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The unfolding science of osseointegration

- *The science of osseodensification*
- *The immune foreign body reaction theory of osseointegration*

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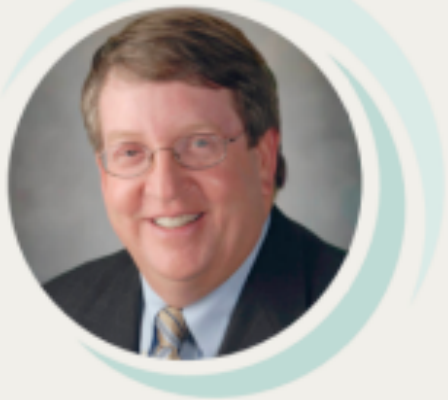
Hybrid Implant Designs – Tissue specific manipulation of the host response

By: David L. Cochran, DDS, MS, PhD, MMSc; Academy News Guest Contributor



Dr. David L. Cochran

I was asked by Dr. **Mehrdad Favagehi** to write a commentary on three articles published in the *Academy News* about hybrid implant designs by Drs. **Daniel Buser**, **Gerald A. Niznick** and **Dennis P. Tarnow**. Let me first say that I highly respect all of these "giants" in the implant field and greatly admire their incredible contributions. Each are very intelligent, and I am both honored and fortunate to call them friends. Both Drs. Buser and Tarnow point out the historical development of hybrid designed implants and then relate these to the development of peri-implantitis.

Dr. Niznick draws an important distinction that has been learned in this history and that is that a surface created by a subtractive process is better than an additive process since roughness is created without porosity. Porosity allows for the creation of microbial niches that are difficult to eradicate. Dr. Niznick also brings up an even more significant issue, which is the creation of implant component interfaces and their location relative to the bone level. This is a largely underappreciated factor with implants, but it is crystal clear that a microbial niche is created in these interfaces which harbors bacterial growth 360 degrees around the implant. The host cannot eliminate the bacteria and their associated inflammation, so the host resorbs bone and uses epithelium to try to isolate the host insult.

Add movement of the components creating the interface (conical and internal being best) and you can appreciate that a significant host response (involving bone loss) is developed. Think about another situation where bacteria penetrate the body in the form of an infected root canal. The body responds by resorbing bone at the apex and forming a cyst which is an epithelial lined cavity since again, the body cannot eliminate the bacteria. Our bodies have a spatial relationship between inflammation and bone that is well documented[1]. It has been called an "extended arm of inflammation"[2] or a "radius of infection"[3] or what Dr. **Dana Graves** and I have published, is an "inflammatory front" that develops and its proximity if close to bone, stimulates bone resorption[4,5]. These relationships today are known as a new area of science called osteoimmunology[6,7].

I would propose however, that there is broader context that should be addressed when considering dental implants and their designs. This broader context can be framed by asking what happens when a foreign material is introduced into the body? Scale makes a difference but the response in the host is always the same, an immune response.

Consider any antigen that is introduced, an immune response develops with antibody production. Larger items are generally isolated by the host (if the host is able to do that). Think of piercings in the body. With soft tissues, the body isolates the foreign body: pierced ears, nose rings, studs, pierced tongues, for example. Even amalgam pieces become embedded in soft tissues as amalgam tattoos. In other words, the host (and immune system) responds in some way to the foreign body.

A dental implant is not different. As a foreign body, we place the implant in both bone tissue and soft tissue. Dental implants should be considered from the point of view of how the specific tissues react to the foreign body being placed into it. What is the host response of the specific tissue where we place the implant? We know that bone tissue tolerates certain metals (and ceramic) well but what is important is that the bone tissue sees this as foreign material and therefore reacts immunologically to wall off the material with new bone tissue to actually protect the rest of the bone and importantly the bone marrow.

You may call this "osseointegration" but it is in fact an osseoimmune reaction to a foreign body, where the host bone walls off the foreign body through an immune reaction and many examples exist where this occurs. Think about fixation devices such as oral surgery plates and screws, distraction devices, knees and hips. Think also of creating an experimental saddle type defect in an edentulous osseous ridge. The bone heals across the cut bone surfaces and walls off the bone marrow[8]. Same concept for broken long bones.

Soft tissue is the other host tissue where the implant is placed. As noted above, soft tissues tolerate foreign material generally well particularly if the epithelium can encapsulate the material. Most pierced ears do not become infected nor do tongue rings. The host immune response is mitigated in these instances due to the epithelial encapsulation. Buser et al.[9] published an interesting finding in 1992 after he placed implants with three different rough surfaces in the soft tissue of animals. He found, "The different surface textures did not influence the healing pattern of the soft tissues…" In other words, the host (immune) response was the same regardless of the surface characteristics. In fact, a "scar-like" soft tissue capsule formed devoid of blood vessels.

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Hybrid Implant Designs... *(continued from page 14)*

Sleeping implants do not get peri-implantitis. It is therefore clear from the above that the implant foreign body itself is compatible with soft tissues and hard (bone) tissue. In other words, the body's immune response forms an equilibrium compatible with tissue health. Our studies on biologic width were the first to show that for non-submerged implants (now called tissue level) the biologic width dimensions and reactions were similar to those found around teeth[10-16].

Our 1997 paper on crestal bone changes revealed that the pathological crestal bone loss that occurred around Brånemark-type implants in the first year was directly associated with second stage surgery and the creation of a contaminated interface between the implant and abutment at uncovering[17]. The amount of bone loss was also directly related to the vertical location of the interface in regard to the bone crest which correlated to the inflammatory response and its spatial relationship to bone. The closer the infection (interface bacteria) to bone, the more bone loss occurred.

Two later publications by our group demonstrated that the host inflammatory reaction was localized adjacent to the interface and consisted of predominantly a PMN response (meaning it was a persistent acute response and did not become chronic). Moving the interface (and associated inflammation) coronally resulted in less bone loss and moving it apically, more bone loss[18,19]. It also explained the 1.5-2.0 mm of first year bone loss around Brånemark implants described as success criteria for these type implants by Albrektsson et al.[20] This bone loss stops as the inflammatory response has moved a sufficient distance from the bone crest[21]. The two Derks studies[22,23] referred to by Buser can also be viewed from an interface perspective where one implant was a tissue level implant (interface away from the bone) compared to two bone level implants with a contaminated interface placed at the bone level and resulted in greater bone loss.

I would posit then that when peri-implantitis occurs it is not related to the implant surface characteristics per se but rather a dysregulation of the equilibrium of the host immune response in the tissues. One can envision that the contaminated interfaces (one source of bacteria) stimulate a host inflammatory reaction in the soft tissues and that this inflammation as it approaches bone results in RANKL production, osteoclast formation and bone loss. Thus peri-implantitis is an immuno-inflammatory reaction that disrupts tissue homeostasis.

Of course, many factors other than plaque accumulation or interface bacteria may stimulate the inflammation or cause the breakdown of the material-host-equilibrium such as cement particles, broken components, compromised host, etc. We have recently published two reviews related to this topic and these are included in the reference list[24,25].

In summary, rather than focus on specific surface characteristics, a broader context is needed where the foreign material (implant)-host immune response is the focus. The majority of implants exist in harmony (equilibrium) with the host soft and hard tissue immune response however, in specific circumstances, this immune equilibrium is disrupted. This disruption can occur in either the soft tissues or in the hard (bone) tissues and can cause a loss of integration (the bone shield). When are we going to stop putting contaminated interfaces at or below the bone crest?

Editor's note: Academy News *recently published an article series about Hybrid Design Implants by Drs. Daniel Buser, Gerald A. Niznick and Dennis P. Tarnow. We received great feedback from our readers about this topic. We asked AO past President and researcher on this topic, David L. Cochran, DDS, PhD to chime in as well. Please use the following QR code for references and also to link to the three previous articles about this subject mentioned by Dr. Cochran.*

- RESPONSE TO DRS. BUSER'S AND TARNOW'S ARTICLES ADVOCATING FOR HYBRID IMPLANTS SURFACES ON ALL DENTAL IMPLANTS: AO ACADEMY NEWS VOL. 33, #1, 2022
- IMPLANT SURFACE DESIGN AO NEWS, VOL. 33,No.2 1-2022 GERALD NIZNICK DMD, MSD - GUEST CONTRIBUTER
- DR. BUSER ACKNOWLEDGES THAT STRAUMANN'S TISSUE-LEVEL IMPLANTS ARE ROUTINELY PLACED ONLY 1MM SUPER-CRESTAL. "THAT IS THE WAY WE HAVE DONE TISSUE LEVEL IMPLANTS SINCE THE LATE 90'S.
- MICRO-MOVEMENT WITHIN A CONICAL CONNECTION PRODUCED TITANIUM PARTICLES THAT CAN MIGRATE AND CAUSE BONE LOSS.
- GEN5 NEW CONNECTION - COMBINING A CONICAL WITH A MORSE TAPER FOR A FRICTION FIT

New theory of osseointegration: The immune foreign body reaction

By: Mehrdad Favagehi, DDS, MS, Academy News *Editor*

Bone loss around implants is widespread. Early pioneers in implant dentistry theorized that some loss of marginal bone levels (MBL) around implants should be considered normal. But that theory doesn't hold as we see many implants without any bone loss.

Even those who believe some loss in MBL is normal, don't dispute the fact that bone loss due to peri-implantitis is a significant problem. A recent meta-analysis reports the overall average prevalence of peri-implantitis is 12%. This translates to a grade of B+ for implant dentistry. How can we improve our record?

Some implant leaders believe that a 12% shortfall may not be just a missing piece of the puzzle, it may represent an unseen black hole requiring a brand new theory to understand osseointegration.

In the early 1900s, a group of astronomers observed small anomalies that could not be explained by Newtonian theories in physics. Albert Einstein's general relativity theory could clearly explain the celestial anomalies including black holes. Einstein's theories transformed science and set the stage for many technologies in our modern lives. Developing new theories has been associated with many scientific breakthroughs.

In his article on page 14, AO past President, Dr. **David L. Cochran** provided us with insights about maintaining MBL, but also pointed out to a new theory of osseointegration based on the new field of osseo-immunology. According to this theory, the immune system works with the bone to mount a foreign body response to a foreign object – the dental implant. In other words, bone formations that we see around implants may be the result of an osseous shield that the host forms to protect itself from the implanted foreign material.

To those who attended the keynote presentation by Dr. **Ole T. Jensen** at our recent Annual Meeting, Dr. Cochran's ideas sound familiar. For a summary of this concept, please see the article on page six provided by Dr. Jensen. According to Dr. Jensen, after three decades even Dr. **Per-Ingvar Brånemark** embraced revisions in the original theory of osseointegration. Dr. **Tomas Albrektsson**, a co-investigator of Dr. Brånemark is also a leading advocate for the new theory of osseointegration based on osseointegration as a foreign body in equilibrium.

Over the years, we've made significant progress in our understanding of the factors that influence bone around implants. We've learned how to minimize bone loss around implants by recognizing patient risk factors, occlusion, implant and restoration position, design, and materials. We've seen the negative influence of sub-gingival cement and the positive influence of adequate peri-implant tissues on the bone around implants. We have explanations for the osseointegration of biocompatible materials based on our understanding of inflammation, wound healing, and bone remodeling. Should we stay on the same course, or do we need a new revolutionary theory as the next step forward?

Our overall record in implant dentistry shows that we still need breakthroughs to prevent peri-implantitis and loss of MBL around some implants. The new immune foreign body reaction theory of osseointegration seems plausible, now we just need scientific evidence to prove it.

The Editor's Editorial is intended to contribute to the dialogue on issues important to implant dentists. The views expressed in the editorial do not necessarily reflect the policy of the Academy of Osseointegration or its board of directors. To provide feedback about this edition, or to contribute as a guest author, please contact me at mfavagehi@yahoo.com. We will endeavor to publish pertinent comments or views when space permits.

Dr. Mehrdad Favagehi

Peri-Implantitis: An educator's perspective

By: Vincent J. Iacono, DMD; Academy News *Guest Contributor*

When educated by Professor Brånemark almost 40 years ago on the novel procedure of osseointegration of dental implants, it was essentially a technique that has evolved into a comprehensive discipline. Implants of machined Grade 1 pure titanium were routinely inserted into the anterior sextant of the edentulous mandible of patients under general anesthesia in an OR setting.

These osseointegrated implants often emerged through alveolar mucosa and supported hybrid prostheses. While peri-implant mucositis and peri-implantitis were not recognized at the time, complications of malposed implants (no CBCT scans), damage to vital structures, "spinners," "sleepers," and implant/restorative fractures were the concerns of the era. Yet times rapidly changed, as we began to place implants at virtually any edentulous site using concepts of guided bone regeneration, ridge expansion, sinus grafts and periodontal plastic surgical procedures.

The implant structures changed to Grade 4 titanium or its alloy for strength and some were plasma sprayed with either titanium or calcium phosphate salts. Peri-implant complications then became a major focus with the recognition that plasma spraying resulted in surfaces that when colonized by the plaque biofilm, tended to lead to significant circumferential alveolar bone resorption with loss of the implants. The plasma spraying days were over!

It was also noted that at times implants with cemented abutment crowns developed peri-implant bone loss. It was determined that this was due to excess cement extruding into the peri-implant area. However, when the protruding cement was removed during an access periodontal flap surgical procedure, there usually was a return to a normal osseointegrated state. It is likely that the adherent cement on the implant threads led to bone resorption and when removed the titanium surface was then able to osseointegrate[1].

The challenge to enhance the kinetics of osseointegration with a greater degree of bone contact led to the use of acid etching with or without sandblasting or expanding the oxide surface of machined/turned titanium implant surfaces to achieve moderate roughness. These implants have been superb for enhancing osseointegration. However, since their use, peri-implant mucositis and peri-implantitis have become significant complications that we have had to address. The incidence of peri-implantitis is increasing and its severity has similar risk factors to periodontitis including but not limited to a dysbiotic biofilm with aberrant host responses, uncontrolled Type-2 diabetes, and an unexplained genetic predisposition[2].

Karoussis and colleagues compared the outcome of implant therapy in patients with or without a history of periodontitis in a 10 year longitudinal study. They demonstrated that the incidence of peri-implantitis at the implant level was 28.6% in patients with a history of periodontitis, while it was 5.8% in patients without a history of periodontitis[3]. It is important that our patients have had appropriate periodontal therapy before implant placement and are compliant with biofilm control and maintenance visits. It is a challenge to treat lesions of peri-implantitis and to decide on whether to remove the failing implant. I leave it to clinical judgement for implant removal as to the extent of bone resorption and existing implant restoration.

For my periodontal residents, the incidence of peri-implantitis has been comparable to that reported in the literature[4,5]. The osseous defects resulting from lack of care for many years have either been severe horizontal bone loss or varying degrees of "moat" like defects. We have found that for lesions with horizontal bone loss, virtually no techniques have led to significant regeneration of bone coronal to the alveolar crest regardless of the thickness of the surrounding peri-implant soft tissue. In these cases, clinical judgement prevails as to apically positioning the soft tissue to the alveolar crest and expose the non-integrated coronal portion of the implant, flap access procedures for implant surface decontamination, or implant removal depending on the extent of bone loss.

We have found successful results for "moat" like defects that are dependent on the distance of the walls of the "moat" to the implant surface. The techniques employed include significant implant decontamination with "standard" acceptable methods[6] (e.g., hydrogen peroxide and saline rinses); followed with the use of a particulate osseous allograft/xenograft/biologic composite graft and covered with a tightly

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Dr. Vincent Iacono

Peri-Implantitis: An educator's perspective (continued from page 21)

adapted resorbable barrier using a "lifesaver" or "poncho" method. Copious lavage with a biologic and post-operative antibiotic are routine. However successful outcomes have not been 100% due to the extent and configuration of alveolar bone loss.

We now frequently use hybrid implants that have highly anodized surfaces at their coronal portions; avoid subcrestal placement of implants that negatively impact on the dynamics of the implant abutment connection; place the smooth surface area coronal to the crest (bone level implants) and through the transmucosal layer (tissue level implants). So, a new era begins with the hope that the incidence of peri-implant inflammation will be reduced. Whether there will be a reduction in the effects of traumatic occlusal forces, release of titanium particles, and implant restorative designs (e.g., zirconia) will depend on the incorporation of evolving guidelines from basic/clinical research.

Unfortunately, I sense that some clinicians are resistant to accept the reasons for complications and follow a biased judgement with a false sense of security. It is critically important that we use our best judgement and follow the accepted guidelines for successful outcomes and not be "clouded" by podium science in decision making.

References for this article are available via scanning this QR code.

IMPLANT SURFACE DESIGN AO NEWS, VOL. 33,No.2 1-2022 GERALD NIZNICK DMD, MSD - GUEST CONTRIBUTER

On implant surface designs

By Gerald Niznick, DMD, MSD, Academy News Guest Contributor

Dr. Daniel Buser's and Dr. Dennis Tarnow's articles in *Academy News*, Vol. 33, No. 1, 2022, advocated for a Hybrid Design (HD) implant surface, claiming it an important factor for avoiding peri-implantitis. A HD implant surface has a smooth neck with a rougher surface on the threaded portion. I agree with their prediction that a HD implant surface will be the prevailing implant design of the future, but I do not believe it is the only, or even the most critical, factor in preventing peri-implantitis.

The cause of peri-implantitis is multi-factorial. While peri-implantitis is related to bone loss, having a textured surface to the top of the implant has been proven to reduce bone recession.[1] Many popular, bone-level implants with well documented success rates do not have a HD surface. Straumann's tissue level implant has a smooth neck intended to be in the soft tissue with the textured surface extending down from the crest of the ridge.

Dr. Buser cites a Swedish 10-year study comparing three implants: Astra, Nobel Biocare and Straumann's Tissue Level implant claiming the latter exhibited significantly less peri-implantitis. Assuming part of the smooth neck of the Straumann TL implant was inserted in bone, which Dr. Buser seems to advocate, this would give it a hybrid bone interface. It also adds the variable that the implant-abutment connection would be supra-crestal. I believe that the stability of the connection and its relationship to the crest of the ridge is at least as important a factor in minimizing peri-implantitis as a hybrid surface.

Historically, exposure of porous implant surfaces, such as TPS and TiUnite, has been recognized to contribute to soft-tissue complications. Straumann replaced TPS with SLA (Sand Blasted/Acid Etched) in 1998 and Nobel introduced TiUltra in 2019, positioning the porous TiUnite 2mm below a relatively smooth collar. Textured surfaces, as differentiated from porous surfaces, have proven successful in preserving crestal bone height when extended to the top of bone level implants.[2]

While it is clear that a textured surface is beneficial at the bone-implant interface and a smooth surface from an oral hygiene standpoint, is beneficial in the trans-mucosal area, the dilemma is that the crest of the ridge is often not flat and will recede from surgical trauma, especially if there is less than 1.5 mm of bone surrounding the implant at time of insertion.

To view Dr. Niznick's full response, please scan the QR code.

Dr. Gerald Niznick

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2. Cavallaro JS Jr. Implant survival and radiographic analysis of proximal bone levels surrounding a contemporary dental implant. Implant Dent. 2011 Apr;20(2):146-56. doi: 10.1097/ID.0b013e31820fbc31. PMID: 21448024.

Editor's note: *The views above reflect the opinion of the author. It's an example of issues raised in lively scientific debates during the 2022 AO Annual Meeting where various groups from North America and Europe presented a 40-year review of the past and made predictions about the future of implant design. In accordance with the theme of the Annual Meeting, we had asked Drs. Daniel Buser and Dennis Tarnow to write articles strictly focusing on implant surface designs from a historical aspect and make predictions about the future.*

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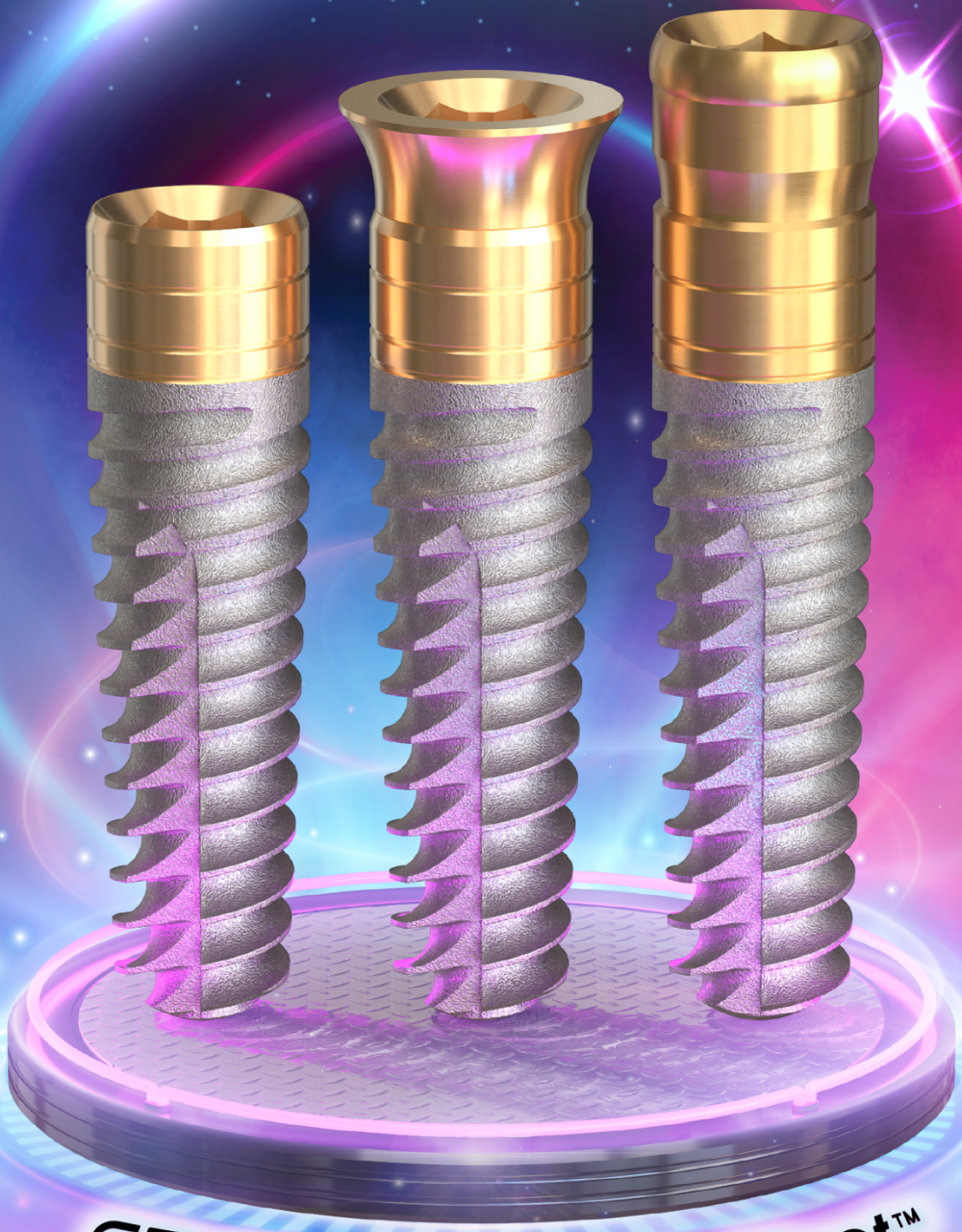

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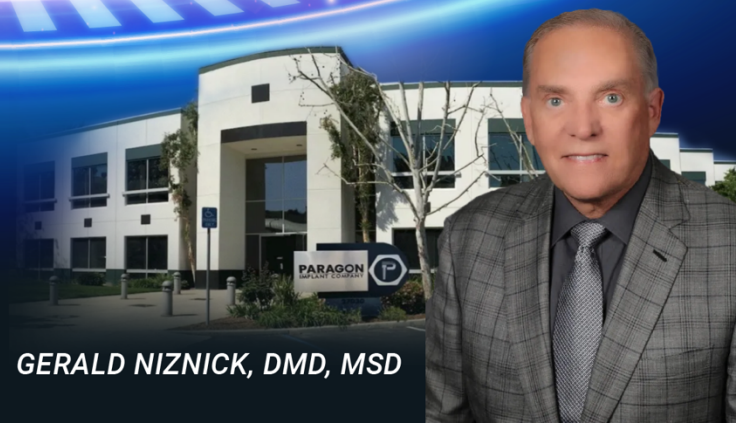

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