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Scale to evaluate the airway in pediatrics "EVAP"

Summary

The most feared catastrophe in Anesthetic practice is the management of the unanticipated Difficult Airway (VAD) in the Pediatric age where the Anesthesiologist does not have sufficient skills and abilities to face a clinical scenario of Emergency Surgery and due to the absence of a Training in the Sub-Specialty of Pediatrics and the Airway, hence the importance that during the training phase as resident physicians of the Specialty, training in the management of AV in pediatric patients and during professional life as an Anesthesiologist General is trained in basic and advanced courses on Pediatric Airway Management to develop technical skills and abilities in stressful and complex situations in the VA to provide efficient, effective and safe patient-centered medical care, due to these scenarios, this Pediatric Airway Clinical Scale is proposed based on Anatomical and Functional variables (EVAP), as well as the classification of Congenital Diseases and Clinical Entities that alter the Anatomy of the Upper Airway to identify an anticipated VAD, to be able to stratify the risk and plan an Efficient Medical Care of the Airway in Emergency and Elective Surgeries in the Pediatric age.

Keywords: Airway (VA), Difficult Airway (VAD), Pediatric Airway Scale (EVAP)

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Introduction

Difficult ventilation with a face mask (MF) during a routine anesthetic induction in a child under 8 years of age is an imminent challenge if VAD was not previously assessed or anticipated, so it is standard to continue ventilating with MF (if necessary) bi-manual) continue to administer 100% oxygen in addition to head and shoulder positioning and request help to continue with the Pediatric Airway (VAP) algorithm in children 1 to 8 years of age.[1]

The ASA defines a difficult airway as the existence of clinical factors that complicate ventilation administered by a face mask or intubation performed by an experienced person. Difficult ventilation is defined as the inability of trained personnel to maintain oxygen saturation above 90% using a face mask, with a fraction of inspired oxygen of 100%. Difficult intubation is defined as the need for three or more attempts to intubate the trachea or more than 10 minutes to achieve it. It is transcendental to remember that the greater the degree of difficulty of the airway, the greater the incidence and severity of complications will be presented.[2] In this paper, the variables and clinical characteristics that affect the functionality of the upper and lower airway in the infant are exposed according to the presence of genetic and congenital diseases and anatomical alterations. Table 1 describes the clinical situations that predispose a VAD.

Table 1

Difficult Airway	Difficulty in performing Face Mask Ventilation or Orotracheal Intubation by an expert person
	Inability to maintain S02 >92% with =2 100% and Ventilation with positive ventilation
	Major gas leak from face mask
Difficult Ventilation	Need to increase gas flow to > 15 L/min and use of oxygen flush more than twice
	no perceptible chest movements
	Need to ventilate with a two-handed mask (Han's classification)
	Need to change operator
Difficult laryngoscopy	Presence of Comarck-Lehane grade III or IV
Difficult Intubation	Requires more than 2 attempts
Hard DSG Insertion	Requires more than two attempts or evidence of trauma during insertion

Complications related to the management of VAP are frequent and sometimes generate morbidity and mortality, mainly affecting newborns and young infants. Within morbidity, inadequate ventilation, apnea, and bronchial obstruction may occur. Pediatric patients have a wide spectrum of diseases, both congenital and acquired, that can affect the airway, making ventilation or intubation difficult. Therefore, it is essential to understand the anatomical differences of the VAP, as well as to become familiar with the diseases and syndromes of the pediatric patient. Currently, there are no studies of the VA carried out in the ER, especially those that analyze predictive factors of difficulty. Nor is there a scale specifically for the pediatric age, since most are classified for the adult patient; however, the scales are useful in the daily work of every physician who manages AS, since it is better to anticipate and/or anticipate a VAD rather than one that occurs unexpectedly or unanticipated. Due to this, in this present writing, a Clinical VAP Scale is proposed based on the anatomical changes of the face, neck and oropharyngeal structures according to pediatric age, the main congenital craniofacial syndromes and sino-bronchial diseases. It has already been mentioned that difficult intubation, inadequate ventilation, apnea, and bronchial obstruction are the direct causes of morbidity. Most of these complications are avoidable and when audited during the medical-legal lawsuits, the following problems of the type of physical infrastructure, medical equipment and management of the complicated environment have been found; It has been the failure to recognize or anticipate problems due to inadequate planning of the AS, inadequate revision of the machine and monitors (omission and imprudence in medical practice), scale of surveillance and insufficient preparation in the face of adverse situations (imprudence).

The anatomical differences of the VA are an angular pillar for the proper and correct management of the VAP. Pediatric patients present a wide spectrum of diseases, both congenital and acquired, that can affect the airway, making intubation and/or ventilation difficult. The differences between the VA of a child and an adult are of great importance for anesthetic management. Newborns and infants are the group with the greatest anatomical differences in the airway.

The differences are in the nostrils, which are small and constitute the main ventilation route. During the first 6 months of life, the tongue is relatively large, the tonsils are prominent, the larynx is small and is positioned more cephalad, leaving the opening glottic at the level of C3-C4 (it reaches C5 at 6 years). The epiglottis of the child is shorter omega-shaped and softer than that of the adult, it forms an angle of about 45 degrees with the base of the tongue, compared to 15-25 degrees in the Adult, its arytenoids are large compared to the glottic opening and there is a progressive reduction in size from the hyoid to the cricoid and the shape of the larynx is conical. The narrowest part of the larynx is at the level of the cricoid cartilage. These characteristics can make it difficult to pass the orotracheal tube. It is important to remember that infants have a larger head and occipital promontory, and a relatively short neck. This produces cervical flexion, which prevents head extension or sniffing position. To correct this flexion angle, a cylindrical bulge of about 10 cm is placed on the edge of the shoulders to help extend the head. and achieve the sniffing position during ventilation and intubation.

Another differential point between both age groups are the physiological variables such as higher oxygen consumption and less oxygen reserve (CRF) in infants and young children. As well as gastric distension, frequent when they are ventilated with a face mask, elevates the diaphragm, further decreasing the functional residual capacity (FRC) and the oxygen reserve. It also decreases lung compliance, which interferes with positive-pressure ventilation, increasing the risk of regurgitation and aspiration. Children are more susceptible to upper airway obstruction due to the greater sensitivity of certain inspiratory muscles to anesthetic agents. VA instrumentation during OI can trigger bronchospasm; Children are more susceptible to airway obstruction because the subglottic epithelium is of a pseudostratified columnar type that responds to instrumental manipulation, inflammation, and a sudden increase in airflow resistance with a valve effect due to negative airway pressure. and progress to a barotrauma. The Presence of bradycardia due to the predominance of the parasympathetic nervous system. Remember that bradycardia is the main response to hypoxemia and heart rate is a determinant of cardiac output and that young infants are dependent on heart rate to maintain adequate cardiac output, so it is important to pay attention to the presence of bradycardia and correct the root cause of its appearance to maintain good hemodynamics in the infant.

In the airway, the infant is more predisposed to lung collapse due to the immaturity of the laryngotracheobronchial structures. Regarding the tracheobronchial tree, the difference in caliber with respect to the adult conditions a greater resistance to the passage of air, favoring its turbulence even during calm breathing due to the greater laxity and vascularization of the mucosa, where a minimum degree of edema, Accumulation of secretions or bronchospasm can exponentially increase resistance to airflow and can quickly lead to airway obstruction. The caliber in the Adult is 8 mm and 4 mm in the child in normal conditions in the presence of edema the caliber in the Adult is 7 mm and in the child 3 mm, which reflects a decrease in the VA lumen in 44% in the Adult and 75% in the infant.

The thoracic wall is also more flexible, with the ribs horizontal, so that breathing and the maintenance of Lung Capacity depend to a great extent on the respiratory muscles. The infant breathes with a lower tidal volume and higher respiratory rate. The lower functional residual capacity entails a lower reserve in apnea and a tendency to hypoventilation.

The diaphragmatic and intercostal muscles of the neonate and infant are poor in type I myofibrils, which allow repetitive movements, so any condition that leads to increased work of breathing can lead to muscle fatigue and respiratory failure. There is also less maturity of the respiratory center, with less response to hypercapnia and pH variations. In short, the effectiveness of ventilation is lower in children, especially in small infants and neonates, when compared to adults. For this reason, the infant rapidly progresses to hypoxemia.[3-5]

The neonate has an increase in cellular oxygen consumption due to its metabolic activity between 2 to 3 times and in the event of a cessation of oxygen supply, it will be quickly depleted. It also has greater closing capacity. The volume of air that remains in the alveoli to prevent their collapse is called the volume or closing capacity. In adults, this volume is proportionally less and coincides with a value that is within the CRF. In neonates and infants, this volume is greater, close to the tidal volume, that is, with a minimal reduction in tidal volume, the small airways close and atelectasis forms, aggravating oxygenation and ventilation problems.[2-7]

A good evaluation through a detailed medical history and physical examination of the head, neck, and cervical spine are important in recognizing Pediatric VAD. Records of previous Anesthesia focused on airway management should be included, especially recording whether ventilation with the face mask was possible or not. The presence of congenital, traumatic or inflammatory diseases, which may make airway management difficult. Knowledge of the syndromes that affect the airway is vital for its management. Micrognathia creates more difficulty in moving the tongue during direct laryngoscopy, preventing observation of the glottis. This situation improves as the child grows (Pierre Robin Syndrome), while anomalies due to Mucopolysaccharidosis such as Klippel-Feil syndrome (Mucopolysaccharidosis type I) orotracheal intubation becomes more difficult as the child grows older because of cervical stiffness.

The history and physical examination should be aimed at looking for a history or the presence of snoring, stridor, apnea, drowsiness, phonation and voice quality, bronchospasm, infectious and inflammatory diseases. In children, those inflammatory or infectious variables of the airway should be considered, such as rhinitis, bronchitis, bronchiolitis, pneumonia, pharyngitis, laryngotracheitis, bronchial hyperreactivity and obstructive sleep apnea, which can lead the patient to severe obstruction due to laryngospasm or bronchospam in seconds. and difficulty in managing the VAD.[2]

Persistent stridor warrants the indication of the VA study. It is defined as a respiratory sound of variable pitch produced by the passage of a turbulent air flow in areas of AV with a decreased caliber. It can be at rest and appreciated with crying or exercise. It can be acute or chronic, congenital or acquired. Depending on the phase of the respiratory cycle in which it is audible, it can point to the level of obstruction: if it is inspiratory, it points to a supraglottic problem, while expiratory is characteristic of the intrathoracic airway. If the stridor is biphasic (inspiratory-expiratory) we must consider glottic, subglottic or extrathoracic tracheal pathology.[5] Another is lingual thyroid localization which is a result of failure of the thyroid gland to descend from the base of the tongue through the thyroglossal duct. Also, the cyst of the thyroglossal duct can be filled with mucus or liquid and if it is located at the base of the tongue it can cause obstruction and stridor.

The most frequent cause of stridor in the pediatric age is laryngomalacia, since it is the most frequent anomaly of the Upper Airway and is of the inspiratory type. The exact causes are not known, but it begins in the first weeks of life and it is believed that it may be due to a delay in the neuromuscular maturation of the supporting structures of the larynx that causes collapse of the supraglottic

structures on inspiration. Most children present mild symptoms with a benign and self-limiting course with resolution in the first year of life (it is good to take this into account in elective surgery in the first year of life). There is a 5 to 10% with serious behavior, conditioning difficulty for feeding with the presence of cyanosis, apnea and can even lead to the development of pulmonary hypertension and cor pulmonale. In these patients, a complete study of the VA is recommended since the coexistence of abnormalities in the lower VA has been described in 17 to 68%.

Biphasic stridor that begins with a history of OTI should be considered subglottic stenosis. Those of the congenital type are usually symmetrical, while those acquired are asymmetrical. The clinic will vary depending on the degree of stenosis. Treatment can be conservative in mild forms, in moderate or severe cases endoscopic or surgical treatment may be required.[5] It is important to point out the different causes of stridor according to the respiratory phase in which it is audible and its anatomical origin, as referred to in the following Table 2.[8]

Table 2

Inspiratory or Extra thoracic	Biphasic	Expiratory or Intrathoracic
Supraglottic: Laryngomalacia	Glottic: paralysis of vocal cords	intrathoracic tracheomalacia
Subglottic: extra thoracic tracheomalacia	Subglottic: fixed subglottic stenosis	bronchomalacia
	Fixed injuries at any level	bronchogenic cysts
		Extrinsic compression (mediastinal masses, vascular rings, lobular emphysema, lymphadenopathy)
		tracheal stenosis

Another cause of acquired subglottic stenosis is gastroesophageal reflux "GERD", which is the rise of gastric contents into the hypopharynx and larynx that can lead to inflammation and edema that further compromise airway patency. It is reported that there is also an association with laryngomalacia because the increased intrathoracic pressure of laryngomalacia can promote gastric reflux in the esophagus and larynx. Reflux can promote laryngopharyngeal edema that causes decreased sensitivity of mucosal receptors and promote collapse and dysfunction of swallowing and microaspiration in children.

Undiagnosed Chronic GERD causes constant damage due to repetitive reflux to the striated epithelium of the respiratory mucosa, making it more reactive to prolonged periods of chronic inflammation with decreased airway lumen at the subglottic level.

Sometimes asthma refractory to treatment can be due to the presence of GERD, however, reflux can be both a cause and a consequence of the respiratory pathology, so ideally, pHmetry can demonstrate a temporal relationship between reflux episodes. and the appearance of wheezing. In cases where this is not possible, it may be useful to detect a pattern of GERD consisting of prolonged episodes during the night (nocturnal cough). Another hidden respiratory symptom of GERD is chronic cough, recurrent pneumonia, pulmonary aspirations, it is essential to look for the presence of long reflux during sleep. It can also be the diverse cause of rhinopharyngeal symptoms such as laryngitis, rhinitis, refractory sinusitis, otitis media, dysphonia.[9] The constant reflux causes the growth of Waldeyer's ring lymphoid tissue (adenoids, lingual tonsil, palatine tonsils and posterior pharyngeal lymphatic tissue) causing obstruction of the oropharyngeal lumen, this condition may lead to the indication of Corrective Adeno-tonsillectomy (Tonsillectomy) and be exposed to a VAD during IOT due to unanticipated subglottic stenosis due to untreated Chronic GERD.

24-hour esophageal pH monitoring has been the most reliable GERD diagnostic method for many years. Recently, the multichannel intraluminal impedance study (IIM) allows the evaluation of esophageal transit and the identification of functional esophageal abnormalities in esophageal motor disorders. It has been shown that GERD induces respiratory symptoms in 95% due to changes in chronic inflammation of the larynx.[10] Vocal cord paralysis is the second most important cause of chronic childhood stridor. It can be congenital or acquired. Congenital Paralysis manifests within the first month of life. Bilateral paralysis produces stridor, cyanosis, and apnea. Unilateral Paralysis presents dysphonia. Causes of unilateral and bilateral paralysis include neurologic disease (18%), myelomeningocele with Arnold-Chiari malformation and hydrocephalus (14%), birth trauma (19%), other (115), malignant and/or familial disease (1 %). In 36% of the congenital disease the cause is not identified, but there may be an association with other congenital diseases at the pulmonary, cardiovascular or esophageal level. Acquired vocal cord palsy can be due to surgical trauma such as repair of a tracheoesophageal fistula or heart defects. When the diagnosis of vocal paralysis is confirmed, cardiological and neurological evaluation is recommended. Tracheostomy is indicated in bilateral paralysis.[8]

It is important to keep in mind the large number of developmental disorders that affect the oral and maxillofacial region. The vast majority of these alterations have been classified as Syndromes of a genetic order, however, not all of them can be described as such, since there are developmental anomalies that appear as a consequence of deficient embryogenesis of the facial region, causing anatomical and functional alterations, far from the specific genetic and chromosomal components.[11] So it is a point to consider in a good physical examination to identify Craniofacial Dimorphisms that alter the permeability and security of the Normal and Difficult Airway in the Pediatric age.

During the physical examination, a search should be made for malformations of the head, neck, and cervical spine. The shape and size of the head (Macrocephaly, Microcephaly), anomalies present in the face such as craniofacial dimorphisms where there are changes in the size and symmetry of its components such as the distance between the eye sockets (hypertelorism with a wide and flattened nasal bridge); Nose (patency of nostrils), mandible and its mobility (micrognathia, its approach to make the airway permeable is with retromolar intubation most of the time), dental prominence, size (macroglossia such as Down syndrome, Beckwith- Wiedemann syndrome), shape of the tongue and palate (Cleft Palate), lips (Cleft lip), implantation of ears (Turner Syndrome that is associated with a thick neck and large cervical circumference), the shape of the ears (microtia). All of these changes are clinical predictors of VAD. In one study it was shown that bilateral microtia is associated in 42% with VAD and 2% with unilateral microtia. The presence of mandibular hypoplasia accompanied by bilateral microtia is associated with VAD in 50% since these variables affect mouth opening, face mask ventilation, direct laryngoscopy, application of supraglottic rescue devices and visualization of the glottis.

Anatomical anomalies at the level of the head, facial, temporomandibular joint, mouth and tongue, nasal, palatal, pharyngeal, laryngeal, trachea, bronchi, neck and cervical spine.

At Head Level, tumor lesions may occur and large head growths may interfere with face mask ventilation and/or laryngoscopy and intubation. Among the tumor anomalies, encephalocele, Klippel-Feil syndrome (cervical vertebrae synostosis), hydrocephalus (to make the airway permeabilized by direct laryngoscopy, the head must be positioned in a lateral position), cleft palate, soft tissue sarcoma, macro and microcephaly, Hurler syndrome (it is the most serious of the mucopolysaccharidoses, with delayed development and physical growth, large tongue, flat face and nasal bridge, prominent forehead).

At the facial level, Apert Syndrome, Crouzon Disease, Pierre-Robin, Treacher Collins Syndrome, Freeman Sheldon Syndrome, Pfeiffer Syndrome are described. In these patients, both the upper and lower VA may be affected; some have choanal atresia, narrowing of the nasopharyngeal space, cleft and deformed palates. These structures cause ventilatory compromise and obstructive apnea that increase as the child grows (except in the Pierre-Robin syndrome sequence, which improves with age) (See Table 3).

Table 3

Craniofacial Syndrome	Genetic trait	Anatomical Features	Evaluation Clinic	AS approach
Kippel-Feil	autosomal dominant and recessive	Fusion 1-2 Vertebrae 4 types of vertebral synostosis, face asymmetry, short neck, low hairline implantation, syringomelia, situs in versus	Limitation of neck movement in flexion-extension	Ventilate with Fastrach Ventilation-intubation Mask, DSG with port for aspiration and intubation: Air q, Supreme, Fibrobronchoscopy, rescue boguie
hurler	MPSI a-1-idoronidose mucopolysacchardosis, Autosomal dominant	Stunting, short stature, short stem, short neck, flat ribs, broad forehead, flat nose, macroglossia, thick lips, square jaw, narrow windpipe	Chronic sinusitis with retronasal discharge, sinobronchial reactivity, degree of difficulty for direct laryngoscopy	Ventilate with face mask with good seal, DSG with suction port and intubation type Air q, Supreme, laryngeal tube, VDL optical blade type with stylet, Air Traq
open	FGFR2 gene mutation. Autosomal Dominant	Craniosynostosis, hypertelorism, exophthalmos, brachycephaly, prominent forehead, low pinna, short neck, syndactyly hands and feet	Prematurity, difficulty for ventilation with MF and sealing, anterior glottis, Comarck-Lehane III and IV	Ventilate with Face mask with good sealing, DSG with suction port and intubation: Air Q, Supreme, Proseal, Igel, VDL optical blade with stylet, rescue bougie, Air traq
Crouzon 's disease	Autosomal dominant, affected FGFR2 gene	Coronosagittal suture fusion, fusion cervical vertebrae C2-C3 and C5-C6, hydrocephalus, hypertelorism, mandibular hypoplasia, flattened forehead, exophthalmos, mandibular prognathism, U-shaped maxilla, short lip and parrot-beaked nose	Difficulty for ventilation with MF and sealing, limitation for cervical flexo-extension, difficulty in positioning the head during laryngoscopy	Pad on shoulders and chest for alignment of oropharyngeal axes, Ventilate with Facial mask with good sealing or Flexible LMA for, DSG for ventilation and intubation type: Frastrach, Air Q, Supreme, VDL with optical blade, direct laryngoscopy in lateral position (hydrocephalus)
Peter-Robin	Malformation sequence in the development of the face and palate	U-shaped cleft palate, severe mandibular hypoplasia, glossoptosis, micrognathia	Difficulty breathing and feeding, risk of Aspiration Pneumonia, difficulty in Ventilation with MF, direct laryngoscopy grade III to IV.	Prior gastric emptying, Ventilate with MF with good seal or GFD with gastric port and intubation: Laryngeal tube, Air Q, Supreme, Proseal, VLG optical blade with stylet, rescue bougie
Teacher -Collins	TCOF1 gene mutation, autosomal dominant	Mircognatia, retrognatia, macrostomy or facial fissure type/of Tessuer is unilateral left and bilateral 10 to 20 %, microtia, palpebral cleft, malar hypoplasia, maxillary convexity	Difficulty ventilating with MF, difficulty for direct laryngoscopy very anterior glottis, Comarck-Lehane IV	Ventilate with Facial mask with good sealing or with DSG with gastric port and Air Q, Supreme type intubation. Igel, rescue bougie, Fibrobronchoscopy
Freeman-Sheldon	Autosomal dominant, homozygous mutations in MYH3 gene, distal Arthrogryposis type A2	Strabismus, hypertelorism, low-set ears, hearing loss, prominent nasolabial folds, whistling face microstomia, high arched palate, small mouth with dental crowding, H-shaped chin with a hole, kyphosis, scoliosis	Difficulty ventilating with MF, poor mouth opening, difficulty for direct laryngoscopy and GFD insertion	Ventilate with MF with good seal, fiberoptic bronchoscopy, rescue bougie

Table Continued...

Craniofacial Syndrome	Genetic trait	Anatomical Features	Evaluation Clinic	AS approach
P.Feiffel	Autosomal Dominant, affected gene FGFR1 and 2	Creaneosynetosis, syndactyly hands and feet, occasional hydrocephalus, cloverleaf brachycephaly, midface hypoplasia, severe proptosis, fat thumbs	Difficulty ventilating with MF, head positioning for oropharyngeal axis alignment during direct laryngoscopy	Place a pad on the shoulders and thorax to position the head and align the oropharyngeal axes, Ventilate with MF or DSG with gastric port and Air Q type intubation, Supreme, direct laryngoscopy in lateral position, rescue bougie
goldenhar	autosomal recessive	Microphthalmia, iris atrophy, coloboma, Behavioral deafness, microtia, mandibular hypoplasia, cervical vertebral anomalies, macrostomy, ojival or cleft palate, dermoid epibular single	Difficulty ventilating with MF due to lack of good sealing, direct laryngoscopy without degree of difficulty	Ventilate with Facial mask with good sealing or with DSG with laryngeal tube type, flexible Classic LMA, Proseal, fastrach for ventilation and intubation, VDL with optical blade with stylet, Air traq with stylet, Rescue bougie
Turner	Partial or complete absence of the X chromosome	Hypothyroidism, immune type, hypogonadism, short stature, low set ears and hair, short, wide neck with skin folds, overweight, aortic valve disease, osteoporosis	Without Difficulty to Ventilate with Face Mask, Conventional Direct Laryngoscopy without degree of difficulty	Ventilate with Face Mask, DSG and rescue Bougie to ventilate and intubate in case of extreme overweight. VDL optical paddle with stylet, perioperative CPAP
markio	Mucopolysaccharidosis type IV A and B, there are two types of deficiency of galactosamine-6,5-Sulfatase and N-galactosidase	Bone deformity, hydrocephalus, kyphosis, vertebral hypoplasia	Difficulty positioning the head and aligning oropharyngeal axes due to hydrocephalus	Place a pad on the shoulders and thorax to align the oropharyngeal axes, direct lateral or medial laryngoscopy, use of DSG to ventilate the laryngeal tube, Igel for Short Surgery. Use of DSG to ventilate and intubate with rescue bouguie
Arnold Chiari	Congenital cerebrospinal malformation	Hydrocephalus, syringomelia, spina bifida, headache, decreased hearing and vision	Difficulty ventilating with a face mask in a neutral medial position, difficulty in direct laryngoscopy in a neutral or medial position	Place a pad on the shoulders and chest to align the oropharyngeal axes for ventilation. Direct laryngoscopy in lateral position. Use of DSG to ventilate Classic LMA type, Igel, Laryngeal tube. Use of Rescue Bouguie

At the Temporomandibular Joint Level, a decrease in mobility or ankylosis may occur, whether congenital, traumatic, inflammatory or infectious. Presence of mandibular hyperplasia-hypoplasia such as micrognathia such as Goldenhar Syndrome (it has auditory and ear abnormalities, mandibular hypoplasia and vertebral anomaly), Pierre-Robin, Turner, Treacher Collins, Morquio (It is a mucopolysaccharidosis type IV-A, lysosomal type disease presenting with bone deformity, hydrocephalus, flexed neck, kyphosis, lumbar hump, pectus carinatum due to vertebral and diaphyseal hypoplasia of the femoral head, ulna, radius as well as joint laxity).[4]

A At the level of mouth and tongue microstomies are presented (small mouth opening as in the flat or flattened face syndrome which presents this variable in addition to large ears and low implantation, hypernasal voice); Burns, tongue hemangiomas, tumors, macroglossia as in Down and Hurler Syndrome, presence of Ludwing's angina, edema secondary to surgery, macroglossitis by manipulation and lingual hematoma. At the level of the nasal, palatal and pharyngeal cavities, there may be choanal atresia, presence of foreign bodies, encephalocele, cleft palate, palatal edema, presence of bruises, tonsillar hypertrophy and adenoids between 4 and 6 years of age, the greatest growth is observed in these (causes obstruction), tumors, retropharyngeal abscess, inflammatory processes (Epidermolysis Bullosa that presents around the mouth a fragile skin with blisters in response to a minor injury, heat, friction, light friction, use of tape for fixation).

laryngomalacia may exist, it is the lack of incomplete development of the rigid structures of the larynx, as a result, there is a weakness and enlargement of the soft structures, which causes a collapse of the soft structures during respiration, causing laryngeal stridor (which improves with time, it is more audible during the sleep phase, feeding and increases in infectious processes), it is related to GERD, so it is important to give conservative treatment for gastroesophageal reflux; Correction of the condition through a surgical procedure involves viewing the larynx and cutting excess soft tissue such as the Ari-epiglottic folds to make more space for air to pass through the trachea. Other anomalies are epiglottitis (alarm signs are continuous drooling, tripod position, and cyanosis); vocal cord paralysis, laryngeal flanges, papillomatosis, foreign body, subglottic stenosis, infections such as laryngeal Croup, traumatic stenosis and edema.

Congenital tracheomalacia can occur at the level of the trachea and bronchi, it is rare and is due to the fact that the cartilage of the trachea has not developed properly. The walls of the trachea are flaccid instead of rigid, with narrowing of the tracheal rings during inhalation causing a turbulent airflow passage characterized by shrill, noisy and vibratory breathing, exacerbated by feeding and inflammatory-infectious processes, is associated with GERD. Other conditions at this level are infectious, tracheitis, mediastinal mass, vascular malformations, foreign body aspiration, edema, acquired tracheal stenosis, fistulas, diverticula, and flanges.

At the Neck and Cervical Spine Level there may be tumors such as giant lymphangiomas, hemangiomas, teratomas, scarring, inflammation, rigid column such as Klippel-Feil Syndrome, Previous surgery, vertebral fracture.[3] The following Table 3 describes the pathologies of genetic and/or congenital affection that alter the anatomical and clinical morphology and the functionality of the AV, it describes the approach strategies for rescue and anticipated planning of the GOES.

Each of these genetic and congenital diseases at some point in the infant's development are subjected to partial or definitive orofacial surgical corrections, as well as craniofacial advances where it is essential to ensure the definitive airway because they are long-lasting corrective surgeries. Changes in the Emergency scenarios where the infant presents some type of Craniofacial Dimorphism should be planned based on what is obtained in the hospital microenvironment, the operator's skills in techniques and devices, and the current VAD algorithms to obtain the Plan A, B, C for securing the airway.

Despite the above, it is important to remember that there is no anatomical factor that can individually predict the possibility of encountering difficulties for tracheal intubation. The predictive value of tests or pre-intubation tests in children is low compared to adult patients.[4] Until now, there is no Unique and Specific Scale to Evaluate Pediatric VA that contains all the variables in context. In the following Table III that brings together a series of useful variables to stratify the Pediatric VA and a better planning of the normal VA and Advance VAD with the purpose of identifying the children with the highest risk and the safest strategies for their management of the VA.

The VAD has a different incidence according to the training of the personnel evaluated, it can be as low among anesthesiologists considered experts in 0.05% or as high in prehospital care in 11%, between these limits the incidence of Pediatric VAD is located in general practitioners, pediatrics and intensivists. The younger the child, the greater the chance of having a VAD. Failure in the management of pediatric VAD requires immediate attention, since the hypoxemia phase occurs in less time, responding to this state with bradycardia and cardiac arrest, brain injury or death.

During the preparation of the VA, time should be taken to evaluate and classify the VA, considering the setting in which it will be addressed, defining the physical state of the child, its functional class, the surgical risk and the urgency of the procedure.[2]

There are few studies of the VA performed in the emergency room, especially those that analyze predictive factors of difficulty. Most of them have been done in anesthesiology rooms, others in intensive care units and some of them in simulation scenarios. The most affected population in terms of morbidity and mortality due to inadequate management of the airway is the newborn and young infants (less than 1 year of age). Within morbidity, inadequate ventilation, barely, and bronchial obstruction can occur. Most of these complications are avoidable and when analyzed, the following problems are detected, such as the failure to recognize and anticipate the VAD, inadequate review of the equipment and monitoring, poor surveillance, insufficient preparation in the face of detected adverse situations and lack of skills. technique, especially in stressful situations.[6]

Complications occur in 43% in children and 30% in adults. Art 8. Therefore, preoperatively evaluating the different causes that can lead to difficult intubation is of vital importance to prevent complications.[7] The following Pediatric Airway Evaluation Scale was based on the most frequent anatomical variables, clinical and congenital entities that alter the morphology and function of the Airway in the Pediatric Age, it represents a practical context to evaluate, stratify and evaluate each scenario clinic of normal and difficult airway. The following proposal is described below, which is the objective of this present Article, to share the "Scale to Evaluate the Pediatric Airway" (EVAP) as shown in Table 4. For the score of this "EVAP" scale, each variable is equivalent to one point and they are stratified into three degrees of complexity according to the points obtained by summation (Table 5). It is important to have the habit of observing and measuring the anatomical structures (mouth, neck, nose, lips) of the upper airway as well as the axes and lines of the craniofacial points (mandible, auricular implant, ocular symmetry, bridge nasal, palate shape, tragus, malar).

Table 4

Variable	Grade I	Grade II	Grade III
Age	< 1 year	neonate	Premature
Weight	Overweight	Obese	Under weight
Head circumference	35cm (+/-1)	> 38cm	<34cm
*** Nasion -chin axis**	53-58mm	>59mm	<52mm
***Chin-tragus axis**	**70** m (+/- 2 to 3)	>74mm	<67mm
Jaw	Normal	Macrognathia, Prognathism	Mandibular hypoplasia, micrognathia, retrognathia
Palate	arched palate	ogival palate	Cleft Palatine and/or Cleft
Tongue/microstomia/ macrostomia	Normoglossia with unilateral microstomia and macrostomia	Macroglossia with unilateral macrostomia	Macroglossia with bilateral macrostomia
Mouth opening	4cm	<4cm	<2cm
Craniofacial Syndrome	down	Pierre-Robin	Treacher -Collins
Respiratory grunt	mild	moderate	severe
crying/anxiety	spontaneous/reactive crying	Reactive crying with moderate agitation	Sustained, uncontrollable crying, excessive agitation

Table Continued..

Variable	Grade I	Grade II	Grade III
Characteristics of Nasal Mucus	thick/opaque	Yellow green	Hyaline, watery, stringy, sticky
Respiratory Tract Infection	Chronicle	subacute	acute
Phonation	infantile or acute	Hoarseness/dysphonia	extremely sharp
Gastroesophageal Reflux Disease "GERD"	mild	Moderate	Severe

Table5

Complexity Degree	Bass	Moderate	Tall
Points	1-3	4-6	>6

The training of the VA in crisis situations is an important part for a correct treatment of the VAD in the Pediatric age. Clinical Scenarios through simulation workshops make a difference in planning and approaching proper management of AV in emergency situations. Assuming that daily practice provides sufficient expertise and training for crisis scenarios is a mistake. Therefore, it is important to prepare with simulation workshops in critical scenarios with high-fidelity mannequins of anatomical and physiological variables of the respiratory tract. Simulation workshops are important together with the surgical work team, using their own resources to become familiar with its operation, developing local management algorithms or hospital microenvironment to face the emerging state of the airway.[10-16]

Conclusion

It is essential to remember that children under 1 year of age are more difficult to intubate and older children with the appropriate maneuvers are easier to manage in their ventilation and oxygenation with a face mask with an adequate one-hand or two-hand seal (Han scale) according to the situation of the Airway. The use of manual CPAP to help stabilize the airway is good practice.[17] Keep in context that in Premature and Neonate children we will face Difficult Ventilation due to the correct sealing of the Face Mask, avoiding leaks during ventilation as well as good anticipated pre-oxygenation leaks. In a percentage of > 50% we will be faced with an extreme anterior glottis opening in grade III and IV of the Comarck-Lehane Scale. These Pediatric Patients, even developing to Adult life, their Glottis will be located anterior during laryngoscopy. Patients with untreated mild and/or moderate Chronic GERD will have a reduction in the caliber of the Airway lumen due to subglottic stenosis, requiring smaller caliber orotracheal tubes corresponding to the corresponding Pediatric Age, even this reduction in the caliber of the lumen extends up to Adulthood; Therefore, it is important to visualize the air column of the airway by radio imaging in the pre-anesthetic evaluation in all patients with the presence of Chronic GERD.

The classification of the VA in the pediatric patient is easy to stratify if the history of VAD, congenital, anatomical and physiological alterations are kept in mind. The purpose of this new Pediatric evaluation scale present in this Article is to provide a more contextualized and practical panorama through an evaluation score that establishes the degree of risk and stratification to anticipate the adequate management of the Pediatric VAD.

Acknowledgments

None.

Conflicts of interest

None.

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