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Increased anal basal pressure in chronic anal fissures may be caused by overreaction of the anal-external sphincter continence reflex

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ABSTRACT

Chronic anal fissure is a painful disorder caused by linear ulcers in the distal anal mucosa. Even though it counts as one of the most common benign anorectal disorders, its precise etiology and pathophysiology remains unclear. Current thinking is that anal fissures are caused by anal trauma and pain, which leads to internal anal sphincter hypertonia. Increased anal basal pressure leads to diminished anodermal blood flow and local ischemia, which delays healing and leads to chronic anal fissure. The current treatment of choice for chronic anal fissure is either lateral internal sphincterotomy or botulinum toxin injections.

In contrast to current thinking, we hypothesize that the external, rather than the internal, anal sphincter is responsible for increased anal basal pressure in patients suffering from chronic anal fissure. We think that damage to the anal mucosa leads to hypersensitivity of the contact receptors of the anal-external sphincter continence reflex, resulting in overreaction of the reflex. Overreaction causes spasm of the external anal sphincter. This in turn leads to increased anal basal pressure, diminished anodermal blood flow, and ischemia. Ischemia, finally, prevents the anal fissure from healing.

Our hypothesis is supported by two findings. The first concerned a chronic anal fissure patient with increased anal basal pressure (170 mmHg) who had undergone lateral sphincterotomy. Directly after the operation, while the submucosal anesthetic was still active, basal anal pressure decreased to 80 mmHg. Seven hours after the operation, when the anesthetic had completely worn off, basal anal pressure increased again to 125 mmHg, even though the internal anal sphincter could no longer be responsible for the increase. Second, in contrast to previous studies, recent studies demonstrated that botulinum toxin influences external anal sphincter activity and, because it is a striated muscle relaxant, it seems reasonable to presume that it affects the striated *external* anal sphincter, rather than the smooth *internal* anal sphincter.

If our hypothesis is proved correct, the treatment option of lateral internal sphincterotomy should be abandoned in patients suffering from chronic anal fissures, since it fails to eliminate the cause of high anal basal pressure. Additionally, lateral internal sphincterotomy may cause damage to the anal-external sphincter continence reflex, resulting in fecal incontinence. Instead, higher doses of botulinum toxin should be administered to those patients suffering from chronic anal fissure who appeared unresponsive to lower doses.

Introduction

Anal fissures, one of the most common benign anorectal disorders, are painful linear ulcers in the anal mucosa, distal to the dentate line. In 80–90% of the cases they are located in the posterior midline. If, with conservative management, anal fissures do not heal within six weeks, the disorder is defined as chronic anal fissure (CAF) [1–4].

To date, the precise etiology and pathophysiology of anal fissures is incompletely understood. Current thinking is that primary anal fissures are caused by local trauma, for example, passage of hard fecal mass, diarrhea, or vaginal delivery [1–4]. A minority of ulcers, classified as secondary anal fissures, are caused by malignancies, anal surgical procedures, or other diseases, such as

Abbreviations: AESCR, anal-external sphincter continence reflex; Botox, botulinum toxin; CAF, chronic anal fissure; EAS, external anal sphincter; IAS, internal anal sphincter; MABP, maximum anal basal pressure.

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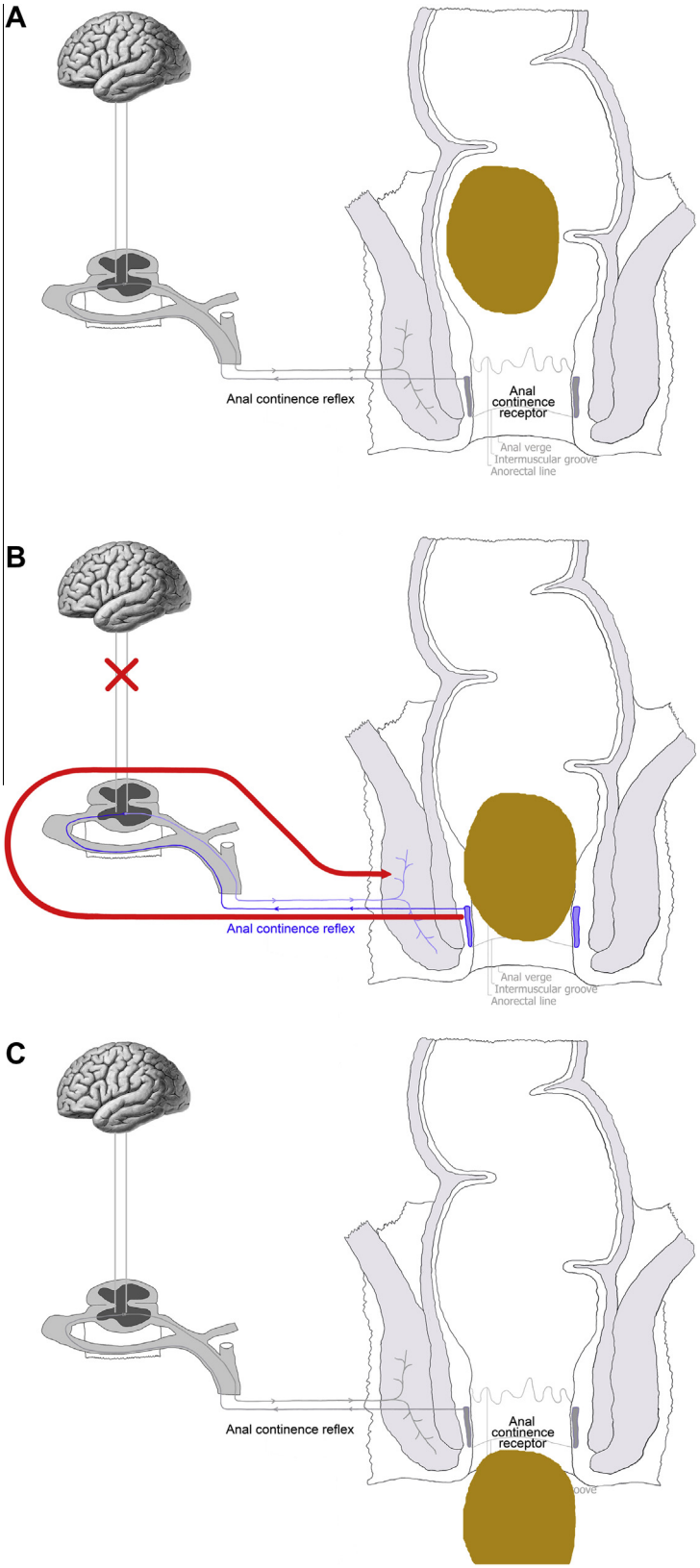

Fig. 1. Diagram representing the stages of fecal continence with the anal-external sphincter continence reflex (AESCR). (A) Fecal content high up in the rectum. The AESCR is at rest. (B) Fecal content in the anal canal. The AESCR activates the external anal sphincter to contract, thus controlling fecal continence. This is a stable situation until urge sensation is reached. Subsequently, one of two possibilities arise, either this status is supported by the brain and the fecal mass moves back up into the rectum, or the body overrules this status by increasing abdominal pressure and relaxing the external anal sphincter, causing the fecal mass to be expelled. (C) After the fecal mass is expelled, the AESCR is once again at rest. Reproduced, with permissions of the publisher, from Broens PM, Penninckx FM, Ochoa JB. Fecal continence revisited: the anal external sphincter continence reflex [16].