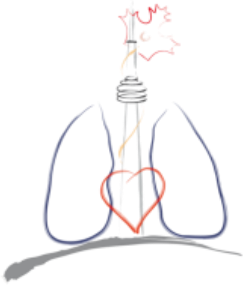
UNIVERSITY OF TORONTO | Interdepartmental Division of Critical Care Medicine



ART SLUTSKY RESEARCH DAY

Interdepartmental Division of Critical Care Medicine
University of Toronto

Tuesday, June 20, 2017

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**

Chair: Dr. Hannah Wunsch

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. A. Mohamed
The Impact of High Flow Nasal Oxygen in the Immunocompromised Critically Ill: A Systematic Review and Meta Analysis

9:15 – 9:30 am **Presentation 2:** Dr. M. Hamilton
Determinants of Depressive Symptoms at One Year after Intensive Care Unit Discharge in Survivors ≥ 7 Days of Mechanical Ventilation: Results from the RECOVER Program

9:30 – 9:45 am **Presentation 3:** Dr. L. Kolodzey (to be presented by S. Pinkney)
Human Factors in Paediatric Critical Care

IDCCM Faculty Presentations I

Session Chair: Dr. Peter Laussen

9:45 – 10:00 am **Featured Speaker:** Dr. Eddy Fan
Topic: Extracorporeal Support for Respiratory Failure in Toronto and Beyond

10:00 – 10:15 am **Featured Speaker:** Dr. Andre Amaral
Topic: A brief (personal) story of Quality Improvement

10:15 - 11:00 am Morning Break

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

Chair: Dr. Margaret Herridge

11:00 – 11:15 am **Presentation 4:** Dr. H. Raheel
Permeability to Albumin in Skin Microvasculature is Regulated by CD36 - Potential Role in Resolving Tissue Edema and Drug Delivery

11:15 – 11:30 am **Presentation 5:** Dr. D. Rozenberg
Clinical Implications of Pre-Transplant Skeletal Muscle Mass, Strength and Exercise Capacity on Functional Recovery in Lung Transplant Recipients after 7 or More Days of Mechanical Ventilation

11:30 – 11:45 am **Presentation 6:** Dr. T. Yoshida
Esophageal Manometry in Acute Respiratory Distress Syndrome (ARDS) – Safer Expiration, Safer Inspiration

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. Margaret Herridge and Hannah Wunsch

1:30 – 1:45 pm **Presentation 7:** Dr. A. Varkouhi
Therapeutic Efficacy of Human Umbilical Cord Mesenchymal Stem Cell-Derived Extracellular Vesicles in Pre-Clinical Model of Acute Respiratory Distress Syndrome

1:45 – 2:00 pm **Presentation 8:** Dr. B. Katira
Cardiogenic Edema Contributes to a Classic Model of Ventilator-Induced Lung Injury

2:00 – 2:15 pm **Presentation 9:** Dr. J. Viau-Lapointe
Outcomes Reported in Studies on Mechanical Ventilation in Critically Ill Pregnant and Postpartum Patients: A Systematic Review.

IDCCM Faculty Presentations II

Session Chair: Dr. Andrew Baker

2:15 – 2:30 pm	**Featured Speaker:** Dr. Gail Annich **Topic:** The Future of Circuitry and Anticoagulation in ECLS
2:30 – 2:45 pm	**Featured Speaker:** Dr. Robert Fowler **Topic:** TBC
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 – 4:15 pm	**Featured Speaker** **Daniel Talmor, MD, MPH** Edward Lowenstein Professor and Chair Department of Anesthesia, Critical Care, and Pain Medicine Beth Israel Deaconess Medical Center and Harvard Medical School **Title:** Pulmonary Physiology at the Bedside *or* How Art Slutsky Made Me Ignorant
4:15 – 5:15 pm	**Presentation of Certificates of Training Completion** **Presentation of Site Teaching Awards** **Presentation of the John Granton and Simon Abramson Teaching Awards** Dr. Dave Hall
5:15 – 5:30 pm	**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 2017
IDCCM Chair: Dr. Laurent Brochard
IDCCM Director of Research: Dr. Margaret Herridge
IDCCM Associate Director of Research: Dr. Hannah Wunsch

GUEST SPEAKERS

Featured Speaker: Daniel Talmor, MD, MPH

Daniel Talmor, MD, MPH, is Chair of the Department of Anesthesia and Critical Care at Beth Israel Deaconess Medical Center (BIDMC) and the Edward Lowenstein Professor of Anesthesia at Harvard Medical School.

Dr. Talmor received his medical degree from Ben Gurion University in Beer-Sheva, Israel. He followed this with a residency in anesthesia at the Soroka Medical Center, also in Beer-Sheva. In 1999 he came to BIDMC, where he completed fellowships in critical care medicine and cardiac anesthesia. He then earned his master's in public health from Harvard School of Public Health.

Dr. Talmor has been an anesthetist at BIDMC since 2001. His research interests are centered on the early identification and treatment of critically ill patients, with a particular focus on the optimal delivery of mechanical ventilation.

Dr. Talmor currently serves as the principal investigator (PI) on two National Heart, Lung, and Blood Institute (NHLBI)-funded studies, one of which is a phase 2 clinical trial testing a novel mode of mechanical ventilation in patients with acute lung injury. The other is a phase 2 clinical trial testing aspirin as a potential treatment to prevent acute lung injury in at-risk patients. He is also the site investigator for the NHLBI network for the Prevention and Treatment of Acute Lung Injury (PETAL).

Dr. Talmor has published over 150 peer reviewed papers as well as multiple chapters, editorials and abstracts. He has lectured extensively around the world.

Faculty Speakers

Dr. Andre Carlos Amaral

The focus of Dr. Amaral's research is on improving quality of care for critically ill patients. Dr. Amaral works within two streams to improve quality: communication, to prevent errors and improve efficiency; and quality assurance, which involves measuring quality and developing novel models for implementing evidence-based medicine.

His work in communication focuses on transitions of care, a necessary, but fragile link in the health care system, and on teamwork during interdisciplinary rounds.

His work in implementation science involves a novel collaboration across Ontario for large-scale quality improvement in a partnership of community and academic intensive care units.

Dr. Gail Annich

In my practice as a Pediatric Intensivist at the Hospital for Sick Children, the use of extracorporeal devices either temporary or permanent, and acute or chronic has proven to be challenging to manage, as these devices and supports are most frequently required for use in the most critically ill, high-risk patients. The blood/biomaterial barrier has not yet been conquered despite many breakthroughs in the biomaterial world so that anticoagulation remains a requirement of these therapies.

My research focuses on this biologic interface and our multidisciplinary group that spans across the fields of medicine, bioengineering, surgery and chemistry does this work. This research group has been successful in stepping towards a solution that will change the risks and improve the outcomes of patients who require such extraordinary supports to live and recover.

Dr. Eddy Fan

Dr. Eddy Fan is an Assistant Professor in the Interdepartmental Division of Critical Care Medicine and the Institute of Health Policy, Management and Evaluation at the University of Toronto and a Staff Intensivist at the University Health Network/Mount Sinai Hospital.

Dr. Fan received his undergraduate degree from the University of Toronto, his medical degree from the University of Western Ontario and a PhD in Clinical Investigation from Johns Hopkins University.

He is currently the Medical Director of the Extracorporeal Life Support Program at the Toronto General Hospital, and the Director of Critical Care Research at the University Health Network/Mount Sinai Hospital.

Dr. Fan's research has focused on advanced life support for acute respiratory failure and patient outcomes from critical illness. These include investigations on the epidemiology and use of mechanical ventilation and extracorporeal life support in patients with ARDS, as well as on the development of ICU-acquired weakness, early rehabilitation in ICU patients, and long-term outcomes in survivors of critical illness.

Dr. Robert Fowler

Dr. Rob Fowler is originally from the Canadian Maritimes, completed medical school at McGill University, Internal Medicine Residency at the University of Toronto and a Critical Care Medicine fellowship and Clinical Epidemiology graduate degree at Stanford University.

He is a critical care physician at Sunnybrook Hospital, the Director for the Dalla Lana School of Public Health's Institute of Health Policy, Management and Evaluation Clinical Epidemiology Program, an Adjunct Scientist at the Ontario Institute for Clinical Evaluative Sciences and a Clinician-Scientist for the Heart and Stroke Foundation.

Dr. Rob Fowler's clinical and academic interests include the access and outcomes of care and end-of-life care for critically ill patients, in Canada and internationally, He has also been involved in clinical care and clinical research of patients during infectious outbreaks in Canada and other regions, including SARS, Influenza A(H1N1), Middle East Respiratory Syndrome, and the 2014 West Africa Ebola outbreak.

ABSTRACT JUDGES

Basic Science Category	Dr. Bilal Ansari Dr. Claudia Dos Santos
Clinical Research/Health Services Research Category	Dr. Victoria McCredie Dr. Geeta Mehta Dr. Orla Smith
Physiology Category	Dr. Ewan Goligher Dr. Max Meineri Dr. Darlene Reid
Quality Category	Dr. Alberto Goffi Dr. John Granton Dr. Sara Gray
Systematic Review/Case Report/Education Category	Dr. Robert Fowler Dr. Mjaye Mazwi Dr. Dominique Piquette

MODERATORS FOR ART SLUTSKY DAY

Basic Science Category I:	Dr. Jane Batt Dr. Haibo Zhang
Basic Science Category II:	Dr. John Laffey
Clinical Research/Health Services Research Category I:	Dr. Matteo Parotto Dr. Damon Scales
Clinical Research/Health Services Research Category II:	Dr. Laveena Munshi TBC
Physiology Category:	Ms. Vagia Campbell Dr. Lorenzo Del Sorbo
Quality Category:	Dr. Jeffrey Singh Dr. Elizabeth Wilcox
Systematic Review/Case Report/Education Category	Dr. Neil Adhikari Dr. Michael Detsky

ORAL PRESENTATION JUDGES

Dr. Laurent Brochard
Dr. Margaret Herridge
Dr. Daniel Talmor
Dr. Hannah Wunsch

THE IMPACT OF HIGH FLOW NASAL OXYGEN IN THE IMMUNOCOMPROMISED CRITICALLY ILL: A SYSTEMATIC REVIEW AND META ANALYSIS

A Mohamed MD, MC. Sklar MD, A Orchanian-Cheff BA, MISt, LD MD, S Mehta MD, L Munshi MD, MSc

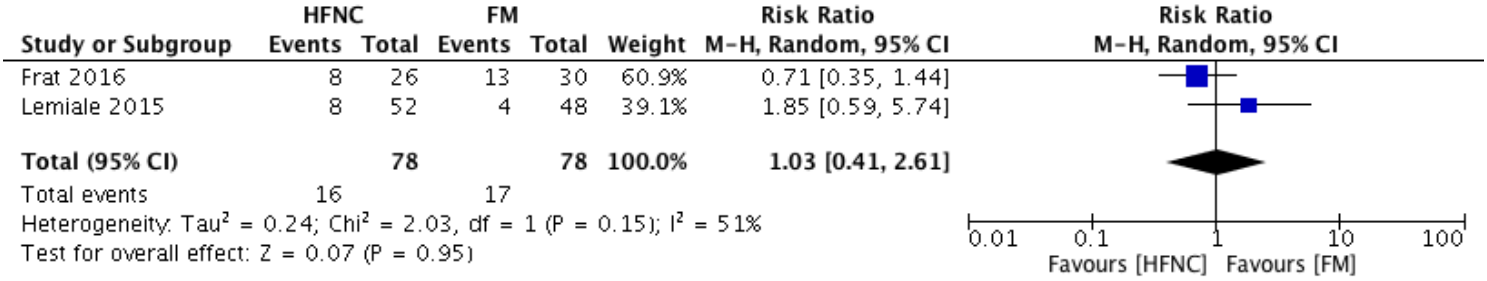
Background: Mortality in immunocompromised patients with respiratory failure presenting to the intensive care unit (ICU) remains high. Those requiring invasive mechanical ventilation are subject to an increased mortality risk. High-flow nasal-cannula has been proposed to be a superior modality of oxygen support and may have a role in preventing invasive mechanical ventilation; however, its role in this population is not clearly defined.

Objectives: To evaluate the effectiveness of high-flow nasal-cannula compared to other modalities of oxygen support (face mask or non-invasive ventilation) in immunocompromised patients with acute respiratory failure
Methods: We systematically searched the major medical databases to identify all publications reporting the use of HFNC. We included any observational studies describing the use of HFNC in IC patients and randomized controlled trials (RCTs) that further compared HFNC to either face mask (FM) oxygen or non-invasive ventilation (NIV) amongst IC patients. We looked at clinical and physiological efficacy across this population and meta-analyzed rates of invasive mechanical ventilation (IMV) and respiratory rate from the RCTs.

Results: Of 3099 studies reviewed, 6 met our inclusion criteria. We included 3 observational studies and 3 randomized controlled trials (428 patients). Hematologic malignancy was the leading etiology of immunosuppression (60%) and pneumonia the leading cause of respiratory failure (52%). Thirty-four percent of high-flow nasal-cannula patients subsequently required invasive mechanical ventilation. Two randomized trials compared high-flow nasal-cannula to face mask, demonstrating no difference in rates of invasive mechanical ventilation (Risk ratio (RR) 1.03, 95% CI 0.41-2.61, I^2=52%). High-flow nasal-cannula compared to non-invasive ventilation (1 randomized trial and 1 observational study with propensity-score matching) was associated with a decreased need for invasive mechanical ventilation (RR 0.54, 95% CI 0.35-0.84, I^2=0%) and with reduced intensive care unit mortality (RR 0.40, 95% CI 0.22-0.70, I^2 = 0%). One possible explanation for this finding of benefit of high flow nasal cannula compared specifically to non-invasive ventilation but not face mask, could be the pressure levels generated in non-invasive compared to pressure transmitted via high flow nasal cannula. During non-invasive ventilation, patients may generate tidal volumes that are above those considered lung protective (>8ml/kg).

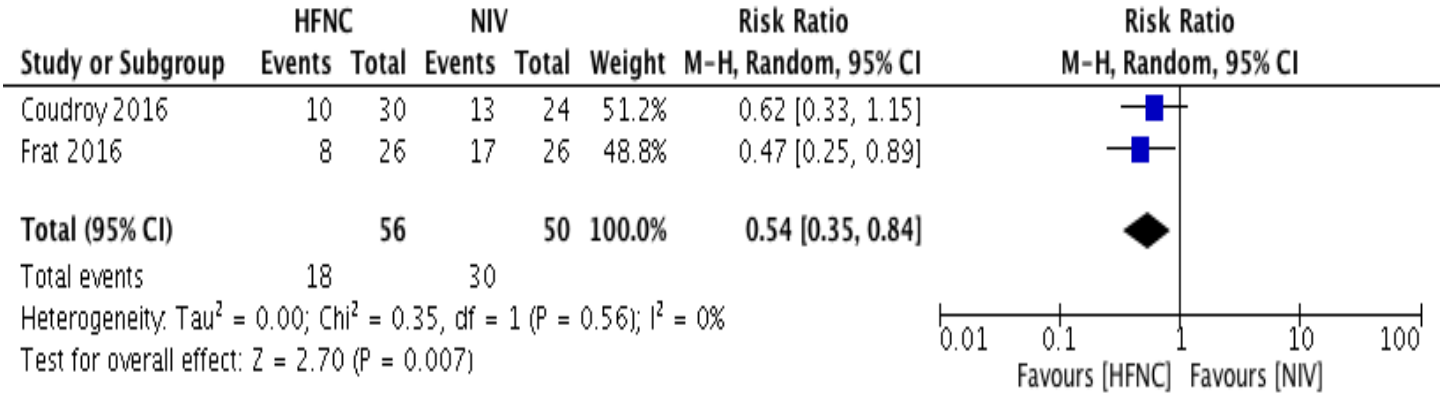
Conclusions: There is a limited body of evidence to guide oxygen therapy in hypoxemic immunocompromised patients. The current studies suggest that high-flow nasal-cannula may be associated with reduced need for invasive mechanical ventilation and improved survival when compared to non-invasive ventilation but not when compared to face mask oxygen. The data to date is intriguing but limited and these findings are meant to be hypothesis generating. More research in supportive oxygen therapies are needed for this population.

The Effect of HFNC vs FM on the need for IMV



These two RCTs (156 patients) evaluated the application of HFNC vs FM, demonstrating no difference in the rates of IMV (Risk ratio (RR) 1.03, 95% confidence interval (CI) 0.41-2.61, p=0.95, I^2=52%).

The Effect of HFNC vs NIV on the need for IMV



HFNC was compared to NIV (1 RCT and 1 observational study with propensity matching, 106 patients), it was associated with a decrease in need for IMV (RR 0.54, 95% CI 0.35-0.84, p=0.007, I^2=0%).

DETERMINANTS OF DEPRESSIVE SYMPTOMS AT ONE YEAR AFTER INTENSIVE CARE UNIT DISCHARGE IN SURVIVORS OF ≥ 7 DAYS OF MECHANICAL VENTILATION: RESULTS FROM THE RECOVER PROGRAM

M Hamilton[1], G Tomlinson[2], L Chu[2], P Robles[2,3], A Matte[2], S Burns[2], C Thomas[2], F Lamontagne[4], N Adhikari[1,5], N Ferguson[1,2], J Freidrich[1,6], J Rudkowski[4,7], Y Skrobik[8], H Meggison[9], J Cameron[3,10,11], M Herridge[1,2] for the RECOVER Program Investigators and the Canadian Critical Care Trials Group.

[1]Interdepartmental Division of Critical Care, University of Toronto, Toronto, Canada [2]Department of Medicine, Interdepartmental Division of Critical Care Medicine, Toronto General Research Institute, Institute of Medical Science, University Health Network, University of Toronto, Toronto, Canada [3]Graduate Department of Rehabilitation Science, University of Toronto, Toronto, Canada [4]Centre de recherché du CHU de Sherbrooke, Université de Sherbrooke, Sherbrooke, Canada [5]Department of Critical Care Medicine, Sunnybrook Health Sciences Centre, Toronto, Canada [6]Departments of Medicine and Critical Care Medicine, St. Michael's Hospital, University of Toronto, Toronto, Canada [7]Departments of General Internal Medicine and Critical Care, St. Joseph's Healthcare, McMaster University, Hamilton, Canada [8]Department of Medicine Division of Critical Care, Maisonneuve Rosemont Hospital, University of Montreal, Montreal, Canada [9]Department of Critical Care, University of Ottawa, Ottawa, Canada [10]Department of Occupational Science and Occupational Therapy, University of Toronto, Toronto, Canada [11]Toronto Rehabilitation Institute, University of Toronto, Toronto, Canada

Background: Moderate to severe depressive symptoms occur in 20-33% of survivors at one year after Intensive Care Unit (ICU) discharge and may have a negative impact on functional outcomes. The extent to which patient and caregiver factors impact the development of depressive symptoms remains unclear.

Objective: We sought to evaluate patient and caregiver factors associated with the development of post-ICU depressive symptoms.

Methods: Using the RECOVER Program cohort of 391 medical/surgical ICU patients, we investigated factors associated with depressive symptoms captured by the Beck Depression Inventory-II (BDI-II) score. We determined the association between BDI-II and baseline characteristics (age, sex, socioeconomic status, Charlson Comorbidity Index (CCI) score and ICU length of stay (LOS)), Functional Independence Measure (FIM) motor subscale score and caregiver characteristics (Caregiver Assistance Scale, Center for Epidemiologic Studies Depression Scale and caregiver BDI-II) using linear regression and linear mixed models at the 3, 6 and 12-month time points. All analyses were preplanned.

Results: BDI-II data was available for 246 patients. Median age at ICU admission was 54 years, 58% were male (n=143) and median LOS was 19 days. Mean BDI-II scores at 3, 6 and 12 months were 12.3, 11.4 and 10.7. Across all time points nearly 17% of patients had a BDI-II score ≥21, indicating moderate-severe depressive symptoms. At each time point, a lower FIM motor subscale score was significantly associated with more severe depressive symptoms. At 6 and 12 months, age had a curvilinear relationship to BDI-II, with the highest depressive symptoms associated with ages 45-50. Over the 12 months, mixed models found more severe depressive symptoms in patients with lower FIM motor subscale scores, those who had not completed secondary education, and the same curvilinear relationship to age. No statistically significant associations were found between patient BDI-II and patient CCI, sex, LOS, income or caregiver variables.

Conclusions: Severity of post-ICU depressive symptoms is associated with less education, greater functional dependency, and has a curvilinear relationship with age. No associations between caregiver variables and patient outcome were identified. Knowledge of risk factors for clinically important depressive symptoms may inform surveillance and targeted mental health follow-up after critical illness. Rehabilitation programs aiming to improve the FIM may serve to modify mood disorders.

Supported By: CIHR, MOH-AFP, John and Gail MacNaughton Family fund, Don and Jane Luck Family fund, Jason Tham and Andrea Chan family fund.

HUMAN FACTORS IN PAEDIATRIC CRITICAL CARE

L Kolodzey[1], S Pinkney[2], M Fan[1], P Sutton[3], A McCormick[3], AM Guerguerian[3,4], P Laussen[3,4], P Trbovich[1,2,5]
[1] Department of Research and Innovation, North York General Hospital, [2] Techna Institute, University Health Network, [3] Department of Critical Care Medicine, The Hospital for Sick Children, [4]Department of Paediatrics, University of Toronto, [5] Institute of Health Policy, Management and Evaluation, University of Toronto

To be presented by: Sonia Pinkney

Introduction & Objective: Paediatric Critical Care is a dynamic and complex sociotechnical work system wherein considerable opportunity for preventable patient harm exists. With a view to improving patient safety at the Hospital for Sick Children (SickKids), the department of Critical Care Medicine launched a collaboration with HumanEra, a human factors research team, to uncover work system factors with the potential to impact patient safety. The objectives of the first phase of this multi-phase collaboration were to: (1) contextualize the critical care environment, and (2) identify a priority research area to be investigated in more depth.

Methods: This observational quality-improvement study utilized naturalistic observation in combination with informal interviews to collect holistic, descriptive, text-based data regarding both patient safety threats and elements of resilience in paediatric critical care. Four human factors researchers observed clinicians at work in both the cardiac and medical-surgical critical care units from November 2016 to March 2017 through approximately 188 hours of shadowing. Observed personnel included staff physicians, fellows, nurses (including charge nurses, clinical support nurses, and nurse practitioners), respiratory therapists, pharmacists, dietitians, and patient service aides. Observation data were subsequently subjected to a multi-framework human factors analysis enabling the characterization of priority clinical activities, implicated work system elements, and the degree and appropriateness of observed behavioural variability.

Results: Through the analysis of over 700 distinct observation notes, 3 clinical activities were highlighted as particularly suitable for a more in-depth human factors analysis: medication processes (ordering, preparation, and administration), surgical/sterile procedures, and rounds. Opportunities for safety improvement as well as areas of resilience were documented. Overall, patient care rounds emerged as a priority research area warranting further study; observed issues included the variable content of the patient report and the inconsistent participation of key care team members, while an example of resilience included the use of continuous monitoring software on rounds to clarify uncertainties regarding historical patient vitals.

Conclusion: This study highlighted actionable opportunities for systems-based safety improvement in multiple facets of paediatric critical care, particularly in relation to patient care rounds. The next phase of the collaboration will thus comprise an REB-approved research study to inform, implement, and evaluate evidence-based interventions to improve rounding efficiency and effectiveness, as well as to reinforce existing successful practices.

PERMEABILITY TO ALBUMIN IN SKIN MICROVASCULATURE IS REGULATED BY CD36 - POTENTIAL ROLE IN RESOLVING TISSUE EDEMA AND DRUG DELIVERY

Hira Raheel[1,2,6], Michael Sugiyama[3, 6], Maria Febbraio[4], Bryan Heit[5], and Warren L. Lee[1-3, 6]
[1]Institute of Medical Science, [2]Cardiovascular Sciences Collaborative Program, [3]Laboratory Medicine and Pathobiology, University of Toronto, [4]Faculty of Medicine and Dentistry, University of Alberta, [5]Department of Microbiology and Immunology, The University of Western Ontario, [6]Interdepartmental Division of Critical Care,
Keenan Research in the Li Ka Shing Knowledge Institute, St. Michael's Hospital

Introduction: Albumin is the most abundant serum protein and its accumulation in the interstitial space leads to tissue edema, a hallmark of sepsis in critical care patients. Under homeostatic conditions the movement of albumin from the vascular lumen to the interstitial space is thought to occur through **transcytosis**, which requires the endocytosis of macromolecules at the luminal surface of an endothelial cell, followed by trafficking to the basolateral membrane and subsequent exocytosis. Despite its physiologic importance, little is known regarding the mechanisms of albumin transcytosis by the vascular endothelium. Our primary objective is to delineate the kinetics and molecular mechanisms of albumin transcytosis by microvascular endothelial cells (MECs) derived from both human lung and skin, two organs that classically manifest edema. We hypothesized that endothelial cells from the microvessels of the skin and lung exhibit different propensities for albumin transcytosis. Delineating the mechanisms of albumin transcytosis is essential for developing strategies to resolve tissue edema and to potentially harness albumin as a tissue-specific drug carrier for a variety of diseases, including diabetes and cancer.

Methods: Albumin transcytosis by primary human lung and dermal MECs was studied by total internal reflection fluorescence (TIRF) microscopy (Armstrong et al, 2015) and transwell assays. For the former, fluorescent albumin was added to the apical surface of confluent monolayers and TIRF was used to record exocytosis at the basal membrane as quantified in a blinded fashion by a Matlab algorithm. The role of the scavenger receptor CD36 was investigated by both molecular and pharmacologic approaches. To study the involvement of CD36 *in vivo,* we obtained endothelial specific CD36 knockout mice and measured basal permeability of the vasculature to albumin using a modified Evans Blue assay.

Results: Transcytosis of fluorophore-tagged albumin by dermal MECs was saturable and partially inhibited by unlabelled albumin. In contrast, transcytosis by lung MECs was not out-competed by unlabelled ligand. Treatment of lung MECs with amiloride, an inhibitor of macropinocytosis, showed 50% reduction in albumin transcytosis, but amiloride had no effect on dermal MECs. These data suggested that albumin transcytosis is receptor-mediated in dermal MECs. We next investigated if the scavenger receptor CD36 may be the receptor for albumin transcytosis in dermal MECs. Over-expression of CD36 in dermal endothelial cells increased albumin endocytosis by more than 2-fold, while treatment with SSO, a CD36 inhibitor, decreased transcytosis of albumin by 50% in dermal MECs but had no effect on lung endothelial cells. Importantly, endothelial-specific *CD36*$^{-/-}$ mice exhibited a 50% reduction in basal permeability to albumin in the skin but not the lung compared to wild-type mice. These endothelial-specific *CD36-deficient* mice also had significantly lower wet-to-dry ratios of the skin than wild-type controls (indicating less tissue edema), while their lungs were indistinguishable.

Conclusion: Tissue edema is a defining feature of critical illness, such as sepsis and Acute Respiratory Distress Syndrome (ARDS). Loss of albumin from blood vessels and its accumulation in the interstitial space leads to edema formation. A reversal of albumin transcytosis may mitigate the development of edema or accelerate its resolution. We have found tissue-specific differences in the endothelial transcytosis of albumin. The receptor CD36 appears to have an essential role in mediating albumin internalization and transcytosis in dermal microvascular endothelial cells both in vitro and in vivo. Conversely, albumin transcytosis in pulmonary endothelium seems to largely occur independently of a receptor, primarily through macropinocytosis. An understanding of the mechanisms through which albumin reaches various tissue sites is essential for the development of therapies to resolve tissue edema. Finally, albumin is being actively investigated as a natural drug carrier; understanding how it is delivered to the tissues will therefore be essential.

Supported By: Natural Sciences and Engineering Research Council of Canada (NSERC) and a Canada Research Chair to WLL.

CLINICAL IMPLICATIONS OF PRE-TRANSPLANT SKELETAL MUSCLE MASS, STRENGTH AND EXERCISE CAPACITY ON FUNCTIONAL RECOVERY IN LUNG TRANSPLANT RECIPIENTS AFTER 7 OR MORE DAYS OF MECHANICAL VENTILATION

D Rozenberg,[1,2] S Mathur,[3] G Tomlinson,[1,4] R Goldstein,[1] P Robles,[4] LM Chu,[4] C Chaparro,[1,2] B Andrade,[2] S Burns,[4] C Thomas,[4] JI Cameron,[4] MS Herridge,[1,4] LG Singer[1,2] In collaboration with the RECOVER Program Investigators.

[1] Department of Medicine, Respirology, University of Toronto; [2]Toronto Lung Transplant Program, UHN; [3] Department of Physical Therapy, University of Toronto; [4]Interdepartmental Division of Critical Care Medicine,UHN –Toronto, ON/Canada

Introduction and Objectives: Lung transplant patients are at risk of peri-operative complications that result in a prolonged course of mechanical ventilation (MV). Unlike other intensive care unit (ICU) patients, all lung transplant recipients undergo rehabilitation prior to their transplant and ICU stay with the goal of optimizing functional recovery after transplantation. We hypothesized that greater pre-transplant skeletal muscle mass, strength and exercise capacity would be associated with better post-transplant functional recovery in lung transplant recipients requiring at least 7 days of MV.

Methods: Single center analysis of lung transplant recipients who required ≥ 7 days of MV peri-operatively and participated in a prospective cohort study (RECOVER) evaluating post-ICU recovery. Pre-transplant skeletal muscle mass (using thoracic computed tomography cross-sectional area), strength (captured with muscle training volumes) and exercise capacity (six-minute walk distance, 6MWD) were evaluated retrospectively. Post-transplant outcomes were the Functional Independence Measure motor subscale (motor FIM, primary outcome at 7 days post-ICU) and secondary outcomes included the Short-Form 36 Physical Component Score (SF-36 PCS), 6MWD and discharge disposition at 3 months post-ICU. Pre-transplant predictors of recovery (age, sex, ICU length of stay (LOS), muscle mass, strength and 6MWD) were identified using multivariable regression.

Results: 80 lung transplant recipients (48% male; median age 57 IQR [44-64] and Lung Allocation Score of 38 IQR [35-44] at time of transplant) were included. 72 (90%) recipients survived to ICU discharge with a total median ICU LOS of 24 IQR [17-36] days. At 7-days post-ICU, these participants required maximal-total assistance (motor FIM; 33 (SD) ± 19), with age being the only independent predictor of motor FIM (β= -5.2 ± 1.7, per 10 years). At three-months post-ICU, lung transplant recipients had persistent functional limitations: motor FIM (80 ± 17, minimal assistance), low 6MWD (63 ± 23% predicted) and SF-36 PCS (35 ± 10) and high rates of discharge to inpatient rehabilitation (43%) versus home. At three-months post-ICU, younger age and shorter ICU LOS were the only independent predictors of better post-transplant motor FIM, greater SF-36 PCS, and a higher likelihood of being discharged home, whereas pre-transplant muscle mass, strength, and 6MWD were not significant.

Conclusion: In a select group of lung transplant recipients surviving 7 or more days of MV, age and ICU LOS were significant determinants of early functional recovery and discharge disposition. The lack of association between pre-transplant skeletal muscle function and post-transplant outcomes highlights the importance of ICU acquired morbidity in this group of patients. These findings may help with discharge planning and facilitate discussions between the healthcare team, patients, and their families about the clinical implications of ≥ 7 days of MV peri-operatively.

Supported By: DR receives salary support from the University of Toronto, Clinician Scientist Training Program and Canadian Institutes of Health Research (CIHR) Vanier Graduate Scholarship.

The RECOVER Program was supported by CIHR, Ministry of Health Innovation Award, University of Toronto Integration Challenge Fund; John and Gail Macnaughton Family Fund, Don and Jane Luck Family Fund and Jason Tham and Andrea Chan Family Fund.

Esophageal Manometry in Acute Respiratory Distress Syndrome (ARDS) – Safer Expiration, Safer Inspiration

T Yoshida[1], MBP Amato[2], DL Greco[3], JC Richard[4], C Lima[2], R Roldan[2], C Morais[2], S Gomes[2], L Brochard[3], BP Kavanagh[1]

[1] Hospital for Sick Children, University of Toronto, [2] Universidade de São Paulo, Brazil, [3] St. Michael's Hospital, University of Toronto, [4] General Hospital of Annecy, France

Introduction: Ventilator-induced lung injury in ARDS occurs mostly in ventilated, non-dependent lung regions. Esophageal manometry (esophageal-pressure, P_{es}) is a classical technique that estimates pleural pressure (P_{pl}) enabling calculation of the transpulmonary pressure (P_L), the difference between airway and P_{pl}, that distends the lung. However, its validity has been questioned. P_{pl} differs with lung region (higher in dorsal and lower in ventral when supine) and P_{es} might be increased by mediastinal weight; thus, it is uncertain in which lung regions esophageal manometry reflects P_L.

We addressed this issue using two approaches: human cadavers, and anesthetized pigs. We compared P_{es} with directly measured P_{pl} in dependent and non-dependent lung, and determined in which regions P_L is reflected by esophageal manometry.

Methods: Human cadavers with preserved elasticity (n=3; Thiel, CER-14-201-08-03.17) and anesthetized pigs with surfactant depletion (n=6; Yorkshire, 40.1±2.9 Kg) were monitored by standard esophageal balloons, while regional P_{pl} was directly measured by surgically inserted pleural sensors (wafer-type, flat-balloon) in non-dependent and dependent pleural spaces. Across a range of PEEP values, regional expiratory P_L was calculated (airway pressure - local P_{pl}; dependent or non-dependent), or by using absolute P_{es}. Regional inspiratory P_L were similarly calculated, and were also estimated using tidal changes in P_{es} (*i.e.* elastance-derived method).

Results: In human cadavers and pigs, expiratory or inspiratory P_L using absolute P_{es} were close to values estimated from dependent pleural sensors, or were intermediate between dependent and non-dependent sensors (adjacent to the esophageal balloon) (Figure 1-A, B). However, inspiratory P_L calculated from tidal changes in P_{es} closely reflected the estimates derived from non-dependent sensors (Figure 2-A, B).

Conclusions: Absolute P_{es} in expiration accurately reflects P_{pl} in the dependent to middle lung regions where atelectasis usually predominates. Thus, using expiratory P_{es} to prevent atelectasis makes sense, and might determine best 'expiration pressure'. The inspiratory change (not absolute value) in P_{es} enables estimation of the local P_{pl} in the non-dependent lung where is the most susceptible to stress during inspiration, and its local P_L may be a more specific marker of risk from ventilation-induced injury than global parameters such as plateau pressure, tidal volume or driving pressure; this might determine best 'inspiratory pressures'.

Figure 1

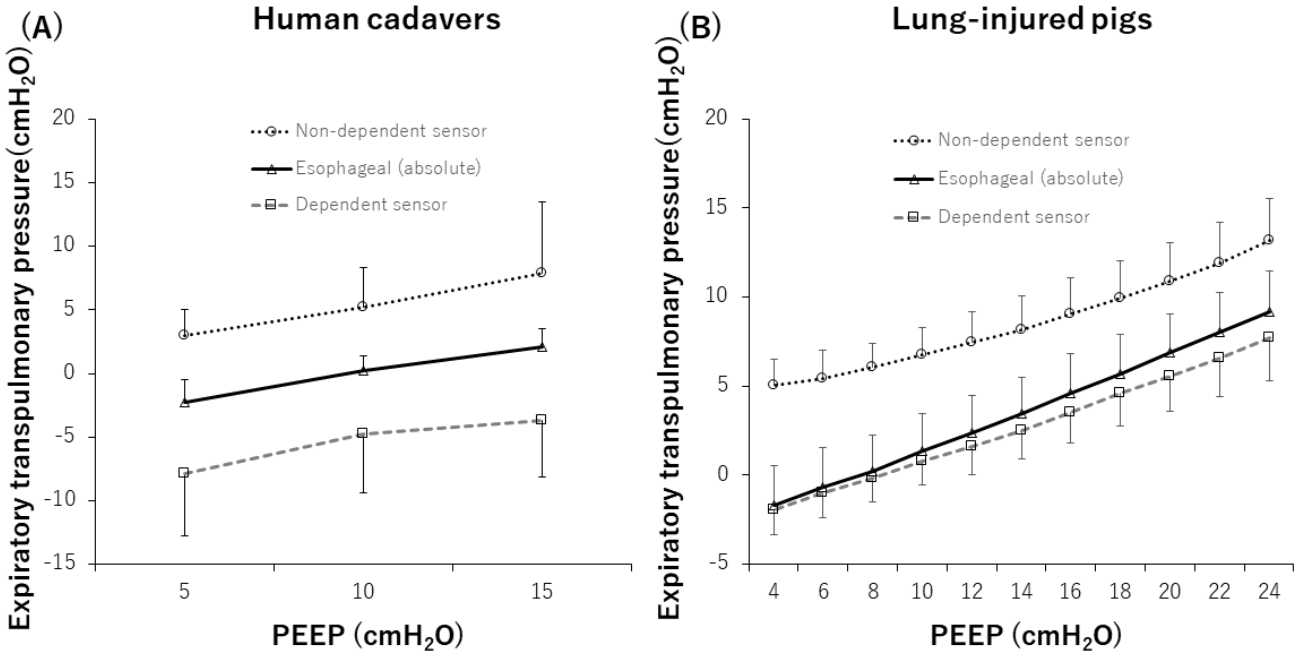
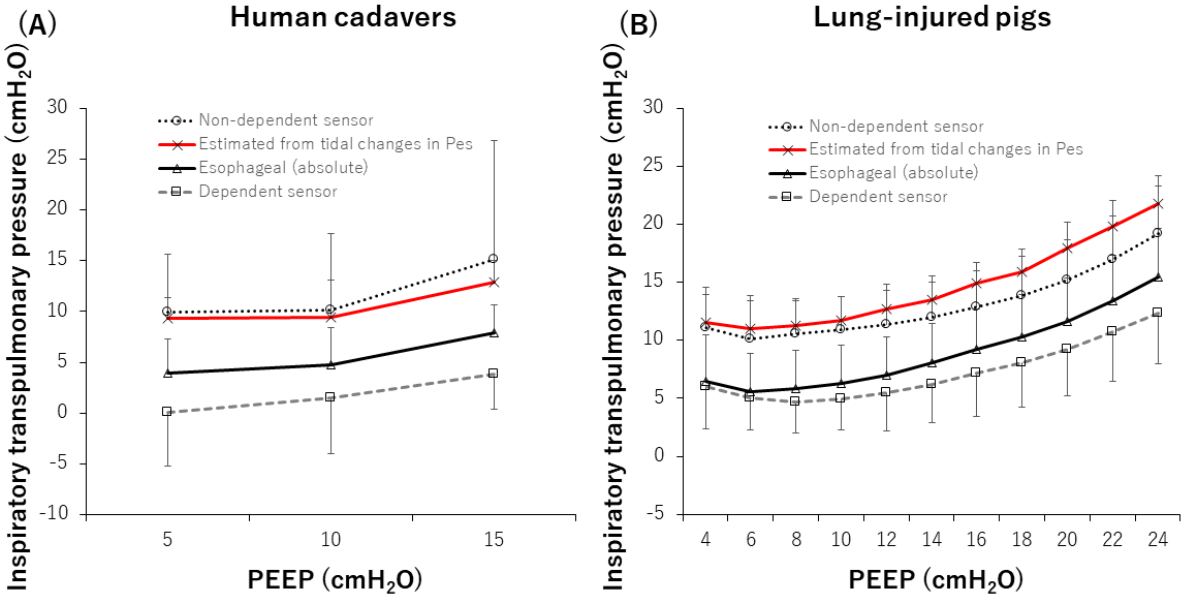


Figure 2



THERAPEUTIC EFFICACY OF HUMAN UMBILICAL CORD MESENCHYMAL STEM CELL-DERIVED EXTRACELLULAR VESICLES IN PRE-CLINICAL MODEL OF ACUTE RESPIRATORY DISTRESS SYNDROME

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Introduction & Objectives: Acute Respiratory Distress Syndrome (ARDS) is a devastating clinical condition, resulting in life threatening acute hypoxic respiratory failure and a 40% risk of death. Over the last decades many novel therapeutics have been evaluated for the treatment of ARDS yet none have proven efficacious, and thus supportive care strategies remain the mainstays of therapy. Mesenchymal Stem Cells (MSCs) are multi-potent cells with strong immunomodulatory properties that constitute an innovative approach with substantial therapeutic promise for ARDS. MSCs release extracellular vesicles (EVs) containing bioactive molecules, such as proteins, mRNAs, microRNAs and lipids, which mediate the cross-talk between MSCs and immune cells. It was reported that EVs could recapitulate the therapeutic effects of stem cell transplantation. Additionally, priming MSCs with specific inflammatory mediators such as IFNγ may improve the immunomodulatory function of these cells and their extracellular vesicles. The aim of this study was to investigate the therapeutic effects of EVs derived from IFNγ-primed and non-primed MSCs in a pre-clinical rat model of ARDS.

Methods: The effects of EVs on anti-inflammatory and anti-bacterial properties of immune cells was investigated in vitro. Extracellular vesicles were isolated from the conditioned medium of primed (IFNγ, 50 ng/mL for 8 hours) or non-primed human umbilical cord MSCs by applying a series of centrifugation steps followed by reconstitution of the EV pellets in PBS. The approximate number and size of EVs were characterized by flowcytometry. The *in vitro* effects of EVs on bacterial phagocytosis/clearance were investigated in a human monocyte cell line (THP1) incubated with EVs for 72 hours. For the animal studies, we induced a pneumonia model of ARDS in male rats by intra-tracheal instillation of *E. coli* (5x10^9 CFU/kg), 30 minutes pre-treatment. Animals were divided into 3 treatment groups: 1. EVs derived from primed cells (n=11), 2. EVs derived from non-primed cells (n=8) and 3. PBS group (n= 12). Animals in EV treatment groups received a dose of about 75 million EVs/kg via tail vain injection. The extent of recovery and phenotype of animals in different treatment groups were assessed 48 hours post-treatment.

Results: Incubation of THP1 cells with EVs derived from IFNγ-primed MSCs significantly enhanced the bacterial phagocytosis and bacterial clearance properties of these cells by 60% and 40%, respectively. In animal studies, treatment with EVs isolated from both primed and non-primed MSCs significantly enhanced animal survival (from 66.7% in PBS group to 91-100% in EVs treated groups, $P<0.05$), while arterial blood oxygenation (at FiO_2= 1.0) was improved only in animals treated with EVs derived from IFNγ-primed MSCs. Additionally, a dramatic increase in the number of macrophages in bronchoalveolar lavage (BAL) fluid of both EV-treated animal groups was observed.

Conclusion: Our results demonstrate that treatment with extracellular vesicles isolated from UC-MSCs can efficiently enhance survival in rats with *E. coli* induced ARDS. In addition, treatment with EVs derived from IFNγ-primed MSCs may enhance their therapeutic effect, by improving the blood oxygenation in ARDS rats. Together with our in vitro findings, it can be concluded that MSC-derived EVs may represent an attractive option for future clinical trials in ARDS.

ORAL PRESENTATION 8

CARDIOGENIC EDEMA CONTRIBUTES TO A CLASSIC MODEL OF VENTILATOR-INDUCES LUNG INJURY

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Introduction: The original *in vivo* study of ventilator-induced lung injury by Webb and Tierney (1974) showed that high V_T (or driving pressure) with zero PEEP caused overwhelming lung injury, thought to be due to lung stretch resulting in high permeability edema. In pilot experiments we reproduced the classic study of Webb and Tierney; the precipitous nature of the edema (in 45/0); prompted us to wonder about a cardiac contribution. We therefore reproduced the experiments and determined the effect and contribution of ventricular function in this model of VILI.

Objective: To reproduce the lung injury and edema in the 'Webb and Tierney' study and investigate the contribution of hemodynamic mechanism.

Methods: Study was conducted in 5 series of experiments. In Series 1, Sprague-Dawley rats (≈400 g) received mechanical ventilation for 60 min according to the protocol of Webb and Tierney (Groups as per airway pressure; 14/0, 30/0, 45/10, 45/0 cmH_2O). Additional experimental series were conducted for groups 45/0 and 45/10 only (20 min duration, to ensure survival of all animals survived), to assess microvascular permeability (Series 2; 4/group), echocardiography (Series 3; 4/group); and right (Series 4; 5/group) and left ventricular pressure (Series 5; 4/group).

Measurements and Main Results: In Series 1, the original Webb & Tierney results were replicated in terms of lung/body weight ratio (45/0 > 45/10 ≈ 30/0 ≈ 14/0; $P<0.05$), and histology. In 45/0, pulmonary edema was overt and rapid, and survival <30 min. Microvascular permeability and total lung water increased in 45/0 (not in 45/10; Series 2). In 45/0, there was cyclical abolition of pulmonary blood flow (Series 3) and significant reduction of right ventricular systolic pressure (Series 4) with each breath. Over 20 min. there was progressive right ventricular dilation along with reduction in ventricular function (Series 3, 4; Figure 1). The left ventricular end-diastolic pressure (effective transmural) progressively increased in 45/0 (not in 45/10; Series 5, Figure 2).

Conclusions: The main mechanism of edema in this model of VILI is increased permeability and increased hydrostatic pressure (the effect of which is magnified when permeability is increased). This classic study that was central to the field of research in ventilator-induced lung injury, involved a substantial component of heart failure.

Supported By: Research funds (BPK) from the Canadian Institutes of Health Research. BPK holds the Dr. Geoffrey Barker Chair in Critical Care Research.

FIGURE 1 – RIGHT VENTRICULAR SIZE AND SYSTOLIC PRESSURE (*SERIES 3 AND 4*): The RV area indexed to the LV (RV:LV Ratio) increased in 45/0 and was unchanged in 45/10 (Panel A). In parallel with the increase in RV size, the RV systolic pressure decreased over time in 45/0, but not in 45/10 (Panel B). All measurements were made in expiration. *P<0.05, 2-way RM ANOVA. ***Abbreviations***: RV right ventricle; LV left ventricle; RVSP right ventricular systolic pressure.

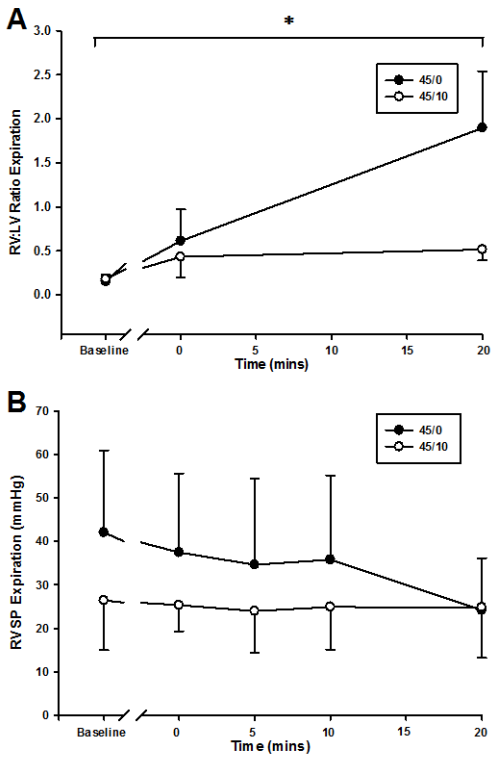

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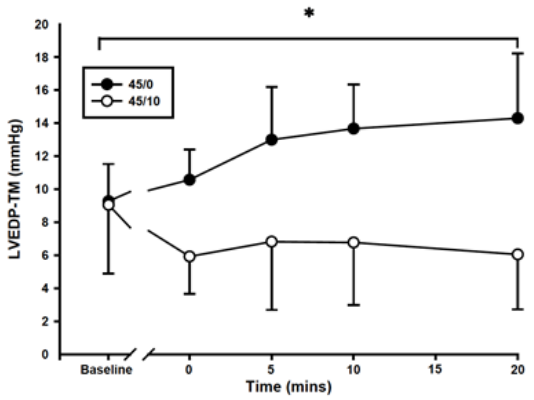
FIGURE 2 – LEFT VENTRICULAR END-DIASTOLIC PRESSURE (*SERIES 5*): The transmural left ventricular end-diastolic pressure increased significantly during the experiment in 45/0, but the values were lower in 45/10. *P<0.05, 2-way RM ANOVA. ***Abbreviations***: LVEDP-TM Transmural left ventricular end-diastolic pressure.



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OUTCOMES REPORTED IN STUDIES ON MECHANICAL VENTILATION IN CRITICALLY ILL PREGNANT AND POSTPARTUM PATIENTS: A SYSTEMATIC REVIEW

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Introduction & Objective: Approximately 0.2 % of pregnant women require mechanical ventilation, but data on this topic are limited and core outcomes (minimal outcomes that should be measured and reported in all clinical trials) have not been elucidated. We aim to develop of a core outcome set (COS) for studies on mechanical ventilation (MV) in pregnant and postpartum patients, and this systematic review identifying reported outcomes is the initial step.

Methods: We performed an electronic search using MEDLINE, EMBASE, CINAHL, the Cochrane Library, PROSPERO and the Joanna Briggs Institute EBP databases (Jan 1980 to Sept 2016). We used terms to identify studies published in English on pregnant or postpartum women requiring invasive or non-invasive MV in the intensive care unit (ICU) or a high-dependency unit. We excluded case reports and case series reporting on 5 or less patients published before the last 15 years. Two authors independently performed title and full text screening and data extraction.

Results: We retrieved 5034 studies and included 74 in the final analysis comprising 47 case reports (63%), 26 case-series (35%) and one case-control study describing mechanical ventilation (MV) in 842 patients.

Most (94%) studies described MV during the antepartum period. Acute respiratory distress syndrome was the most common reason for ventilation (56%), frequently related to influenza (26%). Modes of MV varied and 19% reported use of non-invasive ventilation. Ventilator parameters were specified in 65% of studies. Rescue interventions including neuromuscular blockers, prone positioning, high frequency oscillation ventilation, nitric oxide and extracorporeal membrane oxygenation were used in 34% of studies.

Reported outcomes were highly heterogeneous. Maternal mortality was often not detailed (44, 59%) and the time point of measurement not specified. Frequently reported maternal outcomes included duration of ventilation (54, 75%), ICU (33, 46%), and hospital length of stay (33, 46%). Reported perinatal outcomes included perinatal or neonatal mortality (23, 32%), Apgar score (20, 28%), birth weight (19, 26%), stillbirth, abortion and neonatal ICU admission or MV. Two studies reported neurological or developmental status of the infants. Obstetric details were limited to mode (45, 63%), indication and emergent/elective status of delivery. Three studies mentioned qualitative assessment of functional status and one mentioned mother-child bonding while none reported psychosocial outcomes.

Conclusion: Core outcomes reported in studies of MV during pregnancy are poorly and inconsistently described. To improve the conduct of future research in this rare but important clinical situation, development of a universally accepted COS is essential.

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P2 C Escudero, INTEGRATED miRNA-mRNA ANALYSIS REVEALS TRANSCRIPTOMIC SIGNATURES OF ABNORMAL MUSCLE REPAIR IN PATIENTS WITH ICU-ACQUIRED WEAKNESS.

P3 S Gupta, TYROSINE PHOSPHORYLATED CASPASE-8 REGULATES TOLL-LIKE RECEPTOR 4 ACTIVATION IN INFLAMMATORY SEPTIC NEUTROPHILS.

P4 D Islam, IMPACT OF LUNG MICROENVIRONMENT ON THE PROTECTIVE *VS.* DETRIMENTAL EFFECTS OF MSC ADMINISTRATION IN EXPERIMENTAL ACUTE LUNG INJURY.

P5 M Jerkic, HUMAN UMBILICAL CORD MESENCHYMAL STROMAL/STEM CELLS BENEFICIAL EFFECT IN PRECLINICAL MODEL OF SYSTEMIC POLYMICROBIAL SEPSIS IS DOSE DEPENDENT AND PARTLY MEDIATED BY HEME OXYGENASE 1.

Basic Science Category II

P6 M Jerkic, IL-10 OVEREXPRESSING HUMAN UMBILICAL CORD MESENCHYMAL STROMAL/STEM CELLS (UC-MSCS) ARE MORE EFFECTIVE THAN REGULAR UC-MSCS IN REDUCING SEVERITY OF RODENT *E. COLI* PNEUMONIA.

P7 JK Juss, NEMESIS (NEutrophil MEchanical StabilISation): A NOVEL EXTRACORPOREAL TECHNOLOGY TO MODULATE AND MONITOR PATHOLOGICAL INFLAMMATORY RESPONSES OF NEUTROPHILS (PMN) IN ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS).

P8 R Rabani, HUMAN MESENCHYMAL STROMAL CELLS ENHANCE MACROPHAGES FUNCTION THROUGH PRODUCTION OF PROSTAGLANDIN E2 AND LIPOXIN A4.

P9 A Volchuk, HUMAN MESENCHYMAL STEM/STROMAL CELLS ENHANCE PHAGOCYTOSIS OF IgG-OPSONIZED TARGETS IN HUMAN MONOCYTE-DERIVED MACROPHAGES IN PART BY ENHANCING PHAGOCYTIC Fcγ RECEPTOR EXPRESSION.

P10 CJ Walsh, DNA METHYLATION PATTERNS DIFFERENTIATE SEPTIC AND NON-SEPTIC CRITICALLY ILL PATIENTS.

Clinical Research Category

Clinical Research/Health Services Research I

P11 A Alfares, WEANING PATIENTS WITH ACUTE RESPIRATORY DISTRESS SYNDROME SUPPORTED BY VENO-VENOUS EXTRACORPOREAL MEMBRANE OXYGENATION.

P12 A Alali, PREDICTORS OF INTRACRANIAL HYPERTENSION IN SEVERE TRAUMATIC BRAIN INJURY: DATA FROM THE LATIN AMERICAN BEST-TRIP RANDOMIZED CONTROLLED TRIAL.

P13 BL Ferreyro, ACUTE PROMYELOCYTIC LEUKEMIA IN THE INTENSIVE CARE UNIT: A RETROSPECTIVE ANALYSIS.

P14 JC Gomez, FIBRINOLYSIS SHUTDOWN: DIAGNOSIS WITH ROTEM® AND CLINICAL IMPLICATIONS IN SEVERELY INJURED PATIENTS.

P15 D Junhasavasdikul, A CROSS-SECTIONAL EPIDEMIOLOGICAL PREVALENCE SURVEY OF PATIENTS RECEIVING OXYGEN IN A UNIVERSITY HOSPITAL – THE "WHARF" PROJECT.

Clinical Research/Health Services Research II

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P17 CE Orsso, DEVELOPMENT OF A METHODOLOGY TO EVALUATE ADIPOSITY FROM THORACIC COMPUTED TOMOGRAPHY IN INDIVIDUALS WITH ADVANCED LUNG DISEASE.

P18 T Pham, DO PATIENTS WITH UNILATERAL OPACITIES HAVE A SIMILAR OUTCOME THAN PATIENTS WITH ARDS? AN ANCILLARY ANALYSIS OF THE LUNG SAFE STUDY.

P19 M Urner, PREDICTING THE PRESENCE OF SPONTANEOUS BREATHING IN MECHANICALLY VENTILATED PATIENTS.

P20 T Lee, TRACHEOSTOMY OUTCOMES IN CRITICALLY ILL ELDERLY PATIENTS.

P21 HT Wang, OPIOID PRESCRIBING AFTER HOSPITAL DISCHARGE AMONG ELDERLY CHRONIC OPIOID USERS ADMITTED TO INTENSIVE CARE UNITS IN ONTARIO.

Systematic Review/Case Report/Education

P22 V Brulé, LONG-TERM EXTRA-CORPOREAL MEMBRANE OXYGENATION SUPPORT STRATEGIES IN A CHILD WITH RESPIRATORY FAILURE FROM ACUTE RESPIRATORY DISTRESS SYNDROME AS A BRIDGE TO LUNG TRANSPLANTATION.

P23 J East, CONVERSATIONS ABOUT CPR – PROFESSIONAL JUDGEMENT OR AUTONOMY?

P24 AN Geagea, ANTIMICROBIAL RESISTANCE AND ASSOCIATED INTERVENTIONS IN LOW- AND MIDDLE-INCOME COUNTRIES: SCOPING REVIEW.

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P26 BK Tirupakuzhi Vijayaraghavan, EVIDENCE SUPPORTING THE CLINICAL USES OF PROPORTIONAL ASSIST VENTILATION: A SYSTEMATIC REVIEW AND META-ANALYSIS.

P27 PA Lépine, AN APPLICATION OF MALCOM KNOWLES' PRINCIPLES OF ANDRAGOGY TO LEARNER-CENTRED TEACHING IN THE INTENSIVE CARE UNIT.

Quality Category

P28 PA Lépine, SIMPLIFIED CRITERIA TO DISTINGUISH BETWEEN EXUDATIVE AND TRANSUDATIVE PLEURAL EFFUSIONS.

P29 M GAETANI, FREQUENCY AND TYPE OF MEDICATIONS ADMINISTERED TO CRITICALLY ILL CHILDREN.

P30 S Lalgudi Ganesan, SEIZURE IDENTIFICATION BY CRITICAL CARE PROVIDERS USING QUANTITATIVE ELECTROENCEPHALOGRAPHY (QEEG) DISPLAYS AND DETERMINANTS OF PERFORMANCE.

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P32 C Rassameehirun, WHEN THE CODE BLUE IS REAL: OUTCOMES AND CHARACTERISTICS OF NON-ICU CARDIAC ARRESTS ATTENDED BY A CRITICAL CARE RESPONSE TEAM (CCRT).

P33 S Vakani, DEVELOPMENT OF A STANDARDIZED HANDOVER TOOL FOR COMMUNICATION BETWEEN PHYSICIANS IN THE EMERGENCY DEPARTMENT AND INTENSIVE CARE UNIT.

Physiology Category

P34 L Chen, DIFFUSE AIRWAY CLOSURE IN ACUTE RESPIRATORY DISTRESS SYNDROME.

P35 L Chen, THE RATIO OF MEASURED TO PREDICTED COMPLIANCE: A NEW PHYSIOLOGICAL AND PROGNOSTIC PARAMETER FOR ACUTE RESPIRATORY DISTRESS SYNDROME.

P36 W McClelland, DEFINING THE NORMAL RANGE FOR MAXIMAL SNIFF DIAPHRAGM THICKENING FRACTION IN HEALTHY SUBJECTS.

P37 N Mistry, CARDIOVASCULAR ADAPTATIONS TO MODERATE ANEMIA MAINTAIN CEREBRAL PERFUSION BUT ARE INADEQUATE TO PREVENT RENAL AND SPLANCHNIC TISSUE HYPOXIA.

P38 SE Riegler, DIAPHRAGM ECHOGENICITY IN MECHANICALLY VENTILATED PATIENTS: MEASUREMENT PRECISION AND PRELIMINARY FINDINGS.

P39 MC Sklar, A RANDOMIZED CROSS-OVER PHYSIOLOGICAL STUDY OF HIGH FLOW NASAL OXYGEN CANNULA VERSUS NON-INVASIVE VENTILATION IN ADULT PATIENTS WITH CYSTIC FIBROSIS: THE HIFEN STUDY.

P40 I Telias, ACCURACY OF P0.1 DISPLAYED BY MODERN VENTILATORS – FROM BENCH TO PATIENTS.