Letter to the Editors

I thank Dr. Gold for his comments on my paper *(Descending perineum syndrome: pathophysiology of fecal incontinence, Pelviperineology 2018; 37 (2): 57-62)* and I write to offer further comments.

The premise is that aim of paper was "to understand the types and pathophysiology of fecal incontinence in patients affected by DPS". The conclusions "Urge incontinence, related to external anal sphincter atrophy, is the predominant pattern of fecal incontinence" are supported by instrumental data and are not hypotheses or theories.

The participation of the pelvic floor at the fecal incontinence in the descending perineum syndrome is abundantly addressed in the discussion. "When rectoanal intussusception is combined with levator hiatal widening and levator plate descent, it can become the morphological pathology underlying DPS fecal incontinence[22]. It is difficult to provide a single pathophysiological framework for DPS fecal incontinence. Although a multifactorial etiology seems have an impact on a weak pelvic floor, it is very difficult to understand how much a single factor may destabilize the descending perineum. Surely a descending perineum possesses per se a pathological structure of pelviperineal muscles, perineal body and supportive elements of the endopelvic fascia that can lead to fecal incontinence. For example, lax suspensory ligaments that inactivate striated pelvic muscle forces[23], increased collagen breakdown such as a pathological etiology of urinary incontinence and pelvic organ prolapse[24], the observation that 45% of patients with joint hypermotility, stool evacuatory disorders and abnormal connective tissue also have fecal incontinence not due to sphincter dysfunction[25], are all evidence that an impaired pelvic floor may be associated with fecal incontinence". How much the individual muscles, puborectalis, pubococcygeus and ileococcygeus, are singularly involved in fecal incontinence, it is impossible, in the present state of instrumental diagnostics, to demonstrate such a thing objectively, as suggested by Dr. Gold. Returning to the aims of our paper, they were 1) to describe the clinical profile of fecal incontinence and 2) to identify the main pathophysiological mechanisms of fecal incontinence. As regards 1), we described an extensive data base, 1261 patients. As regards 2) we drew conclusions about pathophysiology based on our data. One view was that Levator ani muscle is part of the pelvic floor and its involvement in the descending perineum syndrome is supported by "levator hiatal widening and levator plate descent". These may well be, as Dr Gold states, hypotheses. As hypotheses, they are there to be questioned, to be proven or disproven, as Dr Gold has done. That is how medical science progresses. We appreciate Dr Gold's comment that the Integral Theory has not as yet been invalidated. Some, even many of the correlations made in our results may well be supportive of the Integral Theory. Some will not be. These would need a critical analysis involving far more than the short comment made here by Dr Gold. We invite Dr Gold and his colleagues to make such an analysis. We would, of course, be happy to assist any such endeavour.

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