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[http://hdl.handle.net/10536/DRO/DU:30086902](http://hdl.handle.net/10536/DRO/DU:30086902)
2014. For 542 surgical procedures during the study period, 62 SSI (11.4%) occurred as compared to 102 cases for 680 in the control period (15%). The adjusted odds ratio of the SSI rate was 0.7319 and was found to be 27% lower post intervention. 

Conclusion: The implementation of the bundle was associated with improved compliance over time and a significant reduction of the SSI rate. This makes the bundle an important tool to improve patient safety.

P32
More effective use of polymyxin-B hemoperfusion for nonoperation cases.

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Introduction: Direct hemoperfusion with polymyxin-B has been reported to improve hemodynamics in postsurgical patients. In 2012, the Japanese Guidelines for the Management of Sepsis were published and mention the efficacy of polymyxin-B direct hemoperfusion. But how to use and the targets of this therapy are varied by facilities. We investigated the effective use of polymyxin-B direct hemoperfusion in nonsurgical patients.

Methods: We analyzed retrospectively all septic shock patients who were treated with polymyxin-B hemoperfusion between January 2008 and December 2012. We checked their mean arterial pressure (MAP), and vasopressor requirement every 30 minutes until stopping treatment.

Results: There were 32 patients under treatment and 11 patients did not need surgical treatment. Even in the nonsurgical group, hemodynamic states and vasopressor requirement was improved after polymyxin-B hemoperfusion started. And the effects were continued over 120 minutes. A second polymyxin-B hemoperfusion treatment was performed in nine patients. In second treatment, MAP increased in the nonsurgical group greater than in the postsurgical group.

Conclusion: Polymyxin-B direct hemoperfusion improves hemodynamic status even in nonsurgical patients. A second polymyxin-B direct hemoperfusion is effective especially in nonsurgical septic shock patients. And if its hemodynamic effect was significantly, long-time treatment should be considered.

P33
Sepsis modulates the human hematopoietic stem cell compartment in peripheral blood and bone marrow.

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Introduction: Efficient fight with infection requires robust production of immunocompetent cells. This response is called emergency hematopoiesis and depends on the proliferation of progenitor cells and awakening dormant hematopoietic stem cells (HSCs) into cycling. As during sepsis an altered immune system is often observed, it seems important to reveal the impact of this syndrome on HSCs. Recent discoveries have shown that HSCs circulate in the peripheral blood and may boost local immune response via paracrine mechanisms and differentiation into myeloid cells. Altogether, these rationales led us to investigate the circulating HSCs in septic patients and in the bone marrow (BM) of septic “humanized mice” transplanted earlier with human HSCs.

Methods: Samples of peripheral blood were collected from 23 patients with sepsis (on days 1 and 3) and 20 healthy volunteers. The following antigens were analyzed by flow cytometry: CD34, CD38, Ki-67, CD133, Lin and CD45. In order to investigate HSCs in their microenvironment, a model of coculture and puncture (CLP) was performed on the NOD.Cg-PrkdcscidIl2rg mice that were transplanted with human cord blood CD34+ cells 8 weeks earlier. BM cells were analyzed 24 hours after CLP by colony-forming unit assay with medium supporting growth of human cells.

Results: Septic patients had a significantly increased (threelfold, P < 0.01) number of CD34+CD38− HSCs on the third day of the disease. Also, the CD133+ HSC number was increased in septic patients, while CD34+CD45− “Lin” progenitors were detected at much lower level than in controls. Interestingly, Ki-67+CD34+ “Lin” cells were fourfold higher in septic patients. Patients with higher number of CD133+ HSCs had significantly lower likelihood of 60-day survival (P = 0.05). Analysis of human HSCs from BM of septic mice revealed significantly compromised hematopoietic colonies output (248 vs. 125 in sham animals). CLP caused also expansion of CD34+CD38− HSCs in BM and absolute increase of Ki-67+CD34+ “Lin” cells (1.5-fold).

Conclusion: In this work we have observed significant changes in circulating HSCs during sepsis. During the disease, dormant HSCs enter the cell cycle (measured by Ki-67 expression) and are mobilized to the peripheral blood. However, the progenitor cells disappear from circulation. Novel use of humanized mice confirmed expansion of early human HSCs in BM during the sepsis model. Despite expansion of the HSC pool, the amount of functional progenitors in BM is decreased in this model. We suggest that HSCs play a significant role in the course of sepsis and may become a new prognostic and therapeutic target.

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P34
Physiological changes after fluid bolus therapy in sepsis: a systematic review of the contemporary literature.

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Introduction: Fluid bolus therapy (FBT) is a ubiquitous intervention in intensive care. However, the physiological effects in the critically ill are poorly understood. Therefore, we systematically reviewed the contemporary literature to determine the current practice and effect of FBT in the management of severe sepsis and septic shock.

Methods: We interrogated the MEDLINE, CENTRAL and EMBASE electronic reference databases using a combination of terms to define a set of records of studies of fluid administration in patients with severe sepsis or septic shock. To achieve contemporary relevance, results were limited to English-language studies in adults between 2010 and 2013.

Results: We identified 22 prospective observational studies, four retrospective observational studies, two quasi-experimental studies, and five randomised controlled trials (RCTs), 41 boluses in total. No RCT compared FBT with alternative interventions. The median fluid bolus was 500 ml (range: 100 to 1,000 ml) administered over 30 minutes (range: 10 to 60 minutes) and 0.9% sodium chloride solution was the most commonly administered. Although 17 studies describe the temporal course of physiological changes after FBT in 31 patient groups, only three studies describe the physiological changes at 60 minutes, and only one study beyond this point (Figure 1). No studies related the physiological changes after FBT with clinically relevant outcomes.

Conclusion: There is a need for obtaining randomised controlled evidence for the physiological effects of FBT in patients with severe sepsis and septic shock beyond the period immediately following its administration.

P35
Forty percent of hospitalizations after severe sepsis are potentially preventable.

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Introduction: Patients are frequently rehospitalized in the 90 days after severe sepsis. The rate of readmission exceeds patients’ baseline rate of hospitalization, and also exceeds the rate after matched nonsepsis hospitalizations [1]. We sought to determine the most common readmission diagnoses after severe sepsis, the extent to which readmissions may be preventable, and whether the pattern of readmission diagnoses differs from that of nonsepsis hospitalizations.

Methods: We studied participants in the US Health and Retirement Study with linked Medicare claims (1998 to 2010) [2]. Using validated methods [3,4], we identified severe sepsis and nonsepsis hospitalizations, then measured 90-day readmissions in each cohort. Using Healthcare Cost & Utilization Project’s Clinical Classification Software [5], we determined the 10 most common readmission diagnoses after severe sepsis. We measured rates of ‘potentially preventable’ readmissions using published definitions [6]. We compared rates of all-cause, potentially preventable, and cause-specific hospitalizations between survivors of severe sepsis and nonsepsis hospitalizations using chi-squared tests.

Results: We identified 3,703 severe sepsis and 44,840 nonsepsis hospitalizations, of which 3,036 (82.0%) and 43,539 (93.1%) survived to discharge, respectively. In the next 90 days, 43.6% of severe sepsis survivors were rehospitalized, compared to 34.8% of nonsepsis survivors, P < 0.001. The top readmission diagnoses following severe sepsis (Table 1) included several recognized potentially preventable diagnoses: heart failure, pneumonia, exacerbation of chronic obstructive pulmonary disease (COPD), and urinary infection. Also common were readmissions for sepsis, acute renal failure, and aspiration pneumonitis, diagnoses that could plausibly be prevented or treated early to prevent hospitalization. Patterns of readmission differed in severe sepsis survivors; rates of readmission for sepsis, renal failure, respiratory failure, device complication, and aspiration pneumonitis were higher and accounted for a greater proportion of the total readmissions. Potentially preventable hospitalizations - infection (sepsis, pneumonia, urinary tract, and skin or soft tissue), heart failure, COPD exacerbation, acute renal failure, and aspiration pneumonitis - accounted for 40.5% of all readmissions after severe sepsis (compared to 25.8% following nonsepsis admission, P < 0.001), and 19.6% of severe sepsis survivors experienced a readmission for one of these diagnoses (compared to 9.5% following a nonsepsis admission, P < 0.001).

Conclusion: Forty percent of hospitalizations after severe sepsis occur for diagnoses that may be preventable. A few disease categories account for a relatively large proportion of the hospitalizations after severe sepsis, suggesting the feasibility of tailoring postdischarge interventions to patient’s personalized risk for these common postsepsis diagnoses.

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References

<table>
<thead>
<tr>
<th>Rank</th>
<th>Diagnosis category</th>
<th>Proportion of all 90-day admissions (%)</th>
<th>Survivors with 90-day admission (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Congestive heart failure, nonhypertensive</td>
<td>10.4</td>
<td>5.7*</td>
</tr>
<tr>
<td>2</td>
<td>Septicemia</td>
<td>9.5*</td>
<td>6.5*</td>
</tr>
<tr>
<td>3</td>
<td>Pneumonia</td>
<td>5.4</td>
<td>3.5*</td>
</tr>
<tr>
<td>4</td>
<td>Rehabilitation care</td>
<td>5.1</td>
<td>3.2</td>
</tr>
<tr>
<td>5</td>
<td>Acute and unspecified renal failure</td>
<td>4.6*</td>
<td>3.2*</td>
</tr>
<tr>
<td>6</td>
<td>Respiratory failure</td>
<td>4.1*</td>
<td>2.5*</td>
</tr>
<tr>
<td>7</td>
<td>Complication of device, implant, or graft</td>
<td>3.5*</td>
<td>2.3*</td>
</tr>
<tr>
<td>8</td>
<td>COPD and bronchiectasis</td>
<td>3.1</td>
<td>1.8</td>
</tr>
<tr>
<td>9</td>
<td>Urinary tract infection</td>
<td>3.1</td>
<td>1.8*</td>
</tr>
<tr>
<td>10</td>
<td>Aspiration pneumonitis</td>
<td>2.8*</td>
<td>1.8*</td>
</tr>
</tbody>
</table>

*Value greater than that of nonsepsis survivors, P ≤ 0.001 for each comparison.

Figure 1(abstract P34) Haemodynamic changes following FBT at 1, 2, 3 and 4 hours

Table 1(abstract P35) Top ten hospitalization diagnoses in the 90 days following severe sepsis