Chapter 2

Chronic Critical Illness in Geriatric Patients

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Key Points

- Chronic critical illness is used to describe patients who survive the initial acute episode of critical illness but persistently remain dependent on intensive care.
- It is typically defined as need for mechanical ventilation for more than 6 hours per day for more than 21 consecutive days with concurrent neurologic changes, endocrine alterations, muscle wasting, predisposition to infection, and changes in body composition, including loss of lean body mass.
- Elderly patients are especially vulnerable to develop chronic critical illness, with its prevalence peaking from 75 to 79 years of age.
- Chronic critical illness involves systemic derangement of immunologic function, persistent inflammation, neurocognitive issues, endocrine imbalance, malnutrition, and muscle wasting.
- Due to elevated levels of catecholamine and glucocorticoids, the metabolism is shifted to the catabolic phase. There is a marked decrease in the pulsatile secretion of anterior pituitary hormones.
- A majority of critically ill patients suffer from neuromuscular weakness, which is broadly classified into critical illness polyneuropathy (CIP), critical illness myopathy (CIM), and combined CIM/CIP.
- Despite the significant burden on the healthcare system afforded by chronic critical illness, there is a lack of guideline-based recommendations regarding management of this patient cohort.

Introduction

Over the last few decades, with significant advances in the field of critical care, the overall mortality of acutely critically ill patients has decreased. These advances include life-sustaining measures that provide artificial support to organs while the patient is recovering from the acute insult [1]. However, a new patient population has emerged that remains dependent on intensive services for their survival for prolonged periods of time. This population is referred to as *chronically critically ill*.

The term *chronically critically ill* was first used by Girard et al. in 1985 to describe patients who survived the initial acute episode of critical illness but persistently remained dependent on intensive care [2,3]. Despite wide recognition of this syndrome, there seems

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to be a lack of consensus regarding its concrete definition [4]. However, most experts agree that prolonged mechanical ventilation, defined as the need for mechanical ventilation for more than 6 hours per day for more than 21 consecutive days, is a hallmark of this disease [1,5]. Other manifestations of this syndrome include neurologic changes, endocrine alterations, muscle wasting, predisposition to infection, and changes in body composition, including loss of lean body mass.

Epidemiology

With improving survival from acute critical illness, the overall incidence of chronic critical illness (CCI) is on the rise. According to recent estimates, CCI has an overall population-based prevalence of 34.4 per 100,000 [6]. Elderly patients are especially vulnerable to develop CCI, with its prevalence peaking from 75 to 79 years of age. There is a decline of CCI among patients older than 80 years because of a higher early mortality among otherwise eligible patients (Figure 2.1).

Chronic critical illness incurs a tremendous clinical and financial burden, with estimated healthcare cost of $10 billion [7]. Despite meticulous and highly skilled care, CCI is associated with poor long-term survival [8], with 20 percent of surviving patients having residual physical and cognitive impairments and less than 10 percent ever returning home after hospitalization [9]. Moreover, with a rise in the aging population and improvements in management of acute illnesses, the incidence of CCI is expected to increase further [10]. Not surprisingly, CCI has been receiving increasing attention from researchers, healthcare providers, and health policymakers. However, strategies to prevent and improve outcomes in patients with CCI still remain clinically challenging.

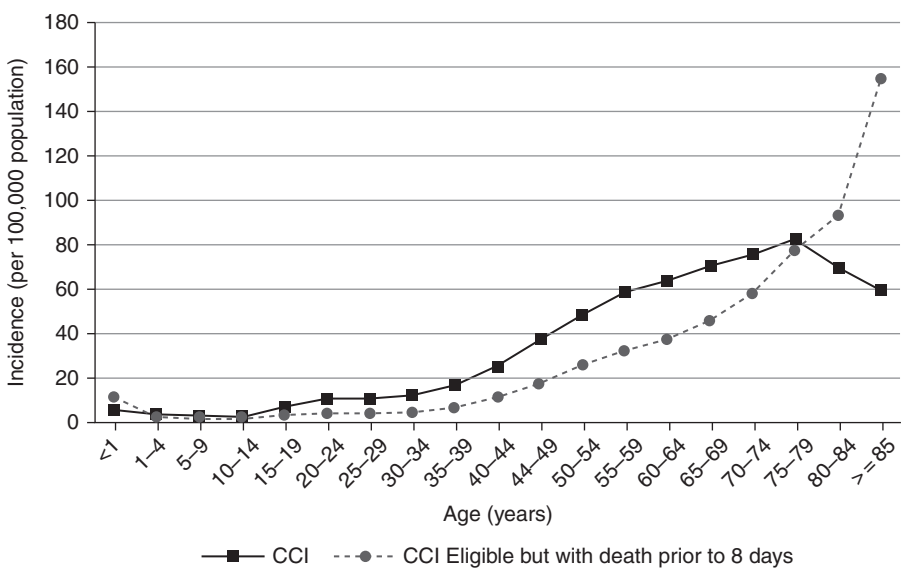

Figure 2.1 Age-specific population-based prevalence of CCI (dark line) and CCI-eligible conditions but with death prior to 8 days (dashed line). Data are for the five-state sample, all years. (*Source:* From Kahn et al. [6].)

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Pathophysiology of Chronic Critical Illness

It is estimated that between 5 and 10 percent of patients with acute critical illness who require mechanical ventilation progress to CCI [3,11,12]. The presence of declining baseline organ function and multiple comorbidities predisposes geriatric patients to prolonged mechanical ventilation and CCI. Although various risk factors [11,13–15] and models [12,16,17] for predicting prolonged mechanical ventilation have been described, their applicability in the geriatric population remains to be validated. Additionally, there are no biomarkers to predict CCI. CCI involves systemic derangement of immunologic function, persistent inflammation, neurocognitive issues, endocrine imbalance, malnutrition, and muscle wasting.

Persistent Inflammation, Immunosuppression, and Catabolism

Patients who develop CCI show signs of persistent systemic inflammatory state. They develop a dysregulated response after an acute insult, which leads to the release of pro-inflammatory cytokines such as interleukin-6 and interleukin-8, which continue to be elevated even after resolution of the initial insult [9]. There are persistently increased levels of circulating glucocorticoids, catecholamines, and prostaglandins, leading to further amplification of the inflammatory milieu.

Enhanced levels and prolonged inflammation lead directly to immunosuppression [18]. There is a marked downregulation of antigen receptors at the cell surface and cell signaling pathways, chemotaxis, antigen presentation, and phagocytosis, which hamper the efficacy of the immune response [19–23]. Not surprisingly, CCI patients have been shown to have absolute lymphocyte depletion, decreased antibody per bound cell, and T-cell downregulation, which manifest clinically as an increased incidence of pneumonia and other nosocomial infections [24].

Due to elevated levels of catecholamine and glucocorticoids, the metabolism is shifted to the catabolic phase [25,26]. This leads to a profound change in body composition and a reduction in lean body mass despite adequate nutritional supplementation [27].

This model of maladaptive body response leading to persistent inflammation, immunosuppression, and catabolic syndrome (PICS) was proposed by Gentile et al. [28]. They proposed that a patient meets PICS criterion if residing in the intensive care unit (ICU) for at least 10 days and having persistent inflammation defined by a C-reactive protein concentration of greater than 150 μg/dl and a retinol-binding protein concentration of less than 10 μg/dl, immunosuppression crudely defined by a total lymphocyte count of less than 800/mm^3, and a catabolic state defined by a serum albumin concentration of less than 3.0 g/dl, a creatinine height index of less than 80 percent, and weight loss greater than 10 percent or body mass index (BMI) less than 18, during the current hospitalization.

Geriatric patients are extremely susceptible to developing PICS. They have a abnormal inflammatory state at baseline, often referred to as *inflamm-aging* [29], and are particularly prone to this dysfunctional cytokine response after an acute insult [30]. This is further exacerbated by immunosenescence, which is characterized by multiple immune-related disorders concomitant with aging [31] (see Chapter 10). It includes decreased number of Langerhans cells, impaired neutrophil and macrophage function, decreased T-cell activation, and decreased cytotoxicity by natural killer (NK) cells [32–38]. Finally, underlying nutritional deficiencies in the elderly are further exacerbated by this state of catabolism accelerating sarcopenia and leading to cachexia [27] (Figure 2.2).

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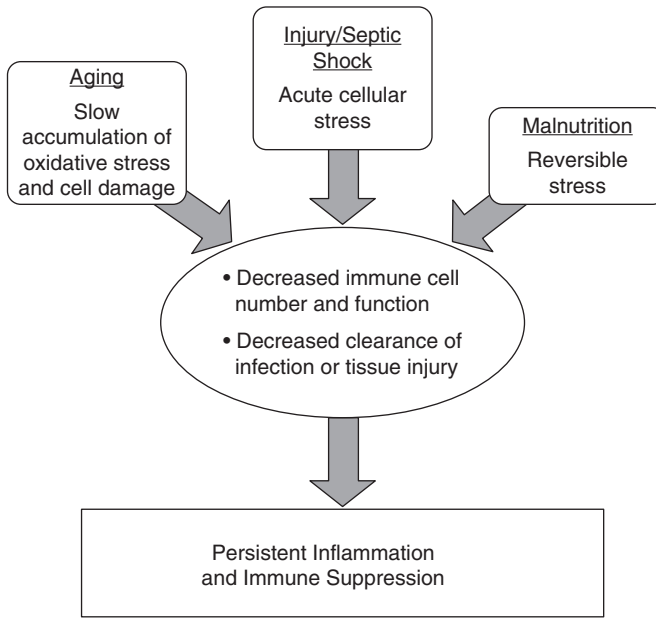

Figure 2.2 Hypothetical representation of the interaction of aging, severe injury, sepsis, and malnutrition and the development of persistent inflammation and immune suppression. (*Source:* From Nomellini et al. [9].)

Clinical Manifestations

In addition to respiratory failure requiring prolonged mechanical ventilation, this syndrome is characterized by a distinct pattern of multiorgan dysfunction. This includes neurologic changes, endocrine alterations, myopathy, loss of lean body mass, and increased susceptibility to infection.

Neurologic Changes

Most patients with CCI suffer from severe neurocognitive dysfunction, with half the survivors being comatose or delirious at hospital discharge [39]. Of these, a majority continue to have neurocognitive deficits at six-months (79 percent) and one-year (71 percent) follow-up [40]. In addition to delirium, neurocognitive issues in patients with CCI include impairments in attention, memory, and executive function [41]. Elderly patients, those with a longer duration of delirium in the acute phase and increased severity of illness, and those affected by multiple complications constitute the patient population at highest risk of developing brain dysfunction [41,42]. Patients with advanced age often have preexisting neurocognitive issues, altered pharmacodynamics, and coexisting diseases, which increase their susceptibility to CCI. The exact mechanism behind the development of these neurocognitive deficits is currently unknown, but it is believed that hypotension, hypoxia, metabolic derangements, and iatrogenic causes such as sedatives may play a role.

In addition, CCI patients incur extreme mental and emotional stress. The presence of tracheostomy (or endotracheal) tubes makes communication difficult. Not surprisingly, more than 40 percent of these patients suffer from anxiety, posttraumatic stress disorder

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(PTSD), and depressive disorders [43–47]. The inability to perform activities of daily living (ADLs), difficulty in communication, the need for mechanical ventilation, and poorly treated pain are among the many factors that contribute to psychiatric issues in this patient population.

Endocrine Changes

Patient with CCI experience endocrinopathies that are often a continuation or sequela of the initial acute critical illness. There is a marked decrease in the pulsatile secretion of anterior pituitary hormones [48–50]. This pulsatile loss of growth hormone correlates with decreased anabolism and promotes catabolic metabolism. Similarly, decreased pulsatile thyroid-stimulating hormone (TSH) secretion leads to low plasma T_3 and T_4 levels [1]. The peripheral conversion of T_4 to T_3 is also decreased [51]. Low T_3 plasma levels correlate with muscle weakness and bone loss. Although the adrenocorticotropic hormone (ACTH) levels are low, cortisol levels remain high. High cortisol levels are probably secondary to decreased cortisol clearance in the chronic inflammatory milieu [52]. High cortisol levels contribute to muscle wasting, hyperglycemia with insulin resistance, decreased wound healing, and increased susceptibility to secondary infection. They also contribute to fluid retention and anasarca, which is commonly seen in patients with CCI. Increased peripheral insulin resistance seen in patients with CCI often leads to prolonged hyperglycemia, which has been shown to correlate with mortality [53]. Male CCI patients have been shown to have extremely low levels of testosterone and have high levels of estrogen, indicating increased aromatization of androgens [54,55]. These abnormalities in the gonadal axis may play a role in promoting a catabolic state in CCI patients because testosterone is the most potent endogenous anabolic steroid [56].

Malnutrition

The stress from critical illness, chronic inflammation and the catabolic state increases the nutritional requirements of CCI patients. Unfortunately, these patients often have poor mentation along with swallowing dysfunction. Consequently, they are unable to meet their dietary requirements, which can lead to profound nutritional deficiencies. Geriatric patients with poor preexisting nutritional state, malabsorption, and poor nutritional reserve are prone to malnutrition after critical illness.

Due to the catabolic state, there also is a shift toward proteolysis and gluconeogenesis. The body preferentially uses muscle proteins as an energy substrate, which leads to substantial muscle breakdown. This has profound effects on respiratory muscle strength, ventilatory capacity, and maximal inspiratory effort, which further complicate weaning from the ventilator in patients with CCI [57,58]. In addition, malnutrition has also been shown to blunt the ventilatory drive [59].

Synthetic function of the liver is impaired. as reflected by hypoalbuminemia, leading to low intravascular oncotic pressure and the development of anasarca [60,61]. Patients with CCI often have deficiency in both micro- and macronutrients. Patients often have vitamin D deficiency, leading to bone resorption [62,63]. Similarly, lack of micronutrients such as carnitine has been linked to mitochondrial dysfunction and multiorgan failure during CCI [64]. Elderly patients with CCI often also have electrolyte imbalances, leading to hypernatremia, hypophosphatemia, and hypomagnesemia, which can further impair mentation and respiratory function [65]. Malnutrition also predisposes patients to have abnormal

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hematopoiesis and immune dysfunction, leading to chronic anemia and increased propensity to have nosocomial infections [66].

Neuromuscular Alterations

A majority of critically ill patients suffer from neuromuscular weakness [67]. Broadly, these disorders are classified into critical illness polyneuropathy (CIP), critical illness myopathy (CIM), combined CIM/CIP, and prolonged neuromuscular blockade. This adversely affects the respiratory muscles, which complicates ventilator weaning, the muscles involved in deglutition, leading to swallowing difficulties and increased risk of aspiration, and the muscles of the extremities, which impairs mobility [68].

Critical illness polyneuropathy is characterized by symmetrical involvement usually of the limbs, especially the lower extremities, weakness of proximal neuromuscular regions (shoulder and hip girdle), and involvement of the respiratory muscles [69]. It usually spares the oculofacial muscles and cranial nerves [70]. Although patients with CIP may exhibit distal sensory loss, it may be difficult to elicit because a significant number of patients with CCI have altered mental status.

Critical illness polyneuropathy is a distal axonal sensorimotor polyneuropathy believed to be secondary to disruption of the blood-brain barrier and neuronal injury by inflammatory mediators [1,71,72]. Electrophysiology studies are generally consistent with a generalized axonal sensorimotor polyneuropathy with low motor and sensory amplitudes [73].

Chronic critical illness patients with CIM have flaccid quadriparesis (proximal greater than distal muscles), difficulty weaning from mechanical ventilation, and often facial muscle weakness [74,75]. Unlike CIP, CIM may be associated with a rise in serum creatine kinase (CK) levels. Muscle histopathologic findings are of myopathy with myosin loss. Electrophysiology studies reveal normal to low motor amplitudes with occasional prolongation of compound muscle action potential [76,77]. Sensory responses are usually preserved.

Critical illness myopathy and CIP can coexist in patients with CCI [78,79]. Patients with CIM have quicker resolution of weakness compared with those with CIP or combined CIM/CIP [80]. Prolonged neuromuscular blockade is a rare form of weakness seen in patients who underwent prolonged neuromuscular junction blockade and had compromised liver or renal function [81]. Train of Four monitoring usually is diagnostic of this form of weakness, although formal testing sometimes may be necessary.

Management of Chronic Critical Illness

Management of CCI patients is an emerging challenge for today's healthcare systems. Despite the significant burden on the healthcare system afforded by CCI, there is a lack of guideline-based recommendations regarding management of this patient cohort.

One of the major challenges remains early identification of patients who meet the definition of CCI. There can also be various venues where these patients receive care, including ICUs, step-down units, weaning units, and floors in acute care hospitals, as well as specialized centers such as long-term acute care hospitals (LTACHs). Variation in care is affected not only by the venue but also by the staffing ratios. The composition of a care team ideally should be multidisciplinary, including physicians, nurses, respiratory therapists, physical therapists, and speech and language specialists, as well as nutritionists who

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continue to deliver critical care in a manner similar to most ICUs. The goal is to create a comprehensive care plan for the patient with the goal of targeting a return to a functional status, as close to before the illness as possible. In many ways, this might be more of a challenge than in the acute care setting, given not only the resource-intensive patient needs but also a background of multiple chronic illnesses, more limited resources, and continued proclivity for clinical decompensation. In fact, the outcomes for these patients remain grim, with a high 1-year mortality rate of 50 to 77 percent [82,83], significant debilitation at discharge [84], multiple transitions in care following incident hospitalization [8], and increased caregiver fatigue and stress [82].

Tracheostomy and Mechanical Ventilation (MV)

Liberation from mechanical ventilation becomes one of the cornerstones of management. The timing of tracheostomy placement in the acute care setting is becoming shorter, with an average recommendation of about 10 days of mechanical ventilation [85]. Patients with tracheostomies often get admitted to chronic care facilities for weaning from ventilator support. Ventilator dependence is an independent cause of diaphragmatic muscle fiber atrophy [86], and mechanical ventilator for as few as 6 days is shown to cause a 30 percent decline in pressure differentials created by diaphragmatic contraction [87], which predisposes to prolonged mechanical ventilation. As in the acute care setting, adherence to a protocol-driven approach for weaning has been shown to decrease days on mechanical ventilation [88]. Despite the absence of evidence specific to chronic care facilities, formulating a daily multidisciplinary plan of care with adherence to objective data points and constant team and family communication can shorten the duration of mechanical ventilation. In this regard, implementing the ABCDEF bundle (i.e., *Assess*, prevent, and manage pain; *Both* spontaneous awakening trials and spontaneous breathing trials; *Choice* of sedation and analgesia; assess, prevent, and manage *Delirium; Early* mobility and exercise; and *Family* engagement and empowerment [89,90]) is likely to improve time to liberation from mechanical ventilation in this patient cohort. Weaning protocols can be effectively managed by respiratory therapists, and in coordination with bedside nursing, physical therapy, and a speech and language specialist, patients can make a robust clinical improvement. Various methodologies, e.g., use of the Rapid Shallow Breathing Index (RSBI) [91] and use of 50 percent lower ventilator support or T-piece trials for spontaneous breathing trials for as long as tolerated by the patient (often >120 minutes) used in the acute setting are routinely part of the process of weaning patients [5]. Patients who prove to be difficult to wean off ventilator support often require a more thorough assessment of barriers to liberation from the ventilator. Integration of bedside ultrasound in the assessment of diaphragmatic contractility in difficult-to-wean patients can be a useful tool to identify and follow such patients [92]. Once weaned from mechanical ventilation, a standardized approach to tracheostomy decannulation is pursued. In chronic care facilities, management of dysphonia as well as dysphagia after tracheostomy requires engagement of a dedicated speech and language specialist to assist patients in regaining normal function.

Analgesia, Sedation, and Delirium

Assessment of pain in geriatric patients with CCI can be quite challenging. While obvious in patients who have undergone surgical procedures (e.g., trauma, neurologic, or surgical ICU patients), pain is probably significantly underestimated in other patients. Overreliance on

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clinical markers of pain (hypertension, tachycardia, sweating, frowning), inability of patients to communicate (due to mechanical ventilation, altered consciousness, and preexisting conditions such as visual and hearing impairment), and lack of validated nonverbal pain assessment tools in patients in the CCI population further complicate management. Routine clinical activities including turns, repositioning, and catheter placement/exchange/removal are all recurrent stimuli of pain. In addition, most patients in the acute care setting likely will be exposed to analgesics (continuous or intermittent infusions during ventilation), and it is important for clinicians to remember to address this in formulating care plans for CCI patients. It is recommended that these patients be treated with a scheduled opiate taper of at least a week's duration if they have been exposed to opiate infusions in their incident hospitalization [90]. While continuous opiate infusions should not be routinely used in most CCI facilities [90], multimodality enteral opiate and nonopiate pain management strategies should be used to manage pain in CCI patients. The goal of analgesia should be such that patients can participate in mobilization and physical therapy while minimizing the side effects of analgesics, including nausea, constipation, sedation, respiratory depression, and the potential for dependence.

Use of sedatives such as propofol, benzodiazepines, and dexmedetomidine is a frequent part of ICU care, especially in mechanically ventilated patients. Despite quality evidence favoring light sedation [93], practice patterns can vary significantly between ICUs. It is therefore not uncommon to see CCI patients who have received high-dose sedatives during their incident hospitalization. Standard recommendations include strict adherence to daily spontaneous awakening trials and use of sedatives only if indicated, starting at half the original dose. Nonbenzodiazepine sedatives are preferred over benzodiazepines [94].

Similar to the acute care setting, delirium in CCI patients has been associated with significantly worse outcomes. It is especially prevalent in geriatric CCI patients given their advanced age, comorbidities, and baseline use of psychotherapeutics. The majority of delirium is hypoactive and goes largely unrecognized, necessitating the use of validated tools such as CAM-ICU [95] in the CCI population. Again, clinicians need to be mindful of the fact that sedatives used in acute care settings, especially benzodiazepines, can increase the risk of delirium significantly. A conservative strategy could include tapering doses of benzodiazepines (if they have been used in acute care setting) in patients with CCI. Significant attention should be paid to delirium prevention, including maintenance of day-night cycle, use of appropriate light cues to promote wakefulness during the day, minimizing noise and clinical interruptions at night to promote sleep, use of restorative visual and hearing aids as soon as the patient is able, and involvement of family visitation and interaction to promote the well-being of patients. Routine use of pharmacologic interventions for the management of delirium is not recommended, and such use should be limited to severe manifestations (e.g., hallucinations, psychosis). Use of daily diaries (written by patients or family members) documenting the patient's stay in a LTACH setting has been shown to help patients with PTSD.

Nutrition

Nutritional assessment and management form the cornerstone for treating geriatric patients with CCI. A specialized approach involving daily assessments by clinicians, nursing staff, and dietitians is of paramount importance in this setting.

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Nutritional Assessment. All CCI patients should undergo periodic nutritional assessment, including one at the time of admission to LTACHs. Commonly used nutritional assessment measures involve anthropometric measures (preadmission dry adjusted weight), comprehensive physical examination (temporal wasting, sarcopenia), evaluation of hypoalbuminemic state with fluid status (ascites, pleural effusion, sacral, scrotal, and pedal edema), daily calorie counts, and laboratory indices such as prealbumin, transferrin, and retinol-binding protein levels [96]. In addition, these measures can be used to determine response to nutritional interventions in CCI patients. Although various screening tools such as the Nutritional Risk Index (NRI), subjective global assessment (SGA), and Mini Nutritional Assessment have been described to assess nutritional risk [97–99], currently no clinically validated tool exists for patients with CCI. Determination of nutritional status thus relies on the multidisciplinary team taking care of the CCI patient [61].

Nutritional Goals. The key strategy of nutritional supplementations in patients with CCI is to replenish the nitrogen deficit by ensuring adequate protein intake, preventing underfeeding/overfeeding, and minimizing nutritional interruptions. Both overfeeding and underfeeding are associated with poor outcomes and increased mortality [100–102]. However, determination of adequate energy requirements in geriatric CCI patients is clinically challenging. Given the difficulties associated with indirect calorimetry, the lack of consensus with regard to the use of predictive equations, and the variability of pathophysiologic states in CCI patients, a target of 20 to 25 kcal/kg adjusted dry weight per day is often recommended by experts [61]. It is important to recognize that this "one size fits all" strategy may not hold true for all CCI patients, especially the elderly, and does not replace the requirement of periodic nutritional assessment in these patients. Similarly, a daily protein intake of 1.5 g/kg is recommended [103]. Patients with impaired wound healing, decubitus ulcers, high ostomy outputs, and undergoing renal replacement therapy usually have higher protein requirements. Overfeeding protein can result in hyperammonemia and azotemia, leading to encephalopathy, hypertonic dehydration, and hypernatremia [96]. Periodic measurement of serum blood urea nitrogen (BUN) and sodium thus is recommended to avoid such "protein overfeeding" scenarios.

Chronic critical illness patients often develop *refeeding syndrome* after reintroduction of carbohydrate-based diets. The key features of this syndrome include acute hypophosphatemia, reduction of thiamine and electrolytes such as magnesium and potassium, acute volume expansion, impaired oxygen delivery, and myocardial injury [51,104]. Hypophosphatemia may affect diaphragmatic muscle function and further impair attempts at weaning from mechanical ventilation. Interruptions in diet thus should be minimized, and a high clinical suspicion of refeeding syndrome should be maintained on resumption of feeding. If patients do develop symptoms indicative of refeeding syndrome, feeds should be restricted to about 1,000 kcal/day with slow increments over time and close electrolyte monitoring.

Chronically ventilated patients often require supplemental feeding because tracheotomy affects the muscles of deglutition. Enteral feeding is often preferred over parenteral feeding due to its lower costs and lower invasiveness [105–108]. Enteral feeding has the advantages of preserving gastrointestinal integrity, reducing bacterial translocation, and modulating immunologic and catabolic responses [61,109,110]. However, enteral feeding is commonly associated with interruptions in feeding, especially for procedures, leading to underfeeding. Semi-elemental feeds are preferred over whole-protein formulations. Choice of appropriate enteral formulations should be based on the patient's underlying pathophysiology, sodium

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status, renal status, and tolerance to a particular formulation [61]. Routine use of "pulmonary formulations" may lead to delayed gastric emptying [111].

Parenteral nutrition is often reserved for patients who are unable to meet their caloric requirements with enteral nutrition alone. Parenteral nutrition is associated with a higher risk of infectious complications than enteral nutrition [108]. Special care should be undertaken to ensure sterility of the central line site, and electrolytes should be monitored closely for patients undergoing parenteral nutrition.

In addition to macronutrients, judicious replenishment of micronutrients is of paramount importance, especially in the geriatric CCI patient population. Vitamin D and pamidronate help with decreasing bone resorption, calcitriol promotes calcium uptake by gastrointestinal tract, vitamin C and zinc sulfate promote wound healing, and carnitine helps in fatty acid oxidation. In addition, pharmacologic supplementation with megesterol, methylphenidate, or mirtazapine may help in stimulating appetite in CCI patients. Thyroid supplementation is often required in these patients based on thyroid function tests. Judicious glycemic control with insulin supplementation forms an integral part of patient management. In addition to intensive insulin management, serial blood sugar checks should be performed to avoid hypoglycemia.

Additional Management Strategies

Early mobilization along with muscle training has been shown to be beneficial in intubated patients [112]. Early physical therapy and mobilization help in decreasing the incidence of pressure ulcers, limb contractures, and deep venous thrombosis in CCI patients. Similarly, whole-body rehabilitation including limb strengthening exercises, trunk control, body posture maintaining exercises, and subsequently ambulation with a wheeled walking aid has been shown to be effective in successful ventilator weaning in patients receiving prolonged mechanical ventilation [113–115]. In addition, such measures enable CCI patients to recover from critical illness myopathies and regain muscle strength and enable them to perform the activities of daily living (ADLs) in the long run. This is especially important for geriatric patients in whom the performance of ADLs is an important milestone toward independent function. Thus early involvement of physical and occupational therapy forms an integral part of CCI management.

Pressure ulcer prevention and aggressive treatment are pivotal in the management of CCI patients because pressure ulcers may progress to osteomyelitis and further complicate the clinical course if left untreated. Pressure ulcer prevention requires daily assessment by the clinical team, timely postural changes, special pressure-reducing mattresses, and barrier ointment application [116,117].

Patients with CCI often have indwelling catheters and intravenous lines. Similar to acute critical care management, daily assessments of the position, functioning, and utility of these lines should be made. Every attempt at early removal of these catheters should be made to prevent further line-related complications.

Communication with Patient and Family Members

Because patients are often unable to participate in decision making regarding continuity of care, families frequently get involved as surrogate decision makers. This can be an emotionally difficult time for most. Depending on the age, quality of life prior to critical illness, and burden of chronic health conditions, the decision to continue intensive care or focus on

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comfort, often comes to the forefront. In the case where a patients' wishes are clearly known, the burden of this decision is somewhat eased for surrogates. However, there is still a significant proportion of the elderly, that might not have communicated their wishes to their family. The nature of modern day nuclear families can further increase the stress of providing care to a chronic critical ill patient, not just from an emotional, but also a financial and caregiver stress perspective. The transformation of a patient from an independent functional status to being ventilator dependent often with concomitant weakness, delirium and skin breakdown can be a traumatic experience for patients and family alike. Not surprisingly, posttraumatic stress disorder is also common amongst caregivers of patients with CCI [118]. In addition, the experiences of individual patients and families are colored by their cultural and religious backgrounds, belief systems, and health literacy. An inclusive and respectful approach is therefore needed to address the needs of this population.

Communication with CCI patients (when cognizant) and their families is of utmost importance in ongoing care. This can occur in varied settings between patient surrogates and the healthcare team, e.g., daily communication at the bedside or a more formalized conference approach. In many cases, clinicians often find themselves in a unique role of providing information, predicting the course of a patient's clinical trajectory, providing emotional support to the family, and eliciting the goals of care from the surrogate decision makers for a patient. This often requires a multidisciplinary approach from the clinical team (including ICU physicians, palliative care physicians, nursing staff, chaplaincy, social work, etc.) and can be even more challenging when dealing with end-of-life decision making in CCI patients [119]. It is also important to anticipate conflicts in decision making and have a consistent approach to deal with such situations as and when they arise. Palliative care should be an integral part of the care plan for CCI patients [3]. An integrated approach by ICU and palliative care teams with repeated contacts to guide patients and families through simple as well as more complex decisions is likely to have better outcomes than a "same size fits all" structured approach [120].

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