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Original Article

Bimaxillary expansion therapy for pediatric sleep-disordered breathing

Stacey D. Quo [a], Nelly Hyunh [b], Christian Guilleminault [c, *]

[a] School of Dentistry, University of California at San Francisco, San Francisco, CA, USA
[b] Faculty of Dentistry, Université de Montréal, Montréal, Canada
[c] Sleep Medicine Division, Stanford University School of Medicine, Stanford, CA, USA

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ABSTRACT

Introduction: The aim of this retrospective study was to evaluate the results of bimaxillary expansion as a treatment option for pediatric sleep-disordered breathing.
Methods: Forty-five children, aged 3–14 years, with sleep-disordered breathing underwent bimaxillary expansion. They were subjected to baseline clinical evaluations, cephalometric X-rays, and polygraphic sleep studies. Three to six months after bimaxillary expansion, posttreatment sleep studies were performed. Data were analyzed with nonparametric Wilcoxon signed-rank test, and Spearman's correlations were performed to correlate cephalometric facial structures to the effectiveness of treatment.
Results: The majority of the children ($n = 30$) showed improvement in their sleep scores and symptoms after bimaxillary expansion. The initial severity of the obstructive sleep apnea (OSA) indicated by the apnea–hypopnea index (AHI) was a much better predictor of positive results. However, in the "mild OSA" group, patients with smaller MP–SN or counterclockwise mandibular growth, worsened with bimaxillary expansion, while patients with clockwise mandibular growth showed greater improvement; in the "severe OSA" group, patients who initially had shorter mandibular base lengths showed lesser AHI improvements.
Conclusions: Bimaxillary expansion can be a treatment option for improving respiratory parameters in children with sleep-disordered breathing. This study also suggests that retrognathia in an anterior growth rotation pattern may not respond to efforts of bimaxillary expansion.

1. Introduction

Obstructive sleep apnea syndrome (OSAS) is the most common sleep-disordered breathing (SDB) abnormality. It is characterized by the abnormal collapse of the upper airway pharyngeal muscles during sleep resulting in sleep disruption [1–3]. In both children and adults, this abnormal collapsibility has been related to the state of sleep itself and intrinsic and extrinsic governing factors. During sleep, the pharyngeal muscle tone and reflex responses are modified, rendering the airway more collapsible. The upper airway has an intrinsic collapsibility that may be characterized by the "critical closing pressure," but extrinsic factors may lead to increased collapsibility. The three external factors that are firmly established to affect the upper airway space are the nasal cavity, and the retropalatal and the retroglossal upper airway space.

Obstructions that affect the mode of breathing can affect the width, length, and height of the maxillomandibular complex with a subsequent impact on the nasal cavity, and retropalatal and retroglossal upper airway space, as shown in both children and experimental animals [4–8]. Craniofacial alterations of a narrowed maxilla, altered tongue position, and narrowed dentition have been described in numerous studies [9–13]. Concomitant changes in the lower jaw have been cited as an altered mandibular posture and clockwise rotation. Similar to the narrowing of the maxilla, narrowing of the mandibular width has been shown [14,15]. Maxillary narrowing has often been described and treated using rapid maxillary expansion (RME), but the mandibular narrowing is seldom mentioned and thus not treated.

RME was first suggested as a therapy for adult OSA in 1998, to address maxillary width alterations [16]. It was based on the previous description of efficacy of RME on other medical conditions

* Corresponding author. Sleep Medicine Division, Stanford University School of Medicine, 450 Broadway St, Pavilion C 2nd Fl MC 5704, Redwood City, CA 94063, USA. Fax: (650) 721 3448.
E-mail address: cguil@stanford.edu (C. Guilleminault).

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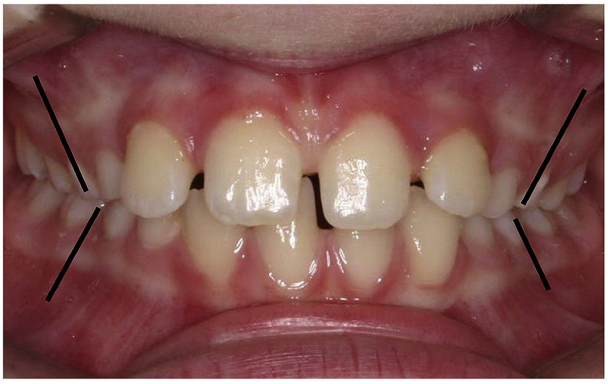

Fig. 1. Depiction of inward lingual tipping of the upper and lower dentition. Legend: Black lines indicate the lingual inclination of the upper and lower teeth.

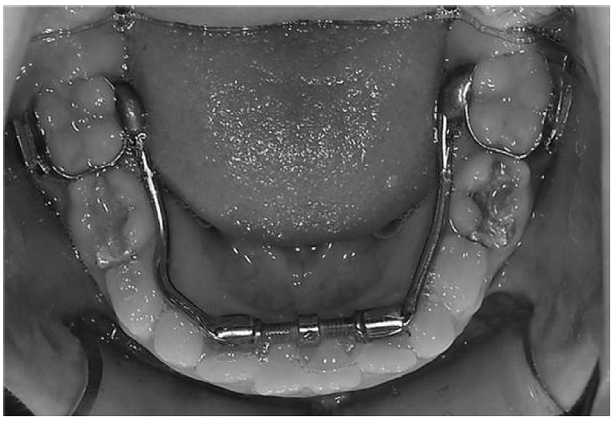

Fig. 2. Lower expander anchored to the permanent lower first molars.

such as enuresis, diaphoresis, allergies, and asthma as early as 1974 [17]. In children, the peak onset of symptoms occurs between two and eight years of age [3]. The long-term health risks of untreated sleep apnea in the pediatric population parallel the same risks as the adult population. The emphasis in effectively treating this disease is on early detection and early treatment. In 2004, Pirelli et al. [18] published the positive impact of RME on a group of OSA children with narrow maxillas and no adenotonsillar hypertrophy, with a recent follow-up study demonstrating treatment stability after a 12-year follow-up [19]. Other RME published studies also describe the variable effects of maxillary expansion in treating children with narrow palates and/or retrognathic mandibles [18,20–23].

Skeletal maxillary expansion using RME can be achieved by applying tension across the intermaxillary or midpalatal synchondrosis/suture. The maxilla can be enlarged in width, while this suture is patent in growing children. Unlike the maxilla, the mandible as one piece of endochondral bone without sutures is not subject to skeletal width expansion, but instead it will only yield dentoalveolar expansion. The skeletal widths (or archforms) of the jaws can be quantified with radiography, but it can also be assessed visually on intraoral examination by the lingual inclination of the maxillary and mandibular dentition (Fig. 1), and on dental casts by the reduced intermolar distance [24].

The aim of this retrospective study differs from all previous maxillary expansion work by assessing the effect of bimaxillary expansion (BE) treatment on 45 children with varying facial types or jaw morphologies who were diagnosed with OSA. Because OSA children often present with variable growth disturbances involving both maxilla and mandible, BE was used as a strategy to address the bimaxillary width distortions that can result from abnormal breathing. The effectiveness of BE as a treatment option for pediatric SDB was first described a decade ago [25], and it refers to skeletal width expansion in the upper jaw and dentoalveolar width expansion in the lower jaw.

2. Materials and methods

2.1. Patient selection

The data were obtained retrospectively from one orthodontic office from children who were diagnosed with OSA and treated with BE between 2001 and 2011. The inclusion criteria were as follows: (1) diagnosis of SDB before the initiation of any orthodontic treatment, (2) treatment with BE without any cotherapy, and (3) completeness of data in the chart (including polysomnography (PSG) reports and lateral cephalograms). The exclusion criteria were as follows: (1) any concurrent soft-tissue surgery (adenoidectomy, tonsillectomy, and turbinectomy), (2) initial and posttreatment PSG studies performed under the control of different certified specialists and sleep laboratories, and (3) absence of syndromic craniofacial anomalies. Forty-five patients' charts satisfied the inclusion and exclusion criteria, and these charts were analyzed.

With a mean age of 7.58 ± 2.82 (range 3–14 years), 45 children (32 boys and 13 girls) reported symptoms of SDB and had undergone a type 1 PSG. Five children with associated neurological syndromes not clinically affecting their overall facial growth (Tourette's syndrome, autism and fetal alcohol syndrome, and cerebral palsy) were also included in this retrospective review. These data were rendered anonymous as requested by the institutional review board (IRB). No sample size calculation was performed, as this is the first study to assess the effect of BE on SDB in children.

2.2. Orthodontic procedures

Fixed bimaxillary screw-activated expansion devices were anchored on the dentition. The upper appliances were the RME type for skeletal expansion, and the lower appliances were placed for dental uprighting and tooth expansion. For patients in the permanent dentition group, the upper appliance was attached to the first bicuspids and the first molars, while the lower appliance was attached to the lower first molars (Fig. 2). For patients in the mixed dentition group, if the upper primary second molars were present, then the appliance was attached to these two teeth on the upper jaw (Fig. 3). If the lower first molars had not erupted,

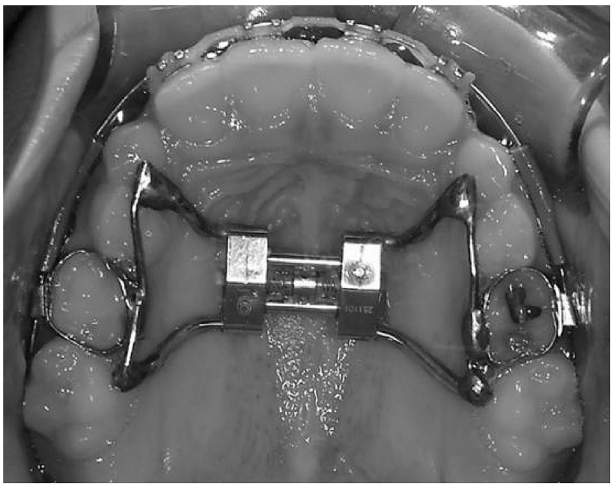

Fig. 3. Design of upper expander in the mixed dentition.