Antibiotics in Patients With Sepsis

CRRT and Anticoagulation

High-Dose Insulin/Glucose Therapy

Thoracic Insufficiency Syndrome

Status Epilepticus in Adults

AUC

MIC

Vd

t₁/₂
FOCUS ON WHAT MATTERS MOST: PATIENT CARE.

When you become a nurse and officer on the U.S. Army or Army Reserve health care team, you’ll be able to continue to work in your community and serve when needed. You’ll be surrounded by health care professionals who share your passion for providing quality patient care.

To learn more about the U.S. Army health care team, call 800-431-6782 or visit healthcare.goarmy.com/at72
Take your knowledge to the next level

Your patients depend on it.

AACN offers the latest certification review courses to assist you with the new CCRN, CCRN-E and CCRN-K exams that went into effect in late 2015. Presented by nationally known subject matter experts, our online review courses will help you prepare for the exams.

Special Offer: AACN is offering individual exam candidates 50 percent off the cost of the following new online review courses when purchased at the time of your exam application:

- Adult CCRN/CCRN-E/CCRN-K Certification Review 2015
- Pediatric CCRN/CCRN-K Certification Review 2015
- Neonatal CCRN/CCRN-K Certification Review 2015

www.aacn.org/ccrnreviewcourse · 800-899-AACN

Brought to you by AACN  |  Leaders in acute and critical care nursing  |  Trusted expertise
Volume management decisions made easy with the 100% noninvasive STARLING™ SV

Dynamic Assessments of fluid responsiveness in Severe Sepsis, Septic Shock, and all Critically-Ill Patients.

Cheetah Medical hemodynamic monitors provide a noninvasive, flow-based, and central measurement of Stroke Volume.

The Cheetah Starling SV provides real-time quantification of volume responsiveness.¹ Using CVP to determine fluid responsiveness has limitations.²

- Nurse-driven dynamic assessments, including Passive Leg Raise (PLR) and the Fluid Bolus (FB) Challenge, directly challenge the heart with volume to measure the response.
- The PLR and FB protocols are included as an option in the NQF0500/CMS SEP1 Severe Sepsis/Septic Shock Management Bundle.
- Δ Stroke Volume Index (ΔSVI) ≥ 10% is highly predictive of fluid responsiveness.
- Sensors can be used for up to 48 hours for rapid assessment and trending of volume status.

Every patient has unique and constantly changing hemodynamic needs. Understanding your patient’s volume status is critical as serious complications are associated with under- and over-resuscitation, including increased morbidity, organ failure, and death.

Fluid vs Complications

Optimal volume

Hypovolemia³ Fluid Overload⁴,⁵,⁶

Challenging conditions that may benefit from dynamic assessments of fluid responsiveness

- Severe Sepsis and Septic Shock
- Adult Respiratory Distress Syndrome
- Congestive Heart Failure
- Acute Kidney Injury
- Subarachnoid Hemorrhage

References

The Starling SV is a trademark of Cheetah Medical, Inc.
Application of Antibiotic Pharmacodynamics and Dosing Principles in Patients With Sepsis

Molly E. Droege, Suzanne L. Van Fleet, and Eric W. Mueller

Page 22

OnlineNOW

Music as Medicine: The Therapeutic Potential of Music for Acute Stroke Patients

Charlene Supnet, April Crow, Sonja Stutzman, and DaiWai Olson

Page e1 (Page 20)

CCN FAST FACTS

Continuous Renal Replacement Therapy and Anticoagulation: What Are the Options?

Molly E. Droege, Suzanne L. Van Fleet, and Eric W. Mueller

Page 42
WE’RE READY WHEN YOU ARE

THE ACE ADVANTAGE

• FULLY COMPATIBLE WITH THE NEW ENFit ISO 80369-3 STANDARD

• SUPPORTS CLOSED ENTERAL FEEDING SYSTEMS

• ON-OFF HANDLE CONTROLS FLUID FLOW—TURN “ON” FOR FEEDING OR “OFF” FOR TUBE IRRIGATION, MEDICINE ADMINISTRATION, GASTRIC RESIDUALS, OR PATIENT AMBULATION

• AUTO-SEAL SYRINGE MED PORT ACCEPTS MOST 60ML ENFit SYRINGES FOR MEDICATION DELIVERY, IRRIGATION AND GASTRIC RESIDUALS

• PROTECTS CLINICIAN FROM SPLASH-BACK

• PROTECTS PATIENT FROM EXTERNAL CONTAMINATION

• NO SOILED LINENS OR FORMULA LOSS DUE TO OPEN CONNECTIONS

FOR MORE INFORMATION PLEASE VISIT
WWW.DALEMED.COM

DALE MEDICAL PRODUCTS, INC.
800-343-3980
WWW.DALEMED.COM

SEE US AT BOOTH #2247 AT NTI
Continuous Renal Replacement Therapy and Anticoagulation: What Are the Options?
Susan Dirkes and Rob Wonnacott
Page 34

COLUMNS

TOXICOLOGY
β-Blocker and Calcium Channel Blocker Poisoning: High-Dose Insulin/Glucose Therapy
Dana Bartlett
Page 45

PEDIATRIC CARE
Use of a Vertical Expandable Prosthetic Titanium Rib in Children With Thoracic Insufficiency Syndrome and Scoliosis
Stephanie L. Watts
Page 52

NEUROLOGY/NEUROSURGERY
Status Epilepticus in Adults: A Review of Diagnosis and Treatment
Thomas Lawson and Susan Yeager
Page 62

DEPARTMENTS

GUEST EDITORIAL
Maintaining Your Momentum: Moving Evidence Into Practice
Mary Beth Flynn Makic and Carol Rauen
Page 13

CERTIFICATION TEST PREP
We Do It for Our Patients
Carol Rauen, Karen Jeffries, and Mary Beth Flynn Makic
Page 74

ASK THE EXPERTS
Best Method for Securing an Endotracheal Tube
Sean G. Smith and Tom Pietrantonio
Page 78

COCHRANE REVIEW SUMMARY
Pulmonary Artery Catheters for Adult Patients in Intensive Care
Adam S. Cooper
Page 80

IN OUR UNIT
The Impact of Family Engagement on Anxiety Levels in a Cardiothoracic Intensive Care Unit
Mariana Skoog, Kerry A. Milner, JoAnne Gatti-Pettito, and Kiran Dintyala
Page 84

ALSO IN THIS ISSUE

Contributors
Page 8

 Corrections
Page 18

OnlineNOW Abstract
Page 20

Book Reviews
Page 91

Acknowledgment of Reviewers
Page 93

Education Directory
Page 94

I Am a Critical Care Nurse
Page 96

AACN Practice Alert: Initial and Ongoing Verification of Feeding Tube Placement in Adults
Page e8

2016 Evidence-Based Solutions Abstracts
Page e14
61% of hospital-acquired pneumonia happens to non-ventilated patients\(^1\)

Address bacterial colonization of the oral cavity, dental plaque and aspiration\(^2\)

Help protect your non-ventilated patients from pneumonia risk with Q•Care\textsuperscript{®} Continue Care\textsuperscript{™} Oral Care Cleansing and Suctioning Systems. Our comprehensive approach incorporates innovative tools and solutions, effective compliance programs and proven clinical outcomes.

Continue your prevention success outside the ICU. Non-ventilator hospital-acquired pneumonia (NV-HAP) has the potential to affect more patients, be more costly, and be as lethal as ventilator-associated pneumonia (VAP).\(^3\)


**Proven results:**
A published study demonstrated a 40% reduction of NV-HAP cases during a 12-month period, resulting in a return on investment of an estimated $1.6 million using a site specific oral care initiative.\(^4\)

Schedule a FREE pneumonia prevention Lunch & Learn at your facility!

Call **800.323.2220** or visit sageproducts.com/NVHAP
Contributors

Author β-Blocker and Calcium Channel Blocker Poisoning: High-Dose Insulin/Glucose Therapy, Dana Bartlett is an information specialist at the Connecticut Poison Control Center, Farmington, Connecticut.

Coauthor of Music as Medicine: The Therapeutic Potential of Music for Acute Stroke Patients, April Crow is a nurse in the inpatient rehabilitation unit of Zale Lipsy University Hospital, Dallas, Texas.

Susan Dirkes, coauthor of Continuous Renal Replacement Therapy and Anticoagulation, is a staff nurse in the surgical intensive care unit and the progressive care unit at the University of Michigan Health System, Ann Arbor, Michigan.

Coauthor of Application of Antibiotic Pharmacodynamics and Dosing Principles in Patients With Sepsis, Molly E. Droege is a clinical pharmacy specialist, trauma, surgery, orthopedics, UC Health–University of Cincinnati Medical Center, and an assistant professor of clinical pharmacy and an adjunct instructor of advanced clinical nursing, University of Cincinnati, Ohio.

Thomas Lawson, coauthor of Status Epilepticus in Adults, is an acute care nurse practitioner in the neuroscience critical care unit at Ohio State University Wexner Medical Center, Columbus, Ohio.

Guest Editorial contributor Mary Beth Flynn Makic is an associate professor, College of Nursing, University of Colorado, Aurora, Colorado. Dr Makic is a recognized expert in critical care and evidence-based practice.

Eric W. Mueller, coauthor of Application of Antibiotic Pharmacodynamics and Dosing Principles in Patients With Sepsis, is an assistant director, clinical services and research, and a clinical pharmacy specialist, critical care, Department of Pharmacy Services, UC Health–University of Cincinnati Medical Center.

Coauthor of Music as Medicine: The Therapeutic Potential of Music for Acute Stroke Patients, DaiWai Olson is an associate professor and director of the Neuroscience Nursing Research Center at University of Texas Southwestern.

Guest Editorial contributor Carol Rauen is an independent clinical nurse specialist and education consultant.

Sonja Stutzman, coauthor of Music as Medicine: The Therapeutic Potential of Music for Acute Stroke Patients, is the clinical research manager for the Neuroscience Nursing Research Center, University of Texas Southwestern Medical Center.

Coauthor of Music as Medicine: The Therapeutic Potential of Music for Acute Stroke Patients, Charlene Supnet is an experienced basic/clinical neuroscience researcher and writer for the Department of Neurology and Neurotherapeutics and the Neuroscience Nursing Research Center, University of Texas Southwestern Medical Center, Dallas, Texas.

Suzanne L. Van Fleet, coauthor of Application of Antibiotic Pharmacodynamics and Dosing Principles in Patients With Sepsis, is a clinical pharmacy specialist, critical care, UC Health–West Chester Hospital, West Chester, Ohio, and an assistant professor of clinical pharmacy and an adjunct instructor of advanced clinical nursing, University of Cincinnati.

Author of Use of a Vertical Expandable Prosthetic Titanium Rib in Children With Thoracic Insufficiency Syndrome and Scoliosis, Stephanie L. Watts is a nurse practitioner in the pediatric intensive care unit/progressive care unit at Children’s Hospital of Philadelphia, Pennsylvania.

Coauthor of Continuous Renal Replacement Therapy and Anticoagulation, Rob Wonnacott is the clinical educator in the surgical intensive care unit at the University of Michigan Health System.

Susan Yeager, coauthor of Status Epilepticus in Adults, is the lead acute care nurse practitioner in the neuroscience critical care unit at Ohio State University Wexner Medical Center and a clinical instructor at The Ohio State University College of Nursing, Columbus, Ohio. CCN
OFIRMEV® (acetaminophen) injection is proven to reduce acute pain, limit opioid exposure, and improve patient satisfaction¹-⁶

The clinical benefit of reduced opioid consumption with OFIRMEV has not been evaluated or demonstrated.

INDICATIONS AND USAGE
OFIRMEV® (acetaminophen) injection is indicated for the management of mild to moderate pain, management of moderate to severe pain with adjunctive opioid analgesics, and reduction of fever.

IMPORTANT RISK INFORMATION

WARNING: RISK OF MEDICATION ERRORS AND HEPATOTOXICITY
Take care when prescribing, preparing, and administering OFIRMEV Injection to avoid dosing errors which could result in accidental overdose and death. In particular, be careful to ensure that:

- the dose in milligrams (mg) and milliliters (mL) is not confused;
- the dosing is based on weight for patients under 50 kg;
- infusion pumps are properly programmed; and
- the total daily dose of acetaminophen from all sources does not exceed maximum daily limits.

OFIRMEV contains acetaminophen. Acetaminophen has been associated with cases of acute liver failure, at times resulting in liver transplant and death. Most of the cases of liver injury are associated with the use of acetaminophen at doses that exceed the recommended maximum daily limits, and often involve more than one acetaminophen-containing product.

Please see additional Important Risk Information and Brief Summary of Full Prescribing Information, including complete boxed warning, on the following pages.
In a major abdominal and pelvic surgery study, patients receiving IV acetaminophen 1 g with IV opioids (n=20) reported significantly lower pain scores vs placebo with IV opioids (n=20), $P<0.01$.

The clinical benefit of reduced opioid consumption with OFIRMEV has not been evaluated or demonstrated.

Percentage of patients, after total hip replacement, rating satisfaction as "good" or "excellent" at bedtime improved $^6,a$ following hip arthroplasty.

**Greater Satisfaction**

Following hip arthroplasty, 85.7% of patients receiving OFIRMEV with PCA morphine (n=30) vs 39.3% of patients receiving placebo with PCA morphine (n=31) reported "good" or "excellent" satisfaction with their pain management at bedtime, $P=0.0018$.

*$^a$ This study was terminated early due to detection of particulates in some placebo vials.
INDICATIONS AND USAGE

OFIRMEV® (acetaminophen) injection is indicated for the management of mild to moderate pain, management of moderate to severe pain with adjunctive opioid analgesics, and reduction of fever.

IMPORTANT RISK INFORMATION

WARNING: RISK OF MEDICATION ERRORS AND HEPATOTOXICITY

Take care when prescribing, preparing, and administering OFIRMEV Injection to avoid dosing errors which could result in accidental overdose and death. In particular, be careful to ensure that:

- the dose in milligrams (mg) and milliliters (mL) is not confused;
- the dosing is based on weight for patients under 50 kg;
- infusion pumps are properly programmed; and
- the total daily dose of acetaminophen from all sources does not exceed maximum daily limits.

OFIRMEV contains acetaminophen. Acetaminophen has been associated with cases of acute liver failure, at times resulting in liver transplant and death. Most of the cases of liver injury are associated with the use of acetaminophen at doses that exceeded the recommended maximum daily limits, and often involve more than one acetaminophen-containing product.

CONTRAINDICATIONS

- Acetaminophen is contraindicated in patients with:
  - known hypersensitivity to acetaminophen or to any of the excipients in the intravenous (IV) formulation.
  - severe hepatic impairment or severe active liver disease.

WARNINGS AND PRECAUTIONS

- Administration of acetaminophen in doses higher than recommended may result in hepatic injury, including the risk of liver failure and death. Do not exceed the maximum recommended daily dose of acetaminophen. The maximum recommended daily dose of acetaminophen includes all routes of acetaminophen administration and all acetaminophen-containing products administered, including combination products. Dosing errors could result in accidental overdose and death.

- Use caution when administering acetaminophen in patients with the following conditions: hepatic impairment or active hepatic disease, alcoholism, chronic malnutrition, severe hypovolemia (e.g., due to dehydration or blood loss), or severe renal impairment (creatinine clearance ≤ 30 mL/min).

- Rarely, acetaminophen may cause serious skin reactions such as acute generalized exanthematous pustulosis (AGEP), Stevens-Johnson Syndrome (SJS), and toxic epidermal necrolysis (TEN), which can be fatal.

- Hypersensitivity and anaphylaxis associated with the use of acetaminophen have been reported. Clinical signs included swelling of the face, mouth, and throat, respiratory distress, urticaria, rash, and pruritus.

- The antipyretic effects of OFIRMEV may mask fever.

ADVERSE REACTIONS

- Serious adverse reactions may include hepatic injury, serious skin reactions, hypersensitivity, and anaphylaxis.

- Common adverse reactions in adults include nausea, vomiting, headache, and insomnia. Common adverse reactions in pediatric patients include nausea, vomiting, constipation, pruritus, agitation, and atelectasis.

USE IN SPECIFIC POPULATIONS

- Pregnancy Category C. OFIRMEV should be given to a pregnant woman only if clearly needed.

- Breastfeeding: While studies with OFIRMEV have not been conducted, acetaminophen is secreted in human milk in small quantities after oral administration.

- Pediatric Use: The effectiveness of OFIRMEV for the treatment of acute pain and fever has not been studied in pediatric patients < 2 years of age.

To report SUSPECTED ADVERSE REACTIONS, contact Mallinckrodt Hospital Products, Inc. at 1-800-778-7898 or the FDA at 1-800-FDA-1088 or www.fda.gov/medwatch. Please see Brief Summary of Full Prescribing Information, including complete boxed warning, on the following page.

References:

INDICATIONS AND USAGE
OFIRMEV is a sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, preservative-free, sterile, clear, colorless, non-pyrogenic, res...
Guest Editorial

Maintaining Your Momentum: Moving Evidence Into Practice
Mary Beth Flynn Makic, RN, PhD, CNS, CCNS
Carol Rauen, RN-BC, MS, CCRN, PCCN, CEN

Ensuring that we provide safe, evidence-based, cost-effective care to all patients is an assumption of today’s health care system. All patients and health care providers should expect a health care system that is committed to preventing harm and improving patient care by having clinicians use evidence-based, safe practices.¹ The Institute of Medicine’s report To Err Is Human clearly articulated the need for health care professionals to embrace evidence-based practice (EBP) to improve outcomes of patient care.² Since that hallmark publication, efforts have been made by multiple organizations to encourage EBP. Several organizations are leading the way by providing EBP resources and toolkits to inform and improve practice: the Institute for Healthcare Improvement,³ The National Quality Forum,¹ the Agency for Healthcare Research and Quality,⁴ the American Association of Critical-Care Nurses (AACN),⁵ and the Society of Critical Care Medicine,⁶ to name just a few. Yet practice outcomes and reviews suggest that barriers persist, preventing daily application of current best evidence in the care of patients.⁷⁻⁹

Barriers range from qualities of hospital systems and organizations, to leadership support, to individual health care professionals not fully embracing EBP interventions as a practice standard.⁷⁻¹¹ In 2010, the Institute of Medicine provided a vision that all clinical decisions would be evidence based by 2020.¹² To meet this goal, we as critical care nurses have an opportunity to lead practice change by fully embracing EBP in our daily practice. Practice interventions wedded in tradition need to be retired, and evidence-based nursing interventions should be consistently implemented in the care of the critically ill patients and families we serve.

Practice knowledge is not stagnant. Evidence supporting practice interventions is dynamic and continually evolving. To ensure that practice is based on the current best evidence, critical care nurses need to have a good understanding of what EBP is. Although multiple definitions of EBP can be found in the literature and it is beyond the scope of this article to provide an in-depth discussion of EBP, several key tenets are present in each definition. Essential elements of EBP include the integration of best research and other forms of evidence to guide practice, viewing clinical expertise as a component in care effectiveness, and considering patients’ preferences, values, and engagement in care decisions as essential to providing optimal evidence-based care to patients and their families.²⁻¹⁸ Embracing EBP as a practice standard requires critical care nurses to be active consumers of current evidence, critically applying evidence-based interventions in practice and retiring traditional ways of providing care.

Authors

Mary Beth Flynn Makic is an associate professor, College of Nursing, University of Colorado, Aurora, Colorado. Dr. Makic is a recognized expert in critical care and evidence-based practice.

Carol Rauen is an independent clinical nurse specialist and education consultant. Carol is a recognized expert in critical care nursing and certification instruction.

Corresponding author: Mary Beth Flynn Makic, RN, PhD, CNS, CCNS, College of Nursing, University of Colorado, 13120 E 19th Ave, Mail Stop C238-19, Aurora, CO 80045 (e-mail: marybeth.makic@ucdenver.edu).

To purchase electronic or print reprints, contact the American Association of Critical-Care Nurses, 101 Columbia, Aliso Viejo, CA 92656. Phone, (800) 899-1712 or (949) 362-2058 (ext 532); fax, (949) 362-2049; e-mail, reprints@aacn.org.

©2016 American Association of Critical-Care Nurses doi: http://dx.doi.org/10.4037/ccn2016568
One challenge lies in the fact that evidence is constantly evolving as we learn more about the effectiveness of various care interventions. As nurses, supporting a spirit of inquiry allows us to remain active learners, gaining new knowledge to guide practice. Research evidence provides the foundation of care interventions, and EBP could not exist without well-done research. Unfortunately, conclusive research evidence may not always be available to guide practice interventions, so nonresearch evidence should be critically examined to support and inform practice. EBP provides a synthesis of research and other forms of evidence to answer clinical questions and guide practice. Nurses need to evaluate the strength of evidence as far as the risk or benefit of the evidence that is guiding practice interventions.

Several tools exist to help clinicians critically evaluate and determine the strength of evidence (ie, the level of evidence). The levels of evidence defined by the AACN provide criteria for evaluating the strength of the evidence used to guide practice. (Readers are referred to a recently published article in Critical Care Nurse that discusses the application of AACN’s levels of evidence to guide clinical practice for more information.) Frequently, research evidence guiding interventions is limited; thus, critical evaluations of all forms of evidence (eg, nonexperimental evidence, national practice alerts, consensus statements, expert opinion, manufacturers’ guidelines) are necessary to guide practice. The ultimate goal is to provide nursing care that is based in a synthesis of current best evidence to optimize patients’ outcomes.

This article is based on a presentation at the AACN’s 2015 National Teaching Institute that took place in San Diego, California. That presentation was the eighth in a series of presentations and articles that challenge critical care nurses to examine the evidence used to guide nursing practice interventions. Almost a decade of practice traditions have been reviewed, and current evidence to support practice has been shared through this series of presentations and articles by experts to enhance practice knowledge and action. A total of 30 nursing practice intervention traditions and current evidence related to them have been presented.

The real challenge now lies with you, the critical care nurse. Are you practicing by current best evidence in your daily practice? And if you are not, why not? What are the barriers that need to be removed to allow you to implement EBP interventions effectively in your daily practice? Practice interventions and traditions that are not based on current evidence need to be retired, or in the spirit of this decade-long effort, practice traditions (otherwise also known as “sacred cow” practices) need to be put out to pasture.

**Traditions in Practice Versus Evidence-Based Interventions**

Practice traditions can be loosely defined as interventions or actions for which the body of evidence no longer supports the action(s), yet the intervention continues to be present in practice. In the past decade, clinical experts were informally contacted and asked to list practice traditions that persisted in critical care environments. A review of the literature was completed, and when evidence could be found to refute the practice tradition, the challenge to improve practice through retiring the practice tradition and apply current best evidence was moved forward in an effort to improve knowledge, nursing care, and patients’ outcomes. Although the evidence for some of the practice traditions presented was not always strong and research was limited, efforts were made to provide rigor in the review of evidence and practice recommendations to enhance critical care nursing practice.

A total of 30 practice traditions were critically reviewed. The interventions have been broadly grouped into 7 nursing practice intervention categories: respiratory, cardiovascular, psychosocial, hospital-acquired conditions, gastrointestinal, neurological, and pediatric. The Table provides a list of the practice traditions reviewed. We encouraged critical care nurses to evaluate their practice and ensure that their practice is based on best evidence and not on tradition.

Although all of the practice traditions reviewed in this series are important and all are within the power of nursing practice to affect patients’ outcomes, a few of the interventions are briefly revisited to encourage EBP adoption. First, we address preventing hospital-acquired conditions. A large volume of evidence addresses bundled and individual interventions to prevent harm to patients as a result of being hospitalized. The frequency
Table: Moving from traditions to evidence-based practice interventions

<table>
<thead>
<tr>
<th>Category</th>
<th>Tradition</th>
<th>Evidence-based intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Respiratory</strong></td>
<td>Saline instillation for secretion removal</td>
<td>Normal saline should not be instilled as a routine step with endotracheal suctioning; instilling saline will not enhance removal of secretions</td>
</tr>
<tr>
<td></td>
<td>Withholding oxygen administration with patients who have chronic obstructive pulmonary disease</td>
<td>Oxygen should not be withheld from patients with chronic obstructive pulmonary disease, who have increased oxygen needs</td>
</tr>
<tr>
<td></td>
<td>Excessive sedation; avoiding sedation and daily awakening practice</td>
<td>Interrupt sedation daily to assess patients’ neurological status and/or readiness for reduced ventilator support or extubation</td>
</tr>
<tr>
<td><strong>Cardiovascular</strong></td>
<td>Blood pressure measurement: arterial catheter and blood pressure cuff are interchangeable</td>
<td>An appropriately sized blood pressure cuff is required for accurate measurement; noninvasive methods and arterial catheter will yield different blood pressure measurements; the appropriate reference level for blood pressure measurement, noninvasive or invasive, is the heart</td>
</tr>
<tr>
<td></td>
<td>Chest tube stripping to maintain patency</td>
<td>Stripping chest tubes may cause patient harm; only milk chest tubes if required to advance fluid into drainage chamber</td>
</tr>
<tr>
<td></td>
<td>Use of Trendelenburg position to improve blood pressure</td>
<td>Avoid using Trendelenburg position; use passive leg raise to increase preload</td>
</tr>
<tr>
<td></td>
<td>Avoid use of small-gauge intravenous catheters for blood administration</td>
<td>A 25-gauge catheter may be used to transfuse blood products and will not cause hemolysis</td>
</tr>
<tr>
<td></td>
<td>Renal-dose dopamine to preserve renal function</td>
<td>Low-dose dopamine does not preserve renal function but may improve cardiac output</td>
</tr>
<tr>
<td></td>
<td>Accuracy of temperature measurement</td>
<td>Accuracy of temperature measurement is very dependent on correct use of the measurement device</td>
</tr>
<tr>
<td></td>
<td>Electrocardiography lead placement</td>
<td>Accurate electrocardiographic assessment and myocardial assessment require proper placement of leads</td>
</tr>
<tr>
<td></td>
<td>Fluid resuscitation practices</td>
<td>Optimal fluid resuscitation may be achieved with crystalloid solutions; human blood products remain the best fluid for patients with blood loss or symptomatic anemia</td>
</tr>
<tr>
<td></td>
<td>Daily weighing of patients for weight-based medication practices</td>
<td>For most medications, using the patient’s admission weight for weight-based medication calculations is encouraged; consult with pharmacist, especially for older patients, patients with higher body mass index, and certain drug categories (eg, antibiotics, chemotherapeutic agents, anticoagulant agents)</td>
</tr>
<tr>
<td></td>
<td>Patient must be supine and flat for hemodynamic monitoring</td>
<td>Supine patients, with legs parallel to the floor, may have head of bed elevated up to 60° for hemodynamic measurements; the key to accurate measurements is the use of a position-specific reference level, by convention, the phlebotastic axis</td>
</tr>
<tr>
<td><strong>Psychosocial</strong></td>
<td>Restricting intensive care unit visitation</td>
<td>Open visitation 24/7 enhances patient/family engagement and does not have adverse physiologic impact on patients’ outcomes</td>
</tr>
<tr>
<td></td>
<td>Limiting cell phone use in the intensive care unit</td>
<td>Policies are needed to address cell phone use to protect patients’ privacy yet allow patients to connect with family</td>
</tr>
<tr>
<td><strong>Hospital-acquired conditions</strong></td>
<td>Early removal of urinary catheters to reduce catheter-associated urinary tract infections is not possible in the intensive care unit</td>
<td>Nurse-driven interventions to reduce catheter-associated infections focus on addressing the need for the catheter, sterile/aseptic catheter insertion, keeping drainage bag below the level of the bladder at all times, daily catheter care, and prompt removal of the catheter</td>
</tr>
<tr>
<td></td>
<td>Addressing infection control practice</td>
<td>Ensuring hand hygiene, proper infection control practices, and antibiotic stewardship are essential roles of nurses</td>
</tr>
<tr>
<td></td>
<td>Prevention of venothromboembolism</td>
<td>Patients must be consistently assessed for risk of venothromboembolism and nurses should ensure that proper mechanical and chemical prophylaxis is part of the daily plan of care review</td>
</tr>
<tr>
<td></td>
<td>Turning every 2 hours is not necessary</td>
<td>Though evidence is limited, frequent turning and mobilizing of patients, at least every 2 hours, is important to prevent complications of immobility</td>
</tr>
<tr>
<td></td>
<td>Is progressive mobility practice possible for intensive care patients?</td>
<td>Progressive mobilization of patients starting with bed mobility and progressing to out-of-bed movement is safe for intubated patients and can reduce patient complications of immobility</td>
</tr>
</tbody>
</table>

Continued
of hospital-acquired conditions remains unacceptably high, with an estimated 1 in 25 patients experiencing a hospital-acquired condition. Preventing hospital-acquired conditions from occurring requires a collaborative effort of all health care professionals, each of whom must be personally accountable.1,27

The key to preventing most hospital-acquired conditions is good infection control practices, starting with hand hygiene. The World Health Organization28 and the Centers for Disease Control and Prevention29 continue to provide evidence that hand hygiene is essential to prevent patient harm. It’s time to simply do it—every patient, every time. In the wake of the Ebola events, editors of the American Journal of Critical Care reminded nurses of the importance of consistent use of hand hygiene and pristine techniques as the foundation for preventing transmission of infections.30 Sadly, evidence continues to suggest that hospital-acquired conditions, especially those related to infection prevention efforts, are often tracked back to inconsistent implementation of proven measures for infection prevention.27,28,30 Critical care nurses can improve practice environments and patient care by modeling infection prevention strategies and helping colleagues adhere to infection prevention protocols.

Vital signs are critical assessment data points used in clinical decision making. Ensuring accurate measurement of vital signs often falls within the purview of the critical care nurse. More research is not needed to guide practice concerning blood pressure cuff size, difference in arterial catheter and noninvasive blood pressure assessment, and positioning of patients for accurate hemodynamic assessment.19,22 Similarly, correct placement of electrocardiography leads and proper assessment of gastric tube placement before gastric feeding are well supported by the evidence.19,21 The goal is for nurses to use the evidence consistently so that accurate assessments and data are provided for health care decisions in the care of our patients.

<table>
<thead>
<tr>
<th>Category</th>
<th>Tradition</th>
<th>Evidence-based intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrointestinal</td>
<td>Assessment of gastric residual volume and aspiration risk every 4 hours</td>
<td>Routine gastric assessment may result in underfeeding patients; assessment of residual volume should be based on patient’s condition rather than a routine</td>
</tr>
<tr>
<td></td>
<td>Use of rectal tubes to manage fecal incontinence</td>
<td>Rectal tubes should not be the first intervention in the management of fecal incontinence; assess and address the cause of the fecal incontinence and try less invasive measures when possible</td>
</tr>
<tr>
<td></td>
<td>Using auscultation as the primary way of verifying gastric tube position in adults</td>
<td>Auscultation should not be used to verify placement of gastric feeding tubes in adult patients</td>
</tr>
<tr>
<td>Neurological</td>
<td>Glasgow Coma Scale for primary neurological assessment</td>
<td>Best clinical practice for neurological assessment includes optimal and consistent use of the Glasgow Coma Scale plus inclusion of other neurological data such as assessment of brain stem reflexes; eye examination, including pupil reactivity and extraocular movement; vital signs; and respiratory rate, depth, and pattern</td>
</tr>
<tr>
<td></td>
<td>Intracranial hypertension management strategies</td>
<td>Modulation of brain volume with osmotic therapy, cerebrospinal fluid with drainage, metabolic suppression, and controlled ventilation</td>
</tr>
<tr>
<td></td>
<td>Sleep hygiene practices</td>
<td>Efforts need to be made to provide patients with uninterrupted sleep while in intensive care unit, reduce environmental noise, and limit unnecessary interventions during the night</td>
</tr>
<tr>
<td></td>
<td>Use of vital signs to assess pain in intubated patients</td>
<td>Physiological parameters are not accurate for pain assessment; behavioral pain assessment tools should be used to assess pain in nonverbal/intubated patients</td>
</tr>
<tr>
<td>Pediatric</td>
<td>Using auscultation as the primary way of verifying gastric tube position in children</td>
<td>Auscultation should not be used to verify placement of gastric feeding tubes in children</td>
</tr>
<tr>
<td></td>
<td>Chest physiotherapy indications</td>
<td>Chest physiotherapy is effective in specific pediatric conditions but is not effective as a routine intervention in the treatment of pneumonia, bronchiolitis, or asthma</td>
</tr>
<tr>
<td></td>
<td>Accuracy of blood pressure measurement in children</td>
<td>Blood pressure cuff should be appropriate size for patient’s arm; initial measurement should be completed by using the auscultatory method and compared with measurements made with noninvasive methods with the patient’s arm level with the patient’s heart</td>
</tr>
</tbody>
</table>

8 Based on information from Rauen et al19,20 and Makic et al.11-24
Management of patients with chronic obstructive pulmonary disease and patients requiring mechanical ventilation, for a variety of reasons, is common within intensive care units. Ensuring that EBP interventions are used should be a cornerstone in the care of these patients. Evidence-based guidelines exist for the use of sedation and analgesia to prevent patient harm, namely, delirium. Research has shown that instilling saline to loosen secretions during routine suctioning harms patients and that withholding oxygen from a patient with chronic obstructive pulmonary disease is not supported and may actually harm the patient.

Research has shown that numerous factors influence EBP, ranging from the individual’s formal or informal leadership within the unit, unit/organizational culture supporting a culture of inquiry and questioning practice, and the availability of resources. Effectively removing practice traditions from daily practice requires active dissemination and diffusion of new knowledge to guide practice interventions. In this decade-long series of presentations and articles, we have tried to encourage a change in practice from tradition to evidence-based nursing interventions. We believe that critical care nurses are in an optimal position to lead the diffusion of knowledge through implementation and consistent application of EBP interventions. Fully embracing EBP in critical care nursing practice will benefit the highly vulnerable patients and families whom we serve.

Evidence-Based Practice and High-Reliability Organizations: Safe Patient Care

One prominent premise of health care reform through EBP as a foundation for care is for all health care professionals to know and apply the current best evidence in daily care of patients to improve outcomes and reduce costs. High-reliability organizations achieve high-quality care through creation of a culture in which persistent mindfulness of patient safety drives care interventions and processes. Several essential elements of EBP align with principles of high-reliability organizations: (1) EBP provides a current body of knowledge that can be applied in the delivery of care to achieve consistent outcomes for patients; (2) nurses should critically evaluate and adapt evidence to meet the unique needs of the critically ill patient, individualizing care and allowing rapid adaptation by the nurse to prevent patient harm; and (3) effective translation of best evidence in practice can be measured through patients’ outcomes (eg, absence of errors). As hospitals continue to face demands to improve the quality and cost-effectiveness of care, critical care nurses can function as leaders modeling EBP that enhances patients’ outcomes.

Transforming care at the bedside requires nurses to develop patient-centered goals and strive to meet those goals through individualized evidence-based interventions. Success also requires nursing leaders who encourage a culture of inquiry and foster continual advancement of practice as the evidence evolves. Teamwork and collaborative practice must be a unit norm. When asking questions is encouraged, traditions in practice can be challenged, providing an opportunity to advance learning and practice through a culture that embraces EBP. Ensuring that critical care nursing practice is based on the current best evidence supports a culture of safety, helping achieve the goal of excellent care for every patient, every time.

Summary

Patients deserve and expect that the care they receive is current and evidence based. Critical care nurses are in an optimal position to lead practice, moving traditions “out to pasture” and embracing EBP interventions. Through the multiple presentations and publications on these sacred cow practice traditions, clinical experts have challenged each of us to critically examine our practice and ensure that we are current, that our care is evidence based. This series is being retired because few new practice traditions or topics are arising. Rather, the lack of adoption of evidence supporting the 30 nursing practice interventions appears to be the persistent problem. It’s time to change the culture in our critical care units to a culture that embraces the translation of evidence into daily practice. Thomas Paine said in 1776, “a long habit of not thinking a thing wrong gives it a superficial appearance of being right. . . .” As critical care nurses, we need to challenge practice traditions that are not supported by the current best evidence so that the superficial appearance of the habit is debunked and evidence-based interventions become the practice norm. CCN

Acknowledgments

We acknowledge the following individuals who have been instrumental in developing and supporting the “Evidence-Based Practice Sacred Cows” series over the years. Without these individuals’ commitment to sharing their expert knowledge through presentations and publications, and the editorial support of Critical
Care Nurse, effectively disseminating EBP knowledge would not have been possible. We sincerely thank you for your contribution to this collective series. But more importantly, we thank you for your engagement in spreading the evidence to support best practice and challenging critical care nurses to apply EBP in daily practice to benefit the patients and families whom we serve. A heartfelt thank you to Linda Bell, RN, MS, Nancy Munro, RN, MS, CCNS, ACNP, Marianne Chulay, RN, PhD, Rich Arbour, RN, MSN, CCNS, CNRN, Elizabeth Bridges, RN, PhD, CCNS, Sarah Martin, RN, MS, CPNP-PC/AC, Suzanne Burns, MN, RN, ACNP, Kathryn VonRueden, RN, MS, ACNS-BC, Ann Villee Potter, RN, MS, Robin Watson, RN, MS, CNS, Pam Shumate, RN, DPNC, CCNS, Kimmuth Jones, RN, DPNC, CCNS, Kathleen Vollman, RN, MSN, CCNS, Anna Fisk, RN, BSN, Jessica Chadwick, RN, MSN, CCNS, Dinah Philbrick, RN, BSN, and JoAnn Grif Aspach, RN, EDD.

Financial Disclosures

None reported.

References


Corrections

In the December Ask the Experts article by Alexander and Zomp, “Best Practices: Full-Dose Delivery of Intravenous Medications via Infusion Pumps” (Crit Care Nurse. 2015;35(6):68-70), the authors would like to clarify that the Alaris pump referred to on page 70 is an older version and that the max rate is now the same for both primary and secondary settings on Alaris pumps (999 mL/h).

©2016 American Association of Critical-Care Nurses
doi: http://dx.doi.org/10.4037/ccn2016628
ReliaFit® is a trademark of Ferndale IP, Inc. ©2013 Eloquest Healthcare®, Inc.

ReliaFit® is a trademark of Ferndale IP, Inc. ©2013 Eloquest Healthcare®, Inc.

CHANGING THE LANDSCAPE

ReliaFit reduces the risk of CAUTIs associated with indwelling catheters1

• Noninvasive design1

ReliaFit offers a superior alternative to condom catheters

• Unique design for superior leak prevention2
• Protects sensitive skin1
• One size fits all1

Contact your sales consultant, call 877.433.7826, or visit ReliaFitDevice.com to learn more about ReliaFit


Visit Booth #3717 at AACN’s NTI 2016 to Learn More!
Music as Medicine: The Therapeutic Potential of Music for Acute Stroke Patients
Charlene Supnet, PhD, April Crow, RN, Sonja Stutzman, PhD, and DaiWai Olson, RN, PhD, CCRN

Nurses caring for patients with acute stroke are likely to administer both music and medication with therapeutic intent. The administration of medication is based on accumulated scientific evidence and tailored to the needs of each patient. However, the therapeutic use of music is generally based on good intentions and anecdotal evidence. This review summarizes and examines the current literature regarding the effectiveness of music in the treatment of critically ill patients and the use of music in neurologically injured patients. The rationale for hypothesis-driven research to explore therapeutic music intervention in acute stroke is compelling. (Critical Care Nurse. 2016;36[2]:e1-e7)

©2016 American Association of Critical-Care Nurses
http://dx.doi.org/10.4037/ccn2016413

Do you have a QR scanner app on your iPhone or Android?
Scan this QR code with your phone to access this article instantly.
There are just so many hours in a day.  
And last we checked, you can’t be in two places at once.

But what if you could?

With Medtronic Health Informatics & Monitoring, you can still be at your patients’ side.  
At home, in the hospital, wherever they are.  
Even if you’re not physically at their side, because we are reinventing what’s humanly possible.

Learn how  
HealthInformaticsMonitoring.com
Application of Antibiotic Pharmacodynamics and Dosing Principles in Patients With Sepsis

Molly E. Droege, PharmD, BCPS
Suzanne L. Van Fleet, PharmD
Eric W. Mueller, PharmD

Sepsis is associated with marked mortality, which may be reduced by prompt initiation of adequate, appropriate doses of antibiotic. Critically ill patients often have physiological changes that reduce blood and tissue concentrations of antibiotic and high rates of multidrug-resistant pathogens, which may affect patients’ outcomes. All critical care professionals, including critical care nurses, should understand antibiotic pharmacokinetics and pharmacodynamics to ensure sound antibiotic dosing and administration strategies for optimal microbial killing and patients’ outcomes. Effective pathogen eradication occurs when the dose of antibiotic reaches or maintains optimal concentrations relative to the minimum inhibitory concentration for the pathogen. Time-dependent antibiotics, such as β-lactams, can be given as extended or continuous infusions. Concentration-dependent antibiotics such as aminoglycosides are optimized by using high, once-daily dosing strategies with serum concentration monitoring. Vancomycin and fluoroquinolones are dependent on both time and concentration above the minimum inhibitory concentration. (Critical Care Nurse. 2016;36(2):22-32)

Sepsis is a major cause of morbidity and mortality in the United States, affecting more than 650 000 patients per year. The spectrum of sepsis to septic shock is associated with crude mortality rates of 20% to 70%. From the onset of hypotension, the probability of survival is reduced by 12% for each hour of delay before administration of adequate empiric antibiotics. Hence, current guidelines advocate early administration of adequate, broad-spectrum, local antibiogram–based antibiotic therapy in appropriate doses to penetrate the suspected site of infection (eg, lungs, cerebral spinal fluid, intra-abdominal fluid). Physiological changes associated with sepsis can complicate antibiotic dosing, effectiveness, and safety.

CE 1.0 hour, Pharma 1.0 hour

This article has been designated for CE contact hour(s). The evaluation tests your knowledge of the following objectives:
1. Discuss the effects of critical illness on pharmacokinetic and pharmacodynamic parameters of commonly used antibiotics
2. Describe the rationale for different dosing strategies for antibiotics used in adult critically ill patients
3. Understand clinical and practical implications of optimizing antibiotic dosage and administration strategies in critically ill patients

To complete evaluation for CE contact hour(s) for test #C1622, visit www.ccnonline.org and click the “CE Articles” button. No CE test fee for AACN members. This test expires on April 1, 2019.

The American Association of Critical-Care Nurses is an accredited provider of continuing nursing education by the American Nurses Credentialing Center’s Commission on Accreditation. AACN has been approved as a provider of continuing education in nursing by the State Boards of Registered Nursing of California (#08036) and Louisiana (#LSBN12).

©2016 American Association of Critical-Care Nurses doi: http://dx.doi.org/10.4037/ccn2016881
Critical care nurses play a crucial role in delivering safe and effective antibiotic treatment to critically ill patients. Therefore, nurses should understand antibiotic properties and dosage strategies that contribute to optimal outcomes in sepsis. In this article, we review pharmacokinetic and pharmacodynamic principles for frequently used antibiotics, with a focus on administration and monitoring for bedside critical care nurses.

Pharmacokinetics and Pharmacodynamics

Pharmacokinetics describes the effect of a patient’s physiology on drug concentrations (ie, the effect of the body on the drug).\(^6\) Drug absorption, distribution, metabolism, and excretion collectively determine drug concentrations within the blood and target tissues (ie, infection sites).\(^7\) Important pharmacokinetic terms include volume of distribution (\(V_d\)), not a physiological volume but rather a proportionality constant that relates serum drug concentration to body drug concentrations; clearance, the process of drug elimination (eg, hepatic metabolism, renal filtration) from the body over time (eg, volume of fluid cleared of drug over time); half-life (\(t_{1/2}\)), the time it takes the drug serum concentration to decrease by half; maximum concentration (\(C_{\text{max}}\)), peak or highest blood concentration after a dose; minimum concentration (\(C_{\text{min}}\)), trough or lowest blood concentration just before the next dose; and area under the curve (AUC), total drug presence in the blood and body during a 24-hour period.\(^6\) Changes in these pharmacokinetic parameters affect the ability of antibiotics to penetrate infection sites and achieve sufficient concentrations. Critically ill patients have many pharmacokinetic derangements because of vasodilatation, capillary leak, third spacing, edema, and changes in cardiac, renal, and hepatic body systems\(^4\) (Table 1).

**Table 1** Pharmacokinetic definitions and alterations in critical illness

<table>
<thead>
<tr>
<th>Pharmacokinetic parameter</th>
<th>Definition</th>
<th>Changes with critical illness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Volume of distribution ((V_d))</td>
<td>Drug-specific hypothetical proportionality constant that relates serum drug concentrations to the amount of drug in the body (eg, L/kg)</td>
<td>Increased after resuscitation (capillary leak, third spacing, fluid shifts)</td>
</tr>
<tr>
<td>Maximum concentration ((C_{\text{max}}))</td>
<td>Maximum or peak serum concentration after a single dose or at steady state (eg, g/L)</td>
<td>Decreased (increased (V_d), hypermetabolic, decreased gastrointestinal absorption)</td>
</tr>
<tr>
<td>Minimum concentration ((C_{\text{min}}))</td>
<td>Minimum or trough serum concentration after a single dose or at steady state (eg, g/L)</td>
<td>Increased (renal or hepatic dysfunction)</td>
</tr>
<tr>
<td>Clearance</td>
<td>Drug elimination from the body over time (eg, mL/min)</td>
<td>Increased (normal renal and hepatic function but cardiovascularly hyperdynamic, drug interactions)</td>
</tr>
<tr>
<td>Half-life ((t_{1/2}))</td>
<td>Time for serum concentration to decrease by 50%, assuming first-order elimination</td>
<td>Decreased (renal or hepatic dysfunction, drug interactions)</td>
</tr>
<tr>
<td>24-hour area under concentration-time curve (AUC)</td>
<td>Body’s total exposure to drug during a 24-hour period</td>
<td>Decreased (increased (V_d), hypermetabolic)</td>
</tr>
</tbody>
</table>

Authors

Molly E. Droge is a clinical pharmacy specialist, trauma, surgery, orthopedics, UC Health–University of Cincinnati Medical Center, and an assistant professor of clinical pharmacy and an instructor of advanced clinical nursing, University of Cincinnati, Cincinnati, Ohio.

Suzanne L. Van Fleet is a clinical pharmacy specialist, critical care, UC Health–West Chester Hospital, West Chester, Ohio, and an assistant professor of clinical pharmacy and an instructor of advanced clinical nursing, University of Cincinnati.

Eric W. Mueller is an assistant director, clinical services and research, and a clinical pharmacy specialist, critical care, Department of Pharmacy Services, UC Health–University of Cincinnati Medical Center. He is also an adjunct associate professor of pharmacy practice and an adjunct instructor of advanced clinical nursing, University of Cincinnati.

Corresponding author: Eric W. Mueller, PharmD, FCCM, FCP, 234 Goodman St, Mail Location 0791, Cincinnati, OH 45291 (e-mail: eric.mueller@uchealth.com).

To purchase electronic or print reprints, contact the American Association of Critical-Care Nurses, 101 Columbia, Aliso Viejo, CA 92656. Phone, (800) 899-1712 or (949) 362-2050 (ext 532); fax, (949) 362-2049; e-mail, reprints@aacn.org.
Pharmacodynamics describes the effect of a drug at its site of action (ie, what the drug does to the body). When applied to antibiotics, pharmacodynamics describes the method by which pathogens are targeted and eradicated. Thus, efficacy is largely determined by the antibiotic concentration at the infection site, microbial load, phase of microbial growth, and minimum inhibitory concentration (MIC) for the pathogen. The MIC is the lowest concentration necessary to inhibit the growth of 90% of the pathogen population in vitro. Conversely, the minimum bactericidal concentration is the lowest concentration necessary to irreversibly inhibit 99.9% of bacterial growth, leading to eradication of the inoculum.

Antibiotic susceptibility is determined by the relationship between MIC and achievable antibiotic concentration. If the MIC is lower than the antibiotic concentration achieved with a safe dose, the pathogen is considered susceptible; an MIC higher than the antibiotic concentration achieved indicates a resistant pathogen. Each antibiotic-pathogen combination has individualized thresholds for determining susceptibility or resistance. The range of concentrations between the MIC and the mutant prevention concentration, or the drug concentration corresponding to the MIC for the least susceptible mutant in a colony, has been called the mutant selection window. Some researchers have recommended that antibiotic concentrations exceed the mutant selection window to avoid development of resistance; however, the clinical relevance of this hypothesis is still being evaluated.

Effective eradication of a pathogen occurs when the dose of antibiotic reaches or maintains optimal concentrations relative to the pathogen MIC. Because proving the drug concentration at the infection site is not typically possible, MIC is used as a surrogate marker for assessing microbiological activity at the site.

Antibiotics are classified as having either time-dependent or concentration-dependent killing (Table 2, Figure 1). Time-dependent pharmacodynamics is described by the duration of time (T) that antibiotic concentrations exceed the pathogen MIC (ie, T > MIC). In contrast, the C max or peak concentration relative to pathogen MIC (ie, C max :MIC ratio) determines efficacy for concentration-dependent antibiotics. Antibiotics with mixed pharmacodynamics include those whose killing is related to both the concentration and time greater than the MIC (ie, AUC:MIC ratio).

Because the relationship between the pharmacokinetics and pharmacodynamics determines antibiotic efficacy, dosing and administration strategies should be...
individualized on the basis of the pathogen-drug-patient interplay to optimize outcomes. Critically ill patients are at risk for unpredictable antibiotic exposure because of previously mentioned physiological derangements.7 Critical care nurses often administer antibiotics with different frequencies, duration of infusions, and monitoring parameters to improve antibiotic efficacy.

**β-Lactams**

β-Lactams are an antimicrobial class that includes cephalosporins, penicillins, carbapenems, and monobactams that have time-dependent killing. β-Lactams bind to penicillin-binding proteins within the cell membrane, leading to weakening of the microbial cell wall, lysis, and pathogen death.11,12 Pharmacodynamic optimization for cephalosporins, penicillins, and carbapenems occurs when T > MIC for at least 60% to 70%, 50%, and 40% of the dosage interval, respectively.13 Some investigators6,12 have suggested that concentrations 4 to 5 times greater than the MIC are required for extended periods to achieve optimal activity.

Most β-lactams except ceftriaxone and nafcillin have low protein binding, primarily renal elimination, and short half-lives ranging from 30 minutes to 2 hours. Therefore, β-lactam regimens require relatively high doses administered multiple times per day with adjustments in dose in patients with renal dysfunction.6,12 Chemically, β-lactams are hydrophilic with a Vₐ of 0.5-0.6 L/kg and may not penetrate all infection sites adequately, such as the central nervous system and skeletal tissue.12

Because of the short half-lives, time-dependent pharmacodynamics, and increasing resistance to β-lactams, interest has grown in alternative dosing and administration methods such as prolonged infusion (ie, intermittent doses infused during a 3- to 6-hour period instead of traditional infusion durations of 30 minutes to 1 hour) and continuous infusion (ie, one-time loading dose followed by continuous infusion to achieve an antibiotic concentration greater than the MIC throughout the course of therapy). A secondary benefit of these administration strategies may be a lower total daily dose of antibiotic. For example, cefepime 2000 mg intravenously infused for 30 minutes every 8 hours compared with 2000 mg intravenously infused for 6 hours twice per day may provide similar or higher concentrations for longer times (Figure 2). At a specific MIC, this strategy would result in similar or longer periods of T > MIC, perhaps greater bacterial killing, and less overall drug use. Most contemporary evaluations of pharmacodynamics-related strategies for administration of β-lactams have focused on piperacillin/tazobactam, cefepime, and carbapenems.

Piperacillin/tazobactam is a penicillin/β-lactamase inhibitor combination commonly used for the treatment of many nosocomial infections, especially when infection with *Pseudomonas aeruginosa* is suspected. Although doses approved by the Food and Drug Administration range from 3.375 to 4.5 g intravenously every 6 hours with each dose infused over 30 minutes, pharmacokinetic models suggest suboptimal T > MICs with these dosages. Conversely, the same dose (eg, 3.375 g) given every 6 to 8 hours but infused over 4 hours significantly increases T > MIC.13 These pharmacodynamic differences may be important for organisms with higher MICs, which are common in critically ill patients at risk for highly resistant pathogens.14 Lodise et al13 retrospectively evaluated a cohort of patients with *P aeruginosa* infections who received piperacillin/tazobactam either as a 30-minute infusion of 3.375 g intravenously every 4 to 6 hours or as a 4-hour infusion of 3.375 g intravenously every 8 hours. Classification and regression tree analysis indicated a decrease in 14-day mortality (12.2% vs 31.6%; *P* = .04) and hospital length of stay (21 days vs 38 days; *P* = .02) in patients with an Acute Physiological and Chronic Health Evaluation-II score of 17 or greater.
who received the extended infusion.\textsuperscript{13} A meta-analysis\textsuperscript{15} of 2 nonblinded randomized control trials and 4 retrospective studies indicated that patients receiving extended or continuous infusion of piperacillin/tazobactam had lower mortality than did patients receiving standard infusion (relative risk, 0.55; 95\% CI, 0.34-0.89). Recent retrospective analyses\textsuperscript{16} have suggested no difference in mortality between extended and intermittent infusion. Although data related to patients’ outcomes are limited,\textsuperscript{17} continuous infusion of the same total daily dose of piperacillin/tazobactam appears to provide T > MIC similar to intermittent 4-hour infusions.\textsuperscript{18} Pharmacoeconomic benefits may also exist because of a decrease in the total daily dose of piperacillin/tazobactam, but further evaluation is warranted.\textsuperscript{19}

Cefepime is a fourth-generation cephalosporin commonly used for nosocomial infections for which prolonged and continuous infusions have been evaluated.\textsuperscript{20-26} The concern about achieving appropriate T > MIC with traditional dosage regimens (eg, 1-2 g every 8-12 hours infused over 30 minutes) for cefepime is similar to the concerns for piperacillin/tazobactam in critically ill patients at risk for resistant pathogens.\textsuperscript{21-25} Data suggest prolonged or continuous infusion strategies may provide higher T > MIC and increase the probability that the infection will be resolved.\textsuperscript{22,23,26} Changing the duration of infusion to 6 hours or administering a single 2000-mg loading dose followed by 24-hour continuous infusion of 4000 mg increases the probability of optimal T > MIC by 30\% across different levels of renal function and pathogen MICs.\textsuperscript{26} Most recent evidence\textsuperscript{12,23} suggests that cefepime 2000 mg intravenously every 8 hours with a 3-hour infusion may be necessary to achieve adequate T > MIC in critically ill patients with normal renal function. Although data are limited on prolonged or continuous infusion of cefepime on patients’ outcomes, observational evidence\textsuperscript{23} suggests that patients infected with \textit{P. aeruginosa} who do not achieve 60\% T > MIC are 8 times more likely to persistently have cultures positive for \textit{P. aeruginosa} than are patients who do achieve 60\% T > MIC. A retrospective quasi-experimental study\textsuperscript{27} of cefepime 2000 mg intravenously every 8 hours with a 30-minute infusion compared with a 4-hour infusion indicated decreased mortality (20\% vs 3\%; \textit{P} = .03) and decreased median hospital length of stay (14.5 days vs 11 days; \textit{P} = .36) and intensive care unit length of stay (18.5 days vs 8 days; \textit{P} = .36) with prolonged infusion.

Carbapenems such as imipenem/cilastatin, doripenem, and meropenem are similar to the other \textit{\beta}-lactams except that they require lower T > MIC for effective bacterial killing.\textsuperscript{6} However, they also tend to have faster clearance and shorter half-lives. Although direct comparison between agents is difficult, imipenem/cilastatin and meropenem 1000 mg intravenously every 8 hours infused over 30 minutes achieve adequate T > MIC in most patients and for organisms with low MICs.\textsuperscript{28,29} However, these regimens may have lower T > MIC in critically ill patients with high MIC organisms. Results of a study\textsuperscript{30} in critically ill patients with ventilator-associated pneumonia (VAP) indicate that meropenem 1000 mg or 2000 mg intravenously every 8 hours with a 3-hour infusion was necessary to achieve T > MIC, especially for infections caused by bacteria with higher MICs.

Using a Monte Carlo simulation and patients with nosocomial pneumonia, Frippiat et al\textsuperscript{31} found that optimum target attainment (40\%-100\% T > MIC) in epithelial lining fluid occurred with meropenem 2000 mg every 8 hours with a 3-hour infusion. Although not tested in critically ill patients, meropenem 3000 mg intravenously infused continuously over 24 hours in healthy individuals attained adequate T > MIC.\textsuperscript{32} Limited information exists on prolonged or continuous infusion of meropenem and patients’ outcomes. In a prospective, multicenter, noninferiority study, Chastre et al\textsuperscript{31} compared standard dosage imipenem/cilastatin with prolonged-infusion doripenem in critically ill patients with VAP. The results suggested that doripenem 500 mg intravenously every 8 hours with a 4-hour infusion was noninferior to imipenem/cilastatin with prolonged-infusion doripenem in critically ill patients with VAP. Despite the pharmacodynamic advantages of prolonged or continuous infusion of \textit{\beta}-lactams, criticisms of studies of this practice include use of mathematical modeling or small sample sizes, unclear applicability for different populations of patients, and limited information.
Practically, limited intravenous access, drug incompatibilities, safety concerns, and drug instability make widespread adoption of prolonged or continuous infusion strategies difficult.

**Fluoroquinolones**

Ciprofloxacin, levofloxacin, and moxifloxacin are the main fluoroquinolones used in practice today. Ciprofloxacin was first introduced in the late 1980s as a second-generation fluoroquinolone along with levofloxacin. Intravenous fluoroquinolones inhibit DNA gyrase and topoisomerase, leading to decreases in DNA transcription, DNA repair, and bacterial replication.

Fluoroquinolones have better tissue penetration (eg, respiratory tissue, cerebrospinal fluid, prostate gland, and skin) with higher $V_d$ and better oral bioavailability than $\beta$-lactams do. Ciprofloxacin and levofloxacin have activity against gram-positive and nosocomial gram-negative pathogens, whereas moxifloxacin has greater activity against anaerobes and gram-positive organisms.

Practically, limited intravenous access, drug incompatibilities, safety concerns, and drug instability make widespread adoption of prolonged or continuous infusion strategies difficult. The Surviving Sepsis Campaign guidelines recommend infusion of antibiotics within the first hour of recognition of septic shock, implying a necessity for standard infusion rates. Adverse events can occur if a large, continuous infusion dose is administered via accidental rapid infusion. Fortunately, use of smart pump technology can successfully aid beside nurses in appropriate administration. Continuous or extended infusions may also require additional intravenous access devices, depending on concomitant administration and compatibility of medications. Patients who require long-term therapy with $\beta$-lactams should be monitored when they go home, with consideration of a change to intermittent infusion, because extended infusion may be difficult at home without oversight from a health care professional. Doses should be changed to standard regimens to ensure that patients are not underdosed at the time of discharge. Overall, practical limitations and the lack of randomized trials may limit the widespread adoption of extended or continuous infusion of $\beta$-lactams.

Fluoroquinolones

Ciprofloxacin, levofloxacin, and moxifloxacin are the main fluoroquinolones used in practice today. Ciprofloxacin was first introduced in the late 1980s as a second-generation fluoroquinolone along with levofloxacin. Intravenous fluoroquinolones inhibit DNA gyrase and topoisomerase, leading to decreases in DNA transcription, DNA repair, and bacterial replication. Fluoroquinolones have better tissue penetration (eg, respiratory tissue, cerebrospinal fluid, prostate gland, and skin) with higher $V_d$ and better oral bioavailability than $\beta$-lactams do. Ciprofloxacin and levofloxacin have activity against gram-positive and nosocomial gram-negative pathogens, whereas moxifloxacin has greater activity against anaerobes and gram-positive organisms.

Practically, limited intravenous access, drug incompatibilities, safety concerns, and drug instability make widespread adoption of prolonged or continuous infusion strategies difficult.

From a pharmacodynamic standpoint, fluoroquinolones have primarily concentration-dependent pathogen killing with some time-dependent features. The primary parameter to describe fluoroquinolone pharmacodynamics is the AUC:MIC ratio; the higher the AUC relative to the bacterial MIC, the greater is bacterial killing. Specifically, an AUC:MIC ratio of 125 hours or greater during the dosing interval has been associated with a higher probability of clinical response and improved outcomes in patients with gram-negative infections. Although most available information is derived from studies on ciprofloxacin, this relationship appears to be similar among the 3 agents. In a study of critically ill patients with *P. aeruginosa* infections, ciprofloxacin 400 mg intravenously every 8 hours had an AUC:MIC ratio of 125 or more hours for 100% and 75% of pathogens with MICs of 0.125 mg/L or less and 0.25 mg/L, respectively, in bronchial secretions. As expected, traditional regimens more often achieve AUC:MIC ratios of 125 hours or greater in organisms with lower MICs (eg, *Klebsiella pneumoniae*, *Escherichia coli*, *Enterobacter aerogenes*, and *Proteus mirabilis*) compared with organisms with higher MICs (eg, *P. aeruginosa* and *Acinetobacter baumannii*). Data on the mutant selection window for
Concentration-dependent bacterial killing, toxic effects related mostly to trough concentrations, and a prolonged postantibiotic effect has led to development of extended-interval aminoglycoside dosing strategies.

Aminoglycosides

Aminoglycosides such as gentamicin, tobramycin, and amikacin are used more commonly again because of their broad spectrum of activity against multidrug-resistant gram-negative organisms, particularly as a part of empiric “double-coverage” in critically ill patients. These medications may also be added to other antibiotics for synergy in the treatment of difficult-to-eradicate gram-positive infections such as endocarditis.

As a class, aminoglycosides have concentration-dependent bactericidal activity via irreversible binding to the 30S ribosomal subunit of susceptible bacteria, inhibiting bacterial protein synthesis. In vitro and clinical outcome studies have shown that Cmax:MIC ratios of 8:1 to 10:1 maximize the bacterial killing and clinical success of aminoglycosides. In addition to their primary antibacterial effect, aminoglycosides have a postantibiotic effect: bacterial killing continues even after the concentration at the infection site (eg, blood, tissue) decreases to less than the bacterial MIC. The postantibiotic effect is due to the intracellular, irreversible activity of aminoglycosides at bacterial ribosomes and ranges from 0.5 to 8 hours, depending on the Cmax achieved.

Aminoglycosides are hydrophilic (Vd 0.2-0.3 L/kg) and doses should be based on ideal body weight or, in obese patients, on adjusted body weight. Aminoglycosides are almost exclusively renally cleared, leading to increased urinary concentrations, a potential advantage in treating urinary tract infections. In contrast, aminoglycosides penetrate lung tissue and cerebrospinal fluid poorly, so higher doses may be required to achieve target Cmax at these infection sites. Primary toxic effects associated with aminoglycosides are nephrotoxic and ototoxic conditions, which have been correlated with elevated trough concentrations and/or prolonged duration of exposure rather than with peak concentration.

The combination of concentration-dependent bacterial killing, toxic effects related mostly to trough concentrations, and a prolonged postantibiotic effect has led to development of extended-interval aminoglycoside dosing strategies. Compared with other strategies, with extended-interval strategies, higher doses are given less often (eg, usual dose given once daily rather than divided and given 3 times a day) to maximize the peak drug concentration and allow time for almost complete drug clearance from the blood (ie, undetectable trough) to avoid prolonged exposure of renal and otic tissues.

If the drug concentration at the site of infection decreases to less than the MIC, the postantibiotic effect allows for a 6- to 8-hour “drug-free” interval during which bacterial regrowth is continually suppressed and the toxic effects of the drug may be minimized. Comparisons of aminoglycoside dosing strategies indicate equivalent or superior efficacy and equivalent or decreased nephrotoxic and ototoxic effects with extended-interval compared with conventional dosing strategies.

Aminoglycosides can be infused quickly, typically in 30 minutes, and are often administered every 24 to every 48 hours in critically ill patients and thus most
likely will not interfere with administration of other antibiotics. Furthermore, aminoglycosides are Y-site compatible with vancomycin and cefepime but have different compatibilities with the other broad-spectrum antibiotics discussed here. Dosages must be individualized, a situation that may be associated with a slight delay in therapy because of the time needed for admixture in the pharmacy.36,59

Aminoglycoside therapy must be monitored closely to ensure efficacy and minimize toxic effects, often necessitating determination of drug levels 1 or 2 times after a dose is administered. Patients in stable condition without contraindications to aminoglycoside therapy can be managed according to an extended-interval aminoglycoside nomogram, also known as the Hartford nomogram, which allows administration of gentamicin or tobramycin 7 mg/kg with dosage adjustments made on the basis of the drug level in a blood sample obtained 8 to 12 hours after administration of the aminoglycoside.51 Critically ill patients, however, have alterations in aminoglycoside V_d and clearance. Aminoglycoside nomograms have not been validated in critically ill patients. Patient-specific pharmacokinetic calculations should be considered for critically ill patients and others with altered V_d by obtaining serum samples for determination of drug level at 2 different times (eg, 3 and 10 hours after administration of a dose) to ensure that the goal for peak level of drug and appropriate dosing interval has been selected.60 Assay of a serum sample obtained at random may also be useful to assess aminoglycoside clearance. Bedside nurses’ understanding of the monitoring plan is imperative so that blood samples for serum drug levels are obtained at appropriate times to ensure dose optimization.

Frequency of monitoring depends on a patient’s clinical status, and no formal recommendations exist to guide practice. Aminoglycoside serum levels may be considered after the initial dose, after dosage adjustments, with changes in renal function, and at least once to twice weekly thereafter to ensure adequate clearance.

**Vancomycin**

Vancomycin is a glycopeptide antibiotic that has been used in critically ill patients for more than 50 years as empiric and definitive treatment of infections caused by methicillin-resistant *Staphylococcus aureus* and other gram-positive pathogens. Vancomycin binds to the D-alanyl-D-alanine cell wall precursor involved in peptidoglycan cross-linking, inhibiting cell wall synthesis.61 This antibiotic is slowly bactericidal and primarily has time-dependent killing, with an AUC:MIC ratio of 400 hours or more or C_{max} greater than 4 to 5 times the MIC best correlated with bacterial killing.62,63 As with fluoroquinolones, the AUC:MIC ratio is a difficult parameter to calculate and apply at the bedside, so monitoring of vancomycin serum trough concentrations as a surrogate is recommended by clinical treatment guidelines.64

Vancomycin is primarily cleared renally and is hydrophilic (V_d 0.4-1 L/kg).65 Guidelines recommend that doses of vancomycin be based on actual body weight; however, limited data are available to guide dosing in obese patients.64 Controversy exists about whether actual or adjusted body weight is most appropriate for weight-based dosing in morbidly obese patients, so therapeutic drug monitoring is essential to ensure efficacy and safety of therapy in these patients.66 Although vancomycin concentrates well in the urine, skin, and soft tissues, it has relatively poor penetration into deeper tissues such as lung and cerebrospinal fluid, necessitating more aggressive dosing strategies for infections in these areas.65

The nephrotoxic and ototoxic effects historically associated with vancomycin most likely were due to impurities in early formulations. Although modern formulations are far less impure, the incidence of nephrotoxic effects is increased with concomitant administration of nephrotoxins such as aminoglycosides and amphotericin B.64,67 Retrospective studies65,66 have revealed other risk factors for the nephrotoxic effects of vancomycin, including prolonged duration of treatment, trough concentrations greater than 15 to 20 mg/L, and total daily dose greater than 4 g, although corroboration through larger, adequately powered prospective studies is warranted. Concomitant piperacillin/tazobactam has also been postulated to increase the risk for nephrotoxic effects, but adequately designed, prospective studies are needed to validate these claims.68-72

Although vancomycin was formerly administered at a fixed dose (eg, 1000 mg intravenously every 12 hours), current guidelines recommend individualized dosing based on the patient’s weight, renal function, and
severity of illness to optimize efficacy and reduce toxic effects. A minimum vancomycin trough serum concentration of 15 mg/L is necessary to achieve an AUC:MIC ratio of 400 hours or greater for bacterial strains with an MIC of 1 mg/L. Trough concentrations of 15 to 20 mg/L should be targeted for complicated infections (eg, bacteremia, endocarditis, osteomyelitis, meningitis, and pneumonia) to maximize vancomycin concentration at the site of infection; troughs of 10 to 15 mg/L may be appropriate for uncomplicated infections (eg, urinary tract, skin, soft tissue). Total daily vancomycin doses of 30 to 40 mg/kg (eg, 15-20 mg/kg every 8 to 12 hours) are often necessary in critically ill patients to achieve these trough concentrations. Initial loading doses (eg, 25-30 mg/kg) may be considered to attain therapeutic levels quickly in critically ill patients in whom the medication might otherwise not reach steady state for several days.

Emergence of methicillin-resistant strains of *S aureus* with higher vancomycin MICs (eg, > 1 mg/L) has necessitated targeting of higher trough levels to achieve an AUC:MIC of 400 hours or greater, challenging the safety and usefulness of vancomycin for infections caused by these organisms. In addition, vancomycin MICs greater than 1 mg/L may be a marker for heterogeneous vancomycin resistance, which could lead to unsuccessful treatment, although methods for MIC determination differ by institution, making this cutoff unpredictable depending on the technique used. Nevertheless, guidelines suggest consideration of alternative therapies for methicillin-resistant strains of *S aureus* with vancomycin MICs of 2 mg/L or higher.

Bedside nurses play an important role in vancomycin monitoring. The so-called red man syndrome characterized by flushing of the face, neck, and upper part of the torso can occur with rapid intravenous infusion. In order to avoid red man syndrome, vancomycin doses must be administered slowly at a rate less than 1 g/h and should be diluted to at least 4 mg/mL. When administered via peripheral catheters. Thus, typical dosages of 15 mg/kg every 8 hours may require several hours per day of infusion time and may contribute markedly to fluid intake. Of the agents discussed, vancomycin is Y-site compatible with meropenem and doripenem, levofloxacin, and the aminoglycosides but has different compatibilities depending on antibiotic concentration for the remaining agents. Dosing must be individualized, a requirement that may necessitate some delay in initiation of therapy because of the need for admixture by the pharmacy.

Steady-state therapeutic trough monitoring is recommended for patients with goal troughs of 15 to 20 mg/L, patients at risk for nephrotoxic effects or with unstable renal function, and patients with prolonged courses of therapy (eg, > 3-5 days). A steady state occurs between the fourth and fifth drug half-life, which typically correlates with the fourth or fifth dose. Monitoring frequency depends on the stability of the patient’s clinical status; subsequent measurements should be made when changes in clinical status occur. Trough concentrations should be checked at least weekly for patients with stable hemodynamic status and more frequently for patients in unstable condition. As is the case with aminoglycosides, bedside nurses must be aware of the monitoring plan for vancomycin to ensure that serum trough concentrations are determined at appropriate times during therapy.

**Conclusions**

Understanding antibiotic-specific pharmacokinetic and pharmacodynamic principles is essential for all health care professionals who provide care for critically ill patients. Pharmacokinetic changes in critically ill patients along with increases in microbes’ resistance to antibiotics have resulted in exploration of new dosing strategies (eg, extended vs continuous infusion of β-lactams), higher doses (eg, vancomycin), and specific monitoring parameters (eg, vancomycin, aminoglycosides). Nursing implications include availability of intravenous catheters, intravenous compatibility of drugs, monitoring for toxic effects and efficacy, timing of administration, and appropriate change of therapy when a patient goes home. Additionally, patients with septic shock may benefit from standard infusion rates to achieve guideline recommendations for administration of antibiotic within 1 hour of the onset of shock. Although additional clinical studies are needed to better define the effects of antibiotics on patients’ outcome and the usefulness of the medications in broader populations of patients, the lack of novel antibiotic agents.
progression of antibiotic resistance, and evolving information about pharmacokinetic changes reinforce considerations for optimizing antibiotic use on the basis of pharmacodynamic properties of available antibiotics, especially in critically ill patients with moderate to severe infections caused by difficult-to-treat pathogens. CCN

Financial Disclosures
None reported.

eLetters
Now that you’ve read the article, create or contribute to an online discussion about this topic using eLetters. Just visit www.ccnonline.org and select the article you want to comment on. In the full-text or PDF view of the article, click “Responses” in the middle column and then “Submit a response.”

dotmore

References
9. Andrews J. Determination of minimum inhibitory concentrations. J Anti-

microbiol. 2001;48(suppl 11):5-16.
18. Kim A, Sutherland CA, Kutli JI, Nicolau DP. Optimal dosing of piperacillin-
20. Burgess DS, Hastings RW, Hardin TC. Pharmacokinetics and pharma-

26. Tam VH, Louie A, Lomaestro BM, Drusano GL. Integration of population pharmacokinetics, a pharmacodynamic target, and microbiologic surve-

28. Novelli A, Ademehri C, Livi P, Fallani S, Maziere T, De Gaudi AR. Pha-

30. Jaruratanasriruk S, Sriwiriyajan S, Punyo J. Comparison of the pharma-
codynamics of meropenem in patients with ventilator-associated pneu-


32. Krueger W, Bultita J, Kinzig-Schippers M, et al. Evaluation by Monte Carlo simulation of the pharmacokinetics of two doses of meropenem administered intermittently or as a continuous infusion in healthy vol-
34. Arnold HM, Hollands JM, Skruply LP, et al. Prolonged infusion antibi-
35. Rotschafer J, Ulmann M. Comparative pharmacodynamics of intermit-
tent and prolonged infusion of piperacillin/tazobactam using Monte Carlo simulation and steady-state pharmacokinetic data from hospital-
With a long history of quality and innovation, you can trust the AnchorFast product.

- Used at over 3,700 hospitals in the United States
- 20 years of demonstrated quality
- Ongoing innovation driven by customer feedback

Learn more at hollister.com/anchorfast
or call 888.740.8999

CAUTION: Federal (USA) Law restricts this device to sale by or on the order of a physician. Prior to use of the AnchorFast oral endotracheal tube fastener, be sure to read the entire product instructions for use package insert that accompanies the product. The Hollister logo and AnchorFast are trademarks of Hollister Incorporated.

© 2016 Hollister Incorporated
Continuous renal replacement therapy is currently used as a standard treatment for acute kidney injury in the intensive care unit, particularly for patients with unstable hemodynamic status. Because this therapy is continuous, for days or weeks, and the extracorporeal blood circuit is large, the circuit is prone to clotting. Several methods of keeping the extracorporeal circuit patent are available, including heparin infusion, flushes with physiological saline, use of thrombin inhibitors, and citrate. This article reviews methods for continuous renal replacement therapy, anticoagulation, efficacy, and implications for bedside critical care. (Critical Care Nurse. 2016;36[2]:34-42)

Continuous renal replacement therapy (CRRT) is often used as a standard treatment for acute kidney injury in the intensive care unit, particularly for patients with unstable hemodynamic status. CRRT is a continuous dialysis therapy with several different techniques and capabilities. For days or weeks, blood is removed from the patient by using various types of double-lumen dialysis catheters, pumped through a dialyzer (typically termed a hemofilter), and returned to the patient via the CRRT machine. During this treatment, plasma water and wastes are removed from the blood gradually and continuously, avoiding the large fluid shifts that accompany intermittent hemodialysis.

CRRT requires pumping the blood through a blood tubing circuit. Any time the blood comes in contact with the tubing, it reacts as if the tubing is a foreign substance. Consequently, when blood is exposed to the tubing, 2 main mechanisms of thrombus formation are activated: the intrinsic and the extrinsic pathways of blood coagulation, platelet adhesion, and platelet activation. In addition, clots tend to form when the blood flows through this tubing because pumping the blood causes turbulence and high shear rates, which activate platelets. Also, the dialysis tubing is not lined with endothelium, the tissue responsible for preventing clotting within the vasculature. Therefore, the CRRT circuit is prone to clotting.
Clot formation in the hemofilt
e, as well as stopping and restarting the CRRT circuit because of frequent alarms, limits the benefits of CRRT by limiting the treatment time, therefore reducing the treatment dose. The circuit for CRRT for adults typically holds approximately 150 to 250 mL, depending on type of circuit and the size of the hemofilt e. Therefore, sudden clotting and an inability to return the patient’s blood can account for potentially large blood loss. In addition, the patient’s hemodynamic status may become unstable because of the loss of blood, and increased fluid administration may be needed. Another important consideration when the CRRT circuit is changed more often than usual because of clotting is the potential for central catheter–associated bloodstream infections. This potential increases every time the CRRT blood tubing is disconnected from the catheter, because the dialysis catheters are central blood catheters. In addition, changing the CRRT circuit often is not only expensive but also time-consuming for bedside nurses, causing an increase in nursing workload.

Several options are available to prevent clotting in the CRRT circuit. In addition to anticoagulation, other aspects of CRRT therapy such as catheter choice and therapy choice may affect clotting in CRRT.

Factors That Affect Clotting Catheters

The type and size of catheter used and the site of insertion are important. Dual-lumen catheters are commonly used for CRRT, and they are placed in large veins to ensure adequate blood flow. Catheters are classified into 2 groups according to the intended use and duration. Acute or short-term catheters are typically used less than 2 weeks; chronic or long-term catheters are often tunneled but can also be used for CRRT.

Some of the long-term catheters may be used for up to several weeks or more. Compared with other types, catheters that are semirigid and longer provide improved blood fl w and less interruption of the therapy.

The site for vascular access in CRRT is also important to maintain a sustained blood fl w without interruptions. The subclavian vein, the femoral vein, and the internal jugular vein are common sites used for access. The subclavian vein has been used often in the past for CRRT access. However, the Kidney Disease Improving Global Outcomes (KDIGO) guidelines recommend using the subclavian vein as a last resort because of the increased incidence of central vein stenosis associated with use of this vein.

The femoral vein is another option for CRRT access, but it is often the least desirable for several reasons. This vein has a high potential for infection if the patient has wounds, diarrhea, or drainage near the site. Patients who have increased intra-abdominal pressure can have increased access pressures, and therefore access alarms may be more frequent than usual when the femoral site is used. In addition, the femoral vein is a site where kinking can occur when a patient is placed in a position such as in the high Fowler’s position to improve ventilation. If early mobility is a priority, the femoral vein may not be a desirable site for long-term treatment.

The right internal jugular vein is the vessel of choice for CRRT because it is the shortest, most direct vascular route to the right atrium and therefore provides potentially better blood fl w. The left internal jugular vein can also be used, but it has a longer and less direct route to the right atrium.

Additionally, arteriovenous grafts or fistulas have been used when other access is limited, although these routes are not the access of choice in CRRT. Grafts or arteriovenous fistulas are typically accessed with large dialysis needles, a procedure that requires careful immobilization of the needles to prevent tearing the graft. The potential for disruption of blood fl w or puncture of the graft or fistula with the needle exists each time the patient is moved or turned. These situations are usually no problem during a typical hemodialysis treatment, but in CRRT, the extremity must be immobilized much longer than in typical hemodialysis because CRRT can last several days. Only a few studies indicate that fistulas or grafts are the access of choice in CRRT, although these access sites were considered safe in 1 study.
Therefore, these types of access are markedly less desirable for CRRT treatments.

Blood Flow Rates

The rate of blood flow is also a factor that can influence the patency of the CRRT circuit. Insufficient rates can be a primary cause of clotting in the CRRT hemofilter. Although blood flow does not need to be as fast as in a hemodialysis treatment, blood should move through the circuit at a rate fast enough to prevent clotting. Typically, blood flow rates in CRRT are 200 to 300 mL/min.

Therapy Choice and Prevention of Clotting

The type of CRRT chosen and the use of fluids can help prevent clotting in some CRRT therapies. Depending on the therapy chosen, fluids in CRRT are administered into the blood before the hemofilter (predilution continuous venousous hemofiltration) or after the hemofilter (postdilution continuous venousous hemofiltration) or infused into the outside compartment of the hemofilter as a dialysate. Infusion of fluids before the filter can cause dilution of the blood and thus reduce the hematocrit of blood entering the hemofilter, leading to less hemocoagulation. Infusion of these same fluids after the filter has no effect on the blood entering the hemofilter. Dialysate fluid used in continuous venousous hemofiltration dialysis does not come in contact with the blood and therefore has no effect on the blood viscosity. So in patients in whom anticoagulation is not desirable, predilution continuous venousous hemofiltration may prevent clotting.

Nurses’ Education

Nurses’ education, knowledge of alarm troubleshooting, and quick response to the alarms are also imperative to minimize circuit disruption and prolong circuit life. Each time an alarm occurs, the blood pump stops in the CRRT machine and blood flow stops. If the blood pump is not restarted quickly, blood stagnation occurs in the extracorporeal circuit, and the blood begins to clot. A well-trained CRRT nurse may be able to identify the source of the alarm and intervene in a timely manner to prevent clotting of blood in the circuit and unnecessary blood loss. However, as mentioned earlier, delays in responding or incorrect diagnosis of the cause of the alarm and subsequent clotting may also lead to time without treatment, reducing fluid and solute removal over time and decreasing treatment efficiency.

Anticoagulation for CRRT

Nonanticoagulant strategies are available for patients who are treated without anticoagulants, including a well-functioning access, high blood flow rates, therapy choice, and quick response to alarms. CRRT circuit clotting is inevitable, so some method of extending the lifespan of the CRRT circuit is often desirable. When anticoagulation is used, the ideal method yields inhibition of platelet activity and thrombotic activity in the circuit, can be easily titrated and reversed, and minimizes complications such as bleeding and other side effects. Although anticoagulation does increase the chance of bleeding, changing the circuit often because of clotting is costly. Therefore, some type of anticoagulation is commonly used.

Saline Flush

The simplest method of clotting prevention is to use no anticoagulant and, instead, flush the circuit on a regular basis with physiological saline. With this option, small amounts of physiological saline, such as 100 mL, are infused into the CRRT circuit before the filter on a regular basis. The saline dilutes the blood travelling through the dialyzer, minimizing the hematocrit of the blood as the blood passes through the dialyzer. This method is simple, but the fluid infused each hour must be accounted for in fluid balance calculations. Obviously, if the fluid is not counted, the patient can experience fluid gain as a result. Studies on flushing with physiological saline have shown mixed benefits in the prevention of circuit clotting. Saline flushes might be a better option for patients who have a high risk for bleeding or have a low platelet count or a prolonged activated partial thromboplastin time (aPTT) or international normalized ratio.

Heparin

Use of heparin is another simple method of anticoagulation. Heparin potentiates antithrombin III, resulting in inhibition of thrombin and factor Xa. Level of
anticoagulation can easily be measured by determining the aPTT.\textsuperscript{1,4,6} Heparin has a half-life of 90 minutes, which can be increased up to 3 hours in patients with renal insufficiency.\textsuperscript{1,4,6,20} Heparin is inexpensive and easy to monitor, and its effects can be reversed with protamine. Studies\textsuperscript{21,22} have indicated that the incidence of bleeding is greater with heparin administration than with other types of anticoagulation. Contraindications for the use of heparin include patient characteristics such as an intolerance to heparin, development of heparin-induced thrombocytopenia, and an increased chance of bleeding.\textsuperscript{1,3,4} Heparin is usually administered as a bolus followed by a continuous intravenous infusion into the arterial side of the CRRT circuit. Various protocols exist for the administration. The aPTT is monitored and typically maintained at 1.5 to 2.0 times normal.\textsuperscript{1} In a study by Uchino et al,\textsuperscript{22} however, the lifespan of the circuit with heparin did not differ significantly from the lifespan when no anticoagulant was used.

**Heparin-Protamine**

Another method of anticoagulation is to infuse heparin, before the filter, into the arterial side of the CRRT circuit and then to infuse protamine into the venous side of the circuit to counteract the effect of the heparin. This method, termed regional heparinization,\textsuperscript{23} is effective, enables anticoagulation of the circuit, and minimizes the risk for bleeding in the patient. Disadvantages of this therapy include difficulties standardizing the protamine dosage and protamine infusion. This method of anticoagulation is also associated with hypotension, anaphylaxis, cardiac depression, leukopenia, and thrombocytopenia.\textsuperscript{1} Although this method has been used and can be effective, its efficacy in prolonging the lifespan of the circuit is variable.\textsuperscript{23}

**Low-Molecular-Weight Heparin**

Low-molecular-weight heparin (LMWH) can also be used in CRRT anticoagulation. Patients treated with LMWH have a lower incidence of heparin-induced thrombocytopenia and have a lower antithrombin affinity than they do with heparin, less activation of platelets and polymorphonuclear cells, and less inactivation by platelet factor-4.\textsuperscript{1,4,24} LMWH is more expensive than heparin, and its effects are harder to reverse with protamine. In addition, a special anticoagulation test (anti Xa assay) is required for monitoring, a characteristic that also increases the cost. Research\textsuperscript{24} has indicated mixed results for whether or not the use of LMWH improves the lifespan of the circuit. Because LMWH is excreted through the kidneys, its effects are prolonged in patients with renal failure.\textsuperscript{1}

**Thrombin Inhibitors**

Thrombin inhibitors are another type of anticoagulant that can be used in CRRT. They inhibit free and clot-bound thrombin and do not react with antibodies to heparin.\textsuperscript{25,26} Thrombin inhibitors such as argatroban are useful in patients who have heparin-induced thrombocytopenia as a result of heparin anticoagulation.\textsuperscript{25} These inhibitors are more expensive than heparin but provide effective anticoagulation. The KDIGO guidelines\textsuperscript{3} recommend the use of thrombin inhibitors rather than other anticoagulants or no anticoagulant. Argatroban is cleared by the liver\textsuperscript{1,25} and has a half-life of 31 to 59 minutes.\textsuperscript{25,26} Dosage includes a loading dose and a maintenance dose adjusted as necessary to keep the aPTT 1 to 3 times the reference level.\textsuperscript{1,27,28}

If bleeding occurs during anticoagulation with argatroban, the infusion can be turned off. In an ex vivo study,\textsuperscript{29} the effects of argatroban could be reversed by using recombinant activated factor VII. In patients with hepatic failure or with multiple organ dysfunction syndrome and sepsis, the clearance of argatroban may be markedly reduced. In a study in animals,\textsuperscript{30} a benefit of thrombin inhibitors was their additional properties of improving the microcirculation and reducing leukocyte adherence, events that may be helpful in patients with septic disseminated intravascular coagulopathy. To date, no studies in humans have been done on this aspect of the inhibitors.

**Citrate**

Use of citrate is an alternative to regional heparinization or use of other anticoagulants to prevent clotting and prolong circuit life. In the United States, citrate is used as an off-label drug. The KDIGO guidelines\textsuperscript{3} recommend the use of citrate for anticoagulation during CRRT in patients who do not have shock or severe liver failure and in facilities that have an established protocol for citrate anticoagulation.
Citrate works by chelating ionized calcium in the blood and therefore inhibits the clotting cascade.\textsuperscript{21,31-34} With some protocols, a citrate solution is infused before the filter in the CRRT circuit in relation to blood flow rate.\textsuperscript{22,34,35} With this method, the amount of ionized calcium in the circuit after the filter is monitored routinely; the typical goal is a concentration less than 0.35 mmol/L to ensure that the calcium level is low enough to suppress clotting.\textsuperscript{21,31,34} The citrate infusion is then adjusted accordingly. Other methods of citrate administration include using a citrate-containing replacement solution infused before the filter, a method that also requires monitoring the concentration of ionized calcium in the circuit after the filter.\textsuperscript{36}

Regardless of the protocol followed for the infusion of citrate, a replacement infusion of calcium is administered at the end of the extracorporeal circuit or via a separate venous access to correct for the calcium that is chelated and also to replace any calcium chelated in the blood or lost into the ultrafiltrate or dialysate.\textsuperscript{31,34,37} The calcium infusion must be administered after the filter or into a central catheter to prevent tissue sloughing and necrosis, which can occur with infiltration.\textsuperscript{31,38} Generally, because calcium interferes with the action of the citrate, calcium-free dialysate or replacement fluids are often used with citrate anticoagulation.\textsuperscript{4} Some researchers,\textsuperscript{39,40} however, have shown that citrate anticoagulation can be effective with the use of calcium-containing replacement fluids, eliminating the need for a separate calcium infusion.

Any remaining citrate that returns to the patient is metabolized to bicarbonate by the liver, kidney, and skeletal muscle.\textsuperscript{1,31,34} Patients with severe liver failure and lactic acidosis may have difficulty with citrate metabolism, leading to an elevated blood level of citrate-chelated calcium. This elevation is a toxic effect of citrate. Its hallmark signs are a low systemic level of ionized calcium and an elevated level of total serum calcium. The toxic effects of citrate can lead to metabolic acidosis and an increased anion gap.\textsuperscript{31} Frequent monitoring of electrolyte levels, especially the level of ionized calcium, and acid-base status is required because of the potential for hypernatremia, metabolic alkalosis, and systemic ionized hypocalcemia.\textsuperscript{1,31}

If citrate is infused as a solution, it can be infused as a mixture of a trisodium citrate solution and an acid-citratedextrose solution.\textsuperscript{31,38} Whatever the choice of citrate, the citrate dose must be titrated to achieve effective anticoagulation.

Depending on the type of CRRT machine used, the citrate and calcium may be infused via independent intravenous pumps. If calcium is administered as an independent infusion, each time the CRRT pump is stopped, the calcium infusion should also be stopped to prevent the adverse effects of hypercalcemia, such as decreased heart rate, hypotension, and coma.\textsuperscript{41} In addition, failure to start the calcium infusion after the citrate infusion has started can cause not only anticoagulation and the potential for bleeding but also hypocalcemia resulting in seizures, bronchospasm, and prolongation of the QT interval.\textsuperscript{42} In some countries outside the United States, CRRT machines are equipped with citrate and calcium pumps that stop when the CRRT machine is stopped. This setup can prevent the citrate and calcium from continuing to infuse when the blood pump is stopped and may improve the safety of the therapy.

Using citrate for anticoagulation not only can provide a longer circuit life and improve the delivered dose for CRRT but also may be associated with less inflammation because of hypocalcemia-induced suppression of intracellular signaling at the cell membrane, which may have anti-inflammatory properties.\textsuperscript{31} A systematic review\textsuperscript{21} of randomized controlled trials on the safety and efficacy of citrate have indicated that citrate is effective in maintaining circuit patency in CRRT when monitoring and adequate protocols are followed. Several studies\textsuperscript{21,36,42,43} indicate that compared with heparin, citrate anticoagulation can reduce the risk of bleeding and the requirement for blood products and causes no increase in episodes of metabolic alkalosis (see Table).

Education of nurses is imperative if a citrate protocol is adopted so that the nursing staff understand the cardiovascular implications of continuous citrate and calcium infusions and the dynamics of citrate metabolism. Implementation of a citrate protocol and ongoing quality assurance helps ensure that patients can be monitored appropriately and managed effectively. Even with a protocol, patients can experience the toxic effects of citrate, especially if they have any degree of hepatic failure. Therefore, the protocol should be individualized.
Platelet-Inhibiting Agents

Prostacyclin (prostaglandin I₂) is a paracrine hormone of the prostaglandin family. It is secreted by the vascular tissue and has various effects related to the sufficiency of blood flow. Prostacyclin is a potent inhibitor of platelet aggregation. The 2 best described effects of prostacyclin are its effects on platelet aggregation and its vasodilatory effects on arterial and venous vessels of all sizes due to relaxation of smooth muscle cells.

As blood travels through the CRRT circuit tubing, it can be activated within the tubing to cause clotting by platelet activation as described earlier. Prostacyclin inhibits platelet binding in the blood in the tubing during CRRT and prevents clotting. Side effects include hypotension, bleeding, and higher cost than other anticoagulants. Currently, in the United States, prostacyclin is not approved for use as an anticoagulant for CRRT, and KDIGO guidelines do not support its use because of a lack of randomized clinical trials.

Summary

Clotting in CRRT is a frequent complication of the therapy. Numerous factors that can influence CRRT circuit life include choice of catheter, catheter access site, blood flow rate, choice of therapy, and choice (if any) of anticoagulation. Frequent clotting in the CRRT circuit can cause many complications, such as excessive blood loss, and unstable clinical status. The decision to use anticoagulation in CRRT should be based on the patient’s condition and the effectiveness of the anticoagulant. As with any therapy used in critically ill patients, proper education of nurses is essential to maximize the effectiveness of the therapy and promote safe patient outcomes.

Financial Disclosures
None reported.
Curos disinfection caps keep IV ports clean and protected.

• Twist On, Stay On
• Colored for Compliance
• Convenient Strip Dispensing

Learn more at www.curos.com


EXERGEN

TemporalScanner™

More than 50 published studies - supporting accuracy from preemies to geriatrics in all areas of care.

The Exergen TemporalScanner™ Temporal Artery Thermometer

• Patients love the TemporalScanner!
• Cost savings of 90% over other thermometry methods
• Lifetime Warranty – unique to thermometry
• Chemical resistant materials stand up to harsh disinfectants
• On-demand, innovative, inserviceing results in successful usage for all levels of nursing skills

To evaluate, email: medical@exergen.com
For general information: www.exergen.com
For clinical information, visit: www.TAThermometry.org
For educational videos, clinical studies, and manuals: www.exergen.com/ww

Exergen Corporation
400 Pleasant Street
Waltham MA 02472
Tel 617-923-9500
Fax 617-923-9511

Learn more at www.curonline.org

Curos disinfection caps keep IV ports clean and protected.

• Twist On, Stay On
• Colored for Compliance
• Convenient Strip Dispensing

Learn more at www.curos.com

CCN Fast Facts

Continuous Renal Replacement Therapy and Anticoagulation: What Are the Options?

Facts
Continuous renal replacement therapy (CRRT) is a standard treatment for acute kidney injury, particularly for patients with unstable hemodynamic status. Because this therapy is continuous, for days or weeks, and the extracorporeal blood circuit is large, the circuit is prone to clotting. Although anticoagulation does increase the chance of bleeding, changing the circuit often because of clotting is costly. Therefore, some type of anticoagulation is commonly used.

Saline Flush
The simplest method of clotting prevention is to use no anticoagulant and, instead, flush the circuit on a regular basis with physiological saline. The saline dilutes the blood travelling through the dialyzer, minimizing the hemoconcentration of the blood as the blood passes through the dialyzer. This method is simple, but the fluid infused each hour must be accounted for in fluid balance calculations.

Heparin
Heparin potentiates antithrombin III, resulting in inhibition of thrombin and factor Xa. Heparin has a half-life of 90 minutes, which can be increased up to 3 hours in patients with renal insufficiency. Heparin is inexpensive and easy to monitor, and its effects can be reversed with protamine.

Heparin-Protamine
Another method is to infuse heparin, before the filter, into the arterial side of the CRRT circuit and then to infuse protamine into the venous side of the circuit to counteract the effect of the heparin. This method, termed regional heparinization, is effective, enables anticoagulation of the circuit, and minimizes the risk for bleeding in the patient.

Low-Molecular-Weight Heparin
Patients treated with LMWH have a lower incidence of heparin-induced thrombocytopenia and have a lower antithrombin affinity than they do with heparin, less activation of platelets and polymorphonuclear cells, and less inactivation by platelet factor-4. LMWH is more expensive than heparin, and its effects are harder to reverse with protamine.

Thrombin Inhibitors
Thrombin inhibitors inhibit free and clot-bound thrombin and do not react with antibodies to heparin. Thrombin inhibitors are useful in patients who have heparin-induced thrombocytopenia as a result of heparin anticoagulation. These inhibitors are more expensive than heparin but provide effective anticoagulation.

Citrate
Citrate works by chelating ionized calcium in the blood and therefore inhibits the clotting cascade. With some protocols, a citrate solution is infused before the filter in the CRRT circuit in relation to blood flow rate.

Nafamostat Mesilate
Nafamostat mesilate inhibits the coagulation system by inhibiting platelet aggregation and coagulation factors. The inhibitor has not been approved for use in the United States for CRRT anticoagulation; it is predominantly used in Japan.

Platelet-Inhibiting Agents
Prostacyclin is a potent inhibitor of platelet aggregation; it inhibits platelet binding in the blood in the tubing during CRRT and prevents clotting. Currently, in the United States, prostacyclin is not approved for use as an anticoagulant for CRRT.

IN SYNC wherever you go.

Introducing Infusomat® Space Pump 2nd Generation

The wireless pump that goes where your patient needs it to, delivering an uninterrupted infusion experience throughout care unit changes. Bringing intuition to infusion with a user-friendly interface, added safety prompts, automatic drug library connections. Synchronized intelligence starts here.

B BraunUSA.com/ Sync
Our Passion Is Making Patient Safety Real

“...I found the curriculum to be challenging but constructed in a way that made goals attainable for those who were employed or learning via distance education.”

-Marianne S. Cosgrove
CRNA, DNAP, APRN Program Director,
Yale-New Haven Hospital School of Nurse Anesthesia

Find the opportunity that inspires you.

Centura Health’s expansive, two-state, fully-integrated health network offers a more diverse range of locations and work settings than any other health provider in the region.

Now hiring ICU RNs for staff and leadership roles throughout Colorado and western Kansas.

Whether you thrive on the energy of a busy Denver-area Level I Trauma Center or prefer the more relaxed feel of a rural or mountain town, we’ll help you find the right nursing opportunity in a community you will love to call home. Enjoy great people, pay and benefits as part of a faith-based, non-profit system that cares about your success! Apply online or join our talent community:

careers.centura.org  (Keyword search: RN ICU)

Denver Metro Area
Email: AmandaHowell@centura.org
Phone: 720-528-0538

Colorado Springs/Southern Colorado
Email: ToddNapikoski@centura.org
Phone: 719-776-4026

Centura Health
Avista Adventist Hospital
Castle Rock Adventist Hospital
Littleton Adventist Hospital
Longmont United Hospital
Mercy Regional Medical Center
OrthoColorado Hospital
Parker Adventist Hospital
Penrose-St. Francis Health Services
Porter Adventist Hospital
St. Anthony Hospital
St. Anthony North Health Campus
St. Anthony Summit Medical Center
St. Catherine Hospital
St. Mary-Corwin Medical Center
St. Thomas More Hospital
Centura Health at Home
Centura Health Physician Group
Colorado Health Neighborhoods
Fight For Life® Colorado

Centura Health is an Equal Opportunity, Non-Nicotine/Non-Tobacco Employer, M/F/D/V.
Overdoses of β-blockers and calcium channel blockers can produce significant morbidity and mortality, and conventional therapies often do not work as treatments for these poisonings. High-dose insulin/glucose therapy has been successful in reversing the cardiotoxic effects of these drugs in cases where the standard therapies have failed, and it appears to be relatively safe. Many successes have been well documented, but the clinical experience consists of case reports, the mechanisms of action are not completely understood, and guidelines for use of the therapy are empirically derived and not standardized. Regardless of these limitations, high-dose insulin/glucose therapy can be effective, it is often recommended by clinical toxicologists and poison control centers, and critical care nurses should be familiar with when and how the therapy is used. (Critical Care Nurse. 2016;36[2]:45-50)

High-dose insulin/glucose therapy, which is also called hyperinsulinemic/euglycemic therapy or insulin-dextrose therapy, has received considerable attention as a treatment for overdoses of β-blockers and calcium channel blockers that are refractory to conventional therapies. Clinical experience using high-dose insulin/glucose to treat cardiotoxic poisonings in humans dates to 1999, and animal experiments before that time provided the theoretical background and the impetus for extension of its use to treat calcium channel blocker overdoses and then β-blocker overdoses.

Despite many articles in medical publications and 15 years of clinical experience with its use, questions remain about how high-dose insulin/glucose therapy works and when and for whom this therapy should be applied. In addition, the dosing guidelines are not standardized. However, high-dose insulin/glucose therapy is commonly used and often recommended for treating β-blocker and calcium channel blocker poisoning, and critical care nurses should be familiar with the therapy.

This article has been designated for CE contact hour(s). The evaluation tests your knowledge of the following objectives:
1. Identify 2 clinical indications for the use of high-dose insulin/glucose therapy
2. Identify 2 mechanisms of action of high-dose insulin/glucose therapy
3. Identify 2 common complications of high-dose insulin/glucose therapy

To complete evaluation for CE contact hour(s) for test #C162, visit www.ccnonline.org and click the “CE Articles” button. No CE test fee for AACN members. This test expires on April 1, 2019.

The American Association of Critical-Care Nurses is an accredited provider of continuing nursing education by the American Nurses Credentialing Center’s Commission on Accreditation. AACN has been approved as a provider of continuing education in nursing by the State Boards of Registered Nursing of California (#0036) and Louisiana (#LSBN12).

©2016 American Association of Critical-Care Nurses doi: http://dx.doi.org/10.4037/ccn2016370
The terms conventional and standard therapy are defined here as atropine, calcium, cardiac pacing, glucagon, intravenous fluids, phosphodiesterase inhibitors, sympathomimetics, and vasopressors.

β-Blockers competitively block the effect of catecholamines at β-adrenergic receptors. This blockade decreases the intracellular production of cyclic adenosine monophosphate (cAMP), and this in turn reduces the amount of intracellular calcium available for cell depolarization and contraction. Calcium channel blockers act as antagonists at L-type voltage-sensitive calcium channels in the myocardium, the vascular bed, and the pancreas. Blockade of these ion channels disrupts membrane depolarization and prevents movement of calcium into the cell, which limits the release of calcium that is stored in the sarcoplasmic reticulum. For both of the drugs, disruption of calcium homeostasis causes a negative chronotropic and inotropic effect and vasodilatation. Calcium channel blocker poisoning also impairs glucose metabolism; this effect is discussed later in the article.

When taken in toxic doses, β-blockers can cause profound bradycardia and hypotension, and the mechanism of action of the drug prevents normal physiological compensation and limits the effectiveness of many standard therapies. An overdose of calcium channel blockers can cause bradycardia, hypotension, and hyperglycemia, and like β-blocker poisoning, severe poisoning due to calcium channel blockers is often refractory to standard therapies.

Note: The toxicity of β-blockers is not limited to beta-blockade. Lipid-soluble β-blockers such as propranolol can cross the blood-brain barrier and may cause seizures. Atebutolol, betaxolol, and propranolol have membrane-stabilizing effects similar to the effects of quinidine and can cause prolongation of the QRS interval. These effects of β-blocker overdose are relatively uncommon and are not discussed in this article.

The toxic doses of β-blockers and calcium channel blockers are not known. The following are evidence-based guidelines that are commonly used by US poison control centers to determine how much of a specific β-blocker or calcium channel blocker may be dangerous. These are guidelines only, and each case must be evaluated individually as patients may experience toxic effects at lower or higher doses (Table 1).

Regular release β-blockers and calcium channel blockers typically begin to affect blood pressure and heart rate several hours after ingestion. The onset of effects after ingestion of sustained release (SR) preparations is variable and may be delayed. A delayed onset of
effects for more than 6 hours and up to 15 hours after ingestion of SR calcium channel blockers has been reported, and the maximum clinical effects produced by overdose also may be delayed. The referral guidelines for β-blockers recommend a minimum of 6 hours of observation after ingestion of an immediate-release (IR) β-blocker and a minimum of 8 hours of observation after ingestion of an SR β-blocker. The referral guidelines for calcium channel blockers state that asymptomatic patients who have ingested an SR product are unlikely to show toxic effects 18 to 24 hours after ingestion.

Mechanisms of Action of High-Dose Insulin/Glucose Therapy

The common opinion is that high-dose insulin/glucose therapy works by 3 mechanisms of action to reverse cardiotoxic poisoning:

1. Positive inotropy: The positive inotropic effects of insulin have been demonstrated in many animal experiments. These effects may be due to the effect of insulin on glucose metabolism and/or to a separate mechanism.
2. Vasodilatation: Insulin dilates peripheral vessels.
3. Metabolic effects: Calcium channel blocker poisoning inhibits the L-type voltage-sensitive ion channels in the pancreas, decreasing insulin release. Insulin resistance is increased, as well, and hyperglycemia is a common effect of calcium channel blocker poisoning. Concurrent with these effects, the hemodynamic effects of overdose “switch” the energy substrate of the myocardium from free fatty acids to glucose, but the decreased insulin release and increased insulin resistance prevent glucose utilization. High-dose insulin/glucose therapy allows myocardial glucose uptake and provides a source of glucose.

These 3 physiological effects increase blood pressure and tissue perfusion in cases of β-blocker and calcium channel blocker poisoning, presumably by (1) dilating the peripheral vasculature, (2) increasing myocardial uptake of glucose, (3) providing the heart with a source of energy, and (4) acting as a positive inotropic agent.

High-dose insulin/glucose therapy has clearly been effective for treating overdoses of β-blockers and calcium channel blockers, but why it is effective is not clear. The positive inotropy, vasodilatation, and changes in glucose metabolism documented in the literature are from tissue and animal experiments involving calcium channel blockers or from what is known of the pharmacological and toxicologic actions of calcium channel blockers and (to a lesser degree) β-blockers. The evidence supporting these mechanisms of action appears to be good, but how—or if—these mechanisms act as the basis of the therapeutic effects of high-dose insulin/glucose therapy has not been proven.

Clinical Experience

Treatment of severely poisoned patients with the conventional therapies often fails to work and inotropic agents and vasopressors can increase systemic vascular resistance and myocardial oxygen demand, effects that are not beneficial in these situations.

Holger et al performed a retrospective review of charts for 12 patients who had cardiogenic shock after overdose of a β-blocker, a calcium channel blocker, a combination of those drugs, a tricyclic antidepressant, or multiple drugs. High-dose insulin/glucose was used as the primary therapy. No patients were given a vasopressor or glucagon, but all 12 were initially given intravenous fluid resuscitation and calcium supplementation. Eleven of the 12 survived. Unlike other studies, a standardized insulin/glucose protocol was used for all of the patients. However, the small number of patients, differing severity of clinical presentation, and the multiple drugs ingested make interpretation of the results difficult.

Shah et al reviewed the literature from 1999 to 2010 for case reports and case series, and they located 28 cases. Most cases involved poisoning with calcium channel blockers, but several involved co-ingestants. Three of the case reports were published as abstracts and 12 were part of 2 case series by Greene et al and Yuan and Kerns that are reviewed next. All of the patients had received conventional therapy at some point: the time at which conventional therapy was applied varied considerably from case to case. The survival rate was 80% (this includes the cases published by Greene et al). Two cases in which this therapy was considered to have failed were published as abstracts, and little clinical information is available. One patient actually received a low dose of insulin, 1 to 2 units per hour; this patient survived.

Greene et al published the results of a prospective observational study of 7 cases of calcium channel blocker...
poisoning for which high-dose insulin/glucose was used. Six of the 7 survived, but the effectiveness and/or relative contribution of high-dose insulin/glucose in these cases cannot be assessed because all 7 patients had taken co-ingestants and all received other therapies such as atropine, intravenous fluids, or cardiac pacing. The authors noted that 3 of the 7 had a significant and sustained increase in blood pressure that was temporally associated with the onset of the therapy and that adverse effects were minimal and not clinically significant.28

Shepherd and Klein-Schwartz23 reviewed the literature from 1996 to 2004. They located 13 cases of calcium channel blocker overdose for which high-dose insulin/glucose was used: all but 3 had been reviewed by Shah et al.27 Two of these 3 cases were published as abstracts; all 3 patients survived and all 3 received conventional therapy in addition to high-dose insulin/glucose.23

Many case reports about the use of high-dose insulin/glucose for the treatment of β-blocker and calcium channel blocker poisoning have been published. Most report success, but failure has been recorded,29 and underreporting of failures is always possible. Published reports do provide support for the use of high-dose insulin/glucose, but those reports cannot be used to make conclusions about (1) the effectiveness of high-dose insulin/glucose, (2) the superiority of high-dose insulin/glucose compared with conventional therapies, or (3) for whom and when high-dose insulin/glucose therapy should be used. The case reports do contain interesting and useful information. But the reports present patients with differing characteristics, a wide range of clinical presentations, co-ingestions of other potentially cardio-toxic medications, different dosing of high-dose insulin/glucose, and the use of other therapies along with high-dose insulin/glucose, so a useful interpretation of the data from these cases is quite difficult. For obvious reasons, no clinical trials have been done to evaluate the effectiveness of high-dose insulin/glucose as a single therapy for treating severely poisoned patients. No direct comparisons of conventional therapies and high-dose insulin/glucose have been done, although animal studies suggest that high-dose insulin/glucose therapy is more effective.30,31 Some authors have stated that high-dose insulin/glucose therapy should be the first treatment used for symptomatic cases of β-blocker or calcium channel blocker overdose2 and that treatment failures may be caused by delaying its use.27 Others think that the proper use of insulin-dextrose therapy is as an adjunct.23,24,28 In a recent article, Levine et al.32 concluded that overdoses of verapamil or diltiazem can be effectively and safely managed with high doses of vasopressors, and they pointed out the relative lack of experience with (and the absence of) proven dosing and administration guidelines for high-dose insulin/glucose therapy. Olson33 noted that significant evidence is lacking for the effectiveness of all the therapies used to treat calcium channel blocker overdose, and he stressed the need for attention to supportive care and the use of pharmacological interventions on a case by case basis.

**Administering and Monitoring High-Dose Insulin/Glucose**

Using a pharmacological intervention properly requires you to know: (1) which patients will benefit, (2) proper dosing, (3) when to start and stop the treatment, (4) adverse effects, (5) monitoring parameters, and (6) how the intervention will be affected by other drugs or therapies that are being used.

Unfortunately, none of those requirements have been conclusively established for the use of high-dose insulin/glucose therapy. Specific dosing regimens have been used, but these regimens were empirically derived and they differ slightly from each other. But regardless of the differences, the goals of each of the dosing regimens and the monitoring parameters are the same: administration of a high dose of insulin while maintaining euglycemia, and close monitoring of serum levels of glucose and potassium. Three examples of available dosing protocols are given in Table 2. These examples are not intended to be taken as definitive guidelines for administering this therapy: decisions on dosing and therapeutic goals should be individualized for each patient. If fluid overload is or may be a concern, then the 1:1 ratio of insulin to normal saline can be changed to 10:1.

The onset of action is typically seen 30 minutes or more after the infusion has started, but a clinical response has been reported within 15 minutes.3,36 Commonly seen adverse effects are hypoglycemia and hypokalemia, but these effects have not been clinically significant and are easily corrected.39 Glucose infusions of 10% and higher
can be irritating to the veins, so a central intravenous catheter may be needed.

No standardized guidelines specify the duration of therapy, and insulin/glucose has been used for as long as 96 hours.37 No guidelines are available for how to taper and discontinue the infusion.1,2 Serum glucose levels should be monitored closely after the infusion has stopped: published reports indicate that insulin levels may remain elevated for 24 hours after high-dose insulin therapy has been stopped.1,3,4 The decision to use high-dose insulin/glucose as the primary therapy or as an adjunct must be made on a case by case basis.

Conclusion

High-dose insulin/glucose therapy has been used successfully for the treatment of β-blocker and calcium channel blocker overdoses. But documentation of why this therapy works, why it fails, and specific, well-proven guidelines for its use is not available. However, the safety profile of this treatment is good and it is commonly used and prescribed; familiarity with the use of high-dose insulin/glucose should be required knowledge for critical care nurses. CCN

Abbreviation: IV, intravenous.

1 These are examples of empirically derived protocols that have been published; they are not all in agreement. There is no standardized protocol, and clinical judgment should prevail when choosing how to use high-dose insulin/glucose therapy.

References


Letters

Now that you’ve read the article, create or contribute to an online discussion about this topic using eLetters. Just visit www.ccnonline.org and select the article you want to comment on. In the full-text or PDF view of the article, click “Responses” in the middle column and then “Submit a response.”

dotmore

BECAUSE INFECTION RISKS ARE EVERYWHERE, SO ARE WE.

Integrated solutions to help you Be The Difference® in preventing HAI.

That’s why PDI is there for you with educational tools, clinical support, and a portfolio of innovative and effective infection prevention solutions—designed to improve patient outcomes and reduce costs.

Infections don’t wait. Neither should you.

pdihc.com/everywhere
The term thoracic insufficiency syndrome (TIS), introduced in 1992 by Campbell and Smith, is defined as “the inability of the thorax to support normal respiration or lung growth.”¹ TIS may cause respiratory insufficiency, as indicated by a requirement for oxygen or a need for respiratory support via noninvasive or invasive positive pressure ventilation. However, the diagnosis of TIS does not depend on the presence of respiratory insufficiency.² Campbell and Smith recommend the following to aid in diagnosing TIS: “a comprehensive history, physical examination, radiographs, computed tomography scans, lung scans, other imaging studies, arterial and capillary blood gases, pulse oximetry, echocardiograms, and, when feasible, pulmonary function testing.” The severity of TIS and whether or not it is progressive must be determined. Mild TIS may need monitoring but not active intervention. Monitoring for mild TIS may include serial radiographs and physical examination every 4 to 6 months. If untreated, progressive TIS can result in respiratory insufficiency. Goals in treating TIS are to restore thoracic volume and function and to allow for growth of the thorax.¹
Scoliosis is a lateral curvature spinal deformity. Congenital scoliosis with fused or absent ribs can cause a restrictive thoracic deformity that may contribute to TIS. Traditional correction of scoliosis with spinal fusion may be problematic in children who have not reached skeletal maturity. This problem is due to the need for continued spinal and thoracic growth to avoid stunted vertical growth of the spine and further worsening of TIS by restricting the linear size of the thorax. Karol et al reported that patients who had thoracic spine fusions before the age of 9 years had a high rate of severe restrictive lung disease as evidenced by smaller forced vital capacities. In addition, the amount of reduction in the height of the thoracic spine was correlated with smaller forced vital capacities, suggesting an increased risk of restrictive lung disease with more extensive fusions. Separating the effect of the reduced height of the thoracic spine from the effect of the spinal deformity on pulmonary function is a challenge, and both may play a part in the development of thoracic insufficiency syndrome in patients with scoliosis who undergo spinal fusions early in life. Additionally, if the cause of TIS is due to unilateral or bilateral thoracic deformities, correction of scoliosis alone will not address the restricted size of the thorax.

The vertical expandable prosthetic titanium rib (VEPTR) was designed by Dr Robert Campbell in 1989 to specifically address the scarcity of treatments to correct the 3-dimensional deformity of the thorax (Figure 1). A VEPTR was first successfully implanted in a patient at Christus Santa Rosa Children’s Hospital in San Antonio, Texas. The goal of treatment with a VEPTR is “to achieve the largest, most symmetrical, most functional thorax by skeletal maturity.”

A detailed description of the commonly used expansion thoracostomy surgical technique and insertion of a VEPTR device was published in 2004. In summary, an expandable titanium prosthesis is inserted on the posterior part of the rib cage (Figure 1). One end is hooked to a superior rib and the other end is attached to an inferior rib, the spine, or the ipsilateral iliac crest. After implantation, the rods are covered by soft tissues and muscle, and the incision is sutured closed. These VEPTR devices are serially expanded 2 to 3 times a year to allow for growth as the child ages.

In this article, I describe current practices related to the VEPTR procedure in children with TIS and scoliosis and the nursing implications for the care of these patients. Topics include indications, postoperative nursing care, potential complications, long-term follow-up, quality-of-life issues, and implications for critical care and advanced practice nurses.

**Indications**

After a successful single-site, feasibility device trial conducted under an investigational device exemption from the Food and Drug Administration from 1992 to 1994, a multicenter trial was conducted. Similar success in the multicenter trial led to the approval of the VEPTR device under the Humanitarian Device Exemption.
Program in 2004. The approved indication for the VEPTR device is treatment of thoracic insufficiency syndrome in infants and children who are more than 6 months old but have not yet reached skeletal maturity. These patients may have TIS caused by congenital scoliosis with fused or absent ribs, neuromuscular or congenital scoliosis without fused ribs, or hypoplastic thorax syndrome (e.g., Jeune syndrome, Jarcho-Levin syndrome, achondroplasia). A humanitarian use device is meant to benefit patients with diseases or conditions that affect fewer than 4000 individuals in the United States per year.

Campbell and Smith describe 4 types of “thoracic volume-depletion deformities” as follows: type I, rib absence and scoliosis; type II, fused ribs and scoliosis; type IIa, foreshortened thorax; and type IIIb, narrowed thorax (Figure 2).

In types I and II, the thoracic cage is affected unilaterally, whereas in types IIIa and IIIb, the thorax is affected bilaterally. First described by Campbell et al., variations of the VEPTR implantation procedure are used to address these types of deformities.

Over the years, surgeons have expanded use of the VEPTR, working to correct spinal curvatures to maximize thoracic capacity while allowing for linear spinal growth. Other conditions in which a VEPTR has been used include multiple congenital spine deformities, early-onset progressive neuromuscular scoliosis, congenital scoliosis, early-onset scoliosis, infantile idiopathic scoliosis, and myelomeningocele kyphosis.

Only a few published articles delineate experience with VEPTR devices in treating TIS in spondylocostal dysplasia, infantile idiopathic scoliosis, and thoracic kyphosis. In a retrospective review published in 2011, Reinker et al. examined VEPTR devices as a means to control progressive kyphoscoliosis. In an article published in Poland in 2011, Latalski et al. included kyphosis associated with myelomeningocele as an indication for a VEPTR.

Postoperative Management

Length of Stay

In a single-institution study by Campbell et al., a total of 41 patients with congenital scoliosis and fused ribs underwent an opening wedge thoracostomy and VEPTR insertion, and 27 of these were followed up for at least 2 years (mean, 5.7 years). For the initial insertion surgery, among the 27 patients followed up, mean stay in the intensive care unit (ICU) was 7.3 days (range, 2-28 days), and the mean total hospital length of stay was 14 days (range, 9-35 days). After initial insertion surgery, 13 of the 27 required a blood transfusion within the first 2 days after surgery, although blood loss was minimal.

In 2005, Emans et al. reported the results of their clinical trial of VEPTR devices in which they assessed the safety and efficacy of the expansion thoracostomy surgical technique and the device. A total of 31 patients had VEPTRs placed and were followed up for as long as 5.2 years. In this study, postoperative recovery occurred in the ICU after the initial surgery, and patients remained intubated from 8 hours to 9 days. One postoperative intervention to promote wound healing was the use of foam protectors to protect the skin over prominent devices. Additionally, Campbell’s surgical procedure for the lengthenings or expansions was modified to minimize wound dehiscence and infection by shifting the deep muscular incision to an area separate from the small skin incision at the original surgical site.

The trial conducted under an investigational device exemption from the Food and Drug Administration provided data on 214 patients who received a VEPTR and an additional retrospective analysis of 20 patients with spondylocostal dysplasia. Ramirez et al. reported that those patients were hospitalized 5 to 29 days (mean, 11 days).
Schulz et al reported on 8 patients who had VEPTRs placed for infantile idiopathic scoliosis. Gadepalli et al published an article on the 29 VEPTR insertions and 57 lengthening procedures performed on 26 patients in one institution. After initial implantation, mean ICU length of stay was 2.72 days, and mean hospital length of stay was 6.72 days. The postoperative course for the lengthening procedures was characterized by mean stays of 0.26 days in the ICU and 2.44 days in the hospital. Three transfusions were necessary for postoperative bleeding in this study, and 4 patients required placement of a chest tube because of pneumothoraces.

All of these studies were relatively small clinical series with relatively short follow-up periods, and the results varied widely from study to study. Of note, the treatment populations were widely heterogeneous. In some studies, the sample consisted mostly of patients with certain rare syndromes, whereas in other studies, the sample included a mixture of patients with various rare diseases. These differences may explain the variations in hospital and ICU lengths of stay.

Monitoring

In an article published in 2010, DiFazio and Tubman provide a detailed review of the important aspects of postoperative nursing care for patients who have undergone VEPTR implantation. Table 1 is a summary of necessary postoperative monitoring. Monitoring hemodynamic parameters is important in the postoperative period. Vital signs should be documented per ICU standards, generally hourly. In 3 studies, minimal intraoperative blood loss was reported; mean estimated blood loss was 68 mL (range, 5-400 mL), 57 mL (range, 4-220 mL), and 75 mL (range, 25-150 mL). However, as with any surgery, patients may still have fluid shifts in the body and thus require close observation of fluid balance. Additionally, patients may lose blood postoperatively through drains, chest tubes, or oozing surgical sites. Practitioners must monitor electrolyte and hemoglobin levels, correct any metabolic derangements, and decide if transfusions are necessary on the basis of the hemoglobin levels. Bedside nurses must complete frequent neurovascular checks, because this surgery is associated with risk for brachial plexus injury and spinal cord injury. Patients may return from the operating room with a chest tube in place. Campbell et al removed chest tubes once drainage was less than 1 mL/kg per day; however, the time of removal may vary from patient to patient and according to the surgeon’s preference. Patients may also return with a closed-suction medical device (Jackson-Pratt drain) in place for collecting fluids from the surgical site.

Pulmonary Function

In their description of the surgical procedure, Campbell et al noted that most patients remain intubated and are treated with mechanical ventilation for 3 days. Children may remain intubated in the ICU after initial placement of the VEPTR device and then progress toward extubation. After extubation, incentive spirometry and chest physiotherapy are initiated to optimize pulmonary toileting. In a multicenter retrospective study of 54 children who had VEPTRs implanted, acute respiratory insufficiency developed in 3 children in the immediate postoperative period. This finding highlights the importance of frequent assessments of respiratory status. Hasler et al reported no postoperative pulmonary problems in their retrospective study of 23 children who had implantation of a VEPTR device to treat non-congenital early-onset spine deformities.

On the basis of my experience in caring for patients with TIS who required tracheostomy and long-term care, the following is a list of key aspects of care for patients with VEPTRs:

- **Vital signs**: Monitor vital signs hourly until stable.
- **Chest tube drainage**: Monitor chest tube drainage until drainage is less than 1 mL/kg per day.
- **Electrolyte levels**: Monitor electrolyte levels daily.
- **Hemoglobin level**: Monitor hemoglobin levels daily.
- **Neurovascular checks**: Perform neurovascular checks hourly until stable.
- **Pulmonary management**: Perform frequent respiratory assessments.
- **Ventilator management and weaning**: Monitor ventilator settings and weaning protocols.
- **Optimization of oxygen saturations**: Monitor oxygen saturation levels regularly.
- **Chest physiotherapy**: Perform chest physiotherapy daily.
- **Incentive spirometry (after extubation)**: Perform incentive spirometry daily.
- **Analgesia management**: Monitor analgesia requirements.
- **Pain assessments**: Monitor pain levels daily.
- **Patient-controlled analgesia or continuous narcotic infusion with as-needed doses**: Monitor analgesia requirements.
- **Conversion to enteral pain medications once bowel function returns**: Monitor enteral pain medications.
- **Skin care**: Monitor skin care needs.

Table 1: Postoperative monitoring

<table>
<thead>
<tr>
<th>Vital signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest tube drainage</td>
</tr>
<tr>
<td>Electrolyte levels</td>
</tr>
<tr>
<td>Hemoglobin level</td>
</tr>
<tr>
<td>Neurovascular checks</td>
</tr>
<tr>
<td>Pulmonary management</td>
</tr>
<tr>
<td>Frequent respiratory assessments</td>
</tr>
<tr>
<td>Ventilator management and weaning</td>
</tr>
<tr>
<td>Optimization of oxygen saturations</td>
</tr>
<tr>
<td>Chest physiotherapy</td>
</tr>
<tr>
<td>Incentive spirometry (after extubation)</td>
</tr>
<tr>
<td>Analgesia management</td>
</tr>
<tr>
<td>Pain assessments</td>
</tr>
<tr>
<td>Patient-controlled analgesia or continuous narcotic infusion with as-needed doses</td>
</tr>
<tr>
<td>Conversion to enteral pain medications once bowel function returns</td>
</tr>
<tr>
<td>Skin care</td>
</tr>
<tr>
<td>Assessment of dressing each shift</td>
</tr>
<tr>
<td>Dressing change per orthopedic surgeon's plan</td>
</tr>
<tr>
<td>Position changes at least every 2 hours</td>
</tr>
<tr>
<td>No lying directly on incision site</td>
</tr>
</tbody>
</table>

CriticalCareNurse Vol 36, No 2, APRIL 2016
ventilator support, increases in ventilator settings may be needed postoperatively, especially if continuous infusion of a narcotic is required for pain management. Stabilization and expansion of the thorax occurs through serial prosthetic lengthenings. Decreases in ventilator settings or the ability to be weaned from mechanical ventilation may occur with this stabilization, although no definitive evidence of improved pulmonary function after VEPTR placement has been reported. Emans et al\textsuperscript{14} reported improvements in lung volumes and spinal curvature, but at the last follow-up, all patients still had TIS. Gadeppalli et al\textsuperscript{19} did not think that placement of a VEPTR improved overall pulmonary status. Some surgeons are more conservative in their expectations of VEPTR placement; their aim is not curative treatment, but rather stabilization of the thorax and improvement in pulmonary function, thereby improving quality of life for the child and the child’s family.

Another challenge is the lack of a consistent method for measuring pulmonary function to determine the impact of VEPTR placement. Hemoglobin levels have been investigated as a surrogate marker of pulmonary function, owing to limitations of the current techniques of computed tomography of the thorax and pulmonary function tests (PFTs), as PFTs can be used only when a child is developmentally able to perform them.\textsuperscript{5,23} Information on patients with tracheostomies and ventilator dependence who undergo VEPTR implantation is limited. Emans et al\textsuperscript{14} reported that 2 patients who had tracheostomies and were ventilator dependent before VEPTR placement no longer required ventilator support at the last follow-up after lengthening or expansions. Campbell et al\textsuperscript{17} recounted that 1 patient was able to be weaned off continuous positive-airway pressure support and supplemental oxygen and have the tracheostomy removed with no residual oxygen requirement.

### Pain Management

Another aspect of VEPTR postoperative management is pain control. Adequate pain management is important. Management relies on frequent pain assessments with appropriate pain scales and the use of devices for patient-controlled analgesia with intravenous narcotics for patients who are able to use such a device. For patients who cannot use a device for patient-controlled analgesia, a continuous narcotic infusion can be used, with as-needed doses available to be administered by a nurse for breakthrough pain. Transition to enteral pain medications can occur when bowel function returns.\textsuperscript{20} In my experience, diazepam may be used as needed for muscle spasms. Pain after expansion or lengthening and revision surgeries can be managed by using as-needed doses of morphine, oxycodone, acetaminophen, and diazepam for muscle spasms.

### Incision Care

The incision site is covered with a foam protective dressing and should be kept dry and intact.\textsuperscript{4} In efforts to prevent wound complications, surgeons place as much muscle as possible over the soft tissues covering the device, aiming to minimize skin sloughing.\textsuperscript{4} Dressings are changed according to the orthopedic surgeon’s plan. The dressing should be inspected each shift by a nurse, and the surgical team should be notified of nonintact dressings or drainage. When dressing changes are performed, the incision should be inspected for any signs of infection. In addition to inspection for drainage, dressings should be assessed for contamination by urine or feces; many patients who have a VEPTR procedure are incontinent because of their age or their disease. Proper postoperative positioning works to promote wound healing. For patients who cannot reposition themselves, their positions should be changed at least every 2 hours. Patients should avoid lying on the incision site because of the risk for skin breakdown or dehiscence. Complications can extend ICU and overall hospital stays.

### Potential Complications

Implantation of a VEPTR device and serial expansions are not without risk. Some of the complications are related to the repetitive nature of these expansions, because the original incision is the portal of entry.\textsuperscript{5} Potential complications are listed in Table 2. Complications that have occurred include wound dehiscence,\textsuperscript{17,24} skin sloughing,\textsuperscript{9,17,22,25} infection of the device or surgical site,\textsuperscript{9,14,15,17-19,21,22,24,26-30} migration of the device,\textsuperscript{12,15-18,21,22,26-28} breakage of the implant,\textsuperscript{9,10,12,13,26} rib fractures,\textsuperscript{10,18,22,25,27} and neurologic injuries such as acute brachial plexus palsy\textsuperscript{17,18,22,25} or spinal cord injury.\textsuperscript{17} A few patients in some studies had respiratory complications, including
pneumothorax intraoperatively,10,12,22 pneumonia,12,18 acute respiratory distress syndrome,17,18 respiratory distress intraoperatively,31 acute respiratory insufficiency immediately postoperatively,22 and chronic respiratory insufficiency due to chest wall stiffness after implantation.22

Emans et al15 reported 12 complications in 31 patients, for a rate of 39%. The complications included aspiration pneumonia leading to acute respiratory distress syndrome, rib fracture, surgical site infection, acute brachial plexus palsy, and refusion of the thoracostomy. The 23 patients in the study by Hasler et al9 had 23 complications; a 22% risk of complication per insertion procedure and a 12% risk per expansion procedure. White et al13 reported complications in 6 of the 14 patients with spine-spine VEPTR implants, a rate of 43%. A retrospective review29 of complications in growing spinal implants revealed an elevated complication rate (2.37 complications per patient) in children with VEPTR compared with the rate in children with hybrid devices (0.86 complications per patient) or dual growing rods (2.06 complications per patient). Campbell et al17 reported 52 complications in 22 patients. The most common major complications were migration of the VEPTR rib device, skin sloughing, and associated infections. In children with infantile idiopathic scoliosis who had VEPTR implantation, 3 of 9 patients (33%) had complications, including erosion of rib at the VEPTR attachment site in 2 patients and 2 weeks of lower extremity pain postoperatively that resolved without intervention.16

Despite the seemingly high rates of complications, the rates are not weighted and many of the studies had small heterogeneous groups of patients, so an accurate reflection of the actual risks of treatment is difficult. However, in a study by Campbell et al,17 the sample of 27 patients was relatively homogeneous, with fused ribs and congenital scoliosis. Campbell et al reported 52 complications in 22 patients. An infection occurred in 11% of the patients, a relatively low infection rate. This rate may be more representative of the actual risk of VEPTR implantation. Although the data are limited, prognoses are poor for patients with the rare diseases that may be treated with VEPTR placement. Akbarinia et al9 reported a 50% mortality rate due to severe restrictive lung disease in patients with Jeune syndrome, and Betz et al13 reported that the syndrome is almost universally fatal within the first year of life. In the study by Betz et al, the mortality rate after VEPTR implantation was 50%, but the patients who survived were 5 years 6 months to 8 years 9 months old at the time the article was published in 2008, a vast improvement compared with the natural death rate. In patients with spondylothoracic dysplasia, early death has been reported.32 Betz et al3 cited research that indicated nearly a 100% mortality rate in children with Jarcho-Levin syndrome; however, Betz et al had a 0% mortality rate after VEPTR implantation in their 24 patients with this syndrome. Although patients undergoing VEPTR insertion and expansions may have significant complications after the procedure, their chances of survival increase dramatically with the use of the VEPTR device.

As stated earlier, the repetitive nature of surgical expansions contributes to wound complications. Additionally, skin sloughing and wound dehiscence are associated with low body weight, a common preoperative problem in TIS patients. Even with aggressive nutritional interventions, including enteral tube feedings via a nasogastric tube or a gastrostomy tube, patients in need of a VEPTR may undergo the implantation of the device while underweight. Surgical site infections are another complication that can cause increases in length of hospital stay, the need for antibiotic therapy, and further
Follow-up of patients as they reach adulthood to monitor long-term effects of VEPTR implantation is an important area of research in which data are limited.

Revision procedures were associated with significantly higher rates of infection (10.3%) than were insertion (8.4%) and lengthening procedures (3.3%). These rates were also higher than rates for spinal fusion insertion (5.5%) and revision (6.3%). Mackenzie et al also examined the pathogens associated with the surgical site infections in these patients. In addition to gram-positive pathogens, 46.5% of the surgical site infections contained one or more gram-negative organisms. A total of 32 of 33 infections (97%) were detected in patients with nonidiopathic scoliosis, and many of the organisms were enteric pathogens, possibly related to contamination of the wound by urine or feces. Hence, perioperative prophylaxis for infections caused by gram-negative organisms should be considered in addition to the commonly provided drugs for gram-positive organisms in these patients.

**Long-term Follow-up**

Expansion surgeries, done via reopening the skin through the original incision and lengthening the VEPTR rod through distraction (moving the inferior and superior ends of the VEPTR device in opposite directions, thereby making the device longer), are generally done every 4 to 6 months until skeletal maturity is reached. Although this course is the one proposed, periods between lengthenings may be greater, as when illnesses and problems arranging travel to the institution produce delays in the expansion surgeries.

Respiratory insufficiency requires additional energy to support breathing and is associated with poor nutritional status. Theoretically, treatment of TIS could improve the ability of a child to grow and gain weight, because some of the energy originally required to breathe could instead be directed toward growth. In a study by Skaggs et al, patients had significant improvement in nutritional status after placement of a VEPTR, as indicated by weight percentile gains.

Multiple techniques have been used to assess improvement in lung function of patients with TIS after VEPTR implantation. The Cobb angle is a measure of the degree of spinal curvature in scoliosis, and a goal in many of the VEPTR studies is to determine if an improvement in the Cobb studies is to determine if an improvement in the Cobb angle correlates with improved lung function. Measurement of lung volume via computed tomography before and after surgery and comparisons with normal lung volume has also been studied to determine if placement of a VEPTR improves pulmonary function.

In a study by Gadepalli et al, the data did not suggest improvement in pulmonary status according to lung volume measured via computed tomography or lung function measured by using PFTs. Caubet et al sought to determine if hemoglobin levels could be used as a marker of pulmonary function. Elevated hemoglobin levels are associated with chronic hypoxia, and in the study of Caubet et al, patients had normalization of previously elevated hemoglobin levels 6 to 24 months after implantation of a VEPTR or a growing rod. Caubet et al suggest that hemoglobin can be used to evaluate the outcomes of these surgical procedures. Standard PFTs in patients old enough and developmentally able to participate in the tests (generally older than 6 or 7 years) are used to determine pulmonary function. Additionally, specialized PFTs have been used in efforts to measure lung function. Other methods may include measurement of arterial or capillary blood gases.
sedation. The results indicated a benefit over time, with the prosthesis allowing for lung growth along with body growth, comparable to that in healthy infants and young children. Long-term follow-up of patients treated with VEPtr devices and growing rods are important in examining improvements in the patients’ TIS, pulmonary function, and, ultimately, long-term survival, which is currently unknown.

**Quality of Life and Future Research**

Only a few articles on the impact of VEPtr on quality of life have been published. In a 2008 study by Vitale et al, the parents of 45 patients who were undergoing VEPtr implantation completed a questionnaire (the Child Health Questionnaire) designed to determine the functional status and psychological well-being of the children and caregiver burden of the parents. The results indicated no significant short-term improvement in quality of life, but Vitale et al postulated that the treatment may maintain the quality of life and the burden on caregivers that might otherwise worsen if the TIS progressed without treatment. The investigators noted that the preoperative scores of TIS patients are among the lowest in pediatric patients, lower than those of children with asthma, epilepsy, heart disease, and childhood cancer. Patients with TIS are extremely ill, and the goal of VEPtr implantation is to improve their quality of life by stabilizing the thorax to maximize pulmonary development. Families of children who require mechanical ventilation or noninvasive ventilation 24 hours a day can benefit from even moderate improvements in pulmonary function after VEPtr placement that might allow the child to tolerate time during the day without ventilatory support.

In the study by Gadeppali et al, a modified Scoliosis Research Society questionnaire was used to assess some aspects of quality of life. Although scores after placement of a VEPtr were positive, they did not differ significantly from preoperative scores. Despite the lack of statistical significance, parental impression of self-image and self-confidence was better postoperatively than it had been preoperatively. The questionnaire was modified from the original, and the modified tool may be insensitive to the issues of TIS because it is intended for use in patients with scoliosis.

Neither the Child Health Questionnaire nor the Scoliosis Research Society instrument is specifically for children with respiratory insufficiency, a characteristic that might limit the usefulness of the tools in patients with TIS who may undergo VEPtr insertion. The paucity of studies on quality of life in children with TIS (with or without surgical intervention such as VEPtr implantation) warrants further research; currently no quality-of-life questionnaire is available for TIS specifically. Other areas for research include determining better measures of improvement of lung function after VEPtr implantation in patients with TIS and further examining quality-of-life issues in these patients.

**Implications for Critical Care Nurses**

Critical care nurses who work in pediatric ICUs need to be knowledgeable about the VEPtr procedure, the necessary postoperative monitoring, and potential complications. Nurses play an important role in supporting patients and the patients’ families during the immediate postoperative period and as patients begin recovering, progressing to the point of transfer to a general unit or discharge from the hospital. Patients with TIS often have extremely rare diseases and will travel to tertiary treatment centers far from their homes for VEPtr placement and management. Being away from their normal support systems both frequently and for potentially longer periods if complications occur can cause stress on the child and the child’s family, adding complexity to the care of these children and their families. Nurses are also ideally positioned to provide education to patients’ families about the expected course in the ICU and the care families will need to provide as the child recovers. One particular area is skin care. The orthopedic surgeon will determine the specific incisional care plan, but the bedside nurse will be demonstrating this care and teaching it to patients’ families.

**Implications for Advanced Practice Nurses**

Collaboration is an important aspect of the role of an advanced practice nurse in the immediate postoperative period to manage the initial recovery after VEPtr implantation as well as the recovery after VEPtr expansions. Because of the repetitive expansions of a VEPtr device, patients and their families most likely...
will have multiple ICU admissions. Advanced practice nurses can develop relationships with patients and patients’ families during this time and provide all of them with further support. Advanced practice nurses can also collaborate with the multidisciplinary team, including personnel from the nutritional, pulmonary, and orthopedic surgery services preoperatively. VEPTR candidates require optimized preoperative nutrition. Adequate nutrition is especially important for wound healing and is also necessary for continued growth between expansion surgeries. Advanced practice nurses are instrumental in coordinating discharge planning and patient-family education for patients who have undergone VEPTR implantation. Planning for discharge may vary with each patient, according to the severity of the patient’s TIS, whether complications occur, and the required level of support and necessary care coordination.

**Conclusion**

Children with TIS and scoliosis have unique needs, and children who undergo VEPTR treatment require specialized care. Published information on the postoperative nursing care for VEPTR patients is limited. Critical care and advanced practice nurses working in the pediatric ICU may encounter these patients in the postoperative period after initial implantation of the VEPTR device and after subsequent surgeries. Knowledge of the postoperative care, potential complications, and long-term follow-up allow advanced practice nurses to safely manage care for these patients and critical care nurses to deliver this care. CCN

**Financial Disclosures**

None reported.

**Letters**

Now that you’ve read the article, create or contribute to an online discussion about this topic using eLetters. Just visit www.ccnonline.org and select the article you want to comment on. In the full-text or PDF view of the article, click “Responses” in the middle column and then “Submit a response.”

**dotmore**

To learn more about pediatric care in the critical care setting, read “Increasing Parental Participation During Rounds in a Pediatric Cardiac Intensive Care Unit” by Blankenship et al in the American Journal of Critical Care, November 2015;24:532-538. Available at www.ajcconline.org.

**References**

Minutes can seem like a lifetime when a nurse encounters a patient having a seizure. A seizure that continues into status epilepticus changes from a stressful situation into a life-threatening emergency. Guidelines for the evaluation and management of status epilepticus published by the Neurocritical Care Society in 2012 are used here to provide definitions and describe the pathophysiology, diagnostic evaluation, treatment options, and nursing considerations when caring for a patient in status epilepticus. With this information, nurses will be able to systematically provide treatment to support patients during this emergency.

**Incidence and Definition**

The incidence of status epilepticus varies by region and age. Incidence ranges from 9.9 to 15.8 per 100 000 persons in Europe to 18.3 to 41 per 100 000 persons in the United States. The distribution is bimodal, with higher incidence in the first decade of life and then dramatically escalating incidence following the fifth decade. Risk of death from status epilepticus ranges from 1.9% to 40%, with the wide variation dependent on age, cause, and duration.
Definition of status epilepticus has varied throughout the decades depending on the duration of seizure. Status epilepticus is currently defined as “5 minutes or more of continuous clinical and/or electrographic seizure activity or recurrent seizure activity without recovery (returning to baseline) between seizures.” The definition of status epilepticus has evolved through the 20th century from “the maximum expression of epilepsy” in 1904, to more than 30 minutes of continuous seizure activity from the 1960s to the early 2000s. The current definition’s more inclusive criteria reflect the need for urgent treatment. Although status epilepticus is defined as 5 minutes or more of clinical or electrographic seizure, refractory status epilepticus is defined as clinical or electrographic seizures after adequate dosing of an initial benzodiazepine and a secondary antiepileptic drug (AED). Others have used the term “super-refractory status epilepticus,” to describe continued status epilepticus 24 hours after administering anesthetic therapy or recurring status epilepticus after reduction in anesthetic dose. Convulsive status epilepticus involves jerking rhythmic movements of the arms and/or legs. Non-convulsive status epilepticus (NCSE) is electrographic, ongoing seizure activity without the motor findings of convulsive status. Table 1 lists signs of NCSE. Regardless of the exact definition, prompt recognition, diagnosis, and emergent treatment are key to minimizing neurologic sequelae. Recognition begins with a basic understanding of the pathophysiology that underlies status epilepticus.

Pathophysiology

Normal neuronal activity occurs through a balance of complex cellular-level mechanisms. The resting membrane potential is maintained across the cell wall of a neuron by the sodium-potassium pump. This mechanism pumps sodium and chloride to high concentrations into the interstitial space and potassium to high concentrations within the cell. When a sufficient stimulus is applied, an action potential is realized and the cell depolarizes. Neurotransmitters excite or inhibit other neurons. Normal brain function is a result of balance between inhibition and excitation. Glutamate is the most common excitatory neurotransmitter and γ-aminobutyric acid (GABA) is the most common inhibitory transmitter. During status epilepticus, failure of the inhibitory GABAergic mechanisms can cause the seizures to become self-sustaining and pharmaco-resistant. Benzodiazepine resistance may develop after 10 to 45 minutes of status epilepticus, possibly because of changes in synaptic physiology that reduce the number of GABA_A receptors. GABA_A-enhancing drugs such as benzodiazepines, barbiturates, propofol, and some anesthetics are effective at stopping seizures by overwhelming the excitation with inhibitory stimulation, but withdrawal of these inhibitory medications may provoke rebound seizures.

Table 1 Possible signs of status epilepticus

<table>
<thead>
<tr>
<th>Nonconvulsive</th>
<th>Convulsive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aggression</td>
<td>Convulsive syncope</td>
</tr>
<tr>
<td>Automatisms</td>
<td>Repetitive decerebrate or decorticate posturing</td>
</tr>
<tr>
<td>Blinking</td>
<td>Involuntary movement disorders</td>
</tr>
<tr>
<td>Crying</td>
<td>Psychogenic nonepileptic spells (pseudo-seizures)</td>
</tr>
<tr>
<td>Delirium</td>
<td></td>
</tr>
<tr>
<td>Delusions</td>
<td></td>
</tr>
<tr>
<td>Echolalia</td>
<td></td>
</tr>
<tr>
<td>Facial twitching</td>
<td></td>
</tr>
<tr>
<td>Laughter</td>
<td></td>
</tr>
<tr>
<td>Nausea/vomiting</td>
<td></td>
</tr>
<tr>
<td>Eye deviation contralateral to lesion</td>
<td></td>
</tr>
<tr>
<td>Perseveration</td>
<td></td>
</tr>
<tr>
<td>Psychosis</td>
<td></td>
</tr>
<tr>
<td>Tremulousness</td>
<td></td>
</tr>
<tr>
<td>Aphasia</td>
<td></td>
</tr>
<tr>
<td>Amnesia</td>
<td></td>
</tr>
<tr>
<td>Catatonia</td>
<td></td>
</tr>
<tr>
<td>Staring</td>
<td></td>
</tr>
</tbody>
</table>

*a Based on information from Bazil and Pedley.*

Authors

Thomas Lawson is an acute care nurse practitioner in the neuroscience critical care unit at Ohio State University Wexner Medical Center, Columbus, Ohio.

Susan Yeager is the lead acute care nurse practitioner in the neuroscience critical care unit at Ohio State University Wexner Medical Center and a clinical instructor at The Ohio State University College of Nursing, Columbus, Ohio.

Corresponding author: Thomas Lawson, BSN, MS, ACNP-BC, Neuroscience Critical Care, The Ohio State University Medical Center Wexner Medical Center, 395 W 12th Ave, 7th Floor, Columbus OH, 43210 (e-mail: thomas.lawson@osumc.edu).

To purchase electronic or print reprints, contact the American Association of Critical-Care Nurses, 101 Columbia, Aliso Viejo, CA 92656. Phone, (800) 899-1712 or (949) 362-2050 (ext 532); fax, (949) 362-2049; e-mail, reprints@aacn.org.
as the inhibitory/excitatory balance is tipped toward excitation.\textsuperscript{10} Seizure activity, whether nonconvulsive or convulsive, causes intense metabolic demands that require increased cerebral blood flow. This workload increases during the first seconds of a seizure.\textsuperscript{12} Even during the first few minutes of a seizure, the normally tight coupling of neuronal activity, energy demand, and cerebral blood flow is decoupled because of the very high energy demands of seizing neurons in the context of limited circulatory delivery of energy to cells that store essentially no energy.\textsuperscript{12,13}

When this increased cellular metabolic demand is not met, neuronal damage and cell death result.\textsuperscript{14,15} Unmet neuronal metabolic demand is not the only factor responsible for cell death. The massive depolarization of the neuronal action potential during seizures leads to excessive release of glutamate, which activates postsynaptic N-methyl-D-aspartate receptors, triggering an influx of calcium into the cell.\textsuperscript{16,17} Maintaining neuronal homeostasis requires large quantities of energy, but neurons do not generally use stored energy and are particularly susceptible to energetic stress.\textsuperscript{13,16} Thus, during an event of status epilepticus, cellular homeostasis fails and high concentrations of intracellular calcium enter the mitochondria, which then lose their membrane potential, and mitochondrial respiration fails with a cascade ending in cell necrosis.\textsuperscript{16,17} The pathophysiologic changes and resulting neuronal injury resulting from status epilepticus demand urgent, aggressive diagnosis and treatment.

**Risk Factors for Seizure**

Various factors can put a patient at higher risk of transitioning from seizure to status epilepticus. Specific factors can have a greater impact within different age groups, but seizure history alone increases a patient’s risk regardless of age. Following a single unprovoked seizure, risk of further seizure is 40% to 52%, and after 2 seizures, the risk is 73%.\textsuperscript{18} Age-related risk factors for status epilepticus are listed in Table 2. Additional causes that decrease seizure threshold can be broken down into structural, infectious, metabolic, and drug-related categories. Examples of structural factors include neoplasms, stroke, or traumatic brain injury. Infection, virus, fever, and metabolic derangements such as hyponatremia, hypoglycemia, hyperglycemia, hepatic encephalopathy, hypocalcemia, and hypomagnesemia also create environments that increase a patient’s risk of status epilepticus.

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Risk factors for status epilepticus by age\textsuperscript{a}</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age, y</strong></td>
<td><strong>Contributing factors</strong></td>
</tr>
<tr>
<td>(&lt;35)</td>
<td>Brain trauma, alcohol withdrawal, brain tumors, and illicit drug use</td>
</tr>
<tr>
<td>(&gt;35)</td>
<td>Cerebrovascular disease, brain tumors, alcohol withdrawal, metabolic disorders such as uremia and electrolyte imbalances, neurodegenerative diseases, and idiopathic causes</td>
</tr>
</tbody>
</table>

\textsuperscript{a} Based on information from Bazil and Pedley.\textsuperscript{4}

**Table 3** | Drugs that can affect seizure threshold\textsuperscript{d} |
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Psychiatric medications</strong></td>
<td>Tricyclic antidepressants, Bupropion, Venlafaxine, Citalopram, Quetiapine</td>
</tr>
<tr>
<td><strong>Recreational drugs</strong></td>
<td>Cocaine, MDMA (ecstasy)</td>
</tr>
<tr>
<td><strong>Antibiotics</strong></td>
<td>β-Lactam antibiotics, Carbapenems, Isoniazid</td>
</tr>
<tr>
<td><strong>Other medications</strong></td>
<td>Carbamazepine/oxcarbazepine, Diphenhydramine, Flumazenil (in benzodiazepine-dependent patients)</td>
</tr>
</tbody>
</table>

\textsuperscript{d} Based on information from Thundiyil et al.\textsuperscript{29}

Toxic effects of drugs as well as some antiepileptic agents can also contribute to seizures (Table 3).\textsuperscript{29} Therefore, when eliciting a patient’s history, a clinician should have a heightened clinical suspicion of status epilepticus if any of these factors is present.

**Patient’s History**

The initial encounter after stabilization should include an interview seeking details of the period surrounding the seizure. This interview should occur with the patient if coherent and with the bedside staff and a knowledgeable family member. Questions should clarify the patient’s seizure history. Of particular importance in regard to a seizure history is information regarding the patient’s AED regimen and adherence, and description of the event including duration, quantity, and character of the convulsions as well as any prodromal features (also known as an aura). Knowledge of what medications have controlled seizures before and what medications have failed may influence the selection of AEDs. Probing for social issues affecting medication adherence may reveal the
necessity of choosing less expensive medications or counseling the patient or family on simple techniques for organizing medications for regular administration.

Assessment of Patients

Initial assessment should begin with a rapid evaluation of airway, breathing, and circulation. Examination of patients with suspected seizures should include a thorough yet rapid visual assessment of motor activity of the face and extremities. The tonic-clonic extremity movements are obvious and are easily identified by lay people. Patients with generalized NCSE may be critically ill and lethargic with or without subtle rhythmic movements. Subtle twitches may occur on any part of the body, may be unilateral or bilateral, and often involve the extraocular muscles or eyelids. NCSE patients with partial seizures may be less impaired: acting confused, aphasis, or staring, and they may show a lateral gaze preference contralateral to the focus of the seizure.¹

Electroencephalography

The bedside nurse caring for a patient in status epilepticus should have a basic knowledge of electroencephalographic (EEG) patterns in order to determine when to call the neurologist to request an official reading. Although it is clearly not the responsibility of the nurse to interpret an EEG, the nurse’s having a basic familiarity with typical EEG patterns and glancing at the bedside EEG monitor while caring for the patient may prompt additional attention by the neurologist and result in earlier identification of seizures. Figures 1 through 3 are examples of typical normal and abnormal EEG findings that may be encountered. A normal EEG shows lower amplitude and irregular frequencies (Figure 1). In NCSE, the amplitude is higher and the pattern is more regular with some similarities to the appearance of ventricular tachycardia on an electrocardiogram (Figure 2). During administration of anesthetic medications such as phenobarbital or propofol, a burst-suppressed pattern is found (Figure 3). Determining the desired frequency of the bursts (the sample EEG in Figure 3 shows 1 burst per page) is useful for anesthetic titration and communication with the prescriber.

Myoclonic muscle artifact reveals a disconcerting EEG pattern, and expert video EEG interpretation is required to discern between myoclonic artifact and
Figure 2  Electroencephalogram of a patient with nonconvulsive status epilepticus.

Figure 3  Electroencephalogram of patient during administration of anesthetic shows burst suppression.
status epilepticus. Use of a single dose of a paralytic agent to eliminate muscle artifact may be required. Timing of EEG application will be different from institution to institution and is likely to vary depending on the resources available from the EEG staff. If radiologic evaluation is also desired, special consideration should be given to the priority of these tests. Determination of the sequence of these diagnostic tests rests on both the clinical condition of the patient and the presumed underlying cause of the seizure. For example, for a patient with known brain cancer who had a witnessed onset of generalized convulsive status epilepticus and stopped convulsing after being given loading doses of lorazepam and fosphenytoin but has not returned to his baseline neurologic status, EEG most likely should be done before imaging as the patient is at high risk for NCSE. Another example is a patient with head trauma who had a seizure following an acute head injury but does not appear to have clinical seizure; in that case, a computed tomography scan of the head should be done to rule out bleeding that should be treated surgically.

In addition to electrographic monitoring, blood samples for laboratory tests should be collected soon after the patient arrives. All patients in status epilepticus should have blood glucose level measured, a complete blood cell count, and a comprehensive metabolic panel including measurement of calcium and magnesium levels. Patients being treated for epilepsy should have AED levels measured if applicable. Depending on the patient’s history and clinical findings, additional laboratory tests including a comprehensive toxicology panel, liver function tests, measurement of troponin levels, collection of blood samples to type and hold, arterial blood gas analysis, and lumbar puncture with measurement of protein and glucose levels, cell count, and culture should be considered.

Except in cases with a clear cause, a radiologic workup to identify cranial abnormalities that may be causing an ictal focus should be undertaken. For example, a computed tomography scan may be obtained to rule out a traumatic hemorrhage and hemorrhagic stroke or magnetic resonance images may be obtained to rule out tumors or acute ischemic stroke.

Management of Patients

Seizures are a medical emergency; if they progress to status epilepticus and are not rapidly treated, permanent neuronal damage may occur within 24 hours. Both clinical and electrographic seizures should be emergently stopped. A prompt diagnosis should be followed by implementation of several therapies concurrently: critical care evaluation, emergency administration of an AED, and a search for the cause of the seizure. Initial management of status epilepticus is summarized in Table 4.

### Table 4 Initial management of status epilepticus

<table>
<thead>
<tr>
<th>Priorities for the first 0-5 min</th>
<th>Priorities for the first 15 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evaluation/treatment of airway with noninvasive management</td>
<td>Intubation if airway or gas exchange is compromised</td>
</tr>
<tr>
<td>Check initial vital signs and telemetry</td>
<td>Second-line intravenous antiepileptic drug</td>
</tr>
<tr>
<td>Obtain intravenous access</td>
<td>Vasopressor support if hypotensive</td>
</tr>
<tr>
<td>Neurological examination</td>
<td>Collect blood samples for laboratory tests (complete blood cell count, Chem 7, calcium, magnesium, antiepileptic drug levels)</td>
</tr>
<tr>
<td>Finger-stick blood glucose</td>
<td>Electrocardiogram</td>
</tr>
<tr>
<td>Administration of dextrose if hypoglycemic</td>
<td></td>
</tr>
<tr>
<td>Administration of benzodiazepine</td>
<td></td>
</tr>
<tr>
<td>(first-line antiepileptic drug)</td>
<td></td>
</tr>
<tr>
<td>Fluid resuscitation if hypotensive</td>
<td></td>
</tr>
<tr>
<td><strong>Priorities for the first 15-60 min</strong></td>
<td><strong>Within first 12-24 h</strong></td>
</tr>
<tr>
<td>Third-line antiepileptic drug for refractory status epilepticus</td>
<td>Magnetic resonance of the brain</td>
</tr>
<tr>
<td>Electroencephalography</td>
<td></td>
</tr>
<tr>
<td>Computed tomography of the head</td>
<td></td>
</tr>
<tr>
<td>Lumbar puncture</td>
<td></td>
</tr>
<tr>
<td>Additional laboratory tests (liver function tests, troponin levels, coagulation tests, arterial blood gases, comprehensive toxicology panel)</td>
<td></td>
</tr>
</tbody>
</table>

Adapted from the Neurocritical Care Society’s guidelines.

### Airway, Breathing, and Circulation

As with all patient emergencies, all management begins with evaluation of airway, breathing, and circulation. Generalized seizure activity impairs mental status and the ability to protect the airway, so the airway should be assessed concurrently with initial AED administration and should be serially reevaluated as therapy is escalated. Initial airway management may involve just provision of airway protection and supplemental oxygen delivery. Initial airway protection maneuvers consist of placing a
Intravenous lorazepam is preferred; an initial dose of 4 mg of lorazepam is recommended for patients in status epilepticus.

Because of this, nurses caring for patients with status epilepticus should clarify with the prescriber how medication and ventilator weaning should be handled to avoid inadvertently causing a patient to go back into status epilepticus.

Most AEDs can induce hypotension, so frequent monitoring of blood pressure is required. Peripheral intravenous catheters should emergently be placed for administration of intravenous AEDs, fluid boluses for hypotension, and other emergency drugs if required. Insertion of a central venous catheter should be considered if status epilepticus is refractory and requires anesthetic EEG burst suppression or if blood pressure is low and vasopressor support is required.

**Antiepileptic Drugs**

The goal of treatment is to rapidly stop status epilepticus and prevent recurrence; thus, the most appropriate route of administration for AEDs is intravenous. Current guidelines for status epilepticus and the emergency neurologic life support protocol recommend parenteral benzodiazepines for emergent initial therapy.

Intravenous lorazepam is preferred, but if intravenous access is not available, other benzodiazepines may be administered via alternative routes. Lorazepam has logistical issues in the prehospital setting (refrigeration requirements and shorter shelf-life), but intramuscular midazolam is at least as effective and is a safe alternative to intravenous lorazepam. An initial dose of 4 mg of lorazepam is recommended. Immediately following benzodiazepine administration, regardless of whether or not the patient has stopped convulsing, another AED should be administered to prevent rebound seizures as the benzodiazepine wears off. Therapeutic levels of a maintenance AED should be obtained urgently. Dosing of AEDs and special considerations are summarized in Table 5.

Several choices of medication are available for second-line AEDs. The most popular is fosphenytoin/phenytoin. Phenytoin is administered at a dose of 20 mg/kg at a rate not to exceed 50 mg/min. Fosphenytoin is a prodrug of phenytoin that has no pharmacological activity before its in vivo conversion to phenytoin. The half-life conversion to phenytoin is 8 to 15 minutes, and once converted, the phenytoin has 100% bioavailability. Fosphenytoin is dosed at 20-mg phenytoin equivalents per kilogram but can be administered at 150-mg phenytoin equivalents per minute, and because it is safe to administer 3 times faster and poses less risk of severe injury if extravasation occurs, fosphenytoin is typically preferred over phenytoin. Phenytoin and fosphenytoin may precipitate bradycardias and hypotension when infused rapidly, so caution should be exercised, particularly when administering a loading dose. Intravenous access, cardiac telemetry, and frequent monitoring of blood pressure are essential. Alternatives to fosphenytoin include valproate sodium 20 to 40 mg/kg intravenously, phenobarbital 20 mg/kg intravenously, levetiracetam 1 to 3 g intravenously, or continuous intravenous infusion of midazolam. Others have suggested lacosamide at doses of 200 to 400 mg intravenously as an alternative second-line agent with the caveat that no
<table>
<thead>
<tr>
<th>Drug/category/mechanism</th>
<th>Dosing</th>
<th>Administration rate</th>
<th>Serious adverse effects</th>
<th>Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intermittently dosed medications</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diazepam</td>
<td>0.15 mg/kg intravenously up to 10 mg, repeat in 5 min, 0.2 mg/kg rectally up to maximum of 30 mg, Alternate 0.2 mg/kg rectally</td>
<td>Up to 5 mg/min</td>
<td>Hypotension, respiratory depression</td>
<td>Short duration, active metabolite</td>
</tr>
<tr>
<td>Lorazepam</td>
<td>0.1 mg/kg intravenously up to 4 mg, repeat in 5-10 min</td>
<td>Up to 2 mg/min</td>
<td>Hypotension, respiratory depression</td>
<td>Short duration, active metabolite, renal elimination</td>
</tr>
<tr>
<td>Midazolam</td>
<td>0.2 mg/kg intramuscularly up to 10 mg</td>
<td></td>
<td>Hypotension, respiratory depression</td>
<td></td>
</tr>
<tr>
<td>Fosphenytoin</td>
<td>20-mg phenytoin equivalent/kg intravenously, may rebolus with 5-mg phenytoin equivalent/kg 10 min after initial loading dose</td>
<td>Up to 150-mg phenytoin equivalent/min</td>
<td>Hypotension, arrhythmias</td>
<td>Cardiac monitoring for loading dose</td>
</tr>
<tr>
<td>Lacosamide</td>
<td>200-400 mg intravenously</td>
<td>200 mg per 15 min</td>
<td>PR prolongation, hypotension</td>
<td>Limited experience in status epilepticus</td>
</tr>
<tr>
<td>Levetiracetam</td>
<td>1000-3000 mg intravenously</td>
<td>2-5 mg/kg per min</td>
<td>Hypotension</td>
<td></td>
</tr>
<tr>
<td>Phenobarbital</td>
<td>20 mg/kg, may give additional 5-10 mg/kg 10 min after initial loading dose</td>
<td>50-100 mg/min intravenously</td>
<td>Hypotension, respiratory depression</td>
<td></td>
</tr>
<tr>
<td>Phenytoin</td>
<td>20 mg/kg, may give additional 5-10 mg/kg 10 min after initial loading dose</td>
<td>Up to 50 mg/min intravenously</td>
<td>Arrythmias, hypotension, purple glove syndrome with extravasation (rare)</td>
<td>Cardiac monitoring during loading dose</td>
</tr>
<tr>
<td>Topiramate</td>
<td>200-400 mg via nasogastric tube or by mouth, 300-1600 mg/d divided into 2-4 doses</td>
<td></td>
<td>Metabolic acidosis</td>
<td>No intravenous formulation available</td>
</tr>
<tr>
<td>Valproate sodium</td>
<td>20-40 mg/kg intravenously, may repeat</td>
<td>3-6 mg/kg per min</td>
<td>Hyperammonemia, pancreatitis, thrombocytopenia, hepatotoxicity</td>
<td>Caution in patients with traumatic brain injury Pregnancy category X</td>
</tr>
<tr>
<td><strong>Refractory/continuous medications</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Midazolam Water-soluble benzodiazepine GABA&lt;sub&gt;B&lt;/sub&gt; enhancement</td>
<td>0.05-2 mg/kg per h, bolus 0.1-0.2 mg/kg and increase rate every 3-4 h</td>
<td></td>
<td>Respiratory depression, hypotension</td>
<td>Requires mechanical ventilation Tachyphylaxis may occur Short duration&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Pentobarbital Barbiturate GABA&lt;sub&gt;B&lt;/sub&gt; enhancement</td>
<td>5-15 mg/kg loading dose followed by infusion at 0.5-5 mg/kg per h</td>
<td></td>
<td>Hypotension, respiratory depression, cardiac depression, paralytic ileus, loss of neuronal function at high doses</td>
<td>Requires mechanical ventilation Half-life 15-60 h&lt;sup&gt;b&lt;/sup&gt; Lowered body temperature and theoretically is neuroprotective&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Propofol Hypnotic sedative Activation of GABA receptors, inhibition of NMDA receptors, and modulation of calcium influx</td>
<td>Start at 20 µg/kg per min, titrate up to 50 µg/kg per min</td>
<td></td>
<td>Hypotension, respiratory depression, cardiac failure, rhabdomyolysis, metabolic acidosis, propofol-related infusion syndrome&lt;sup&gt;d&lt;/sup&gt;&lt;sup&gt;e&lt;/sup&gt;</td>
<td>Requires mechanical ventilation</td>
</tr>
</tbody>
</table>

Abbreviations: GABA, γ-aminobutyric acid; NMDA, N-methyl-D-aspartate.

<sup>a</sup> Adapted from the Neurocritical Care Society’s guidelines.<sup>1</sup>
Phenytoin and fosphenytoin often cause hypotension during the administration of loading doses.

Phenytoin and fosphenytoin often cause hypotension during the administration of loading doses. These medications have considerable side effect profiles that require vigilance to avoid increased morbidity and mortality. Pentobarbital is known to cause immunosuppression and gastric paresis and is associated with elevated rates of ventilator-associated pneumonia, urinary tract infections, and ileus. Propofol has a rapid onset and short duration and is often considered a favorite first-line anesthetic for refractory status epilepticus because of its favorable properties of rapid onset and recovery and no serious drug-drug interactions. The most severe side effect is a rare condition known as propofol-related infusion syndrome (PRIS), typically associated with doses greater than 4 mg/kg per hour for longer than 48 hours, as is characteristic of the usage patterns in status epilepticus. Early findings include hyperlipidemia, acidosis, and elevated creatine kinase levels. Serial daily measurements of arterial blood gases, serum level of creatine kinase, and triglycerides can be used to screen for early signs of PRIS. If unrecognized and untreated, PRIS may result in rhabdomyolysis, renal failure, hypotension, metabolic acidosis, hyperthermia, cardiac dysfunction, and ultimately death.

Midazolam has a strong antiepileptic action and a short duration, and like the others, it is a respiratory and cardiac depressant. The primary disadvantage is tachyphylaxis, which is a rapidly decreasing therapeutic effect following initial administration, which may allow breakthrough seizures as soon as 1 day after therapy is started. The barbiturate pentobarbital is the traditional anesthetic medication used for patients in status epilepticus. Some advantages of pentobarbital are its strong antiepileptic action as well as its tendency to lower core temperature. Mild hypothermia has a neuroprotective effect in status epilepticus. Pentobarbital causes more potent hypotension and cardiorespiratory depression, and pressors are typically required. Gastric paresis, immunosuppression, pneumonia, and splanchnic hypoperfusion leading to gastric, pancreatic, and hepatic complications are more common with barbiturates. The pharmacokinetics of pentobarbital will typically cause a persistent anesthetic effect for several days following discontinuation. Duration of anesthesia before initial attempts to wean a patient off of any of these anesthetic agents is customarily 24 to 48 hours, but few data are available to support this. Maintenance AEDs should be continued during anesthesia and throughout weaning.

Data from randomized controlled trials are available to support this decision because the newer AEDs were not included in older trials. If the patient progresses to refractory status epilepticus, urgent escalation of therapy should be continued. At this point, options include administering another bolus of the same second-line agent that had been given or adding another agent from the ones just discussed. Limited evidence supports expanding the list of appropriate AEDs to include topiramate 200 to 400 mg via nasogastric tube or by mouth. If the status epilepticus remains refractory, induction of anesthesia with continuous intravenous medications is often required. Typical agents include propofol 20 to 200 µg/kg per minute, pentobarbital as a 5- to 15-mg bolus followed by 0.5 to 5 mg/kg per hour, and the aforementioned midazolam as a 0.2-mg/kg bolus followed by an infusion at 0.1 to 0.4 mg/kg per hour. Reevaluation after each of these steps must occur frequently, and subsequent escalation of treatment must be rapid and aggressive to avoid permanent neuronal damage. No data support use of one of these agents over the others.

The most notorious side effect of pentobarbital, midazolam, and propofol infusions is hemodynamic suppression, which warrants close monitoring of vital signs. Before giving a patient a loading dose of phenytoin or fosphenytoin, telemetry should be applied because the drug may precipitate bradyarrhythmias, including high-grade atrioventricular block, asystole, and death. Nurses should anticipate vasopressor use if an anesthetic medication is infusing continuously because anesthetics often cause hypotension. Vasopressor selection should be individualized to each patient’s hemodynamic profile and desired effect.

Although data indicate that intravenous anesthetic drugs such as propofol, midazolam, and high-dose barbiturates have a potent antiepileptic effect, these patients with super-refractory status epilepticus have high morbidity and mortality. In a recent study, researchers found higher rates of infection and death in patients receiving anesthetic agents, even when corrected for critical medical conditions and for the refractoriness of the status epilepticus. It remains unclear whether the anesthetic agents themselves or a selection bias led to this worsened mortality.
Although multiple anesthetic medications exist, no randomized controlled trials with adequate power have been done to compare agents. In a meta-analysis of 121 studies including 1168 patients, the researchers were able to draw only broad conclusions and did not recommend one agent as better than another (it may vary from patient to patient). The Neurocritical Care Society’s guidelines do not recommend anesthetic agent over another, citing limited evidence from less rigorous studies.

Dosing regimens must be adjusted on the basis of the patient and the EEG response. In refractory status epilepticus, the continuous EEG is the primary tool for determining anesthetic dose. Patients who are sedated to the point of burst suppression will often exhibit loss of all brainstem reflexes and appear to have palsy of cranial nerve III with dilated unresponsive pupils. Therefore the need for brain monitoring devices to continually assess patients at risk for herniation should be considered so that unnecessary imaging is not prompted by this examination, which would otherwise be very concerning.

**Nursing Considerations**

**Gastrointestinal**

The anesthetics, particularly barbiturates, can cause gastric paresis and ileus. Postpyloric nutrition in a patient with delayed gastric emptying and a suppressed cough reflex may be a logical approach. Additionally, the American Association of Critical-Care Nurses practice alert on prevention of aspiration recommends elevating the head of the bed to 30° to 45°. Should an ileus occur, a nasogastric tube may be required for gastric decompression to prevent aspiration. Tube feedings administered at a slow rate can promote the health of gastrointestinal membranes to prevent the translocation of organisms across the gastric cell membrane while also minimizing the potential of tube feeding intolerance.

**Dermatologic**

During prolonged EEG monitoring, skin beneath the leads may break down. The scalp should be assessed carefully. A lead holiday in which the EEG monitoring is suspended after a stable medication regimen is established but before weaning off of pentobarbital may be appropriate in some cases. Alternative types of skin preparation and conductive paste may be requested to minimize breakdown, particularly if the patient has a skin allergy to a specific product. After extravasation of phenytoin, purple glove syndrome may develop; this syndrome may be treated with dry heat and elevation, but the patient should be monitored for tissue sloughing or compartment syndrome. Another dermatologic situation that may affect the patient is development of a rash. Certain medications such as phenytoin, carbamazepine, and lamotrigine may cause Stevens-Johnson syndrome or toxic epidermal necrolysis. Although rare, if one of those disorders is ignored and the medication continues to be administered, either Stevens-Johnson syndrome or toxic epidermal necrolysis can evolve into a life-threatening situation.

**Neurologic**

Daily interruption of sedation, although once a popular strategy to reduce oversedation and prolonged mechanical ventilation, does not reduce ventilator days or ICU length of stay but in a Cochrane review, this practice was equivocal. The typical practice in patients with refractory status epilepticus is titrating anesthetics to burst suppression and maintaining this for 36 to 48 hours before weaning. It may be appropriate to perform abbreviated neurologic examinations during deep sedation or anesthesia. In situations where brain death is suspected, the brain death guidelines from the American Academy of Neurology suggest that sedative medications must be withheld for at least 5 half-lives, assuming normal hepatic and renal function, or until drug levels are undetectable before the brain death examination is performed.

Patients with status epilepticus should be kept normothermic. Those receiving continuous anesthetic medications have a tendency to be hypothermic, especially those receiving pentobarbital. Warm air blankets can be used to gently rewarm or maintain normothermia. Intravascular techniques are not often necessary.

**Summary and Conclusions**

Status epilepticus is a medical emergency that requires emergent identification and treatment and urgent follow-up therapies to prevent neuronal death. Although convulsive status epilepticus is obvious, nonconvulsive status epilepticus is less apparent. Whether convulsive or nonconvulsive in nature, emergent treatment for status epilepticus should begin with management of airway, breathing, and circulation followed by
benzodiazepines, urgent follow-up administration of intravenous AEDs, and rapid escalation of therapy, often to general anesthesia, to prevent morbidity and mortality. As with the evolving definition of status epilepticus over the years, ongoing rigorous research is needed to increase knowledge about the drug selection priorities and further development of our understanding of how the GABA-mediated inhibition fails in refractory status epilepticus.1,13 Knowing what to look for in this population of patients, nurses are uniquely positioned to assist with the identification and management of status epilepticus to minimize the untoward and intractable sequela of the natural course of the disease. CCN

Financial Disclosures
None reported.

eletters

Now that you've read the article, create or contribute to an online discussion about this topic using eletters. Just visit www.ccnonline.org and select the article you want to comment on. In the full-text or PDF view of the article, click “Responses” in the middle column and then “Submit a response.”

dotmore


References
29. Trinka E. What is the evidence to use new intravenous AEDs in status epilepticus? Epilepsia. 2011;52(suppl 1):35-38.
45. Shorvon S, Ferlini M. The outcome of therapies in refractory and


Certification Test Prep
We Do It for Our Patients

I was told by a nurse attending a CCRN/PCCN review course that he had no intention of sitting for the certification examination. His response when I asked why was, “I will not be paid any more, my hospital does not pay for the test, and frankly I just don’t see any reason to do it.” When asked why he had come to the review course, he answered “Oh, I like to learn, it was free, and I’m being paid to be here.” I asked the nurses attending the review who had raised their hands indicating their intent to take the examination, all of whom worked for the same health system, why they were going to sit for the examination. The enthusiastic response I got was split among 2 themes: “I’m doing it for myself” and “I’m doing it for my patients.” One of the hallmarks of a professional is selflessness and lifelong learning. Striving for and achieving certification falls into both of these categories.

Adult CCRN Practice Questions
1. Following admission for syncope, a patient reports feeling dizzy. The nurse obtains the following assessment data:
   Blood pressure (BP), 82/50 mm Hg

   ![Blood Pressure Monitor](image)

   **Contributors**

   **Carol Rauen,** RN-BC, MS, CCRN, PCCN, CEN, the department editor, is an independent clinical nurse specialist in The Outer Banks of North Carolina and a staff nurse in the burn trauma intensive care unit at Sentara Norfolk General Hospital in Virginia. She wrote CCRN question 5. Carol welcomes feedback from readers and practice questions from potential contributors at rauen.carol104@gmail.com.

   **Karen Jeffries,** MS, RN, CCRN-K, clinical education specialist at St David’s HealthCare, Austin, Texas, contributed CCRN questions 1 through 4.

   **Mary Beth Flynn Makic,** RN, PhD, CNS, CCNS, FAAN, FNAP, associate professor at the University of Colorado College of Nursing, Anschutz Medical Campus, Aurora, Colorado, wrote the PCCN questions and is teaching the PCCN review at the National Teaching Institute 2016 in New Orleans, Louisiana.

©2016 American Association of Critical-Care Nurses doi: http://dx.doi.org/10.4037/ccn2016791

In addition to calling for a rapid response, the first action of the nurse should be to

A. Prepare a dopamine infusion
B. Administer atropine
C. Administer epinephrine
D. Connect and start transcutaneous pacemaker (TCP)

Test plan topic: Cardiac, 18% of the CCRN questions

2. A patient with a traumatic brain injury (TBI) from a fall has a urine osmolality of 200 mOsm/kg and serum sodium level of 160 mEq/L. The nurse should anticipate an order for

A. 5% Dextrose in water intravenous infusion (IV)
B. 5% Dextrose in normal saline IV
C. A fluid restriction
D. Diuretics

Test plan topic: Endocrine, GI, Renal, Hematology and Integumentary, 20% of the CCRN questions

3. A postoperative gastrointestinal (GI) surgery patient has the following ventilator settings and arterial blood gas (ABG) results:
   Synchronized intermittent mechanical ventilation (SIMV)
   Tidal volume (TV), 500 mL
   Respiratory rate (RR), 12/min, with no spontaneous breaths
   Fraction of inspired oxygen (FiO₂), 60%
   Positive end-expiratory pressure (PEEP), 5 cm H₂O

   ![Ventilator Settings](image)
pH, 7.36
Paco\textsubscript{2}, 37 mm Hg
PaO\textsubscript{2}, 58 mm Hg
HCO\textsubscript{3}\textsuperscript{-}, 26 mmol/L

Which of the following ventilator changes would provide the greatest benefit to the patient?
A. Increase PEEP
B. Decrease TV
C. Decrease FiO\textsubscript{2}
D. Increase RR

Test plan topic: Pulmonary, 17% of the CCRN questions

4. A patient reports shortness of breath and has a blood pressure of 75/50 mm Hg. The following rhythm is on the monitor.

Which of the following would be the priority of care?
A. Vagal stimulation
B. Amiodarone 150 mg
C. Synchronized cardioversion at 100 joules
D. Defibrillation at 200 joules

Test plan topic: Cardiac, 20% of the CCRN questions

5. A patient who sustained blunt trauma from a fall down the stairs describes left shoulder pain and has no obvious shoulder injury. This symptom is most likely:
A. Kernig’s sign, which is indicative of a spinal cord injury
B. Virchow’s sign, which is indicative of clotting from disseminated intravascular coagulation (DIC)
C. Kehr’s sign, which is indicative of a ruptured spleen
D. Murphy’s sign, which is indicative of a ruptured diaphragm

Test plan topic: GI, Endocrine, Renal, Hematology, and Integumentary, 20% of the CCRN questions

Correct Answers and Rationales for Adult CCRN Practice Questions

1. Correct Answer: D

Rationale
The rhythm is a second-degree type II block. External pacing with a TCP can be used until transvenous pacing can be initiated. Dopamine (A) and epinephrine (C) might be considered, but would not be first. Atropine (B) is not recommended, as it may result in further conduction failure and a slower ventricular rate.

Sources

2. Correct Answer: A

Rationale
A low urine osmolality and high serum sodium level indicate intravascular fluid depletion. In this case, the cause is probably diabetes insipidus from the TBI. Dextrose in water will help to rehydrate the patient. Administration of 5% dextrose in normal saline (B) would promote osmotic diuresis, worsening the dehydration. Fluid restriction (C) and diuretics (D) also would increase dehydration.

Source

3. Correct Answer: A

Rationale
Increasing the PEEP will help to reopen closed alveoli to improve oxygenation. Decreasing the TV would decrease the PaO\textsubscript{2}. Increasing the RR (D) would decrease the carbon dioxide further. Decreasing the FiO\textsubscript{2} (C) would decrease the PaO\textsubscript{2} further.

Sources

4. Correct Answer: C

Rationale
Synchronized cardioversion is indicated for unstable supraventricular tachycardia (SVT). If the blood
pressure were stable, vagal maneuvers (A) could be used to slow conduction from the sinoatrial (SA) node to the atrioventricular (AV) node. Amiodarone (B) is used for stable wide QRS complexes and supraventricular arrhythmias. Defibrillation (D) would be used if the patient were pulseless and in ventricular tachycardia.

Sources

5. Correct Answer: C
Rationale
Kehr’s sign is a type of referred pain. The patient complains of left shoulder pain from diaphragm irritation, which could result from things such as rupture of the spleen or diaphragm. Kernig’s sign (A) is a subjective clinical sign of meningitis, and Murphy’s sign (D) is positive when the patient cannot take a deep breath (due to pain) when pressure is being applied over the liver. There is a Virchow’s triad but no Virchow’s sign (B).

Source

PCCN Practice Questions
1. A patient is confused and more withdrawn today. Which factor in the patient’s history is most likely a contributing risk factor for hypoactive delirium?
A. History of depression
B. History of cardiovascular disease
C. Unplanned hospitalization
D. Age greater than 65 years

Test plan topic: Neuro/Multisystem/Behavioral/Psychosocial, 15% of the PCCN questions

2. On postoperative day 3, a patient has onset of a high fever, increased lethargy, tachypnea, and tachycardia. The ABG results are pH 7.21, PaO₂ 72 mm Hg, Paco₂ 30 mm Hg, HCO₃ 18 mmol/L. This presentation and ABG results are consistent with
A. Metabolic acidosis with hypoxia from septic shock
B. Respiratory acidosis from hyperventilation
C. Metabolic alkalosis associated with diarrhea
D. Respiratory alkalosis with hypoxia from pulmonary emboli (PE)

Test plan topic: Pulmonary, 14% of the PCCN questions

3. A progressive care nurse receives a critical value call on a new patient who has a severe metabolic acidosis and a pH of 7.14. Which potential complication should the nurse be concerned about?
A. Hypertension
B. Ventricular fibrillation
C. Respiratory depression
D. Hypokalemia

Test plan topic: Neuro/Multisystem/Behavioral/Psychosocial, 15% of the PCCN questions

4. A patient with new onset atrial fibrillation (A-Fib) reveals to the admitting nurse that he had recently had decreased exercise tolerance, exertional dyspnea, and dizziness. The nurse auscultates a systolic murmur. These symptoms may be caused by
A. Aortic regurgitation
B. Aortic stenosis
C. Mitral regurgitation
D. Mitral stenosis

Test plan topic: Cardiovascular, 33% of the PCCN questions

5. Initial vital signs on a patient admitted for altered mental status and oliguria are blood pressure of 232/123 mm Hg, heart rate 124/min, respiratory rate 12 breaths per minute, oxygen saturation 91% on room air, and an oral temperature 37.2°C (99.2°F). Acute kidney injury (AKI) is suspected from self-administered high dose of ibuprofen for back pain. Which of the following admission orders should receive the highest priority?
A. Administer antihypertensive agents
B. Apply oxygen by nasal cannula
C. Insert an indwelling urinary catheter
D. Place the patient on fall risk precautions
Test plan topic: Endocrine/Hematology/GI/Renal, 18% of the PCCN questions

Correct Answers and Rationales for PCCN Practice Questions

1. Correct Answer: D
Rationale
Many factors, in addition to age, increase the patient’s risk of cognitive disorders such as delirium developing. Sleep deprivation, metabolic changes, environmental changes, pain, and medications can also contribute to new onset confusion.

Source

2. Correct Answer: A
Rationale
Based on the ABG analysis, the patient is experiencing a metabolic acidosis with hypoxia most likely because of a possible postsurgical infection and/or sepsis. Hyperventilation (B) typically presents with a respiratory alkalosis, diarrhea as metabolic acidosis (C), and a PE (D) as respiratory alkalosis with hypoxia.

Source

3. Correct Answer: B
Rationale
The severe acidemia increases the patient’s risk of ventricular fibrillation developing. Acidosis causes vasodilatation, placing the patient at risk of hypotension, not hypertension (A). The compensatory respiratory response to metabolic acidosis would be tachypnea, rather than respiratory depression (C). Acidosis causes potassium to leave the cell, resulting in hyperkalemia, not hypokalemia (D).

Sources

4. Correct Answer: B
Rationale
The classic clinical manifestations of angina, syncope, and heart failure are often late signs associated with advanced aortic stenosis. Earlier symptoms include a systolic murmur, new onset atrial fibrillation, exertional dyspnea and dizziness, exercise intolerance, and complaints of lightheadedness. Symptoms associated with aortic regurgitation (A) include angina and chest tightness with exercise, fatigue, heart palpitations, and shortness of breath with exertion or when lying down. Symptoms of mitral regurgitation (C) also include fatigue and acute shortness of breath, lower extremity edema, and diuresis when resting. Patients with mitral stenosis (D) complain of shortness of breath, fatigue, heart palpitations, and congested cough.

Source

5. Correct Answer: A
Rationale
The AKI is the admission diagnosis, but the hypertensive crisis is the most important priority. Oxygen (B), catheter insertion (C), and safety (D) are all important, but the blood pressure management is the most immediate concern.

Source

AACN Certcorp publishes a study bibliography that identifies the sources from which items are validated. The document may be found in the AACN CertCorPion exam handbook. The contributor of each question written for this column has listed the source used in developing each item.
Ask the Experts

Best Method for Securing an Endotracheal Tube

Q

What is the best practice for securing endotracheal tubes—taping, using twill ties, or using a commercial tube holder?

A

According to the American Heart Association guidelines, postintubation management requires that endotracheal tubes be secured by using either tape or commercial devices.\(^1\) Evidence is currently limited and has yet to confirm a single best method for securing endotracheal tubes. Commercial and non-commercial methods are used to secure endotracheal tubes. Commercial methods are manufactured devices such as the Hollister AnchorFast and Thomas tube holders. Non-commercial methods consist of adhesive tape and nonadhesive twill product. The best method of securing the endotracheal tube would ensure maximum airway security with minimal risk of unwanted movement and/or unplanned extubation, ease of use, facial skin integrity, and infection control.

Airway Security

Unplanned extubation is a major complication occurring in 3% to 16% of patients receiving mechanical ventilation.\(^2\) According to Epstein et al, failed unplanned extubation increased the duration of mechanical ventilation, ICU stay, hospital stay, and need for long-term care.\(^2\) Preventing an unplanned extubation is the priority when securing an endotracheal tube.

In a study published in Respiratory Care, Shimizu et al\(^3\) concluded that the conventional adhesive tape methods had greater extubation force than the 2 endotracheal tube holders and that extubation force varies considerably depending on the width, length, and type of adhesive tape used. Mohammed and Hassan\(^4\) studied endotracheal tube slippage at various time intervals when twill versus adhesive tape was used to secure the tube. They reported that at 120 minutes after the tube was secured, 73% of patients in the twill group had no slippage but only 36% of patients in the adhesive groups had no slippage.

Tasota et al\(^5\) concluded that both internal and external movement of endotracheal tubes was significantly less with commercial endotracheal tube holders than when conventional noncommercial taping methods were used. As a result, nursing staff expressed a higher level of acceptance of commercial endotracheal tube holders as the method of choice for securing endotracheal tubes.

Ease of Use

Ease of use is best described as the ability to manage the endotracheal tube with greater effectiveness and efficiency. Repositioning an endotracheal tube that has been

Authors

Sean G. Smith, RN, BSN, NREMT-P, C-NPT, FP-C, TP-C, CCRN-CMC, CCRN-K, CEN, CFRN, CPEN, and Tom Pietrantonio, BSRT, RRT-RCP, reply:

©2016 American Association of Critical-Care Nurses
doi: http://dx.doi.org/10.4037/ccn2016214
secured with adhesive tape is a time-consuming process that requires 2 clinicians at the bedside and a complete retaping once the endotracheal tube has been repositioned. Twill tape does not rely on adhesive material to secure the endotracheal tube. Instead, the tube is secured by using knots located around the endotracheal tube and the patient’s neck. In a study published in *Chest*, Levy and Griego reported that nurses and respiratory therapists ranked twill as easier to use than tape.

Commercial devices have been designed to allow quick repositioning of endotracheal tubes. Most devices have incorporated a track to which the endotracheal tube can be moved laterally. Studies have demonstrated that commercial devices tend to be more usable than noncommercial methods. Fisher et al. reported that it took less time to move the endotracheal tube when commercially available devices were used than when noncommercial techniques were used.

**Facial Skin Integrity**

Patients’ comfort and skin integrity are primary concerns when securing an endotracheal tube. According to Mohammed and Hassan, at 12 hours after the tube was secured, no patients in the twill group had a severe facial skin reaction compared with 10% of patients in the adhesive tape group. Furthermore, 74% of patients in the twill group had healthy facial skin compared with 37% in the adhesive tape group. Most commercial endotracheal tube holders offer skin-friendly adhesives that eliminate the need for adhesive tape. However, the force exerted on the patient’s face by many of the commercial securing devices may result in discomfort and formation of pressure ulcers, whereas noncommercial techniques are more form-fitting and do not have the same pressure point issues as seen with commercial devices.

**Infection Control**

It is important that nurses and respiratory therapists provide oral care around the endotracheal tube. Booker et al. identified barriers to effective oral care. Restricted access to the oral cavity because of the endotracheal tube and fear of dislodging the endotracheal tube, provoking aspiration, or causing discomfort were some of the factors contributing to ineffective oral care. Mohammed and Hassan reported that at 24 hours after the tube was secured, 80% of patients in the twill group had healthy oral mucosa compared with 37% of patients in the adhesive group.

Commercial endotracheal tube holders facilitate better routine oral care because of their ease of use. The ability to relocate the endotracheal tube allows nurses and respiratory therapists an unobstructed oral cavity for providing oral care without the fear of endotracheal tube dislodgment.

**Conclusion**

Many devices and methods have been evaluated in search of finding the best method to secure an endotracheal tube, and each has its advantages and disadvantages. It is important that hospitals develop and maintain a collaborative multidisciplinary policy that allows all clinicians to be competent and effective when providing postintubation care.

Financial Disclosures

None reported.

References


Cochrane Review Summary

A summary of findings from the Cochrane Library with implications for critical care nursing

Pulmonary Artery Catheters for Adult Patients in Intensive Care

Adam S. Cooper, RN-BC, MSN

**Review Question**

What effect does using pulmonary artery catheters have on mortality, length of stay, and cost of care for adult patients in intensive care units?

**Relevance to Critical Care Nursing**

In 1970, Drs H. J. Swan and William Ganz introduced a flow-directed balloon-tipped catheter that could be used to monitor patients at the bedside using intracardiac pressure tracings without fluoroscopy guidance. Since then, the pulmonary artery catheter (PAC) has been widely used in intensive care units (ICUs) to guide clinicians in the diagnosis and management of critically ill patients.

To summarize the procedure, a PAC is inserted into a vein (jugular, subclavian, or femoral) via an introducer sheath, through the right atrium and right ventricle to the pulmonary artery. While in this position, a PAC can measure filling pressures of the heart, yielding important hemodynamic monitoring information such as pulmonary and systemic vascular resistance, right and left ventricular end-diastolic pressure, and arterial and venous oxygen content.

Even though the PAC is a valuable diagnostic and monitoring tool, there are potential complications. In 2003, the American Society of Anesthesiology convened a task force to review the risks and benefits related to using a PAC. This task force concluded that the majority of complications were related to insertion, with atrial and ventricular arrhythmias being the most common. This task force also determined that deaths attributed to a PAC are rare.

Several studies (between 2003 and 2006) involving ICU patients have been conducted to determine the effect of PACs on patient mortality. These studies did not show an overall benefit of use of a PAC on patient outcomes. As a result, PACs got considerable negative publicity, especially in the United States, leading to a decline in the use of PACs in clinical practice.

A report looking at trends in the use of PACs in the United States, published in 2007, reported a 65% reduction in PAC use among medical ICUs and 63% reduction in PAC use among surgical ICUs from 1993 to 2004. Recently, however, the way the data from these studies were interpreted has been criticized.

Because of the ongoing debate surrounding PACs, the objective of this systematic review was to search and synthesize all of the evidence from randomized controlled trials (RCTs) that examined the use of a
PAC for monitoring critically ill patients and the effect it has on mortality, length of stay (LOS), and cost of care.

**Study Description and Results**

This summary is based on a Cochrane systematic review\(^1\) that included individual patient data from 13 RCTs. The systematic review was an update to a previous systematic review conducted in 2006. The inclusion criteria for this updated review included studies with greater than 50% adult patients (16 years or older) in which a PAC was placed in the ICU setting or during a surgical procedure leading to an ICU admission. The authors excluded studies that examined patients in whom a PAC was placed only for support before organ donation.

**Primary outcome:**
- Hospital mortality (28 days, 30 days, 60 days, or mortality in the ICU)

**Secondary outcomes:**
- LOS in the ICU
- LOS in the hospital
- Costs of hospital care

The authors independently assessed the risk of bias for each study, including selection bias, performance bias, detection bias, attrition bias, reporting bias, and publication bias. The authors resolved any disagreements by reviewing the data together and by discussion.

Risk ratio was used as the measure of treatment effect for the dichotomous data (mortality), and mean difference was used for the continuous data (LOS and costs of care). Chi-squared analysis was used to assess for heterogeneity and, for any inconsistencies, \( I^2 \) was used to describe the percentage of variability in effect estimates that was due to heterogeneity rather than chance. Intention-to-treat analysis was used and a fixed-effect model was applied for the studies that reported hospital costs.

**Summary of Main Results**

- The pooled analysis included a total of 5686 patients who were admitted to the ICU and randomized to either a PAC group or a control group
- The combined mortality outcome for all studies was not significantly different \( (P = .73) \) between the PAC and control groups (relative risk, 1.01; 95% CI, 0.95-1.08; heterogeneity \( \tau^2 = 0.00; \chi^2 = 5.3, df = 11, P = .92; I^2 = 0\%) \)
- The studies found no significant differences in ICU LOS between the PAC group and the control group. Four studies, of a total of 2723 patients, reported the mean (SD) LOS in the ICU, and data were pooled to give a mean difference in days spent in the ICU of 0.50 (95% CI, 0.44-0.55), comparing management with a PAC versus management without a PAC (test for heterogeneity: \( \tau^2 = 0.00; \chi^2 = 0.66, df = 2, P = .72; I^2 = 0\%) \)
- Nine studies reported the overall hospital LOS, and none of them found significant difference between groups
- Four studies collected costs of care based upon hospital charges. One of those studies reported that the mean costs per patient were significantly higher for the mixed venous oxygen saturation \( (\text{SvO}_2) \) PAC group compared with the standard PAC group. In addition to the hospital charges, one study reported the professional fees charged by the anesthesiologists per patient in each group and found that the fees were significantly higher per patient in the PAC group \( (P < .001) \) compared with the control group.

**Nursing Implications**

This systematic review and meta-analysis of individual patient data from 13 RCTs found that the use of a PAC does not increase or decrease mortality, ICU LOS, or overall hospital LOS. Although a few studies found that PACs increased costs, the variability related to geographic location and health care systems makes this element difficult to generalize. This review showed that the use of PACs is not harmful and, with appropriate training, PACs may be used successfully for diagnostic and hemodynamic monitoring in ICU patients in specific clinical scenarios.

For critically ill patients, the PAC provides hemodynamic monitoring data that cannot be measured through less invasive monitoring alternatives. The data that a PAC provide help guide clinicians in
Looking to increase your presence within the nursing community?

AACN content can be tailored to fit your marketing and promotional needs and can be delivered via print or electronic format.

The American Association of Critical-Care Nurses content is available as:

- Customized Article Reprints
- Sponsored Content Collections (by topic, specialty, etc.)
- Licensed content for special publications or marketing campaigns

Electronic content is user friendly, mobile ready, can be posted to your company website, and/or distributed via e-mail or social media campaigns.

Contact us today to discuss the many options available:

Sheridan

Ideas. Delivered.

Matt Neiderer
Matt.neiderer@sheridan.com
800-635-7181 ext. 8265

Managing an often very complicated regimen of treatment options. Since the PAC is a diagnostic and monitoring tool that does not provide a form of treatment, its use cannot change patients’ outcomes unless the information obtained is used appropriately. The significant decline in the use of PACs in recent years may have led to poor or infrequent training, which can lead to complications in use and improper interpretation of results. Therefore, adequate and ongoing training is necessary to provide effective and safe care for patients with a PAC. CCN

Financial Disclosures
None reported.

Reference

www.ccnonline.org
We know you’re busy – busy at work, busy at home, busy all around. That’s why we want to help you find the articles most important to you in each issue of CriticalCareNurse.

Now you can get an early look at what’s coming in each issue of CriticalCareNurse — before the journal arrives in your mailbox — with eTOCs (electronic tables of contents).

eTOCs are designed to help you identify the articles most important to you in each issue—as quickly as possible. eTOCs also provide links to the online version of CriticalCareNurse if you don’t want to wait for the printed issue.

So why wait?
Go to ccn.aacnjournals.org/cgi/alerts and sign up today.
In Our Unit

The Impact of Family Engagement on Anxiety Levels in a Cardiothoracic Intensive Care Unit

Mariana Skoog, DNP, FNP-BC
Kerry A. Milner, RN, DNSc
JoAnne Gatti-Petito, RN, DNP, CNE
Kiran Dintyala, MD, MPH

Having a loved one in the intensive care unit (ICU) can be stressful and anxiety inducing. Anxiety rates in family members of relatives in the ICU have been documented at 80%. Often family needs are not met during the ICU stay, and unmet needs can contribute to increased anxiety. Higher anxiety levels in family members have been reported to persist even 3 months after their relative is discharged, and development of these adverse psychological outcomes is being called post-intensive care syndrome-family.

A recent review of studies on effective nursing interventions for meeting family members’ needs in the ICU published between 2000 and 2013 revealed that high-level evidence from experimental studies is lacking. Facilitated sense-making (FSM) is a relatively new strategy and middle range theory that can guide nursing interventions to support and engage family members of patients in the ICU. A review of MEDLINE and CINAHL up to August 15, 2015, revealed only a feasibility study conducted by Davidson and colleagues, who reported that FSM was both feasible and helpful to family members of ICU patients receiving mechanical ventilation.

Overview of Facilitated Sense-Making

The theory of FSM is based on the concept that family members need to make sense of the ICU and discover their new roles as caregivers. If this goal is not achieved, a family member’s ability to cope with the critical illness may be altered and adverse psychological events such as anxiety may result. The nurse engages the patient’s family in the sense-making process through a series of specific nursing interventions: assisting the family with understanding the ICU environment, identifying and meeting the family members’ information needs, coaching them on how to visit and meet their own needs, providing family support, and providing activities to perform at the bedside. In publications that address family satisfaction in the ICU, each of these interventions has been identified as necessary.

Authors

Mariana Skoog is an advanced practice nurse in the Cardiology Department, Sacred Heart University College of Nursing, Fairfield, Connecticut, and Hartford Hospital Health System, Hartford, Connecticut.

Kerry A. Milner is an assistant professor at Sacred Heart University College of Nursing.

JoAnne Gatti-Petito is an assistant professor at Sacred Heart University College of Nursing.

Kiran Dintyala is an internal medicine physician, Hartford Hospital Health System.

Corresponding author: Kerry A. Milner, RN, DNSc, Sacred Heart University College of Nursing, 5151 Park Avenue, Fairfield, CT 6825 (e-mail: milnerk@sacredheart.edu).

To purchase electronic and print reprints, contact the American Association of Critical-Care Nurses, 101 Columbia, Aliso Viejo, CA 92656. Phone, (800) 809-2273 or (949) 362-2650 (ext 532); fax, (949) 362-2049; e-mail, reprints@aacn.org."
by family members. Intermediate goals include meeting the needs of patients’ family members and continued communication. Outcomes include decreased anxiety and increased comfort and satisfaction.

The purpose of this study was to increase engagement of patients’ family members by implementing FSM in a cardiothoracic ICU and to measure the effect of FSM on family members’ anxiety levels. It was hypothesized that implementing the FSM interventions with patients’ family members would decrease their anxiety levels during the ICU stay. A secondary aim was to assess for differences between sexes or races in anxiety levels before and after FSM.

**Methods**

This study was approved by the institutional review boards at Sacred Heart University and Montefio e Hospital. Patients’ family members completed the State-Trait Anxiety Inventory (STAI) before and after experiencing FSM.

**Setting**

The setting was a cardiothoracic ICU in Montefio e Hospital, a large regional heart center in New York City that treats a large nonwhite, culturally mixed population of patients. The patients admitted to the cardiothoracic ICU were, in general, older adults who had several comorbid conditions and underwent scheduled cardiac surgeries.

**Sample**

Family members and patients who met the following criteria were invited to participate: patients 12 hours or less after cardiothoracic surgery who were admitted to the cardiothoracic ICU and 2 or more family members at least 18 years old who spoke English and had at least a sixth grade self-reported reading/writing level. Prior estimates of the effect size of FSM on anxiety were not available, so a medium effect size, which is considered reasonable for studying most nursing phenomena, was selected. A paired samples t test was used to compare anxiety scores from before to after FSM. By using an effect size of .50, \( \alpha = .05 \), and a power of 80%, it was determined that a sample size of 64 patient/family member dyads was sufficient.

**Measures**

Anxiety was measured by using the STAI form Y. State anxiety is temporary anxiety in response to some threat that is perceived to be present. Trait anxiety describes a personality characteristic of being anxious on a day-to-day basis rather than a temporary feeling of anxiety. Form Y is the most popular version of the instrument and comprises a scale for assessing state anxiety and another scale for assessing trait anxiety, each consisting of 20 statements.

For state anxiety items, participants report how they feel right now by rating the intensity of their anxiety feelings on a 4-point Likert scale: (1) not at all, (2) somewhat, (3) moderately so, and (4) very much so. For trait anxiety items, subjects report how they “generally” feel by rating themselves on a 4-point Likert scale: (1) almost never, (2) sometimes, (3) often, and (4) almost always. The 2 scales are not meant to be combined, and scores can range from 20 to 80 for each scale. The higher the state or trait score, the greater the anxiety level. The STAI form Y has well-established reliability and validity.

Demographic and baseline characteristics were obtained by self-report from each family member and included age, ethnicity/race, family member’s relationship to the patient, social support, education level, employment status, past or present use of anxiolytic agents, and past or present anxiety. These data were collected to allow sample description and to determine if anxiety levels differed between sexes or races.

**Procedures for FSM**

Within 6 hours after cardiothoracic surgery, family members were approached by the principal investigator (M.S.) in the family lounge, where the study purpose was explained and written consent was obtained. At this time, family members were asked to complete the STAI and the FSM intervention was initiated. The FSM interventions were administered by the principal investigator, and each of the interventions was carried out with the participating family members. To improve intervention fidelity, the principal investigator gave each family member a laminated card (see Figure) that has the FSM interventions listed and a check box to record when they are done. Depending on the
family’s needs, some boxes were left unchecked (e.g., manicuring nails). The principal investigator met with each patient’s family members at least 2 times to address needs and reinforce the FSM. The majority of family members completed the STAI after FSM on postoperative day 3 or 4, with only 3 family members completing the STAI after FSM on postoperative day 2.

Data Analysis

Data were analyzed by using SAS 9.3 (SAS Institute, Inc). The level of significance for all tests was set at a P of .05. Descriptive statistics were used to summarize the demographic and baseline characteristics. A dependent t test for continuous measures and a χ² test for categorical measures were used to test for differences between measures before and after FSM.

Results

Data were collected from May 2014 to August 2014. A total of 64 family members completed the STAI before FSM, and 56 family members completed the STAI before and after FSM. For this study, the internal consistency for the state and trait anxiety levels before and after FSM was excellent (α = .99 for both). Test-retest reliability was evaluated by using the Pearson product-moment correlation coefficient, r. A weak correlation was observed between the state anxiety before and after FSM (r = 0.317, N = 56, P = .01). These results indicate a large decrease in state anxiety in the majority of family members after FSM implementation. A very strong correlation was observed between trait anxiety before and trait anxiety after FSM (r = 0.922, n = 56, P = .001). These results indicated that trait anxiety did not change in the majority of family members after FSM implementation. Thus trait anxiety is a stable measure; you would not expect a family member’s personality trait to change during the study period.

Table 1 displays the self-reported demographic and baseline characteristics of all the family members.

The majority of family members were nonwhite, married, and employed with some education past high school. Most of the sample reported no use of anti-anxiety medications, and only 8 family members reported a formal mental health diagnosis.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female sex</td>
<td>38 (59)</td>
</tr>
<tr>
<td>Nonwhite race</td>
<td>39 (61)</td>
</tr>
<tr>
<td>Marital status</td>
<td></td>
</tr>
<tr>
<td>Married/domestic partner</td>
<td>33 (52)</td>
</tr>
<tr>
<td>Divorced/separated</td>
<td>9 (14)</td>
</tr>
<tr>
<td>Widowed</td>
<td>8 (13)</td>
</tr>
<tr>
<td>Single/never married</td>
<td>14 (22)</td>
</tr>
<tr>
<td>Social support, yes</td>
<td>48 (75)</td>
</tr>
<tr>
<td>Education</td>
<td></td>
</tr>
<tr>
<td>Less than high school</td>
<td>3 (5)</td>
</tr>
<tr>
<td>High school/equivalent</td>
<td>21 (33)</td>
</tr>
<tr>
<td>College degree</td>
<td>40 (62)</td>
</tr>
<tr>
<td>Employment</td>
<td></td>
</tr>
<tr>
<td>Employed</td>
<td>48 (75)</td>
</tr>
<tr>
<td>Retired</td>
<td>6 (9)</td>
</tr>
<tr>
<td>Unemployed/not working</td>
<td>9 (14)</td>
</tr>
<tr>
<td>Current use of anxiolytics</td>
<td>6 (9)</td>
</tr>
<tr>
<td>Formal diagnosis of</td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td>5 (8)</td>
</tr>
<tr>
<td>Depression</td>
<td>3 (5)</td>
</tr>
</tbody>
</table>
with anxiety being the most prevalent. The mean age of the family members was 45.54 years (SD, 13.69 years).

Effect of FSM

Table 2 displays the mean levels of state and trait anxiety overall by sex and race before and after FSM. Overall, mean levels of state anxiety decreased after FSM, whereas the mean trait levels before and after FSM were similar. The mean state anxiety levels in both females and males decreased after FSM. Trait anxiety levels did not change from before to after FSM in either sex. The mean state anxiety levels in both whites and nonwhites decreased after FSM, whereas the trait anxiety levels stayed the same.

Table 3 displays the results of the dependent-samples t tests comparing mean state and trait anxiety levels before and after FSM overall and state anxiety level for each of the demographic variables. The overall mean state anxiety levels decreased significantly after FSM ($P = .001$). The overall mean trait anxiety levels before and after FSM did not differ significantly ($P = .46$). Females, males, whites, and nonwhites all had a significant reduction in their mean state anxiety level after completion of FSM. For these subgroups, mean trait anxiety levels were similar before and after FSM.

Associations With Sex

State anxiety levels were significantly higher in females than in males both before FSM ($t_{52} = 3.63, P < .001$) and after FSM ($t_{52} = 3.06, P = .003$).

Associations With Ethnicity

No significant racial differences (white vs nonwhite) were found in mean levels of state anxiety before FSM ($t_{52} = 0.80, P = .42$) or after FSM ($t_{52} = -0.02, P = .98$). Trait anxiety levels were significantly higher in nonwhite than white family members both before FSM ($t_{52} = 2.10, P = .04$) and after FSM ($t_{52} = 2.03, P = .04$).

Anecdotal Findings

Some family members stated that having a nurse to decode and interpret the cardiothoracic ICU environment at the bedside made them feel less anxious and concerned, and more comfortable. Other family members were afraid to touch their loved one because they feared they could cause harm while their relative was receiving mechanical ventilation and connected to various catheters, monitors, and intravenous medications. Family members were visibly relieved when they understood that the monitor alarms could be seen at the nursing station and these alarms were being evaluated even when the nurse was not in the room. Families expressed appreciation for the FSM laminated card because it gave them tangible resources.

Discussion

The overall mean levels of state anxiety decreased significantly after FSM, suggesting that FSM
may be helpful for decreasing family members’ situational anxiety in a cardiothoracic ICU in the first few days. The overall reduction in anxiety of family members after implementation of FSM was similar to that reported in previous ICU intervention studies that evaluated the effect of needs-based education,8 family diaries,9 or brochures and family conferences10 on family member anxiety. However, the overall level of state anxiety observed in this study was higher than the mean level (45.41 [15.27]) reported in a study11 of a Canadian medical surgical ICU. The difference may be explained by different designs, sample sizes, and settings. In the Canadian study, a descriptive correlation design was used to assess 29 family members in the medical surgical ICU, whereas in the current study, state anxiety was assessed before and after FSM in 64 family members in the cardiothoracic ICU.

Females, males, whites, and nonwhites all experienced significant reductions in their mean level state anxiety after the FSM. For females, males, whites, and nonwhites, mean levels of trait anxiety were similar before and after FSM. State and trait anxiety levels were significantly higher in females than in males both before and after FSM. No significant racial differences were found in mean levels of state anxiety before or after FSM. However, nonwhite family members had significantly higher levels of trait anxiety (eg, day-to-day anxiety) than did white family members.

Few studies have looked at differences in family members’ psychological symptoms related to having a relative in the ICU with respect to the sex and race of the family members. For family members of ICU patients at high risk for dying, being female was associated with higher anxiety levels and being female and nonwhite was associated with higher levels of depression.12 Bailey and colleagues11 also reported that female family members had higher anxiety levels than did male family members.

Few high-quality studies of the effectiveness of interventions for families in the ICU have been published. The most recent American College of Critical Care guidelines for support of families of ICU patients were published in 2007 and based on evidence published before 2004.13 A search for guideline revisions on the website of the Society of Critical Care Medicine revealed that revisions are underway. Although this study used a 1-group quasi-experimental design, anecdotal findings also support the FSM intervention. Communicating with patients’ families and teaching the family members how to participate at the bedside and how to perform personal care activities, such as applying lip balm around an endotracheal tube, and applying hand moisturizer to promote touch, made the family members feel comfortable and less anxious.

The significant reduction in situational (state) anxiety levels that were observed in this study may in part be explained by the specificity of the interventions. Researchers in previous studies8,10 concluded that general interventions (eg, informational booklets and support groups) were not as effective as more individualized interventions (eg, targeting family members’ specific needs and using a specific proactive communication technique) for reducing the anxiety experienced by family members.

Limitations

The findings from this study are subject to several limitations. History and maturation were threats to the internal validity. Because no control group was used, it is possible that the situational anxiety (state) was reduced just by virtue of the patient’s condition improving several days after surgery, family becoming accustomed to the ICU, or some other factor not related to FSM. Time on the unit was not controlled for; however, most family members completed the STAI after FSM by postoperative day 3, suggesting that FSM may speed up the processes of reducing anxiety. Data were collected during the summer of 2014, and the number of cardiothoracic surgeries per day was lower than usual; some days, 4 surgeries were performed, and other days just 1 surgery was done.

Implications for Practice

Results suggest that FSM can engage patients’ families in the care of their loved one and may speed up the reduction in situational anxiety experienced by family...
members in the ICU. The items shown in the Figure are the basis of the intervention, and they can be easily implemented in other ICUs with the help of the nursing staff. In addition to measuring situational anxiety level, it would be beneficial to measure family members’ satisfaction with FSM. To truly test the effect of FSM, future studies should use an experimental design with a control group.

Conclusion

Because of the high anxiety experienced by family members of ICU patients, nurses have an opportunity to address these family needs in order to reduce this anxiety. Although few studies have addressed the most effective ways for nurses to help family members reduce their anxiety level, the FSM interventions may provide a framework for guiding the process and increasing engagement of patients’ family members. Findings suggest that FSM may engage families and speed up the reduction in situational anxiety in the immediate postoperative period in the ICU. Moreover, findings suggest that women may be a target group for future intervention. CCN

Financial Disclosures

None reported.

eLetters

Now that you’ve read the article, create or contribute to an online discussion about this topic using eLetters. Just visit www.ccnonline.org and select the article you want to comment on. In the full-text or PDF view of the article, click “Responses” in the middle column and then “Submit a response.”

References


In Our Unit

In Our Unit highlights unique practices, innovations, research, or resourceful solutions to commonly encountered problems in critical care areas and settings where critically ill patients are cared for. If you have an idea for an In Our Unit article, send it to Critical Care Nurse, 101 Columbia, Aliso Viejo, CA 92655; e-mail, ccn@aacn.org.
Concise yet thorough guidance on how to safely and competently care for adult critically ill and progressive care patients

Endorsed by the American Association of Critical-Care Nurses (AACN) and written by top experts in the field, these authoritative textbooks cover all the must-know details on how to care for these types of patients and their families.

NEW EDITIONS!

AVAILABLE AT: www.aacn.org/essentialsbooks
Voices of Angels: Disaster Lessons From Katrina Nurses

Reviewed by Linda Bell, RN, MSN

After a natural disaster, it may be hard to put the events in context. Those of us who did not experience the disaster firsthand depend on the national news or secondhand stories. After Hurricane Katrina, these stories were about the patients or lack of resources, but we did not hear the stories of the nurses who cared for the vulnerable while their own homes, families, and friends were affected by the natural disaster.

Voices of Angels starts with a description of the situation in New Orleans and surrounding areas before the hurricane hit. This introduction explains the mindset of those who were evacuated and those who could not be evacuated. The hurricane itself was just the start of the disaster. What happened and continued to happen in a city surrounded by water as the rain fell, the water levels rose, and the levees gave way was far beyond what anyone could anticipate.

The stories shared by the nurses in Voices of Angels remind us that in natural disasters the basic needs of everyone are at risk. In some hospitals the staff was able to bring family and pets for shelter. With the loss of outside communications, each hospital became an island. Even units within facilities may not have been able to communicate with each other. Elevator transport was lost when the electricity went down. In hospitals where the emergency generators were below the second floor, power was not available. The nurses not only had to deal with their patients’ fears but their own as well.

The nurses’ stories in the first chapters come from different hospitals and types of units. Each chapter ends with a section of lessons learned that can help other facilities prepare for future events.

The last 3 chapters take a look at the issues from a broader perspective of the failures of disaster response from government agencies and the response of The Joint Commission in pulling together experts and agencies to provide guidance for facilities in the future. There are lessons learned in preparing nurses to function before, during, and in the aftermath of a natural disaster.

The Epilogue brings us up to date with what is currently happening and how things have changed. The authors tell us that the importance of the event is reflected in the description of the lives and times of its citizens: all life is now divided in the public mind as the “before Katrina” time, and the “after Katrina” time.

Voices of Angels is a well written account of a major natural and man-made disaster of our lifetime. Read this book and share it with your family and friends.

Linda Bell is a clinical practice specialist at the American Association of Critical-Care Nurses in Aliso Viejo, California.

©2016 American Association of Critical-Care Nurses doi: http://dx.doi.org/10.4037/ccn2016723
Not Part of the Job: How to Take a Stand Against Violence in the Work Setting

Violence toward health care workers has been a growing concern. We hear on the news about hospital shootings, patients or families being abusive to nurses, and other acts of violence that occur in long-term care, outpatient, or home care environments. Not Part of the Job looks at the risk factors; what employees, employers, and managers should know about the particular risks in their environments; and steps that can be taken to prevent and mitigate the effects of violent behavior. Multiple resources are available in the appendices to help assess and plan for your work environment.

The Nature of Suffering and the Goals of Nursing

Although this book has an older copyright date, the information is even more relevant to the health care environment of today. This book tells the story of suffering that nurses see every day in patients, families, and each other. The authors acknowledge that nurses, as caregivers at the bedside, have a unique window into the ways in which patients of all ages and their support systems experience suffering. The Nature of Suffering speaks to the need for effective palliative and end-of-life care to provide what the patients and families need. Meeting the needs of the patients and families in relief of suffering will also help to support nurses as they are providing care.

Atrial Fibrillation: A Multidisciplinary Approach to Improving Patient Outcomes

The first sentence of this book provides an alarming statistic: “atrial fibrillation (AF) is the most important clinical arrhythmia and affects an estimated 33 million people worldwide.” The risk for AF increases with aging, which leaves all of us potentially at risk. Atrial Fibrillation discusses assessment and various medical, surgical, and cardiac catheterization procedures that may be initiated to manage the patient in AF and addresses the roles of the different providers that interact with the individual in both the outpatient and inpatient environment. Case studies are provided at the end of the book.
Affiliation of Reviewers

Critical Care Nurse is pleased to acknowledge and thank the following people who reviewed manuscripts for the journal in 2015.

Abbas K, Abbasi, PhD
Leanne M. Aiken, RN, PhD
Evangelia Aloumiotani, MD
Cathy Alberda, RN, MS
Irene Alexiazi, RN, DNP, NEA-BCC
Abbas Al Al-Mutair, PhD
Georgina Ano, MD, PhD
Michèle Andréouci, MD
Diane Randall Andrews, RN, PhD
Lois Andrews, RN, DNP, CCRN, ACNS-BC
Adria Arboix, MD
Ehrin J. Armstrong, MD
Alexandra R. Aron, RN, MS
Kostoula Arvaniti, MD
Cecile Aubron, MD, PhD
John A. Aucar, MD, MS, FACQ
Donna Audia, RN
Omar Badawi, PharmD
Polly Bailey, RN, APRN-BC, CCN
Sukhminder Jit Singh Bajwa, MD, MBA, FACE
Gerard Barbet, PharmD
Holly Barsier, MD, PhD
James Beaulieu, PharmD
Renee L. Beckstrand, RN, PhD, PCCN, CNE
Greg I. Beer, MD
Janice M. Beitz, RN, PhD
V. Andrew Belk, PharmD, BCPP
Julia Sarah Belesimis, RN, MSN
Nancy Bergstrom, RN, PhD, FAAN
John W. Beickenbosch, MD
Sue Berney, PT, PhD
Judy Beazanson, RN, DSN, CNS-MS, FAHA
Peter Bie, MD
Nancy Blake, RN, NEA-BCC, CCRN
Christopher M. Bland, PharmD, BCPS, FPTDA
Patricia A. Blissitt, RN, PhD, CCRN, CRNP, CNS, CCM, ACNS-BC
Brittany Bone, PAC
Matthew Borgen, MD
Carole Brancif, RN, DNP, PNPP-BC
Elizabeth Bridges, RN, PhD
Kenneth Broocks, RN, DNP
Dorothy Brooten, RN, PhD, FAAN
David W. Brown, MD
Jeremiah Brown, MD
Gregorzej Bulaj, PhD
John F. Butterworth IV, MD
Naomi E. Caball, PhD
Clifton W. Callaway, MD, PhD
Louise Callow, RN, MSN, CPNP
Silvia Galvino-Gutierrez, MD
Margaret Campbell, RN, PhD, FCPN
Robert M. Campbell Jr, MD
Steven Carlisle, MSN, ARNP
Andrew P. Carlson, MD
Kimberly L. Carter, PharmD
Maria Christabelle Castro, RN/BN, COA, MBA, NEBC
Lyn Ceronsky, DNP, GNP, CSNCA, PCCN
James G. Chandler, MD, FACS
Shanmuganathan Chandrasekaran, MD
Helena L. Chang, MS
Theresa M. Chase, RN, MA, ND
Yin-A. Chen, RN, PhD
Cynthia Chernenky, RN, PhD, CNS, AOCN, FAAN
Wisut Cheungsapitorn, MD
Vincel Chol, MD, MS
Michael J. Cisco, MD
Antonio Cittadini, MD
Diane E. Clark, PhD, FAPRN
Karen Clarke, MD
Lory Clukay, RN, PhD, FNSN, CPNS
Alexandre J. Colon, MD
Laurie S. Conklin, MD
Brian T. Conner, RN, PhD, CNE
Lynda S. Cook, RN, MSN, CRNI
Ryan C. Craner, MD
Claire J. Croulfeild, MD
Sherrill Crozin, ARNP
Norma Gracielia Cuelan, RN, PhD, FAAN

Michelle L. Czarnecki, RN-BC, MSN, APNP, CPNP
Rhonda D’Agostino, RN-BC, CPNP, CRNI, CCNC, FCPN
Jennifer Dannemyer, RN, MSN, APN, ACNP, CRNI
Ily A. Danelich, PharmD, BPH
Judy E. Davidson, RN, DNP
Jesse Davidson, MD, MPH
Nicole De Simone, MD, MPH
Bertrand Decaudt, MD
Jonathan W. Decker, PhD, ARNP, PNPC
Christina H. Deeter, MD
Freda Deliery, MD, FNA, RN, PhD
Stephane Delisle, BRT, MS, PhD, FCPM, FSCRT
Carl-Erik Demple, MD
Andrew J. Denault, MD
Bo Deng, MD
Sapana Doshi, PharmD
Alieneck Deshpande, MD, PhD
Helen Dickie, CNS
Sharon Dickison, RN, MSN, CNS-BC, ANP, ACNP
Cristina Dione, MD
Sarah Doll’O’Mallone, MSc, BSc
Amy Donnellan, DNP, CPNP-AC
Michael Dominino, MD
Stacy L. Doyle, RN, MBA
David J. Dryes, MD
Clément Duft, MD
Malcolm Elliott, RN, PhD
Ellen Heidi Elpern, MD
Heidi Engel, PT, DPT
Shane W. English, MD, MS
Milo Engstrom, MD, FACP
Carol Diane Epstein, RN, PhD, FCPM
Jason Ferreira, PharmD, BCPS
Timothy L. Fitzgerald, MD
Kathleen Flattery, DNP, PhD, CCRN, FAAN
Desiree A. Fleck, RN, PhD, CRNP
Julie Flynn, RN, MA/MEd
Vicki Foley, RN, BN, MS, PhD
Daniel L. Fox, MD
Cheri Franklin, RN, MSN, CRNI
Regi Freeman, RN, MSN, CNS
David Gagnon, PharmD
David F. Gardiner, MD
Lohit Garg, MD
Neetika Garg, MD
Melissa R. George, DO
Mostafa Chami, MD
Gordon L. Gillespie, RN, PhD
Donna Gallies, PhD
Amy C. Goo, PharmD
Roman Gokham, PharmD
Carol R. Goss, RN, BC, DNP
Suzanne J. Grant, MD
S. Ryan Greysen, MD, MPH, MA
Julie H. Gross, DMD, MS, MS, RS
Claude Guerin, MD
Loked Gugliani, MD
Samira Haddad, PhD
Carmel Haggerty, RN, CA, MA (Applied) Nm, MEd
Richard L. Hall, MD
Naomi Hammoud, MD, PhD
Marcella Handley, MNSi
Julie Hanley, RN, MSN, CPNP-AC
John P. Harpe, RNCP, MSN
Tajna M. Hartjes, DNP
Lynne Haslam, NP
Kevin Hay, MD, MB, BCA, MRCGP, FCPM
Li He, MD
Joanne Shubert Heil, RN, PharmD
Deborah Henderson, RN, MSN, CNRN
Shoshana J. Herzig, MD, MPH
Laura J. Hinke, MD
John B. Holcombe, MD, FACS
Joel S. Holger, MD
Patrick Honore, MD, PhD
Timothy Hudson, MD
David Y. Hwang, MD
Rebecca Hyas, MD
Francesca Iodice, MD
Ellen Iverson, RN, MPH
Judith Jacobi, PharmD
Gabriella Jaderberg, MD
Mia Jansson, MD
Alexander P. Johnson, RN, MSN, CCNS, ACNP-BC, CCRN
Shannon Johnson, MS
Dennis W. Jones, RN, DNP
Ann L. Jorgensen, ACNS-BO
Esperanza Villanueva Joyce, MSN, BSN, CNS
Deven Juene, MD
Despalk Kaminat, MD, PhD
Biren B. Kamdar, MD, MBA, MHS
April Kapai, DNP, ACNP-BC, FAAN
Fiona E. Karet, RN, PhD, FNASG
Marc Kastrup, MD
Deborah A. Kennedy, MBA, MS, PhD
Dalia Khachamian, PhD, PhD
Mohammad A. Khassawneh, MBBS
Praveen Khilnani, MD
Martha S. Kingson, DNP
Rosanne Kirsch, MD, FPCP, FAAP
Ruth Kleinpel, RN, PhD
Arthur Klein, RN, MS, CPNP, FCPM
Monica Knapp, RN, MSN, CCNS, ACNP-CC, CCRN
Lazaros K. Kouchals, MD
Toni Kosty, RN, MS
Adrienne Kovacs, PhD
Nailay Kumar, MD
Praveen Kumar, MD
Nai Ming Lai, MD
Karino Lachal, MD
Melissa L. Langhan, MD
Grace Larson, RN, MN
Jean-Baptiste Lascaro, MD
Sue Ladet, PhD
Mary Beth Hogan Leaton, MS
Isabel Anne Leidich, MRCP, FRACP, FCPM
Christie M. Lee, MD
Marcia Leveton, MD, FAAPM
Kristyna B. Lewis, RN, MN, CCNC
Pail C. Lewis, PhD
Rodrick Lim, MD
Ruth Lindquist, RN, PhD
Philipp M. Lippe, MD, FACS
Stephen H. Loring, MD
Steven Lucas, PhD
Kengo Maeda, MD
Marc Maeglele, MD
Wendy L. Magee, RN, MSN, NP, FNP
Tim R. Mahara, MD
Thomas Charles Major, PharmD, FACP
Mary Beth Flynn Makic, RN, PhD
Seb Manoach, MD
Paul Mark, MD
Guillia Martensson, RN, PhD
David S. Martin, BS, MB-CHB
Sandria Martins Pereira, PhD
Virginia M. Mason, RN, PhD, ACNS-BC, CCN
Jennifer L. McCandliss, RN, PhD
Natalia S. McAndreas, MS, MSN
Jennifer Meddings, RN, PhD
Katleen C. Merrill, MD
Christian S. Meyhoff, MD, PhD
Marek A. Mirski, MD, PhD
Shakti Bedanta Mishra, MD
Pamela H. Mitchell, RN, PhD, FAHA, FAAN
Kris Mogensen, MS, BS, LDLN, CNOSC
Megan Moore, PhD, MSW
Carole Moore, RN, MSN, ACNP-BC
Shaine A. Morris, MD, MPH
Josh Mugele, MD
Mandy Murphy, RN, CRNP
Kyle P. Murray, PharmD, BCPS
Girish B. Naik, MD
Matthew D. Neal, MD
Kristen Leigh Nelson, MD
Danelle Nielsen, CPNP
Daniel Niven, MD, MS
Michael J. Noto, MD, PhD
Patricia O’Brien, RN, MSN, CPNP-AC
John C. O’Horo, MD, MPH
Karen Nicola Page, RN, ND, RN
Kenya Palmer, MS, MSN, PNPN-BC
Elisabetta Patton, MD
Katerina Pavenksi, MD
Gregory J. Peitz, PharmD, BCPS
Stephen J. Pettig, MD, MS, MPH
Jebeth Pichler, RN, BSN, EDI
Mauro Pirritelli, MD
Theodore Mason Pope, JD, PhD

www.ccnonline.org

CriticalCareNurse Vol 36, No. 2, APRIL 2016

93
Marylin Prusun, PhD, CCNS-BC, CNL, CHFN, FAAN
Sebastien Preaul, MD, PhD
Mitchell A. Pooka, MD, PhD
Christina Purpora, RN, PhD
Suzanne Purvis, RN, DNP, CCNS-BC
Gwen Rempe, RN, PhD
Grace Reynolds, DPA
Sheila Razi, MD
Betina H. Riley, RN, PhD
Leslie Rittenmeyer, RN, PhD, CNS, CNE
Paola Roe-Prior, PhD
Cecelia I. Roseigno, RN, PhD, CNRN
Jennifer E. Rosen, MS, FACC
Philip M. Rosoff, MD, MA
A. Shana Rowland, PharmD, BCPS
Rebecca J. Ruf, RN, DNSc, CCRN
Mary Rummell, RN, MSN, CPNP, FAHA
Heiner Ruchulitz, MD
Anita Rush, RNC, RN
Catherine Jean Ryan, RN, PhD, APN, CCRN
Farhad Salehzadeh, MD
Priya Sampathkumar, MD
Amy Sanders, MD
Jan N. Schievelbein, MD, PhD
David Schell, MD
Margaret L. Schroeder, RN, MN, MSN, PCCN-BC
Daniel Schwarzkopf, MD
Carole Schwobel, MD, PhD
Shelly Schwoedel, RN, MSN, NEA-BC
Elizabeth Scrut, RN, MPH, CCNS, CCRN, PhD
David Severs, MD
Aryeh Shander, MD
Elizabeth Sharpe, DNP, ANP, NNPC-BC, VA-BC
Saadia Sherazi, MD, MS
Robert Sibbald, MD
Sotirisio Siminos, PhD
Venkatesan Sivarajan, MD
Heidi A. Smith, MD
Barbara Sneyers, PharmD
Mary Lou Sole, PhD
Mohammed A. Soliman Hamad, MD
Christine R. Stelmach, MD
Lauren Stewart, PhD
Kyle Elizabeth Stimpert, RN, MSN, ACNP
Lauren Strazulla, MD
Krishna M. Sundar, MD
Benjamin T. Suratt, MD
Joshua T. Swan, PharmD
Keith Mark Sweeze, MD, MA
Paul M. Szumita, PharmD, BCPS
Alexis Tabah, MD
Kimberly L. Tackett, PharmD, MBA, CDE, BCPS
Maged A. Tanios, MD, MPH
Rui Tao, DVM, PhD
Judith Ann Tate, RN, PhD
Sarah Teele, MD
Oliver Michel Theusinger, MD
Elizabeth Joan Thompson, MSN, MBA
Rosemary Timmerman, MSN
Linda Tovée, RN
Glenna Traiger, RN, MSN
Milind S. Tulab, MD, DCH, DNc, FCPS, MNAAMS
Phillip J. Tully, MD
Lyvonne Tumé, MD

Julie M. Turner-Cobb, PhD, C Psychol, ABPP/NS
Beth T. Uhlich, RN, BSN, FACHE, FAAN
Antonio Valentini, MD, PhD
Michael van Manen, MD, PhD
Anne-Loes van Staai, RN, MD, MA, PhD
Wayne Varnadore, MD, BCN (Hon.), PCCP (AP), PCGE
Avelino C. Verceles, MD
Christophe Vermeulen, MD
Mikie Visser, MD
Katherine Vogel Anderson, PharmD
Rais Vohra, MD
Ladislav Volcire, MD, PhD
Dorothy Wade, PhD
Leonie Weidbrodt, CNS
Howard A. Werman, MD
Robert D. White, MD
Roberta G. Williams, MD
Kathleen Willisson, RN, MS
Lisa Wolf, RN, PhD, CAE
Kim M. Wong Luma, MD
M. Neil Woodall, MD
Gina Woody, RN, DNP
Nina Wordf, MD
Perda Yama, MD
Scott W. Yates, MD, MBA, MS, FACP
Timothy I. Yoder, PhD
Howard Yonas, MD
Fernando Godinho Zampieri, MD
Christine Zawistowski, MD
Zhongheng Zhang, MD
Jerry J. Zimmerman, MD
Armin Zittermann, MD

**New Jersey**

**Jackson**

**CCRN PCCN Review Course**

**Date:** April 21-22, 2016  **Place:** Meridian Health Village at Jackson Cranberry Room A & B  **Address:** 27 South Cooks Bridge Rd, Jackson, NJ 08527  **Sponsor:** Jersey Shoreline Chapter of AACN  **Keynote Speaker:** Nicole Kuchpich  **Contact:** MaryEllen Strozak  **Phone:** (732) 530-2543  **E-mail:** mstrozak@meridianhealth.com  **Fee:** $235

**Texas**

**Houston**

**Certification in Legal Nursing Consulting (5-Day Seminar and Online)**

**Date:** June 6-10, 2016  **Place:** Omni Houston Hotel Galleria  **Address:** Four Riverway, Houston, TX 77056  **Sponsor:** Vickie Milazzo Institute  **Keynote Speaker:** Vickie L. Milazzo  **Contact:** Vickie L. Milazzo  **Phone:** (800) 880-0944  **Fax:** (713) 942-8075  **E-mail:** mail@LegalNurse.com  **Website:** www.LegalNurse.com  **Fee:** Varies  **Credits:** 25.3 CEUs  **(5-day seminar), 40 CEUs (online)**

**San Antonio**

**Leadership and Engagement: From the Bedside to the Boardroom**

**Date:** April 28, 2016  **Place:** Embassy Suites  **Address:** 7750 Briarbridge, San Antonio, TX 78230  **Sponsor:** San Antonio Chapter of AACN  **Keynote Speakers:** Vicki Good, David Taylor III, Mauro Ortiz, Cathy White, Elizabeth Hummickt  **Contact:** Suzanne Feliciano  **Phone:** (210) 508-3664  **Fee:** Member/Military, $75; nonmember, $100; student, $40  **Credits:** 7.0 CEUs

**Kansas City**

**CCRN/PCCN Review Course**

**Date:** April 28-29, 2016  **Place:** Evergreen Hospital and Medical Center  **Address:** 12040 NE 128th St, Kirkland, WA 98034  **Sponsor:** North- west Washington Evergreen Chapter and Mountain to Sound Chapter of AACN  **Contact:** Mary Jo Kelly  **E-mails:** education@mtsaacn.org or aacn.nwec@gmail.com  **Fee:** Member, $170; nonmember, $195

**For Education Directory submission guidelines**

contact CCN Education Directory, 101 Columbia Aliso Viejo, CA 92656  **Phone:** (800) 899-1712  **E-mail:** ccn@aacn.org
Your ticket to a great job offer.

Post your CV or résumé today.

You’re admitted to AACN’s Official Career Center. Designed as a comprehensive career resource for nurses of all levels, the Career Center enables you to explore job postings by specialty area, location, and hospital/facility.

→ Search daily job postings on the homepage.
→ Choose from the best career opportunities in nursing.
→ Start now. The perfect job may be waiting for you.

AACNCareerCenter.org
I Am a Critical Care Nurse

Christine B. Lawlor, RN, BSN, CCRN-CSC, is a staff nurse at NorthShore University HealthSystem in Evanston, Illinois.

Why did you become a nurse?
I have 8 siblings and I looked up to my big sister, who was a star orthopedic nurse clinician. I worked as a patient care technician in college and loved the human side of nursing. I knew I couldn’t sit behind a desk; I had to be on the move.

What about your job as a nurse makes you happy?
I’m most happy after an exciting day. Caring for a crashing patient requires real-time integration of theory and skills. Prioritizing physician orders, tests, and procedures and translating those therapies to patients and their families make for both a tense and satisfying day. I strive to embrace any learning opportunity and to pass that knowledge to new nurses. I’m proud to precept many nurses in our unit.

Tell us about an extraordinary experience you’ve had as a critical care nurse.
Achieving certifications in critical care and cardiac surgery has been extraordinary to me. It gave me the confidence to embark on education projects in my hospital. Specifically, I’ve worked in our new simulation lab on several projects. We recently started continuous renal replacement therapy (CRRT) on our unit. A colleague and I worked with the intensive care unit team, renal team, and simulation team to design a CRRT simulation case. We ran this case during our skills day, and the evaluations were spectacular. Our nurses really enjoyed it and learned a lot from the program. We have moved forward to perform all of our skills day in simulation and are working on new simulation cases each year. I am grateful that our organization supports these projects. To be able to practice high-risk, low-volume therapies in a simulated environment is extraordinary.

What are the challenges you encounter and how do you overcome them?
Having too little time to do so much is tough. Wanting to stay with one patient and family but needing to be with another patient has always been a challenge. Learning to organize my day and communicate clearly and effectively with my patients and the multidisciplinary team has been a huge help.

What has your journey as a nurse been like?
My experience over the past 15 years has allowed me to attain and master my knowledge and skills to help patients and their families. I look back on the early days when I was nervous about caring for patients. I still feel those same butterflies, but I’m reassured by my great colleagues because no one is alone in our unit. We support and help each other. Every day brings new challenges and opportunities in nursing.

At the end of a busy day, how do you find balance in your life?
Finding some humor is always a help to diffuse a difficult situation. I enjoy working out with friends. I’m married to a wonderful man and active in my 3 children’s school. Seeing life through a 5-year-old’s point of view helps put things into perspective. Finally, my faith is important and Sunday Mass enriches my life.

What would we be surprised to know about you?
I’m a pathetic Cubs fan and want to see the World Series in Chicago!

How has AACN played a role in your career?
AACN’s certifications have influenced my career tremendously. Certification challenged me to learn and apply that knowledge, giving me confidence to push forward with the education in my unit. I honestly don’t think those educational programs would exist if it weren’t for certification. CCN

©2016 American Association of Critical-Care Nurses
doi: http://dx.doi.org/10.4037/ccn2016646
Aware™ Alarm Management and Reporting tools provide meaningful and actionable patient centric data to identify which alarms are most critical to focus on and manage for individual patients.

Meaningful alarm management gives you the time to do what matters most, responds to actionable alarms and focus on patient care that makes a difference.

Let us help you make AlarmSense. The first step is to become Aware.
IN SYNC
for the way you work.

Integrated Pumps  Integrated Reporting  Integrated EMR

Working together, the sum is greater than its parts.

Introducing the Synchronized Intelligence™ Infusion Platform from B. Braun.
To learn more visit BBraunUSA.com/ Sync