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REVIEW ARTICLE

# Is alveolar cleft reconstruction still controversial? (Review of literature)

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**Abstract** Cleft lip and palate (CL/P) is a frequent congenital malformation that manifests in several varieties including unilateral or bilateral and complete or incomplete. Alveolar cleft reconstruction remains controversial with regard to timing, graft materials, surgical techniques, and methods of evaluation. Many studies have been conducted addressing these points to develop an acceptable universal protocol for managing CL/P. The primary goal of alveolar cleft reconstruction in CL/P patients is to provide a bony bridge at the cleft site that allows maxillary arch continuity, oronasal fistula repair, eruption of the permanent dentition into the newly formed bone, enhances nasal symmetry through providing alar base support, orthodontic movement and placement of osseointegrated implants when indicated. Other goals include improving speech, improvement of periodontal conditions, establishing better oral hygiene, and limiting growth disturbances. In order to rehabilitate oral function in CL/P patients alveolar bone grafting is necessary. Secondary bone grafting is the most widely accepted method for treating alveolar clefts. Autogenous bone graft is the primary source for reconstructing alveolar cleft defects and is currently the preferred grafting material.


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## 1. Introduction

Fifty years ago, the procedures for grafting bone were inconvenient, sporadically used, and lacked clear objectives. Alveolar cleft reconstruction has been one of the most controversial surgical procedures since it was the first at the beginning of the 20th century. There were multiple philosophies and preferred treatment modalities regarding each step in alveolar cleft management including grafting, the most appropriate age, the ideal material, and whether adjunctive procedures such as orthodontic expansion should be used before or after grafting (Horswell and Henderson, 2003).

Cleft lip and/or palate (CL/P) is considered the most prevalent of the common human congenital craniofacial birth defects. The approximate incidence ratio of CL/P has been reported as 1:700 live births. In addition, CL/P is the second most common congenital malformation following clubfoot (Peter and Larsen, 2004).

## 2. Pathogenesis

Cleft palate deformities occur when fusion of palatal shelves fails to occur. These deformities are classified according to the extent of the palatal involvement. Failure of the primary and secondary palate to fuse leads to complete cleft palate, in which the palatal shelves also fail to fuse. Complete palatal clefts are typically associated with uni- or bilateral cleft lip (Sadove et al., 2004). When the facial processes or palatine shelves do not fuse, incomplete palatal clefts might occur which could either affect the primary or secondary palate. Consequently, the incomplete cleft palate can involve only the posterior part of the soft palate, it may extend through the hard palate to the incisive foramen, or it could be confined to the primary palate resulting in alveolar cleft (Jennifer et al., 2007).

CL/P are more often unilateral than bilateral and more common in males than in females. Unilateral defects on the left side occur more often than on the right side. Cleft palate is more common in females and most often associated with other developmental anomalies. Depending on the existence of associated developmental anomalies, CL/P may be classified as syndromic or isolated anomalies (Hagberg et al., 1998). Isolated CL/P is a complex trait that usually results from a combination of hereditary and environmental etiological factors. Previous research to identify the etiological genes and loci responsible for CL/P has suggested that there may be anywhere from 3 to 14 genes involved (Cobourne, 2004). For isolated CL/P, candidate genes and loci have been identified on chromosomes 1, 2, 4, 6, 11, 14, 17and 19 (Blanton et al., 2004).

## 3. Etiology

Environmental factors that contribute to the etiology of facial clefting disorders can be divided into four groups: drugs, chemicals, maternal metabolic imbalances (as folic acid deficiency), and maternal infections. Maternal exposure to alcohol and teratogenic medications such as retinoids, corticosteroids, and anti-convulsants (phenytoin and valproic acid), and folic acid deficiency during the periconceptional period can cause clefting disorders. Consanguineous marriages, maternal diabetes, and obesity have also been linked to an increased risk of orofacial clefts (Eppley et al., 2005).

The embryo undergoes rapid changes in shape and growth between 4 and 8 weeks as the brain expands and the six branchial arches are formed. The first two branchial arches are primarily responsible for the development of the face and the cranium. The development of the face begins from the ectomesenchyme of the neural crest, which forms five prominences: the frontonasal process and two maxillary and mandibular processes (one of each on a side) surrounding a central depression. During the 5th and 6th weeks, the bilateral maxillary processes derived from first branchial arch fuse with the medial nasal process to form the upper lip, alveolus, and primary palate. The lateral nasal process forms the alar structures of the nose. The mandibular processes form the lower lip and jaw. During the 8th week, the bilateral maxillary palatal shelves ascend to an appropriate level above the tongue and then fuse to each other and the primary palate to form the secondary palate (Fig. 1; Sperber et al., 2001).