Original article

Experimental Study No. 5: A prospective endoanal ultrasound study suggests that internal anal sphincter damage is unlikely to be a major cause of fecal incontinence

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Abstract: A prospective endoanal ultrasound study in 80 patients with fecal incontinence (FI) normal external anal sphincters found internal anal sphincter (IAS) thinning in only 39% of patients. This experiment indicates that IAS damage is unlikely to be a major cause of FI.

Key words: Fecal incontinence; Internal anal sphincter; Endoanal ultrasound.

INTRODUCTION

The exact role of the internal anal sphincter (IAS) in the maintenance of fecal continence (FI) is not well understood. Controversy exists as to whether a damaged IAS can cause fecal incontinence. Bartolo and Macdonald [1] report an incidence of up to 40% incontinence of feces and flatus in patients who had undergone complete internal sphincterotomy. Endoanal ultrasound studies in 127 patients after vaginal delivery, demonstrated IAS damage in 49 patients.[2] Sphincteric damage was associated with fecal incontinence (FI) in 10/11 of these patients, implying causation of FI. The number of patients who had IAS and EAS (external anal sphincter) damage was not specified. These data did not fit with one author's(PP) previous experience: [3] of 25 patients with idiopathic FI, 72% had normal IAS and some patients had never been pregnant. Furthermore, reports of FI in 7 patients who had never been pregnant [4] indicate that other factors besides damaged IAS may cause idiopathic FI. The aim of this study was to challenge the internal anal sphincter theory by a prospective review of IAS damage in patients with FI. The fecal incontinence described in this study was of the 'idiopathic' type.

METHODS

There was no patient selection. The mean age of the group (n = 80) was 60.6 years (range 24-88), mean parity 3 (range 0-6). All patients were assessed according to a standard protocol involving a semiquantitative patient-administered questionnaire, urinary diary, 24 hour pad testing, transperineal ultrasound and urodynamics. All the endoanal ultrasound examinations were conducted by a specialist in ultrasonic imaging (JA), using a transrectal Acuson 7 MHz linear array axial sector scanner (focal range 0.5 cm to 8 cm). As this review conforms to the standards established by the NHMRC for ethical quality review, ethics approval was not sought.

RESULTS

Fecal incontinence was defined as involuntary loss of significant liquid or solid material at least once per day. Three patients were nulliparous, and ranged in age from 24 to 88 years. Seventy-eight patients had double incontinence, urinary and fecal, and two had fecal incontinence associated with vaginal prolapse, but no urinary incontinence. Two patients reported repair of a 3rd degree tear during labour, and another had undergone sphincterotomy. At the time of assessment, however, the EAS was intact on direct and ultrasonic assessment in all patients. IAS damage was defined as complete rupture, or thinning to less than 2 mm thickness in some part of the sphincter. Complete IAS rupture was found in one patient, and damage in a further 30 (total 39%). The rest (61%) had normal IAS. All 3 nulliparous patients had normal IAS and EAS.

DISCUSSION

Only 39% of 80 patients had evidence of any IAS damage. The others had no obvious cause for their FI. Even in Sultan's group of 49 patients with IAS defects, only 11 actually had fecal incontinence (22%), and one had no IAS or EAS damage whatsoever.[2] Three nulliparous females with normal IAS in our study confirms previous reports of FI in 7 nulliparous patients,[4] such data being consistent with a congenital connective tissue defect. No alteration in IAS morphology post-operatively was observed on endoanal ultrasound in 3 patients with damaged IAS who were surgically cured of fecal incontinence.[3]

CONCLUSION

Though our results indicate that internal anal sphincter damage is unlikely to be a major direct cause of idiopathic incontinence, its anatomical position mandates a significant role in continence control. We believe this role is limited to creating airtight and watertight closure.

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