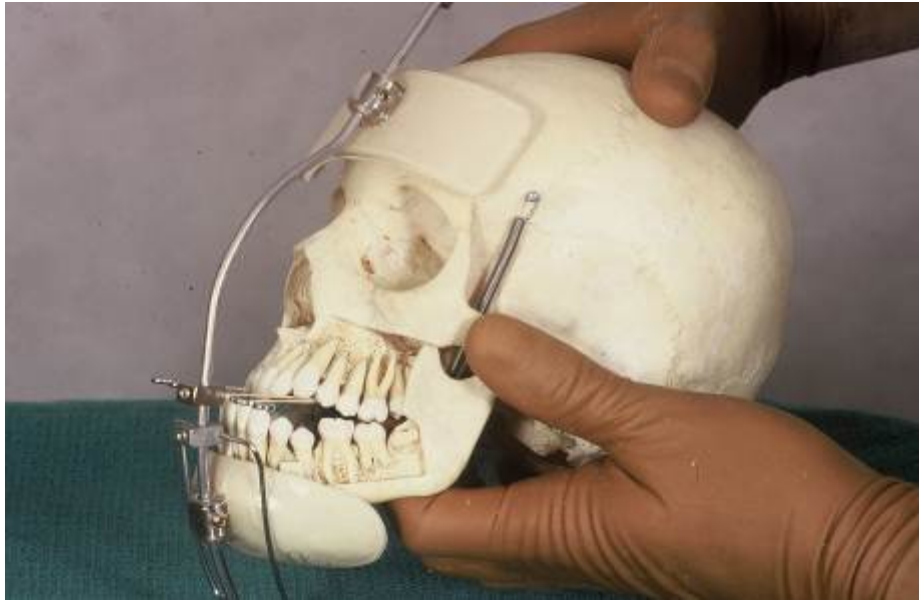


## **PART 1**

### **First in a Series of Six Articles Dealing with** **Airway and Dentofacial Development In Children**



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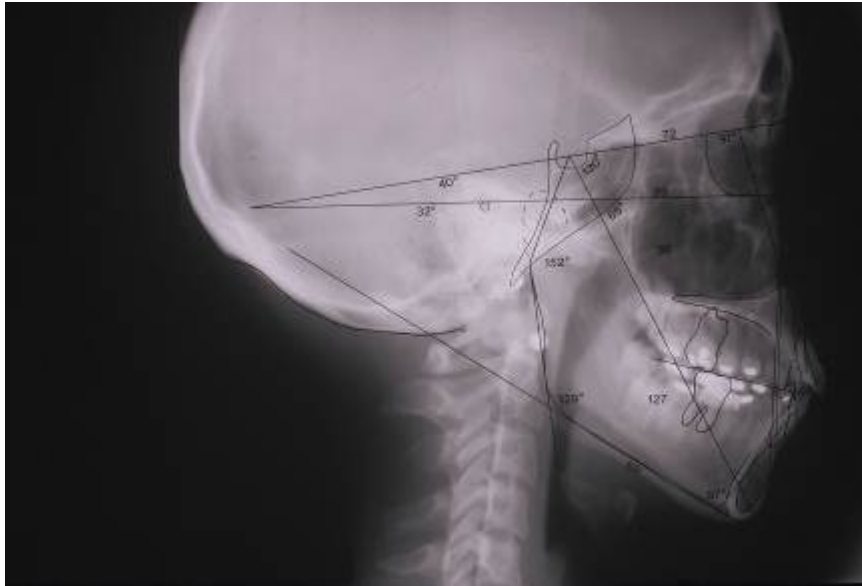
**(Diplomate International Board of Orthodontics)**

Like so many things in life, timing is everything. Parents, family physicians, pediatricians, family dentists, pediatric dentists, ear nose and throat surgeons, friends, family and, especially orthodontists find themselves, in a unique situation where they can change a child's quality of life, literally for the rest of that child's life. Mouth breathing, snoring, excessive daytime sleepiness, obstructive sleep apnea, esthetically displeasing facial features, narrow dental arches, need for future jaw surgery, recurring nasal and sinus infections, sinus pressure headaches, dry raw pharynx, post nasal drip and life long nasal obstruction are some of the quality of life issues that a child or teenager, with a developing long face syndrome, will have to deal with for the rest of his adult life. Especially if the developing long face syndrome is not intercepted.

The child, who has a developing long face syndrome, can have that pathological process intercepted through the use of some unusually simple procedures. Tonsillectomy and adenoidectomy,

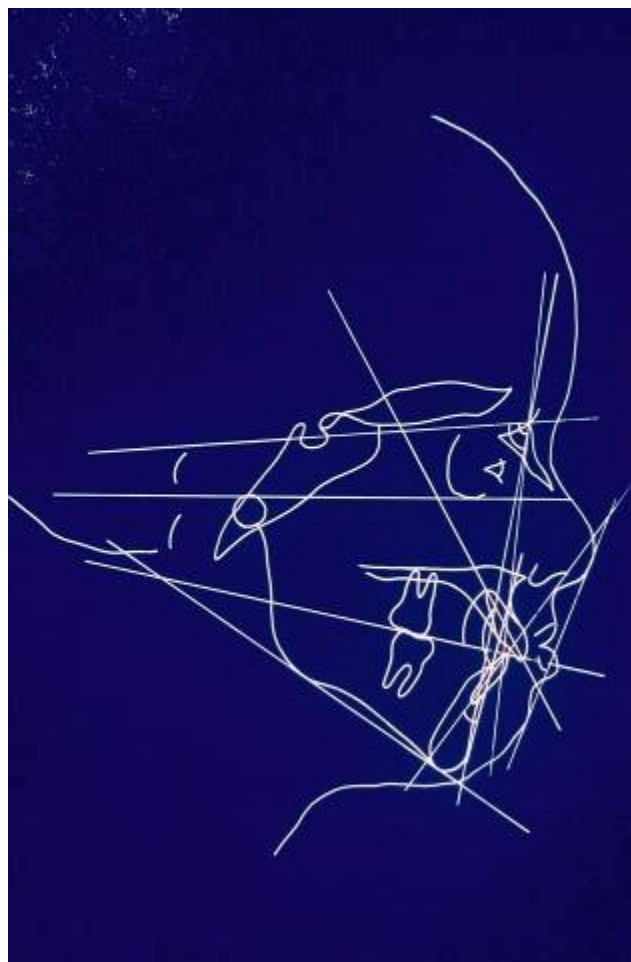
partial resection of the inferior turbinates, maxillary expansion, upper lateral cartilage lateral (alar) rotation and use of a Vertical Chin Cup are some of the simple procedures that can, when done in a timely manner, be utilised to intercept the developing long face syndrome. And at the same time we can greatly improve that child's quality of life.

Unfortunately current internet websites, as well as the government run pubmed.com, offer little in depth information re: the diagnosis of the developing long face syndrome, and virtually nothing regarding interceptive treatment of the developing long face syndrome. However, the research is definitely out there. A few days spent in a good medical school, or dental school library, under the direction of an experienced librarian, can rectify this. Then the clinician needs to find knowledgeable practitioners who can implement the same, and your paediatric patients will thank you for the rest of their life.



**Figure 1.**

**(Lateral cephalometric radiograph of a high angle patient)**



**Figure 2.**

**(Note the prominent antegonial notch on the lower border of the mandible)**

## **Effects of Nasal Airway Obstruction on Facial Growth**

In 1872, CV Tomes<sup>1</sup> described the dentofacial changes associated with nasal airway blockage. He used the term “adenoid facies” because he believed enlarged adenoids were the principle cause of the obstruction. Over one hundred years later, numerous reports concerning this observation have appeared in the dental and medical literature, but the issue still remains controversial.

In 1982, O’Ryan et al<sup>2</sup> critically reviewed the possible relationship between nasorespiratory function and dentofacial morphology, and concluded that they were unable to demonstrate a consistent relationship between obstructed nasorespiratory function, and the adenoid facies in a “long-face” syndrome.

We have attempted to review the available evidence on both sides of this question and propose an aetiologic rationale for the findings. We will also describe a cooperative protocol between primary care physicians and dentists, allergists, oral myologists, otorhinolaryngologists (ENT doctors), and orthodontists for the management of young patients with increased nasal airway resistance.

### **Literature Review**

The initial views of Tomes were later supported by many leading orthodontists, in the 1930s, including Todd et al<sup>3</sup> and Balyeat and Bowen<sup>4</sup>. Angle<sup>5</sup> included airway obstruction as an important aetiologic agent in malocclusion and Ketcham<sup>6</sup> indicated that patients were not receiving the full benefits of medical and dental therapy unless they were fully evaluated by both a rhinologist and an orthodontist. McCoy<sup>7</sup> regarded nasopharyngeal obstruction as an important cause of malocclusion, noting an increase in Class III malocclusion, in his sample with an open mouth posture, and large tonsils.

Moss<sup>8</sup>, in developing a “functional matrix” theory (originally proposed by van der Klaauw<sup>9</sup>), presented a logical rationale for the findings seen in nasally obstructive patients. His view held that bone responded to the influences of function, and adjoining soft tissue. This explained the narrow palate, and long face, seen in some chronic mouth-breathers. In contrast, Hawkins<sup>10</sup>, Howard<sup>11</sup>, and Leech<sup>12</sup> found no relationship between malocclusion and mouth breathing. However, a serious flaw in their studies was the use of Angle’s horizontal classification system, to incorrectly assess vertical dentofacial dysplasias.

In the 1950s, Subtelny<sup>13</sup> and Ricketts<sup>14</sup> examined the effect of nasal airway on facial growth, and concluded that airway obstruction had an important influence on facial form. In recent years, reports by Linder-Aronson and Woodside,<sup>15</sup> Quinn,<sup>16</sup> Rubin,<sup>17</sup> McNamara,<sup>18</sup> Bushey,<sup>19</sup> and Harvold<sup>21</sup> concluded that objective measurements have, in fact, substantiated the finding of chronic mouth-breathing as a casual factor in orthodontic anomalies.

In 1968, Ricketts<sup>22</sup> used the term respiratory obstructive syndrome to describe a constellation of findings, seen in chronic mouth breathers.

Subtelny<sup>23</sup>, in 1974, also indicated that adenotonsillar hypertrophy (marked enlargement of the tonsils and adenoids) could influence facial growth.

Marks<sup>24</sup> studied the role of allergy in orofacial deformities and concluded that nasal obstruction was a significant cause of altered facial growth.

Similar findings were reported by Shapiro and Shapiro<sup>25</sup>. Quinn<sup>16</sup> has cited nasal airway obstruction as the major cause of mandibular prognathism (forward projecting lower jaw), facial asymmetries, and vertical dysplasias.

Linder-Aronson<sup>26</sup> also was able to show that a group of post adenoidectomy patients, who became nasal breathers, had significant craniofacial changes, toward normal. Conversely, persistent mouth-breathers, and the un-operated controls, showed no changes. Harvold<sup>20</sup> has shown skeletal changes in primates, secondary to experimentally produced nasal blockage. After removal of the obstruction, changes toward normality became apparent.

Hannuksela<sup>27</sup> compared 39 Finnish children, with moderate or severe allergic disease, with a control group. She found significantly steeper mandibular (lower jaw) plane angles in the allergic group. This group included children with allergic dermatitis, bronchial asthma, and allergic



rhinitis. Hannuksela noted that children with documented adenoidal hypertrophies (on lateral headplates), had steeper mandibular plane angles.

Recently, Long and McNamara<sup>28</sup> reported 17 cases that developed increased lower facial height following palatopharyngoplasty (flap at the back end of the soft palate used to correct hypernasal voice ). They concluded that the pharyngeal flap procedure increased nasal airway resistance.

Disagreeing with this etiologic contention, Vig<sup>29</sup> stated that, in the absence of documented total nasal obstruction, surgical or other treatment to “improve” nasal respiration remains purely empirical, and difficult to justify from an orthodontic viewpoint. O’Ryan et al<sup>2</sup> reviewed the available literature and found no support for the contention that mouth breathing is a major aetiologic factor in the development of the long-face syndrome (LFS).

Although the primate experiments performed by Harvold obviously cannot be performed on human subjects, technologic advances have permitted the development of sophisticated devices to simultaneously measure nasal and oral resistances. Prospective studies, as performed by

Linder-Aronson,<sup>25</sup> are still necessary for a more objective analysis of mode of respiration, and its influences on facial growth. Establishing a cause-and-effect relationship, between nasorespiratory function, and dentofacial development, is not simple. A dolichocephalic (long narrow face) pattern may be conducive to mouth-breathing, rather than mouth-breathing causing a dolichocephalic appearance. The studies by Shapiro and Shapiro,<sup>26</sup> and Hanuksela,<sup>27</sup> on patients with (nasal) allergy avoid this enigma.

### **Normal Nasal Respiration**

The nose filters, warms, and humidifies the air in preparation for entry into the bronchi and lungs. The functioning nasal airway may also create a certain degree of nasal resistance to facilitate the movements of the diaphragm, and intercostal muscles, in creating negative intrathoracic pressure that, in turn, promotes airflow into the alveoli.<sup>30</sup> (final branchings of the respiratory tree...the primary gas exchange units of the lungs). Appropriate nasal resistance is 2 to 3.5 cm H<sub>2</sub>O/L/sec and produces high tracheobronchial airflow, which improves the oxygenation of the most peripheral pulmonary alveoli. Mouth breathing results in a lower velocity of incoming air, and also eliminates nasal resistance. Suboptimal pulmonary

compliance (the ability of the lungs to stretch in a change in volume relative to an applied change in pressure) is the result. Blood gas studies have revealed that advanced mouth-breathers have 20 percent higher partial pressures of carbon dioxide, and 20 percent lower partial pressures of oxygen in the blood, associated with their lower pulmonary compliance and reduced velocity.<sup>31</sup>

Obstructive sleep apnea is regarded as a complication of nasal and oropharyngeal obstruction. Another less common complication of upper airway compromise is functional pectus excavatum.

Upper airway compromise can also cause cor pulmonale as the result of pulmonary hypertension with associated right ventricular hypertrophy.<sup>32</sup>

### **History and Physical Examination**

The patient should be observed, as he enters the examining room, and sits in the chair. The facial posture should be noted to see if the lips are closed during respiration. Allergic “salutes” and “shiners” are seen commonly in patients experiencing allergic rhinitis. The patient may also give a history of frequent “colds” or “sinus.” A family history for allergy is

likewise important. If either parent or a sibling has an allergic history, there is a 40 percent chance that the patient is allergic.<sup>33</sup> One parent may have eczema alone and then transmit the allergic tendency in the form of allergic rhinitis.

Any history consistent with obstructive sleep apnea or loud snoring should be explored, in detail, and parents should be asked about the sleep patterns of their children. An open mouth posture, while sleeping, may be a supporting sign.

The child should be asked to seal his lips. It should be noted if the child has difficulty breathing through the nose. One nostril can be occluded and the response noted. The same procedure is followed for the other nostril. The one-to-four hour nasal cycle results in the inferior turbinate on one side being engorged for a time, followed by engorgement of the other side. This produces increased nasal resistance in one side of the nasal vault at a time.<sup>34</sup> Patients with septal deviations may be totally obstructed when the nasal cycle occludes the contralateral side.

### Treatment of Nasal Obstruction

Adenoidectomy, with or without tonsillectomy, is indicated if enlarged adenoids (and tonsils) are the cause of upper airway obstruction. The lateral cephalometric x-ray, of the head, provides an excellent view of any adenoid tissue in the epipharynx.



**Figure 3.**

**60% adenoid obstruction**



**Figure 4.**

**80% adenoid obstruction**

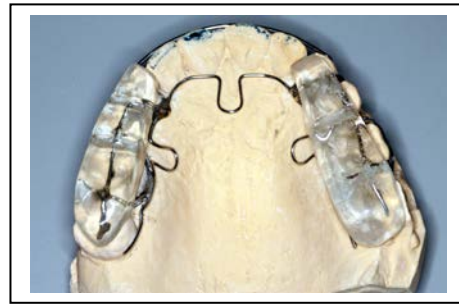
Septal surgery is rarely indicated in the child, but should be considered in the presence of a marked nasal septal deflection with impaction. Cottle,<sup>35</sup>, Jennes,<sup>36</sup>, and Farrior and Connolly<sup>37</sup> have demonstrated that conservative septal surgery, in growing patients, will not have an adverse effect on growth of the boney and cartilaginous nasal vault.

Rapid or semi-rapid maxillary expansion (RME), an orthodontic procedure,<sup>38</sup> is effective in improving the airway by widening the nasal vault.



**Figure 5.**

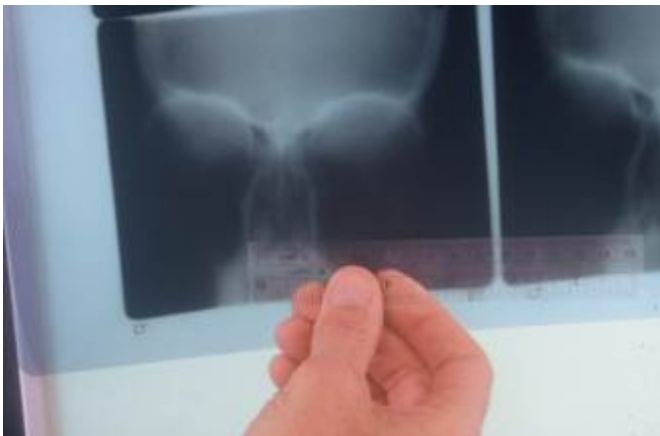
**Banded hyrax**



**Figure 6.**

**Bonded hyrax**

Objective improvement, in the cross-sectional area of the nasal vault, can be documented by pre- and post-expansion PA tomograms or CAT scans.



**Figure 7.**

**(PA tomogram)**



**Figure 8.**

**(CT scan)**

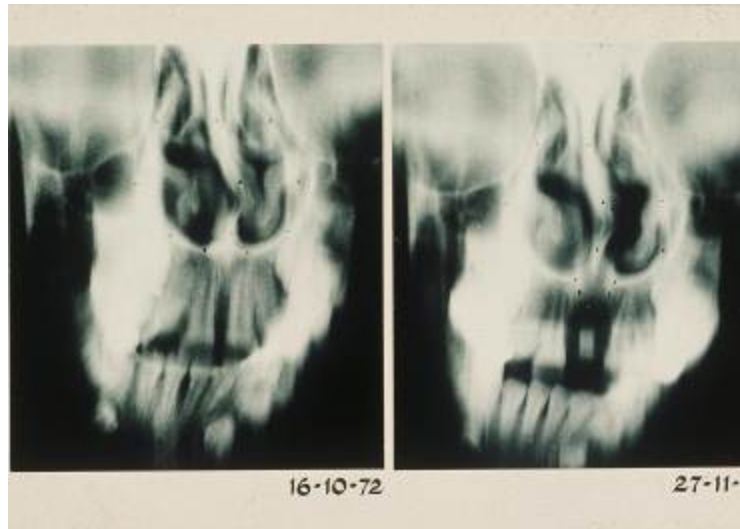
Rhinometric data has supported the efficiency of maxillary expansion in treating nasal obstruction in a child, with a narrow maxilla. In our experience, nonsurgical expansion can be performed between the ages of 3 and 19 years. The rate of expansion is reduced in the older individual/patient.



**Figure 9.**

**(Lateral movement of the inferior turbinates)**

The inferior turbinates move laterally, as the maxillary expansion device expands the maxilla, over a period of 3 weeks. Accordingly, the cross sectional area of the nasal vault is significantly improved.



**Figure 10.**

**(Improvement in septal deviation post maxillary expansion therapy)**

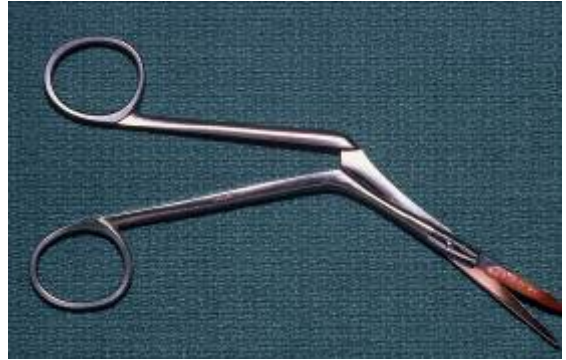
The septum straightens and the cross sectional area of the nasal vault increases, over a three week period of time, as the maxilla is expanded by the maxillary expansion device.

The conditions of patients with vasomotor rhinitis can be improved with cryosurgery or electrosurgery.<sup>39</sup> Chronically enlarged inferior turbinates can be reduced by electrocoagulation,<sup>40,41</sup> or by partial resection.<sup>42-46</sup>





**Figure 11.**



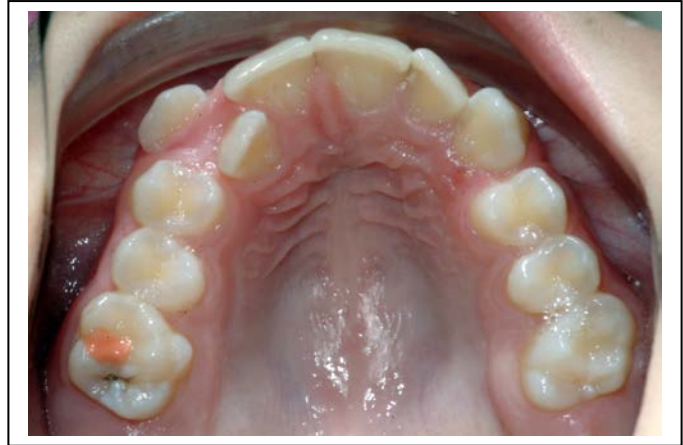
**Figure 12.**

**(Partial resection of enlarged inferior turbinates)**

When the cause of nasal obstruction is allergic rhinitis (with associated hypertrophied tonsils, adenoids and inferior turbinates), a systematic programme of treatment is indicated. In these patients, adenoidectomy is frequently considered; however, removal of the adenoidal pad alone, in cases of untreated nasal allergy, will yield disappointing results because the anterior nasal vault will remain obstructed from inferior turbinate hypertrophy.

Long-standing nasal airway obstruction can lead to a “disuse” atrophy of the lateral crus of the lower lateral cartilage.<sup>30</sup>.

The result is a slit-like external nares associated with a narrow nasal vault and a constricted upper dental arch.



**Figure 13.**

**(Narrow nares and a high palatal vault)**

**(Photo of High Palate)**

After the airway obstruction is corrected, and a normal nasal airway is established, certain patients may still experience nasal collapse on inspiration. These patients could benefit from reconstructive surgery and/or alar dilators.

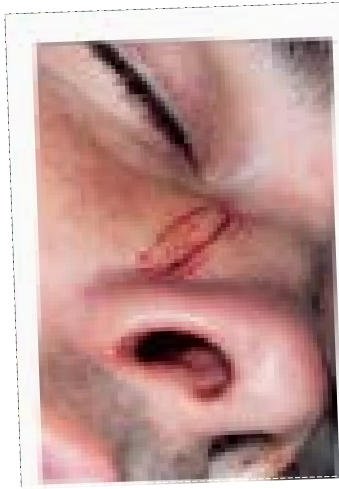
**Note:** Excising an ellipse of skin, and subcutaneous tissue, in the nasofacial fold, in these cases, can **open the nasal valve by rotating the upper lateral cartilage laterally**. The nasofacial fold is highly vascular.

It is recommended that infiltration of ½ % Xylocaine with epinephrine 1:200,000 is placed both superficially, as well as down onto the periosetium, of the nasal bone, and ascending process of the maxilla. Then wait a full 7 minutes (while doing some other part of the operative sequence). Return to the nasaofacial fold, excise a long ellipse of skin, subcutaneous tissue and fascia. Then immediately place a Weck Cell Sponge soaked in 2% Xylocaine with epinephrine 1:5,000 for topical anesthesia and hemostasis, color coded with methylene blue to prevent inadvertent injection, in the wound. And replace the same, several times, as the sponge becomes saturated with blood. Next apply suction to the Weck Cell Sponge and then lightly electrofulgurate specific bleeding points (fourth photograph). Finally do a two layered closure, and apply an ice pack.



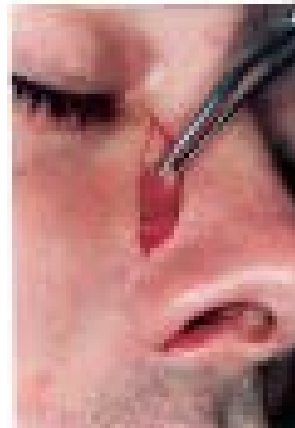
**Figure 14.**

**Infiltration injection**



**Figure 15.**

**Excise skin,  
subcutaneous tissue and fascia**



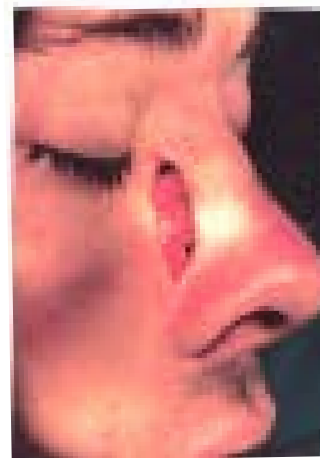
**Figure 16.**

**Placement of a Weck  
Cell Sponge**



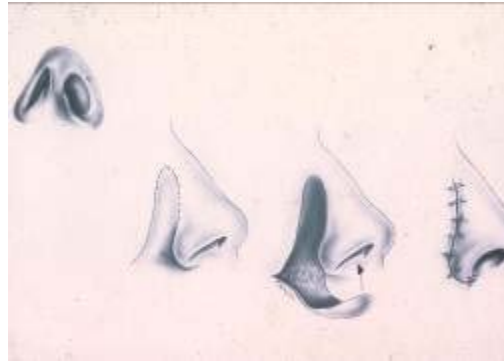
**Figure 17.**

**Two layered closure**



**Figure 18.**

**Apply ice pack**



**Figure 19.**

**Variation for advanced alar collapse ...employing a laterally based nasofacial fold interposition flap...lower right**

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