



THE EFFECT OF DRUGS AND HORMONES ON ORTHODONTIC TOOTH MOVEMENT

Orthodontology

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ABSTRACT

This article aims at reviewing the role of various drugs, systemic factors and hormones in affecting the orthodontic tooth movement and their influence on the osteoclastic and osteoblastic activities. Orthodontic Tooth Movement occurs due to resorption of alveolar bone which is a result of osteoclastic activity. Various drugs and hormones can affect the mediators regulating the osteoclast affecting the orthodontic tooth movement. These drugs can either accelerate the rate of tooth movement or aid in its anchorage.

KEYWORDS

Drugs, Hormone, Orthodontics, Tooth movement, Accelerated Orthodontics, Anchorage

When an orthodontic force is applied it causes the tooth to move from its initial position in the alveolar bone to the desired position as a result of the bone remodelling process. The periodontal ligament plays an important role for the Orthodontic Tooth Movement (OTM). The cells of the periodontal ligament play an important role in this process. Bone remodelling occurs due to osteoblastic and osteoclastic activity which in turn is dependent on the histological changes that take place in the pressure and tension side of the applied force. On the pressure side bone osteoclastic activity is seen leading to resorption of bone while on the pressure side osteoblastic activity leads to bone deposition. This occurs due to inflammatory reaction and cytokine response in the form of Interleukin-1 alpha, Interleukin-1 beta, Tissue Necrosis Factor alpha and Interleukin-6.

Various drugs prescribed on a day-day basis affect this inflammatory reaction. Some can lead to limitation while others can accelerate the rate of tooth movement. Alongside these drugs are systematic factors in form of hormones tend to also affect the tooth movement.

This article helps clinician understand the role of various drugs and their influence on the tooth movement.

The drugs in this article are broadly classified in two categories –

- A. Drugs that limit or suppress Orthodontic Tooth Movement.
 - B. Drugs that accelerate or promote Orthodontic Tooth Movement.
- Suppressor drugs
 - Promoter drugs

SUPPRESSOR DRUGS	PROMOTER DRUGS
Nonsteroidal anti-inflammatory	Prostaglandin
Bisphosphonates	Leukotrienes
Osteoprotegerin	Vitamin D
	Corticosteroids

A.1. Non-Steroidal Anti Inflammatory Drugs:

Commonly used drugs for pain control are Non-Steroidal Anti Inflammatory Drugs also known as NSAIDs. They are known to have analgesic, anti pyretic and anti inflammatory effect^[1]. NSAIDs act by causing inhibition of prostaglandin synthesis^[2]. In 1977 Harrel and colleagues observed that osteoblast like cells that were cultured on orthodontic screws had synthesis of prostaglandins, cemented to the base of the petri dishes^[3]. In 2011 Knop and colleague suggested that due to the prostaglandin inhibition caused by NSAIDs the number of osteoclast like cells, Howship Lacunae and blood vessels reduced throughout the treatment^[4]. De Carlos et al, compared the effect of

selective COX-2 inhibitor and conventional NSAID on the orthodontic tooth movement in rats. They found that both the drugs inhibited the tooth movement but it was found to be partial in case of rofecoxib which is a selective COX-2 (cyclooxygenase-2) inhibitor compared to conventional NSAID in the form of diclofenac^[5,25].

Paracetamol also known as acetaminophen acts on COX-3 inhibitors expressed in the brain and spinal cord compared to NSAIDs which act on COX-1 and COX-2 inhibitors. It has minimal effect on prostaglandin synthesis^[1]. Paracetamol inhibits peripheral synthesis of prostaglandins but the degree and rate of tooth movement is not significantly different compared to the control group at the level of the PDL according to Kehoe^[6]. No effect on tooth movement by paracetamol on rabbits was reported by Roche JJ and colleagues in 1997^[7].

A.2. Bisphosphonates:

Bisphosphonates are used for the therapeutic treatment of diseases like Paget's Disease, Osteoporosis and diseases with excessive bone resorption. Bisphosphonates prevent the development of hematopoietic precursors into osteoclast. According to Zahrowski JJ^[8], bisphosphonates can inhibit the tooth movement and can also lead to increased risk of osteoradionecrosis in the maxilla and mandible. In 1994, Adachi and colleague conducted a study according to which topical administration of Risedronate which is a bisphosphonate in rats, it can be used in orthodontic anchorage and aid in retention^[9]. Liu et al, suggested that localized use of Clodronate reduces the root resorption and the number of osteoclast^[10].

A.3. Osteoprotegerin:

Osteoprotegerin is a glycoprotein that is involved in bone metabolism. It causes inhibition of the osteoclast differentiation and activation^[11]. According to Li Y and colleagues its local delivery adjacent to the anchorage tooth may be used to provide a novel pharmacological in reinforcing orthodontic anchorage^[12]. According to studies performed by Sydorak I et al, in which microsphere encapsulated OPG and non-encapsulated OPG were used in a rodent model of tooth movement to enhance anchorage it was found that a single injection of microsphere encapsulated 1 mg/kg OPG significantly inhibited mesial molar movement whereas a single injection of non-encapsulated 1 mg/kg OPG did not inhibit mesial molar movement. However the single injection of microsphere encapsulated 1 mg/kg OPG enhanced orthodontic anchorage without inhibiting the incisor movement. The study also showed that 5mg/kg of

non-encapsulated OPG every 3 days significantly inhibited mesial molar movement and enhanced the anchorage^[13].

B.1. Prostaglandins:

Prostaglandins are derived from arachidonic acid, belonging to the family of eicosanoids. They are responsible for various cellular and tissue activities as they are responsible for the formation of cyclic AMP. Due to it increasing the body temperature it causes inflammation and pain. It is also responsible for the contraction of the smooth muscles leading to the contraction of the blood vessels. According to Klein and Raisz^[14,23], the osteoclastic activity increases due to the increase in the number of osteoclast leading to more bone resorption.

In orthodontics the first to introduce prostaglandins to control the orthodontic tooth movement were Yamasaki and colleagues^[15], the orthodontic tooth movement became double when locally Prostaglandin E1 or E2 were administered in monkeys. In 1984 Yamasaki et al^[16], carried out clinical cases where in canine retraction was done. On the side that PGE1 injections were given the rate of canine retraction was 1.6 fold increased. There were no side effects observed both radiographically and macroscopically.

B.2. Leukotrienes:

Arachidonic acid is also a precursor for leukotrienes which belong to the family of eicosanoids. In diseases like asthma, allergies and inflammatory conditions leukotrienes have a tendency to increase. In 1989 Mohammed AH and colleagues^[17], discovered that leukotrienes were responsible for increased bone remodelling while the inhibitors of leukotriene receptors had the opposite effect. Thereby, pharmaceutical drugs such as montelukast and zafirlukast can lead to reduced orthodontic tooth movement.

B.3. Vitamin D:

1,25-dihydroxy cholecalciferol is the active metabolite of vitamin D and vitamin D plays an important role in maintenance of calcium in the body. Injection of 1,25-dihydroxy cholecalciferol into the PDL of intraligamentously was done in cats for canine retraction by Collins and Sinclair^[18] in 1988. After 21 days the teeth that had received the injections had moved 60% more than the control teeth. Well balanced bone turnover can also lead to enhanced tooth movement and this is induced by local application of vitamin D according to Kale and colleagues 2004^[19,24].

According to Noor Hasani and colleagues^[20], the canine retraction of patients who received 25pg/0.2 ml calcitriol diluted with 10% of dimethylsulfoxide(DMSO) the rate of tooth movement was 51% faster compared to the control side.

B.4. Corticosteroids:

Corticosteroids are drugs that are used in a wide range of medical conditions. They are known to affect the rate of tooth movement. When given in low doses of 1mg/kg of body weight it is seen that the orthodontic tooth movement is decreased as the osteoclastic activity gets suppressed and in high doses of 15mg/kg of body weight it is seen to increase and cause rapid tooth movement as the osteoclastic activity increases^[21].

In 2004 Kalia and colleagues^[22], had evaluated the effect of short and long term corticosteroid therapy on the orthodontic tooth movement in rats. The results of the study was found that in the group of acute administration of corticosteroids the rate of tooth movement had slowed down. Thereby, patients who are on short term administration of corticosteroids it is better to delay or postpone the orthodontic treatment. However, the study also shows that in case of chronic treatment the rate the tooth movement increased. Thus, in patients who are on long term corticosteroid therapy like chronic asthmatics the orthodontic treatment can be done with minimal adverse effects and with an expectation of faster tooth movement.

CONCLUSION:

Orthodontic tooth movement is greatly influenced by the action of these drugs. A better understanding between the commonly prescribed drugs and their effect on orthodontic tooth movement needs to be developed. If the clinician develops a sound knowledge of drugs, they can use them to their advantage of achieving a rapid tooth movement or gaining a superior anchorage control. It is therefore important to know the medical history and what medication is the patient taking during their orthodontic treatment as it can influence the duration time of the

treatment. A prudent clinician should have the knowledge of pharmacology to know how to divert his patients to achieve a quicker, smoother and less experience of Orthodontics and a thorough skillset to achieve the same.

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