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CONTENTS

EDITORIAL

ANAESTHESIA AND SAFETY

● Dr. Meenu Chadha

''When you make a 'mistake', don't look back at it long. Take the reason of the thing into your mind and then look forward. 'Mistakes' are lessons of wisdom. The past cannot be changed. The future is yet in your power.''

Hugh White

We work in an environment where unreliable delivery of the best care can result in fatal adverse events[1] Anaesthetists have an important role to play in the patient safety. The safety is effectively implemented by the help of guidelines and emerging evidence on safe practice.

The Safe Anaesthesia project (SAFE) was launched in 2010. This is a training initiative of the AAGBI (Association of Anaesthetists of Great Britain and Ireland) and WFSA (World Federation of Societies of Anaesthesiologists). This was started with the main aim of delivering vigilant and competent anaesthesia by the anaesthesia providers.

Year 2015 was a landmark for surgery and anaesthesia. The 68th World Health Assembly (WHA) unanimously passed a resolution on strengthening emergency and essential surgical care and anaesthesia as a part of universal health coverage. The Lancet Commission on Global Surgery (LCoGS) came out with a report “Global Surgery 2030: Evidence and Solutions for achieving Health, Welfare and Economic development “which contained five key messages relating to access to safe and affordable surgery and anaesthesia.

In the present scenario there is a wide discrepancy in safe anaesthesia practice in low and middle income countries (LMICs) and that in high income states. Practical and achievable safety standards should be followed. Use of high quality medications and post-operative pain management should also be taken into consideration.

Anaesthesia has come a long way since 1949 when Robert Macintosh challenged the view that deaths under anesthesia were inevitable and was usually due to anesthetist's failure[2]. Many changes were bought about to reduce human error and the greatest milestone was the foundation of APSF (Anaesthesia patient safety foundation)[3] in 1985. It is now recognized that accidents are inevitable in systems such as healthcare, because of complexity and the latent factors that set up humans to fail[4].

Advances in technology has also played a great role in the safety of anesthesia e.g. pin indexing of cylinders, pulse oximetery and capnography. The one error which was resistant to improvement was medication error. The APSF is addressing medication errors, based on standardization, technology, greater use of pharmacists and culture change[5]. It is mandatory to check all medications[6] before administration and a randomized trial has shown that anaesthetists confirming with key principle of safe drug administration[7] (including checking, using barcodes) resulted in fewer errors. Use of WHO surgical safety check list has also resulted in reduction of errors[8].

Thus we can conclude to state that anesthesia has low specific risk, but has an

important impact on perioperative risks and outcomes. Adverse events in perioperative period are present but are preventable in more than 50% cases. Future research needs to look into more high quality evidence about the effectiveness of patient safety practices, deeper insights into common patterns of preventable events and into implementation issues of surgical checklists and other practices.

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ANAESTHESIA FOR THORACOSCOPIC PROCEDURES

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ABSTRACT

Thoracic anaesthesia has changed the outcome of thoracic surgery. However, it poses significant challenges to the anaesthesiologist. Anaesthesia for video assisted thoracoscopy is based on one lung ventilation(OLV). OLV physiology and its adaptation, the hypoxic pulmonary vasoconstriction are altered by circulatory dynamics, anaesthesia and pulmonary infection. This may predispose the patient to hypoxia during thoracic surgery. Hence, preoperative evaluation should include risk stratification and plan of optimization.Lung isolation techniques are the mainstay for anaesthesia management. Protective ventilatory strategies and judicious fluid administration can reduce the risk of acute lung injury, reexpansion/reperfusion injury after OLV. This article is a review of best practices of management of ventilation, oxygenation and weaning to two lung ventilation

KEY WORDS

Thoracoscopy, Video assisted thoracoscopic surgery, one lung ventilation, hypoxic pulmonary vasoconstriction, double lumen endotracheal tube

INTRODUCTION

The advent of ”minimally invasive” surgeries though advantageous to the surgeon and the patient poses a considerable challenge to the anaesthesiologist. The surgeon and the anaesthesiologist share the same “thoracic space “for surgery and homeostasis. This review intends to familiarize the anaesthesiologist with the technical details of the procedure and the anaesthetic implications.

APPLICABILITY AND SCOPE

Since its inception and first description by Dr. Hans Christian Jacobaeus in 1903[1], minimally invasive thoracic surgery has come a long way.

In the early 1990’s video assisted thoracic surgery (VATS) came into existence with incorporation of video technology and progressed to complex procedures like lobectomy, pneumonectomy, oesophagectomy etc. It offered benefits of less post-operative pain[2], shorter ICU and hospital stay , decreased incidence of post-operative complications, quicker return to work, better cosmoses and decreased morbidity [3-7]. Lung volume reduction surgery is another area where VATS has proved beneficial, especially in patients with disabling emphysema despite medical management.

VATS also offers the advantage of early diagnosis of lung cancer (Stage I, II, IIIa). It gives access to lesions difficult to approach otherwise. It offers a less invasive approach to extensive examination of pleura, mediastinum and lung without rib spreading. According to the latest evidence based guidelines of the American College of physicians (ACCP), lobar resection by VATS has been one of the important advances in thoracic surgery.

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VATS however gives a two dimensional vision, lack of depth of perception and necessitates use of long rigid instruments. The introduction of da Vinci Surgical system with three dimensional vision and endo-wrist technology helped overcome these limitations. It also provided 3 D vision with magnification better than open surgery and precise dissection with improved ergonomics so that a wide plethora of procedures could be performed more effectively. Literature has revealed that robotic surgery for pulmonary resections is safe and efficient and has similar survival rates compared to open and VATS approaches. Robotic platform helps in achieving, with far more finesse lymph node dissection but has the disadvantage of cost, lack of consistent platform availability and lack of tactile feedback[8,9].

Table 1- Diagnostic & Therapeutic indications of thoracoscopy

Indications for diagnostic thoracoscopy

1. Pleural disease-thoracocentesis, tuberculosis
2. Staging-lung cancer, mesothelioma, oesophageal cancer
3. Parenchymal disease- interstitial fibrosis
4. Mediastinal tumors- metastatic lymphoma

Indications for therapeutic thoracoscopy

1. Pleural disease- pleurodesis, decortication, empymectomy
2. Parenchymal disease- wedge resection, lobectomy, pneumonectomy, blebectomy, lung volume reduction.
3. Pericardial disease- stripping, window
4. Mediastinal disease- thymectomy, chylothorax
5. Oesophageal surgery- vagotomy, Heller's myotomy, antireflux procedures
6. Sympathectomy-hyperhidrosis, reflex sympathetic dystrophy
7. Spinal surgery
8. Minimally invasive valve and coronary artery procedures

INDICATIONS

The scope of VATS extends through a spectrum of simple diagnostic to major therapeutic procedures and segmental lung resections.[10,11]. The indications for thoracoscopy are given in Table 1.

PATHOPHYSIOLOGY

The challenges of thoracoscopy are-lateral decubitus physiology, one lung ventilation, potential for massive hemorrhage and proximity of vital structures.

PHYSIOLOGIC EFFECTS OF LUNG ISOLATION –

During two lung ventilation, there is a ventilation perfusion (V/Q) mismatch in the anaesthetized, paralyzed and open chest patient who is in the lateral decubitus position. The main reason for the mismatch is because greater ventilation and less perfusion occur in the nondependent lung and vice versa (Fig 1). The poor ventilation is because of the suboptimal positioning, compression of the lungs by the mediastinum and abdominal viscera and decreased lung volume due to general anaesthesia. Muscle paralysis improves compliance of the open chest and ventilation of the dependent lung[12].

Fig 1 : Diagrammatic representation of the V/Q relationship in Patient with open chest in LDP

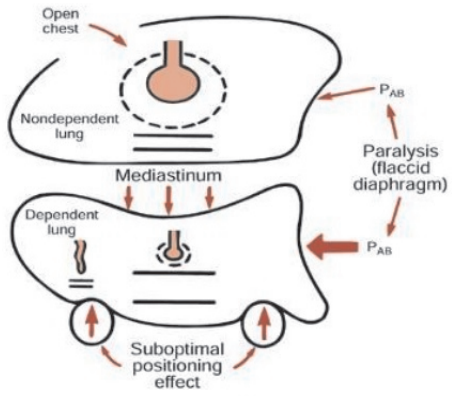
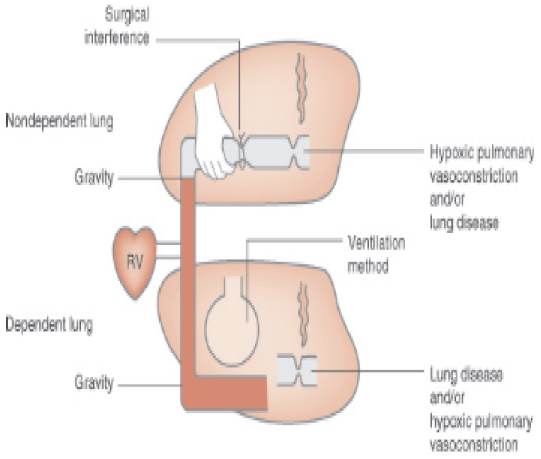


Fig 2 : V/Q Ratio in OLV



When one lung ventilation (OLV) is instituted, a shunt develops. The non-dependent lung is no longer being ventilated but is still being perfused, creating a right to left shunt. In lateral position, there is increase in blood flow to the non-dependent lung due to gravitational force. These effects are significant because the pulmonary system has a much lower blood pressure than the systemic circulation. (Fig 2)

Another adaptation which occurs is the hypoxic pulmonary vasoconstriction (HPV). HPV allows redirection of blood flow to the alveoli with higher oxygen tension thus reducing the V/Q mismatch. This is most beneficial when 30%-70% of the lung becomes hypoxic. The determinants of hypoxic pulmonary vasoconstriction are summarized in Table 2.

PREOPERATIVE EVALUATION AND PREPARATION

The purpose of pre-operative evaluation is not only to give fitness for elective surgery, but to evaluate and implement measures to prepare high risk patients for surgery.

It involves a thorough history and physical examination, laboratory and diagnostic examination and optimization of patient's condition[13]. Patients that require one lung ventilation (OLV) need a thorough assessment of their respiratory function, including mechanical function of the lung, pulmonary parenchymal function and cardiopulmonary reserve [14].

Table 2- Determinants of hypoxic pulmonary vasoconstriction

DECREASED BY

1. Drugs- volatile anaesthetics, GTN, SNP, Calcium channel agonists, PDE, a agonist, β agonists
2. High PAP-poor smooth muscle overcome by pressure
3. Low PAP- creates zone 1 in ventilated lung, non-ventilated lung already zone 1.
4. High PvO_2- reverse diffusion of O_2
5. Low Fio_2 / Low PvO_2- induces HPV in ventilated lung, some blood diverted to non-ventilated lung
6. Hypercapnia- cause pulmonary vasoconstriction in ventilated lung
7. Hypocapnia- causes pulmonary vasodilatation in non-ventilated lung
8. High Airway pressure- increases PVR in ventilated lung
9. Ventilated lung PEEP- diverts blood to non ventilated lung by increasing PVR

MAXIMISED BY

1.Normocapnea 2. Lower airway pressure 3. Normal PAP 4. Use of 50% FiO_2 initially

Assessment of cardiovascular risk:

Cardiovascular evaluation should be the first step in evaluation for lung cancer VATS, as the incidence of MACE (major adverse cardiac events) in lung cancer patients is 2-3 % (ACCP, European Respiratory society (ERS), British Thoracic Society (BTS) guidelines. Thoracic revised cardiac index (ThRCRI) is a valuable tool, based on four parameters. Table-3

TABLE 3- Thoracic Revised Cardiac Index

Pneumonectomy	1.5
Previous heart disease	1.5
Previous stroke or TIA	1.5
Creatinine>2mg/dl	1

Patients with more than 1.5 point, recent cardiac disease or limited exercise tolerance should undergo further cardiac evaluation.

Assessment of respiratory function:

Lung cancer surgical candidates should undergo both spirometry (FEV_1) and measurement of diffusing capacity for CO (DLco)[15-17]. FEV_1 is mainly to assesses air flow limitation while DLco describes function of the alveolar-capillary membrane.

Preoperative FEV_1 and DLco as well as calculated predicted postoperative (ppo) have been independently associated with morbidity and mortality[18-22].

Early postoperative FEV_1 is a better predictor of survival. Patients with preoperative FEV_1 and DLco>80% predicted or ppo FEV_1 and ppoDLco>60% predicted are considered low risk even for pneum-onectomy[15,17]. If both ppo FEV1 and ppoDLco are <60% predicted then it is necessary to evaluate patient's exercise capacity. This could be done with shuttle walk test, stair-climbing or with a cardiopulmonary exercise test (CPET). The latter is a maximal exercise test that assesses both respiratory and cardiac response to stress. Shuttle walk test and stair-climbing are strongly correlated with CPET[23,24]. Patients with an exercise tolerance of >400m on shuttle walk or 22 m on stair climbing, or ppo FEV_1 or ppoDLco 30-60% predicted are considered fit for lobectomy or segmentectomy. Nakahara et al found that patients with a ppoFEV1 greater than 40% had no or only minor post-resection respiratory complications.[25] Major respiratory complications were seen in patients with ppoFEV1<40% and post-operative ventilatory support was needed in those less than 30%.

Patients with VO_2 max >20 mL/kg/min or >75% pred. can undergo pneumonectomy[26,27]. On the contrary those with VO2 max <10-12 mL/kg/min or <35% predicted represents a high risk group and major anatomic resection is contraindicated[28,29]. An intermediate risk group is patients with VO_2 max=10-15 mL/kg/min[30,31]. In this case patient should be informed of the risk and alternative treatments.

ACCP guidelines utilize a stepwise approach for management of lung resection patients, including lung function measurements, low-cost exercise capacity tests and finally CPET.

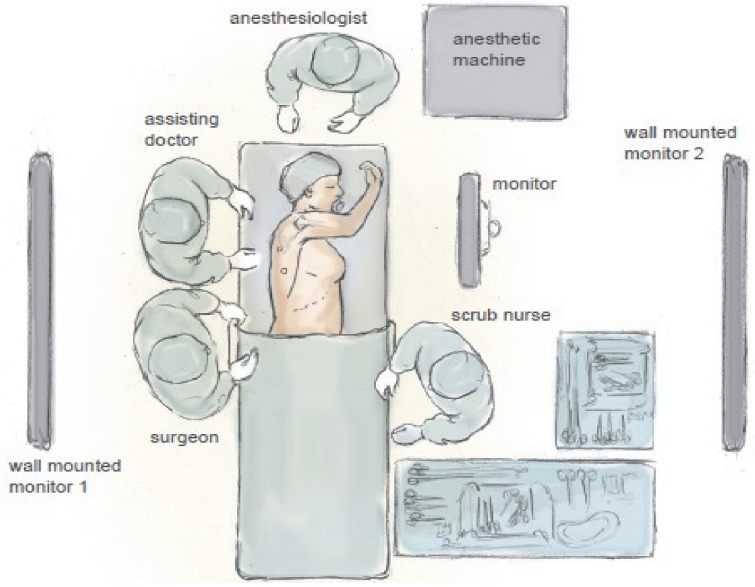
The ERS/ESTS guidelines recommend CPET for those with either FEV_1 or DLco<80% while ppo values are used later in the algorithm. BTS guidelines give a detailed approach for staging the disease but there are no proposed cut-off points for FEV[1] or DLco.

Other tests that can be done are arterial blood gases, minute volume ventilation (MVV), residual volume/total lung capacity (RV/TLC) and forced vital capacity (FVC). There are certain predictors which result in increased post-operative complications (Table4)

TABLE 4- Factors that decrease blood flow to ventilated lung

- High mean airway pressure in ventilated lung secondary to high PEEP.
- Low fraction of inspired oxygen (FiO_2)
- Vasonstrictor medications like dopamine, epinephrine and phenylephinephrine which causes increased vasoconstriction of normoxic vessels
- Intrinsic PEEP from short expiratory time
- High mean airway pressure in ventilated lung secondary to high PEEP.

Fig 3 : Ergonomics in Thoracoscopy



Laboratory tests before the surgery depend on the patient's age, associated co-morbidities and surgical risk, and include complete blood count, basic metabolic panel, renal, liver and coagulation functions and blood grouping and cross matching.

Imaging includes-

Chest x-ray- It is basic, readily available and inexpensive investigation to identify and locate a pulmonary nodule. Lesions as small as 5mm in diameter can be visualized by plain Chest X ray.

CT chest- Is the mainstay in chest imaging. It is very sensitive and specific fordetecting pulmonary nodules and provides accurate information on size, location and character of the nodule

FDG-PET/CT scan- Is a non-invasive functional imaging test that is used for tumor diagnosis, disease staging, and evaluation of treatment response.

Pre-operative bronchoscopy and mediastinoscopy may be needed in patients with bronchogenic carcinoma.

PREOPERATIVE OPTIMISATION

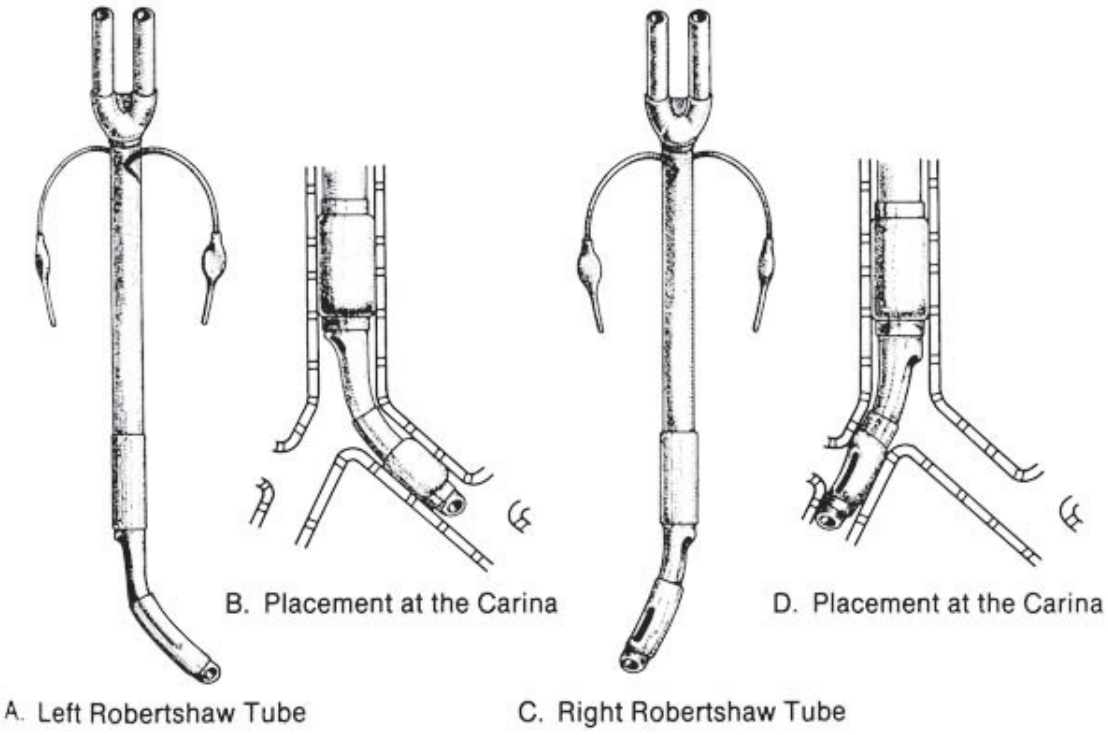
Pulmonary optimization includes

1. Cessation of smoking
2. Preoperative bronchodilators
3. Decreasing viscosity and mobilization of secretions
4. Adjunct therapy to decrease secretions (pharmacological and psychological)
5. Preoperative physiotherapy- preoperative pulmonary rehabilitation (incentive spirometry) has been shown to significantly improve exercise capacity, dyspnea and post-operative morbidity especially in patients for lung volume reduction surgery. Interventions based on moderate to intense aerobic exercises in patients undergoing lung resection, improve FRC and decreases post-operative morbidity[32]

ANAESTHETIC CONSIDERATIONS

VATS requires reorganization of the operating room set up and monitor position. The positions of the surgical team are well depicted in Fig 3.

Fig 4 : Double Lumen Tubes



VATS can be done under local, regional or general anaesthesia (GA).Local anesthesia can be given by infiltration of the thoracic wall or ipsilateral intercostal nerve block.at the level of incision and two spaces above and below. Thoracic epidural or paravertebral block can also be used. An ipsilateral stellate ganglion block can also be given to inhibit the cough reflex occurring as a result of manipulation of the hilum. Visceral pleura can be anaesthetized with topical local anaesthesia. Intravenous sedation can be achieved with intravenous fentanyl, midazolam or propofol.

When VATS is done under local anaesthesia, the patient is breathing spontaneously and partial collapse of the lung occurs when the incision is made and the chest cavity is open. Atelectasis may occur, but this may sometimes provide suboptimal surgical exposure. VATS under local anesthesia is safe for short procedures. Longer surgeries require general anesthesia[33]with one lung ventilation (OLV) for better exposure& a secure airway in lateral position.

LUNG ISOLATION DEVICES

There are three methods of lung isolation

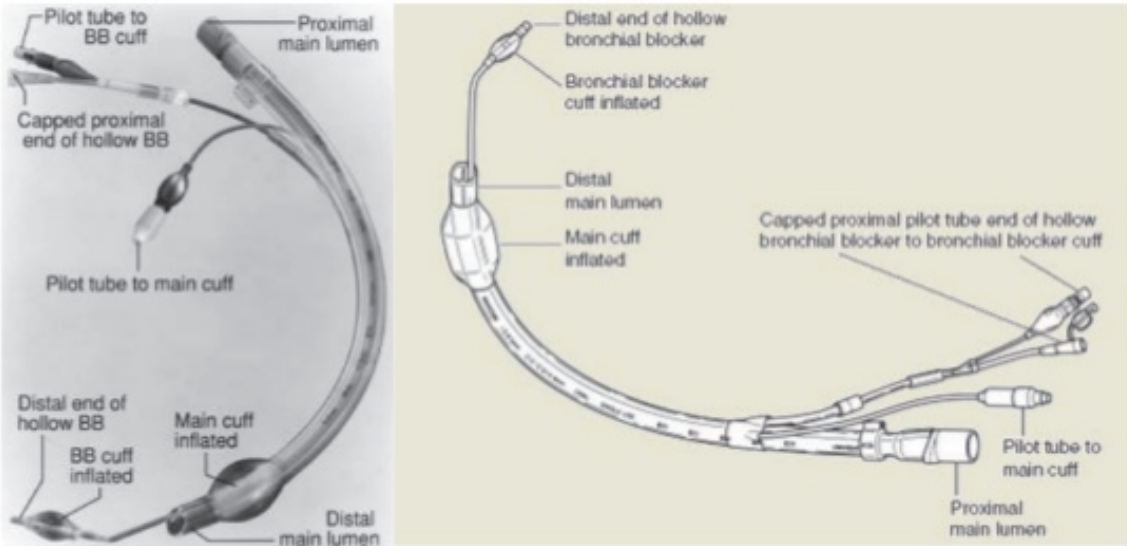
a) Double lumen tubes

b) Bronchial blockers

c) Main stem intubation

DOUBLE LUMEN TUBES- It is a bifurcated tube in different sizes, with two lumina, that can be used to achieve isolation of the right or left lung (Fig 4). It provides lung isolation, allows differential lung ventilation with minimal device manipulation. Most anesthesiologists prefer to use left sided DLT, though the right sided tube may be used for left bronchial resection or distorted anatomy. Practitioners refrain from using right-sided DLT simply to avoid potential occlusion of the right upper lobe orifice. The right DLT endobronchial

Fig 5 : Univent bronchial blocker system



cuff is eccentric, and permits the right upper lobe ventilation slot to ride over the right upper lobe orifice[34-36]. Bumenof et al, suggested that left-sided double-lumen tubes are preferred to right-sided double-lumen tubes because they have a much greater positioning margin of safety.

The complications associated with the use of double lumen tubes, though rare are malpositioning / dislodgement of the bronchial cuff, and airway trauma. Dislodgement of bronchial cuff due to over inflation, surgical manipulation or extension of head and neck can also cause malposition of the tube, necessitating bronchoscopic confirmation of tube placement before and after positioning.

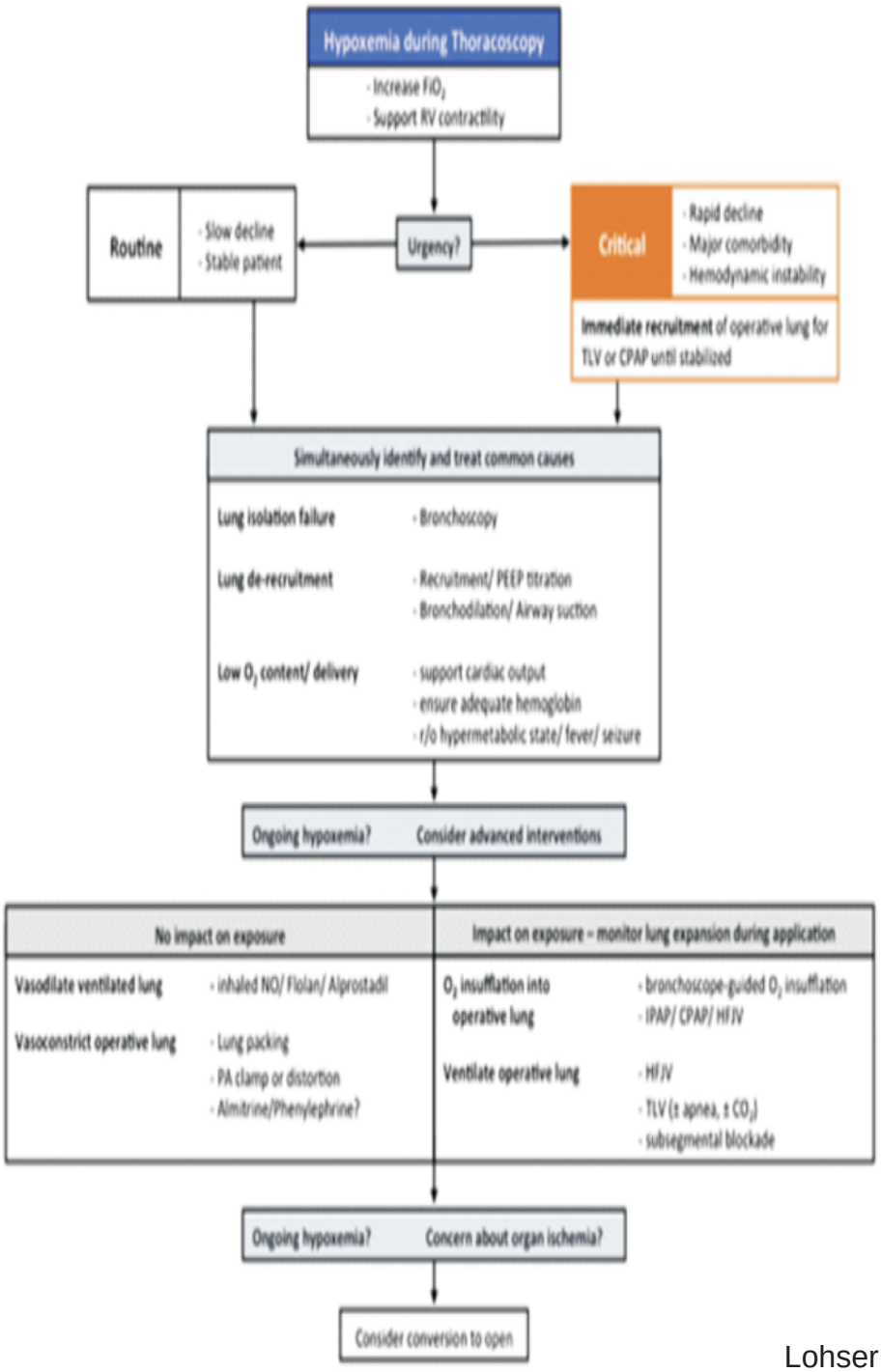
SINGLE LUMEN TUBES WITH BRONCHIAL BLOCKERS- Involves blockade of bronchus to allow lung collapse distal to the occlusion using endobronchial blockers. They include Fogarty, Foley's and Swan ganz catheters, Uninvent tubes and wire guided Endobronchial blockers. (Fig 5).

The recently used bronchial blockers are catheters with one or two distal balloons incorporated at the tip. When the balloon is inflated, airflow to the distal airways beyond it is interrupted.

1. The Arndt blocker: This blocker has a high volume, low pressure balloon and a guide wire within the lumen, to couple it with the bronchoscope and aid placement. The balloon also has distal side holes that help in deflation.
2. Cohen Flexi tip blocker: High volume low pressure balloon with distal Murphy's eyes. At the proximal end it has a wheel twisting device which aids in deflecting the tip into the desired bronchus.
3. The Fuji Uniblocker: This blocker is torque controlled with a silicone balloon that provides gas barrier property to diffusion of gases. Even at maximal cuff inflation (6ml) it doesn't exceed a recommended safety limit of<30mmHg trans mural pressure.
4. EZ blocker: This is a new blocker with a Y shape and independent channels in each port, to block different parts separately.

MAINSTEM INTUBATION- Main stem bronchial intubation with single lumen tube may be used in emergency cases or in children.

Table 5 : Interventions for Management of Intractable Hypoxia during OLV



Lohser 2012

However, with this the exhalation of the non-dependent lung is limited; airway protection at vocal cords is compromised. Repositioning or advancement of the tube may be needed intra-operatively.

VENTILATORY STRATEGIES FOR ONE LUNG VENTILATION-

There is definitely risk of hypoxemia during OLV.Pre-operative prediction of hypoxia risk facilitates decisions of change in lung isolation device, modification of ventilatory technique, pharmacological agents etc.

The predictors can be divided based on patient and procedure or lung pathophysiology

OLV is associated with severe hypoxia at times. Causes of such hypoxia will include failure of O_2 supply or discrepancy in the demand –supply ratio. Specific causes are malpositioned DLT, increased shunt fraction, absorption atelectasis of the ventilated lung, gradual resorption of O_2 in the non- ventilated lung or rarely transfusion associated lung injury (TRALI).

Measures to prevent hypoxemia include avoidance of inhibitors of HPV, use of PEEP to prevent de-recruitment and shunting in non-operative lung. Decreased cardiac output due to neuraxial anaesthesia, parenteral anaesthetic agents or tamponade physiology from CO_2 insufflation will impair mixed venous oxygen concentration, so restoration of normal cardiac output with inotropes may be needed.

Treatment of hypoxemia during VATS – Prompt diagnosis and management is the key to successful treatment of hypoxia under OLV. Malposition of DLT should be ruled out by fiberoptic bronchoscopy. PEEP of <5 cm can be applied to the ventilated lung, with CPAP to the non-ventilated operative lung. Severe hypoxemia calls for resumption of two lung ventilation but other interventions should be tried first. In resistant cases a temporary clamp to the PA to the non-ventilated lung may help. The interventions are summarized in Table 5.

Table 6: Lung protective strategies during OLV for thoracoscopy

a. Maintaining the fraction of inspired oxygen (FiO2) as low as possible to avoid absorption atelectasis and worsening shunt
b. Positive end expiratory pressure (PEEP) above the lower inflection point on the static pressure volume curve.
c. A tidal volume of 5-6 ml/kg, plateau pressure of less than 20 cm water above the PEEP value.
d. Peak inspiratory pressure less than 35 cm of water
e. Respiratory rate 10-18/min
f. Inspiratory: expiratory (I:E) ratio 1:2 or 1:3
g. Permissive hypercapnia
h. Preferential use of pressure limited ventilator modes

Injury from one lung ventilation can result in re expansion pulmonary edema (REPE), acute lung injury (ALI), or acute respiratory distress syndrome (ARDS). Poor fluid dynamics, malnutrition, inflammatory response or REPE predispose the patient to ALI after OLV[37-39]. Protective ventilatory strategies and judicious fluid administration can decrease the risk of ALI[40].

Table 6 illustrates certain lung protective strategies during OLV.

MONITORING

The level of monitoring should be decided on severity of cardiopulmonary compromise and concurrent disease states.

MONITORING OF VATS UNDER LOCAL ANAESTHESIA

In ASA grade I and II adults, VATS may be attempted under local anaesthesia with or without sedation. Minimum mandatory non-invasive monitoring will suffice for procedures

Table 7. Invasive and Noninvasive techniques of cardiac INVASIVE			
Technique	Sample site	Advantage	Disadvantage
Pulmonary artery (PA) catheter	PA	Good assessment of fluid responsiveness	1. Dependent on pulmonary vascular & cardiac anatomy. 2. It is not a continuous process, but a derivation of CO from intermittent sample. 3. Risk of arrhythmias, infection. 4. Recalibration needed with every position change[28]
Pulse index continuous cardiac output (PICCO)Lithium dilution cardiac output (LIDDCO)	CVP line/ peripheral venous line	Accurate[28,29]	1. Assumed aortic diameter based on patient's demography. 2. Measures aortic blood flow and not CO 3.May be influenced by changes in upper and lower body blood flow[30,31]
Oesophageal Doppler monitoring	Oesophagus		Useful only for patients under sedation/GA. Training needed[32]
Flotrach/Vigileo	Arterial line	1. Less invasive than PA. 2. Short time required for set up and acquiring data[29]	1. Ambiguity of combined CO and vascular tone limits its application 2. Cannot give mixed venous O_2 saturation and right heart perfusion pressures 3. Only for adult use. Use in pediatric population is not studied . 4. Not for patients with ventricular assist devices, aortic regurgitation, atrial fibrillation, hypothermia, dynamic autonomic states[33]

NON INVASIVE			
Technique	Sample site	Advantage	Disadvantage
Transesophageal echocardiography (TEE)	Oesophagus	1. Allows real time visualization of anatomy and function[33]. 2. Can detect volume changes early 3. Reliable for diagnosing unidentified intra operative hemodynamic instability due to unknown cause[27]	1. Extensive training required[2]. 2. More liable to iatrogenic errors 3. Equipment is expensive 4. In thoracic surgeries it may be difficult to position the esophageal probe in lateral position or may not allow window because of the open lung[27]
End tidal carbon dioxide ($ETCO_2$)	End tidal gas sample	1. Rapid 2. Real time 3. Reliable for ventilated intubated patients[28]	1. Underestimates CO. 2. Unreliable for spontaneously breathing patients. 3. Unreliable in patients with a shunt fraction>30%
Impedance cardiography (ICG) Thoracic electrical bio impedance (TEB)	Thoracic impedance		Clinical use not yet established

lasting less than half hour, with minimal fluid shifts.

In patients with potential of severe respiratory embarrassment, invasive monitoring, especially real time arterial blood pressure monitoring is highly recommended. A baseline blood gas analysis must be considered.

MONITORING UNDER GENERAL ANAESTHESIA:

In all patients undergoing thoracoscopy under GA, endotracheal intubation is a must.

Along with the basic monitoring like Electrocardiogram (ECG), Pulse oximetry (SpO2), non-invasive blood pressure (NIBP) and end tidal carbon dioxide ($EtCO_2$), one must consider the use of invasive monitoring and monitoring of ventilatory parameters of the anaesthetized patient.

1. Serial blood gas determinations should be done for patients under OLV.
2. Ventilatory parameters and FiO_2 monitoring. During OLV, high ventilating pressures are commonly used to maintain oxygenation, thus predisposing the emphysematous lung to barotrauma. Hence it is important to monitor the ventilating pressures at all times.
3. A FiO_2 of 1.0 is commonly used as it

ensures a PaO2 of over 150 mmHg, but prolonged high FiO2 may be hazardous.

4. Central venous pressure (CVP) monitoring is indicated especially when anticipated blood loss is more than 10% of circulating blood volume, patient has a cardiac disease or lung resections
5. Cardiac output: OLV may necessitate cardiac output monitoring, especially for major surgeries and compromised patients. There are various non-invasive and invasive techniques available to monitor cardiac output[41] (CO) (Table 7)

VENTILATION AT THE COMPLETION OF THORACOSCOPY

At the end of VATS, both the lumina of the double lumen endotracheal tube should be suctioned carefully to remove mucus, debris& blood, before re-inflating the collapsed lung. Both the lungs should be fully expanded for alveolar recruitment. A sustained application of high inflating pressures will be required for some time, till the lung expands fully[42]. A check bronchoscopy may be done to confirm bronchial integrity and remove all debris. Earlier normal saline was poured in the chest cavity to check for leaks.

POST ANAESTHESIA RECOVERY CONSIDERATONS

Post anaesthesia care requires provision of adequate ventilation and oxygenation and early recognition of possible complications. Post-operative analgesia is easier to achieve in VATS as compared to open thoracic procedures. Extubation is preferred where feasible[43]

Once extubated, next focus is on optimizing the lung recruitment and cardiopulmonary physiology, and this is done in three steps:

1. Control of pain- Adequate analgesia reduces sympathetic over activity and thus improves blood flow.
2. Positioning- A semi Fowlers position reduces the risk of aspiration while decreasing the work of breathing and prevents deep vein thrombosis (DVT) in the long run.
3. Oxygenation- Oxygen supplementation for at least 24 hours reduces pulmonary vascular resistance, especially in patients with pre-operative poor lung function.

POST OPERATIVE COMPLICATIONS

For obvious reasons of proximity to vital thoracic structures, thoracoscopy poses a high risk. Immediate complications are tabulated in Table 8.

Table 8: Immediate complications of VATS

Due to DLT or blockers	Trachea bronchial trauma or rupture Vocal cord damage
Due to mismanaged OLV	Hypoxia, REPE, ALI
Due to over enthusiastic hyperinflation	Barotrauma
Instrumentation	Damage to mediastinal structures Pneumothorax Cardiac herniation

Other complications include ongoing hypoxia, alveolar derecruitment and chronic pain though rare

PAIN MANAGEMENT

Routine thoracotomies are associated with severe post-operative pain, 30-40% of them getting converted to chronic pain. Thoracoscopy has a distinct advantage in this regard, as pain is minimal. There is ample literature to support less pain in VATS as compared to open techniques. Walker et al concluded in a study involving 83 VATSv/s 115 open thoracotomies, and found that the opioid

analgesic requirements of VATS patients were much lower than the latter. ($p < 0.001$).The severity and duration of pain after thoracotomy obviously depends on the degree of rib spreading and on the site and modality of thoracotomy incision.

Landreneau et al. report significantly better pain relief with VATS as compared to open surgery in pulmonary resections in the first year. However, there was no significant difference in the incidence of chronic pain after either technique (VATS 22%; thoracotomy 29%), or narcotic usage (VATS 6%; thoracotomy 16%)[44.]

POST OPERATIVE ANALGESIA

1. Systemic opioids: May be given intravenously, intramuscularly, transdermal, transmucosal. Patient controlled analgesia (PCA) pumps allow safe use of opioids by reducing the total dose given. Ketamine in low doses and may be used as an adjunct to opioids[45, 46].
2. Non-steroidal antinflammatory drugs (NSAIDS): NSAIDs should be used cautiously in patients susceptible to renal damage and platelet dysfunction. COX 2 inhibitors may be safer and better adjuncts to NSAIDs, tramadol or opioids.
3. Thoracic epidural (TEA): Epidural analgesia with local anaesthetics may be very useful for open techniques, or where conversion is likely.
4. Thoracic paravertebral nerve block: Percutaneous insertion of local anaesthetic into this space may produce ipsilateral block of the same level.
5. Intercostal nerve block: As effective as TEA, especially for small procedures. Provides adequate unilateral block at same level.
6. Intrapleural or extra pleural infusions of LA
7. Elastomeric pumps for wound infiltrations: This is a safe and effective technique[47]

COST EFFECTIVENESS OF VATS COMPARED TO OPEN PROCEDURES

The cost of treatment is significantly lower with VATS when compared with open surgeries, despite use of expensive equipment.

A study[48]on a total of 3,961 patients (open n=2,907, VATS n=1,054) concluded that VATS was definitely more cost effective than open procedures for the same level of patient risk. The length of stay, risk of adverse events, pneumonia ,arrhythmias, other cardiac events, bleeding and prolonged hospital stay were found to be significantly more prevalent in the open group than in the VATS group. Hospital costs were higher for open versus VATS. Other factors determining the cost were surgeon's experience and the lobe or segment involved. .

CONCLUSION

VATS is a minimally invasive surgery with various advantages. It however requires a much more technical proficiency of both the surgeon and the anesthetist. The probability of conversion should always be explained and taken into account. Conversion from VATS to open thoracotomy is generally for technical reasons and rates range from 1.7-11% in different centers.

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SINUS TACHYCARDIA IN YOUNG PATIENT IS MERELY NOT ALWAYS APPREHENSION; IT MAY BE A MANIFESTATION OF ATRIAL SEPTAL ANEURYSM-A RARE CASE REPORT.

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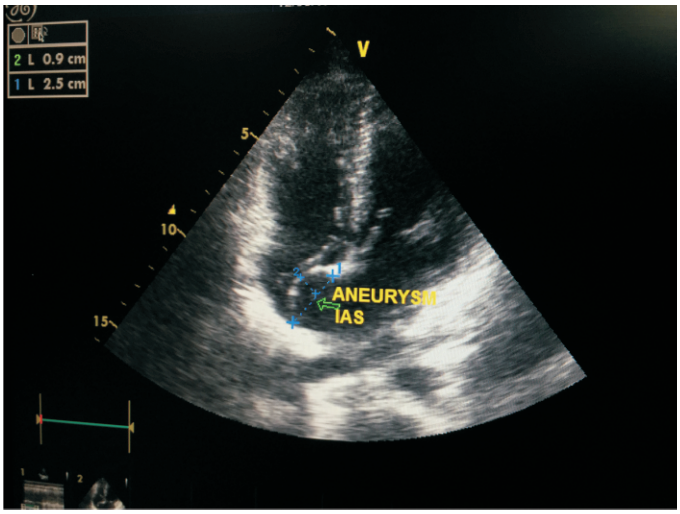
ABSTRACT

Atrial septal aneurysm (ASA) is one the cardiac congenital malformation which either confines itself to the thin fibrous remnant sheet of Fossa ovalis or involves whole of the septum between right and left atrium .This aneurysmal septum looms out either towards right or left atrium during cardiac cycle. Once considered to be a rare now being more frequently diagnosed in patient with wide spread use of 2-D Transthoracic Echocardiography for screening the preanesthetic patient. It is usually asymptomatic, but may be associated with other congenital anomaly like Patent Foramen Ovale, Patent Ductus Arteriosus, or may present with atrial arrhythmias or cryptogenic strokes. Here we report a case of ASA Who was asymptomatic clinically and presented with sinus tachycardia in ECG.



KEY WORDS

Sinus Tachycardia, Atrial Septal Aneurysm, Transthoracic Echocardiography, Congenital Anomaly, Anaesthesia.

INTRODUCTION

Atrial septal aneurysm is one the cardiac congenital malformation which either confines itself to the thin fibrous remnant sheet of Fossa ovalis or involves whole of the septum between right and left atrium .This aneurysmal septum is saccular defect that thrusts out either towards right or left atrium or in both directions during cardiac cycle[1].

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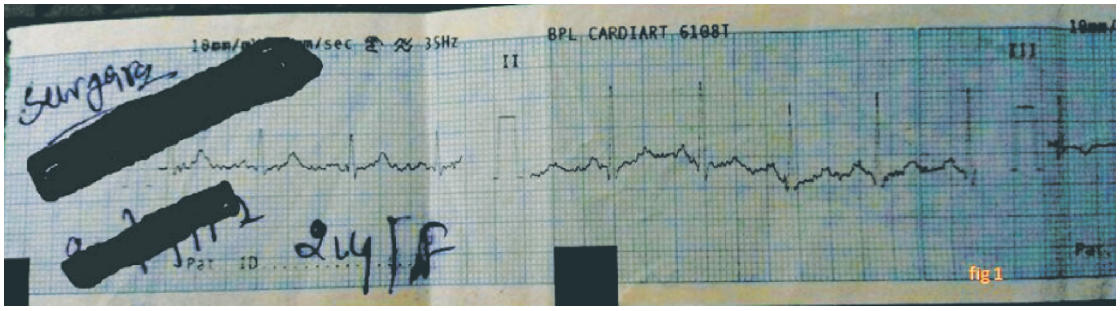
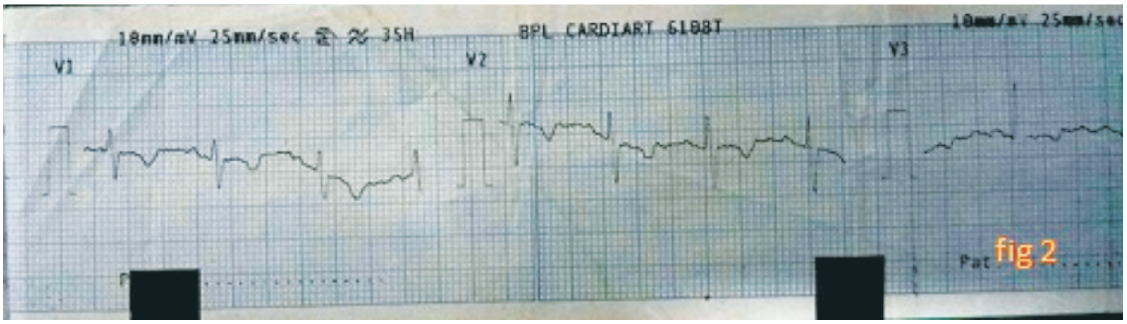


1 Figure 1.ECG leads I, II, III revealing sinus tachycardia



2. Figure2.ECG showing T WAVE INVERSION in leads v1, v2, v3

Although it was considered to be rare , however, with the advent of 2-D transthoracic echocardiography (TTE) and the widespread use of trans esophageal echocardiography (TEE) for screening preoperatively it is being more and more diagnosed in patients.[2] The TTE delineate around 2% of patients are having ASA [1]. It is commonly present along with other congenital heart diseases like Patent foramen ovale(PFO), Patent ductus arteriosus(PDA), Atrial septal defect (ASD), Ebstein's anomaly Ventricular septal defect(VSD), Valvular prolapse (VP), and Tricuspid atresia, as well as with acquired heart diseases i.e., cardiomyopathy, systemic and pulmonary hypertension, ischemic heart disease, valvular disease arrhythmias and thrombus formation. [2]

Here we report a case of ASA in young patient without any symptomology, a written well informed consent was given by the patient for this purpose of publication.

A young female aged 17 years came for preanesthetic consultation scheduled for excision of fibro adenoma breast bilaterally. Apart from surgical problem patient did not have any significant history related to cardiovascular, respiratory or central nervous system .i.e. palpitation, breathlessness, headache or syncopal attack. Clinical examination revealed regular pulse but tachycardia with heart rate of 132 bpm and blood pressure of 110/70mmHg. Peripheral pulses were normal. On auscultation normal heart sounds S1 S2 were heard. Routine laboratory examination revealed hemoglobin 10.4gm%, , serum urea14mg/dl% ,serum creatinine 0.7mg/dl and coagulation profile all within normal range EKG revealed sinus tachycardia with T, wave inversion in lead v1, v2 ,v3 as shown in (figure1,2.) Chest X-ray showed normal cardiac silhouette, no cardiomegaly and clear lung fields. Patient was referred to cardiologist for expert review.TTE

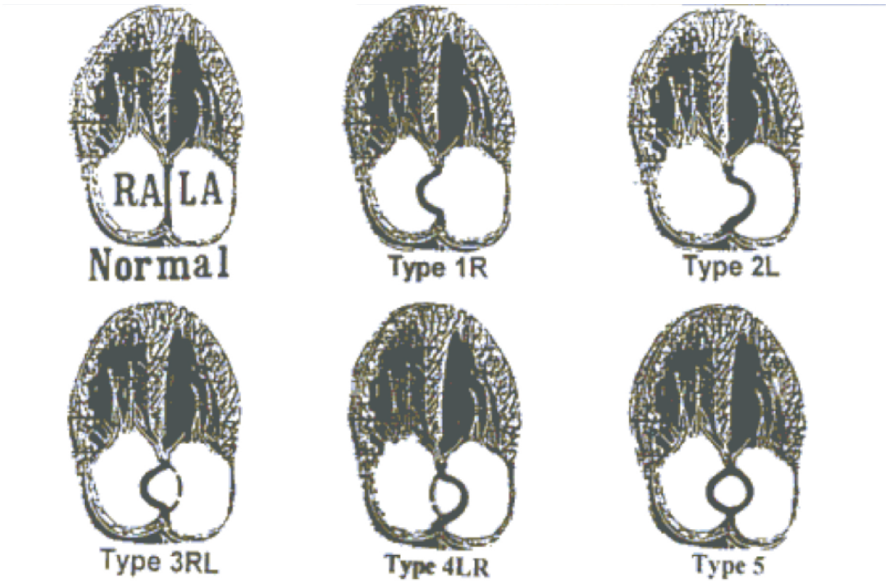


Figure.3. ASA bulging into right atrium (Type 1R) during cardiorespiratory cycle as seen in 2-Dimentional Transthoracic Echocardiography

done which revealed normal size chambers ,valves, intraventricular septum was intact, intra atrial septum showed a bulge in right atrial cavity, signaling TYPE 1R ASA according to the new classification of ASA as shown in figure. 3. No evidence of thrombus in any of the chamber, or associated other congenital anomaly was evident.

Anaesthetic Management

Knowledge of the anatomy and physiology of the congenital anomaly is important for choosing appropriate monitoring and inducing agents. Such patients usually come for repair of septum or closure device. As such in this case with TYPE 1R ASA, without any other congenital problem, we needed to administer anaesthesia for non cardiac surgical procedure of excision of fibro adenoma. Induction done with inj. Midazolam 1mg, Fentanyl 2mcg/kg, Propofol 2mg/kg, Anaesthesia was maintained with variable concentration of Sevoflurane 1-4%. Patients received Oxygen 33% and Nitrous Oxide 67% in spontaneously breathing patients, with LMA in situ. Monitoring was done with NIBP, ECG, RR, HR. SPO2 and Continuous capnography.

After the surgery, sevoflurane was stopped and LMA was removed after eye opening and mouth opening to command. After removal of LMA patient was observed in recovery, then shifted to ward, and finally discharged on very next day.

DISCUSSION.

Atrial septum aneurysm (ASA) is a rare cardiac clinical entity that was first reported by Lang and Posselt in 1934.[3] It is an isolated deformity “saccular” like at the level of Fossa Ovalis which usually balloons out either to right or left atrial chambers, or to both sides at a time during the cardiac cycle. It is a noteworthy cardiac malformation, which is serendipitously diagnosed, and is a well known causative factor for cardio embolic stroke albeit there is no focus of thrombus in aneurysm itself or in left and right atrium when screened via TTE.[4]

Patients with ASA are generally

Table 1.New Classification of ASA

Serial No.	Type of septal Aneurysm	Direction of movement of Aneurysmal Atrial Septum
1	Type 1R	ASA projects from the midline of the atrial septum to the RA throughout the entire cardiorespiratory cycle
2	Type 2L	the ASA protrudes from the midline of the atrial septum to the LA throughout the entire cardiorespiratory cycle
3	Type 3RL	the maximum deflection of the ASA is toward the RA and lesser toward the LA
4	Type 4LR	the maximal excursion of the ASA is toward the LA
5	Type 5	the ASA movement is bidirectional

ASA: Atrial Septal Aneurysm, LA: Left Atrium, RA: Right Atrium.

asymptomatic and do not present with any of the electrocardiogram or clinical findings implicative of ASA.[5] On auscultation a non-ejection systolic click may sporadically be audible possibly produced, as the Inter ASA protrudes and tenses within LA/RA chambers during cardiorespiratory cycle. [6]

Sometimes it may be associated with clinical variables like Cardiovascular embolism, Hypertension, Coronary Artery diseases, Diabetes Mellitus, Valvular prolapse, arrhythmias, valvulopathies, PFO, ASD, [2,5]

Scholz EP,Zitron E,Katus HA et al. reported a case of ASA with in electrocardiogram finding of right atrial enlargement and thereby suggests echocardiography even in asymptomatic patients with abnormal 12-lead electrocardiogram. This reported case presented with asymptomatic tachycardia when further reviewed by cardiologist, shows T wave inversion in lead v1 v2 v3 in ECG (Figure 2.), and ASA in TTE (Figure 3)

Clinical Manifestations attributed to ASA are usually atrial arrhythmias, ventricular tachycardia's[7]. These primary congenital malformations at fossa ovalis or involving the entire interatrial septum result in interatrial pressure difference generating tachyarrhythmia's.[7,8] At times interatrial septal aneurysm itself behave as an arrhythmic focus, generating focal atrial tachycardia. [1,9] In a study delineated by Hanley et al it was observed out of 80 patients 25% of them had atrial arrhythmias. Majority of the reported cases are congenital in nature and are detected incidentally. [1,9]

ASA is also known to cause complications like arterial embolism, the presence of aneurysmal septum physically obstructs the blood flow during cardiac systole and advocates stasis of blood flow, generating a focus of minute LA clots and predisposes to systemic thrombo embolism. [9,8] Mechanism of cardio embolic stroke could be right to left shunting or the thrombogenic properties of aneurysm itself. [4]

Transthoracic Echocardiography is the imaging modality of preference for ASA diagnosis, for routine echocardiography screening or during the event of cardio embolic cerebrovascular stroke and peripheral embolism. In comparison to TTE, TEE is more sensitive in picking up ASA [8.9]

The presence of isolated and uncomplicated ASA usually requires assurance and follows up apart from any specific treatment. A thorough evaluation need to be executed to rule out presence of thrombus in patients with aneurysm. Various therapeutic disciplines for prevention of recurrent stroke in patients with atrial septal aneurysm are commencing medical therapy with antiplatelet

agents, anticoagulants and surgical or percutaneous closure of the defect [4]

Various classification has been laid out since 1985, Longhini et al [10]., then by Hanley et al[1], In 1989 Roudaut et al. [11] In 1991 Pearson et al [9], to describe ASA, later In 1997 Alexander Olivares-Reyes et a[12] based on the direction and movement of ASA proposed a new classification as described in Table 1.

CONCLUSION

Thus the patient reported above had TYPE 1R type of ASA, without any prior thromboembolic events or headache. It was not possible to rule out the cause of rise in left atrial pressures leading to the development of ASA. No clear guidelines exist regarding management of ASA, incidentally found ASA without any symptoms do not require any treatment. Though debate continues on antiplatelet therapy with aspirin alone versus oral anticoagulation therapy with warfarin in patients with PFO. The Lausanne Study, observed hardly any difference in primary end points of recurrent stroke or mortality at the end of 2 years in aspirin alone versus warfarin alone treated groups [13] Most reported cases of ASA are incidental findings[9]. ASA is increasingly gaining clinical significance as it is attributed cardio-embolic stroke and arrhythmias.

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PERIOPERATIVE ATRIAL FIBRILLATION- ANAESTHESIOLOGIST'S PERSPECTIVE

● Dr. Ajita Annachhatre[1]

Abstract-

Atrial fibrillation is the most prevalent arrhythmia. Development in diagnostic and availability of newer modalities of treatment has certainly improved life expectancy in India. Atrial fibrillation has incidence of 1-2 % in old patients of age more than 75 yrs globally.Rheumatic heart disease and atrial fibrillation is another cardiac disease healthcare concern in developing country like India.

Patients with atrial fibrillation or new onset atrial fibrillation pose special concerns and need meticulous management. The anaesthesiologist needs a thorough preoperative evaluation and risk stratification for complications of atrial fibrillation and management of intraoperative sudden onset of atrial fibrillation.Perioperative atrial fibrillation can be safely managed with successful outcome.

Introduction-

Atrial fibrillation is the most prevalent cardiac arrhythmia.[1] It is a disorder of electrical activity in the atrial myocytes.It affects five million people globally.[2] The prevalence increases from 1%-age>60 yrs, 7.2%-age>65 yrs and 10% -age>75 yrs[3,4].Life expectancy in India has increased from 60 yrs to 65 yrs in national survey of 2010 to 2015[5] Rheumatic heart disease is still major healthcare concern in India so the incidence of atrial fibrillation with RHD is 40.7% in Indian cohort as compared with 26.7%globally.Men are more affected globally of but in India females are more at risk because of the RHD and mitral valve diseases.Incidence of atrial fibrillation in India is at approximately around 54 yrs compared with the age of 65 yrs and above in western countries![6]

History

Atrial fibrillation was diagnosed more than a century ago, many stalwarts have done extensive study of atrial fibrillation and treatment of the disease.

1. William Harvey (1578-1657), who first described the circulatory system appropriately, was probably the first to describe fibrillation of the auricles in animals in 1628.
2. The French "clinical pathologist", Jean Baptist de Sénac (1693-1770) was the first who assumed a correlation between "rebellious palpitation" and stenosis of the mitral valve.
3. Robert Adams (1791-1875) also reported in 1827 the association of irregular pulses and mitral stenosis.
4. The discovery of digitalis leaf in 1785 by William Withering (1741-1799) brought relief to patients with atrial fibrillation and congestive heart failure by reducing the ventricular rate.
5. The first human ECG depicting atrial fibrillation was published by Willem Einthoven (1860-1927) in 1906.
6. Carl Julius Rothberger and Heinrich Winterberg in 1909 described direct connection between absolute arrhythmia and atrial fibrillation .

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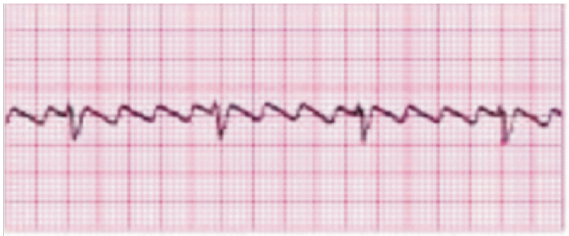
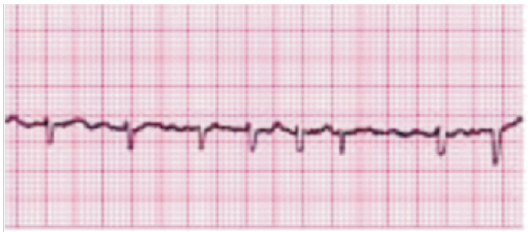
Table 1- Differences in Atrial Fibrillation and Flutter. [From-ACC/AHA Tachyarrhythmia algorhythm 2014]

Defining Criteria and ECG Features		Atrial Fibrillation	Atrial Flutter
(Distinctions here between atrial fibrillation vs atrial flutter; all other characteristics are the same) **Atrial Fibrillation Key:** A classic clinical axiom: *"Irregularly irregular rhythm—with variation in both interval and amplitude from R wave to R wave—is always atrial fibrillation."* This one is dependable. **Atrial Flutter Key:** Flutter waves seen in classic "sawtooth pattern"	**Rate**	■ Wide-ranging ventricular response to atrial rate of 300-400 beats/min	■ Atrial rate 220-350 beats/min ■ Ventricular response = a function of AV node block or conduction of atrial impulses ■ Ventricular response rarely >150-180 beats because of AV node conduction limits
	Rhythm	■ Irregular (classic "irregularly irregular")	■ Regular (unlike atrial fibrillation) ■ Ventricular rhythm often regular ■ Set ratio to atrial rhythm, eg, 2-to-1 or 3-to-1
	P waves	■ Chaotic atrial fibrillatory waves only ■ Creates disturbed baseline	■ No true P waves seen ■ Flutter waves in "sawtooth pattern" is classic
	PR	■ Cannot be measured	
	QRS	■ Remains ≤0.10-0.12 sec unless QRS complex distorted by fibrillation/flutter waves or by conduction defects through ventricles	



Atrial flutter



Atrial fibrillation

7. Sir Thomas Lewis (1881-1945), the father of modem electrocardiography, studied electrophysiological characteristics of atrial fibrillation and has shown that its basic perpetuating mechanism is circus movement of electrical impulse (re-entry).
8. Karel Frederick Wenckebach (1864-1940), Gordon Moe (1915-1989), Bernhard Lown (1921) and Maurits Allessie has described the clinical symptoms.[7]

Definition and Types of atrial fibrillation-

Atrial fibrillation is a supraventricular arrhythmia characterized by complete absence of coordinated atrial contractions.Atrial fibrillation is associated with an irregular and frequently rapid ventricular response if atrioventricular conduction is intact.[8]

Atrial tachycardias can be atrial flutter, premature ventricular contractions and atrial fibrillation.

Table 2- Atrial Fibrillation and Anaesthesia (Copied from anaesthetics.ukzn.ac.Za / libraries / cardiac)

Associated cardiovascular disease	Non-cardiac associations
• Hypertension	• Alcohol
• Coronary artery disease	• Hyperthyroidism
• Cardiomyopathy	• Pulmonary disorders
• Valvular disease (mitral)	• Pulmonary embolism
• Cardiac surgery	• Obstructive sleep apnoea
• Myocarditis	• Thoracic or oesophageal surgery
• Pericarditis	• Sepsis
• Other supraventricular arrhythmia(s) and	• Vagal and sympathetic mechanisms.[5]
• Wolff-Parkinson-White syndrome (WPW)	

Characteristics of Atrial fibrillation and atrial flutter are studied comparatively with ECG findings in Table.1.[9]

Mechanism of atrial fibrillation-

Mechanism is not clear completely but three concepts are important in genesis and maintenance of it.

Changes in atrial structure potentially increases risk of Atrial fibrillation.[10] Changes can be due to cardiac or noncardiac causes . In cardiac diseases like hypertension ,valvular heart disease and coronary artery diseases increase in LA pressure leads to dilatation of atria and increase in wall stress leading to fibrosis, cellular changes disturbing normal conduction pathway.Non cardiac causes leads to infiltrative and inflammatory changes in atrial musculature.[11] Myocardial fibrosis is the commonest cause for atrial fibrillation.[12]These changes alter impulase conduction ,refractoriness generating arrhythmogenic foci.

Three concepts are important in genesis and maintenance of it which are as following-

1. Enhanced automaticity in atrial tissues that extend into the pulmonary veins or venacaval junctions. These are common trigger points of atrial fibrillation. Ablation therapy can completely cure these ectopic foci automaticity.
2. Daughter wavelets formation and maintaining chaotic electrical state, common in chronic atrial fibrillation.Small impulses stimulate groups of atrial cells leading to small twitches of contraction which can be seen as quevering of atria. These weak atrial contractions are not conducted to ventricles and so the rate of atrial contraction is high 450-600/min but ventricular contraction rate is up to 150-180/min.
3. As atrial fibrillation persists it leads to structural and electrical remodelling of atrial tissue causing shortening of effective refractory periods.[13,14]

Types or classification of atrial fibrillation-[15]

First onset atrial fibrillation-

First clinical presentation where the patient is still in atrial fibrillation and the episode has been present for less than 48 hrs.

Paroxysmal atrial fibrillation-

Occurrence of recurrent episodes that typically lasts minutes to hours, but terminated on its own or cardioversion in 7 days.

Persistant atrial fibrillation-

Atrial fibrillation when lasts for more than 7 days, even if terminated later on, cardioversion is required to restore sinus rhythm.

Permanent atrial fibrillation-

When all attempts to restore sinus rhythm have been abandoned.

Etiology of Atrial fibrillation.-

Atrial fibrillation in normal hearts is termed as lone atrial fibrillation.

Causes and risk factors for atrial fibrillation are as follows-[16] Table 2

Management –

Management of atrial fibrillation is one of the major therapeutic challenge. Haemodynamic consequences causing acute complications should be treated immediately. Management basically differs according to type of atrial fibrillation.Long term medical management should consider the major risk of ischaemic stroke and recurrence of atrial fibrillation.Risk stratification for stroke should be done and accordingly treatment should be started.Etiological factors should be treated at the same time to avoid repeated of episodes atrial fibrillation.

Four principles of management are as follows-

1. Restoration of sinus rhythm either by drugs or electrical means.
2. Control of ventricular rate.
3. Prevention of recurrence of paroxysmal or persistent atrial fibrillation after successful restoration of sinus rhythm.
4. Prevention of thromboembolic phenomenon.

1.Restoration of sinus rhythm-

In patients of atrial fibrillation of sudden onset or paroxysmal atrial Fibrillation reverting the arrhythmia to sinus rhythm is beneficial .It can be done either by Electrical means or by pharmacological agents like antiarrhythmics or beta blockers.

A] Direct Current Cardioversion [DCCV] [17]

It involves synchronized direct current electrical shock delivered across the chest wall.Initial shock energy of 200 J for monophasic device. The sequence of energy used is 200 J ; 300 J;and 360 J. Biphasic machines requires lower energies.Both monophasic or biphasic shocks have equal efficacy.

According to 2014 guidelines class I indication are as follows -

1. In pursuing a rhythm-control strategy, cardioversion is recommended for patients with AF or atrial flutter as a method to restore sinus rhythm. If cardioversion is unsuccessful, repeated attempts at direct-current cardioversion may be made after adjusting the location of the electrodes, applying pressure over the electrodes or following administration of an antiarrhythmic medication. (Level of Evidence: B)
2. Cardioversion is recommended when a rapid ventricular response to AF or atrial flutter does not respond promptly to pharmacological therapies and contributes to ongoing myocardial ischemia, hypotension, or HF. (Level of Evidence: C)
3. Cardioversion is recommended for patients with AF or atrial flutter and pre-excitation when tachycardia is associated with hemodynamic instability. (Level of Evidence: C)

Elective Direct Current Cardioversion [DCCV] performed with adequate short acting anaesthesia.Important pre-requisite for this procedure is 2-D Echo confirmation of absence of clot in Left Atrium of heart.Patient should be anticoagulated in unknown duration of onset of atrial fibrillation .

B] Pharmacological Restoration of Sinus rhythm-

Table 3. Rate controlling drugs [copied from-Acta Anaesthesiologica Taiwanica 51 (2013) 34-36]

Category of rate-controlling drugs.

Drug category		Intravenous dosage	Adverse effects
β-blockers	Esmolol	Loading: 0.5 mg/kg over 1 min Maintenance: 0.06–0.2 mg/kg/min	Hypotension, bradycardia, heart block, bronchospasm, and heart failure
	Metoprolol	Bolus 2.5–5 mg over 2 min at 5 min interval up to 3 doses	
	Propranolol	Bolus 0.15 mg/kg	
Ca channel antagonists	Diltiazem	Loading: 0.25 mg/kg over 2 min Maintenance: 5–15 mg/h	Hypotension, bradycardia, heart block, heart failure, And reduces digoxin elimination
	Verapamil	Bolus 0.075–0.15 mg/kg over 2 min	
Digitalis glycoside	Digoxin	Loading: 0.25 mg q. 2 h, up to 1.5 mg Maintenance: 0.125–0.375 mg/d	Ventricular tachyarrhythmias, bradycardia, heart block, drug interaction, and digitalis toxicity
Amiodarone	Amiodarone	Loading: 150 mg over 10 min Maintenance: 0.5–1 mg/min	Hypotension, bradycardia, heart block, pulmonary fibrosis, thyroid and hepatic dysfunction, and warfarin interaction

In clinical practice pharmacological cardioversion is considered in haemodynamically stable patients,it requires 24 hr to few days.[18] Antiarrhythmics are used but most commonly Amiodarone- Class III, Potassium channel blocker is used IV and orally.

2. Control of ventricular rate in paroxysmal, persistant or permanent atrial fibrillation-

   Rate control is achieved by drugs with predominantly affects conduction through AV node Table 3 shows the drugs and dosages used for control of rapid ventricular rate.

3. Prevention of recurrence of atrial fibrillation is done by prescribing oral antiarrhythmic drugs. e.g. Flecainide, Propafanone, amiodarone.

4. Prevention of thromboembolic phenomena-

   Atrial fibrillation is associated with annual 5 % increased risk of thromboembolism.

Left atrial appendage is the most common site for formation of thrombus in persistent and chronic atrial fibrillation.

Risk scoring is adapted by BMJ BAFTA TRIAL.A score for one point is added for risk factor. The CHA2DS2VASc Score gives additional risk factors.[20] (Table 4)

TABLE 4 –A CHA2DS2-VASc Score (Copied from- Atrial fibrillation (AF) Pertooperative Management for Non Cardiac Surgery - ATOTW307)

Scoring System	Criteria	Low risk	Intermediate risk	High risk
CHADS2	C-Congestive Cardiac failure of Echo H- Hypertension (160/90) A>75 years D- Diabetes Mellitus S (2) - Previous Stroke/TIA	0	1	2
CHA2DS2VAS	As above plus Age-1 point if between 65-74 2 points if >75 V- vascular disease- MI/PVD/ aortic disease - 1 point S-Sex Female gender- 1 Point	0	1	2

TABLE 4 –B

Annual risk for stroke according to scoring-

CHADS2 Score	Annual Risk of stroke (%)
0	2
1	3
2	4
3	5
4	8

These patient needs warfarinisation to keep INR of 2-3.Newer Oral vit.K antagonists are also used for this. These patients preoperatively needs bridging therapy with inj. heparin to prevent perioperative clot formation and ischaemic stroke.The decision is with communication with surgeon by weighing the bleeding and stroke risk estimation.

Anaesthesia and Atrial fibrillation-

Three types of scenarios can present-

1. Pateint with pre-existing atrial fibrillation.
2. New onset intraoperative atrial fibrillation.
3. Post –operative atrial fibrillation.

1. Patients with preexisting atrial fibrillation- It needs multidiscplinary approach of Cardiologist, Surgeons and anaesthesiologist.

Key Points-

A. Needs to continue all medical treatment in perioperative period.

B. Anticoagulation and decision of bridging therapy according to type of surgery.

C. Avoid intraoperative electrolyte disturbunces.

D. Avoid sympathetic stimulations which may aggravate atrial fibrillation.

E. Avoid intra operative hypoxia,hypotension. [21]

F. Keep all emergency drugs ready. Defibrillator should be kept ready. Pacing wires and box may be needed in case of conduction block as a side effect of antiarrhythmic drug treatment.

2. Intraoperative new onset atrial fibrillation.

Causes of intraoperative atrial fibrillation are as following-[22] Table 5

Table 5 - Intraoperative causes-[copied From IJA Volume 61 | Issue 9 | September 2017]

Patient	Pathology	Position	Pharmacology
Elderly History of arrhythmias	Cardiac disease Renal disease CNS disorder COPD Electrolyte disturbances	Prone position Lateral position	Premedication Induction agent Muscle relaxant Analgesic Anaphylaxis Vasopressor Inotropic agent Reversal agent Local anaesthetics

Procedures like cardiac surgeries, thoracic surgeries, major fluid shift.CVP and PA cannulation are at more risk of developing atrial fibrillation.

Management is depended on haemodynamic stability of the patient. Systematic approach can give successful outcome. Unstable patients may need immediate cardioversion either electrical of pharmacological. Clinically Stable patients can be treated with rate controlling drugs either beta blockers or calcium channel blockers.Electrolytes imbalance, acidosis any other causes must be ruled out and treated.

3. Post operative atrial fibrillation-

New onset post operative atrial fibrillation has many factors. Management again depend on haemodynamic stability. Diagnosis of cause and correct treatment is warranted.

Factors related to postoperative atrial fibrillation are –[23] Table 6

Table 6 - Post operative risk factors for AF.[Copied From-BJA Atrial fibrillation Continuing Education in Anaesthesia, Critical Care & Pain | Volume 6 Number 6 2006]

- Advanced age	Prolonged p waves on ECG
- Males	A trial dilatation
- Previous AF	High left ventricular end - diastolic pressure
- Cardiac failure	Cardiomegaly on Chest X-ray
-Hypertension	Right coronary artery grafting
- Chronic obstructive airway disease	Prolonged bypass time
Chronic renal failure Previous cardiac surger	Inadequate cardioprotection and hypothermia

Conclusion-

Anaesthesiologists are well versed with managing circulatory disturbances.A proper evaluation of patient with atrial fibrillation with risk stratification will improve outcome with smooth recovery of patient.Intraoperative vigilant monitoring will definitely avoid the haemodynamic complications. Unstable patients requires DCCV .If patient is stable then rate and rhythm control will be sufficient to avoid cardiac as well as other fatal events.

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GUIDELINES AND RECENT CHANGES IN TRAUMA PATIENT AND GOLDEN HOUR MANAGEMENT

● Dr. Meghna Maheshwari[1], Dr. Sadhana Sanwatsarkar[2], Dr. Rohit Mazumdar[3]

Introduction

Trauma is the leading cause of death in the first four decades of life in modern industrialized countries[1].Death from trauma has a trimodal distribution:within seconds to minutes,minutes to hours (GOLDEN HOUR), several days or weeks after the initial injury.The Advanced Trauma Life Support (ATLS) Course was developed in Nebraska and soon adopted by the American College of Surgeons in 1979. The primary focus of ATLS is on the first hour of trauma management, when rapid assessment and resuscitation can be carried out to reduce deaths within the Golden Hour.

One of the most well-known principles in medicine is ‘‘golden hour’’ of trauma, which specifies that patients outcome are improved when patient is transported to a designated trauma centre within an hour of injury[2]. Nearly all emergency medical services (EMS) providers can remember their first exposure to the concept of the “golden hour” with the idea that trauma patients have significantly better survival rates if they reach surgery within 60 minutes of their injury[3]. The “golden hour” summarized by the 3R rule of Dr Donald Trunkey, an academic trauma surgeon is, “Getting the right patient to the right place at the right time.[4,5]” But the concept of golden hour is still questionable in most of countries.

In a 2001 literature review, Lerner EB et al, determined the origin of the term “golden hour”. They cited a series of studies discussing the golden hour, but noticed that those studies often referenced one another and were not accompanied by supporting data or references to other studies. Most frequently the phrase is attributed to Cowley, who used it in 1973 with reference to helicopter transport of injured patients in Maryland

OBJECTIVES

The trauma care process consists of six key steps: detection, reporting, response, on-scene care, and care in transit and transfer to definitive care.[6] Objectives of trauma care involve prompt communication and activation of the system, proper action at the scene of the crash by first responders, and the prompt response of the system or simply offer fastest possible basic life support which includes, airway, breathing, control of bleeding, and transportation of the right patient[7,8].This includes all the appropriate personnel safety precautions, assessment, and treatment of the injured people at the scene, and transport to trauma care facilities while delivering the necessary medical care before arrival at the hospital. Widespread first-aid training is the most important aspect of successful prehospital care[9]

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RECENT CHANGES IN ATLS GUIDELINES[10, 11] (10TH EDITION 2018):-

Initial Assessment

1. A bolus of isotonic solution 1 L for adults and 20ml/kg for morbidity.
2. If a patient is unresponsive to initial crystalloidtherapy, he should receive a blood transfusion.
3. Aggressive and continued volume resuscitation is not a substitute for definitive control of hemorrhage. Paediatric patients< 40 kg should be administered judiciously, as aggressive resuscitation before control of bleeding has been demonstrated to increase mortality and morbidity.

Early use of blood and blood products

1. Early resuscitation with blood and blood products must be considered in patients with evidence of class III and IV hemorrhage.
2. Early administration of blood products at a low ratio of packed red blood cells to plasma and platelets can prevent development of coagulopathy and thrombocytopenia.
3. Management of coagulopathy
4. Uncontrolled blood loss can occur in patients taking antiplatelet or anticoagulant medications.

Prevention

1. Obtain medication list as soon as possible and administer reversal agents as soon as possible.
2. Where available, monitor coagulation with thromboelastography (TEG) or rotational thromboelastometery (ROTEM).Consider administering platelet transfusion, even with normal platelet count.

THORACIC TRAUMA:-

Life Threatening Injuries are

1. Flail chest out
2. Tracheobronchial injury

Life-threatening injuries during primary survey

Airway

1. Airway Obstruction
2. Tracheobronchial Tree Injury

Breathing

1. Tension Pneumothorax
2. Open Pneumothorax

Circulation

1. Massive Haemothorax
2. Cardiac Tamponade
3. Traumatic Circulatory Arrest

Tension pneumothorax

1. When ultrasound is available, tension pneumothorax can be diagnosed using an extended FAST:seashore, bar code or stratosphere sign in M-mode
2. Needle decompression:Recent evidence supports placing the large, over-the-needle catheter at the fifth interspace,slightly anterior to the midaxillary line
3. 28-32 French chest tube for hemothorax (not 36-40Fr)
4. Aortic rupture should be managed with beta blocker, if no contraindications exist. Heart rate control with a short -acting beta blocker (esmolol) to a goal heart rate of< 80 bpm and blood pressure control with a goal MAP 60-70 mm Hgis recommended.

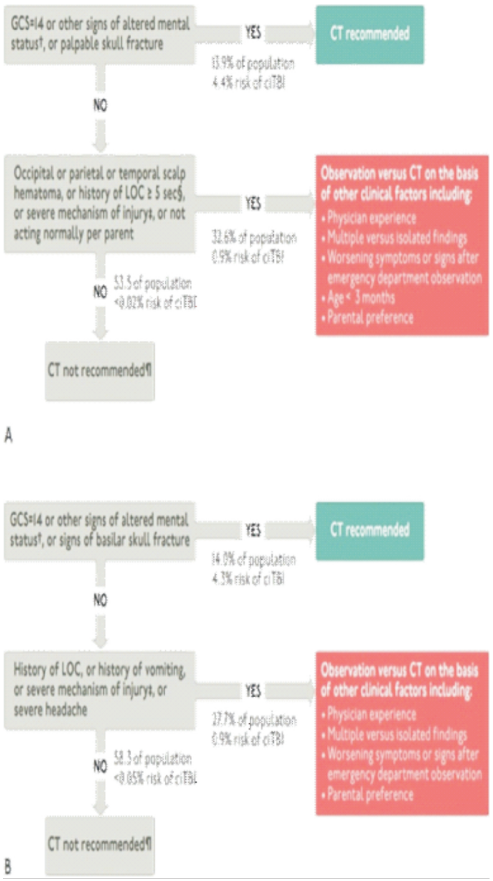
Abdominal and Pelvic Trauma:-

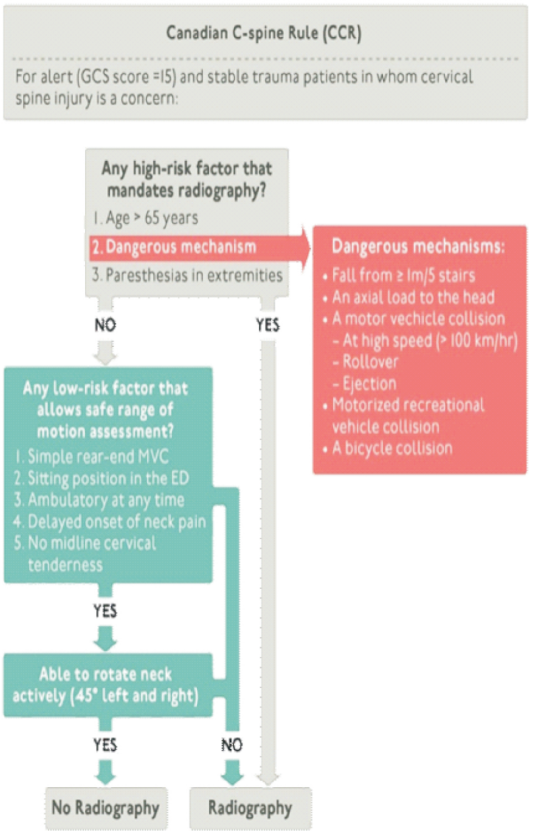
Palpation of prostate gland is no longer recommended for urethral injury.

HEAD TRAUMA:-

1. Detailed guidance on SBP management
2. Anticoagulation reversal guidance
3. Seizure prophylaxis

Table-1





A) Detailed guidance on SBP management-

Maintain SBP at ‡ 100 mmHg for patients 50-69 years or at ‡ 110 mmHg for patients 15-49 years or older than 70 years; this may decrease mortality and improve outcomes.

B) Goals of treatment of brain injury-

ACS Committee on Trauma, January 2015.

A) Clinical Parameters- Systolic BP ‡ 100 mmHg, Temperature 36–38°C

b) Monitoring Parameters-CPP ‡ 60 mm Hg 2, ICP 5–15 mm Hg, PbtO2 ‡ 15 mm Hg, SPO2 ‡ 95%

c) Laboratory Parameters-Glucose 80–180 mg/dl, Hemoglobin ‡ 7 g/dl, INR < 1.4, Na 135–145 meq/dl, PaO2 ‡100 mmHg, $PaCO_2$ 35–45 mmHg, pH 7.35–7.45, Platelets ‡ 75 $X10^3/mm^3$

C) Seizure prophylaxis:-

Prophylactic use of phenytoin (Dilantin) or valproate (Depakote) is not recommended for preventing late posttraumatic seizures (PTS). Phenytoin is recommended to decrease the incidence of early PTS (within 7 days of injury), when the overall benefit is felt to outweigh the complications associated with such treatment. However, early PTS has not been associated with worse outcomes (IIA).

SPINAL CORD AND CORD TRAUMA:-

C - spine protection changed to

'Restriction of spinal motion'

Canadian C-Spine Rule (CCR) and NEXUS Criteria- Table 1

Highlighting risk factor of bilateral femur fractures:-

Compared with patients with unilateral femur fractures, patients with bilateral femur fractures are at higher risk for significant blood loss, severe associated injuries, pulmonary complications, multiple organ failure, and death.

THERMAL INJURY:-

2 ml/kg × weight × % burn adults

3 ml/kg × weight × % burn children

Fluid titrated to urine output

Paediatric trauma:-

1. Needle thoracocentesis site is unchanged to 2nd ICS
2. Limiting crystalloid resuscitation
3. Pediatric Emergency Care Applied Research Network (PECARN) Criteria for Head CT

Pediatric Mass Transfusion Protocol- Initial 20 mL/kg bolus of isotonic crystalloid followed by weight-based blood product resuscitation with 10-20 mL/kg of RBC and 10-20 mL/kg of FFP and platelets are advised

Transfer to definitive care:-

Specific mention of avoiding CT in primary hospital

SBAR template for communication

Do not perform diagnostic procedures (e.g., DPL or CT) that do not change the plan of care.

However, procedures that treat or stabilize an immediately life-threatening condition should be rapidly performed.

Conclusion:-

A trauma care service remains a dynamic field of medicine for care of trauma patients. Therefore, improvements in the field of trauma services are required to ensure "golden hour" compliance for all trauma victims as an achievable goal by coordinating activities between prehospital care and specialized hospital care services. One technical aspect of trauma care is to improve access and to establish a uniform emergency access telephone number. Due to great heterogeneity in the literature, firm conclusions cannot be drawn. However, present literature review provides useful information about the current status of trauma care that will assist in strengthening and expansion of trauma care. A specific and unique model system should be developed to address the needs of the trauma patient. The goal should be to get 'the right patient, to the right place, at the right time, to receive the right care' following trauma.

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