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# Electrophysiological observations on the human pudendo-anal reflex

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SUMMARY A reproducible electrophysiological technique is described to determine the latency of reflex contraction of the external anal sphincter in response to stimulation of the dorsal genital nerve: the pudendo-anal reflex. This was studied in 38 asymptomatic control subjects and 20 women with neurogenic faecal incontinence, supplemented by determination of the mean motor unit potential duration (MUPD) of the external anal sphincter and anorectal manometry. The reflex latency in the control group was 38·5 ± 5·8 (SD) ms and appeared to be independent of age or sex. Three patients with faecal incontinence had absent reflexes; the remainder showed significant prolongation of latency (56 ± 12·2 SD ms) and diminution of amplitude. MUPD was prolonged in incontinence and showed significant correlation with the corresponding reflex latency determination ($\tau = 0{\cdot}56$, $p < 0{\cdot}001$). The latency of this polysynaptic spinal reflex hence provides a reliable index of neuropathy of the external anal sphincter.

There has recently been much interest and controversy about the electrically evoked reflex activity of the external anal sphincter, particularly in relation to faecal incontinence of neurogenic origin.[1–7] Swash *et al* suggested that the latency of the classical anal reflex measured electrophysiologically was significantly increased in idiopathic faecal incontinence.[2 3 8] However, studies by other workers did not confirm the usefulness of the latency of this reflex as an index of pelvic floor neuropathy,[6 7] and raised doubts about the interpretation of the earlier latency measurements. It has since become clear that electrical stimulation of the perianal skin results in direct stimulation of the terminal innervation of the external anal sphincter. This produces the "early" or short-latency responses[4–7 9] that had previously been erroneously interpreted as spinal cord reflexes.[2 3 8] These inconsistences have limited the usefulness of the classical anal reflex for studying the clinical neurophysiology of the pelvic floor.

Electrical stimulation of the dorsal nerve of the glans penis or clitoris evokes a reflex contraction of the external anal sphincter mediated via the sacral spinal cord segments 2, 3, and 4, a modification of the bulbocavernosus reflex described by Bors and Blinn in 1959[10] (fig 1). The availability of modern electrophysiological equipment has enabled more precise studies on the latency of the pudendo-anal reflex.[11] We describe a reproducible method of measuring the latency of this reflex and investigate its variation with age and sex, and in neurogenic faecal incontinence.

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## Patients and methods

Approval for this investigation was obtained from the Ethical Committee of the North Lothian District, Lothian Health Board, Scotland, in June 1983. Informed consent was obtained from all subjects participating in the study. The control group consisted of 25 female and 13 male subjects (age range 23–75 years, mean 45 years). They were hospital patients who had been admitted for minor surgery outwith the alimentary tract and none had any anorectal symptoms. The group with neurogenic faecal incontinence comprised 20 women (age range 37–79 years, mean 59·9 years). The duration of anal incontinence ranged from 6 months to 10 years (mean 1·9 years). All subjects underwent anorectal manometry and measurement of the latency of the pudendo-anal reflex. Determination of the mean motor unit potential duration of the external anal sphincter was performed in all the incontinent patients and in 15 control subjects.

### *Manometry*

Patients were requested to empty their bowel on the morning

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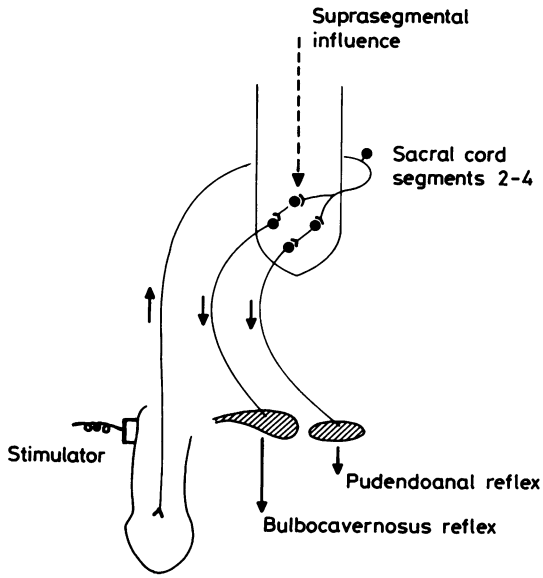


Fig 1 *Diagrammatic representation of the anatomical basis of the bulbocavernosus and pudendo-anal reflexes (modified from Ertekin and Reel[16]).*

of the study. The investigations were performed with the patient in the left lateral position. Basal and squeeze sphincter pressures and sphincter length were measured with a conventional water-filled microballoon and external transducer using a 0·5 cm station-pullthrough method.[12]

*Latency of the pudendo-anal reflex*

Figure 1 illustrates the anatomical basis of the pudendo-anal reflex.[11] It was elicited by electrical stimulation of the dorsal nerve of the glans or clitoris with a felt bipolar suface stimulating electrode (type LBS 53051, Medelec, UK). The reflex contraction of the external anal sphincter was recorded with a bipolar surface stainless-steel anal plug electrode (type 13K78, DISA, Copenhagen) using the Medelec MS92a evoked response unit. Electrode jelly was used to improve electrical contact between the plug electrode and the sphincter. A saline-soaked felt strap wrapped around the right thigh was used as the ground electrode. More than 100 square-wave stimuli (duration 0·1 ms, frequency 2 Hz) were applied and the digitally averaged response displayed on the oscilloscope at a sweep speed of 10 ms/cm with the gain at 10 or 20 $\mu$V/cm and filter settings of 2 Hz–10 kHz. The stimulation voltage was approximately three times the voltage at sensation threshold; typical threshold and stimulation voltages being 30 V and 90 V respectively. In no case was the stimulation reported by the patient to be painful. The procedure was repeated in each subject to ensure reproducibility. The latency of the pudendo-anal reflex was measured from the onset of the sweep (triggered by the stimulus) to the onset of the clearly defined reflex response at the external anal sphincter (fig 2).

*Determination of the mean motor unit potential duration (MUPD) of the external anal sphincter*

A modification of the method described by Bartolo *et al*[13] was used. A standard concentric needle EMG electrode (surface area 0·07 mm$^2$, type 13L49 DISA, Copenhagen) was inserted into the external anal sphincter without anaesthetic via a puncture site 1 cm lateral to the anal orifice to a depth of approximately 250 mm. This was connected via preamplifiers to an oscilloscope (Medelec MS92a, Woking, Surrey, UK). The tonic electrical activity of the sphincter was monitored using a time base of 10 ms/cm with the gain at 100 $\mu$V/cm and filter settings of 20 Hz–10 kHz. Single motor units firing at a steady rate were identified using the delay and trigger facilities incorporated in the apparatus. A

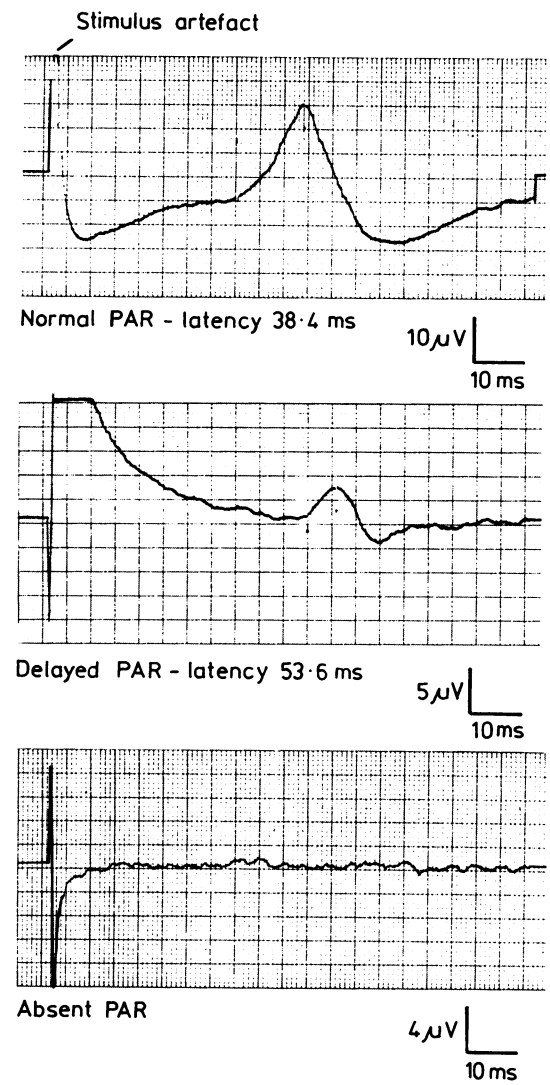


Fig 2 *Typical traces of the pudendo-anal reflex.*

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Table 1 *Anorectal manometry*

| *Parameter* | *Control (n = 25)* | *Faecal incontinence (n = 20)* | *Statistical significance* |
|---|---|---|---|
| Maximum resting pressure (cm $H_2O$) | 100 ± 28 | 60 ± 24·6 | $p < 0·01$ |
| Squeeze pressure (cm $H_2O$) | 167 ± 35 | 86 ± 32·6 | $p < 0·01$ |
| Physiological sphincter length (cm) | 3·2 ± 0·5 | 2·2 ± 0·9 | $p < 0·01$ |

All measurements Mean ± SD.

Table 2 *Normal range and variations with sex and age of the pudendo-anal reflex latency*

| *Subjects* | *Age (Range, mean ± SD, yr)* | *Latency of pudendo-anal reflex (Range, mean ± SD, ms)* | *Statistical significance of differences in latency* |
|---|---|---|---|
| 25 female, 13 male | 23–75 (45 ± 14·6) | 27·2–46·8 (38·5 ± 5·8) | — |
| 10 female (age-matched) | 28–57 (46·4 ± 12·6) | 33–46 (40·7 ± 4·4) | $p > 0·1$ |
| 10 male | 28–59 (47 ± 12·7) | 27·2–46·8 (38·5 ± 6·6) | |
| 10 female ("young") | 23–49 (33·6 ± 9·2) | 33–44·8 (39·6 ± 4·3) | $p > 0·1$ |
| 10 female ("old") | 55–75 (62 ± 6) | 29·2–46 (36·6 ± 6·6) | |

saline-soaked felt strap wrapped around the right thigh was used as the ground electrode. Approximately 100 consecutive action potentials of the same motor unit were digitally averaged on one channel of the oscilloscope and the process repeated on the second channel. When two identical traces were obtained on both channels, the action potential duration for that motor unit was measured from the first deflection from the baseline to the return of the action potential to the baseline. Stable late components were thus easily identified.[13] Permanent recordings were obtained of at least 20 action potentials from the external anal sphincter representing approximately ten recordings from each side of the sphincter. This was made possible by minor movements of the tip of the needle electrode in the sphincter. The arithmetic mean of the 20 recorded potential durations was calculated and represented the mean motor unit potential duration for that sphincter. This was used as an index of neuropathy.[14]

*Statistics*

Non-parametric methods were used. Differences in the manometric and electrophysiological measurements between the various groups were analysed by the Wilcoxon rank sum test. Correlation between the latency of the pudendo-anal reflex and mean motor unit potential duration of the external sphincter was performed by Kendall's rank correlation.

## Results

*Manometry*

Table 1 compares the manometric parameters measured in the two groups. There was a significant reduction in the physiological sphincter length, maximum resting pressure and the squeeze pressure of the external anal sphincter in the incontinent group.

*Electrophysiology*

Reproducible reflex responses were confirmed in all subjects. Reversal of the polarity of the stimulating electrode simply resulted in reversal of the stimulus artefact without altering the shape or latency of the evoked response. "Bifid" responses were observed in some patients and controls, that is, responses with late components. In these cases the latency of the pudendo-anal reflex was taken from the onset of the first response because this represents the shortest measurement. Table 2 lists the latencies of the pudendo-anal reflex in the control group of 38 subjects. These were further subdivided into age-matched male and female groups (n = 10) and "young" and "old" female groups (n = 10). No differences in the latency of the reflex were demonstrable with either sex or age. Table 3 compares the electrophysiological data between the control and incontinent groups. There were no differences in the voltage at sensation threshold and that used for maximal stimulation. However, the latency of the pudendo-anal reflex was significantly prolonged and its amplitude reduced in the incontinent group. In three of these patients the reflex could not be elicited despite several attempts. No differences were apparent in the duration of the reflexly evoked anal sphincter response between the

Table 3 *Electrophysiological anal sphincter data*

| *Parameter* | *Control* | *Faecal incontinence* | *Statistical significance* |
|---|---|---|---|
| | (n = 25) | (n = 20) | |
| Stimulation threshold (Volts) | 35·2 ± 14·5 | 31 ± 8·9 | $p > 0·1$ |
| Stimulation voltage (Volts) | 112 ± 33 | 96·8 ± 20·1 | $p > 0·05$ |
| Latency of pudendo-anal reflex (ms) | 39 ± 5·8 | 56 ± 12·2 (3 absent) | $p < 0·01$ |
| Amplitude of pudendo-anal reflex ($\mu$V) | 4·5 ± 3·4 | 1·95 ± 1·9 | $p < 0·01$ |
| Duration of pudendo-anal reflex (ms) | 16·9 ± 7 | 15·4 ± 5·4 | $p > 0·1$ |
| | (n = 15) | (n = 20) | |
| Mean motor unit potential duration of external sphincter (ms) | 8·66 ± 1·39 | 12·23 ± 1·83 | $p < 0·01$ |

All measurements Mean ± SD.

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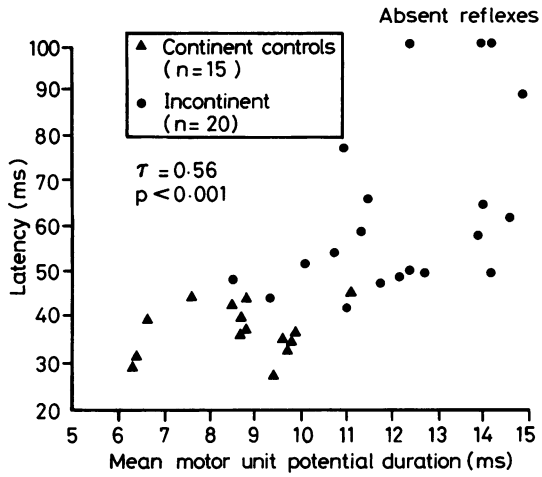


Fig 3 *Scattergram showing correlation between the mean motor unit potential duration of the external anal sphincter and the corresponding latency of the pudendo-anal reflex.*

two groups. The mean MUPD of the external anal sphincter was significantly increased in the incontinent patients. Figure 3 illustrates the high correlation between the latency of the pudendo-anal reflex and the mean MUPD of the respective external anal sphincter ($\tau = 0{\cdot}56$, $p < 0{\cdot}001$; for linear correlation $r = 0{\cdot}69$, $p < 0{\cdot}001$). Taking only the incontinent patients, $\tau = 0{\cdot}4$, $p < 0{\cdot}02$ (for linear correlation, $r = 0{\cdot}5$, $p < 0{\cdot}05$).

## Discussion

Reflexes involving the perineal muscles have stimulated much interest in the neurological evaluation of the conus medullaris and its afferent and efferent connections. These studies are considered to be of particular value in disorders of the genito-urinary system and the pelvic floor musculature.[1-11 15-22]

Since the description of the classical anal reflex by Rossolimo in 1891 using mechanical stimulation of the perianal skin,[23] the reflex has been extensively investigated by more sophisticated electrophysiological methods.[1-9 21] Henry and Swash described a latency for this reflex of 8·3 ± 1·7 (SD) ms in 13 normal subjects[2] and suggested that it was prolonged in faecal incontinence and rectal relapse.[2 3 8] The presence of these short-latency responses has been observed by other workers who also noted later responses of longer duration.[4 9 20] The early reactions have a uniform electrical pattern and show no sign of fatigue. They are not abolished by spinal anaesthesia[4 7] and their latencies are too short for a spinal reflex.[4 5 24] They have therefore been attributed to direct activation of the terminal innervation of the anal sphincter.[4-7 9] Some of the intermediate responses may be due to antidromic stimulation with interaction between neighbouring α-motoneurons in Onuf's nucleus[25] hence resulting in "oligosynaptic" latencies.[6] The "classical" polysynaptic anal reflex is now recognised to have a latency of 50 ± 10·5 (SD) ms.[1] These variable factors have made the precise determination of the latency of the anal reflex difficult and its interpretation controversial.[26]

Reflex reaction of the external anal sphincter can also be provoked by stimulating the glans penis, clitoris, the mucosa and the rectum, urethra and bladder and even the posterior tibial nerve.[27] Stimulation of the dorsal nerve of the penis or clitoris evokes a reflex contraction of the bulbocavernosus muscle: the classical bulbocavernosus reflex[10 15]; (fig 1). Bors and Blinn also described a simultaneous reflex contraction of the anal sphincter in response to this stimulus.[10] This was variously termed the sacral evoked potential,[19] sacral evoked response,[28] sacral reflex,[22 29] pudendal evoked response[20] and pudendal sexual reflex.[30] However, at a recent meeting of the Physiological Society the more accurate terminology of "pudendo-anal reflex" was adopted.[11]

The latency of this response appears to be dependent on the intensity of stimulation and it shows little or no signs of habituation.[26] Hence, the most appropriate electrophysiological method for investigating this reflex is to use a train of high-voltage stimulus impulses and to average digitally the anal sphincter response[26 31 32] as described in this study. The averaging technique helps to obtain a clearly defined response by reducing background activity. The relatively greater distance between recording and stimulating electrodes diminishes the stimulus artefact and, more importantly, eliminates the "direct" short-latency responses such as those observed in the elicitation of the classical anal reflex. The use of a surface anal plug electrode is preferable to concentric needle electrodes because a much larger area of muscle is sampled thus giving a more accurate indication of its function. Voltage was used to measure sensory threshold and stimulation parameters in this study as have many other previous reports, although current probably constitutes a better measurement.

Haldeman *et al* were able to demonstrate a conduction time of approximately 8 ms in the afferent limb of the pudendo-anal reflex by recording evoked potentials over the sacral conus.[20] Marsden *et al*, using percutaneous spinal cord stimulation, measured a latency of approximately 8 ms in the efferent limb of the reflex in normal subjects.[24] This observation was confirmed by Snooks *et al* who also showed prolongation of this latency in neurogenic incontinence,[32] thus demonstrating a lesion in the efferent

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pathway. Hence, assuming a normal latency range of 30–48 ms, the central conduction time for the pudendo-anal reflex has a range of 15–33 ms, thus confirming its polysynaptic nature.

The rather startling prolongation of pudendo-anal reflex latencies observed in neurogenic faecal incontinence (that is, more than expected by a delay in the efferent limb only), and the reduction in the amplitude of the response, suggests that in these patients the afferent impulse has some difficulty in stimulating the spinal α-motoneurons to generate a potential and hence evoke a response from the external anal sphincter. This may be due to a reduction in the number of such neurons innervating the sphincter as is reflected by the larger size of the motor units in these patients suggesting re-innervation.[13] A smaller motoneuron pool must also result in less interaction between them,[33] further "magnifying" the latency differences between normal subjects and patients with defects of sphincter innervation. Although there is much evidence for the presence of a lesion in the efferent side of the reflex arc,[32 34] it has been suggested that abnormalities may also exist in the afferent limb.[35 36] The analogy is clearly with multiple sclerosis in which the latency of the visual evoked response at the cortex is far longer than that expected for any delay in conduction in the optic nerves, thus indicating that the nervous system experiences difficulty in generating the potential.

Despite the wide range in health, the marked increase in latencies in incontinence are clearly of value as indices of neuropathy. Almost all the incontinent patients had latencies in excess of 50 ms (normal range 27·2–46·8 ms). The high correlation of latencies with MUPD is further direct evidence of the value of the pudendo-anal reflex as a predictor of neuropathy of this muscle. Additional evidence of a sphincteric cause of faecal incontinence in the patients in this study is provided by the manometric results (table 1).[8]

Causes of nerve damage other than a stretch injury of the terminal innervation of the sphincter musculature can result in increased latencies. Thus, following glans stimulation, Rushworth found reflexes with a latency of 120 ms in a patient with polyneuritis[37] and Vereecken *et al* recorded a latency of 180 ms in a patient with a T12 fracture.[21] Pedersen *et al* described latencies of up to 200 ms after perianal stimulation in cases where disc protrusion had caused a cauda equina syndrome.[1] Ertekin *et al* described abnormalities of the bulbocavernosus reflex in 40 patients with traumatic or compressive lesions of the conus medullaris or cauda equina.[17] Similar observations were made by Rockswold and Bradley.[38]

The polysynaptic nature of the pudendo-anal reflex and its reproducibility also render it a suitable tool for the electrophysiological exploration of the sacral spinal cord in the absence of neuropathic changes in the external anal sphincter. Hence, it has been used in the investigation of patients with neurogenic disorders of the urinary bladder and of sexual function.[16 28 29] Many of these patients often have radiological evidence of lumbo-sacral spinal dysraphism.[39] Similar observations have been made in some patients with intractable constipation of idiopathic origin.[40]

Electrophysiological measurement of the pudendo-anal reflex latency provides a simple and reliable method of evaluating pelvic floor neuropathy. It also provides information of the neurogenic function of the sacral conus. Its diagnostic value is increased when supplemented by other functional investigations. These include anorectal manometry, EMG (duration of motor units, number of polyphasic potentials, fibre density) and measurement of conduction velocity in the motor innervation of the pelvic floor.[41]

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