Anaesthesia *for Cleft Lip and Palate Surgery*

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Introduction

Cleft lip and palate are the most common craniofacial abnormalities seen worldwide. If left uncorrected, cleft lip can have significant social and psychological consequences for both the child and family. A cleft palate interferes with feeding and speech as a consequence of velo-pharyngeal insufficiency. Clefts are not simply a surgical problem but individuals with clefts or other craniofacial anomalies require the coordinated care of several specialists. These include neonatologists, paediatricians, surgeons, anaesthesiologists, dentists, as well as those in speech pathology, otolaryngology, audiology, genetics, nursing, mental health, and social medicine.[1,2]

Historically, surgery was planned according to "Kilners rule of 10": at 10 weeks of age, a weight of 10 lb and haemoglobin 10gm.dl for cleft lip, and 10 months, 10 kg and a haemoglobin of 10 gm.dl for cleft palate. In recent times, surgery has been performed earlier because the incidence of middle ear infections is less, speech development is more normal, and more effective orthodontic treatment can be instituted. In expert hands it is no longer unusual for neonatal cleft lip repair to be done.[1,3,4] The reduced scarring as a consequence of better wound healing,[5] together with psychological advantages of early bonding and breast-feeding in a neonate are clear, but should be balanced against the difficulties of achieving a good cosmetic result with small, poorly defined tissues.

Prevalence

The prevalence of clefting ranges from 1:300 -1200 live births for cleft lip and 1:2500 for cleft palate.[1-3] These figures vary according to the investigator, the country and racial group studied, or whether the isolated cleft lip or palate, or combined cleft and palate figures are quoted. Cleft deformities are more common in people of Asian descent (1:500), less in Caucasians (1:1000), whilst the African population group has the lowest prevalence (1:2500). The sex ratio in patients with clefts varies. Cleft lip and cleft lip/palate is more prevalent in males whereas cleft palate is more prevalent in females. Males are more likely to have severe disease.

Most clefts are caused by the interaction between genetic and environmental factors.[2] Heredity clearly plays a role. The exact gene locus has yet to be determined, but a number of candidate genes have been identified. There is an increased incidence of clefting in siblings of those with cleft lip or palate; van der Woude syndrome is an autosomal dominant disorder. Parental age is also a factor. There is an increased incidence in children of parents older than 30 years. Environmental factors play a role when the genetically susceptible genotype is triggered. Maternal alcohol and phenytoin sodium consumption have been shown to increase the incidence of both cleft lip and palate.[2]

Cleft lip is unilateral in 75-80% of cases, with the left side more commonly affected; 20-25% are bilateral. A spectrum of abnormality ranging from a notch in the upper lip lateral to the midline, to a bilateral cleft extending up into the nose and alveolar ridge (gums) or hard palate may be seen. Midline clefts are rare and are usually a feature of an underlying syndrome.

The spectrum of cleft palate ranges from a bifid uvula and submucous cleft palate with velo-pharyngeal insufficiency, to cleft soft palate; a V-shaped cleft or a wide U-shaped cleft involving the bony hard palate. Isolated midline cleft palate may represent a different malformation process with different syndrome associations. Velopharyngeal insufficiency leads to regurgitation of milk or food through the nose in babies, and nasal speech in older children. Failure to thrive is a common consequence of these feeding difficulties.[6, 7]

History

Cleft lip repair has a fascinating history, both from the surgical and anaesthetic point of view. Records of the earliest attempts at cleft lip repair in China date back to 390 AD.[1] The repair involved freshening the edges of the cleft and passing straight "needles" through each side of the wound. The wound edges were approximated, using a thread in a figure of eight. As recent as 1930, a wire was still passed through the open structures of the lip, and approximation was achieved by twisting the wire until the sides collapsed together.[1] This all changed with improved surgical techniques and the advent of safer general anaesthesia and muscle relaxants.

Current surgical interventions focus on completing the failed embryological development through complete anatomical dissection and geometric rearrangement of muscle, mucosa, and skin flaps, in an attempt to achieve a more satisfactory functional and cosmetic result.[1]

The development of anaesthesia for cleft lip and palate surgery make interesting reading.[8-12] Innovative techniques introduced by pioneers have impacted significantly on the development of paediatric anaesthesia. In 1847 John Snow reported the use of open drop ether and chloroform on 3-6 week old infants undergoing cleft lip repair. The unprotected airway and the potential for airway obstruction restricted the surgeon to quick simple surgery. Cleft palate surgery was considered too hazardous at the time.

By 1900, pharyngeal insufflation allowing continuous administration of vapour was used. In the 1920's intra-tracheal insufflation using a thin catheter provided better airway protection[10, 12] but it was only later in the 1930's that endotracheal tubes were introduced.

In 1937, Ayre introduced the T-piece.[11] He noticed that by eliminating rebreathing, the popular method at the time, the infants fared better. The addition of the open-ended bag to the T-piece by Jackson Rees allowed manual ventilation. This then became the established technique, and coincided with the advent of muscle relaxants.

Embryology[1, 2]

During facial morphogenesis, neural crest cells migrate into the facial region, where they form the connective tissue, skeletal tissue and all dental tissue except the enamel (ectoderm). Vascular endothelium and muscle are of mesodermal origin.

The facial structures develop from five prominences; the midline fronto-nasal prominence, and bilateral maxillary and mandibular prominences. These fuse around the primitive mouth or stomadeum. The maxillary prominences grow towards each other from both sides and towards the medial nasal prominence, eventually fusing to form the midface. Migration of these cells occurs from approximately 5 weeks gestation and fusion is complete at approximately 9 weeks. The upper lip is derived from the medial nasal and the maxillary processes

Clefting occurs when there is failure of fusion, or diminished mesenchymal penetration between these migrating embryological processes. Cleft lip is the consequence of failed fusion between the medial nasal and maxillary processes on one or both sides. The cleft may affect only the upper lip, or it may extend more deeply into the maxilla and the primary palate (orofacial cleft). Understandably, a variety of complete, incomplete, unilateral and bilateral clefts can occur. If the fusion of palatal shelves is also impaired, the cleft lip is accompanied by cleft palate. This usually occurs at the junction between the central and lateral parts of the upper lip on either side.

Cleft palate is a consequence of partial or total lack of fusion of the palatal shelves, and can occur if there is defective growth of palatal shelves, failure of the shelves to attain a horizontal position, lack of contact between shelves or rupture after fusion of the palatal shelves. The palate consists of two parts. The primary palate, which is anterior to the incisive foramen, forms the alveolus and the lip, and fuses at 5-6 weeks. The incisive foramen is where the anterior palatal vessels and nerves emerge. The secondary palate is posterior to the incisive foramen, and fuses at 7-8 weeks. The maxilla begins to close anteriorly, and the closure proceeds in a posterior direction where it fuses with the vomer plate and the nasal septum.

Fusion may fail in a foetus with micrognathia (mandibular hypoplasia), for example, where the tongue is forced to move up into the roof of the oral cavity during development. This glossoptosis (posterior displacement of tongue) prevents or disrupts the fusion of the palatal shelves.

Associated syndromes

In the majority of children a cleft is an isolated malformation. However, in two surveys of newborns, the incidence of associated malformations ranged from 22-37%. In another 1000 children surveyed by Shprintzen,[13] 50% had one or more major anomalies, and 22% were syndromic (half of these were unknown and considered unique). Isolated cleft palate has the highest incidence of associated malformation (28-47%), whereas cleft lip and palate ranged from 28-37%, and isolated cleft lip 8-13%.

Approximately 150 syndromes have a cleft as a feature[9, 14]. Most are rare, but many have a degree of mandibular hypoplasia that make both airway management and surgery difficult. The more common of these syndromes include the Pierre Robin sequence; Treacher-Collins syndrome; Goldenhar syndrome; 22q deletion (velocardiofacial syndrome); van der Woude syndrome; Fetal alcohol syndrome, Stickler syndrome; and Opitz syndrome. (See Table 1) Cardiac defects are also relatively common, but may be missed.[15] Congenital heart defects have an incidence of 6-8% in the general population. The quoted incidence for the coexistence of clefts and cardiac defects ranges from 1.3-51%.[15-17] Coexistence is not surprising, since environmental and genetic factors that affect the embryological development of the face may also affect the heart, which develops at roughly the same time.

Bilateral clubfeet should alert the anaesthesiologist to other potential

Table 1: Approximately 150 syndromes are associated with cleft and palate. Most are rare. Some of the more common syndromes are listed below.

Syndrome	Important features
Pierre-Robin	Micrognathia, glossoptosis
Treacher-Collins	Micrognathia, malar hypoplasia, ear defect
22q deletion	Cardiac defect, immunodeficiency, calcium
Downs	Cardiac, macroglossia, atlanto-axial joint
Klippel-Feil	Restricted neck movement, renal
Van der Woude	Autosomal dominant, lip pits, missing teeth
Opitz	X-linked, hyperteleorism, hypospadias
Stickler	Autosomal dominant, myopia, deafness, joints
Kabuki	Cardiac, mental retard, facial dysmorphism
Foetal alcohol syndrome	Cardiac, mental retard,
Oro-facial-digital (OFD)	Oral frenula, skeletal dysplasia, renal
Congenital myopathy	Cleft palate, fish mouth, clubfeet, MH

CLINICAL PICTURES

Figure 1: Neonate with severe micrognathia and Pierre Robin syndrome. This 10-day old presented for tracheostomy because of life-threatening episodes of upper airway obstruction. Branchial arch defects often have an associated ear anomaly.

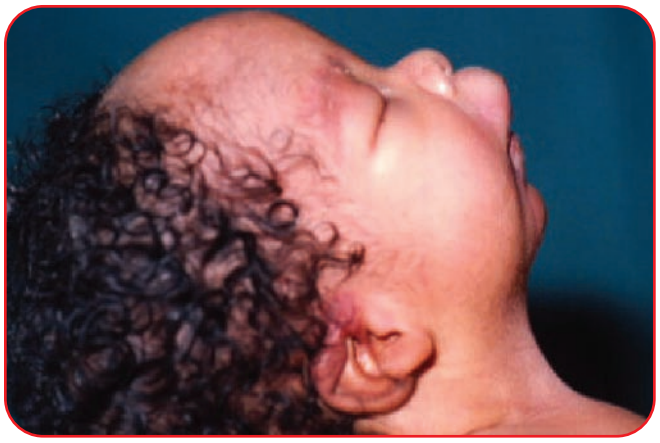


Figure 2: A wide U-shaped palatal cleft is the consequence of failed fusion in the presence of micrognathia and glossoptosis (Pierre-Robin syndrome). This combination, when severe, can cause life threatening upper airway obstruction. Swelling and oedema following surgical repair may worsen the obstruction.

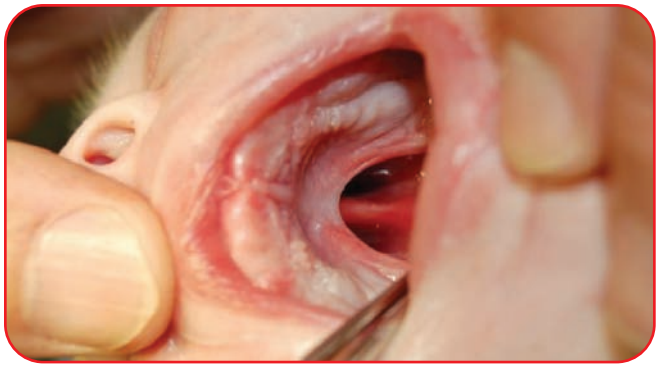


Figure 3: The features of Treacher-Collins syndrome, a branchial arch defect, include maxillary hypoplasia, micrognathia, ear anomalies (microtia) and cleft lip and palate. Congenital cardiac defects should be excluded prior to surgery.

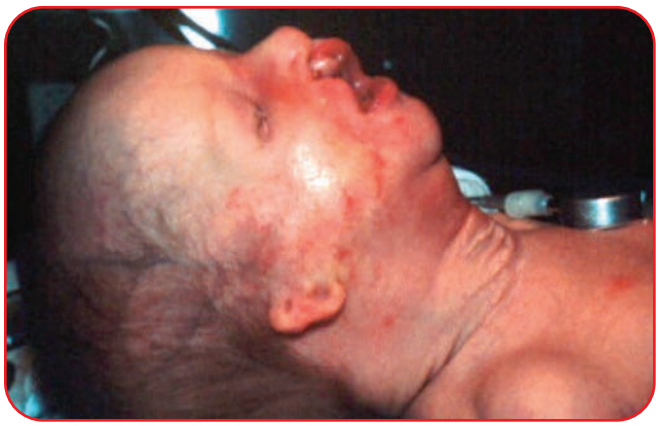


Figure 4: Myopathic facies (expressionless, fish mouth, mild ptosis) are typical features of an underlying congenital myopathy. The combination of delayed milestones, myopathy, cleft palate and clubfeet, are highly suggestive of MH susceptibility.

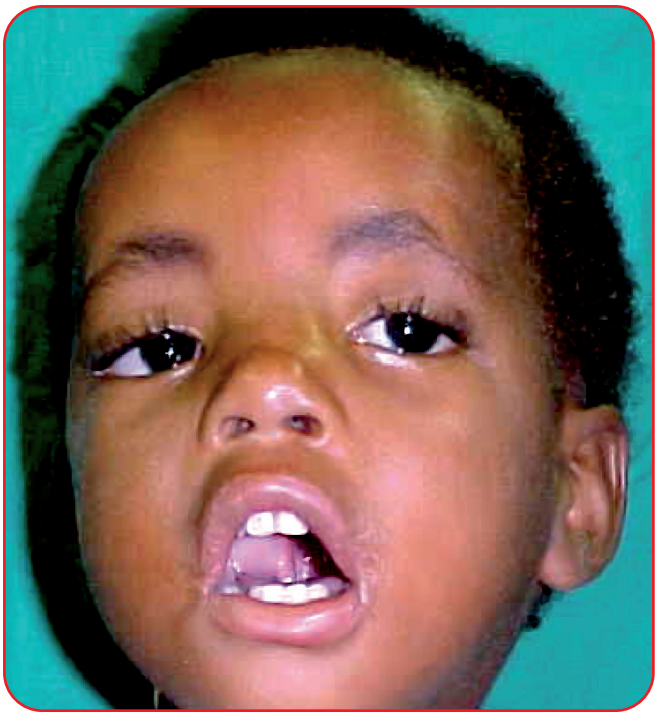
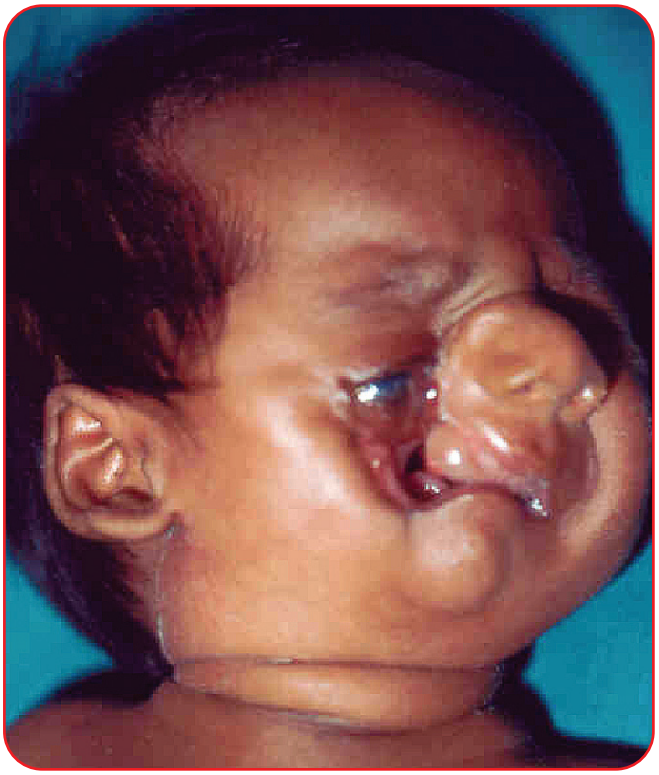


Figure 5: Facial clefts occur when the fusion of the maxillary and fronto-nasal prominences is more incomplete. These infants pose major challenges to both surgeon and anaesthesiologist.



problems. The presence of bilateral clubfeet also increases the likelihood of there being cardiac and renal anomalies. In the author's experience infants with bilateral clubfeet, myopathic facies, cleft palate and delayed milestones from an underlying myopathy, are at risk of developing the signs of malignant hyperthermia (rigidity, hypercarbia).[18]

Anaesthetic considerations

Anaesthesia is relatively straight forward unless there is an associated syndrome, cardiac or renal pathology.[9] There are probably as many anaesthetic techniques described for cleft lip and palate surgery, as there are experts. However there are a number of factors to be considered before providing anaesthesia for these infants.

Preoperative assessment

A thorough preoperative evaluation should reduce the risk of anaesthesia-related morbidity or mortality. Many syndromes associated with cleft lip and palate, are rare. A literature review or an Internet search for details of a particular syndrome prior to surgery is invaluable.

The airway, cardiac defects, renal pathology or other malformations need to be addressed. Many infants fail to thrive. A history of feeding difficulties coupled with a failure to thrive suggests a severe defect. Infants with isolated cleft palate, particularly those with Pierre Robin syndrome, are less likely to thrive than those with combined cleft lip and palate.[6, 7]

Physiological anaemia is likely when surgery is performed at around 3 months of age. Failure to thrive and repeated infections may contribute to the severity of the anaemia. Although blood loss is usually insignificant, the availability of blood should be considered in those where the starting haematocrit is low (<24).

Airway problems

If the infants face is otherwise normal, there should be few problems with either airway maintenance or with laryngoscopy.[19-22] The airway in an infant with abnormal features may challenge even the most skilful anaesthesiologist. Problems with airway management in children with cleft lip or palate were first recognised by Magill almost a century ago.[8, 9] The inherent characteristics of a normal infant's airway can make airway management difficult, particularly in unskilled hands. These airway difficulties may be compounded by a cleft lip or palate.

Preoperative assessment of the airway in infants is not easy when applying methods used in adults, particularly when patient co-operation is required. The pre-operative assessment of the airway in non-syndromic infants can be guided by the findings of two recent studies. Gunawardana, in a prospective study of 800 infants without syndromes, found that 86% of those who had a difficult intubation were associated with a Cormack and Lehane grade lll-lV laryngoscopy; only 2% were associated with a lower grading.[19] The grading was done after external laryngeal pressure was applied. Despite these laryngoscopic findings only 1% - all under 6months - could not be intubated using a styletted endotracheal tube. Bilateral cleft lip, retrognathia and infants under six months were the factors that were associated with difficulty.[19]

In a more recent retrospective study of 985 infants, Xue et al found a 4.77% incidence of difficult laryngoscopy. Age, infants less than 6 months, combined bilateral cleft lip and palate, micrognathia, and to a lesser extent left-sided cleft lip and alveolus, were important risk factors contributing to the difficult laryngoscopy.[20] A similar 1% incidence of failed intubation was recorded in this study. In both these studies a small curved Macintosh blade was used.[19, 20] No studies have looked at the use of a straight blade, which is considered more appropriate for infants.[9]

In order to overcome one of the difficulties associated with the intubation of infants with left-sided defects, some authors have suggested placing a roll of gauze or a dental swab in the defect, to prevent the laryngoscope becoming caught in a deep cleft. In bilateral cleft lip, the vomer or central lip prominence can hinder midline laryngoscopy[21]. A straight laryngoscope blade and a lateral approach may overcome this problem.

Micrognathia or other congenital anomalies involving the upper airway can cause respiratory problems in the immediate postoperative period. Extubation should therefore be delayed until the child is fully awake.[9] Maintaining the airway after deep extubation may be difficult. If subsequent re-instrumentation of the airway is required, disruption of the surgical closure may occur.

Infants with bilateral clefts, wide cleft palates and those with a history of severe feeding difficulties or symptoms of upper respiratory tract infection,[22] have been shown to have a higher incidence of peri-operative respiratory complications. The upper airways of infants with bilateral cleft lip and palate have more reactive airways because they are prone to chronic rhinorrhoea and repeated infections (sinusitis or middle ear infections). The respiratory symptoms may be underestimated because of the structural abnormalities associated with wide clefts.[22] Respiratory depression caused by the inappropriate use of opiate analgesia may worsen these respiratory complications.[4]

Anaesthetic management

Usually no premedication is required for those having early repair. Some anaesthesiologists may use an anti-cholinergic (atropine, glycopyrrolate) to reduce secretions, or to maintain the rate-dependant cardiac output in small infants. Most would use an inhalation induction to ensure that intubation is possible before using muscle relaxants. South-facing RAE tubes are most useful for endotracheal intubation. These RAE tubes have a preformed "knee" that may be too long for a particular infant, and endobronchial intubation may occur. Fixing the "knee" lower on the mandible may overcome this problem. Cutting the tube to an appropriate length is less ideal since the sharp edges thus created may traumatise the airway, unless the end is filed smooth. If a Boyle-Davis gag is used for the palate repair, ensure that the RAE tube does not become snared within the blades of the gag, and when the gag is opened, ensure that the endotracheal tube is not kinked or pushed down into a bronchus.

A throat pack should always be used to prevent soiling of the airway or ingestion of blood. The gauze throat packs usually

that may reduce the incidence of sore throat. Whatever is used, it **must** be removed prior to extubation. Failure to remove the pack has caused death in some of these infants.

Historically, a variety of techniques have been used for maintenance of anaesthesia. The choice is usually dictated by budgetary constraints, rather than patient benefit in many instances. Sevoflurane, isoflurane, halothane and even desflurane are used with or without opiate or regional analgesia.[4,22-26] Total intravenous anaesthesia (TIVA), using a remifentanil–propofol infusion compared favourably with sevoflurane-fentanyl anaesthesia in a small group of infants[23] Ketamine alone or in combination with halothane or ether,[26] is still used in developing countries where resources are limited.[26]

The surgeon usually infiltrates the surgical field with local anaesthetic containing adrenaline.[27] The intraoperative stimulation caused by the endotracheal tube when the head is moved can be overcome by correct tube placement, deepening the level of anaesthesia or by the addition of opiates. Remifentanil is becoming increasingly popular because of its predictable short action, while some advocate small doses of morphine or fentanyl to suppress these airway reflexes. These protective airway reflexes may be suppressed by topical anaesthesia but is probably best avoided. Opiates do minimise postoperative upset, allowing these infants to emerge from anaesthesia slowly with less crying, reducing the risks of bleeding and or swelling. Particular care should be taken with opiates in those who are at risk of apnoea or respiratory obstruction. Similar smooth arousal can be achieved with an infra-orbital nerve block, but without the accompanying risk of apnoea or respiratory depression.

Careful suctioning of the mouth and pharynx and a check for oozing from the surgical site prior to extubation is prudent. Bleeding may delay extubation, but coughing on an endotracheal tube can promote bleeding. The timing of the extubation is therefore important. Extubation in the left lateral position when the child is fully awake is considered safest. Pressure from the Boyles Davis gag may cause swelling, oedema or ischaemia of the tongue in prolonged surgery. A tongue retraction suture, placed by the surgeon at the time of surgery and taped to the cheek, can be used to pull the tongue forward in the event of postoperative airway obstruction.[1]

Post-operative care should be managed in a high care unit, where the infant can be closely monitored. The most common postoperative problems include bleeding and airway obstruction. The nasal airway usually remains patent after cleft lip repair but nasal stents may be indicated. The nasal stents can be dislodged or become occluded with inspissated secretions. Palatal surgery is more likely to cause problems because the nasal and oropharynx are separated. A naso-pharyngeal tube may be indicated for a few days until the swelling subsides in those who are unable to breathe comfortably through their mouth.

Regional anaesthesia

The infra-orbital nerve supplies sensory innervation to the skin and mucous membrane of the upper lip and lower eyelid, the cheek between them and the alae nasi. An infra-orbital nerve block has proved useful in neonates,[4] infants[28-30] and older children[30] undergoing cleft lip repair in combination with general anaesthesia or even under sedation.[30] The block provides postoperative analgesia, without the risk of respiratory depression that may occur when opioid analgesics are used.[4] Bupivacaine with adrenaline is reported to provide a mean duration of 19 hours of analgesia, almost double that provided by lignocaine and adrenaline infiltration in a double-blind study.[28] Patients in this study were more comfortable postoperatively, with less sleep disturbance.

Two approaches to the infra-orbital nerve have been described in children: the intra-oral route.[4,28-30] and the extra-oral approach in neonates[4] and infants.[29] For the intra-oral approach a fine needle is inserted under and into the gingivo-labial fold at the level of the canine tooth (if present) on the ipsilateral side. The needle is directed upward and outward into the canine fossa. The infra-orbital foramen, palpable in older children, lies just below the orbital rim on a line drawn caudad through the centre of the pupil. (In adults the pupil, supra-orbital, infra-orbital, and mental foramina are in the same vertical plane. This does not apply in young infants). The infra-orbital nerve emerges from the foramen in a caudad and medial direction. When the tip of the needle is palpable in the area of the foramen, 0.5 to 1.5 ml of local anaesthetic can be deposited at the exit of the foramen.

The extra-oral approach is useful when the infra-orbital foramen is impalpable. The landmark used is based on a line drawn from the angle of the mouth to the middle of the palpebral fissure.[4] At the point where midpoint of this line (16-17mm from palpebral fissure) and a line equal to half this distance (8mm) drawn from the alae nasi intersect, a fine needle (25-26g) is introduced perpendicular to the skin. When bony contact is made, the needle should be withdrawn slightly and 0.5-1ml of local anaesthetic injected.[4]

The needle should not enter the foramen. Pressure generated by injecting local anaesthetic into the confined space of the narrow infra-orbital canal may result in nerve damage in children.[4,29,30] Another potential danger of entering the foramen is penetration of the flimsy orbital floor, with damage to the orbital contents.

The author believes that the intra-oral approach is potentially dangerous in neonates and small infants, because of the risk of orbital penetration or damage, in view of the close proximity of the infra-orbital foramen to the orbit – a matter of millimetres!

Postoperative Analgesia

Intravenous paracetamol, followed by regular doses of paracetamol usually provides adequate analgesia, particularly when combined with a nerve block[24, 25] An oral opiate (tilidine 1mg.kg or dihydrocodeine) may be necessary for some. Small doses of morphine 0.1mg.kg may be used in selected cases and in older children, where airway obstruction is less likely to be a problem. After palate repair, infants may be reluctant to swallow for approximately 24 hours. A nasogastric tube may be used for feeding, as well as drug administration. Alternatively, rectal analgesia and intravenous fluids may be required, until oral intake is re-established. Ketamine is popular in less developed

provided are rough, and can cause a sore throat post operatively. Small vaginal tampons (Lillets®) are a less abrasive alternative countries, where post-operative observation may be less than optimal.[26]

Conclusion

Anaesthesia for cleft lip and cleft palate surgery is very rewarding. Anaesthesia may range from extremely challenging, if faced with a neonate or infant who has a rare syndrome with a difficult airway and congenital heart disease, to fairly routine in a healthy infant with a simple defect of the upper lip. Both are equally rewarding when the parents see the dramatic improvements after surgery. Simple cosmetic surgery it is not - nor is the anaesthesia!

There are many controversial issues surrounding cleft lip and palate surgery. One issue questions whether cosmetic surgery should be done in the neonatal period. Some believe that only emergency surgery should be done because of the higher risks of anaesthesia in neonates. Ultimately the decision should depend on the skill and expertise of the team involved. The anaesthesiologist and surgeon play a vital role in that team.

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