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Dr. Ronald S. Carlson
4211 Waianae Avenue
Suite 400
Honolulu, Hawaii 96816
808-735-0282

Endodontic-Endotoxemia: Our Current Dilemma

OUR CURRENT DILEMMA IN DENTAL-MEDICINE

For more than 135 years there have been attempts to retain teeth whose nutrient canals have been destroyed by placing gutta-percha or other sealants within the canals.[1] Numerous innovative mechanical techniques have been developed over the years to render the canals sterile and uninhabitable for microorganisms. Virtually all advertisements within the industry trade journals emphasize "mechanical obturation" and not the bio-chemical aspects of sterility of the canal system, or if the canals can be sterilized at all in the short or long term. As reported recently, overemphasis on this aspect of simply filling the empty spaces within the root canal system and its importance has misled the field of endodontology.[2] What is most critical, is that root canal teeth become nesting sites for microbes who, due to the loss of outward hydraulic pressure from within the body of the dental organ since the pulp no longer is living, migrate though the dentin into the peri-radicular tissues, thence into the circulatory system and other tissues. In Dental Clinics of North America, Vol. 18, No. 2, April 1974, Kaare Langeland, D.D.S., Ph.D. reviewed "Root Canal Sealants and Pastes" in which he states: "*The fact that sealers and pastes will contact vital or disintegrating tissue connected with the general circulation allows for generalized distribution by blood and lymph…The resorbability of sealers and pastes has been abundantly demonstrated by gross radiographic evidence. There is, therefore, no doubt that material is transported away from the site where it was originally applied*."

What has been shown today as of equal or more significant importance is the relative sterility of the intra-radicular-canal system.[3,4,5] Current research shows that the significant presence of bacteria in the canal systems lead to the pathogenesis of "apical periodontitis" (AP). This condition (AP) is a chronic/acute inflammation of the tissue about the roots of root filled teeth also designated as "apical granuloma."[21]

Equally significant in consideration for successful endodontic procedures is the presence of bacteria within the microtubules of the dentin itself.[5] These concerns were reported in lectures in Chicago and other major cities in the 1930s and 1940s. Infected dentine was demonstrated to be of great influence on the lack of success of root filled teeth by Weston Price, D.D.S.[6] Recent studies show that dentine can be infected and that controversy exists regarding how to disinfect it clinically.[7] Rather than sterility of the root canal system as an objective of modern endodontics, one can at best employ the term "relative sterility" or disinfecting.

Systemic implications are obvious from more recent work showing that apical periodontitis, an inflammatory process around the apex of a tooth root, is primarily a sequel to microbial infection of the pulp space of teeth and is a remarkably widespread problem.[8]

It is calculated that apical priodontitis is so prevalent that by the age of 50, one in two (50%) experience the disease.[9] By age 62 the statistic rises to 62%. In the U.S. it is estimated that 420 million teeth have received root filings, or in the common vernacular "root canalled."[10]On this basis the average number of root fillings is 2.2 per adult.[11] Readily it can be understood that the magnitude of the problem is not fully comprehended nor appreciated as to its impact upon the general health of the individuals with this condition.[12]

Inflammation has been described as a common reaction of the body to damage of its cells and vascularized tissues. The discovered detailed process of inflammation has revealed its relationship to immune response as being one in the same fundamentally. The five basic symptoms of inflammation, 1) redness (rubor), 2) swelling (tumour), 3) heat (calor), 4) pain (dolor), and 5) loss of function (functio laesa) have been know since ancient times.

In a recent work, a close correlation to periodontal pathology and rheumatoid arthritis was found.[13] In 62.5% of a study population of 1,412 people with rheumatoid arthritis had advanced periodontal disease. The authors went as far as stating there are certain pathogenic similarities between the two clinical entities.

Further, in an article found in the International Endodontic Journal, Vol. 33 Issue 1 Page 1, year 2000, titled "*Root canal treatment and general health: a review of the literature*" the authors state: "There has been an increase in the number of case reports published in the medical literature citing dental infection as an associated factor in several systemic illnesses including; ***uveitis*** (Sela & Sharav 1979), ***intracranial abscesses*** (Holin et al. 1967, Henig et al. 1978, Ingham et al. 1978, Churton & Green 1980, Aldous et al. 1987, Marks et al. 1988, Saal et al. 1988), ***childhood hemiplegia*** (Hamlyn 1978), ***cerebral infarction*** (Syrjanen et al. 1989), ***bacteriospermia and subfertility*** (Bieniek & Riedel 1993), ***necrotizing fasciitis*** (Gallia & Johnson 1981, Steel 1987, Stoykewych et al. 1978), ***mediastinitis*** (Hendler & Quinn 1978, Zachariades et al. 1988, Musgrove & Malden 1989), ***fatal endocarditis*** (Kralovic et al. 1995), ***toxic shock syndrome*** (Egbert et al. 1987, Navazesh et al. 1994), and ***septicemia*** (Lee 1984)."

Infections in the mouth can develop into chronic localized infections or systemic infectious disabilities—conditions we refer to as disease or illness. In the pathogenesis and evolution of glomerular nephropathies, these localized infections play an important role.[14] Renal patients appear to be subject to a variety of dental problems such as periodontal disease, narrowing of the pulp chamber, enamel abnormalities, premature tooth loss and xerostomia. Renal difficulties also often arise after a specific stomatologic (oral) treatment of dental foci. The dental infection can localize in the pulp of the dental organ (tooth), developing acute gangrenous pulpitis. On the other hand, a chronic evolution from gangrenous pulpitis may lead to the formation of dental granulomas, cysts, osteomyelitis, other bone conditions and abscesses in the bone surrounding the root of the tooth. In the evolution of these infections, acute phases may occur, despite the fact that the human host tries to isolate these foci by forming an external layer of fibrin and connective tissue about these lesions, much like the skin protects the hands by forming blisters and then subsequent callous' after working vigorously in the backyard vegetable garden. In these phases, germs or parts of them—toxins—could get into the circulation. They could produce various lesions in other tissues—this process being known as "*effective localization*"—either directly or indirectly by forming immune complexes. These are deposited in different tissues where they can activate different factors like compliments—c reactive proteins for example in the liver. The kidney is often the target organ, since it is the master filter for the vascular system.

Scientific America, May 2001, presented an article titled *Taken to Heart.* Reporting that in a recent examination of 50 plaques scraped out of

human arteries, 72 % contained periodontal pathogens. Two other pathogens that are hot candidates for atherosclerosis were also present: *cytomegalovirus,* which infected 38% of plaques, and *Chlamydia pneumoniae,* which appeared in 18%. Studies have also found antibodies against oral bacteria in the blood, and animal tests have shown that mouth microbes injected into the blood lead to atherosclerosis. Links between periodontal infection (*apical periodontitis included)* and other conditions such as diabetes, chronic respiratory infection, stroke and low birth weight have also emerged. They support the theory that many chronic ill conditions stem from systemic low-grade infections.

A marker known as C-reactive protein or CRP can now identify the systemic low-grade infections causing systemic inflammation. It is most always elevated in patients with periodontitis. Chronic and/or acute inflammation of the peri-radicular ligament may lead to heart disease because it might cause this low-level systemic inflammation since the chemical by-products of the degradation itself along with the autoimmune response may spill over into the blood stream and lymphatic system triggering the liver to make other proteins that inflame the arterial walls and clot blood. Atherosclerosis and, ultimately, heart attack may result.[15]

The American Heart Association's (AHA) web site states: "A growing number of studies have examined whether hs-CRP can predict recurrent cardiovascular disease and stroke and death in different settings. High levels of hs-CRP consistently predict new coronary events in patients with unstable angina and acute myocardial infarction (heart attack). Higher hs-CRP levels also are associated with lower survival rate of these people. Many studies suggest that after adjusting for other prognostic factors, hs-CRP was still useful as a predictor." However, the AHA does not proffer a possible cause to cause of the low-grade systemic infections. This, I believe, will be left to those studying the subtle Oral-Sytemic Linkages as outlined in recent editorials of the Triple O Journal.[2, 16]

CLINICAL EVIDENCE OF ORAL-SYSTEMIC DENTAL INFECTION

With these facts in mind, let us examine the background information derived from a study reported in ODONTOLOGISK REVY, VOLUME 18, SUPPLEMENT 11, 1967 demonstrating apical pathology at the apices of most all teeth examined. The objective of the research was to see if dental x-rays of the root-tips of "root filled" teeth could be used for the purposes of predicting diseased teeth. The summary of this research is as follows:

"The investigation is concerned with a comparison of histological and roentgenological appearance of periapical changes in an attempt to find out whether and to what extent histological changes are reflected in conventional roentenograms. The investigation was carried out on autopsy material and the final analyst was based on 292 cases.

"Roentgenograms were taken of the upper incisors, from which the apical part of the root together with the periapical tissue was afterwards removed with a trepan. Histological sections in the same plane as the roentgenograms were prepared by conventional methods and analyzed in detail with special reference to the root canal, the foramen, the apical and lateral periodontal soft tissue, and the apical hard tissue, including the marrow.

"With the aid of the histologic details, amount and type of inflammatory cells, type of marrow in adjacent marrow spaces, and shape and width of the apical soft tissue, the material fell into three groups…"

And then the researchers go on into details not necessary for our discussion here, for they are lengthy. The findings were simply that, although their primary work in this complex project was regarding the accuracy of dental x-rays distinguishing pathology, what they reported was, yes, x-rays have good accuracy if taken correctly and interpreted properly; and, additionally importantly is that, "…*complete healing after root-filling occurred in only 7% of the cases*…" Reciprocally, what this reports stated was that *93% were not healed*; that pathology, diseased tissue, was present in the majority of teeth having been treated with deep root-fillings at the level of the bone.

Continuing in this line of thought regarding background information, in the journal ENDODONTICS & DENTAL TRAUMATOLOGY, Volume 16, Number 2, April 2000, it was reported that:

"A total of 30 patients were referred for surgical endodontic treatment of teeth with apical periodontitis. The participants were asymptomatic and participation was limited to patients who did not demonstrate the presence of sinus tracts or lesions that required endodontic or periodontal treatment. In Group 1…73% (11 of 15 patients) demonstrated bacterial growth within the periapical lesion…in Group 2…10 samples (67%) of the periapical lesions evaluated demonstrated bacterial contamination."

We must bear in mind that in the BACKGROUND AND PURPOSE of this report it was noted:

"The presence of microbial activity within periapical tissue affected by apical periodontitis has been debated. Micro-biological sampling has been routinely performed to determine if microorganisms can be recovered from the periapical lesion following ineffective endodontic treatment of apical periodontitis. Although several studies have suggested that bacteria maintains a presence in asymptomatic periapical lesions, this theory has not been universally accepted…"

For the past 28 years the author has accumulated data on teeth, which for one reason or another had to be removed. Most of these dental organs were dead, having had "root canals." However, the sample presented here is for only the past several years including 139 teeth (111 people) with root

canal fillings. In every case there was an inflammatory condition that was identified by a board certified pathologist from Queens Hospital Pathology Department.

The summation of these findings is sorted out in the following table:

HISTO-PATHOLICAL STUDIES OF SOFT TISSUE, BONE, ABOUT THE ROOTS OF RC- TEETH DONE AT QUEENS HOSPITAL, HONOLULU, HAWAII 2001-2008

Gender	Age	Endo Tooth #	Pathology	Bacteria
Female 10/6/08	34	3	Periapical Granuloma/Cyst Chronic Inflammation	(+) [Actinomyces=(+)]
Female 10/1/08	51	30	Periapical Granuloma/Scar Reactive Bone	(-)
Female 9/13/08	60	29/30	Thickened partially devitalized bone/granulation tissue	(-)
Male 8/11/08	29	28	Chronic inflammation/ Periapical Granuloma	(+)
Male 8/11/08	29	8,9	Chronic inflammation/ Periapical Granuloma/Scar	(-)
Female 7/14/08	56	3	Acute and Chronic inflammation with Fibrosis and Granulation Tissue	(-)
Female 6/21/08	66	9	Fibrosis, Periapical Scar	(+)
Female 6/18/08	49	7/9	Periapical Granuloma/Scar Chronic inflammation	(+)
Female 6/2/08	55	4	Periapical Granuloma	(-)
Male 4/22/08	58	9	Apical Fibrosis / Scar	(-)
Female 3/19/08	63	8, 6	Periapical granuloma/cyst marked chronic inflammation	(+)
Female 1/22/08	63	14	Periapical Scar (Fibrosis) with focal bacteria	(+)
Female 1/9/08	50	19	Periapicl Granuloma/Cyst, acute/chronic inflammation	(-)
Male 12/5/07	45	5	Granulation tissue/Cyst, chronic inflammation	(-)
Female 12/1/07	34	8/9	Granualtion tissue, marked inflammation, fibrosis	(-)
Female 12/1/07	56	12/13	Granulation tissue, partially devitalized bone, chronic inflammation	(+)
Female 11/9/07	68	13	Periapical Scar/Granuloma with chronic inflammation	(+)
Female 11/1/07	70	28/29	Fragments of reactive focally necrotic bone, fibrosis, chronic inflammation	(-)
Male 12/5/07	53	31	Periapical Granuloma/Cyst marked acute/chronic inflammation	(-)
Male 9/27/07	64	14	Radicular Cyst (Periapical) marked acute / chronic inflammation	(+)
Male 9/18/07	64	19	Periapical Abscess/Granuloma	(+)]
Male 8/24/07	42	2	Periapical Granuloma/ Cyst chronic inflammation and reactive bone changes	(-)
Male 8/13/07	74	19	Periapical Granuloma	(+)

			marked acute chronic inflammation	
Male 8/13/07	74	2	Periapical Granuloma marked acute chronic inflammation	(-)
Male 8/13/07	74	30	Periapical Granuloma marked acute chronic inflammation	(-)
Female 5/23/07	51	4	Periapical Granuloma/Cyst marked acute/chronic inflammation	(+)
Male 5/12/07	62	18	Periapical Granuloma marked acute/chronic inflammation	(--)
Female 4/10/07	32	14	Periapical Granuloma/Scar chronic inflammation	(+)
Female 3/31/07	59	2	Periapicl Granuloma, marked acute/chronic inflammation	(+)
Female 2/1/07	49	29	Periapical Granuloma, marked acute/chronic inflammaton	(+)
Female 5/19/06	62	4	Periapical Granuloma/Chronic infalm	(+)
Female 5/15/06	30	9&10	Periapical Abscess/Granuloma	(+)
Female 4/11/06	52	24	Apical Periodontitis/ Granulation Tissue with Chronic Inflammation.	(--)
Male 4/1/06	57	31	Periapical Granuloma/Scar—Reactive Bone Changes, Chronic Inflammation.	(-)
Female 2/28/06	42	31, 32	Periapical Granuloma & Cyst—Reactive Bone Changes—Marked Acute/Chronic Inflamm.	(-)
Female 2/23/06	42	18, 20	Periapical Granuloma/Scar Marked Chronic/Acute Inflamm—Reactive BC.	(-)
Female 2/15/06	42	15	Periapical Granuloma/ Scar—Reactive Bone Changes	(-)
Female 1/26/06	42	2	Periapical Granuloma with Chronic Inflamm. And Reactive Bone Changes.	(-)
Male 1/25/06	60	19	Periapical Abscess/ Granuloma & Reactive Bone Changes	(+)Actinomyces
Female 12/19/05	88	24,25	Granulation Tissue/ Fibrosis with acute/chronic inflamm.	(+)Actinomyces
Female 12/15/05	45	12	Radicular Cyst (Periapical)	(-)
Male 11/8/05	83	19	Periapical Scar	(+)Actinomyces
Female 10/11/05	57	5	Periapical Granuloma/Cyst	(-)
Female 10/10/05	62	9,10	Periapical Granuloma/Cyst	(-)
Female 7/31/05	54	15	Periapical Granuloma Reactive Bone changes	(-)
Female 7/26/05	64	10	Periapical Granuloma/Cyst	(-)
Female 7/24/05	62	19	Periapical Granuloma/Cyst	(-)
Male 7/16/05	81	5	Granuloma/Scar	(+)Actinomyces
Female 7/06/05	52	9,10	Periapical Granuloma/Cyst	(-)
Female 6/16/05	57	4, 5, 3	Fibrosis & Chronic Inflammtion	(-)
Male 5/18/05	67	4	Periapical Granuloma/Cyst	(-)
Male 5/10/05	73	19	Fibro-Granuloma	(+)Actinomyces
Male 3/16/05	56	4	Periapical Granuloma	(-)
Female 2/15/05	41	3	Radicular Cyst	(+)Actinomyces

Female 2/12/05	61	8,10	Periapical Granulomas	(+)Actinomyces
Male 2/07/05	56	6	Periapical Granuloma	(-)
Female 1/26/05	69	4	Periapical Granuloma	(+)Actinomyces
Female 1/24/05	42	14	Periapical Granuloma	(+)Actinomyces
Female 1/20/05	56	18	Radicular Cyst	(+)Actinomyces
Male 11/27/04	54	9	Chronic Inflammation, Fibrosis, Granulation	(-)
Male 11/20/04	56	5	Apical Periodontitis	(-)
Female 8/31/04	51	9	Periapical Granuloma	(+)Actinomyces
Female 8/16/04	54	3	Periapical Granuloma	(-)
Female 8/16/04	54	30	Periapical Granuloma	(-)
Female 8/04/04	48	19	Periapicl Abscess	(+)Actinomyces
Male 7/22/04	45	3	Radicular Cyst	(+)Actinomyces
Female 7/17/04	19	19	Periapical Granuloma	(-)
Female 7/12/04	55	30	Periapical Granuloma	(-)
Male 5/03/04	50	10	Periapical Scar/Fibroma	(-)
Female 4/12/04	65	7	Racicular Cyst	(+)Actinomyces
Female 1/02/04	61	30	Apical Periodontitis	(+)Actinomyces
Male 12/23/03	55	2	Periapical Granuloma	(-)
Female 10/31/03	32	12	Periapical Granuloma/Cyst	(-)
Female 10/18/03	53	20	Periapical Granuloma/Scar	(-) Biocalex Filled Endo
Female 9/30/03	62	13	Periapical Granuloma/Cyst	(-)
Female 9/19/03	39	14	Apical Granuloma Periapical periodontitis	(-)
Female 8/18/03	69	9	Periapical Granuloma	(-)
Male 7/30/03	44	9	Periapical Granuloma	(-)
Female 7/11/03	42	7, 8, 9, 10	Periapical Granulomas	(+)Actinomyces
Male 6/06/03	38	25	Periapical Granuloma (Chron. Api. Periodon.)	(-)
Female 6/06/03	66	18	Radicular(Periapical) Cyst	(+)Actinomyces
Male 6/04/03	75	7	Devitalized Bone and Fibrosis	(-)
Female 4/01/03	61	20	Apical Fibrosis	(+)Actiinomyces
Female 3/18/03	37	20	Periapical Granuloma/Cyst	(+)Actinomyces
Male 3/17/03	66	9	Periapical Granuloma scar	(+)Actinomyces
Female 2/18/03	61	4	Periapical Granuloma	(+)Actinomyces
Female 2/04/03	80	9	Radicular (Periapical) Cyst	(+)Actinomyces
Male 9/11/02	50	18	Radicular (Periapical) Cyst	(-)
Female 8/08/02	73	19	Periapical Abscess/Cyst	(-)
Female 6/22/02	69	28, 29, 30	Periapical Granulomas	(-)
Female 6/17/02	53	28	Periapical Granuloma with Foreign body mat.	(-)
Male 6/15/02	75	13, 15	Radicular (Periapical) Cyst	(+)Actinomyces
Female 6/04/02	73	11	Periapical Granuloma/Cyst	(-)
Female 6/04/02	75	19	Periapical Granuloma/Scar	(-)
Female 5/22/02	65	20	Periapical Granuloma/Cyst (RBC)	(-)
Female 5/07/02	54	15	Radicular (Periapical) Cyst	(+)Actinomyces
Male 5/06/02	43	14, 30	Periapical Granulomas (Devitalized Bone)	(+)Actinomyces
Female 2/02/02	55	18	Periapical Granuloma	(-)
Male 12/03/01	61	2	Fibrosis, Granulation and Chronic Inflamm.	(-)
Male 10/30/01	43	2	Radicular Cyst and Necrotic Bone	(-)
Male 9/04/01	62	18	Periapical Granuloma (RBChanges)	(+)Actinomyces
Female 8/31/01	54	14	Periapical Granuloma & Dead Bone	(-)
Male 8/27/01	51	3	Periapical Granuloma & RBChanges	(+)Actinomyces
Female 8/01/01	31	10	Periapical Granuloma/Cyst	(+)Actinomyces
Male 7/30/01	58	18	Periapical Abscess/Granuloma (RBChanges)	(-)
Female 6/27/01	58	7, 9	Periapical Granuloma/Cyst	(+)Actinomyces

			with Necrotic Bone	
Female 5/16/01	46	9	Periapical Granuloma and Devitalized Bone	(+)Actinomyces
Male 4/30/01	51	4	Periapical Granuloma/Cyst	(+)Actinomyces
Female 4/19/01	31	14	Periapical Granuloma with RB changes.	(-)
Female 3/20/01	47	5	Periapical Granuloma	(-)
Female 3/14/01	54	19	Periapical Granuloma/Scar	(-)
Female 3/13/01	50	14	Radicular (Periapical) Cyst	(+)Actinomyces
Female 3/12/01	53	13	Periapical Granuloma/Cyst	(+)Actinomyces
Female 2/26/01	53	4	Periapical Abscess/Granuloma	(+)Actinomyces
Female 2/26/01	50	29	Necrotic Bone Inflam. Granulation tissue/fibrosis	(-)
Female 2/17/01	50	3,4	Periapical Granuloma/Cyst	(+)Actinomyces

HEALTHY POSSIBILITIES

Suffice it to say, this clinical research study demonstrates that all teeth may show signs of rejection, since the endodontically treated tooth, due to loss of blood supply and subsequent infection—the essential factors in the definition of "gangrene"—, becomes a nidus of inflammation systemically, an ***inflamed foreign object***. The healthy dental organ, *odonton*, has an external and internal nerve and blood supply as well as intricate lymphatic drainage. Once this is compromised, the internal loss of blood supply renders the shell of the dental organ nothing more than a vessel harboring necrotic debris and micro-vermin.[17]

As one may recall, the death of a tissue (in the instant case the dental organ) due to loss of blood supply is termed *gangrene*. In texts of the early 20th Century the condition expressing as a "dying tooth" was called *gangrenous pulpitis*. This term gave over to the euphemistic term "devitalized tooth." Generally, this term is more acceptable to the patient.

A healthy dental organ has a general out-pressure, overcoming atmospheric pressure and releasing metabolites from the inside of the tooth.[18,] Thus, when the dental organ becomes gangrenous, looses its blood supply, the tooth becomes a "black hole" so-to-speak, a sink of sorts, in another sense a reservoir and allows the infusion of chemicals and microbes from the oral cavity.[19] The subsequent tissue response at the level of the root / bone interface of the dental organ will either be a granuloma, cyst, or cyst-like abscess. More exotic manifestations of the Natural Protection of the human body in an attempt to dissolve the corrupted tooth or so-called "modern dental implants" can be seen in Acute or Chronic Cavitation-Osteonecrosis, large voids either around the dental organ or where it once was. Another moniker for this is the *Giant Cell Granuloma* of yore.[20]

ORAL-SYSTEMIC LINKAGES TO GOOD AND ENDURING HEALTH

In review, historically, dentistry has been aware of the intimate connections between the condition of the dental organ (teeth), supporting structures of the bone, mucosa, the neuro-vascular-muscular system, and more recently, through the work of Dr. Voll, the inter-connections to the Channels of Energy Flow (meridians) found in Chinese Health Practice to general systemic health.

As reported in "Lancet" in 1911, physician Dr. Hunter of the London Fever Clinic identified and clarified the *oral-systemic axis*, causing severe and debilitating diseases such as colitis, chronic upper respiratory infections, chronic urinary tract infections, etc., and the resultant general human physiologic toxemia, which he termed *ORAL SEPSIS*. [22] From his groundbreaking work and lectures, notably at McGill University Medical College commencement in 1912, he demonstrated this relationship in a compelling manner. In fact, it is said that by his work alone the dental profession within the United States reversed its policy from retaining infected root stubs and root filled (root canalled) teeth supporting partial or full dentures to the mass extraction of teeth, identified as the "extractionist movement" in the early 1920s through the 1940s.

Dr. Weston Price, the notable but overlooked dental researcher of the 20th Century, corroborated this understanding during the 1930s, showing in his work for the first time the inclusion of microorganisms within the hard living tissues of the dental organ, the dentine. His work further demonstrated the arthritic and toxic systemic affects of gangrenous teeth upon the health of rabbits by inserting the teeth under their tissues. He spoke actively against the root canal therapy, "deep root fillings," for his work, like Dr. Hunter's, linked the oral infections to general debility.

A third researcher well known in Europe but not in the United States, was German physician Dr. Reinhold Voll who demonstrated with electro-technology, called the "Dermatron" device, the ill effects of infected and malposed teeth, odontons, on systemic health. His personal treatment policy was that until the person addressed the dental condition, his systemic treatments would not effectively help in reducing symptoms.

But with the advent of "modern endodontics" in the early 1950s and the commercialization of the dental profession, dental schools taught and encouraged the retention of the dead dental organ's shell—root canalled teeth—leading to the "save the tooth at all cost" era of dentistry for about 50 years, until year 2000.

In May 2001 "Scientific American" reported on the oral systemic linkage of oral bacteria found in the plaques of atherosclerotic patients,

patients with hardening of the arteries—atherosclerosis.[15] In 72% of the patient samples it was found that bacteria only found in the mouth had made their way through the vascular system and included themselves within the matrix of the vascular plaques. Moreover, the level of CRP—C-Reactive Proteins—were in some cases extremely high and might be a marker for the risk of heart disease. Implicated in these patients were those with acute and chronic periodontitis, an infected inflammatory condition of the roots and supporting structures of the teeth.

The author's research on the oral systemic linkage to good and enduring health reveals the potential deleterious effects of "modern root canal therapy" and "implant dentistry." Apical periodontitis is fundamentally equivalent to *periodontal disease, implant mucositic-granuloma* (dental implant failure), and may be termed, in fact, foreign body tissue reactions—"cleansing phenomenon," the natural physiologic pushing out of dead and dying tissues from within the human body.

With this in mind, one may be wise to avoid unclean foods, water, air and medical procedures.

Thank you for your kind attention, listening and consideration.

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