

1 A comparison of two-months versus two-weeks of internship anaesthesia training

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Introduction

Since the inception of community service in 1998, junior doctors have been required to administer anaesthesia in rural areas with poor or no supervision, despite a lack of technical skills and a need for increased supervision initially.¹

Up until the end of 2006 internship training included a two-week anaesthesia rotation. From 2007, the two-year internship was introduced in Gauteng, with new two-month anaesthesia rotation being instituted from 2008, as part of this two-year internship programme. The goal of this study is to compare the adequacy of internship doctor's knowledge after two-weeks versus two-months of training in anaesthesia.

Methods

After Wits Ethics Committee approval, 108 two-week interns (73% of the intern population) and 107 two-month interns (72% of the intern population) at the Witwatersrand Academic Complex (WAC) were approached at the end of their internship (December 2006 and 2008 respectively). They completed a questionnaire in the form of short questions and case study vignettes, drawn up with a two-tier vetting process to assess basic anaesthetic knowledge as dictated by the HPCSA guidelines.² Demographic data included undergraduate institution and hospital where they had been trained.

Results

The score for the two week interns was 38.95 (14.9) % (mean (SD)). Knowledge of the anaesthetic machine check and anaesthetic pharmacology was inadequate (49% of respondents unable to describe the nitrous oxide pipeline, only 7% of respondents able to give the induction dose of etomidate and 24% unable to list one contraindication to suxamethonium). Analysis of variance showed a difference in the performance of respondents from the different WAC hospitals ($p < 0.05$) and undergraduate institutions ($p < 0.01$).

The score for the two-month interns was 48.95 (21.77) % (median (min,max), an improvement of 10% overall ($p < 0.001$). However, knowledge of the anaesthetic machine check and anaesthetic pharmacology remained inadequate (only 4% of respondents able to give the gauge pressure of a full oxygen cylinder, 63% of respondents unable to give the antidote for a benzodiazepine and 85% unable to give the induction dose of etomidate). Analysis of variance again showed a difference in the performance of respondents from different undergraduate institutions ($p < 0.01$). Further analysis of undergraduate institutions revealed that only 3 out of 6 institutions showed improvement ($p < 0.01$) between two weeks and two months of training in anaesthesia.

Conclusions

- 1) While the two-month anaesthesia rotation appears to improve the anaesthetic knowledge acquired during internship, increased exposure time alone may not be sufficient. Cognisance of other contributing factors should guide the design of the anaesthetic rotation so as to supplement shortfalls (e.g. undergraduate knowledge).
- 2) Anaesthetic knowledge after a two-month internship-training period, as assessed by our questionnaire, shows an improvement over the two-week training period, but still appears to be inadequate for the safe provision of unsupervised anaesthetics during community service.

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2 Adult perioperative fluid management: “Between Scylla and Charybdis”

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Current practices in perioperative fluid management have their origins in the late 1950s and 1960s, when theories regarding salt and water distribution led ultimately to liberal perioperative fluid regimens. Classical teaching assumes replacement of a large preoperative deficit, high maintenance fluid requirements and aggressive replacement of presumed third space losses. Central to this teaching is the avoidance of hypovolaemia. While this remains a crucial objective, recent work suggests that hypervolaemia in the perioperative setting results in a number of deleterious consequences, many of which are seen only in the postoperative period and thus not immediately apparent to the anaesthetist. We are left with the dilemma of trying to avoid hypovolaemia and at the same time, limit fluid excess.

Given the concerns regarding hypervolaemia, it is imperative that we examine our current fluid regimens with a view to minimising potential fluid overload. The fasted patient has less of a fluid deficit than previously assumed and losses due to perspiration are minimal. While significant fluid shifts into the interstitium do occur during surgery, much of this is due to the aggressive use of crystalloid. Despite this evidence liberal fluid regimens continue to be recommended.

This review aims to critically examine some of the accepted principles behind perioperative fluid strategies. The underlying physiology will be examined, with special reference to Starling's forces and the impact of fluid loading on the

endothelial barrier. The nature and magnitude of the perioperative fluid deficit will be discussed, as well as appropriate ways to address it. It will also look at maintenance fluid requirements during surgery and discuss insensible and ‘third space’ losses. Particular attention will be paid to the consequences of hypervolaemia. Finally, it will make practical suggestions regarding possible alterations of our fluid strategies in the perioperative period.

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3 Posttetanic facilitation: A clinical test for safe reversal of nondepolarising neuromuscular blockade.

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Introduction

During the 1970s and 1980s, regaining a train-of-four ratio (TOFR) of 0.7 after administration of non-depolarising neuromuscular blocking drugs, was regarded as an indication of return of mechanical respiratory reserve and the ability to maintain a patent airway.^{1,2} Evidence has accumulated that patients sent to recovery rooms with TOFR less than 0.9 have impaired function of their pharyngeal muscles that predisposes them to regurgitation and aspiration,^{3,4} and an increased risk for developing postoperative pulmonary complications.⁵ Furthermore it appears that a TOFR < 0.9 is associated with decreased chemoreceptor sensitivity to hypoxia.⁶⁻¹² In spite of being able to maintain a sustained head lift and leg lift, patients had difficulty in swallowing, felt uncomfortable and could not sip water through a straw.¹³ Residual neuromuscular block is a major risk factor behind critical events in the immediate postoperative period and should be regarded as a serious adverse event in the same way as we regard ventilatory depression due to opioids and anaesthetic agents.¹⁴

Clinical assessment of neuromuscular blockade by visual and tactile evaluation of the responses to train-of-four stimuli (TOF) to the ulnar nerve at the wrist is imprecise and it has been demonstrated that 20% of experienced anaesthesiologists are unable to detect residual neuromuscular blockade in the presence of a TOFR of 0.4.¹⁵ To make matters worse, devices for objective measurement of TOFR are seldom available in clinical practice. For this reason Viby-Mogenson and others developed the evaluation of double-burst stimulation (DBS), as an improved method for clinical assessment of the adequacy of reversal of non-depolarising neuromuscular blockade (NMB).^{16,17} However it was soon demonstrated that although more sensitive than clinical evaluation of TOF, many anaesthesiologists still could not detect significant residual NMB.¹⁶ There is therefore no guarantee that in spite of careful assessment of DBS, the absence of fade implies adequate, safe reversal of NMB.¹⁸ The purpose of this study was to establish whether the absence of posttetanic facilitation (PTF), long regarded as the "hallmark" of non-depolarising neuromuscular blockade,¹⁹ could be a reliable indicator of adequate reversal of drug effects. The hypotheses were:

1. The presence of PTF is a more sensitive indicator of residual non-depolarising block than DBS.

2. The absence of PTF is a reliable indicator that the TOF-ratio is ≥ 0.9 .

Methods

After obtaining institutional approval for the study, informed, written consent was obtained from 40 adult, ASA I-III patients scheduled for elective surgery of expected duration longer than one hour. In the operating room the following nerve-stimulator devices were attached:

- a) To the "Control arm": an Innervator 272[®] nerve stimulator (Fisher & Paykel) for visual and tactile assessment of NMB.
- b) To the "Test arm": a TOF-Guard[®] monitor (Organon Teknika) for recording of TOFR by accelerometry. The arm, wrist and fingers were immobilised while ensuring free movement of the preloaded thumb according to recommended procedure.²⁰

After induction of anaesthesia with propofol and fentanyl, the TOF-Guard[®] was calibrated, cis-atracurium 0.15mg.kg⁻¹ was administered and the trachea intubated. Controlled ventilation of the lungs followed using 66% nitrous oxide in oxygen and a 0.7 kPa endtidal partial pressure of isoflurane.

On return of four responses to TOF on the control arm, a trained observer who was blinded to the TOFR readings on the Control arm assessed neuromuscular blockade on the Test arm visually and by feel. When it appeared that the four responses to TOF were equal, the observer then switched to assessment of DBS until fade could no longer be detected. At this point patients were randomly allotted to one of two groups. Group-1 was tested for the presence of PTF. In addition the TOFR was determined at which DBS-fade was undetectable. Group-2 was allowed to progress to a TOFR of 0.9 whereupon they were tested for the presence of PTF (to determine whether PTF was present at TOFR ≥ 0.9).

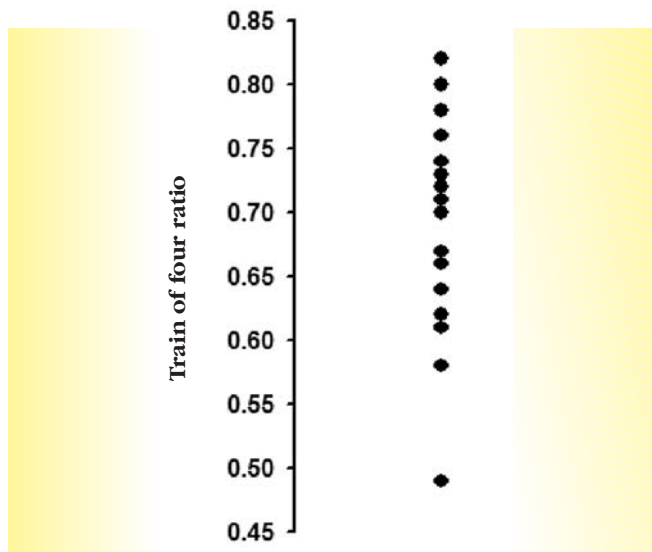
Results

In both groups fade on TOF and DBS were no longer detectable on the Test arm at TOFR of 0.4 and 0.7 respectively. The distribution of the TOFR in Group-1 is illustrated in Figure 1. PTF was present in all of these cases, notably when TOFR was between 0.7 and 0.83. The reliability of PTF to predict the presence of a TOFR < 0.9 is presented in Table 1.

Table1:

Sensitivity % (95% CI)	Specificity % (95% CI)	Positive Likelihood Ratio	Negative Likelihood Ratio	Positive Predictive Value (%)	Negative Predictive Value (%)
100 (83–100)	85.0 (63.1– 96.6)	6.7	0.0	87.0	100.0

Figure 1:



Conclusions

1. DBS is not a reliable method to determine whether a patient has recovered safely after nondepolarising neuromuscular blockade.
2. The presence PTF is a more sensitive indicator of residual non-depolarising block than DBS (when TOFR < 0.9).
3. The absence of PTF is a reliable indicator that the patient has safely recovered from NMB (when TOFR > 0.9).
4. PTF is simple and can easily be performed using any basic nerve stimulator, to ensure safe reversal of NMB.

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95% CI = 95% confidence interval.

4 A comparative study evaluating the effectiveness and safety of the generic sevoflurane (Sojourn, Safeline) and the original sevoflurane (Ultane, Abbott)

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Introduction

This study attempted to evaluate the effectiveness and safety of the generic product of sevoflurane. "Effective" for the purpose of this study refers to the ability of the drug to ensure an acceptable level of general anaesthesia.

Methods

Ethical approval was obtained. Ethics Committee reference number: M08/07/035. Sixty patients were included in this study. Inclusion criteria included: patients scheduled for elective surgery on the gynaecological and orthopaedic lists were used. It was emphasised to the patients that they would receive an appropriate anaesthetic for their particular surgery and if it meant that the best option would be general inhalation anaesthesia, they were asked for consent. Patients who were excluded: patients with a previous reaction to inhalation anaesthesia, malignant hyperthermia history, pregnant patients, children younger than 18 years, patients in whom an alternative anaesthetic technique would be in their best interest, a patient who refuses to participate in the experiment.

Premedication, anaesthetic and postoperative analgesia were standard. Standard monitoring was applied. A BIS monitor was used in every patient and it was accepted that a value between 30 – 45 would imply effective general anaesthesia (read in conjunction with the normal clinical indices of effective anaesthesia). A clinical technologist decided on which of the two options (Sojourn or Ultane) would be used first (by blind card draw). The vaporisers were both in line prior to the commencement of anaesthesia. Hence the study could not be blinded to researcher as she had to adjust the depth of anaesthesia as necessary. The anaesthetic was induced and maintained as clinically relevant with the BIS being stabilised between 30 and 45. Fifteen minutes after stabilisation of the expired inhalation anaesthetic concentration, patient data was obtained. The initial vaporiser was closed and the second vaporiser opened at the same concentration without changing either the ventilation or fresh gas flow. At 5, 10 and 15 minutes after the change over of the ventilator, patient data was again collected. The clinician had the right to adjust the anaesthetic depth as required in the best interest of the patient. However,

once the vaporiser was changed, no changes were done (or were necessary).

Data was stored on an Excel spreadsheet and analysed with the software package Sigmasat. One way repeated measures ANOVA was used. If the test failed the normality test, the one way ANOVA was done on ranks. Post-hoc differences between groups were sought with the Tukey test. Comparison was done between the values obtained after stabilisation plus 15 minutes of the first drug and the subsequent 5, 10 and 15 minutes of the second drug.

Results

- BIS value: ANOVA on ranks were performed. No difference between the groups could be demonstrated ($p = 0.867$). The median values for 15, 5 and 10 minutes were: 38, 37, 36, 36.
- Inspired and expired concentration for the inhalation agent did not differ between groups for inspired ($p = 0.328$) and expired ($p = 0.368$) sevoflurane.
- The saturation ($p = 0.974$) and the alveolar ventilation ($p = 0.854$) did not vary between the 15, 5, 10 and 15 minutes.
- The fresh gas flow, tidal volume and frequency were stable and as per the protocol the p valued = 1.
- The temperature and heart rate ($p = 0.990$ and $p = 0.578$) and not change.
- Changes were detected in the systolic blood pressure.
- The normality test failed and a one way analysis of variants on ranks were performed. Median values for the 15 minutes were 110 mmHg, 5 minutes 115 mmHg, at 10 minutes 120 mmHg and at 15 minutes 117 mmHg. ($H = 17.13$ with 3 degrees of freedom, $p = 0.001$)
- The Tukey test showed that the 15 minutes before change of vaporiser differed from the 10 minutes and 15 minutes post change.

Conclusion

The initial hypothesis was satisfied i.e. there was no difference in the effectiveness of the generic and the original sevoflurane products. No side effects were registered for any of the drugs. There was a variation in systolic pressure after the switch of the anaesthetic agent but this cannot be ascribed to any specific drug.

5 Minimally invasive cardiac output monitoring during spinal anaesthesia for Caesarean section

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Heart rate and blood pressure are appropriately used as surrogate markers of maternal cardiac output (CO) during spinal anaesthesia (SA) for Caesarean section (CS). Maintenance of baseline maternal blood pressure, using phenylephrine, has been shown to produce the closest to zero umbilical arterial base deficit, despite the fact that phenylephrine may depress maternal cardiac output.^{1,2} However, the maximum change in cardiac output has been shown to correlate better with uteroplacental blood flow than upper arm blood pressure.³ The maintenance of blood pressure and maternal cardiac output are therefore both important for maternal safety and comfort, and fetal wellbeing.⁴ Two studies of cardiac output changes during SA for CS are presented, employing pulse wave form analysis (LiDCOplus, LiDCO Ltd, Cambridge, UK).

The first was an observational study of 15 patients with severe preeclampsia, requiring CS for a maternal indication.⁵ CO and systemic vascular resistance (SVR) were derived from the measured stroke volume, heart rate and mean arterial pressure (MAP). In addition, the haemodynamic effects of phenylephrine, and the response to oxytocin were recorded. Haemodynamic values were averaged for defined time intervals. MAP and SVR decreased, and there were significant differences between baseline values and the values from the time of adoption of the supine position until the end of surgery. CO remained stable from induction of SA until the time of request for analgesia. At the time of peak oxytocin effect, CO and HR were higher and SVR lower than at all other time intervals. Bolus phenylephrine increased MAP to above target values, and did not significantly change CO. At the time of recovery from SA, there were no clinically relevant changes from baseline haemodynamic values. This study suggests that SA for CS provides modest afterload reduction in treated patients with severe preeclampsia.

The second study was a prospective, randomised trial comparing the effects of bolus phenylephrine and ephedrine on maternal

CO. Secondary outcomes included the effects of SA and oxytocin on maternal haemodynamics. Pulse wave form analysis and thoracic electrical bioimpedance (BioZ, Cardio Dynamics International, San Diego, CA, USA) were used to estimate stroke volume changes independently in each patient. A 20% decrease in MAP following SA was associated with an increase in HR and CO relative to baseline values. CO decreased following phenylephrine, and increased after ephedrine. After oxytocin, SVR decreased, and heart rate and CO increased. Co-administration of phenylephrine obtunded the changes due to oxytocin. The two CO monitors recorded similar trends in changes in CO.

The use of minimally invasive cardiac output monitors in these two studies has significantly increased our knowledge of the mechanism of SA in healthy and preeclamptic patients, as well as describing the effects of the vasopressors ephedrine and phenylephrine. This should contribute to appropriate patient management and improved safety during SA for CS.

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6 Acute potassium changes during CPB

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Introduction

Cardiopulmonary bypass (CPB) necessary for valve replacements and coronary artery surgery is associated with various metabolic changes intraoperatively which we as anaesthetists must understand to protect our patients. The aim of this review is to look at potassium changes intraoperatively during CPB. Potassium is the principal intracellular cation of our body that plays an important role in the excitability of vital cell membrane physiology, especially in cardiac muscles to maintain resting membrane potentials and cardiac conduction.^{1,2,3,4} Therefore, any extreme intraoperative K⁺ changes should be corrected to prevent arrhythmias that influence cardiac output and oxygen delivery intraoperatively.

Methods

A literature search relevant to physiology and factors influencing potassium was done through a MEDLINE search that produced 17 articles of which 3 articles was applicable and available. Further methods included a search of current textbooks and journal articles.

Results

Potassium concentration rarely remains constant during or after CPB because of various insults to normal physiology. Distribution between the inside and outside of cells depends on intraoperative physiological changes like CO₂ tension, arterial pH, hypothermia⁵ and stress hormones (cortisol, increased endogenous catecholamines, aldosterone, vasoactive substances^{1,2}). Patient determinants like preoperative kidney function and potassium regulation, integrity of renal tubular cells, diuretics, antihypertensives and digitalis use can lead to K⁺ changes during CPB.¹

Pharmacological agents like insulin, glucocorticosteroids and catecholamine infusions causes extracellular and intracellular K⁺ shifts.¹ Preoperative heparin administration^{6,7,8} and additional mannitol, magnesium supplements⁹ and albumin² may also play a role. Further K⁺ changes may be due to acute renal failure which is a recognisable complication of CPB because of the unphysiological non pulsatile bloodflow that upset autoregulatory mechanism of renal blood flow. Other factors are systemic uptake of potassium containing cardioplegia² and myocardial protectants, priming solutions, dialysate, haemodilution⁵ and administration of large amounts of bank blood with high K⁺ concentrations. Bypass

effects (haemolysis, tissue damage, acidosis, inflammatory responses, reperfusion injury) aorta cross clamp time and on-pump time leads to the release of K⁺ through the loss of cell membrane integrity.^{2,10} Causes of hypokalaemia are urinary loss, dilution with K⁺-deficient prime, diuretic therapy and haemodilution with forced diuresis during CPB. The latter two are considered to be the main causes of enhanced electrolyte depletion.

These multiple interacting factors represent potential routes for acute changes in potassium levels and eventual myocardial dysfunction.

Conclusion

Potassium balance disorders during CPB are not widely investigated and there are only a few studies available. The knowledge of potassium is a necessity for a practising anaesthesiologist seeing that the role of potassium in the pathogenesis of cardio arrhythmias is well recognised.

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7 Ischaemic preconditioning – The molecular mechanisms and the influence of general anaesthesia

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Introduction

The modern anaesthetist is in a privileged position to favourably influence cardiac outcomes during surgery.¹ Poor myocardial protection rather than inadequate anatomical repair is recognised as the primary determinant of low cardiac output after cardiac bypass.² The phenomenon of ischaemic preconditioning and more recently post-conditioning,^{3,4} is known to increase the heart's resistance to ischaemic insults.⁵ Ideally, the physician would like to activate the intracellular mechanisms instrumental in bringing about ischaemic preconditioning by means of a pharmacological agent, without inducing ischemia per se.⁶ Specifically, the use of halogenated inhalational anaesthetics to induce preconditioning has garnered attention recently for its role in manipulating the cellular mechanisms instrumental in enhancing the heart's resistance to ischaemic insults.^{1,7} This review details the specific cellular mechanisms involved in ischaemic preconditioning together with a discussion of the pharmacological manipulation of these pathways.

Methods

Medline search used find articles pertaining to ischaemic and anaesthetic induced preconditioning.

Results

Anaesthetic preconditioning with halogenated volatile agents appears to utilise the same cellular apparatus as do ischaemic preconditioning, since a combination of ischaemic and anaesthetic preconditioning does not produce an additive result.⁸

The key intracellular mediator of preconditioning appears to be the Mitochondrial K_{ATP} channels.^{9,10} Halogenated inhalational agents either open the Mito K_{ATP} channels directly or indirectly prime the Mito K_{ATP}, to increase the probability of Mito K_{ATP} channels being in an open state.⁶ PKC may play an important role in activating K_{ATP} channels.¹¹ Opening the Mito K_{ATP} channels results in dissolution of the internal mitochondrial membrane potential and consequently blunts Ca²⁺ overload in the mitochondria and restores functional coupling between the mitochondria and ATP utilising cytosolic sites.

Additionally the Mitochondrial permeability transition pore (mPTP),⁴ NO, Reactive oxygen species (ROS)¹² and intracellular kinases⁷ also appear to have a vital role in anaesthetic preconditioning.

Novel approaches to anaesthetic preconditioning involve inhibition of the Na⁺/H⁺ exchanger.¹³ Recent clinical studies like the EXPEDITION trial showed markedly improved perioperative cardiac outcomes, but the trial was terminated prematurely due to increased focal neurological events on day five. Na⁺/H⁺ exchange inhibitors (eniporide and zoniporide) work by reducing

Sodium accretion during ischemia and secondarily reduced Ca²⁺ levels in the ischaemic myocytes.

Inhibition of RSK-family of kinases with the aim of indirectly inhibiting the Na⁺/H⁺ exchanger is currently under investigation for possible future therapeutic use.

Results

Researchers constantly strive to identify novel ways to induce pre and post conditioning in the myocardium. By understanding the underlying mechanisms of preconditioning, the Anaesthetist will be well prepared to judge the potential merit of emerging therapeutic strategies aimed at improving perioperative cardiac outcomes.

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8 Coagulopathy in renal dysfunction

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Introduction

Coagulopathy in renal dysfunction is a well known phenomenon. Much has been researched and written on the topic and it is clear that the pathogenesis is multifactorial. Most emphasis has been on the uraemic platelet dysfunction but it is now clear that other factors also play an important role.

Methods

MEDLINE Search of review and research articles under the following keywords: Renal dysfunction; Renal failure; Renal disease; Uremia; Platelet dysfunction; Coagulopathy; Bleeding; Thrombosis; Platelet function testing.

Results

Factors such as platelet-endothelial interaction defects, effects of dialysis on coagulation, anaemia of chronic renal failure, and concomitant drug use. The coagulopathy in most cases presents as a hypocoagulable state with bleeding diathesis but under certain conditions there might also be a state of hypercoagulability that may lead to thrombotic complications. The population of patients with renal dysfunction often presents to theatre for a wide range of surgery.

Conclusion

The anaesthetist should have knowledge of the effects of renal dysfunction on coagulation and of the various options to treat and improve the condition to minimise risk to the patient. This

overview lecture will address the pathophysiology, various treatment options and tests available to manage coagulopathy in renal dysfunction.

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9 Airway safety in a tertiary South African hospital and beyond...

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Management of airway is central to practice of anaesthesia. Failure to maintain adequate gas exchange is catastrophic. ASA Closed Claims Analysis shows 37% of adverse outcomes associated with respiratory events were attributable to anaesthesia. Brain damage occurred in 85% and 72% were considered preventable. Care was considered substandard in 90% of claims associated with inadequate ventilation.³

"There is no universal consensus for optimal management and equipment used in difficult airway" Previous work suggests that optimal management of difficult airway relies on anaesthesiologist's experience, skill and familiarity with airway devices, than devices themselves.^{1,2}

Training of Difficult Airway management (DAM) in the Departments of Anaesthesia at University of Pretoria has never been formalised. A model utilised by Baystate Medical centre in the New York³ is being implemented. This is a project in evolution.

Step 1

Standardised DIFFICULT AIRWAY CARTS (DAC) are essential, not only in theatres but in all hospital locations where airways are routinely managed. Directors of ICUs and Casualty departments were approached and contents of DAC were discussed and compiled according to specific requirements. In order to expedite the process, each director was supplied with full lists of disposables in table form as well as product codes and distributors. This information has now been published nationally in SASA guidelines.⁴ Maintenance of the cart and further training in basic and advanced airway management was also discussed.

Step 2

Pertains to training of anaesthetic registrars in standard/advanced

airway management. Baystate model advocates 2 month dedicated airway rotation. Presently Pretoria departments don't have dedicated airway block, but registrars on "Acute Pain" rotation are encouraged to witness all scheduled difficult intubations and to check DAC. Also attendance at a formal workshop is mandatory.

Step 3

Still needs to be developed, a system of assessment of airway competency for registrars

Conclusion

Both personal and systems failures (e.g. no DAM cart) result in unanticipated DAM. Carts and simplified algorithms should be made mandatory in South Africa.

Anaesthesiologists lack DAM training. Teaching of airway skills should be formalised and made essential for registration. Standards for DAM and curricula for CME are essential.

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10 Cricoid pressure: Friend or Foe?

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Cricoid pressure was popularised by Sellick in 1961 as a simple method to reduce the risk of aspiration during induction of anaesthesia. It has since become the standard of care for most anaesthetists. Although it appears to be a mechanically simple and anatomically correct manoeuvre, it is in fact complex and difficult to perform optimally. There is also continued controversy regarding its safety and efficacy. Understanding the role of cricoid pressure in our practice requires knowledge of anatomic relationships, physiology of regurgitation, modes of application, timing, amount of force required to apply it effectively and the impact of cricoid pressure on airway management. With reference to airway management we need to recognise the effects of cricoid pressure on face-mask ventilation, insertion of the laryngeal mask airway (LMA), laryngoscopy and endotracheal tube placement. Additionally we need to be aware of the side effects and potential complications of its use. Current evidence suggests that the widespread and continued application of cricoid pressure in emergency airway management is questionable. Awareness of the benefits and potential problems with the technique will allow the clinician to decide when best to use

the manoeuvre, and whether to eliminate it from one's anaesthetic practice altogether.

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II The clinical characteristics and outcomes of patients with lone atrial fibrillation at Groote Schuur Hospital

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Introduction

Atrial fibrillation (AF) is a common arrhythmia and is often difficult to manage. The classical risk factors for AF include hypertension, valvular disease, thyroid disease, cardiomyopathies, including ischaemic cardiomyopathies. When AF represents an electrophysiological phenomenon in structurally normal hearts it is termed lone AF. There are currently no studies to describe the clinical characteristics and outcomes of patients with lone AF in Africa. This study's purpose is to describe the clinical characteristics and outcomes of Lone AF patients attending Groote Schuur Hospital (GSH).

Methods

A retrospective descriptive study in which 289 medical records of patients with AF at the GSH Cardiac Clinic were reviewed (1992–2006). Clinical data, electrocardiograms, echocardiograms and laboratory results were interrogated to exclude identifiable causes of AF. Information on clinical characteristics and outcomes was entered into a data-entry form. Baseline descriptive statistics were expressed as means and range for continuous variables and counts with percentages for categorical variables.

Results

Forty two patients were identified as having lone AF and had a mean follow-up time of 5.8 years. Males comprised 57% (n = 24) and females 43% (n = 18). The mean age for males was 46 years with no males being older than 65 at diagnosis. The mean age of females was 62.4 years, 55% (n = 10) being less

than 65 (mean age 45) at diagnosis. Fifty percent (n = 21) were white, 36% (n = 15) were mixed race, 7% (n = 3) were black and 7% (n = 3) did not have their race specified. Forty three percent (n = 18) had a normal weight, 36% (n = 15) were overweight and 21% (n = 9) were not specified. Sixty two percent (n = 26) had paroxysmal AF, 38% (n = 16) had chronic AF and 12% (n = 5) progressed from paroxysmal to chronic AF. Presenting complaints were palpitations (73%), dizziness (66%), dyspnoea (46%), near blackouts (41%), chest pain (22%) and fatigue (22%). The mean duration of symptoms prior to diagnosis was 7.7 years. Complications included stroke (10%) (n = 4); tachycardia-related cardiomyopathy (17%) (n = 7) and heart-failure (5%) (n = 2). No mortalities were recorded. Eighty five percent (n = 25) were on betablockers, 22% (n = 9) progressed onto amiodorone, 12% (n = 5) had radiofrequency ablations and 10% (n = 4) eventually had atrioventricular nodal ablations with permanent pacemaker insertion. Sixty eight percent (n = 28) were on warfarin and 27% (n = 11) were on aspirin for prevention of thromboembolic complications. Seven percent (n = 3) had bleeding complications while on anticoagulation therapy.

Conclusions

Lone AF is a relatively uncommon condition with a preponderance to white, thin, middle-aged males. The symptoms of lone AF can be debilitating with associated morbidity but no mortality was recorded in our cohort. Rate control and appropriate anticoagulation are the cornerstones of patient management.

12 Negative pressure pulmonary oedema

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Introduction

Negative Pressure Pulmonary Oedema (NPPE) is associated with upper airway obstruction of various aetiologies. By far the commonest cause is post-operative laryngospasm.¹ In the pre-operative period airway tumors or other airway pathologies are the commonest causes.²

Classically described in males, ASA I-II (who are able to generate significant negative intrathoracic pressure), NPPE has a rapid onset and rapid resolution clinically and radiographically (usually within 12–24 hrs).^{1,3}

NPPE is triggered by the Mueller manoeuvre (inspiration against an obstructed upper airway).³

The ensuing hydrostatic pulmonary oedema is the result of an interesting chain of pathophysiological events, which ultimately lead to increased pulmonary vascular blood volume with resultant increases in pulmonary capillary hydrostatic pressure.¹

Methods

Case study and literature review from 1977 to 2008.

Results

Generation of negative intrathoracic pressure is the catalyst in the pathophysiological cascade.

Firstly, venous return is augmented as a result of decreases in right atrial pressure (due to transmission of negative intrathoracic pressure to the heart) and an increase in mean systemic venous pressure (catecholamine-induced venoconstriction, stimulated by hypoxia and hypercarbia).^{1,3}

Secondly, increased venous return affects ventricular interdependence. Increased right-sided filling pressures result in a left-shift of the interventricular septum, leading to a decrease in left ventricular (LV) diastolic compliance and subsequent decrease in LV stroke volume. Concomitant hypoxia and acidosis further depress myocardial contractility.¹⁻³

Lastly, the net effect of catecholamine-induced increase in systemic vascular resistance and the reduction of intrathoracic pressure is an increased LV transmural pressure. Thus LV wall tension is increased and LV ejection decreased.

The ultimate result is the translocation of blood from the systemic to the pulmonary circulation. According to Starling forces,

hydrostatic oedema^{4,5} ensues. (Pulmonary hydrostatic pressure is increased due to higher pulmonary blood volumes and hypoxic vasoconstriction.)

The traditional classification of pulmonary oedema as either cardiogenic (hydrostatic oedema) or noncardiogenic (increased-permeability oedema)⁶ is challenged by the entity of NPPE. NPPE is a dynamic process that is precipitated by negative surges in the intrathoracic pressure with a resultant elevated hydrostatic pressure in the pulmonary capillary bed. This is despite the integrity of the pulmonary capillary bed being intact and the patient having a structurally normal heart.

Management entails maintaining oxygenation by re-establishing airway patency and application of positive airway pressure (intubation and ventilation is usually described in literature case reports). Diuretics are controversial, as resolution is rapid regardless. Remember to manage the underlying condition (e.g. thyroid goitre causing airway obstruction) if applicable.⁷

Conclusion

It is important to be aware of the entity of negative pressure pulmonary oedema as this directs the caring physician to managing the patient's upper airway obstruction in the appropriate clinical setting.⁸

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13 Is endovascular aortic aneurysm repair intermediate risk noncardiac surgery? A meta-analysis of cardiac morbidity reported in the randomised controlled trials comparing open and endovascular aortic aneurysm repair

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Introduction

The American College of Cardiology (ACC)/ American Heart Association (AHA) guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery has classified endovascular aortic aneurysm repair (EVAR) as intermediate-risk surgery. This guideline is not evidence-based. The aim of this study was to determine the incidence of major cardiac morbidity and mortality reported in the prospective randomised controlled trials of elective open abdominal aortic aneurysm (AAA) repair versus EVAR. By definition, intermediate surgery should have a combined incidence of 30-day cardiac death and nonfatal myocardial infarction of 1 to 5%, and major surgery should exceed 5%.

Methods

We conducted a meta-analysis of randomised controlled trials of open AAA repair versus EVAR for elective surgery. Pubmed Central was searched from 1966 to 2008 and EMBASE from 1996 to week 20 of 2008. The search terms included 'endovascular', 'aorta' and 'randomised'. Data on mortality (all-cause and cardiac), nonfatal myocardial infarction and cardiac death was extracted for all patients on an intention-to-treat basis.

Results

Two-hundred and forty-three potential publications were identified. Eleven publications which reported on five randomised trials met the inclusion criteria. All-cause mortality at 30 days was significantly less in EVAR group compared to open AAA repair (odds ratio (OR) 0.43, 95% confidence interval (CI) 0.26-0.73, $P = 0.002$), but not at 2 years (OR 1.0, 95% CI 0.80-1.26, $P = 0.97$). Thirty-day noncardiovascular mortality was significantly less in the EVAR group (OR 0.45, 95% CI 0.26-0.77, $P = 0.004$) but not cardiovascular mortality (OR 0.40, 95% CI 0.08-1.98, $P = 0.26$). Thirty-day major adverse cardiac events (MACE) were not significantly different between the groups (OR 0.80, 95% CI 0.21-3.0, $P = 0.74$). The incidence of MACE was 2% in both the EVAR and open AAA groups.

Conclusions

There is no prospective randomised evidence to suggest that the cardiac morbidity associated with EVAR is significantly less than open AAA. The low incidence of MACE in the open AAA group raises questions about the validity of the data analysed. Indeed 98% of this data was from a single study with no standard definitions, or surveillance for, cardiac events. Categorisation of EVAR into intermediate or major noncardiac surgery based on the prospective randomised data is therefore impossible.

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14 The paediatric electrocardiogram

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Introduction

Although the basic principles of interpretation of the electrocardiogram (ECG) are identical in children and adults, the paediatric ECG differs significantly from the adult ECG.¹ Interpretation of the paediatric ECG depends on patient age, reflecting the developmental changes in anatomy and physiology that occur in the growing infant or child.^{2,3} In order to avoid errors in interpretation, anaesthesiologists, paediatricians, emergency physicians, etc should be familiar with the normal paediatric ECG and should be aware of the common ECG abnormalities occurring in children.²

Methods

A Pubmed search for recent literature (1990–2008) was conducted in conjunction with the use of textbooks available at the Frik Scott Library at the University of the Free State.

Results

Factors contributing to the dynamic nature of the paediatric ECG include: (1) the anatomical dominance of the right ventricle during the neonatal period due to high pulmonary vascular resistance in utero;^{2,3} (2) increase in vagal tone with ageing;^{2,3} (3) reduced cardiac muscle mass;² (4) developmental changes in body size, position and size of the heart relative to the body, and of the cardiac chambers relative to each other;³ and (5) the presence of maternal autoimmune disease and transplacentally acquired maternal antibodies.^{2,3} The electrocardiographic variables influenced by the above-mentioned factors include the heart rate, P wave morphology, PR interval, mean frontal plane QRS axis, QRS duration, R and S wave amplitudes and progression, QT interval, T wave morphology and atrioventricular conduction,

all demonstrating age-related changes.^{1–4} Another major factor complicating interpretation of the paediatric ECG is congenital heart disease, which affects approximately 1% of newborns.⁵ As the number of infants, children and adults with surgically corrected congenital heart disease is growing – a population that is particularly prone to develop cardiac arrhythmias – knowledge of the arrhythmias commonly associated with congenital heart disease becomes essential.^{5,6}

Conclusion

Accurate interpretation of the paediatric ECG depends on appreciation of the developmental changes in anatomy and physiology and knowledge of the common arrhythmias associated with congenital heart disease.

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