THORACIC ANAESTHESIA UPDATE

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Thoracic surgery is undertaken in only 30-40 units in the UK. National data is collected on number of cases and mortality. Core operations are lobar resection, pneumonectomy for malignant and non-malignant conditions, mediastinoscopy and mediastinotomy, bronchoscopy for diagnostic and interventional reasons, video-assisted thoracoscopic surgery (VATS) for drainage and investigation of effusions, management of air-leaks, management of empyema and operations on the chest wall. Some surgeons undertake both cardiac and thoracic surgery wilst others are dedicated thoracic surgeons. Lung volume reduction surgery and lung transplantation are specialised procedures usually rationalised to specific units or surgeons. Oesophagectomy is now undertaken more commonly by 'general' surgeons. Standard anaesthetic textbooks commonly contain a thoracic chapter.

Bronchoscopy

Rigid bronchoscopy may be for diagnostic or interventional procedures. The latter include stenting, lasering and removal of foreign bodies. Propofol by manual or target controlled infusion has revolutionised the provision of anaesthesia particularly for longer procedures. Cardiovascular responses are easily controlled by alfentanil or remifentanil which both permit rapid awakening. Ventilation by the Sanders technique is still commonly used.

Double lumen tube (DLT) sizes

Suitable PVC tubes are 35, 37, 39, 41 Fr. One suggestion is (for left DLT) to measure the tracheal width on the preop CXR since this predicts the diameter of the left bronchus: > 18 mm 41 Fr, > 16 mm 39 Fr, > 15 mm, 37 Fr and > 12 mm 35 Fr. This is specifically for Mallinckrodt tubes and one study which tested this predictive sizing didn't prove (in my view) it to be very useful.

Left or right DLT

A lively debate continues [1,2]. A right tube may be more expensive, is harder to place so that the right upper lobe is patent, requires fibrescopic checking and may not be possible to place when airway anatomy is abnormal. Early work detailing problems with right tubes has now been followed by studies suggesting that a right DLT placed and checked by fibre-endoscopy has no increased risk of complications. The indications for a right-sided tube are:

- Tumour in left bronchus
- Left pneumonectomy
- Left lung transplantation
- Left tracheobronchial disruption
- Left bronchial stent in situ
- Distorted left bronchial anatomy

Airway trauma due to DLT

A recent review article [3] collected data from the world literature. Between 1972 and 1998, only 33 reports were identified. Reports involved 32 patients with red rubber DLTs and 14 patients with PVC DLTs. This may well just reflect the differing usage of these types of tubes. Authors were contacted to try and ascribe a cause for the airway trauma. A carinal hook, tube-tip irregularities, asymmetric cuff inflation, initial overinflation of the cuff and nitrous oxide induced cuff overinflation were the most common lessons. The bronchus was the most frequent site of injury, followed by tracheal damage followed by tracheo-bronchial tears. Almost half the published reports with PVC DLTs involved Japanese patients. Good practice recommendations to avoid airway trauma are:

- Choose the largest PVC DLT that will fit
- Remove the bronchial 'stylet' once the tip of the tube is past the vocal cords
- Advance the DLT the appropriate distance (based on height)
- Inflate both cuffs slowly and carefully
- Use a 3ml syringe to inflate bronchial cuff
- If nitrous oxide used – fill cuffs with saline or N_2O/O_2 mix
- If nitrous oxide used check cuff pressures intermittently
- Keep intracuff pressures < 30cm H_2O
- Deflate both cuffs before moving or repositioning tube
- Deflate bronchial cuff when not needed

Checking DLT position

It is firmly established that the positioning of a DLT should be by visualisation of chest movement, auscultation, fibre-endoscopy[4] and a suitable pressure/volume/flow profile. A standard intubating fibrescope with nominal diameter 4mm will not pass down a 35 Fr and passes with difficulty down a 37 Fr DLT. Generally these smaller tubes are used in females: This gender discrimination should be overcome by purchasing a fibrescope designed for checking DLT e.g the Olympus LF-DP with a diameter 3.1mm. The sequence of checking is to pass the fibrescope down the tracheal limb to check that the correct bronchus has been intubated and to make certain that it is the correct depth. The bronchial limb of a right tube should always be checked to make certain that the slit in the bronchial cuff is opposite the right upper lobe bronchus. The right upper lobe has no constant anatomy and one should at least consider the desirability of inspecting the airway fibreoptically to make certain that the anatomy will suit a DLT. This can be done by passing the DLT through the vocal cords but not advancing beyond mid-trachea. The fibrescope is placed through the bronchial limb and

is advanced to inspect the anatomy and to guide the tube into position.

Bronchial blockers

Bronchial blockers are making a comeback particulary in the US (see Univent Tube on www.vitaidltd.com/Univent.htm or Arndt Endobronchial blocker set on www.cookgroup.com/cook_critical_care/blocker.html). A blocker is effectively a balloon on a suction catheter and is placed under direct or fibreoptic vision into the bronchus. The trachea is intubated with a normal single lumen tube and the blocker may pass within or outside the tracheal tube. When the blocker is inflated, that lung will not be ventilated. The 'suction catheter' element passing to the tip allows the isolated lung to deflate, or suctioning. A blocker might therefore be used instead of a DLT in a normal patient, be used when it would be difficult to insert a DLT (difficult intubation or paediatric) or when the airway anatomy is not suitable for a DLT. A blocker may also allow continued inflation of one lobe of the operated lung, for example if placed in the right bronchus intermedius, will allow right upper lobe ventilation when the operation is on the lower lobe.

An editorial[5] this year describes the characteristics of the ideal blocker;

● A balloon shape to stabilise it in the bronchus with low pressure/high volume inflation characteristics.

● Be flexible and easy to manipulate into mainstem or lobar bronchus.

● Have a channel for deflation and suction distal to balloon.

● Be adaptable for use internal or external to standard tracheal tube.

● Have a wide variety of sizes for adult and paediatric use.

Tips in the same editorial for insertion of a blocker are:

● Position deflated blocker after intubation when supine

● Particularly useful in left bronchus for left lung deflation

● Push blocker as far distally as possible to avoid migration out of bronchus

● Use preferentially for –'otomies' rather than –'oscopies' because lung deflation is slower

● Use a video camera on the positioning fibrescope to allow an assistant to help. It is a 4 handed task.

One study[6] compared left DLT with left and right bronchial blockers. Left blockers took longer to place but both left DLT and left blocker were satisfactory. Satisfactory lung deflation was obtained in only half of the right blocker cases.

Physiology of one lung ventilation (OLV)

Thoracic surgery is undertaken in the lateral position and the terms non-dependent and dependent lung indicate the operative collapsed lung, and continuously ventilated normal lung respectively. Clinically it is possible to identify four different periods of ventilation - TLV supine; TLV lateral, chest closed; TLV lateral, chest open and OLV. Classic teaching is that in TLV-lateral the dependent lung receives about 60% of the blood flow for gravitational reasons but only 40% of the total ventilation. When OLV is instituted all ventilation goes to the dependent lung and the non-dependent lung blood-flow (40% total) is reduced by 50% through mechanisms including hypoxic pulmonary vasoconstriction. This means that 20% blood flow still passes through the collapsed lung.

Management of hypoxaemia during OLV

● At initiation of OLV the F_1O_2 should be raised at least to 0.5, if not 1.0. This is simple, physiological and effective. The mechanisms diverting blood from the non-dependent lung take time and later in the period of OLV it is possible to reduce the F_1O_2.

● Check the position of the DLT to make certain that all bronchopulmonary segments of the dependent lung are being ventilated.

● PEEP to the lower lung sounds as though it should be effective and fits in with respiratory care given in ITU, but it isn't reliably effective in thoracic surgery and may cause the PaO_2 to fall. An excellent paper[7] details compliance curves on OLV and raises the likelihood that 'excessive' PEEP may produce deleterious increases in pulmonary vascular resistance in the dependent lung. Attempts should be made to individualise the ventilation of the dependent lung by considering tidal volumes, rate of ventilation, I:E ratios, pressure/volume controlled ventilation and amount PEEP artificially produced by the long, narrow lumens of the DLT.

● CPAP with 100% oxygen to the non-dependent lung is effective but inconvenient to the surgeon particularly in thoracoscopy.

● Manipulation of HPV. This is often cited by trainees as a most important factor but Conacher[8] argues coherently against the notion that anaesthetic agent induced failure of HPV is an important or even evident process in clinical anaesthesia in humans.

Hypoxic pulmonary vasoconstriction[9]

HPV was first described in 1946 by Von Euler and Liljestrand who found that when cats breathed an F_1O_2 of 0.105 the pulmonary artery pressure increased. Sympathetic or parasympathetic blockade did not affect this response, and one physiological explanation is that it derives from a direct effect of oxygen on pulmonary arterioles. HPV response is a function of both alveolar *and* mixed venous oxygen tensions and experimental elevation of mixed venous PO_2 above 13 kPa abolishes the process. When the lung is collapsed during OLV it is suggested that HPV decreases the blood flow to that lung by 50%.

Effect of anaesthetic agents on HPV

Early animal work demonstrated a marked (clinically relevant) inhibition of HPV by inhalational but not intravenous agents. It sounds credible that anaesthetic agents with a vasodilatory property would antagonise a physiological process involving vasoconstriction. However, current work supports at worst only a very modest (clinically borderline) impairment of HPV by modern inhalational agents. This revision has been supported by

various criticisms of previous work. An agent may impair the effects of alveolar hypoxia but any reduction in cardiac ouput would reduce mixed venous oxygen and promote HPV. A recent paper[10] examines the effects of increasing isoflurane or desflurane on various parameters during OLV in intact pigs and is worth reading to understand the difficulties of interpreting data. In humans, general anaesthesia with isoflurane is associated with a higher shunt fraction than propofol during OLV, but the prevalence of hypoxemia is similar. Recently[11] no difference in shunt fraction between sevoflurane and propofol could be demonstrated in humans during OLV.

Modification of pulmonary blood flow during OLV

Two approaches are to reduce PVR in the dependent lung or increase PVR in both (or a combination). Inhaled nitric oxide is a potent, short acting pulmonary vasodilator which is supplied only to the dependent ventilated lung during OLV and to regions of high ventilation within that lung. It has been studied in several studies and interestingly has little effect on oxygenation except perhaps in those patients with a low PaO_2 pre NO treatment. A specimen paper[12] investigated the effects of 40 ppm NO on 30 patients undergoing OLV in the standard lateral position. Mean shunt fraction increased from 14% during TLV to 42% with OLV but was not affected further by NO. Mean PaO_2/F_iO_2 (mm Hg) values decreased from 420 on TLV to 170 on one lung but did not change with NO. In patients with a shunt fraction > 45% on OLV, PaO_2/FIO_2 value increased from 84 to 104 with NO. Nitric oxide is a toxic agent and alternatives[13] are being investigated.

In humans[14] almitrine, an intravenously administered pulmonary vasoconstrictor available in France, prevents or limits OLV induced decreases in PaO_2 (this effect had previously been documented in a dog model). In patients anaesthetised with propofol/sufentanil and ventilated with 100% oxygen, an almitrine infusion at 8mcg/kg/min was started at initiation of OLV. With placebo the arterial oxygen tension fell from 430 to 178mmHg over 30 minutes, but with almitrine it fell only to 325mmHg. No changes in CO or PVR were seen with this dose of almitrine.

Pain relief following thoracotomy

A good editorial[15] a year ago considered the topical issues. There is as much debate[16,17] about the use or need for epidural analgesia as in other areas of major surgery. One particular local technique with a good record is paravertebral block insituted either by the anaesthetist percutaneously (see www.nysora.com for technique) or most easily with the catheter placed by the surgeon at the end of surgery. We have used this latter technique at Guy's/St Thomas' for many years, infusing lignocaine 0.5% at 10ml/hr continuously for 48hr on the ward. It is supplemented by NSAID and PCA morphine. Advocates[18] suggest that paravertebral blockade may be more effective with a lower complication rate than epidural.

Post lung resection morbidity/mortality

About 15% - 40% patients develop an arrhythmia which is usually atrial fibrillation, (usually) on the second or third postoperative day. This is more common in older patients[19]and is associated with a longer hospital stay and increased mortality. Various papers have suggested that other factors associated with the development

of AF are ischaemic changes on the ECG, cardiac enlargement, abnormal preoperative exercise test, intraoperative hypotension, postoperative pulmonary oedema, right sided operation and pneumonectomy. It may be indicative of poor cardiovascular reserve. Avenues of research are whether this represents sympathetic stimulation or raised pulmonary artery pressures. Relative sympathetic stimulation may result from intraoperative damage to parasympathetic pathways. A study[20] comparing the effect of bupivacaine with morphine thoracic epidural analgesia on the prevalence of arrythmias in 50 patients showed that bupivacaine was associated with a lower incidence and duration of arrythmia than morphine. Postoperative administration of oxygen[21] to the third postoperative day did not affect the incidence of AF.

The overall mortality rate after pneumonectomy is 7-12% in large[22] or national studies. The causes being pneumonia, sepsis, cardiac causes, DVT/PE etc. A specific problem is post-resection pulmonary oedema. This is more common, and more serious, after pneumonectomy (2-4%) than lobectomy. Criteria are the combination of clinical respiratory distress and hypoxaemia, with diffuse shadowing on CXR in the absence of cardiac dysfunction, pneumonia, sepsis or aspiration.

Slinger provides a thoughtful review[23] of the condition and lists what is 'known':

● Symptoms on postoperative day 2-4

● Radiographic evidence at least 24 hr prior to clinical signs

● More common after right than left pneumonectomy

● High mortality (> 50%)

● Resistant to standard 'pulmonary oedema' treatment

● Associated with fluid overload but not clear cause-effect

● Histological picture of ARDS

● Associated with normal low PAOP and high protein oedema fluid

Alvarez[24] describes the onset and management of the condition in 5 patients. It is clear that overzealous fluid administration is not the cause but may exacerbate the condition. It appears to be due to capillary endothelial leak. Are steroids of use?

Data from the management of ARDS patients suggests that volutrauma may be an important adverse factor. Perhaps OLV should be pressure rather than volume controlled.

Internet resources and thoracic anaesthesia

An article[25] lists many sites. A starting point is the 'related sites' link at the Journal of Cardiothoracic and Vascular Anaesthesia on www.jcardioanesthia.com

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