Use of the pudendo-anal reflex in the treatment of neurogenic faecal incontinence

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Abstract

An electrical stimulator has been devised to treat neurogenic faecal incontinence caused by pudendal nerve neuropathy and works on the basis of repeated stimulation of the pudendo-anal reflex arc. Although conduction in the pudendo-anal reflex arc may be prolonged, and is so in neurogenic faecal incontinence, it must be shown to be present before the method can be used. This stimulation results in an immediate rise in the pressure in the anal canal and a significant increase in the electromyographic activity of the external anal sphincter. Maintenance of the stimulus over a two month period raised the mean resting pressure significantly in the anal canal and increased the reflex and voluntary responses of the external anal sphincter to coughing and squeezing actions respectively. The length of the sphincter was not affected. There was widening of the mean motor unit potential duration, though this was not significant. The resting electromyogram was enhanced after the course of treatment, indicating greater spontaneous activity in the external sphincter. The changes led to seven of the eight patients studied becoming continent at the end of the treatment.

Neurogenic faecal incontinence is an increasing problem in an ageing population. Not all patients are suitable for a post-anal repair, which is the mainstay of surgical treatment.¹ Various electrical stimulators have been designed in the past with the aim of returning function to the external sphincter and pelvic floor. These make use of a surface anal plug electrode² or are implanted with a radiofrequency link outside the body.³ Although some have been successful at first, they have not found continued use, mainly because of wire breakages in the implants and anal pain or mucosal ulceration with the anal plugs.⁴

The pudendo-anal reflex latency⁵ is a useful measurement in the electromyographic investigation of neurogenic faecal incontinence. A prolonged latency from the stimulus artefact to the sphincter response indicates, inter alia, the degree of neuropathy. A portable stimulator has been designed to provide repetitive stimulation of the pudendo-anal reflex which elicits contraction of both the external anal sphincter and the pelvic floor. The method attempts to restore continence by provoking contraction of these muscles selectively through the reflex. Manometric and electrophysiological responses in patients with neurogenic faecal incontinence were studied before and after pudendo-anal reflex stimulation treatment and any clinical change was noted.

Neurophysiological tests performed before and after treatment by the stimulator

	Pre-stimulation (mean (SEM))	Post-stimulation (mean (SEM))	p Value
PARL	55.9 (6.9) ms	65.9 (6.8) ms	p>0.05
PAR amp	37.5 (10.5) μ V	49.5 (9.2) μ V	p<0.01
MUPD	12.7 (3.3) ms	12.8 (3.4) ms	p>0.05
Electromyogram	11.6 (4.9) μ V	26.9 (7.3) μ V	p<0.01

PARL=pudendo-anal reflex latency; PAR amp=pudendo-anal response amplitude; MUPD=motor unit potential duration.

Patients

Eight women with a mean age of 47.5 (range 32-65) years took part in the study, for which ethical permission had been obtained from Lothian Health Board. Parity ranged from 1-5, with a mean of 3.1. All presented with faecal incontinence, were incapacitated socially, and had to wear incontinence pads. During the study each patient acted as her own control. All had an intact pudendo-anal reflex arc with a mean latency of 55.9 ms and a range of 47.2-69.4 ms (Table). The normal latency was mean (SD) of 39 (5.8) ms.⁶

Methods

All measurements were performed at the initial presentation and were repeated after completing an eight week trial of electrical stimulation.

MANOMETRY

A standard water filled microballoon system with external transducer measured the anal canal pressure by a 1 cm station pull through technique. The anal sphincter high pressure zone reflected the functional length of the anal sphincter, whose maximum resting pressure was recorded. The reflex contraction pressure of the external sphincter during a maximum cough was recorded at the point of maximum resting pressure, as was the maximum voluntary squeeze pressure of the sphincter.

Manometric recordings were also made of the inhibitory fall, as the recto-sphincteric reflex, in the resting pressure within the anal canal on rapidly distending the rectal ampulla with a balloon containing 50 ml of air. The anal sphincter pressure was also measured in response to dorso-genital nerve stimulation at the parameters to be used in the subsequent treatment.

ELECTROPHYSIOLOGY

An anal plug electrode was used with an electromyographic integrator to obtain amplitude measurements of the external anal sphincter resting electromyogram and the electromyogram

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gram was also recorded during dorsal nerve stimulation. The pudendo-anal reflex latency and the response amplitude were recorded after applying over one hundred consecutive synchronised impulses to the dorso-genital nerve using an electromyographic multi-function apparatus (Medelec MS 92a, Woking, Surrey, England). The mean motor unit potential duration of the external anal sphincter was calculated from the mean of 20 motor unit potential durations taken at four sites around the anal sphincter circumference.⁷ These were obtained with a concentric needle electrode, using the signal trigger and delay function of the recording equipment. The number of phases in each motor unit potential were counted and the fraction or overall percentage of polyphasic units was calculated for each subject. Each part of a motor unit which lay between two crossings of the base line, including the part of the potential between the onset and the first crossing, was termed a phase.⁸ A polyphasic unit has greater than four phases. Anal sphincter mapping was performed at the time of motor unit potential duration acquisition using a concentric needle electrode to show that the external anal sphincter ring was intact in all cases.

ELECTRICAL STIMULATOR

The portable stimulator, which has a rechargeable nickel cadmium battery power source, provided a train of square wave stimuli to the dorso-genital nerve with fixed frequency of 1 Hz and a duration of 0.1 ms (Fig 1). Saline-soaked felt electrodes were used to apply the skin stimulus in the mid-line at the base of the clitoris identical to that used for the pudendal reflex test. A sub-maximal tolerable stimulation voltage of mean (SD) 135 (15) v was used, which was two

Figure 1: The portable dorsal genital nerve stimulator with the felt stimulating electrodes in an insulating handpiece.

Figure 2: The length of the anal canal (in cm) before and after dorsal genital nerve stimulation, showing *p* value and SEM.

or three times the sensory threshold. The immediate effect of activating the stimulator was to cause a significant rise ($n=8$) in the external anal sphincter electromyographic activity from mean (SEM) 11.6 (1.7) μ V to 44.9 (1.9) μ V ($p<0.01$), with a corresponding significant rise in the anal canal pressure from mean (SEM) 49.1 (4.0) cm H₂O to 89 cm (10.4) cm H₂O ($p<0.01$). Treatment lasted for five minutes on three occasions per day for an eight week course and was self-administered.

STATISTICS

The statistical evaluation for the measurements of the high pressure zone, maximum resting pressure, maximum voluntary squeeze contraction, and cough reflex contraction pressure was done by a Student's *t* test using logarithmic conversion of the data of the paired observations before and after treatment. The remaining data were analysed by the Wilcoxon signed rank test for paired data.

Results

MANOMETRY

The anal canal high pressure zone, representing functional anal sphincter length (Fig 2), was increased from mean (SEM) 1.9 (0.2) cm to 2.6 (0.3) cm after the period of stimulation, but not significantly ($p>0.05$).

The patients with faecal incontinence had individual resting pressures below 60 cm H₂O, which represented their 'continence threshold.' In the anal canal this pressure was significantly increased from mean (SEM) 49.1 (4.0) cm H₂O

Figure 3: The maximum resting pressure recorded in the anal canal before and after the course of dorsal genital nerve stimulation, showing *p* value and SEM.

to 61.2 (4.5) cm H₂O (*p*<0.01) after the course of electrical stimulation (Fig 3).

The pressure elicited by the reflex contraction of the external anal sphincter in response to coughing, the cough reflex contraction pressure (Fig 4), was significantly increased from mean (SEM) 80.6 (8.2) cm H₂O to 106.1 (11.2) cm H₂O after the period of stimulation (*p*<0.01).

The maximum voluntary squeeze contraction of the external anal sphincter (Fig 5) was also increased significantly from mean (SEM) 112 (12.1) cm H₂O to 150 (17.3) cm H₂O after the period of stimulation (*p*<0.01) (Table). The anal canal pressure reduction which followed provoking an internal sphincter recto-sphincteric reflex was not significantly altered by the course of dorsal nerve stimulation (from mean (SEM) 43.8 (3.5) cm H₂O to 45.4 (3.2) cm H₂O (*p*>0.05)).

Figure 4: Maximum pressure recorded in the anal canal during the reflex response to a cough before and after the course of dorsal genital nerve stimulation, showing *p* value and SEM.

Figure 5: Maximum pressure recorded in the anal canal during a maximal voluntary squeeze contraction of the sphincter before and after the course of dorsal genital nerve stimulation, showing *p* value and SEM.

The recto-sphincteric reflex showed that the sphincter could be reflexly inhibited in these patients but this was not changed by treatment (mean (SEM) 43.8 (9.9) cm H₂O to 45.4 (9.1) cm H₂O after treatment).

ELECTROPHYSIOLOGY

The pudendo-anal reflex latencies with a mean (SEM) of 52.9 (2.4) ms were prolonged when compared with the normal range of 39 (0.94) ms,⁶ and were indicative of neurogenic injury (Table). The pudendo-anal reflex response amplitude (Table) was significantly increased from mean (SEM) 37.5 (3.7) μ V to 49.5 (3.3) μ V (*p*<0.1) after the period of stimulation.

The motor unit potential duration of the external anal sphincter was prolonged or widened with a mean (SEM) of 12.7 (1.2) ms when compared with the accepted normal range of 6.9 (0.2) ms (Table). There was also a high mean number of polyphasic motor unit potentials recorded from the external anal sphincter in the treated subjects with a mean (SEM) of 25.9 (9.4)%, the normal being 12%, both of which results are in keeping with reinnervation of the muscle after a previous neurogenic insult.⁷ The motor unit potential duration values were not affected by the course of electrical stimulation with a mean (SEM) of 12.7 (1.2) ms before and 12.8 (1.2) ms after stimulation (*p*>0.05) (Table).

The resting or basal integrated electromyogram of the external anal sphincter was increased significantly from mean (SEM) 11.6 (1.7) μ V to 26.9 (2.6) μ V after the course of stimulation. The external anal sphincter electromyographic response and the pressure responses to activation of the stimulator were also retested. There was a significant increase in the electromyographic response (*n*=8) from mean (SEM) 44.9 (4.9) μ V to 72.3 (5.4) μ V (*p*<0.01) with a concomitant significant increase in the pressure response from

mean (SEM) 89 (10.4) cm H₂O to 128 (13.3) cm H₂O ($p < 0.01$). Seven of the eight subjects had marked clinical improvement and became continent of both faeces and flatus after the course of stimulation. One patient was able to control faeces imperfectly, but not flatus. This patient is the one with the lowest values in Fig 3 before and after stimulation.

Discussion

The pelvic floor and external anal sphincter at rest are in a state of continuous tonic contraction, dependent on a spinal reflex arc completed through the conus medullaris by afferent and efferent pathways in the sacral 2, 3, and 4 segments of the spinal cord. The integrity of the arc can be tested by the pudendo-anal reflex, elicitation of which, in patients with faecal incontinence, produces a measurable contraction of pelvic floor and external sphincter but with an increased latency from stimulation to response. This has been used as an indicator of neurogenic damage. Traction injury to the pudendal and pelvic nerves is associated with dysfunction of the pelvic floor and external anal sphincter which can result in faecal incontinence.⁹ Injury to these nerves also occurs during excessive straining with perineal descent such as occurs during labour.¹⁰ Both types of injury tend to be incomplete, with preservation of the anatomical pathway sufficient for elicitation of the reflex and for using it for treatment. When there is a partial denervation the remaining nerve fibres attempt to reinnervate the muscle fibres by sprouting. Reinnervation of the pelvic floor can take up to two years to be complete depending on the site and degree of the nerve injury.

The women in this study all had reduced spontaneous activity in the external anal sphincter at rest, as was reflected in the low spontaneous integrated electromyographic activity. There was also a weak reflex pressure response to coughing and a reduced voluntary squeeze contraction of the external anal sphincter. This manometric picture coupled with the electromyographic evidence of prolonged pudendo-anal reflex latency, prolonged mean motor unit potential duration, and increased polyphasic potentials confirm the neurogenic nature of the external anal sphincter dysfunction. Where there is a normal recto-sphincteric reflex, rectal filling relaxes the internal anal sphincter. There is then little compensatory action of the external sphincter, which is demonstrably weaker in its barrier action as shown by the poor pressure responses to voluntary and stress reflex contractions of the striated muscle.

Treatments for neurogenic faecal incontinence reflect the spectrum of the condition, which ranges from occasional soiling to frank incontinence. Dietary changes and medication have been advocated to give some predictability of bowel habit.^{11,12} Pelvic floor exercises with physiotherapy are used for urinary stress incontinence but the results are variable.^{13,14} Voluntary sphincter responses can be improved by training devices, and biofeedback has been claimed to be effective.¹⁵⁻¹⁷

The surgical procedure of post-anal repair was devised by Parks,¹ who reported results of 72% fully continent and 12% continent for solid stool,¹⁸ although Keighley¹⁹ reported a lower figure for full continence of 63% with a further 21% continent for solid stool. The use of electrical stimulation for diagnosis in patients with denervated muscle has been practised for over a hundred years.²⁰ The application of Faradic stimulation for the therapy of the anal sphincter has been regarded as unpleasant and without much effect,³ and Caldwell²¹ designed an implantable device for anal sphincter stimulation but with the serious technical problem of cable breakage.

The present method of stimulating the pelvic floor and external anal sphincter makes use of the pudendo-anal reflex and has followed from the observation that the external sphincter contracts during the recording of the reflex. The reflex shows no signs of habituation²² over several minutes of stimulation. A stimulator was therefore designed to produce trains of stimuli similar to those used in the test situation.

All the women in the study were shown to have neurogenic faecal incontinence due to pudendal nerve neuropathy and had a prolonged but intact pudendo-anal reflex before the period of electrical stimulation of the pudendo-anal reflex. After eight weeks of treatment seven out of eight subjects became fully continent of faeces and flatus while one remained incontinent of flatus only. There was no change in the pudendal reflex latency or in the length of the sphincter. The maximum resting anal canal pressure was measured above the 60 cm H₂O pressure which was the 'continence threshold' for the eight patients and the 'protective' cough and squeeze pressures associated with the external sphincter activity were significantly improved.

The improvement in sphincter function is reflected in the enhanced resting electromyogram, which should help raise the spontaneous activity to that in a normal external anal sphincter. The effectiveness of the return of sphincter function needs explanation. It may have resulted from hypertrophy of the remaining innervated muscle fibres giving the sphincter an enhanced activity and thus perhaps making the subjects more aware of its contraction. There was widening of the motor unit potential duration in some patients but this was not significant, indicating that further or reinnervation of the sphincter is an unlikely mechanism. Loss of tone in the pelvic floor could perpetuate the lack of spontaneous activity in the spinal reflex arc since afferent input to the sacral cord is essential for its activity to persist. Simple tightening of the pubo-rectalis portion of the pelvic floor in a post-anal repair, for example, would not persist unless the muscle could actively maintain this position afterwards and electromyographic studies show increased resting activity after the repair in keeping with a reactivation of the pudendo-anal reflex arc. The raised electromyographic and pressure levels during the activation of the stimulator at the end of treatment showed how far a two months' course of treatment can build up these related effects.

In neurogenic faecal incontinence, the

pudendo-anal reflex is almost always present though prolonged, and its presence must be confirmed before an electrical sphincter stimulator dependent on it is used. The evidence suggests that this reflex can be harnessed by repetitive electrical stimulation to aid the restoration of the control of defaecation. The action is likely to be due to stimulation of the external anal sphincter rather than the internal sphincter. The mean basal pressure in the anal canal, which measures both sphincters, had risen but the contribution from the internal anal sphincter to the increased pressure readings had probably not changed because the values obtained on inhibiting it in the recto-sphincteric reflex were not increased. Therefore, it can be deduced that the pressure effect is predominantly on the external sphincter, perhaps accompanied by one on the pelvic floor.

This type of stimulation differs from Faradism, which has been abandoned because it is short-lived and its effects were achieved through the stimulation of local nerve endings only. Studies are proceeding to ascertain the contribution of the present method of pelvic floor stimulation in promoting clinical effects such as the duration of continence and its associated pressure changes, including whether reflex stimulation can be applied to pudendal neuropathy of varying degrees of severity.

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