ANALYSIS OF ARTERIAL BLOOD GAS IN STRIDOR PATIENTS AND THE IMPACT OF EMERGENCY TRACHEOSTOMY

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CERTIFICATE

This is to certify that the dissertation titled **"ANALYSIS OF ARTERIAL BLOOD GAS IN STRIDOR PATIENTS AND THE IMPACT OF EMERGENCY TRACHEOSTOMY"** is a bonafide work done by **Dr.FAYAZ AHMED. S.F**, in partial fulfillment of the requirements for MS (ENT) Branch IV Examination of The Tamilnadu Dr.M.G.R.Medical University to be held in September 2006. The period of study was from January 2004 to March 2006.

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CONTENTS

INTRODUCTION

Establishing and maintaining patent upper airway is the first and most vital step in the basic life support and maintenance of artificial airway is the most fundamental aspect of such support.

The term tracheotomy is derived from the Greek word TOME (to cut) and implies the performance of a non permanent type of surgery. The term tracheotomy was actually coined by Lorenz Heister in 1718. The term tracheostomy is derived from the word STOMOUN (to furnish with an opening) and implies a more permanent opening into the trachea. The international organization for standards has named the act of cutting a hole a tracheotomy. The actual hole and the tube are both called tracheostomy.

Respiration is the utilization of oxygen by the body in the production of energy. Much of the metabolism occurs by aerobic means i.e., it requires the presence of oxygen. The respiratory tract has evolved into a complex series of tubes whose primary function is to allow the exchange of gases across all aerobic cells. The carriage of oxygen and carbon-dioxide to and from tissues, and the exchange of these gases with air, is vital for life.

Life is an acidogenic process and from birth to death, the body is under a constant obligation to balance hydrogen ions (H^+) output against hydrogen ion intake and production.

There are two classes of acids that are physiologically important; carbonic acid and non-carbonic acid. Each day the metabolism of carbohydrates and fats results in the generation of approximately 15,000 mmol of carbon dioxide (CO_2). Although CO_2 is not an acid, it combines with water to form carbonic acid. Lungs remove the CO_2 and therefore prevents accumulation of carbonic acid.

Non-carbonic acids (e.g. Sulphuric acid) are primarily derived from protein metabolism. Only 50-100 mEq/day of acid is produced from those sources and excreted in urine.

The concentration of hydrogen ions is in nanomole range unlike other ions like potassium, sodium, chloride, bicarbonate which are all in millimole range. Even so, the small size of hydrogen ions permits high reactivity with binding sites on proteins, with the result that small changes in hydrogen ion concentration can produce significant alternations in enzyme activity and thus organ dysfunction.

In patients with upper air obstruction, the probability of poor oxygenation, tissue perfusion and waste elimination is very high. Tracheostomy is a life saving surgical procedure performed to provide long term airway access. The objective of tracheostomy is to maintain oxygenation and tissue perfusion.

In arterial blood gas studies (ABGs) this manifests as respiratory and/or metabolic acid base disorder. Ideally these patients should be under continuous, real time blood gas monitoring for immediate intervention. But with the existing facilities the acid base status is assessed only indirectly on a continous basis. The indicators of acid base status are SPO_2, B.P, E.C.G, Urine output etc. These indicators can't be relied always as they are influenced by many other factors. As a compromise Arterial Blood Gas Analysis is done. This study is about the analysis of arterial blood gas changes in stridor patients undergoing emergency tracheostomy in Upgraded Institute of Otorhinolaryngology, Govt. General Hospital, Madras Medical College, Chennai – 600 003.

AIM OF THE STUDY

To study the impact of emergency tracheostomy on Acid-Base and ventilatory status in patients with upper airway obstruction based on the following parameters.

1. Detection of Acid - Base disorders using Arterial Blood Gas (ABG) in patients with upper airway obstruction.

2. Quantification and classification of commonly occurring Acid - Base disorder.

3. Relevance to morbidity and mortality.

4. Impact of tracheostomy on improvement in Acid Base and ventilatory status of patient.

5. Time duration required for improvement in Acid Base status, post tracheostomy.

REVIEW OF LITERATURE

(Historical Review) [1,24,49]

The rich history of tracheostomy stretches back over, the procedure initially was discussed almost simultaneously by both Galen and Aretaeus in the 2nd century AD but neither admitted to performing the operation.

Asclepiades, of Persia in the 2nd century BC (124 BC) is credited with performing first tracheotomy, at that time the only known indication for such surgery was for 'Synanche" or "Cynanche" which referred to nonspecific inflammatory conditions about the larynx, the floor of the mouth, and the head.

In 2nd Century AD, Tracheotomy technique was further defined by Antyllas,(625 –690 AD) a greek surgeon who advised that the 'arteria aspera' (trachea) should be divided at the IIIrd and IVth ring. Obscure reference to tracheotomy in the Ebers Papyrus and Rig veda in 1000-2000 BC.

In 4th century, The Greek ruler Alexander the great is rumored to have performed a tracheotomy himself. He allegedly used the tip of his sword to open the trachea of a chocking soldier.

In the 7th Century, Paul of Aegina, recorded that the physician could be aware that the airway had been entered because a rush of air would be heard and loss of the patient's voice would be noted.

In the 16th Century, the Italian physician Antonio Mura Brasovala, (1490-1554) operated on a near terminal patient with an abscess of the wind pipe in 1546 BC.

Sanctorias (1561-1636) is believed to be the first to use a trocar in the operation and he recommended leaving the cannula in place for a few days following the operation.

Marco Aurelio Serverino (1580-1656) used the tracheotomy to save multiple lives during the 1610 diphtheria epidemic in Naples: he also developed his own version of a trocar.

Hieromymus fabricious ab. Aquapendente (1537-1619) suggests use of a cannula for tracheostomy in 1600.

Nicholar Habicot (1620) French Surgeon Described Four Successful Tracheotomies, One Of These, Performed a 14yr Old Boy, Was Possibly The First Successful Paediatric Tracheostomy And First Recorded A Case Of A Tracheotomy For The Removal Of A Foreign Body By Using Curved Metal Tube – (The Boy Had Attempted To Swallow A Bag Of Gold Coins To Prevent Their Possible Theft, But The Bag Had Become Lodged In The Esophagus And Obstructed The Trachea).

George Martine (1702-1743) the earliest known British tracheotomist suggests use of an inner cannula in the tracheostomy tube in 1730.

Jean Charles Felix Caron (1745-1824) successfully performed the procedure on a 7yr old boy to remove a bean.

Andree, in 1782, recorded performed tracheotomies on paediatric patients.

Tucker & Silverman (1972) found an increase in tracheotomies in the later half of their study and fifty percent of their patients were between the ages of one and five.

George Washington (1799) at Mount Vernon, Virginia, died of an upper air obstruction probably due to acute epiglottitis or an Inflammatory Quinsy.

Goodall reported 28 tracheotomies performed prior to 1825.

Pierre Bretonneau, (1778-1862) published report of a successful tracheotomy in a 5 yr old girl with diphtheria in 1825.

Trousseau (1833) reported 50 of 200 children with diphtheria by performing tracheotomies on them and also stressed techniques for post operative care for the first time.

Chevalier Jackson (1909)[47] described the standard surgical tracheostomy. (in which dissection of the strap muscles was followed by controlled entry into the trachea).

The resistances of tracheostomy tubes can be calculated by rohrer (1915)[52] he accounted for both laminar and turbulency airways with the equation for resistance : $r=k_1v + k_2v_2$. *K1* is a constant reflecting laminar flow, and v represents the flow rate.

Chevalier Jackson (1932) standardizes the technique of tracheotomy and warns against 'High tracheotomy".

Galloway (1943) reported the usefulness of the procedure for respiratory care of patients with poliomyelitis.

Sheldon C.H. (1955)[51] first attempted percutaneous tracheostomy with a Slotted needle and a cutting trocar used to create an opening into the trachea.

Bjork (1960) introduced the concept of suturing an inferiorly based flap consisting of the anterior portion of a single tracheal ring to the inferior skin margin.

Berden (1965) introduced polyvinyl chloride tracheostomy tube.

Mc Donald and stocks (1965) described the use of intubations and respiratory support for neonatal patients.

Toye and Weinstein (1969) developed percutaneous tracheostomy, influenced by the Seldinger technique in method of using a single tapered dilator with a recessed cutting blade.

Ciaglia. P (1985) originated the technique known as percutaneous dilational tracheostomy and described the percutaneous dilational tracheostomy method via the sheldinger approach in 1989.

Schachner. A (1989) developed the rapitrac method of forcibly advancing beveled metal conus over a wire.

Griggs W.M (1990) presented guide wire dilating forceps method.

Donald C. Lanza et al (1990) they conducted a study on Predictive value of the Glasgow coma scale for tracheostomy in Head injured patients.

Friedman M, et al. (1990)[44] introduced "Fingertip" technique to identified the tracheal window in standard tracheostomy method.

Dov Ophir, et al (1990)[53] he used a cricothyroid cannula from a soft, uncuffed, Portex pediatric endotracheal tube of 4 or 4.5 mm diameter as minicricothyrotomy for tracheobronchial toilet.

Marilene B. Wang, et al (1992) they used a needle for entry in to the trachea, a J-tipped guide wire is passed through the needle and progressively larger dilators are used to widen the stoma for insertion of the tracheostomy tube.

Mullis, et al (1993) he measured the Resistances of neonatal, pediatric, and adult tracheostomy tubes using Rotameter and pneumotachometer.

Medhat – Hannallah. M.D. (1995)[54] found the jet stylet to be a useful aid for tracheostomy tube replacement if difficulty is anticipated.

Robert F. Gray et al (1998) followed four steps; inspect the airway for patent, repair obstructive sites, down sizes and cap the tracheostomy tube for a functional trial, and perform decannulation with observation.

Jessica W. Lim, et al (2000) they compared the result of PDT and standard tracheostomy, when performed by the same surgeon.

Rovo, Laszlo M.D. (2000)[55] performed minimally invasive management of Bilateral recurrent nerve injury after Thyroid surgery. This management offers an alternative to tracheostomy in the early period of paralysis, avoids terminal loss of voice quality, by using mono filament non absorbable thread

passed above and under the posterior third of the vocal cord and knotted on the prelaryngeal muscles, permitting the creation of an abducted vocal cord position with the help of endoscopes.

Eliachar, Isaac. M.D, et al (2000)[56] conducted a prospective study of tube free Tracheostomy intended to establish un aided cough and speech by using a new surgical technique using a local tendinous muscular sling was designed to further improve the efficacy of stomal constriction.

Elbert Cheng,et al (2000) he compared the complications of standard tracheostomy and percutaneous dilatational tracheostomy by Meta –analysis.

Kost, Karen. M. et al, (2005) they done a study on Endoscopic Percutaneous Dilatational Tracheostomy of 500 consecutive cases in 2005,October, from1990 to 2003 from that 191 patients underwent PDT using the Ciaglia Percutaneous Tracheostomy introducer Kit and in the remaining 309 patients the Ciaglia Blue Rhino single dilator kit was used.

SURGICAL ANATOMY

SURGICAL ANATOMY OF TRACHEOBRONCHIAL TREE[35]

INTRODUCTION

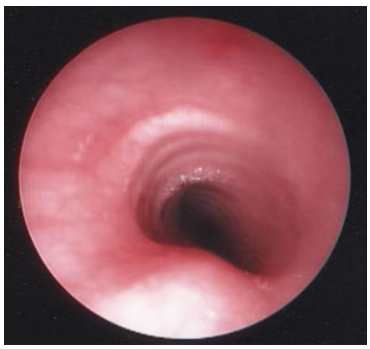
The trachea is a cylindrical tube which extends inferiorly and somewhat posteriorly from the larynx into the thorax, where it bifurcates into the two main stem bronchi. The trachea consists of a series of C- shaped cartilages connected by connective tissue. The structure is supported on its anterior and lateral walls, while the posterior wall is membranous, overlying the esophagus closely. The length of the adult trachea is about 12cm and its cross-sectional area about 2.5cm.Men tends to have a larger trachea than woman. The trachea enters the thorax at approximately the level of the sixth tracheal cartilage. This is somewhat variable depending upon the overall length of the neck (i.e. in persons with a short neck the second or third cartilage may lie at the level of sternum) and body posture and function (during high-pitched phonation and swallowing the upper trachea tends to be elevated in the neck).

The trachea is lined with pseudostratified ciliated epithelium containing large numbers of mucous glands and goblet cells. The glands are found dorsal to the layer of smooth muscle on the posterior wall and in the intercartilaginious spaces. Smooth muscle extends transversely between the free posterior ends of the C-shaped cartilages.

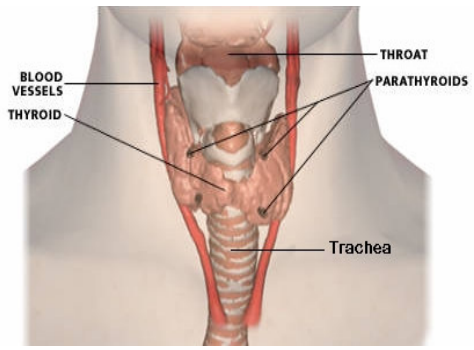
The blood supply to the trachea in the neck is from branches of the inferior thyroid arteries, and its venous drainage ends in the thyroid venous plexus. Its innervation is from vagus and its recurrent branches and the sympathetic system.

The trachea is formed of cartilage and fibromuscular membrane, lined internally by mucosa. It is about 10–11 cm long, descends from the larynx, extending from the level of the sixth cervical to the upper border of the fifth thoracic vertebra, where it divides into right and left principal (pulmonary) bronchi. It lies approximately in the sagittal plane but its point of bifurcation is usually a little to the right. The trachea is mobile and can rapidly alter in length; during deep inspiration the bifurcation may descend to the sixth thoracic vertebral level. Its cylindrical shape is flattened posteriorly so that in transverse section it is shaped, with some individual variation, likes a letter D. Its external transverse diameter is about 2 cm in adult males, and 1.5 cm in adult females. In children it is smaller, more deeply placed and more mobile. The lumen in live adults is about 12 mm in transverse diameter, although this increases after death due to relaxation in the smooth muscle at its posterior aspect. In the first postnatal year, the tracheal diameter does not exceed 3 mm while during later childhood its diameter in millimeters is about equal to age in years. The transverse shape of the lumen is variable, especially in later decades, being round, lunate or flattened.

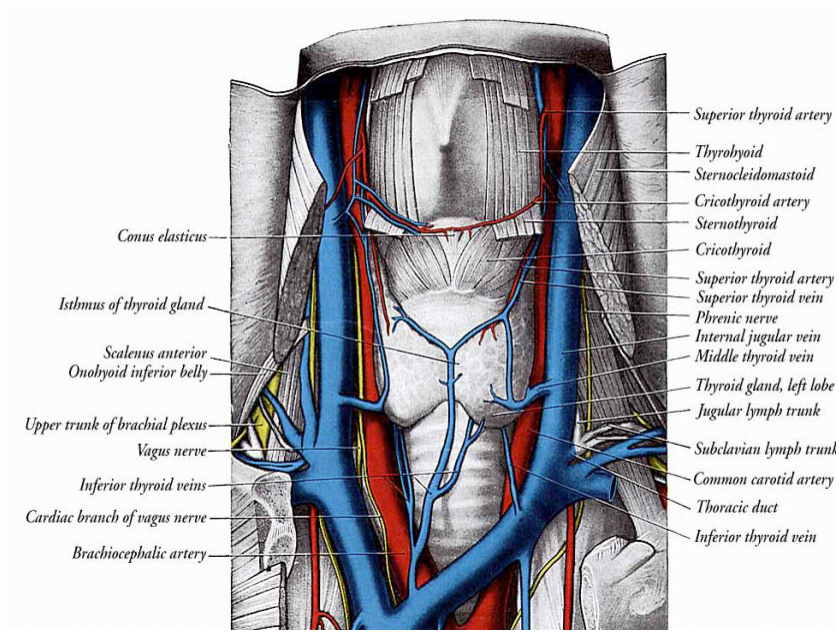
Endoscopic view of normal trachea

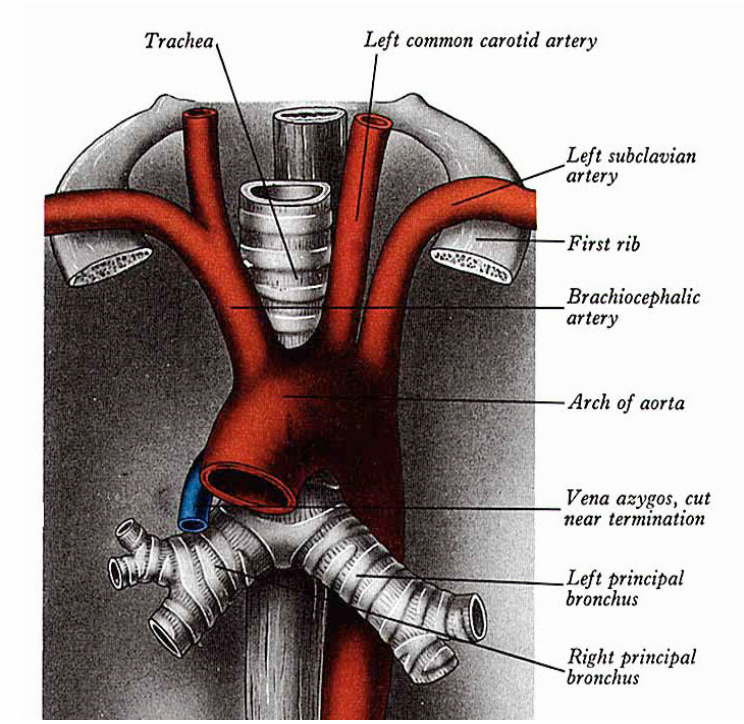


Normal Anatomy of the Trachea

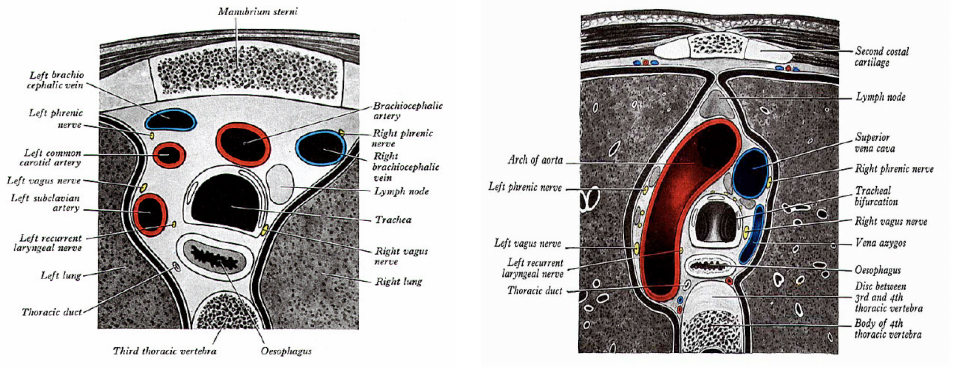


Structure Related to Trachea

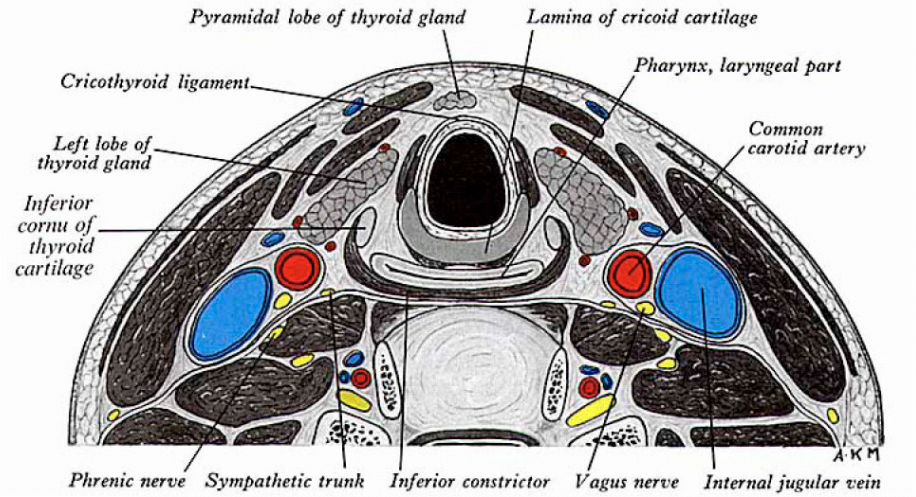


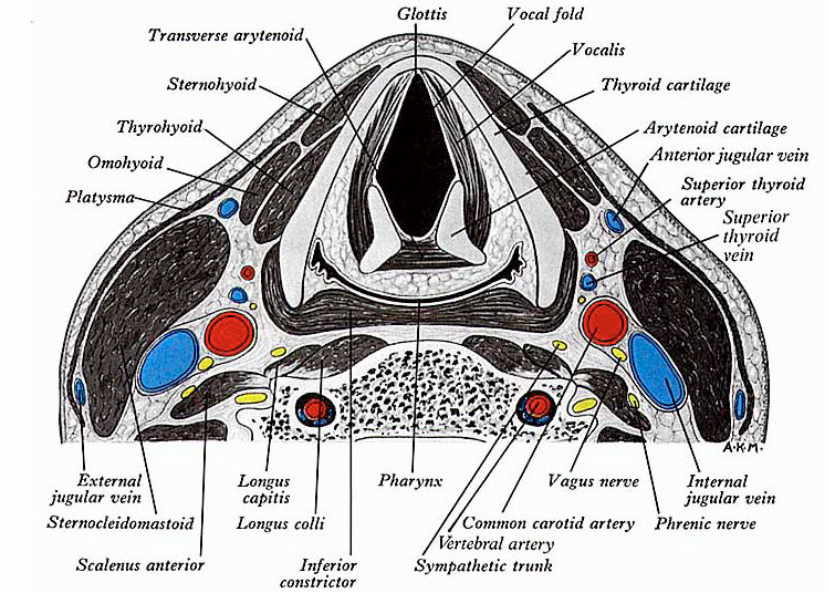


Trans Section Through Mediastinum at the Level of T3 & T4

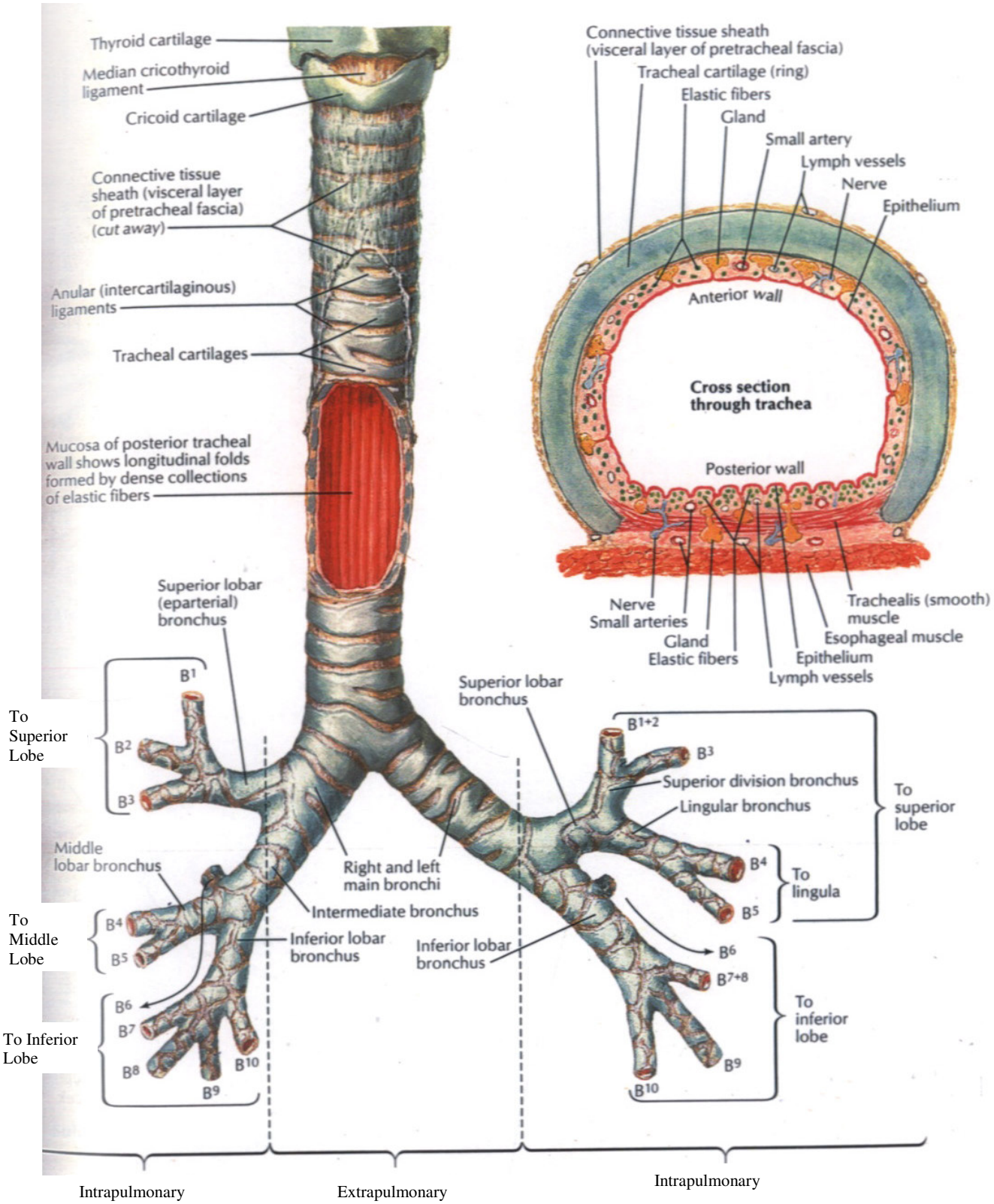


Transverse Section through the Ventral Region of the Neck, between the Fifth and Sixth Cervical Vertebrae: Superior Aspect





TRACHEA AND BRONCHI



RELATIONS OF THE TRACHEA

Cervical Part of the Trachea - Anterior Relations

The cervical trachea is crossed by skin and by the superficial and deep fasciae. It is also crossed by the jugular arch and overlapped by the sternohyoid and sternothyroid muscles. The second to fourth tracheal cartilages are crossed by the isthmus of the thyroid gland, above which an anastomotic artery connects the bilateral superior thyroid arteries; below this and in front are the pretracheal fascia, inferior thyroid veins, thymic remnants and the arteria thyroidea ima (when it exists). In children the brachiocephalic artery crosses obliquely in front of the trachea at or a little above the upper border of the manubrium; the left brachiocephalic vein may also rise a little above this level.

Posterior Relations

Behind the cervical trachea is the oesophagus, running between the trachea and the vertebral column; the recurrent laryngeal nerves ascend on each side, in or near the grooves between the sides of the trachea and oesophagus.

THORACIC PART OF THE TRACHEA

These are the paired lobes of the thyroid gland descending to the fifth or sixth tracheal cartilage, and the common carotid and inferior thyroid arteries.

Anterior Relations

As it descends through the superior mediastinum, the thoracic trachea lies behind the following: the manubrium sterni, the attachments of the

sternohyoid and sternothyroid muscles, the thymic remnants, the inferior thyroid and left brachiocephalic veins, the aortic arch, the brachiocephalic and left common carotid arteries, deep cardiac plexus and some lymph nodes; the brachiocephalic and left common carotid arteries come to lie respectively right and left of the trachea as they diverge upwards into the neck.

Posterior Relations

Behind the trachea is the oesophagus, separating it from the vertebral column.

Lateral Relations

On the right are: the right lung and pleura, right brachiocephalic vein, superior vena cava, right vagus nerve and the azygos vein; on the left: the arch of the aorta, left common carotid and left subclavian arteries.

The left recurrent laryngeal nerve is at first situated between the trachea and aortic arch, then in or just in front of the groove between the trachea and the oesophagus.

Right Principal Bronchus and Its Branches

The right principal bronchus is wider, shorter and more vertical than the left, being about 2.5 cm long. It gives rise to its first branch, the superior lobar bronchus, and then enters the right lung opposite the fifth thoracic vertebra. The greater width and more vertical course of the right principal bronchus explain why foreign bodies enter it more often than the left. The azygos vein arches over it and the right pulmonary artery lies at first inferior, then anterior

to it. After giving off the superior lobar bronchus, which arises posterosuperior to the right pulmonary artery, it crosses the posterior aspect of this artery to enter the pulmonary hilum postero-inferior to the artery, where it divides into middle and an inferior lobar bronchus.

The trachea and extra pulmonary bronchi have a framework of incomplete rings of hyaline cartilage, united by fibrous tissue and smooth muscle and lined internally by mucosa.

Tracheal Cartilages

These vary from 16 to 20 in number, each an imperfect 'ring' surrounding approximately the anterior two-thirds of the tracheal circumference; behind, where they are deficient, the tube is flat and is completed by fibro-elastic tissue and smooth muscle. The cartilages are horizontally stacked, separated by narrow intervals and are about 4 mm vertically and 1 mm in thickness; their external surfaces are vertically flat, their internal surfaces convex. Two or more cartilages often unite, partially or completely, and sometimes bifurcate at their ends. They are composed of hyaline cartilage but may become calcified in the aged. In extrapulmonary bronchi, cartilages are shorter, narrower and less regular but generally similar in shape and arrangement.

The first and last tracheal cartilages differ from the rest , the first cartilage is the broadest. It is often bifurcate at one end and is connected by the cricotracheal ligament to the inferior border of the cricoid and sometimes blended with the cricoid or second cartilage. The last cartilage is centrally thick

and broad and its lower border, the carina, is a triangular hook-shaped process, curving down and backwards between the bronchi. On each side it forms an imperfect ring, enclosing the start of a principal bronchus. The penultimate cartilage is centrally broader than the others.

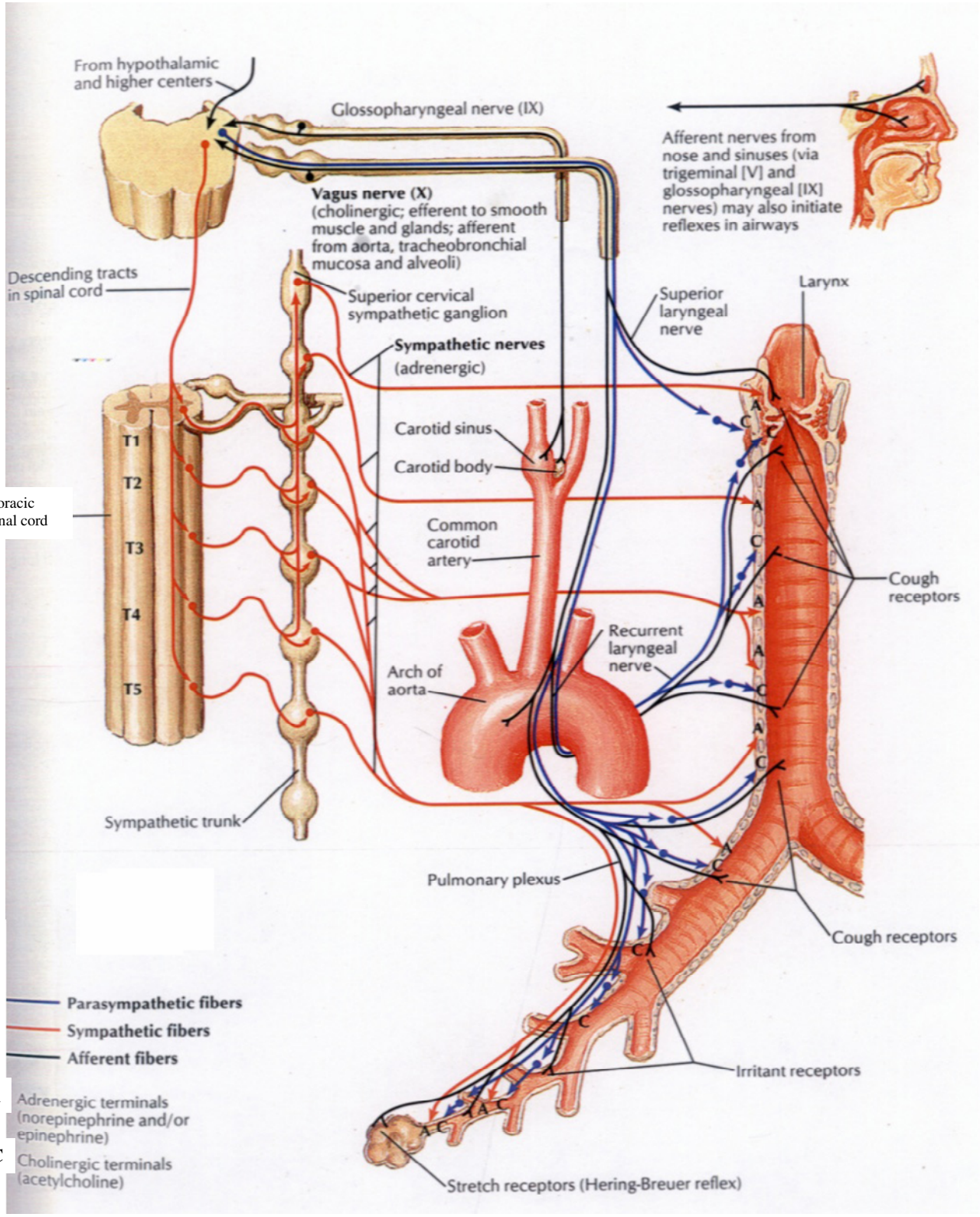
Bronchial Cartilages

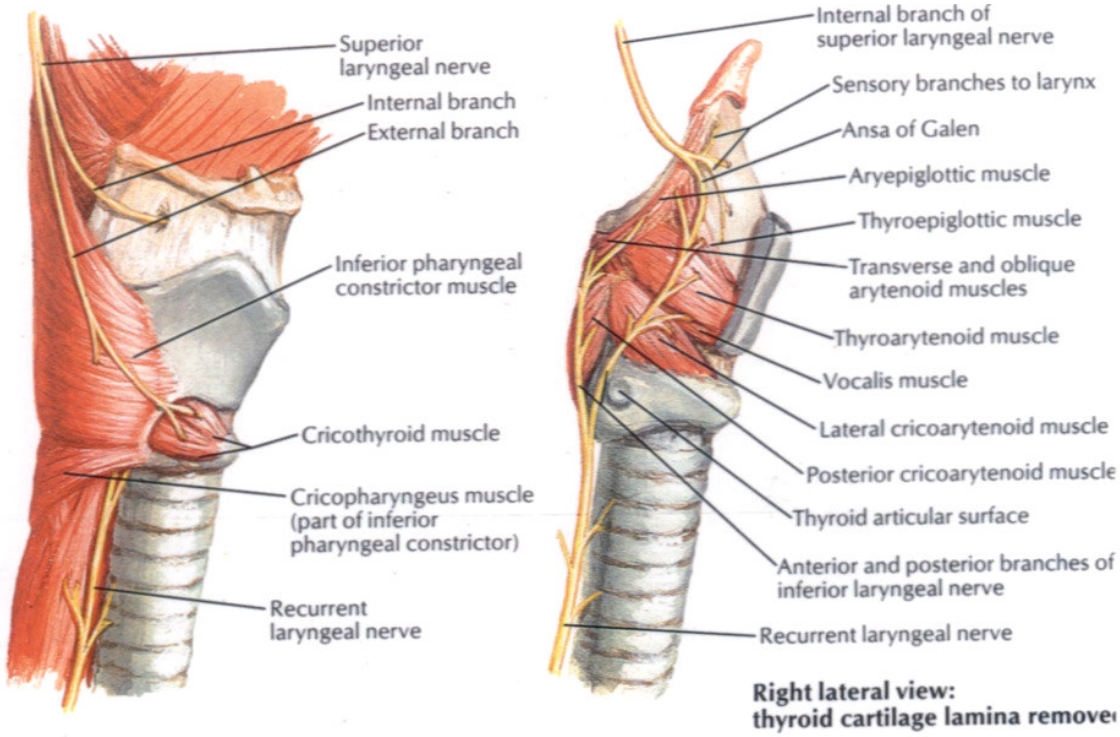
The irregularity of the cartilaginous plates in the extra pulmonary bronchi increases distally; as the major bronchi approach their lungs and lobes, the plates invade their dorsal aspects but never quite encompass their bifurcations. In intrapulmonary bronchi, plates of cartilage progressively form less and less of the bronchial wall, disappearing where the bronchioles begin.

Fibrous Membrane

Each cartilage is enclosed in perichondrium, continuous with a dense fibrous membrane situated between the adjacent cartilages, and filling in the back of the trachea. The perichondrium and membrane are mainly composed of collagen with some elastin fibres; fibres cross each other diagonally, allowing changes in luminal diameter, the elastic component providing some recoil from stretching. Smooth muscle fibres occur in the membrane posteriorly; most are transverse, being attached to the perichondrium at the ends of the cartilages and forming a transverse sheet between them. Contraction, therefore, alters the cross-sectional area of the trachea and bronchi. A few external longitudinal fibres also occur. Smooth muscle in the intrapulmonary bronchi is not attached to cartilages and, where the latter begin to disappear, i.e. in smaller bronchi, contraction may actually obliterate the lumen.

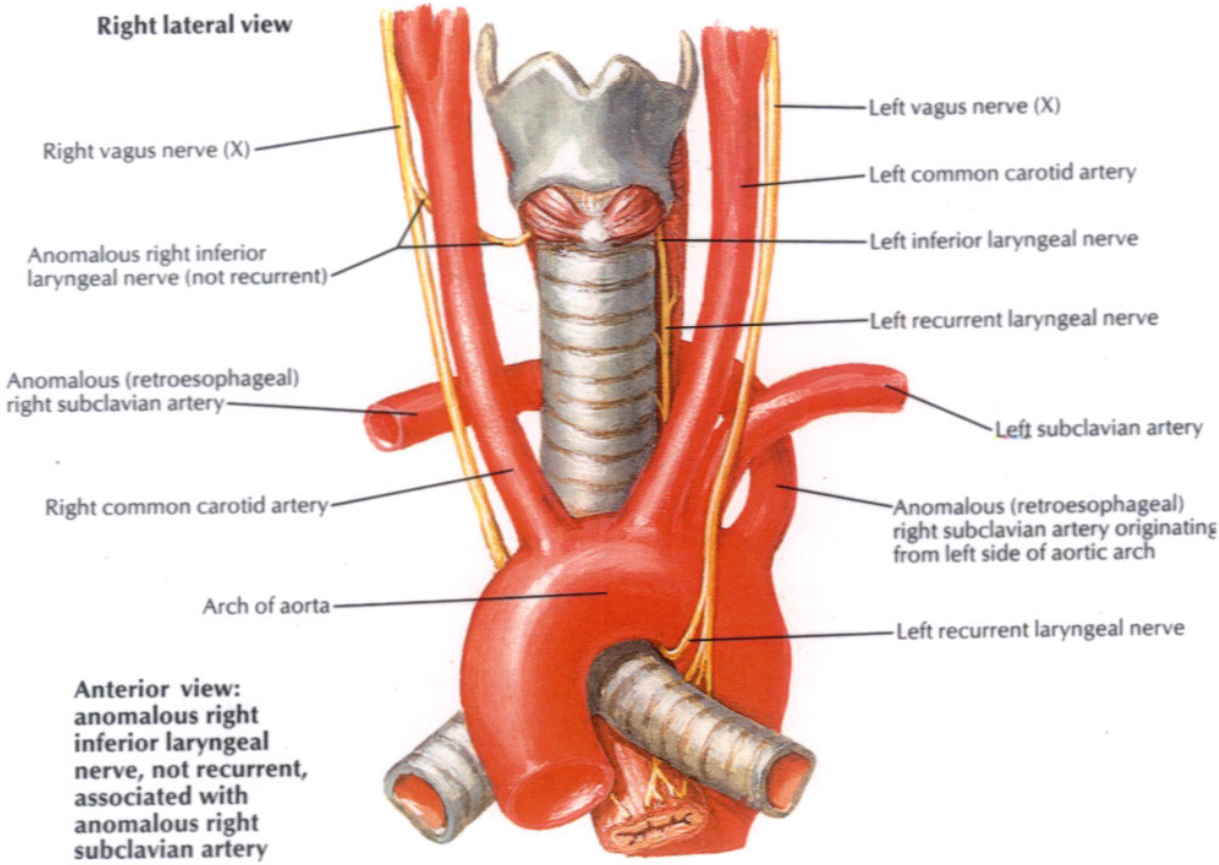
INNERVATION OF TRACHEOBRONCHIAL TREE





Right lateral view

Right lateral view: thyroid cartilage lamina remove



Anterior view: anomalous right inferior laryngeal nerve, not recurrent, associated with anomalous right subclavian artery

Mucosa (Tunica Mucosa)

The mucosa is continuous with and closely resembles that of the larynx above and the intrapulmonary bronchi below, being a layer of pseudostratified ciliated columnar epithelium interspersed with goblet cells, both lying on a basal lamina. Some pseudostratified cells possess unusually large nuclei and may be polytene in chromosomal content. Numerous lymphocytes usually occur deep in the epithelium. The cilia impel mucus towards the laryngeal inlet (aditus). Deep to the basal lamina are a lamina propria with abundant longitudinal elastic fibres and a submucosa of loose connective tissue, containing larger blood vessels, nerves and most of the tubular (tracheal) seromucous glands and lymphoid nodules; external to the submucosa are the perichondrium and the fibrous membrane. Most external of all is the deep fascia, merging with the fascial planes of the surrounding muscles, oesophagus and associated structures.

Vessels, Nerves and Lymphatic drainage

The trachea is supplied with blood mainly by the inferior thyroid arteries, while its thoracic end is also supplied by the bronchial arteries, whose branches ascend to anastomose with the former; all the vessels also supply the oesophagus. Veins draining the trachea end in the inferior thyroid venous plexus. The lymph vessels pass to the pretracheal and paratracheal lymph nodes. The nerve supply is from the tracheal branches of the vagi, recurrent laryngeal nerves and the sympathetic trunks and is distributed to the tracheal muscle and mucosa. Sympathetic nerve endings evoke bronchodilatation by

releasing catecholamines; they may also exert a direct adrinergic effect on glandular acini in the bronchi. Parasympathetic activity which is cholinergic causes broncho-constriction. Many small postsynaptic ganglia occur in the autonomic plexuses of the tracheal and bronchial walls. Afferent fibres include those with sensorimotor functions and can mediate local inflammatory influences by means of their collateral terminae which can release neuropeptides to trigger mast cell degranulation. The tracheal lymphatic drain to the pretracheal and paratracheal group of nodes.

The trachea is about 2 cm wide and extends almost vertically in the midline from the cricoid cartilage to the sternal angle, inclining slightly to the right. The right principal bronchus runs from the trachea down to the right for 2.5 cm to the right hilum behind the sternal end of the right third costal cartilage. The left principal bronchus runs for 5 cm more obliquely to its left and down to the hilum behind the left third costal cartilage, 3.5 cm from midline. The trachea may be opened by median vertical incision above the thyroid isthmus (high tracheotomy) or below it (low tracheotomy), the latter being more difficult because the trachea recedes as it descends and has hazardous anterior relations. The trachea may be compressed by pathological enlargements of the thyroid gland, thymus and aortic arch.

PATHOPHYSIOLOGY OF TRACHEO BRONCHIAL TREE

In quiet breathing, air normally enters through the nose and is warmed and humidified in the nasal passage, approaching body temperature and 100 percent humidity by the time it reaches the midtrachea. Tracheostomy bypasses the upper airways, including the nose / mouth, pharynx, and larynx, alters normal upper as well as lower airway functions.

The patency of the trachea is supported by C- shaped cartilages opening posteriorly. Bundles of smooth muscle fibres are present mostly in the posterior membranous part and are attached to both ends of the semicircular cartilages. Contraction of these muscles reduces tracheal compliance and tracheal diameter but at the same time stabilizes against dynamic compression. In the smaller airways in the lung parenchyma, the cartilage becomes more irregular. Their patency is maintained in part by the elastic recoil and interdependence of parenchymal lung tissues. In peripheral airways the lumen is probably stabilized by the presence of pulmonary surfactant, which reduces the surface tension of the alveolar and small airway lining.

Tracheal walls are lined with pseudostratified epithelium consists of ciliated cells, noncilated serous and brush cells, and abundant mucus secreting goblet cells. The submucosa contains numerous serous and mucous cell glands, which are major contributors of the mucus in the respiratory tract. The mucosal surface is covered by a serous fluid layer in which the cilia beat. During quiet inspiration, airflow through the trachea is largely laminar, although turbulence does develop in to lower tracheas the flow rate increases. When dry air reaches the trachea either by mouth breathing or via an endotracheal tube, there is very poor adjustment of temperature and humidity, resulting in drying of the tracheobronchial mucosa.

During quiet breathing the patency of the lower trachea is supported by negative pleural pressure. During forced expiration pleural pressure increases considerably above the atmospheric pressure and in turn increases alveolar pressure. The resultant pressure gradient between the alveoli and the airway opening at the mouth (atmospheric) produces the expiratory flow. In the periphery of the lung. The pressure within the airway is higher than the pleural pressure because of the elastic recoil of the lung. As the air moves downstream from the periphery toward the major airways, airway pressure decreases and at some point becomes identical to the pleural or tissue pressure surrounding the airway. This point is termed the equal pressure point (EPP).

Downstream from the EPP to the lower trachea at the thoracic inlet, airway pressure becomes lower than the surrounding pleural pressure and consequently these airways are subjected to dynamic compression. The membranous parts of the trachea and major bronchi are invaginated into the airway lumen and the cross section becomes crescent-shaped or even nearly occluded. Under these circumstances the expiratory flow rates become effort-independent. Dynamic compression is an Integral part of the coughing mechanism, in which an increase in air flow velocity in the affected central airway helps to propel mucus toward the mouth.

During forced inspiration, the lower trachea and bronchi are inflated by surrounding negative pleural pressure. The upper trachea, by contrast, is subjected to dynamic compression, the degree of which depends on the patency of the larynx and above and on the tracheal compliance. Smooth muscle tone decreases the tracheal compliance and the cross-sectional area but stabilizes

both the upper and lower trachea by resisting dynamic compression. In laryngotracheomalacia severe limitation of inspiratory air flow may occur during forced inspiration.

The flow pattern in the central airways below the carina is turbulent, particularly when the flow rate is increased. In peripheral airways, dramatic increases in the total cross-sectional area and reduction in flow velocity cause the air flow pattern to become laminar. At the level of the alveolar ducts and alveolar sacs, the velocity becomes so low that gas exchange depends largely on molecular diffusion.

PHYSIOLOGIC ALTERATIONS WITH TRACHEOSTOMY

Effect on airway resistance

Air flow resistance of the upper airway with nose breathing (including nose, pharynx, and larynx) is as much as 80 percent of the total airway resistance, and with mouth breathing it is nearly 50 percent. Thus, theoretically, there should be a significant reduction in total airway resistance with tracheostomy. In reality, however, flow resistance through the tracheostomy cannula may be as high as or even higher than that through the normal upper airways, because of the cannula's relatively small diameter (7 to 8 mm ID for an adult-sized cannula, and the flow resistance is decreased drastically when the tracheostomy is performed to alleviate severe upper airway obstruction, which occurs most commonly at the level of the cricoid cartilage or at the glottis. Removal of the severe obstruction results in a marked reduction in the work of breathing and oxygen consumption and may relieve the patient's

sensation of respiratory distress and suffocation. Sudden relief of severe upper airway obstruction is occasionally followed by a sudden onset of pulmonary edema, particularly in children. The etiology of this pulmonary edema is not clear, but a marked negative airway pressure produced by inspiratory effort against obstruction (Muller's maneuver) with increased capillary-alveolar transmural pressure is thought to be responsible, together with catechol-mediated shift of pulmonary blood volume and increased pulmonary capillary permeability due to tissue hypoxia.

A tracheostomy reduces the anatomical dead space by as much as 100 ml in adults. This may be of some help in patients, such as those with emphysema, whose tidal volume is deceased in relation to physiological dead space. More careful control of oxygen therapy in recent years has decreased the indication of tracheostomy in chronic obstructive lung disease.

Effect on Gas Temperature and Humidification

During quiet tidal breathing, the inspired air is warmed and humidified through the nasal passages. By the time it reaches the nasopharynx, air with an ambient temperature of 23°C is warmed to approximately 33°C and fully saturated. Since the trachea is ill-equipped to humidify inspired air, bypassing of the upper airway causes cold, dry air to reach the carina and beyond. When cold air was inspired, the bronchial air temperature fell by 2.5°C, and with increasing ventilation through the mouth, it dropped to an average of 27°C. Although such a reduction in bronchial air temperature was transient, it is likely that the lack of nasal air conditioning in tracheostomized patients would cause drying of tracheobronchial mucosa.

It is generally recognized that air inspired via a tracheostomy should be humidified. However, humidification of inspired air is not practiced widely for home care or even for hospital care of patients with chronic tracheostomy. These patients are prone to atelectasis owing to drying of mucous secretions and pulmonary infection due in part to a lack of a normal filtering mechanism of particulate matter and to a decreased or absent mucociliary clearance mechanism. Infants and children with long-term tracheostomy appear particularly vulnerable to airway and pulmonary complications with associated high morbidly and mortality.

Effect on Cough Mechanism

Cough is an important and powerful adjunctive mechanism to expel material such as foreign bodies and excessive secretions, which may not be cleared effectively by the usual airway defense mechanisms such as mucociliary transport and macrophages. There are three basic phases in the cough mechanism. It usually starts with a rapid and brief inspiration of air deeper than the usual resting tidal volume (inspiratory phase). The glottis is then closed tightly for a brief moment. During this time the expiratory muscles of the thorax and abdomen contract actively, raising pressure in the abdominal, pleural and alveolar spaces to 50 to 100 mmHg or more (compressive phase). Then the glottis is suddenly opened actively (expiratory phase). Expiratory flow accelerates rapidly, reaching a peak flow of 10 L/s or more within 50 ms. at the same time, the lower trachea and bronchi are subjected to dynamic compression. This produces a transient spike of flow at a velocity that may approach 250 ms, or three-quarters of the speed of sound. Oscillation of airway

tissue and air cause an explosive sound and may facilitate the dislodging of secretions from the airway wall into the moving air stream for removal. Since dynamic compression of the lower trachea and major bronchi appears to be the most important mechanism in coughing, effective airway clearance is possible in tracheostomized patients by cough like maneuvers not utilizing the closure of the larynx. The effectiveness of airway clearance, however, would be diminished in these patients since their ability to produce high air now Velocity is compromised. This is particularly true in those with muscle weakness, pain, and airway obstruction. In these patients alternative means of clearing airway secretions, such as endotracheal suctioning and artificial coughing are needed.

Effect on Laryngeal Closure Reflex

Tracheostomy is not infrequently complicated by aspiration of food particles and resultant pulmonary infection, particularly in infants and children. There has been a suggestion, based on clinical observation, that coordination of the laryngeal reflexes of respiration and deglutition may be lost following prolonged tracheostomy, and that after tracheal decannulation it is necessary for infants and children to relearn the swallow-laryngeal closure reflex. In the pharyngeal phase of swallowing, the epiglottis tilts posteriorly to cover the laryngeal inlet; the glottis then closes, the esophagus opens, and the peristaltic wave forces the bolus of food into the upper esophagus. The entire process occurs in less than 1s, while respiration is reflexly interrupted for only a fraction of a respiratory cycle. Such a glottic closure has been thought to be phylogenetically primitive and physiologically stable.

These effects of tracheostomy on the regulation of airway resistance and the protection of the lower airways from aspiration have important clinical significance. Laryngeal adductor dysfunction may result in the failure of glottic closure, resulting in aspiration of food materials. Before the tracheal decannulation the medullary respiration centre must readjust to the change from breathing through the tracheostomy to breathing through the upper airway and the larynx.

BIOCHEMICAL CHANGES IN UPPER AIRWAY OBSTRUCTION

Main Biochemical Changes

1. Arterial hypoxia (hypoxemia)
2. Retention of CO2 (hypercapnia)
3. Respiratory and metabolic acidosis (decreased blood ph)
    a. Increased lactic acid accumulation
    b. Increased carbonic acid accumulation
    c. If slow compensation occurs, decreased alveolar PO_2 (< 50mm) or arterial PO_2 (< 70%) causes stimulation of carotid and aortic bodies (< 40 mm Hg = 70% decrease of O_2 in Hb)

SEQUENCES TO OBSTRUCTION

Increased respiratory effort

Tachycardia

Peripheral vaso constriction

Hypertension

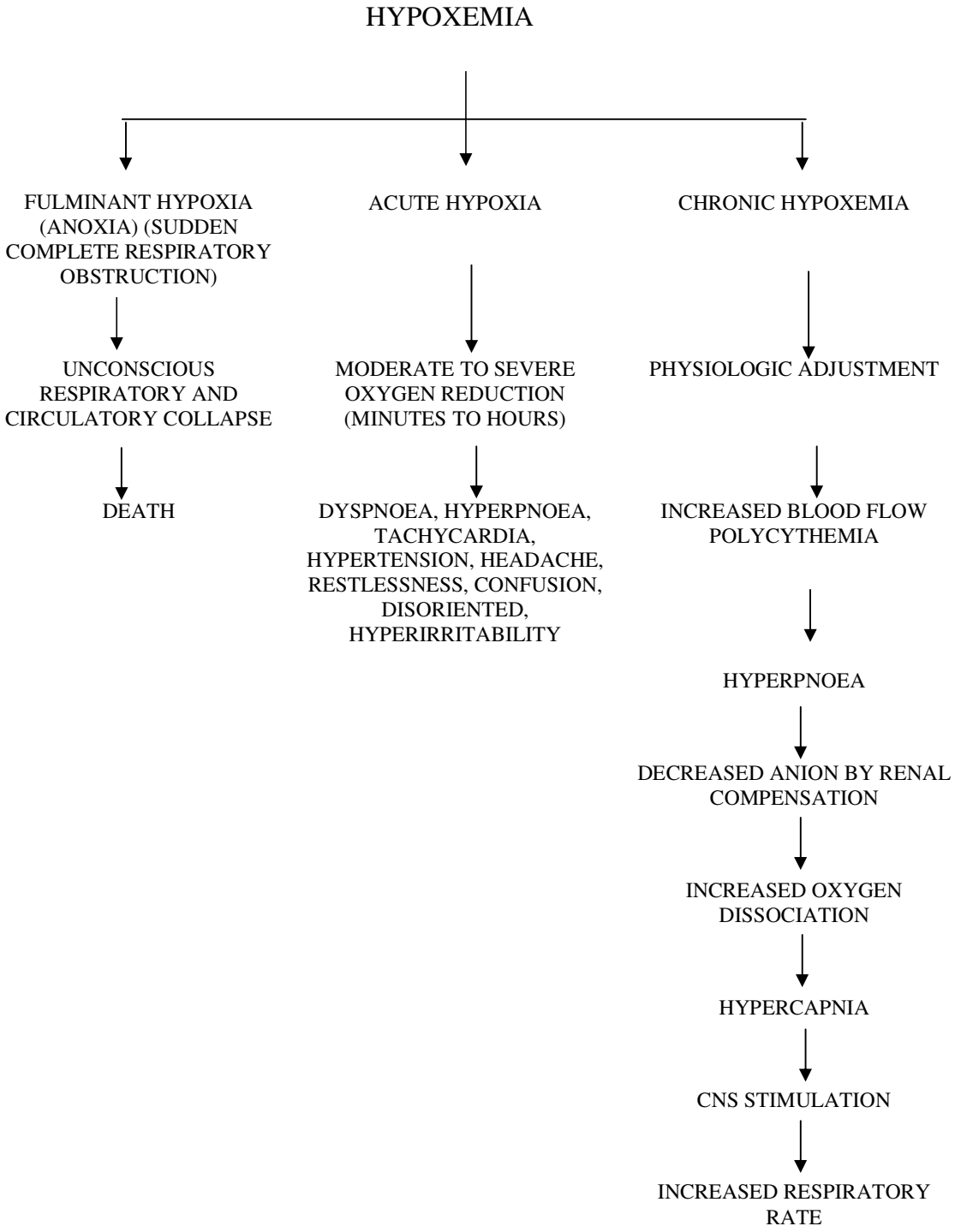
Increased Pulmonary vascular resistance

Increased adrenergic activity

Increased cerebral cortical activity (chemoreceptor stimulation)

Alveolar hypoventilation

PATHOPHYSIOLOGY OF UPPER AIRWAY OBSTRUCTION



ACID- BASE PHYSIOLOGY

The pH of the body fluids affects the electrical charges of the chemical substances throughout the body and hence is of great importance. Under normal circumstances the hydrogen ion concentration varies very little from the normal value of 40nm/L. Acid and base are continuously added to the extra cellular fluid (ECF). The process of hydrogen ion regulation involves 3 basic step,

1. Chemical buffering by ECF and ICF buffers
2. Control of partial pressure of CO_2 in the blood by alterations in alveolar ventilation.
3. Control of plasma bicarbonate concentration by changes in renal H+ excretion.

Bronsted - Lowrey Definition

Acid is a H^+ (proton) Donor.

Base is a H^+ (proton) acceptor.

60% of the acid load is buffered in the ICF. The most important ICF buffer is the imidazole ring in histidine. The bicarbonate carbonic acid buffer system, in the presence of carbonic anhydrase, high solubility of CO_2, ability of kidney to synthesize and eliminate HCO_3 and efficient removal of CO_2 by the lungs, becomes a very effective and powerful buffer system of the body.

Henderson's equation is a non-logarithmic version of law of mass action which best numerically expresses the relationship between H^+, PCO_2 and HCO_3.

$$H^+ \text{ nmol/L} = \frac{24 \times PC_2 \text{mmHg}}{(HCO_3^-) \text{ mmol/L}}$$

The logarithmic version of this equation is the Henderson Hasselbach equation. pH is the negative logarithm of (H^+) in nmol/L

$$pH = 6.1 + \log \frac{(HCO_3^-) \text{ mmol/L}}{0.03 \times PCO_2 \text{ mmHg}}$$

The acid base status of the ECF is customarily evaluated in terms of HCO_3^- and $PaCO_2$. By convention the CO_2 is referred as the respiratory component and HCO_3 as the metabolic component.

Normal ABG values:

pH	➔	7.36 to 7.44
$PaCO_2$	➔	35-45 mmHg (4.7 to 6.00 Kpa)
Actual HCO_3^-	➔	21-28 mmol/L
Standard HCO_3^-	➔	21-27 mmol/L
Base Excess	➔	$\pm$ 2 mmol/L
PaO_2	➔	Over 90mmHg (12.0 KPa) on room air)

Compensatory Mechanism

The body tries to compensate for any derangements of acid base balance by trying to bring the pH back to normal. This it does by changing of the 2 factors namely $PaCO_2$ and (HCO_3^-), by altering the other factors in the same direction.

The compensatory process attempt to restore (H^+) to normal, but is usually not complete. Over compensation does not occur.

The respiratory compensation for a primary metabolic disorder is almost immediate (it starts within a few minutes and becomes maximal within an hour), where as the renal compensation for a primary respiratory disorder is much slower in onset, taking several hours to start and 1-2 days to become maximal.

When two or more primary acid-base disorders are present, the condition is referred to as a mixed acid-base disorder.

ACID BASE DISORDERS

The types are:

1. Respiratory acidosis.
2. Respiratory Alkalosis
3. Metabolic acidosis
4. Metabolic alkalosis.

RESPIRATORY ACIDOSIS

It is another name for hypercapnia. It may be a direct result of a decrease in the alveolar ventilation due to any cause. It presents as a combination of high H+ with high $PaCO_2$.

ABG Findings

	Respiratory Acidosis	Metabolic Compensation
pH	↓↓	↓
$PaCO_2$	↑	↑
H^+	↑	↑↑
HCO_3^-	N/↑	↑
Base excess	N	↑

Causes

Acute and chronic respiratory acidosis can be caused by

1. Inhibition of medullary respiratory centre.
2. Disorders of the respiratory muscle and chest wall.
3. Upper airway obstruction.

4. Disorders affecting gas exchange across pulmonary capillary.
5. Mechanical ventilation.

Effects

1. CO_2 narcosis
2. Sympathetic Stimulation
3. Arrthymias
4. Pulmonary vasoconstriction

RESPIRATORY ALKALOSIS

It is also known as hypocapnia. It is usually a result of an increase in the alveolar ventilation. If presents as a low (H^+) in combination with a low $PaCO_2$.

ABG Findings

	Respiratory Alkalosis	**Metabolic Compensation**
pH	↑↑	↓
$PaCO_2$	↓↓	↓↓
H^+	↓	↓/N
HCO_3^-	↓	↓↓
Base excess	N	↓

Causes

They include

1. Hypoxaemia

2. Pulmonary disease
3. Direct stimulation of medullary respiratory centre
4. Mechanical ventilation

Effects

1. Myocardial irritability
2. CNS irritability.
3. Hypophosphataemia

METABOLIC ACIDOSIS

This disorder is caused by either an increased loss of bicarbonate or a failure of new renal bicarbonate generation. The most important lab clue to this type of acidosis is an entity called anion gap which calculates the unmeasured anions in the plasma like lactate and ketone bodies.

ABG Findings

	Metabolic Acidosis	**Respiratory Compensation**
pH	↓↓↓	↓↓
$PaCO_2$	N	↓
H^+	↑↑	↑
HCO_3^-	↓↓	↓↓↓
Base excess	↓↓	↓↓

Causes

These include inability to excrete dietary H^+ load as in renal failure or renal tubular acidosis. Increased H^+ production as in lactic acidosis, Diabetic Keto Acidosis (DKA) and HCO_3^- loss as in diarrhea.

Effects

1. Hyperventilation
2. Venticular arrhythmias
3. Myocardial Depression
4. Hyperkalaemia
5. Vasodilatation

METABOLIC ALKALOSIS

A combination of low H^+ ion concentration and high HCO_3^- occurs in this disorder which occurs due to increased production of bicarbonate or contraction of ECF volume.

ABG Findings

	Metabolic Alkalosis	**Respiratory Compensation**
pH	↑↑	↑
$PaCO_2$	N	↑↑↑
H^+	↑↑	N
HCO_3^-	↑↑	↑↑
Base excess	↑↑	↑↑

Causes

It includes H^+ ion loss due to vomiting and renal loss and retention of HCO_3^- due to massive blood transfusion and milk alkali syndrome.

Effects

1. Parasthesia
2. Corpopedal Spasm
3. Arrythmias

ARTERIAL BLOOD GAS SAMPLE

An arterial sample can be obtained by either a percutaneous puncture of an artery or aspiration from an indwelling arterial cannula.

If the sample is taken incorrectly, the results of the analysis will be invalid.

Common sites of arterial cannulation

1. Radial artery at the wrist.
2. Brachial artery at the cubital fossa.
3. Femoral artery below the inguinal ligament.

Allen's Test

It is a test to evaluate the patency of ulnar circulation and patency of collaterals supplying the palmar arch. The patient is asked to clench his fist and raise his hand to exsanguinate the palm. Examiner applies pressure over the radial and ulnar artery at the wrist. Patient opens the palm revealing a pale hand. The compression over the ulnar artery is released. Distinct pink colouration occurring within 8 seconds indicates good ulnar circulation. Consequently, the puncture of radial artery is not contra indicated in this patient.

Procedure

Explain the procedure to patient and obtain consent. The wrist is extended by 20-30^0 to bring the radial artery superficially. Under strict aseptic

ARTERIAL BLOOD GAS ANALYSIS

precautions the radial artery pulsation is felt just proximal to the proximal transverse skin crease at the wrist. 0.2 to 0.5ml of local anaesthetic is injected around the artery.

A 20G cannula on needle, is flushed with heparin saline (2 I.U. heparin SO_4/ml). The needle is inserted with bevel facing upwards at an angle of 30^0 to skin and advanced towards the artery.

On puncture of the artery, blood flash is noticed in the hub. The tip of the catheter is then advanced into the artery. Free flow of arterial blood is confirmed and the catheter is secured with tape. A 3 way tap is connected to cannula and flushed with heparin saline to prevent blockage. Indwelling arterial cannula can be retained for 24 to 36 hours and permit serial ABG estimation.

Securing blood sample

Attach heparinised syringe to 3 way tap. Draw 2ml of blood and discard. This ensures that subsequent sample is fresh blood and not diluted by the flushing solution between the tap and the artery. A fresh heparinised syringe is attached again to the tap and 1 to 2ml of blood is gently aspirated and the sample is immediately subjected to analysis.

Key points during ABG analysis

- To avoid iatrogenic errors the sample should be analysed within 3 to 5 minute.
- No air bubbles to be allowed in the sample.

- If transport time is expected to be more than 5 minutes the sample must be kept in melting ice.

- Blood from a hypothermic patient should be warmed to 37^0C before interpretation.

- Patients inspired oxygen concentration is necessary for interpretation of result.

Removal of Catheter

After catheter removal, firm pressure has to be applied to the puncture site for a minimum of 5 minutes before inspecting the area for swelling and bleeding. This will prevent formation of haematoma.

MATERIALS AND METHODS

The study was designed as a prospective cohort study and was conducted between January 2004 to March 2006 at the Upgraded Institute of Otorhinolaryngology, Govt. General Hospital.

After ethics committee approval and informed consent from the patient, all patients attending ENT outpatient department and causalty with stridor and satisfying all the inclusion criteria were drafted in the study.

The inclusion criteria included

1. Patients of either sex aged between 30 to 80 years.
2. Patients with stridor due to upper airway obstruction.
3. Emergency tracheostomy was deemed to be the treatment of choice.

Patients with the following problems were excluded from the study group

1. All intubated, mechanically ventilated patients in medical ICU.
2. Neurosurgical patients requiring tracheostomy for tracheobronchial toileting.
3. Patient with uncorrectable bleeding disorders
   BT > 10mins
   CT > 15 min.
4. Patients with insufficient collaterals in palmar arch.

5. Patient whose pre-operative ABG when analyzed using Miller's ABG assessment protocol revealed a mixed type of acid base disorder indicating the additional presence of a metabolic component.

The patients initially drafted in the study were dropped if their ABG showed complex mixed type of disorders. In all patients requiring active surgical intervention for relief of acute upper airway obstruction, the decision of to perform the tracheostomy or not and when to perform the tracheostomy was left to the decision of the primary treating surgeon (i.e. the admitting unit).

All patients on admission received a I.V. access with 18G cannula and fluid resuscitation with balanced salt solution. The received oxygen 6L/min through mask and their vital parameters were monitored.

Pre-Operative Evaluation

It includes a detailed history and through clinical evaluation. Demographic profiles were recorded and attention was paid to possible risk factors in the disease process. All vital parameters were recorded and continually monitored.

Pre-Operative investigations included

1. Compete haemogram
2. Renal function test → Blood urea, S.creatinine, S.electrolytes
3. Coagulation profile →BT, CT

4. Urine analysis
5. ECG
6. X-ray chest PA view
7. X-ray neck - AP, Lat view
8. E.N.T. Examination

ARTERIAL BLOOD GAS (ABG)

Once it was decided to perform tracheostomy, duty anaesthesiologist / intensivist was contacted. Under his guidance, with strict aseptic precautions and local anaesthesia the left radial artery was cannulated using a 20G cannula and the cannula was secured. The cannula was kept patent by intermittently flushing it with heparin saline (1000 I.U. of heparin sulphate in 500ml NS).

In a 2 ml heparinized, de-aired syringe 1ml of arterial blood sample was drawn and was immediately subjected to ABG analysis using Bayer Health Care and Chiron International.

If the ABG revealed mixed or complex acid-base disorders due to varied causes, the tracheostomy was proceeded with, but the patient was dropped from the study group.

If ABG analysis revealed a respiratory acidosis fitting with the picture expected in upper airway obstruction, the patient was included in the study.

Performance of tracheostomy

Patient was shifted to emergency Operation Theatre after checking I.V access, patient was connected to minimum mandatory monitoring including ECG, ANIBP, SaO_2 and $ETCO_2$.

The duty anaesthetist provided monitored anaesthesia care and appropriate I.V. sedation. He was on stand by for intervention and resuscitation if the condition of the patient deteriorates.

SURGICAL PROCEDURE[40]

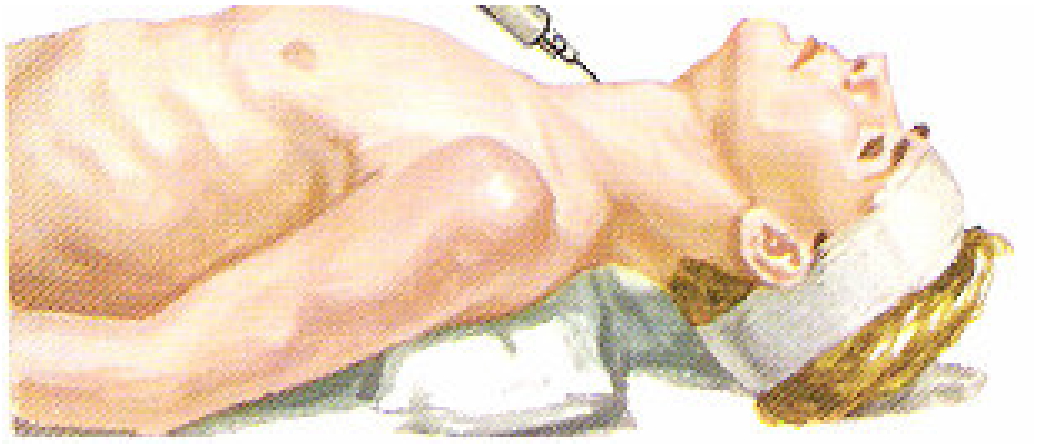
Trachostomy may be satisfactorily performed under local anaesthesia and this may be indicated in a patient with an obstructive lesion. Local anaesthesia is obtained after the test for sensitivity and then by injection of the skin and subcutaneous tissues with 2 percent xylocaine and 1:200000 adrenaline. Before the trachea is opened 0.5ml of 2 percent xylocaine should be injected into the tracheal lumen.

The patient's neck is placed in hyper extension so that the larynx and trachea are prominent. This also allows the trachea to be elevated in relation to the supra sternal notch. The operation is difficult to perform in patients with short thick neck or with disease in the tracheostomy site.

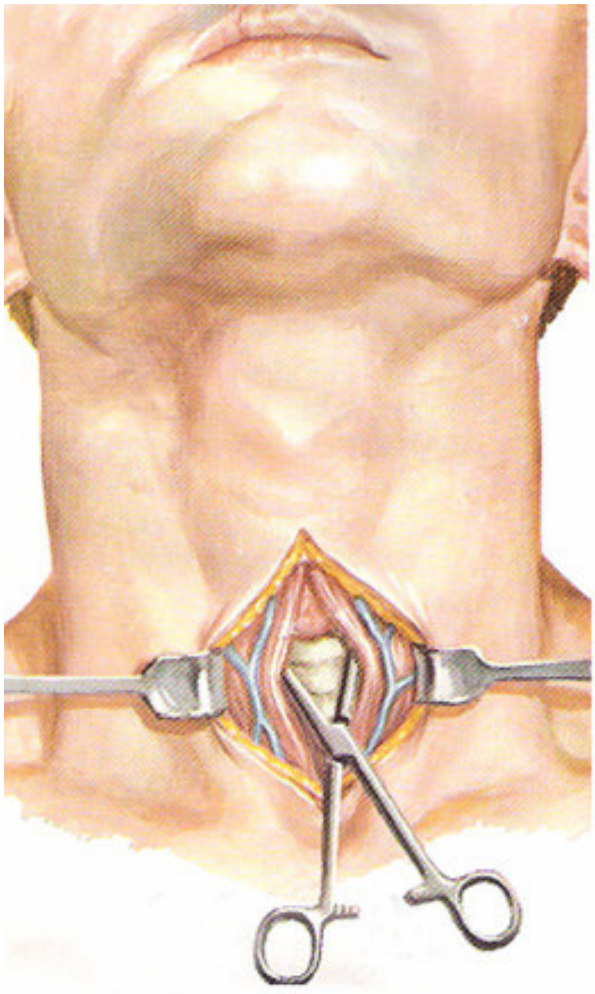
Vertical incision, approximately 5cms in length is made from the lower border of cricoid cartilage to the suprasternal notch (Burns space) in the midline.

The fibrous median raphe in the interval between the right and left sternohyoid muscles is defined and separated with blunt dissection. The sternothyroid muscles on a deeper plane are identified and retracted laterally.

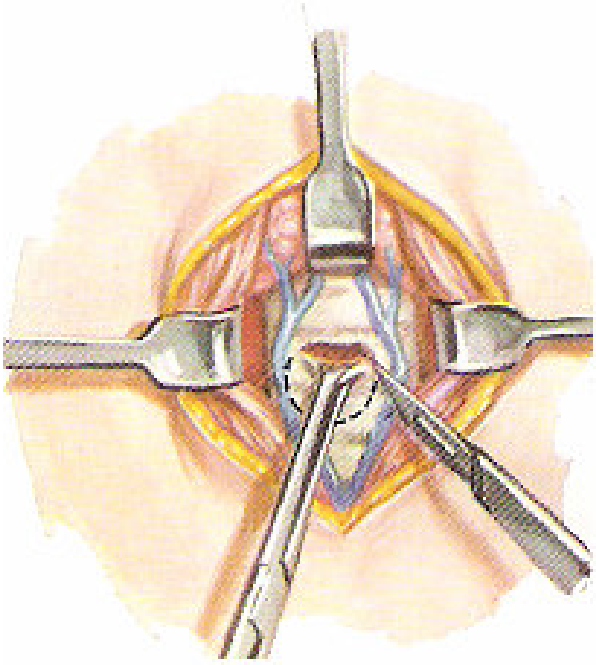
The thyroid gland and part of the trachea will then be visible. Anatomical variation in the size and postion of the thyroid isthmus are to be expected. The thyroid isthmus may be small, not interfering with the approach to the trachea but in most patients it is of sufficient size to need dividing. A small horizontal or vertical incision is made through the pretracheal fascia over the lower border of the cricoid cartilage so that a small hemostat can be



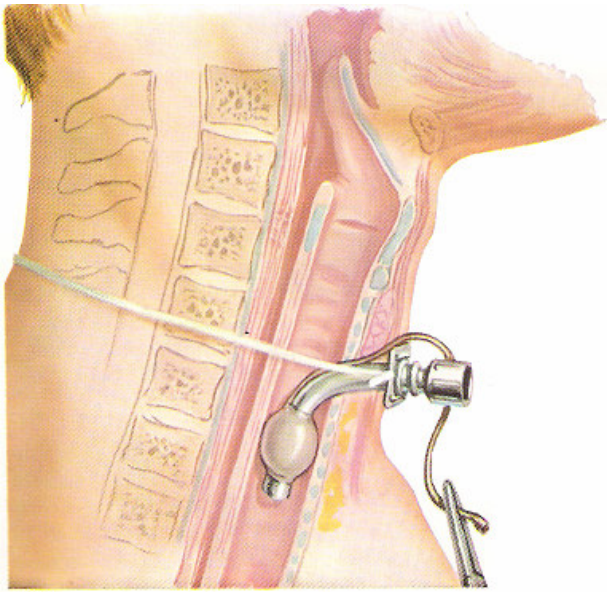
POSITION THE PATIENT: SHOULDERS ELEVATED, NECK EXTENDED.
INFILTRATION OF LOCAL ANESTHETIC



INCISION: VERTICAL MIDLINE
STRAP MUSCLES SEPARATED
BY HEMOSTAT



THYROID ISTHMUS ELEVATED; PRETRACHEAL VEINS SEPARATED; WINDOW EXCISED IN TRACHEA

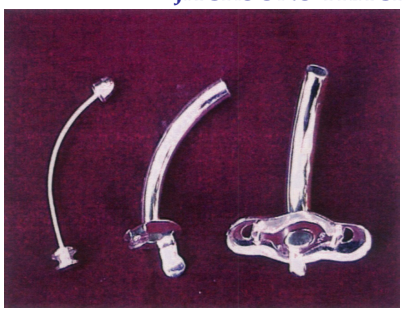


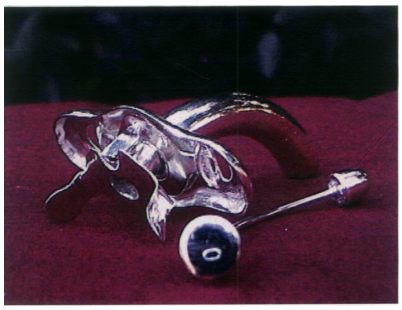
TRACHEOSTOMY TUBE INSERTED AND TIED IN PLACE WITH UMBILICAL TAPE, CUFF INFLATED AFTER INTRODUCTION

FULLERS TRACHEOSTOMY TUBES

PORTEX CUFFED TRACHEOSTOMY TUBE

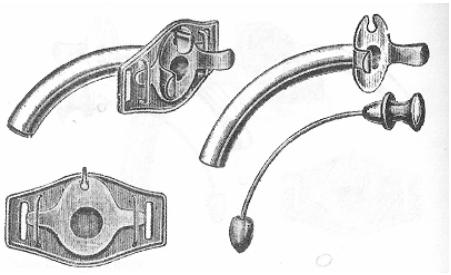
JACKSONS TRACHEOSTOMY TUBES





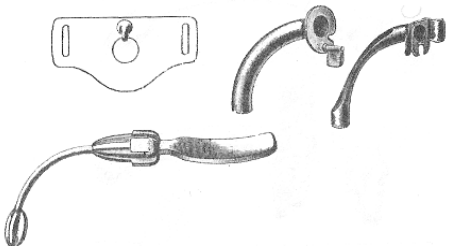
ANCIENT TRACHEOSTOMY TUBES

NEGUS TRACHEOSTOMY TUBE

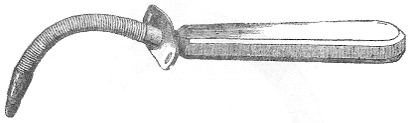


SILVER, WITH RECESSED VALVED INNER TUBE, LAIN INNER TUBE WITH PILOT

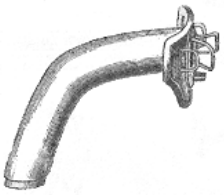
CUBLEY'S TRACHEOSTOMY TUBE. Silver with pilot



HOWSE'S TRACHEOSTOMY TUBE

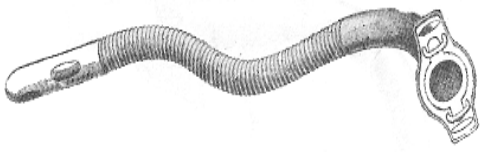


PARKER'S TRACHEOSTOMY TUBE

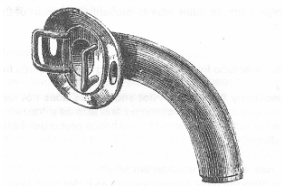


SILVER WITH PILOT

KONIG'S FLEXIBLE SILVER TRACHEOSTOMY TUBES

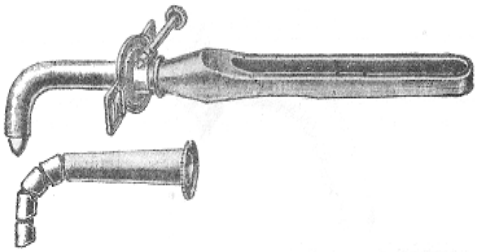


EDINBURGH PATTERN TRACHEOSTOMY TUBE

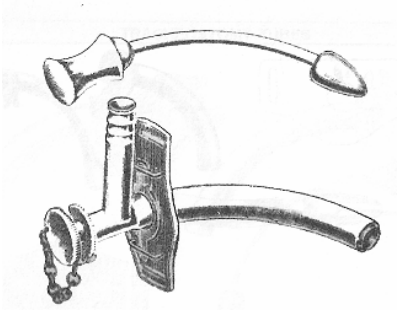


WITH MOVABLE SHIELD, SILVER WITH PILOT

DURHAM'S LOBSTER TAIL TRACHEOSTOMY TUBE

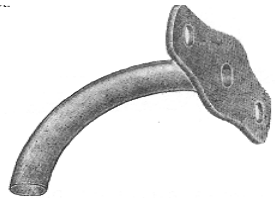


McMATH'S TRACHEOSTOMY TUBE

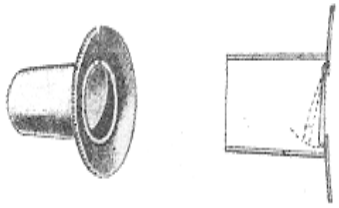


SILVER, WITH TUBE AND ANGLED SIDE BRANCH WITH PLUG STOPPER

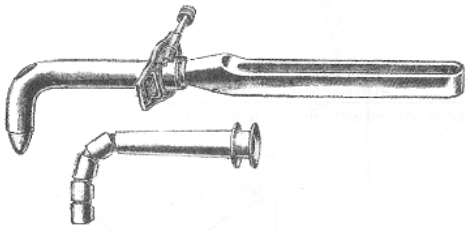
MORRANT'S BAKER'S TRACHEOSTOMY TUBES



De SANTI'S VALVES

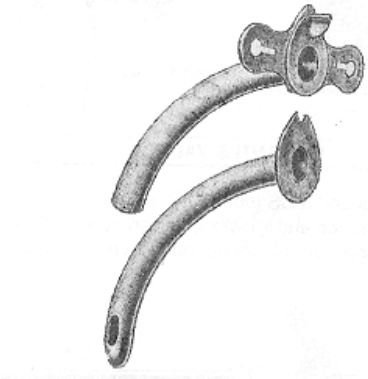


DURHAMS LOBSTER TAIL TRACHEOSTOMY TUBE



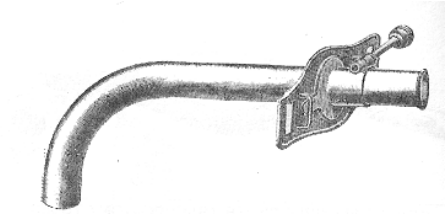
SILVER, WITH OPENING ON BEND AN FITTED WITH VALVED INNER TUBE AND PILOTS

PORTMANNS TRACHEOSTOMY TUBE

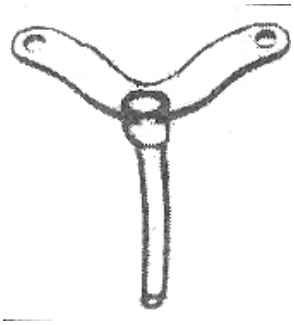


PORTEX NON CUFFED TRACHEOSTOMY TUBE

DURHAMS LOBSTER TAIL TRACHEOSTOMY TUBES, JUMBO PATTERN

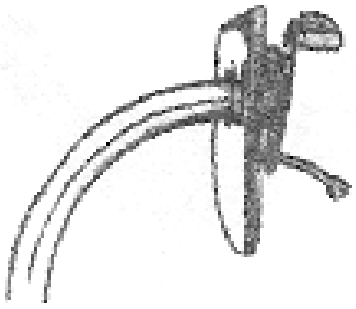


WARNE FRANKLIN TUBE



SHILEY PLASTIC TUBES

FENESTRATED OUTER TUBE



"An Ivory Tracheostomy Set" was used in the 1800's by Physicians who performed Tracheostomy for children suffering from epidemics of Diptheria and Polio !

Antique tracheostomy tubes made of silver, used in 1800's

WORLD'S FIRST SCULPTURED TRACHEOSTOMY PATIENT

A statue in the historical museum of Venice, dating back to 740 A.D.

inserted into the incision and directed inferiorly behind the thyroid isthmus and anerior to trachea. After the proper plane of cleavage between thyroid isthmus and trachea has been determined with the small haemostat, a larger haemostat is inserted to completely separate the thyroid isthmus from the anterior tracheal wall by blunt dissection.

To perform a tracheostomy at the proper level, it is often necessary to transect and ligate the thyroid isthmus. Occasionally the isthmus is small or so placed that ligation is not necessary. For quicker exploration of trachea thyroid isthmus can be retracted superiorly or inferiorly.

Before the trachea is opened complete haemostasis must be obtained. A suction tube with a catheter attached should be ready for aspiration of the trachea. At this stage sutures may be inserted into the skin edges in anticipation of closure of the lateral parts of the wound after the tube has been inserted. The trachea is retracted in an anterosuperior direction by inserting a tracheal hook below the cricoid cartilage. A transverse incision is made into the inter cartilaginous membrane below the second or third ring and then converted into a circular opening by holding the upper and lower margins in turn with strong forceps and removing the cartilage with a knife. Alternatively a ring punch can be used. The first tracheal ring must on no account be disturbed.

The type of tracheostomy tube which will be required in the immediate postoperative period should be selected. A soft cuffed tube will be needed if anaesthesia is to be continued and positive pressure ventilation is required, or if the accumulation of secretions in the trachea from laryngeal spill over is to be prevented. If the operation is for simple airway obstruction, a silver

tracheostomy tube or a softer synthetic tube can be used. The later tubes are provided with an obturator to help insertion through the opening in the anterior tracheal wall. The obturator is then removed and replaced by the inner tube.

Tracheostomy tube position is retained by tapes passed around the neck and secured by a reef knot on one side of the neck. It is important that the patient's head is well flexed when the ties are knotted. Otherwise the ties may become slack when the patient sits up in bed with the head forward, resulting in the possible displacement. An antibiotic impregnated gauze is packed around the tube and the lateral margins of the wound loosely approximated with the skin sutures. There should be sufficient space remaining around the tube to minimize the danger of subcutaneous emphysema.

If there is a lack of experienced nursing care immediately after tracheostomy, a flap of trachea based inferiorly and sutured to the skin margin of the incision will retain an airway in the event of the tracheostomy tube being accidentally displaced. It also makes reintroduction of the tube easier but is more likelihood of tethering of the skin to the trachea during healing.

Post operative

Immediately after completion of tracheostomy arterial blood sample was drawn again from the arterial catheter strictly adhering to all the protocols. It was subjected to ABG analysis. The patient was shifted to post - operative ward where he received Oxygen through tracheostome, I.V. fluids and monitoring. As explained above arterial blood sample, where drawn 12 hours and 24 hours, after performance of tracheostomy and ABG analysis was done.

All the results were tabulated, expressed as mean ± SD and subjected to statistical analysis. The paired t test was used by the statistician to derive the t value. The P value was calculated. P value of <0.001 was taken to be statistically significant.

COMPLICATION OF TRACHEOSTOMY[43,49]

Intraoperative Complications (Immediate)

1. Venous bleeding from communicating vessels of the anterior jugular vein or thyroid vein or innominate vein.
2. Arterial bleeding from thyroid vessel, innominate artery or less frequently from carotid.
3. Cardiac arrest due to excessive adrenaline, increased pH, CO_2, wash out, increased K, respiratory alkalosis.
4. Injury to tracheal wall.
5. Injury to paratracheal structures – recurrent laryngeal nerve, oesophagus, cricoid, thyroid cartilage.
6. Air embolism
7. Obstruction of bronchi by blood or secretions.
8. Hypotension.

Early post operative complications

1. Apnoea due to carbon dioxide washout
2. Dislodgement or displacement of the tube
3. Surgical emphysema
4. Pneumo thorax, pneumo mediastinum, atelectasis.
5. Tube obstruction due to excessive scabs and crusts.
6. Infection, profuse bronchorrhoea pneumonia
7. Dysphagia, aerophagia.
8. Recurrent respiratory obstruction
9. Tracheitis sicca
10. Tracheo Oesophageal fistula
11. False passage, accidental decannulation
12. Tracheo – arterial fistula.

Late complications

1. Tracheal stenosis
2. Difficulty in decannulation
3. Tracheo – cutaneous fistula, scar
4. Keloid formation
5. Tracheal granulation

DANGERS OF HIGH TRACHEOSTOMY

1. Sub glottic stenosis
2. Damage to cricoid cartilage
3. Arytenoid fixation
4. Perichondritis

DANGERS OF LOW TRACHEOSTOMY

1. Injury to inferior thyroid vein
2. Injury to innominate vein or artery

POST OPERATIVE CARE

NURSING CARE

1. A well trained nurse and medical staff should take care of the patient in the first few days.
2. They should observe strict aseptic precautions, with mask, gloves, catheter, powerful suction apparatus and keep a note book and bell by the side of the patient.
3. X-Ray soft tissue neck and chest should be ordered to note the position of the tube and to observe complications, like surgical emphysema and pneumothorax.

FIXATION OF TRACHEOSTOMY TUBE

1. Tapes are tied with head in the neutral or flexed position with reef knot.

2. Tapes should not be too tight or too loose.

3. Better to stitch the collar of the tube to the skin initially and secure it with tape to the neck.

SUCTIONING

1. Frequent suction is necessary in the early periods because there is increase production of secretion and patient cannot cough out the secretions effectively.

2. Suctioning should be accomplished with sterile soft rubber catheter less than ½ of the internal diameter of the tube.

3. 'Y' tube or side opening in the catheter is necessary to allow insertion of catheter into the trachea without suction. Suction is applied only during withdrawal of the catheter. Prolonged or frequent suctioning should be avoided.

4. Suctioning for every 30 mts, for the first 48 hrs (varies from person to person) Duration of suction is less than 20 seconds. Atleast one suction per 4 hrs.

HUMIDIFICATION

1. To prevent drying and crusting of the secretions
2. The air bypasses the normal route and directly goes to the trachea which necessitates humidification.

METHODS OF HUMIDIFICATION

1. Wet gauze soaked in water kept over the tracheostomy (periodically droplet of distilled water or saline is added over the cloth).
2. Hot water bath humidifier
3. Nebuliser delivering cold droplets.
4. Heat and humidity exchanger.
5. Humidified air through the ‘ T ’ tube
6. Ultra sonic humidifier
7. Steam tent
8. Fan ventilator
9. 15 drops of normal saline instillation per minute or 5ml of normal saline per hour (all the secretions and the crusts are cleaned easily by this method).

TRACHEOSTOMY TUBE CHANGE

1. It is necessary to change the tube for the first 24 to 48 hours, by this time the track will be epithelialised and tracheal opening will be readily formed.

2. Inner tube should be cleaned and changed every 2-3 hours for the first 3 days and as needed thereafter. Generally outer tube is changed after a week.

3. If there is any difficulty the patient is shifted to the theatre, with the help of tracheal dilator, he tube should be reinserted again.

4. While changing the tube, the emergency set is to be kept ready to tackle any situation.

5. The patient's attender is trained to clean and change the tube.

CUFF CARE

1. First 12 hours not to deflate the cuff.

2. After 12 hours deflate the cuff 5 minutes for every hour till the patient is accustomed to the tracheostomy.

3. Cuff pressure maintained between 20-30 cm H_2O to prevent complications.

FEEDING

1. In the immediate post operative periods, intravenous fluids are given, thereafter slowly start oral feeding.

2. Dysphagia is due to

    - bulk of the tube
    - subglottic oedema
    - Inflated cuff pressing over the oesophagus

3. Patient should be encouraged to take feeds in the sitting posture.

PHYSIOTHERAPY (BREATHING EXERCISE)

1. This plays an important role in the management of tracheostomy especially in the initial stages.

2. This is important in the elderly, debilitated, weak patient who cannot cough out the secretions effectively. Antibiotic is started along with physiotherapy.

3. All patients always need post operative breathing exercise by a physiotherapist.

RESULTS AND OBSERVATION

This study was designed as a prospective cohort study involving patients with upper airway obstruction with stridor and requiring emergency tracheostomies. The impact of the disease process on the patients ABG values and effect of tracheostomy of ABG changes were documented and statistically analyzed. All values were expressed as mean ± SD values were analyzed using t value and p values were derived and statistical significance of data was interpreted.

DEMOGRAPHIC DATA

Table: 1

AGE DISTRIBUTION

Age group	Male	Female	Total	Percentage
30-39 yrs	3	0	3	10%
40-49yrs	2	1	3	10%
50-59 yrs	2	0	2	6.66%
60-69yrs	16	1	17	56.66%
70-79 yrs	5	0	5	16.66%

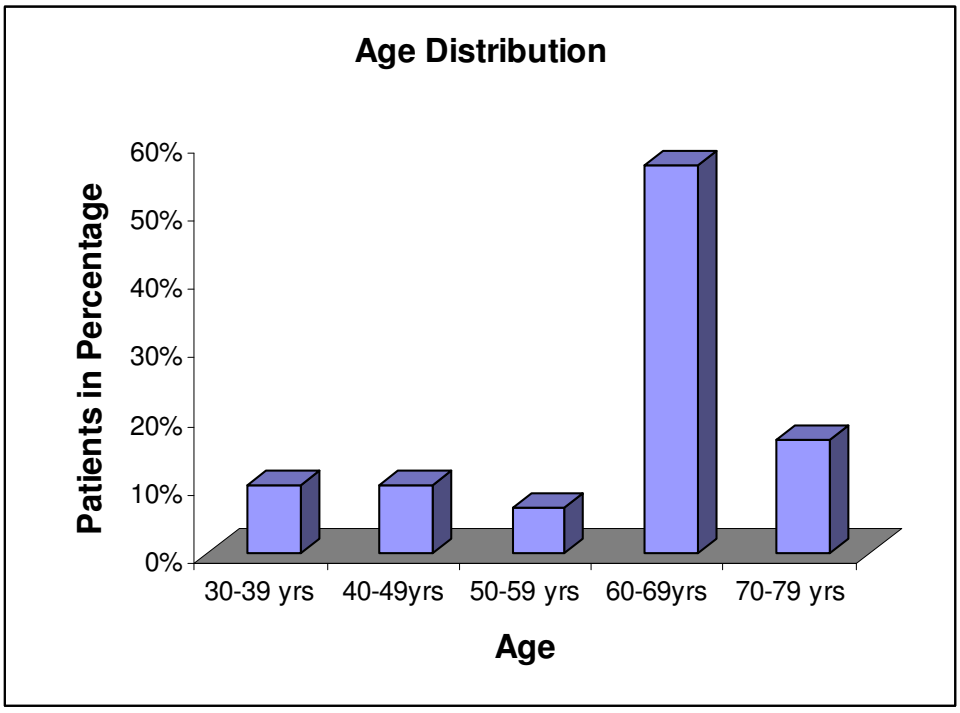
From the above data it is found that there is an increase predominance in the age group of 60-69 years (6^{th} Decade).

Table: 2

SEX DISTRIBUTION

Sex	No of Patients	Percentage
Male	28	93.33%
Female	2	6.66%

From the above data it is found that there was a male preponderance in this study and 93.33% of our patients were male.


Age Distribution
Patients in Percentage
60%
50%
40%
30%
20%
10%
0%
30-39 yrs
40-49yrs
50-59 yrs
60-69yrs
70-79 yrs
Age

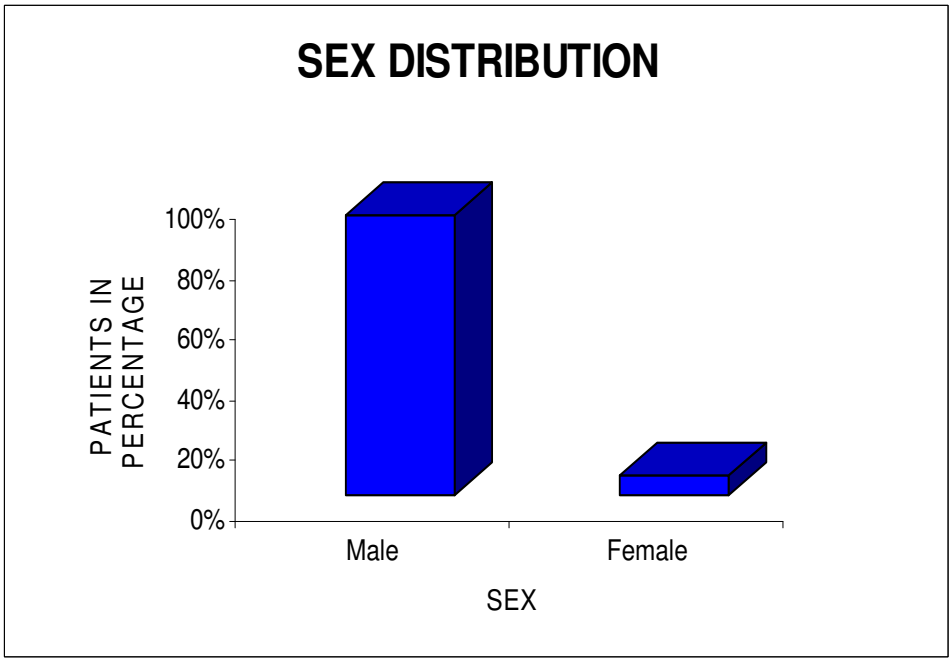

SEX DISTRIBUTION
PATIENTS IN PERCENTAGE
100%
80%
60%
40%
20%
0%
Male
Female
SEX

Table: 3

ETIOLOGY OF STRIDOR

S.No.	Causes	No. of cases	Percentage
1.	Glottic growth	7	23.3
2.	Supraglottic growth	5	16.66
3.	Subglottic growth	1	3.3
4.	Hypopharyngeal growth		
	i. Pyriform fossa growth	10	33.3
	ii. Post cricoid growth	4	13.3
	iii. Post pharyngeal growth	-	-
	Total	**14**	**46.6**
5.	Others i. bilateral abductor palsy	3	10

In the present study, commonest etiology factor causing stridor was malignant growth hypophaynx followed by glottic growth and supraglottic growth and bilateral abductor palsy.

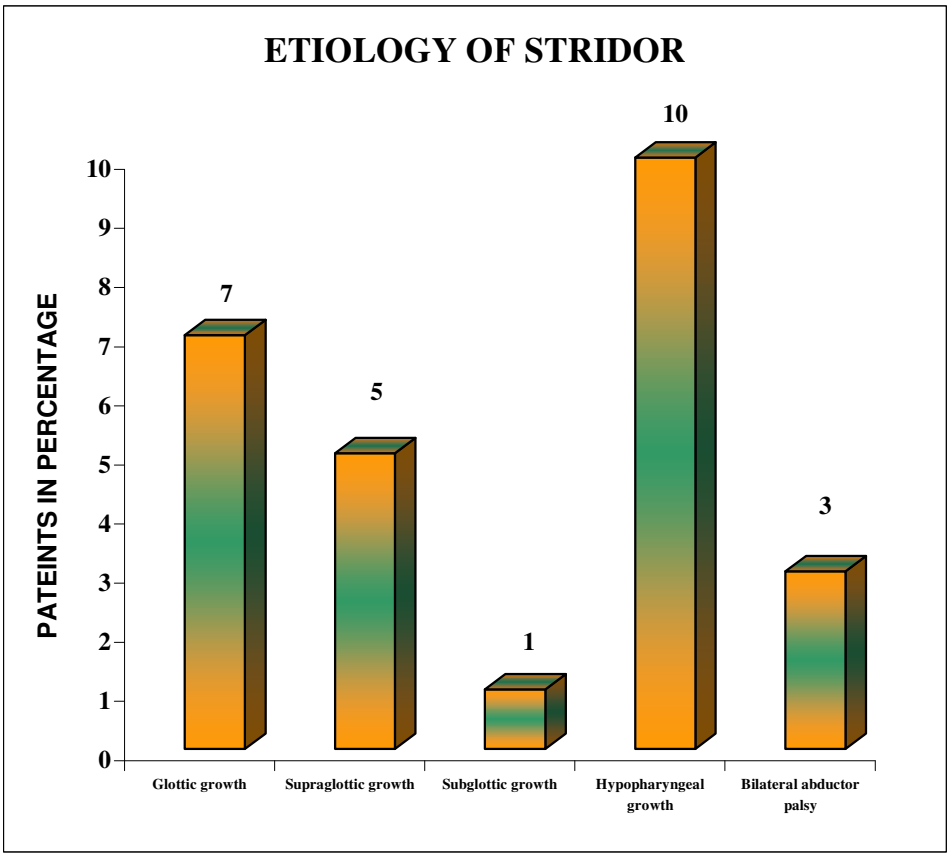

ETIOLOGY OF STRIDOR
PATEINTS IN PERCENTAGE
0
1
2
3
4
5
6
7
8
9
10
7
5
1
10
3
Glottic growth
Supraglottic growth
Subglottic growth
Hypopharyngeal growth
Bilateral abductor palsy

Table: 4

RISK FACTORS ANALYSIS

	No of patients	Percentage
Tobacco Abuse	25	83.33%
Alcohol	20	66.66 %
Both	19	63.33%
Pan Chewing	5	16.66%
Nothing Identified	4	13.33%

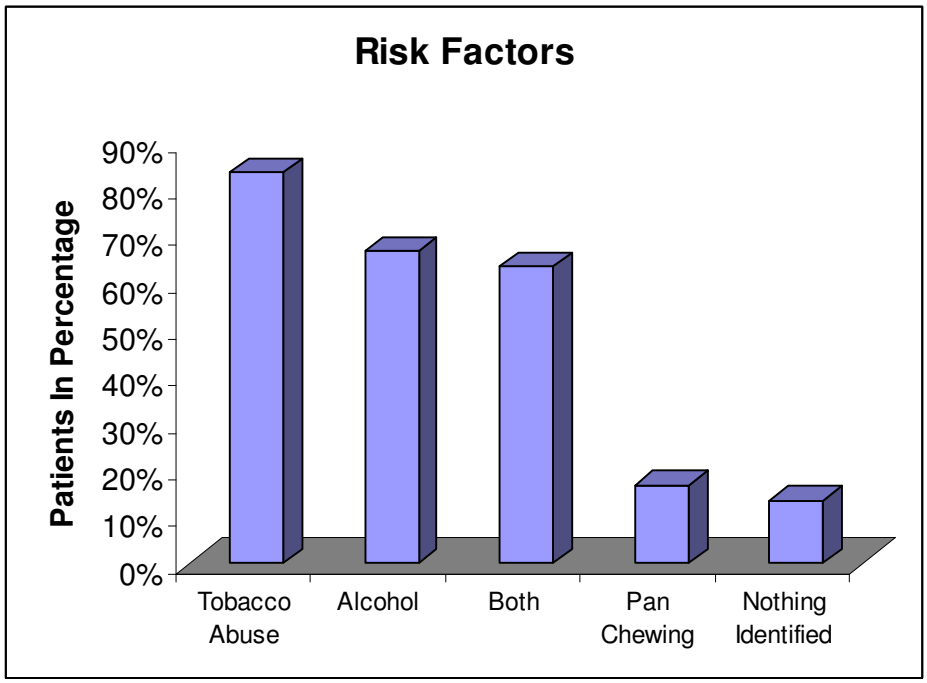
An analysis of the risk factors implicated in production of laryngeal growths in this study reveals that 83.33% of patients were Tobacco Abusers. Hence tobacco abusers were the commonest risk factor in this study group. The other risk factors detected in this study group were alcoholics 66.66%. both risk groups had a percentage of 63.33% followed by pan chewing of 16.66%. No risk factor was seen in 13.33%.

Table : 5

INCIDENCE OF TRACHEOSTOMY TUBE IN OUR PATIENTS

S. No	Tracheostomy tube	Cases	Percentage (Present Series)
1	Fuller's Biflanged metal tracheostomy tube	20	66.66%
2	Portex Cuffed tracheostomy tube	10	33.33%

Out of 30 cases of emergency tracheostomy, 20 cases (66.66%) were operated using Fuller's Biflanged Metal Tracheostomy tube and 10 cases (33.33%) with Portex cuffed tracheostomy tube due to availability of these tubes in our department of ENT.



INCIDENCE OF TRACHEOSTOMY TUBE

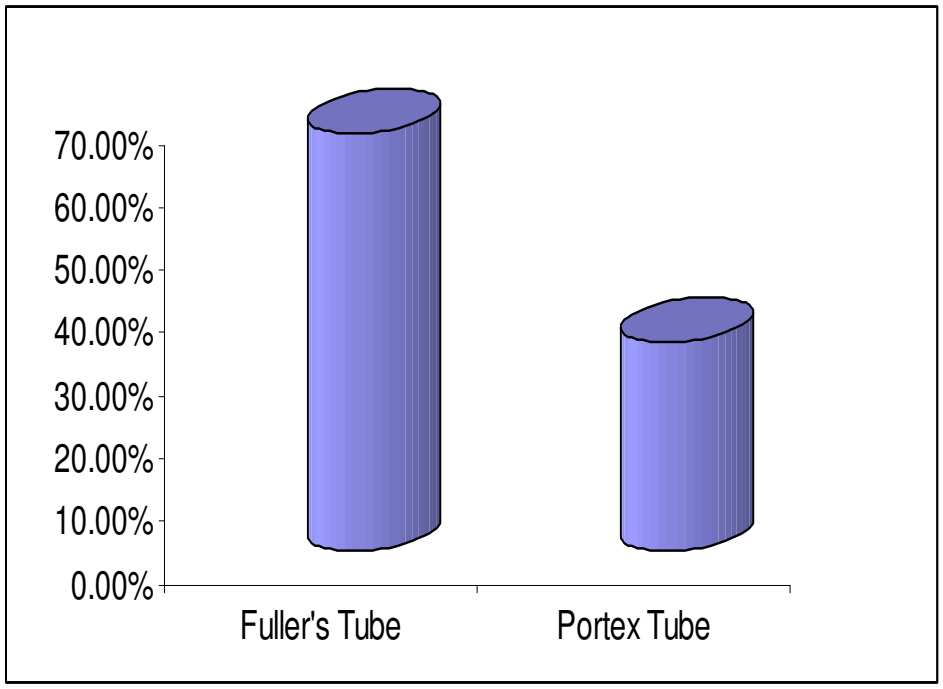


Table : 6

COMPLICATIONS IN OUR SERIES

S. No.	Nature	Complications	No. of Patients	Percentage
1.	Immediate	1. Bleeding	6	20%
		2. Cardiac Arrest		
		3.local Damage (Tracheal / paratracheal)		
		4. Air Embolism		
		5. Apnoea	3	10%
2.	Early	1. Tube Dislodgment	7	23.3%
		2. Surgical emphysema	6	10%
		3. Pneumothorax / Pneumomediastinum	-	-
		4. Scabs / Crusts	4	13.3%
		5.Infection	4	13.3%
		6. Dysphagia	6	20%
		7. Tracheo oesophageal fistula	-	-
		8. Tracheo inominate fistula	-	-
		9. Tracheitis Sicca	-	-
		10. Pharyngo cutaneous fistula	-	-
3.	Late	1.Tracheal Stenosis	-	-
		2. tracheo cutaneous fistula	-	-

The most common complications were scab and crust formation, bleeding, tube dislodgment and dysphagia. Emergency tracheostomy resulted in more acute complications usually than with elective tracheostomy. Surgical emphysema and apnoea was significant more with severity of stridor and more with patients suffering from acute respiratory obstruction due to laryngeal malignancy. Infection was attributed to poor hygiene of the patient.

Table : 7

ANALYSIS OF PRE-TRACHEOSTOMY ABG FOR PH

PH		Patients	Percentage
<7.30	Severe acidosis	8	26.6%
7.31 to 7.35	Mild acidosis	19	63.3%
7.36 to 7.45	Normal ABG	3	10%

Pre tracheostomy ABG shows 8 patients had severe respiratory acidosis. Majority of the patients (19/30) had mild uncompensated respiratory acidosis. 3 patients had normal PH. The commonest Acid-base disturbance in this study group was a acute primary, uncompensated Respiratory acidosis of mild to moderate severity.

Table : 8

ANALYSIS OF PRE TRACHEOSTOMY ABG FOR PO_2

PO_2	Patients	Percentage
Moderate hypoxaemia <80mm Hg	30	100%
Mild Hypoxaemia 80 - 90mm Hg	0	0
Normoxia >90mm Hg	0	0

Pretracheostomy ABG shows moderate to severe hypoxaemia in all the patients in this study. The study reveals that airway obstruction produces severe hypoxaemia, which can be life threatening if not treated urgently. Decreased PO_2 reveals a moderate to severe hypoxic hypoxaemia due to significant decrease in airflow to the lungs.

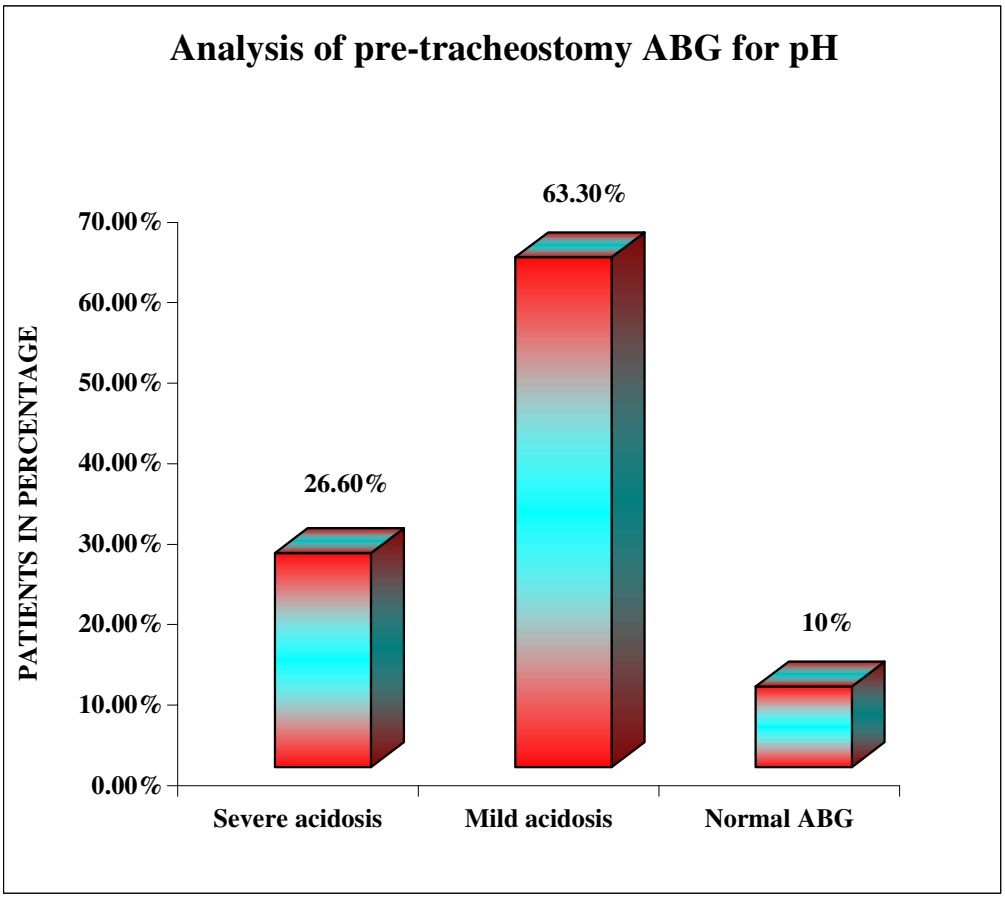

Analysis of pre-tracheostomy ABG for pH
PATIENTS IN PERCENTAGE
70.00%
60.00%
50.00%
40.00%
30.00%
20.00%
10.00%
0.00%
26.60%
63.30%
10%
Severe acidosis
Mild acidosis
Normal ABG

Table : 9

ANALYSIS OF PRE TRACHEOSTOMY ABG FOR PCO_2

PCO_2	Patients	Percentage
Hypocarbia <35mm Hg	0	0
Normocarbia 36-44mm Hg	0	0
Hypercarbia ≥45mm Hg	30	100%

Pre tracheostomy ABG shows hypercarbia in 100% of patients undergoing tracheostomy. This reveals a ventilatory failure which is a type II respiratory failure. This leads to alveolar hypoventilation and retention of Co_2 producing hypercarbia and acidosis.

Table: 10

ANALYSIS OF PRE – TRACHEOSTOMY ABG FOR HCO_3

HCO_3	Patients	Percentage
<18mmol/L	11	36.6
18-24mmol/L	19	63.3
>24 mmol/L	0	-

Analysis of bicarbonate values show that no patient had a metabolic disorder or mixed type of acid base disorder. The patient showing a normal serum bicarbonate, could reflect the initiation but not the completion of compensation. The normal compensation to primary respiratory acid is by the renal mechanism of increased conservation and production of HCO_3 ion.

Analysis of Pre Tracheostomy ABG For Hco_3

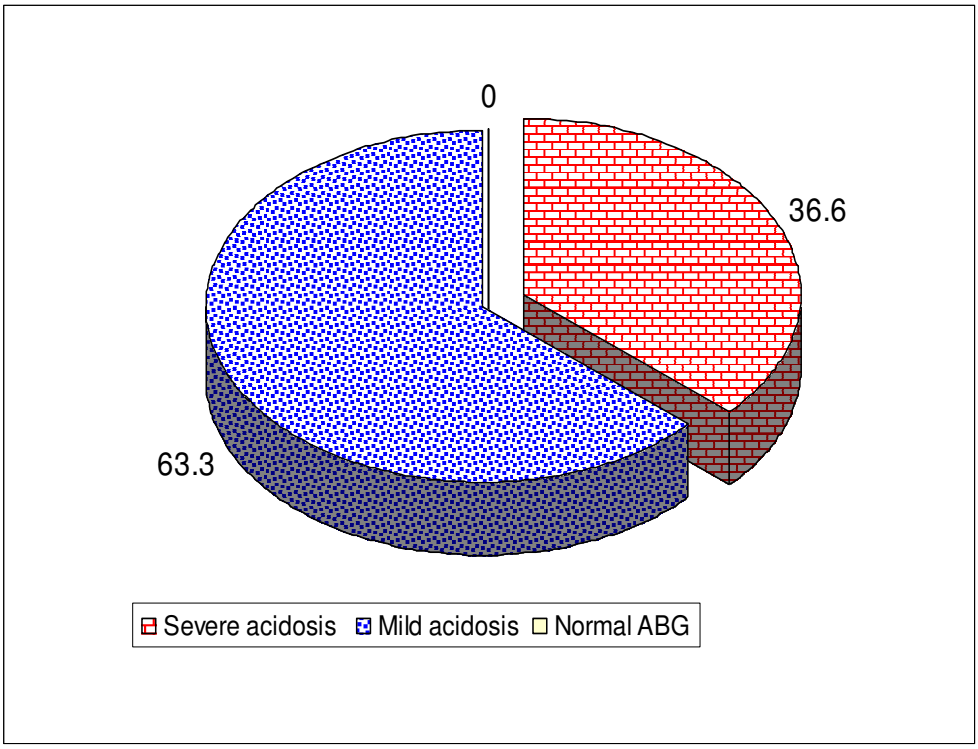


Table: 11
IMPACT OF TRACHEOSTOMY ON PH

	Severe acidosis <7.30	**Mild acidosis 7.31 to 7.35**	**Normal ABG 7.36 to 7.45**
Pre op	8 (26.6%)	19 (63.3%)	3(10%)
Immediate Post op	1(3.3%)	9(30%)	20(66.6%)
12 hrs Post op	0	0	30(100%)
24 hrs post op	0	0	30(100%)

PH	**Mean**	**S.D**	**T value**	**P value**	**Statistical significance**
PH preop	7.316	0.033	-	-	-
PH Immediate	7.370	0.031	12.55	<0.001	Significant
PH 12 Hours	7.394	0.023	13.83	<0.001	Significant
PH 24 Hours	7.407	0.021	15.91	<0.001	Significant

The PH values were found to improve with tracheostomy Immediately after tracheostomy patients with acidosis decreased drastically from 26.6% to 3.3%. Within 12 hours of tracheostomy all patients had normalization in their PH value and did not show any further deterioration. The difference between preop PH and postop PH changes was statistically significant in all the groups.

Impact of Tracheostomy on pH

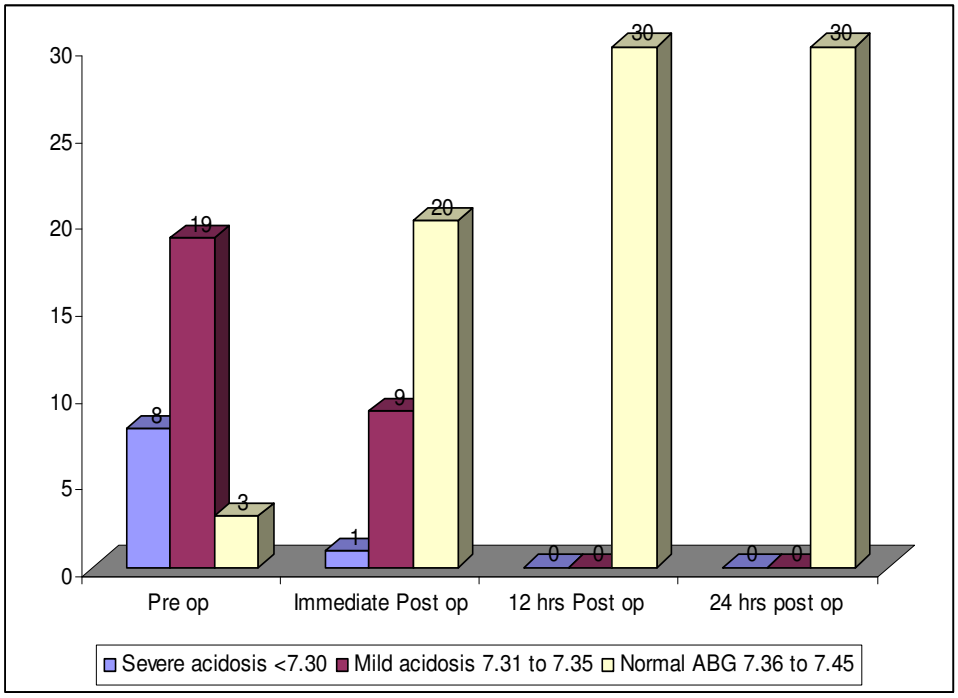


Table : 12

]IMPACT OF TRACHEOSTOMY ON PO_2

	Moderate Hypoxaemia <80mm Hg	**Mild Hypoxaemia <80-90mm Hg**	**Normoxia > 90mm Hg**
Pre op	30 (100 %)	0 (0%)	0 (0%)
Immediate	12(40%)	18 (60%)	0 (0%)
12 Hrs Postop	1 (3.3%)	10 (33.3%)	19 (633%)
24Hrs Postop	0(%)	3 (10%)	27(90%)

PO_2	**Mean**	**SD**	**T value**	**P value**	**Statically significance**
PO_2 Pre op	63.227	5.329	-	-	-
PO_2 Immediately	81.302	5.878	15.52	<0.001	Significant
PO_2 12Hrs	90.214	3.021	25.53	<0.001	Significant
PO_2 24Hrs	92.223	1.737	31.81	<0.001	Significant

Analysis of the PO_2 values reveal that patients with upper airway obstruction had moderate to severe Hypoxaemia. It incidence was 100%. Tracheostomy provided relief from Hypoxaemia in 19/30 (63.3%) of patients, Over a 12 hours period. Immediate but limited improvement occurred in 18/30 (60%) patients immediately after tracheostomy. 27/30 (90%) achieved normoxia within 24 hours after performance of tracheostomy.

Oxygenation improved after tracheostomy and the changes among the postop values over preop values were a statistically significant. The hypoxic hypoxaemia that occurs due to decreased airflow to the lungs due to the obstruction produced by the growth. Tracheostomy relieves this obstruction and normalizes oxygenation.

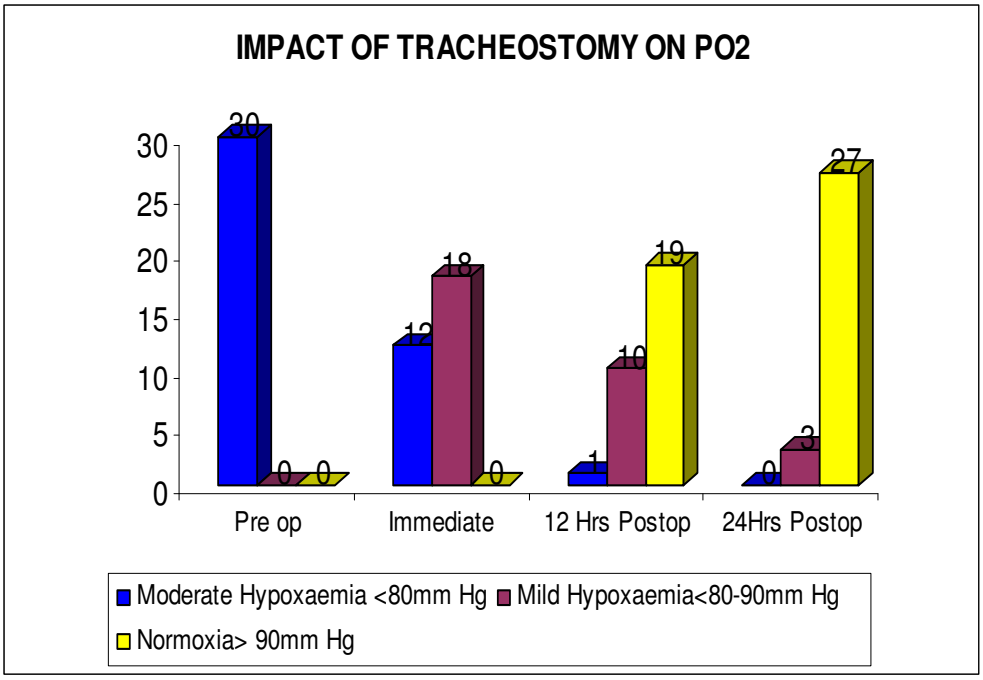

IMPACT OF TRACHEOSTOMY ON PO2
30
25
20
15
10
5
0
30
0
0
12
18
0
1
10
19
0
3
27
Pre op
Immediate
12 Hrs Postop
24Hrs Postop
Moderate Hypoxaemia <80mm Hg
Mild Hypoxaemia<80-90mm Hg
Normoxia> 90mm Hg

Table: 13

IMPACT OF TRACHEOSTOMY ON PCO_2

	Hypocarbia ≤ 35mm/Hg	**Normacarbia 36-44mm/Hg**	**Hypercarbia ≥ 45mm/Hg**
Pro op	0 (0%)	0 (0%)	30 (100%)
Immediate	0 (0%)	7 (23.3%)	23(76.6%)
12hrspost op	1 (3.3%)	22 (73.3%)	7 (23.3%)
24hrspost op	1 (3.3%)	28(93.3%)	1 (3.3%)

	Mean	**SD**	**t value**	**p value**	**Statistical significance**
PCO_2 pre	58.080	4.918	-	-	-
PCO_2 3mm	45.770	4.468	20.59	<0.001	Significant
PCO_2 12hrs	42.033	2.832	23.10	<0.001	Significant
PCO_2 24hrs	41.089	2.732	22.60	<0.001	Significant

Tracheostomy decreased PCO_2 values, by statistically significant, margins. This showed that upper, airway growths with stridor produced alveolar hypoventilation with retention of CO_2 and significant hypercarbia in all patients. Following tracheostomy CO_2 values normalized over a period of 12 to 24 hours is majority of the patients 73.3% patients reached normocarbia within 12 hours and 93.3% patients required 24 hours for normalization of CO_2 values.

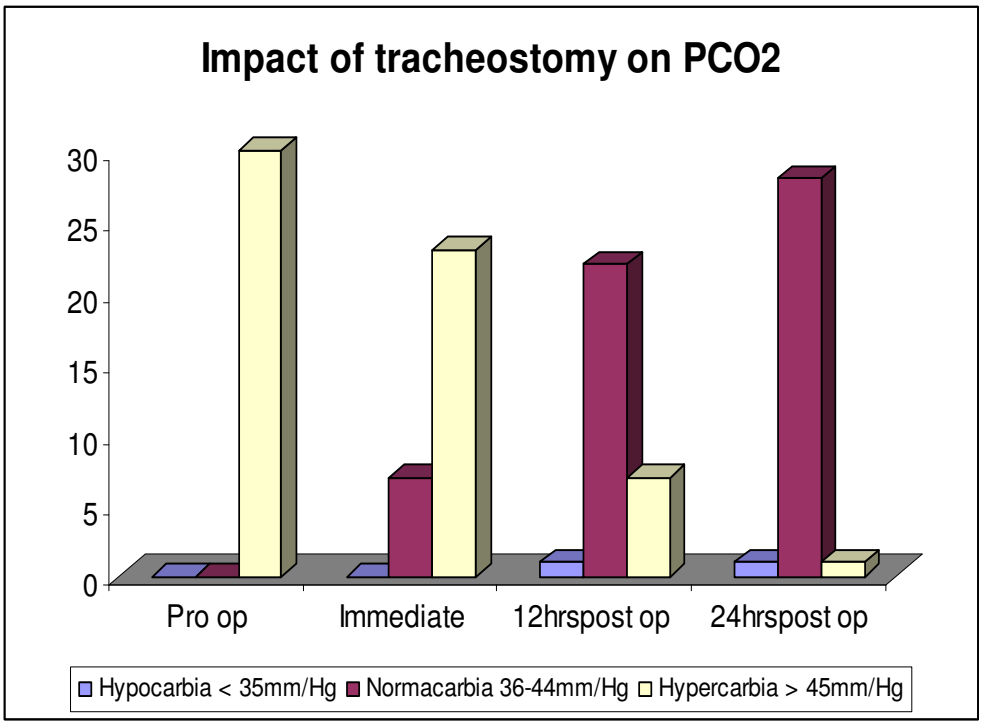

Impact of tracheostomy on PCO2
30
25
20
15
10
5
0
Pro op
Immediate
12hrspost op
24hrspost op
Hypocarbia < 35mm/Hg
Normacarbia 36-44mm/Hg
Hypercarbia > 45mm/Hg

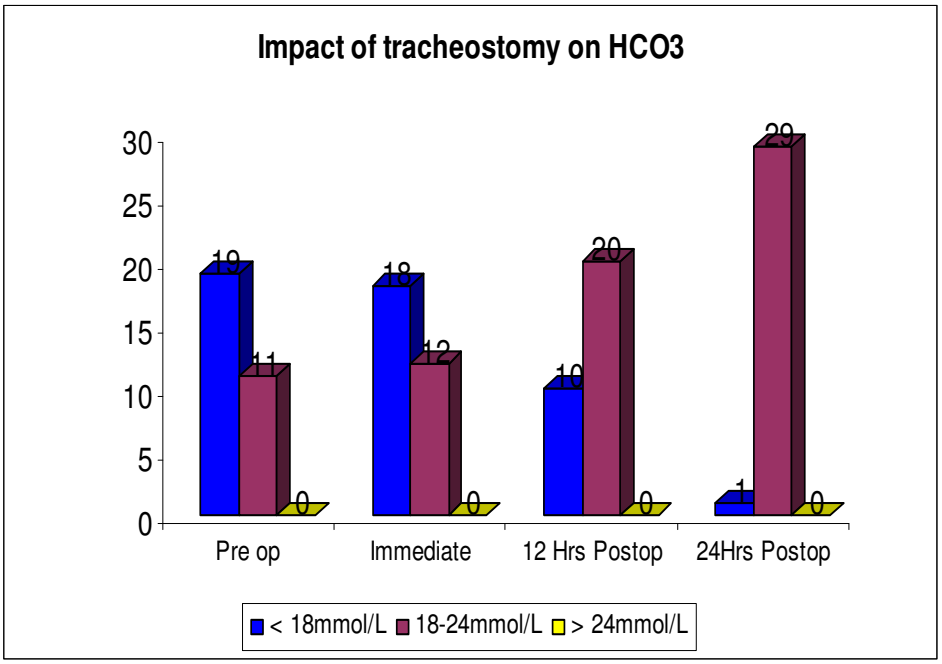
Table : 14

IMPACT OF TRACHEOSTOMY ON HCO_3

	< 18mmol/L	**18-24mmol/L**	**> 24mmol/L**
Pre op	19 (63.3%)	11 (36.6%)	0
Immediate	18 (60%)	12 (40%)	0
12hrs post op	10 (33.3%)	20 (66.6%)	0
24hrs post op	1 (3.3%)	29(96.3%)	0

	Mean	**SD**	**t value**	**P value**	**Statistical significant**
HCO_3	18.063	1.793	-	-	-
HCO_3 Imm	18.367	1.785	4.82	<0.001	Significant
HCO_3 12 hrs	18.970	1.580	7.10	<0.001	Significant
HCO_3 12 hrs	19.677	1.268	10.11	<0.001	Significant

Analysis of bicarbonate valves is critical for this study because it rules out acidosis due to metabolic disorders, where HCO_3^- values are grossly within normal range. It also rules out acid base disorders due to both respiratory and metabolic component that can co-exist in the patients. In most of the cases, the respiratory acidosis is acute and hence no compensation has occurred. Some cases showed initiation of compensation by renal conservation of HCO_3 but the pH still reflects the primary pathology. Tracheostomy produced statistically significant change amongst all the group.


Impact of tracheostomy on HCO3
30
25
20
15
10
5
0
19
11
0
18
12
0
10
20
0
1
29
0
Pre op
Immediate
12 Hrs Postop
24Hrs Postop
< 18mmol/L
18-24mmol/L
> 24mmol/L

DISCUSSION

In our study an attempt was made to analyse the arterial blood gas in tracheostomy patients Pre – operatively and post operatively in the following manner.

- Among the 30 cases studied (Table 3), 14 cases accounting to about 46.66% were hypopharyngeal growth followed by glottic growth which is present in 7 cases accounting to about 23.33%. Supraglottic growth were present in 5 cases and 3 cases of bilateral abductor palsy and 1 case of subgottic growth. Over all primary malignancy in this study was found to be more in hypopharyngeal growth accounting to about 46.66% which is in conformity with known statistical analysis (Meuden Hall; W. Head New Surgery 1980).

- 17 of 30 patients (Table 1) were between 60–69 years of age and this group constitutes 56.66% of the cases. Patient in the age group of 70–79 years were 5, 3 were present in both 30 – 39 years and 40 – 49 years age group and 2 were between 50 – 59 years. The observations is significant in that it shows and similar occurrence of malignancy when compared to western statistics showing. 6^{th} decade.(Althan and Cadman 1986). The mean age was lower than that reported in western studies such as Crofts et al[29]., Friedman et al[33]., because of lower life expectancy than western countries. Berrouschut et al reported from his study, the average was 58 + 13 (25 – 85 years). Keygu M.F. et al., and Dass and Bose series showed laryngeal malignancy occurs most commonly in age group of 50 – 60 (50%). Vega M.F. et al., Robin et al., (1991) and Dass bag series showed similar observation.

- Comparing the males with the females (Table2) 28 out of the 30 were males and the remaining 2 were females. This sex ratio of 14:1 comparable with value 15 : 1 in literature and other studies. But in western countries this sex ratio has come down to 5:1 and is being attributed to be increase in smoking and alcohol abuse among females in India, smoking and alcohol abuse among women is still very less so that 14% sex ratio is obtained in his study. Hazard et al., reported from his study the male 13 patients and female 11 patients, mean age of 61±18. Crofts et al.,[29] reported the total number of patients 28, from that 19 males and 9 female patients of age in years of 59.4 ± 18.3.

- Tobacco abuse added with alcohol abuse (Table 4) is identifiable in 19 of the patients. Out of this 19, 5 are also Pan chewers. Apart from this category 5 had the habit of only tobacco abuse and one had the habit of only alcohol abuse. Family history of cancer indicating genetic predisposition, presence of other cancers, previous irradiation for else other disease, industrial pollution were also considered to be one of the risk factors which in our study is conspicuously absent. No risk factor has been identified in 4 of these patients.

- Patients present to our OP department often in very late stages. This is attributed to poor health education and prevalence of self medication in our people. Poverty and lack self care also adds to the late presentation. On the other hand tumour may be asymptomatic at early stages and becomes symptomatic only in late stage. This late presentation has led the patients to us with Stridor, where emergency tracheostomy is needed. In our study we included only cases of upper airway obstruction

who presented to us with stridor. In the study tracheostomy was done for all cases of laryngotracheal trauma which was in conformity with a study by Mehta (1987)[9] of PGIMER, Chandigarh.

Complications recorded in present series (Table 6)

- Haemorrhage (Primary) is common with sharp dissection than with blunt dissection and is more in emergency situations. The late complications of bleeding is mainly due to metal tube crusting, irritation with metal tubes and is in conformity with known practices. The common bleeding places are anterior jugular vein and thyroid veins.

- Subcutaneous emphysema and apnea has accounted for 3 cases i.e., 10% and the cause being emergency. Subcutaneous emphysema has been related to use of very small incision or use of very large incision and usage of tracheostomy tube of small size has also been a cause for it. Metal tracheostomy tube produce more subcutaneous emphysema than non metal tube. Apnea has been related to severity of stridor and overextension due to usage of sand bag during tracheostomy has also been a cause for apnea.

- Use of cuffed tracheostomy tube has characteristically produced important complications like obstruction due to crust formation. Another common complications is the displacement of tracheostomy tube and this is due to neck extension being maintained during tracheostomy and also due to shorter length of the tracheostomy tubes in patient with large fatty neck.

- In our study (Table 5) we have used only Fuller's biflanged metal tracheostomy tube and portex cuffed tracheostomy tube, due to availability of these tubes in our Department of ENT. We have observed the usage of Fuller's metal tracheostomy tube, the incidence of subcutaneous emphysema and anterior displacement of tracheostomy tube is more common but it is very easy to maintain hygiene, thereby preventing crust formation.

- Friedman et al[33], have reported an incidence of 15% of mild bleeding and pneumothorax 4%.

- Crofts et al[29], they reported the intra operative and post operative complications include minor and major bleeding of 11%, pneumothorax of 4%, subcutaneous emphysema of 4%, atelectasis of 4% and cuff leak of 7%.

Incision and Anaesthesia

- In all our cases of emergency tracheostomy, we used vertical incision.

- All cases were operated in local anaesthesia

    1. Since the patient is already in respiratory distress and during induction of general anaesthesia, there is a possibility of respiratory muscle paralysis.

    2. To prevent tumour embolus during intubation.

Pre and Post tracheostomy Assessment of pH (Table 7 & 11)

Pre tracheostomy ABG shows 8 patients had severe respiratory acidosis. Majority of the patients (19/30) had mild uncompensated respiratory acidosis. 3 patients had normal PH. The commonest Acid-base disturbance in this study group was a acute primary, uncompensated Respiratory acidosis of mild to moderate severity. The PH values were found to improve with tracheostomy Immediately after tracheostomy patients with acidosis decreased drastically from 26.6% to 3.3%. Within 12 hours of tracheostomy all patients had normalization in their PH value and did not show any further deterioration. The difference between preop PH and postop PH changes was statistically significant in all the groups.

Pre and Post tracheostomy assessment of PO_2 (Table 8 & 12)

Pretracheostomy ABG shows moderate to severe hypoxaemia in all the patients in this study. The study reveals that airway obstruction produces severe hypoxaemia, which can be life threatening if not treated urgently. Decreased PO_2 reveals a moderate to severe hypoxic hypoxaemia due to significant decrease in airflow to the lungs.

Analysis of the PO2 values reveal that patients with upper airway obstruction had moderate to severe Hypoxaemia. It incidence was 100%. Tracheostomy provided relief from Hypoxaemia in 19/30 (63.3%) of patients. Over a12 hours period Immediate but limited improvement occurred in 18/30 (60%) immediately after tracheostomy. 27/30 (90%) achieved normoxia within 24 hours after performance of tracheostomy.

Oxygenation improved after tracheostomy and the changes among the postop values over preop values were a statistically significant. The hypoxic hypoxaemia that occurs due to decreased airflow to the lungs due to the obstruction produced by the growth. Tracheostomy relieves this obstruction and normalizes oxygenation.

Pre and Post tracheostomy assessment of PCO_2 (Table 9 & 13)

Pre tracheostomy ABG shows hypercarbia in 100% of patients undergoing tracheostomy. This reveals a ventilatory failure which is a type II respiratory failure. This leads to alveolar hypoventilation and retention of Co_2 producing hypercarbia and acidosis.

Tracheostomy decreased PCO_2 values, by statistically significant, margins. This showed that upper airway growths with stridor produced alveolar hypoventilation with retention of CO_2 and significant hypercarbia in all patients. Following tracheostomy CO_2 values normalized over a period of 12 to 24 hours in majority of the patients. 73.3% reached normocarbia within 12 hours, and 93.3% required 24 hours for normalization of CO_2 values.

Pre and Post tracheostomy assessment of HCO_3 (Table 10 & 14)

Almost 43 cases were studied and 30 cases were selected according to Millers ABG criteria and analysis of bicarbonate values show that no patient had a metabolic disorder or mixed type of acid base disorder. The patient showing a normal serum bicarbonate, could reflect the initiation but not the completion of compensation. The normal compensation to primary respiratory acid is by the renal mechanism of increased conservation and production of HCO_3 ion.

Analysis of bicarbonate valves is critical for this study because it rules out acidosis due to metabolic disorders, where HCO_3^- values are grossly within normal range. It also rules out acid base disorders due to both respiratory and metabolic component that can co-exist in the patient., in most of the cases, the respiratory acidosis is acute and hence no compensation has occurred. Some cases showed initiation of compensation by renal conservation of HCO_3 but the pH still reflects the primary pathology. Tracheostomy produced statistically significant change amongst all the group.

SUMMARY

1. The commonest cause of upper airway obstruction with stridor is hypopharyngeal growth.

2. It is common in males and the commonest associated risk factor is tobacco abuse.

3. The commonest acid-base disturbance in these patients is acute primary uncompensated respiratory acidosis of mild to moderate severity.

4. These patients also have arterial hypoxic hypoxemia and hypercarbia reflecting alveolar hypoventilation.

5. Tracheostomy is the treatment of choice for urgent intervention in this life threatening emergency.

6. Tracheostomy should be performed under L.A. with monitored anesthesia care and i.V. Sedation.

7. Tracheostomy with supplemental O_2 following it, produce normalization of pH and PO_2 starting immediately after procedure and is usually completed within 12 hours.

8. Improvements in PCO_2 and HCO_3 also start immediately following tracheostomy and 12 to 24 hours are required for normalization of values.

9. Serial arterial blood samples and ABG analysis is a very effective diagnostic tool to predict the morbidity and mortality of patients with upper airway obstruction.

10. Serial ABG values can help in monitoring the efficacy at the surgical intervention for the airway obstruction problem.

11. The commonest complications related to tracheostomy under L.A. were Bleeding and tube dislodgment.

CONCLUSION

- Patients with upper airway obstructions commonly have acute primary uncompensated respiratory acidosis.
- Tracheostomy under L.A. rapidly improved the acid base and ventilatory status by relieving the obstruction as evidenced by statistically significant improvement in ABG value.
- ABG is very useful diagnostic tool in upper airway obstruction.

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PROFORMA

Name :

Age / Sex :

Diagnosis :

Features

Stridor
Tracheal tug
Accessory muscles
Intercostal indrawing
Paradoxical movement

Vital signs

PR :
BP :
SaO2 :
Temp :
Weight :

ENT Examination

Glottic Growth
Supraglottic Growth
Subglottic Growth
Hypopharyngeal Growth
i. Pyriform fossa Growth
ii. Post Cricoid Growth
iii. Posterior Pharyngeal Wall Growth
Others
i. Bilateral Abductor Palsy

Risk factors

Tobacco Abusers:
Alcoholics:
Both:
Pan Chewers:
None:

Investigations

Hb
PCV
BT
CT
Urine — Alb / Sugar
Blood Urea
Serum Creatinine
Serum Electrolytes
ECG
X– Ray Chest PA View
X–Ray soft tissue neck AP/LAT view
Barium Swallow
CT neck

Arterial Cannulation

- Artery cannulated
- Site
- Cannula size
- No. of attempts
- Complications
- Failure
- Hematoma
- Venous Sample

Surgical Procedure

Anaesthesia –

Surgery

Type of Tube : Portex Cuffed Tube / Fullers Tracheostomy Tube

A.B.G Analysis

	pH	**PO_2**	**PCo_2**	**HCo_3**	**Interpretation**
Pre – Op					
Immediate Post OP					
Post – OP 12 hrs					
Post – OP 24 hrs					

Post OP vital signs

	0 hours	**6 hours**	**12 hours**	**24 hours**
PR				
BP				
SaO_2				

MASTER CHART

S. No.	Name	Age / Sex	I.P.No.	Diagnosis	Tracheostomy Tube	Habits	Pre Tracheostomy Analysis of A.B.G.				Post - Tracheostomy Analysis of A.B.G.											
											Immediately				12 Hrs				24 hours			
							PH	PO2	PCo2	HCo3	PH	PO2	PCo2	HCo3	PH	PO2	PCo2	HCo3	PH	PO2	PCo2	HCo3
1	Vijayaraghavan	65/M	676896	Growth Larynx	Fullers Tube	S+A	7.31	65.9	61.02	16.6	7.34	72.6	52.2	16.6	7.43	79.4	40.4	17.2	7.43	92.6	36.9	18.4
2	Kathirvel	60/M	677050	Growth Larynx	Fullers Tube	S+A+P	7.34	74	47.5	18.7	7.38	86	36.4	19.2	7.42	88	35.6	19.6	7.44	96	35	19.4
3	Perumal	62/M	678722	Hypopharyngeal Growth (Pyriform Fossa)	Fullers Tube	S+A	7.3	62.6	60.08	16.3	7.39	85	41	16.5	7.41	89	38.6	17	7.42	90	37.5	18.8
4	Thulasidas	62/M	682299	Hypopharyngeal Growth (Pyriform Fossa)	Portex Tube	S+A	7.24	59.6	58.7	19.6	7.3	78	50.8	20.2	7.38	92	43.4	20.6	7.39	92.6	43	21
5	Muniyandi	65/M	685602	Hypopharyngeal Growth (Pyriform Fossa)	Fullers Tube	S+A	7.4	70.8	52.8	20.4	7.41	89.4	45.6	20.4	7.41	94	44.2	20.4	7.42	94.8	42.4	20.6
6	Velu	65/M	687012	Hypopharyngeal Growth (Pyriform Fossa)	Fullers Tube	S+A+P	7.32	63.6	59.08	16.6	7.38	84.8	46.4	16.8	7.39	89	41.6	17.4	7.41	90	40.08	18.8
7	Krishnaiah	65/M	688122	Growth Larynx	Fullers Tube	S	7.28	64.8	60.05	17.2	7.34	79.2	48.2	17	7.38	92.02	44.6	18.6	7.39	94	40.5	19
8	Giribalan	60/M	691904	Hypopharyngeal Growth (Pyriform Fossa)	Portex Tube	S+A	7.34	66.2	57.4	19.8	7.35	86.4	43.2	19.8	7.37	94.6	42.8	20.2	7.38	94.6	42.6	20.6
9	Subbaiah	70/M	695614	Hypopharyngeal Growth (Pyriform Fossa)	Fullers Tube	S+A	7.32	66.7	64.5	16	7.38	74.4	45.2	16.4	7.44	90.5	38.6	17.6	7.44	93.5	37.5	18.8
10	Navaneethan	75/M	697802	Bilateral Abductor Palsy	Fullers Tube	S+A	7.31	57.6	62.3	16.6	7.34	82.1	53.2	16.9	7.36	90.6	45.4	17.2	7.36	91	45	18.4
11	Munuswamy	35/M	698954	Growth Larynx	Fullers Tube	S	7.27	73.2	60.86	16.4	7.32	89.9	46.5	16.8	7.38	95.2	40.4	17.3	7.39	95	40	19
12	Vasu	55/M	700279	Growth Larynx		S+A	7.41	60.4	56.5	20.1	7.41	85.4	48	20.6	7.41	89.6	44.3	20.8	7.41	93	43.4	21
13	Rani	40/F	708749	Hypopharyngeal Growth (Post Cricoid)	Portex Tube	-	7.32	62.1	57	18.6	7.37	82	46	19.4	7.38	91.2	43.6	19.6	7.39	92.4	43.6	19.8
14	Rajammal	60/F	719203	Hypopharyngeal Growth (Post Cricoid)	Portex Tube	-	7.32	59	61	17	7.38	81.6	52	17.6	7.39	92	46.5	19.2	7.41	93.2	42	19.4
15	Thangavelu	75/M	735868	Hypopharyngeal Growth (Pyriform Fossa)	Portex Tube	S+A	7.29	61.7	58.6	17.2	7.35	73	45	17.2	7.36	88.4	43.4	18.4	7.39	90.6	41.6	19

MASTER CHART

S. No.	Name	Age / Sex	I.P.No.	Diagnosis	Tracheostomy Tube	Habits	Pre Tracheostomy Analysis of A.B.G.				Post - Tracheostomy Analysis of A.B.G.											
											Immediately				12 Hrs				24 hours			
							PH	PO2	PCo2	HCo3	PH	PO2	PCo2	HCo3	PH	PO2	PCo2	HCo3	PH	PO2	PCo2	HCo3
16	Velu	31/M	736110	Bilateral Abductor Palsy	Portex Tube	S	7.31	58.7	62.1	16.8	7.36	85	47.8	17.6	7.41	90.6	43	18.8	7.42	92.4	41	19.4
17	Natesan	55/M	749277	Growth Larynx	Fullers Tube	S+A+P	7.34	71.6	49.8	21	7.41	84.6	38.6	21.2	7.42	91.4	38	21.4	7.44	92	36.8	21.8
18	Natarajan	62/M	751291	Supraglotic Growth	Fullers Tube	S+A	7.3	63.2	59.7	17.4	7.36	74	46	18	7.38	86.8	42	19.6	7.41	93.6	42	20
19	Durai Raj	60/M	751736	Growth Larynx	Fullers Tube	S+A	7.31	54.5	61.6	16.7	7.38	88	47.2	17	7.39	91	43.6	17.4	7.39	89.6	43.2	18.8
20	Devaraj	60/M	752851	Growth Larynx	Portex Tube	S+A	7.31	57.9	63	17	7.41	84.2	45	17.4	7.41	91.4	42.8	18.4	7.41	92	42.2	19.4
21	Venkataraman	47/M	753023	Hypopharyngeal Growth (Post Cricoid)	Fullers Tube	-	7.31	68.7	48.6	22	7.38	79	39.8	22	7.42	87.4	37.4	22	7.43	91.4	37	22.2
22	Ganesan	60/M	755924	Supraglotic Growth	Fullers Tube	S	7.31	57.1	60.7	16.8	7.35	68.4	47.2	17.4	7.37	85.6	43.4	18.4	7.41	91.8	43	19
23	Shyam Prasad	31/M	757330	Sub Glottic Growth	Fullers Tube	-	7.32	60.2	57.2	16.7	7.41	83	45	17	7.41	90.6	43	17.2	7.41	93.2	42	18.1
24	Kumar	79/M	760494	Supraglotic Growth	Fullers Tube	S+A+P	7.36	72.6	49	20.8	7.41	85.5	39	21	7.42	90.7	38.2	21.2	7.44	89.6	38.4	21.6
25	Natarajan	64/M	773037	Hypopharyngeal Growth (Pyriform Fossa)	Fullers Tube	S+A	7.31	61.1	59	17.2	7.38	78.2	44	17.4	7.38	90.2	41	19.2	7.38	90.6	43.8	20.2
26	Arul	45/M	777692	Hypopharyngeal Growth (Pyriform Fossa)	Portex Tube	S+A	7.31	59.8	63	16.4	7.37	77	53.6	16.4	7.37	91.8	45.6	16.6	7.39	91	44.2	17.2
27	Narayanan	60/M	777934	Hypopharyngeal Growth (Pyriform Fossa)	Fullers Tube	A	7.33	65.4	48	21.8	7.41	87	38.8	22	7.42	93.4	37.4	22	7.42	89	37.8	22.4
28	Jagadesan	70/M	778977	Supraglotic Growth	Portex Tube	S	7.29	56.9	62.6	17.6	7.32	69.6	51.4	18.8	7.36	88.2	44.8	19.4	7.39	92.4	44	20.4
29	Vellaiah	65/M	782297	Supraglotic Growth	Fullers Tube	S+A+P	7.32	58.4	61.5	17.8	7.36	78.6	45.2	17.2	7.39	89.4	43.6	17.4	7.41	92.8	43	18.6
30	Selvam	60/M	782792	Hypopharyngeal Growth (Pyriform Fossa)	Portex Tube	S	7.28	62.6	59.2	18.8	7.34	87.2	44.8	19.2	7.36	92.4	43.8	19	7.39	92	43.2	19.2