

Experimental Study No. 4: Abdominal pressure increase during anorectal closure is secondary to striated pelvic muscle contraction

PETER PETROS (*) - MICHAEL SWASH (**)

(*) Royal Perth Hospital, University of Western Australia

(**) Dept of Neurology, The Royal London Hospital, London, UK

Abstract: Abdominal pressure measurements during squeezing and straining simultaneous with vaginal EMG indicate that anorectal closure is not caused by raised intra-abdominal pressure, but by pelvic muscle contraction.

Key words: Anorectal closure; Raised intra-abdominal pressure; Integral theory.

INTRODUCTION

Though questions have been raised concerning the validity of valvular-type theories for anorectal closure,¹ for many physicians, such theories still have currency, possibly because of their seductive simplicity: raised intra-abdominal pressure presses the rectum down to increase the anorectal angle, and effect closure.² The aims of this study were to measure the abdominal pressures during “squeezing” (which interrupts defecation) and straining (which accelerates defecation). Simultaneous with this, surface cylindrical EMG electrodes were placed in the posterior fornix of vagina to confirm (or not) pelvic muscle contraction during the pressure rise. The Musculoelastic Theory as outlined in paper No. 1, predicts that organ movements are not caused by rise in intra-abdominal pressure. Rather it is pelvic muscle contraction which causes organ stretching and anorectal closure, the rise in intraabdominal pressure being secondary.

METHODS

The pressure and EMG studies were performed in 24 patients with no history of fecal dysfunction. Mean age was 50.5 years (range 29-71), parity 3 (range 1-7). Gaeltec micro-tip transducers were positioned in the bladder to measure the abdominal pressure during straining and also, “squeezing”. Simultaneous EMG recordings were taken from the posterior vaginal fornix using a cylindrical probe. Within this group, 20 patients had urinary incontinence, and 4 were asymptomatic. Separate radiological studies were performed during straining and squeezing on another occasion.

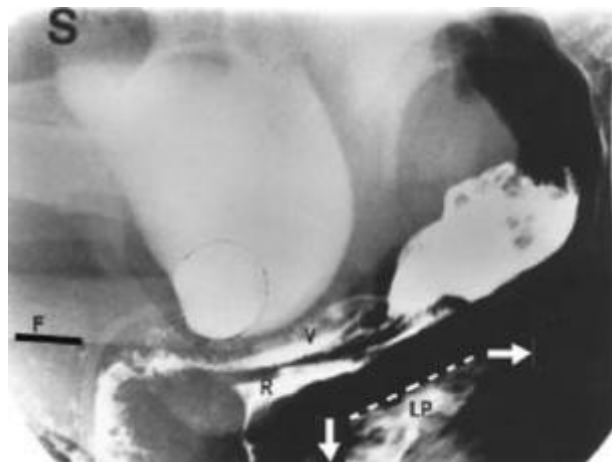


Fig. 1. – Straining X-ray, sitting position. With reference to “F”, lower border of femur, bladder, proximal vagina (V) and rectum (R) are stretched backwards and downwards during straining. Note downward angulation of levator plate (LP).

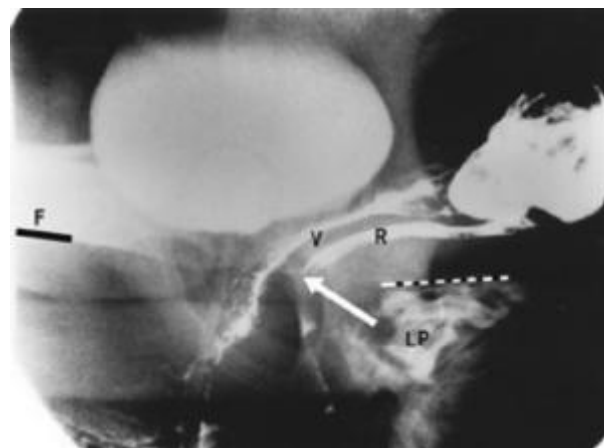


Fig. 2. – “Squeezing” X-ray, sitting position. With reference to “F”, lower border of femur, bladder, proximal vagina (V) and rectum (R) are stretched upwards and forwards. Note horizontal position of levator plate (LP).

RESULTS

Straining (Fig. 1) caused a similar rise in pressure (mean 29.75 cm water, range 0-72 cm) to that for “squeezing” (Fig. 2) (mean 24.5cm water, range 4-92 cm). EMG activity was noted in the posterior fornix of the vagina (the site of the puborectalis and levator plate muscles) both during straining and squeezing (Fig. 3).

DISCUSSION

Raised intra-abdominal pressure, a mainstay of valve-type theories for continence per se is unlikely to be a primary factor in anorectal closure. The bladder, proximal vagina and rectum are stretched backwards and downwards during straining (Fig. 1) and elevated forwards during ‘squeezing’

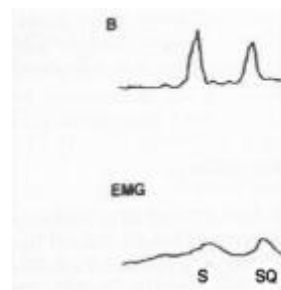


Fig. 3. – Simultaneous bladder and EMG measurements from the posterior vaginal fornix during straining (S) and squeezing (SQ). B = abdominal pressure as recorded in the bladder.

(Fig. 2) yet similar abdominal pressures were recorded. EMG activity simultaneous with pressure rise was recorded (Fig. 3), for both 'squeezing' and straining, indicating that the organ movements observed (Figs. 1, 2) were most likely activated by muscle activity. Furthermore, if raised intra-abdominal pressure were a factor in anorectal closure, straining during defecation, a common event, would immediately cause cessation of emptying, not the acceleration generally observed.

Sturmdorf (1919)³ explained the anatomical basis for our observations of raised intra-abdominal pressure during pelvic floor contraction: "the levator ani is the tensor of the pelvic fascia, the antagonist of the diaphragm and the abdominal muscles, contracting when these opposing muscles contract and relaxing when they relax".

CONCLUSIONS

The results do not support the pressure theory's statement that anorectal intra-abdominal pressure rise is a major factor in maintaining anorectal continence.

REFERENCES

1. Bartolo DCC, and Macdonald ADH. Fecal continence and defecation. In: *The Pelvic Floor, Its functions and disorders*, Eds Pemberton J, Swash M, Henry MM, WB Saunders, London, 2002; 77-83.
2. Parks AG. Anorectal incontinence. *Proc Royal Soc Med* 1975; 68: 681-90.
3. Sturmdorf A. The levator ani muscle. In: *Gynoplastic Technology*, FA Davis, Philadelphia, 1919; 109-114.

Correspondence to:

PETER PETROS

E-mail: kvinno@highway1.com.au