Cleft lip and palate: diagnosis and management

Cleft lip and palate is the most common congenital facial anomaly in children, which can affect appearance, speech, hearing, growth, psychosocial wellbeing and social integration. This article provides an overview of the condition for the benefit of all health-care professionals.

Cleft lip and palate is the most common congenital facial anomaly in children with approximately 1000 new cases per year in the UK (Mossey and Castilia, 2003). Clefts can affect appearance, speech, hearing, growth, psychosocial wellbeing and social integration (Dixon et al, 2011). Despite its prevalence a nationwide survey of parental perceptions on cleft lip and palate care in 2006 demonstrated a disproportionate lack of knowledge and understanding about the condition (Cleft Lip and Palate Association, 2007). This article gives an overview of the condition for the benefit of health-care professionals not working in a specialist paediatric cleft unit.

Classification

Clefts of the lip and palate can be broadly classified into clefts of the lip and/or palate and isolated cleft palate. They are epidemiologically, embryologically and aetiologically unique.

Cleft lip and/or palate

The commonest form is cleft lip and/or palate, which involves disruption of the embryological tissue planes above the lip that may continue into the hard and/or soft palate. Cleft lip is defined as a congenital abnormality anterior to the incisive foramen. The cleft can be complete, incomplete, unilateral or bilateral and may or may not involve the alveolus.

Clefts of the lip may extend to involve the primary palate and the secondary palate. Cleft lip and/or palate accounts for around 60% of referrals (whereas the other 40% are composed of isolated cleft palates) (Mosahebi and Kangesu, 2006). Of the cleft lip and/or palate referrals the breakdown is as follows:

- 25% cleft lip alone (unilateral or bilateral)
- 25% unilateral cleft lip and palate
- 10% bilateral cleft lip and palate.

The prevalence varies with ethnicity and the type of cleft. Asian and Amerindian ethnicities have the greatest reported birth prevalence rates, as high as 1 in 500, while European derived populations have prevalence rates of approximately 1 in 1000 (intermediate) and patients of African origin have the lowest reported birth prevalence rates at 1 in 2500. There are also differences between the laterality of clefts and between genders, with the left side of the lip involved more commonly than the right side and males being twice as likely to be affected than females (Mossey et al, 2009).

Approximately 35% of cleft lip and/or palate patients have another congenital anomaly and there are 200 syndromal associations (Wong and Hägg, 2004). The inheritance patterns are broadly characterized into chromosomal (trisomy 13 or 21), Mendelian (van der Woude syndrome) or sporadic.

Cleft palate

Cleft palate is defined as a congenital abnormality of the secondary palate: this forms part of the hard palate and all of the soft palate. This includes:

- Submucous clefts
- Partial clefts of the soft palate
- Complete clefts of the soft palate with incomplete clefts of the hard palate
- Complete clefts of the soft and hard palate

Isolated cleft palates account for the remaining 40% of referrals. Although the incidence is racially uniform at 0.5 per 1000 live births there is up to a 50% association with any one of 400 syndromes including single gene defects such as Treacher Collins syndrome, Stickler syndrome, Pierre Robin syndrome and velocardiofacial syndrome (Wong and Hägg, 2004).

Embryology and pathology

Between the 4th and 10th weeks of embryogenesis a series of coordinated cellular activities (migration, growth,

Dr Bilal G Taib *is Academic Foundation Year 2 Doctor in the Department of Gastroenterology, Royal Liverpool University Hospital, Liverpool L7 8XP,* **Dr Adnan G Taib** *is Academic Foundation Year 1 Doctor in the Department of Cardiology, Royal Derby Hospital, Derby,* **Mr Andrew C Swift** *is Consultant Ear, Nose and Throat Surgeon and Rhinologist in the Department of Otorhinolaryngology, Head & Neck Surgery and Thyroid, Aintree University Hospital, Liverpool, and* **Mr Simon van Eeden** *is Consultant Cleft and Maxillofacial Surgeon in the Department of Cleft and Maxillofacial Surgery, Alder Hey Children's Hospital Foundation Trust, Liverpool*

Correspondence to: Dr BG Taib (bgani56@gmail.com)

differentiation and apoptosis) occur resulting in the development of the primary and eventually the secondary palate. The primary palate is a triangular area of the hard palate, anterior to the incisive foramen. It includes a portion of alveolar ridge, as well as the four incisor teeth. At approximately 5 weeks' gestational age the primary palate begins to form by the growth then subsequent fusion of the medial nasal, lateral nasal and maxillary processes. The lateral nasal processes before their union with the maxillary processes undergo rapid mitotic division rendering them susceptible to teratogenic insults or growth disturbances resulting in primary palate defects (Sperber, 2002). Another potential deficiency of the palate may arise from a lack of mesodermal reinforcement along the lines of fusion (Tewfik et al, 2015). This is therefore a complex process susceptible to toxins especially if there is an underlying genetic predisposition. Animal studies have shown that uncoupling of embryonic cellular processes governing the midface, through alterations in proteins such as sonic hedgehog, bone morphogenetic and fibroblast growth factor proteins can result in the presence of a cleft lip and/or palate or an isolated cleft palate (Cox, 2004).

The secondary palate forms the remaining hard palate and the entirety of the soft palate. It arises from two separate palatal shelves extending from the maxillary processes during the 6th week of embryogenesis. The shelves are initially oriented vertically on either side of the tongue. With head extension and mandibular growth, the tongue is withdrawn allowing the palatal shelves to assume a horizontal position and meet in the midline. This is followed by subsequent midline fusion of the hard and soft palate (*Figure 1*).

By the end of the tenth week the primary palate, secondary palate and nasal septum have fused. This divides the oral and nasal cavities into separate entities allowing them to function simultaneously. Theories suggest that some clefts of the secondary palate occur because of failure of the intervening tongue to withdraw, which then impedes elevation and subsequent fusion of the palatal shelves (Tewfik et al, 2015).

Aetiology

The causes of isolated cleft palate and cleft lip and/or palate remain largely unknown but are thought to be a combination of genetic and environmental factors.

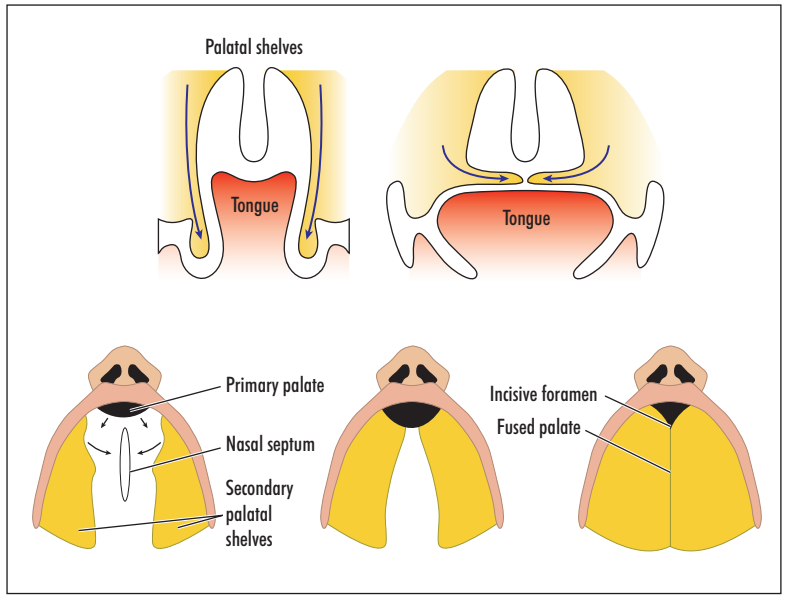
Many clefts run in families and these may be syndromic or non-syndromic occurrences. For non-syndromic clefts, the risk of unaffected parents with a cleft lip and/or palate child having another affected child is 4%. Similarly, if one parent is affected by a non-syndromic cleft lip and/or palate the chance of having an affected child is 4% (Goodacre and Swan, 2011). Cases of isolated cleft palate show a familial clustering effect. For cleft lip and/or palate it is estimated that there is a 60% concordance rate in monozygotic twins and 10% in dizygotic twins (Grosen et al, 2011). The commonest syndrome associated with cleft lip and palate is van der Woude syndrome (Dixon et al, 2011), while the most frequent syndrome associated with cleft palate is 22q11 deletion syndrome (Fullman and Boyer, 2012). Through understanding these various syndromes, many genes have been identified which may contribute to non-syndromic cleft lip and/or palate; however, an isolated gene is yet to be established as the sole cause.

A number of environmental causes for clefting have been identified. Smoking during pregnancy has a dose–response relationship with orofacial clefting (Little et al, 2004). Estimations suggest that smoking contributes to 4% of cleft lip and/or palate (Honein et al, 2007). Alcohol consumption during pregnancy also increases the risk of isolated cleft palate (Goodacre and Swan, 2011). Other maternal risk factors include the use of steroids, anticonvulsants (phenobarbital and phenytoin), retinoids, diabetes and nutritional deficiencies (zinc, folic acid and vitamin A) (Lorente and Miller, 1978; Park-Wyllie et al, 2000).

Diagnosis

Antenatal scans commonly carried out at 20 weeks' gestation provide up to 45% of the diagnoses of cleft lip and/or palate (Cleft Lip and Palate Association, 2007). In 2012 39% (406 children) of children had their cleft diagnosed during the antenatal period; 56% of cleft lips, 79% of unilateral cleft lip and palates and 78% of bilateral cleft lip and palates were diagnosed antenatally while only 1.1% of cleft palates were diagnosed antenatally (Fitzsimons et al, 2013). Although antenatal diagnosis allows parents to prepare themselves for the baby's birth and subsequent care pathway, it may negatively impact the rest of pregnancy as a result of the psychological burden on parents. It is therefore important to provide parents with appropriate and timely support. Once an

Figure 1. Formation of the secondary palate.



antenatal diagnosis has been made, the scanning unit is obligated to refer the parent(s) to the designated regional cleft network within 24 hours. Once this referral has been received the specialist cleft nurse will contact the parents within 24 hours in accordance with national guidelines. Subsequently the clinical nurse specialist will arrange a meeting in person with the parent(s) to provide the necessary support and information and, if appropriate, arrange a meeting with the cleft team.

Of those children not diagnosed antenatally in 2012, 72% were diagnosed at birth. Most of the cleft lips, unilateral cleft lip and palates and bilateral cleft lip and palates (82.5%) were diagnosed at birth but 32.4% of the cleft palates were not identified until later, with 6.9% of these being diagnosed beyond 1 month after birth (Fitzsimons et al, 2013). This reflects the fact that isolated cleft palates are more challenging to detect on the newborn examination. This is caused by a failure to directly visualize the palate adequately using a torch and a tongue depressor, but instead relying on diagnosis through digital palpation of the palate (Habel et al, 2006). In both groups delayed detection inadvertently increases the psychological burden on parents.

Submucosal clefts may also be overlooked at birth if visual inspection of the palate is not undertaken. They can present with the 'classic triad' of features: a bifid uvula, a zona pellucida (central zone of lucency in the soft palate) and a bony defect of the hard palate (Calnan, 1954).

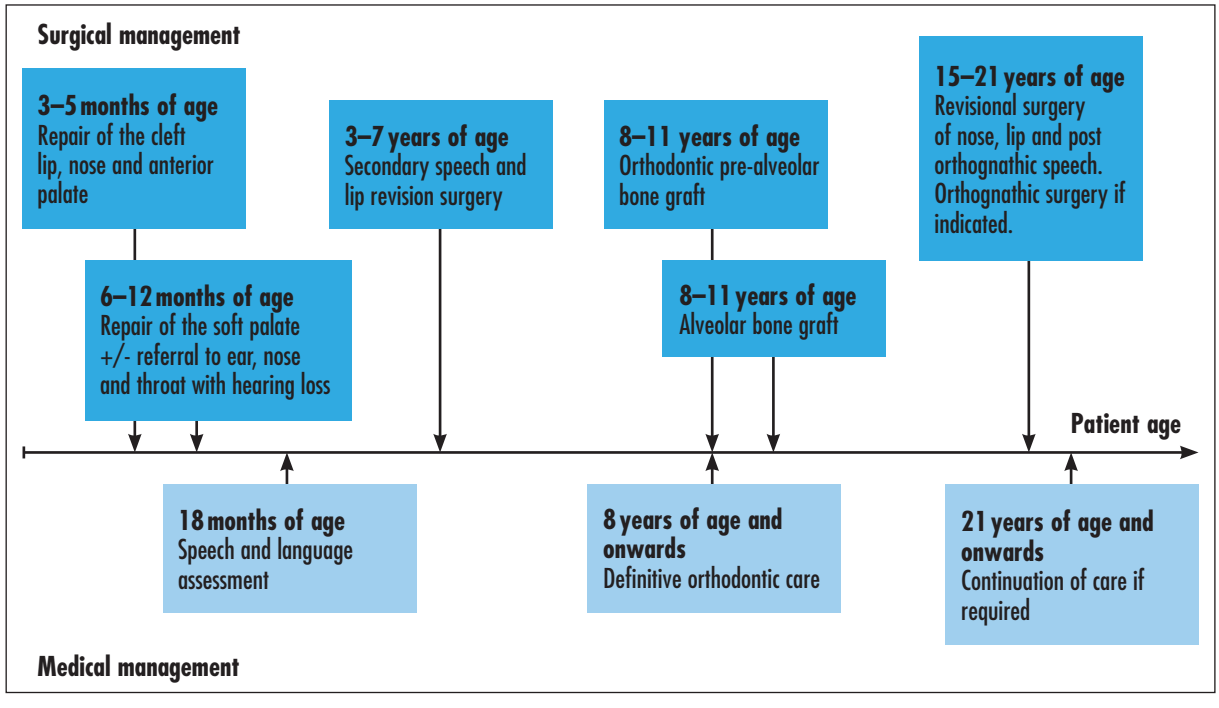
Management

Since the 1990s there has been a large change in the delivery of cleft care in the UK from a locally based, poorly coordinated service to a regional, centralized hub and spoke system. This re-configuration followed the Clinical Standards Advisory Group report published in 1998, which recommended that cleft care should be reorganized and centralized. The report found that there were 57 cleft centres in the UK with poorer patient outcomes when compared to centralized models of care in Europe. There are now nine regional cleft centres configured on a hub and spoke basis enabling multidisciplinary care, high volume operating, pooling of resources and a focus on audit and quality of care. The surgery is carried out within the regional centre and multidisciplinary treatment and support is provided thereafter from infancy to early adulthood both at the regional centre and also more locally in outreach clinics. Initial reports from the follow-up Clinical Service Advisory Group II study (Sandy et al, 2012) show an improvement in outcomes for cleft patients when compared to the original Clinical Service Advisory Group study (Bearn et al, 2001).

The number of primary surgical operations will vary according to cleft type, ranging from one operation to repair a cleft lip and isolated cleft palate to four operations to repair a bilateral cleft lip and palate. This may be followed by speech surgery for those with velopharyngeal incompetence, revisional lip and nose surgery to improve appearance and function and orthognathic surgery to improve skeletal and occlusal relationships (*Figure 2*).

Children born with cleft lip and palate may have feeding difficulties, psychosocial issues, speech and language problems, facial growth retardation, dental irregularities and hearing loss. The aim of the cleft care pathway, which spans from infancy to adulthood, is to minimize the impact of such sequelae (*Table 1*). This is achieved through a multidisciplinary team approach:

Figure 2. Timeline summarizing the management of cleft lip and palate.



- Clinical nurse specialists provide support to expectant parents, perinatal care, feeding advice, home visits and perioperative care. They ensure continuity of care from the cleft centres
- Psychologists specialize in minimizing the psychological impact of the cleft (including treatment) on the children and their families' lives
- Cleft surgeons undertake the surgical management
- Speech and language therapists are responsible for the identification, assessment and management of speech and language disorders
- Paediatric dentists provide general oral hygiene and dental advice, monitor dental health and development, and prescribe necessary interventional dental care
- Orthodontists monitor dental development and jaw growth. They are involved in the perioperative management of alveolar bone grafting, and provide interceptive orthodontics (teeth straightening) for non-orthognathic and orthognathic cases
- Paediatricians are vital in addressing any associated medical problems in cleft patients
- Audiovestibular physicians and audiologists monitor hearing on a regular basis as children with cleft palates have a higher incidence of otitis media with effusion and chronic ear disease
- Ear, nose and throat surgeons carry out ventilation tube insertion; they liaise closely with cleft surgeons in order to couple any operative procedures
- Geneticists provide advice on genetic diagnosis, management and on the reproductive implications of having a cleft.

Primary surgery

The aim of primary surgery is to minimize the stigmata of the cleft, restore normal function including speech and hearing, and promote normal growth and psychosocial development.

Good evidence for one technique or protocol over another is still lacking within the cleft community because of low numbers of cases, the time taken to evaluate the technique or protocol (birth to adulthood) and the difficulties associated with randomized controlled trials in this cohort of patients. There is therefore a wide variation in protocols, techniques and timing for the closure of clefts of the lip and/or palate. However, good longitudinal cohort studies have been reported in the literature and these form the basis for protocols and timings used in many of the cleft centres in the UK (Semb, 1991).

In the UK the first surgery is undertaken once feeding patterns have been established and birth weight recovered. Cleft lip and nose repair usually takes place between 3 and 5 months of age. If there is a complete cleft of the lip and palate the hard palate is usually repaired at the same time as the lip (*Figure 3*). There are many different techniques described for lip closure with rotation-advancement techniques being the most widely used across the world (Sitzman et al, 2008).

Isolated cleft palate repair and repair of the soft palate in unilateral cleft lip and palate and bilateral cleft lip and palate cases usually takes place between 6 and 9 months. In the UK a high proportion of cleft surgeons repair the soft palate by carrying out a radical dissection of the abnormal palatal musculature to pos-

Table 1. The main complications and subsequent management of cleft palate and/or lip and isolated cleft palate

Problems associated with cleft palate and/or cleft lip	Complication	Management
Feeding	Babies with cleft palates have a fistula between the oral and nasal cavities. The baby is unable to compress the breast, between the tongue and palate, to generate a negative suction pressure. This prevents the newborn from breastfeeding	In babies with clefts of the palate an orthodontic teat and squeezable bottle are introduced soon after birth. This helps control the rate of feeding and overcome the pressure gradient (Masarei et al, 2007)
Hearing loss	The incidence of otitis media with effusion has been reported as high as 97% in cleft palate patients (Dhillon, 1988). Eustachian tube patency is compromised because of a lack of tensor muscle fibre anchorage. The sequelae of otitis media with effusion includes physical phenomena such as cholesteatomas, atelectasis and ossicular fixation but also developmental consequences impacting on speech, social integration and educational attainment (Sharma and Nanda, 2009)	These patients require regular audiology surveillance. Prophylactic ventilation tubes have a complication rate of ~25% and as 50% of cases of 'glue ear' resolves spontaneously within 3 months a more conservative treatment approach has prevailed (Gani et al, 2012). This includes the use of hearing aids as an alternative to ventilation tubes
Speech	Clefts of the palate may lead to velopharyngeal insufficiency which is commonly associated with nasal air escape. This resonance disorder affects both the intelligibility and confidence of these children (Sie, 2006)	It is important to monitor speech in all children with a history of cleft palate. Timely palatal repair will normalize speech in the majority of cleft palate patients, but up to 50% of cases will require speech therapy and a proportion (5–50%) will require further surgery
Dentition	Dental irregularities often occur when the cleft involves the alveolus. Dental anomalies range from supernumerary teeth, altered teeth dimensions and development (Vettore and Sousa Campos, 2011)	The aim of dental surveillance and treatment, which begins at birth, is to normalize tooth eruption, mask maxillary hypoplasia and provide a functional and aesthetic bite (Mosahebi and Kangesu, 2006)

teriorly reposition the palatal muscles and reconstitute the muscular slings of the velum as described by Brian Sommerlad in the UK. Use of this technique has produced the best speech outcomes for cleft palate repair (Sommerlad, 2003).

Patients with a submucosal cleft do not always need surgical management. Surgery is indicated if the infant presents with feeding difficulties related to velopharyngeal incompetence and in older patients presenting with speech difficulties secondary to velopharyngeal insufficiency. Repair is as described for soft palate repair above. Caution should also be exercised in patients with sub-mucosal clefts in which an adenoidectomy is indicated (e.g. for chronic ear conditions). The removal of the adenoids may lead to velopharyngeal insufficiency with subsequent speech problems necessitating a combined cleft and ear, nose and throat assessment.

Those patients with an alveolar cleft will need bone grafting to restore the continuity of the alveolar arch to support dental development and growth. This is not usually carried out at the time of the initial lip repair (because of poor growth results following early bone grafting) but is delayed until the eruption of either the permanent upper lateral incisor or canine on the cleft side. This is known as secondary bone grafting and was pioneered by Boyne and Sands, New Jersey, USA, in the 1970s. They showed that the bone defect is best filled with osteogenic cancellous bone fragments taken from the iliac crest, during the mixed dentition phase (between 7–11 years old), ideally when the root of the lateral incisor or canine is half to two-thirds formed. This allows tooth eruption to occur through grafted bone while the graft supports the maxillary arch. This then reconstitutes the maxillary alveolus and facilitates definitive orthodontic treatment (Lilja, 2009). However, there is still variation in practice internationally with regard to the timing of alveolar bone grafting and the donor site used.

Secondary surgery

Secondary surgery in cleft patients usually takes place in order to:

- Normalize palatal function and therefore speech in patients with clefts of the palate
- Repair oro-nasal fistulae
- Address aesthetic concerns relating to lip and nasal appearance
- Correct facial growth disturbances.

The timing of secondary surgery is dictated by both national specifications and patient/parental concerns. As far as speech is concerned the aim is to have speech within normal limits by the time the child is 5 years of age. This includes both palatal function (which can be corrected surgically) and articulation (which can be corrected with speech and language therapy). Functional oro-nasal fistulae and those impacting on the child's quality of life need to be repaired as soon as possible.

Lip revision surgery is carried out on patient or parental request, as long as the expectations are reasonable, and can be carried out any age.

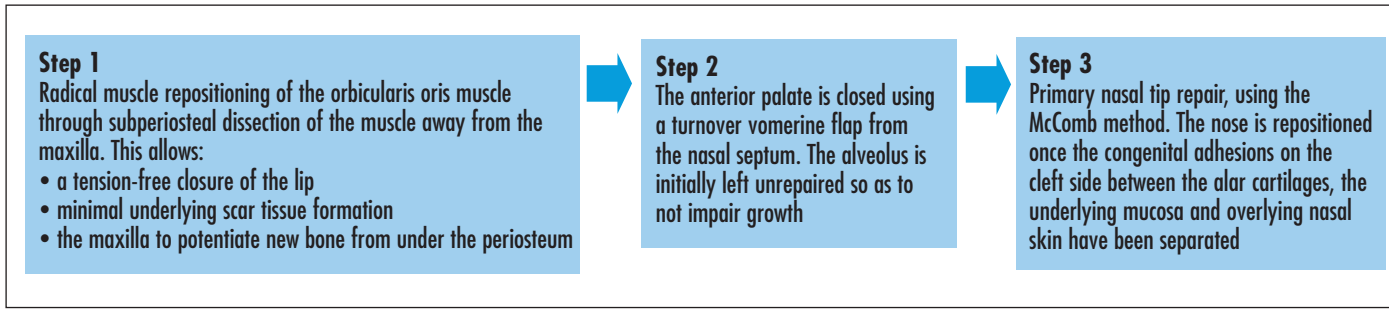
Orthognathic surgery to correct facial growth disturbance is indicated in up to 80% of patients according to some studies. Apart from embryological and intrinsic deficiencies in bone formation, surgical intervention in cleft patients is thought to have a major effect on craniofacial growth in these patients, with the maxilla being most affected (Berkowitz, 2006). Maxillary hypoplasia results in a class III skeletal relationship and a class III malocclusion with a characteristic facial appearance that is best resolved with psychological support and orthognathic surgery during late adolescence at the end of growth. Orthognathic surgery may involve a combination of maxillary, mandibular and chin surgery to address individual skeletal and occlusal needs.

Similarly definitive rhinoplasty to address both functional and aesthetic concerns should be carried out once growth has been completed and should follow orthognathic surgery. In some cases, however, earlier surgery may be indicated to address severe psychosocial issues.

Immediate postoperative emergencies

The two most commonly encountered problems immediately after primary cleft surgery are bleeding and airway obstruction. Infants are obligate nasal breathers and may therefore have difficulty breathing following narrowing of the nasal airway during cleft lip and anterior palate repair. Suctioning techniques and gentle placement of a nasopharyngeal airway can help alleviate problems with the airway (Mosahebi and Kangesu, 2006).

Figure 3. Early surgical repair of cleft lip, nose and anterior palate at 3 months of age.



Airway obstruction following posterior palate repair is more difficult to manage. A combination of narrowing of the velopharyngeal gap, soft tissue swelling, intra-palatal haematoma and nasal obstruction all contribute to respiratory obstruction (Agrawal, 2009). Again placement of a nasopharyngeal tube will alleviate airway obstruction in most of these cases but should be carried out with care to avoid damage to the repair.

Significant postoperative bleeding, although rare, is a surgical emergency. Minimal oozing is expected within the first 12 hours of surgery. The most common bleeding sites are from the bare membranous bony palate and from the edges of the mucoperiosteal flap (Agrawal, 2009). In cases where bleeding is profuse and sustained the surgeon should be immediately alerted and the child prepared for urgent return to theatre.

Conclusions

The diagnosis of a cleft lip and/or palate is only the first step of the care which continues right through to adulthood. The complex multidisciplinary team approach is delivered through a centralized system which has satellite services. **BJHM**

Conflict of interest: none.

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KEY POINTS

- Cleft lip and or palate and isolated cleft palate are epidemiologically, embryologically and aetiologically unique.
- All newborn examinations require direct visualization of the palate and not just digital palpation.
- In the UK cleft lip and nose repair usually takes place between 3 and 5 months of age and palatal repair usually takes place between 6 and 9 months.
- Cleft care pathway provides multidisciplinary support well in adult life.
- Airway obstruction and bleeding are the two most common postoperative complications.