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The Bulletin of the Hong Kong College of Anaesthesiologists

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Pre-oxygenation Using Circle Absorber

To the Editor:

Yeo and colleagues show that the effectiveness of pre-oxygenation using a circle absorber depends upon the fresh gas flow of oxygen.[1] The accompaniment and aim of pre-oxygenation is denitrogenation which is maximized by breathing an atmosphere containing no nitrogen. Unfortunately, although the circle breathing attachment is a very efficient system for economizing in the use of anaesthetic gases during maintenance, it is an inefficient breathing-attachment for changing inspired concentrations rapidly (because of the recirculation of expired gas). Ideally for economy in the use of gases, a non-rebreathing attachment such as the Magill attachment (Mapleson A) needs to be used for pre-oxygenation,[2] although the use of a second breathing attachment may add to the complexity and cost.

Bell has argued that routine pre-oxygenation should be a new 'minimum standard' of care.[3] However such a practice would markedly increase oxygen usage but pre-oxygenation studies by Drummond and Park,[4] Gold,[5] and McCrory and Matthews have demonstrated that three maximal deep-inspirations are as effective as three to four minutes of normal respiration using pure oxygen.[6] Baraka and associates have gone further and suggest that a single vital-capacity breath,[7] following forced exhalation, can achieve adequate pre-oxygenation within 30 seconds. Yeo and colleagues' method uses 40 litres of oxygen for pre-oxygenation whereas Baraka's method, perhaps using a six litre bag charged with pure oxygen, could reduce the amount of oxygen used to six litres. This use of a larger bag and temporarily higher flow rates of oxygen (to fill it) would achieve the desired economy whilst pre-oxygenating a patient with a circle.

Brigadier Ivan T Houghton
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In response:

Dr Houghton is correct in pointing out that although the circle system is very economical and efficient in allowing the use of low fresh gas flow during anaesthetic maintenance, it is not the ideal breathing circuit for pre-oxygenation. There has been no defined

criterion to represent an adequate level of pre-oxygenation, and different studies had used different endpoints for assessment. We have defined adequate pre-oxygenation as end-tidal oxygen concentration above 90%. In our study, we hope to reproduce a scenario similar to our day-to-day clinical practice, where one may preoxygenate the patient in the operating theatre without changing anaesthetic machine or breathing system. There are other benefits in using the same breathing system, such as a decrease in staff exposure to anesthetic gas during disconnection, a reduction in the risk of cross infection, and possibly avoiding mishaps from misconnection.

Using the circle system, a high fresh gas flow is required to wash out nitrogen, so that during the next breathe, the patient may inhale high concentration of oxygen for successful pre-oxygenation. Any residual nitrogen within the breathing circuit may actually hinder the pre-oxygenation process. It will be useful to find out if using a six litre bag charged with pure oxygen or other breathing pattern with circle system, could reduce the amount of oxygen used but still achieving the desired economy of pre-oxygenating a patient with the circle system.

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Indications of Dexmedetomidine in the Intensive Care Setting

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In the intensive care unit (ICU), most patients require sedation and analgesia to prevent delirium, allay anxiety, relieve pain, encourage sleep, facilitate mechanical ventilation, and modulate the physiologic responses to stress, such as tachycardia and hypertension. No currently available medication provides sedation and analgesia at the same time without significant respiratory depression. Therefore, at present, polypharmacy is routinely prescribed for patients for analgesia and sedation in the ICU to provide the ptimal therapy. Drugs used for sedation include: analgesics such as opioids, sedatives such as gamma butyric acid (GABA) receptor stimulants (e.g. propofol and midazolam), or neuroleptics. All of these agents have adverse consequences associated with their use, such as respiratory depression, abuse potential, lack of orientation and cooperation, delirium, hypotension, and tolerance.[1-6]

Dexmedetomidine is a novel lipophilic imidazole derivative with a higher affinity for α_2-adrenoceptors compared to the prototype drug, clonidine. Dexmedetomidine acts on the locus ceruleus / norepinephrine axis rather than the GABA receptor, providing sedation like non-REM sleep.[7,8] Its high quality of sedative, analgesic and sympatholytic effects blunt the cardiovascular responses (hypertension, tachycardia) without clinically significant respiratory depression and reduce the frequency of delirium in ICU. A number of pharmacological studies support the efficacy of this drug as well as studies on the mechanism of action have been conducted in various species. The appearance of a sedative analgesic effect and an anti-anxiety action has been confirmed at the clinical dose. Another novel advantage of this agent is that the sedated patient may be aroused and demonstrate normal cognitive ability. Therefore a neurological assessment could be performed whenever required. Dexmedetomidine was approved on January, 2004, by Japanese regulatory authority and launched in Japan on May, 2004. We introduced this agent in August, 2004, and started using it for the patients who required sedation and analgesia in intensive care setting. In this article, we present our experiences in the clinical use of dexmedetomidine at our center as well as essential points of the administration methods and the best cases of use.

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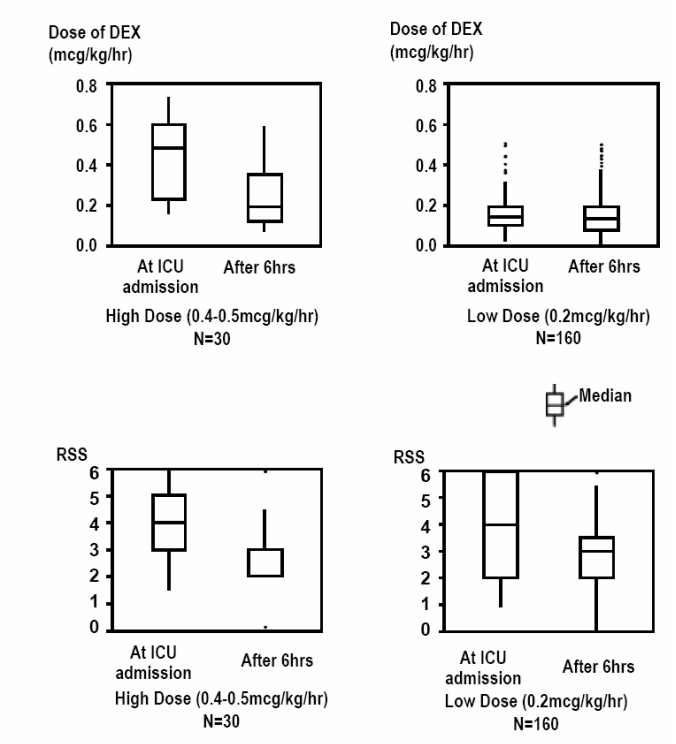
Dosage and Administration

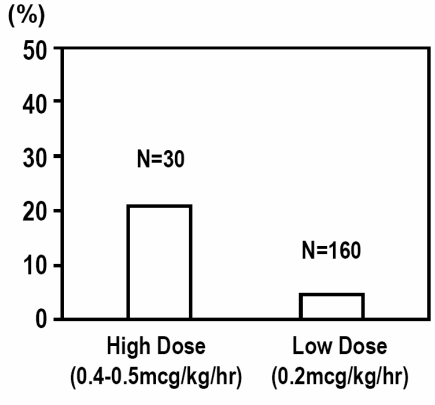
Our first impression on dexmedetomidine is that the sedative action is mild as compared to midazolam or propofol. Rapid increase in the infusion rate did not achieve a profound

Figure 1. Ramsey sedation score (RSS) in high and low dosing groups.



sedation, causing bradycardia or decrease in blood pressure. We then correlated the dose of dexmedetomidine was studied using the Ramsay sedation score as an index of the depth of sedation. Dexmedetomidine, which is not an intravenous anesthetic agent, did not induce sedation as deeper than a score higher than 4 in most patients at the approved maintenance infusion rate up to 0.7 μg/kg/h. In contrast, midazolam or propofol at increasing doses resulted in a sedation score ≥ 4. Dexmedetomidine was then administered at two doses: high dose (0.4 to 0.5 μg/kg/h) and low dose (0.2 μg/kg/h) doses to correlate with the sedation score 2 to 6 hours after drug administration. Comparison of the depth of sedation revealed that the target sedation score of 2 to 3 was achieved even in the low dose group. There was no significant difference in sedation scores between the two dose groups. Sedation was slightly more profound in the high dose group. It was also found that the low dose was appropriate to achieve arousal 6 hours after administration (Figure 1). On the other hand, cardiovascular complications such as bradycardia and hypotension occurred in about 20% of patients in the high dose group, but in the low dose group, the incidence was 25% less. The above dose-finding study results led us to administer dexmedetomidine at a relatively lower dose for a longer time in increasing

Figure 2. Incidence of bradycardia and hypotension in high and low dosing groups.



patients (Figure 2). Dexmedetomidine was used in about 30% of ICU patients when it was first introduced to us, but the rate of using dexmedetomidine has been now approaching 70%.

Use by disease

Figure 3 shows status of dexmedetomidine administration in each department of our hospital. Dexmedetomidine was used in more than 80% of patients who underwent cardiac surgery or surgery for esophageal, gastrointestinal, or lung cancer, but in only about 10% of patients after neurosurgery. Regarding to pathological condition, dexmedetomidine was used in about 80% of cases with respiratory failure, but required to be switched to other sedative agents about 24 hours after initiation of infusion in most patients. It appears that it is difficult to administer dexmedetomidine to patients with respiratory failure and sepsis.

Sedation was controlled to reach a sedation score of 2 to 3 at an infusion rate of 0.2 μg/kg/h or less. However, increased dose was required for cases with respiratory failure or sepsis. They often require switching to other sedative agents. Such dose escalation would lead to an increase in the incidence of adverse reactions (bradycardia and hypotension). Even in such cases, management with dexmedetomidine free from respiratory depression was useful during invasive procedures such as weaning of mechanical ventilator or immediately before leaving the ICU in the case of sepsis (Figure 4).

Incidence of delirium in the postoperative cases of esophageal cancer

Reduction in the incidence of delirium was studied as a potential advantage for dexmedetomidine. Prior to dexmedetomidine was introduced to our hospital (non-dexmedetomidine treated group), delirium at the ICU occurred in about 40% of patients on the following day after surgery for esophageal cancer, but the incidence was lowered to about 10% after dexmedetomidine was used (dexmedetomidine treated group). However, occurrence of delirium was observed in about 30% of patients after the patients were transferred to the general ward from the ICU following completion of dexmedetomidine administration (Figure 5). These study results strongly suggested that dexmedetomidine is useful in clinical use for delirium.

Experience in pediatric use

In children, most concerned is age-dependent difference in the sedative effect. In our hospital, dexmedetomidine is used for about

Figure 3. Percentage use of dexmedetomidine

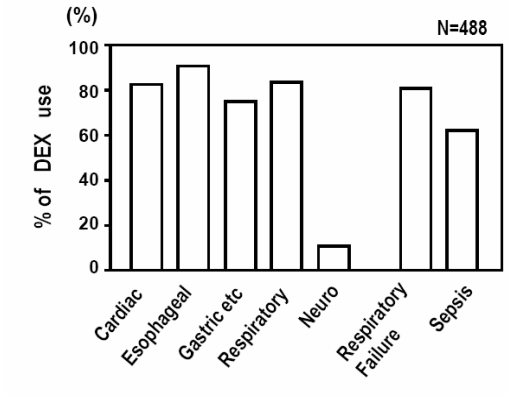


Figure 4. Dexmedetomidine infusion rate (6 hours after start of infusion)

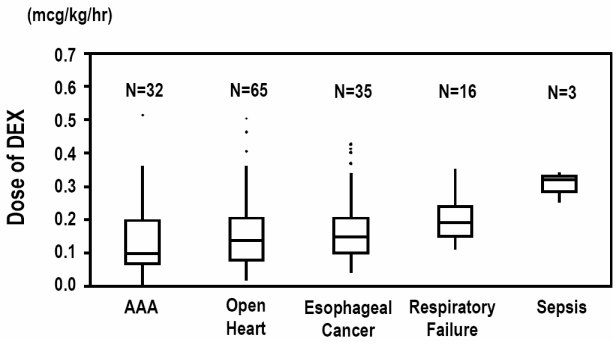
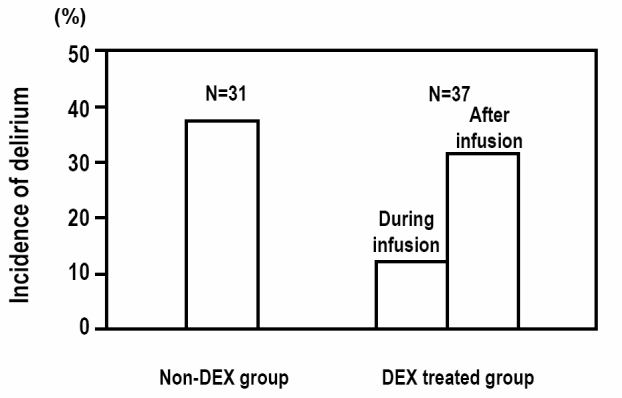


Figure 5. Incidence of delirium after oesophageal surgery.



60% of infants under one month of age. It is quite difficult to judge sedation in infants, and therefore, strict monitoring of hemodynamics is inevitable. Figure 6 shows the percentage of the dexmedetomidine treated children and average dose by age. Dexmedetomidine was used in 70% of infants under 1 year of age, small children under 6 years of age, and children aged 6 years or over, as is for adults. In small children under 6 years of age, the dose is more than that for adults to stabilize the pulse rate and blood pressure (0.3 to 0.4 μg/kg/h). In children, it appeared that dexmedetomidine is less effective during moderate or major procedures, as in the cases of adults. However, dexmedetomidine is almost free from respiratory depression, and thus allowing its use even after extubation. In the cases after Fontan operation where management of pulmonary vascular resistance is required, dexmedetomidine was found to be useful, because of its advantage for early extubation.

Conclusion

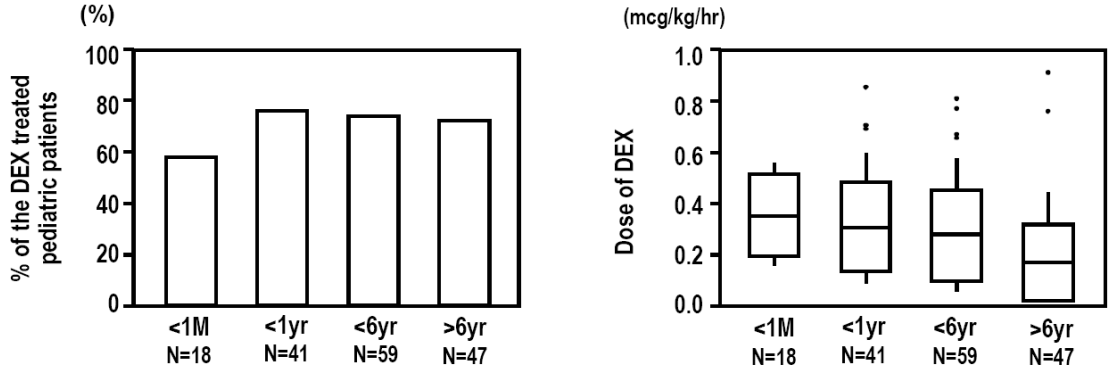
Dexmedetomidine is indicated for the postoperative cases where delirium is expected after admission to ICU or respiratory depression is of concern. To circumvent the cardiovascular adverse reactions, the dose is considered appropriate around 0.2 μg/kg/hr. Although the dose for children tends to be increased, it would not be advisable to increase the dose. The combination with other sedative agents may be considered, if sedation is insufficient.

Acknowledgments

The author gratefully acknowledges the contributions of the ICU staff in Osaka City General Medical Center and Professor Shinichi Nishi, MD PhD for his guidance. The author also thanks Kazuko Taiji of Hospira Japan for her informative support.

The author and Professor Shinichi Nishi, are the advisors of the dexmedetomidine clinical research for Hospira, Japan.

Figure 6. Percentage of patients treated with dexmedetomidine and the average dose range.



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COX-3: biochemistry, pharmacology, relevance to paracetamol and future prospects

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Cyclooxygenase (COX) is the key enzyme in the rate-limiting step that converts arachidonic acid to prostaglandins and thromboxane.[1] There are two commonly known cyclooxygenase: COX-1 and COX-2. Recently a new COX, a splice variant of COX-1 called COX-3 was discovered. COX-3 messenger ribonucleic acid (mRNA) is expressed in canine cerebral cortex, but the resultant enzyme has been characterized with limited success in humans. Drugs such as paracetamol and phenacetin inhibit COX-3. A COX-3 pathway may be the primary central mechanism by which these drugs exert their analgesic and anti-pyretic effects.[2]

The aim of this review was to summarize the biochemistry of COX-3 and discusse its clinical relevance with respect to paracetamol.

Methods

Sources for this review include Medline 1980-2005 (searched under the following Medical subject headings: Cyclooxygenase, COX-3, Paracetamol, acetaminophen, splice variants) and PubMed (www.ncbi.nlm.nih.gov/ entrez) 1980-2004. The bibliographies of the included studies were also scanned for additional references (reference dredging).

History

Acetylsalicylic acid (aspirin) has been used since 1899. However it was not until 1971 that Sir John Vane identified COX as the molecular target for aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDS).[3] Between 1989 - 1992 a new isoform COX-2 was discovered by three independent laboratories.[4-6] The elucidation of the structure and then the cloning of the gene for COX-1 and COX-2 in 1991 revolutionized the understanding of NSAID pharmacology by providing a clear pathway for prostaglandin regulation.[7,8] However there is still no explanation for the mechanism of action for drugs like paracetamol. Flower and Vane have postulated in 1972 that paracetamol inhibits prostaglandin synthesis in the brain.[9] However, a definite enzyme for this inhibition could not be found for a considerable period of time until in 2002.

It has been postulated for the past 10 years that there are other isoforms of the COX enzyme, which may be inhibited by paracetamol.[10] It was postulated that the enzyme COX-3 existed although it had not been isolated. Originally in 1999, Simmons *et al* postulated that COX-3 might be immunologically related to COX-2, when they demonstrated an inducible paracetamol sensitive COX-2 enzyme with reduced sensitivity to NSAIDs.[11]

Willoughby *et al*, in 2000, hypothesized that COX-3 may act in the resolution phase of

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Glossary

Process of transcription and translation

(transcription) *(translation)*

DNA → nuclear RNA → messenger RNA (mRNA) → transfer (tRNA) → amino acid → polypeptide protein

Transcription

The synthesis of RNA from a DNA template. It only requires one strand of the DNA double helix. It is comprised of initiation, elongation and termination. Initiation is the binding of RNA polymerase to the double stranded DNA. Elongation is the addition of nucleotides to the 3′ end of the chain, forming the pre-mRNA transcript. Splicing then occurs, which is the removal of introns and joining of exons. Termination is the recognition of the termination sequence and release of messenger RNA polymerase. Finally a tail is added to the mRNA transcript.

Nuclear RNA

Unedited mRNA transcripts, or pre-mRNAs

Transfer RNA

Small RNA molecules that carry amino acids to the ribosome for polymerization into a polypeptide

Messenger RNA

An RNA molecule transcribed from the DNA of a gene, and from which a protein is translated by the action of ribosomes. The basic function of the nucleotide sequence of mRNA is to determine the amino acid sequence in proteins.

Intron

A non-coding sequence of DNA that is initially copied into RNA but is cut out of the final RNA transcript. Also called an intervening sequence.

Codon

A group of three nucleotides that specifies addition of one of the 20 amino acids during translation of an mRNA into a polypeptide

Exon

The region of a gene that contains the code for producing the gene's protein. Each exon codes for a specific portion of the complete protein.

Splicing

The process by which introns are excised out of the primary messenger RNA and exons are joined together to generate a mature messenger RNA. Heterogeneous nuclear RNA is capped and polyadenylated.

Isoforms

Different forms of a protein that may be produced from different genes, or from the same gene by alternative splicing

Dimerization

A reaction where two identical molecules, the monomers, are unified without significant structural changes of the educts in the combined entity that is usually called dimer.

Splice-site mutation/Frameshift

A mutation in which there is an addition or deletion of one, two or a small number (not a multiple of three) of nucleotides that causes the codon reading frame to shift to one of two others from the point of the mutation during translation. Consequently the amino acid sequence of the protein is altered from the point of the mutation to the carboxy terminus. Such mutations can result in one or more introns remaining in the mRNA and can disrupt the generation of the protein product.

Translation

Translation is the process of converting the mRNA codon sequences into an amino acid sequence. It is composed of initiation, elongation, translocation and termination. Initiation and elongation occur when the ribosome recognizes the starting codon (a sequence of 3 bases) on the mRNA strand and binds to it. The ribosome has sites, which allow another enzyme, tRNA to bind to the mRNA. On tRNA, there is an anticodon that is used to match the codon on the mRNA. tRNA also has a single unit of amino acid attached to it. As the ribosome travels down the mRNA one codon at a time, another tRNA is attached to the mRNA at one of the ribosome site. The first tRNA is released, but the amino acid that is attached to the first tRNA is now moved to the second tRNA, and binds to its amino acid. This translocation continues on, and a long chain of amino acid (protein), is formed.

When the entire unit reaches the end codon on the mRNA, it falls apart and a newly formed protein is released. This is the termination.

Northern blot

A technique for transferring RNA fragments separated by electrophoresis to a nitrocellulose filter, on which specific fragments can then be detected by their hybridisation to probes, which may be labelled radioactively.

Western blot

A technique for analysing and identifying proteins. The proteins are separated by electrophoresis in gel, and then transferred ("blotted") onto a piece of treated pater, where they bind in the same pattern as they formed in the gel.

acute inflammatory responses, separate to COX-1 and COX-2.[12] This pathway produces anti-inflammatory compounds that include the cyclopentenone prostaglandin family.[13] They concluded that it might have implications in the treatment of chronic inflammatory diseases such as rheumatoid arthritis. It was not until 2002 that Simmon's group isolated COX-3 mRNA in canine cerebral cortex and showed that resultant proteins can be inhibited by antipyretics like paracetamol.[1]

Biochemistry

COX-1 and COX-2

COX-1 is the constitutional isoform, expressed under physiological conditions and is important in maintaining renal function, platelet function and the gastrointestinal epithelium.[2] COX-2 is the inducible isoform that is expressed when there is inflammation. COX-2 also has homeostatic roles in the kidney, cardiovascular system and the brain.

Prostaglandin I_2 has previously been shown to be the predominant product of COX reaction in endothelium that results in inhibition of platelet aggregation, vasodilatation, and prevents the proliferation of vascular smooth-muscle cells in vitro. Although it was initially thought to be produced by COX-1, it is actually produced by COX-2. It was then postulated that COX-2 selective inhibitors reduce PGI_2 and theoretically can increase thrombotic risks.[14]

Recent studies, Vioxx Gastrointestinal Outcomes Research (VIGOR) and Adenomatous Polyp Prevention on Vioxx (APPROVe), have shown that a highly selective COX-2 inhibitor, rofecoxib, increases the risks of heart attacks and cerebral vascular accidents. About 3.5% of active treatment patients suffered myocardial infarction as opposed to 1.9% of patients who received placebos. The drug company, Merck, has withdrawn it from the market voluntarily in October 2004.[15]

Discovery of COX-3 in dogs

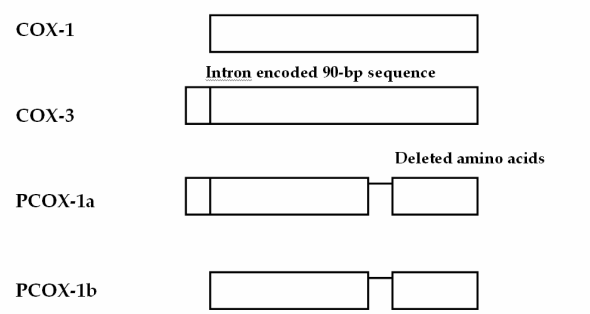
The discovery of COX-3 mRNA was a major breakthrough when Simmons' laboratory detected COX-3 mRNA using canine brain cells in 2002. First, a deoxyribonucleic acid (DNA) library of canine cerebral cortex was constructed. Two mRNAs with COX-1 coding was then transcribed using Northern blot analysis. A Northern blot is a technique used for transferring RNA fragments separated by electrophoresis to a filter, on which probes can then detect specific fragments.[1]

When the RNAs were studied, it was discovered that they were 75% similar to intron 1 of human COX-1 gene. Normally during splicing, introns (non-coding DNA sequence that is initially copied into RNA, "junk" DNA found in the middle of a gene sequence and with unknown function) are removed and exons (encoding regions of gene that produce the protein) are joined together to form a continuous RNA coding sequence. This intron-1 containing COX-1 was therefore very unusual. When the Northern blot was repeated using radiolabeled intron 1-specific probe, multiple intron-1 containing splice variants were expressed. Multiple COX-1 isoenzymes were derived as a result. Figure 1 shows a graphical depiction of the splice variants of COX-1 mRNA.

Analysis of the RNAs suggested that they could translate into previously uncharacterised COX-1 related proteins. The major protein product was named COX-3 because it was pharmacologically different to COX-1 and COX-2 even though it came from the COX-1 gene. It shared all the catalytic features and structural features of COX-1 and COX-2. Canine COX-3 is a 613 amino acid protein with a molecular weight of 65kDa.[1] However, inhibition studies with analgesics/antipyretics showed that it had different enzymatic properties to COX1 and COX2.

Two other isoenzymes were produced in the process. They were called partial COX-1a

Figure 1. COX-1 mRNA and its slice variants. Modified from Chandrasekharan N *et al*.[2]



(PCOX-1a) and partial COX-1b (PCOX-1b). PCOX-1a and PCOX-1b lack detectable COX activity.[1] These partial COX isoenzymes did not synthesise prostaglandins but bind heme and may have other enzymatic properties.[8]

COX-3 in other species

Other laboratories have isolated COX-3 in other animal models. In insect cells, COX-3 has 20% of the activity of COX-1.[8]

Kis and Snipes' laboratory have successfully isolated the putative COX-3 mRNA in rat's brain. They found that it was present in all brain cell types except neurons, and cerebral endothelial cells such as the pituitary, hypothalamus and choroid plexus had the highest expression. The expression of COX-3 mRNA was 30% of that for COX-1 mRNA in brain micro-vessels. It was postulated that cells constitutively express COX-3 because the expression of COX-3 did not change with lipopolysaccharide challenge. Bacterial infection causes lipopolysaccharide release and this stimulated inflammation.[16,17]

Human COX-3?

In humans, the existence of COX-3 at the nucleotide sequence is questionable. Intron 1 DNA sequence is found in humans. Intron-1 containing COX-1 mRNA is much larger than the COX-1 message (5.2 *vs* 2.6 kb). Intron retention during splicing is poorly understood.[1] There have been concerns that the larger mRNA

may not be transported from the nucleus to endoplasmic reticulum and be glycosylated (an important step for enzyme folding and activity). N-linked glycosylation was subsequently demonstrated in COX-3 insect cells.[1]

Frame reading (the reading of the succession of codons determined by reading nucleotides in groups of three) is part of translation. Translation is the process in which specific functional proteins are produced. Human COX-1 mRNA containing intron-1 disrupts the triplet reading frame leading to the creation of a premature termination codon and results in a truncated protein product. This is called frame shift. Abnormal amino acid sequence results and the proteins are non-functional.

Chandrasekharan *et al* suggested that if the full length of intron 1 is retained, then human COX-3 differs by one nucleotide from that in dogs and thus shift the remainder of the protein out of frame. However, partial or modified retention of intron 1 may occur in human COX-3.[1]

Dinchuk *et al* have also attempted to characterize human COX-3 by cloning 24 separate COX-1 cDNA. Intron 1 of human COX-1 is 94 nucleotides long and shifts the sequence of human COX-3 out of frame with respect to the open reading frame of COX-1. They concluded that a full-length, catalytically active form of COX-3 did not exist in humans and human COX-3 was "in the wrong frame".[18]

Simmons' laboratory, on the other hand, demonstrated that the presence of intron-1 containing COX-1 mRNA produced proteins. They used immunoblots, an anti-COX-3 directed antibody, and demonstrated that it competed with intron-1 peptide.[8] An immunoblot is a technique for analysing or identifying proteins via antigen-antibody specific reactions, is also known as Western blot. Other studies may have failed to identify the protein because they may have used non-specific antibody testing. Simmons showed that the resultant proteins are likely to contain a sequence encoded by intron-1.[8] The retention of intron 1 adds 30-34 amino acids to the resultant protein depending on the mammalian species.[19] Human COX-3 contains 633 amino acids.[20]

Structure and sites of action

All the information for the biosynthesis of a native protein is encoded in the DNA. The sequence of the DNA, and thus the RNA, determines the 3-D structure of the product. Retention of intron 1 in the COX-1 mRNA would alter folding, dimerization and active sites of action of COX-3. The steps to protein folding are first the transition of a random coil to secondary elements. The elements cooperate and eventually polypeptide subunits are formed and these assemble to become a dimer and finally a native protein.[21]

The enzymatic properties of COX-3 are different to that of COX-1 and COX-2 as indicated by its lower potency to produce prostaglandin E2 and different inhibition potencies by NSAIDS (Table 1).[1,22]

The retention of intron-1 may alter the site where mRNA is polyadenylated. The functional significance of intron retention and alternative polyadenylation is not known and needs to be fully elucidated.

The catalytic domains of COX-1 and COX-2 each contain 2 active sites, the cyclooxgenase active site and the peroxidase active site. Each step is performed at distinct sites on the same protein. The first step of action is the binding of arachidonic acid to the COX site and cyclization to form an unstable intermediate, an endoperoxide. This is a cyclooxygenase reaction. Then the endoperoxide binds to the peroxidase site and a reduction reaction occurs to form a stable prostaglandin H_2 (PGH_2). This is a classical peroxidase reaction. All other prostaglandins are made from PGH_2.[22,23]

Subcellular localization studies indicate that COX-3 is a membrane bound enzyme.[1] It is likely that COX-3 contains similar catalytic domains but will bind to substrates differently.

Postulated Function of COX-3

In 2000, using Western blot analysis, Willoughby *et al* originally identified a new enzyme protein, postulated to be COX-3, which was expressed during the resolution of inflammation. They concluded that the inducible COX-3 might be essential during remission period in chronic inflammatory disease. Its expression was spatially and temporally separate from prostaglandin producing COX-1 and COX-2. It was postulated that COX-3 might be an anti-inflammatory compound belonging to the cyclopentenone prostaglandin family. They suggested that drugs that induced COX-3 might be important and drugs that inhibited COX-3 might be deleterious.[12,13]

COX-3 may also be involved in the development of Alzheimer's disease. The concentration of COX-2 is raised in Alzheimer's disease. COX enzymes potentiate inflammatory neuropathology. Patients on chronic NSAIDS have a lower incidence of developing Alzheimer's disease.[24] COX-3 may play a role in the disease process as high levels of COX-3 in the brain have been found in patients suffering from Alzheimer's disease. A recent study has shown that COX-3 has ancillary roles in membrane-based COX signaling. It might be vital in the development of Alzheimer's disease when basal levels of COX-1 or COX-2 expression are persistently high.[25]

COX-3 may be involved in the development of cancer. COX-1 is over expressed in ovarian adenocarcinomas and cervical carcinomas.[26,27] COX-1 activity potentiates the differentiation of acute promyelocytic leukemia.[28] As COX-3 is a COX-1 splice variant, it may be expressed in some forms of cancer and its modification may change the course of mutagenesis. COX-3 (COX-1 splice variant) mRNA levels have been demonstrated to be elevated in colorectal tumours and reduced after NSAID treatment to normal levels in colonic mucosa in rats.[29]

However, it is postulated that the major function of COX-3 is that it may be involved in the main chemical pathway by which paracetamol acts in the central nervous system. It is unknown whether COX-3 mRNA can produce an enzyme that will convert arachidonic acid to prostaglandins, ie. a COX. However it is unlikely that the body would produce an enzyme in high quantities in the brain that serves no purpose. COX-3 mRNA accounts for 30% of COX-1 mRNA levels. Also, the expression of COX-3 mRNA is strong in sites which are targets of antipyretic action of paracetamol – the hypothalamus, pituitary and choroid plexus.[16] A study looking at murine astrocytic and microglial cDNA has found that acute inflammation does not stimulate the expression of COX-3.[30]

Relevance with paracetamol

Paracetamol was first used in medicine in 1893.[31] Since then it has become the most widely used anti-pyretic and analgesic agent. Its mechanism of action is still not fully elucidated despite years of research.

Flower and Vane first postulated that paracetamol has a central mechanism of action. At physiological pH range it exists in the largely unionised form, therefore easily crossing the blood brain barrier.[9] Studies have consistently shown that paracetamol inhibits prostaglandin synthesis in the central nervous system. Enantiomers of NSAIDS that are devoid of peripheral cyclooxygenase effects have been shown to be effective in the central nervous system.[32,33]

Paracetamol is a potent analgesic and anti-pyretic agent but lacks anti-inflammatory activity. Its action is highly selective. It decreases levels of prostaglandin metabolites in urine but not in platelets or stomach mucosa.

Prostaglandins released by cerebral endothelium are important in the regulation of vascular tone and in the development of fever. Formation of prostaglandins occurs when COX-2 is induced by endotoxin in the endothelial cells of hypothalamic blood vessels. The prostaglandins formed penetrate into the organum vasculosum laminae terminalis to produce fever. Paracetamol may inhibit the enzyme in endothelial cells or exert its antipyretic action centrally.[34]

In 2002, Boutaud *et al*, using human umbilical vein endothelial cell, demonstrated one possible mechanism of action of paracetamol. They showed that paracetamol inhibited prostaglandin H synthase-2 (PGH_2) by acting as a reducing agent (the second step in the biosynthesis of prostaglandins). It stimulated COX activity at low concentration (0.1-1mM) and inhibited COX at high concentrations. This is because excess reducing agents will inhibit the tyrosyl feedback loop and inhibit COX.[23,35] Adding peroxide to increase the oxidative state of enzymes will reverse the action of paracetamol. In the brain, where peroxide levels are not raised in fever, paracetamol is effective.

Paracetamol is a very weak COX-1 or COX-2 inhibitor at therapeutic concentrations. Different studies have quoted different IC_{50} values, with IC_{50} values varying from 100 M to >6.6mM.[23,36,37]

Paracetamol is poorly effective at inflammatory sites, where there are raised peroxide levels. The peroxide levels are also high in platelets.[35] It does not inhibit COX-1 in the stomach, which may be the reason why paracetamol does not damage the stomach mucosa. Lung COX-1, however, is most sensitive to paracetamol inhibition. There is a positive association with increase in asthma attacks and frequent paracetamol use.[38]

In the brain, paracetamol appears to selectively inhibit COX enzymes other than COX-1 or COX-2. In 2000, Simmons *et al* have identified a COX activity which is more susceptible to paracetamol inhibition than COX-1 or COX-2 using chicken embryo fibroblasts. They concluded that increased susceptibility is due to cell-specific factors that may the due to the induction a new COX-3.[22]

When the COX-3 mRNA was inserted into baculovirus insect cells they produced a novel COX that produced prostaglandins. The inhibition of the protein by NSAIDS and paracetamol (COX-3) was compared with COX-1 and COX-2. It was discovered that COX-3 was more sensitive to paracetamol than COX-1 and COX-2 even at low concentrations of substrate (5M arachidonic acid).[1,19]

The sensitivities of COX-3 to paracetamol and pyrazolone drugs, such as dipyrone, are summarised in Table 1.

It should be noted that in Chandrasekharan's study canine COX-3 was compared with murine COX-1 and murine COX-2. The DNA of intron 1 in the dog, human and mouse are very similar.[19]

NSAIDS that inhibit COX-1 also inhibit COX-3. COX-3 actions are not independent from COX-1. Lower IC_{50} values for COX-3 suggest that COX-3 is blocked before COX-1 and this effect might last longer than COX-1 blockade.[20]

Prostaglandin E_2 (PGE_2) is an important product in inflammation and the end product of

Table 1. IC_{50} values of analgesics and NSAIDS

	IC_{50} (M)		
Drug	**COX-1**	**COX-2**	**COX-3**
Aspirin	10	>1000	3.1
Diclofenac	0.035	0.041	0.008
Paracetamol	>1000	>1000	460
Phenacetin	>1000	>1000	102
Dipyrone	350	>1000	52

(Modified from Chandrasekharan *et al*.[2] All assays were carried out in the presence of 30 M arachidonic acid.)

cyclooxygenase reaction. It affects monocyte maturation, inhibits proliferation of T-lymphocytes and is an autocrine factor in dendritic cell maturation. It has been described as the ultimate mediator to generate hyperthermia during fever. Paracetamol decreases prostaglandin E_2 production in a dose dependent manner in cerebral microvasculature. The co-incidence of high levels of COX-3 and high levels of paracetamol-sensitive levels of cerebral endothelial cells may suggest a possible interaction.[20,39]

In a study comparing hypothermia, decreased PGE_2 levels and paracetamol in knock out mice, COX-3 appears to be the determining enzyme involved. COX-1 knock out mice show 70% decrease in baseline brain PGE_2 levels, but these levels are not reduced further by paracetamol. COX-2 knock out mice show unaltered PGE_2 levels with paracetamol. Meanwhile, COX-3 inhibitors - antipyrine and aminopyrine, reduced brain PGE_2 levels even in normal mice. Therefore the researchers concluded that parace-tamol reduces PGE2 by inhibiting COX-3.[40]

Canine COX-3 is inhibited by paracetamol at physiological concentrations. IC_{50} by paracetamol is 460 μM. The therapeutic serum concentration of paracetamol is 66-132 μM in humans however (or 10-20 g/ml). Therefore COX-3 activity does not completely explain the action of paracetamol as this plasma concentration of IC_{50} is difficult to achieve with the usual oral dose of 0.5-1.0 g.[41,42]

Future prospects

Proper nomenclature of COX enzymes will be required as more genetic isoforms are discovered. It is confusing that an enzyme that comes from a COX-1 gene is named COX-3 and not COX-1a. The term "COX-3" should be reserved for the protein produced by an independent COX-3 gene that is yet to be identified.[19] Some studies have used the term COX-1 variant (COX-1v) instead.

Currently several companies are developing COX-3 inhibitors to enable research in the functional role of COX-3.[19,43] The use of computer software to predict protein function may become more important as technology advances. It may be the first step in characterizing possible function of new variants.[44]

Pharmaceutical companies should develop new COX reagents, antibodies, primers and screening kits specific for each COX variant found. They should also be species specific.[19]

Currently, COX-1 knock out mice are deficient in both COX-1 and COX-3. No specific knock out mice for each COX variant has been developed. The development of specific COX-3 knock out mice is likely to be challenging.[19]

COX-3 has only been recently characterized and it is still not clearly demonstrated in human tissue. Its exact pathophysiological role is still unknown. Few publiccations exist on the subject. Presences of functional relevant COX-1 splice variants in humans need to be verified.[13,37] Further work on COX-3, particularly at physiological concentration of its substrate, arachidonic acid, is required to elucidate its actions.[45]

Conclusion

Currently the only evidence of COX-3 comes from Simmons' laboratory. There is a lack of confirmation and extension of these results in other laboratories. Concrete evidence for COX-3 is reported in two papers to date. One demonstrated the existence of COX-3 mRNA in canine cerebral cortex and the other used immunoblots and antibodies to prove that functional proteins in humans are produced by the splice variant COX-1 mRNA.

A functional COX-3 in human is yet to be isolated. The identification of additional functional COXs in the generation of bioactive autacoids can lead to the development of more

selective drugs.[20] Further study is required to search for a functional COX-3 protein.

It is currently too early to comment on the possible use of a COX-3 inhibitor, as the physiological action of COX-3 is still unknown. Whether selective COX-3 inhibition is feasible is also questionable as it has similarities in its action with COX-1. NSAIDS can have unpredictable effects because of interaction with various COX isoforms.[46]

The discovery of COX-3 warrants the search for further COX variants and other paracetamol sensitive antipyretic proteins. More work on whether paracetamol work selectively through this enzyme in vivo is required.[19]

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Compliance with Intensive Care Admission Guidelines on Triage

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SUMMARY

Appropriate utilization of expensive resources has become an important issue due to limited health budgets. Admission to the intensive care unit (ICU) should be reserved for those patients with reversible medical conditions who have a "reasonable prospect of substantial recovery". The Task Force of the American College of Critical Care Medicine, Society of Critical Care Medicine has introduced guidelines for prioritizing admission. These define those that will benefit most from ICU (Priority 1) to those that will not benefit at all (Priority 4). We aimed to test our compliance to the model and to critically analyze its usefulness as a triage tool.

We performed a prospective audit of all adult referrals for admission to our 22 bed multidisciplinary University ICU for one month. Patients were categorized according to the guidelines by two investigators not involved in the triage decision. The proportion of patients admitted in each priority group was calculated.

The audit consisted of 117 patients. The mean age was 58 and the mean Mortality Prediction Model at admission (MPM II_0) 0.37. There was an inverse relationship between Priority category and admission rate. The priority categories correlated well in terms of MPM II_0 and mortality. Based on MPM II_0 there was no significant difference in admission rate between different severity groups and the proportion of those with moderate to high MPM II_0 admitted were high. There was an overall reduction in mortality amongst each severity groups but significantly higher in those with high MPM II_0.

Our triage decisions complied well with the guidelines. This is evident from the high admission rate in the Priority 1 group and a reducing admission rate in the subsequent categories. The model correlated well in terms of the severity of underlying illness and mortality between different categories. However, it gives little insight to the spectrum of illness within groups and hence it's usefulness as a triage tool is limited.

Keywords: Intensive care medicine; Admission criteria; Triage; Prediction model; Resource allocation

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When evaluating a patient with critical illness for intensive care unit (ICU) admission, the attending physician must determine the underlying diagnosis, treatment modalities and prognosis.

In the United States, critical care beds account for approximately 8% of hospital beds, 28% of charges for acute hospital care come from ICU and comprise >1% of gross national product.[1] Due to the limited health budget, appropriate

utilization of medical expenses is essential. In the ideal situation, patients would be accepted or discharge strictly according on their potential to benefit from ICU care. In agreement with the principles of beneficence, nonmaleficence, autonomy and justice in resource distribution, ICU should be reserved for those patients with reversible medical conditions who have a "reasonable prospect of substantial recovery".

Various triage recommendations for cardiac, trauma and patients with gastrointestinal hemorrhage have developed by the North American and European Societies of Critical Care Medicine.[3-5] The Task Force of the American College of Critical Care Medicine, Society of critical Care Medicine introduced a series of guidelines for prioritizing admission.[2] One of which was the *Prioritization Model*. These define those that will benefit most from ICU (Priority 1) to those that will not benefit at all (Priority 4). We aimed to test our compliance to the model and to critically analyze its usefulness as a triage tool.

Materials and Methods

The Prince of Wales Hospital in Hong Kong is a multidisciplinary university tertiary referral center with 400 beds. Our ICU functions as a "closed" unit with 22 beds. Both surgical and medical patients are accepted into the unit and are managed by physicians trained in critical care medicine.

We performed a prospective audit of all adult referrals for admission to our ICU from November to December 2001. The attending medical personnel involved in the referral also obtains the following information: hospital admission date; date and time of the triage decision; patient's age and gender; diagnosis precipitating ICU referral; the number of beds available at the time.

Data forms were reviewed by two independent investigators not involved in the triage decision within 24 hours for accuracy. Patients were then categorized according to the *prioritization model* and the mortality prediction model at admission (MPM II_0) calculated at admission or at the time of triage.[6] Their actual hospital mortality was also prospectively collected.

Data analysis

*P*atients' demographic and clinical data were recorded in a computerized database. All parametric results are presented as mean ± standard error of the mean (SEM) and between group means were compared by analysis of variance. Other non-parametric results were obtained by χ^2 analysis. A *P* value of less than 0.05 was considered as significant.

Results

During the one month study period, a total of 117 patients were referred to ICU for admission. According to the *prioritization model*, we found an inverse relationship between the rate of admission and priority category (Table 1). Of these, 93 (79%) patients were admitted and 24 (21%) were refused. The admission diagnoses of the triaged patients are shown in Table 2. There were no differences in age and gender between the priority groups of the admitted patients (Table 3).

The mean mortality prediction model at admission (MPM II_0) of each respective category group correlated well with that of predicted. Category 2 and category 3 had the lowest and highest average value respectively whilst category 1 patients' average value was in between of the two categories.

Amongst the 93 patients admitted, they were further divided into four equal MPM II_0

Table 1. Composition of triaged patient

Priority Category	Number of patients (% of total)	Number admitted (% of admission)
1	84 (72%)	79 (94%)
2	17 (15%)	11 (65%)
3	7 (6%)	3 (43%)
4	9 (7%)	0 (0%)

Table 2. Admission diagnoses of triaged patient.

	Admitted Number (%)	Not admitted Number (%)	Total Number (%)
Respiratory failure	27 (29%)	7 (29%)	34 (29%)
Neurosurgical	15 (16%)	1 (4%)	16 (14%)
Cardiac	12 (13%)	2 (8%)	14 (12%)
Airway problems	7 (8%)	2 (8%)	9 (8%)
General surgical	7 (8%)	2 (8%)	9 (8%)
Trauma	6 (6%)	3 (13%)	9 (8%)
Neurological	5 (5%)	3 (13%)	8 (7%)
Sepsis	4 (4%)	3 (13%)	7 (6%)
Renal failure	4 (4%)	-	4 (3%)
Cardiothoracic	2 (2%)	-	2 (2%)
Vascular	2 (2%)	-	2 (2%)
Urological	1 (1%)	-	1 (1%)
Hepatic	1 (1%)	1 (4%)	2 (2%)
Total	93 (100%)	24 (100%)	117 (100%)

groups, 0-0.25 (n = 43), 0.26-0.50 (n = 29), 0.51-0.75 (n = 19) and 0.76-1.0 (n = 14).

According to this MPM II_0 subdivision, there was no significant relationship between the severity of illness and overall ICU admission. Figure 1 (P = 0.43). Ideally, we should admit all Category 1 patients but due to our limited budget a small percentage of them were denied. Those patients that were refused had low to medium range MPM II_0 while no patients with moderate to high MPM II_0 range were denied admission on the basis of resource.

Based on the prioritization model, hospital outcome of the admitted patients also correlated well as expected (Figure 2). Category 2 (9%) and category 3 (33%) patients had the lowest and highest mortality, respectively. Category 1 patients (18%) had an intermediate mortality between the two.

Despite the overall improvement in survival as a group, there was a clear increased in mortality within the high MPM II_0 subgroup (P < 0.001). Similarly, within category 1 patients there was an increased mortality within the high MPM II_0 group (P < 0.006).

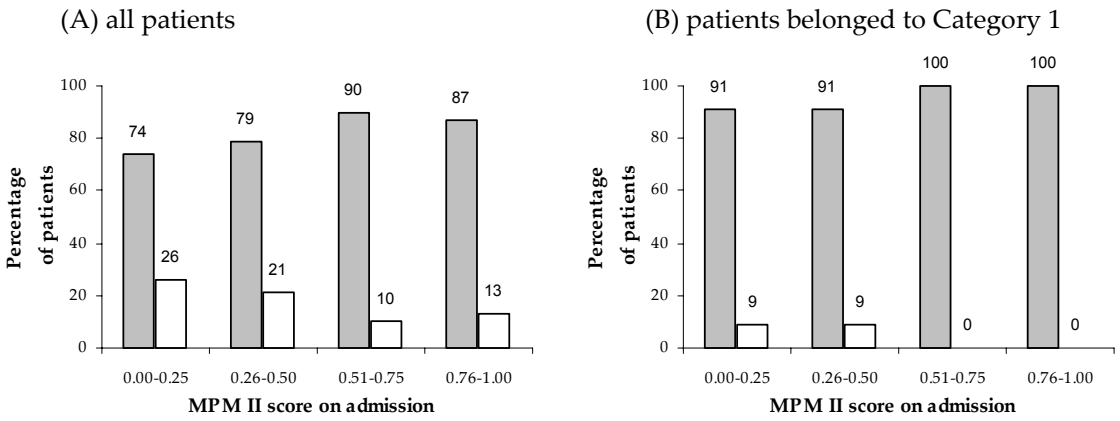
Discussion

The present study is the first to use the "Prioritization Model" to audit ICU admission in Hong Kong. It is also the first to evaluate the usefulness of the model as a guideline in determining triage decisions. During the study period our unit complied well with the admission guideline. This is evident from the inverse relationship between "Priority category" and the proportion of patients admitted. There was a high percentage of admission in category 1 as these patients would benefit most from ICU admission. On the contrary, low admission rate

Table 3. Characteristics of admitted patients.

	Category 1	Category 2	Category 3	P value
Number	79	11	3	
Age	58 ± 18	50 ± 19	64 ± 10	0.35
Gender				
Male	52 (83%)	9 (14%)	2 (3%)	0.57
Female	27 (90%)	2 (7%)	1 (3%)	

Figure 1. Proportion of admission according to MPM II_0 (Admitted ▩ ; not admitted □).



was found amongst category 3 and no admission for category 4 as these patients would benefit little from ICU admission.

Respiratory failure, cardiac failure and neurological interventions (both elective and emergency) comprised the majority of our admissions. Both neurosurgical and cardiac patients had the highest odds ratio for admission (4.42 and 1.63, respectively). This is in keeping with a previous study, which also demonstrated a high likelihood of cardiac admission[7]. However, due to the limited size of studied sample the 95% confidence intervals were too wide to be statistically significant.

When using MPM II_0 as a surrogate measure of disease severity, it correlated well with the "Prioritization Model" categories. Category 2 patients had the lowest MPM II_0, as a majority of these patients required only intensive monitoring. Hence their expected MPM II_0 should be the lowest. In contrast, category 3 patients had the highest MPM II_0 as these patients had a reduced likelihood of recovery because underlying disease or of their acute illness. While category 1 patient had a predicted mean MPM II_0 between the two groups.

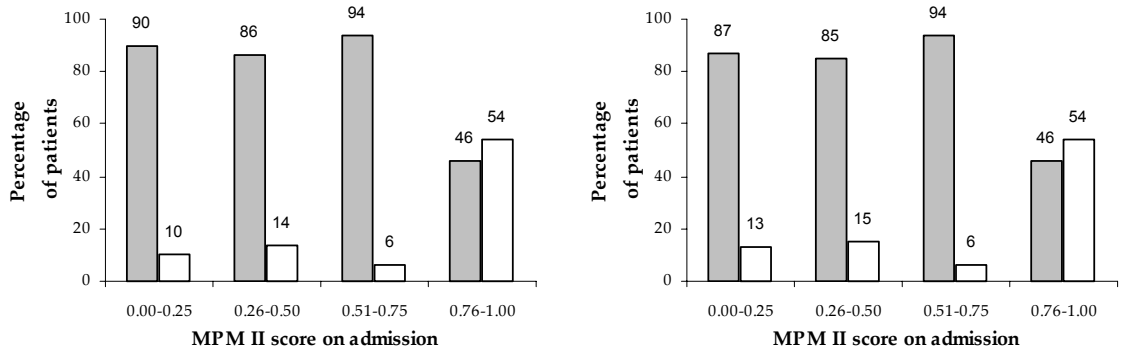
Similarly, when patients were grouped according to the subdivisions, the proportion of admission was inversely related to the MPM II_0. Despite this, 14% of patients with a high severity score were actually admitted; these were the patients that we thought would benefit from ICU. In fact, the admission rates were not statistically different between the defined MPM II_0 subdivisions. Furthermore, within category 1, all patients with moderate and high severity score were admitted. The consequence of this was demonstrated by the hospital outcome of these patients, as we will discuss later on.

The expected mortality also correlated well with the prioritization model. Category 3 and category 2 patients had the highest and lowest mortality respectively while category 1 group's hospital mortality was between the two. The actual mortality also correlated well according to the MPM II_0.

Although the overall mortality in our "closed" ICU was lower than the expected mortality within each group,[7,8,9] there was a significantly higher mortality amongst those with a high MPM II_0 (Figure 2). Again, similar results were found within the Category 1 patients; those with high MPM II_0 score had a higher mortality rate.

In view of our limited resources, the question is not only whether these patients should ever be admitted to ICU. But it is how to

Figure 3. Hospital outcome classified according to MPM II_0. (Survived ▭ ; Dead □).



select out those who are severely ill when we make a triage decision. This raised a number of issues.

Are we too liberal in regards to ICU admissions? Over the years of British influence, Hong Kong has slowly advanced into a westernized society, and as well as the people's attitude towards health. It is not uncommon for patients' relatives to push for the "best" of care despite the high likelihood of poor outcome. The referring teams may also initiate this pattern of admission. As most critically ill patients are not capable of giving consent a paternalistic altitude by the referring team may reflect the number of patients with high MPM II_0 being referred and subsequently admitted.[10] Certain primary teams were almost always granted ICU admission, in particular with the surgical teams. This is in keeping with previous study by Sprung *et al*. They have also shown that surgical status has a positive predictive impact on triage decision.[11] In our study, neurosurgery, cardiothoracic and vascular surgery had the highest odds ratio for admission and they also had the highest $MPMII_0$ scores. Does it simply reflect to types of patients selected or does it reflect the type of pattern of referrals. In fact, another study also demonstrated that political powers and medical provincialism overrode medical suitability in the provision of the critically ill[12]. These factors will no doubt be difficult to resolve.

Although the above guideline is ethically sound and medically appropriate the implementation of such a guideline in a real life situation may be difficult.[13]

Was there an intrinsic problem in our admission system? Almost all patients (96%) in category 1 were admitted according to the "Prioritization model"; as these patients would benefit the most. However, amongst them included a proportion with high MPM II_0 scores. Were these patients justified for admission? Was the ICU director or in-charged physician informed of these "borderline" patients, as according to the guidelines.[3-5] If so, what were the factors that prompted for their admission?

Could the "Prioritization model" allow us to determine the appropriateness of ICU admission? According to the prioritization model, patients that belonged to category 4 should have the lowest priority for ICU admission. Under these circumstances, the ICU director should determine the suitability for admission on an individual basis. From the

above, even though there was no patient admitted under category 4, there were a significant proportion of critically ill patients, with high MPM II_0 scores admitted under category 1. Accordingly, what determines the difference between a high MPM II_0 category 1 patient and that of category 4 patient? Or, in another words, when does a patient become so ill that death is imminent or irreversible? Since others have demonstrated the importance of the severity of underlying illness in making a triage decision it would seem that the underlying physiological parameters are more appropriate when formulating such a decision.[14,15]

Although the "Prioritization Model" is useful in predicting in-between group MPM II_0 scores and in-between group mortality, it is less useful in determining the intra-group differences. Furthermore, it gives little insight on the spectrum of severity of the underlying illness; especially within category.

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The Recovery Profile of Sevoflurane *versus* Isoflurane in Patients Undergoing Eye Surgery Monitored with Auditory Evoked Potential

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SUMMARY

Auditory evoked potential (AEP) monitoring device provide practitioners with information about the hypnotic component of anesthesia. With this guidance, we may titrate the amount of anesthetic agent given to patient to maintain an adequate level of anesthesia. This should avoid awareness during anesthesia, provide a better recovery profile, minimize side effects and hopefully allow more economical use of expensive anesthetic. Newer and more expensive volatile anesthetics such as sevoflurane has lower blood:gas partition coefficient. It allows a more rapid recovery from anesthesia. We performed a prospective, double blind randomized control study to assess the differences in recovery profile after sevoflurane (study group n = 20) and isoflurane (control group n = 20) anesthesia using the A-line ARX index titrated anesthetic technique in patients undergoing retinal surgery. The sevoflurane group did not show any significant advantage over isoflurane group in recovery profile including time to emergence, time to respond to commands, time to extubation, time to regain orientation and recovery room discharge.

Keywords: Anesthetic depth monitoring; Auditory evoked potential; Retinal surgery; Recovery profle; Sevoflurane

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Newer volatile anesthetics such as sevoflurane and desflurane are reported to have a better recovery profile compared with older agents because of their lower blood:gas solubility. However, the cost per minimal alveolar concentration (MAC) of these volatile agents is 2 to 3 times of isoflurane. In order to justify the increased expenditure, a cost:benefit ratio need to be demonstrated.[1-4]

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Early studies comparing the recovery profile of sevoflurane and isoflurane rely on clinical judgment of anesthetists to assess and establish equianesthetic state. New techniques to monitor depth of anesthesia such as auditory evoked potential (AEP) and bispectral index (BIS) have been introduced which are both technically easy and effective for detecting whether patients were awake or asleep.[5]

AEP is a response of brain to any acoustic stimuli. Changes in latency and amplitude of AEP wave are highly correlated with transition from awake to loss of consciousness. However, it is a weak electrical signal wrapped in the EEG background activity. Extraction of AEP requires advanced signal processing. The AEP monitor (Danmeter, Odense, Denmark) permits fast

extraction of AEP. It features ARX-modeling, a mathematical method that can extract the AEP within a few repetitions. This reduces the delay to as much as 2 to 6 seconds. AEP is a complex signal; therefore it is desirable to map the AEP into an index (autoregressive AEP index, AAI) for easy interpretation but containing the same information of AEP. The AEP monitor calculates an AAI continuously and this appears in the display as a single number and a graphic curve.

AAI value shows appropriate sensitivity to general anesthesia with similar graded changes over a whole range of anesthetic agents. AAI is typically more than 60 when patient is awake and decreases when the patient is anesthetized; loss of consciousness occurs when the AAI is below 30. The AEP index offers a better discriminatory power over BIS in describing the transition from conscious to unconscious state.[5] Therefore, AAI should be helpful in guiding the anesthetist to achieve an adequate level of hypnosis with minimal amount of anesthetics.[6-13]

We performed a prospective double blind randomized control study to assess the recovery profile of sevoflurane (study group) *versus* isoflurane (control group) using AEP to titrate the amount of anesthetic required in patients undergoing retinal surgery.

Methods and Materials

Approval was obtained from the Research Ethics Committee of the Queen Elizabeth Hospital. Forty patients aged between 18 and 60 years, American Society of Anesthesiologists physical status class 1 to 3, scheduled for elective retinal surgery were recruited. Based on the results of a pilot study and previous studies, a sample size of 20 groups should be adequate to detect 30% difference in the time to response to command and tracheal extubation with a power of 0.8 at an α value of 0.05.[16-18] Written informed consent was obtained from each patient.

Patients were randomly allocated to receive either isoflurane or sevoflurane anesthesia according to a computer-generated random number table. Those with known history of psychiatric disorders, hearing impairment, allergy to any drugs given during anesthesia or refusal were excluded. Since the intensity of the 'clicks' sound (70 dB) generated by AEP monitor was louder than normal conversation, we assumed patient had normal hearing if the patient could hear the anesthetist during the pre-operative assessment. Both the patient and the observer were blinded to the volatile anesthetic being used for maintenance of anesthesia.

Pre-anesthetic preparation

No pre-medication was given. On arrival to operating theatre, standard monitoring including pulse oximetry, non-invasive blood pressure, electrocardiogram were applied. Intravenous infusion was set up. Muscle relaxation was monitored by nerve stimulator (TOF Watch, Organon Teknika)

AEP monitoring

The AEP monitor was used. The skin over the intended electrode placement was cleaned with alcohol and scrubbing to achieve low electrode impedance (< 10 kΩ). Three electrodes were then applied o over the middle of forehead (white), left forehead (green) and left mastoid (black) according to manufacturer's recommendation. The headphone was then fitted to the patient. Adequate time was allowed for the readings to stabilize before commencement of anesthesia.[14]

Induction

Standardized anesthesia technique was adopted. While pre-oxygenating patients with 100% oxygen for 2 to 3 minutes, baseline values for AAI and hemodynamic variables were obtained. Anesthetic induction was achieved with fentanyl 1.5 μg/kg and propofol 1-3 mg/kg Atracurium 0.5 mg/kg were given to facilitate tracheal intubation.

Maintenance

Anesthesia was maintained with either sevoflurane or isoflurane in an oxygen:air mixture with inspired fraction of oxygen (fiO_2) between 0.3 and 0.5. Total fresh gas flow was kept at 100 ml/kg/min for the first 10 minutes and then 2 L/min for the maintenance phase.

The end tidal volatile anesthetics concentration was set at 1.3 MAC initially i.e. isoflurane 1.5% and sevoflurane 2.5% and then titrated to maintain the AAI between 20 and 30. The stepwise titration of volatile anesthetic concentration was adjusted according to the vaporizer markings. If AAI was below 20 for past 3 minutes, volatile anesthetic vaporizer was turned down half of the marking.

All patients were mechanically ventilated to maintain an end-tidal carbon dioxide concentration of 32-36 mmHg. Muscle relaxation was maintained by atracurium bolus to keep the count ≤ 1. Time and amount of last dose of atracurium as well as the total amount of atracurium administered were recorded.

Bolus doses of morphine 1.5 mg IVI were administered to maintain adequate analgesia in the presence of persistent elevation in heart rate (> 100 beat/min) or mean arterial pressure (> 20% of baseline) when AAI < 30. Other drugs such as ephedrine or anti-hypertensive drugs administrated during the surgery were recorded.

Hemodynamic variables were recorded every 5 minutes while End-tidal volatile anesthetics concentration and AAI were recorded every 10 minutes. The time of completion of last stitch of surgery was taken as the end time. At the end of surgery, neostigmine 0.05 mg/kg and atropine 0.02 mg/kg were given to reverse the muscle relaxation.

Measurements of Recovery Profile[3,15-17]

Patients were called at 15 seconds intervals by a blinded observer who did not know the volatile anesthetic used.

Table 1. Discharge criteria

(1) alert and orientated to time and place,
(2) conversant and cooperative,
(3) stable vital signs for at least 30 minutes,
(4) able to sit-up without dizziness and/or nausea,
(5) pain is tolerable,
(6) Aldrete score ≥ 8.

Recovery times were defined as follows:

(1) Emergence was the time to eye opening;
(2) Response to commands was the time to correct response to verbal commands (hand squeeze);
(3) Extubation was the time to removal of tracheal tube.
(4) Orientation was the time to ability to state name, date of birth and current location.
(5) Recovery discharge was the time to fulfill the recovery room discharge criteria (Table 1).

Data Collection

We recorded adverse events such as bradycardia (HR < 50 beats/min) and hypotension (mean arterial pressure < 20% of baseline) during anesthesia. The amount of analgesic and antiemetics given were also recorded. Before discharge from the recovery room, patients were asked whether they can recall any of the intraoperative events such as hearing any sound or feeling any pain during the anaesthesia.

Statistical analysis

Data are expressed as mean ± standard deviations. Student *t* test was used for continuous variables including the amount of anesthetic and analgesic drugs given, and the time of recovery profile. Non-parametric variables, including number of patients with bradycardia, postoperative nausea and vomiting, pain and those requiring vasopressor therapy

Table 1. Characteristics of patients receiving sevoflurane or isoflurane.

	Sevoflurane	Isoflurane	*P* value
Number of patients	20	20	
Age (years)	40 ± 16	45 ± 15	0.30
Weight (kg)	63 ± 9	63 ± 12	0.97
ASA physical status (I/II)	12/8	14/6	0.51
Duration of surgery (min)	114 ± 41	125 ± 34	0.39
Duration of anesthesia (min)	133 ± 40	143 ± 34	0.37

Values are number or mean ± standard deviation

were analyzed using χ^2 test. A P value < 0.05 was considered statistically significant.

Results

A total of 40 patients were recruited. Two patients were excluded because of noisy signals causing interference to AEP monitoring. Patients receiving sevoflurane or isoflurane were comparable with respect to age, weight, ASA physical status, duration of surgery and anesthesia (Table 1).

Anesthetic usage is summarized in Table 2. The amount of morphine, atracurium and volatile anesthetics used during surgery were similar between groups. The average AAI were similar between groups throughout the surgery. Recovery times did not differ between groups (Table 2).

The incidence of adverse events was similar between groups (Table 3). The postoperative course was uncomplicated in all patients. No patient recalled intraoperative event in the recovery room.

Discussion

Sevoflurane is a less soluble compared to isoflurane and previous studies have shown that the use of sevoflurane allowed faster recovery and earlier discharge from the recovery room than isoflurane.[3,17-20] These studies depended on clinical judgment to assess adequate depth of anesthesia such as hemodynamic response and eye signs of anesthesia, which had been shown to be unreliable. Titration according to pre-determined end-tidal concentration of volatile anesthetic to reflect anesthetic effect is also unreliable

In this study, we adopted the AEP monitor to titrate the amount of volatile anesthetics to achieve a predetermined depth of anesthesia. We demonstrated that sevoflurane offered no advantage with respect to recovery profiles compared with isoflurane.

Table 2. Drug usage and recovery times

	Sevoflurane	Isoflurane	*P* value
Drug usage			
atracurium (mg/kg/h)	0.63 ± 0.12	0.58 ± 0.12	0.28
volatile anesthetic (MAC)	0.66 ± 0.13	0.50 ± 0.11	0.32
morphine (mg/kg/h)	0.03 ± 0.01	0.02 ± 0.08	0.10
Recovery times to			
emergence(s)	299 ± 140	253 ± 80	0.21
response to commands (s)	356 ± 169	289 ± 88	0.13
tracheal extubation (s)	399 ± 174	355 ± 90	0.32
orientation(s)	548 ± 205	613 ± 296	0.42
recovery discharge (min)	42 ± 6	41 ± 4	0.69

Values are number or mean ± standard deviation

Table 4. Adverse effects

	Sevoflurane	Isoflurane	*P* value
Bradycardia	4 (20)	2 (10)	0.38
Postoperative nausea and vomiting	2 (10)	3 (15)	0.63
Use of ephedrine	2 (10)	3 (15)	0.63
Significant postoperative pain	3 (15)	2 (10)	0.63

Values are number (%).

One of the possible explanations may be related to the duration of anesthesia. The recovery endpoints for less soluble anesthetics such as sevoflurane may be less sensitive to the duration of anesthesia. It appeared that after a short anesthetic (less than one hour), there is minimal difference in recovery would between any of the volatile anesthetics. We believe this is because there is insufficient time to saturate the body tissues. The difference was significant if the anesthesia time is longer than 3 hours.[3] This was demonstrated by Eger *et al* in a study of rodents in which the rapid recovery of less soluble anesthetics, such as sevoflurane or desflurane, was difficult to demonstrate after anesthetic exposure of less than one hour compared with longer anesthetics.[21] It also has been shown in rodents that the differences in times to recovery endpoints between anesthetics are smaller when low concentrations of the anesthetic are used.

In our study, the duration of anesthesia was about 1-2 hours. In keeping with the animal data, the difference in recovery profile between sevoflurane and isoflurane may not be apparent.[3] The average concentration of sevoflurane and isoflurane was 0.6 and 0.5 MAC, respectively, and may not influence the recovery profile.

We chose those patients with retinal surgery because the surgical stimulus was not as intense compared with other major operations such as laparotomy. In our study, surgical stimulus would not lead to patient's movement because of inadequate level of anesthesia relative to the stimulus. Hence, effect of anesthesia is better demonstrated without confounding factor of inadequate analgesia.

Besides, patients with retinal surgery usually require relatively higher concentration of volatile anesthetic to ensure adequate depth of anesthesia as nitrous oxide is commonly avoided in anticipation of intraocular gas injection during surgery. We wish to avoid nitrous oxide because Barr *et al* showed that it reacted with AAI in a non-linear fashion.[22]

As in other studies using bispectral index for anesthetic titration, the anesthesia was maintained at relatively 'light plane' and the amount of muscle relaxant required monitored to avoid movement during the surgery.[18] Other drugs, like ephedrine, may have an effect on the AAI. Mori *et al* suggested ephedrine can cause excitation of the central nervous system during light anesthesia and the AAI value may increase.[23] We found no difference in the amount of ephedrine used in our study and there was no significant difference in consumption between the two groups.

The limitation of the AEP monitoring is that it is dependent on the transmission of auditory signals. Therefore patients with hearing problems, psychiatric illness, and previous ear surgery cannot be benefited from AEP monitoring.

In summary, our study showed that sevoflurane has no advantage over isoflurane with respect to recovery profile on patient undergoing retinal surgery with AEP monitoring anesthesia. The use of AEP to monitor the level of hypnosis could assist the

anesthetists in titrating the amount of anesthetic used to the required level of anesthesia.

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Thank you, Edward!

Dr Edward Ho has recently resigned from the Council and the Hospital Authority and is now staying in the United Kingdom. Edward has been serving the College in various Boards and Committees, including the Assistant Treasurer, member of the Formal Project committee and Examiner in the Final Fellowship Examination.

The Council would like thank Edward for all his contributions to the local anesthetic community. Fellows and members, who wish to get in touch with Edward, should contact the College office for details.

The Council has also resolved to appoint Dr CH Koo as the new Assistant Treasurer.

Honorary Treasurer's Report for Financial Year 2005

This is my first report on the financial status of the Hong Kong College of Anaesthesiologists. I am very thankful that I inherited a very wealthy position. I have the pleasure to announce that our college is very healthy financially. With the combined efforts of many dedicated fellows, in year 2005, we managed to have a handsome surplus of HK$1,040,454. Together with the retained surplus brought forwarded from 2004, our total assets stood at HK$11,662,221.

Income

Our main source of revenue came from annual subscriptions by members and fellows. Seventeen new members and thirty new fellows joined the College last year. Income from subscriptions was HK$1,021,851. Interest rate started to climb in 2005 and the income from this source went up to HK$90,576. The 2005 Annual Scientific Meeting was also a successful event and our share of profit was HK$229,955. We also had solid income from various courses and the total amount was HK$130,458. Our share of income from the Institute of Clinical Stimulation (ICS) was HK$3,230. Two intermediate and two final examinations, one pain examination, one intensive care examination and four exit assessments were conducted in the year 2005. Income from these activities amounted to HK$79,027.

Expenditure

Total expenditure by our college in year 2005 was HK$723,574. The major expense was in salary. Other major expense was for maintenance of the college chamber.

Annual Subscription

It was resolved that the annual subscription for year 2006 would stand at:

Fellows	HK$ 2,500
Members	HK$ 1,250
Overseas Fellows	HK$ 625
Overseas Members	HK$ 313
Members and fellows > 65 years	HK$ 50

Appreciation

The total assets of our college have reached over ten million dollars by end of 2005. This is not easy considering we do not have many fellows and members compared to other colleges. This healthy financial status obviously is the result of the tremendous efforts of many of our hardworking colleagues to whom I would like to say thank you again.

Dr Anne Kwan
Honorary Treasurer
June 2006

AUDITORS' REPORT TO FELLOWS AND MEMBERS OF THE HONG KONG COLLEGE OF ANAESTHESIOLOGISTS

(incorporated in Hong Kong with liability limited by guarantee)

We have audited the accounts on pages 152 to 160 which have been prepared in accordance with accounting principles generally accepted in Hong Kong.

RESPECTIVE RESPONSIBILITIES OF COUNCIL MEMBERS AND AUDITORS

The Companies Ordinance requires the directors to prepare accounts which give a true and fair view. In preparing accounts which give a true and fair view it is fundamental that appropriate accounting policies are selected and applied consistently.

It is our responsibility to form an independent opinion, based on our audit, on those accounts and to report our opinion solely to you, as a body, in accordance with section 141 of the Companies Ordinance, and for no other purpose. We do not assume responsibility towards or accept liability to any other person for the content of this report.

BASIS OF OPINION

We conducted our audit in accordance with Statements of Auditing Standards issued by the Hong Kong Institute of Certified Public Accountants. An audit includes examination, on a test basis, of evidence relevant to the amounts and disclosures in the accounts. It also includes an assessment of the significant estimates and judgments made by the council members in the preparation of the accounts, and of whether the accounting policies are appropriate to the college's circumstances, consistently applied and adequately disclosed.

We planned and performed our audit so as to obtain all the information and explanations which we considered necessary in order to provide us with sufficient evidence to give reasonable assurance as to whether the accounts are free from material misstatement. In forming our opinion we also evaluated the overall adequacy of the presentation of information in the accounts. We believe that our audit provides a reasonable basis for our opinion.

OPINION

In our opinion the accounts give a true and fair view of the state of the college's affairs as at 31st December, 2005 and of its surplus for the year then ended and have been properly prepared in accordance with the Companies Ordinance.

R. Kadir & Company
Certified Public Accountants (practicing)

HONG KONG

THE HONG KONG COLLEGE OF ANAESTHESIOLOGISTS
INCOME AND EXPENDITURE ACCOUNT FOR THE YEAR ENDED 31st DECEMBER, 2005

	Notes	2005	2004
		HK$	HK$
TURNOVER			
Members' entrance fees and subscriptions	4	1,021,851	1,007, 876
OTHER REVENUE			
Donation		15,350	511
Interest income		90,576	14,394
Miscellaneous		193,581	68,524
Surplus from events :-			
Annual scientific meeting	5	229,955	247,244
Institute of Clinical Simulation	6	3,230	6,609
Courses	7	130,458	118,271
Examination	8	79,027	233,472
		742,177	689,025
TOTAL REVENUE		1,764,028	1,696,901
EXPENDITURE			
Audit fee		10,000	7,000
Bank charges		1,496	914
Crown rent and rates		7,242	7,161
Depreciation		29,886	29,630
Insurance		36,035	35,932
Management fee		73,980	73,980
Members' subscriptions written off		18,900	43,750
Miscellaneous		78,468	55,638
Mandatory provident fund		11,317	10,300
Office supplies		32,722	41,573
Postages, stationery & printing of educational materials		149,361	164,925
Salary		270,720	289,225
Subscriptions		3,447	3,408
		723,574	763,436
SURPLUS FOR THE YEAR		1,040,454	933,465
RETAINED SURPLUS BROUGHT FORWARD		10,621,767	9,688,302
RETAINED SURPLUS CARRIED FORWARD		11,662,221	10,621,767

THE HONG KONG COLLEGE OF ANAESTHESIOLOGISTS
BALANCE SHEET AS AT 31ST DECEMBER, 2005

	Note	2005 HK$	2004 HK$
NON CURRENT ASSETS			
Fixed assets	10	23,524	52,130
Held-to-Maturity debt securities	11	5,100,000	1,099,230
		5,123,524	1,151,360
CURRENT ASSETS			
Held-to-maturity investments	11	1,099,833	-
Accounts receivable		77,864	56,870
Cash and bank balances		5,769,077	10,068,915
Prepayment		27,500	122,831
		6,974,274	10,248,616
LESS : CURRENT LIABILITIES			
Accounts payable and accruals		430,927	778,184
Receipt in advance		4,650	25
		435,577	778,209
NET CURRENT ASSETS		6,538,697	9,470,407
NET ASSETS		11,662,221	10,621,767
Represented by :			
RETAINED SURPLUS		11,662,221	10,621,767

THE HONG KONG COLLEGE OF ANAESTHESIOLOGISTS
FOR THE YEAR ENDED 31ST DECEMBER, 2005
STATEMENT OF CHANGES IN EQUITY

	2005 HK$	2004 HK$
Opening balance - Total equity	10,621,767	9,688,302
Net surplus for the year	1,040,454	933,465
Closing balance - Total equity	11,662,221	10,621,767

THE HONG KONG COLLEGE OF ANAESTHESIOLOGISTS
CASH FOLW STATEMENT
FOR THE YEAR ENDED 31ST DECEMBER, 2005

	2005 HK$	2004 HK$
Operating activities		
Surplus for the year	1,040,454	933,465
Depreciation	29,886	29,630
Interest income	(90,576)	(14,394)
Operating surplus before working capital changes	979,764	948,701
(Increase)/Decrease in accounts receivable	(20,994)	185,218
(Decrease)/Increase in trade and other payable	(347,257)	202,416
Decrease/(Increase) in prepayment	95,331	(122,831)
Increase/(Decrease) in receipt in advance	4,625	(2,425)
Net cash inflow from operating activities	711,469	1,211,079
Investing activities		
Payment for purchase of fixed assets	(1,280)	(56,248)
Payment for purchase of certificate of deposit	(5,100,000)	(1,099,230)
Interest received	89,973	14,394
(Decrease) / increase in cash and cash equivalents	(4,299,838)	69,995
Cash and cash equivalents at 1st January	10,068,915	9,998,920
Cash and cash equivalents at 31st December	5,769,077	10,068,915

THE HONG KONG COLLEGE OF ANAESTHESIOLOGISTS
NOTES TO THE ACCOUNTS

1. INCORPORATION

The college was incorporated on 26th September, 1989 under the Companies Ordinance and its liability is limited by guarantee. The address of its registered office and principal place of business is Room 807, Hong Kong Academy of Medicine Building, 99 Wong Chuk Hang Road, Aberdeen, Hong Kong. Its principal activity is the promotion of the knowledge on anaesthesiology among the members of the College.

Under the provisions of the college's Memorandum and Articles of Association, every member shall, in the event of the college being wound up, contribute to the assets of the college to the extent of HK$100. At 31st December, 2005, the college had 468 members.

2. PRINCIPAL ACCOUNTING POLICIES

The principal accounting policies adopted in the preparation of the accounts are set out below. These policies have been consistently applied to all the years presented, unless otherwise stated

(a) Basis of preparation

The College's financial statements have been prepared in accordance with Hong Kong Financial Reporting Standards ("HKFRSs"). The financial statements are prepared under the historical cost convention, except that the held-to-maturity investments are stated at their amortized cost as explained in note 2 (c).

The preparation of financial statements in conformity with HKFRSs requires the use of certain critical accounting estimates. It also requires management to exercise its judgment in the process of applying the appropriate accounting policies.

The adoption of new/revised HKFRSs

In 2005, the College adopted the new/revised standards and interpretations of HKFRSs below, which are relevant to its operations.

HKAS 1	Presentation of Financial Statements
HKAS 7	Cash Flow Statements
HKAS 32	Financial Instruments : Disclosures and Presentation
HKAS 39	Financial instruments : Recognition and Measurement

The adoption of revised HKAS 1 and 7 has no material effects on the College's financial performance and positions. The adoption of HKASs 32 and 39 has resulted in a change in the accounting policy relating to the measurement of held-to-maturity investments which has been described in note 2 (c).

All the changes in the accounting policies have been made in accordance with the transition provisions in the respective standards, wherever applicable. The adoption of these new/revised standards does not require to amend the 2004 comparatives. The HKAS 39 does not permit to recognize, derecognize and measure financial assets and liabilities in accordance with this standard on a retrospective basis. The College applied the previous SSAP 24 "Accounting for investments in securities" to investments in securities for the 2004 comparative information. The effect of the accounting differences between SSAP 24 and HKAS 39 is insignificant and therefore no adjustment on the opening reserve at 1st January, 2005.

(b) Revenue recognition

Donation is recorded on the cash basis. Entrance and subscription income is recognized when the right to receive payment is established. Interest income is recognized on a time-proportion basis using the effective interest method. Revenue on other events is recognized when the right to receive such revenue has been established.

(c) Held-to-maturity debt securities

From 1st January, 2004 to 31st December, 2004:

Held-to-maturity investments are stated in the balance sheet at cost plus/less any discount/premium amortized to date. The discount or premium is amortized over the period to maturity and included as interest income/expense in the income statement. Provision is made when there is a diminution in value other than temporary.

The carrying amounts of held-to-maturity investments are reviewed at the balance sheet date in order to assess whether the credit risk and whether the carrying amounts are expected to be recovered. Provisions are made when carrying amounts are not expected to be recovered and are recognized in the income statement.

From 1st January, 2005 onwards:

Held-to-maturity investments are non-derivative financial assets with fixed or determinable payments and fixed maturities that the College's management has the positive intention and

ability to hold to maturity. They are included in non-current assets, except for those with maturities less than 12 months from the balance sheet date, which are classified as current assets. Held-to-maturity investments are carried at amortized cost using the effective interest method.

The fair value of the held-to-maturity investments are based on the current bid prices offered by the dealers/issuers.

(d) Property, plant and equipment

Property, plant and equipment are stated at historical cost less accumulated depreciation and impairment losses. Historical cost includes expenditure that is directly attributable to the acquisition of the items.

Subsequent expenditure is charged to the asset's carrying amount only when it is probable that future economic benefits associated with the item will flow to the College and the cost of the item can be measured reliably. All other repairs and maintenance costs are expensed in the income statement during the financial period in which they are incurred.

Depreciation of property, plant and equipment is calculated to allocate cost to their residual values over their estimated useful lives, as follows:

Furniture and equipment	5 years
Computers	3⅓ years

The assets' residual values and useful lives are reviewed, and adjusted if appropriate, at each balance sheet date.

An asset's carrying amount is written down immediately to its recoverable amount if the asset's carrying amount is greater than its estimated recoverable amount.

(e) Cash and cash equivalents

Cash and cash equivalents include cash in hand, deposits held at call with banks, other short-term highly liquid investments with original maturities of three months or less, and bank overdrafts. Bank overdrafts are shown within borrowings in current liabilities on the balance sheet.

(f) Employee benefits

The company has established a mandatory provident fund scheme ("MPF Scheme") in Hong Kong. The assets of the MPF Scheme are held in separate trustee-administered funds. Both the company and the employees are required to contribute 5% of the employees' relevant income, subject to a maximum of HK$1,000 per employee per month. The company's contributions to the MPF Scheme are expensed as incurred.

(g) Trade and other receivables

Trade and other receivables are recognized initially at fair value and subsequently measured at amortized cost using the effectively interest methods, less provision for impairment losses for bad and doubtful debts. Short term receivables with no stated interest rate and the effect of discounting being immaterial that are measured at their original invoice amount less provision for impairment losses for bad and doubtful debts, if any.

A provision for impairment of trade and other receivables is established when there is objective evidence that the company will not be able to collect all amounts due according to the original terms of receivables. The amount of the provision is the difference between the assets's carrying amount and the present value of estimated future cash flows, discounted at the effective interest rate. The amount of the provision is recognized in the income statement.

(h) Trade and other payables

Trade and other payables are initially measured at fair value and after initial recognition, at amortized cost, except for short-term payables with no stated interest rate and the effect of discounting being immaterial, that are measured at their original invoice amount.

The company has established a mandatory provident fund scheme ("MPF Scheme") in Hong Kong. The assets of the MPF Scheme are held in separate trustee-administered funds. Both the company and the employees are required to contribute 5% of the employee's relevant income, subject to a maximum of HK$1,000 per employee per month. The company's contributions to the MPF Scheme are expensed as incurred.

3. FINANCIAL RISK MANAGEMENT

(a) Financial risk factors

The College's activities expose it to credit risk and liquidity risk as described below. The College's overall risk management policies focus on minimizing the potential adverse effects on the College by closely monitoring the individual exposure as follows:

(1) Credit risk

The College has no significant concentrations of credit risk. It has policies in place to ensure that income from membership subscription and other events are primarily collected in cash.

(2) Liquidity risk

The College's policy is to regularly monitor current and expected liquidity requirements to ensure that it maintains sufficient reserves of cash and cash equivalents to meet its liquidity requirements in the short and longer term.

(b) Fair value estimation

The nominal value less estimated credit adjustments of account receivables and payables are assumed to approximate their fair values.

4. CRITICAL ACCOUNTING ESTIMATES AND JUDGEMENT

The College's management makes assumptions, estimates and judgments in the process of applying the company's accounting policies that affect the assets, liabilities, income and expenses in the financial statements prepared in accordance with HKFRSs. The assumptions, estimates and judgments are based on historical experience and other factors that are believed to be reasonable under the circumstances. While the management reviews their judgments, estimates and assumptions continuously, the actual results will seldom equal to the estimates.

Critical judgments in applying the company's accounting policies

Certain critical judgments in applying the company's accounting policies are set out as follows:

Held-to-maturity investments

The College follows the guidance of HKAS 39 on classifying non-derivative financial assets with fixed or determinable payments and fixed maturity as held-to-maturity. This classification requires significant judgment. In making this judgment, the College evaluates its intention and ability to hold such investments to maturity.

If the College fails to keep these investments to maturity other than for specific circumstances explained in HKAS 39, it will be required to reclassify the whole class as available-for-sale. The investments would therefore be measured at fair value and not amortized cost.

If the class of held-to-maturity investments is tainted, the fair value would decrease by HK$55,889, with a corresponding entry in the fair value reserve.

5. ANNUAL SCIENTIFIC MEETING

	2005 HK$	2004 HK$
Income	1,311,300	1,083,850
Less : Cost and expenses	851,390	589,363
	459,910	494,487
Less : 50% profit shared with ISAP	229,955	247,243
	229,955	247,244

From the year ended 31st December, 2005, the combined scientific meeting was organized jointly with The Society of Anaesthesia of Hong Kong ("SAHK"). The College and the SAHK agreed to share the income and expenses of the meeting equally.

6. INSTITUTE OF CLINICAL SIMULATION

	2005 HK$	2004 HK$
Income	88,000	99,359
Less : Cost and expenses	81,540	86,141
	6,460	13,218
Less : 50% profit shared with the North District Hospital	3,230	6,609
	3,230	6,609

The Institute of Clinical Simulation was organized jointly with the North District Hospital ("the Hospital"). The college and the hospital agreed to share the income and expenses of ICS equally.

7. COURSES

	2005 HK$	2004 HK$
Income	235,200	307,600
Less : Cost and expenses	104,742	189,329
Surplus	130,458	118,271

8. EXAMINATION

	2005 HK$	2004 HK$
Income	582,220	755,300
Less : Cost and expenses	503,193	521,828
Surplus	79,027	233,472

9. STAFF COSTS

	2005 HK$	2004 HK$
Salaries	270,720	289,225
Mandatory provident fund	11,317	10,300
	282,037	299,525

10. FIXED ASSETS

	Furniture and Equipment HK$	Computers HK$	Total HK$
At 1st January, 2004			
Cost	225,464	25,869	251,333
Accumulated depreciation	199,952	25,869	225,821
Net book amount	25,512	---	25,512
At 31st December, 2004			
Opening net book amount	25,512	---	25,512
Additions	---	56,248	56,248
Depreciation	12,756	16,874	29,630
Closing net book amount	12,756	39,374	52,130
At 31st December, 2004			
Cost	225,464	82,117	307,581
Accumulated depreciation	212,708	42,743	255,451
Net book amount	12,756	39,374	52,130
At 31st December, 2005			
Opening net book amount	12,756	39,374	52,130
Additions	1,280	---	1,280
Depreciation	13,012	16,874	29,886
Closing net book amount	1,024	22,500	23,524
At 31st December, 2005			
Cost	226,744	82,117	308,861
Accumulated depreciation	225,720	59,617	285,337
Net book amount	1,024	22,500	23,524

11. HELD-TO-MATURITY DEBT SECURITIES

	2005 HK$	2004 HK$
Held-to-maturity investments, at amortized cost:		
Unlisted debt securities in Hong Kong traded on inactive markets	1,099,833	1,099,230
Less: non-current portion	-------	1,099,230
Current portion	1,099,833	-------
Certificate of deposit (non-current portion)	5,100,000	-------
	6,199,833	2,198,460
Fair value of held-to-maturity investments	6,143,944	1,112,886

The held-to-maturity debt securities represent HK$1,100,000 HKSAR Government Bond ("Bond") and HK$5,100,000 HSBC Certificate of deposit ("CD") which was acquired on 26th July, 2004 and 31st October, 2005 respectively. For the Bond, interest is calculated based on the effective interest rate at 2.17% per annum on the principal amount of the bond up to the date of maturity on 24th July, 2006. For the CD, interest is calculated based on the effective interest rate at 3.4% per annum on the principal amount of the CD up to 30th April, 2007.

12. TAXATION

The college is exempt from Hong Kong profits tax by reason of being a charitable institution.

13. APPROVAL OF ACCOUNTS

The accounts set out on pages 152 to 160 were approved by the Council at the 17th Annual General Meeting on Friday, 16th June 2006.

Approved Formal Projects

NG, Vincent	Comparison of Forced air-warming and Electric warming blanket for Maintenance of Body Temperature in Total Knee Replacement.
SO, Alpha	COX-3: biochemistry, pharmacology, relevance to paracetamol and future prospects
HUSSAIN, Assad	Intranasal ketamine premedication in children
CHANG, HK	Case report: Anaesthetic management of a child with Spinal Muscular Atrophy, Obstructive Sleep Apnoea Syndrome and Asthma
MA, Elina	Report and Mini Review of Management of a Case of Placenta Increta in a Patient with Placenta Praevia Type IV
CHAN, Stanley	Reliability using finger palpation of pilot balloon in estimating endotracheal tube cuff pressure: comparison between Portex® Soft-Seal and Curity®

Peter Kam's Courses ….

REVISION TUTORIAL COURSE IN ANAESTHESIOLOGY 2006

This year Professor Peter Kam will again be running two REVISION TUTORIAL COURSES:

	Basic science in Anesthesiology	***Clinical Anaesthesiology***
Time	20 November - 1 December, 2006	2 December - 9 December 2006
Contents	2 weeks "fulltime" course containing lectures, tutorials and mock viva	7½ -day course with interactive lectures, tutorials and mock viva sessions
Target audience	Trainees preparing for the Intermediate Fellowship Examination	Trainees preparing for the Final Fellowship Examination
Venue	Queen Elizabeth Hospital	Queen Elizabeth Hospital
Maximum number	30	30
Fee	HK$ 2,000 Registered HKCA trainee HK$ 4,000 for non-HKCA member	HK$ 1,500 Registered HKCA trainee HK$ 3,000 for non-HKCA member
Deadline for application	4th November, 2006	11th November 2006

Details are subjected to change without prior notice.

If you have any queries concerning the course, please contact Mr. Daniel Tso, Administrative Executive at 2871 8833. Further information can also be obtained at the College website www.hkca.edu.hk.

Drs CH KOO and Douglas FOK,
Course Coordinators, Department of Anaesthesia, Queen Elizabeth Hospital

Board of Education

Supervisor of Training meeting

The Board of Education is going to hold a Supervisor of Training (SOT) meeting in the coming Annual Scientific Meeting in Anaesthesiology 2006 on 18 November 2006. The aim of this meeting is to collect feedbacks or questions from trainees and/or SOTs related to the anesthesia vocational training and documenttation.

Please forward your comments and opinions on the Vocational Training Guide or the eLogBook to the Training Officer (*via the Administrative Executive*) of the College or the SOT of your hospital by mail, e-mail or fax.

Revision Tutorial Courses 2006

This year, the revision tutorial courses hosted by Professor Peter Kam will start on 20 November for the Intermediate Fellowship Examination candidates and 2 December for Final Fellowship Examination candidates. Applications for both courses have commenced. For details, please see the advertisement in this issue of the Bulletin, the College website, ask your SOT or the Administrative Executive of the College.

New Diploma of Pain Management Program

The Board of Pain Medicine has announced a new vocational training program and curriculum towards the *Diploma of Pain Management*. The new program and curriculum will start on 2 January 2008. For detail of the changes, please see the Board of Pain Medicine announcement in this issue of the HKCA Bulletin or the HKCA website.

CME/CPD

The Academy has passed the notion that starting from 1 January 2008, no more than 75 points can be awarded for passive participation in a cycle. It is also proposed that from 1 January 2011, the limit for passive participation will be 60. Fellow consultation letters have already been sent from the College. Forum communication focused to private hospitals by HKAM Education Committee will be planned in due course for further consultation.

Current list of accredited CPD activities by HKCA:

Self-study (Remarks: College approved self study program and or self assessment program)

Chairing/Presenting at 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/Undergraduate Teaching (Undergraduate and postgraduate teaching may not be accepted as a form of CME/CPD. Consideration can be given to development of teaching materials.)

Conducting Examinations

Quality Assurance and Medical Audits

Mortality & Morbidity Meetings (Participation in mortality & morbidity meetings is accepted as a form of CME/CPD)

Quality assurance activities (such as clinical/surgical review and audit, clinical governance, peer review of operative practice, activities that examine and evaluate the clinical care of patients, are accepted as a form of CME/CPD)

Postgraduate Courses (Attending a course leading to postgraduate qualification can be accepted as a form of CME/CPD)

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

Reviewers of Hong Kong Medical Journal and other Indexed Journals (*e.g.* Participation in reviewing articles submitted to HKMJ and other indexed journals is accepted as a form of CME/CPD)

Board of Pain Medicine

Diploma of Pain Management Program

With the development of pain medicine in Hong Kong, the Board of Pain Medicine felt that it is the correct time to review the training curriculum and guidelines for the *Diploma of Pain Management (HKCA) program*. In November 2005, the Board of Pain Medicine established a Review Subcommittee for the Training Program and Examination. They were lead by Drs MC Chu and TW Lee, respectively. The Boards of Education and Examination were consulted during the review. The subcommittees reported on the recommended changes in May 2006. These were approved by the Board and was endorsed by the Council on 30 August 2006. The Board of Pain Medicine (HKCA) would like to announce that a new training program towards the *Diploma of Pain Management* will commence on 2 January 2008. The new examination format will take place in October 2008.

The following table summarizes the major changes that will take place after the new changes take effect.

	Current program	New program
Training		
1	**DPM trainee does not need to attend and log pain related clinical activities in other specialties and disciplines.**	**DPM trainee has to attend and log pain related clinical activities in other specialties and disciplines. The trainee has to attend a total of not less than 12 sessions, in not less than 2 different specialties or disciplines, and not less than 3 sessions in each specialties or disciplines (PM1.V2, Clause 6).**
2	**Presently the DPM trainee has two years to complete his/her one-year vocational training, and has to complete the project and examination within three years of completion of accredited training.**	**The examination should be passed and the project has to be completed and approved within 3 years of commencement, *instead of completion*, of training. Failing that, the trainee is required to rejoin the entire training program (PM1.V2, Clause 8.4).**
Examination		
3	**No viva.**	**A One hour viva examination consisting of 2 parts, with 2 examiners at each table will be held (PM5.V2, Clause 4.3).**
4	**Paper I: consists of eight short questions, six to be answered within one hour.**	**Paper I: consists of four compulsory questions and four optional short questions, six to be answered within one hour.**
	Paper II: consists of three clinical scenarios each of them has three questions, two scenarios to be answered within one hour.	**Paper II: consists of two compulsory clinical scenarios each of them has three questions to be answered within one hour (PM5.V2, Clause 4.3).**

PP Chen
Chairman, Board of Pain Medicine

THE HONG KONG COLLEGE OF ANAESTHESIOLOGISTS GUIDELINES ON TRAINING FOR THE DIPLOMA IN PAIN MANAGEMENT

INTRODUCTION

The Hong Kong College of Anaesthesiologists ("the College") conducts organized post-fellowship training in pain management leading to the Diploma in Pain Management (Dip Pain Mgt (HKCA)).

1. Entry requirements:
   - 1.1 In possession of FHKCA *or,*
   - 1.2 Current trainees who have completed at least 5 years of vocational training in Anaesthesiology recognised by the College, and have passed the final examination of Anaesthesiology by the College or other equivalent colleges.
2. Applicants must register and obtain approval from the College before commencing training. Training prior to approval may be considered upon the discretion of the College for up to 3 months.
3. Application forms are available by writing to the college office or can be downloaded from the college website. The College will inform the applicants upon receiving the completed application form on the commencement date of pain management training.
4. Duration of training:
   - 4.1 Each trainee shall have not less than 12 months of full time training in a post approved by the College for Dip Pain Mgt (HKCA) training. The training period may overlap with the vocational training for Anaesthesiology.
   - 4.2 Full time training is defined as a minimum of 7 sessions per week per trainee of clinical or academic exposure to pain management exclusively. Each session shall be not less than 3 hours.
   - 4.3 At least 6 months of training shall be completed after obtaining FHKCA.
   - 4.4 In the event of interrupted training,
     - 4.4.1 Training shall be conducted in blocks of not less than 3 months.
     - 4.4.2 The training programme shall be completed within 24 months of the commencement date. Trainees unable to complete the training programme may be required to extend their training period.
5. The number of patients managed and logged by each trainee within the training programme shall be not less than:
   - 5.1 One hundred (100) new patients with chronic cancer or non-cancer pain. New patients are defined as being new to the trainee AND to the service. Each patient shall be considered new to one trainee only; *and*
   - 5.2 Two hundred (200) patients with acute or postoperative pain.
   - 5.3 Trainees unable to achieve the caseload may be required to extend their training programme upon the discretion of the College.
6. The trainee is required to attend and log pain related clinical activities of no less than 2 of the following specialties, for not less than 12 sessions during their training programme. Trainees unable to attend these activities may be required to extend their training programme upon the discretion of the College.
   - 6.1 Psychiatry
   - 6.2 Neurology
   - 6.3 Neurosurgery
   - 6.4 Oncology
   - 6.5 Palliative Medicine
   - 6.6 Rehabilitation Medicine
   - 6.7 Rheumatology
   - 6.8 Clinical Psychology
   - 6.9 Physiotherapy
   - 6.10 Occupational Therapy
7. Trainees shall keep a logbook for documentation of all training activities. The logbook has to be endorsed by the Supervisor of Training upon completion of training, and submitted to the College upon application for the Diploma.
8. Trainees are required to submit a project related to pain management.
   - 8.1 The topic shall be related to acute, chronic or cancer pain management.
   - 8.2 The project can be in the form of a clinical trial, a case report with mini-review, or other format acceptable to the college.
   - 8.3 Prospective approval is required from the College. Projects presented for accreditation towards other qualifications, such as Anaesthesiology training, shall not be considered for accreditation towards the Dip Pain Mgt (HKCA).
   - 8.4 The project must be completed and approved by the College within 3 years of commencement of training. Trainee unable to have their project approved may be required to rejoin the entire training again.
9. Trainees shall submit a training progress report upon completion of every 6 months of accredited training, including the completion of training at 12 months. The training progress report has to be endorsed by the Supervisor of Training.
10. Trainees must satisfy the examiners in a Dip Pain Mgt (HKCA) examination conducted by the College
    - 10.1 Only trainees who are registered and commenced training in Dip Pain Mgt (HKCA), have submitted all required documents, and have paid the appropriate fee are eligible to present for the examination.
    - 10.2 The format of the examination shall be determined by the Council of the College on recommendation of the Board of Examinations.
    - 10.3 The examination must be passed within 3 years of commencement of training. Otherwise, these

candidates have to rejoin the entire training programme again.

11. The accredited training time, caseload, attendances, examination passed and the approved project will lapse 3 years after commencement of the approved training period in pain medicine. Doctors failing to obtain the diploma within 3 years of commencement of training will have to rejoin the entire training programme and repeat all the training requirements again.

PM1.V2 Approved by the Council, 30 August 2006

THE HONG KONG COLLEGE OF ANAESTHESIOLOGISTS ADMINISTRATIVE INSTRUCTIONS ON EXAMINATION FOR THE DIPLOMA IN PAIN MANAGEMENT

1. INTRODUCTION

Persons who wish to be educated as a diploma holder in Pain Management must undertake a structured programme of training and assessment. This document sets out the examination requirements for assessment of trainees for the diploma in pain management.

2. ELIGIBILITY

2.1 The Diploma in Pain Management of the Hong Kong College of Anaesthesiologists may be granted to a medical graduate whose credentials are acceptable to the Council and who complies with the following regulations.

2.2 Candidates for the Diploma will be required to pass the examination for the Diploma in Pain Management and complete the specified training. (Refer PM1.V2 HKCA 2006)

2.3 Application to present for the examination must be made on the approved form, together with the required documents and the prescribed fee, and shall be received by the Secretary at least one month before the commencement date of the examination.

2.3.1 Each application shall be considered by the Board of Examinations.

2.3.2 A candidate may appeal against a decision of the Board to the Appeal Committee.

2.3.3 A candidate approved by the Board, or by the Council on appeal, may present before the appointed examiners.

2.3.4 A candidate whose withdrawal is received by the Secretary, in writing, before the published date on which entries close will be refunded the examination fee, less an administrative charge.

2.3.5 A candidate whose entry has been accepted and who withdraws from the examination on or after the date on which entries close, or who fails to attend at the examination, shall forfeit the examination fee.

2.3.6 The Board may, on production by the candidate of satisfactory evidence of a medical or compassionate nature, refund a proportion of the fee paid, provided that the refund is applied for within seven days of withdrawal or failure to appear.

2.3.7 The Court of Examiners for the Examination may refuse to proceed with the examination of a candidate, infringes the regulations, or is considered by the examiners to be guilty of behaviour prejudicial to the conduct of the examinations.

3. THE EXAMINATION

3.1 Candidates who apply for admission to the Examination must

3.1.1 have registered with HKCA as a trainee for the Diploma in Pain Management.

3.1.2 have completed six months of training in an accredited training position for the Diploma in Pain Management.

3.2 All candidates applying to sit the Examination must be in an approved training post for Pain Management or within three years of being in a training post in Pain Management.

3.3 Although a candidate may pass the Examination, the Diploma in Pain Management of the Hong Kong College of Anaesthesiologists will not be awarded until the full training requirements for the Diploma in Pain Management are fulfilled.

4. FORMAT OF EXAMINATIONS

4.1 Examinations will be held at times to be determined by the Council. All examinations will be held in Hong Kong and examination fees shall be determined by the Council.

4.2 The examination questions will be set according to the curriculum. (Refer PM4 HKCA)

4.3 The format of the Examination will be two written papers and one 1 hour viva:

4.3.1 Paper I: consists of four compulsory questions and four optional short questions, six to be answered within one hour.

4.3.2 Paper II: consists of two compulsory clinical scenarios each of them has three questions to be answered within one hour.

4.3.3 A One hour viva examination consisting of 2 parts, with 2 examiners at each table will be held.

5. ADMINISTRATION OF THE EXAMINAT-IONS

The administration of the examinations will be by the Board of Examinations who will

5.1 Appoint a Court of Examiners for the examination.

5.2 Conduct the examinations.

5.3 Notify successful and unsuccessful candidates.

5.4 Provide such information to unsuccessful candidates as will enable them to attempt future examinations with an improved chance of success.

5.5 Recommend the names of examiners to the Council.

6. EXAMINERS

6.1 Examiners shall be recruited from qualified persons who, in the opinion of the Council, are themselves sufficiently well informed to be capable of evaluating the knowledge of the candidates.

6.2 Appointment as an examiner shall be by the Council on the recommendation of the Board of Examinations.

6.3 Appointment shall be for a term of two years, and appointees shall be eligible for reappointment up to a total of six years, subject to the Council's discretion.

6.4 There shall be at least one external examiner participating in each examination.

6.5 Appointment of external examiners shall be by the Council on the recommendation of the Pain Management Committee of HKCA.

7. REPRESENTATIONS AND APPEALS

7.1 A candidate, or any person on behalf of that candidate, wishing to make representations with regard to the conduct of an examination, or to appeal against any result, must address such representation or appeal to the Appeal Committee in writing within one week of the end of that particular examination.

7.2 The Appeal Committee will make recommendation to the Council, whose decision shall be final.

PM5.V2 Approved by the Council, 30 August 2006

Future Meetings:

HONG KONG
20-21 October, 2006

SCIENTIFIC SYMPOSIUM ON EMERGENCY MEDICINE 2006
Venue: Hong Kong Academy of Medicine Jockey Club Building
Contact: Hong Kong Academy of Medicine; Ms Lenora Yung / Ms Jessie Chow; Phone: (852) 2871 8841 / 2871 8787; Fax: (852) 2871 8898; Email: lenora@hkam.org.hk / jessie@hkam.org.hk

SINGAPORE
6-10 November 2006

12th ASIAN AUSTRALASIAN CONGRESS OF ANAESTHESIOLOGISTS (AACA)
Theme: The Art and Science of Anaesthesiology.
Venue: Suntec Singapore Convention and Exhibition Centre.
Contact: A/Prof Dr Yew-Weng CHAN, Organising Chairman. Tel: (65) 6330 6834 Fax: (65) 6336 2123 Email: aaca2006@pacificworld.com; Website: www.aaca2006.com

HONG KONG
17-19 November, 2006

ANNUAL SCIENTIFIC MEETING 2006
Theme: Cardiothoracic and Vascular Anaesthesia
Venue: Hong Kong Convention and Exhibition Centre
Contact: ASM 2006 Secretariat, c/o International Conference Consultants, Ltd. Unit 301, 3/F The Centre Mark, 287-299 Queen's Road Central, Hong Kong, Tel: (852) 25599973; Fax: (852) 25479528; Email: asm2006@icc.com.hk; Website: www.hkca.edu.hk/asm2006.htm

Chepstow Wales, UK
23-24 November, 2006

ANNUAL SCIENTIFIC MEETING OF THE UK SOCIETY FOR INTRAVENOUS ANAESTHESIA
Venue: Marriott St. Pierre Hotel & Country Club.
Contact: Dr William McFadzean, Consultant Anaesthetist. Morriston Hospital, Swansea SA6 6NL. Tel: +44 (0)1792 703279 Email: meetings@sivauk.org
Website: www.sivauk.org/StPierreChepstow.htm

HONG KONG
2-3 December, 2006

HONG KONG COLLEGE OF ORTHOPAEDIC SURGEONS - INTERNATIONAL SYMPOSIUM ON DISASTER MEDICAL RESPONSE
Venue: Hong Kong Academy of Medicine Jockey Club Building
Contact: Symposium Secretariat The Hong Kong College of Orthopaedic Surgeons Room 905, 9/F, Hong Kong Academy of Medicine Jockey Club Building 99 Wong Chuk Hang Road Aberdeen, Hong Kong Tel: (852) 2871 8722 Fax: (852) 2873 4077 E-mail: hkcos@hkcos.org.hk Website: www.hkcos.org.hk

Kathmandu
NEPAL
22 - 24 February, 2007

7TH CONGRESS OF THE SOUTH ASIAN CONFEDERATION OF ANAESTHESIOLOGISTS
Contact: Clinic-Health Care Centre (Pvt.) Ltd. Naya Bazar - Balaju, Ring Road Crossing, P.O. Box 4602, Kathmandu, Nepal. Tel: 977 1 4351627 Fax: 977 1 4351654 Email: info@7thsacacongress2007.com Website: 7thsacacongress2007.com

HONG KONG
14-18 March, 2007

5TH INTERNATIONAL CONFERENCE ON PAIN CONTROL AND REGIONAL ANAESTHESIA
Venue: Sheraton Hotel and Towers TST
Contact: OPTIONS Fax: 44 (0)870 0132940; www.optionsglobal.com

Melbourne
AUSTRALIA
26 - 29 May, 2007

2007 ANZCA ASM
Theme: "Perioperative Medicine - Evidence and Practice".
Contact: Ms Juliette Mullumby. ANZCA, 630 St Kilda Road, Melbourne 3004. Tel: 03 9510 6299 Fax: 03 9510 6786 Email: jmullumby@anzca.edu.au
Website:www.anzca2007asm.com

Fellowship Examinations 2007

Intermediate Fellowship Examinations

Examination Fee: $ 6,000

February / April	**Date**
Written	23 February 2007 (Fri)
Oral	13/14 April 2007 (Fri/Sat)
Closing Date	12 Jan 2007 (Fri)

June / August	**Date**
Written	22 June 2007 (Fri)
Oral	10/11 Aug 2007 (Fri/Sat)
Closing Date	11 May 2007 (Fri)

Final Fellowship Examination in Anaesthesiology

Examination Fee: $ 9,500

March / May	**Date**
Written	23 March 2007 (Fri)
Oral/OSCE	11-13 May 2007 (Fri-Sun)
Closing Date	9 February 2007 (Fri)

July / September	**Date**
Written	20 July 2007 (Fri)
Oral/OSCE	7-9 Sept 2007 (Fri-Sun)
Closing Date	8 June 2007 (Fri)

Application forms are available from Supervisors of Training and HKCA Office.

Board of Examination

Intermediate Fellowship Examination July/August 2006

CHAN, Lai Mei
CHAN, On Yi
CHIANG, Chi Sum James
HO, Chi Yu
JOENG, Kin Ying Alice
KANDAMBY, Darshana Hewa
LAU, King Yin
LEE, Wai Ping
LEUNG, King Hei
NG, Kwun Tung
SUN, Chi Hong Nicholas
TAI, Man Ting, Florence
TAM, Tak King Dhugal
WAT, Chun Yin
WONG, Chi Pan
WONG, Hoi Kay Tiffany
WONG, Tsz Kin
YUNG, Hoi Ling

Eighteen out of 21 candidates who took part in this sitting passed the examination. The College is grateful to Dr Leslie Gemmell of RCA, for his assistance as external examiner during the examination. The Prize of the Intermediate Fellowship Examination was awarded to Dr CHAN, Lai Mei.

Final Fellowship Examination July/September 2006

KAM, Hau Chi
KONG, Kau Fung Vincent
LAM, Chi Shan
LEUNG, Kam Kin
WONG, Lai Sze Grace

Successful candidates with the Final Examiners
From Left to right: Drs Brian Sweeney (RCA), Vincent Kong, KK Leung, Geoffrey Mullins (ANZCA), Geoffrey Lam, Grace Wong and PT Chui (Chairman).

Five out of 10 candidates passed the examination. The HKCA Final Fellowship Examination Prize was awarded to Dr WONG Lai Sze, Grace. The College is grateful to Dr Brian Sweeney of RCA, and Dr Geoffrey Mullins of ANZCA for their assistance as external examiners during the examination.

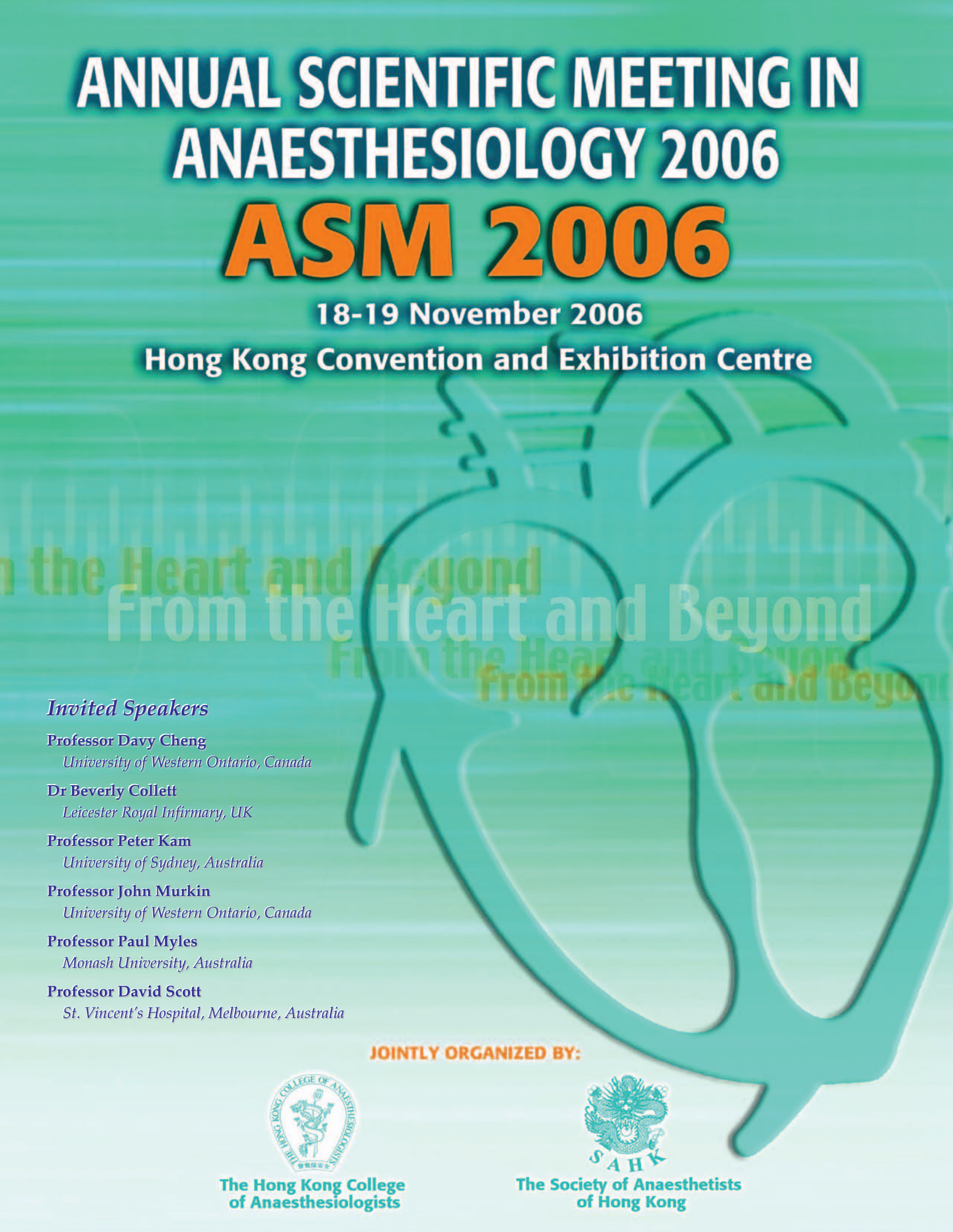
Dr PT Chui
Chairman
Board of Examination

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

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14th - 18th March 2007

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HONG KONG

5TH INTERNATIONAL CONFERENCE on Pain Control and Regional Anaesthesia

Abstract submission deadline : 22nd December 2006

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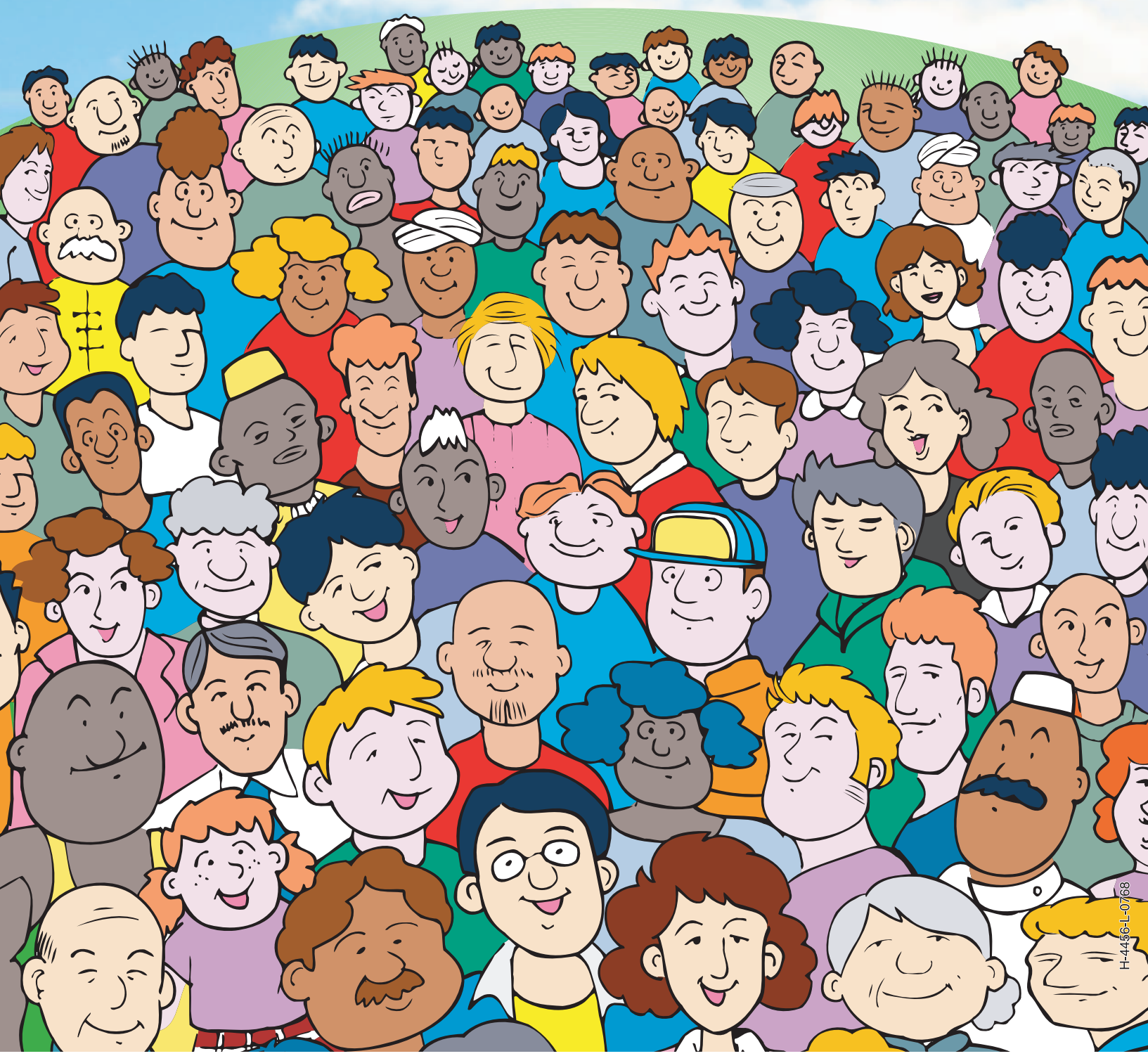

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Assistant	Cherry WONG

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TY Chan	

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Joseph Lui	Theresa Hui
Agnes Cheng	

Organizer, Basic Science Course: CH Koo, Aaron Lai
Organizers, Clinical Anaesthesiology Courses (Informative course and Crash course): Douglas Fok and Eric So
Chairman, The Institute of Clinical Simulation: PP Chen (Chairman)