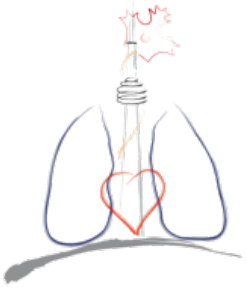
UNIVERSITY OF TORONTO | Interdepartmental Division of Critical Care Medicine



ART SLUTSKY RESEARCH DAY

Interdepartmental Division of Critical Care Medicine
University of Toronto

Tuesday, June 19, 2018

Campbell Conference Facility
Munk School of Global Affairs
Trinity Site, 1 Devonshire Place
University of Toronto

8:00 –8:55 am Registration and Light Breakfast

8:00 – 8:45 am Poster Set-Up

Posters should be on display until **3:00 pm** so they are available for viewing both at the designated time with the authors (see below), and informally (without authors) at coffee breaks and lunch.

Please set up posters before the start of the oral presentations to avoid interruptions.

We will supply VELCRO (we are not allowed to use pins on these rented boards)

8:55 – 9:00 am **Welcome and Introductory Remarks**

IDCCM Research Director: Dr. Margaret Herridge

9:00 – 9:45 am **Oral Presentations 1: Mix of all abstract types**

Chairs: Drs. Peter Laussen and Damon Scales

Each presentation is 10 min followed by 5 min discussion.

All speakers will be requested to adhere to the time limit.

9:00 – 9:15 am **Presentation 1**: Dr. Lu Chen
Diffuse Airway Closure and Surfactant Degradation Biomarker in Acute Respiratory Distress Syndrome

9:15 – 9:30 am **Presentation 2:** Dr. Saptharishi Lalgudi Ganesan
Factors Reducing the Accuracy of Seizure Identification Using Quantitative Electroencephalography Displays in the Intensive Care Unit

9:30 – 9:45 am **Presentation 3:** Dr. James East
Needs Assessment for Medical Errors in Residency Training – A Qualitative Study

IDCCM Faculty Presentations I

Session Chairs: Drs. Anne-Marie Guerguerian and Brian Cuthbertson

9:45 – 10:00 am **Featured Speaker:** Dr. David Mazer
Topic: Restrictive or Liberal Red-Cell Transfusion for Cardiac Surgery
New England Journal of Medicine 2017; 377:2133-2144

10:00 – 10:15 am **Featured Speaker:** Dr. Elizabeth Wilcox
Topic: Neurocognitive Outcomes and Sleep

10:15 - 11:00 am Morning Break

11:00 – 11:45 am **Oral Presentations 2: Mix of all abstract types**

Chairs: Drs. Jan Friedrich and David Hall

11:00 – 11:15 am **Presentation 4:** Dr. Bhushan Katira
Abrupt Deflation After Sustained Inflation Causes Lung Injury

11:15 – 11:30 am **Presentation 5:** Ms. Victoria Mintsopoulous
Ultrasound and Microbubbles to Deliver Therapy to the Injured Lung in ARDS

11:30 – 11:45 am **Presentation 6:** Dr. Tai Pham
Ethical Decisions in Patients with Severe Acute Respiratory Failure – Determining Factors, Time of Decisions and Impact on Outcome: The Edecide Project. An Ancillary Analysis of Lung Safe Study

11:45 am – 1:30 pm **Lunch and Poster Viewing**

11:45 am – 12:45 pm Poster judging

Presenting authors must be with their posters

12:45 pm – 1:30 pm Lunch and unaccompanied poster viewing

1:20 pm – 1:30 pm Break

1:30 – 2:15 pm **Oral presentations 3: Mix of all abstract types**

Chairs: Drs. Briseda Mema, Mjaye Mazwi and Haifa Mtaweh

1:30 – 1:45 pm **Presentation 7:** Ms. Rebecca Sinyi
An In Situ Neurocritical Care Simulation Curriculum to Foster Ongoing Acquisition of knowledge and Crisis Resource Management Skills within the Interprofessional ICU Team

1:45 – 2:00 pm **Presentation 8:** Dr. Chris Walsh
Multi-Cohort Transcriptional Meta-Analysis of Muscle Diseases Identifies a Gene Signature of Disease Severity

2:00 – 2:15 pm **Presentation 9:** Dr. Daniel Lane
Bayesian Analysis of the Quick Sequential Organ Failure Assessment (Qsofa Score) to Predict Intensive Care Unit Admission and Mortality Among Patients with Infections Transported by Paramedics

IDCCM Faculty Presentations II

Session Chairs: Drs. Natalie Wong, Jeff Singh, Niall Ferguson

2:15 – 2:30 pm **Featured Speaker:** Dr. Chris Parshuram
Topic: Effect of a Pediatric Early Warning System of All-Cause Mortality in Hospitalized Pediatric Patients: the EPOCH Randomized Clinical Trial
The Journal of the American Medical Association, 319(10), February 2018

2:30 – 2:45 pm	**Featured Speaker:** Dr. Victoria McCredie **Topic:** Building Bridges and Bridging Minds in Neurocritical Care
2:45 – 3:15 pm	Afternoon Break
3:15 – 3:45 pm	**State of Research in the IDCCM, University of Toronto** Dr. Laurent Brochard
3:45 pm	**Introduction of Visiting Professor** Dr. Laurent Brochard
3:45 – 4:15 pm	**Featured Speaker** **Marc Moss, MD** Roger S. Mitchell Professor of Medicine Vice Chair of Clinical Research, Department of Medicine, University of Colorado Denver-Anschutz Medical Campus President, American Thoracic Society **Title:** Uncovering the Silent Epidemic of Burnout Syndrome and Psychological Distress in Critical Care Healthcare Professionals
4:15 – 4:45 pm	**Presentation of Certificates of Training Completion** Dr. David Hall and Ms. Stephanie Nardella **Presentation of Site Teaching Awards** **Presentation of The John Granton Teaching Award, Simon Abrahamson & John Laffey Contribution Awards**
4:45 – 5:00 pm	**Andrew Sass-Kortsak Award Announcement** Dr. Laurent Brochard **Presentation of Research Day Prizes and Closing Remarks** Drs. Margaret Herridge and Hannah Wunsch

Thank you!

Have another great year of research and hope to see you next year!

Art Slutsky Research Day 2018
IDCCM Chair: Dr. Laurent Brochard
IDCCM Director of Research: Dr. Margaret Herridge
IDCCM Associate Director of Research: Dr. Hannah Wunsch

GUEST SPEAKERS

Featured Speaker

Dr. Marc Moss

Marc Moss is the Roger S. Mitchell Professor of Medicine and Vice Chair of Clinical Research for the Department of Medicine at the University of Colorado School of Medicine.

Dr. Moss has a longstanding interest in clinical and translational research involving critical care research for over two decades, and he has held continuous NIH funding as a Principal Investigator for over 18 consecutive years. More specifically, Dr. Moss's research interests include identifying new treatment modalities for patients with the Acute Respiratory Distress Syndrome (ARDS), exploring the diagnosis and treatment of neuromuscular dysfunction in critically ill patients who require mechanical ventilation, and studying burnout syndrome, posttraumatic stress disorder, and wellness in healthcare professionals.

Dr. Moss is also the principal investigator for the Colorado center in the NHLBI sponsored Prevention and Early Treatment of Acute Lung Injury (PETAL) network. Based on his expertise in clinical/translational research and mentoring, Dr. Moss served as the Program Director for the Education, Training, and Career Development Core of the Colorado Clinical Translational Sciences Institute (CCTSI) from 2008-2016.

He also served as the Chair of the American Thoracic Society (ATS) International Conference Committee from 2009 -2011 and as their President from 2017-2018.

Faculty Speakers

Dr. David Mazer

David Mazer, M.D. is a Professor in the Departments of Anesthesia and Physiology at the University of Toronto.

He received his medical training at the University of Saskatchewan (MD 1978) and worked as a family physician in Inuvik NWT, New Zealand, and Saskatchewan before attending the University of Toronto to complete his residency in Anesthesia (FRCPC 1985).

He subsequently completed his fellowship training in cardiovascular anesthesia research at the University of California San Francisco and has been actively engaged in clinical practice and research in Cardiac anesthesia and critical care since 1987.

He has been Associate Editor of the Canadian Journal of Anesthesia, Coordinator of Critical Care and Medical Director of CVICU at St. Michael's Hospital and Vice-Chair for Research for the Department of Anesthesia at the University of Toronto. He currently is Chair of the Research Ethics Board of St. Michael's Hospital.

Dr. Mazer's translational research has focused on improving the practice of Cardiac Anesthesia. His research program focuses on perioperative blood conservation, cardiac physiology and perioperative organ protection. He has co-authored over 100 peer-reviewed publications, including practice guidelines and position statements related to perioperative transfusion and hemostasis from the Society of Thoracic Surgeons, Society of Cardiovascular Anesthesiologists and the Canadian Cardiovascular Society. He was a lead anesthesia investigator in the BART trial and is the principal investigator for TRICS III, a global study of transfusion triggers in cardiac surgery.

Dr. Christopher Parshuram

Dr. Parshuram is a staff physician in the Department of Critical Care Medicine at The Hospital for Sick Children, a Senior Scientist in Child Health Evaluation Sciences at the SickKids Research Institute.

At the University of Toronto he is a Professor in the Department of Paediatrics and is faculty in Critical Care Medicine and Health Policy, Management and Evaluation.

Dr. Parshuram is formally trained in clinical epidemiology, and has expertise in cardiac arrest prevention, reducing errors that are associated with medications, and preventing fatigue in healthcare workers.

He has received peer-reviewed research funding from the Heart and Stroke Foundation of Canada, the Society of Critical Care Medicine, and the Canadian Institutes of Health Research.

Dr. Elizabeth Wilcox

Elizabeth Wilcox received her medical degree from the University of Calgary, and completed her training in internal medicine and adult critical care medicine at the University of Toronto.

After completing research training at the London School of Hygiene and Tropical Medicine in the United Kingdom, she returned to join the critical care faculty at the University of Toronto and the Toronto Western Hospital.

Her research is focused on the organization of critical care services and long-term outcomes from critical illness.

She is currently conducting a large, externally-funded, multicentre cohort study which is evaluating the association of genetic, EEG and sleep to cognitive outcomes and wellness in survivors of critical illness (COGWELL).

Dr. Victoria McCredie

Victoria trained in acute medicine at the University of Manchester, moving to Canada in 2009 to complete fellowships in critical care and neurocritical care medicine, both at the University of Toronto. Victoria went on to complete her Ph.D. in clinical epidemiology at the University of Toronto examining the relationship between processes of care, structure, and outcome for critically ill patients with acute brain injury.

In particular, she studied the withdrawal of life-sustaining therapies, airway management strategies and the organization of critical care services for traumatic brain injured patients.

Victoria's ongoing research program focuses on the organization of critical care for subarachnoid hemorrhage patients, airway management strategies for acutely brain-injured patients, and the development of an integrated neurocritical care clinical informatics platform to evaluate pathophysiologic mechanisms contributing to secondary brain injury during critical illness.

ABSTRACT JUDGES

Basic Science Category	Dr. Jane Batt Dr. Warren Lee
Clinical Research/Health Services Research Category	Dr. Stephen Lapinsky Dr. Margaret Herridge
Physiology Category	Dr. Alberto Goffi Dr. John Granton
Quality Category	Dr. Michael-Alice Moga Dr. Gail Annich
Systematic Review/Case Report/Education Category	Dr. Neill Adhikari Dr. Christie Lee Dr. Dominique Piquette

POSTER JUDGES FOR ART SLUTSKY DAY

Basic Science Category I:	Dr. Haibo Zhang Dr. Aleksandra Leligdowicz
Basic Science Category II:	Dr. Andrew Baker Dr. John Marshall
Clinical Research/Health Services Research Category I:	Dr. Sangeeta Mehta Dr. Matteo Parotto
Clinical Research/Health Services Research Category II:	Dr. Alberto Goffi Dr. Victoria McCredie
Physiology Category I:	Ms. Vagia Campbell Dr. Darlene Reid
Physiology Category II:	Dr. Ewan Goligher Mr. Thomas Piraino
Quality Category:	Dr. Gail Annich Dr. Elizabeth Wilcox
Systematic Review/Case Report/Education Category I:	Dr. Michael Detsky Dr. Orla Smith
Systematic Review/Case Report/Education Category II:	Dr. Laveena Munshi Dr. Anne-Marie Guerguerian

ORAL PRESENTATION JUDGES

Dr. Laurent Brochard Dr. Marc Moss Dr. Margaret Herridge Dr. Hannah Wunsch

DIFFUSE AIRWAY CLOSURE AND SURFACTANT DEGRADATION BIOMARKER IN ACUTE RESPIRATORY DISTRESS SYNDROME

L Chen[1,2], C Lu[3], A Demoule[4], L Brochard[1,2]

1. Interdepartmental Division of Critical Care Medicine, University of Toronto, Toronto, Canada
2. Department of Critical Care, St Michael's Hospital, Keenan Research Centre and Li Ka Shing Knowledge Institute, Toronto, Canada
3. Department of Pediatric Intensive Care, Beijing Children's Hospital, Capital Medical University
4. Unité de Réanimation et de Surveillance continue, Groupe hospitalier Pitié-Salpêtrière, Paris, France

Introduction: "Atelectrauma" was proposed to describe the lung injury probably caused by cyclic closing and reopening of small distal airways and alveoli. Muscedere and his colleagues found in isolated, lavaged rat lungs ventilated with 6 mL/kg of tidal volume (1) that positive end-expiratory pressure (PEEP) below the so-called "lower inflection point" generated significant small airway and alveolar injury in histological examination. However, none of studies linked bronchiolar injury in vivo with airway closing-reopening phenomenon.

In a recent study (2), we found that around one third of patients with acute respiratory distress syndrome (ARDS) presented complete airway closure with an airway opening pressure (AOP) higher than 5 cmH_2O. By integrating results from other physiological studies, we speculated that complete airway closure could be caused by diffuse liquid segments in terminal airways. In the present study, we wanted to know whether the presence of diffuse airway closure is associated with biomarkers of surfactant degradation and collagen turn-over measured in bronchoalveolar lavage (BAL) fluid.

Methods: This is a retrospective analysis of a cohort of ARDS patients in whom were measured pressure-volume (P-V) curves and were collected BAL fluid within 24 hours (3). Patients had severe or moderate ARDS (80 mmHg ≤ PaO2/FiO2 ≤ 200 mmHg). All patients were ventilated with a 6 ml/kg of predicted body weight tidal volume. PEEP was titrated individually according to oxygenation, hemodynamics, and plateau pressure. Plateau pressure was kept at 30 cm H_2O or below.

Patients respiratory system P-V curves were measured by using the oscillating flow insufflation (4) starting at zero end-expiratory pressure (ZEEP). Tracheal pressure was estimated by subtracting resistive pressures during oscillated inspiratory flow from the total airway pressure. We used the tracheal pressure as an approximation of elastic pressure and constructed P-V curves to identify airway closure (Figure 1). The blocked circuit P-V curve was constructed using the compliance of ventilator circuit (1.7 ml/cmH_2O) obtained on a bench. AOP was defined as the tracheal pressure at which gas volume delivered to a patient became 8.5 ml greater than the volume compressed in an occluded circuit. A patient was defined as having diffuse airway closure if the AOP from ZEEP was greater than or equal to 5 cmH_2O.

After separating patients into two groups (patients with/without airway closure), we compared the following biomarkers: activity of type-IIA secreted phospholipase A2 (sPLA2), an enzyme involved in surfactant degradation; type III procollagen peptide (PCP III), a marker of collagen deposition; matrix metalloproteinase 2 (MMP-2), for collagen degradation; and tumor necrosis factor-alpha (TNF-α) for inflammation (3).

Results: All patients in the cohort (n=23) were involved in the current analysis. Average age was 55±17 years (mean ± standard deviation) and PaO_2/FiO_2 133±49 mmHg. They were ventilated with a PEEP of 8±2 cmH_2O. Eleven (48%) of them exhibited diffuse airway closure with AOP 9±5 cmH_2O (Figure 1). We compared general characteristics, respiratory parameters, and biomarkers between patients with or without airway closure (Table 1). sPLA2 activity was significantly higher in the presence of airway closure.

Conclusions: Our data suggest that diffuse airway closure is associated with an increase in sPLA2 activity in the BAL fluid– a biomarker of surfactant degradation. This supports the hypothesis that diffuse airway closure is caused by intra-bronchial liquid segment formation and an increase in surface tension.

References

1. Muscedere JG, Mullen JB, Gan K, Slutsky AS. Tidal ventilation at low airway pressures can augment lung injury. Am J Respir Crit Care Med 1994; 149: 1327-1334.
2. Chen L, Del Sorbo L, Grieco DL, Shklar O, Junhasavasdikul D, Telias I, Fan E, Brochard L. Airway Closure in Acute Respiratory Distress Syndrome: An Underestimated and Misinterpreted Phenomenon. Am J Respir Crit Care Med 2018; 197: 132-136.
3. Demoule A, Decailliot F, Jonson B, Christov C, Maitre B, Touqui L, Brochard L, Delclaux C. Relationship between pressure-volume curve and markers for collagen turn-over in early acute respiratory distress syndrome. Intensive Care Med 2006; 32: 413-420.
4. Jonson B, Richard JC, Straus C, Mancebo J, Lemaire F, Brochard L. Pressure-volume curves and compliance in acute lung injury: evidence of recruitment above the lower inflection point. Am J Respir Crit Care Med 1999; 159: 1172-1178.

Figure 1. Panel A is a representative patient who present complete airway closure during oscillating flow inflation. The blue line with circle marker denotes the patient's respiratory system pressure-volume curve and the red line with plus sign denotes the curve of the occluded ventilator circuit. Panel B is a representative patient who did not present complete airway closure. AOP = Airway Opening Pressure.

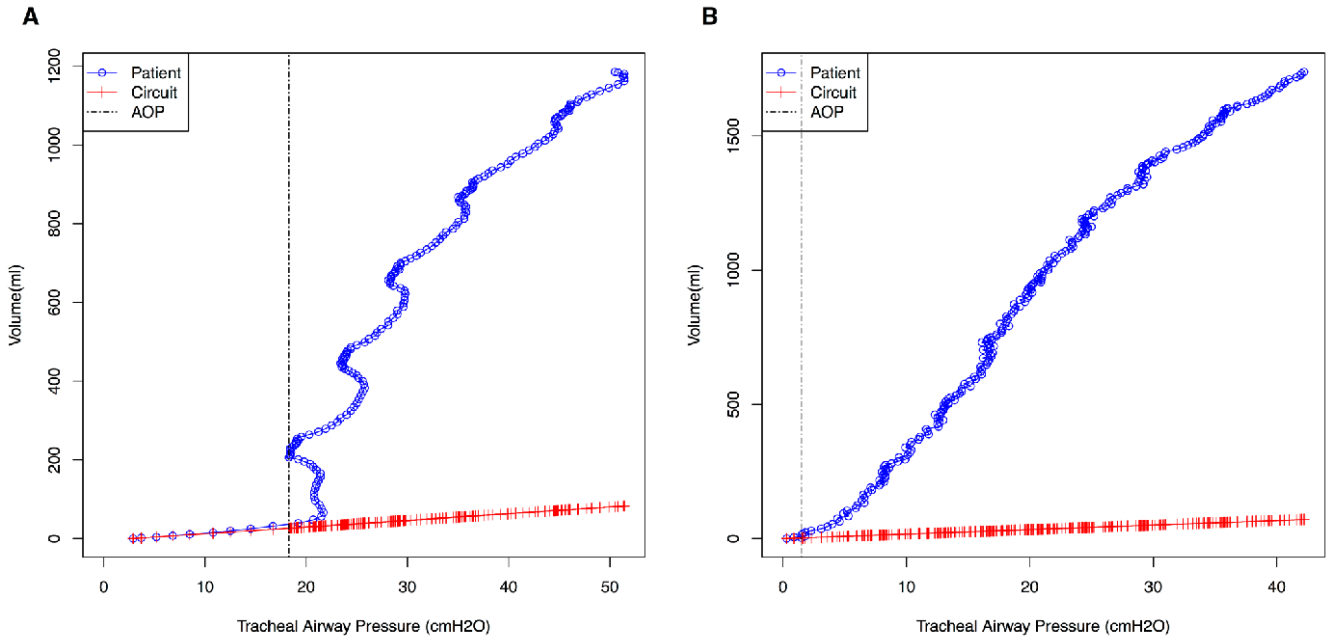


Table 1. Comparison of the main characteristics of patients with airway closure vs. patients without airway closure *

	Airway Closure (n =11)	**No Airway Closure (n=12)**	***P* Value**
General characteristics			
Age – years	62 [50-69]	54 [46-69]	0.667
BMI – kg/m^2	25 [22-31]	25 [21-26]	0.477
SAPS II	47 [45-71]	39 [30-60]	0.204
Respiratory parameters			
Clinical PEEP – cmH_2O	9 [8-10]	9 [8-10]	0.975
Crs at PEEP – ml/cmH_2O	32 [28-50]	34 [29-36]	0.689
$PaCO_2$ – mmHg	52 [46-64]	49 [42-57]	0.413
PaO_2/FiO_2 – mmHg	96 [81-152]	166 [109-183]	0.079
Biomarkers in BAL			
sPLA2 activity – (U/ml)	416 [28-1407]	21 [20-31]	**0.034**
PCP III – (U/ml)	0.38 [0.07-1.70]	0.14 [0.03-1.67]	0.456
MMP-2 – (ng/ml)	7.04 [4.43-14.64]	4.04 [0.34-8.29]	0.157
TNF-α – (ng/ml)	14 [0-35]	4 [0-67]	0.975

* All data were recorded at the day of performing BAL. Continuous variables were described as described as median [interquartile ranges] and compared using Mann-Whitney Test.

FACTORS REDUCING THE ACCURACY OF SEIZURE IDENTIFICATION USING QUANTITATIVE ELECTROENCEPHALOGRAPHY DISPLAYS IN THE INTENSIVE CARE UNIT

S Lalgudi Ganesan[1], CP Stewart[2], EG Atenafu[3], A Ochi[2], H Otsubo[2], C Go[2], CD Hahn[2]

1. Department of Critical Care Medicine, The Hospital for Sick Children, University of Toronto
2. Division of Neurology, Department of Paediatrics, The Hospital for Sick Children, University of Toronto
3. Department of Biostatistics, Princess Margaret Hospital, University Health Network, University of Toronto

Introduction & Objectives: Non-convulsive seizures are common in critically ill and are detectable only by continuous electroencephalography. Quantitative electroencephalography (QEEG) trending displays are being used increasingly commonly to facilitate seizure identification during such continuous EEG monitoring. Our previous work showed that the accuracy of seizure identification using QEEG is lower in some recordings, even for EEG experts. In this study, we sought to determine which seizure, EEG recording and patient characteristics make seizure identification on QEEG more difficult.

Methods: Three neurophysiologists were independently asked to mark all suspected seizures in 27 continuous EEG recordings from 22 critically ill children that were processed using two QEEG techniques: amplitude-integrated EEG (aEEG) and color density spectral array (CDSA). Seizures confirmed by a board-certified neurophysiologist on review of the raw EEG constituted the comparison standard. We analyzed the following factors for their influence on the probability of correct seizure identification: age, number of seizures per recording, seizure burden (per hour), seizure distribution and spectral characteristics of individual seizures such as power and frequency (both absolute and relative to the immediate pre-ictal background). Independent predictors of poor seizure detectability were identified using a multivariable correlation (Recording characteristics) and reduced regression modeling with autoregressive covariance (seizure characteristics).

Results: Sensitivity was higher in recordings from older children (CDSA>aEEG), and those with higher seizure count and higher seizure burden, for both CDSA and aEEG (figure 1). Seizure detectability declined with decreasing seizure duration (figure 2). Using CDSA, seizure identification rates were 65% for seizure durations of >180s, 62% for 60-179s, 44% for 30-59s and 16% for less than 30s. Using aEEG, seizure identification rates were 81% for seizures lasting >180s, 67% for 60-179s, 38% for 30-59s and 13% for less than 30s. Focal seizures were more difficult to detect on CDSA/aEEG (26%/33%) compared to both hemispheric seizures (44%/53%) and generalized seizures (69%/64%). With CDSA, independent predictors of poor seizure detectability were: younger age, lower number of seizures per recording, lower ictal/pre-ictal spectral edge frequency at 50% power, lower ictal/pre-ictal spectral edge frequency at 95% power, and lower ictal/pre-ictal average total power. With aEEG, independent predictors were the same except age and ictal/pre-ictal spectral edge frequency at 95% power, which were not in the final aEEG model.

Conclusions: Independent factors reducing seizure detectability were younger age (only CDSA), lower number of seizures per recording, shorter duration, focal distribution and a lower ratio of ictal-interictal power as well as the frequency at 50% and 95% power (only CDSA). We also demonstrate that the ratios of the spectral features of ictal and interictal periods are vital determinants of seizure detectability. These insights will inform the optimal use of these displays and aid the design of newer QEEG trends that enhance the contrast between ictal and interictal periods, to achieve more accurate seizure identification

Supported by: Canadian Institutes of Health Research (CIHR), Physician Services Incorporated (PSI) Foundation, RETSRACOMP/C-BMH Integrative Research Fellowship awards.

Figure 1. Impact of age, number of seizures per recording and seizure burden on sensitivity for seizure identification using QEEG

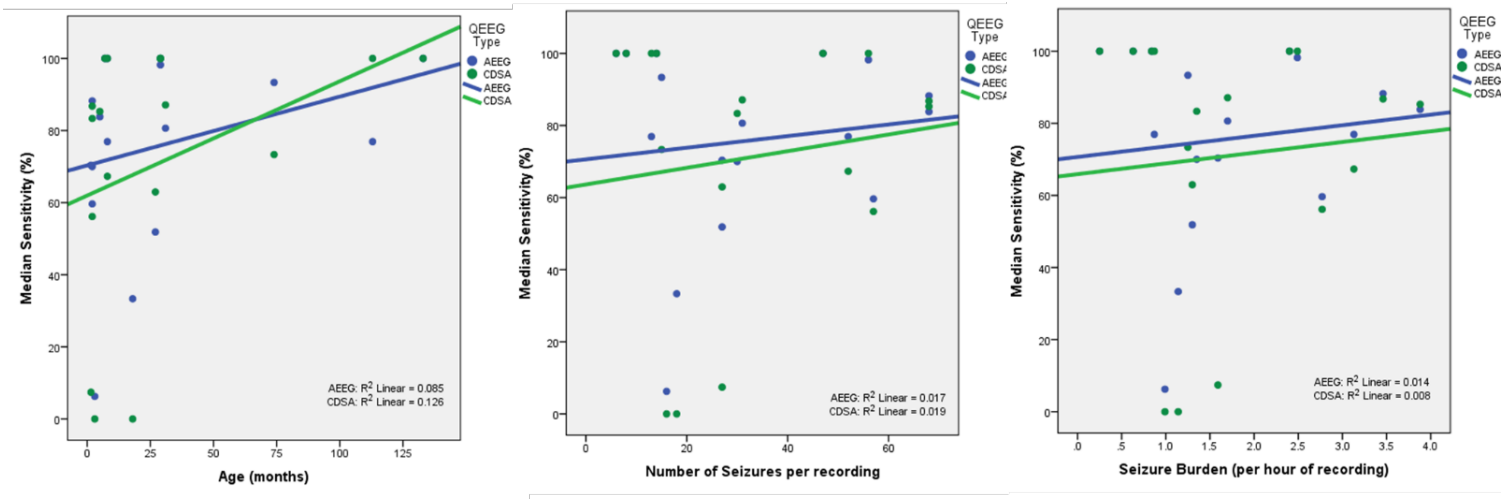
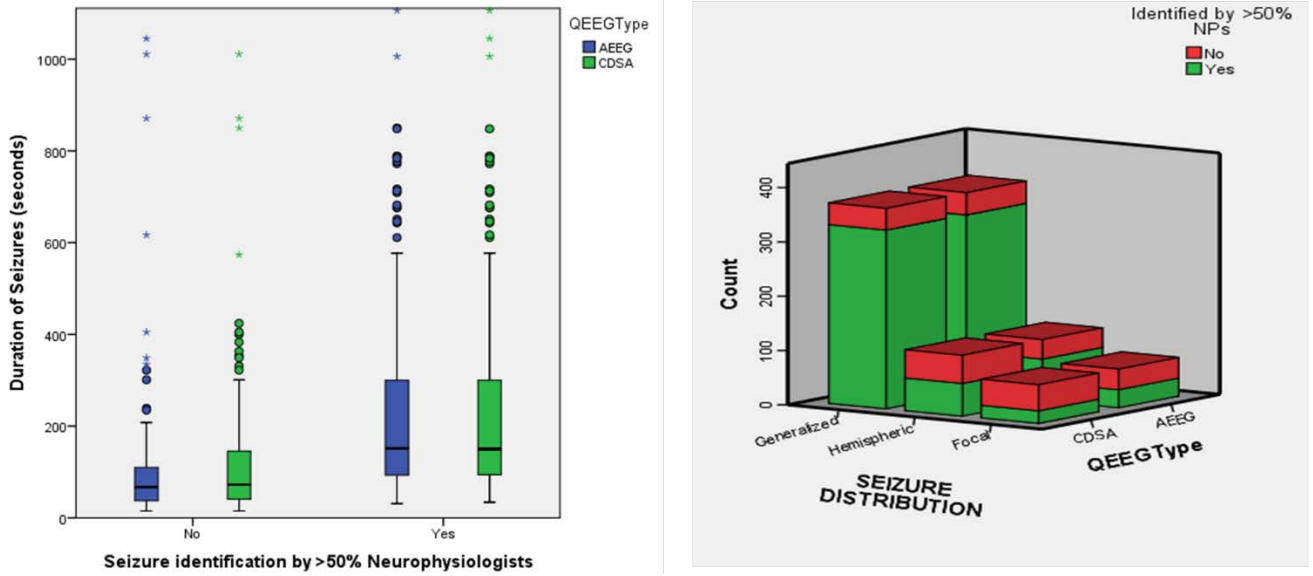


Figure 2. Comparison of seizure duration and distribution among seizures identified by less than 50% and more than 50% of neurophysiologists



NEEDS ASSESSMENT FOR MEDICAL ERRORS IN RESIDENCY TRAINING – A QUALITATIVE STUDY

East, JM.[1], Hawryluck, L.

1. Department of Critical Care, University of Toronto

Introduction: Medical error in practice and how physicians deal with the impact of these errors has become and emerging issue since the publication of *To Err is Human*[1]. Addressing medical errors, from disclosure to coping skills, has not been consistently taught in postgraduate training. Moreover little is known of trainees' perceptions of error events, their disclosure to patients and perceptions of the need for support. This qualitative study explored how medical errors were handled, mentorship of such events and the perceived educational and support needs of General Internal Medicine trainees at the University of Toronto in order to understand the lived experience and impact of medical errors in their clinical practice and gain insight regarding potential interventions to improve in dealing with such events.

Methods: Purposive sampling was used to recruit 7 GIM trainees at various levels of training for semi-structured interviews. Interviews were conducted in person by the PI (JE). The interview guide was based on research team's literature review on medical error and experience in clinical practice and training. Interviews were audio-recorded and transcribed verbatim. Grounded theory with concurrent thematic data analysis using NVIVO software was performed. Transcripts were coded inductively and iteratively. The transcripts were read and coded independently by two of investigators (JE and LH). The coding scheme was refined and expanded using the constant comparative method. Data collection ceased when conceptual saturation was achieved.

Results: Coding identified several themes. Emotional responses to error included detachment, guilt, blame, saving face, burnout and minimization of responsibility. Trainee perceptions of the error varied widely including variable reporting/disclosure based on perceived importance and impact of the event, supervising physician's response to the event, and professional reputation. Personal learning and professional development varied depending on ownership of responsibility, self-reflection and morbidity of the event. Short comings in current systemic support structures included ongoing culture of barriers to transparent discussion between staff and trainees, lack of appropriate forum for discussion of effects on sense of self and on clinical practice. Participants identified perceived benefits of error discussion. Themes surround risks of not seeking open discussion about error included burnout, anxiety, feelings of professional/social isolation and hiding errors in the future. To improve education/support around errors, participants identified small group discussions tailored to residents' needs. Characteristics of staff facilitators, necessity of time protection from clinical responsibility, ideal group make-up of participants and potential perceived harms were also identified as key to any successful future program development.

Conclusions: Overall, these themes illustrated that despite several interventions provided to support residents through training, there persists, at minimum, a perception of lack of support that could be contributing to burn out, anxiety and loss of potential learning opportunities for clinical practice and support network development. Small group facilitated discussions appear to meet several of the illustrated pitfalls of the current support network provided and appear well perceived by trainees. We believe that a pilot study should be conducted to further analyze the benefits and perceptions of such an intervention.

References

Institute of Medicine. 2000. *To Err is Human: Building a Safer Health System*. Washington, DC: The National Academies Press. https://doi.org/10.17226/9728

ABRUPT DEFLATION AFTER SUSTAINED INFLATION CAUSES LUNG INJURY

BH Katira[1,2,3], D Engelberts[1], G Otulakowski[1], RE Giesinger[4], T Yoshida[1,2,3], M Post[1], WM Kuebler[5,6,7], KA Connelly[5], BP Kavanagh[1,2,3]

Affiliations: *[1]Translational Medicine, The Research Institute, Hospital for Sick Children, Toronto, Canada; [2]Departments of Critical Care Medicine and Anesthesia, Hospital for Sick Children, University of Toronto, Toronto, Canada; [3]Interdepartmental Division of Critical Care Medicine, University of Toronto, Toronto, Canada; [4]Division of Neonatology, Department of Pediatrics, Hospital for Sick Children, University of Toronto, Toronto, Canada; [5]Keenan Research Centre for Biomedical Sciences, St. Michael's Hospital, Toronto, Canada; [6]Departments of Surgery and Physiology, University of Toronto, Toronto, Canada; Institute of Physiology, Charité–Universitätsmedizin Berlin, Germany.*

Introduction: Clinicians managing mechanical ventilation for patients with ARDS concentrate on 'setting' ventilator parameters, such tidal volume (V_T), PEEP and driving pressure; but, the circumstances surrounding abrupt disconnection of the ventilator (*e.g.* suctioning, circuit changes, transport) usually receive little consideration.

Hypothesis: To determine if abrupt deflation after sustained inflation causes lung injury beyond simple atelectasis.

Methods: Healthy Sprague Dawley rats were anaesthetised, mechanically ventilated (V_T 6 mL/kg), and randomized to: sustained inflation (increasing PEEP in increments of 3 cmH_2O over 70 min, up to 11 cmH_2O) followed by abrupt deflation to zero PEEP and continued ventilation for 30 min; or, to control ventilation (V_T 6 mL/kg, PEEP 3 cmH_2O) for 100 min. Respiratory compliance, oxygenation, lung wet-to-dry weight ratio, BAL protein, and histology were assessed. Additional experiments were performed to determine the timing of lung leakage (timed BAL performed following intravenous Evans Blue, to measure leakage of protein); the impact of gradual *vs*. rapid deflation; ultrastructural changes using scanning electron microscopy; hemodynamic assessment with echocardiography and cardiac catheterization (Millar catheter); and, the impact of LV afterload reduction using IV infusion of sodium nitroprusside (SNP).

Results: Abrupt deflation following sustained inflation caused acute lung injury (compliance 0.48±1.0 *vs*. 0.82±0.2 $mL \cdot cmH_20^{-1}$, SpO_2 67±23.5 *vs.* 91±4.4%, wet/dry ratio 6.1±0.6 *vs*. 4.6±0.4: $P<0.01$, *vs*. control). Vascular leak (Evans Blue detected in BAL) was absent before deflation and maximal by 10 min thereafter; ultrastructural injury was predominantly endothelial. At deflation, pulmonary blood flow, LV preload, systemic blood pressure and LVEDP increased precipitously. The rise in LVEDP was sufficient to cause capillary stress failure and was proportional to the degree of injury. Sodium nitroprusside prevented increases in LVEDP and prevented injury. Injury did not occur if deflation was gradual.

Conclusion: Abrupt deflation after sustained inflation caused acute lung injury that appeared to be mediated by acute LV decompensation, elevated pulmonary microvascular pressure and endothelial damage.

Significance: The significance of these findings is unknown. However, if demonstrated in patients, deflation injury may constitute an occult injury unnoticed during routine care and potentially contributing to negative results in RCTs of sustained inflation (*e.g.* PEEP, recruitment maneuvers, HFOV) in patients with ARDS.

Support: *Canadian Institutes of Health Research (BPK and KAC), Dr Geoffrey Barker Chair in Critical Care Research (BPK), Ministry of Research and Innovation and Science, Ontario (KAC), & Restracomp (BHK).*

ULTRASOUND AND MICROBUBBLES TO DELIVER THERAPY TO THE INJURED LUNG IN ARDS

V Mintsopoulos[1,2], M Sugiyama[2,3], A Goffi[1,4,5], A Slutsky[1,4], H Leong-Poi[1,4], R Karshafian[6] and WL Lee[1,2,3,4]

[1]Keenan Research Centre for Biomedical Sciences, St. Michael's Hospital, Toronto, Canada
[2]Institute of Medical Science, University of Toronto, Canada
[3]Department of Laboratory Medicine and Pathobiology, University of Toronto, Canada
[4]Department of Medicine, Interdepartmental Division of Critical Care and the Division of Respirology, Toronto, Canada
[5]University Health Network, Toronto, Canada
[6]Department of Physics, Ryerson University, Toronto, Canada

Introduction and Objectives: Acute respiratory distress syndrome (ARDS) is a common diagnosis in ICUs and has a mortality rate approaching 40%; most cases are due to bacterial or viral pneumonia. Leakage of the alveolar-capillary membrane leads to pulmonary edema and decreased lung compliance. Although frontal radiographs suggest diffuse disease, most patients exhibit heterogeneous injury on CT scans, with relatively normal-appearing areas abutting densely consolidated or collapsed regions. This heterogeneity makes treatment difficult. For example, mechanical ventilation preferentially inflates healthier regions causing over-distension and lung damage; similarly, inhaled drugs cannot be targeted to the most injured areas. Parenterally-administered drugs go to all organs, predisposing to off-target effects. Targeting therapy to the injured regions of the lung is one of the most intractable problems in critical care.

Ultrasound-destruction of microbubbles (USMB) has been demonstrated to increase drug uptake by tissues, whether the drug is encapsulated by the microbubbles or merely in their vicinity. This has never been attempted in the lung since air scatters ultrasound energy. We reasoned that the heterogeneous injury of ARDS would allow USMB to preferentially deliver drugs to injured lung areas, since ultrasound will only penetrate fluid-filled or collapsed lung regions. Furthermore, the microbubbles themselves may accumulate in injured regions due to vascular leakage.

To test this hypothesis, we developed small and large animal models of lung injury. We first sought to use USMB to enhance delivery of the aminoglycoside antibiotic gentamicin to the lung in *E. coli*-induced ARDS. While most gram-negative isolates remain sensitive to aminoglycosides, their use is discouraged by clinical societies because of poor bioavailability in the lung and because even therapeutic doses are associated with nephro- and oto-toxicity. Next, to establish feasibility in a large animal and to establish generalizability of the technique, we developed a model of porcine pneumonia and tested whether USMB can enhance delivery of gentamicin or genetic material to the injured lung.

Methods: Male C57BL/6 mice were infected intratracheally with $5X10^7$ colony forming units (CFU) of *E. coli* and developed hypoxemia and lung edema. Six hours post infection (hpi), 1.5mg/kg of gentamicin was given intraperitoneally followed 30-min later by $1X10^9$ Definity™ microbubbles via tail vein and administration of thoracic ultrasound for 5 min. 30 min later we measured gentamicin levels in the bronchoalveolar lavage fluid (BALF) and lung homogenates; in other mice, lungs were homogenized 2 h after ultrasound to measure bacterial colonies.

For the large-animal model, female pigs (25-35kg) were administered $1X10^{11}$ CFU of *E. coli* into the right middle lobe (RML) via bronchoscope; the same animal's uninfected left upper lobe (LUL) served as an internal control. 24 hpi, pigs were administered 5 mg/kg of gentamicin and Definity microbubbles ($7.5X10^9$) IV simultaneously with thoracic ultrasound for 10 min over the RML; BAL was performed 30 min later in the RML and the LUL of each animal. In separate pigs, in lieu of antibiotics, Alexa-fluor 555-(fluorophore)-tagged RNA

was charged-coupled to microbubbles and administered IV during thoracic ultrasound. After sacrifice, lungs were analyzed by fluorescent microscopy to measure fluorophore-tagged RNA deposition in the RML versus the LUL.

Results: In both infected and control animals, oxygen saturation levels were not affected by USMB acutely or for 48 hours afterwards, establishing safety. Bacterial growth was reduced almost 10-fold in mice that received gentamicin and USMB compared to controls (PBS alone; gentamicin alone; gentamicin and microbubbles; ultrasound and microbubbles alone) ($p=0.001$ by ANOVA, $p<0.05$ or 0.01 by *post-hoc* t-tests). USMB caused a 2-fold increase in gentamicin levels in the BALF and lung homogenates ($p<0.05$) compared to controls that received only gentamicin. We observed a similar increase in gentamicin in BAL from the right middle lobe of infected pigs compared to the same animal's uninjured left upper lobe. Finally, our pig model also demonstrated enhanced deposition of fluorophore-tagged RNA in the RML compared to the same animal's LUL.

Conclusion: USMB significantly enhanced delivery of drugs and miRNA to the injured lung in two *in vivo* models of pneumonia. In the gentamicin model, this permitted a ten-fold increase in bacterial killing at a low systemic dose of gentamicin. Feasibility in humans is suggested by the porcine model. USMB may therefore allow for targeted delivery of therapies – antibiotics, antivirals, genes – to the injured lung in ARDS.

Supported By: CIHR-NSERC CHRP grant, NSERC Engage, OCE-VIP1, iBEST, and a Canada Research Chair to WLL.

ETHICAL DECISIONS IN PATIENTS WITH SEVERE ACUTE RESPIRATORY FAILURE - DETERMINING FACTORS, TIME OF DECISION AND IMPACT ON OUTCOME: THE EDECIDE PROJECT. AN ANCILLARY ANALYSIS OF LUNG SAFE STUDY

Tai Pham[1,2]*, Marie Berleur[3]*, Jean-Damien Ricard[3,4], Masatoshi Uchida[5], Taylor Thompson[6], Jonathan Messika[3,4], Didier Dreyfuss[3,4], Damien Roux[3,4] for the LUNG SAFE investigators.

**equally contributed to this study*

[1]University of Toronto, Interdepartmental Division of Critical Care, Toronto, Canada
[2]Li Ka Shing Knowledge Institute, Keenan Research Centre for Biomedical Science, St. Michael's Hospital,Toronto, Canada
[3]Assistance Publique des Höpitaux de Paris, Intensive Care Unit, Colombes, France
[4]Paris Diderot University, INSERM UMR 1137 IAME, Paris, France
[5]Dokkyo medical University, Department of Emergency and Critical Care Medicine, Tochigi, Japan
[6]Massachussett General Hospital, Department of Medicine, Division of Pulmonary and Critical Care, Boston, USA

Introduction and Objectives: Withholding and withdrawing life-sustaining therapy (LST) are complex ethical decisions made in many critically ill patients. End of life decisions (EOL) are part of intensive care daily practice and they may be influenced by many factors beyond patient's characteristics and severity. We aimed to describe EOL decisions in patients with acute respiratory failureand their impact on patients' prognosis.

Methods: The centers participating to the international observational LUNG SAFE study included all patients with acute hypoxemic respiratory failure over a one-month period. More than 450 ICU from 50 countries were involved in the project. Demographic, clinical and biological data were compared between patients with and without decision of LST limitation. We also compared LST decisions according to the model of physician/patient relationships. Countries were classified in directed physician decisions or patient autonomy based on published data. This classification was validated by countries coordinators.

Results: A total of 4041 hypoxemic patients were included. Overall mortality was 37.2%. A decision of withholding or withdrawing LST was reported in 948 patients (23.4%). Mortality in patients with EOL decisions was 84.4% (N=800).
In bivariate analysis, patients with a decision of LST limitation were older, had a lower body weight, a higher SOFA score, were more frequently hospitalized for a medical condition; they also presented more frequent active neoplasia/immunosuppression or chronic liver failure ($p<0.001$ for all). Patients admitted for non-cardiogenic shock had more LST limitation decision ($p=0.018$). Patients admitted after trauma, drug overdose or pulmonary contusion were less likely to have a LST limitation decision ($p<0.001$). EOL decisions were less frequent in lower-middle income countries as compared to high and middle-high income countries ($p<0.001$) and less frequent in countries with physician directed relationship model than in countries with patient autonomy model.
Almost half of the 1503 patients who died during their hospital staydid not receive a decision of LST limitation (46.8%, N=703).

Conclusion: Decisions of LST limitation are frequent in the ICU, concerning almost one quarter of hypoxemic patients. Not only patients' characteristics but also health system (patient-physician relationship, gross domestic product) seem to impact these decisions. Almost half of the patients who eventually died in hospital had not been the subject of a decision of LST limitation.

Supported By: ESICM trials group

References

Bellani G, Laffey JG, Pham T, Fan E, Brochard L, Esteban A, et al. Epidemiology, patterns of care, and mortality for patients with acute respiratory distress syndrome in intensive care units in 50 countries. JAMA. 2016;315:788–800.

Characteristics	No limitation N=3093	Limitation **decision N=948**	**p-value**
Age, year	60.1±16.9	66.8±15.2	<0.001
Weight, kg	78.7±24.0	74.2±20.4	<0.001
SOFA score	9.0±4.0	10.2±4.3	<0.001
Active neoplasia	242 (7.8%)	121 (12.8%)	<0.001
Immunosuppression	550 (17.8%)	258 (27.2%)	<0.001
Chronic cardiac failure	298 (9.6%)	120 (12.7%)	0.009
Chronic liver failure	95 (3.1%)	65 (6.9%)	<0.001
Trauma	159 (5.1%)	18 (1.9%)	<0.001
Non cardiogenic shock	205 (6.6%)	85 (9.0%)	0.018
Overdose	70 (2.3%)	9 (0.9%)	0.015
lower-middle income country	124 (4.0%)	14 (1.5%)	0.015
Physician directed relationship	1390 (44.9%)	367 (38.7%)	<0.001

Table: Characteristics of patients with Acute Hypoxemic Respiratory Failure according to the decision of limiting LST. Data are shon as mean ±SD or N(%)

ORAL PRESENTATION 7

AN *IN SITU* NEUROCRITICAL CARE SIMULATION CURRICULUM TO FOSTER ONGOING ACQUISITION OF KNOWLEDGE AND CRISIS RESOURCE MANAGEMENT SKILLS WITHIN THE INTERPROFESSIONAL ICU TEAM

R Sinyi[1], V McCredie[1,2], B Giammarioli[1,2], J Singh[1,2], A Goffi[1,2]

[1] *Toronto Western Hospital MSNICU, University Health Network*
[2] *Interdepartmental Division of Critical Care Medicine, University of Toronto*

Introduction & Objective: Simulation has become an integral part of medical education and the fidelity of the simulation appears to play an important role. Both the physical (i.e., the manikin and the environment) and psychological (i.e., scenario design and emotional response) dimensions of fidelity are essential components of a successful learning experience. Simulation conducted in the actual clinical environment (*in situ* simulation) facilitates participation from multiple disciplines and may promote enhanced engagement and facilitate increased knowledge uptake. Our objective was to create an *in situ* neurocritical care simulation curriculum at Toronto Western Hospital MSNICU to foster ongoing acquisition of knowledge and crisis resource management (CRM) skills within the interprofessional ICU team.

Methods: We developed a series of acute neurocritical care scenarios (e.g., seizures, subarachnoid hemorrhage, elevated intracranial hypertension, traumatic brain injury, ischemic stroke, difficult airway in a comatose patient) focusing on key knowledge objectives and essential components of CRM skills, such as effective communication, situational awareness, resource utilization, problem-solving and leadership. Each scenario requires participation of multiple professions (nurses, respiratory therapists, physicians), and is followed by a debrief to allow participants to reflect on personal and team performance, provide feedback on areas of strength and weakness, and highlight opportunities for improvement. We created a pre-curriculum survey and a post-simulation session self-reflective form to capture needs, knowledge and CRM skills uptake. At the end of the curriculum implementation we are planning a post-intervention survey.

Results: Over the past year we conducted 12 simulation sessions. 47 nurses completed the pre-curriculum survey (55.3% with less than 9 years of experience and 44.7% with more than 10 years of clinical experience). Although the vast majority of the participants felt prepared and comfortable in managing most of the common neurocritical care emergencies, a significant percentage only felt somewhat or not comfortable in the management of acute ischemic stroke (19%), meningitis/encephalitis (11%), post-operative oncological neurosurgical patients (11%), traumatic brain injury (7%), acute non-traumatic weakness (8.5%) and spinal cord injuries (8.5%). Poor communication with the team and lack of support were considered important factors that negatively impact the management of neurocritically ill patients. The quality of the teamwork was considered poor by 13%, acceptable by 22%, good by 28% and very good by 35% of the nurses. The majority of participants (65%) felt workload distribution and appropriate delegation were effectively undertaken. We collected 35 post-simulation self-reflective forms (22 nurses, 8 respiratory therapists, 1 resident, 3 others), with 88% of the respondents reporting the acquisition of new knowledge and 79% felt to have improved their teamwork skills. Several CRM skills deficiencies were identified, mostly related to the domains of communication, situational awareness, resource utilization and anticipation of future needs.

Conclusion: Our neurocritical care simulation curriculum seems to be an effective way to improve knowledge, confidence and CRM skills, especially communication, situational awareness and resource utilization/anticipation, for the entire interdisciplinary ICU team. Although time consuming and labor-intensive, *in situ* simulation appears to be an excellent training tool, promoting learners' engagement and knowledge uptake and recall. We are planning to expand the curriculum to non-neurocritical care emergencies.

Supported By: *TWH Krembil Awards*

ORAL PRESENTATION 8

MULTI-COHORT TRANSCRIPTIONAL META-ANALYSIS OF MUSCLE DISEASES IDENTIFIES A GENE SIGNATURE OF DISEASE SEVERITY

CJ Walsh[1,2], J Batt[1,2], M.S. Herridge[4], S. Mathur[5], GD Bader[6], P Hu[7], P Khatri[8,9], and CC. dos Santos[1,3]
Submitted on behalf of the MEND-ICU Group and Canadian Critical Care Translational Biology Group

1. Keenan and Li Ka Shing Knowledge Institute of Saint Michael's Hospital, Toronto, Ontario, Canada.
2. Institute of Medical Sciences and Department of Medicine, University of Toronto, Toronto, Ontario, Canada.
3. Interdepartmental Division of Critical Care, University of Toronto, Toronto, Ontario, Canada
4. University Health Network, Interdepartmental Division of Critical Care, University of Toronto, Toronto, Ontario, Canada
5. Department of Physical Therapy, University of Toronto, Toronto, Ontario, Canada
6. The Donnelly Center, University of Toronto, Toronto, Ontario, Canada
7. Department of Biochemistry and Medical Genetics, University of Manitoba, Winnipeg, Manitoba, Canada
8. Stanford University School of Medicine, Stanford,CA, USA
9. Stanford Institute for Immunity, Transplantation and Infection (ITI), Stanford Center for Biomedical Informatics Research (BMIR), Department of Medicine, Stanford University, Stanford, CA, USA

Introduction and Objective: Muscle diseases share common pathological features suggesting common underlying mechanisms. We hypothesized there is a common set of genes dysregulated across muscle diseases compared to healthy muscle and that these genes correlate with severity of muscle disease. We performed meta-analysis of transcriptional profiles from muscle biopsies of human muscle diseases and healthy controls.

Methods: Studies obtained from public microarray repositories fulfilling quality criteria were divided into six categories: i) Immobility, ii) inflammatory myopathies, iii) ICU acquired weakness (ICUAW), iv) congenital muscle diseases, v) chronic systemic diseases, vi) motor neuron disease. Patient cohorts were separated in discovery and validation cohorts retaining roughly equal proportions of samples for the disease categories. To remove bias towards a specific muscle disease category we repeated the meta-analysis five times by removing data sets corresponding to one disease class at a time in a "leave-one-disease-out analysis".

Results: Using 490 muscle tissue samples from 22 cohorts we identified 86 up-regulated and 33 down-regulated genes which were similarly dysregulated in 17 addition patient cohorts (689 samples) including five categories of muscle diseases. This gene signature inversely correlated with muscle mass in ICUAW ($r = -0.70$, $p=8.62\times10^{-6}$) and correlated with functional independence score ($r=-0.54$, $p=1.67\times10^{-3}$). The signature also correlated with histologic assessment of muscle atrophy in ALS ($r=0.84$, $p = 5.10\times10^{-3}$) and inversely correlated with shoulder abduction strength ($r=-0.74$, $p=0.022$) in ALS. Finally, removing the genes common to muscle disease from meta-analysis of only ICUAW cohorts revealed uniquely down-regulated muscle development and contraction genes specific to ICUAW.

Conclusions: Our results identify a conserved muscle disease transcriptional signature associated with clinical and histologic disease severity which may have future applications as a biomarker. ICUAW-specific gene dysregulation affecting muscle contraction and regeneration may be important therapeutic targets.

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BAYESIAN ANALYSIS OF THE QUICK SEQUENTIAL ORGAN FAILURE ASSESSMENT (QSOFA SCORE) TO PREDICT INTENSIVE CARE UNIT ADMISSION AND MORTALITY AMONG PATIENTS WITH INFECTIONS TRANSPORTED BY PARAMEDICS

D Lane [1], R Saskin[1,2], D Scales[1,2,3,4]

[1.] *Institute of Health Policy, Management and Evaluation, Dalla Lana School of Public Health, University of Toronto*
[2.] *Institute of Clinical Evaluative Sciences, University Health Network, Toronto, Ontario, Canada*
[3.] *Department of Critical Care Medicine, Sunnybrook Health Sciences Centre, Ontario, Canada*
[4.] *Interdepartmental Division of Critical Care, University of Toronto, Ontario, Canada*

Introduction: The quick Sequential Organ Failure Assessment (qSOFA) score was developed to provide clinicians with a *quick* assessment to identify patients with latent organ failure possibly consistent with sepsis, or patients with infections at high-risk for mortality. This study uses a Bayesian analysis to assess the prediction of Intensive Care Unit (ICU) admission or in-hospital mortality using the qSOFA score among patients transported by paramedics.

Methods: A one-year cohort of adult patients transported by paramedics in a large, provincial EMS system was linked to Emergency Department (ED) and in-hospital administrative databases, then patients were restricted to those with ED diagnosed infection. The primary outcomes of interest were direct 1) ICU admission or ED death, or 2) in-hospital mortality. A Bayesian binomial regression model was constructed for each outcome using Hamiltonian Markov-Chain Monte-Carlo sampling, with clinical characteristics of the qSOFA score dichotomized per the original thresholds (respiratory rate greater than 21, Glasgow Coma Scale less than 15, systolic blood pressure 100 mmHg or less), and uninformative priors. Discrimination was assessed using posterior predictions to calculate a “Bayesian” C statistic, and calibration was assessed with calibration plots of observed and predicted probabilities for each outcome.

Results: A total of 9,920 patients with ED diagnosed infection were included. 251 (2.5%) patients were admitted directly to the ICU or died in the ED, and 890 (9.0%) patients died in-hospital. The C statistic for predicting ICU admission or ED death was 0.798; however, the calibration curve had significant overlap of predictions across all levels of the qSOFA score (Figure 1). For predicting in-hospital mortality, the C statistic was 0.728 and the calibration curve had no overlap of predictions (Figure 2), with a predicted mortality of 0.5 (IQR 0.47-0.53) for patients with a qSOFA score of 3.

Conclusion: The qSOFA score is limited for predicting ICU admission or ED death, but is good for predicting in-hospital mortality of patients with ED diagnosed infection.

Figure 1: Calibration for qSOFA score predicting direct ICU admission or ED death

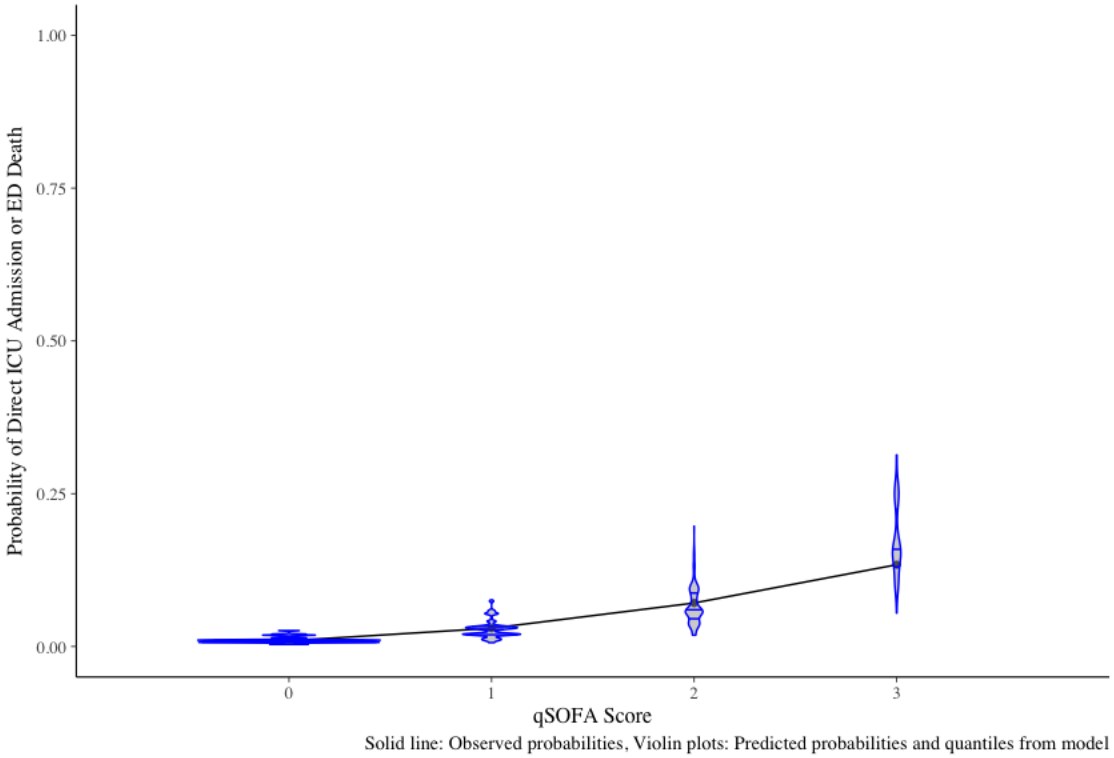
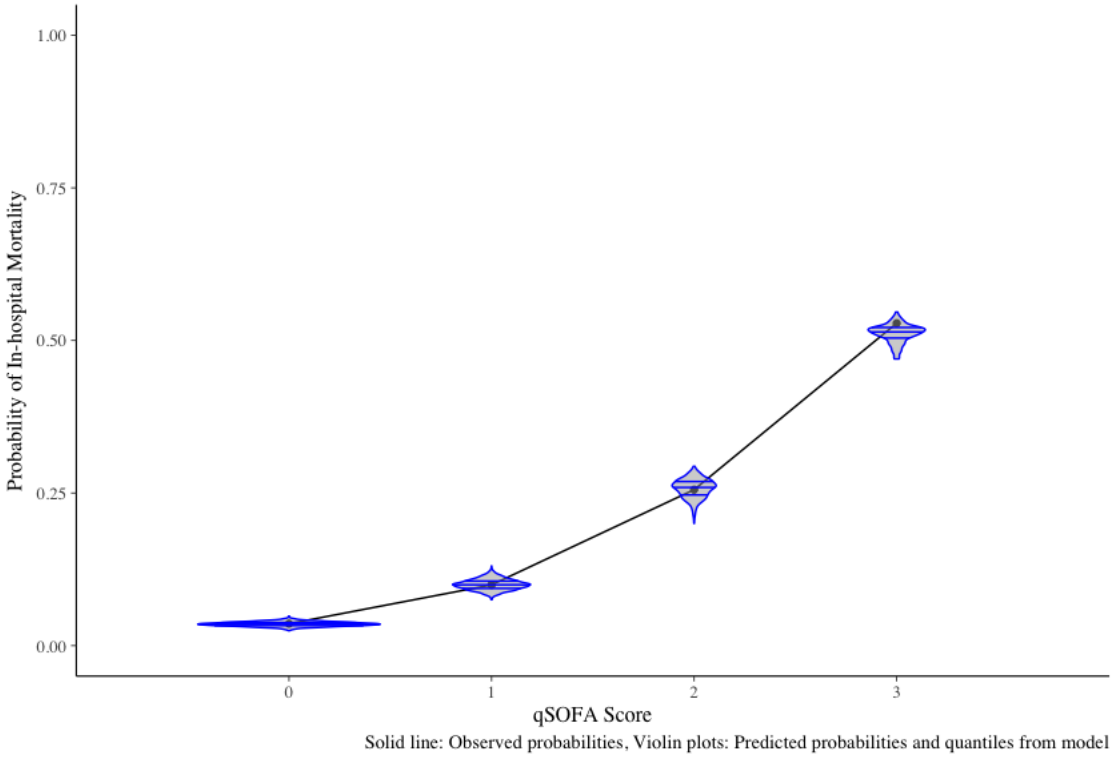


Figure 2: Calibration for qSOFA score predicting in-hospital mortality



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