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Alimentary tract and pancreas

Combined sensory and motor deficit in primary neuropathic faecal incontinence

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SUMMARY Eleven patients with idiopathic faecal incontinence (IFI) and nine normal controls were studied with techniques of mucosal electrosensitivity and rectal distension for the quantitative assessment of anal and rectal sensation and with manometric and electromyographic tests for the assessment of anorectal motor function. The tests of motor function showed pelvic floor motor neuropathy in the patients with IFI, compared with controls, anal canal resting and voluntary contraction pressures were significantly ($p<0.05$, $p<0.002$) lower, pudendal nerve terminal motor latency and external anal sphincter fibre density were significantly ($p<0.05$, $p<0.05$) raised. The results of mucosal electrosensitivity (MES) disclosed a sensory deficit in the anal canal in patients with IFI, compared with controls, MES threshold was significantly ($p<0.002$) higher. Sensory thresholds to rectal distension were similar in the two groups. This study shows that sensory deficit of the anal canal occurs in combination with the motor neuropathy of the anal canal musculature in primary neuropathic faecal incontinence.

There has been renewed interest in the sensory aspects of anorectal function in health and disease. This is a region where two different types of innervation meet. The rectum and anal canal (above Hiltons' white line) are derived from endoderm and have an autonomic nerve supply through the inferior hypogastric plexus, which consists of sympathetic and parasympathetic fibres. Sensation is mediated by the parasympathetic fibres S2,3.[1] The lower part of the anal canal is derived from ectoderm and has a somatic nerve supply through the inferior rectal branch of the pudendal nerve S2,3,4 and the perineal branch of S4.[1] The pudendal and perineal nerves are mixed motor and sensory nerves.

Free nerve endings, organised endings and fast conducting myelinated fibres are present throughout the length of the anal canal including the region above Hiltons' line.[2] No free nerve endings or organised endings have been identified in the adjacent rectal mucosa, though many unmyelinated fibres related to the submucosal plexus have been found. The importance of anal canal sensation in maintaining continence has been much studied. Duthie and Bennett[3] have showed an anal sampling mechanism which allows discrimination of rectal contents. Read and Read[4] found that continence to saline infused into the rectum was not impaired in normal subjects after application of a local anaesthetic to the anal mucosa. A study of anorectal function after major resections of the sacrum with unilateral and bilateral loss of the sacral nerves demonstrated impaired discrimination of sensation in all patients but functional impairment of continence only in patients with bilateral nerve loss.[5] After excision of the rectum and coloanal anastomosis sphincter function and appreciation of impending evacuation remains unaffected, as do the normal inhibitory reflexes elicited by balloon distension.[6]

Incontinent patients often report that they have no sense of impending or actual incontinence. Methods for quantifying anal sensation by mucosal electrosensitivity (MES)[7] and rectal sensation by rectal distension, have been described.[8–11] The purpose of this study was to determine whether a sensory deficit exists in patients with primary neuropathic faecal incontinence.

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Methods

PATIENTS

The subjects were placed in the left lateral position for all of the tests.

TESTS OF MOTOR FUNCTION

Anal manometry, functional anal canal length, resting and maximum squeeze pressures

These were measured using a closed water filled system consisting of a 4 mm diameter microballoon mounted on a polyethylene tube 2 mm in diameter connected to a pressure transducer (Statham Instruments, Inc, Hato Ray, PR) and a pen recorder (Devices, London). The system was calibrated in a centimetre of water at ambient temperature and pressure. The balloon was lubricated with K-Y jelly (Johnson and Johnson Ltd, Slough) and gently inserted into the rectum; the pressure was continuously monitored as the balloon was removed in 1 cm stations. This allowed the resting pressure and functional length of the anal canal to be recorded. The balloon was reinserted into the anal canal to record the voluntary squeeze pressure.

Pudendal nerve terminal motor latency (PNTML)

This was measured using a specially constructed finger stall,[12] with stimulating electrodes mounted at the tip and recording electrodes mounted at the base connected to a standard electromyographic recording machine (MS6, Medelec, Woking, Surrey). The stimulator was inserted into the rectum and the pudendal nerve on each side was stimulated with a supramaximal stimulus of 50 volts of 0·1 millisecond duration. The action potential of the external anal sphincter was recorded by the base electrodes of the stimulator allowing measurement of the latency of conduction.[13]

Single fibre electromyography

Using a sterilised fine needle electrode with recording surface of 25 μm diameter (Medelec SF25533031) inserted into the external sphincter through the perianal skin, serial recordings of 20 different single muscle fibre electrical potentials were made. Analysis of the tracings allowed the calculation of fibre density,[13,14] an index of motor unit clustering; a consequence of denervation and subsequent reinnervation.

TESTS OF SENSATION

Mucosal electrosensitivity (MES)

This was measured using a specially constructed probe with two platinum electrodes placed 1 cm apart towards the tip of a 10 FG polyvinyl catheter graduated in centimetres. Copper wires connected to the electrodes passed to a constant current generator (Department of medical physics BESA, UK) supplying a 0·1 msec square wave stimulus at a constant rate of 5 pulses per second. The probe was lubricated with K-Y jelly and inserted into the upper, middle and lower thirds of the anal canal as determined by functional anal canal length. The current across the electrodes was increased in increments of 0·1 mA, until a threshold of sensation was reported by the patient: usually as a burning or tingling sensation. Two further recordings were taken at each site and the lowest mA reading taken as the result.

Rectal sensation

This was tested by means of an adapted party balloon inserted into the rectum and inflated slowly in 5 ml increments with water at 37°C. Records were made of the volumes required to give (i) a constant sensation of fullness, (ii) the first sensation of desire to defecate; when inflation was continued until (iii) the maximum tolerated volume (usually associated with an irresistible urge to defecate) was reached.

Statistical analysis of the data was by the Mann-Whitney U-test.

PATIENTS

Twenty subjects have been studied using these techniques: 11 patients with idiopathic faecal incontinence (IFI) (eight women, mean age 56·2 (25–83) years) and nine normal controls (NC) (four women, mean age 56·6 (25–73) years). Patients with incontinence had no identifiable predisposing cause, all had normal neurological findings on clinical examination, normal sigmoidoscopy and proctoscopy and all were incontinent of solid stool. Of the eight women

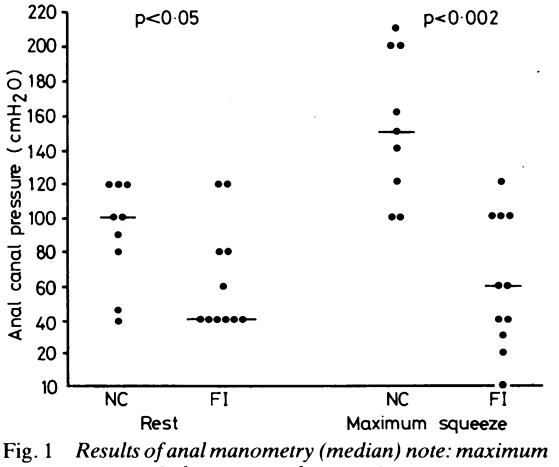

Fig. 1 *Results of anal manometry (median) note: maximum squeeze pressure is the pressure above resting pressure generated by voluntary contraction of the external anal sphincter.*

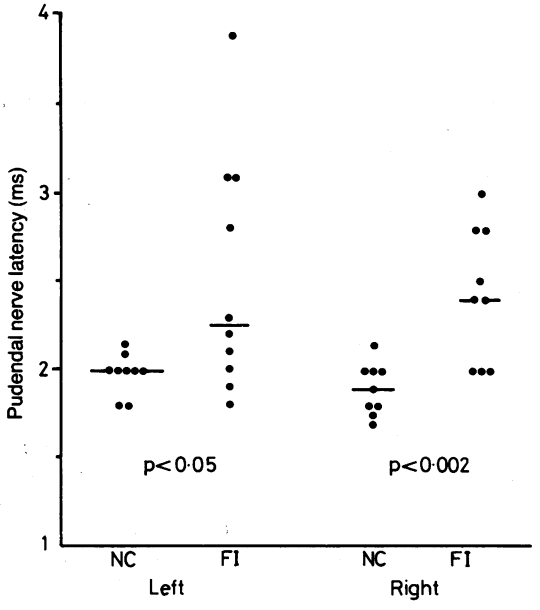

Fig. 2 *Results of PNTML (median).*

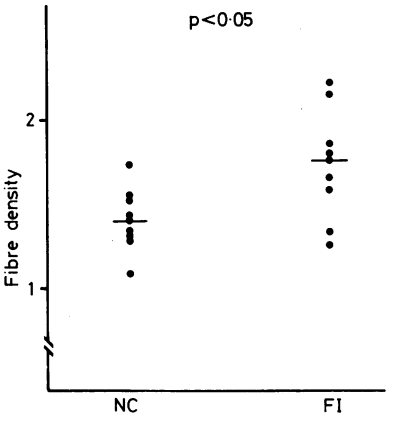

Fig. 3 *Results of FD (median).*

with IFI, three were nulliparous, four multiparous and in four their last delivery preceded the onset of incontinence by at least 20 years. Normal controls were volunteers with no history of diabetes mellitus or local anorectal disorder and all had normal sigmoidoscopic and proctoscopic findings. Two of the four women controls were multiparous and one nulliparous.

Informed consent was obtained in all cases. The study was approved by Brent Area Health Authority Ethical Committee.

Results (Table)

The functional length of the anal canal was significantly ($p<0·01$) shorter in the patients with IFI, than in controls. The tests of motor function confirmed a significant neuropathy in the incontinent patients. Resting pressure and maximum squeeze pressure were lower ($p<0·05$ and $p<0·002$) (Fig. 1). Pudendal nerve latency was increased, right ($p<0·002$) and left ($p<0·05$) (Fig. 2). Fibre density was also increased ($p<0·05$) (Fig. 3).

The thresholds to constant current stimulation in the upper, middle and lower thirds of the anal canal in the two groups were different. In normal subjects the threshold ranged from 1·7 to 7·2 mAmp. The results in the incontinent patients showed a significant increase in threshold at all levels of the anal canal,

Table *Summary of results*

	IFI Median (range)	*NC Median (range)*	*Mann-Witney U p value*
Anal canal length	3 (2–4) cm	4 (3–4)	(p<0·01)
Squeeze pressure	60 (0–120) cm H_2O	150 (100–210)	(p<0·002)
Resting pressure	40 (40–120) cm H_2O	100 (40–120)	(p<0·05)
Right PNL	2·4 (2·0–3·0) ms	2·0 (1·7–2·5)	(p<0·002)
Left PNL	2·25 (1·8–3·9) ms	2·0 (1·8–2·15)	(p<0·05)
Fibre density	1·73 (1·21–2·18)	1·38 (1·06–1·69)	(p<0·05)
MES			
Upper ⅓	18·4 (5·2–25) mA	5·3 (3·9–6·9)	(p<0·002)
Middle ⅓	10·1 (5·7–21·4) mA	3·7 (1·7–7·2)	(p<0·002)
Lower ⅓	8·9 (3·1–21·6) mA	4·4 (3·1–6·0)	(p<0·02)
Rectal distension			
Fullness	95 (50–230) ml H_2O	80 (20–200)	(NS)
Urge vol	125 (60–250) ml H_2O	120 (40–250)	(NS)
Max tol vol	150 (100–280) ml H_2O	150 (70–270)	(NS)

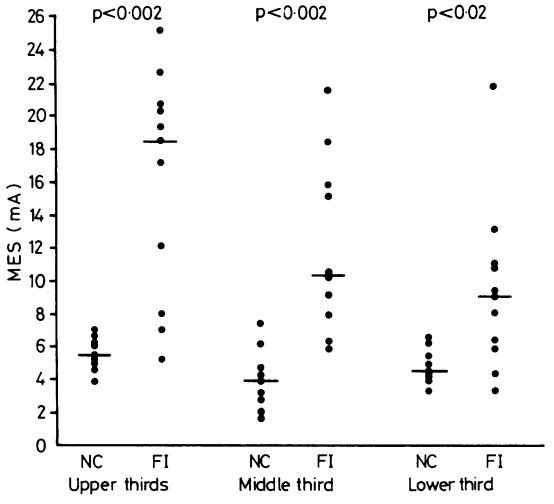

Fig. 4 *Results of MES (median).*

upper third ($p<0.002$), middle third ($p<0.002$) and lower third ($p<0.05$) (Fig. 4).

The results of rectal distension, at the three sensory levels, showed a wide range of values in normal controls and patients with IFI (Fig. 5).

Discussion

This is the first report of a coexisting sensory and motor deficit in a group of patients with IFI. Measurements of MES, in this study, suggested a sensory deficit in the anal canal at all levels in the patients with IFI. It is not known which nerve endings are stimulated by the MES technique, but it would appear to give a reliable quantitative measurement of the threshold to stimulation in the anal canal, which was described by all patients as a tingling, or burning sensation. On the other hand, there was a wide distribution of values obtained by rectal distension for the three sensory thresholds and no difference between the two groups could be detected. The finding of a sensory deficit in the anal canal with apparently normal rectal sensation in IFI may be because of the dual innervation of this region. The rectum is innervated by the autonomic nerves of the pelvic plexus which, in contrast with the somatic supply of the anal canal by the pudendal nerves and perineal branches of S4, are susceptible to different pathology; in addition they have a different anatomical position in relation to the pelvic floor. The wide range of results obtained in testing sensory thresholds to rectal distension can be explained by the absence of specialised nerve endings in rectal mucosa.[2] An alternative explanation is that the receptors which detect the stimulus lay outside the rectum, in the pelvic floor and adjacent structures.

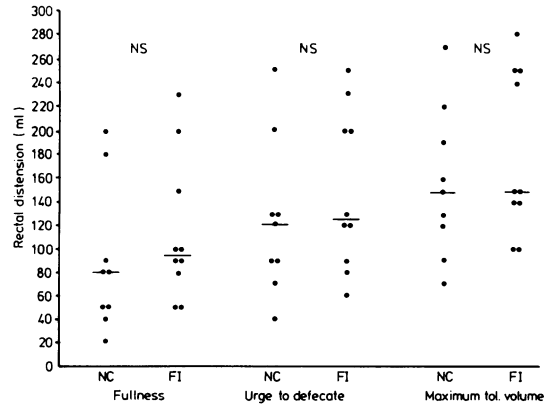

Fig. 5 *Results of rectal distension (median).*

The results of the motor tests in this study showed a weakness of the anal sphincter musculature and pudendal neuropathy as the primary cause, as shown by the increased PNTML and FD in the IFI group. This observation confirms previous findings which have established that pelvic floor neuropathy is the commonest cause of idiopathic faecal incontinence.[12]

The role of sensation in maintaining continence is still unclear, but this study shows that a significant sensory deficit in the anal canal is present in combination with a motor neuropathy in a group of patients with primary neuropathic faecal incontinence.

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References

1 Last RJ, *Anatomy: regional and applied.* London: Churchill Livingstone, 1978.
2 Duthie HL, Gairns FW. Sensory nerve-endings and sensation in the anal region of man. *Br J Surg* 1960; **47:** 585–94.
3 Duthie HL, Bennett RC. The relation of sensation in the anal canal to the functional anal sphincter: a possible factor in anal incontintence. *Gut* 1963; **4:** 179–82.
4 Read Maria G, Read NW. Role of anorectal sensation in preserving continence. *Gut* 1982; **23:** 345–7.
5 Gunterberg B, Kewenter J, Petersen I, Stener B. Anorectal function after major resections of the sacrum with bilateral or unilateral sacrifice of sacral nerves. *Br J Surg* 1976; **63:** 546–54.
6 Lane RHS, Parks AG. Function of the anal sphincters following colo-anal anastomosis. *Br J Surg* 1977; **64:** 596–9.
7 Roe AM, Bartolo DCC, Mortensen McC NJ. New

method of assessment of anal sensation in various anorectal disorders. *Br J Surg* 1986; **73:** 310–2.
8 Ihre T. Studies on anal function in continent and incontinent patients. *Scand J Gastroenterol* 1974; **9:** suppl 25.
9 Farthing MJG, Lennard-Jones JE. Sensibility of the rectum to distension and the anorectal distension reflex in ulcerative colitis. *Gut* 1978; **19:** 64–9.
10 Shouler P, Keighley MRB. Changes in colorectal function in severe idiopathic constipation. *Gastroenterology* 1986; **90:** 414–20.
11 Wald A, Tunuguntla AK. Anorectal sensorimotor dysfunction in faecal incontinence and diabetes mellitus. *N Engl J Med* 1984; **310:** 1282–7.
12 Kiff ES, Swash M. Slowed conduction in the pudendal nerves in idiopathic (neurogenic) faecal incontinence. *Br J Surg* 1984; **71:** 614–6.
13 Stalberg E, Thiele B. Motor unit fibre density in the extensor digitorum communis muscle. *J Neurol Neurosurg Psychiatry* 1975; **38:** 874–80.
14 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.