Treatment of Mandibular Prognathism

Hong-Po Chang,* Yu-Chuan Tseng, Hsin-Fu Chang

Mandibular prognathism (MP) or skeletal Class III malocclusion with a prognathic mandible is one of the most severe maxillofacial deformities. Facial growth modification can be an effective method of resolving skeletal Class III jaw discrepancies in growing children with dentofacial orthopedic appliances including the chin cup, face mask, maxillary protraction combined with chin cup traction and the Fränkel functional regulator III appliance. Orthognathic surgery in conjunction with orthodontic treatment is required for the correction of adult MP. The two most commonly applied surgical procedures to correct MP are sagittal split ramus osteotomy (SSRO) and intraoral vertical ramus osteotomy. Both procedures are suitable for patients in whom a desirable occlusal relationship can be obtained with a setback of the mandible, and each has its own advantages and disadvantages. In bilateral SSRO, the intentional osteotomy of the posterior part of the distal segment can offer long-term positioned stability. This may be attributable to reduction of tension in the pterygomasseteric sling that applies force in the posterior mandible. While various environmental factors have been found to contribute to the development of MP, heredity plays a substantial role. The relative contributions of genetic and environmental components in the etiology of MP are unclear. The recent identification of the genetic susceptibilities to MP constitutes the first step toward understanding the molecular pathogenesis of MP. Further studies in molecular biology are needed to identify the gene–environment interactions associated with the phenotypic diversity of MP and the heterogenic developmental mechanisms thought to be responsible for them. [J Formos Med Assoc 2006;105(10):781–790]

Key Words: dentofacial orthopedics, environmental factors, genetics, mandibular prognathism, morphogenetic basis, orthognathic surgery

In the early 1900s, Angle,1 the father of modern orthodontics, described three basic types of malocclusion for dental occlusion: Class I, II and III malocclusions. Class III malocclusion is defined by the mandibular first permanent molar being “mesial”, i.e. forward to normal in its relationship with the maxillary first molar. Lischer2 later termed Angle’s Class III malocclusion as mesio-oclusion. This method of categorization, however, does not provide information about the developmental mechanisms by which the observed occlusal relationship has been reached. A relatively high prevalence of Class III malocclusion, from 15% to 23%, has been observed in Asian Mongoloid populations of Taiwanese, Japanese, Korean and Chinese.3-6 In contrast, most studies reported an incidence of this class of malocclusion in American, European and African Caucasian populations below 5%.7-9 Class III malocclusion is thus a common clinical problem in orthodontic patients of Asian or Mongoloid descent.10-12

Studies indicate that 63–73% of Class III malocclusions are of skeletal type.4,13 Such skeletal cases result from growth disharmony between the mandible and maxilla, thus producing a concave...
facial profile. Patients with skeletal Class III malocclusion can exhibit mandibular protrusion, maxillary retrusion or a combination of the two.\textsuperscript{4,8} Mandibular prognathism (MP) or skeletal Class III malocclusion with a prognathic mandible has long been viewed as one of the most severe maxillofacial deformities.\textsuperscript{9}

The etiologic factors of this skeletal type of Class III malocclusion have not been fully unraveled, remain incompletely understood, and the regions of the craniofacial complex that are affected by various treatment modalities have not been delineated explicitly. The aims of this review are to rationalize morphologic and etiologic components and to identify areas where further research is needed to fully delineate the basis of MP and its clinical management.

**Maxillofacial Complex in MP**

Cephalometric and geometric morphometric studies\textsuperscript{14–19} have shown that the deformations in subjects with Class III malocclusion may represent a developmental shortening of the palatomaxillary complex and elongation of the mandible anteroposteriorly, which leads to the appearance of a retrognathic midface and prognathic mandibular profile.

MP may be due to a hypoplastic and/or retropositioned maxilla, a greater total length and anterior positioning of the mandible.\textsuperscript{17} Our previous study in adults showed that the position of the maxilla relative to the anterior cranial base showed no significant difference between MP and normal occlusion groups.\textsuperscript{18} Nevertheless, there were significantly shorter palatal and maxillary lengths in the prognathic group. The total length and anterior positioning of the mandible are particularly suitable criteria for the differential diagnostic evaluation of MP, as revealed by discriminant analysis.\textsuperscript{17} The midfacial deformations may represent a developmental diminution of the palatomaxillary complex anteroposteriorly that allies with the vertical shortening of midfacial height anteriorly.\textsuperscript{18} The changes in mandibular morphology noted for this skeletal Class III deformity may represent a developmental elongation of the mandible anteroposteriorly, leading to the appearance of a prognathic facial profile.\textsuperscript{18–20}

**Morphogenetic Basis for MP**

The results of studies of the cranial base in subjects with Class III malocclusion and MP compared with normal occlusion controls showed that the greatest between-group differences occurred in the posterior cranial base region.\textsuperscript{18,21} It was concluded that shortening and angular bending of the cranial base, and a diminished angle between the cranial base and ascending ramus, may be associated with the formation of MP, and with the appearance of a Class III facial morphology of retrognathic midface and/or prognathic mandible.\textsuperscript{18,21} Besides the more acute articular or saddle angle in the cranial base, individuals with MP had a more obtuse frontal-nasal angle, presumably associated with a retrusive midfacial profile.\textsuperscript{21}

The shape of the cranial base appears to be established during fetal development,\textsuperscript{22–24} and remains relatively stable during postnatal growth.\textsuperscript{25,26} Kerr\textsuperscript{27} found that saddle angle was one of the few craniofacial parameters that varied little during the growth period from 5 to 15 years of age. An extensive longitudinal study by Bhatia and Leighton\textsuperscript{28} confirmed such stability in both sexes, although there was wide interindividual variability. A prominent feature of this early growth is progressive flattening of the cranial base during late prenatal development.\textsuperscript{22,23,29,30} Therefore, a Class III cranial base morphology may be established very early in development, possibly at the prenatal stage.\textsuperscript{31} Moreover, a Class III morphology may arise, not because of increased cranial base flexion, but rather because of deficient orthocephalization, or failure of the cranial base to flatten anteroposteriorly. However, cranial base flexure is not the only factor involved in determining malocclusion. Scott\textsuperscript{32,33} suggested that several factors determine or influence the jaw position and, consequently, occlusion in individual cases. The three
principal factors involved are the cranial base angle, the extent to which the mandible and maxilla are moved forward in relation to the cranium, and the amount of surface bone deposition along the facial profile from nasion to menton.\textsuperscript{32,33}

The anterior cranial base provides the template that establishes the horizontal length of the midfacial complex, which is also relatively short in Class III malocclusion.\textsuperscript{34} Therefore, the cranial base has a role in the final positioning of the midface and mandible that could account for the clinical presentation of mandibular protrusion and/or maxillary retrusion in individuals with MP. However, conflicting data in the literature\textsuperscript{35–37} suggest that anterior cranial base length might not play an important role in the pathogenesis of Class III malocclusion. The nasion may be quite variable in its position during growth and, thus, may contribute to the contradictory findings.\textsuperscript{37,38} Another possible explanation for such findings is that the foramen cecum of frontal bone is the anterior anatomic limit of the anterior cranial base,\textsuperscript{39} and the nasion may not be appropriate for characterizing anterior cranial base configuration.

Decreased angulation between the anterior and posterior cranial bases, particularly associated with the articular (Ar), was also noted in studies of adults with MP and children with Class III malocclusion. Thus, anterior displacement of the temporomandibular joint appears to be demonstrable in subjects with Class III malocclusion and MP. The resulting prognathic face, characterized by shortening and angular bending of the cranial base, and a diminished angle between the cranial base and mandibular ramus,\textsuperscript{40} provides an indication of apparent cranial base kyphosis, associated with the appearance of a Class III facial morphology.

Ethnic Variations in Craniofacial Form: Structural Basis of Basicranium for MP

Class III malocclusion shows a relatively low prevalence in Caucasian populations, whereas its prevalence has been observed to be much higher in Asians. Our analysis of local differences in cranial base configuration on cephalographs between European–American and four Asian groups (Taiwanese, Japanese, Korean, Chinese) in young adults with normal Class I molar occlusion, using thin-plate spline and strain tensor graphical analyses,\textsuperscript{41} showed that the greatest differences occurred in the anterior portion of the cranial base and upper midface region, which generally consisted of horizontal compression. The posterior-most cranial base region also showed horizontal compression between the Bolton point (Bo) and Ar with forward compression of the Ar. This represented frontal and facial flatness (the characteristic feature of Asian faces)\textsuperscript{42–44} and anterior displacement of the temporomandibular joint. These findings indicated that the composite result is a relative retrusion of the nasomaxillary complex and a more forward relative placement of the mandible. This results in a greater tendency toward a prognathic mandible and/or retrognathic midface and a Class III type of malocclusion in Asian populations. Therefore, it appears that Class III malocclusions are prevalent to a greater degree in Asian populations and even Class I and II cases may exhibit an underlying Class III character.\textsuperscript{45,46}

Etiologic Factors of MP

Heredity of MP

MP is one of the best-known facial genetic traits or phenotypes. It is also well recognized that ethnicity is a risk factor for MP; the highest incidence has been observed in Asian populations and the lowest in Caucasian populations as previously discussed. The existence of familial aggregation of MP suggests that genetic components play an important role in its etiology. Numerous studies have shown a significantly higher incidence of this phenotype in the relatives of affected probands.\textsuperscript{47–50} In the offspring of affected parents, extensive studies of Japanese families showed a frequency of 31% if the father was affected, 18% if the mother was affected and 40% if both parents were affected.\textsuperscript{47,51} In siblings of affected probands, Litton et al.\textsuperscript{52}
found a frequency of 13% irrespective of sex. Concordance for MP among twin pairs collected from published reports was 81.0–83.3% for monozygotic and 10.0–13.3% for dizygotic twin pairs.\textsuperscript{53,54} However, the inheritance pattern of MP is controversial; findings have been reported suggesting autosomal-recessive inheritance,\textsuperscript{47,55} autosomal-dominant inheritance,\textsuperscript{49,56} dominant inheritance with incomplete penetrance\textsuperscript{18,57,58} or a polygenic model of transmission.\textsuperscript{52}

The Habsburgs, one of Europe’s foremost royal families, are famous not only for the duration of their reign and brilliance of their leadership, but also because they represent one of the few examples of the inheritance patterns of facial characteristics. The term “Habsburg jaw” has been coined to describe the prognathic mandible seen in 23 successive generations of this family.\textsuperscript{57,59,60} Males were more severely affected than females. The facial characteristics of the royal Habsburgs included MP, thickened lower lip, prominent, often misshapen nose, flat malar areas (maxillary hypoplasia) and mildly everted lower eyelids.\textsuperscript{57,59,60} The Habsburgs suffered from various other ailments including asthma, gout, dropsy, epilepsy and melancholia. The early Habsburgs were unfortunate enough to not have been acquainted with the laws of heredity and the consequences of consanguineous marriage or inbreeding.

Although there appears to be a strong familial tendency in the development of MP, the rate of developing MP in patients with a positive family background (49.3%) is not higher than those with a negative one (50.7%).\textsuperscript{61} Therefore, the prevalence of MP may depend on candidate genes being expressed, with gene–environment interactions determining the severity of MP. The relative contributions of genetic and environmental components in the etiology of MP are unclear. Little is known about the interaction between genetic and environmental factors in the causation of MP.

Recent progress in molecular genetics has enabled the genetic determinant to be approached directly. Yamaguchi et al\textsuperscript{62} performed a genome-wide linkage analysis identifying three chromosomal loci (including 1p36, 6q25 and 19p13.2) susceptible to MP with 90 affected sibling pairs from 50 Japanese and 40 Korean populations. The replication of these results with an independent dataset should facilitate the positional cloning of the gene or genes that influence the development of MP. Therefore, large-scale studies of well-defined families, including those of other ethnicities, are warranted to confirm the evidence of linkage to MP.

Environmental factors associated with MP

Various environmental factors have been found to contribute to the development of MP, including congenital anatomic defects (cleft lip–cleft palate),\textsuperscript{63,64} endocrine disturbance (acromegaly, gigantism, pituitary adenomas),\textsuperscript{20,55,63,65,66} nasoairway obstruction (enlarged tonsils),\textsuperscript{20,67} habitual posture (habit of protruding the mandible)\textsuperscript{23,70} and trauma (instrumental deliveries).\textsuperscript{68,69}

Determination of tongue position is important in the diagnosis of certain clinical conditions, such as MP, dentoalveolar crossbite or bialveolar protrusion of teeth. These clinical conditions can be associated with forward tongue position and/or enlarged tonsils.\textsuperscript{20,67} A lower pharyngeal width of more than 15 mm suggests anterior positioning of the tongue, either as a result of habitual posture or due to an enlargement of the tonsils.\textsuperscript{65} The lower pharyngeal width is measured from the intersection of the posterior border of the tongue and the inferior border of the mandible to the closest point on the posterior pharyngeal wall.\textsuperscript{57}

MP may result from pituitary adenoma with acromegaly.\textsuperscript{56} Pituitary adenomas are benign tumors located in the sella turcica and usually associated with hypersecretion of pituitary hormones. One of these hormones is growth hormone (GH). The GH-secreting pituitary adenoma leads to acromegaly, which is a highly disproportionate growth of the mandible and facial bones in postpubertal patients, mainly as a result of reactivation of the subcondylar growth zones and also due to peristomal bone apposition.\textsuperscript{66}

Patients with cleft lip–cleft palate clearly exhibit the underlying potential for midfacial deficiency or skeletal Class III growth as a result of the
original deformity and subsequent multiple operations necessary for its repair. Cleft cases with midfacial deficiency usually underdevelop anteroposteriorly and also vertically, leading the accentuation of the relative mandibular protrusion in maxillary retrognathism or MP.

**Dentofacial Orthopedic Therapy in Growing Class III Children**

Treatment of MP or skeletal Class III malocclusion in growing children remains one of the most challenging problems confronting the practicing orthodontist. A number of treatment protocols have been used to address skeletal Class III cases, including the chin cup, face mask, maxillary protraction combined with chin cup traction and the Fränkel functional regulator III appliance.

The chin cup is recommended in growing patients who have a moderately protrusive mandible and a relatively normal anteroposterior position and maxilla size. While chin cup therapy for MP has been used for a long time, varying levels of success have been reported. Differences in these findings may be causally related to the duration of treatment, level of force utilized in the appliance and/or the age of the patient being treated. This orthopedic appliance is effective for mandibular prognathic patients in late deciduous or early mixed dentition. After resolution of the intermaxillary skeletal imbalance, both the amount of chin cup force and the duration of wear are reduced. The appliance is then used as a retainer for the remainder of the treatment period.

Maxillary protraction is recommended for developing skeletal Class III patients with maxillary deficiency. For a growing patient who suffers from maxillary retrognathia with or without mild MP, maxillary protraction with a face mask is an adequate treatment method. Maxillary protraction therapy is sometimes combined with palatal expansion in the belief that palatal expansion may disrupt the circummaxillary suture system and thus enhance the protraction effect of the face mask. However, controlled prospective randomized clinical trials by Turley and Vaughn et al demonstrated that maxillary protraction therapy with or without palatal expansion produced equivalent changes in the dentofacial complex that combined to improve the skeletal and dental Class III correction. Their results suggest that the indication for palatal expansion should be based on clinical criteria (such as maxillary constriction or space deficiency) other than assisting the Class III correction.

Appliances that combine maxillary protraction and chin cup traction are appropriate for skeletal Class III patients showing both midface deficiency and moderate MP. Combining maxillary protraction therapy for the midface deficiency with the necessary mandibular retraction strategy often produces satisfactory results. Skeletal Class III patients with midface deficiency and MP are often less difficult to treat than patients with MP alone, since some improvement may be obtained in the midface by maxillary protraction, and some in the mandible by chin cup therapy. They may not respond as well if the strategies are focused on one region only.

The ultimate treatment goal for skeletal Class III patients should not only be the correction of the jaw relationship and negative incisal overjet related to mesial occlusion at that stage, but also the stabilization of the intermaxillary skeletal and dental relationships resulting from orthopedic appliance treatment. Thus, close observation and follow-up of midfacial and mandibular growth during adolescence, particularly during the second or third stage of orthodontic treatment, are essential.

Using dried human skulls with strain gauges, Omatsu and Kawamoto investigated the effects of a chin cup on the pediatric craniofacial skeleton. This included consideration of different directions of traction force imparted by the chin cup. They found that when the direction of traction was 20° more vertical than the chin–condyle line, i.e. through the occlusal surfaces of the molars, the vertical-pull chin cup (VPCC) produced strong vertical compression stress on the maxillary molars.
suggestive of induction of counterclockwise rotation of the mandible. Therefore, this treatment strategy may prevent the relapse of treated skeletal Class III malocclusion. The effects of orthopedic force produced by VPCC were correlated with the cephalometric and histologic observations during use of this appliance in animal studies.\textsuperscript{83, 84}

**Orthognathic Surgery Combined with Orthodontic Therapy in Adult MP**

Facial growth modification can be an effective method of resolving skeletal Class III jaw discrepancies with dentofacial orthopedic appliances. Nevertheless, continued growth in early adulthood may detract from treatment results obtained in childhood or adolescence. Orthognathic surgery in conjunction with orthodontic treatment would be required for correction of this problem in adults.\textsuperscript{85}

Skeletal Class III patients make up a considerable percentage of the orthognathic surgery population. A number of surgical techniques can be used for the treatment of maxillofacial deformities. According to the location of the sagittal jaw problem in adult skeletal Class III cases, orthognathic surgical treatment is accomplished by a mandibular setback, a maxillary advancement or a combination of the two. Several methods have been proposed for surgical correction of MP. The two most commonly applied surgical procedures to correct MP are sagittal split ramus osteotomy (SSRO)\textsuperscript{86, 87} and intraoral vertical ramus osteotomy.\textsuperscript{88-90} Both are suitable for patients in whom a desirable occlusal relationship can be obtained with setback of the mandible, each having its own advantages.

Dentoalveolar compensation is characteristic of skeletal Class III malocclusion. In prognathic patients, dentoalveolar compensation is common in both maxillary and mandibular arches. Prognathic patients compensate for the intermaxillary skeletal dysplasia during mandibular protrusion by lingual tipping of the mandibular incisors with alveolar process. The lower incisors and alveolar retroinclination may result from a restraining effect of the orbicularis oris musculature on the crowns as the roots are carried forward by the prognathic mandible.\textsuperscript{4} In contrast, the upper incisors and upper alveolar process in prognathic adults are more proclined compared with normal controls. The upper incisors with alveolar process may be tipped labially by the tongue while the mandible is prognathic.\textsuperscript{4} Such compensatory dentoalveolar changes should be eliminated from both arches during orthodontic treatment presurgically or postsurgically.\textsuperscript{91} Orthodontic preparation can eliminate dentoalveolar compensations by aligning the teeth over the basal bone to allow maximal repositioning of the mandible.

The diagnosis and planning of treatment for patients with these maxillofacial deformities can be complex and challenging. It is important to determine the quantity of necessary surgical correction of the prognathic mandible and/or retrognathic midface. This will allow design of the desired correction possible by an orthodontic preparation. Considering the needs of each patient, the therapeutic goal is to eliminate dentoalveolar compensation. An orthodontic preparation with dentoalveolar decompenstation will allow an increase in the quantity of surgical correction, making better functional and aesthetic results possible.

Relapse after maxillofacial surgery for the correction of jaw deformities is distressing to both clinicians and patients. The muscular factor is regarded as being most important in postoperative relapse following mandibular setback.\textsuperscript{92} The most reliable fixation method to reduce postoperative relapse is rigid fixation because it provides stability of the postoperative position and reduces relapse.\textsuperscript{93, 94} The other consideration is to reduce muscular force, since postoperative relapse could be exacerbated by tension in the pterygomasseteric sling\textsuperscript{95} or by postoperative contracture of the operated soft tissue and muscles.\textsuperscript{96} Additional methods for preventing postoperative relapse were considered with glossectomy and condylar positioning. In Class III cases, when the condyles are displaced inferoanteriorly, the condyles tend to return into
the fossa, which thereby reduces the likelihood of postoperative relapse.97,98

Rigid fixation has been recognized as a successful method for preventing relapse, although applying the distal ostectomy technique (intentional ostectomy of the posterior part of the distal segment) in addition to bilateral SSRO is even more effective as it offers even better long-term positional stability.98 This may be due to the reduction of tension in the pterygomasseteric sling that applies force in the posterior mandible.

For the practicing orthodontist and oral maxillofacial surgeon, modern molecular techniques may appear to be of little relevance. However, MP and cleft lip–cleft palate have a genetic background. It is important for clinicians to be well informed of the molecular background of these conditions, such as distraction osteogenesis and bone grafting.

Conclusion

Further research into the growth changes and/or treatment effects of the VPCC with the direction of traction through the occlusal surfaces of maxillary and mandibular molars on a large number of patients with skeletal Class III malocclusion is needed.

Newer techniques of patient management such as distraction osteogenesis of the maxillary complex and orthodontic implant therapy99 require rigorous investigation and assessment. Therapies that are targeted at regional dysmorphoses may produce a more stable posttreatment outcome.

One good example is distraction osteogenesis, which, as a mainstay in bone engineering, has significantly improved reconstructive maxillofacial procedures. However, the molecular mechanisms governing the formation of new bone in the intersegmental gap of gradually distracted bone segments remain largely unclear. It has been recently implicated that a growing number of cytokines (i.e. growth factors) are intimately involved in the regulation of bone synthesis and turnover. The gene regulation of these numerous cytokines during distraction osteogenesis has yet to be characterized. This will ultimately guide the development of targeted strategies designed to accelerate bone healing.

Future work will employ molecular genetics to identify candidate genes within the human genome to predict those individuals most likely to develop MP. Further studies in molecular biology are needed to disclose the gene–environment interactions associated with the phenotypic diversity of MP and the heterogenic developmental mechanisms thought to be responsible for them. Identification of candidate genes will permit early clinical diagnosis and intervention, as the growing craniofacial complex may be amenable to prophylactic treatments. Identification of the susceptible genes in the linkage regions will pave the way for insights into the molecular pathways that cause MP, especially overgrowth of the mandible, and may lead to the development of novel therapeutic tools.

References


51. Schulze C, Wiese W. On the heredity of prognathism. Fortschr Kieferorthop 1965;26:213–29. [In German]