

Critical Care News

Lung Protection Symposium and Workshop Summary with focus on – Lung Recruitment and Neurally Adjusted Ventilatory Assist - NAVA

CRITICAL CARE NEWS is published by MAQUET Critical Care.
Maquet Critical Care AB
171 54 Solna, Sweden
Phone: +46 (0)8 730 73 00
www.maquet.com
©Maquet Critical Care 2009. All rights reserved.
Publisher: Fredrik Wetterhall
Editor-in-chief: Kris Rydholm Överby
Contributing editor: Judith Marichalar-Sundholm
Order No. MX-0554
Printed in Sweden
www.criticalcarenews.com
info@criticalcarenews.com

The views, opinions and assertions expressed in the interviews are strictly those of the interviewed and do not necessarily reflect or represent the views of Maquet Critical Care AB.

©Maquet Critical Care AB, 2009.

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted in any form or by any other means, electronic, mechanical, photocopying, recording, or otherwise, without the prior written permission of the copyright owner.

The following designations are registered or pending trademarks of MAQUET Critical Care AB: Servo-i®, Automode®, Open Lung Tool®, NAVA®



Participants registering for the three day Lung Protection Symposium and Workshop

Lung Protection Symposium and Workshop Summary with focus on – Lung Recruitment and Neurally Adjusted Ventilatory Assist - NAVA

Over 100 participating physicians from Hong Kong, Macao, Taiwan, and the People's Republic of China attended a three day Lung Protection Symposium and Workshop at the Prince of Wales Hospital in Hong Kong in February, for an educational opportunity to learn about lung recruitment and NAVA.

The activity, which was the third Lung Protection Symposium hosted by the Prince of Wales Hospital in recent years, featured a faculty of globally renowned guest speakers including Dr Fernando Suarez Sipmann of the Fundación Jiménez Díaz Hospital in Madrid, and Dr Charles Gomersall of the Prince of Wales Hospital in Hong Kong.

The participants were welcomed officially by CM Leung, CEO of Maquet Hong Kong, who co-hosted the activity together with the Prince of Wales Hospital. The three day program featured lectures by the guest speaking faculty, followed by two days of hands-on workshop in the physiology laboratory.

Lung recruitment – the concepts, the practical approach, and ideas about how to improve the strategy at bedside

Fernando Suarez Sipmann of Hospital Fundación Jiménez Díaz in Madrid, has a primary interest in research in mechanical ventilation and respiratory physiology, and has been working in the areas of research and treatment of patients with lung recruitment since 1996. He introduced his presentations as lectures on lung protective ventilation, especially lung recruitment and PEEP as closely linked elements of a lung protective strategy. Dr Suarez Sipmann highlighted the classic pressure-volume curve in relation to ventilator induced lung injury as a frame about how application of mechanical ventilation has an impact on patient outcome. Theoretically, the PV curve shows all of the possibilities to ventilate a specific lung mechanically. Given the different combinations, exceedingly high volume or pressure in the right upper area of the curve, subjects the tissue to mechanical stress, with risk of rupture of alveolar walls. Ventilating at those pressures over time can aggravate respiratory failure. The other extreme is the region of low volume and pressures, down left where the lung tends to collapse. Mechanical ventilation forces bring several dangers in the collapsed region, principally stress by continuously opening and closing small airways and alveoli. Dr Suarez Sipmann highlighted the concept of ventilating ideally in the safe zone, i.e. away from the area of over distention and away from the collapsed zone.

Landmark lung recruitment study

Dr Suarez Sipmann referred to the authors that first introduced the term “lung protection” in clinical practice, in the study by Amato et al in 1998 in the New England Journal of Medicine, which was a turning point in mechanical ventilation. The authors combined all those concepts that were thought at that time to have a protective effect on the lung: to reduce tidal volume and plateau pressure allowing CO_2 to rise



Dr Fernando Suarez Sipmann of Madrid lectured on lung recruitment concepts and bedside strategy

preventing the lung from overdistention and to apply higher levels of PEEP based on the lower inflection point of the PV curve to minimize collapse, and putting all of these strategies together introducing the term of lung protective ventilation strategy. A reduction in mortality of up to 30% in patients in the protective strategy, compared to a conventional group in this study taught that the way the physician turns knobs on the ventilator, without the need of expensive drugs and treatments, has in itself an impact on patient's outcomes, according to Dr Suarez Sipmann.

He stated that this landmark study was followed by a number of other clinical studies that tried to reproduce the benefits of lung protective ventilation strategies, some with positive results, some negative. The most important one was by the NIH group in the NEJM, which is also called the ARDSNet trial. After enrolling 800 patients this study could demonstrate that simply adjusting one intervention – limiting the tidal volume – had a positive impact on patient's outcome.



Fernando Suarez Sipmann of Hospital Fundación Jiménez Díaz in Madrid

Protective ventilation – two different views

Dr Suarez Sipmann illustrated an example of a patient with ARDS with a CT scan showing aeration of upper non-dependent regions combined with extensive collapse in the dorsal dependent lung regions. This is the scenario for a heterogeneous distribution of ventilation with the boundary regions of aerated and collapsed regions, submitted to a high mechanical stress. He stated that when facing such a condition there are two different views of how to best manage this patient.

One current view may be termed as “permissive atelectasis”. This view is represented by the ARDSNet approach, where the ventilation strategy is

adapted to the aerated lung which due to its reduced size has been termed the baby lung. In this approach, it is recommended to reduce tidal volume to minimize overdistention but tolerate atelectasis, in the hope that the collapsed lung will recover over time and that the mechanical stress to the aerated lung can be minimized.

The other current view of managing this condition is what Dr Suarez Sipmann calls “the Open Lung approach”, which is a ventilation strategy aimed at actively restoring and maintaining the size of the ventilated lung. This current of understanding emphasizes that atelectasis is a pathological condition of the lung, per se harmful. Their presence results in poor oxygenation and mechanics, a more heterogeneous

distribution of tidal ventilation facilitating the overdistention of the aerated regions and promoting cyclic opening and closing of the boundary regions.

Importantly, both views aim at protecting the lung sharing the idea of limiting overdistention and tidal recruitment. In the Open Lung approach according to Dr Suarez Sipmann the major difference resides in the attempt to re-expand the collapsed lung by means of a recruitment maneuver and, specifically, in the use of PEEP which in such a strategy aims at stabilizing the recruited lung at end-expiration.

According to Dr Suarez Sipmann, only tidal volume limitation might not be enough to fully protect the lung from ventilation induced lung injury. Recent



Dr Suarez Sipmann has been involved with research and clinical application of lung recruitment maneuvers for many years

studies have shown that in a certain population of ARDS patients with higher amounts of collapse, tidal volume limitation as recommended by the ARDSnet approach resulted in areas of overdistention that appeared as areas of excessive aeration in CT scans at end-inspiration. Dr Suarez Sipmann referred to the study by Terragni et al in AJRCCM 2007, which illustrated that the two main mechanisms of ventilation induced lung injury, overdistention and tidal recruitment, could be present when applying the ARDSnet strategy. Therefore, we cannot assume that just limiting tidal volume to a certain extent, is enough to protect the lung in all patients, according to Dr Suarez Sipmann.

Definition of Lung Recruitment and procedure in theory

Dr Suarez Sipmann defined lung recruitment as the “re-expansion of

previously collapsed lung units by means of a brief controlled increase in transpulmonary pressure that results in an instantaneous change of the lung’s morphological and physiological condition”. He highlighted the concepts of a controlled increase in transpulmonary pressure, so that the physician is aware and in control at each step of the recruitment process.

In terms of re-expanding the lung and keeping it open, Dr Suarez Sipmann explained that the objective in this stage of recruitment is to shift ventilation from the inflation to the deflation limb of the PV relationship, i.e. establishing tidal ventilation in the outer deflation limb, around the point of maximum curvature, away from the collapse region. To achieve this the lung must first stay a brief period on the overdistention part of the curve, which is the only way to have access to the deflation portion where we want to establish ventilation. The minimum

level of PEEP must be found that is able to maintain tidal ventilation exactly at this region where collapse is avoided, according to Dr Suarez Sipmann.

Dr Suarez Sipmann illustrated changes in lung aeration in the recruitment procedure by means of a PV curve with inspiration and expiration of a sick ARDS patient, presented as CT scans, showing that setting ventilation along the inspiratory limb of the PV curve is associated to the the presence of collapse.

Dr Suarez Sipmann stated that if the areas of the lung that are closed are recovered and stabilized, they will increase the functional lung units improving gas exchange, and lung mechanics and more importantly contributes to minimize ventilation induced lung injury.

Dr Suarez Sipmann further illustrated the differences between establishing ventilation in the inflation as compared to the deflation PV limb. In the same patient with pulmonary ARDS, stepwise increases of 2 cm H₂O of PEEP while maintaining the same tidal volume and respiratory rate provided modest changes in oxygenation and compliance. However when PEEP steps were reduced after recruitment, maintaining the same ventilatory settings, there was a marked increase in oxygenation and compliance until the critical level of PEEP, the closing pressure was reached. This means that provided the patient can be effectively kept at this level, ventilation can be maintained at lower pressures and tidal volumes over the long term, in a better physiological condition, as presented by Dr Suarez Sipmann.

Different types of lung recruitment maneuvers?

Dr Suarez Sipmann stated that life is not simple at the bedside, and recruitment is not a simple procedure. There is an ongoing debate on which is the best recruitment maneuver, and published data on patient outcome are still not available. Since the landmark study by Amato et al in NEJM 1998, a remarkably long list of clinical studies on

lung recruitment have been published. According to Dr Suarez Sipmann, lung recruitment must be aimed at recruiting as much lung as possible to efficiently establish a lung protective strategy. When only transient improvements in oxygenation or lung mechanics are sought recruitment is reduced to a matter of “lung cosmetics” not a rational ventilation strategy. In such a context its use is not justified.

The group of Amato has published an important recent study (Borges, AJRCCM 2008) where many important concepts of lung recruitment were described, and where they validated the criterion of PaO₂ for defining a fully recruited lung in 25 ARDS patients. Whenever the PaO₂ was less than 350, there was significant collapse seen on the CT scan. A sequential recruitment maneuver with different levels of pressure was applied, and in patients who were fully recruited according to PaO₂ criteria (PaO₂/FiO₂ > 300 mmHg) and CT criteria also showed an increase in static compliance of 15%.

From the many published clinical studies describing different lung recruitment maneuvers, Dr Suarez Sipmann emphasized primarily three types of recruitment maneuvers, including the most frequently described in literature; the CPAP maneuver, or the so called 40/40 maneuver. Although the CPAP maneuver is the most frequently described in the literature, in up to 40-50% of the studies, Dr Suarez Sipmann believes that this is not the best strategy and should not be used. The patient remains in apnea and maximal level of pressures are maintained for the entire period significantly increasing the hemodynamic effects.

He strongly recommends a cycling recruitment maneuver in Pressure controlled ventilation. He described two types of cycling maneuvers, one called single pulse in Pressure Control ventilation, which is recommended for patients in less severe disease, for example in postoperative patients where pressures can be increased in two or three short steps to the recruitment level, generally in the range of 45 cm H₂O. The other cycling maneuver consists in

applying several sequential incremental PEEP steps, where the maneuver can be individualized to the particular patient. From baseline, a level of PEEP is selected to be high enough to maintain the entire recruited lung stable usually in the range of 25 cm H₂O in ARDS patients, being this the level of PEEP maintained along the recruitment sequence. As shown by Borges et al, different recruitment levels can then be explored by increasing PEEP in 5 cm H₂O steps above baseline while maintaining a fixed inspiratory pressure gradient for 2 minutes and returning to baseline PEEP after each step for evaluation. If Open Lung criteria have been obtained according to oxygenation and/or lung mechanics no further levels of pressure need to be explored. If not, the next level of inspiratory recruitment pressure is explored.

Dr Suarez Sipmann described time dependency as a factor, with the need to apply a certain amount of pressure for a certain amount of time. There is a minimum threshold pressure, the critical opening pressure, that has to be overcome otherwise no significant recruitment effect, independently of the time it is applied can be expected. As the lung gets sicker, more time and higher pressure are needed according to Dr Suarez Sipmann.

Higher pressures in ARDS to optimize lung recruitment in ARDS

Dr Suarez Sipmann illustrated the complexity of the situation in regard to the heterogeneity of the ARDS lung: lungs with significant collapse show a vertical gravitational gradient of opening pressures, which means that the alveoli at the dorsal, dependent region require much higher opening pressures than those in the ventral non-dependent regions.

Dr Suarez Sipmann referred to the recruitment pressures in ARDS lungs reported by Borges et al in the AJRCCM in the distribution of opening pressures in severe ARDS patients. Although over 50% were fully recruitable with pressures of 45 cm H₂O, the most diseased lungs required up to 60 cm H₂O in some

cases. According to Dr. Suarez Sipmann this study highlighted the importance of individualizing the recruitment pressures to each particular condition looking for the minimum pressure that results in full lung recruitment.

Dr Suarez Sipmann went on to illustrate the occurrence of important uncontrolled increases in airway pressures associated with routine interventions such as bagging. He showed an example where bagging pressures were recorded with repeated pressure peaks over 50 cm H₂O –with one of the bags resulting in pressures over 80 cm H₂O. The effects of handbagging, which occurs frequently in routine clinical practice, can sometimes result in much higher pressures than those reached during controlled recruitment maneuvers.

Lung recruitment side effects

Dr Suarez Sipmann reported that in a systematic review in AJRCCM 2008 of 40 different studies with over 1200 patients, in different situations submitted to lung recruitment, the number of side effects reported are surprisingly low either before, during or after the recruitment procedure. He stated that before pressurizing the patient, a good volemic status and a stable hemodynamic condition must be confirmed. He outlined that the hemodynamic effects of lung recruitment are mainly caused by a decreased venous return, increased right ventricular afterload and compromised left ventricular filling due to ventricular interdependence. The major effects include reduced cardiac output, and reduced systolic arterial pressure. “However, when performing a recruitment maneuver in a protocolized way most of the time these side effects during recruitment maneuvers are small in magnitude, preventable and self limited”, stated Dr Suarez Sipmann. He went on to explain how hemodynamics depends on lung condition and volemia. Dr Suarez Sipmann explained: “As compliance decreases, the transmission of alveolar pressure to the pulmonary circulation also decreases so that sicker patients needing higher recruitment pressures are more protected against hemodynamic side effects.



Mr Arne Lindy of Maquet Critical Care, thanking Dr Suarez Sipmann

Dr Suarez Sipmann outlined that the type of recruitment maneuver is an important factor in influencing the hemodynamic response. This was systematically assessed in an experimental study where different lung injury models were submitted to different recruitment maneuvers by Dr Marini's group. They could show that sustained inflation (the 40/40 maneuver), caused the most important decrease in cardiac output. Best tolerated was a cycling Pressure Control maneuver, with maximum decreases of 20 - 30% in cardiac output, which recovered to normal within a few minutes after the recruitment maneuver. Dr Suarez Sipmann stated "The 40/40 is not the best approach, the pressures are not enough, patients are apneic, so the thorax is continuously pressurized, causing major hemodynamic effects." In terms of hemodynamic management in lung recruitment, Dr Suarez Sipmann recommended a protocol of baseline ventilation, and an adequately resuscitated patient with stable cardiac output and mean arterial pressure. He outlined one of his ongoing studies, looking at

the impact of right ventricular and left ventricular output during recruitment maneuvers in ARDS patients.

Rationale for using Pressure Control ventilation during recruitment

According to Dr Suarez Sipmann, the rationale for using Pressure Control during recruitment includes the following factors:

- Strict control over inspiratory pressures
- No pendelluft phenomenon: new recruited regions always receive fresh gas from the ventilator
- Cycling allows pressure relief during expiration

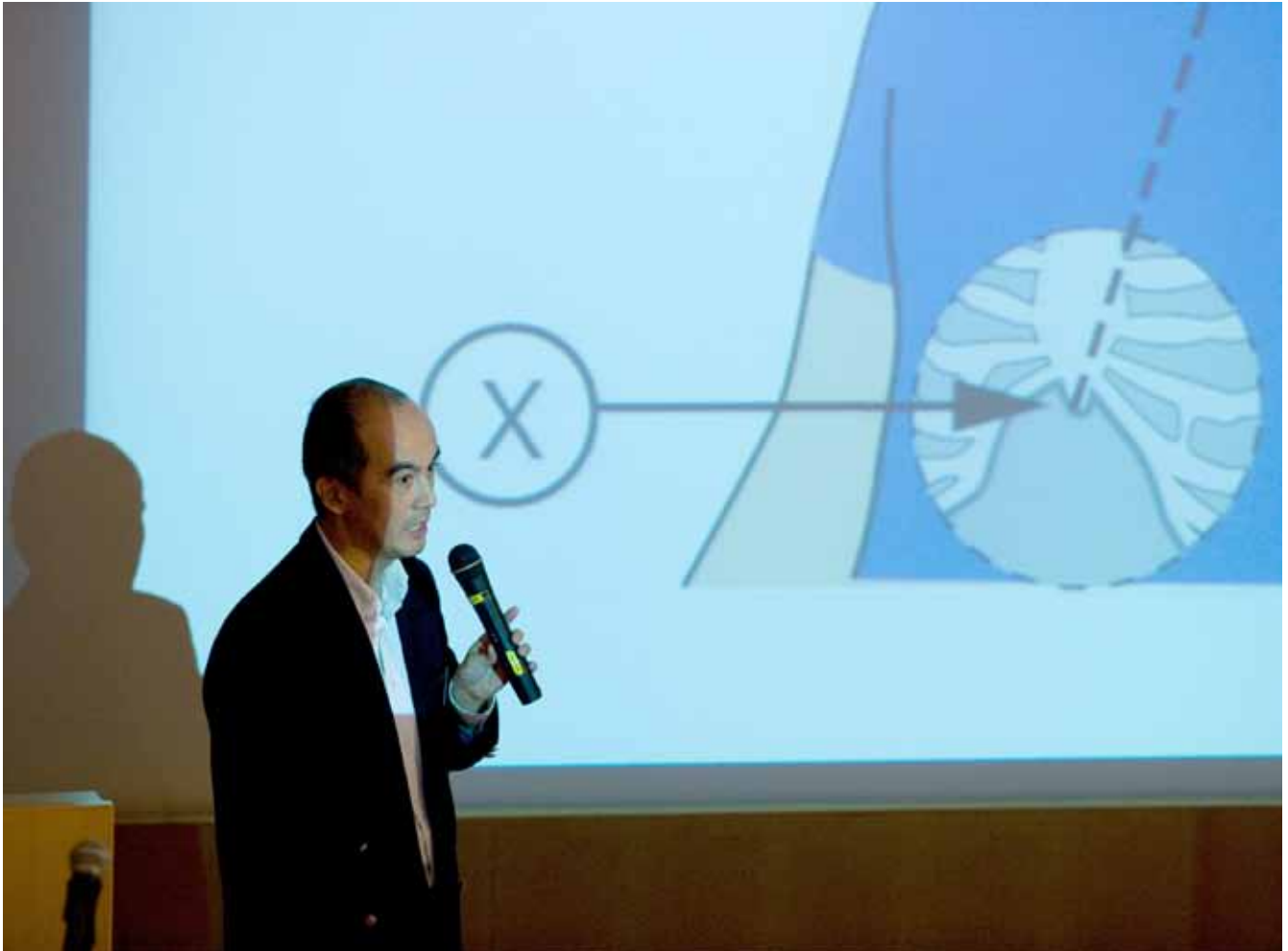
The role of PEEP

He went on to state that the NIH trial paid less attention to the importance of PEEP, which was selected as the lowest possible level for a needed FiO_2 to achieve a specific oxygenation target. He stated that this table has no physiologic rationale which is one of the major critiques to the NIH

-ARDSNet trial according to Dr Suarez.

Dr Suarez Sipmann also referred to 3 fairly recent studies focusing on high versus low PEEP; the ALVEOLI (U.S. study) reported in NEJM 2004, the LOVS study (Canada) reported in 2008, and the EXPRESS study from France, also reported in 2008 both in JAMA. He commented: "There are now three large randomized clinical trials that could not demonstrate a clear benefit of higher levels of PEEP on survival." There was however, a trend to a beneficial effect of the use of higher levels of PEEP and clear benefits on important secondary endpoints, that support the recommendation of using higher levels of PEEP. Except for the Express study, PEEP was selected according to a PEEP/ FiO_2 table, only looking at oxygenation. This resulted in a constantly higher plateau pressure in the high PEEP group as compared to the lower PEEP groups. According to Dr Suarez Sipmann the lack of control of the increase in plateau pressures may have offset the potential benefits of the use of higher PEEP in these studies.

Clinical application of Neurally Adjusted Ventilatory Assist – NAVA



Dr Charles Gomersall of the Intensive Care Department of the Prince of Wales Hospital in Hong Kong, presented the concept and clinical application of NAVA

Dr Charles Gomersall of the Intensive Care Department of the Prince of Wales Hospital in Hong Kong has been familiar with the clinical application of Neurally Adjusted Ventilatory Assist, or NAVA in his intensive care department since late 2007. He started his presentation by presenting the physiological concept of NAVA, where a patient's neural respiratory signals, or Edi – electrical activity of the diaphragm, were used to trigger the ventilator according to the patient's own requirements. Dr Gomersall outlined the differences in the concept of NAVA compared to conventional pneumatic mechanical ventilation by explaining: "Conventional ventilator technology uses a pressure drop or flow reversal to initiate

the assist delivered to the patient. With NAVA, the electrical discharge of the diaphragm is captured by an Edi catheter fitted with an electrode array. The signal is then passed to the ventilator, which executes both triggering and flow control in proportion to the received Edi signal."

Practical set up of NAVA

Dr Gomersall briefly reviewed the components needed for NAVA, consisting of ventilator, software, NAVA module, and Edi catheter with 10 electrodes. He reviewed the NEX measurement formula and procedure to estimate the insertion length of the Edi catheter needed. Dr

Gomersall went on to explain: "When the Edi catheter is in place, you see esophageal ECG – an added benefit of the Edi catheter. I am waiting for a broad complex tachycardia, so that I can say that I diagnosed a type of tachycardia from a nasogastric tube and ventilator. Verify the position of the Edi catheter by means of the ECG waveforms."

Dr Gomersall also presented information about what NAVA levels entail. He described the NAVA level as an amplification factor. In the NAVA mode the SERVO-i will amplify each Edi sample as determined by the set NAVA level, after the following formula: $P = \text{NAVA level} \times (\text{Edi signal} - \text{Edi min}) + \text{PEEP}$. The

amplification depends on the selected NAVA level and results in a smoothly rising pressure. The shape of the pressure curve mirrors the Edi signal until the Edi signal drops to 70% of peak Edi. The ventilator cycles to expiration, and the pressure will drop to the set PEEP level. Dr Gomersall explained: "Not only does NAVA give you a way of triggering the ventilator, the profile of the breath is determined by the patient's effort."

To address the question if NAVA unloads respiratory muscles, Dr Gomersall referred to the literature, where a study in healthy volunteers comparing 0 and high levels of NAVA support (Sinderby et al, *Chest* 2007; 131(13): 711-717), NAVA allows the patient to take the same tidal volume while making less effort, according to Dr Gomersall.

Dr Gomersall also showed data from a patient in his intensive care unit, where the NAVA level was 1.5 cm H₂O/ μ v, increasing the NAVA level to 3 cm H₂O/ μ v resulted in a reduction in Edi with maintained tidal volume. This indicates that the respiratory work was taken over from the patient by the ventilator.

Ventilatory dyssynchrony and its consequences

Dr Gomersall addressed the subject of ventilatory dyssynchrony and its consequences, referring to Thille et al (*Intensive Care Med* 2006; 32(10):1515-1522) where the authors studied 62 patients triggering the ventilator. They investigated the impact of dyssynchrony in assist controlled and Pressure Support ventilation. The main outcome of the study was that the length of mechanical ventilation was much extended in patients with dyssynchrony.

He summarized that from an outcome perspective, patients with an asynchrony index higher than 10% were likely to have a longer time on mechanical ventilation and a higher incidence of tracheostomy.

Does NAVA have an effect on asynchrony?

Dr Gomersall addressed this question

by presenting some of the original pre-clinical scientific studies, comparing NAVA and Pressure Support ventilation, in low, medium and high levels. He said: "It is difficult to set equivalent level of NAVA to Pressure Support, but if you used peak pressure, the waveforms are different, and pressure time curve is different in NAVA. Tidal volumes don't change much with NAVA. In terms of peak airway pressure and tidal volume there is a contrast between NAVA and Pressure Support modes," according to Dr Gomersall. "As you increase the NAVA level the peak airway pressure and the tidal volume doesn't change much. When you increase the inspiratory pressure level in Pressure Support, the peak airway pressure and tidal volumes increase markedly. Pressure time product of diaphragm or work of breathing of diaphragm, with NAVA as you increase level, the work goes down. With Pressure Support, it falls initially, but then rises."

In terms of trigger delay, with NAVA the trigger delay is not really a factor, as presented by Dr Gomersall. With Pressure Support the trigger delay increases as you increase the level of Pressure Support, reflecting hyperinflation induced trigger load. According to Dr Gomersall, the explanation is that what happens in Pressure Support is that as the pressure and therefore the tidal volume goes up there is more gas trapping, resulting in trigger delay. On one hand work of breathing decreases by increasing Pressure Support, on the other hand it is harder to trigger as Pressure Support levels are increased.

Human data

Dr Gomersall reviewed a study by Colombo et al consisting of 14 patients in Pressure Support and NAVA, with equivalent levels of Pressure Support and NAVA on maximum voluntary breaths. Edi was suppressed in Pressure Support compared to NAVA. In high levels of Pressure Support the triggering was not effective. Diaphragmatic activity in Pressure Support was found to be variable, and tidal volume to be constant in Pressure Support. In the same study NAVA provided variable

Edi and variable tidal volume, as the patient is controlling tidal volume much more effectively than in Pressure Support, according to Dr Gomersall.

He also stated that the grouped data from this study showed that neural inspiratory time does not change with level of support. "Inspiratory time increases in Pressure Support as you increase level of Pressure Support. Neural expiratory time and flow time goes up in Pressure Support. The reason for neural expiratory time increasing in increasing levels of Pressure Support, is that the inspiratory time is being artificially prolonged, the patient is unable to exhale when they desire, and expiration is delayed," as explained by Dr Gomersall.

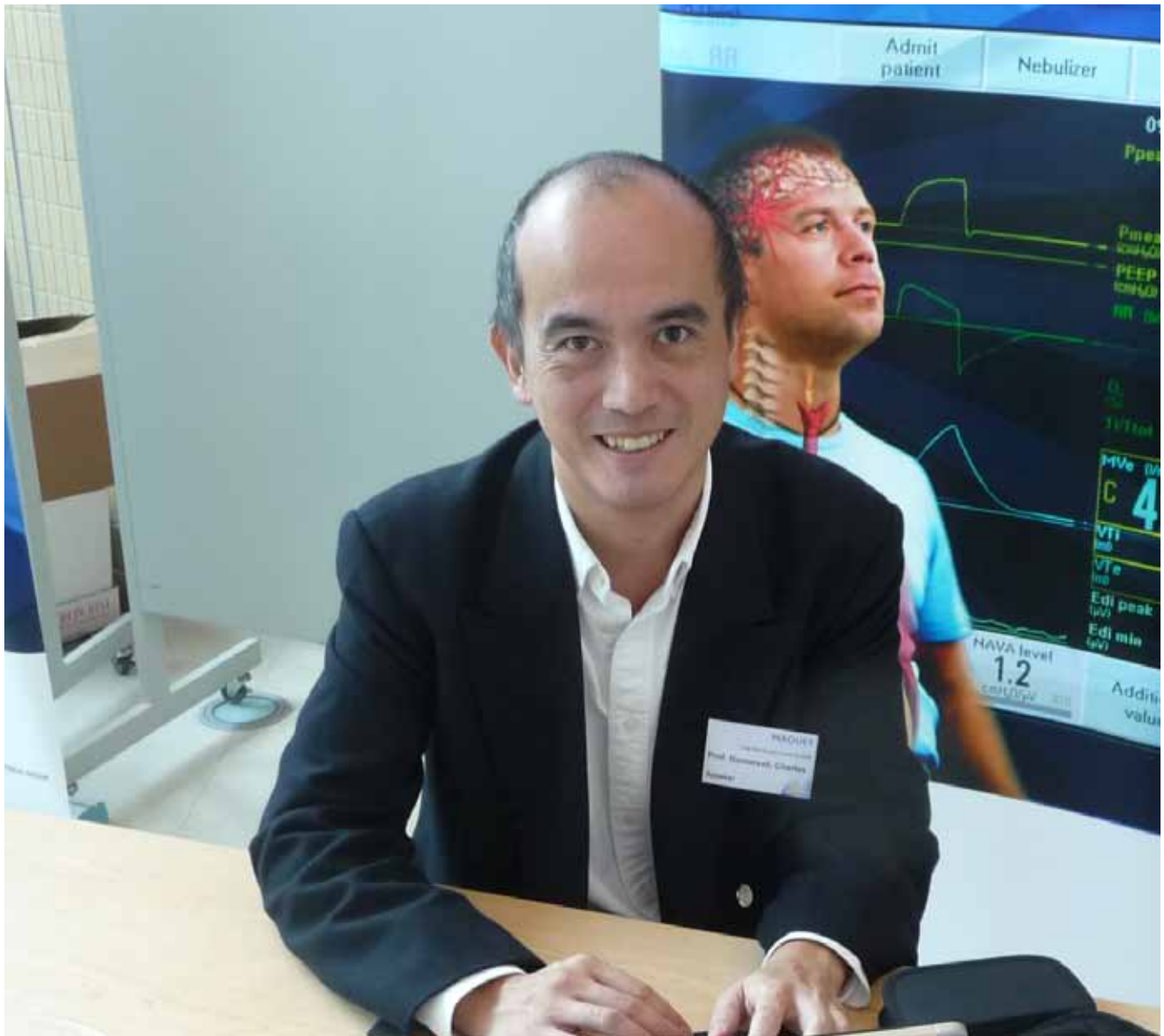
He summarized the study by stating that using a cut-off of asynchrony index greater than 10, five patients in the Pressure Support group were found to be asynchronous, but none in NAVA group.

NAVA experience in clinical practice

Dr Gomersall said that he did not yet have systematic data, and for this reason preferred to present a few patient cases from his practice.

Intermittent dyspnea

This patient case was an obese lady with neuropathia, who was difficult to wean and had intermittent dyspnea on Pressure Support ventilation. A bronchoscope was placed for observation, and in her trachea it was found to be moving inwards on inspiration. It revealed some collapse down to about one-third of its normal diameter. The patient had PEEP and Pressure Support, of which some was lost during bronchoscopy. The ICU team interpreted that there was signal trigger delay and that the ventilator did not deliver breaths as fast as the patient desired. When the patient was taken off the ventilator, no dyspnea occurred, however she was not strong enough to stay off the ventilator for a long period of time. She was started on NAVA, which obliterated trigger delays, and clinically



Dr Charles Gomersall

she was much more comfortable. The patient was now easily weaned on NAVA.

Cheyne-Stokes

Dr Gomersall and his colleagues have experienced some advantage with NAVA in the category of patients with Cheyne-Stokes respiration, with crescendo-decrescendo pattern of breathing in rate and depth, with periods of apnea. He explained: "If you ventilate these patients on Pressure Support, you get standard tidal volume for each time they trigger the ventilator. So instead of getting a crescendo and decrescendo in rate and depth, you get constant values, which

exacerbates the problem of apnea, so that the ventilator, even if set at max apnea time, goes into back-up ventilation, therefore it is difficult in getting the patient to breathe more. If you take these patients off the ventilator, the issue of apnea becomes less of a problem, but these patients are not able to tolerate spontaneous breathing for long periods of time. In this group, when we put in the Edi catheter and switch to NAVA, it is much easier to wean these patients, since they have control; the tidal volume is dependent on their respiratory effort."

Dr Gomersall also illustrated experience with examples of ventilation in PRVC,

with significant dyssynchrony between PRVC breaths and Edi and no correlation between PRVC breaths and attempts to breathe. He added "NAVA gets perfect synchrony in these patients. In a recent patient with a flow pattern that was odd, she was attempting to breathe between breaths with failure to trigger; perfect synchrony was obtained in switching to NAVA. Dr Gomersall concluded his presentation by summarizing that NAVA is a promising new mode of ventilation, simple to use, and will effectively off-load respiratory muscles. He stated that NAVA reduces dyssynchrony.

Hands-on physiology workshop and participant feedback



Some of the many workshop participants

The purpose of the workshop was to give the participants the opportunities to perform recruitment maneuvers while individualizing treatment to the specific lung, with extensive monitoring of the respiratory as well as the hemodynamic side. On the respiratory screen, the Open Lung tool, trends on time scale on pressures, inspiratory and expiratory tidal volumes, and the inspiratory dynamic compliance and tidal CO₂ elimination were utilized for lung protective recruitment maneuvers.

The workshop also gave the opportunity to observe electrical activity of the diaphragm by means of Edi signals and to monitor patient-ventilator synchrony by means of NAVA.

Critical Care News spoke with some of the participants of the lung protective symposium and workshop, to hear their feedback after the sessions.

Dr Dennis Kin Long Wong, Macao Government Conde de São Januário Hospital

I am an ICU physician and medical officer treating adult patients in a government hospital in Macau. I had read to prepare before coming here to join the two day lung protective course. We can have a real chance to practice here to consolidate our clinical knowledge, which is a nice opportunity for us.

When returning home after the workshop, will you be trying to do recruitment maneuvers?

Yes, I will definitely try, on ARDS patients and maybe ALI patients and some severe pneumonia patients to start out with. I will start with a few patients first, we have 10 beds in our ICU which is a general medical/surgical/neurological ICU. The symposium and workshop have been very good, I learned a lot. I would like to join similar courses in the future.



Dr Wong is Medical Officer at the Macao Government Hospital ICU



Dr Hsinkuo Kao and Mrs Shiu Hui Qua, from Taiwan

Intensivist Dr Hsinkuo Kao, and Mrs Shiu Hui Qua, Respiratory Therapist, Taiwan

Dr Kao: We work at the UGH hospital in Taipei, where we currently have a 30 bed ICU, which is on the way to increasing to 36 beds. It is a medical ICU; most patients belong to the medical care program.

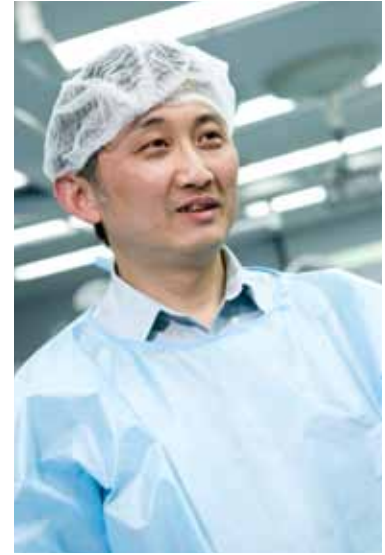
Are you familiar with lung recruitment procedures?

Dr Kao: Yes – we have had meetings about lung recruitment, and we perform these maneuvers in our patients, we select patients with ARDS and we look at hemodynamics as a major individual factor in performing the maneuvers. We know the patients conditions and the situation to stabilize, we perform the lung recruitment maneuvers and we check by chest x-ray. In our department, ARDS is often complicated by pneumonia, so that in these patients with pneumonia related ARDS we perform these maneuvers.

How will you teach your fellow respiratory therapists about lung recruitment?

Mrs. Qua: As Director of Respiratory Therapy, I have 40 RT therapists that report to me. We have education by coworkers but are more interested in this technique, and we need doctor approval to teach the technique, in order to support it and apply it. Education is the first step and we have other physicians that perform various types of recruitment maneuvers in our hospital, but we know we can monitor useful parameters with the Open Lung tool and dynamic compliance as a new element in our treatment practice. In the past we have perhaps performed lung recruitment by more traditional methods, but it was not apparent exactly what was happening to our patients, and there is more data today to support different methods than in the past.

Dr Kao: By using the Open Lung tool, I can see parameters that might help me to diagnose and understand the



Dr Zhou Suming of The People's Hospital of Jiangsu Province, PRC

results at bedside. This has been a very valuable workshop for us. We see the lung recruitment effects in ARDS patients today, but I think we are finding the value of recruitment maneuvers in other types of patients as well.

Zhou Suming, Director of ICU, The People's Hospital of Jiangsu Province, People's Republic of China

It was the first time I have seen lung recruitment maneuvers. We have SERVO-i in our ICU and sometimes we see ARDS patients, so that is the basis of my interest in attending this workshop. My intensive care unit is 10 beds, and it is a geriatric ICU, we see a lot of COPD, and sometimes we see surgical patients as well.

Are you interested in NAVA?

I think that has been the most interesting aspect of the workshop for me, it is very hard for us sometimes to wait for the ventilation to gather information; NAVA might give us information at bedside. With geriatric patients and issues in regard to sleep quality at night, NAVA might be an interesting opportunity for us in future. This information, as well as the lectures and questions and workshops have been very useful. ■

Biographies

Fernando Suarez Sipmann

conducted his initial Study of Medicine at the University of Navarra, Pamplona, Spain and earned his degree there in 1990. During the years of 1991 and 1996, he specialized in Intensive Care Medicine at the Fundación Jiménez Díaz University Hospital of the Universidad Autónoma in Madrid. Since then he has been working as a consultant in Intensive Care Medicine at the same institution.

He earned his PhD degree at the Department of Clinical Physiology of the University of Uppsala, Sweden in 2008. He is internationally known for his research in intensive care, is well-known within the lecture circuit, and has been a frequently invited speaker, main lecturer or chairman in international congresses, symposia and other venues during the past ten years.

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 anesthesia 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 heavily involved in the CoBaTRICE project.

References

- 1) Costa EL, Borges JB, Melo A, Suarez-Sipmann F, Toufen C Jr, Bohm SH, Amato MB. Bedside estimation of recruitable alveolar collapses and hyperdistension by electrical impedance tomography. *Intensive Care Med* 2009; 35(6): 1132-1137.
- 2) Borges JB, Carvalho CR, Amato MB. Ventilation strategies for acute lung injury and acute respiratory distress syndrome. *JAMA* 2008; 300(1): 41-42.
- 3) Fan E, Wilcox ME, Brower RG, Stewart TE, Mehta S, Lapinsky SE, O Meade M, Ferguson ND. Recruitment maneuvers for acute lung injury – A systematic review. *AJRCCM* 2008; 178: 1156-1163.
- 4) Villar J, Perez-Mendez L, Lopez J, Belda J, Blanco J, Saralegui I, Suarez-Sipmann F, Lopez J, Lubillo S, Kacmarek RM, HELP Network. An early PEEP/FIO2 trial identifies different degrees of lung injury in patients with acute respiratory distress syndrome. *AJRCCM* 2007; 176(8): 795-804.
- 5) Suarez-Sipmann F, Bohm SH, Tusman G, Pesch T, Thamm O, Reismann H, Reske A, Magnusson A, Hedenstierna G. Use of dynamic compliance for open lung positive end-expiratory pressure titration in an experimental study. *Crit Care Med* 2007; 35(1): 214-221.
- 6) Borges JB, Okamoto VN, Matos GF, Caramaz MP, Arantes PR, Barros F, Souza CE, Victorino JA, Kacmarek RM, Barbas CS, Cavalho CR, Amato MB. Reversibility of lung collapse and hypoxemia in early acute respiratory distress syndrome. *AJRCCM* 2006; 174(3): 268-278.
- 7) Amato MBP, Barbas CSV, Maderios DM, Magaldi RB, Schettino GP, Lorenzi-Filho G, Kariralla RA, Deheinzelin D, Munoz C, Oliveira R, Takagaki TY, Carlvahlo CRR. Effect of a protective ventilation strategy on mortality in acute respiratory distress syndrome. *N Engl J Med* 1998; 338(6): 347-354.
- 8) Lecomte F, Brander L, Jalde F, Beck J, Qui H, Elie C, Slutsky AS, Brunet F, Sinderby C. Physiological response to increasing levels of neurally adjusted ventilatory assist (NAVA). *Respir Phys & Neurobiology* 2009; doi: 10.1016/j. resp 2009-02-015.
- 9) Brander L, Leong Poi H, Beck J, Brunet F, Hutchison SJ, Slutsky AS, Sinderby C. Titration and implementation of Neurally Adjusted Ventilatory Assist in critically ill patients. *Chest* 2008; Doi 10.1378/chest.08-1747.
- 10) Colombo D, Cammarota G, Bergamaschi V, De Lucia M, Della Corte F, Navalesi P. Physiologic response to varying levels of pressure support and neurally adjusted ventilatory assist in patients with acute respiratory failure. *Intensive Care Med* 2008; doi 10.1007/s00134-008-1208-3.
- 11) Sinderby C, Beck J. Neurally Adjusted Ventilatory Assist (NAVA): An Update and Summary of Experiences. *Neth J Crit Care* 2007; 11(5): 243-252.
- 12) Sinderby C, Navalesi P, Beck J, Skrobik Y, Comtois N, Friberg S, Gottfried SB, Lindstrom L. Neural control of mechanical ventilation in respiratory failure. *Nat Med* 1999; 5(12): 1433-1436.
- 13) Sinderby C, Beck J, Spahija J, de Marche M, Lacroix J, Navalesi P, Slutsky AS. Inspiratory muscle unloading by neurally adjusted ventilatory assist during maximal inspiratory efforts in healthy subjects. *Chest* 2007; 131(3): 711-717.
- 14) Thille AW, Rodriquez P, Cabello B, Lellouche F, Brochard L. Patient-ventilator asynchrony during assisted mechanical ventilation. *Intensive Care Med* 2006; 32 (10): 1515-1522.