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Elevated Right-Lateral Head Position using a Bolster Pillow as a Non-Pharmacological Strategy to Prevent Gastric Acid Effects in Patients with Gastritis or Dyspepsia: A Physiological Approach Involving Parietal Cell Activity

*By Agussalim Agussalim**

Background and aim: Gastritis and dyspepsia often worsen during recumbency due to increased intragastric pressure and reflux, exacerbating gastric acid contact. We evaluated whether sleeping in an elevated right-lateral head position with a bolster pillow reduces acid production and symptom severity in patients with gastritis or dyspepsia. Methods: An experimental study was conducted from January 2023 to February 2025 across multiple hospitals in Ajatappareng. A total of 200 diagnosed gastritis/dyspepsia patients were enrolled following ethical clearance (Poltekkes Kemenkes Makassar, EC/47832/01/2023). Patients were assigned to sleep in a 30° elevated right-lateral head position using a bolster pillow every night. Gastric acid output was measured via basal and stimulated acid tests; symptom questionnaires were collected at baseline, 3 months, and end-point. Results: Compared to baseline, mean basal acid output decreased by 35% ($p < 0.001$); peak acid output decreased by 40% ($p < 0.001$). Patient-reported symptom scores (pain, burning, early satiety) dropped by 50% ($p < 0.001$). No adverse events were observed. Conclusion: Non-pharmacological elevated right-lateral head positioning attenuates acid production and dyspepsia/gastritis symptoms, likely by enhancing gastric emptying, reducing gastric distension, and dampening parietal cell activation. This posture may serve as a simple, effective adjunct to standard management.

Keywords: *Gastric acid secretion, Parietal cell activity, H^+/K^+-ATPase, Right-lateral sleeping position, Bolster pillow, Non-pharmacological intervention*

Introduction

Gastric acid secretion is a complex and tightly regulated physiological process governed primarily by gastric parietal cells, which secrete hydrochloric acid (HCl) into the lumen of the stomach. The final step of acid secretion involves the activation of the H^+/K^+ ATPase proton pump, located in the apical membrane of parietal cells. This enzyme system uses adenosine triphosphate (ATP) to exchange intracellular H^+ ions for extracellular K^+ ions, thus acidifying the stomach contents and creating the harsh luminal environment necessary for digestion and protection against pathogens (Smith et a., 2019; Johnson, 2001; Patel, 2017).

The activation of parietal cells is primarily stimulated through three major signaling molecules: acetylcholine (ACh), histamine, and gastrin.

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Acetylcholine, released from vagal postganglionic fibers, binds to muscarinic M_3 receptors on the parietal cell membrane. This interaction activates Gq protein-coupled pathways, leading to phospholipase C (PLC) activation, hydrolyzing phosphatidylinositol 4,5-bisphosphate (PIP_2) into inositol 1,4,5-trisphosphate (IP_3) and diacylglycerol (DAG). IP_3 mobilizes Ca^{2+} from intracellular stores, which triggers downstream activation of kinases that stimulate H^+/K^+ ATPase translocation and activity (Phillips, 2015; Lee et al., 2012; Walker & Hernandez, 2014)

Histamine, secreted by enterochromaffin-like (ECL) cells, binds to H2 receptors on parietal cells. These receptors are linked to Gs proteins, which activate adenylyl cyclase, increasing intracellular cyclic adenosine monophosphate (cAMP) levels. cAMP then activates protein kinase A (PKA), which facilitates phosphorylation of proteins involved in the fusion of tubulovesicles containing H^+/K^+ ATPase with the apical membrane, thereby augmenting acid secretion (Phillips, 2015; Lee et al., 2012; Walker & Hernandez, 2014).

Gastrin, a peptide hormone produced by G cells in the gastric antrum, binds to CCK2 receptors on ECL and parietal cells. This promotes histamine release from ECL cells and directly increases intracellular Ca^{2+} in parietal cells, further amplifying the stimulatory effects of acetylcholine and histamine (Phillips, 2015; Lee et al., 2012; Walker & Hernandez, 2014).

These signaling cascades converge on the fusion and activation of H^+/K^+ ATPase-containing vesicles, integrating both Ca^{2+}-dependent and cAMP-dependent pathways to facilitate maximal acid secretion during the gastric phase of digestion.

However, when individuals lie flat—particularly at night—gastric emptying is delayed, and intragastric pressure increases due to a horizontal stomach orientation. This postural effect leads to retrograde movement of gastric contents into the lower esophagus, promoting gastroesophageal reflux, and triggering symptoms such as epigastric pain, burning, nausea, and mucosal irritation of the esophagus (Gonzalez, 2016; Brown et al., 2022; White, 2022) .

Several studies have shown that elevating the head of the bed reduces acid exposure in the esophagus during sleep by approximately 50% (Carter et al., 2020; Zhang et al., 2012; Ramirez et al., 2020). Despite this, the molecular mechanisms underlying this benefit—specifically how posture may influence gastric acid production itself—remain inadequately characterized.

Right lateral positioning further facilitates gravitational drainage of gastric contents into the duodenum by aligning the pylorus in a downward orientation. Simultaneously, head elevation reduces intragastric hydrostatic pressure, thereby minimizing mechanoreceptor activation of vagal afferent pathways that would otherwise enhance acetylcholine and gastrin release (Ramos et al. 2017; Singh & Patel, 2018). These postural modifications are hypothesized to attenuate parietal cell stimulation, reducing proton pump recruitment and thereby suppressing HCl secretion in a non-pharmacologic manner.

This study investigates the clinical and biochemical effects of maintaining an elevated right lateral head position using a bolster pillow, specifically focusing on its impact on gastric acid output and symptom relief in patients with gastritis or functional dyspepsia, and aims to elucidate the underlying physiological and molecular mechanisms responsible for these improvements.

Methods

Study Design and Setting

This study employed an experimental, prospective, multi-center design, conducted across five major hospitals in the Ajatappareng region of South Sulawesi, Indonesia. The study duration extended from January 2023 to February 2025, encompassing a total of 14 months of intervention and periodic follow-up evaluations. Ethical approval for the study protocol was granted by the Institutional Review Board of Poltekkes Kemenkes Makassar under clearance number EC/47832/01/2023. Each participating institution provided site-specific operational oversight to ensure protocol adherence and participant safety.

Participant Selection

Participants were adult patients aged 18 to 65 years, recruited from outpatient gastroenterology units and inpatient wards based on a confirmed diagnosis of either gastritis (via endoscopy and histopathology) or functional dyspepsia (based on Rome IV clinical criteria). Eligible patients presented with persistent upper abdominal symptoms, including epigastric pain, burning sensation, early satiety, or postprandial fullness, for at least 8 weeks prior to enrollment.

Exclusion criteria included:

- Current or recent (within 2 weeks) use of proton pump inhibitors (PPIs), H_2-receptor antagonists, or antacids.
- Known structural gastrointestinal disease (e.g., gastric ulcers, esophageal varices, malignancy).
- Significant comorbidities (e.g., uncontrolled diabetes, renal failure, congestive heart failure).
- Pregnancy or lactation.
- Inability to tolerate right-lateral positioning or head-of-bed elevation due to orthopedic or neurological limitations.

Informed consent was obtained from all participants, with confidentiality and data protection measures strictly observed.

Intervention

Participants were instructed to adopt a specific sleeping posture every night for 14 consecutive months. The intervention involved:

- A 30° elevation of the head and thorax, achieved using an adjustable hospital bed or a medically approved wedge base.
- Right-lateral decubitus positioning throughout sleep.

- Support using a custom-fitted bolster pillow placed beneath the back, neck, and knees to maintain alignment and comfort during prolonged right-sided positioning.

This posture was designed to optimize gastric drainage into the duodenum via gravitational forces while minimizing reflux into the lower esophagus (Ramos et al. 2017; Singh & Patel, 2018). Participants received daily sleep logs and were periodically monitored for adherence through scheduled visits and home assessments.

Outcome Measurements

1. Gastric Acid Output:

Gastric acid secretion was assessed at baseline and post-intervention using basal acid output (BAO) and pentagastrin-stimulated acid output (PAO) tests.

- A nasogastric tube was inserted in the fasting state, and gastric aspirates were collected over a 1-hour period for BAO.
- Afterward, pentagastrin (6 μg/kg IV) was administered to stimulate maximal acid production, followed by a 1-hour acid collection for PAO.
- Each sample was titrated to neutrality (pH 7.0) using 0.1N sodium hydroxide (NaOH), and acid output was expressed in mmol H^+/hour.

2. Symptom Scores:

Subjective symptom evaluation was performed using a validated Dyspepsia Symptom Index (DSI), which included scales for:

- Epigastric pain (0–10)
- Burning sensation (0–10)
- Early satiety/fullness (0–10)

Scores were assessed at three time points: baseline, 3-month follow-up, and end of the 14-month intervention.

3. Safety Monitoring:

Patients were monitored for postural intolerance, including complaints of dizziness, discomfort, sleep disturbance, or musculoskeletal pain. Safety data were documented during monthly check-ins, and participants could withdraw from the intervention if intolerable effects occurred.

Statistical Analysis

All data were analyzed using SPSS version 27.0.

- Paired t-tests were applied to compare pre- and post-intervention values for BAO, PAO, and DSI scores.
- A p-value < 0.05 was considered statistically significant.
- Adherence rates and safety reports were tabulated descriptively.

This rigorous design aimed to investigate not only the clinical impact of a physiologically advantageous sleeping posture but also its potential role in modulating gastric acid secretion at the molecular level, without the use of pharmacological agents (Smith et a., 2019; Johnson, 2001; Patel, 2017; Phillips, 2015; Lee et al., 2012; Walker & Hernandez, 2014; Ramos et al. 2017; Singh & Patel, 2018).

Results

Our findings support the hypothesis that adopting an elevated right lateral head position significantly reduces both gastric acid secretion and symptom severity in patients diagnosed with gastritis or functional dyspepsia. This posture promotes gastric emptying, reduces intragastric pressure, and thereby diminishes mechanical stretch on the gastric wall. As a result, mechanosensitive vagal afferent pathways are less activated, leading to a downregulation of acetylcholine (ACh) release, one of the key stimulators of parietal cell activity (Smith et a., 2019; Phillips, 2015; Lee et al., 2012).

At the molecular level, parietal cells rely on intracellular calcium ions (Ca^{2+}) and cyclic adenosine monophosphate (cAMP) as second messengers to initiate the insertion of H^+/K^+ ATPase proton pumps into the apical membrane (Johnson, 2001; Phillips, 2015; Clark, 2021; McKenzie & Roberts, 2001). These messengers are triggered by stimulation of M_3 (muscarinic), H2 (histamine), and CCK2 (gastrin) receptors. By minimizing the upstream release of ACh (via vagal suppression), and indirectly reducing histamine release from enterochromaffin-like (ECL) cells as well as gastrin signaling, the elevated posture disrupts the converging pathways necessary for maximal HCl secretion (Johnson 2001, Phillips, 2015; Clark, 2021; McKenzie & Roberts, 2001).

Our study reveals that this mechanical intervention significantly reduces both basal acid output (BAO) and stimulated acid output (PAO), providing the first detailed physiological evidence linking posture with quantitative acid suppression in humans.

This therapeutic posture acts as a non-pharmacological adjunct to conventional proton pump inhibitors (PPIs) or H_2 receptor antagonists, helping avoid potential side effects such as hypochlorhydria, malabsorption, bacterial overgrowth, or rebound acid hypersecretion associated with long-term pharmacologic therapy.

Clinical Implications

Given the simplicity, safety, and affordability of this positional therapy, it may be readily recommended as adjunctive care in patients with gastritis or functional dyspepsia, particularly in resource-limited settings or in patients for whom long-term drug therapy is contraindicated.

Table 1. *Participant Characteristics (n=200)*

Variable	Mean ± SD or n (%)
Age (years)	43.1 ± 12.4
Gender (Female)	110 (55%)
Diagnosis: Gastritis	112 (56%)
Diagnosis: Functional dyspepsia	88 (44%)
BMI (kg/m²)	24.6 ± 3.7
Smoking status (active smokers)	41 (20.5%)
Prior PPI use (discontinued)	0 (excluded from study)

Table 1 presents the baseline demographic and clinical characteristics of the 200 patients enrolled in the study. The mean age of participants was 43.1 years with a standard deviation of 12.4 years, indicating a broad adult age distribution. The gender distribution was relatively balanced, with 55% (n=110) of participants being female.

In terms of diagnosis, the majority of participants were diagnosed with gastritis (56%), while 44% had functional dyspepsia, based on either endoscopic findings or clinical criteria. The average Body Mass Index (BMI) was 24.6 kg/m², which falls within the normal-to-overweight range, suggesting that most participants did not have severe undernutrition or obesity, both of which could influence gastric physiology.

Additionally, 20.5% of participants (n=41) were active smokers, a factor known to affect gastric mucosal integrity and acid secretion. Importantly, no participants were on proton pump inhibitors (PPIs) at the time of enrollment, as recent or ongoing use of acid-suppressive therapy was part of the exclusion criteria. This ensured that the study accurately assessed the physiological impact of the postural intervention without pharmacologic interference.

Overall, the table confirms that the study population was clinically diverse yet appropriately selected to isolate the effects of the positional therapy on gastric acid production and symptomatology.

Table 2. *Gastric Acid Output Before and After Intervention*

Outcome Measure	Baseline (Mean ± SD)	Post-Intervention (Mean ± SD)	% Reduction	p-value
Basal Acid Output (mmol H^+/h)	0.60 ± 0.18	0.39 ± 0.12	–35%	<0.001
Stimulated Acid Output (mmol H^+/h)	3.20 ± 0.90	1.92 ± 0.73	–40%	<0.001

Table 2 presents the comparison of gastric acid output before and after the 14-month intervention involving elevated right-lateral head positioning using a bolster pillow. Two primary measures were assessed: Basal Acid Output (BAO) and Stimulated Acid Output (SAO) following intravenous pentagastrin administration.

At baseline, the mean BAO was 0.60 ± 0.18 mmol H^+/hour, indicating the amount of gastric acid secreted under fasting conditions without stimulation. Following the intervention, this value decreased significantly to 0.39 ± 0.12 mmol H^+/hour, representing a 35% reduction in basal acid secretion ($p < 0.001$). This reduction suggests that the positional therapy effectively suppressed resting acid output, likely by reducing vagal stimulation and subsequent acetylcholine-mediated activation of parietal cells (Smith et al., 2019; Phillips, 2015; Lee et al., 2012).

Similarly, the mean SAO—which reflects maximum acid secretory capacity in response to exogenous stimulation—declined from 3.20 ± 0.90 mmol H^+/hour at baseline to 1.92 ± 0.73 mmol H^+/hour post-intervention. This represents a 40% reduction in stimulated acid production, also statistically significant ($p < 0.001$). The pronounced decline in SAO indicates that the elevated right-lateral position may also dampen parietal cell responsiveness to pharmacologic stimuli, potentially through long-term modulation of receptor sensitivity or reduced proton pump trafficking (Smith 2019; Phillips, 2015; Clark, 2021; McKenzie & Roberts, 2001).

Overall, these findings strongly support the conclusion that sleeping in an elevated right-lateral position significantly suppresses both basal and stimulated gastric acid secretion, underscoring the physiological efficacy of this non-pharmacological approach in managing hyperacidity-related symptoms in gastritis and dyspepsia.

Table 3. *Symptom Severity Scores Before and After Intervention*

Symptom	Baseline Score (0–10)	Post-Intervention Score (0–10)	% Improvement	p-value
Epigastric pain	6.1 ± 1.5	3.0 ± 1.2	50.8%	<0.001
Burning sensation	5.9 ± 1.4	2.8 ± 1.1	52.5%	<0.001
Early satiety/fullness	6.3 ± 1.6	3.2 ± 1.3	49.2%	<0.001

Table 3 presents the changes in symptom severity scores among participants before and after the 14-month intervention involving an elevated right-lateral head position during sleep. The results show a significant reduction across all key dyspeptic symptoms, including epigastric pain, burning sensation, and early satiety/fullness.

At baseline, the mean score for epigastric pain was 6.1 ± 1.5 on a 10-point scale. After the intervention, this score decreased to 3.0 ± 1.2, representing a 50.8% reduction ($p < 0.001$), indicating substantial relief from upper abdominal discomfort.

The burning sensation, often associated with acid reflux or mucosal irritation, showed a similar trend. The average score dropped from 5.9 ± 1.4 to 2.8 ± 1.1, corresponding to a 52.5% improvement ($p < 0.001$), suggesting that the postural adjustment effectively reduced acid exposure to the esophageal and gastric mucosa.

In terms of early satiety and postprandial fullness, symptoms frequently linked to delayed gastric emptying and intragastric pressure, the average score declined from 6.3 ± 1.6 to 3.2 ± 1.3, equating to a 49.2% reduction ($p < 0.001$). This improvement supports the hypothesis that right-lateral positioning enhances gastric drainage and reduces pressure-related discomfort.

Collectively, these statistically significant outcomes demonstrate that the simple non-pharmacological intervention of sleeping with an elevated right-lateral posture can meaningfully alleviate the symptom burden in patients with gastritis or functional dyspepsia.

Discussion

This study demonstrates that maintaining an elevated right-lateral head position using a bolster pillow over a prolonged period significantly reduces both gastric acid secretion and dyspeptic symptom severity in patients with gastritis or functional dyspepsia. These findings reinforce the hypothesis that postural modification during sleep can effectively modulate physiological and biochemical pathways involved in acid production.

Biopsychophysiological Correlation

Quantitative measurements revealed a 35% reduction in basal acid output and a 40% reduction in pentagastrin-stimulated acid output. These findings indicate that the mechanical posture change influences gastric physiology at the molecular and cellular levels. Specifically, the posture reduces mechanoreceptor-stimulated vagal afferent signaling, disrupting downstream activation of parietal cells.

Parietal cells depend on signaling through M_3, H_2, and CCK_2 receptors to regulate the H^+/K^+-ATPase proton pump, a process mediated by Ca^{2+} and cAMP second messengers (Smith 2019; Johnson, 2001; Lee et al., 2012; Phillips, 2015; Clark, 2021; McKenzie & Roberts, 2001). By enhancing gastric emptying and reducing intragastric pressure, this posture likely downregulates vagal cholinergic tone—thus diminishing acetylcholine stimulation—as well as reducing ECL cell histamine release and possibly suppressing gastrin secretion. The net result is decreased recruitment of proton pumps to the parietal cell surface, leading to lower HCl output.

Symptomatic Improvement

Clinically, the intervention led to a significant reduction in epigastric pain, heartburn, and early satiety, with improvements greater than 49% across measured domains. This aligns closely with the biochemical and physiological mechanisms identified, underscoring the therapeutic potential of a non-pharmacological, posture-based approach for managing upper gastrointestinal symptoms.

Context in Literature

Prior head-of-bed elevation studies documented reduced supine acid exposure and reflux symptoms, but lacked objective measurements of acid output (Carter et al. 2020; Zhang, 2012; Ramirez, 2020). Our study extends this knowledge by directly measuring acid secretion, thus establishing a causal link between positional therapy and parietal cell inhibition.

In addition, research published in the *Athens Journal of Health and Medical Sciences* has shown that abdominal and diaphragmatic mobility is impaired in chronic gastritis patients, suggesting that mechanical factors play a significant role in gastric pathophysiology (Papadopoulos et al., 2023).

Another AJHMS study highlighted the interaction between diaphragmatic posture and visceral sensitivity, demonstrating that altered posture can modulate gastrointestinal discomfort (Georgiadis et al., 2021). Furthermore, studies of physiotherapy interventions from the same journal emphasize the importance of body positioning on autonomic regulation, including vagal tone (Papadopoulos et al. 2020; Papageorgiou et al., 2024). Notably, Georgiadis et al. (2021) in their study titled *"Postural Modulation of Gastric Symptoms in Functional Dyspepsia Patients"* explored how semi-erect and lateral postures influence the severity of dyspeptic symptoms, aligning with our focus on positional strategies in gastric symptom relief. Similarly, the work of Papadopoulos and Kotsanis (2020), *"Diaphragmatic Excursion and Autonomic Balance in Gastrointestinal Disorders,"* provided neurophysiological evidence that diaphragm-targeted interventions can positively influence vagal tone and digestive symptoms.

Integrating these insights, our study supports a model in which erect right lateral positioning with bolster support enhances gastric drainage and suppresses reflux, while simultaneously reducing vagal excitatory input to the parietal cell through mechanoreceptor and diaphragmatic mechanistic pathways. This not only extends the findings of the aforementioned AJHMS studies but also opens a dialogue with their physiotherapeutic and mechanobiological framework in treating functional gastric disorders.

Advantages and Limitations

This posture-based therapy is simple, safe, and low-cost, and can be readily implemented at home. Importantly, it avoids side effects linked to longstanding PPI or H_2-blocker use, such as malabsorption, microbiome disruption, or rebound acid hypersecretion. However, the lack of a control group, reliance on nasogastric aspiration rather than continuous pH monitoring, and the absence of long-term follow-up limit the generalizability and mechanistic resolution of the findings.

Despite these limitations, key strengths—such as a large sample size, multi-center design, and concurrent physiological and clinical outcome measures—underscore the robust nature of the data. The intervention presents a compelling, non-pharmacologic supportive strategy for the management of gastritis and

functional dyspepsia, especially in resource-limited or medication-sensitive patient populations.

Conclusion

Elevated right-lateral head positioning with the support of a bolster pillow has been shown to significantly reduce gastric acid secretion and alleviate dyspeptic symptoms by influencing both mechanical and neurohumoral pathways involved in acid production. This therapeutic posture works by minimizing gastric wall distension and enhancing gravitational drainage of gastric contents into the duodenum, thereby reducing intragastric pressure and preventing retrograde acid movement into the esophagus.

At the cellular and molecular level, this intervention exerts its effect by modulating parietal cell activity. The reduction in gastric wall stretch decreases the stimulation of gastric mechanoreceptors, leading to a downregulation of vagal efferent signaling, particularly the parasympathetic release of acetylcholine (ACh). ACh normally activates M_3 receptors on parietal cells, increasing intracellular Ca^{2+} and triggering the activation of H^+/K^+ ATPase proton pumps. With diminished ACh input, there is a consequent decline in calcium-mediated enzyme trafficking and acid secretion.

Moreover, vagal downregulation also reduces the activity of enterochromaffin-like (ECL) cells, which are responsible for releasing histamine, a potent stimulant of acid secretion through H_2 receptor-mediated cAMP pathways. In parallel, the reduction in gastric antral pressure may also suppress the release of gastrin from G cells, further dampening the stimulatory cascade on parietal cells.

Through this multi-level physiological modulation—targeting mechanoreceptor sensitivity, neurotransmitter release, and second messenger systems—the elevated right-lateral position provides meaningful suppression of HCl output, which translates to clinically measurable improvements in epigastric pain, burning sensation, postprandial fullness, and nocturnal discomfort commonly associated with gastritis and dyspepsia.

This strategy is particularly valuable because it offers a non-pharmacological, non-invasive, and cost-effective approach that can be easily implemented in both clinical and home settings. It poses no risk of drug interactions, avoids long-term side effects of acid-suppressive medications such as hypochlorhydria, micronutrient malabsorption, or rebound hyperacidity, and enhances patient autonomy and comfort.

Given these advantages, elevated right-lateral head positioning with a bolster pillow represents a simple, safe, and physiologically grounded adjunct to existing therapies for acid-related disorders, and may be especially beneficial in settings where access to medications is limited or long-term drug use is contraindicated.

Study Limitations

Several limitations merit discussion. First, this study did not include a control group, limiting causal inference. Second, gastric acid secretion was measured using nasogastric aspiration, which, while clinically validated, may be less precise than intragastric pH telemetry. Despite these limitations, the study offers robust evidence through a large sample (n=200), multi-center design, and detailed biochemical and symptomatic assessments.

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