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Management of Refractory Mechanical Ventilation Weaning in a Geriatric Neurocritical Patient with ARDS and COPD Using the ISCCM 2023 Guidelines: A Case Report

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A B S T R A C T

Mechanical ventilator weaning failure occurs in 20–30% of critically ill patients, with the risk significantly amplified by chronic obstructive pulmonary disease (COPD), acute neurological impairment, and severe hypoxemia. A 72-year-old female with COPD developed acute respiratory distress syndrome (ARDS) secondary to hospital-acquired pneumonia (HAP) following a re-craniotomy for an epidural hematoma. Following an initial extubation failure marked by hypercapnia ($PaCO_2$ 50.7 mmHg), the patient required reintubation. Her initial PaO_2/FiO_2 ratio of 127 mmHg indicated severe gas exchange impairment. Management utilized the 2023 Indian Society of Critical Care Medicine (ISCCM) guidelines. To facilitate weaning, the sedation strategy was transitioned from an initial thiopental infusion to dexmedetomidine, while continuous electrocardiographic monitoring was employed during the rapid correction of severe hypokalemia. The integration of early percutaneous dilatational tracheostomy (PDT), targeted diuresis, and resolution of ventilator-induced diaphragmatic dysfunction (VIDD) improved her PaO_2/FiO_2 ratio to 295 mmHg. In conclusion, successful ventilator liberation in complex neurocritical cases requires a rigorous, multidisciplinary approach. Integrating the ISCCM ABCDEFGHI bundle ensures the systematic correction of pathophysiological barriers, metabolic derangements, and sedation accumulation, leading to favorable clinical outcomes.

1. Introduction

The primary objective of mechanical ventilator weaning is to systematically minimize the duration of invasive ventilatory support, thereby mitigating the profound iatrogenic risks associated with prolonged endotracheal intubation.[1] While mechanical ventilation is a life-saving intervention for acute respiratory failure, it inherently bypasses the natural defense mechanisms of the upper respiratory tract, exposing the patient to severe hospital-acquired complications. Among the most critical of these are ventilator-associated pneumonia and ventilator-associated tracheobronchitis. These infectious pathologies are characterized by the microaspiration of virulent, nosocomial oropharyngeal flora past the endotracheal tube cuff, leading to an intense inflammatory cascade within the pulmonary parenchyma. Furthermore, prolonged positive pressure ventilation induces direct mechanical trauma to the pulmonary architecture, precipitating barotrauma from elevated airway pressures and volutrauma from alveolar overdistension. Beyond the

pulmonary parenchyma, the artificial airway itself induces airway trauma, increasing the risk of tracheal stenosis, mucosal ulceration, and vocal cord dysfunction. Prolonged controlled mechanical ventilation also triggers a rapid and deleterious physiological phenomenon known as ventilator-induced diaphragmatic dysfunction. This condition is characterized by a rapid onset of diaphragmatic myofibrillar atrophy and proteolysis, which can begin within hours of initiating mandatory ventilation, profoundly weakening the primary muscle of inspiration and complicating future liberation attempts. Consequently, the overarching goal of critical care management is to reduce the overall duration of mechanical ventilation to decrease both morbidity and overall intensive care unit mortality.

The transition from controlled mechanical ventilation to spontaneous breathing requires the fulfillment of rigorous standard readiness criteria. These criteria serve as physiological checkpoints to ensure the patient possesses the adequate cardiopulmonary reserve to tolerate the immense work of breathing required upon extubation. The foundational prerequisite is the substantial resolution or stabilization of the underlying primary pathology that initially necessitated intubation.[2] Following this, adequate oxygenation must be objectively demonstrated. Clinicians typically require a ratio of arterial oxygen partial pressure to fractional inspired oxygen (PaO_2/FiO_2) greater than or equal to 150 millimeters of mercury, or a peripheral capillary oxygen saturation greater than or equal to 90 percent while requiring a fractional inspired oxygen concentration of 40 percent or less. Additionally, the patient must tolerate a positive end-expiratory pressure of 5 centimeters of water or less, indicating a resolution of widespread alveolar collapse and an improvement in functional residual capacity. Hemodynamic stability is equally paramount; the patient must demonstrate adequate tissue perfusion and maintain a mean arterial pressure within physiological limits while requiring minimal or no vasopressor and inotropic support. Furthermore, the patient must exhibit an adequate level of consciousness, an intact cough reflex, and the ability to manage oropharyngeal secretions, ensuring the patency and protection of the airway post-extubation.

Despite the rigorous application of these readiness criteria, weaning failure remains a formidable and pervasive challenge in modern intensive care medicine. Weaning failure is classically defined by two adverse clinical outcomes: the inability to tolerate a spontaneous breathing trial or the necessity for reintubation within 48 hours following a planned extubation.[3] During a spontaneous breathing trial, failure manifests through profound physiological distress, including severe tachypnea, tachycardia, hypertensive urgencies, diaphoresis, the use of accessory muscles of respiration, paradoxical abdominal motion, and progressive hypercapnia leading to respiratory acidosis and altered mental status. Epidemiological data highlight the magnitude of this clinical hurdle, indicating that weaning failure affects approximately 20 to 30 percent of all mechanically ventilated patients globally.

The pathophysiology underlying weaning failure is exceptionally multifactorial, representing a complex interplay of systemic derangements rather than a singular organ deficit. This physiological complexity is particularly pronounced in geriatric populations.[4] The aging process inherently diminishes physiological reserves, characterized by decreased chest wall compliance, senile pulmonary hyperinflation, and a natural decline in central respiratory drive. When these age-related changes are compounded by frailty syndromes, generalized sarcopenia, and a high burden of chronic comorbidities, the geriatric patient faces a dramatically contracted margin for error during the weaning process.

Retrospective analyses and robust clinical registries indicate that specific patient cohorts exhibit disproportionately high rates of weaning failure. Notably, failure occurs in up to 61 percent of patients with pre-existing chronic respiratory disorders, most prominently chronic obstructive pulmonary disease. In chronic obstructive pulmonary disease, the

underlying pathophysiology involves chronic airflow limitation and the destruction of lung parenchyma, leading to dynamic hyperinflation. During mechanical ventilation, these patients frequently develop intrinsic positive end-expiratory pressure, commonly referred to as auto-PEEP. This auto-PEEP places a massive inspiratory threshold load on the already flattened, mechanically disadvantaged diaphragm. When support is withdrawn during a weaning trial, the patient often cannot generate the negative intrathoracic pressure required to overcome this threshold load, leading to rapid respiratory muscle fatigue and hypercapnic respiratory failure.[5]

Similarly, weaning failure occurs in approximately 41 percent of patients suffering from acute neurological impairment. In the neurocritical care setting, the cerebral pathology directly disrupts the central control of respiration located within the medulla and pons. Patients with severe traumatic brain injury, massive cerebrovascular accidents, or extensive intracranial hemorrhages often exhibit irregular breathing patterns, a blunted hypercapnic drive, and an inability to protect their airway due to profound bulbar dysfunction.[6] Furthermore, the management of neurocritical patients requires a delicate physiological balancing act; interventions designed to lower intracranial pressure, such as deep sedation, osmotic diuresis, and targeted temperature management, directly antagonize the awake, interactive, and euvolemic state required for successful ventilator liberation.

Furthermore, weaning failure is observed in 38 percent of patients presenting with severe hypoxemia, frequently driven by acute respiratory distress syndrome. In acute respiratory distress syndrome, widespread endothelial and epithelial injury leads to protein-rich pulmonary edema, alveolar collapse, and a profound reduction in static lung compliance.[7] The resulting right-to-left intrapulmonary shunting creates severe ventilation-perfusion mismatches. Although the acute inflammatory phase may resolve, the subsequent fibroproliferative phase leaves the lungs stiff and difficult to expand, requiring an immense mechanical effort from the respiratory muscles that often exceeds the patient's physiological capacity during a spontaneous breathing trial.

When a geriatric patient experiences the simultaneous convergence of these pathologies—specifically, concurrent acute brain injury requiring surgical intervention and severe pulmonary compromise stemming from chronic obstructive pulmonary disease and acute lung injury—the ventilatory management strategy becomes an extraordinary clinical challenge. The critical care team must continuously balance neuroprotective targets against lung-protective ventilation principles. Neuroprotection often mandates the strict avoidance of hypercapnia to prevent cerebral vasodilation and subsequent intracranial hypertension, requiring higher minute ventilations. Conversely, lung-protective ventilation emphasizes low tidal volumes and the acceptance of permissive hypercapnia to prevent mechanical lung injury. Navigating this diametrically opposed physiological crosstalk, known as the brain-lung axis, requires continuous, highly individualized adjustments to critical care parameters.[8]

To navigate the weaning process, critical care societies have developed comprehensive frameworks. International standards, particularly the guidelines jointly published by the American Thoracic Society and the American College of Chest Physicians, predominantly focus on the mechanical and procedural algorithms for conducting spontaneous breathing trials. These guidelines provide robust evidence regarding the optimal duration of trials, the choice between T-piece and pressure support ventilation, and the specific mechanical parameters that predict extubation success. However, while these algorithms are highly effective for general intensive care populations, they often fall short in cases of multimodal, refractory weaning failure where the barrier to extubation is systemic rather than purely respiratory.[9]

In response to the limitations of purely respiratory-focused algorithms, the 2023 Indian Society of Critical

Care Medicine guidelines offer a paradigm shift by providing a broader, more holistic, and systemic framework for difficult weaning scenarios. These guidelines emphasize the systematic identification, quantification, and correction of underlying metabolic, pharmacological, and physiological barriers that covertly sabotage the respiratory muscles. This systemic approach is encapsulated in the highly structured ABCDEFGHI bundle, making it exceptionally suitable for highly complex, resource-intensive tertiary care settings where multimodal failure is common and single-organ algorithms are insufficient.

The ISCCM framework mandates a rigorous evaluation of every physiological domain before committing to extubation. It requires optimizing Airway patency and managing anatomic dead space, often through interventions like percutaneous dilatational tracheostomy. It demands the optimization of Breathing mechanics by minimizing auto-PEEP and addressing severe bronchospasm. It dictates strict Cardiovascular optimization, ensuring euvolemia and treating occult myocardial ischemia that might precipitate cardiogenic pulmonary edema during the stress of weaning. It specifically highlights the danger of Delirium and the accumulation of sedative Drugs, advocating for the rapid clearance of neuro-depressants to restore central drive. Crucially, it focuses on Electrolyte optimization, recognizing that severe deficiencies in serum potassium, magnesium, and phosphate directly impair the generation of action potentials and the excitation-contraction coupling of the respiratory musculature. The framework also enforces meticulous Fluid management to drive a negative fluid balance and resolve interstitial lung water, emphasizes Gastrointestinal motility and optimal caloric provision to reverse malnutrition, and ensures the optimization of Hemoglobin levels alongside the eradication of underlying Infections to reduce systemic metabolic demand. Only by systematically resolving these diverse physiological barriers can a patient with highly complex, overlapping morbidities be safely transitioned to spontaneous breathing.[10]

The aim of this case report is to delineate the multidisciplinary management and physiological optimization required to successfully wean a highly complex geriatric patient who repeatedly failed standard liberation protocols. This study is novel because it presents a rare and exceptionally challenging pathophysiological triad: severe neurotrauma requiring an emergent re-craniotomy, underlying severe chronic obstructive pulmonary disease, and acute respiratory distress syndrome secondary to multidrug-resistant hospital-acquired pneumonia. Furthermore, this report explicitly demonstrates the clinical efficacy of stepping beyond traditional mechanical algorithms by integrating early percutaneous dilatational tracheostomy with the holistic, structured ISCCM 2023 weaning protocols—specifically the comprehensive correction of metabolic and fluid derangements—to achieve successful and safe ventilator liberation in a highly vulnerable patient.

2. Case Presentation

Ethical consideration

The ethical integrity of this case report is anchored in strict adherence to the principles outlined in the Declaration of Helsinki regarding medical research involving human subjects. Written informed consent was obtained directly from the patient's legally authorized representative, given the patient's compromised neurological status during the acute phase of her critical illness. This consent explicitly authorized the publication of her clinical narrative and completely anonymized radiological data for educational and scientific advancement. To guarantee absolute confidentiality, all personally identifiable information, including specific dates of admission, exact geographic identifiers beyond the hospital level, and recognizable demographic details, was systematically redacted from the manuscript. Furthermore, the institutional review board at Dr. Moewardi Regional General Hospital granted an exemption for this retrospective single-case report,

confirming that the dissemination of these clinical findings poses no risk to the patient while offering substantial instructional value to the broader critical care and neuroanesthesiology communities.

A 72-year-old woman was referred to the emergency department with a depressed level of consciousness following a fall in her bathroom seven days prior. Initial non-contrast head computed tomography (CT) at the referring hospital revealed an intracranial hemorrhage, for which she underwent a left-sided craniotomy. Persistent intracranial bleeding prompted a re-craniotomy 24 hours later to evacuate a right frontal epidural hematoma and right frontotemporoparietal subarachnoid hemorrhage. After one week of hospitalization without neurological improvement, she was transferred to the tertiary care Intensive Care Unit (ICU) at Dr. Moewardi Regional General Hospital, Surakarta, Indonesia, for advanced management (Tables 1 and 2).

Upon ICU admission, her medical history was notable for COPD, previously confirmed via spirometry, demonstrating an FEV_1/FVC ratio <0.70 with FEV_1 >80%. Admission chest radiography confirmed bilateral pneumonia. After initial stabilization and mechanical ventilation, the patient met weaning readiness criteria and was extubated on day two of her ICU stay. Post-extubation, she was supported with high-flow nasal cannula (HFNC) at 50 L/min and an FiO_2 of 50%. Her initial post-extubation parameters were stable: Glasgow Coma Scale (GCS) E3M6V4, SpO_2 97%, blood pressure 155/89 mmHg, and respiratory rate (RR) 16–22 breaths/min. However, 24 hours post-extubation (ICU day three), she suffered acute respiratory failure and neurological decline. She became somnolent (GCS E3M4V3) with an RR of 36 breaths/min and SpO_2 88%, despite HFNC escalation to a flow rate of 60 L/min and FiO_2 of 60%. Arterial blood gas (ABG) analysis confirmed acute-on-chronic type II respiratory failure with hypercapnia: pH 7.574, $PaCO_2$ 50.7 mmHg, PaO_2 73.3 mmHg, bicarbonate 27.3 mmol/L, and a PaO_2/FiO_2 ratio of 127 mmHg. Laboratory findings showed leukocytosis (29,300/mm³) and anemia (hemoglobin 8.8 g/dL).

Table 1. Clinical Timeline, Interventions, and Respiratory Mechanics

Comprehensive daily tracking of ventilator modes, gas exchange, fluid balance, and sedation strategies.

ICU DAY	GCS	VENTILATOR MODE	PIP / PS (CMH_2O)	PEEP (CMH_2O)	FIO_2 (%)	PAO_2/FIO_2 (MMHG)	FLUID BALANCE	SEDATION STRATEGY
Day 1	E3M6V4	PCV	PIP: 22	8	50	**210**	+1200 mL	Thiopental / Morphine
Day 2	E3M6V4	HFNC	N/A	N/A	50	**255**	+800 mL	Dexmedetomidine
Day 3	E3M4V3	PCV Reintubation	PIP: 25	10	50	**127**	+1500 mL	Thiopental resumed
Day 5	E2M4V(t)	PSIMV PDT placed	PS: 14	8	40	**180**	+900 mL	Transition to Dexmedetomidine
Day 10	E3M5V(t)	CPAP Failed	PS: 10	5	40	**165**	+Cumulative	Dexmedetomidine / Paracetamol
Day 11	E4M6V(t)	PSV	PS: 6	5	30	**295**	-800 mL Diuresis	Dexmedetomidine weaning
Day 12	E4M6V(t)	T-piece SBT Success	N/A	N/A	30	**310**	-500 mL	Off sedation
Day 16	E4M6V(t)	Trach Mask	N/A	N/A	21	**>350**	Euvolemic	**Discharged to Ward**

The patient was diagnosed with ARDS secondary to HAP and COPD exacerbation, complicated by cerebral edema. She was immediately reintubated. To ensure precise lung-protective ventilation while overcoming high airway resistance, she was placed on Pressure-Controlled Ventilation (PCV). Her settings were optimized to a Peak Inspiratory Pressure (PIP) of 25 cmH_2O and a PEEP of 10 cmH_2O, yielding a driving pressure of 15 cmH_2O. The FiO_2 was set at 50%. Her Clinical Pulmonary Infection Score (CPIS) exceeded 6, and subsequent sputum cultures isolated *Pseudomonas aeruginosa* (day 5) and *Acinetobacter baumannii* (day 11).

Table 2. Summary of Clinical Findings on Admission

Baseline demographic, neurological, and cardiopulmonary profiles upon transfer to the tertiary ICU.

CLINICAL PARAMETER	DIAGNOSTIC FINDING / STATUS
1. DEMOGRAPHICS & HISTORICAL CONTEXT	
Age & Gender	72-year-old Female
Mechanism of Injury	Ground-level fall (bathroom) 7 days prior to ICU transfer
Pre-existing Comorbidities	Chronic Obstructive Pulmonary Disease (COPD) Chronic
2. NEUROLOGICAL STATUS & NEUROSURGICAL HISTORY	
Primary Brain Injury	• Right frontal epidural hematoma Acute • Right frontotemporoparietal subarachnoid hemorrhage
Surgical Interventions	• Initial left-sided craniotomy (at referring hospital) • Right-sided re-craniotomy for persistent bleeding (24 hours post-initial surgery) Surgical
Neurological Presentation	Depressed level of consciousness with no improvement after one week of hospitalization
3. PULMONARY & DIAGNOSTIC STATUS	
Historical Spirometry	FEV_1/FVC ratio < 0.70 with FEV_1 > 80% (Confirming COPD baseline)
Admission Radiography	Bilateral pulmonary opacities / Bilateral pneumonia Acute
Initial Airway Status	Intubated and mechanically ventilated upon arrival for airway protection and stabilization
4. BASELINE VITAL SIGNS (INITIAL POST-EXTUBATION STABILITY, DAY 2)	
Neurological (GCS)	E3M6V4 Stable
Hemodynamics	Blood Pressure: 155/89 mmHg
Respiratory Rate	16–22 breaths/min
Oxygenation	SpO_2 97% on High-Flow Nasal Cannula (HFNC: 50 L/min, FiO_2 50%)

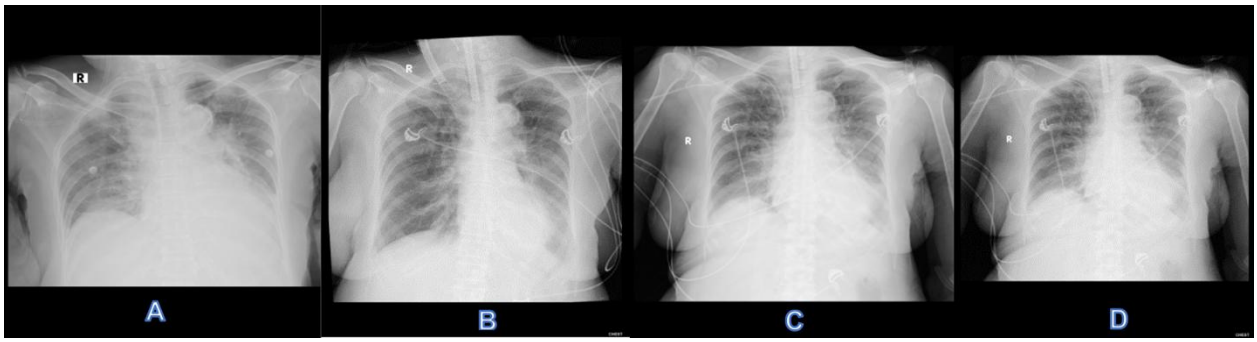

Figure 1. Radiological Evidence. The chest radiograph demonstrates bilateral pulmonary opacities consistent with ARDS. A: first day in ICU; B: Third day in ICU; C: 8th day in ICU; D: 11th day in ICU.

Neuroprotective sedation was required. Due to transient propofol supply chain limitations, a continuous thiopental infusion (50 mg/hour) was utilized initially to maintain strict intracranial pressure (ICP) control. However, recognizing that thiopental exhibits zero-order kinetics at high doses and possesses a prolonged context-sensitive half-time in geriatric patients due to extensive lipid distribution, the clinical team aggressively prioritized its cessation to facilitate eventual weaning. Sedation was transitioned to dexmedetomidine (0.2–0.7 μg/kg/hour), providing anxiolysis and sympathetic blunting without suppressing respiratory drive. Furthermore, analgesia provided via continuous morphine infusion was transitioned to intravenous paracetamol to prevent the accumulation of morphine-6-glucuronide (M6G), an active metabolite known to cause profound respiratory depression and altered mental status in elderly patients with fluctuating renal clearance (Table 3).

On ICU day five, early percutaneous dilatational tracheostomy (PDT) was performed to reduce anatomic dead space and decrease the work of breathing. The ventilator mode was transitioned to pressure support intermittent mandatory ventilation (PSIMV) temporarily to manage severe patient-ventilator asynchrony; however, acknowledging that SIMV can prolong weaning, she was transitioned to pure pressure support ventilation (PSV) as soon as synchrony was re-established. By day ten, during an observation period on continuous positive airway pressure (CPAP), she demonstrated severe weaning intolerance marked by tachypnea, tachycardia, and visible discomfort. A comprehensive pathophysiological evaluation revealed a profound metabolic crisis: serum potassium 2.4 mmol/L, serum magnesium 1.1 mg/dL, and serum albumin 2.3 g/dL. Because rapid correction of hypokalemia poses a high risk of arrhythmogenesis, potassium chloride (50 mEq over 6 hours) was administered under strict, continuous electrocardiographic (ECG) monitoring, alongside a continuous magnesium sulfate infusion (0.5 g/hour). Concurrent fluid overload was aggressively managed via continuous furosemide (5 mg/hour) to enforce a negative daily fluid balance, and albumin 25% was transfused. Following these interventions, the patient tolerated minimal support (pressure support 6 cmH_2O, PEEP 5 cmH_2O) by ICU day 11, with her PaO_2/FiO_2 ratio recovering robustly to 295 mmHg. On day 12, she successfully passed a 120-minute SBT, was liberated to a tracheostomy mask, and was discharged from the ICU on day 16.

3. Discussion

The successful liberation of this patient from mechanical ventilation required a profound understanding of and navigation through the complex, intersecting, and often competing pathophysiological demands of her neurocritical status and chronic pulmonary disease.

Table 3. Diagnosis and Treatment

Comprehensive summary of the clinical trajectory and multidisciplinary management.

1. COMPREHENSIVE DIAGNOSIS	
Primary Acute Pathologies	• Pulmonary Acute Respiratory Distress Syndrome (ARDS) secondary to Hospital-Acquired Pneumonia (HAP). • Infectious Sputum isolates: *Pseudomonas aeruginosa* and *Acinetobacter baumannii*. • Pulmonary Type II Respiratory Failure with hypercapnia ($PaCO_2$ 50.7 mmHg).
Underlying / Chronic Conditions	• Pulmonary Chronic Obstructive Pulmonary Disease (COPD) with auto-PEEP generation. • Neurological Status post re-craniotomy for right frontal epidural hematoma and right frontotemporoparietal subarachnoid hemorrhage.
Secondary Complications	• Metabolic Severe hypokalemia (2.4 mmol/L), hypomagnesemia (1.1 mg/dL), and hypoalbuminemia (2.3 g/dL). • Muscular Suspected Ventilator-Induced Diaphragmatic Dysfunction (VIDD). • Fluid Significant cumulative fluid overload contributing to interstitial pulmonary edema.
2. TREATMENT AND INTERVENTIONS	
Respiratory Support	• Reintubation and application of Pressure-Controlled Ventilation (PCV). • Early Percutaneous Dilatational Tracheostomy (PDT) performed on Day 5 to reduce airway resistance. • Transition to synchronous Pressure Support Ventilation (PSV) to counter diaphragmatic atrophy.
Pharmacotherapy & Sedation	• Sedation Immediate cessation of continuous thiopental; transitioned to dexmedetomidine (0.2–0.7 µg/kg/hour) to preserve respiratory drive. • Analgesia Transitioned from morphine to intravenous paracetamol to avoid active metabolite accumulation. • Antimicrobial Targeted antibiotic escalation based on susceptibility testing for isolated pathogens.
Metabolic & Fluid Optimization	• Continuous ECG-monitored potassium chloride infusion (50 mEq over 6 hours) and magnesium sulfate (0.5 g/hour). • Aggressive targeted diuresis via continuous furosemide (5 mg/hour). • Albumin 25% transfusion to increase intravascular oncotic pressure and enforce a negative fluid balance.

The patient's initial weaning failure on the third day of her intensive care admission was not an isolated pulmonary event, but rather the catastrophic culmination of the acute development of acute respiratory distress syndrome triggered by hospital-acquired pneumonia, superimposed on her severely compromised baseline respiratory mechanics from underlying chronic obstructive pulmonary disease.

To fully appreciate the magnitude of this failure, one must first deconstruct the baseline ventilatory limitations imposed by chronic obstructive pulmonary disease. In such patients, the architectural integrity of the lung parenchyma is degraded, resulting in a profound loss of elastic recoil and the destruction of alveolar tethering.[11] During exhalation, this loss of structural support leads to premature dynamic airway collapse, trapping air within the distal alveoli. Consequently, the patient requires a significantly prolonged expiratory time to fully empty the lungs. When the respiratory rate increases due to stress or infection, there is insufficient time for complete exhalation, leading to dynamic hyperinflation and the progressive generation of intrinsic positive end-expiratory pressure, clinically referred to as auto-PEEP. This auto-PEEP places an immense mechanical burden on the respiratory muscles; the diaphragm is pushed downward into a flattened, mechanically disadvantaged position, fundamentally altering the zone of apposition and severely impairing its force-generating capacity.

Upon this fragile baseline, the patient sustained a severe superimposed acute lung injury. The invasion of the lower respiratory tract by highly virulent, multidrug-resistant gram-negative pathogens, specifically *Pseudomonas aeruginosa* and *Acinetobacter baumannii*, triggered an overwhelming local inflammatory cascade. These pathogens release potent endotoxins and lipopolysaccharides that bind to pattern recognition receptors on alveolar macrophages, initiating a massive release of pro-inflammatory cytokines, including interleukin-1, interleukin-6, and tumor necrosis factor-alpha.[12] This cytokine storm orchestrates the rapid influx of neutrophils into the alveolar space. While attempting to eradicate the invading bacteria, these neutrophils release reactive oxygen species, proteases, and neutrophil extracellular traps, which inadvertently cause widespread collateral damage to the delicate alveolar-capillary membrane.[13]

The destruction of this barrier results in a profound increase in alveolar-capillary permeability. Protein-rich exudative fluid floods the alveolar spaces, diluting and inactivating pulmonary surfactant. The loss of surfactant dramatically increases alveolar surface tension, leading to widespread microatelectasis and a catastrophic reduction in static lung compliance. Mathematically, static lung compliance is defined as the tidal volume delivered divided by the difference between the alveolar plateau pressure and the total positive end-expiratory pressure.[14] As the alveolar units filled with inflammatory exudate and collapsed, the lungs became exceedingly stiff. Consequently, the driving pressure required from the ventilator, or the muscular effort required from the patient, to deliver a physiological tidal volume increased exponentially. Concurrently, her altered mental status stemming from the intracranial hemorrhages and necessary neurosurgical interventions severely blunted her central medullary respiratory drive and impaired her ability to protect her airway and clear copious purulent secretions. This triad of massive mechanical load, weakened muscular capacity, and depressed central drive inevitably culminated in rapid, hypercapnic type II respiratory failure upon her initial extubation.

A pivotal pathophysiological barrier discovered during her subsequent failed weaning attempt on day ten was the presence of severe, compounding metabolic derangements, specifically profound hypokalemia and hypomagnesemia. The generation of muscular force required for spontaneous breathing relies entirely on the precise electrophysiological balance of the sarcolemma. Potassium is the primary intracellular cation responsible for maintaining the resting membrane potential of muscle cells.[15] Severe hypokalemia hyperpolarizes the resting membrane potential, drastically increasing the threshold required to initiate an action potential. Furthermore, magnesium acts as an essential cofactor for the sodium-potassium adenosine triphosphatase pump and is critical for regulating calcium release and reuptake from the sarcoplasmic reticulum during the excitation-contraction coupling cycle. The profound depletion of these key electrolytes directly paralyzed

the biochemical machinery of the diaphragm and accessory respiratory muscles, severely impairing their contractility.[16]

However, this electrolyte depletion did not occur in isolation. Given the patient's mandatory requirement for controlled mechanical ventilation over the preceding days to manage her intracranial pressure and severe hypoxemia, ventilator-induced diaphragmatic dysfunction undoubtedly played a major compounding role. This phenomenon represents a rapid and specific form of disuse atrophy that occurs when the ventilator completely assumes the work of breathing, mechanically unloading the diaphragm.[17] Within hours of initiating controlled mechanical ventilation, a massive upregulation of proteolytic pathways occurs within the diaphragmatic myocytes, most notably the ubiquitin-proteasome system and the calpain-caspase cascades.

These pathways aggressively target and degrade myosin heavy chains, leading to physical myofibrillar atrophy, a reduction in muscle fiber cross-sectional area, and a significant drop in force-generating capacity. Furthermore, the absence of muscular contraction leads to intense mitochondrial dysfunction and the unchecked production of reactive oxygen species within the muscle tissue, further accelerating structural damage. When the clinical team attempted to transition the patient to continuous positive airway pressure on day ten, this structurally atrophied and biochemically depleted diaphragm was suddenly forced to contract against the massive resistive and elastic loads of her chronic obstructive pulmonary disease and resolving acute respiratory distress syndrome. The combination of structural protein loss and functional electrolyte depletion directly caused overwhelming respiratory muscle weakness, resulting in the clinically observed tachypnea, tachycardia, severe dyspnea, and ultimate weaning intolerance.

Another critical variable contributing to the refractory nature of her respiratory failure was the insidious development of fluid overload. In the critical care environment, patients frequently accumulate a massive positive fluid balance during the initial days of admission. This accumulation is driven by the necessity for aggressive initial hemodynamic resuscitation, the administration of obligatory carrier fluids for continuous intravenous medications, and the specific fluid requirements associated with neurosurgical interventions and maintaining cerebral perfusion.[18]

This systemic fluid overload directly and severely exacerbates pulmonary dysfunction. According to the Starling equation governing capillary fluid dynamics, an increase in intravascular hydrostatic pressure forces fluid out of the pulmonary capillaries and into the pulmonary interstitium. This extravascular lung water physically thickens the alveolar-capillary membrane. From a gas exchange perspective, this thickening exponentially increases the diffusion distance for oxygen, leading to refractory hypoxemia. From a mechanical perspective, the engorgement of the pulmonary interstitium with fluid further stiffens the lung parenchyma, sharply decreasing compliance and exponentially increasing the work of breathing required to inflate the lungs. Clinical outcome studies consistently indicate that a cumulative positive fluid balance exceeding ten percent of the patient's baseline body weight is an independent predictor of prolonged mechanical ventilation and an exponentially higher risk of weaning failure.[19]

To overcome this formidable barrier, the clinical team instituted a highly targeted deresuscitation strategy. By initiating a continuous infusion of furosemide, a potent loop diuretic that inhibits the sodium-potassium-chloride symporter in the thick ascending limb of the loop of Henle, the team forced the kidneys to excrete massive volumes of sodium and water. Concurrently, to ensure this diuresis mobilized fluid from the pulmonary interstitium rather than simply depleting the intravascular volume and risking hemodynamic collapse, hyperoncotic albumin was administered. The albumin artificially raised the plasma colloid osmotic pressure, pulling the extravascular lung water back into the vascular space where the furosemide could effectively clear it. This strategy successfully achieved a strict negative fluid

balance, resolving the interstitial edema, dramatically improving lung compliance, and directly facilitating the robust improvement of her oxygenation parameters.[20]

The successful liberation of this patient was ultimately achieved through the rigorous application of the 2023 Indian Society of Critical Care Medicine Committee on Weaning guidelines. Rather than relying solely on arbitrary mechanical thresholds, this framework provided the highly structured, algorithmic, and multidimensional approach necessary to systematically identify and reverse the compounding variables keeping the patient tethered to the ventilator. The clinical team systematically mapped every aspect of the patient's care to the comprehensive ABCDEFGHI screening criteria, successfully transforming a refractory, multi-organ failure state into a coordinated extubation success (Table 4).

A - Airway and Anatomy: The team recognized that the endotracheal tube itself imposed a massive resistive load on the patient. Following the principles of Poiseuille's law, breathing through a long, narrow artificial airway significantly increases the work of breathing. This was addressed via the performance of an early percutaneous dilatational tracheostomy on day five. This intervention drastically reduced anatomic dead space, bypassed the resistance of the upper airway and the endotracheal tube, and facilitated far superior pulmonary toilet for her copious pneumonia-induced secretions.

B - Breathing Mechanics: To mitigate the severe auto-PEEP inherent to her chronic obstructive pulmonary disease and to actively reverse the progressive Ventilator-Induced Diaphragmatic Dysfunction, the team transitioned the ventilatory strategy from heavily controlled modes to pure pressure support ventilation. This crucial shift allowed the patient to dictate her own respiratory rate and inspiratory flow, promoting patient-ventilator synchrony and encouraging active, physiological diaphragmatic contraction to rebuild lost muscle mass.

C - Circulation: Hemodynamic stability was aggressively maintained to ensure a euvolemic state and a mean arterial pressure strictly greater than 65 millimeters of mercury. This was absolutely vital to maintain an adequate cerebral perfusion pressure in the aftermath of her traumatic brain injury and re-craniotomy, utilizing targeted osmotic therapy with mannitol only when clinically indicated by neurological deterioration.

D - Drugs and Delirium: The pharmacological management of sedation was recognized as a major barrier. The initial use of thiopental and morphine, while necessary for acute intracranial pressure control, proved detrimental for weaning. These highly lipophilic agents exhibit massive context-sensitive half-times in elderly patients, accumulating in adipose tissue and causing prolonged central nervous system and respiratory depression long after the infusions are stopped. The clinical team executed a critical pivot, transitioning to dexmedetomidine, a highly selective alpha-2 adrenergic agonist. This provided excellent anxiolysis and blunted the sympathetic stress response without suppressing the medullary respiratory centers. Concurrently, transitioning to paracetamol provided adequate analgesia without the respiratory depressive effects of accumulating opiate metabolites.

E - Electrolytes: As previously delineated, the team executed an aggressive, continuously electrocardiographically monitored correction of her profound hypokalemia and hypomagnesemia, systematically restoring the electrochemical gradients required for forceful and sustained muscular contractility.

F - Fluid Balance: The team transitioned the patient from the inevitable positive balance of her acute resuscitation phase to a strict, protocolized negative fluid balance. The synergistic use of loop diuretics and targeted albumin transfusions effectively resolved her alveolar and interstitial edema, clearing the physical barriers to gas exchange and lung expansion.

G - Gastrointestinal: The profound energetic demands placed on the respiratory muscles during the weaning process require optimal nutritional support. The team ensured appropriate, carefully titrated enteral nutrition to fuel the cellular metabolism of the diaphragm, avoiding both the underfeeding that prevents tissue repair and the overfeeding that drives excess carbon dioxide production and exacerbates hypercapnia.

H - Heme and Infection: The underlying infectious catalyst driving the acute respiratory distress syndrome was systematically eradicated through targeted, culture-directed antimicrobial escalation specifically designed to penetrate the lung parenchyma and neutralize the multidrug-resistant *P. aeruginosa* and *A. baumannii*.

I - Interventions: Finally, the clinical team refrained from premature extubation attempts until all preceding physiological domains were optimized. Only when the metabolic, fluid, and infectious parameters were stabilized did they conduct a structured, protocolized spontaneous breathing trial using a T-piece. This served as a definitive physiological stress test, proving the patient possessed the cardiopulmonary reserve to safely separate from positive pressure support.

Table 4. Implementation of the ISCCM ABCDEFGHI Weaning Bundle

Systematic identification and targeted correction of pathophysiological barriers to mechanical ventilator liberation.

BUNDLE ELEMENT	CLINICAL ASSESSMENT & TARGETED INTERVENTION
A Airway & Anatomy	High anatomical airway resistance noted. **Early percutaneous dilatational tracheostomy (PDT)** was placed on Day 5 to significantly reduce anatomic dead space and resistance, facilitating enhanced pulmonary toilet.
B Breathing Mechanics	Mitigation of auto-PEEP and targeted management of Ventilator-Induced Diaphragmatic Dysfunction (VIDD). The ventilatory strategy was transitioned from controlled modes (PSIMV) to **synchronous pure Pressure Support Ventilation (PSV)** to encourage active, synchronous diaphragmatic work.
C Circulation	Hemodynamics were aggressively managed to maintain a euvolemic state and ensure adequate cerebral perfusion pressure. **Mean Arterial Pressure (MAP) > 65 mmHg** was maintained, utilizing targeted osmotic therapy with mannitol only when clinically indicated.
D Drugs & Delirium	A critical pharmacological pivot was executed. Accumulative neuro-depressants (thiopental and morphine) were completely halted and transitioned to **dexmedetomidine and intravenous paracetamol**, thereby preserving the central medullary respiratory drive while providing anxiolysis.
E Electrolytes	Profound hypokalemia (2.4 mmol/L) and hypomagnesemia (1.1 mg/dL) were identified as major barriers. **Aggressive, continuous ECG-monitored correction** with potassium chloride and magnesium sulfate was executed to restore muscular contractility and resting membrane potentials.
F Fluid Balance	The patient was transitioned from an acute positive fluid balance to a **strict negative balance**. This was achieved via continuous infusions of loop diuretics (furosemide) combined with albumin administration to successfully resolve alveolar and interstitial edema.
G Gastrointestinal	Appropriate, carefully titrated **enteral nutrition** was ensured to fuel the massive energetic cellular demands of the respiratory musculature during the weaning phase, avoiding both malnutrition and overfeeding.
H Heme & Infection	The underlying infectious catalyst driving the acute respiratory distress syndrome was eradicated through **targeted antimicrobial escalation** specifically aimed at the isolated *Pseudomonas aeruginosa* and *Acinetobacter baumannii*.
I Interventions	Only after all preceding physiological domains were systematically optimized did the team conduct a **structured, protocolized 120-minute Spontaneous Breathing Trial (SBT)** using a T-piece, which was executed successfully on Day 12.

4. Conclusion

Ventilator weaning failure in geriatric patients presenting with intersecting neurological trauma, chronic progressive respiratory disease, and acute invasive infectious pathologies represents an exceptionally complex clinical scenario that demands precise, systematic, and highly individualized management. The physiological margin for error in this demographic is exceedingly narrow, and failure to account for even one deranged parameter can precipitate a rapid cardiopulmonary collapse during the transition to spontaneous breathing. This case explicitly demonstrates the profound clinical utility of the 2023 Indian Society of Critical Care Medicine Committee guidelines in organizing and delivering advanced critical care. By rigorously identifying and correcting hidden metabolic and electrolyte derangements, strategically transitioning to neuroprotective and respiratory-sparing sedation profiles, strictly managing the insidious threat of fluid overload, actively resolving structural diaphragmatic dysfunction, and utilizing the mechanical advantages of early percutaneous dilatational tracheostomy, successful liberation from mechanical ventilation can be achieved. This protocolized, systemic approach proves that safe extubation is possible even in highly vulnerable patients burdened by the formidable, overlapping triad of severe acute respiratory distress syndrome, chronic obstructive pulmonary disease, and acute catastrophic brain injury.

5. References

1. Omar A, Taha A, Elmetwally T, Sivadasan P, Khalil MA. Early transition to airway pressure release ventilation may facilitate weaning and improve the outcome of acute respiratory distress syndrome patients. Egyptian J Intensive Care Emergency Med. 2021; 1(1): 1–14.
2. Aldabayan YS, Tolba AA, Alrajeh AM, Ahmed AT, Mahgoub AA, Glalah AAA, et al. Factors affecting mechanical ventilator weaning success and 28-day survival among patients with acute respiratory distress syndrome secondary to COVID-19. SAGE Open Nurs. 2023; 9: 23779608231187248.
3. Marshall G, Sanguinet J, Batra S, Foreman MJ, Peruchini J, Lopez S, et al. Association between ventilator-associated events and implementation of acute respiratory distress syndrome (ARDS) ventilator weaning protocol. Am J Infect Control. 2023; 51(12): 1321–3.
4. McNamara L, Baedorf Kassis E. Optimal positive-end expiratory pressure weaning in acute respiratory distress syndrome patients. Curr Opin Crit Care. 2024; 30(1): 85–8.
5. Feng Y, Sun Q, Guan C, Wang S, Wang P, Hu D. Effect of early pulmonary rehabilitation training on the prognosis of patients with acute respiratory distress syndrome after weaning of invasive mechanical ventilation in the intensive care unit. Zhonghua Wei Zhong Bing Ji Jiu Yi Xue. 2024; 36(3): 286–92.
6. Hermann M, König S, Laxar D, Krall C, Kraft F, Krenn K, et al. Low-frequency ventilation may facilitate weaning in acute respiratory distress syndrome treated with extracorporeal membrane oxygenation: a randomized controlled trial. J Clin Med. 2024; 13(17): 5094.
7. Duan Z, Xie H, Zhong H, Hu S, Chen X, Liu Z. Predictors for successful weaning from veno-venous extracorporeal membrane oxygenation in patients with severe acute respiratory distress syndrome. Risk Manag Healthc Policy. 2025; 18: 471–7.
8. P Thacker H, Dhekane A, Wadhwa N, Patil S. Itolizumab in addressing symptoms of acute respiratory distress syndrome (ARDS) with weaning off oxygen requirements in a COVID-19 patient: a case study. Indian J Immunol Respir Med. 2021; 6(1): 58–61.
9. López Rodríguez CE, Amezcua Gutiérrez MA, Gasca Aldama JC, Garduño López J. ΔPocc as a predictor in the weaning protocol from mechanical ventilation in patients with acute

respiratory distress syndrome. Am J Respir Crit Care Med. 2025; 211(Suppl_1): A1519.

10. Huang Y-Q, Yu P, Xiang D-D, Gan Q. Diaphragm ultrasound for predicting weaning success in post-cardiac surgery acute respiratory distress syndrome patients: a prospective observational study in China. Acute Crit Care. 2025; 40(3): 435–43.
11. Caroli A, Algera AG, van Meenen D, Schultz MJ, Paulus F, Neto AS, et al. Effect of a lower vs. Higher positive end-expiratory pressure strategy on clinically relevant outcomes in ICU patients without acute respiratory distress syndrome: Bayesian re-analysis of the REstricted vs. Liberal positive end-expiratory pressure in patients without acute respiratory distress syndrome (RELAx) randomized clinical trial. Crit Care Med. 2026.
12. Binh NG, Manabe T, Co DX, Thach PT, Tuan DQ, Van Cuong B, et al. Tuberculosis-induced acute respiratory distress syndrome treated with veno-venous extracorporeal membrane oxygenation. Respir Med Case Rep. 2019; 28(100900): 100900.
13. Maranatha D, Rahardjo P, Lusman R. Evolution of chest CT scan manifestations in a patient recovered from COVID-19 severe pneumonia with acute respiratory distress syndrome. Respir Med Case Rep. 2021; 32(101342): 101342.
14. Supranoto YTN, Negara IMPW. Meta-analysis of higher PEEP strategies' effects on mortality rates and inflammatory mediators in patients with ARDS: a perspective review on patients with severe COVID-19-associated ARDS. Solo J Anesth Pain Critical Care. 2023; 3(2): 75.
15. Cigolini MP, Schneider B, Bellé NL, Garcia TS, Moraes RB. Association between chest tomographic changes and weaning from mechanical ventilation in patients with severe acute respiratory syndrome due to SARS-CoV-2: a retrospective cohort study. J Emerg Crit Care Med. 2026; 10: 1–1.
16. Čučković M. Ultrasound guided assessment of the diaphragm in predicting success of weaning from mechanical ventilation. Lijec Vjesn. 2026; 148(1–2).
17. Ghiani A, Tsitouras K, Lutfi A, Barnikel M, Kempa AT, Kneidinger N. Tracheostomy timing and weaning outcomes following prolonged mechanical ventilation. BMC Pulm Med. 2026.
18. Béduneau G, Pham T, Schortgen F, Piquilloud L, Zogheib E, Jonas M, et al. Epidemiology of weaning outcome according to a new definition. The WIND study. Am J Respir Crit Care Med. 2017; 195(6): 772–83.
19. Guia M, Ciobanu LD, Sreedharan JK, Abdelrahim ME, Gonçalves G, Cabrita B, et al. The role of non-invasive ventilation in weaning and decannulating critically ill patients with tracheostomy: a narrative review of the literature. Pulmonology. 2021; 27(1): 43–51.
20. Dres M, Estellat C, Baudel J-L, Beloncle F, Cousty J, Galbois A, et al. Comparison of a preventive or curative strategy of fluid removal on the weaning of mechanical ventilation: a study protocol for a multicentre randomised open-label parallel-group trial. BMJ Open. 2021; 11(8): e048286.