"ANALYSING ASSOCIATION BETWEEN FLUID BALANCE AND EXTUBATION SUCCESS"

BY

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ABSTRACT

Introduction: Water constitutes approximately 45–75% of total body weight and is essential for physiological functions including metabolism, temperature regulation, and tissue maintenance. In critically ill, mechanically ventilated patients, fluid homeostasis is disrupted due to reliance on intravenous fluids and impaired physiological responses. Improper fluid management may lead to pulmonary complications such as edema, which can negatively impact extubation outcomes and overall prognosis. Evidence increasingly suggests that maintaining an optimal fluid balance may improve extubation success and reduce morbidity.

Objectives: Primary Objective: To examine the relationship between fluid balance from the day of intubation and extubation outcomes in patients mechanically ventilated for more than 24 hours who underwent successful spontaneous breathing trials (SBTs).

Secondary Objective: To derive an optimal input/output (I/O) ratio to guide fluid administration in critically ill patients.

Materials and Method: A prospective observational study was conducted over 12 months at BLDE, Shri B.M Patil Medical College Hospital and Research Centre, Vijayapura. The study included adult patients admitted to the ICCU and CCU who were intubated for medical reasons and mechanically ventilated for over 24 hours. Patients who completed an SBT using a T-piece or CPAP mode and underwent their first (index) extubation were included. Patients intubated before admission, post-operative or trauma cases, those extubated within 24 hours, or who underwent tracheostomy were excluded. Data were analyzed using JMP Pro 16 and SPSS v20. Statistical tests included the independent t-

test, Mann-Whitney U test, chi-square test, and ROC curve analysis. A p-value <0.05 was considered statistically significant.

Results: Among categorical variables such as gender, primary diagnosis, type of SBT, diuretic use, and dialysis, no significant associations were found with extubation outcomes. However, cumulative fluid balance showed a statistically significant difference between extubation success and failure groups. Other continuous variables like age, individual fluid input/output volumes, and duration of mechanical ventilation did not predict extubation outcomes. ROC analysis for the I/O ratio yielded an AUC of 0.558 with a non-significant p-value (0.140), indicating only marginal discriminative ability. A cut-off ratio of 1.129 offered moderate sensitivity and specificity but lacked predictive reliability as a standalone marker.

Conclusion: Cumulative fluid balance emerged as a potential key factor in predicting extubation outcomes, whereas the fluid input/output ratio alone did not serve as a reliable predictor. These findings underscore the complexity of fluid management in critically ill patients and support the integration of fluid balance metrics into broader, multivariate predictive models. Future studies should focus on refining these models to improve extubation success rates and patient outcomes.

Keywords: Cumulative fluid balance, mechanical ventilation, extubation failure, input/output ratio, critically ill, fluid management, spontaneous breathing trial.

LIST OF ABBREVIATIONS

- **AUC** - Area Under Curve
- **ADH** – Anti Diuretic Hormone
- **ANP** – Atrial Natriuretic Peptide
- **AVP** – Arginine Vasopressin
- **CKD** - Chronic Kidney Disease
- **CS** - Chi-Square
- **DBP** - Diastolic Blood Pressure
- **d** - Margin of Error
- **ED** - Emergency Department
- **ECF** – Extracellular fluid
- **IV** – Intravenous
- **ICF** – Intracellular fluid
- **K+** - Potassium Ion
- **mg/dL** - Milligrams per Deciliter
- **min-max** - Minimum-Maximum
- **mmHg** - Millimeters of Mercury
- **mmol/L** - Millimoles per Liter
- **Na+** - Sodium Ion
- **NPV** - Negative Predictive Value
- **NS** - Not Significant
- **n** - Sample Size
- **OR** - Odds Ratio
- **p** - Population Proportion
- **p-value** - Probability Value

- **PPV** - Positive Predictive Value
- **ROC** - Receiver Operating Characteristic
- **SBP** - Systolic Blood Pressure
- **SD** - Standard Deviation
- **SPSS** - Statistical Package for the Social Sciences
- **t-test** - Student's t-test
- **Z-score** - Standard Score

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INTRODUCTION.

About 45 to 75% of the human body weight is made up of water, making it one of the fundamental aspects of human life. Water is essential for metabolism, heat regulation, the structure and maintenance of tissues. The daily water requirement is estimated to be 20–60 mL/kg/day. In normal adults this requirement is met by water intake from drinking and eating foods. However, in critically ill and intubated patients, the main water source is IV fluids. This poses a significant issue: patients cannot respond to their internal homeostasis, and fluids are administered externally without full knowledge of their physiological status (1,2).

Water leaves the body through urine, feces, and insensible losses such as respiration and sweating. In conditions like burns and sepsis, there is a substantial fluid shift into the interstitial space due to peripheral vasodilation and inflammation. This causes physiological trapping of total body water, effectively rendering it unavailable. The kidney plays a major role in water regulation—about two-thirds of the water reaching the proximal convoluted tubules is reabsorbed through active sodium transport. The water entering the collecting duct is regulated by antidiuretic hormone (arginine vasopressin), secreted from the pituitary. This hormone maintains an approximate urine output of 0.5 mL/kg/hour in healthy adults. All these factors—fluid intake and solutes, vasopressin's hormonal activity, hypothalamic control, renal excretion of solutes and water, and physiological stressors causing fluid retention—make fluid balance a complex physiological process that requires utmost attention when caring for the critically ill(3,4,5).

Fluid regulation in and around the pulmonary microcirculation is another area of interest. The exchange of fluids in the pulmonary microvasculature is maintained by Starling forces. Fluid is pushed out of the capillaries due to

hydrostatic pressure, or it can be pulled into the interstitium due to interstitial oncotic pressure. Recent studies suggest that albumin leaks out of the pulmonary capillaries through transcellular pathways to maintain hydration of the interstitium. This albumin is eventually cleared through the lymphatic system. Since there are no lymphatic vessels at the pulmonary capillary bed, the newly formed interstitial fluid moves along the interstitium to the perivascular and peribronchial areas, where it drains into lymphatic vessels and later to the hilar nodes. The estimated pulmonary lymphatic flow is 20 mL/hour. Lymph flow is hindered by positive intrathoracic pressure caused by mechanical ventilation, which contributes to the development of pulmonary edema. Fluids in the interstitium are prevented from entering the alveoli through tight junctions and active sodium transporters, which pump sodium to the interstitial space, pulling water along with it out of the alveoli. Pulmonary edema develops in two stages. Initially, there is interstitial edema characterized by peribronchial and perivascular interstitial tissue engorgement. Alveolar function is not affected at this stage, but as fluid accumulation worsens, it overwhelms the alveoli's capability to clear the fluids, leading to alveolar edema and loss of function. Similarly, in ARDS, an excessive or positive fluid balance has been shown to be associated with severity, duration of mechanical ventilation, and mortality due to worsening pulmonary edema. A conservative fluid strategy has proven beneficial by reducing length of ICU stay and duration of mechanical ventilation in children and adults, making it a therapeutic strategy in ARDS (6,7,8,9).

long duration mechanical ventilation is associated with increased mortality and poor prognosis. there have been studies substantiating the benefit of maintaining a negative fluid balance in the day prior to extubation. similary studies have shown positive fluid balance as a risk factor for re-intubation. Although past research has demonstrated that aggressive fluid resuscitation improves

outcomes, the patients may suffer as a result of this mindset. Emerging research from a variety of sources suggests that ICU patients who achieve a negative fluid balance have better results in terms of morbidity and death. In sepsis and kidney disorders, a positively tilting fluid balance is linked to an increased risk of mortality. Moreover, it has been demonstrated to lengthen mechanical ventilation times and cause pulmonary problems. Pathophysiologically, a positive fluid balance will lead to an increase in capillary leakage, which will increase lung volume and result in a decrease in lung compliance, which will cause respiratory distress during the attempt at spontaneous breathing and immediately after extubation, ultimately leading to extubation failure. As a result, this study is planned as a prospective observational study to provide more knowledge about fluid balance in critically sick patients and to help with the appropriate titration of resuscitative fluids following first resuscitation.(10,11,12)

AIM AND OBJECTIVE

Primary Objective: To examine the relationship between fluid balance from the day of intubation and extubation outcomes in a range of patients who underwent successful trials of spontaneous breathing and were mechanically ventilated for longer than 24 hours.

Secondary Objective: To derive an optimal input to output ratio, to aid in fluid administration of critically ill patients.

REVIEW OF LITERATURE

FLUID BALANCE: REGULATION OF WATER INTAKE AND LOSS

Fluid balance is a state of equilibrium where the body's intake matches its output. This balance is regulated by mechanisms involving thirst, kidney function, and hormonal control to maintain homeostasis. Water intake is influenced by thirst, habits, and socioeconomic factors. Additionally, water comes from food and is produced through the metabolic oxidation of fats and carbohydrates(13).

Fluid intake is primarily controlled by thirst, which individuals perceive as dryness in the mouth and throat due to decreased salivary secretions. A subtle change in serum osmolarity is detected by osmoreceptors in the hypothalamus, relayed to the cerebrum, and consciously experienced as thirst. The osmoreceptors in the anterior hypothalamus shrink in response to dehydration-induced hypertonicity, with just a 1-2% rise in serum osmolarity triggering the thirst response. In conditions involving extracellular fluid loss, such as hemorrhage and gastrointestinal loss, baroreceptors in the carotid sinus and aortic arch—along with volume receptors in the cardiac atria and great vessels—stimulate the thirst center. This mechanism activates only when plasma volume drops by more than 10%. The renal system also helps detect volume loss through kidney volume receptors, which activate the renin-angiotensin system to preserve fluids. These two mechanisms typically work together, as seen during excessive sweating from strenuous exercise or in tropical climates. Since sweat is a hypotonic fluid, its excessive loss increases serum osmolarity and reduces plasma volume, leading to both intracellular and extracellular fluid loss (13,14,15,16).

Water is primarily lost through the kidneys, with normal urinary output being around 1500ml/day for an average healthy adult. The renal system maintains strict control over homeostasis, with urine concentration varying from 400ml/day to 23L/day depending on the body's needs. While fluid intake can be reduced to zero, the combined fluid output through urine and insensible losses cannot fall below 1200ml/day. When the body detects decreased sodium levels, renin—stored in the juxtaglomerular apparatus—is released into the plasma. This occurs through three main mechanisms(14,17).

First, reduced ECF volume leads to increased sympathetic activity in arterioles via the baroreceptor reflex. The sympathetic nerves innervating the juxtaglomerular cells in the renal afferent arteriole stimulate renin release. Second, decreased wall tension in the afferent arterioles from reduced renal perfusion triggers renin release, with the arteriole acting as a baroreceptor. Third, reduced NaCl delivery to the macula densa activates cyclo-oxygenase-2, which releases PGE2 and facilitates renin release. Once in the bloodstream, renin converts angiotensinogen (an alpha2-globulin plasma protein) into angiotensin I. Surface endothelial cells contain angiotensin converting enzyme (ACE), which rapidly converts angiotensin I to angiotensin II by removing two amino acids. Angiotensin II is the primary hormone in sodium regulation. It acts as a potent vasoconstrictor, causing vasoconstriction of the efferent arteriole. Angiotensin II also induces sodium reabsorption from the proximal tubules by activating Na+/H+ exchange. This angiotensin II then acts on the AT1 receptor of the adrenal cortex to release aldosterone. It also stimulates ADH release and aids in water retention(18,19,20).

Other routes of water loss occur through the skin and lungs (known as insensible loss) and through the GI tract. Invisible water loss through the skin amounts to about 450ml per day in temperate climates. The respiratory tract loses around 250-350ml per day through evaporation, while 200ml of water is

lost through feces. These insensible losses vary depending on climate, temperature, and humidity. Sweat evaporation is the body's only mechanism for controlling hyperthermia. When exercising in hot environments, individuals can lose up to 1-2L of water per hour, leading to dehydration and hyperosmolarity of the extracellular space. Sweat is hypotonic compared to ECF, containing 25-50 mmol/L of sodium, which means that during heavy sweating, proportionally more water is lost than sodium.

BODY COMPARTMENTS AND FLUID COMPOSITION.

Fluids in the human body are constantly in flux through various regulatory mechanisms to maintain equilibrium across body compartments. The intracellular environment makes up 40% of total body weight and maintaining appropriate osmolality within cells is crucial for healthy cellular metabolic activity. The extracellular fluid compartment comprises 20% of body weight and can be further divided into plasma (5%) and interstitial space. Transcellular fluid, as in CSF, aqueous humour of eye, renal tubular fluid accounts for another 1 to 2%. In pathological conditions, additional compartments may form depending on the disease's cause and location (21).

The chemical composition differs significantly between extracellular and intracellular spaces. The extracellular space contains higher concentrations of sodium, chloride, proteins, and bicarbonates, with low levels of potassium, magnesium, and phosphates. While the interstitial space is similar to the extracellular space, it has relatively lower protein concentrations. In contrast, the intracellular environment contains high levels of phosphate, magnesium, and proteins but minimal levels of sodium, chloride, and bicarbonates (22).

Osmolarity—the number of particles in a liter of fluid—is one of the main regulatory gradients for fluid balance within the cellular environment. Blood normally maintains an osmolarity of 286 mOsmoles/L. When osmolarity

deviates from this level, it creates either a hypo-osmolar (below normal) or hyperosmolar (above normal) state. Blood maintains its osmolarity through two primary mechanisms: absorbing solutes from the alimentary canal and secreting solutes into the gastrointestinal tract or urine. Plasma proteins (like albumin), glucose from the gastrointestinal tract, and urea from protein metabolism also help maintain blood osmolarity although to a smaller extent. The osmolarity of the extracellular fluid (ECF) and intracellular fluid (ICF) must remain balanced. If not, fluid will move from the hypo-osmolar compartment to the hyperosmolar compartment, causing cells to either shrink or swell depending on the direction of water movement (21, 23,24).

Altering the osmolality of the ECF induces a predictable change in the size of the intra and extracellular compartments. adding water to the ECF, reduces its osmolarity. causing a shift of water from ECF into the intracellular space, till the osmolarity comes to equilibrium. this causes the cells to swell up. the end osmolarity of both compartments will be diminished. whereas if instead of water isotonic saline is introduced to the ECF, being isotonic, it will increase the size of extracellular space with no change in osmotic pressure, and so no movement of water into or out of the cells. similarly adding hypertonic saline to the ECF pulls water from the ICF, shrinking the cells and increasing intracellular osmolarity (21,24).

STARLINGS FORCES:

In 1894, Starling explained the basic forces producing fluid shift between intracellular and extracellular spaces. These forces are tissue pressure (Pt), capillary pressure (Pc), plasma colloidal osmotic pressure (Op), and tissue colloidal pressure (Ot). In 1963, Guyton and colleagues introduced the concept of interstitial fluid pressure and the physiological behavior of the interstitium. Hydrostatic pressure (Pc) pushes plasma out of circulation toward the

endothelial surface. The fluid's ability to cross the membrane depends on the membrane's permeability and surface area. However, plasma proteins and other large molecules cannot pass through the capillary membrane and remain in the intravascular space, exerting an opposing force that pulls fluids back into the plasma. This force is known as oncotic or osmotic pressure (Oc) (25).

Therefore, net fluid movement = K(Pc + Pt) - (Oc + Ot), where K is the filtration coefficient, which accounts for membrane permeability and surface area.

Starling hypothesized that capillary filtration (Pc - Pt) equals oncotic absorptive pressures (Oc - Ot). He proposed that capillary fluid exchange is self-regulated—when Pc changes, Pt and Ot adjust in opposing ways to counterbalance it. In 1920, Landis's landmark study modified Starling's hypothesis. Landis found that increased capillary filtration resulted from increased capillary surface area rather than hydrostatic pressure. He termed the tissues' ability to prevent edema formation the "margin of safety." Years later, Guyton redefined this phenomenon as the "tissue edema safety factor." When capillary pressure rises beyond this safety factor, interstitial edema develops (26,21,39).

ELECTROLYTES AND ION BALANCE.

Electrolytes exist as ions in plasma. Positively charged ions are called cations, while negatively charged ions are called anions. These ions maintain electrical neutrality by existing in equilibrium, meaning the total number of anions equals the total number of cations. Sodium is the major cation in the ECF and the primary determinant of plasma volume. While other cations like potassium and magnesium are present in the ECF, their concentrations are relatively negligible compared to sodium. Water diffuses to regions with high sodium content to maintain fluid osmolarity. Plasma sodium ranges between 135-145 mmol/L.

Sodium mainly comes from dietary consumption, averaging 10-15mg per day. It is excreted primarily through urine (approximately 10g per day) and sweat (around 0.5g). The glomerulus filters sodium mainly in the proximal convoluted tubules, making kidneys the primary regulators of sodium and ECF volume. Hormonal regulation occurs through aldosterone, which aids in sodium retention via the renin-angiotensin-aldosterone system (RAAS) triggered by low circulating ECF. Conversely, excess ECF volume triggers the release of atrial natriuretic peptide, which aids in sodium excretion and reduces ECF volume (27).

Potassium is the primary cation in ICF, with an intracellular concentration of 150mmol/L compared to 3.5-5mmol/L in the ECF. Like sodium, potassium comes mainly from dietary intake and is regulated by the RAAS system, intracellular buffering, and nephron reabsorption. Beta-2 agonism and insulin promote intracellular uptake of potassium. Na+/K+ ATPase pumps in the cell membrane regulate intracellular potassium. Potassium naturally leaves cells due to the electrochemical gradient, reducing intracellular osmolarity. This movement prompts fluid to enter the cells, causing swelling. To prevent this, intracellular sodium is pumped out using ATP to maintain osmotic equilibrium. Potassium excretion is strictly regulated: most is reabsorbed at the PCT, and filtered potassium reaching the distal nephron is reabsorbed through the actions of both aldosterone and arginine vasopressin (AVP)(28).

Phosphates are the primary intracellular anions, with 80% stored in bones and regulated by parathyroid hormone and Vitamin D. Phosphates filter freely in the glomerulus and are reabsorbed with sodium; 20% of filtered phosphates are excreted in urine. Intracellular proteins also act as anions to maintain electrical neutrality. In the ECF, chlorides and bicarbonates serve as the primary anions (28).

HORMONAL REGULATION OF FLUIDS.

Several key hormones govern fluid balance by regulating water and electrolytes to maintain homeostasis. Among these, Antidiuretic hormone (ADH), aldosterone, and atrial natriuretic peptide (ANP) play crucial roles.

ADH is a nine-amino-acid peptide hormone that increases the permeability of renal collecting ducts to water, thereby reabsorbing water without sodium (free water reabsorption) and creating concentrated urine. ADH is produced in the hypothalamus (supraoptic and paraventricular nuclei) and stored in the posterior pituitary. It is also called arginine vasopressin (AVP) due to its vasopressor activity. The secretion of ADH is controlled by hyperosmolality and hypovolemia; ADH is the only mechanism by which both thirst and hyperosmolality regulate fluid balance together.

Osmoreceptors located in the supraoptic nuclei are stimulated by the osmotic gradient between cytoplasm and plasma. Since sodium is the principal solute in the ECF compartment, plasma sodium is the major osmotic factor controlling ADH secretion. The carotid sinus baroreceptors contain stretch receptors that stimulate the paraventricular nuclei of the hypothalamus. With reduced stretch, these receptors relay impulses to the vasomotor center of the brainstem and cause ADH release.

The osmotic stimulus is more sensitive than the volume-directed stimulus—even a 1% change in osmolality can trigger this pathway, whereas only substantial volume depletion of the ECF (more than 10%) will trigger ADH release. Volume regulation overrides osmoregulation, and once stimulated, volume regulation triggers more ADH release. Other factors controlling ADH release include nausea, hypoglycemia, and angiotensin II stimulation.

Endogenous opiates and high doses of morphine also stimulate ADH release, while ethanol inhibits its release.

ADH acts on the P cells (principal cells) of the collecting duct through ADH receptors (V2 receptors) situated on the basolateral membrane. This triggers cyclic adenosine monophosphate and stimulates aquaporin-CD into the luminal membrane, increasing the water permeability of the collecting duct. V2 receptor activation also stimulates prostaglandin E2 synthesis, which opposes ADH's action and forms a negative feedback loop. V1a receptors, found on vascular smooth muscles, cause vasoconstrictor effects through phosphoinositol pathway activation. V1a receptor stimulation also promotes procoagulant factors and platelet adhesion. Additionally, ADH stimulates potassium excretion in the distal nephron (29,30,31).

The renin-angiotensin-aldosterone system is another hormone-driven regulatory pathway of fluid balance. Renin release is regulated by renal vascular receptors, macula densa receptors, renal sympathetic nerves, plasma sodium concentration, and catecholamines. Renin, the rate-limiting enzymatic step in angiotensin II synthesis, is synthesized, stored, and released from the juxtaglomerular cells of the afferent arterioles into intravascular space. There are three mechanisms for renin release. First, the JG cells of the macula densa act as high-pressure baroreceptors and detect changes in blood pressure. An increase in renal arterial pressure inhibits renin release, while decreased renal artery pressure (as in severe hypovolemia) reduces stretch, decreases intracellular calcium concentration, and stimulates renin release. Second, sodium delivery to the thick ascending limb of the loop of Henle affects renin release. Increased sodium delivery inhibits renin release, while decreased sodium delivery to the distal nephron is detected by the macula densa, triggering renin release. This release is regulated by upregulation of cyclooxygenase-2 in the macula densa, which increases PGE2 and thereby

increases intracellular cAMP. Third, since JG cells are innervated by sympathetic nerve fibers, renal sympathetic activation and beta-adrenergic receptor stimulation increase renin release.

Increased renin release leads to increased circulating angiotensin II, which causes vasoconstriction of renal and systemic arterioles, increasing total peripheral resistance and blood pressure. In the brain, angiotensin II stimulates sympathetic drive to the heart and vasculature, which increases cardiac output and total peripheral resistance. Angiotensin II promotes water retention by increasing ADH release, stimulating thirst, and expanding blood volume. It also causes renal sodium conservation through aldosterone stimulation from the adrenal cortex. Beyond aldosterone activity, angiotensin acts through AT1 receptors in the luminal and basolateral membranes of proximal and distal nephron segments to cause sodium resorption. It also increases sodium-chloride cotransporter activity in the distal tubules and collecting ducts. Through these direct and indirect pathways, angiotensin promotes sodium and water retention (18,19,20,34).

Aldosterone is a steroid (mineralocorticoid) hormone synthesized in the zona glomerulosa of the adrenal cortex. It is the final endocrine stimulus in the renin-angiotensin-aldosterone system (RAAS). Aldosterone is stimulated by angiotensin II, as discussed previously, and by increased circulating potassium, which enhances the activity of steroidogenic enzymes and steroidogenic acute regulatory protein (StAR). It is regulated through a negative feedback loop with blood pressure—when blood pressure drops, aldosterone release increases. The P cells of the collecting duct are aldosterone-sensitive cells containing mineralocorticoid receptors (MR). Aldosterone binds to these receptors to form an aldosterone-MR complex, which regulates specific gene transcription. This induces increased opening of ENaC channels and sodium-potassium ATPase

activity, leading to increased sodium reabsorption with variable chloride ion reabsorption and potassium secretion (32,33).

Atrial natriuretic peptide (ANP) is another hormone involved in fluid balance. It is unique because it corrects low circulating volume by decreasing its secretion. As its name suggests, ANP promotes natriuresis (sodium excretion). Synthesized and stored in atrial myocytes, ANP is released when the atrial wall stretches—consequently, low circulating volume inhibits its release. Its activity, mediated through guanylyl cyclase, has many synergistic effects on renal circulation and water excretion. ANP decreases renin release, independently inhibits aldosterone, and reduces AVP release. Its actions include direct and indirect inhibition of sodium reabsorption in the glomerulus, inhibition of angiotensin II-stimulated sodium reabsorption, inhibition of renin secretion by reducing macula densa tonicity, and inhibition of aldosterone-mediated sodium reabsorption in the distal nephron(35).

FLUID PHYSIOLOGY IN LUNG

Optimum gas diffusion in the air-blood barrier is possible only through adequate control of ECF water volume to prevent edema-induced tissue swelling and alveolar edema. The normal air-blood barrier does not exceed 0.2 micrometers. An extremely low microvascular permeability and dynamic remodeling of the interstitial matrix prevent alveolar edema. The air-blood barrier (ABB) is a specialized structure that minimizes thickness and maximizes surface area (100m^2). The ABB has a "thin portion" covering the alveolar surface that facilitates gaseous exchange, and a "thick portion" containing the extracellular matrix and lymphatics, serving as a fluid reservoir. Type 1 epithelial cells form the major component covering up to 95% of the alveolar surface, while the remaining 5% is made up of type 2 epithelial cells. The ABB regulates microvascular permeability and provides mechanical support through

endothelial cells, the interstitial compartment, and other structures like collagen, elastic fibers, and proteoglycans.

In physiological conditions, the lung interstitial compartment maintains a sub-atmospheric hydrostatic pressure (~10 cmH2O), with fluid movement governed by Starling's equation. The Starling pressure gradients, controlled by hydrostatic and oncotic forces, regulate transcapillary and transepithelial water exchange, ensuring minimal water accumulation in normal conditions. The lung's water balance is measured using the wet-to-dry ratio (W/D). Fluid permeability in the alveolar compartment is restricted due to low hydrostatic conductance and high protein reflection coefficient, making the epithelium nearly impermeable to proteins. The alveolar capillary network exhibits structural compartmentation, with the true alveolar district having a vast surface area but minimal contribution to albumin flux. In the physiological state, fluid exchange remains minimal due to the interplay of low permeability and Starling gradients. However, alterations in alveolar pressure, particularly during mechanical ventilation, could significantly impact fluid dynamics and potentially lead to lung edema.

under normal conditions the hydrastatic pressure is around -10 cmH2O, but in response to edemagenic conditions, like hyperventilation or increased cardiac output during exercise it can rise upto +5 cmH2O, this is due to hyalurinan capturing the excess interstitial water and morphing into a gel like structure that increases steric hinderance and prevents excessive fluid accumulation. as long as microvascular permeability remains intact this mechanism acts as a safety factor against edema. Experimental studies using a biomimetic system has shown that increased extracellular matrix rigidity enhances barrier properties. Additionally, respiratory mechanics assessed through low-frequency forced oscillation technique (FOT) in mechanically ventilated rats suggests that early lung tissue changes can be detected before severe fluid accumulation occurs.

However, if the interstitial matrix structure is compromised, hydrostatic pressure drops to 0 cmH_2O, leading to a critical increase in W/D ratio and loss of this protective mechanism, making lung edema more likely.

The lungs have a complex defense system to maintain fluid balance by controlling microvascular filtration. When fluid begins to accumulate, the initial response occurs in the thick portion of the air-blood barrier (ABB), which contains lymphatic vessels. When the water-to-dry weight ratio (W/D) reaches about 5.5, endothelial cells swell to 45% above their normal volume, while most of the excess fluid (approximately 85%) is stored in the thick portion of the ABB. The thin ABB, being relatively rigid, channels fluid through a high-resistance pathway to the more flexible thick ABB.

Pulmonary lymphatics play a crucial role in fluid homeostasis. These vessels extend deep within the pulmonary lobule, forming a peripheral network that reaches the perimicrovascular district near alveoli, alveolar ducts, and interalveolar septa. These lymphatic vessels then merge into larger conducting pathways along the broncho-vascular bundle. Under normal conditions, the baseline lymph flow rate is 0.063 ml/min/100 g of lung tissue. While only 4% of fluid removal occurs through alveolar clearance via active sodium transport, most fluid is reabsorbed into capillaries through Starling forces. The lymphatics drain approximately 18% of the remaining fluid.

Lymphatic flow is capable of increasing significantly in response to excess filtration, providing a negative-feedback mechanism to help regulate extravascular lung volume. Notably, lymph flow is directly proportional to lung weight gain, which reflects microvascular filtration rate. Lymphatic vessels generate a subatmospheric pressure that facilitates interstitial fluid movement. Mathematical models suggest that, under normal conditions, a threefold increase in lymphatic flow can stabilize interstitial pressure at approximately 2

cmH_2O. However, in cases of excessive interstitial hydration, the extracellular matrix degrades, lowering tissue resistance and increasing fluid movement toward lymphatics. Experimental lung injury studies have demonstrated that lymph flow can increase up to 10-fold.

Mechanical ventilation and positive end-expiratory pressure (PEEP) can hinder lymphatic drainage by increasing intrathoracic and central venous pressures, exacerbating pulmonary edema. When lung W/D surpasses 35% of baseline (~6.25), lymphatic clearance becomes insufficient, leading to alveolar flooding. As fluid accumulates in the lungs, alveolar compliance declines due to the loss of functional airspaces.

Mathematical models suggest that interstitial pressure helps buffer and even reverse transcapillary Starling forces. However, excessive fluid buildup compresses capillaries, reducing their surface area and decreasing permeability (Kf). Studies confirm that edematous lung regions experience reduced blood flow even when vasodilators are administered. Precapillary vasoconstriction serves as an additional protective mechanism, redistributing blood from edematous to healthier lung regions. Individuals vary in their susceptibility to pulmonary edema based on their capillary recruitment patterns, which are influenced by exercise and hypoxia exposure. While higher pulmonary capillary density improves oxygen transport, it also increases the risk of pulmonary edema due to greater filtration surface area. Pulmonary hypertension during hypoxia is believed to be a protective response driven by precapillary vasoconstriction, which limits excessive microvascular filtration.

Several interventions have been explored for preventing pulmonary edema. Gadolinium has been shown to protect against lung injury by blocking stretch-activated cation channels that otherwise increase intracellular calcium and microvascular permeability. Another potential strategy targets NADPH oxidase

type 2 (NOX2), a major source of reactive oxygen species (ROS) in the lung. NOX2 inhibitors have been proposed to mitigate inflammation associated with oxidative lung injury. Additionally, dietary antioxidants may offer a protective effect against oxidative stress-related lung damage in mechanically ventilated patients.

In conclusion, pulmonary lymphatics and interstitial pressure play crucial roles in maintaining lung fluid balance and preventing edema. However, excessive microvascular filtration, mechanical ventilation, and loss of interstitial matrix integrity can overwhelm these compensatory mechanisms, leading to alveolar flooding and decreased lung compliance. Precapillary vasoconstriction, lymphatic drainage enhancement, and pharmacological interventions targeting oxidative stress and endothelial permeability represent promising strategies for managing pulmonary edema (36-42).

MEASUREMENT OF TOTAL BODY WATER

Total Body Water (TBW) is a critical component of body composition and essential for clinical assessments, particularly in fluid balance disorders. Scientists initially estimated TBW by comparing the weights of fresh versus desiccated animal carcasses. Later advancements introduced tracer dilution techniques using markers such as tritiated water (^3H_2O), deuterium oxide (^2H_2O or heavy water), antipyrine, and less commonly, urea, thiourea, and ethanol. ^2H_2O is now preferred because it's non-radioactive and safer for clinical use. Water labeled with stable isotope ^18O (H_2^18O) serves as another option.

These isotope-based methods slightly overestimate TBW because of exchange reactions with body constituents. ^2H exchanges with protein protons while ^18O reacts with inorganic pools, with ^2H showing a higher exchange rate. As

a result, TBW estimates using ^2H_2O are about 3.5% higher than those from H_2^18O.

Recent efforts have produced less invasive methods, including Dual-Energy X-ray Absorptiometry (DEXA), air displacement plethysmography, nuclear magnetic resonance spectroscopy, and bioelectrical impedance analysis (BIA). BIA has become the clinical standard due to its affordability and ease of use, particularly for managing chronic dialysis patients. While these newer methods use empirical formulas derived from reference methods, their accuracy depends on underlying assumptions that may not apply to all patients.

Anthropometric formulas calculate TBW using height, weight, age, gender, and ethnicity through statistical regression. Though widely used in adults, these formulas have key limitations. They don't account for individual variations in body composition, especially obesity. Since fat tissue contains minimal water, TBW estimates can differ substantially between individuals with similar measurements but different fat content, leading to high standard errors.

Water balance abnormalities also affect formula accuracy. These formulas don't properly account for body weight changes from fluid shifts, typically using weight coefficients below 0.5 L/kg. This leads to TBW underestimation in fluid retention and overestimation in dehydration. The Chertow formula, designed for dialysis patients, improves accuracy by including diabetes mellitus and various interaction terms, but still relies on average fluid gains from studied populations.

Determining whether an individual has abnormal TBW remains challenging. Siri's model suggests that fat-free mass (FFM) contains about 73% water under normal conditions, making TBW relative to FFM a potential indicator of water balance. While BIA and DEXA can measure FFM, they assume this 73% ratio applies universally—which isn't always true. More accurate reference methods

like total body potassium (TBK) measurement exist but aren't practical for routine clinical use. The field needs better methods to measure TBW and assess water balance in clinical practice. (43-45)

FLUID BALANCE AND MECHANICAL VENTILATOR:

There exists a complex relationship between mechanical ventilation (MV) and intravenous (IV) fluid management in critically ill patients. These essential interventions can independently and synergistically affect hemodynamics, gas exchange, and organ function. While fluid therapy helps restore hemodynamic stability and organ perfusion, the optimal fluid balance remains debated. Similarly, MV—though lifesaving—can affect cardiovascular function and lead to ventilator-induced lung injury (VILI), particularly depending on ventilator settings.

Heart–lung interactions are fundamental to understanding MV's effects. Positive pressure ventilation raises intrathoracic pressure, reducing venous return and right ventricular preload. This affects cardiac output and organ perfusion, with the right ventricle being especially vulnerable due to its lower contractile reserve. Moreover, transpulmonary and pleural pressures influence capillary filtration pressures, potentially worsening lung edema, particularly in ARDS.

Fluid management approaches range from restrictive to liberal, though these terms lack consistent definitions across studies. Liberal fluid therapy may temporarily boost cardiac output but risks endothelial damage, lung edema, and organ dysfunction through increased hydrostatic pressures and capillary leakage. However, overly restrictive approaches may cause hypoperfusion, acute kidney injury, and slower recovery, especially during early sepsis

treatment. Evidence points to the need for individualized, goal-directed fluid therapy based on patient condition and continuous monitoring.

MV mode and settings significantly affect outcomes. Controlled ventilation with protective strategies (low tidal volume and moderate-to-high PEEP) reduces VILI and improves organ protection. Yet high PEEP can compromise renal perfusion and cardiac output. Assisted ventilation, like pressure support ventilation (PSV), requires less sedation and enables spontaneous breathing but risks patient self-inflicted lung injury (P-SILI) from excessive inspiratory efforts. This can increase lung transvascular filtration pressures and, combined with liberal fluids, lead to alveolar edema and inflammation.

The review highlights that **the interaction between MV and fluid therapy** works both ways and carries clinical significance. Research shows that combining liberal fluids with PSV worsens lung injury, while restrictive fluids with PSV reduce epithelial damage. High PEEP plus liberal fluids can also intensify lung injury. These findings emphasize the importance of coordinating ventilation and fluid strategies.

Despite strong experimental data, **clinical evidence remains limited**, and standard guidelines are lacking. However, current consensus supports an integrated approach that customizes MV and fluid strategies to each patient's condition, balancing risks and benefits. While awaiting further research to clarify these interactions, clinicians should rely on continuous monitoring and personalized care plans to optimize both MV and fluid therapy. (46-49)

FLUID BALANCE AND CRITICALLY ILL PATIENTS

A recent study by J Lee et al (2014) demonstrated that positive fluid balance at ICU discharge correlates with increased 90-day mortality risk, particularly in patients with cardiac or renal dysfunction. The research investigated how fluid

balance during and after critical illness affects outcomes, revealing that excess fluid accumulation leads to worse results.

The findings showed that each additional liter of positive isotonic fluid balance at ICU discharge increased the hazard ratio (HR) for 90-day post-discharge mortality by 1.04. Within-ICU mortality also increased, with an HR of 1.07 per additional liter—indicating that the increased mortality risk wasn't merely due to survivorship bias. Fluid balance remained a significant mortality predictor even after adjusting for demographics, comorbidities, ICU type, illness severity and duration, acute kidney injury (AKI), renal function at discharge, total administered fluids, and peak fluid balance.

At ICU discharge, the mean total fluid balance was 2.6 ± 5.2 liters. Patients in higher fluid balance quartiles exhibited significantly higher unadjusted mortality rates. These patterns were most pronounced in patients with congestive heart failure (CHF), acute kidney injury, and renal insufficiency—groups susceptible to fluid retention and volume overload. For these patients, positive fluid balance strongly predicted poor outcomes, emphasizing the need for personalized fluid management in critically ill patients with fluid-affecting comorbidities.

Importantly, adjusted analyses revealed that positive fluid balance didn't significantly affect outcomes in patients without predisposition to fluid retention or in those who survived beyond 90 days post-discharge. This suggests that fluid overload primarily affects a specific, vulnerable group of ICU patients.

The study found that achieving normal fluid balance (euvolaemia) by ICU discharge improved survival, especially in patients who had experienced the highest peak fluid balances during their stay. This underscores the importance of active fluid management and reduction strategies before discharge to improve long-term outcomes.

In conclusion, the research highlights the crucial role of careful fluid balance monitoring and management in ICU patients, particularly at discharge. Positive fluid balance independently predicted 90-day post-ICU discharge mortality, even after adjusting for multiple factors. The results advocate for careful fluid management, especially in patients with cardiac or renal conditions, and indicate that achieving normal fluid balance before ICU discharge may improve survival after critical illness (50).

Older adults face particular vulnerability to dehydration and electrolyte disturbances due to multiple factors: physical limitations restricting fluid access, iatrogenic causes such as polypharmacy and diuretics, socioeconomic challenges, and cognitive impairments. Incontinence-related embarrassment can lead to intentional fluid restriction. Dehydration frequently causes hospital readmissions, particularly after surgeries like ileostomy or hip fractures, and correlates with increased mortality (51).

Physiologically, aging results in reduced total body water, decreased renal function, and impaired urine concentration ability. These changes increase vulnerability to both dehydration and fluid overload, especially during physiological stress such as surgery or illness. Older adults also experience a blunted thirst response and hormonal changes, including reduced renin and aldosterone levels, and elevated atrial natriuretic peptide (ANP), further compromising fluid balance.

Renal senescence, marked by glomerular loss, reduced glomerular filtration rate (GFR), and diminished tubular function, impairs the kidney's ability to regulate sodium, potassium, and acid-base balance. Age-related hormonal changes disrupt the renin-angiotensin-aldosterone system (RAAS), increasing the risk of hypovolaemia and dysnatraemia. Antidiuretic hormone (ADH) regulation also

changes, with possible nocturnal deficits and reduced renal sensitivity, complicating water conservation during dehydration.

Dysnatraemia, especially hyponatraemia, stands as the most common electrolyte imbalance in the elderly and is linked to increased risks of fractures, osteoporosis, and mortality. Hypernatraemia, though less frequent, carries a higher mortality rate. These abnormalities typically stem from underlying illnesses, medication effects, or inappropriate fluid management. Electrolyte imbalances such as hyperkalaemia and hypomagnesaemia are also common due to impaired renal excretion and dietary factors, potentially leading to serious outcomes like arrhythmias and myocardial infarction.

Environmental stressors, particularly heatwaves, worsen dehydration risks in older adults due to impaired thermoregulation and reduced sweat production. Cognitive impairments, especially dementia, increase the risk, as affected individuals may forget to drink fluids, creating a vicious cycle of dehydration and cognitive decline.

Despite their prevalence and consequences, dehydration and electrolyte abnormalities in older adults often go undiagnosed due to subtle, nonspecific symptoms like dry mouth, dizziness, and apathy, which may be mistaken for normal aging. Accurate diagnosis requires clinical vigilance and appropriate laboratory testing. Poor hydration knowledge among care staff and challenges in fluid monitoring in institutional settings further contribute to missed diagnoses.

Ultimately, while treatable, fluid and electrolyte imbalances in the elderly require a multidisciplinary approach, enhanced caregiver education, and careful monitoring to reduce morbidity, mortality, and healthcare burdens in this growing population.

A systematic review and meta-analysis by Messmer AS et al (2020) examined the relationship between fluid overload (FO), positive cumulative fluid balance (CFB), and mortality in critically ill patients. The findings revealed significant associations between both FO and positive CFB with increased mortality. The adjusted relative risk for mortality was 8.83 for FO and 2.15 for positive CFB after three days in the intensive care unit (ICU). Additionally, mortality risk increased by a factor of 1.19 for each additional liter of positive fluid balance.

Subgroup analyses showed that this association was strongest in critically ill patients with acute kidney injury (AKI), sepsis, respiratory failure, and those who had undergone surgery. These findings highlight the vital importance of fluid management in the ICU setting.

The study demonstrates that FO and positive CFB correlate with increased mortality in critically ill patients, emphasizing the necessity for careful fluid monitoring and management. However, these results should be interpreted cautiously given the limitations in current literature, including varying definitions of fluid overload and inconsistent assessment timepoints. (52)

Another study by Slobod D et al (2019) examined the relationship between volume status and clinical outcomes in mechanically ventilated ICU patients using bioimpedance analysis. The researchers found a significant link between volume overload—measured by the extracellular water to total body water (ECW/TBW) ratio—and extended mechanical ventilation duration. Each 1% increase in the ECW/TBW ratio on day 1 corresponded to a 1.2-fold increase in ventilator days.

Participants required a median of 5 ventilator days. Logistic regression analysis confirmed the relationship between ECW/TBW ratio and ventilation duration, though ICU mortality showed no significant association after adjusting for age, sex, and APACHE II score (OR 1.2, 95% CI: 0.6–2.3, P = 0.75). These results

suggest that unrecognized or unmanaged volume overload may delay recovery and extend mechanical ventilation time.

Technical interference from medical equipment prevented enrollment of 20% of eligible patients, highlighting practical limitations of bioimpedance use in the ICU. Despite this constraint, the study demonstrated that bioimpedance offers an objective, non-invasive method for assessing volume status, which could help personalize fluid management.

The researchers concluded that bioimpedance-based volume assessment could complement traditional clinical evaluation, leading to better outcomes through precise fluid management and reduced ventilator dependence. (53)

in 2011 Flori HR et al, conducted another observational study examined the relationship between positive fluid balance and clinical outcomes in pediatric patients with acute lung injury (ALI). The findings demonstrated that a persistently positive fluid balance was significantly associated with increased mortality and a longer duration of mechanical ventilation. Importantly, these associations remained independent of the severity of the oxygenation defect and the presence of multiple organ system failures.

Multivariate analysis confirmed that increasing fluid balance correlated with higher mortality, and the study found no significant interactions between fluid balance and the presence of cardiovascular or renal failure. These results mirror those observed in adult populations and suggest that excessive fluid accumulation may similarly be harmful in the pediatric ALI context.

The study concludes that positive fluid balance may be deleterious in pediatric ALI patients and underscores the need for further investigation. Specifically, the authors advocate for a large-scale, prospective randomized controlled trial to evaluate the effects of liberal versus conservative fluid management strategies in this vulnerable population (54).

This large observational study by Van Mourik N et al (2019) examined the association between cumulative fluid balance and clinical outcomes in intensive care unit (ICU) patients diagnosed with Acute Respiratory Distress Syndrome (ARDS). The findings revealed that a more positive cumulative fluid balance on day 7 was independently associated with increased 28-day mortality, prolonged mechanical ventilation, and extended ICU stay. Specifically, the adjusted odds ratios for 28-day mortality increased progressively with higher fluid balance categories: 2.1 for Group II, 3.3 for Group III, and 7.9 for Group IV, indicating a non-linear dose-response relationship.

Patients with higher cumulative fluid balances were found to have significantly fewer ventilator-free days at day 28 and a longer ICU length of stay. Additionally, non-survivors demonstrated consistently higher cumulative fluid balances compared to survivors during the first week of ICU admission. The study also noted that continuous venovenous hemofiltration (CVVH) and sepsis were associated with both increased mortality and higher cumulative fluid balances.

The study concludes that a more positive cumulative fluid balance is a strong and independent predictor of adverse outcomes in ARDS patients. These findings underscore the clinical importance of adopting a restrictive fluid management strategy in this patient population to potentially improve survival and reduce the duration of mechanical ventilation and ICU stay.

Another study by Zhou D et al(2025), examined the association between fluid balance and hospital mortality in sepsis patients undergoing mechanical ventilation (MV), with a specific focus on whether ventilation settings—particularly the level of positive end-expiratory pressure (PEEP) and mode of ventilation—modify this relationship. Fluid resuscitation is essential in early sepsis management to improve intravascular volume and perfusion, but

excessive fluid accumulation is known to be associated with poor outcomes. This research aimed to clarify the interaction between fluid balance and mechanical ventilation settings in the early phase of ICU admission.

Data were collected during the first 24 hours of ICU stay, including fluid input, output, arterial blood gas, SOFA score, SAPS II score, and the use of vasopressors or continuous renal replacement therapy (CRRT). Results indicated that non-survivors had significantly higher fluid input and retention during the first 6, 12, 18, and 24 hours. A U-shaped association was observed between cumulative fluid balance and hospital mortality, with both negative and positive balances linked to increased risk of death. Notably, cumulative positive fluid balance was associated with mortality only within the first 18–24 hours, while negative fluid balance was consistently harmful.

Importantly, the study found that the **level of PEEP**, but not the mode of ventilation, significantly modified the relationship between fluid balance and mortality. High PEEP levels exacerbated the detrimental effects of positive fluid balance during the early stages. These findings underscore the importance of considering PEEP levels when planning fluid management strategies in mechanically ventilated sepsis patients.

MATERIALS AND METHODS

Study Design

It is a prospective observational study that was done to determine the relation between cumulative fluid balance and extubation outcomes. The study was done at Shri B M Patil Medical College, Vijayapura, in patients who were admitted to ICCU and CCU with invasive mechanical ventilation. The study ran for about 12 months.

Study Population

Intubated patients admitted to ICCU and CCU of BLDE, Shri B.M Patil Medical College Hospital and Research Centre, Vijayapura from May 2023 to December 2024.

Inclusion Criteria

- All the patients aged older than 18 years.
- Patients intubated for medical cause.
- Invasive mechanical ventilation for >24hours.
- Successful completion of SBT with T piece trial or by CPAP mode.
- Only first extubation (index extubation) will be considered in the study.

Exclusion Criteria

- Already intubated at time of admission.
- Extubated within one day of admission.
- Self extubations.
- Post-Op cases
- Trauma or surgical cases.
- Long duration intubation leading to tracheostomy

Definition Of Terms

Planned extubation – Extubation following spontaneous breathing trial with T piece or by PS/CPAP mode.

Extubation failure – development of any of the following within 72 hours post-extubation;

- Stridor
- Inability to protect airway – (poor cough, excess secretions)
- Hemodynamic instability.
- Worsening of oxygenation.
- Worsening of pH with rise in $PaCO_2$
- Initiation of NIV
- Or reintubated.

Sample Size Calculation

With anticipated Sensitivity and specificity For Extubation failure 60% and 59.5% respectively, considering the prevalence of Extubation failure 24% (ref), at precision of 1% and 95%confidence, the required sample size is 385.

Formula used is—

$$N=\frac{Z^2 P(1-p)}{\Delta^2}$$

N ill be (a+c) if we use sensitivity as p

N= (a+c)/Prevalence

Statistical Analysis

- The data obtained will be entered in a Microsoft Excel sheet, and statistical analysis will be performed using JMP Pro 16 and statistical package for the social sciences (Version 20).
- Results will be presented as Mean (Median) ±SD, inter quartile range, counts and percentages and diagrams.
- For normally distributed continuous variables between two groups will be compared using independent t test for not normally distributed variables Mann Whitney U test will used.
- Categorical variables will be compared using Chi square test.
- ROC Curve analysis will be applied to find optimal input output ratio along with Sensitivity, Specificity.
- $p<0.05$ will be considered statistically significant. All statistical tests will perform two tailed.

Data Collection Method

Baseline data -- Day of admission, age, sex, underlying acute and chronic disorder.

For all extubation following successful SBT the following data were collected:

- Total duration of mechanical ventilation
- Cumulative Fluid Input
- Cumulative Fluid Output
- Net Cumulative Fluid Balance
- Net Cumulative fluid Input to Output ratio
- Use of diuretics
- Use of dialysis
- Application of NIV support or reintubated, for extubation failure group.

Ethical Considerations

The study was conducted in compliance with the ethical standards of the Institutional Ethical Review Board. Written informed consent was obtained from all participants before inclusion. Confidentiality and anonymity of patient data were strictly maintained.

Study Limitations

1. The study was conducted in a single center, which may limit its generalizability.
2. Calculation of fluid output, only considers urine output, as insensible losses cannot be calculated..
3. Impact of cardiovascular and renal comorbidities not extensively studied.

RESULTS

DEMOGRAPHIC AND CLINICAL CHARACTERISTICS OF THE STUDY POPULATION

A total of 370 patients who underwent mechanical ventilation followed by extubation were included in the study. The demographic and clinical profiles of the patients are summarized below.

The majority of the study population were male, comprising 255 patients (68.9%), while females accounted for 115 patients (31.1%). The most common primary diagnosis leading to mechanical ventilation was respiratory disease, observed in 111 patients, followed closely by cardiovascular disease in 104 patients. Other primary disease categories included general or miscellaneous conditions (n = 63), neurological conditions (n = 55), and renal diseases (n = 37).

Regarding comorbidities, 158 patients (42.7%) had no known comorbid conditions. Among those with comorbidities, cardiovascular disease was the most prevalent (n = 60), followed by diabetes mellitus (n = 39) and respiratory comorbidities (n = 31). The remaining patients had neurological, renal, or multiple coexisting comorbid conditions.

Two types of spontaneous breathing trials (SBTs) were employed prior to extubation. The T-piece trial was used in 196 patients, whereas 174 patients underwent SBT using CPAP mode. Diuretic therapy was administered to 74 patients, while 296 did not receive diuretics during the peri-extubation period. Additionally, 45 patients required dialysis during their ICU stay, whereas the majority (n = 325) did not.

The overall extubation success rate was high, with 304 patients (82.2%) successfully extubated. Extubation failure occurred in 66 patients (17.8%). Among those who failed extubation, 39 required reintubation, while 27 were transitioned to non-invasive ventilation (NIV) as an alternative post-extubation support strategy.

COMPARISON OF CLINICAL VARIABLES BETWEEN GROUPS

Independent Samples T-Test

		Statistic	P
Age	**Mann-Whitney U**	9552	0.542
Cumulative Fluid Input (mL)	**Mann-Whitney U**	8830	0.127
Duration of Mechanical Ventilation (Days)	**Mann-Whitney U**	9987	0.955
Cumulative Fluid Balance (mL)	**Mann-Whitney U**	8410	0.040
Cumulative Fluid Output (mL)	**Mann-Whitney U**	8982	0.182
Fluid Input/Output Ratio	**Mann-Whitney U**	8870	0.140

Note. $H_a\ \mu_0 \neq \mu_1$

An independent samples analysis was conducted to compare key demographic and clinical parameters between two groups, likely corresponding to extubation outcomes (success vs. failure), using the Mann-Whitney U test due to non-normal distribution of variables. The null hypothesis (H_0: $\mu_0 = \mu_1$) assumed no difference between the two groups, with the alternative hypothesis (H_a: $\mu_0 \neq \mu_1$) indicating a statistically significant difference.

Age

The comparison of patient age between groups yielded a Mann-Whitney U statistic of 9552 with a p-value of 0.542, indicating no statistically significant difference in age between the groups. This suggests that age did not significantly influence extubation outcomes in this cohort.

Cumulative Fluid Input

The cumulative fluid input showed a U statistic of 8830 and a p-value of 0.127, which was not statistically significant. While fluid input volumes varied between groups, the difference was not sufficient to reject the null hypothesis.

Duration of Mechanical Ventilation

For the duration of mechanical ventilation, the analysis returned a U statistic of 9987 and a p-value of 0.955, indicating no significant difference in the length of mechanical ventilation between the groups. This suggests that the duration of mechanical support did not significantly affect the extubation outcome.

Cumulative Fluid Balance

A statistically significant difference was observed in cumulative fluid balance between the two groups, with a U statistic of 8410 and a p-value of 0.040. This finding suggests that patients with different extubation outcomes had significantly different net fluid balances. Specifically, it implies that fluid balance may be a relevant clinical marker in predicting extubation success or failure, warranting further investigation.

Cumulative Fluid Output

The comparison of cumulative fluid output produced a U statistic of 8982 and a p-value of 0.182, indicating no statistically significant difference. Thus, fluid output alone may not be a key factor differentiating outcomes in this sample.

Fluid Input/Output Ratio

The fluid input/output ratio had a U statistic of 8870 and a p-value of 0.140, which was not statistically significant. Although this metric accounts for the relationship between fluid input and output, it did not differ meaningfully between the groups in this analysis.

Among all variables analyzed, only cumulative fluid balance demonstrated a statistically significant difference between groups. This suggests that net fluid status may play a critical role in extubation outcomes and could serve as a valuable parameter in clinical decision-making. Other variables, including age, mechanical ventilation duration, and fluid management metrics (input, output, and ratio), did not show statistically significant differences between the groups.

COMPARISON OF DEMOGRAPHIC AND CLINICAL CHARACTERISTICS BETWEEN EXTUBATION SUCCESS AND FAILURE GROUPS

Group Descriptives

	Group	N	Mean	SD
Age	**Extubation Failure**	66	46.98	21.222
	Extubation Success	304	48.49	20.317
Cumulative Fluid Input (mL)	**Extubation Failure**	66	12452.11	7504.344
	Extubation Success	304	11802.85	9164.727
Duration of Mechanical Ventilation (Days)	**Extubation Failure**	66	5.74	2.544
	Extubation Success	304	5.76	2.605
Cumulative Fluid Balance (mL)	**Extubation Failure**	66	1613.03	2072.283
	Extubation Success	304	1058.93	1730.252
Cumulative Fluid Output (mL)	**Extubation Failure**	66	10839.08	6534.971
	Extubation Success	304	10743.92	8832.422

Group Descriptives

	Group	N	Mean	SD
Fluid Input/Output Ratio	**Extubation Failure**	66	1.16	0.150
	Extubation Success	304	1.13	0.150

The study population of 370 patients was stratified into two groups based on extubation outcomes: extubation success (n = 304) and extubation failure (n = 66). Group comparisons were performed using descriptive statistics and chi-square tests to evaluate associations across demographic and clinical variables.

Age

The mean age of patients in the extubation failure group was 46.98 years (SD = 21.22), compared to 48.49 years (SD = 20.32) in the extubation success group. The difference in age was not statistically significant, indicating that age was not a differentiating factor between those who succeeded and those who failed extubation.

Cumulative Fluid Input

Patients in the failure group had a slightly higher mean cumulative fluid input (12,452.11 mL, SD = 7504.34) compared to those who had a successful extubation (11,802.85 mL, SD = 9164.73). However, this difference was not statistically significant.

Duration of Mechanical Ventilation

The average duration of mechanical ventilation was comparable between the two groups, with a mean of 5.74 days (SD = 2.54) in the failure group and 5.76 days (SD = 2.61) in the success group. No significant difference was observed.

Cumulative Fluid Balance

Patients who experienced extubation failure had a higher mean cumulative fluid balance (1613.03 mL, SD = 2072.28) than those who were successfully extubated (1058.93 mL, SD = 1730.25). This difference, while not statistically significant in the descriptive analysis, showed significance in earlier inferential testing ($p = 0.040$), suggesting fluid balance may be an important factor in extubation outcomes.

Cumulative Fluid Output

Cumulative fluid output was similar between groups: 10,839.08 mL (SD = 6534.97) in the failure group and 10,743.92 mL (SD = 8832.42) in the success group. No significant difference was found.

Fluid Input/Output Ratio

The fluid input/output ratio was marginally higher in the failure group (mean = 1.16, SD = 0.150) than in the success group (mean = 1.13, SD = 0.150), though this difference was not statistically significant.

CATEGORICAL VARIABLES AND CHI-SQUARE ANALYSES

Gender

Contingency Tables

	GROUP		
Gender	**Extubation Failure**	**Extubation Success**	**Total**
Female	22	93	115
Male	44	211	255
Total	66	304	370

χ^2 Tests

	Value	df	P
χ^2	0.190	1	0.663
N	370		

Among the extubation failure group, 22 were female and 44 were male. In the success group, 93 were female and 211 were male. A chi-square test indicated no significant association between gender and extubation outcome ($\chi^2 = 0.190$, df = 1, p = 0.663).

Primary Disease

Contingency Tables

	GROUP		
Primary Disease	**Extubation Failure**	**Extubation Success**	**Total**
Cardiovascular	16	88	104
General/Other	14	49	63
Neurological	9	46	55
Renal	5	32	37
Respiratory	22	89	111
Total	66	304	370

χ^2 Tests

	Value	**df**	**p**
χ^2	2.10	4	0.717
N	370		

Primary disease distribution did not differ significantly between groups (χ^2 = 2.10, df = 4, p = 0.717). While respiratory disease was the most common diagnosis overall, it was similarly distributed among success and failure groups (22 failures vs. 89 successes). Other conditions like cardiovascular, renal, neurological, and general/other were also proportionally represented.

Type of Spontaneous Breathing Trial (SBT)

Contingency Tables

	GROUP		
Type of Spontaneous Breathing Trial	**Extubation Failure**	**Extubation Success**	**Total**
CPAP mode	29	145	174
T piece trial	37	159	196
Total	66	304	370

χ^2 Tests Chi Square Test

	Value	df	p
χ^2	0.307	1	0.579
N	370		

Of the 66 patients who failed extubation, 29 underwent CPAP and 37 underwent T-piece trials. In the success group, 145 used CPAP and 159 used T-piece. No statistically significant association was observed between type of SBT and extubation outcome ($\chi^2 = 0.307$, df = 1, $p = 0.579$).

Use of Diuretics

Contingency Tables

	GROUP		
Use of Diuretics	**Extubation Failure**	**Extubation Success**	**Total**
No	55	241	296
Yes	11	63	74
Total	66	304	370

χ^2 Tests

	Value	**df**	**p**
χ^2	0.558	1	0.455
N	370		

Diuretic use was recorded in 11 of the patients who failed extubation and in 63 of those who succeeded. The use of diuretics was not significantly associated with extubation outcome ($\chi^2 = 0.558$, df = 1, p = 0.455).

Dialysis

Contingency Tables

	GROUP		
Dialysis	**Extubation Failure**	**Extubation Success**	**Total**
No	56	269	325
Yes	10	35	45
Total	66	304	370

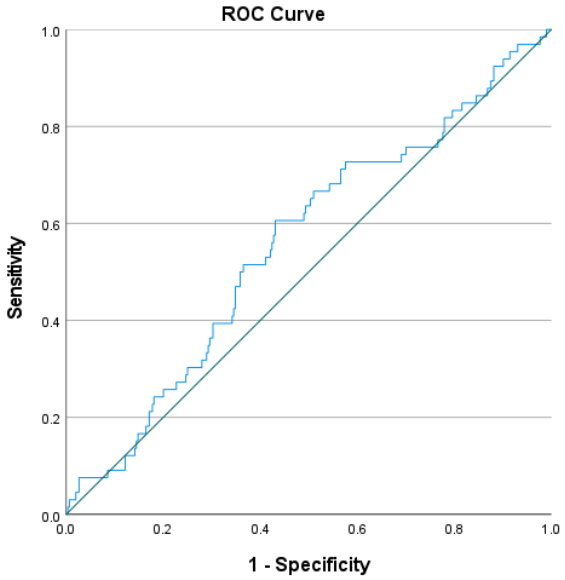
χ^2 Tests

	Value	df	p
χ^2	1.02	2	0.602
N	370		

Dialysis was administered to 10 patients in the failure group and 35 in the success group. The chi-square test revealed no statistically significant relationship between dialysis requirement and extubation outcome ($\chi^2 = 1.02$, df = 2, p = 0.602).

Receiver Operating Characteristic (ROC) Analysis of Fluid Input/Output Ratio

To evaluate the predictive value of the fluid input/output (I/O) ratio in determining extubation outcomes, a Receiver Operating Characteristic (ROC) analysis was conducted. This analysis assesses the discriminative ability of the I/O ratio to correctly classify patients into extubation success or failure groups.



Area Under the Curve (AUC)

The area under the ROC curve (AUC) for the fluid I/O ratio was 0.558 with a standard error of 0.039 and an asymptotic significance (p-value) of 0.140. The 95% confidence interval ranged from 0.482 to 0.634.

While the AUC is slightly above 0.5—suggesting some discriminative power—it is not statistically significant, as indicated by the p-value (> 0.05). This implies that the fluid input/output ratio does not provide a strong or reliable predictive signal for extubation outcomes in this sample population.

Area Under the Curve

Test Result Variable(s): Fluid Input/Output Ratio

Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
.558	.039	.140	.482	.634

a. Under the nonparametric assumption

b. Null hypothesis: true area = 0.5

Cut-off Point Analysis

Cutt off				Sensitivity	Specificity	
1.129084372462370				56.90789	60.60606	

A specific cut-off value of 1.129 was evaluated. At this threshold:

- Sensitivity (true positive rate) was 56.91%
- Specificity (true negative rate) was 60.61%

These values indicate moderate classification performance. In practical terms, using an I/O ratio of 1.129 as a decision threshold would correctly identify approximately 57% of patients who would experience extubation failure and correctly exclude approximately 61% of those who would not. However, these rates are relatively modest and, combined with the non-significant AUC, suggest limited clinical utility of the I/O ratio as a standalone predictor.

In summary, none of the categorical variables—gender, primary disease, type of spontaneous breathing trial, diuretic use, or dialysis—were significantly associated with extubation outcomes. Among the continuous variables, cumulative fluid balance showed a statistically significant difference between

groups in prior inferential analysis and may represent a critical factor influencing extubation success. Other clinical metrics, including age, fluid input/output, and mechanical ventilation duration, were not predictive of extubation failure in this sample.

The ROC analysis of the fluid input/output ratio demonstrated only marginal discriminative ability in predicting extubation outcomes, with an AUC of 0.558 and non-significant p-value (0.140). Although a cut-off value of 1.129 offered moderate sensitivity and specificity, the overall findings do not support the use of the I/O ratio as a sole predictive marker for extubation success or failure in this cohort. Future studies may consider incorporating this parameter into multivariate models alongside other clinical indicators to enhance predictive accuracy.

DISCUSSION

This study aimed to evaluate demographic and clinical predictors associated with extubation outcomes in mechanically ventilated patients, with a particular focus on fluid balance and its components. Extubation failure, a clinically significant complication associated with increased morbidity, mortality, and healthcare costs, was observed in 17.8% of patients in this cohort. Understanding factors that differentiate successful from failed extubation may help clinicians optimize timing and supportive care strategies in the ICU.

Demographic and Clinical Characteristics

The demographic profile of the study population aligns with existing literature, with a male predominance (68.9%) and a wide age range (mean ~48 years). No significant difference in age or gender was observed between the extubation success and failure groups, suggesting that demographic factors alone may not be reliable indicators of extubation readiness.

Primary diagnoses among patients included respiratory, cardiovascular, neurological, renal, and general/other conditions, with respiratory causes being the most common (30%). Importantly, primary disease distribution did not significantly differ between groups, indicating that underlying etiology alone may not sufficiently predict extubation outcomes. This finding supports the notion that extubation failure is likely multifactorial and not determined by diagnosis alone.

Fluid Balance and Mechanical Ventilation Parameters

One of the core objectives of this study was to investigate the role of fluid balance in extubation outcomes. While cumulative fluid input, output, and the input/output (I/O) ratio did not differ significantly between groups, cumulative fluid balance emerged as a statistically significant factor, with patients

experiencing extubation failure exhibiting higher mean fluid balance than those who succeeded (1613.03 mL vs. 1058.93 mL, p = 0.040). This finding aligns with prior research suggesting that positive fluid balance may impair respiratory function, particularly by contributing to pulmonary edema, impaired gas exchange, and respiratory muscle weakness.

Although the mean fluid I/O ratio was marginally higher in the failure group (1.16 vs. 1.13), this difference did not reach statistical significance. ROC analysis for the I/O ratio confirmed its limited discriminative power, with an AUC of 0.558 (p = 0.140), sensitivity of 56.9%, and specificity of 60.6% at a cut-off value of 1.129. These findings indicate that while a higher I/O ratio may reflect subclinical fluid retention, it is not a strong standalone predictor for extubation failure and should be interpreted in the context of cumulative balance and clinical presentation.

The duration of mechanical ventilation was not significantly different between groups, averaging around 5.7 days in both cohorts. This contrasts with some earlier studies that identified prolonged ventilation as a risk factor for extubation failure. The relatively short ventilation duration in both groups may reflect adherence to current weaning protocols and early extubation practices in the study setting.

Other Clinical Parameters

Use of diuretics, dialysis, and type of spontaneous breathing trial (SBT) also showed no significant association with extubation outcomes. While diuretics were more commonly used in the failure group, this did not reach statistical significance, and their effect on fluid status and respiratory recovery warrants further exploration in prospective studies.

Similarly, no significant differences were found between CPAP and T-piece SBT modalities in terms of extubation success. These findings are consistent

with prior randomized controlled trials that have shown comparable outcomes between these SBT types in terms of predicting extubation readiness.

Clinical Implications

The findings of this study emphasize the importance of cumulative fluid balance as a modifiable factor potentially associated with extubation success similar to previous studies (57). Although fluid input/output ratios and other individual fluid parameters did not reach statistical significance, net balance remains a critical integrative measure of patient volume status. Clinicians should consider adopting a conservative fluid management strategy in the peri-extubation period, particularly in patients at high risk for respiratory compromise.

Furthermore, the lack of association between extubation outcomes and other commonly assessed variables (age, gender, duration of ventilation, and SBT type) suggests that multifactorial risk assessment tools may be more appropriate than reliance on single parameters. These tools may include cumulative fluid metrics, in addition to hemodynamic stability, neurological status, respiratory muscle strength, and disease-specific factors.

Limitations

This study has several limitations. First, it was observational and retrospective in nature, which limits causal inference. Second, the sample was derived from a single center, which may affect generalizability. Third, data on some potentially important predictors such as respiratory muscle performance, blood gas parameters, and weaning indices were not included. Lastly, while fluid balance was a focus, variability in fluid management practices, including diuretic timing and fluid resuscitation strategies, was not controlled for and may confound findings.

Recommendations for Future Research

Future studies should aim to validate these findings in larger, multicenter cohorts and incorporate additional variables such as lung ultrasound, biomarkers of cardiac function, and dynamic measures of respiratory performance. Randomized trials exploring fluid management protocols in the context of weaning and extubation could also help delineate causal relationships.

Moreover, development of a multivariate risk prediction model incorporating fluid balance, disease severity, ventilatory parameters, and clinical assessments may aid clinicians in identifying patients at risk for extubation failure with greater accuracy.

CONCLUSION

In conclusion, this study highlights cumulative fluid balance as a significant factor associated with extubation failure in mechanically ventilated patients. Other variables, including fluid input/output ratios, mechanical ventilation duration, and spontaneous breathing trial type, did not show a significant association. These findings support a more nuanced, integrative approach to extubation readiness that includes assessment of fluid status alongside established clinical criteria.

SUMMARY

This dissertation explored the demographic and clinical characteristics associated with extubation outcomes in mechanically ventilated adult patients, with a primary focus on fluid balance and its potential role in predicting extubation success or failure. The investigation was conducted on a cohort of 370 patients, stratified into extubation success (n = 304) and failure (n = 66) groups, with multiple variables assessed using both descriptive and inferential statistics.

The demographic analysis revealed that most patients were male (68.9%) with a mean age of approximately 48 years. No statistically significant differences in age or gender were observed between those who succeeded and those who failed extubation. Likewise, the distribution of primary diagnoses—including respiratory, cardiovascular, renal, neurological, and general/other conditions—was not significantly associated with extubation outcomes.

A central component of this study was the evaluation of fluid balance-related parameters, including cumulative fluid input, cumulative fluid output, fluid input/output (I/O) ratio, and cumulative fluid balance. Among these, cumulative fluid balance was the only variable that significantly differed between the two groups ($p = 0.040$), with patients in the extubation failure group having a notably higher positive fluid balance. This finding supports the hypothesis that fluid overload may impair respiratory function and contribute to unsuccessful weaning from mechanical ventilation.

Other factors, such as duration of mechanical ventilation, use of diuretics, dialysis, and type of spontaneous breathing trial (CPAP vs. T-piece), were not significantly associated with extubation outcomes. These results suggest that

while such clinical practices are important in patient management, they may not independently predict the success of extubation.

A Receiver Operating Characteristic (ROC) analysis was performed to assess the predictive accuracy of the fluid input/output ratio. Although the area under the curve (AUC) was 0.558, indicating some degree of discrimination, the result was not statistically significant ($p = 0.140$), and sensitivity and specificity were moderate at best. These findings reinforce that the I/O ratio lacks sufficient predictive power as a standalone indicator for extubation outcomes.

Overall, this dissertation adds to the growing body of literature indicating that net fluid balance is a potentially modifiable risk factor in extubation planning. While other routine clinical variables did not show significant associations with extubation outcomes in this study, the role of fluid management, particularly in avoiding excessive positive balances, appears to be of clinical relevance.

This study also highlighted several limitations, including its retrospective design, single-center data source, and limited availability of certain physiological and laboratory parameters that may further influence extubation outcomes. Despite these limitations, the findings contribute valuable insights into ICU extubation practices and point to fluid balance as a target for intervention.

Future research should aim to validate these results in larger, prospective, and multicentric settings. Additionally, integrating fluid metrics into comprehensive predictive models alongside clinical, respiratory, and neurological indicators may improve extubation decision-making and patient outcomes in critical care settings.

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ANNEXURE – I

ETHICAL CLEARANCE CERTIFICATE

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 915/2023-24 10/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "ANALYSING ASSOCIATION BETWEEN FLUID BALANCE AND EXTUBATION SUCCESS".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.VIVEK MATHEW EAPEN

NAME OF THE GUIDE: DR.B.P.KATTIMANI, ASSOCIATE PROFESSOR, DEPT. OF EMERGENCY MEDICINE AND CRITICAL CARE.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA
Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA
MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103, Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

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ANNEXURE II

INFORMED CONSENT FORM

BLDEDU'S SHRI B. M. PATIL
MEDICAL COLLEGE HOSPITAL AND
RESEARCHCENTRE,

VIJAYAPURA- 586103

TITLE OF THE PROJECT –

"**ANALYSING ASSOCIATION BETWEEN FLUID BALANCE AND EXTUBATION SUCCESS**."

PRINCIPAL INVESTIGATOR - DR VIVEK MATHEW EAPEN

P.G. GUIDE NAME - DR. B. P. KATTIMANI

CHAIRMAN ETHICAL COMMITTEE

All aspects of this consent form are explained to the patient in the language understood by him/her.

1) PURPOSE OF RESEARCH:

I have been informed about this study. I have also been given a free choice of participation inthis study.

2) PROCEDURE:

I am aware that in addition to routine care received, I will be asked a series of questions bythe investigator. I have been asked to undergo the necessary investigations and treatment, which will help the investigator in this study.

3) RISK AND DISCOMFORTS:

It is understandable that I may experience some pain and discomfort during the examination orduring my treatment. This is mainly the result of my condition, and the procedure of this studyis not expected to exaggerate these feelings that are associated with the usual course of treatment.

4) BENEFITS:

I understand that participation in this study will help patients' survival and better outcome.

5) CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of Hospital records and will be subject to confidentiality and privacy regulation. Information of a sensitive personal nature will not be a part of the medical records but will be stored in the investigator's research file and identified only by a code number. The code-key connecting name to numbers will be kept in a separate location. If the data are used for publication in the medical literature orfor teaching purposes, no name will be used, and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand that I may see the photographs and videotapes and hear the audiotapes before giving this permission.

6) REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time. Dr VIVEK MATHEW EAPEN is available to answer my questions or concerns. I understand that it will be informed me about any significant new findings discovered during the course of the study, which might influence my continued participation. If, during the study or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am awarethat the social worker of the hospital is available to talk with me. A copy of this consent form will be given to me to keep for careful reading.

7)REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand about the voluntary participation and that I may refuse to participate or may, withdraw consent or discontinue participation in the study at any time without prejudice to my present or future care at this hospital. I also understand that Dr VIVEK MATHEW EAPEN may terminate my participation in the study after he has explained the reasons for doing so and has helped arrangefor my continued care by my own physician or physical therapist if this is appropriate.

8)INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation inthis study if such injury were reported promptly, the appropriate treatment would be available to me, but no further compensation would be provided. I understand that by my agreement to participate in this study, I am not waiving any of my legal rights. I have explained in detail the purpose of the research, the procedures required and the possible risks and benefits to the best ofmy ability in the patient's own language

_____ _____

DR. VIVEK MATHEW EAPEN

(INVESTIGATOR)

DATE:

II) STUDY SUBJECT CONSENT STATEMENT

I confirm that DR. VIVEK MATHEW EAPEN has explained to me the purpose of the research, thestudy procedures that I will undergo, and the possible risks and discomforts as well as benefits that I may experience in my own language. I have read the form and understand this consent.

Therefore, I agree to give consent to participate as a subject in this research project.

_____

_____

Participant / Guardian Date:

_____

Witness signature

ANNEXURE III

B.L.D.E (DEEMED TO BE UNIVERSITY)

SHRI B M PATIL MEDICAL COLLEGE,

VIJAYAPURA, KARNATAKA

SCHEME OF CASE TAKING

INFORMANT:

Name: Case number:

Age: IP number:

Gender: DOA:

Contact number: DOI:

Duration of mechanical ventilation: DOE:

Primary Disease:

Commorbidities:

SBT --- T piece trial / CPAP mode.

POST- EXTUBATION:

Cumulative Fluid Input:

Cumulative Fluid Output:

Cumulative Fluid Balance:

Cumulative Fluid Input to output Ratio:

Diuretics --- USED / NOT USED

Dialysis – Yes/No

If, extubation Failure: Reintubated or kept on NIV

CURRICULUM-VITAE

NAME DR.BABU KATTIMANI

DESIGNATION PROFESSOR

DEPARTMENT OF EMERGENCY MEDICINE

CONTACT: 9342594480

EDUCATION M.S.GENERAL SURGERY,

PRESENT DESIGNATION: PROFESSOR,

DEPARTMENT OF EMERGENCY MEDICINE

SHRI B M PATIL MEDICAL COLLEGE AND

RESEARCH CENTER

VIJAYAPURA, KARNATAKA

BIO-DATA

INVESTIGATOR NAME: DR VIVEK MATHEW EAPEN

QUALIFICATION: M.B.B.S

KARNATAKA MEDICAL

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Mir- "The Greens",
Ettamkallu, Karakulam P.O
Thiruvananthapuram
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PHONE NUMBER: **8891179604**

ANNEXURE III

MASTER CHART

SL. NO:	Age	Gender	Primary Disease	Comorbidity	Duration of Mechanical Ventilation (Days)	Type of Spontaneous Breathing Trial	Cumulative Fluid Input (mL)	Cumulative Fluid Output (mL)	Cumulative Fluid Balance (mL)	Fluid Input/Output Ratio	Use of Diuretics	Dialysis	Extubation Outcome	If Extubation Failure, Outcome
1	75	Female	Cardiovascular	Renal	4	T piece trial	5017	3965	1052	1.265322	No	No	Success	N/A
2	68	Female	General/Other	None	2	CPAP mode	6228	6772	-544	0.919669	No	No	Success	N/A
3	37	Male	General/Other	None	9	CPAP mode	11542	9392	2150	1.228918	No	No	Success	N/A
4	83	Male	Renal	Cardiovascular	4	T piece trial	5434	5700	-266	0.953333	Yes	No	Success	N/A
5	35	Male	Respiratory	None	9	CPAP mode	10817	9676	1141	1.117921	No	No	Success	N/A
6	41	Male	Cardiovascular	Respiratory	9	CPAP mode	9618	9300	318	1.034194	No	No	Success	N/A
7	61	Female	General/Other	None	6	T piece trial	7015	6237	778	1.124739	No	No	Success	N/A
8	42	Male	Respiratory	Diabetes Mellitus, Respiratory	2	CPAP mode	9560	7830	1730	1.220945	No	No	Failure	Reintubated
9	62	Male	Respiratory	Renal, Diabetes Mellitus	8	CPAP mode	7438	5415	2023	1.373592	No	No	Success	N/A
10	19	Female	Renal	None	6	T piece trial	8664	7128	1536	1.215488	No	No	Failure	NIV
11	19	Female	Neurological	Diabetes Mellitus	9	CPAP mode	26043	23141	2902	1.125405	No	Yes	Success	N/A
12	68	Female	Respiratory	Neurological	10	T piece trial	14033	12043	1990	1.165241	No	No	Failure	NIV
13	49	Male	General/Other	Diabetes Mellitus	8	CPAP mode	31367	22189	9178	1.413628	No	No	Success	N/A
14	78	Male	General/Other	Cardiovascular, Respiratory	8	CPAP mode	7781	6238	1543	1.247355	Yes	No	Success	N/A
15	73	Male	General/Other	Diabetes Mellitus	2	T piece trial	3469	3220	249	1.077329	No	No	Success	N/A
16	24	Female	Respiratory	None	5	T piece trial	18420	16004	2416	1.150962	Yes	No	Success	N/A
17	19	Male	Respiratory	None	3	CPAP mode	6182	6493	-311	0.952102	No	No	Success	N/A
18	32	Male	Cardiovascular	None	3	T piece trial	2461	2056	405	1.196984	Yes	No	Success	N/A
19	83	Female	Neurological	None	3	T piece trial	8007	6963	1044	1.149935	No	No	Success	N/A
20	42	Male	Renal	Cardiovascular, Diabetes Mellitus	5	CPAP mode	4081	4418	-337	0.923721	No	No	Success	N/A
21	80	Female	Cardiovascular	Respiratory	2	CPAP mode	2050	2050	0	1	No	No	Success	N/A
22	18	Female	Cardiovascular	Cardiovascular	5	CPAP mode	4691	4380	311	1.071005	No	No	Success	N/A
23	36	Male	Renal	None	3	T piece trial	4553	4401	152	1.034538	No	No	Success	N/A
24	46	Female	Respiratory	Respiratory	6	T piece trial	25041	21947	3094	1.140976	No	Yes	Failure	NIV
25	31	Male	Neurological	None	4	T piece trial	10727	7791	2936	1.376845	Yes	No	Success	N/A
26	85	Female	General/Other	None	9	T piece trial	33554	23699	9855	1.41584	No	No	Failure	NIV
27	77	Male	Cardiovascular	Neurological, Diabetes Mellitus	5	CPAP mode	6993	6553	440	1.067145	Yes	No	Success	N/A
28	47	Male	Respiratory	Diabetes Mellitus	2	T piece trial	8597	7758	839	1.108146	No	No	Failure	NIV
29	78	Male	Respiratory	None	2	CPAP mode	7898	6456	1442	1.223358	No	No	Failure	Reintubated
30	21	Female	Cardiovascular	Diabetes Mellitus, Cardiovascular	4	T piece trial	3648	3304	344	1.104116	Yes	No	Failure	Reintubated
31	64	Female	General/Other	None	10	CPAP mode	30095	30675	-580	0.981092	No	No	Failure	Reintubated
32	25	Female	Renal	Diabetes Mellitus	8	CPAP mode	12614	11215	1399	1.124744	No	No	Success	N/A
33	29	Male	General/Other	Diabetes Mellitus	3	CPAP mode	10237	8986	1251	1.139217	No	Yes	Failure	Reintubated
34	56	Female	Respiratory	None	10	T piece trial	31025	26858	4167	1.155149	No	No	Success	N/A
35	64	Male	General/Other	Diabetes Mellitus	3	CPAP mode	3862	2807	1055	1.375846	No	No	Success	N/A
36	24	Female	Respiratory	None	4	CPAP mode	18043	18215	-172	0.990557	No	No	Failure	NIV
37	19	Female	Cardiovascular	None	3	T piece trial	4997	4979	18	1.003615	No	No	Success	N/A
38	40	Female	Respiratory	None	4	CPAP mode	14725	15858	-1133	0.928553	No	No	Success	N/A
39	28	Female	Neurological	None	7	T piece trial	32342	34724	-2382	0.931402	No	No	Success	N/A
40	26	Female	Cardiovascular	Diabetes Mellitus	10	T piece trial	14635	14089	546	1.038754	No	Yes	Success	N/A
41	64	Female	General/Other	Neurological, Diabetes Mellitus	10	CPAP mode	42393	41557	836	1.020117	No	No	Success	N/A
42	49	Female	Respiratory	Cardiovascular	8	CPAP mode	7550	8095	-545	0.932674	Yes	No	Success	N/A
43	26	Female	Neurological	Cardiovascular	4	T piece trial	3803	3906	-103	0.97363	Yes	No	Success	N/A
44	54	Male	Respiratory	None	5	CPAP mode	13866	10084	3782	1.37505	No	No	Success	N/A
45	62	Female	Cardiovascular	None	7	T piece trial	9330	9824	-494	0.949715	No	No	Failure	NIV
46	70	Male	General/Other	Cardiovascular	6	T piece trial	8809	9495	-686	0.927751	No	No	Success	N/A
47	78	Male	General/Other	Neurological, Diabetes Mellitus	3	T piece trial	11764	11672	92	1.007882	No	No	Success	N/A
48	70	Female	Respiratory	Respiratory	7	CPAP mode	9902	10772	-870	0.919235	No	No	Failure	Reintubated
49	27	Male	Respiratory	Diabetes Mellitus	4	T piece trial	9792	8226	1566	1.190372	No	No	Failure	NIV
50	53	Male	Respiratory	None	9	CPAP mode	15997	11421	4576	1.400665	No	No	Success	N/A
51	59	Male	Cardiovascular	Respiratory	3	CPAP mode	3227	2995	232	1.077462	No	No	Success	N/A
52	56	Male	General/Other	Cardiovascular	2	CPAP mode	2828	2214	614	1.277326	Yes	No	Success	N/A
53	23	Male	Cardiovascular	None	10	CPAP mode	11741	12496	-755	0.939581	No	No	Failure	NIV
54	24	Male	Neurological	None	4	T piece trial	13161	9923	3238	1.326313	No	No	Success	N/A
55	19	Male	Respiratory	Diabetes Mellitus	2	CPAP mode	3661	3005	656	1.218303	No	No	Success	N/A
56	22	Male	Cardiovascular	None	8	T piece trial	10155	8996	1159	1.128835	No	No	Success	N/A
57	30	Male	Renal	None	6	T piece trial	5027	4140	887	1.214251	Yes	Yes	Success	N/A
58	66	Male	Cardiovascular	Neurological, Renal	6	CPAP mode	7798	8535	-737	0.91365	No	No	Success	N/A
59	50	Male	Respiratory	Cardiovascular	2	T piece trial	2523	2001	522	1.26087	No	No	Success	N/A
60	82	Female	Respiratory	None	10	T piece trial	19561	18938	623	1.032897	No	No	Success	N/A
61	66	Male	General/Other	Cardiovascular	8	CPAP mode	12617	9579	3038	1.317152	Yes	Yes	Failure	Reintubated
62	36	Male	Renal	Cardiovascular	9	T piece trial	12410	12673	-263	0.979247	Yes	No	Success	N/A
63	28	Female	Cardiovascular	Cardiovascular	10	T piece trial	16124	14837	1287	1.086743	No	No	Failure	Reintubated
64	52	Male	Respiratory	None	7	T piece trial	26309	24816	1493	1.060163	No	No	Success	N/A
65	57	Male	Cardiovascular	None	6	T piece trial	5676	4482	1194	1.266399	No	Yes	Success	N/A
66	21	Female	General/Other	None	7	CPAP mode	5650	5529	121	1.021885	No	Yes	Failure	Reintubated
67	32	Male	Neurological	Cardiovascular	3	T piece trial	4354	3755	599	1.159521	Yes	No	Success	N/A
68	69	Male	Neurological	Respiratory, Renal	5	T piece trial	6800	5544	1256	1.226551	No	No	Success	N/A
69	21	Male	Respiratory	Cardiovascular	4	T piece trial	5233	3799	1434	1.377468	No	No	Success	N/A
70	69	Male	Respiratory	None	8	T piece trial	8722	8528	194	1.022749	No	No	Success	N/A
71	29	Male	Renal	Diabetes Mellitus	10	CPAP mode	12200	10674	1526	1.142964	Yes	No	Success	N/A
72	19	Male	Cardiovascular	None	6	T piece trial	7335	6911	424	1.061351	No	No	Success	N/A
73	79	Male	Respiratory	Neurological, Cardiovascular	7	CPAP mode	11551	9307	2244	1.241109	No	No	Success	N/A
74	35	Male	Respiratory	None	3	T piece trial	3718	4088	-370	0.909491	No	No	Success	N/A
75	22	Male	Neurological	None	4	CPAP mode	15906	16563	-657	0.960333	No	No	Success	N/A
76	25	Male	Respiratory	Cardiovascular	4	CPAP mode	5513	5636	-123	0.978176	No	No	Failure	Reintubated
77	58	Male	Neurological	None	10	CPAP mode	32830	32477	353	1.010869	No	No	Success	N/A
78	35	Female	Respiratory	None	8	CPAP mode	12719	13762	-1043	0.924212	No	No	Success	N/A
79	31	Male	General/Other	None	9	CPAP mode	13694	10286	3408	1.331324	No	No	Success	N/A
80	29	Male	Respiratory	None	10	T piece trial	36218	33470	2748	1.082103	No	No	Success	N/A
81	43	Male	Neurological	Respiratory	7	T piece trial	25410	20776	4634	1.223046	No	No	Success	N/A
82	51	Female	Cardiovascular	Respiratory	5	T piece trial	4846	3543	1303	1.367767	No	No	Success	N/A
83	53	Male	Cardiovascular	Respiratory	2	CPAP mode	2428	2000	428	1.214	Yes	No	Success	N/A
84	25	Male	Renal	Cardiovascular	9	T piece trial	8701	7391	1310	1.177243	No	No	Success	N/A
85	51	Male	Respiratory	None	8	CPAP mode	19892	17646	2246	1.127281	Yes	No	Success	N/A
86	24	Male	Cardiovascular	None	3	CPAP mode	4223	3987	236	1.059192	No	No	Success	N/A
87	68	Male	Respiratory	Cardiovascular, Diabetes Mellitus	9	T piece trial	15936	11961	3975	1.33233	Yes	No	Success	N/A
88	70	Male	Neurological	Cardiovascular	6	T piece trial	5112	3812	1300	1.341028	Yes	No	Failure	Reintubated
89	29	Female	Cardiovascular	None	8	CPAP mode	6595	5176	1419	1.27415	No	No	Success	N/A
90	58	Male	Neurological	Diabetes Mellitus	5	CPAP mode	20375	15587	4788	1.307179	No	No	Failure	Reintubated
91	66	Male	General/Other	Neurological, Renal	6	T piece trial	5341	3775	1566	1.414834	No	No	Success	N/A
92	27	Female	Cardiovascular	None	9	T piece trial	11189	8991	2198	1.244467	No	No	Success	N/A
93	68	Male	Renal	Neurological, Renal	2	T piece trial	3376	3184	192	1.060302	Yes	No	Success	N/A
94	37	Male	Respiratory	None	2	T piece trial	4642	3669	973	1.265195	Yes	Yes	Success	N/A
95	71	Male	Neurological	Respiratory	5	CPAP mode	13358	10592	2766	1.26114	No	No	Success	N/A
96	66	Male	Respiratory	None	4	T piece trial	12669	9215	3454	1.374824	No	No	Success	N/A
97	64	Male	Neurological	Respiratory, Diabetes Mellitus	3	T piece trial	13019	10944	2075	1.189602	No	No	Failure	Reintubated
98	69	Male	Respiratory	Cardiovascular, Neurological	4	T piece trial	3444	3512	-68	0.980638	No	No	Success	N/A
99	49	Male	Cardiovascular	Respiratory	2	CPAP mode	3288	3048	240	1.07874	Yes	Yes	Success	N/A

100	68	Male	Cardiovascular	Cardiovascular	2	CPAP mode	1721	1476	245	1.165989	No	No	Success	N/A
101	29	Male	Cardiovascular	None	7	CPAP mode	8704	9324	-620	0.933505	No	No	Success	N/A
102	73	Male	Cardiovascular	None	6	CPAP mode	8452	7817	635	1.081233	No	No	Success	N/A
103	67	Male	Cardiovascular	Renal, Cardiovascular	9	T piece trial	7780	7801	-21	0.997308	No	No	Failure	NIV
104	81	Male	Respiratory	Renal	3	CPAP mode	3545	3878	-333	0.914131	Yes	No	Success	N/A
105	21	Male	Respiratory	Cardiovascular	5	T piece trial	8863	9227	-364	0.960551	Yes	No	Success	N/A
106	52	Male	Cardiovascular	Cardiovascular	9	T piece trial	8197	8517	-320	0.962428	Yes	No	Success	N/A
107	67	Male	Renal	None	7	T piece trial	7295	6930	365	1.05267	No	No	Success	N/A
108	21	Male	General/Other	None	2	T piece trial	7850	7986	-136	0.98297	No	No	Success	N/A
109	48	Female	General/Other	None	5	CPAP mode	22760	23211	-451	0.98057	No	No	Success	N/A
110	40	Female	Renal	None	2	CPAP mode	3308	3543	-235	0.933672	No	No	Success	N/A
111	81	Female	Neurological	None	3	CPAP mode	8811	6571	2240	1.340892	No	No	Success	N/A
112	26	Male	Neurological	None	6	T piece trial	21952	17087	4865	1.284719	No	No	Success	N/A
113	73	Male	Respiratory	Respiratory	9	T piece trial	17512	19254	-1742	0.909525	No	No	Success	N/A
114	68	Male	Neurological	Respiratory, Renal	9	T piece trial	10805	10388	417	1.040142	Yes	Yes	Success	N/A
115	77	Male	Respiratory	Cardiovascular, Diabetes Mellitus	4	T piece trial	5451	3929	1522	1.387376	No	No	Success	N/A
116	73	Female	General/Other	Renal, Cardiovascular	5	T piece trial	4839	4531	308	1.067976	No	No	Success	N/A
117	84	Male	Neurological	None	7	CPAP mode	19172	19192	-20	0.998958	No	No	Success	N/A
118	84	Male	Respiratory	None	4	T piece trial	7651	5419	2232	1.411884	No	No	Success	N/A
119	29	Female	Cardiovascular	None	3	CPAP mode	2686	2704	-18	0.993343	No	No	Failure	Reintubated
120	62	Male	Cardiovascular	Cardiovascular, Respiratory	7	T piece trial	10174	9407	767	1.081535	No	No	Success	N/A
121	83	Male	Cardiovascular	Neurological, Cardiovascular	6	T piece trial	8854	8546	308	1.03604	No	No	Success	N/A
122	31	Male	Cardiovascular	None	6	CPAP mode	6503	5967	536	1.089827	Yes	No	Success	N/A
123	33	Male	Respiratory	None	6	CPAP mode	21374	15929	5445	1.341829	No	No	Success	N/A
124	68	Male	Respiratory	None	6	T piece trial	25234	18031	7203	1.399479	No	No	Failure	Reintubated
125	46	Female	Respiratory	None	4	T piece trial	5428	4031	1397	1.346564	No	Yes	Success	N/A
126	35	Female	Cardiovascular	None	8	T piece trial	12085	10701	1384	1.129334	No	No	Failure	Reintubated
127	27	Female	Cardiovascular	Cardiovascular, Diabetes Mellitus	7	T piece trial	9448	7519	1929	1.25655	No	No	Success	N/A
128	21	Female	Cardiovascular	Cardiovascular	5	CPAP mode	8838	9269	-431	0.953501	No	No	Success	N/A
129	81	Male	Cardiovascular	Neurological, Diabetes Mellitus	3	T piece trial	3018	3032	-14	0.995383	No	No	Success	N/A
130	29	Male	Respiratory	None	10	CPAP mode	28804	29042	-238	0.991805	No	No	Success	N/A
131	24	Female	Respiratory	None	2	T piece trial	4426	3274	1152	1.351863	No	No	Success	N/A
132	62	Male	Cardiovascular	None	5	CPAP mode	4874	3627	1247	1.34381	No	Yes	Success	N/A
133	53	Female	Neurological	None	2	CPAP mode	6221	5388	833	1.154603	No	No	Success	N/A
134	22	Female	Respiratory	Diabetes Mellitus	7	T piece trial	21097	16236	4861	1.299396	No	No	Success	N/A
135	57	Male	Respiratory	Diabetes Mellitus	5	CPAP mode	13240	13993	-753	0.946187	No	No	Success	N/A
136	37	Male	Respiratory	Cardiovascular	6	T piece trial	7360	5315	2045	1.38476	Yes	Yes	Success	N/A
137	61	Male	Renal	None	6	CPAP mode	6773	6841	-68	0.99006	No	No	Success	N/A
138	29	Male	Respiratory	None	10	T piece trial	14362	12318	2044	1.165936	No	No	Success	N/A
139	55	Male	Cardiovascular	Cardiovascular	2	CPAP mode	1736	1574	162	1.102922	No	No	Success	N/A
140	77	Female	Neurological	Neurological	3	T piece trial	8644	8482	162	1.019099	No	Yes	Success	N/A
141	23	Male	General/Other	None	7	T piece trial	32341	26789	5552	1.207249	No	No	Success	N/A
142	59	Male	General/Other	Diabetes Mellitus, Respiratory	6	CPAP mode	18253	16990	1263	1.074338	No	No	Success	N/A
143	83	Female	Cardiovascular	Respiratory	8	CPAP mode	9782	9794	-12	0.998775	No	No	Success	N/A
144	77	Female	General/Other	None	4	CPAP mode	16957	12666	4291	1.338781	No	No	Failure	NIV
145	34	Male	Respiratory	Cardiovascular	4	CPAP mode	3766	3415	351	1.102782	Yes	No	Success	N/A
146	64	Male	Cardiovascular	Cardiovascular, Renal	3	T piece trial	4931	4466	465	1.10412	Yes	No	Success	N/A
147	36	Male	Neurological	Diabetes Mellitus	4	CPAP mode	17710	15247	2463	1.16154	Yes	No	Success	N/A
148	45	Male	Renal	None	2	T piece trial	2490	2587	-97	0.962505	No	No	Success	N/A
149	64	Female	Cardiovascular	Cardiovascular, Renal	6	CPAP mode	7165	6453	712	1.110336	Yes	No	Success	N/A
150	41	Male	Renal	None	6	T piece trial	6259	6274	-15	0.997609	No	No	Success	N/A
151	75	Male	Respiratory	Respiratory, Neurological	8	T piece trial	16652	14962	1690	1.112953	No	No	Failure	NIV
152	30	Male	Neurological	Diabetes Mellitus	5	T piece trial	23507	19356	4151	1.214455	No	No	Failure	Reintubated
153	66	Male	Cardiovascular	Renal	4	T piece trial	6513	5266	1247	1.236802	No	No	Success	N/A
154	80	Female	Neurological	Cardiovascular	2	CPAP mode	1995	1475	520	1.352542	No	No	Success	N/A
155	25	Male	Respiratory	Cardiovascular	9	CPAP mode	12572	12518	54	1.004314	Yes	Yes	Success	N/A
156	55	Male	Cardiovascular	None	4	T piece trial	6930	4872	2058	1.422414	No	No	Success	N/A
157	80	Male	General/Other	Neurological	3	CPAP mode	9572	8200	1372	1.167317	No	No	Failure	NIV
158	23	Male	General/Other	Diabetes Mellitus	5	CPAP mode	20295	15048	5247	1.348684	No	No	Success	N/A
159	40	Female	Respiratory	Cardiovascular	4	T piece trial	3337	3451	-114	0.966966	No	No	Success	N/A
160	49	Female	Cardiovascular	None	2	CPAP mode	3437	2596	841	1.32396	No	No	Success	N/A
161	51	Male	Neurological	None	4	CPAP mode	18328	17943	385	1.021457	No	No	Success	N/A
162	30	Male	Respiratory	None	3	CPAP mode	14051	15057	-1006	0.933187	No	No	Success	N/A
163	62	Male	Cardiovascular	Diabetes Mellitus, Neurological	9	T piece trial	9305	6908	2397	1.346989	No	No	Success	N/A
164	61	Male	Respiratory	Cardiovascular	7	CPAP mode	11620	12459	-839	0.932659	Yes	No	Success	N/A
165	44	Male	Renal	None	10	T piece trial	11915	9213	2702	1.293281	No	No	Success	N/A
166	81	Female	Renal	Neurological, Diabetes Mellitus	9	CPAP mode	12709	11517	1192	1.103499	No	No	Success	N/A
167	48	Female	Respiratory	Diabetes Mellitus	4	CPAP mode	14204	14277	-73	0.994887	No	No	Success	N/A
168	20	Male	General/Other	Cardiovascular	6	CPAP mode	5415	3879	1536	1.395978	No	No	Failure	Reintubated
169	22	Male	Cardiovascular	Diabetes Mellitus	8	T piece trial	11993	10674	1319	1.123571	No	No	Success	N/A
170	29	Male	Cardiovascular	None	6	T piece trial	10729	7966	2763	1.346849	No	No	Failure	Reintubated
171	37	Male	Neurological	None	9	CPAP mode	27521	29736	-2215	0.925511	No	No	Success	N/A
172	53	Female	General/Other	Respiratory	6	T piece trial	25292	26900	-1608	0.940223	No	No	Failure	NIV
173	33	Male	Cardiovascular	Cardiovascular	2	CPAP mode	2758	2368	390	1.164696	Yes	No	Success	N/A
174	47	Male	Cardiovascular	Respiratory	6	T piece trial	6482	5935	547	1.092165	No	Yes	Success	N/A
175	31	Female	Cardiovascular	None	5	T piece trial	7620	5678	1942	1.342022	No	No	Success	N/A
176	76	Male	Renal	Renal	7	CPAP mode	9778	9555	223	1.023339	No	Yes	Success	N/A
177	70	Male	Cardiovascular	Cardiovascular	4	CPAP mode	6567	5252	1315	1.250381	No	No	Success	N/A
178	20	Female	Respiratory	None	6	CPAP mode	16805	18036	-1231	0.931748	No	No	Success	N/A
179	26	Female	Respiratory	None	3	CPAP mode	12219	11015	1204	1.109305	No	No	Success	N/A
180	23	Male	Respiratory	None	7	T piece trial	17654	13473	4181	1.310324	No	No	Failure	Reintubated
181	74	Female	Respiratory	Cardiovascular	7	T piece trial	6248	4998	1250	1.2501	Yes	No	Failure	Reintubated
182	35	Male	Renal	None	6	T piece trial	6647	4935	1712	1.34691	No	No	Failure	Reintubated
183	29	Male	General/Other	None	7	CPAP mode	22585	19803	2782	1.140484	No	No	Failure	NIV
184	20	Male	Cardiovascular	None	8	CPAP mode	12546	10460	2086	1.199426	No	No	Success	N/A
185	25	Male	Neurological	None	6	T piece trial	15395	10998	4397	1.3998	No	No	Success	N/A
186	61	Male	Cardiovascular	Renal, Neurological	2	CPAP mode	1914	2082	-168	0.919308	Yes	No	Success	N/A
187	58	Male	Renal	Cardiovascular	9	T piece trial	7688	5843	1845	1.315762	No	Yes	Success	N/A
188	84	Male	Cardiovascular	None	5	T piece trial	5386	5338	48	1.008992	No	Yes	Success	N/A
189	54	Female	General/Other	None	10	T piece trial	27510	20993	6517	1.310437	No	No	Success	N/A
190	53	Female	Cardiovascular	Diabetes Mellitus	7	T piece trial	9886	7966	1920	1.241024	No	No	Success	N/A
191	48	Female	Respiratory	None	2	T piece trial	4105	4166	-61	0.985358	No	No	Success	N/A
192	33	Male	Neurological	None	4	CPAP mode	11649	8818	2831	1.321048	No	No	Failure	NIV
193	57	Female	General/Other	None	4	CPAP mode	13690	10939	2751	1.251486	No	No	Success	N/A
194	66	Male	Neurological	Renal, Neurological	5	CPAP mode	7185	6043	1142	1.188979	Yes	Yes	Failure	Reintubated
195	44	Male	Cardiovascular	Cardiovascular	7	T piece trial	8451	8047	404	1.050205	No	No	Success	N/A
196	21	Male	Cardiovascular	None	8	T piece trial	10179	9793	386	1.039416	No	No	Success	N/A
197	78	Male	Respiratory	Cardiovascular	5	T piece trial	8116	6735	1381	1.205048	No	No	Success	N/A
198	49	Female	Respiratory	Diabetes Mellitus	7	T piece trial	10794	9319	1475	1.158279	Yes	Ye	Success	N/A
199	21	Male	General/Other	None	2	CPAP mode	7291	7390	-99	0.986604	No	No	Success	N/A

200	27	Male	Neurological	None	9	CPAP mode	37653	32366	5287	1.16335	No	No	Success	N/A
201	19	Female	Cardiovascular	None	8	T piece trial	9237	6827	2410	1.35301	No	No	Success	N/A
202	61	Male	Cardiovascular	Neurological, Renal	10	T piece trial	11362	10822	540	1.049898	No	No	Success	N/A
203	36	Male	Neurological	Cardiovascular	4	T piece trial	3058	2807	251	1.089419	No	Yes	Success	N/A
204	44	Male	Respiratory	None	3	CPAP mode	5507	5734	-227	0.960412	No	No	Failure	Reintubated
205	63	Male	Renal	Neurological, Diabetes Mellitus	2	CPAP mode	3125	2285	840	1.367615	No	No	Success	N/A
206	34	Male	General/Other	Diabetes Mellitus	4	CPAP mode	14938	14526	412	1.028363	No	No	Success	N/A
207	71	Female	Cardiovascular	Neurological	6	T piece trial	7471	8115	-644	0.920641	No	No	Success	N/A
208	55	Male	Renal	None	3	T piece trial	3777	3064	713	1.232702	No	No	Success	N/A
209	30	Male	Neurological	None	4	CPAP mode	18314	18418	-104	0.994353	No	No	Success	N/A
210	57	Female	Cardiovascular	Respiratory	5	T piece trial	7469	5289	2180	1.412176	No	No	Success	N/A
211	28	Male	Cardiovascular	Cardiovascular	8	CPAP mode	9345	7673	1672	1.217907	No	No	Failure	Reintubated
212	81	Male	Cardiovascular	Respiratory, Cardiovascular	4	T piece trial	5486	5542	-56	0.989895	Yes	No	Success	N/A
213	85	Male	Respiratory	Diabetes Mellitus, Renal	10	CPAP mode	8061	8832	-771	0.912704	No	No	Failure	Reintubated
214	28	Male	Cardiovascular	Diabetes Mellitus	9	T piece trial	10731	8108	2623	1.323508	No	No	Failure	Reintubated
215	23	Male	Cardiovascular	Cardiovascular, Diabetes Mellitus	5	CPAP mode	5487	4231	1256	1.296857	No	No	Success	N/A
216	80	Male	General/Other	Renal, Respiratory	2	CPAP mode	2404	1733	671	1.38719	Yes	Yes	Success	N/A
217	18	Male	Neurological	None	2	CPAP mode	8554	8637	-83	0.99039	Yes	Yes	Failure	Reintubated
218	18	Male	Respiratory	None	2	CPAP mode	6723	6018	705	1.117149	No	No	Success	N/A
219	70	Male	Cardiovascular	Respiratory, Cardiovascular	2	CPAP mode	1894	1661	233	1.140277	No	No	Success	N/A
220	25	Female	Cardiovascular	Cardiovascular	9	CPAP mode	15876	11922	3954	1.331656	No	No	Failure	Reintubated
221	64	Female	Cardiovascular	Renal, Respiratory	4	T piece trial	3074	3378	-304	0.910006	No	No	Success	N/A
222	65	Female	Neurological	Neurological	7	T piece trial	17794	15764	2030	1.128774	No	No	Success	N/A
223	70	Male	Cardiovascular	Diabetes Mellitus, Renal	4	T piece trial	3020	2865	155	1.054101	Yes	Yes	Success	N/A
224	66	Male	Respiratory	Diabetes Mellitus, Neurological	6	CPAP mode	6024	5375	649	1.120744	No	No	Success	N/A
225	37	Male	Neurological	Cardiovascular	8	CPAP mode	11330	9461	1869	1.197548	Yes	Yes	Success	N/A
226	56	Male	General/Other	Diabetes Mellitus	3	T piece trial	12155	11190	965	1.086238	No	No	Success	N/A
227	60	Female	General/Other	Respiratory	7	CPAP mode	18609	13279	5330	1.401386	No	No	Failure	NIV
228	27	Male	Respiratory	None	7	CPAP mode	5328	5796	-468	0.919255	No	No	Success	N/A
229	51	Male	Cardiovascular	None	4	CPAP mode	6771	7314	-543	0.925759	No	No	Success	N/A
230	82	Male	Respiratory	Cardiovascular, Diabetes Mellitus	7	CPAP mode	5621	5102	519	1.101725	Yes	No	Success	N/A
231	43	Male	General/Other	Cardiovascular, Respiratory	6	CPAP mode	10623	8667	1956	1.225684	No	No	Success	N/A
232	38	Male	Neurological	None	3	T piece trial	12413	11112	1301	1.117081	No	No	Success	N/A
233	27	Male	General/Other	None	10	T piece trial	42558	41557	1001	1.024087	No	No	Success	N/A
234	57	Female	Respiratory	Diabetes Mellitus	9	T piece trial	7959	5736	2223	1.387552	Yes	No	Success	N/A
235	24	Male	Renal	None	4	T piece trial	4164	4487	-323	0.928014	No	No	Success	N/A
236	62	Female	Renal	None	9	T piece trial	16034	14131	1903	1.134668	No	No	Failure	NIV
237	31	Male	Respiratory	Cardiovascular	10	CPAP mode	16975	16594	381	1.02296	Yes	No	Success	N/A
238	32	Male	Respiratory	Diabetes Mellitus	4	CPAP mode	18358	19605	-1247	0.936394	No	No	Success	N/A
239	55	Male	Cardiovascular	Cardiovascular	4	CPAP mode	5011	5132	-121	0.976422	Yes	No	Success	N/A
240	27	Female	Cardiovascular	None	3	CPAP mode	3095	2668	427	1.160045	No	No	Success	N/A
241	62	Female	Neurological	Renal, Diabetes Mellitus	9	T piece trial	15462	15831	-369	0.976691	No	Yes	Success	N/A
242	25	Male	Cardiovascular	Cardiovascular	2	T piece trial	1870	1387	483	1.348234	Yes	No	Success	N/A
243	29	Female	General/Other	None	9	T piece trial	34888	37293	-2405	0.935511	No	No	Success	N/A
244	21	Male	Cardiovascular	None	8	CPAP mode	13111	11135	1976	1.177458	No	No	Success	N/A
245	54	Male	Neurological	None	8	T piece trial	28434	23912	4522	1.18911	No	Yes	Failure	NIV
246	85	Male	General/Other	Cardiovascular	4	CPAP mode	5800	4454	1346	1.3022	Yes	No	Success	N/A
247	46	Male	General/Other	Respiratory	2	T piece trial	7896	7158	738	1.103101	No	Yes	Success	N/A
248	59	Male	Respiratory	Cardiovascular	5	T piece trial	6352	6326	26	1.00411	No	No	Success	N/A
249	25	Male	General/Other	None	10	CPAP mode	32262	33949	-1687	0.950308	No	No	Success	N/A
250	40	Male	Respiratory	None	6	CPAP mode	25112	26171	-1059	0.959535	No	No	Success	N/A
251	45	Female	Respiratory	Cardiovascular	2	T piece trial	3041	3102	-61	0.980335	No	No	Success	N/A
252	52	Male	Respiratory	Diabetes Mellitus	6	T piece trial	11294	8128	3166	1.389518	No	No	Success	N/A
253	78	Male	Renal	Respiratory, Renal	9	CPAP mode	9515	7177	2338	1.325763	Yes	No	Success	N/A
254	25	Male	Respiratory	Diabetes Mellitus	9	T piece trial	13499	9684	3815	1.393949	Yes	No	Success	N/A
255	22	Female	Respiratory	Diabetes Mellitus	6	T piece trial	18263	16282	1981	1.121668	No	No	Success	N/A
256	68	Male	Renal	Renal, Respiratory	10	CPAP mode	17806	16675	1131	1.067826	Yes	No	Success	N/A
257	62	Male	General/Other	None	2	T piece trial	9491	9877	-386	0.960919	No	No	Success	N/A
258	29	Male	Respiratory	None	5	CPAP mode	7425	5422	2003	1.369421	No	No	Success	N/A
259	61	Male	Neurological	Diabetes Mellitus	4	T piece trial	13083	11435	1648	1.144119	No	No	Success	N/A
260	24	Male	General/Other	Cardiovascular	3	CPAP mode	3510	2638	872	1.330553	No	Yes	Success	N/A
261	38	Male	Cardiovascular	None	9	T piece trial	8607	9344	-737	0.921126	No	No	Success	N/A
262	38	Female	Renal	Cardiovascular	8	CPAP mode	6769	6079	690	1.113506	Yes	No	Success	N/A
263	52	Female	Respiratory	Respiratory	10	T piece trial	39963	32010	7953	1.248454	No	No	Success	N/A
264	53	Male	Neurological	Cardiovascular	3	CPAP mode	3672	2779	893	1.321339	No	Yes	Success	N/A
265	71	Male	Neurological	None	5	T piece trial	21098	18495	2603	1.140741	No	No	Success	N/A
266	78	Female	Renal	Cardiovascular	7	T piece trial	8222	7170	1052	1.146722	Yes	No	Failure	NIV
267	54	Male	Cardiovascular	Diabetes Mellitus, Respiratory	4	T piece trial	4872	5290	-418	0.920983	No	No	Success	N/A
268	77	Male	Cardiovascular	Cardiovascular	9	T piece trial	12349	11447	902	1.078798	Yes	No	Failure	Reintubated
269	57	Male	Cardiovascular	None	4	T piece trial	5694	5094	600	1.117786	No	No	Success	N/A
270	34	Female	Respiratory	None	3	T piece trial	3263	2698	565	1.209414	No	No	Success	N/A
271	32	Female	Neurological	None	4	T piece trial	12269	10614	1655	1.155926	No	No	Success	N/A
272	62	Male	General/Other	None	7	T piece trial	22775	16335	6440	1.394245	No	No	Success	N/A
273	73	Male	Respiratory	Neurological, Cardiovascular	3	T piece trial	2903	2727	176	1.06454	Yes	No	Success	N/A
274	41	Male	Cardiovascular	None	9	CPAP mode	13321	10167	3154	1.310219	No	No	Success	N/A
275	45	Female	Cardiovascular	None	2	CPAP mode	2681	2479	202	1.081484	No	No	Success	N/A
276	30	Male	Respiratory	Diabetes Mellitus	8	T piece trial	16303	11993	4310	1.359376	No	No	Success	N/A
277	24	Male	Cardiovascular	Diabetes Mellitus	3	CPAP mode	3226	2390	836	1.349791	No	No	Success	N/A
278	51	Male	Respiratory	None	7	T piece trial	30595	24254	6341	1.261441	No	Yes	Failure	Reintubated
279	30	Male	Respiratory	None	3	T piece trial	5985	6457	-472	0.926901	No	No	Success	N/A
280	26	Male	Renal	None	3	CPAP mode	3689	3557	132	1.03711	No	No	Success	N/A
281	22	Male	Respiratory	None	9	T piece trial	36255	32983	3272	1.099203	No	No	Success	N/A
282	53	Female	Neurological	None	3	T piece trial	10553	9200	1353	1.147065	No	No	Success	N/A
283	19	Male	Respiratory	None	8	CPAP mode	38286	38255	31	1.00081	No	No	Success	N/A
284	26	Male	Cardiovascular	None	4	CPAP mode	6333	5976	357	1.059739	No	No	Success	N/A
285	46	Female	Renal	Cardiovascular	9	T piece trial	14200	12775	1425	1.111546	Yes	No	Failure	Reintubated
286	26	Female	General/Other	Cardiovascular	8	CPAP mode	9959	9709	250	1.025749	Yes	No	Success	N/A
287	37	Female	Neurological	Diabetes Mellitus	9	CPAP mode	23813	20803	3010	1.144691	No	No	Success	N/A
288	32	Male	Respiratory	Cardiovascular	8	T piece trial	13847	11008	2839	1.257903	Yes	No	Success	N/A
289	38	Male	General/Other	None	7	CPAP mode	18570	19832	-1262	0.936365	No	No	Success	N/A
290	71	Male	Neurological	Cardiovascular	8	CPAP mode	9285	6660	2625	1.394144	Yes	No	Success	N/A
291	78	Male	Renal	Renal, Diabetes Mellitus	6	T piece trial	5257	5588	-331	0.940766	No	Yes	Success	N/A
292	56	Female	Cardiovascular	None	8	CPAP mode	9095	7493	1602	1.2138	No	No	Success	N/A
293	63	Male	General/Other	Neurological, Renal	2	CPAP mode	1820	1434	386	1.269177	Yes	Yes	Success	N/A
294	40	Male	Neurological	Respiratory	2	T piece trial	7321	6782	539	1.079475	Yes	No	Failure	Reintubated
295	29	Female	Renal	None	10	T piece trial	8055	8763	-708	0.919206	No	No	Success	N/A
296	73	Male	Respiratory	None	6	T piece trial	22300	17572	4728	1.269064	No	Yes	Success	N/A
297	44	Male	Respiratory	None	6	CPAP mode	15545	16034	-489	0.969502	No	No	Success	N/A
298	67	Male	Respiratory	None	4	T piece trial	10766	9825	941	1.095776	No	No	Success	N/A
299	61	Male	Respiratory	Diabetes Mellitus	6	T piece trial	22312	16584	5728	1.345393	No	No	Success	N/A

300	85	Male	Cardiovascular	None	4	CPAP mode	4386	3959	427	1.107856	No	No	Success	N/A
301	22	Female	General/Other	Cardiovascular, Diabetes Mellitus	10	T piece trial	12263	11645	618	1.05307	Yes	No	Success	N/A
302	32	Female	Renal	Cardiovascular	10	T piece trial	14354	13676	678	1.049576	No	No	Success	N/A
303	43	Female	Cardiovascular	Respiratory	7	T piece trial	11288	11090	198	1.017854	No	No	Success	N/A
304	59	Male	Neurological	Cardiovascular	2	T piece trial	3504	3152	352	1.111675	No	No	Success	N/A
305	19	Female	Cardiovascular	None	8	CPAP mode	13637	11211	2426	1.216395	Yes	No	Success	N/A
306	42	Male	Neurological	None	8	T piece trial	32962	34331	-1369	0.960124	No	No	Success	N/A
307	27	Male	Respiratory	None	5	T piece trial	21697	23288	-1591	0.931682	No	No	Failure	NIV
308	22	Female	General/Other	None	9	CPAP mode	37440	33587	3853	1.114717	Yes	Yes	Success	N/A
309	77	Male	Cardiovascular	Neurological	7	T piece trial	10411	8728	1683	1.192828	No	No	Success	N/A
310	22	Male	Cardiovascular	Cardiovascular, Diabetes Mellitus	5	CPAP mode	7617	6398	1219	1.190528	No	No	Success	N/A
311	61	Male	General/Other	Respiratory, Renal	5	CPAP mode	6176	4450	1726	1.387865	No	Yes	Success	N/A
312	72	Female	General/Other	Neurological, Diabetes Mellitus	3	CPAP mode	11293	10828	465	1.042944	No	Yes	Success	N/A
313	20	Male	Cardiovascular	Cardiovascular	3	T piece trial	4763	3491	1272	1.364366	No	No	Failure	NIV
314	80	Male	Neurological	Respiratory, Renal	5	CPAP mode	7553	6113	1440	1.235564	Yes	No	Success	N/A
315	55	Male	Respiratory	Cardiovascular	6	CPAP mode	6827	6668	159	1.023845	Yes	No	Failure	NIV
316	45	Female	Cardiovascular	Respiratory, Cardiovascular	9	CPAP mode	15559	15498	61	1.003936	No	No	Success	N/A
317	53	Female	Cardiovascular	Respiratory, Diabetes Mellitus	7	T piece trial	7415	7282	133	1.018264	No	No	Success	N/A
318	65	Male	Cardiovascular	None	5	T piece trial	7058	7206	-148	0.979462	No	No	Success	N/A
319	71	Female	General/Other	Respiratory	8	T piece trial	26452	23054	3398	1.147393	No	No	Success	N/A
320	77	Male	Respiratory	None	9	T piece trial	24242	19331	4911	1.254048	No	No	Success	N/A
321	38	Female	Neurological	Diabetes Mellitus	3	T piece trial	13743	11844	1899	1.160334	No	No	Success	N/A
322	30	Male	Respiratory	None	8	T piece trial	11188	9557	1631	1.17066	No	No	Failure	Reintubated
323	64	Male	Cardiovascular	Respiratory	3	T piece trial	4558	4885	-327	0.93306	No	No	Failure	NIV
324	64	Male	General/Other	Renal, Respiratory	3	CPAP mode	3242	2535	707	1.278895	No	No	Failure	Reintubated
325	61	Male	Cardiovascular	Cardiovascular	9	CPAP mode	12174	9358	2816	1.300919	No	No	Success	N/A
326	64	Male	General/Other	Cardiovascular, Renal	3	CPAP mode	3627	3940	-313	0.920558	No	No	Success	N/A
327	27	Male	Respiratory	None	10	CPAP mode	43945	47831	-3886	0.918756	No	No	Success	N/A
328	29	Female	General/Other	Diabetes Mellitus	2	T piece trial	9381	9927	-546	0.944998	No	Yes	Failure	Reintubated
329	41	Male	Renal	Respiratory, Diabetes Mellitus	10	CPAP mode	10674	7827	2847	1.363741	No	No	Success	N/A
330	81	Male	Respiratory	Respiratory	2	CPAP mode	7758	6301	1457	1.231233	No	No	Failure	Reintubated
331	23	Female	Neurological	None	3	CPAP mode	13706	11567	2139	1.184923	No	No	Success	N/A
332	84	Male	Neurological	Renal	7	CPAP mode	10032	9273	759	1.081851	Yes	No	Success	N/A
333	62	Female	Cardiovascular	Neurological	7	T piece trial	6555	5139	1416	1.27554	No	No	Success	N/A
334	67	Male	General/Other	Respiratory	2	T piece trial	8078	7464	614	1.082262	No	No	Success	N/A
335	81	Female	Neurological	Respiratory	8	T piece trial	22983	20506	2477	1.120794	No	No	Success	N/A
336	40	Male	Respiratory	None	9	CPAP mode	36575	36435	140	1.003842	No	No	Success	N/A
337	84	Female	Cardiovascular	Respiratory, Neurological	3	T piece trial	4208	3125	1083	1.34656	No	No	Success	N/A
338	34	Male	Respiratory	None	9	CPAP mode	9536	9221	315	1.034161	No	No	Success	N/A
339	49	Male	Respiratory	Respiratory	10	T piece trial	41138	39338	1800	1.045757	No	No	Success	N/A
340	65	Female	Cardiovascular	Renal, Diabetes Mellitus	7	T piece trial	11240	12224	-984	0.919503	No	No	Success	N/A
341	26	Male	Respiratory	Diabetes Mellitus	6	T piece trial	16296	16286	10	1.000614	No	No	Success	N/A
342	74	Male	Renal	Respiratory	10	T piece trial	10023	8239	1784	1.216531	No	No	Success	N/A
343	81	Female	Neurological	Respiratory	10	T piece trial	34677	37474	-2797	0.925362	No	No	Success	N/A
344	36	Female	Respiratory	Cardiovascular	2	CPAP mode	3600	3211	389	1.121146	Yes	No	Success	N/A
345	29	Female	Cardiovascular	None	4	T piece trial	6715	5643	1072	1.18997	No	No	Success	N/A
346	31	Female	Neurological	None	10	CPAP mode	30609	32348	-1739	0.946241	No	No	Success	N/A
347	26	Female	Respiratory	None	3	CPAP mode	9672	8088	1584	1.195846	No	No	Success	N/A
348	24	Male	Cardiovascular	Cardiovascular	5	CPAP mode	5751	5560	191	1.034353	No	No	Success	N/A
349	20	Male	Respiratory	None	5	CPAP mode	20793	19339	1454	1.075185	No	No	Failure	NIV
350	61	Male	Cardiovascular	Neurological, Renal	3	CPAP mode	3637	2940	697	1.237075	Yes	No	Success	N/A
351	78	Male	Renal	Diabetes Mellitus	4	T piece trial	6183	6100	83	1.013607	No	No	Success	N/A
352	65	Male	General/Other	None	4	T piece trial	16552	14795	1757	1.118756	No	Yes	Success	N/A
353	20	Male	General/Other	None	2	T piece trial	6030	5059	971	1.191935	No	Yes	Failure	NIV
354	26	Male	Renal	None	7	T piece trial	7015	5787	1228	1.2122	No	No	Success	N/A
355	57	Male	Respiratory	Cardiovascular	9	CPAP mode	7970	8449	-479	0.943307	No	Yes	Success	N/A
356	28	Male	Cardiovascular	None	4	T piece trial	6806	4812	1994	1.414381	No	No	Failure	Reintubated
357	25	Male	Respiratory	Cardiovascular, Diabetes Mellitus	6	T piece trial	6914	5131	1783	1.347496	No	No	Success	N/A
358	84	Male	Respiratory	Cardiovascular, Renal	2	CPAP mode	2501	2377	124	1.052167	Yes	Yes	Success	N/A
359	31	Female	Cardiovascular	None	8	CPAP mode	12035	13055	-1020	0.921869	No	No	Success	N/A
360	45	Male	Cardiovascular	Respiratory	2	T piece trial	3118	2492	626	1.251204	No	No	Failure	NIV
361	69	Female	General/Other	Diabetes Mellitus	10	T piece trial	34455	32846	1609	1.048986	No	No	Success	N/A
362	29	Male	General/Other	Cardiovascular, Diabetes Mellitus	9	CPAP mode	13273	11977	1296	1.108207	Yes	No	Success	N/A
363	47	Male	Respiratory	Respiratory	8	T piece trial	22396	15910	6486	1.407668	No	No	Success	N/A
364	48	Female	General/Other	Respiratory	8	T piece trial	29063	31188	-2125	0.931865	No	No	Success	N/A
365	32	Female	Cardiovascular	None	6	T piece trial	5334	4699	635	1.135135	No	No	Success	N/A
366	18	Male	General/Other	None	6	T piece trial	26014	19098	6916	1.362132	No	No	Success	N/A
367	62	Male	Respiratory	Diabetes Mellitus, Renal	6	CPAP mode	5418	5850	-432	0.926154	No	No	Failure	Reintubated
368	77	Male	Respiratory	Respiratory, Renal	2	CPAP mode	3224	3291	-67	0.979641	Yes	Yes	Success	N/A
369	19	Male	Respiratory	None	9	CPAP mode	35003	32728	2275	1.069512	No	No	Success	N/A
370	84	Male	Respiratory	Neurological, Respiratory	2	T piece trial	7904	5978	1926	1.322181	No	No	Success	N/A

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