

Re: NICO Historical Review

Source: <http://sci.tech-archive.net/Archive/sci.med.dentistry/2005-03/3114.html>

From: John Chewter (john_at_LESS_SPAMchewter.f9.co.uk)

Date: 03/18/05

Date: Fri, 18 Mar 2005 15:21:53 +0000 (UTC)

My claim is that you do not understand the long medical terms that you quote.

This is backed up by your comments showing non-comprehension.

Case Proven

--

John Chewter

<http://www.keyneimage.co.uk>

"Joel M. Eichen" <joeleichen@yahoo.com> wrote in message
news:sj1315setievto2evl5iqvft454mkno53@4ax.com...

> On Fri, 18 Mar 2005 05:33:45 GMT, "LadyLollipop"

> <LadyLollipop@insightbb.com> wrote:

>

>>

>>"John Chewter" <john@LESS_SPAMchewter.f9.co.uk> wrote in message

>>news:d1cnqs\$cj6\$1@hercules.btinternet.com...

>>> Jan - do tell us that you understood all these papers? Some of the words

>>> are longer than 'Marmalade, Can you understand trans-marmaladic words?

>>

>>John, do back up you claims and you lies, rather than stalk me, then do

>>post

>>something besides criticisms of everything I post.

>

>

> John!

>

> Yeah, Jan wants to know about YOU claims and YOU lies

>

>

> (This is not standard English but its heard of the street.)

>

> YOU friend,

>

> Joel

>

>

>

>

>>

>>You have number of of questions you have never answered, is that your

>>problem?

>>

>>Shall I ask them again, and post you insults, rather than answers.

sci.med.dentistry: Re: NICO Historical Review

>>
>>Do grow up, John.
>>
>>As a claimed amalgamist, I asked you why you didn't post any articles
>>showing us why you believe amalgams are toxic, here is your reply:
>>
>>> LL Have you ever posted any studies of this toxicity????
>>
>>
>>JC Certainly not. I am an imaging specialist.
>>
>>So I kindly suggest, you shut up.
>>
>>LL
>>
>>> John Chewter
>>> <http://www.keyneimage.co.uk>
>>> "LadyLollipop" <LadyLollipop@insightbb.com> wrote in message
>>> news:TW8_d.76748\$Ze3.41700@attbi_s51...
>>>> <http://maxillofacialcenter.com/NICOhistory.html>
>>>>
>>>> The History of Maxillofacial Osteonecrosis (NICO)
>>>>
>>>> @The Maxillofacial Center for Diagnostics & Research
>>>>
>>>> Other Links
>>>>
>>>> NICO Clinical Page
>>>> NICO Home Page
>>>> Home Page
>>>>
>>>>
>>>>
>>>> Topics
>>>> Historical Overview
>>>> 1800-1930
>>>> 1930-1970
>>>> 1970-1990
>>>> 1990-2000
>>>> References
>>>> Tables
>>>> Painful osteonecrosis/osteomyelitis, or "phossy jaw," of upper and
>>>> lower jaws sloughed out when dentist tried to
>>>> extract several teeth because of "toothache." Source: American
>>>> Journal of Dental Science, 1859.
>>>>
>>>>
>>>> The Maxillofacial Center, 165 Scott Avenue, Suite 100, Morgantown, WV
>>>> 26508 USA
>>>> Phone: 304-292-4429 Fax: 304-291-5149 Email: MFC@aol.com
>>>>
>>>>
>>>> -----
>>>>
>>>> History of Maxillofacial Osteonecrosis (NICO)
>>>>
>>>> First described in 1794 in a case of septic necrosis of the femoral
>>>> head,
>>>> this enigmatic disease is as old as the dinosaurs but has been poorly
>>>> understood and has such subtle radiographic changes that until recently
>>>> it was seldom diagnosed prior to end-stage damage.[11-13] Contemporary

sci.med.dentistry: Re: NICO Historical Review

>>>> research has so enhanced our understanding of its basic pathophysiology
>>>> that it now bears little resemblance to the entity once known as
>>>> "aseptic
>>>> osteomyelitis."
>>>>
>>>> Heightened awareness and improved imaging techniques have confirmed
>>>> this
>>>> once rare disorder to be one of the most common of bone disorders. In
>>>> certain diseases, such as lupus erythematosus, almost a third of
>>>> patients
>>>> may be affected.[9] IO is able to affect any bone of the human
>>>> skeleton
>>>> and is represented by a large number of orthopedic diseases now seen as
>>>> simple anatomic- and age-related variations of intramedullary ischemia
>>>> and infarction.[1-5,9,14,15]
>>>>
>>>> The old, overly-simplified histopathologic definition of IO as massive
>>>> loss of osteocytes without pus is now substantially expanded to include
>>>> specific and often subtle signs of ischemic marrow damage which may not
>>>> even include obviously dead tissues.[2-9,14-16] Histopathologically
>>>> less
>>>> severe or nascent involvement has begun to be consolidated under a
>>>> common
>>>> diagnostic term, bone marrow edema (Table 1), and the disease is now
>>>> known primarily as a vascular disorder readily influenced by a variety
>>>> of
>>>> risk factors or trigger events ("hits") which promote
>>>> thrombosis.[7,9,17-21] Persons with multifocal IO are more likely to
>>>> suffer from systemic risk factors than those with single site
>>>> involvement
>>>> and the great majority of patients have inherited or acquired a
>>>> systemic
>>>> tendency toward fibrin generation (Table 2) which predisposes them to
>>>> microinfarction and ischemic marrow damage.[8,9,15-22]
>>>>
>>>> Usually associated with pain, IO can nevertheless show a surprising
>>>> capacity to remain painless until great destruction has occurred, even
>>>> to
>>>> the point of joint collapse for hip lesions -- there is little
>>>> correlation between the degree of bone involvement and the intensity of
>>>> associated pain.[5,9] The pain can take on a neuralgic character but
>>>> its
>>>> etiology is primarily a function of intraosseous fluid dynamics and
>>>> inflammatory mediators rather than damaged nerves, as discussed
>>>> later.[4,5,9,11,14-16]
>>>>
>>>> Top Of This Page
>>>>
>>>>
>>>> -----
>>>>
>>>> The Pre-Antibiotic Era: 1850-1930.
>>>>
>>>> IO of the maxillofacial region is not new to dentistry. During the
>>>> pre-antibiotic era "phossy jaw" and other forms of "chemical
>>>> osteomyelitis" resulted from environmental pollutants, such as lead and
>>>> the phosphorus used in safety matches, as well as from popular
>>>> medications containing mercury, arsenic or bismuth.[23-29] This
>>>> disease
>>>> was well established by 1867, did not often occur in individuals with
>>>> good gingival health, and appeared to "attack" the mandible first.[25]
>>>> It was associated with localized or generalized deep ache or pain,

sci.med.dentistry: Re: NICO Historical Review

>>>> 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.[24] Occasionally, large
>>>> fragments
>>>> of necrotic bone would come out with the tooth, sometimes involving
>>>> much
>>>> of an entire quadrant, as depicted in the figure at the top of this
>>>> page.
>>>> Apparently, Lorinser of Vienna in 1845 was the first to call attention
>>>> to
>>>> the problem.[25]
>>>>
>>>> Less severe cases of maxillofacial osteonecrosis were discussed in the
>>>> classic 1898 oral pathology text by Barrett,[28] wherein he described
>>>> "caries" and "necrosis" of bone with cellular "devitalization" and
>>>> "inhibition of nutrient currents," characterized by a slowly
>>>> progressive
>>>> "breaking down" of the "territory" of marrow tissues receiving those
>>>> nutrients and resulting in little or no production of granulation
>>>> tissue.
>>>> He had no suggested etiology for his cases. Thirty years earlier and
>>>> more than a century ahead of his time, Noel[27] separated bone caries
>>>> into two distinct categories: "bone death" and the less intense
>>>> "reduced
>>>> vitality." Even earlier, the 1848 text by Thomas Bond[23], which
>>>> appears
>>>> to be the first true oral pathology text, was the first book to discuss
>>>> bone necrosis as such, emphasizing that this disease did not require
>>>> abscessed teeth or gums, could result in the complete death of bone.
>>>> Bond mentioned that "necrosis may be caused by any means which destroys
>>>> the nutrition of the bone or any part of it"-- usually from
>>>> "constitutional vitiations, or defects of nutrition consequent upon
>>>> general pravity." His recommended treatment: "when necrosis has taken
>>>> place, the bone must be removed."
>>>>
>>>> G. V. Black,[29] the father of modern dentistry, described in 1915 an
>>>> osteomyelitis look-alike disease which he called "chronic osteitis." He
>>>> described slow bone death "cell by cell" with the creation of alveolar
>>>> intramedullary "cavities" up to 5 cm. in size and wondered about its
>>>> unique ability to produce extensive bone destruction without pus,
>>>> without
>>>> redness and swelling of the overlying tissues, without an increase in
>>>> the
>>>> patient's body temperature, and often without pain. His suggestion to
>>>> curette diseased bone reiterated the treatment proposed by Ferguson[24]
>>>> in 1868 and by Bond[23] in 1848. Around the same time period
>>>> osteonecrosis of the hip in children was being recognized by the
>>>> author's
>>>> whose names would eventually be affixed to that disease, i.e. the
>>>> Legg-Calvé-Perthes disease.[31-33]
>>>>
>>>> Top Of This Page
>>>>
>>>>
>>>> -----
>>>>
>>>> The Forgotten Decades: 1930-1970.
>>>>
>>>> For most of the twentieth century this disease was largely forgotten by

sci.med.dentistry: Re: NICO Historical Review

>>>> the dental profession, although a few investigators made significant
>>>> contributions to the advancement of our understanding. Wilensky[24]
>>>> and
>>>> Hankey[25] suggested that persistent regional necrosis in osteomyelitis
>>>> of the jaws was secondary to vascular insufficiency, while Brosch[26]
>>>> described the potential for hollow medullary spaces to enlarge and
>>>> coalesce one with another. Thoma[37,38] was likely the first to
>>>> specifically correlate this "residual infection" or "osteitis" with old
>>>> extraction sites, many of which demonstrated focal "necrotic exudates,"
>>>> fibrosis and "osteoclastic resorption" of surrounding bone. His
>>>> observations were affirmed in 1955 by Box,[34] who reported a very
>>>> large
>>>> series of limited intraosseous cavitations or "vacuolations" in old
>>>> extraction sites with no production of pus or bony sequestra. Box was
>>>> especially intrigued by the radiographic subtlety of the disease, by
>>>> its
>>>> multifocal nature, its localized tenderness without inflammatory signs,
>>>> and the neuralgia-like nature of accompanying pain.

>>>>

>>>> Top Of This Page

>>>>

>>>>

>>>> -----

>>>>

>>>> The Over-Emphasis of Pain: 1970-1990.

>>>>

>>>> The 1970s and 1980s saw a strong emphasis placed on the neuralgia-like
>>>> pains often accompanying osteonecrosis of the maxillofacial region, an
>>>> influence embodied in the currently popular diagnostic name NICO
>>>> (neuralgia-inducing cavitational osteonecrosis).[40-48] Significant or
>>>> complete pain reduction was achieved in chronic "idiopathic" facial
>>>> pain

>>>> by the simple expedient of decortication and curettage of damaged
>>>> alveolar bone (Table 3), supporting the contention by neural
>>>> researchers

>>>> that persistent odontogenic and osseous disease can be important
>>>> contributing factors for such neuralgias.44-49 None of these
>>>> investigations included a control group, nor has any facial neuralgia
>>>> follow-up study. Ethical considerations and the ever-present potential
>>>> for silent or subclinical disease will likely prevent valid control
>>>> groups from being identified, but a 1995 NICO follow-up investigation
>>>> confirming earlier surgical successes went so far as to guarantee
>>>> patient

>>>> anonymity, to use a well-established pain evaluation instrument instead
>>>> of surgeon records to determine outcomes, and to use a third party to
>>>> collect and analyze data in order to reduce potential biases.[50]

>>>>

>>>> Unfortunately, the major emphasis on the association with neuralgic
>>>> pain

>>>> initiated significant controversy among professionals treating
>>>> "idiopathic" facial pain and kept involved researchers from focusing on
>>>> other features of the disease process, such as more appropriate
>>>> diagnoses

>>>> and diagnostic techniques, and better understanding of the
>>>> pathophysiology and pathoetiology of the disease. Early lesions were
>>>> diagnosed by a number of independent pathologists as chronic
>>>> osteomyelitis, and microorganisms cultured from many NICO lesions,
>>>> combined with occasional facial pain relief with antibiotic therapy,
>>>> assured that these cases would be diagnosed and treated as chronic
>>>> osteomyelitis. And yet, a significant number of patients did not
>>>> respond

>>>> in a fashion appropriate to that diagnosis. This led some

sci.med.dentistry: Re: NICO Historical Review

>>>> investigators
>>>> to seek alternative interpretations for the biological behavior and
>>>> histopathology. A critical shift in perspective (return to the original
>>>> concepts?) occurred in 1989 when this odd alveolar disease began to be
>>>> viewed primarily as a problem of compromised medullary blood flow
>>>> driven
>>>> by progressive thrombosis, rather than as a unique infection unknown to
>>>> other bones.[56,57]
>>>>
>>>> This new perspective as a maxillofacial manifestation of IO provided,
>>>> for
>>>> the first time, a logical explanation for the curiously multifocal
>>>> nature
>>>> of the disease; its frequent intermingling of ischemically damaged and
>>>> normal marrow (also influenced by the perfusion irregularities of fatty
>>>> marrow[58]), its frequent lack of inflammatory cells, its remarkably
>>>> chronic and recurring character, its deep bone pain and varied pain
>>>> syndromes, its relatively high failure rate with local interventions,
>>>> and
>>>> its primary localization at the ends of the arterial inflow (retromolar
>>>> and subcrestal alveolar regions) where weak, irregular blood flow
>>>> favors
>>>> the formation of intravascular thrombi.[5,7,9,14,15,59]
>>>>
>>>> This is not to say that intraosseous microorganisms do not represent a
>>>> significant risk factor or triggering mechanism for thrombosis in these
>>>> stagnant zones of cancellous bone. Affected bone is ideal fodder for
>>>> periodontal and periapical bacteria chronically stimulating
>>>> inflammatory
>>>> and immune responses.[60-62] Impaired medullary circulation prevents
>>>> proper healing in these instances and the chronic infection, in turn,
>>>> enhances local and systemic clotting. This further exacerbates the
>>>> medullary ischemia and initiates a slow, ever-increasing spiral of
>>>> thrombosis and microinfarction with progressively elevating
>>>> intramedullary pressures, additional thrombosis, and frequent
>>>> propagation
>>>> of spontaneous pain. Prothrombotic factors, especially fibrinogen, also
>>>> allow increased adherence of bacteria to thrombin-activated endothelial
>>>> cells.[63]
>>>>
>>>> Top Of This Page
>>>>
>>>>
>>>> -----
>>>>
>>>> Decade of Major Advances: 1990-2000.
>>>>
>>>> Once it became clear that this disease of the jaws resembled avascular
>>>> necrosis of other bones, investigators used newly available laboratory
>>>> tests, including allele-specific polymerase chain reaction, to identify
>>>> in NICO patients heritable disorders predisposing to adverse thrombotic
>>>> events. At least 72% proved to be afflicted with a variety of such
>>>> disorders, as compared to 70-87% for patients with IO of the hip and
>>>> knee.[9,19-21,64-67 This was seen as a major breakthrough, eventuating
>>>> in
>>>> the use of anticoagulants (without surgery or antibiotics) in persons
>>>> with NICO and hip osteonecrosis.[68-71] Although not all affected
>>>> individuals benefited, the significant pain relief experienced by a
>>>> large
>>>> proportion of treated patients confirms an association in those persons
>>>> between the symptoms of IO and the hypercoagulable disorders.[69,71]
>>>>

sci.med.dentistry: Re: NICO Historical Review

>>>> Viewing NICO as the oral manifestation of a systemic disease also
>>>> allowed
>>>> application of the clinicopathologic qualities of long bone disease to
>>>> maxillofacial cases, especially the use of diagnostic imaging
>>>> techniques
>>>> such as 99technetium-MDP (99mTc-MDP) scintigraphy and Single Proton
>>>> Emission Computed Tomography (SPECT) scans, instead of the indium and
>>>> gallium scans typically used for bone infections.[64,72-74] The small
>>>> number of chronic inflammatory cells found in NICO lesions makes
>>>> radioisotopes which attach to leukocytes much less useful than those
>>>> which attach to new or exposed bone matrix. There is usually a small
>>>> amount of ongoing healing in IO lesions and so they present as "hot
>>>> spots" of increased radioisotope uptake, with "cold spots" of extremely
>>>> reduced uptake in the occasional severely desiccated lesion. Newly
>>>> developed 99mTc isotopes directed at fibrin "-chain peptide may prove
>>>> useful for patients actively forming microclots.[75]
>>>>
>>>> A substantial proportion (25-35%) of scans will be falsely negative
>>>> because the disease has long periods during which no bone is destroyed
>>>> or
>>>> regenerated, even as symptoms and marrow damage progress. This holds
>>>> true
>>>> regardless of the affected bone, but maxillofacial involvement suffers
>>>> from an unexpected false-negative phenomenon: radiologists not attuned
>>>> to
>>>> jawbone ischemia often interpret a hot spot of alveolar bone as
>>>> "normal,"
>>>> presuming it to relate to ubiquitous dental and periodontal disease. We
>>>> recommend, therefore, that the surgeon review all films interpreted as
>>>> negative. Thin-sliced spiral CT scans and ultrasonic scans have also
>>>> proven effective in localizing NICO, although they require very careful
>>>> evaluation.[76] MRI scans are valuable for the rounded ends of bones
>>>> but
>>>> in our experience are of little benefit in alveolar cases.[77,78]
>>>>
>>>> In a similar fashion the more contemporary histopathologic features of
>>>> ischemic osteonecrosis and bone marrow edema (its less severe
>>>> counterpart) often overlooked by or unfamiliar to oral pathologists,
>>>> could be applied to maxillofacial examples with the notable caveat that
>>>> there are no features of cortical collapse in jaw lesions and
>>>> odontogenic
>>>> infections are often superimposed.[1-9,57] Additionally, microscopic
>>>> evaluation of maxillofacial biopsy samples is made much more difficult
>>>> by
>>>> the small number and size of available curettage fragments, especially
>>>> when an intramedullary cavitation exists, in contradistinction to the
>>>> large specimens available for study after resection and core biopsies
>>>> of
>>>> long bone cases. Recent analyses have, significantly, reported that
>>>> almost 3/4 of jawbone biopsy samples of ischemic osteonecrosis and NICO
>>>> can be classified as the histologically more subtle variants called
>>>> bone
>>>> marrow edema or regional ischemic osteoporosis.[81,82] This has helped
>>>> to explain why some oral pathologists, certainly not the majority, have
>>>> difficulty distinguishing the classic features from "normal" bone and
>>>> bone marrow.
>>>>
>>>> The first microscopic review of a large series of biopsied cases of
>>>> NICO
>>>> was reported during the 1990s, as was the first necropsy
>>>> example.[57,82]
>>>> These papers strongly emphasized the multifocal nature of the disease,

sci.med.dentistry: Re: NICO Historical Review

>>>> while others reinforced the strong association between chronic facial
>>>> pain and inflammatory or ischemic marrow disease.[55,83] In this
>>>> light,
>>>> one of the most important advances was the refinement of the old
>>>> anesthesia/hyperesthesia and microanesthesia diagnostic tests to more
>>>> successfully localize areas of medullary disease in facial pain
>>>> patients.[84-86]
>>>>
>>>> During the 1990s sophisticated assays were also applied, for the first
>>>> time, to maxillofacial osteonecrosis. Haley and Pendergras[87] used a
>>>> well-established neurotoxicity assay on a very large number of tissue
>>>> samples, finding almost all to be extremely toxic -- often more toxic
>>>> than hydrogen sulfide, the chemical normally used to establish maximum
>>>> level of neurotoxicity. The exact nature of the toxin is not yet
>>>> known,
>>>> but the discovery of the neurotoxicity led some to question whether or
>>>> not this process damaged the peripheral nerve myelin of the alveolar
>>>> nerves. This idea was further stimulated by the finding in a small
>>>> number of NICO biopsy samples of an unusual form of nonwallerian
>>>> degeneration in the majority of visible nerves.[55] To this end the
>>>> blood of another small sample of NICO patients was evaluated by a
>>>> newly-established assay which, for the first time, allowed the
>>>> determination of circulating antibodies against peripheral nerve
>>>> myelin.[88] The sera of healthy humans normally show none of these
>>>> antibodies, as was true for a few of the NICO patients, but other NICO
>>>> patients had antibody levels as high as or higher than those found in
>>>> the
>>>> classic demyelination disease, the Guillain-Barré syndrome.[89,90]
>>>> This
>>>> suggests chronic exposure of the peripheral myelin to the immune
>>>> system,
>>>> either as a primary attack (autoimmune) or secondary to myelin exposed
>>>> or
>>>> partially destroyed by a local inflammatory/ischemic phenomenon.
>>>>
>>>> While some patients had no such antibodies, others demonstrated
>>>> Elevated
>>>> levels of circulating anti-peripheral nerve myelin (anti-PNM)
>>>> antibodies
>>>> have been found in NICO patients, suggesting .120-122 Chronic nerve
>>>> damage is likely enhanced by the very high levels of neurotoxicity
>>>> found
>>>> by bioassay in virtually all tissue samples of maxillofacial
>>>> osteonecrosis, although the responsible neurotoxins have not yet been
>>>> identified.123
>>>>
>>>> Top Of This Page
>>>>
>>>>
>>>> -----
>>>>
>>>> References
>>>>
>>>> 1. Burwell RG, Harrison MHM (eds). Symposium: Perthes' disease. Clin
>>>> Orthop 1986; 209:2-161,234.
>>>>
>>>> 2. Milgram JW. Radiologic and histologic pathology of nontumorous
>>>> diseases of bones and joints. Northbrook, Illinois, Northbrook
>>>> Publishing, 1990, vol 2, p 868.
>>>>
>>>> 3. Ono K, ed. Symposium: recent advances in avascular necrosis. Clin
>>>> Orthop 1992; 277:2.

sci.med.dentistry: Re: NICO Historical Review

- >>>>
>>>> 4. Schoutens A, Arlet J, Gardeniers JWM, Hughes SPF. Bone circulation
>>>> and vascularization in normal and pathological conditions. New York,
>>>> Plenum Press, 1993.
>>>>
>>>> 5. Steinberg ME, Steinberg DR. Osteonecrosis. In: Kelly WN, Harris ED
>>>> Jr, Ruddy S, Sledge CB. Textbook of rheumatology (4th ed).
>>>> Philadelphia,
>>>> W.B. Saunders; 1993, p 1628.
>>>>
>>>> 6. Mazieres B. Osteonecrosis. In: Klippel JH, Dieppe PA (eds).
>>>> Rheumatology. St. Louis, Mosby; 1994, 41.1.
>>>>
>>>> 7. Bullough, PG. Orthopaedic pathology (3rd ed). Baltimore,
>>>> Mosby-Wolfe,
>>>> 1997.
>>>>
>>>> 8. Jones JP Jr. Osteonecrosis. In: Koopman WJ (ed). Arthritis and
>>>> allied
>>>> conditions; a textbook of Rheumatology (13th ed). Baltimore, Williams &
>>>> Wilkins, 1997,1923
>>>>
>>>> 9. Urbaniak JR, Jones, JP Jr (eds). Osteonecrosis -- etiology,
>>>> diagnosis, and treatment. American Academy of Orthopaedic Surgeons;
>>>> Chicago, Illinois, 1997.
>>>>
>>>> 10. Phemister, DB. Repair of bone in the presence of aseptic necrosis
>>>> resulting from fractures, transplantsations, and vascular obstruction. J
>>>> Bone Joint Surg 1930; 12:769.
>>>>
>>>> 11. Russell J. A practical essay on a certain disease of bones termed
>>>> necrosis. Edinburgh, Neill and Co, 1794.
>>>>
>>>> 12. Martin LD, Rothschild BM. Paleopathology and diving monasaurs.
>>>> Amer
>>>> Scientist 1989; 77:460.
>>>>
>>>> 13. Norgard MJ, Carpenter JT, Conrad ME. Bone marrow necrosis and
>>>> degeneration. Arch Intern Med 1979; 139:905.
>>>>
>>>> 14. Arlet J, Mazieres B (eds). Bone circulation and bone necrosis.
>>>> Heidelberg, Springer-Verlag, 1990.
>>>>
>>>> 15. Ficat RP. Idiopathic bone necrosis of the femoral head: early
>>>> diagnosis and treatment. J Bone Joint Surg 1985; 67B:3.
>>>>
>>>> 16. Arnoldi CC. Intraosseous engorgement-pain syndromes. The
>>>> pathomechanism of pain. In: Arlet J, Mazieres B (eds). Bone circulation
>>>> and bone necrosis. Proceedings of the IVth International Symposium on
>>>> Bone Circulation, Toulouse (France), 17th-19th September, 1987. New
>>>> York,
>>>> Springer-Verlag, 1990.
>>>>
>>>> 17. Jones JP Jr. Intravascular coagulation and osteonecrosis. Clin
>>>> Orthop 1992; 277:41.
>>>>
>>>> 18. Van Veldhuizen PJ, Neff J, Murphey MD, et al. Decreased
>>>> fibrinolytic
>>>> potential in patients with idiopathic avascular necrosis and transient
>>>> osteoporosis of the hip. Am J Hematol 1993; 44:243.
>>>>
>>>> 19. Glueck CJ, Freiberg R, Glueck HI, et al. Hypofibrinolysis; a

sci.med.dentistry: Re: NICO Historical Review

>>>> common,
>>>> major cause of osteonecrosis. Am J Hematol 1994; 45:156.
>>>>
>>>> 20. Glueck CJ, Crawford A, Roy D, Freiberg R, et al. Association of
>>>> antithrombotic factor deficiencies and hypofibrinolysis with
>>>> Legg-Perthes
>>>> Disease. J Bone Joint Surg 1996; 78A:3.
>>>>
>>>> 21. Glueck CJ, Freiberg R, Gruppo R, Crawford A, et al. Thrombophilia
>>>> and hypofibrinolysis: reversible pathogenetic etiologies of
>>>> osteonecrosis. In: Urbaniak Jr, Jones, JP Jr (eds). Osteonecrosis:
>>>> etiology, diagnosis, and treatment. Chicago, Illinois, American Academy
>>>> of Orthopaedic Surgeons, 1997 p 105.
>>>>
>>>> 22. Mont MA, Jones LC, La Porte DM, et al. Symptomatic multifocal
>>>> osteonecrosis: a multicenter study. Clin Orthop 1999; 369:312.
>>>>
>>>> 23. Bond TE Jr. A practical treatise on dental medicine. Philadelphia:
>>>> Lindsay & Blakiston, 1848.
>>>>
>>>> 24. Am J Dent Sc 1859 [phossy jaw photo]
>>>>
>>>> 25. Anonymous. Necrosis of the lower jaw in makers of Lucifer matches.
>>>> Am J Dent Science 1867; 1 (series 3):96-97.
>>>>
>>>> 26. Ferguson W. New treatment of necrosis. Am J Dent Science 1868; 1
>>>> (series 3):189.
>>>>
>>>> 27. Noel HR. A lecture on caries and necrosis of bone. Am J Dent
>>>> Science
>>>> 1868; 1 (series 3):425, 482.
>>>>
>>>> 28. Barrett WC. Oral pathology and practice. Philadelphia, S.S. White
>>>> Dental Mfg Co, 1898.
>>>>
>>>> 29. Black GV. A work on special dental pathology (2nd ed). Chicago,
>>>> Medico_Dental Publ Co, 1915.
>>>>
>>>> 30. [old: discolored nerve]
>>>>
>>>> 31. Calve J Sur une forme particuliere de pseudo-coxalgie greffee sur
>>>> des formations caracteristiques de l'extremite superieure du femur. Rev
>>>> Chir 1910; 42:54-84.
>>>>
>>>> 32. Legg AT. An obscure affection of the hip joint. Boston Med Surg J
>>>> 1910; 162:202-204.
>>>>
>>>> 33. Perthes G. Uber Osteochondritis Deformans Juvenalis. Arch Klin
>>>> Chir
>>>> 1913; 101:779-807.
>>>>
>>>> 34. Wilensky AO. Osteomyelitis of the jaw. Arch Surg 1932; 25:215.
>>>>
>>>> 35. Hankey GT. Osteomyelitis (necrosis) of the jaws: it pathology and
>>>> treatment. Brit Dent J 1938; 63:552.
>>>>
>>>> 36. Brosch F. Die histopathologische Grundlage der Symptome bei
>>>> Keifer-osteomyelitis. Deutsch Zahnartz Zeit 1958; 13:426.
>>>>
>>>> 37. Thoma KH. Clinical pathology of the jaws, with a histologic and
>>>> roentgen study of practical cases. Baltimore, Charles C. Thomas, 1934,
>>>> p

sci.med.dentistry: Re: NICO Historical Review

>>>> 94.
>>>>
>>>> 38. Thoma KH. Oral surgery (vol 1). St. Louis, C V Mosby; 1948, p
>>>> 773.
>>>>
>>>> 39. Box RM. Post-extraction oral sepsis. Ontario Dent J 1955:
>>>> Oct/Nov:1.
>>>>
>>>> 40. Ratner EJ, Person P, Kleinman DJ, et al. Jawbone cavities and
>>>> trigeminal and atypical facial neuralgias. Oral Surg Oral Med Oral
>>>> Pathol
>>>> 1979; 48:3.
>>>>
>>>> 41. Roberts AM, Person P. Etiology and treatment of idiopathic
>>>> trigeminal and atypical facial neuralgias. Oral Surg Oral Med Oral
>>>> Pathol
>>>> 1979; 48:298.
>>>>
>>>> 42. Shaber EP, Krol AJ. Trigeminal neuralgia __ a new treatment
>>>> concept.
>>>> Oral Surg Oral Med Oral Pathol 1980; 49:286.
>>>>
>>>> 43. Mathis BJ, Oatis GW, Grisius RJ. Jaw bone cavities associated with
>>>> facial pain syndromes: case reports. Milit Med 1981; 146:719.
>>>>
>>>> 44. Demerath RR, Sist T. Treatment of osteocavitation lesions in
>>>> facial
>>>> pain patients: preliminary results. J Dent Res 1982; 61:218.
>>>>
>>>> 45. Wang M, Xiwei J, Qingrong I, Sanyou Z. [A study of the relation
>>>> between the various trigger zones of idiopathic trigeminal neuralgia
>>>> and
>>>> jaw bone cavities.] Acta Acad Med Sichuan 1982; 13:233.
>>>>
>>>> 46. Grechko VE, Puzin MN. [Odontogenic trigeminal neuralgia] Zh
>>>> Nevroptol Psikhiatr 1984; 84:1655.
>>>>
>>>> 47. Roberts AM, Person P, Chandran NB, et al. Further observations on
>>>> dental parameters of trigeminal and atypical facial neuralgias. Oral
>>>> Surg
>>>> Oral Med Oral Pathol 1984; 58:121.
>>>>
>>>> 48. Ratner EJ, Langer B, Evins ML. Alveolar cavitation
>>>> osteopathosis -- manifestations of an infectious process and its
>>>> implication in the causation of chronic pain. J Periodontol 1986;
>>>> 57:593.
>>>>
>>>> 49. Harris W: Forward. In: Wartenberg R: Neuritis, sensory neuritis,
>>>> neuralgia, a clinical study with review of the literature. New York,
>>>> Oxford University Press, 1958, p vii.
>>>>
>>>> 50. Mumford JM. Role of the dentist in trigeminal neuralgia. Pain
>>>> 1978;
>>>> 5:83.
>>>>
>>>> 51. Sisk AL. Management of chronic orofacial pain: surgical treatment
>>>> of
>>>> chronic facial pain. Anesth Prog 1983; 30:180.
>>>>
>>>> 52. Fromm GH, Terrence CF, Maroon JCL: Trigeminal neuralgia; current
>>>> concepts regarding etiology and pathogenesis. Arch Neurol 1984;
>>>> 41:1204.

sci.med.dentistry: Re: NICO Historical Review

>>>>
>>>> 53. Sessle BJ. Neurobiology of orofacial pain. Dent Clin North Am
>>>> 1987;
>>>> 31:595.
>>>>
>>>> 54. Raskin NH. Headache, 2nd ed. New York, New York, Churchill
>>>> Livingstone, 1988.
>>>>
>>>> 55. Bouquot JE, Christian J. Long-term effects of jawbone curettage on
>>>> the pain of facial neuralgia; treatment results in neuralgia-inducing
>>>> cavitational osteonecrosis. J Oral Maxillofac Surg 1995; 53:387.
>>>>
>>>> 56. Bouquot JE, Roberts AM, Person, P, Christian J. The histopathology
>>>> of neuralgia-inducing cavitational osteonecrosis (NICO). J Dent Res
>>>> 1989;
>>>> 68:952.
>>>>
>>>> 57. Bouquot JE, Roberts AM, Person P, et al. NICO (neuralgia inducing
>>>> cavitational osteonecrosis): osteomyelitis in 224 jawbone samples from
>>>> patients with facial neuralgias. Oral Surg Oral Med Oral Pathol 1992;
>>>> 73:307.
>>>>
>>>> Byron MA. A clinicopathologic review of 2278 NICO cases
>>>> (neuralgia-inducing cavitational osteonecrosis). Master's Thesis
>>>> (Endodontics). Morgantown, WV: West Virginia University, 1994.
>>>>
>>>> Bouquot J, McMahon R. Ischemic osteonecrosis of the jaws in 2,023
>>>> patients with facial pain. J Oral Pathol Med 1996; 25:271.
>>>>
>>>> Bouquot JE, McMahon RE. Ischemic alveolar osteonecrosis in 2,023
>>>> patients with chronic facial pain. J Orofacial Pain 1997; 11:180.
>>>>
>>>> Odell EW, Morgan PR. Biopsy pathology of the oral tissues. London:
>>>> Chapman & Hall, 1998: 268-270.
>>>>
>>>> 58. Kiaer T. Bone perfusion and oxygenation. Animal experiments and
>>>> clinical observations. Acta Orthop Scand 1994; 65 (Suppl 257):1.
>>>>
>>>> 59. Hiroshi I, Matsuno T, Kaneda K. Prognosis of early stage avascular
>>>> necrosis of the femoral head. Clin Orthop 1999; 358:149.
>>>>
>>>> 60. Lerner UH. Regulation of bone metabolism by the kallikrein-kinin
>>>> system, the coagulation cascade, and the acute-phase reactants. Oral
>>>> Surg
>>>> Oral Med Oral Pathol 1994; 78:481.
>>>>
>>>> 61. Stashenko P, Want C-Y, Tani-Ishii N, Yu SM. Pathogenesis of
>>>> induced
>>>> rat periapical lesions. Oral Surg Oral Med Oral Pathol 1994; 78:494.
>>>>
>>>> 62. Torabinejad M. Mediators of acute and chronic periradicular
>>>> lesions.
>>>> Oral Surg Oral Med Oral Pathol 1994; 78:511.
>>>>
>>>> 63. Shenkman B, Runinstein E, Tamarin I, et al. Staphylococcus aureus
>>>> adherence to thrombin-treated endothelial cells is mediated by
>>>> fibrinogen
>>>> but not by platelets. J Lab Clin Med 2000; 135:43.
>>>>
>>>> 64. Neville B, Damm D, Allen C, Bouquot JE. Oral & maxillofacial
>>>> pathology. Philadelphia: W. B. Saunders, 1995, p 631.
>>>>

sci.med.dentistry: Re: NICO Historical Review

- >>>> 65. Glueck CJ, McMahon RE, Bouquot JE, et al. Thrombophilia, hypofibrinolysis and osteonecrosis of the jaws. Oral Surg Oral Med Oral Pathol 1996; 81:557.
- >>>> 66. Gruppo R, Glueck CJ, McMahon RE, et al. The pathophysiology of osteonecrosis of the jaw: anticardiolipin antibodies, thrombophilia, and hypofibrinolysis. J Lab Clin Med 1996; 127: 481.
- >>>> 67. Glueck CJ, McMahon RE, Bouquot JE, Tripplet D, et al. Heterozygosity for the Leiden mutation V gene, a common pathoetiology for osteonecrosis of the jaw with thrombophilia augmented by exogenous estrogens. J Lab Clin Med 1997; 130:540.
- >>>> 68. Glueck CJ, Freiberg R, Glueck HI, et al. Idiopathic osteonecrosis, hypofibrinolysis, high plasminogen activator inhibitor, high lipoprotein (a), and therapy with stanozolol. Am J Hematol 1995; 48:213.
- >>>> 69. Glueck CJ, McMahon RE, Bouquot JE, Tracy T, et al.. Preliminary pilot study of the treatment of thrombophilia and hypofibrinolysis and the amelioration of the pain of osteonecrosis of the jaws. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998; 85:64.
- >>>> 70. Hammerschmidt DE. Thrombophilic osteonecrosis: another chapter. J Lab Clin Med 1997; 130:451.
- >>>> 71. Glueck CJ. NICO and anticoagulation therapy. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998; 86:5.
- >>>> 72. Langlais RP, Langland OE, Nortje CJ. Diagnostic imaging of the jaws. Baltimore: Williams & Wilkins; 1994:393.
- >>>> 73. Boudreau RJ, Griffiths HJ. Bone infarcts and osteonecrosis. In: Collier BD Jr, Fogelman I, Rosenthal L. Skeletal nuclear medicine. St. Louis: Mosby, 1996;293.
- >>>> 74. Bouquot JE, LaMarche MG. Ischemic osteonecrosis under fixed partial denture pontics: radiographic and microscopic features in 38 patients with chronic pain. J Pros Dent 1999; 81:148.
- >>>> 75. Thakur ML, Pallela VR, Consigny PM, et al. Imaging vascular thrombosis with 99mTc-labeled fibrin "-chain peptide. J Nucl Med 2000; 41:161.
- >>>> 76. Nicol K, Klingman J, Holt J, et al. Ultrasonic gum/jaw bone detection instrument. Thesis. Socorro, New Mexico, New Mexico Institute of Mining & Technology, 1996.
- >>>> 77. Larheim TA, Westesson P-L, Hicks D, Eriksson L, et al. Osteonecrosis of the temporomandibular joint: correlation of magnetic resonance imaging and histology. J Oral Maxillofac Surg 1999; 57:888.
- >>>> 78. Sano T, Westesson P-L, Larheim TA, Rubin SJ, et al. Osteoarthritis

sci.med.dentistry: Re: NICO Historical Review

>>>> and abnormal bone marrow of the mandibular condyle. Oral Surg Oral Med
>>>> Oral Pathol Oral Radiol Endod 1999; 87:243.
>>>>
>>>> 80. Bouquot J, McMahon R. Bone marrow edema syndrome: new disease or
>>>> early presentation of ischemic osteonecrosis? J Oral Pathol Med 1998;
>>>> 27:346.
>>>>
>>>> 81. Bouquot J, McMahon R. Bone marrow edema syndrome, independent
>>>> disease or early presentation of ischemic osteonecrosis? Proceedings,
>>>> Annual Meeting, American Academy of Oral & Maxillofacial Pathology;
>>>> Williamsburg, Virginia; April, 2000.
>>>>
>>>> 82. Adams WR, Spolnick KJ, Bouquot JE. Maxillofacial osteonecrosis in
>>>> a
>>>> patient with multiple facial pains. J Oral Pathol Med 1999; 28:423-432.
>>>>
>>>> 83. McMahon RE, Griep J, Marfurt CP, et al. Local anesthetic effects
>>>> in
>>>> the presence of chronic osteomyelitis/necrosis of the mandible:
>>>> implications for localizing the etiologic sites of referred trigeminal
>>>> pain. J Craniomand Pract 1995; 13:212-226.
>>>>
>>>> 84. Brown RS, Hinderstein B, Reynolds DC, et al. Using anesthetic
>>>> localization to diagnose oral and dental pain. J Amer Dent Assoc 1995;
>>>> 126: 633-641.
>>>>
>>>> 85. McMahon RE, Adams W, Spolnik K. Diagnostic anesthesia for referred
>>>> trigeminal pain, Part I. Compendium Cont Educ Dent 1992; 11:870-881.
>>>>
>>>> 86. McMahon RE, Adams W, Spolnik K. Diagnostic anesthesia for referred
>>>> trigeminal pain, Part II. Compendium Cont Educ Dent 1992; 11:980-997.
>>>>
>>>> 87. Haley BE, Pendergrass JC. <http://www.altcorp.com>. [Affinity
>>>> Labeling
>>>> Technologies; University of Kentucky]
>>>>
>>>> 88. Koski CL. Humoral mechanisms in immune neuropathies. Neurol Clin
>>>> 1992, 10:629.
>>>>
>>>> 89. McMahon R, Bouquot J, Mahan P, Gremillion H. Elevated serum
>>>> peripheral nerve anti-myelin antibody titers in atypical facial pain
>>>> patients with NICO. J Orofacial Pain 1994; 8:104.
>>>>
>>>> 90. McMahon R, Bouquot J, Mahan P, Saxen M. Elevated anti-myelin
>>>> antibodies in patients with maxillofacial osteonecrosis (NICO). J Oral
>>>> Pathol Med 1998; 27:345-346.
>>>>
>>>> Top Of This Page
>>>>
>>>>
>>>> -----
>>>>
>>>> Table 1: Alternative diagnostic names used for bone marrow
>>>> edema and ischemic osteonecrosis.1-9,14-16
>>>>
>>>> Bone Marrow Edema
>>>> Ischemic Osteonecrosis
>>>>
>>>> Arlet Type I osteonecrosis
>>>> Bone compartment disease
>>>> Bone marrow edema syndrome
>>>> Chronic traumatic edema

sci.med.dentistry: Re: NICO Historical Review

>>>> Medullary engorgement-pain syndrome
>>>> Migratory osteolysis
>>>> Migratory osteoporosis
>>>> NICO *
>>>> Post-traumatic painful osteoporosis
>>>> Post-traumatic reflex dystrophy
>>>> Primary algodystrophy
>>>> Regional ischemic osteoporosis
>>>> Regional osteoporosis
>>>> Roentgenologic transient osteoporosis
>>>> Sudeck's disease (RSD) **
>>>> Transient bone marrow edema syndrome
>>>> Transient demineralization
>>>> Transient ischemic osteoporosis
>>>> Transient marrow edema
>>>> Transient osteoporosis
>>>> Transitory demineralization in pregnancy
>>>> Aseptic necrosis
>>>> Aseptic osteomyelitis
>>>> Aseptic osteonecrosis
>>>> Avascular necrosis
>>>> Bone infarction
>>>> Coronary disease of bone
>>>> Ischemic necrosis
>>>> NICO *
>>>> Osteochondrosis desiccans
>>>> Perthe's disease

>>>> * NICO: neuralgia-inducing cavitational osteonecrosis
>>>> ** RSD: reflex sympathetic dystrophy

>>>> Return to Text Top Of This Page

>>>> Table 2: Coagulation disorders found in patients with ischemic
>>>> osteonecrosis of the hips, knees and jaws. These are compared to the
>>>> proportions found in patients with deep vein thrombosis of soft tissues
>>>> and with the normal population. Resulting proportions do not total 100%
>>>> because some patients had multiple disorders. Modified from Bouquot JE,
>>>> LaMarche MG. J Pros Dent 1999; 81:148-158.

>>>> Normal Population
>>>> Deep Vein Thrombosis
>>>> Osteonecrosis
>>>> Thrombophilia
>>>> Hereditary types*
>>>> 2-5%
>>>> 5-9%
>>>> 50-70%
>>>> Acquired types
>>>> 3-7%
>>>> 20-50%
>>>> 33%

