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AN EAGLE EYE VIEW ON SNEHAPANA AND KETONES- A REVIEW ARTICLE

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ABSTRACT

The therapy known as snehapana causes the body to become greasy, liquefied, soft, and moisturized. Because of this attraction, our bodies are thought of as the essence of oils and fats. As a result, this study is being prepared to monitor changes in body fat levels. Ketone bodies are the name given to the trio of acetoacetate, ß-hydroxybutyrate, and acetone. These compounds produce energy and are water soluble. The exception is acetone, which cannot be digested and is easily expelled through the lungs. True ketones are acetoacetate and acetone, but B-hydroxybutyrate lacks a keto group. Thus to asseses the Acetone gas through the breath test this protocol is designed by using Ketone Breath Analyser. Snehana treatment is a procedure that involves giving sneha to accomplish the intended impact within a set time frame as opposed to just giving some sneha, regardless of time or duration. The maximum amount of time required for the Snehana technique is Prakarsa Kala.

KEYWORDS: Snehapana, Ketone, Ketosis, Ketone breath test.

INTRODUCTION

The therapy known as snehapana causes the body to become greasy, liquefied, soft, and moisturized. Because of this attraction, our bodies are thought of as the essence of oils and fats. As a result, this study is being prepared to monitor changes in body fat levels. Ketone bodies are the name given to the trio of acetoacetate, ß-hydroxybutyrate, and acetone. These compounds produce energy and are water soluble. The exception is acetone, which cannot be digested and is easily expelled through the lungs. True ketones are acetoacetate and acetone, but B-hydroxybutyrate lacks a keto group. Thus to asseses the Acetone gas through the breath test this protocol is designed by using Ketone Breath Analyser.

MATERIALS AND METHODS

Snehana and Swedana

Bahya Snehana (External application) and Abhyantara Snehana (oral administration) are both included in Snehana Chikitsa. Abhyantara Snehapana is administered before Virechana and Vamana Karma, then Sarvanga Abhyanga and Sarvanga Swedana.[1]

Nirama should be observed, and then the Avastha shodhana technique should be used. After observing the Jeerna Ahara Lakshanas of the previous meal and while the patient is on an empty stomach, the recommended Sneha should be given at the time of Suryodaya Kala. Snehapana should last for a minimum of three days and a maximum of seven days.[2]

Importance of snehana

The actions of Snehana and Swedana before Shodhana were described by Acharya Charaka. Snehana is in charge of increasing vishyandata (Fluidity) and the subsequent Vriddhi of the doshas at their respective places. Dosha paka and the elimination of sroto avarodha (Obstruction in micro channels) will be handled by Swedana. As a result, the doshas will start to travel from Shakha towards the kostha.[4]

Abhyantara snehapana

Snehana is given the utmost priority throughout the entire treatise of Ayurveda. There are two general ways to perform snehana: internally (abhyantara) and externally (bahya).[5]

The five techniques for internal body cleaning were specifically created to allow the biological system to self-regenerate, restore to normal, and stop pathogenesis from

progressing. It is necessary for the body to be properly and safely prepared in order for this to occur, which is where prvakarma in the form of snehana and svedana becomes importan.[6]

According to Shodhananga Snehapana, snehapana is performed till the creation of "samyak Sneha Siddhi Lakshanas," which can be accomplished in Mridu, Madhyama, and Krura Koshta, respectively, in 3, 5, and 7 days.[7]

Shodhanaga Snehapana often has a 7-day maximum lifespan before creating Sneha satmya. However, snehapana can be restarted after a one-day break if the Samyak Sneha siddhi lakshanas are not achieved in 7 days.[8]

There are two ways to administer Shodhananga Snehapana:
A. Arohana Snehapana.
B. Sadyo Snehapana.

Arohana Snehapana: Increasing pattern of Sneha dosage:
Vagbhata was the one who originally suggested starting snehapana with hrsiyasi matra. The context of the Sneha Adyaya of the Astanga Hrdaya Sutra, however, is explained. Arunadutta describes how to achieve the target dose in great detail. If the desired dose is uttama matra, start with hsiyasi, move on to hrsva, madhyama, and finally uttama. If the aim is the madhyama matra, start with hrsiyasi, then hsva, and finally the madhyama.[9]

In his commentry on Cha.Si.1/6, Cakrpni provides a hint. There, he states that performing Snehana karma26 after seven days is forbidden since Sneha mtr brings Satmya into the body. This means that Mtra should be set up so that the body doesn't get used to it. Since the body develops accustomed to Sneha if it is taken in a constant dosage and Sneha fails to produce the necessary Klinnata of Dos, Sodhana cannot be performed if the Dos are not Klinna (Utklista). The Samyak Snehana Lakas version of the Klinnata has its description.[10]

It is explicitly stated in the sixth-century Kalyanakaraka and the ninth-century Vangasena that anyone posted for snehapana should drink ghrta or taila in escalating doses (K.Ka.chi.22 and Vang.Sena79).

Sadyo snehapana

This kind of snehapana is administered in massive amounts to quickly or instantly establish Sneha Siddhi Lakshanas. Bala, Vriddha, Mrudu kosthi, Balaheena, Alpa Doshavastha, Raja Samipya, and Sneha Pariahara Asahishnu are typical cases of sadyo snehapana.[11]

Sneha can also be administered using the following 2 methods:

A. Accha Peya
B. Pravicharana Sneha

A. Accha Peya - This is simply administering Sneha on its alone, without combining it with any other foods.[12]
B. Pravicharana Sneha is simply administering Sneha by combining it with various food items in the form of 24 odanadi or 64 Rasa Pravicharanas.[13]

The Shodhananga Snehapana is thought to be superior to Accha Peya.

Shodhananga snehapana vidhi

The type of snehana therapy known as shodhananaga snehapana is carried out before to panchakarma procedures. The goal of sodhana therapy is to purify the body by releasing the accumulated morbid humours that cause sickness, creating the perfect environment for the body to function properly.[14]

On the day of snehapana, when the rising sun has attained the color of golden yellow orange (Pratapta kanakara pita lohita savitari) and the patient is displaying Ahara Jeerna Lakshanas (Signs of digestions of previous night meal), with the exception of Kshudha (hunger), snehapana is administered after performing auspicious rituals.[15] The patient should be watched for Sneha Jeeryamana Lakshanas (signs of Sneha being digested) and Sneha jeerna Lakshanas (signs of Sneha being digested) after Snehapana.

Sodhananga Snehapna's Importance

Sodhana treatments are important processes, or Pradhana Karma, in the therapy plan. They are preceded by specified antecedent acts, or Purva Karma, and are followed by specific remedial acts, or Paschat Karma. The appropriate mobilization of dosas from the channels, which is accomplished with the aid of Snehana and Svedana, is essential to the success of the entire Sodhana treatment.[16]

Out of these two, the sodhanänga snehapäna is a crucial procedure that directs and determines the Sodhana treatment's overall outcome. Snehana, also known as oleation therapy, is a treatment that gives the body Snigdhata, or oiliness, and also makes it simple to extract vitiated dosas. Prior to Vamana and Virecana Karma, Sneha must be administered in a precise manner in order to produce the intended effect; it is not just a matter of delivering Sneha regardless of dose, time, length, etc.[17]

The goal of Snehana therapy is to get the Dosas located in the peripheral tissues to the Kosta so they may be easily thrown out13. This is to get the body ready for Sodhana Karma.[18]

According to Samhitas, Snehana and Svedana are crucial before Sodhana because without them, the body will crumble like dry wood if Sodhana is administered. The Dosas that are stuck to the walls of little Channels are unfastened by Sneha. Dalhana explains that Snehana is necessary in order to bring the Sakhgata Dosas into Kosta. Caraka provides a similar result to how contents simply and effortlessly separate from a smooth container. Similar to Kaphadi Dosas, they were effortlessly evacuated from the oleated bodies. Similar to how a klista Mala of any fabric can be quickly washed by water if it is removed from its location, Malas can be easily evacuated out of Sodhana if they are made Utklişta by Purva karmas, i.e. Snehana and Svedana.[19]

Optimum duration of the snehapana

Snehana treatment is a procedure that involves giving sneha to accomplish the intended impact within a set time frame as opposed to just giving some sneha, regardless of time or duration. The maximum amount of time required for the Snehana technique is Prakarsa Kala.[20]

Snehana therapy can only be administered for a total of seven days, according to reports. Once more, this time frame varies according on the patients' Koshta. Therefore, the Prakarga Kala for Krüra Kosta is the specified 7-day period. Sneha Prakarga Kala in the case of Madhyama Koşta may be five days long, but for Mrdu Kosta, a three-day period is regarded as Sneha Prakarsa Kala.[21]

Sneha matra[22,23]

1. Ashtanga Sangraha and Charaka Samhita both use the Madhyama Matra, a dose that digests in 12 hours.

2. Ashtanga Hridaya's Uttama Matra, a dose that digests in 24 hours
3. Chakrapani's commentary on Uttama and Madhyama Matra

Time of snehapana

Sneha needs to be consumed when the previous night's diet has fully digested and there isn't any sign of hunger for the upcoming meal. The precise timing of Snehapana in the morning as well as at dusk, when the color of the sky turns golden yellow and red, has been elaborated by Acharya Sushruta.[24]

Various assesemnt made during the Snehapana are as follows

Sneha Jeeryamana and Jeerna lakshanas[25]

Sneha Jeeryamana Lakshanas	Sneha Jeerna Lakshanas
Shiroruja	Shirorujadi Jeeryamana Lakshana Prashamana
Bhrama	Vatanulomana
Nishthiva	Swasthya
Murcha	Kshut pravritti
Sada	Trishna Pravritti
Arati	Udgara Shuddhi
Klama,trishna and daha	Laghuta

Samyak sneha siddhi lakshanas

Lakshanas	Cha.Sa[26]	Su.Sa[27]	A.H[28]	Sha.Sa[29]
Vatanulomana	+	-	+	+
Deepta agni	+	-	+	+
Snigdha varchas	+	-	+	+
Asamhata varchas	+	-	+	+
Anga mardava	+	-	+	+
Snigdha anga	+	-	+	+
Snigdha twak	-	+	-	-
Vit shaithilya	-	+	-	-
Glani	-	+	-	+
Anga laghavata	-	+	-	+
Adhastat Sneha darshana	-	+	-	-
Snehodwega	-	+	-	+
Vimalendriyata	-	-	-	+

Asnigdha lakshanas

Lakshanas	Cha.Sa[30]	Su.Sa[31]	A.H[32]	Sha.Sa[33]
Grathita Purisha	+	+	-	-
Ruskha purisha	+	+	-	-
Agnimandya	+	+	-	-
Vata pratoloma gati	+	+	-	-

Khara Gatra	+	-	-	-
Ruskha Gatra	+	-	-	-
Uro Vidaha	-	+	-	-
Dourbalya	-	+	-	-
Durvarnata	-	+	-	-
Anna Pachana Krichhrata	-	+	-	-
Susnigdha Lakshana Viparyayata	-	-	+	+

Atisnigdha lakshanas

Lakshanas	**Cha.Sa**[34]	**Su.Sa**[35]	**A.H**[36]	**Sha.Sa**[37]
Panduta	+	-	-	-
Gourava	+	-	-	-
Jadya	+	-	-	-
Purishasya avipakvata	+	-	-	-
Tandra	+	+	-	-
Aruchi	+	-	-	-
Utklesha	+	+	-	-
Mukha srava	+	+	+	+
Guda Srava	-	-	+	-
Grana Srava	-	-	+	-
Guda Daha	+	+	-	+
Bhakta Dwesha	-	+	-	+
Pravahika	-	+	-	+
Purishati Pravrutti	-	+	-	-

Lipid metabolism

Digestion of lipid

Since enzymes are soluble in aqueous solution and lipid molecules are entirely immiscible with water, the digestion of lipids provides a challenge not posed by other dietary components. Bile salts' emulsification of lipids provides a solution to this issue. Fats must first be emulsified into tiny droplets before they can be digested, which speeds up the process. In the mouth or stomach, there is little to no digestion because:[38]

- The stomach's and mouth's secretions don't contain a large amount of lipase.
- There is no mechanism for emulsifying lipids.
- The gastric secretion's acidic pH is detrimental to the digestion of lipids.

The small intestine is the primary location of lipid digestion, where dietary fat is subjected to its main digestive processes employing enzymes released by the pancreas.[39]

Digestion in mouth

The dorsal surface of the tongue secretes a lingual lipase, although in humans, this enzyme is not as important as it is in rats and mice.[40]

Digestion in stomach

The stomach secretes three lipases that preferentially hydrolyze short and medium chain fatty acids (containing 12 or fewer carbon atoms) from dietary triacylglycerols. These lipases, which are only active at neutral pH, are the gastric lipase, lingual lipase, and gastric lipase. Because their stomach pH is closest to neutral and their diets frequently include milk lipids (cow's milk), which contain triacylglycerols with a high percentage of short and medium chain fatty acids, they are most active in newborns and young children. Overall, dietary lipids are not processed in the stomach or the mouth in adults because of the acidic pH of the stomach.[41]

Longer chain fatty acids dissolve in the fat droplets and move on to the duodenum, whereas the released hydrophilic short and medium chain fatty acids are absorbed through the stomach wall and enter the portal vein.[42]

Digestion in small intestine

The duodenum is where the stomach's acidic chyme, which contains dietary fat, exits the stomach and enters the small intestine. The duodenal mucosa is stimulated to secrete the enteric hormones secretin and cholecystokinin (pancreozymin) when the acidic chyme from the stomach enters the duodenum.[43]

The gallbladder contracts as a result of cholecystokinin's action, releasing bile into the small intestine. Additionally, cholecystokinin affects the exocrine cells of the pancreas, leading them to secrete lipase-containing digesting enzymes. Additionally, cholecystokinin slows down gastric motility, which delays the passage of gastric contents into the small intestine.[44]

Review on ketones

As ketones are a consequence of fat metabolism, the process through which they are created is known as ketosis, which is characterized as A condition known as ketosis is characterized by sporadic increases in blood and interstitial fluid levels of acetoacetic acid, -hydroxybutyric acid, and acetone several times above normal.[45]

Because acetoacetic acid is a kind of keto acid. Ketone bodies are the collective name for the three chemicals. Starvation, diabetes, and occasionally even consuming a diet that is almost exclusively composed of fat have all been linked to the development of ketosis. Since there are no carbohydrates present, persons who are starving or consuming a high-fat diet absorb almost no carbohydrates.[46]

When the body doesn't use carbs as fuel, practically all of its energy must come from the metabolism of fats.

Acetone is a volatile chemical that is produced during ketosis and is released in small amounts in the exhaled air of the lungs, giving the breath an acetone odor that is widely employed as a diagnostic indicator of ketosis.[47]

Fat Storage and Synthesis are Important.

Two factors make fat production from carbs particularly significant:

1. The ability of distinct body cells to store carbohydrates in the form of glycogen is typically very limited; the liver, skeletal muscles, and all other body tissues combined can only store a maximum of a few hundred grams of glycogen. In contrast, adipose tissue has the capacity to store several kilograms of fat. As a result, fat production offers a way to store extra energy from consumed carbs (and proteins) for later usage. In fact, the average person has over 150 times more energy stored in fat than they do in carbohydrates.[48]
2. The number of calories in a gram of fat is nearly 2.5 times greater than that in a gram of glycogen. Therefore, for a given weight gain, a person may store several times as much energy in the form of fat as they can in the form of carbohydrates, which is extremely significant when an animal needs to be highly mobile in order to survive.[49]

Ketone body metabolism

Only when there is an optimum balance between fat and carbohydrate degradation does the acetyl-CoA produced during fatty acid oxidation enter the acid cycle. Acetyl-CoA in the liver, on the other hand, has a different fate if fat breakdown predominates.[50]

The availability of oxaloacetate for the entry of acetyl-CoA into the citric acid cycle is the cause of this.

If carbohydrate is unavailable or incorrectly utilized, citrate is formed, but the concentration of oxaloacetate is reduced. Because oxaloacetate, which is required to generate glucose, is unavailable during fasting or diabetes, the first stage of the citric acid cycle (condensation of oxaloacetate with acetyl-CoA) cannot occur. Acetoacetate and B-hydroxybutyrate are formed from acetyl-CoA under these conditions. It demonstrates the adage (proverb) "Fats Burn in the Flame of Carbohydrates."[51]

Acetoacetate, B-hydroxybutyrate, and acetone are known collectively as ketone bodies. These are energy-producing compounds that are water soluble. Acetone, on the other hand, is an exception since it cannot be digested and is easily expelled through the lungs. True ketones are acetoacetate and acetone, whereas B hydroxybutyrate lacks a keto group.[52]

Ketogenesis (Ketone Bodies Formation):

Acetoacetate formation: From acetyl-CoA, acetoacetate is produced in three steps:[53]

- Acetoacetyl-CoA is created when two acetyl-CoA molecules come together. The thiolysis stage in the oxidation of fatty acids is reversed in this reaction, which is catalyzed by thiolase.
- HMG-CoA synthase then catalyzes the condensation of acetoacetyl-CoA with another acetyl-CoA molecule to produce hydroxy-methylglutaryl-CoA (HMG-CoA).
- The mitochondrial enzyme HMG-CoA lyase then converts B-HMG-CoA to acetyl-CoA and acetoacetate.

B-hydroxybutyrate formation[54]

- The enzyme B-hydroxybutyrate dehydrogenase reduces acetoacetate in the mitochondrial matrix to produce B-hydroxybutyrate.
- In blood, the ratio of [B-hydroxybutyrate]/[acetoacetate] ranges from 1:1 to 10:1. This ratio is based on the redox state, or the ratio of mitochondrial NADH to NAD*.

Acetone production[55]

- Additionally, Acetoacetate is slowly and nonenzymatically decarboxylated to acetone.
- Total ketone body levels in healthy individuals' blood often don't rise above 0.2 mmol/L.

Utilizing ketone bodies

- The liver is the only organ where ketone bodies are made since it has an active enzymatic

mechanism for converting acetoacetyl-CoA to acetoacetate. However, the liver is unable to use ketone bodies because it lacks the specific enzyme CoA-transferase needed to activate ketone bodies.[67]

- From the liver mitochondria, acetoacetate, B-hydroxybutyrate, and acetone permeate into the circulation and are carried to peripheral tissues. These involve the conversion of B-hydroxybutyrate back to acetoacetate and the subsequent reactivation of acetoacetate to acetoacetyl-CoA.[56]

Ketone bodies activation[57]

Acetoacetate can be activated to acetoacetyl-CoA via two distinct pathways catalyzed by:

- ATP and COA-SH in the presence of thiokinase
- succinyl-CoA in the presence of COA-transferase.

Employing ketone bodies[58]

- B-hydroxybutyrate is initially converted back to acetoacetate in peripheral tissues, where it is subsequently reactivated into acetoacetyl-CoA.
- Following these processes, thiolase cleaves the acetoacetyl-CoA to produce two molecules of acetyl-CoA, which can subsequently be oxidized in the citric acid cycle to produce H, O, and CO2.
- It takes a while for acetone to undergo further metabolism. Acetone is expelled by the lungs since it is a volatile substance. Ketone body metabolism (synthesis and breakdown) is shown schematically.

Objectivity of ketogenesis[59]

- When acetyl-CoA is not being oxidized in the citric acid cycle, the liver can continue to oxidize increasing amounts of fatty acids by producing and exporting ketone bodies to extra hepatic organs. Citric acid cycle intermediates are used up during famine for gluconeogenesis, which slows down the citric acid cycle's oxidation of acetyl-CoA and diverts them to the creation of ketone bodies.
- Acetoacetate and B-hydroxybutyrate provide an alternate source of energy for extrahepatic tissues such skeletal muscle, heart muscle, renal cortex, and other organs when there is a lack of carbohydrates, such as in hunger and diabetes mellitus.
- Long-term hunger reduces the brain's need for glucose by supplying ketone bodies with 75% of its energy requirements.

- Furthermore, the liver only has a little quantity of coenzyme A, and since the majority of it is bound to acetyl-COA, B-oxidation is slowed down due to the lack of free coenzyme A. Ketone body production and export release coenzyme A, enabling ongoing fatty acid oxidation.
- Ketone bodies can be thought of as transportable, water-soluble acetyl-CoA derivatives. As a result, unlike the other lipids, they do not need to be carried by albumin or included in lipoproteins.
- Acetoacetate controls the metabolism of lipids as well. Low levels of lipolysis in adipose tissue are caused by high levels of acetoacetate in the blood, which indicates an abundance of acetyl units.

Control over ketogenesis

Ketogenesis is controlled at three crucial phases.

1. In the liver, free fatty acids serve as the building blocks for ketone bodies. Triacylglyceni in adipose tissue undergo lipolysis, which releases free fatty acids. As a result, the variables governing the release of free fatty acids from adipose tissue have a role in regulating ketogenesis.[60]
2. After being activated to acyl-CoA during liver absorption, free fatty acids have two main routes available to them:

a. mitochondrial oxidation to acetyl-CoA or ketone bodies
b. Esterification to triacylglycerol.

The entrance of acyl-CoA (active fatty acid) into mitochondria via carnitine determines which route is used. As a result, the rate-regulating step for the oxidation of fatty acids is carnitine.[61]

Prior to -oxidation, the activity of the enzyme carnitine-acyl-transferase I (CAT-I) controls the entry of long chain fatty acyl groups into mitochondria.

Its low activity in the fed state causes fatty acid oxidation to be suppressed and triacylglycerol esterification to be stimulated. Due to its increased activity during famine, triacylglycerol esterification is reduced while fatty acid oxidation increases.[62]

3. The acetyl-CoA that is produced during B-oxidation in the liver mitochondria then has two potential outcomes;

a. By way of the citric acid cycle, it might be oxidized to CO.
b. In order to create ketone bodies, it enters the ketogenesis pathway.

The availability of oxaloacetate for the production of citrate determines the entry of acetyl-CoA into the citric acid cycle. Oxaloacetate concentrations are so low that there is very little acetyl-CoA entering the citric acid pathway, which favors the synthesis of ketone bodies. If carbohydrate is unavailable or used poorly, such as when fasting or when a person has diabetes, the concentration of oxaloacetate is reduced. Acetyl-CoA is redirected to the production of acetoacetate under these circumstances.[63]

Disorders caused by ketone metabolism

Ketosis

The amount of ketone bodies in blood is typically relatively low—less than 0.2 mmol/L—but it can rise to extraordinarily high levels during fasting and in diabetes mellitus. Gluconeogenesis depletes the citric acid cycle's intermediaries under famine, directing acetyl-CoA to the synthesis of ketone bodies.[64]

Extrahepatic tissues in untreated diabetes are unable to adequately absorb glucose from the blood for use as fuel or for conversion to triacylglycerol when the insulin level is insufficient. Malonyl-CoA levels drop under these circumstances, which is a prerequisite for the production of fatty acids.[65]

Fatty acids enter the mitochondria to be converted to acetyl-CoA, which cannot pass through the citric acid cycle because the cycle's intermediates have been drawn off for use as substrates in gluconeogenesis. A decrease in malonyl-CoA levels releases the inhibition of carnitine acyltransferase-I.[66]

Beyond the capacity of extrahepatic tissues to metabolize them, the ensuing buildup of acetyl-CoA increases the ketone body production. The disease known as ketoacidosis is brought on by elevated blood levels of acetoacetate and B-hydroxybutyrate.

A coma and death may result from severe acidosis. Ketonemia, or an increase in the concentration of ketone bodies in the blood, eventually results in ketonuria, or the excretion of ketone bodies in the urine. Ketosis refers to the combined state of ketonuria and ketonemia.[67]

Ketone bodies can accumulate to levels of 90 mg/100 mL in blood (normal is 3 mg/100 mL) and 5000 mg/24 hr in urine (normal rate is 125 mg/24 hr) in patients with uncontrolled diabetes. The blood of diabetics also contains acetone along with B-hydroxybutyrate and acetoacetate. Diabetics' breath contains acetone, a highly flammable substance that has a sweet, fruity odor. Because of the breath's acetone odor, a diabetic in a coma can occasionally be mistaken for intoxicated.[68]

Ketoacidosis[69]

Increased concentrations of acetoacetate and B-hydroxybutyrate, which are both fairly potent acids, result in metabolic acidosis by lowering the pH of the blood. Ketoacidosis is the medical term for the acidosis brought on by an excess of ketone bodies. When present at high concentrations in the blood, acetoacetate and -hydroxybutyrate are buffered by the bicarbonate buffer's HCO3 (alkali) portion. Ketoacidosis is brought on by the overuse of HCO3, which depletes the alkali reserve. Ketoacidosis is common in type I diabetes mellitus but relatively uncommon in type II diabetes.

Ketoacidosis is partially treated by hyperventilation, which lowers pCO2 and lowers H2CO3concentration.

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