



BOWEL NO 4 - FUNCTIONAL GASTRO-INTESTINAL DISORDERS V DYSFUNCTIONAL HUMAN BEINGS; MICROMANAGEMENT OF MILIEU INTERIOR PART I – ALEA IACTA EST - THE DIE HAS BEEN CAST

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ABSTRACT

Humans are suffering from Nonorganic Gastro-Intestinal Disorders (NGIDs) classified as “Functional Gastro-Intestinal Disorders (FGIDs)”. The controversies, quality, nature and size of FGIDs including the view by Katzka et al are defined. Variability between different cohorts and their definition of symptom complex is addressed under “calling spade a spade” section, follows the current concerns effervesce. It follows brief sketch of investigations and management issues, precise explanation of gas/bloating, Ayurvedic and Yoga management is introduced. Symptoms of GERD and IBS frequently overlap.

KEYWORDS: Functional Gastro Intestinal Disorders – FGIDs, Disorders of Gut-Brain Interactions – DGBIs, Functional Dyspepsia – FD, Irritable Bowel Syndrome – IBS; Diverticular Disease – DD; Symptomatic Uncomplicated Diverticular Disease – SUDD.

(1) INTRODUCTION

Humans are allegedly beseeched by functional gastro-intestinal disorders (FGIDs); but are there FGIDs or dysfunctional human beings (DHBs) contracting FGIDs? This issue deals with nature and controversies on FGIDs raised by different authors, calling spade a spade with varied views by various authors including effervescing concerns and medically unexplained symptoms (MUS). Burkitt’s experience in Africa (1971), allodynia symptoms, current concerns on role of dietary fiber in Westernised society, various investigating methods are briefly described as their role is limited in FGIDs; clinical phenotype of Gas and bloating syndrome has been discussed as non-issue. Age-old biophysiology of gas phenotype explaining the Ayurvedic concept and definition of gas (Vāyu) with its five types of classifications and their functions has been briefly stated including positive role of *Apāna Vāyu* in FGIDs. Role of ‘*Yoga Āsan*’ is introduced in improving and ameliorating

FGIDs, besides its health related quality of life (HRQOL).

(2) Nature and controversy about FGIDs

This has aroused vast interest in FGIDs since 1980s as its scourge increased. Sperber *et al*^[1] put it, “The functional gastro-intestinal disorders (FGIDs)... are gastro-intestinal disorders (GIDs) allegedly related to any combination of motility disturbance, visceral hypersensitivity, altered mucosal and immune function, altered gut microbiota and altered central nervous system processing... Published studies have involved highly variable diagnostic criteria... For irritable bowel syndrome (IBS) and functional dyspepsia (FD), the 2 most researched disorders, reported prevalence estimates are very broad (1.1%–45.0% for IBS, and 1.8%–57.0% for FD). Unquote.” The microorganic nature of FGIDs arising from systemic, metabolic or motility disorders, gut dysbiosis, inflammation, etc, is not addressed in Rome IV criteria, (Ray G and Ghoshal^[2]). Bergh *et al*^[3]

postulate, “The relationship between conscious experience of physical symptoms and indicators of objective physiological dysfunction is highly variable and depends on characteristics of person, context and their interaction. This relationship often breaks down... in case of ‘medically unexplained’ or functional somatic symptoms violating basic assumption in medicine that physical symptom has pathophysiological causes. Unquote.” Yet another group, viz, Oshima and Miwa^[4] observed contrarily, “Functional dyspepsia (FD) and irritable bowel syndrome (IBS) are among the most widely recognized FGIDs. It is characteristic of the FGIDs that each entity has no specific objective findings and that they overlap with each other. Therefore, these entities are syndromes, and their diagnosis and treatment are similar. Unquote” Mittal in his Commentary^[5], “Montreal, Rome, and Lyon Consensus - Will They Resolve the Conundrum of Gastroesophageal Reflux Disease”, observed, “With the advent of PPIs, the relationship between erosive reflux disease and acid reflux has become clear, the dictum “no acid no esophagitis” is indeed true. However, what is not true is “no acid no heartburn/or other presumed symptoms of GERD.” In patients with true nonerosive disease, that is, symptoms and abnormal reflux parameters on pH testing, the relief of symptoms is the same as in patients with erosive esophagitis. However, uncontrolled studies found that in patients with acid-hypersensitive esophagus, improvement in symptoms was barely above placebo. Reflux hypersensitivity is defined as positive association between symptoms of heartburn with acid reflux on pH monitoring; however, an association between the 2 does not establish that heartburn is caused by acid. It might very well be that the acid is an “innocent bystander” at the time of heartburn. Unquote.” Mittal’s view may appear polemic; it does show a logical path to work on, which might become torchbearer to solve the FGIDs’ conundrum; the die is cast (*alea iacta est*). Mittal^[5] continues, “Among 3229 participants taking daily PPIs, 54% had persistent GERD symptoms.” He^[5] continues, “Most of the epidemiological studies used a standardized questionnaire that equated heartburn and regurgitation symptoms with GERD, a misunderstanding that was initiated and is being perpetuated by Montreal definition of GERD.... Studies have found that a large number of patients with typical esophageal GERD symptoms did not have GERD when they were assessed with objective testing. This is even more true (sic) for extra-esophageal symptoms of GERD, such as angina-like esophageal pain, worsening or difficult-to-treat asthma, posterior laryngitis, chronic cough, dental erosions, and disordered sleep... Adequate PPI therapy healed esophagitis in >95% of patients with erosive disease, but it did not result in relief of heartburn in a proportionally similar number of patients. Forty percent of patients with Barrett’s esophagus (severe form of GERD) have no history of heartburn or other GERD symptoms. These observations provide an important clue that symptoms of heartburn should not be considered synonymous with GERD, which is true even in patients with erosive

esophagitis... With the advent of PPIs, relationship between erosive reflux disease and acid reflux has become clear, the dictum “no acid no esophagitis” is indeed true. However what is not true is no acid no heartburn/or other presumed symptoms of GERD... Contractions of the circular and longitudinal muscle of esophagus reduce blood flow to the esophageal wall and it may be that *relative ischemia* of esophageal wall is the cause of a heartburn sensation. Distention of esophagus induced by reflux contents is another possible cause of heartburn and chest pain. Distention of esophagus by a balloon induces heartburn and chest pain at a low and high degree of distention, respectively... Studies conducted in late 1980s and early 1990s clearly proved that there was poor correlation between chest pain/heartburn symptoms and recordable events by manometry and pH monitoring in patients with esophageal motility disorders... What will resolve the conundrum of GERD? Symptom-based definition of GERD is a major limitation of epidemiological studies and the majority of data suggest that PPI-resistant heartburn is not true GERD. Esophageal events, such as sustained contraction of longitudinal muscles of esophagus, low perfusion of esophageal wall, esophageal distention, and possibly other events in periphery (esophagus) that are not recorded by clinically used monitoring techniques, can cause symptoms that are also associated with classical GERD and acid reflux... Patients not responding to PPI therapy are currently labeled as recalcitrant GERD or nonresponsive GERD, and often end up having one or the other types of antireflux surgery if pH monitoring studies conducted off PPI therapy show abnormal reflux scores in these patients. Ironically, when studied on PPI therapy, majority of these patients do not have any acid reflux. I propose that the correct term to use for these patients is *recalcitrant heartburn* rather than recalcitrant GERD and these patients should not be subjected to antireflux surgery. Only patients with troublesome regurgitation, and pulmonary symptoms suspected to be due to GER, with evidence of GER on pH monitoring off PPI therapy, are appropriate candidates for antireflux surgery. (Author’s caveat: No surgery at all!). I used the term suspected because there are no good tests to prove whether GERD is indeed the cause of pulmonary symptoms, even if these patients are found to have reflux on pH monitoring of esophagus... The consensus meetings held in great cities like Montreal, Rome, and Lyon will not resolve the conundrum of GERD. What is required is the realization that heartburn, esophageal pain, and regurgitation are visceral symptoms and, unlike somatic symptoms, they are not stimulus-specific (ie, GERD or acid).... Progress in the field will only occur if we start calling spade a spade, that is, stop calling every heartburn, esophageal pain, regurgitation, and extraesophageal symptom GERD or a phenotype of GERD. Only diligent and innovative research into better understanding of the genesis of heartburn, esophageal pain and other GERD symptoms will advance the field that afflicts 10%–30% of population worldwide and

consumes hundreds of billions of dollars. Unquote.” The spade is hidden in faulty Western lifestyle. Kahrilas^[6] lamented his dismay for ‘refractory heartburn’ in these words, “From a management viewpoint, the objective is to reduce the patient’s symptom burden, irrespective of causality. However at this point we have only a failed management strategy and no diagnosis. Unquote.” Kahrilas^[7] cautioned further, “These patients with symptomatic GERD (called nonerosive reflux disease and endoscopy-negative reflux disease) present a

diagnostic challenge. Diagnostic tests, like ambulatory pH monitoring, acid perfusion test and intraesophageal balloon distension have limited reliability in patients with... GERD, whose symptoms may exhibit poor correlation with acid exposure or mechanostimulation? Unquote.”

In yet another study by Katzka *et al*^[8], present their view-point about GERD and have also produced a Table that is reproduced hereunder for ready reference.

Table 1: Major GERD phenotypes along with clinically important modulating clinical considerations.

GERD Syndrome	Modulating Clinical Considerations
Non-erosive or endoscopy-negative reflux disease (NERD)	<ul style="list-style-type: none"> • When defined by physiological testing, very similar to low-grade esophagitis • When defined by symptom assessment, overlaps with GERD hypersensitivity and functional heartburn
GERD hypersensitivity	<ul style="list-style-type: none"> • Conceptually differentiated by positive or negative symptom association on reflux testing
Functional heartburn	<ul style="list-style-type: none"> • In practice, these entities can be clinically indistinguishable
Erosive esophagitis, low grade (LA grade A or B)	<ul style="list-style-type: none"> • LA grade A esophagitis can be found in about 6% of asymptomatic controls making it a non-specific finding
Erosive esophagitis, high grade (LA grade C or D)	<ul style="list-style-type: none"> • Grossly abnormal EGJ function with supine reflux and abnormal esophageal acid clearance • Usually associated with hiatus hernia
Barrett’s esophagus	<ul style="list-style-type: none"> • Endoscopic spectrum from intestinal metaplasia at the EGJ to short-segment to long-segment (>3 cm) • Important biological spectrum from non-dysplastic metaplasia to low-grade dysplasia to high-grade dysplasia
Reflux chest pain syndrome	<ul style="list-style-type: none"> • “Non-cardiac” chest pain along with physiological evidence of GERD or accompanied by typical reflux symptoms is much more amenable to GERD therapy than chest pain without these features
Regurgitation-dominant reflux disease	<ul style="list-style-type: none"> • Indicative of grossly incompetent EGJ barrier with large volume reflux • Need to differentiate from rumination and achalasia
Laryngopharyngeal reflux (LPR)	<ul style="list-style-type: none"> • Though reflux may contribute, it is rarely the dominant pathophysiology- generally, there are important cofactors
Chronic cough	<ul style="list-style-type: none"> • Strongly driven by neuronal hypersensitivity • More amenable to GERD therapy when accompanied by typical reflux symptoms

<https://europepmc.org/articles/PMC6960363/table/T1/> (Access 11/12/25)

Katzka *et al*^[8] state in *Phenotypes of Gastroesophageal Reflux Disease: Where Rome, Lyon, and Montreal Meet*, “From a conventional pathophysiological perspective, GERD is conceptualized as incompetence of antireflux barrier at esophagogastric junction; the more severe that incompetence, the worse the disease.” It has been shown that lower esophageal sphincter mechanism and GERD as well as Hiatus Hernia are unrelated etiopathologically. They^[8] continued, “However, it is increasingly clear that many presentations of GERD represent distinct phenotypes with unique predisposing cofactors and pathophysiology outside of this paradigm. Three major consensus initiatives have grappled with this dilemma (the Montreal Consensus, The Rome Foundation, and the Lyon Consensus), each from a different perspective. Montreal struggled to define the disease, Rome sought to characterize its functional attributes, while Lyon examined its physiological attributes. Here, we merge the 3 perspectives, developing the concept that what has come to be known as GERD is actually a family of syndromes with complex matrix of contributing

pathophysiology. A corollary to this is that the concept of one size fits all to therapeutics does not apply, and that although escalating treatment with proton pump inhibitors (PPIs) may be pertinent to healing esophagitis, its applicability beyond that is highly questionable. Similarly, failing to recognize the modulating effects of anxiety, hypervigilance, and visceral and central hypersensitivity on symptom severity has greatly oversimplified the problem. That oversimplification has led to excessive use of PPIs for everything captured under the GERD umbrella and shown a broad spectrum of syndromes less amenable to PPI therapy in any dose. It is with this in mind that we delineate this precision medicine concept of GERD. Unquote.” These conceptual juggleries only add to the conundrum and reminds the idiom, “As many pipers, as many tunes”!

(3) Calling “spade a spade”

To dare is to do (*audere est facere*). When we call ‘a spade a spade’; it truly becomes a torchbearer and guides towards solution of FGIDs. As Bergh *et al*^[3] expressed,

“The relationship between conscious experience of physical symptoms and indicators of objective physiological dysfunction is highly variable and depends on characteristics of the person, the context and their interaction. This relationship often breaks down entirely in case of ‘medically unexplained’ or ‘functional somatic’ symptoms, violating the basic assumption in medicine that physical symptoms have physiological causes. Unquote.” Rief and Broadbent^[9] summed up medically unexplained symptoms (MUS), “We summarize psychological mechanisms that have been linked to development and maintenance of MUS. Many models postulate that patients with MUS misinterpret physical sensations and show other cognitive abnormalities (eg, an over-exclusive concept of health) that play major role in symptom development. While there is strong evidence for the role of cognitive aspects, there is less evidence for their interaction with perceptual features (eg, perceptual sensitivity, lowered perceptual threshold). Unquote.” Drossman and Hasler^[10] provides an operational definition and classification system for FGIDs, discuss the process and changes that occurred from Rome III to Rome IV; history, conceptual and scientific understanding of FGIDs taxonomically with biopsychosocial model in 33 adult and 17 (upgraded 20) pediatric FGIDs. The successive two WWs led to ‘The Great Economic Depression’ plunging the global population in economic hardship and food scarcity. The Great Depression post-WW I was a devastating and prolonged economic recession that followed the crash of the United States stock market in 1929. It lasted through 1941, same year that the U.S. entered World War II. The Depression ran from 1929 to 1941 (Segal^[11]). This has been discussed in ‘Gut Issue 1’ fully. This caused obvious malnutrition and gut disorders fully declared by the work of few earlier workers. Burkitt’s^[12] work in Africa led him to propose, “These diseases are rare or unknown in communities who have deviated little from their traditional way of life, and a rise in their frequency follows adoption of Western customs. Available evidence suggests that all these diseases were rare or uncommon even in the Western world a century ago and that they are rare or unknown in undomesticated animals. Some appear or increase in frequency within a few years of exposure to new environmental factor, others not until several decades later. Unquote.” He listed a number of diseases viz, appendicitis, diverticular disease, ulcerative colitis, polyps, cancer of large bowel, oesophagitis, cirrhosis, chronic pancreatitis, Crohn’s disease, rectal hypersensitivity, anal fissures, coronary disease, gallstones, obesity, hiatus hernia, haemorrhoids, herniae, varicose veins. These are reaffirmed in 2011 by, “Quinn M J – Origins of Western diseases”.^[13] Quinn^[13] notes, “Recent gynaecological studies show that childbirth, constipation, trauma and surgery cause injuries to autonomic nerves at different anatomical sites in the female pelvis resulting in endometriosis, adenomyosis and fibroids. Re-growth of abnormal nerves cause allodynic symptoms (light touch causing pain or discomfort) some years later including vulvodinia,

dyspareunia, dysmenorrhea, irritative bladder and bowel symptoms. Unquote.” These phenotypes in females make one wonder, “How complex human biophysiology is, beyond modern psychophysiological epistemology”? The compound gynecological, urological and GIT illness in cohorts result in aberrant allodynic neuropsychological injuries are striking to explain by current anatomical, physiopathological epistemology. Yet its toll on HRQOL of cohorts cannot be denied or explained logistically.

(4) Current Concerns Effervesce – Next quarter century from 1930s on passed until gut was medically accepted as public health hazard. Some Western workers like Peter Cleave, G. D. Campbell, Hugh Trowell and D P Burkitt did emphasize role of dietary fibre. Cummings and Engineer stated^[14], “However, between 1966 and 1972, Denis Burkitt, a surgeon who had recently returned from Africa, brought together ideas from a range of disciplines together with observations from his own experience to propose a *radical view* of the role of fibre in human health. Burkitt came late to the fibre story but built on the work of three physicians. a surgeon... and a biochemist... to propose that diets low in fibre increase risk of CHD, obesity, diabetes, dental caries, various vascular disorders and large bowel conditions such as cancer, appendicitis and diverticulosis. Simply grouping these diseases together as having a common cause was groundbreaking. Proposing fibre as key stimulated much research but also controversy. Credit for dietary fibre hypothesis is given largely to Burkitt who became known as the ‘Fibre Man’. unquote” Painter and Burkitt^[15] (1971) reaffirmed, “Diverticulitis first became a clinical problem at the turn of the century, and the term ‘diverticulosis’ first appeared in 1914. As recently as 1916, disease was not important enough to merit a mention in textbooks. Though present incidence of diverticulosis is unknown, it is certainly endemic in our aged citizens. This dramatic increase in incidence occurred in only 70 years and cannot possibly be explained on *genetic basis*. This change might be due to observer error and be apparent rather than real, but we believe that their writings show that clinicians of last century were just as capable as those of today recognizing diverticulitis. We believe that there is another possibility-namely, that colon’s environment has changed and that diverticula are caused by diet of so-called civilized countries. Unquote.” Burkitt^[12] published in 1973 on dietary fibre while working in Kampala, Uganda; stated (*but ignored*), “A number of diseases of major importance are characteristic of modern Western civilization. These diseases are rare or unknown in communities who have deviated little from their traditional way of life, and a rise in their frequency follows adoption of Western customs. Unquote.”

(5) Investigations: FGIDs being functional phenotypes without any structural or biochemical abnormality; no investigation modality of any type shall be helpful. Hence it may be futile to waste space by indulging in

discussing them. However, a broad outline is mentioned for completion. Assessment of esophageal Functions are conveniently classified into four groups: (A) Tests to detect structural abnormalities of esophagus, (B) Tests to detect functional abnormalities of esophagus, (C) Tests to detect increased esophageal exposure to gastric juice, (D) Tests of duodenogastric function as they relate to esophageal disease. Esophageal pH monitoring is the gold standard for diagnosing GERD, but barium studies and endoscopy can also play a role in evaluating for hiatal hernia and Barrett metaplasia respectively.

(6) Gas and bloating conundrum: A dossier received online in *Author's inbox*^[16] stated, "This Rome Foundation Grand Rounds presentation, begins on May 1, 2024, will appeal to individuals interested in evaluating and treating one of the most prevalent clusters of gastrointestinal symptoms – *gas and bloating*. These symptoms develop for a variety of different reasons. They are reported by a large number of patients with disorders of gut-brain interaction, such as *irritable bowel syndrome* and *functional dyspepsia*. Unquote." This iterates "gas and bloating", clubbing with IBS and FD. This vignette merely adds to the enigma than clarifying the phenotype of FGIDs. Word 'gas' is defamed globally and patients get PPIs/H2blockers/PCABs prescribed. This is pharmacophysiologically harmful and unscientific in longterm. These drugs do have side-effects in longterm use. Gas in bowels is normal biophysiological byproduct of digestion of foods that is defamed ignorantly; barring few pathological conditions, eg, bowel inflammation or GI obstruction, in which case specific treatment is mandated instead of *ad libitum* PPIs etc. Is presence of gas in bowels abnormal or its absence is abnormal? Its absence is certainly worrying normally. Further if 'Gas' will not be present in gut, then where-else? It is impossible to quantify gas in any individual at any time due to vast physical and physiopathological variations. Sadly its major clinical pitfall is that undue prescriptions of H2 blockers, PPIs or PCABs, antacids, tricyclic or other empirical drugs are done for years on for no gain but for positive toxicity. Since etiopathology of FGIDs is ambiguous but side/toxic effects of drugs lead to adverse psychosomatic phenotype issues. Thus use of phenotype, "Gas and bloating" is cautioned to employ diligently. Their misinterpretation has led to change FGIDs' to DGBIs for no scientific basis. If soma becomes perturbed, psyche is only a resultant effect: *Mens sana in corpore sano* (healthy mind in healthy body, ie, psychosomatic = psycho+soma; DGBIs!)." Even a mild physical discomfort leads to disturbed mind until the physical stimulus is resolved. Some cohorts may suffer from labile mental health (allodynia); beware of such cohorts and treat them sensitively.

Its precise etiopathology is very indistinct, exemplified in Rome Foundation Grand Rounds presentation where symptoms of FGIDs are clubbed together upper and lower GIT, eg, "...disorders of gut-brain interaction, such as IBS and FD." There is overlap between various

syndrome phenotypes making FGIDs, ie, GERD/FD, IBS/DD indeterminate; even IBD (Inflammatory Bowel Disease, Maryam et al^[17]) is felt as variant of IBS/DD; further complicates pathophysiology of FGIDs. Strate and Morris^[18] iterated, "Diverticular disease, once a rarely diagnosed medical curiosity, is now one of the most common gastrointestinal disorders among inpatients and outpatients. Painter and Burkitt^[15] first documented a large increase in prevalence of diverticular disease beginning at the time of industrial revolution and noted differences in prevalence between Western and Eastern countries. These views led to the hypothesis that DD resulted from dietary fiber deficiency. According to this theory, a diet low in fiber resulted in small-caliber stools, increased intracolonic pressures, and herniation of colonic mucosa through muscular layers adjacent to vasa recta. Diverticulitis was thought to ensue when a diverticulum became obstructed with stool, resulting in fecal stasis, mucosal trauma and ischemia. Unquote."

(7) Ayurvedic Biophysiology Phenotype of Gas (Vāyu) formation: In Eastern medical system, viz 'Āyurved', gas (wind) is called 'Vāyu, alias Vāta and Pavana'; is vast word with diverse meanings in different contexts. For current purpose, we keep limited as hereunder: A life-wind or vital air (vayu) of five kinds is: (1) Udān (□ □ □ □): Upward energy that is creative (2) Prān (□ □ □ □): Inward energy that inspires (3) Samān (□ □ □ □): Inward energy that churns (4) Vyān (□ □ □ □): Pervasive energy that circulates (5) Apān (□ □ □ □): Downward energy that eliminates. The five vāyus are also known as 'Panch Prānās' or 'Panch Vāyus'. They are movements or functions of prana or life force in yoga. The vāyus govern different areas of body and physical and subtle activities. Here we concentrate this discussion on 'Apāna Vāyu'. Those interested more in 'Panch Vāyus' should peruse relevant works like Singhdeo.^[19] Word 'Prāna' refers to *vital life force or energy* that sustains us. Prāna flows by *Nādi*, subtle energy channels spread throughout body. They pass through Chakrās, as Prāna rises upwards, as 'Kundalini Shakti'. There are also five layers or sheaths in the body, known as *Pancha Koshās*. Of these five *Koshās*, second one is *Prānāyāma* consists of *five Prānās (five Vāyus)* that are responsible for various bodily functions, balances and activities. The functioning of Panch Prānās come from Āyurved and *Apān Vāyu* is responsible for elimination and is associated with organs between navel and perineum, including small and large intestines, kidneys, urinary and reproductive systems. *Apān Vāyu* is responsible for elimination of waste such as feces, gas (flatus) and urine. It also controls forceful expulsion of breath and influences sense of smell, contributes to body stability and its sphere of influence is from navel to rectum. *Apān Vāyu* causes semen ejaculation, menstrual cycle for women and expulsion of fetus during childbirth. This *Prān Vāyu* flows downwards and stimulates down and outward movement of wastes. It is linked with Root Chakra – The Earth Element (Muladhārā Chakra^[20]). Its imbalance can cause

depression, lethargy, poor elimination, constipation, diarrhea, piles (Girdhar et al^[21]; Halpern^[22]). *Panch Prànās* are looked at as important factor to balance and improve functioning. With *Āsana*, hip openers and prone postures work on *Apān Vāyu*, forward bending and sitting *Āsana* work on *Samān Vāyu*, backbending and dynamic movements work on *Prān*, Inversions and certain sitting *Āsanas* work on *Udān* and balancing and standing *Yogā* postures work on *Vyān*. *Panch Prànās* are interlinked and dependent on each other. With practice of *Yogā*, one can balance, activate and encourage harmony of *Prān Vāyus*.

(8) Yoga (Āsana) Art and Practice: It is vast subject involving physical, mental, philosophical and spiritual education. Details of this subject is out of place here and Author's endeavour limits to its physical, mental wellbeing and fitness to ward off FGIDs as well as keep fully energised in daily routine. 'Maharishi Patanjali' enunciated, Yoga has Eight Limbs but we shall be concerned here only with its Third Limb – 'Āsana' that deals with physical exercises to energise body to prepare for next Spiritual project of 'Samadhi' or 'Enlightenment'. According to Yoga Statistics^[23], "The increasing popularity of yoga in the U.S. is evident from Yoga in America study of 2016, which observed significant rise in yoga practitioners from 17.8% in 2012 to 28% in 2016. The economic impact of this growing trend is substantial, with yoga industry's value reaching \$37.46 billion globally in 2019 and projected to escalate to \$66.23 billion by 2027, as per Zippia. This data underscores yoga's expanding appeal and its substantial contribution to the wellness and fitness industry worldwide. The global yoga industry is a major force in wellness and fitness sector, with a valuation over \$88 billion. This figure is expected to grow even more to \$215 billion by 2025, around the world there are more than 300 million individuals practicing yoga. The period from 2010 to 2021 saw a remarkable surge in yoga's popularity, with an increase of 63.8%, showcasing that more and more people are getting interested in it. Unquote." Huang^[24] narrates, "In the U.S., about 1 out of 6 adults say they practice yoga, according to new survey data published Wednesday by Centers for Disease Control and Prevention. About 80% are practicing to improve their health, and some 30% are using it to treat and manage pain. "Yoga is a complementary health approach used to promote health and well-being," says Nazik Elgaddal, an IT specialist at the CDC's National Center for Health Statistics who co-authored a data brief on the topic." The stretching and strengthening exercise has been shown to reduce stress and help with some types of neck and back pain. Unquote.* Author (Octogenarian) practices himself and vouches it's psychophysical and biophysiological phenotype advantages from personal practice. Yoga poses apparently look simple flexible and stretchable prosaic exercises but their impact and experiences at psychosomatic and mental well being

are profound resulting in overall increased work output and job satisfaction: "proof of pudding is in eating".

(9) CONCLUSION

Humans are allegedly beseeched by FGIDs but are there FGIDs or dysfunctional human beings (DHBs) contracting FGIDs? Modern human society is suffering from FGIDs that are self-inflicted by humans themselves. Nature and controversy about FGIDs as expressed by various global schools and calling spade a spade with varied views by various authors including medically unexplained symptoms (MUS) are highlighted. Katzka et al in a study present their opinion on GERD in a chart; facsimile of it is reproduced indicating its heterogeneity. Different schools are playing their tunes ignoring the real etiopathology that lie in modern Western lifestyle and dinner plate. Despite efforts by some Western workers like Burkitt, undue waywardness resisting easy way in lifestyle is being adopted. We must swallow our hubris to look within, ie, in our dinner plate instead of modern high-tech investigations at undue economic burden. Age-old biophysiology of gas phenotype explaining the Ayurvedic concept and Biophysiology Phenotype of definition of gas (Vāyu) with its five classifications and their functions have been initiated including positive role of *Apāna Vāyu* in FGIDs and their functions has been introduced for the first time. Fictitious clinical phenotype of Gas and bloating syndromes has also been discussed as non-issue. The therapeutic positive role of 'Yoga Āsana's' has been introduced in this vignette to ameliorate FGIDs besides their health related quality of life (HRQOL).

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