



SEPSIS PROTOCOL DESIGN

ED Recognition and Risk-stratification

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STOPPING SEPSIS
Saving Lives in Pennsylvania



Declarations

- NIH funding (U series grant, PETAL network, NHLBI; R series grant, ProACT trial, NIGMS)
- Deputy Editor, *Annals of Emergency Medicine*
- Royalties from writings (*Tintinalli's Comprehensive Study Guide; ED Critical Care; UpToDate*)
- Expert consulting
- Unpaid role on steering committee for Ferring International (selepressin in septic shock)
- Led ACEP sepsis DART initiative

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Why are we here?

- You've heard the sepsis definitions and burden
- The ED is the initial site of hospital based care for @75-80% who ultimately are diagnosed with sepsis in any form
 - EMS brings about 75% of those suffering from sepsis to hospital
- Concentration of providers and resources
 - NQF 0-500 and CMS Sep-1 measure
- Early care matters

▶ What do the new trials show

- All *built* on early recognition – Rivers et al EGDT; Jones et al lactate clearance; ProCESS/ARISE/ProMISE
 - We know that earlier matters in ‘real life’ – Kaukonen *JAMA* 2014
- Once recognized, prompt ED-based “aggressive” (sic) therapy is key step
- Reassessment and titration
- There is no one-best way
 - Fluid
 - Source control (ATB)
 - Respiratory and cardiovascular support
 - Surveil and limit other organ failure

The Challenge in the ED

- Signal: Noise (CDC and NHACMS)
 - 140 million ED visits 2014; 5-6:1 discharged : admitted
 - ED presenting complaints: Fever #1 in children, # 3 in adults; some form of infection is # 5, 7, 9 in adults – diagnosis pattern similar
 - Across U.S., @ 5 million have fever on ED presentation, with @ 450k having non-exposure hypothermia
 - But, 550-600k will have sepsis, with death happening 15-35% of the time

The Challenge in the ED

- Tools
 - Vital signs – best availability at start, vary widely after; age issues
 - BP – what cut point?
 - RR – accuracy?
 - Shock Index (HR/SBP; >0.8 bad)
 - Temp measurements
 - Complaints – see before.
 - Sick and not so sick look the same often
 - Testing – no one ‘test’ exists
 - Easy things – CBC, basic labs, source testing – help with infection detection but not sensitive or specific alone
 - Who needs more testing? What – lactate? Others? Invasive?

Two Bedside Data Approaches

- Oldest school – gestalt. Not well examined.
- Older school - SIRS (variable performance, low specificity for sepsis).
 - Variables: $T > 38$ or < 36 ; $HR > 90/\text{min}$; $RR > 20/\text{min}$; $WBC > 14\text{k}$
 - 2 or more = likely **but** misses 15% (Kaukonen et al *NEJM* 2015)
 - Only 1 means many/most wont have sepsis
 - Mortality goes up with more but ? transition point
- New school – qSOFA (SEP-3, *JAMA* 2016). Specific for poor outcomes
 - Variables: $SBP \leq 100$ or lower; $RR \geq 22/\text{min}$ or more; altered mental status
 - Two or more – if infected, get busy with resuscitation and assessing organ function

Potential Solutions

- Recognize that two subgroups exist, and require approaches for each with some overlap
 - **Obvious** infection with organ dysfunction – AMS, hypotension, tachycardia, resp distress (qSOFA or multiple SIRS)
 - Right into care path – Fluid bolus, ATB, source specimens, resp/circulatory support
 - New “septic shock” group (SEP-3) – ongoing hypotension requiring VP after volume infusions with lactate elevation = highest mortality – most aggressive approaches
 - **Not so obvious** –
 - **Two or more looks, triggered off presence or history of fever/low temp**
 - Additive over time
 - Test – **esp. lactate** – when unsure or if one other ‘sign’
 - Pediatric accommodations
 - Protocolize
 - Triage/order sheet/e-surveillance all options

Potential Solutions

- Require **assessment** impact
 - Processes – data integrity, tool use, time of events
 - Outcomes – hit target or hit improvement
 - Diagnosed with a some form of sepsis
 - Mortality and non-mortal outcomes
- Require attack **gaps**
 - Plans
 - Trends
 - Reports