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In some respects, it seems relatively early for a second edition to be published seven years after the first one. However, the subject of mini-implants in orthodontics is a dynamic and rapidly evolving field in terms of research publications, clinical techniques, collective comprehension and clinical insight. Some of these refinements appear subtle, but nevertheless represent clinically significant refinements in both our comprehension and applications. Therefore, this second edition has arisen from the realisation that both experienced orthodontists and those new to mini-implant anchorage will benefit from an updated appraisal of this progressive field and its ever-increasing applications in clinical orthodontics.

For example, our understanding of the nature of three-dimensional control of target tooth movements has greatly increased in the last 10 years. This has resulted from the initial recognition and then comprehension of biomechanical side-effects observed during the early years of mini-implant usage (and described in Chapter 1). This increased understanding has been matched with the introduction of relatively simple but effective clinical adjuncts such as powerarms (as described in Chapters 7 and 9). These have greatly enhanced our biomechanical control of anterior and posterior tooth movements. Consequently, we are now much more able to control not only anchorage but the delivery of bodily tooth movements in all three dimensions. However, it is important to acknowledge that mini-implants do not provide ‘miracle’ solutions since orthodontics still has to overcome biological limitations such as the severe alveolar bone deficiencies in hypodontia cases (discussed in Chapter 9).

Over the last 10 years, existing clinical techniques have been refined, and become more standardised and evidence based, as exemplified by protocols for molar intrusion in the treatment of anterior openbites (Chapter 10). The outer envelope of achievable orthodontic treatments has also been further expanded. This is amply illustrated by the inclusion of a new chapter in this edition on mini-implanted anchored maxillary expansion (Chapter 13). Last, but not least, orthodontic diagnostic imaging options have changed dramatically in recent years with the dissemination of lower dose cone beam computed tomography (CBCT) machines and software. This has had a positive impact on the use of mini-implants with the introduction of 3D imaging and planning techniques (described in Chapter 5).

However, one key theme has been continued from the first edition. The second edition still provides a combination of pragmatic clinical advice on common orthodontic clinical problems, based on a synthesis of the literature evidence base, judgement, experience and clinical insight. Therefore, I hope that you enjoy reading this textbook and that I have done service to my orthodontic peers and, indeed, to Sir Isaac Newton, the physics-based father of anchorage concepts.

Richard Cousley
2019
This chapter could alternatively have been titled ‘the advantages and disadvantages’ or, more trendily, ‘the pros and cons’ of mini-implants, since it describes what we have to gain and possibly lose from their use. However, before we embark on these details, it’s important to summarise what is meant by orthodontic mini-implants and how we’ve arrived at current clinical applications.

1.1 The Origins of Orthodontic Bone Anchorage

Orthodontic-specific skeletal fixtures were developed from two distinct sources:

- restorative implants
- maxillofacial surgical plating kits [1].

Orthodontic implants were first produced in the 1990s by modification of dental implant designs, making them shorter (e.g. 4–6 mm length) and wider (e.g. 3 mm diameter). However, they retained the crucial requirement for osseointegration, which is a direct structural and functional union of bone with the implant surface causing clinical ankylosis of the fixture. In contrast, orthodontic miniplates and mini-implants (miniscrews) are derived from bone fixation technology, and primarily rely on mechanical retention rather than osseointegration. In effect, modification of the maxillofacial bone plate design, adding a transmucosal neck and intraoral head, resulted in the miniplate, whilst adaption of the fixation screw design produced the mini-implant. Since the start of this millennium, a wide variety of customised orthodontic mini-implants have been produced and these are now used in the vast majority of orthodontic bone anchorage applications. Orthodontic implants are no longer in standard use and the invasive nature of miniplates tends to limit their use to orthopaedic traction (e.g. Class III) cases or occasionally where the alveolar and palatal sites are too limited for mini-implant usage (as exemplified in Chapter 8).

1.2 The Evolution of Mini-implant Biomechanics

Hindsight is a wonderful tool, especially with new treatment modalities such as orthodontic mini-implants. Much has changed in my clinical practice since I first used mini-implants, back in 2003. And when one looks at the early texts on mini-implants (including the first edition of this textbook), the evolution of techniques is also very apparent. The mini-implant evolution in the 10 years from circa 2005 to 2015 may be best summarised as shown in Table 1.1.

1.3 3D Anchorage Indications

This gradual refinement of mini-implant techniques has been accompanied by a substantial increase in the range of clinical applications for mini-implants. The proportion of these uses will vary between orthodontists, depending on their individual caseloads, and even on financial and cultural influences. Overall, it’s best to subdivide modern anchorage control according to each of the three dimensions and ‘other’ applications, with common examples for each category listed below.
1.4 Using the Right Terminology

Unfortunately, a misleading array of terms has been used for bone anchorage devices (BADs) and their applications in both journals and the commercial literature. Essentially, it is best to encompass all types of fixtures which provide skeletal anchorage under the umbrella terms BADs or temporary anchorage devices (TADs), although the latter term does not indicate the essential role of bone in this anchorage. This book covers only one of the three types of BADs: mini-implants. Whilst the terms mini-implant and miniscrew are used interchangeably in the literature, it is erroneous to use the terms microscrews or microimplants since these fixtures are small (mini) and not microscopic. I prefer the term mini-implant since it conveys the small size and implantable nature of these temporary fixtures.

Second, there appears to be much misunderstanding over whether mini-implants osseointegrate. Most mini-implants are made from either titanium or titanium alloy and histological studies show variable levels of bone–implant contact (BIC) [2,3]. However, it is misleading to refer to this as osseointegration. Rather, clinical usage and percussion indicate that mini-implants are mechanically retained (like bone fixation screws) rather than forming a clinically discernible ankylosis with the bone. Hence, mini-implants can be immediately loaded and easily unscrew, usually without anaesthetic, at any time after insertion. This may be because of their relatively smooth surface and possibly because the surface contact is more a physical phenomenon than a biochemical one.

Table 1.1 The clinical evolution of orthodontic mini-implant anchorage is subdivided into three chronological stages, with a description of the main focus at each stage along with representative clinical examples and associated side-effects

<table>
<thead>
<tr>
<th>Stage of evolution</th>
<th>Clinical focus</th>
<th>Technique examples</th>
<th>Side-effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Reliable anchorage</td>
<td>Direct anchorage, from alveolar sites</td>
<td>Vertical effects of oblique traction, e.g. lateral openbites and uncontrolled incisor movements</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Indirect anchorage, especially palatal sites</td>
<td>Hidden anchorage loss due to failings of connecting anchorage components</td>
</tr>
<tr>
<td>2</td>
<td>Minimised side‐effects</td>
<td>Traction powerarms</td>
<td>Prevention of incisor extrusion/retroclination</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rigid transpalatal auxiliaries</td>
<td>Prevention of molar buccal/palatal tipping movements during intrusion</td>
</tr>
<tr>
<td>3</td>
<td>Optimised target tooth movements (in addition to anchorage control)</td>
<td>Controlled 3D tooth movements during: incisor retraction, molar distalisation, molar intrusion</td>
<td>Bodily movement of target teeth, e.g. torque control during incisor retraction, bodily distalisation of molars, vertical molar intrusion movements</td>
</tr>
</tbody>
</table>

1.5 Principal Design Features

Most mini-implants have three constituent parts: the head, neck and body (Figure 1.1), and are fabricated from a titanium alloy such as surgical grade 5 (Ti-6Al-4V). Grade 5 machined (smooth) titanium alloy is a good choice for mini-implants because it supports rapid cell proliferation and has good cytocompatibility (which is better than stainless steel) and cell adhesion [4,5]. The head is the platform which connects to orthodontic appliances or elastic traction. The neck is the part that traverses the mucosa. The body is the endosseous section with threads around a core and a tapered tip. Mini-implants were initially available only in self-tapping (non-drilling) forms whereby a full-depth pilot hole had to be drilled before mini-implant insertion. However, many self-drilling screws are now available. These have a tapered body shape with sharp tips and threads, and are inserted in a corkscrew-like manner. Full-depth predrilling is avoided, although shallow perforation of the cortex is still advantageous where the cortex is thick or dense, for example the posterior mandible and palate.

1.6 Clinical Indications for Mini-implants

Mini-implant usage may be broadly divided according to the case application and form of anchorage.

1.6.1 Routine Cases

- Cases with high anchorage demands, such as retraction of prominent upper incisors or centreline correction (especially where unilateral anchorage only is required). Orthodontists new to mini-implant use may find it easiest to introduce them into their clinical practice in such
cases since the other aspects of the treatment are usually uncomplicated, enabling the orthodontist to readily recognise the anchorage effects and gain experience.

- Adults and older adolescents who wouldn’t comply well with other anchorage options, especially headgear.
- Where extrusive tooth movements would be unfavourable (risking an anterior openbite or vertical excess).

1.6.2 Complex Cases

- Where conventional biomechanics would be limited, for example molar intrusion to correct an anterior openbite.
- Where conventional dental anchorage is limited by an inadequate number of anchor teeth (due to tooth loss or hypodontia) or periodontal support.

1.6.3 Direct and Indirect Anchorage

Direct loading is when traction is applied from the mini-implant's head to an appliance, typically with elastic chain or nickel titanium (NiTi) coil springs (Figure 1.2a). Indirect loading involves using the mini-implant to reinforce anchor teeth, from which traction is applied (Figure 1.2b,c). The most commonly shown example of this involves mid-palatal mini-implant(s) anchorage of the first molars. This approach is advocated because of the high success rates of parasagittal mini-implants, even in adolescents. For example, a retrospective study of 384 parasagittal mini-implants inserted by Dr Björn Ludwig (in Germany) gave a 98% success rate [6]. Whilst indirect anchorage also has a potential advantage of avoiding some potential biomechanical side-effects (discussed later in this chapter), it carries the risk of insidious anchorage loss through flexing of the intermediary wire connection and undetected tipping or bodily translation of the mini-implant. This has been reported to cause up to 0.5 mm of movement (anchorage loss) of the anchor tooth, in any one plane, when it is connected by a short piece of 0.019 x 0.025 in steel wire to a mini-implant [7], and 1–1.4 mm mean anteroposterior mesial drift of indirect molars in cases treated with bimaxillary incisor retraction [8].

Becker et al. [9] recently published a meta-analysis of en masse retraction treatments involving direct (buccal mini-implants) and indirect (palatal) anchorage [9]. Their results show that direct traction techniques provided better anchorage control than indirect anchorage approaches in both anteroposterior and vertical planes. This appears to have been due to occult mesial migration of some palatal mini-implants and bending of the transpalatal arch (TPA) component. Therefore, it was recognised that direct anchorage provides better outcomes in terms of anchorage control despite the possibility of higher mini-implant stability in midpalate sites. Interestingly, the favourable biomechanical effects of direct anchorage, such as controlled bodily movement of target teeth, were not studied in this meta-analysis, but represent an additional clinical benefit of direct anchorage usage [10–14]. This aspect will be discussed in detail at the end of this chapter.

In summary, I prefer to use direct anchorage wherever possible and this will be elucidated in the clinical scenario chapters. A key exception to this rule occurs in young patients whose bone immaturity means that the higher success rates of midpalate sites negates the biomechanical limitations of indirect anchorage. In effect, direct anchorage prioritises biomechanical considerations and indirect anchorage focuses on anatomical factors.

1.7 Benefits and Potential Mini-implant Complications

Mini-implants have been shown to provide maximum anchorage along with the following benefits.

- No need for additional patient compliance (over and above the compliance required for fixed appliance treatment).
- Flexible timing for anchorage control, such that mini-implant anchorage may be ‘switched’ on and off at virtually
any stage in treatment. This differs from conventional options where the anchorage, such as headgear, needs to be applied at the outset and is very difficult to add later in treatment.

- Greater predictability of both the treatment mechanics and clinical outcomes. For example, one can now confidently retract the labial segment without anchorage or torque loss in a controlled manner (as described in Chapter 7).

- Reduced treatment time, especially where it’s more efficient to move groups of teeth rather than subdivide movements in an attempt to spare anchorage demands. This is exemplified by *en masse* retraction of the canine and incisor teeth in a single phase, rather than two-phase retraction of the canines then incisors. A randomised trial showed a four-month time saving in this respect [15].

- 3D anchorage control. Traditionally, orthodontists think of anchorage reinforcement in the anteroposterior dimension, with much less emphasis on vertical and transverse anchorage. However, now that it’s feasible to control anchorage in all three dimensions, orthodontics can truly aim to correct 3D malocclusion traits.

However, a number of risks and side-effects have been observed over the years with mini-implant clinical usage and in the research literature. Fortunately, these are reversible in most clinical situations, but it is important to consider them in an effort to maximise mini-implant treatment success and to provide informed patient consent. The main risks are described in the following sections.

### 1.8 Mini-implant Success and Failure

Failure of a mini-implant is the ‘risk’ that one ought to focus on most in day-to-day clinical terms. Mini-implant failure means that it cannot be used for its intended clinical
loading/anchorage purposes. Conversely, mini-implant success is generally defined as the fixture remaining stable under continuous orthodontic loading for a minimum period of six months, although many studies have used one year as the minimum observation time. One of the most recent overviews of success rates has been provided by a meta-analysis of 3250 mini-implants, which showed a combined rate of 86% [16]. When the effect of insertion site is analysed, there is a consensus in the literature that the success rate varies according to the jaw involved, at approximately 80% and 90% for alveolar sites in the mandible and maxilla respectively, and up to 99% in the midpalate [17–30]. Conversely, success rates for infrayzygomatic sites are relatively low, at 78%, as reported in a study of 30 consecutive Caucasian patients [31]. This may seem counter-intuitive since the mandible is generally regarded as the stronger jaw bone, but the reasons for this paradox will be explained in Chapter 2.

Interestingly, mini-implants with minor mobility may still be graded as successful. This is evident clinically by slight rotational or lateral movement of the mini-implant on manipulation. This is painless and consequently asymptomatic for the patient. It is easily resolved by tightening the mini-implant, usually by a clockwise turn (insertional rotation), provided that this does not submerge the head, and without the need for anaesthesia. Notably, for Infinitas mini-implants, one complete turn equates to 0.7 mm further insertion. However, if the mini-implant displays obvious lateral mobility with light digital pressure then this indicates failure and the mini-implant should be removed.

Fortunately, most mini-implant failures become clinically evident within the first few months of insertion [20,24,25], enabling early replacement or a modification to the treatment plan. However, it is important to realise that replacement mini-implants still have much lower success rates than primary insertions [32,33]. For example, a recent study of 471 mini-implants showed primary and secondary (replacement) success rates of 85% and 58% for maxillary buccal sites and 79% and 77% respectively for midpalate sites [33]. This marked drop in success rates for buccal, but not palatal, insertion sites suggests that the midpalate ought to be considered if a maxillary buccal mini-implant fails unless there has been an obvious and rectifiable explanation for the failure. Therefore, it is important to determine the likely reason for failure and undertake remedial clinical steps, such as root divergence, to favourably alter the chances of success for a secondary buccal site insertion. On the plus side, when a mini-implant feels firm after two months in situ then normal orthodontic forces may be applied with confidence.

Mini-implant failures are staged according to the time taken for this to manifest after insertion.

- Primary failure occurs when a mini-implant is clinically mobile at the time of insertion. This is due to inadequate cortical bone support in terms of its thickness and density, or close mini-implant proximity to an adjacent tooth root or incorrect insertion technique. These factors will be fully discussed in the relevant sections.
- Secondary failure refers to a situation where the mini-implant is initially stable but then exhibits mobility, usually after 1–2 months. This delayed instability is due to bone necrosis around the mini-implant threads, which may result from thermal bone damage (during pilot drilling), excessive insertion torque, excessively close proximity to a tooth root, traction overload, or a combination of these.

Most mini-implant failures become clinically evident within the first few months of insertion, enabling early replacement.

1.9 Medical Contraindications

There are no absolute medical contraindications which specifically apply to orthodontic mini-implants. Conditions, such as diabetes mellitus and immunosuppression, which are relative contraindications to orthodontic treatment in general must be considered in terms of soft tissue hyperplasia and infection risks. However, if the patient has good oral hygiene then comprehensive treatment may proceed as normal. Older, especially female, patients with osteoporosis may present problems in terms of reduced bone support and hence mini-implant stability, but this can be accounted for in terms of insertion site and force application considerations. The increasing number of older patients on bisphosphonate drug treatment are a specific group which may limit orthodontic treatment, especially against tooth extractions (because of osteonecrosis risks). Whilst I have successfully treated patients taking oral bisphosphonates with routine orthodontic treatment (e.g. alignment), I have no experience of using mini-implants in this group of patients and am unaware of any literature published on this.

1.10 Root/Periodontal Damage

Multiple clinical and animal studies have been conducted with the aim of intentionally inflicting damage on tooth roots, using both pilot drills and self-drilling mini-implants [34–42]. Fortunately, these studies have consistently shown that traumatised root surfaces are repaired within 12 weeks by cellular cementum and periodontal regeneration,
provided that there is no infection portal present (which is usually the case). The cementum repair even occurred when root dentine was fully exposed [34]. Orthodontists can also be reassured that there are no known reports of tooth ankylosis or loss arising from mini-implant use. This may be because, in normal clinical usage, if a self-drilling mini-implant contacts a root then the insertion stalls and its tip will become blunt, preventing extensive penetration of the root tissues. Furthermore, the patient is likely to complain of pain (from periodontal pain receptors) even before root contact occurs. If the root is actually contacted then the orthodontist is also likely to feel a sharp increase in insertion torque [43].

So if a mini-implant doesn’t actually contact a root surface, is there still scope for indirect damage? A recent finite element analysis study indicated that less than 1 mm of separation of the mini-implant and the adjacent root surface may still risk root resorption because a transfer of stress through the thin layer of bone causes an osteoclastic reaction beside the root surface [44]. However, this has not been validated by animal or clinical studies, and the reciprocal effect on bone remodelling around the mini-implant is more likely to have a negative impact. For example, a histological analysis of mini-implants inserted in a dog model showed a significant reduction in BIC where the implant body contacted the root or even just the bundle bone (around the periodontal ligament) [45]. Therefore, it is reasonable to conclude that any irreversible effect from close proximity of a mini-implant and a tooth root will be on the mini-implant: it will have an increased risk of failure (by becoming mobile) rather than the tooth being irreversibly damaged [43,46–50].

**1.11 Perforation of Nasal and Maxillary Sinus Floors**

Concerns have been raised in the literature that mini-implant perforation of the nasomaxillary cavities (Figure 1.3) may result in either infection or the creation of a fistula. However, the consensus based on dental implant research is that a soft tissue lining rapidly forms over the end of a perforating fixture, and that mini-implant sites heal by bone infill because of the narrow width of the explantation hole. Motoyoshi et al. [51] investigated clinical effects in a retrospective study where 82 mini-implants had been inserted mesial and buccal to the maxillary first molar [51].

![Figure 1.3](image)

**Figure 1.3** Coronal slice views of a CBCT scan of the maxilla (a) before and (b) one month after insertion of mini-implants in palatal alveolar sites. The mini-implant, sited distal to the right maxillary first molar, has been inserted at a relatively vertical inclination and has perforated the maxillary sinus, as seen in (b). However, this was asymptomatic and there has been no change in the clarity of the maxillary sinus.

They found perforation of the maxillary sinus in 10% of the sites, but with no sinusitis symptoms, nor differences in insertion torque and secondary stability. In contrast, a study of infrrazygomatic insertions showed that 78% penetrated the maxillary sinus at this site [52]. Whilst these were apparently asymptomatic, mucosal thickening was seen on cone beam computed tomography (CBCT) in 88% of these sites where the mini-implant penetrated by at least 1 mm. Therefore, in order to maximise bone engagement and minimise both patient discomfort and possible sinus disease, it is generally recommended that maxillary alveolar insertion sites should be within 8 mm of the alveolar crest in dentate areas, and at a more coronal level where maxillary molars are absent. The infrrazygomatic crest is not recommended for this reason.

**1.12 Damage to Neurovascular Tissues**

Disruption of the inferior dental, mental or greater palatine nerves and blood vessels is highly unlikely given their relative distance from standard insertion sites. The nasopalatine nerve is closer to potential anterior palatal insertion sites, but this can be readily avoided if recommended midpalatal insertion procedures are followed; for example,
midpalatal insertion sites ought to be distal to the transverse level of the maxillary canines.

**1.13 Mini-implant Fracture**

Mini-implant fracture is thankfully a rare occurrence nowadays since most mini-implant materials and designs do not easily fracture within the normal torque limits in clinical practice \[53,54\]. However, some studies, aimed at fracturing mini-implants in hard acrylic blocks, have failed to allow for the low insertion torques experienced with many designs and especially in the case of self-drilling body versions. Therefore, fractures due to poor clinical technique may be attributed incorrectly to mini-implant weakness.

Fracture may occur during insertion, but also on removal if the mini-implant has been preweakened. Fracture of the mini-implant tip may occur when a root is inadvertently contacted (i.e. the insertion position and/or angle is incorrect) or when the insertion angle is altered with the mini-implant partially inserted through the cortical plate. This is most likely to occur due to incorrect technique and/or clinical inexperience. Fracture of the main section of a mini-implant body is a particular risk, on either insertion or removal, with mini-implants which feature a narrow diameter and cylindrical body design (Figure 1.4) \[55,56\] or when excessive insertion torque occurs (e.g. in the posterior mandible with dense, thick cortical bone). If a mini-implant fractures on removal, flush with the bone surface, and the retained part is unlikely to impede any remaining tooth movements, then it may be left *in situ* because of the biocompatibility of titanium alloy (Figure 1.4c). In the rare event that removal of a fractured part is indicated then this involves creating access by raising a small mucoperiosteal flap, trephination of a narrow collar of bone around the mini-implant end, and then derotation of the fractured fragment using a Weingarts or mosquitos-like instrument.

**1.14 Pain**

There is often an expectation that high levels of pain will occur but the opposite is true, such that some patients appear to feel virtually no discomfort during and after insertion \[57,58\]. The majority of patients appear to experience mild pressure-related pain at the time of insertion and up to 24 hours of low-level pain thereafter. This is self-limiting, controlled by simple analgesics (e.g. paracetamol or ibuprofen) and comparable (but of shorter duration) to other orthodontic experiences, such as the effects of separators and aligning archwires \[59\], and certainly much less than premolar tooth extractions \[60\]. The latter comparison is beneficial when it comes to explaining the likely pain experience to patients who already have a fixed appliance *in situ*.

Assuming that the superficial soft tissues have been adequately anaesthetised, mini-implant insertions cause dental pain because of the pressure wave generated by insertion of a rigid fixture into a confined bone space. While there are no pain receptors within the bone tissues, if the pressure dissipates further it will reach the periodontal tissues of adjacent teeth, and hence stimulate their periodontal pain receptors. The patient will feel this as dental pain in the affected tooth. Fortunately, I think that it’s within the orthodontist’s scope to proactively reduce the level of pressure discomfort by diverging the roots of adjacent teeth prior to insertion (as described in Chapter 5). This creates more interproximal space and hence a greater distance between the mini-implant site and the adjacent periodontal pain receptors. While there is no clinical evidence to support (or refute) this hypothesis, my experience is that patients with increased interproximal spaces complain of less pain both at the time of insertion and afterwards.

When it comes to mini-implant removal, local anaesthesia is usually not required and indeed, patients find that the injection sensation is worse than the actual discomfort of

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Figure 1.4  Intraoral radiographs taken after (a) insertion of a cylindrically shaped mini-implant mesial to the maxillary first molar, and (b) its fracture near the coronal end of the body. The initial fracture line is visible in the intact mini-implant. (c) Sectional orthopantomograph (OPG) showing the (asymptomatic) retained mini-implant body over five years later.
An exception to this rule may occur when a mini-implant is being removed from the anterior region where there has been some soft tissue overgrowth of the mini-implant head, especially when it has been inserted in loose mucosa (Figure 1.5). For all removal procedures, it is essential that the mini-implant is unwound the entire way out of the implant hole since any attempt to pull it out will result in soft tissue pain where the threads catch on the mucosa. In addition, the orthodontist should ensure that the screwdriver is fully engaged on the mini-implant at the start of explantation, since premature disengagement during the removal process will cause pain because of the implant's mobility within the soft tissue envelope.

The most common soft tissue issue related to mini-implant usage is chronic low-grade peri-implant inflammation. This is analogous to gingivitis around the mini-implant neck, and is usually superficial and self-limiting. It is more likely if the mini-implant is either inserted into an area of mobile mucosa (Figure 1.6) or overinserted (partially submerged) in attached gingiva (Figure 1.7). If tissue hyperplasia fails to resolve with oral hygiene measures, and either interferes with use of the mini-implant or causes patient discomfort, then the mini-implant should be removed. Fortunately, acute infections are rarely seen. For example, an audit of my first 500 mini-implant insertions confirmed that only one patient had returned to clinic within several days with a painful, inflamed soft tissue swelling around a mini-implant. This episode of acute infection was readily resolved by immediate explantation of the mini-implant, without the need for antibiotics.

It is also unlikely that peri-implant colonisation by specific pathogenic bacteria is responsible for infection problems in failed mini-implants, as demonstrated by microbiological studies (comparing successful and failed mini-implant flora) [62–65]. Instead, soft tissue causes of failure are probably related to generalised inflammation effects. Even then, it is likely that the soft tissue influences on stability are small compared with other factors such as root proximity.

The labial or buccal mucosa adjacent to a mini-implant head may occasionally be traumatised, manifesting as a mucosal ulcer (Figure 1.8). This is most likely to occur if the mini-implant has a prominent profile (a long head and neck distance from the tissue surface) or sharp edges, or it is inserted in or near loose mucosa. Conversely, mucosal hyperplasia or ulceration may occur if the traction auxiliary (in direct anchorage scenarios) impinges excessively on the underlying mucosa, especially in the presence of poor oral hygiene (Figure 1.9).

1.15 Soft Tissue Problems

The majority of patients appear to experience mild pressure-related pain at the time of insertion.

1.16 Mini-implant Migration

This depends on the head (and neck) to body ratio, on the degree of bone support (stability), and on the relative force level. In effect, both self-tapping and self-drilling mini-implants may tip and/or translate bodily in the direction of the applied force [66–70]. This is problematic if it causes the mini-implant head to approximate an adjacent bracket or crown and cause soft tissue impingement or difficulty in utilising the mini-implant head.
Figure 1.6  (a) Photograph of lower anterior mini-implants immediately after insertion. (b) Hyperplasia of the loose sulcular mucosa around the right mini-implant one month after insertion. (c) Effective oral hygiene measures have resolved the hyperplastic tissue problem on the right side eight weeks later, enabling the continued use of this mini-implant.

Figure 1.7  (a) Hyperplasia of the palatal mucosa covering an overinserted mini-implant in the palatal alveolar site between the left molars. (b) Normal tissue appearance after simple excision of the hyperplastic tissue and replacement of this mini-implant. Minor peri-implant hyperplasia is seen on the right side.
1.17 Biomechanical Side-effects

In many respects, conventional fixed appliances often only exhibit subtle biomechanical side-effects such as frictional binding, tooth tipping and anchorage loss, because these effects are usually localised to single teeth or a group of several teeth. For example, traction applied at the coronal level (to a bracket) may result in tipping and poorly controlled bodily movement of that tooth. Since the adjunctive use of mini-implants provides more profound anchorage, active in all three dimensions and extrinsic to the fixed appliance, the side-effects may also be more strongly expressed and affect the entire arch (when continuous arch mechanics are utilised).

Two pertinent examples of this occur if oblique traction is applied from a mini-implant directly to a canine bracket for anterior segment retraction, with either a flexible or a rigid archwire in place. In the first instance, this oblique vector of traction encourages the canine to tip distally causing the flexible archwire to exhibit a ‘rollercoaster’ bowing phenomenon (Figure 1.10a). In the latter situation, the oblique traction causes a rigid archwire to rotate the entire arch (around its centre of rotation near the premolar apices), causing a combination of incisor extrusion and retroclination and molar intrusion (Figure 1.10b). This manifests clinically as a molar openbite, with development of a vertical step between the first and second molar teeth (if the second molar is unconnected).

Figure 1.8 Labial ulceration caused by this mandibular mini-implant’s insertion at the mucogingival junction and by the active movement of the adjacent labial sulcus.

Figure 1.9 (a) Elastomeric traction auxiliary in contact with the alveolar mucosa following insertion of maxillary buccal mini-implants. (b) Maxillary alveolar ulcerative gingivitis after one month with the powerchain in situ, along with generalised gingival hyperplasia resulting from poor oral hygiene. (c) Photograph taken a further four months later with new traction applied following an improvement in oral hygiene.
1.18 Factors Affecting Mini-implant Success

A large number of mini-implant research papers have been published in the orthodontic (and to a lesser extent the surgical and dental implant) literature at an ever-increasing rate since the start of this millennium. This collective evidence provides a sound basis for mini-implant usage, although it may be difficult for orthodontists and dental colleagues to keep track of all this new information. Consequently, Chapters 2–4 aim to collate and summarise the essential findings of the most relevant scientific and clinical research papers, in order that orthodontists may both understand and maximise their clinical usage of mini-implants. In general, the factors determining success may be divided into three categories and these will be discussed sequentially in the next three chapters.

References


Maximising Mini-implant Success

Patient (Anatomical) Factors

The influences on mini-implant success rates are generally subdivided into three categories: patient, mini-implant (design), and technique factors. These will be discussed accordingly in this book, beginning with patient factors in this chapter.

Patient factors may be subdivided as:

- macro – somatic and general patient factors
- mini – insertion site anatomy
- micro – bone characteristics.

There is now a consensus in the literature that mini-implant success tends to be unaffected by patient gender, anteroposterior (Class I, II, or III) skeletal relationship, dental crowding, periodontal, and temporomandibular status. Therefore, these factors will not be discussed in detail. Having said that, my clinical experience is that gender may have an indirect influence in individual cases in terms of male–female variations in bone characteristics since these factors clearly do affect stability.

The basis and clinical consequences of these bone influences are summarised below. However, it’s first worth defining some of the relevant terminology.

- Primary stability – the initial support for the mini-implant, due to its physical engagement in the cortical and cancellous bone. This is clinically reflected by the final insertion torque. Its influence reduces over several weeks as secondary stability supersedes it.
- Secondary stability – the long-term bone support for the mini-implant. This is due to (reactive) bone remodelling around the mini-implant body. On one hand, bone resorption leads to a loss of primary stability, whilst emerging bone deposition increases the secondary stability.
- Torque – measured in Newton centimetres (Ncm – the same Sir Isaac Newton who described the fundamentals of anchorage in his third Law). Insertion torque is the rotational resistance to a mini-implant being wound into the bone. A manual screwdriver readily reflects this in terms of how easy or difficult it is to turn/rotate the screwdriver: low forces mean low primary stability, while high forces indicate high primary stability. Some mini-implant handpieces give a digital read-out of the torque, or allow the orthodontist to set the maximum insertion torque.

2.1 Cortical Bone Thickness and Density

A combination of clinical, animal and artificial bone studies has demonstrated that the most important patient determinants of primary stability are the density and thickness of the maxillary and mandibular cortical plates. This helps to explain the variations seen in clinical studies of mini-implant success rates where both anatomical sites and individuals differ in terms of the cortical bone layer’s quantity and quality [1]. The key facts to consider are as follows.

- Cortical bone thickness (depth) is generally regarded as ranging from 1 to 2 mm (Figure 2.1) and generally increases towards the apical aspect of the alveolus. However, a recent micro-computed tomography (CT) study of cadavers has shown frequent areas of the maxillary and anterior buccal cortex with less than 1 mm cortex depth (compared to a mean of 1.3 mm palatal alveolar cortex depth and 2 mm or more in the posterior mandible) [2]. In the maxillary alveolus, cortical thickness peaks both mesial and distal to the canines (in the region of the canine eminence) and the first molars, which partly accounts for the frequent use of these sites for anterior and posterior anchorage points, respectively. Notably, the maxillary alveolar cortex is thicker on the palatal than the buccal side, which contributes to the value of palatal alveolar insertions in anterior openbite correction (discussed in Chapter 10), and the highest alveolar values for both jaws occur in mandibular molar sites [2–10].

- An increase in either the cortical thickness or density leads to an increase in insertion torque (the resistance to rotational insertion movements) [11–17]. Thickness and
density are co-dependent factors, with density appearing to be the more influential in terms of mini-implant primary stability [13,14]. The density of the underlying cancellous bone is much less relevant, and hence has less influence on insertion torque [14].

- The ideal range of maximum insertion torque appears to be 5–15 Ncm for alveolar sites [12,18–23]. Interestingly, in my experience, the maximum torque for midpalate sites is often higher, peaking between 15 and 20 Ncm in adults. This has been corroborated by a retrospective study of consecutive midpalate insertions (in adolescents and adults) where 90% had 10–25 Ncm final torque readings [24]. Maximum torque occurs during final seating of the mini-implant and is felt as an increase in resistance on turning a manual screwdriver, such that difficulty in digital rotation typically equates to the top of this torque range. This is clinically valid without it being necessary to measure this in individual patients. In effect, low torque equates to poor primary stability (inadequate cortical support) and excessive torque results in secondary failure because microscopic bone stress leads to microfractures and subclinical ischaemic necrosis around the mini-implant threads [25]. This manifests clinically as the mini-implant screwing in with little resistance at the low end of the torque scale, and it being difficult to manually turn the screwdriver at the high end. Such excessive torque, especially in posterior mandibular sites, may be avoided by initial perforation of the cortical plate, as described later.

- Cortical bone thickness and density are both greater in the mandible than in the maxilla [26]. Our initial instinctive view is that the mandible provides greater primary stability because we regard it as a ‘tougher’ bone. However, mandibular success rates (reported in the literature) are less than those for the maxilla. This is because greater amounts of cortical thickness and density cause excessive insertion torque, which reflects high levels of peri-implant bone stress. This localised stress results in microscopic bone necrosis around the threads and hence secondary mini-implant failure [19].

- Cancellous bone, which has a similar density in both jaws [26–28], has often been suggested to have little effect on primary stability, except when the cortex is less than 1 mm. This occurs in some patients’ maxillary buccal alveolar sites, and the cortical plate provides inadequate stability on its own [2]. In such sites, engagement of the cancellous bone does contribute to mini-implant stability, as demonstrated in an animal bone study [29]. This may account for the positive association between higher cancellous bone density and mini-implant success rates in a study of 127 maxillary buccal mini-implants [30]. Cancellous bone may also influence secondary stability in the long term, by stabilising the mini-implant body against migration and tipping [15,17,31]. This requires a mini-implant with a relatively longer body (e.g. 9 mm length) to engage sufficient cancellous bone area.

2.2 Interproximal Space

The literature provides data on the average amount of interproximal space available for mini-implant insertion, but it is crucial to understand that there is wide individual variation depending on the adjacent teeth’s root sizes, shapes (degree of root taper and curvature) and alignment (i.e. root proximity or separation). Arguably, this almost means that ‘average’ figures are meaningless in individual patients, since each site must be assessed individually for bone volume (Figure 2.2). However, mean measurements do provide useful information such as highlighting that there is more space available on the palatal than the buccal aspect of the posterior maxillary alveolus (e.g. 5 and 3 mm, respectively). This is due to the differential number and shape of the molar roots, specifically the single palatal versus the two buccal roots of the molars (Figure 2.1).

Assuming reasonable tooth alignment, the typical buccal alveolar insertion sites for the maxilla are mesial to the first molar, and adjacent to the canines and central incisors, and for the mandible, adjacent to the molars and premolars [32]. Crucially, limited interproximal space is no longer regarded as a significant barrier since it may be increased

The most important patient determinants of primary stability are the density and thickness of the cortical plates.