

# Approaches in Critical Care

a publication for trauma and critical care professionals

## Sepsis

### ■ Four Minnesota Case Reports

Sepsis following H1N1 infection

Sepsis in an immunosuppressed cancer patient

Post-partum sepsis

Peritoneal septic shock

### ■ EMS Perspectives

The pre-hospital management of suspected sepsis

### ■ Critical Care Profile

James W. Leatherman, MD,  
director of Hennepin's  
medical intensive care unit



## Dear Readers:



I am not a fan of medical dramas like *ER* and *Grey's Anatomy* but when I do tune in I sometimes chuckle at how often patients are shot, knifed, burned, taken hostage (often while in the emergency department), beaten, and/or struck with the oddest afflictions and illnesses. Meanwhile, a new survey (see page 15) shows that the majority of Americans aren't even familiar with the word "sepsis" – a condition that kills more than 500 Americans every day and often is followed by long-term physical and cognitive disabilities.

In this issue, we review four cases of sepsis in patients with diverse circumstances. Robert Ball, EMT-P, describes pre-hospital care for the potential sepsis patient while James Leatherman, MD, director of Hennepin County Medical Center's medical intensive care unit, offers wisdom on diagnosing and managing sepsis in the emergency department. Taken together, this issue is a thorough look at how sepsis preys on patients and how some of Minnesota's most experienced clinicians manage the condition.

The theme for the next issue will be pediatric trauma care. If you have an interesting case study you'd like to contribute, see the author's guidelines on the *Approaches in Critical Care* Web site at [www.hcmc.org/approaches](http://www.hcmc.org/approaches). We'd love to hear from you.

Sincerely,

A handwritten signature in white ink that reads "Michelle Biros". The signature is fluid and cursive.

Michelle H. Biros, MD, MS  
*Approaches in Critical Care* Editor-in-Chief  
Department of Emergency Medicine  
Hennepin County Medical Center



Hennepin County **Medical Center**

Every Life Matters



# Approaches in Critical Care

## Approaches in Critical Care

### Editor-in-Chief

Michelle Biros, MD, MS

### Managing Editor

Linda Zespy

### EMS Perspectives Editor

Robert Ball, EMT-P

### Graphic Designer

Karen Olson

### Public Relations Director

Tom Hayes

### Patient Care Director, Critical Care and Emergency Services

Kendall Hicks, RN

### Patient Care Director, Behavioral and Rehabilitative Services

Joanne Hall, RN

### Printer

Sexton Printing

### Photographers

Raoul Benavides

Karen Olson

### Clinical Reviewers

James Leatherman, MD

### Events Calendar Editor

Susan Altmann

a publication for trauma and critical care professionals

### Case Reports

- 2 Sepsis following H1N1 infection  
Brian Driver, MD
- 5 Sepsis in an immunosuppressed cancer patient  
Lisa Hayden, MD
- 6 Post-partum sepsis  
Rob Beyer, MD and Jamie Karambay, MD
- 7 Peritoneal septic shock  
Justin Kane, MD
- 11 **Critical Care Profile**  
James Leatherman, MD, medical director of Hennepin's medical intensive care unit
- 14 **EMS Perspectives**  
Prehospital management of suspected sepsis
- 16 **Calendar of Events**
- 18 **News Notes**

### To submit an article

Contact the managing editor at [approaches@hcmcd.org](mailto:approaches@hcmcd.org). The editors reserve the right to reject the editorial or scientific materials for publication in *Approaches in Critical Care*. The views expressed in this journal do not necessarily represent those of Hennepin County Medical Center, or its staff members.

### Copyright

Copyright 2011, Hennepin County Medical Center. *Approaches in Critical Care* is published twice per year by Hennepin County Medical Center, 701 Park Avenue, Minneapolis, Minnesota 55415.

# Sepsis: Four Case Reports



More than 200,000 Americans die each year from sepsis. Misdiagnosis or late diagnosis is an especially grave problem since sepsis can progress rapidly. After severe sepsis, many patients experience declines in long-term physical and mental functioning. Some patients, especially those with pre-existing chronic diseases, may experience permanent organ damage.

A high index of suspicion and a comprehensive care plan can help prevent long-term effects of sepsis. The following four Minnesota case studies describe the diagnosis and initial management of patients with sepsis arising from a variety of medical conditions.

raphy showed patchy bilateral infiltrates. (See Figure One.) Oseltamivir and broad-spectrum antibiotics were initiated. He was admitted to the medical intensive care unit for treatment of sepsis and observation of his respiratory status as it was thought he was at high risk for rapid decompensation.

The patient was maintained on high-flow oxygen by face mask until 36 hours into his hospital stay, at which point he was intubated for progressive respiratory fatigue. Blood cultures grew group A streptococcus; viral serologies sent to the Minnesota Department of Public Health returned positive for novel H1N1 influenza.

A few days after admission, he developed severe acute respiratory distress syndrome (ARDS) requiring aggressive ventilatory support including high-positive, end-expiratory pressure with numerous recruitment maneuvers, heavy sedation with continuous paralysis and inhaled epoprostenol, and a low tidal volume strategy. Proper oxygenation remained a challenge despite these numerous interventions. The patient also developed septic shock requiring vasopressors and acute tubular necrosis causing anuria, which eventually required continuous renal replacement therapy (CRRT). A course of stress-dose methylprednisolone was attempted with limited improvement.

His clinical status fluctuated day to day but after approximately ten days he seemed to be improving slowly. Based on culture results, antibiotics were narrowed. His supplemental oxygen needs lessened and he no longer required vasopressor support for his blood pressure. Conventional dialysis was started in lieu of CRRT.

He continued on this course until hospital day 22 when he rapidly decompensated with severe hypotension necessitating almost every available vasopressor, including phenylephrine, vasopressin, norepinephrine, and epinephrine as well as hydrocortisone. Extremely broad-spectrum antibiotics were started and oseltamivir was re-administered.

## Sepsis following H1N1 infection

by Brian Driver, MD  
Division of Emergency Medicine and  
Internal Medicine  
Hennepin County Medical Center

### Case report

A previously healthy 29 year-old man presented to the emergency department (ED) during the height of H1N1 influenza season in 2009 with three days of cough, fever, sore throat, upper respiratory congestion, headache, and myalgias. He was discharged to home with cough syrup, acetaminophen with codeine, loratadine, and pseudoephedrine.

Two days later, he returned to the ED with worsening shortness of breath and hemoptysis. He was febrile, tachycardic, tachypneic with moderate respiratory distress, and hypoxemic on room air. Chest radiog-

Late that morning, he went into ventricular fibrillation cardiac arrest, later found to be secondary to acute hyperkalemia and/or severe sepsis. He was resuscitated after approximately ten minutes of high-quality CPR with advanced cardiac life support. Activated protein C (APC) was subsequently started later in the day. A stool sample returned positive for clostridium difficile toxin, which was thought to be cause of his sudden decline.

On hospital day 24 an echocardiogram showed biventricular failure with an ejection fraction of 20%, which was thought to be due to stress cardiomyopathy from overwhelming sepsis. This was not the cause of his hypotension, which at this point had improved, and he was off vasopressor support.

Again, he began to slowly improve with continued intensive therapy. Antibiotics were again narrowed and eventually stopped. His pulmonary mechanics improved and he was able to be easily oxygenated. The patient's kidneys began to make urine and dialysis eventually was stopped. A repeat echocardiogram showed normal ejection fraction.

On hospital day 34, a tracheostomy was placed (see Figure Two) and, after continued improvement and adequate oxygenation off positive pressure ventilation, he was transferred to the floor on hospital day 45. On hospital day 48, he was transferred to Hennepin's Knapp Rehabilitation Center. The patient spent 11 days at Knapp regaining some of his lost strength. Mild impairments in attention and verbal reasoning, which was noted on admission, had improved by discharge. He was followed at Hennepin in the months subsequent and returned successfully to work five months after his initial admission.

### Discussion

The traditional definition of sepsis usually is thought to be hypotension in the face of a severe infection. A more formal definition conceived in 1991 distinguishes systemic inflammatory response syndrome (SIRS) from sepsis. Criteria for SIRS include:

- ▶ Body temperature greater than 38 degrees Celsius or less than 36 degrees Celsius
- ▶ Heart rate greater than 90 beats per minute
- ▶ Respiratory rate greater than 20, or hyperventilation with PaCO<sub>2</sub> less than 32 mmHg
- ▶ White blood cell count greater than 12,000/mm<sup>3</sup> or less than 4,000/mm<sup>3</sup>, or more than 10% mature neutrophils, such as bands

A patient meeting two or more of these criteria has SIRS, which can be infectious or non-infectious. Sepsis is defined as SIRS with a suspected or con-

firmed infectious source. Sepsis complicated by organ dysfunction, hypotension, or elevated lactate is termed severe sepsis. Hypotension refractory to intravenous fluid resuscitation is defined as septic shock.

Sepsis is usually thought of as a disease of elders but just five days before this patient was intubated in the intensive care unit (ICU) he was a robust, healthy young man with no medical problems. While the incidence of sepsis after the neonatal period remains low (less than one per 1,000 population per year) until age 30, a significant number (42%) of sepsis cases occur in patients younger than 65 years old. In patients older than 30, the incidence of sepsis increases slowly with age until it increases sharply at age 60. Overall mortality averages 25-30%.

Because of the high mortality associated with sepsis, rapid and proper treatment is essential. Standard treatment includes early antibiotic administration, source control, goal-directed resuscitation, and support of vital organ function. Activated protein C, as used in this case, is an option in selected patients. There are many more novel and controversial therapies that are beyond the scope of this discussion.

Early administration of broad-spectrum antibiotics is essential in the treatment of sepsis. Retrospective studies have shown increasing mortality for every hour that antibiotics are delayed and, in multivariate analysis, time-to-antibiotic-administration was the strongest predictor of eventual patient mortality.

Antibiotics are selected based on the suspected site of infection and local patterns of antibiotic resistance as well as host susceptibility and co-morbidities. The initial choice of antibiotics must be broad, covering all potential organisms, and concerns about generating antimicrobial resistance are unfounded in these dire circumstances. There is time for antibiotic narrowing when the patient stabilizes and cultures return. Cultures ideally are obtained prior to antibiotic administration.

Equally important in the treatment of sepsis is the concept of source control, which is the removal or control of the focus of infection in order to limit the ongoing inflammatory response and, in some cases, to decrease further spread of infection. Again, this is ideally completed as rapidly as possible, depending on the ease of the source-control procedure.

Interventions can be divided into three groups:

- ▶ Drainage of a local focus or abscess
- ▶ Debridement of necrotic tissue
- ▶ Removal of an infected medical device

Simple source-control procedures include replacement of Foley catheters and exchange of non-tunneled central venous catheters. More complex interventions involve removal of peritoneal dialysis catheters and tunneled central venous catheters, endoscopic relief of biliary obstruction, and percutaneous nephrostomy in pyelonephritis with obstruction. Invasive procedures include replacement of orthopedic hardware or prosthetic heart valves and debridement of necrotic tissue in intestinal ischemia and necrotizing fasciitis. Removing these foci can be life-saving. It is crucial that every patient with sepsis be evaluated for a potential focus of infection. Risks and benefits of intervention must be weighed in the context of the patient's wishes and overall clinical picture.

Early goal-directed resuscitation, an outline of which appeared in a landmark article by Rivers in 2001, should be performed concurrently with early antibiotics and source control. While the development of this protocol was a milestone for sepsis care, it is acceptable to deviate from these guidelines if patient circumstances dictate, and protocols will vary between institutions. For example, in an under-resuscitated patient in whom bedside ultrasound shows excellent myocardial contractility, inotropic agents may not be appropriate. The ideal endpoint of resuscitation is not a physiologic parameter but rather restoration of normal tissue perfusion and reduction of mortality.

Fluid resuscitation in sepsis should be generous. A goal central venous pressure (CVP) of 8-12 mmHg has been advocated but use of CVP to guide fluid therapy remains controversial. Evaluation of the inferior vena cava via bedside ultrasound also may help to noninvasively determine volume status. If hypotension persists after fluid resuscitation, vasopressor agents are appropriate. It is important to note that tissue hypoperfusion can persist despite normal physiologic parameters.

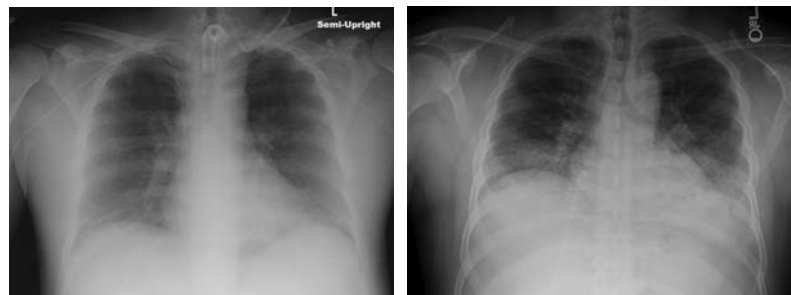
In the future, real-time tissue perfusion monitoring using near-infrared spectroscopy will likely provide an excellent endpoint. For now, however, lab tests such as lactate and superior vena cava oxygen saturation (ScvO<sub>2</sub>) are used as surrogates of tissue hypoperfusion. While the original article by Rivers, et al, guided resuscitation by serial ScvO<sub>2</sub> values, recent multivariate analysis found lactate non-clearance (defined as less than a 10% decrease) to be more predictive of in-hospital mortality than ScvO<sub>2</sub>. A 2009 randomized controlled trial demonstrated serial lactate values to be non-inferior to ScvO<sub>2</sub> as an endpoint in resuscitation.

Numerous inflammatory and cytokine changes are found in sepsis and a procoagulant state is created.

This is thought in part to be due to decreases in APC levels. When APC is administered as an intravenous infusion, it is thought to act as an anticoagulant, helping open microvasculature and increase tissue perfusion. It also may inhibit the systemic inflammatory response. The PROWESS study, published in 2001, which evaluated the safety and efficacy of drotrecogin alfa (activated) on 28-day, all-cause mortality, showed a 19.4% reduction in the relative risk of death in severe sepsis with APC treatment, with a number needed to treat of 16.4. Subgroup analysis, however, demonstrated patients with multi-organ failure and high acute physiology and chronic health evaluation II (APACHE II) scores received most of the benefit. There were numerous criticisms of this study, including mid-study protocol and master cell lot changes as well as the fact that a treatment group should not be determined by subgroup analysis. Further studies showed no benefit in children or adults with single-organ failure and APACHE II scores less than 25. Further research is ongoing, which will help determine future guidelines.

Current recommendations are to administer APC to adult patients with sepsis-induced organ dysfunction associated with a clinical assessment of high risk of death. Most of these will have APACHE II scores more than 25 or multiple organ failure. There are numerous contraindications to APC, mostly revolving around conditions that increase the risk of bleeding, which should be referenced prior to APC administration. Patients should receive APC within 24 hours of the onset of sepsis for maximal benefit.

Sepsis can generate a myriad of clinical scenarios and prompt and proper treatment is essential. Executing the objectives of early antibiotics, source control, and goal-directed therapy will help save lives. New and controversial therapies are both here and forthcoming, though their use is currently applicable only in select patients. ■



**Figure One.** The chest radiograph at admission (image at left) showed extensive bilateral basilar infiltrates consistent with severe pneumonia and developing ARDS.

**Figure Two.** While the image (at right) shows that infiltrates are significantly improved at discharge, the patient now has both a tracheostomy tube and right-sided peripherally inserted central catheter as evidence of his arduous ICU course.

## Sepsis in an immunosuppressed cancer patient

by Lisa Hayden, MD

Departments of Emergency Medicine and Internal Medicine  
Hennepin County Medical Center

### Case report

A 59 year-old male with a history of metastatic squamous cell lung carcinoma presented to the emergency department (ED) with altered mental status, as reported by his wife. At the time, the patient was undergoing chemotherapy with carboplatin/gemcitabine. Paramedics reported an initial systolic blood pressure of 88 mmHg and a heart rate of 104 beats per minute.

During his initial ED evaluation, he became unresponsive. Cardiac monitoring revealed thirty seconds of asystole and he was apneic. He regained consciousness with a small dose of Narcan® and a brief period of bag valve mask ventilation to restore respiration. Emergent intubation was not required initially. Administration of two liters of normal saline improved both blood pressure and mentation. An indwelling Foley catheter was placed with an initial 10 milliliters of urinary output.

A chest radiograph (Figure One) showed probable pneumonia, and broad-spectrum antibiotics were initiated. Initial blood tests showed acute renal failure with a creatinine of 3.6, hyperkalemia at 5.8, and a metabolic acidosis with a bicarbonate level of 15 and an anion gap of 24. Complete blood cell count showed a white blood cell count of 200/mm<sup>3</sup>, hemoglobin of 5.7 g/dL, and platelet count of 6,000/mm<sup>3</sup>. The initial international normalized ratio was 2.2 and the lactate was 2.2 mmol/L. The patient continued to receive aggressive fluid resuscitation, which continued to improve blood pressure. He was transferred to the medical intensive care unit where he again decompensated and required intubation for respiratory failure.

After intubation, the patient again became hypotensive. Central venous and radial arterial lines were placed for hemodynamic monitoring. The initial central venous pressure (CVP) reading was 8 mmHg. Fluids were continued and packed red blood cells were given to treat his anemia. Vasopressor support with norepinephrine resulted in an initially adequate blood pressure response. Urine output remained adequate. Filgrastim for neutropenia was initiated upon recommendation of the patient's oncologist.

The clinical course of this patient was complicated by the development of atrial fibrillation with a rapid ventricular response, managed with aggressive electrolyte replacement and esmolol, which was given

with phenylephrine to maintain blood pressure. On hospital day three, the patient developed gastrointestinal hemorrhage, managed with Protonix and octreotide and multiple blood product transfusions, including packed red blood cells, platelets, and fresh frozen plasma. He did not receive endoscopy or colonoscopy and his bleeding resolved spontaneously.

Initial blood cultures showed pan-sensitive escherichia coli and appropriate antibiotics were maintained. Despite these aggressive measures, on hospital day four, the patient again became hypotensive and manifested worsening multi-organ failure and probable disseminated intravascular coagulopathy with elevated coagulation studies and profound thrombocytopenia. He required additional vasopressor support with norepinephrine and vasopressin. Stress-dose steroids were also given. His acidosis worsened and he became anuric. A Quinton™ femoral catheter was placed in anticipation of dialysis. On hospital day five, his family made the decision to withdraw care and the patient died.

### Discussion

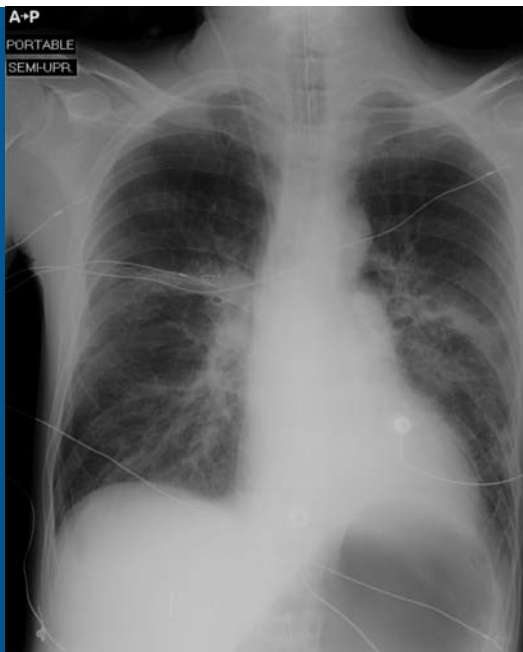
Early, goal-directed therapy for sepsis, outlined by Rivers in 2001, emphasized early and aggressive fluid management to achieve a CVP  $\geq$  8-12, vasopressors to maintain mean arterial pressure (MAP)  $\geq$  65 mmHg, and inotropes/blood transfusion to maintain SvcO<sub>2</sub>  $\geq$  70%. Further guidelines were established in 2008 by the Surviving Sepsis Campaign, which is an initiative of the European Society of Intensive Care Medicine, the International Sepsis Forum, and the Society of Critical Care Medicine.

The Surviving Sepsis Campaign guidelines provide evidence-based recommendations for sepsis management that can be applied to any patient presenting with severe infection and signs of end-organ compromise. The cornerstones of early management include source recognition and control, placement of appropriate hemodynamic monitoring devices, aggressive fluid resuscitation, early, broad-spectrum antibiotics, vasopressor support to maintain a MAP  $\geq$  65 mmHg, early airway management, thoughtful and appropriate use of red blood cell transfusion for anemia, and indications for inotropes. Further considerations include glucose control, stress ulcer and deep-vein-thrombosis prophylaxis, indications for steroids and renal replacement therapy, and the use of activated protein C. A final and important aspect of these guidelines is the discussion of a limited-care plan when the patient's prognosis is poor.

Guidelines are useful in directing early and aggressive intervention in patients who have a severe infection. However, as the case above illustrates, morbidity and

mortality may occur despite appropriate management. This case is an example of septic shock complicated by immunosuppression from chemotherapy.

The patient's immune system became overwhelmed and he rapidly progressed to multi-organ failure refractory to aggressive therapy. Despite appropriate therapy in accordance with recent guidelines, the patient did not recover. Given the limited life expectancy due to metastatic cancer, the decision to terminate care was considered appropriate. ■



**Figure One.** Chest x-ray at the time of admission, showing perihilar and bibasilar opacities with increased interstitial markings and Kerley B lines.

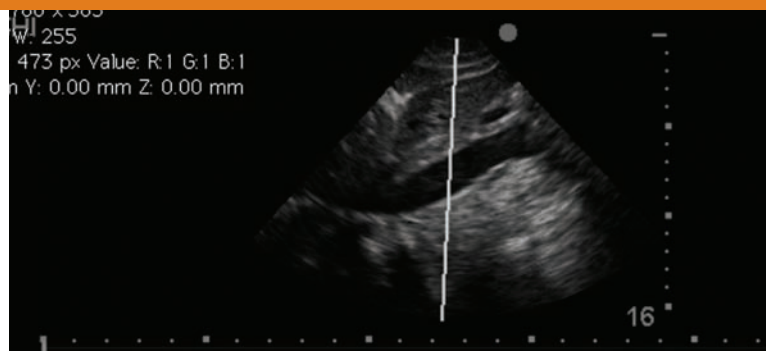
## Post-partum sepsis

by Rob Beyer, MD and Jamie Karambay, MD  
Department of Emergency Medicine  
Hennepin County Medical Center

### Case report

A 22 year-old healthy female presented to the emergency department (ED) with a low-grade fever that had been present for three days. A rash had appeared the previous night on her hands and, when she woke up in the morning, the rash covered her entire upper body, face and upper extremities. Thirteen days prior to presenting to the ED, the patient had an uncomplicated vaginal delivery. This was her first pregnancy. She was doing well post-partum with exception of minimal vaginal bleeding and a persistent malodorous vaginal discharge.

Initially, her blood pressure in the ED was 84/59 mmHg, pulse 138, respiratory rate 24, SpO<sub>2</sub> 98%, and temperature of 39.1 degrees Celsius. Her examination



**Figure One.** This ultrasound image shows a full inferior vena cava that does not collapse with spontaneous respirations.

was remarkable for a sand-paper-like, erythematous rash over her upper body involving her face, extremities, and torso. Her skin was warm and dry. She had weak peripheral pulses but her mentation was normal. Labs were drawn, including two sets of blood cultures, and the patient was given acetaminophen, clindamycin, gentamycin, and four liters of normal saline with minimal improvement in blood pressure or heart rate.

Because the etiology of patient's hypotension was unknown, a bedside cardiac ultrasound was performed, which revealed a full inferior vena cava, non-collapsing with inspiration and expiration. (See Figure One.) Her heart was hyperdynamic and she had no ultrasonic evidence of intra-abdominal or pelvic free fluid. This suggested that the patient's preload and cardiac function were adequate and that her hypotension was from an afterload problem consistent with distributive shock and sepsis. She was transferred to the stabilization room for further resuscitation and hemodynamic monitoring.

In the stabilization room, her vital signs were blood pressure 88/37, pulse 148, respiratory rate 18, and SpO<sub>2</sub> of 96%. She had two large-bore intravenous lines placed and was given two additional liters of normal saline on pressure bags. A left triple-lumen central venous catheter was placed under ultrasound guidance. Levophed® was begun.

Her pelvic examination revealed no retained foreign bodies, no cervical motion tenderness, and no adnexal tenderness or masses on exam. However, the exam was remarkable for a purulent bloody vaginal discharge. A sample of the discharge was sent for Gram stain and culture. A Foley catheter was placed with the return of clear urine. She received two more liters of normal saline and her blood pressure improved to 132/67. She received a total of eight liters of normal saline while in the ED.

When her labs returned, they revealed a white blood cell count of 19.8 and stable hemoglobin. A computed tomography scan of her pelvis and abdomen did not reveal a source of her infection. She was admitted to

the medical ICU with a presumed diagnosis of toxic shock syndrome (TSS) vs. puerperal sepsis.

The patient remained on several antibiotics in the intensive care unit, including clindamycin, vancomycin, and Zosyn® (piperacillin and tazobactam injection). After 36 hours the patient was tapered off vasopressor support. The rash resolved on hospital day two. The patient's blood and urine cultures returned negative. Her vaginal swab returned with "normal flora" including Gram-positive cocci; however, further speciation was not performed. The patient was transferred out of the ICU and discharged several days later with the diagnosis of puerperal sepsis with a component of TSS.

### Discussion

Toxic shock syndrome is a toxin-mediated, systemic inflammatory response syndrome manifested by invasive infection with *Staphylococcus aureus* (*S. aureus* or staph) or Group A streptococcus (GAS). The syndrome gained notoriety in 1980 when 812 cases of TSS were reported in previously healthy females and were associated with the use of high-absorbency tampons. Fifty percent of TSS cases are nonmenstrual cases affecting both sexes. The Centers of Disease Control (CDC) estimate 220 cases of postpartum sepsis due to GAS each year.

Staph and GAS are common pathogens that less commonly cause severe invasive disease. Following invasive infection and significant bacterial burden, exotoxins are released that act as superantigens and activate large numbers of T cells. This leads to the release of large quantities of inflammatory cytokines and manifests as shock and multi-organ failure. Patients commonly present with the classic triad of fever, hypotension, and skin manifestations. A diffuse macular erythroderma often is followed by desquamation one to three weeks later. Vomiting, diarrhea, renal failure, coagulopathies, altered mental status, and other symptoms indicative of multiple-organ failure are often experienced.

Staphylococcal cases are commonly associated with superinfection of various wounds, burns, or surgical sites whereas GAS cases are associated with more severe soft-tissue infections, such as necrotizing fasciitis. Staphylococcal and streptococcal cases are slightly distinct with staph more often affecting younger and female patients. In GAS cases, a rash is less common; very painful soft-tissue infections are more frequent. Mortality for GAS is significantly higher at 30-70%.

The diagnosis is based upon the clinical presentation using a CDC case definition, although cases are

variable, and TSS may be present even if all criteria are not satisfied. The mainstay of treatment is supportive care with extensive fluid replacement, sometimes 10 or more liters a day. Vasopressor support should be initiated if shock is refractory to fluids. Surgical consultation for debridement is important, especially in streptococcal cases. Clindamycin is considered first-line treatment for TSS due to its ability to suppress toxin synthesis. Various regimens have been proposed using vancomycin, piperacillin/tazobactam, or a carbapenem in addition to clindamycin. Intravenous immunoglobulin has been suggested (though the benefit is unproven) for cases refractory to antibiotics fluids and vasopressors. ■

### Peritoneal septic shock

by Justin Kane, MD  
Departments of Emergency and Internal Medicine  
Hennepin County Medical Center

#### Case report

A 56 year-old male with a Stage IVB diffuse large B-cell non-Hodgkin's lymphoma presented to the ED with abdominal pain. He had prior treatment including R-CHOP chemotherapy but PET-CT scans performed earlier in the month had demonstrated extensive peritoneal neoplastic soft-tissue thickening in the abdomen; therefore, two days prior, he had been switched to a R-ICE chemotherapy regimen. The radiology report at that time did not mention peritoneal fluid.

On the day of admission, he reported worsening lower abdominal pain, nausea, and non-bloody emesis. Initially he was afebrile with stable vital signs including blood pressure of 110/75 mmHg and a heart rate in the 110s. A CT scan of the abdomen was completed showing possible omental metastasis and new peritoneal fluid not consistent with blood. Blood and urine cultures were obtained. During his evaluation, he acutely decompensated with an altered mental status, a systolic blood pressure in the 70-80s, and a heart rate to the 140s. He became febrile to 103.3 degrees Fahrenheit. He was transferred to the stabilization room, where improved vascular access was obtained via a second large-bore intravenous line. Fluids were initiated wide open (three liters initially on pressure bags) and a focused assessment with sonography in trauma (FAST) exam was done, which demonstrated peritoneal fluid. (See Figure One.)

Antibiotic treatment was started for presumed sepsis. Initial antibiotics included Zosyn® (piperacillin and tazobactam injection), vancomycin, and tobramycin to cover resistant pseudomonas, which was a



**Figure One.** This image shows free peritoneal fluid in Morison's pouch.

previous problem noted on chart review. After discussion of code status, the patient was orotracheally intubated for airway protection and increasing respiratory distress in the setting of developing systemic inflammatory response syndrome. A central line was placed for central venous pressure (CVP) monitoring and improved access. As the patient's vital signs were not improving with fluids after six liters, Levophed® was started and stress-dose steroids were given.

Upon arrival to the medical intensive care unit, CVP monitoring demonstrated continued fluid depletion. However, with continued hypotension, tachycardia, and decreased urine output, vasopressin was started in addition to the Levophed®. His vital signs began to stabilize with these measures.

With fluids continuing, attention was focused on finding a source of his infection. Bedside ultrasound again revealed peritoneal fluid. This was felt to be new, given recent radiographic examination, and a likely infectious source. A sample was obtained by ultrasound guidance. Within the hour, results returned on Gram stain suggested a polymicrobial peritoneal infection. As a result, despite no free air noted in the abdomen during the CT scan, surgery was consulted and the patient underwent an emergent exploratory laparotomy.

No viscus perforations were evident on inspection and the tumor load did not appear to be as high as anticipated. The bowel was poorly perfused but not necrotic. In the meantime, Gram stain from the blood returned, showing Gram-negative rods. An infectious disease specialist consulted in the case recommended doripenem, daptomycin, ciprofloxacin, and continued Tobramycin coverage. With continued aggressive fluid resuscitation, he received close to 17 liters within the first 24 hours of hospitalization. His CVP normalized, his urine output returned, and he was slowly weaned off Levophed®. Eventually *Escherichia coli*

(*E. coli*) grew out of his peritoneal fluid and his initial blood culture growth, which was sensitive to ceftriaxone.

### Discussion

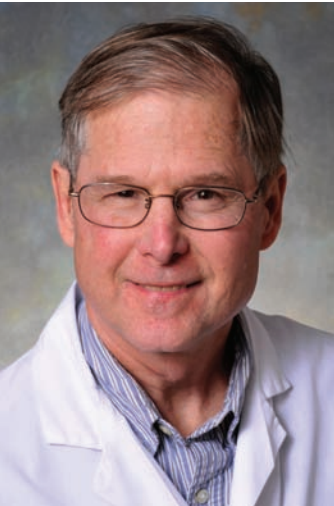
This patient's case illustrates distributive septic shock secondary to Gram-negative peritonitis. The aggressive management following his rapid deterioration while in the ED demonstrates how early, goal-directed therapy with fluids, antibiotics, and pressor support can help curb the end-organ effects of low perfusion commonly observed in septic shock. A crucial aspect of this case was identification of the intra-abdominal source of infection. The early decision to take the patient to the operating room (OR) was based on early information that the ascitic fluid contained multiple organisms. The distinction between spontaneous vs. secondary bacterial peritonitis is important. If secondary bacterial peritonitis is missed, mortality reaches 100% without surgical intervention. Yet, operative intervention on secondary peritonitis also carries a high mortality.

While this patient did not have observable free air in the abdomen, the report of possible polymicrobial ascitic fluid led to a strong suspicion that he may have a perforation (possibly a micro-perforation). Polymicrobial or fungal ascitic fluid indicates secondary peritonitis. In review, it was a single organism that infected both the peritoneum and blood but the decision to go to the OR was based on the critical clinical presentation, new ascitic fluid not previously seen on a very recent PET-CT scan, the suspicion of polymicrobes on Gram stain, and the consequence of missing a perforation that would almost certainly be fatal. ■

### Bibliographies/Suggested Readings

- Angus DC et al. Epidemiology of severe sepsis in the United States: Analysis of incidence, outcome, and associated costs of care. *Crit Care Med* 2001;29:1303-10.
- Dellinger R, Levy M, Carlet J, et al. Surviving Sepsis Campaign: International guidelines for management of severe sepsis and septic shock: 2008. *Crit Care Med* 2008;36:296-327.
- Jean-Louis V et al. Evolving concepts in sepsis definitions. *Crit Care Clin* 2009;25:665-675.
- Jones AE et al. Sepsis-induced tissue hypoperfusion. *Crit Care Clin* 2009;25:769-779.
- Marshall JC et al. Source control in the management of severe sepsis and septic shock: An evidence-based review. *Crit Care Med* 2004;32:S513-26.
- Rivers E et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med* 2001;345:1368-77.
- Toussaint S et al. Activated protein C for sepsis. *N Engl J Med* 2009;361:2646-52.

## Q and A with James Leatherman, MD



James W. Leatherman, MD

In his 25-year career, intensivist James Leatherman, MD, has treated hundreds of patients with sepsis. As the director of the medical intensive care unit at Hennepin County Medical Center, he has seen an evolution in how—and, more important, how fast—treatments are provided to patients with sepsis.

### ***How has sepsis care changed during your career?***

Antibiotics are a cornerstone in the treatment of sepsis but until relatively recently the critical importance of rapid administration had not been emphasized. Current guidelines recommend giving initial broad-spectrum antibiotics within one hour of presentation. From a practical standpoint, this means patients need to receive antibiotics in the emergency department. Antibiotics alone are inadequate to manage sepsis; identification and elimination of the infectious source also is essential and may require surgical intervention. (See case report on page 7.) Guidelines recommend that a definitive source control ideally should be accomplished within six hours of presentation. Again, while the importance of source control has been known for many decades, the emphasis more recently has been on the importance of an expedited approach.

Another change has been the speed with which hemodynamic resuscitation is accomplished. Although fluids and vasopressors have been used for many decades for septic shock, current guidelines emphasize rapid, large-volume fluid resuscitation in the emergency department and early use of vasopressors as needed. Current sepsis guidelines also emphasize targeting a central venous pressure (CVP) of 8-12 mmHg and a superior vena cava oxygen saturation (ScvO<sub>2</sub>) under 70% rather than just trying to achieve a mean arterial pressure goal.

According to the guidelines for early goal-directed therapy, after adequate fluid

resuscitation, the goal of a ScvO<sub>2</sub> under 70% may be achieved with red blood cell transfusions if hematocrit is less than 30% or by dobutamine if the hematocrit is over 30%. However, despite these published guidelines, it remains controversial whether targeting a specific CVP and use of red blood cell transfusions and dobutamine to achieve a ScvO<sub>2</sub> target are really beneficial. While pretty much everyone agrees on the importance of rapid administration of broad-spectrum antibiotics, aggressive fluid resuscitation, and use of pressors (usually norepinephrine) when fluids are inadequate to reverse hypotension, there is divided opinion on whether you need to routinely measure CVP or to give either blood transfusions or dobutamine during the first few hours of management.

Another change in management has been in the use of corticosteroids. Some patients with severe sepsis have relative adrenal insufficiency and may benefit from steroids. The most recent guidelines call for administration of empiric steroids (hydrocortisone 50 mg every 6 hours) for the subset of patients whose shock is proving difficult to reverse with fluids and vasopressors but not to those patients with stable hemodynamics.

In other words, steroid use is a clinical judgment call. The good news is that while steroids do not seem to change the outcome of sepsis when applied to all comers, they do not seem to have a negative impact. Therefore, the clinician does not need to fret too much as to whether steroids are necessary and it seems perfectly reasonable to give them to patients whose shock is difficult to reverse.

Stress hyperglycemia is common in sepsis, especially if steroids are used. Currently, a middle ground between tight and exceedingly loose glucose control is advocated, targeting a glucose in the range of 140-180 mg/dl.

Other changes in management of patients with severe sepsis relate to important

aspects of supportive care. For example, currently nutrition is almost always given enterally and is usually started within a day or two of admission. Ventilator support for acute lung injury, a common occurrence in severe sepsis, has also undergone dramatic changes in the last two decades. Previously, large tidal volumes were used to achieve a normal blood gas. Today, low tidal volume ventilation (~6 ml/kg of ideal body weight) is the standard of care. Management of renal failure has also changed. Hemodynamically compromised patients are now routinely treated with continuous renal replacement rather than less well tolerated intermittent hemodialysis.

Finally, newer therapies for sepsis are available or under study. An example is the use of activated protein-C as described in the case report on sepsis following H1N1 infection. (See page 2).

***Is sepsis often undiagnosed or diagnosed later than it should be?***

It can be. Given the enormous impact of severe sepsis, which kills more patients each year than myocardial infarction, there has been increased emphasis on getting clinicians to recognize it earlier so that we can intervene before potentially irreversible organ injury occurs. Sepsis sometimes isn't recognized because a patient may not have a fever at presentation and, in the absence of a fever, clinicians may not consider an underlying infection. Common features of severe sepsis include alteration in mental status, lactic acidosis, and hypotension. When otherwise unexplained, these features should alert the clinician to the possibility of sepsis in the afebrile patient.

***How has the recent emergence of new infectious diseases like H1N1 affected sepsis care?***

Certainly, with an H1N1 (or influenza A) outbreak, affected patients are at risk for secondary bacterial pneumonias and may develop severe sepsis. I think a bigger threat than new pathogens may be the development of antibiotic resistance in our old bacteria. We certainly saw this with methicillin-resistant staphylococcus aureus (MRSA). At one time, MRSA was relatively uncommon and was limited to patients who were in hospitals or long-term health care facilities. Now MRSA is well established in the community and is a common cause of community-acquired skin and soft-tissue infection and may be a cause of pneumonia occurring outside the hospital.

Of even greater potential concern is the rise of highly resistant Gram-negative organisms. Although this has not yet become a major problem locally, we are

starting to see more resistant Gram-negatives than we did a few years ago. It is likely only a matter of time before we begin to see increasing numbers of resistant Gram-negative infections as the cause of severe sepsis. At present, there aren't many new antibiotics directed against Gram negatives in the pipeline and this has created a lot of concern in the infectious disease community.

Fortunately, resistant Gram-negative infections have been pretty much confined to hospitals and health care facilities but, as was the case with MRSA, it is possible that these could begin to be a problem in the community. This is why it is important for all of us to practice good antibiotic stewardship. While broad-spectrum antibiotics are essential when patients first present with severe sepsis—if you pick the wrong drug the patient may not survive—it is important that we change to the narrowest-spectrum agent that covers the organism that is eventually grown in culture.

***Are there any major changes in sepsis care coming down the pipeline?***

There have been a number of different antibody therapies tried over the years. The theory was that certain monoclonal antibodies would block key cytokines involved in the body's response to sepsis. At one time, there was extraordinary excitement about this idea but it turned out that none of the antibodies were effective. Because of this, there is currently a lot of skepticism about new treatments. Anything that seems promising will be subjected to very rigorous evaluation. But that's as it should be. Sepsis is probably the most complex critical illnesses we see in the intensive care unit and it seems unlikely that a single antibody or molecule will be able to magically reverse the process.

*“...a high index of suspicion for diagnosing sepsis is important so that empiric treatment can be given as soon as possible. Ideally, antibiotics should be given within an hour of presentation.”*

### ***As an intensivist, what do you tell providers about initial management of sepsis?***

First, a high index of suspicion for diagnosing sepsis is important so that empiric treatment can be given as soon as possible. Ideally, antibiotics should be given within an hour of presentation. The initial antibiotics are designed to cover all possible pathogens; this usually requires two and sometimes three antibiotics to cover Gram positives, including MRSA, and Gram-negatives.

Also, you need to cover anaerobes if there is a suspected intra-abdominal source and Legionella if there is a pulmonary source. There's no reason to be gunshy about giving a lot of antibiotics early on; we should always narrow them down later. Maintain a low threshold for intubation and mechanical ventilation in a patient with increased work of breathing, severe hypoxemia, and marked encephalopathy. Also, early and aggressive fluid administration is important; hypotensive patients with suspected sepsis should receive two liters initially and then additional saline boluses based on response. Fluid-refractory hypotension usually is due to excessive arterial vasodilation and a pressor such as norepinephrine often is necessary to keep the mean arterial pressure above 65 mmHg.

Published guidelines for fluid resuscitation based on CVP remain somewhat controversial. As a general rule, it is better to err on the side of more rather than less. It also has been advocated that the  $SvcO_2$  be measured; if it is below 70% this suggests that  $O_2$  delivery is inadequate. A somewhat complex and controversial pathway based on the CVP and the  $SvcO_2$  has been advocated—so-called early goal-directed therapy—which may encourage red blood cell transfusion or dobutamine or both in the early management.

Personally, I am not convinced of the need for this type of algorithmic approach and ongoing studies may help clarify whether it is necessary. However, if a low  $SvcO_2$  persists after adequate fluid resuscitation, cardiac output likely is inadequate. Either more volume loading or possibly inotropic therapy may be needed.

For me, the real take-home message in managing sepsis is the following: give antibiotics early, give lots of fluid, and support blood pressure with a vasopressor when hypotension can not be reversed with fluids. Whether blood transfusions or dobutamine have a key role in the early management of sepsis remains to be seen. ■

## **Initial Resuscitation (First Six Hours) for Sepsis**

### **Guidelines as Summarized by the Surviving Sepsis Campaign**

Begin resuscitation immediately in patients with hypotension or elevated serum lactate  $\geq 4$  mmol/L; do not delay pending ICU admission.

#### **Resuscitation goals:**

- ▶ Central venous pressure (CVP) 8-12 mmHg\*\*
- ▶ Mean arterial pressure  $\geq 65$  mmHg
- ▶ Urine output  $\geq 0.5$  ml/kg/hr
- ▶ Central venous (superior vena cava) oxygen saturation  $\geq 70\%$  or mixed venous oxygen saturation  $\geq 65\%$

#### **If venous $O_2$ saturation target not achieved:**

- ▶ Consider additional fluid;
- ▶ Transfuse packed red blood cells if required to hematocrit of  $\geq 30\%$ ; AND/OR
- ▶ Dobutamine infusion max 20 mcg/kg/min

\*\* A higher target CVP of 12-15 mmHg is recommended in the presence of mechanical ventilation or pre-existing decreased ventricular compliance.

Source: Guidelines for Managing Severe Sepsis and Septic Shock brochure. Summarized by the Surviving Sepsis Campaign International Guidelines for Management of Severe Sepsis and Septic Shock: 2008, condensed from Dellinger RP, Levy MM, Carlet JM, et al: Surviving Sepsis Campaign: International guidelines for management of severe sepsis and septic shock. Intensive Care Medicine. 2008; 34:17-60 and Crit Care Med. 2008; 36(1): 296-327.



Hennepin County **Medical Center**  
**Hennepin Connect**  
Physician Referral & Consult Line

---

Rapid access to Hennepin physicians  
for referrals and consults

**Services available 24/7**  
**1-800-424-4262**

6 1 2 - 8 7 3 - 4 2 6 2



## EMS Perspectives: Prehospital management of suspected sepsis

by Robert Ball, EMT-P  
Operations Supervisor, Emergency Medical Services  
Hennepin County Medical Center

*“In severe sepsis, the burden of the infection and the body's response result in an insult to other organ systems.”*

When we think of hazards in the community, traffic-related injuries and deaths often dominate the discussion. But one killer, sepsis, claims over five times as many lives. In the U.S., over 750,000 patients are treated for severe sepsis each year. Of these, about 215,000—nearly 30 percent—die.

While hospitals often emphasize the nosocomial infections that can lead to sepsis, two-thirds of sepsis cases arrive in the emergency department, most transported by ambulance. To make headway in the fight against sepsis, it's helpful to have a basic understanding of the illness, its triggers, symptoms, and treatment.

### **Sepsis definition**

Put simply, sepsis is the body's response to infection. In uncomplicated sepsis, infection may be localized, with the same redness and swelling of an infected minor laceration, or it may be generalized, as seen in patients with gastroenteritis and other illnesses. Every year, millions of people feel the effects of uncomplicated infection without requiring medical care. However, in some cases, the body's ability to combat

the infection falters or is overwhelmed by the infectious agent. At this point, the infection can develop into severe sepsis.

In severe sepsis, the burden of the infection and the body's response result in an insult to other organ systems. Patients developing severe sepsis appear very ill and often struggle to manage simple daily care functions. They often present as feverish but some patients—particularly those who may be developing septic shock—may be afebrile, or even mildly hypothermic.

The most severe form of sepsis, septic shock, combines the insult on multiple organs due to infection with hypotension unresponsive to fluid challenges alone. Patients in septic shock may rapidly progress into respiratory or cardiac arrest.

### **Identifying sepsis**

Unlike trauma, strokes or myocardial infarctions, sepsis requires medical detective work; definitive diagnosis, which requires lab work and cultured specimens, can't be made in the field. However, sepsis's high morbidity and mortality requires

the emergency services provider to identify those patients who are at risk of severe sepsis. Several indicators should raise suspicion for sepsis:

- ▶ **Age.** Both infants (although not premature infants) and elders are at higher risk for severe sepsis. While infants are a small percentage of sepsis patients, they have less capacity to compensate and can become very ill very quickly. On the end of the spectrum, over half of the patients seen for severe sepsis in the U.S. are over the age of 65.
- ▶ **Immunocompromised status.** Patients who have acquired immune deficiency syndrome, hepatitis, or those receiving chemotherapy are often at high risk for sepsis.
- ▶ **Invasive treatments.** The body normally does an excellent job of protecting itself from infectious agents but some necessary medical treatments defeat these defenses and place the patient at risk for sepsis. Tracheostomies, urinary catheters, and long-term intravenous catheters all can be access points for infection.

The next step in identifying potential patients with sepsis is to look for signs of infection:

- ▶ **Fever.** Patients in severe sepsis or septic shock may no longer be febrile, however most were at one point in the course of their illness.
- ▶ **Malaise.** Patients may have difficulty with the simplest tasks, such as getting to the bathroom or feeding themselves.
- ▶ **Localized signs.** Depending on the cause of the infection, you may see signs or symptoms specific to the body area involved. Sepsis in the lungs may cause dyspnea, rapid and shallow respiration, and a productive cough of virulent (pus-filled) sputum. Urological sepsis may cause pain/discomfort with urination, pus and/or sediment in the urine, and foul-smelling urine. Patients who perform intermittent catheterization or have an in-dwelling catheter are chronically at risk for urinary tract infections and sepsis. Infections to the central nervous system, such as meningitis or encephalitis, can lead to sepsis and cause headaches, neck pain, and/or stiffness. Skin infections leading to sepsis may involve redness and/or rashes.

### Treatment

Prehospital care for sepsis involves supporting respiration and circulation. Unless the patient is in extremis or in respiratory arrest, respiratory support will largely involve administering low-flow oxygen in a manner best tolerated by the patient. For most patients, this will mean providing four to six liters per minute of oxygen by nasal cannula.

Basic care for circulatory support involves positioning the patient comfortably on the stretcher. They may need or want their head elevated to a Fowler (semi-recumbent) position to allow them to breathe easier. For advanced providers, establishing an intravenous line and providing fluid support is an important step. The amount of fluid varies. For patients in septic shock, it's not uncommon to run large-bore intravenous lines wide-open and/or to administer vaso-pressors such as dopamine.

Ultimately, the goal is to maintain perfusion without overdoing it. The amount of time the patient spends with a mean arterial pressure (MAP) of <65 has been identified as a predictor of mortality. By the same token, sepsis patients over-treated with pressors to the point of having a MAP of >70 are also known to have a higher incidence of mortality and morbidity due to the effects of the pressor agents on the body.

Calculating MAP is relatively simple. The equation to estimate MAP is  $[(\text{diastolic BP} \times 2) + \text{systolic BP}] / 3$ . So, for example, a textbook "normal" blood pressure of 120/80 has a MAP of 93.3  $[(2 \times 80=160) + 120 = 280] / 3 = 93.3$ . To extrapolate, maintaining a MAP over 65 may involve maintaining a blood pressure akin to approximately 90/50. Beyond that, the goal is to have the body recover and increase MAP as it heals.

### One final consideration

As emergency services providers, we care for many patients with a myriad of infections and contagious illnesses. The possible presence of infectious agents has made body substance isolation (BSI) or universal precautions an important standard of care for all patients but this is particularly important in the suspected sepsis patient. Proper precautions will reduce your risk of becoming septic as well.

While sepsis may not receive the attention that cardiac conditions and trauma care receive, sepsis annually claims almost 32 times the U.S. combat deaths to date in both Iraq and Afghanistan. Quickly identifying potential sepsis patients, providing supportive care, and quickly moving them to facilities that can provide definitive care can help make sure more of these patients walk out of the hospital. ■

### Bibliography/Suggested Readings

- Wang HE, Shapiro NI, Angus DC, Yealy DM. National estimates of severe sepsis in United States emergency departments. *Crit Care Med* 2007; 35(8):1928-1936.
- Surviving Sepsis Campaign, Society of Critical Care Medicine 2010. Available at [www.survivingsepsis.org](http://www.survivingsepsis.org).
- Takala J. Should we target blood pressure in sepsis? *Crit Care Med* 2010;38(10):S613-S619.

**To confirm hours and location of courses and to register, visit [www.hcmc.org](http://www.hcmc.org) and click on "Professional Education and Training."** For questions or additional information, contact Susan Altmann in the medical education department at Hennepin County Medical Center at (612) 873-5681 or [susan.altmann@hcmcd.org](mailto:susan.altmann@hcmcd.org) unless another contact person is provided. Classes are at Hennepin unless otherwise indicated. Many courses fill quickly; please register early to avoid being wait-listed.

## **Advanced Cardiac Life Support for Providers (AHA)**

February 9 and 10, March 29 and 30, April 19 and 20, May 17 and 18

## **Off-Site Advanced Cardiac Life Support (ASHI)**

March 2 and 3 in Redwood Falls, April 8 and 9 in Marshall

## **Advanced Cardiac Life Support Provider Renewal (AHA)**

February 10, March 30, April 20, May 18

## **Advanced Cardiac Life Support Provider Renewal (ASHI) for Hennepin Staff**

January 18, February 16, April 6, May 12

## **Advanced Cardiac Life Support for Experienced Providers (AHA)**

January 19, February 28

## **Advanced Cardiac Life Support Instructor (AHA)**

March 8 and 9

## **Advanced Cardiac Life Support Instructor Renewal (AHA)**

March 9

## **Emergency Medical Technician Basic**

February 1 and March 31, March 21 and April 8

## **Emergency Medical Technician Refresher**

January 24 and 26, February 2 and 4, March 2 and 4, March 15 and 17 (held at the Edina Training Center), March 17 and 19

## **First Responder**

April 11 and 15

## **First Responder Refresher**

March 10 and 11

## **Healthcare Provider Cardiopulmonary Resuscitation**

January 10, March 7, April 18

## **Heartsaver Automated External Defibrillator and Cardiopulmonary Resuscitation**

February 7

## **Cardiopulmonary Resuscitation Instructor**

April 26

## **Cardiopulmonary Resuscitation Instructor Renewals**

April 26

## **Cardiopulmonary Resuscitation/Basic Life Support for Hennepin Staff**

January 12, February 8, March 2, April 12, May 11

## **Infant-Child Cardiopulmonary Resuscitation**

January 21, February 9, March 18, April 13, May 20

## **MD Cardiopulmonary Resuscitation for Hennepin Staff**

January 7, February 4, March 4, March 16, April 1, April 20, May 6

## **Advanced Pediatric Life Support**

March 22 and 23

## **Trauma Nursing Core Course**

May 24 and 25

## **Trauma Nursing Core Course Renewal**

January 20, May 4

## **Advanced Trauma Life Support**

January 27 and 28, March 15 and 16, May 2 and 3

## **Pediatric Advanced Life Support for Providers (AHA)**

February 1 and 2, March 17 and 18, May 9 and 10

## **Pediatric Advanced Life Support Renewal (AHA)**

February 2, March 18, April 27, May 10

## **Pediatric Advanced Life Support Instructor (AHA)**

May 26 and 27

## **Pediatric Advanced Life Support Instructor Renewal (AHA)**

May 27

# News Notes



*Minnesota's...*

## Level I Pediatric Trauma Center

**Little Lives. Big Futures.**

### Hennepin is verified as a Level I Pediatric Trauma Center

In fall 2010, Hennepin County Medical Center was verified as a Level I Pediatric Trauma Center by the American College of Surgeons (ACS). This verification—the highest possible—recognizes Hennepin's distinctive expertise in caring for critically ill and injured children.

In 1989, Hennepin became Minnesota's first Level I Trauma Center for both adults and children. Though Hennepin has been providing pediatric trauma services for many years, the ACS recently changed their pediatric trauma criteria and encouraged adult trauma centers that also took care of many children to pursue a separate verification.

Providing service to the community as a Level I Pediatric Trauma Center means that Hennepin provides:

- ▶ 24-hour in-house coverage by general surgeons
- ▶ Prompt availability of physicians from varying specialties, including orthopedic surgery, neurosurgery, plastic surgery, anesthesiology, emergency medicine, radiology, oral and maxillofacial surgery, and pediatric critical care

- ▶ A pediatric operating room that is always available
- ▶ Priority status for pediatric lab work and radiology
- ▶ Experienced, highly trained pediatric trauma nursing staff and trauma nurse clinicians

In addition, Hennepin is the home of a nationally recognized Traumatic Brain Injury Center, which includes a pediatric brain injury program.

### Most Americans are not familiar with the term “sepsis”

Survey results released in October 2010 showed that three out of five Americans were not familiar with the term “sepsis,” even though the condition kills 200,000 Americans per year. Among older adults, the lack of familiarity was even more widespread.

The study, commissioned by the Feinstein Institute for Medical Research, part of the North Shore-Long Island Jewish Health System, involved 1,000 American adults.



Photo on left: New Brooklyn Park Clinic opened in November.

Photo on right: Officials cut the ribbon at Whittier Clinic, which opened in October.

### Hennepin opens new neighborhood and community clinics

Hennepin County Medical Center opened three new sites of service this fall as part of an expansion of its neighborhood and community clinics.

**Whittier Clinic** is a 59,000 square foot primary care clinic and multi-specialty center located at 2810 Nicollet Avenue in South Minneapolis. This new, state-of-the-art, multi-specialty center opened in October and replaced the Family Medical Center on Lake Street. Primary care provided at the clinic includes family medicine, pediatrics, obstetrics and gynecology. Available specialty care includes orthopedics, cardiology, sports medicine, integrative health, physical therapy, and surgery.

The new **Brooklyn Park Clinic** in the Village Creek neighborhood at the corner of Zane Avenue and Brooklyn Boulevard provides family-centered primary care to children and adults, including family medicine, pediatrics, obstetrics and gynecology, and certified nurse-midwives.

The Brooklyn Park Clinic will complement the services provided to northwest metro residents at the Brooklyn Center Clinic, which offers primary care for adults and specialty care including rheumatology, cardiology, surgery and geriatrics.

In the southwest metro, Hennepin County Medical Center now operates two retail clinics in renovated Walmart stores in Bloomington and Eden Prairie. These clinics complement the care provided through nearby full-service primary care clinics—Richfield Clinic in the Hub Shopping Center in Richfield and the East Lake Clinic, located at 2700 East Lake Street in Minneapolis.

### Study explores link between sepsis and functional disability

Moderate to severe cognitive impairment is more than three times as likely after an episode of sepsis and more than four times as likely when patients are older adults, according to a longitudinal, population-based study published in the Journal of the American Medical Association (JAMA) on October 27, 2010. Overall, nearly 60% of the patients in the study who were hospitalized with severe sepsis experienced worse cognitive or physical functioning by their first post-sepsis assessment.

Researchers sought to rule out pre-existing health conditions as the cause of cognitive/physical decline by analyzing results from the Health and Retirement Study, which included prospective cognitive and functional evaluations for more than 9,000 patients along with linked Medicare claims data. Among this group, 516 survived severe sepsis and 4,517 survived a non-sepsis-related hospitalization. The mean age of hospitalization was 76.9 years.

An accompanying JAMA editorial recommended that physicians use these results to talk to patients and families about probable outcomes and to plan care that addresses longer-term survival and functional outcomes.

## Did you train at Hennepin?

**We're looking for you.**

You are an important member of an exclusive group of physicians who share Hennepin County Medical Center's expertise and knowledge with the people of the Upper Midwest. Hennepin is committed to continue a learning and sharing relationship with our alumni and would like to stay in touch.

Please submit your contact information at

**[HCMC.org/alumni.html](http://HCMC.org/alumni.html)**

or to R. Hoppentrath, 701 Park Ave., Mpls, MN 55415

## For more information

To download additional resources for critical care physicians, please visit the *Approaches in Critical Care* Web site at [www.hcmc.org/approaches](http://www.hcmc.org/approaches).

There, you'll find:

- ▶ An electronic version of *Approaches in Critical Care* that you can email to colleagues
- ▶ A link to Surviving Sepsis campaign materials.
- ▶ Protocols, educational materials, and many other resources from past issues.



Hennepin County **Medical Center**

Every Life Matters

**CHANGE SERVICE REQUESTED**

PRESORTED  
STANDARD  
U.S. POSTAGE  
**PAID**  
MINNEAPOLIS, MN  
PERMIT NO. 3273

Hennepin County Medical Center is a Level I Trauma Center and public teaching hospital repeatedly recognized as one of America's best hospitals by *U.S. News & World Report*. As one of the largest and oldest hospitals in Minnesota, with 469 staffed beds and more than 102,000 emergency services visits per year at our downtown Minneapolis campus, we are committed to provide the best possible care to every patient we serve today; to search for new ways to improve the care we will provide tomorrow; to educate health care providers for the future; and to ensure access to health care for all.

