BACKGROUND: Rapid response systems are still in development, and their practices vary significantly from hospital to hospital. Although the literature supports their value and a four-arm structure, it is unclear within these arms (afferent, efferent, quality assurance, administrative arms) which processes and procedures are responsible for their efficacy. This article reports the evolution of a rapid response system over many years at four institutions and considers the key elements that likely contribute to its efficacy.

METHODS: Retrospective evaluation of the processes, procedures, and outcomes of an adult general-ward rapid response system as it evolved, at four non-affiliated community medical centers, spanning 2 decades of development and refinement. System and patient outcomes examined included the number of rapid response system activations/1,000 admissions, time to rapid response system activation and/or interventions, cardiac arrest rate, and/or hospital mortality over time.

RESULTS: In the three hospitals that collected control and intervention data, there was significant increase in earlier and total number of rapid response system activations, more rapid administration of protocolized interventions, and associated decreases in cardiac arrest rate and hospital mortality of the respective population. In all four institutions three important common rapid response system processes were identified: early identification of at-risk patient using a novel focused bedside-assessment tool, leading to classification of the pathophysiologic process, linked to goal-directed intervention protocols.

CONCLUSIONS: Our review of a rapid response system that evolved over 20 years across four unrelated institutions revealed a common care pathway that coupled a focused bedside at-risk patient assessment leading to pathophysiologic classification of the patients decline linked to goal-directed intervention protocols. We speculate that the improved outcomes observed are a consequence of effective implementation and coupling of these three processes, as they are important in identifying and treating early the signs of tissue hypoxia and hypoperfusion, which remain the basic pathophysiologic threats of acute deterioration.

KEY WORDS: cardiac arrest; failure to rescue; quality improvement; rapid response system; resuscitation; severity scoring systems

In the United States, it is estimated that more than 400,000 inhospital preventable deaths occur annually (1). Many occur on general wards and are preceded by clear and significant vital sign abnormalities that may be underappreciated or unrecognized (2, 3). In response, hospitals have implemented rapid response systems (RRS). Although these systems are not uniform and still evolving, there is increasing evidence that RRS are effective and should have a four-arm structure: an administrative arm that provides oversite
and resources, an afferent arm/bedside clinician that recognizes patient deterioration and calls the efferent arm/response team, and a quality assurance arm (4). The quality assurance component reviews program compliance, and outcomes with the administrative arm ensuring system policies and procedures are continuously updated. However, it is still unclear, within this structure, which processes or interventions contribute most to RRS effectiveness (5, 6).

RRSs use different several different approaches to improve the early recognition and treatment of the deteriorating patient including early warning scores (EWS) to trigger concern and need for further patient evaluation or intervention. A variety of single parameter or aggregated EWSs are used for the early detection of patient deterioration. These scores are commonly used, and some have shown significant value in reducing failure to rescue, yet to date, published studies have demonstrated mixed results (7, 8). EWS do not provide immediate insight into what may be wrong with the patient and what to do about it. Indeed, before interventions can begin, the patient must be reexamined to understand the pathophysiology responsible for its activation. In addition to EWS, mnemonics such as Airway, Breathing, Circulation, Disability/neuro, Exposure (ABCDE) to identify skin and extremity injury have been used by trauma teams and RRSs to quickly identify, prioritize, and direct treatments in life-threatening conditions (9, 10).

The early identification of the specific, sometimes subtle, early signs and symptoms heralding acute decline coupled with best practice interventions are important acute care process in trauma, acute myocardial infarction, stroke, etc. and are likely important for inhospital RRSs. In this review, we report the evolution of an RRS at four different institutions over 20 years and consider components that may have contributed most to the improved outcomes observed.

**MATERIALS AND METHODS**

**Retrospective Analysis**

We retrospectively reviewed the development, evolution, and refinement of an RRS at four nonaffiliated community medical centers spanning 2 decades. The number of RRS activations/1,000 admissions and/or time to RRS activation/timeliness of interventions, cardiac arrest rate, and/or hospital mortality of the respective populations were examined.

**System Evolution**

*Implementation Development and Evolution of the RRS.* Development and implementation of our four-arm RRS began in 1999 after several hospital sentinel events of unrecognized shock prompted action. Initially, the focus was only on patients with one of the five classic forms of shock (i.e., hypovolemic, septic/distributive, cardiogenic, obstructive, and anaphylactic) for which was created, based on clinical experience and the literature, an empiric focused Early Warning-bedside patient Assessment (EWA). It was comprised of the seven common signs of organ hypoperfusion (i.e., altered mental status, hypothermia, hypotension, tachypnea, prolonged capillary refill time [CRT], oliguria, and increased lactic acid (LA) or base deficit (BD)) (11, 12). The presence of two or more of these seven parameters activated the “shock” team to deliver a treatment protocol based on Dr. Max Harry Wiel “Ventilation and Infusion of volume, and then the need for Pressors and pump support, Pharmacologic interventions and/or Specific interventions” (VIPPS) algorithm, which addressed optimizing VIPPS interventions (13).

This approach rapidly recognized and treated patients with one or more of the five classic types of shock (14), but it did not deal adequately with a significant number of at-risk patients with hypoxia (e.g., airway compromise, aspiration, decompensated congestive heart failure, chronic obstructive pulmonary disease, etc.). Therefore, in 2007, Sao2 less than 90% with increasing oxygen requirement was added to the EWA as a further life-threatening pathophysiologic derangement, and the VIPPS protocol was amended by dividing it into two parts. The first part emphasized the protection and maintenance of the Airway, the provision of supplemental Oxygen and noninvasive or invasive Ventilation if needed (AOV) to decrease work of breathing, which is often overlooked in a patient with persistent hypoperfusion but without obvious respiratory difficulty. Such patients benefit significantly from resting the intercostal and diaphragmatic muscles by ventilatory support and have been demonstrated to reduce the mortality of patients in shock (15, 16). Decreasing the work of breathing, even if it does not look excessive, allows blood to be redirected from the respiratory muscles to more vital organs (e.g., heart, brain, and kidneys). This leads to a decrease in oxygen consumption, reduced endogenous catecholamine release, lower systemic vascular resistance, and improved
cardiac output, with better match of oxygen delivery to consumption.

The second part of the protocol “Infusion of volume and assessing need for Pressors and/or pump support, other Pharmacologic interventions (antibiotics, steroids, etc.) and/or Specific interventions” (IPPS) is implemented if correction of hypoxia, work of breathing, and hypercapnia did not reverse the patient’s abnormalities (i.e., altered mental status, hypothermia, hypotension, tachypnea, prolonged CRT, oliguria, and increased LA or BD), and hypoperfusion persisted. Once the primary pathophysiology is identified as one or more of the following: hypoxia, or one or more of the five classic forms of shock (hypovolemic/hemorrhagic, sepsis/distributive, cardiac, obstructive, or anaphylactic); more specific goal-directed protocols are implemented (9, 10). These interventions prompted by the AOV-IPPS interventions mnemonic (Table 1), taught in series but often implemented in parallel, are the basic therapies for patients suffering from hypoxia and/or hypoperfusion and must be addressed before embarking on more advanced resuscitation.

**Development of the Bedside EWA.** In addition, in 2007, three additional parameters of fever, pain, and

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**TABLE 1.**

**AOV-IPPS Interventions Titrated to Goals**

| Airway                          | Jaw thrust                  |
|------|-----------------------------|
|      | Racemic epinephrine for strider |
|      | Nasal trumpet or oral airway, if needed |
|      | High-flow NC, BIPAP, or intubation if above is not effective in relieving airway problems |
| Oxygen to keep $\text{Sao}_2 > 92\%$ | Increase $\text{FIO}_2$ |
|      | If on 100% rebreather mask patient still hypoxic turn $\text{O}_2$ meter to flush if the above is not effective, setup for high-flow NC, BIPAP, and/or intubation |
| Ventilatory support early, putting the respiratory system to rest (allowing cardiac output to be redirected from the bellows to the abdominal organs) | Bronchodilators for prolong exhalation or wheezing |
|      | High-flow NC or BIPAP |
|      | If the above is not effective, then consider intubation |
| Infusion of volume, per fluid challenge protocol, if fluids thought to be tolerated, i.e., not in pulmonary edema or unable to oxygenate | Pressors/pump, if MAP < 60 mm Hg |
|      | Pressor as ordered by physician to get MAP ≥ target |
|      | If patient on a vasoconstrictor and MAP > 60 mm Hg and goals not met add dobutamine starting at 2 $\mu$g/kg, titrate dose as ordered and tolerated |
|      | Inotropes as ordered by physician to urine output > 25 cc/hr, capillary refill time ≤ 3 s, improved mental status, decreasing lactic acid, $\text{ScVO}_2 ≥$ and/or cardiac output to target |
|      | Treat tachy and brady dyarhythmas to optimize heart rate |
|      | Pressors/inotropes titrated to goals. As goals are met, wean vasoactive drugs, starting with pressors, as tolerated |
| Pharmacy | Antibiotics for sepsis |
|      | Albuterol, steroids, H1, and H2 blockers for anaphylaxis |
|      | Dobutamine and/or milrinone and/or nipride for persistent cardiogenic shock with MAP > 60 |
|      | Thrombolytics for pulmonary embolus with shock or severe right ventricular dysfunction |
|      | Other |

(Continued)
abnormal heart rate (HR) were added to the bedside EWA, because these “minor signs” are so frequently assessed on the general wards and can appropriately trigger concern. Abnormal HRs are nonspecific and often easily managed (e.g., atrial fibrillation, paroxysmal tachycardia, etc.) along with fever or pain without signs of hypoxia or hypoperfusion that are associated with benign outcomes (17, 18). Nevertheless, changes in these three parameters may be an indication of early deterioration. Therefore, when newly present, they triggered further patient evaluation/observation. These three minor signs (HR, fever, and pain) combined with $\text{Sao}_2$ and the seven perfusion parameters of mental status, hypothermia, blood pressure, respiratory rate, CRT, urine output, and LA/BD collectively became known as the “Ten Signs of Vitality” (10-SOV), an empirically derived, rapid, simple, bedside assessment for the prompt detection of patients at-risk for acute decline. Since hyperthermia and hypothermia are considered differently within the 10-SOV, it is a comprised of 11 interpretations of the 10 vital signs that reflect tissue hypoxia or hypoperfusion.

Any change in a patient’s condition that causes concern, or any change in a routinely measured vital sign (i.e., temperature, HR, respiratory rate, blood pressure, pain, and $\text{Sao}_2$) triggers a full 10-SOV assessment. If only one minor or major sign is newly abnormal, then patient monitoring was intensified with more frequent bedside evaluations. Since the presence of two or more vital sign abnormalities significantly increases the risk of deterioration (19), the rapid response team (RRT) was activated if two or more of the eight “major” parameters of hypoxia and/or hypoperfusion were abnormal (Fig. 1).

The Current Care Process. The current iteration of this evolving RRS was implemented in the fourth hospital in 2011. Patient surveillance starts with the 10-SOV parameters being recorded at the beginning of each shift as part of the routine nursing assessment. It is repeated at any time a new routine vital sign abnormality occurs or if any significant concern in the

| Specific interventions | Resuscitation goals |
|------------------------|---------------------|
| Endoscopy for upper gastrointestinal bleed | Improving mental status |
| Surgery for acute abdomen | $\text{Sao}_2 > 90\%$ |
| Pericardiocentesis or to operating room for tamponade | Decrease work of breathing |
| Others as indicated | MAP $> 60$ mm Hg |

$\text{AOV-IPPS} = \text{maintain Airway, the provision of supplemental Oxygen and noninvasive or invasive Ventilation if needed Infusion of volume and assessing need for Pressors and/or pump support, other Pharmacologic interventions (antibiotics, steroids, etc.) and/or Specific interventions, BiPAP = bileval Inspiratory positive airway pressure, MAP = mean arterial pressure, NC = nasal cannula.}$

$\text{These goals are based on normalizing the eight “major” 10 signs of vitality parameters reflecting hypoxia and or hypoperfusion and if central line available mixed venous oxygen saturation is included per physician order.}$

$\text{AOV-IPPS protocol for treatment of all patients identified to at-risk for critical Illness by the Ten Signs of Vitality (10-SOV) Early Warning bedside patient Assessment. Patients are initially classified as suffering primarily from “hypoxia” prompting AOV interventions or “hypoperfusion” requiring both AOV and IPPS treatments. Once the patient’s pathology is better defined, more specific protocols are implemented for hypoxia, hypovolemia, sepsis, cardiogenic, obstruction of blood flow, or anaphylaxis (9, 10) until resuscitation goals are met.}$
patient’s condition develops. When activation criteria are recognized, at the bedside, the nurse initiates AOV resuscitation as indicated, for example, airway management, supplemental oxygen, bronchodilators, “ambu-bag,” etc., while awaiting arrival of the RRT. The response team attempts to classify, based on the 10-SOV criteria, the patient’s primary pathophysiology as either “hypoxia” and/or “hypoperfusion” and continues the AOV management algorithm while adding IPPS interventions if hypoperfusion is identified and/or persists until resuscitation goals are met (Fig. 1; Table 1).

This three-component general ward at-risk patient management system (i.e., 10-SOV bedside at-risk patient assessment and six pathophysiological pathway classifications coupled with AOV-IPPS protocolized interventions) has been embedded in the hospital culture by extensive staff education, audits, and compliance measures. This includes mandatory entry of all 10-SOV parameters into the electronic medical record, which are continuously monitored electronically for their capture at the beginning of each shift and when there is a new vital sign abnormality identified. When 10-SOV activation criteria are met (i.e., two new “major” abnormalities; Fig. 1), audits for timelines of RRT mobilization are conducted. Nurse managers are alerted to any variations,

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**Figure 1.** Flow diagram demonstrating the linkage of ten signs of vitality patient track and trigger early warning assessment (10-SOV TT-EWA) with primary pathophysiology initially identified as primarily hypoxia or hypoperfusion prompting Airway maintenance, the provision of supplemental Oxygen and non-invasive or invasive Ventilation (AOV) and Infusion of volume and assessing need for Pressors and/or pump support, other Pharmacologic interventions (IPPS) treatment protocol as indicated. The 10 vital signs contain 11 parameters (hyperthermia and hypothermia are evaluated differently) and require abnormalities of two of the eight “major” parameters below the line to identify the patient to be at risk. This triggers an RRT response and AOV resuscitation; if hypoperfusion persists or develops, IPPS resuscitation is also begun while optimizing AOV interventions. These interventions are taught in series but are often implemented in parallel. BE = base excess, CRT = capillary refill time, HR = heart rate, LA = lactic acid, LOC = level of consciousness/mental status, Low T = hypothermia, RR = respiratory rate, SIRS = systemic inflammatory response syndrome, T = fever, UO = urine output.
which they remedy by providing feedback and ongoing education to their staff. The response team AOV-IPPS interventions are reinforced through case studies, simulation, and team debriefings. They are initially applied to all six pathophysiology classifications and subsequently tailored according to the underlying condition identified (11, 12).

**RESULTS**

These described RRS care processes serially evolved at four hospitals over 2 decades (11, 14, 20–22) and demonstrated significant improvement in outcomes within the three of hospitals that had reportable data (Table 2) (11, 14, 20, 21). The first iteration (hospitals 1 and 2) addressed the five classic forms of shock and demonstrated a significant reduction in the time to perform key interventions with mortality falling proportionally as time to treatment decreased (14, 20). In hospital 1, identification of patients in shock doubled, and hypovolemic and septic shock mortality was significantly reduced (Table 2) (14). This program was subsequently adopted by a second hospital San Francisco Bay area in 2003 with similar results (20) and by a third hospital in north-central California in 2005 (Table 3) (22).

The fourth and current iteration of the program was developed at a 450-bed regional central-California medical center. At this hospital, as previously reported, RRS activations increased from 10.2 to 48.8/1,000 discharges ($p < 0.001$) and a decrease of ward cardiac arrest decreased from 3.1 to 2.4/1,000 discharges ($p = 0.04$), hospital mortality from 3.8% to 3.2% ($p < 0.001$), and the observed-to-expected mortality ratio from 1.5 to 1.0 ($p < 0.001$) (Table 2) (21).

Previously unpublished analysis (2019) at this hospital found that patients serially identified to be at-risk by the 10-SOV criteria, 87% (4,521/5,184) could be classified primarily within one of the six physiologic derangements (Table 3). Hypoxia and hypoperfusion accounted for 47% and 53%, respectively. The latter was comprised of patients with hypovolemia (18.7%), sepsis (15.4%), primary cardiac problems (15%), obstruction of blood flow (3.5%), and anaphylaxis (0.5%). Table 3 displays the percent transferred to a higher level of care, hospital mortality, and length of stay (LOS) for patients in each of the six pathophysiologic subgroups with hypoxia and sepsis having the highest mortality, 31% and 29%, respectively, and longest LOS of 11 days.

These RRS that evolved over 20 years were associated with improved patient outcomes and shared a common approach that provided for “prompt identification at the bedside of early patient decline, categorization of at-risk patients into specific pathophysiologic pathways” coupled to “protocolized treatments.”

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**TABLE 2.**

Rapid Response System Outcomes of Three Hospitals

| Hospital No./Time Frame | No. of Patients | Mean Time to Three Key Interventions or Rapid Response Team Activations/1,000 Admissions | Mortality |
|-------------------------|----------------|---------------------------------------------------------------------------------|-----------|
|                         |                | C  | I  | C  | I  | p   | C  | I  | p   |
| 1/2000–2005             | 85             | 426| 108 min | 30 min | <0.001 | 50% | 10% | <0.001 |
| 2/2003–2005             | 68             | 63 | 390 min | 54 min | <0.001 | 51% | 27% | <0.001 |
| 4/2008–2013             | 28,914         | 39,802 | 10.2/1,000* | 48.8/1,000 | <0.001 | 3.8% | 3.2% | <0.001 |

C = control cohort, I = intervention cohort.

*Rapid response team activations/1,000 hospital admissions.

Summary of results of the three hospitals with control and intervention data available before and after implementation of a bedside at-risk patient assessment, pathophysiologic classifications and Airway, the provision of supplemental oxygen and noninvasive or invasive ventilation if needed infusion of volume and assessing need for pressors and/or pump support, other pharmacologic interventions (antibiotics, steroids, etc.), and/or specific interventions care system. Hospitals 1 and 2 looked at only patients with conventional forms of shock for 5- and 1-yr intervention periods, respectively. Hospital 4 was a larger facility and included general ward patients at risk for both hypoxia and shock. All patients observed on the general wards by the rapid response systems were included, as opposed to treated, accounting for the large increase in the number of study participants.
DISCUSSION

This report supports the concept that linkage of “focused bedside patient assessment, pathophysiologic classification,” and “goal-directed intervention protocols” within a four-arm RRS was associated with a significant increased number of patients identified to be at-risk, reduced time to RRS activation and/or treatment, and lowered cardiac arrest rates and/or hospital mortality of the respective populations.

The structure of effective acute care systems requires early identification of the relevant population (afferent arm), which triggers prompt delivery of indicated interventions (efferent arm). For these systems to function well and continuously improve the administrative-arm needs to provide necessary resources based on system data regarding compliance and effectiveness (quality assurance-arm). This study is a report of the performance of a four-arm RRS as it evolved over 20 years in four different hospitals. Within each hospital, we attempted to identify common important RRS processes that accounted for their success. The shared care pathway that emerged was the coupling of a “bedside assessment for the early identification of at-risk patients,” prompting “pathophysiologic classification” of their decline, linked to “protocolized interventions” as the key functions that were likely important system components responsible for improved patient outcomes.

It is probable that other factors were also important, such as ongoing staff education to ensure system compliance and performance audits to promote continuous improvement (21). Nurses were all educated on the pathophysiology reflected by any abnormality of each of the 10 vital signs (Fig. 1). The entry by the bedside registered nurse of all 10-SOV parameters into the electronic medical record was required by policy at the beginning of each shift, or when a new vital sign abnormality occurred and again by the RRT, if activated, upon arrival to the bedside. Audits for compliance were monitored manually in the first three hospitals and electronically in the fourth. Improvements were incremental as this approach became part of each hospital’s culture. These educational and compliance measures are essential in assuring that the “three key care processes identified” are successfully performed.

No healthcare system depends on a single component nor is it possible to identify which component most determines outcome, as all will inevitably depend on each other. Nevertheless, we believe the 10-SOV EWA has advantages over EWS as it is performed and triggered when criteria are met at the bedside and promptly identifies specific critical pathophysiology in real time that nurses can be taught to recognize, understand, and quickly initiate treatment.

Our analysis is a retrospective review of an evolutionary process, without any precisely defined start and finish—process is ongoing. Control data were not available from one of the four institutions as their preintervention period was documented in

| Table 3. Distribution and Outcomes of At-Risk Patients by Primary Pathophysiology at Hospital #4 |
|---------------------------------------------------------------|
| **Rapid Response Team Patients Classified by Risk Type**      |
|                                                              |
| Hypoxia          | Hypovolemia | Septic | Cardiogenic | Obstructive | Anaphylactic | Population, % (n) |
| 47 (2,124)       | 18.7 (845)  | 15.4 (698) | 15 (670) | 3.5 (162) | 0.5 (22) | <0.001* |
| Transfer to higher level of carea, % (n)                     |
| 51 (1,064)       | 49 (412)    | 63 (438)    | 52 (344) | 54 (86) | 55.5 (12) | <0.001c |
| Mortality, % (n)                                           |
| 31 (662)         | 25 (214)    | 29 (200)    | 18 (123) | 27 (43) | 9 (2)   | <0.001c |
| Length of stay median/d (interquartile range)               |
| 11 (6–19)        | 9 (4–17)    | 11 (6–19)   | 8 (4.25–14) | 10 (5–15.75) | 6 (3.25–9) | <0.001d |

*Pearson goodness of fit test.
+cTransfer to higher level of care included intermediate unit or ICU.
+Fishers exact test.
+aKruskal-Wallis test.

Distribution and outcomes in hospital 4 of rapid response team patients of which 87% (4,521/5,184) who met activation criteria were able to be classified within one of the six pathophysiologic pathways.
a legacy database that no longer exists. However, in the three different hospitals that complete data was available, clinical deterioration was recognized progressively more rapidly, and inhospital mortality of the respective population steadily declined making it more likely that the key RRS processes identified were in part responsible for the improved patient outcomes.

In acute care, most improvements are small and attributable to more conscientious and effective implementation of many items of care rather than a single factor. To define precisely the factors that improve evolving complex adaptive systems such as a RRS is difficult: no two patients are ever the same, and diseases and their treatments are constantly changing. Nevertheless, for most at-risk patients, hypoxia and/or hypoperfusion remain the basic pathophysiologic threats that must be promptly recognized and rapidly reversed to prevent further acute deterioration. Our review identified an easily adaptable common approach that successfully accomplished this in unrelated hospitals over many years. Further incremental improvement requires that advances in best practice continue to be absorbed into this evolving system.

CONCLUSIONS

Our review of an RRS that evolved serially at four institutions over many years revealed an important common theme or care process that coupled an “At-risk Patient Bedside Assessment, Pathophysiologic Classification of Patient Decline,” linked to “Goal Directed Interventions,” which identified and promptly treated patients with early signs of tissue hypoxia and/or hypoperfusion and was associated with improved system and patient outcomes.

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