with detailed clinical findings and course of events, the diagnosis, with the help of various imaging modality, can be achieved earlier, in most patients.

In the modern dental profession, it is only recently when severe cases associated with bisphosphonates came to light, that the issue of ONj has been brought to the attention of a majority of dentists. At present, the focus is mostly on bisphosphonates associated cases. However, the pharmaceutical manufacturers of bisphosphonates drugs such as Merck and Novartis have stated that ONj in patients on this class of drug, can be related to a pre-existing condition, coagulopathy, anemia, infection, use of corticosteroids, alcoholism and other conditions already known to be associated with ONj in absence of bisphosphonate therapy. The implication is that bisphosphonates may not be the initiating cause of ONj and that other pre-existing or concurrent systemic and/or local dental factors are involved.^(73,74)

Since ONj has been diagnosed in many patients who did not take bisphosphonates, it is thus logical to assume that bisphosphonates are not the only factor in ONj. While the oversuppression of bone turnover seems to play a major role in aggravating the disease process, other factors can and do initiate the pathophysiological mechanisms responsible for ONj. In non-



The photographs are of the left posterior area of the maxilla of a post-menopausal woman diagnosed with systemic osteoporosis who has received IV bisphosphonates. The site corresponds to the location of the upper left third molar and maxillary tuberosity. The patient made no mention of her IV-bisphosphonate injections on her medical questionnaire. She was unaware of the risk of ONj when treated with IV bisphosphonates.

Photo # 1: Appearance of soft tissue in the left maxillary tuberosity area – normal clinical appearance with no visible sign of inflammation.

Photo # 2: Appearance of bone cortex upon soft tissue flap exposure – note the eroded cortical shell. This is prior to any probing or exploration.

Photo # 3: The cortical shell is probed here using a standard periodontal probe. The probe easily penetrated the eroded cortex and there was no resistance to deeper probing in the area of the bone normally occupied by cancellous bone tissue.

Photo # 4: A larger window has been prepared in the cortex to expose the medullary area. Note the large osteocavitation filled with some tissue debris at the bottom and the paper thin cortical bone at the margin of the access window. The osteocavitation defect is devoid of any trabecular structure, which has already collapsed, leaving a thinning, partially eroded cortical shell.

Photo # 5: The tissue debris removed from the medullary defect. Essentially fragments of necrotic bone trabeculae and necrotic bone marrow, typical of osteonecrosis.

bisphosphonate cases of ONj, it is mainly the cancellous portion of the bone and its marrow content that are involved in the disease process. The first stage is an oedema of the bone marrow initiated by a bone infarct, which is itself modulated by numerous etiological factors, leading to fibrous degeneration of the bone marrow as a result of hypoxia and gradual loss of mineral bone density characteristic of ischaemic osteoporosis. Further deterioration can be triggered by additional bone infarcts leading to anoxia and a localized areas of osteonecrosis within the osteoporotic cancellous bone. Secondary events such as dental infection, injection of local anaesthetics with vasoconstrictors such as epinephrine and trauma can add further complications to the disease process and chronic non-suppurative osteomyelitis can also be associated with ONj.⁽⁷⁵⁻⁸⁷⁾

However, in patients on bisphosphonates, the cortical bone is also frequently involved as well. Spontaneous exposure of necrotic bone tissue through the oral soft tissues or following non-healing bone exposure after routine dental surgery, characteristics of this form of ONj, may be the result of late diagnosis of a disease process that has been masked by the oversuppression of osteoclastic activity, allowing pre-existing etiological factors to further aggravate bone damage.

RECOMMENDED TREATMENTS:

The treatment should be tailored to the individual patient according to the etiological factors involved and the severity of the disease process. With oral osteoporosis the emphasis should be on good nutrient absorption and metabolic wastes elimination through a healthy gastro-intestinal function, effective hepatic metabolism of toxicants such as exogenous oestrogens, endogenous acetaldehyde and heavy metals, a balanced diet, healthy lifestyle, assessment of factors related to potential coagulopathies, and treatment of periodontal diseases and other oral and dental infections. If patients are on bisphosphonates, the need for this form of therapy should be re-assessed in light of the most recent research.

In cases of advanced oral ischaemic osteoporosis and/or ONj that are not bisphosphonates related, clinical evidence has shown that surgically removing the damaged marrow, usually by curettage and decortication, will eliminate the problem (and the pain) in 74% of patients with jaw involvement.⁽⁸⁸⁾ Repeat surgeries, usual-

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ly smaller procedures than the first, may be required, and almost a third of jawbone patients will need surgery in one or more other parts of the jaws because the disease so frequently present multiple lesions, i.e. multiple sites in the same or similar bones, with normal marrow in between. In the hip, at least half of all patients will get the disease in the opposite hip over time; this pattern occurs in the jaws as well. Recently, it has been found that some osteonecrosis patients respond to anticoagulation therapies alone. The earlier the diagnosis the better the prognosis. Research is ongoing on other non-surgical therapeutic modalities that could alone or in combination with surgery further improve the prognosis and reduce the morbidity of ONJ. A greater emphasis on minimizing or correcting known etiological factors is necessary while further research is conducted on CIBD such as oral osteoporosis and ONJ.

In patients with bisphosphonates-associated ONJ, the response to surgical treatment is usually poor.⁽⁸⁹⁾ Conservative debridement of necrotic bone, pain control, infection management, use of antimicrobial oral rinses, and withdrawal of bisphosphonates are preferable to aggressive surgical measures for treating this form of ONJ.⁽⁹⁰⁾ Although an effective treatment for bisphosphonate-associated bone lesions has not yet been established,⁽⁹¹⁾ and this is unlikely to occur until this form of ONJ is better understood, there has been clinical reports of some improvement after 6 months or more of complete cessation of bisphosphonate therapy.⁽⁹²⁾

PREVENTION:

An ounce of prevention is worth a pound of cure. This is particularly true in CIBDs which are by nature hidden into the deepest and least easily visible part of our body, the trabecular bone tissue.

- Prevention of CIBD involves the elimination or appropriate modification of factors that can initiate, predispose or increase the likelihood of CIBD occurring.
- Implementing of good oral hygiene programs to avoid dental caries and gum disease in order to avoid infection and potential trauma.
- The use of biocompatible, non-metallic, dental materials. Manufacturers should provide written results of biocompatibility testing according to ANSI / ISO standards. Avoidance of vasoconstrictors such as epinephrine in patients at risk. In routine dental surgery for exodontia, complete removal of infected periradicular tissue should be emphasized.
- If decay is present, early diagnosis and interception is indicated.
- Inclusion of chronic radicular infection in the assessment of treatment outcome in endodontic therapy, in addition to symptomatology and radiographic criterion. Full informed consent on the potential implications of chronic dental infections.
- The use of minimally invasive techniques whenever possible (minimally invasive dental medicine).
- Dietary and lifestyle choices such as organic foods, reduction of refined and processed foods such as sucrose and bleached flour which are a known cause of dental diseases. Avoidance of source of heavy metals such as cadmium, lead and mercury. Superphosphate fertilizers and tobacco are a known source of cadmium exposure in humans,^(93,94,95) dental amalgams are the main source of mercury exposure for the non-industrially exposed population.⁽⁹⁶⁾ Some brands of dental amalgams have also been shown to be a source of other toxic metals such as cadmium, indium, lead and in some case antimony. These metals, released from amalgam particles in the GI tract, were found in the liver, kidneys, lungs and blood or research animals.^(97,98,99)

- Emphasis on healthy gastro-intestinal and hepatic function, especially in patients with multiple metallic restorations such as amalgam or/and chronic infections such as gum disease or devitalized teeth. An important reminder is that patients cannot efficiently detoxify if the two major organs of detoxification and elimination are dysfunctional.

CONCLUSION:

There is still much to be learned about osteoporosis / osteonecrosis of the jaws. Health professionals have a duty of care to their patients in the prevention and early diagnosis of jaw osteoporosis and/ or earlier stages of ONJ. The quality of the prognosis is inversely related to the severity of the disease process. Greater awareness allows early diagnosis and early treatments, especially non surgical modalities to reduce coagulopathy, improve bone nutrition and reduce bone heavy metals content. Etiological factors have to be considered carefully in determining the best course of action in the diagnosis and treatment of ONJ. In patients with CIBD, more attention should be paid to GI tract health and hepatic function. The key role of the gut in nutrients absorption and toxins elimination in combination with the liver cannot be overemphasized, especially in ONJ.

Dentists, physicians, and natural health practitioners need to collaborate in an integrative manner in the treatment and management of jaw osteoporosis / osteonecrosis since such CIBD have both dental and systemic components that can have severe consequences on health as well as dental treatment decisions.

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Introduction to Through-Transmission Alveolar Ultrasonography (TAU) in Dental Medicine

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ABSTRACT: Through-transmission alveolar ultrasonography (TAU) is a novel imaging modality in dental medicine. A brief introduction to through-transmission ultrasonography (TTU) is followed by a description of the first commercially available TAU device, the Cavitat CAV 4000 (Cavitat Medical Technologies, Inc., Alba, TX). Recent associations between systemic osteoporosis, oral osteoporosis, periodontal diseases, and cardiovascular diseases underline the importance of early detection and treatment of oral cancellous bone pathologies associated with low bone density (LBD), such as regional ischemic osteoporosis, chronic nonsuppurative osteomyelitis, bone marrow edema, and cavitational ischemic osteonecrosis (osteocavitation). While the impact of osteoporosis on maxillofacial bones is acknowledged, there is a lack of reliable prevalence rate, and the National Institutes of Health (NIH) recommend that more attention should be paid to skeletal health, especially in persons with conditions known to be associated with secondary osteoporosis. TAU, a safe and effective imaging modality, can be a valuable tool in research as well as for the clinical assessment of alveolar cancellous bone pathologies associated with LBD and ischemia.

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Ultrasonography imaging technology, a safe and minimally invasive procedure, has been in use in medicine since the 1940s. This well proven modality is used to examine many parts of the body. Most commonly, pulse-echo ultrasound is used during the first, second, or third trimester of pregnancy. The American Institute of Ultrasound in Medicine indicates that the prudent use of diagnostic ultrasound for medical indications offers benefits that outweigh any risks that may be present, even in obstetrics. In spite of this, its use in dental medicine has been limited and mainly restricted to soft tissue applications such as parotid gland pathologies.¹ More recent research has focused on potential applications of ultrasound for caries detection,^{2,3} imaging of the TMJ,⁴⁻⁶ gingival thickness assessment in periodontology,⁷ mandibular fracture evaluation,⁸ and imaging of the cancellous portion of the maxillary and mandibular bones. Some investigators have been able to assess, to a certain degree, osteolytic lesions of the mandible, and even periapical infections, with pulse echo-sonography.^{9,10}

In pulse-echo ultrasonography, the imaging capability is entirely dependent on reflected sound. The ultrasound wave pulsed by the transducer is reflected back to the

source, which also works as a receiver. The ultrasound wave characteristics are analyzed and converted into an image displayed on a monitor. Since a bone/soft tissue interface is highly reflective, a high fraction of the ultrasound is reflected, converted into heat by absorption or scattered by bone. This causes what is called an acoustic shadow, as structures in or behind the bone surface cannot be imaged by using conventional pulse-echo, B-mode grey-scale imaging.

Thus pulse-echo ultrasound, as used in other medical fields, has been of little diagnostic value for imaging the medullary portion of bones, hence it's limited use in dental medicine where imaging of calcified tissues is of primary importance.

Through-transmission ultrasonography (TTU), however, does not rely on reflected sound waves for imaging. In TTU, the system will only measure the acoustic wave going through a given medium instead of the echo being reflected back at the transducer.

The reflective method, also known as pulse-echo ultrasonography, has been the modality of choice in medical imaging since the 1940s. However, the past decade has seen a resurgence of TTU with the development and commercial introduction of a number of through-transmission systems.

Through-transmission ultrasonographic imaging of the cancellous portion of the alveolar bone as an aid to assessing alveolar cancellous bone density and quality is the focus of this article.

Display Modes

There are various types of displays for ultrasound imaging. Presently the only TTU device commercially available for imaging the alveolar bone is the Cavitat Ultrasonograph CAV 4000 (Cavitat Medical Technologies, Inc., Alba, TX) which uses a modified C-Mode display and is defined as through-transmission alveolar ultrasonography (TAU).

Unlike the B-Mode, which is a 2D display of a tissue section along the propagation axis of the beam, C-Mode is a plane view of a slice of the specimen perpendicular to the ultrasound beam axis (**Figure 1**). C-Mode can be used in pulse-echo or through-transmission. In the through-transmission mode, the image contrast is derived primarily from differences in attenuation and/or time of flight of the ultrasound wave as it passes through the entire specimen.¹¹ Until recently it was used mainly in non-destructive type testing in industry.

The Cavitat CAV 4000 uses a 2D color display (**Figure 2**) that is extrapolated as a 3D view (**Figure 3**) that can be rotated or magnified by the operator.

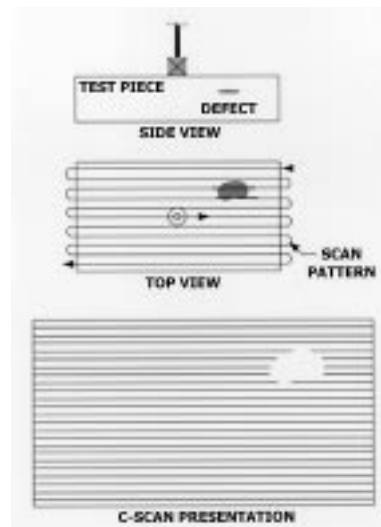


Figure 1
Schematic representation of C-Mode.

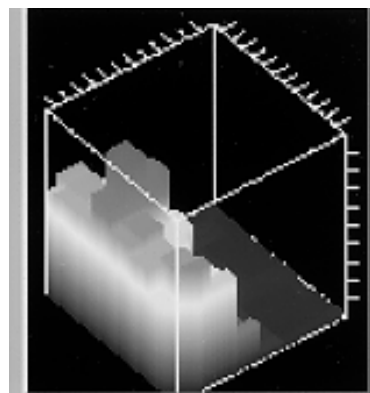
Tissue Types

The characteristics of ultrasound are closely related to the media which can be broadly divided into gas, liquid and solid. Tissues, because of their high liquid content,



Figure 2 (above) 2D T Scan.

Figure 3 (below) 3D T Scan.



can be considered liquids. Because biological tissues are heterogenous, the propagation of ultrasound varies according to tissue type (**Table 1**).

Of particular interest in TAU is fat, cortical and cancellous bone. Cortical bone is the thick external layer of the alveolar ridge encapsulating the cancellous part. Of the two, cancellous bone is the more complex tissue. From an acoustical point of view, it is far from an "ideal medium," since it consists of an anisotropic, heterogeneous open-celled framework of mineralized tissue saturated in marrow fluid. Normal cancellous bone, including jawbone, consists of moist, relatively dense, relatively uniform and highly connected trabeculae.¹²⁻¹⁵

The specific region of interest in TAU is the alveolar process which is integrated into the maxillary and mandibular body. It is defined as the part of the maxilla and the mandible that forms and supports the sockets of the teeth. Anatomically, no distinct boundaries exist between the body of the maxilla or the mandible and their respective alveolar processes. The marrow spaces in the cancellous bone of the alveolar process may contain hematopoietic marrow, but they usually contain fatty marrow.¹⁶

As complex as healthy cancellous bone can be, it is even more complex in a pathological state. The level of complexity depends on the pathological nature of the changes in the cancellous bone. These changes can affect both the calcified structure and the soft tissue marrow.

Osteoporosis

Osteoporosis is a disease characterized by low bone mass and structural deterioration of bone tissue, leading

to bone fragility and an increased susceptibility to fractures, especially of the hip, spine, and wrist. It is generally categorized as primary (idiopathic) or secondary, depending on the absence or presence of associated medical conditions, surgical procedures or medications known to be associated with accelerated bone loss.¹⁷ Secondary osteoporosis is sometimes a purely local phenomenon, confined to particular bones or parts of them.¹⁸

Osteoporosis is a different disease from osteomalacia. It results from abnormal organic matrix formation rather than abnormal bone calcification. In general, in osteoporosis, the osteoblastic activity is less than normal, and consequently the rate of bone deposition is depressed. Multiple factors can be involved, including but not limited to, lack of use of bones, malnutrition to the extent that sufficient protein matrix cannot be formed, deficiency in vitamin C (which is apparently necessary for the secretion of intercellular substances by all cells including osteoblasts), postmenopausal lack of estrogen secretion (estrogens have an osteoblastic stimulating activity).¹⁹ The increase in venous thromboembolism related to HRT must also be evaluated when estrogen replacement is considered in osteoporosis.²⁰ A recent study concluded that estrogen plus progestin increases the risk of ischemic stroke in generally healthy postmenopausal women. Excess risk for all strokes attributed to estrogen plus progestin appeared to be present in all subgroups of women examined.²¹ Ischemia can also have deleterious effects on bone mineralization.^{22,23}

The diagnosis of osteoporosis is complicated by the fact that osteoporotic bone loss cannot be detected by ordi-

Table 1
Typical Ultrasound Velocity, Characteristic Acoustic Impedance and Attenuation in Different Biological Tissues for Temperatures Between 20°C and 37°C. These Values Based on Published Data by Wells³⁹ and Njeh⁸³ Are Only Indicative Due to Biological Variability

| Tissue | Ultrasound propagation velocity V (m/s) | Characteristic acoustic impedance Z ($10^6 \text{ kg m}^{-2}\text{s}^{-1}$) | Slope of attenuation coefficient (dB cm ⁻¹ MHz ⁻¹) |
|-----------------|--|--|--|
| Water | 1480 (20° C) | 1.48 | 0.002 |
| Air | 340 (20° C) | 0.00044 | 1.2 |
| Blood | 1566 | 1.66 | 0.2 |
| Cancellous bone | 1450-1800 | - | 10 - 40 |
| Cortical bone | 3000-4000 | 4.0 - 8.0 | 5 |
| Fat | 1450 | 1.38 | 0.6 - 0.8 |
| Liver | 1560 | 1.65 | 0.6 - 0.9 |
| Muscle | 1550-1660 | 1.65 - 1.74 | 0.5 - 3.5 |
| Skin | 1600 | 1.6 | 2.4 |

nary clinical examination, even with the help of routine blood tests or x-rays.²⁴ Thus, special investigations using the most modern methods are necessary. TAU is such a method.

Velocity of Sound and Cancellous Bone

Velocity of sound in cancellous bone has a definite linear correlation with density.^{25,26} Velocity is a good indicator of bone density loss caused by such factors as thinning of the trabecular network, found in primary osteoporosis, and loss of trabecular structure, found in more severe forms of osteoporosis such as ischemic osteoporosis and osteonecrosis.²⁷⁻²⁹

Attenuation of Sound and Cancellous Bone

Attenuation of sound in cancellous bone does not have a linear relationship with density.³⁰ The strongest correlation between attenuation and density exists at lower density and only over a limited density range.³¹ In the published literature, explanations for this nonlinear behavior center around the role of scattering and absorption.^{25,31,32} Since the data has been almost exclusively qualitative, there is a need to develop a more detailed understanding of attenuation in cancellous bone, because factors other than bone density are involved.

Bone Marrow and Ultrasound Transmission

A number of studies have looked at the influence of bone marrow on ultrasound. Alves,³³ who measured bovine cancellous bone at one MHz, found an increase in velocity of 2.9% when bone marrow was replaced by water, but a decrease of 8.8% in specific attenuation under the same conditions. In human cancellous bone, Nicholson³⁴ found that marrow decreased velocity and increased attenuation compared to water saturated bone. Of particular interest is the fact that bone marrow seems to have a proportionally greater effect on attenuation than velocity.

Cancellous Bone—The Role of Ischemia in Osteoporosis and TAU

Osteoporotic trabecular bone is characterized by relative dehydration, trabecular thinning, trabecular disruption (loss of connectivity, loss of continuity), and irregularity in the calcified structure.³⁵

Under ischemic conditions, numerous pathological changes in the bone marrow and trabeculae of oral cancellous bone have been documented. Microscopically, areas of “apparent fatty degeneration and/or necrosis, often with pooled fat from destroyed adipose cells (oil cysts) and with marrow fibrosis (reticular fatty degeneration)” are seen. These changes are present even if “most

bony trabeculae appear at first glance viable, mature, and otherwise normal, but closer inspection demonstrates focal loss of osteocytes and variable micro cracking (splitting along natural cleavage planes).” “The microscopic features are similar to those of ischemic or aseptic osteonecrosis of long bones, corticosteroid-induced osteonecrosis, and the osteomyelitis of caisson (deep-sea diver’s) disease.”³⁶

In the cancellous portion of the femoral head, it is not uncommon to find trabeculae with apparently intact osteocytes which seem to be “alive” but are no longer synthesizing collagen.³⁷ This appears to be consistent with the findings in alveolar cancellous bone.

Collagen in trabecular bone, or lack thereof, has also been shown to have a greater effect on attenuation than velocity. Hoffmeister, et al., reported in decollagenized cancellous bone specimens, an increase in attenuation of between 35 and 77% compared to a decrease in velocity of 10 to 12%. In demineralized specimens the increase in attenuation was between 44 and 58% and the decrease in velocity of 19-39%.³⁸

In theory, the formation of areas of pooled fat (oil cysts) in diseased bone marrow should have a relevant influence on acoustic behavior. The available data seems to point in that direction. Fat itself is significantly more attenuating than water while velocity is only slightly less in fat than water (**Table 1**). Thus a higher concentration of fat in a specific area of medullary bone should have a greater effect on attenuation than velocity.

Since ischemically damaged cancellous bone will exhibit significant degenerative marrow changes before any easily detectable structural changes in the trabecular structure, the effects of such changes on ultrasound transmission need to be further studied.

Another factor is specular reflectance due to the smooth surfaces of steatocytes and oil cysts which will further attenuate acoustic energy. While velocity is a good indicator of cancellous bone mineral density, as discussed earlier, could attenuation be a better indicator of qualitative changes such as degenerative marrow changes in the absence of any significant structural alteration of oral cancellous bone? A hypothesis that remains to be proven.

Njeh, et al.,²⁶ raised this very issue of the distinct abnormality of ultrasound wave propagation in low density cancellous bone stating that further studies were clearly needed to find an explanation. One possible explanation that we propose is that while low density bone can be simply the result of thinning of the trabecular structure with a healthy bone marrow content, it can also be the result of a more severe pathological process in which loss of bone density occurs only in the later stages

of the disease and is preceded by a pathological degeneration of the bone marrow content as a result of ischemia³⁷⁻³⁹ and/or other known or unidentified oral and/or systemic factors.

Through-Transmission Ultrasonography (TTU) Devices

As mentioned previously, a number of TTU devices, called Quantitative Ultrasound System (QUS), are now commercially available. All the QUS devices are designed as bone densitometer including through-transmission alveolar ultrasonography (TAU).

These systems have been developed mainly for use in medicine as an alternative to ionizing radiation for assessing bone mineral density (BMD). Loss of bone density, or osteoporosis, a significant medical problem in the human population, is usually diagnosed based on information obtained with x-ray based devices such as quantitative computed tomography (QCT) and single or dual energy x-ray absorptiometry (SXA-DXA), which is relatively expensive and bulky medical imaging devices that have the disadvantage of using ionizing radiation.

In most dental offices, bone density and quality is assessed using intraoral radiography and/or orthopantomography, which are excellent imaging tools when cortical damage is involved, but have been shown to have significant limitations when cancellous bone is concerned.⁴⁰⁻⁴⁵ Although recent advances in digital radiography have enhanced radiographic imaging, the limitations regarding cancellous bone imaging²⁴ as well as the dangers of ionizing radiations are still an issue. Thus, additional imaging modalities are required to assist dental clinicians in their effort to measure oral BMD, changes in trabecular bone density/quality and to detect oral osteoporosis, especially since an association between reduced BMD and periodontal status has now been established and there is evidence that patients with systemic osteoporosis are likely to have decreased oral bone density as well.^{46,47}

TAU and the Cavitat Ultrasonograph CAV 4000

The TAU device, the Cavitat Ultrasonograph CAV 4000, was developed by engineers at Cavitat Medical Technologies. It became commercially available in February 2002 and uses ultrasound attenuation (UA), time of flight (TOF), and a third proprietary method of signal analysis to assess medullary bone quality and density.

The device is based on a frequency of 2.5 MHz. The inventors have found this frequency to be optimal for penetration through alveolar bone. The acoustic wave is

pulsed at a rate of 27,000 per ms via a proprietary flat piezoceramic transducer with an acoustic intensity of 0.4mW/cm². These parameters are significantly different from other commercially available devices. For example, reflective devices will use a repetition rate of between 1000 to 5000 times per second and most manufacturers of QUS devices use a lower range of ultrasound frequencies, ranging from 0.25 to 1.25 MHz.

The display includes a simulated oscilloscope showing the percentage of maximum amplitude of the acoustic wave in its y axis and time in ms on its x axis. The operator can observe the rate of attenuation of the wave in real time.

Attenuation is measured as the signal strength of the acoustic wave against the intraoral sensor array which is made up of 64 individual transducers bonded to a square piezoelectric membrane. The transducers convert the energy of the acoustic signal into a microelectrical signal analyzed by an AMD central processor and proprietary software to display a 2D and 3D color image. Information from each transducer of the sensor array is displayed as a square in the 2D view and as a vertical column in the 3D view. Each exposure represents the average of at least eight separate acoustic pulses against each of the 64 transducers of the array. The total image is made up of 64 squares/columns. The image is color coded to display 256 levels, or units, of attenuation and the height of each column is linked to the level of attenuation. Normal bone is imaged as dark green and increasingly attenuated bone is represented by light green, yellow/green, yellow (moderate loss), orange, orange/red, and red (most severe attenuation). Accordingly, maximum column height is associated with dark green (256 units) and minimum column height with dark red (six units).

The colors displayed are nonlinear and a color palette is always visible on the screen for immediate reference while reading the image (**Figure 4**).

According to the manufacturer, it is the lack of connectivity and thickness of trabeculae that determine signal strength (attenuation) and therefore, the height of the three-dimensional column display.

The patent on which the Cavitat CAV 4000 is based and the FDA 150K market approval indicate attenuation as the mechanism of action. In addition, the manufacturer explains that TOF is used to localize intra-medullary areas of LBD (personal communication with Cavitat Medical Technologies, 8/25/03). TOF is displayed on the x axis of the oscilloscope. Thus, TOF is the measure of the time taken, in microseconds, by the acoustic wave to travel through tissues, from the extra-oral transducer to the intraoral sensor array:

$$\text{TOF} = d/V_u$$



Figure 4
Nonlinear color palette.

Where d is the distance between the transducer and sensor and V_u is the velocity of ultrasound per square millimeter.

The Cavitat Ultrasonograph CAV 4000 is a pulse-transmission ultrasonic imaging device specifically designed for imaging the cancellous bone inside the alveolar ridges of the maxillary and mandibular bones (**Figure 5**).

Scanning Technique

A food-grade coupling gel (aloe vera) is applied to the extraoral transducer (emitter) to insure good acoustic conductivity and is placed on the facial skin of the patient. Gel may also be applied intraorally in the buccal sulcus to insure continuous acoustic contact between the buccal mucosa and the gingival tissues, especially in cases where the anatomical contour of the buccal aspect of the ridge is conducive to air bubble entrapment. The incoming ultrasound waves are received by the digital sensor array which is imbedded in the tip of a rigid wand, so careful positioning of the tip relative to the extraoral transducer and the alveolar ridge is important. A sterile transparent plastic sleeve is filled with coupling gel and positioned over the tip and any bubbles inside the gel must be carefully extruded. Gel is also applied on the tip once the sleeve is in place to insure good acoustic conductivity. The tip is positioned intra-orally on the lingual or palatal surface of the alveolar ridge, parallel to the extraoral transducer and the alveolar ridge. The goal is to have a perpendicular path of entry of the acoustic wave



Figure 5
Cavitat Ultrasonograph with extraoral transducer and intraoral digital sensor array.

into the alveolar ridge so as to minimize refraction (**Figure 6**).

The process of TAU imaging is known alternatively as a Cavitat Scan, a TAU Scan or τ Scan. A "recall" button allows the operator to access stored 3D data from previous τ Scans (**Figure 7**). The information on the monitor can be printed as a form which also includes patient details. A copy can be provided to the patient immediately following the τ Scan (**Figure 8**).

As with any new imaging technique, the scanning procedure and image interpretation requires training and a certain amount of skill as placement of the sensor and transducer in relation to anatomical structures is very important. Hence, this is a user sensitive technique and the above instructions are a simple introduction. Training is definitely required and should be provided by professionals who have expertise in TAU as well as in the relevant clinical sciences.

TAU Grading

A grading system based on a histopathological confirmation of 285 scanned alveolar sites has been developed for the Cavitat Ultrasonograph and presented to the FDA as part of the approval process.^{51,52} As explained previously, each column is divided into 256 units of attenuation displayed in a nonlinear color scale. The green range represents a column height of 129-256 units, while yellow represents a column height of 91-128 units, brown of 75-90 units, orange of 65-74 units and red of 6-64 units. As described in **Table 2**, the grading system is designed to classify the degree of alveolar cancellous bone attenuation so as to facilitate image interpretation and treatment planning.

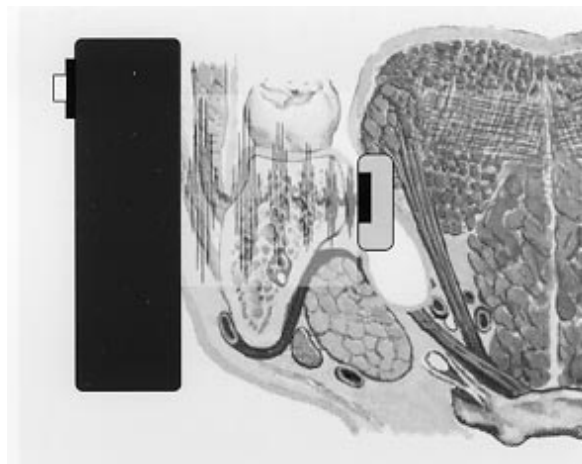


Figure 6
Proper positioning of transducer and intraoral sensor array.

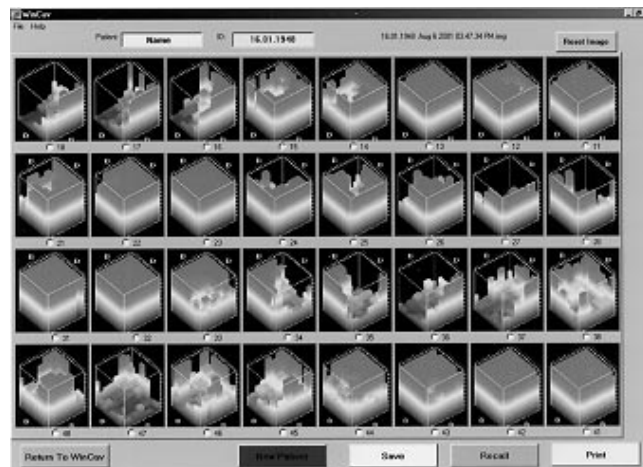


Figure 7
3D data display of all four quadrants.

At present, based on available clinical evidence, it is suggested that only high grade (grade III & IV) lesions should be considered for conservative surgical exploration in the absence of very strong radiographic changes.

Limitations and Error Sources in TAU

TAU measures pulsed ultrasound transmission through 32 different alveolar sites in the oral cavity. This is more

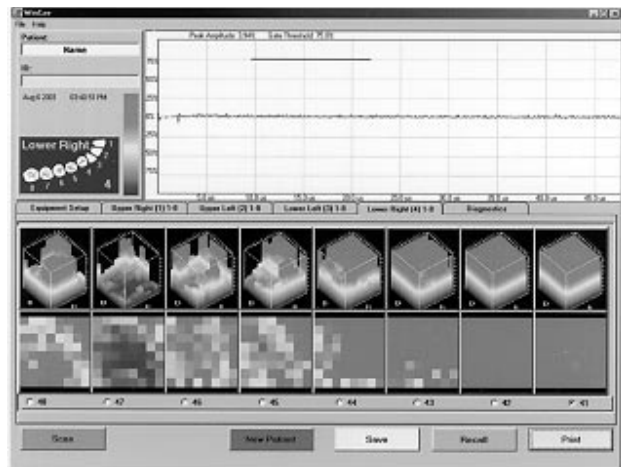


Figure 8
Information as displayed on the Cavitat CAV 4000 LCD screen.

sites than any other bone densitometer on the market, as far as the author is aware. The maxillary and mandibular bones have a unique combination of morphological structure and associated dental elements.

Potential sources of error when imaging the maxillary and mandibular alveolar bones in vivo with TAU are related to:

- anatomical configuration of the specimen;
- transducer and sensor array positioning;

Table 2
Grading Categories for Individual 3D Cube Images (64 columns in each) of T Scans

| τ Scan Grade | Description* |
|-------------------|--|
| 0 | Cube shows no loss of column height and is 100% green; or mild loss of column height in less than 1/4 of columns; and/or moderate to severe loss of column height in less than 3 nonadjacent columns |
| 1 | Cube shows mild loss of column height in more than 1/4 of columns; and/or moderate loss of column height in less than 1/4 of the columns; and/or severe loss of height in less than 1/8 of the columns |
| 2 | Cube shows moderate loss of column height in 1/4 to 1/2 of columns; and/or severe loss of height in less than 1/4 of columns |
| 3 | Cube shows moderate loss of column height in more than 1/2 of columns; and/or severe loss of column height in 1/4 to 1/2 of columns |
| 4 | Cube shows severe loss of column height in more than 1/2 of columns |

*Definition of loss of column height: mild (crown is green, less than 1/3 loss of height); moderate (crown is yellow or brown, 1/3 to 2/3 loss of height); severe (crown is orange or red, more than 2/3 loss of height).

- bone properties and tissue thickness;
- acoustic transmission between the various media;
- patient cooperation.

Limitations related to anatomical configuration can interfere with the proper positioning of the sensor array against the lingual or palatal aspect of the alveolar ridge.

Errors induced by variations in the positioning of the transducer and sensor array are related to geometric optics which will impact on the accuracy of the imaging since, unlike x-rays that always travel in a straight line, acoustic radiation is sensitive to the angle of entry as described by Snell's law which describes the relationship between the angles and the velocities of the waves. Snell's law equates the ratio of material velocities V_{L1} , and V_{L2} to the ratio of the angle of incident (\sin_1) and refraction (\sin_2) angle, as shown in the equation and graphic representation (**Figure 9**).

The formula (**Figure 9**) is valid for longitudinal waves which exist both in liquids and solids. Since cancellous bone is a heterogenous, anisotropic, dual phase medium, shear waves are also formed. This is called mode conversion (**Figure 10**).

Errors induced by bone properties include the variability of bone thickness, bone marrow composition and the ratio of cortical to trabecular bone. Since attenuation and TOF are dependent on distance travelled by the acoustic wave, measurements based on a standardized thickness instead of actual thickness are less than optimal. As long as the goal is to identify a change in bone density and/or quality, small variations in thickness are not as significant and this has been confirmed by a recent study involving the author and showing a high degree of correlation

between high grade TAU Scans and intramedullary pathological changes.⁵⁵ However, when the goal is the precise spatial localization of a defect in the specimen, accurate measurement of the thickness of the specimen as well as the thickness of soft tissue relative to the thickness of the alveolar ridge is necessary.

Another potential source of error with the Cavitat Ultrasonograph is the lack of a built in mechanism to regularly check calibration of the device. Manufacturers of QUS devices provide a phantom to allow the operator to verify the calibration of their devices on a daily basis. This can be done manually or through software integrated with the device. The Cavitat Ultrasonograph is calibrated by the manufacturer but a phantom or other suitable mechanism is not provided to allow the operator to routinely verify if the device is still properly calibrated.

For these reasons and others that may yet to be identified, the author recommends that the three golden rules of ultrasound imaging be followed⁴⁸:

1. Use more than one image to make an interpretation;
2. Just because a feature is displayed do not consider that it is necessarily real;
3. Just because a feature is not displayed do not consider that it is necessarily not there.

Effectiveness of TAU

Early investigations with porcine mandibles have determined the safety of the generated sound waves and also determined the optimal frequency for complete transmission through alveolar bone as well as the detection of intra-medullary defects⁴⁹ (**Figure 11**).

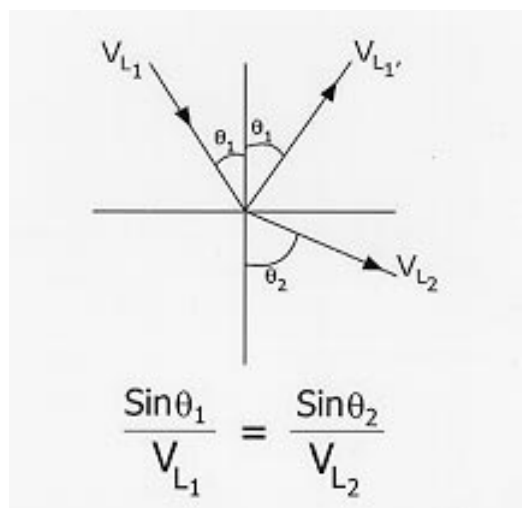


Figure 9
Refraction of an acoustic wave between two mediums of the same type but with different acoustic impedance.

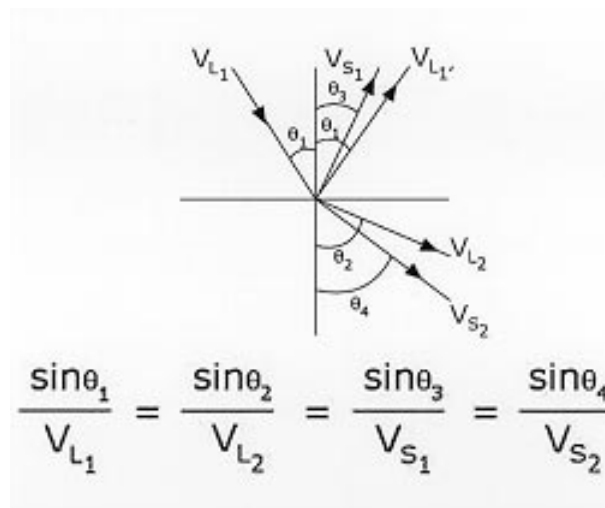


Figure 10
In solids shear waves are produced and are refracted at a different angle due to their lower velocity.

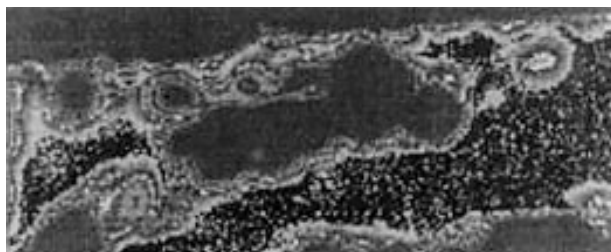


Figure 11

The first prototype version of the Cavitat was tested by the New Mexico Institute of Technology on porcine jaws for safety and effectiveness. Holes were drilled in the jaws and sound was then sent through the jaws in an attempt to see the holes. The sonogram shows several rounded holes (the drilled holes), but also shows several irregular hollow marrow spaces.

As an ultrasonic imaging device, the Cavitat Ultrasonograph was tested for acoustic output in accordance with Food and Drug Administration (USA) requirements for diagnostic ultrasound to confirm the safety of the device. The effectiveness of the device was demonstrated by two clinical studies in which both radiographs and ultrasound based, 3-dimensional images of alveolar sites were compared and confirmed with histopathology examination. The result of the studies confirmed that the device is effective as an adjunct to standard radiographic evaluation and clinical diagnostic procedures.^{51,52}

The TAU device received regulatory approval from the FDA in February 2002 and from Health Canada in July 2002.⁵³

Additional clinical studies have confirmed the effectiveness of the Cavitat Ultrasonograph for alveolar cancellous bone density and quality evaluation. One study compared radiographic images and τ scan images from 72 human subjects and 170 scanned alveolar sites which had been biopsied and given a microscopic diagnosis of osteoporotic or ischemic medullary disease. The images were graded according to a 4-point scale relative to severity of image alteration from normal. Of the 72 patients, 68% were women, and 82% were 40-69 years of age at diagnosis. Of the 170 imaged sites, both jaws were evenly distributed and 57% of the lesions were located in the retromolar/third molar region; 83% were in edentulous areas. Thirty-five (35%) of the radiographs were completely "normal" (false negative tests), while only one lesion was completely "normal" on τ scans. The average grade for radiographs of osteoporotic regions was 1.1 (median: 1; 95% CL: 0.92-1.22) compared to an average grade of 3.4 (median: 4; 95% CL: 3.18-3.43) for τ scans. The average grade for radiographs of ischemically damaged bone was 0.8 (median: 1; 95% CL: 0.65-1.01), compared to 3.5 (median: 4; 95% CL: 3.39-3.61) for τ

scans. Eighty-six percent (86%) of positive scans were high-grade, i.e., grade III & IV lesions while only 9% of positive radiographs were high-grade. Therefore, it was concluded that τ scan typically showed great image alteration for ischemic and osteoporotic bone and that TAU imagery appeared to be significantly superior to routine dental radiology for detection of osteoporotic and ischemically diseased alveolar cancellous bone.⁵⁴

Another study⁵⁵ was conducted to correlate τ scans with medullary histopathology in a large number of cases and to determine the proportion of false positive results. The study was based on a total of 3,522 scanned alveolar sites with 339 sites, in 125 patients, biopsied after τ scanning. Diagnoses were categorized into five broad disease types, which were then correlated with graded (4-point grade scale, see section entitled TAU Grading) τ images of the biopsied sites. Average patient age was 52 years, with 65% being female. Seventy-seven percent (77%) of lesions were in the molar/retromolar region. There was a slight predilection for the maxilla. Osteoporotic and ischemically damaged bone accounted for 61% of all microscopic diagnoses and 74% of suspicious sites had high-grade τ images, i.e., were Grade III or IV. The mean grade for these two disease types was 3.5 (median: 4) compared to 1.8 for normal bone; 95% CIs did not overlap. The level of false positive τ scans was approximately 2%, so the conclusion was that τ imaging appears to be an excellent adjunctive tool for identifying ischemically damaged and osteoporotic alveolar bone. However, the t -test is technique-sensitive, and mistakes in technique can give false positive results, so the authors recommended that only images rated as Grade III or IV be considered for surgical exploration.

It should be noted that in the above-mentioned study, TAU demonstrated a relatively poor ability to detect periapical and odontogenic inflammatory lesions. Odontogenic cyst and fibroma, while well demarcated on radiographs, scanned as grade 0 or 1, i.e., negative τ scans. This is likely due to the fact that sound travels so well within the peripheral bony wall surrounding most periapical pathoses, developmental odontogenic cysts and benign odontogenic neoplasms. Conversely poorly radiographically demarcated lesions lacking a high-conduction peripheral wall, such as a myxoma, scanned as a high Grade III lesion.

Clinical Significance of TAU for Oral Bone Densitometry

The National Institute of Health (NIH) considers osteoporosis as a devastating disorder with significant physiological, psychological, and financial consequences.

While its impact on craniofacial bones is acknowledged, there is a lack of reliable prevalence rate and the NIH recommends that more attention should be paid to the skeletal health, especially in persons with conditions known to be associated with secondary osteoporosis.⁵⁶

Recent investigators have demonstrated a significant association between BMD of the mandible and the peripheral skeleton in postmenopausal women. Some studies also have linked low BMD of the mandible and the peripheral skeleton with alveolar bone loss of the mandible and tooth loss.

In a review of the literature in 1997, Hildebolt⁵⁷ concluded that an association between osteoporosis and oral bone loss existed while recommending additional longitudinal studies. He suggested that inexpensive methods must be developed for sensitive and specific measures of oral bone loss.

More recent studies^{58,59} have cited osteoporosis as a risk factor for periodontal disease even while their association is still not well understood. There is evidence that patients with systemic osteoporosis are likely to have decreased oral bone density, which may affect treatment decisions. Further, patients with decreased bone mineral density, indicative of osteoporosis, may be at a higher risk for periodontitis. Therefore, osteoporosis, could be considered a risk factor for periodontitis.⁶⁰

In another study⁶¹ based on 11,655 subjects in the US (5733 males and 5922 females), possible association of periodontal disease with femoral BMD were investigated. Women with high calculus scores and low BMD had significantly more clinical tooth attachment loss than those with normal BMD and similar calculus scores. These findings suggest that in presence of high calculus score, females with osteoporosis were at increased risk for tooth attachment loss. Other studies^{62,63} have concluded that available evidence supported the role of systemic bone loss in the development of tooth loss among postmenopausal women. However, because so many possible factors contribute to the development of osteoporosis and periodontal diseases, it is difficult to establish a direct correlation between tooth loss, bone loss and loss of attachment resulting from periodontitis and decreased BMD associated with osteoporosis, though studies are ongoing.⁵⁰

In general, the interpretation of the existing literature is difficult due to the different methods used to measure osteoporosis and oral bone loss. Most of the studies so far are confined to postmenopausal women, and several other acquired, behavioral, local and systemic factors contributing to bone loss such as genetics, hormone intake, gender, age, race, smoking, diet, oral hygiene, occlusal trauma, psychological stress, socio-economic, as

well as immunological factors need to be addressed when assessing the relationship between osteoporosis and periodontitis.

Numerous studies have established an association between periodontal diseases and various systemic diseases including cardiovascular diseases. For example, DeStefano, et al.⁶⁴ focused on the contribution of periodontitis and analyzed coronary heart disease and mortality outcomes in nearly 10,000 subjects followed for 14 years longitudinally in the NHANES I study. Periodontitis for this cohort study was assessed with the periodontal index. Overall, subjects with periodontitis had a 25% increased risk for coronary heart disease relative to those with minimal periodontal disease. This association occurred after adjustments for potential confounders like age, gender, race, education, marital status, systolic blood pressure, total cholesterol levels, body mass index, diabetes, physical activity, alcohol consumption, poverty and cigarette smoking. For males younger than 50 years, periodontitis more strongly affected the incidence of coronary heart disease with a relative risk of 1.72.

Lately, data regarding the periodontal microbial challenge support the biological plausibility of the associations seen in human population studies. Hertzberg, et al.,^{65,66} have reported that two oral microbes, streptococcus sanguis and p. gingivalis, express a collagen-like platelet aggregation-associated protein that can stimulate thrombotic events. Genco, et al.⁶⁷ presented preliminary data that suggest an odds ratio of 2.8 for subjects harboring p. gingivalis in periodontal pockets and exhibiting a myocardial infarction. In addition, Zambon, et al.,⁶⁸ recently isolated DNA sequences specific for periodontal pathogens like P. gingivalis and A. actinomycetemcomitans from human atheroma specimens using polymerase chain reaction (PCR) techniques. Other nonperiodontal infectious agents like clamidia pneumoniae, heliobacter pylori, herpes simplex, and cytomegalovirus have been previously detected in atheromatous lesions using similar methods, and further support an infectious etiology for cardiovascular disease.⁶⁹

There is also mounting evidence supporting the association of cardiovascular diseases and osteoporosis. Calcification is a common feature of atherosclerotic plaques, and osteoporosis is associated with both atherosclerosis and vascular calcification.^{70,71}

The association of periodontal diseases with oral/systemic osteoporosis and cardiovascular diseases, in light of the association of cardiovascular diseases with osteoporosis, is interesting since other medullary oral bone pathologies associated with LBD such as regional ischemic osteoporosis (RIO), bone marrow edema (BME) and ischemic osteonecrosis (IO) with its many hollow,

air-filled spaces and its dry bone and fibrous marrow, have been identified^{72,73} and linked with hypercoagulability,⁷⁴⁻⁷⁸ a known risk factor in cardiovascular diseases. IO has also been associated with chronic facial pain.⁷⁹⁻⁸² The role of hypercoagulability in osteoporosis and periodontal diseases is thus an issue that warrants further research.

The available evidence supports early detection of oral osteoporosis as a sound clinical practice in the prevention and treatment of oral and systemic diseases associated with oral and systemic osteoporosis.

TAU has been demonstrated to be effective in imaging LBD in cancellous alveolar bone, and low bone density (LBD) is a characteristic of osteoporosis and related bone pathologies. The efficacy of the use of ultrasound in this application is also supported by previously published data showing a significant linear correlation between ultrasound velocity and cancellous bone density.²⁶

TAU can provide valuable additional information on alveolar bone density which, used in combination with radiography, can assist the clinician in the diagnosis and treatment of cancellous bone pathologies associated with LDB (**Figure 12**).

Conclusions

TAU imaging is a safe, effective medical modality and a valuable clinical tool that can help clinicians in the early detection, diagnosis and treatment of the various forms of oral osteoporosis and associated oral pathologies.

Information derived from a τ scan is not meant to be used purely on its own, but as part of a comprehensive

clinical assessment using other established imaging modalities and diagnostic procedures. It is then that information on bone density and quality derived from TAU is most useful and can be used in various fields of dental medicine, including periodontology, maxillofacial surgery, maxillofacial pathology, implantology, endodontics and general dentistry.

The Cavitat Ultrasonograph has a small footprint that can be installed in almost any dental surgery to complement existing radiographic equipment and to provide additional diagnostic information on oral bone density and quality. It could also facilitate clinical research into oral etiological factors involved in various form of oral osteoporosis which, in combination with known systemic factors, could be aggravating factors in the disease processes.

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Disclaimer: *The author does not have any financial interest in Cavitat Medical Technologies, Inc. He has purchased his own Cavitat Ultrasonograph and, although his name is listed on the company Board of Scientific Advisors, he has not received from Cavitat Medical Technologies any incentive, financial or otherwise, for his services.*

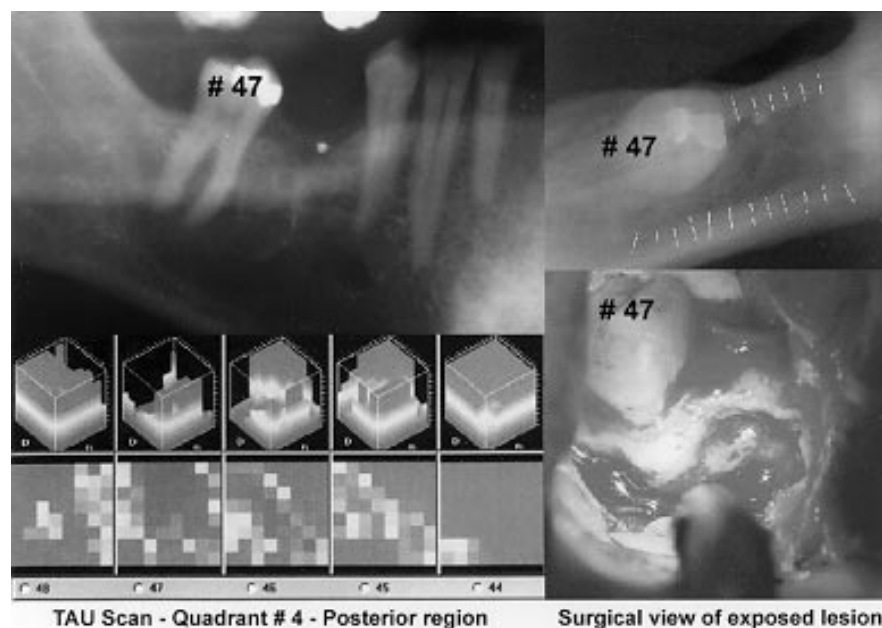


Figure 12

Lower right quadrant: a comparison of roentgenographic imaging, TAU imaging, and the actual lesion in the cancellous bone. The margins of the lesion have been highlighted on the occlusal film which shows the lesion more clearly than the orthopantomographic view, because the internal aspect of the occlusal cortical plate was damaged. The histopathological diagnosis in this particular case was regional ischemic osteoporosis (RIO). Tooth #47 (US #31) was vital and retained. This is a good example of the diagnostic benefits derived from the combination of roentgenographic and ultrasonographic imaging.

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United States Patent [19]

Jones et al.

[11] **Patent Number:** 6,030,221[45] **Date of Patent:** Feb. 29, 2000

[54] **ULTRASONIC APPARATUS AND FOR
PRECISELY LOCATING CAVITATIONS
WITHIN JAWBONES AND THE LIKE**

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[73] Assignee: **Cavitac, Inc.**

[21] Appl. No.: **09/021,951**

[22] Filed: **Feb. 11, 1998**

[51] **Int. Cl.**⁷ **A61C 5/00**; A61B 8/00

[52] **U.S. Cl.** **433/215**; 128/660.02; 128/660.01

[58] **Field of Search** 433/215; 128/660.02,
128/660.01; 601/2

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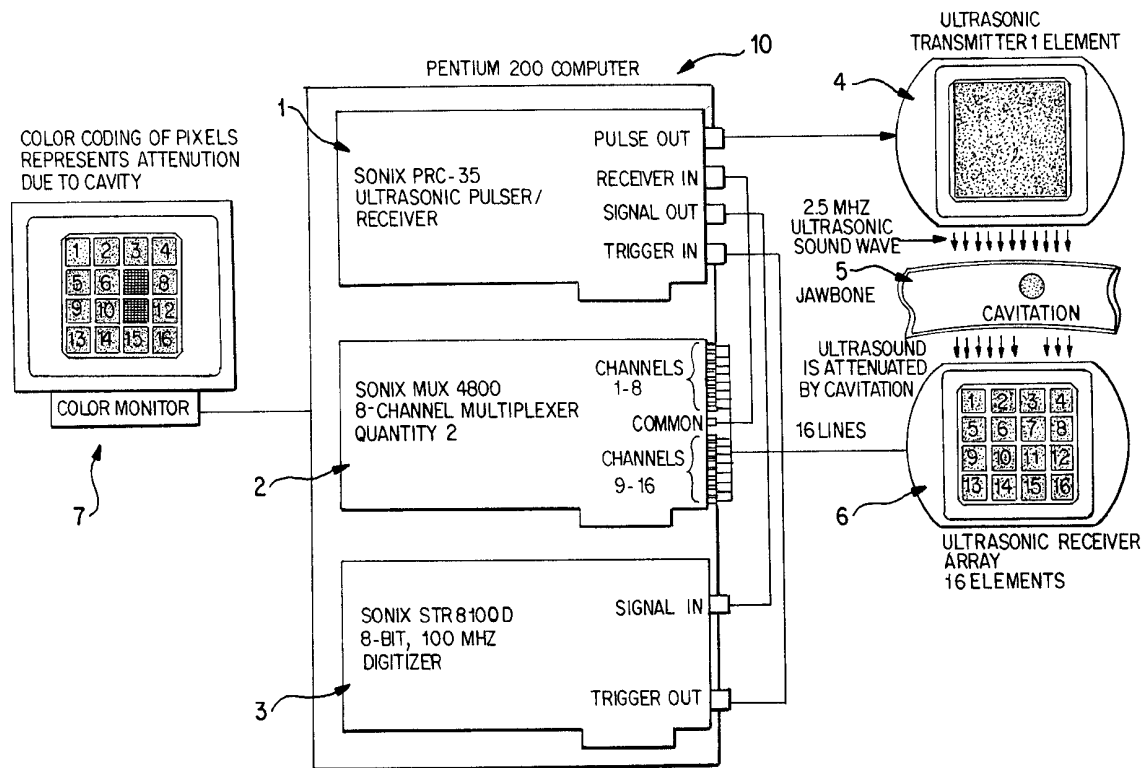
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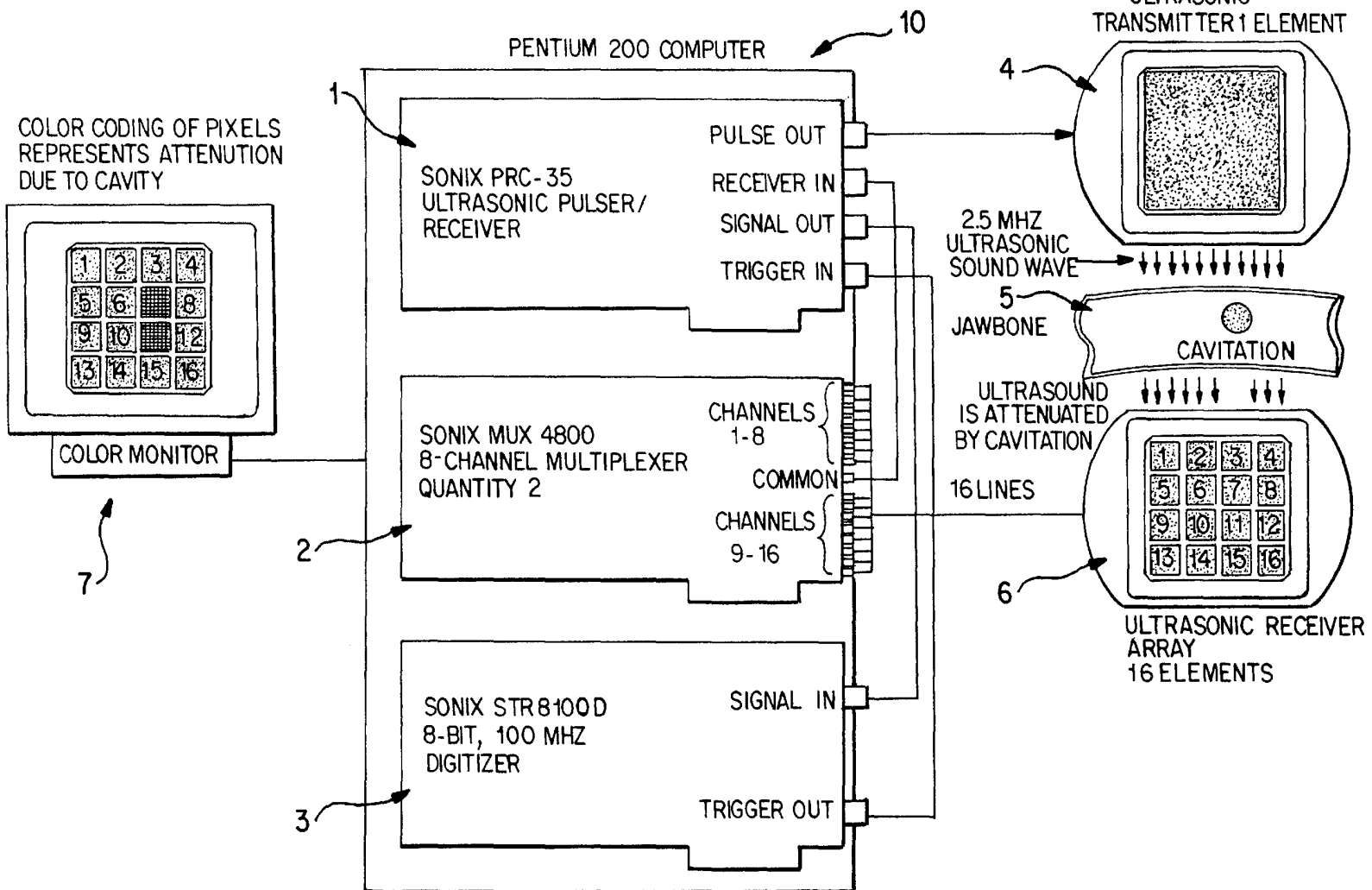
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Lenahan P.L.L.C.

[57] **ABSTRACT**

A method and apparatus detects cavitation in the jawbone of human. The apparatus generates an ultrasonic pulse and passes the pulse through the jawbone of a human. The pulse is detected by an ultrasonic receiving unit. Attenuations in the amplitude of the pulse are detected and displayed on a color monitor. The color monitor allows the detection of cavitations by interpreting color codes in a 4×4 matrix displayed on the monitor.

19 Claims, 1 Drawing Sheet



ULTRASONIC APPARATUS AND FOR PRECISELY LOCATING CAVITATIONS WITHIN JAWBONES AND THE LIKE

BACKGROUND AND SUMMARY OF THE INVENTION

The present invention relates to an apparatus and a method for locating cavitations and the like in the jawbone of a living being. More particularly, the present invention relates to an apparatus which comprises an ultrasonic transmitter and an ultrasonic receiver oppositely arranged around the jawbone of a patient to implement a method which introduces ultrasonic sound waves and determines the attenuation thereof. Thereby, a precise location and size of cavitations can be determined.

Ultrasound has long been used for a variety of applications including use in the medical field. Its predominant use has been to obtain two-dimensional soft tissue images, e.g. in a developing fetus or in a kidney. U.S. Pat. No. 5,402,781 describes a technique for measuring bone density and diagnosing osteoporosis using a frequency generator and a power amplifier to drive a transducer for inducing continuous vibration in hard tissue. Other bone and tissue analyzers are described in U.S. Pat. Nos. 4,610,255; 5,006,984; 5,518,0008; and 5,651,363.

In contrast to other techniques, ultrasound does not subject a patient to radiation. Also, it is less costly and the equipment is typically smaller in size and easier to use than X-ray equipment and the like.

More recently, ultrasound has been used to characterize the physical properties of cancellous bones using velocity of sound and broadband ultrasonic attenuation parameters which relate to elasticity, density and structure of bones. The attenuation of ultrasound in bone is derived by comparing signal amplitudes transmitted through cancellous bone with that of the same signal transmitted through water. In this connection, see *Role of Ultrasound in Assessment of Osteoporosis* (<http://www.mccueplc.com/ultrasnd.htm>).

Some recent medical studies suggest that there are significant health risks associated with jawbone infections resulting from cavities in the jawbone (hereinafter, cavitations). There is an emerging view of a relationship between chronic medical diseases and jawbone infections, e.g., emaciation, rheumatic diseases, heart and other circulatory problems. See, for example, Levy and Huggins, *Routine Dental Extractions Routinely Produce Cavitations*, Journal of Advancement in Medicine, pp. 235-249 (Vol. 9, No. 4, Winter 1996), George E. Meining D.D.D., F.A.C.D., *Root Canal Cover-Up*, 1996.

We have recognized that a considerable advantage to patient wellbeing can be gained by the ability to accurately detect the location of such cavitations within the jawbone of a human patient and that ultrasound technology is the most effective way to provide this ability.

The use of ultrasound technology in the dental field is known and has generally been limited to root canal treatment. During a root canal procedure, removing infected nerve endings and filling with an inert substance are required. Before the tooth under treatment is filled with a sterile substance, the canals of the teeth containing the infected nerve endings require cleaning to ensure that the entire nerve ending is removed and thus to prevent later reinfection. During this nerve removal and cleaning, the depths of the canals are increased. To aid in this procedure, prior art techniques disclose the use of ultrasound to either detect the depth of root canals, or to diagnose the health of teeth tissue (i.e., teeth pulp).

Another use for ultrasound technology in the dental field has been in the diagnosis of periodontal pockets. Periodontists can use metal probes to determine the depths of periodontal pockets during inspections for gum disease. The prior art teaches the use of ultrasound to determine the depths of periodontal pockets, with deeper pockets indicating the likelihood of gum disease. Thus, the need for the invasive probing associated with the prior art methods of measuring dental pockets would be eliminated.

One known method and apparatus for performing ultrasound measurements is described in U.S. Pat. No. 5,100,318. The presence of diseased gum tissue by measuring the depth of the periodontal pocket along an outer surface of a tooth. In particular, a first ultrasonic pulse travel path having a fixed, reflected delay time and a second ultrasound pulse travel path having a variable, reflected delay time are established. The difference between the fixed, reflected time delay time of the first ultrasonic pulse echo pulse reflected at the gum line and the variable, reflected delay time of a second ultrasonic echo pulse reflected from the bone surface at the bottom of the periodontal pocket is measured. The difference between these reflected delay times is displayed and indicates the depth measurement for the periodontal pocket.

U.S. Pat. No. 5,115,813 describes an ultrasound based measurement method and apparatus for examining dental tissue, in particular teeth. The method involves the use of ultrasound to determine the health of teeth by subjecting them to a low frequency vibration and determining the intensity and delay of returning echo signals in relation to the pulse transmitted to the teeth under examination. The intensity and delay of the echo signals is used to form a picture which dentists can use to determine the overall health of dental tissue (i.e., teeth pulp, root paths, etc.).

Dental root canal diagnosis and treatment are the subjects of U.S. Pat. No. 5,295,833. During a root canal procedure, a dentist uses a probing tool to clean the roots of an infected tooth. Usually the root is enlarged to ensure the complete removal of the nerve from the canal path. This procedure is generally performed based on the experience of the dentist and is, more or less, completed using trial and error. Dental diagnosing and treating equipment is proposed in this patent document to enable a dentist to determine the exact depth of root canals during such root canal procedures. Ultrasound is used in this known approach to detect the depth of the canal, thus improving the less precise conventional root canal cleaning procedure.

U.S. Pat. No. 4,485,823 also proposes a dental diagnostic apparatus using ultrasound. That is, the apparatus is intended to measure environmental tissue of the teeth and numerically identify the degree of health. To effect this measurement, the apparatus is provided with an oscillation converter for converting electrical oscillation into mechanical oscillation. A probe is connected to the converter and is brought into contact with a patient's tooth for applying the mechanical oscillation thereto. The patient has a means for actuation when he or she detects the sense threshold of the oscillation applied through the probe. This approach is intended to allow the dentist to more specifically diagnose the health of the tissue without visual examination.

Similarly, U.S. Pat. No. 3,883,954 discloses the use of acoustic vibrations produced by dental occlusions for providing a viewable display. U.S. Pat. No. 3,094,115 proposes to provide a tooth mobility measuring instrument using a piezoelectric transducer in a small probe intended for ready insertion into an oral cavity. A percussion instrument is

described in U.S. Pat. No. 4,499,906 whereby the degree of looseness of teeth is determined. Finally, U.S. Pat. No. 4,673,352 shows a device which uses transmit times of an ultrasonic pulse to measure relative jaw positions and movements.

Whereas the use of ultrasound has in the past been limited to soft tissue imaging, bone analysis and treatment of disorders of the teeth (i.e., root canals and gum disease), it is an object of the present invention to provide an apparatus and novel method using ultrasound for detecting with great accuracy cavitations within a person's jawbone, not the teeth themselves, thereby permitting medical personnel to undertake corrective action and prevent further complications or harmful side effects.

The foregoing object has been achieved in accordance with the present invention by generating an output pulse with an ultrasonic pulser/receiver and by configuring a multiplexer in order to output a single output to a common output from a plurality of sequentially selected input channels. An ultrasonic sound wave passed through the jawbone of a patient is subsequently received by a multiple-element (e.g., 16 elements) ultrasonic receiver array coupled to the multiplexer and converted into an electrical signal. An ultrasonic sound wave is generated using a single element ultrasonic transmitter or the like coupled to the ultrasonic pulser/receiver based on the digital output pulse generated by the ultrasonic pulser/receiver. A digital output trigger is produced and the electrical signal is converted into a digital value using a digitizer.

Cavitations within the jawbone of a patient are therefore located quickly, easily and precisely by displaying on the monitor the digital value produced from the signals of each of the array elements representing the attenuation of a sound wave generated by the ultrasonic transmitter element that is passed through the jawbone. Thus, the medical profession now has the ability to more easily treat cavitations which increasingly are believed to cause health problems.

BRIEF DESCRIPTION OF THE DRAWING

Other objects, advantages and novel features of the present invention will become apparent from the following detailed description of the invention when considered in conjunction with the accompanying drawing wherein the sole FIGURE is a schematic diagram of the novel cavitation detecting arrangement which, in relation to the jawbone of a patient, precisely detects cavitations in accordance with the method of the present invention.

DETAILED DESCRIPTION OF THE DRAWING

The sole FIGURE shows a portion of the jawbone **5** of a patient in which the presence and the location of cavitation represented by the solid dark circle is detected by emitting an ultrasonic sound wave via a one-element ultrasonic pulse transmitted by an ultrasonic pulser/receiver **1**. The ultrasonic pulser/receiver **1** of the type used to implement the invention is well known in the art and can be, for example, a commercially available one such as a SONIX PRC-35 manufactured by Sonix, Inc. of Springfield, Va. The location or inclusion of a cavitation within the jawbone **5** has the effect of attenuating the amplitude of the sound wave (shown by the parallel lines above the jawbone **5**) as it passes through the jawbone **5**.

A sixteen channel multiplexer (in practice, a unit comprising two 8-channel multiplexers, such as a Sonix model MUX 4800), is connected with the ultrasonic pulser/receiver **1** and a digitizer **3** such as a commercially available SONIX

STR8 100D, 8-bit, 100 MX digitizer also produced by Sonix, Inc. Multiple channels of the multiplexer **2** are sequentially selected. The one-element ultrasonic transmitter element **4** is excited by the pulser/receiver **1** when the latter is triggered by the digitizer **3** to produce an ultrasonic sound wave having a frequency of, for example, about 2.5 MHz. The ultrasonic sound wave is passed through the patient's jawbone **5** to detect and precisely locate the presence of one or more cavitations within the jawbone **5** by virtue of the attenuation of the strength of the wave amplitude.

In order to precisely determine and depict the exact location of the cavitations, a 16-element ultrasonic receiver array **6** is connected to the bus of the multiplexer **2** to detect the sound wave whose amplitude is proportional to the attenuation through the jawbone **5**. The ultrasonic receiver array **6** is positioned on the side of the patient's jawbone **5** opposite to the transmitter **4** and comprises an array of multiple transducer elements corresponding to the channels of the multiplexer **2**, e.g. sixteen in the illustrated embodiment. It will, of course, be understood that the number of channels and array elements can be varied without departing from the scope of the present invention.

The sound wave is converted into an electrical signal by the ultrasonic pulser/receiver array **6** and sent to the multiplexer **2**. The multiplexer **2** sequentially routes one of a plurality of inputs to a "COMMON" output.

This common output is fed to the digitizer **3**, where the electrical signal is converted into an 8-bit digital value that represents 256 levels. A processor, e.g. a "Pentium"® 200, located within a computer **10** processes the 8-bit digital data and displays, on a color monitor **7**, a 4 x 4 color coded image representing the attenuation of the sound wave through the patient's jawbone **5**. This attenuation represents the presence and precise location of a cavitation within the jawbone **5**. Of course, the results can also be printed out, remotely displayed at another location and/or stored for subsequent retrieval.

Although the invention has been described and illustrated in detail, it is to be clearly understood that the same is by way of illustration and example, and is not to be taken by way of limitation. The spirit and scope of the present invention are to be limited only by the terms of the appended claims.

What is claimed is:

1. An apparatus for detecting bone cavitations, comprising:
 - an ultrasonic pulser/receiver for generating an output pulse;
 - an ultrasonic transmitter coupled to the ultrasonic pulser/receiver for generating an ultrasonic sound wave based on the output pulse generated by the ultrasonic pulser/receiver;
 - a multiplexer operatively coupled to the pulser/receiver and configured to output a single output signal to a common output thereof from a plurality of input channels therein;
 - an ultrasonic receiver array operatively coupled to the multiplexer for receiving an attenuated ultrasonic sound wave passed through the bone and converting the same into an electrical signal supplied to the input channels of the multiplexer;
 - a digitizer for receiving a single output signal and converting the same into a digital value representing a predetermined number of levels;

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a processor for building up a color coded image from the data obtained from each channel representative of an extent of attenuations of the sound wave through the bone; and

a device for displaying the color coded image.

2. An apparatus according to claim 1, wherein the ultrasonic array has a plurality of receiving elements.

3. An apparatus according to claim 1, wherein the frequency of the output pulse is in the range of about 2.5 MHz.

4. An apparatus according to claim 3, wherein the frequency of the sound wave based on the output pulse is about 2.5 MHz.

5. An apparatus according to claim 3, wherein the digital output trigger has a frequency of about 100 MHz.

6. An apparatus according to claim 1, wherein the digital value is comprised of 8-bits representing 256 levels of data.

7. An apparatus according to claim 1, wherein the displaying device is a monitor for visual display.

8. A method for detecting a bone cavitation, comprising the steps of

locating an ultrasonic receiver array adjacent one side of a bone region to be examined;

locating an ultrasonic transmitter adjacent a side of the bone region opposite the receiver array;

sequentially selecting data from the receiver array;

generating and supplying a pulse signal to the transmitter so as to cause emission of an ultrasonic wave directed toward the bone region and toward the receiver array adjacent the opposite side of the bone region;

converting an attenuated sound wave received at sequentially selected portions of the receiver array into an input signal to form a succession of input signals; and

6

processing the input signals to obtain color-coded data representative of sound attenuation measuring the extent and location of the bone cavitation.

9. A method according to claim 8, wherein the bone region is a jawbone of a human being.

10. A method according to claim 8, wherein the generated pulse signal has a frequency is in the range of about 2.5 MHz.

11. A method according to claim 10, wherein the emitted sound wave from the transmitter has a frequency of about 2.5 MHz.

12. A method according to claim 8, wherein the processing step includes digitizing a common output signal based on the succession of input signals.

13. A method according to claim 12, wherein the digitizing step assigns a digital value to data from respective channels corresponding to the instantaneously selected portions of the receiver array.

14. A method according to claim 13, wherein the digital value is comprised of 8-bits representing 256 data levels.

15. A method according to claim 12, wherein the digitizing step is performed by a digitizer having an operating frequency of about 100 MHz.

16. A method according to claim 8, wherein the processing step includes the step of visually displaying the processed color-coded data.

17. A method according to claim 16, wherein the displayed color coded data is in the form of a 4x4 image.

18. A method according to claim 8, wherein the ultrasonic receiver array comprises multiple elements corresponding to the sequentially selected portions.

19. A method according to claim 8, wherein the ultrasonic transmitter is a single-element type.

* * * * *

(Per 21 CFR § 801.109)

510(k) SUMMARY

FEB 15 2002

I. General Information on Submitter:

Name: CAVITAT Medical Technologies, Inc.
10691 E. Bethany Drive, Suite 900
Aurora, Colorado 80014
Tele: (303) 755-2688
Fax: (303) 755-2699

II. General Information on Device

Name: CAVITAT™ (Ultrasonograph)

Classification Name: Extraoral Source X-Ray System

III. Predicate Devices:

Existing extraoral X-ray devices and
MYSONO 201 Diagnostic Ultrasound System and Transducers (K003121)

IV. Description of the Device:

The CAVITAT™ (Ultrasonograph) CAV 40000-1 or CAV 40000-3 with WIN/CAV Software (Release 1.05) consists of a transmitter, pulsing transducer, receiver, and a dedicated computer using an AMD processor. The computer system includes a LCD monitor, a color inkjet printer, and patented circuitry installed by CAVITAT Medical Technologies, the manufacturer of the device. The transmitter sends an electrical signal to the transducer which converts it into a pulsed ultrasonic sound wave that is transmitted at a rate of 27,000 sound pulses per microsecond, clocked at 2.5 megahertz at 1,042 feet per second at 98°F. These ultrasonic pulses travel through alveolar bone, and are received by the receiver membrane, which converts it into an electrical signal. It is this electrical signal that is displayed on the computer monitor. The display represents an average of two to eight pulses received when the button/foot pedal is pushed. The frequency used by the CAVITAT™ (Ultrasonograph) does not record the presence of soft tissue. It is the lack of interconnectivity and thickness of trabeculae that determines signal strength, and therefore the height of the three-dimensional column display. The colors of the columns displayed are non-linear.

V. Intended Use:

The CAVITAT™ (Ultrasonograph) CAV 40000-1 or CAV 40000-3 with WIN/CAV Software (Release 1.05) provides an ultrasound-based, three-dimensional image of the alveolar process of the maxilla and mandible as an adjunct to standard radiographic evaluation and clinical diagnostic procedures.

The clinical significance and correlation of the CAVITAT™ (Ultrasonograph) images, including column height and color grading, has not been established for specific osseous pathology, or normal bone. Positive images represent alveolar regions that attenuate ultrasound signals.

VI. Technological Characteristics of Device Compared to Predicate Devices:

The technological characteristics of the CAVITAT™ (Ultrasonograph) are identical to those of a diagnostic pulse-echo ultrasound device, with the exception of the fact that the CAVITAT™ (Ultrasonograph) measures the signal that passes through the bone rather than the return or echo signal. Positive images represent alveolar regions that attenuate ultrasound signals.

VII. Summary of Safety and Effectiveness Data

The CAVITAT™ (Ultrasonograph) was tested for acoustic output in accordance with FDA requirements for diagnostic ultrasound to confirm the safety of the device. The device met or exceeded all applicable FDA requirements. The effectiveness of the CAVITAT™ (Ultrasonograph) was demonstrated by a clinical study in which both radiographs and ultrasound-based, three-dimensional images of 92 alveolar sites were compared and confirmed with histopathologic examination. The results from the clinical study confirmed that the device is effective as an adjunct to standard radiographic evaluation and clinical diagnostic procedures.



DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service

FEB 15 2002

Food and Drug Administration
9200 Corporate Boulevard
Rockville MD 20850

Cavitat Medical Technologies, Inc.
C/O Mr. Larry R. Pilot
Mckenna and Cuneo, L.L.P.
1900 K Street, N. W.
Washington, D.C. 20006-1108

Re: K011147

Trade/Device Name: Cavitat™ (Ultrasonograph), CAV 40000-1 and CAV
40000-3 with WIN/CAV Software (Release 1.05)
Regulation Number: 872.1800
Regulation Name: Extraoral Source X-Ray System
Regulatory Class: II
Product Code: EHD
Dated: January 14, 2002
Received: January 14, 2002

Dear Mr. Pilot :

We have reviewed your Section 510(k) premarket notification of intent to market the device referenced above and have determined the device is substantially equivalent (for the indications for use stated in the enclosure) to legally marketed predicate devices marketed in interstate commerce prior to May 28, 1976, the enactment date of the Medical Device Amendments, or to devices that have been reclassified in accordance with the provisions of the Federal Food, Drug, and Cosmetic Act (Act) that do not require approval of a premarket approval application (PMA). You may, therefore, market the device, subject to the general controls provisions of the Act. The general controls provisions of the Act include requirements for annual registration, listing of devices, good manufacturing practice, labeling, and prohibitions against misbranding and adulteration.

If your device is classified (see above) into either class II (Special Controls) or class III (PMA), it may be subject to such additional controls. Existing major regulations affecting your device can be found in the Code of Federal Regulations, Title 21, Parts 800 to 898. In addition, FDA may publish further announcements concerning your device in the Federal Register.

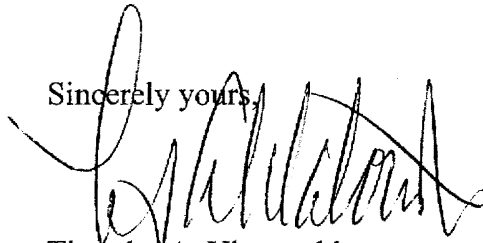
Please be advised that FDA's issuance of a substantial equivalence determination does not mean that FDA has made a determination that your device complies with other requirements

of the Act or any Federal statutes and regulations administered by other Federal agencies. You must comply with all the Act's requirements, including, but not limited to: registration and listing (21 CFR Part 807); labeling (21 CFR Part 801); good manufacturing practice requirements as set forth in the quality systems (QS) regulation (21 CFR Part 820); and if applicable, the electronic product radiation control provisions (Sections 531-542 of the Act); 21 CFR 1000-1050.

This letter will allow you to begin marketing your device as described in your Section 510(k) premarket notification. The FDA finding of substantial equivalence of your device to a legally marketed predicate device results in a classification for your device and thus, permits your device to proceed to the market.

If you desire specific advice for your device on our labeling regulation (21 CFR Part 801 and additionally 21 CFR Part 809.10 for in vitro diagnostic devices), please contact the Office of Compliance at (301) 594-4613. Additionally, for questions on the promotion and advertising of your device, please contact the Office of Compliance at (301) 594-4639. Also, please note the regulation entitled, "Misbranding by reference to premarket notification" (21CFR Part 807.97). Other general information on your responsibilities under the Act may be obtained from the Division of Small Manufacturers, International and Consumer Assistance at its toll-free number (800) 638-2041 or (301) 443-6597 or at its Internet address <http://www.fda.gov/cdrh/dsma/dsmamain.html>

Sincerely yours,

A handwritten signature in black ink, appearing to read 'Timothy A. Ulatowski', written over a horizontal line.

Timothy A. Ulatowski
Director

Division of Dental, Infection Control
and General Hospital Devices
Office of Device Evaluation
Center for Devices and
Radiological Health

Enclosure

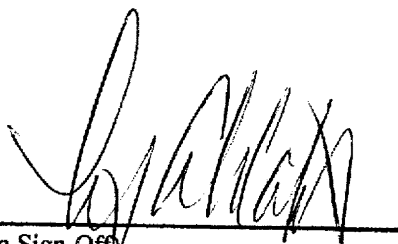
510(k) Number: K011147

Device Name: CAVITAT™ (Ultrasonograph)

Indications for Use:

The CAVITAT™ Ultrasonograph CAV 40000-1 or CAV 40000-3 with WIN/CAV Software (Release 1.05) provides an ultrasound-based, three-dimensional image of the alveolar process of the maxilla and mandible as an adjunct to standard radiographic evaluation and clinical diagnostic procedures.

The clinical significance and correlation of the CAVITAT™ (Ultrasonograph) images, including column height and color grading, has not been established for specific osseous pathology, or normal bone. Positive images represent alveolar regions that attenuate ultrasound signals.



(Division Sign-Off)
Division of Dental, Infection Control,
and General Hospital Devices

510(k) Number K011147

PLEASE DO NOT WRITE BELOW THIS LINE. CONTINUE ON ANOTHER PAGE IF NEEDED.

Concurrence of CDRH, Office of Device Evaluation (ODE)

Prescription Use ✓

OR

Over-The-Counter Use _____

CAVITAT™ Ultrasonograph

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|--|--|--|
| Received.....1/30/04 Scientific Review.....2/8/04 IAOMT Board Review.....3/25/04 Reevaluation..... 7/24/05 | Scientific Review Root Canals & Cavitations | Approval.....3/25/04 Provisional Approval No Opinion No Approval |
| <p>Explanation of IAOMT position: In the next paragraph the applicant request that the Cavitat Ultrasonograph be also approved as an IAOMT Standard of Care. The IAOMT fully appreciates the true value of the Cavitat Ultrasonograph. However IAOMT Approval as a Standard of Care in addition to the Scientific Review could place the membership in legal jeopardy if the Cavitat Ultrasonograph is not used below by a member for those purposes. With this in mind, the IAOMT fully supports the use of this technology for the reasons described below. The IAOMT encourages anyone doing these procedures to seriously consider using the Cavitat Ultrasonograph but at this time the IAOMT must be cautious in defining this as a Standard of Care.</p> <p>Applicant's Request: It is recommended that the CAVITAT™ Ultrasonograph be an approved Scientific Review and Standard of Care of the IAOMT for the following reasons:</p> <ol style="list-style-type: none"> 1. The device is used to determine the general health of the jawbone before any dental procedure is done. 2. The incidence of necrotic bone lesions is estimated in the general population to be from 40% to 80%. Utilization of this technology would reduce the failure rate of dental procedures performed on ischemic bone, which is currently estimated to be 40%. Necrotic bone tissue is reported to give off toxins that can affect the general health as well as the integrity of dental health. When used in conjunction with a panograph, detection and diagnosis of jawbone lesions approaches 98 to 100% in accuracy. X-rays alone show 27% at best and only when 60% or more of the cortical bone is destroyed. 3. The results of Cavitat™ testing should be evaluated with the panograph as this provides a higher percentage of accurate diagnosis than either procedure alone. The dental professional can now accurately select or rule out appropriateness of certain dental procedures for patients seeking restorative or reconstructive dental procedures. 4. Diagnosis of necrotic jawbone can also benefit the medical professional as this diagnosis provides the potential cause of chronic illness whose etiology is unknown and the illness cannot otherwise be explained. It opens a new avenue of investigation for the physician and the opportunity to work with the dental professional to assist the patient in recovery of health. <ol style="list-style-type: none"> 1. The CAVITAT™ Ultrasonograph provides: <ol style="list-style-type: none"> A) An ultrasound-based 3-D color image of the alveolar process of the maxilla and mandible. B) An extremely accurate quantitative analysis of the alveolar process. C) Real-time and 3-D image of each tooth site that can be moved vertically or mesial/distal to scan a larger area while in use, and the image can also be rotated, zoomed in or zoomed out to better evaluate the total condition of the bone. D) Detection and precise measuring of low bone density to aid medical professionals in diagnosing areas of possible concern before a dental procedure such as an implant or root canal. E) Color coded images that distinguish the intensity of destruction: <ol style="list-style-type: none"> 1. Red = significant bone loss and density 2. Yellow = moderate bone loss and density 3. Green = no bone loss. F) Capable of detecting jawbone defects from 1 millimeter and up in size 2. Benefits: <ol style="list-style-type: none"> A) Information can be read and interpreted immediately B) Quick and thorough scanning of the entire jawbone C) Real time digital scanning D) No radiation E) Very user-friendly F) Low cost operation, uses no chemicals or film G) Mobile <p>No other imaging device has been able to match the accuracy of Cavitat™. X-rays show 27% at best and only when 60% or more of the cortical bone is destroyed. The CAVITAT™ Ultrasonograph when used in conjunction with a panograph will provide 98 to 100% of the information needed for an accurate clinical diagnosis allowing the practitioner to confirm or rule out suspect areas that cannot be clearly imaged by x-ray alone.</p> <p>It is the only instrument designed exclusively for quantitative bone analysis.</p> | | |

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| Name of Scientific Review: CAVITAT™ Ultrasonograph |
| Alternative name(s) of Scientific Review: Ultrasonographic imaging of jawbone to detect low jawbone bone density |
| This Scientific Review is related to Medicine & Dentistry |
| This Scientific Review is Equipment |
| <p>Purpose of the Scientific Review: This equipment is used for:</p> <ol style="list-style-type: none"> 1. Detection of low bone density in the jawbone. It provides an ultrasound-based 3-D image of the alveolar process of the maxilla and mandible. It performs a quantitative analysis of the alveolar process. 2. To better evaluate the total condition of the jawbone by precisely measuring necrosis of the jawbone to aid medical professionals in diagnosing Bone Marrow Edema Syndrome, Neuralgia Inducing Cavitational Osteo-necrosis, Osteomyelitis and Periodontal Pockets of the Buccal Bone. <p>The latest scientific studies have proven these lesions to be highly neurotoxic (reference website: www.testfoundation.org). These toxins inhibit protein and enzyme absorption essential for all cellular functions contributing to or inducing systemic cellular disease.</p> |
| <p>Scientific Review History: The CAVITAT™ was developed by Bob Jones, a former pilot, who suffered from an extremely severe, debilitating osteonecrosis of multiples sites in his jawbone.</p> <p>Initially, the inventor developed a transducer, a device that sends sound waves through the jawbone at an ultrasound wave strength determined to be effective and then a digitized array was developed that is capable of interpreting the strength of the signal after it has passed through the bone.</p> <p>Once the signal data is acquired, it is sent to a computer that converts the signal to a digital perspective 3-D color image. User-friendly software was developed to operate and convert the signals from analog to digital (WIN/CAV) in order to project the color image to the computer screen. All components of the Cavitat™ are patented by the inventor</p> |
| <p>A brief description of the Scientific Review Initial research was done at New Mexico Institute of Mining & Technology, Socorro, New Mexico, on pigs' jaws and confirmed that the ultrasound wave strength frequency was the proper setting to image accurately and safely. Following this research, CAVITAT™ Medical Technologies, Inc. was established to take this revolutionary imaging system through testing and into production. The Generation 3 field model of the CAVITAT™ Ultrasonograph was built and used for several years by doctors who participated in independent clinical trials under the administration of Dr. Jerry Bouquot of The Maxillofacial Center for Diagnostics & Research, Morgantown, West Virginia. Using the data obtained from clinical trials, a Generation 4 production model was built and sold to doctors as a custom built system.</p> <p>Data from clinical trials and Dr. Bouquot's study was presented to the FDA for full review with the 510(k) application. Although not normally required in the FDA 510(k) process, CAVITAT™ Medical Technologies, Inc., prepared and submitted in excess of 12,000 scans, biopsies and panos to the FDA as supporting evidence. Dr. Bouquot personally took in excess of 900 scans and supporting data to the FDA where 392 were randomly selected and reviewed by the FDA committee. The CAVITAT™ legal team commented that this was the largest amount of research data ever presented to the FDA, to their knowledge, for a dental device for a 510(k) clearance.</p> <p>The FDA requires the scan be a prescription item. The FDA designated the instrument as an Ultrasonograph and listed it as a Class II device with no restrictions on marketing.</p> |
| A specific description of this Scientific Review: Contact the manufacturer for specific directions of use which would be part of training with the equipment purchase |
| Manufacturer(s) & Distributer(s): CAVITAT™ Medical Technologies, Inc. is the manufacturer/distributor, 10730 E. Bethany Drive, Ste. 112, Aurora, Colorado 80014, (303) 755-2688, FAX (303) 755-2699 Website: www.cavitat.com , |

E-mail: cavitat@cavitat.com

Scientific Literature: *Medullary and Odontogenic Disease in the Painful Jaw: Clinicopathologic Review of 500 Consecutive Lesions*, The Journal of Craniomandibular Practice, October 2002, volume 20, Number 4.

Published abstracts relative to Through-transmission Alveolar Ultrasonography (TAU) as of September 19, 2002.
More information at www.maxillofacialcenter.com, (click osteonecrosis), as follows:

1. Bouquot, W, Martin, G, Wroblewski. Computer-based thru-transmission sonography (CTS) imaging of ischemic osteonecrosis of the jaws – a preliminary investigation of 6 cadaver jaws and 15 pain patients. Oral Surg Oral Med Oral Pathol Oral Radiol Endo 2001; 92:550. Presented orally to the American Academy of Oral & Maxillofacial Pathology, Chicago, Illinois, April, 2001.
2. Bouquot J, Margolis M, Shankland W, Imbeau J. Through-transmission Alveolar Ultrasonography (TAU) – A new technology for evaluation of medullary diseases. Correlation with histopathology of 285 scanned jaw sites. Oral Surg, Oral Med Oral Pathol, Oral Radiol Endod 2002; 94:210. Presented orally to the American Academy of Oral & Maxillofacial Pathology, New Orleans, April, 2002.
3. Bouquot JE, Shankland WE II, Margolis M. Through-transmission alveolar ultrasonography (TAU) – new technology for evaluation of bone density and desiccation. Comparison with radiology of 170 biopsied alveolar sites of osteoporotic and ischemic disease. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002; 93:214-215. Presented orally to the American Academy of Oral Medicine, Ft. Lauderdale, April, 2002.
4. Bouquot JE, Shankland WE II, Margolis M, Glaros W. Through-transmission Alveolar Ultrasonography (TAU) – new technology for detection of low bone density of the jaws. Comparison with radiology for 92 osteoporotic alveolar sites with histopathologic confirmation. J Oral Pathol Med 2002; 31:289-290. Presented orally to the 11th Biennial Congress of the International Association of Oral Pathologists, Singapore, August, 2002.
5. Bouquot, JE, Through-transmission alveolar sonography (TAU) – new technology for the evaluation of low bone density and ischemic disease. Correlation with histopathology of 339 scanned alveolar sites. This paper was presented to the American Academy of Oral and Maxillofacial Pathology, New Orleans, Louisiana, April, 2002.

Jawbone Cavitations: Infarction, Infection & Systemic Disease, Susan Stockton, MA; Townsend Letter for DOCTORS & PATIENTS – April 2002.

Margolis, Michael D. DDS, Candidate, Doctor of Integrative Medicine, Daniel F. Farrier, MD, CCN, Doctor of Integrative Medicine (CUIM), David S. Margolis, B.S., - Doctoral Thesis: *Accurate Detection of Jawbone Lesions and Osteolytic Abnormalities Using the CAVITAT™ Ultrasonogram Imaging System: A clinical Study Utilizing Photoaffinity Labeling and Histological Assessments*

Legal Aspects of this Scientific Review: The CAVITAT™ Ultrasonograph has the following clearances and certifications:

Food and Drug Administration: FDA Clearance #K011147 was granted February 15, 2002 with the following indication for use:

“Indications for Use: the CAVITAT™ Ultrasonograph CAV 40000-1 or CAV 40000-3 with WIN/CAV software (Release 1.05) provides an ultrasound-based, three dimensional image of the alveolar process of the maxilla and mandible as an adjunct to standard radiographic evaluation and clinical diagnostic procedures.”

Prior to review of data by the FDA, a third party designee of the FDA in California cleared the CAVITAT™ Ultrasonograph for its complete safety and efficacy – February 16, 2001.

Health Canada: CSA #37968; approved 5/17/02

Federal Communications Commission: FCC – 3/27/02

Commission of Europe: CE – 3/27/02

Commission of Medical Europe: CME –3/27/02

Applicant Name: CAVITAT™ Medical Technologies, Inc.

Office Phone: