

Damage to the innervation of the voluntary anal and periurethral sphincter musculature in incontinence: an electrophysiological study

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SUMMARY In 40 women with idiopathic (neurogenic) faecal incontinence, 20 of whom also had stress urinary incontinence, single fibre EMG studies showed an increased fibre density in the external anal sphincter muscle. All these patients showed excessive descent of the pelvic floor on straining. The mean terminal motor latencies in the pudendal and perineal nerves, measured by a digitally-directed intrarectal stimulating technique, were increased when compared with 20 control subjects ($p < 0.01$). The perineal nerve terminal motor latency was more markedly increased in the 20 patients with double incontinence than in those with faecal incontinence alone ($p < 0.01$). These results provide direct electrophysiological evidence of damage to the innervation of the pelvic floor musculature in idiopathic faecal and double incontinence, and imply that idiopathic stress urinary incontinence may have a similar cause.

Histological and electrophysiological studies have revealed denervation of the pelvic floor musculature in 75% of patients with idiopathic (neurogenic) faecal incontinence.¹⁻³ Kiff and Swash have provided direct evidence of damage to the innervation of the perianal musculature, consisting of an increase in the terminal motor latency in the pudendal nerves measured by recording the compound muscle action potential in the external anal sphincter muscle after stimulating the pudendal nerves at the level of the ischial spines.⁴ In addition to supplying the external anal sphincter muscle, the pudendal nerves via their perineal branches also innervate the periurethral striated sphincter musculature.⁵ About 12% of our patients with neurogenic faecal incontinence also suffer from urinary incontinence of stress type.¹ These observations have therefore suggested to us that denervation of the periurethral striated musculature may be a factor in some patients with urinary incontinence, perhaps particularly in women in whom urinary incontinence of gradual onset in middle life is associated with perineal descent, and in those who have experienced a difficult labour. This suggestion is consistent with the clinical finding

of weakness of the anterior pelvic floor musculature in idiopathic urinary incontinence.⁶ We have therefore investigated the innervation of the voluntary anal and urinary sphincter muscle in incontinence and in normal subjects, using new electrophysiological techniques.

Patients

Twenty patients with faecal incontinence aged 22-75 years (mean 52 years \pm 18 (SD)) and twenty with faecal and urinary (double) incontinence aged 22-80 years (mean 54 \pm 16 (SD)) were studied; all 40 patients were women. Patients with sphincter damage due to anorectal surgery, direct obstetric trauma, or other injury were excluded. None of these patients suffered from diabetes mellitus or neurological disease. Twenty additional women, aged 20-60 years (mean 34 years \pm 10 (SD)) attending for follow up after treatment of colonic polyps or minor anorectal conditions, all with a normal defaecatory pattern, were used as controls.

Patients with faecal incontinence

Twelve of these 20 patients were incontinent of solid stool, the remaining eight being incontinent of liquid stool only. None were incontinent of urine. The average duration of faecal incontinence was 4.5 years (range 0.5-15 years). Seven patients gave histories of defaecation straining and constipation. The average duration of defaecation straining was 16.6 years (range 1-20 years). Seven patients had a complete rectal prolapse.

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Fourteen patients were parous (mean parity 1.9, range 1–4). Nine patients reported a difficult obstetric history; two had large babies, two prolonged labour, two forceps deliveries, one breech delivery, one caesarian section for failure to progress and two patients had perineal tears not involving the external anal sphincter muscle. In the nine women who reported a difficult delivery the mean length of time from first delivery to the onset of faecal incontinence was 25.3 years (range 6–46 years). Two patients reported the onset of faecal incontinence immediately after their first deliveries. Neither of these patients had evidence of direct sphincter damage. Five patients had undergone hysterectomy, four via an abdominal procedure.

On examination perineal descent, to an abnormal degree,⁷ was present in 11 patients. The anal reflex was absent in 12 patients.

Patients with double incontinence

Seventeen of the 20 patients were incontinent of solid stool and three were incontinent of liquid stool and flatus. The average duration of faecal incontinence was 7 years (range 0.5–34 years), and of stress urinary incontinence was 13 years (range 2–40 years). Eight patients gave a history of defaecation straining and constipation. The average length of history of straining was 14 years (range 2–30 years). Three patients had a complete rectal prolapse.

Fifteen patients were parous (average parity 2.3 years, range 1–14 years). Six patients reported difficult deliveries; two prolonged labour, three forceps deliveries, four breech deliveries, one caesarian section for failure to progress and two perineal tears without external anal sphincter damage.

The mean time interval between the first delivery to the onset of faecal incontinence was 27 years (range 4.5–40 years); and of urinary incontinence 9 years (range 2–40 years). Five patients reported the onset of faecal incontinence immediately after delivery and two patients reported urinary incontinence immediately after delivery. The average duration of urinary incontinence was 13 years (range 2–40 years). Eleven patients in this group had undergone hysterectomy; eight via an abdominal procedure.

On examination 12 patients had obvious perineal descent on straining.⁷ In eleven patients the anal reflex was absent.

Methods

Ano-rectal manometry

Resting tone and maximal voluntary contraction in the anal canal sphincter zone were measured using standard techniques.³

Single fibre electromyography (EMG) of the external anal sphincter

Single fibre EMG is an established technique in the investigation of patients with neuromuscular disease.⁸ This technique enables recordings to be made from several individual muscle fibres supplied by branches of the same motor axon in a circumscribed area of the external anal sphincter muscle. In our studies of the external anal sphincter a standard Medelec SF25 single fibre EMG needle electrode was used in conjunction with the Medelec MS6 EMG apparatus. The normal basal activity of the external anal

sphincter was increased by applying 150 g of traction to a 30 ml volume filled Miller-Abbott balloon placed within the lower rectum.² The mean number of muscle fibre action potentials recorded in 20 different sites, through four skin insertions is called the *Fibre density* and represents the mean number of muscle fibres innervated by one motor unit within the uptake area of the electrode. In neurogenic disorders the fibre density is raised, indicating reinnervation by collateral axonal sprouting.

Transrectal digitally directed pudendal nerve terminal motor latency measurement

This method was developed from the technique of electroejaculation described by Brindley for use in patients with impotence due to paraplegia.⁹ The device consists of a rubber finger stall having two metal stimulating electrodes at its tip, and two metal circular surface electrode plates for recording mounted 3 cm proximally at the base of the finger.⁴ Stimulation of the pudendal nerves on either side was performed using square wave supramaximal stimuli of 0.1 ms duration and about 50 volts. Two recordings of five consecutive traces at 1 ms intervals were made on both sides of the pelvis. The latency of the response was measured on the paper print out from the onset of the stimulus to the onset of the response.

Digitally directed perineal nerve terminal latency

The technique of transrectal digitally directed pudendal nerve stimulation⁴ has been modified to record the latency of the response of the periurethral striated musculature to pudendal nerve stimulation. A surface electrode mounted on a size 14 Foley catheter was inserted into the lower third of the urethra. The compound muscle action potential response recorded from the periurethral sphincter musculature, after stimulation of the pudendal (perineal branch) nerve per-rectally, was displayed on the Medelec MS6 apparatus. In order to study the responses of the voluntary anal and urethral sphincter musculature the patient was placed in the left lateral position. A ground electrode was strapped to the upper thigh.

Measurement of perineal position and descent

The method employed was described and standardised by Henry and colleagues.⁷ The device used consists of a graduated latex cylinder capable of free movement within a steel frame. It possesses two vertical limbs which can be moved across a horizontal rack so that their bases rest on the patients' ischial tuberosities. With the patient in the left lateral position, the central latex cylinder is brought into contact with the perineal skin. A centimetre scale on the cylinder allows measurements of the perineal plane in relation to the ischial tuberosities, first at rest and then when the patient bears down as in defaecation. It is therefore possible to determine both the position of the perineal plane in relation to the plane of the ischial tuberosities, and the total excursion occurring during perineal descent.

Results

Ano-rectal manometry

The resting tone in the patients with faecal inconti-

nence ranged from 5 to 76 cm of water (mean 32.3 cm of water \pm 17.9 (SD)) and in the patients with double incontinence resting tone ranged from 10 to 100 cm of water (mean 38.1 cm of water \pm 33.4 (SD)). Maximal voluntary contraction pressures ranged from 15 to 140 cm of water (mean 48.0 cm of water \pm 31.9 (SD)) in faecally incontinent patients and 10 to 150 cm of water (mean 39.2 cm of water \pm 33.3 (SD)) in patients with double incontinence; normal range 80 to 200 cm of water (mean 120 cm of water).³ In the control subjects these values fell within the normal ranges.

Single-fibre EMG of the external anal sphincter muscle

In the patients with faecal incontinence the mean fibre density in the external anal sphincter muscle was 2.1 \pm 0.3 (SD); normal 1.5 \pm 0.16 (SD).² In the patients with double incontinence the mean fibre density in the external anal sphincter muscle was 2.0 \pm 0.3 (SD).

Digitally directed pudendal and perineal nerve terminal motor latencies

The mean (right and left nerves) pudendal and perineal nerve terminal motor latencies in the patients with faecal incontinence were 2.6 ms \pm 0.8 (SD) and 3.2 ms \pm 0.6 (SD) respectively. In the control subjects the pudendal and perineal nerve

terminal latencies were 1.9 ms \pm 0.2 (SD) and 2.4 ms \pm 0.2 (SD) respectively (fig 1). Using the Wilcoxon Rank Sum Test both pudendal and perineal nerve terminal latencies were significantly increased ($p < 0.01$) when compared to the control values.

The mean (right and left nerves) pudendal and perineal nerve terminal motor latencies in the patients with double incontinence were 2.7 ms \pm 0.5 (SD) and 4.3 ms \pm 1.6 (SD) respectively (fig 1). Both the pudendal ($p < 0.01$) and perineal ($p < 0.01$) nerve terminal motor latencies were significantly greater than the control values. The perineal nerve terminal motor latency was also increased ($p < 0.01$) in the patients with double incontinence when compared to the patients with faecal incontinence alone, but there was no significant difference in pudendal nerve terminal motor latencies in these two groups of patients.

The mean differences between the perineal and pudendal terminal nerve motor latencies in the patients with faecal and double incontinence were; 0.7 ms \pm 0.5 (SD) and 1.6 ms \pm 1.3 (SD) respectively. In the normal subjects the mean difference between the perineal and pudendal nerve terminal motor latencies was 0.5 ms \pm 0.2 (SD). If the difference between perineal and pudendal nerve terminal motor latencies is expressed as a percentage of the total perineal nerve terminal motor latency the values are; 20.8% \pm 11.8 (SD), 33.2% \pm 14.7 (SD) and 18.8% \pm 5.5 (SD) for the patients with faecal incontinence, double incontinence and normal subjects respectively.

Perineal descent

The mean distance of the perineal plane above the plane of the ischial tuberosities at rest in the women with faecal incontinence was 1.2 cm \pm 0.5 (SD) (range 0.25 to 2.25 cm) and in those with double incontinence was 1.0 cm \pm 0.4 (SD) (range 0.5 to 1.5 cm). The mean distance on straining below the plane of the ischial tuberosities in the faecal incontinent group was 1.2 cm \pm 0.4 (SD) (range 1 to 2.25 cm) and in the double incontinent group was 1.4 cm \pm 0.8 (SD) (range 0.25 to 3 cm). The normal values in our laboratory are; at rest 2.5 cm \pm 0.6 (SD) above the plane of the ischial tuberosities and on straining 0.9 cm \pm 1.0 (SD) above the plane of the ischial tuberosities.⁷

Discussion

The clinical, anorectal manometric and single fibre EMG abnormalities of the external anal sphincter found in all 40 patients with faecal incontinence are similar to those previously reported^{2,3} in the syndrome of idiopathic neurogenic faecal inconti-

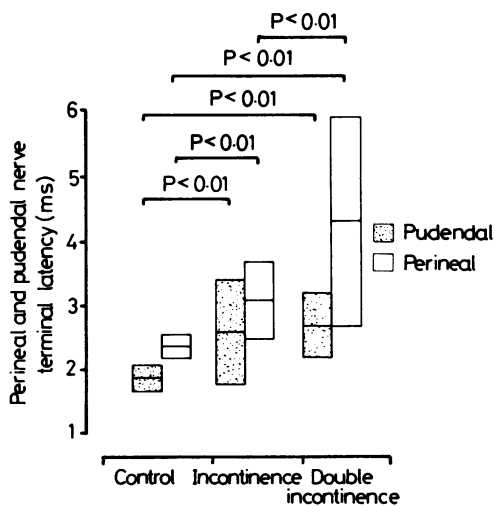


Fig 1 Differences in the mean pudendal (anal sphincter) and perineal (urethral sphincter) nerve terminal motor latencies for incontinent and doubly incontinent patients represented as one standard deviation either side of the mean.

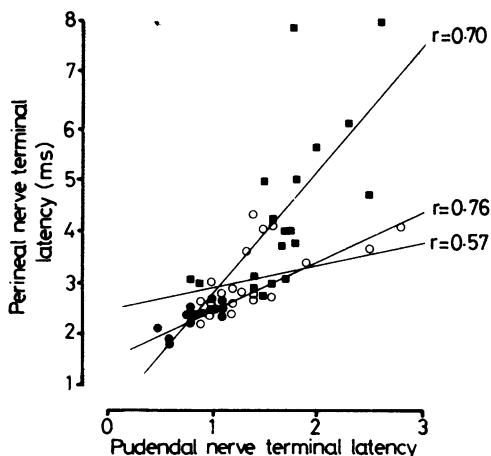


Fig 2 Linear regression lines and correlation coefficients for perineal and pudendal nerve terminal motor latencies in normal subjects ● faecal incontinent ○, and doubly incontinent ■, patients.

nence¹⁰ indicating that these patients conform to the pattern of abnormality found in that form of anorectal incontinence we have found to be due to denervation of the pelvic floor musculature.¹⁰ The single fibre EMG fibre density in the external anal sphincter was similarly raised in the patients with double incontinence and in those with faecal incontinence alone.

The periurethral striated musculature plays an important role in the maintenance of urinary continence.¹¹ This musculature is innervated by the pudendal nerves. The urethra itself has an additional intrinsic (intramural) component of striated muscle which is supplied by somatic efferent fibres travelling in the pelvic nerves.¹²

This study has revealed significant increases in the distal motor latencies in the pudendal and perineal nerves in patients with faecal incontinence and double incontinence compared with control subjects. The perineal nerve terminal motor latencies were significantly greater in patients with double incontinence than in those with faecal incontinence alone. The correlation coefficients for the pudendal and perineal nerve terminal motor latencies were 0.7 in the doubly incontinent patients, 0.5 in those with incontinence alone, and 0.8 in the normal subjects (fig 2). Thus in those with double incontinence there was a closer correlation than in those with faecal incontinence alone, both nerves being damaged in the former group and only the branch of the pudendal nerve supplying the external anal sphincter muscle in the latter. Likewise a close correlation

existed between pudendal and perineal nerve terminal motor latencies in the normal subjects. These findings, indicating slowing of motor nerve conduction in the distal parts of the pudendal and perineal nerves provides direct confirmation of our hypothesis that idiopathic faecal incontinence is due to damage to the nerve supply to the muscles of the pelvic floor,¹³ and suggests that the urinary incontinence that accompanies this disorder may have a similar cause. Since this stress incontinence is similar in all clinical respects to the common form of stress urinary incontinence unassociated with faecal incontinence it is implied that this may also be associated with partial denervation of the voluntary striated musculature of the periurethral sphincter muscle, and of the anterior pelvic floor.

Twenty three of our 40 patients with faecal incontinence had perineal descent.⁷ This is consistent with the hypothesis that in idiopathic (neurogenic) faecal incontinence damage to the innervation of the pelvic floor may occur from stretch-induced injury.^{7,10} Injuries to the nerve supply of the pelvic floor musculature may also be associated with trauma during child birth (15 patients), and with excessive and prolonged defaecation straining (15 patients). In patients with double incontinence, in whom the percentage difference between the perineal and pudendal nerve terminal motor latencies fell within the normal range, and yet both latencies were increased (5 of 20), it is suggested that the nerve lesion was proximal to the bifurcation of the pudendal nerve into branches innervating the external anal sphincter and periurethral sphincter muscles.¹¹ Previous work¹⁴ using transcutaneous spinal stimulation has suggested that this nerve lesion is situated peripherally and that it also involves the non-pudendal somatic innervation of the pelvic floor derived from pelvic nerves.¹⁴ However, in the majority of patients (15 of 20) in the double incontinence group the perineal nerve terminal motor latency was greatly increased. In the patients with faecal incontinence alone the perineal branch of the pudendal nerve was relatively spared.

These results suggest a new approach to the investigation of faecal and double incontinence in women, using direct electrophysiological techniques for the measurement of motor nerve terminal latencies to the anterior pelvic floor musculature. Further, this approach has implications for understanding idiopathic stress urinary incontinence.

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