Dear colleagues,

I offer you a scientific analysis of diagnostic methods that have recently been used for functional constipation (FC).

Introduction. Unfortunately, recently the idea of scientific analysis has lost its original meaning. Scientific analysis should be based on honest and compelling scientific evidence.

**First**, a scientific fact should not contradict common sense. For example, in the article by Koppen et al, the authors came to the absurd conclusion that the upper limit of the normal width of the rectum in children aged 1-6 years is 6.5 cm, that is, the same as in adults [1]. This conclusion made because of significant violations in scientific research [2].

**Secondly**, erroneous selection criteria for patients to determine normal limits lead not only to erroneous conclusions but also to a false idea of the normal anatomy and physiology of anorectum, pathological physiology of the FC, as well as unreasonable surgical interventions. For example, Koppen et al, based on the erroneous determination of the maximum border of the norm of the rectum in children, concluded that segmental colonic dilation is possible [3]. In this case, the formal logic is followed. If a 2-year-old child has a width of the rectum less than 6.5 cm, which, according to the authors of the article, is the norm, then the expansion of the sigmoid and descending intestines is a segmental expansion of an unknown nature. The authors did not measure the length of the anal canal and the width of the rectum, and did not determine the anorectal reflexes. They compared width at different places in the sigmoid and descending gut with high-amplitude propagating contractions (HAPCs). It turned out that the colonic segments with HAPCs had a significantly smaller median diameter than colonic segments without HAPCs. This study made no sense because the expanded
segments of the colon with fibrotic changes in the wall and the loss of Cajal cells cannot contract as well as narrow segments. The authors' conclusions about the possibility of segmental colonic dilation are erroneous. In addition, the conclusion that “the combination of manometry testing and contrast enema studies may help guide surgical management of children with FC by identifying dysmotile colonic segment” does not follow from this work. Meanwhile, based on false conclusions, the authors resect the expanded sigmoid colon, leaving the megarectum untouched [4].

Thirdly, any new research should be based on the results of previously published scientific research and the experience of previous generations. For example, it is known that FC occurs in childhood as a result of a deliberate delay in bowel movements, which leads to the accumulation of a large number of feces in the rectum. The next attempt to defecate causes pain and re-delay defecation. There is a vicious circle, as a result of which there is an expansion of the rectum and a gradual expansion of the left parts of the colon. As a result of the mismatch between the width of the feces formed in the megarectum with the throughput of the anal canal, the function of the puborectalis muscle is damaged. This so-called descending perineal syndrome is accompanied by fecal incontinence [5]. This is a brief description of the most important elements of the pathophysiology of the FC. But they are not even mentioned in the works of this group of authors, as if over the past 200 years there have been no studies in this field of knowledge. These authors produce a sigmoid resection though it is not physiological since it does not eliminate the mismatch between the width of the feces and the throughput of the anal canal. And as the experience of previous generations showed, a dolichosigma resection does not relieve patients of problems [6,7].

Fourth, the consensus is a way of political compromise. No congress decisions, including the Rome criteria, are scientific achievements. Discussion by the multidisciplinary team combining motility gastroenterology, psychology, and

**Fifth,** the value of scientific research does not depend on the fame of the author. This obvious and necessary principle for the development of science is ignored in pediatric journals, as evidenced by the publication of works by Koppen et al.

**Sixth,** FC begins in childhood, and if it is not cured, it continues in an adult. Thus, the pathophysiology of FC in children and adults is the same. However, the severity of the pathological process is more pronounced in adults. This means that pediatric surgeons should consider studies of the pathophysiology of FC in adults, but indications for surgical treatment in children should be sharply limited.

**Measurement of colon transit**

Colonic transit investigation in adults and children has led to the concept of three types of constipation: normal transit constipation, slow transit constipation, and evacuation disorder. With scintigraphy and marker studies, STC can be further quantified as general colonic inertia, defined as the retention of marker throughout the entire colon; or left-sided delay, characterized by retention primarily in descending and sigmoid colon [8]. It is suggested that transit studies help to guide treatment. "For example, in patients that have failed all non-surgical therapies, partial or total colectomy may be recommended" [8]. Some authors based on a scintigraphic evaluation of colonic transit in children believe that STC and functional fecal retention are two forms of severe intractable constipation in childhood. It is argued that STC is characterized by the delayed passage of fecal matter through the proximal (right) colon, whereas functional fecal retention describes delayed transit in the rectosigmoid (left) region only [9]. As an analysis of this study showed, the authors mistakenly rated a widened and elongated
sigmoid colon located to the right of the midline as part of the right half of the colon [10].

The notion that slow transit constipation and evacuation disorder are different diseases, arose based on X-ray studies of Gladman et al [11]. An analysis of this work showed that using non-physiological techniques, the authors came to the absurd conclusion that 6.5-6.3 cm is the upper limit of the normal width of the rectum [12]. It is enough to draw a circle of such a diameter in order to understand that feces of this size cannot form in the rectum of a healthy person. Moreover, feces of such a diameter do not pass through the anal canal without damaging it. Due to this error, in patients with FC in which the width of the rectum was less than 6.3-6.5 cm, obstruction, as the cause of FC, was excluded. Therefore, there was an idea of STC, as the reasons for the FC. Surprisingly, articles on slow transit constipation ignore studies on the pathophysiology of FC.

**Pathophysiology of FC.**

Based on the fact, that constipation never occurs in patients with a colostomy, reasons for the development of FC are presumed in a disturbance in the anorectum [13]. The most common pathogenesis of FC is a vicious cycle that begins at the age of 2-4 years with painful defecation and leads to stool-withholding behavior as a result. During the withholding, rectal mucosa absorbs water from the fecal mass, which becomes harder and larger as time passes and ultimately defecation becomes difficult. Therefore, when the desire to pass stools comes, children adopt the retentive posture, hide from parents till the urge pass of. Passage of this fecal mass is painful and sometimes results in anal fissures, which further aggravate pain and precipitate stool withholding. Accumulation of stools in the rectum causes gradual dilatation leading to megarectum resulting in loss of rectal sensation and urge for defecation [14,15].
Our studies have confirmed that in all children with FC, the rectum enlarged relative to the age norm. Based on the X-ray examination, we subdivide patients with FC into 3 stages of megacolon and measure the length of the functioning part of the anal canal, which allows us to determine the degree of damage to the puborectalis muscle (PRM) [12,16]. The PRM is stretched under the influence of broad feces, the force of its contraction decreases, which leads to fecal incontinence. This condition is called descending perineum syndrome (DPS), which is usually diagnosed during defecography. We define DPS during a barium enema as a shortening of the function portion of the anal canal [5].

In patients with a normal anal sphincter, the puborectalis muscle relaxes and the anorectal angle widens, thus straightening the rectum and permitting the passage of stool during defecation. However, in patients with outlet obstruction from paradoxical puborectalis contraction, a paradoxical contraction of the puborectalis muscle occurs, resulting in a decrease in the anorectal angle, thus prohibiting the normal passage of stool. Some authors use the term dyssynergic defecation (DD). Some authors use the terms DD and PPC interchangeably [16]. Analysis of DD allows us to explain the mechanics of this pathological phenomenon. It is known that an increase in pressure in the rectum to a certain level causes a rectoanal inhibitory reflex, which results in relaxation of the internal anal sphincter (IAS), which is inhibited by a simultaneous contraction of the external anal sphincter (EAS) and PRM. The defecation reflex occurs at a higher rectal pressure. In patients with FC, which is always accompanied by megarectum, during an attempt to defecate, rectal pressure does not reach the threshold level of defecation. Therefore, instead of a defecation reflex, a rectoanal inhibitory reflex is provoked, i.e. contraction of EAS and PRM is not paradoxical, but a normal reaction of the anal canal to the enlarged rectum. Four types of DD depend on the degree of megacolon [17,18].
Thus, the pathogenesis of FC begins with a delay in feces in the rectum, followed by the development of megarectum and megacolon. The mismatch between the width of the feces with the throughput of the anal canal leads to damage to the PRM, which is accompanied by fecal incontinence.

The colon is part of the digestive system. The rectum and anal canal have a connection with other parts of this system. In healthy volunteers, in response to dilatation of the lower esophageal sphincter, a significant increase of the rectal pressure was shown. Shafik has suggested that this reaction is conducted through the mesenteric plexus from the esophagus down along the gut wall to the rectum, initiating rectal contractions [19]. Impacted feces in the rectum caused a deceleration of gastric emptying [20]. In response to the dilation of the balloon in the rectum, the decrease of pressure in the jejunum and ileum was seen. According to Shafik, this reaction inhibits the transit of the small intestine, allowing the rectum to expel its contents [21]. Voluntary suppression of defecation in healthy controls has been demonstrated to prolong total and regional colonic transit times indicating that a functional evacuation disorder influences the right colon [22].

Most patients with chronic childhood constipation had decreased innervation of the rectal mucosa [23]. Adachi et al found specific downregulation of c-kit in intestinal cells of Cajal (ICCs) in adults with sigmoid megacolon. The ICCs are detected as a pacemaker of gastrointestinal movement and express c-kit and CD34 [24]. Absence or intertenial interruption of the continuous connection tissue layer between the circular and longitudinal muscle of the muscular propria in the colon of patients with constipation has been termed aplastic or atrophic desmosis, respectively. Atrophic hypoganglionosis or atrophic desmosis with loss of myenteric plexus connective tissue is detected in the wall of the colon removed in adults with chronic idiopathic constipation [25]. Histological examination of surgical samples showed vacuolar degeneration of nerve plexuses as well as of
the muscular propria, which also showed fibrosis in its outer layers in patients with constipation [26].

Currently, most researchers have accepted the hypothesis of the different origins of STC and functional outlet obstruction constipation. The cause of STC is considered a primary neurogenic or myogenic disorder of the colon, and obstruction constipation is due to the impaired function of the anal canal. The only evidence for this separation is the normal width of the rectum in some patients with chronic constipation, which excludes the obstructive nature of the disease. All supporters of this hypothesis refer to a work where, based on a violation of scientific principles, the upper limit of the norm of the width of the rectum was erroneously established as 6.5-6.3 cm [11,27]. In FC, which always occurs due to obstruction, a large amount of feces is retained in the rectum and sigmoid colon, which causes the expansion of these departments. Since the sigmoid colon has a mesentery, it lengthens in 100% of cases. Thus, an obvious sign of obstruction is the sigmoid loop leaving the pelvis. If normally the width of the sigmoid colon is always smaller than the other parts of the colon, then with FD it is always wider than the descending colon [18]. "Dolichocolon, defined as an elongated colon, should not be seen as a cause of constipation" [28].

STC is not a cause, but a consequence of obstructive constipation. Firstly, because patients with a colostomy do not have constipation. Secondly, obstructive constipation is a chronic intestinal obstruction. It is not so pronounced as in Hirschsprung's disease, in which severe enterocolitis occurs. The fecal mass expand the rectum, and the left half of the colon. Enhanced and often ineffective peristalsis with retention of large volumes of stool leads to non-specific chronic inflammation with damage to the intestinal innervation, muscle damage of the colonic wall and the appearance of fibrosis. The longer the stool stays in the left half of the colon, the slower the movement of the bolus in the right half of the
colon. Moreover, the small bowel motor dysfunction was frequently associated with a co-existent rectal evacuatory disorder [29].

So why was paradoxical anal contraction observed in the constipated patients with distal delay but in none of the subjects with colon inertia [30, 31]? As shown above, an attempt to defecate in the presence of a widened rectum causes anorectal inhibitory reflex, i.e. relaxation of the internal anal sphincter and contraction of the puborectalis muscle and the external anal sphincter. In more severe cases of FC stretching and weakening of the puborectalis muscle occurs, which is defined as the descending perineal syndrome (DPS). In such cases, an increase in the rectal pressure is not accompanied by a contraction of the puborectalis muscle (Figure 1) [18]. In patients with DPS, chronic constipation is associated with fecal incontinence due to weakness in PRM. In the article, Benninga et al showed, that based on objective marker studies, their findings suggest the existence of pediatric slow-transit constipation. This entity can be recognized by clinical features, most importantly nighttime soiling, and a palpable rectal mass [32]. Thus, a severe form of STC (colonic inertia) indicates a more severe form of obstructive constipation.
Figure 1. Lateral radiographs of the anorectal zone. (A) In a healthy child, the rectum of normal width has vertical and horizontal branches. As a result of IAS relaxation, barium penetrated the upper part of the anal canal in front of the enema tip. The posterior wall of the anal canal is pressed to the tip as a result of the PRM contraction. The axis of the anal canal is shifted forward relative to the axis of the rectum. (B) Patient with Hirschsprung disease. Due to the absence of ganglia in the rectum, there is no neural connection between the rectal wall and PRM. As a result, PRM is not contracted and is not relaxed. So, the rectum is represented only by the vertical branch. The aganglionic rectum has a normal width since there is no colitis. Recto-sigmoid index <1. (C) Early-stage FC. As a result of the expansion of the rectum, its horizontal branch disappeared. The anal canal seems long due to PRM edema. This is evident by the impression on the lower wall of the rectum (arrow). (D) Due to the weakness of PRM, the contrast agent
penetrates the upper part of the anal canal behind the enema tip. (E) Descending perineum syndrome. A sharp expansion of the rectum is combined with a sharp shortening of the functioning anal canal.

**Conclusion.** Different degrees of STC are proportional to the severity of obstructive constipation. However, the study of the speed of transit through the colon in terms of accuracy and amount of information is inferior to the barium enema, according to our proposed method. From the point of view of formal logic, the study of transit through the colon in patients with FC has no practical significance. Moreover, there remains a difficulty in assigning specific treatments to subtypes of constipation based on transit measures [33] Other scintigraphic studies found that colonic retention is equivalent in groups of patients with or without a severe evacuation disorder [29].

**High-resolution manometry of the colon.**

A measure of the contractile activity that moves content through the colon is largely achieved with colonic manometry. In both adults and children with constipation, colonic manometry has identified abnormal colonic contractility, largely portrayed as an absent or diminished frequency of high amplitude propagating sequences (contraction). In addition, abnormal responses to physiological stimuli, such as a high-calorie meal, or morning waking have been detailed. As with transit studies these measures have been used to assist in the treatment of patients [8]. In adults, different types of constipation showed different colonic motility patterns and morphological changes in the colonic wall. For example, Li et al based on the amplitude, intensity, and trends in peristaltic contraction recorded by high-resolution colonic manometry, observed 117 patients with STC and 34 with functional outlet obstruction constipation. After an overall evaluation, 26, 23, 27, and 75 patients were treated with total colectomy, subtotal colectomy, local excision, and conservative treatment respectively [26].
The movement of a bolus along the colon obeys the law of the intestine [34]. Normally, the peristaltic wave is contracted to complete overlap of the lumen. Continuing to contract, it creates pressure on the wall, which through the intramural nervous system leads to relaxation of the distal segment, through which the bolus is pushed out by the contracted segment of the intestine (Figure 2, A). In FC in those segments of the colon where there are significant expansion and damage to the nerve element and muscle fibers, the force of contraction of the intestinal wall is weakened, the proximal segment does not overlap the intestinal lumen with the result that the pressure in the intestine is low and does not cause the distal segment to open (Figure 2, B). Therefore, in patients with constipation a reduced frequency of high amplitude propagating sequences, in comparison to healthy control, is one of the most reported findings. Moreover, low amplitude propagating sequences have been reported with a reduced or similar frequency in comparison to healthy control. Thus, pathological patterns of mobility are due to the expansion of the lumen of the colon and degenerative changes in its wall, which is caused by stagnation of feces. The greater the degree of megacolon and megarectum, the more pronounced symptoms of dysmotility.
Figure 2. Colonic motility in normal and with FC. (A) Scheme of the Bayliss-Snarling’s gut law. The peristaltic wave is contracted to complete overlap of the lumen. Continuing to contract, it creates pressure on the wall, which leads to relaxation of the distal segment, through which the bolus (b) is pushed out into the next segment. (B) In FC the force of contraction of the intestinal wall is weakened, the proximal segment does not overlap the intestinal lumen with the result that the pressure in the intestine is low and does not cause the distal segment to open. (C) X-ray of the abdomen 24 hours after taking barium. You can see the movement of separated barium portions. In front of the functional sphincter, several portions merge into one (red oval). During motion, the volume of portions decreases and their density. (D) This process as a result of water absorption continues in the rectum. (E) In a patient with FC and megacolon of the 3rd degree (the arrow shows the wall of the ascending colon), both motor function and speed of movement along the colon cannot be normal.

Conclusion. Based on the analysis of the literature, we concluded that the FC in children and adults is obstructive. At severe forms of PC which are accompanied by fecal incontinence and in which there is no "paradoxical" contraction of PRM, there is always a megarectum, and PRM incompetency, i.e. descending perineal syndrome. High-resolution colonic manometry in children begins after sedation, an hour after the introduction of the manometry catheter and X-ray control of its location. The study lasts several hours. It allows us to differentiate severe cases of FC from milder ones. Moreover, without scientific justification, it is believed that in severe cases there is a special form of neuropathy of the colon, which is subject to surgical treatment [1,35]. X-ray examination lasts no more than 30
minutes. On the frontal and lateral radiographs, there is accurate information about the size of each of the parts of the colon and the length of the anal canal, which allows you to make an accurate diagnosis and determine the degree of damage to the large intestine [12].

**High-resolution anorectal manometry**

Authors using high-resolution anorectal manometry compare different characteristics of the rectum and anal canal in patients with FC and in healthy individuals. Dinning et al rated these studies as follows: "High-resolution anorectal and colonic manometry provide a more comprehensive characterization of motility patterns and coordinated activity; this may help to improve our understanding of the normal physiology and pathophysiology in these regions. To date, however, no published study has conclusively demonstrated a clinical, diagnostic, or interventional advantage over conventional manometry» [36].

This characteristic is very superficial because: firstly, in none of the articles is there a reference to the normal physiology of the colon and anorectum. Secondly, there is no basic understanding of the pathological physiology of FC. For example, there is a lack of understanding of the mechanism of the so-called paradoxical reduction of PRM. None of the articles describes descending perineum syndrome. Thirdly, the upper limit of the norm of the width of the rectum for adults and children is determined with serious violations of scientific research, which led to results that are contrary to common sense. It creates most doctors and researchers a false idea about the true state of the problem.

Therefore, HRM cannot "help to improve our understanding of the normal physiology and pathophysiology in these regions" in the absence of a basic understanding of these problems.

**X-ray examination**
The method I use is based on knowledge of the standards for the width of different parts of the colon, including the rectum, as well as the length of the anal canal in children of different ages (Table 1). In adults, the maximum border of the width of the rectum is 3.5 - 4.8 (3.95 ± 0.21 cm), and the length of the anal canal is 3.4 - 4.2 (4.08 ± 0.07 cm). In FC, the width of the rectum and sigmoid colon is greater than the maximum normal limit in 100% of cases. The descending colon is expanded in 70% of cases. The average width of the right colon does not go beyond the norm.

Table 1. Normal sizes of the colon, rectum and anal canal in children of different ages.

<table>
<thead>
<tr>
<th>Age</th>
<th>Anal canal length</th>
<th>Widths of different parts of the intestine (cm)</th>
<th>Height of the cecum dome</th>
<th>Volume of the colon (ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-11 months</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>1.7 - 2.5</td>
<td>1.3 - 3.0</td>
<td>1.4 - 2.0</td>
<td>2.3 - 2.7</td>
</tr>
<tr>
<td></td>
<td>2.2±0.15</td>
<td>2.2±0.09</td>
<td>1.73±0.06</td>
<td>2.05±0.06</td>
</tr>
<tr>
<td>1-3 years</td>
<td>7</td>
<td>2.3 - 2.8</td>
<td>3.0 - 3.7</td>
<td>2.4 - 3.8</td>
</tr>
<tr>
<td></td>
<td>2.55±0.10</td>
<td>3.21±0.11</td>
<td>2.11±0.08</td>
<td>2.54±0.08</td>
</tr>
<tr>
<td>4-7 years</td>
<td>9</td>
<td>2.5 - 3.6</td>
<td>3.0 - 3.9</td>
<td>2.4 - 2.6</td>
</tr>
<tr>
<td></td>
<td>3.17±0.14</td>
<td>3.43±0.14</td>
<td>2.52±0.03</td>
<td>2.76±0.07</td>
</tr>
<tr>
<td>8-10 years</td>
<td>18</td>
<td>2.6 - 3.7</td>
<td>3.2 - 4.1</td>
<td>2.1 - 2.6</td>
</tr>
<tr>
<td></td>
<td>3.11±0.10</td>
<td>3.72±0.05</td>
<td>2.41±0.03</td>
<td>2.81±0.06</td>
</tr>
<tr>
<td>11-15 years</td>
<td>13</td>
<td>3.1 - 3.9</td>
<td>3.6 - 4.6</td>
<td>2.2 - 2.6</td>
</tr>
<tr>
<td></td>
<td>3.43±0.10</td>
<td>3.95±0.07</td>
<td>2.36±0.03</td>
<td>2.82±0.05</td>
</tr>
</tbody>
</table>

The method of barium enema and the method of analysis of the obtained data can be found in the articles that are published on my site [5,7,12]. The following is an example of the use of an X-ray examination, which, unlike HRM, lasts no more than 20-30 minutes after 2 radiographs are performed (Figure 3).
Figure 3. X-ray examination of a 12-year-old boy who was admitted to the hospital with complaints of abdominal pain and fecal incontinence. (A) Lateral radiograph of anorectum. The diameter of the X-ray contrast marker near the anus is 1.6 cm. The length of the functioning anal canal (blue line) is 1.9 cm (the minimum norm is 3.1 cm). The length of the normal anal canal (red line) from the anus to the pubococcygeal line is 3.9 cm (norm). The rectum 8 cm wide does not have a horizontal branch (the upper limit of the norm is 4.1 cm). (B) The sigmoid colon is elongated, so goes beyond the pelvis. Its width is 3.5 cm (the upper limit of the norm is 2.6 cm), and its haustration is asymmetric. The contour of the descending colon is uneven and not clear, which indicates an inflammatory process. Conclusion FC obstructive nature with pronounced megarectum with a descending perineum syndrome. Fecal incontinence is due to PRM failure and secondary colitis.

Knowledge of the pathophysiology of FC allows us to state with absolute certainty without a manometric study that in patients with FC the threshold of sensitivity and the capacity of the rectum is significantly increased, as well as a sharp decrease of the tone of the anal canal.

In order not to be unfounded, I propose to analyze the article by Gasior A. et al [37], in which all six of the above principles of scientific research are violated.
1. "All 31 patients had a contrast enema prior intervention and 87% of those were abnormal having either a dilated rectosigmoid or a redundant sigmoid loop". From this phrase, it follows that 3 (13%) of the children were operated on, even though their sigmoid colon was not lengthened and expanded. An abdominal radiograph is included in the article as evidence of elongation and expansion of the sigmoid colon (Figure 4).

Figure 4. The caption to the figure does not explain the important clinical and radiological symptoms. On the frontal abdominal radiograph, performed in a 6-7-year-old child, fecal impaction is determined. A contrast enema performed in such conditions makes no sense because it is possible to judge the size of the intestine only after the fecal disimpaction.

The article does not mention the width of the rectum and the length of the anal canal, although the radiograph shows, that the widest fecal stone located precisely in the rectum, which indicates a violation of the throughput of the anal canal. There is no indication in the article about the obstructive nature of the disease and
the descending perineum syndrome, which is absolutely necessary for understanding the causes of chronic constipation and fecal incontinence.

2. There is no scientific substantiation of indications for surgical treatment in the article, and the two principles that were used by the authors contradict common sense. FC is known to be a chronic disease requiring long-term treatment. In children, a long period of timely cleansing of the rectum leads to a complete recovery, as the throughput of the anal canal increases with growth. If during this time the rectum has ceased to expand, there comes a time when the width of the feces formed in the rectum come into correspondence with the width of the anal canal.

A) Failure of the week program with high dose Senna, cannot be an indication for surgical treatment since it has no scientific basis. This is not only meaningless but also a dangerous experiment [38].

B) Colonic manometry is not a diagnostic method. Its results cannot serve as an indication for surgery. But it was the combination of these two false methods that led, at least to 3 meaningless operations, where the sigmoid colon was of normal size. Our study showed that in patients with FC, the expansion of the rectum in 100% of cases is combined with the expansion of the sigmoid colon. Based on this, we can state that in these 3 cases the rectum was also of normal width. Chronic constipation and fecal incontinence in these patients is explained by the initial phase of the disease. Secondly, the authors, as can be seen from the above, have erroneous criteria for assessing the size of the colon [1].

3. There is no justification for the surgical treatment of children with FC at the age of 4 years. There is no justification for the surgical treatment of children with FC without using a variety of conservative methods, including botox injection to the intersphincteric plane or the EAS, which may offer an excellent minimally invasive therapy with low morbidity and good outcome [39].
4. The resection of megadichosigma does not eliminate the main cause of the disease, since the sharply expanded rectum, located retroperitoneally, will continue to form fecal masses of large diameter, and therefore the discrepancy between the large width of the fecal masses and the throughput of the anal canal will remain. Secondly, the weakness of the puborectalis muscle will not disappear, which will allow the more liquid feces coming from the descending intestine to pass, predisposing to fecal incontinence. As Duhamel showed, this operation does not relieve patients of problems [6]. Figure 5 shows the result after the removal of the megadichosigma. Does it really take another 10 years to reach the conclusion reached by surgeons from previous generations?

![Figure 5](image_url)

*Note the discrepancy in the caliber of the rectal cuff and the sigmoid colon.*

**Figure 5.** After an intraperitoneal rectosigmoid anastomosis, the enlarged rectum forms broad stool poorly passed through the anal canal. (radiograph from an article by Glasser et al. [40].)
The above analysis shows that articles by pediatric surgeons from the Center for Colorectal and Pelvic Reconstruction from Nationwide Children's Hospital in Columbus cannot be considered scientific. It is surprising how these articles received the approval of the ethics committee, why reviewers gave a positive assessment of these works. The publication of these works opened a Pandora’s box, as a result of which pediatric journals publish articles with the same unforgivable errors. An example is an article by De la Torre et al. [41].

The authors "reviewed the charts of patients with a diagnosis of chronic idiopathic constipation with megarectosigmoid confirmed in all by contrast enema". Cases of fecal impaction are shown as a demonstration (Figure 6).

Figure 6. Cases with fecal impaction from article De la Torre et al [41]. (a) The authors introduced a contrast agent in fecal impaction, which is an acute surgical condition. It arose sharply, and the wall of the sigmoid colon, possessing elasticity, after disimpaction, can significantly contract. Proximal to the huge fecal stone identifies symptoms indicating severe colitis. (b) On a lateral CT scan in a 16-year-old patient with fecal impaction, feces are in the expanded rectum proximal to the pubococcygeal line (the red line is drawn by me). This indicates the normal function of the puborectalis muscle.
Such studies do not make any sense, firstly, because they were performed during fecal impaction. Secondly, because there are no criteria for the degree of megarectum. This characteristic is very important since all patients with FC have an expansion of the rectum, but not everyone needs to be operated on. Thirdly, there is no assessment of the state of the puborectalis muscle. If it is weakened, then the connection of the rectum with the proximal part of the sigmoid colon, which brings to the rectum relatively liquid feces, will inevitably lead to fecal incontinence.

Indications for the surgery are copied from the articles of the colorectal group from Columbus. "In 9 patients, indication for surgery was chronic intake (> 4 years) of a daily high dose of Senna with failed weaning trials. The parents in this group refused to continue giving their children the laxative. The indication in the other 4 patients was because they could not tolerate the stimulant laxative and rejected the rectal enemas. Thus, these patients continued to have fecal impactions and pseudofecal incontinence. In this group, 2 teenagers with long-standing severe pseudofecal incontinence suffered depression and anxiety and 1 of them attempted suicide".

The use of this method not only has no scientific justification but is dangerous [38]. Change laxative - this is the only logical conclusion from this text. You can understand the children and their parents who, after the unbearable torment caused by the high doses of Senna, agreed to the operation. But it is impossible to understand surgeons who blindly followed up on non-responsible recommendations. The article has no radiological or clinical justification for the surgical treatment of the patients' ages ranged from 21 months to 19 years (median, 8 years). Surgery in a child with FC under the age of one year is obviously absurd.
From the point of view of the pathological physiology of FC, the transanal proximal rectosigmoidectomy method described by the authors is quite justified and has an advantage over sigmoidectomy, since most of the rectum is removed and the large volume of stool is not collected in the remainder of the rectum.

Even though patients with FC over 18 years old are recent pediatric patients and adult doctors have much more experience of treatment than pediatricians do, all the references in this article are limited to pediatric surgeons. Because of this, a gap between knowledge in pediatric and adult literature arose. For example, the authors of this article claim, "Pelvic outlet obstruction was ruled out in all these patients". Diagnostic tests for the diagnosis of pelvic outlet obstruction or obstructed defecation syndrome include intestinal transit studies, anorectal manometry, defecography, ball expulsion, and anal sphincter electromyography. Firstly, the authors did not produce any of the above studies. Secondly, a sharp expansion of the rectum (megarectum) is proof of anal obstruction.

Dear colleagues,

I suggest you try the above method of radiological diagnosis in patients with FC and give it your assessment.

Respectfully,

Michael Levin, MD, PhD, DSc. Radiologist,

Department of Pediatric Radiology of the 1-st State Hospital, Minsk, Belarus.

Dorot-Netanya Geriatric Medical Center, Israel.

Amnon VeTamar, 1/2, Netanya, 42202, Israel.

nivel70@hotmail.com; michael.levin@dorot.health.gov.il

https://orcid.org/0000-0001-7830-1944
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