Meeting report

8th Annual Toronto Critical Care Medicine Symposium, 30 October–1 November 2003, Toronto, Ontario, Canada

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The eighth annual Toronto Critical Care Symposium was held from 30 October to 1 November 2003, in downtown Toronto, Ontario, Canada. This symposium is Canada's premier critical care conference and attracts participants from across the country, the United States, Europe, Australia and Asia. The attendance this year was in excess of 900 people and included the disciplines of medicine, nursing, respiratory therapy and other allied health care professionals. The themes of this year's meeting included sepsis, organ donation, blood conservation strategies, acute lung injury and ethics. In addition, many of the plenary addresses reviewed the recent severe acute respiratory syndrome (SARS) crisis. The discussion surrounding SARS was particularly poignant as Toronto was the North American city most affected by the outbreak.

Severe acute respiratory syndrome

John Marshall (University of Toronto) kicked off the meeting with an informative review of the epidemiology of SARS: 'How it got from A to B'. The origin of the disease in humans may have developed through contact with animal reservoirs for the virus. Indeed, the first affected individuals were food handlers who routinely came into contact with animals such as the Civet Cat that are sold as a delicacy in Chinese markets.

The Metropole Hotel in the Kowloon province of Hong Kong has become perhaps one of the most infamous hotels in the world as it is regarded as the source of the index cases for SARS in Toronto, Hong Kong, Singapore and Hanoi. Upon return to Toronto one of the exposed hotel guests became ill and was taken care of by her son at home. Although she never sought medical attention (she eventually died), her son became ill and sought medical attention at Scarborough Grace Hospital in Toronto. It was with this visit that the health care system in Toronto became involved in the spread of SARS, with devastating consequences. There were a total of 375 cases in Ontario and there are presently 43 deaths attributed to SARS.

In Toronto the primary spread of the disease was within the hospitals of the Greater Toronto area, essentially making SARS a 'nosocomial' illness. Following the initial outbreak a second wave of infection occurred in Toronto following exposure of patients and hospital staff to an elderly male patient in a convalescent home recovering from a hip fracture. SARS II, therefore, was probably the result of premature relaxation of infection control measures and the late recognition of epidemiological linkages between cases.

Why Toronto? This is a question that is somewhat difficult to answer. Marshall felt that several factors conspired to make Toronto a probable target. Ease of travel, being a large cosmopolitan city, inadequate facilities and lax infection control were all cited as contributing factors. However, just plan bad luck was probably the largest contributor to the choice of Toronto by the SARS coronavirus

The medical system was ill prepared for SARS. Indeed, the successful control of the infection was made through sheer will and tireless determination of individuals. Judith Stein (University of Toronto) focused on several of the lessons to be learned from the way SARS was handled. Through her own investigative efforts she drew some distressing distinctions between the private and public sectors' ability to deal with crisis in any form. She paralleled the private sectors response to the September 11th terror attacks in the United States with the public sector's ability to deal with the SARS crisis.

ARDS = acute respiratory distress syndrome; HGMB1 = high mobility group B1; ICU = intensive care unit; IL = interleukin; MOF = multi-organ failure; NDD = neurological determination of brain death; PaO_2/FiO_2 = arterial oxygen tension/inspired oxygen ratio; SARS = severe acute respiratory syndrome; VAP = ventilator-associated pneumonia.

Most of the major financial institutions in North America have plans in place for disasters to allow them to rapidly and effectively continue to provide service to their customers. Planning for the unexpected is all part of a sound 'business continuity plan' and not to do so would be foolish and would probably result in loss of revenue and possibly bankruptcy during times of crisis. These business leaders not only ensure that systems and infrastructure have redundancy, but ensure that the people who are integral for the basic function of the organization are backed up. Redundancy is not a 'bad word' in the private sector; it is actually good business when applied properly.

Stein has presented these thoughts to deputy ministers in the Canadian government and has received what she interpreted as a less than receptive response. However, Stein provided the audience with a sound argument for the development of an action plan and the notion that the health care sector cannot afford to ignore the 'back up' of systems and human resources – the next time we may not be so fortunate.

Continuing on the SARS theme, Donald Low (University of Toronto) presented a further review on the outbreak in Toronto. Low remains an integral member of the local, provincial and international effort to control the spread of this disease. He was truly on the 'front lines' during the crisis in Toronto. Low described the two phases of the outbreak that occurred in Toronto and relayed the absolute devastation that was felt by the entire health care community and the public when the second outbreak of SARS occurred in Toronto. However, he passed on some lessons that were learned, that may help limit spread if SARS rears its ugly head again. The importance of infection control procedures and lasting vigilance was emphasized.

The period of infectivity and the range of incubation periods have been a cause for concern and debate. The disease in general is not infectious prior to the onset of symptoms, but the incubation period has been variably reported and in some instances may extend beyond 10 days. At present, however, the 10-day incubation, and hence quarantine, period remains. The disease itself peaks in infectivity at the 7-day to 10-day mark. In addition, there does seem to be the phenomenon of 'super spreaders', who may have an increased capacity to infect those that come into contact with them. There is generally increased risk of spread with prolonged patient contact, in a linear fashion. At present there is no evidence of a prolonged carrier state.

Patients can develop severe pneumonia and acute lung injury once ill with SARS. Stephen Lapinsky (University of Toronto) presented a review of Toronto's intensive care unit (ICU) experience with SARS. The mortality associated with a SARS patient who required admission to the ICU was about 40–50%. Multiple therapies were attempted, such as antivirals, steroids and a variety of modes of mechanical ventilation [1,2]. Of particular interest to the audience was the safety precautions required while caring for a SARS patient. Certain procedures have been identified as high risk for health care workers, not the least of which is assisting or performing intubation. High-risk procedures combined with the high-risk SARS patient require extraordinarily strict precautions and the use of full protective equipment.

Transfusion medicine

The session on transfusion practices began with Dean Fergusson's (University of Ottawa) update on the effectiveness of leukoreduction (the removal of white blood cells from donated blood). Leukoreduction essentially reduces the average white blood cell content in a unit of packed cells from 3.0×10^9 to 2.5×10^5. The rationale for leukoreduction is to minimize exposure to transfused leukocytes, which can lead to adverse transfusion reactions or alterations in immunity or inflammation. Since 1999 all blood in Canada has been leukoreduced. A large Canadian study recently examined the effect of this change in practice in adult high-risk surgical patients [3]. Fergusson reviewed the findings of their trial that showed a reduction in mortality, fever and antibiotic use, but failed to demonstrate a change in serious nosocomial infection. Similarly, when they evaluated the effects in neonates (<1250 g) there was evidence of decreased morbidity and ICU stay following the implementation of leukoreduction [4].

A reduction in transfusion rates in the ICU may help to reduce possible harm from blood products. Between 20 and 30% of all blood transfusions occur in the ICU setting, and 85% of patients present in an ICU for longer than 1 week receive a transfusion [5]. Higher transfusion rates have been associated with an increased length of stay and increased mortality versus matched controls [6]. Robert Fowler (University of Toronto) proposed several approaches to decrease the frequency of, and possibly the need for, transfusion in the ICU. Perioperatively, the use of autologous donation, the use of normovolemic hemodilution and the use of cell recovery are possible options. Implementation of a lower hemoglobin threshold for transfusion, limitation in the use of 'routine' daily laboratory work, the use of antifibrinolytics, the use of erythropoietin and a reduction in the amount of phlebotomy performed for the purposes of laboratory testing were all suggested in the ICU. In fact, one ICU study indicated that phlebotomy rates and volumes directly accounted for 30% of all blood transfusions [5]. The use of pediatric phlebotomy tubes in adults and having closed arterial line systems to reduce wastage are viable options that have been shown to significantly reduce blood loss from phlebotomy [7,8].

A large multicenter trial is underway to look at the effectiveness of some of these simple measures to reduce transfusion. The role of blood substitutes remains futuristic.

David Mazar (University of Toronto) reviewed the stormy experience to date with blood substitutes as an alternative to blood products. Although many alternatives are on the horizon, safety concerns and lack of proof of efficacy have limited their clinical application.

Many of the aforementioned issues have become topical in critical care, in no small part due to the work of Paul Hebert (University of Ottawa). In a talk appropriately entitled, 'Still 70?', Hebert revisited the question of what hemoglobin value should be the threshold for transfusion. It seems that clinical practice remains inconsistent. Many people maintain that a value of 70 g/l remains the hemoglobin threshold for transfusion. However, what people think and what they do in daily practice seems to be different. This disparity was emphasized in the anemia and blood transfusion in critically ill patients (ABC) trial. In that study of the transfusion practice of 146 European ICUs, the average hemoglobin at which patients were transfused was 84 ± 1.3 g/l [6]. More randomized controlled trials are needed to answer the question more definitively, particularly in patients with ischemic heart disease. Hebert hinted that a value greater than 70 g/l might be reasonable in patients with ischemic heart disease, with 80 g/l being a possible threshold.

End-of-life care

Deborah Cook (McMaster University) presented 'Predictions and end of life decisions in the ICU', a topic that was sobering to many in the audience and gave food for thought surrounding clinicians' end-of-life decisions in a critical care setting. Given a standard scenario, there was a wide degree of variation in ICU clinicians' decisions about the appropriate levels of care for the given patient [9]. In addition, the confidence of these management choices was highest when the decisions were extreme in nature (i.e. withdrawal of life support) [10]. Factors that predict the implementation of withdrawal of life support include the use of inotropes or vasopressors, physician prediction of an ICU survival of less than 10%, physician prediction of poor residual cognitive function and physician perception of the patient's desire to limit life support [11]. Interestingly, physician prediction of ICU mortality is the greatest independent predictor of ICU death, even greater than the Acute Physiology and Chronic Health Evaluation II score or the multiple organ dysfunction score. In summation, Cook stressed that uncertainty exists, as demonstrated in multiple studies examining confidence, predictions, perceptions and discomfort in end-of-life decisions.

Moving on from physicians' perceptions in end-of-life issues, Daren Heyland's (Queen's University) discussion of the experience of dying from the perspective of the patient and of the family brought to light some areas of patient care that require greater attention [12]. The majority of deaths in Canada occur in a hospital setting, from medial (noncancer) causes; however, palliative care seems oriented to cancer patients [13]. With regards to dying in an ICU, the majority of family members rated their satisfaction with overall care as excellent or very good [14]. For Canadian ICU patients and their family members the most important aspect of achieving quality end-of-life care was having trust in the doctors looking after you. Problem areas that were identified included a perceived gap between patient and family needs, and a shortfall in services to assist with care upon discharge from hospital. To improve patient satisfaction during end of life these gaps need to be targeted by concentrating on continuity of care, on patient–doctor relationships and on relief of symptoms.

The experience in adults was contrasted to that in pediatrics. In pediatric hospitals 80% of inhospital deaths occur in the pediatric or neonatal ICU. Yet there is minimal evidence-based literature about end-of-life care or palliative care in pediatric ICUs. Jacques Lacroix (University of Montreal) emphasized during his presentation the importance of a multidisciplinary approach to end-of-life care in children. Caregivers rarely decide on their own about a child's end-of-life wishes, as parents' views are involved in the decisions [15,16]. The greatest source of conflict is secondary to poor communication, and this conflict can have serious detrimental effects on the child's care [17]. To prevent conflict, the information given to parents needs to be consistent. They should be reassured that nothing will be done to hasten the death of the child and that drugs will be used only for comfort and palliation [18].

The European experience surrounding issues of organ donation was reviewed by Jean-Louis Vincent (University of Brussels). He based his philosophical discussion on the four main pillars of medical ethics: autonomy, beneficence, nonmaleficence and distributive justice. During his discussion, Vincent emphasized that any decisions about organ donation needed to represent a balance of these four concepts. Vincent contrasted the North American style of obtained consent for organ donation with the approach of presumed consent practiced in some European centers. In the latter situation, citizens have to register their objection to organ donation or it is presumed that they are willing donors. It was emphasized, however, that if a family of a brain-dead patient indicated that they felt their loved one was against donating, then those wishes should and would be respected. Finally, the issue of nonheart-beating donors was touched upon, and further debate is almost certain as this is becoming a hot topic in transplant and critical care medicine.

To bring the session on end-of-life care to a conclusion, Sam Shemie (McGill University) presented a review of the determination of brain death in Canada. The concept of brain death is medically and legally accepted as death in Canada and a majority of countries worldwide [19]. Despite this national and international acceptance of brain death, however, there is no global consensus in diagnostic criteria [20]. In the United States the concept is one of whole brain

death, in the United Kingdom it is one of brainstem death, and finally in Canada there is no clear distinction between whole brain death versus brainstem death.

A forum sponsored by the Canadian Council for Donation and Transplantation (Severe Brain Injury to Neurological Determination of Brain Death) was held in Canada in April 2003 to reach a national consensus on optimal management of patients with severe brain injury, and in particular for those who evolve to brain death. These recommendations are in the process of national distribution. Shemie drew our attention to the distinction between brain death (a concept that lacks clarity) and the neurological determination of brain death (NDD), which is the process and procedure to determine death based on neurological criteria. The current evidence base for existing NDD guidelines is inadequate, and clear standards for NDD and the qualifications of physicians performing NDD need to be clarified.

A variety of recommendations came out of this Canadian forum. Accordingly, their definition of NDD was irreversible loss of the capacity of consciousness combined with the irreversible loss of brainstem reflexes, including the capacity to breathe. The physicians performing this testing must have an independent license and have skill and knowledge in the care of brain-injured patients. Recommendations were put forth for the minimal clinical criteria for NDD, including the appropriate brain stem reflexes to be tested. Confounding factors need to be absent in the diagnosis of brain death, such as shock, hypothermia (< 34°C), metabolic disorders causing reversible coma, nerve or muscle dysfunction accounting for unresponsiveness, and medications or toxins accounting for the clinical state. With regards to the apnea test, the recommendation was to achieve $PaCO_2 \geq 60$ mmHg and ≥ 20 mmHg above baseline, and pH ≤ 7.28. A physician also needs to observe the patient throughout the entire test.

The panel concluded that there is no medical basis for the existing laws stating that two determinations of death are required for organ donation. This led to the recommendation that although two determinations of brain death are required by law, there should be no fixed examination interval. Finally, NDD is primarily a clinical diagnosis, which only requires ancillary testing if there is an inability to complete any of the minimum clinical examination criteria or there are confounding factors that cannot be resolved. In this situation the ancillary test must demonstrate the absence of intracranial blood flow to diagnose brain death. Electroencephalogram is no longer recommended.

The report also focused on a variety of other issues including timing of declaration, reporting and legal issues surrounding brain death. It should also be noted that the forum had specific recommendations for children younger than 1 year of age and newborns that differ from the aforementioned recommendations.

Nosocomial infections

Nosocomial infections affect about 30% of patients in ICUs and are on the increase [21]. The Sepsis Occurrence in the Acutely Ill Patient study undertaken by the European Sepsis Network taught us that Gram-negative and Gram-positive infections seem to be equally prevalent. This study also showed increased mortality and increased ICU length of stay in patients who acquired a nosocomial infection. The lungs seem to be the most common location of infection. During his presentation, Vincent reviewed possible modalities to reduce the incidence of ventilator-associated pneumonia (VAP), including noninvasive ventilation, selective digestive decontamination and continuous subglottic aspiration [21]. He concluded by promoting an integrated care team for infected ICU patients. This team should include a microbiologist, an infectious disease consultant, infection control and a pharmacist.

Gram-positive organisms are the most common source of bacteremia in the ICU and their prevalence is increasing, as is the problem of antibiotic resistance. In particular, methicillin-resistant *Staphylococcus aureus* is a significant problem in industrialized nations [22]. Mervyn Singer (London, UK) acknowledged that the dilemma is becoming particularly bad in the United Kingdom, with specific regions affected by methicillin-resistant *Staphylococcus aureus* [23]. The question of how to adequately treat Gram-positive bacteremia is not so clear. The duration of therapy has traditionally been extended in comparison with other bacterial infections. However, whether this is appropriate is uncertain. It seems that if a microbiologist becomes involved in the care of these patients, then the treatment courses are usually shorter in duration. Current treatment options include glycopeptides (vancomycin), synercid and linezolid.

The long-standing dogma of isolating these patients must also be looked at more critically. Singer emphasized that isolated patients may actually receive suboptimal care with less frequent visits be the medical team [24]. In addition, the isolation does not seem to reduce cross-infection rates in high prevalence units. In summary, Singer impressed upon the audience that methicillin-resistant *Staphylococcus aureus* is probably here to stay and that the best combination of antibiotics and infection control practices remains to be determined.

Improving diagnostic precision and the appropriate utilization of antibiotics may be one strategy to reduce the emergence of multiresistant pathogens. Using VAP as a model for discussion, Heyland reviewed the 'Impact of diagnostic strategies on patient outcome'. VAP has an attributable mortality of 5.8% and increases the length of stay by 4.3 days [25]. The importance of correct antibiotic selection from the start of treatment was stressed, since inappropriate antibiotic coverage from the outset has been shown to increase morbidity and mortality [26–28]. Therefore, if VAP is

strongly suspected, the empiric use of broader spectrum antibiotics is probably the best approach. However, the use of broad-spectrum antibiotics can lead to increased resistance, cost and toxicity. To help combat these drawbacks of broad-spectrum antibiotics, Heyland proposed a 'de-escalation' of antibiotic coverage once culture results are available. There is presently no gold standard for the diagnosis of VAP.

The use of bronchoscopy to help diagnose VAP has been shown to alter the amount and types of antibiotics used, but the effect on mortality is questionable [29–32]. Heyland also eluded to the use of serum-based markers, such as procalcotonin, IL-6 and IL-8, to help diagnose VAP in the future [33]. A novel method of ruling out the diagnosis is through the interpretation of the waveform that is generated during the performance of the partial thromboplastin time assay. The presence of an abnormal waveform is associated with increased mortality and can be a sensitive and specific indicator of disseminated intravascular coagulation. The waveform changes may also precede the onset of clinical disease by 48 hours [34]. There seems to be a high negative predicative value if a specific type of waveform is absent, possibly helping to rule out infection.

Although not classically felt to be a nosocomial infection, the threat of spread through donated blood makes West Nile a potential nosocomial pathogen. The potential impact on critically ill patients was highlighted in a report that reviewed the spread of West Nile to several solid organ transplant patients. They acquired the disease from an infected donor who initially was exposed from blood he received during his initial resuscitation for multiple trauma [35]. In his presentation, James Brunton (University of Toronto) reviewed the epidemiology and effects of the West Nile virus. The cases of West Nile are both geographically and temporally localized. The disease is most prevalent in the northeastern United States and the highest incidence occurs in August and September. However, there has been a gradual westward progression of the disease in the past few years.

The clinical course of West Nile follows a very generalized pattern, which unfortunately can make it hard to diagnose. The prodrome is generally 2–7 days followed by delirium, which is often attributed to another etiology. The patient may then deteriorate and have a decreased level of consciousness, a lower motor neuron pattern of weakness and respiratory failure. The recovery phase can be varied in duration and at times protracted. Diagnosis can be aided by West Nile viral cultures and serology. In fact, blood is now tested prior to donation, which should hopefully reduce the risk of transmission. The treatment of West Nile is really just in its infancy. Intravenous immune globulin has experienced conflicting results in case reports. Interferon alpha 2b is an investigational treatment that may offer hope. Finally, human trials may be on the horizon for the development of a vaccine.

Novel strategies for the treatment of sepsis

Several targets for modulation of the inflammatory response have been identified and are a source of ongoing investigations. One such treatment target is the high mobility group B1 (HGMB1), a nonhistone chromosomal protein. There is evidence that HGMB1 may be involved as a late mediator of inflammation with cytokine-like activities [36]. It is capable of inducing lung injury, liver injury and gut injury in mice [37,38]. Mitchell Fink (University of Pittsburgh) presented his research on novel strategies for inhibiting the effects of HGMB1. The administration of ethyl pyruvate has been shown to successfully modulate the inflammatory response in animal models of sepsis. Work is underway to evaluate its effects in humans. Other potential modulators of HGMB1 include an anti-HGMB1 monoclonal antibody and a recombinant segment (box A) of HGMB1 [39]. Interestingly, it seems that strategies targeting HGMB1 may potentially offer benefit even in late sepsis.

Gregory Downey (University of Toronto) succinctly reviewed the importance of the modulation of signaling pathways in sepsis and in lung injury. The mechanisms of lung injury are complex and can be divided into early (proximal) and late events. One of the first events is bacterial endotoxin lipopolysaccharide binding to toll-like receptors. If this pathway can be interrupted early then the cascade that follows may be stopped or reduced. One possible way to achieve this is by the use of agents that bind lipopolysaccharide, such as antibodies. Techniques to interrupt in the later stages of the sepsis cascade include antioxidants, such as alpha-tocopherol (vitamin E). Interruption of these late stages has been shown in murine models to confer protection even if given 1 hour after the initiating stimulus.

Singer concluded this section by providing an interesting teleological perspective on multi-organ failure (MOF). He proposed that MOF represents cellular shutdown and is a late-stage adaptive response to a prolonged insult. He challenged the audience that meddling with the immune response to prevent MOF may be counter-productive. In defense of this proposal, Singer argued that tissue oxygen levels increase and oxygen consumption decreases during sepsis [40–42]. This is associated with mitochondrial inhibition, yet ATP levels are often preserved [43]. Indeed, during MOF there is little histological evidence of cell death, and if the patient survives so do the organs [44,45]. In a sense, the cells of major organ systems go into a state of hibernation and re-emerge when the illness is resolving. The analogy of hibernation not only applies to the animal kingdom, but can also be appreciated in the phenomenon of 'hibernating' myocardium. Cellular shutdown leading to organ failure may thus just be an adaptive response or a 'last-ditch' effort at organ preservation.

Mechanical ventilation

The session surrounding mechanical ventilation began with Tom Stewart's (University of Toronto) discussion of lung

recruitment. Lung recruitment may have a role in cases of difficult oxygenation or ventilation and as an early protective strategy in patients with acute lung injury. Evidence does suggest that recruitment maneuvers lead to increased oxygenation, at least in the short term [46]. The success of lung recruitment maneuvers to increase the PaO_2/FiO_2 ratio depends on the stage of acute respiratory distress syndrome (ARDS), with increased success early in the course. Other factors include the severity of illness, the type of ARDS, patient positioning and positive end-expiratory pressure settings postmaneuver [47–50]. Recruitment maneuvers have been shown to have low complication rates, with hemodynamic consequences being the most frequent [48]. There are a variety of ways to perform recruitment maneuvers, but most of the maneuvers require the patient to be heavily sedated. Stewart also presented some promising interim analysis of the Lung Open Ventilation Study demonstrating the safety of recruitment maneuvers.

On the flip side of the previous topic, Brian Kavanagh (University of Toronto) presented the implications of lung derecruitment. He stressed that there are a variety of etiologies of lung derecruitment and that atelectasis is not straightforward [51]. The impact of atelectasis on the pulmonary vasculature may be greater than appreciated. In addition, atelectasis may lead to lung injury directly, through changes in microvascular permeability [52]. The effectiveness of modalities to reverse this injury depends on timing, the nature of the lung injury and the approach used. The utility of early prevention of derecruitment requires further study.

'The chest wall vs. the abdomen' was the subject presented by Antonio Pesenti (Ospedale S. Gerardo, Monza, Italy). He eloquently described the physiological interaction between the abdomen and the chest wall during the respiratory cycle. In the supine patient the pressure of the abdominal contents is directed on the diaphragm in a cephelad direction. This subsequently causes atelectasis in the dependent lung zones near the diaphragm. Increases in both chest wall compliance and abdominal pressure will exaggerate this effect. In fact, by decreasing chest wall compliance through the experimental use of weights directly applied to the chest wall, one can demonstrate a reduction in V/Q mismatch and an improvement in oxygenation. Pesenti left the audience with four simple words to remind us of the importance of the abdomen in respiratory mechanics: 'breathe with your belly'.

Arthur Slutsky (University of Toronto) followed with the topic 'Mechanical ventilation and multiple organ dysfunction'. The lung seems to be in a unique situation to contribute to multiple organ dysfunction. It receives all of the systemic circulation and contains 25–50% of the marginated neutrophil pool. A variety of studies have suggested the connection between lung injury and multiple organ dysfunction. In particular, lung overinflation without positive end-expiratory pressure has been demonstrated to promote bacteremia, and there is pulmonary to systemic translocation of endotoxin [53,54]. In addition, Slutsky discussed ongoing animal research that is looking into the relationship of injurious ventilatory strategies and apoptosis of organs distal to the lung (e.g. kidney, gut). A reduction in cell death was noted in the lung protective strategies in this animal model. Overall, both biochemical and biomechanical injury to the lungs causes the release of a variety of inflammatory mediators, such as cytokines, complement and neutrophils [55]. These mediators in turn may lead to MOF and contribute to the morbidity and mortality beyond the initial lung injury.

As many of the aforementioned studies have noted, mechanical ventilation can lead to injury to both the lung and to other organs. Evidence is mounting that lung protective strategies may reduce the harm to patients secondary to injurious methods of ventilation. However, the knowledge of lung protective strategies has not translated into their widespread use in practice. Gordon Rubenfeld (Washington University) cited many reasons for this discord. Failure to recognize mild forms of acute lung injury/ARDS and lack of knowledge surrounding lung protection are continuing problems. In addition, physician reluctance was seen as a barrier to implementation. The solution to this failure to apply the literature to everyday practice requires several approaches. Better training and improved documentation of patient's ventilation parameters at the bedside are starting points. However, Rubenfeld indicated that standard protocols for ventilation might be the most efficient and effective solution.

In a case of what is old is new again, the issues surrounding surfactant in lung-injured patients were revisited by Neil MacIntyre (Duke University). The rationale for using surfactant includes better mechanical function, anti-inflammatory properties and strong evidence in infant respiratory distress syndrome and in animal models. Improved mechanical function relates to less regional overdistention when adequate surfactant is available. Previous attempts to study the use of surfactant replacement in ARDS have not shown an improvement in outcome. However, these studies were complicated by inefficient delivery systems and surfactant preparations that may have been inadequate [56]. More recent studies have pointed to improved oxygenation and possibly a trend towards decreased mortality [57]. Issues that may be important in the effectiveness of surfactant as a therapy include the nature of the lung injury (direct versus indirect), the timing, the protein/phospholipid content and the dosing route.

The emerging technology of neurally controlled ventilators was presented by Christer Sinderby (University of Toronto). Asynchrony between the patient and the ventilator is common in the critically ill. This often leads to ineffective ventilation, to ventilator-related complications (e.g. pneumothorax) and to

increases in sedation. Neurally controlled ventilators would have the timing and possibly the magnitude of ventilatory assistance controlled via a neural input from the patient. Diaphragmatic electrical activity can be monitored and can act as a signal to in effect "inform" the ventilator about a patient's respiratory cycle. With neurally adjusted ventilators the amount of mechanical support varies from moment to moment according to a mathematical function that converts diaphragmatic electrical activity to ventilator flow and pressure. This mode of ventilation has significant relevance as it stands to improve patient–ventilator synchrony, to reduce requirements for sedation and to facilitate weaning. Future research includes the use of neural-controlled ventilation in neonates and as a noninvasive modality [58].

After patients leave the critical care unit many critical care staff lose contact with them and have little perspective on their quality of life in the future. Margaret Herridge (University of Toronto) presented a review of research looking at long-term outcomes in patients with ARDS [59]. Patients were assessed 3, 6 and 12 months after discharge from the critical care unit. With regards to the pulmonary function tests, the lung volumes approached normal values by the 12-month mark, but the diffusion capacity remained substantially lower than predicted. The distance walked over 6 min (a measure of patient functioning) was less than predicted at 12 months. This was attributed to global muscle weakness and wasting, to foot drop, to immobility of large joints and to dyspnea. Surprisingly, 49% of patients had returned to work 1 year after discharge, with the majority in their original position. The absence of systemic corticosteroids, the lack of acquired illness during the ICU stay and the rapid resolution of lung injury were associated with higher functional levels at 1 year.

Herridge's data call upon us to evaluate strategies to improve patient outcomes further by altering how we care for patients in the ICU (steroids, neuromuscular blockade, nutrition) as well as developing programs to promote rehabilitation both during and after the patient's ICU stay. Herridge's results challenge us to expand the role of the ICU physician as an advocate for patient care during their convalescence.

Pesenti wrapped up the session on mechanical ventilation with his talk 'How I manage ARDS'. He began by reminding the audience that mechanical ventilation and ARDS are highly linked. With this in mind, should we be attempting to ventilate non-normal critically ill patients to normal lung blood gases? There is a price to pay for attempting this. With controlled mechanical ventilation our goals should be low tidal volumes and low plateau pressures. Permissive hypercapnia allows greater latitude in providing these less injurious ventilations to the patient with ARDS [60,61].

Pesenti presented his rationale for the use of volume control over pressure control ventilation. The use of higher levels of positive end-expiratory pressure and recruitment maneuvers (including prone positioning) was encouraged. In his discussion of prone positioning, Pesenti reviewed the available data and suggested that a good indicator of survival in the prone patient is the responsiveness of the $PaCO_2$. The benefits of early weaning and conversion to pressure support ventilation were also stressed. Studies have shown that selected groups of ventilated patients can tolerate pressure support very well early in their ICU course [62]. These patients get the benefits of pressure support earlier, which include less sedation, decreased intrathoracic pressure, improved tidal volume distribution and less respiratory muscle atrophy.

Conclusion

This summary is just a sample of the presentations at the 2003 Toronto Critical Care Medicine Symposium. Many other presentations touched on subjects including medical errors, sedation, team building, management of cardiac failure and specific pediatric critical care issues. Particularly entertaining sessions were several pro/con debates that occurred at the end of the symposium. The topics debated were lung protective strategies, nontherapeutic ventilation and genomics.

Overall the 3-day conference was an enormous success. Planning is already underway for next year, 21–23 October 2004. Save the dates.

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Conference Calendar

2004

28–31 January 2004
6th Annual Anesthesia and Critical Care Challenges for Clinicians
St John, Virgin Islands
http://www.uchicago.edu/bsd/cme/

1–6 February 2004
12th Winter Symposium on Intensive Care Medicine
St Moritz, Switzerland
http://www.intensive.org

21–25 February 2004
33rd Critical Care Congress
Orlando, USA
http://www.sccm.org/education/index.asp

25–29 February 2004
The Canadian Critical Care Conference
British Columbia, Canada
http://canadiancriticalcare.ca/ccc/

For further information about these and other conferences see our online Conference Calender at http://ccforum.com/conferences/