A side-effect of bonded retention wires: the "wire syndrome": part 1

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ABSTRACT

Excessive version may be observed in groups of teeth bonded together in fixed retention by a metal wire in a very small percentage of patients. Although quite uncommon, these erratic movements are dangerous for both dental and periodontal health.

We precisely describe this phenomena in both jaws, to enable early detection, before periodontal impact becomes irreversible.

What attitude is appropriate to this “wire syndrome,” and how can it be prevented?

KEY WORDS

Bonded retention, wire syndrome, gingival recession, torque

INTRODUCTION

After multibracket orthodontic treatment, it is essential to apply bonded retention to maintain incisor rotation and intercanine distance. The retention can use simple, twisted or plaited metal wires, individual plates or flexible metal chains. The composition of composite studs also varies, with varying resistance to rupture and wear

Bonding may be direct or indirect².

The present article seeks to describe canine and/or incisor displacement despite perfect retention bonding on all teeth.

We collated and analyzed 60 cases in our casebook and those of colleagues who provided us with photographic documentation.

We shall refer to this phenomenon, which can take various forms, as “wire syndrome”.

It has been partially reported in the literature for some years now, under the term “severe complications”⑧, or “unexpected complications of retention wires”⁴, but to our knowledge has never previously been made explicit or described in the maxillary arcade.

Detecting “wire syndrome” may help avoid severe and sometimes irreversible periodontal complications. We therefore consider it essential to describe it precisely so as to prevent onset when ablating retention. After a description of the syndrome, we make recommendations for screening.

In the cases discussed here, bonding used various techniques: direct, adapting the wire on the actual teeth, or indirect,
preparing wire and studs on a plaster or silicone mold. The wires in question were multi-strand, plaited, twisted or composed of a single smooth strand, and varied in diameter.

Wire syndrome, as we shall see, may implicate all types of wire, regardless of bonding technique.

A mechanical study of the underlying process is underway, and will be the focus of a second report.

OBSERVATION AND DESCRIPTION OF THE VARIOUS ASPECTS OF WIRE SYNDROME

Wire syndrome was mainly observed in bonded retention using twisted round 3- or 6-strand wires, but a very comparable phenomenon was found with plaited wires and flat-link chains, and even with single-strand 0.032-inch steel canine-to-canine rods.

Incidence in a Dutch study of 221 cases at 3 and 5 years’ follow-up after ablation was 2.7%. Although low, this rate is worrying, as patients wearing this kind of retention are not usually being monitored.

The displacements seen in figure 1 were unrelated to the original malpositioning. It was not the kind of relapse found with broken or lost wires, but authentic new malpositioning despite retention perfectly bonded on each tooth (fig. 1).

WIRE SYNDROME IN THE MANDIBLE

In the most frequent form of wire syndrome, the canines show excessive version, which is always inversed if both canines are involved (figs 2 and 3). The wire remains perfectly bonded to both teeth.

Figure 1
Wire syndrome inducing version in both canines. The wire was well bonded. The observed movement was clearly not a case of relapse. a: mandibular arcade before treatment; b: day of retention fitting; c: wire syndrome 4 years after bonding.
Concomitantly, the incisors are projected frontally, leading to tip-to-tip occlusion and, in case of thin periodontium, to root exposure (fig. 4).

In 29 of the 40 cases of “mandibular syndrome” the right canine was in coronovestibular version and in 11 the left canine was in coronolinguval version (fig. 5).

Sometimes, canine version, although abnormal, is negligible and it is the central incisor that shows very strong radiculovestibular (fig. 6) or coronovestibular version (fig. 7).

In some cases, interincisor centers are disaligned and the incisor axis is tilted toward the canine, which is in coronovestibular version (fig. 8).
Figure 5
Collection of 6 distinct cases of wire syndrome in which excessive canine version is inverted between the left and right canines.

Figure 6
Abnormal right central incisor version inducing very severe exposure. Note also coronovestibular version of right canine and coronolingual version of left canine. (Copyright, Dr D. Wiechmann)

Figure 7
Excessive right central incisor coronovestibular version associated with anterior position in all teeth of the group and inverted canine version.
Radiologic aspect of excessive canine version

On panoramic X-ray, the roots of teeth in excessive version seem to have vanished: in reality, they are outside the view (fig. 9).

More modern radiology methods, such as cone-beam CT (CBCT), are useful to analyze root position in severely malpositioned teeth (fig. 10).

After treatment, the roots can recenter in the cortex as seen on CBCT\(^4\). In contrast, Farrel\(^3\), in a case report, failed to recenter the root in the cortex on successive CBCT views.

There are as yet no human studies demonstrating that roots can be reproducibly reintroduced in the cortex. Animal studies\(^12\) showed positive results, but we still do not know whether human translation is feasible\(^11\).

Root recentering can be clearly seen in the panoramic radiograph shown in fig. 11.

The above cases were extreme, with intervals between wire fitting

Figure 8
a: Deviation of mid-points and rightward distovestibular version of incisor-canine group; b: inverse leftward deviation with periodontal impact on 32 and 33, displaced frontally outside the alveolar bone.

Figure 9
a: Panoramic control radiograph after device ablation; b: panoramic view 4 years after retention wire fitting, the left canine root is outside the radiologic frame.
and syndrome detection of 4 to 13 years. Obviously, the earlier the detection, the less severe the impact (fig. 12). The aim of the present article is, precisely, to enable practitioners to detect wire syndrome within 2 years of bonding, so as to be able to remove the toxic retention system during the period in which patients are normally followed up.

Figure 10
In this very severe case (13 years after device ablation), CBCT was prescribed. The left mandibular canine root is clearly outside the cortex (slices 9-11).

Figure 11
Case shown in figure 9 (a) before and (b) after re-treatment. 33 root version was corrected, but with moderate resorption.
A SIDE-EFFECT OF BONDED RETENTION WIRES: THE “WIRE SYNDROME”: PART 1

If the phenomenon is detected soon enough after bonding the wire, effects will be more moderate and resolution easier.

MAXILLARY WIRE SYNDROME

Bonded retention is less frequently applied to the maxilla, as it is often difficult to adjust in occlusion. Residual closed bite introduces interference between the wire and the free edges of the mandibular incisors. This can often be got around by bonding only the 4 incisors, in a 2-2” form. Wire syndrome may also occur with this type of wire when fitted to the maxilla.

To our knowledge, such side-effects of bonded retention have not previously been described in the maxillary arcade.

We found far fewer cases involving the maxilla than the mandible: just 20 out of 60; this was probably because bonded retention is less common there. Only 2 cases involved the canines, as many of these wires did not include them.

Maxillary wire syndrome with canine impact (fig. 13)

When wire syndrome involves a maxillary tooth, the smile is immediately affected and the
patient is not slow to consult (fig. 14).
We found more cases when retention was from one lateral incisor to the other (so-called “2-2”).

One of the 2 lateral incisors tilts in coronovestibular version despite stable bonding (fig. 15).
In principle, the esthetic blemish quickly brings the patient to consultation.

Figure 13
Right canine in strong coronovestibular version. Wire still bonded to all teeth.

Figure 14
Wire bonded to 13-23, with coronovestibular movement in 13. The impact on the smile quickly brought the patient to consultation.

Figure 15
a and b: Wire fitted on the day of device ablation. c and d: Maxillary wire syndrome involving 12. Wire bonded, and crown in coronovestibular version.
WIRE SYNDROME IN NON-TWISTED WIRES

We found some images of wire syndrome involving 0.036 inch single-strand wires bonded only to the canines via small plates resembling the base of a bracket. Such images were rare, but show that the syndrome can also arise with non-twisted wires (figs 16 and 17).

There was also a case of incipient wire syndrome involving a flat plaited Ortho-Flex chain (fig. 18).

Figure 16
Translation of canine block, as with twisted wire. Translation seems to be without version. (Copyright, Le May Orthodontics) a: Retention arch (0.036 inch) bonded only to canines, after device ablation; b: wire syndrome with space opening and anterior translation of 32 and 33; c: after re-treatment.

Figure 17
a: Retention arch just after device ablation; b: wire syndrome. (Copyright, Le May Orthodontics).
The phenomenon thus may arise with plaited as well as twisted wires, flat-link chains and even single-strand canine-to-canine retention without incisor bonding.

RECOMMENDATIONS

In case of wire syndrome, the bonded retention system should be removed as quickly as possible. Patients feel even the smallest tooth movements. Clinical examination and, above all, history-taking are very important for screening.

“Tension” is clearly felt by the patient, with relief on wire ablation.

It may be helpful to wait for 6 months to 1 year before resuming malposition treatment. In some cases, spontaneous recovery reduces the time to re-treatment (fig. 19).

In certain cases, the return to normal is spectacular (fig. 20).

Even so, 2 precautions should be taken in ablating the wire:

– Many patients admit to “playing with the wire” with their tongue. Tongue position at rest and in swallowing should therefore be checked, with rehabilitation in case of doubt. Behavioral counseling is indispensable.

– Parafunctions such as nail-biting have been implicated in this phenomenon by some authors, although no purely mechanical explanation can be given.

To avoid rapid return to rotation during surveillance, slight proximal

Figure 18
Incipient wire syndrome (9 months after device ablation): right canine in coronovestibular version. Retention is by Ortho-Flex Tech flat plaited wire.
enamel reduction may be indicated if the teeth are gritted.

If movement is simple, correction can be achieved with a simple splint (fig. 21).

If, after a period of surveillance, no spontaneous improvement is found, then bonded retention should be resumed to correct version (figs 22 and 23).

In severe recession, as in figure 6, treatment should be resumed as soon as the wire is ablated (fig. 24).

If the patient refuses resumption of treatment, the wire should nevertheless be removed. Otherwise, there is a risk of definite aggravation, for which we could be blamed. Bonetti presented a case at 4 and 5 years post-treatment. Wire syndrome induced severe gum recession. The patient at first refused treatment; the wire was left, inducing severe gum recession.

PERIODONTAL CONSEQUENCES OF WIRE SYNDROME AND RECOMMENDATIONS

Torque and translation induce alveolar thinning and, in case of thin periodontium, root exposure (fig. 25).

The syndrome needs to be identified so as to ablate the wire before any periodontal action: surgery would

Figure 19
Discovery of wire syndrome, ablation, and partial spontaneous correction within 1 year.
Figure 20
a: Anterior and rightward displacement of incisor block, with mid-point deviation and tip-top-tip incisor occlusion; b: 3 months after wire ablation, without re-treatment, spontaneous incisor-canine block repositioning is seen.

Figure 21
Figure 22
Resumption of maxillary treatment by exclusively lingual technique (Win device). Well calculated enamel reduction corrected mid-point alignment and allowed lateral incisor repositioning.

Figure 23
Smile before and after resumption of exclusively maxillary treatment.
Figure 24
Resumption of treatment to correct abnormal version in 41 and 31; a: discovery of wire syndrome; b: wire ablation and immediate resumption of treatment (Win device); c: at 9 months’ treatment; d: fitting a new 0.0175 inch 6-strand wire from 34 to 44. (Copyright, Dirk Wiechmann).

Figure 25
a: End of treatment; b: retention wire from 33 to 43; c: wire syndrome onset with severe recession on 31 and 41. Right maxillary canine in coronolinguval version; d: space formed between 42 and 43.
be ineffective if the progressive mechanism continued to propel the root out of the alveolar bone.

Conversely, early periodontal surgery undertaken as soon as the “toxic” wire has been ablated is pointless, as spontaneous improvement is systematic, even without resumed treatment.

The tooth then needs repositioning by an orthodontic apparatus as well as possible within the bone, to allow intervention on the remaining lesion (fig. 26).

If the periodontal lesion is too severe, treatment should be resumed immediately, with periodontal graft at the end (fig. 27).

If the patient refuses resumption of treatment, the retention wire has to be ablated, to avoid aggravation⁵. The patient often fears relapse, and should be appropriately informed.

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**Figure 26**

*a: End of treatment; b: 4 years after device ablation, onset of wire syndrome with severe gum recession on 32; c: 9 months after retention wire ablation. Tooth recentered and lesion beginning to resolve. Treatment can be resumed.*
Figure 27
Same patient as in figure 6. a: Wire syndrome. The root of 41 has moved into the vestibule and the tooth is in severe radiculovestibular version; b: wire ablation and immediate resumption of treatment. Partial spontaneous correction of recession during treatment; c: end of treatment and residual lesion; d: 15 days after epithelial-connective graft. (Copyright, Dr D. Wiechmann; periodontal treatment, Dr. Axel Berens).

CONCLUSION

If its various forms are known, wire syndrome will not be overlooked on check-up. It may induce severe periodontal lesions. The wire should be removed immediately and the teeth placed under surveillance. Resumed treatment is often necessary.

Incidence is extremely low, but impact for both patient and practitioner is severe.

It is important to inform the patient that wearing a retention wire is not without risk and that check-ups are necessary at least twice yearly after the first two years. Any abnormal movement that the patient may detect could prove progressive and is a reason for taking an appointment.

We are all concerned by this phenomenon, even if bonded wires are not our own choice for retention. The syndrome is generally detected by the patient several years after the wire has been fitted and the practitioner to whom the patient turns may well not be the one that fitted the wire. It would be useful for family doctors to be aware of this kind of movement so as to be able to decide to ablate the wire or to refer the patient to his or her orthodontist. A study of the likely causes of wire syndrome and of the mechanical processes underlying such excessive version is underway and a further report is forthcoming.
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Note: The present study is ongoing, and we would be glad if readers contributed with cases of “wire syndrome”.

Conflict of interest
The authors declare no conflicts of interest.

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