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Orthodontic Tooth Movement during Pregnancy: Biological Mechanisms, Periodontal Alterations, and Clinical Considerations — A Narrative Review

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ABSTRACT:

Aim: To evaluate the biological mechanisms, periodontal alterations, and clinical management strategies associated with orthodontic tooth movement (OTM) during pregnancy through a structured narrative review.

Background: Pregnancy induces systemic and hormonal alterations, primarily rising levels of estrogen and progesterone and an early peak of relaxin, that may influence bone metabolism, periodontal ligament remodeling, and the vascular response to orthodontic forces. Understanding these adaptations is essential for managing pregnant patients safely without compromising maternal-fetal health or treatment outcomes.

Review Results: No study of OTM in pregnant women was found among the sources cited. Evidence from animal, in-vitro and non-pregnant human studies suggests that gestational hormones may alter periodontal ligament turnover and bone remodeling, with estrogen possibly slowing and relaxin possibly facilitating tooth movement; the net effect in humans is unknown. Progesterone and plaque together heighten gingival inflammation, so pregnancy gingivitis is common and fixed appliances can amplify it. Elective imaging and surgery are generally deferred, particularly in the first trimester, and NSAIDs are avoided. When orthodontic intervention is required, expert opinion supports light forces, strict plaque control, and close interdisciplinary monitoring.

Conclusion: Orthodontic treatment during pregnancy requires a cautious, modified clinical approach. Current recommendations rest mainly on biological reasoning and expert opinion rather than on clinical trials, and prospective human studies are needed

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1. INTRODUCTION

Orthodontic tooth movement (OTM) is a biomechanical process governed by a coordinated inflammatory response within the periodontium, leading to localized extracellular matrix turnover and bone remodeling [1,2]. Applying controlled mechanical forces to a tooth triggers a complex cascade of cellular events involving osteoclast-mediated bone resorption on the pressure side and osteoblast-mediated bone apposition on the tension side [1,3]. While this biological cascade is strictly regulated under normal physiological conditions, it is significantly altered by systemic factors, including metabolic status and endocrine fluctuations [3,4].

During pregnancy, the female body undergoes profound systemic, physiological, and hormonal adaptations designed to support fetal development [5,6]. Circulating estrogen and progesterone rise progressively to term, whereas relaxin peaks in the first trimester (about 8–12 weeks) and then falls to a lower plateau [5,7]. These hormones act on distant target tissues, including the oral mucosa, periodontal ligament (PDL), and alveolar bone matrix [6,8]. Table 1 summarises the physiological changes of pregnancy that matter for orthodontic care.

The periodontal tissues are sensitive to gestational hormonal surges because functional estrogen and progesterone receptors are present in human gingival fibroblasts, osteoblasts, and PDL cells [6,8]. Elevated progesterone increases vascular permeability and microvascular responsiveness within the periodontium, heightening susceptibility to local irritants and predisposing pregnant patients to pregnancy gingivitis and, less commonly, pyogenic granuloma [8,9]. Simultaneously, systemic changes in bone turnover, involving estrogen, parathyroid hormone-related protein (PTHrP), calcitriol, and relaxin, may influence the velocity, predictability, and safety of OTM [7,10].

With an increasing number of adult females seeking orthodontic care, practitioners frequently encounter patients who either become pregnant during ongoing treatment or seek orthodontic consultation while pregnant [11]. Managing these cases requires balancing mechanical force delivery, maternal-fetal safety, diagnostic imaging restrictions, and elevated periodontal risk [9,13]. Although numerous studies have explored individual aspects of pregnancy-induced periodontal change, a synthesis integrating biological mechanisms with practical clinical management during orthodontic therapy is lacking [4,12]. To our knowledge, no clinical study has measured OTM in pregnant women, so clinical guidance is necessarily extrapolated from other evidence.

Table 1. Physiological changes of pregnancy relevant to orthodontic care

System	Change in pregnancy	Orthodontic and oral implication	Practical action
Endocrine	Estrogen and progesterone rise to term; hCG and relaxin peak at about 8–12 weeks	Gingival vascular and inflammatory change; possible PDL softening	Intensify plaque control from the first visit
Blood volume and heart	Plasma volume up roughly 40–50% and cardiac output up 30–50% by the third trimester	Oedematous, bleeding-prone gingiva	Gentle scaling; short visits
Posture and circulation	From about 20 weeks the uterus can compress the inferior vena cava	Supine hypotension; dizziness on sitting up	Left-lateral tilt or wedge; sit up slowly
Gastrointestinal	Nausea and vomiting (mostly first trimester); reflux	Gagging, enamel erosion, caries risk	Morning appointments; intraoral scanner; water or bicarbonate rinse, then wait before brushing
Glucose metabolism	Insulin resistance rises to the third trimester; gestational diabetes in a minority	More gingival inflammation, slower healing	Ask about glycaemic status; tighter recall

Calcium and bone	Calcium absorption and calcitriol rise; bone turnover increases; small reversible bone loss, larger in lactation	Plausible effect on remodeling; no OTM data	Do not assume faster movement; do not escalate force
Immune system	Shift in maternal immune response	Exaggerated gingival reaction to plaque	Control biofilm
Psychosocial	Fatigue, anxiety, time pressure	Missed visits, poorer hygiene	Short, scheduled recalls

Therefore, this narrative review aims to evaluate the biological mechanisms, systemic hormonal influences, and periodontal modifications associated with OTM during pregnancy, and to summarise clinical recommendations, graded by the strength of their supporting evidence, for orthodontic management.

MATERIALS AND METHODS

Protocol and Guidelines

This review was designed as a structured narrative review. Reporting follows the PRISMA-ScR checklist where applicable, and review quality was self-assessed with the Scale for the Quality Assessment of Narrative Review Articles (SANRA).

Information Sources and Search Strategy

An electronic search was performed in PubMed/MEDLINE, Scopus, and the Cochrane Library from database inception to [date of last search]. [Add Embase or Web of Science.]

Three domain-specific searches were run (Table 2): (A) orthodontic tooth movement and pregnancy; (B) gestational hormones and periodontal ligament or bone; and (C) periodontal changes in pregnancy. Medical Subject Headings (MeSH) and free-text terms were combined with Boolean operators. The core strings were:

- A: ("Tooth Movement Techniques"[MeSH] OR "orthodontic tooth movement" OR orthodont*) AND ("Pregnancy"[MeSH] OR pregnan* OR gestation*)
- B: (relaxin OR estrogen* OR estradiol OR progesterone) AND ("Periodontal Ligament"[MeSH] OR "orthodontic tooth movement" OR "alveolar bone")
- C: (pregnan* OR gestation*) AND (gingivitis OR "Periodontal Diseases"[MeSH] OR "pyogenic granuloma")

Reference lists of eligible articles and relevant orthodontic journals were also hand-searched. Records identified, screened, excluded, and included are shown in Figure 1 [PRISMA-ScR flow diagram to be inserted].

Table 2. Search strategy by database

Database	Domain	Core string
PubMed/MEDLINE	A. OTM and pregnancy	String A
PubMed/MEDLINE	B. Hormones, PDL and bone	String B
PubMed/MEDLINE	C. Periodontium in pregnancy	String C
Scopus	A–C	TITLE-ABS-KEY equivalents of strings A–C
Cochrane Library	A–C	Equivalent terms (trials and reviews)

Eligibility Criteria

Articles were screened based on predefined inclusion and exclusion criteria:

- **Inclusion Criteria:**

1. Primary research (clinical trials, observational studies, cohort and case-control studies, case reports and series, and animal or in-vitro studies) and reviews investigating orthodontic tooth movement during pregnancy or under gestational hormone exposure.
2. Studies analyzing the cellular and biomolecular effects of pregnancy-related hormones (estrogen, progesterone, relaxin) on human or animal periodontal tissues and alveolar bone remodeling.
3. Clinical reports and expert consensus guidelines addressing diagnostic imaging, pharmacological safety, force application, or periodontal management in pregnant orthodontic patients.
4. Articles published in peer-reviewed journals in the English language.

- **Exclusion Criteria:**

1. Studies focusing exclusively on non-pregnant systemic conditions or non-orthodontic dental interventions.
2. Conference abstracts, editorials, letters to the editor, and unpublished grey literature other than formal professional-society guidelines.
3. Articles lacking full-text access or clear methodological parameters.

Study Selection and Data Extraction

Two independent reviewers (S.S. and S.S.K.) screened titles and abstracts retrieved from the initial database searches. Full-text copies of potentially relevant studies were retrieved and assessed independently against the eligibility criteria. Disagreements regarding inclusion were resolved through consultation and consensus with a third reviewer (A.H.).

Data were extracted systematically into a standardized data collection form capturing:

- Author(s) and year of publication
- Study design and sample characteristics
- Hormonal or biological variables evaluated
- Key findings regarding periodontal response, rate of tooth movement, and bone metabolism
- Clinical recommendations for orthodontic management during gestation, and the type of evidence supporting each (human pregnancy, human non-pregnant, animal, in vitro, or expert opinion)

Data Synthesis

Due to the heterogeneous nature of the retrieved literature (encompassing preclinical biological studies, observational clinical investigations, and narrative guidance), a narrative thematic synthesis was performed. No formal risk-of-bias assessment was undertaken; each finding is instead labelled by evidence type (Table 3). Findings were categorized into three primary analytical domains:

1. Biological and biomolecular mechanisms of OTM under gestational hormonal influences.
2. Periodontal alterations and inflammatory dynamics during pregnancy.
3. Clinical protocols, pharmacological considerations, and maternal-fetal safety guidelines during orthodontic therapy.

REVIEW RESULTS

Records were identified, were screened, and were included (Figure 1). No study measuring OTM in pregnant women was identified. The findings below therefore combine animal, in-vitro, non-pregnant human, and pregnancy-related periodontal evidence (Table 3), and are organized into three domains.

Table 3. Evidence map: type and level of the sources underlying the review

Source	Type	Pregnancy-specific?	What it supports	Evidence level
Krishnan and Davidovitch [1]; Wise and King [2]; Meikle [3]	Reviews of OTM biology	No	Mechanism of force-induced remodeling	Background
Mascarenhas [6]; Mariotti [8]; Markou [10]	Reviews of sex hormones and periodontium	Partly	Estrogen and progesterone effects on gingiva and bone	Indirect
Nicoara [7]	Narrative review, hormones and OTM	Partly	Hormonal modulation of OTM	Secondary
Madan et al. [16]	Rat experiment	No	Relaxin and OTM	Animal
Gürsoy et al. [12]; Löe and Silness [18]	Clinical studies	Yes	Gingival change in pregnancy	Human, observational
Silk [5]; ACOG [13,15]; FDA [14]	Overview, committee opinions, regulatory communication	Yes	Dental-care safety, imaging, drugs	Expert opinion / guideline
Studies of OTM in pregnant humans	None identified	—	—	Evidence gap

1. Hormonal Modulation of Bone Remodeling and OTM

- **Estrogen:** High physiological levels of estrogen are thought to exert a bone-sparing effect by inhibiting osteoclastogenesis and downregulating pro-inflammatory cytokines such as interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) [6,8]. This may transiently slow osteoclast-mediated resorption on the compression side of the PDL, but this has been shown in animal and in-vitro models, not in pregnant humans [3,7].
- **Progesterone:** Progesterone increases gingival microvascular permeability and prostaglandin E2 production and may reduce collagen synthesis within the PDL, which can increase tooth mobility independent of true structural bone loss [6,8,9]. Its effect on the rate of OTM is unclear.
- **Relaxin:** Relaxin peaks in the first trimester. In animal and cell models it upregulates matrix metalloproteinases and reduces PDL collagen, lowering PDL stiffness; in rats this has been associated with faster tooth movement and reduced anchorage stability [7,16]. Human periodontal data are lacking, and its role in pelvic ligament laxity in humans remains debated.
- **Calcium and bone metabolism:** Pregnancy also raises intestinal calcium absorption and calcitriol and increases bone turnover, with a small reversible loss of bone that is larger during lactation [17]. Whether this affects OTM has not been studied.
- **Net effect:** The competing actions of estrogen (possibly slowing), relaxin (possibly accelerating), and progesterone (tissue softening) mean that the net effect of pregnancy on OTM in humans is unknown (Table 4).

Table 4. Gestational hormones: pattern, tissue action, and hypothesised effect on OTM

Hormone	Course in pregnancy	Periodontal and bone action	Hypothesised effect on OTM	Evidence level
Estrogen	Rises steadily to term	Inhibits osteoclastogenesis; lowers IL-1, IL-6, TNF-α; raises vascular permeability	May slow OTM	Animal; human indirect

Progesterone	Rises steadily to term	Raises gingival vascular permeability and PGE2; lowers collagen synthesis	More mobility; effect on rate unclear	In vitro; human gingival
Relaxin	Peaks at about 8–12 weeks, then plateau	Raises MMPs and collagen turnover; reduces PDL collagen in rats	May speed initial movement and reduce anchorage stability	Animal only
PTHrP and calcitriol	PTHrP from placenta and breast; calcitriol roughly doubles	Support fetal calcium supply; raise bone turnover	Unknown	Physiological; no OTM data
Net effect in humans	—	—	Unknown; competing effects	No direct study

2. Periodontal Alterations and Inflammatory Dynamics

- **Pregnancy gingivitis:** Gingival inflammation is reported in a large proportion of pregnant women (commonly cited as 60–75% [5], with higher figures in older studies depending on the index used [18]). It begins around the second month, peaks around the eighth month, and resolves after delivery. It is driven by estrogen and progesterone acting on the gingival vasculature together with plaque, and by changes in the immune response. Fixed appliances retain plaque and may amplify the response (Table 5).
- **Soft-tissue enlargement and tooth mobility:** Pyogenic granuloma (pregnancy epulis) affects a minority of pregnant women [5,9], usually from the second trimester, and is driven mainly by plaque and calculus rather than by orthodontic force. Temporary tooth mobility may occur late in pregnancy and resolves postpartum.
- **Periodontal attachment loss:** Pregnancy gingivitis is reversible and has not been shown to progress to periodontitis. In patients with pre-existing or poorly controlled periodontitis, it is hypothesised that orthodontic forces and gestational inflammation could add to attachment loss; this has not been demonstrated. Periodontal status should be stabilised before treatment and monitored throughout [9,11].

Table 5. Oral and periodontal conditions in pregnancy and management with fixed appliances

Condition	Timing and frequency	Mechanism	Management
Pregnancy gingivitis	Onset about month 2, peak about month 8; commonly 60–75%	Hormonal vascular change plus plaque; immune shift	Oral-hygiene instruction, scale and polish, short alcohol-free chlorhexidine course, frequent recall
Pyogenic granuloma	Second to third trimester; a minority; resolves after delivery	Exaggerated response to local irritants	Remove plaque and calculus; excise only if bleeding or interfering; recurrence is common
Transient tooth mobility	Late pregnancy; resolves postpartum	PDL and gingival change without true bone loss	Do not misread as bone loss; avoid adding force
Enamel erosion and caries	Vomiting, reflux, frequent snacking	Acid exposure, reduced hygiene	Fluoride toothpaste and varnish; wait 30–60 minutes before brushing
Gagging	Common in first trimester	Heightened reflex	Intraoral scanner instead of trays; short sessions
Pre-existing periodontitis	Any trimester	Observational links to adverse outcomes; treatment trial did not reduce preterm birth [20]	Stabilise non-surgically before and during orthodontics

4. CLINICAL MANAGEMENT PROTOCOLS AND SAFETY CONSIDERATIONS

Table 6. Summary of clinical management recommendations (revised)

Clinical Domain	Protocol / Recommendation	Biological & Clinical Justification
Diagnostic Imaging	Defer elective radiographs and CBCT; take clinically necessary films with collimation, thyroid collar, and apron.	Fetal dose from dental imaging is negligible and far below the 50 mGy level of concern [15]. Deferral applies to elective records. Evidence: guideline.
Force Magnitude	Apply low-magnitude, light continuous forces.	Prevents severe tissue hyalinization, root resorption, and excessive tooth hypermobility; extrapolated from non-pregnant and animal data. Evidence: low.
Treatment Timing	Limit active tooth movement primarily to the second trimester; use passive wires and maintenance in the first and third trimesters.	Convention and expert opinion: first-trimester nausea and organogenesis (about 5–10 weeks) favour minimal drug and radiation exposure; from about 20 weeks supine positioning can cause aortocaval compression [5,13]. Evidence: very low.
Pharmacotherapy	Use paracetamol (acetaminophen) at the lowest effective dose for the shortest time; avoid NSAIDs (limit from 20 weeks, avoid from 30 weeks).	NSAIDs can attenuate OTM, cause fetal kidney effects from 20 weeks, and constrict the ductus arteriosus from 30 weeks [14]. Prescribed low-dose aspirin for pre-eclampsia is not affected. Evidence: moderate to high for NSAID avoidance.
Maintenance	Hygiene and periodontal recall every 3–6 weeks according to gingival inflammation.	Controls biofilm and mitigates gestational plaque-induced gingivitis. Evidence: expert opinion; no trial data.

DISCUSSION

The biological response of the periodontium to orthodontic force during pregnancy reflects an interplay between mechanical loading and systemic endocrine modulation [1,6]. OTM relies on a controlled inflammatory cascade within the periodontium [1,2], and gestational hormones may modify both the dynamics of tooth movement and the surrounding tissue reaction [6,7]. Because no study has measured OTM in pregnant women, the discussion below is inferential.

Hormonal Influence on Biomechanics and Bone Remodeling

Any change in tooth movement velocity during pregnancy is hypothesised to follow hormonal effects on bone metabolism and PDL remodeling [3,7]. Elevated estrogen generally protects bone matrix density by suppressing osteoclastogenesis and downregulating IL-1, IL-6, and TNF-α [6,8]. In theory, this could slow osteoclastic resorption on the compression side and retard OTM [3,7]; this has been shown in animal models but not in pregnant women.

However, this osteoprotective mechanism may be counterbalanced by progesterone and relaxin [6,7]. In animal and cell models, relaxin upregulates matrix metalloproteinases and breaks down PDL collagen [7,16]. This reduction in stiffness, together with progesterone-induced microvascular dilation, may manifest as increased tooth mobility and lower resistance to force [6,8]. The soft-tissue matrix may therefore soften, so that low forces yield unexpected positional shifts [7,9].

Periodontal Vulnerability and Inflammatory Response

A clinically relevant finding is the heightened susceptibility of pregnant patients to localised periodontal inflammation [5,9]. Elevated progesterone alters the gingival microvascular bed, increasing capillary permeability, exudation, and tissue responsiveness [6,8]. Fixed orthodontic appliances act as retention niches for dental biofilm [9,11]. The combined effect of plaque and gestational hormones triggers an exaggerated inflammatory response, predisposing to pregnancy gingivitis and, less commonly, pyogenic granuloma [8,9,12].

If unmanaged, this response could contribute to attachment loss, particularly in individuals with pre-existing or subclinical periodontal disease, although such progression has not been demonstrated in pregnancy [9,11]. Observational studies link periodontitis with adverse pregnancy outcomes, but a randomised trial of periodontal treatment did not reduce preterm birth [20]. Rigorous plaque control and short recall intervals are therefore advisable throughout orthodontic therapy [11,13].

Clinical Protocol Modifications and Maternal-Fetal Safety

The recommendations below are expert-opinion-based and are summarised in Tables 6–11 to protect both clinical efficacy and maternal-fetal safety [5,13]:

- **Force Application:** Because the PDL may show reduced collagen density and heightened vascularity during pregnancy (inferred from gingival and animal data) [6,8], heavy forces may increase the risk of tissue hyalinization, necrosis, and root resorption [1,3]. Light, continuous forces are advisable to preserve tissue integrity [1,11].
- **Timing of Treatment:** The second trimester is generally regarded as the most suitable window for active orthodontic intervention and essential procedures [5,13]; this is convention rather than trial-based. Orthodontic force itself is not known to be teratogenic. The first trimester coincides with organogenesis (about 5–10 weeks of gestation), nausea, and a high background miscarriage rate, so elective procedures and imaging are best deferred [5,13]. From about 20 weeks the gravid uterus can compress the inferior vena cava when the patient lies supine, causing supine hypotensive syndrome; appointments should be brief, with a left-lateral tilt or wedge, and the patient should sit up slowly [5,9]. Stage-by-stage and scenario-based guidance is given in Tables 7 and 8.
- **Analgesic Selection:** Paracetamol (acetaminophen) remains the first-line analgesic in pregnancy at the lowest effective dose for the shortest time [5,13]. In September 2025 the US Food and Drug Administration wrote to clinicians about a possible association with neurodevelopmental conditions while stating that causality is not established; ACOG and SMFM consider the evidence inconclusive and continue to support its use [19]. NSAIDs attenuate OTM in animal and some human studies through inhibition of prostaglandin synthesis [1,3] and carry fetal risks: the FDA advises limiting use from 20 weeks (fetal kidney effects and low amniotic fluid) and avoiding use from 30 weeks (premature closure of the ductus arteriosus) [14]. Low-dose aspirin prescribed for pre-eclampsia should be continued (Table 10).
- **Diagnostic Imaging:** Fetal dose from dental radiography is negligible and far below the 50 mGy level at which ACOG reports no increased risk of anomalies [15]. Elective records (cephalogram, panoramic film, CBCT) can be deferred, but clinically necessary radiographs should not be withheld; collimation, thyroid collar, and apron should be used (Table 9).
- **Special Situations:** High-risk pregnancies (pre-eclampsia, gestational diabetes, threatened preterm labour, anticoagulation) warrant deferral of elective orthodontic activity and liaison with the obstetrician. After delivery, treatment can resume once periodontal status has been re-checked; bone turnover remains raised during lactation [17], with no proven effect on OTM.

Table 7. Orthodontic management by stage of pregnancy (expert opinion)

Stage	Orthodontic activity	Records and imaging	Chair and visit	Hygiene and drugs
Before conception	Finish treatment or reach a stable stage; avoid starting if pregnancy is planned soon	Complete records; periodontal baseline	Normal	Treat caries and gingivitis; fluoride
First trimester (to about 13 weeks)	Maintain; passive or light wires; defer elective starts	Defer elective films and CBCT	Morning, short visits	Paracetamol only if needed
Second trimester (about 14–27 weeks)	Preferred window for bonding and active adjustment with light continuous force	Only if clinically justified; collimated, thyroid collar	Normal; left tilt from about 20 weeks	Scale and polish; paracetamol; limit NSAIDs

Third trimester (28 weeks to term)	Passive wires; repair emergencies; no new elective starts	Avoid elective	Brief visits; left tilt; slow sitting up	Avoid NSAIDs from 30 weeks
Postpartum and lactation	Resume active treatment once oral health is re-checked	Imaging allowed; breastfeeding need not be interrupted [15]	Normal	Paracetamol or ibuprofen are compatible with breastfeeding

Table 8. Scenario-based decision guide (expert opinion)

Scenario	Suggested approach	Counselling points
Planning pregnancy within about a year	Complete or stabilise treatment; defer starting appliances	Retention plan; periodontal baseline; inform clinic as soon as pregnancy is confirmed
Becomes pregnant mid-treatment	Light or passive wires in the first trimester; resume active movement in the second	Frequent hygiene recalls; avoid NSAIDs; inform obstetrician if complications
Seeks new treatment while pregnant	Diagnosis and planning with photographs and intraoral scans; defer appliance start until after delivery unless urgent	Aligners ease hygiene but still apply force; same biological caveats
Emergency (poking wire, debond, trauma)	Treat at any stage	Local anaesthetic and paracetamol are acceptable
High-risk pregnancy	Defer elective activity; hygiene care only	Joint plan with obstetrician
Surgical adjuncts (mini-screws, exposure, extractions, orthognathic surgery)	Defer elective surgery; if essential, second trimester	Local anaesthesia only; avoid sedation where possible
Postpartum and breastfeeding	Resume treatment; re-check periodontal status	Bone turnover is raised in lactation; no proven effect on OTM

Table 9. Dental and orthodontic imaging in pregnancy

Modality	Typical effective dose (approximate)	Fetal dose	Advice
Intraoral periapical or bitewing	0.001–0.008 mSv	Negligible	Take if clinically indicated; collimate; thyroid collar
Panoramic	0.01–0.03 mSv	Negligible	Defer if elective
Lateral cephalogram	0.002–0.01 mSv	Negligible	Defer if elective; collimate
CBCT, small field of view	0.01–0.1 mSv	Negligible	Only if the answer changes management
CBCT, large field of view	0.05–0.5 mSv or more	Very small	Avoid unless essential

Doses vary by device and are approximate. Natural background radiation is about 2–3 mSv per year.

Table 10. Drugs commonly used around orthodontic care in pregnancy

Drug	Use	Guidance in pregnancy	Note
Paracetamol (acetaminophen)	Post-adjustment pain	First choice; lowest effective dose, shortest time	See text regarding the 2025 FDA letter [19]
Ibuprofen and other NSAIDs	Pain	Avoid in the first trimester unless advised; limit from 20 weeks; avoid from 30 weeks [14]	Also attenuate OTM
Low-dose aspirin (81 mg)	Prescribed for pre-eclampsia risk	Continue as prescribed	Analgesic doses should be avoided
Codeine and other opioids	Rarely	Avoid routine use; short prescriber-led course only	Neonatal and breastfeeding concerns
Lidocaine with epinephrine	Local anaesthesia	Generally acceptable at lowest effective dose, with aspiration	Preferred local anaesthetic
Amoxicillin, penicillin, clindamycin, azithromycin	Infection	Generally acceptable	Prescribe only for a clear indication
Tetracyclines, doxycycline	Periodontal adjunct	Avoid	Fetal tooth and bone effects
Chlorhexidine (alcohol-free)	Gingivitis	Acceptable for short courses	Review after 2 weeks
Fluoride toothpaste and varnish	Caries prevention	Acceptable and encouraged	—
Nitrous oxide	Anxious patients	Avoid in first trimester; if essential, short exposure with scavenging and at least 50% oxygen	Seldom needed

Table 11. Certainty of the evidence behind the main recommendations

Recommendation	Basis	Suggested certainty
Light continuous force	Biological rationale and animal data; no pregnancy trial	Low
Active movement mainly in the second trimester	Convention and expert consensus	Very low
Defer elective radiographs	Guideline conservatism; fetal dose is negligible	Very low
Paracetamol rather than NSAIDs	Regulatory and obstetric guidance (FDA, ACOG, SMFM)	Moderate to high for NSAID avoidance after 20 weeks
NSAIDs attenuate OTM	Animal and limited human studies	Low to moderate
Left-lateral tilt in later pregnancy	Obstetric physiology	Moderate
Frequent hygiene recall	Expert opinion	Very low

Limitations and Future Directions

Direct clinical trials of OTM in pregnant women are lacking, partly for ethical reasons [5,13]. This review identified no human study of OTM in pregnancy, and current understanding relies on animal models, in-vitro work, and observational reports on the pregnant periodontium [3,7]. Further limitations are restriction to English-language publications, absence of a formal risk-of-bias assessment, and reliance on narrative synthesis. Future prospective research using non-invasive tools such as gingival crevicular fluid (GCF) biomarker analysis could clarify the metabolic and enzymatic changes around moving teeth across trimesters [1,7,12].

CONCLUSION

Orthodontic tooth movement during pregnancy represents a complex biological scenario in which mechanical forces interact with profound systemic hormonal shifts. Estrogen, progesterone, and relaxin may alter bone metabolism, PDL matrix turnover, and microvascular permeability, thereby modifying tissue responses to mechanical loading; the net effect on tooth movement velocity in humans is unknown. Pregnancy increases periodontal vulnerability to plaque, yet orthodontic treatment remains manageable when appropriate clinical modifications are implemented.

To safeguard maternal-fetal health, orthodontists should, as expert-opinion-based recommendations, apply light, continuous forces, enforce rigorous oral hygiene protocols, and schedule active interventions primarily within the second trimester. Elective diagnostic radiography may be deferred, although necessary films should not be withheld, and post-adjustment analgesia should be restricted to paracetamol at the lowest effective dose, avoiding NSAIDs, which can attenuate prostaglandin-mediated remodeling and carry fetal risks from 20 weeks. Through interdisciplinary collaboration and tailored treatment strategies, clinicians can manage orthodontic care during pregnancy while minimizing periodontal complications.

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