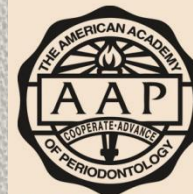


# THE ROOTS OF PERIODONTOLOGY

NMDHA 24<sup>th</sup> Annual Scientific Session  
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# A word about the value of history.....

Those who don't know history are destined to repeat it.  
Edmond Burke (1729-1797)

Those of you who don't remember the past are  
condemned to repeat it.

- George Santayana, 1890



# The History of Dentistry

- Barbers and blacksmiths were the first dentists in America
- 1840 – opening of the first dental school, University of Baltimore
- 1859 – ADA was founded, oldest and largest national Dental Society in the world
- 1890 – Dr. John Riggs describes periodontal disease
  - Calls it Riggs disease or pyorrhea
  - Marketed Anti-Riggs mouthwash (156 proof)
- 1896 – Dr. G.V. Black who is known as the father of modern dentistry, describes cavity preparations
- 1906 – Dr. C. Edmund Kells exposes first dental radiograph; also the first to use female dental assistants and surgical aspirators
- 1929 – Orthodontics recognized as first dental specialty



# History of Periodontics

- 1914 – Dr. Grace Rodgers Spalding and Dr. Gillette Hayden (both physicians) formed the American Academy of Oral Prophylaxis and Periodontology
- 1919 – became the American Academy of Periodontology
- Circa 1941 - Periodontics recognized as a dental specialty



# History of Hygiene

- 1867 – Lucy Hobbs Taylor graduated from Ohio College of Dental Surgery as a hygienist
- 1884 – paper presented at the NY 1<sup>st</sup> District Dental Society meeting advocating teeth cleaning for prevention, done by “staff”
- 1923 – formation of ADHA





# Historical names of periodontitis

- Loculosis
- Blennorrhea gingivae
- Periostitis
- Alveolodental periostitis
- Infectious arthrodental gingivitis
- Phagedenic pericementitis
- Expulsive gingivitis
- Symptomatic alveolar arthritis
- Smutz pyorrhea
- Riggs disease
- Periodontoclasia
- Pyorrhea alveolaris





# The roots of Periodontology

- The roots of the dental profession including periodontics and dental hygiene were not concocted by some entrepreneur.
- They evolved out of a recognition of dental diseases which affected a large segment of the population and had the potential to cause pain, illness and at times morbidity or mortality
- Early attempts at controlling dental diseases were **empirical guesses** based on observation. This stands in contrast to our current **evidence based treatment**.
- Logically, one of the first things to ascertain when dealing with a disease is to determine the cause of the disease. Additionally, there has to be an understanding of the natural history of the disease so that it can be treated at different stages and its progression evaluated.



# The cause of periodontal disease (periodontitis)?

- Early attempts at causation of periodontal problems hinted at plaque, but more generally it was debris. Primate cultures frequently demonstrate rudimentary hygiene practices
- The classic study demonstrating the cause effect relationship of plaque and gingivitis was done in 1965 by H. Loe
- This really opened the door to modern ideas of efficient plaque removal and plaque control
- 1985 Spindel et al published an article saying plaque really was not the cause of dental diseases



# Disease Activity in Periodontal Disease

- Plaque causes decay and periodontal disease. If plaque removal is not effective, you will suffer from dental disease. If you have gingivitis and fail to have it treated, it will go deeper and you will develop periodontitis. If you continue to ignore it, you will lose your teeth.
- How does this match what you are telling your patients?



# Disease activity in Periodontal Diseases

- This was the paradigm until the early 1980s. This worked for most people, however, we still had glaring discrepancies. Some patients with great plaque control had continued disease and some patients with no plaque control had no disease.
- Goodson, Haffajee, Socransky et al published several papers that described initiation and progression of periodontal disease as measured by attachment loss (1982).
  - Disease activity is episodic
  - Disease activity is characterized by bursts of activity
  - The best predictor of an at risk site.....a deep pocket
  - One of the poorest predictors of disease activity was/ is.....



# Disease activity

- One of the poorest predictors of disease activity was bleeding on probing
- The positive predictive value of BOP is about 32% (Haffajee et al, 1983)
- Lang et al, (1986) reported that a site that has not bled in the last 4 maintenance appointments is likely to remain healthy while a site that has bled 3 or 4 times out of 4 times has a 30% chance of breakdown.
- A coin flip is 50/50. But it has no predictive value
- This tells us that gingivitis does not necessarily progress to periodontitis



# Disease activity

- As the old paradigms stretched and broke new ideas were investigated
- Bacteria definitely are involved, but there were host factors as well as local factors. As time progressed, more and more host factors were (and are being) identified
- Michalowicz studies of the early 1990s using data from twins raised together and apart really started to identify genetic links in periodontitis
- Genetics now definitely play a part in treatment planning
  - Probably a resistance factor defect
  - Parental tooth loss from periodontal disease is gaining importance

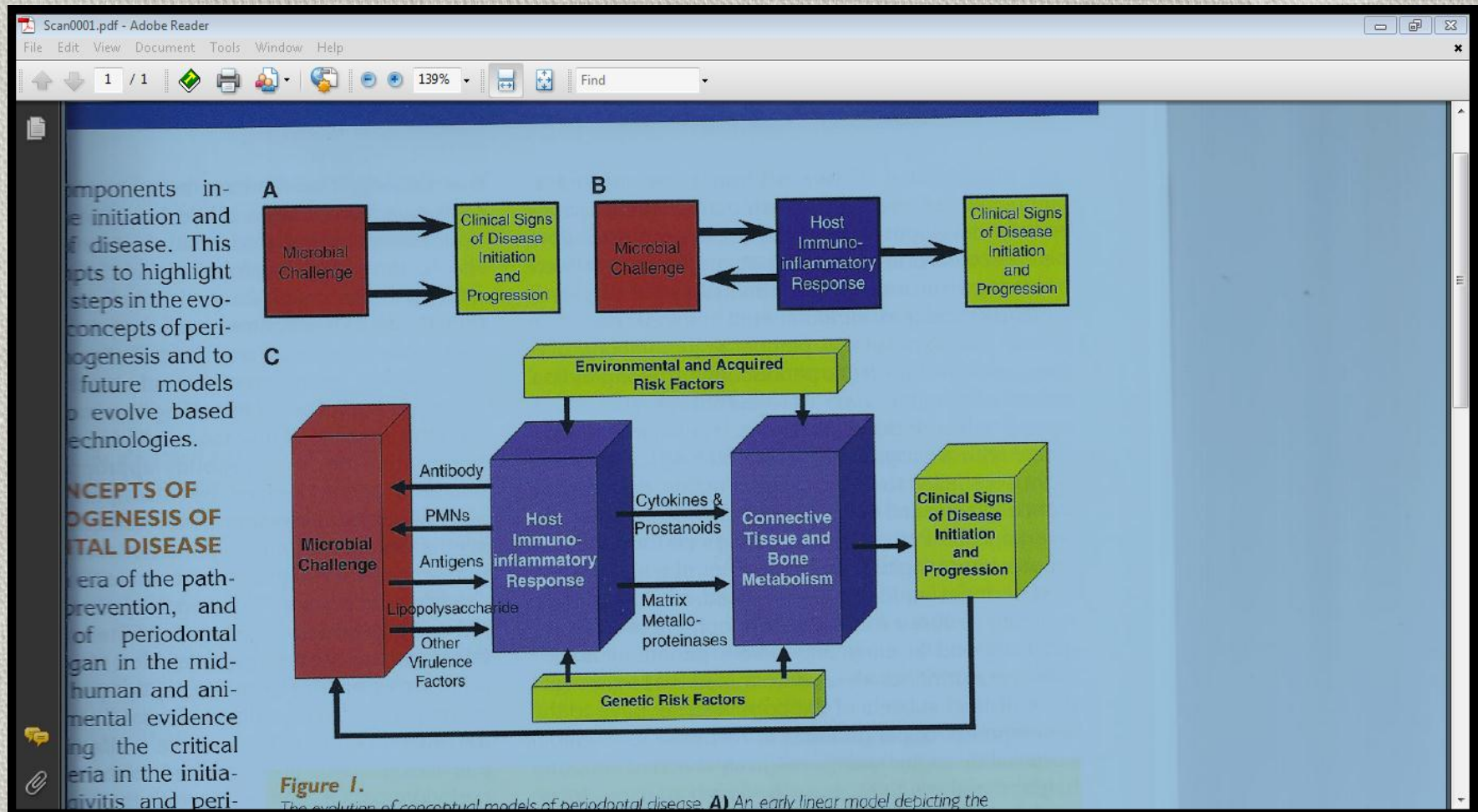


# Current models of disease activity

- Now we are realizing how complicated this is
  - Genetics are a factor. Not everyone is susceptible
  - The immune system function and dysfunction are important
  - The inflammatory response sets off local and systemic responses that are yet to be completely understood
  - We can safely say plaque control is critical to controlling periodontal disease, but there are a host of other factors to consider
  - This has become more apparent as we have started to see the interplay of oral and systemic disease



# Diagram of disease activity





# Disease activity in periodontitis: What I tell my patients

- The latest information available shows that about 80% of the population suffers from some form of periodontal disease. But not everyone is susceptible.....
- We know that periodontal disease is not a continuously progressing disease. It starts and stops and undergoes “bursts” of activity. We are poor at predicting where and when these bursts will happen, but we do know that they are most likely to happen in areas of deep pockets. Other things that increase risk for disease progression are stress, smoking .....
- Our treatment goal then is to get rid of the deeper pockets to reduce chances of disease progression. We don't cure this disease...we try and control it and hopefully prevent progression
- If you have the disease, we need to treat at several levels
  - Your homecare and overall health
  - Our professional care which may be non-surgical or surgical
  - Our maintenance care



# Why the history of disease activity?

- Throughout the young history of dentistry, treatment has mirrored the concept of the disease
- We need an accurate perception of the disease to provide proper treatment
- Initially treatment involved cutting out the affected tissue
- As the discovery of bacterial involvement matured, treatments to get rid of bacteria were central
- Treatments now that address only bacteria are inadequate
- Treatments that ignore overall health are inadequate
- Treatments that involve great plaque control are still essential!



# Links between systemic and oral disease

The AAP was started by physicians who saw a relationship between oral health and overall health

- Dr. Robert Koch (1870) described the germ theory of disease causation. This ushered in the age of microbiology
- W.D. Miller and W. Hunter (1900s) were dentists that were convinced that oral disease was responsible for disseminated disease. (Focal Theory of Infection)
- Up until 1960 common thinking was that you got 3 sets of teeth
- Up until the 1960s, a physician would tell patients to have their teeth removed for health reasons
- Thoden van Velzan described how oral problems could affect overall health (J Clin Periodontol, 1984) **Focal infection revisited**
- Have seen a resurgence of information in the last 16 years that is showing the link between oral and systemic disease
- Sentinel event was a 1996 study by Offenbacher et al relating pre-term, low birth weight babies to periodontal disease.



# Systemic and oral disease links

- In the late 90's the evidence began to mount regarding the link between cardiovascular disease (CVD) and periodontal disease
- Links were already known with diabetes, but relationships with renal and respiratory diseases were also attracting attention
- More importantly people started to look past what was associated with periodontal disease and they started to investigate the common denominator.....inflammation



# What about inflammation?

- We all had to study inflammation somewhere along the line
- Inflammation is a rapid, non-specific response to injury
- Cardinal signs of inflammation are pain, swelling, heat, redness and loss of function. The severity of the symptoms really dictate the magnitude of the inflammatory response
- An inflammatory response results in specific and non-specific mediators that can be measured
- C-reactive protein is a non-specific inflammatory mediator that can be measured in peripheral blood
- Many different diseases, known as **chronic inflammatory diseases of aging**, result in **and are affected by** high CRP levels



# Chronic inflammatory diseases of aging

- Diabetes
- Arthritis
- Alzheimer's disease
- Pancreatic cancer
- Head and neck cancer
- Asthma
- Osteoporosis
- Kidney disease
- Metabolic syndrome





# Systemic and oral disease links

- Current consensus is that inflammation is a common feature and thereby is a risk factor in common at least, synergistic at worst
- Even though the inflammatory response is non-specific, pathways resulting in previously mentioned diseases are very specific and are influenced by genetics as well as by environment
- Turning off inflammation is not automatic. Exercise, diet (Omega 3s) and rest are probably as important as any factors in overall management of inflammation
- More is appearing in the professional literature in regards to natural compounds.
  - Aparna et al. A comparative evaluation of the antibacterial efficacy of honey in vitro and antiplaque efficacy in a 4 day plaque regrowth model in vivo. Sept 2012 issue of JOP. The conclusion was that honey has an antibacterial and antiplaque activity. The rinse was 1.8 mg of honey per ml of distilled water.



# Why the history of the theory of focal infection?

- We are healthcare providers, not mechanics
- Our treatments are not just for dental health, they are for overall health
- A regimen of oral health should not neglect the rest of the body
- We don't fall into the trap that periodontal disease causes.....
  - We instead, want to be aware of systemic diseases that complicate periodontal disease and systemic diseases that are complicated by periodontal disease



# How has changing paradigms affected treatments for periodontal diseases?

- Non-surgical periodontal treatment
- Surgical periodontal treatment





# Evolution of treatments in Non-surgical periodontics

- Ancient cultures long ago conclude that some kind of oral cleaning was necessary to prevent oral problems, i.e. pain, odor, bleeding.
- By the 1900s we were ready to get rid of anything that was considered infected....teeth, gums, bone. As our understanding increased, it became an all out war on “infected cementum”
- 1965 Harold Loe et al did an ingenious study paralleling plaque accumulation and gingivitis formation. The conclusion was plaque is bad and we need to get rid of it on a daily basis
- It wasn't until the mid 1980's that it was argued that plaque removal (not total cementum removal) was the goal. Calculus removal was required because calculus harbors plaque (Waerhaug, O'Leary, Lindhe)
- Microscopic and ultra-structural studies verified the horrific – we don't get all the calculus off of teeth that we instrument.....and yet most patients have positive clinical results



# Evolutions of treatment in non-surgical periodontics

- Our initial preparation (SCRIP) primarily dealt with removal of junk...accretions, soft material, and selected hard material
- Scaling and root planing techniques were refined and changed as understanding of periodontal diseases changed.
- Clinical endpoints moved from completion of the mechanics to observation of patient response
  - Glassy hard cementum
  - Improvement in periodontal parameters
- Ultrasonics were introduced and instrumentation was refined.



# Evolution of treatments in Non-surgical periodontics

- Aside from these researchers we have actual clinicians adding their findings to periodontal literature
- The Ramfjord studies (1980's) were beneficial for the documentation of the clinical benefits of SCRP, for setting the one month reevaluation appointment and for setting the time interval for periodontal maintenance
- Removing granulation tissue during surgery or gingival curettage did not have any impact on healing
- The importance of periodontal maintenance was realized
- The move was on to make non-surgical treatment more effective
  - Keyes Technique
  - Fiber-optic instruments



# Recent adjuncts to SCRP

- What about local delivery of medicaments?
- What about the laser?





# Local delivery antimicrobials

- The temptation to put something in pockets that will have a sustained effect or improve clinical parameters is not new
- In the 1970s it was anti-formin
- In the 1980s it was betadine
- More recently it has been tetracyclines or chlorhexidine
- Most recently, alendronate, clarithromycin



# AAP position paper on local delivery antimicrobials

- LDAs deliver high concentrations of active ingredient in a vehicle that results in prolonged levels
- Use of LDAs with scrp result in less than 0.5mm probing depth improvements which is far out-shadowed by scrp. Effect on ALs were less than probing depths
- This included studies with Atridox (doxycycline hyclate), Arestin (minocycline microspheres), and PerioChip (CHX in gelatin matrix)



# AAP position paper on local delivery antimicrobials

- LDAs have not been shown to be effective in treating periodontal abscesses, furcations, peri-implantitis or special problems like smokers
- LDAs do not address anatomical problems inherent with disease and thus have not been shown to reduce the need for surgery or improve tooth retention **or to be cost effective**



# Tetracyclines

- Tetracyclines show the most promise
  - Anti-collagenolytic
  - Prevent epithelial migration
  - Are highly acidic
  - Have an extremely low to non-existent sensitivity rate
  - Can cause photosensitivity
  - Concentrate in the gingival sulcus (1600 mg/ml)
  - They are dirt cheap
  - They are active against *Aggregatibacter actinomycetemcomitans*
  - So why not give them orally



# Non-surgical periodontics with a laser

- Laser use has received attention since the early 1990s
- The 3 most commonly touted benefits of the laser are
  - Laser curettage or pocket debridement
  - Pocket sterilization or reduction of bacterial loads
  - Scaling and root planing



# Non-surgical periodontics with a laser

- Published statement from AAP in April 2011
- Laser mediated sulcular debridement has no benefit on pocket reduction over scaling and root planing and there is minimal evidence to support it as a monotherapy to SRP
- Pocket sterilization or reduction of bacteria; lasers have been unpredictable and inconsistent in reducing bacterial load beyond that provided by SRP or when provided as a monotherapy
- Use of lasers for SRP. Er:YAG lasers have been shown in vitro to remove calculus and negate endotoxin and have some potential, however, inability to observe the target area has resulted in root and cementum damage that is inconsistent with normal healing and functional attachment. More study is required.



# Non-surgical periodontics with a laser

- Ciannopoulou et al, Aug 2012. No apparent advantage in levels of acute inflammatory mediators when using a laser
- At this point it is not as good as scrp, it doesn't add to the benefit of scrp and it likely does damage to root surfaces



# The history of non-surgical treatment?

- The areas where the most important strides have occurred are the ones that increase our ability to clean root surfaces
- Splinter analogy...we are removing splinters and letting the body heal
- We need to be conscious of the rest of the body as well as the goal of your treatment.
- The goal should not just be good clinical parameters but, reduction in all risk factors and if possible, to provide an avenue for *tissue regeneration*



# Non-surgical periodontics

## The current state of practice

- Complete cementum removal is not desirable or advisable
- Gingival curettage is not necessary or beneficial
- Removal of local factors is still the goal
- Scrp will result in an average 0.4mm loss of attachment when performed on a healthy area
- Systemic antibiotics in conjunction with SCRP have repeatedly been shown to be of little value in the short term and no value long-term for sustained or improved clinical parameters
- Local delivery benefits are not commensurate with their cost in that they do not provide consistent long-term advantages in tooth retention
- The laser has as yet been of little value in non-surgical treatments



# Evolution of surgical periodontics

- The model in medicine is that if something is causing a problem and you can live without it.....get rid of it.
- The appendix is a modern day example
- Tonsils are a modern day example that really are an example of the fallacy with that kind of treatment
- This kind of thinking led to the early practice of tooth extraction for any reason
- As information about the disease was presented, treatments changed.
- Osteotomy (ledge and wedge, from the 1930s) was for removal of infected bone as in the diagnosis of periostitis
- Gingivectomy was used when it was considered that the gingival was at fault (Ramjord, Glickman, Goldman, Orban)



# Evolution of surgical periodontics

- The deep end....an unembellished gingivectomy was done without any initial calculus removal.
- The work of Warhaug and Loe in describing the role of plaque really opened the door for modern kinds of flap surgery
- When removal of calculus became the goal (instead of removal of sick tissue), different flap surgeries came into vogue
- As the dream of regeneration was brought into focus by Bowers et al in the early 1980s, flap surgery took on a new dimension



# Regeneration

- The formation of new attachment apparatus around a previously diseased tooth. Regeneration requires new bone, new cementum and a functional PDL
- This has become an achievable goal in selected kinds of defects
- It has been enhanced by the use of membranes
- Strong histologic evidence suggests most times we treat, we get repair which is in essence a scar. The real question is the ability of repair to resist continued or new episodes of disease activity



# The need for surgery in periodontics.....

The primary reason for surgery in periodontics has gone from a mindset of resection and removing damaged parts to enhancing a climate where regeneration can take place.

In this day and age, the primary reason for periodontal surgery is.....



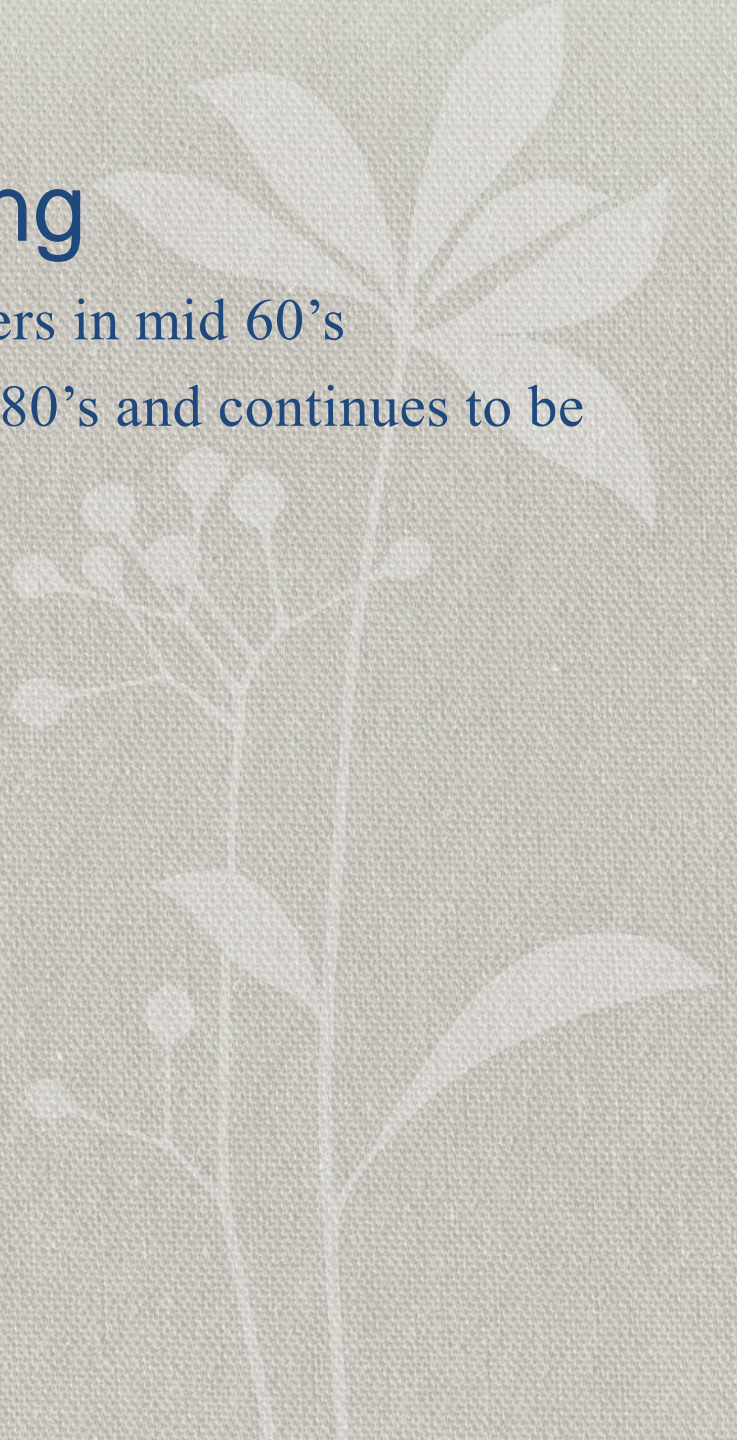
# The primary reason for surgery in periodontics is.....

- Access for debridement
- Secondary reasons include exploration, root amputation, biopsy etc
- The price to pay for surgical treatment of a healthy area.....loss of 1.2mm of attachment
- This has led to the popularity of MIS (minimally invasive surgery)
  - Surgery that involves only the affected surfaces
  - Far less invasive
  - Much less dramatic for patients
  - Equal or better results



# Surgical soft tissue grafting

- Free gingival graft was developed by Nabers in mid 60's
- The connective tissue graft evolved in the 80's and continues to be refined
  - Recombinant materials
  - Allografts
  - Autografts
  - Grafting in children has all but disappeared





# The laser in surgical periodontics

- A laser can be used in surgical periodontics. It is of particular value in cauterization, frenectomy and implant uncovering.
- Its slow
- Its can be painful post-operatively
- It harms cementum
- Break through bleeding





# The history lesson.....

- Dentistry, periodontics and dental hygiene are relatively young
- Dentistry in general and periodontics in particular have been and are presently on the leading edge of evidence based treatment. We look to reliable research and evidence that a particular treatment works or is beneficial.
- This has taken us out of the realm of ‘relief of pain’ to defining, treating and preventing disease
- This has taken us out of the realm of tooth mechanics and gum plumbers to health care providers
- Don’t allow marketing to take us back to mechanics



# Where are we headed?

- New and more effective instruments for debridement
- Recombinant genetic materials to grow bone, cementum and PDL
- Prevention – we need to defeat the “return to baseline” phenomena
- Managing inflammations associated with periodontal disease
  - Development of risk profiles for diabetes, periodontal disease and CVD that can be used in dental and medical offices
  - Develop risk criteria for embolic events in dental patients
  - Aggressively managing periodontal disease in diabetic, heart patients
  - Use of omega-3 fatty acid and ASA in treating chronic infection
  - Treatment can't just be to remove inflammation, it must return to homeostasis
  - New looks at local delivery



# • Questions?





# trivia

- Who set the interval to see your dentist at six months?
- Who set pmt at 3 mo and why?
- Gracey curettes....what was Gracey's first name? What was his first name?

