Common Craniofacial Anomalies: The Facial Dysostoses

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Learning Objectives: After studying this article, the participant should be able to: 1. Understand the etiology and pathogenesis of facial dysostosis syndromes. 2. Recognize and classify common facial dysostoses. 3. Understand the different management plans for the reconstruction of facial dysostoses.

The wide spectrum of craniofacial malformations makes classification difficult. A simple classification system allows an overview of the current understanding of the etiology, assessment, and treatment of the most frequently encountered craniofacial anomalies. Facial dysostoses are reviewed on the basis of their diverse etiology, pathogenesis, anatomy, and treatment. Conditions discussed include craniofacial microsomia, Goldenhar syndrome, Treacher Collins syndrome, Nager syndrome, Binder syndrome, and Pierre Robin sequence. Approaches to the surgical management of these conditions are reviewed. (Plast. Reconstr. Surg. 110: 1714, 2002.)

The wide spectrum of craniofacial malformations makes them difficult to classify. Gorlin believes that our limited understanding of the embryology and etiology of the malformations restricts efforts at classification. In 1981 the Committee on Nomenclature and Classification of Craniofacial Anomalies of the American Cleft Palate Association grouped craniofacial disorders according to their diverse etiology, anatomy, and treatment. They proposed a practical and simple classification system of five categories that allows an overview of our current understanding of the etiology, assessment, and treatment of the most frequently encountered craniofacial anomalies:

I. Facial clefts/encephaloceles and dysostosis
II. Atrophy/hypoplasia
III. Neoplasia/hyperplasia
IV. Craniosynostosis
V. Unclassified

The broad category of facial clefts/encephaloceles and facial dysostosis is extensive. In this article, deformities of craniofacial dysostosis will be reviewed and discussed, as will the surgical correction of these craniofacial anomalies.

In 1976 Tessier described an anatomical classification system whereby a number is assigned to each of the malformations according to its position relative to the sagittal midline. This system has become internationally accepted and allows concise and effective communication between clinicians.

Van der Meulen and coworkers tried to correlate clinical features of the disorders with embryologic events. The authors envisioned the craniofacial skeleton as developing along a helical course symbolized by the letter S. Their scheme uses “focal fetal dysplasia” in preference to “cleft” for an arrest in skin, muscle, or bone development and names the dysplastic anomaly after the area(s) involved. In malformations characterized by dysostoses, an additional distinction is made between transformation defects and developmental arrests occurring before fusion of the facial processes.

The deformity known as maxillozygomatic dysplasia is equivalent to Tessier No. 6 cleft, or incomplete Treacher Collins syndrome. The complete form of Treacher Collins is characterized by zygomaticomandibular dys-
plasia and corresponds to Tessier Nos. 6, 7, and 8 clefts. Temporoauromandibular dysplasia is also known as auromandibular dysostosis, hemifacial microsomia, or first and second branchial arch syndrome.

Maxillomandibular dysplasia is a failure of the maxillary and mandibular processes to fuse, resulting in macrostomia. Mandibular dysplasias, exemplified by the Pierre Robin sequence, consist of micrognathia, glossoptosis, and respiratory distress.

**Craniofacial Microsomia**

Craniofacial or hemifacial microsomia is the term most frequently used to describe the first and second branchial arch syndrome, which correlates with a Tessier No. 7 cleft. Thomson was the first to suggest in 1843 that the malformation was caused by imperfect development of the first two anterior branchial arches. Clinical manifestations of craniofacial microsomia are underdevelopment of the external and middle ear and underdevelopment of the mandible, zygoma, maxilla, temporal bone, facial muscles, muscles of mastication, palatal muscles, tongue, and parotid gland. Macrostomia, a first branchial cleft sinus, and possible involvement of any or all cranial nerves are also features.

The birth incidence of craniofacial microsomia is approximately 1 in 4000, and approximately 10 percent of cases are bilateral. The possible etiology is thought to relate to hematoma of the embryonic stapedial artery injuring the developing first and second branchial arches. The expression of craniofacial microsomia is variable, with isolated microtia considered to be a microform of craniofacial microsomia. In its fullest expression, the craniofacial microsomia syndrome is made up of a constellation of congenitally malformed facial structures arising from the embryonic first and second visceral arches, the intervening first pharyngeal pouch and first branchial cleft, and the primordia of the temporal bone.

Murray and colleagues suggested that craniofacial microsomia is a progressive skeletal and soft-tissue deformity that worsens over time. Subsequently, Polley et al. performed longitudinal cephalometric analyses of 26 patients with untreated hemifacial microsomia and concluded that the deformity is nonprogressive and a phenomenon of growth, not worsening disease. More recently, Kearns et al. studied 67 patients with untreated hemifacial microsomia and again suggested that the process of asymmetry is indeed progressive. Presentations of the disease may vary from simple preauricular skin tags through hypoplasia or aplasia of skeletal and soft-tissue elements, with management dependent on the severity of the defect.

The deformities of craniofacial microsomia can be broadly considered as being skeletal, auricular, and soft-tissue. Pruzansky was the first to describe the mandibular deficiency in craniofacial microsomia and identified three types:

- **Type I:** Mild hypoplasia of the ramus, and the body of the mandible is minimally affected.
- **Type II:** Condyle and ramus are small, the head of the condyle is flattened, the glenoid fossa is absent, the condyle is hinged on a flat and often convex intratemporal surface, and the coronoid process may be absent.
- **Type III:** Ramus is reduced to a thin lamella of bone or is completely absent; no evidence of a temporomandibular joint (Fig. 1).

This classification was modified by Mulliken and Kaban, who added a clinically useful subdivision of type II into type IIA (Fig. 2), in which the glenoid fossa-condyle relationship is maintained and the temporomandibular joint is functional, and type IIB, in which the glenoid fossa-condyle relationship is not maintained and the temporomandibular joint is nonfunctional. The Kaban classification system is now widely accepted in classifying mandibular deficiency and is used to determine treatment protocols for mandibular deficiency syndromes.

Munro’s classification extended the skeletal anomaly to not only classify the mandible but also to include the orbit. This system aims at providing a basis for surgical reconstruction and consists of five types of skeletal abnormality. Type I shows a complete but hypoplastic facial skeleton, and types II through V show progressively severe absence of part of the skeleton. Type II has an absent mandibular condyle and part of the ramus. Type III also lacks the zygomatic arch and glenoid fossa. In type IV, the above findings are associated with posterior and medial displacement of the lateral orbital wall. Type V further presents with a micro-orbit or an inferiorly displaced orbit.

The auricular deformity of craniofacial mi-
crosomia was classified by Meurman, who recognized the following three grades of deformity: grade I, distinctly smaller malformed auricle but all components are present; grade II, only a vertical remnant of cartilage and skin is present, with atresia of the external meatus; grade III, complete or nearly complete absence of the auricle.20,22

Incorporating the skeletal (S), auricular (A), and soft-tissue (T) anomalies, David and colleagues23 proposed a multisystem classification of hemifacial microsomia in the tumor, node, metastasis style. The physical manifestations of hemifacial microsomia are graded according to five levels of skeletal deformity (S1 to S5) equivalent to the Pruzansky classification for S1 to S3, with S4 representing orbital involvement and S5 representing orbital dystopia. The auricular deformity (A0 to A3) is similar to the classification described by Meurman,20 and the soft-tissue deficiency (T1 to T3) is graded as mild, moderate, or severe. The SAT system

![Image]

Fig. 1. (Above, left and right) Severe expression of craniofacial microsomia with Pruzansky type III mandibular hypoplasia, severe soft-tissue hypoplasia, and microtia. (Below) Three-dimensional computed tomographic reconstruction shows Pruzansky type III mandibular hypoplasia.
enables classification of the skeletal, auricular, and soft-tissue deformity and allows a comprehensive and staged approach to skeletal and soft-tissue reconstruction (Fig. 3).

The timing of surgical intervention in craniofacial microsomia must be integrated to address the multiple tissue deficiencies. Macrostomia should be corrected in the first few months of life, similar to the timing of cleft lip repair. Most authors believe that surgery for moderate-to-severe craniofacial microsomia should be performed when the child is 5 to 6 years old, rather than waiting until the child is older and facial growth is complete. The mandibular deformity is usually compensated for first. It was believed in the past that repositioning the jaw would unlock the growth potential of the functional matrix to allow more normal growth of the mandible, although it does remove the abnormal growth tendencies of the maxilla. In mild cases of mandibular deformity, Posnick prefers to wait for skeletal maturity and uses traditional orthognathic surgery to achieve favorable aesthetic results. For mild cases, mandibular surgery may involve contour surgery with or without genioplasty once skeletal maturity is reached. The advent of distraction osteogenesis allows early correction of cases of mandibular deformity up to type IIB, whereas in cases of type III deformity, a costochondral graft is usually indicated, although many au-

FIG. 2. Moderate expression of right craniofacial microsomia, with deviation of menton to right secondary to mandibular asymmetry (Pruzansky type IIA), and right microtia.

FIG. 3. SAT classification system for craniofacial microsomia allows classification of the skeletal (S), auricular (A), and soft-tissue (T) anomalies and provides a treatment plan. (From David, D. J., Mahatumarat, C., and Cooter, R. D. Hemifacial microsomia: A multisystem classification. Plast. Reconstr. Surg. 80: 525, 1987; used with permission.)
thors have noted unpredictable overgrowth of costochondral grafts.\textsuperscript{8,16–18,27,33} Ross\textsuperscript{34} reviewed 48 cases of severe craniofacial microsomia treated with costochondral grafts at The Hospital for Sick Children. The success rates were higher when the children were operated on earlier (85 percent for ages 3 to 7 years versus 50 percent for those older than 14 years). Equal growth with the other normal side was seen in 46 percent of cases, undergrowth in 15 percent, and overgrowth in 39 percent. Given the option of early or delayed surgery, the author favors early surgery at age 4 to 5 years, citing a higher graft success rate, the psychosocial advantage of attaining improved facial symmetry at a younger age, and the additional benefit that erupting teeth will assume a more normal position, making future orthodontic treatment less difficult.

Munro\textsuperscript{16,18,27} advocates much more extensive surgery, operating on the maxilla at the same time as the mandible. In orbitozygomatic hypoplasia, Posnick\textsuperscript{24} performs reconstruction using split cranial bone grafts at age 5 to 7, stating that at age 7 the cranio-orbitozygomatic complex is nearly mature, allowing reconstruction of an adult-size vault, orbit, and cheekbone. Also, the thickness of the calvarium at that age makes for an easier harvest of split calvarial bone grafts. Ultimately, refinements to the mature skeleton may be needed, and patients may require sagittal split osteotomy, Le Fort I osteotomy, or genioplasty to achieve a symmetrical and aesthetic result.\textsuperscript{24}

At times, the soft-tissue deformity in craniofacial microsomia must also be addressed to attain a long-term aesthetic result, and this usually follows skeletal reconstruction. Past treatment methods have included silicone fluid injections,\textsuperscript{35} which are contraindicated because of long-term complications, and injections of lipoaspirated fat, which have met with mixed results.\textsuperscript{26} Autologous fat injections, however, certainly have a place in the correction of mild contour irregularities. Muscular atrophy of pedicled or free muscle flaps presents a problem for calculating the final volume needed for the repair,\textsuperscript{37} and microvascular free tissue transfer of dermis fat flaps, as described by La Rossa et al.\textsuperscript{38} and Upton et al.,\textsuperscript{39} is preferred for large-volume soft-tissue deficiency augmentation.

\textbf{Goldenhar Syndrome}

The features of Goldenhar syndrome (ocularauriculovertebral dysplasia) resemble those of craniofacial microsomia except that Goldenhar syndrome is characteristically bilateral. Differentiating features include epibulbar dermoids and vertebral anomalies\textsuperscript{10} (Fig. 4). Occurrence is believed to be sporadic, with only a weak genetic component. Management protocols are similar to those used in cases of craniofacial microsomia.

\textbf{Treacher Collins Syndrome}

The first reference to mandibulofacial dysostosis in the medical literature was made by Berry in 1889. Berry described the physical symptoms and speculated about the heritable transmission of the deformity. His treatise was certainly much more detailed than the two cases reported 11 years later by Treacher Collins, after whom the syndrome was named. In Europe, the deformity is known as the Franceschetti-Zwahlen-Klein syndrome on the basis of those authors’ 1949 monograph summarizing the world literature.\textsuperscript{41} Treacher Collins syndrome is variably expressed; the \textit{incomplete} form should be designated as Treacher Collins syn-

![Fig. 4. Goldenhar syndrome. Skeletal deformity similar to craniofacial microsomia (Pruzanovsky type III mandibular hypoplasia) with residual eyelid deformity following excision of epibulbar dermoid.](image-url)
drome and the complete form as Franceschetti syndrome.

Treacher Collins syndrome represents a manifestation of the Tessier Nos. 6, 7, and 8 clefts. It is inherited as an autosomal dominant trait with an incidence of 1 in 10,000 live births. Bilaterality is always present, and although phenotypic expression is variable, the deformity is nearly always symmetrical (Figs. 5 and 6). The gene for Treacher Collins syndrome was identified in 1991, when it was mapped to chromosome 5 by Dixon and colleagues from a study of 12 families. Later that year, the genetic locus was refined to bands 5q31.3→q33.3.

The pathogenesis of Treacher Collins syndrome remains unknown. Sulik and colleagues were able to produce the facial abnormalities of Treacher Collins in mice by administration of isotretinoin, suggesting that the syndrome can be triggered by disruption of vitamin A metabolism. There is also an apparent correlation between frequency of mutation and advanced paternal age.

The typical features of Treacher Collins syndrome include:

![Fig. 5. Treacher Collins syndrome. The skeletal features of mandibular hypoplasia are similar to those of craniofacial microsomia with a propensity for bilaterality. Note the palpebral fissures sloping downward laterally (antimongoloid slant); hypoplasia of the facial bones, especially the malar bones (bilateral presence of zygomas demonstrating variable expression); malformation of the external ear; and absence of eyelashes in the medial third of the lower eyelid.]

![Fig. 6. (Above) A child with Treacher Collins syndrome following repair of bilateral lower lid colobomata and bilateral mandibular distraction osteogenesis. Note the palpebral fissures sloping downward laterally (antimongoloid slant), with coloboma of the outer portion of the lower lid; hypoplasia (aplasia) of the facial bones, especially the malar bones and mandible; malformation of the external, middle, and inner ear; atypical hair growth in the form of tongue-shaped processes of the hairline extending toward the cheeks; and absence of eyelashes in the medial third of the lower eyelid. Zygomatic reconstruction with split rib grafts is planned, followed by bilateral microtia reconstruction. (Below) Three-dimensional computed tomographic reconstruction of a patient with Treacher Collins syndrome demonstrating hypoplasia of the facial bones with near total aplasia of the malar bones.]

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• palpebral fissures sloping downward laterally (antimongoloid slant), with coloboma of the outer portion of the lower lid and, rarely, the upper lid
• hypoplasia (aplasia) of the facial bones, especially the malar bones and mandible
• malformation of the external ear and, occasionally, the middle and inner ear
• macrostomia, high palate, abnormal position and malocclusion of the teeth
• blind fistula between the angles of the mouth and the ears
• atypical hair growth in the form of tongue-shaped processes of the hairline extending toward the cheeks
• absence of eyelashes in at least the medial third of the lower eyelid

In newborns with the syndrome, the overwhelming priority is airway management. Shprintzen et al. noted that some patients have marked narrowing of the airway (pharyngeal diameter <1 cm in some cases), and Behrents et al. described extreme shortening of the mandible with severe lower face retrusion. Combined, these malformations can explain the obstructive sleep apnea and reports of neonatal death associated with cases of Treacher Collins syndrome. Tracheostomy may be necessary, although distraction osteogenesis of the mandible in the neonatal period has been used to avert it.

The eyelid coloboma must be addressed to protect the cornea. Tessier addresses the problem with a Z-plasty, and Jackson uses a full-thickness skin-tarsal plate flap from the upper lid for reconstruction of the lower lid. Correction of the coloboma must be addressed acutely, and despite the description of multiple techniques to address the problem, an “operated-on look” is hard to avoid.

Primary reconstructive surgery in Treacher Collins syndrome is directed to the maxilla, mandible, and zygoma and to the soft-tissue deficiencies of the eye, ear, and malar regions. Pruzansky type I and II mandibular defects can be corrected with distraction osteogenesis in children younger than 10 years. Type III defects may require costochondral rib grafts, which are best performed during the mixed dentition phase (age 6 to 10 years). Posnick et al. augment hypoplastic zygomas with onlay nonvascularized bone grafts, without significant graft resorption or change in contour over time. Others prefer vascularized calvarial bone flaps or osteotomy and advancement, finding the resorption of nonvascularized bone to be high. Van der Meulen and associates prefer a temporal osteoperiosteal flap, and Psillakis et al. describe a temporal aponeurosis-vascularized outer table fascial flap. Autogenous cartilage is the preferred form of auricular reconstruction, and soft-tissue augmentation can be performed with dermal grafts or free-tissue transfer.

Orthognathic surgery and refinement procedures with onlay grafts and soft-tissue augmentation can be beneficial to children aged 10 to 19 years. Freihofer describes a combination of well-known orthognathic techniques for the correction of Treacher Collins syndrome. The patients are treated in two to three operative sessions beginning early in the second decade of life. The main components are chin advancement with concomitant malar osteotomies in the first session. In the second operation, the chin prominence is moved further forward by simultaneous vertical movement of the maxilla, sagittal split osteotomy, and body osteotomy of the mandible.

NAGER SYNDROME

The Nager anomaly (acrofacial dysostosis) is rare. It is inherited as an autosomal recessive trait, and patients have craniofacial features similar to mandibulofacial dysostosis, coupled with preaxial reduction defects of the upper and, sometimes, the lower limbs (Fig. 7). In the upper limbs, there is hypoplasia or agenesis of the thumbs and radius and of one or more metacarpals. Unlike in Treacher Collins, lower eyelid colobomas are not as frequent but cleft palate is practically universal. Affected individuals are also typically of short stature and have subnormal intelligence.

BINDER SYNDROME

Zuckerkandl in 1882 described an anomaly in the anterior nasal floor in which the normal crest that separates the nasal floor from the anterior surface of the maxilla was absent and, instead, a small pit (the fossa prenasalis) constituted the inferior margin of the piriform aperture. In 1939, Noyes described a patient with a flat nasal tip sitting on a retruded maxillonasal base, and in 1962, von Binder described a syndrome consisting of a short nose with a flat bridge, absent frontonasal angle, absent anterior nasal spine, limited nasal mucosa, short columella and acute nasolabial ang-
perialar flatness, convex upper lip, and a tendency to class III occlusion. Occasionally there may be hypoplastic frontal sinuses. Von Binder postulated these defects were caused by rhinocphalic dysplasia, which he called “maxillonasal dysostosis.” Since that time, the condition has been known as maxillonasal dysplasia or Binder syndrome.66

Posnick and Tompson67 note that the physical findings of Binder syndrome are the result of hypoplasia (depression) of the anterior nasal floor (fossa prenasalis) and localized symmetric maxillary hypoplasia of the alar rim regions. From the basal view, typical variations from normal include a retracted columella-lip junction; lack of normal triangular flare at the nasal base, a perpendicular alar-cheek junction; convex upper nasal tip with a wide, shallow philtrum; crescent-shaped nostrils without a sill; low-set and flat nasal tip; and stretched and shallow Cupid’s bow (Figs. 8 and 9).68 The most striking characteristics of the nose are vertical shortening, lack of tip projection, perialar flattening, and an acute nasolabial angle. Holmstrom68 found a hereditary connection in 16 percent of 50 patients with Binder syndrome, and inheritance may be as an autosomal recessive trait with incomplete penetration.69,70

Surgical correction of the deformities of Binder syndrome is complex.67,71 Techniques for nasal reconstruction aim at increasing nasal length and tip projection and have been described by Posnick and Tompson,67 Holmstrom,71 Jackson et al.,72 and Banks and Tan-
Reconstructive options include Le Fort II osteotomy, Le Fort I osteotomy, a combination of Le Fort II and I osteotomies, compensatory orthodontic alignment of the teeth, and infraorbital rim augmentation. Nasal reconstruction may involve both autogenous and homogenous bone and cartilage grafts extending up the columella and over the dorsum, from the radix to the tip.

Disappointing long-term results thought to be caused by bone graft resorption were challenged by Rune and Aberg. They followed up 11 patients for 40 months and found a reduction of graft length of 28 percent. Despite this, there was no alteration in the achieved improvement in nasal length or tip projection.

Banks and Tanner approach the nasal deformity in Binder syndrome by lifting the facial mask through a coronal incision and reaching the nasal floor through an incision in the upper buccal sulcus. The nasal soft tissues, including the alar cartilages, are mobilized. The nose is lengthened and tip projection is achieved with a cantilever graft of lyophilized cartilage.

Wolfe describes a technique of nasofrontal osteotomy to lengthen the nose in cases of posttraumatic shortening and Binder syndrome. McCollum et al. review the literature and provide long-term follow-up of two patients—one treated with traditional orthognathic surgery and the other with a growth center implant to the nose.

PIERRE ROBIN SEQUENCE

In 1923, Pierre Robin, a French stomatologist, identified the physical characteristics of a combination of anomalies that were once considered a syndrome but are known now as the Pierre Robin sequence. The characteristic features of the Pierre Robin sequence are retrogenia, glossoptosis, and airway obstruction (Fig. 10). Although many infants with Pierre Robin sequence have micrognathia, Randall points out that retrogenia better describes the condition of the jaws in the disorder because it is the posterior displacement of the chin that predisposes to glossoptosis. An associated high-arched midline cleft of the soft palate and, occasionally, of the hard palate is also present in about 50 percent of cases. The sequence shows great etiologic heterogeneity, with as many as 18 associated syndromes.
The glossoptosis in Pierre Robin sequence can begin a vicious sequence of events, with airway obstruction, increased energy expenditure, and decreased caloric intake from impaired feeding. Afflicted infants typically fail to thrive because of respiratory and feeding difficulties, and if these problems are ignored, cardiac failure and death may ensue.

In 1946, Douglas reported a greater than 50 percent mortality rate with conservative treatment of Pierre Robin sequence. It is now clear that the key to successful medical treatment of infants with Pierre Robin sequence is to hold the infant prone to relieve the glossoptosis and open the airway. In some cases, this position must be maintained 24 hours a day, even during feeding, baths, and diaper changing.

Although most infants can be successfully treated conservatively, a few will require surgical intervention. If medical treatment failed to relieve the symptoms of airway obstruction, the infant previously would have been considered for tongue-lip adhesion or tracheostomy. The advent of distraction osteogenesis provides an alternative for addressing airway obstruction. Denny et al. describe a series of 10 patients who were treated with distraction osteogenesis of the mandible. Two of three patients with indwelling tracheostomies were successfully decannulated within 6 weeks. All children showed clinical improvement after mandibular distraction, with a mean effective airway increase after distraction of 67.5 percent.


Self-Assessment Examination follows on the next page.
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1. WHICH OF THE FOLLOWING IS CHARACTERISTIC OF TREACHER COLLINS SYNDROME?
   A) Autosomal recessive
   B) Bilaterality
   C) Microstomia
   D) Mongoloid slant of palpebral fissure
   E) Frontal bossing

2. THE MAJOR DIFFERENCES BETWEEN THE FACIAL ANOMALIES IN NAGER'S SYNDROME AND TREACHER
   COLLINS SYNDROME IS:
   A) Auricular deformity
   B) Facial bone hypoplasia
   C) Cleft palate
   D) Coloboma
   E) Antimongoloid slant of palpebral fissure

3. WHICH OF THE FOLLOWING IS THE PRIMARY DIFFERENTIATOR BETWEEN CRANIOFACIAL
   MICROsomia AND GOLDENHAR SYNDROME?
   A) Degree of mandibular involvement
   B) Extent of auricular involvement
   C) Presence of macrostomia
   D) Bilaterality
   E) Presence of coloboma

4. WHICH OF THE FOLLOWING COMPONENTS OF CRANIOFACIAL MICROsomia SHOULD BE
   CORRECTED FIRST?
   A) Auricular deformity
   B) Macrostomia
   C) Mandibular ramus deficiency
   D) Mandibular body deficiency
   E) Temporal mandibular joint abnormalities

5. IN THE MAJORITY OF PATIENTS WITH PIERRE ROBIN SEQUENCE, AIRWAY OBSTRUCTION CAN BE
   SUCCESSFULLY MANAGED WITH:
   A) Prone positioning
   B) Oral-tracheal intubation
   C) Lip-tongue adhesion
   D) Tracheostomy
   E) Mandibular distraction

6. THE PHYSICAL FINDINGS IN BINDER'S SYNDROME ARE PRIMARILY ATTRIBUTABLE TO HYPOPLASIA
   OF THE:
   A) Medial orbital wall
   B) Anterior wall of the maxilla
   C) Nasal septum
   D) Anterior nasal floor
   E) Anterior cranial base

To complete the examination for CME credit, turn to page 1824 for instructions and the response form.