

Critical Care News

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Arkansas Children's Hospital, Little Rock, AK



Improving ventilatory care processes at an award-winning children's hospital

In the course of our research and production of each issue of Critical Care News, we have the unique opportunity to visit many different ICU departments and to meet ICU staff from all corners of the world. Every ICU and each staff member is truly unique. But intensive care facilities in every country face a number of common challenges: more sophisticated therapies, more technologically advanced equipment, the logistics of medical staffing, and the threat of new disease conditions.

Improving ventilatory care processes and procedures: focus and challenges

In this issue of Critical Care News, we highlight a number of ICU centers around the world where changes in ventilatory care procedures have had an impact on staff treatment routines and patient outcomes.

Tailoring pediatric and neonatal ventilation therapy

The feature article focuses on the challenges of refining ventilatory care processes in one of

“Kids are definitely very different to adults in terms of anatomy and physiology. They are more than just a smaller patient, they are dynamically different.”

Professor Mark Heulitt MD,
Arkansas Children's Hospital

the largest children's hospitals in the United States. At Arkansas Children's Hospital, extensive ongoing research is the basis for tailoring new ventilatory treatments and procedures to pediatric and neonatal patients, with positive outcomes, in a private non-profit medical center environment. Dr Mark Heulitt outlines some of the physiological obstacles in treating the smallest of patients, and his innovative research methods in validating new therapeutic approaches.

Lung-protective strategies

The continuing focus on lung-protective strategies is reviewed in an interview about atelectasis in anesthesia and intensive care with Professor Göran Hedenstierna of Uppsala, Sweden, who is internationally acknowledged for his expertise and research within the field. The interview is accompanied by articles about post-graduate lung-protective sessions and workshops held at the Uppsala Academic Hospital in Sweden and the University of Amsterdam Medical Center in the Netherlands.

ICU epidemic management

The global concern of managing epidemics in the intensive care environment is the basis of the article about the experiences with the SARS outbreak at Prince of Wales Hospital, Hong

Kong, where new precautions and routines were implemented, and a comprehensive strategic planning process is underway for future ICU crisis management scenarios.

New post-op cardiac weaning procedures

The fast pace and patient volumes in the CVICU environment involve special challenges that require special solutions. The CVICU at Lund University Hospital in Sweden shares its new practices and procedures for facilitating the weaning processes that have been developed, refined and documented in recent years.

Advanced in-flight ventilatory therapy

Increasing population densities in metropolitan areas is a global phenomenon that has accelerated over the past decade. The trend

of transferring ICU patients to specialized hospitals is growing, and requires the same ventilation treatment strategies as in the ICU. Assistant Professor Gerhard Kuhnle shares his experience of the challenges of treating ventilator patients in transport, and solutions for providing sophisticated bedside therapy in-flight at the University Hospital of Grosshadern, Munich.

Global dedication

Despite the challenges of dynamically changing ICU environments around the world, we can identify one universal quality that is shared at every institution: the dedication of the ICU staff to providing the best treatment and care for their patients. ■



Ventilation procedures for intensive care air transports



Post-graduate lung recruitment workshop



Two years post SARS: the experience and need for preparedness

Arkansas Children's Hospital is a non-profit organization and one of the largest institutions for children in the United States. It has received national acclaim as one of the best children's hospitals in the country, with recognition from Child magazine and U.S. News and World Report.

The hospital's Pediatric Intensive Care Unit, Neonatal Intensive Care Unit, Cardiovascular Intensive Care Unit and Burn Unit admit and treat over 3,000 patients a year. Many of these patients are transported to the hospital by means of two helicopters from a surrounding five-state region. The extremely high patient acuity at this institution, and comprehensive research programs conducted at the non-profit Arkansas Children's Hospital Research Institute, require continuous refinement of ventilatory care procedures by staff for the broad range of conditions that the children present with.

Critical Care News talked to Professor Mark Heulitt MD about his experiences of the challenges in research and treatment of pediatric and neonatal patients.

Lung protective strategies in an award-winning children's hospital lead to reduction of ARDS mortality from 50% to 12%



Professor Mark Heulitt, MD has conducted research for many years. He is Associate Medical Director of Respiratory Care Services at Arkansas Children's Hospital and Director of the Applied Respiratory Physiology Lab at Arkansas Children's Research Institute

Can you briefly describe the unique environment here, in the hospital and in the PICU?

I have been working here for 15 years. I did two fellowships, one in neonatology at Duke University in North Carolina and one in pediatric critical care medicine at Southwestern University in Texas. I came here to Arkansas Children's in 1990, and started as an Assistant Professor. But now I am a full Professor in Pediatrics, Physiology and Biophysics.

This hospital is an amazing place to work. We are constantly expanding, and it is truly an institution focused on children's needs. We invest a lot of effort, energy and money in ensuring that this is a good environment for our children. Everyone likes to work here, as it is a very positive environment where the attitude is always "how can we help this?" and never "I can't do that."

We had a problem recently. I called one of the senior management and said something wasn't working and it was fixed within 45 minutes. The focus here on service is phenomenal.

The unique aspect of our PICU is that we are



Dr Heulitt and Child-life Specialist Esther Pipkin support a 15-year-old quadriplegic accident victim with a computer interface to communicate with

one of the largest in the US, especially when you take into account that we don't have post-op heart patients here; our post-op heart children go to a separate ICU within the facility, the CVICU. There are PICUs that are larger than ours, but they are a mix of post-op hearts, general medical and general surgical cases. Our unit is medical, with neurosurgical, trauma and other post-op surgical cases. A 26-bed PICU like we have here with that kind of mix is very unique. Our PICU nurses are great; they could work anywhere in the hospital since they do just about everything here. For example, you could have one neuro ICU patient with increased cranial pressure, and in the next bed you could have a septic shock patient. Our PICU nurses are well trained and can take care of a diverse range of problems.

It's amazing how the environment of the PICU changes from day to day and hour to hour. Right now in the summer we see a lot of neuro ICU patients. Being the only children's hospital for the state, we see an enormous number of patients being brought in for trauma in the summertime; car accidents, other vehicular accidents, near drownings, and so on. We had

a young man here from Dallas who was run over by a boat, with a severed leg. It's a really diverse group of patients.

We are a non-profit institution. The hospital has a contract with the university for our services, as the university is our employer.

We are active in research and recognized for this, as well as for our advanced facilities and the way we treat our patients.

Which area do you receive patients from?

What is interesting about Arkansas Children's is that we are in the center of the state of Arkansas. We get our regional patients from Missouri to the north, and from Texas to the west. From the south we get a lot of patients from Louisiana, and from the East they come from Tennessee and Mississippi.

We have patients from this large area since we have facilities that the other states just don't have. There are other children's hospitals in this region but they don't have the broad specialty areas we offer.

What are some of the challenges of pediatric and neonatal ventilation therapies, as opposed to treating adult patients?

Kids are definitely very different to adults in terms of anatomy and physiology. They are more than just a smaller patient. They are dynamically different. We like to term them as "God's work in progress." The way they breathe is different. So a ventilator must meet certain needs in the pediatric patient that are different to the adult patient. In the newborn period, a child may breathe 60 breaths per minute or more. The ventilator has to cycle every second, in contrast to every 6 seconds for an adult patient. This puts a demand on the technology of the ventilator to rapidly respond to the patient's needs. In one cycle per second there are an enormous number of things that must occur – prior to when the ventilator is triggered, to after the ventilator is triggered, to the phase of when air departs the lung during expiration. During that entire cycle there are many factors that can disrupt the synchrony of the patient and ventilator. The key to pediatric and neonatal patients is to have the

technology capable to interface rapidly, and that is the nice thing about the SERVO-i ventilators and our use of them. There are two key components that really make a difference. One is a sophisticated flow delivery system that allows extremely rapid response between the patient and the ventilator. It is truly unique. It is active even during the expiratory phase, and it samples 2,000 times per second, which is amazing. The other key component is an interface that allows the information to be processed through the ventilator and can also provide information output to the physician for interpretation. At my research laboratory, we have worked with a number of models to evaluate the SERVO-i and continuing developments to it. We can ventilate animals as small as 25 grams with the SERVO-i with no difficulties. With our animal models, we can replicate some of what we are doing up here in the ICU, as well as evaluating and trying out new ideas as we make technological changes.

Children are unique not only in terms of development of their lungs, but also of their brains. The ability to interface on a number of different levels is very important. I speak at a lot of meetings and lectures, and I am usually the lone pediatric representative. But what works in adults does not work in children; it is more than simply an issue of size. The ventilator we use has to have the capability to take into account these dynamic differences we see in the children, to meet the patients' needs. When we talk about ventilation, we are talking about three things: the equipment, the patients and decision-making. With this new technology, by providing better information, the clinician is able to make better decisions. In previous years, we didn't have this information. You put the patient on the ventilator and the patient and ventilator fought back and forth, until the patient became exhausted and did what the ventilator wanted to do. Now we have the capability to form an interface between the patient and the ventilator, to allow the clinician to make decisions. Our objective here in our patient care and in our research is to decide what are the best decisions to give the best care and get the best outcomes. The best outcome for us is the shortest time on the ventilator, getting the patients off quickly with the minimum amount of complications.

What clinical difference does sampling 2,000 times a second make?

It really makes a difference in our research. When you look at sampling 100 times per second, or 200 times per second, there is an enormous amount of information that is lost. In the respiratory cycle there are certain



Cardiologist Eudice Fontenot and pediatric cardiac surgeon Imamura Michiaki attend a 4-year-old fontanelle patient. She was on bypass for 7 hours, and will be treated with PRVC for 6-8 hours prior to weaning

phases that are very dynamic. An enormous amount of information is necessary for the patient to be synchronous with the ventilator, for the patient to be comfortable, and for gas to be delivered properly. These dynamics occur during those phases, and if you're not adjusting for that information, the patient can head in the wrong direction, especially in pediatric and neonatal patients with these short cycles. In adult patients it may not be as important, but it is always important in cases where the patient and the ventilator are asynchronous. The ventilator technology pays its dividends in that subset of patients who are on the ventilator for a long period of time and are very sick. This is where these differences in speed are really significant. We all believe that these patients should be breathing spontaneously, and not heavily sedated with blocking agents as there are a lot of problems associated with these. That will delay the healing process.

What different categories do you have throughout the year?

We get the whole gamut of pediatric problems. From a seasonal standpoint, we see a lot of trauma in the summertime. In the fall months,

we see more respiratory problems as temperatures drop and the asthmatics start to suffer. In wintertime, we see patients with RSV – respiratory syncytial virus. This is partly due to the fact of the changing environment. We see some patients here with unique metabolic disease; we have a large sickle-cell population in this state, we have CF patients and follow them to adult stages. Other types of infections that we are seeing right now include pertussis – there is a very large outbreak right now. Children and babies are coming in prior to their immunizations. Pertussis in adults and immunized children presents with the same symptoms as a cold. But babies and children who are not immunized can be severely affected. It is a unique disease that requires a ventilator to meet our needs, since pertussis patients have conditions that can change dramatically in a short period of time. The patient may be on the ventilator and be doing well, and suddenly have a paroxysmal coughing episode. A lot of dynamics can happen, and this is when a ventilator with response of 2,000 samples per second can make a big difference. What can you do? Give the patient high doses of drugs and paralysis agents, and wait for them to recover, but that is not good since it will

extend the time on the ventilator. We prefer the patients and ventilator to work together in a way that the patient's needs are being met.

We see everything. We also have a very large burn unit here, with a director who is an ex-burn victim himself. We work very closely together. Burn patients who are very complicated from a respiratory point of view are transferred up here to the PICU. If the burns are more complicated, we cover the respiratory needs in the burn unit.

What range in ages do you see in your PICU patients?

We take care of babies who have been sent home, but come back to the hospital with difficulties. So our age groups range from neonates up to patients of 21 years of age. But in some cases we do admit older patients who may have a unique need that requires treatment in our center, which no other center can administer. So we do have patients in their 40s and 50s who also come for support on our ECMO system.

Do you have any standard ventilation protocols for different patient categories?

We have pretty much standardized our approach to PRVC-Volume Support. When a patient comes in we use PRVC with a protocol, and when they begin to trigger, we switch them over to Volume Support. We also use Automode to allow the patient and ventilator to interact. We keep them on Automode and Volume Support until the FiO_2 is less than 30%, the PEEP is 6 or less and peak inspiratory pressure is 20 cm H_2O or less. At that point, we extubate. We have outstanding results utilizing this approach. In ARDS patients, we

have reduced mortality rates from 50% to 12% since 1991.

What are your clinical experiences with regard to upper airway mechanisms in small children?

We do a lot of non-invasive ventilation support, without endotracheal tubes. To be able to do non-invasive ventilation with SERVO-i will serve a lot of patients here who need positive pressure support, but don't need an endotracheal tube. In my research, we look at the airway as it responds to bronchodilators, and what happens to children. When children grow, their lungs grow faster than their airways do. So the airways take some time to catch up, and many people don't realize this important fact. We have been able to demonstrate this in a rat model with constricted airways, to simulate the development that happens in children. So we can look at what happens when children become sick, and what happens when we put them on positive pressure ventilation. This teaches us, and supports the educational effort, that a one-month old is much different from a one-year old, who will differ greatly from a five-year-old, who is in sharp contrast to a ten-year-old. We need to meet very specific needs in each different age group, and we need to identify their requirements: pharmacologically, mechanically and from technique and decision-making standpoints.

In my research we intubate rats from 2 weeks of age on, or about 25 grams in weight. With the technique we have developed here, we can visualize the airway with the tube in, and extubate and recover the rats with a 96-98%

survival rate. This allows us to study them over time, to monitor developmental changes that occur rapidly, as rats reach maturity within 2-3 months. The human developmental rate is obviously much slower, and would require years to do the same research we are doing on the animal models. It gives us the capability to observe these dynamic processes, which we can apply by means of the ventilator to other research, such as gene therapy in asthma. We can take different points of research and combine them and streamline them to provide more information than we have had in the past.

Your institution has gradually replaced the SERVO 300 ventilators with the SERVO-i platform. From a clinical perspective, how does SERVO-i compare to the previous product generation?

The SERVO 300 was an outstanding ventilator, but a bit intimidating. From my perspective, the SERVO 300 took time and had a steep learning curve. The SERVO-i interface is phenomenal: in a very short period of time you can learn to use it and get a lot of information. It is very intuitive. The graphics allow for physiological output and data that is minimally filtered, and the rapid response rate and sampling are so high that you can easily interpret the graphics and see what is going on with your patient. Some of the problems with other graphic packages or interfaces are that they are so heavily filtered or have sample rates so low that you clinically lose the decision-making opportunities. Every breath looks exactly the same and the patient looks wonderful in graphics, but terrible at the bedside. Something is missing, and it is usually the inferior graphic interface that is hiding something. The SERVO-i graphic interface is also beneficial from a teaching aspect, since I can teach a resident about some of the activity on the screen, and we can go back and identify key events and learn about them. The answer is not to heavily sedate the patient, but to solve the problem and fix what is going on in that particular condition.

We are still using some of the same modes as we did with the SERVO 300, but some of the advances in SERVO-i in terms of weaning support are distinctly different. And some developments such as the Open Lung Tool allow us to see changes in our patients that were not possible before. I personally use the Open Lung Tool in patients with pulmonary collapse or severe atelectasis, where their peak inspiratory pressure is no greater than 35. In these types of situations we are beginning to use the Open Lung Tool instead of the oscillator. But not every



PICU staff members Jay Duncan MD, Patricia Bryon (PICU social worker), Mark Heullitt MD, Sharon M Goodman MD and Matt Jaeger MD

lung is recruitable. There are a number of dynamic processes going on. But if you need to re-establish the lungs to establish adequate gas exchange, the Open Lung Tool may be used.

We recently had an interesting case relating to this: a young man with gram-negative septic shock. In the old days, that would be somebody who would not do very well, with septic shock, lung disease, and a downward spiral. Part of the reason for that downward spiral would be that even as we started curing the sepsis, we would be damaging the lungs, which would deteriorate. In this young man, we had the septic shock under control within 48 hours, and his lungs are doing great. I started out with the Open Lung Tool and went over to adequate levels of PEEP. The Open Lung Tool helped to define the levels of PEEP that would be adequate, and he slowly started to respond positively.

We have done a lot of training with the Open Lung Tool, and we have provided training to physicians from other institutions as well. We are thinking about putting together a symposium for mechanical ventilation every other year in the U.S. The European counterpart in Montreux, Switzerland is a great opportunity for physicians to get together and learn and define new opportunities for treatments. We hope to have a lung recruitment symposium here in our research facilities during 2007, and offer lung recruitment training possibilities for remote participants who can't physically join us. There are a lot of opportunities for teaching, and physicians want to learn how to protect the

lungs earlier, from the first breath.

How have you managed the transfer of technology from a practical perspective?

We have 54 SERVO-i units in the PICU. We had 50 SERVO 300s, and we slowly phased in the new technology, and donated some and released some. We have a relationship with an organization in California, who have helped us transfer equipment to a hospital in the Philippines – the National Children's Hospital in Manila – as we have upgraded our fleet. It is all working equipment that they can make good use of, and it is a good feeling that it can be used to save other children's lives.

All the intensive care units now have SERVO-i. When you have one platform, it is so much easier to deal with. There is no perfect system out there; they all have their positive and negative points. But when you have three or four different ventilator models within an institution, you are constantly faced with negatives, as it generates confusion for the staff, for the rotating staff, for the clinicians, for staff wherever you need to use it right. We use SERVO-i as our mainstay. We use the oscillator as our secondary option, and we use ECMO in our tertiary care. Between these three, we can meet any patient's needs. Since introduction of SERVO-i and the Open Lung Tool, we are using much less of the others. It's now rare for us to use ECMO. And high frequency is now only used if we cannot recruit the patient's lungs

with conventional ventilation.

The SERVO-i is extremely accurate in its measurements of volume, in contrast to SERVO 300, which was not at that level. We recognize that 1 cc or 2 cc per kilo can make a significant difference. We know that we need to know more in pediatric and neonatal patients. As clinicians, we need to know that we are accurately delivering volumes. And that is a benefit of using SERVO-i.

What do you see as the most significant development in ventilation therapies over the past decade?

Definitely lung-protective strategies. I wrote the first review paper in 1994 or 1995 on recommending protective ventilatory strategies from a pediatric standpoint, with 150 references. Lung protective strategies are currently the major therapeutic consideration. In the future, I think the focus will be patient-ventilator synchrony, and what happens when patients are on the ventilator 24 hours a day. Marco Ranieri presented some fascinating research recently about what happens when patients are sleeping and how they interact with ventilators. It started me thinking along the same lines in pediatrics, since this is an area where we know very little. When you look at logs, it is not uncommon that patients have problems at night. We really need to focus our attention on how the patient and ventilator interface together. Currently, the Open Lung strategy and other lung recruitment



Laura Huber, RRT-NPS in NICU with 1.5 kg twin born at 30 weeks gestation, treated for respiratory distress syndrome

strategies, are the methods that are most positively affecting outcomes. We now know what damages the lungs as it relates to pulmonary overdistention and we are currently focusing on ventilator strategies to address this understanding. We know what pressures are destructive, we know about overdistention

and lack of adequate PEEP, and everybody is in agreement. And we are starting to agree on what protects the lungs, levels of PEEP and volumes. The next step is to fold all of this together and to recognize what is the best way to open and close lungs. That could be a major focus for the next five years. But the Open

Lung/lung protective strategies are still important to research, especially in children. We have seen that by simply modifying our approaches in children, we have improved mortality, without any big studies. But we still need to address these issues. ■

Biography

Mark Heulitt, MD, FAARC, FCCP, FCCM, is Professor of Pediatrics, Physiology and Biophysics at the University of Arkansas Medical Sciences School of Medicine; Associate Medical Director of Respiratory Care Services at Arkansas Children's Hospital; and Director of the Applied Respiratory Physiology Laboratory at Arkansas Children's Research Institute.

He initiated his training as medical doctor at Far Eastern University in Manila, Philippines in 1982, with internship, residency and the position of Pediatric Chief Resident at St. Luke's-Roosevelt Hospital at Columbia University during 1982-1986. His Neonatal-Perinatal Fellowship was obtained at Duke University Medical Center 1986-1988, and his Pediatric Critical Care Fellowship at Southwestern Medical School, Children's Medical Center, University of Texas from 1988-1990.

He started working at the Arkansas Children's Hospital in 1990 as Assistant Professor Pediatrics in Critical Care Medicine, and was named Co-Medical Director, Mobile ECMO, in 1992. He has held his current position as Associate Medical Director of Respiratory Care Services since 1993.

Mark Heulitt has won numerous awards and honors within the areas of critical care medicine and respiratory care, including the Strathmore's Who's Who list for 2001-2002 and the A Gerald Shapiro, MD Award from the New Jersey Society AARC for Outstanding Leadership in Respiratory Care in 2003. He has held several editorial assignments and positions for Critical Care Medicine, Pediatric Critical Care Medicine, Respiratory Care, and the Society of Critical Care Medicine.

Mark Heulitt has conducted substantial research in the area of critical care medicine and published numerous articles in peer-reviewed publications.

References

- 1) Carmack J, Torres A, Anders M, Wilson S, Holt S, Heulitt MJ. Comparison of work of breathing in spontaneously breathing young lambs during continuous positive airway pressure and pressure support ventilation with and without flow triggering utilizing the servo 300 ventilator. *Respir Care* 1995; 40:28-34.
- 2) Torres A, Anders M, Wilson S, Heulitt MJ, Yeh P. Evaluation of a method for measuring minute volumes during bias flow ventilation. *Respir Care* 1995; 40:22-27.
- 3) Heulitt MJ, Anders M, Benham D. Acute respiratory distress syndrome in pediatric patients: redirecting therapy to reduce iatrogenic lung injury. *Respir Care* 1995; 40:74-85.
- 4) Heulitt MJ, Torres A, Anders M, Wilson S, Carmack J. Comparison of total resistive work of breathing in two generations of ventilators in an animal model. *Pediatr Pulmonol* 1996; 22:58-66.
- 5) Fiser R, Torres A, Holt S, Wilson S, Heulitt M. Isolation valve increases work of breathing in a mechanically ventilated pediatric animal. *Respir Care* 1997; 42(7):688-692.
- 6) Heulitt MJ, Desmond B. Lung protective strategies in pediatric patients with ARDS. *Respir Care* 1998; 43(11): 952-960.
- 7) Heulitt MJ, Sanders RC, Holt SJ, Rhodes SM, Thurman TL. Comparison of total work of breathing during CPAP between two neonatal ventilators in a neonatal animal model. *Pediatric Critical Care Medicine* 2000; 1(2):170-175.
- 8) Holt SJ, Sanders RC, Thurman TL, Heulitt MJ. An evaluation of automode, a computer-controlled ventilator mode with the Siemens Servo 300A, using a porcine model. *Respir Care* 2001; 46(1):26-36.
- 9) Sanders RC, Thurman T, Holt SJ, Taft K, Heulitt MJ. Work of breathing associated with pressure support ventilation in two different ventilators. *Pediatr Pulmonol* 2001; 32:62-70.
- 10) Heulitt MJ, Holt SJ, Thurman TL, Wilson SW. Effects of continuous positive airway pressure/positive end expiratory pressure and pressure support ventilation on work of breathing utilizing an animal model. *Respir Care* 2003; 48(7):689-696.
- 11) Heulitt MJ, Wankum P, Holt SJ, Thurman TL, Hall RA, Simpson P. Reliability of measured tidal volume in mechanically ventilated young pigs. *Intensive Care Medicine* 2005 (in press).
- 12) Heulitt MJ. Patient Interactive Ventilation. *International Journal of Intensive Care*, October 2003.
- 13) Heulitt MJ, Jones SM, Holt SJ, Thurman TL. Measurement of respiratory mechanics in developing rats: comparison of two methods. *Journal of Applied Physiology* (submitted).

Books

Heulitt MJ. Physiology of the Respiratory System. In: Fuhrman B, Zimmerman J, editors. *Pediatric Critical Care 3rd Edition*. Mosby; 2005.

Heulitt MJ. Support Modes of Ventilation. Weaning from Mechanical Ventilation in Pediatric Patients. In: Papadakos PJ, Lachman B, editors. *Mechanical Ventilation: Clinical Applications and Pathophysiology*. 1st edition. Elsevier; 2005.

Heulitt MJ. Co-section editor physiology/pathophysiology chapter Respiratory Diseases and clinical section Clinical Disorders of Respiratory Conditions. In: Slonim AD, Pollack MM editors. *Pediatric Critical Care Medicine*, 3rd edition. Lippincott Williams & Wilkins 2005.

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| Respiratory Training and Education |

There are 151 respiratory therapists on the staff at Arkansas Children's Hospital to serve the PICU, NICU, CVICU and Burn Unit, as well as patient air and land transports. Coordinating training and education procedures may be a challenge in this dynamic environment, but is of great significance for optimal workflow. Critical Care News spoke with respiratory therapists Randy Willis, staff development/clinical specialist for NICU, CVICU and PICU, and Ben Downs, staff development and education in respiratory care.



Ben Downs, RRT, Staff Development and Education and Randy Willis, RRT, Staff Development/Clinical Specialist for NICU, CVICU and PICU discuss the challenges of training and education

There is an extensive RT staff at this institution. How do you handle continuing education and training?

Randy Willis: Once a year we hold a conference to help our staff stay current, which is also offered to staff at hospitals in surrounding states. We conduct continuing education at this conference to assess skills validation and to discuss any new needs or issues that have developed that need to be addressed in education and training.

Ben Downs: We also have a new monthly program that recently started, which we are very proud of: the RCS Educational Rounds. It's a wonderful opportunity for respiratory therapists to come together and review case studies and presentations on special topics. We also have

physicians participating in the program. It has been very successful, and we are already booked into next year. It is also an opportunity for respiratory therapists to earn Continuing Education (CE) credits towards their licenses.

What is your objective with regard to continuing education and training?

Randy Willis: Our objective is to provide our RT staff with a minimum of 12 training opportunities per year. But I think that Ben and I have averaged at least 50 educational opportunities for our staff to attend this past year, either in meetings and presentations, or online training.

Ben Downs: We work together to develop a hospital online program for RTs to read about special new procedures, or to refresh skills in

procedures that they have not used recently. We put together in-services online, visual tools as well as written tools that they can review and refer to.

What do you do about specific modes of ventilation or new techniques to educate and train on?

Randy Willis: This requires a lot of in-servicing. One example is our Burn Unit. They have used a lot of BiVent in their ventilation therapy, which the rest of us were not too familiar with. We suggested strategies to physicians to streamline our management. We contacted MAQUET, who coordinated with an RT specialist in Springfield, Missouri, who helped us set up new protocols and guidelines for respiratory management in our Burn Unit.

Ben Downs: We have some pretty good tools to help us disseminate information and help with in-services. This is important since we have a large RT staff here: 151 respiratory therapists, which means that training everyone can be a challenge at times. But we have in-services. When incorporating something new for everyone, it is especially important that everyone understands this new information.

You have upgraded your ventilator fleet with new models in several ICU departments in recent years. How did you manage staff training?

Randy Willis: We now use SERVO-i in all of our intensive care units: Burn, CV, NICU, and PICU. We initially started switching in the PICU, Burn Unit and NICU. We transitioned with 20 SERVO-i ventilators, and developed in-servicing for our staff to train on the new equipment. We contacted MAQUET, who sent in a clinical specialist and helped us set up a program. The specialist was here for a week, basically 24 hours a day, to help us implement the program initially. Even though we initially had had a few SERVO-i units that many of our therapists had exposure to, we wanted comprehensive training when the more extensive fleet was coming in.

Ben Downs: Most people here were familiar with the SERVO 300, but there were some jumps to understanding the new technology. A lot of the basic principles were the same, though, which made for an easier transition.

From an RT standpoint, what was your experience of the transition process?

Randy Willis: It went smoothly and flawlessly from our perspective. We like the new modes such as SIMV-PRVC, especially up in the nursery. It allows us to provide the babies with pressure-supported breaths spontaneously and still have a controlled breath that is pressure regulated. It has become our primary mode in the nursery.

Ben Downs: We had the basic skills from the SERVO 300. When we switched over to SERVO-i, the biggest thing was going from a ventilator where you turned knobs to touchscreens and graphic presentation of information. The learning curve on the pre-use check and flow module took a little time. We had some challenges in teaching everyone how we were addressing the circuit compensation, making sure that everyone understood how to handle it, especially with our smallest patients.

How does having one platform in all departments affect the respiratory therapists?

Randy Willis: That works well for us, especially with 151 respiratory therapists in all of our ICUs. Some of the RTs are dedicated to the PICU, NICU, Burn Unit, etc; we have 3-4 core therapists on every shift, but we also have a large number of staff who rotate between the units. SERVO-i works great in this respect. Staff are pulled to different units, depending on

patient volumes. They find the same ventilator and can provide therapy no matter where they are for that particular day, for infants or larger pediatric patients. It greatly minimizes the risk for errors, which easily happen if a hospital is using five or six ventilator models. It's scary enough if you are used to working in one unit and are pulled into another department needing extra staff. It's reassuring to have the same familiar ventilator platform wherever you go.

What are the proportions of helicopter transports to ambulance transports?

The majority come in by helicopter. Ambulances are used for cases where weather prohibits air transport, or the helicopter is down for routine maintenance.

Do the respiratory therapists accompany patient transport flights?

We have different teams that accompany the different types of patients. For nursery patients, a nurse and a therapist. For all other transports there is a physician, a nurse and a respiratory therapist in attendance. For twins, we have double teams from the nursery.

How many patient transports are planned, versus emergency?

For the nursery they are almost always unplanned. We don't know until they deliver the babies. The patients will be coming from smaller hospitals, and we are taking them, but many of these are emergency situations with patients who need to be stabilized. We have roughly 150-170 patient air transports per month, so the numbers are quite substantial. There are three teams on call at all times, with a fourth team that can be called in if needed.

What are the most important factors in terms of treatment when transporting a ventilated patient by helicopter?

Ideally, the patient should receive the same ventilation therapy in air transport as he does in the ICU, since we are dealing with the same problems and complications. The traditional transport ventilators are limited therapeutically to just a few ventilation modes. When we first started out in the nursery we saw babies coming in on pressure-controlled ventilation. If we could match that situation with PRVC, we could keep the same lung protective strategy and follow the patient clinically in a more optimal manner. ■



Respiratory care in the NICU

Comprehensive respiratory research |

The Arkansas Children’s Hospital Research Institute (ACHRI) is a non-profit organization owned by Arkansas Children’s Hospital. It provides research programs in infectious disease, endocrinology, osteogenesis and pediatric pharmacology, and is home to the Center for Applied Research and Evaluation. Critical Care News met with Mark Heulitt and research respiratory therapist Shirley Holt, RRT, to hear about the intensive care research projects to evaluate and modify ventilation therapies for the smallest of neonatal patients.



The laboratory has developed research models in ventilating animals as small as 10 grams

Can you tell us about some of the innovative research and work on methods you are conducting here?

Mark Heulitt: We have a number of computer systems measuring in milliseconds when we conduct ventilation therapy research on animals. We can validate directly from the ventilator to the computer system, and at all times look at this information together and analyze it. That allows us to change things when we are trying to validate new equipment, or evaluate protocols that need adjustments. We also have a state-of-the-art blood gas system, as well as Aerogen nebulizers. Our research lab is essentially a mini ICU; we have all the capabilities that the intensive care department offers right here in our laboratory.

We also use a forced oscillation respiratory mechanics system. This is a computerized ventilator that is connected to a cylinder and piston. The computer knows the position of the piston at all times and the diameter of the cylinder. This system allows us to utilize a forced oscillation technique, to make respiratory mechanics measurements. We are not

dependent upon gas flow moving in and out of the lungs; we are dependent upon measurements moving in the system. When utilizing this system, we are able to determine the resistance of the endotracheal tube and subtract it. This gives us the capability of obtaining very accurate measurements in very small models – animals down to 10 g. The system is extremely sensitive. We are currently using this on intubated subjects, and are modifying it for use in non-invasive ventilation.

Shirley Holt: We have modified masks for use in non-invasive ventilation in our pig models, and we can intubate and ventilate rats as small as two weeks old. To intubate our rats, we use IV catheters. We can subtract the resistance in the system, so we can get very accurate measurements in developmental models. Since rats grow so quickly, they are ideal models for respiratory dynamics. The same measurements would take years in human growth development. We have different sized cylinders, so we can treat and analyze patient data from animals as small as 10 g up to 10 kg in size.



Shirley Holt, RRT and Dr Heulitt in their state-of-the-art research facility

ID#	Sex	Arterial			Pulmonary Arterial			Venous			Pulmonary
		Flow	Pressure	Volume	Flow	Pressure	Volume	Flow	Pressure		
100	127	98.00	74.00	84.00	31.00	17.00	24.00	0.00	0.00	100.00	
101	128	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
102	129	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
103	134	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
104	134	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
105	123	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
106	122	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
107	121	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
108	120	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
109	119	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
110	118	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
111	117	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
112	116	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
113	115	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
114	114	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
115	113	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
116	112	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
117	111	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
118	110	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
119	109	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
120	108	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
121	107	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
122	106	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
123	105	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
124	104	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
125	103	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
126	102	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
127	101	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	
128	100	100.00	76.00	86.00	31.00	17.00	24.00	0.00	0.00	100.00	

The Data Acquisition Record streamlines research parameters from the tracheal tube and ventilator in regard to hemodynamics, airway functions and cardio/pulmonary monitoring

Our data collection system allows us to download information collected at the patient’s airway, as well as hemodynamic and continuous blood gas information. In addition we utilize the SERVO-i ventilator, which is microprocessor based, and have the capability to receive a signal directly from the ventilator that allows us to see when the inspiratory valve opens. This lets us see when the patient took a breath and when the ventilator responded to that breath. It enables us to do very sophisticated research.

Are there any problems with leakage with the rats intubated with IV catheters?

Shirley Holt: No, we use several different sizes. For the tiniest animals we use the smallest IV catheters available, and for larger animals we go up to the 14 Fr size. We almost never have leakage problems. ■

| Maintaining cost efficiencies in respiratory care |

Providing quality respiratory care service in a non-profit environment at one of the largest children's hospitals in the United States creates special challenges for administrators who must also maintain cost efficiencies. Critical Care News discussed these challenges with Patty Burge, RRT, Director for Respiratory Services at Arkansas Children's Hospital.



The intensive care environment is oriented towards children, with bright colors, murals and light. The institution was recently entirely remodeled



Patty Burge, RRT, Director for Respiratory Services

This is a non-profit organization. What are your objectives regarding cost efficiency for respiratory therapy services?

Our main objective is quality patient care. As far as cost savings are concerned, we have found it very beneficial to have one ventilator platform for use throughout the institution. I have a very large staff of 181 employees. To ensure that everyone is proficient on the equipment to be used, it has been very beneficial to streamline everyone's training to one single ventilator platform.

We have one of the largest RT departments in the United States. The therapists are working throughout the hospital, and in all of the intensive care environments. We have a very high acuity here; we average around 50 ventilators, where the average children's hospital has between 25 and 30 ventilators running on any given day. We have 112 intensive care beds, which also contributes to our high acuity.

What were the key factors that contributed to the decision to invest in one fleet of ventilators?

We have used SERVO ventilators for many years. We had the 900B and 900C, and the SERVO 300, so the SERVO-i was a natural progression. It's

ideal because it can be used on patients from tiny infants all the way up to adults.

We started by replacing the SERVO 900. We usually purchase about 20 ventilators at any given time, so the replacement process occurred naturally and gradually over a period of time.

A big advantage has been the work of Dr Heulitt and education of the medical staff in the uses of SERVO-i. They respect his research and they have accepted his recommendations for use of the ventilator.



Will there be continued growth of respiratory services in the future?

I started here as a supervisor of the pulmonary lab in June 1980. At that time there were only about 18 respiratory therapists. We have seen enormous growth and the acuity is extremely high. We are the only children's hospital in a large area, and I am sure that the patient admissions will continue to increase in the coming years. ■

Pediatric patient on pet therapy day. The Arkansas Children's staff may enroll their pets in a three week training program. When pets are approved, they visit the hospital once a week, at the wards and in play areas

The Post-Graduate Lung Recruitment Workshop at the Academic University Hospital in Uppsala was attended by a broad range of anesthesiologists and intensive care physicians from many countries around the world. One of the most appreciated sessions was a lecture by Professor Göran Hedenstierna, Professor of Clinical Physiology and head of the hospital Nuclear Department. Professor Hedenstierna is well known to the global anesthesia and intensive care communities, having conducted substantial research in anesthesia, lung mechanics and regulation of vascular tone for many years. Critical Care News met Professor Hedenstierna to discuss his lecture topics of lung collapse and airway closure.

Mechanisms of atelectasis in anesthesia and intensive care



Professor Göran Hedenstierna in the Department of Clinical Physiology, with view of Uppsala Castle in background

Can you tell us about your experience in regard to the relationship of lung collapse and airway closure?

Lung collapse and airway closure are common phenomena during anesthesia. The changes we see during anesthesia are also seen in the ICU, but much more pronounced.

The FRC is reduced by about 20% during anesthesia. The interesting point is that when you are upright, you may have an FRC of about 3 liters, depending on body size. If you lie supine, it will be reduced by about 0.7 or 0.8 liters. Abdominal organs push up the diaphragm, which reduces the FRC. And then we are going to anesthetize, which will bring it

down to about 2 liters, which means we are close to the residual volume. Residual volume is the volume you have in the lungs at maximum expiration. It is important to realize this simple fact: when we are treating patients during anesthesia in the supine position, the lung volume is at residual volume.

Can that have an effect on the patency of the lung tissue?

That is what we asked 20-25 years ago. In the CT scans of the anesthetized subject, it is evident that the diaphragm and liver have moved cranially, and consequently reduced FRC. The major cause of reduced FRC in anesthesia is cranial displacement of the diaphragm.

We found something interesting at the bottom of those lung CT scans that looked like a "roaring sea." We conducted animal experiments, and CT scans showed a large amount of densities. Some animals have severe hypoxemia in the supine position. When we removed the lungs of some of the animals and made a histological assessment, we found normal tissue and some completely collapsed tissue, with a very sharp border. This corresponds to atelectasis, with some edema and congestion. When we presented this for the first time, it was a new discovery.

So we established that the dense area was atelectasis, and we wondered whether it was due to anesthesia or something else. We moved on to patients who were awake and spontaneously breathing, and found no densities in the CT. We thereafter induced anesthesia in these patients with a face mask, under spontaneous breathing, and found the densities, or atelectasis, appearing almost immediately. When we paralyzed the patients, the densities increased. We know that the anesthetic is the cause of atelectasis, and that we do not need to paralyze a patient to induce atelectasis.

What else was established in these first studies with regard to atelectasis?

In post-operative CT scans of these patients, we found that these densities, or atelectasis, remain for one hour after anesthesia, or even for several days. You may see a slow decrease in atelectasis over time, but more than a quarter may still have atelectasis on the fifth day post surgery.

From these first studies we concluded that atelectasis during anesthesia is found during spontaneous breathing and artificial ventilation, with intravenous and inhalational anesthetics, and that the atelectasis will last a few days post-operatively, and may cause post-op pulmonary complications. Loss of muscle tone is a pre-requisite for developing atelectasis.

We could also conclude that about 90% of all patients will develop atelectasis during anesthesia, and that 3-4% of lung area (basal) is affected, with at least 10-15% of lung tissue involved. This cannot be seen on an X-ray. In

uneventful anesthesia, it's standard to leave the patient with at least 10-15% of the lung collapsed.

After thoracic surgery, atelectasis is much more pronounced. Over 40% of lung tissue is collapsed, with a very slow reopening of the tissue. Most of the atelectasis is near the diaphragm.

What in your opinion is the relationship of shunt in atelectasis?

Shunt (perfusion of the lung that is not oxygenated) is nearly always caused by atelectasis in the anesthetized, otherwise lung-healthy subject. This is also the major cause of acute respiratory failure in the intensive care setting, but there, another cause of collapse can be fluid filling of the alveoli. But shunt is the major cause of hypoxemia, or impeded oxygenation, in both cases.

Shunt was established to have a direct correlation to atelectasis in anesthesia. We also found that it is related to intensive care. We did a CT and gamma camera SPECT study in patients, with CT scan and vertical distribution of ventilation and perfusion in the same lung segment. We found atelectasis in the bottom of both lungs, and could see how the ventilation and perfusion were distributed in the lung, from top to bottom.

The perfusion increased down the lung – a common distribution pattern when the patient is awake, during anesthesia, and in the ICU, provided you do not have a vascular abnormality. There was a slight decrease in perfusion at the bottom of the lungs.

In the healthy and awake subjects, ventilation also increases along the lung, similar to the perfusion. But in the anesthetized subject, we have a very different pattern. Most of the ventilation goes to the upper half, so there is a clear mismatch. In the upper half, ventilation is in excess of perfusion. In the lower half, we have less ventilation than perfusion. And at the bottom, we have no ventilation at all. This corresponds to the atelectasis.

So why do we have a decrease in ventilation in a zone above the atelectasis? That is due to airway closure. When you exhale, you can get closure of air ducts, a normal phenomenon. It is lung volume dependent. In healthy adult subjects, sitting up, there will be no airway closure during normal breathing. But if they lie down, they will suffer from airway closure as lung volume decreases. During anesthesia the further decrease in lung volume caused airway closure in all subjects above 30 years of age. If airways remain closed all the time, the gas in

closed regions will be absorbed. The airways that open for inspiration and close for expiration will allow for a certain level of gas exchange, but it is reduced. And that can explain the decrease of ventilation in the zone above the atelectasis.

An important issue for many physicians is how to manage airway closure with regard to aeration and recruitment. Do the airways have an important physiological role that is not always fully appreciated?

Yes, they do indeed. In fact, we should remember that airway closure is a normal phenomenon, which is more obvious or dominating if lung volume is reduced. When lung volume is reduced, alveoli decrease in size, and the airways do as well. The decrease in lung volume, with the subsequent decrease in airway caliber, promotes airway closure. So during anesthesia, or in intensive care when using muscle paralysis or sedatives, you reduce or eliminate respiratory and other muscle tone. Then lung volume is reduced, and you provoke or promote airway closure. So this is a normal phenomenon made worse by the decrease in lung volume.

Airway closure was first demonstrated in the mid-60s. People who know about airway closure have thought that it is something that occurs in diseased conditions. It was proposed as a measure of early obstructive lung disease before any other sign of the disease was existent. For some reason, people have disregarded the fact that it is a normal phenomenon with impact on oxygenation of lungs. In fact with age, oxygenation of blood is impaired; PaO₂ successively decreases as we get older. The explanation is airway closure.

Airway closure and atelectasis explain as much as three-quarters of oxygen impairment during anesthesia.

What can we do about airway closure and atelectasis?

In regard to airway closure, we can elevate FRC by means of PEEP. In regard to atelectasis there are a number of possibilities: give PEEP, increase muscle tone, "sigh" the patient, and avoid high O₂ concentrations (FiO₂), as this can affect the absorption of gas.

What happens to the lung physiologically when PEEP is administered or discontinued?

CT studies show that in patients with large atelectasis (>25% of lung tissue) giving PEEP

of 10 cm of water almost eliminates the atelectasis. If PEEP is discontinued all the atelectasis returns, so there is no lasting effect. When we discontinue the PEEP for any reason, such as suctioning, the atelectasis will return within a minute.

We learned about the lung physiological effects of PEEP by looking at the perfusion distribution to anesthetized patients with gamma camera technique. We could see that at 10 cm of water, PEEP redistributes with more perfusion going to the lower lung. If we have atelectasis in the lower portion, we get shunt with ZEEP. We can also get shunt with PEEP, even if the atelectasis is reduced, since the perfusion has been forced down. In general, we do not see an improvement in PaO₂ by applying a standard PEEP in anesthesia.

As a routine tool, PEEP will not improve oxygenation. Hewlett et al. in 1974 concluded that there was no place for the indiscriminate use of PEEP in routine anesthesia. We can conclude that PEEP has no effect on PaO₂ in unselected patients, has no remaining effect after discontinuation, and decreases the cardiac output.

What about the use of PEEP in the intensive care setting?

Yes, you can recruit lung tissue that has collapsed by increasing airway pressure. When you apply a PEEP, you have to increase the inspiratory pressure in order to insufflate the air and gas into the lungs. The increased airway pressure required when applying PEEP will help open up atelectatic regions. Strictly speaking, it is not the PEEP per se that recruits alveoli; it is the airway pressure, or pressure above PEEP that is responsible for the recruitment phase. The PEEP will prevent re-collapse. The problem with PEEP is that there is no lasting effect. As soon as it is discontinued, the alveoli will collapse again within a minute. Many people might not yet realize this fact. So when you give PEEP and think that you have opened up the lung, but stop using it, the lung will collapse. Another thing with PEEP is that it will affect the distribution of blood flow in an unfavorable way. It might be needed in order to keep the lung open, but one should realize that PEEP is not the ultimate tool. It is a tool that may be valuable in the intensive care setting, but it may not have the same value in anesthesia.

In contrast to PEEP, what we call vital capacity maneuvers (lung recruitment procedures) do have a lasting effect. We may ask why one technique does not have a long lasting effect when another one does. With

PEEP, we open up unstable alveoli, but the alveoli remain unstable and need continuous support by means of airway pressure. The difference with vital capacity maneuvers is that they restore the stability of the alveoli.

When a low compliance lung is treated with a vital capacity maneuver, the alveoli become stable because surfactant spreads on the alveoli surface. To get this distribution of surfactant, you need a vital capacity maneuver, at a higher airway pressure compared to a normal size tidal breath, which is not enough to open up collapsed areas and generate new surfactant.

What effects do increasing muscle tone or “sighing” the patient have on atelectasis?

In regard to preserving or increasing muscle tone, it has been demonstrated by Tokics et al. that ketamine prevents atelectasis but if paralysis is added, collapse occurs as with other anesthetics. We also determined that phrenic nerve stimulation that tenses the diaphragm reduces atelectasis, but it is very difficult to carry out.

In regarding to “sighing” the patient as a recruitment maneuver, we established early in anesthesia, with lung healthy patients in CT scans, that atelectasis was present at atmospheric pressure. We then inflated the lung at a pressure of 10 cm H₂O, and nothing happened to the atelectasis. We inflated the lung with 20 cm H₂O, and nothing happened. The atelectasis was similar to how it was at atmospheric pressure. So what I was taught 40 years ago, to give a double tidal volume now and then, has no effect. You do not open any atelectatic tissue by inflating the lung with 20 cm H₂O in healthy subjects. However, this is not the case in ARDS patients, where you can open up tissue! We finally opened up the lung at 40 cm H₂O. This will have a lasting effect, as established by Rothen in 1993. But 40 cm of airway pressure during a longer period of time can stop the heart. We have found that eight or nine seconds is enough to open what you may be able to recruit.

“Sighing” the patient requires a vital capacity maneuver. One can ask about the risks of barotrauma, volutrauma, and the short-term decrease in cardiac output.

What is your experience in research of the use of 100% oxygen?

In patients who are ventilated at 100% oxygen after recruitment maneuvers, there are no lasting effects. Oxygen concentration has a very strong influence on atelectasis. We can

conclude that a vital capacity maneuver is efficient with a moderate FiO₂, but with 100% O₂, atelectasis will return within five minutes of ventilation. This leads to the question of whether pre-oxygenation at 3-4 minutes during anesthesia induction causes atelectasis, and whether we should avoid pre-oxygenation.

We looked at a group of patients whose pre-oxygenation was limited to 30% oxygen. When they were awake there was no atelectasis. When they were anesthetized, there was no atelectasis either. At 30 minutes of anesthesia, there was a tiny amount of atelectasis. If you look at a time sequence, you see that 30% oxygen is associated with very little atelectasis over time. So we have learned to avoid a high FiO₂ during anesthesia induction and during anesthesia, but to balance this benefit with the risk of hypoxemia.

In a recent study by Edmark et al., anesthesia was induced in patient groups with different O₂ concentrations. One group received 100% O₂, one group received 80% O₂, and the third group received 60% O₂. The groups with 60% and 80% pre-oxygenation were associated with very little atelectasis. But the 100% group was associated with broad levels of atelectasis. Apnea tolerance was also studied in the same groups, and it may be said that pre-oxygenation at 80% O₂ will not be too dangerous for these patients.

The benefit of lowering O₂ concentration during the induction of anesthesia is that there is less atelectasis, and recruitment maneuvers may be used afterward to retain this effect. Magnusson et al. established that post-oxygenation after surgery and before extubation may have an effect on atelectasis.

The mechanisms of atelectasis during anesthesia are loss of muscle tone (fall in FRC, increase in airway closure), high inspired oxygen concentration (> 80%), and impaired surfactant function.

The reason why atelectasis reappears so fast when we discontinue PEEP must be explained by surfactant impairment. Collapsed alveoli mean that the surfactant will be destroyed. If we open up the alveoli, with additional PEEP, the surfactant function is not restored, and the alveoli remain unstable. The combination of these factors sets off a rapid chain of events that leads to atelectasis in just minutes.

However, if we make a vital capacity maneuver, surfactant is released from its production sites and delivered to the alveolar wall and bronchi. The vital capacity maneuver restores surfactant function, and the alveoli remain stable.

How does this situation in anesthetized patients differ from ARDS patients in the ICU?

Collapse in the ARDS situation differs somewhat. Not only is there atelectasis, but also fluids that fill the lungs. Gattinoni studied these patients with CT, and found densities in the lower parts of the lung. Neumann et al. studied the effect of breathing on atelectasis in ARDS patients, and established that there is considerable collapse with certain PEEP levels in the models. PEEP of 20 or 25 is necessary to reduce the cycling collapse. In ARDS patients, a certain amount of atelectasis and fluids can be seen at end-expiration. At inspiration we will open up some of them, but there is further collapse at the next expiration. This cyclic collapse may be more harmful to the lung than the continuous collapse that the other parts of the lung may be exposed to, or even than over distension of tissue that is aerated all the time.

With regard to atelectasis, can we modify the ventilatory support by adding spontaneous breathing?

I am interested in that, since we see such differences between the mechanically ventilated and spontaneously breathing healthy lung. Christian Putensen in the early-to-mid-90s performed Airway Pressure Release Ventilation (APRV) experiments using dogs. In mechanical ventilation, the PaO₂ increased when spontaneous breathing was present. Shunt was also reduced in these subjects. Putensen and colleagues also established that in human patients on APRV, PaO₂ can be improved in the presence of spontaneous breathing with reduced shunt as well.

How do spontaneous breaths improve gas exchange, and what are the effects on lung volume, atelectasis, ventilation and perfusion?

There is a beneficial effect on aeration, as demonstrated in studies with CT scans. There is also an effect of the APRV on ventilation distribution. In a gamma camera study by Neumann et al, from diaphragm to apex, and from posterior to anterior, the ventilation was shown in more apical and anterior regions. With APRV, we are able to distribute some ventilation to other regions, where we have more perfusion.

Thoracic EIT (Electrical Impedance Tomography) has also been used to show how ventilation is distributed and the effects of respiratory modes on regional ventilation.



Professor Göran Hedenstierna

In Pressure Control, most of the ventilation goes to the upper region. In APRV there is ventilation in the upper part, which is being mechanically ventilated, but more in the lower part where spontaneous breathing is evident. If we switch to CPAP, there is mainly spontaneous breathing in the lower part. So you can see how it varies in regard to distribution of the spontaneous breath and the mechanical breath.

Why do we have these differences?

When you have a relaxed diaphragm during mechanical ventilation, there is a certain amount of pathology and atelectasis consolidation. When we inflate the lung by increasing airway pressure, we push away the diaphragm and elevate the rib cage to some extent. The displacement of the diaphragm will be mainly in the upper part of the diaphragm because the pressure in the upper, anterior part of the abdomen is lower. If we say that

we have an edematous patient with ascites and fluid, the pressure will increase along the abdomen, corresponding to 1 cm H₂O to 1 cm of distance. If there is a 20 cm distance, there will be a 20 cm higher pressure in the lower part than in the upper part. Therefore it is easier to push away the diaphragm in the upper part than in the lower part. So we get a preferential displacement of the diaphragm anteriorly in the supine position.

Now with spontaneous breathing, we have a completely different displacement. Most of the movement is in the dorsal part. This is because the diaphragm in this situation becomes an active muscle, moving itself. We have more muscle fibers in the dorsal section, and an elongation of the fibers in this area compared to the anterior part. This elongation makes the fibers stronger. The muscle is stronger and moves more forcefully in the dorsal part than the anterior part, during an active spontaneous breath.

In synchronized ventilatory support, what is the best way to trigger the ventilator, by flow, pressure or EMG?

This is an area of interest. Despite some of the benefits of APRV, I don't think it is the perfect tool. This sustained non-synchronized pattern of behavior cannot be ideal. If we look at the triggering of the ventilator, Martin Tobin et al. demonstrated that diaphragm contraction causes

muscle contraction after some time, which lowers airway pressure. But there is a time lapse until the pressure has dropped enough in the ventilator to trigger a breath. So from when the EMG signal is transmitted from the brain, there is a delay of half a second or more before the ventilator provides a breath to the patient.

In recent times, Sinderby et al. have researched the use of a catheter to enable the recording of the diaphragmatic excitation,

or EMG. If the diaphragm provides a good signal, this could be a shortcut with less delay. The signal is also proportional to the demand of the patient. So we can use the magnitude of the signal to determine the tidal volume. If this could be made into a clinically usable tool, it could be very useful because you would have a type of pressure support that is totally controlled by the patient, provided he or she is delivering a signal from the diaphragm. ■

Biography

Göran Hedenstierna was named Professor in Clinical Physiology at Uppsala University in 1988, and held the position of Chairman of Clinical Physiology for 14 years. He has been Chairman of the Department of Nuclear Medicine at Uppsala University Hospital since 2002. He has also been visiting or honorary professor at numerous university institutions in the US, France, China and Italy.

He has published over 430 scientific papers and reviews, primarily in his scientific research areas of atelectasis and gas exchange, lung edema and nitric oxide. He holds a number of scientific committee positions as chairman or member in institutions such as the Swedish Research Council, Swedish Heart and Lung Foundation, the Fleischner Society, the Royal College of Anaesthetists and the German Society of Anaesthetists.

Göran Hedenstierna has won numerous awards, including G Göransson's Young Scientist's Award 1974, Thureus Award for Excellence in Medicine 1989, T. Sjöstrand Lecturer in Clinical Physiology 1993, Radiometer Prize (3) 1995, Martin Holmdahl Lecture in Anaesthesia 1997, Litchfield Lecture Oxford 1998, and Erik Huslfed Lecture Copenhagen 2000.

References

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- 1) Wabba, RW. Perioperative Functional Residual Capacity. *Can J Anesth* 1991; 38:384-400.
- 2) Brismar, B, Hedenstierna G, Lundquist H, Strandberg A, Svensson L, Tokics L. Pulmonary Densities During Anesthesia with Muscular Relaxation: a Proposal of Atelectasis. *Anesthesiology* 1985; 62:422-428.
- 3) Gunnarsson L, Tokics L, Gustavsson H, Hedenstierna G. Influence of Age on Atelectasis Formation and Gas Exchange Impairment During General Anesthesia. *Br J Anaesth* 1991; 66:423-432.
- 4) Tokics L, Hedenstierna G, Svensson L, Brismar B, Cederlund T, Lundquist H, Strandberg A. / Distribution and Correlation to Atelectasis in Anesthetized Paralyzed Humans *J Appl Physiol* 1996; 81:1822-1833.
- 5) Tokics L, Strandberg Å, Brismar B, Lundquist H, Hedenstierna G. Computerized Tomography of the Chest and Gas Exchange Measurements During Ketamine Anaesthesia. *Acta Anaesthesiol Scand* 1987; 32:684-692.
- 6) Hedenstierna G, Tokics L, Lundquist H, Andersson T, Strandberg A, Brismar B. Phrenic Nerve Stimulation During Halothane Anesthesia. Effects of Atelectasis. *Anesthesiology* 1994; 80:751-760.
- 7) Rothen HU, Sporre B, Engberg G, Wegenius G, Hedenstierna G. Re-expansion of Atelectasis During General Anesthesia: a Computed Tomography Study. *Br J Anaesth* 1983; 71:788-795
- 8) Rothen HU, Sporre B, Engberg G, Wegenius G, Reber A, Hedenstierna G. Prevention of Atelectasis During General Anaesthesia. *Lancet* 1995; 346(8973):514-5.
- 9) Edmark L, Kostova-Aherdan K, Enlund M, Hedenstierna G. Optimal Oxygen Concentration During Induction of General Anesthesia. *Anesthesiology* 2003; 98:28-33.
- 10) Benoît Z, Wicky S, Fischer J-F, Frascarolo P, Chapuis C, Spahn DR, Magnusson L. The Effect of Increased FiO₂ Before Tracheal Extubation on Postoperative Atelectasis. *Anesth Analg* 2002; 95:1777-1781.
- 11) Neumann P, Berglund JE, Mondéjar EF, Magnusson A, Hedenstierna G. Effect of Different Pressure Levels on the Dynamics of Lung Collapse and Recruitment in Oleic-Acid induced Lung Injury. *Am J Respir Crit Care Med* 1998; 158 (5), 1636-1643.
- 12) Putensen C, Rasanen J, Lopez FA. Ventilation-Perfusion Distributions During Mechanical Ventilation with Superimposed Spontaneous Breathing in Canine Lung Injury. *Am J Respir Crit Care Med* 1994; 150(1): 101-108.
- 13) Putensen C, Rasanen J, Lopez FA. Interfacing between Spontaneous Breathing and Mechanical Ventilation Affects Ventilation-Perfusion Distributions in Experimental Bronchoconstriction. *Am J Respir Crit Care Med* 1995; 151(4); 993-999.
- 14) Putensen C, Mutz NJ, Putensen-Himmer G, Zinserling J. Spontaneous Breathing During Ventilatory Support Improves Ventilation-Perfusion Distributions in Patients with Acute Respiratory Distress Syndrome. *Am J Respir Crit Care Med* 1999; 159 (4):1241-1248.
- 15) Froese AB, Bryan CH. Effects of Anesthesia and Paralysis on Diaphragmatic Mechanics in Man. *Anesthesiology* 1974; 41:242-255.
- 16) Tobin MJ. Advances in Mechanical Ventilation. *N Engl J Med* 2001; 344 (26): 1986-1996.
- 17) Sinderby C, Navalesi P, Beck J, Skrobik Y, Comtois N, Friberg S, Gottfried SB, Lindström L. Neural Control of Mechanical Ventilation in Respiratory Failure. *Nat Med* 1999; 5(12): 1433-1436.

Post-graduate workshop in lung recruitment: concept, principles and practical clinical application



Professor Hedenstierna with workshop physicians

After the fundamental lecture in the mechanisms of atelectasis, workshop participants were given an overview of the inherent risks of ventilator-induced lung injury (VILI) in older methods of mechanical ventilation with high tidal volumes and low PEEP, such as initiation or maintenance of an inflammatory response, causing or accelerating lung injury. The limitations of applying pressure-volume loops in clinical practice were also discussed.

The concept and method of delivery for lung-protective ventilation management, guided by the physiological response of the patient, was also reviewed in the workshop. The recommendations for practice, ventilatory objectives and sequence of management decision-making as published by Marini and Gattinoni were included, as well as other publications and recruitment material tools.

The following aspects of interventional assessment were highlighted in the workshop:

- Guidance and standardization for a prescribed recruitment maneuver
- Identification of collapse point – PEEP titration
- Monitoring of breath-by-breath vital parameters for evaluation of the patient response to intervention
- Identification of patients unlikely to benefit from a recruitment maneuver
- Assessment of the patient response to intervention, by means of dynamic compliance and carbon dioxide excretion trends

Over a two-day period in the physiological laboratory, each workshop participant was given several opportunities to apply the course recruitment maneuver in clinical practice.

Lung recruitment maneuver

- 1) Assessment of baseline clinical situation.
- 2) Application of increased end inspiratory pressure (EIP), with corresponding increase in VT_{CO_2} (tidal elimination) as activated alveoli participate in gas exchange.
- 3) Monitoring of the plateau state of VT_{CO_2} , when no additional alveoli may be recruited.
- 4) A slow step-by-step decrease in PEEP to identify the maximum value of dynamic compliance ($C_{dyn\ i}$) as indication of lung compartments closing again. Collapse pressure was identified as the corresponding plateau/max value of $C_{dyn\ i}$ and VT_{CO_2} .
- 5) The lung was re-recruited at the previously used opening pressure.
- 6) PEEP was established at 2 cm H_2O above collapse pressure, and ventilation was maintained at this level.

The post-graduate lung recruitment course held in April at the Academic Hospital in Uppsala, Sweden attracted many international participants, with attending physicians from Scandinavia, Central Europe, Russia and Singapore. Critical Care News asked some of the participants about feedback after attending the educational and lab sessions.

Dr Csaba Micskei, anesthesiology and intensive care, Hungary

Why did you choose to attend this course?

My boss, Professor Béla Fülesdi, urged me to attend because we hope to offer a ventilatory course for colleagues in Hungary. We offer a resuscitation course, and we want to offer a ventilatory support course for post-graduate training at our hospital.

What is the most valuable thing you have learned from this course?

To use this physiological method and tool and become familiar with it, in order to see the ventilation curves and physiology from another perspective. After this course, I hope to use the recruitment maneuver; this course is a good practice run, giving physiological training before treating the patients.

Dr Luis Telo, pneumologist and intensive care physician, Portugal

What was your goal in attending the course?

I don't do recruitment maneuvers, but I am interested in the concept, and I wanted to learn how to do it.

What is the most valuable thing you have learned from the course?

To understand the mechanics of the lung, and the physiological process of opening the alveoli and keeping them open.

Will you try working with recruitment maneuvers when you return to Portugal?

Yes, I think so. We have two intensive care units at my hospital, with about five beds each. So we are a small group at my hospital and I



Dr Luis Telo, pneumologist and intensive care physician, Portugal



Dr Csaba Micskei, anesthesiology and intensive care, Hungary



Dr Beata Oscarsson and Dr Ingemar Lindström, Sweden, anesthesiologists and intensive care physicians

think some of my colleagues will be interested in learning about lung recruitment as well.

Dr Beata Oscarsson and Dr Ingemar Lindström, Sweden, anesthesiologists and intensive care physicians

What were your expectations of this course?

We expected to learn how to use these procedures and instruments when working with severely ill patients.

We are impressed by the international scope of this training. It has been a benefit to meet doctors from many other countries.

What was the most beneficial aspect of the training?

The fact that we could learn how to use the method and tool for clinical support in a physiological environment in the labs. It would have been difficult to learn about these recruitment maneuvers from books or manuals only.

Did you find the concept and procedure easy or difficult?

Once you have learned the basics, and can set reasonable expectations about how to run the

procedure and the clinical principles involved, it is very easy. However, it needs reflection and practice. But we are eager to use this concept, routine and tool on patients as soon as we get back home.

We suspect that in patients with different disease processes, there may be some challenges when applying this method. But that is why it is so valuable to learn the fundamentals in this setting.

We have a trend to have our patients less sedated on the ventilator, so this could be valuable to use on the sickest patients, in order to achieve spontaneous breathing more quickly.

Dr John Gannon, United Kingdom

Can you tell us about you background and why you are attending this course?

I am an anesthesiologist, with an interest in intensive care. That's the way most of the units in the United Kingdom are managed; they are run by anesthesiologists with an interest in critical care medicine. Our critical care environment is comprised of a high-dependency area and a critical care area. We have a total of 16 beds, broken down roughly to nine level three beds, and seven high dependency beds, but the match depends on what the needs are.

We recently purchased eight SERVO-i ventilators, and I learned about this course from our local MAQUET representative. I was interested in the lung recruitment procedure, and I thought this would be a useful way to learn about the concept, and become familiar with the mechanism of using it.

What course experiences have been most beneficial to you?

The hands-on experience with the recruitment procedure, before I start using it on my patients. But it was also beneficial to share experiences with the international delegates; it is interesting to meet fellow physicians from other parts of Europe and the world.

So you intend to use this methodology and tool on patients?

Yes, and hopefully I will be able to disseminate the knowledge back to my colleagues when I return home.

It has been valuable to train these recruitment maneuvers in a physiological lab environment, since you have a hesitancy to apply new methodologies directly on your patients for the first time. This also gave us the ability to try out some theories that perhaps otherwise we would not get the opportunity to do. ■



Dr John Gannon, United Kingdom

Faculty members of the VUMC University Hospital in Amsterdam were inspired to start their own series of post-graduate workshop courses after attending a session at the Uppsala University Hospital last year. Within a few months, they organized their first educational workshop in the series. Critical Care News met the educational faculty and lab participants after a comprehensive workshop session.

Alveolar recruitment: physiological basis and strategies, tools and application



Some members of the workshop faculty from the Amsterdam VUMC Medical Center are Drs Dick G Markhorst, PICU, Hagen Biermann, ICU and Jan Jaap Spijkstra, ICU

The Pathophysiology of ARDS and Consequences for Treatment was the subject of the lecture held by Professor Johan Groeneveld MD, PhD. After thoroughly detailing ARDS definition and pathogenesis from early to late stages, Professor Groeneveld summarized that the pathophysiology of ARDS depends on the underlying cause, whether direct or indirect, and that ARDS is a dynamic process dependent on injury, repair, effect of regional blood flow and ventilation. He also underlined the need for a simple, reproducible pathophysiological quantitative and specific test for ARDS.

Dr Jan Jaap Spijkstra presented the next

topic, Ventilator Induced Lung Injury, with particular reference to volutrauma and shear stress. He highlighted a review of the literature, with the growing insight that what was previously referred to as barotrauma should be defined as volutrauma. He underlined that the uneven distribution of the lung lesions in ARDS may easily lead to regional over-inflation and additional lung damage. The causes of shear stress were explained, and the role of shear stress in the development of VILI was discussed. The ways in which volutrauma and shear stress may lead to biotrauma were outlined, and subsequently the role of

biotrauma and the systemic progression to sepsis and multi-organ failure.

Dr Dick G Markhorst lectured on the topic of PEEP. He underlined the clinical benefit and role of PEEP in more uniform distribution, as well as alveolar PV relationship and vertical pressure gradients. The physiological conditions of alveolar interdependence and the effects of repetitive opening and closing, end-expiratory atelectasis and anti-inspiratory over-distention were outlined in detail. Dr Markhorst also presented the significance of pulmonary perfusion and the effects of PEEP on hemodynamic parameters, as well as the risks of excessive PEEP. He concluded his lecture with a comprehensive literature review, focusing on what has been reported as the optimal PEEP in recent years, and the ARDSnet data. He cautioned that a generic level of PEEP is not the objective; it is necessary to address the level of PEEP required by each individual patient dependent upon lung injury severity, in order to reach therapeutic goals supported by the current best evidence.

Alveolar Recruitment Strategies and Comparison of Techniques was presented in a lecture by Dr Hagen Biermann, with an overview of key principles, methods, experimental studies and clinical trials, with special emphasis on efficacy and hazards.

The aspects of prone position, adequate PEEP and tidal volumes, lowest acceptable FiO_2 and spontaneous efforts were emphasized. He also posed the question of whether the lung should always be opened, with respect to primary and secondary ARDS. Recruitment maneuver effectiveness, lavage in ARDS, oleic acid injury and a pneumonia model were discussed, with reference to an intercomparison of three models, with three levels of post-recruitment PEEP in regard to sustained inflation, incremental PEEP and high-level pressure control. Dr Biermann concluded that pressure-controlled ventilation should be preferred to

sustained inflation, and that the use of hemodynamic monitoring is essential during recruitment maneuvers. He also stressed the importance of repeating recruitment after patient disconnections, position changes or deterioration of mechanics and/or PaO₂.

Dr Jan Jaap Spijkstra followed with the topic of Lung Protective Ventilation, with reference to ventilator settings, tidal volumes and PEEP currently used in ICU departments all over the world. He presented an in-depth review of low-volume ventilation used in clinical trials of lung

protective mechanical ventilation in ALI patients. The contradictory results so far were discussed, as well as the conclusion that low tidal volumes are beneficial for patients, not only lowering mortality and morbidity, but also reducing the extent of biotrauma. Trials with different levels of PEEP were also reviewed, with emphasis on the disappointing results and the possible explanations for them.

Dr Hagen Biermann also gave the final lecture, on the subject of Lung Recruitment with the Open Lung Tool. He outlined the mechanisms of

collapse, lung weight and height, gas adsorption, and the relationship between chest wall and abdominal pressures. The tool parameters were illustrated with graphical breath-to-breath observation of end inspiratory, PEEP and inspired tidal volumes. Changes in lung mechanics during a recruitment maneuver were illustrated with real-time monitoring, as well as graphic visualization of measured and calculated values. The function for analysis of opening and closing airway pressures was reviewed, and an example of a step-by-step recruitment maneuver was given. ■

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References

- 1) Bernard GR, Artigas A, Brigham KL, Carlet J, Falke K, Hudson L, Lamy M, Legall JR, Morris A, Spragg R. The American-European Consensus Conference on ARDS. *AJRCCM* 1994; 149:818-824.
- 2) Murray JF, Matthay MA, Luce JM, Flick MR. An expanded definition of the adult respiratory distress syndrome. *Am Rev Respir Dis* 1988; 138(3):720-723.
- 3) Arif SK, Verheij J, Groeneveld ABJ, Rajmakers PGHM. Hypoproteinemia as a marker of acute respiratory distress syndrome in critically ill patients with pulmonary edema. *Int Care Med* 2002; 28:310-317.
- 4) Groeneveld ABJ, Rajmakers PGHM, Jaap Teule GJ, Lambertus G. The 67Gallium pulmonary leak index in assessing the severity and course of the adult respiratory distress syndrome. *Crit Care Med* 1996; 24:9:1467-1472.
- 5) Gattinoni L, Carioni P, Pelosi P, Goodman LR. What has computed tomography taught us about the acute respiratory distress syndrome? *Am J Respir Crit Care Med* 2001; 164:1701-1711.
- 6) Groeneveld ABJ. Vascular pharmacology of acute lung injury and acute respiratory distress syndrome. *Vasc Pharmacol* 2003; 39:247-256.
- 7) Groeneveld ABJ, Plötz FB, van Genderingen HR. Monitoring the permeability edema of ventilator-associated lung injury. *Crit Care Med* 2005; 33(1):250-253.
- 8) Gajic O, Lee J, Doerr CH, Berios JC, Myers JL, Hubmayer RD. Ventilator-induced cell wounding and repair in the intact lung. *Am J Respir Crit Care Med* 2003; 167:1057-1063.
- 9) Dreyfuss D, Martin-Lefevre L, Saumon G. Hyperinflation-induced lung injury during alveolar flooding in rats. *Am J Respir Crit Care Med* 1999; 159:1752-1757.
- 10) Suh GY, Koh Y, Chung MP. Repeated derecruitments accentuate lung injury during mechanical ventilation. *Crit Care Med* 2002; 30:1848-1853.
- 11) Ranieri VM, Guinta F, Suter PM, Slutsky AS. Mechanical ventilation as a mediator of multisystem organ failure in acute respiratory distress syndrome. *JAMA* 2000; 284:43-44.
- 12) Mead J, Takishima T, Leith D. Stress distribution in lungs: a model of pulmonary elasticity. *J Appl Physiol* 1970; 28:596-608.
- 13) Salazar E, Knowles JH. An analysis of pressure-volume characteristics of the lungs. *J Appl Physiol* 1964; 19:97-104.
- 14) Markhorst DG, van Genderingen HR, van Vught AJ. Static pressure-volume curve characteristics are moderate estimators of optimal airway pressures in a mathematical model of (primary/pulmonary) acute respiratory distress syndrome. *Int Care Med* 2004; 30:2086-2093.
- 15) De Durante G, delTurco M, Rustichini L, Cosimini P, Glunza F, Hudson LD, Slutsky AS, Ranieri VM. ARDSNet lower tidal volume ventilatory strategy may generate intrinsic positive end-expiratory pressure in patients with acute respiratory distress syndrome. *Am J Respir Crit Care Med* 2002; 165:1271-1274.
- 16) Gattinoni L, Pelosi P, Suter PM, Pedoto A, Vercesi P, Lissoni A. Acute respiratory distress syndrome caused by pulmonary and extrapulmonary disease. Different syndromes? *Am J Respir Crit Care Med* 1998; 158(1):3-11.
- 17) Villar J. The use of positive end-expiratory pressure in the management of the acute respiratory distress syndrome. *Minerva Anestesiol* 2005; 71:265-272.
- 18) Marini J, Gattinoni L. Ventilatory management of acute respiratory distress syndrome: a consensus of two. *Crit Care Med* 2004; 32:No. 1.
- 19) Crotti S, Mascheroni D, Caironi P, Pelosi P, Ronzoni G, Mondino M, Marini JJ, Gattinoni L. Recruitment and derecruitment during acute respiratory failure: a clinical study. *Am J Respir Crit Care med* 2001; 164(1):131-140.
- 20) Grasso S, Mascia L, DelTurco M, Malacarne P, Giunta F, Brochard L, Slutsky AS, Ranieri MV. Effects of recruiting maneuvers in patients with acute respiratory distress syndrome ventilated with protected ventilatory strategy. *Anesthesiol* 2002; 96(4):795-802.
- 21) Lim SC, Adams AB, Simonson DA, Dries DJ, Broccard AF, Hotchkiss JR, Marini JJ. Intercomparison of recruitment maneuver efficacy in three models of acute lung injury. *Crit Care Med* 2004; 32(12):2371-2377.
- 22) ARDSNetwork. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. *N Engl J Med* 2000; 342(18):1301-1308.
- 23) Amato MBP, Barbas CSV, Medeiros DM, Magaldi RB, Schettino GP, Lorenzi-Filho G, Kariralla RA, Deheinzeln D, Munoz C, Oliveira R, Takagaki TY, Carlvalho CRR. Effect of a protective ventilation strategy on mortality in the acute respiratory distress syndrome. *N Engl J Med* 1998; 338(6):347-354.

In the physiology laboratory after a lavage procedure, workshop participants had the opportunity to initiate the recruitment maneuver in a stepwise manner: determining the critical opening and closing pressures while monitoring dynamic compliance and VT_{CO_2} levels. Re-recruitment was then initiated by setting EIP to deliver a tidal volume of 7 ml/kg, and ventilation was maintained by setting a PEEP of 2 cm/H₂O above the critical closing pressure.

Critical Care News got impressions of the course from two groups of participants.



Workshop participants DAH de Gast-Baulcher, PICU fellow; WJ Engelbrecht, anesthesiologist and fellow Intensive Care Medicine; and JE Steenhuisen, anesthesiology fellow

Why are you interested in lung recruitment?

WJ Engelbrecht, anesthesiologist and ICU fellow:

We are dealing with critically ill patients, from the OR to the ICU, so we are interested in lung-protective strategies. The subjects and the sessions today were very good.

Would you be able to do a recruitment maneuver on patients after the session today?

JE Steenhuisen, anesthesiology fellow:

Yes. In the past, we have mostly used PEEP titration and pressure gradients, or inspiratory hold. This concept and tool is quite new and I think there are advantages to it.

What about in the PICU, are you interested in applying the recruitment steps you learned today?

DAH de Gast-Bakker, pediatrician, PICU fellow:

We have been recruiting by means of inspiratory hold, depending on the patient and disease categories. Some children just don't have enough lung volume, so we have mixed problems. I think we should try the stepwise recruitment protocol and maneuvers we learned about today. We always need to think about being able to change what we are doing.

Workshop participants participating in lung recruitment maneuvers



What aspects of the workshop were of most value to you?

RJ Trof and TMD Nguyen, ICU fellows: The theoretical part was good. It is important to know the background to what we are applying in practice. It is also valuable to see how it works in practice.

We are already doing recruitment maneuvers with end tidal CO₂. We are starting to learn this in the unit, and are already a little familiar with it.

What is your opinion of the stepwise recruitment maneuver, in comparison to others?

EWJ Schubert, ICU fellow: The good thing about the stepwise procedure is that you get parameters to measure what you are doing, in contrast to other types of recruitment. Using the Open Lung Tool, you can see what you are achieving and get more patient information. This approach seems superior.

It may take more time in some patients, especially unstable cases where there might be hypotension. However, this procedure clearly has fewer disadvantages than other procedures. ■



ICU fellows RJ Trof, EWJ Schubert and TMD Nguyen gave feedback after the sessions



Professor Johan Groeneveld lectured on pathophysiology of ARDS

It's a scenario every ICU in the world needs to prepare for but dreads to face: the outbreak of a global infectious disease epidemic. The Prince of Wales Hospital ICU in Hong Kong had the media eyes of the world upon it as it struggled to treat patients, protect staff and learn and educate about the SARS virus. Critical Care News met Professor Charles Gomersall, who shared his experiences with SARS in the ICU, and outlined important precautions and actions for other ICU departments to keep in mind for the future.

Two years post SARS: the experience and the need for a state of preparedness



Professor Charles Gomersall, MD

Tell us about the first experience at your institution – the first patient who presented with symptoms, and how the staff handled it.

I got a call from the Professor of Medicine saying “we have an outbreak.” We had known for some time that something was going on in China. We didn't know what it was; we thought it might be avian flu. In retrospect, we are pretty glad that it was not! We were already quite wary about atypical pneumonias. The Professor of Medicine thought they might ventilate the patients non-invasively at first, and then send them down to us in the ICU. We were actually quite lucky in that we had a patient in the unit a few weeks earlier with multi-resistant TB, so respiratory protection had recently been a consideration. We had already decided that for seriously infectious disease, we were not going to ventilate patients non-invasively, because of the mask leaks, very high flows and therefore

greater chances of dispersing organisms.

It was really quite a frightening experience at the time because we were seeing a relatively high proportion of patients who were colleagues from the medical department, with a disease that seemed to be rapidly progressive. And as we were not sure what it was, we did not know how to treat it. It wasn't clear that the patients were not all going to die. At that time, given the size of the local hospital outbreak, it seemed really quite infectious. Patients were predominantly presenting with respiratory failure. They were admitted to the ICU slightly earlier than usual, partly because many of them were colleagues.

What happened in the ICU when subsequent SARS patients started to be admitted?

People started to feel that they were at significant risk of getting this disease. They

were scared. However, we had an idea of what to do early on: we had admitted a patient from a private hospital with an atypical pneumonia, who subsequently tested positive to SARS when we tested serum months later, although we didn't know at that time. We took similar precautions with the subsequent SARS patients as we did with her.

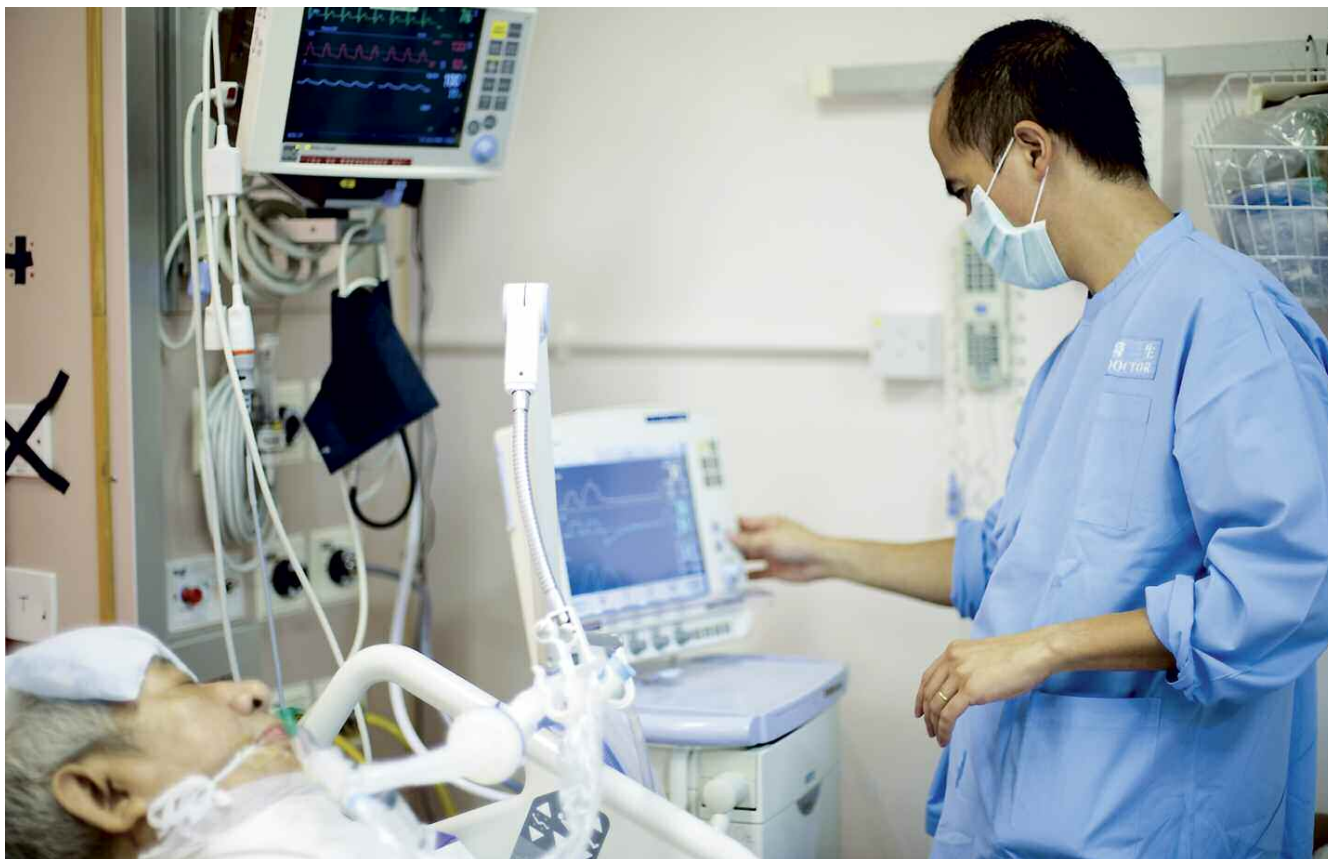
What was the clinical progression for the majority of patients after admission to ICU, in terms of ARDS or organ failure?

The typical progression was single organ disease. We experienced very minor other organ failure; what cardiovascular failure we saw was partly related to the policy of keeping the patients very dry. Because this was a predominantly respiratory problem, we tended to keep the patients very dry so that if they did develop a shunt they tended to be a little more anatomic than they would have been if we had fluid loaded them as we normally do. The patients who came to us were almost exclusively respiratory, about 80% ARDS.

Interestingly, not all of the SARS patients with ARDS needed ventilation. We found that there was a very high rate of air leaks, pneumothorax, even in the non-ventilated patients. So we were very reluctant to ventilate people unless we absolutely had to.

What percentage of patients were ventilated?

About 70% of the ARDS patients were on ventilation. It gave us a little bit of insight into separating the lung changes that occur as a result of ARDS from those that occur with mechanical ventilation. We have CT images of the lungs of patients in the sub-acute phase and these were similar for both ventilated and non-ventilated patients.



Dr Charles Gomersall with ARDS patient

The big difference from the normal ARDS patients was that the overt barotrauma was much more obvious. Overall in our ventilated patients we had 26% overt barotrauma rate. The Singaporeans had 23% and the Canadians had 34%. These are all very high percentages, much higher than typical ARDS patients, so it may have been due to the SARS disease process.

What were your early experiences in contacts with health authorities and other ICU physicians, in Hong Kong and in other nations that were experiencing the epidemic?

We put most of what little we knew about the disease on our website, and in reviewing the traffic, there were a lot of hits. Many people were viewing our infection control procedures. One of the great advantages of the Internet is that you can update it regularly, so we were probably updating those pages every two or three days as we were learning ourselves from the process. It also allowed us to update the outcomes, because we knew how many patients were being admitted to the hospital with SARS, and we knew how many patients

had died. It gave us some sort of idea of what the mortality was.

In regard to the national health authorities, the Hong Kong outbreak was pretty much centered here, so the situation was more us saying what we needed, than the authorities saying this is what we can help you with. In a sense, the authorities were grasping for solutions as well, as nobody initially knew the depth or how encompassing the situation was. It was very much a bottom-up approach, rather than top down. We said, "this is what we need, and this, this, this and this." There was considerable discontent among ground level staff, as we essentially had to not only deal with patients, but also work out what we needed and why. Other hospitals, which were perhaps less vocal, ended up with lower levels of protection than we did. Certainly in intensive care, our absolute volume is relatively small, so it is much easier to say, "we are having this – end of story."

It was quite chaotic in many ways, for example things like mask fitting. The N95 masks have a very variable fit; what will fit you may not fit me. So the function of the mask is entirely dependent on having a very good fit, and the mask fitting was set up by a member of the

department acting essentially on his own, and getting the funding. The initial machinery was funded by the department and not the hospital. There were a lot of examples like that, where an individual here would sort out a problem at ground level and that solution was passed on, rather than being passed down.

When you are busy coping with the patients, it can be difficult to deal with these sorts of issues as well. Another difficulty for us was that the authorities changed their strategic plan almost on a daily basis. At one point they were saying that we needed to set up an additional 40 ICU beds in a week or two. Our response was, "are you sure you want this, because it will cost a lot of money?" We called MAQUET, who were fantastic and told us that the next 40 ventilators to come out of the warehouse were ours. When we told the hospital, we were informed that they couldn't afford it. So two days later we were reducing the order because of the dynamics of the situation. But because of our relationship with the local MAQUET representatives, it was nice to know that the dialogue was working and that the capacity was there for us if we needed it. In the end, we opened up another eight beds in the ICU, with an additional eight ventilators

and monitors. Possibly even more important was the scavenging system. We had not routinely used scavenging before, so we did not have it on every ventilator. But that has now become our practice: every patient in the ICU has scavenging of respiratory gas.

Which type of ventilation strategy did you apply initially? Did you modify the ventilation treatment strategy as you treated more SARS patients?

Initially we mostly used a mixture. We used some Volume Control, some Pressure Control, some SIMV/Volume Control and Pressure Support. Then the volume of patients made it apparent that since we had to have more staff who were less familiar with intensive care, the sensible thing to do would be to standardize treatment with PRVC, allowing us to control both the pressures and the volumes. We had a standard PRVC algorithm, largely based on the ARDSnet criteria. From then on, we used PRVC for virtually everybody.

Which types of medical treatments did you apply to the patients, and was this modified by experience during the epidemic?

With regard to fluids, we tended to keep people on the dry side. When we subsequently analyzed our data on a univariate analysis, we found that one of the factors associated with survival

was being kept dry. Now this may be because those who developed bacterial superinfection needed more fluid, and of course those who developed bacterial superinfection did worse. So it may not be a causal relationship at all.

There were a number of treatments that were suggested on the basis of relatively little evidence. It was an interesting period where people were willing to try all sorts of things without there necessarily being a sound scientific basis for them. We used steroids for some, pentaglobulin and convalescent serum, but resisted the use of other treatments. We were also surprised by how many people rang us up to give us advice on the disease. We heard from a number of countries, urologists, general practitioners – it was quite extraordinary how many people were moved to give us advice on a disease none of them had ever seen!

How did it feel to have the eyes of the world upon you in the ICU?

The difficulty came when many of the treatment suggestions were suddenly announced in the press as working solutions. And the patients' relatives would come and say, "Well, why don't you try this?" It was a somewhat sad feature of SARS, in that there was a lot of communication of supposed medical information being spread through the newspapers, rather than through established methods. It is interesting that in the time since then, none of these treatments that

were reported in news conferences as being useful have been published as effective treatments in peer-reviewed journals. One would hope that next time we have an epidemic, this aspect can be avoided.

What is your institution's view on the involvement of the World Health Organization during and after the epidemic?

During the outbreak, it clearly had a role in disease control on a wider basis in a major pandemic, but at the local ICU level it had virtually none. Afterwards, the WHO set up a research group to look at a randomized controlled trial to be ready to run should there be another outbreak of SARS. That is not yet ready; two years after SARS we still don't have a randomized controlled trial. From an ICU point of view, it seems almost naïve to believe that we can run a randomized controlled trial. We still don't know enough about the epidemiology to ensure that the groups are balanced. In intensive care, we have been through this experience time and time again, trying to make the jump to the final step and get the answer in a randomized controlled trial, without doing the primary steps you need to do. My own feeling is that we will not be in a position to do a randomized controlled trial next time around, if there is a next time around.

The WHO does have some standard protocols. Other than that, I don't think there is the data to give any clear answers. In retrospect, one wishes that one had collected more data. But at that time, we were scrambling just to cope, and the data that we did collect was not what we would have wished.

How many SARS patients were treated at this institution, and what were their outcomes?

About 68 patients. They had a very long length of stay compared to our usual ICU patients; we did nothing else than look after SARS patients for three months. The average median ICU length of stay was about eight days compared to our normal length of stay of about three days. Mortality was about 25%. Approximately 85% of patients met the criteria for ARDS and about 50% of patients required mechanical ventilation.

What was the profile for prognosis in cases with the worst outcomes?

There was certainly nothing that would allow you to reliably predict outcome, apart from the fact that older patients did worse. In the data from



Dr Gomersall and ICU staff members. A unique team spirit supported the challenges the staff faced during the outbreak



Dr Charles Gomersall adjusting settings for a patient who will soon be released from the ICU

the physicians' analysis, old age as a cut-off was 45. Since I was 43 at that time, and the director of the ICU was 42, we were a little bit worried!

What was comforting, particularly to all of us with families, was that children did incredibly well during this epidemic. Few children needed to go to the ICU at all, and none of the children died. SARS was a relatively mild disease in children – in contrast to avian flu, which is very severe in young people.

What types of infection control routines and protocols did your ICU establish?

Basically we took precautions for airborne disease. It is still not entirely clear how SARS is spread. It seems to be predominantly droplet spread, but it has been suggested that it may also be airborne. In the ICU we had a segregated entrance and exit, we wore an N95 mask, a face-shield, hat, gloves, a waterproof gown to the ankles, and shoe covers. And the staff spent the entire day like that. Putting on all of the kit in the right order probably took about 5-10 minutes. There would be someone there to check we did it properly, because it was incredibly easy to miss a step, get the order wrong, find the hat wasn't covering properly, etc. Of course you had to make sure not to touch your face, and we developed a culture within the ICU where everyone looked after each other.

The exhaust gas from all ventilated patients was scavenged. We stopped using hot water

humidifiers; we only used HMEFs with high-efficiency viral bacterial filters, both at the Y-piece and before the expiratory cassette. We also put a viral filter between the mask and the valve of the self-inflating resuscitators. And we always made sure that the patient was paralyzed before intubation to prevent coughing. When we did intubate, we added a hood to cover everything except our eyes. The intubation was quite difficult; because of the high pneumothorax rate, we tended to intubate patients quite late. They would be getting close to 100% oxygen. We developed a special tight-fitting mask with an oxygen supply, which meant that when we intubated them, if they had already desaturated they were desaturating on 100% oxygen. And while you were waiting for the relaxant to work, they desaturated even further. That was scary. You knew that the saturation would continue to fall, but since you had the hood on, you could not auscultate their chests. It was very difficult to ascertain whether the tube was in the right place.

Most of us worked on the principle that the most senior person available should intubate, as intubation was perceived as a very high-risk procedure in these patients. It was very important from a morale point of view that the senior people were in there, getting exposed as much as everybody else. In fact, morale was extraordinarily good in the ICU, maybe even better than at any other time. We ascribe this partly to the fact that the consultants were in there all day, and

were involved in the high-risk procedures. It was also important from a learning point of view. No matter how many times you think through a procedure, it is not until you do it that you can realize that there may be a flaw. By doing it, you can make things safer and better. It was quite apparent to the staff that we were always looking for ways to make things safer, for them as well as for us. On an ordinary day, I was physically present in the ICU from eight in the morning until eight in the evening. I tended not to eat or drink in the hospital during that time period. So it would be a twelve-hour day without food or fluids. At the end of the day, the high points would be removing your N95 mask, since it is incredibly uncomfortable, and taking a drink.

Often we did two weeks continuous call in the ICU, and then 10 days doing research and administration, and teaching material for SARS. But during those 10 days I would not be seeing any patients, and 10 days is about the incubation period for SARS. After those 10 days, I would go home for four days, and then start the cycle again.

How was the staff affected by the epidemic? Did any members acquire the disease?

Five ICU staff contracted the disease and one had to be admitted to the ICU. Four of the five were members of the permanent ICU staff, and one volunteered to work in the ICU during the outbreak.

One of the striking things about the whole epidemic was that people who could easily have avoided the exposure volunteered to come and work in high-risk areas. Staff from endoscopy, who could have had three months off, came and volunteered to work in our ICU. Staff from other hospitals volunteered here as well. There really was an overwhelming response from people who really had no obligation to work with us. This was one of the factors that made SARS much easier, because on those days where you were tired of the whole thing, you only needed to look at others who were coming in and did not need to be there. It was very inspiring. We have about 100 permanent staff in the ICU, and only one did not turn up to work in the ICU. She had previously contracted TB in the ICU, and had two children at home with respiratory problems, so it was quite understandable. All of our other ICU staff worked through the epidemic. This is in contrast to some other places we heard of.

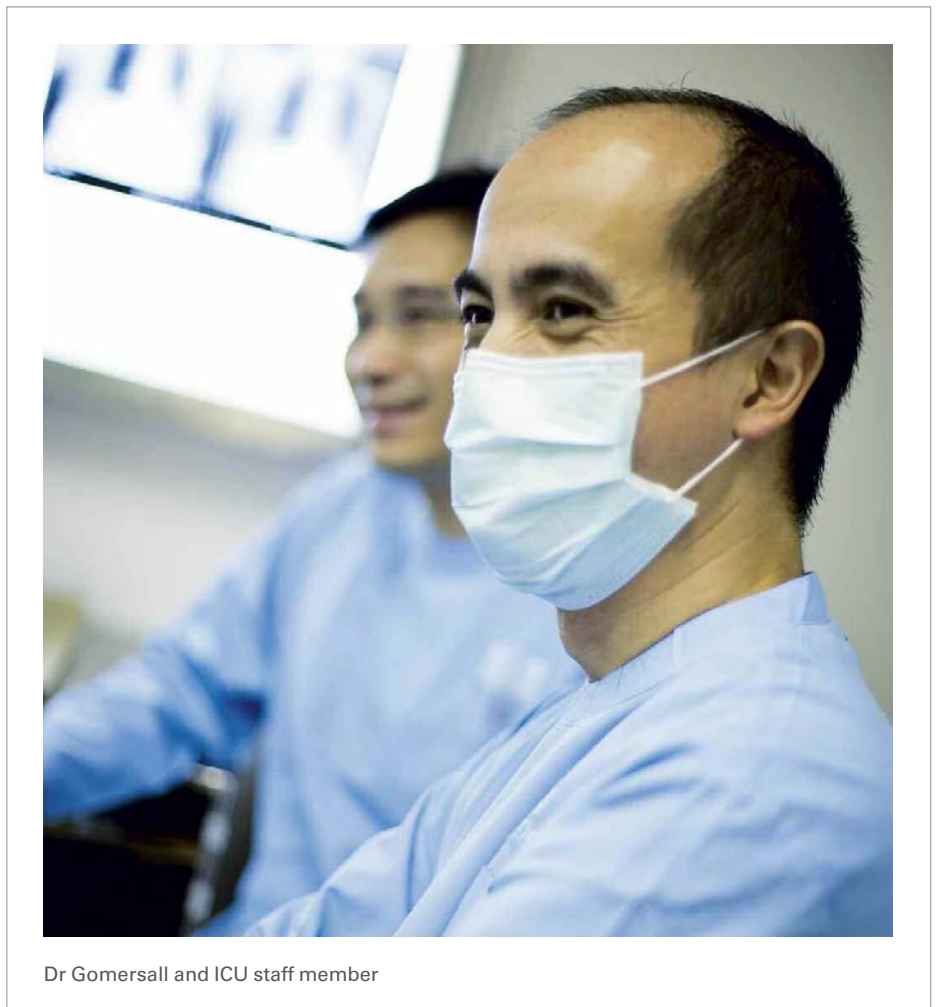
The community was incredibly supportive as well. We have heard of some other places where the medical community was shunned, but our experience was quite the opposite. I had people come up to me in the street and thank me for what I was doing.

What types of weaning strategies did you apply to the SARS patients?

We predominantly used Pressure Support. We found it quite difficult with Volume Support – the patients had very variable effort and could take a huge breath, then a small one, and it wasn't tracking closely enough. Pressure Support was used partly because of the infection risks of using an ordinary t-piece with high flow; we were concerned this would result in suspended particles that would travel further. We devised a variant of a system that has been described before, using a heat and moisture exchange filter – putting oxygen through the gas sampling port and attaching a t-piece to the distal end of the HMEF. But work of breathing through that is relatively high. In the end, we used that as a trial prior to extubation, rather than the weaning method. We were fairly clear early on that we would need some sort of filter in the circuit, but much of the process was developed through trial and error.

When did you notice that the epidemic seemed to be tapering off?

The outbreak started on March 13, 2003. By mid-May, it was clearly tapering off. We admitted our last patient in June 2003.



Dr Gomersall and ICU staff member

How did your institutional cooperation with national and international health authorities develop in the aftermath of the epidemic? Can you outline the state of preparedness that ICU staff should maintain in the event of a similar outbreak?

We have actually recently had a joint working group with the Singaporeans, to prepare a document on how to expand an ICU in an epidemic, and what preparations to make. It became clear from our discussions that staffing was the main issue. Contrary to a lot of what is written about the need to purchase equipment, equipment is almost certainly not going to be the limiting factor. From a staffing point of view, you cannot afford to dilute your nursing expertise too much and that means you need to keep a balance of permanent staff and temporary staff. Also you must calculate that a significant proportion of your staff may be ill. There is no point trying to prepare for more patients than you will have the staff to take care of. Then you have to prepare for staff training. An epidemic and expanded staff requires preparation for training beforehand, for both existing and

additional staff. You need to identify people who might best work in your ICU during an epidemic and train them, so you can call on them in a hurry. There is no point in rostering someone to work in the ICU who is not up for the job. All you are going to do is to expose them without any benefit to the patients. The same thing is true for high-risk procedures in this scenario. If you ask a junior staff member to do something and they fail and you end up doing it yourself, you have exposed two individuals to risk.

What is the most important advice you can give to other ICU departments around the world to prepare for an epidemic of this nature?

To choose the staff you need selectively, and to keep track of the staff who have worked in your ICU in the past. Certainly on the nursing side, there are individuals who might have worked in your unit in the past, know the layout and routines, and are probably the quickest people to retrain. But if you don't have a list, you can't contact them. Staffing is the major concern for preparedness, with much emphasis on infection

control. If you want to stop infection, you need to have a tight routine and control from the very start of an outbreak. Procedures need to be in place so that they can be rolled out, as there is no time to think up or establish routines once an epidemic has started. A formal mask fitting takes about 15 minutes, and that is dependent upon the first mask you try fitting perfectly. Every mask after that takes 15 minutes. If you average two masks per individual, that makes 30 minutes per staff member. These types of issues need to be sorted out in advance. You must also require that people wear the masks from the outset. They are incredibly uncomfortable, and initially there are a lot of complaints, but it is vital that you enforce compliance. Cases from the Canadian experience may illustrate this, where they had variable use of personal protection equipment, and large numbers of staff became infected. You have to be meticulous in requiring the use of masks for all staff members.

We talked about the higher vulnerability of children to Avian flu. Are you monitoring the current situation in other Asian countries with some concern?

We have had avian flu previously in Hong Kong, and it is a more frightening disease as it appears

to be much more complex; it is a multi-organ disease and it affects a different age group. It is frightening for us who don't do a lot of pediatrics. The pediatric ICU in this hospital has only four beds, which means it would be overwhelmed with work and the adult ICU would have to take up the overflow. But in the adult ICU, we don't have the equipment or recent pediatric clinical experience. In terms of ventilators we have the SERVO-i Universal, which will take care of pediatric patients, but we don't have the tubing, disposables, central lines and other items needed for pediatrics. We have discussed this with the PICU at the hospital, and they are in fact stocking the equipment needed, to prepare for any eventual overflow to the adult ICU. In terms of preparing for avian flu, the adult ICUs need to

consider the fact that they may be treating a different patient category, and need to prepare for this. The other worrying thing is obviously the aspect of multi-organ failure. More renal replacement therapy would certainly be needed, and there are the difficulties of renal replacement therapy in children. That would need to be part of any preparedness training program; you have to think about the need of training adult intensivists to look after children, which is quite a challenge.

We have two training packages, a general one for the ICU which is available on our website, and one in process that is geared to ICU disaster training. ICU staff members from other departments who would like more information on these packages should visit www.aic.cuhk.edu.hk/web8/BASIC.htm. ■

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References

- 1) Gomersall CD, Joynt GM, Lam P, Li T, Yap F, Lam D, Buckley TA, Sung JY, Hui DS, Antonio GE, Ahuja AT, Leung P. Short-term outcome of critically ill patients with severe acute respiratory syndrome. *Intensive Care Med* 2004; 30:381-387.
- 2) Derrick JL, Gomersall CD. Surgical helmets and SARS infection. *CDC online* 2004; 10:2. www.cdc.gov/incidod/EID/vol10no2/03-0764.htm.
- 3) Lee N, Hui D, Wu A, Chan P, Cameron P, Joynt GM, Ahuja A, Yung MY, Leung CB, To KF, Lui SF, Szeto CC, Chung S, Sung JY. A major outbreak of severe acute respiratory syndrome in Hong Kong. *N Eng J Med*. 2003; 348:1986-1994.
- 4) Tsang KW, Ho PL, Gaik CO, Wilson KY, Wang T, Chan-Yeung M, Lam WK, Seto WH, Yam LY, Cheung TM, Wong PC, Lam B, Ip MS, Chan J, Yuen KY, Lai KN. A cluster of cases of severe acute respiratory syndrome in Hong Kong. *N Eng J Med*. 2003; 10.1056/NEJMoa030666.
- 5) Drazen JM. Case clusters of the severe acute respiratory syndrome. *N Eng J Med* 2003; 348:6-7.
- 6) Poutanen SM, Low DE, Henry B, Finkelstein S, Rose D, Green K, Tellier R, Draker R, Adachi D, Ayers M, Chan AK, Skowronski DM, Salit I, Simor AE, Slutsky AS, Doyle PW, Kraiden M, Petric M, Brunham RC, McGeer AJ. Identification of severe acute respiratory syndrome in Canada. *N Eng J Med* 2003; 348:1995-2005.
- 7) Drosten C, Günther S, Preiser W, van der Werf S, Brodt H-R, Becker S, Rabenau H, Panning M, Kolesnikova L, Fouchier RAM, Berger A, Burguière A-M, Cinatl J, Eickmann M, Escriou N, Grywna K, Kramme S, Manuguerra J-C, Müller S, Rickerts V, Stürmer M, Vieth S, Klenk H-D, Osterhaus ADME, Schmitz H, Doerr HW. Identification of a novel coronavirus in patients with severe acute respiratory syndrome. *N Eng J Med* 2003; 348:1967-1976.
- 8) Ksiazek TG, Erdman D, Goldsmith CS, Zaki SR, Peret T, Emery S, Tong S, Urbani C, Comer JA, Lim W, Rollin PE, Dowell SF, Ling A-E, Humphrey CD, Shieh W-J, Guarner J, Paddock CD, Rota P, Fields B, DeRisi J, Yang J-Y, Cox N, Hughes JM, LeDuc JW, Bellini WJ, Anderson LJ. A novel coronavirus associated with severe acute respiratory syndrome. *N Eng J Med*. 2003; 348:1953-1966.
- 9) Tsang KW, Lam WK. Management of severe acute respiratory syndrome: the Hong Kong University experience. *Am J Respir Crit Care Med* 2003; 168:417-424.
- 10) Fowler RA, Lapinsky SE, Hallett D, Detsky AS, Sibbald WJ, Slutsky AS, Stewart TE. Critically ill patients with severe acute respiratory syndrome. *JAMA* 2003; 290:367-373.
- 11) Lew TWK, Kwek T-K, Tai D, Earnest A, Loo S, Singh K, Kwan KM, Chan Y, Yim CF, Beck SL, Kor AC, Yap WS, Chelliah YR, Lai YC, Goh S-K. Acute respiratory distress syndrome in critically ill patients with severe acute respiratory syndrome. *JAMA* 2003; 290:374-380.

Biography

Dr Charles Gomersall is Associate Professor of the Department of Anaesthesia and Intensive Care at the Chinese University of Hong Kong. He received his undergraduate training at the Westminster Medical School, University of London. His postgraduate training has taken place in internal medicine at St. George's Hospital, London; in anaesthesia at St. Mary's Hospital in London, and in intensive care at the Prince of Wales Hospital in Hong Kong.

Charles Gomersall has conducted scientific research in special interest areas, which currently include triage, antibiotic pharmacokinetics and probiotics. He is currently Editor of ICU Web. This website for ICU healthcare professionals receives approximately 65000 visits per month. Charles Gomersall has also been initiator, editor and major author of BASIC (Basic Assessment and Support in Intensive Care), and the Very BASIC and Not So BASIC courses. He is also presently heavily involved in the CoBaTRICE project.

It's a situation that CICU staff around the world are familiar with: an aging population with more complex coronary disease requiring advanced and comprehensive surgeries. And many of these departments are under pressure to economize; on staffing, equipment, and treatment time. Reducing the time on ventilator for cardiac surgery patients is of great interest from economic and work volume perspectives.

The Cardiovascular Intensive Care Unit (CVICU) at Lund University Hospital Heart and Lung Center in Sweden has met the challenge of implementing new procedures to wean cardiac patients more quickly, while maintaining safety and ensuring that the quality of care is not compromised. Critical Care News met Lars Algotsson, MD, PhD, and registered nurse Göran Claesson of the Cardiovascular ICU to discuss their experiences with the new procedures for weaning.

New weaning procedures for post-op cardiac patients



Göran Claesson, RN and staff member extubating a patient who underwent PCI with PTCA and stent to distal LAD. He suffered cardiogenic shock and was resuscitated and treated with IABP and ventilator in the CICU for 20 hours until extubation

How did you come to develop and implement your new procedures for weaning post-op cardiac surgery patients?

Dr Algotsson: We were upgrading the ventilator fleet in the department. The classic SERVO 900C was in part developed in cooperation with physicians here in Lund. The CVICU and other departments in the hospital chose SERVO-i about a year ago, partly because of this cooperation and earlier experiences. We had the opportunity to try non-invasive ventilation here in the CICU, and Göran Claesson was educating other staff in the department. They accepted the new ventilator extremely quickly. It was a simple machine to adapt to. We were planning to test non-invasive ventilation in patients with pulmonary edema; eight patients in two weeks. But that resulted in 28 patients in three weeks. Everyone thought the protocol was good, simple and user friendly.

We found that patients who had pulmonary edema and were difficult to oxygenate were successfully treated with non-invasive ventilation.

Based on these positive experiences, we decided to implement Automode in the weaning process for post-operative patients, and to take the opportunity to initiate the current weaning study, where we learned much more about the system in clinical situations in post-operative patients.

What methods for weaning did you previously follow?

Dr Algotsson: Previously we worked with pressure supportive modes and the ASB



Göran Claesson, registered nurse, CICU

function for weaning. That was a routine that worked well and provided cost efficiencies with shorter times on ventilator; in 1999 we had 11 hours post-operative ventilatory therapy after heart surgeries, and that was reduced to 6 hours in 2003.

Can you describe the new weaning procedures?

Göran Claesson: Our patients have high risk factors. But we have an objective that the average patient is weaned from the ventilator 4-6 hours post-op. We have been collecting statistics in our weaning times for many years.

Dr Algotsson: We implemented a program with PRVC and Automode using SERVO-i ventilators. This means we can compare our experiences of pressure-supported modes in weaning with PRVC and Automode, and Volume Support. Our current experience is this: patients with heart failure and pulmonary edema are not always suitable to ventilate with pressure-supportive modes. In these types of patients, the tidal

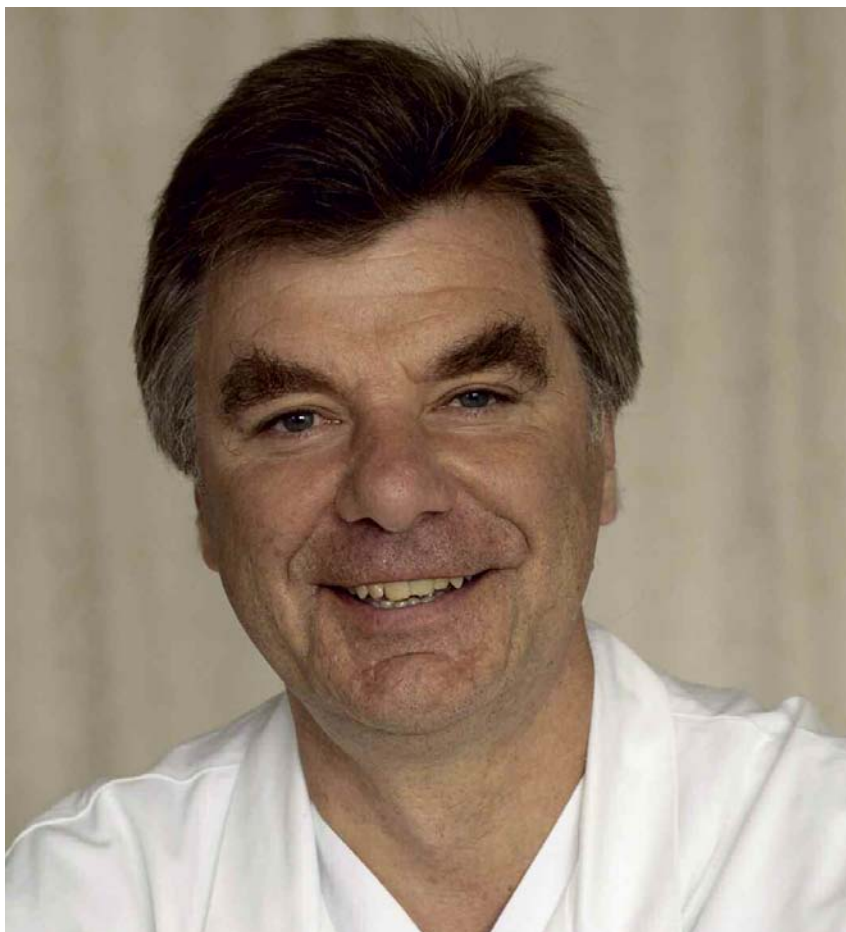
volumes decrease over time if the heart failure progresses and the pulmonary edema increases. It was a pleasure to see how Volume Support and PRVC (Pressure Regulated Volume Control) worked, and even more pleasing to have them connected to Automode, which automatically adapts to the patient's changing clinical situation. It is an excellent ventilation therapy for surgical patients in the CVICU department.

When did you start the current study?

Göran Claesson: The current study was started in March this year. We were going to conduct the study on 40 patients, which has just been completed, with excellent results so far, and the study has now been extended.

What types of patient categories are coming from surgery?

Dr Algotsson: The majority are bypasses, but a great deal of valve replacements as well. We even have a steady stream of patients coming in after PCI in the cathlab. We have 16 beds in the



Professor Lars Algotsson, MD



The Lund CVICU staff have been tracking modifications in their weaning protocols and collecting outcome data for many years

CICU now, and have processed about 2, 700 patients annually. Of these, 1, 400 are cardiac surgeries, 250 non-surgical, 500 are pulmonary surgeries, and we also previously had about 700 pacemaker patients included in this number. Regarding PCI patients, the majority have multi-vessel disease and very distal stenoses. Our biggest problem is that we have a long waiting list, which never seems to recede. We have a lot of patients with heart failure and several assist-device programs with different pump systems. We have about 20-25 heart or lung transplants per year, so we meet a variety of complicated cases. Our post-operative patients usually stay about a day, and we have no step-down department; patients usually go directly to the medical ward. The majority of patients leave us after 12-18 hours.

So it's also important, from a work volume perspective, to get patients off the ventilators as quickly as possible?

Dr Algotsson: Absolutely. We encourage spontaneous breathing in the ventilator as early as possible, to remove sedation. We have made a computerized model of a program to docu-

ment our process. We have analyzed records of patient outcomes from a five-year test period from 1999 to 2004 and a control period from 1996 to 2000, and compared with when we implemented the new treatment routines with spontaneous breathing in the ventilator as a part of the new routines to extubate earlier and obtain hemodynamic stability. We calculate that we have saved 240 patients from an ICU stay of more than 48 hours – patients who would perhaps have been here for three, four or five days. From this perspective, we have saved a substantial amount of money for this institution.

What criteria do you use to select cardiac patients for weaning according to the new procedure?

Göran Claesson: We have modified the process when implementing the Automode procedure: patients must be hemodynamically stable and have appropriate body temperature. They must have successful surgical outcomes and have no bleeding. They should be awake and able to communicate with us, and be able to move their arms, legs and head. We use very light sedation. When we begin extubation, they

have already been spontaneously breathing with the ventilator for quite some time.

One of the interesting aspects of Automode is that patients appreciate that the ventilator has adapted to their needs, and that they can talk; they are awake and alert, and quite anxious to have the tube removed.

How many patients have you treated using this new procedure?

Dr Algotsson: We are now using this procedure in almost all of our patients. We can see that it is much easier on the patients, and it has become quickly established within our organization. When we move to new facilities this autumn, it will be implemented as the standard treatment protocol for most cases.

I was a little worried initially that it would be difficult to implement the new process. Generally, it takes a long time for staff to adapt to new working routines. Ten years ago, every patient here was lying with a y-piece, and spontaneously breathing. We had to educate that the new procedure meant that the patients would be spontaneously breathing with the ventilator, to maintain PEEP and avoid atelectasis,

and then be extubated. When we implemented the Automode protocol, I was a little concerned that we would not maintain this concept, which I felt was a winner. But it has gone beyond our expectations, and I see the patients in control of their own breathing patterns. Since the ventilator delivers a volume, it is just for the patients to determine their own pace.

What impact has the new weaning procedure had on staff?

Göran Claesson: From a staff perspective, we found that Automode is easy and simplifies the care process; it has reduced the need for staff interventions with the ventilator, since it automatically responds to the patient situation and need. There are also fewer alarms in this process. This means we have more time to focus on the patient. I have worked at the hospital as an anesthesia nurse since 1983, and have been in the CICU for five years, so I have been able to follow the processes and

protocols, from the old to the new.

This was an entirely new concept for us when we started implementing Automode in weaning this year. We were a bit concerned in the beginning as to whether the ventilator would respond appropriately to patient need. But we were quickly impressed by how well it works. And especially after these comprehensive surgeries, it is nice to see how positively and quickly the patients respond with this new procedure. We generally see them sitting up in bed reading the newspaper the day after surgery, which is fantastic for us. We can note that there have been developments in anesthesia that also contribute to this situation. They are using fewer morphine-based preparations, and more short-term anesthesia drugs.

After training, the entire staff has adapted quickly to this new procedure. Twice a year we report statistics on how we are doing with ventilator hours, and so forth. Our objectives are well known to everyone working here, and we all want to achieve these objectives. ■

Biography

Dr Lars Algotsson, MD, PhD, began working at Lund University Hospital in 1983, and has been Director of the Cardiovascular ICU since 1994.

He has special interests in cardiovascular hemodynamics, heart failure treatment and transplantation, and has refereed for a number of medical journals.

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References

- 1) Carlucci A, Delmastro M, Rubini F, Fracchia C, Nava S. Changes in the practice of non-invasive ventilation in treating COPD patients over 8 years. *Intensive Care Med* 2003; 3: 419-425.
- 2) Brochard L, Mancebo J, Wysocki M. Noninvasive ventilation for acute exacerbations of chronic obstructive pulmonary disease. *N Engl J Med* 1995; 333: 817-822.
- 3) Nilsson J, Algotsson L, Höglund P, Lührs C, Brandt J. EuroSCORE predicts intensive care unit stay and costs of open heart surgery. *Ann Thorac Surg* 2004; 78:1528-1534.
- 4) Nilsson J, Algotsson L, Höglund P, Lührs C, Brandt J. Early mortality in coronary bypass surgery: the EuroSCORE versus The Society of Thoracic Surgeons risk algorithm. *Ann Thorac Surg* 2004; 77: 1235-1239.
- 5) Gold J. Invited commentary of Early mortality in coronary bypass surgery: the EuroSCORE versus The Society of Thoracic Surgeons risk algorithm. *Ann Thorac Surg* 2004; 77:1239-1240.
- 6) Stohr IM, Albes JM, Franke U, Wippermann J, Cohnert TU, Huttemann E, Wahlers T. Outcome of patients after cardiac surgery transferred to other hospitals following prolonged intensive care stay. *Thorac Cardiovasc Surg* 2002; 50 (6): 329-332.
- 7) Cimochoowski GE, Harostock MD, Foldes PJ. Minimal operative mortality in patients undergoing coronary artery bypass with significant left ventricular dysfunction by maximization of metabolic and mechanical support. *J Thorac Cardiovasc Surg* 1997; 113(4): 655-664.



Dr Lars Algotsson and Göran Claesson with cardiogenic shock patient

Many metropolitan area hospitals are facing difficulties in densely populated urban areas: how to transfer intensive care patients to centers where there are open ICU beds or adequate staffing this week, or specialized centers for advanced treatment care needs. In addition to these logistics, transferring the ventilated patient from one ICU to another poses additional therapeutic challenges. The University Hospital System in Munich has defined a systematic approach to ICU patient treatment and transport.

Ventilation procedures for intensive care air transports



Assistant Professor Dr Gerhard Kuhnle, Anesthesia Director of Intensive Care Transports, University of Munich hospital system

How many intensive care patients are transported here on an annual basis?

In 2004 we had 1,900 patients by land and 860 by air transport, or a total of 2,760 patients. This represents primarily the area of Bavaria and Baden Wurttemberg, where we are receiving patients, but sometimes the patients can be transported from here, to Hamburg and Berlin.

What are the proportions of emergency and planned air transports of intensive care patients?

Of total patients in air transports, about two-thirds are intensive care patients and about one-third are from acute emergency situations. Of the intensive care patients, approximately 50% are emergency and 50% are planned transports.

Most of these patients are coming from smaller hospitals in Bavaria and some are coming from central hospitals in Munich, smaller hospitals without special care units, or without cardiac or neurosurgery, for example.

Are these transports generally during daylight hours, or are they day/night transports?

Our helicopter and ambulance services run day and night. But at night, the helicopter primarily does emergency cases.

What is the average transport time for a ventilated patient by helicopter?

It depends entirely on the patient situation; we have flight times of a few minutes up to several hours. On one occasion last year, we took a ventilated patient from Marseille to Munich, so it can vary. But in general, for the majority of intensive care patients, transport time is up to one hour in the air.



Medic Dirk Baumann and Dr Gerhard Kuhle prior to transport

Which types of intensive care patient categories do you primarily transport by planned air transport?

There are a wide variety of intensive care cases: ARDS, infants, trauma, neurological cases and cardiac patients, heart failure, or pericardial infusion and coronary syndromes, coming from a peripheral hospital for specialized surgery.

The infants are often premature, but there are also many full-term infants coming here for specialized surgery for congenital defects.

In these types of patient transports, what type of clinical performance is required from the ventilator?

It depends on the patient of course. Cardiac and neurological patients are often sedated and on controlled ventilation. In these patients it is pretty straightforward, since they have normal lungs, and normal resistance and compliance. But in the ARDS patients, we need a good intensive care ventilator, delivering pressure

supported and pressure controlled therapy, or in very severe cases extracorporeal membrane oxygenation (ECMO), also during the transport. Pressure controlled ventilation is also sometimes needed for infants.

Is there a profile or any type of clinical criteria intensive care patients must fulfill to be transported by air?

The indication for air transport is dependent upon the patient. A patient with ARDS with unstable respiratory mechanics is stabilized on the ward of the remitting hospital. Other patients may be unstable and need hemofiltration, and then we try to transport them as urgently as possible. Each underlying disease or condition is different. We have no exclusion criteria, but I would not transport patients with acute bleeding, for example, or a ruptured aortic aneurysm. They require extensive blood transfusions, and that can be difficult to manage in the air. In our experience, almost every other patient, depending on their

situation, may be transported by air or land. It is difficult to intubate or put in IV lines or a CVC during transport, but we have done this when necessary. If these types of interventions are needed, often we usually want to make sure that they are already in place prior to transport.

What are the types of ventilation strategies or modes commonly used during transport?

We have all modes of ventilation, protocols coming with high PEEP and high peak pressure, sedated patients, some patients with non-invasive ventilation by mask, for example with cystic fibrosis or lung fibrosis, coming to the transplant center here.

What is the normal range of trigger settings used during transport?

We try to determine what is good for that particular patient: for a patient on supported ventilation, we use flow triggering, which is best in my experience. But controlled ventilation is

used for sedated patients, who are in the majority. Primarily, we try to maintain the same ventilation strategy initiated by the remitting hospital, continuing the same strategy during air transport until the patient reaches our center here. However, there are cases where we try to adapt a good ventilation strategy during the transport. If the patient is not being optimally ventilated when we receive him, we titrate the settings and adjust to our own ventilation strategies enroute to this hospital.

What are some of the challenges in treating these ventilation patients in air transports?

The main challenge is the transport situation itself: you have a lot of equipment – several IV lines where you want to avoid disturbance, the ventilation circuit and tubes. The most important thing is to avoid leakage or disconnection in the ventilatory circle. In ARDS or ALI patients, the challenge is to improve patient oxygenation and

recruiting lung area, or improving the ventilatory strategies of the remitting hospital.

What are the contrasts in ventilation treatment during air transport with an intensive care ventilator compared to traditional transport ventilators?

It is generally the same contrast that you have in the hospital, coming from the OR or in the ICU. Generally, uncomplicated and sedated patients do not require advanced ventilatory therapy. But if you have a patient with respiratory failure, whether in the ICU or in the air, you need a good intensive care ventilator. But since you never know what the next patient's condition will be, it is better to have both solutions – the intensive care ventilator for complex cases, and the transport ventilator for general cases. I think that only about 50% of our total transport patients require a sophisticated intensive care ventilator, but in those patients it is really a necessity. If we

did not have it, we could not transport these patients. In air transports, we frequently use the intensive care ventilator even in uncomplicated patients. The unit is already there and can simply be switched on. About 30% of our ventilated patients get hand ventilation from the remitting hospital to the ambulance or helicopter, where they are put on the intensive care ventilator and treated with supportive modes.

What types of preparations are needed for planned air transport of an intensive care patient; at the remitting hospital, and at Grosshadern where the patient is received?

It always depends on the underlying disease and the condition of the patient. Uncomplicated patients usually require no further preparation. Critically ill patients, on the other hand, with heart failure, respiratory failure or sepsis for example, often require hemodynamic stabilization, e.g. catecholamines, improved



Dr Gerhard Kuhnle with intensive care transport patient and ventilator

ventilator strategies, nebulization of illoprost or NO ventilation, and sometimes insertion of central venous catheters or arterial catheters. The most demanding patients are the ARDS patients, since they are frequently in a critical and unstable condition when we receive them.

What are the average transport times by land?

We have a lot of transports from one Munich hospital to the other, for specialized types of surgeries. These transports are usually 30 minutes by land, and from ICU to ICU about 90 minutes. For hospitals in Bavaria outside of Munich, there can be a wider range of times. There can be extreme cases too: we had an intensive care patient transport to Bonn last year by land, when the weather did not permit air transport.

How big is the medical team that accompanies each intensive care patient by air?

In the majority of cases, the medical team consists of one doctor and one medic per patient. In some cases we might have three members in the medical crew. But it is a calculation of the crew weight and the total weight with fuel that determines how many medical team members the helicopter pilot will permit.

How do ventilated patients who are not sedated generally experience the air transport process?

I usually give a light sedation, but most of the patients are not nervous or fearful of the flight. They are generally comfortable, and they have headphones so they can talk with the medical staff members.

Can you give us an example of a "worst case scenario" for a ventilated patient during helicopter transport?

The worst case scenario is losing the airway in a patient who could not be intubated conventionally, for example a dysmorphic newborn, or discovering empty oxygen tanks and a patient with respiratory failure in need of an FiO_2 of 1.0.

What is the history behind the intensive care transport culture here at this institution?

The first helicopter came in 1991, so we have a lot of experience with ARDS, transplant and emergency patients in air transports. Most strategies coming were the same for years, but



Helicopter interior and ICU patient

some new ones from the ICU are also used in transport as well, such as; nitric oxide, non-invasive ventilation, lung recruitment and illoprost nebulization. Treatment strategies that are developing in the ICU are adapted for treating ICU patients in air transports.

We had fewer emergency patients in 1991, and more planned transports at that time. But in recent years there has been a trend in Germany moving towards more emergency air transports. There are also more helicopters available now than there were ten years ago. Accidents outside the cities, or accidents at night, call for helicopter assistance.

What do you think will be the future trends in regard to transport of intensive care patients? Will there be increasing numbers, requirements or special demands in future?

The trend we have seen in recent years is one I think will continue. There are more and more critically ill patients being remitted by smaller hospitals to the large research centers. There are also more problems with intensive care department capacities that we are seeing more frequently, due to lack of beds or staff. Therefore the need for inter-hospital transfer with intensive care facilities will increase. In addition, the requirements for the quality of air transport will also increase, as well as the need

for continuing therapy during air transport.

Weight is the major problem for air transport, so every kilogram is important. Lighter weight and more advanced equipment to be used for more sophisticated ICU therapies is needed in future development to meet these future directions and trends. ■

Biography

Dr Gerhard Kuhnle is Assistant Professor of Anesthesiology and Intensive Care at the University of Munich. He conducted his medical studies at the Universities of Tuebingen and Munich during the years of 1981 and 1988, and conducted research during the years of 1988 and 1993 at the Department of Surgical Research, University of Munich. He was named Professor and has been employed at the Department of Anesthesiology, University of Munich, since 1994, and is currently a Director of Inter-Hospital Intensive Care Transport at the University of Munich hospital system. His special interests are in the areas of critical care medicine (ARDS, ventilation, inter-hospital transport) and anesthesia (surgery, neurosurgery, gynecology and obstetrics, ENT surgery, pediatric anesthesia, regional anesthesia), as well as pain therapy.

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