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The effect of vibratory stimulus oninterleukin 1ß levels and its correlation with the rate of tooth movement during retraction- A split mouth study

Piush Kumar[1], Shreya Agrawal[2], Payal Sharma[1]*, Shubhangi Jain[1], Divya Shetty[1], Bhupender Singh[1]

[1]Dept. of I.T.S Centre for Dental Studies and Research, Ghaziabad, Uttar Pradesh, India
[2]Private Practitioner, India

Abstract

Aim and Objectives: Prolonged orthodontic treatment duration is a major concern in clinical practice. Techniques aimed at accelerating tooth movement include both biochemical and physical stimulation methods. This study evaluated the effect of vibratory stimulus on Interleukin-1β (IL-1β) levels in gingival crevicular fluid (GCF) and its correlation with the rate of canine retraction.

Materials and Methods: A split-mouth clinical study was conducted on 10 patients undergoing maxillary first premolar extractions. Canine retraction was performed using NiTi coil springs on both sides; one side received additional vibratory stimulus via electric toothbrush use (8 minutes twice daily for 8 weeks). GCF samples were collected at 0 (T0), 24 hours (T1), 4 weeks (T2), and 8 weeks (T3). IL-1β levels were analyzed using ELISA. Tooth movement was measured using digital calipers, and pain perception was assessed using a visual analog scale (VAS). Statistical analysis was conducted using Wilcoxon signed-rank test, paired t-test, and Pearson's correlation.

Results: Both groups showed a significant increase in IL-1β levels at 24 hours, followed by a gradual decline toward baseline by 8 weeks. There was no statistically significant difference in the rate of tooth movement or IL-1β levels between the vibratory and control groups. However, patients in the vibratory group reported significantly lower VAS pain scores.

Conclusion: Vibratory stimulus does not enhance the rate of tooth movement or sustain elevated IL-1β levels but is effective in reducing orthodontic pain. No correlation was found between IL-1β levels and rate of tooth movement.

Keywords: Orthodontic tooth movement, Interleukin-1beta, Orthodontic appliances, Fixed.

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1. Introduction

Orthodontic treatment is widely sought across the globe and is practiced in nearly every nation, irrespective of development status. One of the significant challenges in orthodontics remains the prolonged duration of treatment. Consequently, much of the innovation in the field is focused on strategies to shorten treatment time.[1] A deeper understanding of the biological mechanisms governing tooth movement is essential for designing interventions that can enhance this process. Based on insights into these biological processes, two principal strategies have emerged to accelerate tooth movement: either directly activating the target cells through physical, chemical, or artificial methods to boost their numbers and activity, or indirectly prompting the body to recruit and activate more of these target cells.[2]

When orthodontic forces are applied, periodontal ligament cells—including fibroblasts and osteoblasts—release signaling compounds such as neurotransmitters, arachidonic acid derivatives, and cytokines. These molecules play a key role in initiating the biological events responsible for bone remodeling, particularly resorption and formation of alveolar bone.[3,4] Such mediators are secreted at various phases of tooth movement and can be sampled through gingival crevicular fluid (GCF).[5]

*Corresponding author: Payal Sharma
Email: shubhangi.jain.92@gmail.com

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The significance of these mediators in controlling the pace of tooth movement is highlighted in studies where their inhibition led to a slower rate of movement.[2] On the other hand, increasing their activity—for instance, through the injection of prostaglandins (PGs) into the periodontal ligament in animal models—has been shown to enhance osteoclast activity and tooth movement.[6]

Another strategy to upregulate these mediators involves stimulating the body to naturally produce them in higher quantities.[2] This can be accomplished either through localized surgical interventions near the extraction site—like corticotomies or micro-osteoperforations—or by employing device-assisted methods. These include the application of direct electrical currents, pulsed electromagnetic fields, static magnetic fields, vibrational stimulation, or low-level laser therapy.[1]

Nishimura et al.[7] reported that vibratory stimulation enhanced the speed of tooth movement in rats without harming periodontal tissues. Their findings also indicated activation of the RANK/RANKL pathway, which is essential for osteoclast development, in response to vibrational forces.

Research by Grieve et al.,[8] Lee et al.,[9] and Ren et al.[10] demonstrated a marked increase in IL-1β levels within the periodontium during orthodontic movement, with these changes detectable in GCF samples. Furthermore, Leethanakul et al.[11] found that IL-1β production was amplified when vibratory forces were applied in conjunction with orthodontic forces.

Despite these findings, limited data exist on how different bone remodeling mediators respond to the combination of vibratory and orthodontic stimuli. Such investigations could provide deeper insight into the role of vibrational therapy in orthodontics. Assessing GCF levels is a minimally invasive yet effective method to study fluctuations in inflammatory markers, particularly in in-vivo experiments.[11] Therefore, the aim of the present clinical research was to assess how vibrational stimulation influences IL-1β concentrations and whether these changes are associated with the rate of tooth movement during the retraction phase of orthodontic treatment.

Accordingly, the null hypothesis stated that applying vibratory stimulation during orthodontic retraction does not significantly affect IL-1β levels in gingival crevicular fluid

2. Materials and Methods

2.1. Clinical and periodontal evaluation of subjects before case selection

Subjects were selected for each group after a brief case history. A sterile mouth mirror and William's graduated periodontal probe was used to clinically examine the periodontal status. For each subject, the plaque index,[12] gingival index[13] and probing depth[14] scores were recorded. The same investigator collected all clinical data. Fixed appliance with MBT (0.022"x0.028") prescription metal brackets was placed in all the patients subjects qualifying the selection criteria, after taking informed consent. Ethical approval for the study was taken from the institution`s ethical committee. Initial phase of leveling and alignment was completed and routine archwire sequence was followed till working archwire of 0.018" stainless steel.

2.2. Experimental design

Sample size calculation was done from a previous study by Leethanakul et al wherein for an effect size of 0.85a sample of 10 patients (20 sites), would provide an adequate statistical power of 95% to detect a significant difference. was determined that a sample of 10 patients would be sufficient to The study was conducted in 10 orthodontic patients requiring fixed orthodontic treatment requiring maxillary 1st premolar extraction and distal retraction of the canines. Patients with good general health, no relevant medical history, no antimicrobial therapy within previous 6 months, healthy periodontal tissues and gingival index and plaque index showing mild score were included in the study.

In this split-mouth design, canine retraction was done by NiTi closed coil spring with a force of 150 gm on both sides of the arch. The NiTi spring was placed between canine and molar, and the force level was measured using Correx gauge.

On one side of the arch (randomly selected by coin toss) the patients were asked to use a vibratory tooth brush for added stimulation for a minimum of 8 minutes twice a day for 2 months. The allocation was done and noted by third individual who was not involved in the study as the clinician or an assessor. The allocation was concealed from the clinician and the assessor. The patients were instructed not to clean their teeth with the electric toothbrush.

The patients were allowed to take anti-inflammatory medicine, if required, but in such cases, the patients were no longer to be included in the study.

From each test group, GCF samples were collected at four different time intervals so as to obtain a total of 80 samples (40 for control and 40 for experimental side). GCF was collected from the distobuccal gingival sulcus of maxillary canine of both sides at various time intervals i.e., 0 day (T_0), 24 hours (T_1) and 4 weeks (T_2) and 8 weeks (T_3) after application of force.(**Table 1,2**). The NiTi coil spring was reactivated at 4 weeks and 8 weeks before collection of the GCF samples.

For each test group, the amount of extraction space was measured on models of three different time intervals, i.e., at 0 day (T_0),4 weeks(T_2)and 8 weeks (T_3) to calculate the amount of retraction. The measurements were repeated after 1 week to determine the intra-examiner reliability.

2.3. For collection of gingival crevicular fluid

1 µl GCF was collected from the distobuccal gingival sulcus of maxillary canines bilaterally with white color-coded 1-5 µl calibrated volumetric micro capillary pipette. All GCF samples were collected in the morning hours during the study period to avoid any diurnal variation. Supragingival plaque, if present, was removed from these teeth for sampling. The teeth were gently dried with an air spray and isolated with cotton rolls. Saliva ejector was used to avoid salivary contamination. The GCF collected was immediately transferred to Eppendrof tube and was stored at -20^{0}C till the time of the assay for IL 1β analysis. ELISA was used for analysis of the samples and the values obtained were tabulated. All data was subjected for statistical analysis.

2.4. For measuring the rate of tooth movement

For the measurement of the rate of tooth movement, the extraction space present distal to canine was measured using Digital Caliper with an accuracy of 0.01 mm. The measurement of the space was done at three time intervals, i.e., at 0 days (T_0), 4 weeks (T_2) and 8 weeks (T_3). The tabulated values were subjected to statistical analysis.

Any patient who did not have the required plaque index and gingival index at the end of the study was excluded from the data.

2.5. For assessment of pain

The participants were asked to assess their level of discomfort 12 hours after beginning canine retraction, for both control and test sides on different 100-mm visual analog scales. They were also asked to do the same 12 hours after every reactivation i.e. at 4 weeks and 8 weeks.

2.6. Statistical analysis

The data was subjected to statistical analysis using SPSS (Statistical Package for Social Sciences) version 20.0 statistical analysis software. The descriptive statistics including the mean, standard deviation, minimum and maximum values were calculated for each of the variables in both groups. Wilcoxon signed rank test was used to compare the rate of canine retraction between vibratory stimulation assisted and conventional mechanics group. Wilcoxon signed rank test was also used to compare the VAS scores at different time intervals, and between the groups. The inter-observer and intra-observer reliability was compared using paired t test and Dahlberg error measurement. Karl Pearson's correlation formula was used to find the correlation between IL-1β and the rate of tooth movement. The significance was set a $p<0.05$.

3. Results

The results revealed no significant difference was seen in the gingival status (gingival index, periodontal index and probing depth) at the beginning and end of study period, also between test and control sides. Both Paired t test and Dahlberg's formula indicated for a good inter-observer and intra-observer reliability for space measurement on the study models.

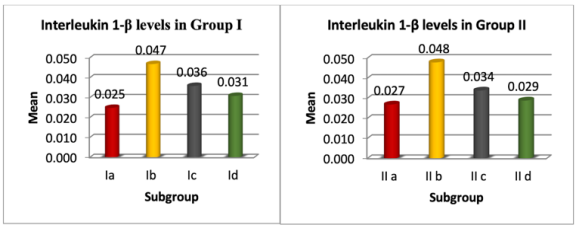

Figure 1: Comparison of remaining extraction space in Group I and Group II at different time intervals.

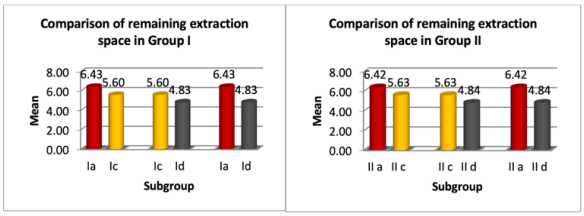

Figure 2: IL-1β levels in Group I and II at different time intervals.

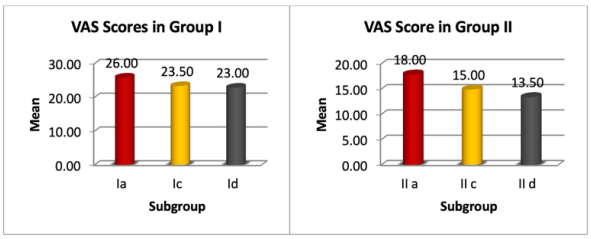

Figure 3: VAS scores in Group I and Group II at different time intervals.

Table 1 shows the Comparison of remaining extraction space in the Groups at different time intervals in group 1 and 2. In group 1 there was a consistent reduction in remaining extraction space from 6.43 ± 0.18 mm to 4.83±0.21 mm with a statistically significant difference between all the time intervals on pairwise comparison($p<0.05$). A similar trend was observed in group 2 wherein reduction in remaining extraction space was observed from 6.42 ± 0.20 mm reducing to 4.84±0.21 with a statistically significant difference ($p<0.05$). A Comparison of remaining extraction space in Group I and Group II at different time intervals revealed no statistically significant difference between the 2 groups at any of the 3 timepoints(a, p=0.739)(c, p=0.564)(d, p=0.902). (Table 2). Comparison of rate of tooth movement in Group I and Group II at different time intervals [T_0-T_2, p=0.206], [T_2-T_3, p=0.317], [T_0-T_3, p=0.608] also revealed no statistically significant difference between the two groups.(Table 3)

Table 4 shows the Interleukin 1-β levels in Group I (control) at different time intervals showed the highest levels at T1 (0.047 ± 0.009 pg/µl) followed by T2, T3 and the least at T0 (0.025 ± 0.005 pg/µl). There was a statistically significant difference observed between the time points. [p=0.005] [T0,T3;p=0.021] [T0,T2; p=0.024] Similar trend

was observed for group II. Highest levels for T1 (0.048 ± 0.011 pg/µl) followed by T2, T3 and least for T0 (0.027 ± 0.005 pg/µl). There was a statistically significant difference also observed for group II at different timepoints (p=0.005) except for the comparison of T0 and T3(p=0.483). Table 5 shows the comparison of IL-1β in Group I and Group II at different time intervals. Group II showed a greater Interleukin 1-β levels than group I at T0 and T1 with a statistically significant difference at T0 timepoint (p=0.046). At T2 and T3 group I showed greater interleukin levels than group II with no statistically significant different at any of the two timepoints. The VAS scores were higher for group I for all timepoints. The scores showed a reduction from T0 to T3 in both group 1 and II.(Table 6) There was no statistically significant difference between the VAS scores for different timepoints for group I, However there was a significant difference between T0 and T3 for group II. (p=0.024). There was a statistically significant greater VAS score for group I compared to group II for at all timepoints.(Table 7) (a, p=0.016;b, p=0.004;c, p=0.002) **Table 8** shows the correlation between Interleukin 1-β levels and rate of tooth movement in Group I and II. No significant correlation was found between Interleukin 1- β levels and rate of tooth movement in Group I or Group II.

Table 1: Comparison of remaining extraction space in the Groups at different time intervals.

Sub group	Mean ± SD	Mean±SD difference	P value (Wilcoxon signed rank test)
Ia	6.43 ± 0.18	0.83±0.01	0.004*
Ic	5.60 ± 0.17		
Ic	5.60 ± 0.17	0.77±0.04	0.004*
Id	4.83 ± 0.21		
Ia	6.43 ± 0.18	1.60±0.03	0.005*
Id	4.83 ± 0.21		
IIa	6.42±0.20	0.79±0.00	0.004*
IIc	5.63±0.20		
IIc	5.63±0.20	0.79±0.01	0.002*
IId	4.84±0.21		
IIa	6.42±0.20	1.58±0.01	0.004*
IId	4.84±0.21		

P>0.05, non-significant; P<0.05*, significant; P<0.001** highly significant

Table 2: Comparison of remaining extraction space in Group I and Group II at different time intervals.

Time interval	Group I	Group II	Mean±SD difference	P value (Wilcoxon signed rank test)
	Mean ± SD	Mean ± SD		
a	6.43 ± 0.18	6.42 ± 0.20	0.01±0.02	0.739
c	5.60 ± 0.17	5.63 ± 0.20	0.03±0.03	0.564
d	4.83 ± 0.21	4.84 ± 0.21	0.01±0.00	0.902

P>0.05, non-significant; P<0.05* significant; P<0.001**, highly significant

Table 3: Comparison of rate of tooth movement in Group I and Group II at different time intervals.

Time interval	Group I	Group II	P value (Wilcoxon signed rank test)
	Mean±SD	Mean±SD	
Subgroup(a-c)	0.83±0.01	0.79±0.00	0.206
Subgroup(c-d)	0.77±0.04	0.79±0.01	0.317
Subgroup(a-d)	1.60±0.03	1.58±0.01	0.608

P>0.05, non-significant; P<0.05*, significant; P<0.001**, highly significant

Table 4: Comparison of IL-1β in Group I at different time intervals.

Sub group	Mean±SD	Mean±SD difference	P value (Wilcoxon signed rank test)
Ia(T0)	0.025 ± 0.005	0.022±0.004	0.005*
Ib(T1)	0.047 ± 0.009		
Ia(T0)	0.025 ± 0.005	0.011±0.001	0.005*
Ic(T2)	0.036 ± 0.004		
Ia(T0)	0.025 ± 0.005	0.006±0.001	0.021*
Id(T3)	0.031 ± 0.004		
Ib(T1)	0.047 ± 0.009	0.011±0.005	0.005*
Ic(T2)	0.036 ± 0.004		
Ib(T1)	0.047 ± 0.009	0.016±0.005	0.005*
Id(T3)	0.031 ± 0.004		
Ic(T2)	0.036 ± 0.004	0.005±0.000	0.005*

Id(T3)	0.031 ± 0.004		
IIa(T0)	0.027 ± 0.005	0.021±0.006	0.005*
IIb(T1)	0.048 ± 0.011		
IIa(T0)	0.027 ± 0.005	0.007±0.000	0.024*
IIc(T2)	0.034 ± 0.005		
IIa(T0)	0.027 ± 0.005	0.002±0.000	0.483
IId(T3)	0.029 ± 0.005		
IIb(T1)	0.048 ± 0.011	0.014±0.006	0.005*
IIc(T2)	0.034 ± 0.005		
IIb(T1)	0.048 ± 0.011	0.019±0.006	0.005*
IId(T3)	0.029 ± 0.005		
IIc(T2)	0.034 ± 0.005	0.005±0.000	0.005*
IId(T3)	0.029 ± 0.005		

P>0.05, non-significant; P<0.05*, significant; P<0.001**, highly significant

Table 5: Comparison of IL-1β in Group I and Group II at different time intervals.

Time interval	Group I	Group II	Mean±SD difference	P value (Wilcoxon signed rank test)
	Mean ± SD	Mean ± SD		
a	0.025 ± 0.005	0.027 ± 0.005	0.002±0.01	0.046*
b	0.047 ± 0.009	0.048 ± 0.011	0.001±0.002	0.414
c	0.036 ± 0.004	0.034 ± 0.005	0.002±0.001	0.313
d	0.031 ± 0.004	0.029 ± 0.005	0.002±0.001	0.182

P>0.05, non-significant; P<0.05*, significant; P<0.001**, highly significant

Table 6: Comparison of VAS scores in Group I and Group II at different time intervals.

Sub groups	Mean ± SD	Mean±SD difference	P value (Wilcoxon signed rank test)
Ia	26.00 ±4.59	2.50±0.15	0.132
Ic	23.50 ± 4.74		
Ic	23.50 ± 4.74	0.50±0.09	0.564
Id	23.00 ± 4.83		
Ia	26.00 ± 4.59	3.00±0.24	0.058
Id	23.00 ± 4.83		
IIa	18.00 ± 4.83	3.00±0.75	0.119
IIc	15.00 ± 4.08		
IIc	15.00 ± 4.08	1.50±0.03	0.317
IId	13.50 ± 4.12		
IIa	18.00 ± 4.83	4.50±0.71	0.024*
IId	13.50 ± 4.12		

P>0.05, non-significant; P<0.05*, significant; P<0.001**, highly significant

Table 7: Comparison of VAS scores between Group I and Group II at different time intervals.

Time interval	Group I	Group II	Mean±SD difference	P value (Wilcoxon signed rank test)
	Mean ± SD	Mean ± SD		
a	26.00 ± 4.59	18.00 ± 4.83	8.00±0.24	0.016*
c	23.50 ± 4.74	15.00 ± 4.08	8.50±0.66	0.004*
d	23.00 ± 4.83	13.50 ± 4.12	9.50±0.71	0.002*

P>0.05, non-significant; P<0.05*, significant; P<0.001**, highly significant

Table 8: Correlation between IL-1β and rate of tooth movement in Group I.

Correlation between IL-1β & rate of tooth movement	Correlation coefficient (Karl Pearson's correlation formula)	P value
Ia	0.270	0.451
Ic	0.584	0.076
Id	0.624	0.054
IIa	0.369	0.294
IIc	0.001	0.998
IId	0.329	0.353

P>0.05, non-significant; P<0.05*, significant; P<0.001**, highly significant

4. Discussion

Orthodontic tooth movement is governed by a dynamic interplay of molecular proteins that coordinate bone remodeling. One of the main drawbacks in orthodontic treatment is the extended duration required, which has prompted ongoing exploration into methods that can speed up tooth movement.[15]

Several studies have examined the effect of vibrational forces used in conjunction with fixed appliances, but their findings have been inconsistent. While researchers like Nishimura[7] and Pavlin[17] observed an increase in the rate of movement due to vibratory input, others such as Dibiase[18] and Katchooi[19] reported no significant benefits. These discrepancies may stem from variables such as vibration frequency, application time, the device used, whether the research was conducted on humans or animals, and the subjects' age.

This study utilized an electric toothbrush to provide vibratory stimulation during canine retraction, owing to its affordability, ease of use, and high patient acceptance. The participants were young adults undergoing individual canine retraction.

According to published data, orthodontic forces can cause notable changes in the composition of gingival crevicular fluid (GCF), particularly in markers linked to inflammation, bone remodeling, and tissue breakdown.[20] Kapoor et al.,[21] in a systematic review, concluded that cytokine levels in GCF typically peak around 24 hours post-force application and gradually return to baseline. Mechanical compression applied to osteoblasts upregulates IL-1β and IL-6, both of which are pro-inflammatory cytokines involved in osteoclast formation and activity.[22]

Sakamoto et al.[16] found that in rats, vibratory forces significantly enhanced RANKL expression and activated NF-κB translocation in osteocytes located on the compression side of alveolar bone during orthodontic movement. Thus, this study aimed to stimulate an early cellular response in the periodontium through vibration.[7] Kapoor et al. have emphasized IL-1 as a crucial factor in modulating tooth movement, and researchers like Nunes[5] and Saito[25] have similarly documented increased IL-1β levels during orthodontic activity.

Consequently, this study measured IL-1β concentrations in GCF during the application of vibratory stimuli. Due to the short durations of earlier studies, long-term molecular responses remain unclear; hence, the current study tracked both IL-1β levels and tooth movement over two months.

Previous researchers, including Drummon[20] and Kapoor,[21] have proposed using GCF as a diagnostic tool to monitor biochemical changes associated with orthodontic movement. In addition, some investigations have indicated that vibration may alter pain perception during treatment.[21,24] Therefore, a secondary aim of this study was to assess how vibration affects patient discomfort during retraction.

Results showed that the space distal to the canines decreased steadily in both test and control groups. No significant difference in retraction was found between the groups at various time intervals, indicating a consistent rate of space closure. The average retraction rate ranged from 0.77±0.04 mm to 0.83±0.01 mm per month—within the typical 0.5–1.5 mm/month range in conventional treatments.

When comparing sides, the rate of tooth movement was similar with or without vibratory stimulation, aligning with findings from DiBiase,[25] Katchooi,[19] and Woodhouse.[26] On the other hand, Nishimura[7] and Pavlin[17] reported increased movement with vibration. However, Nishimura used a custom device in rats, and Pavlin used a 30 Hz Acceladent device, which differs from the present study's method. Miles[27] also found no improvement in movement rate with Acceladent.

Further analysis of IL-1β levels revealed a sharp increase at 24 hours in both groups compared to baseline, consistent with findings from Iwasaki,[28] Nunes,[5] and Kapoor.[21] The levels decreased by week 4 and returned near baseline by week 8. Grieve et al.[8] and Lee et al.[9] noted similar trends: IL-1β spiking early and declining unless reactivation occurs, potentially explaining the slightly elevated levels seen at week 4.

Interestingly, baseline IL-1β levels were higher on the test side than on the control, possibly due to vibratory stimulation an hour before GCF sampling. However, no significant differences were noted between the groups at 24 hours, 4 weeks, or 8 weeks. This suggests that while vibration may cause an early spike, its long-term impact on IL-1β levels is minimal. These findings are consistent with Ren,[10] who noted that IL-1β surges initially after force application but does not influence sustained movement. Leethanakul[11] found IL-1β increased with vibration on the pressure side but not the tension side—our results may differ due to shorter (8-minute) stimulation versus their 15-minute protocol.

Pain is a common side effect of orthodontic therapy.[29] Patient pain was measured using the VAS scale. While the control side showed no major variation over time, the test side revealed a significant drop in pain scores from T0 to T3. Overall, test group scores were consistently lower, an encouraging result considering Miles[30] found no benefit of Acceladent in pain reduction. In contrast, Celebi[29] observed that vibration could indeed lower orthodontic pain.

Pain mechanisms in orthodontics are thought to involve hyperalgesia and release of pain-related compounds such as Substance P, Histamine, Enkephalins, Dopamine, Serotonin, Glycine, Prostaglandins, Leukotrienes, and cytokines.[31] These substances contribute to the pain experienced during

tooth movement. It is proposed that vibration may relieve pressure in the periodontium, enhancing circulation and clearing inflammatory by-products. Another possible explanation for reduced discomfort lies in the "gate control theory," where non-painful stimuli inhibit pain signals through alternate nerve pathways.[32]

To summarize, more research is needed to fully evaluate the efficiency of vibratory stimulation when combined with orthodontic forces for expedited movement. Future studies should explore optimal vibration frequencies and durations, use larger participant pools, and assess longer-term outcomes. Special attention should also be paid to patient comfort and pain reduction—key factors in treatment compliance and satisfaction.

5. Conclusions

1. Vibratory stimulus does not affect IL-1β levels in GCF. It just helps in early release of the cytokine.
2. No correlation exists between the rate of canine retraction and IL-1β levels in GCF, with or without application of vibratory stimulus.
3. The vibratory stimulus may reduce the amount of pain during orthodontic retraction, improving the patient acceptance of the appliance and the treatment protocols. Further studies can shed more light on the usefulness of vibratory stimulus and it's role in reducing orthodontic treatment induced pain.

6. Source of Funding

None.

7. Conflict of Interest

None.

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