

What this compound is and where it is used

Propylene glycol is a solvent and carrier employed in a variety of medical and nonmedical products. In clinical settings, it is most important because it can appear in **IV medications**, especially certain sedatives and other formulations that need a liquid base. It can also be included in some **oral products** and **topical products**. Most exposures are harmless, but large or prolonged exposure can create a **toxicity** problem, especially in hospitalized patients.

This is important because propylene glycol is not just an ingredient; it is a compound that is *metabolized* by the body into other substances. Under the right circumstances, those **metabolites** can affect acid-base balance and lead to abnormal serum chemistry findings. In some patients, the clinical picture includes **high anion gap** findings, especially when the exposure is significant or renal clearance is impaired.

From a diagnostic workup standpoint, propylene glycol exposure is often overlooked unless the medication list is reviewed closely. That is why laboratory interpretation must be paired with clinical correlation. The product source, route of exposure, and duration all influence whether propylene glycol becomes clinically relevant.

How the anion gap is calculated

The **anion gap** is a computed value used to help identify certain forms of **metabolic acidosis**. It represents the variation between observed cations and measured anions in the blood. The most common **anion gap formula** uses **serum sodium**, **serum chloride**, and **serum bicarbonate**.

A simplified version is:

anion gap = serum sodium – (serum chloride + serum bicarbonate)

An **anion gap calculator** performs this process and can assist clinicians and patients see whether the result falls in a normal span or suggests an acid-base disorder. Because the result relies on the measured electrolyte values, even small electrolyte changes can alter the number.

The anion gap is useful because unmeasured anions can build up in the blood during conditions such as lactic acidosis, ketoacidosis, or toxin ingestion. When interpreting the number, clinicians also consider **albumin**, since low albumin can reduce the measured gap and hide a clinically important issue. That is why the **albumin-corrected anion gap** is often more informative than the unadjusted result.

How propylene glycol can increase the anion gap

Indeed, propylene glycol can cause a **high anion gap** in the setting of **propylene glycol toxicity**. The mechanism is usually indirect rather than instant. After exposure, propylene glycol is converted into acidic compounds, including **organic acids**, which can contribute to **high anion gap metabolic acidosis**. In addition, the body may develop a concurrent **lactic acidosis**, which also increases the gap.

Another important clue is the **osmolar gap**. Propylene glycol itself elevates serum **osmolarity**, so early toxicity may present with an elevated osmolar gap before the anion gap rises. As metabolism advances, the parent compound decreases while acidic **metabolites** build up, shifting the pattern from isolated osmolar gap elevation to a combined osmolar gap and high anion gap presentation.

This progression is why the point in time of testing matters. A patient may initially have a high osmolar gap and later show progressive acidosis, elevated lactate, and a rising anion gap. In other words, propylene glycol can be

part of a combined laboratory pattern that changes over time.

Typical causes of high anion gap metabolic acidosis

Propylene glycol is only one possible cause of **metabolic acidosis**. A comprehensive **differential diagnosis** is essential whenever the anion gap is elevated. Typical causes include **ketoacidosis**, **lactic acidosis**, **renal failure**, and use of **toxic alcohols**. All of these can produce a similar lab pattern, but the underlying mechanism is different.

Ketoacidosis is often seen with diabetes, starvation, or prolonged vomiting, while lactic acidosis may occur with shock, sepsis, hypoperfusion, or certain drugs and toxins. Renal failure can raise the gap because the kidneys cannot clear acid effectively, allowing unmeasured acids to accumulate. Toxic alcohols, such as methanol or ethylene glycol, can also lead to an elevated anion gap and osmolar gap pattern.

This is why laboratory interpretation should never rely on a single number alone. The anion gap calculator can identify a worrisome result, but the final diagnosis depends on the clinical context, medication exposure, and additional testing.

Signs and signs of propylene glycol toxicity

The clinical signs of **propylene glycol toxicity** can be nonspecific at first. Patients may experience **altered mental status**, **hypotension**, and **tachypnea** as the pH disturbance worsens. Tachypnea often reflects respiratory compensation for acidosis. Some patients may also display signs of reduced perfusion or sedation, depending on the total exposure and the drugs involved.

A rising **serum lactate** can be an important clue, especially when the clinical picture suggests an unexplained acid-base problem. Elevated lactate does not confirm propylene glycol as the cause, but it strengthens concern when combined with medication exposure, abnormal serum chemistry, and a high anion gap. Because the symptoms overlap with other illnesses, **clinical correlation** is critical.

Severe cases can progress quickly, particularly when the patient has impaired clearance or multiple risk factors. A careful review of medications, infusion history, and serial labs is often the best way to recognize the problem.

Tests used to evaluate suspected poisoning

If propylene glycol contact is suspected, the evaluation usually includes **serum osmolality**, determination of the **osmolar gap**, a **blood gas**, and evaluation of **renal function**. These studies help determine whether the patient has a mixed toxic and metabolic picture.

Serum osmolality is compared with the calculated osmolality to identify an osmolar gap. A widened gap suggests unmeasured osmotically active substances, which may include propylene glycol or other toxic alcohols. The blood gas helps define the severity of the acid-base disorder and shows whether the patient has metabolic acidosis with respiratory compensation. Renal function testing is important because reduced clearance can worsen toxicity and prolong exposure.

Other lab studies often include serum lactate, electrolytes, and repeat chemistry panels. Serial testing can show whether the anion gap is rising or resolving after intervention. In many cases, the pattern of serum chemistry abnormalities provides the strongest evidence before specialized toxin levels are available.

How to understand an Anion Gap Calculator output

An **anion gap calculator** is valuable, but the output should consistently be interpreted in context. Initially, determine whether the value is in [Learn here](#) the **normal range** for the lab used. Normal ranges can shift somewhat depending on the analyzer and whether potassium is included in the formula. A value that is borderline in one lab may be definitely out of range in another.

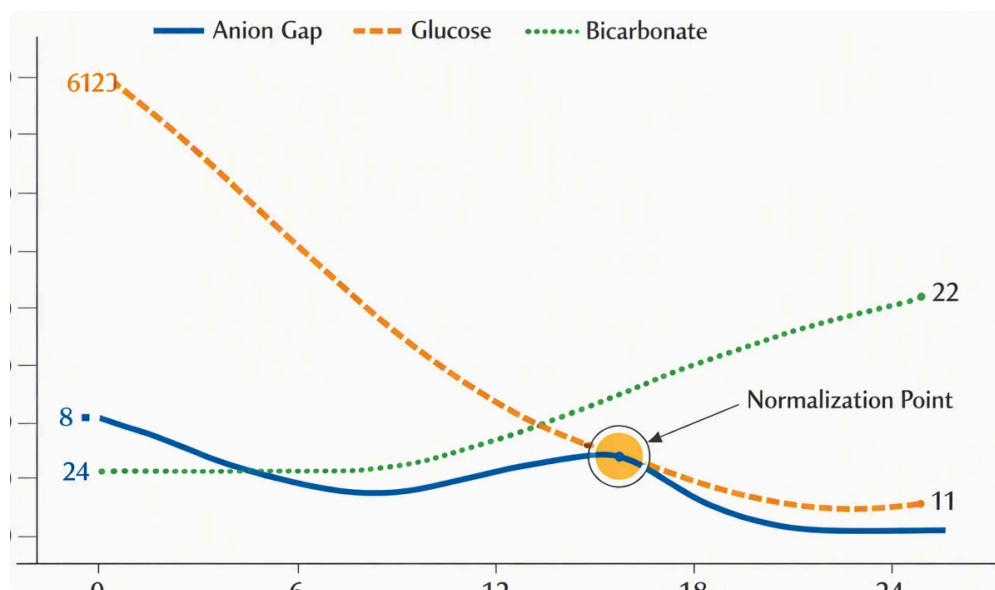
Afterward, evaluate the possibility of hypoalbuminemia. Because **albumin** is a major unmeasured anion, low albumin can hide a true **acidosis**. An **albumin-corrected anion gap** gives a better estimate of the underlying acid burden when albumin is reduced. This adjustment is most useful in critically ill patients, where albumin is often low.

In the end, ask whether the result fits the overall picture. A high anion gap with normal lactate and normal ketones may suggest a toxin, while a high gap with elevated lactate may indicate tissue hypoperfusion, sepsis, or propylene glycol toxicity. Good interpretation depends on **clinical context**, not just the number.

When propylene glycol contact becomes hazardous

The risk rises with **dose-dependent toxicity**, extended exposure, and reduced ability to clear the compound. This matters especially with some **benzodiazepines** and other **intravenous medications** that contain propylene glycol as a solvent. Ongoing or high-dose **infusion** can lead to accumulation over time.

Renal impairment raises risk because the kidneys have a key role in eliminating the compound and its byproducts. If renal clearance is reduced, propylene glycol and its metabolites may accumulate, contributing to osmolar gap elevation, lactic acidosis, and high anion gap metabolic acidosis.



Risk peaks when multiple factors stack together: high medication dose, prolonged infusion, critical illness, dehydration, and impaired kidney function. In that setting, monitoring should be more frequent and clinicians should stay highly alert for toxicity.

Treatment and management of suspected propylene glycol toxicity

The first step in management is **discontinuation** of the suspected source. Halting the offending medication or exposure can stop additional buildup. Depending on the severity of the case, the patient may also need **supportive care**, including fluid resuscitation, correction of electrolyte abnormalities, and treatment of acidosis.

Monitoring is important after the exposure is stopped. Serial serum chemistry testing, blood gas assessment, serum lactate, and renal function checks help show whether the acid-base disorder is getting better. If the patient has severe symptoms, rapidly worsening acidosis, or significant renal dysfunction, more aggressive treatment may be needed.

Hemodialysis can be considered in severe cases because it helps remove propylene glycol and correct associated metabolic derangements. It may be especially useful when there is significant acidosis, hemodynamic instability, or impaired renal clearance. The decision is based on the full clinical picture rather than the anion gap alone.

When to obtain urgent medical evaluation

Prompt evaluation is warranted if there are **emergency symptoms** such as progressive confusion, severe weakness, respiratory distress, collapse, or evidence of shock. A patient with suspected **toxic exposure** and a rapidly changing condition should not wait for regular follow-up.

Worrisome findings include **severe acidosis**, markedly low bicarbonate, persistent hypotension, or fast-rising lactate. These findings may signal a serious acid-base disorder that needs urgent treatment. If propylene glycol exposure is possible, a prompt **medical assessment** can clarify whether the patient needs hospital monitoring, medication changes, or hemodialysis.

As the disorder can overlap with other causes of high anion gap metabolic acidosis, clinicians should evaluate the entire presentation early. Rapid assessment of medication exposure, serum osmolality, blood gas results, and renal function can prevent delays in care.

Frequently asked questions about propylene glycol and the anion gap

Can propylene glycol cause a high anion gap?

It can. Propylene glycol can cause a **high anion gap**, especially when exposure is marked or prolonged. It may first raise the **osmolar gap** and then, as it is metabolized into acidic **metabolites**, contribute to **high anion gap metabolic acidosis** and **lactic acidosis**.

What is the difference between an anion gap and an osmolar gap?

The **anion gap** represents unmeasured charged particles and helps identify causes of **metabolic acidosis**. The **osmolar gap** shows the difference between measured and calculated **osmolality** and suggests unmeasured dissolved substances, such as propylene glycol or other **toxic alcohols**. Both are helpful, but they serve different questions.

Which medications contain propylene glycol?

Propylene glycol can be found in some **intravenous medications**, including certain **benzodiazepines**, sedatives, and other formulations that use it as a solvent. It may also appear in some **oral products** and **topical products**. The exact formulation depends on the drug and manufacturer, so the medication list should be checked carefully.

What indications may indicate propylene glycol toxicity?

Potential manifestations include **altered mental status**, **low blood pressure**, **increased respiratory rate**, and signs of increasing acidosis. An elevated **serum lactate** may also be present. As these findings are nonspecific,

they must be assessed with history of exposure, lab values, and total clinical correlation.

How is propylene glycol toxicity treated?

Management usually starts with **discontinuation** of the source and **supportive care**. Clinicians observe the patient closely with repeat labs, including blood gas, serum chemistry, and renal function. In severe cases, **hemodialysis** may be used to help remove the toxin and improve severe acidosis.