Pathologic paediatric conditions associated with a compromised airway

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Purpose. The purpose was to describe pathologic paediatric conditions associated with airway compromise adversely affecting dental treatment with sedation and general anaesthesia.

Methods. A review of available literature was completed, identifying pathologic paediatric conditions predisposing to airway compromise.

Results. Airway-related deaths are uncommon, but respiratory complication represents the greatest cause of morbidity and mortality during the administration of general anaesthesia. Differences in anatomy and physiology of the paediatric and adult airway contribute to the child's predisposition to rapid development of airway compromise and respiratory failure; juvenile rheumatoid

Introduction

Literature reports that approximately onequarter of more than 1500 liability claims received by the American Society of Anaesthesiologists were related to adverse respiratory events during general anaesthesia. Although airway-related deaths are uncommon, respiratory complications in paediatric anaesthesia represent the greatest cause of morbidity and mortality during the administration of general anesthesia¹. Literature substantiates this claim, describing that careless patient evaluation prior to administration of general anaesthesia has resulted in failure to identify conditions which may contribute to difficult airway management². It therefore

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arthritis, cervical spine injury, morbid obesity, and prematurity represent only a few conditions contributing to potential airway compromise of which the paediatric clinician needs to be aware. In all cases, thorough physical examination prior to treatment is mandated to affect a positive treatment outcome.

Conclusions. Successful management of children and adolescents with a compromised airway begins with identification of the problem through a detailed medical history and physical examination. Due to the likely fragile nature of many of these patients, and possibility of concomitant medical conditions affecting airway management, dental treatment needs necessitating pharmacological management are best treated in a controlled setting such as the operating room, where a patent airway can be maintained.

becomes imperative that physical examination identify both pre-existing medical conditions contributing to airway compromise and a history positive for past difficulty in airway management. Subsequently, thorough and detailed history and physical examination are mandated to effect a positive sedation and general anaesthesia outcome. Neglect in ascertaining a detailed history can lead to adverse respiratory events during and after sedation and general anaesthesia, including atelactasis, bronchospasm, hypoxaemia, and respiratory failure³.

In order to understand the complications that may result during paediatric anaesthesia, it is important to understand the physiologic differences between the adult and paediatric airways. Anatomy of the paediatric airway predisposes the child patient to rapid development of a compromised airway and respiratory failure. As children have an inherently higher basal metabolic rate than their adult

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counterpart, for example, oxygen consumption in children is approximately twice that in adults. Resultant to children's increased demand for oxygen is a concomitant increase in alveolar ventilation, defined as the volume of gas per unit time reaching the alveoli. Similarly, the ratio of minute ventilation to functional residual capacity is increased in children, indicating that the child patient does not have sufficient oxygen reserve for longterm perfusion should hypoventilation occur. Consequently, should apnoea develop, loss of airway patency occur, or inadequate ventilation be performed, the child patient is predisposed to more rapid desaturation and development of hypoxaemia.

Anatomical differences between paediatric and adult airways also account for the risk of desaturation and complication during sedation and anaesthesia. Children's airways are smaller and funnel shaped, with a narrow and stiffer epiglottis. The more compliant airway of children, supported by less cartilage, makes the child more prone to airway obstruction by mucus, oedema, or aspiration of vomitus or other contents. Additionally, children's intraoral structures are large relative to their body size. Infants and children, for example, have a large tongue capable of complete airway obstruction if displaced too far posteriorly; grossly enlarged tonsils can result in the same outcome. Children may also have a higher epiglottis relative to adults, which when combined with larger tissue sizes, correlates to a greater tissue mass occupying a smaller space. This reality may make it more difficult to visualize anatomical landmarks during laryngoscopy and establish and maintain a patent airway during emergencies⁴.

The intent of this study is to describe pathologic paediatric conditions associated with a compromised airway which the paediatric provider may encounter in practice. As each of these conditions presents significant risk to outpatient parenteral and enteral moderate sedation, it is highly recommended that paediatric patients identified with the pathologic conditions reviewed below be treated under general anaesthesia. The risk of complications, including bronchospasm, hypoxa-

emia, apnoea, and respiratory failure, remain the same with administration of general anaesthesia. Patient safety is, however, not compromised due to the controlled operating room environment, where advanced equipment and skilled anaesthesiologists are readilv available in instances of emergency. Congenital anomalies may also contribute to a compromised airway during provision of treatment, although these conditions will not be discussed in this review. As described earlier, a thorough history and physical examination prior to treatment is essential to affect a positive treatment outcome, while avoiding adverse respiratory events before, during, or after the provision of treatment.

Supraglottic infections

Acute epiglottitis

Acute epiglottitis, an inflammation of the epiglottis, results from bacterial infection with Haemophilus influenzae B and most often presents in boys between three and 7 years old⁵. Typically characterized by distress, the clinical course is abrupt; within 6-24 h, the inflammation may progress to cardiorespiratory arrest and death. Patients may present with fever up to 40 °C, inspiratory stridor, muffled voice, and difficulty in swallowing, and individuals affected are usually postured upright or leaning forward. Typically, the oral cavity is associated with excessive salivation and drooling, and the epiglottis is oedematous in texture and cherry red in colour. Acute epiglottic inflammation may also be accompanied by pulmonary oedema, pericarditis, and septic arthritis, and if upper airway obstruction is not treated promptly and effectively, this condition can be fatal. As many as 6% of children identified with epiglottitis and without an artificial airway die. Yet, if an artificial airway is established, either by nasotracheal intubation or tracheostomy, the percentage of fatal outcomes drops to $<1\%^6$.

Croup (laryngotracheobronchitis)

Croup, resulting from parainfluenza virus infection, usually affects children between

6 months and 5 years and is characterized by a slow and gradual onset. The infection manifests clinically with mild fever, stridor on inspiration, and hoarseness of voice, although there is only minimal pharyngitis and the normal. Radiographic epiglottis appears evaluation often reveals subglottic narrowing, producing the characteristic inverted V-shaped appearance of the upper airway of Steeple's sign⁵. Yet radiographic appearance does not correlate well with disease severity, and Steeple's sign may be radiographically present in a patient without croup as a normal anatomic variant.

Antiviral agents have been proved to be beneficial against parainfluenza virus and many other viral aetiological agents of croup. Approximately 15% of patients experiencing croup have a strong family tendency of disease⁶.

Facial cellulitis

Facial cellulitis results from an unresolved abscess spreading through facial planes of soft tissue in the head and neck. Various patterns of cellulitis have been documented as sequelae to dental infection, but two forms, Ludwig's angina and cavernous sinus thrombosis, are especially threatening.

Ludwig's angina

Ludwig's angina defines an aggressive, rapidly progressing cellulitis involving the sublingual, submandibular, and submental spaces and was first described in 1836. Patients presented with a marked neck swelling, progressing to encompass facial spaces between the larynx and floor of the mouth, as Ludwig described diffuse, indurated oedema of the submandibular and sublingual spaces. The condition was almost universally fatal.

Although uncommon, this cellulitis most often affects children, young adults, and immunocompromised patients following dental infection. Extreme pain, swelling, and oedema occur in nearly all patients, and fever, fetid breath, and trismus can also occur; in its most severe form Ludwig's angina can result in swelling extending to the clavicles. More commonly, spread of infection into the sublingual space results in posterior elevation, enlargement, and protrusion of the tongue, causing dysphagia, dysphonia, and difficulty in respiration. Spread of infection into the submandibular facial plane often manifests with neck tenderness above the hyoid bone, whereas laryngeal oedema suggests involvement of the lateral pharyngeal space. More general signs and symptoms suggestive of spread of infection into any of these facial planes include tenderness in the floor of the mouth, restricted neck movement, dvsarthria, and sore throat⁷. Tachycardia and tachypnoea, stridor, and a need to remain upright in order to breathe, however, indicate airway obstruction secondary to oedema, swelling, and distortion of airway anatomy. Airway mismanagement of patients presenting with these latter symptoms of infection may end in death⁸.

Additionally, in rare clinical circumstances, bilateral sialadenitis or sialolithiasis may manifest as a Ludwig's angina infection. In order to avoid clinical mismanagement, such cases should be managed as a Ludwig's angina until a definitive diagnosis is made otherwise⁹.

Cavernous sinus thrombosis

Encased between the meningeal and periosteal lavers of the dura is the cavernous sinus, a susceptible site for spread of infection involving maxillary teeth. Cavernous sinus thrombosis may result when dental infection involving the maxillary anterior teeth perforates the maxillary buccal plate and extends into the canine space, eventually spreading into the cavernous sinus. Similarly, infection affecting maxillary premolars and molars may result in cavernous sinus thrombosis if an abscess is severe enough to perforate the buccal cortical plate and extend into the maxillary sinus, ptyergopalatine space, or infratemporal fossa. In such cases, dental infection reaches the cavernous sinus at the cranial vault.

Clinically, cavernous sinus thrombosis presents with a range of symptoms, although infection can usually be identified by oedematous swelling in the lateral nasal and periorbital areas. Yet, patients may also experience advanced toxaemia. In such cases, signs and symptoms of infection may range from tachycardia and tachypnoea to irregular breathing and meningitis. In the very worst cases, brain abscesses, and death if untreated, may result^{10,11}.

Benign tumours

Laryngeal papillomatosis

Laryngeal papillomatosis, the most common benign childhood laryngeal tumour diagnosed in approximately 1500 children per year¹², typically affects children prior to age 7 and is first identified by a change in voice character. The benign growths are likely a tissue response to human papilloma virus, and although malignant transformation is uncommon, degeneration can occur in older children. These papillomas usually spontaneously regress at puberty, although it is not impossible that they progress into adulthood¹³.

Some degree of airway obstruction affects approximately 40% of patients with this condition. Despite the tumour's benign character, the location of the growths and their tendency to recur make the condition troublesome for those patients undergoing sedation and general anaesthesia¹⁴.

Lymphangioma

A lymphangioma is a benign hamartomatous tumour of lymphatic tissues, most likely representing a developmental malformation arising from sequestrations of lymph tissue failing to communicate normally with the rest of the lymphatic system; it is unlikely that these growths are true neoplasms¹⁵. The most common lymphangioma subtypes include lymphangioma simplex and cavernous and cystic lymphangioma.

Approximately 50% of lymphangiomas are noted at birth, and 90% develop by 2 years of age. Most tumours are noted in the head and neck region, with up to 75% of all growths occurring there. Oral lymphangiomas, for example, are frequently noted developing on the anterior two-thirds of the tongue and may result in macroglossia; other oral lymphangiomas may cause swelling of the mouth and lips as well as involvement of the upper respiratory tract. Growths that compromise respiratory function can result in dysphagia with increasing size, and rapid tumour growth secondary to upper respiratory tract infection or blocked lymphatic drainage can further exacerbate already-compromised respiratory function¹⁶.

Oftentimes, secondary haemorrhage into lymphatic spaces may result in a purple appearance of the lymphatic tumour¹⁷.

Trauma

Cervical spine injury

Cervical spine injury, occurring in <2% of children with multi-system trauma, is associated with significant morbidity and mortality, occurs with an incidence of approximately 40 per 1 million cases, and is most often a result of blunt force cervical trauma¹⁸; Down's syndrome and hypoplasia or absence of the odontoid process are also common aetiologies for spinal instability subsequent to trauma. Bony traumatic injury usually occurs in children under 8 years old and affects cervical vertebrae C_1 through C_4 , although trauma affecting stability of the occiput, C_1 , or C_2 has the most serious implications. If chronic instability results as a consequence of trauma, progressive myelopathy may result; acute impingement and death are possible with severe instability^{19–21}.

Head or facial injury

Management of the paediatric patient with a history of maxillofacial trauma must consider anatomical and physiological differences between children and adults, concomitant injury, the child's stage of growth and development, and the patient's specific injuries and the areas those injuries affect²². The most serious complication following trauma is airway obstruction, although facial trauma in the absence of airway compromise rarely causes life-threatening conditions. Obstruction may be resultant secondary to laryngeal

fracture, bony disruption, and bony displacement, and airway compromise may be immediate or delayed. Aspiration of foreign objects or body fluids may result in sudden compromise whereas soft tissue damage or displacement, haemorrhage and haematoma, and oedema may cause delayed airway obstruction post-trauma.

Other serious sequelae to maxillofacial trauma include cerebral damage, midface deformity, and undiagnosed cervical spine injury; the serious implications of the last injury were discussed above^{23,24}. Knowledge of potential adverse outcomes of head and facial injury provides useful strategies for patient care and prevention of complications²⁵.

Rare conditions

Juvenile rheumatoid arthritis

An uncommon condition, with a reported incidence of approximately 13.9/100,000 children per year among Caucasians⁶, juvenile rheumatoid arthritis is important to the dental clinician because of its affect on both the cervical spine and temporomandibular joint. Injuries involving atlanto-axial subluxation and cervical spine fusion are more frequently described in the literature^{26,27}, but the temporomandibular joint is affected in as many as 40% of patients with RA. Typically, joint involvement is bilateral and occurs late in the disease with signs and symptoms including stiffness, crepitation, pain to palpation, and trismus; clenching of the teeth on one side results in pain on the contralateral joint. Limited range of mandibular motion often results because of synovitis on the joint.

Although ankylosis of the temporomandibular joint in rheumatoid arthritis is infrequent, this condition can impact the sedation provider, as destruction of the condylar head may result in micrognathia, a receding chin, and resultant malocclusion^{28–31}. In cases of ankylosis, radiographic presentation of the condyle is also altered: the condylar head is flattened, the temporal fossa irregularly shaped, and the condyle anteriorly displaced. Permanent joint subluxation is radiographically and clinically apparent in severe cases of ankylosis and has been reported in the litera-ture^{32,33}.

Cricoarytenoid arthritis is also common in patients with generalized rheumatoid arthritis and is most commonly associated with pain on swallowing, hoarseness of voice, dyspnoea, stridor, and tenderness of the lar-ynx^{9,34}.

Temporomandibular joint ankylosis

Ankylosis describes a fusion of body parts, is characterized as fibrous or bony, and can be classified as either intra- or extra-articular. Fibrous ankylosis typically results from infection and is more commonly seen in the temporomandibular joint, especially after traumainduced haemorrhage and haemarthrosis; conversely, osseous ankylosis results subsequent to nonhaemorrhagic infection. Intraarticular ankylosis of the temporomandibular joint is characterized by destruction of the meniscus and temporal fossa, thickening and flattening of the condular head, and a narrowing of the joint space, whereas extra-articular involvement produces an external fibrous or osseous encapsulation of the joint and is less common. Literature documents that post-traumatic infection accounts for approximately one-half of temporomandibular joint ankylosis, representing its most common cause, whereas 30% of all cases result from aseptic trauma³⁵. The remaining 20% of cases result from rheumatoid arthritis or are idiopathic³⁶.

Temporomandibular joint ankylosis primarily occurs in the first decade of life, affecting males and females equally. Although most cases are unilateral, joint ankylosis causes an inability to open the mouth and is characterized by an opening shift of the mandible toward the ankylosed side; the condition gradually worsens with time and may be characterized by pain, tenderness, and malocclusion. In severe cases, the mandible may be almost completely immobile, and as tissues occupy the joint space, the mandible may gradually protrude. In young children with hemifacial microsomia severe ankylosis, may become clinically apparent because of diminished mandibular growth on the affected side³⁵.

Goitre

A goitre is an enlargement of the thyroid gland and may be broadly categorized into four subtypes. A congenital goitre is typically sporadic in occurrence and may be a consequence of a synthesis defect of foetal thyroxine, T_4 ; fortunately, such a defect in hormone synthesis is extremely rare, with a reported incidence of 1/30.000-50.000 live births. Second, an endemic goitre (cretinism) is associated with an iodine deficiency in which a goitre results from compensatory hypertrophy or hyperplasia of thyroid tissue to overcome this deficiency. Third, a sporadic goitre results from defective synthesis of thyroid hormone and is often diagnosed by its early onset, occurrence in siblings, and characteristic hypothyroidism. Finally, an intratracheal goitre results from the ectopic location of thyroid tissue within the trachea. If tracheal obstructive manifestations are mild, administration of L-thyroxine may be used in management to decrease goitre size. In the most severe cases of obstruction, surgical intervention is mandated^{6,37,38}.

Carcinomas

Oropharyngeal carcinomas and neoplasms of the oral cavity, although rarely reported in the paediatric population, combined comprise approximately 30,000 new cases of cancer per year in the USA³⁹. The former are usually larger in size than oral neoplasms and commonly first present with pain and difficulty swallowing. Approximately 75% of lesions originate from the tonsilar and soft palate areas, and the remainder from the base of the tongue. At the time of diagnosis, 8% of oropharyngeal lesions have metastasized to surrounding tissues; the more posterior and inferior the tumour at time of diagnosis, the greater the chance of lymphatic spread.

Similarly, irradiation to the head and neck to treat childhood malignancy often induces changes in soft tissues of the larynx, affecting both size and mobility. For example, in supra107

glottic laryngeal carcinoma and pharyngeal abscess formation, the unobstructed airway in the conscious patient becomes difficult to secure in the sedated patient⁴⁰.

Miscellaneous

Morbid obesity

Morbidly obese patients, or those with a body mass index >40 for both height and weight, present with both compromised respiratory and cardiovascular systems, restrictive lung disease that is many times severe, and variations in airway physiology compared to thinner individuals. Commonly, accumulation of adipose tissue on the neck and thorax results in a decrease in chest wall compliance and concomitant increase in airway resistance. Likewise, loss of pharyngeal muscle tone adversely affects pharyngeal patency, leading to upper airway narrowing, turbulent airflow, and snoring $^{41-43}$. Upper airway obstruction in morbidly obese patients resultant to spreading facial and cervical infection can be potentially fatal due to the inability to secure an already compromised airway^{22,44}.

Obesity is of increasing concern to the dental provider, especially because of the increasing prevalence of this condition. The most recent NHANES survey, conducted between 1999 and 2002, identified 16% of children are obese and an additional 31% are at risk for becoming obese. These new statistics represent an approximate 300% increase in childhood obesity since the 1960s⁶.

Gastro-oesophageal reflux disease

Gastro-oesophageal reflux disease (GERD) is relatively common, affecting approximately 7% of the population daily and 36% of individuals on a monthly basis⁴⁵, and is defined as the regurgitation of acidic stomach contents into the oesophagus. This condition is characterized by posterior laryngitis, hypertrophic interarytenoid tissue, and oedematous vocal folds, as well as vocal fold granulation tissue; supraglottic oedema and subglottic inflammation may also be clinically detectable. It is also possible that already-existing chronic laryngeal oedema may be exacerbated by GERD⁴⁶.

Literature critique and analysis suggest that GERD has a causative role in subglottic stenosis, recurrent croup, chronic cough, and apnoea, and is also a significant cofactor in laryngomalacia, true vocal cord nodules, and recurrent choanal stenosis. Literature also supports that GERD is an important inflammatory cofactor in chronic sinusitis, otitis, and bronchitis and may result from chronic illness in older patients^{19,20,47}.

Finally, although supraglottic stenosis and collapse have not been identified to occur commonly in association with GERD, its occurrence has been documented in the literature. Such stenosis, if present, compromises a clinician's ability to secure and maintain a patent airway⁴⁸.

Supraglottic airway stenosis

Supraglottic airway stenosis refers to conditions that affect the diameter of the glottic lumen and should be among the differential diagnosis of patients experiencing chronic upper airway obstruction following tracheal or laryngeal surgery⁴⁸. However, conditions resulting in supraglottic stenosis may be both congenital and acquired. The former is associated with laryngomalacia and anterior placement of supraglottic and intra-arytenoid webs, and the latter with trauma.

Clinical findings in patients with supraglottic stenosis include thickened laryngeal soft tissues, a posteriorly displaced epiglottis, and anterior placement of otherwise posterior supraglottic structures. In some cases of stenosis, collapse of the epiglottis may be observed resultant to anterior pressure or a lack of anterior support entirely. The latter can occur due to relaxation of either the hypoepiglottic ligament or suspensory muscles of the hyoid bone⁴⁹.

Finally, acquired airway stenosis represents the most serious long-term complication of endotracheal intubation in children. In such cases, trauma from mechanical abrasion and pressure necrosis of laryngeal tissues cause ulcerations that, when healed, result in granulation and scar tissue formation. Although most granulation tissue resolves without adverse sequelae, some does become fibrous scar tissue, contracting and decreasing the size of the glottic lumen, resulting in an irregularly shaped glottic opening. Similarly, if the duct of a mucous gland is obstructed by scar tissue formation following traumatic intubation, a cyst may develop resultant to mucous accumulation in the duct. If significant enough in size, the laryngeal lumen size and shape may be compromised, resulting in airway compromise⁵⁰.

Premature birth

Prematurity is medically defined as liveborn infants who are delivered prior to 37 weeks gestation, and low birthweight, defined as a mass <2500 g, is often a consequence of premature birth⁶. Resultant to medical advances, however, there has been continued and gradual improvement in the survival rates of premature infants weighing <1500 g at birth, or so-called very low birthweight infants. Because of increases in survival rates of premature infants, though, the prevalence of bronchopulmonary dysplasia and its milder form, chronic lung disease of prematurity, have increased.

In children affected by bronchopulmonary dysplasia, initial air trapping usually improves within the first 4 years of life, but small airway obstruction which is slow to improve is suggestive of dysanaptic lung growth; other severe complications include asthma-like symptoms, airway hyper-reactivity, increased airway resistance, and apnoea. These varied pulmonary complications of bronchopulmonary dysplasia remain long-term abnormalities^{51–55}.

Conclusion

Treatment options exist for completing needed dental care in those patients presenting with the conditions described above, including in-office treatment with moderate sedation medications as well as treatment under general anaesthesia. Yet due to the likely fragile condition of many of these patients, and possibility of concomitant medical conditions affecting airway management, it is advisable that extensive dental needs necessitating pharmacological intervention be completed in a controlled environment. In these instances, patient safety is assured, loss of the airway can be avoided, and rescue efforts can be initiated, if indicated. In-office moderate sedation may be contraindicated, and only small, short procedures should be completed by individuals who are adequately trained; advanced airway training is imperative for the patient who presents with an already compromised airway, as in those cases described above. Should an emergency fail to be identified, changes in airway function may result in hypoventilation, desaturation, and in the worst cases, death. In these instances, a regulated clinical environment is required, and it becomes imperative that dental treatment is rendered in a controlled clinical setting such as the operating room to assure and maintain patient safety, avoiding emergencies.

In each of the cases described, however, the most successful patient management begins with identification of the problem and a thorough understanding of the patient's medical history. All patients with potentially compromised airways should be evaluated for mandibular opening and visibility of oropharyngeal landmarks upon opening, as well as for the size of the mandibular space. Additionally, known anatomical factors contributing to difficult airway management must be accounted for in patient care. Nonetheless, when a team approach and effective communication are employed in patient care, complications before, during, and after treatment with sedation and general anaesthesia can be avoided.

What this paper adds

- Review of paediatric versus adult airway physiology.
- Review of pathologic conditions which the paediatric provider may encounter in practice.

Why this paper is important to paediatric dentists

- Importance in ascertaining a thorough medical history prior to treatment.
- Patient safety in provision of dental treatment.
- Ability to differentiate between normal and abnormal airway conditions and make recommendations for treatment in dental office versus operating room.

References

- 1 Caplan RA, Posner KL, Ward RJ, Cheney FW. Adverse respiratory events in anesthesia: a closed claims analysis. *Anesthesiology* 1990; **72**: 828–833.
- 2 Ovassapian A, Glassenberg R, Randel GI, Klock A, Mesnick PS, Klafta JM. The unexpected difficult airway and lingual tonsil hyperplasia: a case series and a review of the literature. *Anesthesiology* 2002; **97**: 124–132.
- 3 Cote CJ, Notterman DA, Karl HW, Weinberg JA, McCloskey C. Adverse sedation events in pediatrics: a critical incident analysis of contributing factors. *Pediatrics* 2000; **105**: 805–814.
- 4 Saxen MA, Wilson S, Paravecchio R. Anesthesia for pediatric dentistry. *Dent Clin North Am* 1999; **43**: 231–245. vi.
- 5 Diaz JH. Croup and epiglottitis in children: the anesthesiologist as diagnostician. *Anesth Analg* 1985; **64**: 621–633.
- 6 Kliegman R, Nelson WE. *Nelson Textbook of Pediatrics,* 18th edn. Philadelphia, PA: Saunders, 2007.
- 7 Hartmann RW Jr. Ludwig's angina in children. *Am Fam Physician* 1999; **60**: 109–112.
- 8 Britt JC, Josephson GD, Gross CW. Ludwig's angina in the pediatric population: report of a case and review of the literature. *Int J Pediatr Otorhinolaryngol* 2000; **52**: 79–87.
- 9 Honrado CP, Lam SM, Karen M. Bilateral submandibular gland infection presenting as Ludwig's angina: first report of a case. *Ear Nose Throat J* 2001; **80**: 217–218. 222–223.
- 10 Chen HW, Su CP, Su DH, Chen YC. Septic cavernous sinus thrombosis: an unusual and fatal disease. *J Formos Med Assoc* 2006; **105**: 203–209.
- 11 Dolan RW, Chowdhury K. Diagnosis and treatment of intracranial complications of paranasal sinus infections. *J Oral Maxillofac Surg* 1995; **53**: 1080– 1087.
- 12 Dancey DR, Chamberlain DW, Krajden M, Palefsky J, Alberti PW, Downey GP. Successful treatment of juvenile laryngeal papillomatosis-related multicystic lung disease with cidofovir: case report and review of the literature. *Chest* 2000; **118**: 1210–1214.
- 13 Berry FA. Juvenile papillomatosis and airway obstruction. *Anesth Analg* 1997; **84**: 1395–1396.
- 14 Mikkelsen PG. Laryngeal papillomatosis with airway obstruction in an infant. *Acta Anaesthesiol Scand* 2001; **45**: 645–648.
- 15 Brennan TD, Miller AS, Chen SY. Lymphangiomas of the oral cavity: a clinicopathologic, immunohistochemical, and electron-microscopic study. *J Oral Maxillofac Surg* 1997; **55**: 932–935.
- 16 Thompson BM, Welna JO, Kasperbauer JL, Van Ess JM, Marienau ME. Childhood airway manifestations of lymphangioma: a case report. *AANA J* 2004; **72**: 280–283.
- 17 Bryan Y, Chwals W, Ovassapian A. Sedation and fiberoptic intubation of a neonate with a cystic hygroma. *Acta Anaesthesiol Scand* 2005; **49**: 122–123.

- 18 Hanson JA, Blackmore CC, Mann FA, Wilson AJ. Cervical spine injury: a clinical decision rule to identify high-risk patients for helical CT screening. *AJR Am J Roentgenol* 2000; **174**: 713–717.
- 19 Doolan LA, O'Brien JF. Safe intubation in cervical spine injury. *Anaesth Intensive Care* 1985; **13**: 319–324.
- 20 Hensinger RN. Congenital anomalies of the cervical spine. *Clin Orthop Relat Res* 1991; **00**: 16–38.
- 21 Wong JK, Tongier WK, Armbruster SC, White PF. Use of the intubating laryngeal mask airway to facilitate awake orotracheal intubation in patients with cervical spine disorders. *J Clin Anesth* 1999; **11**: 346–348.
- 22 Combes X, Jabre P, Jbeili C *et al.* Prehospital standardization of medical airway management: incidence and risk factors of difficult airway. *Acad Emerg Med* 2006; **13**: 828–834.
- 23 Ardekian L, Rosen D, Klein Y, Peled M, Michaelson M, Laufer D. Life-threatening complications and irreversible damage following maxillofacial trauma. *Injury* 1998; **29**: 253–256.
- 24 Verschueren DS, Bell RB, Bagheri SC, Dierks EJ, Potter BE. Management of laryngo-tracheal injuries associated with craniomaxillofacial trauma. *J Oral Maxillofac Surg* 2006; **64**: 203–214.
- 25 Alvi A, Doherty T, Lewen G. Facial fractures and concomitant injuries in trauma patients. *Laryngoscope* 2003; **113**: 102–106.
- 26 Espada G, Babini JC, Maldonado-Cocco JA, Garcia-Morteo O. Radiologic review: the cervical spine in juvenile rheumatoid arthritis. *Semin Arthritis Rheum* 1988; **17**: 185–195.
- 27 Nathan FF, Bickel WH. Spontaneous axial subluxation in a child as the first sign of juvenile rheumatoid arthritis. *J Bone Joint Surg Am* 1968; **50**: 1675–1678.
- 28 Ronchezel MV, Hilario MO, Goldenberg J *et al.* Temporomandibular joint and mandibular growth alterations in patients with juvenile rheumatoid arthritis. *J Rheumatol* 1995; **22**: 1956–1961.
- 29 Stabrun AE. Impaired mandibular growth and micrognathic development in children with juvenile rheumatoid arthritis. A longitudinal study of lateral cephalographs. *Eur J Orthod* 1991; **13**: 423–434.
- 30 Svensson B, Larsson A, Adell R. The mandibular condyle in juvenile chronic arthritis patients with mandibular hypoplasia: a clinical and histological study. *Int J Oral Maxillofac Surg* 2001; **30**: 300–305.
- 31 Twilt M, Mobers SM, Arends LR, ten Cate R, van Suijlekom-Smit L. Temporomandibular involvement in juvenile idiopathic arthritis. *J Rheumatol* 2004; **31**: 1418–1422.
- 32 Hakala P, Randell T. Intubation difficulties in patients with rheumatoid arthritis. A retrospective analysis. *Acta Anaesthesiol Scand* 1998; **42**: 195–198.
- 33 Larheim TA, Dale K, Tveito L. Radiographic abnormalities of the temporomandibular joint in children with juvenile rheumatoid arthritis. *Acta Radiol Diagn (Stockh)* 1981; **22**: 277–284.

- 34 Ovassapian A, Tuncbilek M, Weitzel EK, Joshi CW. Airway management in adult patients with deep neck infections: a case series and review of the literature. *Anesth Analg* 2005; **100**: 585–589.
- 35 Chidzonga MM. Temporomandibular joint ankylosis: review of thirty-two cases. *Br J Oral Maxillofac Surg* 1999; **37**: 123–126.
- 36 Gogalniceanu D, Trandafir V, Chiriac R, Gogalniceanu P. Temporomandibular joint ankylosis. A possible complication in juvenile psoriatic rheumatism. *Rev Med Chir Soc Med Nat Iasi* 2005; **109**: 652–659.
- 37 Jaruratanasirikul S, Leethanaporn K, Suchat K. The natural clinical course of children with an initial diagnosis of simple goiter: a 5-year longitudinal follow-up. *J Pediatr Endocrinol Metab* 2000; **13**: 1109–1113.
- 38 Voyagis GS, Kyriakos KP. The effect of goiter on endotracheal intubation. *Anesth Analg* 1997; 84: 611–612.
- 39 Smith BD, Smith GL, Carter D, Sasaki CT, Haffty BG. Prognostic significance of vascular endothelial growth factor protein levels in oral and oropharyngeal squamous cell carcinoma. *J Clin Oncol* 2000; **18**: 2046–2052.
- 40 Delbridge L, Sutherland J, Somerville H, Steinbeck K, Stevens G. Thyroid surgery and anaesthesia following head and neck irradiation for childhood malignancy. *Aust N Z J Surg* 2000; **70**: 490–492.
- 41 Baker S, Yagiela JA. Obesity: a complicating factor for sedation in children. *Pediatr Dent* 2006; 28: 487– 493.
- 42 Strollo PJ Jr, Rogers RM. Obstructive sleep apnea. *N Engl J Med* 1996; **334**: 99–104.
- 43 Zerah F, Harf A, Perlemuter L, Lorino H, Lorino AM, Atlan G. Effects of obesity on respiratory resistance. *Chest* 1993; **103**: 1470–1476.
- 44 Puchner W, Obwegeser J, Puhringer FK. Use of remifentanil for awake fiberoptic intubation in a morbidly obese patient with severe inflammation of the neck. *Acta Anaesthesiol Scand* 2002; **46**: 473–476.
- 45 Chiba N, De Gara CJ, Wilkinson JM, Hunt RH. Speed of healing and symptom relief in grade II to IV gastroesophageal reflux disease: a meta-analysis. *Gastroenterology* 1997; **112**: 1798–1810.
- 46 Burton DM, Pransky SM, Katz RM, Kearns DB, Seid AB. Pediatric airway manifestations of gastroesophageal reflux. *Ann Otol Rhinol Laryngol* 1992; **101**: 742–749.
- 47 Yellon RF, Goldberg H. Update on gastroesophageal reflux disease in pediatric airway disorders. *Am J Med* 2001; 111(Suppl 8A): 78S–84S.
- 48 Walner DL, Holinger LD. Supraglottic stenosis in infants and children. A preliminary report. *Arch Otolaryngol Head Neck Surg* 1997; **123**: 337–341.
- 49 Belmont JR, Grundfast K. Congenital laryngeal stridor (laryngomalacia): etiologic factors and associated disorders. Ann Otol Rhinol Laryngol 1984; 93: 430–437.

- 50 Liu H, Chen JC, Holinger LD, Gonzalez-Crussi F. Histopathologic fundamentals of acquired laryngeal stenosis. *Pediatr Pathol Lab Med* 1995; 15: 655–677.
- 51 Coates AL, Bergsteinsson H, Desmond K, Outerbridge EW, Beaudry PH. Long-term pulmonary sequelae of premature birth with and without idiopathic respiratory distress syndrome. *J Pediatr* 1977; **90**: 611–616.
- 52 Galdes-Sebaldt M, Sheller JR, Grogaard J, Stahlman M. Prematurity is associated with abnormal airway function in childhood. *Pediatr Pulmonol* 1989; **7**: 259–264.
- 53 Halvorsen T, Skadberg BT, Eide GE, Roksund O, Aksnes L, Oymar K. Characteristics of asthma and airway hyper-responsiveness after premature birth. *Pediatr Allergy Immunol* 2005; **16**: 487–494.
- 54 Kennedy JD, Edward LJ, Bates DJ *et al.* Effects of birthweight and oxygen supplementation on lung function in late childhood in children of very low birth weight. *Pediatr Pulmonol* 2000; **30**: 32–40.
- 55 Walther-Larsen S, Rasmussen LS. The former preterm infant and risk of post-operative apnoea: recommendations for management. *Acta Anaesthesiol Scand* 2006; **50**: 888–893.

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