

# Metabolic alkalosis in intensive care: A narrative review.

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## Abstract

**Introduction:** In the intensive care unit (ICU), metabolic alkalosis is the most common acid–base disorder. A link has been observed between this disorder and an increased risk of death. However, this increased mortality is likely due primarily to the severity of the underlying disease rather than to metabolic alkalosis itself.

**Objective of the review:** This article is a narrative review aimed at updating the diagnosis, treatment, and prognosis of metabolic alkalosis in the context of intensive care management.

#### Key points of the review:

Metabolic alkalosis often develops during a hospital stay and is usually a consequence of intensive care unit (ICU) interventions.

Mortality in patients with a pH of 7.55–7.56 is 41%, that in patients with a pH of 7.57–7.59 is 47%, that in patients with a pH of 7.60–7.64 is 65%, and that in patients with a pH of 7.65–7.70 is 80%.

Hypokalemia is a significant alteration in metabolic alkalosis, causing muscle weakness and paralysis, areflexia, ileus, ST-segment depression, the presence of a U wave, and QT prolongation.

**Conclusion:** The underlying etiology has a significant influence on mortality associated with metabolic alkalosis. Therapeutic interventions have been limited in guiding adequate fluid management in patients with or without chloride sensitivity; the role of carbonic anhydrase inhibitor-type diuretics has increased, with varying evidence regarding their usefulness.

**Keywords:** Metabolic alkalosis, acid–base status, alkalaemia, intensive care, potassium.

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In the intensive care unit (ICU), unlike other hospital settings, disorders in water and electrolyte homeostasis and acid–base status occur more frequently and are associated with an increase in adverse outcomes; however, in many cases, these conditions are potentially preventable [1–3]. In the ICU, metabolic alkalosis is the most common acid–base balance disorder [4–5]. A connection between this disorder and an increased risk of death has been observed. However, this increased mortality is likely due mainly to the severity of the underlying disease rather than directly to metabolic alkalosis [1].

Metabolic alkalosis usually develops during the hospital stay and is often a consequence of intensive care unit (ICU) interventions [4–6]. The maintenance of acid–base homeostasis, which is mediated by buffer systems at both the intracellular and extracellular levels, is essential for effective therapeutic interventions [7]. Therefore, the early recognition of these conditions has implications for the management and prevention of complications [8].

The analysis of pH as an indirect measure of the concentration of hydrogen ions allows the identification of pathological states such as acidemia ( $[H^+] > 40$  nmol/L) or alkalemia ( $[H^+] < 40$  nmol/L), revealing these situations as a consequence of changes in hydrogen ion concentrations [9]. Similarly, changes in the plasma bicarbonate  $[HCO_3^-]$  concentration generate primary metabolic alterations such as metabolic alkalosis, the most common disorder in up to 51% of cases, and induce systemic conditions such as vasoconstriction, cardiac arrhythmias, alterations in consciousness, decreased cerebral and myocardial perfusion, increased affinity of  $O_2$  for hemoglobin and reduced release of  $O_2$ , as well as an increase in the binding of ionic calcium with albumin [3,7].

The objective of this review is to describe the role of metabolic alkalosis in critically ill patients and how it contributes to systemic reactions, which are often associated with an increase in mortality in critically ill patients.

## Definition and epidemiology

Alkalosis is defined as an arterial pH greater than the normal pH of the body ( $> 7.45$ ). This is mainly due to the increase in the loss of nonitratable acids, which results in an excess of  $HCO_3^-$  or a decrease in  $H^+$  concentrations. An arterial  $pH > 7.45$  and an excess of bases (BE)  $> +3$  mmol/L are generally classified as metabolic alkalosis [10].

However, upon admission, acidosis (whether respiratory or metabolic) is usually more common. During the stay in the unit and after interventions are carried out, isolated or mixed metabolic alkalosis appears to be the most prevalent acid–base disorder in the ICU [4,5]. For example, in a study carried out by Okusawa et al., 87.5% of 293 surgical patients achieved a normal acid–base balance before surgery. After surgery, 50.5% of the patients developed metabolic alkalosis [11].

The mortality associated with metabolic alkalosis is directly proportional to the increase in pH. The mortality of patients at pH 7.55–

7.56 is 41%, that of patients at pH 7.57–7.59 is 47%, that of patients at pH 7.60–7.64 is 65%, and that of patients at pH 7.65–7.70 is 80% [12–14]. Therefore, the correction and prevention of metabolic alkalosis are important prognostic factors, although the evidence is somewhat controversial.

## Pathophysiology

Among the etiologies associated with the development of metabolic alkalosis are the therapeutic interventions used to aggressively treat circulatory shock, acidemia, volume overload, severe coagulopathy, respiratory failure, intestinal obstruction, and acute respiratory failure. In Table 1, several factors are presented according to the interpretation used to evaluate the acid–base balance [6,15].

Metabolic alkalosis results from the gain of a base or the loss of an acid. In most cases, this will cause an increase in pH and  $HCO_3^-$ . Under normal conditions, the kidney excretes large amounts of  $HCO_3^-$ . The development of metabolic alkalosis requires not only a phase during which  $HCO_3^-$  is added or acid is lost, but also a maintenance phase during which the kidney is unable to excrete  $HCO_3^-$  [6].

Under the premise of the existence of pathophysiological processes that generate and perpetuate metabolic alkalosis, two groups of etiological mechanisms are described: those of gastrointestinal and renal origin. The first mechanism involves an increase in the absorption of calcium and potassium exchange resins, the loss of hydrogen ions associated with vomiting, the use of a nasogastric tube, and the presence of alkalotic diarrhea [16,17]. At the renal level, the use of thiazide and loop diuretics, redistribution of hydrogen ions secondary to hypokalemia and hypovolemia, excess mineralocorticoids, posthypercapnia states and associated endocrinological conditions such as Conn syndrome are the leading causes of this disease [15].

On the other hand, perpetuating processes such as hypokalemia, hyperaldosteronism, and hypovolemia are responsible for maintaining or worsening the state of alkalosis. In a similar way to what occurs in hyperaldosteronism, aspects such as hypokalemia and hypovolemia create a feedback cycle, since the decrease in blood pressure is sensed by the juxtaglomerular cells of the kidney, thus generating the release of renin and consequently activating the renin–angiotensin–aldosterone system (RAAS) to promote the excretion of potassium at the renal level and therefore produce hypokalemia [15].

Some mechanisms converge and can promote the generation of metabolic alkalosis at the renal level, such as a decrease in bicarbonate production in the intercalated cells of the collecting duct. This results in a decreased renal excretion of hydrogen ions and an increase at the extracellular level, which is compensated by carbonic anhydrase in the distal and collecting tubules. This metalloenzyme is present not only in kidney tissue but also in cardiac, hepatic, and central nervous system tissues. Additionally, its catalytic function generates a double bond by eliminating molecules of  $H_2O$ ,  $CO_2$ , and  $HN_3$ , which facilitates the ionization of  $CO_2$  into carbonic acid, producing a proton



(H<sup>+</sup>) and a bicarbonate anion (HCO<sub>3</sub><sup>-</sup>). In metabolic alkalosis, this reaction mediated by AC-II and AC-IV is reversed through the conversion of bicarbonate back into CO<sub>2</sub> and water, facilitating its elimination in the lungs and lowering blood pH [9, 15, 18].

On the other hand, metabolic alkalosis caused by the loss of hydrogen ions at the renal level results from the use of thiazide or loop diuretics, which target the Na–Cl cotransporter in the distal convoluted tubule and the Na–K–2Cl cotransporter in the ascending branch of the loop of Henle. These medications increase potassium excretion, leading to hypotension and the redistribution of extracellular hydrogen ion concentrations [18].

At the respiratory level, metabolic alkalosis stimulates the primary center of respiratory control, temporarily increasing the respiratory rate. However, in patients with pulmonary pathology, the respiratory rate decreases, leading to alveolar hypoventilation, which is sustained, resulting in CO<sub>2</sub> retention and secondary hypoxemia. At the cardiac level, the increase in pH raises the sensitivity of myocardial cells to catecholamines, facilitating a positive chronotropic effect. This increase in automatism heightens the oxygen demands in the myocardium while lowering the oxygen pressure at the peripheral level. If this process continues over time, tissue hypoxia ensues, making it more difficult for hemoglobin to release oxygen. Ultimately, the reduced oxygen supply to myocardial cells diminishes their contractile function [6,18].

On the other hand, metabolic alkalosis promotes the entry of calcium into the cell by stimulating the sodium/calcium exchanger. This increase in calcium enhances the vascular expression of the enzyme endothelial nitric oxide synthetase (eNOS), which explains the observed peripheral arterial vasodilation, the reduction in peripheral vascular resistance, and consequently, hypotension. In contrast, the opposite effect occurs in cerebral vessels, where an increase in pH, associated with hypoxemia, causes vasoconstriction, decreases cerebral blood flow, and leads to alterations in the state of consciousness [19].

In patients with metabolic alkalosis, elevated pH and decreased levels of hydrogen ions in the extracellular space activate the K<sup>+</sup>/H<sup>+</sup> exchange pump, facilitating potassium entry into the cell and inducing hypokalemia through redistribution. The resulting hypokalemia alters the action potential of cardiomyocytes, increasing their susceptibility to arrhythmias and cardiac arrest through three mechanisms:

1. An increase in the electronegativity of the membrane, which raises the resting membrane potential, also activates voltage-dependent Na<sup>+</sup> channels. This increases the maximum rate of Na<sup>+</sup> entry into the cell and steepens phase zero, shortening its duration.
2. An increased delta between the resting membrane potential and the depolarization threshold potential results in a decrease in myocyte excitability.
3. Potassium conductivity decreases during phases 2 and 3 of the action potential, extending repolarization [6,18].

Secondary hypokalemia in smooth muscle alters the muscle's action potential. It increases the membrane's electronegativity, preventing myocyte contraction and leading to progressive weakness and paralysis of the diaphragm, which causes respiratory failure. At the intestinal level, it results in alterations in intestinal transit, such as paralytic ileus [18].

In metabolic alkalosis, the increase in pH creates an alkaline environment that enables calcium to bind strongly to plasma proteins and reduces ionized calcium, leading to hypocalcemia. This condition primarily affects the neuromuscular junction, where calcium facilitates the release of neurotransmitters into the synaptic cleft. At the neuromuscular junction, this contributes to the development of tetany, hemiballism, tonic-clonic seizures, psychosis, and dementia [6].

## Clinical manifestations

The clinical manifestations of metabolic alkalosis range from alterations of the central nervous system to muscle damage, increasing the risk of fatal arrhythmias [18, 20, 21]. These relate to severe disturbances in electrolyte levels of sodium, potassium, calcium, chloride, hydrogen, phosphate, or albumin, which lead to changes in the anion gap [22]. The effects of alkalosis on the body are varied and are often linked to related electrolyte disorders. [18].

Its impact on the body occurs at various levels. This includes the central nervous system (ranging from confusion to coma), the peripheral nervous system (with neuropathic symptoms such as

**Table 1.** Causes of metabolic alkalosis

<b>Traditional model (Bicarbonate)</b>	<b>Physicochemical Model (Stewart)</b>
<p><b>Exogenous bicarbonate</b>                      Infusion of sodium bicarbonate solution.                      Infusion of bicarbonate precursors:</p> <ul style="list-style-type: none"> <li>• Sodium Acetate (Plasma-Lyte 148 Intravenous Fluid)</li> <li>• Sodium gluconate (Plasma-Lyte 148 intravenous fluid)</li> <li>• Sodium lactate (Hartmann's intravenous fluid)</li> <li>• Sodium citrate (citrate liquid for RRT)</li> </ul>	<p><b>Increased difference of strong ions</b>                      Chloride depletion relative to sodium:</p> <ul style="list-style-type: none"> <li>• Loop and thiazide diuretics.</li> <li>• Vomiting and loss of nasogastric fluid.</li> <li>• Hypokalemia.</li> <li>• Compensation for hypercapnia.</li> </ul> <p><b>Excess sodium in relation to chloride:</b></p> <ul style="list-style-type: none"> <li>• Infusion of baking soda or bicarbonate precursors.</li> <li>• Activation of the renin-angiotensin-aldosterone system (RAAS).</li> <li>• Excess mineralocorticoids (aldosterone).</li> </ul> <p>ATOT reduction</p> <ul style="list-style-type: none"> <li>• Hypoproteinemia.</li> <li>• Hypoalbuminemia.</li> </ul>
<p><b>Endogenous bicarbonate</b>                      Diuretics:  <ul style="list-style-type: none"> <li>• Thiazides</li> <li>• Loop (furosemide)</li> </ul>                     Compensation for hypercapnia.                      Ion loss (hydrogen)                      Vomiting                      Nasogastric tube leaks.</p>	
<p>Reproduced from Park et al. [15] ATOT: refers to the total concentration of weak anions, which mainly includes albumin, phosphates and bicarbonate in the blood.</p>	



paresthesia and weakness), the myocardium (resulting in arrhythmia), and the skeletal muscle (causing weakness and spasms), among others [18].

Hypokalemia is a significant alteration in metabolic alkalosis. It can lead to muscle weakness, muscle paralysis, areflexia, intestinal ileus, and severe electrocardiographic changes, such as ST-segment depression and the presence of U waves, potentially resulting in QT complex prolongation. [23]. Hypokalemia causes metabolic alkalosis through a variety of mechanisms, including the movement of  $K^+$  out of cells in exchange for  $H^+$  ions, increased renal ammonia excretion, and the activation of  $H^+K^+$  ATPase in the cortical collecting duct [6]. Therefore, the evaluation of hypokalemia is a relevant variable in the management of patients with metabolic alkalosis.

Another electrolyte commonly affected by alkalosis disorders is calcium. Alkalosis results in a decrease in ionized calcium and an increase in  $pCO_2$ . Many neurological symptoms associated with alkalaemia, such as paresthesia, tetany, muscle spasm, and seizures, are secondary to hypocalcemia [6].

The antecedents of medical history, such as aldosteronism and cystic fibrosis, are associated with the presence of metabolic alkalosis [24]. In cystic fibrosis, the excessive loss of sodium, chloride, and water can lead to dehydration, as well as hyponatremic and hypochloremic effects [25]. This may result in alterations in glomerular filtration and activate the renin–angiotensin–aldosterone system, ultimately causing changes in sodium–potassium pumps at the renal level that favor metabolic alkalosis [26]. Aldosteronism presents with disturbances in the internal environment, including hypokalemia, hypercalciuria, and metabolic alkalosis, along with nonspecific symptoms such as generalized weakness, constipation, and lassitude [27, 28].

## Diagnosis

The diagnosis of metabolic alkalosis is based on the identification of a serum pH ( $pH > 7.45$ ) accompanied by a plasma bicarbonate concentration greater than 27 mEq/L, as measured through an arterial blood gas [18,29]. Confirmation of the diagnosis of metabolic alkalosis is supported not only by arterial blood gas but also by identifying an increase in the anion gap [30]. The measurement of chlorine in urine is the most pertinent since it can differentiate alkalosis due to chloride depletion from chloride-resistant alkalosis, a relevant fact in the therapeutic orientation [24]. The measurement of potassium levels is also helpful for diagnosis, together with the evaluation of volemia status [24].

A thorough clinical history must complement the diagnostic approach to identify exogenous, pharmacological, and pathological causes that may be responsible for the changes in pH [18]. The medical history can also provide clues about the presence of diseases such as primary aldosteronism or cystic fibrosis, as well as possible drugs that cause alkalosis, including penicillin and carbenicillins [18]. The physical examination should look for signs of volume depletion that could guide the etiology and eventual therapeutic indications. In addition to arterial or venous blood gases, electrolytes, especially

sodium, potassium, magnesium, phosphorus, chlorine, and calcium, should be measured completely [6,18].

Metabolic alkalosis can be divided into two main categories according to whether the volume of the extracellular fluid (ECF) is contracted or expanded [18]. Depending on the systemic blood pressure and electrolytes in the urine, patients with metabolic alkalosis are divided into a group sensitive to chloride ( $Cl^-$  in urine  $<20$  mmol/L) and a group resistant to chloride ( $Cl^-$  in urine  $> 20$  mmol/L) [18]. Additional etiological studies, including hormonal evaluation, are beyond the scope of this review.

## Treatment

Treatment for metabolic alkalosis depends on the underlying cause. The pH level at which treatment should begin is not clearly established and remains at the discretion of the treating physicians [6]. Since the morbidity associated with alkalaemia can increase above a pH of 7.55, this is a reasonable target value [6]. Due to the effects of hypokalemia on  $H^+$  transport to cells and on the renal reabsorption of  $HCO_3^-$ , hypokalemia must be treated intensively. Whenever possible, volume depletion should be addressed and corrected [6].

First, in alkalosis with a reduction in the extracellular volume (urinary  $Cl^- <20$  mmol/L), treatment focuses on correcting the factors that precipitate or maintain alkalosis. These factors include a decrease in the glomerular filtration rate (GFR) due to hypovolemia, chlorine deficiency, and hypokalemia. In this regard, administering intravenous fluids containing chlorine can expand the intravascular volume, improve renal perfusion, and increase glomerular filtration, thereby disrupting the tubular reabsorption of sodium, potassium, bicarbonate, chlorine, and water, and contributing to the excretion of bicarbonate. A practical approach to monitor the response to fluid therapy is by tracking urinary chloride levels, as an increase in these levels would indicate sufficient volume expansion. Replenishing  $K^+$  to address hypokalemia reduces ammoniogenesis and the generation of new  $HCO_3^-$ , in addition to decreasing the absorption of  $HCO_3^-$ . Both correcting hypovolemia and managing hypokalemia help address chlorine-dependent alkalotic states [18].

On the other hand, those that are not sensitive to chlorine should be noted. In cases where a patient has hypervolemia and high blood pressure, urinary chloride levels exceed 20 mmol/L. In these situations, it is crucial to identify the underlying etiology, especially if there is an increase in mineralocorticoid activity, and to address potassium disorders. If an excess of mineralocorticoids is found, eliminating the endogenous source of the mineralocorticoid can help resolve the acid–base balance disorder. Additionally, exogenous sources should be identified and limited; see Table 1 [18].

Another therapeutic intervention to consider is the infusion of hydrochloric acid, which has been reported and described in the literature since the 1960s [30]. The adverse effects of this intervention are varied; it is not a first-line indication, and it has been recommended for refractory cases [31]. A publication in surgical patients, which included 30 participants, reported that the most frequent



etiology of metabolic alkalosis was the use of diuretics; correction of metabolic alkalosis was achieved, monitored by changes in  $p\text{CO}_2$ , which is well tolerated and has a low rate of adverse effects [31]. However, its therapeutic utility and the metabolic alkalosis situations it addresses have not yet been clarified.

A special scenario in intensive care involves metabolic alkalosis in patients with acute respiratory failure, a common situation that may delay the process of releasing ventilatory assistance. In this context, carbonic anhydrase inhibitors block renal reabsorption of bicarbonate, thereby reversing metabolic alkalosis. Specifically, patients with chronic obstructive pulmonary disease (COPD) and chronic respiratory acidosis show increased renal bicarbonate reabsorption to compensate for the acidic pH resulting from respiratory disorders. When a patient with COPD exacerbation needs ventilatory support, the  $p\text{CO}_2$  decreases, while the bicarbonate level may remain elevated, leading to a condition known as posthypercapnic metabolic alkalosis (PHA) [32]. The alkalaemia that arises in this manner could suppress the respiratory drive, leading to further deterioration of ventilation with delayed weaning from invasive ventilatory support [33].

A recent meta-analysis addressing this situation revealed that using carbonic anhydrase inhibitors could positively impact the gasometric behavior of these patients, reducing the duration of mechanical ventilation [34]. However, the evidence concerning the real benefits of this pharmacological intervention remains controversial [35]. Regarding patients receiving noninvasive mechanical ventilation, the available evidence is even more limited, with no benefits reported in the literature to date [36]. Although acetazolamide effectively improves alkalaemia in patients with PHA, its impact on severe outcomes, such as the duration of mechanical ventilation, hospital stay, and mortality, may be limited by factors like patient complexity and coadministered medications, especially other types of diuretics [32]. Publications with more rigorous methodology are needed to clarify the role of acetazolamide in managing metabolic alkalosis.

## Prognosis

Several studies have demonstrated that metabolic alkalosis is associated with increased morbidity and mortality [5, 14]. There is evidence of a rise in mortality alongside the increase in pH [6]. However, it is unclear whether the increase in mortality is due to the alkalaemia itself or the underlying conditions responsible for the change in pH [6]. Kru et al. evaluated the behavior of metabolic alkalosis in the context of sepsis and septic shock. In this retrospective cohort study, no association could be demonstrated between metabolic alkalosis and mortality at 30 days and one year. However, metabolic alkalosis was associated with a more extended ICU stay (6 versus 2 days,  $P < 0.001$ ) [37]. Therefore, establishing and maintaining this condition may lead to worse outcomes; being preventable, early identification is crucial [38, 39].

## Conclusions

Metabolic alkalosis is the most common acid–base balance disorder in intensive care and is generally the result of interventions routinely used in the ICU. Its prognostic implications are not entirely clear; however, it has been associated with an increase in mortality that may be synergistic with the underlying etiology. Therapeutic interventions have been limited to guiding the proper management of fluids in scenarios of sensitivity or insensitivity to chlorine, and the role of carbonic anhydrase inhibitor-type diuretics has increased, with mixed evidence regarding their effectiveness. Studies aimed at developing more beneficial therapeutic interventions are necessary for diverse populations in intensive care.

### Abbreviations

COPD: chronic obstructive pulmonary disease.  
 $\text{HCO}_3^-$ : Bicarbonate.  
ECL: Extracellular fluid.  
ICU: intensive care unit.

### Supplementary information

The supplementary materials have not been declared.

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### Authors' contributions

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### Consent for publication

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### Conflicts of interest

The authors declare that they have no conflicts of interest.

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