Scholars Journal of Medical Case Reports

OPEN ACCESS

Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

Medical Science

Air Swallowing and Breathing Exercises Reduce the Severity of Acute Gastroesophageal Reflux Symptoms and Gives a Clue into the Role of Oxygen in Digestion: Case Report with Extended Discussion

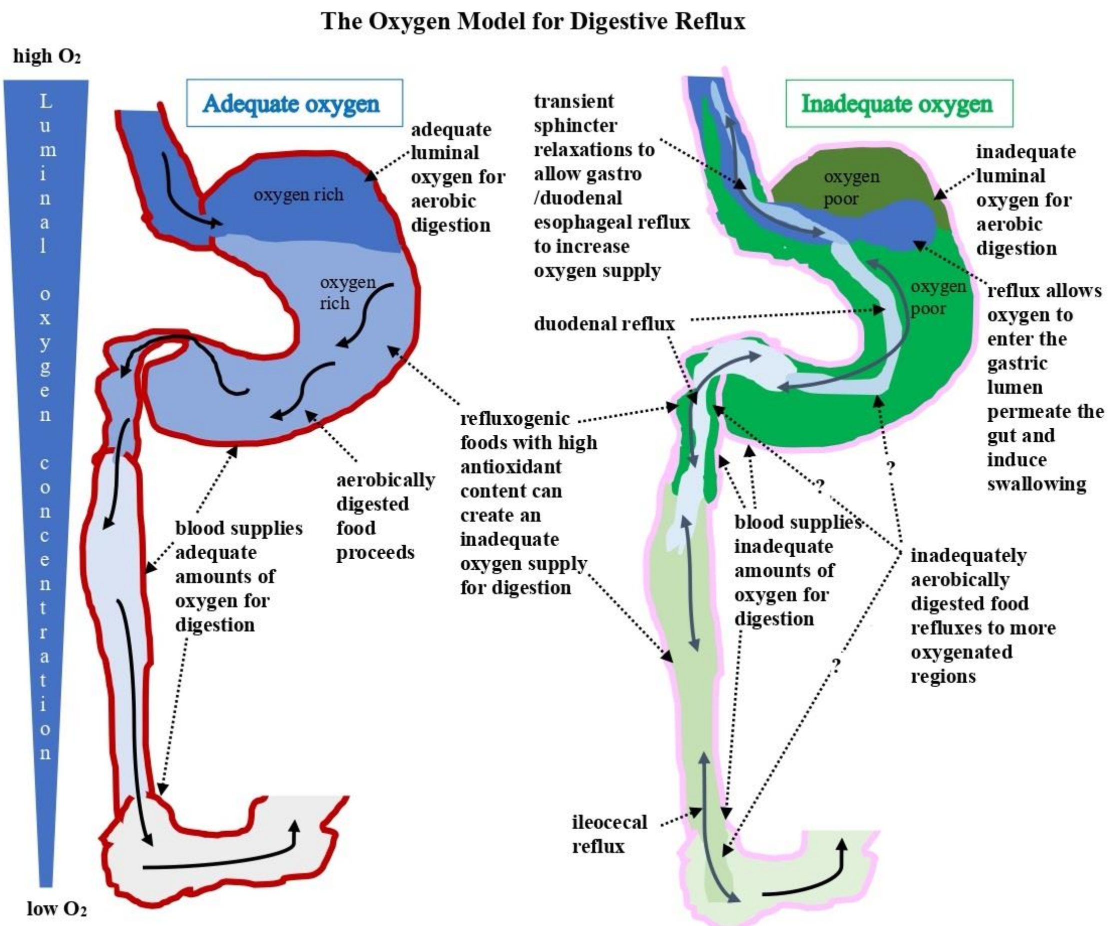
Thomas J. Hurr[1*]

[1]South Australian Reflux Research Unit, Adelaide, South Australia, Australia,15 Lanor Avenue, Millswood. Adelaide, South Australia, 5034. Australia

DOI: https://doi.org/10.36347/sjmcr.2025.v13i11.023 | **Received:** 19.09.2025 | **Accepted:** 06.11.2025 | **Published:** 10.11.2025

***Corresponding author:** Thomas J. Hurr
South Australian Reflux Research Unit, Adelaide, South Australia, Australia,15 Lanor Avenue, Millswood. Adelaide, South Australia, 5034. Australia

Abstract **Case Report**



Graphical Abstract:

The hypothesised oxygen model for digestive reflux. Reflux of digesting food to the more oxygenated oesophagus /pharynx regions is triggered by refluxogenic foods like coffee and orange juice which are high in antioxidants that react with oxygen in the lumen to creating an inadequate supply necessary for aerobic digestion, resulting in gastroesophageal reflux. Several air swallowing and deep breathing events over 30 seconds, when reflux symptoms occur, increase oxygen supply and the probability that food can be aerobically digested as required, potentially reducing the number of reflux events and reflux symptoms, but excessive air swallowing can cause indigestion, bloating and burping.

A case is reported where a patient, after using a tooth whitening gel containing the active ingredient hydrogen peroxide over 2 days, found the expected onset of postprandial gastroesophageal reflux symptoms (RS) did not occur. Oxygen in air dissolves in water contained in the gut lumen to form hydrogen peroxide and so the role of oxygen, rather than potentially toxic hydrogen peroxide, was investigated as a treatment for RS. Air swallowing can provide oxygen to the gut lumen and breathing exercises can supply oxygen from the blood to the gut epithelium. Air swallowing and breathing exercises (ASBE) were performed over 34 days and it was found a single ASBE session (3-8 ASBE over 30 seconds) brought temporary relief from RS over 1-2 hours. Up to 3 sessions of ASBE were required and resolved RS over 74% of days reducing the need to take antacid tablets. To understand the impact oxygen could have on RS, the oxidation potential for oxygen, calculated as mmol electrons /100 ml (2-3 breaths) of swallowed air was calculated and compared to the reported values for the antioxidant content of various foods. It was found that 100 ml of swallowed air has the potential to oxidize 1 serve of coffee, red wine or orange juice, known refluxogenic or trigger foods that can cause RS. Based on the finding that ASBE can reduce RS and a brief review of the role oxygen has in digestion, a hypothetical oxygen model for digestive reflux was proposed. For the model, the reflux of digesting food to the more oxygenated oesophageal regions is triggered by refluxogenic foods high in antioxidants that react with oxygen in the lumen to create an inadequate supply necessary for aerobic digestion, resulting in gastroesophageal reflux. As the ASBE did not resolve all RS with ≈ 26% remaining unresolved, oxygen supply was considered only a part of the complex digestive reflux mechanism.

Keywords: air swallow, antacids, antioxidants, coffee, gastroesophageal reflux, GERD, hypoxia, IBD, oxygen, redox.

1.0 INTRODUCTION

A patient, after using a tooth whitening gel containing the active ingredient hydrogen peroxide over 2 days, reported the expected onset of postprandial gastroesophageal reflux symptoms (RS) did not occur. This observation, together with the knowledge that oxygen from air can dissolve in water contained in the gut to form hydrogen peroxide, initiated the investigation into the potential benefits of air swallowing and breathing exercises (ASBE). Oxygen supply is likely the most central part of food digestion and it is not surprising that to digest an average meal, to produce molecules suitable for absorption and to maintain a healthy microbiome, a significant amount of oxygen, including luminal oxygen, is required. A half equation for oxygen and the formation of hydrogen peroxide on dissolving in water, allow a calculation of the maximum number of electrons able to be accepted during oxidation, when a 100 ml of air is swallowed. These values can be compared to reported values for the redox active antioxidant in food [1]. A brief review of the literature for air and oxygens association with gastroesophageal reflux disease (GERD) and other regions of the digestive system, give a clue to its central role. A hypothetical model is proposed suggesting that a diet consisting of foods requiring significant amounts of oxygen for digestion or containing high concentrations of antioxidants, can create a lack of luminal oxygen, resulting in the reflux of digesting food to regions of higher oxygen content and induce swallowing to increase oxygen supply and prevent hypoxia or anoxia.

2.0 CASE REPORT

A case is presented of a 65 year old male with over a ten year history of postprandial RS that usually occur most nights an hour or more after a meal, often becoming troublesome when lying down to sleep. No other diseases were reported or prescription medication currently used. After using a tooth whitening gel containing hydrogen peroxide (7.5%) in retainers covering the teeth for 60 minutes over 2 days, the expected onset of RS did not occur. Reflux symptoms were reported to be a feeling or awareness that stomach contents could reflux into the throat and were typically managed using antacid tablets containing 300mg calcium carbonate, taken before sleep. Consumption of refluxogenic foods like coffee and alcohol, 1-2 hours before sleep and large evening meals were minimised but not avoided altogether.

Awareness that hydrogen peroxide contained oxygen compounds that could also be obtained from ASBE and may be beneficial for digestion, ASBE was typically undertaken 1-2 hours after a meal when RS occurred. Symptoms would be scored nightly over 50 days as no symptoms 0, mild 1, moderate 2 and severe symptom 3. More complex RS scoring methods have been developed but may not necessarily be more accurate [2].

An ASBE involved breathing air to fill the mouth and deliberately swallowing the air and included several deep breaths over ≈ 30 seconds. Up to 3-8 ASBE were done at any one time forming a ASBE session, performed when RS occurred. The first ASBE session often only brought temporary relief and had to be repeated, with up to 3 ASBE sessions required over 1-4 hours. Any discomfort resulting from ASBE or for whatever reason, the process would be discontinued and antacid tablets could also be used as required to manage RS. The patient reported no significant side effects from ASBE but noted occasional burping and occasional mild transient indigestion immediately after ASBE of short duration.

3.0 RESULTS

Air swallowing and breathing exercises

Over the 50 days, 10/50 days had no RS and for 6/50 days antacids were used in preference to ASBE due to going to sleep soon after a meal with no time available to do the 3 ASBE sessions usually required.

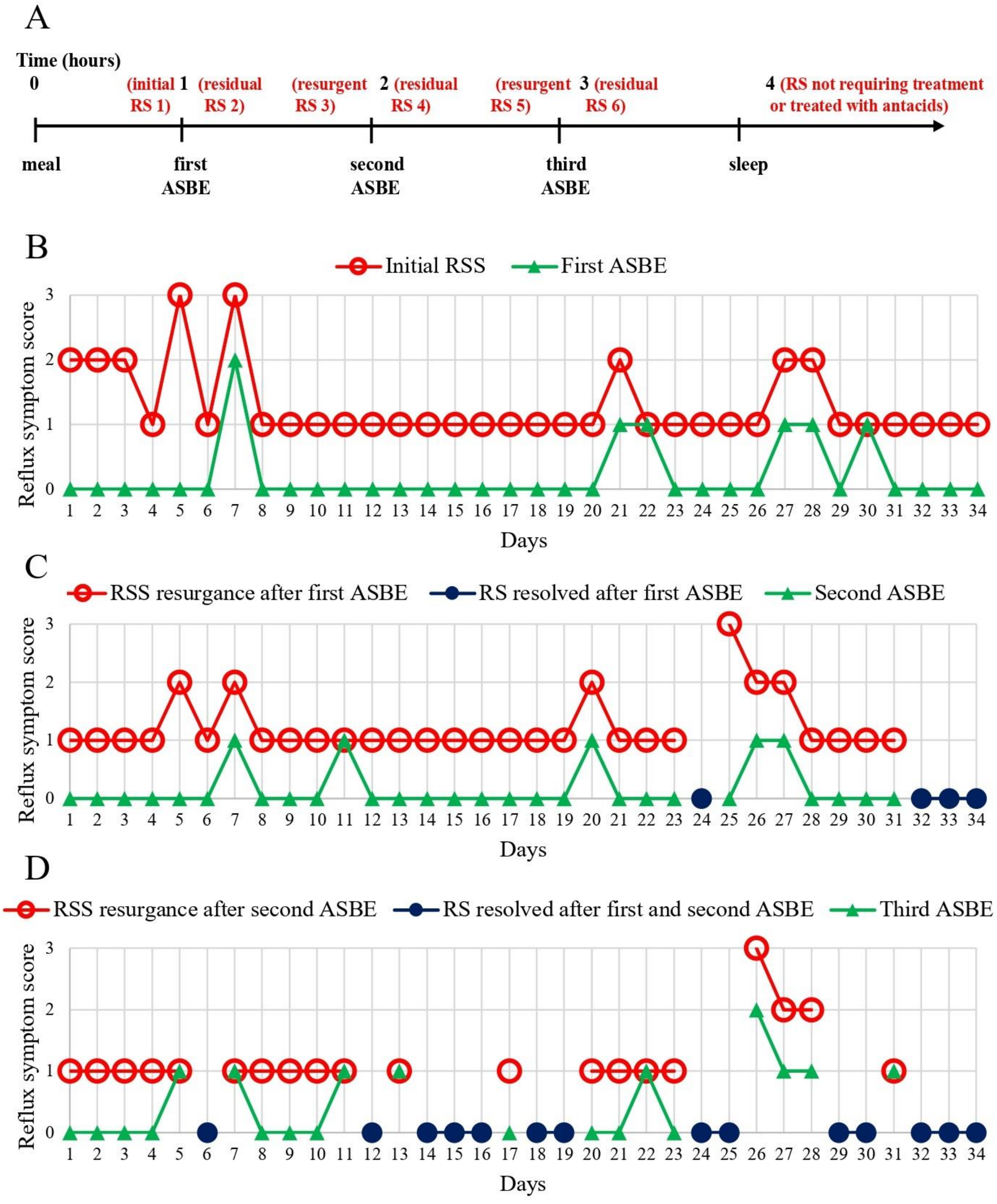


Figure 1: Changes in the RS score over 34 days from ASBE sessions: (A) typical timeline between meals with an initial RS score recorded before 3 ASBE sessions followed by an additional 5 further RS scores, totaling up to 6 RS scores /evening meal, (RS 1 – RS 6) before sleep, (B) the first ASBE session brought temporary relief to 28/34 days with RS persisting for 6/34 days (days 7,11,20,26,27), (C) over the following ≈ 1-2 hours, RS resolved for 4/34 days (days 24,32,33,34) but returned for 30/34 days requiring a second ASBE session, bringing temporary relief to RS for 25/30 days with RS persisting for 5/30 days (days 7,11,20,26,27), (D) again over a further ≈ 1-2 hours, RS returned for 20/34 days requiring a third ASBE session, fully resolving RS for 11/20 days with RS persisting for 9/20 days (days 5,7,11,13,22,26,27,28,31), overall ASBE sessions resolved 25/34 ≈ 74% of RS events over ≈ 1-4 hours with 9/34 remaining unresolved

For 34 days, up to 3 ASBE sessions were undertaken to manage RS with up to 6 RS scores recorded, Fig. 1A.

- The first ASBE session occurred ≈ 1-2 hours after the meal when RS emerged, bringing temporary relief to RS for 28/34 days, reducing the severity of RS for 4/34 days (days 7, 21, 27, 28) with RS unchanged for 2/34 days (days 22, 30), Fig. 1B.
- About 1-2 hours after the first ASBE session, RS for 4/34 days (days 24, 32, 33, 34) had fully resolved but RS for 30/34 days had re-emerged, requiring a second ASBE session, Fig. 1C.

- The second ASBE session bringing temporary relief to RS for 25/30 days, reducing the severity of RS for 4/30 days (days 7, 20, 26, 27) with RS unchanged for 1/30 days (day 11), Fig. 1C.
- About 1-2 hours after the second ASBE session, RS for 10/30 days (days 6, 12, 14, 15, 16, 18, 19, 25, 29, 30) had fully resolved (in addition to days 24, 32, 33, 34 after the first ASBE) but RS for 20/30 days had re-emerging, requiring a third ASBE session, Fig. 1D.
- The third ASBE session fully resolved RS for 11/20 days (days 1, 2, 3, 4, 8, 9, 10, 17, 20, 21, 23) but with RS persisting for 9/20 days (days 5, 7, 11, 13, 22, 26, 27, 28, 31) with 7 days requiring the use of antacids and 2 days (5, 7) not requiring antacid use, Fig. 1D.
- In summary the 3 ASBE sessions fully resolved RS for 25/34 ≈ 74% of days and with RS for 27/34 ≈ 79% of days not requiring the use of antacids.

It is uncertain how much the RS scores would have reduced naturally over time, independently of ASBE sessions, potentially inflating the benefit of ASBE, however ASBE had reduced the use of antacids to manage RS.

Relief from ASBE could take 10 minutes to occur after the session, while antacid tablets worked more rapidly, indicating redox reactions have a slower rate than acid neutralization. It was claimed that RS that developed approximately 30 minutes after drinking coffee in the mornings could be reduced by 2-3 ASBE of 20-30 seconds, but RS score values were not recorded. The patient also found ASBE sessions worthwhile and would continue as a treatment option for RS at the end of the 50 days, as the need for antacids had been reduced. There was no trend observed showing the severity or frequency of RS had reduced over the 50 days (34 days ASBE) from the ASBE. It is not known if air swallowing or breathing exercises alone or both together, provide the best option for RS reduction and resolution.

4.0 DISCUSSION

To understand the finding that ASBE can reduce RS, a discussion with background information regarding the role of hydrogen peroxide, oxygen reactivity and supply, antioxidants in food and a brief literature review of the role of oxygen in the gut was undertaken, resulting in a hypothetical oxygen model for reflux.

4.1 Hydrogen peroxide in humans and its formation by oxygen in water

Hydrogen peroxide could be considered a toxic substance with no beneficial role however it is produced by every cell in the body and is important physiologically but when produced in excess, may cause disease [3]. For example, excessive production of hydrogen peroxide has been found to reduce lower esophageal sphincter tone in human esophagitis [4]. Although hydrogen peroxide can form when oxygen dissolves in water, at high concentrations it can be toxic. This was found when a solution of hydrogen peroxide (3%) when unintendedly ingested over several weeks, as part of a dental mouth wash, causing chemical gastritis and colitis that completely resolved on cessation [5]. Hydrogen peroxide is contained in human milk ($H_2O_2 \approx 10^{-5}$ mol/L), found to promote gastric motility with an antimicrobial role in murine studies and may enhance gastric emptying in neonates [6]. The antimicrobial properties of hydrogen peroxide ($H_2O_2 \approx 0.1$-0.03 mol/L) were also shown by the eradication of *Helicobacter pylori* in vivo and in vitro from animal models [7].

The molecular oxygen present in air has a hypothetical redox half equation showing oxygen can react as an oxidizing reagent:

$$O_2 + 4e^- \rightarrow 2O^{2-} \quad (1)$$

is soluble in water:

$$O_{2(g)} \leftrightharpoons O_{2(aq)} \quad (2)$$

and can generate hydrogen peroxide which can also react as an oxidizing agent:

$$O_2 + 2H^+ + 2e^- \rightarrow H_2O_2 \quad (3)$$

$$H_2O_2 + 2H^+ + 2e^- \rightarrow 2H_2O \quad (4)$$

$$O_2 + 4H^+ + 4e^- \rightarrow 2H_2O \quad (5)$$

with oxygen able to accept a maximum of 4 electrons per mole as part of the oxidation process [8]. Many other reactive oxygen species (ROS), in addition to hydrogen peroxide, can be formed including highly reactive oxygen free radicals that can remove a hydrogen atom from unsaturated fatty acids [9, 10]. The thermodynamics and kinetics of redox reactions involving oxygen and the digestion of food can be pH dependant, as shown by eqs. (3-5) and catalysed by metals (Fenton reaction) [8-12].

4.2 The oxidizing potential of oxygen in 100 ml swallowed air in mmol electrons

For 100 ml of swallowed air contained in the stomach, assuming 21% of the volume was oxygen or 21ml, with 78% nitrogen and 1% other gases including water vapor, then the moles of swallowed oxygen can be calculated at standard temperatures and pressure from the ideal gas equation:

$$P\,V = n\,R\,T \quad (6)$$

For oxygen, assuming a pressure (P) of 1 atmosphere or 101,325 pascals, volume (V) as 21 ml /100 ml of air giving $V = 21/10^6$ m^3, gas constant R = 8.314 and temperature of 25° C or 273.15 + 25 = 298.15 K then:

$$n = PV/RT = 101325 \times (21/10^6)/8.314 \times 298.15 = 8.58 \times 10^{-4} \text{ mol oxygen} \quad (7)$$

or the number of moles of oxygen $O_2 \approx 0.86$ mmol /100 ml of swallowed air.

From eq. (1) with a maximum of 4e /mole of oxygen that can be accepted, then from eq. (7):

0.858 x 4 = 3.43 mmol electrons /100 ml air (8)

is the maximum number of electrons that can be accepted by 21 ml of oxygen (100 ml air) from antioxidants or electron rich food substances, as part of the oxidation process.

4.3 The oxidizing potential of oxygen in 100 ml drinking water in mmol electrons

The concentration for pure oxygen, that can dissolve in water is on average $O_2 \approx 1.22$ mmol /L (pH ≈ 1-14, low ionic strength solutions) at 25°C and 1 atmosphere (atm) with solubility increasing with pressure but decreasing with increasing electrolyte concentration and temperature [13,14]. Air at 21% oxygen, has a partial pressure P ≈ 0.21atm resulting in the concentration of $O_2 \approx 0.21x1.22 \approx 0.256$ mmol/ L in water (≈ 0.0256 mmol /100 ml water) at 25° C, [14].

From eqs. (3- 5), with up to 4e /mole of oxygen available:

0.0256x4 ≈ 0.10 mmol electrons /100 ml water (dissolved oxygen) (9)

is the maximum number of electrons that can be accepted by oxygen from air dissolved in 100ml of water.

Swallowed air has a concentration of $O_2 \approx 0.86$ mmol /100 ml air compared to $O_2 \approx 0.0256$ mmol /100 ml dissolved in water. As such, air contains ≈ 34x more oxygen than oxygen dissolved in water, at the same volume and temperature. Oxygen dissolved in water may be more reactive towards food contents than oxygen gas at the lumen/aqueous interface, but the depleted oxygen from water by reaction with food would allow more oxygen from air to dissolve in accordance with the equilibrium eq. (2).

4.4 Systemic oxygen supply to the human body

During the inhalation of air, with a 21% oxygen content, oxygen in the alveoli is exchanged for the waste product, carbon dioxide from cellular energy production and exhaled as 16% oxygen and 5% carbon dioxide [15]. An adult at rest, exchanges ≈ 500 ml of air per breath (tidal volume) with 12 breaths a minute, resulting in ≈ 6 litres /minute air ventilation [15]. With moderate exercise the breathing rate can increase to 20 breaths /minute with ≈ 2 litres /breath resulting in ≈ 40 litres /minute ventilation [15]. As such, from the values above, the amount of pure oxygen /minute, when breathing at rest, can be calculated as $O_2 \approx (0.21\text{-}0.16) \times 6 \approx 0.3$ litres /minute or $O_2 \approx 300$ ml /minute and with moderate exercise $O_2 \approx (0.21\text{-}0.16) \times 40 \approx 2$ litres /minute. In awake healthy subjects at rest oxygen consumption, has been reported to be $O_2 \approx 250$ ml pure oxygen /min [16]. As 100 ml of swallowed air contains 21 ml oxygen, then 21/250 or ≈ 8% of the bodies systemic oxygen consumption/minute can be made available within the gastric lumen, indicating multiple air swallowing can provide a significant increase in gastric oxygen availability for aerobic digestion when at rest.

4.5 Air swallowing, breathing, luminal oxidation of lipids and the microbiome

Air swallowing naturally occurs on swallowing and when food is consumed. The swallowing of a 10 ml liquid bolus including 8-32 ml of air, which can result in belching, but patients with excessive belching, did not have more frequent gaseous gastroesophageal reflux [17]. Swallowed air has a higher partial pressure of oxygen in the stomach than in the blood, allowing oxygen to be directly adsorbed by diffusion [18].

A meta-analysis of 7 studies on the effects of breathing exercises on patients with GERD found reduced symptoms, improved quality of life and a decrease in acid suppression use, possibly due to strengthening the anti-reflux barrier function [19]. Diaphragmatic breathing exercises have been shown to reduce the symptoms of GERD and reduce acid exposure, attributed to respiratory physiotherapy, however the proof of the mechanism of action was reported to be lacking [20, 21]

In murine models it was found that bacteria in the lumen consumed more oxygen than bacteria adherent to the mucosa throughout the length of the gut [22]. Oxygen was not only consumed by bacteria but was also involved in direct chemical oxidation processes of dietary lipids and this may be one of the processes responsible for oxygen consumption [22]. It was also found that oxygen levels were highest in the duodenum with luminal oxygen levels higher than the intravascular levels, followed by the stomach with lower oxygen levels but with similar luminal and intravascular oxygen levels [22]. The existence of oxygen, other than in the stomach, was found by using a swallowed electronic capsule capable of sensing different gases, finding oxygen in the jejunum, ilium, and with a high fibre diet, the colon, with excess hydrogen mitigated by oxidation [23].

It has been reported that 5% of total blood volume is present in the gut during fasting increasing to 30% following the ingestion of a meal, creating daily fluctuations [24-26]. The oxygen gradient for the small intestinal epithelial cells, villus and lumen are all at physiologically low O_2 levels (2-4%) and depicted as tissue in constant low grade inflammation [24, 25, 26]. Hypoxia and hyperoxia can alter the microbiome content of the gut however ASBE did not show a trend towards increasing or decreasing RS over the 50 days (34 with ASBE) suggesting any alteration of the microbiome did not greatly influence RS for this case report [26].

In summary, oxidative processes using luminal oxygen for digestion have been found to occur through the digestive regions of the gut indicating an oxygen demand, but it is uncertain how much of this is supplied by air swallowing or systemically from the circulating haemoglobin in blood. Dietary lipids can be considered a potential trigger food and if luminal oxygen is directly involved in their oxidation, may have an important role in reducing RS, provided enough can be supplied from either swallowing or the blood supply.

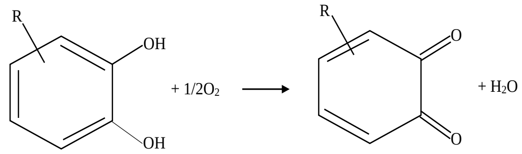
4.6 Antioxidants, foods and their electron content

Foods when consumed for digestion can undergo oxidation or reduction (redox reactions) to involve the addition of oxygen, electron /hydrogen atom exchange or free radical reactions, as part of the digestive process [1, 27]. The redox-activity of 1113 food samples have been ranked according to their antioxidant contents, as mmol electrons /100 g or mmol /serving size with the aim of assessing the health benefits of antioxidants [1]. This was achieved by using the FRAP (ferric reducing ability of plasma) test by measuring the relative oxidizing ability of foods towards 2,4,6,tripyridyl-*s*-triazine (TPTZ) as TPTZ-Fe(III)Cl_3 which on mixing with food samples, accepts electrons, to form a deep blue TPTZ-Fe(II)Cl_2 [1]. Foods that have relatively high antioxidant capacity per serve, include coffee (2.96 mmol/serve), chocolate (2.52 mmol /serve), red wine (2.2 mmol /serve), pineapple juice (1.86 mmol /serve), oranges (1.26 mmol /serve), and iced tea (0.88 mmol /serve), often considered as trigger foods for RS with high refluxogenic scores [1, 28]. Foods with the lowest antioxidant activity include fats and oils (0.187-0.531 mmol /serve), meat products (0.052-0.509 mmol /serve) and substitutes, poultry products (0.072-0.388 mmol/serve), fish and sea food (0.025-0.141 mmol /serve) with the lowest values for eggs and egg dishes (0.009-0.047 mmol /serving) [1]. Fats and oils despite the electrons available from the alkenes in polyunsaturated fats, were found to have low antioxidant activity, possibly due to any number of variables including slow reaction rate with the TPTZ-Fe^{3+} (a 4-minute incubation time was used), solubility, pH, ionic strength (electron conduction) and temperature [1].

Ideally it is best to measure both the pro-oxidant and antioxidant properties of food as no single test would reflect the many possible redox reactions for in vivo digestion [27]. If both pro-oxidant and antioxidant properties were measured, using the FRAP and 3 additional testing methods, coffee and orange juice were found to have very high antioxidant potential with sausage and white chocolate having both high antioxidant and dietary lipid pro-oxidant properties at the same time [27].

Based on the calculated value in mmol electrons /100 ml of swallowed air, oxygen has the capacity to oxidize 1 serve of coffee, red wine or orange juice but not the highest scoring foods like 1 serve of blackberries (5.746 mmol /serving), walnuts (3.721 mmol /serving), strawberries (3.584 mmol /serving) or artichokes (3.559 mmol /serving), based on the FRAP analysis [1].

The polyphenols in red wine, which contain multiple aromatic hydroxy groups, have been reported to totally inhibit dietary lipid peroxidation when saliva is present in the acidic environment of the stomach [29]. Foods containing polyphenols can react directly with oxygen in air turning food brown as shown from a slice of apple [30]. Polyphenols are likely to react directly with luminal oxygen depleting supply and in this example, removing hydrogen to form water, with the oxidation process not necessarily adding more oxygen atoms on forming the oxidised product.



4.7 Oxygens association with reflux and the digestion process

Numerous studies give background information and provide a clue to a possible relationship between systemic or luminal oxygen supply, RS and GERD in Table 1 and oxygens role in other gut regions, Table 2 with some comments provided.

Table 1: Oxygen, reflux and the digestion process

Chronic obstructive pulmonary disease (COPD) associated with abnormally high prevalence of oesophageal acid reflux where oxygen desaturation can coincide with periods of acid reflux in some patients.	31
Marked improvement in RS for patients with obstructive sleep apnea (OSA) treated with continuous positive air pressure (PAP) and nasal PAP suggested as a new treatment for nocturnal reflux disease.	32,33
The effect of PAP on productive cough was found to be mainly mediated through decreased in GERD.	34
Acute oxygen desaturation associated with pulmonary aspiration in patients with GERD and laryngopharyngeal reflux.	35
Gastric mucosal oxygen delivery and red blood cell flux decreased by 50% during hypothermic cardiopulmonary bypass despite constant systemic oxygen delivery. Comment: although the mechanisms were not discussed for the sedated patients, this result may indicate luminal oxygen has a role in increasing gastric mucosal oxygen levels in awake subjects.	36
Chewing gum is associated with an increase in air swallowing and a reduction in RS.	37,38
Consumption of carbonated beverages is associated with laryngopharyngeal reflux and GERD. Comment: the mechanism was not discussed but could involve the displacement of oxygen from the lumen by carbon dioxide, changing anerobic digestion conditions.	39

Magnetic resonance imaging of oxygen saturated water, claimed to increase health and athlete performance, found molecular oxygen remained present in the stomach or duodenum for ≈ 45 minutes. Comment: the use of oxygen saturated water to relieve acute reflux symptoms was not reported.	40
Upper airway obstruction in newborn lambs had no significant effect over 3 hours but after 6 hours, mildly increased the number of gastroesophageal reflux events. Comment: hypoxia is likely to create reflux events.	41
Hypoxia of the esophageal mucosa related to ischemic heart disease leads to a decrease in both esophageal tissue resistance and lower sphincter dysfunction, leading factors in the development of GERD.	42
Air swallowing could promote belching but did not facilitate acid gastroesophageal reflux in both healthy and GERD patients. Comment: air swallowing may reduce the acidity of reflux.	43,44
Frequent belching was common in patients with functional dyspepsia who were found to swallow more air than controls (up to 80% incidence) resulting in an increased incidence of non-acid gaseous gastroesophageal reflux, with no signs of pathological acid reflux. Comment: gaseous reflux is likely to involve gas exchange with air in the oesophagus /pharynx prior to returning to the stomach.	45
The gas component of the stomach remains relatively constant at ≈ 98-109±63 ml over 1 hour while digesting 500 ml of soup, even at low liquid volumes of 294±74 ml. Comment: retention of luminal gas during gastric digestion suggests an important role.	46
Oxygen demand is associated with high energy processes, like the transport functions of intestinal epithelial for electrolytes including Na^+/K^+-ATPase with activity either increased or decreased during periods of hypoxia. Hypoxia has been found to reduce acid secretion and gastric emptying in murine models. Comment: how oxygen demand influences H^+/K^+-ATPase activity, an important component of GERD, requires further investigation.	47,48, 49

Table 2: Both systemic and luminal oxygen with redox, IBD, HIF, oxygen in the gut and the microbiome but an association with bile and coloileal reflux was not found

The metabolism of food, as it passes through the gut, involves specific regional redox potentials (Eh) and pH values with the oral cavity Eh ≈ 800-1000 mV with pH ≈ 7-8, the most oxidizing part of the gastric system due to the large amount of oxygen, followed by the stomach with a Eh ≈ 400-500 mV, pH ≈ 1-5, both forming the more aerobic regions of the digestive system, followed by the ileum Eh ≈ 0-(-200) mV, pH ≈ 7-8 and colon Eh ≈ -200-(-300) mV with pH ≈ 5-6 with both forming the more anaerobic and reducing regions with low oxygen levels. Comment: redox potentials indicate the location of the oxidizing and reducing environments of the gut, presumably with different microbiome content, with reaction thermodynamics and kinetics likely pH dependent.	50
Biopsy specimen classified 20 aerobic and 12 anaerobic bacteria in the gastric mucosa with the fundus redox potential ≈ 400-500 mV and the colon ≈ -200-(-300) mV with the redox balance influenced by the bacteria present and therefore antibiotics, both implicated in the development of diseases. Comment: the oxidizing environment of the stomach does not contain exclusively aerobic bacteria.	51
Cellular reductive stress, the counterpart of oxidative stress is associated with a shift in the redox balance to a more reducing state, leading to inflammatory conditions, distorted transport, failed oxidation and metabolic syndrome.	52
Colonic fermentation of indigestible carbohydrates in 9 patients with symptomatic GERD, increased transient lower sphincter relaxations (TLESRs), the number of acid reflux episodes and GERD symptoms. Comment: relaxation of the pyloric and /or ileocecal sphincters and an association with luminal oxygen supply was not investigated.	53
The role of oxygen in inflammatory bowel disease (IBD) may result from diminished tissue oxygen levels (anoxia) initiating hypoxia inducible factor (HIF) pathways caused by a loss in intestinal epithelial barrier function, allowing luminal contents access to underlying cells.	54
Conditions that increase the epithelial oxygen content for intact epithelia, which may include the presence of dietary fibre, can create an imbalance in the anerobic /aerobic bacteria ratio, resulting in dysbiosis in the colon.	55
Regurgitation of duodenal bile contents into the stomach and oesophagus, known as bile reflux, can present as bile acids in the oesophagus and is associated with GERD and hypopharyngeal cancer.	56
Coloileal reflux, classified as mild, moderate or severe has been reported at a 46.5% frequency from the analysis of 715 barium enemas, with the causes and consequences not widely investigated.	57,58
Oxygen pressure in the lumen of the small intestine <10 mm Hg and in the lumen and sigmoid colon < 3-11 mm Hg with the role of the epithelial-lumen interface and luminal oxygen concentrations in intestinal diseases not currently known.	59

4.8 Air columns in the esophagus

For 110 subjects, computerized tomography (CT) scans were used to determine the average length of the esophagus as 21.7(range 16.8-26.4) cm with air columns frequently found with a maximum length of 2.32 cm and an average length of 1.05±0.5 cm and could be detected along the entire length of the esophagus [60]. Food was found in the esophagus for 9% of subjects and considered normal [60]. In another study, 36% (74/200) of normal chest radiographs and 50 % (101/200) with abnormal radiographs found air was present in the esophagus [61]. CT scans found air columns were frequently found in the esophagus in a study of 59 GERD patients with maximum mean size of 3.088±1.495 cm and for patients with heartburn the mean column size was 1.367±0.671 cm with air columns especially in the middle to lower parts [62]. These results indicate that it is not unusual to find both air columns and sometimes food in the esophagus on CT scanning.

4.9 The oxygen model for digestive reflux

This case report has found ASBE can reduce RS in one individual but despite this limitation, ASBE must increase the oxygen concentration in the gastric lumen and other intestinal regions, increasing the probability of the necessary successful aerobic digestion of food. Increasing oxygen supply increases the probability for the oxidation of refluxogenic foods, changing their composition and therefore potentially reducing their influence on producing RS.

A hypothetical model is proposed suggesting that if the oxygen supply necessary for aerobic digestion is unavailable, the food /liquid contents can reflux from the stomach or duodenum towards more oxygenated regions of the oesophagus /pharynx or even the mouth, increasing swallowing and air supply and reducing cellular hypoxia /anoxia (Graphical Abstract). Reflux between regions with different Eh and pH may also be beneficial suggesting a possible bidirectional component for optimal human digestion. Reflux can also occur between the colon and the small intestine, although not normally refluxing into the oesophagus. Due to the possibility of multiple reflux processes, the model was termed the digestive reflux model, rather than the gastroesophageal reflux model.

Tables 1 and 2 provide supporting evidence and a clue into the role oxygen has in both preventing or initiating digestive RS in particular:

- ASBE in the current case report reduced RS and the need to use antacids treatments,
- breathing exercises were found to reduce the symptoms of GERD and reduce acid suppression medication [19,21,20],
- PAP or positive airway pressure (increased luminal oxygen) suggested as a new treatment for GERD [33],
- a remarkably high prevalence of oxygen desaturation associated with gastroesophageal in patients with RS [63],
- acute oxygen desaturation (hypoxia) associated with pulmonary aspiration in patients with GERD and laryngopharyngeal reflux suggesting reduced oxygen supply could initiate reflux [35].
- colonic fermentation of fibre can increase transient lower sphincter relaxations (TLESR) and GERD symptoms and ingestion of fibre has been associated with increased oxygen concentration in the bowel, indicating TLESR (also associated with many other foods) may be initiated to increase luminal oxygen supply throughout the gut, but evidence is lacking [23, 53, 59, 64].
- during CT scanning of the normal esophagus and for GERD patients, air columns are frequently seen and can have a maximum size of 3.088±1.495 cm, indicating the esophagus can be a region that contains air columns [60,61,62].

Why the systemic blood supply cannot deliver sufficient oxygen for digestion may be due to reduces respiratory drive as associated with obesity hypoventilation syndrome and a greater oxygen demand occurring postprandially, particularly if sleeping soon after meals [65]. A unifying oxygen related model for reflux would support the suggestion that GERD, functional dyspepsia and irritable bowel syndrome could be overlapping disorders [66].

CONCLUSION

The process of ASBE was found to resolve or reduce most postprandial RS, although multiple ASBE sessions are required over several hours, reducing the need to use antacid treatments. A 100 ml of oxygen from air swallowing was shown to have the capacity to oxidise several refluxogenic foods changing their composition and therefore potentially reducing their influence on producing RS. A brief literature review gave a clue into the central role oxygen, both systemic and luminal has in the digestion process. A hypothetical model was proposed that suggests if there is insufficient systemic or luminal oxygen available to carry out the necessary redox reactions, regions of the gut may initiate reflux of digesting food to regions of higher luminal oxygen concentration. Further studies are required to determine what the role oxygen may have in digestive reflux diseases.

Conflict of interest: No conflict of interest was declared.

Funding: No financial support was received for this article.

Ethical approval and informed consent:

This manuscript adheres to the national and international ethical guidelines for research on human subjects. Written informed consent was obtained and the patient has read the manuscript and approved it for publication.

Abbreviations: air swallowing and breathing exercises (ASBE), reflux symptoms (RS).

REFERENCES

1. Halvorsen BL, Carlsen MH, Phillips KM, Bøhn SK, *et al.*, Content of redox-active compounds (ie, antioxidants) in foods consumed in the United States. Am J Clin Nutr. 2006; 84:95-135.
2. Hurr TJ. The six-question gastroesophageal reflux disease questionnaire (GerdQ) cannot accurately quantify reflux and reflux-associated symptoms frequency. Gastroenterol Report. 2022; 43:1-2.
3. Pravda J. Hydrogen peroxide and disease: towards a unified system of pathogenesis and therapeutics. Molecular Med. 2020; 26:1-10.
4. Cheng L, Harnett KM, Cao W, Liu F, *et al.*,Hydrogen peroxide reduces lower esophageal sphincter tone in human esophagitis. Gastroenterol. 2005; 129:1675-1685.
5. Zanelli M, Ragazzi, M, De Marco L. (2017) Chemical gastritis and colitis related to hydrogen peroxide mouthwash. Br J Clin Pharmacol, 2017;83:427–428.
6. Fajardo AF, Sobchak C, Shifrin Y, Pan J, et al. Hydrogen peroxide promotes gastric motility in the newborn rat. Pediatric Res. 2018;84:751-756.
7. Di J, Zhang J, Cao L, Huang TT, Zang J, *et al.*,Hydrogen peroxide-mediated oxygen enrichment eradicates *helicobacter pylori* in vitro and in vivo. Antimicrob Agents Chemother. 2020;64: e02192-19.
8. Nutting JE, Gerken JB, Stamoulis AG, Burns DL, *et al.*, How should I think about voltage? what is overpotential? Establishing an organic chemistry intuition for electrochemistry. J. Org. Chem. 2021; 86:15875–15885.
9. Domínguez R, Pateiro M, Gagaoua M, Barba FJ, *et al.*,A Comprehensive review on lipid oxidation in meat and meat products. Antioxidants. 2019; 8:429-460.
10. Wang D, Xiao H, Lyu X, Chen H, Wei F. Lipid oxidation in food science and nutritional health: A comprehensive review. Oil Crop Science. 2023; 8:35-44.
11. Ibanez JG, Hernandez-Esparza M, Doria-Serrano C, Fregoso-Infante A, *et al.*, (2007). Chemistry fundamentals, part A. Environmental chemistry fundamentals (1st ed.). Springer New York. 2007; 2:11-42.
12. Krishtalik LI. pH-dependent redox potential: how to use it correctly in the activation energy analysis. Biochimica et Biophysica Acta. 2003; 1604:13-21.
13. Bok F, Mogg HC, Brendler V. The solubility of oxygen in water and saline solutions. Front. Nucl. Eng. 2023; 1158109:1-25.
14. Xing W, Yin M, Lv Q, Hu Y, *et al.*, Oxygen solubility, diffusion coefficient, and solution viscosity in Rotating electrode methods and oxygen reduction electrocatalysts. Ed. Xing W, Yin G, Zang J. Elsevier. 2014; 1:1-31.
15. Pleil J, Ariel Greer Wallace M, Davis MD, Matty CM. The physics of human breathing: flow, timing, volume, and pressure parameters for normal, on-demand, and ventilator respiration. J Breath Res. 2021; 15:1-24.
16. Hahm TS, Jeong H, Ahn HJ. Systemic oxygen delivery during one-lung ventilation: comparison between propofol and sevoflurane anaesthesia in a randomised controlled trial. J Clin Med. 2019;8:1438-1448.
17. Bredenoord AJ, Weusten BL, Sifrim D, Timmer R, *et al.*, Aerophagia, gastric, and supragastric belching: a study using intraluminal electrical impedance monitoring. Gut. 2004;53:1561-1565.
18. Zhao C, Liu R, Zhou Y, Zheng R. *et al.*, Ignored roles of gases in digestive diseases. Biomed. Technol. 2023;3:1-10.
19. Qiu K, Wang J, Chen B, Wang H, *et al.*, The effect of breathing exercises on patients with GERD: a meta-analysis. Ann Palliat Med. 2020; 9:405-413.
20. Halland M, Bharucha AE, Crowell MD, Ravi K, *et al.*,Effects of diaphragmatic breathing on the pathophysiology and treatment of upright gastroesophageal reflux: A randomized controlled trial. Am J Gastroenterol. 2021;116:86-94.
21. Zdrhova L, Bitnar P, Balihar K, Kolar P, *et al.*, Breathing exercises in gastroesophageal reflux disease: a systematic review. Dysphagia. 2023; 38:609-621.
22. Friedman ES, Bittinger K, Esipova TV, Hou L, *et al.*,Microbes vs. chemistry in the origin of the anaerobic gut lumen. PANS. 2018;115:4170-4175.
23. Kalantar-Zadeh K, Berean KJ, Ha N, Chrimes AF, *et al.*, A human pilot trial of ingestible electronic capsules capable of sensing different gases in the gut. Nature Electronics. 2018; 1:79-87.
24. Zeitouni NE, Chotikatum S, Köckritz-Blickwede M, Naim HY. The impact of hypoxia on intestinal epithelial cell functions: consequences for invasion by bacterial pathogens. Mol Cell Pediatr. 2016; 3:1-9.
25. Colgan SP, Taylor CT. Hypoxia: an alarm signal during intestinal inflamation. Nat Rev Gastroenterol Heptol. 2010;7:281-287.
26. Dai N, Gu J, Luo Y, Tao Y, *et al.*, Impact of hyperoxia on the gut during critical illness. Critical Care. 2024; 28:1-12.
27. Vahid F, Wagener L, Leners B, Bohn T. Pro- and antioxidant effect of food items and matrices during simulated in vitro digestion. Foods. 2023;12:1719-1730.
28. Lechien JR, Bobin F, Mouawad F, Zelenik K. *et al.*, Development of scores assessing the refluxogenic potential of diet of patients with laryngopharyngeal reflux. Eur Arch Otorhinolaryngol. 2019; 276:3389-3404.
29. Gorelik S, Kohen R, Ligumsky M, Kanner J. Saliva plays a dual role in oxidation process in stomach medium. Arch Biochem Biophys. 2007; 458:236-243.
30. Murata M. Food chemistry and biochemistry of enzymatic browning. Food Sci Tech Res. 2022; 28:1-12.

31. Casanova C, Baudet JS, del Valle Velasco M, Martin JM. *et al.,* Increased gastro-oesophageal reflux disease in patients with severe COPD. Eur Respir J. 2004; 23:841-5.
32. Green BT, Broughton WA, O'Connor JB. Marked improvement in nocturnal gastroesophageal reflux in a large cohort of patients with obstructive sleep apnea treated with continuous positive airway pressure. Arch Intern Med. 2003; 163:41–45.
33. Kerr P, Shoenut JP, Steens RD, Millar T. *et al.,* Nasal continuous positive airway pressure. A new treatment for nocturnal gastroesophageal reflux? J Clin Gastroenterol. 1993; 17:276-280.
34. Emilsson ÖI, Aspelund T, Janson C, Benediktsdottir B, *et al.,* Positive airway pressure treatment affects respiratory symptoms and gastro-oesophageal reflux: the Icelandic sleep apnea cohort study. ERJ Open Res. 2023; 9:00387.
35. Weerasinghe DP, Burton L, Chicco P, Pearson M, *et al.,* Acute oxygen desaturation characterizes pulmonary aspiration in patients with gastroesophageal reflux disease and laryngopharyngeal reflux. Physiol Rep. 2022;10: e15367:1-9.
36. Sicsic JC, Duranteau J, Corbineau H, Antoun S, *et al.,* Gastric mucosal oxygen delivery decreases during cardiopulmonary bypass despite constant systemic oxygen delivery. Anesth Analg. 1998; 86:455-60.
37. Moazzez R, Bartlett D, Anggiansah A. The effect of chewing sugar-free gum on gastro-esophageal reflux. J Dent Res. 2005; 84:1062-1065.
38. Silva AC, Aprile LR, Dantas RO. Effect of gum chewing on air swallowing, saliva swallowing and belching. Arq Gastroenterol. 2015; 52:190-194.
39. Wang M, Mo T, Tan J, Dai Y, Li X. Risk factor-related lifestyle habits of patients with laryngopharyngeal reflux. Ear Nose Throat J. 2022; 0:1-10.
40. Nestle N, Wunderlich A, Nüssle-Kügele K. In vivo observation of oxygen-supersaturated water in the human mouth and stomach. Magn Reson Imaging. 2004; 22:551-556.
41. Michaud A, Jia WL, Djeddi D, Samson N, *et al.,* Effects of upper airway obstruction or hypoxia on gastroesophageal reflux in newborn lambs. Pediatr Res. 2021; 89:496-501.
42. Oparin A, Vnukova A. The role of endothelial dysfunction in the mechanism of gastroesophageal reflux disease development in patients with ischemic heart disease. Acta Clin Croat. 2017;56:635-639.
43. Bredenoord AJ, Weusten BL, Timmer R, Akkermans LM, *et al.,* Relationships between air swallowing, intragastric air, belching and gastro-oesophageal reflux. Neurogastroenterol Motil. 2005;17:341-347.
44. Bredenoord AJ, Weusten BL, Timmer R, Smout AJ. Air swallowing, belching, and reflux in patients with gastroesophageal reflux disease. Am J Gastroenterol. 2006;101:1721-1726.
45. Conchillo JM, Selimah M, Bredenoord AJ, Samsom M, *et al.,* Air swallowing, belching, acid and non-acid reflux in patients with functional dyspepsia. Aliment Pharmacol Ther. 2007; 25:965-971.
46. Bertoli D, Mark EB, Liao D, Brock C, *et al.,* A novel MRI-based three-dimensional model of stomach volume, surface area, and geometry in response to gastric filling and emptying. Neurogastroenterol Motil. 2023;35: e14497-12.
47. Ward JB, Keely SJ, Keely SJ. Oxygen in the regulation of intestinal epithelial transport. J Physiol. 2014; 592:2473-89.
48. Engevik A, Kaji I, Goldenring JR. The physiology of the gastric parietal cell. Physio Rev. 2020; 100:573-602.
49. Yamaji R, Sakamoto M, Miyataki K, Nakano Y. Hypoxia inhibits gastric emptying and gastric acid secretion in conscious rats. J Nutr. 1996; 126:673-680.
50. Kaliaperumal V, Ramadass B. A perspective on gut health: The redox potential and pH. Gastroenterol Hepatol and Endosc Pract. 2023; 3:12-16.
51. Panigrahi MK, Kaliaperumal V, Akella A, Venugopal G, *et al.,*Mapping microbiome-redox spectrum and evaluating microbial-redox index in chronic gastritis. Sci Rep. 2022; 12:8450-8564.
52. Pérez-Torres I, Guarner-Lans V, Rubio-Ruiz ME. Reductive stress in inflammation-associated diseases and the pro-oxidant effect of antioxidant agents. Int J Mol Sci. 2017; 18:2098.
53. Piche T, des Varannes SB, Sacher-Huvelin S, Holst JJ, *et al.,*Colonic fermentation influences lower esophageal sphincter function in gastroesophageal reflux disease. Gastroenterology. 2003; 124:894-902.
54. Manresa MC, Taylor CT. Hypoxia inducible factor (HIF) hydroxylases as regulators of intestinal epithelial barrier function. Cell Mol Gastroenterol Hepatol. 2017; 3:303-315.
55. Litvac Y, Byndloss MX, Bäumler AJ. Colonocyte metabolism shapes the gut microbiota. Science. 2018; 362:1-8.
56. Vageli DP, Doukas SG, Doukas PG, Judson BL. Bile reflux and hypopharyngeal cancer (review). Oncol Rep. 2021; 46:244-258.
57. Machado WM, Morceli J. Prevalence, classification and characteristics of the coloileal reflux diagnosed by barium enema. Radiologia Brasileira. 2006; 39:1-9.
58. Machado WM, Miranda JR, Morceli J, Padovani CR. The small bowel flora in individuals with cecoileal reflux. Arq Gastroenterol. 2008; 45:212-218.
59. Singhal R, Shah YM. Oxygen battle in the gut: hypoxia and hypoxia inducible factors in metabolic and inflammatory responses in the intestine. J Biol Chem. 2020;295:10493-10505.

60. Schraufnagel DE, Michel JC, Sheppard TJ, Saffold PC, Kondos GT. CT of the normal esophagus to define the normal air column and its extent and distribution. AJR Am J Roentgenol. 2008; 191:748-52.
61. Proto AV, Lane EJ. Air in the esophagus: a frequent radiographic finding. AJR Am J Roentgenol. 1977; 129:433-40.
62. Moosavi A, Raji H, Teimoori M, Ghourchian S. Air column in esophagus and symptoms of gastroesophageal reflux disease. BMC Medical Imaging. 2012; 12:1-7.
63. Salvador R, Watson TJ, Herbella F, Dubecz A, *et al.*, Association of gastroesophageal reflux and O_2 desaturation: a novel study of simultaneous 24-h MII-pH and continuous pulse oximetry. J Gastrointest Surg. 2009; 13:854-861.
64. Özenoğlu A, Anul N, Özçelikçi B. The relationship of gastroesophageal reflux with nutritional habits and mental disorders. Hum Nutr Metab. 2023; 33:1-7.
65. 65.Ghimire P, Sankari A, Kaul P. Pickwickian syndrome. Stat Pearls. Stat Pearls Publishing, Treasure Island Florida. 2024:1-13.
66. de Bortoli N, Tolone S, Frazzoni M, Martinucci I, *et al.*,Gastroesophageal reflux disease, functional dyspepsia and irritable bowel syndrome: common overlapping gastrointestinal disorders. Ann Gastroenterol. 2018; 31:639-648.