SOUVENIR PROGRAMME & ABSTRACT BOOK

5th National Conference on Intensive Care

DATE

22 – 24 JUNE 2007

VENUE

SHANGRI-LA HOTEL
KUALA LUMPUR, MALAYSIA

Organised by

Intensive Care Section
Malaysian Society of Anaesthesiologists

In conjunction with

Ministry of Health Malaysia
(Anaesthetic and Intensive Care Services)

CONTENTS

MESSAGE FROM THE CHAIRPERSON, INTENSIVE CARE SECTION, MSA & CHAIRPERSON, ORGANISING COMMITTEE, 5TH NCIC

I am privileged to have been associated with the National Conference on Intensive Care (NCIC) since its inception five years ago and observed its growth over the years. It is organised to be a truly scientific meeting without the pomp and frills. I like to think that the NCIC is well received because it has achieved its goal of providing the most up-to-date information on the care of the critically ill. The topics chosen have been current and relevant to the day-to-day management of patients in the ICU. The speakers have been carefully selected to ensure high quality of delivery.

I would think that the NCIC not only promotes continuous professional development, it has also enhanced the professional standing of intensive care in the medical fraternity in this country. More clinicians are now pursuing intensive care as a subspecialty and this is indeed very encouraging. The development of a critical mass of intensivists is crucial in the advancement of intensive care and towards improving outcome of the critically ill.

The Organising Committee would like to thank you for your continued support and wish you a fruitful meeting.

Dr Ng Siew Hian

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ACKNOWLEDGEMENTS

The Organising Committee of the 5th National Conference on Intensive Care expresses its deep appreciation to the following for their support and contributions:

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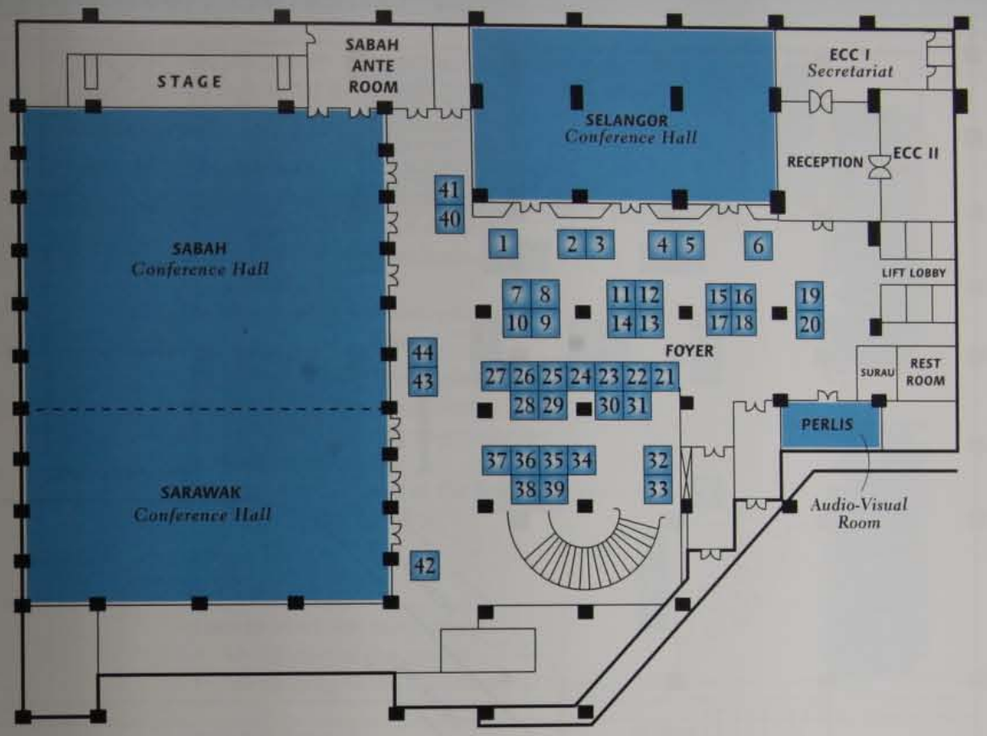

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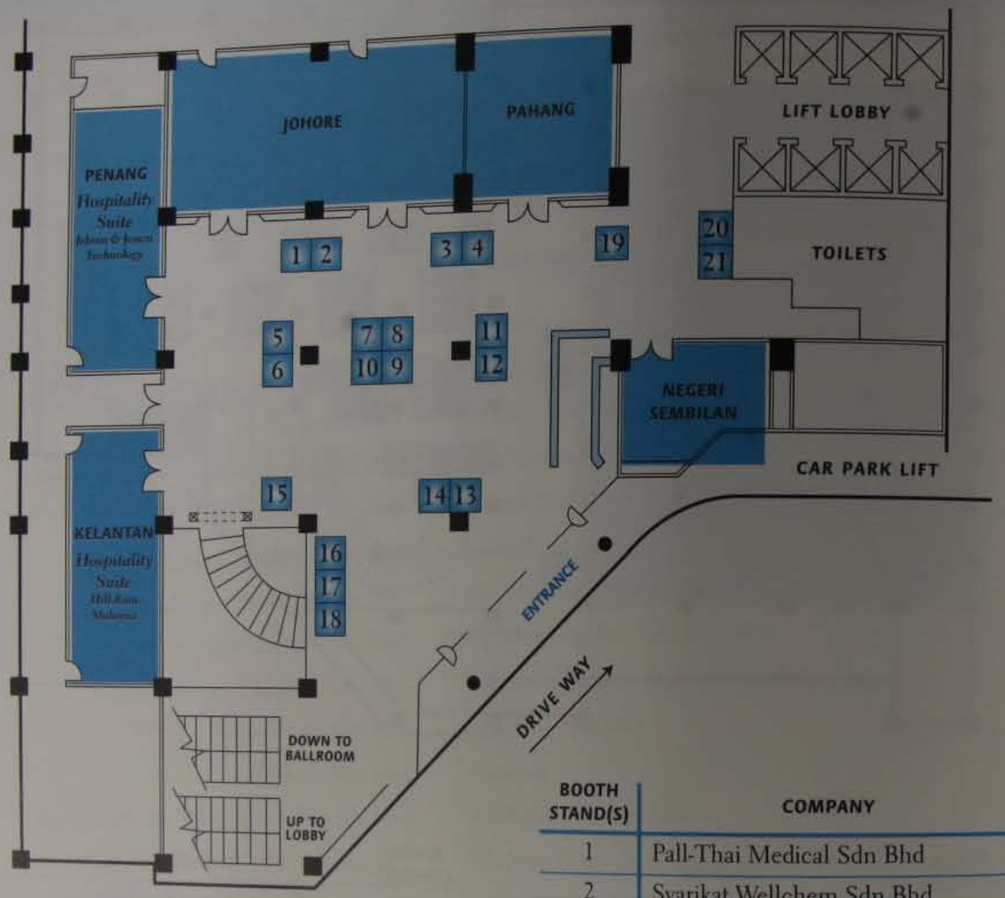

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PRE-CONFERENCE WORKSHOPS

21 JUNE 2007, THURSDAY

1300 – 1700	**WORKSHOP 1 – BRONCHOSCOPY** **"The Use Of Bronchoscopy In Intensive Care"**	KEDAH / SELANGOR
1300 – 1400	REGISTRATION	
1400 – 1445	**Workshop 1** *Coordinator:* Lim Boon Kor • Anatomy of the bronchial tree • Indications of bronchoscopy • Chest X rays • Hands-on bronchoscopies and bronchoscopy simulator	
1445 – 1530	**Workshop 2** *Coordinator:* Nor'Azim Mohd Yunos • Preparation of the patient • Monitoring during bronchoscopy • Equipment • Care and cleaning of the bronchoscope • Safety of the operator	
1530 – 1600	TEA	
1600 – 1645	**Workshop 3** *Coordinator:* Toh Khay Wee • Ventilation of the patient • Monitoring of the percutaneous tracheostomy	

1400 – 1730	**WORKSHOP 2** **"Improving Clinical Outcome In Intensive Care Blood Purification"** *(Sponsored by Gambro/T-Medic)*	PERAK
1400 – 1430	REGISTRATION Video Presentation "Discover flexibility of the next generation of blood purification system – Prismaflex" *(Speaker from T-Medic Sdn Bhd)*	
1430 – 1445	Welcome Speech Shanti Rudra Deva	
1445 – 1530	Renal replacement therapy in critically ill patient with sepsis: Does dosing affect survival and renal recovery Ian K S Tan	
1530 – 1600	Case studies Ian K S Tan	
1600 – 1615	TEA	
1615 – 1700	Liver support in the intensive care unit Alexander Chiu	
1700 – 1730	Q & A	

SCIENTIFIC PROGRAMME SUMMARY

Time \ Date	22 June 2007 Friday	23 June 2007 Saturday	24 June 2007 Sunday
0800 – 1730	REGISTRATION		
0830 – 0900	OPENING CEREMONY	PLENARY 2	PLENARY 4
0900 – 0930	PLENARY 1		
0930 – 1000		PLENARY 3	PLENARY 5
1000 – 1030	TEA / TRADE EXHIBITION	TEA / TRADE EXHIBITION	TEA / TRADE EXHIBITION
1030 – 1100	SYMPOSIUM 1 Respiratory (1)	SYMPOSIUM 5 Sepsis	SYMPOSIUM 10 Neurologic
1100 – 1130			
1130 – 1200	SYMPOSIUM 2 Paediatrics	SYMPOSIUM 6 Paediatrics	SYMPOSIUM 11 Intensive Care For Nurses (2)
1200 – 1230			
1230 – 1330	LUNCH FRIDAY PRAYERS	LUNCH	LUNCH
1330 – 1430			
1430 – 1500	SYMPOSIUM 3 Haemodynamics	SYMPOSIUM 7 Respiratory (2)	
1500 – 1530			
1530 – 1600	SYMPOSIUM 4 Miscellaneous	SYMPOSIUM 8 Metabolic / Renal	
1600 – 1630			
1630 – 1700	TEA / TRADE EXHIBITION	TEA / TRADE EXHIBITION	
1700 – 1730	TEA SYMPOSIUM	FREE PAPER PRESENTATIONS	
1730 – 1800			
	INTERACTIVE SESSION (I) Haemodynamics	INTERACTIVE SESSION (III) Respiratory	
	INTERACTIVE SESSION (II) Data Interpretation	SYMPOSIUM 9 Intensive Care For Nurses (1)	

PROGRAMME
22 JUNE 2007, FRIDAY

	Sabah	Sarawak	Selangor
0800 – 1730	REGISTRATION		
0830 – 0900	**OPENING CEREMONY**		
0900 – 1000	**PLENARY 1** *Chairperson: Tai Li Ling* Ethics in intensive care practice *Dermot Phelan*		
1000 – 1030	TEA / TRADE EXHIBITION		
1030 – 1230	**SYMPOSIUM 1** **Respiratory (1)** *Chairpersons: Suresh Rao / Ahmad Shaltut Othman* • Ventilator induced lung injury (pg 10-11) *Mohd Basri Mat Nor* • Acute lung injury – Oxygenation strategies (pg 12) *Kwek Tong Kiat* • Pharmacologic therapy for ARDS – Has it failed? (pg 13-14) *Nik Abdullah Nik Mohamad* • Patient-ventilator synchrony (pg 15) *Andrew Bersten*	**SYMPOSIUM 2** **Paediatrics** *Chairperson: Teh Keng Hwang* • Pharmacological approaches to the ventilated paediatric patient (pg 16) *Tang Swee Fong* • Current practices in transfusion medicine in the PICU (pg 17) *Stephen Jacobe* • Support for the failing heart (pg 18) *Loh Tiee Foong*	**INTERACTIVE SESSION (I)** **Haemodynamics** *Moderators: Nor'Azim Mohd Yunos / Mahazir Kassim*
1230 – 1430	LUNCH / Friday Prayers		
1430 – 1630	**SYMPOSIUM 3** **Haemodynamics** *Chairpersons: Nor'Azim Mohd Yunos / Wan Nasruddin Wan Ismail* • Predicting fluid responsiveness in the critically ill (pg 19) *Tai Li Ling* • Management of systolic and diastolic heart failure (pg 20) *Anthony McLean* • Perioperative myocardial ischaemia (pg 21) *Toh Khay Wee* • Acute pulmonary embolism *Ian K S Tan*	**SYMPOSIUM 4** **Miscellaneous** *Chairpersons: Tan Cheng Cheng / Noor Airini Ibrahim* • Are severity of illness scores useful to predict outcome? (pg 22) *Chen Fun Gee* • How to expand the ICU in an outbreak – What you can and can't do (pg 23) *Gavin Joynt* • Dealing with families (pg 24) *Shanti Rudra Deva* • Care of the dying patient *Dermot Phelan*	**INTERACTIVE SESSION (II)** **Data Interpretation** *Moderator: Lim Chew Har*
1630 – 1700	TEA / TRADE EXHIBITION		
1700 – 1800	**TEA SYMPOSIUM** *(Sponsored by Edwards Lifesciences)* *Chairperson: Ng Siew Hian* The role of minimally invasive cardiac output monitoring in improving perioperative outcome *Can Tong Joo*		

VENTILATOR INDUCED LUNG INJURY

Mohd Basri bin Mat Nor

Department of Anaesthesiology and Intensive Care, Faculty of Medicine, International Islamic University Malaysia, Kuantan, Pahang, Malaysia

Mechanical ventilation is a life-saving therapy and is the cornerstone of therapy for patients with acute respiratory failure. Its widespread use was initiated in the mid-1950s for the treatment of paralytic poliomyelitis. Our understanding of the mechanisms of gas exchange and pulmonary mechanics in relation to mechanical ventilation has increased tremendously. However, especially for the past 20 years it is generally accepted that mechanical ventilation per se can initiate lung injury in a previously healthy lung as well as can exacerbate preexisting lung injury and contribute to patient morbidity and mortality. Today the aim of mechanical ventilation is to oxygenate and ventilate without causing or induce further ventilator induced lung injury (VILI). There are a number of mechanisms that can lead to the development of VILI. Two major mechanisms of VILI are cyclic alveolar over-distension and repetitive alveolar collapse and reopening.

Barotrauma refers to gross air leaks (including pneumothorax or pneumomediastinum) that correlate with high levels of peak airway pressure and positive end expiratory pressure (PEEP), high tidal volume and gas trapping. *Volutrauma* refers to more subtle type of lung injury that can occur secondary to pulmonary overdistension induced by mechanical ventilation. Many investigators have shown that cyclic overdistension of lung units can lead to development of pulmonary oedema, diffuse alveolar damage, increased epithelial permeability and increased microvascular permeability. The term volutrauma has been introduced to indicate that the critical variable for injury is not the airway pressure per se but rather lung volume or end-inspiratory stretch.

Meanwhile, ventilation at low lung volumes can also cause parenchymal damage due to alveolar collapse, termed atelectrauma. *Atelectrauma* refers to the damage that can occur when lungs are allowed to become atelectatic and then re-expanded. The repetitive opening and closing of these atelectatic alveoli provoked by conventional mechanical ventilation can cause excessive alveolar wall strain that triggers the inflammatory cascade.

Biotrauma, a relatively newly described response to mechanical ventilation is characterized by the release of inflammatory mediators from the cells within the lung. These mediators can cause further injury to lung tissue and to other organ systems. A number of studies have provided evidence that mechanical ventilation of injured lungs can exacerbate lung injury and lead to additional inflammatory response. Most experimental studies clearly demonstrate that overstretching lung cells and allowing recruitment and de recruitment of the lung can lead to an increased in lung cytokines. Under conditions in which there is increased lung permeability, these cytokines may translocate from the alveolar space to the systemic circulation. The concept of biotraumas may help explain why most patients who die with the acute respiratory distress syndrome (ARDS) succumb not because their lungs fail but because of the development of multiple organ dysfunction syndrome (MODS) involving both the lungs and other organs.

All forms of mechanical trauma to the alveoli may lead to surfactant dysfunction, epithelial and endothelial cell injury with increased alveolar capillary permeability, inflammatory mediator release, neutrophil infiltration, lung microphage activation and bacterial translocation. Each of these mechanisms can in turn exacerbate the local injury and initiate or potentiate a systemic inflammatory response.

Patients with acute lung injury (ALI) and ARDS are particularly at high risk of VILI since their lungs are already inflamed and heterogeneously damaged. The lungs in patients with ARDS can be thought of as consisting of 3 zones: collapsed lung regions, lung region that are open to recruitment and over distended regions. Thus a ventilatory strategy that may be protective for one region of the lung may be injurious for a neighboring region. The relatively healthy zones of the lung that have higher compliance tend to receive

the bulk of delivered tidal volumes and subjected to more stress and strain than consolidated alveoli. Therefore the strategies to minimize volutrauma and atelectrauma can directly compete.

The understanding of lung pathophysiology and the role of VILI has led to advances in lung protective strategy for the mechanical ventilation support of patients with severe respiratory failure. These include low tidal volume ventilation, permissive hypercapnia, open lung approach and recruitment maneuvers.

Based on the ARDS Network trials and others detailing the open lung approach, most clinicians today avoid high-peak inspiratory pressures, use low tidal volumes and apply appropriate levels of PEEP to encourage lung recruitment and avoid cyclic atelectasis. This approach is the current 'gold standard' for mechanical ventilatory support and avoiding additional VILI in patients with ALI/ARDS. In patients with ALI/ARDS, plasma interleukine-6 and 8 are associated with morbidity and mortality. Lower tidal volume ventilation in the ARDS Network prospective, randomized trial was also associated with a more rapid attenuation of the inflammatory response. However, there have been some barriers to widespread implementation of the low tidal volume ventilation strategy particularly with regard to patient discomfort and tachypnea and concerns about hypercapnia, acidosis and hypoxaemia.

The paradigm for mechanical ventilation in patients with ALI and ARDS has evolved in the last 10 years from a goal of normalizing blood gases to one of avoiding VILI while maintaining adequate gas exchange. One of the potential method may be useful in the avoidance of VILI is high-frequency oscillation ventilation (HFOV). HFOV theoretically satisfies all the goals of a lung protective strategy and offer several potential advantages over conventional ventilation (CV). HFOV achieves gas exchange by delivering very small tidal volumes at frequencies ranging from 3 to 15 Hz. Potential advantages over CV include delivery of smaller tidal volumes (VT smaller than the anatomical dead space), limiting alveolar over-distension, promoting more alveolar recruitment, maintenance of a constant mean Paw during inspiration and expiration thus preventing end-expiratory collapse. In adults, two prospective randomized control trials, however, while showing no mortality benefit, have suggested that HFOV compared to CV is a safe and effective ventilation strategy for patients with ARDS. Several studies suggest that HFOV may improve outcomes if used early in the course of ARDS. Although the exact severity threshold at which to initiate a trial of HFOV remains unclear, an emerging approach includes the following severity criteria:

- $FiO_2 > 0.60$ and $SpO_2 < 88\%$ on CMV with PEEP > 15cm H_2O or
- Plateau pressures > 30 cm H_2O or,
- Mean airway pressure > 24 cmH_2O.

While promising, the use of HFOV as a primary mode for lung-protection in ARDS needs further investigation before it is widely adopted as routine elinical practice. At the current practice, the usual indication for HFOV in adults should be as rescue therapy for patients with severe hypoxemic respiratory failure who are not responding to conventional ventilation.

Future pharmacology therapy for VILI may be aimed at mitigating biotrauma by the use of anti-inflammatory agents, antibodies and gene transfer approaches. The use of an agent to effect interleukin-1b blockade or intra-tracheal instillation of antibodies tumor necrosis factor (TNF)-alpha have shown some promise in animal studies.

In summary, the current goal of mechanical ventilation is to avoid VILI while at the same time maintaining adequate oxygenation and ventilation. HFOV is in theory the ideal 'lung protective' method. Future studies should compare HFOV with open lung-protective strategy to determine whether one strategy is superior and whether earlier initiation might improve outcomes. Although the initial inciting event during VILI may be mechanical in nature, it may trigger the release of inflammatory mediators. Biotrauma may be the missing link between the pulmonary pathophysiology of ARDS and the pathophysiology of MODS and may explain the observation that many patients with ARDS die from MODS, not hypoxemia. These concept may lead to a paradigm shift in which therapy of VILI is not only minimizing the physical forces causing injury, but also on modulating biotrauma using anti-inflammatory or anti-apoptotic interventions to limit the extrapulmonary consequences of ventilator induced inflammation.

ACUTE LUNG INJURY – OXYGENATION STRATEGIES

Kwek Tong Kiat

Department of Anaesthesiology, Tan Tock Seng Hospital, Singapore

Acute Lung Injury (ALI) and Acute Respiratory Distress Syndrome (ARDS) are relatively common with incidences of 20-50 and 15-30 cases per 100 000 persons per year respectively. ARDS and ALI are associated with pathologically complex changes in the lung, manifested by an early exudative phase followed by proliferative and fibrotic phases.

The treatment of ALI and ARDS is mainly supportive, with many patients requiring mechanical ventilatory support. Hypoxemia refractory to supplemental oxygen often complicates the clinical course and is a direct cause of death in 16-20% of deaths for ARDS.

THE STRATEGY FOR OPTIMIZING OXYGENATION IN ALI AND ARDS INCLUDE THE FOLLOWING:

1. Initiating mechanical ventilatory support to reduce the work of breathing.
2. Maintaining euvolemia and normal blood pressures without excessive elevations of extravascular lung water.
3. Employing a low tidal volume lung protective ventilation strategy to reduce ventilator associated lung injuries (VALI).
4. Using adequate PEEP to maintain end expiratory lung volumes.
5. Opening the lungs and keeping them open. The respiratory system compliance in ARDS is reduced with the PV curve shifted downwards and to the right. Recruitment maneuvers (RM), generating high transpulmonary pressures, are required to open these collapsed lung units and higher PEEP to keep these units recruited. The effect on oxygenation is variable with some showing dramatic improvements but no change in others. Performing RM as part of the ventilation strategy has not been shown to improve clinical outcomes.
6. Turning a patient prone is reported to improve oxygenation in 60-70% of ARDS patients but not found to improve outcomes in randomized controlled trials. Problems reported include accidental extubation, facial edema, pressures sores and intolerances of early enteral feeding.
7. Selective pulmonary vasodilators such as inhaled nitric oxide and aerosolized prostacycline have been found to provide short-term improvements in oxygenation but no improvement in outcomes. They are used mainly as rescue therapy for life-threatening hypoxemia.

PHARMACOLOGIC THERAPY FOR ARDS – HAS IT FAILED?

N M Nik Abdullah

Department of Anaesthesiology and Intensive Care, Health Campus, Universiti Sains Malaysia, Kelantan, Malaysia

Acutely ill patients with severe respiratory failure are common problems in the ICU world-wide today. Acute respiratory distress syndrome (ARDS) represents the most severe form of pulmonary organ dysfunction with a significant increase in morbidity and mortality. In the United States, it has been estimated that there are 150,000 cases of ARDS per year, an incidence of about 75 cases per 100,000 persons. Management of critically ill ARDS patients involves lung protective ventilation and in addition, several pharmacologic adjunctive therapies which are being tried and are discussed below.

INHALED NITRIC OXIDE

Inhaled nitric oxide (NO) has been shown to acutely improve oxygenation by dilating pulmonary vessels supplying ventilated alveolar units and improving V/Q matching. It has a short half life in blood and the vascular effects are limited to the pulmonary circulation. The combination of rapid clinical improvement and lack of risks has led to the widespread usage of inhaled NO in ARDS. However, in the absence of conclusive evidence of efficacy, UK Consensus Guidelines advised that NO should only be administered after ventilation had been optimized and that the dose administered (commonly 5–20 ppm) should be adjusted to the lowest possible level needed for efficacy. Inhaled NO is efficacious in the short term and has a place as rescue therapy. Many studies show little benefit from long-term administration of NO in ARDS and data from Scandinavia showed no evidence of benefit in patients administered NO over a 3-year period.

ANTI-INFLAMMATORY

Anti-inflammatory treatment has been proposed in view of the histopathologic findings of inflammatory and fibrotic activity in late ARDS, with disrupted repair mechanisms; although trials of nonsteroidal anti-inflammatory agents have been ineffective.

Earlier there had been great enthusiasm by clinicians in the use of corticosteroids in the management of ARDS. Early findings show therapeutic benefit, but the results of later studies failed to show efficacy in preventing lung injury or in modifying mortality. Gattinoni et al., 1998 demonstrated that high-dose methylprednisolone was not useful in early treatment of ARDS. However, Kacmarec R. Met al.,2006 showed that corticosteroid is effective in aiding resolution of lung injury in late fibroproliferative ARDS although the dose and duration of therapy remain debatable.

A variety of other anti-inflammatory agents have been administered to patients with ARDS including prostaglandin E_1, antiendotoxin antibodies, interleukin-1 receptor antagonists, platelet-activating factor receptor antagonists, antitumour necrosis factor antibodies, and ibuprofen, but none of these has proven successful in reducing mortality. Ketoconazole, a thromboxane A_2 synthetase inhibitor, appeared to prevent ARDS in a small number of patients, but it was not shown to be clinically efficacious in other study. (G. B. Bernard, ARDS Net, 1997).

ANTIOXIDANT

N-acetylcysteine (NAC) showed improved static lung compliance, pulmonary vascular resistance and chest x-ray scores as well as reducing the number of days that patients suffered from a low FIO_2/PaO_2 ratio in some studies. Patients with mild to moderate lung injury showed similar improvements although no difference was found in mortality, length of ventilatory support, or improvement in oxygenation in those with established ARDS. A recent, unpublished, large-scale phase III trial of procysteine has been stopped early due to concerns about excess mortality in the treatment group of the study. Currently, there is little evidence that intravenous NAC is of benefit to patients with ARDS.

SURFACTANT REPLACEMENT THERAPY

Surfactant abnormalities were one of the first pathophysiologic abnormalities described in patients with ARDS. Thus, surfactant replacement therapy has been proposed as an adjunctive and anti-inflammatory treatment in ARDS. Current data suggest that the pattern of ventilation may affect conversion of large functional surfactant aggregates to small minimally functional forms. However, a large randomized, controlled clinical trial testing the efficacy of aerosolized surfactant replacement showed no benefit on oxygenation and survival.

PARTIAL LIQUID VENTILATION

The use of partial liquid ventilation (PLV) using perfluorocarbons has been proposed as a method to open lung units and enhance gas exchange. Perfluorocarbons support oxygen transport and have a high vapor pressure so that they are rapidly volatilized and exhaled from the lungs. Unfortunately, preliminary reports of Phase II/III studies of perfluorocarbon therapy failed to show a benefit on survival. At the present time, PLV remains an experimental approach that is not recommended for use as a routine treatment to support oxygenation.

SUMMARY

Lung protective mechanical ventilation strategy remains the main therapy that has been shown to reduce mortality and the development of organ failure in patients with ARDS. At the present time, the roles of pharmacologic and adjunctive therapies are being actively investigated and are found to be less effective in the treatment of ARDS.

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PATIENT-VENTILATOR SYNCHRONY

Andrew D Bersten

Department of Critical Care Medicine, Flinders University and Flinders Medical Centre, Adelaide, South Australia

Patient-ventilator dyssynchrony, also termed 'fighting the ventilator', is found during both intubated ventilation and during non-invasive positive pressure ventilation (NIPPV). It may cause distress and agitation, diaphoresis, tachycardia, hypertension, and potentially myocardial ischemia, tachypnoea, hypoxemia, and weaning failure.

Immediate intervention including hand ventilation, intubation or re-intubation, may be required; however, a more measured approach is usually possible. In the first instance airway patency and circuit connection must be confirmed. During NIPPV, as positive pressure splints open the airway, secretions, foreign body, airway swelling and mask leak need to be considered. During intubated ventilation problems with the endotracheal tube such as secretions, blood, foreign body, kinking, or displacement, and cuff herniation must be excluded. Changes in the clinical state such as pneumothorax, pulmonary edema, bronchospasm, and dynamic hyperinflation may also result in altered neural drive and dyssynchrony.

Dysfunction of the ventilator can then be considered, and is most conveniently divided into three parts of the respiratory cycle, which are most easily identified by respiratory waveform analysis.

1. INITIATION OF INSPIRATION

a. Dynamic hyperinflation and intrinsic PEEP reduces sensitivity of the ventilator trigger. This may be improved by a lower level of extrinsic PEEP.

b. Poor trigger sensitivity may impede initiation of inspiration, while this is less of an issue with modern ventilators, it can become important during high patient demand or respiratory muscle insufficiency. In general flow triggering is preferable as autocycling is less likely.

c. Autocycling, a triggered assisted or supported breath without patient effort, may occur due to excessively low trigger threshold or due to Pao or flow distortion as commonly seen with large cardiogenic oscillations, hiccups, condensate in tubing or a circuit leak.

2. INSPIRATION

a. If there is inadequate flow during inspiration, such as low flow rates during assist-control ventilation, flow starvation may occur. This is evident as a scalloped pressure-time trace.

b. Excessively rapid development of the set delivered pressure, a rapid rise time, may be detected by an overshoot in airway pressure; an excessively long rise time will be seen as a rounded inspiratory flow profile, similar to that seen with continued inspiratory muscle effort.

3. TERMINATION OF INSPIRATION

a. Ventilator inspiratory time can be greater or less than the patient's neural inspiratory time.

PHARMACOLOGICAL APPROACHES TO THE VENTILATED PAEDIATRIC PATIENT

Tang Swee Fong

Department of Paediatrics, Faculty of Medicine, Universiti Kebangsaan Malaysia, Kuala Lumpur, Malaysia

Severe respiratory failure (including acute lung injury and acute respiratory distress syndrome) continues to be a significant cause of morbidity and mortality, not only in adults but also in paediatric patients. In addition to advances in ventilatory management, several pharmacological interventions have been investigated to improve survival in paediatric patients. These have included inhaled nitric oxide, surfactant therapy and heliox.

Inhaled nitric oxide, a selective pulmonary vasodilator, has been used in acute respiratory distress syndrome to improve ventilation-perfusion matching and reduce pulmonary hypertension. Studies in paediatric patients with acute respiratory failure have shown improvements in oxygenation with inhaled nitric oxide but its use have not been associated with improved survival. Exogenous surfactant administration in acute hypoxaemic respiratory failure remains of unclear benefit in adult patients. More recently however, exogenous administration of calf surfactant in paediatric patients was shown to not only improve oxygenation but reduce the days on ventilator as well as days to discharge from intensive care. There has also been some evidence to suggest that heliox, used in the early acute phase of asthma, can improve gas flow and carbon dioxide elimination, while reducing peak inspiratory pressures and the potential risk of barotrauma and air leak.

Despite the promising results that some of these therapies have shown much work needs to be done especially in the paediatric population to determine the efficacy of these therapies. As our understanding of acute respiratory distress syndrome evolves, promising new therapeutic agents are being explored.

CURRENT PRACTICES IN TRANSFUSION MEDICINE IN THE PICU

Stephen Jacobe

Helen Macmillan Paediatric Intensive Care Unit, The Children's Hospital at Westmead, New South Wales, Australia

It has been accepted wisdom for many years that treating anaemia by administering allogeneic blood transfusions would improve oxygen delivery and this would lead to improved patient outcomes. Optimising oxygen delivery in the way was felt to be particularly important in critically ill medical and perioperative patients in intensive care units. Blood transfusion is not a benign intervention, however, with a number of risks including infection, transfusion-related acute lung injury (TRALI) and transfusion-related immunomodulation (TRIM). Running a safe and reliable blood collection and supply service is also costly.

The TRICC study involved randomisation of adult intensive care patients to either a "restrictive" or "liberal" blood transfusion threshold.[1] A non-statistically significant trend towards a lower mortality in the restrictive group was found, and other outcomes were also comparable. A number of observational studies have also failed to find any apparent benefit from a more liberal transfusion policy, with a suggestion of worse outcomes in patients receiving more transfusions.[2,3] As a result of these findings, the current recommendation for most adult ICU patients is to adopt the lower transfusion trigger of 7.0 g/dL.

The result of the TRIPICU study were recently published.[4] This study of paediatric intensive care unit patients also failed to show any benefit from transfusing patients at a haemoglobin level of 9.5 g/dL as opposed to 7.0 g/dL. There may be subgroups of patients, however, where a higher haemoglobin may be beneficial including premature infants[5,6], patients with cyanotic heart disease, and adults with ischaemic heart disease.[7]

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SUPPORT FOR THE FAILING HEART

Loh Tsee Foong
K K Women's & Children's Hospital, Singapore

The most important initial approach to heart failure is identification of the cause and specific therapy if possible. In acute onset heart failure, intensive medical therapy is used to stabilize patient. Children with congenital heart condition should have their respiratory, haemodynamics and nutritional status optimised before surgical repair or palliation is rendered. In non-structural or chronic heart failure, medical therapy is targeted at symptomatic relief as well as reversing ventricular re-modeling.

Medical therapy includes diuretics, inotropic support, drugs modulating renin-angiotensin axis and beta-blockers. Newer forms of therapy include use of recombinant natriuretic peptide, aldosterone and arrhythmia control using biventricular pacing.

Where medical therapy has failed and surgical or palliation therapy is not possible, mechanical cardiovascular support is possible. When short-term recovery is expected, extracorporeal membrane oxygenation is warranted. ECMO provides both pulmonary and cardiac support but it can also be used as a bridge to transplantation. Most paediatric heart failure is biventricular, which exclude intra-aortic balloon pump as a viable option.

Patients needing long-term support with adequate pulmonary function, ventricular assist devices (VAD) are the better choice. Although there are wide choices, it is less readily available as they are used in specialised centers and service support is important. Patient size has been a traditional hindrance in paediatric age group but gains have been made in this area notwithstanding most tertiary center experience with VAD are limited. VAD are also used as a bridge to transplant. Use of VAD as destination therapy needs to take into consideration resource allocation and ethical discussions. Devices can deliver blood flow in pulsatile or rotary manner. There are advantages and disadvantages of between LVAD and ECMO, so both types of support can be complementary.

When heart failure can no longer be sustained medically or mechanically, cardiac transplantation should be considered. Cardiac transplantation maybe required on an elective or emergent basis. International Society for Heart, Lung Transplantation registry has shown improved survival over the last 20 years but mostly in centers that perform a critical mass of this procedure.

PREDICTING FLUID RESPONSIVENESS IN THE CRITICALLY ILL

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

Fluid administration or volume expansion is frequently used in critically ill patients to improve haemodynamics. From the Frank-Starling relationship, an increase in preload will induce a significant increase in stroke volume only if the ventricle operates on the ascending portion of the curve (condition of ventricular preload dependence) than if the ventricle operates on the flat portion of the curve (preload independence). There is a need for predictive markers of fluid responsiveness in order to select patients who could benefit from volume expansion and to avoid ineffective or deleterious fluid therapy in the non-responders.

Parameters used as indices of fluid responsiveness can be divided into static and dynamic markers.

STATIC MARKERS:

– intracardiac pressures e.g. CVP, PAOP

– ventricular end-diastolic volumes e.g. RVEDV, LVEDV, GEDV

DYNAMIC MARKERS USE HEART-LUNG INTERACTIONS:

– pulse pressure variation (PPV)

– stroke volume variation (SVV)

– descending aortic blood flow variation e.g. descending aortic blood velocity, diameter of descending aorta

Markers of cardiac preload regardless of their methods of measurement are poor indicators of preload dependency and thus are poor predictors of fluid responsiveness.

Mechanical ventilation induces cyclic alterations in ventricular filling, and in consequence stroke volume and cardiac output. Respiratory variation of dynamic parameters has been demonstrated to be reliable markers of fluid responsiveness. However, in the presence of spontaneous breathing, the indices of fluid responsiveness that use heart-lung interactions such as PPV and SVV are no longer reliable.

The limitations of the use of the respiratory variation of dynamic markers include the influence of tidal volume delivered, presence of spontaneous breathing activity or arrhythmias.

Passive leg raising which is a simple bedside manoeuvre used to increase cardiac preload has been demonstrated to be a valuable tool for predicting fluid responsiveness. This dynamic method remains reliable in patients with mechanical ventilation regardless of their cardiac rhythm and their own breathing activity.

MANAGEMENT OF SYSTOLIC AND DIASTOLIC HEART FAILURE

Anthony McLean

Nepean Hospital, Penrith, Sydney, New South Wales, Australia

Cardiac dysfunction is commonly found in the Intensive Care patient population with studies identifying around 30% of all patients admitted to a general ICU having such. The aging population is likely to increase this proportion, some of whom will have underlying heart failure complications affecting their ICU stay. It is important to assess cardiac function and identify what actual abnormalities are present – systolic failure, diastolic failure, mixed systolic/diastolic failure, valvular dysfunction, predominantly left or right heart dysfunction etc. Clinical examination and invasive pressure measurements are often insufficient with echocardiography being the tool par excellence.

Once the diagnosis of isolated systolic dysfunction is made, goal directed therapy can be instituted. Acute decompensating heart failure may be on the background of chronic disease (eg previous infarcts, idiopathic cardiomyopathy) or de novo (severe sepsis). Management consists of optimal fluid loading, inotropes, reduction of afterload where possible, and neurohormonal manipulation. Underlying correctable coronary lesions should be identified early and where possible dealt with quickly. Identifying optimal fluid loading cannot usually rely on a single examination or modality. The combination of clinical assessment, including SLR or pulse pressure variation, with a diagnostic technique such as PiCCO or echocardiography is often necessary. Unfortunately the CVP and PCWP are not always helpful guides to preload responsiveness in the patient with multiorgan failure. Once preload is optimised but inadequate cardiac output is still a problem then vasoactive agents should be considered. The traditional use of adrenergic agents is being challenged from a number of angles including lack of supportive published data, theoretical negative properties in this patient population, as well as good data on the beneficial effects of betablockers in the chronic heart failure population. Newer agents such as levosimendan are theoretically attractive but clinical benefits are anecdoctal rather than strong trial data. In terms of afterload reduction the application of mechanical devices such as IABP should be considered early rather than later.

The approach to left ventricular diastolic dysfunction has to be carefully considered as effective treatment modalities are few, in contrast to therapies available in systolic dysfunction. Drug therapy in the acute situation is limited and the emphasis is on increasing diastolic filling period (reducing heartrate) and limiting the use of inotropic agents. Obviously the mixture of systolic and diastolic dysfunction makes management more complicated.

PERIOPERATIVE MYOCARDIAL ISCHAEMIA

Toh Khay Wee

Subang Jaya Medical Centre, Selangor, Malaysia

It is estimated that 1% of patients experience a cardiac event that carries a mortality of 15 to 25%. In high risk surgery like vascular surgery, the risk of a cardiac event can reach 34% with a mortality of 25 to 40%. In certain developed countries up to 30% of patients presenting for non cardiac surgery have risk factors for coronary artery disease. In Malaysia, we could be facing similar levels due to a rising life expectancy and increasing incidence of obesity in our population.

The pathophysiology of perioperative myocardial ischaemia arises out of a combination of coronary occlusion, increasing myocardial oxygen demand and a reduction in myocardial oxygen delivery. In fact, up to 50% of perioperative myocardial infarctions show no evidence of an occluding thrombus which may indicate an imbalance between demand and supply. Furthermore, plaque rupture leading to thrombosis can occur even without the prior formation of a stenosed coronary vessel. This has led to the concept of the 'vulnerable plaque'.

Risk indices have been developed to stratify patients according to their risk of developing perioperative cardiac events. One of the most well known of risk indices is the Goldman Cardiac Risk Factors (1977). However, newer risk indices like the Lee Index (1999) have superseded it with better discriminatory power. Various non invasive tests can be used to further stratify this population of at risk patients. Of these tests, dobutamine stress echo has been shown to have the highest sensitivity and specificity in detecting patients at risk. However, all of these tests suffer from a poor positive predictive value i.e. a large proportion of those who test positive do not subsequently develop a cardiac event. In an effort to standardize the management of patients at risk of perioperative myocardial ischaemia and reduce unnecessary testing, the American College of Cardiology has devised a set of guidelines that is based on expert opinion. However, all the current tests only detect coronary stenosis and not the 'vulnerable plaque' which accounts for 50% of perioperative myocardial infarctions. Of all the inflammatory markers available, highly sensitive C reactive protein has shown the most promise in identifying the 'vulnerable plaque'.

Risk reduction strategies can be widely categorized into the pharmacological and revascularization strategies. Pharmacological risk reduction strategies that have been shown to be effective include the beta blockers, statins and alpha 2 agonists. However, all the pharmacological trials suffer from small sample sizes and many were retrospective in nature limiting the power of the studies. We wait in anticipation of the PeriOperative ISchaemic Evaluation (POISE) trial which is the first multicentre double blind randomized trial to evaluate the effectiveness of beta blockers to reduce cardiac events in at risk patients. Risk reduction in the form of coronary bypass grafting and percutaneous revascularization may be logical. However, the risk of revascularization has to be added to the risk of surgery without revascularization and this may exceed the risk of proceeding with surgery without revascularization. In fact, the CARP trial has shown that there were no differences in mortality at 2 years and troponin levels between the two groups. The current recommendation for revascularization is only limited to patients who would otherwise have needed it independent of their non cardiac surgery. Furthermore, revascularization may increase mortality if non cardiac surgery is undertaken within 6 weeks of the revascularization procedure. Current advice for patients at risk on aspirin is to continue aspirin throughout surgery even though there is a slight increase in bleeding.

Perioperative myocardial infarction is difficult to detect and is often missed as they tend to be silent. In high risk groups, it may be advisable to perform serial electrocardiograms and troponin measurements. The view of perioperative myocardial damage has now changed from an 'all or none' binary event to a wide spectrum of increasing myocardial damage. This has been made possible by the measurement of troponin levels which have been shown to correlate with long term outcomes. The management of perioperative myocardial infarction is mainly based on expert opinion as there are very few trials. The decision to pursue interventional or medical therapy should be individualized based on the category and severity of infarction, type of surgery and the need for haemostasis.

ARE SEVERITY OF ILLNESS SCORES USEFUL TO PREDICT OUTCOME?

Chen Fun Gee

Department of Anaesthesia, National University Hospital, Singapore

High levels of ICU use at the end of life has attracted interest in the ability to predict prospectively which patients will not survive despite ICU care. Over the last 20 years numerous models have been developed that quantifies physiologic derangements and comorbidities and from which estimates of survival, length of stay and resource use are based. Most of the models rely on large computerized databases of patients admitted to a variety of ICUs in different hospitals and using logistic regression equations provide probabilities of mortality and to a lesser extent length of stay.

The evaluation of any model rests on 2 fundamental concept: discrimination and calibaration[1]. Discrimination refers to the model's ability to distinguish whether a patient died or survive. It is measured by the area under the receiver-operating characteristics curve which plots sensitivity against specificity over a wide range of mortality cut-off points. Generally the higher the value, the better the accuracy and predictive power and current models are better than 0.8. Calibration refers to how well the predicted outcome compares with the observed outcome throughout the range of risk. These 'goodness-of-fit" analyses are displayed as bar graphs that plot actual to predicted mortality or Hosmer and Lemeshow chi-square-type statistic. In general Hosmer-Lemeshow statistic lower than 15 and P values of 0.2 to 0.8 are considered good.

The Acute Physiological And Chronic Health Evaluation (APACHE) score was developed by Knaus et in 1981[2]. It had 2 parts, the acute physiology score (APS) and the chronic health evaluation score (CHE). It consisted of 34 physiological variable collected over a period of 32 hours after ICU admission and there were no probability calculations of death. The APACHE was improved in 1985 and incorporated several changes which made it one of the most popular scoring system used in the world[3]. The APS variables were reduced to 12 and the period of data collection reduced to the first 24 hours of ICU admission. It allowed calculation of individual risk of death.

The APACHE II score was extensively validated in different populations in different clinical conditions. Our validation in Singapore yielded ROC of 0.87 and this was similar to reports in the UK ((0.835)[4]. In 1991 Knaus refined the APACHE II into the APACHE III with increases in number of physiological variables data collected from 12 to 17 in the first 24 hours of ICU admission[5]. Mortality prediction was calculated from the APS, age, CHE, source of ICU admission, length of hospital stay prior to ICU admission, coefficients for surgery and increased numbers of diagnostic categories. The discriminatory ability increased to a ROC of 0.89. The Hosmer-Lemeshow statistic was Chi square 48.71, P<0.001. More recently Zimmerman reported the use of the APACHE IV in 110558 ICU patients[6]. The new score was modified from the APACHE III using refined statistical techniques. The reported discrimination ability was ROC 0.88. Calibration was excellent with the Hosmer-Lemeshow statistic of 16.8 and P=0.08.

Sinuff et al reviewed the literature comparing physician vs scoring system prediction of ICU or hospital mortality of critically ill patients[7]. Physicians were better at predicting outcomes compared with scoring systems (ROC 0.85 vs 0.63).

At the present state, severity scores allow calculation of "risk adjusted" mortality rates for groups of patients with similar conditions, allowing quality improvements and clinical trials to be carried out. The ability to predict outcomes for individual patients for rationing decisions is at present not sufficiently accurate for us to incorporate in our critical care practice.

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HOW TO EXPAND THE ICU IN AN OUTBREAK – WHAT YOU CAN AND CAN'T DO

Gavin M Joynt

Department of Anaesthesia and Intensive Care, The Chinese University of Hong Kong, Hong Kong

Intensive care units are expensive facilities and as a consequence intensive care units are usually maximally utilized. An additional requirement for intensive care facilities is likely to occur during an epidemic. Any additional requirement has the potential to overwhelm existing intensive care resources and therefore it may become necessary to rapidly increase the capability of existing intensive care facilities. The experience and recommendations of a consensus conference held in Hong Kong is presented and describes the views of clinicians, nursing, engineering and other supporting staff from Hong Kong and Singapore who had direct experience of expanding intensive care services in response to the epidemic of severe acute respiratory syndrome (SARS). Recommendations for adequate expansion are made on the basis that a reasonable standard of ICU care will be maintained. An assessment of the need for additional staffing is made, also based on maintenance of reasonable standards of ICU care in outbreak conditions. The requirement for training of anticipated additional staff, established infection control procedures, good communication procedures and the resolution of anticipated ethical dilemmas is stressed. Certain preparations for expansion should be completed in advance. These include the training of reserve staff, fit testing of negative pressure respirators, sourcing of material and designs that will allow physical modifications to the ICU and additional equipment supply sourcing.

DEALING WITH FAMILIES

Shanti Rudra Deva

Department of Anaesthesia and Intensive Care, Hospital Kuala Lumpur, Kuala Lumpur, Malaysia

The critically ill patients due to their complexity of disease require a lot of time and effort for their care by the intensive care physician and nurses. It can be a very stressful time for all the healthcare workers in the intensive care. The families of the patient are equally stressed and anxious as they are unable to communicate with their loved one and understand the complexity of the problems.

Communication between the intensive care physician and the families is of paramount importance to maintain a healthy relationship. Families should not be made to feel abandon and marginalised. In fact, most families rate a clinician's good communication skills over their clinical acumen. Information appears to be greatest need for all families when their loved ones are in the intensive care. This information-seeking behavior has been described as a way in which families cope with stress and anxiety.

A simple maneuver such as the provision of an information brochure has been shown to help families comprehend the complexity of the problems when their loved one is admitted into the intensive care. Another novel approach is allocate protected time. Allocation of protected time allows families to ask questions and receive updates after the team's daily ICU rounds and that way understand the disease progress of their loved one, good or bad. The feedback from the families where protected time has been allocated is overwhelmingly positive and appreciative. A French study has shown that when daily updates are provided, only half the families wanted to share in the actual decision-making process and understand better when there is a need to institute limitation or withdrawal of treatment.

Long gone are the days when ICU staff can spend all their time and effort on the patient and ignore the families at the end of the bed. It is clear that families need and deserve our attention. Communication with family members of the critically ill is crucial from the beginning as it can circumvent conflicts and improve family's satisfaction on the care that has been provided in the intensive care.

PROGRAMME
23 JUNE 2007, SATURDAY

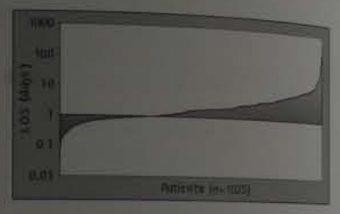
0800 – 1730	REGISTRATION		
0830 – 0930	**PLENARY 2** — Sabah *Chairperson: Tang Swee Fong* Chronically ill children in the PICU (pg 26) *Stephen Jacobe*		
0930 – 1030	**PLENARY 3** — Sabah *Chairperson: Tang Swee Fong* Practising evidence-based medicine when there are no randomized controlled trials (pg 27) *Gavin Joynt*		
1030 – 1100	TEA / TRADE EXHIBITION		
1100 – 1300	Sabah **SYMPOSIUM 5** **Sepsis** *Chairpersons: Shanti Rudra Deva / V Sivasakthi* • Tropical infections in ICU *Noor Azrini Ibrahim* • Candida colonization vs infection (pg 28) *Louisa Y L Chan* • Use of vasopressin in septic shock (pg 29) *Anthony McLean* • Albumin in the treatment of sepsis (pg 30) *Chen Fun Gee*	1100 – 1230 Sarawak **SYMPOSIUM 6** **Paediatrics** *Chairperson: Jimmy Lee* • Nutritional support in the critically ill child (pg 31) *Teh Keng Hwang* • Is non-invasive ventilation beneficial in children? (pg 32-33) *Lee Lean See* • Sedation in PICU *Thavaranjithan*	Selangor **INTERACTIVE SESSION (III)** **Respiratory** *Moderator: Sekar Shanmugam*
1300 – 1430	LUNCH		
1430 – 1630	Sabah **SYMPOSIUM 7** **Respiratory (2)** *Chairpersons: Lim Chew Har / Marlizan Md Yusoff* • Non-invasive ventilation – What are the limits? (pg 34) *Louisa Y L Chan* • Percutaneous tracheostomy – Has it lived up to the promise? (pg 35) *Syed Rozaidi Wafa* • Weaning from mechanical ventilation: Science or art? (pg 36) *Gavin Joynt* • Unilateral lung disease (pg 37) *Andrew Bersten*	Sarawak **SYMPOSIUM 8** **Metabolic / Renal** *Chairpersons: Toh Khay Wee / Nik Abdullah Nik Mohamad* • Drug dosing in acute renal failure and RRT *Ian K S Tan* • Haemostatic drugs in the bleeding patient (pg 38) *Ahmad Shaltut Othman* • Intensive insulin therapy – Where are we? (pg 39-41) *Tan Cheng Cheng* • Use of steroids in the critically ill *Jenny Tong May Ceok*	Selangor **SYMPOSIUM 9** **Intensive Care For Nurses (1)** *Chairpersons: Asmarawati Mohd Yatim / Nik Azman Nik Adib* • Head of bed elevation (pg 42-44) *Anita Alias* • Reducing medication errors (pg 45) *V Sivasakthi* • Nurse-based protocols (pg 46) *Mahamarowi Omar* • Comfort and distress in ICU (pg 47) *Tai Li Ling*
1630 – 1700	TEA / TRADE EXHIBITION		
1700 – 1800	Selangor **FREE PAPER PRESENTATIONS** (pg 48-53) *Chairperson: Tai Li Ling*		

CHRONICALLY ILL CHILDREN IN THE PICU

Stephen Jacobe

Helen Macmillan Paediatric Intensive Care Unit, The Children's Hospital at Westmead, New South Wales, Australia

Ongoing advances in paediatric intensive care medicine have led to an improvement in care such that many children who previously would have died after serious acute illnesses or very high-risk, complex surgery now survive. Fortunately, most of these patients return to their premorbid level of functioning, however, some require prolonged mechanical ventilation or other technological support. Although few in number, these patients impose a large burden on often limited paediatric intensive care resources. In the financial year ending 2006 at the Children's Hospital at Westmead, for example, 32 patients stayed longer than 3 weeks, and 22 patients (2% of total patients) with a LOS greater than 4 weeks accounted for over 40% of all PICU bed days.



A small number of these patients require prolonged ventilatory support over months to years, and it can be easily argued that the Intensive Care Unit is an inappropriate environment for the longer-term care of these patients. This is for a number of reasons including: this represents an inappropriate use of a limited resource; the long-term psychosocial needs of the patient and family may be difficult to comprehensively address; the model of care in a critical care setting is not focused towards chronic medical issues; and, in distinction to adult practice, children have specific developmental and educational needs which are often not met in the paediatric intensive care setting. Alternative care environments include long-term ventilation units (LTVU's) within hospitals, but perhaps the ideal arrangement is to organise enough support to allow the patient to be safely cared for at home. Establishing LTVU's or home ventilation programs can be very resource intensive. In addition to the impact on hospital resource utilisation, each such patient's "chronic critical illness" has a profound impact on their family, the paediatric intensive care unit team, and indeed the wider health system generally.

Without accurate prognostic indicators it is impossible to discriminate which of these patients will improve to survive eventually without technological support and which may develop complications and die. Prospective research is required to clarify these issues, as well as developing and sharing management strategies to enable these patients to be discharged from hospital as soon as possible.

PRACTICING EVIDENCE-BASED MEDICINE WHEN THERE ARE NO RANDOMIZED CONTROLLED TRIALS

Gavin M Joynt

Department of Anaesthesia and Intensive Care, The Chinese University of Hong Kong, Hong Kong

Randomised controlled trails (RCTs) provide information in a specific way. In particular, they are the "gold standard" method to establish the effect of interventions. This is primarily because of the ability of a well designed RCT to provide equipoise and rule out significant bias. However, to make a RCT feasible several things are necessary. The condition tested needs to be reasonably common, the study needs to be ethically viable, costs need to be acceptable, and the outcome must be measurable in a reasonably short time. The quality of a RCT may also depend on the ability to adequately blind the intervention and assessment of the response. In ICU patients it is very difficult to conduct meaningful RCTs as many of the above cannot be easily realized. Once completed, external validity and therefore generalization of results can be problematic because RCTs are often performed on very specific groups of patients. Several alternative methods of generating evidence to support our practice in ICU exist and for practical reasons are either more useful to answer questions of evidence, or offer an alternative to RCTs. These include the use of experimental, quasi-experimental and observational studies. In ICU we need to learn to utilize observational studies, cohort studies, case control studies and other methods that synthesize existing information in a systematic way to ensure that our practice in individual cases is based on "best evidence".

CANDIDA COLONIZATION VS INFECTION

Louisa Chan
Malaysia

The distinction between candidal colonization and infection is often not clear cut in the ICU population. Risk factors and clinical signs are non specific and microbiological techniques lack sensitivity. Whilst infection can be confirmed by a positive culture from sterile site and treated accordingly, the management of events leading up from colonisation to invasive candidiasis remains the area of interest. There are 4 approaches to the presence of candida: watchful waiting, prophylaxis, preemptive treatment and empirical treatment.

THE FACTORS TO CONSIDER BEFORE THE CLINICIAN EMBARKS ON A TREATMENT APPROACH ARE:

- patient risk factors (presence of breakdown of normal barriers, artificial devices allowing biofilm formation, immune dysfunction and selection pressures)
- site of positivity (the number of sites and colonisation density),
- clinical signs and symptoms
- local epidemiological data on fungal infections eg albicans or non albicans spp, rates of colonization and blood stream infections

Watchful waiting- no antifungals instituted e.g. with candiduria, may elect to remove/change urinary catheter and reculture.

Prophylaxis- usually azole antifungals. Well established in very high risk groups e.g. BMT, solid organ transplants, prolong neutropenia.

Pre emptive- early antifungal therapy administered to patients with evidence of substantial colonization in the presence of multiple risk factors for candidal infection

Emprical-instituting antifungals to symptomatic patients deemed to be at high risk of invasive fungal infection prior to any positive sterile site cultures.

Once decision to treat with antifungals are made,

- Ensure correct dose and adequate duration
- Take note of important drug interactions and monitor side effects +/- drug levels

Use of antifungals does not negate the need to reduce risk factors, maintain good hand hygiene, meticulous technique for vascular catheter insertions and rational use of antibiotics.

USE OF VASOPRESSIN IN SEPTIC SHOCK

Anthony McLean
Nepean Hospital, Penrith, Sydney, New South Wales, Australia

Vasopressin is a nonapeptide. In 1896 it was discovered that pituitary extracts had a vasopressor effect. It wasn't until 1950 that vasopressin and ADH were discovered to be the same agent. It was not surprising that potential benefits in vasodilatory shock were predicted in addition to its role in Diabetes Insipidus and oesophageal variceal haemorrhage. Three vasopressor receptors are present –V^1R,V^2R, and V^3R, active in vascular tissue, kidney and pituitary respectively. Oxytocin receptors are also nonselective VP receptors. In the late 1990s, evidence that vasopressin levels, although transiently initially high, were rapidly depleted in sepsis and septic shock compared to other types of shock. This appeared to be due to depletion of stores in the neurohypophysis.

Some small studies indicating benefit using vasopressin to replace or reduce nor epinephrine doses in septic shock then appeared in the literature. These were accompanied by reports that high doses of vasopressin resulted in a worse outcome. Larger trials were then undertaken with results currently published in abstact form only.

The VANNISH trial was a multicentre Australian study examining the effect of a vasopressin infusion in early hyperdynamic septic shock on the endpoints of resolution of septic shock at the times of 72 hours and 7 days, in addition to effects on cardiac and hepatic performance. The results were that baseline serum vasopressin levels were higher than previously reported, there appeared to be a detrimental effect on cardiac and hepatic function, and that shock resolved in the great majority of patients (95.8%) by 72 hours. The high baseline vsopressin levels raised concerns and now the study is awaiting a control group of nonseptic patients for comparison. The VASST study was a large

multicentre study across 27 Canadian and Australian ICUs comparing lowdose (0.01-0.04 units/ml) vasopressin to norepinephrine infusion in septic patients. 6229 patients were screened and 779 enrolled. There was no difference in outcome in severe septic shock with decreased mortality in the less severe group. The definition of severity was based on dosage of inotropes used.

In summary the use of vasopressin may have benefit in less severe sepsis but what little evidence does exist for its use is only for low dose infusions (0.01-0.04 u/ml).

ALBUMIN IN THE TREATMENT OF SEPSIS

Chen Fun Gee

Department of Anaesthesia, National University Hospital, Singapore

A Cochrane meta-analysis suggested that albumin for the treatment of hypovolemia, hypoalbuminemia and burns in critically ill patients increased the risk of mortality[1]. Subsequently the saline vs albumin fluid evaluation (SAFE) study reported no important difference in the overall risk of death for adults given albumin or saline for fluid resuscitation in intensive care units[2]. The subgroup analysis of patients with severe sepsis in the study showed relative risk reduction with the use of albumin (0.87. CI 0.74 – 1.02) but this was not statistically significant.

Albumin is a globular, nonglycosylated negatively charged plasma protein containing 585 amino acids. Synthesized in the liver, it is the most abundant protein in the circulation (60% of total plasma protein). Normal serum albumin levels 35 – 45 g/l. Its main functions include maintenance of colloidal oncotic pressure, ligand binding, anti-oxidant effects and endometrial stabilization. In critical illness decreased synthesis and increased leakage from the vascular compartment results in hypoalbuminemia[3].

In a meta-analysis involving 291433 patients, JL Vincent reported that hypoalbuminemia was a potent dosed dependent independent predictor of poor outcome. Each 10 g/l decline in serum concentration raised the odds of mortality by 137%, morbidity by 89% and length of ICU and hospital stay by 28% and 71% respectively. Causality is an issue: whether hypoalbuminemia directly contributes to poor outcomes and albumin replacement therapy bestows benefit or hypoalbuminemia merely serves as a marker for a pathologic process in which case albumin replacement would be ineffective[4].

Dubois et al compared the use of IV albumin vs no albumin in 100 patients who had albumin levels < 31 g/dl prospectively randomized controlled trial. He found improvements in the SOFA scores but no difference in the secondary endpoints of mortality, ICU LOS, use of diuretics and fluid balance[5].

The SAFE study investigators look at whether albumin administration in patients with a serum albumin of < 25 g/l improved outcome. They reported no significant interaction between baseline serum albumin concentration and type of fluid administered on mortality, LOS in ICU or hospital, duration of RRT or mechanical ventilation[6].

There is no prospective randomized controlled trial specifically in septic patients on the use of IV albumin on the endpoints of mortality. Available data shows poor outcomes with hypoalbuminemia common with sepsis in the intensive care. No data exists to show that correction of the hypoalbuminemia with IV albumin improves survival. Until clinical trials designed to answer this question become available, the decision to withhold or administer IV albumin in septic patients should be guided by the clinical situation.

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NUTRITIONAL SUPPORT IN THE CRITICALLY ILL CHILD

Teh Keng Hwang

Department of Paediatrics, Hospital Alor Setar, Alor Setar, Kedah, Malaysia

Adequate nutritional support is an important aspect of the clinical management of pediatric intensive care patients. Critical illness increased nutritional requirements, increased catabolic state and result in a negative nitrogen balance. The aim of nutritional support is to prevent calorie or protein malnutrition, improve survival and reduce mortality and morbidity.

Nutritional assessment includes both subjective and objective assessment using anthropometric (weight, height, mid arm circumference, skin fold thickness) and laboratory evaluation (visceral protein pool, acute phase protein and nitrogen and energy expenditure)

Nutrition targets are to support the basal metabolic requirements and limit protein loss. The measured energy expenditure (MEE) is less than the predicted energy expenditure (PEE) unlike hypermetabolism in adults. PEE is estimated by using the equations eg Harris Benedict, Schofield, FAO/WHO/UNU equations. The MEE is lower in the critically ill child who are frequently sedated, and on mechanical ventilation with reduced work of breathing. The current practice is to provide caloric amounts to meet the MEE. During periods of critical illness the utilization of nutrients is inhibited by stress and inflammatory mediators. Therefore calories should be provided in a thoughtful manner Overfeeding may cause hepatic steatosis, impaired liver function hyperglycemia. Undernutrition affects wound healing, impaired immune response and altered organ function.

Nutritional support can be given via enteral or parenteral route. Early enteral nutrition is the preferred method. It keeps the intestine active (trophic effect) and reduce bacterial translocation. Parenteral nutrition is indicated in the presence of intestinal failure. Requirements can be divided into macronutrients and micronutrients

In recent years the focus is on immunonutrition (modified nutrition support regime) the use of a number of nutrients to modify inflammatory response and reduce secondary organ dysfunction. Nutrients such as arginine, glutamine, nucleotides and omega 3 fatty acids are being studied to see their effect in the pediatric setting.

IS NON-INVASIVE VENTILATION BENEFICIAL IN CHILDREN?

Lee Lean See

Paediatric Intensive Care Unit, Paediatric Institute, Hospital Kuala Lumpur, Kuala Lumpur, Malaysia

Non Invasive Ventilation refers to mechanical ventilation of the lung without an artificial airway, ie without endotracheal tube, tracheostomy or LMA – laryngeal mask airway.

This discussion focuses on Non Invasive Positive Pressure Ventilation (NPPV) in children with ACUTE respiratory insufficiency in the acute setting in Paediatric ICU and high dependency setup.

The indications, benefits and role of NPPV in Acute Respiratory Failure in Adults are clear and well established. NPPV is advocated as first line adjunct therapy to standard treatment in Acute Respiratory Failure in Acute Exacerbation of Chronic Obstructive Pulmonary Disease[14,15]. It has been shown to improve respiratory symptoms and gas exchange, reduce the need for intubation and its associated complications, lower mortality and reduce ICU and hospital stay[14,15]. However, the role of NPPV in the heterogenous group of acute hypoxaemic and mixed respiratory failure is less strong[16] and in severe acute asthma, remains controversial[14].

Nevertheless, experience among the paediatric population is more limited. Literature is sparse and the few published data pertaining to non invasive ventilation in acute respiratory failure in children are mainly case reports, case series and uncontrolled studies. To date, there are NO randomized controlled trials or any published guidelines.

Several of the more prominent studies are outlined[5-8]. These uncontrolled case studies and series described and illustrated NPPV as effective and safe in a wide spectrum of respiratory diseases in acute hypoxaemia and hypercarbic respiratory failure in the paediatric population, although the sample size is rather small, the largest being 114 children[7]. These studies include children aged 2 weeks to 20 years, previously well or children with underlying neurological and neuromuscular diseases, cardiac disease, respiratory disorders, immunocompromised states and post surgery with acute respiratory deterioration and insufficiency principally related to pneumonias and atelectasis and also a few, minor stridor and upper airway obstruction and rarer, acute chest syndrome in sickle cell disease. A few case reports also describe the successful use of NPPV in severe acute asthma[10-12] and as a weaning strategy to facilitate extubation[13] in small groups of patient.

NPPV is deemed effective as it was shown to improve respiratory status, gas exchange and oxygenation and with success rates ranging from 77%[7] to over 90%[5,6] at avoiding intubation in these groups of patients. NPPV is regarded as safe as adverse effects were few and minor with short term use, largely skin irritation and ulceration related to interface and this healed rapidly and completely. Mortality was directly related to severe progressive respiratory disease and not considered linked to the use of NPPV in these patients.

Other outcome measures namely morbidity, mortality and survival, length of PICU and hospital stay related to NPPV and cost-effectiveness of NPPV therapy were not discussed or studied.

Although the current limited data demonstrates favourable and promising results, the role and benefits of NPPV in children with Ac Respiratory Failure is still not clearly defined. We await more substantial evidence and large prospective randomized controlled trials in the near future.

As such, it is recommended that a trial of NPPV be applied and attempted in selected groups of patients in EARLY Acute Respiratory Insufficiency by a skilful, experienced, motivated and committed team consisting of an attending paediatrician, intensivist or anaesthetist and medical officers, a nursing team and

physiotherapists with strict continuous monitoring and ongoing reassessments in a paediatric intensive care unit or intermediate high dependency care setup. It is imperative to identify children who fail to make improvements or have deteriorated, and hence, to urgently intubate for conventional ventilation.

NPPV may be also considered as an option in children in whom intubation and invasive ventilation is deemed unsuitable[5], in places where NPPV is available.

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NON-INVASIVE VENTILATION – WHAT ARE THE LIMITS?

Louisa Chan
Malaysia

UNDERSTAND THE PRIMARY PATHOLOGY, DISEASE COURSE AND PREVAILING EVIDENCE

- Accepted indications :acute hypercapnoeic respiratory failure from COAD / APO, chronic hypercapnoeic respiratory failure, hypoxaemic respiratory failure in the immunecompromised
- May be useful (weak evidence) in Acute Asthma (but the indications for intubation remains. An Asthmatic that dies on NIV is unacceptable), weaning from invasive ventilation, post extubation failure (depending on the causes), thoracic trauma

Be well versed with the contraindications

Ensure that underlying medical treatment is also optimal

Note the physiological limits- constellation of worsening vital signs, conscious state and gas exchange

Know the limits of the medical staff ie level of experience and training, location of NIV provision, adequacy of monitoring

Patient's wishes

HENCE PICK THE RIGHT PATIENTS FOR THE THERAPY

- Improve technical aspects eg interface and settings to improve patient tolerance
- Close monitoring: act (Intubate!) before physiologically decompensated
- Watch for complications of NIV

The use of NIV should be subjected to regular audit

PERCUTANEOUS TRACHEOSTOMY – HAS IT LIVED UP TO THE PROMISE?

Syed Rozaidi Wafa
Department of Anaesthesiology, Hospital Universiti Kebangsaan Malaysia, Kuala Lumpur, Malaysia

Tracheostomies is not something new. It has been well described throughout history. The earliest recorded tracheostomy was done nearly 3000 years ago described in the Indian medical book Rig-Veda. But it was only in the last decades did percutaneous tracheostomies (PCT) come about in the ICU setting. This was due to the rapid development in new techniques and equipment, and of course the need for such a procedure to be done in the ICU setting.

The so call perc' promise is to have a procedure that is 'safe', 'easy to do' and 'cost effective'in the hope it would eventually replace or near replace the surgical tracheostomy (ST). There are numerous studies cited with regards to PCT and it's comparison to ST, all with at time opposing results. The few metaanalysis that have been done comparing PCT versus ST have shown the results to be supportive for both techniques but in general PCT seems to have a slight advantage over ST mainly it reduces time, costs and patients' stress as well as allowing better cosmetic results and a lower rate of stoma infection.

MOST TRIALS AND EDITORIALS SUPPORT PCT PROVIDED THE FOLLOWING CAVEATS ARE OBSERVED:

- Patient selection is crucial – pts that are not ideal candidates can pose serious risk.
- No written consent.
- Anatomically difficult patients.
- Previous tracheostomy.
- Significant uncorrected coagulopathy / trombocytopaenia.
- Patients on PEEP >> 10 cmH2O.
- Patients with significant raised ICP.
- Expertise. Procedure is challenging – best left to experienced physicians with thorough knowledge of anatomy of region, surgical / airway experience.

The long-lasting debate between ST and PCT is far off to be solved with both proponents on either side of the fence supporting or opposing either technique. Randomized control trials are difficult to perform, the greatest problem being human anatomical diversity and genetic predisposition to aberrant scars or vascular abnormalities. Therefore like most procedures, a proper database that is regularly reviewed must be kept in order to ensure the "promise" made is fulfilled.

WEANING FROM MECHANICAL VENTILATION: SCIENCE OR ART?

Gavin M Joynt

Department of Anaesthesia and Intensive Care, The Chinese University of Hong Kong, Hong Kong

Mechanical ventilation is not a therapeutic intervention in itself. Even though the life-support provided by mechanical ventilation is considered essential to allow the underlying pathology to resolve, mechanical ventilation itself has been consistently shown to produce harmful effects. Once it is established that mechanical ventilation is no longer necessary, it becomes important to terminate it as soon as possible. The term "weaning" suggests a consistent, stepwise and gradual process of reduction in mechanical ventilatory support. However, removing mechanical ventilation is frequently not a stepwise process but a one step liberation of the patient from mechanical ventilatory support. Both approaches may have a place in clinical practice. Controversy surrounds the process and procedures of weaning or liberation from mechanical ventilation and while it is possible to provide a scientific framework for considering the various options available, considerable judgment is also required when applying competing options.

In particular, clinicians are often wrong when predicting who can or cannot be successfully weaned. It would therefore be very useful to have a set of objective criteria that would allow us to predict when weaning should start and that it would ultimately be successful. Unfortunately there is little evidence to support any such criteria. A number of suggestions have been made, however, and some have moderate predictive power. Assuming a reasonable starting point is chosen, methods of weaning vary from the stepwise reduction of the frequency of mechanical breaths, to immediate liberation following a successful extubation trial (using a T-piece or pressure support trial period). Given several weaning options, considerable judgement is required to minimize weaning failure in individulas. The use of extubation as an end point for weaning may be common, but is not always valid.

UNILATERAL LUNG DISEASE

Andrew D Bersten

Department of Critical Care Medicine, Flinders University and Flinders Medical Centre, Adelaide, South Australia

In the healthy lung ventilation and perfusion are generally well matched, however, there are regional differences with a combination of well ventilated but poorly perfused lung units contributing to alveolar dead space, and well matched lung units, and well perfused poorly ventilated lung units contributing to intrapulmonary shunt. These latter lung units are typically found in dependent lung. In both hydrostatic pulmonary edema and acute lung injury an increase in lung weight results in greater dependent lung collapse and hypoxemia due to increased intrapulmonary shunt.

Acute hypoxemic respiratory failure affecting both lungs is generally responsive to PEEP, with recruitment of alveoli reducing intrapulmonary shunt and improving gas exchange. However, in unilateral lung disease PEEP has complex effects on the distribution of blood flow and ventilation, leading to less predictable improvement in oxygenation.

Common causes of acute hypoxemic respiratory failure from unilateral lung disease include community or hospital acquired pneumonia, aspiration pneumonia, pulmonary contusion, and lobar or lung collapse; less common causes include unilateral pulmonary edema due to asymmetric mitral regurgitation and re-expansion pulmonary edema. Left lower lobe collapse is a common cause of unilateral lung disease, however, this may be the obvious manifestation of unrecognized bilateral lung disease due to the added influence of the mediastinum which tends to reduce the forces keeping the adjacent lung aerated. In addition, unilateral lung conditions such as pneumonia and aspiration may result in acute lung injury and subsequent bilateral lung disease.

Oxygenation may fail to improve, or worsen, following application of PEEP in unilateral lung disease due relative overinflation of the 'healthy' lung leading to a unilateral increase in pulmonary vascular resistance with increased pulmonary blood flow in the diseased lung. This will tend to increase intrapulmonary shunt and reduce the beneficial effects due to recruitment of lung units in the diseased lung. Positioning the patient with the diseased lung down may improve oxygenation. Occasionally differential lung ventilation is required; however, the outcome is usually determined by the underlying disease.

HAEMOSTATIC DRUGS IN THE BLEEDING PATIENT

Ahmad Shaltut Othman

Department of Anaesthesiology, Hospital Alor Setar, Alor Setar, Kedah, Malaysia

Uncontrolled hemorrhage is one of the leading causes of potentially preventable death. Improving our ability to control hemorrhage may represent the next major hurdle in reducing mortality. To decrease the mortality from hemorrhage, new techniques, devices, and drugs for hemorrhage control should be applied.

One of the modern methods of homeostasis is the use of injectable haemostatics as a pharmacologic manipulation of the coagulation cascade. These drugs must be simple to store and use, and must be rapidly effective and directed at life-threatening hemorrhage.

A large body of evidence supports the use of antifibrinolytic agents for the management of bleeding in elective surgery and cardiac surgery patients. The drugs used are tranexamic acid (trans-4-aminomethy lcyclohexane- 1-carboxylic acid) with suggested dosages of 10 to 15 mg/kg followed by an infusion of 1 to 5 mg/kg per hour, aminocaproic acid 100 to 150 mg/kg followed by 15 mg/kg per hour, or (after a test dose) aprotinin 2 million kallikrein inhibitory units (KIU) immediately followed by 500,000 KIU/hour in an intravenous infusion. Antifibrinolytic therapy should be stopped once bleeding has been adequately controlled.

The risk of precipitated thrombosis with the use of antifibrinolytic agents has been of major theoretical concern; however, the Cochrane review of antifibrinolytics cites studies that included more than 8,000 patients and demonstrated no increased risk of either arterial or venous thrombotic events. Anyway a study performed in cardiac surgery patients reported in 2006 showed that there was indeed a risk of acute renal failure, myocardial infarction, heart failure, as well as stroke and encephalopathy with the use of aprotinin.

Perhaps most intriguing is the use of recombinant factor VIIa (rFVIIa) as an intravenous adjunct for hemorrhage control. The use of recombinant activated coagulation factor VII (rFVIIa) is considered if major bleeding persists despite standard attempts to control bleeding and best-practice use of blood components. Although the dose of rFVIIa is truly unknown, an initial dose of approximately 60μg/kg may be appropriate. If hemostasis is not achieved within 30 to 60 min, consideration may be given to a second dose; however, the cost-effectiveness of repeated dosing is questionable given the limited number of experiences reported to date. The use of rFVIIa may increase the risk of thromboembolic complications.

Wide implementation of advances such as pharmacologic modulation of the clotting cascade will have positive effects on patient outcome in acute bleeding. Thus focusing research activity on developing innovative new concepts and technologies that allow control of hemorrhage in the earliest phases of care is very important.

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INTENSIVE INSULIN THERAPY – WHERE ARE WE?

Tan Cheng Cheng

Hospital Sultanah Aminah, Johor Bahru, Johor, Malaysia

Currently, four truly randomized clinical trials evaluating the clinical benefit of tight glucose control using intensive insulin therapy have been performed.

The first randomized trial is the large, single-center prospective randomized controlled study of intensive insulin treatment in the surgical intensive care unit (ICU) by Van den Berghe and colleagues1 published in the New England Journal of Medicine in the year 2001. In this study, a total of 1548 patients were enrolled. Intensive insulin therapy (maintenance of blood glucose at a level between 4.4 to 6.1 mmol/L) reduced overall ICU mortality from 8.0 per cent with conventional treatment (maintenance of blood glucose at a level between 10.0 to 11.1 mmol/L) to 4.6 per cent (p=0.04). The in-hospital mortality was also lower in the intensive insulin therapy group (7.2 versus 10.9%, p=0.01). Intensive insulin therapy also reduced bloodstream infections by 46%, acute renal failure requiring dialysis or hemofiltration by 41%, the median number of red cell transfusions by 50%, and critical illness polyneuropathy by 44%. However episodes of hypoglycaemia (blood glucose <2.2 mmol/L) were more common in the intensive insulin therapy group (5.0 versus 0.7%).

The results were impressive. However critics of the study question the validity of the study. The limitations or concerns[2,3,4,5,6] were:

1. It was not blinded (though the investigators could hardly be blinded), which raises the possibility of both conscious and unconscious bias.
2. Most patients were recruited after surgery (about 63% from cardiac surgery) from a single center
3. Recruited patients received intravenous glucose on arrival at the ICU at 200 to 300 g /day (the equivalent of 2-3 L of 10% glucose per day), an unusual practice among most ICUs
4. This regimen was followed by the initiation of total parenteral nutrition, or enteral feeding, or combined feeding for all patients (presumably including cardiac surgery patients) within 24 hours, also an unusual practice
5. The mortality of the cardiac surgery patients in the conventional treatment group was 5.1%, double the national average in Australia and 5 times that in a hospital where one of the authors (R.B.)7 works
6. The mortality in relation to the severity of illness among patients in the conventional group was relatively high
7. The relative reduction in mortality was extremely high: 34% for a decrease of only 2.8 mmol/L in morning glucose levels
8. The risk of hypoglycaemia with its potential morbidity was 5.0% in the intensive insulin therapy group compared to 0.7% in the conventional treatment group

Indeed a strong critique of the Van den Berghe study suggested that a more reasonable conclusion for that study might have been that administration of excessive intravenous glucose without strong attempts to control its consequences increases mortality in critically ill surgical patients[2].

The other 3 trials are the VISEP study, the GluControl study and "Intensive insulin therapy in the medical ICU" study by Van den Berghe et al who carried out the first trial.

The VISEP (Efficacy of volume substitution and insulin therapy in severe sepsis) trial8 is a prospective randomized multi-center study on the influence of colloid versus crystalloid volume resuscitation and of intensive versus conventional insulin therapy on outcome in patients with severe sepsis and septic shock

This trial from Germany started in April 2003 was designed to randomize 600 patients with medical or surgical severe sepsis to conventional or intensive insulin therapy. However the trial was discontinued permanently after recruitment of 488 patients because of identical mortality rates in the treatment group and in the control groups but a higher incidence of hypoglycemia in the intensive therapy arm (12.1% versus 2.1%).

The GluControl (Glucontrol study: comparing the effects of two glucose control regimens by insulin in intensive care unit patients)[9] is a prospective, randomized, controlled, multi-center study. The study aimed to compare the effects of two regimens of insulin therapy, respectively titrated to achieve a blood sugar level between 4.4 and 6.1 mmol/L (80 and 110 mg/dl, respectively) and between 7.8 and 10.0 mmol/L (140 and 180 mg/dl, respectively) with regards to mortality and morbidity in a mixed population of critically ill patients (around 3000 patients). At the European Society of Intensive Care Medicine 2006 Congress, the results of the GluControl study were presented, concluding that there was no significant benefit of tight glucose control in this study. In fact, the study which started in November 2004 was stopped prematurely because of safety concerns about the high incidence of hypoglycemia beforehand, and could therefore be underpowered for their primary endpoint: mortality.

The "Intensive insulin therapy in the medical ICU"[10] was the second part of Dr Van den Burghe's intensive insulin therapy. The study was carried out on 1200 patients who were considered to need intensive care for at least three days in the medical ICU. The result failed to show any decrease in mortality (40.0% in the conventional-treatment group versus 37.3% in the intensive-treatment group, p=0.33) though there was a significant reduction in morbidity by the prevention of newly acquired kidney injury, accelerated weaning from mechanical ventilation, and accelerated discharge from the ICU and the hospital. The most surprising part of the study was the analysis of the subset of patients who stayed in the ICU for less than three days. Among 433 patients who stayed in the ICU for less than three days, mortality was greater in the intensive insulin therapy group. In contrast, among 767 patients who stayed in the ICU for three or more days, in-hospital mortality in the 386 patients who received intensive insulin therapy was reduced from 52.5 to 43.0 percent (p=0.009) and morbidity was also reduced.

In the era of evidence-based medicine, these 4 randomized controlled trials do not give us conclusive evidence on whether tight glucose control should be the standard of care in the management of critically ill patients and if it is to be the standard of care, what should the target glucose level be.

Fortunately, a large prospective, multi-center study is now well underway, the NICE-SUGAR (Normoglycemia in Intensive Care Evaluation and Survival Using Glucose Algorithm Regulation) study. The NICE-SUGAR study is a multi-centre, open label, randomized controlled trial of blood glucose management with an intensive insulin regimen to maintain blood glucose between 4.5 - 6.0 mmol/L versus an insulin regimen maintaining blood glucose less than 10.0 mmol/L with insulin being infused if blood glucose exceeds 10.0 mmol/L, and adjusted when needed to maintain blood glucose between 8.0 – 10.0 mmol/L. The primary aim of the study is to compare the effects of the two blood glucose targets on 90 day all-cause mortality in Intensive Care patients who are predicted on admission to stay in the ICU for at least one full calendar day. The study aims to enroll a total of 5000 patients, 4500 patients recruited in Australia and New Zealand and 500 in Canada and it has already started in April 2005.

What should we do in the interim period while waiting for the results of this robust study?

Here are a couple of good advices.

1. As Dr Atul Malhotra wrote in the editorial of the same issue of NEJM - " In my opinion, a reasonable approach would be to provide adequate exogenous insulin to achieve target glucose values of less than 8.3 mmol/L , at least during the first three days in the ICU. If critical illness persists beyond three days despite the provision of other proven therapies and resuscitation, a goal of normoglycemia (4.4 to 6.1 mmol/L) could then be considered to maximize the potential benefits. This approach would allow time for a gradual increase in calories in enteral feedings which should minimize hypoglycemic complications". OR

2. As Dr Angus and Abraham suggested last year – "...It may be valuable to remember that, although the evidence for tight glycemic control does not yet support a grade A recommendation, it does appear to be stronger than that for continuing our existing practice of tolerating hyperglycemia. Thus, we should probably explore ways to introduce some form of tight glucose control during this interim period that seems feasible and safe given local considerations. Once better evidence is available, we can modify our plans accordingly."

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HEAD OF BED ELEVATION

Anita Alias

Department of Anaesthesia and Intensive Care, Hospital Melaka, Melaka, Malaysia

INTRODUCTION

Nosocomial pneumonia is a leading cause of morbidity and mortality from hospital-acquired infections with an associated crude mortality rate of approximately 30 percent[1]. These infections also place a strain on ICU resources with an associated average increase in ICU stay of 4.3 days[2]. Prevention of ventilator-associated pneumonia (VAP) is now recognized by the Joint Commission on Accreditation of Healthcare Organizations (JCAHO) as one of its "core measures" for monitoring and improving patient care among the critically ill[3].

The pathogenesis of VAP is generally recognized to consist of two steps: 1) bacterial colonization of the stomach and oropharynx, and 2) subsequent pulmonary aspiration of contaminated secretions[1].

Mechanically ventilated patients are prone to gastric bacterial colonization due to the widespread use of histamine-2 (H2) receptor blockers and proton pump inhibitors for the prevention of gastrointestinal stress ulceration. Indwelling nasogastric and nasoenteric feeding tubes decrease the competance of the lower esopheageal sphincter, increasing the potential for aspiration. Strategies to reduce the incidence of VAP are typically aimed at reducing the colonization of the aerodigestive tract, decreasing the incidence of aspiration, or both.

EVIDENCE DEFINITIONS

- Class I: Prospective randomized controlled trial.
- Class II: Prospective clinical study or retrospective analysis of reliable data. Includes observational, cohort, prevalence, or case control studies.
- Class III: Retrospective study. Includes database or registry reviews, large series of case reports, expert opinion.
- Technology assessment: A technology study which does not lend itself to classification in the above-mentioned format. Devices are evaluated in terms of their accuracy, reliability, therapeutic potential, or cost effectiveness.

LEVEL OF RECOMMENDATION DEFINITIONS

- Level 1: Convincingly justifiable based on available scientific information alone. Usually based on Class I data or strong Class II evidence if randomized testing is inappropriate. Conversely, low quality or contradictory Class I data may be insufficient to support a Level I recommendation.
- Level 2: Reasonably justifiable based on available scientific evidence and strongly supported by expert opinion. Usually supported by Class II data or a preponderance of Class III evidence.
- Level 3: Supported by available data, but scientific evidence is lacking. Generally supported by Class III data.

Maintenance of the head at greater than 30-45° has been suggested as a clinically useful method for reducing a patient's risk of VAP and ICU mortality. Head of bed elevation also reduces intracranial pressure (ICP) and optimizes cerebral perfusion pressure (CPP) in patients with closed head injury[4].

LITERATURE REVIEW

Torres et al. performed a prospective, randomized, two-period crossover trial in 19 mechanically ventilated medical ICU patients in which gastric secretions were radiolabelled with Technetium-99m sulphur colloid[5]. Patients were randomly placed in either the supine or semirecumbent (45-degree angle) position and the presence of radioactivity in bronchial secretions was subsequently assessed. All patients had nasogastric tubes in place. Forty-eight hours later, the study was repeated in each patient using the alternate position. All patients demonstrated an increase in radioactivity count illustrating that pulmonary aspiration of gastric secretions occurs, regardless of patient position. The radioactivity recovered in the endobronchial samples

of semirecumbent patients, however, was significantly lower than that of supine patients (p=0.036) confirming that head of bed elevation is significantly protective. The authors concluded that supine positioning promotes the development of VAP and that semirecumbent positioning of mechanically ventilated patients is a simple and effective means to minimizing aspiration of gastric contents (Class II).

Kollef carried out a prospective descriptive cohort study of 277 mechanically ventilated patients of whom 43 developed VAP while 234 did not[6]. Univariate and multivariate analyses were subsequently performed to identify risk factors that were independently associated with VAP and mortality. Age, organ failure, prior antibiotic administration, and supine head positioning (30-degree angle) during the first 24 hours of mechanical ventilation were all independently associated with VAP in multivariate analysis.

Supine position and organ failure were independently associated with patient mortality in multivariate analysis. VAP occurred in 34% of supine patients and 11% of semirecumbent patients (p<0.001). ICU mortality was 30% in supine patients and 8.9% in semirecumbent patients (p<0.001) (Class II).

Drakulovic, Torres, et al. subsequently performed a prospective, randomized trial of supine vs. semirecumbent (45-degree angle) positioning in the prevention of nosocomial pneumonia among 86 mechanically ventilated medical ICU patients[7]. The study was terminated early during a planned interim analysis due to the finding of a statistically significant difference in pneumonia between patient groups. Microbiologically confirmed pneumonia occurred in 5% of semirecumbent patients and 23% in supine patients (p=0.018; 95% CI 4-33%). The risk reduction associated with semirecumbent positioning was 78%. In a multivariate analysis of risk factors associated with development of pneumonia, enteral nutrition (odds ratio 11.8) and supine body position (odds ratio 6.1) were identified as significant independent risk factors. The study showed a trend towards a reduction in mortality (18% in semirecumbent patients and 28% in supine patients (p=0.289)), but the trial was not powered to detect such a difference if present (Class I).

Durward et al. performed a prospective evaluation of the impact of supine vs. various semirecumbent positions (15, 30, and 60 degrees) on ICP, CPP, and CVP in patients with a Glasgow Coma Score of ≤ 8 and traumatic closed head injury or near-drowning[4]. ICP was highest in all patients in the supine position and decreased significantly at 15 and 30 degrees of elevation while maintaining CPP and cardiac index. Elevation to 60 degrees caused a fall in CPP and cardiac index, an increase in CVP, and a variable response in ICP (Class II).

In other related studies; Cerebrovascular dynamics with head-of-bed elevation in patients with mild or moderate vasospasm after aneurysmal subarachnoid hemorrhage by Patricia A. Blissitt, et al. In patients with aneurysmal subarachnoid hemorrhage, elevation of the head of the bed during vasospasm has been limited in an attempt to minimize vasospasm or its sequelae or both. Consequently, some patients have remained on bed rest for weeks. The objectives were to determine how elevations of the head of the bed of 20° and 45° affect cerebrovascular dynamics in adult patients with mild or moderate vasospasm after aneurysmal subarachnoid hemorrhage and to describe the response of mild or moderate vasospasm to head-of-bed elevations of 20° and 45° with respect to variables such as grade of subarachnoid hemorrhage and degree of vasospasm. A within-patient repeated-measures design was used. The head of the bed was positioned in the sequence of 0°-20°-45°-0° in 20 patients with mild or moderate vasospasm between days 3 and 14 after aneurysmal subarachnoid hemorrhage. Continuous transcranial Doppler recordings were obtained for 2 to 5 minutes after allowing approximately 2 minutes for stabilization in each position. No patterns or trends indicated that having the head of the bed elevated increases vasospasm. As a group, there were no significant differences within patients at the different positions of the head of the bed. Utilizing repeated-measures analysis of variance, P values ranged from .34 to .97, well beyond .05. No neurological deterioration occurred. Conclusions, in general, elevation of the head of the bed did not cause harmful changes in cerebral blood flow related to vasospasm.

ACCURACY OF CLINICAL EVALUATION OF HEAD OF BED ELEVATION

Nasir Awan; Chanaka Seneviratne; Zenia Ceniza; Taek S Yoon; et al

Chest; Oct 2005; 128, 4

PURPOSE
Maintaining head of bed elevation (HOBE) greater than 30° has been shown to decrease the incidence of ventilator associated pneumonia. We prospectively studied the accuracy of clinical estimation of HOBE.

METHODS
HOBE was set at two levels 30° and 45° with the use of a protractor. Nurses and physicians were asked to determine the HOBE angle. The position of the observer, whether the estimation was performed at the foot or the side of the bed, was evaluated.

RESULTS
One hundred and fifty nurses and fifty physicians participated in the study. Overall, when HOBE was 30°, the average clinical estimation was 50° (P<0.001). When the HOBE was 45°, the average clinical estimation was 70° (P<0.001). When the observer was at the foot of the bed, only 32 (16%) correctly estimated the position versus 90 (45%) of the observers correctly estimated bed position from the side of the bed (P = 0.001). There was no difference between physicians and nurses in the accuracy of the clinical estimation of HOBE.

CONCLUSION
Clinical evaluation of the angle of HOBE tends to overestimate the angle of elevation. Measurement of the HOBE from the side rather than the foot of the bed are more accurate.

SUMMARY
Nosocomial pneumonia is commonplace in the intensive care unit (ICU) and is associated with pulmonary aspiration of contaminated gastric secretions. Semirecumbent patient positioning (head of bed elevation) significantly decreases the incidence of both pulmonary aspiration as well as subsequent development of bacterial pneumonia and may be associated with reduced ICU mortality.

RECOMMENDATIONS

Level 1
The head of a patient's bed should be elevated to a minimum of 30 degrees or greater, as clinically tolerated, at all times to reduce aspiration of contaminated oropharyngeal secretions and subsequent development of ventilator-associated pneumonia (VAP).

Level 2
The head of a patient's bed should be elevated to a minimum of 30 degrees or greater, as clinically tolerated, at all times to reduce patient mortality. In patients with closed head injury, the head of a patient's bed should be elevated to 30 degrees at all times to reduce intracranial pressure (ICP) and maintain cerebral perfusion pressure (CPP).

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REDUCING MEDICATION ERRORS

V Sivasakthi
Department of Anaesthesia and Intensive Care, Hospital Melaka, Melaka, Malaysia

The issue of medication errors has been the focus of scrutiny because these contribute directly to patient morbidity and mortality. A desire to provide patients with optimum and safe care fuels practitioners to create strategies to reduce the likelihood of administration errors occurring.

A report entitled "Building a safer NHS for patients – Improving Medication Safety" reviewed the causes and frequency of medication error and identified models of good practice to improve medication safety.

Causes of errors were complex and were divided into systems errors and those caused by individual health care professional issues. It was found that systems weakness that predisposed to human errors were important and recommended checks and error traps that should be built into all medication processes, including prescribing, dispensing and drug administration. Introduction of well designed information management solutions to reduce the scope for mistakes and lapses in medication were recommended. Mostly, medication errors were attributed to documentation issues, including: illegible handwriting, misunderstanding abbreviations, misplaced decimal points, misreading and misinterpreting written orders. Several human factors were attributed to potential causes of medication errors, including: stress, fatigue, knowledge and skill deficits. Environmental factors, namely, interruptions and distractions during the administration of medications, also attributed to potential errors.

Key steps for safer prescribing included active management and review of long term repeat prescribing; clear treatment plans shared with all professionals involved in a patient's care; and double checking of all complex dose calculations. Other specific interventions include standardising prescription writing and rules and eliminating abbreviations and limiting verbal medication orders.

Greater use of information technology, including implementation of electronic care records and effective electronic prescribing systems Computerized Physician Order Entry (CPOE), Personal Digital Assistant (PDA) and internet are useful. Appropriate training should be provided for all health staff in all settings where medicines are given. Drugs should be checked by a second person in high risk circumstances, including intravenous infusions and complex calculations. There should be development of clear procedures for the documentation of allergies to the drugs and the use of alerts for allergies. Training and assessment of competence in paediatric drug therapy, including calculations of doses and infusion rates, should be introduced to reduce the risk of drug errors in children.

Additional general recommendations embraced by most organisations include adopting a systems oriented approach to medication errors, creating a culture of safety and improving medication error identification and reporting and also supporting nurses in providing best practice.

NURSE-BASED PROTOCOLS

Mahamarowi Omar
Department of Anaesthesiology, School of Medical Sciences, Universiti Sains Malaysia, Kubang Kerian, Kelantan, Malaysia

Intensive care (IC) involves management of patients with vast varieties of problems, and that they are associated with rapid and dynamic physiological changes. As such it poses a major challenge, and thus the goals of successful ICU management would include continuous close observation and monitoring of patients, having good clinical judgment and appropriate interventions. Variations in this clinical management have been associated with suboptimal outcome and in increased cost.

Management based on protocols is a very useful strategy to standardize ICU care, principally by limiting variation, thereby it would be able to reduce complications, decrease length of stay, and improve outcome and costs.

However, the nature of intensive care makes it difficult to conduct blinded, randomized controlled clinical trials, to identify the specific type of science required for evidence-based medicine, and for protocol development and implementations. Furthermore, there are so many protocols in existence, with many variations between different hospitals and from one country to another.

ICU nurses are highly professional staff with adequate level of competency and thus would be able to intuitively know about intensive care principles. The Nurse-Based protocol approach to ICU patient care would provide the potential for widespread application as it is not routinely feasible for the clinician to be constantly present at the bedside.

ICU nurse-based protocols have been successfully implemented in various areas including sedation, analgesia and paralyzing methods, enteral feeding, tight glycaemic control, weaning from ventilator, antibiotic policies, infection control and deep vein thrombosis prophylaxis.

COMFORT AND DISTRESS IN ICU

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

The first intensive care units were set up in the 1940s – 50s with the priority of saving lives. Today, 20% of all patients dying in the U.S., die in an ICU. In a large North American study, life support was limited in 70% of ICU patients who died. Therefore, ensuring comfort and dealing with end-of-life must be part of the management of the critically ill.

The ICU is a stressful environment for the patient. Unfamiliar environment with too much noise, bright light, bustling activity, and physical disturbance is disturbing to the patient. Pain and fear are ever-present for patients. There is lack of communication with patients who are sedated. The patient who is stressed may present with depression, disorientation, hallucinations or even manic behaviours. It is important to provide comfort to the all the patients in the ICU by providing emotional support, effective communication and symptom control e.g. for pain, anxiety, sleep disturbance and hunger/ thirst.

Patients' family members may experience emotional stress levels as great as that of the patient. The most pressing need of family members is to receive clear, understandable and honest information about the patients' condition. Family members want reassurance concerning comfort measures and notification of deterioration of their loved one's status. Satisfaction of family members of patient who died in ICU is shown to be associated with adequate communication, respect and compassion shown to patient and family members and good decision making by the physician.

Nurses are important figures in the ICU. They are the link between patients and patients' families and also between patients and physicians. The ICU environment can be equally stressful for the nurses working there. High workload and poor staffing ratios are associated with burnout, low job satisfaction and increased stress among nurses. Feelings of helplessness when watching a patient suffer, dealing with death and dying, lack of support from colleagues and interpersonal conflicts are some of the other stressors faced by the nursing staffs. Increased stress among nurses is related to poor patient care and adverse patient events. Some of the methods that have successfully reduced the level of stress among the nurses include: regular staff meetings and discussions to communicate feelings and improve support; organised and efficient work functions and environment; frequent in-service educational sessions to improve skills and confidence; scheduled rotation of unit assignments; and adequate staffing.

"To cure sometimes, to relieve often, to comfort always"

FREE PAPER PRESENTATIONS
23 JUNE 2007, SATURDAY

A RELATIONSHIP OF VASCULAR PEDICLE WIDTH AND VENTILATOR PARAMETERS IN VENTILATED PATIENTS

L Mohd Fahmi[1], M Rhendra Hardy[2], Y Azura Sharena[3], Y Rohaizan[4], N M Nik Abdullah[5]
[1,2,5]Department of Anaesthesiology, [3,4]Department of Radiology, School of Medical Sciences, Universiti Sains Malaysia, Kubang Kerian, Kelantan, Malaysia

BACKGROUND
Chest radiograph is the most common diagnostic imaging technique in Intensive Care Unit, particularly important in assessment of patient's intravascular volume. However, this technique is underutilized and vascular pedicle width will be one of the solutions.

OBJECTIVES
The objective of this study is to determine relationship of Vascular Pedicle Width (VPW), Positive End Expiratory Pressure (PEEP) and Peak Inspiratory Pressure (PIP).

METHODOLOGY
Prospective, randomized study on adult patients whom were ventilated in the Intensive Care Unit and Neuroscience Intensive Care Unit of Hospital Universiti Sains Malaysia between May 2006 until December 2006. One hundred and forty patients were chosen based on inclusion and exclusion criteria. PEEP and PIP were measured within one hour after chest radiograph taken. VPW was measured on digitalized chest radiograph by Radiology Researcher at separate occasion without clinical data.

RESULTS
There was significant linear relationship between PEEP and VPW ($p<0.05$, CI 0.00 – 0.97 cmH2O) with those who had PEEP of 10 cm H2O have VPW wider for 4.9mm. However, there was no significant linear relationship between PIP and VPW. There was no interaction between independent variables and multicollinearity problem.

CONCLUSION
Vascular Pedicle Width can be used in Intensive Care Unit, even in ventilated patients. VPW is easy to interpret, practical, non invasive, and cost efficient. However, level of PEEP should take into consideration. Therefore, measurement of VPW in ventilated patient can play a crucial role in Intensive Care Unit and act as added value for chest radiograph.

P 02

BLIND BRONCHIAL ASPIRATE CULTURE AND SENSITIVITY: AN INVESTIGATION TO BE TIED WITH CLINICAL GROUNDS

Tan Cheng Cheng, Mahazir Kassim, Mohd Faizal Zuhri, S Balan
Department of Anaesthesiology and Intensive Care, Hospital Sultanah Aminah Johor Bahru, Johor, Malaysia

OBJECTIVE
To study the blind bronchial aspirate culture and sensitivity done in the intensive care unit (ICU) in Hospital Sultanah Aminah and its clinical significance.

METHODS
Blind bronchial aspirate culture and sensitivity (C&S) were done as part of the septic workout in ICU patients suspected of having infection. The results were recorded on a culture and sensitivity form. Those results which grew organisms were then decided on clinical grounds and chest radiography to see if they were real infections, that is pneumonia or contaminations/colonisations. A review of these results in year 2005 was carried out. Cultures done on admission to ICU were excluded.

RESULTS
There was a total of 1012 admissions. After exclusion of cultures done on admission, 506 blind bronchial aspirate cultures were available for analysis. Of these 506 blind bronchial aspirate cultures, 377 (74.5%) cultures grew organisms. Of the 377 cultures with growth, 133 (35.2%) isolates were real infections while 244 (64.7%) were contaminations/ colonisations. The top 3 organisms for real infections were Acinetobacter, Pseudomonas and Klebseilla, while the top 3 organisms for contaminations/colonisations were Acinetobacter, Klebseilla and Pseudomonas. There were no statistically significant difference in terms of sensitivity of real or contaminated/colonized organisms for both Acinetobacter and Klebseilla. For Pseudomonas, 66% of the organisms which caused real infections were multi-resistant while 45.3% of contaminate/colonized organisms were multi-resistant ($p<0.05$).

CONCLUSION
About 75% of blind bronchial aspirate C&S done as part of septic workout grew organisms but only about 35% of those organisms grown caused pneumonia. Hence blind bronchial aspirate C&S had a high yield but its significance needs to be correlated with clinical parameters and chest radiography.

P 03

POSITIVE BLOOD CULTURE AND SENSITIVITY AND ITS CLINICAL SIGNIFICANCE

Tan Cheng Cheng, Mahazir Kassim, Mohd Faizal Zuhri, Murniati Mustafa, S Balan
Department of Anaesthesiology and Intensive Care, Hospital Sultanah Aminah Johor Bahru, Johor, Malaysia

OBJECTIVE
To study the blood culture and sensitivity done in the intensive care unit (ICU) and to determine its clinical significance.

METHODS
Blood culture and sensitivity (C&S) results done in ICU were recorded on a culture and sensitivity form. Those results which grew organisms were decided on clinical grounds to see if they were real infections or contaminations/colonisations. A review of these results in year 2005 was carried out.

RESULTS
There was a total of 634 blood C&S done in the ICU in the year 2005. Of these 634 blood C&S, 360 (56.8%) cultures grew organisms. Of the 360 positive cultures, 74 (20.6%) cultures yielding a total of 101 organisms were identified to be contaminated or of no clinical significance. Of the 101 contaminated organisms, gram negative bacilli made up 46.5% followed by gram positive cocci which made up 36.7%. Of the remaining 286 positive cultures yielding 327 organisms, the top 3 organisms were Acinetobacter (20.8%), Pseudomonas (19.6%) and Klebseilla (18.0%). There was no statistical significance between infected and contaminated blood yielding more than one organism ($p=1$). With regards to resistant strains for the 3 common organisms, 51.5% of Acinetobacter, 48.4% of Pseudomonas and 37.3% of Klebseilla were multi-resistant.

CONCLUSION
About 21% of the blood C&S done in our ICU were contaminated and almost half were due to gram-negative bacilli. Multi-resistant Acinetobacter was our most common infecting organism.

IMPACT OF EARLY ANTIBIOTICS ON SEVERE SEPSIS – ARE WE DOING A GOOD JOB?

Shahla Siddiqui, Nawal Salahuddin, Adeel Raza, Junaid Razzak
Department of Anaesthesiology, Aga Khan University, Karachi, Pakistan

BACKGROUND
Despite improvements in technology and health care services, mortality rates from severe sepsis have remained unchanged over the past few decades. Exciting new data are emerging about the benefits of early, aggressive management in the Emergency room. The objective of this study was to assess the promptness of antibiotic administration to patients presenting with sepsis and the effects on survival and length of hospitalization.

METHODS
Consecutive, adult patients presenting with Systemic Inflammatory Response Syndrome (SIRS) to the emergency department of the Aga Khan University hospital were enrolled in a prospective, observational study from February to June 2006. Source of sepsis, timing and appropriateness of antibiotic administration, resuscitative measures in the ER were recorded. The patient was followed until death or discharge. Univariate, multivariate regression modeling and one-way ANOVA were used to examine the effects of various variables on survival and for significant differences between timing of antibiotic administration and survival; two-sided p values <0.05 were considered significant.

RESULTS
One hundred and eleven patients were enrolled. Severe sepsis was present in 52% patients; the most frequent organism isolated was Salmonella typhi (30%). Overall mortality was 35.1%. One hundred (90.1%) patients received intravenous antibiotics in the Emergency room; average time from triage to actual administration was 2.48 (± 1.86) hours. The timing of antibiotic administration was significantly associated with survival (F statistic 2.17, p 0.003).Using a Cox Regression model, we were able to demonstrate that survival dropped acutely with every hourly delay in antibiotic administration. On multivariate analysis, use of vasopressors (adjusted OR 23.89, 95% CI 2.16,263, p 0.01) and Escherichia coli sepsis (adjusted OR 6.22, 95% CI 1.21,32, p 0.03) were adversely related with mortality.

CONCLUSIONS
We demonstrated that in the population presenting to our emergency room, each hourly delay in antibiotic administration was associated with an increase in mortality.

TRIAL OF INHALED ALPROSTADIL TO IMPROVE HYPOXIA AND PULMONARY HYPERTENSION – A PRELIMINARY REPORT

S Siddiqui[1], N Salahuddin[2], S Khan[3], R Manasia[6], S Zubair[4], A Gilani[5]
[1]*Department of Anaesthesia* , AKUH, [2]*Department of Medicine*, AKUH, [3]*Research Assistant*, AKUH, [4]*Pharmacist*, AKUH, [5]*Professor Pharmacology*, AKUH, [6]*Head Nurse*, ICU, AKUH

INTRODUCTION
Aerosolized PGE1 (Alprostadil) can result in a selective pulmonary vasodilatation without affecting the systemic blood pressure as shown in preliminary studies/case reports. No large trials exist for this type of use of the drug so far. Furthermore, aerosolized PGE2 can improve gas exchange and pulmonary shunt as occurs in Adult Respiratory Distress Syndrome (ARDS).

METHODS
This is a randomized, prospective, double blinded study which was funded by the PMRC*. A research assistant together with the Principal Investigator recruited 21 patients in the multidisciplinary ICU at AKUH. Inclusion criteria included all adult patients with ARDS and/or PA pressures > 35 mmHg on PA catheter or suspected on clinical grounds. A transthoracic echo was performed as well as recording PA pressures on echo and P/F ratio prior to drug administration. Subsequently each patient was randomized in the pharmacy in a block computerized randomization to either case or control. Cases received nebulized PGE1 over 30 minutes in the ICU. Following this the echo and arterial blood gases were repeated.

RESULTS
Our results on the paired T -test before and after the drug showed a p -value of 0.035 for PaO2/ FiO2 ratio and a p-value of 0.000 for PA pressures, both of which were strongly significant.

CONCLUSION
Our study is a preliminary report which shows that there is a significant improvement in PA pressures and oxygenation after administration of nebulised alprostadil.

ACKNOWLEDGEMENTS
Pakistan Medical Research Council*, S. Fatimi, A. Raza, Z. Bhimani, S. Dhakum, L. Sheikh, Q. Hoda.

PROGRAMME
24 JUNE 2007, SUNDAY

0830 – 0930	**PLENARY 4** (Sabah) *Chairperson: Syed Rozaidi Wafa* Shock – Revisited (pg 55) *Anthony McLean*	
0930 – 1030	**PLENARY 5** (Sabah) *Chairperson: Syed Rozaidi Wafa* Acute pulmonary edema: The non-invasive ventilation paradox (pg 56) *Andrew Bersten*	
1030 – 1100	TEA / TRADE EXHIBITION	
1100 – 1300	**SYMPOSIUM 10** (Sabah) **Neurologic** *Chairpersons: Mohd Basri Mat Nor / Sekar Shanmugam* • Status epilepticus: Monitoring and treatment (pg 57) *Santhi Puvanarajah* • Improving neurological outcome after CPR (pg 58) *M Kauthaman* • Monitoring for optimal sedation in the ICU (pg 59) *Kwek Tong Kiat* • The use of muscle relaxants in Intensive Care Unit (ICU) (pg 60) *Amarawati Mohd Yatim*	**SYMPOSIUM 11** (Selangor) **Intensive Care For Nurses (2)** *Chairpersons: Anita Alias / Rohana Mohamed* • Ventilator-associated pneumonia (pg 61) *Shanti Rudra Deja* • Prevention of catheter-related infections (pg 62-66) *Marlizan Md Yusoff* • Post-operative respiratory complications *Noor Airyn Ibrahim* • Challenges of enteral feeding in the critically ill *Jenny Tong May Cenk*
1300 – 1400	LUNCH (Sarawak)	

SHOCK – REVISITED

Anthony McLean
Nepean Hospital, Penrith, Sydney, New South Wales, Australia

The word SHOCK has a myriad of different definitions with only one pertaining to the medical lexicon. The medical term 'shock' is described in the Concise English Dictionary as "an acute medical condition associated with a fall in blood pressure, caused by loss of blood, severe burns, sudden emotional stress etc". Physicians, particularly critical care physicians would dismiss this definition as totally inadequate, yet agreement by clincians on a generally acceptable definition is elusive. The Merck manual describes it as "a state in which blood flow to, and perfusion of, peripheral tissues are inadequate to sustain life because of insufficient cardiac output or maldistribution of peripheral blood flow, usually associated with hypotension and oliguria". Perhaps more acceptable to clinicians this definition is possibly misleading because if shock can occur without hypotension then how do we recognise it? The term shock is applied to conditions where there is threatened or actual global or regional ischaemia, and the conditions that can bring this about are multiple, including acute cardiac decompensation from whatever cause, and circulatory collapse where the cardiac function may be normal or even increased. Perhaps therefore a better definition of shock is 'inadequate cellular perfusion and /or oxygen uptake with consequent tissue hypoxia and organ failure'. The clinical parameters such as hypotension, blood volume reduction, peripheral limb hypoperfusion, confusion and impaired consciousness and oliguria with deteriorating renal function, don't actual define shock but do assist in diagnosing the presence of shock.

Whatever the definition of the shock there is likely to be agreement that identification of the shocked patient should be rapid and based on clinical findings. The initial diagnosis is suggested by decreased urine output, poor limb and skin perfusion, signs of cerebral hypoperfusion and haemodynamic instability which usually includes, although not always, hypotension. Early diagnosis and subsequent early goal directed therapy has been demonstrated to improve organ function and reduce mortality. This approach has been incorporated into treatment algorithms best exemplified by the 'Saving Sepsis Campaign' .

A number of approaches on describing types of shock have been promolgated over the years but the favoured one dates back to that proposed by Weil over 30 years ago: namely the division into cardiogenic, hypovolemic, distributive (septic) and obstructive.

Still associated with a high mortality it is not surprising that controversy exists in regard to a number of areas including diagnosis, monitoring and treatment. Some current important debates are in regard to the importance of serum lactate levels, newer techniques on monitoring peripheral perfusion, at which level of anaemia a blood transfusion should be given, choice of vasoactive agents, and the place of naloxone in the management of shock.

ACUTE PULMONARY EDEMA: THE NON-INVASIVE VENTILATION PARADOX

Andrew D Bersten

Department of Critical Care Medicine, Flinders University and Flinders Medical Centre, Adelaide, South Australia

Non-invasive positive pressure ventilation (NIPPV) has become established therapy for patients with acute pulmonary edema (APE). Following early empirical use of mask continuous positive airway pressure (CPAP), we found it resulted in faster improvement in respiratory and cardiovascular physiology and reduced intubation rate compared to comparable inspired oxygen concentration (FiO2), with both groups receiving usual medical care[1]. Numerous subsequent studies have confirmed these findings, and meta-analysis of these also demonstrate benefit; CPAP reduces mortality (RR 0·59, 95% CI 0·38–0·90, NNT 10), and while bilevel PAP tends to reduce mortality (RR 0·63, 95% CI 0·37–1·10) there is no difference between bilevel and CPAP[2]. However, there is weak evidence of an increase in the incidence of new myocardial infarction with bilevel ventilation versus CPAP was recorded (RR 1·49, 95% CI 0·92–2·42).

The physiologic basis for the effectiveness of NIPPV includes reduced LV preload and LV afterload, lung recruitment and increased FRC with improved oxygenation and reduced work of breathing. While the initial basis for APE is a sudden elevation in pulmonary microvascular pressure (Pcap) resulting in hydrostatic lung injury, this also leads to lung injury which commonly occurs on the background of homeostatic changes in the lung due to chronic heart failure. There is increased bidirectional protein flux across the alveolar epithelium, and alveolitis with elevated levels of plasma TNF-_ that persist for some days[3,4]. Consequently hydrostatic pulmonary edema provides a fertile basis for lung injury that may be exacerbated by ventilatory stress (hydrostatic lung injury [HLI]; two hit hypothesis). If APE fails to improve within 12 hours intubation and ventilation should be considered as a strategy to reduce the impact of HLI.

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STATUS EPILEPTICUS: MONITORING AND TREATMENT

Santhi Puvanarajah

Department of Medicine, Hospital Kuala Lumpur, Kuala Lumpur, Malaysia

Status epilepticus (SE) is a neurological emergency that demands prompt and aggressive treatment by the managing team of medical professionals at any hospital. This is because the risk of mortality and morbidity rises with prolonged duration of continuous seizures and the cerebral damage incurred. It is essential that both overt and electroencephalographic discharges are controlled in these situations.

General lines of treatment include stabilization of cardiorespiratory function, arranging the necessary investigations, initiation of antiepileptic medication, correcting metabolic abnormalities and closely monitoring and correcting for hypotension, cardiac arrhythmias, hyperthermia, lactic acidosis, rhabdomyolysis and cerebral edema. Electroencephalographic (EEG) monitoring plays an important role in assessment of response to antiepileptic medication and the depth of anesthesia induced.

Establishing protocols for SE treatment and the wide dissemination and proper implementation of such clinical practice go a long way to reducing mortality and morbidity otherwise associated with this condition.

IMPROVING NEUROLOGICAL OUTCOME AFTER CPR

Kauthaman Mahendran
Department of Medicine, Melaka Hospital, Melaka, Malaysia

Although 'CPR' relates to heart and lungs, the aim is survival of a functional brain. The most effective way of achieving this is providing prompt and effective CPR. Towards this end we need to lobby and work towards realizing the dream of a strong chain of survival in our respective communities. Questions we should be asking ourselves include:

1. How much have we done to encourage public CPR awareness and training?
2. Have we worked hard enough to create public access defibrillation (PAD) sites?
3. How effective is our ambulance response service?
4. How effective is our CPR training program for in-house health care staff.?
5. Do we have the well maintained basic equipment in the proper places; bag-mask devices, ETTs, LMAs, AEDs, ETC02 monitors, drugs?
6. Are CPR audits in place in our institutions?

Post resuscitation care is the second determinant of neurological outcome. High quality scientific evidence is scant in this area. Numerous treatments have been proposed. Interventions that will be discussed include: creating mild hypothermia, treating hyperthermia, ventilation induced normocarbia, BP control, glucose control, seizure control, dysrhythmia control, fibrinolytics, erythropoetin and coenzyme Q10.

It is not easy to predict neurological outcome in comatose post resuscitation victims. Educated guesses can be made from knowledge of the victim's pre morbid status, medical diagnosis, cardiac rhythm at arrest, delay in CPR, and time to return of spontaneous circulation (ROSC). Objective markers are continually being evaluated. Some key physical signs, serum markers, somato-sensory evoked potentials and EEGs will be discussed.

The third level will be rehabilitation of the survivor. Nurses, physiotherapists, occupational therapists and rehabilitation medicine experts play their parts here.

It should not be forgotten that prevention of conditions that have a high probability of leading to a cardiac arrest situation should receive the most of our time, money and energy. This begins with encouraging a healthy lifestyle, recognizing and controlling risk factors and close monitoring of in-patients. This may, sadly, be still wanting.

MONITORING FOR OPTIMAL SEDATION IN THE ICU

Kwek Tong Kiat
Department of Anaesthesiology, Tan Tock Seng Hospital, Singapore

The critically ill patient is often anxious, confused, uncomfortable and in pain in our intensive care units (ICU). These patients will require sedation, analgesia or both for at least part of their stay in the ICU. Sedation can minimize agitation, promote synchrony with mechanical ventilation and facilitate nursing and procedures performed at the bedside.

Inappropriate administration of sedation can lead to potentially serious consequences. Insufficient sedation may cause life-threatening agitation precipitating myocardial ischaemia or ventilator dyssynchrony. Excessive sedation has been shown to prolong the duration of mechanical ventilation, increase the incidence of ventilator-associated pneumonia, and increase the ICU length of stay. In a recent report by Kress et al., it was found that daily interruption of the sedative infusion in the ICU led to shorter durations of mechanical ventilation and ICU stays without increased complications.

The clinical practice guidelines of the Society of Critical Care Medicine emphasise the need for goal-directed delivery of sedation and recommends the use of a validated sedation assessment scale to monitor and titrate sedation in the ICU. The ideal sedation scale should be simple to compute, have adequate well-defined categories for the range of sedation and agitation noted and have good reliability and validity when used in ICU patients.

The Ramsay Scale, first developed in 1974, remains the most widely used sedation scale in the ICU. This is despite the scale lacking behavioural descriptors and definitions to separate the various levels. This and more recently introduced scales such as the Sedation Agitation Scale and the Richmond Agitation Sedation Scale will be discussed.

Objective testing of the level of sedation may be useful during very deep sedation or when neuromuscular blockade is used, preventing the use of sedation scales. These modalities include monitoring plasma drug concentrations, heart rate variability, continuous EEG and the Bispectral Index monitor.

THE USE OF MUSCLE RELAXANTS IN INTENSIVE CARE UNIT (ICU)

Asmarawati Mohamad Yatim
Department of Anesthesia and Intensive Care, Hospital Tengku Ampuan Afzan, Kuantan, Pahang, Malaysia

The use of muscle relaxants in the ICU for reasons other than the placement of an endotracheal tube is often guided by individual practitioner preference than by standards based on evidence-based medicine. Common reasons for the use of muscle relaxants in the ICU are to facilitate mechanical ventilation, manage raised intracranial pressure, treatment of muscle spasms and decrease oxygen consumption.

None of the muscle relaxants currently available satisfy all the criteria for an ideal agent in the ICU. The commonly used nondepolarizing agents today are the aminosteroidal compounds (pancuronium, vecuronium, rocuronium) and the benzylisoquinolinium compounds (atracurium, cisatracurium). Monitoring the depth of neuromuscular blockade is highly recommended. Patients receiving neuromuscular blocking agents (NMBA) should be assessed both clinically and by train-of-four (TOF) monitoring with the aim of maintaining one or two twitches.

Prolonged recovery following the use of NMBAs is the most common complication of pharmacologic neuromuscular blockade. The usual presentation is increased in the time to recovery, often 50-100% longer than predicted by pharmacologic parameter. This can be due to accumulation of the parent NMBA, active metabolites or drug-drug interactions with NMBAs. There are other forms of skeletal muscle weakness with multifactorial etiologies linked to the prolonged used of NMBA which includes acute quadriplegic myopathy syndrome and critical illness polyneuropathy.

In the appropriate ICU patient population and for specific indications, neuromuscular blockade can be useful for optimal management. However the benefits and the associated risks of neuromuscular blockade must be reviewed constantly so that potential adverse effects can be minimized.

VENTILATOR ASSOCIATED PNEUMONIA

Shanti Rudra Deva
Department of Anaesthesia and Intensive Care, Hospital Kuala Lumpur, Kuala Lumpur, Malaysia

Ventilator associated pneumonia or VAP is defined as pneumonia occurring in patients more than 48 hours after endotracheal intubation and mechanical ventilation. It is one of the more common ICU acquired infection with an incidence of about 10 – 20%.

The development of VAP is associated with prolonged ventilation, ICU as well as hospital stay. This inevitably would increase the cost thereby representing a major health care burden. Prevention of VAP is the cornerstone in reducing the incidence and other adverse events.

An understanding of the pathogenesis of VAP would help tailor the strategies aimed at preventing this nosocomial infection. Aspiration of oropharyngeal and gastric contents into the lower airways is the main mechanism in which VAP occurs. Inhalation of pathogens from contaminated aerosols and direct inoculation could also result in VAP though this is less common.

Hand washing is perhaps the single most important general infection control measure taken in the prevention of VAP. Specific preventive strategies are targeted at preventing aspiration. This would include placing the patient in the semi-recumbent position, optimizing endotracheal tube pressure and preventing unplanned extubation as well as reintubation.

As intubation has been shown to increase the incidence of VAP, early liberation from ventilation is warranted. Daily sedation vacation and weaning protocols can decrease time spent on the ventilator. Non invasive ventilation should be used whenever possible in selected patients with respiratory failure.

Great efforts should be made to decrease the incidence of VAP as it represents a major health care burden as well as morbidity and mortality. Preventing VAP should be made an important focus for quality improvement and infection control in the ICU.

PREVENTION OF CATHETER-RELATED INFECTIONS

Marlizan Md Yusoff

Department of Anaesthesiology, Hospital Universiti Kebangsaan Malaysia, Kuala Lumpur, Malaysia

INTRODUCTION

The use of intravascular devices in hospital practice is ubiquitous. Several hundred thousand of intravascular catheters are purchased each year by hospitals in Malaysia. Use of these devices place large number of patients at risk for catheter-related infections (CRIs), which remain a major complication in modern medicine. They increase patient morbidity, affect quality of patient care, and generate substantial hospital costs.

Most serious infections are associated with central venous catheters rather than small peripheral catheters; this is particularly evident in intensive care units (ICUs). An average of 5.3 central line-associated bloodstream infections (CLA-BSI) per 1000 catheter-days in ICUs with attributable mortality between 12% and 25% in prospective studies. The estimated cost per infection is approximately $25,000 and about 500 to 4000 patients die annually of CLA-BSI in U.S. ICUs. A large proportion of these infections are preventable and therefore their prevention should become a priority target of health-care systems.

DEFINITIONS

Catheter-related infections (CRIs) include colonization of the device, skin exit-site infection and device-related bloodstream infection.

Colonised catheter: growth of > 15 colony forming units (CFU) or 103 (quantitative) from a proximal or distal catheter segment in the absence of accompanying clinical symptoms and signs.

Exit-site infection: Microbiologically documented as a positive (semi) quantitative catheter culture in the presence of clinical signs of infection (erythema, tenderness, induration or purulence) at the insertion site of any vascular access and/or clinically documented as a clinical infection (erythema, tenderness, induration or purulence) at the insertion site.

Catheter-related blood stream infection: Isolation of the same organism from the catheter segment as from a peripheral blood culture in a patient with signs of infection and in the absence of another source.

PATHOPHYSIOLOGY

Four distinct pathways may be identified in the infection process of CRIs. The two major pathways are the external and internal bacterial colonization of the catheter surface, both eventually leading to catheter tip colonization, with the potential for subsequent bacteraemia.

Extrinsic mechanisms associated with developing catheter sepsis include infection from the skin and insertion site; contamination from the hub and then internally spread; and contamination from the hub and then internally spread; and contamination of drugs or fluids administered through the catheter. Bacteraemia seeding to catheter is an intrinsic mechanism.

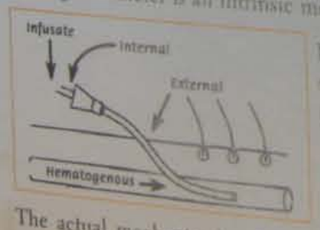

Figure 1 External & internal catheter colonization pathways involving skin insertion site, & hub respectively. Additional pathways include microbial contamination of the infusate & hematogenous seeding

The actual mechanics by which the organisms colonize the catheter is important. The key factors for pathogenesis include bacterial adherence and host defence mechanisms. Host glycoproteins, such as fibrinogen, fibronectin, collagen and laminin, adsorbed on the surface of intravenous devices, form a layer that enhances bacterial adherence to foreign material, in particular Staphylococcus aureus and coagulase-

negative staphylococci (CNS). In addition, some strains produce 'slime' conferring some protection against antimicrobial agents and interfering with neutrophil function. Polyvinyl chloride or polytheneare more prone to this film developing than some other materials such as silicone.

RISK FACTORS FOR CATHETER INFECTION

Host risk factors

Site: Subclavian is a lower risk than internal jugular and femoral

Catheter material: antibacterial catheters may reduce infection, antiseptic catheters reduce colonization.

Number of lumens: multilumen catheters increase the infection risk

Number of administration through the lines

Dressing type frequency of changes

Skin preparation: CHG better than povidone-iodine

Experience of technique of personnel

Occurrence of bacteraemia

Tunnelling: often used for long term access but the data is contentious

MICROBIOLOGY

The organisms involved in catheter sepsis are many and varied. Most of the micro-organisms in CRIs arise from the skin flora. CNS (60% Staphylococcus epidermidis) are the leading bacteria cultured from catheters. Staph aureus and MRSA are prevalent but enterococci are not uncommon.

Gram-negative bacilli are responsible for a higher proportion of CRIs in ICU than in non-ICU patients. They are due to colonization of invasive monitoring pressure systems, complicated remote infections, or a high degree of orotracheal colonization.

Candida spp. have emerged as important pathogens of CRIs and account for a high proportion of the dramatic increase in the rate of candidaemia over the last decades.

TREATMENT STRATEGIES

The most important aspect of treatment is a high index of suspicion that leads to removal of the infected device. Catheter retention may result in a several-fold higher risk for recurrence of bloodstream infection. Although fever and bacteraemia are likely to resolve rapidly after removal of the line, appropriate antibiotics are indicated (5-7 days for uncomplicated CNS; 10-14 days for S. aureus, gram-negative organisms and fungi, and 4-6 weeks if there is evidence of endocarditis, infected thrombus, osteomyelitis, or clinical line sepsis is still present after 3 days). Lines removed should be cultured. If an infection is present it is best to avoid replacing the central venous catheter (CVC) if possible for few days. In situations where it is uncertain if the line is implicated in infection some advocate replacing a new line over a wire.

Futhermore, recent data suggested that a transoesophageal echocardiogram may help to identify vegetation (s) particularly in gram positive bacteraemia which require specific management. In any case, antimicrobial agents should then be adapted according to susceptibility testing. Relapse, continuous fever, or bacteraemia, despite removal of the catheter is consistent with the suspicion of a persistent focus of infection. Following completion of treatment, careful follow-up is required due to the frequent occurrence of late complications.

PREVENTION

More than 50% of patients admitted to ICUs are already colonized at the time of admission with the organism responsible for subsequent infection. Nevertheless, the prevention of CRIs relies on careful control of all the factors associated with the colonization of vascular accesses by microorganisms; evidence-based guidelines and preventive measures have been published by the Hospital Infections Control Practices Advisory Committee.

Four major risk factors are associated with increased CRI rate: cutaneous colonization of the insertion site, moisture under the dressing, prolonged catheter time, and the technique of care and placement of the

CVC. The strategies to prevent CRIs have been focused on implementing processes to mitigate or prevent these risk factors, with the primary focal being the insertion procedure itself. However, most of these measures are supported by clinical studies with a limited strength of evidence. Some are discussed below.

HAND HYGIENE

Appropriate hand hygiene is the cornerstone of any infection prevention program. A strict adherence to hand hygiene measures (hand washing and/or hand disinfection) and to aseptic techniques in caring for patients and devices is the key requirement of these precautions.

There have been persistent reports of low-level compliance with hand hygiene, particularly in ICUs. Experience with alcohol-based hand rubs showed that hand disinfection may reduce hand contamination more than hand washing. This may also save precious time in the ICU where theoretically almost two-thirds of the staff's working time could be required for optimal adherence to infection control guidelines. The CDC guidelines do recommend that when hands are visibly soiled, they should be washed with soap and water (either a non-antimicrobial or antimicrobial).

However, following successful interventions, compliance with hand hygiene decreased again over the next few months. Recently, electronic monitoring and voice-activated prompts to remind the caregiver to perform hand hygiene resulted in improved compliance. Strategies to improve hand hygiene need to be comprehensive and include education, real-time feedback, and the necessary organizational changes to support a sustain program focused on hand hygiene as a major safety initiative.

MAXIMAL STERILE BARRIERS

Maximal sterile barriers (MSBs) have also been shown to reduce CRIs by improving sterile technique during catheter insertion. When using MSBs, the person inserting the central line wears a cap, face mask, sterile body gown, and sterile gloves, and uses a full-size drape to cover the patient from head to toe. Meanwhile, skin preparation should include hair cutting rather than shaving.

One study found a 6-fold higher rate of catheter-related septicaemia when minimal sterile barriers (sterile gloves and small drape) were used instead of MSBs. The recent CDC guidelines on central line management rate MSBs has the highest level evidence for reducing CVC infections and recommends adopting this procedure, as do other experts in the field. Although research studies have not evaluated what the assisting personnel should wear, several groups or guidelines recommend that minimum practice should be universal precautions unless the nurse comes into contact or crosses over the sterile field.

CHLORHEXIDINE FOR SKIN ASEPSIS

Rigorous cleansing and disinfection of the insertion site is regarded as a key point. Povidone iodine has been the most widely used antiseptic for cleansing skin prior to central line insertion in Malaysia. Its combination with alcohol (70%) is effective, but chlorhexidine gluconate (CHG) has been shown to be superior in preventing CVC colonization. In a recent meta-analysis, the use of CHG rather than povidone iodine was found to reduce the risk of CRIs by approximately 50% in hospitalized patients who require short-term catheterization.

Besides reducing CRI rates, there are several other advantages of the use of CHG for skin asepsis. Two percent of CHG in tincture of isopropyl alcohol has rapid bactericidal activity, and is effective within 30 seconds after application versus a 2-minute period for povidone iodine. In addition, CHG provides persistent bactericidal activity on the skin and maintains its activity in the presence of other organic material.

Chlorhexidine also has less allergic response and minimal systemic absorption compared with povidone iodine.

When using CHG tincture preparation, a back-and-forth, up-and-down motion is used to promote penetration of the cleanser within multiple layers of the epidermis. It provides antisepsis when wet and crossing over of previously cleaned areas does not cause contamination. Chlorhexidine solution is clear, making it difficult for the user to know the area that has been cleaned. The manufacturer has added a teal coloration to help improve visibility of the prepared site; however at present only the larger sized CHG applicators have the teal colouring. The smaller size CHG applicators and vials still remain clear of any colour.

SITE OF INSERTION: AVOID FEMORAL LINES

Insertion of CVCs can lead to serious and sometimes life-threatening complications, whether of mechanical, infectious, or thrombotic origin. The insertion chosen can influence the incidence and type of such complications.

A higher rate of infectious and thrombotic complications associated with the used of femoral catheter compared to subclavian catheter had been demonstrated in ramdomized control trial. There are no studies thus far, comparing complication rates between subclavian and internal jugular lines. The internal jugular area, although easier to access, allows the maintenance of an occlusive dressing extremely difficult, and is repeatedly exposed to colonized oral secretions, making the risk for infectious complications potentially high. In the absence of contraindications to catheterisation at either site, selection of the subclavian site is preferred based on available evidence that this site is associated with less risk of infectious and thrombotic complications.

AVOID/REMOVE UNNECESSARY LINES

One of the most effective strategies to prevent central line infection is to eliminate or reduce a patient's exposure to a CVC. The decision to place a CVC must be well thought out by the healthcare team, because of the risk it brings to the process. In addition, once placed, there should be periodic, if not daily assessment, of its continued need, with emphasis on prompt removal.

LINE CARE AND LINE AND TUBING CHANGES

A transparent, semipermeable polyurethane dressing has many advantages over gauze but both have shown no difference in infection rates with use as long as they are changed appropriately. The benefits of a transparent dressing include the ability to evaluate the insertion site while the dressing is in place, wicking of moisture away from the skin, and less frequent dressing changes compared with standard gauze and tape dressings. The CDC guidelines recommend routine changing of transparent dressing every 7 days and gauze every 2 days, and whenever either dressing is soiled or nonadherent. Antibiotic ointment at the catheter site should be avoided, as it promotes fungal infections and antibiotic resistance.

The current CDC recommendation is to replace intravenous administration sets, including secondary sets and add-on devices, no more frequently than a 72-hour interval, unless catheter-related infection is suspected or documented. However, a recent systematic review of studies evaluating 96-hour versus 72-hour intravenous administration set changes found no difference in rates of phlebitis.

CATHETER REPLACEMENT AND/OR GUIDEWIRE EXCHANGE

The duration of catheterization has been linked to the risk of CRIs, particularly after 7 days, but systematic routine replacement of central lines has failed to prove its efficacy in decreasing the risk.

Guidewire exchange may increase the likelihood of infection of the new catheter, but reduces the rate of complications associated with CVC placement in a new site, which may be technically difficult, particularly in severely ill patients. Randomized prospective studies failed to detect any preventive benefit associated with guidewire exchange compared to insertion at a new site. For many experts, guidewire exchange with systematic (semi) quantitative culture of the catheter tip is mandatory in any case of sepsis without clinical evidence of another source of infection. This allows removal of the exchanged catheter and mandates further insertion at a new site only if the culture of the removed material is positive.

ANTIBIOTIC AND ANTISEPTIC-COATED CATHETERS

Catheters coated with chlorhexidine/silver sulfadiazine, impregnated on both the external and internal surfaces with minocycline/rifampin or platinum/silver-impregnated catheters have been shown to reduce the risk for CRIs compared with standard noncoated catheters. In a meta-analysis and a cost-effectiveness analysis, Veenstra et al. suggested that the use of chlorhexidine-sulphadiazine-impregnated catheters decreased the incidence of catheter-related bloodstream infection (CR-BSI) by between 1.2% and 3.4%, corresponding to a cost saving between USD68 and USD391 per catheter used.

As compared to the chlorhexidine/sulphadiazine-coated catheters the minocycline/rifampicin-impregnated catheter was reported to be associated with significantly lower colonization (relative risk 0.35; CI 0.24-0.55) and CR-BSIs (relative risk 0.08; CI 0.01-0.63).

The duration of catheter placement may well have played a role. Impregnated catheters failed to prevent CRIs in neutropenic patient cancer patients with a mean catheterization time of 20 days as compared to 6,7 and 8.3 days for others. Therefore, the potential benefit of these devices may be lost after 7-10 days.

EDUCATIONAL PROGRAMS

Sherertz et al. recently reported that an educational program of physicians in training could decrease the risk of CRIs. A 1-day course on infection control practices and on procedures of vascular access insertion was shown to reduce the infection rate by 73%, from 3.3 to 2.4 per 1000 CVC-days. The educational program included a 1-h introduction to basic infection control principles (handhygiene, isolation and barrier use, handling of patients with resistant organisms). Thereafter, these nursing staffs and physicians rotated through a series of 1-h stations, during which they received 5-15 min of didactic instruction followed by hands-on instruction that was overseen by faculty members. Participants were also instructed to change dressings and intravenous tubing every 3 days and not to adhere to fixed schedules for changing CVCs.

Sherertz et al. also estimated that their program was associated with a cost saving of at least $63 000, perhaps exceeding $800 000. This may represent the salary of one-full time infection control nurse per unit involved in the programme for 1 month to 1 year. Although the precise attributable costs of CRIs remain to be determined, using a conservative approach, it was estimated to correspond to the annual salary of three to six fulltime infection control nurses.

The behavioural changes may have played a key role in the success of these educational programs, which were based on multimodal and multidisciplinary approaches, including communication and education tools, active participation and positive feedback, and systematic involvement of institution leaders.

CONCLUSION

Prevention of infection is the foundation of any severe sepsis management program. CRIs are one of the most prevalent healthcare-associated infections in ICUs. A comprehensive prevention program that included handhygiene, use of MSB precautions, chlorhexidine skin antisepsis, avoidance of femoral lines and staff education resulted in a significant reduction of CRIs. Although the true impact of the introduction of antibiotic/antiseptic-coated catheters in a unit remains to be determined, the data extrapolated from the studies, which explored the impact of educational programs, suggest that educational strategies targeted at vascular-access reduction should be implemented in any unit before considering the use of coated catheters. Nevertheless, the use of such sophisticated devices should probably be considered if the infection rates remain higher than those reported in the NNIS despite the introduction of other preventive measures.

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POSTER PRESENTATIONS

PP 01

DOES FEEDING REGIMEN AFFECT THE INCIDENCE OF VENTILATOR-ASSOCIATED PNEUMONIA? : A STUDY IN HOSPITAL MELAKA

T C Lim, Sivasakthi V

Department of Anaesthesiology and Intensive Care, Hospital Melaka, Melaka, Malaysia

The incidence of ventilator-associated pneumonia (VAP) in Malaysia was estimated to be approximately 23% and in Hospital Melaka, the incidence was 17.3%. Numerous factors including feeding regimen have been associated with the incidence of VAP. Our study therefore looked at the relationship between two modes of feeding to the incidence of VAP in Hospital Melaka. We conducted a prospective randomized study over the duration of three months in 2007 in our Intensive Care Unit (ICU). The study was performed on 40 patients, of whom 20 patients received intermittent enteral feeding and another 20 patients received continuous enteral feeding. They were then followed up daily in order to diagnose VAP. The diagnosis was made based on a set of specific criteria. Other factors studied were patients' tolerance and outcome. There were no significant difference between groups of patients in age, sex, ethnicity, volume of daily gastric aspiration, Acute Physiology and Chronic Health Evaluation II Score (APACHE II), Simplified Acute Physiology Score II (SAPS II) and outcome. Differences in incidence of VAP was not significant (p=0.548), between intermittent and continuous enteral feeding. The incidence of VAP in the intermittent enteral feeding group was 10 % compared to 5% in continuous enteral feeding group. We can therefore conclude by saying that feeding regimen does not affect the incidence of VAP in patients ventilated in ICU.

PP 02

N-ACETYL CYSTEINE IN ACUTE LIVER FAILURE [NON PARACETAMOL INDUCED] – CASE REPORT

Jaya Raj, Norzalina E

Department of Anaesthesiology and Intensive Care, Sarawak General Hospital, Kuching, Sarawak, Malaysia

BACKGROUND

N-acetyl cysteine has been widely used as a mucolytic agent for more than 50 years and is the treatment of choice in paracetamol induced hepatotoxicity, which is the leading cause of liver failure in countries like the UK,USA and Denmark. N-acetyl cysteine has also other uses in clinical practice such as its addition to conventional therapy in the treatment of HIV infection , in the prevention of radio contrast induced nephropathy, cardioprotection in ischemia-reperfusion or myocardial injury and in cardiopulmonary bypass. N-acetyl cysteine is also used as an adjunct in the treatment of non paracetamol induced acute liver failure though controversial and the results are not very convincing and inconsistent , but has yielded some beneficial and promising outcomes. The use of N-acetyl cysteine in non paracetamol induced liver failure, though questionable, have generated lot of interest amongst clinicians and intensivists. N-acetyl cysteine acts directly as an antioxidant and also as a scavenger for oxygen radicals [or more appropriately known as reactive oxygen species], hence reducing the hypoxic insult and prevents further destruction of the hepatocytes following reperfusion injury. It stimulates the the synthesis of glutathione which is an important endogenous intracellular antioxidant. N-acetyl cysteine also helps in modulating pro-inflammmatory cytokine response.

CASE REPORT

27 year old Chinese man with history of swimming and fishing at nearby pond presented with 4 days history of fever and feeling unwell. He was obese (BMI-40) clinically jaundiced, altered conscious level, was in severe sepsis. Blood investigation results revealed a Total White Cell count of 24K/uL, Platelets of 10,000K/uL which later decreased to 6,000K/uL. His blood electrolytes were also deranged, Sodium was 117mmol/L, Urea 35mmol/L and Creatinine 357 umol/L and deranged liver function test with Total bilirubin of 321umol/L and Direct Bilirubin 255umol/L which and markedly raised Creatine Phosphokinase [2076U/L] and Lactate Dehydrogenase of 1058U/L. Coagulation profile was unremarkable. His blood pressure was supported by intravenous infusion of Dopamine and required continuous fluid resuscitation. Clinical Diagnosis of ? Leptospirosis ? Dengue Hemorrhagic fever was made. He was started on intravenous Meropenem 1g 8hourly. He required intubation and ventilatory support as his conscious level deteriorated, and was brought to The Intensive Care for further management. In The ICU he was fluid resuscitated adequately to achieve a CVP of 12-15mmHg and a urine output of 0.5-1ml/kg. Dobutamine and Noradrenaline infusions were started to maintain hemodynamic stability. Cerebral protection therapy was instituted in view of his hepatic encephalopathy and deepening jaundice. Mixed venous sample was obtained from the right atrium upon insertion of central venous line and SVO2 was 59%. Blood sugar levels were kept under tight control. Total peripheral nutrition and glamine were started on day 2 of admission. His Liver function worsened [Total bilirubin was 411ìmol/L]. However there was no deterioration in the coagulation profile. N-acetyl cysteine infusion was started at 21mls/h on day 2 of admission to The Intensive Care, for 48h [at a dose of 100mg/Kg/hour, total dose of 9000mg/day].There was marked improvement in his general condition as his liver enzymes showed a down going trend. His renal function improved as evidenced by urea and creatinine showing a decreasing trend with continuous hydration. His SVO2 improved to 79% on Day 3 of ICU. Total parenteral nutrition and glamine was continued along with his antibiotics. His hemodynamic parameters required only minimal Noradrenaline infusion. He was successfully weaned of the ventilator support and inotropes and extubated well on Day 5 ICU. He was discharged well to the ward.

CONCLUSION

The use of N acetyl cysteine in addition to the other treatment modalities instituted did improve the overall outcome in our patient with acute liver failure by virtue of its anti-oxidant properties and also a scavenger for oxygen radicals hence improving oxygenation to the liver.