

<<Stating the obvious!!>>

Soft pink tissue of the pulp is dependent on its normal hard dentine shell for protection. As a price of this protection, the pulp contributes to closed symbiosis with its immediate environment with which it's confluent; mainly dentine and periodontal ligament. So,, pulp and periodontal ligament consist of two interdependent anatomical and functional entities. Pulp responses to different stimuli do not necessarily end at the "magical" border of the apical foramen; they can extend following the continuity of the connective tissue toward periodontal ligament and alveolar bone.

Periapical inflammation is the extension of the pulpal inflammatory process into the periapex and periapical lesions are areas of reaction to what has occurred within the root canal system.

When bacteria or toxins - from a necrotic pulp - advance beyond the apex periapical inflammation is established. First tissue affected is the periodontal ligament that shows radiographically widening around the apex of the root. This first tissue affected is also the last structure that returns to its original architecture and function.

So the incipient lesion start from the periodontal ligament around the apex of the root and this is the first tissue that is affected by inflammation and the last tissue - when healing occurs - that returns to its original function.

<<Concept!!>>

Attachment apparatus (PDL) is the vital organ of the tooth,,, viability of the tooth in the arch depends more on the health of attachment

apparatus than on the presence of vital pulp tissue within its root canal system.

<<Ready? Repeat! Repeat! Repeat!>>

In other words, viability of the tooth depends mainly on the condition of the attachment apparatus (PDL).

This means that "function and durability" of the tooth depend on its attachment apparatus.

Destruction of that vital organ (attachment apparatus) impairs the ultimate retention of the tooth.

<<Memorized!!>>

Even though root canal treatment involves working inside the root canal within the tooth, periapical tissues and their reaction determine the outcome of treatment – success or failure and conclusive healing in such cases.

<<Epic Battles!!>>

Outside the tooth the "battle" is joined with full resources of the body – in this battle full resources of the body are utilized .. the histological make-up of these lesions and special environmental conditions within the periodontal ligament will permit tolerance of edema and will conclusively affect the healing and repair process in this lesion.

<<A Fairy Tale!!>>

Canal anatomic and physiologic characteristics that contribute to this biological interdependence of pulp and periodontal tissues includes embryologic origination, vascular circulation, innervations, lymphatic

system, anatomic defense, and repair mechanism.

<<Back in time>>

The dental papilla forms the pulp and the dental sac forms cementum. Around 8 weeks of fetal life the first beginning of the dental papilla which will become the future dental pulp is seen, concurrently the mesenchyme surrounding the outside of the developing tooth will form the dental sac which will give the tissues of the periodontium – PDL, cementum, and alveolar bone.

Branches of the dental artery provide the pulp with blood, before entering the apical foramen arterioles of the pulp course through the periodontal ligament where they rarify. A plethora of arterio-venous anastomosis is usually observed between the pupal and periodontal vessels in this region. Another thing, nerve fibers before going upward through the apical foramen they can send branches to the periodontal ligament,,, this means that any inflammatory reaction in the periodontal ligament will affect the pulp tissue and vice versa – their circulation and innervation are the same.

<<Paradigm shift!!>>

The poly-dynamic character of the pulp - that is recently considered the base of new biological modalities in treating pulpal diseases – because the rich blood supply and the arterio-venous anastomosis in this area are reason why areas of pulp inflammation and necrosis co-exist with areas of healthy pulp tissue.

<<'ENDO' RULES!!>>

Multiple foramina are the rule rather than the exception!

<<Oorah!!>>

Main blood supply of the pulp enters through the apical foramina – these foramina are usually flood with periodontal tissue which is later replaced by cementum and dentine.

Communication between the pulp and periodontal ligament is not limited to the apical region, occasionally during the formation of the root sheath a break can develop in the continuity of the root sheath producing a small gap,, when this happens dentinogenesis does not take place opposing to that defect and the result is a small accessory canal between the dental sac and the pulp, usually it is filled physiologically with periodontal tissue,,, detection of these canals radiographically is extremely difficult and in the majority of case they become evident following obturation of the root canal system .. their presence can be suspected when there is a lesion on the lateral, middle, or bifurcation aspects of the tooth. As for their distribution, they are present at every level; in anterior teeth they are more frequently noted in the periapical lesion while a great many accessory canals are seen in the furcation area and account for the frequent presence of furcation radiolucencies in molar teeth.

<<So!>>

Following a periodontal disease lateral canals are exposed to the septic environment of the periodontal pocket and oral cavity ... when the periodontal lesion is deep also resorption can be seen within the root canal system opposite to the lateral canals – as the lesion is getting bigger the resorption becomes more obvious.

<<Leaks!!>>

The floor of an infected molar is more permeable than that of a non-infected tooth,,,

this means that there is a constant flow of material directly through the floor of chamber between the pulp and adjacent tissues ... This means that the inflammation of severe intensity in this region is due to the thin wall of the furca and also the normal presence of marrow spaces in this region which are susceptible to invasion in this area ,, the radiographic appearance (radiolucency) of the furcation is usually due to an auxiliary canal in the region or inflammatory products extending through the thin furca (pulpal floor that is more permeable in such case)

This communication between pulp and periodontal tissues is through dentinal tubules; despite their very small diameter, bacteria and their toxins can penetrate these tubules toward the pulp to cause damage.

<<More and more!!>>

There are also other factors that can affect the establishment of the endo-perio lesion; anatomical anomalies such as the enamel pearls which are small enamel droplets formed on the surfaces of or between the roots and they contain many opening through which pulps of these teeth could be affected, also there is vertical developmental radicular groove in the crown of upper central and lateral teeth in the region of the cingulum; usually it can cause untreatable periodontal condition and ultimately this will affect the vitality of the pulp ... and also iatrogenic factors such as perforation that can contribute in the establishment of endo-perio disease ...

<<'Pulpal' ATTACK!!>>

As for the effect of pulpal inflammation on the periodontium; pulpal inflammation does not always cause severe periodontal changes. What

happens is that the bacteria or their toxins can find their way through the apical foramen - accessory and multiple foramina - or through extension of the periapical granulomatous lesion and drain through the gingival sulcus - through the soft tissues - and cause periodontal damage.

So,, bacteria and their toxins that immigrated from the root canal system can results in the establishment of the periapical inflammation and periodontal changes and the formation of periodontal pocket draining via the gingival sulcus.

<<'Periodontal' ATTACK!!>>

As for the effect of the periodontal lesion on the pulp, these periodontal pockets extended to the apex can affect the vitality of the tooth causing damage ranging from hyperemia to necrosis.

<<An eye for an eye!!>>

Just as products from inflamed pulpal tissue can cause periapical inflammation, periodontal disease can cause pulpitis – retrograde or secondary pulpitis associated with periodontal disease and a vicious circle can establish >> periodontal disease results in inflamed pulp and the inflamed pulp will perpetuate the periodontal disease ...

<<Everything has a dark side!!>>

Also the effect of periodontal treatment should be considered; as we know when cementum is removed protection is lost so we will have sensitivity that can be also attributed to the inflammatory changes or hemorrhages in the pulp tissue.

<<18 lines to Classification Department>>

Moreover, this process is similar to the induction of acute inflammation of the pulp following cavity preparation – in deep scaling (especially in the furcation area of molar teeth) will result in loss blood circulation to a small area this will ultimately produce pain spasm and ultimate death of pulpal cells; a phenomenon comparable to the angina cardial attack! .. So what happens practically is that we will have a small area of infarction that develops in the pulp followed by coagulation necrosis.

<<Looking at the bright side!!>>

Of course not all 'curettements' of teeth will terminate with pulpal damage ...

<<Moving on>>

Attachment apparatus could be affected variety of diseases; endodontic, periodontal or occlusal origin.

<<Welcome to 'Classification Department'>>

As for their classification, they are classified on the basis of their etiology, clinical 'symptomatology', primary location of the lesion, and therapeutic treatment of the lesion.

*** we will depend on the "primary lesion" and "therapeutic treatment" of the lesion in our classification ***

According to that we have:

- Class I Endo-Perio lesion >> that is primarily or pure endodontic (pulpal/periapical) – due to inflammation or necrosis of the pulp. In other words, the primary cause of that

lesion is pulpal (endodontic) in origin, but! Symptoms clinically and radiographically simulate periodontal disease.

- Class II Endo-Perio lesion >> the primary cause is periodontal in origin, but! Symptoms clinically and radiographically simulate endodontic disease.
- Class III (true combined) Endo-Perio lesion >> both endo and perio lesions exist in the same tooth; and here we have two subcategories; (i) the lesion is pure periodontal but treatment requires endodontic therapy, (ii) the lesion is pulpal but treatment requires surgical periodontal therapy.

--- American terminology in diagnosis of pulpal and periapical diseases / 2013

<<Which is which?!>>

We have true puzzlement in endodontic diagnosis, because there is no single pulp testing technique can reliably diagnose all pulpal conditions neither it has been proven to be superior in all aspects ... <<simply>> "we do not have the ideal test!" ... so we have many limitations in diagnosis of endodontic diseases.

<<Schism!!>>

Historically, there are no dramatic changes or consensus for pulpal status in health or disease; and this is the reason for the diversity of opinions ...

<<Disgrace!!>>

50% of traumatized anterior teeth that reacted negatively to conventional pulp testing contained vital pulps when examined histologically ...

So clinical tests available can only test the ability of the pulp to respond to a stimulus which does not represent our best clinical judgment for the actual status of the pulp ..

<<DO NOT TRESPASS!!>>

The border line between 'reversibility' and 'irreversibility' of the pulp is very shallow, it is not easy to cross the line to be definite. According to that, treatment modalities is changing - even for the irreversibly inflamed pulp - recently.

So the straight forward diagnostic outcome in the clinic becomes more difficult to interpret particularly in the posterior teeth (molars) where the area will ache overall ...

<<CONFUSION!!>>

There is no test that we apply to the pulp itself. What we do that we apply stimulus to the external surface of the tooth (dentine or enamel) and we ask the patient to register response; we rely on his/her answer! Pain receptors of the patient are very vague; perception of pain differs from patient to another, besides that we do not have any measurement scale of pain!

<<It's a GOAT, even if it FLIES!!>>

In the lack of objective tests, it will not represent the true clinical situation of the pulp. So the same readings might be interpreted differently between dentists!

So diagnostic process is not a pure science, it is more of being "skill" ...

Practitioners should be equipped with the basic requirements and skills including; 'art of listening', knowledge, training, interest, curiosity, patience, and - above all - common sense ...

<<One step back>>

In 'Class I' which is 'pure endo' (the main cause is endodontic in origin with the symptoms of periodontal disease) >> the most significant sign is isolated pocket in a 'clean mouth' – the patient does not have periodontal disease in other areas of the mouth,,, it is very rare to have severe periodontal disease involving only

one tooth while all other teeth are relatively normal. Basis for diagnosis; is the loss of vitality – a 'strong' sign usually obtained by electric pulp tester. In other words, it is a must to have a non-vital tooth in order to say that this lesion is 'Class I' Endo-Perio lesion.

While the presence of pulpotomy, pulp capping, large restorations, deep carious lesion, or considerable diminishing of the pulp canal space; all these are signs that an endodontic disease or a primary pulpal disease is present. Periapical inflammation can find a pathway extending from the apex through the soft tissues and bone forming what "appears to be" a periodontal pocket. Such cases can be traced by gutta-percha radiographically. Here usually there is no true furcation invasion, because there is no communication between that radiolucency (in the furcation area) with true periodontal pocket. This is a "sinus tract" – not a true periodontal pocket, which means that there is epithelium proliferation, but! No

*Dental students
are HUNTERS!!
Hunting
'requirements'*

periodontal pocket formation. Periodontal probing in these cases is a 'conical probing' – short, abrupt probing. Usually the so-called pocket closes (heals) with only endodontic treatment. The radiolucency in the furcation area heals perfectly as well following the endodontic treatment. That means that probing depths will back to normal from mesial, buccal, to distal (all sites); a result which is rare - if possible – to obtain when we have a periodontal disease so rapidly. Worth saying that the root surface in that so-called pocket should not be scraped because - as we said - the attachment apparatus is the vital organ of the tooth; if we have viable periodontal ligament cells we do not want to remove them, because - later on - this will affect the healing process.

<<To sum it up>>

Class I endo-perio lesion "looks as if" periodontal therapy is needed, the primary cause is endodontic that requires endodontic therapy only, heals rapidly with an excellent prognosis.

<<C'mon! Let's pay Torabinejad a visit>>

<http://www.youtube.com/watch?v=nvM4zDF6stc>

<<Advertisement>>

Asia-Pacific endodontic confederation – president; Dr.Ibrahim Abu-Tahun; announces an international conference in 'Endodontology'; in which Dr.Torabinejad will be one of the lecturers of this conference. This will be held in Amman from 8th to 10th of April, 2015.

<<Back on track>>

In 'Class II' which is 'pure perio' (the predominant condition is periodontal) – the primary cause is periodontal. Symptoms might simulate endodontic disease. Periodontal probing will show increased pocket depth with plaque and calculus formation and the bony lesion is usually more widespread and generalized – so mouth must be examined for the existence of periodontal disease in other areas; this is very good indication that class II endo-perio lesion is present. Pulp testing should indicate vital response. Symptoms like draining sinus tract, loss of bone and/or soft tissue support, tenderness to percussion mobility, and swelling; all these are confused with endodontic diagnosis <<Be Careful!!>> .. A tooth with no restoration, caries-free - or only minimal restoration -, no fracture, and normal response to electric pulp tester; mostly indicates class II endo-perio lesion.

<<Back to Torabinejad>>

<http://www.youtube.com/watch?v=pPe0MO52TyE>

<<Stepping forward>>

In 'Class III' both 'endo and perio lesions' co-exist in the same tooth, hence the name 'true combined' endo-perio lesion. Any portion of the periodontium can be affected by extensive pulpal inflammation. A lesion that appears to be continuous from crestal bone to periapex could be two lesions that have joined – while a lesion that is extending from the periapex towards the crestal bone is a periapical lesion, and a lesion that is extending from the gingival sulcus apically is a periodontal breakdown. Both lesions could co-exist in the same tooth. Such

lesion could be primarily pulpal in origin with secondary apposition of periodontal disease, it could be primarily periodontal disease with extension to pulpal tissue, or it could be also pulpal and periodontal lesions in which disease processes exist independently in both tissues. Criteria for diagnosis; patient does have periodontal disease in multiple areas of the mouth. If both periodontal and endodontic diseases are present the treatment and prognosis change – if endodontic treatment only is performed the lesion will heal to the point where the periodontal lesion begins. On the other hand, if periodontal treatment only is performed the lesion will heal to the point where the endodontic lesion begins.

<<Keeping it simple>>

Perio treatment alone will induce healing of the perio component of the lesion while endo treatment alone will induce healing of the endo component of the lesion. So,,, in such cases - combined treatment is needed - endodontic treatment should be performed “first” – or at least at the same time of the periodontal treatment. Although the overall prognosis in these cases is questionable, endodontic component has a better chance for resolution – better prognosis.

<<Why?!>>

Because pathological content of the pulp cavity will provide the periodontium with a source of infection – if left untreated; so this will retard the healing process. When a periapical lesion heals, the incidence of recurrence is much less than - incomparable with - the periodontal lesion – that usually needs periodic maintenance.

*Looks like that
'requirements'
hunting season had
never been opened!*

<<20 lines to finish!!>>

To prevent periodontal complications; we can treat these teeth with non-setting calcium hydroxide; in order to induce fast remission of symptoms in these cases – this will improve the micro-environment within the root.

Endodontic therapy and root amputation as well may be required to gain healing for a periodontal (only) problem.

Typical indication where we have one tooth (multi-rooted) that is involved while the other teeth are relatively healthy or normal; so we do root canal treatment for that root (to be saved) and sacrifice the (non-healing) other one by doing root amputation.

<<5 lines to finish>>

Vertical root fracture is a ‘typical’ “endo-perio” lesion.

<<Worth knowing>>

Vital pulp does not always mean a normal pulp!
Inflamed pulp is a vital one!

<<Cheers!! WE DID IT!! OORAH!!>>

<<¡Perdóneme!>>