Intensive News

Highlights from the 36th International Symposium on Intensive Care and Emergency Medicine, ISICEM

Brussels 15th-18th March 2016
Presented **VANISH-Study Results** (Vasopressin vs. Norepinephrine) and reminded of rationale for vasopressin in septic shock:

- Relative vasopressin deficiency
- Powerful vasoconstrictor
- Acts on V1 receptors (vasoconstriction) but also on V2 receptors, which may have a role in septic shock

**WHY DO WE NEED MORE VASOPRESSORS?**

- Lower doses of multiple vasopressors more efficacious than high doses of one class
- High dose catecholamines have significant toxicity
- Catecholamines are prone to tachyphylaxis
- There are reasons why evolution gave us more than one vasopressor!!

*Reported about ATHOS-study (Angiotensin-II for treatment of high-output shock)*
Five arguments to initiate norepinephrine early

1. Duration and degree of hypotension associated with increased mortality
2. NE increases cardiac output, when initiated early
3. NE improves microcirculation, when initiated early,
4. Early initiation of vasopressors prevents harmful fluid overload
5. Delayed initiation of vasopressors associated with increased mortality

Jean-Louis Teboul
Highlightled Lectures at ISICEM 2016 / Vasopressors and Betablockers

Prof. Dr. Mervyn Singer
University Hospital College, London

„DECATECHOLAMINIZATION“
Dangers in using high dose catecholamines

- Tachyarrhythmia, Digital ischaemia
- Immunosuppression
- Stimulation of bacterial growth + virulence
- Metabolic modulation

BETABLOCKERS IN SEPSIS
- Appear to benefit sicker subjects with elevated heart rate
- Mechanism of action supporting benefits are unclear

HIGH DOSE CATECHOLAMINE - HIGH MORTALITY (>90%)
Recommendation:
- Do not exceed 1mcg/kg/min (4mg/h) norepinephrine
- Do not switch to other catecholamines
- Switch to vasopressin in refractory shock

Prof. Claude Martin
Marseille
Radomized trials


Reviews and Editorials


Literature Update /
ß-Blocker vs. Calcium-Channel-Blocker

| Atrial Fibrillation during Sepsis: |
i.v. Beta-Blocker show significantly lower hospital mortality than Calcium-Channel-Blocker, Digoxin or Amiodaron |


<table>
<thead>
<tr>
<th>ß-Blocker vs. Kalziumkanal-Blocker</th>
<th>RR (95% CI)</th>
<th>P for interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>All patients</td>
<td>N = 18,720</td>
<td>0.92 (0.86-0.97)</td>
</tr>
<tr>
<td>Pre-existing AF</td>
<td>N = 15,076</td>
<td>0.94 (0.99-1.01)</td>
</tr>
<tr>
<td>New-onset AF</td>
<td>N = 3,174</td>
<td>0.99 (0.86-1.15)</td>
</tr>
<tr>
<td>No vasopressor</td>
<td>N = 13,360</td>
<td>0.98 (0.91-1.06)</td>
</tr>
<tr>
<td>Vasopressor</td>
<td>N = 4,946</td>
<td>0.86 (0.79-0.94)</td>
</tr>
<tr>
<td>No heart failure</td>
<td>N = 10,710</td>
<td>0.90 (0.83-0.98)</td>
</tr>
<tr>
<td>Heart failure</td>
<td>N = 7,890</td>
<td>0.87 (0.80-0.95)</td>
</tr>
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<table>
<thead>
<tr>
<th>ß-Blocker vs. Digoxin</th>
<th>RR (95% CI)</th>
<th>P for interaction</th>
</tr>
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<tbody>
<tr>
<td>All patients</td>
<td>N = 13,994</td>
<td>0.79 (0.75-0.84)</td>
</tr>
<tr>
<td>Pre-existing AF</td>
<td>N = 12,142</td>
<td>0.77 (0.72-0.82)</td>
</tr>
<tr>
<td>New-onset AF</td>
<td>N = 1,932</td>
<td>0.75 (0.64-0.88)</td>
</tr>
<tr>
<td>No vasopressor</td>
<td>N = 8,546</td>
<td>0.80 (0.73-0.88)</td>
</tr>
<tr>
<td>Vasopressor</td>
<td>N = 5,228</td>
<td>0.79 (0.73-0.86)</td>
</tr>
<tr>
<td>No heart failure</td>
<td>N = 7,028</td>
<td>0.73 (0.67-0.80)</td>
</tr>
<tr>
<td>Heart failure</td>
<td>N = 7,142</td>
<td>0.82 (0.75-0.89)</td>
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<table>
<thead>
<tr>
<th>ß-Blocker vs. Amiodaron</th>
<th>RR (95% CI)</th>
<th>P for interaction</th>
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<tbody>
<tr>
<td>All patients</td>
<td>N = 10,190</td>
<td>0.65 (0.61-0.69)</td>
</tr>
<tr>
<td>Pre-existing AF</td>
<td>N = 8,088</td>
<td>0.63 (0.58-0.67)</td>
</tr>
<tr>
<td>New-onset AF</td>
<td>N = 2,372</td>
<td>0.67 (0.59-0.77)</td>
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<tr>
<td>No vasopressor</td>
<td>N = 4,464</td>
<td>0.73 (0.65-0.81)</td>
</tr>
<tr>
<td>Vasopressor</td>
<td>N = 5,378</td>
<td>0.64 (0.59-0.69)</td>
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<tr>
<td>No heart failure</td>
<td>N = 5,562</td>
<td>0.62 (0.57-0.67)</td>
</tr>
<tr>
<td>Heart failure</td>
<td>N = 5,054</td>
<td>0.66 (0.60-0.72)</td>
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Kohortenstudie an 39.693 Sepsis-Patienten: ß-Blocker zeigten geringere Spitals-Mortalität als Kalziumkanal-Blocker, Digoxin oder Amiodaron

**ESMOLOL in Myocardial Infarction**


   This pilot study shows that esmolol can decrease myocardial damage and improve cardiovascular function recovery in STEMI patients.

**ESMOLOL in Takotsubo Cardiomyopathy**


   This cohort supports esmolol is best choice to treat takotsubo myocardial dysfunction. Patients tolerate well high dose of esmolol infused over 24h.
ESMOLOL in Perioperative Surgery


Two recent studies\(^1,2\) showing esmolol adjunct potentiates anesthesia and helps optimizing patient management.

Two studies\(^4,5\) and two reviews\(^3,6\) published in 2015 on the same topic showing interest for this technique.
Literature Update / Norepinephrine

Norepinephrine - Not Too Much, Not Too Long

A threshold of 0.5 mcg/kg/min NE is associated with > 2x increase of mortality

Literature Update / Vasopressin

VASOPRESSIN in septic shock


One recent study finding improved survival in vasopressin group vs norepinephrine.


One recent metaanalysis supporting potential benefits of vasopressin.