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CANADIAN ASSOCIATION OF CRITICAL CARE NURSES

Canadian Association of Critical Care Nurses

Vision statement

All critical care nurses provide the highest standard of patient and family centred care through an engaging, vibrant, educated and research driven specialized community.

Mission statement

We engage and inform Canadian Critical Care nurses through education and networking and provide a strong unified national identity.

Values and beliefs statement

Our core values and beliefs are:

- Excellence and Leadership
  - Collaboration and partnership
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- Dignity and Humanity
  - Respectful, healing and humane critical care environments
  - Combining compassion and technology to advocate and promote excellence
- Integrity and Honesty
  - Accountability and the courage to speak up for our beliefs
  - Promoting open and honest relationships

Pathways to success

1. **Leadership:**
   - Lead collaborative teams in critical care interprofessional initiatives
   - Develop, revise and evaluate CACCN Standards of Care and Position Statements
   - Develop a political advocacy plan

2. **Education:**
   - Provision of excellence in education
   - Advocate for critical care certification
3. **Communication & Partnership:**
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   - Enhancement and expansion of communication with our members
4. **Research:**
   - Encouraging, supporting, facilitating to advance the field of critical care
5. **Membership:**
   - Strive for a steady and continued increase in CACCN membership

Implementation of the SLEEP-MAD mnemonic for improving sleep quality in the intensive care unit: A pilot study

By Jinglin Tang, PharmD, Gloria Su, PharmD, Vincent H. Mabasa, PharmD, Christine Thomas, BScN, RN, Katherine Suchorowski, BScN, RN

Abstract

Background: *Patients report inadequate sleep as one of the most stressful factors of their intensive care unit (ICU) admission. SLEEP-MAD is a mnemonic that has been developed as a standardized nursing tool to help improve patient sleep quality in the ICU setting. As a patient care bundle, SLEEP-MAD stands for Sedatives and stimulants, Lights, Earplugs, Environmental disturbances, Pain assessment, Medications, Activity, and Delirium.*

Methods: *We conducted an observational, prospective, single-site quality improvement pilot study in the Burnaby Hospital ICU to assess the feasibility and outcomes of SLEEP-MAD mnemonic implementation. The study consisted of three phases: pre-mnemonic implementation, training and education, and post-mnemonic implementation.*

Results: *The primary outcome of patient sleep quality was improved post-mnemonic implementation, as per consistently higher Richards-Campbell Sleep Questionnaire (RCSQ) scores (difference range of 4.9 to 10.5 when analyzed on a per patient basis). However, the RCSQ score differences were not found to be statistically significant. This study also supported feasibility of incorporating the SLEEP-MAD mnemonic into nursing workflow in the ICU. As a secondary outcome, mnemonic compliance rate was 72%. Other secondary outcomes included no observed statistical differences in incidence of delirium and mortality pre- versus post-mnemonic implementation. Longer ICU length of stay and increased use of sedatives were observed in the post-phase, potentially indicating sicker patients.*

Conclusion: *This pilot study demonstrated improved patient sleep quality with SLEEP-MAD mnemonic implementation, but no statistical significance. It is necessary and feasible to conduct larger validation studies to confirm this observation.*

Key words: critical care, intensive care, sleep, quality improvement, standardized approach, SLEEP-MAD mnemonic

Tang, J., Su, G., Mabasa, V.H., Thomas, C., & Suchorowski, K. (2019). Implementation of the SLEEP-MAD mnemonic for improving sleep quality in the intensive care unit: A pilot study. *The Canadian Journal of Critical Care Nursing, 30*(4), 5–9.

Background

Poor sleep is a significant deterrent to optimal patient care in the intensive care unit (ICU) setting. Patients report inadequate sleep as one of the most stressful factors of their ICU admission (Beltrami et al., 2015; Kaplow, 2016). Negative sequelae associated with sleep deprivation include increased cardiovascular morbidity and mortality, decline in respiratory status, metabolic disturbances, weakened immune system, delirium, and negative mental health outcomes (Beltrami et al., 2015; Salas & Gamaldo, 2008).

SLEEP-MAD is a mnemonic that has been developed as a standardized nursing tool to help improve patient sleep quality and outcomes in the ICU setting. As an evidence-based patient care bundle consisting of both pharmacological and non-pharmacological measures, SLEEP-MAD stands for Sedatives and stimulants, Lights, Earplugs, Environmental disturbances, Pain assessment, Medications, Activity, and Delirium (Table 1) (Mabasa, Suchorowski, Thomas, & Su, 2018). To date, SLEEP-MAD is the first standardized approach developed for ICU nurses and nursing students as an aid to a systematic assessment and implementation of sleep improvement interventions in critically ill patients.

The primary objectives of this study were to assess the feasibility and impact of SLEEP-MAD mnemonic implementation on patient sleep quality in the Burnaby Hospital ICU.

Table 1. *The SLEEP-MAD Mnemonic*

S	Sedatives and Stimulants	Administering stimulants in the morning and sedatives in the evening.
L	Lights	Lights on, curtains open during the daytime. Dimmed lights and eye masks at night.
E	Earplugs	Earplugs to minimize noise.
E	Environmental disturbances	Bundling nursing patient care activities at night, minimizing ambient noise, controlling room temperature.
P	Pain assessment	Optimizing pain management.
M	Medications	Pharmacological measures as a last resort, minimizing withdrawal from sedatives or substances patients were on at home.
A	Activity	Increased stimulation and patient activity during the daytime.
D	Delirium	Optimizing delirium management.

Methods

This was an observational, prospective, single-site, quality improvement pilot study conducted in the Burnaby Hospital ICU. The study consisted of three phases: pre-mnemonic implementation, training and education, and post-mnemonic implementation. In the pre-mnemonic phase, baseline data were collected to assess patient sleep quality in the ICU prior to SLEEP-MAD mnemonic implementation. This was followed by a one-week training and education period for the ICU nursing team, as an introduction to the concepts outlined in the SLEEP-MAD mnemonic. Follow-up data were then collected in the post-mnemonic phase as a comparator to assess patient sleep quality and outcomes after mnemonic implementation.

The primary outcome of patient sleep quality was measured using the Richards-Campbell Sleep Questionnaire (RCSQ). The RCSQ consists of five questions assessing each of the following five domains of sleep: depth, latency, time awake, return to sleep, and overall quality. Scores range from 0 to 100 with higher scores indicating better sleep. This sleep measurement tool has been validated for use in the ICU population. Nursing and patient self-responses showed a moderate correlation, with nursing responses tending to overestimate patient sleep quality (Kamdar et al., 2012). Survey responses were recorded twice each morning to assess patient sleep quality from the previous night: once by the night-shift nurses prior to end of shift and once by the patient themselves, if capable. For data analysis, only one survey response per patient per night was included. In situations where both patients' self-responses and nursing responses were available, the former was used.

The secondary objectives of this study were to evaluate compliance rates of SLEEP-MAD mnemonic implementation and to evaluate the impact of SLEEP-MAD mnemonic implementation on incidence of delirium, ICU length of stay, ICU mortality, and use of sedatives. Mnemonic compliance was reported by the nurses each night as an addendum to the post-phase RCSQ. Compliance was indicated when nurses integrated the SLEEP-MAD process with their patient care activities. Delirium was assessed once per nursing shift using the Intensive Care Delirium Screening Checklist (ICDSC). Information on ICU length of stay and patient status on discharge or end of study phase, whichever came first, were obtained from the ICU patient census records. Use of sedatives was determined through patient chart review of daily medication administration records and flowsheets. At the time of data collection each morning, any sedating medications administered within the past 24 hours were documented. These measurement procedures and tools were standardized between the pre and post phases.

Data collection occurred between November 2017 and February 2018. The pre- and post-mnemonic phases were approximately one month in duration, and convenience sampling was employed with a target of 30 new patients per group. This target was chosen based on expected ICU patient admission rates within the allotted study timeframe. All patients 18 years or older admitted for at least one night in the ICU during the pre and post data collection phases were included in the study. Patients were excluded if they had conditions that would impair accurate completion of the RCSQ. Such conditions included: severe dementia, acute traumatic brain injury, acute stroke, acute hepatic encephalopathy, acute seizures, excessive sedation as defined by a Richmond Agitation Sedation Scale (RASS) goal less than or equal to 3, anoxic brain injury, acute alcohol or illicit drug abuse, or receiving neuromuscular blockade agents. Patients were also excluded if they maintained a non-critically ill status despite being physically located in the ICU. Such patients included those receiving comfort care, off-service post-op sleep apnea monitoring, or ready to transfer out and awaiting bed placement. ICU nurses screened all their admitted patients within the study timeframe and selected only those who satisfied the aforementioned criteria to be included in the study.

As this was a quality improvement study, ethics approval was waived by the Fraser Health Research Ethics Board.

Data Analysis

Descriptive and inferential statistics were calculated with SPSS Statistics Software Version 21. For inferential statistics, both parametric and nonparametric tests were employed, and data sets were analyzed twice. Unpaired t-tests and Mann-Whitney tests were used for continuous variables and the Pearson's Chi-squared test was used for binomial variables. The primary outcome of patient sleep quality was analyzed on both a per patient and per patient night basis. For secondary outcomes, incidence of mortality and ICU length of stay were analyzed on a per patient basis while mnemonic compliance, incidence of delirium, and use of sedatives were analyzed on a per patient night basis.

Results

A total of 34 patients and 104 patient nights were captured in the pre-mnemonic phase, and 48 patients and 145 patient nights were captured in the post-mnemonic phase. There were two carry-over patients in the post-mnemonic phase giving a total of 46 new patients. Overall, patient characteristics were mostly similar across the two groups (Table 2). Two statistically significant differences were observed; there was less use of sedatives prior to the patient's ICU stay (20.8% post versus 50% pre) and more mechanical ventilation or BiPAP use overnight (57.9% post versus 39.4% pre) in the post-mnemonic phase than in the pre-mnemonic phase.

RCSQ scores were consistently higher in the post-mnemonic phase than in the pre-mnemonic phase across all domains when analyzed from both a per patient and per patient night perspective (Table 3). When analyzed from a per patient perspective shown in Figure 1, the score differences between the pre- and post-mnemonic groups ranged from 4.9 to 10.5. The overall RCSQ score was 66.6 (SD 18.1) in the post-mnemonic group versus 58.9 (SD 24.3) in the pre-mnemonic group. When analyzed from a per patient night perspective shown in Figure 2, the score differences ranged from 2.9 to 7.3. The overall RCSQ score was 63.1 (SD 25.1) in the post-mnemonic group versus 58.7 (SD 26.6) in the pre-mnemonic group. However, when statistical tests were conducted, these score differences

Table 2. *Baseline characteristics for the pre-SLEEP-MAD and post-SLEEP-MAD patient cohorts (N = number of patients, n = number of patient nights)*

Characteristic	Pre-SLEEP-MAD N=34, n=104	Post-SLEEP-MAD N=48, n=145
Age, mean ± SD (years)	60.6 ± 18.7	66.7 ± 18.1
Male, N (%)	18 (52.9)	21 (43.8)
Female, N (%)	16 (47.1)	27 (56.3)
On sedatives prior to ICU admission, N (%)	17 (50.0)	10 (20.8)*
ICU admission diagnosis, N (%)		
CNS	4 (11.8)	4 (8.3)
Respiratory	13 (38.2)	16 (33.3)
Cardiovascular	7 (20.6)	10 (20.8)
GI/GU	1 (2.9)	7 (14.6)
Sepsis	4 (11.8)	5 (10.4)
Other	5 (14.7)	6 (12.5)
Received mechanical ventilation/BIPAP overnight, n (%)	41 (39.4)	84 (57.9)*

**P-value <0.05*

did not show statistical significance. In terms of who completed the RCSQ, the proportion of patient versus nurse completion was similar in both the pre- and post-mnemonic phases. The observed patient to nurse RCSQ completion ratio was 46.2%:53.8% in the post-mnemonic phase and 47.1%:52.9% in the pre-mnemonic phase.

The secondary outcome results are shown in Tables 4 and 5. Table 4 shows that the mnemonic compliance rate post implementation was 72% (104/145), and that the statistically significant difference among the secondary outcomes was an increased ICU length of stay in the post-mnemonic phase (5 days SD 9.8 post versus 2 days SD 3 pre). The extent of sedative use between the two groups was only analyzed descriptively. Table 5 shows the frequency and amount of the most commonly used sedatives observed in pre- and post-mnemonic phases. Overall, there appears to be an increased use of sedatives in the post-mnemonic phase in terms of both higher frequency and amount used.

Discussion

Implementation of the SLEEP-MAD mnemonic appeared to improve patient sleep quality in the ICU, as measured by RCSQ score increases of 2.9 to 10.5 units. Although current literature does not indicate a minimal clinically important difference for the RCSQ scoring system, this measurement tool is largely subjective and any difference observed could

Table 3. *Numerical values of the Richards-Campbell Sleep Questionnaire Responses on a per patient and per patient night basis (N = number of patients, n = number of patient nights)*

Criteria	Pre-SLEEP-MAD N=34, n=104		Post-SLEEP-MAD N=48, n=145		Difference (Post minus Pre)		P-value	
	Per night	Avg per patient	Per night	Avg per patient	Per night	Avg per patient	Per night	Avg per patient
Sleep depth, mean ± SD	56.5 ± 28.8	58.8 ± 26.5	63.8 ± 27.2	66.2 ± 20.5	7.3	7.4	0.051	0.157
Sleep latency, mean ± SD	58.1 ± 28.7	56.9 ± 26.7	64.1 ± 29.5	67.4 ± 22.6	6	10.5	0.111	0.058
Time awake, mean ± SD	59.0 ± 27.9	58.9 ± 24.8	61.9 ± 29.9	65.3 ± 22.1	2.9	6.4	0.445	0.222
Return to sleep, mean ± SD	59.2 ± 31.1	58.8 ± 27.8	62.3 ± 30.3	67.4 ± 21.4	3.1	8.6	0.427	0.116
Quality of sleep, mean ± SD	60.5 ± 31.5	61.3 ± 28.0	63.4 ± 29.5	66.2 ± 20.0	2.9	4.9	0.460	0.364
Overall, mean ± SD	58.7 ± 26.6	58.9 ± 24.3	63.1 ± 25.1	66.6 ± 18.1	4.4	7.7	0.181	0.106

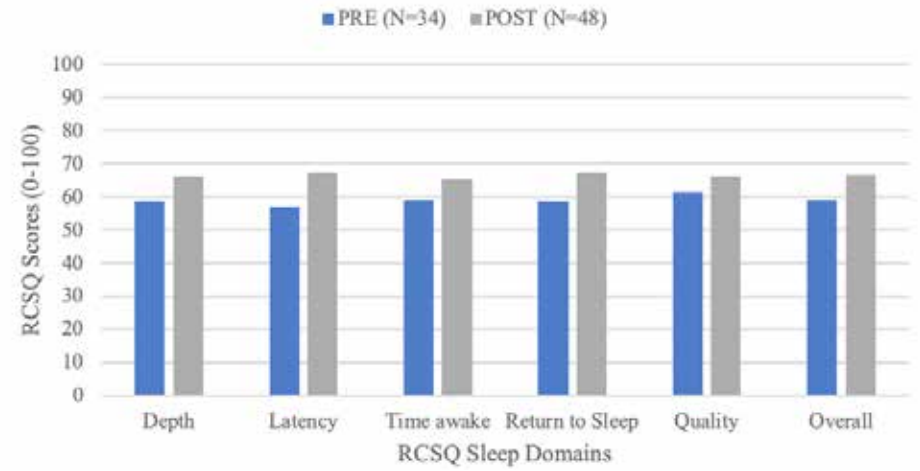

Figure 1. *Graphical comparison of RCSQ scores on a per patient basis*

be considered clinically significant. The findings of this study did not reach statistical significance, however, for which some potential explanations exist. Contrary to hypothesis, patients in the post-mnemonic phase had an increased ICU length of stay and sedative use. This increased ICU length of stay, along with increased mechanical ventilation observed in the post-mnemonic phase, indicate sicker patients, a factor that may be a potential confounder impairing sleep quality in the post-mnemonic phase. Furthermore, both increased sedative use and overnight mechanical ventilation or BIPAP in the post-mnemonic phase could impair sleep architecture, decreasing time spent in the restorative stages of sleep and resulting in poorer sleep quality (Knauert, Malik, & Kamdar, 2014). While a 72% compliance rate was achieved, this could be improved further to optimize results of SLEEP-MAD implementation.

Published results from similar studies are varied. A bundled care approach including both non-pharmacological and pharmacological components implemented by Kamdar et al. (2013) in the medical ICU at Johns Hopkins demonstrated significant improvements in perceived night-time noise and reduction in delirium outcomes. However, similar to this study, improvement in sleep quality did not reach statistical significance. A

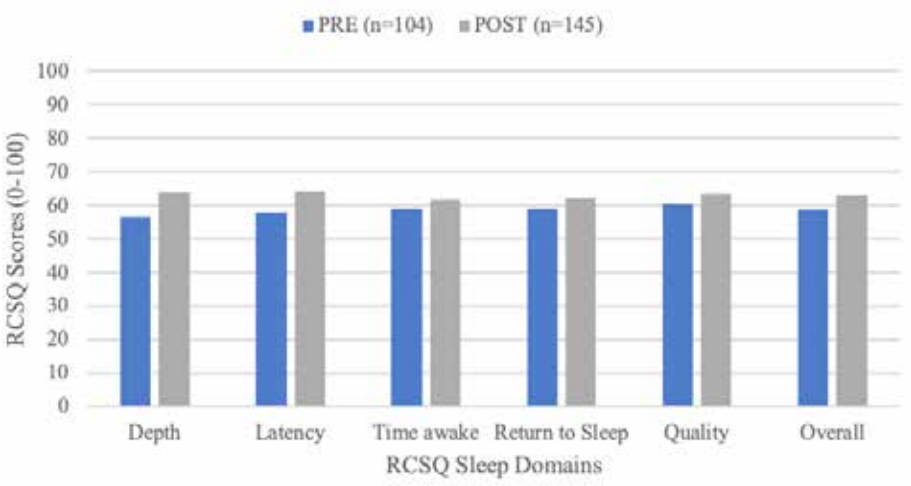

Figure 2. *Graphical comparison of RCSQ scores on a per patient night basis*

Table 4. *Secondary outcomes: Compliance, delirium, ICU length of stay and mortality (N = number of patients, n = number of patient nights)*

Outcome	Pre-SLEEP-MAD N=34, n=104	Post-SLEEP-MAD N=48, n=145	P-value
Mnemonic compliance, n (%)	--	104 (72)	--
Patient nights with delirium, n (%)	6 (5.8)	14 (9.7)	0.311
ICU length of stay (in days), median (interquartile range)	2 (3)	5 (9.8)	0.002*
Expired on discharge from ICU or end of study phase, N (%)	0 (0)	4 (8.3)	0.138

**P-value <0.05*

Table 5. *Secondary outcome: uses of sedatives*

Drug	Pre-SLEEPMAD n=104		Post-SLEEPMAD n=145	
	Frequency, n (%)	Amount (mg)[a]	Frequency, n (%)	Amount (mg)[a]
Opioids[b]	60 (57.7)	57.5	86 (59.3)	96.0
Melatonin	33 (31.7)	1.9	44 (30.3)	1.8
Propofol	13 (12.5)	103.2	30 (20.7)	264.2
Benzodiazepines[c]	2 (1.9)	0.02	18 (12.4)	0.23
Zopiclone	2 (1.9)	0.11	15 (10.3)	0.81
Quetiapine	2 (1.9)	0.48	11 (7.6)	2.8

[a] *Average per night*
[b] *Amount in oral morphine equivalents*
[c] *Amount in oral lorazepam dose equivalents*

study conducted by Patel, Baldwin, Bunting, and Laha (2014) in the United Kingdom implemented only a non-pharmacological bundled care approach. Their results showed statistical significance in terms of increased mean sleep efficiency index, reduced ICU sound and light levels, reduced number of awakenings overnight caused by nursing care activities, and reduced delirium outcomes (less time spent in delirium and reduced incidence of delirium). Despite other authors exploring bundled care approaches to patient sleep improvement in the ICU, the SLEEP-MAD mnemonic is the first developed systematic approach, and this study demonstrates promising results. These results support the feasibility of educating and training critical care nurses to incorporate the SLEEP-MAD process into their workflow, as well as using the RCSQ to evaluate patient sleep quality.

Limitations

There are some limitations that must be considered for future studies. As this was a small pilot study, expanding the scope of future studies to include multiple sites would provide more statistical power and enable further exploration of potential SLEEP-MAD mnemonic benefits. Additionally, the one-week training and education period in this study may not have been long enough to capture all nursing staff, especially those who mostly work night shifts, and may not have provided enough time for the nurses to adapt to and consistently apply the SLEEP-MAD process. Prolonging the education and training period may lead to improved compliance rates and better sleep outcomes. Lastly, sample selection of this study was not randomized, and some baseline characteristics were imbalanced between the pre- and post-mnemonic groups, increasing the risk of confounding factors. The small sample size of this study also presented a limitation to regression analysis. Employing randomization, matched cohorts, or regression analysis in larger future studies would mitigate this limitation.

Conclusion

The findings of this pilot study showed feasibility of and improved patient sleep quality with SLEEP-MAD mnemonic implementation. However, the differences observed in sleep quality between pre- and post-SLEEP-MAD mnemonic implementation were not statistically significant. It is necessary to conduct larger validation studies to confirm these results. Future studies should employ randomization, matched cohorts, or regression analysis and a prolonged training period to optimize results.

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Implications for Nurses

1. SLEEP-MAD advocates for optimization of patient care in intensive care areas through improving sleep quality of critically ill patients.
2. SLEEP-MAD proposes a standardized approach that can be easily incorporated into nursing workflow within critical care areas.
3. The SLEEP-MAD approach can be a useful teaching tool when precepting and mentoring nursing students.

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Submissions Open	October 15, 2019
Submissions Deadline	January 10, 2020
Acceptance Notification	March 6, 2020

PRESENTATION FORMATS

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- **Fast and Focused: 45-minute sessions**
  - designed to speak to a well-defined, singular topic.
- **Concurrent: 60-minute sessions**
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- **Mastery: 120 minute sessions**
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- Printed poster
- Maximum size: horizontal print; 7' 7" wide and 3' 9" high (91" wide by 45" high; 231cm wide by 114cm high)

ABSTRACT GUIDELINES / SUBMISSION REQUIRED COMPONENTS

Abstracts for consideration at Dynamics 2020 should be of interest to critical care providers and high-acuity health care professionals. **Refer to the Dynamics Abstract Submission Process at caccn.ca for full details.**

Language	Abstracts may be submitted in English or French.
Title	Ensure the title represents the intended content of oral session or poster presentation.
Population Focus	Identify the patient population of the proposed presentation.
Purpose / Goals	Provide a brief summary of the main message(s) of the oral session or poster presentation. The purpose / goals will be used in the conference brochure.
Learning Outcomes	Provide two (2) to five (5) learning outcomes for your session.
Oral Session / Poster Description	Oral and poster presentations must reflect current, evidence-based, evidence-informed best practice. Clearly describe the content of the presentation/ poster, identifying key points and relevance to critical care providers and high-acuity health care professionals. Poster presentations should also include (as appropriate): scope, methods, theoretical framework, process, results and conclusions.
References	A maximum of three (3) references to be submitted. All references must be formatted in APA 6th edition.

ABSTRACT SELECTION DYNAMICS PLANNING COMMITTEE

All submitted abstracts will be peer-reviewed for relevance, quality and fit with CACCN's mission/vision and conference educational objectives. Decisions of the Conference Education and Evaluation Committee / Dynamics 2020 Local Operations Committee will be final.

QUESTIONS MAY BE DIRECTED TO

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Photo Credit: Tourism Windsor Essex Pelee Island

The ABCCs of sepsis: A framework for understanding the pathophysiology of sepsis

By El Ladha, RN, Michelle House-Kokan, RN, MSN, CNCC(C), Mary Gillespie, RN, MSN, CNCC(C)

Abstract

Sepsis is a common diagnosis with a high mortality and is a leading cause of in-hospital death. Sepsis manifests in increasing severity along a continuum that begins with systemic inflammatory response syndrome, and progresses to sepsis, septic shock, and multiple organ dysfunction syndrome. During their careers, all critical care nurses will likely care for patients experiencing sepsis. However, this is a challenging task, as treatment modalities and guidelines change in response to our evolving understanding of this complex disorder. To provide quality care in the face of these changes, it is imperative that critical care nurses have a solid understanding of the complex pathophysiology of sepsis. In this article, a unique framework for simplifying, understanding, and recalling the pathophysiology of sepsis is presented as the "ABCCs of sepsis", and the impact of this pathophysiology on a patient's cellular oxygen supply and demand balance is described. The links between the pathophysiology and the patient's overall clinical presentation and consequent treatment are clarified. The ABCCs framework supports nurses in understanding the pathophysiology of sepsis, and the rationale behind treatment protocols. This, in turn, promotes effective clinical decision-making during provision of both medical and nursing care. The ABCCs framework can also be used as a teaching tool by critical care nurse educators to help nurses understand this complex and serious syndrome.

Key words: sepsis, SIRS, multiple organ dysfunction syndrome, pathophysiology, inflammatory response

Ladha, E., House-Kokan, M., & Gillespie, M. (2019). The ABCCs of sepsis: A framework for understanding the pathophysiology of sepsis. *Canadian Journal of Critical Care Nursing, 30*(4), 12–21.

Sepsis is a common diagnosis with an overall mortality rate of around 30% and is the leading cause of in-hospital death (Urden, Stacy, & Lough, 2018). During their careers, all critical care nurses will likely care for patients experiencing sepsis. However, this is a challenging task, as treatment modalities for sepsis change frequently and our understanding of this complex disorder evolves (Cawcutt & Peters, 2014). To provide quality care in the face of ever-changing treatment guidelines, it is imperative that critical care nurses have a solid understanding of the complex pathophysiology of sepsis. The "ABCCs of Sepsis" is a framework for simplifying and understanding the pathophysiology of sepsis, its effects on cellular oxygen supply and demand balance, and the links to the patient's overall clinical presentation and consequent treatment. This framework supports nurses in understanding not only the pathophysiology of sepsis, but also the rationale behind treatment protocols. This, in turn, promotes effective clinical decision-making during provision of both medical and nursing care. Also, critical care nurse educators can use the ABCCs framework as a teaching tool to help nurses understand this complex syndrome.

Sepsis: What Is It?

Case study part 1

Melisa is a 48-year-old woman with a two-week history of "flu". She has seen her primary care provider twice in this period of time with fever, sore throat, cough, and skin rash. Last week she was started on a seven-day course of penicillin for suspected streptococcal infection. Today she presents to the ER with her husband, with worsening symptoms and shortness of breath. On arrival, her temperature is 39.3° C, blood pressure 90/60mmHg, cardiac rhythm sinus tachycardia of 125/minute, and SpO_2 is 85%. Her respirations are shallow at a rate of 28. She appears exhausted and is drowsy. Sepsis is suspected. Immediately, bloodwork is taken, including blood cultures, lactate, electrolytes, creatinine, complete blood count and coagulation screen. She is given 2 litres of normal saline intravenously, and placed on face mask with FiO_2 100%. Broad spectrum antibiotics are started. Her arterial blood gas on oxygen shows pH 7.45, PaO_2 85, $PaCO_2$ 31, bicarbonate 21 and SaO_2 0.89. She is sedated, intubated and mechanically ventilated, and a triple lumen central line is placed before she is transferred to the ICU.

As the current understanding of sepsis and related conditions such as septic shock has evolved over the past three decades (Singer et al., 2016), the definition of sepsis has been revised several times. Up until recently, sepsis was defined as the presence of infection accompanied by the clinical systemic manifestations of infection, moving along a continuum of increasing severity. This continuum began with systemic inflammatory response syndrome (SIRS)/sepsis, and progressed through severe sepsis, septic shock, and multiple organ dysfunction syndrome (MODS) (Dellinger et al., 2013). Most recently, sepsis has been defined by the Sepsis-3 Task Force as "life-threatening organ dysfunction caused by a dysregulated host response to infection" (Singer et al., 2016, p. 6). Sepsis continues to be understood as existing on a continuum of severity that begins with infection (placing a patient at risk for sepsis), and encompasses sepsis and septic shock. In the 2016 definition, sepsis is differentiated from uncomplicated infection, since not all patients who develop infection will develop sepsis. However, patients who do experience sepsis have a 'dysregulated' inflammatory response to the infection, which is widespread throughout the whole body, and can result in organ dysfunction. Septic shock is recognized as a subset of sepsis in which circulatory, cellular, and metabolic alterations are associated with a higher mortality rate than sepsis

alone (Kleinpell & Schorr, 2016). Septic shock is identified clinically when patients: 1) fulfill the definition of sepsis; 2) have persistent hypotension despite adequate fluid replacement, have a lactate ≥ 2.0 mmol/L, and require vasopressors to maintain mean arterial pressure (MAP) ≥ 65 mmHg (Singer et al., 2016).

SIRS and MODS

While these two syndromes are no longer specifically included in the sepsis continuum, they are still important related concepts. MODS can occur if the dysregulation in sepsis and septic shock worsens and organ dysfunction increases. However, it is important to note that MODS can also occur when noninfectious processes trigger dysregulated host responses, e.g., SIRS. SIRS is the normal inflammatory response of the body 'gone out of control'. The normal body response to infection or injury is a regulated inflammatory response, localized around the injury or infection to fight it and promote healing. However, in some instances, this response becomes dysregulated, and becomes magnified and self-perpetuating, thus exerting its effect on the whole body—which gives rise to a systemic inflammatory response, or SIRS. A dysregulated host immune response is common to both SIRS and sepsis, but their origins are different. While sepsis only arises from infection, SIRS can be triggered by a variety of insults, including infection, trauma, thermal injury, pancreatitis, massive blood replacement, hepatic necrosis, and others.

The acute inflammatory response

To understand the dysregulated immune response that underlies the pathophysiology of sepsis, it is necessary to understand the normal acute inflammatory response. Typically, when a microorganism enters the body, or if there is an injury, various inflammatory cells, as well as chemical mediators from cells and from plasma, are released and several plasma enzyme cascades are activated. The intent is to keep the sources of infection or response to injury localized and controlled, and to prepare the body for longer-term healing. The normal acute (local) inflammatory response can be summarized as "VIPP": vascular responses; immune responses; platelet-related actions; and plasma protein responses (Figure 1). The sequence of these four responses depends on the patient's situation and the extent of the insult, but all are part of the inflammatory process.

V: Vascular response.

When tissue damage occurs (injury, microbe entrance, etc.), cytokines such as interleukins, tumour necrosis factor, platelet activating factor, and interferons are released. Cytokines are chemical messengers that mediate the inflammatory response and that trigger local capillary vasodilation and increased capillary permeability. This results in increased blood flow to the area, and fluid "leaks" out of the vessel into the affected tissue. The blood remaining in the capillary becomes more viscous and flows more slowly, and clotting occurs. These effects are further potentiated by other substances in the "VIPP" process.

I: Immune response.

At the same time, the immune response is initiated with the movement of leukocytes into the area of injury through the process of emigration through the permeable capillary walls. Guided by chemicals in the tissues in a process called chemotaxis, neutrophils find and degrade microbes and cellular debris. Monocytes emigrate into the area of injury, enlarge and become macrophages, then destroy microbes and phagocytize foreign matter. Mast cells that live in the tissues degranulate, releasing histamine and prompting the release of other inflammatory mediators. Notably, histamine promotes further vasodilation and increased capillary permeability.

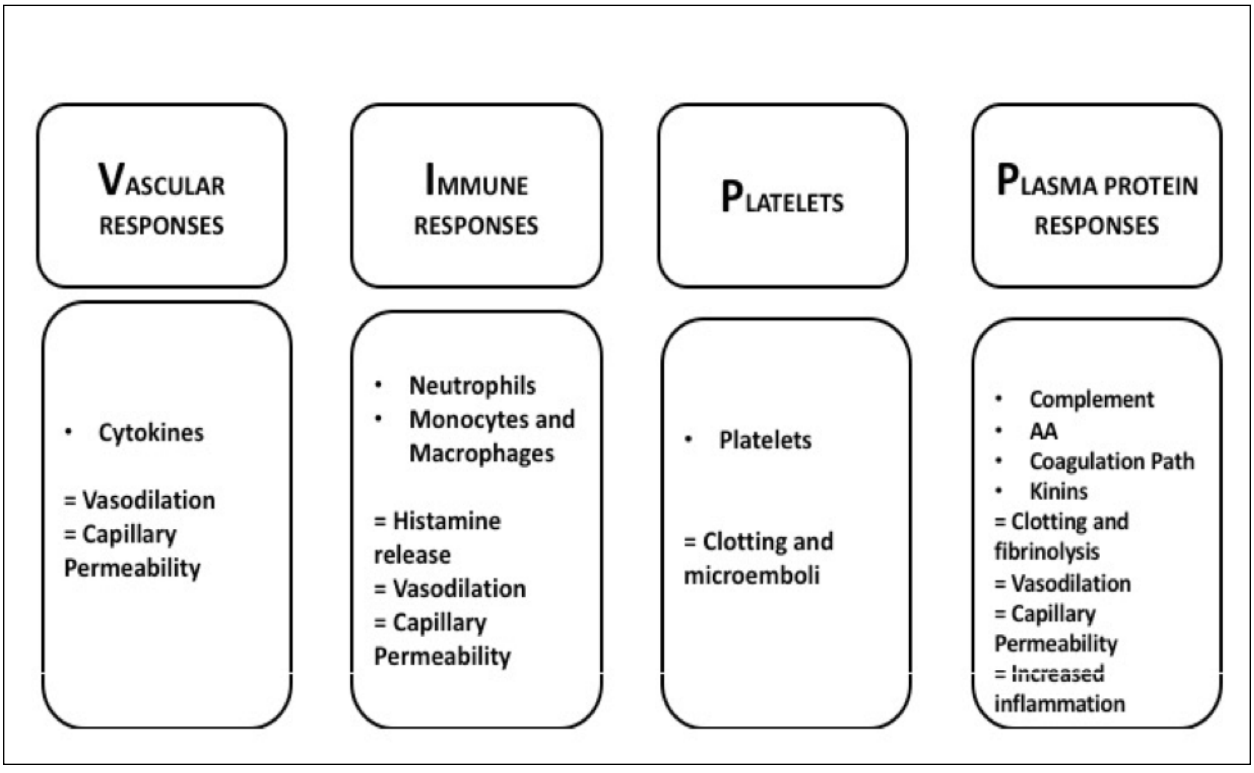

Figure 1. *Local Inflammatory Response: VIPP*

P: Platelet-related response.
The platelet-related response includes increased clotting at the site of injury. This process traps exudates, microorganisms, and foreign bodies. Platelets also play a role in the recruitment and activation of leukocytes (Ferrer-Acosta, Gonzalez, Fernandez, & Valance, 2014).

P: Plasma protein response.
The plasma protein/enzyme cascades that are active in the immune response include the kinin system, the coagulation system, complement system, and arachidonic acid pathway, among others. The end results of these activated systems are increased clotting and micro emboli formation, vasodilation, increased capillary permeability, stimulation of pain receptors, promotion of phagocytosis, and intensification of the inflammatory response.

ABCCs

Case Study, Part 2

In the ICU, central venous pressure (CVP) monitoring is established and a radial arterial line is placed. Melisa is not awake, but rouses to stimulation. Her blood pressure varies between 98/50 and 90/48 mmHg, with a MAP averaging 62 mmHg. Her heart rate is 120 beats per minute and regular, and her CVP is 7 mmHg. Her capillary refill is brisk, and her peripheral pulses are very strong. She has a small amount of edema in both feet and ankles. Melisa's breath sounds are coarse throughout, with scattered crackles bilaterally. She is ventilated on assist-control mode with a set respiratory rate of 14/minute, tidal volume 350 mL, PEEP 5 cm, and FiO_2 0.60. A nasogastric tube is in situ, draining small amounts of green fluid. A Foley catheter is draining an average of 35 ml/hour of amber urine. Her bloodwork arrives.

During sepsis, the normal inflammatory response becomes excessive, or dysregulated. In Figure 2, the acute inflammatory response is represented in the top three boxes: infection/injury triggers a normal inflammatory response, which, in turn, prompts the release of various cytokines. These cytokines signal for more help by activating other chemical inflammatory mediators that are derived from within the plasma or within cells. The inflammatory mediators set in motion a series of pathways, or cascades that create and perpetuate the inflammatory response. Four key chemical mediators and their associated pathways are the primary drivers of the pathophysiology of sepsis: Arachidonic acid (and its associated metabolites); kinins (represented here by Bradykinin); Coagulation cascade; and Complement system. These are the ABCCs of sepsis, and we will examine each, in turn.

Table 1. *Laboratory results*

Laboratory Test	1300 h (ICU Admission)	Normal Values
WBC	18.6	4.0–11.0 x 109/L
HGB	85	120–160 g/L
Platelets	133	150–400 x 109/L
Neutrophils	11.8	2.3–7.7 x 109/L
Bands	5.2	<0.6 x 109/L
Urea	8.5	2.5–8.0 mmol/L
Creatinine	128	40–120 µmol/L
Glucose	9.7	3.5–6.0 mmol/L
Lactate	3.2	0.5–1.8 mmol/L

Arachidonic acid

This cell-derived inflammatory mediator is a highly reactive substance activated by hypoxia, ischemia, endotoxin, catecholamines, and tissue injury (Urden et al., 2018), and exerts its effects through two pathways: lipoxygenase and cyclooxygenase. The lipoxygenase pathway produces leukotrienes and lipoxins, while the cyclooxygenase pathway produces prostaglandins, prostacyclins, and thromboxanes. Each of these end products, referred to as arachidonic acid (AA) metabolites, creates a specific physiologic response (Figure 3). The predominant effects of the AA pathway are vasodilation, increased capillary permeability, and pain. In the context of the normal inflammatory response, the release of AA is helpful, as it creates initial vasoconstriction around the injury (which helps keep it localized), followed quickly by vasodilation that increases blood flow into the area. It also triggers increased capillary permeability, allowing inflammatory cells to migrate from the vascular space into the affected tissues. However, in the dysregulated response that characterizes sepsis, the effects of AA (and its metabolites)

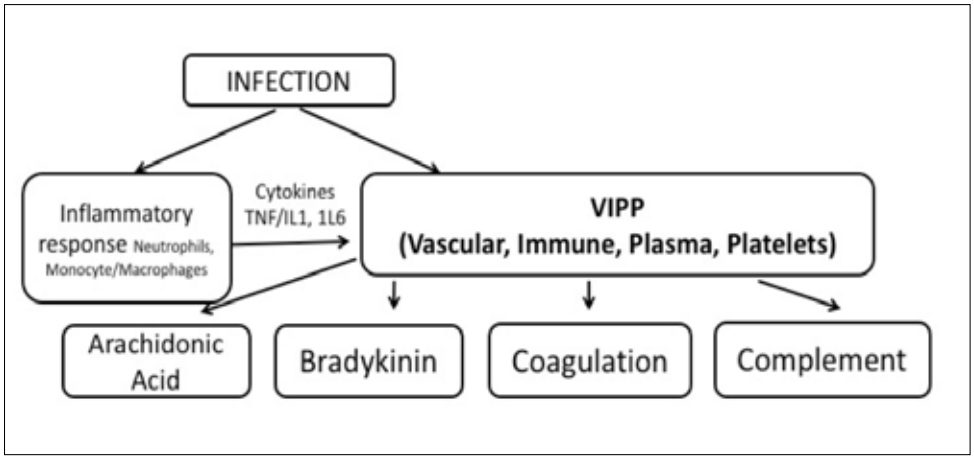

Figure 2. *ABCCs*

become widespread and act on a systemic level, creating haphazard vasoconstriction and vasodilation throughout the body. This results in maldistribution of blood flow: some organ systems do not receive adequate oxygen, while others receive more than they need. Where oxygen supply does not meet cellular oxygen need, the potential for cellular dysfunction exists. AA also causes increased capillary permeability. As this effect becomes widespread, increased fluid shifts from vascular spaces to interstitial and intracellular spaces occur, resulting in loss of circulating volume and decreased preload. Additional effects occur, depending on where the fluid shifts. For example, when fluid shifts occur in the lungs, ventilation and gas exchange become impaired.

Bradykinin

Bradykinin (kinins) are plasma-derived inflammatory mediators activated as part of a normal, acute inflammatory response. Tissue injury (direct or related to microorganisms) activates Hageman factor (Factor XII in the coagulation cascade). Hageman factor stimulates the prekallikrein system, ultimately resulting in the release of bradykinin (Figure 4). The prekallikrein system can also be triggered by the complement system.

Bradykinin is a potent vasodilator, increases capillary permeability, and also stimulates mast cells at the site of injury, resulting in release of histamine. As histamine is also a vasodilator, this adds to the overall vasodilatory effects. Further, mast cells stimulate the AA pathway, additionally amplifying the vasodilation and increased capillary permeability.

So, like AA and its metabolites, the kinin system, especially bradykinin, is helpful in supporting a local inflammatory response to injury/infection. However, when the response becomes dysregulated during sepsis, the impact of systemic vasodilation and increased capillary permeability is significant for the patient. Profound vasodilation, coupled with increased capillary permeability creates a profound decrease in both preload and afterload. Finally, the systemic effects of kinins are magnified by interactions that occur between the plasma protein cascades: these interrelationships underpin the self-perpetuation of sepsis pathophysiology.

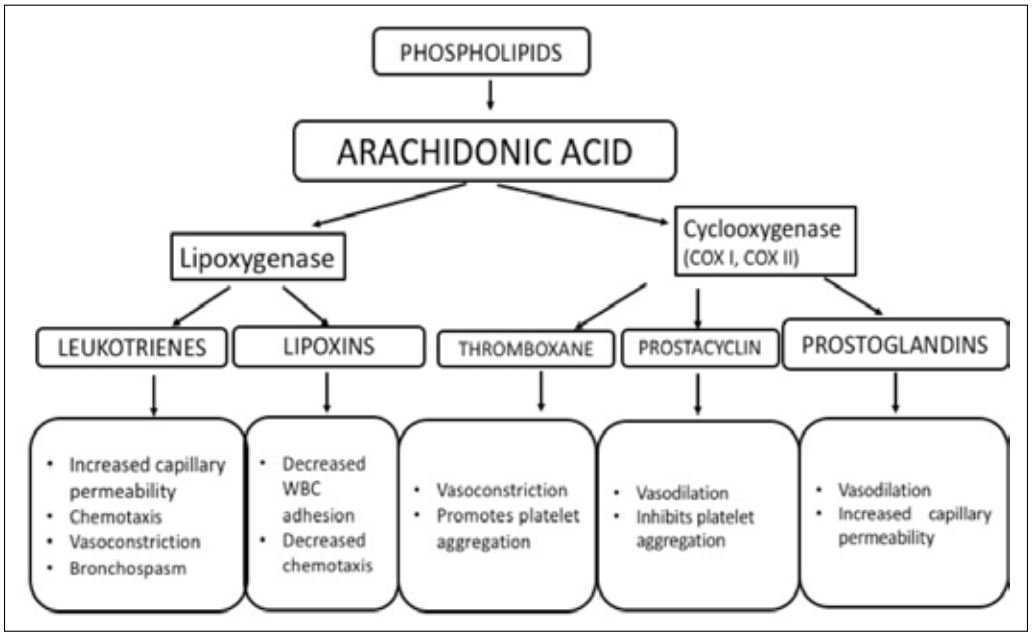

Figure 3. *Arachidonic Acid Pathway*

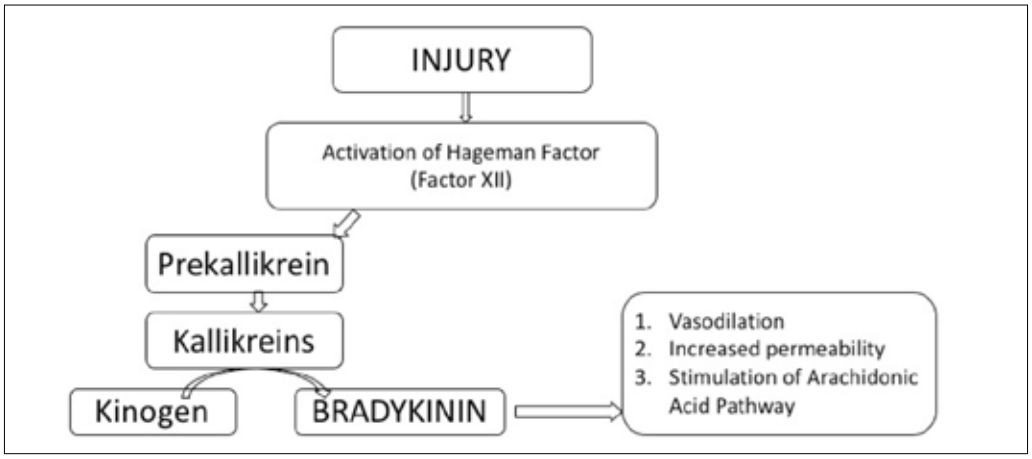

Figure 4. *Bradykinin Pathway*

Coagulation

In the acute inflammatory response, coagulation plays an important role in localizing the injury. A review of the coagulation pathways is beyond the scope of this article, but Figure 5 provides a basic overview of intrinsic, extrinsic, and common pathways. Note that the extrinsic pathway is triggered by tissue factor released from damaged tissues and that the intrinsic pathway is triggered when Hageman factor/Factor XII is activated by exposure to collagen fibres at the site of injury. The two pathways join at the common pathway, which, ultimately, results in formation of a fibrin mesh to which red blood cells and platelets adhere, forming a clot. Platelets in the clot release thromboxane A2, which attracts more platelets to the area, and increases their stickiness, furthering clot formation. Finally, the fibrinolytic pathway facilitates the breakdown and dissolution of the clot when no longer required. However, in sepsis, the normal coagulation process is changed in two ways. First, the coagulation process becomes systemic in its effects. Second, procoagulant aspects of the clotting cascade remain effective, but anticoagulant effects (the fibrinolytic pathway) fail. This failure arises from effects of cytokines released in the inflammatory process that stimulate plasminogen activator inhibitor. This substance prevents plasminogen activators from converting plasminogen to plasmin. The lack of plasmin means that clot breakdown cannot occur. This imbalance is very important in the systemic effects of coagulation in sepsis: the end result is formation of microemboli throughout the systemic circulation, with no mechanism for them to break down. Microemboli impede blood flow through the microvasculature, decreasing cellular oxygen supply and contributing to organ dysfunction.

The coagulation cascade also interacts with other cascades in the ABCCs (Figure 6). Hageman factor triggers release of

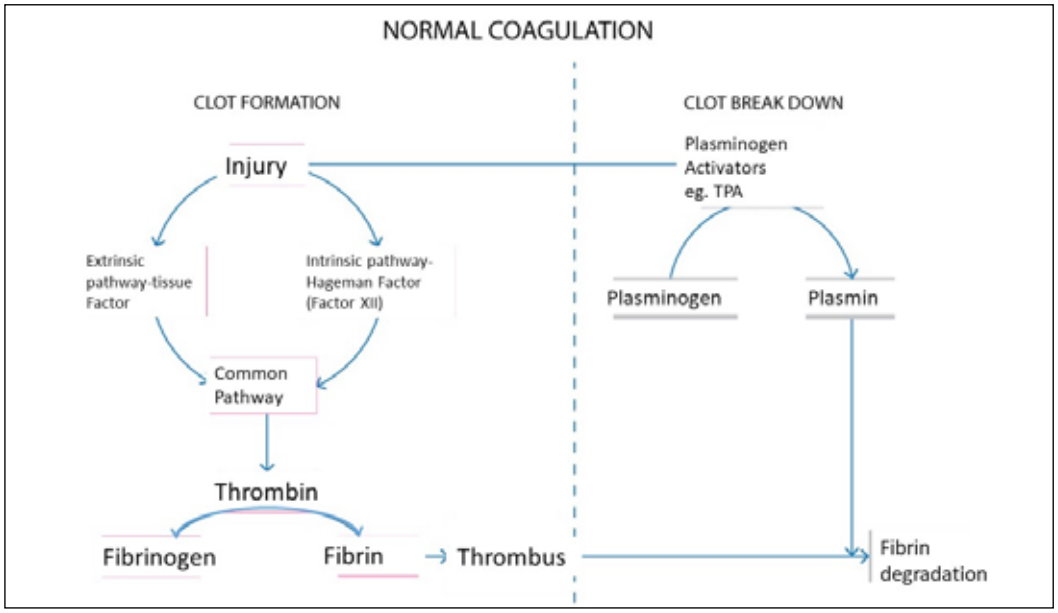

Figure 5. *Normal Coagulation*

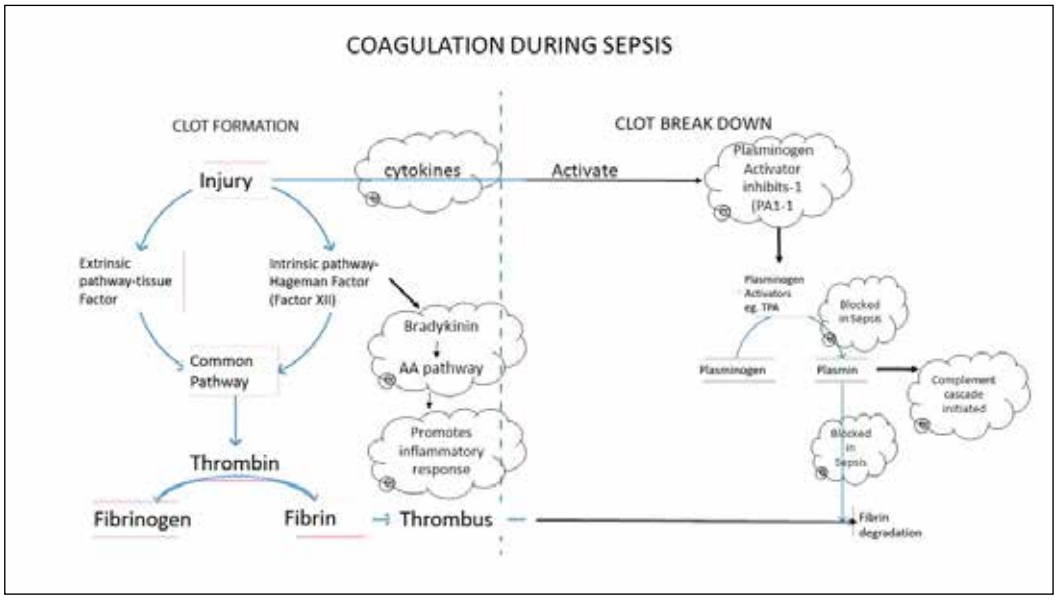

Figure 6. *Coagulation During Sepsis*

bradykinin, which, in turn, stimulates the AA pathway. In addition, plasmin will activate the complement system for a period of time, although this effect fades as sepsis progresses and the fibrinolytic systems becomes increasingly suppressed.

Complement

The second "C" in the ABCCs of sepsis refers to the complement system. This complex group of proteins form a plasma-derived inflammatory mediator cascade that exerts effects through three pathways: classical, alternative, and lectin. Together, these pathways stimulate leukocytes, and promote phagocytosis and immune and inflammatory responses (Figure 7), (Urden et al., 2018). Within these functions, two complement actions are particularly notable. First, the complement system is instrumental in the formation of a membrane attacking complex (MAC) that breaks down the cell membrane of a foreign microorganism, killing it. Second, the complement system "coats" pathogens in a process called opsonization, which more easily allows detection and ingestion by phagocytes. See Figure 8.

The complement cascade is interconnected to other components within the inflammatory response. In particular, released complement proteins stimulate mast cells, which then release histamine and promote vasodilation. As a localized response, the effects of the complement system assist in containing and responding to an infection/injury by promoting phagocytosis and supporting other aspects of the inflammatory response. However, when its effects become systemic during a dysregulated response in sepsis, it becomes harmful: it intensifies inflammatory responses from other cascades, damages endothelium, and causes cell death.

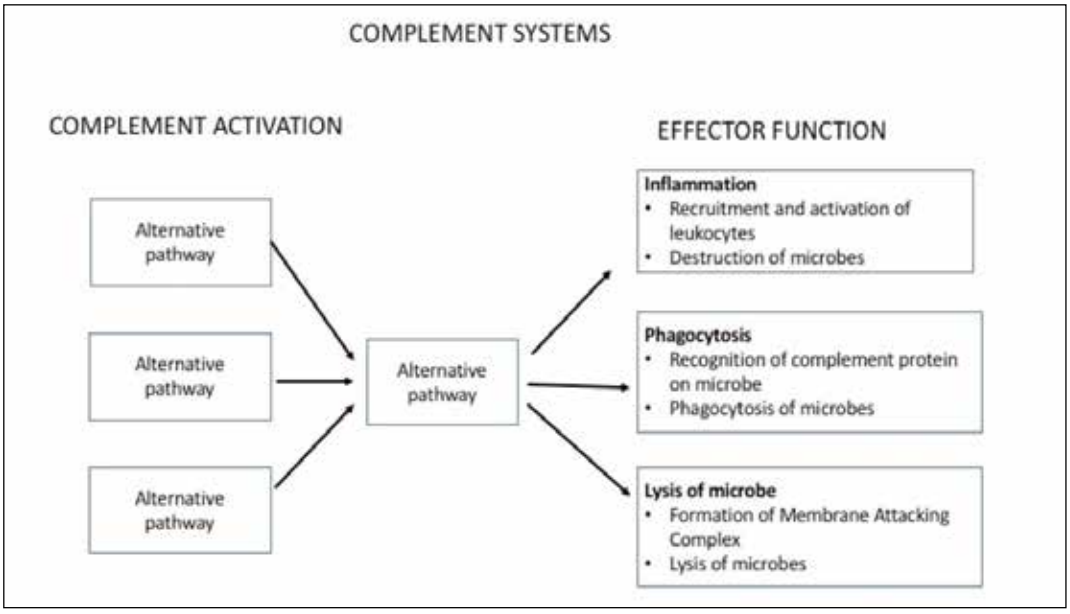

Figure 7. *Complement Systems*

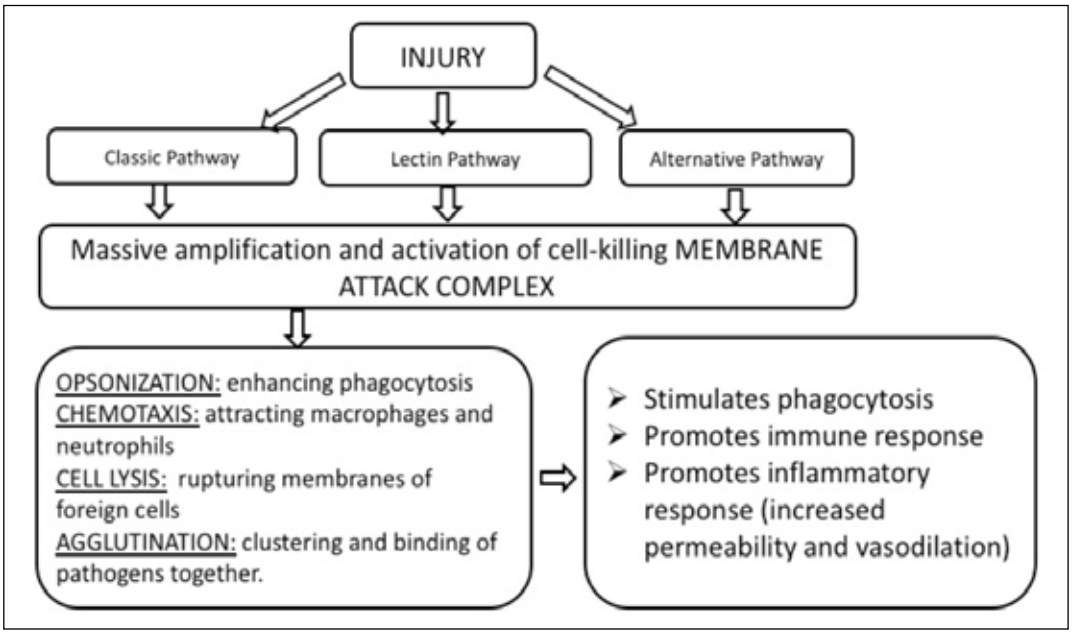

Figure 8. *Complement Pathway*

ABCCs of sepsis: Overall effects

The responses that occur during the dysregulated inflammatory responses associated with sepsis can be summarized into five primary outcomes: systemic vasodilation, increased capillary permeability, inappropriate clotting in the microvasculature, maldistribution of blood flow, and diminished myocardial contractility. Figure 9 depicts the entirety of the ABCCs of sepsis as a framework.

The pathophysiology of the first four outcomes has been addressed in previous discussion. Diminished myocardial contractility in sepsis is multifactorial and presents in varying degrees. Causative factors include release of circulating myocardial depressant factors (MDF), although the exact nature of MDF has not yet been ascertained. Ongoing research suggests some cytokines, nitric oxide, and prostanoids as possible stimuli for MDF production (Kakihana, Ito, Nakahara, Yamiguchi, Tomotsugu, 2016). However, regardless of the causative factors, decreased myocardial contractility adds to the complex pathophysiological presentation of sepsis. In combination, these pathophysiological changes result in poor end-organ perfusion, decreased cellular oxygenation, and potential for organ dysfunction.

ABCCs practice: Case study question 1.

Look back at Melisa's story, and identify evidence of the ABCCs of sepsis (dysregulated host response) from her clinical picture. You may wish to compare your responses to the Answer Key at the end of this article.

Recognizing Sepsis

The clinical picture of sepsis can vary tremendously depending on the site and severity of infection, the patient's pre-existing condition and co-morbidities, and interventions used in management. With this variation, however, the five primary outcomes of the ABCCs of sepsis (see above discussion) give rise to some commonalities in clinical presentation (Figure 10).

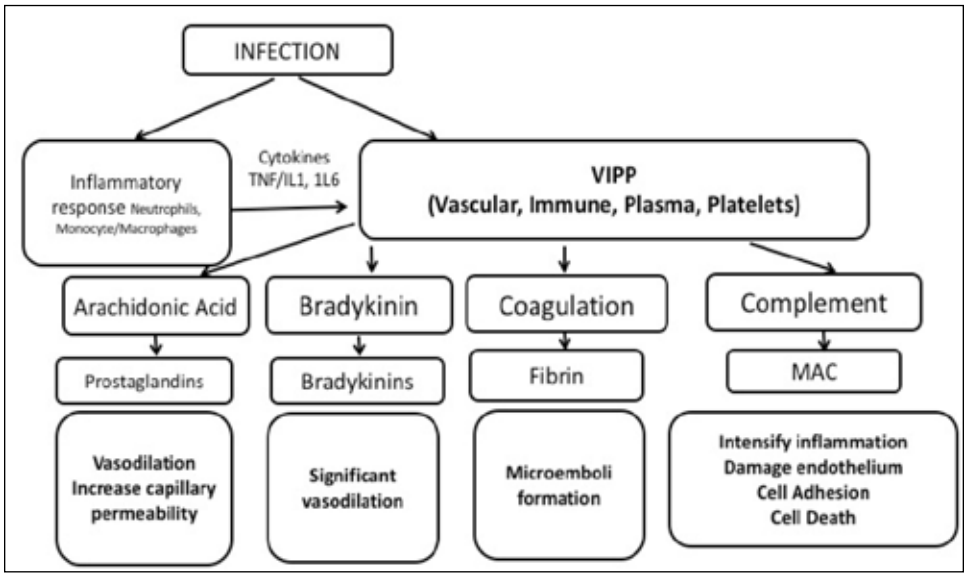

Figure 9. *The ABCCs of Sepsis Framework*

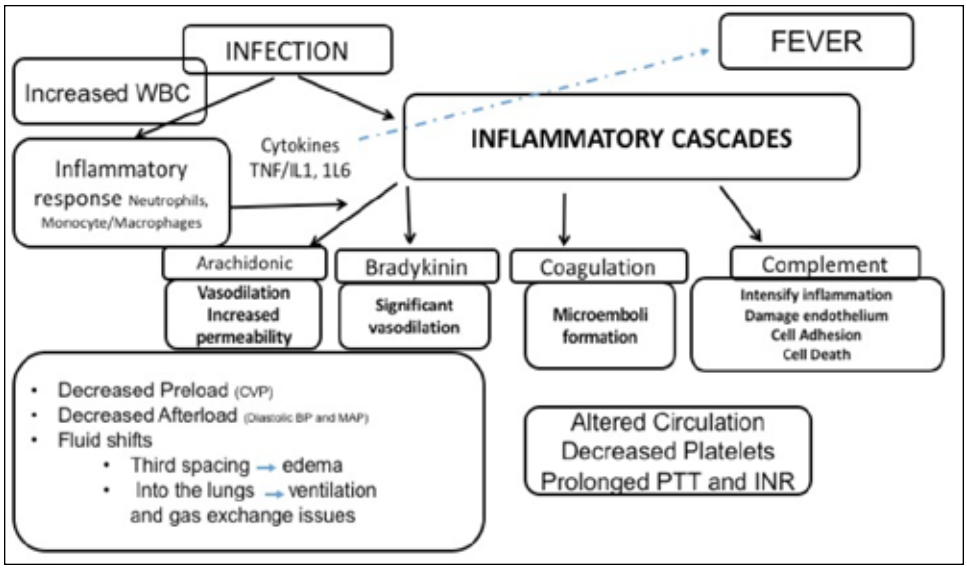

Figure 10. *Presentation of Sepsis*

Table 2. *Sequential organ failure assessment (SOFA) score system*

	SOFA score				
Organ System	**0**	**1**	**2**	**3**	**4**
Respiratory: PO_2/FiO_2, mmHg	≥400 (53.3)	<400 (53.3)	<300 (40)	<200 (26.7) with respiratory support	<100 (13.3) with respiratory support
Coagulation: Platelets, x 103/mm^3	≥150	<150	<100	<50	<50
Liver: Bilirubin, mg/dL	<1.2	1.2–1.9	2.0–5.9	6.0–11.9	>12.0
Cardiovascular	MAP ≥70 mmHg	MAP <70 mmHg	Dopamine <5 or Dobutamine (any dose)[a]	Dopamine 5.1–15 or epinephrine ≤0.1 or norepinephrine ≤0.1[a]	Dopamine > 15 or epinephrine >0.1 or norepinephrine >0.1[a]
CNS: Glasgow Coma Scale	15	13-14	10-12	6-9	<6
Renal: Creatinine, mg/dL					
Urine output, mL/d	<1.2	1.2-1.9	2.0-3.4	3.5-4.9	
<500	<5.0				
<200					

Note: Adapted from Marik, P. & Abdalsamih, M. (2017). SIRS, qSOFA, and new sepsis definition. Journal of Thoracic Disease, 9(4), 943-945.

[a] Catecholamine doses are given as mcg/kg/minute for at least 1 hour. FiO_2: fraction of inspired oxygen; MAP: mean arterial pressure; PO_2: partial pressure of oxygen

Oxygen supply/demand and cellular oxygenation

The oxygen supply and demand imbalance associated with sepsis pathophysiology results in decreased end-organ perfusion and decreased cellular oxygenation. Within this, the maldistribution of blood flow contributes significantly to impaired cellular oxygenation. When cellular oxygenation is inadequate, cells convert from aerobic to anaerobic metabolism. The by-product of anaerobic metabolism is lactic acid, measured in the blood as lactate. Thus, serum lactate, as well as the presence of metabolic acidosis, serve as indicators of end-organ perfusion. Clinically, end-organ perfusion is assessed by signs and symptoms associated with dysfunction within each organ system (e.g., decreased level of consciousness).

Scoring systems

In addition to clinical presentation, scoring systems can assist in early recognition of sepsis and septic shock (Tables 2 & 3). The qSOFA score system can increase early detection of sepsis in community, emergency room, and ward settings (Singer et al., 2016). The qSOFA score is calculated from clinical data only (blood pressure, mentation, and respiratory rate) to permit quick and easy identification of adult patients with suspected infections who may be developing sepsis. qSOFA is not diagnostic, but a score of ≥ 2 should prompt clinicians to further assess and initiate/escalate treatment. The SOFA score, on the other hand, is used to quantify sepsis-related organ failure (Singer et al., 2016). A higher SOFA score is associated with increased probability of mortality.

Table 3. *Quick Sequential Organ Failure Assessment (qSOFA) Score*

qSOFA (Quick SOFA) Criteria	**Points**
Respiratory rate ≥22/minute	1
Change in mental status	1
Systolic blood pressure ≤100 mmHg	1

Note: Adapted from Marik, P. & Abdalsamih, M. (2017). SIRS, qSOFA, and new sepsis definition. Journal of Thoracic Disease, 9(4), 943-945.

The ABCDs of Sepsis Management

Case study part 3

Melisa has been in the ICU for 18 hours now. Still waiting for blood cultures to arrive, the intensivist has ordered ongoing broad-spectrum antibiotic therapy. Over the last 12 hours,

Melisa's temperature has remained elevated (38.8 C), and her MAP has decreased, averaging 58 mmHg, in spite of a her CVP of 7 mmHg. She was given another 500 mL bolus of saline, after which her MAP was unchanged, and her CVP increased to 9 mmHg while her SpO_2 fell to 85%. Currently, crackles are audible throughout her lungs, as are scattered wheezes. Her urine output is averaging 25 ml/hour of amber urine, and her lactate is 3.9 mmol/L. She has been somewhat restless and appears uncomfortable with the endotracheal tube. Based on these findings, her current interventions include: Norepinephrine 5 mcg/minute with a MAP goal of 65 mmHg; FiO_2 0.65, PEEP 7.5 cmH_2O; nebulized salbutamol and ipratropium bromide for bronchospasm; tube feeds initiated at 30 mL/hour; and a sliding-scale insulin infusion at 2 units/hour. Morphine and midazolam are infusing at 2 mg/hour each to achieve a RASS goal of -3.

Two broad goals underlie the management of patients with sepsis: a) to locate and remove the source of infection (and the trigger for the acute inflammatory response); and b) to support physiological function until the dysregulated inflammatory response subsides. Evidence-based guidance to meeting these goals is provided in the *Surviving Sepsis Campaign (SSC) 2016 Guidelines* (Rhodes et al., 2017) that inform management of patients with sepsis and septic shock, and typically form the basis of hospital protocols for sepsis management. A 2018 update to the SSC Guidelines that emphasizes immediate recognition, resuscitation, and management has recently been released (Levy, Evans, & Rhodes, 2018). Readers are referred to the Surviving Sepsis Campaign website at www.survivingsepsis.org for further details.

To simplify and support recall of medical and nursing management strategies included in the SSC bundles, we offer a further framework: the ABCDs of sepsis management. While most management strategies are medical, critical care nurses must carry out and make clinical decisions regarding these interventions. This framework supports nurses in clinical decision making in providing both medical treatment and nursing care. With the overall goals of sepsis management focused on restoring and maintaining adequate cellular oxygenation and tissue perfusion, all interventions are directed towards improving oxygen supply and minimizing metabolic oxygen demand.

A: Assessment, airway and antibiotics

Early identification and strategies to eliminate the source of sepsis are critical. Obtain blood cultures and lactate immediately, and identify and exclude any potential source of infection, such as the removal of any intravascular lines that may be a source (Rhodes et al., 2017). Maintaining an airway is essential to support adequate ventilation and gas exchange. Insert an artificial airway if needed. Antibiotics are a cornerstone of sepsis management. As soon as blood cultures are obtained, broad spectrum IV antibiotics should be administered. Reassess antimicrobial therapy when the gram stain and cultures are available.

B: Breathing

Use mechanical ventilation as needed to support ventilation and oxygenation. Choose appropriate modes to minimize barotrauma and volutrauma secondary to poor compliance, using PEEP to maximize gas exchange. Maximize V/Q matching with appropriate positioning, and manage any bronchospasm that arises due to leukotrienes in the arachidonic acid cascade.

C: Cardiac Output

All three components of stroke volume are potentially impacted in sepsis and will require management. Initial and ongoing preload (and tissue perfusion) assessment should be completed according to the SSC Guidelines, with early, adequate fluid resuscitation using 30 mL/kg of crystalloid fluid initially. Low afterload should be managed with norepinephrine as the first choice, with the addition of vasopressin, or possibly epinephrine, if needed, to achieve a MAP goal of > 65 mmHg. Dopamine is only to be used for patients with a low risk of tachydysrhythmias. Contractility is addressed with adequate preload via fluid resuscitation. However, if signs of myocardial dysfunction are present, or if there is evidence of poor tissue perfusion despite adequate fluid resuscitation and use of vasopressors, an inotrope may be utilized. Dobutamine is the inotrope of choice, using normalizing lactate levels as a titration end point (Rhodes et al., 2017).

D: Drugs and demand

Decreasing metabolic oxygen demand is an important management strategy in sepsis and in optimizing oxygen supply and demand balance. Ensuring adequate sedation according to a sedation scale goal, as well as adequate analgesia, is imperative. The benefit of fever arising from the inflammatory response must be weighed against the disadvantage of the associated increase in metabolic demand. If the patient is actively cooled, the potential for shivering and subsequent increase in metabolic demand must be anticipated and managed.

The term "drugs" is used in the ABCDs of sepsis management to categorize other supportive strategies. These include glycemic control via a protocol, stress ulcer prophylaxis for patients with risk factors, and the possible use of steroids for patients in refractory shock. While not a drug, providing early enteral nutrition, as tolerated, is necessary in the patient with sepsis.

ABCDs practice: Case study question 2.

Use the ABCDs of sepsis management to organize and critique Melisa's treatment so far. You may wish to compare your answer to the Answer Key in the Appendix.

Conclusion

Sepsis is a very common diagnosis in the critical care setting, associated with very high morbidity and mortality. It is also a very complex disorder, involving multiple body systems and physiologic pathways that result in cellular oxygen supply and demand imbalances and subsequent complex

care needs. In addition, treatment guidelines continue to evolve as more research is done, which further challenges medical and nursing management. This article presented a framework for understanding and recalling the complex pathophysiology of sepsis, as well as linked the framework to typical patient presentation and possible treatment modalities. We offer this framework as a tool that assists critical care nurses' clinical reasoning and clinical decision making when caring for patients experiencing sepsis. Further, the framework is recommended for use by critical care nurse educators in teaching/learning about sepsis.

Implications for Nurses

- Sepsis is deadly and must be treated early and aggressively. Its pathophysiology directly impacts the patient's clinical presentation. Being cognizant of sepsis pathophysiology can prompt early recognition of sepsis and/or advancing sepsis.
- Sepsis pathophysiology is complex, but can be taught, understood, recalled, and applied in critical care nursing practice more easily using this conceptual framework.
- Sepsis treatment is directed specifically at its underlying pathophysiology and can be taught, understood, recalled and applied in critical care nursing practice more easily using the ABCCs of Sepsis pathophysiology framework in conjunction with the ABCDs of Sepsis Management framework.

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Appendix

Answer Key

1. What evidence of infection and the ABCCs of sepsis can you identify in Melisa's clinical presentation?

• *Infection: WBC 18.6, high neutrophils and band cells, fever*

• *Arachidonic Acid*

- Vasodilation: widened pulse pressure, low diastolic blood pressure, strong peripheral pulses, brisk capillary refill. Has had significant fluid resuscitation but CVP is only 7 mmHg (increased vascular capacity) = decreased preload and decreased afterload

- Increased Capillary Permeability: crackles in lungs, CVP only 7 mmHg (fluid shifts = decreased preload), requires mechanical ventilation (ventilation and gas exchange issues due to decreased compliance, V/Q mismatch, and impaired diffusion), slight edema in hands and feet

• Bradykinin (Cytokines): vasodilation: widened pulse pressure, low diastolic blood pressure, strong pedal pulses, brisk capillary refill, = decreased afterload and decreased preload; fever (interleukins act on the hypothalamus and adjust the temperature set point)

• Coagulation Cascade: low platelets, low hemoglobin, no INR/PTT available

• Complement System: intensifying inflammation effects in other systems, cell death will be occurring but not evident yet (this will be apparent in decreased organ function).

2. ABCDs of Sepsis Management:

• A: Assessment, Airway and Antibiotics

- Blood cultures and lactate drawn in ER as soon as sepsis is suspected

- SpO_2 monitored in ER, 100% oxygen by face mask administered

- Broad spectrum antibiotics started right away in ER following collection of blood and body fluid cultures.

• B: Breathing

- Intubated and mechanically ventilated to address poor ventilation and gas exchange; FiO_2 increased to address poor oxygenation; PEEP increased to address poor oxygenation (Note: it will be important to monitor airway pressures with the increase in PEEP)

• C: Cardiac Output

- Preload management: early fluid resuscitation in ER, followed by ongoing fluid management in ICU following CVP and physical assessment information

- Afterload management: Norepinephrine infusion with MAP goal established for adequate tissue perfusion at this time. Vasopressin may be added if needed.

- Contractility: managed via adequate preload management at the moment. Dobutamine is agent of choice if evidence of decreased preload.

• D: Drugs and Demand

- Morphine and midazolam infusions to achieve a set RASS goal in order to provide adequate analgesia and sedation, and reduce oxygen demand

- Mechanical ventilation also reduces oxygen demand

- No treatment for fever at this time

- Insulin infusion for glycemic control

- No stress ulcer prophylaxis established as yet

- Tube feeds initiated

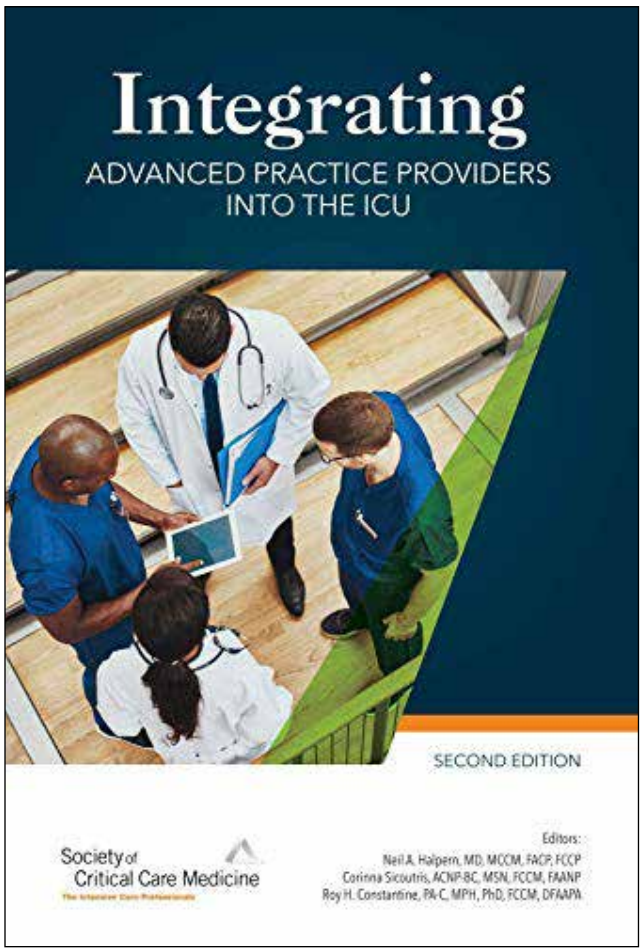
Book Review

Halpern, N.A., Sicoutris, C., & Constantine, R.H. (Eds.) (2019). *Integrating Advanced Practice Providers into the ICU*. Mount Prospect, IL: Society of Critical Care Medicine.

Critical care is an evolving specialty designed to care for patients during their most vulnerable times. As critical care medicine has progressed and grown over the past several decades, so has the team caring for these patients and their families. Most critical care units have developed an interdisciplinary team to ensure all patients have access to comprehensive, appropriate, and timely care focused on improving not only mortality, but also long-term patient outcomes. Nurse practitioners (NPs) or advanced care providers (APPs) have taken on active and evolving roles on the critical care team. Halpern, Sicoutris and Constantine (2019) have recently published the second edition of the Society of Critical Care Medicine's "*Integrating Advanced Practice Providers into the ICU*", a step-by-step guide on the implementation and integration of APPs into critical care. This edited collection provides a realistic guide to not only plan for the implementation of an APP, but also how to implement and evaluate the role, while also providing important insights on potential barriers that may be encountered.

Halpern, Sicoutris and Constantine built on the success of the first edition published in 2012 by engaging a wide range of experts including advanced practice nurses and physicians to provide input on this new edition. Notable experts that contributed in the writing of individual chapters included authors such as Kleinpell, Grabenkort, and Kapu. Kleinpell is an expert and lead researcher in the area of integrating APPs into critical care and was the original author of ground-breaking research published in 2008 that described the benefits of APPs in critical care (Kleinpell, Ely, & Grabenkort, 2008). Grabenkort has published work on APPs education, training and integration into practice (Grabenkort, Meissen, Gregg, & Coopersmith, 2017). Kapu has also published work describing the integration of nurse practitioners into the intensive care unit at Vanderbilt (Kapu, Thomson-Smith, & Jones, 2012). The variety of expertise engaged in the book provides a wholesome perspective on the benefits of APPs, and a logical stepwise approach for their successful integration into critical care areas.

The guide includes information on two different APPs: nurse practitioners and physician assistants, and while for the Canadian context nurse practitioners are the more common APP utilized, there is a growing number of physician assistants beginning to emerge in Canada. Each chapter provides a summary of an important topic that must be considered for the successful implementation, evaluation and sustainability of APPs. The writing style of the guide is such that the reader does not need to be an APP to understand key concepts but provides a realistic approach for the implementation of a new role, or information to consider improving upon an existing role. Key concepts such as understanding the benefit of the APP role are discussed and provide the reader the opportunity to consider why an organization may integrate the APP role as an adjunct



or alternative provider as a member of the interdisciplinary team. The book also highlights the importance of engaging all key stakeholders early to enable the team to build the role description, thereby ensuring that everyone understands how the new APP fits within the team and the expectations of the role. This early role definition also helps the organization ensure they are recruiting the best individual for the role.

Other key concepts covered includes the initial on-boarding process, fellowship training or previous experience required, the ongoing evaluation process of the APP and the role. APP specific topics of training (e.g., ventilator training, radiographic reading, etc.) and examples of outcome measures (e.g., expectations of when an APP may be independent with specific tasks/skills) are provided and aid in the planning for the introduction of an APP into critical care. Sample timelines and metrics—both patient-care related (e.g. readmission rates, length of stay, patient transfer, etc.) and hospital productivity (e.g. ,total number of patient encounters, cost savings, etc.) for organizational evaluation are also provided as a reference to help guide organizations in what to reasonably expect from this role.

The book is primarily written for an American audience and, thus, includes a chapter devoted to billing and one focused on credentialing and privileging. While the Canadian context of APP salary or financial reimbursement is very different from

the American model, it still provides information to consider about the relative cost of an APP compared to that of a traditional physician-dominated model of care. The chapter on credentialing, again while American focused, provides insight into processes that must be investigated and prepared for prior to the recruitment and implementation of an APP role. Other important topics that are covered include consideration of the critical care APP role utilization outside the walls of the ICU, in either role development/mentorship role, hospital and community outreach programs, and in research.

Overall, Halpern, Sicoutris and Constantine (2019) provide a detailed guide for organizations and/or individuals interested in planning or implementing an APP into their critical care team. It provides important information to help ensure the success of such a role, thereby enhancing the interdisciplinary team to provide optimal patient care.

About the reviewer

Sarah Crowe, MN, PMD-NP(F), NP, CNCC(C), is a Nurse Practitioner for the Fraser Health Authority. She is a current member of the CACCN Board of Directors holding the positions of National Treasurer, Co-Chair of the Conference Education and Evaluation Committee and Chair of the Dynamics of Critical Care Conference 2020. She is passionate about critical care and hopes to inspire others with her leadership.

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Research Review

Citation

Khan, N., Jackson, D., Stayt, L., & Walthall, H. (2019). Factors influencing nurses' intention to leave adult critical care settings. *Nursing in Critical Care, 24*(1), 24–32.

Background

A shortage of registered nurses persists globally; this shortage is particularly problematic among specialty nurses including critical care. Focusing on the retention of critical care nurses is essential in order to mitigate significant costs to the healthcare system (i.e., costs associated with turnover), as well as the direct impact shortages have on patient care (i.e., quality and safety).

Purpose of the Study

To explore factors that may influence nurses' intent to leave (ITL) adult critical care areas.

Research Approach and Methods

A mixed-methods systematic review of the literature modelled on the Joanna Brigg's Institute guidelines for systematic reviews (The Joanna Brigg's Institute, 2014).

Search Methods

A search strategy was developed using Lavender et al.'s (2016) List, Keep, and Delete approach and with the assistance of a librarian. Published literature from 2005-2016 was searched and included six databases (British Nursing Index (BNI); Cumulative Index to Nursing and Allied Health Literature (CINAHL); PubMed; PsychINFO; Embase and Health B Elite). Search terms and Medical Subject Headings (MeSH) were identified and listed by the authors. The search was limited to adult critical care settings. Inclusion criteria was identified as "international research published from 2005 in English looking at factors influencing nurses' ITL adult critical care areas" (Khan et al., 2019, p. 25). Exclusion criteria was specified as "[p]rimary data from non-critical care areas and neonatal and paediatric critical care" (Khan et al., 2019, p. 25).

Search Outcomes and Quality Appraisal

The final search outcome yielded 15 studies to be included in the literature review (n=2 qualitative studies; n=13 cross-sectional studies) (PRISMA flow diagram provided in original publication). Quality appraisal was completed by two authors independently using the National Institute for Health and Care Excellence qualitative and quantitative checklists (NICE, 2012a, 2012b). Narrative results of the quality appraisal were reported by the authors.

Data Extraction

Data extraction was completed by the first author and verified by the co-authors. Study details were extracted (e.g., authors, year of publication, country), as were findings reported in the primary studies that related to factors influencing nurses' ITL in adult critical care areas (Khan et al., 2019).

Synthesis

Data synthesis was completed using a thematic approach articulated by Braun and Clarke (2006).

Findings

Results reported from the quantitative data reflected 16,794 critical care nurses across 585 intensive units and 12 countries. Qualitative data were reflective experiences described by 24 critical care nurses. The following themes and subthemes were identified:

Theme 1 – Quality of work environment (subthemes: Social aspects, Organizational aspects, Physical aspects). Social aspects were the need for a supportive work environment that enable the nurses to voice their concerns. Organizational aspects were access to resources, professional development opportunities, structured career trajectory, relationship with nurse managers, organizational and systemic demands, and lack of recognition. Physical aspects included limited working space, constant noise, inadequate staffing and physical and emotional toll of the job.

Theme 2 – Nature of working relationships (subthemes: relationships with patients and relatives, relationships with manager and colleagues, relationships with medical colleagues). Positive encounters with patients and families were viewed as rewarding to nurses, while situations of conflict influenced nurses' ITL. Feelings of camaraderie and fellowship empowered nurses and created a healthy work environment, while negative experiences with colleagues and managers were considered stressful and increased ITL. The exclusion of nurses from decision-making discussions contributed to negative nurse-physician relationship and increased nurses' ITL.

Theme 3 – Traumatic and stressful workplace experiences. Workplace experiences such as those of caring for dying patients, withdrawing life-sustaining treatments, and futile care were viewed as stressful and increased nurses' ITL.

Commentary

Using a systematic approach to search and review the literature on the topic was an appropriate method to explore and synthesize what was currently known about nurses' ITL in adult critical care settings. A thematic approach facilitated succinct descriptions of identified themes and relevant subthemes on the topic. As is consistent with review methodology, a detailed account of the search methods, quality appraisal and data extraction were described. The authors note that a number of articles are excluded because they were "irrelevant records." However, it is unclear what this means. The authors provided a detailed discussion of the findings and also highlight strengths and limitations of the review from their perspective.

The introduction and background sections provided by the authors clearly situates the reader within relevant context and key literature on the topic. As a result, the need for a review is justified.

Interestingly, despite identifying that the review was based on The Joanna Briggs Institute methodology for mixed-methods systematic reviews, the authors did not incorporate Joanna Briggs Institute Quality Appraisal tools. While the NICE tools are commonly used in review methodology, there is no rationale provided regarding this decision. Additionally, there are no tables included that highlight the findings of the quality appraisal and the reader is left with only the brief narrative description provided by the authors.

Regarding the reporting of qualitative data, there are no illustrative quotes included in the findings. While not a mandatory requirement of Joanna Briggs Institute review methodology, the use of illustrative quotes can add important context to the review findings.

The results of this review provide greater insight into the factors that may contribute to nurses' ITL in adult critical care settings. Future reviews on the topic might consider comparing ITL with intention to stay. It could be potentially limiting to assume that an absence of factors contributing to nurses' ITL would translate into increased intention to stay. In order to address the issue of nursing shortage, concurrent efforts need to be taken to reduce nurses' intention to leave and to increase the intention to stay. The question we like to leave the readers with is: In your experience as a critical care nurse, what made you want to leave and what made you want to stay?

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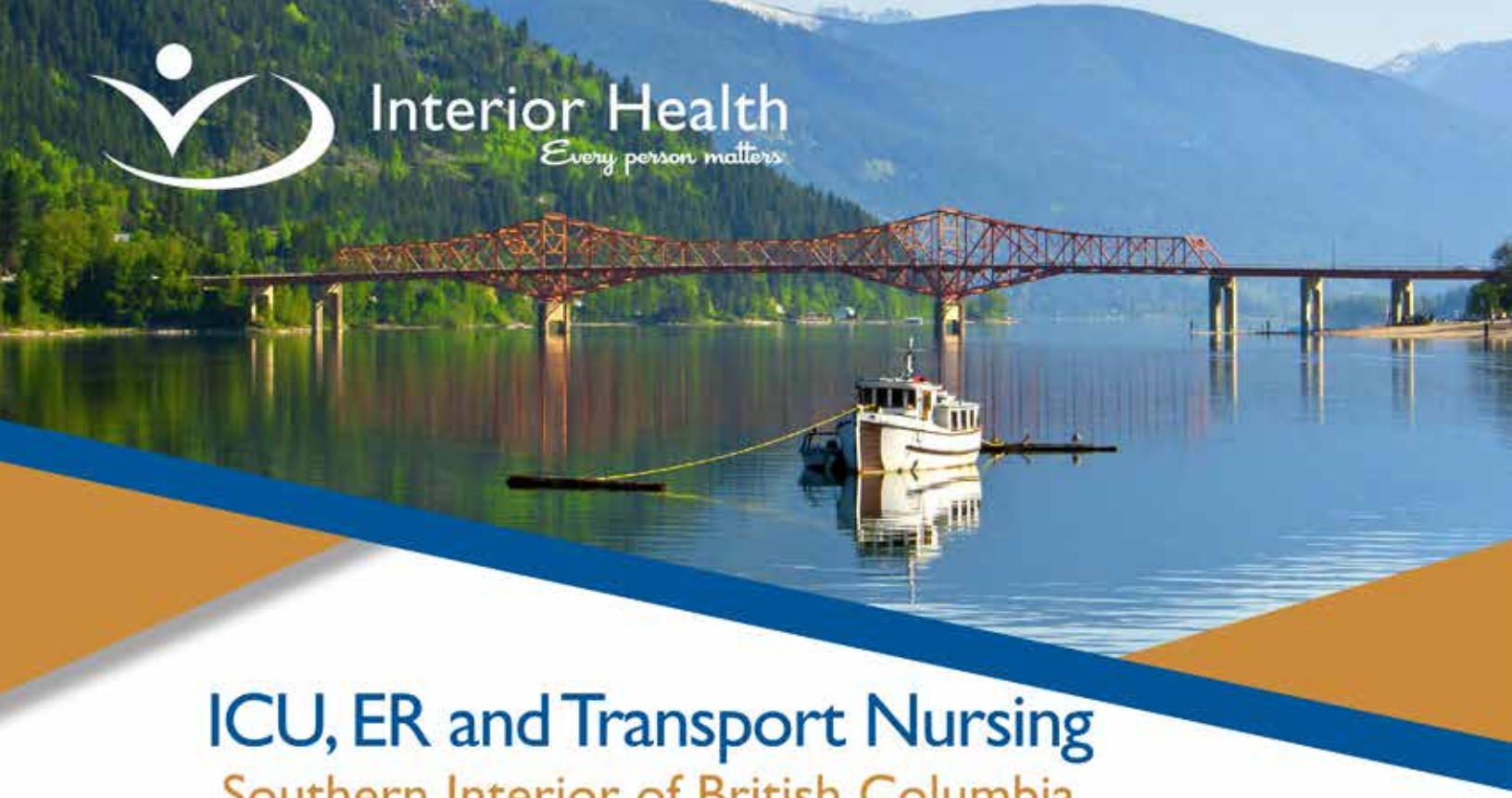

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