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Editorial

Is anorectal assessment really useful?

The present issue of *REED* includes two papers examining the usefulness of anorectal function assessment (1,2) where authors wonder when may this testing provide relevant information for patient care.

The distal portion of the gastrointestinal (GI) tract, the anorectum, has controlled closure mechanisms that allow continence on the one hand and voluntary fecal evacuation on the other. Key elements for this task include the rectum, acting like a reservoir, and ano-perineal muscles, which exert the closing function. These elements are controlled by a complex series of neuronal circuits within the intestinal wall itself and via afferent and efferent fibers from and to the extrinsic nervous system. Basically, anorectal function changes clinically manifest as incontinence, distal constipation, and ano-perineal pain. The fact that some functional changes may secondarily lead to organic disorders should also be borne in mind (3,4).

When dysfunction is suspected in one of these mechanisms there is an indication for anorectal function assessment. However, to which extent may these tests identify changes accounting for the origin of symptoms and help in therapy planning? The answer is that, for patient assessment, a consistent, complete evaluation of the various functional components is mandatory, since clinical alterations are usually multifactorial (3,5). A set of tests is now available to explore the neuromuscular aspects of the GI tract and perineal floor. Each test provides partial information; their joint assessment provides an overview of anorectal function and allows a pathophysiological interpretation of clinical changes, as well as therapy guidance. Otherwise, we may find patients with unaccounted for manifestations – incontinence with normal sphincters (1), anal fissure without sphincter hypertonus (2), and idiopathic constipation or perineal pain with no identifiable cause. Furthermore, given the high anorectal capability for functional compensation, an isolated disorder may have no clinical impact, which in turn renders normal value measurements difficult.

¿What should be assessed and how can results be interpreted?

Four groups of parameters will provide information on the rectum's functional status, ano-perineal muscle activity, control neural pathways, and evacuation capacity. Identifying specific changes may help in patient management.

1. *Rectal function changes*. The rectum is usually empty and the fecal bolus gives rise to perceived tenesmus on arrival. To assess rectal function in a laboratory the rectum is gradually distended with a bag or balloon – intrarectal pressure is measured to estimate distensibility, as is perceived distension at various levels as an index of sensitivity (3,6).

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—*Decreased rectal capacity/increased sensitivity*. This type of changes may result in incontinence and defecatory urgency. The arrival in the rectum of small fecal volumes causes rectal wall hypertension and fecal leaking from overflow. Fecal bolus volume should be reduced in these patients, as well as fiber ingestion, to prevent overflow in a insufficient rectum.

–*Increased rectal capacity/decreased sensitivity*. In the presence of constipation, mainly from expulsion difficulties, rectal capacity usually increases and rectal perception is usually reduced to some extent. Patients with neurological changes commonly present with decreased rectal sensitivity, even complete anesthesia in case of medullary section (7). Defective sensitivity usually results in fecal retention but may occasionally be associated with incontinence, actually from a decrease in perceived rectal filling. Reduced rectal sensitivity is only clinically relevant when severe (7), and sensory rehabilitation therapy through biofeedback may be attempted in such cases. Patients with complete rectal anesthesia and no perceived tenesmus should be instructed to evacuate on a schedule, even with a regular regimen of cleaning enemas to keep the rectum free from potentially leaking remnants.

2. *Ano-perineal muscular activity changes*. The anal canal is surrounded by an inner sphincter made up of smooth muscle that is constantly in a sustained tonic contraction except for brief episodic relaxation events. The outer sphincter surrounds this inner sphincter and includes striated muscle allowing the induction of additional voluntary contractions when needed. Sphincter function may be assessed using conventional manometry (3,5,6).

—*Decreased contractile capacity* may be associated with incontinence of gas, fluid feces or even stools of normal consistency. It should be noted that, besides anal sphincters, perineal floor muscles, especially the levator ani, also plays a significant role for continence maintenance – a perineal change may explain incontinence with preserved sphincters. Muscle insufficiency may be managed with rehabilitation using biofeedback (8).

—*Increased internal sphincter pressure* is a pathophysiological mechanism for anal fissure, and a good means to assess therapy (2). On the other hand, *puborectal muscle contraction* results in levator ani syndrome with chronic proctalgia, which usually worsens during prolonged sitting and subsides with lying down (5).

3. *Neuropathic changes*.

—*Intrinsic innervation* is provided by the extended myenteric plexus and specifically controls internal sphincter function. To assess intrinsic innervation in the laboratory the inhibitory anorectal reflex is used (3,6). This test is clinically useful in patients with suspected Hirschprung's disease, that is, with severe constipation since childhood. A present reflex rules out this disease. A repeatedly absent reflex suggests a disordered myenteric plexus and is an indication for rectal biopsy in order to confirm aganglionosis.

—*Extrinsic innervation* includes a number of efferent motor pathways that control external sphincter and perineal floor function. An increase in intraabdominal pressure stimulates mechanoreceptors probably on the perineal floor, and triggers a multisynaptic spinal reflex that contracts the external sphincter (3,6,7). Physiologically, this reflex automatically closes the anal canal and preserves continence during efforts. In case of pudendal neuropathy (e,g., patients with a history of obstetric trauma, diabetes, or alcoholism) or of medullary lesions in sacral segments (e.g., spina bifida) this reflex is impaired and stress incontinence may arise (7).

4. *Defecatory disorders*. The defecatory maneuver includes an abdominal compression associated with sphincter relaxation, which allows the fecal bolus to be ex-

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pelled. In order to assess the defecatory maneuver in the laboratory the increase in intrarectal pressure and the fall in anal pressure are simultaneously measured with a manometric probe (3,6). A relatively common cause of impaired defecatory maneuver is defective anal relaxation (3,5), which may give rise to two relevant consequences.

—Defecation usually empties the rectum. In case of defective expulsion *incomplete evacuation* may ensue with feces retained in the rectum. This disorder may result in incontinence, particularly when sphincter weakness is associated.

—On the other hand, patients with incomplete anal relaxation usually exert excessive abdominal compression to make up for expelling difficulties. This *ano-perineal trauma* may bring about functional proctalgia events because of painful striated muscle contracture or anal lesions such as fissure or hemorrhoids. A high proportion of patients with anal fissure have no hypertonus (2,5), and the lesion usually results from defecatory impairment. Repeat perineal stress may in the long run induce stress-related perineal damage (4), and even jeopardize continence. In fact, constipation is the sole functional disorder that may independently predict a poor response of incontinence to rehabilitation (9).

Defecatory disorders may be managed with muscle coordination techniques using biofeedback (10-12) whereby patients learn to relax their anal canal while preventing excessive abdominal compression.

Conclusion

An assessment of anorectal function is indicated in patients with anal incontinence, suspected expulsion defects (with or without associated lesions), and anoperineal pain, including anal fissure. The usefulness of such tests depends on a systematic assessment of functional parameters, and their overall interpretation. As novel techniques are developed to assess relevant anorectal function parameters, both the diagnostic sensitivity of these tests and their implications for treatment increase.

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