Guest Editorial

Root resorption revisited: The paradigm of force effect on root resorption: *Is a 'paradigm shift' needed in order to learn more about the phenomenon?*

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What do we know about the orthodontic root resorption phenomenon? Our answer is: not very much. Actually, practically, almost nothing. Yes, almost nothing. When we say almost nothing we mean that we know a lot about the 'other process' that takes place around the roots during orthodontic treatment, but almost nothing about the one that occurs in the apical region and is responsible for the root shortening phenomenon. For several years we have tried to convince the profession that data from short term studies cannot be extrapolated to long term treatments. Only recently, we have seen in the literature some doubts about the association between short-term in vivo studies and long term treatment related to root shortening. We were not the first ones to publish those doubts.

Albin Oppenheim, in his 1942 iconic paper,[1] had suggested a change in what was then the main paradigm related to root resorption (RR). To our surprise, this paradigm that emerged in the beginning of the 20th century, almost 100 years ago, remained so protected that even today, the Angle Orthodontist still publishes it.[2]

"I would suggest that the terms root resorption and cementum resorption be not used interchangeably. While root resorption denotes a real shortening of the root, the cause of which is not yet known, cementum resorption means only a loss of tooth substance not necessarily localized at the apex. The cause for its occurrence is well known. While root resorptions occur relatively seldom, cementum resorptions are always present in orthodontic procedures." Those phrases, that can be said even today, are quotations from the original 1942 article by Oppenheim.

"What do you imagine when you hear the expression 'root resorption' related to orthodontic treatment?" was the question of a verbal survey we made among dentists and orthodontists while attending a local convention. All of them, with no exception, described the well-known apical root shortening phenomenon. No respondent said that this expression referred to the immediate surface root resorption seen following force application. Those survey's results were in our minds when we went to present a lecture on RR update in front of residents in one prestigious orthodontic department in Israel. There, we began our lecture by asking the residents to list the parameters that make our teeth susceptible to orthodontic root resorption (ORR). The relatively long list began with the most well-known parameter, namely, the force, its level, direction, continuous versus intermittent etc. Genetics was mentioned among other parameters but nobody could point to the gene or the genetic combination that might stand behind what we relate to ORR. We are sure that similar answers would have been received if this question were raised among experienced professionals. As you can imagine, the references for the cited knowledge were mainly from more than 50 years of short term in-vivo studies. When we critically analyzed several studies, the residents were surprised to understand that, in contrast to what might have been concluded, real apical root shortening (ARS) was never demonstrated in those studies. All of them revealed only the initial cemental surface resorption that is the normal and expected reaction to the applied forces, namely the local defense mechanism at its best.

We all know that there is no tooth movement without orthodontically induced inflammation. We all know that this inflammation affects the bone and cementum, initially by surface resorption. We all know that most cemental resorption is later fully remodeled. We further know that no study ever demonstrated any relation between the amounts of this initial cemental surface resorption, on any tooth surface, to ARS. Therefore, how is it possible, although known to the researchers, that there are no changes in the definitions of ORR? How is it possible that editors continue to publish studies with clinical conclusions that might confuse the profession in this matter?

Thomas Kuhn, a philosopher of science, taught us that the scientific fields go through periodic "paradigm shifts" rather than progressing in a linear and continuous way, and that these shifts open up new ways to

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understanding that further study is needed in fields that scientists would never have considered valid before.[3] In the last few years, we tried several times to make Oppenheim's suggested paradigm shift alive again. However, it seems that the profession, even today, is not ready to make this paradigm shift. Initially we presented the profession the OIIRR[4] definition, (1st shift) that became partly acceptable by several researchers and, later, the term Orthodontitis[4] (2nd shift). While presenting the last paradigm shift, we differentiated between the normal defense mechanism of the PDL to any level of the applied force, namely the Instrumental Orthodontitis (referred by Oppenheim as cemental resorption) and the root shortening reaction, namely Instrumental Detrimental Orthodontitis (referred by Oppenheim as root resorption).

We think that our 2nd paradigm shift can really serve as the basis for a major change in the concept of ARS, from the devastating side effect of the treatment into what it really is: an inevitable defense mechanism outcome of the involved tissues. The level of this defense mechanism (measured by the amount of ARS) is determined by unknown or yet unproved factors (see the suggested list below) and, as far as our 100 years of professional experience taught us, in most cases does not affect the longevity, vitality, color and/or function of the teeth injured by this defense mechanism activation. The fact that this 2nd paradigm shift was ignored does not mean that it does not exist. We believe that studying it, discussing it out loud, might bring the change that we are so eager to know. Medical history is full of theories that were born many years before they could have been proved. Probably the most famous one was presented by Ignaz Semmelweis', the physician who, in the mid-19th century, demonstrated the importance of hygiene as a means to save lives in medicine without knowing the explanation to it.

Our main question is why does the profession not accept the fact that, during those 100 years, there is not even one study that found or confirmed the main question asked initially by Oppenheim (the subtext of his quoted phrases) and later by us: Is there a connection/relation/association between the results of short term in-vivo studies (up to 4 months) that describe histological/physiological/pathological changes on the root surfaces as a reaction to the applied forces, that most of them might be reversible, to the apical root shortening that is detected a few months later by imaging (X-rays, CBCT)? The second question is: How is it possible, although this connection was never found/verified, that almost each study implicates those short term results, with no methodological basis or proof, to our daily clinical treatment ways/systems?

We think that our objective is not to answer those questions, but to enlighten some alternatives or suggest ways to reassess this confusing field.

We believe that it is time that the profession should consider the theory that the initial reaction to the orthodontic force is like any other defense mechanisms that the body is abundant with. The initial PDL reaction, although different in each one of us, to the applied force, affects the root surfaces as well as the bone surfaces. Each surface reacts differently but, as Oppenheim said in his publication, it reacts. Without it, no tooth movement is possible. He also made clear that, under the microscope, the cementum demonstrates not only resorption sites but areas of new secondary cementum as well. On the cemental side, the zone that we are interested in, we see the activation of this defense mechanism, initiated by resorption. However, when enough time is given (several days), in most instances, many of the resorbed lacunae are being filled and no morphological changes of the root or cementum can be detected. The 'cemental lines' are the sole traces identified following this full cycle. This reaction can and probably should be different in heavy versus light or continuous versus intermittent forces (for example, the surface area of the craters), or on different root's surfaces. However, this reaction is not necessarily related, until proven otherwise, to the ARS that is seen following a much longer treatment time, which is much beyond the legal/ethical acceptable in-vivo study length.

What makes the apical zone more vulnerable to force activation? We know that this zone is totally different from other root surfaces and, unfortunately, it did not get the attention we think it should, especially from the researchers. Here is a partial list of parameters that differentiate the apex from other root surfaces (not including the 1/3 gingival part) that can be investigated:

1. The cementum in the apical zone is more cellular than on the other root surfaces.
2. The apex engulfs the bundle of blood vessels and nerves that supply the tooth.
3. The apex is the only place where the dentin/predentin and the cementum/pre-cementum meet. This junction might be a vulnerable point, similar to the cemento-enamel junction, where invasive cervical resorption begins. The apex is the only place where this cemento-dentin junction and the dentin/predentin are directly exposed to the outside surrounding of the root. On other surfaces, the dentin/predentin are away from the PDL.
4. During almost every movement, the apex moves the most (root wise), since this part is at the edges of the moving levers.

5. The apical zone anatomy is totally different from the cervical zone. When we apply force, let's say perpendicular to the tooth long axis, the cervical crestal bone is more bendable than the bone in the apical zone and, further, fluids can easily be moved from the PDL crestal zone to the oral cavity faster than in the surrounding apical bone spaces. As a result, the expected apical pressure is higher than the cervical pressure, and the defense mechanism can easily reach the insufficiency point there. It does not mean that the higher the pressure, the more ARS is presented, since we do not know whether there is an insufficiency point to the process.
6. We all know that deciduous teeth without successors, especially in the premaxilla, demonstrate, during life, resorption that is radiographically very similar to orthodontic apical root shortening. Does the apical zone contain cells that can be activated during orthodontic treatment and resorb the apex?

We gave 6 ideas here that, as far as we know, were never studied. Each one by itself (and for sure as a combination) can be **the reason** for the yet unsolved issue: What is really behind shortening of the roots during orthodontic treatment?

The current enduring paradigm that denies that there is activation of the cycle of the defense mechanism, (resorption and apposition) and puts most of its effort in studying the force (amount, direction etc.), implicating those findings as the cause of apical root shortening, actually places each one of us in danger of being sued in court for using, for example, too heavy forces that shortened a patient's roots. We all know that: a. nobody can really measure the actual force applied in all directions on the teeth during treatment, and b. this paradigm is derived from short term (mostly up to 1 month) in-vivo studies that, in none of them, as far as we know, ARS, that can be detected on X-rays, was demonstrated.

REFERENCES

1. Oppenheim A. Human tissue response to orthodontic intervention to short and long duration. Am J Orthod. 1942; 28:263–301.
2. Ozkalayci N, Karadeniz EI, Elekdag-Turk S, et al. Effect of continuous versus intermittent orthodontic force on root resorption: A microcomputed tomography study. Angle Orthod. 2018; 88:733–739.
3. Thomas S Kuhn. The Structure of Scientific Revolutions. University of Chicago press. Publisher, 1996.
4. Brezniak N, Wasserstein A. Orthodontically induced inflammatory root resorption. Part II: The clinical aspects. Angle Orthod. 2002;72:180–184.
5. Brezniak N and Wasserstein A. Orthodontic Root Resorption: A New Perspective. Angle Orthod. 2016; 66: 1056–1057.