02 - 311 Approach to the Patient with Critical Illness

311 Approach to the Patient with Critical Illness

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Approach to the Patient

with Critical Illness The care of critically ill patients requires a thorough understand ing of pathophysiology and centers initially on the resuscitation of patients at the extremes of physiologic deterioration. This resuscita tion is often fast-paced and occurs early when a detailed awareness of the patient's chronic medical problems may not yet be possible. While physiologic stabilization is taking place, intensivists attempt to gather important background medical information to supplement the real-time assessment of the patient's current physiologic condition. Numerous tools are available to assist intensivists in the assessment of pathophysiology and management of incipient organ failure, offer ing a window of opportunity for diagnosing and treating underlying disease(s) in a stabilized patient. However, despite these tools, ongo ing clinical bedside assessment is imperative for care of the critically ill patient. Indeed, the use of interventions to support the patient, such as mechanical ventilation and renal replacement therapy, is commonplace in the intensive care unit (ICU). An appreciation of the risks and benefits of such aggressive and often invasive interven tions is vital to ensure an optimal outcome. Nonetheless, intensivists must recognize when a patient's chances for recovery are remote or nonexistent and must counsel and comfort dying patients and their significant others if an initial trial of invasive supportive care is either not effective or is not appropriate for the patient's current condition. Critical care physicians often must redirect the goals of care from resuscitation and cure to comfort when the resolution of an underly ing illness is not possible. The COVID-19 pandemic has heightened the need and priority for effective critical care practices (Chap. 205), as well as the need for additional support for post-ICU care and recovery for ICU survivors TABLE 311-1 Calculation of SOFA Scorea SYSTEM

Respiration Pao2/FIo2, mmHg (kPa) ≥400 (53.3) <400 (53.3) <300 (40) <200 (26.7) with respiratory support Coagulation Platelets, × 103/μL ≥150 <150 <100 <50 <20 Liver Bilirubin, mg/dL (μmol/L) <1.2 (20) 1.2–1.9 (20–32) 2.0–5.9 (33–101) 6.0–11.9 (102–204)

12.0 (204) Cardiovascular MAP ≥70 mmHg MAP <70 mmHg Dopamine <5 or

dobutamine (any dose)b Dopamine 5.1–15 or

epinephrine ≤0.1 or norepinephrine ≤0.1b Central nervous system Glasgow Coma Scalec

13–14 10–12 6–9 <6 Renal Creatinine, mg/dL (μmol/L)

<1.2 (110) 1.2–1.9 (110–170) 2.0–3.4 (171–299) 3.5–4.9 (300–440) or <500 or urine output, mL/d aAdapted from JL Vincent et al: Working Group on Sepsis-Related Problems of the European Society of Intensive Care Medicine. The SOFA (Sepsis-related Organ Failure Assessment) score to describe organ dysfunction/failure. Intensive Care Med 22(7):707, 1996. bCatecholamine doses are given as μg/kg per min for at least 1 h. cGlasgow Coma Scale scores range from 3 to 15; higher score indicates better neurological function. Abbreviations: FIo2, fraction of inspired oxygen; MAP, mean arterial pressure; Pao2, partial pressure of oxygen.

Critical Care Medicine PART 8 ASSESSMENT OF ILLNESS SEVERITY In the ICU, illnesses are frequently categorized by degree of severity. Numerous severity-of-illness (SOI) scoring systems have been devel oped and validated over the past three decades. Although these scoring systems have been validated as tools to assess populations of critically ill patients, their utility in predicting individual patient outcomes at the bedside is not clear. Their utility may be more applicable toward defining patient populations for clinical trial outcomes and broader epidemiologic studies. SOI scores are also useful in guiding hospital administrative policies, directing the allocation of resources such as nursing and ancillary care, and assisting in assessments of quality of ICU care over time. Scoring system validations are based on the prem ise that age, chronic medical illnesses, and derangements from normal physiology are associated with increased mortality rates. All existing SOI scoring systems are derived from patients who have already been admitted to the ICU. Nevertheless, there has been increased recent clinical use of scoring systems due to revised consensus guidelines for definitions of sepsis, as will be detailed below. The most commonly utilized scoring systems are the SOFA (Sequen tial Organ Failure Assessment) and the APACHE (Acute Physiology and Chronic Health Evaluation). There has been more recent interest in the use of a “quick” SOFA, or qSOFA, scoring system for prognosti cation of sepsis outcomes. ■ ■THE SOFA SCORING SYSTEM The SOFA scoring system is composed of scores from six organ systems, graded from 0 to 4 according to the degree of dysfunction (Table 311-1). The score accounts for clinical interventions; it can be measured repeatedly (i.e., each day), and rising scores correlate with increasing mortality. The most recent sepsis consensus conference guidelines incorporated an increase of at least two points in the SOFA score from baseline as diagnostic of sepsis in the setting of suspected or documented infection. Patients with suspected infection can be predicted to have poor outcomes typical of sepsis if they have at least two of the following clinical criteria: respiratory rate ≥22 breaths/min, altered mental status, or systolic blood pressure ≤100 mmHg. Recently, a new bedside clinical score using two or more of the above clinical criteria has emerged and is termed quick SOFA (qSOFA). qSOFA is intended to screen patients for risk of poor outcomes from sepsis from SCORE <100 (13.3) with respiratory support Dopamine >15 or epinephrine >0.1 or norepinephrine >0.1b

> 5.0 (440) or <200

out-of-hospital, emergency department, and hospital ward settings. qSOFA was not developed, nor is it currently recommended, as a sep sis diagnostic screening tool, but studies are investigating its utility as such, especially in resource-poor settings that may not have the ability to measure all the components of the SOFA score.

■ ■THE APACHE II SCORING SYSTEM The APACHE II system is the most commonly used SOI scoring system in North America. Age, type of ICU admission (after elective surgery vs nonsurgical or after emergency surgery), chronic health problems, and 12 physiologic variables (the worst values for each in the first 24 h after ICU admission) are used to derive a score. The predicted hospital mortality rate is derived from a formula that takes into account the APACHE II score, the need for emergency surgery, and a weighted, disease-specific diagnostic category (Table 311-2). The relationship between APACHE II score and mortality risk is illus trated in Fig. 311-1. Updated versions of the APACHE scoring system (APACHE III and APACHE IV) have been published. PART 8 Critical Care Medicine ■ ■OTHER SCORING SYSTEMS There are numerous other scoring systems that have been developed, and there are ongoing studies evaluating their utility. In particular, TABLE 311-2 Calculation of Acute Physiology and Chronic Health Evaluation II (APACHE II) Scorea Acute Physiology Score SCORE +4 +3 +2 +1 +0 +1 +2 +3 +4 Rectal temperature (°C) ≥41 39.0–40.9 38.5–38.9 36.0–38.4 34.0–35.9 32.0–33.9 30.0–31.9 ≤29.9 Mean blood pressure (mmHg) ≥160 130–159 110–129 70–109 50–69 ≤49 Heart rate (beats/min) ≥180 140–179 110–139 70–109 55–69 40–54 ≤39 Respiratory rate (breaths/min) ≥50 35–49 25–34 12–24 10–11 6–9 ≤5 Arterial pH ≥7.70 7.60–7.69 7.50–7.59 7.33–7.49 7.25–7.32 7.15–7.24 <7.15 Oxygenation If FIo2 >0.5, use (A – a) Do2 ≥500 350–499 200–349 If FIo2 ≤0.5, use Pao2

> 70 61–70 55–60 <55 Serum sodium (meq/L) ≥180 160–179 155–159 150–154 130–149 120–129 111–119 ≤110 Serum potassium (meq/L) ≥7.0 6.0–6.9 5.5–5.9 3.5–5.4 3.0–3.4 2.5–2.9 <2.5 Serum creatinine (mg/dL) ≥3.5 2.0–3.4 1.5–1.9 0.6–1.4 <0.6 Hematocrit (%) ≥60 50–59.9 46–49.9 30–45.9 20–29.9 <20 WBC count (103/mL) ≥40 20–39.9 15–19.9 3–14.9 1–2.9 <1 Glasgow Coma Scoreb,c EYE OPENING VERBAL (NONINTUBATED) VERBAL (INTUBATED) MOTOR ACTIVITY 4—Spontaneous 5—Oriented and talks 5—Seems able to talk 6—Verbal command 3—Verbal stimuli 4—Disoriented and talks 3—Questionable ability to talk 5—Localizes to pain 2—Painful stimuli 3—Inappropriate words 1—Generally unresponsive 4—Withdraws from pain 1—No response 2—Incomprehensible sounds 3—Decorticate 1—No response 2—Decerebrate 1—No response Points Assigned to Age and Chronic Disease AGE, YEARS SCORE <45

45–54

55–64

65–74

≥75

CHRONIC HEALTH (HISTORY OF CHRONIC CONDITIONS)d SCORE None

If patient is admitted after elective surgery

If patient is admitted after emergency surgery or for reason other than after elective surgery

aThe APACHE II score is the sum of the acute physiology score (vital signs, oxygenation, laboratory values), the Glasgow coma score, age, and chronic health points. The worst values during the first 24 h in the ICU should be used. For serum creatinine, double the point score for acute renal failure. bGlasgow coma score (GCS) = eye-opening score + verbal (intubated or nonintubated) score + motor score. cFor GCS component of acute physiology score, subtract GCS from 15 to obtain points assigned. dHepatic: cirrhosis with portal hypertension or encephalopathy; cardiovascular: class IV angina (at rest or with minimal self-care activities); pulmonary: chronic hypoxemia or hypercapnia, polycythemia, ventilator dependence; renal: chronic peritoneal or hemodialysis; immune: immunocompromised host. Abbreviations: (A – a) Do2, alveolar-arterial oxygen difference; FIo2, fraction of inspired oxygen; Pao2, partial pressure of oxygen; WBC, white blood cell count.

there is increasing interest in utilizing electronic health medical record scoring systems that might better incorporate larger and real-time data sets from patients, and that can alert providers to patients at risk for sepsis and/or poor outcomes from clinical illness. SHOCK (SEE ALSO CHAP. 314) ■ ■INITIAL EVALUATION Shock, a common condition necessitating ICU admission or occurring in the course of critical care, is defined by the presence of multisys tem end-organ hypoperfusion. Clinical indicators include reduced mean arterial pressure (MAP), tachycardia, tachypnea, cool skin and extremities, acute altered mental status, and oliguria. The end result of multiorgan hypoperfusion is tissue hypoxia, often with accompanying lactic acidosis. Because the MAP is the product of cardiac output and systemic vascular resistance (SVR), reductions in blood pressure can be caused by decreases in cardiac output and/or SVR. Accordingly, once shock is contemplated, the initial evaluation of a hypotensive patient should include an early bedside assessment of the adequacy of cardiac output (Fig. 311-2). Clinical evidence of diminished car diac output includes a narrow pulse pressure (systolic blood pressure minus diastolic blood pressure)—a marker that correlates with stroke <200

Mortality rate, %

0–4 10–14 5–9 15–19 25–29 35+ 20–24 30–34 APACHE II Score FIGURE 311-1 APACHE II survival curve. Blue, nonoperative; green, postoperative. volume—and cool extremities with delayed capillary refill, colloquially termed “cold shock.” It is important to palpate proximal extremities (e.g., thigh region) rather than distal extremities to determine rela tive “coolness,” because patients with peripheral vascular disease may always have cool distal extremities. Signs of increased cardiac output include a widened pulse pressure (particularly with a reduced diastolic pressure), warm extremities with bounding pulses, and rapid capillary refill, colloquially termed “warm shock.” If a hypotensive patient has clinical signs of increased cardiac output, it can be inferred that the reduced blood pressure is from decreased SVR. SHOCK Cold, clammy extremities

Warm, bounding extremities Low cardiac output High cardiac output May convert to Septic shock, liver failure JVP, crackles JVP, orthostasis Heart is “full” (cardiogenic shock) Antibiotics, aggressive resuscitation Evaluate for myocardial ischemia Heart is “empty” (hypovolemic shock) Consider echocardiogram, invasive vascular monitoring Intravenous fluids No improvement Inotropes, afterload reduction What does not fit? Adrenal crisis, right heart syndrome, pericardial disease Consider echocardiogram, invasive vascular monitoring FIGURE 311-2 Approach to the patient in shock. JVP, jugular venous pressure.

In hypotensive patients with signs of reduced cardiac output, an assessment of intravascular volume status is appropriate. A hypoten sive patient with decreased intravascular volume status may have a history suggesting hemorrhage or other volume losses (e.g., vomiting, diarrhea, polyuria). Although evidence of a reduced jugular venous pressure (JVP) is often sought, static measures of right atrial pressure do not predict fluid responsiveness reliably; the change in right atrial pressure as a function of spontaneous respiration is a better predictor of fluid responsiveness (Fig. 311-3). Patients with fluid-responsive (i.e., hypovolemic) shock also may manifest large changes in pulse pressure as a function of respiration during mechanical ventilation (Fig. 311-4). Other bedside metrics can help with judging whether a patient remains fluid-responsive, including responses to volume chal lenge or a straight leg raise (that increases venous return) that correlate with improved perfusion. Such tools include judging changes in JVP or central venous oxygen saturation, assessing changes in pulse pres sure variation, determining changes in inferior vena cava collapse by ultrasound, and examining changes in left ventricular stroke volume using echocardiography. None of these measurements has been shown to be independently correlative, but a combination of these assessments with clinical judgment can help determine whether a patient remains volume-responsive. A hypotensive patient with increased intravascular volume and cardiac dysfunction may have S3 and/or S4 gallops on examination, increased JVP, extremity edema, and crackles on lung auscultation. The chest x-ray may show cardiomegaly, widening of the vascular pedicle, Kerley B lines, and pulmonary edema. There is increasing use of ultrasonography in place of chest radiography in resource-limited settings. Chest pain and electrocardiographic changes consistent with ischemia may be noted (Chap. 316).

CHAPTER 311 Approach to the Patient with Critical Illness In hypotensive patients with clinical evidence of increased cardiac output, a search for causes of decreased SVR is appropriate. These patients usually require targeted initial volume resuscitation (as described above), after an initial fluid bolus, to achieve euvolemia, and often require vasopressors to maintain vascular tone. The most com mon cause of high-cardiac-output hypotension is sepsis (Chap. 315). Patients with suspected sepsis should receive early broad-spectrum, appropriately dosed antibiotics and source control when feasible. Other causes of high-cardiac output hypotension include liver failure, severe pancreatitis, adrenal insufficiency, burns, trauma, anaphylaxis, thyro toxicosis, and peripheral arteriovenous shunts. Insertion of lines for monitoring and caring for critically ill patients may be necessary. Over the last two decades, management of shock has improved to the point where not all patients will require central venous and arterial lines, and recent sepsis guidelines support use of peripheral lines for pressors administration, if needed, while central access is being obtained. However, if a patient demonstrates that shock is not quickly resolving, as indicated by a persistent need for vasopres sors and/or repeated measurement of the JVP and/or central venous O2 saturation, then insertion of an arterial line for monitoring blood pressures and arterial blood gases, as well as a central venous line for administration of vasoactive agents and monitoring of the JVP

and/ or central venous O2 saturation, may be required. Ideally, lines should be inserted under sterile conditions using a protocolized checklist approach, and lines should be removed as soon as they are no longer necessary to avoid risk of line-associated infection. In summary, the most common categories of shock are hypovole mic, cardiogenic, and high-cardiac-output with decreased SVR (highoutput hypotension). Certainly, more than one category can occur simultaneously (e.g., hypovolemic and septic shock). It may often be the case that an initial presentation with septic shock can present a cardiac strain, especially in patients with underlying heart dysfunction, such that later cardiac insufficiency may arise. The initial assessment of a patient in shock should take only a few minutes. It is important that aggressive targeted resuscitation is instituted on the basis of the initial assessment, particularly since early resuscitation from septic and cardiogenic shock may improve survival (see below). If the initial bedside assessment yields equivocal or confounding data, more objective assessments such as ultrasound/ echocardiography may be useful as described above. In spontaneously

Spontaneous inspiration Pressure Time FIGURE 311-3 Right atrial pressure change during spontaneous respiration in a patient with shock whose cardiac output will increase in response to intravenous fluid administration. The right atrial pressure decreases from 7 mmHg to 4 mmHg. The horizontal bar marks the time of spontaneous inspiration. breathing patients, inferior vena cava collapse seen on ultrasound may predict a fluid-responsive state. Increasingly, ultrasound of the thorax and abdomen is used by intensivists as an extension of the physi cal examination to assess rapidly imputed filling volumes, adequacy of cardiac performance, and for indices of other specific conditions (e.g., pericardial tamponade, pulmonary embolus, pulmonary edema, pneumothorax). The goal of aggressive resuscitation is to reestablish adequate tissue perfusion and thus to prevent or minimize end-organ injury. It is equally important not to over-resuscitate patients, as it is increasingly appreciated that excess fluid resuscitation is likely not beneficial. Thus, targeted fluid resuscitation is the goal. PART 8 Critical Care Medicine ■ ■MECHANICAL VENTILATORY SUPPORT

(SEE ALSO CHAP. 313) During the initial resuscitation of patients in shock, principles of advanced cardiac life support should be followed. An early assess ment of the ability of a patient to protect their airway and to maintain adequate gas exchange is mandatory. Early intubation and mechanical ventilation often are required. Reasons for the institution of endotra cheal intubation and mechanical ventilation include acute hypoxemic respiratory failure and ventilatory failure, which frequently accompany shock. Acute hypoxemic respiratory failure may occur in patients with cardiogenic shock and pulmonary edema (Chap. 316) as well as in those who are in septic shock with pneumonia or acute respiratory dis tress syndrome (ARDS) (Chaps. 204, 312, and 315). Ventilatory failure often occurs as a consequence of an increased load on the respiratory system in the form of acute metabolic (often lactic) or respiratory aci dosis, or decreased lung compliance (e.g., from pulmonary edema or pneumonia). Inadequate perfusion to respiratory muscles in the setting of shock may be another reason for early intubation and mechanical ventilation. Normally, the respiratory muscles receive a very small per centage of the cardiac output. However, in patients who are in shock with respiratory distress, the percentage of cardiac output dedicated to respiratory muscles may increase by 10-fold or more. Lactic acid production from inefficient respiratory muscle activity can present an additional ventilatory load. Mechanical ventilation may relieve the work of breathing and allow redistribution of a limited cardiac output to other vital organs. Patients demonstrate respiratory distress by an inability to speak full sentences, accessory use of respiratory muscles, paradoxical abdominal muscle activity, extreme tachypnea (>40

breaths/min), and decreasing respiratory rate despite an increasing drive to breathe. When patients with shock are supported with mechanical ventilation, a major goal is for the ventilator to initially assume all or the majority of the work of

Time FIGURE 311-4 Pulse pressure change during mechanical ventilation in a patient with shock whose cardiac output will increase in response to intravenous fluid administration. The pulse pressure (systolic minus diastolic blood pressure) changes during mechanical ventilation in a patient with septic shock.

breathing, facilitating a state of mini mal respiratory muscle work. With the institution of mechanical ventilation for shock, further declines in MAP are frequently seen. The reasons include impeded venous return from positivepressure ventilation, reduced endog enous catecholamine secretion once the stress associated with respiratory failure abates, and the actions of drugs used to facilitate endotracheal intubation (e.g., propofol, opiates). Patients with right heart dysfunction or preexisting pulmonary hypertension may also have diminished cardiac output related to the increases in right ventric ular afterload resulting from positive-pressure ventilation. Accordingly, hypotension should be anticipated during and following endotracheal intubation. Because many of these patients may be fluid-responsive, IV volume administration should be considered, and vasopressor support peri-intubation may also be necessary. Figure 311-2 summarizes the diag nosis and treatment of different types of shock. For further discussion of individual forms of shock, see Chaps. 314, 315, and 316. RESPIRATORY FAILURE Respiratory failure is one of the most common reasons for ICU admis sion. In some ICUs, ≥75% of patients require mechanical ventilation during their stay. Respiratory failure can be categorized mechanistically on the basis of pathophysiologic derangements in respiratory function. ■ ■TYPE I: ACUTE HYPOXEMIC

RESPIRATORY FAILURE This type of respiratory failure occurs with alveolar flooding and sub sequent ventilation-perfusion mismatch and intrapulmonary shunt physiology. Alveolar flooding may be a consequence of pulmonary edema, lung injury, pneumonia, or alveolar hemorrhage. Pulmonary edema can be further categorized as occurring due to elevated pul monary microvascular pressures, as seen in heart failure and intravas cular volume overload or ARDS (“low-pressure pulmonary edema,” Chap. 312). This syndrome is defined by acute onset (≤1 week) of bilateral opacities on chest imaging that are not fully explained by car diac failure or fluid overload and often includes ventilation-perfusion mismatch and shunt physiology requiring positive end-expiratory pressure (PEEP). A new global definition for ARDS has been proposed that does not rely upon chest radiography, arterial blood gases, or use of ventilatory support with PEEP. Type I respiratory failure occurs in clinical settings such as sepsis, gastric aspiration, pneumonia, COVID-19 (Chap. 205), near-drowning, multiple blood transfusions, and pancre atitis. The mortality rate among patients with ARDS was traditionally very high (50–70%), although changes in patient care have led to mortality rates closer to 30% (see below). The COVID-19 pandemic resulted in a substantially increased incidence of viral-mediated ARDS. It is well established that mechanical ventilation of patients with ARDS may propagate lung injury. As seen in Fig. 311-5, the pressurevolume relationship of the lung in ARDS is not linear. Alveoli may collapse at very low lung volumes. Animal studies have suggested that repeated stretching and overdistention of injured alveoli dur ing mechanical ventilation can further injure the lung. Concern over this alveolar overdistention, termed ventilator-induced “volutrauma,” led to a multicenter, randomized, prospective trial comparing traditional ventilator strategies for ARDS (large tidal volume: 12 mL/kg

of ideal body weight) with a low tidal volume (6 mL/kg of ideal body weight). This study showed a dramatic reduction in mortality rate in the low-tidal-volume group from that in the high-tidal-volume group (31 vs 39.8%). Other studies have suggested that large tidal volumes may lead to ARDS in patients who initially do

Pressure (mmHg)

Alveoli D

C Upper inflection point Volume, mL

B Lower inflection point A

Pressure, cmH2O FIGURE 311-5 Pressure-volume relationship in the lungs of a patient with acute respiratory distress syndrome (ARDS). At the lower inflection point, collapsed alveoli begin to open and lung compliance changes. At the upper deflection point, alveoli become overdistended. The shape and size of alveoli are illustrated at the top of the figure. not have this problem. Prone positioning has been shown to improve survival in those with severe ARDS and has been more broadly applied in many centers in COVID-19 ARDS. Select patients may benefit from neuromuscular blockade in ARDS. In addition, a “fluid-conservative” management strategy (maintaining a low central venous pressure [CVP] or pulmonary capillary wedge pressure [PCWP]) is associated with fewer days of mechanical ventilation than a “fluid-liberal” strat egy (maintaining a relatively high CVP or PCWP) in ARDS in those patients who have been resuscitated from shock. There is growing interest in avoiding intubation in patients with ARDS by the use of a variety of devices, such as masks, high-flow oxygen delivery systems, and helmets for respiratory support that were more broadly applied during the COVID pandemic and likely more so in resource-limited settings when feasible; however, this must be balanced by concern that higher tidal volumes during spontaneous breathing with these devices could result in progression of preexisting lung injury. ■ ■TYPE II RESPIRATORY FAILURE:

HYPERCAPNEIC RESPIRATORY FAILURE This type of respiratory failure is a consequence of alveolar hypoventi lation and results from the inability to eliminate carbon dioxide effec tively. Mechanisms are categorized by impaired central nervous system (CNS) drive to breathe (colloquially termed, “won’t breathe”), impaired strength with failure of neuromuscular function in the respiratory sys tem, and increased load(s) on the respiratory system (with the latter two colloquially termed, “can’t breathe”). Reasons for diminished CNS drive to breathe include drug overdose, brainstem injury, sleep-disordered breathing, and severe hypothyroidism. Reduced strength can be due to impaired neuromuscular transmission (e.g., myasthenia gravis, Guillain-Barré syndrome, amyotrophic lateral sclerosis) or respiratory muscle weakness (e.g., myopathy, electrolyte derangements, fatigue). The overall load on the respiratory system can be subclassified into resistive loads (e.g., bronchospasm), loads due to reduced lung compli ance (e.g., alveolar edema, atelectasis, intrinsic positive end-expiratory pressure [auto-PEEP]—see below), loads due to reduced chest wall compliance (e.g., pneumothorax, pleural effusion, abdominal disten tion), and loads due to increased minute ventilation requirements (e.g., pulmonary embolus with increased dead-space fraction, sepsis). The mainstays of therapy for hypercapnic respiratory failure are directed at reversing the underlying cause(s) of ventilatory failure. Noninvasive positive-pressure

ventilation with a tight-fitting facial or nasal mask, with avoidance of endotracheal intubation, may stabilize these patients in certain circumstances. This approach has been shown to be beneficial in treating patients with exacerbations of chronic obstructive pulmonary disease; it has been tested less extensively in

other kinds of respiratory failure but may be attempted nonetheless with close monitoring in the absence of contraindications (e.g., hemo dynamic instability, inability to protect the airway, respiratory arrest, significant airway secretions, significant aspiration risk).

■ ■TYPE III RESPIRATORY FAILURE:

LUNG ATELECTASIS This form of respiratory failure results from lung atelectasis. Because atelectasis occurs so commonly in the perioperative period, this form is also called perioperative respiratory failure. After general anesthesia, decreases in functional residual capacity lead to collapse of dependent lung units. Such atelectasis can be treated by frequent changes in posi tion, chest physiotherapy, upright positioning, and control of incisional and/or abdominal pain. Noninvasive positive-pressure ventilation may also be used to reverse regional atelectasis. ■ ■TYPE IV RESPIRATORY FAILURE:

METABOLIC DEMANDS This form most often results from hypoperfusion of respiratory muscles in patients in shock. Normally, respiratory muscles consume <5% of total cardiac output and oxygen delivery. Patients in shock often experience respiratory distress due to pulmonary edema (e.g., in car diogenic shock), lactic acidosis, and anemia. In this setting, up to 40% of cardiac output may be distributed to the respiratory muscles. Intuba tion and mechanical ventilation can allow redistribution of the cardiac output away from the respiratory muscles and back to vital organs while the shock is treated. In addition, other causes of significant metabolic acidosis might require ventilatory support while reversal of the underlying cause of the acidosis is addressed. CHAPTER 311 Approach to the Patient with Critical Illness CARE OF THE MECHANICALLY VENTILATED PATIENT Mechanically ventilated patients frequently require sedatives and anal gesics. Opiates are the mainstay of therapy for analgesia in mechani cally ventilated patients. After adequate pain control has been ensured, additional indications for sedation include anxiolysis; treatment of subjective dyspnea; reduction of autonomic hyperactivity, which may precipitate myocardial ischemia; and reduction of total O2 consump tion (Vo2). Nonbenzodiazepine sedatives are preferred because ben zodiazepines are associated with increased delirium and worse patient outcomes. The neuromuscular blocking agent cisatracurium is occasionally used to facilitate mechanical ventilation in patients with profound ventilator dyssynchrony despite optimal sedation, particularly in the setting of severe ARDS. Use of these agents may result in prolonged weakness—a myopathy known as the postparalytic syndrome. For this reason, neuromuscular blocking agents typically are used as a last resort when aggressive sedation fails to achieve patient–ventilator synchrony. Because neuromuscular blocking agents result in phar macologic paralysis without altering mental status, sedative-induced amnesia is mandatory when these agents are administered. Amnesia can be achieved reliably with propofol and benzodi azepines such as lorazepam and midazolam. Outside the setting of pharmacologic paralysis, few data support the idea that amnesia is mandatory in all patients who require intubation and mechanical ventilation. Because many of these critically ill patients have impaired hepatic and renal function, sedatives and opiates may accumulate when given for prolonged periods. A nursing protocol–driven approach to sedation of

mechanically ventilated patients or daily interruption of sedative infusions paired with daily spontaneous breathing trials has been shown to prevent excessive drug accumulation and shorten the duration of both mechanical ventilation and ICU stay (see below). (See also Chap. 313.) Whereas a thorough understanding of the pathophysiology of respiratory failure is essential for optimal patient care, recognition of a patient's readiness to be liberated from mechani cal ventilation is likewise important. Several studies have shown that daily spontaneous breathing trials can identify patients who are ready for extubation. Accordingly, all intubated, mechanically ventilated patients should undergo daily screening of respiratory function.

If oxygenation is stable (i.e., Pao2/FIo2 [partial pressure of oxygen/ fraction of inspired oxygen] >200 and PEEP ≤5 cmH2O), cough and airway reflexes are intact, and no vasopressor agents or sedatives are being administered, the patient has passed the screening test and should undergo a spontaneous breathing trial (SBT). If sedatives are being administered, the patient can undergo a spontaneous awak ening trial (SAT), as well, to determine if they are able to maintain adequate alertness and respiratory status without sedatives. The SAT/ SBT trial consists of a period of breathing through the endotracheal tube without significant ventilator support (continuous positive airway pressure [CPAP] of 5 cmH2O with or without low-level pressure sup port [e.g., 5 cmH2O] and an open T-piece breathing system have all been validated) for 30–120 min. The spontaneous breathing trial is declared a failure and stopped if any of the following occur: (1) respiratory rate

> 35/min for >5 min, (2) O2 saturation <90%, (3) heart rate >140/min or a 20% increase or decrease from baseline, (4) systolic blood pressure <90 mmHg or >180 mmHg, or (5) increased anxiety or diaphoresis. If, at the end of the spontaneous breathing trial, none of the above events has occurred, the patient can be considered for an extubation trial. Such protocol-driven approaches to patient care can have an important impact on the duration of mechanical ventilation and ICU stay. Despite such a careful approach to liberation from mechanical ventilation, up to 10% of patients develop respiratory distress after extubation and may require resumption of mechanical ventilation. Many of these patients will require reintubation. The use of noninvasive ventilation as a res cue strategy in patients in whom extubation fails may be associated in some patients with worse outcomes than are obtained with immediate reintubation. Some studies suggest that there are subgroups of patients who might benefit from administration of noninvasive ventilation or high-flow nasal oxygen therapy upon extubation, as it is believed that low levels of PEEP and/or inspiratory flow delivered by these devices after extubation may be helpful.

PART 8 Critical Care Medicine MULTIORGAN SYSTEM FAILURE Multiorgan system failure, which is commonly associated with critical illness, is defined by the simultaneous presence of physiologic dysfunc tion and/or failure of two or more organs. Typically, this syndrome occurs in the setting of severe sepsis, shock of any kind, severe inflam matory conditions such as pancreatitis, and trauma. The fact that multiorgan system failure occurs commonly in the ICU is a testament to our current ability to stabilize and support single-organ failure. The ability to support single-organ failure aggressively (e.g., by mechanical ventilation or by renal replacement therapy) has reduced rates of

early mortality in critical illness. As a result, it is less common for critically ill patients to die in the initial stages of resuscitation. Instead, many patients succumb to critical illness later in the ICU stay, after the initial presenting problem may have been stabilized. Although there is debate regarding specific definitions of organ fail ure, several general principles governing the syndrome of multiorgan system failure apply. First, organ failure, no matter how it is defined, must persist beyond 24 h. Second, mortality risk increases with the accrual of failing organs. Third, the prognosis worsens with increased duration of organ failure. These observations remain true across vari ous critical care settings (e.g., medical vs surgical). MONITORING IN THE ICU Because respiratory failure and circulatory failure are common in critically ill patients, monitoring of the respiratory and cardiovascu lar systems is undertaken frequently. Evaluation of respiratory gas exchange is routine in critical illness. The “gold standard” remains arte rial blood-gas analysis, in which pH, Pao2, partial pressure of carbon dioxide (Pco2), and O2 saturation are measured directly. With arterial blood-gas analysis, the two main functions of the lung—oxygenation of arterial blood and elimination of CO2—can be assessed directly. In fact, the arterial blood pH, which has a profound effect on the drive to breathe, can be assessed only by such sampling. Venous pH obtained through an indwelling central venous line can approximate arterial pH. Although sampling of arterial blood is generally safe and may be undertaken more frequently through insertion of a temporary

indwelling arterial line, it may be painful and cannot provide continu ous information. In light of these limitations, noninvasive monitoring of respiratory function is often employed. ■ ■PULSE OXIMETRY The most commonly utilized noninvasive technique for monitoring respiratory function, pulse oximetry takes advantage of differences in the absorptive properties of oxygenated and deoxygenated hemo globin. At wavelengths of 660 nm, oxyhemoglobin reflects light more effectively than does deoxyhemoglobin, whereas the reverse is true in the infrared spectrum (940 nm). A pulse oximeter passes both wavelengths of light through a perfused digit such as a finger, and the relative intensity of light transmission at these two wavelengths is recorded. From this information, the relative percentage of oxyhe moglobin is derived. Since arterial pulsations produce phasic changes in the intensity of transmitted light, the pulse oximeter is designed to detect only light of alternating intensity. This feature allows distinction of arterial and venous blood O2 saturations. Studies during the COVID pandemic noted that pulse oximetry overestimates oxygen saturation in patients with darker skin, thus making correlation with arterial Pao2 more imperative. ■ ■RESPIRATORY SYSTEM MECHANICS Respiratory system mechanics can be measured in patients during mechanical ventilation (Chap. 313). When volume-controlled modes of mechanical ventilation are used, accompanying airway pressures can easily be measured as long as the patient is breathing passively. The peak airway pressure is determined by two variables: airway resistance and respiratory system compliance. At the end of inspira tion, inspiratory flow can be stopped transiently. This end-inspiratory pause (plateau pressure) is a static measurement, affected only by respiratory system compliance and not by airway resistance. There fore, during volume-controlled ventilation, the difference between the peak (airway resistance + respiratory system compliance) and plateau (respiratory system compliance only) airway pressures pro vides a quantitative assessment of airway resistance. Accordingly, dur ing volume-controlled ventilation, patients with increases in airway resistance typically have increased peak airway pressures as well as abnormally high gradients between peak and plateau airway pressures (typically >10–15 cmH2O) at a constant inspiratory flow rate of 1 L/s. The compliance of the respiratory system is defined by the change in volume of the respiratory system per unit change in pressure; thus, a

quantitative assessment of compliance is provided by the tidal volume divided by the plateau pressure minus PEEP. The respiratory system can be divided into two components: the lungs and the chest wall. Normally, respiratory system compliance is ~100 mL/cmH2O. Pathophysiologic processes such as pleural effu sions, pneumothorax, and increased abdominal girth all reduce chest wall compliance. Lung compliance may be reduced by pneumonia, pulmonary edema, alveolar hemorrhage, interstitial lung disease, or auto-PEEP. Accordingly, patients with abnormalities in compliance of the respiratory system (lungs and/or chest wall) typically have elevated peak and plateau airway pressures but a normal gradient between these two pressures. Auto-PEEP occurs when there is insufficient time for emptying of alveoli before the next inspiratory cycle. Because the alveoli have not decompressed completely, alveolar pressure remains positive at the end of exhalation (functional residual capacity). This phenomenon results most commonly from obstruction of distal airways in disease processes such as asthma and COPD. Auto-PEEP with resulting alveolar overdistention may result in diminished lung compliance, reflected by abnormally increased plateau airway pres sures. Modern mechanical ventilators allow breath-to-breath display of pressure and flow, permitting detection of potential problems such as patient–ventilator dyssynchrony, airflow obstruction, and auto-PEEP (Fig. 311-6). ■ ■CIRCULATORY STATUS Oxygen delivery (Qo2) is a function of cardiac output and the content of O2 in the arterial blood (Cao2). The Cao2 is determined by the

cmH2O Pressure–Time

L/s Flow–Time 1.2

–1.2 FIGURE 311-6 Increased airway resistance with auto–positive end-expiratory pressure (PEEP). The top waveform (airway pressure vs time) shows a large difference between the peak airway pressure (80 cmH2O) and the plateau airway pressure (20 cmH2O). The bottom waveform (flow vs time) demonstrates airflow throughout expiration (reflected by the flow tracing on the negative portion of the abscissa) that persists up to the next inspiratory effort. hemoglobin concentration, the arterial hemoglobin saturation, and dissolved O2 not bound to hemoglobin. For normal adults: Qo2 = 50 dL/min × (1.39 × 15 g/dL [hemoglobin concentration]

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× 1.0 [hemoglobin % saturation] + 0.0031 × 10 [Pao2])
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= 50 dL/min (cardiac output) × 21.6 mL O2 per dL blood (Cao2)

= 1058 mL O2 per min It is apparent that nearly all the O2 delivered to tissues is bound to hemoglobin and that the dissolved O2 (Pao2) contributes very little to O2 content in arterial blood or to O2 delivery. Normally, the content of O2 in mixed venous blood (C–vo2) is 15.76 mL/dL since the mixed venous blood is 75% saturated. Therefore, the normal tissue extraction ratio for O2 is Cao2 – C–vo2/Cao2 ([21.16 – 15.76]/21.16) or ~25%. A pulmonary artery catheter (see discussion below) allows measurements of O2 delivery and the O2 extraction ratio. Information on the venous O2 saturation allows assessment of global tissue perfusion. A reduced venous O2 saturation may be caused by inad equate cardiac output, reduced hemoglobin concentration, and/or reduced arterial O2 saturation. An abnormally high oxygen consumption (Vo2) may also lead to a reduced venous O2 saturation if O2 delivery is not concomi tantly increased. Abnormally increased Vo2 in peripheral tissues may be caused by problems such as fever, agitation, shivering, and

thyrotoxicosis. The pulmonary artery catheter originally was designed as a tool to guide therapy for acute myocardial infarction but has been used in the ICU for evaluation and treatment of a variety of other conditions, such as ARDS, septic shock, congestive heart failure, and acute renal failure. This device has never been validated as a tool associated with reduction in morbidity and mortality rates. Indeed, despite numerous prospec tive studies, mortality or morbidity rate benefits associated with use of the pulmonary artery catheter have never been reported in any setting. Accordingly, it appears that routine pulmonary artery catheterization is not indicated as a means of monitoring and characterizing circulatory status in most critically ill patients, especially as monitoring of the venous O2 saturation via an indwelling central venous line has proven helpful in many critical illness settings. However, there are still select circumstances where pulmonary artery catheterization may prove helpful when used by those with appropriate experience in its insertion and data interpretation. PREVENTION OF COMPLICATIONS OF CRITICAL ILLNESS ■ ■SEPSIS IN THE CRITICAL CARE UNIT (See also Chap. 315) Sepsis is defined as life-threatening organ dys function (i.e., an increase in SOFA of 2 points or more) caused by a

dysregulated response to infection. Poor outcomes can be anticipated in patients with two or more of the following: respiratory rate ≥22 breaths/min, altered mentation, and systolic blood pressure ≤100 mmHg. Sepsis is a leading cause of death in noncoronary ICUs in the United States, with case rates expected to increase as the population ages and a higher percentage of people are vulnerable to infection.

■ ■NOSOCOMIAL INFECTIONS IN THE ICU Many therapeutic interventions in the ICU are invasive and predis pose patients to infectious complications. These interventions include endotracheal intubation, indwelling vascular catheters, transurethral bladder catheters, and other catheters placed into sterile body cavi ties (e.g., tube thoracostomy, percutaneous intraabdominal drainage catheterization). The longer such devices remain in place, the more prone to infections patients become from these devices. For example, ventilator-associated events such as ventilator-associated pneumonia correlate strongly with the duration of intubation and mechanical ven tilation. Therefore, an important aspect of preventive care is the timely removal of invasive devices as soon as they are no longer needed. Moreover, multidrug-resistant organisms are commonplace in the ICU.

CHAPTER 311 Infection control is critical in the ICU. Care bundles, which include measures such as frequent hand washing, are effective but underuti lized strategies. Other components of care bundles, such as protective isolation of patients colonized or infected by drug-resistant organisms, are also commonly used. Studies evaluating multifaceted, evidencebased strategies to decrease catheter-related bloodstream infections have shown improved outcomes with strict adherence to measures such as hand washing, full-barrier precautions during catheter inser tion, chlorhexidine skin preparation, avoidance of the femoral site, and timely catheter removal. Approach to the Patient with Critical Illness ■ ■DEEP-VENOUS THROMBOSIS (DVT)

(SEE ALSO CHAP. 290) All ICU patients are at high risk for this complication because of their predilection for immobility. Therefore, all should receive some form of prophylaxis against DVT if feasible. The most commonly employed forms of prophylaxis are subcutaneous chemoprophylaxis (e.g., lowdose heparin) injections and sequential compression devices for the lower extremities. Observational studies report an alarming incidence of DVTs despite the use of these standard

prophylactic regimens. Fur thermore, heparin prophylaxis may result in heparin-induced throm bocytopenia, another nosocomial complication in critically ill patients. Low-molecular-weight heparins such as enoxaparin are more effec tive than unfractionated heparin for DVT prophylaxis in high-risk patients (e.g., those undergoing orthopedic surgery) and are associ ated with a lower incidence of heparin-induced thrombocytopenia, although their use may be limited in patients with renal dysfunction given their renal clearance. ■ ■STRESS ULCERS Prophylaxis against stress ulcers is not necessary for all ICU patients. It should only be administered to high-risk patients, such as those with coagulopathy or respiratory failure requiring mechanical ventilation. While there has been debate about the optimal agent for stress ulcer prophylaxis, a number of recent studies have supported improved efficacy of proton pump inhibitors (PPIs) in reducing bleeding risk compared with other agents (e.g., histamine-2 receptor antagonist [H2 blocker] or sucralfate). There exist concerns for increased risk of pneu monia and Clostridium difficile colitis with PPIs compared with other agents, although the data are not definitive, and the improved efficacy of PPIs in patients at high risk for stress ulcers may outweigh these potential infectious risks. ■ ■NUTRITION AND GLYCEMIC CONTROL Nutrition and glycemic control are important issues that may be asso ciated with respiratory failure, impaired wound healing, and dysfunc tional immune response in critically ill patients. Early enteral feeding is reasonable, with some data suggesting that permissive underfeeding of nonprotein calories is not inferior to full-goal feeding. Certainly, enteral feeding, if possible, is preferred over parenteral nutrition, which

is associated with numerous complications, including hyperglycemia, fatty liver, cholestasis, and sepsis. When parenteral feeding is necessary to supplement enteral nutrition, delaying this intervention until day 8 in the ICU results in better recovery and fewer ICU-related complica tions. Tight glucose control has been an area of controversy in critical care. Although one study showed a significant mortality benefit when glucose levels were aggressively normalized in a large group of surgical ICU patients, other studies of both medical and surgical ICU patients suggested that tight glucose control resulted in increased rates of mortality likely attributable, in part, to hypoglycemic episodes. Thus, current guidelines suggest targeting glucose levels of ≤180 mg/dL in critically ill patients, rather than targeting tighter control.

■ ■ICU-ACQUIRED WEAKNESS ICU-acquired weakness occurs frequently in patients who survive critical illness. Both neuropathies and myopathies have been described, most commonly after ~1 week in the ICU. The mechanisms behind ICU-acquired weakness syndromes are poorly understood, and they are known to present with heterogeneous muscle pathophysiol ogy. Very early physical and occupational therapy in mechanically ventilated patients reportedly results in significant improvements in functional independence at hospital discharge as well as in reduced durations of mechanical ventilation and delirium. PART 8 Critical Care Medicine ■ ■ANEMIA Studies have shown that most ICU patients are anemic as a result of chronic inflammation. Phlebotomy also contributes to ICU anemia. A large multicenter study involving patients in many different ICU set tings challenged the conventional notion that a hemoglobin level of 100 g/L (10 g/dL) is needed in critically ill patients, with similar out comes noted in those whose transfusion trigger was 7 g/dL. Red blood cell transfusion is associated with impairment of immune function and increased risk of infections as well as of ARDS and volume overload, all of which may explain the findings in this study. A conservative transfu sion strategy has shown similar outcomes in septic shock, postcardiac surgery, and post–hip surgery patients. ■ ■ACUTE KIDNEY FAILURE (See also Chap. 321) Acute kidney failure occurs in a significant per centage of critically ill patients. The

most common underlying etiology is acute tubular necrosis, usually precipitated by hypoperfusion and/or nephrotoxic agents. Currently, no pharmacologic agents are available for prevention of kidney injury in critical illness. Studies have shown convincingly that low-dose dopamine, fenoldopam, and vasopressin are not effective in protecting the kidneys from acute injury. NEUROLOGIC DYSFUNCTION IN CRITICALLY ILL PATIENTS ■ ■DELIRIUM (See also Chap. 29) Delirium is defined by (1) an acute onset of changes or fluctuations in mental status, (2) inattention, (3) disorga nized thinking, and (4) an altered level of consciousness (i.e., a state other than alertness). Delirium is reported to occur in a wide range of mechanically ventilated ICU patients and can be detected by the Confusion Assessment Method for the ICU (CAM-ICU) or the Inten sive Care Delirium Screening Checklist (ICDSC). These tools are used to ask patients to answer simple questions and perform simple tasks and can be used readily at the bedside. The differential diagnosis of delirium in ICU patients is broad and includes infectious etiologies (including sepsis), medications (particularly sedatives and analgesics), drug withdrawal, metabolic/electrolyte derangements, intracranial pathology (e.g., stroke, intracranial hemorrhage), seizures, hypoxia, hypertensive crisis, shock, and vitamin deficiencies (particularly thia mine). The etiology of a patient's ICU delirium impacts the prognosis. Those with persistent ICU delirium not related to sedatives have increases in length of hospital stay, time on mechanical ventilation, cognitive impairment at hospital discharge, and 6-month mortality rate. Interventions to reduce ICU delirium are limited. The sedative dexmedetomidine has been less strongly associated with ICU delirium

than midazolam in some studies. In addition, very early physical and occupational therapy in mechanically ventilated patients has been demonstrated to reduce delirium. ■ ■ANOXIC CEREBRAL INJURY (See also Chap. 318) This condition is common after cardiac arrest and often results in severe and permanent brain injury in survivors. Active cooling of patients to 33°C after cardiac arrest is controversial, with some studies showing improved neurologic outcomes and oth ers showing no such improvement when compared to maintaining normothermia. Certainly, patients post cardiac arrest should have a temperature targeted to no higher than normothermia. ■ ■STROKE (See also Chap. 437) Stroke is a common cause of neurologic critical illness. Hypertension must be managed carefully, because abrupt reduc tions in blood pressure may be associated with further brain ischemia and injury. Acute ischemic stroke treated with tissue plasminogen activator (tPA) has an improved neurologic outcome when treatment is given within 4.5 h of onset of symptoms, with likely increased benefit associated with earlier administration. The mortality rate is not reduced when tPA is compared with placebo, despite the improved neurologic outcome. The risk of cerebral hemorrhage is significantly higher in patients given tPA. No consistent overall benefit is seen when tPA ther apy is given beyond 4.5 h after symptom onset. Heparin has not been convincingly shown to improve outcomes in patients with acute isch emic stroke. Decompressive craniectomy is a surgical procedure that relieves increased intracranial pressure in the setting of space-occupying brain lesions or brain swelling from stroke; available evidence suggests that this procedure may improve survival among select patients (e.g., ≤55 years of age), albeit at a cost of increased disability for some. ■ ■SUBARACHNOID HEMORRHAGE (See also Chap. 437) Subarachnoid hemorrhage may occur secondary to aneurysm rupture and is often complicated by cerebral vasospasm, re-bleeding, and hydrocephalus. Vasospasm can be detected by either transcranial Doppler assessment or cerebral angiography; it is typi cally treated with the calcium channel blocker nimodipine, aggressive IV fluid administration to avoid hypovolemia, and therapy aimed at maintaining adequate central perfusion pressure, typically with vaso active drugs such as phenylephrine. IV fluids and vasoactive drugs (hypertensive

hypervolemic therapy) are used to overcome the cerebral vasospasm. Early surgical clipping or endovascular coiling of aneu rysms is advocated to prevent complications related to re-bleeding. Hydrocephalus, typically heralded by a decreased level of conscious ness, may require ventriculostomy drainage. ■ ■STATUS EPILEPTICUS (SEE ALSO CHAP. 436) Recurrent or relentless seizure activity is a medical emergency. Ces sation of seizure activity is required to prevent irreversible neurologic injury. Lorazepam is the most effective benzodiazepine for treating status epilepticus and is the treatment of choice for controlling seizures acutely. Maintenance of seizure control should be effected with a load ing dose of fosphenytoin, valproate, or levetiracetam, as these agents have been shown to have similar efficacy and side effects. ■ ■BRAIN DEATH (See also Chap. 318) Although deaths of critically ill patients usually are attributable to irreversible cessation of circulatory and respiratory function, a diagnosis of death also may be established by irreversible cessation of all functions of the entire brain, including the brainstem, even if circulatory and respiratory functions remain intact on artificial life support. Such a diagnosis requires demonstration of the absence of cerebral function (no response to any external stimulus) and brain stem functions (e.g., unreactive pupils, lack of ocular movement in response to head turning or ice-water irrigation of ear canals, positive apnea test [no drive to breathe]). Many U.S. institutions have a protocol based upon their state’s requirements for declaration of brain death. Absence of brain function must have an established cause and be per manent without possibility of recovery; a sedative effect, hypothermia,

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