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BOWEL NO 7 - FUNCTIONAL GASTRO-INTESTINAL DISORDERS V DYSFUNCTIONAL HUMAN BEINGS, INCITE HYPERTENSIVE COLONIC SYNDROME - MICROMANAGING MILIEU INTERIOR -CAUSA LATET, VIS EST NOTISSIMA (CAUSE IS HIDDEN; RESULT IS KNOWN)

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ABSTRACT

For past over half a century, medical faculty have been viciously grilling herself on pathophysiological phenotype of 'Functional Gastro-Intestinal Disorders (FGIDs). In this monograph, etiophysiopathology of *hypertensive colonic syndrome* (HCS), anatomical aspects of large intestine, alongwith irritable bowel syndrome (IBS) and diverticular disease (DD), gut dysmotility causing HCS shall be addressed. Modern Western lifestyle issues are correlated to etiopathology of FGIDs. 'Functional fault' lies in lifestyle abuse instead of GIT. GERD and IBS symptoms frequently overlap; signifying that entire gut from stomodeum to proctodeum acts in unison as one unit though medical faculty has not appreciated its phenotype essence. Truely there are no FGIDs but only Dysfunctional Human Beings (DHBs) contracting FGIDs. "They condemn what they do not understand (*Damnant quod non intellegunt*)"

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; Hypertensive Colonic Syndrome – HCS.

(1) INTRODUCTION

Hypertensive Colonic Syndrome (HCS) is an innovative physiopathological concept and this monograph discusses the subject in ensuing sections: large intestine (gross anatomy and applied microanatomy); microstructure of submucosa of large intestine; blood supply of colon and pathogenesis of diverticular disease (DD), putative etiopathology of irritable bowel syndrome (IBS); which is also harbinger and mechanism of HCS. Importance of 'fart' (flatulence or flatus) in health related quality of life (HRQOL) with a mnemonic, "fart a while – zest all the while" for better HRQOL, besides, a brief comment about FODMAP is described.

(2) Preliminary Prologue: World Health Organization (WHO) defines health as a "State of complete physical, mental and social well being and not merely the absence of disease" (NIOS[2], Schramme[3]). Despite efforts of WHO; arguably, humans are afflicted with Functional Gastro-Intestinal Disorders (FGIDs). Factually there are no FGIDs, but only 'Dysfunctional Human Beings – DHBs' contracting FGIDs. Sperber *et al*[4] published global study data of FGIDs that result in significant global health care costs and impaired health-related quality of life (HRQOL) (Teoli and Bhardwaj[5]). Published studies have involved highly variable diagnostic criteria, study populations, questionnaires and

data collection methods. Thus, given the large methodological heterogeneity, it is inappropriate to pool individual prevalence rates and we are left with an unanswered question as to whether differences in prevalence rates seen among individual countries in prior surveys reflect genuine differences between populations or are due to methodological differences between studies. Fletcher[6] stated, "Judgments about clinical heterogeneity are qualitative, do not involve any calculations and can be made by putting forward a convincing argument about similarities (or differences) between the trials. Unquote" We eat and drink unconcerned presuming Gastro-Intestinal Tract (GIT) is trash bag and anything going wrong is fault of GIT and designate them "FGIDs" heuristically.

(3) Large Intestine – gross anatomy: Large intestine includes appendix, cecum, entire colon (ascending, transverse, descending, and sigmoid), rectum and proximal anal canal upto internal anal sphincter. Colon is shorter lengthwise (about 1 to 1.5 meter long; 6 cm to 9 cm wide) compared to small intestine (6.5 to 7.5 meters long; 2 to 3 cm wide) but has much wider lumen; hence called large for its width. Colon is notable by presence of appendices epiploicae, haustra and teniae coli. Appendices epiploicae are small protrusions of peritoneum present in entire colon except appendix and rectum. They contain adipose tissue, blood vessels, lymphatics; prominent on transverse and sigmoid colon. Haustra are small sacculations giving large intestine false segmental appearance. They are created by semilunar folds on internal surface of colon. Teniae are three long bands of smooth muscle on outer wall of colon except at appendix and rectum, where teniae coli converge to form continuous outer long smooth muscle layer. Two of taeniae in sigmoid colon are located on antimesenteric border of colon and third is located on mesenteric border. The muscle layer becomes progressively thicker in more distal colon, particularly in sigmoid colon, and taeniae eventually fuse in proximal rectum, a landmark useful for identifying distal extent of resection when performing a sigmoid colectomy. In distal rectum, inner circular smooth muscle condenses to form internal anal sphincter. Cecum is widest part of colon about 7.5 to 8 cm wide and has thinnest muscle wall prone to perforation but least to obstruction. Sigmoid colon is fairly mobile with a narrow base mesentery prone to volvulus and narrower part of colon prone to obstruction and diverticular disease (DD). Its mobility makes it lie in right side abdomen disguising appendicitis. Good knowledge of anatomy and pathophysiology of digestive tract will help diagnose and manage GI disorders, optimize surgical procedures and enrich patient care (Omole *et al*[7]).

(4) Large Intestine – applied microanatomy: Colon layers from lumen outward consist of mucosa, submucosa, nuscularis externa, and adventitia/serosa. Mucosa forms epithelium, intestinal glands, lamina propria and muscularis mucosa. There are *no plicae circulares or villi* in colon but temporary folds only. Submucosa formed of loose network of connective tissue and muscularis externa containing inner circular and outer longitudinal smooth muscle forming three teniae coli. Outermost layer, viz, serosa covers transverse and sigmoid colons hinged to posterior parietes by a mesentery containing loose connective tissue, adipocytes, blood vessels and nerves. Ascending and descending colons are partly retroperitoneal and their posterior layer, viz, adventitia is formed by collagen tissue.

(5) The submucosa of large intestine is a loose fibrous connective tissue layer situated between muscularis mucosa and muscularis propria. It supports, neuro-vascular supply, fibroblasts, mast cells, blood and lymphatic vessels, lymphoid aggregates that may extend from lamina propria containing network of collagen and elastin fibers providing support and elasticity to the layer responsible for absorption of water, salts and excretion of waste products. Submucosa is thicker in large intestine, allows greater storage and fermentation of fecal material and contains nerve plexus (Meissner's plexus) of nonmyelinated postganglionic sympathetic fibers and parasympathetic ganglion cells.

(6) Blood supply of colon portends pathogenesis of DD: Colon is supplied blood by superior (SMA) and inferior mesenteric arteries (IMA) that is highly variable. Arterioles from SMA/IMA form a marginal artery in majority cohorts on inner border of colon from which smaller arteries run toward colon forming arterial arcade from which smaller arterioles (vasa-recta) penetrate circular muscle beside teniae coli to enter submucosa and mucosa. Vasa-recta create tiny weak spots at point of entrance through circular muscle, precursor for *pulsion diverticulae* formation due to high intraluminal colonic pressure, progenitor of HCS. Diverticula generally occur in parallel rows along the mesenteric side of the antimesenteric taeniae; with progression, an additional row of diverticula may be found between the antimesenteric taeniae. Microscopic studies of areas of colon with early small diverticula demonstrate areas of thinning due to presumed focal microscopic muscle atrophy. With progression, a clear-cut defect in muscle occurs, usually at the site of penetration of a vessel through gaps in circular muscle. Interestingly, diverticula remain isolated to the circular muscle, and rarely, if ever, penetrate through taeniae. HCS is created by abnormal colon motility perhaps by low natural dietary fiber resulting in small fecal volume, insufficient for propulsion by motility of colon; structural abnormalities of colonic wall created by senescence, though physiopathology phenotype is highly complex, ill-understood and multipronged including Western lifestyle. In pulsion diverticulosis, mucosa and muscularis mucosa only herniate along ports of entry of vasa-recta between taeniae; more so in sigmoid colon with high intraluminal tension as sigmoid is narrowest part of colon and its contents are thicker.

(7) Putative Etiopathology of HCS harbinger of IBS/DD: It is observed that human digestive system is suitable for *herbivorous* meal rich in natural fiber that gives large volume to feces and also generates some gaseous products essential for proper motility, health of gut microbiota and all the desired physical and biophysiological phenotypes. These biochemical products are essential for normal physiological phenotypes that prevent generation of FGIDs. Apropos to digestive process, some amount of gases are also generated as byproduct, which are essential for motility and digestion. Sadly, word – 'Gas' is denigrated as odious etiopathological factor causing FGIDs or IBS/DD. Instead, this phenotype must be regarded as prophylactic factor against FGIDs and IBS/DD, including inflammatory bowel disease (IBD) and is discussed more in 'Fart' section. Strate and Morris[8] iterate that DD was a rarely diagnosed medical curiosity, is now one of the most common gastrointestinal disorders among inpatients and outpatients. Painter and Burkitt[9] (1971) documented a large increase in prevalence of DD beginning at the time of industrial revolution and noted differences in prevalence between Western and Eastern countries. These insights 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 colon mucosa through portals by vasa recta in muscle layer. Diverticulitis was thought to arise when diverticulum became inflamed and obstructed with stool, resulting in fecal stasis, mucosal trauma, and ischemia. Sadly '*dietary fiber deficiency*' hypothesis is ignored. They[9] stated, "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... This dramatic increase in incidence occurred in only 70 years and cannot possibly be explained on *genetic basis*." Unquote. Genetic factor may be rare involvement in etiopathology of FGIDs, IBS/DD, constipation or obstipation or some organic disease like IBD. High fiber diet is golden key (*Clavis aurea)*; is ignored yet it is 'essential condition without which FGIDs could not be' (*conditio sine qua non*). Drossman[10] recalls, "The paradigm shift began with the Manning Criteria publication in 1977, a precursor of the Rome Criteria... Our goal was to fight back against the narrative that nothing is wrong if it's not seen in a test... Symptom-based criteria allowed us to understand the patients' symptoms, regardless of the presence of structural or physiological findings. Unquote." Apart from food; even fasting can help repel FGIDs (Semnani-Azad *et al*[11]). Fasting is practiced since antiquity in India every month on a schedule, while Islamic practice in Ramadan yearly from dawn to dusk with night-time eating ad libitum is practiced since long. Collier[12] advises about fasting, "To improve health, goal should be to lose weight by reducing the total amount of calories consumed... rather than focusing on when those calories are consumed." He[12] continues, "If you [don't] eat two days a week, and limit what you eat the other five days, you will lose weight... I'm not sure it works any better than cutting down slightly seven days a week. Unquote." He[12] explains, "The one that we've studied a lot, and designed experiments to test, is the hypothesis that during the fasting period, cells are under a mild stress... and they respond to the stress adaptively by enhancing their ability to cope with stress and, maybe, to resist disease. Unquote." The dictum reminds, "There are no facts, only interpretations (*Tatsachen gibt es nicht, nur Interpretationen* – Nietzsche[13]).

(8) Mechanism of Hypertensive Colon Syndrome: Study of colon motility is difficult subject. It forms important function and regulatory mechanism of physiopathology phenotype of colon disorders. Colon exhibits three types of motility actions, ie, peristalsis, segmentation and mass movements (giant migrating contractions – GMCs) detailed in 'Issue 5, subsection 16-17'. Camilleri[14] briefly addressed various phenotypes in FGIDs including gut motility, "...changes in barrier function, alterations of the intraluminal milieu within the gastrointestinal tract including effects of endogenous slowly produced substances like bile acids and short chain fatty acids, and exogenous substances including dietary and product of microbes that are commensal or introduced as part of an infectious process. In addition, there are also disturbances of gastrointestinal motor function. Unquote." Hobson and Roberts[15] stated, "Painter and colleagues performed sigmoid manometry and cineradiography in patients with and without diverticular disease. They found that fecal material normally passes through the length of the colon with minimal change in pressure. However, when two haustral contractions occur in a given segment of the colon at the same time, it can cause temporary isolation of that segment of bowel, a process called segmentation. Unquote" Thus, the colon seemed to function not as a tube, but as individual compartments generating high pressures. Contraction of the bowel wall subsequently causes locally increased intracolonic pressure, resulting in functional obstruction. In this way, segmentation creates a pulsion force causing visible distension of the local diverticula. The high pressures resulting from segmentation in turn lead to focal muscular atrophy and subsequent mucosal herniation. Pulsion diverticula occur most frequently in sigmoid colon because lumen of colon is narrowest resulting in generation of highest pressures. Arfdwidsson[16] using intraluminal pressure recording showed that segments of the sigmoid that were beset with diverticula were capable of producing higher pressures than the normal bowel when stimulated by eating and emotion and by prostigmine. Painter[17] combined pressure recording using open-ended tubes (balloons do not record pressure in bowel) with simultaneous cineradiography. This revealed that localised intracolonic pressures were generated by segmentation of the colon. These pressures are produced by contraction of colonic muscle and this finding is supported by the observation that, if this muscle is

paralysed by probanthine, intraluminal pressure falls to basal level and generation of localised pressures ceases. When segmented, colon acts as a series of "little bladders" whose outflow is obstructed at both ends and in each segment or "bladder" high pressures may develop. This mechanism is responsible for the pulsion force that causes diverticula to be extruded. Furthermore, morphine, which is commonly used as analgesic in acute diverticulitis, raises pressures in sigmoid.

The segmentation movements are involved in mixing, propulsion and expulsion of feces and it solely depends upon its biophysical texture. Diet with sufficient high fibre is biophysiologically conducive for colon motility to propel feces and avoid 'slow transit constipation'. Fiber content of diet helps in two ways. One, it is less sticky to mucosa due to abundant vegetable fiber devoid of proteinaceous bioglues; secondly it adds to fecal bulk helps to fill the lumen of colon and facilitates propulsive segmentation movements and GMCs, thereby nullify generation of intramural colonic hypertension. This biophysiological intervention applies to solid fecal as well as 'gaseous mass'. In fact, passage of flatus is far more important than passage of feces. One may take a little liberty in evacuation of feces for a moment or even days but any delay in release of flatus of even seconds will result in 'retrograde giant contractions (RGCs)', which are harbingers of visceral hypersensitivity phenotypes and resultant FGIDs. Simple dietary biophysiological intervention for phenotype modification avoids the development of intramural colonic hypertension, thereby DD/IBS and HCS. Prather and Ortiz-Camacho[18] observed, "Constipation and sequelae of constipation, such as hemorrhoids, are rarely described in Third World countries. The consumption of a fiber-depleted Western diet has been implicated in constipation and other disorders. Although current American Dietetic Association daily recommendation for fiber is 25 g, few adults consume this level of fiber. Increased dietary fiber intake accelerates colonic transit and produces more frequent, bulky stools. Unquote." The slow transition movement of colon results in functional constipation. The precise quantity of fiber consumption may vary between different individual cohorts due to innate anatomical and physiopathological variations besides the quality and quantity of daily dietary consumption. Thus the daily supplementation of fiber in diet may vary between various cohorts and it will also vary in daily routine. It is decided by the cohorts intelligibly. McKeown *et al*[19] observed, "Modern day diets are very different from those of our ancestors, which contained substantially more fibre because they consumed a variety of plant based foods. Ancestral humans might have consumed as much as 100 g of fibre daily. Today, adults in North America consume an average of 17 g of dietary fibre daily; intakes are slightly higher in European countries (18 g to 24 g a day). The definition of dietary fibre has evolved from "remnants of plant cells that are resistant to digestion by human enzymes" in 1970s to the more complex global definition outlined in 2009 by the Codex Alimentarius Commission. Although there has been considerable debate over past few decades about terminology and analytical methodology used to define dietary fibre, most definitions now include carbohydrates with three or more monomeric units. This includes some well known prebiotic fibres, such as fructans and inulins. Despite scientific advances in the field, consumer confusion persists, and current intakes of dietary fibre fall short in many populations worldwide. Unquote." Quinn[20] quotes, "DP Burkitt found that Africans eating low calorie, plant-based diets had short intestinal transit times (14 h vs. 40 h), increased stool weights (464 g vs. 110 g per day), and reduced rates of 'constipation' compared to Europeans. Unquote." He[20] continued, "Burkitt also viewed squatting as an advantageous method of defaecation (and childbirth) compared to Western methods, though he did not identify the adverse neuropathic consequences. In recent years, KW Heaton found that physical efforts complicated 20–30% of Western bowel movements. One percent of adults opened their bowels less than once each week, and, 0.3% less than once each month. Recent studies confirm that persistent straining during defaecation causes neuropathic pelvic injuries in every organ in the nulliparous female pelvis. Unquote."

Dhingra *et al*[21] defines dietary fiber, "Dietary fibre is that part of plant material in the diet which is resistant to enzymatic digestion which includes cellulose, noncellulosic polysaccharides such as hemicellulose, pectic substances, gums, mucilages and non-carbohydrate component lignin. The diets rich in fibre such as cereals, nuts, fruits and vegetables have positive effect on health since their consumption has been related to decreased incidence of several diseases. Dietary fibre can be used in various functional foods like bakery, drinks, beverages and meat products. Influence of different processing treatments (like extrusion-cooking, canning, grinding, boiling, frying) alter physico-chemical properties of dietary fibre and improves their functionality. Unquote."

(9) 'Fart awhile, Zest all the while' and Health Related Quality of Life: 'Quality of Life' is vast subject but here, we shall concentrate on "Health Related Quality of Life (HRQoL), (Teoli and Bhardwaj[4])." Important factor that affects FGIDs exclusively relates to release of flatulence more than passing feces and is discussed hereunder, but there can be no debate who deny the principles (*contra principia negantem non est disputandum*). Word fart (the phrase 'not worth a fart', ie, worthless) is intentionally used to draw necessary focus. One fart is millions of dollars worth, which are spent to get rid of FGIDs. Fart is bad enough in civility but not attending to it is worse to the cohort biophysiopathologically. The trio, "Fart, fart and fart" is used than more civilised words – flatulence or flatus. The metaphor, "Either 'fart' from below; *else* fart from

above"; means cry with visceral hypersensitivity phenotypes and all its ensuing sequela, ie, abdominal discomfort, vague or even severe pains, so-called ill-famed 'gas', bloating, IBS/DD, GERD/FD, even IBD; anorexia, constipation, diarrhoea. Postoperatively, first thing surgeon asks patient, "Have you broken wind instead of passed poop"? Passage of gas (fart) and absence of abdominal distension with bowel sounds signifies return of normal gut motility as a sign of return of normal GIT function after postoperative ileus; signal to resume oral intake.

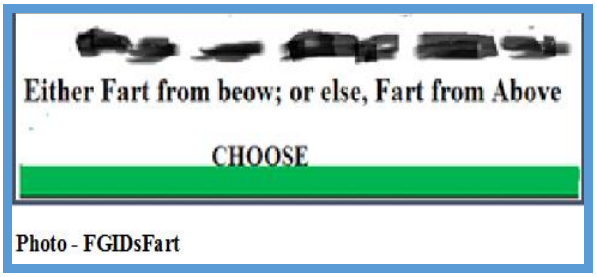

Photo - FGIDsFart

This rectangular box showing broken ragged mound-like art at top is sign of melancholy that turns into zest as soon as 'fart' occurs; is shown in bottom *uniform green block*. This Idiomatic phrase, "*Fart awhile, zest all the while*"; signifies experiencing life fully, even if it means uncivil in *fart* than holding back and stay joyful in "zest". "Fart awhile" refers to experiencing normal and sometimes unavoidable involuntary phenotype awful to audience but biophysiologically healthy to cohort warding off phenotypes like visceral hypersensitivity etc. "Zest all the while" is metaphorical expression and "don't let minor events or inconveniences stop you living joyful life free of FGIDs, with fart". This *involuntary* GIT intervention can help, rid of dreaded visceral hypersensitivity, altered central nervous system processing, dysbiosis and gut motility disorders that ultimately lead to miserable FGIDs. Our GIT functioning is like motor car, if, timely repair of one loose nut is ignored; it leads to dysfunction of other parts in 'cascade effect' of entire system. Similar events occur with GIT by interfering with flatulence. Initial nuts of Human GIT are disordered motility and dysbiosis, though these are poorly understood. Slowly their dysfunction leads to others until the whole system gives up making it unserviceable and sick of; we call FGIDs. It must be noted that biophysiologically, farting is involuntary, unpredictable phenotype so much so that urge may not feel at one moment and urgently in next blink of moment, comes up. Its release must be assisted come what may, lest suffer retrograde giant contractions (RGC) in colon, ensuant visceral hypersensitivity and motility disorders. When this physiopathological phenotype becomes chronic; is called FGIDs, linking with Gut-Brain Axis or bidirectional 'microbiota-gut-brain axis'. Loh *et al*[22] assert, "Accumulating evidence has unveiled the bidirectional communication between the gut microbiome and central nervous system (CNS), referred to as "microbiota–gut–brain axis". Although gut and brain are anatomically separated, several pathways by which gut microbiota communicates with CNS have been proposed. These include modulation of immune system, vagus nerve, enteric nervous system (ENS), neuroendocrine system and circulatory system via the production of neuroactive substances, metabolites and hormones. Unquote." No matter how best we investigate; will be fruitless except for a good clinical history taking. Author always asks cohorts in history taking session, "Does farting helps you feel better and relieved of discomfort"? Invariably the response is in affirmative in majority and this concludes the history taking session. Interestingly, whether the symptoms are related to GERD/FD or IBS/DD; overtly, etiopathology of phenotypes remain cryptic: if cause is taken away, effect ends (*ablata causa tollitur efectus*); mostly, lie in one 'flatulence (fart)'.

Buchanan and Tuma[23] quote economic burden, "It is estimated that the economic impact of postoperative ileus (POI) is approximately 750 million dollars annually in the United States alone. Unquote." They[23] continue explaining its *Pathophysiology*: The exact mechanism and causes of POI are incompletely understood. However, the pathophysiology of POI can be attributed to the following three categories of mechanisms: neurogenic, inflammatory, and pharmacologic. The autonomic nervous system plays a major role in gastrointestinal motility. The parasympathetic system stimulates motility while sympathetic system inhibits motility. Increased sympathetic stimulation plays a role in inhibition of gastrointestinal motility after surgery. Hormones and neurotransmitters, such as nitric oxide, calcitonin-gene-related peptide and corticotropin-releasing factor may induce development of POI. POI is more likely to develop after prolonged major surgical procedures and general anesthesia with excessive gastrointestinal manipulation or interruption. Postoperative pain medication, particularly opioids, promotes development or worsening of POI due to their known inhibitory effect on gut motility. Unquote."

In a relevant clinical phenotype about constipation (a syndrome of FGIDs), studied in a systematic review on idiopathic acquired megacolon by Cuda *et al*[24], concluded: (A) Acquired Megacolon (AMC) is poorly understood and diagnostic criteria remain obscure. Many symptoms including constipation, distension, abdominal pain and a poor sense of wellbeing are attributed to AMC. Pathophysiology, natural history and effective symptom management, although speculated, are unknown. (B) The common features of an adult presentation were constipation, distension, gas distress and abdominal pain. In comparison, children presented with faecal incontinence and impaction. (C) There may well be a considerable overlap between symptoms associated with AMC and Constipation Predominant Irritable Bowel Syndrome given 20% of constipated patients has an AMC. Brummer et al. (1962) supports

this, estimating 30% of patients with constipation have an AMC. (D) AMC is a disease of exclusion. Ruling out an organic cause for this condition is pertinent. The absence of hypogangliosis has more consistent results when compared with anorectal reflex testing, although it does carry more risk. (E) Based on data collated in this review, we propose the following criteria for the diagnosis of AMC: (1) exclusion of organic disease by rectal biopsy or intact anorectal inhibitory reflex; (2) sigmoid diameter of ~10 cm on abdominal X-ray or barium enema; (3) symptoms including constipation, distension, abdominal pain and gas distress. (F) Whether AMC is single entity or group of heterogeneous conditions is unknown, neither is its relationship to other constipation predominant conditions. It may well be that patients with AMC are misdiagnosed as having Constipation Predominant IBS. The natural history of this condition and optimal forms of management are yet to be elucidated. (G) Further research is required on pathophysiology of the condition, protocols for conservative treatment and place of surgery for intractable disease. Unquote." Author feels that it is surrender as there is no obvious etiopathology and the pertinent issues of concern are ignored. They have used one terminology-*Gas distress* that is an illdefined hypothetical phenotype and to avoid confusion, unqualified word – '*Gas'* itself must be avoided. It has been explained earlier that gas is both normal and abnormal phenotype depending on clinical perspective. Author feels that absence of 'gas' in GIT is definitely abnormal than its presence, the debate may be about its size or pattern. There is no system in body except GIT where gas is found normally. Presence of gas everywhere else in human body is definitely pathological. Therefore use of unqualified phenotype Gas in GIT is advised cautiously. Constantin *et al*[25] describe, "Idiopathic megacolon (IM) is a condition characterized by an enlarged colon and aperistaltic syndrome in absence of a detectable cause. The main symptom is considered chronic constipation, refractory to drug treatment and without surgical indication. IM affects both sexes and symptoms develop early in childhood or adulthood. The condition has a relatively unknown etiopathogenesis, but according to nosological framework, term IM primarily excludes congenital nerve plexus disorders (Hirschsprung's disease) or colonic changes secondary to a systemic disorder (colonic pseudo obstruction). The terminology used in literature for this pathology can lead to confusion by name of either "idiopathic (mega)colon" or "acquired megacolon" depending on authors; referring to same pathological entity. Unquote."

(10) Classification of Hypertonic Colonic Syndrome: The Original Hinchey Classification (1978) is a system used to grade the severity of acute complicated diverticulitis based on CT or intraoperative surgical findings, helping to guide treatment decisions. As advanced imaging modalities and conservative treatments have improved, this classification has been considered increasingly unsuitable for determining the treatment strategy for complicated diverticulitis and more modifications have been described with no better clinical advantage. It has been modified over time, often known as the Modified Hinchey Classification described by Tochigi *et al*[26] in their monograph to better reflect CT findings and include earlier stages of the disease. This system is based on intraoperative findings which limits its preoperative clinical value. To overcome this deficiency, I have tried to develop a preoperative classification system. Author classifies 'Hypertensive Colonic Syndrome' in following types based on clinical observations preoperatively. Group A = Abdomen is soft on palpation with some tenderness alongwith a feel of vague lumpiness in left iliac fossa. Group B = Phenotypes of Group A alongwith tenderness all along the area of colon except central abdomen. Group C = Entire abdomen is tender, there may be a lump in left lower abdomen and signs of ascending cholangitis, ascites may or not be present, pyrexia. Group D = Frank signs of peritonitis besides signs in Group C. Except these phenotypes, there may be isolated phenotypes of pain in left hypochondrium (Left Hypochondrium Syndrome), or pain in right hypochondrium (Right Hypochondrium Syndrome); related to Splenic and/or Hepatic flexures.

(11) Low FODMAP diet: Low FODMAP (fermentable oligo-, di-, and monosaccharides and polyols) diet, involves cutting out certain foods that are poorly absorbed are reckoned to result in gas symptoms. This type of diet can be advisable, *temporarily*, for IBS-diarrhea prone cohorts until their GIT has resumed normalcy. Author resents its blind promotion because such a diet must not be advised blindly on long term basis, lest it itself become a cause of FGIDs. De facto, a low residue or low fiber diet (low FODMAP) is the cause of FGIDs. Hence blind promotion of low FODMAP diet in long run is unbiophysiological and must be prudently advised. Few dietary products like chewing gums, and carbonated beverages such as soda or beer and such products and aerophagia can cause temporary bloating and better curtailed. FODMAP diet must be directed towards specific GIT phenotypes. Catassi *et al*[27] remarked, "The criteria for inclusion in FODMAPs list are not fully defined. Although low-FODMAP diet can have a positive impact on symptoms of IBS, particularly bloating and diarrhea, quality of evidence is lower than optimal, due to frequent methodological flaws, particularly lack of proper control group and/or lack of blinding. In particular, it remains to be proven whether this regimen is superior to conventional IBS diets. The drastic reduction of FODMAP intake has physiological consequences, eg, on intestinal microbiome and colonocyte metabolism, which are still poorly understood. A low-FODMAP diet imposes an important restriction of dietary choices due to elimination of some staple foods, such as wheat derivatives, lactose-containing dairy products, many vegetables and pulses, and several types of fruits. For this reason, patients may be at risk of reduced intake of

fiber, calcium, iron, zinc, folate, B and D vitamins, and natural antioxidants. Unquote."

(12) Conclusion: With reference to 'WHO' on HRQOL, FGIDs are *non sequitur* (it does not follow) and factually there are no FGIDs, but only DHBs contracting FGIDs. A new syndrome, viz, HCS has been suggested and anatomical structure of colon including its submucosa and reference to blood supply is described as prelude to etiophysiopathological understanding of HCS and IBS/DD. Disorders of motility and microbiota are harbingers of HCS. 'Gas and bloating' phenotype is a physiopathological misnomer due to biophysiological misconception. "*Fart awhile; Zest all the while*" forms important phenotype to ensure HRQOL. Word fart is intently used to draw necessary focus because disorders of *fart* depend upon healthy physiopathology of gut which in turn depends upon healthy motility and colon microbiota. Good natural dietary fiber ensures preceding colon phenotypes and avoids issues of 'gas, bloating'. This phrase, "*Fart awhile, zest all the while*"; signifies experiencing life fully, even if it means uncivil but stay joyful in "zest", and is discussed to lay stress for HRQOL. Issues of lifestyle, clean potable water, dietary factors, physiotherapy and other traits like bowel, urinary, sexual hygiene, etc, besides anatomical aspects of colon, etiopathology of mechanism of HCS that is both consequence and cause of constipation, DD/IBS and their management protocols are devolved. A classification plan of HCS is suggested based on clinical presentation. The role of Fodmap diet in relation to FGIDs is defined. There is misconception, whereby effect is mistaken for cause, eg, GERD/FD, IBS/DD, HCS are effects of the cause that lie in Western lifestyle. One must concentrate on colon motility and microbiota/microbiome phenotype biophysiopathology, failure of which leads to FGIDs including even IBD. Correcting the DHBs shall correct the FGIDs and HCS.

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