Rules

• A single static PowerPoint slide is permitted (no slide transitions, animations or 'movement' of any description, the slide is to be presented from the beginning of the oration).
• No additional electronic media (e.g. sound and video files) are permitted.
• No additional props (e.g. costumes, musical instruments, laboratory equipment) are permitted.
• Presentations are limited to 3 minutes maximum, and presenters will have points deducted if they exceed the 3 minutes.
• Presentations are to be spoken word (e.g. no poems, raps or songs).
• Presentations are to commence from the stage (e.g. no walking through the audience).
• Presentations are considered to have commenced when a presenter starts her/his presentation through movement or speech.
• The decision of the adjudicating panel is final.
Judging Criteria

Comprehension & Content

• Did the presentation provide an understanding of the background to the research question being addressed and its significance?
• Did the presentation clearly describe the key results of the research including conclusions and outcomes?
• Did the presentation follow a clear and logical sequence?
• Was the thesis topic, key results, and research significance and outcomes communicated in language appropriate to a non-specialist audience?
• Did the speaker avoid scientific jargon, explain terminology, and provide adequate background information to illustrate points?
• Did the presenter spend adequate time on each element of their presentation - or did they elaborate for too long on one aspect or was the presentation rushed?
Engagement & Communication

• Did the oration make the audience want to know more?
• Was the presenter careful not to trivialise or overly generalise their research?
• Did the presenter convey enthusiasm for their research?
• Did the presenter capture and maintain the audience's attention?
• Did the speaker have sufficient vocal range, maintain a steady pace, and have a confident stance?
• Did the PowerPoint slide enhance the presentation - was it clear, legible, and concise?
Today, August 4 SPATS Presenters:

from 8-930AM EST

1. Brian Patchett
2. Will Okoniewski
3. Chandler Annesi
4. Justin Uphus
5. Tharusan Thevathasan
6. Nishad Bhatta
7. Thomas Mahood
8. Noel Britton
Construct database of 477 allergic profiles

- D1: 4.3
- Maple: 11.6
- Birch: 10.6
- Elm: 9.28
- White Ash: 12.8
- D2: 4.87
- Cat Dander: 11.0
- Dog Dander: 0.81
- C. herbarum: 17.1
- A. alternata: 36.1
- Oak: 6.01
- Ragweed: 5.90
- Aspergillus: 5.55
- Cockroach: 0.18
- Penicillium: 6.66
- Cedar: 2.69
- Walnut Tree: 13.0
- Sycamore: 11.4
- Cottonwood: 8.03
- Mulberry: 0.22
- C. dactylon: 0.13
- P. pratense: 0.13
- Pigweed: 5.09
- Mugwort: 7.35
- Sheep Sorrel: 5.19

Gaussian Mixture Modeling

Higher serum IgE & Eosinophil count

A Framework for the Clinical Interpretation of the Allergic Poly-Sensitized Asthmatic

Worsening Obstruction
Better Acute Glycemic Control During Pulmonary Exacerbations Is Associated with Longer Time to Next Admission in Pediatric Cystic Fibrosis

BACKGROUND
It is unknown if acute glucose control is associated with time between acute CF exacerbations.

RESULTS
Poor glycemic control was associated with shorter time to next hospitalization:
• Basic model HR=1.76, p=0.042
• Fully-adjusted model HR=2.05, p=0.016

This was largely driven by patients who completed treatment at home (not pictured):
• Basic model HR=2.2, p=0.065
• Fully-adjusted model HR=3.4, p=0.04

METHODS
• 164 inpatient CF exacerbations (2010-2016)
• Analyzed glucose control as area under the curve (AUC)
• Multiple-event adjusted survival analysis with two models (basic and fully-adjusted)

TAKE-HOME POINTS
In CF patients hospitalized for a pulmonary exacerbation, poor acute glycemic control is associated with shorter time to next hospital admission.
Long-Term Outcomes in Bronchopulmonary Dysplasia Requiring Tracheostomy: A Boston Children’s Cohort

Demographics and comorbidities for BPD subjects with and without tracheostomy.

<table>
<thead>
<tr>
<th></th>
<th>tBPD</th>
<th>sBPD</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean Gestational Age</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>in months (SD)</td>
<td>27.06 (2.63)</td>
<td>26.64 (2.34)</td>
<td>0.043</td>
</tr>
<tr>
<td><strong>DEMOGRAPHICS</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male (%)</td>
<td>32/49 (65.31)</td>
<td>30/49 (61.22)</td>
<td>0.834</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White (%)</td>
<td>29/44 (65.91)</td>
<td>34/42 (80.95)</td>
<td>0.021*</td>
</tr>
<tr>
<td>Black (%)</td>
<td>14/44 (31.82)</td>
<td>4/42 (9.52)</td>
<td></td>
</tr>
<tr>
<td>Asian (%)</td>
<td>1/44 (2.27)</td>
<td>4/42 (9.52)</td>
<td></td>
</tr>
<tr>
<td>Hispanic/Latino (%)</td>
<td>13/40 (32.5)</td>
<td>2/34 (5.88)</td>
<td>0.008*</td>
</tr>
<tr>
<td><strong>COMORBIDITIES</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subglottic Stenosis (%)</td>
<td>30/48 (62.5)</td>
<td>0/49 (0)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Pulmonary Hypertension (%)</td>
<td>18/48 (37.5)</td>
<td>8/49 (16.33)</td>
<td>0.023*</td>
</tr>
</tbody>
</table>

Childhood best lung function controlling for gestational age and multiple gestation

<table>
<thead>
<tr>
<th></th>
<th>( \beta )</th>
<th>95% CI</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV(_1) % Predicted</td>
<td>-16.44</td>
<td>(-26.92, -5.97)</td>
<td>0.003*</td>
</tr>
<tr>
<td>FVC % Predicted</td>
<td>-12.38</td>
<td>(-23.10, -1.65)</td>
<td>0.024*</td>
</tr>
<tr>
<td>FEV(_1)/FVC</td>
<td>-6.891</td>
<td>(-13.80, 0.02)</td>
<td>0.051</td>
</tr>
</tbody>
</table>

Neurodevelopmental outcomes

(a) Cognitive
(b) Gross Motor
(c) Fine Motor

* P<0.05

Chandler Annesi
Boston University School of Medicine
cannesi@bu.edu
Vasoresponders

Non-vasoresponders

Vienna Definition of Vasoresponse
10% Drop in mPAP

Vanderbilt Definition of Vasoresponse
10 mmHg Drop in mPAP

Classic Definition of Vasoresponse
10 mmHg Drop in mPAP to below 40 mmHg

Years from initial catheterization

Survival from Death or Lung Transplant

Vasoresponders

Non-vasoresponders

Years from initial catheterization

Survival from Death or Lung Transplant

Vasoresponders

Non-vasoresponders

Nitric oxide vasoresponder

Nitric oxide NON-vasoresponder
Introduction

- Without an absolute indication for organ support, there is equipoise over who may benefit from postoperative critical care.
- Utilisation of critical care is correlated with critical care bed availability which varies stochastically.
- **Objective:** To investigate the causal effects of postoperative critical care versus surgical ward admission on patient morbidity and mortality with consideration of critical care bed strain.

Methods

<table>
<thead>
<tr>
<th>Study design</th>
<th>Prospective, international, multicentric cohort study in 248 hospitals in the United Kingdom, Australia and New Zealand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient population</td>
<td>21,935 adult patients undergoing inpatient surgery without an absolute indication for postoperative critical care</td>
</tr>
<tr>
<td>Primary outcomes</td>
<td>Postoperative Morbidity Survey (POMS) on day 7, 30-day and 60-day mortality</td>
</tr>
<tr>
<td>Primary analysis</td>
<td>Multivariable regression with 29 demographic and perioperative predictor variables (observed confounding)</td>
</tr>
<tr>
<td>Secondary analysis</td>
<td>Instrumental variable method with instruments on critical care bed strain (observed and unobserved confounding)</td>
</tr>
</tbody>
</table>

Conclusion

Although postoperative critical care admission means patients are more likely to incur short-term morbidity, it confers longer-term mortality benefits (at 30 and 60 days).

### Primary outcomes

<table>
<thead>
<tr>
<th>7-day morbidity</th>
<th>30-day mortality</th>
<th>60-day mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unadjusted</td>
<td>Adjusted</td>
<td>Instrumental</td>
</tr>
<tr>
<td>(without confounders)</td>
<td>multivariable regression</td>
<td>variable method</td>
</tr>
<tr>
<td><strong>Risk ratio</strong></td>
<td><strong>Risk ratio</strong></td>
<td><strong>Risk ratio</strong></td>
</tr>
<tr>
<td>*** p &lt;0.001</td>
<td>*** p &lt;0.001</td>
<td>*** p &lt;0.001</td>
</tr>
</tbody>
</table>

### Mortality risk stratified by Surgical Outcome Risk Tool

<table>
<thead>
<tr>
<th>Surgical risk groups</th>
<th>30-day mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;10%</td>
<td>&gt;9%</td>
</tr>
<tr>
<td>&gt;8%</td>
<td>&gt;7%</td>
</tr>
<tr>
<td>&gt;6%</td>
<td>&gt;5%</td>
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<tr>
<td>&gt;4%</td>
<td>&gt;3%</td>
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<tr>
<td>&gt;3%</td>
<td>&gt;2%</td>
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<tr>
<td>&gt;2%</td>
<td>&gt;1%</td>
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<table>
<thead>
<tr>
<th>60-day mortality</th>
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</thead>
<tbody>
<tr>
<td>&gt;10%</td>
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<td>&gt;9%</td>
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<tr>
<td>&gt;8%</td>
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<tr>
<td>&gt;7%</td>
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<td>&gt;6%</td>
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<td>&gt;2%</td>
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<td>&gt;1%</td>
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</table>

<table>
<thead>
<tr>
<th>SORT risk groups</th>
<th>Surgical risk groups</th>
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<tbody>
<tr>
<td>&gt;10%</td>
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<tr>
<td>&gt;2%</td>
<td>&gt;1%</td>
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</table>

Risk ratio (95% Confidence Interval) between critical care versus ward admission after surgery.
Clinical Question
What is the Prevalence of Comorbidities in Pleural Effusion (PE) in Developing Countries & How does their Presence Impact Treatment & Prognosis?

Study Design
1 Year (Jan – Dec 2018) Retrospective Audit of Discharge Summaries of Pleural Effusion at BPKIHS, Nepal for Studying the Prevalence of comorbidities & their effect on treatment outcomes.

Results
45% Patients with PE had Comorbidities

Pleural Effusion (PE) with Comorbidities were:
- Loculated PE
- ICTD Complication
- Required fibrinolytics
- Hospital Stays

Multi-Morbidity in PE cluster around the Risk of:
- Tobacco Smoking
- Cardio-Metabolic Disease
- Alcohol Abuse

Conclusions
Patients with Pleural Effusion in Developing Countries have a high Prevalence of Comorbidities and their Presence indicates towards Worse Prognosis

Presence and Pattern of Comorbidities in Patients with Pleural Effusion: Audit of Pulmonary Discharge Summaries from Developing Country
N. Bhatta 1, K. Bhandari 1, D.A. Bhattacharjee 1, D.R. Mishra 2, A.B. Acharya 1, N. Bhatta 2
AJRCCM2020;201:A1562
nishadstar7@gmail.com
Can Prenylation Alter Lung Inflammation?

Thomas Mahood (PhD Candidate) ummahoot@myumanitoba.ca
PI: Dr. Andrew Halayko

Prenylation: A post-translational modification of a protein using a lipid molecule (isoprenoid)

Simvastatin ——— HMG-CoA

Hypothesis: Inhibiting prenyltransferases can blunt proinflammatory cytokine release

Prenyltransferases (PT):
- Found in all types of lung cells
- Gene expression ↑ in COPD patients

COPD Lung Fibroblasts:
- PT abundance and activity ↑ during cigarette smoke exposure
- Protein targets ↑ abundance at the cell membrane during cigarette smoke exposure

- Lung fibroblast response to pro-inflammatory stimuli is partially mediated by PTs
- PT inhibitors should be considered for evaluation using pre-clinical models of smoking associated lung inflammation and disease
Of 100 ICU patients...

- 40 are mechanically ventilated.
- 10 have ARDS.
- 3 ARDS survivors are unable to return to work due to ARDS-related health problems.
- 4 die due to ARDS.