



It is estimated that approximately 50% of the population – to some degree - grind and clench their teeth.

However, despite being prevalent throughout the UK and worryingly on the rise, bruxism often remains overlooked and underdiagnosed. Whilst, for some, bruxing doesn't cause any serious symptoms, for many – the habitual bruxers - the side effects are painful and persistent. Here we discuss the potential causes of bruxism, look at the typical symptoms and explore the possible treatments that aim to help alleviate those suffering.

### What is bruxism?

Originating from the Greek 'brugmos', meaning the 'gnashing of teeth', bruxism is characterised by grinding and clenching – the typical definition used in the UK today. A subconscious activity, bruxism can occur both whilst the patient is awake (diurnal) or asleep (nocturnal).

The latter, commonly referred to as nocturnal or sleep bruxism, is the most wide-spread, affecting almost 80% of bruxers. Often characterised by heavier grinding, sleep bruxism is generally deemed highly destructive when compared to diurnal.

Stereotypically rhythmic and sustained, it is suggested – though figures do fluctuate - that with nocturnal bruxism, nearly 40% more pressure can be exerted on the teeth compared to normal habitual behaviour, such as speaking or eating. It is due to the body's poor motor control during sleep that allows nocturnal bruxism to be so harmful.

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This vastly exaggerated pressure exerted during sleep (by the temporalis, masseter, pterygoids, digastric and stylohyoid muscles) means that bruxism can be responsible for a host of painful and persistent problems such as migraines, tinnitus, poor sleep quality, neck ache and temporomandibular joint dysfunction (TMD). It is the main cause of tooth wear and breakage, disorders of the jaw (pain and limited movement), and headaches and migraines.

However, as a subconscious parafunction, very few sleep bruxism sufferers are aware that they are bruxing. As a result, their local GP is typically their first port of call, unless specifically experiencing tooth wear and/or breakages.

Whilst both children and adults can be affected, it is believed to be most common in 25-44 year olds.

### **Why do we brux?**

The cause is unclear - there are many cited reasons for bruxism dependent on where you look, though it rarely occurs as a standalone.

Found to be linked to multiple sleep disorders, it has been found to occur in periods of wakening during the night, with possible links to sleep disorders such as Obstructive Sleep Apnoea (OSA) though there are also associations between bruxism and other sleep conditions – sleep talking, hypnagogic (state of consciousness between sleep and wakefulness) hallucinations and many more. Various lifestyle factors and, commonly, stress and anxiety, are also linked to bruxism.

Equally, there has always remained a strong relationship with occlusion (or, more accurately, malocclusion), though competing concepts have resulted in much historic, and indeed current, conflict in the dental world when it comes to bruxism and TMD. An interesting paper by Dr Barry Glassman and Dr Don Malizia, *The Curious History of Occlusion in Dentistry*, looks into this and helps us to understand the variance when it comes to the root causes: 'Competing concepts of occlusion have been at the centre of conflicting TMD camps over the years.'

Despite the fact that the use of the abbreviation TMD itself is controversial, the paper goes on to explain that occlusion as the cause of jaw issues such as bruxism is somewhat outdated and based primarily on empirical studies that hold their stance weakly against the more recent evidence-based studies. Equally, a study by Michilotti (2006) demonstrated that introducing interferences in to an otherwise 'perfect occlusion', did not result in symptoms presenting.

In the very early 20th Century, Dr Mór Károlyi – a Hungarian doctor and student of József Arkövy, the founder of the Hungarian scientific dentistry) – discusses the role of occlusion in bruxism (among one of the first found doing so) but his paper fails to cite references. Effectively theorising, Károlyi suggests that 'abnormality of occlusal structure was a basis for abnormal temporomandibular joint function, abnormal masticatory muscle function, periodontal disease and bruxism'. This theory has been continuously communicated, and is referenced in many 21st Century textbooks as fact.

Further strengthened over the years by well-known names such as Goodfriend and Costen, these studies are also questionable when it comes to standing up under criticism.

Dissecting cadaver heads in the 1920s, Goodfriend was looking to show a relationship between occlusion and joint anatomy. Glassman and Maliza explain of this study: "There was no control group, and he had no clinical or social histories on these cadavers. Nevertheless, he drew the conclusion that "muscle spasms, external injuries, deleterious habits, and stressful life situations unquestionably play an important role in the predisposition and exacerbation of craniomandibular disorders." According to the paper, therapy should consist of "re-establishment of harmonious balanced dental occlusion that supports adequate maxillomandibular relationships and positions the closed-jaw condyle relationship in the

forecenter of the temporal fossa.”

Costen used 11 case reports of patients with reduced vertical dimension who reported having ear symptoms, including altered hearing and dizziness. Observing symptoms changes when increasing vertical dimensions, Costen made the assumption that altered pressure on the joints was responsible.

According to Glassman and Malizia, Costen ‘births’ the idea of posterior joint support (and what is now termed the ‘occlusion/pain and dysfunction connection”) hypothesising that occlusion, including vertical dimension, is directly related to TMJ pathology and that the altered pressure on the joint acts as the cause for glossopharyngeal neuralgia and altered Eustachian tube function. All this despite stating in his paper that the mechanics of occlusion are not included...

1956 sees an advance in the the relationship of TMJ ‘disturbances’ and occlusion: Sears reported predictable effectiveness in resolving dental and non-dental symptoms of TMJ disorders by the use of a pivot appliance, now more commonly referred to as the Gelb appliance (after Harold Gelb who used these to treat joint pain in his patients, the posterior pivot supports the jaw joint during clenching in patients with symptomatic temporomandibular joints).

Further to these studies being based of empirical research, much of this is now outdated and some, having delved deeper (and more scientifically) into this area of study, have been shown to be discredited (i.e. the relationship between ‘posterior support’ and the development of degenerative changes in the joint). There are also studies that link occlusion and TMJ disorders but that disagree with previous elements (such as Ramfjord who suggested vertical dimension was irrelevant but that ‘any type of occlusal interference was key’) and many other strong studies that demonstrate no evidence of malocclusion and ‘TMD’.

Despite the history, and the worrying detail that, as Glassman and Malizia put it, the ‘old maps’ govern and direct dental education when it comes to occlusion and TMJ concerns, there is much now that suggests nocturnal bruxism is a centrally mediated disorder.

The fact of the matter is that occlusion – maximum intercuspation – is not common, i.e. our maxilla and our mandible spend approximately 20 minutes in any form of contact within any given two hour period. By simple extension, this cannot be the only reason for some of the symptoms that bruxism presents. This is not to say that malocclusion may not be a contributing factor but simply that the dogmatic approach of ‘ideal occlusion’ may be somewhat flawed, and not, necessarily, provide the answers that we are looking for.

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In turn, this allows for other considerations - those previously mentioned - to be taken in to account. Out of the many associations with nocturnal bruxism, the most common (circa 70%) appears to be stress or anxiety. There are also links with:

- sleep disorders – such as sleep apnoea (obstructive sleep apneics are more likely to brux, though this is not fully understood), talking in ones sleep and semi-conscious hallucinations.
- medication – such as anti-psychotic and anti-depressant drugs.
- life-style choice - alcohol, smoking, the use of drugs.

There also appears to be a genetic predisposition towards tooth grinding, as well demographics, and even educational status.

Despite the multiple associations, most support that bruxism is noticeably exacerbated by stress, a commonly cited contributing factor of bruxism. Our current society, in which people are expected to ‘have it all’, has resulted in increasing pressure and stress, and may well go some way in explaining the rising numbers suffering from bruxism.

**What is clear, is that the common myth that links poor occlusion as the cause of bruxism, generally fails to be supported by literature. There are many patients with ‘bad’ occlusion that show no signs of parafunction, and plenty that have strong occlusion and yet suffer daily from sleep bruxism.**

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## Possible treatment options

Due to the overwhelming percentage of patients who likely suffer from bruxism due to stress and anxiety, behavioural therapy may play an important role. Stress management should be included in patient discussions, and this is where the GP may have an important role to play.

However, for habitual bruxers that suffer debilitating symptoms, a dental appliance may be the only option.

The concern here is that the variety available is vast, with some being available off-the-shelf from the chemist. These are cheap, but not effective – fitting less well than one made by a dentist, and often resulting in low compliancy and little impact.

The same sort of guard – typically made from a soft material - can be custom-made by the dentist, providing a physical barrier between the patient's teeth. This simple concept aims to simply protect the teeth from further damage. However, these devices often last little over a year due to the material and the force that the patient can exert whilst asleep. Furthermore, there is evidence to suggest that soft guards increase muscle activity, resulting in increased bruxism: Okeson found that hard splints significantly reduced muscle activity in 80% of participants, whilst the soft guards significantly increased activity in 50%.

Alternatively, a hard splint may result in greater discomfort, again diminishing patient compliancy, and may still cause stress fractures.

As a result, the SCi (Sleep Clenching inhibitor, known as the NTI-tss in America) was developed – a partial arch splint designed to provide greater comfort and heightened muscle relaxation: by altering the force vectors, and in turn, reducing EMG levels by as much as 80%.

The SCi reduces parafunctional intensity of the temporalis, masseters and, to a degree, the lateral pterygoids, eliminating posterior and canine contact, and thereby reducing temporalis clenching. By inhibiting unwanted muscle activity in such ways, the tension which often results in jaw and neck pain, headaches and migraines, is greatly reduced.

The SCi offers a custom-made fit and minimal discomfort - a prefabricated acrylic matrix is custom fitted over the patient's upper or lower incisors using a thermoplastic material to obtain a 'snap' fit. Both chairside and laboratory-made SCis are available. Compliancy is high and results are impressive:

There is no better method on the market to switch off chronic bruxing. This treatment increases patient loyalty because you've provided something for them that's improved their lifestyle by reducing or eliminating some of their chronic symptoms.

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The SCi is a cornerstone in my dental practice.

*Dr. Pav Khaira. Dental Surgeon and Facial Aesthetics Practitioner at The Old Church Dental Practice and founder of the Migraine Care Institute.*

**Exclusive providers of the SCi splint, please visit us at [s4sdental.com](http://s4sdental.com) or contact us on (0) +44 114 250 0176, for more information.**

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