

## ***Routine Dental Extractions Routinely Produce Cavitations***

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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).

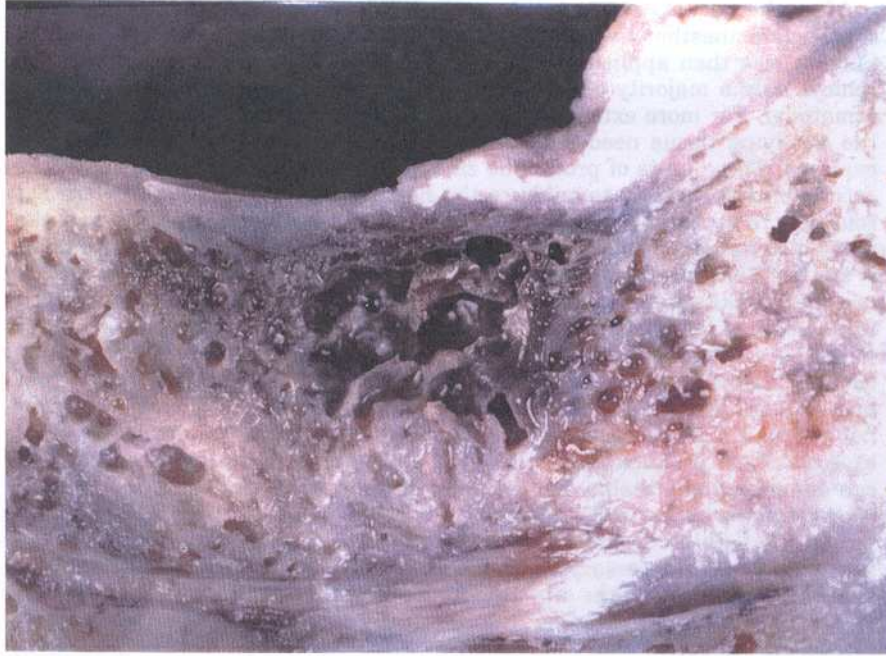
Black's findings were either ignored, forgotten, or not given proper credence. Certainly, they were never assimilated into dentistry and dental teaching. In the 1970s, however, CVs were "re-discovered" and correlations with previously termed idiopathic pain syndromes were suggested. Patients with atypical 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 cavitation 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. Porphyrins, disorders in which





FIGURE 2A  
Mandible Enhanced

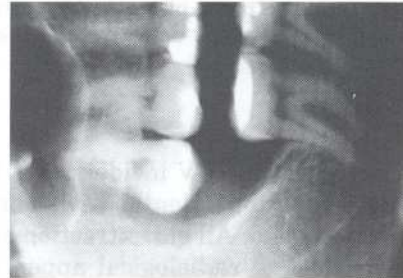


FIGURE 2B  
Mandible Unenhanced



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

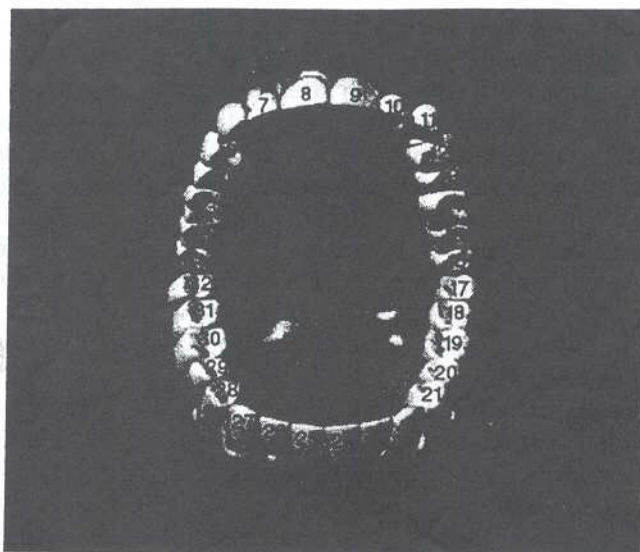
## Results

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 under-represents 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-

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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 serous 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.

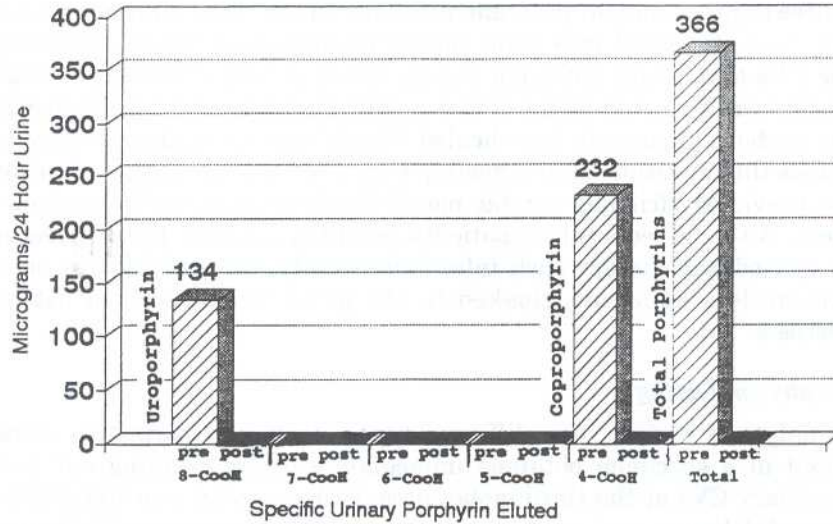


FIGURE 4

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 realignment of the teeth. When the first bicuspid 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 periodental 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

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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

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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-

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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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