Contents

Pages

Editorial
4 What is New in the 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care?

Special Article
11 The Science and Art Behind the Enhanced Stabilization of Sevoflurane.

Clinical Investigations
14 Cuffed Oropharyngeal Airway in Basic Airway Management.
20 Co-administration of Phenylephrine to Prevent Systemic Hypotension During Induction of Anesthesia with Propofol.
26 Pre-oxygenation Using Circle Absorber.
32 Shunting After Release of Aortic Cross-Clamp During Coronary Artery Bypass Graft Surgery.
36 Periprostatic Local Anesthetic Block for Prostatic Biopsy. A Prospective Controlled Study.

Case Report
40 A Case Report of Being Awake at Bispectral Index of 40.

College Business
46 Report – Board of Education
47 Reports – Boards of Pain Medicine and Intensive Care Medicine
48 Formal Projects
49 College Press Release

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Results: Present the results in logical sequence in the text, tables, and illustrations.

Discussion: Emphasize the new and important aspects of the study and conclusions that follow from them. Include in the Discussion the implications of the findings and their limitations and relate the observations to other relevant studies. Link the conclusions with goals of the study but avoid unqualified statements and conclusions not completely supported by the data.

Abbreviations and Units of Measurement
Units of measurement: Measurements of distance/length and weight must be expressed in metric units only. Clinical laboratory and hematologic data must be expressed in SI units with, if desired, present conventional metric units in parentheses. Continue using abbreviations consistently; do not revert to the spelled-out term.

References
All references must be available to all readers. Cite only references to books and articles or abstracts published in peer-reviewed journals. Number references consecutively in the order in which they are first mentioned in the text. Double-space between all lines of each reference and between references when typing the reference page. Identify references in text, tables, and legends by arabic numerals. References must be verified by the author(s) against the original documents, and the entire list must be checked for nonduplication. Use the style of the examples below:

Examples:
Standard journal articles (List all the authors when four or less; when five or more, list only the first three and add et al.;): Lam DW, Chui PT. A prospective evaluation of a microcoagulation analyzer. Bull HK Coll Anaesthesiol 2005;14:9-12.

Tables
Type each table double-spaced on a separate sheet. Number tables consecutively and supply a brief title for each. Give each column a short or abbreviated heading. Place explanatory matter in footnotes, not in the heading. In footnotes, define all abbreviations that are used in each table. Repeat definition if the abbreviation is used in a subsequent table. For footnotes, use lower-case italicized letters in alphabetical order. Cite each table in the text in consecutive order.

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Computer generated figures are satisfactory for publication but authors should be aware that most figures will be reduced in size and should design their illustrations accordingly. Each figure should be identified by number. Color figures may be published at the discretion of the Editor-in-Chief. Figures should be cited in the text in consecutive order. If a figure has been published, acknowledge the original source and submit written permission from both the author and the publisher to reproduce the material. Define all abbreviations used in each illustration. Repeat definition if the abbreviation is used in a subsequent legend.
Editorial

What is New in the 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care?

It has been 5 years since the last guideline on cardiopulmonary resuscitation (CPR) and emergency cardiovascular care (ECC) was published by the American Heart Association (AHA).1 Over this period, international experts on resuscitation evaluated different research findings and presented their observations in the 2005 International Consensus Conference on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations in Dallas. Their inputs were incorporated into the 2005 guideline for CPR and ECC.2

Since few victims of cardiac arrest received CPR and even fewer received good quality CPR, the aim of the 2005 guideline is to simplify information so that rescuers will learn, remember and deliver proper and early CPR. It is envisaged that this approach will improve survival from cardiac arrest and life-threatening emergencies.

In this article, we wish to draw your attention to the new principles of CPR and ECC. We encourage our fellows and members to read the original articles that are listed in the references.2

Major Changes
Table 1 summarizes the key changes in the latest guideline. In a nutshell, the major changes are:
(1) Effective chest compressions (push hard, push fast) at 100 compressions per minute. The aim is to allow complete chest recoil after each compression, and minimize interruptions in chest compression.
(2) Universal compression-to-ventilation ratio for all lone rescuers. i.e. 30:2 for all victims from infants (excluding newborns) to adults.
(3) Each rescue breath should be given over 1 second to make the chest rise. One should avoid too many, too large or too forceful breaths (Table 2).
(4) Attempt only one shock for each episode of defibrillation, this should be followed by immediate CPR. There should be no intervening rhythm (or pulse) check in between administering a shock and resumption of CPR with chest compression.
(5) The 2005 guideline endorsed the 2003 ILCOR recommendation for the use of automated external defibrillators (AEDs) in children 1-8 years old (and older). The use of a child dose reduction system should be adopted if available.

Other useful information
(1) There are insufficient data to recommend CPR before defibrillation in victims of ventricular fibrillation (VF). Therefore, lay rescuer should use AED as soon as available. Emergency Medical Services (EMS) rescuer may give about 5 cycles (about 2 minutes) of CPR before attempting defibrillation for treatment of out-of-hospital VF or pulseless ventricular tachycardia (VT) when the EMS response (call-to-arrival) interval is > 4-5 minutes or when EMS responders do not witness the arrest.
(2) The latest recommended shock doses for defibrillation are:
Adult: Biphasic truncated exponential waveform: 150-200 J for initial shock, same or higher energy for subsequent shocks
Biphasic rectilinear waveform: 120 J for initial shock, same or higher energy for subsequent shocks
Monophasic waveform: 360 J for initial and subsequent shocks
Children: Same dose for monophasic or biphasic waveform (initial shock - 2 J/kg; subsequent shocks - same or higher energy (2-4 J/kg)
(3) There is lack of documented effect of
vasopressors and antiarrhythmics in improving long-term outcome from cardiac arrest, therefore sequence of CPR de-emphasizes drug administration and re-emphasizes basic life support. Providers should not interrupt compressions to check the rhythm after a shock is delivered until about 5 cycles or 2 minutes of CPR are provided. Check for a pulse by healthcare provider if an organized rhythm is present.

(4) If given, vasopressors or antiarrhythmics should be administered during CPR as soon as possible after rhythm checks; either preceding or following the shock delivery. The most important part of the sequence is to minimize interruptions in chest compressions.

(5) CPR and shock delivery should be coordinated such that it can be delivered as soon as possible after chest compressions with minimal interruption. Studies have shown that a reduction in the interval between compression and shock delivery by as little as 15 seconds can increase the predicted shock success.

Summary
(1) Adult Resuscitation (Figure 1)
Compression to ventilation ratio is 30:2 in 1-rescuer or 2-rescuers CPR (applied to all lay rescuer resuscitation).

(2) Newborn resuscitation
(a) There is no benefit for suctioning of the meconium-stained infant if (s)he is vigorous.
(b) Establishment of effective ventilation is the most important intervention in newborn resuscitation.
(c) There is inadequate data to indicate whether 100% oxygen is superior to room air.

(3) Pediatric resuscitation (Figure 2)
(a) Compression to ventilation ratio for infants and children (up to the onset of puberty) is 30:2 in 1-rescuer or all lay rescuer resuscitation and 15:2 in 2-rescuer CPR.
(b) Emphasis is being placed on the provision of effective compression and ventilation.
(c) Routine use of high-dose epinephrine is not beneficial. On the contrary, this may partially increase the rates of morbidity and mortality.

(4) The Acute Coronary Syndrome Task Force
(a) There is emphasis on first hour of therapy.
(b) The guideline reaffirms the recommendation of out-of-hospital performance and pre-arrival transmission of either 12-lead electrocardiogram or their interpretation to the receiving hospital to reduce time to reperfusion.

(5) The Stroke Task Force
(a) The guideline reaffirms the use of tissue plasminogen activator (tPA) therapy for acute ischemic stroke, adhere vigorously to eligibility criteria.
(b) Dedicated stroke unit and multidisciplinary team improve survival rates, functional outcomes, and quality of life for patients with acute stroke.

(6) The First Aid Task Force evaluates the evidence supporting a number of important first aid therapies:
(a) Use of direct pressure tourniquets for control of hemorrhage
(b) Treatment of ingestion and environmental emergencies.

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Reference
Table 1. Key changes of the 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation (CPR) and Emergency Cardiovascular Care (ECC).

<table>
<thead>
<tr>
<th>2005 Recommendation</th>
<th>2000 Recommendation</th>
<th>Explanation</th>
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<tbody>
<tr>
<td><strong>Basic Life Support</strong></td>
<td></td>
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<tr>
<td>Increased emphasis on delivery of effective chest compressions</td>
<td>Emphasized the first three links in the Chain of Survival: early access, early CPR, and early defibrillation. Stated early CPR significantly improved survival. Named early defibrillation as the single greatest determinant of survival for adult victims of cardiac arrest.</td>
<td>When chest compressions are interrupted, blood flow stops. Limiting interruptions to chest compressions will result in greater survival. In any given series (cycle) of chest compressions, earlier compressions are less effective than later ones. Therefore, fewer interruptions increase the percentage of effective chest compressions. Allowing the chest wall to fully “recoil” or return to its normal position between compressions results in better re-filling of blood in the heart, which allows more blood to be pumped to the rest of the body during the next compression.</td>
</tr>
<tr>
<td>Single CPR compression-to-ventilation ratio 30:2 for all rescuers responding alone to victims of any age, except newborns.</td>
<td>A compression to ventilation ratio of 15 to 2 was recommended for adult CPR; a ratio of 5 to 1 for child and infant CPR.</td>
<td>A single ratio will make learning the correct procedure for responding to victims of all ages easier and increase the likelihood that a rescuer will remember the steps of CPR during an emergency.</td>
</tr>
<tr>
<td>CPR for newborns is the same as 2000 guidelines recommendation.</td>
<td>Three compressions for every one breath should be given to newborns, totaling 90 compressions and 30 breaths per minute.</td>
<td>The new ratio also helps reduce interruptions in chest compressions (see explanation above).</td>
</tr>
<tr>
<td>AED programs should be implemented in public locations where a relatively high likelihood of witnessed cardiac arrest (e.g. airports, casinos, sports facilities and businesses).</td>
<td>Key elements of successful AED programs were recommended, including healthcare provider oversight, training of likely rescuers, link to local EMS system and process of continuous quality improvement.</td>
<td>Some AEDs do not require a medical prescription, so healthcare provider oversight of AED programs is not mandatory.</td>
</tr>
<tr>
<td>A single shock from a defibrillator, followed by immediate CPR for two minutes, beginning with chest compressions, should be used to treat cardiac arrest caused by ventricular fibrillation (VF-the abnormal heart rhythm responsible for most cardiac arrests).</td>
<td>Up to three shocks in a series were recommended to treat cardiac arrest with a “shockable” rhythm before returning to chest compressions; the heart rhythm was evaluated before and after each shock.</td>
<td>Repeated cycles of rhythm analysis and shock result in delays of up to 37 or more seconds before the first post-shock chest compressions are delivered. Most defibrillators eliminate VF more than 85 percent of the time. If the first shock fails, immediate CPR (before trying another shock) is likely to contribute to the success of a subsequent shock. Even when a shock eliminates VF, it may take several minutes for the heart to pump blood effectively, even if a normal heart rhythm returns. A brief period of chest compressions can deliver oxygen to the heart during this post-shock period, increasing the likelihood that the heart will begin to effectively pump blood on its own.</td>
</tr>
<tr>
<td>After giving two rescue breaths, lay rescuers no longer check for signs of circulation before beginning chest compressions.</td>
<td>After giving two rescuer breaths, lay rescuers were instructed to check for signs of circulation (normal breathing, coughing or movement). Lay rescuers gave rescue breathing without chest compressions to victims with signs of circulation who were not breathing normally.</td>
<td>Lay providers cannot reliably detect the presence of circulation in a victim. Great harm can be done when rescuers don’t do chest compressions when they’re needed. Relatively minimal harm can be done by providing chest compressions when they aren’t needed. Therefore, the new guidelines do not recommend that lay rescuers look for “signs of circulation” before delivering chest compressions. This eliminates the chance that lay rescuers might not recognize true cardiac arrest, and reduces delays to chest compressions. Eliminating instructions to look for signs of circulation and for delivering “rescue breathing without chest compressions” reduces the number of skills required for lay rescuers. This makes it more likely that the lay provider will learn and remember the steps of CPR.</td>
</tr>
<tr>
<td>Dispatchers should be trained to recognize the symptoms of Acute Coronary Syndromes (ACS), and advise patients with symptoms of ACS without history of aspirin allergy or gastrointestinal bleeding to chew 160-325 mg of aspirin while awaiting the arrival of EMS providers.</td>
<td>Dispatchers were not instructed to recognize ACS or recommend aspirin.</td>
<td>Early administration of aspirin has been associated with decreased mortality rates in several clinical trials. Many studies have demonstrated the safety of aspirin administration.</td>
</tr>
<tr>
<td>2005 Recommendation</td>
<td>2000 Recommendation</td>
<td>Explanation</td>
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<tr>
<td>Advanced Cardiac Life Support</td>
<td>Heart rhythm analysis, delivery of shocks and selection of drug therapies resulted in frequent interruptions to CPR.</td>
<td>Studies show that providing continuous CPR outweighs the potential effects of drug therapies, so interruptions should be minimized.</td>
</tr>
<tr>
<td>Basic Life Support (BLS) skills are the priority in treating cardiac arrest. Providers must minimize interruptions to chest compressions.</td>
<td>No specific neurologic signs indicated the potential for successful resuscitation.</td>
<td>New research suggests there are specific clinical signs, such as certain brain responses to stimuli that correlate strongly with death or poor brain function following resuscitative efforts. More research is needed to predict potential for survival during resuscitation.</td>
</tr>
<tr>
<td>New neurological tests and evaluations given 24 hours and 72 hours after resuscitation can predict survival to hospital discharge.</td>
<td>Mild hypothermia may be beneficial....but hypothermia should not be induced actively after resuscitation from cardiac arrest. (Position was updated in a 2003 science statement from the International Liaison Committee on Resuscitation, which supported induced hypothermia following resuscitation.)</td>
<td>In two randomized clinical trials, induced hypothermia (cooling within minutes to hours after the return of spontaneous circulation) resulted in improved survival and brain function in adults who remained comatose after initial resuscitation from out of hospital VF cardiac arrest.</td>
</tr>
<tr>
<td>Unconscious adult patients with return of spontaneous circulation after out-of-hospital cardiac arrest should be cooled for 12-24 hours to 32-34 °C when the initial rhythm was ventricular fibrillation. Similar therapy may be beneficial for patients with non-VF arrest out of hospital or for in-hospital arrest.</td>
<td>Administration of tPA was recommended for carefully selected patients with acute ischemic stroke if they had no contraindications to fibrinolytic therapy and if the drug can be administered within 3 hours of the onset of stroke symptoms.</td>
<td>National Institute of Neurological Disorders and Stroke (NINDS) results have been supported by subsequent one year follow up, reanalysis of the NINDS data and a meta analysis. Additional trials supported the NINDS results.</td>
</tr>
<tr>
<td>Tissue plasminogen activator (tPA) is recommended for carefully selected patients with acute ischemic stroke, but cautions that tPA must be administered in the setting of a clearly defined protocol and institutional commitment.</td>
<td></td>
<td>Note: Higher complications of hemorrhage following tPA was reported in one study when participating hospitals did not require strict adherence to NINDS protocols.</td>
</tr>
</tbody>
</table>
Figure 1. Advanced Cardiac Life Support (ACLS) pulseless arrest algorithm.
Figure 2. Pediatric Advanced Life Support (PALS) pulseless arrest algorithm.
**Table 2.** Summary of basic life support - ABCD Maneuvers for Infants, Children, and Adults (*newborn information not included*). This table is originally taken from: 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Part 3: Overview of CPR. *Circulation* 2005;112 [Suppl I]: IV-12-IV-18.

<table>
<thead>
<tr>
<th>Maneuver</th>
<th>Adult Lay rescuer: ≥ 8 years</th>
<th>Child Lay rescuer: 1 to 8 years</th>
<th>Infant Under 1 year of age</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Activate</strong> Emergency Response number (lone rescuer)</td>
<td>Activate when victim found unresponsive HCP: If asphyxial arrest likely, call after 5 cycles (2 minutes) of CPR</td>
<td>Activate after performing 5 cycles of CPR For sudden, witnessed collapse, activate after verifying that victim unresponsive</td>
<td></td>
</tr>
<tr>
<td><strong>Airway</strong> Head tilt–chin lift (HCP: suspected trauma, use jaw thrust)</td>
<td>2 effective breaths at 1 second/breath</td>
<td>2 effective breaths at 1 second/breath</td>
<td></td>
</tr>
<tr>
<td><strong>Breathing</strong> Initial</td>
<td>10 to 12 breaths/min (approximately 1 breath every 5-6 s)</td>
<td>12 to 20 breaths/min (approximately 1 breath every 3-5 s)</td>
<td></td>
</tr>
<tr>
<td>HCP: Rescue breathing without chest compressions</td>
<td>8 to 10 breaths/min (approximately 1 breath every 6 – 8 seconds)</td>
<td>Back slaps and chest thrusts</td>
<td></td>
</tr>
<tr>
<td>Foreign-body airway obstruction</td>
<td>Abdominal thrusts</td>
<td>Brachial or femoral</td>
<td></td>
</tr>
<tr>
<td><strong>Circulation</strong> HCP: Pulse check (≤ 10 sec)</td>
<td>Carotid (HCP can use femoral in child)</td>
<td>Just below nipple line (lower half of sternum)</td>
<td></td>
</tr>
<tr>
<td>Compression landmarks</td>
<td>Lower half of sternum, between nipples</td>
<td>Brachial or femoral</td>
<td></td>
</tr>
<tr>
<td>Compression method Push hard and fast</td>
<td>Heel of one hand, other hand on top</td>
<td>One hand: Heel of one hand or 2 hands as for adults</td>
<td></td>
</tr>
<tr>
<td>Allow complete recoil</td>
<td></td>
<td>2 or 3 fingers HCP (2 rescuers): 2 thumb–encircling hands</td>
<td></td>
</tr>
<tr>
<td>Compression depth</td>
<td>1.5 to 2 inches</td>
<td>Approximately one third to one half the depth of the chest</td>
<td></td>
</tr>
<tr>
<td>Compression rate</td>
<td>Approximately 100/min</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Compression-ventilation ratio</td>
<td>30:2 (one or two rescuers)</td>
<td>30:2 (single rescuer) HCP: 15:2 (2 rescuers)</td>
<td>No recommendation for infants &lt;1 year of age</td>
</tr>
<tr>
<td><strong>Defibrillation</strong> AED</td>
<td>Use adult pads Do not use child pads</td>
<td>Use AED after 5 cycles of CPR (out of hospital). Use pediatric system for child 1 to 8 years if available If child pads / system not available, use adult AED and pads</td>
<td>No recommendation for infants &lt;1 year of age</td>
</tr>
<tr>
<td>HCP: For out-of-hospital response may provide 5 cycles/2 minutes of CPR before shock if response &gt; 4-5 minutes and arrest not witnessed.</td>
<td></td>
<td>HCP: For sudden collapse (out of hospital) or in-hospital arrest use AED as soon as available.</td>
<td></td>
</tr>
</tbody>
</table>

Note: Maneuvers used by only Healthcare Providers are indicated by "HCP."

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Sevoflurane is the leading inhalation anaesthetic agent worldwide. Since its launch in 1994, sevoflurane (SEVOrane®, Ultane®) has been utilized as an anesthetic agent in more than 190,000,000 patients globally. As a result of this extensive use, Abbott has accumulated a large amount of experience regarding the clinical performance of sevoflurane and in related issues that may affect its clinical use. One such area relates to product stability during manufacturing, transportation, storage, and use. A description of two examples of the results of this extensive experience follows.

In late 1996, a limited number of bottles of sevoflurane were reported to have a cloudy appearance and a non-characteristic pungent odor when initially opened. The drug in these bottles was tracked to a specific manufacturing lot of sevoflurane. On investigation, this lot of product was noted to have a level of acidity outside of Abbott’s acceptable specification range and contained hydrogen fluoride (HF) which is highly corrosive to skin and mucous membranes, and with respiratory exposure can result in life-threatening respiratory toxicity. In some cases, the glass bottles then used to package the product had deteriorated. Abbott immediately removed all affected product from the marketplace. No patient injury was reported.

Investigation by Abbott researchers revealed that several materials may react with sevoflurane, resulting in a degradation profile matching that found in the affected bottles. These materials included aluminum oxide, iron oxide and other species known as Lewis acids. Figure 1 shows the Lewis acid degradation reaction, resulting in the production of HF, among other degradants.

Figure 1. Lewis acid degradation reaction.

Further research was conducted to better characterize the previously unrecognized reaction of sevoflurane with the ubiquitous Lewis acids, and to find a method to prevent its reoccurrence. As noted above, Abbott investigators determined that one source of Lewis acids is aluminum oxide, a natural component of glass. In the circumstance where sevoflurane is packaged in a glass container, if the sevoflurane Lewis acid degradation process has already been initiated by Lewis acids in the environment, the HF generated may proceed to attack the glass surface, potentially exposing additional Lewis acids in the glass (aluminum oxide) to the sevoflurane product, further...
fueling the process. Abbott refers to this process as glass attack (Figure 2).

After extensive investigation, Abbott’s researchers found that the addition of a sufficient amount of water was effective in inhibiting the Lewis acid degradation process, thus enhancing the stability of sevoflurane. Among several Lewis acid inhibitors, water was selected as the stabilizing agent of choice because in the concentration selected, it is effective, biocompatible, stable, and does not affect sevoflurane’s clinical performance. By preferentially combining with Lewis acids with which sevoflurane may come in contact during storage and use, the added water renders these substances non-reactive in sevoflurane. Figure 3 illustrates this process.

The discovery of the stabilizing effect of water was considered scientifically important and novel and resulted in the issuing of sevoflurane

Figure 3. Excess of water molecules completely blocks the Lewis acid reaction cascade.
formulation patents worldwide. All sevoflurane product manufactured and sold by Abbott today contains a carefully controlled quantity of water within the product formulation.

In conjunction with the investigation to characterize and prevent the Lewis acid degradation process, Abbott also worked to change the packaging of sevoflurane from a glass bottle to a lighter, less breakable container. Finding a suitable plastic for the new sevoflurane container required considerable investigation. The selected material had to be resistant to chemical reaction with sevoflurane, not permit leaching and release of impurities into the sevoflurane product, provide a vapor barrier to transpiration of sevoflurane, and also not be a source of Lewis acids. Abbott researchers found a suitable material for this purpose in the plastic Polyethylene Naphthalate (PEN)\(^4\). Therefore, Abbott has converted sevoflurane’s container from glass bottles to PEN bottles across most of the world.

The purpose of this article is to highlight improvements Abbott has made in the formulation and packaging of SEVOrane\(^5\)/Ultane\(^6\). Abbott will continue to monitor the performance of the product throughout the world.

References

Cuffed Oropharyngeal Airway in Basic Airway Management

A Prospective Randomized Clinical Trial

1Bassanio LAW, 2PT CHUI, 3SK NG, 4David C CHUNG
Department of Anaesthesia and Intensive Care, Prince of Wales Hospital, Shatin, Hong Kong

SUMMARY

Adequate ventilation is essential during cardiopulmonary resuscitation. Tracheal intubation, although considered to be the gold standard for airway management, it is skill dependent and is not suitable for those without adequate training. A new airway device, the cuffed oropharyngeal airway (COPA), may offer an alternative for the use in basic airway management prior to the arrival of skilled personnel in the emergency situation.

In this study, we compared the effectiveness of COPA, facemask alone and facemask with Guedel airway in ventilating 60 anesthetized patients. As untrained subjects, 60 medical students were recruited to ventilate 60 patients with the 3 devices for 12 breaths per device. The time to first recordable breath, peak inspiratory pressure (PIP), end-tidal carbon dioxide concentration (ETCO₂), inspired and expired tidal volume and the leak volume were measured.

The time required to establish the first breath was the longest with COPA. This was associated with an increase in ETCO₂ but lower PIP. The number of successful ventilations was fewer than facemask with Guedel airway. Facemask alone resulted in shorter time to establish the first breath but the number of successful breaths was the least and the leak volume was the highest. Facemask with Guedel airway achieved the highest number of successful breaths together with the lowest leak volume.

We concluded that facemask with appropriate sized Guedel airway offered the best option for the lesser-trained. COPA cannot be recommended as the first choice in basic airway management.

Keywords: Cardiopulmonary resuscitation; Airway management; Facemask; Guedel airway; Cuffed oropharyngeal Airway.
of the tongue anteriorly. This forms an air tight seal with the posterior pharynx and elevates the epiglottis from the posterior pharyngeal wall to provide a clear airway.5

In a group of 100 spontaneously breathing anaesthetized patients, Brimacombe and Berry showed that the COPA provided a clear airway in 98% of the patients and had a low complication rate.6 The insertion of COPA is easy and does not required long period of training. Gerard et al investigated the use of COPA to ventilate training resuscitation manikins in a group of lesser-trained hospital staff (stretcher-bearer, nurse and midwife).7 There was a higher success rate in effective ventilation using COPA than facemask.

We hypothesized that COPA may improve airway patency and assist in manual ventilation by lesser-trained personnel during basic airway management. In this prospective, randomized clinical study we compared the effectiveness of manual ventilation through COPA, with facemask alone, or facemask with Guedel airway by lesser-trained personnel (medical students) in anaesthetized apneic patients.

Materials and Methods
This study was approved by the Clinical Research Ethics Committee of the Chinese University of Hong Kong. We recruited sixty patients of American Society of Anesthesiologists physical status class 1 or 2 Mallampati class 1 or 2, age between 18 to 65 years. All patients were scheduled for elective surgery requiring general anesthesia, tracheal intubation and muscle relaxant. We excluded patients who were obese with a body mass index > 30. Patients who were pregnant or patients with a history of gastroesophageal reflux, or having loose teeth, crowns, or dental prostheses were also excluded.

Sixty final year medical students, during their 4 weeks’ attachment to the anesthesia module, were enrolled to represent lesser-trained personnel. The medical students had received practical instructions on basic airway skills using airway manikins, including the triple airway maneuver, manual ventilation using facemask with and without Guedel airway, but not with COPA.

All patients were seen preoperatively and a written informed consent obtained. Premedication was not prescribed. On arrival in the operating theatre, intravenous access was secured. Monitoring included heart rate, noninvasive arterial pressure and electrocardiography, inspired oxygen concentration, oxygen saturation, end-tidal carbon dioxide concentration (ETCO2). Peak inspiratory airway pressure (PIP), inspired (VTi) and expired (VTe) tidal volumes were measured by side stream spirometry (Datex AS3, Helsinki Finland). The investigators chose the appropriate size Guedel airway and COPA for each patient.

In the presence of two specialist anesthetists, patients were preoxygenated for 3 minutes prior to induction of anesthesia with intravenous fentanyl 1 µg/kg, propofol 1-2 mg/kg and vecuronium 0.08-0.1 mg/kg. Anesthesia was maintained by propofol infusion at a rate of 10 mg/kg/h. Following induction of anesthesia, a specialist anesthetist ventilated the patient for 3 minutes to allow the onset of neuromuscular blockade as monitored by nerve stimulator. Each medical student was then assigned to one of the 60 fasting anaesthetized apneic patients to ventilate the patient’s lung with a self-inflating bag with COPA alone, facemask alone and facemask with Guedel airway. The order of airway devices was randomized by pre-coded envelopes. Oxygen was delivered to the reservoir bag of the Laerdal manual resuscitator (Laerdal Medical Corporation, Norway) at 8 L/min. During manual ventilation with each airway device, respiratory parameters, including VTi, VTe, ETCO2, and PIP, were recorded over 10 consecutive manual breaths. The first 2 breaths were considered as warm-up breaths. If the medical student failed to insert the Guedel airway or the COPA in 30 seconds, the investigator would take over insertion of the airway device. No assistance was given to the medical students during the manual ventilation.
We decided to discontinue the study if patient’s pulse oximetry oxygen saturation fell below 94%. The time taken by the medical student from the start of inserting the airway device to the delivery of a first recordable breath (time to first breath) was noted.

Because the participating medical student might not be able to ventilate the patient with a correct technique or maintain an effective seal for each of the 10 consecutive breaths, therefore not every attempted breath was recordable e.g. the monitor would record the absence of inspired and expired tidal volume, ETCO₂, PIP etc. when there was an ineffective seal. Therefore, we defined the following outcome variables:

1. an attempted breath is any breath attempted by the medical student whether or not there is data recorded on the monitor;
2. a failed breath is an attempted breath without generation of recorded data;
3. a recordable breath is an attempted breath with any recorded data;
4. a successful breath is a breath with recorded VTₑ > 400 ml according to the International Liaison Committee on Resuscitation.

Based on these data, we calculated the following for each of the airway devices:

1. the average PIP of all the recordable breaths;
2. the average ETCO₂ of all the recordable breaths;
3. the average VTᵢ of all the recordable breaths;
4. the average VTₑ of all the recordable breaths;
5. the average leak volume (i.e. VTᵢ-VTₑ) of all the recordable breaths;
6. the number of recordable breaths out of the total number of attempted breaths (n = 600);
7. the number of successful breaths out of the total number of attempted breaths.

Subsequently, general anesthesia was continued according to the preference of the individual anesthetist.

**Statistics**

Continuous data are presented as mean ± standard deviation (SD). Categorical data are presented as counts (percentage). Continuous data between stages were compared using one-way analysis of variance with repeated measures, followed by Bonferroni Correction for post hoc comparison. Categorical data between airway devices were compared using χ² test. The Statistical Package of Social Science (SPSS; version 9.0, Chicago, IL) was used for data analysis. P values less than 0.05 was considered as statistically significant.

**Results**

Sixty final year medical students were enrolled to ventilate one of the 60 recruited patients (10 males; 50 female) for the study. The mean (± SD) age and weight of the patients were 44.1 ± 13.4 years and 60.8 ± 11.8 kg, respectively. The patients suffered no complication during the study.

The results are summarized in Table 2. There are statistically significant differences between the 3 airway devices regarding the time to establish the first breath, PIP, ETCO₂, VTᵢ, VTₑ, the leak volume as well as number of recordable and successful breaths. Post hoc comparisons showed the following findings:

1. COPA had the longest time to establish the first breath, whereas facemask alone had the shortest time to establish first breath.
2. Facemask alone had higher PIP than COPA and facemask with Guedel airway;
3. COPA had highest ETCO₂ compared with facemask alone or facemask with Guedel airway;
4. Facemask alone had highest VTᵢ compared with COPA or facemask with Guedel airway;
5. COPA had lowest VTₑ compared with facemask alone or facemask with Guedel airway;
6. facemask alone had highest leak volume compared with COPA or facemask with Guedel airway.

Moreover, facemask with Guedel airway had the highest number of recordable and successful breaths while facemask alone had the lowest number of recordable and successful breaths.
Discussion

Tracheal intubation remains the “gold standard” for airway management in cardiopulmonary resuscitation (CPR) because it protects the trachea from aspiration which neither COPA nor oral Guedel airway could offer. However, tracheal intubation requires the presence of skilled personnel, e.g. the anesthetist, who may not be immediately available.\(^9\)\(^{11}\) Attempts at intubation by lesser-trained personnel are far less likely to be successful than insertion of COPA or oral Guedel airway. Most lesser-trained personnel involved in CPR would use facemask alone or facemask with Guedel airway as the first method to achieve ventilation of the patients.

In this study, we have demonstrated that the use of facemask alone is associated with the shortest time to establish the first breath but resulted in the highest leak volume and the lowest number of recordable and successful breaths. This observation implies that although the facemask is easy to apply, it is highly skill dependent and is unreliable with inexperienced personnel. It is also difficult to maintain an effective seal. Although we hypothesized that COPA is a better alternative airway device, our results showed otherwise. The use of COPA is associated with the longest time to establish the first breath, achieving fewer effective breaths compared with facemask and Guedel airway. One advantage of COPA is that its use is associated with the lowest peak inspiratory pressure. This may be associated with a lower chance of inadvertent gastric insufflation. The inability to generate high airway pressure may be related to the intrinsic design of the device. The use of COPA to ventilate patients with low lung compliance or high airway resistance may be inadvisable, as high airway pressure cannot be generated.

The use of facemask with Guedel airway appears to be the best technique to provide the greatest number of recordable and successful breaths (tidal volume > 400 ml). The time to establish the first breath is not excessively prolonged compared with facemask alone. Moreover, the average PIP required to produce

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**Table 1.** Respiratory parameters during airway management with cuffed oropharyngeal airway (COPA), facemask alone and facemask with Guedel airway.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>COPA</th>
<th>Facemask</th>
<th>Facemask with Guedel airway</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time to first breath (sec)</td>
<td>21.3 ± 15.2</td>
<td>9.7 ± 5.3</td>
<td>12.5 ± 4.7</td>
<td>(P &lt; 0.001^a)</td>
</tr>
<tr>
<td>Average peak inspiratory pressure (cmH(_2)O)</td>
<td>15.7 ± 4.4</td>
<td>18.7 ± 6.5</td>
<td>16.3 ± 4.8</td>
<td>(P &lt; 0.001^b)</td>
</tr>
<tr>
<td>Average end-tidal carbon dioxide concentration (kPa)</td>
<td>4.6 ± 0.6</td>
<td>4.2 ± 0.8</td>
<td>4.3 ± 0.6</td>
<td>(P &lt; 0.001^c)</td>
</tr>
<tr>
<td>Average inspiratory tidal volume (ml)</td>
<td>780 ± 278</td>
<td>880 ± 246</td>
<td>814 ± 223</td>
<td>(P = 0.002^d)</td>
</tr>
<tr>
<td>Average expiratory tidal volume (ml)</td>
<td>641 ± 164</td>
<td>699 ± 174</td>
<td>685 ± 154</td>
<td>(P = 0.011^e)</td>
</tr>
<tr>
<td>Average leak volume (ml)</td>
<td>138 ± 145</td>
<td>182 ± 158</td>
<td>130 ± 141</td>
<td>(P = 0.003^f)</td>
</tr>
<tr>
<td>Number of recordable breaths (total 600 breaths)</td>
<td>504/600</td>
<td>467/600</td>
<td>548/600</td>
<td>(P &lt; 0.001^g)</td>
</tr>
<tr>
<td>Number of successful breaths (total 600 breaths)</td>
<td>474/600</td>
<td>455/600</td>
<td>533/600</td>
<td>(P &lt; 0.001^h)</td>
</tr>
</tbody>
</table>

* One-way repeated-measures analysis of variance; \(^{*}\chi^2\) test

\(^a\) COPA vs Facemask: \(P < 0.01\); COPA vs Guedel airway: \(P < 0.01\); Facemask vs Guedel airway: \(P = 0.39\).

\(^b\) COPA vs Facemask: \(P < 0.01\); COPA vs Guedel airway: \(P = 0.27\); Facemask vs Guedel airway: \(P < 0.01\).

\(^c\) COPA vs Facemask: \(P < 0.01\); COPA vs Guedel airway: \(P < 0.01\); Facemask vs Guedel airway: \(P = 0.02\).

\(^d\) COPA vs Facemask: \(P < 0.01\); COPA vs Guedel airway: \(P = 0.53\); Facemask vs Guedel airway: \(P < 0.01\).

\(^e\) COPA vs Facemask: \(P = 0.01\); COPA vs Guedel airway: \(P = 0.05\); Facemask vs Guedel airway: \(P = 0.175\).

\(^f\) COPA vs Facemask: \(P = 0.02\); COPA vs Guedel airway: \(P = 0.35\); Facemask vs Guedel airway: \(P = 0.002\).
successful breaths is lower than the facemask alone. This observation is not surprising as the Guedel airway opens the upper airway to relieve partial or complete obstruction that frequently present in unconscious patients. Therefore, the presence of a Guedel airway may decrease the upper airway resistance leading to easy ventilation of the lungs at lower airway pressure than with facemask alone.

We acknowledge that our study is not without limitations. Firstly, medical students may not be good representatives for the lesser-trained personnel involving in CPR. In out-of-hospital cardiac arrest, the first responder is frequently an ambulance officer whereas in in-hospital cardiac arrest, the ward nurse is usually the first person providing resuscitation. However, medical students represent an easily available group of lesser-trained personnel who are expected to provide CPR in the ward once after their graduation.

Secondly, the use of fasted anesthetized apneic patients in a highly controlled environment may not mimic the real stressful situation of resuscitating a patient with cardiac arrest in the general ward environment.

Thirdly, most of the students, during their attachment to the anesthetic module, have limited experience with the use of facemask and/or Guedel airway for ventilation; none has any exposure to COPA. This “prior” experience may explain the higher success rate of using facemask with Guedel airway in comparison to COPA.

Finally, the monitor was unable to record all the data of the attempted breaths for practical reasons such as excessive leakage with ineffective seal. This may make the overall calculation less accurate. However, this is not surprising to us because the students we recruited are inexperienced in basic airway skill.

In summary, we have demonstrated that the use of facemask with Guedel airway is associated with greatest number of recordable breaths and successful breaths, followed by the use of COPA, while the use of facemask alone is associated with least number of recordable and successful breaths. This suggests that, of the three options, facemask with Guedel airway is the most effective way to ventilate an apneic patient by lesser-trained personnel. We recommend the use of facemask with an appropriate sized oral Guedel airway as the best option for lesser-trained personnel to ventilate apneic patients. The use of COPA cannot be recommended as the first choice in basic airway management.

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Co-administration of Phenylephrine to Prevent Systemic Hypotension During Induction of Anesthesia With Propofol

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SUMMARY

Hypotension is a well known complication of propofol administration. In a double-blind, randomized, controlled study, we studied the efficacy of using phenylephrine, an α-agonist, in preventing propofol induced hypotension. Sixty elderly (≥ 55 years) patients were randomly allocated to receive either phenylephrine 0.1 mg, 0.2 mg, 0.3 mg or saline immediately following propofol i.v. 2 mg/kg. Hemodynamic parameters were recorded every minute for 5 minutes.

In the saline group, systolic arterial pressure decreased markedly by 26% from the baseline at the first minute and continued until the fourth minute, representing a total of 32% decrease from the baseline ($P = 0.0001$). All the phenylephrine study groups prevented a decrease in arterial pressure. Phenylephrine 0.2 mg and 0.3 mg increased arterial pressure in first 1-2 minutes (maximum 15%) but did not reach statistical significance. There was a decease in heart rate in all groups ($P = 0.0001$). These results showed that prophylactic use of phenylephrine prevent a decrease in blood pressure following propofol injection in elderly patients. The smallest dose 0.1 mg among the three doses is the optimal one achieving such effect in this study.

Keywords: Intravenous anesthetic, Propofol; Complication, Hypotension; Vasopressor, Phenylephrine; Elderly

Propofol is a widely used intravenous induction agent for general anesthesia. However, it is associated with significant hemodynamic changes, especially a decrease in arterial pressure. The reduction in arterial pressure, although rarely important for young and healthy individuals, can be detrimental to the elderly patients. These patients are more sensitive to propofol and hypotension may adversely affect tissue perfusion and oxygenation leading to myocardial and cerebral ischemia.

A number of methods have been suggested in the literature to prevent hypotension associated with propofol. These include fluid preload, prophylactic use of ephedrine, atropine or glycopyrrolate or metaraminol, although the results were conflicting. Phenylephrine is an α1 agonist and potent peripheral vasoconstrictor. It has been widely used for treating hypotension.

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of various causes with incremental doses of 0.1 - 0.2 mg every 5 to 10 minutes or infusion. However, its role in preventing propofol-induced hypo-tension has not been studied.

The aim of this study was to evaluate the efficacy of phenylephrine 0.1, 0.2 or 0.3 mg given immediately following propofol induction in the prevention of hypotension caused by propofol in patients ≥ 55 years.

Methods

After approval by hospital ethics committee, sixty patients of American Society of Anesthesiologists physical status class I or II, aged 55 years or older, scheduled for elective surgery during general anesthesia were recruited into the study. Written consent for the study was obtained before surgery. Patients with history of uncontrolled hypertension, ischemic heart disease, congestive heart failure, cerebrovascular disease, thyroid disease or pre-operative baseline systolic arterial pressure ≥ 200 mmHg or diastolic blood pressure ≥ 100 mmHg were excluded. Patients were randomized into four groups: (1) phenylephrine 0.1 mg, (2) 0.2 mg, (3) 0.3 mg groups or (4) placebo group using sealed envelopes. Phenylephrine doses (study drug) were diluted in 5 ml saline and placebo consisted of 5 ml saline alone. An anesthetic medical officer, not involved in the study, prepared the drugs in identical syringes.

Premedication was not prescribed. Upon arrival in the operating theatre, baseline arterial pressure and heart rate were measured. A 20 gauge intravenous cannula was then inserted into the dorsum of the hand under local anesthetics and patients were pre-oxygenated for 3 minutes. Thereafter, patients were induced with a bolus of propofol 2.0 mg/kg. Subsequently study drug or placebo bolus was given. Further increments of propofol 0.5 mg/kg was given if necessary until loss of consciousness with loss of eyelash reflexes.

All patients breathed a mixture of nitrous oxide 66% in oxygen, and respiration was assisted gently by facemask for apneic patients for 5 minutes after induction of anesthesia. The study period was defined as 5 minutes following induction or there were signs of recovery from anesthesia such as eye or limb movements. Electrocardiogram and pulse oximetry were monitored continuously. Arterial pressure and heart rate were measured by oscillotonometry every minute after induction for 5 minutes. Rescue medication with phenylephrine 0.1 mg was given immediately if there was a single systolic arterial pressure reading ≤ 80 mmHg and atropine 0.4 mg was given if heart rate ≤ 40 beats/min. Following the study period, anesthesia was continued as required for the operation. No intravenous fluid was given before or during the study period.

This study aimed to assess whether a bolus dose of phenylephrine would prevent a significant decrease in arterial pressure (20%) associated with propofol induction. The secondary aim was to determine the minimum dose required among the three study groups. Based on a 20% change from mean (± standard deviation, SD) systolic pressure of 143 ± 23 mmHg, 15 patients in each group were required at an α value of 0.05 and power of 90%. Population characteristics were compared among groups using one-way ANOVA. Hemodynamic data was analyzed using factorial ANOVA with repeated measures. Differences were considered statistically significant when \( P < 0.05 \).

Results

Patient characteristics were similar among groups. (Table 1). Induction of anesthesia was associated with a decrease in systolic arterial pressure in the saline group \( (P = 0.0001) \). The marked decrease in mean arterial pressure (-26% from baseline) occurred in first minute after induction and such effect continued until the fourth minute (-32% drop from baseline, Figure 1). Statistical analysis revealed significant difference between saline group and other groups \( (P = 0.0001) \), yet there was no significant difference among the three study groups \( (P = 0.07) \). There was no
significant decrease in arterial pressure in three phenylephrine study groups. There was an increase in arterial pressure after phenylephrine 0.2 and 0.3 mg in first two minutes (Figure 1). The highest mean arterial pressure was 175 mmHg in patients receiving phenylephrine 0.3 mg (i.e. 15% increase from baseline).

Heart rate decreased significantly in all four groups after induction ($P = 0.001$, Figure 2). There was no significant difference among groups ($P = 0.89$). The decrease of heart rate ranged between 0 to 51% from baseline, however only one patient in phenylephrine 0.3 mg group needed atropine 0.4 mg for bradycardia (37 beat/min at first minute post-induction). Two patients in saline group needed phenylephrine 0.1 mg as rescue for hypotension.

None of the patients had electrocardiographic abnormality. There was no oxygen desaturation during the study period. No patient suffered from neurologic deficit was noted after recovery from anesthesia.

**Discussion**

Our study confirms that induction of anesthesia with propofol is associated with significant arterial hypotension in patients older than 55 years. Phenylephrine in doses of 0.1, 0.2 or 0.3 mg administered immediately after propofol induction can prevent such a decrease in systolic arterial pressure.

Phenylephrine is an $\alpha_1$ agonist and potent peripheral vasoconstrictor commonly used in our daily practice of anesthesia. As decrease in systemic vascular resistance accompanied by vasodilation is thought to be the predominant mechanism causing propofol induced hypotension,$^{1,2}$ a potent vasoconstrictor such as phenylephrine may prevent such an effect. Furthermore, as a direct $\alpha_1$ agonist, phenylephrine has immediate onset of action and short duration of effect - a pharmacokinetic profile that matches well with propofol induced hypotension. We chose three study doses of phenylephrine based on manufacturer’s recommendation which suggests 0.1 - 0.5 mg bolus as slow injection. Although bolus doses of phenylephrine 0.2 or 0.3 mg may be considered as higher than that usually used, we believe such doses are still safe in selected patients. We used propofol as the sole induction agent without premedication or opioid to avoid additional effects on hemodynamic stability.

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**Table 1.** Patient characteristics, hemodynamic measurements and total dose of propofol used in patients receiving saline or phenylephrine after induction of anesthesia.

<table>
<thead>
<tr>
<th></th>
<th>Saline</th>
<th>Phenylephrine</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.1mg</td>
<td>0.2mg</td>
</tr>
<tr>
<td>Number of patients</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>Age (years)</td>
<td>61±5.5</td>
<td>65±7.3</td>
</tr>
<tr>
<td>Sex (M/F)</td>
<td>4/11</td>
<td>6/9</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>59.9±8.9</td>
<td>63.2±9.5</td>
</tr>
<tr>
<td>American Society of Anesthesiologists physical status class (I/II)</td>
<td>5/10</td>
<td>6/9</td>
</tr>
<tr>
<td>Propofol dose (mg)</td>
<td>120±18</td>
<td>126±19</td>
</tr>
<tr>
<td>Baseline systolic arterial pressure (mmHg)</td>
<td>144±15</td>
<td>149±18</td>
</tr>
<tr>
<td>Baseline mean arterial pressure (mmHg)</td>
<td>108±14</td>
<td>107±13</td>
</tr>
<tr>
<td>Baseline heart rate (beat/min)</td>
<td>74±16</td>
<td>78±11</td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation.
Figure 1. Changes in systolic arterial pressure from baseline to 5 min after induction of anesthesia. Point estimates are mean and the error bar refers to standard deviation.

![Graph showing changes in systolic arterial pressure](image1)

Figure 2. Changes in heart rate from baseline to 5 min after induction of anesthesia. Point estimates are mean and the error bar refers to standard deviation.

![Graph showing changes in heart rate](image2)
patients and complicating the interpretation of the results.

Non-invasive blood pressure measurements at one minute intervals were thought to be sufficiently accurate to detect the changes in systolic pressure although individual readings have been shown to have 10% variability. \(^1\) Invasive arterial pressure monitoring would give us a continuous reading with greater accuracy, but it was not thought to be ethically justifiable for this study.

Different mechanisms have been proposed to explain propofol induced hypotension. Some studies have shown a significant decrease in vascular resistance with little change in cardiac output,\(^1,12\) while others suggested that propofol causes a reduction in both cardiac output and vascular resistance.\(^2,13-15\) Metaraminol and phenylephrine are both potent vasopressors. Although a recent study using metaraminol 0.5 mg did not show an effect on attenuating propofol induced hypotension in patients older than 55 years,\(^3\) we have been able to demonstrate that phenylephrine at dose of 0.1 mg or above given immediately following propofol can prevent hypotension in similar group of patients. Chiu et al proposed that in older patients, reduction in cardiac output might be the predominant mechanism making metaraminol an ineffective choice as prophylactic vasopressor.\(^3\) Based on our study results, however, we propose that the predominant mechanism of propofol induced hypotension is reduced vascular resistance rather than reduced cardiac output. Our results are consistent with Claeyss et al who showed that arterial hypotension associated with propofol induction was mainly due to decreased afterload without compensatory increase in heart rate or cardiac output.\(^1\) Turner et al also showed that fluid preload was ineffective in preventing propofol induced hypotension in young healthy patients, and thus proposed that decreased vascular resistance was the mechanism of hypotension instead of reduced cardiac output.\(^3\)

We observed that there was an increase in systolic arterial pressure in the initial two minutes in the phenylephrine 0.2 and 0.3 mg groups, but it did not reach statistical significance. Goertz et al showed that a bolus of phenylephrine 3 µg/kg did not impair left ventricular function or increase end-systolic left ventricular wall stress when it restored blood pressure and cardiac filling after postural hypotension induced by isoflurane anesthesia in head-up tilt position.\(^3\)

We also observed a significantly reduction in heart rate among all study groups \((P = 0.0001)\). This was likely due to central vagotonic action of propofol and nitrous oxide and oxygen inhalational mixture.\(^1,17\) Phenylephrine, in contrast, did not produce additional bradycardia in this study, although one patient in 0.3 mg group required atropine rescue. Propofol induced reduction in heart rate and myocardial depression are associated with a significant decrease in cardiac oxygen consumption and global myocardial oxygen supply and demand ratio is therefore preserved.\(^18,19\) Despite this, arterial hypotension may occasionally lead to local myocardial or cerebral ischemia in area supplied by a stenotic artery.\(^18\) Thus there is a need to prevent hypotension with propofol induction.

Our study used a high power of 90% to detect a 29 mmHg difference in systolic arterial pressure among treatment groups. Therefore, phenylephrine at dose of 0.1 mg can be considered an effective choice in preventing propofol induced hypotension in patients older than 55.

In conclusion, the concomitant use of phenylephrine at propofol induction is able to prevent hypotension in patients older than 55 years and the ideal dose is 0.1 mg.

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Pre-oxygenation Using Circle Absorber

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SUMMARY

The authors determined the optimal fresh oxygen flow rate for preoxygenation using the circle system. We also evaluated whether flushing the circle system with oxygen will affect the rate of preoxygenation.

Our data showed that fresh gas flow rate is a significant factor in determining the rate of successful preoxygenation; the higher the oxygen flow rate, the quicker and more reliable is preoxygenation. Also flushing the circle breathing system with oxygen was shown to make preoxygenation faster. However, the effect of increased flow rate disappeared when the rate exceeded 6 L/min with the system flushed with oxygen and 8 L/min with the system flushed with air. We also showed that there was a risk of breathing hypoxic gas mixture when using a low fresh gas flow rate especially when the breathing system had not been flushed with oxygen. This may have an adverse effect on patient during induction of anesthesia. Despite having apparently leak-proof facemasks, some subjects still failed to be preoxygenated by the normally recommended method.

We would propose that it is most economical and appropriate to preoxygenate a patient with oxygen flow rate of 8 L/min for 5 minutes without necessarily prior flushing of the breathing system with oxygen.

Keywords: Preoxygenation, Fresh gas flow rate, End-tidal oxygen concentration

Pre-oxygenation is a routine practice in modern-day anesthesia.1,2 By breathing 100% oxygen, the nitrogen in the lungs is replaced with oxygen, and potentially increases the oxygen available for body use from 450 to 3,000 ml during apnea.3 This reduces the incidence of hypoxia during induction of anesthesia. A number of factors may affect the effectiveness of preoxygenation. These include the duration of preoxygenation, respiratory pattern, choice of anesthetic breathing system, technique of applying the facemask. Studies on preoxygenation had been conducted using various breathing systems4-6 and breathing patterns,7,8 and different methods had been used for assessing endpoints.9-10 Despite all these, textbooks recommendations on methods of preoxygenation varies, with the duration of preoxygenation ranges between 2 to 10 minutes, using various anesthetic breathing systems and oxygen flow rates.1,12-14

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The circle breathing system is one of the most economical and commonly used anesthetic breathing circuits in modern day anesthesia. It would be logical to think that higher fresh gas flow rate is more effective in preoxygenation, as oxygen displaces the nitrogen out of the system. It thus prevents rebreathing of nitrogen and expedites preoxygenation. Nevertheless, when the oxygen delivery exceeds the patient’s respiratory requirement and the volume required to washout the nitrogen in the breathing system, the excess fresh oxygen is vented via the scavenging systems and is therefore wasted. Also, prior to the start of preoxygenation, the circle system may contain gaseous mixture of variable composition. This volume of gas may affect the inspired oxygen concentration during preoxygenation. Hamilton and Eastwood investigated “denitrogenation” with volunteers breathing using a circle system with oxygen flow rate of 1-5 and 10 L/min and showed that denitrogenation to < 5% of expired nitrogen concentration occurred within 3 minutes at an oxygen flow rate of 4 L/min. But the study was performed under laboratory conditions in an unspecified manner with unknown number of volunteers.

The objectives of the present study are firstly to determine the optimal fresh gas flow rate of oxygen for preoxygenation using the circle circuit in a clinical setting and secondly to determined if flushing the circuit with oxygen will affect the rate of preoxygenation.

Methods
After obtaining approval from the local ethics committee, twenty healthy volunteers were studied with informed consent. Their demographic details including age, sex, height and weight were recorded. None of them had ever smoked. The study was carried out in the operating theatre suite. The facemasks (Anatomical Facepieces, BOC of sizes 2, 3, 4 and 5) were used for preoxygenation. These masks were chosen such that no leak was detectable when applied to subjects’ face with them exhaling using maximal force. At the time of preoxygenation, the baseline heart rate, systolic and diastolic arterial pressure, body temperature and their arterial oxygen saturation were recorded. The inspired and expired concentration of oxygen and carbon dioxide were measured continuously using a photoacoustic and magnetoacoustic gas monitor (Type 1304, Brüel and Kjær, Nærum, Denmark). The circle breathing system was checked to make sure that it was functioning properly and leak-free, and the soda lime was not exhausted. The Narkomed 2 anesthesia machine, the circle breathing system and the 2-litre reservoir bag were flushed through with oxygen to ensure delivery of 100% oxygen, as judged by the inspired oxygen analyzer. The pressure relief valve of the circle circuit was fully open and the gas sampling tubing for the gas analyzer was placed as close to the volunteer's mouth as possible. The volunteer was asked to lie supine and breathe normally via the facemask for 5 minutes. The fresh gas flow was set at rates of 2, 4, 6, 8 and 10 L/min of oxygen and showed that denitrogenation to < 5% of expired nitrogen concentration occurred within 3 minutes at an oxygen flow rate of 4 L/min. But the study was performed under laboratory conditions in an unspecified manner with unknown number of volunteers.

Statistics
The association between fresh gas flow rate, the type of gas used for flushing, and the success in pre-oxygenation at 3 minutes and 5 minutes after commencement of preoxygenation were
analyzed with logistic regression using a marginal model. The mathematical approach has been discussed by Diggle et al. The calculations were performed using generalized estimating equation in S-PLUS 4.5 (StatSci Division, Seattle, WA).
Results
The median (inter-quartile range, IQR) weight, height and body mass index were 53.4 (50-65.8) kg, 165 (158.5-170) cm and 21.3 (18.8-23.0) kg/m², respectively.

The changes of end-tidal oxygen concentration with different fresh gas flow rate and flushing media are shown in Figure 1. When using the “normal” preoxygenation technique, ten of the volunteers did not achieve successful preoxygenation at 3 minutes despite an oxygen flow rate of 6 L/min and flushing with oxygen. Nevertheless, nine out of these ten volunteers were successfully preoxygenated at 5 minutes.

Marginal model analysis revealed that success in preoxygenation at 3 minutes is significantly associated with an increase in fresh gas flow rate, odds ratio (95% confidence interval, CI) 1.6 (1.4 - 1.8), \( P < 0.0001 \) and changing the flushing medium from air to oxygen, odds ratio 2.8 (1.6 - 4.9) \( P < 0.0001 \), Figure 3). Body mass index was however not a significant factor (\( P = 0.20 \)). Similarly, at 5 minutes, increase in fresh gas flow rate and changing the flushing medium from air to oxygen increased the likelihood of successful preoxygenation, \( P < 0.0001 \) and \( < 0.0001 \), respectively.

Discussion
Machlin et al suggested that end-tidal oxygen fraction should be used to determine the success of preoxygenation.\(^9\) This method was chosen in our study because it is non-invasive and directly measures the changes of the oxygen content in the lungs. End-tidal oxygen tension \( \geq 630 \) mmHg was defined as successful preoxygenation. To our knowledge, there has only been one study reported on the effect of changing the oxygen supply flow rate on preoxygenation. In 1955, Hamilton et al showed that volunteers achieved 95% denitrogenation within 3 minutes when breathing via a circle breathing system with fresh gas flow of 4 L/min.\(^4\) Nevertheless, the methodology used was not clearly defined. In addition, the study was conducted in laboratory conditions where the facemasks were “tightly strapped .” This technique however is not applicable in daily clinical situations. The number of volunteers in the quoted study was not specified and besides, the numbers of trials for some preoxygenation settings were different.

**Figure 3.** Success of preoxygenation at different fresh flow rate using a circle breathing system flushed with air (squares) or 100% oxygen (circles) at 3 (open symbols) or 5 minutes (filled symbols).
Our study showed that fresh gas flow rate is a significant factor in determining the speed of successful preoxygenation; the higher the oxygen flow rate, the quicker and more reliable is preoxygenation. Also flushing the breathing system with 100% oxygen expedites preoxygenation. However, the effect of increased flow rate disappears when the rate exceeds 6 L/min with the breathing system flushed with oxygen and 8 L/min with breathing system flushed with air.

Even after 5 minutes of breathing 100% oxygen, there is still one subject not preoxygenated by the preset criteria. Because of this, we were unable to recommend a fresh gas flow rate which will ensure successful preoxygenation. Despite that, we would propose that it is most economical and appropriate to preoxygenate with oxygen flow rate of 8 L/min for 5 minutes without the need of prior flushing the circle breathing system with oxygen.

There is a definite risk of breathing hypoxic gas mixture if fresh gas flow is set at a low rate (< 4 L/min) when breathing through the circle breathing system, especially if it has not been flushed with 100% oxygen. This is because nitrogen recirculates and accumulates in the breathing system. The effect can actually adversely affect the safety of preoxygenation, which could become dangerous when overlooked.

Nearly all volunteers found it difficult to breathe through the circle breathing system especially with a low flow rate. They tended to hyperventilate despite being asked to breathe normally. This may explain why many patients do not tolerate tight-fitting facemasks, leading to gas leak and thus failure of preoxygenation. Other than mask leak, denitrogenation is also affected by factors such as relationship between functional residual capacity and closing volume, where air pockets (containing nitrogen) are trapped in lungs. These factors delayed denitrogenation.

Success of preoxygenation may also be influenced by factors affecting oxygen uptake from alveolar (e.g. lung diseases, hemoglobin level and cardiac output), or oxygen utilization (e.g. body temperature and activity, disease status, pregnancy and age). Further studies are required to delineate the possible relationship between preoxygenation and functional residual capacity and other factors as described above.

In summary, we should beware that traditionally taught method of preoxygenation may not be successful in all patients and it is pertinent to preoxygenate, especially high risk patients, with a higher fresh gas flow rate and for longer period to ensure adequate preoxygenation.

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Shunting After Release of Aortic Cross-Clamp During Coronary Artery Bypass Graft Surgery:

Is Lung Ventilation Necessary Before Separation From Bypass?

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SUMMARY

The ventilatory practice after release of aortic cross-clamp during coronary artery bypass graft (CABG) surgery differs among anesthesiologists. In this study, we evaluated the safety and efficacy of withholding lung ventilation until completion of proximal aortic anastomosis.

Sixteen patients scheduled for CABG surgery requiring cardiopulmonary bypass (CBP) were studied. Lung ventilation was stopped after commencement of CBP and was resumed following final suture of proximal graft anastomosis. Blood gases were measured from the patients and heart lung machine before and 10 minutes after release of aortic cross-clamp, and during suturing of the final proximal anastomosis. The calculated shunt fraction was less than 5% at all sampling time points ($P = 0.91$). The degree of shunt after release of aortic cross-clamp without lung ventilation is minimal and did not differ from the baseline. Oxygen saturation and tension measured from the patients’ blood and the arterial circuit of the heart lung machine was identical.

Keywords: Coronary artery bypass graft surgery, Shunt, Oxygen tension, Cardiopulmonary bypass

During coronary artery bypass graft (CABG) surgery, the classic teaching is to resume lung ventilation after release of aortic cross-clamp. The rationale is to minimize the potential intrapulmonary shunt once as the patient’s heart retributes to the forward blood flow.

However lung ventilation may disturb the surgical field and thus increase the surgical difficulty in fashioning proximal graft anastomoses. Therefore, ventilatory practices after release of aortic cross-clamp differ among anesthesiologists.

In this study, we evaluated the safety and efficacy of withholding lung ventilation until completion of the final proximal anastomosis.
We measured the shunt fraction during proximal anastomosis with CBP.

Methods

The study was approved by the institutional review board of the Hospital Authority, Hong Kong West Cluster. Written informed consent was obtained from all patients. Patients were eligible for the study if they were scheduled for CABG requiring cardiopulmonary bypass (CBP). Patients were excluded if they have evidence of cardiac valve dysfunction, left ventricular ejection fraction < 50%, or history of pulmonary disease with resting oxygen saturation < 95%.

All patients received midazolam (7.5-15 mg) or lorazepam (0.5-1.0 mg) orally, one hour before the scheduled operation. Upon arrival in the operating room, routine monitoring including arterial, and central venous catheter were inserted. Electrocardiogram leads II and V5, pulse oximetry, rectal and nasopharyngeal temperature probes, and urinary catheter were also placed.

Nitroglycerin was started at a rate of 0.5 µg/kg/min after central venous cannulation. Fentanyl, etomidate, rocuronium or pancuronium were given during induction of anesthesia. Anesthesia was then maintained with isoflurane before CBP, propofol infusion and additional doses of fentanyl was given during CBP. The total dose of fentanyl was 15-20 µg/kg.

After tracheal intubation, the lungs were ventilated with intermittent positive pressure ventilation (Aestiva 5, Datex-Ohmeda/Servo 900C, Siemens) using 40% oxygen in air. Tidal volume and respiratory rate were adjusted to maintain an arterial carbon dioxide tension of 35-45 mmHg. Positive end expiratory pressure of 4 cmH2O was also applied.

Heparin 3 mg/kg was administrated before CBP. An activated clotting time (ACT) was maintained > 480 s during CBP with additional doses of heparin as required. CBP was established with a Sarns 8000 heart lung machine, using roller pump and membrane oxygenator. A hard shell open venous reservoir was used. Monitoring consisted of arterial pressure, reservoir level, mixed venous oxygen saturation, blood temperature. Flow rate was set at 2.4 L/m². Radial artery pressure was maintained at 50-85 mmHg, and vasoactive drug were given as required. CABG was performed in routine fashion using saphenous vein, radial and/or left internal mammary arterial grafts.

Lung ventilation was stopped and continuous positive airway pressure (CPAP), 4 cmH2O, was applied during CBP and it was withheld until completion of proximal anastomosis. Serial blood samples were collected from the pump arterial and venous lines. Patient’s arterial blood was also sampled. Arterial and venous blood was analyzed by a blood gas machine (pHOx plus, Nova Biomedical) immediately before aortic cross-clamp release, 10 minutes after aortic cross-clamp release and during suturing of the final proximal anastomosis.

 Oxygen content in the blood samples were calculated from the oxygen carrying capacity equation:

\[
CO_2 = 1.34 \times Hb \times SpO_2 + 0.003 \times PO_2
\]

Where
- \(CCO_2\) = oxygen content (ml/dl)
- \(Hb\) = hemoglobin concentration (g/dl)
- \(SpO_2\) = oxygen saturation (%)
- \(PO_2\) = oxygen tension (mmHg)

We then calculated the shunt fraction by the following modified shunt equation:

\[
\frac{Q_{sp}}{Q_t} = \frac{CcO_2 - CaO_2}{CcO_2 - CvO_2} \times 100\%
\]

Where
- \(Q_{sp}\) = shunt flow (L/min)
- \(Q_t\) = total forward flow (L/min)
- \(CcO_2\) = end-capillary (or pump arterial line) oxygen content
- \(CaO_2\) = Arterial oxygen content
- \(CvO_2\) = mixed venous (pump venous line) oxygen content
Data were presented as means ± standard deviation (SD). Changes in shunt fractions were analyzed by one-way analysis of variance. A P value less than 0.05 was considered as statistically significant.

We estimated the sample size by assuming a 50% change in shunt fraction. Based on a mean shunt fraction of 2.0 and a standard deviation of 1.4, we calculated that 14 subjects were required to achieve 80% power at an α value of 0.05.

**Results**

A total of 16 patients (11 male and 5 female) were recruited. The median (range) age and body mass index were 65 (29-72) years and 24.5 (19.0-29.7) kg/m², respectively. On average, each patient had three grafts. The median (range) duration of CBP and aortic cross-clamp were 105 (67-141) min and 60 (38-97) min, respectively. The time period spent when ventilation was withheld during proximal anastomosis was 27 (16-41) min.

The oxygen saturation in the pump arterial line and the patient’s systemic blood at all sampling time points were identical. The lowest oxygen saturation and oxygen tension measured from the patient’s systemic blood were above 98% and 180 mmHg respectively.

The shunt fraction before and 10 minutes after release of aortic cross-clamp were 2.1 ± 1.4 and 2.0 ± 1.4, respectively. Shunt fraction remained unchanged during proximal aortic anastomoses, 1.9 ± 1.4. One-way analysis of variance showed no difference between the calculated shunt measured at all three different sampling time points, P = 0.91.

**Discussion**

Several studies have evaluated the various methods of lung management during CBP, in order to minimize post-CBP pulmonary dysfunction. However, none has proved useful. Current suggestions are to encourage CPAP but avoid 100% oxygen or lung ventilation.

Pizov et al showed that administration of 100% oxygen during bypass may lead to absorption atelectasis and oxygen toxicity. Berry et al showed that the difference between alveolar and arterial oxygen partial pressure was lower during the first 30 minutes of CPB but was increased afterward. Loeckinger et al also showed that the application of CPAP during bypass significantly improved pulmonary gas exchange after surgery. In common to Berry’s findings, the reduced difference in alveolar-arterial oxygen partial pressure was not persistent in the postoperative period and did not influence the postoperative pulmonary morbidity. Stanley et al estimated the postoperative shunt fraction and lung compliance in animals ventilated with different methods during CPB. They found that lung ventilation during CPB might actually increase the shunt fraction and decrease the lung compliance. Chancy et al reported similar findings in human.

Although the ideal mode of lung ventilation during CBP in preventing postoperative pulmonary dysfunction remains controversial, little research has focused on the study of lung management after aortic cross-clamp release. Classic teaching is to resume lung ventilation after release of aortic cross-clamp and weaning of CPB. The rationale is to minimize the potential shunting effect as the patient’s heart re-contributes to the forward blood flow. Our study showed that shunting was minimal during this period and carried no clinical or statistical significance.

A previous study showed significant decreases in systemic oxygen tension in non-ventilated patients. However, this study was conducted at the end of CBP with all the coronary graft anastomoses completed. The venous return to the heart was artificially increased with partial occlusion of the extracorporeal venous circuit. Therefore, there was significant forward blood flow through the pulmonary vasculature, allowing some filling of
the cardiac chambers and subsequent systemic and pulmonary ejection.

Our study was conducted during the routine surgical procedure and there was no occlusion of venous line until separation from bypass. As the heart was not ejecting, the resulting cardiac output during the study with such a small preload became negligible. Therefore, it did not result in shunting.

Although comparison with a similar group of patients undergoing CABG, but utilizing a strategy of lung ventilation would have been ideal, our main purpose in this study was to demonstrate the efficacy and safety of an alternative strategy, we feel that the study has satisfactorily fulfill our aim.

This was a small, simple and prospective descriptive study aiming to show the safety and efficacy of the non-ventilation strategy. Although we did not investigate the relationship between the level of surgical difficulty during proximal graft anastomosis and the mode of ventilation, resumption of lung ventilation after aortic cross-clamp release often disturbs the surgical field, creating increased difficulty in suturing of the proximal anastomoses.8

We concluded that the strategy of non-ventilation is a safe and efficacious technique for ventilatory management after aortic cross-clamp release in CABG surgery.

References


Periprostatic Local Anesthetic Block for Prostatic Biopsy
A Prospective Controlled Study

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SUMMARY

A prospective randomized trial was performed to evaluate the efficacy of periprostatic block with 2% lignocaine and adrenaline 1:200,000. A total of 99 patients undergoing transrectal ultrasound (TRUS) guided prostatic biopsy as day care only procedures were studied. The mean pain scores during the procedure were improved in the local anesthetic group compared with placebo. However, the statistical difference disappeared two hours after injection. There was no difference in the incidence of surgical complication between groups.

Keywords: Transurethral ultrasound guided Prostatic biopsy, Local anesthetics, Pain

Prostate cancers are diagnosed by transrectal ultrasound (TRUS) guided biopsy. Originally a transperineal needle puncture was used after local anesthetic infiltration. But even then it was a painful procedure. With the development of transrectal needle insertion in the late 1980’s the use of local anesthetic was discontinued because it was thought that rectal wall puncture and transrectal biopsies were painless. However, significant proportion of patients reports discomfort or pain. This may have arose from puncture of the prostatic capsule and the stroma. Nevertheless, some patients also find the introduction and maintenance of the ultrasound probe within the rectum uncomfortable. Previous studies have evaluated the role of local anesthesia. The rectal administration of lignocaine gel has no impact on the tolerance to prostatic biopsy. Other reports suggested that periprostatic block using plain lignocaine did not improve pain relief. In the latter studies, there were no difference in the amount of hematuria, hematochezia, hematospermia or infection rate between groups with or without the use of local anesthetics. Adrenaline may cause vasoconstriction and less postoperative hemorrhage. By using lignocaine with adrenaline, 2% instead of plain 1% lignocaine
can be used without increasing the risk of toxicity.

We prospectively evaluated the safety and efficacy of periprostatic local anesthesia using 10 ml 2% lignocaine with adrenaline 1:200,000 during prostatic biopsy guided by TRUS.

Methods

Patients referred to surgical department for TRUS guided prostatic biopsy in day surgery ward over a 3-month period were recruited for the study. All patients gave written informed consents. Patients were excluded if there was a history of allergy to lignocaine or adrenaline. Patients were randomized to receive the same volume of either local anesthetics or normal saline. The solution for injection was prepared by the anesthetist. The patient, nurse assistant and urologist were unaware of the treatment allocation.

All patients were examined in left lateral decubitus position. The TRUS probe was fitted with the needle guide and repositioned in the rectum. Study drug was injected by the urologist using a long 22G spinal needle through the needle guide. The tip of the needle was advanced into the right of the gland apex. If there was resistance to injection when the needle was within prostatic tissue, the needle was slowly withdrawn under direct vision and once immediately outside the prostate capsule with the needle in Denonviller’s fascia, the resistance dissipated and 5 ml of study drug was injected easily. The solution separated and spreaded along the tissue planes of Denonviller’s fascia, bathing the entire posterior surface of the gland, from the apex to the seminal vesicles. A second injection of 5 ml was then given at the same site on the left side of the gland. Biopsy was then taken immediately.

Patients were assessed by nurse assistant for any symptoms of local anesthetic toxicity, especially, circumoral numbness and tinnitus. After completion of biopsy, patients were asked to score the severity of pain (1) in middle of biopsy and (2) at the end of biopsy using a visual analogue scale (0-100). Same question was asked 2 hours later and upon discharge home. On next day, patients were contacted again by nurse through telephone. They were asked about pain and satisfaction score by verbal analogue scale (0-10) and surgical complication including hematuria, hematospermia, hematochezia, retention of urine and fever. Finally, patients were asked whether they agreed for a second biopsy using the same anesthetic technique.

The following surgical complications were defined:
(1) Hematuria - blood in urine
(2) Hematospermia - blood in sperm
(3) Retention of urine - cannot pass urine
(4) Fever - temperature > 38 °C

Continuous data were tested by unpaired $t$ test and category data were analyzed by $\chi^2$ test. Data were mean (95% confidence intervals, CI). A $P$ value < 0.05 was considered significant.

Results

One hundred patients were recruited at the start of study. One patient was excluded due to

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technical problems after recruitment. A total of 50 patients received local anesthetics and 49 received saline. The age and weight between groups were similar (Table 1). The number of patients who could not be contacted on day 1 was not significantly different between groups. There were three patients in the saline group requiring admission to the hospital.

The occurrence of surgical complications, such as retention of urine, hematuria, hematospermia, hematochezia and fever) on day 1 was similar between groups (Table 2).

The mean (95% CI) pain scores in patients receiving local anesthetics during the middle of biopsy, 24.7 (18.8-30.6) was lower than that in the placebo group, 36.2 (28.0-44.5), $P = 0.02$. Similarly, there was lower pain score at the end of biopsy, $P = 0.01$.

The mean pain scores two hours after the procedure and upon discharge were also less in local anesthetic group although they were not statistically significant. Pain score on day 1 were similar between groups. Patient satisfaction score were also similar (Table 3). No patients suffered from allergy or local anesthetic toxicity.

**Discussion**

A significant portion of patients does not tolerate TRUS guided biopsy. This will discourage suspicious patients from repeated biopsy. Moreover, many patients suffered from surgical complications. Rectal anesthetic gel was ineffective and the value of an analgesic suppository has not been systematically evaluated.

In our study, the pain scores in the middle and at the end of the procedure were both lower in local anesthetic group. However the patient satisfaction was similar between groups. This may reflect that the pain relief was inadequate and the duration of action was not sufficiently long enough despite adrenaline was added.
No patient had signs or symptoms of local anesthetic toxicity. The toxic limit can be exceeded if concentrated lignocaine is used. This limit can be increased by the addition of adrenaline. Our results show that given “proper” doses of lignocaine, periprostatic block may work. One possible explanation of failure of local anesthetic instillation in previous studies was that 1% lignocaine is not enough when deposited in the vascular periprostatic capsule. Our data however, suggested even 2% lignocaine with adrenaline does not produce a very long duration of analgesia.

The bleeding complications (hematuria, hematochezia, hematospermia) were similar in both groups. The vasoconstriction by adrenaline may be insufficient to decrease bleeding. One other explanation may lie in the anatomy of the prostatic blood vessels. It is postulated that the rigid prostatic stroma may hold these vessels open, thus making them less able to constrict appropriately. Incidences of urinary retention and fever were similar in both groups.

The method of periprostatic infiltration was developed empirically by considering prostatic anatomy. The gland receives sympathetic and parasympathetic nerve supplies to both the glandular and stromal elements. The nerves join the neurovascular bundles, which pass along the posterolateral margins of the gland between the capsule and Denovillier’s fascia, and pierce the capsule, particularly at the base and apex in the 4 and 8 o’clock position. Thus, local anesthetic introduced at these points will anesthetize the whole gland.

In conclusion, periprostatic local anesthetic improves the quality of pain control, however, the duration of analgesic effect need to be longer to provide better patient satisfaction. Further study may be needed for another choice of local anesthetic.

References

A Case Report of Being Awake at Bispectral Index of 40

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SUMMARY

Monitoring level of consciousness in patients requiring general anesthesia has been gaining popularity. BIS values below 60 are suggested to correlate with adequate hypnosis. We report a case of a patient being awake and obeyed commands at BIS of 40 in the immediate postoperative period.

Keywords: Bispectral index; Hypnosis; Awareness; Monitoring; Fentanyl

Intraoperative awareness is one of the undesirable outcomes that anesthetists aim to avoid during general anesthesia. Awareness is identified as having explicit memory (a conscious recollection of previous experiences) and/or implicit memory (changes in performance or behavior that are produced by previous experience). Postoperatively, a structured interview is required to reveal explicit awareness (recall test or recognition test), whereas detection of implicit recall needs psychological testing. It is possible that stimuli delivered to an unconscious, adequately anesthetized patient may, in some way, be retained in memory. Patients with intra-operative awareness encountered multiple problems like pain, sensation of weakness or paralysis, feelings of helplessness, anxiety, panic and impending death. Some patients may also experience sleep disturbance, nightmare, daytime anxiety, fear of surgery and post-traumatic stress disorder. Recent estimates of the incidence of recall of awareness range from 0.16-0.29% in general surgery. Autonomic responses to light anesthesia and pain such as tachycardia, sweating, lacrimation, skin conductance, pupil dilation have been used as indicators of awareness. Others methods used to monitor awareness include isolated forearm technique, lower esophageal motility. With the introduction of bispectral index (BIS, Aspect Medical Systems, Newtown, MA) monitoring, anesthetists have an additional tool to assess the level of sedation and hypnosis. It is suggested that this may result in better titration of anesthesia and minimize awareness during general anesthesia. BIS is a dimensionless variable of 0 - 100 that correlates with the degree of sedation and hypnosis. BIS values of 40-60...
are proposed to correlate with an adequate depth of anesthesia with low probability of awareness. There were two large scale studies on BIS to prevent unintentional awareness. The B-Aware trial is a double blind randomized controlled trial. It examined high awareness risk patients, including patients undergoing cardiac surgery, trauma surgery, rigid bronchoscopy, and cesarean section. In this study, BIS-guided anesthesia reduced the risk of awareness by 82%.

The SAFE-2 trial compared the incidence of awareness with a retrospective cohort of patients showing that the use of BIS monitoring reduced the incidence of awareness by 78%.

However, the reliability of these monitoring in different anesthetic applications needs to be justified. We reported a patient responding to verbal commands at a BIS value of 40.

Case report

A 70-year-old female with known history of myocardial infarction and a ventricular aneurysm was scheduled for laparoscopic right hemicolectomy for carcinoma of the transverse colon. She was taking aspirin, nitrates and metoprolol. Pre-operative investigation showed a hemoglobin concentration of 99 g/L. Before induction, her arterial pressure and heart rate were 152/85 mmHg and 55 beats/min, respectively. She was given fentanyl 100 µg and etomidate 4 mg at induction of anesthesia. Rocuronium 30 mg was administrated to facilitate tracheal intubation. Standard monitoring (including electrocardio-graphy, non-invasive blood pressure, pulse oximetry and temperature) was applied. Arterial and central venous catheters were inserted in view of the potential fluctuation of arterial pressure. Initially, general anesthesia was maintained with 0.5% sevoflurane. The lungs were ventilated with intermittently positive-pressure ventilation with a mixture of 50% oxygen and 50% nitrous oxide. After a temporary rise in blood pressure during intubation, the systolic arterial pressure decreased gradually to < 90 mmHg at 15 minutes after tracheal intubation.

In order to avoid myocardial depression, sevoflurane and nitrous oxide were switched off. Anesthesia was then maintained by midazolam infusion 1 mg/h, fentanyl infusion 100 µg/h. An S/5 Datex-Ohmeda M-BIS module with BIS quarto XP sensor was used to monitor anesthetic drug effect. The BIS monitor was set with a scale of 100 µV, a smoothing rate of 15 sec and filters were applied. Nitroglycerin infusion was started for the control of arterial pressure (20 to 200 µg/min).

Intraoperatively, her heart rate was stable and BIS numbers were kept between 25 and 45. BIS number was solid during surgery. Electromyographic signals were within the range of 24-38 dB. Operation lasted for three hours and blood loss was about 200 ml. Body temperature was between 36.5-37° C and further boluses of fentanyl 100 µg i.v. were given for episodes of hypertension.

After three hours operation, BIS numbers remained < 50 and the patient was transferred to the recovery room. Midazolam and fentanyl infusion were ceased, but nitroglycerin and cisatracurium infusion were continued. BIS varied between 30 and 40. Two hours after the operation, cisatracurium was stopped. Half hour later, the patient started to breathe spontaneously and was able to respond to verbal command (opening eyes and protruding tongue). The BIS number was 40 and started to increase to 63 about 15 minutes later (Figure 1). Patient was re-sedated with midazolam 1 mg and fentanyl 150 µg and then she was sent to intensive care unit. The trachea was extubated the next day. When she was asked about any recall of the operation and the period of being awake in the recovery room, she could not remember these events. During the intra-operative and postoperative period in the recovery room, there were no tachycardia, no lacrimation, no sweating or dilated pupils.

Discussion

Awareness during general anesthesia is a horrifying and distressing experience. It may
occur with or without conscious recall. Even without conscious recall, awareness in the intraoperative period may act as a psychic irritant and it may cause post-traumatic stress disorder in the patient.\textsuperscript{12} The relationship between consciousness and memory formation during general anesthesia is complex and is incompletely understood. Our patient was fully awake, opened eyes and protruded her tongue, in response to verbal command at a BIS value of 40. This value is usually considered to have a low probability of awareness. Although the patient had no conscious recall of the events in the operating and recovery room, the BIS values below 45 in the intraoperative period probably did not signify unconsciousness in our patient. The amnesic effect of midazolam is well documented and this may be the reason for the lack of conscious recall.\textsuperscript{13}

Using narcotic based anesthesia with or without volatile anesthetic agent increase the risk of awareness. Detection of awareness by clinical signs and hemodynamic changes is known to be unreliable especially when muscle relaxant is used.\textsuperscript{2,14} In our patient, the only clinical sign which might suggest awareness, was the episodes of hypertension. Nevertheless, this was difficult to interpret.

The BIS monitor has been approved by the Federal Drug Administration in USA as an aid to monitor the anesthetic effects.\textsuperscript{16} Its usefulness in the detection of awareness is controversial. Many advantages have been proposed for the use of BIS monitor. Titration of the depth of anesthesia according to BIS has been found to facilitate emergence and recovery from general anesthesia.\textsuperscript{18} These findings have been associated with cost savings with less consumption of anesthetic agents and recovery room resources.\textsuperscript{19}

Nevertheless, the usefulness of BIS in monitoring the depth of anesthesia has been
challenged. BIS is an empiric, statistically derived measurement of electroencephalographic (EEG) variables. It assumed that these EEG features will correlate with hypnosis irrespective of the anesthetic agents used. The mechanism for the state of anesthesia is however complex. It will be an over-simplification if one assumes that all anesthetic agents have similar mechanism of action. The accuracy of BIS in the correlation with unconsciousness may also be drug specific. Unconsciousness and BIS values have no correlation when ketamine is used. There is no change in BIS value when increasing the concentration of nitrous oxide administration until the loss of response to verbal command in the patient. Barr et al studied BIS and propofol induced hypnosis in volunteers. They found that in general, BIS decreased with increasing sedation but there were large individual variations so that it could not reliably differentiate the state of being awake and unconscious during propofol infusion in individual patients. One subject was awake at BIS of 51.

The usefulness of BIS in measuring the depth of anesthesia in narcotic based anesthesia as in our patient is not encouraging. Barr et al studied BIS in fentanyl and midazolam anesthesia for coronary bypass surgery. BIS did not correlate with midazolam and fentanyl drug concentrations. During clinically adequate anesthesia, BIS varied from 36-91 in different patients. They concluded that BIS was not a measure of the depth of anesthesia in this drug combination. Barr et al also studied the effects on the BIS during medium to high doses of fentanyl induction. When fentanyl alone was used at induction, patients lost their responses to verbal command at BIS of 45-94. One patient was awake at BIS value of 43. They concluded that BIS did not correlate with the hypnotic effect of fentanyl. The detection of awareness is more difficult when muscle relaxants are used. The sign of inadequate anesthesia such as an increase in respiratory rate and somatic movement are lost. The usefulness of BIS, in these situations is doubtful. Measner et al reported that the BIS of a volunteer declined from 98 to 33 and 9, respectively in two occasions when muscle relaxant alone was administered. A case of explicit intraoperative recall under general anesthesia using nitrous oxide, oxygen and sevoflurane in cardiac surgery at BIS of 47 had been reported.

In conclusion, we reported a case of being awake and obeying command at a BIS value of 40 for a patient in the immediate postoperative period. This adds to the evidence that the BIS value of less than 60, the general accepted criterion for adequate anesthesia, does not guarantee unconsciousness. The suggestion of using BIS in guiding the dose of general anesthetic agents given in order to reduce the expenditure and the side effects of the anesthetic agents as well as speeding the recovery from anesthesia needs to be cautioned.

References


28. Mychaskiw G II, Horowitz M, Sachdev V,


30. Doli M, Gajraj RJ, Mantzaridis H, Kenny GN.


**Congratulations!**

Professor Peter Kam, FHKCA (Hon) was appointed as the Nuffield Professor of Anaesthetics, The University of Sydney, Chairman and Head, Department of Anaesthetics, Royal Prince Alfred Hospital.
Subspecialties Training Survey

After the implementation of the new VTG, the Board of Education would like to review the progress of the College’s trainees so far. A survey for various subspecialties training of our trainees under the new Vocational Training Guide (VTG) will be conducted. All anesthesia trainees whose vocational training starts after 1 January 2002 will be asked to participate in the survey. The information obtained will have an important reference for our future training requirements. All trainees attending the Exit Assessment on or after 1st January 2008 will be assessed based on the new VTG.

Please completed the survey forms and send them to Dr CH Koo, Training Officer of the Hong Kong College of Anaesthesiologists, Department of Anaesthesiology, Queen Elizabeth Hospital, 1/F, Block D, 30 Gascoigne Road, Kowloon before 31 March, 2006.

Review on continuous medical education/continuing professional development (CME/CPD) by the Hong Kong Academy of Medicine:

It was agreed in principle by the Education Committee of the Hong Kong Academy of Medicine that; (awaiting the formal endorsement by the Council of the Academy):

1. The number of CME/CPD points required for Fellows of different specialties and subspecialties should be the same, and a Fellow accredited for more than one specialty/subspecialty within the same College should not be required to obtain more than 90 points.

2. Some specialty or subspecialty-specific CME/CPD activities should be allowed to suit the needs of different Colleges.

3. CME/CPD requirements for subspecialties should not be made harder to achieve than that for the specialties. Mandatory elements should be applicable across all specialties/subspecialties within the same College.

4. Starting from 1 January 2008, all Colleges would set a capping for passive CME, so that Fellows would have to do other activities which may include CPD. The capping should be at 75 points or below.

5. Starting from 1 January 2011, mandatory CPD would take place.

6. Flexibility would be allowed for individual Colleges should they want to take a faster pace in implementing mandatory CPD.

The College would like to provide fellows with flexible and wider choices of learning and to facilitate our Fellows in gaining CPD points. The following activities are defined by our College as CPD activities:

- **Self-study**
  - Remarks: College approved self study programmes and/or self assessment programmes.

- **Active Participation**
  - Remarks: speaker, chairman, panelist or presenter in formally College approved activity

- **Publications**
  - Remarks: in peer reviewed journals approved by the College

- **Research**
  - Remarks: with publication in peer reviewed journals approved by the College

- **Development of new technologies or services**

- **Postgraduate teaching: approved by the College**

- **Conducting examinations**

- **Quality assurance and medical audits**
  - Clinical/surgical review and audit
  - Clinical governance
  - Activities that examine and evaluate the clinical care of patients

- **Mortality and morbidity meetings**
  - Postgraduate courses: approved by the College

- **Development of CME/CPD materials**

- **Activities for Improvement of Patient Care**
  - Information technology training
  - Interpersonal and communication skill training
  - Skills laboratory learning
  - Virtual reality learning
  - Grand rounds in training units

Dr YF Chow
Chairman
Board of Education
Board of Pain Medicine

The Diploma of Pain Management (HKCA) is now almost 10 years old. Over 30 trainees have joined the training programme since then, and almost the same number has sat for the examination. While the training for Dip Pain Mgt has matured over time, the pain medicine has also progressed enormously in all parts of the world. In view of the changing status of pain medicine in Hong Kong and elsewhere, the Board of Pain Medicine felt that it is time to review the Diploma of Pain Management training programme including the curriculum and examination. A sub-committee was formed recently to look into this and in the next month or so, the committee will make its recommendation to the Board and then to Council.

The next Dip Pain Mgt examination will be held on 27 October 2006. Our external examiner this time will be Dr Beverly Collett, Consultant Pain Specialist at the Leicester Royal Infirmary, England. Dr Collett is also the current President of the Pain Society of Great Britain and will be a guest faculty at the Annual Scientific Meeting 2006 in November.

The Board would also like to announce that Dr Kong Suet Kei is the new Supervisor of Training for Dip Pain Mgt at the Pamela Youde Nethersole Hospital.

Dr PP Chen
Chairman
Board of Pain Medicine

Board of Intensive Care Medicine

Final Fellowship Examinations in Intensive Care

The Board of Intensive Care is pleased to announce that wherever possible two examinations in Intensive Care Medicine will be held each year.

Please consult College for the closing dates for application for examinations.

The dates for the examinations for 2006 are:

(1) Written paper 2nd May 2006
    Clinical and Oral 10th June 2006
(2) Written paper 3rd October 2006
    Clinical and Oral 11th November 2006

Dr Peggy Tan
Board of Intensive Care Medicine
Approved Formal Projects

CHU, Ka Lai  The EXIT procedure for tracheal intubation in a fetus with cystic hygroma.
NG, Yuen Chong  Agreement between plasma ionized calcium and albumin-adjusted calcium concentrations in critically ill patients.
SIN, Leonard  Prophylactic anti-emetic therapy after laparoscopic cholecystectomy: Comparative study of tropisetron, dexamethasone and a combination of these two drugs.
WONG, Ian  Effect of prophylactic subcutaneous single injection of naloxone on prevention of intrathecal morphine-induced pruritus in post caesarean patients.
FUNG, Nga Yin  A comparison between sevoflurane/remifentanil and propofol/remifentanil anaesthesia in providing conditions for somatosensory evoked potential monitoring during scoliosis corrective surgery.
TONG, Kam Chiu  Revision of right total hip arthroplasty in a case of ankylosing spondylitis.
FONG, Siu Yan  A randomized study in comparing levobupivacaine versus racemic bupivacaine in epidural analgesia for labor pain.

The Formal Project Prize was established by the College Council. This Prize is awarded to the best paper presented at the Formal Project Prize Session, usually held as part of the College Annual Scientific Meeting (ASM)* or any other meeting approved by the Council. Registered Trainees in Anesthesia, Intensive Care or Pain Medicine and Fellows within one year of award of the corresponding Diploma of Fellowship are invited to submit the abstract of their formal projects to the organizing committee of the ASM for consideration of the award. Projects that have previously been published as a full manuscript or have been presented in another local or overseas meeting will also be considered. However, projects that have previously entered in another Formal Project Prize competition will be excluded.

The Chairman of the Board of Education will appoint at least two judges to select a number of projects for presentation during the “Formal Project Prize Session” at the ASM. The criteria for selection will be based on the scientific content of the submitted abstracts. The final assessment for the award will also include the quality of performance during the presentation and discussion afterwards.

The College reserves the right not to award the Prize if none of the project achieves a sufficiently high standard.

*The ASM 2006 “From the Heart and Beyond” will be held in the Convention and Exhibition Center on 18-19 November, 2006. Please visit the conference web page, http://www.hkca.edu.hk/asm2006.htm, for further details.
The Council has resolved on the admission for Fellow “ad eundem” 2006. The same applies to application for both the specialty of Anaesthesiology and the sub-speciality of Intensive Care Medicine.

A. Introduction:
The regulations regarding admission of Fellow ad eundem as governed by the By-laws of the Hong Kong College of Anaesthesiologists stipulates that:

- 2.3.5 Election of candidates as Fellow ad eundem shall be by the Council.
- 2.3.6 Such election shall be held at the meeting of the Council prior to the Annual General Meeting in each year but so that the number of Fellows ad eundem so admitted in each year shall not exceed a number to be determined each year by the Council; such election shall be by ballot and to be admitted a candidate shall receive the favorable vote of three quarters of the number of the members of the Council present.

HKCA recognizes the importance and contribution of anaesthesiologists possessing overseas anaesthesia qualifications who have decided to come and work in Hong Kong. On the other hand, HKCA has to address the issue of supply and demand of specialist anaesthesiologists. At the present moment, HKCA is able to produce enough specialist anaesthesiologists to satisfy the needs of Hong Kong people. It would be reasonable to set a quota for the number of “Fellows ad eundem” to be admitted each year.

B. After the Annual General Meeting of 2005, the procedure for election of “Fellow ad eundem” will follow strictly by-law 2.3.6 and

B.1 Not more than 5 will be admitted at such occasion.
B.2 Each application will be vetted according to the spirit laid down in By-law 2.3.2.7, which stipulates that the applicant should have demonstrated a contribution to the advancement of the pursuits of the College in practice, education or research.
B.3 If there are more than 5 applications, the most deserving ones, according to By-law 2.3.2.7 will be admitted. The total number of admissions may be less than 5.
B.4 Unsuccessful applicants may appeal to the Appeals Committee against the decision of the Board of Censors, or they may apply again in future when their contributions may have increased over time.

C. Procedures for application
C.1 The deadline for the annual application for admission as “Fellow ad eundem” will be announced 6 months in advance.
C.2 Applicants should complete the Fellowship Application Form and send it to the College secretariat together with supporting documents before the prescribed deadline.
C.3 All applications will be considered together.
C.4 Short listed applicants will be invited to attend an “Exit Assessment”.
C.5 Successful applicants will be informed in due course.

The College would like to invite applications for Fellowship “ad eundem” 2006.

1. All applications are to be received by the published deadline of 13 April 2006. (Late application will not be considered)
2. The applications will be vetted by a select committee approved by the College Council.
3. Only short-listed applicants will be invited to attend the next available Exit Assessment (i.e. 15 June 2006).
4. The Exit Assessment panel will make a final recommendation to the College Council
5. The resolution of College Council will be ratified at the Annual General Meeting 2006 (no later than 28 July 2006).
6. Seniority date of Fellowship will be the date of the Annual General Meeting.
7. Fellowships ad eundem will then be recommended to the Academy for admission as fellow in the respective specialty.
Press Release 1 March 2006

It was noted recently that there is public concern regarding the safe use of sedative and anaesthetic drugs for minor surgery. The Hong Kong College of Anaesthesiologists and the Society of Anaesthetists of Hong Kong would like to make the following statement:

The development of modern anaesthesiology makes complicated major surgery possible. With appropriate facilities, judicious use of the analgesic, sedative and anaesthetic drugs, it is now possible to perform diagnostic or minor surgical procedures outside operating room and even outside the hospital. However, there are potential serious side effects associated with the use of these drugs. They range from unconsciousness, respiratory depression, seizures and cardiac arrest; and could be lethal if not managed accordingly. To be qualified as specialist anaesthetists in Hong Kong, medical practitioners are required to undergo a minimum of six years of postgraduate training and pass three professional examinations. Upon completion of training, Fellowship of Hong Kong College of Anaesthesiologists and subsequently Fellowship of Hong Kong Academy of Medicine would be awarded. Practicing specialist anaesthetists are required to register with the Specialist Register of the Medical Council of Hong Kong and hence would be under the regulation of the Medical Council.

Although there is currently no regulation to restrict the use of anaesthetic drugs only by specialist anaesthetists, practitioners are urged to observe the following precautions related to the use of analgesic, sedative and anaesthetic drugs:

1. Thorough understanding of the drugs to be administered including the pharmacology and side effects.
2. A pre-procedural assessment of the patient’s physical status to determine the suitability for sedative drugs.
3. Other than the operating medical practitioner or the nurse required for the procedure, another medical practitioner or a qualified nurse trained in resuscitation should be assigned for monitoring of the patient.
4. Resuscitation drugs and equipment should be available at the location of the procedure.

Patients should be well informed the risks inherited with the use of these analgesic, sedative and anaesthetic drugs. Specialist anaesthetists should be consulted if needed. Guidelines and recommendations regarding the safe use of sedative drugs can be found in various international institutions, and a suggested guideline is also available from the Hong Kong College of Anaesthesiologists’ web page http://www.hkca.edu.hk.

The Hong Kong College of Anaesthesiologists
The Society of Anaesthetists of Hong Kong

香港麻醉科醫學院及香港麻醉科學會就近日社會公眾對麻醉藥物的安全問題引起關注，特發表以下聲明：

麻醉學的發展令很多大型複雜的手術得以進行，而麻醉、鎮靜及止痛藥物的普及亦讓病人在醫院外也能接受一些小型手術和檢查。但「水能載舟，亦能覆舟」，麻醉藥帶來的風險實在不容忽視。過量的鎮靜及止痛藥物能令病人轉迅間失去知覺，停止呼吸；而過量的麻醉藥亦可引起併發症如痙攣，心臟停頓等；若未能及早發現和提供適當的治療；嚴重的可引致死亡。麻醉專科醫生的職責，就是讓病人安全地接受手術和檢查。在香港要成為麻醉專科醫生，需要在醫科畢業後接受六年專科培訓和通過三重專業考試，才能取得有關專業資格。本港執業的麻醉專科醫生均在醫務委員會的專科名冊上登記。

雖然至今尚未有法例規管使用上述藥物的人士必須持有特定的專業資格，但我们認為任何人士在使用麻醉、鎮靜及止痛藥物的時候，必需注意以下幾點：

(一) 充分了解麻醉藥的藥性和使用方法
(二) 為病人作麻醉前檢查和風險評估
(三) 有接受過適當訓練包括急救的專職醫護人員在旁負責監察病人的狀況
(四) 備有充足的急救器材和藥物

香港麻醉科醫學院及其他專業團體對小型手術和檢查施行鎮靜的守則則提供了清晰的指引，詳情可瀏覽香港麻醉科醫學院網頁 (www.hkca.edu.hk)。病人亦有權清楚了解麻醉的性質，若有疑問可向你的醫生或任何麻醉專科醫生查詢。
Fellowship Examinations 2006

Intermediate Fellowship Examinations
Examination Fee: $6,000

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Final Fellowship Examination in Anaesthesiology
Examination Fee: $9,500

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Final Fellowship Examination in Intensive Care Medicine
Examination Fee: $10,000

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Diploma in Pain Medicine
Examination Fee: $5,000

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Exit Assessments for Year 2006

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<tbody>
<tr>
<td>13 April 2006 Thursday</td>
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<tr>
<td>15 June 2006 Thursday</td>
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<tr>
<td>12 October 2006 Thursday</td>
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Trainees who qualified to apply for fellowship are recommended to have their applications arrived at the HKCA office at least 21 days before the scheduled Exit Assessment, to allow time for processing.

Application forms are available from Supervisors of Training and HKCA Office.
<table>
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<tr>
<th>Location</th>
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<tr>
<td>8-9 May, 2006</td>
<td>Theme: “From Policy to Practice, 政策與實踐”</td>
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<tr>
<td></td>
<td>Venue: Hong Kong Convention and Exhibition Center</td>
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<td></td>
<td>Contact: Hospital Authority Convention 2006 Secretariat Room 209N Hospital</td>
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<tr>
<td></td>
<td>Authority Building, 147B Argyle Street, Kowloon, Hong Kong SAR.</td>
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<tr>
<td></td>
<td>Tel: (852) 23006808; Fax: (852) 28950937; Email: <a href="mailto:hac@ha.org.hk">hac@ha.org.hk</a></td>
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<td><strong>Adelaide, AUSTRALIA</strong></td>
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<tr>
<td></td>
<td>Contact: Mr Christopher Boundy, South Australian Postgraduate Medical Education</td>
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<td></td>
<td>Association Inc (SAPMEA) Tel: 08 8274 6060 Fax: 08 8274 6000</td>
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<tr>
<td></td>
<td>Email: <a href="mailto:admin@sapmea.asn.au">admin@sapmea.asn.au</a></td>
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<td></td>
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<tr>
<td></td>
<td>Contact: Scientific secretariat, Erasmus MC, Dept. of Neurosurgery, Room Ba-463,</td>
</tr>
<tr>
<td></td>
<td>P.O. Box 2040, 3000 CA Rotterdam, The Netherlands; Phone: +31 (0)10 - 463 40 75;</td>
</tr>
<tr>
<td></td>
<td>Fax +31 (0)10 - 463 40 75; E-mail: <a href="mailto:m.vangemerden@erasmusmc.nl">m.vangemerden@erasmusmc.nl</a></td>
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<td><strong>Madrid, SPAIN</strong></td>
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<td><strong>Toronto, CANADA</strong></td>
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<td>16 - 20 June</td>
<td>Venue: Toronto. Contact: 1 Eglinton Avenue East, Suite 208, Toronto, Ontario</td>
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<tr>
<td></td>
<td>Canada M4P 3A1. Tel: 416 480 0602 Fax: 416 480 0320 Email: <a href="mailto:meetings@cas.ca">meetings@cas.ca</a></td>
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<td><strong>ANAESTHETISTS</strong></td>
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<tr>
<td></td>
<td>Contact: Organizers Australia. PO Box 1237, Milton, Qld 4064. Tel: +61 (0)7 3371 0333</td>
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<td></td>
<td>Unit 301, 3/F The Centre Mark, 287-299 Queen’s Road Central, Hong Kong,</td>
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52
THE INSTITUTE OF CLINICAL SIMULATION
A Collaboration between the Hong Kong College of Anaesthesiologists and the North District Hospital

Workshop in
Airway Management for Nurses
Organized by
The Hong Kong College of Anaesthesiologists
Institute of Clinical Simulation
North District Hospital

Aim: To enhance the knowledge and skill in airway management
Target Participant: All Nurses
Date: 20 May 2006 or 16 September 2006 or 18 November 2006
Time: 08:30AM - 12:30PM
Venue: ICS, 3/f, North District Hospital
Workshop Design: Lecture, Video, Skill Stations
Course fee: HK$ 350.00
Accreditation: 3.5 CNE points
Class Capacity: 16 Persons
Enquiry: College Secretariat at 28718833
Mr. Allick Chiu DOM (OT & SSO) NDH at 26838885

Application form can be downloaded from
The Hong Kong College of Anaesthesiologists Intranet www.hkca.edu.hk or Department of Anaesthesia & Operating Services NDH Intranet

(Application form can be downloaded from the College website: www.hkca.edu.hk)
Management of Anaesthetic Crisis (EMAC) course

(Accredited by the Australian and New Zealand College of Anaesthetists)

EMAC is a simulator-based course catered to management of anaesthetic crises developed by Australian and New Zealand College of Anaesthetists. It is comprised of 5 half-day modules, namely Human Performance, Cardiovascular Emergencies, Airway, Anaesthetic Emergencies and Trauma.

Venue: Institute of Clinical Simulation
       North District Hospital
       9 Po Kin Road, Sheung Shui

Date: 10-12 June 2006
       0800 hr to 1700 hr on 10 and 11 June, 2006
       0800 hr to 1230 hr on 12 June, 2006

       21-22 October 2006
       0800 hr to 1700 hr on 21 and 22 October, 2006
       0800 hr to 1230 hr on 23 October, 2006

CME points: HKCA 20 points

Max participants: 8

Fee: HK$4,000 per head

Format: Each registrant will participate in:
(1) Lectures
(2) Skills stations
(3) An introduction on the METI Simulator, the anesthetic machine for use in the workshop and the theories of crisis management
(4) Allocated time for hands-on crisis scenario management on the METI Simulator, rotating through different roles and handling different scenarios

Trainees starting training program on or after 1st January 2003 are required to complete the EMAC course or its equivalent.
Accredited for CME by
Hong Kong College of Anaesthesiology
(10 points per year) & Royal College of Anaesthetists (UK)

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ANNUAL SCIENTIFIC MEETING IN ANAESTHESIOLOGY 2006
ASM 2006
18-19 November 2006
Hong Kong Convention and Exhibition Centre

Invited Speakers
Professor Davy Cheng
University of Western Ontario, Canada

Dr Beverly Collett
Leicester Royal Infirmary, UK

Professor Peter Kam
University of Sydney, Australia

Professor John Murkin
University of Western Ontario, Canada

Professor Paul Myles
Monash University, Australia

Professor David Scott
St. Vincent’s Hospital, Melbourne, Australia

JOINTLY ORGANIZED BY:
The Hong Kong College of Anaesthesiologists
The Society of Anaesthetists of Hong Kong
Over a hundred million worldwide exposures since 1994*

*Based upon Abbott in-house data, as of Nov. 2003.
Abbott Laboratories Limited 20/F, AIA Tower, 183 Electric Road, North Point, Hong Kong. Tel: 2566 8711
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Reference:
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<th><strong>Board of Examination</strong></th>
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<th><strong>Board of Pain Medicine</strong></th>
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Organizer, Basic Science Course: CH Koo, Aaron Lai  
Organizers, Clinical Anaesthetics Courses (Informative course and Crash course): Douglas Fok and Eric So  
Chairman, The Institute of Clinical Simulation: KM Ho (Chairman)