Int J Colorect Dis (1987) 2:93–95

International Journal of Colorectal Disease

Relation between perineal descent and pudendal nerve damage in idiopathic faecal incontinence

P. N. Jones, D. Z. Lubowski, M. Swash and M. M. Henry

Sir Alan Parks Physiology Unit, St. Marks Hospital, London, UK

Abstract. In 60 patients with idiopathic anorectal incontinence, without neurological disease, there was a significant relationship, shown by regression analysis, between the pudendal nerve terminal motor latency and the extent of perineal descent during straining (r 0.59; $p<0.001$), and the plane of the perineum on straining (r −0.61; $p<0.001$). These data are consistent with the suggestion that perineal descent can lead to stretch-induced damage to the perineal nerves in this condition.

In idiopathic faecal incontinence there is denervation, and therefore weakness, of the pelvic floor sphincter muscles [1–6]. This is associated with partial reinnervation of the external anal sphincter and puborectalis muscles [4, 5, 7], and with an increase in the terminal motor latency in the inferior rectal branches of the pudendal nerves [8, 9]. A similar abnormality has been reported in the perineal branches of the pudendal nerves in patients with idiopathic stress incontinence of urine [9, 10]. In about a fifth of patients with incontinence there is electrophysiological evidence of damage to the innervation of the external anal sphincter and puborectalis muscles in the cauda equina nerve roots [11]. We have suggested that damage to the distal portions of the innervation of these striated sphincter muscles result, in part from stretch injury to these nerves during the perineal descent that occurs with repeated defaecation straining [1, 2, 7, 12]. This may be initiated by abnormal bowel habit [13, 14], or by nerve damage caused during childbirth [15]. In previous observations in patients with perineal descent we have noted histological evidence of denervation and reinnervation in the striated pelvic floor sphincter muscles [12], and an increased pudendal nerve terminal motor latency (PNTML) [13], but have not explored the relation between the extent of perineal descent on straining and the PNTML. In this paper we describe the relation between these two variables in a group of patients with idiopathic (neurogenic) faecal incontinence.

Patients and methods

Sixty patients with idiopathic faecal incontinence [16], referred to the Physiology Department at St Mark's Hospital for investigation, but otherwise unselected, were studied. These consisted of 50 women and 10 men. None of these patients had neurological disease, and none gave a history of trauma to the pelvic floor.

Measurement of perineal descent

In each patient the position of the perineal plane at the anus was measured with respect to the plane of the ischial tuberosities using the St Mark's Hospital perineometer [7, 12]. The plane of the perineum was defined at rest (rest position) and during a maximal defaecatory strain (strain position). This method has been described previously [7, 10, 12, 15]. In 106 normal subjects previously studied in our laboratory the perineum was 2.5 cm (SD 0.6) *above* the plane of the ischial tuberosities at rest, but only 0.9 cm (SD 1.0) *above* this plane during straining [12]. The technique of measurement of perineal descent we have used provides a measure of the clinical phenomenon [12]. Although this technique probably underestimates the amount of perineal descent recorded by radiological measurement of the plane of the anorectal angle [17], the clinical measurement is reproducible, and does not involve exposure to X-irradiation.

Pudendal nerve terminal motor latency (PNTML)

The PNTML was measured using the standard method we have described previously [8, 18]. Transrectal stimulation of the pudendal nerves is achieved using supra-maximal stimuli delivered by a glove-mounted stimulating and recording electrode array, and the latency of the compound muscle action potential evoked in the external anal sphincter muscle (PNTML) is measured with cursors on the oscilloscope screen of the EMG apparatus or on paper recordings. The mean value of the pudendal nerve terminal motor latencies on the two sides in normal subjects is 2.0 ms (SD 0.2) [7, 8, 13, 15].

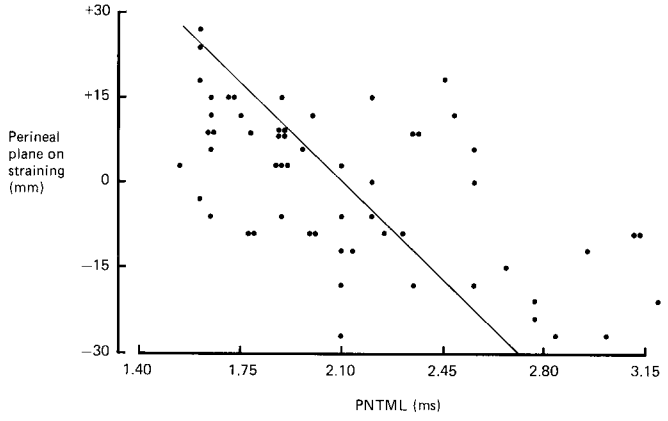

Fig. 1. Relation between perineal plane on straining and the pudendal nerve terminal motor latency (PNTML). Positive values for the perineal plane on the ordinate indicate a position of the perinaeum above the trans-ischial plane, and negative values a position below this plane. (Regression analysis $r - 0.61$; $p < 0.001$)

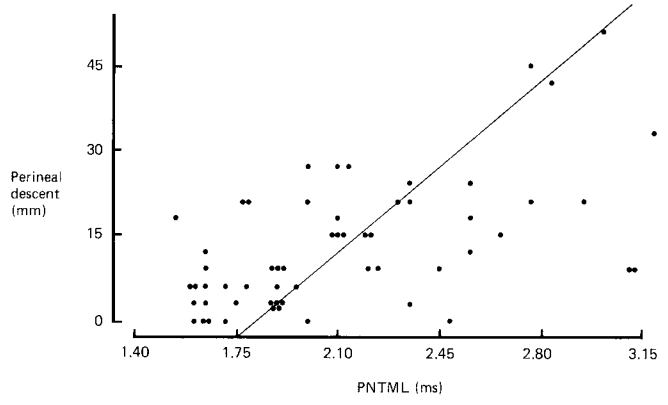

Fig. 2. Relation between extent of perineal descent on straining, expressed as the difference between the perineal plane at rest and the perineal plane on straining, and the pudendal nerve terminal motor latency (PNTML). (Regression analysis r 0.589; $p < 0.001$)

Results

In the 60 patients with anorectal incontinence the mean perineal plane at rest was 1.3 cm (SD 0.8) *above* the plane of the ischial tuberosities. During straining the perineum descended to a mean position 0.1 cm (SD 1.4) *below* the plane of the ischial tuberosities. The mean difference between these rest and strain positions was 1.3 cm (SD 1.7).

The mean PNTML in the 60 patients was 2.15 ms (SD 1.07). Regression analysis showed that the plane of the perineum during straining was correlated with the PNTML (r –0.610; $p < 0.001$), and that the difference between the perineal plane at rest and on straining was also correlated with the PNTML (r 0.589; $p < 0.001$) (see Figs. 1 and 2). There was no relation between the perineal plane at rest and the PNTML.

Discussion

Parks and colleagues [18] described abnormal perineal descent as a clinical syndrome but we now prefer to regard it as a physical sign indicative of weakness of the pelvic floor musculature, usually due to denervation. It is a common clinical feature in patients with faecal incontinence. In the patients with idiopathic faecal incontinence reported here linear regression analysis showed that there was a significant, positive correlation between an increasing PNTML and increasingly abnormal descent of the perineum on straining (Figs. 1 and 2), but not with the plane of the pelvic floor in the resting state. The mean PNTML was increased in most of our patients.

The PNTML is a measure of fastest motor conduction in the pudendal nerves. Since this fastest latency is not influenced by the presence of increased numbers of slowly conducting, damaged axons, the PNTML does not give a quantitative estimation of the extent of abnormality in the nerve. Similarly, the PNTML cannot be used as an indicator of normality or abnormality in the muscles innervated by this nerve. EMG, or single fibre EMG, is a better index of the latter since it will reveal evidence of partial reinnervation, a feature of axonal damage that is relevant to the histological findings of denervation and reinnervation in the pelvic floor muscles. Thus a normal PNTML does not indicate that the innervation is normal, only that fastest motor conduction is normal in this nerve. However, the correlation we have observed between the increase in the PNTML and the extent of perineal descent on straining is consistent with our suggestion that the latter is a factor leading to damage to the pelvic floor innervation in anorectal incontinence [1–4, 8–15, 19].

Perineal descent and damage to the pelvic floor innervation are inter-related phenomena [2, 12–16, 19]. Abnormal perineal descent can lead to damage to the pudendal and pelvic innervations of the external anal sphincter and puborectalis muscles, and damage to these innervations, as occurs in childbirth [15] or with cauda equina lesions [20], causes pelvic floor weakness and perineal descent. The latter can lead to progressive damage to the innervation of the striated sphincter musculature from recurring stretch-induced damage to these nerves [2–4, 7–11], leading to increasing weakness of the pelvic floor. Bartolo et al. [21] found that in continent women with perineal descent there was EMG evidence of reinnervation in both the external anal sphincter and puborectalis muscles. The correlation of the PNTML with perineal descent on straining that we have noted in idiopathic faecal incontinence is consistent with our formulation of the importance of perineal descent in the gradually

progressive course characteristic of this clinical syndrome. This concept is important in that it implies that medical or surgical measures to reduce defaecation straining and perineal descent may prevent progressive denervation of these perineal sphincter muscles, and thus prevent the development of incontinence.

Acknowledgements. We thank the St Mark's Hospital Research Foundation for financial support. DZL was supported by the RACS Edward Lumley Research Fellowship. PNJ, a medical student at Leeds University Medical School, was attached to the laboratory during an elective period.

References

1. Swash M (1985) New concepts in incontinence. Br Med J 290:4–5
2. Parks AG, Swash M, Urich H (1977) Sphincter denervation in anorectal incontinence and rectal prolapse. Gut 18:656–665
3. Beersiek F, Parks AG, Swash M (1979) Pathogenesis of anorectal incontinence; a histometric study of the anal sphincter musculature. J Neurol Sci 42:111–127
4. Neill ME, Swash M (1980) Increased motor unit fibre density in the external anal sphincter muscle in anorectal incontinence; a single fibre EMG study. J Neurol Neurosurg Psychiatry 43:343–347
5. Bartolo DCC, Jarratt JA, Read MG, Donnelly TC, Read NW (1983) The role of partial denervation of the puborectalis muscle in idiopathic faecal incontinence. Br J Surg 70:664–667
6. Womack NR, Morrison JFB, Williams NS (1986) The role of pelvic floor denervation in the aetiology of idiopathic faecal incontinence. Br J Surg 73:404–407
7. Henry MM, Swash M (1985) Coloproctology and the pelvic floor. Butterworth, London, pp 406
8. Kiff ES, Swash M (1984) Slowed conduction in the pudendal nerves in idiopathic (neurogenic) faecal incontinence. Br J Surg 71:614–616
9. Snooks SJ, Barnes PRH, Swash M (1984) Damage to the innervation of the voluntary anal and periurethral sphincter musculature in incontinence. J Neurol Neurosurg Psychiatry 47:1269–1273
10. Snooks SJ, Badenoch D, Tiptaft R, Swash M (1985) Perineal nerve damage in genuine stress urinary incontinence; an electrophysiological study. Br J Urol 57:422–426
11. Snooks SJ, Henry MM, Swash M (1985) Abnormalities in central and peripheral nerve conduction in anorectal incontinence. J R Soc Med 78:294–300
12. Henry MM, Parks AG, Swash M (1982) The pelvic floor musculature in the descending perineum syndrome. Br J Surg 69:470–472
13. Kiff ES, Barnes PRH, Swash M (1984) Evidence of pudendal neuropathy in patients with perineal descent and chronic straining at stool. Gut 25:1279–1282
14. Snooks SJ, Nicholls RJ, Henry MM, Swash M (1985) Electrophysiological and manometric assessment of the pelvic floor in solitary rectal ulcer syndrome. Br J Surg 72:131–133
15. Snooks SJ, Swash M, Setchell M, Henry MM (1984) Injury to the pelvic floor sphincter musculature in childbirth. Lancet 2:546–550
16. Parks AG (1975) Anorectal incontinence. Proc R Soc Med 68:681–690
17. Oettle GJ, Roe AM, Bartolo DCC, Mortensen NJ McC (1985) What is the best way of measuring perineal descent? A comparison of radiographic and clinical methods. Br J Surg 72:999–1001
18. Parks AG, Porter NH, Hardcastle J (1966) The syndrome of the descending perineum. Proc R Soc Med 59:477–482
19. Snooks SJ, Barnes PRH, Swash M, Henry MM (1985) Damage to the innervation of the pelvic floor musculature in chronic constipation. Gastroenterology 89:971–981
20. Swash M, Snooks SJ (1986) Slowed motor conduction in lumbosacral nerve roots in cauda equina lesions; a new diagnostic technique. J Neurol Neurosurg Psychiatry 49: 808–816
21. Bartolo DCC, Roe AM, Mortensen NJ McC (1986) The relationship between perineal descent and denervation of the puborectalis in continent patients. Int J Colorect Dis 1:91–95

Accepted 9 March 1987

Dr. M. Swash
St Mark's Hospital
City Road
London EC1 V2PS
UK