ABSTRACT
Masseteric hypertrophy, popularly called the benign masseteric hypertrophy (BMH) or idiopathic masseteric hypertrophy (IMH) is a relatively uncommon condition characterized by asymptomatic enlargement of one or both masseter muscles and is rarely accompanied by pain, which is often confused with parotid swelling. Most frequently, clinicians are consulted for swelling of angle of mandible which needs to be corrected for cosmetic reasons. Although it is tempting to produce malocclusions, bruxism, clenching, temporomandibular joint disorders, the etiology in majority of the cases are unknown. The purpose of this review is to have a discussion on the effects of masseter muscle on craniofacial morphology, its implication in different types of malocclusion and contemporary orthodontic management and stability.

Keywords: Masseter muscle, Hypertrophy, Craniofacial morphology, Growth pattern.

INTRODUCTION
Masseteric hypertrophy was first described by Legg in 1800 in a 10-year-old girl who had concurrent idiopathic temporalis muscle hypertrophy.1 The masseter muscle, one of the important muscles for mastication located lateral to the ramus of mandible plays an important role in facial esthetics. A hypertrophied muscle will alter facial symmetry, generating discomfort and negative cosmetic impact in many patients (Figs 1A to C). In some patients, this can produce functional alterations of the mandible leading to bruxism, prognathism, clenching and trismus. It may affect anyone regardless of age, gender and ethnicity, however most studies state that adolescents and young adults are more prone to this condition.2 Also it is believed that this condition runs in certain types of ethnic groups, particularly Asian descents and is predominantly seen in males.2,3 Even though there are several theoretical considerations about the etiology of masseteric hypertrophy, the exact cause still remains unclear. Several authors claim that emotional stress leading to chronic forceful clenching of the jaws results in hypertrophy of the muscle.4,5

Masseteric hypertrophy must be accurately diagnosed, as it may be mistaken for other diseases like benign tumors of the parotid gland, benign and malignant tumors of the masseter, unilateral compensatory hyperplasia of masseter (due to hypoplasia of the contralateral side) and lymphangiomas.2 Diagnosis is often based on clinical examination through extraoral palpation of an uninflamed muscle during forceful clenching, awareness and disfigurement of lower third of face. Investigations may even include imaging techniques like computed tomographic (CT) scan to rule out other soft tissue lesions. The purpose of this review is to establish the relationship between mandibular muscle architecture and craniofacial morphology, challenges in management of hypertrophy of masseter muscle with contemporary orthodontic and surgical correction and its long-term stability.

ANATOMICAL AND FUNCTIONAL PERSPECTIVE
The relationship between muscle activity and craniofacial morphology has been studied extensively and reports are innumerable in orthodontic literature.2,4,6-10 Masticatory muscle function and architecture correlates well with the morphologic features of craniomandibular apparatus to which the muscles are geometrically related. Bite force studies have documented diminished occlusal force at the molar occlusal plane in long face adults and increased bite force in deep bite cases.5,11 This difference is not due to the muscle mass or architecture, rather due to the loss of mechanical force in oblique arrangement of muscles in relation to occlusal plane.3,8

Many authors have described the relationship between cross-sectional area of muscles of mandible particularly masseter muscle and facial morphology.5,7 A common finding is that the masseter muscles have large cross section in subjects with short anterior face height and reduced gonial angle. There is often a relation existing between form and function,8 but it is not known whether a genetically determined facial morphology dictates the strength of masseter muscle or a strong musculature influences the form of face.5 In animal
GROWTH PATTERN, BITE FORCE AND ELEVATOR MUSCLES

It was Bjork\textsuperscript{10} who first described the growth rotation of mandible and its relevance in orthodontics. Forward rotating brachyfacial types tend to have deep overbite and backward rotating subjects tend to have open bite. Many investigators argued that these brachyfacial patterns have strong bite force due to the arrangement of mandibular muscles with reduced lower anterior face height which seems to resist vertical posterior dentoalveolar development.\textsuperscript{7-10} The maximum bite force also varies with skeletal craniofacial morphology, decreasing with increasing vertical facial relationships, the ratio between anterior and posterior facial height, mandibular inclination and gonial angle.\textsuperscript{18}

It has been proposed that bite force reflects the geometry of the lever system of the mandible.\textsuperscript{19} That is, the elevator muscles appear to have greater mechanical advantage when the ramus is more vertical and the gonial angle relatively acute. However, the interaction is probably more complex because craniofacial form seems to be determined, at least in part, by the biomechanics of the masticatory muscles. Thus, analyses of structures by CT have shown that the jaw elevator muscles exert influence on their adjacent local skeletal sites by mechanical stresses and that the maximum electromyographic activity in the jaw elevator muscles during clenching is highest in subjects with a square facial type.\textsuperscript{18}

The correlation between occlusal contact area and bite force have been investigated by many authors\textsuperscript{13-15} and they showed that with an increase of the clenching level from 30\% to 100\%, the occlusal contact area is doubled. Due to the biomechanics of the jaw elevator muscles and the lever system of the mandible, the occlusal force is greater on the molars than on the incisors. Correspondingly, occlusal tooth contacts are most frequent in the posterior region. They concluded that the number of occlusal contacts is a stronger determinant of muscle action and bite force than the number of teeth present. All these studies showed that when the mandibular elevator muscle force increase due to hypertrophy, the bite force would also increase and the occlusal contact area is doubled predominantly in the posterior region than anterior region.

ORTHODONTIC CONSIDERATIONS

Treatment planning in orthodontics is not based entirely on biomechanical considerations, but it also requires an awareness
of the craniofacial muscular environment of each patient. The muscles of the maxilla and mandible are of paramount importance in the etiology of malocclusions and jaw deformities as well as in active treatment and long-term stability. Schudy20 believed that variations in the vertical dimension of the face were more significant in identifying facial types than variations in the anteroposterior dimension.

Most orthodontic techniques tend to produce extrusion of posterior tooth, and this extrusion appears to increases the vertical dimension during treatment. In patients with strong masseteric activity, there appears to be a tendency for depression of the molars during chewing or swallowing. Thus, it might be difficult to produce permanent extrusion of molars and backward rotation of mandible in such patients.25 Thus, the recognition of different muscular patterns, growth rotations of the mandible, and profile would influence the premolar extraction decision in each patient.21,22 In deciding the extraction pattern, brachyfacial patients with strong masseteric activity tend to reduce the face height and deepen the bite when premolars are extracted. According to Gianelly,23 the leeway space would allow the relief of crowding in up to 75% of patients, enabling much orthodontic treatment to be carried out without premolar extractions. This might seem to be an ideal approach to the treatment of brachyfacial patients. Many investigators have reported that in such cases orthodontic treatment should be started in late mixed dentition utilizing the ‘E’ space without extraction for management of mild crowding and proclination.24,25

MANAGEMENT OF MASSETER HYPERTROPHY

There are various modalities for management of masseteric hypertrophy including nonsurgical and surgical methods (Table 1). Nonsurgical means of management include use of various drugs-like injecting botulinum toxin-A (Botox) locally over the hypertrophied muscle, radiofrequency electrocoagulation, antianxiety drugs, muscle relaxants, use of splints, physiotherapy and occlusal adjustments. The current surgical options include resection of masseter muscle, surgical reduction of angle of mandible and neurectomy of masseter nerve.

Surgical treatment was proposed for the first time by Gurney in 1947. The procedure consists of a submandibular incision and the removal of 3/4 to 2/3 of all muscle tissue available from the muscle upper aponeurosis to the lower mandibular border.26 Surgical management involves surgical excision of muscle mass with or without bone prominence, a procedure called angloplasty is mainly aimed at improving the esthetic appearance of the patient. In the beginning, the extraoral approach was widely indicated, because it offered better visualization. Although the extraoral approach provides good access to the muscle and angle of the mandible, it also has the distinct disadvantage of producing a surgical scar that may not be acceptable, along with a risk of damage to the mandibular branch of the facial nerve. The intraoral approach reduces the possibility of direct injury to the marginal branch.

However, with the development of new surgical materials and techniques (rotation instruments, surgical saws, specific retractors and more recently, intraoral endoscopy), the intraoral approach has become a good option. In 1951, Converse resected both bone and muscle by excision of the internal layer of the masseter muscle and reduction of the thickened bone in the region of the mandibular angle, via an intraoral approach, which is now considered the surgical treatment of choice.27 Immediately after surgery, it is necessary to apply a fixed compression bandage to the masseteric region in order to obliterate dead space which would otherwise be filled by a hematoma which in turn leads to prolonged postoperative trismus. After removal of the bandage it is necessary to institute mouth opening exercises. Kim and Kameyama,28 in their reported case suggested that angloplasty without muscle resection is also a treatment of choice in some cases of masseteric hypertrophy. Other investigators also documented that surgical resection of mandibular angle alone would give good treatment outcomes.29 The surgical resection itself is having limitations like difficulty in removing great proportions of muscle and bone

| Table 1: Treatment modalities for masseter hypertrophy |
|---------------------------------|---------------------------------|---------------------------------|
| **Etiology**                    | **Nonsurgical methods**         | **Surgical methods**            |
| 1. Emotional stress, anxiety and masseteric hyperfunction | 1. Pharmacotherapy—anxiolytics, muscle relaxants and antidepressants |  |
| 2. Occlusal interferences, chronic bruxism and functional habits | 2. Dental and orthodontic management—dental restorations, correction of premature occlusal contacts, splints, prevention and correction of parafunctional habits |  |
| 3. Esthetic impairment due to unilateral or bilateral moderate hypertrophy | 3. Botulinum toxin A injection into the muscle | Intraoral and extraoral surgical resection of muscle size, removal of angle of mandible, neurectomy of masseter nerve, resection of buccal pad of fat |
| 4. Severe masseteric hypertrophy with esthetic and functional problems | 4. Radiofrequency electrocoagulation for volumetric reduction |  |
in an accurate and symmetric manner and inaccurate surgery results in resection of different proportions between each side, resulting in an asymmetric facial deformity. Other complications from surgical excision of masseter include hematoma formation, facial nerve paralysis, infection, mouth opening limitation and sequelae from general anesthesia.

Hwang et al in their clinical study reported that selective neurectomy of masseter nerve results in muscular atrophy and subsequent reduction of hypertrophic mass of masseter muscle. Precision, bilateral symmetry and predictability of long-term results are doubtful with this technique.

**BOTULINUM TOxin FOR MASSETER HYPERtROPHY**

Injection of botulinum toxin type A into the masseter muscle was first introduced by Smyth, and Moore and Wood in 1994 and considered a less invasive modality for cosmetic correction of the lower face. Botulinum toxin type A injection is reported to be a safe and effective treatment modality in orofacial dystonies, sialorrhea, Frey’s syndrome and muscle hypertrophies.

Botulinum toxin type A is a powerful neurotoxin which is produced by the anaerobic organism *Clostridium botulinum* and when injected into a muscle causes interference with the neurotransmitter mechanism producing selective paralysis and subsequent atrophy of the muscle. The procedure involves percutaneous injection of botulinum toxin A, 50 to 75 units using 2 ml syringe and 25 gauge needle on the center of lower third of hypertrophic muscle at three points which are located 1 cm between each other. The biggest disadvantage of botulinum toxin therapy is that the treatment effect wears away and reverts to the original condition in 6 months. Unlike surgical excision of muscle tissue that reduces the actual number of muscle cells, botulinum toxin type A only reduces muscle volume temporarily.

Even though botulinum therapy does produce a temporary improvement in lower facial thirds, the anatomical effects on craniofacial parts has not been studied extensively. Chi Yang Tsai et al in their animal study argued that the contraction of muscle decreases when the drug is administered, this would produce minimum periosteal activity and remodeling changes to the neighboring bone and thereby craniofacial morphology is altered. The functional effects of this therapy has been studied by Ahn and Kim, and stated that the muscle atrophy as a result of injection causes a reduction of bite force and it gradually recovers by 12 weeks. This argument is supported by Kim et al in their investigation and claimed that the reduction of bite force experienced had little effect on mastication.

Although the use of botulinum toxin injections appear to have certain advantages over surgery, the efficacy and reliability of this procedure is questionable due to lack of available data in terms of randomized controlled clinical trials.

**RADIOFREQUENCY ELECTROCOAGULATION FOR HYPERtROPHY**

Radiofrequency coagulation is the procedure by which an electric current is used as an alternative energy source which causes ionic agitation, leads to tissue coagulation through frictional heating (60-80°C) to denature proteins. Even though this is relatively a new procedure for masseteric hyperactivity, this technique has been used for medical purposes mainly as an alternative procedure for cancer therapy. This ultimately produces a focal necrosis of the hypertrophied masseter muscle without any adverse effects away from the site at which the current is delivered. Few studies documented that this procedure is considered to be a definite option for management of masseteric hypertrophy, but the reliability in terms of long-term results, side effects and complications has to be verified by further studies.

**EFFECTS OF SURGICAL AND NONSURGICAL MANAGEMENT**

There are various factors to be considered in deciding a surgical excision or nonsurgical plan like injection of botulinum toxin or radiofrequency coagulation. The first factor to be considered important in treatment decision is the severity of hypertrophy. A study by Kim et al evaluated the effects of muscle volume reduction following botulinum toxin and surgery, and showed that surgical treatment can reduce up to two-thirds or more of the total volume and botulinum toxin injection can reduce only one-third in its maximum response. Therefore, botulinum toxin injection must be considered only when there is no need for a great amount of volume reduction. Therefore, for severe cases, surgical treatment is indicated. Another important factor is the presence or absence of bone involvement which produce hypertrophy and esthetic problems. Both botulinum toxin injection and radiofrequency electrocoagulation act only on the muscular component of the deformity, resulting in a limited outcome, if there is an associated bone deformity. On the other hand, surgical treatment can manage both muscular and osseous components. Longevity and predictability of the treatment are another concern in management of this condition. Many authors suggest that recurrence following botox injection will occur within 6 months as the pharmacological effect of botulinum toxin type A have a mean duration of 3 months.

**CONCLUSION**

Many literatures on facial morphology and mandibular masticatory muscles particularly masseteric hypertrophy would seem to confirm the relationship between form and function. Vertical corrections achieved during active treatment have to be considered with special care in patients with hyper-masseteric activity and brachyfacial pattern because the strong musculature associated with such cases tend to depress the
extrusion of posterior tooth. The primary objective in management of these cases is improvement in facial esthetics and to decrease the amount of biting forces which depress the posterior. In extreme hypertrophic masseter muscles with facial disfigurement, surgical excision of the muscle with or without angloplasty would be a better choice whereas for moderate situations, noninvasive techniques like injection of botulinum toxin would produce satisfactory results. The long-term effects of botulinum toxin and radiofrequency electrocoagulation as a noninvasive procedure for this condition is to be documented with the help of randomized controlled clinical trials.

REFERENCES