Gut, 1991, 32, 345-346

Gut

Leading article

The internal anal sphincter – new insights into faecal incontinence

Application of neurological techniques to the pelvic floor over the past 10 years has increased our understanding of the pathophysiology of idiopathic faecal incontinence.[1-7] We now realise that damage to the innervation of the striated external anal sphincter and puborectalis muscles leads to progressive denervation with muscle fibrosis and weakness. These abnormalities can be shown clinically using manometry, measurement of the conduction time of the innervating nerves, and electromyography with either concentric or single fibre techniques. These neurological changes are thought in many instances to be the result of ongoing damage from earlier childbirth.[8]

The sphincter mechanism is a delicate balance of both striated pelvic floor and visceral smooth muscles. Yet the role of the smooth muscle internal anal sphincter in idiopathic faecal incontinence has been sadly neglected. Over the last two years, however, refinement of physiological measurement techniques, new modes of sphincter imaging, and the application of in vitro pharmacological studies have begun to shed light on the pronounced abnormalities of the internal anal sphincter which exist in this condition.

The first clue that the internal anal sphincter may be abnormal in this condition came from manometric studies which showed that two thirds of patients with idiopathic faecal incontinence have a reduced resting anal canal pressure.[4] Approximately 85% of this resting tone is contributed to by the internal anal sphincter.[9 10] If the sphincter mechanism is experimentally stressed by the infusion of saline, most patients with idiopathic faecal incontinence leak at a lower volume than normal.[11] In many of these patients inappropriate sphincter relaxation is thought to contribute to this leakage. More recently prolonged manometric recordings in the anal canal have shown that the internal anal sphincter undergoes spontaneous episodes of relaxation, resulting in transient falls in anal pressure. A study at rest has shown that incontinent patients exhibit more episodes of relaxation and that the pressure fall is lower than in control subjects.[12] In addition, fewer incontinent subjects showed a compensatory protective increase in external anal sphincter electrical activity. Lower than normal rectal volumes were required in some of these patients to produce sphincter relaxation. These findings suggest that the control of internal anal sphincter function is likely to be abnormal in these patients. Further evidence comes from prolonged ambulatory measurement of sphincter pressure using solid state pressure transducers and data collection. Twenty four hour studies have confirmed that spontaneous transient relaxations of the internal anal sphincter are more frequent[13] and of longer duration in idiopathic faecal incontinence.

Because both the internal and external anal sphincters exert an effect on anal pressures it would be useful to monitor internal anal sphincter activity directly. The introduction of electromyography of the sphincter allows confirmation that simultaneously measured falls in anal pressure are related to sphincter relaxation. Smooth muscle electromyographic activity can be recorded with either surface electrodes[14] or two fine hooked wire electrodes.[15] A study of patients with faecal incontinence using surface electrodes to record internal sphincter electrical activity showed that in incontinence the slow wave activity present in controls was often not recordable.[16] A recent study with bipolar wire electrodes has shown that there is a correlation between the frequency of the internal anal sphincter smooth muscle electrical oscillation and the resting anal pressure. In addition, the frequency of the slow wave activity was reduced in faecal incontinence.[15] This change in underlying electrical rhythm requires confirmation.

Another major development which provides insight into the structure of the sphincter is anal endosonography. For the first time we are able to image the sphincters and clearly distinguish between the smooth echogenic appearance of the internal anal sphincter and the characteristic heterogeneous echo of striated external anal sphincter.[17] In a study which used anal manometry and endosonography, there was a strong correlation between the endosonographic thickness of the internal anal sphincter and the resting pressure. Incontinent patients with a lower than normal resting pressure were found to have an abnormally thin sphincter. In addition, some patients with idiopathic incontinence were found to have an unsuspected external anal sphincter defect.[18]

Ultrastructural studies of the internal anal sphincter show pronounced abnormalities in patients with faecal incontinence.[19] The main abnormalities seen using electron microscopy and light microscopy are loss of smooth muscle cells, increased collagen fibril content, stretching of elastic tissue, and disruption of the normal architecture.

Can we relate these observed functional and structural findings in patients with idiopathic faecal incontinence to changes in the innervation of the internal anal sphincter? In vitro pharmacological studies have provided valuable information about abnormalities in both the extrinsic and intrinsic innervation of the internal anal sphincter.[20 21] The sphincter receives its intrinsic innervation directly from the myenteric and possibly other gut wall plexuses. The exact

nature and role of the autonomic extrinsic innervation is more controversial, but most likely comprises both a sympathetic supply which causes contraction and a parasympathetic supply whose function may be inhibitory,[22 23] may maintain tone,[24] or possibly modulate control of the sympathetic extrinsic and intrinsic inhibitory nerves.[25 26]

Organ bath studies of internal anal sphincter muscle from patients with idiopathic incontinence, removed during post-anal repair, have shown pronounced pharmacological abnormalities.[20 21] In faecal incontinence there is a decreased contractile sensitivity to noradrenaline, suggesting abnormal extrinsic α adrenergic innervation. The response to electrical field stimulation is also altered, suggesting abnormal intrinsic innervation. The latter is a non-adrenergic, non-cholinergic innervation whose activity is modified by the sympathetic extrinsic innervation. These pharmacological changes suggest that denervation may be important in causing decreased internal anal sphincter function and provide a rationale for future drug treatment. Changes in the resting pressure, electrical activity, and spontaneous relaxations of the internal anal sphincter may also be due to an abnormality of the innervation of the sphincter.

We can now appreciate the crucial nature of the contributions that both the smooth and the striated muscles make in maintaining continence. Idiopathic faecal incontinence is almost certainly not just one homogeneous condition, but a heterogeneous syndrome with one final disabling symptom. We must strive to identify these subgroups, using both clinical and in vitro methods, if we are to arrive at specific treatments.

C T M SPEAKMAN
M A KAMM

St Mark's Hospital,
City Road,
London EC1V 2PS

Correspondence to: Dr M A Kamm

1 Parks AG, Swash M, Urich H. Sphincter denervation in anorectal incontinence and rectal prolapse. *Gut* 1977; **19**: 656–65.
2 Beersiek F, Parks AG, Swash M. Pathogenesis of anorectal incontinence: a histometric study of the anal canal musculature. *J Neurol Sci* 1979; **42**: 111–27.
3 Neill ME, Swash M. Increased motor unit fibre density in the external anal sphincter muscle in anorectal incontinence: a single fibre EMG study. *J Neurol Neurosurg Psychiatry* 1980; **43**: 343–7.
4 Neill ME, Parks AG, Swash M. Physiological studies of the pelvic floor in idiopathic faecal incontinence and rectal prolapse. *Br J Surg* 1981; **68**: 531–6.
5 Kiff ES, Swash M. Slowed conduction in the pudendal nerves in idiopathic (neurogenic) faecal incontinence. *Br J Surg* 1984; **71**: 614–6.
6 Bartolo DCC, Jarratt JA, Read MG, Donnelly TC, Read NW. The role of partial denervation of the puborectalis muscle in idiopathic faecal incontinence. *Br J Surg* 1983; **70**: 664–7.
7 Womack NR, Morrison JFB, Williams NS. The role of pelvic floor denervation in the aetiology of idiopathic faecal incontinence. *Br J Surg* 1986; **73**: 404–7.
8 Snooks SJ, Swash M, Setchell M, Henry MM. Injury to innervation of pelvic floor sphincter musculature in childbirth. *Lancet* 1984; ii: 546–50.
9 Frenckner B, Euler CV. Influence of pudendal block on the function of the anal sphincters. *Gut* 1975; **16**: 482–9.
10 Duthie HL, Watts JM. Contribution of the external anal sphincter to the pressure zone in the anal canal. *Gut* 1965; **6**: 64–8.
11 Read NW, Haynes WG, Bartolo DCC, *et al.* Use of anorectal manometry during rectal infusion of saline to investigate sphincter function in incontinent patients. *Gastroenterology* 1983; **85**: 105–13.
12 Sun WM, Read NW, Miner PB, Kerrigan DD, Donnelly TC. The role of transient internal sphincter relaxation in faecal incontinence? *Int J Colorectal Dis* 1990; **5**: 31–6.
13 Kumar D, Waldron DL, Williams NS. Home assessment of anorectal motility and external sphincter EMG in idiopathic faecal incontinence. *Br J Surg* 1989; **76**: 635–6.
14 Wankling WJ, Brown BH, Collins CD, Duthie HL. Basal electrical activity in the anal canal in man. *Gut* 1968; **9**: 457–60.
15 Duthie GS, Miller R, Bartolo DCC. Internal anal sphincter electromyographic frequency is related to anal canal resting pressure. Both are reduced in idiopathic faecal incontinence. *Gut* 1990; **31**: A619.
16 Lubowski DZ, Nicholls RJ, Burleigh DE, Swash M. Internal anal sphincter in neurogenic fecal incontinence. *Gastroenterology* 1988; **95**: 997–1002.
17 Law PJ, Bartram CI. Anal endosonography – technique and normal anatomy. *Gastrointest Radiol* 1989; **14**: 349–53.
18 Law PJ, Kamm MA, Bartram CI. Anal sphincter imaging in faecal incontinence using endosonography – demonstration of unsuspected defects. *Gut* 1990; **31**: A605.
19 Swash M, Gray A, Lubowski DZ, Nicholls RJ. Ultrastructural changes in internal anal sphincter in neurogenic faecal incontinence. *Gut* 1988; **29**: 1692–8.
20 Speakman CTM, Hoyle CHV, Kamm MA, Henry MM, Nicholls RJ, Burnstock G. Adrenergic control of the internal anal sphincter is abnormal in patients with idiopathic faecal incontinence. *Br J Surg* 1990; **77**: 134–44.
21 Speakman CTM, Hoyle CHV, Kamm MA, Henry MM, Nicholls RJ, Burnstock G. Alpha adrenergic and intrinsic innervation of the internal anal sphincter is abnormal in idiopathic faecal incontinence. *Gut* 1990; **31**: A605.
22 Frencker B, Ihre T. Influence of autonomic nerves on the internal anal sphincter in man. *Gut* 1976; **17**: 306–12.
23 Penninckx F, Kerremans R, Beckers J. Pharmacological characteristics of the non-striated anorectal musculature in cats. *Gut* 1973; **14**: 393–8.
24 Meunier P, Mollard P. Control of the internal anal sphincter (manometric study with human subjects). *Pflugers Arch* 1977; **370**: 233–9.
25 Carlstedt A, Nordgren S, Fasth S, Appelgren L, Hulten L. Sympathetic nervous influence on the internal anal sphincter and rectum in man. *Int J Colorectal Dis* 1988; **3**: 90–5.
26 Bouvier M, Gonella J. Nervous control of the internal anal sphincter of the cat. *J Physiol* 1981; **310**: 457–69.