SHOCK TEAM AND RAPID RESPONSE SYSTEM

STaRRS

MODULE

EARLIER IDENTIFICATION AND TREATMENT OF AT-RISK PATIENTS

10 Signs of Vitality

KRITIKUS FOUNDATION

Society of Critical Care Medicine
The Intensive Care Professionals

Forward

A growing number of hospitals are implementing Rapid Response Systems (RRS) to reduce morbidity and mortality of at risk patients. Recognition and treatment of these patients is often delayed or inadequate. Increased awareness of delay in recognition and treatment of critically ill patients is responsible for the expansion of RRS and is driving initiatives of the Institute of Health Improvement (IHI), Society of Critical Medicine and Joint Commission to improve outcomes in this patient population. Joint Commission has mandated (2009) that hospitals implement solutions that recognize early and rapidly treat at risk patients. This manual, created by practicing experts in the field, has been developed to assist in implementing and/or improving an existing RRS.

Acknowledgment

This STaRRS Program was initially developed through the support of Kritikus Foundation, Redding Medical Center, Fremont-Rideout Health Group, Kaweah Delta Health Care District located in northern California. We wish to thank the Society of Critical Care Medicine for their confidence and support of this Rapid Response System Program and Laurie R. Grier, MD, Medical Director MICU, Professor of Medicine at Louisiana State University for the STaRRS acronym.

Authors

Frank Sebat, MS, MD, FCCM, FCCP
Director
Kritikus Foundation | Redding, California
Cardiovascular Intensive Care Unit | Visalia, California

Geoffrey K. Lighthall, MD, PhD
Assistant Professor
Anesthesia and Critical Care
Stanford University School of Medicine | Palo Alto, California

Ruth M. Kleinpell PhD. RN FAAN FAANP FCCM
Professor and Teacher Practitioner
Rush University Medical Center | Chicago, Illinois

Marie R. Baldisseri, MD, FCCM
Associate Professor of Critical Care Medicine
University of Pittsburgh Medical Center
648 Scaife Hall, 3550 Terrace Street | Pittsburgh, Pennsylvania

Randy S. Wax, MD, MEd, FRCPC
Medical Director
Program for Resuscitation Education and Patient Safety Mount | Sinai Hospital Assistant Professor and Clinician-Educator | Department of Medicine University of Toronto | Toronto, Canada

Deborah J. Shoffner
Administrator
Kritikus Foundation | Redding, California
Critical Care Research
Fremont-Rideout Health Group | Yuba City, California

Susan Henderson, MSN, NP, CCRN
Critical Care Educator and Director of Clinical Education
Fremont-Rideout Health Group | Yuba City, California

Michael Buist M.B.Ch.B
Director of Intensive Care
Dandenong Hospital | Melbourne, Australia
Associate Professor | Monash University | Melbourne, Australia

Bradford D. Winters, PhD, MD
Critical Care Medicine and Anesthesia
Johns Hopkins University School of Medicine
Medical Director | Adult Rapid Response Team program

Leland K. Beggs, MD
Medical Director of Critical Care and Performance Improvement
Kaweah Delta Health Care District Hospital | Visalia, California

Emma Camarena, RN, MSN, CNS, CCRN
Clinical Nurse Specialist-Critical Care Services
Kaweah Delta Health Care District Hospital | Visalia, California

Keith Jones, BSN, RN, PHN, CCRN
Clinical Educator for Critical Care
Kaweah Delta Health Care District Hospital | Visalia, California

Authors

Dr. Frank Sebat, MS, MD, FCCP, FCCM
Dr. Sebat is a Fellow of the American College of Critical Care Medicine and American College of Chest Physicians and a member of the Society of Critical Care Medicine Medical Emergency Team Task Force. He is currently the Medical Director of the Cardiovascular Intensive Care Unit at Kaweah Delta Health Care District Hospital, Visalia, CA and Kritikus Foundation, Redding, CA. He has published, lectured and consulted nationally on the early recognition and treatment of critical illness and he and his colleagues have developed and implemented programs to improve the care of these patients within the pre-hospital and hospital setting. Dr. Sebat brings an emphasis on “back to basics “in training health care professionals to recognize and treat at-risk patients. He refers to this as the Vince Lombardi (former coach of Green Bay Packers football team) approach to critical care, meaning that focusing on the basic “blocking and tackling” issues in a critically ill patient will improve outcomes more than the “razzle dazzle” interventions.

Geoffrey K. Lighthall, MD, PhD
Dr. Lighthall is an Assistant Professor for the Department of Anesthesia at Stanford University's School of Medicine in Stanford, CA, as well as Staff Physician in Intensive Care and Anesthesia at the Department of Veterans Affairs Medical Center in Palo Alto, CA. Dr. Lighthall started one of the first medical emergency teams in the VA system, and is active in assisting other hospitals with the development of rapid response systems and associated training programs. He is equally active in the development of critical care educational programs based on patient simulation. Geoff is a principal investigator on a number of studies concerning the role of simulation training in Critical Care education. Additionally, he developed the Stanford Critical Events Project which uses in-situ simulation to uncover latent errors in healthcare delivery systems.

Ruth Kleinpell PhD RN FAAN FCCM
Dr. Kleinpell is a fellow of the American Academy of Nursing, the American Academy of Nurse Practitioners, the Institute of Medicine of Chicago and the American College of Critical Care Medicine. She is currently a Professor at Rush University College of Nursing and a Teacher-Practitioner in Critical Care at Rush University Medical Center in Chicago, IL. In addition, she is a Nurse Practitioner at Our Lady of the Resurrection Medical Center in Chicago, IL. She is an active member of several critical care organizations including the Society of Critical Care Medicine, American College of Chest Physicians, and the American Association of Critical Care Nurses. She has published and presented on a number of clinical topics including implementation of rapid response teams.

Marie R. Baldisseri, MD, FCCM
Dr. Baldisseri is an Associate Professor of Critical Care Medicine at the University of Pittsburgh School of Medicine and is Chief of Critical Care Services at Magee-Women's Hospital in the University of Pittsburgh Health System. She completed a fellowship in Critical Care Medicine under Drs. Ake Grenvik and Peter Safar at the University of Pittsburgh Medical Center. She remained as faculty at the University of Pittsburgh and has served as the Medical Director of the Neurosurgical Intensive Care Unit and currently is the Medical Director of the ICU at Magee-Women's Hospital. She has been actively involved in the Society of Critical Care Medicine and presently serves as the Society's liaison to the Institute for Healthcare Improvement (IHI). She is the Chair of the Society's Fundamental Critical Care Support (FCCS) Steering Committee and has traveled extensively both nationally and internationally on behalf of the Society promoting the FCCS course. For the past three years, she served as Chair of the Society's International Task Force on Medical Emergency Teams and has published and lectured extensively on Medical Emergency Teams.

Randy S. Wax, MD, MEd, FRCPC
Dr. Wax is an Assistant Professor and Clinician-Educator in the Department of Medicine, University of Toronto. He works as a staff intensivist in the medical-surgical ICU, and is an attending physician on the "ACCESS" Critical Care Response Team, at Mount Sinai Hospital in Toronto. He is also Medical Director of the Program for Resuscitation Education and Patient Safety (PREPS), incorporating principles of adult education and simulation technology in teaching interprofessional resuscitation teams. Dr. Wax is the Course Director for the Critical Care Response Team course provided to hospitals throughout Ontario in conjunction with the Canadian Resuscitation Institute and the Ministry of Health and Long-term Care of the Province of Ontario. He also is the course co-director for the Rapid Response Team Training Course and member of the MET/RRS Task Force for the Society of Critical Care Medicine. Dr. Wax has been very active in assisting institutions and organizations that are developing and refining their approach to rapid response systems, having authored multiple textbook chapters and simulation learning cases, speaking frequently at international conferences, and providing consultative services.

Deborah J. Shoffner
Deborah is the Administrator of Critical Care Research, Fremont-Rideout Health Group, Marysville, CA and Kritikus Foundation, Redding, CA and is involved in process-centered medical informatics, graphics design, production of text and video nursing and physician education materials.

Susan Henderson, MSN, NP, CCRN
Susan is the Critical Care Educator and Director of Clinical Education at Fremont-Rideout Health Group Marysville, CA and continues to practice critical care nursing at the bed side. She has over 25 years experience as a Critical Care Nurse and received her MSN degree from UCLA while completing the Acute Care Nurse Practitioner program. Sue is a member of the American Association of Critical Care Nurses and Sigma Theta Tau. She is a clinical nurse practitioner specializing in critical care and neuro sciences. She was involved at the ground level in the development of the Ten Signs of Vitality and STaRRS and currently evaluating and upgrading these programs. She has lectured widely of this back to basics approach on earlier recognition and intervention of at-risk patients.

Michael Buist, MBChB, FRACP, FJFICM
Dr. Buist is a full time clinician in the disciplines of intensive care and internal medicine. He has published widely on the rationale and effectiveness of Rapid Response Teams.

Bradford D. Winters, PhD, MD
Dr. Winters is an Anesthesiologist and Critical Care physician in the department of anesthesiology and Critical Care Medicine at the Johns Hopkins University School of Medicine where he attends and teaches in the Cardiac Surgical, General Surgical and Neurological/Neurosurgical critical care units. He is the medical director of the hospital's Adult Rapid Response Team program and is pursuing research interests in patient safety and quality of health care delivery as a member of the Johns Hopkins Quality and Safety Research Group. He has published on effectiveness of RRS and is especially interested in technological solutions to early identification of deteriorating patients.

Leland K. Beggs, MD
Leland K Beggs received his medical degree from Wayne State University, Detroit, MI and his post graduate training at University of California Irvine Healthcare, Irvine, CA. He is the current medical director of Critical Care and Performance Improvement at Kaweah Delta Health Care District (KDHCD). He has served in these roles since 1988 and 1995 respectively. He was one of the developers of the current KDHCD rapid response system and is currently working on rapid response systems, process improvement projects to more rapidly identify and treat those patients with severe sepsis and septic shock.

Emma Camarena, MSN, RN, CNS, CCRN
Emma Camarena is the Clinical Specialist for Critical Care Services (ICU, CVICU, ICCU and ED) at Kaweah Delta Health Care District, Visalia, CA. She has over 12 years of critical care nurse and received her MSN-CNS degree from California State University, Fresno, CA. Emma is a member of the American Association of Critical Care Nurses and National Association of Clinical Nurse Specialist. Emma's role is translating evidenced-based research into bedside nursing practice.

Keith Jones, BSN, RN, PHN, CCRN
Keith Jones is the Clinical Educator for Critical Care Services (ICU, CVICU, ICCU and Centralized Monitoring) at Kaweah Delta Health Care District, Visalia, CA. He has over 6 years experience in his current role as the Clinical Educator and over 12 years of critical care nursing. He received his BSN-PHN degree from California State University, Bakersfield, CA. Keith is a member of the American Association of Critical Care Nurses and the Central California Critical Care Consortium. Keith's role is developing and implementing evidence based education programs, protocols, policies and procedures in critical care, advancing nursing education through didactic, high fidelity simulation, and bedside support. Keith has plays an intricate role in the development and education of Kaweah's Critical Care Nurse Intern (CCNI) program which transitions the non critical care nurse or newly graduated nurse into a successful, skilled critical care nurse.

Table of Contents

CHAPTER 1

INTRODUCTION

Frank Sebat, MS, MD, FCCP, FCCM
Bradford D. Winters, PhD, MD

Background and Need

A hospital-wide program that educates clinicians to identify early and rapidly treat life-threatening conditions, with a team response and protocols based upon best practice guidelines will improve outcomes.

Recognition and treatment of critical illness is often delayed or inadequate.[1, 2] This is due to insufficient knowledge of bedside nurses, respiratory therapists and other frontline (primary bedside providers) health care professionals needed to recognize various forms of critical illness early, lack of empowerment of frontline providers to start therapy independently, limited intensive care unit (ICU) /hospital resources and lack of institutional-wide systems to facilitate early recognition and rapid best-practice treatment of at-risk patients. [1-5] The therapeutic arsenal for severe sepsis, shock, and respiratory failure has grown in the last decade with the use of drotrecogin alpha, early goal-directed resuscitation for severe sepsis, low tidal volume ventilation, and conservative fluid management for acute lung injury.[6-8] Unfortunately, therapeutic advances often do not quickly translate into benefits at the bedside [9-14]. The delay between availability of proven therapies and their adoption by physicians or institutions can be attributed in part to the inability to keep abreast of new developments using evidence-based medicine and resistance to change due to ingrained patterns of behavior and lack of institutional systems that facilitate adoption of new best practice therapies.

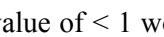
Significant alterations in vital signs, SpO_2, urine output and/or neurologic status in the field or the general hospital ward necessitate a call to and a response from the primary physician or consultant or the emergency department, delaying assessment and treatment. In addition, response from 'clinicians in training, is often delayed due to lack of experience or education in recognition and of treatment critical illness early.[15] The model of medical education historically has been notification of the least experienced member of the team, i.e. the intern, when a patient issue or crisis is identified. When that person perceives a lack of knowledge or skills, then the next least experienced physician is contacted and so on in a hierarchal fashion. In a medical crisis, this delay can lead to decompensation and disaster.

If critical illness is not recognized and treated during a narrow window of opportunity, tissue hypoxia develops and initiates a cascade of events leading to multi-organ failure and death. [4, 6] Fifty to eighty-four percent of in-hospital cardiac arrests are preceded by unappreciated physiologic instability, especially abnormalities of pulse rate, respiratory rate, mental status and SpO_2.(3, 4) In septic shock, mortality varies from 28 to 50%. Despite major advances in the therapeutic armamentarium, septic shock alone has been estimated to claim 90,000 lives per year in the United States. [16, 17] Primary respiratory failure and/or significant deterioration in neurologic status can quickly lead to hypoxia (hypoxic shock) followed by cardiovascular decompensation and arrest. Due to the high frequency and mortality of these clinical syndromes, a comprehensive systems-based approach to rapidly identify and treat these at-risk patients is only now evolving and is the basis for this manual.

A hospital-wide program that educates and assists clinicians in early identification and rapid treatment of life-threatening conditions, with a team response and protocols based upon best practice guidelines will improve outcomes. This approach has been successful in cardiac arrest, trauma and acute myocardial infarction (AMI) and stroke, where non-physician personnel identify patients, initiate frontline therapy and mobilize institutional resources. Patients with primary respiratory failure, deteriorating neurologic status or hemodynamic instability are a significant at-risk group who have benefited from this approach with

demonstrated decreased time to key interventions and mortality yearly over five years.[18] Rapid Response Systems (RRS) started with code blue teams utilizing protocol driven ACLS therapy. This approach evolved into organized early intervention systems i.e., trauma, acute myocardial infarction, stroke, and most recently medical emergency teams/rapid response teams (MET/RRT). A MET or RRT is a team led by a physician or nurse, respectively, usually activated by nurses, that quickly responds to evaluate and treat patients with clinical deterioration. Many observational reports and one single institution cluster-randomized study have shown that this approach reduces hospital cardiac arrests and mortality. In contrast, a recent multicenter, cluster-randomized trial (MERIT) showed no significant change in the incidence of cardiac arrest, unplanned ICU admissions or unexpected death.[19] Potential limitation of the MET/RRT approach include failure to change clinicians' behavior, limited effect of the team due to lack of standardized therapy, and the application to patients with a wide range of illness severity that might mask improvement in high-risk groups. Additionally, the afferent limb of the MET/RRT strategy (where the deterioration is identified based on "alert criteria") may not be optimal such that critical deteriorations are still being acted upon too late even with a MET/RRT service in place. The failure of the MERIT study to confirm the observational studies has been the subject of much discussion. Several potential factors have been identified including the short six months duration of the intervention phase not being adequate to influence hospital culture, the Hawthorne effect influencing the control hospitals and inadequate exposure of the population to the intervention.

While in theory a randomized cluster design with multiple centers is optimal for this type of intervention, in practice this design would be difficult to repeat, owing to the very broad implementation of RRS and their continued rapid growth. The Institute for Health Improvement, the Society of Critical Care Medicine and the American Heart Association have assisted in the implementation of a RRS in over 3000 hospitals in the U.S.[20, 21] These efforts, combined with the recent Joint Commission patient safety goals requirement for developing systems to better respond to deteriorating general ward patients that likely will further increase the implementation of such teams, results in making MET/RRTs nearly ubiquitous. Thus, enrolling a cohort of control patients in a multi-center trial free of bias may not be possible. In table 1 is a summary of the currently available studies on RRS. Nine of the ten show that the RRS or MET, a group of physicians and nurses activated by frontline personnel to promptly evaluate and treat deteriorating patients, led to either a reduction of in-hospital cardiac arrests or postoperative morbidity and mortality.[22, 23] However, the largest randomized multicenter trial showed no significant change in the incidence of cardiac arrest, unplanned ICU admissions or unexpected death.[19] Potential limitations of this study are discussed above. A recent RRS study focused on high-risk patients in shock led to a dramatically improved survival, decreasing mortality from 40% to11% by year five.[18]

The following tables (Tables 1, 2) and forest plots (Figures 1, 2) summarize the currently available RRS outcome studies. The forest plots combined effect of **similar studies** and show a 23% and 37% relative reduction in hospital death and cardiac arrest () with RRS implementation. In the forest plots a value of 1 would indicate no effect. A value of < 1 would be the relative reduction in risk of death or cardiac arrest. A value of > 1 would be the relative increase of these events.

Study, year		Country	Study Design	Relative Risk Reduction Mortality (CI 95%)	Relative Risk Reduction Cardio-respiratory Arrests (CI 95%)
Bristow,	2000 [24]	Australia	Concurrent cohort	1.06 (0.92-1.23)	0.94 (0.72-1.22)
Buist,	2002 [25]	Australia	historical	0.87 (0.73-1.04)	0.50 (0.35-0.72)
Bellomo,	2003 [22]	Australia	historical	0.74 (0.70-0.79)	0.35 (0.22-0.56)
Bellomo,	2004 [23]	Australia	historical	0.64 (0.45-0.92)	na
Kenward,	2004 [26]	UK	historical	0.99 (0.91-1.07)	0.92 (0.72-1.17)
Priestley,	2004 [27]	UK	Cluster-randomized	0.52 (0.32-0.85)	na
Devita,	2004 [28]	USA	historical		0.81 (0.71-0.93)
Garcea,	2004 [29]	UK	historical	0.52 (0.35-0.77)	na
MERIT,	2005 [19]	Australia	Cluster randomized	1.03 (0.83-1.27)	0.94 (0.79-1.13)
Jolley,	2007 [30]	USA	historical	1.00 (0.91-1.1)	0.86 (0.69-1.08)
Dacey,	2007 [31]	USA	historical	0.83 (0.73-0.95)	0.39 (0.27-0.57)
Mailey *	2006 [32]	USA	historical	na	na

Table 1. List of controlled trials of RRSs. Some trials were not included in Figure 1 secondary to inadequate numerator/denominator data *Mailey et al., 2006 [32] and Tolchin, 2007 [33] report significant reductions in mortality (25% and 19.2%, respectively) and cardio-respiratory arrest (30.6% by Tolchin) as well but their RRSs were part of a composite safety initiative that included other interventions so attributing the reductions solely to the RRS is not possible.

Long-term Studies, author, year		Country	Study Design	Reduction in Mortality (odds ratio)	Reduction in Cardio-respiratory Arrest (odds ratio or % reduction)
Jones,	2005 [34]	Australia	historical	na	0.47 (0.35-0.62)
Jones,	2007 [35]	Australia	Historical (surgical patients)	0.85 (0.69-1.05)	na
Jones,	2007 [35]	Australia	Historical (medical patients)	1.26 (1.13-1.39)	na
Buist,	2007 [36]	Australia	historical	na	73% over 6 years
Sebat*,	2007 [37]	USA	historical	0.15 by year 5 See legend	

Table 2. Long term Studies. Several studies have examined sustainability of reductions in mortality and cardio-respiratory arrest over several years. Jones et al., 2007 [35], reported on both medical and surgical patients over 4 years separately in their publication. For surgical patients years 1 and 3 exhibited statistically significant reductions, while years 2 and 4 had point estimates in favor of the RRS but did not reach significance. Medical patients actually had increased mortality all four years, though incidence of cardiac arrest was significantly decreased raising the question of sustainability of initially reported success. However, *Sebat et al., 2007 [37] used a specific disease subset of patients in shock and reported a significant reduction of 22% reduction of mortality odds ratio each year over five years and 28.2% (40% - 11.8%) absolute reduction in mortality by year five.

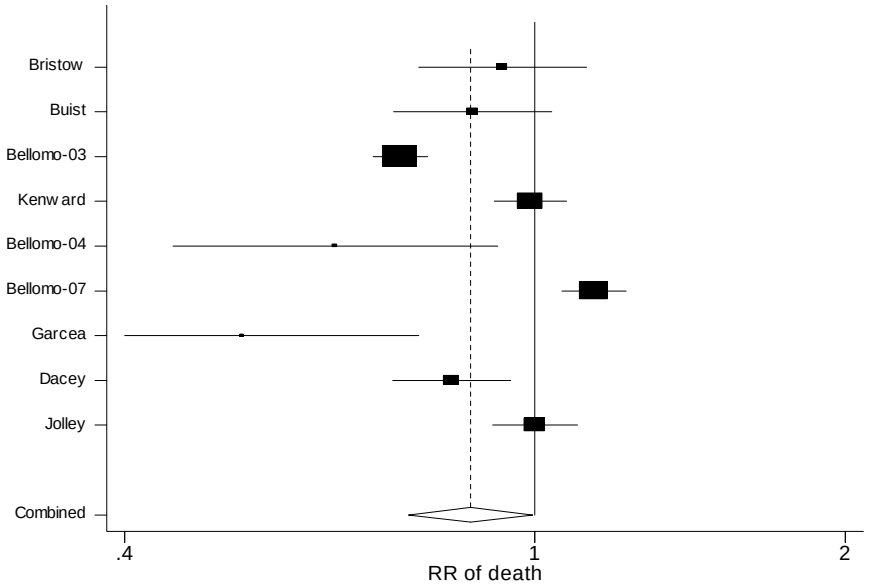


Figure 1. Meta-Analysis of Mortality: relative risk reduction with RRS in place: non-randomized studies. Combined relative risk of death with RRS in place is 0.866 95% CI=07.754-.995, p=0.042. Additional studies exist but do not provide appropriate numerator and denominator data to allow meta-analysis. Results calculated based on the random effects model.

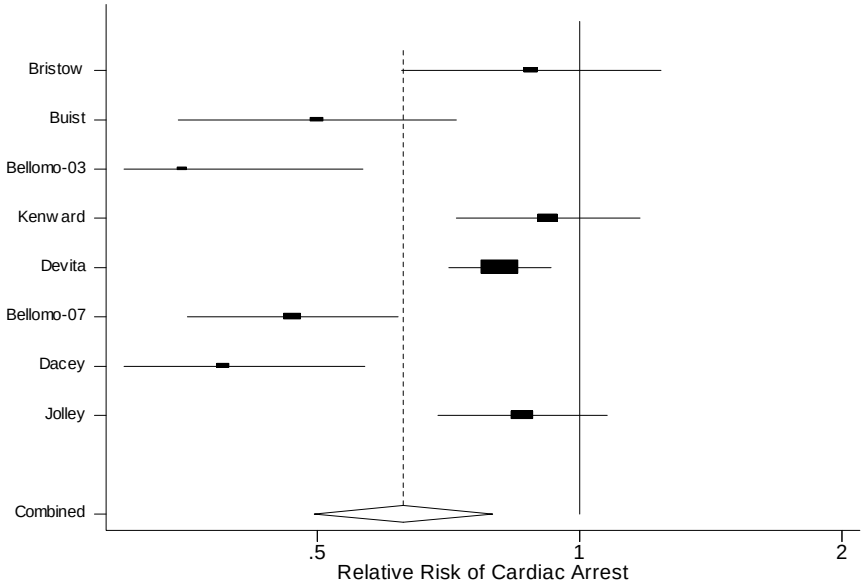
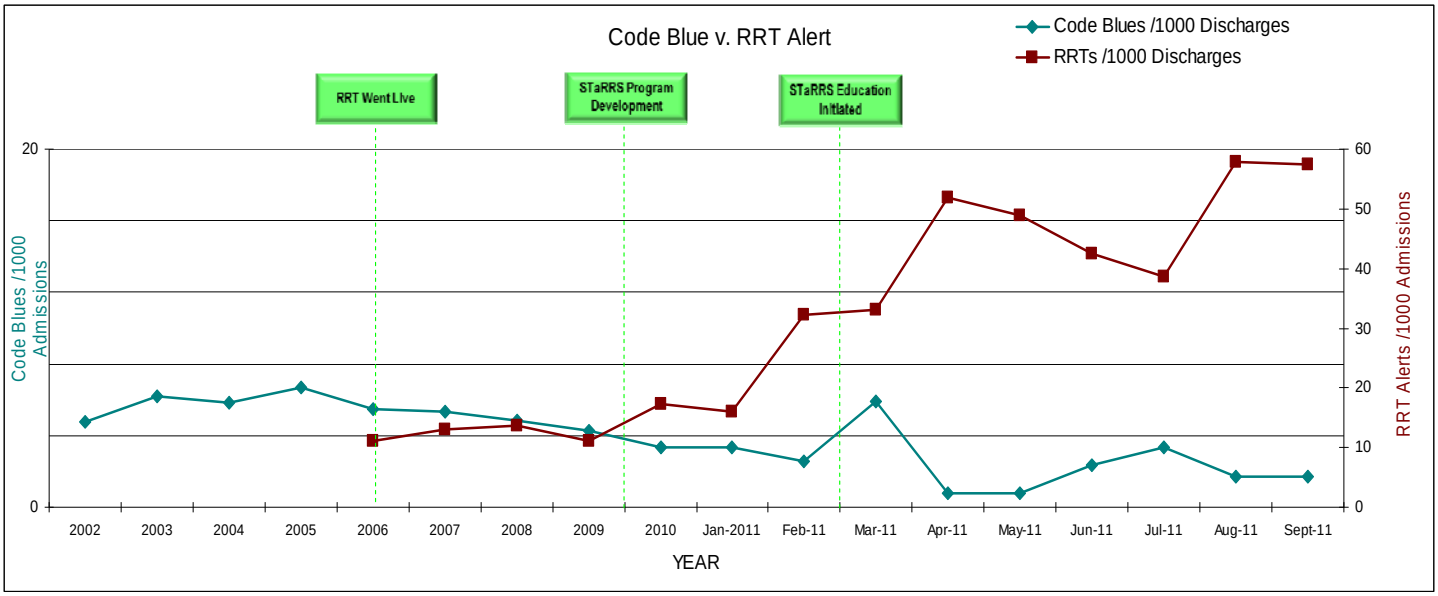


Figure 2. Meta-analysis of cardiac arrest: relative risk of cardiac and/or respiratory arrest with RRS in place. Combined relative risk of arrest is 0.628 95% CI=0.496-0.795, p=0.0001. Additional studies exist but do not provide appropriate numerator and denominator data to allow meta-analysis. Results calculated based on the random effects model.

Due to the prolonged time frame to conduct a large randomized trial, ethical concerns regarding randomization and barriers described above these studies may be the best data available for sometime to support continued expansion of RRS. Other types of RRS, i.e., Code blue, Trauma AMI and Stroke Teams, have been implemented based on less rigorous data and yet have saved countless lives. We believe this data is compelling enough that community hospitals as well as academic medical centers have the tools needed to implement a RRS designed to rescue earlier at-risk patients.

In the United States, between 4% and 16% of hospitalized patients suffer an adverse event many of which are preventable. [1, 18] As a result of this and available outcome data, Joint Commission in 2009 required all hospitals to have a system in place to recognize and rapidly address the needs of at-risk patients.

The STaRRS Program at Kaweah Delta Medical Center was developed in 2010 with education for all the medical / surgical floor nurses completed in May 2011. The graph below illustrates the benefit of bedside provider education focused on earlier recognition of at-risk patients using an expanded set of vital signs—the 10 SOV. Consistent with prior studies, RRS alerts quadrupled with a decrease in the incidence of code blues by more than half. It is expected that hospital mortality and unplanned transfers to higher level of care will decrease over time as provider adoption of this program increases.



References

1. Sebat F, Johnson D, Musthafa AA, Watnik M, Moore S, Henry K, Saari M: A multidisciplinary community hospital program for early and rapid resuscitation of shock in nontrauma patients. *Chest* 2005, 127(5):1729-1743.
2. Lundberg JS, Perl TM, Wiblin T, Costigan MD, Dawson J, Nettleman MD, Wenzel RP: Septic shock: an analysis of outcomes for patients with onset on hospital wards versus intensive care units. *Crit Care Med* 1998, 26(6):1020-1024.
3. Garrard C, Young D: Suboptimal care of patients before admission to intensive care is caused by a failure to appreciate or apply the ABCs of life support. *Bmj* 1998, 316(7148):1841-1842.
4. Buist MD, Jarmolowski E, Burton PR, Bernard SA, Waxman BP, Anderson J: Recognising clinical instability in hospital patients before cardiac arrest or unplanned admission to intensive care. A pilot study in a tertiary-care hospital. *Med J Aust* 1999, 171(1):22-25.
5. Carcillo JA, Davis AL, Zaritsky A: Role of early fluid resuscitation in pediatric septic shock. *Jama* 1991, 266(9):1242-1245.
6. Rivers E, Nguyen B, Havstad S, Ressler J, Muzzin A, Knoblich B, Peterson E, Tomlanovich M: Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med* 2001, 345(19):1368-1377.
7. Annane D, Bellissant E, Bollaert PE, Briegel J, Keh D, Kupfer Y: Corticosteroids for severe sepsis and septic shock: a systematic review and meta-analysis. *Bmj* 2004, 329(7464):480.
8. Bernard GR, Vincent JL, Laterre PF, LaRosa SP, Dhainaut JF, Lopez-Rodriguez A, Steingrub JS, Garber GE, Helterbrand JD, Ely EW *et al*: Efficacy and safety of recombinant human activated protein C for severe sepsis. *N Engl J Med* 2001, 344(10):699-709.
9. McGlynn EA, Asch SM, Adams J, Keesey J, Hicks J, DeCristofaro A, Kerr EA: The quality of health care delivered to adults in the United States. *N Engl J Med* 2003, 348(26):2635-2645.
10. Kalhan R, Mikkelsen M, Dedhiya P, Christie J, Gaughan C, Lanken PN, Finkel B, Gallop R, Fuchs BD: Underuse of lung protective ventilation: analysis of potential factors to explain physician behavior. *Crit Care Med* 2006, 34(2):300-306.
11. Berwick DM: Disseminating innovations in health care. *Jama* 2003, 289(15):1969-1975.
12. Asch SM, Kerr EA, Keesey J, Adams JL, Setodji CM, Malik S, McGlynn EA: Who is at greatest risk for receiving poor-quality health care? *N Engl J Med* 2006, 354(11):1147-1156.
13. Weinert CR, Gross CR, Marinelli WA: Impact of randomized trial results on acute lung injury ventilator therapy in teaching hospitals. *Am J Respir Crit Care Med* 2003, 167(10):1304-1309.
14. Young MP, Manning HL, Wilson DL, Mette SA, Riker RR, Leiter JC, Liu SK, Bates JT, Parsons PE: Ventilation of patients with acute lung injury and acute respiratory distress syndrome: has new evidence changed clinical practice? *Crit Care Med* 2004, 32(6):1260-1265.
15. Smith GB, Poplett N: Knowledge of aspects of acute care in trainee doctors. *Postgrad Med J* 2002, 78(920):335-338.
16. Angus DC, Wax RS: Epidemiology of sepsis: an update. *Crit Care Med* 2001, 29(7 Suppl):S109-116.
17. Wenzel RP: Treating sepsis. *N Engl J Med* 2002, 347(13):966-967.
18. Sebat F, Musthafa AA, Johnson D, Kramer AA, Shoffner D, Eliason M, Henry K, Spurlock B: Effect of a rapid response system for patients in shock on time to treatment and mortality during 5 years*. *Crit Care Med* 2007.
19. Hillman K, Chen J, Cretikos M, Bellomo R, Brown D, Doig G, Finfer S, Flabouris A: Introduction of the medical emergency team (MET) system: a cluster-randomised controlled trial. *Lancet* 2005, 365(9477):2091-2097.
20. Berwick DM, MD, MPP: Redesigning Hospital Care. In: *2nd Annual International Summit on Redesigning Hospital Care: 2006; Atalanta, GA*; 2006.
21. Baldisseri MR, MD; Teres, Daniel, MD, FCCM: Rapid Response System Training. In: *Society of Critical Care Medicine: Excellence in Qualtiy and Safety in Critical Care: 2006; Baltimore, Maryland, USA*; 2006.
22. Bellomo R, Goldsmith D, Uchino S, Buckmaster J, Hart GK, Opdam H, Silvester W, Doolan L, Gutteridge G: A prospective before-and-after trial of a medical emergency team. *Med J Aust* 2003, 179(6):283-287.
23. Bellomo R, Goldsmith D, Uchino S, Buckmaster J, Hart G, Opdam H, Silvester W, Doolan L, Gutteridge G: Prospective controlled trial of effect of medical emergency team on postoperative morbidity and mortality rates. *Crit Care Med* 2004, 32(4):916-921.
24. Bristow PJ, Hillman KM, Chey T, Daffurn K, Jacques TC, Norman SL, Bishop GF, Simmons EG: Rates of in-hospital arrests, deaths and intensive care admissions: the effect of a medical emergency team, vol. 173; 2000.
25. Buist MD, Moore GE, Bernard SA, Waxman BP, Anderson JN, Nguyen TV: Effects of a medical emergency team on reduction of incidence of and mortality from unexpected cardiac arrests in hospital: preliminary study. *Bmj* 2002, 324(7334):387-390.
26. Kenward G, Castle N, Hodgetts T, Shaikh L: Evaluation of a medical emergency team one year after implementation. *Resuscitation* 2004, 61(3):257-263.
27. Priestley G, Watson W, Rashidian A, Mozley C, Russell D, Wilson J, Cope J, Hart D, Kay D, Cowley K *et al*: Introducing Critical Care Outreach: a ward-randomised trial of phased introduction in a general hospital. *Intensive Care Med* 2004, 30(7):1398-1404.
28. DeVita MA, Braithwaite RS, Mahidhara R, Stuart S, Foraida M, Simmons RL: Use of medical emergency team responses to reduce hospital cardiopulmonary arrests. *Qual Saf Health Care* 2004, 13(4):251-254.
29. Garcea G, Thomasset S, McClelland L, Leslie A, Berry DP: Impact of a critical care outreach team on critical care readmissions and mortality. *Acta Anaesthesiol Scand* 2004, 48(9):1096-1100.
30. Jolley J, Bendyk H, Holaday B, Lombardozzi KA, Harmon C: Rapid response teams: do they make a difference? *Dimens Crit Care Nurs* 2007, 26(6):253-260; quiz 261-252.
31. Dacey MJ, Mirza ER, Wilcox V, Doherty M, Mello J, Boyer A, Gates J, Brothers T, Baute R: The effect of a rapid response team on major clinical outcome measures in a community hospital. *Crit Care Med* 2007, 35(9):2076-2082.

32. Mailey J, Digiovine B, Baillod D, Gnam G, Jordan J, Rubinfeld I: Reducing hospital standardized mortality rate with early interventions. *J Trauma Nurs* 2006, 13(4):178-182.
33. Tolchin S, Brush R, Lange P, Bates P, Garbo JJ: Eliminating preventable death at Ascension Health. *Jt Comm J Qual Patient Saf* 2007, 33(3):145-154.
34. Jones D, Bellomo R, Bates S, Warrillow S, Goldsmith D, Hart G, Opdam H, Gutteridge G: Long term effect of a medical emergency team on cardiac arrests in a teaching hospital. *Crit Care* 2005, 9(6):R808-815.
35. Jones D, Opdam H, Egi M, Goldsmith D, Bates S, Gutteridge G, Kattula A, Bellomo R: Long-term effect of a Medical Emergency Team on mortality in a teaching hospital. *Resuscitation* 2007, 74(2):235-241.
36. Buist M, Harrison J, Abaloz E, Van Dyke S: Six year audit of cardiac arrests and medical emergency team calls in an Australian outer metropolitan teaching hospital. *Bmj* 2007, 335(7631):1210-1212.
37. Sebat F, Musthafa AA, Johnson D, Kramer AA, Shoffner D, Eliason M, Henry K, Spurlock B: Effect of a rapid response system for patients in shock on time to treatment and mortality during 5 years. *Crit Care Med* 2007, 35(11):2568-2575.

CHAPTER 2

PHYSIOLOGY OF SHOCK STATES

Geoffrey K. Lighthall, MD, PhD
Marie R. Baldisseri, MD, FCCM
Frank Sebat, MS, MD, FCCP, FCCM

Many hospital deaths are *preventable.* In vulnerable patients subtle *early* events set into motion a critical interruption of oxygen delivery to multiple vital organs. This O_2 supply/demand mismatch, leading to multiple organ hypoxia and dysfunction, is defined as shock. Although shock is traditionally considered a consequence of hemodynamic instability, oxygen delivery can also be impaired by problems related to severe anemia, airway compromise or respiratory failure. This chapter describes the key pathophysiologic derangements underlying shock states and their progression to organ dysfunction and failure. Different shock types will also be presented. Understanding the pathophysiology of shock provides an appreciation for the need of early recognition and rapid treatment to interrupt the shock cycle, the foundation of any RRS.

Shock:

Hypovolemic
Septic or Distributive
Cardiogenic
Obstructive
Anaphylactic
Hypoxic

Common pathways of shock

Shock is a result of the body's inability to get oxygen in sufficient quantities to the vital organs resulting in tissue hypoxia. Secondary events of all shock states include to a lesser or greater extent, systemic inflammation and activation of coagulation resulting in microcirculatory stasis. Tissue hypoxia, whether due to inability to protect the airway, lung disease or decreased blood flow to vital tissues, reduces the energy yield of carbohydrate metabolism and hence the energy available to maintain cellular function. The vast majority of critical illness events evolve from alterations in oxygen availability, delivery and utilization. The overarching concept of tissue perfusion, while fundamental to the pathophysiology of shock and proper resuscitation, is often hard to define, and often lacks a direct measure early in the process. At its core, however, are two key physiologic relationships that can and should be evaluated at the bedside of every patient you encounter:

1. The relationship between the body's oxygen supply and demand.
2. The adequacy of blood pressure and blood flow through key organs.

O_2 Arterial Content
- *SaO_2% x 1.34ml O_2/dl x Hb gm/dl (normal 15-18 mlO_2/dl)*

O_2 Delivery
- *O_2 content x CO/L/min x 10 (normal 750-900 ml)*

O_2 Extraction Ratio
- *O_2 tissue consumption / O_2 delivery (normal 25%)*

O_2 Mixed Venous Saturation
- *Arterial O_2 Sat minus % extracted (normal 65-75%)*

1. Oxygen Supply and Demand.

Mammalian evolution has delivered an efficient means of harvesting energy in the form of oxidative metabolism and its generation of ATP. Oxygen drives (serves as an electron acceptor) the mitochondrial respiratory chain, providing nearly ten times the energy yield of anaerobic metabolism. Without this additional energy, cellular functions such as active transport of molecules across membranes, production of proteins and cell integrity cannot be maintained.

Oxygen supply to the tissues is dependent on arterial O_2 Hb saturation, quantity of hemoglobin (O_2 content) and cardiac output. Dissolved oxygen is usually negligible compared to that bound by hemoglobin. Thus with an adequate Hb, much of the discussion of oxygen supply and demand revolves around the evaluation of the oxyhemoglobin saturation of arterial and mixed venous blood. In a healthy resting state, there is an excess of oxygen delivery compared to demand, which in most circumstances means that only 25% of the blood's oxygen is consumed. The corresponding central venous saturation (blood returning to the right side of the heart) is therefore approximately 75%. Central venous saturation in principle can serve as a dynamic measure of oxygen supply versus demand. Accordingly, normalization of central oxygen saturation has become an accepted goal in resuscitation of patients with septic shock.

Arterial O_2 saturation, quantity of hemoglobin, and cardiac output determine the supply of oxygen to the tissues. Metabolic and physical activities determine oxygen demand. The O_2 supply/demand relationships are demonstrated in Figure 1. As discussed above, oxygen consumption is generally not limited by supply; this "luxury perfusion" is represented by the flat or supply independent zone of the graph. When O_2 delivery does not meet demand, represented by the sloping parts of the graph, anaerobic metabolism and cell injury result. Uncorrected shock states are in the pathologic supply dependent region. Increases in metabolic state or vigorous exercise can also cause the curve to shift upwards to much higher levels of oxygenation consumption (VO_2) and therefore leave the supply independence zone and lead to a lactic acidosis.

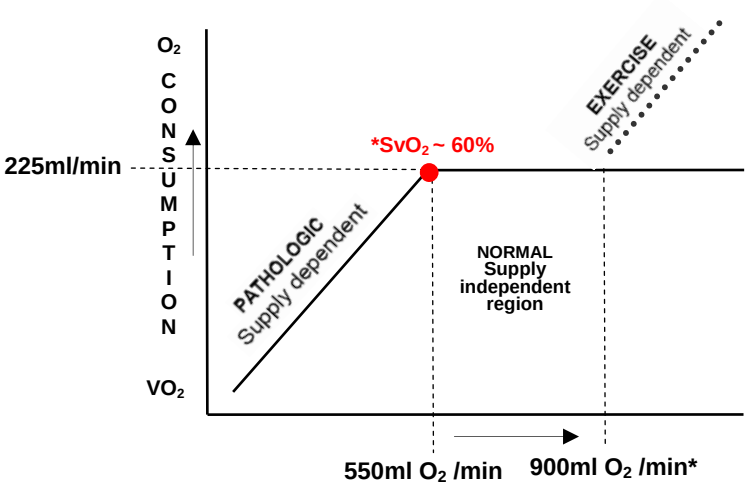


Figure 1. The relationship between oxygen utilization (VO_2) and oxygen delivery (DO_2) and tissue hypoxia).
The shoulder ● of the graph divides the supply independent (normal) and supply dependent (pathologic) regions of tissue oxygen consumption. As delivery decreases in the supply-independent region, oxygen extraction increases so that oxygen utilization can remain constant. When delivery crosses the critical threshold and into the supply dependent region, metabolism becomes anaerobic and lactic acid accumulates. At the left shoulder ● O_2 delivery has decreased to 550 ml/min and consumption has remained at 225ml/min (225/550=60% O_2 Hb sat) at which point continued drop of O_2 supply makes further O_2 extraction difficult and results in tissue hypoxia. *5,000ml/min blood Hb 14 gm/100ml blood x 96 % O_2 sat x 1.34 ml O_2 /100 ml blood = 900 ml O_2/min delivered.

The derivation of oxygen delivery is hemoglobin level, arterial oxygen saturation and cardiac output. When one suspects that oxygen delivery is decreased, the problem is due to one or more of the following problems: inadequate hemoglobin (unable to carry oxygen); inadequate arterial oxygenation saturation (airway compromise, respiratory failure); or inadequate cardiac output (not enough forward flow or pressure to get oxygenated hemoglobin to vital organs). Any significant perturbation to systems controlling these parameters can lead to potentially catastrophic drops in oxygen delivery and subsequent organ compromise. Earlier manifestations of inadequate tissue oxygen delivery include decreased SaO_2, anxiety, apathy, lethargy, tachypnea, prolonged capillary refill, oliguria, EKG changes, increased base deficit on an ABG (decreased bicarbonate) or increased levels of lactic acid. A late and often irreversible manifestation of tissue hypoxia is multi-organ failure.

It is less common, but no less important to consider increased O_2 demand as a contributing factor in shock. For example, increased work of breathing can create significant diversion of blood flow from other key organs to the diaphragm and intercostal muscles. Likewise, energy consumption increases 8% for each degree of temperature increase (Celsius). Other causes of increased O_2 demand are seizures, vigorous physical activity seen in delirium and thyrotoxicosis. In the early phases of shock, excessive work of breathing is generally under-recognized and undertreated. The other causes of excessive O_2 demand are generally recognized and addressed.

2. Blood Pressure

Blood pressure is a highly regulated parameter influenced by fluid shifts and activity of renal, endocrine, and autonomic nervous systems. Regulation of tissue perfusion depends on maintaining blood pressure within an adequate range; however, this range differs between individual patients. This autoregulation of tissue perfusion is diagrammed in Figure 2. It is important to consider that with age, preexisting hypertension, and vascular disease, the autoregulatory zone for a given patient is likely to be shifted to the right as shown by the red line. Thus knowing a patient's baseline or outpatient blood pressure is important to appropriately interpreting whether a blood pressure measurement is "normal" or sufficient for that patient.

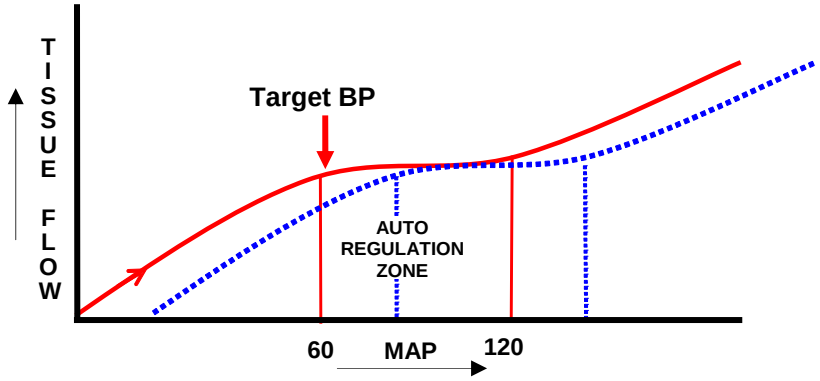


Figure 2. Autoregulation of tissue blood flow vs. blood pressure. Despite changes in blood pressure, blood flow to tissues can remain constant as long as the blood pressure remains within a certain range. This phenomenon is known as autoregulation. The autoregulatory curve for a normotensive heart is depicted in blue. In contrast, patients with hypertension have rightwardly shifted curves (red); early clinical recognition of decreased organ perfusion are often the only clues to an inappropriately low blood pressure for an individual patient, despite being in the "normal" range for the general population.

Abnormalities in blood pressure, principally low pressures, may have several possible explanations:

A. Pathologic hypotension secondary to decreased cardiac output or vascular resistance resulting from disease or iatrogenic factors.
B. Population variation—some people have normal low pressure with adequate tissue perfusion.
C. Therapeutic targeting—many patients with systolic heart failure are given agents that reduce systemic vascular resistance (afterload), lowering systolic pressures to the 90s in an effort to improve stroke volume and cardiac output. In this case, cardiac output increases so that tissue perfusion is improved in spite of lower blood pressure.

A mean arterial pressure <60 mmHg should be considered inappropriately low unless patient history confirms a chronically well tolerated low blood pressure AND current patient assessment demonstrates no signs of inadequate tissue perfusion. Although a mean arterial pressure >60 mmHg is usually adequate to perfuse vital organs, one must always be vigilant for historical features suggesting a shifted autoregulatory curve OR clinical signs of hypoperfusion. False reassurance due to a blood pressure >60 mmHg is a common pitfall for health care providers leading to delayed recognition and treatment of shock.

A. Falsely concluding that "everything is okay." Even though the blood pressure is noted to be low, or worse is low but unrecognized due to measurement error, the health care provider does not perform an exhaustive search for signs of tissue hypoperfusion. Patients in shock frequently do not exhibit all the signs and symptoms of shock. Clinicians who are overly optimistic will focus on the absence of some signs and symptoms of shock while ignoring those that are present.
B. Failure to consider the patient's baseline. Blood flow through key organs is relatively constant over a wide range of pressures, but that *range* can shift to higher values in patients who are older, and particularly in patients with chronic or uncontrolled hypertension and peripheral vascular disease.
C. A mean BP <60 will result in decrease coronary artery perfusion leading to myocardial dysfunction, further reducing coronary artery perfusion. Thus regardless of the initial cause, all uncorrected shock eventually develops a component of cardiogenic shock

These errors can be prevented with some fundamental changes in the approach to hypotension. One should take abnormal vital signs seriously, and assume that *something is definitely wrong until proven otherwise.* "Everything is okay" becomes the diagnosis of exclusion rather than an early assumption. An exhaustive assessment of organ perfusion should always be conducted when hypotension is noted including: neurologic status; respiratory rate; capillary refill; ST segment changes; urine output; changes in serum creatinine; presence of bowel sounds; and measurement of base deficit on an ABG or lactic acid. Hypotension should be rapidly reversed if there is evidence of end organ hypoperfusion

Most to least accurate blood pressure measurement:

Arterial line
Doppler with manual cuff
Occultation with manual cuff
Automated blood pressure cuff

Determining whether a given blood pressure for a patient is adequate is one thing; measuring blood pressure accurately is another. Determination of blood pressure by a non-invasive automated cuff in at-risk or unstable patients is frequently inaccurate. The commonly used Dynamap is error prone when there is low stroke volume or increased vascular resistance and frequently over-estimates BP in this setting. Hypotension is frequently missed by automated blood pressure cuffs in at-risk patients due to increased vascular resistance from endogenous catecholamines or from decreased stroke volume from hypovolemia or myocardial dysfunction. A second confirmatory method of BP measurement in an at-risk patient is mandatory.

An integrated picture of blood pressure and flow

Blood pressure (MAP) is a function of the cardiac output (CO) and the systemic vascular resistance (SVR) as represented in the equation below.

$$MAP = CO \bullet SVR$$

Accordingly, not all blood pressures are created equally; one can have a MAP of 60 that derives from any of the scenarios listed below in Table 1.

Table 1

	MAP* (80-110)	**CO** (3-6 L/min)	**CVP** (0-4)	**SVR** (800-1200)	**Examples** (normal range)
1	60	2	20	1600	cardiogenic shock
2	60	8	3	570	septic shock
3	60	2	2	2320	hemorrhage, hypovolemia

* MAP, mean arterial pressure (units=mmHg); CO, cardiac output; CVP, central venous pressure (units=mmHg); SVR, systemic vascular resistance (units =dyn·s/cm5), calculated using SVR = [(MAP-CVP)/CO] * 80

It is important to consider not only the numerical value of the blood pressure, but how the body is creating that pressure. For example, chest compressions in CPR can briefly generate high arterial pressure (SBP >150mmHg), but produces only minimal forward blood flow (i.e., CI ~0.8 L/min). Conversely, in a partially resuscitated septic patient, blood flow can be significantly elevated (CO >8 L/min) while a vasodilated state renders him hypotensive and unable to perfuse vital tissue beds (i.e., MAP <50mmHg).

Accordingly, a normal blood pressure does not assure that *perfusion* is normal. This underscores the point that *each patient evaluation is incomplete without deliberate consideration of (1) the oxygen supply / demand relationship, and (2) adequacy of the components of blood pressure CO and SVR.* For example, consider a penetrating trauma victim with a blood volume loss of 25-30%. The blood pressure may be normal in this supine patient, but with loss of hemoglobin, low cardiac preload and output, and an extremely high vascular tone (example 3 in Table 1), this patient's effective blood flow and tissue oxygen delivery are dangerously low.

If measured, this patient's global oxygen deficit will likely be detected by an elevated serum lactate. Diagnosing the low cardiac output state can be facilitated by noting the combined findings of delayed capillary refill, narrow pulse pressure (when HR is normal or elevated) and low urine output, as well as increased respiratory rate, altered mental status, and decreased $ScvO_2$ or new metabolic acidossis. While classic parameters of cardiovascular physiology are often used to define and describe shock states, invasive devices and direct measurement of these parameters are not nearly as valuable as formulating the right questions and thoughts at the time of bedside evaluation.

In chapter 3, we will provide a detailed introduction to the Ten Signs of Vitality (10 SOV) as a framework for evaluating patients at risk for decompensation. All of these signs relate to evaluating the important concepts presented in this chapter, mainly: (1) oxygen supply versus demand, and (2) blood pressure and flow. The 10 SOV are listed below along with their relationships to the physiologic relationships just mentioned.

10 Important Clinical Signs	Relevance to The Pathophysiology of Shock
Temperature	Increased metabolic demand and oxygen consumption in the setting of poor supply
Heart rate	Increased heart rate reflects a compensatory mechanism for poor supply or increased demand for oxygen
Pain	Sympathetic stimulation, increased oxygen consumption, risk for overall low oxygen delivery
Respiratory rate	Increased respiratory rate due direct respiratory center stimulation by SIRS related factors, increased CO_2 production , increased demand for oxygen or metabolic acidosis
Arterial oxygen saturation	Low O_2 sat leads to decreased oxygen delivery − more critical in setting of high tissue demand
Blood pressure	If decreased, can compromise organ blood flow
Level of consciousness	A sensitive indicator of brain perfusion
Capillary refill	Corresponds to peripheral skin perfusion; slow refill often suggests high vascular peripheral tone in setting of poor cardiac output
Urine output	Often a sensitive indicator of abdominal organ perfusion
$ScvO_2$ or base deficit or lactic acid	Decrease in $ScvO_2$ or increase in BE or LA reflects imbalance of oxygen supply (CO, Hb, SaO_2) to demand (temperature, work of breathing, increase muscle activity)

An abnormality in one of the 10 SOV should trigger an assessment of all ten. The conclusion that the patient does not have a significant O_2 supply/demand mismatch then becomes a diagnosis of exclusion. The burden of proof is on the clinician.

Organ Dysfunction Resulting from Shock

Shock, as a result of inadequate arterial oxygen content, blood pressure or cardiac output leads to vital organ dysfunction. Regardless of the initial cause of shock, the end results are multi-organ dysfunction, failure and death. It is important to stress that all shock eventually leads to decreased coronary artery perfusion and myocardial depressant factors (i.e., in sepsis) that result in cardiogenic shock (Figure 3). It is not uncommon to see healthy athletes or young people with a decrease in left ventricular ejection fraction (i.e., from 65% to 30%) as a result of septic shock or severe prolonged hypotension.

In all forms of shock, tissue factors are released that activate the inflammatory and coagulation pathways. This cascade of events triggered by tissue hypoxia is less severe in the case of hypoxic, hypovolemic or cardiogenic shock as compared to distributive shock associated with the systemic inflammatory response syndrome (SIRS). Tissue factors released in SIRS due to pancreatitis, trauma, burns, infection, etc., cause fever, tachypnea, decrease in systemic vascular resistance with hypotension, increase in WBCs and/or bands (immature white cells) and organ dysfunction. SIRS is a potent activator of the inflammatory and coagulation pathways resulting in vascular endothelial injury and capillary leak resulting in increased third space fluid, depletion of intravascular volume and microvascular stasis. Sepsis, defined as SIRS triggered by infection, is associated with increased inflammation and microvascular coagulopathy, due to the release of tissue factors from injured endothelial cells and toxins from the bacterial cell wall (endotoxins), cytoplasm (exotoxin, toxic shock) or both. This leads to capillary leak, severe third spacing and microvascular stasis. These problems are compounded by increased oxygen demand at a time when volume deficits due to third spacing compromise cardiac output and hence decrease oxygen delivery.

In the later stages of shock, a different set of factors interfere with energy production and oxidative metabolism. Despite a normal to high level of oxygen delivery, chemical mediators can compromise mitochondrial function so that oxygen extraction and utilization is impaired. The resulting low tissue oxygen extraction ratio (<20% O_2/100ml consumption) is reflected in the common observation of a seemingly normal or high mixed venous oxygen hemoglobin saturation (70-85%) in the setting of organ dysfunction due to suspected hypoperfusion. Nonetheless, the clinical studies conducted on sepsis resuscitation support the idea of optimizing oxygen delivery in the earlier phases of sever sepsis. Accordingly, resuscitation from shock should be considered to occur in two phases. Initial early aggressive resuscitation with goal-directed therapy to *maximize* oxygen delivery improves outcome for patients in early septic shock (1). Beyond the first few hours, as mitochondrial dysfunction and other cellular metabolic dysfunction becomes established, aggressive attempts to augment oxygen delivery to *supernormal* levels does not improve organ dysfunction, and has been associated with increased mortality.(4, 5)

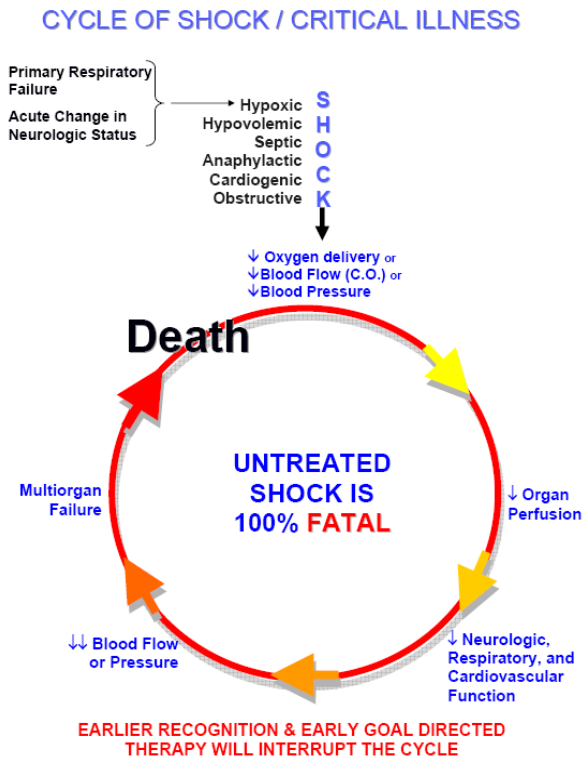


Figure 3. Cycle of Shock

The manifestations of *early* organ dysfunction in shock are subtle and unless rapidly recognized and corrected, a death spiral ensues (Figure 3). Hypoperfusion of the brain from sepsis will, for example, cause confusion, decrease in airway protection and ventilatory dysfunction leading to an unwanted set of pulmonary complications in addition to the initial problem with vascular resistance. Decrease in cardiac function or vasomotor tone arising as the primary cause of shock can likewise initiate the cycle. When loss of vascular tone, oxygen uptake or cardiac performance are secondary processes, they accelerate the death spiral even further.

Types of Shock

Shock states have been classically categorized according to major perturbations in circulatory homeostasis. Low cardiac output on the basis of inadequate preload has been termed hypovolemic shock; hemorrhagic shock is a variant of hypovolemic shock which is due to the loss of red cell mass (hence oxygen carrying capacity) as well as cardiac preload. Inadequate cardiac pump function is called cardiogenic shock; this state can also appear as a secondary process following episodes of hypoxemia or poor oxygen delivery due to hypotension. Distributive shock results from the inability to maintain vascular tone, either from biochemically-based endothelium/ vascular muscle dysfunction or from denervation. Distributive shock is the most severe manifestation of sepsis, allergy (anaphylaxis), pancreatitis, spinal cord injury and adrenal failure. Obstructive shock refers to mechanical impairment of either cardiac filling or expulsion, and includes processes such as tension pneumothorax, cardiac tamponade, valvular stenosis, and pulmonary embolus. Hypoxic shock refers to the end organ sequelae of prolonged hypoxemia. For example pneumonia and airway obstruction can lead to impaired cardiac performance (systolic and diastolic dysfunction), diaphragm muscle fatigue (hypercarbia and worse hypoxemia) and CNS depression (hypoventilation, poor protective reflexes and aspiration) that can combine to create a death spiral that overwhelms normal compensatory mechanisms.

Shock:
Septic or Distributive
Hypovolemic
Cardiogenic
Obstructive
Anaphylactic
Hypoxic

Summary

All shock states result in a decrease in O_2 delivery to vital organs. One is rarely called to the bedside for the diagnosis of shock; rather one is usually summoned to evaluate findings such as change in mental status, blood pressure, respiratory or heart rate that are non-specific. The clinician must maintain a high level of suspicion that a shock state may be evolving or exist. The next chapter will elaborate further on the 10 SOV as a tool to aid in the evaluation of patients at risk. The remaining chapters of the book continue to emphasize the 10 SOV for early recognition of deteriorating patients and lead to the "AOV/VIPPS" approach to goal directed resuscitation. Understanding the physiologic relationships that are perturbed when the body experiences respiratory (hypoxic) shock or circulatory shock will better prepare clinicians to identify earlier, evaluate and treat patients at risk for organ dysfunction and death.

References

1. Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med 2001;345(19):1368-77.
2. ? For debate. Respiratory Rate: The Neglected Vital Sign. Med J Aust 2008.
3. Cooper DJ, Michael Buist. Vitalness of Vital Signs, and MET. Med J Aust 2008.
4. Hayes MA, Yau EH, Timmins AC, Hinds CJ, Watson D. Response of critically ill patients to treatment aimed at achieving supranormal oxygen delivery and consumption. Relationship to outcome. Chest 1993;103(3):886-95.
5. Gattinoni L, Brazzi L, Pelosi P, et al. A trial of goal-oriented hemodynamic therapy in critically ill patients. SvO2 Collaborative Group. N Engl J Med 1995;333(16):1025-32.

Other related articles:

- Sax FL, Charlson ME. Medical patients at high risk for catastrophic deterioration. Crit Care Med 1987; 15:510-515
- Lundberg JS, Perl TM, Wiblin T, et al. Septic shock: an analysis of outcomes for patients with onset on hospital wards versus intensive care units. Crit Care Med 1998; 26:1020-1024
- Fanning R, Lighthall G. The Rapid Response Team: Bringing Critical Care Medicine out of the ICU. Pulmonary and Critical Care Medicine Update 2007; 21 Lesson 2.
- Lighthall G, Pearl R. How best to provide volume resuscitation in the critically ill. Journal of Critical Illness 2003;18(6):252-60

CHAPTER 3

EARLY RECOGNITION AND TREATMENT OF AT-RISK PATIENTS

Frank Sebat, MS, MD, FCCP, FCCM
Susan Henderson, MSN, NP, CCRN

Traditional vital signs including pulse, temperature, respirations, and skin color, have been utilized since the mid 1800s *(Notes on Nursing, Observations of the Sick*, 1859) to assess at-risk patients. By the early 1900s, the sphyngometer had been developed and blood pressure was added as a vital sign. These key physiologic variables remained the corner stone of separating stable from at-risk patients for the next 100 years. By incorporating additional signs, such as level of consciousness, SaO_2 (replacing skin color) capillary refill, urinary output and $ScvO_2$, base deficit, or lactic acid into the traditionally used vital signs, we can create a more complete, accurate and rapid bedside patient evaluation. .

Figure 1

YOUR PAGING NUMBER GOES HERE

CALLING CRITERIA EXAMPLES OF WHEN TO CALL ACCESS	
A IRWAY	• Obstructed • Stridor • Excessive secretions
B REATHING	• Respiratory rate ≤ 8 or ≥30 • Distressed breathing • Saturation <90% on oxygen
C IRCULATION	• Systolic BP ≤90 or ≥200 mmHg • BP decrease >40 mmHg • Heart rate ≤40 or ≥130 • Low urine output (≤100ml in 4 hr)
D ISABILITY	• Altered level of consciousness • Sudden loss of movement

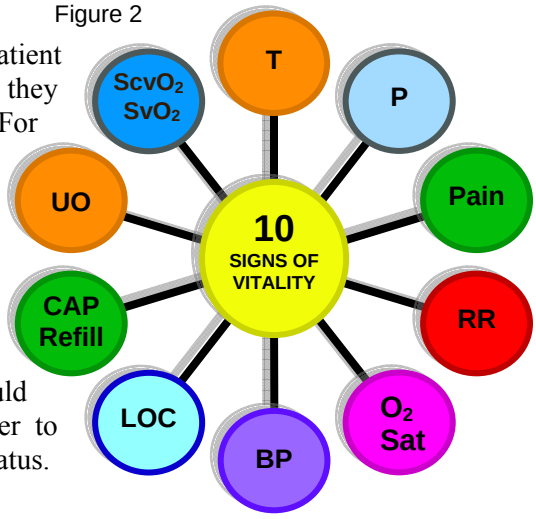
Conventionally most RRS medical emergency teams have utilized six vital signs: pulse, respiratory rate, SaO_2, blood pressure, urine output and level of consciousness. These activation criteria placed in the A·B·C·D pneumonic as seen in Figure 1 have the advantage of simplicity and sensitivity (capturing most, if not all, patients at-risk), but may more often mobilize intensive resources that are not needed (false positive).

Activation criteria, which can be developed, that maintain sensitivity while increasing specificity, are desirable. An example of this is the 10 Signs of Vitality (10 SOV) which adds capillary refill, temperature, pain and a measure of adequate global O_2 delivery, i.e., lactic acid, $ScvO_2$, or base deficit (Figure 2) to the activation criteria. The 10 SOV can be used singularly as conventional activation criteria or in combination. Expanding the vital signs and using them in combination will likely increase sensitivity (capturing most patient at-risk), specificity (reducing false positive activation) and identify earlier patients at-risk.

10 Signs of Vitality

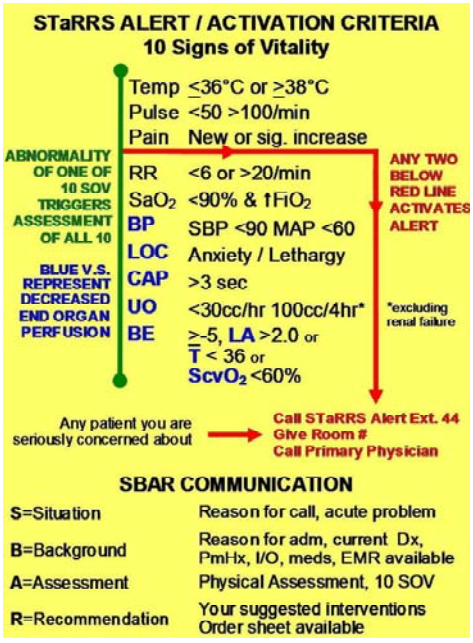
Temperature, pulse and pain are important triggers for a further patient assessment using the other seven signs of vitality. However, **alone** they do not necessarily identify a patient at-risk for decompensation. For example, a patient could have a very low heart rate i.e., 36 but mentating fine with good BP, urine output and SaO_2. Under these circumstances the patient is well perfused and, assuming the heart rate is not continuing to deteriorate, the patient is at low risk which allows time for consulting with the patient's physicians. The same can generally be said for abnormalities of temperature or pain associated with an otherwise normal assessment. However, a new significant abnormality in temperature, pulse or pain should trigger an assessment of all ten vital signs and be used together to determine the patient's neurologic, respiratory and hemodynamic status.

Figure 2



The remaining seven signs of vitality ***respiratory rate, SaO_2, blood pressure, level of consciousness, capillary refill, urinary output and $ScvO_2$ / base deficit / lactic acid*** are more sensitive and specific as early warning triggers compared to ***temperature, pulse and pain***. By lowering the RR threshold to 20 and coupling two of these seven signs for activation, at-risk patients will be identified earlier with increased specificity reducing unnecessary expenditure of resources

Figure 3



The 10 SOV can be used singularly as conventional activation criteria or in combination (Figure 3). By utilizing an abnormality of any one of the 10 SOV to trigger a full assessment of all ten and then combining any two abnormalities that fall below the red line, the most appropriate patients will be identified earlier. The rest of this chapter explains the importance, implication and what to do about abnormalities in respiratory rate, SaO_2, blood pressure, capillary refill, urinary output and $ScvO_2$ /BE /lactic acid.

Respiratory Rate

At a time when hospital staff are becoming increasingly dependant on new technologies, there is renewed interest in using the respiratory rate, the "neglected vital sign" as a means of detecting at-risk patients.[1] It is a timely reminder that understanding, documenting and acting on changes in patients' simple vital signs is of fundamental importance to clinical outcomes. An abnormal respiratory rate (high or low) is a highly reliable predictor of life threatening clinical events, but daily documentation of this simple number in many hospitals is remarkably poor. [1, 2]

Respiratory rate above 20 is the most sensitive sign of at-risk patients though least specific (i.e., shock, SIRS, respiratory insufficiency, pain, anxiety, etc). A respiratory rate above 20, coupled with other abnormalities such as altered mental status, hypotension, oliguria, poor capillary refill, lactic acidosis, etc., improves its specificity. Medical Emergency Teams (MET) who have lowered their respiratory rate threshold for activation from mid-30s to 20s have identified more at-risk patients. [3-10] The importance of this vital sign cannot be over-emphasized. Tachypnea (RR>20) is always present in at-risk patients excepting of narcotic and sedative over-medication.

SaO_2

"Hypoxia Kills" - as the patient's arterial oxygenation saturation decreases, all organ functions become impaired. As the central nervous system becomes hypoxic, loss of control of the airway, coupled with decreased intercostal and diaphragmatic muscle contraction, will lead to further hypoxia. If this process is not reversed, bradycardia followed by asystolic arrest will often occur. Measuring arterial hemoglobin saturation by oximetry is a quick and accurate method of rapidly assessing the patient's arterial oxygenation and is key in the assessment of at-risk patients. Normal arterial saturation falls between 94%-99% and is significantly abnormal when < 90%. A deteriorating SaO_2 with a change in the respiratory rate (RR) rapidly identifies the cause of the patient's hypoxia. Decreased SaO_2 with RR <12 is seen with CNS depression from sedatives or narcotics. Conversely a decreased SaO_2 with RR >20, is indicative of an excited CNS and is seen with systemic problems i.e., shock, sepsis SIRS or primary pulmonary problems.

Knowing the pitfalls in SaO_2 measurement will reduce error. A good pulse wave form correlating with the patient's heart rate generally assures that the reading is accurate. Accurate pulse oximeters can be difficult to obtain with poor circulation as reflected with a poor wave form and should be confirmed with an ABG.
Correcting arterial hypoxia is of paramount importance. Remember - hypoxia kills, hypercapnia without hypoxia does not.

Blood Pressure

Determining whether a given blood pressure for a patient is adequate is one thing; measuring blood pressure accurately is another. Accurate blood pressure measurement is key in assessing patients at-risk yet blood pressure determined by a non-invasive automated cuff in unstable patients can frequently be misleading.

Most to least accurate blood pressure measurement:

Arterial line

Doppler with manual cuff

Auscultation with manual cuff

Automated blood pressure cuff

Plysmography blood pressure determination utilizes changes in the arm circumference with arterial pulse at the site of measurement between systole and diastole. Small differences in tissue volume due to small pulse pressure from low stroke volume or increased vascular resistance introduce significant error in automated blood pressure measurement. As a result, hypotension can be missed in at-risk patients due to increased vascular resistance from endogenous catecholamines or from decreased stroke volume from hypovolemia or myocardial dysfunction. When proximal vascular restriction is not present, a properly transduced arterial line is the most accurate blood pressure measurement, followed by doppler determination of systolic blood pressure with a manual blood pressure cuff. Auscultation with a manual blood pressure cuff is next most accurate, with automated blood pressure cuff the least accurate.

If one of the 10 SOV suggests the patient is at-risk and the dinamap blood pressure is adequate, a doppler or other method to check the patients systolic blood pressure should be performed to confirm the pressure. A doppler systolic blood pressure is often measured by placing the probe on the brachial artery in the antecubital fossa by extending the arm and rotating the palm anterior. Once the sound of the arterial pulse is easily detectable (a brisk whŏŏŏf, whŏŏŏf sound, not to be confused with a much slower drawn out venous return sound), the blood pressure cuff is inflated to the point at which the pulse sound disappears, reflecting the systolic blood pressure. Remember, a hypotensive patient may go unrecognized due to inaccurate automated BP determination unless a second confirmatory method is utilized.

Level of Consciousness

The brain is one of the most (clinically apparent) sensitive organs to changes in perfusion. As CNS perfusion decreases, mentation goes through stages of anxiety, apathy, lethargy, stupor and eventually coma. This sequence of progression is quite different in a patient who becomes confused, agitated and then delirious. The latter reflects sedative or alcohol withdrawal, stimulant ingestion or acute psychosis. (Figure3)

In low-flow states or hypotension, the brain and heart will attempt to maintain perfusion by sacrificing blood flow to the skin, extremities and abdomen. Thus, alterations in mental status, combined with other signs of hypoperfusion, are serious indicators that the patient is at-risk for decompensation. As mental status deteriorates, the ability to protect the airway and maintain strong intercostal and diaphragmatic muscle contraction is reduced leading rapidly to cardio-pulmonary compromise. Efforts must be made to rapidly reverse the underlying cause and if not possible, to take measures to protect the airway and maintain ventilation.

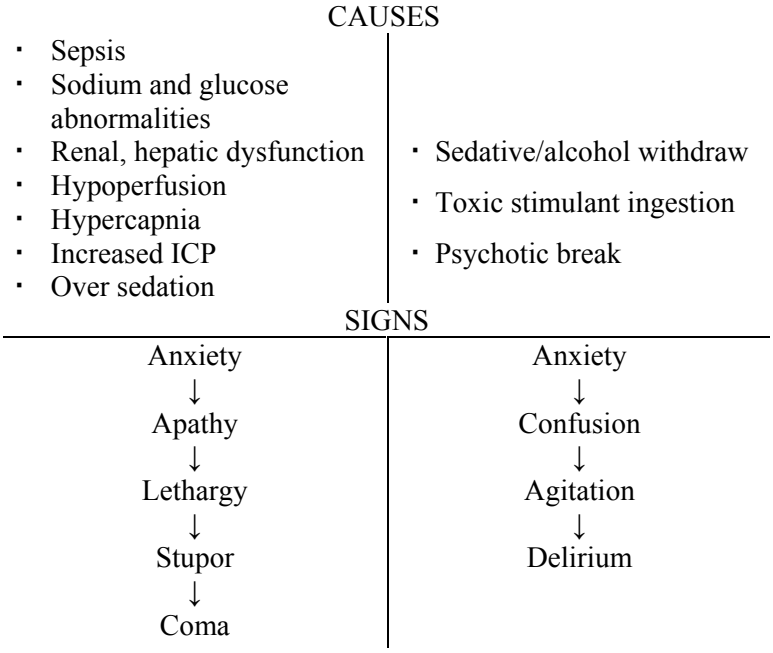


Figure 3. Causes and pathways of altered mental status

A thorough assessment of the change in level of consciousness includes focused neurologic exam using the Glasgow Coma Scale (GCS), assessment of the other nine signs of vitality, electrolytes, ABG, and medication review. An acute neurologic change often reflects a systemic process, i.e., medications, other metabolic causes, and/or a decreased cerebral blood flow of oxygen. When a deteriorating level of consciousness is associated with decreasing oxygen saturation or a GCS of <8, the patient is at immediate risk for sudden respiratory failure due to hypoxia or loss of airway. Airway-Oxygen-Ventilation (AOV) resuscitation should be immediately implemented (explained below).

Capillary Refill

The skin and extremities are the least vital organs and when a patient becomes stressed from illness the first organs to lose perfusion are the least vital ones. Capillary refill reflects extremity skin perfusion. It can be a very useful sign that the body is attempting to divert blood flow from less vital to more vital tissues. It can also reflect cool extremities due to exposure or loss of circulation to an isolated extremity though these causes are generally easy to rule out. In a recent study of patients being admitted into the Intensive Care Unit those with capillary refill > 4.5 sec were associated with 77% progressing to worsening organ function and increasing lactic acid ($p < 0.05$). [11]

A normal capillary refill is $\leq$ 2sec. An increase in extremity capillary refill >3 seconds is not specific, but is very sensitive in identifying a patient at-risk and needs to be interpreted in light of the other 9 SOV. For example, a patient who has a capillary refill of five seconds without altered pulse, blood pressure, respiratory rate, level of consciousness, O_2 saturation, oliguria, or low $ScvO_2$ may have poor circulation to the extremities, but is not at-risk for sudden cardio-respiratory collapse. Poor capillary refill should prompt further assessment of the remaining 9SOV. New altered mental status or oliguria coupled with capillary refill >3 sec can reflect a drop in cardiac output or blood pressure. Capillary refill is under utilized in patient assessment due to increased reliance on technology and the lack of large randomized controlled trials in adults although smaller observations [12-14] and years of experience suggest its usefulness. We believe, and as one study demonstrates, utility of capillary refill is **not** great as an isolated finding but can be significant when combined with the other vital signs. [14]

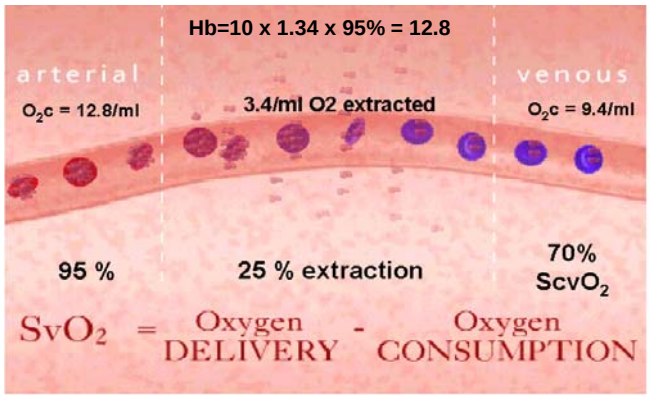
Urine Output is our window to abdominal organ perfusion. The renal artery is the largest end organ artery in the abdomen since the kidney's primary job is to filter the circulation. When renal artery blood flow decreases due to a systemic problem such as hypovolemia, all other abdominal organs will also have a decrease in blood flow. When oliguria is present one has to assess whether this is due to a systemic problem or a localized renal injury. Assessing the other nine signs of vitality will assist in determining whether oliguria is due to a pre-renal problem (i.e., low CO) or a renal issue (i.e., ARF). New oliguria in the presence of other vital sign abnormalities requires further evaluation. When oliguria is secondary to shock, the kidneys and other abdominal organs will become ischemic thus, even after renal recovery, an ischemic colon, small bowel or gallbladder may continue to be an ongoing source of SIRS.

$ScvO_2$ / Base Excess <-5 / Lactic Acid

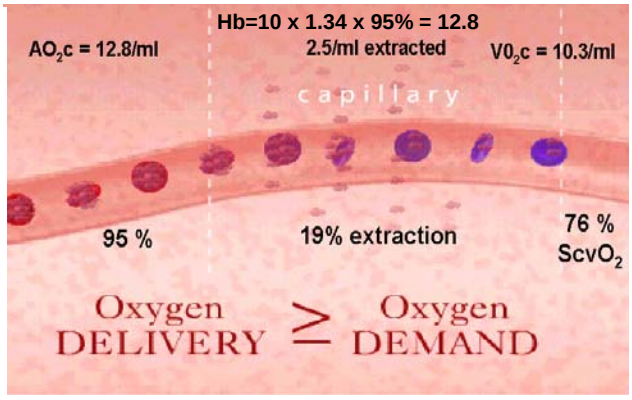
$ScvO_2$ less than 70% or base excess <-5 or/ lactic acid > 2 are indicators of the adequacy of global oxygen delivery. Measuring saturation of the hemoglobin returning to the right atrium after the tissues have extracted their oxygen needs through a CVP line ($ScvO_2$) can be used to evaluate the balance between O_2 delivery and demand (see chapter 2).

Oxygen delivery (hemoglobin, arterial oxygen saturation, cardiac output) minus oxygen utilization by the tissues (normal 25% of delivered O_2) leaves a residual quantity of O_2 (Approximately 70%) bound to Hb in the mixed venous blood (Figure 4). When the hemoglobin, arterial oxygen saturation, and tissue oxygen consumption are constant then any decrease in $ScvO_2$ reflects a decrease in cardiac output. It has become common practice in evaluating patients who are septic or may have other causes for low cardiac output to check the venous saturation through a central line placed in the right atrium. Again other variables being constant, a decrease in $ScvO_2$ reflects an increase tissue oxygen extraction due to decreased cardiac output and becomes a marker for the need of aggressive resuscitation to improve either heart rate, stroke volume, BP or any combination thereof.

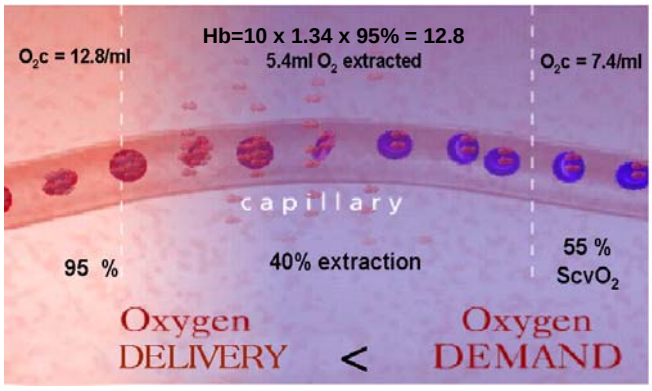
Figure 4



Normal CO and Demand



High Cardiac Output States or Tissue Shunting



Inadequate Cardiac Output

In shock states, more blood is delivered to the chest and head than to the abdomen. As a result, upper right atrial oxygen saturation is higher than lower right atrial saturation. This leads to a 5-8% higher $ScvO_2$ when measured from the upper right atrium compared to a mixed venous pulmonary artery sample (SvO_2) for patients in shock; therefore a $ScvO_2$ of 70% is ~ equal to SvO_2 of 65% and are respectively goals for resuscitation of shock.

$ScvO_2$ < 70

- Assess SaO_2, Hb, temperature, work of breathing and muscle activity
- If the above are unchanged and are acceptable then ↓$ScvO_2$ is due to **inadequate C.O.**
- Assess the other nine SOV and anticipate communicating and addressing SaO_2, Hb, ↑work of breathing or muscle activity, volume status, cardiac function

Conclusion

- Based on the 10 SOV assessment, anticipate and implement:
  - Respiratory / airway support by protocol
  - Volume / cardiovascular support by protocol
  - SBAR communication with MD
  - Neurologic support (Narcan / Ramazicon)
  - Change in medications
  - It has been suggested that "MET teams alone may be a simplistic "band-aid" response to a complex problem in our hospitals. A greater recognition of the importance of those vital signs together with greater commitment to investment in education, training, and perhaps to real-time emergency information communication systems, may be the key to improved outcomes." [2]

AOV / VIPPS WHAT TO DO ABOUT IT

Early identification of patients at-risk is an important part of the solution to improve patient outcomes. Rapid application of basic resuscitation is the remaining part of the solution and is far more effective in reducing morbidity and mortality than late application of advanced therapies. For example, support of the respiratory system, volume resuscitation and early administration of antibiotic therapy in sepsis will have a far greater impact on the outcome of patients with severe sepsis than later application of activated protein C. Once a patient at-risk is recognized early, it is imperative that we systematically apply best practice resuscitation. This can be facilitated by utilizing the pneumonic AOV/VIPPS as described below. More advanced therapies should also be applied early, when indicated.

All life saving interventions include assuring an adequate airway, arterial oxygenation, ventilation and circulation. Put simply, "*air needs to go in and out and blood needs to go round and round*." The pneumonic AOV (Airway, Oxygen, Ventilation) and VIPPS (Ventilation, Infusion of Volume, Pressors and Pharmacy and Specific intervention) assists in preventing us from overlooking these fundamental principals.

AOV Treatment

Airway. In assessing patients with hypoxia, tachpnea, increase work of breathing etc. determine if air is easily moving in and out of the chest if not extending the head back while thrusting the jaw forward. If this does not improve air movement then inserting a nasal airway can relieve oropharyngeal obstruction in obese or neurologically impaired patients. If these maneuvers are ineffective, intubation may be necessary. Once the airway is established, the next step is to administer enough oxygen to provide arterial oxygenation (i.e., SaO_2 >90%) and to assess ventilation of both lungs. Once adequate arterial oxygenation is established it is important to not over-ventilate the patient (i.e., large tidal volumes or fast rates) which can increase air trapping, intrathoracic pressures, risk of barotraumas while decreasing venous return, cardiac output and blood pressure. Remember, hypoxia kills; hypercapnia does not.

Patients, who are not ventilating appropriately, with or without hypoxia, can be divided into two categories; those who won't breathe due to CNS depression and those who can't breathe due to an airway, respiratory muscle, or lung parenchyma problem. Remember, AOV can be lifesaving regardless of the problem.

Primary Neurologic / Hypoxic Shock - WON'T BREATHE

SPECIFIC CAUSES	TREATMENT
• Narcotics / Sedatives	**A**irway (Naloxone / Flumazenil)
• ↑Ammonia and Hypothyroidism	**O**xygen
• Brain stem stroke / lesion	**V**entilation

Primary Respiratory Failure - CAN'T BREATHE

SPECIFIC CAUSES	TREATMENT — AOV
· Airway obstruction	· Jaw thrust with chin extension, nasal airway, NT suction, intubation
· Pneumonia · ARDS · PE } pulmonary shunt	· Postural drainage, ventilatory support · Low tidal volume ventilation, conservative fluid management, PEEP · Thrombolytics
· Asthma / COPD	· Bronchodilators, steroids, ventilatory support
· Muscle weakness (dead space)	· Ventilatory support

VIPPS Treatment

1969 and 1976, Dr. Max Harry Weil, a pioneer in the field of Critical Care Medicine, first described the ABCs of hemodynamic resuscitation, utilizing the pneumonic VIPPS [15, 16]. This included **V**entilation (and oxygenation), **I**nfusion of volume to restore circulation, **P**ressors needed to assure coronary artery and other vital organ perfusion (MAP ≥60) and to address any other problems with the **P**ump i.e., bradyarrhythmias or tachyarrhythmias, valvular problems etc. In addition the clinician should also be thinking about what **P**harmacological interventions are appropriate for his/her patient; for example are we rapidly administering appropriate antibiotic therapy for sepsis or steroids for anaphylaxis? Lastly, are there **S**pecific interventions that could rapidly reverse the patient's condition such as laparotomy for a perforated abdominal viscous or endoscopy for GI bleeding.

A Airway
O Oxygen
V Ventilation

V Ventilation (continue)
I Infusion of volume
P Pressors / Pump
P Pharmacy
S Specific interventions

All of these interventions are basic "bread and butter" care with one or more all too frequently underutilized or overlooked. Although our interventions may or should be administered in parallel, it is important to serially evaluate our interventions using the AOV/VIPPS pneumonic so we are maximizing basic care before proceeding to advanced resuscitation. A patient in cardiogenic shock with a respiratory rate of 30, but who does not look like they need to be intubated, will still benefit from maximum support of the respiratory system. This could include noninvasive ventilatory support (BiPAP) which allows the cardiac output, currently being used by intercostal muscles and diaphragm, to be diverted to the heart, brain or abdomen. Supporting the respiratory system in septic shock has repeatedly demonstrated benefit with decreased mortality. [17-19]

Nobody is ever requested to "evaluate a patient in distributive shock." More likely, it will be a call to evaluate someone with tachycardia, depressed metal status or some other non-specific finding. Since the treatments for all types of shock are initially the same (AOV / VIPPS) but later in their course the treatment is diametrically different, it is tempting to hold off treatment until a definitive diagnosis is made. We encourage a more active strategy. The key questions that need to arise are (1) is this patient showing early manifestations of shock, and (2) if so, what type of shock is it? The 10 SOV will provide a sensitive indication of risk and indicate the need for further diagnostic and therapeutic support. In the first minute or two of patient evaluation, one also should evaluate intravascular volume status and vascular tone. Understanding which of these parameters is abnormal allows one to stabilize the patient with the most appropriate type of empiric therapy as diagrammed below.

Volume	High Vascular Tone	Low Vascular Tone
Depleted	Hypovolemia, hemorrhage **Rx:** fluids, blood	Distributive shock before fluid resuscitation **Rx:** fluids
Adequately replaced	Cardiac problem* **Rx;** Inotrope ,Vasodilator	Distributive shock after fluids **Rx:** vasoconstrictor

*Includes cardiogenic and obstructive shock, valvular regurgitation and pulmonary hypertension

The main value of this scheme is to avoid the time and problems associated with giving excess amounts of fluid to patients who are fluid replete and to avoid attempts at raising blood pressure with vasoconstrictors in patients that are already maximally vasoconstricted. Note also the term "Cardiac problem" appears while the terms cardiogenic shock and obstructive shock do not. Findings such as rales and ECG abnormalities may help the clinician differentiate the problems that are lumped together as "Cardiac," but really there are no easy answers here. Inotropes may be useful in some cases, but harmful in others. An urgent echocardiogram is really needed to differentiate the various etiologies. Hypoxic shock can lead to types of circulatory dysfunction described here and so despite proper attention to airway patency, administration of oxygen and respiratory support, the hemodynamic sequelae require additional support.

AOV-VIPPS; the ABCs of Early Critical Illness/ Shock Resuscitation

AOVentilation

- Airway
- Supplemental Oxygen
- Early Ventilatory Support
- Adequate Hemoglobin to get $ScvO_2 > 65\%$.

Infusion of VOL

- 16 ga IVs
- Central line
- Crystalloid/ Colloid to get CVP > 12 and or $ScvO_2 > 65\%$
- Packed red blood cells for Hgb < 9, < 10 for persistent Shock and or $ScvO_2 > 65\%$

Pressors / Pump

- MAP $\leq$ 60
  - Levophed for Septic Shock
  - Dopamine or Levophed for Cardiogenic Shock
  - Add Dobutamine if MAP > 60 to get $ScvO_2 > 65$
- Treat dysrhythmia
- Rule out tamponade, ischemia, PE, or valvular abnormality

Pharmacy

- Antibiotics, activated protein C for sepsis
- Albuterol, steroids, H1 and H2 blockers for anaphylaxis
- Dobutamine / Nipride for cardiogenic shock with MAP > 60
- Thrombolytics for PE
- Other

Specific

- Endoscopy for upper GI bleed
- O.R. for ruptured Abdominal Aortic Aneurysm or perforated viscus
- Pericardiocentesis for tamponade

Remember!

The air needs to go in and out and

The blood needs to go round and round

References

1. Cretikos MA, Bellomo R, Hillman K, Chen J, Finfer S, Flabouris A: Respiratory rate: the neglected vital sign. *Med J Aust* 2008; 188(11):657-659
2. Cooper DJ, Buist MD: Vitalness of vital signs, and medical emergency teams. *Med J Aust* 2008; 188(11):630-631
3. Hodgetts TJ, Kenward G, Vlachonikolis IG, Payne S, Castle N: The identification of risk factors for cardiac arrest and formulation of activation criteria to alert a medical emergency team. *Resuscitation* 2002; 54(2):125-131
4. McBride J, Knight D, Piper J, Smith GB: Long-term effect of introducing an early warning score on respiratory rate charting on general wards. *Resuscitation* 2005; 65(1):41-44
5. Ryan H, Cadman C, Hann L: Setting standards for assessment of ward patients at risk of deterioration. *Br J Nurs* 2004; 13(20):1186-1190
6. Hogan J: Why don't nurses monitor the respiratory rates of patients? *Br J Nurs* 2006; 15(9):489-492
7. Fieselmann JF, Hendryx MS, Helms CM, Wakefield DS: Respiratory rate predicts cardiopulmonary arrest for internal medicine inpatients. *J Gen Intern Med* 1993; 8(7):354-360
8. Cretikos M, Chen J, Hillman K, Bellomo R, Finfer S, Flabouris A: The objective medical emergency team activation criteria: a case-control study. *Resuscitation* 2007; 73(1):62-72
9. Goldhill DR, McNarry AF: Physiological abnormalities in early warning scores are related to mortality in adult inpatients. *Br J Anaesth* 2004; 92(6):882-884
10. Harrison GA, Jacques TC, Kilborn G, McLaws ML: The prevalence of recordings of the signs of critical conditions and emergency responses in hospital wards--the SOCCER study. *Resuscitation* 2005; 65(2):149-157
11. Lima A, Jansen TC, van Bommel J, Ince C, Bakker J: The prognostic value of the subjective assessment of peripheral perfusion in critically ill patients. *Crit Care Med* 2009; 37(3):934-938
12. Gorelick MH, Shaw KN, Murphy KO, Baker MD: Effect of fever on capillary refill time. *Pediatr Emerg Care* 1997; 13(5):305-307
13. Tibby SM, Hatherill M, Murdoch IA: Capillary refill and core-peripheral temperature gap as indicators of haemodynamic status in paediatric intensive care patients. *Arch Dis Child* 1999; 80(2):163-166
14. Gorelick MH, Shaw KN, Murphy KO: Validity and reliability of clinical signs in the diagnosis of dehydration in children. *Pediatrics* 1997; 99(5):E6
15. Weil MH, Shubin H: The "VIP" approach to the bedside management of shock. *Jama* 1969; 207(2):337-340
16. Weil MH SH: **The "VIP" approach to the bedside management of shock.** Hagerstown, MD: Harper & Row; 1976.
17. Rivers E, Nguyen B, Havstad S, Ressler J, Muzzin A, Knoblich B, Peterson E, Tomlanovich M: Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med* 2001; 345(19):1368-1377
18. Sebat F, Musthafa AA, Johnson D, Kramer AA, Shoffner D, Eliason M, Henry K, Spurlock B: Effect of a rapid response system for patients in shock on time to treatment and mortality during 5 years. *Crit Care Med* 2007; 35(11):2568-2575
19. Chalmers JP, Tiller DJ: Effects of treatment on the mortality rate in septicaemia. *Br Med J* 1969; 2(653):338-341