Australian Society of Orthodontists

THE UNIVERSITY OF SYDNEY

FACT OR FALLACY ?

PART 1

Creating Brighter Futures

EDITION 4 2022

FACT OR FALLACY? PART 1

In this, the first of a two-part series, a selection of controversial topics within the field of orthodontics will be addressed. Some of these issues have been the subject of much debate for decades.

1. All large maxillary labial frena need Frenectomies

Careful diagnosis is required when assessing spacing, the presence of a prominent frenum and whether or not the frenum requires removal. Patients should not be subjected to unnecessary surgery.

Not all upper midline diastemas are associated with a prominent frenum. Some diastemas are associated with conditions such as generalised spacing, ectopic eruption of teeth, supernumeraries or a tooth size discrepancy (Bolton's Discrepancy) between upper and lower anterior teeth.

Nevertheless, a significant proportion of persistent midline diastemas are associated with enlarged frena.[1,2] Midline diastemas are considered aesthetically undesirable, by both laypeople and dentists, when they are greater than two millimetres in width.[1] (Fig 1).

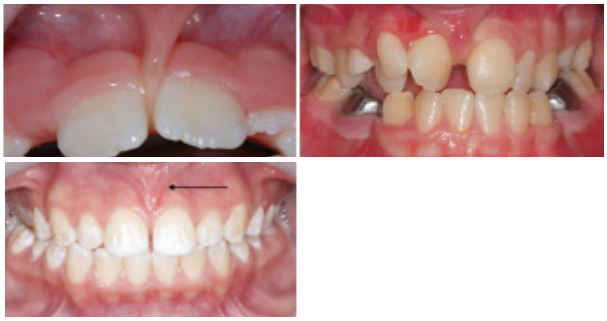

Fig 1 a,b and c. Large frena with diastemas

A large labial frenum can be identified by the movement or blanching of the interdental papilla when the frenum is stretched.[4,5] (Fig 1a). There are numerous reasons cited for a frenectomy to be undertaken, including for periodontal, soft tissue and prosthetic considerations, however, from an orthodontic perspective, the presence of a midline diastema is the major motive.

It is not abnormal for an upper midline diastema with a prominent frenum to exist in the deciduous and mixed dentition. Usually with both growth and the eruption of further permanent teeth, in particular the upper canines, the diastema closes naturally and the frenum recedes.

In some cases, a prominent frenum can exist as a result of the diastema. When central incisors erupt widely separated from each other, continued existence of a prominent frenum is likely, because frenal atrophy from pressure application does not occur.[6] There are two schools of thought on the mechanism by which frenal attachments may cause a diastema. One proposed mechanism is that the bulk of frenal fibres physically impedes physiological approximation of central incisors. An alternative mechanism suggests that the frenal fibres between the central incisors interrupt the transeptal fibres of the periodontal ligament resulting in a weak link in the transeptal fibre network.

Large frenal attachments have been suggested as an impediment to orthodontically closing a diastema. However, studies indicate that the probability of diastema closure is the same, in the long term, with or without a frenectomy.[7] Occasionally after space closure, a large frenum can become difficult to keep clean and inflammation may ensue. (Fig 2). The evidence for an increased rate of space closure with pre orthodontic frenectomy is weak.[8] It has been suggested that orthodontically closing a diastema can create sufficient pressure to cause some or complete atrophy of an enlarged frenum. (Fig 3).

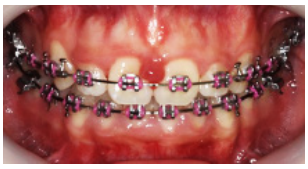

Fig 2. Inflamed frenum after space closure.

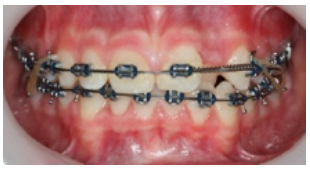

Fig 3. Partial remodelling of the frenum after space closure

There is a recommendation that if a frenectomy is undertaken before closure of the diastema, orthodontic movement should be resumed immediately to prevent scar tissue becoming an obstacle to space closure. Instead, this healing tissue may assist with tooth position stabilisation by acting as a natural retainer.[9] Although scar tissue may be an unwanted sequela of early surgery, the improved surgical access, in some cases, may be a consideration for the frenectomy to be performed prior to closure of the space.

Persistent labial frena have been well documented as being associated with a higher risk of orthodontic relapse following space closure.[10] The best predictor for relapse is pre-treatment diastema size and presence of one family member with a similar condition.[11.] Frenum size appears to be less of an influencing factor. Relapse can be minimised with the use of a fixed retainer, now commonly used after orthodontic treatment, which is a much less invasive intervention than a frenectomy.[11,12] Clinicians must always be mindful to avoid unnecessary surgery and be aware of the associated morbidity and risks.

In summary, in those orthodontic cases where a frenectomy is required, a common clinical approach is to perform space closure prior to frenectomy to aid stability through scar tissue contraction and avoidance of scar tissue becoming an impediment to space closure. This delay in undertaking a frenectomy also allows the clinician to assess whether there may be sufficient natural frenal retraction with growth and orthodontic space closure to avoid a frenectomy altogether.

2. Incisor proclination causes gingival recession

A reported long-term complication of orthodontic treatment can be gingival recession. Gingival recession is defined as "the apical movement of the marginal gingival tissue resulting in exposure of the root surface."[13] Gingival recession can result in poor aesthetics, root sensitivity, loss of periodontal support, difficult maintenance of oral hygiene and increased susceptibility to caries.[14] There are many potential causes and predisposing factors leading to gingival recession, as listed in Figure 4.[13,15]

Trauma e.g tooth brushing	Inadequate plaque control inducing inflammation	High muscle attachment and frenal pull
Chronic Periodontal disease	Host immune response	Occlusal trauma
Thin tissue biotype	Genetic factors	Piercings
Height of keratinised and attached gingiva	Tooth malposition, including proclination	Iatrogenic eg. Orthodontic, prosthetic or periodontal treatment
Narrow mandibular symphysis	Alveolar bone dehiscence	

Fig 4. Possible causes or predisposing factors of gingival recession

Mandibular incisors are the teeth most often thought to be affected by gingival recession which, anecdotally, is strongly associated with proclination. Lower incisors may be proclined during orthodontic treatment for a number of reasons, including correction of a Class II malocclusion, resolution of crowding and levelling of a deep Curve of Spee.[13] (Fig 5). This can result in labially prominent teeth covered with an inadequate or absent labial plate of bone or suboptimal thickness of keratinised gingiva.[14]

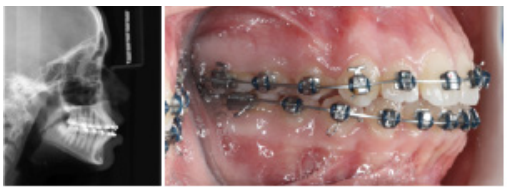

Fig 5. Proclined incisors potentially leading to long term gingival recession

Whether orthodontic tooth movement is a cause of gingival recession, or whether the alveolar bone and gingiva adapt to the new position of the tooth is a controversial issue.[16] Some authors support the theory that orthodontic tooth movement does not necessarily cause soft tissue recession, but rather the resulting thin gingiva caused by the forward tooth movement may predispose these teeth to developing soft tissue defects in the presence of bacterial plaque or trauma.[17]

Despite the reported association between incisor proclination and gingival recession, there is a lack of scientific consensus to support this theory.

The existing studies are weak, and there is a distinct lack of randomised control trials. The vast majority of published studies and systematic reviews have concluded that there is a lack of evidence to support the relationship between proclination and recession.[13,15,18-22] One systematic review which did support the theory stated that:

"More proclined teeth compared with less proclined teeth or untreated teeth had in most studies a higher occurrence or severity of gingival recession".

However, the conclusion from this paper was unconvincing stating that "a cause-effect relationship could be present but that this was supported by a low level of evidence and that statistically significant differences between proclined and non-proclined incisors is small and the clinical consequence questionable."[23] It is postulated that gingiva with a thick biotype can more safely accommodate proclination than gingiva with a thin biotype. If gingival recession does occur gingival grafting may be required.[24]

Although the scientific evidence to support the association between proclination and recession is very weak, the anecdotal evidence cannot be ignored. Therefore, it is prudent for the clinician to take precautionary steps to limit this possible negative side effect. Orthodontic tooth movement, especially in the facio-lingual dimension should be preceded by a thorough clinical examination to determine the quality and quantity of the hard and soft tissues overlying the teeth to be moved.

3. Fixed retainers cause periodontal disease

Fixed retainers are typically made of stainless steel, gold or nickel titanium wires, or fibre-reinforced composite. They are bonded, after orthodontic treatment, to the lingual or palatal surfaces of teeth with composite resin. (Fig 6.)

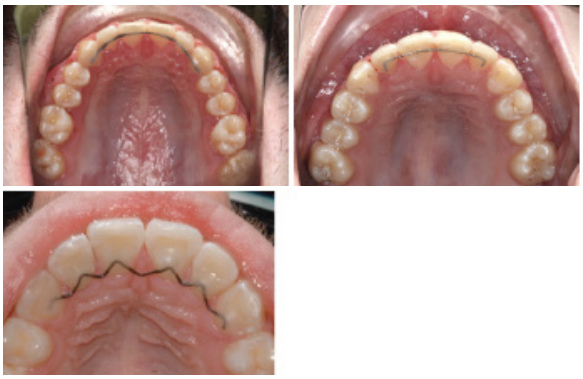

Fig 6. Examples of fixed retainers

Fixed retainers are now widely used and have many advantages. However, their potential periodontal implications and the possible difficulty in maintaining oral hygiene in their presence, has been a long-standing topic of contention.

The presence of a lingual wire and/or bonding resin will complicate the ability of a patient to maintain optimal oral hygiene. Additionally, the design of many fixed lingual retainers prevents the patient utilising traditional interdental floss. In these circumstances the patient has the option to use Superfloss, interdental brushes and/or an AirFlosser to achieve adequate plaque removal.

Conversely, if pretreatment irregular lower incisors are not retained long term, they will eventually move back towards their original positions. This resultant 'recrowding' could potentially increase plaque accumulation with consequent gingival inflammation and adverse long term periodontal consequences.

A recent systematic review of twenty-nine studies, examining the current literature pertaining to this topic, was published.[25] The literature was largely of low quality in nature yielding inconclusive statements. However, it did highlight that there are potential periodontal concerns, particularly with respect to plaque accumulation, gingival inflammation, and changes in the gingival crevicular fluid.[26] While the studies did not report any severe periodontal side-effects, there was a suggestion that fibre-reinforced composite retainers may cause more harm than wire retainers.

Due to the chronic nature of periodontal disease, high quality long-term data will be required to elucidate whether or not fixed retainers significantly contribute to this disease[27]. Unfortunately, the current calibre of data is grossly lacking, with follow-up times often only spanning four to five years.

Nevertheless, the systematic review concluded that:

"Fixed retainers seem to be a retention strategy that is compatible with periodontal health, or at least not related to severe detrimental effects on the periodontium"[25].

Retention plans for patients should be individualised considering the original malocclusion, the patient's oral hygiene practices, their periodontal susceptibility to disease and their perceived ability for long-term cooperation.

A one-size-fits-all retention approach is inappropriate management.[27]

References available upon request

Past issues of Brighter Futures can be accessed at: www.aso.org.au/resources/brighter-futures-newsletters

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Brighter Futures is published by the Australian Society of Orthodontists (NSW Branch) Inc. in conjunction with the Orthodontic Discipline at the University of Sydney.

The newsletter is intended to help keep the dental profession updated about contemporary orthodontics, and also to help foster co-operation within the dental team.

Without the generous support of Henry Schein, who are an integral part of the dental team, this publication would not be possible.

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