

Moving Our Critically Ill Patients: Mobility Barriers and Benefits

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In the last quarter-century, research developments have led to improvements in diagnosis and resuscitation of critically ill patients. With these improvements, survival for many populations of critically ill patients has increased [1–6]. With these improvements in mortality, the field of critical care has seen increased opportunities to affect the immediate posthospital quality of life for survivors of critical illness. The concept of a successful outcome for an ICU patient has gone through meaningful maturation in the last decade. Not too long ago, for clinical trials in critical care, being alive at 28 days or alive and off the ventilator were the sole endpoints. Examination of ICU processes of care has led to longer periods of observation to determine survival endpoints. Recently, critical care experts also have focused on management factors that may affect survivors' quality of life. Many investigators have become interested in aspects of ICU practice that may affect the speed with which a person may return to premorbid function [7,8]. Authors' descriptions of ICU sequelae long after the 28-day mark have demonstrated mental and physical limitations [7,8]. This issue of *Critical Care Clinics* will explore care of the critically ill patient from the viewpoint of how ICU mobilization may benefit patients and how usual practice may inhibit the delivery of mobilization.

Mobility as a therapy

Some may view mobility in the context of the resources required to turn an ICU patient in bed for a bath, or pulling the patient back up to the head of the bed, or mobilizing a patient from the bed to a stretcher for transport.

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Some hospitals have gone as far as instituting a lift team to help bedside caregivers deal with these tasks. It is the intent of the articles in this issue of *Critical Care Clinics* that the term ICU mobility be equated with exercise and with therapy. In the ICU context, mobility can be viewed as a therapy just as exercise prescriptions are written for chronic obstructive pulmonary disease (COPD) and myocardial infarction (MI) patients in the cardiopulmonary rehabilitation setting. Like other therapies, ICU mobility and exercise can be thought of in terms of quantity of dose, duration of dose, and frequency of administration. Importantly, as has been seen with other ICU practice and therapies, mobility could be approached with protocolization. Unlike ICU pharmaceutical therapies, there are virtually no ICU data guiding current ICU mobility therapy decisions.

Despite a general notion by many groups that mobility and exercise may play a strong role in speeding the return to preacute illness functional status, the exact manner in which to administer mobility and the safety of administering mobility maneuvers to critically ill patients are not clear. Overall, mobility intervention research for ICU patients has not received much attention.

Impact of inactivity for critically ill patients

The deconditioning effects of acute illness are extrapolated from studies on acute inactivity, with a great deal of the information stemming from studies of young healthy persons put on bed rest in space programs and low-gravity research [9]. Recorded alterations include changes in mood, coordination, muscle strength, balance, and work tolerance [10]. Immobilization of laboratory animals long has been a model of deconditioning investigation. Both of these types of investigations have provided many insights into the muscle, nerve, bone, and cardiovascular system changes seen in people during an illness that directly (eg, leg cast) or indirectly (eg, ICU sedation for mechanical ventilation) requires immobility.

Deconditioning may be described as the multiple changes in organ system physiology that are induced by inactivity and reversed by activity [10]. In the clinical setting, acute deconditioning refers to changes that occur within days to a few weeks of a sudden decrease in activity. Chronic deconditioning refers to changes occurring over months and years. Geriatric exercise studies have provided some insight into the chronic deconditioning effects of sedentary lifestyles; there are few geriatric or other deconditioning studies that specifically address the acute effects of an ICU stay. There have been some inroads however, and investigators and ICU caregivers have become more aware that neuromuscular abnormalities develop as a consequence of critical illness. These neuromuscular abnormalities can be found in many patients who stay for as briefly as 1 week in the ICU [11]. Electromyography and histological studies in critically ill patients have revealed that acute myopathy may coexist with acute neuropathies [11], and both have been observed frequently (eg, in patients with sepsis) [12,13].

Muscle function in critically ill patients

Investigators estimate that for a young, healthy adult, 45% of the human body weight is muscle [14]. For many caregivers who work in the ICU setting, there is awareness of the overall weakness patients demonstrate even after a few days of mechanical ventilation. Theories have been developed, from a wide range of clinical investigation, to explain the possible causes of ICU-related muscle weakness. Few data, however, are directly from ICU patient study. For ICU patients, the standard functional muscle tests such as maximal voluntary isometric contraction or muscle function estimated by the 0 to 5 scale may not be applicable when the ICU patients are unconscious [12]. Much of the muscle weakness experienced in ICU patients, therefore, may go without notice or documentation, particularly early in a patient's ICU course. Reviews of the effect of inactivity on muscle strength have noted that skeletal muscle strength may decline by 1% to 1.5% per day of strict bed rest [10,11,14]. Data from patient studies with limbs immobilized by casting and animal studies with castings point out that the decline in strength may be even more significant, with predictions as high as 5% to 6% per day [15–19].

Atrophy, loss of maximum force generation, change in type or density of muscle fibers, change in density of mitochondria, lessons from other myopathies, and the syndrome now known as critical illness ICU polymyoneuropathy are important concepts from the literature. Examination of these types of data may help caregivers understand more fully how these concepts may relate to the impact of inflammation and immobility on ICU patients' muscle strength.

An approach to muscle fiber classification subdivides fibers into type I (slow twitch) and type II (fast twitch, glycolytic). The type I fibers are labeled as red, because they contain myoglobin and mitochondria. Type I fiber metabolism is aerobic, and the fibers function in relatively sustained contractions. Type II fibers lack myoglobin, contain few mitochondria, and are termed white. They are thought to derive energy from anaerobic glycolysis. Type II fibers work in short durations of contraction. The distribution of fiber type varies among the muscles of the body, depending on the muscle function (eg, whether or not a muscle is involved in sustained contraction, such as maintaining posture). The proportion also varies with training. Sprinters will increase the number of type II fibers, whereas marathon runners will increase the number of type I fibers in certain leg muscles.

Muscle atrophy is a complex, active remodeling process. Many studies have shown that without physical activity, skeletal muscles begin to lose protein. With atrophy from inactivity comes change in selective fiber type (in some studies, strength of type I or slow-twitch fibers, appears less affected than type II or fast-twitch fibers). There can be reduction in the cross-sectional area of the individual muscle fibers, changes in the satellite cells, and an alteration in the distribution and size of the capillaries and

connective tissue [20]. The antigravity muscle groups are located in the legs, trunk, and neck. They function primarily to support the body and are particularly rested when a patient assumes a horizontal position in bed. Antigravity muscles have been observed to lose contractile proteins with a corresponding increase in noncontractile tissue content, including collagen, while the total number of muscle fibers remains unchanged during an episode of immobilization. Type I fibers of the antigravity muscles lose myofibrils, resulting in a decreased cross-sectional area, in response to reduced physical activity [14,21–23].

There are separate phenotypes of muscle atrophy. Much data are available on primary atrophy, which is related to disuse (eg, bed rest, space flight, denervation, limb casting). More relevant to ICU patients may be secondary atrophy, or those muscle changes related to a pathology. Biochemical studies have begun to elucidate the very complex pathways involved. It is not known to what extent various gene programming mechanisms are activated in primary and secondary muscle atrophy [20]. All forms of atrophy seem to have increased mRNA for polyubiquitin [20]. The polyubiquitin series of enzymes links the chains of the polypeptide cofactor ubiquitin onto proteins that enable subsequent degradation of the protein [24]. This linking of ubiquitin leads to the protein's recognition by the 26S proteasome, a large multi-subunit, multicatalytic protease complex that degrades ubiquitinated proteins to small peptides [25]. There are three main enzymatic components involved in the binding of ubiquitin onto muscle proteins targeted for degradation. E1, an Ub-activating enzyme, and E2s, Ub-carrier or conjugating proteins, initiate ubiquitin in the conjugation step. The principle enzyme, however, in this overall mechanism is E3 (Ub–protein ligase). E3 couples the activated ubiquitin to the protein substrate. There are over 1000 E3s in cells, which give a specificity to this system, because only a single E1 protein and a few dozen E2 proteins exist in cells [26]. In most types of muscle atrophy, overall rates of protein synthesis are suppressed, and rates of protein degradation are consistently elevated. This response accounts for most of the rapid loss of muscle protein. In fasting, protein breakdown in muscle provides the organism with a source of amino acids for gluconeogenesis. In various animal models of human diseases (eg, fasting, diabetes, cancer wasting, acidosis of chronic renal failure, sepsis, atrophy from inactivity, denervation, and glucocorticoid treatment), most of the accelerated proteolysis in muscle appears to be caused by an activation of the ubiquitin-dependent proteolytic system in muscles [27]. An important aspect to consider for future ICU mobility study is that for ICU patients, muscle changes may be caused by both inactivity and inflammation.

When muscles atrophy, information from laboratory preparation studies indicate that the strength of fast-twitch (type II) fibers appears to decline at an accelerated rate compared with declines in strength measured within slow-twitch (type I) fibers [14]. Fast-twitch (type II) fibers are involved with quick intense bursts of muscle contraction for strength. They quickly

demonstrate fatigue thought secondary to their primary use of glycolytic processes. Slow-twitch (type I) fibers use oxidative processes. Antigravity muscles contain slow-twitch fibers and involve less intense, prolonged, endurance contractions. Whether a result from type I or type II alteration, when whole muscle groups are considered, the antigravity muscles appear to selectively atrophy as a result of immobilization to a greater extent than non-antigravity muscles. With immobilization, the antigravity muscles of the calf and back appear to lose strength with bed rest at an accelerated rate as compared with muscles involved with grip strength [14]. With immobilization, as antigravity muscles lose contractile proteins, there is an increase in noncontractile tissue content, including collagen. Total numbers of muscle fibers seem to remain unchanged as a result of immobilization. For ICU patients on bed rest, it is suspected that type I fibers of the antigravity muscles lose myofilaments (cross-sectional area) [14]. This finding may be very relevant to ICU patients, because the muscle groups that may lose strength most quickly as a result of immobilization or bed rest are the groups involved with maintaining posture, transferring position, and ambulation [14]. It is believed that in the ICU setting, skeletal muscle, following such an atrophy mechanism, has the potential to regain contractile protein content and strength, because the number of muscle fibers is not affected by immobilization initially.

Mechanisms of muscle pathology in ICU patients

Critically ill patients may lose significant muscle mass by the time of hospital discharge. Herridge and colleagues [7] reported an 18% reduction in body weight by the time of discharge for patients who had acute respiratory distress syndrome (ARDS). Wagenmakers [28] comments that more than 1.5 kg of skeletal muscle mass per day and up to 50% of the total muscle mass in 2 weeks time may be experienced by ICU patients.

Wagenmakers has theorized that a functional denervation may be associated with critical illness. Certainly dysfunction occurs in situations of non-depolarizing neuromuscular blocking agents and corticosteroid administration, but also in patients with prolonged illness. Specific theories regarding the illness-associated reduction in frequency and intensity of nerve impulses arriving at the muscle membrane have been developed. Gene expression itself is suspected to be affected by the character of the nerve impulse that arrives at the motor endplate. The nerve impulse itself works as a message that may alter gene expression in specific fibers differently (type I or type II); therefore, the enzyme expression may be affected differentially within these specific fiber types. Nerve impulse characteristics are suspected of affecting mitochondrial content. Another alteration of skeletal muscle function following critical illness is a loss of oxidative capacity. Decreases in the activity of muscle mitochondrial enzymes in animal models and in critically ill ICU patients have been observed [29,30].

Wagenmakers suspects that the oxidative capacity decreases to very low levels within days after arrival at the ICU, and this implies that ICU patients may depend on glycolysis with lactate formation at very low muscle workloads (eg, work of ventilation). This also implies that skeletal muscles could fatigue rapidly during any type of sustained contractions. In their recovery phase from critical illness, patients are likely to experience the equivalent stress of intense exercise (high heart rates and ventilation rates) even at low exercise intensities encountered regularly in daily life. Many post-ICU patients complain of severe physical limitations for a prolonged time [7,31]. For this reason, they may have very low physical activities for months following hospital discharge. Both the rapid fall in mitochondrial content on admission to the ICU and these exercise limitations during recovery may prevent a rapid recovery of muscle function (strength) and oxidative capacity (endurance performance).

In a model of critical illness that administered intraperitoneal zymosan to rats, muscle force for zymosan-treated animals was only 10% of control animals. These changes were attributed to marked decreases in muscle mass associated with this acute inflammation model [32]. Eikermann and colleagues [12] studied evoked muscle fatigue in the hand muscles of sepsis patients with multiple organ failure and compared the results with normal volunteers immobilized with a hand cast. These studies were conducted to determine whether sepsis adds something more to muscle dysfunction than immobilization alone. This study was an attempt to tease out mechanistic differences between ICU-associated atrophy and atrophy seen in studies of inactivity or enforced bed rest. The authors noted that few if any data on maximum skeletal muscle force and fatigue in patients with sepsis had been reported previously. These compound variables of peripheral motor nerve and muscle function examine muscle weakness as a loss of power. The result is decreased motor function and fatigue of the conduction of the impulse as an exercise-induced decrease in the capacity to generate force or power output. The authors concluded that using this approach might yield more sensitive information for ICU patients than using nerve conduction velocity studies alone. In this study, maximum force and the compound action potential of the ulnar nerve were decreased markedly in sepsis patients. Interestingly, fatigue and ulnar nerve conduction velocity did not differ from volunteers, and a decrement of the compound muscle action potential was not observed with nerve stimulation frequencies up to 40 Hz. All patients with critical illness polyneuropathy (CIP), and an additional 50% of those without, had significant muscle weakness. The authors concluded that peripheral muscle force is decreased markedly in sepsis, without evidence for an increased fatigability of the impulse. They believed that the muscle weakness demonstrated was most likely caused by a sepsis-induced myopathy when the nerve conduction studies were normal. This study may point to differences in the more classic laboratory findings of normal volunteers becoming immobilized as compared with actual ICU

patient findings. Muscle findings in the ICU patients for the most part have been reported through isolated muscle biopsies, which, although important to document from a research prospective, carry at least some minimal risk to research subjects in the midst of their ICU stay. The authors emphasized that there was a difference in the type of defect noted between the normal immobilized volunteers and the sepsis patients in the muscle performance. They were able to determine this difference without requiring a muscle biopsy [12]. These data show that muscle weakness occurs even in the patients without electrophysiological signs of CIP. Thus, evoked action potential muscle force measurement may be more sensitive to detect relevant muscular abnormalities in critically ill patients [12].

Interestingly, insulin therapy in critically ill patients has been correlated with a decrease in neuromuscular dysfunction. Not only did tight control of hyperglycemia in critical illness with intensive insulin treatment improve outcome in a population of postoperative patients, it was shown to counter hyperglycemia by increased skeletal muscle glucose uptake (up-regulation of skeletal GLUT-4 and hexokinase II mRNA levels) rather than exerting an effect in control of hepatic insulin resistance and increased gluconeogenesis [33–35].

When many of these factors are considered specifically in the elderly ICU population, it may become more clear why elderly patients can take longer to recover than acuity matched controls [36]. The elderly ICU population may experience prolonged functional difficulties because of their relative decreased premorbid muscle mass compared with younger ICU patients and decreased exercise tolerance at baseline. The impact of immobility may be a more pronounced limitation in elderly patients. This may be because of elderly patients' baseline functional limits as compared with younger ICU patients.

Role of ICU-associated neuropathies

Patients with prolonged critical illness are at risk for developing compression neuropathies [37]. Two of the more common compression neuropathies for ICU patients involve the ulnar nerve at the elbow's retrocondylar groove and the peroneal nerve as it passes superficially over the fibular neck. Sensory deficits may be detected when there is involvement of the ulnar nerve, with deficits on the volar and dorsal surfaces of the fourth and fifth digits and weakness of the palmaris brevis, abductor digit minimi, and flexor digit minimi muscles, resulting in weakness of finger adduction and abduction. Involvement of the peroneal nerve is associated with weakness of ankle dorsiflexion and eversion. There may be resulting numbness of the lateral aspect of the leg and dorsum of foot. The motor and sensory deficits from these compression neuropathies may not be apparent for some time in ICU patients. Diagnostic delays in detecting ICU-related neuropathies are related to sedation and general level of consciousness of the ICU patient.

These ICU-associated neuropathies are typically self-limiting. Some patients, however, experience neurological deficits for months following their discharge from the hospital.

Effect of ICU inactivity on bone and cardiovascular function

Muscle tissue unfortunately is not alone in demonstrating alterations in ICU patients as a result of immobility. Like muscle, bone and cardiovascular systems respond to acute decreases in physical activity [14,16,38–41]. A concern for ICU-oriented mobility programs will be cardiovascular assessment and optimization of cardiovascular function. Certainly the data from outpatient study show that improvement of physical fitness decreases cardiovascular mortality risk by upwards of 51% [42]. In the ICU setting, reversible myocardial dysfunction can develop in critically ill patients, many of whom do not have primary heart disease. This syndrome is associated with systolic dysfunction, segmental contractility disturbances, and electrocardiographic changes [43]. Several authors have reported on the specific cardiovascular effects of deconditioning [38,39,44,45]. From immobilization studies, detrimental effects have been shown to occur on both the heart tissue and peripheral cardiovascular system. Orthostatic intolerance is believed to be the result of a baroreceptor dysfunction, and cardiovascular deconditioning reduces orthostatic tolerance.

From space exploration studies, several key factors have become associated with deconditioning of the cardiovascular system. These findings would include: volume decreases in blood and interstitial fluid, decrease of arterial diastolic pressure, decrease in left ventricular stroke volume, left ventricular mass decreases, and carotid baroreceptors resetting. At re-entry into earth's gravity, astronauts may experience dizziness, tachycardia and palpitations, an inability to assume the standing position (orthostatic intolerance), presyncopal feelings caused by postural stress, and reduced exercise capacity [46]. What remains to be clarified is how applicable these findings are to the heterogeneous ICU populations, and how well the counter-efforts applied to otherwise healthy astronauts to inhibit these changes would work for ICU patients.

Are there data regarding either the positive or negative effects of bed rest?

In a piece published in the early 1940's, Dock [47] noted that in the 19th century, bed rest was offered primarily as treatment of many disorders. For hospitals, there remains an association between beds and patients. Even today, hospitals are identified using the parameter of number of beds.

For many areas of health care, the therapeutic value of bed rest has been questioned [48]. In the postpartum period and for those patients with an uncomplicated MI, safe reductions in hospital stays have occurred in the last

few decades [49–55]. Patients within these clinical entities were able to get out of bed much sooner than what was appreciated even 30 years ago [56]. It would be reasonable to critically assess the need for bed rest early in an acute illness for ICU patients. Future study may allow for the establishment of guidelines to define the period in which some form of ICU exercise therapy yields positive results.

Brown and colleagues [57] reported on adverse outcomes associated with different levels of mobility during hospitalization of 498 patients aged 70 and older. The authors assessed these in-patients' mobility level, demonstrated on hospital wards, with a numeric value. Sixteen percent of the overall group were found to have low levels of mobility, and 32% were deemed intermediate in their activity. When the authors compared the low mobility group with the high mobility group, there was a graded association with adverse outcomes, such as a decline in activities for daily living (ADLs), a new need for institutionalization such as a nursing home, or death. Interestingly, bed rest was a specific order at some point during hospitalization in 33% of the patients. Routinely, mobility was limited involuntarily (bed rest orders), and almost 60% of bed rest episodes in the lowest mobility group had no documented medical indication [57].

Potential barriers to the delivery of mobility therapy in the ICU setting

Safety concerns

There are potential reasons why mobility therapy might be withheld by a caregiver early in a patient's ICU stay. ICU caregivers might be concerned that early mobility might risk an immediate adverse event. These events could include a dislodgement of a vascular access device or an endotracheal tube. Applying too aggressive of an ICU mobility program to patients on mechanical ventilation with altered mental status could be viewed by some as a potential for an orthopedic, plastic surgery, or even neurosurgical complication. Caregivers worry that early ICU mobility for some patient situations might cause a patient death from inadvertent removal of the endotracheal tube. There is also the fear that passive movement may cause further decrease in already marginal oxygenation or hemodynamic parameters. Control of pain and discomfort sometimes are viewed as competing themes with wakefulness and movement. There are few if any clinical studies that help caregivers define the risks associated with movement of critically ill patients. More difficult may be defining the risks involved in not moving critically ill patients.

When patients become more interactive, some caregivers may feel that active patient participation in movement will risk undo stress on the pulmonary system or cardiac system, causing irreversible hypoxemia, or a dysrhythmia leading to a preventable death. The potential risks of early ICU mobility are listed easily, whereas the benefits of ICU mobility therapy

are more illusive in their identification. At this point in time, the benefits of early ICU mobility are only implied by various literature in chronic disease states when exercise programs were administered.

Recently there have been reports addressing the safety of mobilizing patients with pulmonary emboli, including patients with submassive pulmonary emboli. The authors of a recent report believed that in light of their data, bed rest has no influence on the risk of developing pulmonary emboli among patients with acute deep vein thrombosis of the lower limbs already receiving appropriate therapy. Additionally, no outcome improvement could be shown by the influence of bed rest, even in patients presenting with acute submassive pulmonary emboli [58].

Multiplicity of vascular access

In ICUs, it is not uncommon for high-acuity patients to require more than one vascular access device. For patients with previous ICU stays that required vascular interventions, current prolonged ICU stay, or an ongoing coagulopathy, the ease with which new devices can be placed may be difficult. Therefore, indwelling devices for difficult-access patients come to be viewed as a precious commodity and are protected often at the cost of mobilization for fear of dislodgement. In patients requiring hemodialysis or continuous renal replacement therapies, mobility of the hip may be restricted because of the desire to maintain adequate flows through dialysis catheters placed in the femoral position. For these reasons, ICU-associated hardware in general may be conscious or unconscious causes of restrictions to the mobilization of ICU patients. There are few if any studies discussing the range of motion (ROM) tolerated for patients with vascular access devices in various anatomic sites.

Sedation

A goal for bedside caregivers is to maintain ICU patients in a calm and pain-free state. Achieving this goal and allowing clear mentation is often difficult, particularly early in their ICU course. In the last decade, ICU caregivers have been made aware of the deleterious adverse effects not only of prolonged neuromuscular blockade, but also of prolonged use of anxiolytic and analgesic therapies [11,59]. Many hospitals have addressed this aspect of ICU care with implementation of general sedation protocols that include some form of a daily awakening technique.

Cost barriers

Surprisingly, there are no studies that report efficacy of passive ROM in the ICU setting for the high-acuity patient. Therefore, justifying the cost of physical therapy positions targeted to the ICU is difficult in light of the current lack of safety or efficacy data. Therefore, although it may seem reasonable to request from hospital administrators the work force to

perform these tasks, when viewed from the administrators' point of view, there is little in the literature to justify these expenditures in labor. A discussion of barriers to ICU mobility would have to include the lack of data that aggressive passive ROM for the high-acuity ICU patient brings about either a quality improvement or an overall hospital cost reduction.

In contrast to work force requests, despite not having abundant data to support ICU specialty beds, there appears to be more widespread administrative acceptance of arguments for specialty beds than for the labor to move ICU patients. There may be reluctance on the part of hospital administrators to invest in human labor for the promotion of ICU mobility, because of lack of literature to support the value to patient outcomes.

In addition to work force costs, there may be reluctance to justify the cost of mobility aids. The price tag for specialty chairs, tilt tables, walkers, and portable ventilators are often difficult for hospital administrators to cover when there are few data to support the use of these devices, particularly any data to demonstrate improvement in morbidity or mortality.

Obesity

There is literature that both supports and refutes the argument that obesity carries an independent risk of death for ICU patients [60–62]. For mobility practice, an assessment of barriers necessarily would include the weight of ICU patients. In the United States, obesity has risen dramatically during the past several decades and is now among the nation's leading health problems. According to the National Health and Nutrition Examination Surveys (NHANES), a series of surveys conducted periodically by the US Department of Health and Human Services agencies, the number of overweight and obese Americans continues to rise. Between NHANES 1976 to 1980 and NHANES 1999 to 2002, the percentage of obese adults nearly doubled [63]. This means that more than 60 million adults in the United States are obese. Common sense may dictate that maintaining the human resources to adequately address ICU mobility concerns of morbidly obese patients represents more cost than those required for patients with a normal body mass index (BMI). For a population suffering an epidemic of obesity, however, ICU staffing concerns for obese ICU patients may not have kept pace uniformly in the United States. Very likely, ICU mobility concerns for the morbidly obese ICU patient in the future will become more of a challenge for hospitals to address adequately.

Time restraints

Time may be a factor in the reduction of attention to the mobility concerns of ICU patients. Certainly, there are perceptions from physicians, nurses, and physical therapists that the amount of daily hospital documentation has increased. For an ICU nurse particularly, the amount of time required to accomplish the necessary documentation may continue to rise.

In an 8- or 12-hour shift, there is only so much time a practitioner may spend on direct mobilization care of the patient and still achieve other care goals. Hospital administrators set and articulate each individual institution's priorities; hospital staff accomplishes patient goals according to these priorities. If insurance carriers, lawyers, and administrators continue to favor increasing volumes of documentation, staff likely will continue to prioritize care accordingly and accomplish the requested goals. In a recent study, it was determined that ICU patients were not turned every 2 hours despite staff's knowledge of the goal of every 2-hour turning [64]. One caveat about this report on turning of ICU patients is that the authors did not report what the other nursing tasks accomplished during the shift were [64]. Lack of turning does not directly translate to a lack of care for the patient, but that other goals possibly had exceeded mobility care in priority.

In a nursing survey addressing availability of time for direct patient care, staff nurse responses suggested a 6% decline in direct time spent on patient care in just a 3-year period from 1999 to 2001 [65]. Time devoted to charting and care documentation was given as the reason for most of this deficit.

Are there benefits to mobility?

In general, studies on inpatient medical rehabilitation are based on patients who survived until the post-ICU setting to qualify for the rehabilitation unit. There is some degree of a selection bias within inpatient rehabilitation studies, because most subjects are those whose performance allowed survival to a floor bed setting. Traditional rehabilitation programs, whether they are conducted in hospital, in a rehabilitation in-patient facility, or as an outpatient facility, do not typically commence while the patients are in an ICU setting. Therefore, the applicability of rehabilitation data to ICU patients may be limited.

In the ICU, besides relying on a common-sense foundation to the argument supporting the early administration of mobility therapy to ICU patients, there are numerous publications demonstrating an inpatient benefit to early exercise therapies. Munin and colleagues [66] started rehabilitation for patients aged greater than 70 following knee and hip replacement. Those starting on day 3 versus day 7 had shorter total lengths of stay, 12 days versus 15 days, and lower costs. Functional assessments reached preset endpoints sooner in the early rehabilitation group.

Mundy and colleagues [67] studied inpatients with community acquired pneumonia admitted to non-ICU beds. The patients randomized to early mobilization, (sitting out of bed or ambulating for 20 minutes starting on the first hospital day), demonstrated a shorter length of stay in the hospital (5.8 days versus 6.9 days). There were no adverse events reported for the early mobilization group.

For the ICU patient, there may be helpful correlations from the cardiac rehabilitation literature. Formal phase II cardiac rehabilitation and exercise

training programs have shown benefits in exercise capacity, lipid profiles, weight changes, autonomic function, subsequent hospitalization costs, and cardiovascular morbidity and mortality [68–71]. Interestingly, there are reports of over 50% reduction in the prevalence of depressive symptoms in cardiac patients undergoing active rehabilitation [68,69,72–74]. In a report that studied lower extremity clot development in post-MI patients, ambulating in the first 3 days post-MI compared with bed rest for the first 5 days, venous thrombosis was reduced by early ambulation [75].

One study examined whether muscle wasting in critically ill patients could be prevented with stretching alone [76]. Continuous passive motion for three 3-hour sessions over 7 days was applied to one leg of five separate critically ill adults. Protein loss and water gain were less in the treated leg compared with the control leg that received routine nursing care [76].

Siebens and colleagues [10] reported their findings with an inpatient population of older adults with nondisabling medical and surgical diagnoses. The intervention group received an inpatient general exercise program with encouragement to continue the program at home, self-administered. Although the intervention group did not show a decrease in length of stay, the authors demonstrated better function in instrumental ADLs at 1 month in those receiving the inpatient exercise program [10].

Mobility delivery options

What is passive range of motion?

When searching through various textbooks, the reader will find fairly consistent, although frequently superficial, definitions of passive ROM. Few definitions offer mechanistic explanations for the proposed goals of passive ROM, that being preservation of the range of the joint. Most definitions reflect that performing passive ROM exercises consists of repeated movement of a joint within the available limits (range) of the joint. There are no studies that set out to specifically determine the benefits of passive ROM exercise in ICU patients [9]. Technically, passive ROM differs from what is described as a prolonged muscle stretch. A prolonged muscle stretch usually implies holding a muscle or group of muscles in a lengthened position for a period. The notion of splinting a joint follows from this notion (ie, that passive muscle stretch leads to maintenance of a joint's baseline range) [77].

The musculoskeletal system has been described as having three characteristics: muscular strength, endurance, and flexibility. Muscular strength (a dynamic phenomenon) is the maximum force a muscle or muscle group generates at a given velocity. Muscular endurance is the ability of a muscle or muscle group to perform repeated contractions against a load for a prolonged time [78]. Flexibility can be thought of as the ROM about a joint, and research suggests that flexibility decreases with age [78].

What does passive range of motion offer ICU patients?

Synovial fluid movement within the joint space depends upon the active or the passive motion of joints. Much of the synovial structure depends on synovial fluid for adequate nutrition and maintenance. Immobility of a joint is thought to lead to synovial fluid stasis. Without movement, there is increased intra-articular fluid volume and increased pressure; this can cause heightened tension, pain, and decreased ROM of a joint. Simple joint motion creates fluctuations in intra-articular pressure, which decreases fluid stasis. In general, flexion of a joint increases intra-articular pressure, and extension decreases it. Active motion produces more profound intra-articular pressure changes when compared with passive motion [79].

Since the early 1970s, positive effects of continuous passive motion (CPM) devices have been demonstrated in the recovery of a large array of common joint pathologies [80–82]. Passive motion studies of the knee using radiolabeled tracers demonstrated that the clearance rates of synovial fluid can be increased under conditions of passive motion [83]. Moreover, animal models of hemarthrosis and septic arthritis demonstrated improved healing and recovery under conditions of continuous passive motion manipulation [81,82]. Applying passive range of motion techniques may mimic some characteristics of CPM devices. But for the ICU patient with systemic illness, the benefit of these CPM devices is unclear [84].

Electrical stimulation as a means to achieve muscle contraction

It has been shown that pulmonary rehabilitation is able to improve exercise tolerance in patients with COPD [85,86]. The possibility exists that for ICU patients, as has been shown for COPD outpatients, acute skeletal muscle dysfunction is a potentially remediable effect of immobilization and a reason for exercise intolerance [87]. Electrical stimulation of lower extremity muscles was performed in a study of bed-bound patients on mechanical ventilation who had severe end-stage COPD. These chronically mechanically ventilated COPD patients were chosen because of their likelihood to display extreme dysfunction of their skeletal muscles. These patients already had experienced a considerable period of immobility before enrollment in this particular study. Electrical stimulation was applied only to the lower limbs twice a day for 30 minutes. Surface electrodes were positioned bilaterally on the quadriceps femoris and on the vastus glutei. The muscle strength score was similar in the two groups at admission. Muscle strength improved significantly in the patients who received electrical stimulation and decreased the number of days needed to transfer from bed to chair (11 days versus 14 days) [88].

Tilt table therapy

An interesting report of a survey response from Australian physiotherapists demonstrated that the most common reasons for inclusion of

tilt table treatment in ICU patients were to: facilitate weight bearing, prevent muscle contractures, improve lower limb strength, and improve patient alertness [89]. Standing with assistance of the tilt table seemed to be a technique used by the most of the responding physiotherapists working in Australian ICUs. In their report, the authors site that standing with assistance of the tilt table is a method to reintroduce patients to the vertical position when they are unable to stand by themselves. Despite the lack of clinical trials, tilting was included as a treatment modality in the recent statement by British physiotherapists working in critical care [89,90].

Specialty beds

There is research demonstrating that the use of kinetic therapy beds decreases the incidence of pulmonary complications such as nosocomial pneumonias in critically ill patients. Much of the information available, however, has been associated with the manufacturers of the specialty beds. The overall utility of these devices is unclear given the considerable expense of these beds and the lack of definitive cost-benefit analyses.

Active range of motion

For ICUs in developed countries, it is not easy to point to unifying guidelines regarding the initiation, frequency, and intensity of either passive or active ROM. A survey of European hospitals demonstrated a great range of responses in regards to staffing and training for ICU-dedicated physiotherapy [91]. In a Canadian survey, few of the responding physiotherapists indicated that ICU mobilization of the intubated, mechanically ventilated patient was common place [92]. Minimizing anxiolytic and anesthetic medication by balancing ICU patient comfort against anxiety and pain is a difficult goal to achieve. It is particularly difficult early in the course of an ICU patient's acute organ dysfunction. An important ICU patient recovery milestone is reaching the level of mentation that permits interaction with staff. It is at this point that the ICU patient may carry out active participation in ROM exercises. Structured protocols for early ICU application of exercise have begun to be reported just recently. Bailey and colleagues [93], have described their experience with dedicated physical therapy to a respiratory ICU. Formal consensus conference statements are needed that include all aspects of critical care providers, particularly physical therapists. These consensus conference statements hopefully will help develop guidelines that will draw attention to safety, heighten efficiency, and promote regularity in the delivery of mobility to ICU patients. Working through these guidelines will draw attention to areas in need of formal research. Hopefully, through future study, the efficacy of these mobility practices may be determined.

Summary

Mobility has been recognized as a component of primary, secondary, and tertiary prevention of overall disease morbidity and mortality [10]. ICU data are limited as to the role mobility exercise might play in treating the effects of acute illness and acute deconditioning. An increased attention to data generation regarding ICU mobility programs would be necessary to support the notion that ICU deconditioning may be potentially reversible or preventable.

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