MALAYSIAN SOCIETY OF ANAESTHESIOLOGISTS

Year Book 2011/2012

MALAYSIAN SOCIETY OF ANAESTHESIOLOGISTS YEAR BOOK 2011/2012

CONTENTS

Foreword

It is with pleasure that the Malaysian Society of Anaesthesiologists (MSA) publishes its fourth issue of the Year Book 2011/2012 themed "*Subspecialties in Anaesthesia*"

As other issues have done in the past, this Year Book continues to promote and showcase the progress in professional development within our fraternity. The interest towards subspecialties is growing and its importance cannot be understated. Therefore, it is indeed welcoming that this Year Book has chosen to highlight the growing interest of our fraternity towards subspecialties. It has been and will continue to be the goal of the MSA to encourage our diverse group of members to march forth and keep abreast with recent developments in our field. Through this we stand fortified in knowledge.

It is delightful that this Year Book is released in conjunction with the World Anaesthesia Day. We, as a society and a group of professionals, should take pride that the world and globe celebrate us. So I urge one and all of you to commemorate the day with pride and recognition. What better way than to showcase our academic strength and literary contribution via this issue.

I wish to thank the contributors for their effort and time to make this issue a reality. I appreciate the determination and the perseverance in seeing your contribution to publication. It is no secret that our fraternity is at its developmental stage in reference to publications and research. It is through these projects that the MSA seeks to encourage the determination and confidence of our members in the field of research.

To the respected reviewers, a sincere appreciation for the guidance and efforts to navigate this book to success. You are the torch bearers and once again your work towards keeping the standard of this book is sublime.

The efforts put forth by our determined editors are very evident. In spite of the tight deadline and the numerous obstacles, they have succeeded in their quest of seeing this book published.

I am sure the end product makes the experience more than worthwhile.

I trust our members will benefit from this Year Book and I congratulate all involved for a job well done.

Datin Dr V Sivasakthi
President
Malaysian Society of Anaesthesiologists

Preface

The theme for this edition of the MSA Year Book 2011/2012 is "*Subspecialties in Anaesthesia*". In choosing this theme we acknowledge the fast changing tapestry of our specialty that has kept abreast with the medical development and trend of time.

The journey from the start to the finishing line for this edition of the MSA Year Book 2011/2012 has never been short of a dull moment. From onset of the project, it has been a race against time to complete this assignment and to publish this book in time for the National Anaesthesia Day Celebration in 2012. And voila it is finally here!!

It is with immense pleasure and pride that we exhibit a showcase of very young and talented contributors to the Year Book. This Year Book has seen the toil of a very determined and obliging group of writers who have raced along with us to see this book to completion. It is indeed the very hallmark of pure determination and it is the will of these writers that bear testament to this publication.

One must not over look the contributions of the esteemed group of reviewers. These individuals as custodians to the quality of the Year Book, rallied with suggestions and ideas to compliment the articles reviewed by them. It is with promptness, patience and bountiful guidance that the articles are deservingly published.

Every quest completed renders one wiser. We, as editors, have been humbled from this experience of the many accounts and obstacles along the way. We sought solace with the fortitude and the determination of the majority of the novice contributors. We found strength from the reviewers who painstakingly attempted to steer this edition to success. We developed confidence working together as a team.

So, as the year draws nearer to its end and the new year emerging, let's learn to celebrate ourselves as Anaesthesiologists and let's learn to showcase ourselves with pride to our fraternity, country and the world. Through the publication of this Malaysian Society of Anaesthesiologists' Year Book, may we continue to garner interest and strength. Let us celebrate with the release of this edition in conjunction with the WORLD ANAESTHESIA DAY with pride.

Dr Shireen Sree Jayan
Dr Vanitha Sivanaser
Co-Editors
MSA Year Book 2011/2012

Acknowledgements - Reviewers

We would like to acknowledge the contributions of the following reviewers:

Dr Anuradha Pathmanathan
Prince Court Medical Centre

Dr Azrin Mohd Azidin
Hospital Kuala Lumpur

Professor Dr Chan Yoo Kuen
University Malaya Medical Centre

Dr Hari Krishnan
Sime Darby Medical Centre Ara Damansara

Dr Felicia Lim Siew Kiau
Hospital Kuala Lumpur
Universiti Kebangsaan Malaysia Medical Centre

Professor Dr Lim Thiam Aun
Universiti Putra Malaysia

Dr Lim Wee Leong
Hospital Sungai Buloh

Dr Ling Kwong Ung
Sime Darby Medical Centre Ara Damansara

Professor Dr Marzida Mansor
University Malaya Medical Centre

Associate Professor Dr Mohd Basri Mat Nor
International Islamic University Malaysia

Dr Raveenthiran a/l Rasiah
DEMC Specialist Hospital Shah Alam

Dr Shahridan Mohd Fathil
Alexandra Hospital, Singapore

Dr Thong Chwee Ling
Kuala Lumpur Sports Medicine Centre

Ultrasound Guided Regional Anaesthesia - Study on Dual-View Technique for Anterior Visualization of The Sciatic Nerve

Azrin Mohd Azidin, Amiruddin Nik Mohamed Kamil, Mohd Salleh Abdul Samad, Shahridan Mohd Fathil
Department of Anaesthesiology and Intensive Care, Hospital Kuala Lumpur, Malaysia

ABSTRACT

Background: Identifying sciatic nerve from anterior thigh is difficult due to its depth, inconsistent surface anatomy and its location posterior to the femur. In this study we evaluated the ease of identification of sciatic nerve using 'dual view' technique in comparison with 2 other techniques.

Objective: To evaluate the identification rate of anterior approach to the sciatic nerve using the short-axis view at the proximal third, mid-thigh and using 'dual view' technique

Methods: After approval from Institutional Review Board , 52 anterior thigh sonograms were evaluated in patients who consented for this procedure. This was a study to compare sonographic identification rates of sciatic nerve using conventional anterior thigh transverse view scanning at proximal third (technique 1) and mid thigh (technique 2), with a 'dual view' technique (technique 3) which involves using both transverse and longitudinal axis views to pinpoint the sciatic nerve.

Results: Anterior visualization of the sciatic nerve using transverse axis scans at the mid thigh were found to have higher identification rates compared to the scans over the proximal thigh (82.7% v 73.1%). 'Dual view' technique yielded the highest visualization rates among the 3 techniques (92.3%). Mean skin to nerve distances were 7.15 ± (1.54) cm, 6.75 ± (1.57) cm and 6.37 ± (1.36)cm with proximal thigh transverse scan, mid thigh and 'dual view' technique respectively. Mean femur to nerve distances was 4.52 ± (0.75) cm, 5.04 ± (0.67) cm and 4.77 ± (0.60) cm with similar techniques.

Conclusion: 'Dual view' technique gives a higher percentage of success in identifying the sciatic nerve using the anterior approach compared to conventional transverse view scan.

Anterior approach to the sciatic nerve has several advantages over the posterior or lithotomy techniques. With the anterior approach (classical approach by Beck, or modified Chelley-Delaunay technique)[1,2] the block can be performed with the patient in the supine position, particularly beneficial in patients who have compromised mobility due to trauma, arthritis or obesity.[2] Both the sciatic and the femoral nerve can be blocked without needing to change position. Identifying sciatic nerve from the anterior thigh is difficult due to its depth, inconsistent surface anatomy and its location posterior to the femur. Internally rotating the femur may help expose the nerve, so that it is made amenable to approaches from the anterior, at or above the level of the lesser trochanter.[3] Both techniques (classical approach by Beck, or modified Chelley-Delaunay technique) are equally difficult technically and as a result, the anterior approach is the least popular of the three techniques to block the sciatic nerve.[4]

This anterior approach is made slightly easier with the use of ultrasound, as the nerve can be "seen" and traced along its course provided it is correctly identified.[5] The landmarks of importance are the adductor groups and the hamstrings, distinguished by the different echo texture of the fibres, and also the lateral intermuscular septum.[6] However, as the muscle groups in this region are relatively thick, the sciatic nerve, which supposedly is hyper-echoic and oval to circular in shape,[7] appears thinned

out. With the background of thick muscles with fascia layers, the flattened sciatic nerve can be difficult to identify in the transverse view, especially at the proximal thigh.

The ultrasound guided technique to block the sciatic has been described as the proximal thigh, and the mid-thigh approach. Using a curved low frequency transducer, the sciatic nerve is viewed at its short-axis. Three techniques to identify the nerve have been illustrated in one of the many regional anaesthesia websites - www.neuraxiom.com:

The 'big triangle' technique
The sciatic is situated as the third point of a triangle, straight down from the femoral vessels, whereby the vessels and the femur are the other two points of the triangle.

The 'small triangle' technique
The sciatic is the apex of a small triangle, bounded medially by the semitendinosus and semimembranosus muscle, beneath the adductor group.

'Flashing the adductor' technique
With the use of nerve stimulator, and advancing the stimulating needle in Doppler mode, the adductor group of muscles are displayed as a flashing 'indicator light'. As the stimulating needle bypasses the adductor magnus and goes into the fascia containing the sciatic nerve, the 'flashes' will disappear-indicating the close proximity of the sciatic nerve.

We have observed that scanning the sciatic nerve in its long-axis view, will help to correctly identify the depth of the sciatic nerve, especially if it is difficult to confirm in the transverse view. The probe can be placed antero-medially to the femur along the length of the thigh, and the nerve will appear as a distinctly continuous hyper-echoic structure with a characteristic fascicular pattern. When the structure is followed distally, it will be seen to diverge into its two branches. This 'diverging highway' appearance will help indicate the point where the sciatic nerve gives its common peroneal and tibial branches. The probe can be brought back proximally before the point of divergence, and the nerve can be blocked at any point proximally throughout its course in the thigh, in its transverse view.

We have seen that with this 'dual view' technique, the sciatic nerve may be easier to identify, and can act as a confirmatory maneuver in cases where the sciatic is not well defined.

OBJECTIVE

To evaluate the identification rate of the anterior approach to the sciatic nerve using the short-axis view at the proximal third, mid-thigh using 'dual view' technique.

METHODOLOGY

A thigh sonogram of 52 patients, were obtained in General Operation Theatre of Hospital Kuala Lumpur, after explanation and consent was taken, prior to surgery.

INCLUSION CRITERIA

All preoperative patients with American Society of Anaesthesiologist (ASA) Classification I - 3, and post operative cases with Post Operative Recovery Score of 6 who consented for procedure.

EXCLUSION CRITERIA

All patients without consent and patients aged less than 12

Patients were positioned supine with the thigh to be imaged exposed up to the inguinal region. An anaesthesiologist who has done at least twenty

anterior scanning of the thigh, will conduct this procedure. In cases of uncertainty, opinion of another anaesthesiologist with at least the similar ability is sought. The thigh was divided into three regions, the proximal thigh, mid thigh and distal thigh. To visualize the sciatic nerve, a curved-array low frequency transducer (M Turbo, Sonosite) was placed at the proximal third in its transverse view (technique 1) and then at the mid thigh region in its transverse axis (Technique 2). The nerve was seen as an oval or elliptical hyper-echoic structure deep to the adductor group of muscles. The depths from the skin and the distance of the sciatic nerve to the femur were recorded. Images seen on any part in that divided area were documented according to it designated thirds.

The ultrasound transducer was then rotated 90 degrees, to image the sciatic nerve in its longitudinal axis. Confirmation was by the appearance of continuous hyper-echoic structure with a characteristic fascicular pattern, which diverges as the probe was moved distally (Diverging Highway sign). Once this was seen, the probe was moved back to the point before this divergence, traced until the most proximal point where the image can be identified clearly, and was then rotated back to visualize the sciatic nerve in its transverse axis (Technique 3). The distance to the femur, and its depth to the skin at this point was recorded.

Other data recorded were demographics, weight, ASA classification and co-morbidity. Statistical analysis of the compiled data was done via SPSS software version 12:0

RESULTS

49 were sonogram of male thighs and 3 were female. 31 patients were Malay, while 11, 4 and 6 were Chinese, Indian and other races respectively (chart 1). Mean age, weight and ASA classification are as illustrated in Table 1.

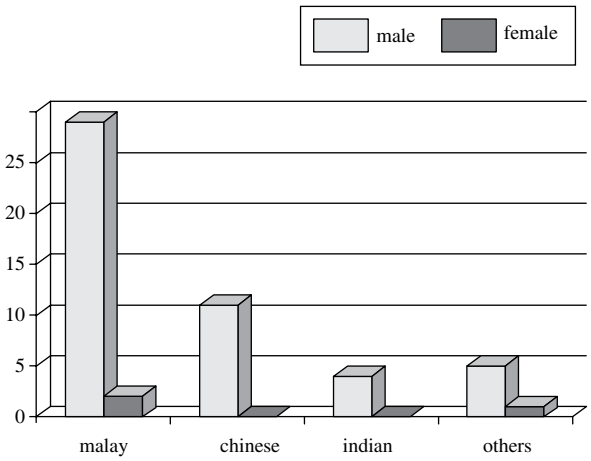


CHART 1: Chart showing number of patients, in relation to race and gender

TABLE 1: Table showing age, weight and ASA classification of patients

Age (years)	39.9 ± 18.1
Weight (kg)	63.0 ± 13.0
ASA Classification	
ASA 1	42 (80.8%)
ASA 2	9 (17.3%)
ASA 3	1 (1.9%)

Mean distances of sciatic nerve to skin (skin to nerve) and to femur (femur to nerve) using short axis view at proximal thigh (PT), mid thigh (MT) and at the most proximal point the sciatic nerve is seen using dual view technique are shown in Table 2. At proximal thigh, the mean skin to nerve distance was 7.15 ± 1.54 cm and the femur nerve distance was 4.52 ± 0.75 cm. Using short axis view at mid thigh region, the mean distances were 6.75 ± 1.57 cm and 5.04 ± 0.67cm respectively. With dual view technique, the sciatic nerve could be seen at mean distance of 6.37 ±1.36 cm from skin, and 4.77 ± 0.60 cm from femur.

TABLE 2: Table showing skin-nerve and femur-nerve distances from various techniques, and numbers (percentage) of sciatic nerve detection

Location of ultrasound probe (view)	Skin-nerve distance (cm) ± SD	Femur-nerve distance (cm) ± SD	Number of sciatic nerve detection
Proximal thigh (short-axis view) - Technique 1	7.15 ± (1.54)	4.52 ± (0.75)	38 of 52 (73.1%)
Mid thigh (short-axis view) - Technique 2	6.75 ± (1.57)	5.04 ±(0.67)	43 of 52 (82.7%)
Dual view technique (short axis with long-axis view) at its most proximal site -Technique 3	6.37 ± (1.36)	4.77 ± (0.60)	48 of 52 (92.3%)

The sciatic nerve was able to be identified in 38 out of 52 sonograms at the proximal thigh, giving a detection rate of 73.1% with the transverse/short axis view. Using short axis view at mid thigh region, 43 sciatic nerves were successfully identified, giving an identification rate of 82.7%. However, dual view technique yielded the highest identification rate of 92.3%, out of 52 thigh sonograms.

As for co-morbidity, most of the cases were ASA 1 patients, with nine (9) ASA II. Patients with ASA II mostly have hypertension and or diabetes with or without neuropathy. Only one (1) patient was classified as ASA class III.

There were four patients in whom the sciatic nerves were not identifiable at all with either of the views. One patient had hypertension, diabetes and cerebro-vascular injury, while another patient has diabetes and chronic limb ischaemia. Another patient only had hypertension alone, while another patient was a healthy ASA I, 36 year old patient.

DISCUSSION

Regional block of the sciatic nerve using the anterior approach is made feasible with the use of ultrasound.[8,9] Quoted success rates have been documented to be the same as the posterior approach.[9] However, identifying the sciatic nerve can be difficult anteriorly due to its location behind the femur and its anisotropic appearance, especially in the proximal third (Figure 1). Visualizing the sciatic nerve in the middle of the thigh is made slightly easier as its hyper echoic shadow stands out in the background of relatively hypo echoic muscle fibres (Figure 2). Our study showed that the identification rate based on appearance alone is higher, when visualizing the nerve using the transverse view in the mid-thigh region compared to the proximal third.

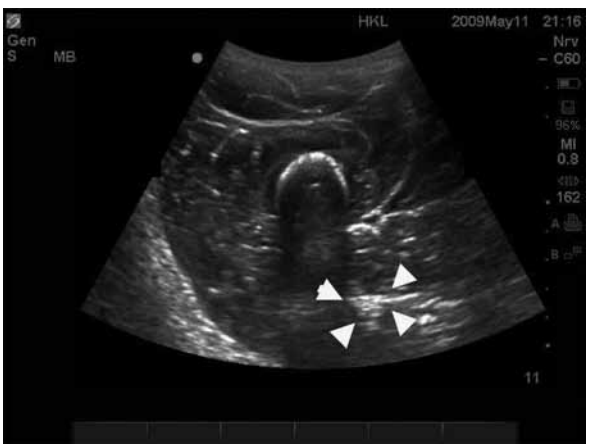


FIGURE 1: Transverse view of the sciatic nerve in the anterior thigh scan at the proximal third

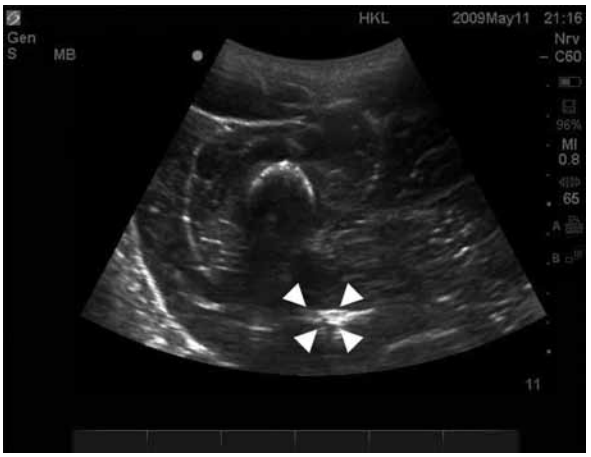


FIGURE 2: A transverse view of the sciatic nerve in the anterior thigh scans at the mid thigh

However, using 'Dual view' technique, the identification rate was found to be the highest compared to the two previous techniques. Confirmation of the sciatic nerve under ultrasound, can be made by its continuous appearance, and also as it diverges distally along its course towards the popliteal fossa ('Diverging Highway' sign-Figure 3). This technique can be used as an extra maneuver to confirm the location of sciatic nerve, besides scanning in the transverse view at either of the two points (Figure 4). Local anaesthetic can be deposited at the most proximal point seen; hence more sensory areas will be covered for block.

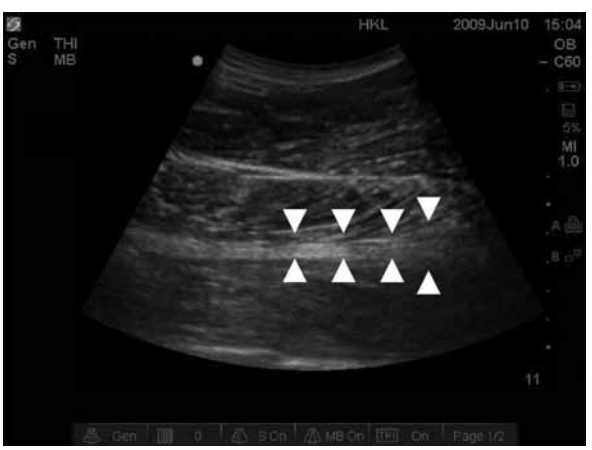


FIGURE 3: A longitudinal view of the sciatic nerve with the transducer over the antero-medial aspect of the thigh. Distally, the sciatic nerve can be seen to diverge to its branches ('Diverging Highway' sign)

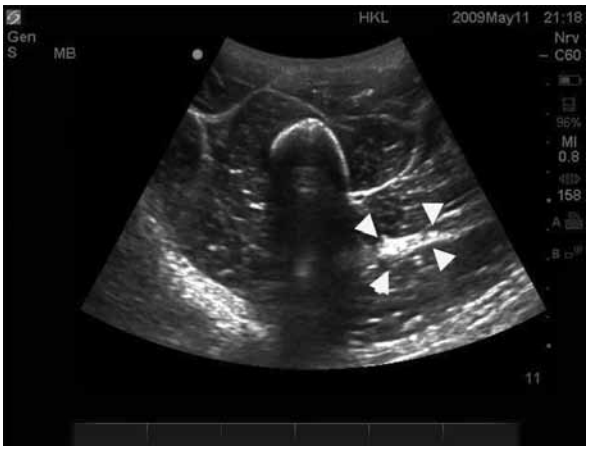


FIGURE 4: A transverse view of the sciatic nerve in the anterior thigh scans at the proximal thigh using 'dual view' technique. The sciatic nerve was viewed in a longitudinal axis first before having the transducer rotated to view the sciatic nerve in its transverse axis

In our observation, the sciatic nerves of 2 sonograms were wrongly identified during transverse scan at the proximal third region. After using dual-view technique, they were found not to be the sciatic nerve. Among the three techniques of visualizing the sciatic nerve anteriorly, this 'dual view' technique was found to have the highest identification rate, with the mid-thigh transverse view being the second most successful. The proximal thigh transverse view was the least successful technique in correctly identifying the location of the sciatic nerve anteriorly based on appearance alone.

There were no distinct relationship in the distances between skin to nerve, and femur to nerve among the three techniques. The mean depths for the three methods were 7.15 cm, 6.75 cm and 6.37 cm respectively. It was expected that the depth (skin to nerve) at the mid-thigh would be more than the proximal third, as the nerve comes out from the pelvis and lies superficially in the popliteal fossa, but this was not found to be the case. This could be due to the fact that, at the mid thigh region, the sciatic nerve would have coursed itself directly beneath the femur, and to visualize it requires the transducer to be moved medially. Shorter skin

to nerve distances could be found because of the elliptical cross-sectional shape of the thigh.

Mean distance of the sciatic nerve using 'dual view' technique was also found to be the shortest compared to the other two techniques. This observation can be explained by the way in which the distances were measured from the ultrasound screen. Measurements of the distances were taken from the center of the screen, to the superior-most part of the transverse view (short-axis view) of the sciatic nerve. As the transducer was placed antero-medially on the thigh and due to the cross-sectional shape, the distance can be seen to be smaller compared to its antero-posterior distances. It is unclear whether the shorter mean distance from the skin is the reason why the 'dual view' technique apparently has a higher success rate. We have had success in identifying the nerve using this technique even though it is deeply seated. Another reason for its higher success rate is that the additional maneuver, which is visualizing the nerve in its longitudinal axis, act as a validating sign which suggests the targeted structure which we thought could be the sciatic nerve, is indeed correct.

The number of cases was relatively small to look into the relationship between co-morbidities in relation to the ease of identification. Only 3 patients were female and no analysis of ease of identification was made for weight or body mass index. Most of the patients recruited were ASA I cases, and in almost all patients, the sciatic nerve can be seen using any of the three techniques, except for 4 patients. One patient had bilateral below knee amputation done secondary to ischaemic neuropathy and in this case, the nerve was not seen. In another patient, the sciatic nerve could not be identified even though he had no co-morbidity. In view of this small number, any correlation could not be made.

A combination of the short axis and longitudinal view will lead to a higher success rate in visualizing the sciatic nerve. With this approach, the depth of the nerve can be identified along its course, and the injectate can be deposited as proximal as possible covering a larger sensory block distribution. Another advantage of this technique is that, the difficulty in identifying the sciatic nerve in its transverse view alone can be minimized as this technique can also be used as a confirmatory maneuver to ascertain its location deep to the structures.

CONCLUSION

Based on our findings, we recommend the use of 'dual view' technique to visualize the sciatic nerve for regional block via the anterior approach.

References

1. Beck GP. Anterior approach to sciatic nerve block. *Anesthesiology* 1963;**24**:222-4
2. Chelley JE, Delaunay L. A new anterior approach to the sciatic nerve block. *Anesthesiology* 1999;**91**:1655-60
3. Vloka JD, Hadzic A, April E, Thys DM. Anterior approach to the sciatic nerve block: the effects of leg rotation. *Anesthesia Analgesia* 2001;**92**:460-2
4. Hadzic A, Vlodka JD, Kuroda MM, Koorn R, Birnbach DJ. The practice of peripheral nerve blocks In the United States: A national survey. *Reg Anesth Pain Med* 1998;**23**: 241-6
5. Alon YBA, Bigeleissen P, Chelly JE. Ultrasound guided antero-medial sciatic approach. *Anesthesiology* 2008;**109**A340
6. Barrington MJ, Lai SL, Briggs CA, Ivanusic JJ, Gledhill SR. Ultrasound guided midthigh to sciatic nerve block: a comparison with the posterior approach. *Anesth Analg* 2009 Feb;**108**(2):660-5
7. Tsui BCH, Ozelsel TJP. Ultrasound-Guided Anterior Sciatic nerve Block Using a Longitudinal Approach: "Expanding the View". *Reg Anesth Pain Med* 2008 May-June;**3**(33):275-6
8. Chan VW, Nova H, Abbas S, McCartney CJL, Perlas A, Xu DQ. Ultrasound examination and localization of the sciatic nerve: A volunteer study. *Anesthesiology* 2006;**104**:309-314
9. Ota J, Sakura S, Hara K, Saito Y. Ultrasound-guided anterior approach to sciatic nerve block: a comparison with the posterior approach. *Anesth Analg* 2009 Feb;**108**(2): 660-5

Anaesthesia for Thoracic Surgery: A Brief Summary

Noorjahan Haneem binti Md Hashim
Senior Lecturer, Universiti Malaya
Clinical Specialist, University Malaya Medical Centre

Anaesthesia for thoracic surgery presents management problems even to most experienced anaesthesiologists. The patients may present at any age, with multiple co-morbidities, poor cardio-respiratory reserves from the presenting illness and for procedures ranging from diagnostic to therapeutic.

The aim of this article is to provide anaesthesiology trainees with a guideline of common management of such patients undergoing thoracic surgery. The discussion will be divided into pre-operative, intra-operative and post-operative management.

PREOPERATIVE MANAGEMENT

The main aims of the pre-operative management for thoracic surgery are pre-anaesthetic assessment, risk-stratification and optimisation.

Pre-anaesthetic assessment
A thorough history and detailed physical examination could not be more important in these patients. In addition to the assessment of the patients' co-morbidities, cardio-respiratory status and smoking history, it is important to assess the effects of the presenting illness ie intrathoracic pathology on the patients. Therefore, it is important for the anaesthesiologists to understand how these pathologies present and their impact on the patients' management.[1]

Symptoms like cough, sputum, chest pain, dyspnoea and wheezing are recognised as *bronchopulmonary symptoms*.[1] These symptoms arise from bronchial irritation, ulceration, obstruction, infection distal to the obstruction or a combination of these causes.

Patients with tumours extending beyond the lungs may present with orthopnoea, expiratory wheeze (tracheal compression), pleural effusion (pleura), pain (chest wall or pleura), dysphagia (oesophagus), superior vena cava obstruction (superior vena cava), hoarseness of voice (recurrent laryngeal nerve), Horner's syndrome and arm pain and weakness (brachial plexus). These symptoms are classified as *extrapulmonary intrathoracic symptoms*.[1]

Presence of paraneoplastic syndromes (*extrathoracic non-metastatic symptoms*)[1] are due to secretions of hormones or hormone-like substances by pulmonary tumours.

Non-specific symptoms associated with chronic illnesses and malignancy such as loss of weight and appetite, lethargy, anaemia should be elicited and optimised.

Symptoms of extrathoracic metastasis should be elicited and investigated as they would alter the course of patients' management.

Another important aspect of assessment is the assessment of the airway. In addition to the standard assessment of ability to bag mask ventilate and intubation, emphasis needs to be placed in the assessment of airway patency distal to the larynx. Patients may present with extrapulmonary intrathoracic symptoms. The position patient feels most comfortable in should be elicited and documented in the presence of a mediastinal mass.

Radiological assessment (chest radiograph and CT scan of the thorax) is extremely important as it will impact our induction method, choice of airway devices and allocation of resources.

Risk stratification
Another important part of pre-operative assessment is risk stratification-the prediction of post–operative mortality and morbidity (pulmonary and non-pulmonary).

The Thoracoscore[2] is a relatively new validated[3] scoring system that predicts in hospital **mortality** after thoracic surgery which only uses nine

variables: age ≥65, sex (male), dyspnoea score ≥3, ASA status ≥3, WHO performance status classification ≥3, priority of surgery (emergency vs elective), diagnosis group (malignancy vs non-malignancy), procedure class (pneumonectomy vs non-pneumonectomy), presence of ≥3 co-morbidities (including smoking, history of cancer, hypertension, chronic obstructive airway disease, heart disease, diabetes mellitus, peripheral vascular disease, obesity).

Factors that increase the risks of pulmonary complications are age ≥70 years,[5,6,7] smoking,[5,6] emergency surgery,[5] chronic lung disease,[6,7] BMI ≥30,[6] ASA ≥3,[6,7,8,9] renal failure,[7] poor nutritional status,[7] prolonged anaesthesia and surgical[7,8] times, and the need for postoperative ventilation of >48 minutes.[8]

Further prediction of postoperative pulmonary complications can be done via three related but independent assessments,[9] gold standards are marked *:

i) respiratory mechanics: spirometry: patients with decreased FEV1,[6] predicted postoperative FEV1*[6, 9]

ii) lung parenchymal function: blood gases (PaO2<60, PaCO2>45),[9] gas exchange capacity via diffusion capacity of carbon monoxide, DLCO*,[4,6,9] and

iii) cardiopulmonary interaction: maximal oxygen consumption*, VO2 max <15ml/kg/min,[4,9] incremental shuttle walk test <400 m,[4,6,9] six-minute walk test , 400m,[6] stair climbing test <2 flights.[10]

Pre-operative optimisation

It is very important to optimise the patients' general condition and modifiable risk factors. This requires a multidisciplinary approach, and should involve the patients and their caregivers.

Optimisation of co-morbidities, correction of anaemia at least 24 hours prior to surgery, improvement of nutritional (both weight gain in malnourished and weight loss in obese patients) and exercise tolerance status, weight loss and psychological preparation and motivation are vital aspects.

Four parallel processes are done to optimise these patients' respiratory condition pre-operatively:[1]

- smoking cessation;
- bronchodilators (in patients with bronchospasm-with β-agonists, aminophylline and steroids);
- secretions management by loosening and removal of secretions (airway and systemic hydration, mucolytics and expectorants, antibiotics, postural drainage, cough and physiotherapy);
- preparation for post-operative care (incentive spirometry, encourage all the above, discussion of analgesia options).

INTRAOPERATIVE MANAGEMENT

The aims of intra-operative management are maintenance of normoxia, normocarbia, haemodynamic stability, prevention of lung injury, providing adequate surgical exposure while minimising the need for controlled ventilation, minimising post-operative pulmonary complications and optimising analgesia.

Monitoring[1,11]

All patients undergoing general anaesthesia for thoracic surgery will require standard monitoring (electrocardiogram, non-invasive blood pressure, pulse oximetry, and capnometry), temperature monitoring, monitoring of the inspired gases, especially inspired oxygen fraction. Neuromuscular monitoring, though not mandatory in our country, is recommended to balance the need of providing good surgical conditions and the risk of overdose of neuromuscular blockers.

The use of additional monitors depend on the patients' condition and the nature of the surgical procedures. Patients with cardiorespiratory diseases, obesity, at extremes of age, as well as those undergoing procedures that carry an increased risk of bleeding or needing single lung

ventilation will require invasive blood pressure monitoring, monitoring of intraoperative blood gasses, airway pressures and urine output.

Central venous pressure is usually not monitored as its reading is affected by changing positions, PEEP, and surgical manipulation rather than changes in preload.[11]

Positioning
The patients may be placed in the supine (bronchoscopy, mediastinoscopy, thymectomy) or the lateral (pleurodesis, thorascopy, lung resection) positions. Pressure points are to be padded, intravenous lines accessible and flowing. It is important to remember to check the position of the endotracheal tube after every change in positioning. Care of the dependent brachial plexus and axillary artery as well as the urine catheter are important as well.

Anaesthesia technique
Some procedures (pleurodesis, thoracoscopy, flexible bronchoscopy) may be done under topical or local anaesthesia. Other procedures are done under general anaesthesia.

Induction
Monitoring should be established prior to induction.

Appropriate *airway* equipment should be prepared-masks, oropharyngeal airways, larygoscopes, endotracheal tubes and choice of lung isolation methods[12] (if single lung ventilation is required, see Table 1[13] and bronchoscope (flexible fibreoptic bronchoscope to be used in lung isolation for confirmation of placement, and/or rigid bronchoscope when managing patients with tracheal tumours and anterior mediastinal masses.[14] The role of supraglottic devices in thoracic surgery is currently limited in flexible bronchoscopic examination of the airway,[15] though, using i-gel and with a bronchial blocker has been described.[16]

Patients with potential difficult intubation or ventilation may be induced with with inhaled sevoflurane. Other patients may be induced intravenously with Fentanyl 1-2 mcg/kg and propofol 2-4 mg/kg or thiopentone 4-6 mg/kg with a neuromuscular blocker.

Patients with myasthenia gravis presenting for thymectomy would require dose adjustment of neuromuscular blocker, as low as 10-25% of ED95,[17] preferably, atracurium for its non-organ dependant metabolism.

It is best to withhold neuromuscular blockers in patients with bronchopleural fistula[13] and anterior mediastinal masses.[14]

Maintenance
Anaesthesia can be maintained with either inhalational agents (sevoflurane, desflurane), intravenous agents (propofol, remifentanyl) or a combination of both with muscle relaxation (atracurium or rocuronium boluses).

Procedures that require general anaesthesia are best done with positive pressure ventilation.[18,19] Procedures on unilateral lung or pleura are usually done with one lung ventilation. The best option is IPPV, with PEEP in the dependant lung and CPAP in the non-dependant lung as it reduces atelectasis of the lungs and mediastinal shift which will worsen V/Q mismatch, and prevents lung injury. Lung recruitment during and after one lung ventilation[19] can be done to avoid postoperative atelectasis and lung injury.

Three techniques are commonly used for lung isolation (Table 2). Table 3 show steps in the management of intraoperative hypoxaemia.[18]

POST-OPERATIVE MANAGEMENT

Patients should be extubated as soon as possible (pending haemodynamic stability, gas exchange and mechanics of respiration). Prolonged IPPV stresses suture lines, increases the risk of air leaks and barotrauma and increases the risk of infection.

Oxygen therapy[1] should be continued and weaned off slowly to compensate for the V/Q mismatch from OLV and intraoperative positioning.

Patients at high risk of developing post-operative complications (determined pre-operatively and depending on intraoperative events) are to be managed in the intensive care unit.[20,21]

Transport to the intensive care should adhere to guidelines of transporting other critically patients.[22]

Chest drains should be unclamped during transfer and during IPPV.[22] Care should be taken so that the drainage chamber is below the chest at all times to prevent backflow.[22] The optimal water level is 2cm above the drainage tube.[23] High volume, low pressure suction pumps (at 10 to 20 cmH2O) are used to drain haemothorax.[24]

Monitoring should be directed towards early detection of complications. Common complications are:

Pulmonary:
Respiratory insufficiency caused by oedema, atelectasis, small airway obstruction by secretions or blood, pneumonia or pulmonary oedema. Optimising analgesia, chest physiotherapy, antibiotics, appropriate weaning of oxygen therapy are important measures to reduce the morbidity and mortality.

Bronchial disruption causing bronchopleural fistula and the potentially fatal pneumothorax may occur. Sudden bubbling of chest drains, inadequate gas exchange and haemodynamic instability are signs to be monitored.

Pneumothorax may also occur in the contralateral side due to high ventilator pressures, pleural injury during surgical manipulation, insertion of central venous lines and epidural or paravertebral catheters.

Non-pulmonary:
Haemorrhage[1] may be caused by slippage of suture of major vessels or bleeding from raw surfaces and bronchial and intercostal arteries. The chest drain output is to be monitored closely. Any output of more than 200ml/hr is indicative. As absence of drainage does not preclude haemorrhage as the drains may be blocked, it is also important to monitor the patients haemodynamics and general condition.

Right heart failure[1]:
Major lung resection results in increased right ventricular afterload from the reduction in the cross-sectional area of the pulmonary vasculature leading to right heart failure. Patients with pulmonary complications causing increased pulmonary blood flow and increased pulmonary hypoxic vasoconstriction are at increased risk of developing this complication.

Patients present with increased CVP, arrhythmias and signs of low cardiac output. Treatment includes heart rate and rhythm control, optimising fluid status and inotropic support with control of precipitating factors.

Neural injuries[1]:
Accidental injuries to the phrenic, recurrent laryngeal and vagus nerves may occur intraoperatively. Patients with recurrent laryngeal nerve injury may present with upper airway obstruction post extubation, requiring immediate re-intubation. Patients with phrenic nerve injury will have weaning difficulties despite not having pulmonary complications. Patients with vagus nerve injury may present with bowel atony and ileus.

Analgesia[25,26]:
Options include high thoracic epidural, paravertebral blocks, intercostal nerve blocks, interpleural nerve blocks, intravenous opioids and parenteral or oral NSAIDS. Whenever possible, a multimodal approach is best.

Physiotherapy[27]:
is extremely important in preventing postoperative pulmonary complications. This study shows that physiotherapy commenced in the preoperative period and followed through in the postoperative period markedly reduces these complications.

SUMMARY

The anaesthetic management of thoracic patients is very challenging. A thorough understanding of the patients' condition, the physiology of illness and intervention is very important. The management of these patients require a multidisciplinary approach and patient and carer involvement is very important.

TABLE I: Absolute indications for lung isolation

1. protective isolation: in patients with massive lung haemorrhage or infection
2. control of distribution of ventilation: • bronchopleural or bronchopleurocutaneous fistula • large cysts/bullae • major bronchial disruption
3. unilateral lung lavage
4. video assisted thoracoscopy

TABLE II: The advantages and disadvantages of each technique of lung isolation

	DLT	**Bronchial blocker**	**Endobronchial intubation**
advantages	• Allows separation and isolation • Rapid conversion from one lung ventilation to two lungs ventilation and vice versa • Allows bilateral suction • Allows CPAP to non-dependant lung	• Easier insertion • Less problems with displacement during patient positioning • No need for tube change postoperatively • Allows deflation of a specific lobe	• Easy and quick intubation
disadvantages	• Contraindicated in endoluminal tumours • Relative contraindication in difficult airway • Aspiration risk • Changing to single lumen ETT post-op may be difficult	• Slow inflation/deflation • Bronchial blocker lumen easily blocked • Difficulty in suctioning the blocked lung • High pressure cuff	• More likely to block upper lobe bronchi • Limited suction of operated lung • Unable to ventilate operated lung

TABLE III: Management of intra-operative hypoxia

1. Increase FIO2 to 1
2. Call for help
3. Check connections, oxygen supply
4. Check haemodynamic status
5. Apply PEEP to dependant lung
6. Apply CPAP to non-dependant lung
7. Rule out obstruction, pneumothorax, tube displacement
8. Return to DLV
9. Clamp non-dependant pulmonary artery

References

1. Benumof LB, Alfery DD, Anaesthesia for thoracic surgery. Anesthesia 4th ed. 1994, Ed Miller RD Churchill Livingstone **52**:1663-1756
2. Falcoz PE, Conti M, Brouchet L,Chocron S, Puyraveau M, Mercier M, Etievent JP,Dahan M. The Thoracic Surgery Scoring System (Thoracoscore): Risk model for in-hospital death in 15,183 patients requiring thoracic surgery. *J Thorac Cardiovasc Surg* 2007;**133**:325-332
3. Chamogeorgakis TP, Connery CP, Bhora F, Nabong A, Toumpoulis IK. Thoracoscore predicts midterm mortality in patients undergoing thoracic surgery. *J Thorac Cardiovasc Surg* 2007;**134**:883-887
4. Lim E, Baldwin D, Beckles M, et al. Guidelines on the radical management of patients with lung cancer. *Thorax* 2010;**65**(III):iii1-iii27
5. Wong DH, Weber EC, Schell MJ, Wong BA, Anderson CT, Barker SJ, Factors associated with postoperative pulmonary complications in patients with severe chronic obstructive pulmonary disease. *Anaesth Anal* 1995;**80**:276-84
6. Agostini P, et al Postoperative pulmonary complications following thoracic surgery are there any modifiable risk factors. *Thorax* 2010;**65**(9):815-818
7. Rock P, Rich PB. Postoperative pulmonary complications. *Curr Opin Anaesthesiology* 2003;**16**:123-132
8. Stephan F, Boucheseiche S, Hollande J, Flahault A, Cheffi A, Bazelly B, Bonnet F. Pulmonary complications following lung resection*A comprehensive analysis of incidence and possible risk factors. *Chest* 2000;**118**:1263-1270
9. Slinger P, Darling G. Principles and practise of anaesthesia for thoracic surgery. 2011, Ed Slinger. Springer Science and Business Media
10. Biccard BM. Relationship between the inability to climb two flights of stairs and outcome after major non-cardiac surgery: Implications for the pre-operative assessment of functional capacity. *Anaesthesia* 2005;**60**:588-593
11. Kittnar O, Cardiac preload: haemodynamic physiology during thoracic surgery. *Curr Opin Anaesthesiology* 2011;**24**:21-23
12. Brodsky J. Anaesthesia for thoracic surgery. A practice of anaesthesia 6th ed. 1995, Cohen PJ. Wylie & Churchill: 1148-1170
13. Banerjee A. Anaesthesia and myasthenia gravis. *Anaesthesia tutorial of the week* 2008. http://www.totw.anaesthesiologists.org
14. Thoracic anaesthesia. Mitchell Anaesthetic Notes. FRCA UK. http://www.frca.co.uk
15. Slinger P, Karsli C. Management of the patients with a large anterior mediastinal mass: recurring myths. *Curr Opin Anaesthesiology* 2007;**20**:1-3
16. Fearson DZ, Nesbilt JC. The LMA: a new standard for airway evaluation in thoracic surgery. *Ann Thorac Surg* 1997;**63**(3):768-772
17. Arevalo Ludena J, Arcas Bellas JJ, Lopez Perez Y, Cuarental Garua A, Alvarez-Rementena Carbmell R. Placement of a bronchial blocker through the i-gel supraglottic airway device for single lung ventilation:preliminary study. *European Journal of Anaesthesia* 2010;**27**(47):257

18. Rippin BD, One lung ventilation. *Anaesthesia tutorial of the week* 2009

19. Grichnik KP, Shaw A. Update on one lung ventilation :the use of continuous positive airway pressure ventilation and positive end-expiritory pressure ventilation-clinical application. *Curr Opin Anaesthesiology* 2009;**22**:23-30

20. Pedoto A, Heerdt PM. Postoperative care after pulmonary resection: postanaesthesia care unit versus intensive care unit. *Curr Opin Anaesthesiology* 2009;**22**:50-55

21. Campos JH, Fastrack in thoracic anaesthesia and surgery. *Curr Opin Anaesthesiology* 2009;**22**:1-3

22. Day D. Keeping patients safe during intrahospital transport. *Crit Care Nurse* 2010;**80**(4):18-32

23. Lit M. Nursing management of chest drains. http//www.hkresp.com

24. Paramasivam E, Bodenham A. Airleaks, pneumothorax and chest drains. *Contin Educ Anaes Crit Care Pain* 2008;**8**(6):204-209

25. Scott NB, Wound infiltration for surgery. *Anaesthesia* 2010;**65**(1):67-75

26. Dalya DJ, Myles PS. Update on the role of paravertebral blocks for thoracic surgery: are they worth it? *Curr Opin Anaesthesiology* 2009;**22**:38-43

27. Skin N, Cassara EL. Pre-operative pulmonary evaluation and therapy for surgical patients. JAMA. 211:787-

Anaesthesia for Bariatric Surgery

Suresh Venugobal
Senior Lecturer, Anaesthetist and Intensivist, University Malaya Medical Centre

INTRODUCTION

Obesity occurs when someone carries too much fat for their build and gender. Obesity is a major health issue for most countries not only in terms of costs but also due to the concomittent increase in the incidence of chronic non communicable medical ailments. It is now an endemic and the facts are that worldwide obesity has more than doubled since 1980 and in 2008 there were 1.5 billion adults who were overweight and out of these, 200 million men and 300 million women were obese.[1]

The WHO definition using the body mass index (BMI) is as follows:

- Normal - BMI 18.6 to 24.9
- Overweight - BMI ≥ 25
- Obese - BMI ≥ 30
- Morbid obesity (35 if there is concomitant obesity associated disease) - BMI ≥ 40
- Super morbid obesity - BMI ≥ 55

The scale differs for Asians with normal defined as BMI of 18.5 to 22.9.

WHO has also classified obesity in other ways:

- Class I BMI 30-34.99
- Class II BMI 35-39.99
- Class III BMI equal or greater than 40

The above scale has been criticised as it does not indicate the percentage of fat. Furthermore discrepencies in BMI are evident between genders despite having the same percentage of fat.

Circumference of the waist can be used as a simple measure of obesity.

- Increased risk of health problems Men ≥ 94cm, Women ≥ 80cm.
- Greatly increased risk Men ≥ 102cm, Women ≥ 88cm.

Bio impedance and skin fold thickness can also be used to estimate lean body mass.

The pathology in obesity arises due to the energy imbalance between calory consumption and expenditure. This is the result of increased intake of energy dense food that is high in fat, salt and sugar but low in vitamins, minerals and micronutrients. This effect is pronounced when there is reduction of physical activity due to a myriad of effects brought forward with urbanisation.

The consequences are daunting;

- Cardiovascular disease (mainly stroke and heart disease)
- Diabetes Mellitus
- Musculoskeletal disorders
- Certain types of cancer

SURGICAL INDICATION AND PROCEDURES

Treatment of the overweight patient involves a multidisciplinary approach. This involves diet with lifestyle modification, increasing physical activity and approved drug therapy. However the above non invasive approach can rarely sustain weight loss.

In the United Kingdom, the National Institute for Health and Clinical Excellence (NICE)[2], recommends bariatric surgery as an intervention when non surgical measures fail to achieve or maintain weight loss for 6 months. The groups offered are:

- Patients with BMI 35 to 40 with obesity associated disease (example diabetes mellitus and obstructive sleep apnoea).
- BMI 40 to 50 with no medical ailments.
- As first line treatment for BMI ≥ 50.

Based on US research, extremely obese patients could gain 3 years life expectancy if offered bariatric surgery.[3] Bariatric comes from the Greek word *baros* indicating weight and *iatric*, medical treatment. The surgical procedures are divided into malabsorptive or restrictive or combination of both. Restrictive surgery includes vertical banded gastroplasty and gastric banding surgery. Pure malabsorptive procedures are jejuna-ileal bypass or biliopancreatic bypasses which are rarely performed these days. Laparoscopic Roux en Y gastric bypass combines both the restrictive and malabsorptive mechanisms.

The gold standard remains Roux en Y gastric bypass surgery (RYGB). This involves anastomosing the proximal gastric pouch to a proximal segment of jejunum, bypassing most of the stomach and the entire duodenum. It is the most effective bariatric procedure to achieve sustained weight loss. Patients can benefit and maintain weight loss upto 50% of the excess weight and occassionally may Achieve resolution of Type II diabetes mellitus. This procedure is performed laparoscopically and has better patient outcomes compared to open procedures. However this procedure becomes technically difficult when the patient is more than 160 kgs.

Adjustable gastric banding surgery involves inserting an adjustable inflatable band around the proximal stomach to limit intake. This technique has a shorter learning curve and is less complicated compared to gastric bypass surgery. Excess weight loss of up to 50% can be achieved and maintained. Its complication rate is documented at up to 19% and this includes pouch herniation, band erosion, oesophagitis and mechanical failure.

Other restrictive procedures include sleeve gastrectomy and vertical banded gastroplasty.

PREOPERATIVE ASSESSMENT

The assessment of these individuals requires a multidisciplinary approach. This involves the participation of surgeons, anaesthetists, endocrinologists, specialist nurses, dieticians and psychologists. Ideally all these patients should be seen at a bariatric clinic where a registered nurse does the initial screening. The nurse then arranges for a multidisciplinary meeting where the respective teams can perform further assessments. The aim of the preoperative assessments includes optimising the patient's physiomental parameters and assessing suitability for surgery with the quest of improving outcome.Ideally the anaesthetist involved is the one who manages these cases on a regular basis. He routinely begins the assessment at the combined meeting which is usually commenced12 weeks in advance.

Patients are usually started on a low calorie diet to ensure weight loss of 5 to 10 % over a 6 month period. There is evidence this leads to less postoperative morbidity especially after gastric bypass surgery.

Obese patients have to be evaluated thoroughly. They have issues with almost all of their physiological systems.

- **Airway and Respiration.**

While weight alone is not a predictor of a difficult airway and intubation, obese patients are assumed to have a difficult airway. Brodsky has shown that obesity in association with large neck circumference and Mallampathi score of III and above are associated with a difficult intubation. It is estimated that 5% of obese patients will have a difficult airway if the neck circumference is 40 cm as opposed to 35% if the neck circumference is 60 cm.[4] However the current available evidence is not strong enough to recommend the routine use of the awake fibreoptic intubation.

Obstructive sleep apnoea is associated with obesity. It is estimated that 40 to 90% of obese patients have OSA. This group of patients are at increased risks. Features that point to the presence of OSA include increased neck circumference (more than 43 cm for male and 40 cm for female), Mallampati score of 3 or more, retrognathia, lateral peritonsillar swelling,

macroglossia, tonsillar hypertrophy and nasal abnormality. Other factors include snoring and daytime sleepiness or somnolence. Epworth Sleepiness Scale can be used to score the patients. Patients scoring more than 12 should be referred for a sleep study. If scores are positive for OSA, they are offered Non Invasive Positive Pressure Ventilation (BIPAP).

Obese patients tend to exhibit shallow rapid breathing due to their lower chest wall and lung compliance. The closing capacity encroachs on their tidal breathing. As a result many will demonstrate hypoxia secondary to atelectasis, however demonstrable hypercarbia may not be present. A thorough history and examination will elucidate the basal respiratory function. Cessation of smoking for at least 8 weeks should be ascertained. General investigations include routine blood investigations, arterial blood gas and chest radiograph. A lung function test may not be necessary all the time. A referral to a chest physician is indicated if other lung ailments are present or example Bronchial Asthma which may require optimistion.

- **Cardiovascular**

All bariatric surgery patients should be evaluated for hypertension (systemic and pulmonary), ischemic heart disease and heart failure. This is difficult as the symptoms may be obscured by their sedentary lifestyle. Obesity especially the android variant is associated with ischemic heart disease. Physical examination may not reveal much either.

Effort tolerance will provide some clue to the general fitness level. It can be assessed using the metabolic equivalents (MET) with a score of 4 indicating moderate functional capacity.

Perioperative cardiac risk should be assessed in accordance with the AHA/ACC guidelines. Based on the revised cardiac risk index and functional capacity, patients can be identified if they need further cardiac evaluation. If METs is more than four with no identifiable risk factors, there is no need for further evaluation. Patients who are sedentary and have 2 or more risk factors need to be referred to the cardiologist for further investigation. This includes nuclear scanning or pharmacological stress testing for those who are not fit. Appropriate patients may be investigated using an angiogram, which is the gold standard.

Cardiopulmonary exercise testing can be considered in patients who can exercise. Patients who have a VO_2 max of less than 15.8 mls/kg/min are at higher perioperative risks.[5] Presence of pulmonary hypertension is difficult to pick up clinically. Echocardiogram can be used to detect pulmonary hypertension based on the presence of tricuspid regurgitation.

- **Gastrointestinal, liver and renal systems**

All obese patients are at risk for gastroesophageal reflux. Appropriate measures should be taken preoperatively to reduce the gastric acidity and risk of aspiration. The liver enzymes are usually elevated in the obese and a good proportion of them demonstrate fatty liver. However the metabolic function of the liver remains normal. Renal clearance of drugs are increased because of the increased glomerular filtration rate (GFR). Patients coming for repeat bariatric surgery may present with other problems. Apart from reflux, they may be at risk of nutritional deficiencies including folate, Vitamin B_{12}, calcium and iron. It is essential for their peripheral nervous system be evaluated (folate, Vit B_{12}), and coagulation checked (Vit K deficient).[6]

- **Neurological system**

Obese patients are at risk of postoperative neuropathies and thus any pre-existing neuropathy should be recorded.

Apart from the above concerns, patients should be evaluated for ease of arterial and venous cannulation to facilitate administration of fluids and for monitoring. Premedication should be directed towards anxiolysis and reduction of gastric acidity and volume. Benzodiazepines appear safe; nevertheless it should be given with caution in patients with OSA. Usually a

combination of non particulate antacid (sodium citrate) and proton pump inhibitor / H_2 receptor antagonist is prescribed. Thromboembolatic prophylaxis must be considered as all obese patients are considered at risk for DVT and PE. The preferred choice of drug against the development of DVT and PE are the low molecular weight heparins in combination with mechanical prophylaxis (pneumatic calf compression). Bioavailability of LMWH continues to be debated and these drugs should ideally be started preoperatively. In certain subgroups of patients, it is recommended that an inferior vena cava filter be inserted to prevent PE.

The anaesthetist should also evaluate all medications taken by the patient preoperatively. All medications should be continued except for oral hypoglycaemics, which should be substituted before surgery with insulin to ensure tight glycaemic control. Patients should be asked if they are consuming any herbal medications or any medical treatment that is used for weight reduction (sibutramine and orlistat). Anaesthetists should be aware of side effects of these drugs which include isolated cases of hypertension. There does not seem to be any drug interaction between these drugs and anaesthetic agents.

PERIOPERATIVE RISKS

The obesity surgery-mortality risk score (OS-MRS) has been validated for patients undergoing bariatric surgery.

1. BMI > 50
2. Male
3. Hypertension
4. Risk factors for pulmonary embolism
5. Age more than 45

Each factor is allocated one point. They are classified as low risk for scores of 0-1 (mortality rate 0.31%), intermediate risk for a score 2-3 (mortality rate 1.9%) and high risk for a score of 4-5 (mortality rate 7.5%)[7]

INTRAOPERATIVE MANAGEMENT

It is indeed challenging to manage these patients for bariatric surgery. The first consideration has to be both patients and staff safety. The regular operating tables can manage weights of up to 220 kg but there are special operating tables which have extra width and take more weight for bigger patients. There should be enough staff present to position the patient as well as equipment like sliders to ease movement of patients to and fro the operating table. There has to be enough straps to ensure the patient does not slip. Some theatres may have the bean bag which can be moulded to prevent patient movement especially during reverse Tredelenberg position.

As these patients are prone to nerve damage, care must be taken to avoid traction of nerve roots. Both brachial plexus and sciatic nerve damage have been reported. Warner has described in a retrospective study that ulna nerve neuropathy has been demonstrated in 29% of obese patients (BMI >38) compared to 1% in the non obese population.[8]

Induction and intubation is generally done via rapid sequence induction to prevent gastric aspiration. To prevent hypoxaemia the patient is generally placed head up about 25 degrees and preoxygenated. Stacking is applied where pillows or blanket are put under the occiput to ensure the chin is higher than the chest to ease intubation.[9] Neck circumference has been shown to predict difficult intubation, especially for those with more than 60 cm circumference. The anaesthetist should judge how best to secure the airway. A Ryles tube is also inserted to decompress the stomach for ease of surgery.

Dosing for the anaesthetic drugs is dependent on the pharmacological properties of the drug, whether lipophilic or hydrophilic in nature. Generally lipophilic drugs are given according to total body weight and hydrophilic drugs are given according to lean body mass. There are exceptions to the rule. Most muscle relaxants and propofol are dosed according to lean body mass.Drugs dosed according to total body weight

include thiopentone, opioids (with the exception of remifentanil), benzodiazepines, succinylcholine and atracurium. Caution is applied when calculating the thiopentone dose using total body weight. A lower induction dose per kilogram body weight is required compared to lean patients.[10]

Inhalational agents of choice remain agents with fast offset like desflurane and sevoflurane which has less lipophilicity.

Generally invasive arterial monitoring is applied due to conical shape of the arm. Rarely would a central line be needed unless it is difficult to get peripheral venous access. Ultrasound will be useful to localise the vessels.

Laparascopy will present an extra challenge to the maintenance of adequate ventilation in these individuals. Lung protective strategy and the usage of PEEP are important to prevent hypoxia and atelectasis post operatively. Generally rates of up to 24 bpm and tidal volume of 8 to 10 mls/kg of ideal body weight are used and the plateau pressure is kept less than 35 cmH_20. The anaesthetist must make sure the intra abdominal pressure is kept below 15 mm Hg and complete muscle relaxation is employed to ease surgery.

Fluid management is usually based on lean body mass (20 to 40 % above ideal body weight). Ryle's tube and gastric tube insertion remains an important procedure not only to check anastomosis with methylene blue dye but also as a conduit for creating the gastric pouch.

Rhabdomyolysis can occur especially in patients with BMI of more than 50 and surgery longer than 4 hours.

POSTOPERATIVE MANAGEMENT

All patients should be assessed for extubation unless any untoward operative issues are encounted. They should be positioned in the sitting position, fully awake, assessed for full recovery from muscle relaxation before being extubated.

All patients need to be in a monitored environment post operatively. Generally high dependency care is appropriate unless the patient has risk factors, in which case Intensive Care would be appropriate. This risks includes age of more than 45, cardiopulmonary issues including ischemic heart disease with ejection fraction less than 40%, BMI>50, severe OSA.

OSA patients will need to be on NIPPV/CPAP post operatively. There is evidence pointing to the safety of these devices post operatively without causing anastomotic leakage.[6] Obesity hypoventilation syndrome will be more difficult to manage. These patients are more prone to become hypercapnic post operatively and will benefit from intensive unit care management and regular sampling of arterial gases to evaluate carbon dioxide levels.

Pain has to be treated with multimodal approach, including the use of COX 2 inhibitors/ NSAIDS, paracetamol and opioids. Neuraxial blocks like epidural may be technically difficult. Since most procedures are performed laparascopically, the use for epidural has declined. Some surgeons may be concerned with the use of NSAIDS in view of the risk of gastric ulcers. Usually morphine is given via patient controlled analgesia technique.

Atelectasis still remains a major problem. Chest physiotherapy, deep breathing exercise and incentive spirometry remains the mainstay of treatment.

The other major concern is the risk of venous thrombo embolism. Low molecular weight heparin should be started preoperatively and continued postoperatively. In addition pneumatic calf compressors should be applied and patients should be encouraged to mobilise by the first post operative day. There is evidence that twice daily dosing is efficacious without increasing the rate of bleeding.[11]

References

1. WHO media centre. Obesity and overweight.
2. NICE guidelines.
3. Schauer DP. Decision modelling to estimate the impact of gastric bypass surgery on life expectancy for the treatment of morbid obesity. *Archives of Surgery* 2010 Jan;**145**(1):57-62
4. Brodsky JB. Morbid obesity and tracheal intubation. *Anesth. Analg.* 2002;**94**:732-36
5. McCullough PA. Cardiorespiratory fitness and short term complications after bariatrc surgery. *Chest* 2006;**130**:517-25
6. Babatunde O. Anaesthetic considerations for bariatric surgery. *Anesth. Analg.* 2002;**95**(6):1793-1805.
7. DeMaria EJ. Obesity surgery mortality risk score: proposal for clinically useful score to predict mortality risk in patients undergoing gastric bypass. *Surg. Obes. Relat. Dis.* 2007 Mar;**3**(2):134-40
8. Warner MA. Ulnar neuropathy in surgical patients. *Anestesiology* 1999 Jan;**90**(1):54-9
9. Myatt J. Airway management in obese patients. *Current Anaesthesia and Critical Care* 2010 Feb;**21**(1):9-15
10. Jung D. Thiopentone disposition in lean and obese patients undergoing surgery. *Anesthesiology* 1982;**56**:269-74
11. Scholten DJ. A comparison of two different prophylactic dose regime of low molecular weight heparin in bariatric surgery. *Obes. Surg*. 2002;**12**:19-24

Case Report: Anaesthetic Experience of the Surgical Separation of Thoraco-Omphalopagus Twins in Paediatric Institute, Hospital Kuala Lumpur

Muhammad Habibullah, Thavaranjitham Sandrasegaram, Hamidah Ismail, Nik Azizah, Intan Zarina
Department of Anaesthesiology, Paediatric Institute, Hospital Kuala Lumpur

INTRODUCTION

Several medical and surgical aspects of the separation of complex conjoined twins have been discussed,[1,2,3,4] and also carefully reviewed.[5,6,7,8] Perioperative aspects concerning the anaesthesiologist, such as preoperative evaluation,[9,10,11,12] monitoring, airway management, ventilatory support, anaesthetic pharmacology and fluid therapy can be found in the literature. We describe the anaesthetic management of the surgical separation of thoraco-omphalopagus twins in Paediatric Institute, Hospital Kuala Lumpur.

CASE REPORT

Pre-operative period
A set of a female thoraco-omphalophagus twins were delivered by caesarean section at 39 weeks of gestational age. Antenatal MRI showed sharing of liver and diaphragm with possible sharing of pericardium of the heart. Their combined birth weight was 5.20kg. The Apgar scores of twin A and B were 8, 9 at 1 and 5 min respectively after delivery.

Thorough pre-operative evaluation was initiated including routine blood and urine analysis, coagulation screen, plain x-rays, ultrasound scans, computed tomography (CT) scans and echocardiography (ECHO). Abdominal ultrasonography revealed fused livers. ECHO findings revealed 2 hearts sharing the pericardium at the apex but with separate myocardium. There was no evidence of gastrointestinal tract (GIT) communication between the twins seen on GI contrast studies. CT scan abdomen and thorax revealed fused liver segments at segment IV, V and VIII with separate hepatic and portal veins. They shared the pericardium at the level of the apex. Each twin was with it's own organs: stomach, spleen, gallbladder, pancreas, urinary bladder and a pair of kidneys.

Planning and rehearsals
Representatives of the participating services from Paediatric Surgery, the Paediatric anaesthetist, the Paediatric intensivist, the Paediatric Radiologist, nursing and operating room support staff met on several occasions to finalize the plan for the surgery to ensure the smooth separation of twins. The surgical and anaesthesia plan, staffing, location of anaesthetic equipment and surgical equipment, patient positioning, plan for repositioning and moving one of the twins to the adjacent operation room after separation were discussed.

A contingency plan for transfusion of blood and blood products was discussed with the blood bank. The anaesthesia monitoring system in our largest operating theatre was modified to allow each anaesthetic team to orient and view both twins' vital parameters separately.

FIGURE 1:
Thoraco-omphalopagus twins before surgery.

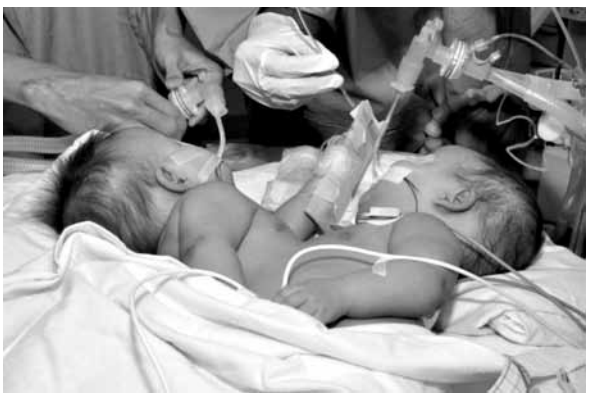


FIGURE 2: Endotracheal tube securement.

Peri-operative

The babies were separated on 19 November 2011, at an age of 3.8 months and combined weight of 11.0 kg. They were not premedicated. Each twin had a venous access. Dedicated teams of anaesthetists for each child along with duplication of all monitoring and equipment in each operating room were organized. Standard monitoring consisting of SpO2, ECG, and NIBP were instituted. Induction was carried simultaneously for both babies with fentanyl, thiopentone and atracurium. Mask ventilation and orotracheal intubations were done sequentially.

Right radial artery and right internal jugular veins were cannulated under ultrasound guidance. Arterial BP and CVP were monitored. Urinary bladder was catheterized for urine output measurement and nasopharyngeal temperature probe was inserted for temperature monitoring. Antibiotics, Cefotaxime and Metronidazole were administered as antibiotic prophylaxis for both twins following induction of anaesthesia.

Anaesthesia was maintained with a mixture of sevoflurane and oxygen with FiO_2 of 0.33 in both twins. Body temperature was kept at 36–37°C throughout the operation by a warmed air mattress. Blood and fluid losses were replaced accordingly. The twins were separated after 1 h 30 min into the surgery. After separation, twin B was transferred to an adjacent operating theatre for further surgery, while twin A remained in the same operating theatre. The vital signs for both twins were stable throughout the operation. Twin A was transfused with 60 mls of blood, 50 ml of 5% albumin and 540 mls of crystalloids, and twin B was transfused with 100 mls of blood, 100 mls of 5% albumin and 450 mls of crystalloid.

Initial attempts at closure of the thoracoabdominal defect, led to hypotension and ventilatory compromise in both twins. Synthetic tissue was therefore used for closure of the defect, and delayed skin closure was planned. The total anaesthetic time was 5 h 45 min for twin A and 7 h 10 min for twin B. After the operation, both twins were transferred to the Paediatric Intensive Care Unit for ventilatory support and intensive monitoring.

On the Day 2 post surgery, both twins were brought into the operation theatre again for closure of the defect. The procedure was well tolerated. The twins were extubated 5 days post closure and successfully transferred to the general ward on Day 7.

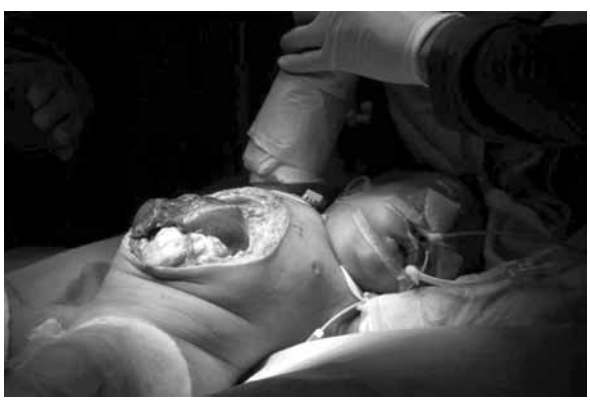


FIGURE 3: Skin deficit prior to closure.

DISCUSSION

Conjoined twins, also known as Siamese twins, are one of the rarest, most interesting, and challenging congenital malformations. The exact incidence of conjoined twins is unknown, but the estimated incidence is 1 in 50 000 live births. The rate in the United States is 1 in 100 000 births. Increased incidences range from 1 : 14 000 to 1 :

25 000 in Asia, India, Pakistan, Thailand and in Africa, especially East Africa, Nigeria and South Africa.[5,21]

The theory on the genesis of conjoined twins is incomplete division of the inner cell mass after the first 7 days when the monozygotic twinning process is thought to occur, which is between 13 and 16 days after fertilization. Conjoined twins are classified by their most prominent site of connection.Such sites include the thorax (thoracopagus) 40%, abdomen (omphaolpagus, xiphophagus) 33%, sacrum (pygopagus) 19%, pelvis (ischiopagus), or skull (craniopagus) 2%. Some authors group thoracopagus and omphalopagus together as thoraco-omphalopagus and, therefore, account for 73% of reported instances.[7]

The first case report of surgical separation of conjoined xiphopagus twins was in 945 AD from Constantinople when one of the twins died and the other survived for 3 days.[13] The first successful separation of xiphopagus twins was performed by Konig in 1689.[7] The separation was by tightening and necrosing the band of tissue between the twins. The first successful separation for thoracopagus twins, with at least one twin surviving, was performed in 1900, for pygopagus twins in 1912 and for craniopagus twins in 1952. In 1966, the first separation of ischiopagus tetrapus twins in which both twins survived was reported.[7] Anaesthetic management for separation of conjoined twins was first published by Hall *et al*. from Maryland in 1957.[14] Subsequently, many reports of anaesthetic management of conjoined twins have been published, including MRI study and separation.[15,16,17,18,19,20] Cardiopulmonary bypass and combined caudal and general anaesthesia for separations of conjoined twins have also been used.[18,19]

The timing for the separation of conjoined twins is important. Separation is best performed on an elective basis when the twins are 9–12 months old. Operative survival was 50% in those operated in the neonatal period, but 90% in those over 4 months of age.[6]

Preoperative assessment and planning, with interdisciplinary communication and cooperation, is vital to the success of the surgery. This surgery requires a dedicated team of surgeons, anaesthetists and staff for each child, and, consequently, duplication of all monitoring equipment in one operating room is necessary. Meticulous attention to detail, monitoring and vigilance are mandatory. Planning for the post-operative period in the Paediatric Intensive Care Unit (PICU), as well as the babies' rehabilitation is essential from the time of the initial admission to discharge.

As conjoined twins are joined in different postures, positioning during the operation can sometimes be difficult. Caution should be exercised with the cardiovascular system, pressure areas and hyperextension of joints and extremities during the procedure. Tracheal intubation can be difficult in craniopagus or thoracopagus twins. During induction of anaesthesia, induction agents and muscle relaxants given to one twin can result in sedation, airway obstruction, hypoventilation or apnoea in the other twin. Anaesthesia should be induced after it is known that both twins can be mask-ventilated. Then induction can either be by an intravenous or inhalation technique. If a problem with mask ventilation occurs, anaesthesia should be cautiously induced while spontaneous breathing is maintained. Intubation when awake may be required in the case of suspected difficult intubation and significant cross-circulation between twins. This may not be an attractive option for older conjoined twins. Moreover, coughing and straining during awake intubation can lead to haemodynamic disturbances in both twins.[19] Fibreoptic intubation in conjoined twins has recently been reported.[19]

We conclude that treatment of thoraco-omphalopagus twinning is a complex and multidisciplinary task involving strict cooperation between different medical personnel. Teamwork is essential to the management of these twins throughout a series of diagnostic and separation procedures, but the availability of two distinct teams of anaesthesiologists specifically experienced in paediatric surgery is very important.

References

1. Synhorst D, Matlak M, Roan Y, et all. Separation of conjoined thoracopagus twins joined at the right atria. *Am J Cardiol* 1979;**43**:662-665.
2. Bloch EC & Karis JH. Cardiopagus in neonatal thoracopagus twins: Anesthetic management. *Anesth Analg* 1980;**59**:304-307.
3. Brown DL, Holubec DM, Towle DJ et al. Anesthetic management of thoracopagus twins undergoing cardiopagus separation. *Anesthesiology* 1985;**62**:679-682.
4. Hoshina H, Tanaka O, Obara HT et al. Thoracopagus conjoined twins: Management of anesthetic induction and postoperative chest wall defect. *Anesthesiology* 1987;**66**:424-426.
5. Diaz JH, Furman EB. Perioperative management of conjoined twins. *Anesthesiology* 1987;**67**:965-973.
6. O'Neill JA, Holcomb GW, Schnaufer L et al. Surgical experience with thirteen conjoined twins. *Ann Surg* 1988;**208**:299-312.
7. Hoyle RM. Surgical separation of conjoined twins. *Surg Gynecol Obstet* 1990;**170**:549-562.
8. Holcomb GW, O'Neill JA. Conjoined twins. In: Ashcraft K W, Holder T M, eds. Pediatric surgery. *WB Saunders Company* 1993:948–955.
9. Patel R, Fox K, Dawson J et al. Cardiovascular anomalies in thoracopagus twins and the importance of preoperative cardiac evaluation. *Br Heart J* 1977;**39**:1254-1258.
10. Dev V, Pothineni RB, Rohatgi ME et al. Echo-Doppler assessment of cardiac status in conjoined (thoraco-omphalopagus) twins. *Pediatr Cardiol* 1990;**11**:91-92.
11. Danford DA, McManus BM, Nielsen SM et al, Definition of inseparably fused ventricular myocardium in thoracopagus: Fetal Echocardiographic utility and pathologic refinement. *Pediatr Cardiol* 1993;**14**:242-246.
12. Wu MH, Lai YC, Lo HM et al. Assessment of electromyocardial continuity in conjoined (thoracopagus) twins. *Am J Cardiol* 1992;**69**:830-832.
13. Pentogalos GE, Lascaratos JG. A surgical operation performed on Siamese twins during the tenth century in Byzantium. *Bull His Med* 1984;**58**:99–102.
14. Hall KD, Merzig J, Norris FH. Case report: separation of craniopagus. *Anesthesiology* 1957;**18**:908–910.
15. Ballantine RIW, Jackson I. Anaesthesia for separation of craniopagus twins. *Br Med J* 1964;**1**:1339–1340.
16. Tandan GC, Gode GR, Kalle NR et al. Anesthetic management for surgical separation of thoracopagus twins. *Anesthesiology* 1970;**33**:116–119.
17. Furman EB, Roman G, Hairabet J et al. Management of anesthesia for surgical separation of newborn conjoined twins. *Anesthesiology* 1971;**34**:95–101.
18. Suan C, Ojeda F, Garcia-Perla JL et al. Anaesthetic management of the surgical separation of a pair of thoracopagus-cardiopagus twins. *Paed Anaesth* 1998;**8**: 255–257.
19. Greenberg M, Frankville DD, Hilfiker M. Separation of omphalopagus conjoined twins using combined caudal epidural-general anesthesia. *Can J Anaesth* 2001;**48**: 478–482.
20. Clemessy J, Brusset M, Frot CM et al. Anaesthetic management for ischiopagus tetrapus conjoined twins separation. *Paed Anaesth* 1996;**6**:160–161.
21. Thomas JM, Lopez JT. Conjoined twins- the anaesthetic management of 15 sets from 1991-2002. *Paediatric Anaesthesia* 2004;**14**:117-129.

Haemodynamic Optimization and Fluid Therapy in the Surgical Patient

Hema Malini Manogharan
Consultant Anesthesiologist, Hospital Port Dickson

Haemodynamic optimization of surgical patients during the perioperative period aims to improve outcomes. It has been frequently referred to as goal-directed therapy (GDT), a term that has been used for nearly 30 years to describe methods of optimizing fluid and haemodynamic status. This term has never been standardized, and means different things to different people, as it was earlier coined for the early management of sepsis in the emergency department and intensive care units.[1] In the perioperative period, we have always depended on intravenous fluid resuscitation in order to maintain a patient's physiological function and to replace fluids lost.

We have always relied on non-invasive blood pressure (NIBP), heart rate, urine output, and occasionally arterial blood pressure and central venous pressure (CVP) for assessment of fluid status in the surgical patient. However, these are all known to be poor indicators of intravascular volume and cardiac output. During and after surgery, blood pressure is decreased by anaesthetic and analgesic drugs, and urinary output is decreased by the release of stress hormones. In healthy volunteers, heart rate and BP remained relatively unchanged despite a 25% loss of blood volume.[2] One systematic review showed that CVP is unable to identify which patient needed more fluids, and concluded that CVP poorly reflected fluid responsiveness when routinely measured in the intensive care, operating room, or emergency departments.[3]

There is little doubt that hypovolaemia leads to poor tissue perfusion, suboptimal organ function, organ failure and death. Fluid overload, on the other hand, may be just as harmful as hypovolaemia. Iatrogenic fluid overload has been shown to decrease pulmonary function, hamper gut motility and decrease subcutaneous oxygen tension.[4-7] Associations between intraoperative fluid overload and complications as well as mortality have been demonstrated following major surgical procedures.[8-11] Recent clinical trials have shown fluid overload following gastrointestinal surgery cause poor outcomes.[8,11]

Current 'standard fluid therapy' include replacement of fluid loss (by basal fluid requirements, insensible loss through surgical site, loss to third space and blood loss) and maintenance of physiological functions (i.e. 'preloading' in neuroaxial blocks). It is generally agreed that these losses should be replaced; however the usual disagreements that arise are regarding the timing of when to replace, the amount to replace and the type of fluid used for replacement. These replacements are very subjective and vary from person to person.

The *insensible fluid* loss in normal conditions is approximately 10ml/kg/day, and this does not change much during surgery. About 2/3 of this volume is lost through the skin and 1/3 from the airways. The loss through airways depend on the humidity of the inhaled air (inhalation of or ventilation with 100% water saturated air causes negligible loss, while inhalation of dry air leads to losses approximately 0.5ml/kg/hr).[12] Fasting preoperatively also inevitably leads to loss of water. The *evaporative loss* from the surgical wounds depend on the size of the incision and the exposure of the intestines. The loss from completely exteriorised viscera decreases by 50% after 20 minutes13 and wrapping the exteriorised viscera in plastic or wet abdominal packs reduces evaporative loss by 87.5%.[14]

The *loss to third space* can be divided into an anatomical and a non-anatomical loss.[15,16] The anatomical third space loss represents pathological accumulation of fluid in the extracellular space (ECV). Before, during or after surgery, the disease and/or trauma may cause fluid accumulation in the transcellular or interstitial space and cause an expansion of the ECV. Examples of this are ascites, pleural effusion or oedema in the interstitial spaces. Whereas, the volume of ascitic and pleural

fluids can be emptied through drains or during surgery and measured accurately, the volume of fluid accumulated in the interstitial space is more difficult to assess. It is highly influenced by the administration of intravenous fluids. In a study of rabbits, it was found that the formation of a small bowel anastomosis caused an oedema in the surrounding tissue by 5-10% without administration of intravenous fluids. The oedema doubled when 15mls/kg/hour of intravenous fluids were administered.[16] If equivalent changes occurred in humans, such oedema may accumulate around a small or large bowel anastomosis, depending on the volume of intravenous fluids administered. The non-anatomical third space loss (or deficit in functional extracellular volume) was earlier believed to occur in surgical trauma patients and patients undergoing abdominal surgery caused by a contraction of ECV; however many investigators have found that this so-called third space does not exist.[17]

Replacement of lost blood with a crystalloid demands infusions of double or triple the volume because the crystalloid is dispersed throughout the entire extracellular space. This causes an expansion of the interstitial space, with postoperative oedema formation and body weight gain. A colloid, on the other hand, stays in the vascular space for a longer time and seems to be a more expedient choice for replacement of lost blood. However, trials of colloids versus crystalloids have shown diverging results.

Urine in large volumes cannot be expected during surgery, both because the release of stress hormones reduce the excretion of salt and water, and because anaesthesia may cause hypotension. It is, however, important to distinguish between the anaesthesia-induced hypotension and hypovolaemia. The first is caused by vasodilatation and may reduce the glomerular function rate (GFR) but not the arterial blood pressure to the renal parenchyma. Hypovolaemia, on the other hand, reduces both GFR and renal blood supply and may cause renal failure. It is not at all evident that a large urinary output is necessary to prevent postoperative renal failure, nor that a small urinary output is associated with renal failure in the absence of hypovolaemia.[18-19] A small urine output during surgery is therefore acceptable as long as hypovolaemia is not the cause.

Neuroaxial blockade (eg. epidural analgesia used in many abdominal surgeries along with general anaesthesia) causes a relaxation of the vascular bed innervated by the affected segments of the spinal cord. This causes a decrease in peripheral vascular resistance with a decrease in arterial blood pressure. There is also venodilation causing venous pooling and decreased venous return to the heart. Despite the fact that cardiac output and peripheral blood flow may be unaltered, it is common to respond to this decrease in BP by giving either 500-1000mls of crystalloids or colloids intravenously. However, this treatment has not been shown to be effective. Earlier non-randomised trials and retrospective trials[20-22] suggested fluid preloading to reduce the incidence of hypotension in 20-35% of patients, but this has not been confirmed in clinical randomised trials of preloading versus no preloading. Fluid preloading did not significantly improve the drop in blood pressure or reduce the requirement for pressor agents.[23-26]

In a large multicentre trial of 172 patients randomized to receive either a restricted or a standard perioperative fluid regimen, Brandstrup et al.[8] showed that restriction of fluids significantly reduced the number of complications after colorectal surgery. A review of intraoperative fluid restriction in gastrointestinal surgery concluded that judicious perioperative fluid therapy can improve outcome after major surgery, recommending a balanced approach to fluid management.[27] In thoracic surgery, there is a trend towards a 'dry' regimen, with evidence to suggest that this reduces postoperative pulmonary complications.[28] With all these regimens, the consensus is to avoid tissue hypoperfusion, activation of the systemic inflammatory response and multiple organ failure, yet at the same time prevent fluid overload.

As we now come to realise that too much fluids and too little fluids are detrimental to the surgical patient, how do we get it just right? This leads to the question whether monitoring of stroke volume (SV) and cardiac output (CO) improves the ability to optimize fluid and haemodynamic status.

Perioperative haemodynamic optimization or 'goal-directed therapy' was first described in the 1980's when the pulmonary artery catheter (PAC) was used to guide fluid and inotrope administration[29] based on targets from earlier work by Shoemaker et al.[30] These oxygen-targeted approaches were able to show a survival benefit in many studies.[31-33] Despite these results, the technique was not widely adopted as it required significant resources, was very labour intensive and relied on information from the PAC. As several observational studies showed increased mortalities associated with the PAC, GDT that depended on the PAC fell out of favour.[34]

The last 10 years have seen the introduction of many minimally invasive cardiac output (CO) technologies such as oesophageal Doppler (CardioQ™), arterial pressure waveform analysis devices providing stroke volume variation (SVV) and pulse pressure variation (PPV) (eg PiCCO™, Vigileo™), and monitors based on bioimpedance and bioreactance technology.[35] This has enabled us to monitor and optimize SV, SVV, CO and other haemodynamic variables without the need for a PAC. These monitors are minimally invasive and relatively easy to operate. The change in SV, SVV and CO in response to a fluid challenge can be used to assess volume responsiveness based on the algorithms provided.

When a patient is hypovolaemic, this will result in a high SVV (>10%). An IV fluid challenge will typically result in >10% increase in SV or CO, with a resultant reduction in SVV. This means the patient has a 'recruitable' SV and is fluid-responsive. In the perioperative settings, fluid challenges should be considered until the SV no longer increases by 10% or SVV is < 10%. SVV and PPV have also been shown to be superior in predicting volume responsiveness in controlled mechanically ventilated patients.[36]

A number of studies have demonstrated that perioperative volume optimization is beneficial and results in improved outcomes with lower complication rates and shorter hospital stay.[37] In spite of these findings, we still do not perform haemodynamic optimization in major surgeries and high risk surgeries. This may be due to several factors. One possible factor is that as anaesthesiologists, we like to see immediate results and benefits from optimization, which is usually not obvious in the intraoperative and early post-operative periods. Equipment that is less user friendly and our scepticism towards the concept may also play a role. Furthermore, the absence of a large multicentre trial is a significant factor. Many of the studies done are single centre trials and as such are usually underpowered to detect a mortality difference.

Two excellent systematic reviews by Hamilton et al. and Gurgel et al. examined various 'goal-directed' interventions for haemodynamic optimization of patients undergoing major surgery. Hamilton et al.[38] specifically investigated haemodynamic intervention and showed a significant reduction in complications with modern invasive devices that are comparable with the PAC optimization. Gurgel et al.[39] on the other hand looked at use of tissue perfusion markers such as lactate and central venous oxygen saturations, and also showed a clear benefit with haemodynamic optimization.

The burden of complications and mortality for surgical patients is becoming increasingly understood. Interest in perioperative haemodynamic optimization and protocolized fluid administration continues to grow. It is easy to accomplish for major surgeries, makes physiologic sense, and has a growing evidence base. A reduction in complication rates and shorter hospital stays has been widely demonstrated across surgical types. There is emerging evidence that optimization during the perioperative period may be associated with a long-term (15years) survival benefit in high-risk patients.[40] Furthermore, Enhanced Recovery After Surgery (ERAS) programmes are currently driving increased interest in haemodynamic optimization.[41]

As major surgery is associated with a significant and quantifiable rate of morbidity and mortality, haemodynamic targeted therapy can reduce this risk. This reduction in postoperative complications can have a long lasting effect on the survival of the patient. It will also shorten hospital stay and reduce multiple costly interventions, indirectly

saving significant amounts of health care resources. In short, haemodynamic optimization using a variety of protocolized fluid regimes and minimally invasive technologies may be a key step in improving short, intermediate, and long-term outcomes in the surgical patient.

References

1. Rivers E, Nguyen B, Havstad S, Ressler J, Muzzin A, Knoblich B, Peterson E, Tomlanovich M. Early goal-directed therapy in treatment of severe sepsis and septic shock. *N Engl J Med* 2001;**345**:1368-77
2. Hamilton-Davies C, Mythen MG, Salmon JB, Jacobson D, Shukla A, Webb AR. Comparison of commonly used clinical indicators of hypovolemia with gastrointestinal tonometry. *Intensive Care Med* 1997;**23**:276-81
3. Marik PE, Baram M, Vahid B. Does central venous pressure predict fluid responsiveness? A systematic review of the literature and the tale of seven mares. *Chest* 2008;**134**:172-8
4. Holte K, Jensen P, Kehlet H. Physiologic effects of intravenous fluid administration in healthy volunteers. *Anesth Analg* 2003;**96**:1504-1509
5. Lobo DN, Bostock KA, Neal KR et al. Effect of salt and water balance on recovery of gastrointestinal function after elective colonic resection : A randomised controlled trial. *Lancet* 2002;**359**:1812-1818
6. Nisanevich V, Felsenstein I, Almogy G et al. Effect of intraoperative fluid management on outcome after intra-abdominal surgery. *Anesthesiology* 2005;**103**:25-32
7. Lang K, Boldt J, Suttner S, Haisch G. Colloids versus crystalloids and tissue oxygen tension in patients undergoing major abdominal surgery. *Anesth Analg* 2001;**93**:405-409
8. Brandstrup B, Tonnesen H, Beier-Holgersen R et al. The Danish study group on perioperative fluid therapy. Effects of intravenous fluid restriction on postoperative complications: comparison of two perioperative fluid regimes. A randomised assessor blinded multicentre trial. *Ann Surg* 2003;**238**:641-648
9. Arieff Al. Fatal postoperative pulmonary oedema. Pathogenesis and literature review. *Chest* 1999;**115**: 1371-1377
10. Bennett-Guerro E, Feierman DE, Barclay GR et al. Preoperative and intraoperative predictors of postoperative morbidity, poor graft function and early rejection in 190 patients undergoing liver transplantation. *Arch Surg* 2001;**136**:1177-1183
11. Lowell JA, Schifferdecker C, Driscoll DF et al. postoperative fluid overload: not a benign problem. *Crit Care M* 1990;**18**:728-733
12. Reithner L, Johannson H, Strouth L. Insensible perspiration during anaesthesia and surgery. *Acta Anesthesiol Scand* 1980;**24**:362-366
13. Lamke LO, Nielsson GE, Reithner HL. Water loss by evaporation from the abdominal cavity during surgery. *Acta Chir Scand* 1977;**143**:279-284
14. Roe CF. Effect of bowel exposure on body temperature during surgical operations. *Am J Surg* 1971;**122**:13-15
15. Carrico CJ, Canizaro PC, Shires GT. Fluid resuscitation following injury: rationale for the use of balanced salts. *Crit Care Med* 1976;**4**:279-284
16. Chan STF, Kapadia CR, Johnson AW et al. Extracellular fluid volume expansion and third space sequestration at the site of small bowel anastomosis. *Br J Surg* 1983;**70**: 36-39
17. Brandstrup B. Fluid therapy for the surgical patient. *Best Prac & Research Cl Anaest* 2005;**20**:265-283
18. Alpert RA, Roizen MF, Hamilton WK et al. Intraoperative urinary output does not predict postoperative renal function in patients undergoing abdominal aortic revascularization. *Surgery* 1984;**95**:707-711
19. Priano LL, Smith JD, Cohen JL, Everts EE. Intravenous fluid administration and urine output in radical neck surgery. *Head Neck* 1993;**15**:208-215
20. Clark RB, Thompson DS, Thompson CH. Prevention of spinal hypotension associated with caesarean section. *Anesthesiology* 1976;**45**:670-674
21. Fanelli G, Casati A, Berti M. Incidence of hypotension and bradycardia during intergrated epidural/general anesthesia. An epidemiological observational study on 1200 consecutive patients. Italian study group on intergrated anaesthesia. *Minerva Anestesiologica* 1998;**64**:313-319
22. Wollman SB, Marx GF. Acute hydration for prevention of hypotension of spinal anaesthesia in parturients. *Anesthesiology* 1968;**29**:374-380
23. Jackson R, Reid JA, Thornburn J. Volume preloading is not essential to prevent spinal-induced hypotension in Caesarean section. *Br J Anaesth* 1995;**75**:262-265

24. Kinsella SM, Pirlet M, Mills MS et al. Randomized study of intravenous fluid preloads before epidural analgesia during labour. *Br J Anaesth* 2000;**85**:311-313

25. Rout CC, Rocke DA, Levin J et al. A re-evaluation in the role of crystalloid preload in the prevention of hypotension associated with spinal anaesthesia for elective caesarean section. *Anaesthesiology* 1993;**79**:262-269

26. Park GE, Hauch MA, Curlin F et al. The effects of varying volumes of cystalloid administration before caesarean section on maternal haemodynamics and colloid osmotic pressure. *Anaesth Analg* 1996;**83**:299-303

27. Joshi GP. Intraoperative fluid restriction improves outcome after major elective gastrointestinal surgery. *Anaesth Analg* 2005;**101**:601-605

28. Slinger P. Perioperative fluid management for thoracic surgery: the puzzle of postpneumonectomy pulmonary edema. *J Cardiothoracic Vasc Anesth* 1995;**9**:442-451

29. Shoemaker WC, Montgomery ES, Kaplan E et al. Prospective trial of supranormal values of survivors as therapeutic goals in high risk surgical patients. *Chest* 1988;**94**:1176-86

30. Shoemaker WC, Montgomery ES, Kaplan E, Elwyn DH. Physiologic patterns in surviving and non-surviving shock patients: use of sequential cardiorespiratory variables in defining criteria for therapeutic goals and early warning of death. *Arch Surg* 1973;**106**:630-636

31. Lobo SM, Salgado PF, Castillo VG et al. Effects of maximising oxygen delivery on morbidity and mortality in high-risk surgical patients. *Crit Care Med* 2000;**28**: 3396-3404

32. Polonen P, Ruokonen E et al. A prospective, randomized study of goal-oriented hemodynamic therapy in cardiac surgical patients. *Anaesth analg* 2000;**90**:1052-1059

33. Wilson J, Woods I, Fawcett J et al. Reducing the risk of major elective surgery: randomized controlled trial of preoperative optimization of oxygen delivery. *BMJ* 1999;**318**:1099-1103

34. Connors AF Jr, Speroff T, Dawson NV et al. The effectiveness of right heart catheterization in the initial care of critically ill patients. SUPPORT investigators. *JAMA* 1996;**276**:889-897

35. Funk DJ, Moretti EW, Gan TJ. Minimally invasive cardiac output monitoring in the perioperative setting. *Anaesth Analg* 2009;**108**:887-897

36. Marik PE, Cavallazi R, Vasu T, Hirani A. Dynamic changes in the arterial waveform derived variablesand fluid responsiveness in mechanically ventilated patients; a systematic review of the literature. *Crit Care Med* 2009;**37**:2642-2647

37. Giglio MT, Marucci M, Testini M, Brienza N. Goal-directed hemodynamic therapy and gastrointestinal complications in major surgery: a meta-analysis of randomized controlled trials. *Br J Anaesth* 2009;**103**:637-646

38. Hamilton MA, Cecconi M, Rhodes A. A systematic review and meta-analysis on the use of pre-emptive hemodynamic intervention to improve postoperative outcomes in moderate and high risk surgical patients. *Anaesth Analg* 2011;**112**:1392-1402

39. Gurgel ST, do Nascimento P Jr. Maintaining tissue perfusion in high-risk surgical patients: a systematic review of randomized clinical trials. *Anaesth Analg* 2011;**112**:1384-1391

40. Rhodes A, Cecconi M, Hamilton M et al. Goal-directed therapy in high-risk surgical patients: a 15 year follow-up study. I*ntensive Care Med* 2010;**36**:1327-1332

41. Lassen K, Soop M, Nygren J, et al. Consensus review of optimal perioperative care in colorectal surgery: Enhanced Recovery After Surgery (ERAS) Group recommendations. *Arch Surg* 2009;**144**:961-969

Do We Need to Know More About Ischaemic Optic Neuropathy?

Vanitha Sivanaser
Consultant Anaesthesiologist & NeuroAnaesthetist, Hospital Pulau Pinang

INTRODUCTION

Ischemic optic neuropathy is a rare but important complication that can cause permanent visual loss especially so after spine surgery in the prone position. Postoperative vision loss (POVL) has garnered interesting alarm over the past 15 years, resulting in increased litigation. The magnitude of the problem has been addressed by the American Society of Anesthesiologists and they developed a Post Operative Visual Loss Registry to better understand and evaluate this catastrophic complication.

The pathophysiology of Ischemic Optic Neuropathy (ION) is poorly understood and its risk factors remain speculative. As there is no known treatment for this acquired condition, it is imperative that we as anaesthetists should have an increased understanding of this condition.

DEFINITION AND CLINICAL FEATURES

Postoperative vision loss (POVL) encompasses Central Retinal Artery Occlusion (CRVO), Ischemic Optic Neuropathy (ION) and cortical blindness.

FIGURE 1: The major differences are tabulated below

Central Retinal Artery Occlusion (CRVO)	**Cortical Blindness**	**Ischemic Optic Neuropathy (ION)**
Incidence: • Second most common cause of POVL[1]	• Common after cardiothoracic surgery	• Frequent occurrence after lumbar spine surgery
Causes: • Mainly caused by embolic strokes or as a consequence of direct external pressure on the eyes.	• Is a result of infract of visual areas in the occipital lobe or may be caused by stokes that may be embolic or thrombotic in nature	• Most common cause of POVL
Clinical Features • Characterized by retinal pallor and "cherry red spots" on fundoscopic examination.[1]	• Associated with other features of stroke. • Eye essentially normal.	2 different forms are recognized based on the distribution of the optic nerve ischemic • Anterior Ischemic Optic Neuropathy (AION) • Posterior Ischemic Optic Neuropathy (PION)

FIGURE 2: Major Differences between Anterior Ischemic Optic Neuropathy (AION) and Posterior Ischemic Optic Neuropathy

Anterior Ischemic Optic Neuropathy (AION)	Posterior Ischemic Optic Neuropathy (PION)
• Onset of blindness is usually delayed from 48 hours to a week after surgery.[2] • Symptoms rarely occur upon awakening from anaesthesia. • Usually painless.	Onset of symptoms are upon awakening (60%) and is not uncommon to have symptoms within 24 hours (40%)[2]
• Binocular vision involvement is frequent (50-60%)[2]	
• Onsets of symptoms are accompanied by optic nerve oedema. Hemorrhagic spots may or may not be present on fundoscopy.	• Optic disc appears normal at time of onset of symptoms
	• Affects men more frequently(>70%) than women
	• Frequently encountered in lumbar spine surgery.[2] • Occurrences also reported in cervical and thoracic surgeries.

THE ANATOMY OF THE OPTIC CANAL

Basic understanding of the optic canal is required to understand the development of ION. It is beyond the scope of this article to describe the anatomy of the globe in detail, an excellent review has been published by Hayreh[3] for further detailed reading.

A comprehensive overview is described below for the brief understanding of this article. The optic canal is enclosed within a closed cavity, bounded anteriorly by the globe, and laterally by the bony structures of the frontal and maxillary sinuses. The posterior boundary of the canal opens within the cerebral structures. Cerebrospinal fluid surrounds the neurovascular bundle up to the globe. The optic nerve transverses the optic canal in its prechiasmic portion.

BLOOD SUPPLY AND THE BLOOD FLOW TO THE OPTIC NERVE

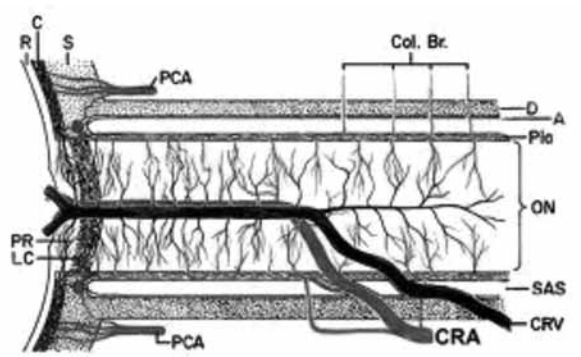
The anterior portion of the optic nerve has rich arterial supply and is supplied by the left and right posterior ciliary arteries, the peripapillary choroid arteries and short posterior ciliary artery. Their sectoral distribution might explain the occurrences of segmental vision loss.

The posterior portion of the optic nerve is supplied by multiple arteries. Branches from the central retinal artery (CRA) combine with the vessels derived from the hypophyseal artery and provide the vascular supply to the posterior portion.

The anterior and posterior portion of the optic nerve are richly supplied while in contrast the mid portion of the optic nerve within the optic canal is supplied only the pial vascular plexus derived from the arterial extensions of the anterior and posterior blood supplies and the intraneural branches of the central retinal artery. The relative lack in the vascular supply to the mid portion of the nerve renders it at an increased risk for ischemia and it is this portion of the nerve that is thought to be related to PION.

Venous drainage occurs primarily via the central retinal vein and the small orbital veins. There are retinociliary collaterals to the peripapillary choroidal veins and drainage through these collaterals can be substantial in central retinal vein thrombosis.

FIGURE3: Blood supply of the optic nerve by the central retinal artery



R-retina, C-choroid S-sclera, PCA- posterior ciliary artery,CRA –central retinal artery, D-dural sheath, A-arachnoid membranr, Pis- pia mater, ON-optic nerve, SAS-subarachnoid space.

AUTOREGULATION AND OCULAR PERFUSION PRESSURE

Blood flow to any organ depends on the perfusion pressure, resistance to flow, rheology of blood and the presence or not of auto regulation.

Blood flow to the human eye is autoregulated and is similar to that of the brain. High blood pressure (BP) and high intraocular pressure may alter the autoregulatory mechanisms. Autoregulation allows a constant blood flow over a pressure range despite fluctuations in perfusion pressure (which in the eye translates into the pressure difference between the arterial blood entering the eye and the venous blood existing the eye). Above and below this pressure range, compensatory mechanisms are exhausted and vasomotor adjustments become linearly related to perfusion pressure.

Similar to the cerebral perfusion pressure, the ocular perfusion pressure (OPP) is calculated as the differences between mean arterial BP and the intraocular pressure (IOP) as it is similar to the pressure in the vein leaving the eyes. Studies on the monkey have demonstrated the relationship between the systemic BP, IOP and ocular blood flow (4). The study demonstrated that when IOP increased, the decrease in blood flow in the optic nerve was dependent on the systemic blood pressure. At higher pressures ocular blood flow showed no demonstrable change in ocular blood flow. However at lower systemic pressures, a significant decrease in ocular blood flow was evident. In humans, the lower level of auto regulation is determined at the mean BP of 35mmHg.[4] This translates into the understanding that increasing IOP in a setting of low systemic blood pressure will significantly drop OPP.

INTRAOCULAR PRESSURE

Normal intraocular pressure is about 27mm Hg. Prone positioning can increase the IOP up to three times the baseline pressure. This increases the risk in susceptible individuals for inadequate ocular nerve perfusion.[5] Hvidberg et all demonstrated the linear relationship between increasing PaCO2 and IOP, stressing the importance of ventilation on the IOP and ocular blood flow.[6]

To date there remains large retrospective studies and case reports on POVL and scientific evidence remains speculative. The following are current evidences on the existing literature on POVL.

INCIDENCE

Those at high risks are reported as patients undergoing spine surgery in prone positioning and patients undergoing cardiac bypass surgery. It is difficult to estimate the exact incidence and figures, despite recent publications of retrospective case control studies encompassing large number of surgeries and patients.[7,8] The accuracy of the true incidence is questionable as most data was extrapolated from billing codes, thus making

comparisons between reported complications difficult. Having established the limitations of the above analysis, the reported estimated incidence of ION lies between 0.1 overall and 0.3% in spine surgery.

CAUSES

The speculative causative factors contributing to the development of the ION are described below and are enumerated as patient related factors and surgical related factors. Patients related factors comprise age, sex, cup to disc ratio and concomitant risk factors.

a) Age
A retrospective analysis involving 5.6 million patients by Shen et al demonstrated that patients 18 years and younger undergoing spinal surgery were at higher risk to experience POVL in the form of cortical blindness than older patients. This held true for only spine surgery and was not demonstrated in other surgeries.[2] For patients over the age of 50 years, ION was the commonest form of POVL and again this was demonstrated following spine surgery.

b) Sex
Quoting the same study by Shen et al, men were found to be more affected than women with a 1.3 times higher Odds Ratio for all types of POVL. Men were also more frequent by a factor of two to develop ION.[2]

c) Type of surgery
Postoperative vision loss occurs more frequently in cardiac and orthopedic spine fusion surgery compared to other surgeries.[1,7]

Those undergoing lumbar spine surgery are likely to develop ION, particularly AION compared to those undergoing cervical or thoracic spine surgeries.[1]

d) Surgical experience, surgical emergencies and length of surgery
Surgical experience and likewise cases listed under elective and non elective surgery does not have any impact on the development of POVL.[8] A strong correlation between the duration of surgery and the development of ION is demonstrated in the ASA POVL registry. The registry has reported that most cases of ION reported were associated with surgery lasting more than 6 hours or longer. A staggering 94% of the cases reported were subjected to prolong surgeries.[9]

e) Positioning
Cheng et al demonstrated that IOP rises significantly in prone position.[5] This can potentially decrease the orbital perfusion pressures to critical values. Periorbital swelling and eye lid oedema are common in prone surgeries and does not contribute to the development of POVL.

CRAO arising from thromboembolism may occur as a consequence of head rotation as a result of showers of plaques from the anterior carotid artery.[10]

The use of different frames for positioning and Mayfield pins without any compression of the orbits does not seem to influence the incidence of POVL.[8]

f) Direct Compression
Increased IOP due to direct compression of the eye is implicated in POVL related to central retinal artery occlusion.[1]

g) Anatomical variations and size of the cup to disc ratio
Doro et al demonstrated that individuals with small cup to disc ratio are at an increased risk to develop ION.[11] How this translates unto the daily practice of the anaesthesiologists in prevention of ION remains to be sorted out.

Of importance with regards to the surgical related factors, prone positioning of the patient, duration of surgery and intraoperative blood losses are confirmed factors that increase susceptibility to development of ION.[8]

h) Increase venous pressure
Lee demonstrated that IOP is partially dependent on central venous pressure in prone positioning

of patients during spine surgery. This strong statement translates that increased IOP can result from an increase in venous pressure within the eye. Therefore large amounts of fluid infused during surgery can contribute to venous congestion of the eye. This is made worse with prone positioning.[12] IOP also increases with direct pressure on the abdomen as a consequence of increased venous pressure. The postulated mechanism is said to be an equivalent of a compartment syndrome at the level of the optic canal reducing arterial blood flow to the optic nerve.

i) Blood loss and anemia.
The ASA POVL registry reports blood loss more than 1000mls and more as one of the determinants for the development of POVL.[9] Interestingly and not surprisingly anemia is reported to increase the Odds Ratio for POVL in spine surgery. This finding did not hold true for cardiac and other surgeries. The report does not specify the haemoglobin or haematocrit levels. The lowest haematocrit and haemoglobin levels for all reported cases were 30%.[8]

j) Blood transfusion
Shen et al, reported, that amongst all the reviewed surgeries, spine surgery seemed to be the only one in which blood transfusion did not seem to be associated with POVL.[8]

k) Arterial hypotension
Intraoperative arterial hypotension and prolonged drops in systolic pressure have been reported as notorious perpetrators to the development of POVL in many cited case reports.[13] This statement has been disputed in the recent retrospective analysis, where hypotension was not found to be an independent risk factor.[8]

l) Vasopressor
There are anecdotal and theoretical literatures on the use of vasopressors .Currently, there exists no evidence to support the use or not of vasopressors. However, the use of direct acting vasoconstrictors (phenylephrine) is controversial for vasoconstriction of the arterioles of the optic nerve may ensure.

ANAESTHETIC CONSIDERATION

The importance of mitigating this potential catastrophe cannot be understated. Armed with the knowledge of risk factors, it is prudent to formulate the anaesthetic plan with the preoperative management.

At our local setting, sporadic reports of ION have increased the attention of both the surgeons and anaesthetists alike. Increasing the awareness of this catastrophic complication can allow for proper screening of high risk patients. These so called high-risk patients should be adequately pre warned of this potential complication.

The following measures should be considered in patients at risk for ION:

a) Planning of surgery, Positioning and Duration
Communication with the surgeon is beneficial and all concerns need to be brought forth, in an effort to decrease the length of time that the patient remains in prone position. Staging when feasible (anterior posterior spinal fusion), should be discussed with the surgeon.

Proper positioning with minimal compression on the abdomen and avoidance of any extrinsic direct pressure on the eyes should be made. Encourage a reverse Trendelenberg of 10 - 15% to decrease the venous and decrease IOP in the prone position. It is prudent to avoid venous congestion in prolonged surgery (longer than 6 hours) and where blood loss of more than 1 liter occurs. A recent literature review of case reports and retrospective studies indentified a staggering incidence of 83% of ION, with the above stated as major culprits.[14]

b) Hemodynamic Management.
Arterial hypotension should be avoided and blood pressure should be maintained at preoperative levels. No concrete evidence is available determining the lower limit of tolerance for adequate perfusion pressure of the optic nerve.

It is advisable to avoid anaemia and current literature suggests keeping haemoglobin around 100g/l and haematocrit around 30%.[8]

As with all types of surgery, balanced fluid management is advisable. Infusion of large amount of crystalloids with decreasing serum osmolality should be avoided as this could further contribute to an increase in compartmental pressures and venous congestions.

c) Ventilation
Normoventilation and avoidance of hypercapnia will prevent increases in IOP during surgery.[5]

TREATMENT

Though not routinely performed in our local practice, it would be advisable that visual field examination be included in the immediate postoperative screening of high risk patients. Follow up visits should be recommended as a substantial number of patients have no presenting symptoms upon awakening from anaesthesia.

Immediate consultation with an ophthalmologist is imperative if the patient reports any visual symptoms. Available symptomatic treatment should be instituted immediately without further delays in awaiting confirmation of diagnosis. Treatment options and protocols are available and include the use of mannitol and intravenous steroids. Head elevation in an effort to decrease any venous congestion is encouraged. Anecdotel reports on elevation of mean arterial blood pressure and correction of haemoglobin have been reported as part of the treatment protocol. The surgical treatment such as optic nerve fenestration is rarely performed due to its poor results.

CONCLUSION

ION remains a devastating complication following anaesthesia particularly in lumbar spine surgeries in the prone position[15] and in cardiac surgeries.

The challenge of ensuring the safety of the patient under anaesthesia is our responsibility to prevent devastating complications. While acknowledging that risk factors remain speculative and prospective studies are unfeasible due to the ethical nature involved, what remains to be highlighted is the understanding of the basic anatomy and physiology of the eye and preventive measures that need to be taken.
The American Society of Anesthesiologists Task Force on Perioperative Blindness in 2006 have enumerated assumed risk factors to be prolonged surgery, prone surgery, anaemia, increased surgical blood loss, and prolonged arterial hypotension.[16]

Armed with the above knowledge of risk factors, what remains within the direct control of prevention is aggressive and sound preoperative assessment and intraoperative management to maintain all physiological parameters within the norm, and the importance of postoperative screening to ensure good outcome.

References

1. Myers, M.A., et al., Visual loss as a complication of spine surgery. A review of 37 cases. *Spine (Phila Pa 1976)* 1997;**22**(12):1325-9.
2. Ho, V.T., et al., Ischemic optic neuropathy following spine surgery. *J Neurosurg Anesthesiol* 2005;**17**(1):38-44.
3. Hayreh, S.S., Ischemic optic neuropathy. *Prog Retin Eye Res* 2009;**28**(1):34-62.
4. Liang, Y., et al., Impact of systemic blood pressure on the relationship between intraocular pressure and blood flow in the optic nerve head of nonhuman primates. *Invest Ophthalmol Vis Sci* 2009;**50**(5):2154-60.
5. Cheng, M.A., et al., The effect of prone positioning on intraocular pressure in anesthetized patients. *Anesthesiology* 2001;**95**(6):1351-5.

6. Hvidberg, A., S.V. Kessing, and A. Fernandes, Effect of changes in PCO2 and body positions on intraocular pressure during general anaesthesia. *Acta Ophthalmol (Copenh)* 1981;**59**(4):465-75.
7. Holy, S.E., et al., Perioperative ischemic optic neuropathy: a case control analysis of 126,666 surgical procedures at a single institution. *Anesthesiology* 2009;**110**(2):246-53.
8. Shen, Y., M. Drum, and S. Roth, The prevalence of perioperative visual loss in the United States: a 10-year study from 1996 to 2005 of spinal, orthopedic, cardiac, and general surgery. *Anesth Analg* 2009;**109**(5):1534-45.
9. Lee, L.A., et al., The American Society of Anesthesiologists Postoperative Visual Loss Registry: analysis of 93 spine surgery cases with postoperative visual loss. *Anesthesiology* 2006;**105**(4):652-9; quiz 867-8.
10. Delattre, O., et al., Spinal surgery and ophthalmic complications: a French survey with review of 17 cases. *J Spinal Disord Tech* 2007;**20**(4):302-7.
11. Doro, S. and S. Lessell, Cup-disc ratio and ischemic optic neuropathy. *Arch Ophthalmol* 1985;**103**(8):1143-4.
12. Rizzo, J.F., 3rd and S. Lessell, Posterior ischemic optic neuropathy during general surgery. *Am J Ophthalmol* 1987;**103**(6):808-11.
13. Brown, R.H., J.F. Schauble, and N.R. Miller, Anemia and hypotension as contributors to perioperative loss of vision. *Anesthesiology* 1994;**80**(1):222-6.
14. Lee, L.A., et al., Postoperative ischemic optic neuropathy. *Spine (Phila Pa 1976)* 2010;**35**(9 Suppl):S105-16.
15. Tempelhoff, R., An optic nerve at risk and a prolonged surgery in the prone position: time to reconsider? *Anesthesiology* 2008;**108**(5):775-6.
16. American Society of Anesthesiologists Task Force on Perioperative, B., Practice advisory for perioperative visual loss associated with spine surgery: a report by the American Society of Anesthesiologists Task Force on Perioperative Blindness. *Anesthesiology* 2006;**104**(6):1319-28.

Aeromedical Transportation of the Critically Ill: A Review

Gunalan Palari Arumugam
Consultant Anaesthesiologist, Hospital Pantai Ipoh

HISTORY

Since the invention of airplanes in the early 1900s, man has conquered the skies for various reasons, be it social, economic or political dominance. Distances that used to take days and months were covered in hours. With the popularity of air travel in recent times, flying on a plane is now considered a norm for the young to the old rather than a luxury. Although the science of aeromedicine is relatively new, there have been many applications of its use, especially in transportation of the critically ill patient. Aeromedical services are now considered an integral part of any modern emergency health care system. The origins of aeromedical transportation unfortunately had its beginning in rather depressing states of war to be exact the First World War where wounded soldiers were flown off to the nearest medical facility. The first organized aeromedical services, although primitive by today's standards were credited to the Inland Mission's Aerial Medical Service in 1928, the service which is now popularly known as the Royal Flying Doctor Service of Australia.

It was not till the Korean War in the late 1950s and later the Vietnam War in the early 1970s that aeromedical services had shown a clear impact on improving care for the injured. Over 400 000 patients were airlifted to hospitals during the two conflicts. Helicopters were used for the rapid removal of injured troops from close to the point of injury. The concept of 'scoop and run' was first coined here as there was hardly any time to do any meaningful life-saving procedures while dodging enemy bullets. These services may have accounted, at least partly, for the much lower mortality rate of those wounded in the conflicts when compared with those injured in previous wars.

The success of the military paved the way for civilian application of these services. Civilian operators slowly began to embrace the concept of transporting patients especially from areas such as highways, remote, inhospitable or difficult terrain. Among the world's first civilian helicopter air ambulance services was the Swiss Air Rescue Association (known as REGA) which started in 1952 mainly helping out people in mountainous regions of the Swiss Alps. By the late 1960s and early 1970s most countries in Europe and North America started providing their own civilian and not military operated helicopters and airplanes to provide services. This then evolved into a much more established service which included interhospital transportations as well as use of the services for organ procurement under the transplant programme. Running parallel to helicopter services were services provided by the fixed wing operators mainly using turbo propeller as well as jet planes that are custom built for aeromedical purposes. For the long distance transportations across continents, for a select group of patients they can be safely moved on commercial airlines as well.

In Malaysia, aeromedical transportation has been in place for more than 3 decades. The Sarawak Flying Doctor Service was first introduced in 1973 to provide basic health services to people living in remote areas. The State of Sabah is now on the verge of becoming the first state in Malaysia to have a dedicated rotary wing services for interhospital transfers. The Military has also played its part by providing both the Nuri Helicopters as well as the C130 Hercules for critically ill patients who require advance medical care in the bigger cities. The Fire and Rescue Department based out of Subang have also performed mercy flights for medical emergencies especially involving the Orang Asli community.

ISSUES/ADAPTATIONS WHILE UNDERGOING AEROMEDICAL TRANSPORTATION

The majority of studies conducted to determine the effect of the flight environment to the body and mind were mostly done by the military as well as the aerospace industry as manned missions to the moon and outer space resulted in an explosion of interest in the science over the last couple of decades. For the military the amount of investment spent in the technology necessitated both protection of the money spent in ensuring that only the fittest pilots are chosen to command the fighter planes without causing harm to them and the property. As a result of research on the aircrew, most of the findings of these studies were usually accepted as best practices for the civilian environment as well. Most patients will usually be accompanied by physicians who are trained to handle emergencies while inflight. They are also expected to understand the various physiological and physical constraints that may have an effect on the patient being transferred. In brief, some of these constraints can be divided into 2 broad categories:

A. Stresses of the Flight Environment to the Patient

i. Hypoxia can occur as a result of decreased amount of oxygen in cabin atmosphere despite cabin pressurization. Cabin pressures are typically kept at between 6000 to 8000 feet above sea level at a cruising altitude of 35,000 to 40,000 feet. This level is usually associated with a drop of 25% of the partial pressure of oxygen when compared to sea level. Most passengers are able to tolerate these levels, but there are some specifically with respiratory or cardiac diseases that may have some element of desaturation which cannot be tolerated for long periods of time. Typically these changes are seen for patients with pneumonia, chronic obstructive airway disease, recent myocardial infarction or heart failure and sepsis. Supplemental oxygen will usually be required for these patients. Some patients with pre-existing obstructive sleep apnea who are dependent on their CPAP machines are now allowed to bring the portable machines on board with advanced airlines clearance provided that the machines are compatible with the airlines in-flight power supply.

ii. Using the principles of Boyles Law where gas expands with increase in elevation and drop in atmospheric pressure 1 liter of air at sea level will swell to 1.5 liter at 10,000ft. These changes in barometric pressure can have an effect on patients who may have trapped gas in closed spaces. Those with sinusitis, ear infection, pneumothorax, post abdominal surgery and laparoscopic surgeries as well as patients with retinal detachment who may have had SF6 gas bubble injection are at risk of experiencing gas expansion.

iii. Cabin pressurization is a function of the aircraft that allows a passenger to tolerate flying without significant physical and physiological derangements that could occur as explained above. On ground with doors open, cabin & ambient pressure is the same but as the aircraft takes off after the doors are closed, there is ambient air that is fed into the cabin, whilst valves (at the rear) control the release. These valves act to maintain the cabin pressure at a controlled rate (normally while climbing at 300ft per minute to the desired altitude and while descending at 500ft per minute to the desired altitude). The maximum allowable differential limit between ambient and internal pressure is determined by the aircraft manufacturer and is purely a function of design of the fuselage in other words the strength/weight requirements of the aircraft. For some medical conditions where flying at sea level cabin pressure is needed, these usually will require air ambulances and the pilots will have to be informed prior to the launch of the mission. If they are to maintain sea level cabin

pressure throughout the flight, the altitude of the aircraft is limited and there will be increased resistance to the flight, resulting in more fuel consumption and more stops for refueling. A typical example of when a sea level cabin pressure will be mandatory is when transporting a diver who has Caisson's disease. When divers ascend too rapidly, nitrogen bubbles that form in blood can expand and cause acute gas embolism that can be fatal if flying on normal cabin pressure of 6000 to 8000 feet.

iv. Gravitational forces refer to the effect of gravity as well as acceleration forces as the airplane reaches its cruising speed and deceleration forces as the plane comes to a stop. Some hemodynamic changes as well as shift in fluid distribution maybe seen, but whether it is clinically significant and requiring a change in patient position (head up or down) is yet to be proven.

v. Thermal refers to the cold cabin environment that may be of concern especially when transporting neonates. Most of them will require nursing care in transport incubators.

vi. Vibration effects more commonly occurs in rotary wing rather than fixed transfers and may worsen motion sickness or increase incidences of nausea and vomiting as such prophylactic anti-emetics will usually be given prior to any transfers.

vii. Increased noise levels as a result of turbulence and movement of people in small confined spaces decreases the ability to relax or concentrate. Typically, clients who have had recent hospitalization for mental illness (esp. manic depression or schizophrenia), while may be stable on ground, may "lose it" in noisy environment. Therefore we upgrade (fewer people around, and quieter), and the medical team is usually prepared with a preloaded syringe of sedatives in the event it is needed.

viii. Lower limb swelling and fluid accumulation is third spaces most commonly on long-distance flights. These changes are especially important for those in casts or with chronic heart or lung problems. The patients are upgraded to business class seats for extra space and the ability to elevate the lower limbs to increase circulation. DVT prophylaxis is usually given for the high risk group such as those who are obese and have had recent surgery especially lower limb operations provided no contraindications.

ix. Fatigue is the end-product of all the other stresses associated with altitude and flight especially noise, vibration and hypoxia. Deconditioned client (in hospital for extended period, or even short stay for the chronically ill) are at higher risk.

B. Patient factors which are anything that places a strain on the ability of a human to perform at their optimum level. These factors are divided into

i. Physical: Weight (neonates will need special incubators to deal with hypothermia), overweight patients may need specially adapted stretchers and specific planes, some doors maybe too small to allow loading), height (patients who are too tall and need lower limb elevation may need first or business class seating arrangements.

ii. Psychological: Most are anxious to return home to their normal surroundings; as such they are visibly more anxious. Some patients may have been in a deprived sleep state causing them to be more fatigued and irritable and will most often require an anxiolytic.

iii. Physiological: These are factors related to the main organ functions such as cardiovascular and respiratory which are the 2 main systems that receive the closest scrutiny during transfers. Hemodynamic parameters as well as oxygen requirements are closely

watched prior to the transfer itself so that any required equipment and drugs such as syringe pumps, inotropes, oxygen tanks or portable oxygen concentrators are prepared in advance.

iv. Psychosocial: There are numerous motivation factors such as financial difficulties and other personal commitments that may cloud the patient's or the family's decision in wanting to undergo the transportation earlier than would be medically recommended. Any decision to be made should not be at the expense of risk to life and is guided by the treating physicians as well as the team involved in the transfer.

v. Pathological processes refers to the disease for which the patient is suffering from for which the aeromedical transportation is being conducted or a disease from which he has recovered and is now being transported to his residence. Both the current as well as the patient's pre-existing medical conditions are obtained in detail so that adequate preparation is made to anticipate any events that can happen during the transfer.

RECENT ADVANCEMENTS

It is without a doubt that oxygen is almost always required and it is in this area that quite a number of significant changes have occurred over the past few decades. From what used to be a situation where a number of bulky cylinders of oxygen were used to be carried, we now have portable oxygen concentrators that can be used for patients who are not requiring a high flow rate.

For air ambulance transfers, high pressure oxygen cylinders are slowly being replaced with liquid oxygen cylinders, as such the jet air ambulance will only need to have few stops for refueling purposes and not necessarily replacement of oxygen tanks. Previously, the jet either has to carry all the required amount of oxygen on board or have them replaced at each refueling stops as required. However, the Federal Aviation Authority (FAA) has not approved use of liquid oxygen on commercial flight. However, with the incident on flight QF30 in 2008 where failure of a high pressure oxygen cylinder caused rupture of the aircrafts fuselage which led to rapid cabin decompression and necessitated an emergency landing in Manila, it may cause a slight rethink on whether liquid oxygen can be a viable option.

Oxygen is highly flammable and none the more when it is used in areas where there is heavy artillery fired directed against a plane or helicopter. With that in mind, there is new equipment called MOVES which is the acronym for Monitoring, Oxygen, Ventilation and Suction that has been developed by Thornhill Research based in Canada and being tested currently by the US military. This machine allows a patient to be ventilated with multiple modes to a Fi02 of about 80 to 85% without use of compressed oxygen cylinders but using the ambient air alone and at the same time allows monitoring of ECG, capnography, invasive and non-invasive pressures as well as suction. The entire machine replaces individual pieces of equipment that has to be carried separately, thus making MOVES highly attractive for emergency responders in disaster and war torn areas.

FUTURE IN MALAYSIA

Of late, there have been a number of clinical specialists who have shown interest to develop the field of aeromedicine further in the country. Interested clinicians, namely from the three disciplines quite closely linked to this field which is Aviation Medicine, Anaesthesiology and Emergency Medicine have embarked on training programmes of late and will look at forging more ties internationally where some of our doctors can go for training as well as working with aviation providers locally to further enhance both interhospital transfers as well as emergency first response especially for road trauma where delays to get immediate medical attention to the victims still exist.

References

1. Martin T Aeromedical transportation: A clinical guide. 2nd ed. : Ashgate; 2006
2. Reinhart R Basic flight physiology. 3rd ed.: McGraw-Hills; 2008.
3. Sarawak State Health Department. [homepage on the Internet]. No date [cited 2011 Dec 1]. Available from: The Sarawak State Health Department, Web site: http://jknsarawak.moh.gov.my/en
4. Australian Transport Safety Bureau. ATSB interim factual report into the Qantas Boeing 747 depressurisation occurrence, 475 km north-west of Manila, Philippines, 25 July 2008. [homepage on the Internet]. 2009 March 6 [cited 2011 Dec 1] Available from:, Web site: http://www.atsb.gov.au/newsroom/2009/release/2009_03.aspx
5. Thornhill Research Inc., MOVES. [homepage on the Internet]. 2011 [cited 2011 Dec 1]. Available from:, Web site: http://www.thornhillresearch.com/p-moves.html

N-Acetylcysteine in The Treatment of Non-Paracetamol Induced Acute Liver Failure

Tai Li Ling
Department of Anaesthesia and Intensive Care, Hospital Kuala Lumpur

Acute liver failure (ALF) is a condition in which there is a rapid decline in hepatic function characterised by jaundice, coagulopathy (international normalised ratio, INR ≥ 1.5) and hepatic encephalopathy in patients with no evidence of prior liver disease.[1] A classification system proposed by O'Grady and colleagues used the terms *hyperacute, acute and subacute* hepatic failure to describe patients in whom encephalopathy develops within 7 days, 7 – 28 days and 4 – 12 weeks of the onset of jaundice, respectively.[2] Although jaundice is considered a defining feature of ALF, it may not always be present, particularly in hyperacute presentations.

Paracetamol overdose is the most common cause of ALF in the USA and western Europe, while in the developing countries, infections by hepatitis A, B and D viruses predominate.[3,4,5] Other causes of acute liver failure include other drug-induced hepatitis, autoimmune hepatitis, ischaemic injury as a result of systemic hypotension in sepsis or cardiac failure, Wilson disease, fatty liver of pregnancy, and HELLP (haemolysis, elevated liver enzymes, low platelets) syndrome.

ALF is relatively uncommon but remains a rapidly progressive and frequently fatal condition. Many ALF patients develop infectious, cardiopulmonary, or renal complications that can progress to multi-organ failure. Because of its rarity, ALF has been difficult to study in depth and very few controlled therapy trials have been performed. Short-term transplant-free survival varied greatly, from 67% for patients with paracetamol-related liver failure to 25% and 17% for those with other drug reactions and liver failure of indeterminate cause, respectively. Liver transplantation has improved survival in these patients and currently, overall one-year survival with transplantation is greater than 65%.[4] However, liver transplantation is not readily available in many countries and alternative therapies need to be explored.

N-acetylcysteine (NAC) has been used in the treatment of paracetamol overdose for more than 30 years.[8] Early NAC administration is highly effective in preventing hepatocellular necrosis, by acting as an antidote in replenishing depleted stores of glutathione.[9,10] The effects of NAC seem to go beyond this, as there is evidence that continued infusion of NAC has beneficial effects in patients with established paracetamol-induced ALF, suggesting that it works through other mechanisms in the treatment of ALF.[11] Emerging data suggest that NAC may also have a role in non-paracetamol-induced ALF.

N-ACTEYLCYSTEINE AS A PRECURSOR OF GLUTATHIONE

Glutathione (γ-glutamylcysteinylglycine; GSH) is the primary endogenous anti-oxidant in the body and the liver and lungs are the primary sites of synthesis. It is formed from 3 amino acids i.e. glutamatic acid, cysteine and glycine. Cysteine, a sulphur-containing amino acid is the "rate-limiting" amino-acid for the production of glutathione. NAC is the acetylated derivative of the amino acid L-cysteine and is a precursor for glutathione.

In acute paracetamol overdose, metabolism of paracetamol by conjugation becomes saturated, and excess paracetamol is oxidatively metabolised by the CYP to the reactive metabolite N-acetyl-p-benzoquinone (NAPQI). NAPQI has an extremely short half-life and is rapidly conjugated with glutathione, a sulfhydryl donor, and is renally excreted. Under conditions of excessive NAPQI formation, or reduction in glutathione stores by approximately 70%, NAPQI covalently binds to the cysteinyl sulfhydryl groups of cellular proteins, forming NAPQI-protein adducts that mediate mitochondrial injury, leading to hepatocyte death and ultimately centrilobular liver necrosis. Similar

enzymatic reactions occur in extrahepatic organs, e.g. the kidney, and contribute to some degree of extrahepatic organ dysfunction.

N-ACETYLCYSTEINE AS AN ANTI-OXIDANT AND ANTI-INFLAMMATORY AGENT

NAC is also known as a thiol anti-oxidant as it contains a sulfyhdryl (–SH) group. The hydrogen atom in the sulfhydryl group acts as an electron for neutralising free radicals like superoxide radical (O_2-), hydroxyl radical (OH-) and hydrogen peroxide (H_2O_2).

In acute liver injury, hepatocytes death initiates immunological reactions, including both innate and adaptive immune responses, resulting in the activation of other cells of the innate immune system, including Kupffer cells (KC), natural killer (NK) cells, and natural killer T (NKT) cells. These cells contribute to the progression of liver injury by producing proinfammatory mediators and secreting chemokines to further recruit infammatory cells to the liver. KC produce various cytokines and other mediators, including prostanoids, nitric oxide, and reactive oxygen radicals.

N-ACETYLCYSTEINE IMPROVES HAEMODYNAMICS AND OXYGEN TRANSPORT

NAC enhances the activity of nitric oxide-soluble guanylate cyclase system to generate the second messenger cyclic guanosine monophosphate (cGMP). cGMP activates protein kinase resulting in phosphorylation of protein that regulates smooth muscle relaxation. The resultant microcirculatory vasodilation improves blood flow and enhances oxygen delivery to peripheral tissues. It had been shown that plasma levels of cyclic cGMP were increased further by 200% after NAC infusion in ALF.[12]

In a small, uncontrolled study it was suggested that NAC improves oxygen delivery and consumption when used both in paracetamol and non-paracetamol-induced ALF.[13]

N-ACETYLCYSTEINE IN NON-PARACETAMOL-INDUCED ALF: CURRENT CLINICAL EVIDENCE

The role of NAC in limiting liver injury and improving prognosis in patients with ALF following paracetamol overdose has been well documented in large case series and a small, controlled trial.[10,14,15,16]

NAC has shown efficacy in non-paracetamol-induced ALF as well. There are two separate case reports on two children who survived ALF induced by clove oil poisoning who were treated with intravenous NAC.[17,18] In a case series on the use of intravenous NAC in non-paracetamol-induced ALF, four of seven patients survived.[19] There was also significant improvement in mean peak prothrombin time, serum factor V, aspartate aminotransferase and alanine aminotransferase levels in the four patients who had full recovery.

A case series on the use of intravenous NAC in eight adults with serologically confirmed dengue-associated ALF was reported from Sri Lanka.[20] All five patients with encephalopathy grades I – II recovered completely and well at follow-up at 2 months, while the three patients with encephalopathy grades III – IV died. No patients had adverse events attributable to NAC. In Singapore, Lim reported on a child with dengue-associated fulminant liver failure who was treated successfully with intravenous NAC.[21]

In a case-control study, Mumtaz et al. studied the role of oral NAC in 47 patients with non-paracetamol induced ALF.[22] More than 90 % of the cases of ALF were caused by acute viral hepatitis. There was a statistically significant survival in the NAC treated group, 47% vs. 27% in the control group.

A prospective, randomised, double-blinded, placebo-controlled trial of intravenous NAC in ALF not caused by paracetamol overdose was carried out by the US Acute Liver Failure Study Group.[23] Spontaneous survival in subjects with grade I - II hepatic encephalopathy at randomisation was significantly better in those who received NAC compared to those who received placebo (52% vs. 30%, $p = 0.02$). However, the primary end point of

the study, overall survival (spontaneous survival and survival after liver transplant) at 3 weeks, was not significantly better in patients who received NAC than in those who received placebo (70% vs. 67%, $p = 0.57$). Spontaneous survival was not improved in NAC recipients with grade III – IV encephalopathy compared to those in the placebo group (9% vs. 22%, $p = 0.18$). NAC appears to be favorable for drug-induced and hepatitis B virus-associated ALF, and less favorable for autoimmune hepatitis-induced ALF.

A retrospective review of 170 children with non-paracetamol-induced ALF in King's College Hospital suggested that NAC administration was associated with a shorter length of hospital stay, higher incidence of spontaneous survival and better survival after liver transplantation over children treated with standard care without NAC.[24] However, a recent randomised, double-blinded, placebo-controlled study presented at the 2011 Digestive Disease Week meeting showed that intravenous NAC does not improve one-year survival in paediatric patients with non-paracetamol-induced ALF.[25] One-year survival with NAC was 73%, compared with 82% with placebo ($p = 0.20$). Moreover, NAC administration was associated with a significantly lower probability of one-year transplant-free survival compared with placebo (35% vs. 53%, $p = 0.04$).

DOSAGE RECOMMENDATION OF *N*-ACETYLCYSTEINE

Different regimens of NAC have been used in the treatment of paracetamol overdose. NAC may be administered orally or intravenously. Intravenous NAC is more widely used. The initial loading dose of 150 mg/kg is infused over a period of 15 - 60 minutes, followed by an infusion of 50 mg/kg over a 4-hour period, and finally an infusion of 100 mg/kg over 16 hours. The dose does not require adjustment for renal or hepatic impairment.

There is suggestion that the 20-hour intravenous NAC regimen is suboptimal in patients with delayed presentation, chronic paracetamol overdose or in whom ALF develops. It has been raised recently in the literature that the treatment course of NAC should be based on clinical end points.[26] The Consensus Statement on guidelines for the management of paracetamol poisoning in Australia and New Zealand recommends that NAC be continued at the rate of 6.25 mg/kg/hr (150 mg/kg/24 hours) until there is clinical and biochemical evidence of improvement.[27] It is suggested for treatment to be continued till encephalopathy resolves and the alanine aminotransferase, creatinine concentrations and INR have substantially improved or until the patient is transferred to operation theatre for liver transplantation.

The regimen for intravenous NAC in non-paracetamol-induced ALF has not been established. The dose for paracetamol overdose is typically used.

SIDE-EFFECTS AND TOXICITY OF *N*-ACETYLCYSTEINE

NAC is a safe drug with a wide toxic-therapeutic window. The most common side-effects associated with NAC are nausea, vomiting, diarrhoea, abdominal pain. Rarely, it can cause rashes, fever, headache and drowsiness. Infrequently, allergic reactions due to histamine release occur and can consist of rash, pruritis, angioedema, bronchospasm, tachycardia, and hypotension. Most of these side-effects are self-limited or resolved with either the use of antihistamine drugs or by lowering the infusion rate.

Based on information in the Hunter Area Toxicology Service database in Australia, 9.3% of the 399 patients who were treated with intravenous NAC for paracetamol toxicity, had an adverse drug reaction.[28] Anaphylactoid reactions occurred in 1.8% of all patients treated with intravenous NAC. None of the deaths in paracetamol overdose was attributed to NAC.

Adverse effects which included maculopapular rash, cardiac reactions (bradycardia, tachycardia), mild dizziness and peripheral oedema were noted in only 11% of the 111 children treated with

intravenous NAC for non-paracetamol induced ALF in the King's College Hospital.[24] NAC was discontinued in a child who developed a florid maculopapular rash and bronchospasm due to an allergic reaction to NAC.

CONCLUSION

There is current evidence to suggest that NAC may be beneficial in adults with early grades of encephalopathy resulting from non-paracetamol-induced ALF. However, it does not support the broad use of NAC in non-paracetamol-induced ALF in children.

References

1. Polson J, Lee WM. American Association for the Study of Liver Disease. AASLD position paper: the management of acute liver failure. *Hepatology* 2005;**41**:1179–1197.
2. O'Grady JG, Schalm S,Williams R. Acute liver failure: redefining the syndromes. *Lancet* 1993;**342**:273-275.
3. Hadem J, Stiefel P, Bahr MJ, et al. Prognostic implications of lactate, bilirubin, and etiology in German patients with acute liver failure. *Clin Gastroenterol Hepatology* 2008;**6**:339–345.
4. Ostapowicz G, Fontana RJ, Schiodt FV. Results of a prospective study of acute liver failure at 17 tertiary care centers in the United States. *Ann Intern Med* 2002;**137**:947–954.
5. Sarwar S, Khan AA, Alam A. Predictors of fatal outcome in fulminant hepatic failure. *J Coll Physicians Surg Pak* 2006;**16**:112–116.
6. Khuroo MS, Kamili S. Aetiology and prognostic factors in acute liver failure in India. *J Viral Hepatitis* 2003;**10**:224–231.
7. Mudawi HMY, Yousif BA. Fulminant hepatic failure in an African setting: etiology, clinical course, and predictors of mortality. *Dig Dis Sci* 2007;**52**:3266–3269.
8. Scalley RD, Conner CS. Acetaminophen poisoning: a case report of the use of acetylcysteine. *Am J Hosp Pharm* 1978;**35**:964–967
9. Prescott LF, Illingworth RN, Critchley JA, Stewart MJ, Adam RD, Proudfoot AT. Intravenous *N*-acetylcystine: the treatment of choice for paracetamol poisoning. *Br Med J* 1979;**2**:1097-1100.
10. Smilkestein MJ, Knapp JL, Kulig KW, Rumak BH. Efficacy of oral *N*-acetylcysteine in the treatment of acetaminophen overdose. *N Engl J Med* 1988;**319**:1557-1562.
11. Harrison PM, Keays R, Bray GP, Alexander GJ, Williams R. Improved outcome of paracetamol-induced fulminant hepatic failure by late administration of acetylcysteine. *Lancet* 1990;**335**:1572-1573.
12. Harrison P, Wendon J, Williams R. Evidence of increased guanylate cyclase activation by acetylcysteine in fulminant hepatic failure. *Hepatol* 1996;**23**:1067-1072.
13. Harrison PM, Wendon JA, Gimson AE, Alexander GJ, Williams R. Improvement by acetylcysteine of hemodynamics and oxygen transport in fulminant hepatic failure. *N Engl J Med* 1991;**324**:1852-1857.
14. Smilkstein, M. J. et al. Acetaminophen overdose: a 48-hour intravenous *N*-acetylcysteine treatment protocol. *Ann Emerg Med* 1991;**20**:1058–1063.
15. Harrison PM, Keays, R, Bray GP, Alexander GJ, Williams R. improved outcome of paracetamol-induced fulminant hepatic failure by late administration of acetylcysteine. *Lancet* 1990;**335**:1572–1573.
16. Keays R, Harrison PM, Wendon JA, Forbes A, Gove C, Alexander GJ, Williams R. Intravenous acetylcysteine in paracetamol induced fulminant hepatic failure: a prospective controlled trial. *Br Med J* 1991;**303**:1026–1029.
17. Janes SE, Price CS, Thomas D. Essential oil poisoning: *N*-acetylcysteine for eugenol-induced hepatic failure and analysis of a national database. *Eur J Pediatr* 2005;**164**:520-522
18. Eisen JS, Koren G, Juurlink DN, Ng VL. *N*-acetylcysteine for the treatment of clove oil-induced fulminant hepatic failure. *J Clin Toxicol* 2004;**42**:89-92
19. Ben-Ari Z, Vaknin H, Tur-Kaspa R. *N*-acetylcysteine in acute hepatic failure (non-paracetamol induced). *Hepatogastroenterol* 2000;**47**:786-789.
20. Kumarasena RS, Senanayake SM, Sivaraman K, de Silva AP. Intravenous *N*-acetylcysteine in dengue-associated acute liver failure. *Hepatol Int* 2010;**4**:533-534
21. Lim G, Lee JH. *N*-acetylcysteine in children with dengue-associated liver failure: A case report. *J Trop Pediatr* 2011; First published online: December 23, 2011

22. Mumtaz K, Azam Z, Hamid S, Abid S, Memon S, Shah HA. Role of *N*-acetylcysteine in adults with non-acetaminophen induced acute liver failure in a center without the facility of liver transplantation. *Hepatol Int* 2009;**3**:563–570
23. Lee WM, Hynan LS, Rossaro L, Fontana RJ, Stravitz RT, Larson AM, et al. The Acute Liver Failure Study Group. Intravenous *N*-acetylcysteine improves transplant-free survival in early stage nonacetaminophen acute liver failure. *Gastroenterology* 2009;**137**:856–864
24. Kortsalioudaki C, Taylor RM, Cheeseman P, Bansal S, Mieli-Vergani G, Dhawan A. Safety and efficacy of *N*-acetylcysteine in children with non-acetaminophen-induced acute liver failure. *Liver Transpl* 2008;**14**:25-30.
25. Squires R, Dhawan A, Alonso E, Narkewicet M. A prospective clinical trial shows that intravenous *N*-acetylcysteine (NAC) does not improve survival in pediatric patients with non-acetaminophen acute liver failure. *Gastroenterology* 2011;**140**:S-897
26. Heard KJ. Acetylcysteine for acetaminophen poisoning. *N Engl J Med* 2008;**359**:285-292.
27. Daly FF, Fountain JS, Murray L, Graudins A, Buckley NA: Guide- lines for the management of paracetamol poisoning in Australia and New Zealand - explanation and elaboration: a consensus statement from clinical toxicologists consulting to the Australasian poisons information centres. *Med J Aust* 2008;**188**:296-301.
28. Ian M Whyte, Barbara Francis, Andrew H Dawson. Safety and efficacy of intravenous *N*-acetylcysteine for acetaminophen overdose: analysis of the Hunter Area Toxicology Service (HATS) database. *Curr Med Res Opin* 2007;**23**:2359-2368

Robotic Urosurgery: Ventilating the Difficult Lung, A Retrospective Study in National Urology Institute, Kuala Lumpur Hospital

Amiruddin N. M. Kamil, Azrin M. Azidin, Noorulhana S. Hadzrami
Department of Anaesthesiology and Intensive Care, Hospital Kuala Lumpur, Malaysia

INTRODUCTION

Robotic surgery refers to a surgical technology that works on a computer-assisted electromechanical device as an interface between the surgeon and the patient.[1] It functions by mechanically translating surgeon's movements from a visually enhanced master console to a remote surgical cart which houses mechanical arms that mimics the same movements on the patients. This achieves a higher degree of precision and control than normally possible.

The use of robotic-assisted laparoscopic prostatectomy (RALP) for prostate cancer has grown tremendously in Malaysia over the last 5 years. Current estimates indicate that around 80% of all prostatectomies in the National Urology Institute, Malaysia are done robotically,[2] and this figure is likely to increase as more surgeons gain robotic experience. RALP is the most common procedure done among all urological robotic procedures and it is the most physiologically demanding for patients and challenging to the attending anaesthesiologists. Other urological procedures commonly done with robotic assistance are nephrectomy, pyeloplasty and radical cystectomy.

Difficulty in achieving adequate ventilation is one of the common challenges encountered during robotic procedures.[3] Major contributing factors for this are the Trendelenburg position for optimal surgical access and the use of carbon dioxide pneumoperitoneum. High ventilatory pressures with appropriate minute ventilation are required to offset pressures from low compliant lungs in an attempt to maintain normocarbia and minimize acid base changes. Other anaesthetic considerations which contribute to the effects on ventilatory parameters, are interstitial lung oedema, or rarely; a potential venous air embolism and deep venous thrombosis. There should be low tolerance for conversion to open surgery should any problems arise.[4]

OBJECTIVE

We evaluated anaesthetic data on robotic urological surgery done in Kuala Lumpur Hospital from year 2008 - 2010. In particular, we observed the ventilatory pressures changes and the subsequent acid base changes during a particular robotic procedure.

METHODOLOGY

This is a retrospective study of the anaesthetic and ventilatory management for robotic urological procedures. The data was extracted from the patient's surgical notes, anaesthesia chart and also from the local audit form, which every anaesthesiologist has to fill when anaesthetising patients for a robotic procedure. Data collected revealed that all patients for robotic procedures had general anaesthesia, routine and invasive haemodynamic monitors (arterial line, with or without central venous line), IPPV with pressure control ventilation, fluid therapy guided by serial arterial blood gases which was done 2-hourly or more frequent than that.

Both robotic prostatectomy and cystectomy groups were performed in supine position, with maximal Trendelenburg (40^0 head-down), while nephrectomy and pyeloplasty groups were performed in lateral position, with minimal Trendelenburg of 25^0 head-down. Observed data include demography, BMI, amount of fluid used, estimated blood loss, duration of surgery, ventilatory and airway pressures, and acid base changes. All obtained data were tabulated and analyzed using SPSS software version 16.

RESULTS

A total of 73 patients underwent urological robotic procedures in National Urology Institute from 2008 to 2010. Robotic prostatectomy had the highest number of patients (48 patients), compared to other procedures such as robotic cystectomy (or cystoprostatectomy) which involved 16 patients, followed by robotic pyeloplasty (5 patients) and lastly, robotic nephrectomy (4 patients).

The mean age was older (>60 years old) with higher BMI (>25 BMI) in prostatectomy, cystectomy and nephrectomy groups compared to those in the pyeloplasty group (<22 years old, <18 BMI). Total fluid used was highest in robotic cystectomy group due to long hour surgery, followed by robotic prostatectomy and others. The total fluid infused per hour between the groups is highest in the nephrectomy group; however this was not statistically significant. Duration of anaesthesia was longest in robotic cystectomy procedure. (Table 1)

TABLE I: Demographic data

Variables	Robotic prostatectomy	Robotic cystectomy	Robotic pyeloplasty	Robotic nephrectomy
Age (years)	66.7 ± 5.56	58.2 ±11.09	22.2 ± 7.328	63.5 ± 28.05
Weight (kg)	65.7 ± 11.42	63.5 ± 17.26	49.1 ± 8.80	70.5 ± 9.68
Body mass index	24.1 ± 4.37	24.4 ± 6.871	18.9 ± 3.57	30.8 ± 0.00
Total IV fluid (ml)	3067.7 ± 185.08	4680.0 ± 600.81	2100.0 ± 600.00	2375.0 ± 125.00
IV fluid per hour (ml/h)	345.10 ± 144.11	386.33 ± 170.21	366.72 ± 175.67	461.31 ± 133.73
Estimated blood loss (mls)	462.5 ± 45.23	838.46 ± 174.51	187.5 ± 12.5	237.5 ± 37.5
Duration of anaesthesia (min)	539.2 ± 13.82	748.4 ± 47.61	325.0 ± 27.30	330.0 ± 79.37

We performed a multivariable analysis for difficulty in ventilation parameters (highest Inspired pressure (IP), highest airway pressure (Pmax)) and found no correlation between age, weight, total fluid used and duration of anaesthesia to difficulty in ventilation. However, there was a mild correlation (r=0.44, p=0.041) between body mass index (BMI) and difficulty in ventilation in all types of surgery.

The baseline insufflation pressure used in these surgeries was between 12-15 cm H2O, and pressures were up to 20 cm H2O during the surgery; the highest increment was seen in the robotic nephrectomy. Ventilation requirements, as shown by the rise in inspiratory pressure (IP) and minute ventilation (MV) was highest in the robotic cystectomy group which demonstrated a 126% increase in IP and MV of 7.22, followed by prostatectomy group and other group. The subsequent rise in airway pressure (Pmax) was highest in cystectomy group (110% increment), followed by prostatectomy group (68% increment), compared to other groups. The percentage rise in ETCO2 from baseline and the degree of rise in pCO2 was seen highest in the cystectomy (56% and 41% respectively) compared to other groups.

TABLE II: Table showing ventilatory changes with types of robotic procedures

Variables	Robotic Prostatectomy	Robotic Cystectomy	Robotic Pyeloplasty	Robotic Nephrectomy	P value
Baseline Insufflation P (cm H2O)	13.54 ± 3.29	12.63 ± 3.12	15.80 ± 2.49	13.00 ± 4.00	0.29
Highest Insufflation P (cm H2O)	19.32 ± 2.24	19.56 ± 1.86	19.00 ± 2.23	20.75 ± 1.50	0.59
Baseline IP (cm H2O)	11.87 ± 3.19	11.19 ± 2.37	9.80 ± 1.64	12.00 ± 2.45	0.45
Highest IP (cm H2O)	24.65 ± 3.78	24.75 ± 4.63	18.20 ± 3.83	23.00 ± 7.70	0.016
IP difference (%)	116.74 ± 7.22	126.72 ± 11.66	85.58 ± 9.47	94.74 ± 38.14	0.33
MV Highest (i/min)	7.21 ± 2.22	7.22 ± 1.31	6.18 ± 1.88	5.70 ± 1.61	0.94
Baseline RR (/min)	11.09 ± 1.57	11.31 ± 2.02	10.20 ± 1.48	10.00 ± 0.00	0.35
Highest RR (/min)	17.98 ± 3.86	18.56 ± 2.83	16.60 ± 3.13	18.00 ± 4.00	0.77
Baseline Pmax (cm H2O)	18.67 ± 5.63	15.27 ± 2.94	17.40 ± 1.95	24.67 ± 7.23	0.021
Highest Pmax (cm H2O)	29.81 ± 4.78	31.13 ± 4.19	23.00 ± 3.24	31.25 ± 2.99	0.007
Pmax difference (%)	68.00 ± 5.92	109.99 ± 10.70	34.22 ± 12.97	29.24 ± 22.73	0.0001
Baseline EtCO2 (mm Hg)	34.5 ± 3.80	32.6 ± 2.80	33.8 ± 3.11	34.2 ± 3.61	0.22
Highest EtCO2 (mmHg)	50.08 ± 9.33	51.25 ± 10.31	47.20 ± 1.92	50.50 ± 13.20	0.87
% Rise in EtCO2 (%)	46.13 ± 4.39	55.95 ± 9.68	40.46 ± 5.53	39.63 ± 14.15	0.64

A multivariable analysis for bicarbonate level (HCO3), base deficit (BE), end tidal CO2 (EtCO2), pCO2 and acidosis (pH), have shown that the acidosis was contributed mainly by the respiratory component. It was demonstrated by the 30-40% rise in pCO2, compared to a small 4-5% drop in the bicarbonate level (HCO3) in all groups. The degree of acidosis, as shown by the lowest pH is worst in robotic nephrectomy (7.17), followed by robotic cystectomy (7.21), robotic prostatectomy (7.22) and the least affected in the robotic pyeloplasty group. Though the acidosis in robotic nephrectomy group was mainly contributed by the respiratory component, it was also made worse by metabolic component as demonstrated by the pre-existing baseline acidosis and also the high base deficit in that group.

We compared the ventilation data between the group which required maximal Trendelenburg position (robotic prostatectomy and robotic cystectomy) and the group with minimal Trendelenburg (robotic nephrectomy and robotic pyeloplasty) and found that the group with maximal Trendelenburg required higher inspiratory pressure (IP) resulting in higher Pmax (78% increase from baseline), compared to the group which required minimal Trendelenburg (32% increase from baseline).

TABLE III: Table showing acid base changes with types of robotic procedures

Variables	Robotic Prostatectomy	Robotic Cystectomy	Robotic Pyeloplasty	Robotic Nephrectomy	P value
Baseline pCO2 (mm Hg)	41.7 ± 9.42	40.0 ± 12.51	37.8 ± 3.86	46.2 ± 6.56	0.61
Highest PaCO2 (mmHg)	53.49 ±11.17	53.13 ± 17.71	48.00 ± 0.00	54.00 ± 9.89	0.98
EtCO2-PaCO2 difference	7.26 ± 9.03	8.28 ± 12.82	3.25 ± 5.19	10.22 ± 8.97	0.76
Baseline pH	7.32 ± 0.08	7.33 ± 0.10	7.31 ± 0.06	7.26 ± 0.09	0.63
Lowest pH	7.22 ± 0.08	7.21 ± 0.10	7.34 ± 0.05	7.17 ± 0.06	0.087
Baseline HCO3 (mmol/l)	21.10 ± 3.05	20.71 ± 2.37	20.60 ± 1.00	20.62 ± 3.25	0.95
Lowest HCO3 (mmol/l)	19.55 ± 2.41	19.87 ± 3.19	19.63 ± 3.67	19.53 ± 1.91	0.98
Baseline BE	-4.74 ± 3.46	-4.81 ± 2.76	-5.58 ± 1.55	-6.73 ± 3.47	0.67
Lowest BE	-7.37 ± 3.08	-8.13 ± 2.65	-5.33 ± 2.57	-8.23 ± 2.07	0.46

TABLE IV: Table showing airway and ventilatory pressure comparison between two groups in terms of patient's position during the surgery.

Variables	45-degree Trendelenburg group (Robotic Prostatectomy & Cystectomy)	25-degree Trendelenburg group (Robotic Pyeloplasty & Nephrectomy)	P value
Highest IP (cm H2O)	24.68 ± 3.98	20.33 ± 6.00	0.033
Baseline IP (cm H2O)	11.69 ± 2.99	10.78 ± 2.22	0.55
IP difference (%)	119.36 ± 47.78	89.65 ± 49.29	0.52
Highest MV	7.23 ± 2.00	5.97 ± 1.67	0.37
Highest PEEP (cm H2O)	7.06 ± 1.63	7.11 ± 1.69	0.99
Highest Pmax (cm H2O)	30.13 ± 4.64	26.67 ± 5.24	0.37
Pmax difference from baseline (%)	78.50 ± 43.87	32.35 ± 30.50	0.33

FIGURE 1: Figure showing mean Pmax difference from baseline Inspired Pressure (in Percentage) with various Robotic Procedures

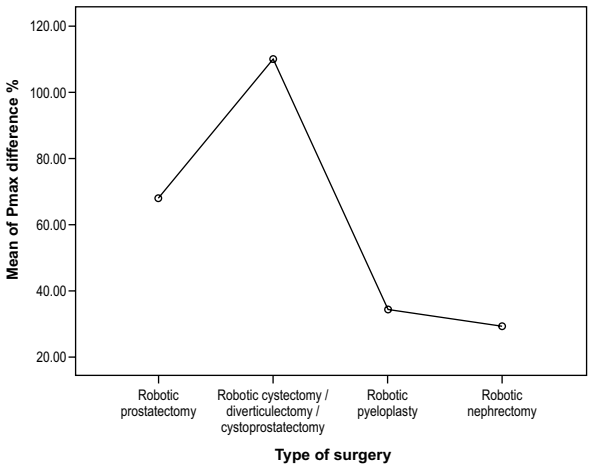


FIGURE 2: Figure showing mean difference of Inspired Pressure from baseline (in Percentage) with various robotic procedures.

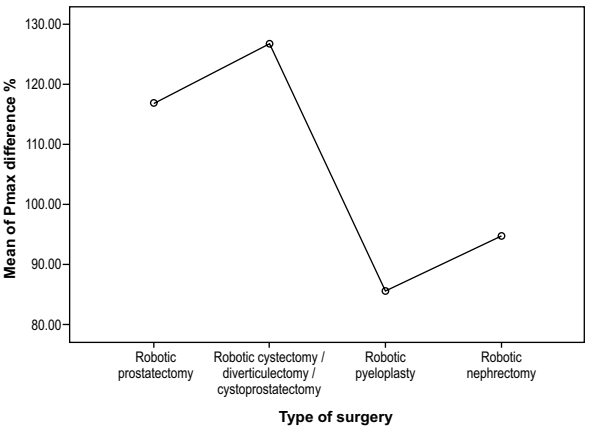
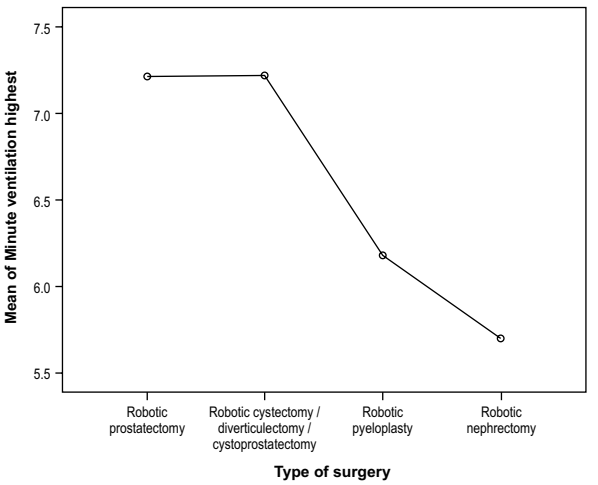


FIGURE 3: Figure showing highest Minute Ventilation with various Robotic Procedures



DISCUSSION

Ventilatory requirements were found to be highest in the robotic procedures of cystectomy and prostatectomy groups compared to robotic nephrectomy and pyeloplasty, which observed the lowest ventilatory requirements. These changes are likely observed due to the need to overcome the effect of gravity on respiratory physiological changes during Trendelenburg position which are made worse by pneumoperitoneum.[2] Maintaining adequate ventilation will help minimise acid base changes that are likely to occur resulting from:

- Worsening respiratory acidosis (low lung compliance, interstitial lung oedema)[2,3]
- Worsening metabolic acidosis (conservative intraoperative fluid management, preoperative preparation of patients for surgery, pneumoperitoneum effects on splanchnic and renal flow)[3]
- Worsening acidosis will increase cerebral blood flow and increase intracranial pressure.[4,5]

Therefore it is imperative that adequate ventilation is maintained especially in robotic procedures whereby Trendelenburg position is used.

Providing adequate ventilation at the expense of reducing barotrauma was a challenge we faced in our series. The longer the duration of surgery, observed dynamic changes in the cardio respiratory system required adjustments in ventilatory parameters. Targeting EtCO2 of 40 mmHg and below and Pmax of less than 40 cm H2O to achieve an acceptable tidal volume; which has been easily achieved in other studies,[6,7] has not been replicated in our series. Our target EtCO2 used was high normocapnia (40-45 mmHg) to allow for Pmax of less than 40 cmH2O.

Trendelenburg position reduces Functional Residual Capacity (FRC), predisposes to atelectasis, and reduced lung compliance from displaced abdominal contents.[3] Increased pulmonary venous pressure from increased pulmonary blood content causes most of the lung to be below the left atrium and hence its vasculature to lie in West zone 4 (Pa>Pi>PV>PA).[9] This may cause development of pulmonary interstitial oedema even though there are studies that indicate there was no significant change in the intrathoracic blood volume.[10,11] The added interstitial pressure will hasten alveolar closure before the next inspiration and hinder adequate gas exchange especially when the expiratory time is relatively long. Shortened expiratory time may prevent alveolar from closing earlier than usual as the next cycle of inspiration will help keep the airway patent for gas exchange to occur. We found that inspiratory to expiratory time ratio of 1:1.5 could be beneficial to improve ventilation especially when the inflating pressures are dangerously high.

We used pressure controlled ventilation in our patients in order to achieve a desirable tidal volume within a safe pressure range, compared to volume controlled ventilation in other studies.[6,7,8] However, we had a case in whom significant pneumothorax ensued post operatively which required drainage. A few of our patients developed subcutaneous emphysema which subsequently resolved within twenty four hours. Mean respiratory rates for our series during Trendelenburg position were 16-18 breaths per minute, from baseline of 10-12 breaths per minute, mean PMax were 29 to 31 cmH2O with at least twice the baseline minute ventilation for

procedures that require Trendelenburg position. We postulate that for procedures with ventilatory difficulties subject to longer duration of surgery, similar ventilatory strategies will be able to provide adequate minute ventilation at safer inspired and peak inflating pressures to counter the ongoing changes in ventilation perfusion characteristics.

There are limitations to this study. This study is a retrospective audit from robotic cases done from 2008 to 2010. We found that anaesthesia for the cases were being handled by multiple anaesthesiologists with different levels of experience in handling robotic procedures. Problems with missing data were also encountered.

CONCLUSION

Robotic urological procedures are generally challenging to the anaesthetist. Anaesthesia for robotic prostatectomy and cystectomy is more demanding compared to pyeloplasty and nephrectomy due to extreme Trendelenberg position and longer operating time. Ventilation difficulty, combined with restrictive fluid management results in mixed respiratory and metabolic acidosis. Anaesthetic management key points are good ventilatory support and judicious fluid management.

References

1. A Consensus Document on Robotic Surgery. Position Papers/Statements published on November 2007 by the Society of American Gastrointestinal and Endoscopic Surgeons (SAGES)
2. Senarai Prosedur Tahunan Dewan Bedah Urologi, HKL
3. MJ Sullivan, EAM Frost, MW Lew. Anesthetic care of the patient for robotic surgery. *M.E.J ANESTH* 2008;**19**(5): 967-982.
4. S Baltayian. A brief review: anaesthesia for robotic prostatectomy. *J Robotic Surg* 2008;**2**:59-66.
5. Grabowski JE, Talamini MA. Physiological Effects of Pneumoperitoneum. *J Gastrointest Surg* 2009;**13**:1009–1016
6. Kalmar AF, Foubert L, Hendrickx JFA , Mottrie A, Absalom A, Mortier EP, Struys MMRF. Influence of steep Trendelenburg position and CO2 pneumoperitoneum on cardiovascular, cerebrovascular, and respiratory homeostasis during robotic prostatectomy?? JOURNAL
7. Park EY, Koo BN, Min KT, Nam SH. The Effect of Pneumoperitoneum in the steep Trendelenburg position on cerebral oxygenation. *Acta Anaesthesiol Scand* 2009;**53**: 895-899
8. Lestar M, Gunnarsson L, Lagerstrand L, Wiklund P, Odeberg-Wernermann S. Hemodynamic Perturbations During Robot-Assisted Laparoscopic Radical Prostatectomy in 45° Trendelenburg Position. *Anesth Analg* 2011;**113**:1069-75
9. Hong JY, Lee SJ, Rha KH, Roh GU, Kwon SY, Kil HK. Effects of Thoracic Epidural Analgesia Combined with General Anesthesia on Intraoperative Ventilation/Oxygenation and Postoperative Pulmonary Complications in Robot-Assisted Laparoscopic Radical Prostatectomy. *Journal of Endourology* 2009;**23**:1843-1849
10. Respiratory Physiology 8th edition (Respiratory Physiology: The Essentials Lippincott Williams & Wilkins 2008)
11. Hofer CK, Zalunardo MP, Klaghofer R, et al. Changes in intrathoracic blood volume associated with pneumoperitoneum and positioning *Acta Anaesthesiol Scand* 2002;**46**:303-308
12. Meininger D, Westphal K, Bremerich DH, Runkel H, Probst M, Zwissler B, Byhahn C. Effects of Posture and Prolonged Pneumoperitoneum on Hemodynamic Parameters during Laparoscopy. *World J Surg* 2008;**32**:1400-1405

Ultrasound Guided Infraclavicular Block - What, Why and How?

Lim Teng Cheow
Anaesthesiologist, Department of Anaesthesia and Intensive Care, Hospital Melaka

INTRODUCTION

Brachial plexus block essentially involves an injection of a local anaesthetic solution to surround the brachial plexus and it may be performed via four main approaches, namely the interscalene, supraclavicular, infraclavicular and axillary. The Infraclavicular approach of the brachial plexus block provides effective surgical anaesthesia of the lower arm and hand.[1,2,3] With the refinement and advancement in the ultrasound technology, it has been utilized increasingly to perform this block in the recent years.[4,5,6]

HISTORY

The infraclavicular approach of brachial plexus block or the infraclavicular block (ICB) was introduced by Bazy in 1917 as an alternative to axillary and supraclavicular approaches. In this very early description of ICB, the needle was inserted below the clavicle at a point medial to the coracoid process and was advanced along a line linking the coracoid process and Chassaignac's tubercle (the line of anesthesia).[7] It was also described in 1922 by Gaston P Labat.[8] This technique failed to gain popularity in the early years of the 20th century. In the effort to overcome the limitations of the axillary approach of brachial plexus block, an alternative technique was described by Raj et al in 1975. In his technique, Chassaignac's tubercle, brachial and subclavian arteries were used as the landmark. The point of entry was at 1 inch inferior to the midpoint of clavicle and the needle is then directed laterally at 45° to skin, aiming to axillary artery.[9] Unfortunately, the widespread practice of Raj's technique seems not to have materialized.[10] Since then, modifications have been designed mainly to reduce the complications associated with this technique, particularly pneumothorax as well as to improve the quality of the block. The term 'coracoid block' was first coined by Whiffler in 1981 whereby the needle was inserted perpendicularly at a point medial and caudal to coracoid process on a line from subclavian artery to axillary artery.[11] Many years later, Kilka et al published the vertical infraclavicular approach of brachial plexus block in 1995, which described the introduction of needle perpendicularly just under the mid-clavicular point (Kilka's point) and this had really ignited the interest in ICB.[12] The coracoid approach of ICB was then popularized by Wilson et al in 1998. In this revised method, perpendicular needle introduction was done at a point located 2 cm medial and caudal to the tip of coracoid process.[13] As the vertical infraclavicular technique introduced by Kilka et al carried high risk of pneumothorax, while the landmarks used in the original Raj's technique were not always easily identified,[14] Borgeat et al has revised the technique in 2001, and this essentially was a modification of the Raj's technique. The injection was done at a point 1 cm inferior to the midpoint of clavicle, with needle aimed 45-60°to skin towards emergence of the axillary artery as close as possible to lateral border of pectoralis major. Apart from using the more easily identifiable landmarks, this modification was also made to facilitate the placement of the catheter which was thought to be difficult or impossible in the earlier techniques.[15] Ootaki et al have subsequently published one of the early papers of ultrasound guided ICB. They have proposed the injection of the local anaesthetic solution around the subclavian artery without the visualization of brachial plexus.[6] Two year later, Sandhu et al described an alternative approach of performing ultrasound-guided ICB. This new technique involved the administration of multiple injections of the local anaesthetic solution around each cord of the brachial plexus.[5] Klaastad et al in 2004 published another technique which was known as lateral sagittal technique and this is now a popular approach in the performance of ultrasound guided ICB. In this new technique, needle is introduced parasagitally adjacent to the most medial point of coracoid process and anterior

surface of clavicle.[16,17] Following this, papers were then published on ultrasound guided lateral sagittal approach of ICB.[18,19,20]

WHY ULTRASOUND?

The utilization of ultrasound has provided numerous benefits in the performance of peripheral nerve blocks, including ICB. It permits the visualization of all the three cords of the brachial plexus block.[5,21,22,23] In conjunction to this, repeated injections are made possible at the same site should the block begin to resolve. Ultrasound guidance is particularly useful in patients with amputated distal upper extremities which will make neurostimulation of distal parts impossible.[5] Besides the visualization of the target nerves, ultrasound also enables direct visualization of relevant anatomical structures. Blood vessels, particular axillary artery can be easily identified and subsequently this dictates the site of injection and minimizes complication of vessel injury.[18] As structures like axillary artery and vein can be seen directly, there have been reports describing the performance of ultrasound guided infraclavicular block in patients on anticoagulant medication.[24] The visualization of the trajectory of needle beneath the skin is useful in the control and aiming of needle movement. This may lead to several advantages, namely reduction of block performance time,[18] reduction of number of needle passes and thus minimizes patients' discomfort.[25,26] Inadvertent puncture of the axillary vein which was reported to occur in up to 30% of ICB performed with conventional methods can be minimized with the use of ultrasound.[27] The pattern of spread of local anaesthetic solution can be seen directly under ultrasound and this is again associated with several advantages. It serves as a predictor of a successful and effective block by observing the spread to the posterior aspect of axillary artery.[28] Ultimately, the success rate of the block can be increased.[18] As the local anaesthetic can be deposited in close proximity to the nerves under direct visualization, the onset of the block can be hastened while the duration of the block may be also prolonged particularly in paediatric patients.[4,18,29] In addition, it is also evident from papers that the dose of local anaesthetics administered may be reduced.[21,30]

ULTRASOUND ONLY OR ULTRASOUND WITH NEUROSTIMULATION?

With or without sonographic guidance, the performance of ICB with neurostimulation as the end point for a local anaesthetic injection produces success rates of 80-90%.[31,32,33,34] In comparison to this, performance of ICB by utilizing ultrasonic visualization of local anaesthetic spread as the end point was reported to yield a success rate of 90-95%.[5,6] The utilization of neurostimulation in the location of the brachial plexus is associated with several difficulties. The neurostimulation is not always successful in eliciting a motor response even when the stimulating needle is placed immediately adjacent to a nerve.[35] In certain cases, the interpretation of distal motor responses at cord level may not be simple and easy.[36] In addition, the neurostimulation may be technically difficult in the infraclavicular site due to the deep position of the cords.[37] The use of neurostimulation was further discouraged in a paper by Beach et al in 2006. It was pointed out that block success was not successfully predicted by the ability to produce a twitch response.[37]

RELATED FUNCTIONAL ANATOMY OF BRACHIAL PLEXUS AND INDICATIONS OF ICB

Infraclavicular block generally exert its action at the level of cord within the brachial plexus system. The blockade of axillary and medical brachial cutaneous (arm) nerves are unreliable in comparison to that of median, ulnar, radial, musculocutaneous and medial antebrachial cutaneous (forearm) nerves. Axillary nerve was reported to be blocked in 9-73% of the patients,[33,38] while the blockade of medial brachial cutaneous nerve of arm was achieved in 44-97% of the patients.[32,39,40,41] The poor blockade of medical brachial cutaneous nerve of arm may be related to the fact that it exits the medial cord quite early within the brachial plexus.[33] Unfortunately, there is no firm conclusion drawn as regards to association of blockade of medial brachial cutaneous nerve of arm and tourniquet pain. Jandard et al[33] has found a good correlation between tourniquet pain and failure of blockade of medial brachial cutaneous nerve of arm while other

authors were not appeared to be convinced by this. They noted that patients were able to tolerate the application of tourniquet without any additional block or analgesic despite the low success rate of blockade of medial cutaneous nerve of the arm.[42,43,44] The good tolerance to tourniquet application may be related to the possibility of the spread of local anaesthetic solution to block the intercostobrachial nerve which arises from second intercostal space. It crosses through the axillary fat layer and lies very close to the axillary vein before supplying the skin of the axilla and arm.[22] Unreliable blockade of axillary nerve negates its use in the provision of surgical anaesthesia in humeral surgeries.[45] Thus, ICB is well-suited in cases involving areas below the mid-humerus,[23] as well as for surgeries in the areas of forearm, wrist and hand.[15,32,46]

WHY THE INFRACLAVICULAR BLOCK?

In comparison to a supraclavicular block, the ICB was proven to be either equivalent or superior in terms of onset and efficacy of block.[34,45,47] The inferiority of supraclavicular block is mainly related to the unreliable quality of block in the areas supplied by ulnar and median nerves. It is believed that the poor quality of supraclavicular block may be due to the poor visualization of certain parts of the plexus in ultrasound-guided approach[45] or anatomical variations especially related to inferior trunk of the brachial plexus.[48] The later may probably explain the poor analgesia of the ulnar and median nerves which had contribution from the inferior trunk. Generally, the incidence of pneumothorax following supraclavicular block was quoted to be ranging between 0.5 to 6%[49]prior to the era of ultrasound, and although this complication may be likely reduced with aid of ultrasound,[50] it does not serve as a fail-safe technique to avoid pneumothorax during the supraclavicular block.[51] Horner syndrome is another common complication and it was reported to occur in 20-90% of patients following supraclavicular blocks performed using traditional modalities.[52] Tran et al[53] has reported that incidence of Horner syndrome was higher in patients undergoing supraclavicular block as compared to those with ICB. The similar result was also noted in a study by Koscielniak et al.[45] This is related to the blockade of cervical sympathetic chain as it normally lies very closely to the brachial plexus in the supraclavicular area.[53] The risk of diaphragmatic paralysis was found to be more readily associated with supraclavicular block then ICB.[45] This impairment in the diaphragmatic movement is due to the blockade of phrenic nerve and for this reason, it was also suggested that supraclavicular block should not be performed in patients with significant respiratory dysfunction or contralateral hemidiaphragmatic paralysis.[50]

Prior to the introduction of ultrasound, axillary block had lower success rate in comparison to ICB as it provided poorer quality of block of axillary and musculocutaneous nerves.[39,55,56] With the utilization of ultrasound, the success rate of axillary block has improved tremendously.[57,58,59] As musculocutaneous nerve leaves neurovascular compartment early at the level of coracoid process, axillary block does not provide reliable block of this nerve to provide surgical anaesthesia in forearm surgery.[56] Hence additional block of musculocutaneous nerve is necessary in the performance of axillary block,[60,61] whereas this additional block is not necessary in ICB. This nerve can be identified separately with ultrasound guidance. As it takes longer time to perform an axillary block, the procedural pain and number of needle passes are significantly increased respectively.[32,53,62] As it takes a longer time for the local anaesthetic to diffuse into the more proximal larger cords of the brachial plexus in ICB in comparison to more distal smaller terminal nerves in axillary block, the onset time of axillary block was reported to be faster in some studies.[39] In contrary, Tran et al did not find any significant difference in the onset time between axillary block and ICB.[53] In addition to these, the axillary block may not be suitable in patients with pain from upper limb trauma or fracture as it has to be done with the arm abducted, while ICB although not truly ideal, may be performed with the upper limb placed in neutral position.[38,39]

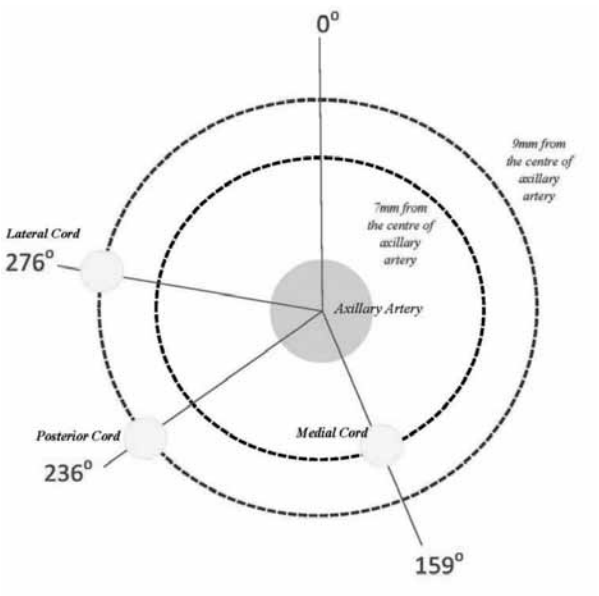
Ultrasound guided ICB is mainly limited by the poor visualization of the infraclavicular anatomy which occurs commonly in obese patients.[18,63] This difficulty is due to the relative deeper location of the brachial plexus in infraclavicular region

in comparison to axillary and supraclavicular approaches to the brachial plexus.[64] Technical improvements in ultrasound technology may probably address this limitation by production of better quality images in order to further improve the success rate.[18]

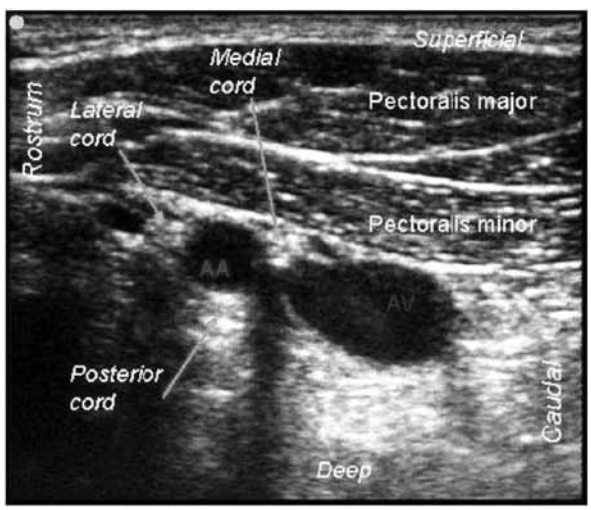
INFRACLAVICULAR SONOANATOMY AND SITE OF DEPOSITION OF LOCAL ANAESTHETIC

Although the magnetic resonance imaging (MRI) study showed that the cords of brachial plexus were found within 2 cm form the centre of axillary artery,[65] the identification of the nerve structures by ultrasound can be difficult.[18,63] As the axillary artery is easily identified, it serves as a basic landmark for ultrasound guided ICB.[66,67] In a study by Sauter et al,[68] it was noted that the anatomy of brachial plexus especially the orientation of cords around the axillary artery varies widely among individuals. All these cords were found to be distributed mainly between III and XI clock position.

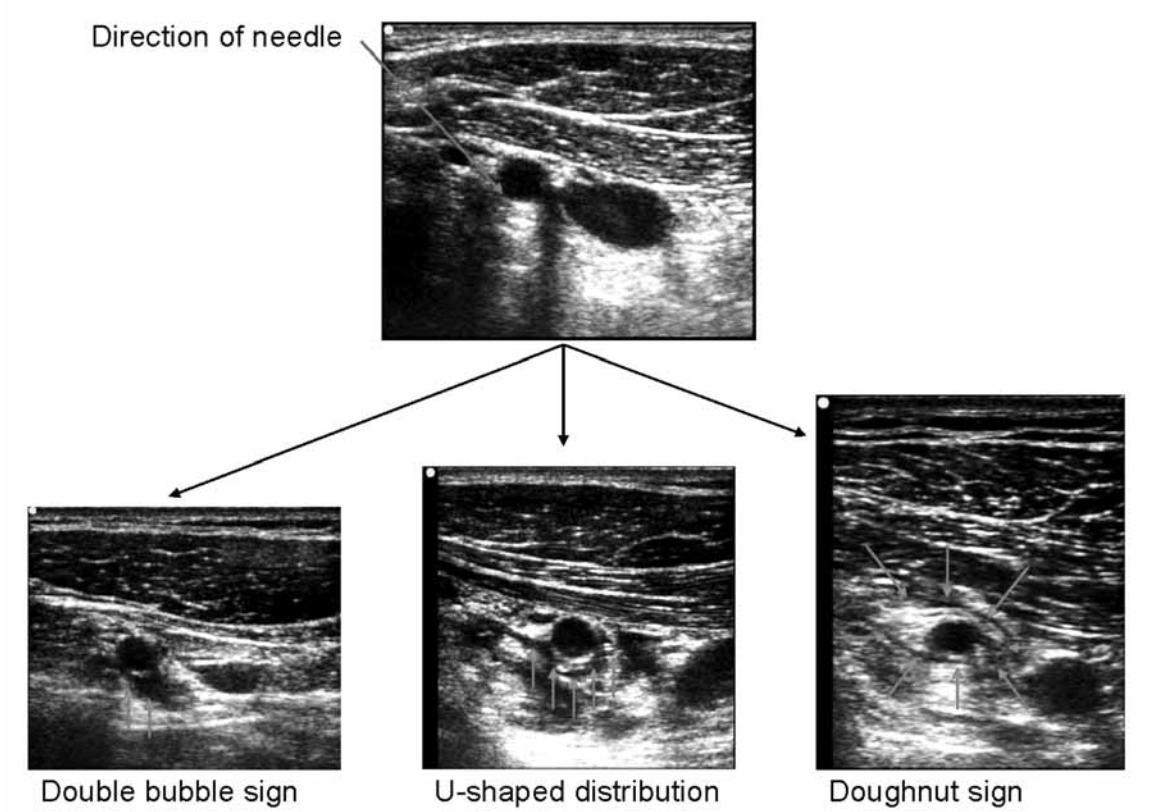
Owing to the significant interindividual difference in the distribution of the cords, an average point with shortest distances to these cords should be determined. This point was then determined to be at VIII clock position. Injection and deposition of local anaesthetic solution at this point generally leads to efficient spread of the solution to the cords. The local anaesthetic solution initially spread deep to axillary artery, followed by subsequent spread to the lateral and ultimately superior aspect of axillary vessels.[69] In practice, the needle should be directed in-plane with ultrasound probe towards the posterior aspect of axillary artery in close proximity to the posterior cord. Under the guidance of ultrasound, several patterns of spread of local anaesthetic were described to predict a good and reliable block, such as U-shaped distribution pattern, double bubble sign and doughnut sign.[6,18,70]

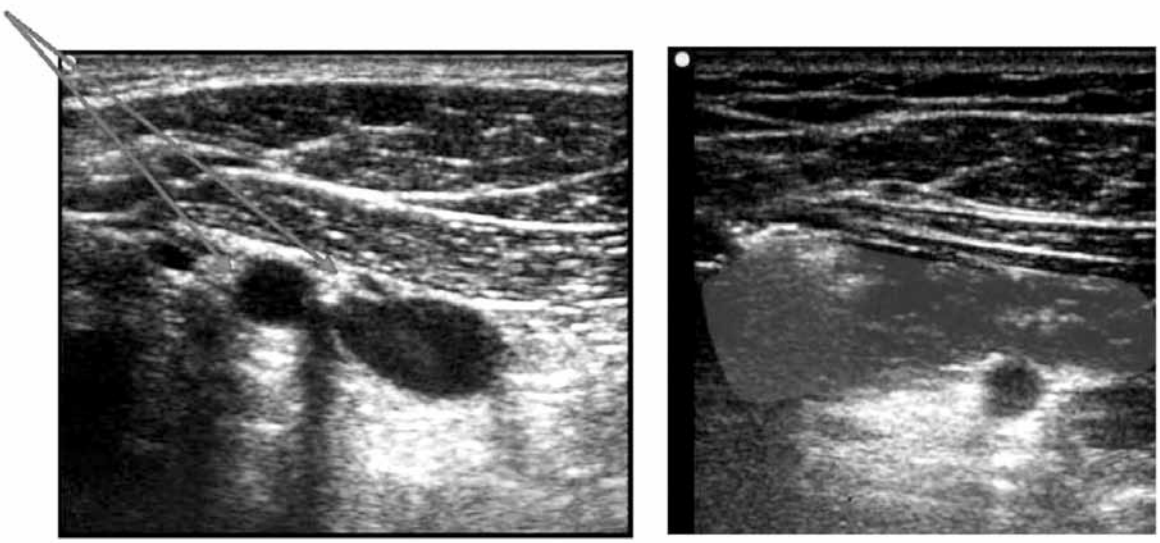


Position of the cords of brachial plexus. *Distance from centre of cord to the centre of axillary artery (presented as median with 5th and 95th percentile): Lateral cord 9 (4-18 mm) at 276° (263°-321°), Posterior cord 9 (4-12 mm) at 236° (189°-261°) and Medical cord 7 (4-11 mm) at 159º (90°-270°).* Adapted from Sauter AR, Smith HJ, Stubhaug A, et al. Anesth Analg 2006:103:1574-6.



Typical sonographic view of the infraclavicular region, with ultrasound probe placed in parasagittal plane medial to coracoid process. *All the cords are found to be distributed mainly between III and XI clock position in most of the subjects. AA= axillary artery, AV= axillary vein.*





Deposition of local anaesthetic solution around the lateral and medial cords causes its spread to be more concentrated at the superior and anterior aspect of axillary artery. which is represented by an area coloured with blue. Subsequently, the posterior cord is spared. The red solid arrows depict the direction of needle placement.

If local anaesthetic is deposited around the lateral or medial cords, the solution will concentrate at the superior and anterior aspect of axillary artery with the posterior cord spared and unblocked. This obviously predicts a failed ICB.[69]

SINGLE VS MULTIPLE INJECTIONS

The authors of one of the earliest ultrasound guided ICB study proposed a multiple injection technique to block each cord under the guidance of ultrasound.[5] However, it was noted 7 years later that the success rate and onset of surgical anaesthesia were not indeed enhanced by a triple injection technique as compared to a single injection technique which deposited the local anaesthetic at a point posterior to axillary artery.[71]

POSITIONING OF THE ARM

Significant difference in distance from skin to axillary artery was found following arm abduction. By abducting the arm by 90°, neurovascular bundle is brought to a more superficial plane to facilitate the performance of ICB.[72] In addition to this, arm abduction also pulls the brachial plexus away from the thoracic wall and eventually reduces the possibility of pleural puncture.[23] Although arm abduction may shorten the distance between the target nerve and skin, this obviously may not be always possible especially in patients with a very painful arm.

APPROACHES

To date, numerous approaches have been reported in the performance of ultrasound guided ICB. One of the commonest approaches is the lateral sagittal in-plane approach, in which the ultrasound probe is placed medial to coracoid process with the needle advanced in plane with ultrasound probe. In the out-of-plane approach, the ultrasound probe is placed below the clavicle and the needle is introduced via an out-of-plane technique from the upper border of the probe. It was described in paediatric subjects to allow more space between the ultrasound probe and clavicle to facilitate needle manipulation.[4,73] The posterior in-plane approach was described by Hebbard et al in 2007.[74] It was believed that this approach would improve the visualization of needle during advancement and hence allowing better alignment of needle with neuromuscular structure.

COMPLICATIONS

Complications have been reported following ICB, regardless of methods used to locate the brachial plexus. Although ultrasound technique has been used extensively in the performance of brachial plexus block, little data exists regarding the frequency of complications.[75] Even in expert hands, ultrasound guidance may only reduce but unable to eliminate totally the complications of an intraneural or intravascular injection.[22,76,77] The in-plane approach of ultrasound-guided nerve blocks may give the operator a false sense of safety with respect to needle-to-nerve proximity and it is not impossible to lose the sight of needle tip.[78] Following this, some authors were in the opinion that application of ultrasound may possibly increase the risks particularly neurological complications.[79] As regards to this, there have been a few cases of intraneural injection of local anaesthetic solution reported despite the use of real-time ultrasound guidance.[76,80,81] Although none of the cases described were related to ICB, the similar limitation of ultrasound-guided technique may still be very likely applicable to ultrasound-guided ICB. In a study done by Fredrickson et al[82] to analyze the relation of neurological complication and ultrasound guided peripheral nerve blocks, approximately 6% of the patients with ultrasound-guided ICB developed neurological symptoms within the first 6 months and from these, only 0.8% of the patients have the symptoms clearly related to the block. Luckily, none of the patients had prolonged complications with neurological symptoms persisted beyond 6 months following the block. Neurological complications are most of the time associated with procedure-related paraesthesia, which is the electric shock-like sensation during the procedure of brachial plexus block.[82] Intravascular injection of local anaesthetics

may still occur and this is particularly possible if spread of local anaesthetic is unable to be visualized under ultrasound imaging. Application of ultrasound may be beneficial in this scenario as failure to observe the spread of initial volume of local anaesthetic would stop the operator from injecting further and the sequalae from this intravenous injection of local anaesthetic could be minimized.[83] Pneumothorax is another complication which was reported following ultrasound guided ICB. Similar to the complications mentioned before, it may be related to poor visibility of needle during its advancement.[84] Hence, the consistent real time imaging of needle trajectory, identification of neighbouring structures and recognition of initial spread of local anaesthetic are vital in the prevention of complications related to performance of ultrasound-guided infraclavicular block.

CONCLUSION

Ultrasound-guided ICB is an effective mode of anaesthesia for surgeries of the lower arm and hand. Ultrasound guidance has provided benefits and this to a certain extent has increased the safety profile and success rate of the block. It can be even performed successfully without the need of neurostimulation. Although the injection is performed under direct ultrasound visualization, operators must be always aware of the pitfalls in this technique and necessary precautions should be practiced to prevent complications.

References

1. Rodriguez J, Taboada-Muniz M, Barcena M, et al. Median versus musculocutaneous nerve response with single-injection infraclavicular coracoid block. *RegAnesth Pain Med* 2004;534-8
2. Lecamwasam H, Mayfield J, Rosow L, et al. Stimulation of posterior cord predicts successful infraclavicular block. *AnesthAnalg* 2006;**102**:1564-8
3. Minville V, Fourcade O, Bourdet B, et al. The optimal motor response for infraclavicular brachial plexus block. *AnesthAnalg* 2007;**104**:448-51
4. Marhofer P, Sitzwohl C, Grehel M, et al. Ultrasound guidance for infraclavicular brachial plexus anaesthesia in children. *Anaesthesia* 2004;**59**:642-6
5. Sandhu NS, Capan LM. Ultrasound-guided infraclavicular brachial plexus block. *Br J Anaesth* 2002;**89**:254-9
6. Ootaki C, Hayashi H, Amano M. Ultrasound-guided infraclavicular brachial plexus block: an alternative technique to anatomical landmark-guided approaches. *RegAnesth Pain Med* 2000;**25**:600-4
7. Bazy L. The infraclavicular block. In: Pauchet V, Sourdat P, Larboure J editors. L'AnesthesieRegionale. Paris, France: *G DoinetCie* 1917;222-5
8. Labat G. Brachial plexus block: details of technique. *AnesthAnalg* 1927;**6**:81-2
9. Raj PP, Montgomery SJ, Nettles D, et al. Infraclavicular brachial plexus block: a new approach. *Anesth Analg* 1973;**52**:897-904
10. Rodriguez J, Barcena M, Rodriguez V, et al. Infraclavicular brachial plexus block effects on respiratory function and extent of the block. *RegAnesth Pain Med* 1998;**23**:564-8
11. Whiffler K. Coracoid block-a safe and easy technique. *Br J Anaesth* 1981;**53**:845-8
12. Kilka HG, Geiger P, Mehrkens HH. Die vertikaleinfraklaviulareBlokade des Plexus brachialis: EineneueMethodezurAnaesthesie der oberenExtremitat-eineanatomische und klinischeStudie. *Anaesthetist* 1995;**44**:339-44
13. Wilson JL, Brown DL, Wong GY, et al. Infraclavicular brachial plexus block: parasagittal anatomy important to the coracoid technique. *Anesth Analg* 1998;**87**:870-3
14. Tountas CP, Bergman RA. Arteries. In: Tountas CP, BergmanRA, eds. Anatomic variations of the upper extremity. *New York: Churchill Livingstone* 1993:187–92.
15. Borgeat A, Ekatadramis G, Dumont C.An evaluation of the infraclavicular block via a modified approach of the Raj technique. *AnesthAnalg* 2001;**93**:436-41
16. Klaastad O, Jorgen Smith H, Smedby O, et al. A novel infraclavicular brachial plexus block: the lateral and sagittal technique, developed by magnetic resonance imaging studies. *AnesthAnalg* 2004;**98**:252-6
17. Gurkan Y, Hosten T, Solak M, et al. Lateral sagittal infraclavicular block: clinical experience in 380 patients. *ActaAnaesthesiolScand* 2008;**52**:262-6

18. Dingemans E, Williams SR, Arcand G, et al. Neurostimulation in ultrasound-guided infraclavicular block: a prospective randomized trial. *AnesthAnalg* 2007;**104**:1275-80
19. Sauter AR, Dodgson MS, Stubhaug A, et al. Electrical nerve stimulation or ultrasound guidance for lateral sagittal infraclavicular blocks: a randomized controlled, observer-blinded comparative study. *AnesthAnalg* 2008;**106**:1910-5
20. Taboada M, Rodriques J, Amor M, et al. Is ultrasound guidance superior to conventional nerve stimulation for coracoid infraclavicular brachial plexus block? *RegAnesth Pain Med* 2009;**34**:357-60
21. Sandhu NS, Bahnival CS, Capan LM. Feasibility of infraclavicular block with a reduced volume of lidocaine with sonographic guidance. *J Ultrasound Med* 2006;**25**:51-6
22. Sandhu NS, Manne JS, Medabalmi PK, et al. Sonographically guided infraclavicular brachial plexus block in adults. A retrospective analysis of 1146 cases. *J Ultrasound Med* 2006;**25**:1555-61
23. Bigeleisen P, Wilson M. A comparison of two techniques for ultrasound guided infraclavicular brachial plexus block. *Br J Anaesth* 2006;**96**:502-7
24. Bigeleisen P. Ultrasound-guided infraclavicular block in an anticoagulated and anesthetizedpatient. *AnesthAnalg* 2007;**104**:1285-7
25. Prebaugh SL, Williams BA, Kentor ML. Ultrasound guidance with nerve stimulation reduces time necessary for resident peripheral nerve blockade. *RegAnesth Pain Med* 2007;**32**:448-54
26. Domingo-Triado V, Selfa S, Martinez F, et al. Ultrasound guidance for lateral midfermoral sciatic nerve block: a prospective, comparative, randomized study. *AnesthAnalg* 2007;**104**:1270-4
27. Grau T. Ultrasonography in current practice of regional anaesthesia. *Best Prac Res ClinAnaesthesiol* 2005;**19**:175-200
28. Porter JM, McCartney CJL, Chan VWS. Needle placement and injection posterior to the axillary artery may predict successful infraclavicular brachial plexus block: a report of three cases. *Can J Anaesth* 2005;**52**:69-73
29. Soeding PF, Sha S, Royse CF, et al. A randomized trial of ultrasound-guided brachial plexus anaesthesia in upper limb surgery. *AnesthIntens Care* 2005;**33**:719-25
30. Sandhu NS, Maharlouei B, Patel B, et al. Simultaneous bilateral infraclavicular brachial plexus block with low-dose lidocaine using ultrasound guidance. *Anesthesiology* 2006;**104**:199-201
31. Desroches J. The infraclavicular brachial plexus block by the coracoid approach is clinically effective: an observational study of 150 patients. *Can J Anaesth* 2003;**50**:253-7
32. Deleuze A, Gentili ME, Marret E, et al. A comparison of a single-stimulation lateral infraclavicular plexus block with a triple stimulation axillary block. *RegAnesth Pain Med* 2003;**28**:89-94
33. Jandard C, Gentili ME, Girard F, et al. Infraclavicular block with lateral approach and nerve stimulation: extent of anesthesia and adverse effects. *RegAnesth Pain Med* 2002;**27**:37-42
34. Arcand G, William S, Chouinard P, et al. Ultrasound-guided infraclavicular versus supraclavicular block. *AnesthAnalg* 2005;**101**:886-90
35. Urmey WF, Stanton J. Inability to consistently elicit a motor response following sensory paresthesia during interscalene block administration. *Anesthesiology* 2002;**96**:552-4
36. Borene SC, Edwards JN, Boezaart AP. At the cords, the pinkie towards: Interpreting infraclavicular motor response to neurostimulation. *RegAnesth Pain Med* 2004;**29**:125-9
37. Beach ML, Sited BD, Gallagher JD. Use of nerve stimulator does not improve the efficacy of ultrasound-guided supraclavicular nerve blocks. *J Clin Anaesth* 2006;**18**:580-4
38. Koscielniak-Nielsen ZJ, Rotboll-Nielsen P, Risby Mortensen C. A comparison of coracoids and axillary approaches to the brachial plexus. *Acta Anaesthesiol Scand* 2000;**44**:274-7
39. Kapral S, Jandrsits O, Schabernig C, et al. Lateral infraclavicular plexus block vs axillary block for hand and forearm surgery. *Acta Anaesthesiol Scand* 1999;**43**:1047-52
40. Gaertner E, Estebe JP, Zamfir A, et al. Infraclavicular plexus block: multiple injection versus single injection. *Reg Anesth Pain Med* 2002;**27**:590-4
41. Gurkan Y, Acar S, Solak M, et al. Comparison of nerve stimulation vs ultrasound-guided lateral sagittal infraclavicular block. *Acta Anaesthesiol Scand* 2008;**52**:851-855
42. Desroches J. The infraclavicular brachial plexus block by the coracoid approach is clinically effective: an observational study of 150 patients. *Can J Anaesth* 2003;**50**:253-7

43. Minville V, Amathieu R, Luc NG, et al. Infraclavicular brachial plexus block versus humeral approach: comparison of anesthetic time and efficacy. *Anesth Analg* 2005;**101**:1198 –201
44. Minville V, Fourcade O, Idabouk L, et al. Infraclavicular brachial plexus block versus humeral block in trauma patients: a comparison of patient comfort. *Anesth Analg* 2006;**102**:912–6
45. Koscielniak-Nielsen ZJ, Frederiksen BS, Rasmussen H, et al. A comparison of ultrasound-guided supraclavicular and infraclavicular blocks for upper extremity surgery. *Acta Anaesthesiol Scand* 2009;**53**:620-6
46. Koscielniak-Nielsen ZJ, Rasmussen H, Hesselbjerg L, et al. Clinical evaluation of the lateral sagittal infraclavicular block developed by MRI studies. *Reg Anesth Pain Med* 2005;**30**:329-34
47. Fredrickson MJ, Patel A, Young S, et al. Speed of onset of corner pocket supraclavicular and infraclavicular ultrasound guided brachial plexus block: a randomised observer-blinded comparison. *Anaesthesia* 2009;**64**:738-44
48. Royse CF, Sha S, Soeding PF, et al. Anatomical study of the brachial plexus using surface ultrasound. *Anaesth Intens Care* 2006;**34**:203-10
49. Brown DL, Bridenbaugh LD. The upper extremity: Somatic block, In: Cousins MJ, Bridenbaugh PO, eds. Neural Blockade in Clinical Anesthesia and Management of Pain, 3rd ed. Philadelphia, *PA: Lippincott-Raven* 1998:345-71
50. Perlas A, Lobo G, Lo N, et al. Ultrasound-guided supraclavicular block: outcome of 510 consecutive cases. *Reg Anesth Pain Med* 2009;**34**:171-6
51. Bhatia A, Lai J, Chan CWS, et al. Pneumothorax as a complication of the ultrasound-guided supraclavicular approach for brachial plexus block. *Anesth Analg* 2010;**111**:817–9
52. Neal JM, Hebl JR, Gerancher JC, et al. Brachial plexus anesthesia: essentials of our current understanding. *Reg Anesth Pain Med* 2002;**27**:402-28
53. Tran DQT, Russo G, Munoz L, et al. A prospective, randomised comparison between ultrasound-guided supraclavicular, infraclavicular and axillary brachial plexus blocks. *Reg Anesth Pain Med* 2009;**34**:366-71
54. Yang CW, Kwon HU, Cho CK, et al. A comparison of infraclavicular and supraclavicular approaches to the brachial plexus using neurostimulation. *Korean J Anesthesiol* 2010;**58**:260-6
55. Fleischmann E, Marhofer P, Greher M, et al. Brachial plexus anesthesia in children: lateral infraclavicular vs axillary approach. *Paediatr Anaesth* 2003;**13**:103-8
56. Rettig HC, Gielen JM, Boerma E, et al. A comparison of the vertical infraclavicular and axillary approaches for brachial plexus anaesthesia. *Acta Anaesthesiol Scand* 2005;**49**:1501-8
57. Casati A, Danelli G, Baciarello M,et al. A prospective, randomized comparison between ultrasound and nerve stimulation guidance for multiple injection axillary brachial plexus block. *Anesthesiology* 2007;**106**:992-6
58. Sites BD, Beach ML, Spence BC, et al. Ultrasound guidance improves the success rate of a perivascular axillary plexus block. *Acta Anaesthesiol Scand* 2006;**50**:678-84
59. Chan VWS, Perlas A, McCartney CJL, et al. Ultrasound guidance improves the success rate of axillary brachial plexus block. *Can J Anaesth* 2007;**54**:176-82
60. Sia S, Bartoli M. Selective ulnar nerve localization is not essential for axillary brachial block using a multiple nerve stimulation technique. *Reg Anesth Pain Med* 2001;**26**:12-6
61. Coventry DM, Barker KF, Thomson M. Comparison of two neurostimulation technique for axillary brachialis plexus blockade. *Br J Anaesth* 2001;**86**:80-3
62. Koscielniak-Nielsen ZJ, Rasmussen H, Hesselbjerg L, et al. Infraclavicular block causes less discomfort than axillary block in ambulatory patients. *Acta Anaesthesiol Scand* 2005;**49**:1030-4
63. Perlas A, Chan VWS, Simon M. Brachial plexus examination and localization using ultrasound and electrical stimulation: a volunteer study. *Anesthesiology* 2003,**99**:429-35
64. Mirza F, Brown AR. Ultrasound-guided regional anesthesia for procedures of the upper extremity. Anesthesiol Res Pract 2011, Article ID 579824, doi:10.1155/2011/579824
65. Klasstad O, Smith HJ, Smedby O, et al. A novel infraclavicular brachial plexus: the lateral and sagittal technique, developed by magnetic resonance imaging studies. *Anesth Analg* 2004;**98**:252-6
66. Retzi G, Kapral S, Greher M, et al. Ultrasonographic findings of the axillary part of the brachial plexus. *Anesth Analg* 2001;**92**:171-5
67. Yang WT, Chui PT, Metrewell C. Anatomy of the normal brachial plexus revealed by sonography and the role of sonographic guidance in anesthesia of brachial plexus. *Am J Roentgenol* 1998;**171**:1631-8

68. Sauter AR, Smith HJ, Stubhaug A, et al. Use of magnetic resonance imaging to define the anatomical location closest to all three cords of infraclavicular brachial plexus. *Anesth Analg* 2006;**103**:1574-6

69. Bloc S, Garnier T, Komly B, et al. Spread of injectate associated with radial or median nerve type motor response during infraclavicular brachial plexus block: an ultrasound evaluation. *Reg Anesth Pain Med* 2007;**32**:130-5

70. Tran de QH, Charghi R, Finlayson RJ. The double bubbles sign for successful infraclavicular brachial plexus blockade. *Anesth Analg* 2006;**103**:1048-9

71. Desgagnes MC, Levesque S, Dion N, et al. A comparison of a single or triple injection technique for ultrasound-guided infraclavicular block: a prospective randomized controlled study. *Anesth Analg* 2009;**109**:668-72

72. Ruiz A, Sala X, Bargallo X, et al. The influence of arm abduction or anatomic relations of infraclavicular brachial plexus: an ultrasound study. *Anesth Analg* 2009;**108**:364-7

73. De Jose Maria B, Banus E, Egea MN, et al. Ultrasound guided supraclavicular vs infraclavicular brachial plexus blocks in children. *Pediatr Anesth* 2008;**18**:838-44

74. Hebbard P, Royse C. Ultrasound-guided posterior approach to the infraclavicular brachial plexus. *Anaesthesia* 2007;**62**:539

75. Hadzic A, Sala-Blanch X, Xu D. Ultasound guidance may reduce but not eliminate complications of peripheral nerve blocks. *Anesthesiology* 2008;**108**:557-8

76. Russon K, Blanco R. Accidental intraneural injection into the musculocutaneous nerve visualized with ultrasound. *Anaesth Analg* 2007;**105**:1504-5

77. Sites BD, Spence BC, Gallagher J, et al. Regional anesthesia meets ultrasound: a speciality in transition. *Acta Anaesthesiol Scand* 2008:456-66

78. Sites BD, Spence BC, Gallagher JD, et al. Characterizing novice behavior associated with learning ultrasound-guided peripheral regional anesthesia. *Reg Anesth Pain Med* 2007;**32**:107-15

79. Hebl JR, Ultrasound-guided regional anesthesia and the prevention of neurologic injury: fact or fiction? *Anesthesiology* 2008;**108**:186-8

80. Bigeleisen PE. Nerve puncture and apparent intraneural injection during ultrasound-guided axillary block does not invariably result in neurologic injury. *Anesthesiology* 2006;**105**:779-83

81. Schafhalter-Zoppoth I, Zeitz ID, Gray AT. Inadvertent femoral nerve impalement and intraneural injection visualized by ultrasound. *Anesth Analg* 2004;**99**:627-8

82. Fredrickson MJ, Kilfoyle DH. Neurological complication analysis of 1000 ultrasound guided peripheral nerve blocks for elective orthopaedic surgery: a prospective study. *Anaesthesia* 2009;**64**:836-44

83. Martinez Navas A, Tabla Gonzalez RO. Ultrasound-guided technique allowed early detection of intravascular injection during an infraclavicular brachial plexus block. *Acta Anaesthesiol Scand* 2009;**53**:968-70

84. Koscielniak-Nioelsen ZJ, Rasmussen H, Hesselbjerg L. Pneumothorax afer an ultrasound guided lateral sagittal infraclavicular block. *Acta Anaesthesiol Scand* 2008;**58**:1175-7

Continuous Lumbar Plexus Block with a Local Anaesthetic in the Management of Severe Cancer Pain – A Case Report

Ng Kim Swan
Consultant Anaesthesiologist & Pain Specialist, Hospital Selayang

INTRODUCTION

In recent years Continuous Lumbar Plexus Catheter Block (CLPB) using local anaesthetics has gained popularity in the management of acute pain especially with advances in catheter design and block techniques, e.g. ultrasound guided. Reports in the literature have so far been confined to its use in acute post-operative pain.[1] Single shot lumbar plexus block for postoperative analgesia in patients with hip fractures operated under spinal anaesthesia significantly reduced the requirement of supplemental analgesics in the first 24 hrs.[2] Another study using continuous lumbar plexus block in combination with post operative PCA morphine showed reduced opioid requirements and pain intensity, with consequent reduction in opioid-related side effects, and enhanced patient satisfaction.[3] However, the use of CLPB with local anaesthetics for the management of cancer pain is uncommon.

We report a case of successful continuous lumbar plexus block in a 36 year old man with severe pain from advanced adenocarcinoma of the caecum with infiltration of the abdominal wall and lateral extension into the right iliacus muscle.

CASE HISTORY

A 36 year old man diagnosed with recurrent adenocarcinoma of caecum with infiltration to the anterior abdominal wall and right iliacus muscle complained of severe pain at the anterior abdominal wall and the right groin with resulting immobility of the right lower limb especially at the hip. His left lower limb was completely unaffected by pain, with full range of motion of all joints. Although he was bed-bound because of the pain and immobility of the right lower limb (RLL), he was able to move himself in bed independently, using both upper limb (UL) and the left lower limb (LLL).

Surgical intervention, chemotherapy and radiotherapy had failed to control the cancer, and he also complained of colicky abdominal pain from the recurrent primary tumour.

When first seen by the pain management team, he was still complaining of severe pain (pain score 10/10) despite continuous subcutaneous (SC) infusion of morphine 400 mg and ketamine 200mg over 24 hours, transdermal fentanyl 200 mcg/hour with breakthrough doses of morphine 25mg SC and buscopan 20mg IV. Initially, an epidural catheter was inserted at L1/2 and a mixture of bupivacaine and fentanyl was infused at 10 ml/hour. This provided good pain relief for his leg and abdominal pain – his pain score reduced to 2/10 and the dose of SC morphine and fentanyl were reduced. However, he was very unhappy with the side effects of the epidural which included nausea & vomiting as well as numbness and weakness of both lower limbs due to sensory and motor block. When the epidural catheter slipped out 3 weeks later, we offered him a continuous intrathecal block instead; however he refused because of fear of bilateral sensory & motor block. He demanded: "I want to have no pain in my right leg but at the same time I want my left leg to be able to move so that I can move on my bed without assistance". In view of that, he was offered a right lumbar plexus (Psoas compartment) block, which we hoped would give him analgesia with motor and sensory block confined to the right lower limb.

METHOD OF LUMBAR PLEXUS CATHETER INSERTION

The patient was monitored and sedated during the procedure, which was done under aseptic technique. A conventional approach of lumbar plexus block was performed using the Contiplex ® Touhy set (18GX159mm). The skin was punctured at a point 1cm above the intercristine line and 4cm from the midline and a nerve stimulator was used to locate the lumbar plexus (presence of "patella dance"). A total of 20ml of 0.75% ropivacaine was injected slowly with frequent aspiration to avoid inadvertent intravascular injection. Subsequently a lumbar plexus catheter was inserted through the Contiplex ® Touhy needle into the psoas compartment. Continuous stimulation of the plexus was elicited by stimuplex and persistent quadriceps twitch & patella dance during the catheter insertion confirmed that the catheter was in place. The catheter was tunnelled for 7cm through the subcutaneous layer, then secured using a size 3.0 silk suture. A continuous infusion of 0.375% ropivacaine at 10ml/hour was then started. The local anesthetic was later changed to 0.2% ropivacaine at same infusion rate.

RESULTS

After insertion of the lumbar plexus catheter, there was marked pain relief – his pain score reduced to 1/10. Sensory block in the distribution of the right femoral, lateral femoral cutaneous and obturator nerves was elicited, confirming correct catheter placement. Although the patient was unable to flex his right hip, he was able to move the right knee with no pain at all; at the same time, movement in his left lower limb was unaffected. This pain relief was maintained with a continuous infusion of ropivacaine 0.2% at 10ml/hour in the ward. The patient's opioid requirements reduced further (being required mainly for the abdominal pain) and both the patient and the palliative care team were very satisfied. The analgesia was maintained until the patient passed away in the hospital 4 weeks later.

DISCUSSION

More than 75% of all patients with advanced cancer experience moderate to severe pain.[4] The pain may be associated with direct infiltration of tissues, organs and/or nerves by the tumour, pathologic fracture, surgery, or may be due to chemotherapy or radiation therapy. Unrelieved cancer pain dramatically affects quality of life, leads to decreased physical activity and social interaction, and increased morbidity and suffering.[5,6,7]

Although WHO says that more than 90% of cancer pain can be relieved using simple means, the remaining 5-10% require more sophisticated methods of pain relief, including invasive procedures. The more commonly used invasive blocks are neuroablation or central neuraxial blocks.[8] In recent years, with advances in technology, we are able to perform nerve and plexus blocks more reliably and are able to provide continuous blockade using catheter techniques. A search of the literature over the last 15 years showed anaesthetic interventions on cancer pain are mostly central neuraxial block using neurolytic agents or local anaesthetics; only one case on the unilateral right inguinal paravascular (lumbar plexus) neurolytic block was reported by Abdullah et al[9] for pain relief in a patient with pathological fracture of the acetabulum secondary to carcinoma of the lung; in this particular patient, 6% phenol was used after radiographic confirmation of the catheter position was obtained.

In our patient, only local anaesthetic infusion was used without neurolysis to achieve good analgesia. Although the catheter placement was not confirmed radiologically, the clinical effect reassured us that the catheter was within the psoas compartment. More importantly, the analgesic effect reported by the patient was very satisfactory.

In other similar cases, a central neuraxial block with an implanted intrathecal catheter would be the preferred option for pain relief. Furthermore, this patient's pain was not confined to his right hip but also involved the abdominal wall and he had visceral

pain from sub-acute intestinal obstruction due to the primary tumor, making a central neuraxial block even more suitable.

In this patient however, although we achieved pain relief with an epidural infusion of local anaesthetic, it was unacceptable to him due to the effect on the left lower limb - his remaining "good" leg. After discussion, we discovered that the patient was more concerned about the pain that was causing immobility of his right lower limb, rather than pain in the abdomen.

Anatomically, the psoas muscle receives its nerve supply from the anterior branches of the lumbar plexus. The iliacus is a flat triangular muscle which fills the iliac fossa and is innervated by the anterior branches of femoral nerve which is part of the lumbar plexus. Its fibers converge into the tendon of the psoas muscle which contributes to hip flexion. A lumbar plexus catheter infusion would block the nerve roots of L2,3,4 which supply the hip joint and upper part of the femur. With this block, we were able to achieve unilateral pain relief with a preservation of motor control over the unaffected limb. This resulted in a high level of patient satisfaction, which is one of the important factors that we should consider when we make a management plan for pain control in patients with advanced cancer.

CONCLUSION

This case report shows that lumbar plexus blocks are not confined to use in acute pain and demonstrates the feasibility of placing a lumbar plexus catheter via a conventional approach followed by continuous infusion of local anesthetic for relief of intractable cancer pain in a patient who demanded a unilateral sensory block.

FIGURE 1: LTP, two weeks before he passed away.

"I want to have no pain in my right leg but at the same time I want my left leg to be able to move so that I can move on my bed without assistance"

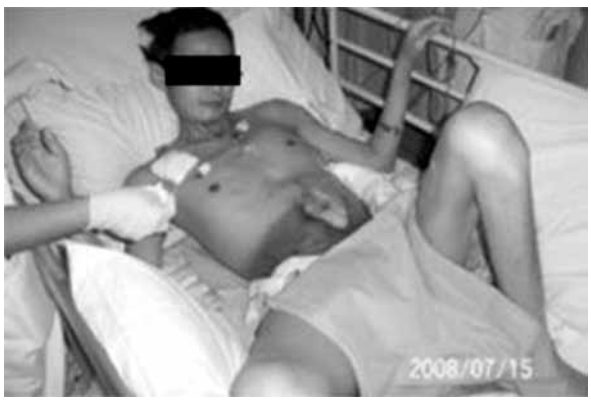


FIGURE 2: Pelvic CT Scan showing tumour arising from the caecum infiltrating the abdominal wall anteriorly and the right iliacus muscle laterally

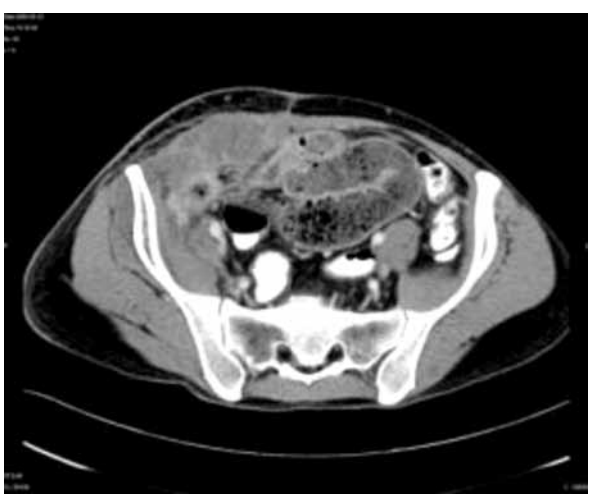
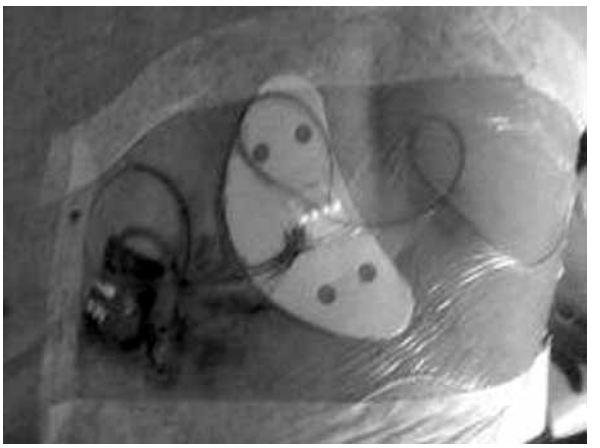


FIGURE 3: Anchoring of the lumbar plexus catheter



References

1. Patrick DB, Radu L, Gilles B, Pablo L and Elisabeth GM Continuous lumbar plexus block: Use of radiography to determine catheter tip location *Reg Anesth Pain Med* 2003;**28**(2):135-139.
2. Uma S, Aditya K, Surekha S, Anjum N, Vineeta G, Roli M,.Lumbar plexus block for post-operative analgesia following hip surgery : A comparison of "3 in 1" and psoas compartment block. *Indian J. Anaesth* 2007;**51**(2):127-130
3. Zafar IS, M. Soledad C, William D, Roman S and Daniel BC. Continuous Lumbar Plexus Block Provides Improved Analgesia With Fewer Side Effects Compared With Systemic Opioids After Hip Arthroplasty: A Randomized Controlled Trial *Reg Anesth Pain Med* 2007;**32**(5):135-139.
4. Oster MW, Vizel M, Turgeon LR. Pain of terminal cancer patients. *Arch Intern Med* 1979;**138**:1801-1802
5. Ashburn MA, Lipman AG. Management of pain in the cancer patient. *Anesth Analg* 1993;**76**:402-416
6. Cherny NI. The management of cancer pain. *CA Cancer J Clin*. 2000;**50**:70-116.
7. Portenoy RK, Lesage P. Management of cancer pain. *Lancet*. 1999;**353**:1695-1700.
8. Allen W. Burton. Interventional Management of Cancer Pain *CME* Jan 29th 2005, www.medscape.com/viewarticle/467488 accessed xxxx (KS, You need to put in the date you looked at the website).
9. Abdullah M. Kaki, Geraint W. Lewis. Inguinal paravascular (Lumbar plexus) neurolytic block – description of a catheter technique: case report. *Regional Anesthesia and Pain Medicine* 1998;**23**(2):214-21

"Wish I had more practice!"

Thiruselvi Subramaniam
Senior Lecturer, International Medical University

INTRODUCTION

Have we not, at some point in our career, wished that we had a bit more practice doing a procedure or handling a crisis situation? There is nothing ideal in the way we practice medicine sometimes and it is not the lack of knowledge but the lack of practice and perhaps even lack of exposure as doctors at different levels of care or area of practice are exposed to different levels or type of crisis. Consider this usual scenario;

An anaesthetic medical officer is called to intubate a trauma patient in A&E with respiratory insufficiency. Imagine this: the doctor arrives there and finds that there is chaos in casualty which we know is a regular norm when there is an unstable patient with multiple injuries needing input from more than one department. Everyone is calling out for something or the other as the patient has a bleeding open wound with a fractured long bone and a distended abdomen suggesting an intra-abdominal injury. There is a lack of communication and coordination in the acute area, with more than one person doing the same activity, lacking economy. The young doctor notices that the patient is an obese individual with a short neck who has facial injury. The patient is tachypoenic, restless and hypotensive with an oxygen saturation of 85%, a 20 gauge branula in the cubital area with a colloid as the intravenous solution. The young doctor realises that he may need to perform a fiberoptic intubation, something that he has seen his specialist do but once during his eight months of training in anaesthesia. He calls in his specialist who he knows has not done a fiberoptic intubation in a while as well.

What possibly goes through his mind is 'What if my specialist doesn't get here on time! I wish I had attended the airway workshop earlier this month; I would have had some exposure at least!"

In instances like this, even before we even realise it, patient safety that is usually uppermost in all our minds takes the backstage. Consider the alternative scenario if each and every individual in that team had been exposed to a scenario like this in a safe controlled environment several times before.

HISTORY OF SIMULATION

Simulation has been around for centuries; models were used to learn about surface anatomy and anatomical structures in the early period. It is baffling how medical simulation appears to have lagged behind other fields like the military, aviation industry, space programme and nuclear power industry because looking from the patient safety perspective, it should be on par or more advanced. The positive note to this is that we have caught on and appear to be fast catching up.[1,2]

Asmund Laerdal from Norway (a toy manufacturer), was one of the pioneers of CPR when he, in collaboration with anaesthetists developed "Resusci Anne", a part-task trainer to teach mouth-to-mouth resuscitation in the 1960s. Since then, there has been an ongoing evolution in simulators with production of more interactive and sophisticated models used for teaching and training resuscitation as well as basic and complex skills in medicine. The first realistic simulator was produced in the late 1960s (SimOne) by Abrahamson and Denson which had output for peripheral pulse and heart sounds but no output for electronic monitors and was eventually phased out.

In the 1980s, simulation was revived again, one group headed by David Gaba, developed a comprehensive anaesthesia simulaton environment (CASE) that later became known as Medsim while another group led by Michael. G and JS Gravenstein developed the Gainesville anaesthesia simulator (GAS) that has now come to be known as METI (Medical Education technologies, Inc.).[1,2]

WHAT IS SIMULATION?

It is the art of creating realism; as it means to imitate / replicate a task, a situation, and an environment

to portray real life circumstances in which medical services are provided. Simulation sets a safe and comfortable environment for learning, allowing health care workers from all level of expertise and discipline to learn at their own pace.

From the perspective of fidelity (quality of accuracy), simulations can be classified into two different groups: high fidelity and low fidelity. High fidelity simulations utilize very realistic materials and equipment to represent the task(s) that the participant must perform. Low fidelity simulations are less realistic, such as role play or part–task trainer to practice chest compression. There is no direct relationship between fidelity and technology, in fact it is a flexible one and they are used interchangeably to achieve a good learning outcome. (Figure I)

The major components in a simulation are the simulators (equipment that range from low to high fidelity using simple to complex technology), standardized patients and an appropriate environment (Figure II).

Simulators can be selectively used for an individual or group of trainees and be conveniently classified into 3 categories: part task trainers (Figure III), computer-enhanced mannequins (Figure I & III) and virtual reality simulators.[3,4] Hybrid simulation use combination of simulators and standardized patients and thus significantly increase the level of fidelity and as an added advantage aid in intimate or embarrassing physical examinations.[5,6]

FIGURE 1: Fidelity and technology.[5]

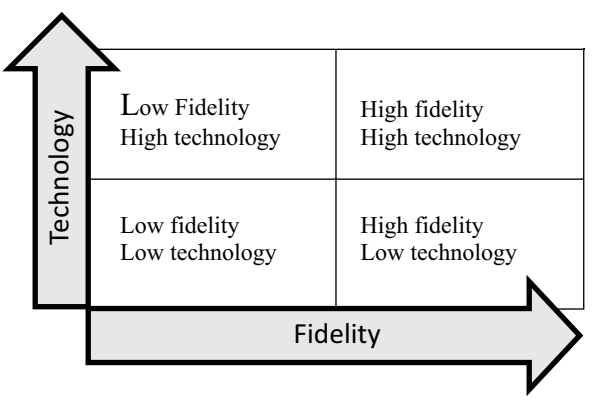


FIGURE 2: Learning environment

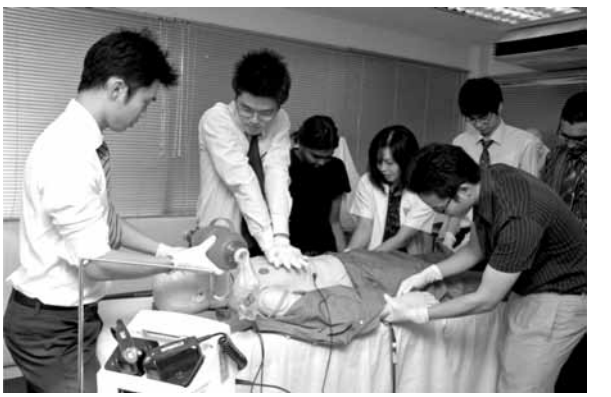


FIGURE 3: Part task trainer

FIGURE 4: Medium fidelity, high technology

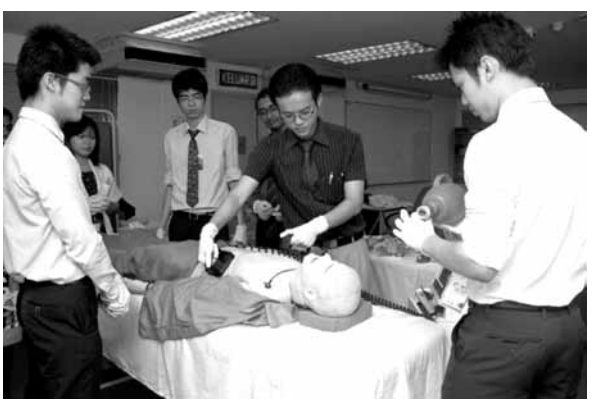


TABLE I: Benefits of Simulation

- Appropriate for all; undergraduates, junior residents, practicing health professionals.
- Safe and minimally stressed environment
- Practised at one's own time
- Required "patient" is always available
- Skills can be sharpened with repeated practice without fear of" hurting" the patient
- Ready and available variety of scenarios at all times
- Training can be tailored to individuals
- Students/trainees gain confidence
- Cultivates divergent thinking
- Feedbacks enhance learning
- Learn from mistakes
- Competency assessment in medical education and throughout medical career

WHAT DRIVES SIMULATION?

Patient safety is foremost in the driver's seat that is pacing this movement towards institutionalising simulation across health care. In the recent years, medical education has increasingly employed the use of simulation in the quest to improve the quality of young doctors coming out into the medical world. This is to meet the demands of the dynamic change in the health care delivery, expectations of the public and the well documented skill deficiencies in fresh graduate doctors.[2,7,8,9, and 10]

To Err Is Human,[11] the report by the Institute of Medicine opened our eyes to the fact that present day health care has resulted in a significant number of deaths due to medical error and since then there has been a surge in efforts made to address patient safety in the healthcare system as well as the undergraduate medical education seeing the need to produce young doctors inculcated with awareness of the possible medical errors and how to avoid or minimize them. Thus, patient safety, a relevant topic not emphasized or taught in the traditional medical education is now increasingly being integrated into the medical curriculum of many universities.

Worldwide, anaesthesiologists make up only a small percentage of the physicians; however anaesthesiology is acknowledged as the leading medical speciality in addressing patient safety and has used simulation to minimize errors and sharpen skills since the 1960s[1,2,12] The formation of Anaesthesia Patient Safety Foundation in 1985 was a landmark that brings together many fields of health care that have a common goal; patient safety. [12]

WHERE ARE WE HEADED?

Simulated teaching has gained a lot of mileage especially since the second half of the 20th century and appears to be picking up speed across the board in the medical community.[1,2,13] Crisis management courses and airway workshops have been conducted using simulation in this country but with only a few established centres it is not as regular as it should be. Simulated teaching and learning is not new it certainly is to some, therefore making it more difficult to teach and establish a clamouring interest in young doctors to participate in any simulation workshop.

'Best Evidence Medical Education' (BEME) systematic review on; the features and uses of high-fidelity medical simulations that lead to effective learning appears to show evidence that high-fidelity medical simulations facilitate learning among trainees when used appropriately.[13]

Evolving issues and drawbacks;

- Patient safety.
- More young doctors entering the practising market; change in doctor: patient ratio affecting number of procedures they perform to
- Less variety of disease presentation (earlier diagnosis, more awareness among public and lower threshold for seeking help early)
- Very few established simulation centres and those available are centred in very few selected areas thus making it inaccessible to practitioners.

- Exposure to diagnosis and management of conditions/diseases that are specific to certain areas/countries are missed.

Ideal future trends;

- Woven into undergraduate curriculum.
- Utilized for training clinical phase students before patient exposure/contact
- Aid assessment in competency at all levels
- House officers trained before being allowed to perform invasive skills harmful to patients.
- Practising doctors undergo regular training to keep up with the trends and sharpen skills.
- Simulation centres accessible to private and government practitioners to upgrade or sharpen their skills.
- Multidisciplinary team training; teamwork and coordination skills among different level of health care workers can be optimized.

CONCLUSION

We seem to be a little slow to catch up though we have started for some time now in Malaysia. Simulated teaching is here to stay and more research needs to be done here on the outcome of this method of training to validate and use it as a tool in delivery of undergraduate and postgraduate curriculum. Generally, evidence show a positive trend and that is promising when looked from the perspective of undergraduate and postgraduate education, patient safety, as well as maintaining a continued level of expertise among working health care givers.

We will not want to be in the hands of a novice if admitted for a procedure for who better versed with the follies that can occur than us!

References

1. Gaba DM. The future vision of simulation in health care. *Qual Saf Health Care* 2004;**13**(Suppl 1):i2-i10.
2. Bradly P. The history of simulation in medical education and possible future directions. *Medical Education* 2006;**40**:254–262
3. Reznek MA. Current status of simulation in education and research. In: Loyd GE, Lake CL, Greenberg RB, eds. Practical Health Care Simulations. *Philadelphia, PA: Elsevier Mosby*; 2004:27–47.
4. Scalese R J, Obeso VT, Issenberg SB. Simulation technology in medical education. *J Gen Intern Med* **23**(Suppl 1):46-9 2007
5. Improving instructional simulation methods (ISIM). Gordon Center for Research in Medical Education (GCRME). University of Miami Miller School of Medicine and The Peter M. Winter Institute for Simulation, Education and Research (WISER), University of Pittsburgh Medical Center
6. Simulation in medicine – a knol by dianesliwka. http://knol.google.com/k/simulation-in-medicine.
7. Carter R, Aitchison M, Mufti G, Scott R. Catheterisation: your urethra in their hands. *BMJ* 1990;**301**:905.
8. Cartwright MS, Reynolds PS, Rodriguez ZM, Breyer WA, Cruz JM. Lumbar puncture experience among medical school graduates: the need for formal procedural skills training. *Med Educ* 2005;**39**(4):437.
9. Feher M, Harris-St John K, Lant A. Blood pressure measurement by junior hospital doctors – a gap in medical education? *Health Trends* 1992;**24**(2):59–61.
10. Maguire GP, Rutter DR. History taking for medical students. Deficiencies in performance. *Lancet* 1976;**2**: 556–8.
11. Kohn LT, Corrigan JM, Donaldson MS. (Eds) (1999) To err is human: Building a safer health system. Washington, DC: *National Academy Press*.
12. Gaba DM. Anaesthesiology as a model for patient safety in health care. *BMJ 2000* **vol 320**:785-8.
13. Issenberg SB, McGaghie WC, Petrusa ER, Gordon DL, Scalese RJ. Features and uses of high–fidelity medical simulations that lead to effective learning: a BEME systematic review. *Medical Teacher*, **Vol 27**, No.1, 2005, pp. 10–28.

Case Study: Anaesthesia Implications in Positioning and Surgical Brainstem Manipulation in Posterior Fossa

Ramanesh Mageswaran
Consultant Anaesthesiologist, Hospital Kuala Lumpur

INTRODUCTION

We describe a case of a 16 year old male who presented with a posterior fossa tumour. This case presented with three admissions to the Neurosurgery Department with three operative procedures done. The issues of hemodynamics and positioning in posterior fossa surgery are highlighted and discussed.

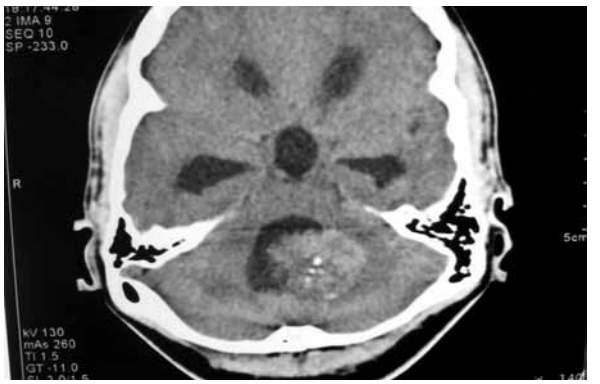
A brief history of his admissions and operative course is discussed below.

FIRST ADMISSION

A 16 year old male patient presented on 17th May 2011 with symptoms of raised intracranial pressure and syncopal attacks. His Glasgow Coma Scale (GCS) was full with cerebellar signs of nystagmus and unsteady gait. Systemic examination of other organs was unremarkable.

The CT scan of the brain (Figure 1 below) showed a fourth ventricle tumour with intraventricular hydrocephalus. His family opted to take him home against medical advice.

FIGURE 1: CT scan brain on admission



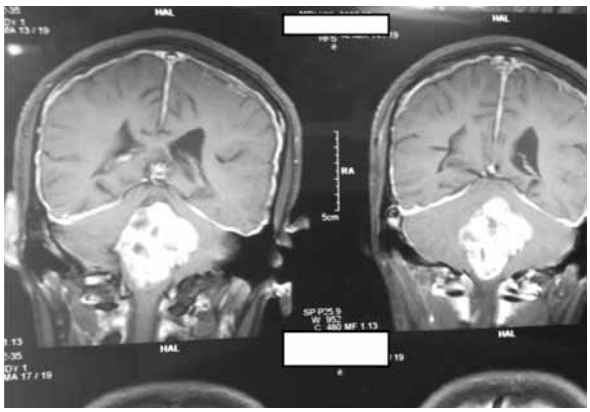
SECOND ADMISSION

One month later he was admitted with worsening headaches and blurring of vision. He underwent a Ventricular Peritoneal (VP) shunt for hydrocephalus in supine position. Intraoperative course was uneventful. He was extubated with an intact cognitive function.

Following the above, he was scheduled for a craniotomy and excision of the posterior fossa tumour. His Glasgow Coma Scale was full and clinically his headaches improved. His blood pressure was 100/60mmHg and heart rate was 100 beats/min while his blood investigations were unremarkable.

His MRI showed a huge posterior fossa tumour (Figure 2), measuring 4.2 x 4.9 x 6.1 cm.

FIGURE 2: MRI brain showing a huge posterior fossa tumor

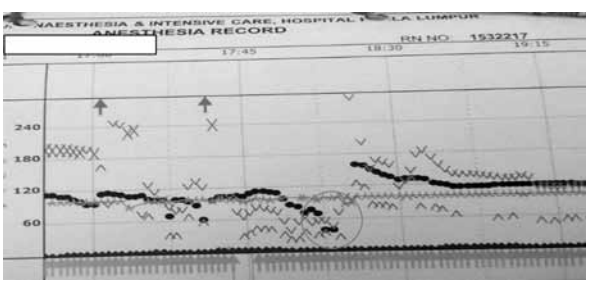


He was induced with intravenous Propofol 130mg (2mg/kg), Fentanyl 100mcg and Rocoronium Bromide 50mg. The trachea was intubated with a 7.5mm armoured endotracheal tube. General anaesthesia was maintained with Oxygen and air in Sevoflurane 2%. Remifentanil infusion using

TCI (Minto Model Effect Site Concentration) was started at 0.5ng/ml and gradually increased to 2ng/ml.

He was positioned prone and this was followed by insertion of Mayfield's head clamps. Hemodynamic parameters were stable. Fifteen minutes after positioning, and prior to the start of the operative procedure there was notable hypotension and bradycardia (BP-60/40mmHg, HR-40 beats/min). Remifentanil infusion was stopped and he was given IV Atropine 0.6mg. There was no improvement noticed and subsequently he was given IV Ephedrine 12mg and IV Adrenaline 200mcg. Despite the above measures, he developed asystole with no pulse and arterial tracing.

FIGURE 3: Intraoperative Record



The surgeon was informed and he was immediately turned supine. With the cardiac monitor showing ventricular fibrillation, CPR was commenced. Resuscitation included defibrillation with 300J and 1 mg of IV Adrenaline. After 2 minutes, he was in sinus rhythm with palpable pulses. The arterial blood pressure was 80/40mmHg with a heart rate of 116 beats/min. Intravenous Dopamine 10mcg/kg/min was started. The ABG showed pH-7.16, O_2-425mmHg, CO_2-52mmHg, Base Excess- -9.2, Lactate-5.1. His pupils were initially dilated and subsequently were 3mm reactive bilaterally.

He was sedated and ventilated in the Neuro ICU. On review the next morning, he was obeying commands. He was then extubated in the afternoon and transferred out of the Neuro ICU by evening, with a full GCS. Once again the family discharged him against medical advice. He was discharged with oral Dexamethasone 8 mg thrice daily.

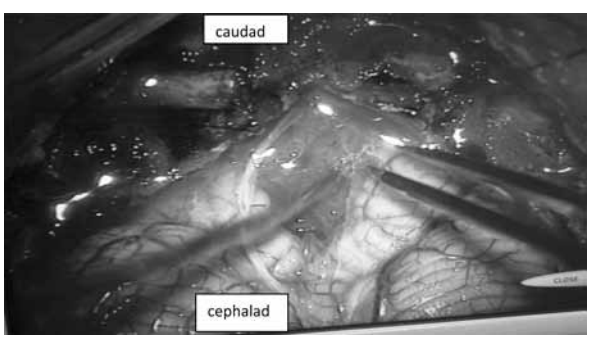
THIRD ADMISSION

The patient came back again three months later, with persistent headaches and difficulty in swallowing. He was scheduled for a craniotomy and an excision of the posterior fossa tumour. Although GCS remained full, this time around he had involvement of the IX cranial nerve. Preoperatively his blood pressure was 110-120/70-80mmHg and with a heart rate of 100-110/min.

Anaesthesia was induced in a similar manner as that of the 2nd procedure. This time continuous monitoring of his hemodynamics (IABP – Intra Arterial Blood Pressure and ECG - Electrocardiography) was maintained during positioning from supine to prone. Five hours into the operation, his heart rate dropped from 120 to 70 beats/min with a mean arterial pressure of 55mmHg. The surgeon was informed and Intravenous Dopamine infusion was started to maintain a mean arterial pressure of 70mmHg.

With inotropic support, the blood pressure was maintained at 120/60mmHg and heart rate was 110-120 beats/min. He experienced a few more episodes of sudden drop in heart rate from 120 to 70 beats/min. Intravenous Atropine 0.6mg was administere and the surgeon was informed. Subsequently the surgical approach was altered. The surgeon started to resect the tumour from

FIGURE 4: Surgical Resection of Tumuor



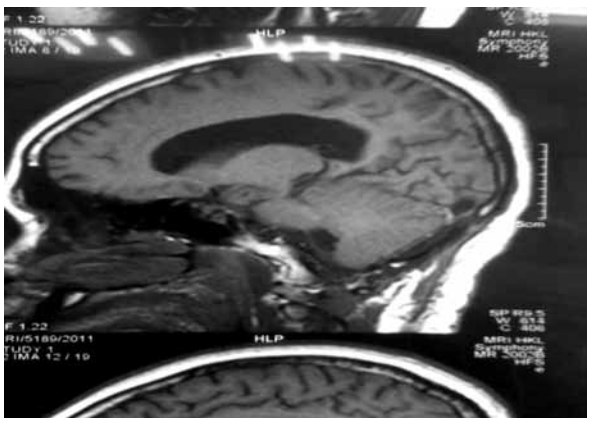
the midbrain to the medulla to minimize the hemodynamic disturbances. The heart rate maintained at 100 to 110 beats/min through-out the operation. His mean arterial pressure was 80mmHg, the inotropes were weaned off.

Post operative findings showed the ventral and lower portion of the tumour directly attached to the medulla causing compression and kinking of the upper medulla.

He was transferred post operatively to the Neuro ICU and planned for delayed emergence in view of the labile hemodynamics intraoperatively and the anticipated oedema due to manipulation of the brainstem. The following day he was opening eyes to call and obeying commands. His cough and gag were present but weak and he subsequently underwent a tracheostomy. He was discharged from Neuro ICU on the 8th post operative day.

Currently, he is undergoing rehabilitation and physiotherapy in the neurosurgical ward.

FIGURE 5: MRI brain post operatively



DISCUSSION

The posterior fossa houses the brainstem and cerebellum. Surgical manipulation of the brainstem especially the pons and medulla can cause rapid haemodynamic changes for example arrhythmias, hypotension, hypertension or even cardiac arrest[1] (Table 1). We postulate that the gravitational impact of the tumour onto the medulla may have aggravated the hemodynamic disturbances. The prone position may also exacerbate hypovolaemia by further reducing venous return and cardiac output.[3]

We highlight the importance of maintaining continuous hemodynamic monitoring (IABP and ECG) while positioning prone for rapid detection of the changes which may precede cardiac arrest.

TABLE I: Effects of Surgical Brainstem Manipulation[2]

Effect of Surgical Brainstem Manipulation		
Brainstem Area	Signs	Changes seen on Monitoring
Cranial Nerve (CN) V	Hypertension, Bradycardia	Arterial pressure, Electrocardiography (ECG)
CN VII	Facial Muscle movement	Electromyography
CN X	Hypotension, Bradycardia	Arterial pressure, ECG
Pons, Medulla	Arrhythmias, hypotension/hypertension, tachycardia/bradycardia, irregular breathing pattern	Arterial pressure, ECG, end-tidal carbon dioxide monitor

The use of Remifentanil in children is associated with significant bradycardia. However, in our case with remifentanil infusion TCI mode at 2 ng/ml target concentration, it's highly unlikely that it can cause haemodynamic disturbances like hypotension and bradycardia.[4]

The issue of sitting versus prone position depends largely on the surgeon and his preference. Posterior fossa surgery in prone position has been favoured because perioperative complications were judged to be less frequent and in particular less severe than in sitting position.[4]

It is important to remember that anticholinergics should be given as a last resort in order that we do not mask cardiovascular responsiveness to surgical manipulation of brainstem structures. Vagally induced bradycardia is a useful indicator for potential brainstem injury. The communication with the surgeon is extremely important at this stage.[1]

This case illustrates the danger of positioning from supine to prone position may result in cardiac arrest. The importance of awareness of this catastrophic complication is important especially so when dealing with posterior fossa tumours. We recommended continuous monitoring during positioning supine to prone position. This allows for early detection of any untoward hemodynamic disturbance and allows for early intervention.

References

1. Gupta A. Anaesthesia for Posterior Fossa Surgery. *Anaesth Intensive Care* 2002;**88**:12-17.
2. Cotrell JE,Young WL. Anaesthesia for Posterior Fossa Surgery. Cotrell and Young's Neuroanaesthesia, *5th Edition* 2010;203-17.
3. Julian B, James R, Jasmeet S, Cardiac arrest during surgery and ventilation in the prone position: a case report and systematic review. *Resuscitation* 2001;**50**:233-38
4. Marsh D.F., Hodkinson B. Remifentanil in paediatric anaesthetic practice. *Anaesthesia* 2009;**64**:301-08.
5. Gilles A.O, Mohamed H, Philippe G.M, et al, Is the sitting or the prone position best for surgery for posterior fossa tumours in children?. *Paediatric Anaesthesia* 2001;**11**:541-47.
6. Hockey B, Leslie K, Williams D.Dexamethasone for Intracranial neurosurgery and anaesthesia. *Journal of Clinical Neuroscience* 2009;**16**:1389-93.
7. Porter J, Pigeon C, Cunnigham A. The Sitting Position In Neurosurgery: A Critical Appraisal. *Br J Anaesth* 1999;**82**:117-28.
8. Domaingue CM. Anaesthesia for Neurosurgery in the Sitting; A Practical Approach. *Anaesth Intensive Care* 2005;**33**:323-31.
9. Harrison EA, Mackersie A, McEwan A. The sitting position for neurosurgery in children: a review of 16 years' experience. *Br J Anaesth* 2002;**88**:12-17.