1. INTRODUCTION

1.1. Introduction to craniofacial surgery

Craniofacial surgery is a growing subspecialty that seeks to manage congenital and acquired malformations of the face, skull and jaw. Craniofacial surgeons deal with a vast range of conditions including craniosynostosis and craniofacial clefts, as well as various miscellaneous congenital malformations. This chapter aims to cover the basic anatomy and embryology of the skull and palate, and then focuses on the common craniosynostosis syndromes, cleft lip and palate (CL/P).

Craniosynostosis dates back to 100 BC, when it was initially described by Hippocrates. It is defined as the premature fusion of one or more cranial sutures either during development in utero or soon after birth. More boys are affected than girls, with the sagittal suture being involved most often. It can be classified into syndromic and non-syndromic, with the former being less common (15–40%) despite over 100 reported syndromes (Cohen, 2000).

CL/P is one of the most common congenital craniofacial deformities encountered in newborns, affecting 1 in 600 live births. The defect develops from abnormalities in the growth of the foetal facial skeleton leaving an opening – a cleft. The most common forms that occur involve both the lip and palate (45%), the palate (40%) or the lip (15%) alone and are either unilateral or bilateral. Infants born with these conditions may also have other associated abnormalities, for example as part of Pierre Robin syndrome (Bailey et al., 2013).
1.2. Embryological development

1.2.1. Skull embryology

The skull develops from the mesenchyme and is comprised of the (1) neurocranium, which envelopes the brain; and (2) viscerocranium, which constitutes the facial skeleton.

The chondrocranium, which is the initial cartilaginous form of the neurocranium, undergoes endochondral ossification to form the bones of the base of the skull. This is followed by intramembranous ossification, forming the cranial vault (calvaria). The skull bones are separated by connective tissue, which constitutes the fibrous joints of the suture planes. The skull undergoes moulding during foetal life owing to its soft and malleable nature.

The cartilaginous viscerocranium is derived from the first two pairs of pharyngeal arches. The first arch cartilage is responsible for formation of the malleus and incus. The second arch cartilage results in the production of the stapes and styloid process. Following ossification, the membranous viscerocranium forms, resulting in formation of the squamous, temporal, zygomatic and maxillary bones (Moore et al., 2013; Sadler and Langman, 2009).

1.2.2. Embryology of the palate

Palatogenesis stems from two embryological structures. The primary palate arises as the median palatine process during the 6th week of gestation after fusion of the median nasal processes. This contributes to the formation of the pre-maxillary part of the maxilla, which lies anterior to the incisive foramen. The secondary palate contributes to the formation of the hard and soft palate located posterior to the incisive foramen. It develops from the lateral palatine processes at the 6th week of gestation. These processes subsequently fuse within the median plane as well as with the posterior portion of the primary palate and the nasal septum (Moore et al., 2013; Sadler and Langman 2009).

1.3. Functional anatomy

The cranium encloses the brain, the meninges and the remainder of the intracranial contents. The neurocranium is made up of eight bones and consists of a cap, the calvaria and a base (Figure 12.1). The cranial base is comprised mainly of the sphenoidal and temporal bones. The calvarium is formed by the frontal, occipital and parietal bones. The viscerocranium represents the facial skeleton and made up of three single bones and six paired bones (Sinnatamby and Last, 2011).

Sutures are immoveable fibrous joints that unite the calvaria. In neonates, the fontanelles represent the normal, incomplete suture fusion points that facilitate expansion of the neurocranium during the early growth of the brain. The two major ones are the anterior (or frontal) and posterior (or occipital) fontanelles (Figure 12.2). The timed fusion of the fibrous sutures of a newborn is well co-ordinated with brain growth, enabling it to expand to its optimum size before fusing (Moore et al., 2013).
Figure 12.1. Lateral view of skull anatomy. Bones of the neurocranium include the frontal, ethmoidal, sphenoidal, occipital and two sets of bones on either side of the skull: the temporal and parietal. The viscerocranium is made up of the following bones: mandible, vomer, ethmoid (three single bones), maxillae, inferior nasal conchae, zygomatic, palatine, nasal and lacrimal bones (six paired bones).

Figure 12.2. Illustration of the main skull sutures (superior view). The coronal suture lies between the frontal and parietal bones; the metopic suture lies between the frontal bone; the lambdoidal suture connects the parietal bones to the occipital bone; the sagittal suture lies between the parietal bones; and the squamosal suture connects the parietal and temporal bones. The posterior fontanelles commonly close at 1–2 months of age, whereas the anterior fontanelles shut at 9–18 months.
The hard palate is a bony structure which constitutes the roof of the mouth and is comprised of the palatine processes of the maxilla, as well as the horizontal plate of the palatine bone. It primarily functions in speech production and feeding. The soft palate is an aponeurotic extension commencing at the posterior aspect of the hard palate. It is an associated mobile flap which aids the functions of the hard palate. It is made up of a series of muscles which include tensor veli palatini, levator veli palatini, palatoglossus and palatopharyngeus (Drake et al., 2010).

### 1.4. Developmental abnormalities

#### 1.4.1. Cleft abnormalities

In complete cleft palate, an opening is evident between the two palatal processes throughout its complete length so that the nose and mouth are in communication. In incomplete cleft palate, the two halves of the palate unite from front to backwards; thus, the anterior part is normal. The last parts to fuse are the two halves of the uvula resulting in (1) a bifid uvula; (2) a bifid soft palate along its course; or (3) a bifid soft palate with involvement of the posterior part of the hard palate. Velopharyngeal inadequacy is observed in a submucous cleft palate: rather than forming a transverse sling across the posterior soft palate, the levator veli palatini muscles insert abnormally onto the posterior aspect of hard palate (Bailey et al., 2013; Cuschieri et al., 2003).

A failure of the maxillary prominence (on the affected side) to join with the merged medial nasal prominences results in a unilateral cleft lip (Figure 12.3A). A bilateral cleft lip, however, develops from the failure of mesenchymal masses of the maxillary prominences to meet and merge with the merged

![Figure 12.3](image-url). A. A unilateral left-sided CL/P deformity in a neonate, showing the abnormal communication. B. A bilateral CL/P defect present in a newborn.
medial nasal prominences (Figure 12.3B). In addition, mesodermal deficiencies result in a median cleft lip by partial or complete failure of the medial nasal prominences to merge and form the intermaxillary segment (Moore et al., 2013).

### 1.4.2. Craniosynostosis abnormalities

The sutures involved and deformities observed in craniosynostosis are described in Table 12.1 (Alden et al., 1999).

**Table 12.1.** Developmental patterns of craniosynostosis.

<table>
<thead>
<tr>
<th>Suture</th>
<th>Cranial deformity</th>
<th>Description</th>
<th>Incidence</th>
<th>Radiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sagittal</td>
<td>Scaphocephaly</td>
<td>Tall, narrow skull</td>
<td>1/5,000</td>
<td>Long narrow skull</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Small anterior fontanelle</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Absence of mid-line sagittal suture</td>
</tr>
<tr>
<td>Bilateral coronal</td>
<td>Brachycephaly</td>
<td>Short anteroposterior dimension and bitemporal widening</td>
<td>1/150,000</td>
<td>Absence or sclerosis of sutures</td>
</tr>
<tr>
<td>Bilateral coronal</td>
<td>Turricephaly</td>
<td>Tower skull with increased height of the cranium</td>
<td>1/17,000</td>
<td>Bilateral harlequin eye sign</td>
</tr>
<tr>
<td>(untreated)</td>
<td></td>
<td></td>
<td></td>
<td>Upward and outward peaking of orbital contour</td>
</tr>
<tr>
<td>Unilateral coronal</td>
<td>Plagiocephaly</td>
<td>Ipsilateral frontal flattening; contralateral frontal bossing</td>
<td>1/25,000</td>
<td>Oval orbits</td>
</tr>
<tr>
<td>Metopic</td>
<td>Trigonocephaly</td>
<td>Keel-shaped anterior skull with pointy forehead</td>
<td></td>
<td>Hypertelorism</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Small frontal bone with hyperostosis</td>
</tr>
<tr>
<td>Multiple</td>
<td>Turricephaly</td>
<td>Pointed head with a posteriorly tilted head</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Kleeblattschädel</td>
<td>Trilobed ‘cloverleaf’ cranium</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Virchow postulated in 1851 that the cranial deformities observed were caused by the premature fusion of skull sutures. Virchow’s law states that brain growth is restricted along a plane perpendicular to the fused suture with compensatory overgrowth occurring at the non-fused suture sites resulting in the cranial deformities described. The commonest synostosed suture in craniosynostosis is the sagittal suture (40–55%), followed by the coronal (20–25%), metopic (5–15%) and lamboid (<5%) sutures.

*Source:* Adapted from Marks and Marks (1997).
2. CRANIOSYNOSTOSIS SYNDROMES

2.1. Crouzon syndrome

Affecting 1 in 25,000 births, this syndrome may be inherited and is manifested in a similar manner to Apert syndrome with comparable skull and ocular characteristics but more prominent midface hypoplasia. Mandibular prognathism with lower jaw retrusion, a high palate or cleft palate may also be present. A distinguishing noticeable feature is the presence of normal hands. Raised intracranial pressure (ICP) may result in irreversible loss of vision due to atrophy of the optic disc and must be reversed immediately (Padmanabhan et al., 2011).

2.2. Apert syndrome

Apert syndrome manifests with bicornal synostosis that largely develops sporadically; however, some are inherited in an autosomal dominant pattern through mutation of the FGFR2 gene. Craniofacial abnormalities are characterised by a turribrachycephaly, low-set ears and a small beaked nose (Figure 12.4). Typically, the midface is hypoplastic and pseudoprognathism is observed, in which class III malocclusion protrudes the mandible forward, causing an anterior open bite. Ocular features include hypertelorism and protruded eyeballs due to shallow orbits and exotropia. Obstructive sleep apnoea develops from reduced opening of the nasal choanae (Kabbani and Raghuvir, 2004).

![Figure 12.4. Facial characteristics associated with Apert syndrome. A. Frontal view B. Profile view. Note the flat, elongated head with bitemporal widening and occipital flattening. The midface is hypoplastic with a small, beaked nose and low-set ears. Pseudoprognathism is also observed in this syndrome.](image-url)
Other characteristic signs include congenital hand deformities. Syndactyly (fusion of the upper and lower limb digits) is observed commonly in the 2nd, 3rd and 4th digits. Mental retardation due to raised ICP or hydrocephalus may ensue if treatment is delayed.

### 2.3. Pfeiffer syndrome

As described by Pfeiffer in 1964, mutation of the *FGFR2* gene is apparent. The syndrome is inherited in an autosomal dominant manner. Broad thumbs and a widened big toe classically characterise this syndrome; however, these can be difficult to detect. Occasionally, syndactyly involving 2nd and 3rd digits is apparent. The craniofacial anomalies are similar to those of Apert and Crouzon syndromes, with mid-face hypoplasia, proptosis and hypertelorism. In addition, the nose is downturned and the nasal bridge is flat. Mental retardation, although uncommon, may also be observed (Thorne *et al.*, 2014).

### 2.4. Muenke syndrome

Caused by a *GFGR3* gene mutation, this syndrome has features overlapping with those of other GFGR-related craniosynostosis. More distinctive characteristics are unilateral or bilateral coronal synostosis, hypoplastic midface, broad toes and brachydactyly. Asymmetry of the face is also noticed. Ipsilaterally, the superior orbital rim and eyebrows are raised, the forehead is flattened and ears are displaced anteriorly. Contralaterally, there is prominent frontal bossing with eyebrow depression. Hearing loss is a common feature; thus, hearing assessment at a young age is vital to prevent learning difficulties (Agochukwu *et al.*, 1993).

### 2.5. Saethre–Chotzen syndrome

This syndrome is caused by mutation of the *TWIST1* gene. It is commonly characterised by brachycephaly, a low anterior hairline, bilateral ptosis and facial asymmetry. Irregularity of the face is commonly accompanied by nasal septum deviation and maxillary hypoplasia. The intelligence and midface is unaffected in this autosomal dominant disorder.

### 2.6. Management of craniosynostosis syndromes

#### 2.6.1. Key to early intervention

Rapid correction of craniofacial dysmorphism is essential to take advantage of the fast-growing brain within the first 12 months of age. Management of raised ICP prevents disabling complications such as
blindness and mental retardation. Linear craniectomy and fragmentation of the vault may provide early temporary protection of the brain until definitive craniofacial procedures are undertaken. Tarsorrhaphy may be considered to protect the cornea of the eye. Early intervention may also be of psychosocial benefit for the child if the disfigurement is corrected at a young age.

### 2.6.2. Definitive craniofacial surgery

#### 2.6.2.1. Fronto-orbital advancement

A coronal incision is made with the aim of releasing the synostosed suture to decompress the cranial vault. A frontal craniotomy is carried out to reshape the vault and elevate the frontal bone. The supraorbital bar is also advanced providing protection to the eyeball and improving cosmesis.

#### 2.6.2.2. Le Fort osteotomy

When the facial skeletal growth is complete, facial re-contouring can be offered for cosmetic and functional purposes (Figure 12.5A). (1) **Le Fort I osteotomy** is indicated for class III malocclusion following complete maturation of the maxilla and mandible. Either the mandible is protruding or the maxilla is retruded; thus, the nose, malar and upper maxilla bones are shifted forward. (2) **Le Fort II osteotomy** is indicated when the nasomaxillary area is retruded. (3) **Le Fort III osteotomy** involves the simultaneous movement of the lower orbit, maxilla and zygoma as a single unit. The bones are merged together and fixated at the nasolacrimal junction, lateral orbital wall and pterygomaxillary fissure. This procedure is usually performed between the ages of 7 and 9 years. At this age, orbital growth is near completion and can thus withstand fixation with screws. This, however, may need to be performed at a younger age if upper airway obstruction or ocular complications ensue. This technique may be combined with facial bipartition to correct hypertelorism, downslanting palpebral fissures and midface concavity (Thorne et al., 2014).

#### 2.6.2.3. Monobloc osteotomy

This procedure advances the fronto-orbital and Le Fort III segments simultaneously in one fragment to correct midface retrusion (Figure 12.5B). The age at which this is performed is debated among surgeons regarding whether it should be carried out early or left until complete skeletal development at puberty. Upper airway obstruction or severe exorbitism, however, may command early intervention.

The monobloc procedure alone remains controversial because of high infection rates. Contamination of the dead space created between the frontal lobe and bone develops from direct communication between the anterior cranial fossa and the paranasal sinus and nasopharynx. A staged procedure involving fronto-orbital advancement followed by Le Fort III or the use of monobloc distraction has thus become the more favourable technique for syndromic craniosynostosis. Distraction permits growth of the soft tissue envelope with monobloc advancement retaining the barrier between the nasal cavity and
anterior fossa. Other advantages include a reduction in both surgery time and relapse rates. However, patients require further surgery to remove embedded devices following distraction surgery and must wear a halo frame around their head for several months (Bradley et al., 2006).

2.7. Detecting sinister signs

Raised ICP can be detected clinically by examining for papilloedema and optic disc atrophy on fundoscopy. At later stages, plain skull radiography may show a ‘thumb printing’ or ‘beaten copper’ appearance and towering of the head (i.e. turricephaly) may develop (Staatz et al., 2007). Intraparenchymal monitoring using pressure-sensitive filaments remains the gold standard for measuring ICP.

Orbital signs must be recognised and managed appropriately. Exorbitism results in exposure of the cornea and subsequently keratitis, corneal ulcerations and possibly loss of vision. A thorough examination involving the assessment of visual acuity and slit lamp biomicroscopy is essential.

Hydrocephalus, although rare, can develop in syndromic craniosynostosis (commonly Apert syndrome). It may present with signs of raised ICP, which may be difficult to distinguish from these syndromes. Pre-operative computed tomography or ultrasound scanning detects those at a high risk, who are then best managed with a ventriculoperitoneal shunt (Marks and Marks, 1997).

3. CLEFT LIP AND PALATE

CL/P deformities in neonates are evident at birth and are recognised by the presence of clefts affecting the lip and/or palate with potential nasal extension. However, this may not be visible in the case of
submucous clefts. The aetiology is believed to be a combination of environmental and genetic influences. Environmental factors include drug exposure during pregnancy (steroids, diazepam, phenytoin). A positive family history predisposes toward an increased incidence of the anomaly (Table 12.2). Although the occurrence of CL/P in an isolated form is the usual mode of presentation, it can also be associated with a syndrome, of which Pierre Robin syndrome is the commonest (Bailey et al., 2013).

One of the most common classification systems employed for CL/P is the LAHSHAL method (Figure 12.6). This system incorporates several features of the pathology, defining whether the CL/P lesion is present in combination or isolation, is unilateral or bilateral, and complete or incomplete (Thorne et al., 2014).

Submucosal cleft is a variant cleft palate deformity that often goes undiagnosed in neonates because the deformity lies underneath the covering of the mucosa. Both the hard and soft palate can be affected and it has an incidence of 1:1250–1:6000. It is often characterised by a triad of signs: (1) a bifid uvula; (2) a translucent zone in the soft palate; and (3) the presence of a bony notch in the posterior edge of the hard palate. However, it is often detected incidentally after investigating for nasal speech. Complications include velopharyngeal insufficiency, otitis media and speech difficulties (Neligan et al., 2013).

### 3.1. Management of cleft lip and palate

A multidisciplinary approach is key to the management of cleft deformities. The team of specialists should include plastic and maxillofacial surgeons, orthodontists, speech therapists, psychologists and dieticians. All forms of CL/P defects, with the exception of isolated cleft palate, can be detected during intra-uterine life by ultrasound. This may be ordered after 18 weeks of gestation have elapsed. The antenatal diagnosis is important because it allows clinicians to counsel parents in advance and plan the appropriate treatment protocols (Bailey et al., 2013; Cuschieri et al., 2003).

#### 3.1.1. Neonatal care

Initial neonatal care should centre on airway protection with the aid of nasopharyngeal tubes as required. Nursing in the prone position and labioglossopexy (tongue–lip adhesion) in more serious cases can be employed to prevent obstruction by the tongue. Moreover, the inability of newborn babies with CL/P
to produce a significant negative intraoral pressure impedes their suckling ability and thus nutritional status. This can be overcome with a special feeding teat (Bailey et al., 2013).

### 3.1.2. Pre-surgical work up

Good planning prior to the procedure is vital to provide a good prognosis and involves different methods to objectively delineate the abnormality. It can also include moulding techniques, which attempt to reduce the magnitude of the deformity. The child must be in good health prior to the procedure and should not have any respiratory infections. Prior to surgical intervention, detailed anthropometric measurements are made of the CL/P deformity to accurately guide the reconstruction.

### 3.1.3. Photography

This is important for assessing cosmetic improvements after surgery. It enables pre- and post-operative comparisons to evaluate the success of the procedure. Serial photographs can help track facial development as the child gets older.

### 3.1.4. Moulding

This intervention aids surgical revision and is based on the malleable nature of tissues at an early age. Its implementation improves the surgical prognosis because some anatomical continuity is restored prior to surgery. Common techniques used to mould the tissues include lip taping, alveolar moulding and nasoalveolar moulding, as well as usage of the Latham device.
Lip taping provides mechanical pressure on the tissues to help mould them into shape and reduce the need for early orthodontia. Alveolar moulding involves the application of a plate that helps to adjust palatal growth. When an additional nasal stent is employed, the process can be modified to nasoalveolar moulding. However, this technique can lead to ulceration and mega nostril. The Latham device offers a mode of active moulding; however, it hasn’t gained wide acceptance because it is carried out under general anaesthesia and can cause growth disturbances (Bailey et al., 2013).

3.1.5. Primary surgery

The principles of surgery aim to restore the anatomical continuity of the defects so as to improve functional ability, improve the aesthetic outlook and encourage normal facial development. The time at which surgical intervention is recommended varies according to the type of CL/P present (Table 12.3). The ‘rule of 10’ can also aid in determining when to operate. This entails haemoglobin in excess of 10 g/dl, an age of 10 weeks and a weight of 10 lbs for cleft lip. In a cleft palate case, typically a 10-month-old infant weighing more than 10 kg with a total leukocyte count of less than 10,000/μl are operated on.

The basic principle of repair involves surgical incisions directed toward restoring the displaced tissues to their normal positions. It involves the use of local flaps to reconstruct the defects, as well as repositioning and suturing the displaced muscle fibres. Millard’s rotational advancement flap is the most common approach employed in the case of unilateral cleft lip. It involves advancement of a mucocutaneous flap from the lateral portion of the lip into the cleft within the superior portion of the lip. It can also be used in a one- or two-stage form for bilateral cleft lip. A triangular flap technique (Tennison–Randall method) can be used to repair cleft lip but is reserved for more severe cases. The length of the medial lip can be extended by the application of a triangular flap from the lower part of the lateral lip. A drawback of this technique is that the scar may not be as cosmetically acceptable.

<table>
<thead>
<tr>
<th>Table 12.3. Timing of primary surgery in CL/P defects.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cleft lip alone</td>
</tr>
<tr>
<td>Unilateral: single operation at 5–6 months</td>
</tr>
<tr>
<td>Bilateral: single operation at 4–5 months</td>
</tr>
<tr>
<td>Cleft palate alone</td>
</tr>
<tr>
<td>Soft palate only: one operation at 6 months</td>
</tr>
<tr>
<td>Soft and hard palate: two operations with soft palate at 6 months and hard palate at 15–18 months</td>
</tr>
<tr>
<td>Cleft lip and palate</td>
</tr>
<tr>
<td>Unilateral: two operations with cleft lip and soft palate at 5–6 months. Hard palate with lip revision at 15–18 months</td>
</tr>
<tr>
<td>Bilateral: two operations</td>
</tr>
<tr>
<td>Cleft lip and soft palate at 4–5 months. Hard palate with lip revision at 15–18 months</td>
</tr>
</tbody>
</table>

Source: Adapted from Bailey et al. (2013).
The Von Langenbeck procedure is the oldest cleft palate operation and is still being used today. It involves the production of bipedicled mucoperiosteal flaps from the edge of the cleft which are then advanced medially. By maintaining the anterior attachment of the flap with the alveolar margin, it enables the flap to be bipedicled. Newer techniques such as V–Y retroposition of the palate can also be performed and are typically conducted at 12–18 months of age.

Asymptomatic submucosal cleft palates are managed conservatively; however, in the presence of speech defects and velopharyngeal incompetence, surgical correction is an accepted treatment modality. Surgery focuses on palatal repair and may be associated with pharyngoplasty. Furlow palatoplasty is one of the surgical techniques used and is effective when there is a small velopharyngeal gap (Thorne et al., 2014; Zenn et al., 2012; Marks and Marks, 1997).

### 3.1.6. Secondary surgery

Surgical revision should not be conducted until 2 years after the primary procedure. It aims to correct residual defects that have persisted or disproportional development of the face that may require augmentation. Common procedures include rhinoplasty, veloplasty, alveolar bone grafting, orthognathic surgery and pharyngoplasty.

#### 3.1.6.1. Alveolar bone grafting

This revisional procedure is conducted to correct residual alveolar clefts with the aim of promoting normal dental development and reducing the incidence of oronasal fistulas. Pre-surgical orthodontic treatment is often beneficial for improving the post-operative outcome. Alveolar bone grafting is best conducted between the ages of 8 and 11 years and employs cancellous bone from the iliac crest or tibia plateau. It has borne very good results until now and is thus a popular procedure.

#### 3.1.6.2. Orthognathic surgery

This is typically performed to correct poor maxillary growth but is delayed until facial growth has reached completion at 16–19 years of age. Poor maxillary growth can contribute to pseudoprognathism and may not be amenable to orthodontic device therapy alone.

#### 3.1.6.3. Septorhinoplasty

Open septrhinoplasty is effective for correcting persistent nasal cartilaginous deformities, usually on the side of the cleft deformity. The two most common deformities revised are a collapsed lateral nasal cartilage on the side of the cleft and dislocation of nasal septum into the non-cleft nostril.
Pharyngoplasty or the pharyngeal flap procedure is effective in correcting velopharyngeal insufficiency and aids in improving soft palate function. The procedure usually involves the anterior transposition of posterior pharyngeal wall tissue to reduce the velopharyngeal gap by suturing two palatopharyngeal muscles in the middle. There are three types of pharyngoplasty in practice: (1) a single flap from the posterior pharyngeal wall united to the posterior section of the soft palate; (2) bilateral mucomuscular flaps transposed from the lateral pharyngeal walls to obtain a horizontal position on the posterior pharyngeal wall; and (3) an implant used behind the mucosa of the posterior pharyngeal wall (Thorne et al., 2014; Zenn et al., 2012; Marks and Marks, 1997).

### 3.1.7. Post-operative care

Complications may ensue immediately after surgery, including wound dehiscence and infection. Late complications are more common and have been summarised in Table 12.4. In the post-operative period, good oral hygiene is essential and patients are started prophylactically on broad-spectrum antibiotics. Dehiscence secondary to infection should not be re-sutured until the lip has become soft. It is also recommended to commence a liquid diet within a few hours of surgery. Elbow restraints can be helpful in attempting to prevent the child from physically tampering with the repaired facial structures. To conclude, both CL/P and craniosyntosis syndromes have a strong genetic heritage and their respective management are closely tailored to each individual patient (summarised in Figure 12.7).
Figure 12.7. Summary of craniosynostosis syndromes and CL/P.

REFERENCES


