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White Paper

American Association of Oral and Maxillofacial Surgeons

Craniofacial Anomalies

Craniofacial anomalies (CFAs) are congenital abnormalities in the bone or soft tissue of the face or head and comprise a wide range of heterogeneous conditions with many associated syndromes. Some CFAs and their associated syndromes are relatively common, such as cleft lip without cleft palate, which has an estimated prevalence of 1 in 940 newborn babies.[1] Others are rarer, such as Crouzon syndrome, which has an estimated prevalence of 16.5 per 1,000,000 births and results in the flattening of the back and top of the head, shallow eye sockets, retrusion (concavity) of the middle face and protrusion of the lower jaw.[2] Patients born with a CFA often will present with multiple auxiliary syndromes associated with the initial CFA. For example, about 350 different syndromes are associated with facial clefts, including hypodontia, malocclusion and airway obstruction. Babies and children with CFAs will have difficulty eating (including regurgitation and aspiration), ear infections, hearing loss, permanent speech impediments, gross jaw deformities of various types and a broad range of dental-related challenges – all of which are justifications for unavoidable surgical management.

Cleft lip repair is typically performed when a child is 6 to 12 weeks old and may require multiple surgeries depending on the extent of repair needed. Cleft palate treatment is more extensive and requires a multisurgical, multispecialty team approach over the course of 18 years. Initial surgeries create a functional palate, reduce the chances that fluid will develop in the middle ears, and aid in the proper development of the teeth and facial bones. Later surgeries are performed to account for the child's growth and development to ensure continued proper function as the child matures. This multistage surgical approach can result in significant improvement in a child's quality of life, ability to eat, breathe and talk. Other CFAs may require a similar course of treatment. Surgical intervention may be required early in life but often will be delayed until the patient has developed to a point that surgical correction will be optimized.

As a member of the team of multispecialty providers, oral and maxillofacial surgeons (OMSs) play an important role in the carefully orchestrated, multistage correctional approach for CFA patients. The goal is to help restore the jaw and facial structures, leading to normal function and appearance. The standard of care for treatment must consider function, appearance, nutrition, speech, hearing, and emotional and psychological development.

While insurance carriers generally provide coverage for early treatment of CFAs, procedures performed later in a child's life are frequently denied because of a "lack of medical necessity" or being deemed "cosmetic" in nature. Surgical and dental procedures related to CFAs are performed not for esthetics but for reconstructive purposes to address medical sequelae resulting from the congenital condition. Frequently denied treatments include the placement of dental implants, orthodontia and orthognathic treatment – all of which are necessary to restore function due to the initial oral cleft condition.

AAOMS supports legislation that requires insurers to provide coverage for all aspects of the treatment plan for CFA cases. Such coverage is necessary to ensure quality of life for these patients. AAOMS also supports medical coverage of orthodontic treatment and dental implants for patients with craniofacial anomalies.

References:

1 *Parker SE, Mai CT, Canfield MA, Rickard R, Wang Y, Meyer RE, Anderson P, Mason CA, Collins JS, Kirby RS, Correa A; for the National Birth Defects Prevention Network. Updated national birth prevalence estimates for selected birth defects in the United States, 2004-2006. Birth Defects Research (Part A): Clinical and Molecular Teratology 2010;88:1008-16.*

2 *Cohen Jr MM, Kreiborg S. Birth prevalence studies of the Crouzon syndrome: comparison of direct and indirect methods. Clin Genet 1992;41:12–15.*

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