

Apana Vayu and Suppression of Natural Urges: Neurophysiological Mechanisms Connecting to Pelvic Autonomic Dysregulation and Disease

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Abstract

Ayurveda identifies thirteen non-suppressible natural urges (vegās), whose suppression (vega vidharana) is a primary cause of disease. Among the thirteen types of urges, defecation, urination, flatus, and ejaculation urges are governed by Apana Vayu, a sub-dosha of Vata localised in the pelvic region. The neurophysiological mechanisms underlying the signs and symptoms of non-suppressible natural urges remain underexplored. This narrative review compares classical Ayurvedic descriptions of vega vidharana with established neurophysiology, focusing on defecation, micturition, flatulence, and ejaculation reflexes. Suppression of defecation leads to constipation, headache, and abdominal distension, correlating with sustained sphincter contraction due to sympathetic overactivity. Suppression of micturition causes bladder pain and dysuria, matching inhibition of sacral parasympathetic outflow. Suppression of flatulence results in distension, pain, and cardiac discomfort from retained gases irritating the colonic myenteric plexus. Suppression of ejaculation causes genital pain, urinary obstruction, and eventual impotence, reflecting sustained sympathetic tone at T12–L2 and pudendal overactivity. Apana Vayu functions parallel to the motor outputs of sacral parasympathetic (S2–S4), lumbar sympathetic (T12–L2), and pudendal nerves. Habitual suppression produces chronic autonomic dysregulation, visceral hyposensitivity, and pelvic floor incoordination, aligning with modern understanding of neuroplastic changes in pelvic reflexes. *Apana Vayu* thus represents the integrated neural output of sacral and lumbar autonomic centres coordinating pelvic elimination, and suppression of its urges produces predictable, physiologically explainable disease patterns. Respecting natural urges is not merely traditional advice but a neurophysiologically sound preventive health measure.

Keywords: Apana Vayu, Adharniya Vega, Defecation reflex, Ejaculation, Micturition reflex, Neurophysiology, Non-suppressible urges, Vega dharana.

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Introduction

The human body is a remarkably complex system equipped with various mechanisms to balance or eliminate potentially harmful substances. To facilitate this elimination, the body generates natural urges (vegās). These urges are of two types: suppressible and non-suppressible. Suppressible urges should be intentionally controlled to prevent disease, whereas non-suppressible urges, if suppressed, lead to illness. Natural urges are vital phenomena that indicate normal biological functioning. This is a fundamental concept in Ayurveda. Although this aspect of Ayurveda is highly significant, it remains largely overlooked. Ayurveda emphasises prevention. By respecting the body's natural urges, we promote balanced mind-body health and keep diseases at bay. Vegās arise as part of normal bodily functions to fulfil physiological demands. Vegās are naturally created to help eliminate toxins produced within the body. Evacuating these urges at the right time and in the right place is essential for maintaining perfect health

and homeostasis. Therefore, eliminating urges rather than suppressing them is crucial for achieving positive health. Various lifestyles, such as shift work, stressful jobs, lack of exercise, and nightlife, often lead people to suppress natural urges frequently. Common examples include suppressing a sneeze during a meeting, ignoring hunger because of work or shyness, holding in urine while watching a favourite television show or movie, suppressing flatus or a yawn in public, or avoiding defecation because of a lack of facilities. If done repeatedly, such suppression leads to illness, and frequent illness may shorten lifespan. Occasional suppression of a Vega may be harmless, but habitual suppression harms health and can become a primary cause of sickness. Therefore, Ayurveda strongly recommends against suppressing any natural urge. These are the body's natural calls, which must be heeded as they arise to maintain bodily balance and eliminate elements that could cause imbalance. Suppression may disturb normal physiology and affect organ

systems, eventually leading to various diseases. Acharya Charak has dedicated a chapter in the Sutra Sthana on suppressible and non-suppressible urges. Acharya Vagbhata and Acharya Charak describe thirteen non-suppressible urges that should never be retained. These non-suppressible urges are: urine, defecation, sexual desires (release of semen), flatus, vomiting, sneezing, belching (eructation), yawning, hunger, thirst, tears, sleep and exertion-induced dyspnoea. Acharya Vagbhata mentions non-suppressible urges similar to Charak, except for kasa vega in place of udgaar.

While non-suppressible urges should never be suppressed, it is equally important not to forcibly initiate an urge, as this may lead to the development of another disease. Vagbhata mentioned that all diseases arise from Vega Vidharana (suppression of natural urges) and Vega Udirana (premature initiation of urges). One must always heed these urges, as suppressing them can lead to various health problems. If a constipated person forcibly induces the defecation reflex, it may lead to a pile-like condition. In this paper, we are exploring the physiological mechanisms and disease development related to the suppression of urges such as flatus, defecation, micturition, and semen. These urges arise from the normal functioning of apana vayu. The location and function of apana vayu, as described by various acharyas, are listed in the Table 1.

Table 1: Anatomical Location and Functions of Apana Vayu¹

	Charak Samhit a ²	Sushruta Samhit a ³	Ashtanga Hridaya ⁴	Ashtanga Sangraha ⁵
Location	Vrishan a (testicles), Vasti (urinary bladder), Medhra (penis), Nabhi (umbilicus), Uru (thighs), Vakshyana (inguinal region) and Guda(anus)	Pakvadhana (Large intestine)	apana desha (perineal region) and it traverses along sroni (pelvis), vasti (urinary bladder), medhra (external genital apparatus of each	Rectum, moves along the urinary bladder, penis, scrotum, and groin

			sex) and uru (thighs)	
Function	Ejaculation, Micturition, defecation, expulsion of menstrual blood and fetus.	Elimination of samirana (flatus), sakrit (faeces), mutra (urine), sukra (semen), garbha (fetus), artava (menstrual fluid)	Expulsion of sukra (semen), artava (menstrual blood), sakrit (faeces), mutra (urine), and garbha (the product of conception that is fetus and placenta)	Elimination of faeces, urine, semen, menstrual fluid and foetus

The somatosensory pathway plays a pivotal role in the perception and conscious awareness of Adharniya Vega (non-suppressible urges) such as the urge to defecate, micturition, flatus, and ejaculation. These visceral urges begin when mechanical or chemical stimuli, such as distension of the bladder, rectum, or seminal vesicles, activate specialised mechanoreceptors and nociceptors in the walls of these hollow organs. The resulting sensory signals are transmitted via first-order afferent neurons travelling through the pelvic, hypogastric, and pudendal nerves to the dorsal root ganglia and subsequently to the spinal cord (sacral and lumbar segments). From there, second-order neurons ascend through the spinothalamic tracts to the thalamus, where the signals are relayed to the somatosensory cortex and insular cortex for conscious perception of urge intensity, location, and unpleasantness. This entire pathway, from peripheral receptor to cortical awareness, enables the individual to recognise and respond to the body's natural calls⁶.

In the context of Adharniya Vega, the conscious suppression of these urges driven by social, occupational, or psychological factors involves top-down cortical inhibition of the spinal reflex arcs. The cerebral cortex (especially the prefrontal cortex and anterior cingulate cortex) exerts inhibitory control over the sacral parasympathetic outflow, the somatic pudendal nerve, and the sympathetic

hypogastric plexus, thereby overriding the autonomic spinal reflexes that would normally facilitate elimination. However, habitual or prolonged suppression disrupts the normal somatosensory processing by causing desensitisation of stretch receptors, sustained sympathetic tone, and chronic pelvic floor tension, leading to altered afferent signaling and pathological feedback loops. This neurophysiological interference manifests as the classical symptoms described in Ayurveda—pain, distension, obstruction, and systemic disturbances.

1. The Neurophysiology of the Ejaculation Reflex

Emission and ejaculation are the culmination of the male sexual act. When the sexual stimulus becomes extremely intense, the reflex centres of the spinal cord begin to emit sympathetic impulses that leave the cord at T12 to L2 and pass to the genital organs through the hypogastric and pelvic sympathetic nerve plexuses to initiate emission, the forerunner of ejaculation.

Emission: Emission begins with contraction of the vas deferens and the ampulla to cause expulsion of sperm into the internal urethra. Then, contractions of the muscular coat of the prostate gland, followed by contractions of the seminal vesicles, expel prostatic and seminal fluid into the urethra, thereby forcing the sperm forward. These fluids mix in the internal urethra with mucus already secreted by the bulbourethral glands to form semen. The process to this point is emission.

Ejaculation: The filling of the internal urethra with semen elicits sensory signals that are transmitted through the pudendal nerves to the sacral regions of the cord, giving the feeling of sudden fullness in the internal genital organs. These sensory signals also excite rhythmical contraction of the internal genital organs and cause contraction of the ischiocavernosus and bulbocavernosus muscles, which compress the bases of the penile erectile tissue. These effects together cause rhythmical, wavelike increases in pressure in both the erectile tissue of the penis and the genital ducts and urethra, which “ejaculate” the semen from the urethra to the exterior. This final process is called ejaculation⁷.

1.1 Suppression of Urge of Ejaculation

Suppression of Vega of Shukra (Semen) results in Medra & vrushan Shool (Swelling and pain in the genital parts, especially in penis & testicles), Angamarda (body pain), Hridhya pida (discomfort in the region of the heart), Mutraavarodha⁸ (obstruction during urination), mushkshophya (enlargement of the scrotum), formation of Shukrashamari⁹ (seminal calculi) and over a period of time, the person may suffer from sandata¹⁰ (impotency). These symptoms result from the suppression of Vega of Shukra. Suppression of sex desires causes nervous diseases like neurosis and psychological maldevelopment¹¹.

1.2 Medra & Vrushan Shool (Pain in Penis and Testicles)

Mechanism: Suppression of ejaculation leads to sustained sympathetic stimulation via the hypogastric nerve (T12–L2), causing prolonged contraction of the vas deferens, epididymis, and seminal vesicles. This results in congestion of the genital organs with blood and seminal fluid. Continued vasocongestion causes local venous stasis, increased hydrostatic pressure, and tissue oedema, leading to swelling and pain in the penis (Medra Shool) and testicles (Vrushan Shool). The unrelieved distension of the reproductive ducts activates visceral nociceptors, generating pain signals transmitted via the pelvic and hypogastric plexuses¹².

1.3. Angamarda (Bodyache)

Mechanism: The sustained sympathetic outflow and pelvic muscle tension during suppression of ejaculation create a state of generalised neuromuscular tension. Afferent nociceptive signals from the congested pelvic organs lead to central sensitisation and referred pain patterns. Additionally, the build-up of metabolic waste products (e.g., lactic acid) in skeletal muscles from sustained isometric contraction of the pelvic and perineal muscles contributes to generalised body ache (Angamarda). Prolonged sympathetic activation also releases inflammatory mediators like prostaglandins, which may induce systemic myalgia¹³.

1.4. Hridhya vyatha (Pain in cardiac region)

Mechanism: Suppression of ejaculation triggers intense sympathetic nervous system activation (T12–L2 outflow), leading to the release of catecholamines (epinephrine and norepinephrine). These hormones increase heart rate, cardiac contractility, and blood pressure via beta-adrenergic stimulation. The resulting tachycardia, palpitations, and chest discomfort constitute Hridhya vyatha¹⁴. Additionally, vagal afferent signals from congested pelvic organs can trigger reflex cardiac autonomic disturbances that mimic cardiac distress. This is not organic heart disease but a neuro-autonomic phenomenon.

1.5. Mutraavarodha (Retention of urine)

Mechanism: The prostate gland and urethral sphincters share sympathetic and somatic innervation with the reproductive organs. During ejaculation suppression, sustained sympathetic tone maintains contraction of the internal urethral sphincter (via the hypogastric nerve) and of the smooth muscle of the prostate. Simultaneously, the pelvic floor muscles (bulbocavernosus and ischiocavernosus) remain contracted via pudendal nerve stimulation. This coordinated contraction, intended to prevent semen outflow, mechanically compresses the prostatic urethra, resulting in partial or complete obstruction during urination¹⁵ (Mutraavarodha). Chronic suppression desensitises

stretch receptors, causing the sympathetic nervous system to relax (slightly) the detrusor muscle and contract the trigone/internal urethral sphincter, promoting urine storage and inhibiting micturition¹⁶.

1.6. Mushkshoppa¹⁷ (Inflammation in both testicles)

Mechanism: Chronic suppression of ejaculation leads to persistent congestion of the epididymis, spermatic cord, and testicular venous plexus (pampiniform plexus). Unrelieved vascular engorgement causes fluid transudation into the interstitial spaces, resulting in scrotal oedema and enlargement. Additionally, prolonged sympathetic vasoconstriction followed by reactive vasodilation contributes to the pooling of blood and lymphatic fluid in the scrotal tissues¹⁸.

1.7. Shukrashamari¹⁹ (Formation of Seminal Concretions)

Mechanism: Retention of seminal fluid for prolonged periods causes stagnation, leading to supersaturation of its constituents fructose, proteins, zinc, and citric acid. This promotes precipitation and crystallisation within the seminal vesicles, vas deferens, and prostatic ducts. Over time, these crystallised deposits aggregate to form microscopic or macroscopic concretions²⁰ (Shukrashamari). These calculi may later cause obstruction, pain, and infertility.

1.8. Sandata²¹ (Impotency)

Mechanism: Over time, habitual suppression of ejaculation or habitual excessive ejaculation leads to neurovascular fatigue, desensitisation, and impotency. Chronic sympathetic overactivity results in downregulation of adrenergic receptors and impaired nitric oxide (NO) mediated vasodilation, essential for penile erection. Persistent congestion and microinflammation may cause fibrosis of the corpora cavernosa²². Psychologically, repeated suppression may lead to performance anxiety and loss of libido. Neurogenic factors include pudendal nerve dysfunction and altered spinal reflex excitability. Collectively, these lead to erectile dysfunction, termed Sandata.

2. The Neurophysiology of the Defecation Reflex

When stool enters the rectum, it immediately prompts the urge to defecate. This occurs through the coordinated action of two distinct reflexes. The first is a built-in reflex controlled by the enteric nervous system. As stool enters and stretches the rectal walls, sensory signals are picked up by the myenteric plexus. This triggers a series of wave-like muscle contractions that push stool downward from the descending colon, through the sigmoid colon, and into the rectum. These contractions also signal the internal anal sphincter an involuntary muscle to relax. At this point, if consciously relax the external anal sphincter, which is under voluntary control, defecation happens naturally. The second reflex involves the parasympathetic nervous system—the part responsible for rest and digestion. When stool

stretches the rectum, nerve endings send signals up to the spinal cord through sensory nerve fibres. The spinal cord then sends reflex signals back down through the pelvic nerves to the descending colon, sigmoid colon, and rectum. These parasympathetic signals powerfully amplify the muscle contractions and further relax the internal anal sphincter. Again, defecation occurs when voluntarily relax the external anal sphincter.

Meanwhile, sympathetic nerve fibres from the superior rectal and hypogastric plexuses act in the opposite direction, stimulating and maintaining contraction of the internal anal sphincter, helping to hold stool when the timing isn't right. The external anal sphincter, which can be consciously controlled, is innervated by the somatic pudendal nerve, allowing voluntary control over when to release or retain stool²³.

2.1 Suppression of the urge to Defecate

A person who suppresses the urge to defecate (Vega of Purisha) develops Pakwashaya Shool (colic pain), Shiroshool (headache), Vata apravarthanam (Retention of flatus), Purish apravarthanam (Retention of stool), Pindiko dveshteana (cramps in calf muscles) and adhmanam²⁴ (distension of abdomen). Bad odour from the mouth (Halitosis) and sometimes Mukhen malapravrutti²⁵ (stool comes out from mouth). These symptoms are caused by suppression of Vega of Purisha.

2.2 Pakwashaya Shool (Colic pain)

Mechanism: Suppression retains faecal matter in the Pakwashaya (large intestine), causing its distension. This activates visceral stretch receptors and mechanoreceptors, transmitting pain signals via afferent fibres (sympathetic) to the spinal cord²⁶. Retained faeces also cause sustained contraction of the intestinal smooth muscle (spasm), leading to colicky or aching pain.

2.3 Shiroshool (Headache)

Pain and distension caused by fecal retention activate the vagus nerve, triggering reflex vasodilation of cerebral blood vessels. At the same time, abdominal discomfort can stimulate sympathetic responses, leading to peripheral vasoconstriction and slight hypertension. The interplay of these changes in cerebral blood flow and reflex mechanisms results in a tension-type or vascular headache²⁷.

2.4. Vata Apravarthanam (Retention of Flatus)

Mechanism: Suppressing defecation involves voluntary contraction of the external anal sphincter (via the pudendal nerve). This mechanically prevents flatus expulsion. Additionally, retained faeces physically obstruct the rectal ampulla, creating a barrier. The colonic myenteric plexus continues peristalsis, but gas accumulates, leading to obstruction of flatus passage²⁸ (Vata Apravarthanam).

2.5. Purish Aapravarthanam (Retention of faeces)

Mechanism: Repeated suppression inhibits the parasympathetic sacral outflow (pelvic nerves) that stimulates defecation reflexes. The rectal wall accommodates to chronic distension, reducing stretch receptor sensitivity. The internal anal sphincter remains contracted due to unopposed sympathetic tone. Faeces become harder and drier as water is absorbed. This combination results in constipation²⁹(Purish Aapravarthanam).

2.6. Pindikō Dveshteana (Cramps in Calf Muscles)

Mechanism: This is a classic example of referred muscle spasm. Prolonged straining and voluntary pelvic muscle contraction can alter lumbosacral spinal reflexes. Also, electrolyte imbalances (e.g., mild hypokalemia) from altered colonic absorption and the shared spinal innervation (L4–S2) of the pelvic organs and calf muscles may cause reflex muscle cramping³⁰.

2.7 Aadhmanam (Distension of Abdomen)

Mechanism: Retention of faeces and flatus leads to accumulation of intraluminal contents. Colonic bacteria ferment undigested carbohydrates, producing gases³¹ (carbon dioxide, hydrogen, methane). When expulsion is blocked and absorption is overwhelmed, the colon and small intestine distend, leading to visible abdominal distension (Aadhmanam).

2.8 Bad Odour from the Mouth (Halitosis)

Mechanism: Prolonged retention leads to bacterial putrefaction of faeces, producing foul-smelling compounds like indole, skatole, and hydrogen sulfide³². These volatile substances are absorbed into the portal circulation and, bypassing complete hepatic metabolism, are excreted via the lungs in exhaled air, resulting in halitosis³³.

2.9 Mukhen Vitpravrutti (Stool Comes Out from Mouth – Faecal Vomiting)

Mechanism: This is a sign of severe, advanced intestinal obstruction. Chronic suppression causes complete mechanical obstruction in the large intestine. The colon cannot accommodate more contents, initiating reverse peristalsis (retrograde contractions). Intestinal contents are forced proximally, distending the small intestine until the gastroesophageal sphincter fails. This culminates in feculent vomiting³⁴, a medical emergency indicating impending bowel perforation or paralytic ileus.

3. The Neurophysiology of the Micturition Reflex

The micturition reflex is initiated by the stretch reflex. When the bladder fills with urine and pressure rises, sensory stretch receptors in the bladder wall are stimulated and send signals to the sacral segment of the cord through the pelvic nerve. Then it reflexively sends signals back to the bladder via parasympathetic motor fibres, triggering micturition. It involves coordination among the central, autonomic, and somatic nervous systems³⁵. The micturition reflex is an autonomic spinal cord reflex, but it can be inhibited and facilitated by the

pons and several centres in the cerebral cortex. The smooth muscle of the bladder (detrusor) and trigone are innervated by both sympathetic nerve fibres from the lumbar spinal cord and parasympathetic fibres from the sacral spinal cord. Stimulation of sympathetic fibres causes contraction of the trigone and relaxation of the detrusor muscle. Stimulation of parasympathetic fibres causes contraction of the detrusor muscle and relaxation of the trigone. The urethral sphincter is innervated by the somatic pudendal nerve. These nerves coordinatively help in micturition³⁶.

3.1 Suppression of Urge of Micturition

Suppressing the vega of mutra causes pain in the body, especially Basti Shool (pain in the bladder), Mehana Shoola (pain in the phallus), Mutrakruchata (Dysuria), Shiroruja (headache), Vinama (bending of the body), and Anaha³⁷ (distension of the abdomen). Over time, this person may develop bladder and urinary tract diseases. These symptoms result from suppression of Vega of Mutra.

3.2 Basti Shool (Pain in the Bladder)

Mechanism: Suppression of micturition causes continued filling and distension of the urinary bladder beyond its normal capacity (~300–400 mL). Stretch receptors in the bladder wall (detrusor muscle) are excessively stimulated, sending intense afferent signals via the pelvic nerve (S2–S4) to the sacral spinal cord. The sustained distension activates visceral nociceptors, generating pain perceived in the suprapubic and bladder region (Basti Shool). Prolonged stretching also causes detrusor ischemia due to compression of intramural blood vessels, releasing inflammatory mediators (e.g., substance P, prostaglandins) that further sensitise pain fibres³⁸.

3.3 Mehana Shoola (Pain in phallus)

Mechanism: Voluntary suppression of micturition involves sustained contraction of the external urethral sphincter via the pudendal nerve (somatic, S2–S4) and continued contraction of the internal urethral sphincter via sympathetic stimulation (hypogastric nerve, T12–L2). This sustained sphincter spasm compresses the urethral mucosa and surrounding tissues, causing ischemic pain and discomfort along the penile urethra³⁹ (Mehana Shoola). Additionally, increased intravesical pressure is transmitted to the posterior urethra, stimulating nociceptors in the urethral wall.

3.4 Mutrakruchata (Dysuria)

Mechanism: Chronic or repeated suppression of micturition leads to detrusor muscle overdistension and subsequent hypocontractility. The stretch receptors become desensitised, reducing the efficiency of the micturition reflex arc (pelvic nerve afferents → sacral parasympathetic efferents). The external and internal urethral sphincters remain in a state of relative spasm due to persistent voluntary and sympathetic tone. When the person eventually attempts to void, the detrusor contraction is weak, and sphincter relaxation is incomplete, resulting in

straining, poor stream, and difficulty in initiating or completing urination (Mutrakruchata). This may progress to acute or chronic urinary retention⁴⁰.

3.5 Shirorooja (Headache)

Mechanism: Retained urine causes progressive bladder distension, which activates the pelvic and hypogastric afferents, generating intense visceral pain signals. This noxious stimulation triggers a viscerosomatic reflex, leading to sympathetic activation and peripheral vasoconstriction. Simultaneously, vagal afferents from the distended bladder can cause reflex cerebral vasodilation. The combination of altered cerebral blood flow, autonomic imbalance, and referred pain pathways from pelvic to cranial autonomic centres results in headache (Shirorooja). In severe cases, acute urinary retention can cause hypertension and autonomic dysreflexia (in susceptible individuals), further exacerbating headache⁴¹.

3.6 Vinama (Bending of the body)

Mechanism: Severe discomfort, suprapubic pain, and urinary urgency lead the individual to adopt a protective posture-bending forward (Vinama). This position reduces intra-abdominal pressure on the bladder and provides some relief by relaxing pelvic floor muscles and reducing tension on the vesical ligaments. In cases of acute retention, the palpable distended bladder causes visible lower abdominal bulging, and the patient assumes a stooped posture to alleviate pain. This posture is an involuntary musculoskeletal response to severe visceral pain⁴².

3.7 Anaha (Distension of the lower Abdomen)

Mechanism: Retention of urine causes significant bladder enlargement, which displaces intestinal loops upward and laterally, leading to visible lower abdominal distension (Anaha). Simultaneously, bladder distension and pain can cause reflex inhibition of gastrointestinal motility via sympathetic overactivity (via splanchnic nerves), thereby reducing intestinal peristalsis and gas transit. This results in accumulation of intestinal gas and contents, contributing to abdominal bloating and distension. Severe urinary retention with a distended bladder can mimic or coexist with intestinal obstruction⁴³.

3.8 Diseases of Bladder and Urinary Tract (Long-term Complications)

Mechanism: Habitual suppression of micturition predisposes to chronic urinary stasis, which allows bacterial growth in the residual urine, leading to recurrent urinary tract infections (cystitis, urethritis). Chronic overdistension causes detrusor muscle damage, fibrosis, and loss of compliance, resulting in a hypotonic or atonic bladder. Sustained high intravesical pressure can cause vesicoureteral reflux, hydronephrosis, and eventually renal damage. Stagnant urine promotes crystal formation and stone development (vesical calculi). Prolonged sphincter spasm leads to detrusor-sphincter dyssynergia, further worsening voiding dysfunction⁴⁴. These

collectively constitute the spectrum of "diseases of the bladder and urinary tract".

4. Neurophysiology of the Flatulence Reflex

Gases can enter the gastrointestinal tract from three sources: 1. Swallowed air 2. Gases formed in the gut because of bacterial action. 3. Gases that diffuse from the blood into the gastrointestinal tract. Colonic bacilli are present in the absorbing colon. An average of 7-10 litres of gas forms in the large intestine every day, whereas an average of 0.6 litres is expelled through the anus. The remainder is absorbed through the intestinal mucosa into the blood and expelled through the lungs. Bacterial activity produces vitamin K, vitamin B12, thiamine, riboflavin, and gases such as carbon dioxide, hydrogen, and methane, which contribute to flatus⁴⁵. Excessive gas expulsion results from irritation of the large intestine, which promotes rapid peristaltic expulsion of gas through the anus before it can be absorbed. Due to gastrointestinal motility, flatus is eliminated through the anus. The myenteric plexus controls this gastrointestinal motility and is strongly stimulated by the parasympathetic nervous system⁴⁶. The actual odour of flatus is due to gases such as indole, skatole, mercaptans, and hydrogen sulphide.

4.1 Suppression of the urge to pass flatulence

Suppression of Vega of Apana vayu causes Mala avrodha (obstruction to pass flatus and faeces), Mutra rodha (urine retention), Aadhmanam (distension of abdomen), Vedana (pain), Klama⁴⁷ (fatigue), Udavarta (retrograde intestinal movements), Drstivadha (loss of vision), Agnivadha (loss of digestion) and Hrdroga⁴⁸ (epigastric diseases). These symptoms result from Apana vayu suppressing Vega.

4.2 Vin and Vata Sang (Retention of faeces and flatus)

Mechanism: Voluntary suppression of flatus involves sustained contraction of the external anal sphincter (via pudendal nerve) and internal anal sphincter (via sympathetic fibres from hypogastric plexus). The retained faeces physically obstruct the rectal ampulla, creating a mechanical barrier. The colonic myenteric plexus continues to generate peristaltic waves, but the combination of sphincter spasm, faecal impaction, and loss of rectal stretch receptor sensitivity prevents expulsion of both flatus and faeces (Mala Avrodha). This leads to progressive constipation and inability to pass gas⁴⁹.

4.3 Mutra Sang (Retention of Urine)

Mechanism: Severe distension of the colon and rectum from retained flatus and faeces causes mechanical compression of the urinary bladder. This compression reduces bladder capacity and interferes with normal detrusor contraction. Additionally, the shared sacral parasympathetic innervation (pelvic nerves, S2–S4) for both defecation and micturition results in reflex inhibition of bladder emptying when colonic distension is excessive⁵⁰. Sustained pelvic floor muscle tension (from anal sphincter

contraction) further compresses the urethra, making voiding difficult. This results in urinary retention⁵¹ (Mutra Rodha).

4.4 Aadhmanam (Distension of Abdomen)

Mechanism: Retention of flatus and faeces causes progressive accumulation of gases and intraluminal contents in the colon. Colonic bacteria ferment undigested carbohydrates, producing gases like carbon dioxide, hydrogen, and methane. When expulsion is blocked and absorption through the intestinal mucosa is overwhelmed, gases accumulate, causing progressive distension of the colonic and small intestinal walls (Aadhmanam). Stretching of the bowel wall activates mechanoreceptors and stretch receptors, further inhibiting normal peristalsis through the enterogastric reflex and perpetuating the cycle of distension⁵².

4.5 Vedana (Pain)

Mechanism: Retained flatus and faeces cause progressive colonic distension, activating visceral stretch receptors and mechanoreceptors. This generates intense afferent signals via sympathetic (splanchnic, hypogastric) and parasympathetic (pelvic) nerves to the spinal cord. The resulting pain is perceived as colicky or cramping abdominal pain (Koshtashool). Additionally, increased intraluminal pressure compromises mucosal blood flow, causing ischemia and the release of inflammatory mediators (e.g., prostaglandins, substance P) that sensitise nociceptors, thereby exacerbating pain⁵³.

4.6 Klama (Fatigue)

Mechanism: Prolonged abdominal distension and pain from retained flatus stimulate the sympathetic nervous system (splanchnic nerves) and activate the hypothalamic-pituitary-adrenal (HPA) axis, leading to increased cortisol release. Chronic visceral nociceptive input causes central fatigue by activating descending inhibitory pathways⁵⁴. Additionally, impaired gastrointestinal motility reduces nutrient absorption and alters the gut microbiome, potentially causing mild anaemia or vitamin deficiencies. The combination of sympathetic overactivation, neuroendocrine stress response, and impaired nutrition results in generalised debility and fatigue (Klama).

4.7 Udavarta (Retrograde Intestinal Movements)

Mechanism: Suppression of flatus prevents the normal downward (apana) movement of gases through the colon. The retained gases, along with intestinal contents, increase intraluminal pressure, stimulating the myenteric plexus and initiating reverse peristalsis (retrograde contractions). This abnormal upward movement of gas and intestinal contents from the descending colon toward the transverse colon, jejunum, and even the stomach causes pain, distension, and a sensation of "upward movement of vayu" (Udavarta). The retrograde propagation of peristaltic waves is mediated by altered enteric neural signaling, where inhibitory

neurotransmitters (e.g., nitric oxide, VIP) are overwhelmed by excitatory cholinergic activity in the proximal direction⁵⁵.

4.8 Agnivadha (Loss of digestion)

Mechanism: Intestinal distension and faecal retention activate the enterogastric reflex, which inhibits gastric motility and emptying. The distended colon stimulates vagal afferents that send inhibitory signals to the gastric fundus and antrum, reducing gastric contractions and delaying gastric emptying. This results in early satiety, anorexia, and a feeling of fullness. Chronic constipation also alters the gut microbiome, reduces ghrelin secretion, and increases leptin, further suppressing appetite⁵⁶. The suppression of the "digestive fire" (Agni) is a direct consequence of impaired gastrointestinal motility.

4.9 Hrdroga (Epigastric distress)

Mechanism: Severe abdominal distension from retained flatus and faeces increases intra-abdominal pressure, which is transmitted to the thoracic cavity through the diaphragm. This reduces venous return (preload) and compresses the inferior vena cava, decreasing cardiac output. Reflex sympathetic activation causes tachycardia and hypertension. In severe cases, the vagal stimulation from extreme colonic distension can cause bradyarrhythmias, hypotension, and even syncope. Chronic constipation and straining (Valsalva manoeuvre during attempted defecation) can precipitate angina, arrhythmias, or myocardial infarction in susceptible individuals⁵⁷. This is the classical explanation for Hridayaroga-cardiac discomfort or disease resulting from chronic suppression of Apana Vayu.

Discussion

This review clarifies the neurophysiological basis for the Ayurvedic concept of Apana Vayu and the potential health issues that may arise from suppressing its associated urges. The main finding is that the functions attributed to Apana Vayu—such as the expulsion of faeces, urine, flatus, and semen—correlate precisely with the integrated motor and autonomic outputs from the sacral (S2–S4) and lumbar (T12–L2) spinal segments, which are mediated by the pelvic, hypogastric, and pudendal nerves. Therefore, Apana Vayu can be understood not as a metaphysical concept, but as a functional representation of the pelvic autonomic-somatic neural network. The analysis illustrates a consistent pathophysiological mechanism underlying Vega Vidharana: habitual voluntary suppression disrupts autonomic spinal reflexes, leading to chronic dysregulation. During defecation, micturition, flatulence, and ejaculation, persistent cortical inhibition sustains sphincter contraction and sympathetic tone while suppressing the necessary parasympathetic outflow required for proper elimination or release. This results in predictable outcomes, including visceral distension, smooth muscle spasm, congestion, and receptor desensitisation.

The Ayurvedic principle that "occasional suppression is harmless but habitual suppression is pathogenic" is supported by modern understanding of neuroplasticity and autonomic adaptation. Chronic suppression alters afferent signalling and reflex excitability, establishing pathological feedback loops that manifest as functional gastrointestinal and urological disorders. This integrative framework has important clinical implications. It suggests that many common pelvic floor and autonomic disorders may be partially preventable through simple behavioural interventions, namely respecting and responding to natural urges. Moreover, it provides a neurophysiological rationale for incorporating Ayurvedic principles into preventive medicine and rehabilitation strategies, such as biofeedback and pelvic floor training.

Conclusion

This comprehensive review establishes a robust and scientifically coherent connection between the Ayurvedic concept of Apana Vayu and the principles of neurophysiology. The evidence strongly supports the interpretation of Apana

Vayu as the integrated neural output of the pelvic autonomic and somatic nervous systems, orchestrating the essential functions of elimination and reproduction. The suppression of its associated urges (Adharniya Vega), specifically defecation, micturition, flatulence, and ejaculation, produces predictable and clinically recognisable disease patterns that are entirely explicable through modern physiological mechanisms. The symptoms described in classical texts are not empirical observations of disease but are direct manifestations of chronic autonomic dysregulation, sustained muscle tension, visceral congestion, and impaired reflex coordination, driven by a persistent override of brainstem and spinal reflexes by higher cortical centres. The convergence of Ayurvedic wisdom and modern physiology presented here offers a powerful model for integrating traditional knowledge into contemporary medical practice. It validates the Ayurvedic principle that respecting the body's natural calls is a fundamental aspect of preventive medicine.

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