Update on the pathophysiology of rectal prolapse

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For decades, rectal prolapse has been hypothesized to be caused due to laxity or weakness of the pelvic floor muscles. Childbearing certainly plays a role in the development of pelvic floor laxity in females. However, 50% of females with rectal prolapse are nulliparous. Furthermore, this hypothesis does not explain the incidence of rectal prolapse in males. It has been suggested that this condition is associated with psychiatric disorders (1-3).

Herein, we suggest a novel hypothesis to explain the pathogenesis of rectal prolapse in both males and females. We hypothesize that straining causes an increase in the intra-abdominal pressure, leading to inversion of the rectal wall at the level of the pelvic peritoneal reflection (cul-de-sac, i.e., rectovesical pouch in males or rectouterine pouch in females), which eventually presents clinically as rectal prolapse.

The rectum is located anterior to the sacrum. It passes through the pelvic diaphragm separating the rectum into pelvic and anal parts. The anterior wall of the upper rectum is covered by peritoneum, whereas the middle and lower parts lie below the peritoneal reflection (4). While being completely embedded in the extraperitoneum and supported by the levator muscles and anal sphincter complex, the lower part of the rectum is well fixed and able to sustain increased intra-abdominal pressure. We hypothesize that straining causes increased intra-peritoneal pressure, which caudally pushes the peritoneal reflection along with the anterior wall of the rectum. We believe that an increase in intra-abdominal pressure forces a redundant and hypermobile part of the rectum caudally across the level of cul-de-sac and through the anorectal hiatus. In the presence of a sufficiently long and mobile rectum, the typical telescoping appearance of the rectal wall prolapsing through the anus is observed. On the other hand, an internal intussusception occurs in patients with a shorter and less redundant rectum.

We suggest that the surgical procedures currently performed for rectal prolapse further support our hypothesis. Fixation of the rectum to the sacrum resolves this condition by preventing its caudal displacement without any intervention on the pelvic floor. Alternatively, in the Altemeier's procedure, transanal resection of the redundant rectum or plication of the rectal wall is performed (5), which results in a shorter rectum, thereby supporting our hypothesis. Therefore, we believe that the etiology underlying rectal prolapse depends on the rectal anatomy rather than on an intrinsic dysfunction of the pelvic floor muscles. The anatomical variations of a redundant rectum or hypermobility of the rectal wall may predispose to rectal prolapse in certain individuals.

We built a simulator to test our hypothesis. A tubular balloon was insufflated, and its tip was fixed at the only opening of a closed box. This apparatus represents the rectum and sigmoid colon within the abdominal cavity as shown in the illustration (Figure 1). Moreover, a piston was installed to increase the pressure in the box. Upon

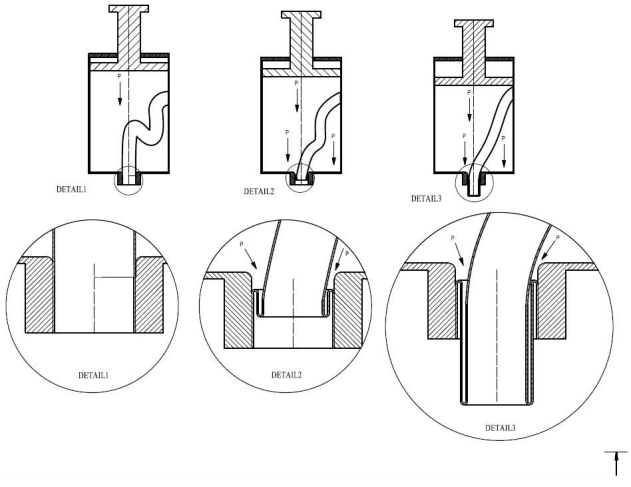

Figure 1. Illustration of prolapse upon increasing the pressure with a piston in the closed box.

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increasing the pressure, inversion (or intussusception) of the balloon at the point where it was fixed to the outlet was initially noticed. Further increase in pressure caused the balloon to prolapse completely through the outlet. We believe that this simulation clearly demonstrates the process of rectal prolapse and supports our hypothesis.

Further clinical research is needed to evaluate the actual length of the rectum and the degree of its motility in patients with rectal prolapse and to compare these data with those of a control population. Additional radiological imaging to check for levator muscle defects may further test our hypothesis.

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