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A STUDY OF THE EFFECT OF RENAL PELVIC AND URETERIC DISTENSION ON THE ANORECTAL FUNCTION WITH IDENTIFICATION OF THE "RENO-ANAL REFLEX"

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1. ABSTRACT

Renal or ureteral diseases are often associated with gastrointestinal symptoms. In this communication, we studied the effect of renal pelvic or ureteric distension on the anorectal function in 12 healthy volunteers. A 3F balloon-tipped catheter was introduced into the renal pelvis. The balloon was filled with saline in increments of 2 ml up to 12 ml and the pressure response of the rectum and anal canal was recorded. Balloon filling was performed twice: rapidly (1ml/sec) and slowly (1 ml/min). The test was repeated in the ureter with balloon fillings in increments of 0.25 ml up to 1 ml. The electromyographic (EMG) response of the external anal sphincter to distension of the renal pelvis or ureter was evaluated. The pressure response of the rectum, anal canal and external anal sphincter to distension of the anesthetised renal pelvis or ureter was then determined. The test was repeated after external anal sphincter anaesthetisation. Rapid renal pelvic or ureteric distension increased the anal canal pressure ($p < 0.05$) but did not change the rectal pressure ($p > 0.05$). With 10 and 12 ml rapid renal pelvis distension and with 0.75 and 1 ml rapid ureteric distension, the EMG activity of the external anal sphincter increased and was accompanied with flank and anal pain. Slow renal pelvic or ureteric distension caused no significant change in the anal or rectal pressures ($p > 0.05$) or in the EMG activity of the external anal sphincter and no anal pain was perceived. Distension of the anesthetised renal pelvis or ureter effected no changes in anal or rectal pressures ($p > 0.05$). The anesthetised external anal sphincter as well as the anal or rectal pressure did not respond to renal pelvis or ureteric distension. In conclusion, rapid renal pelvic or ureteric distension stimulates the mechanoreceptors in the renal pelvis or ureteric wall leading to reflex external anal sphincter contraction. This leads to elevation of the anal canal pressure. The findings suggest the possible involvement of a "reno-anal reflex" which is evoked on rapid renal pelvis or ureteric distension. Slow distension does not seem to trigger such a reflex.

2. INTRODUCTION

Renal or ureteral diseases are often associated with gastrointestinal symptoms. Patients with acute pyelonephritis present not only with a localized flank pain, chills, fever and symptoms of vesical irritation but also with abdominal pain and distension (1-5). Renal colic is commonly associated with nausea, vomiting and abdominal distension (6-10). In some patients, the pain is felt deep in the pelvis and anus. Retrograde urograms which overdistend the renal pelvis may cause nausea, vomiting and a cramp-like pain in the abdomen (11-13). In other urologic conditions, like urolithiasis, which may be urologically silent, gastrointestinal symptoms may be present, leading to diagnostic confusion (14-15).

The current communication describes the effect of renal pelvic and ureteric distension on the anorectal function.

Table 1. Normal pressures in the urinary tract, rectum & anal canal

SITE	PRESSURE cm H_2O	
	Mean	Range
Renal pelvis	5.8 ± 1.3	3 - 9
Ureter	6.3 ± 1.9	3 - 8
Rectum	6.7 ± 1.5	4 - 7
Anal canal	73.8 ± 6.2	60-85

3. MATERIAL AND METHODS

3.1 Subjects

12 healthy volunteers (9 men and 3 women) signed an informed consent before entering into the study. The study was approved by the Internal Review Board at our institution. Mean age was 38.6 ± 11.3 SD years (range 26-44). They had no history of urinary troubles. Urinalysis and sonography of the urinary tract were normal. Physical examination, including neurologic assessment, was also normal.

3.2 Methods

With the subject lying in a supine position, a 3F balloon-tipped ureteric catheter with a metallic clip at its end for fluoroscopic control was introduced into the renal pelvis. The balloon measured 0.5 cm in diameter and was made of latex (London Rubber Industries Ltd, London, UK). The catheter was connected to a strain gauge pressure transducer (Statham, 230B, Oxnard, California, USA). Pressure measurements started with the gauge at zero level. The filling of the balloon when it was not in the renal pelvis (i.e. in air) showed no transient pressure rise.

The pressures in the anal canal and rectum were simultaneously measured by means of a two-channel microtip catheter (Wiest Urocompact, California, USA). The catheter was placed 8-10 cm from the anal orifice so that the distal transducer resided in the rectum. The proximal transducer was adjusted to lie in the anal canal 2-3 cm from the anal orifice. The basal pressures in the renal pelvis as well as in the rectum and anal canal were recorded. The balloon in the renal pelvis was filled with saline in increments of 2 ml up to 12 ml and the pressure response of the renal pelvis, rectum and anal canal to renal pelvic distension by the balloon was recorded. The catheter was then withdrawn from the renal pelvis to the ureter using a mechanical device for automatic catheter withdrawal (902, Disa, Copenhagen, Denmark).

The position of the catheter was fluoroscopically controlled. The balloon in the ureter was filled with saline in increments of 0.25 ml up to 1 ml and the pressure response of the ureter, anal canal and rectum was recorded while the balloon was situated in the upper, middle and lower third of the ureter.

Renal pelvic and ureteric distension was done twice: once rapidly with a balloon filling at a rate of 1 ml/s and another time slowly at a rate of 1 ml/min.

3.2.1 External anal sphincter electromyography

The electromyographic (EMG) activity of the external anal sphincter to balloon distension of the renal pelvis and ureter was evaluated.

A concenteric needle EMG electrode was introduced into the external anal sphincter using the technique previously described (16). The normality of the sphincter had been ascertained by EMG testing before starting the experiment.

3.2.2 Anesthetisation of the renal pelvis, ureter and external anal sphincter

The renal pelvis and ureter were anesthetised by administration of 5 ml of 2% xylocaine (Astra, S.dert.lje, Sweden) diluted with 15 ml of saline using a 3 F ureteric catheter which was introduced through the ureteric orifice into the renal pelvis.

The pressure response of the anal canal, rectum and external anal sphincter to separate distension of the renal pelvis and ureter in its upper, middle and lower third was registered after 20 minutes of local anesthesia and 2 hours later when the effect of the anesthetic had waned. On another day, the test was repeated using saline instead of xylocaine.

The external anal sphincter was anesthetised by injecting 1 ml of 2% xylocaine diluted with 2 ml normal saline. A 23 gauge needle was introduced into the perianal skin, 0.5-1 cm lateral to the anal verge and 0.5 cm deep, and 1.5 ml of the anesthetic solution was injected on each side of the anal orifice. The EMG activity of the external anal sphincter as well as the rectal and anal canal pressure response to renal pelvic and ureteric distension was determined after 20 minutes of anesthetisation and 2 hours later when the anesthetic effect had disappeared.

To assure reproducibility of the results, the pressure measurements were done at least twice in each individual and the mean value was calculated.

3.2.3 Statistical analysis

The results were analysed statistically using the Student's t test. Differences assumed significance at $p < 0.05$ and values were given as mean ± standard deviation.

4- RESULTS AND DISCUSSION

The tests were completed and evaluated in all the volunteers with no adverse effects. The mean basal pressures are shown in table 1.

4.1 Effect of renal pelvic distension on the anal and rectal pressures

The effect of rapid renal pelvic distension in increments of 2 ml of saline on the pressures in the renal pelvis, rectum and anal canal is shown in figure 1. The renal

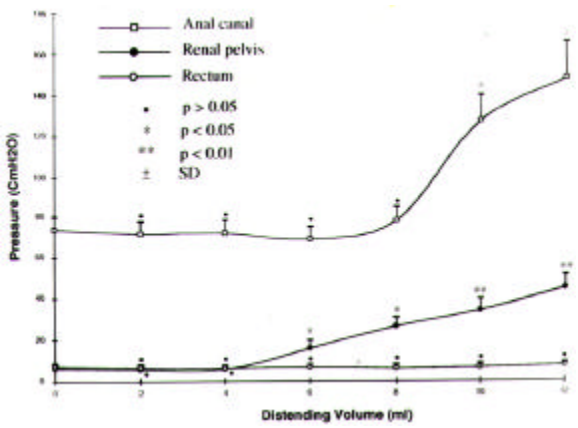

Figure 1: Effect of rapid renal pelvic distension in increments of 2 ml of saline on the pressure in the renal pelvis, rectum and anal canal.

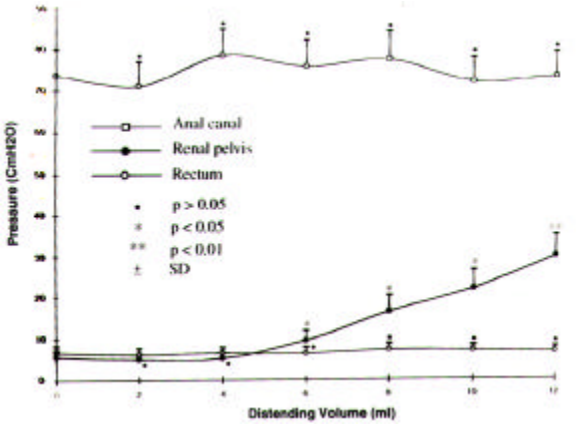

Figure 2: Effect of slow renal pelvic distension in increments of 2 ml of saline on the pressure in the renal pelvis, rectum and anal canal.

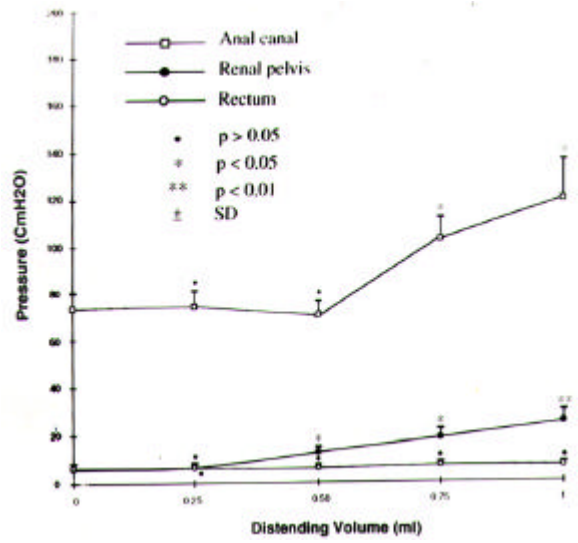

Figure 3: Effect of rapid ureteric distension in increments of 0.25 ml of saline on the pressure in the ureter, rectum and anal canal.

pelvic pressure increased significantly ($p < 0.05$) on distension with 6 ml of saline. The more the renal pelvis was distended, the more the renal pelvic pressure increased whereas the rectum did not record significant pressure changes ($p > 0.05$). Meanwhile, the anal canal pressure increased significantly on renal pelvic distension with 10 and 12 ml of saline ($p < 0.05$; figure 1). We did not try distension of the renal pelvis with more than 12 ml of saline due to possibility of injury.

The subjects felt renal and anal pain only on rapid renal pelvic distension with 10 and 12 ml of saline (table 2); no pain was felt with 2-8 ml distension. The renal pain occurred in the flank and was a dull ache while the anal pain was felt in the anus and lower pelvic area as a sharp stabbing ache. The severity of the flank and anal pain increased with more renal pelvic distension, was felt as long as the distension was maintained and disappeared upon release of the distension.

Slow renal pelvic distension led to rise in renal pelvic pressure with volumes greater than 6 ml ($p < 0.05$). However, the pressure rise was significantly lower than that produced by rapid distension ($p < 0.05$; figure 2). No significant rectal or anal pressure changes occurred ($p > 0.05$; figure 2). Flank pain was felt with 10 to 12 ml distension, but anal pain did not occur.

4.2 Effect of ureteric distension on the anal and rectal pressure

Rapid distension of the upper third of the ureter showed significant ureteric pressure rise with 0.5, 0.75 and 1 ml distension ($p < 0.05$; $p < 0.05$; $p < 0.01$, respectively; figure 3). The ureteric pressure continued to rise as the ureteric distension increased. While the rectum exhibited no significant pressure rise ($p > 0.05$) upon ureteric distension, a significant pressure increase was recorded in the anal canal with 0.75 and 1 ml distension ($p < 0.05$; $p < 0.05$, respectively; figure 3). Flank and anal pain were felt on ureteric distension with 0.75 and 1 ml of saline. The flank pain occurred as a dull ache while the anal pain was sharp and stabbing and was felt in the anus and lower pelvic area. The flank pain occurred with a distending ureteric volume less than that of the anal pain. Both the flank and anal pain disappeared with the release of the ureteric distension.

Slow ureteric distension caused ureteric pressure rise with 0.75 and 1 ml of saline and led to flank pain (figure 4). Meanwhile, no significant rectal or anal pressure changes ($p > 0.05$) occurred and no anal pain was felt. The ureteric pressure rise was significantly lower ($p < 0.05$) than that caused by rapid ureteric distension (figures. 3-4).

Distension of the middle and lower third of the ureter produced results similar to those produced by the upper third distension with no significant difference ($p > 0.05$). We did not try ureteric distension with more than 1 ml in view of possibility of injury.

4.3 Effect of renal pelvic and ureteric distension on the external anal sphincter EMG

The basal activity of the external anal sphincter showed a mean amplitude of motor unit action potentials of 102.4 ± 26.2 µV (range 80-121). This basal EMG showed no significant change of activity on rapid renal pelvic distension up to 8 ml (figure 5). A significant increase of

Table 2: Incidence of flank and anal pain on rapid renal pelvic distension

DISTENDING VOLUME (ml)	**PAIN**	
	Renal	Anal
2	-	-
4	-	-
6	-	-
8	-	-
10	+	+
12	+	+

Table 3: Incidence of flank and anal pain on rapid ureteric distension

DISTENDING VOLUME (ml)	**PAIN**	
	Renal	Anal
0.25	-	-
0.5	+	-
0.75	+	+
1.00	+	+

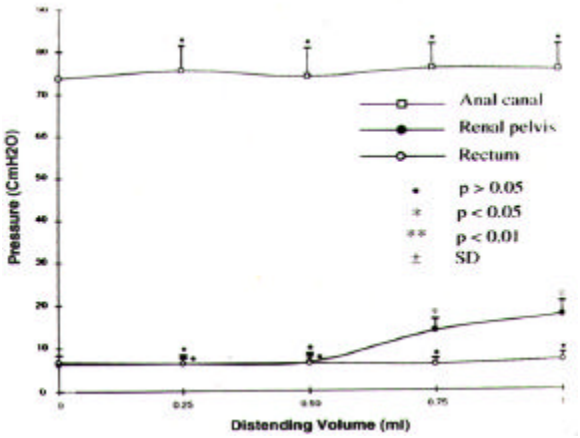

Figure 4: Effect of slow ureteric distension in increments of 0.25 ml of saline on the pressure in the ureter, rectum and anal canal.

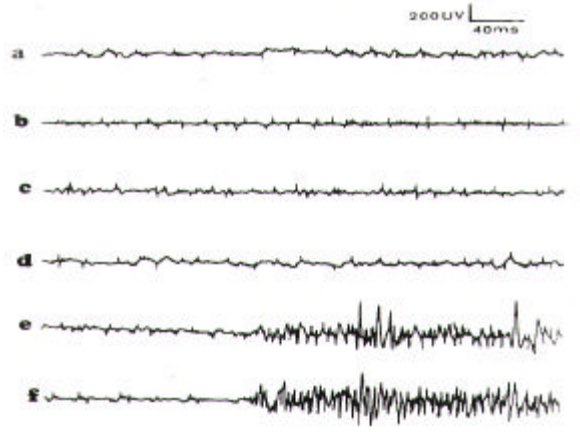

Figure 5: The EMG activity of the external anal sphincter on rapid renal pelvic distension with a) 2, b) 4, c) 6, d) 8, e)10 and f)12 ml of saline.

EMG activity occurred with 10 and 12 ml distension and the activity was more on distension with 12 ml than with 10 ml (figure 5). The motor unit action potentials recorded a mean of 586.3 ± 82.8 µV (range 418-750) at 10 ml renal pelvic distension and 674.6 ± 94.5 µV (range 533-890) at 12 ml. The EMG activity returned to the basal value on release of distension. The increased external anal sphincter activity was associated with anal and lower pelvic pain which disappeared upon release of renal pelvic distension.

The external anal sphincter exhibited increased EMG activity only with 0.75 and 1 ml rapid distension of the upper third of the ureter (figure 6). It recorded mean motor unit action potentials of 528.6 ± 78.2 µV (range 427-780) with 0.75 ml distension and 612.8 ± 88.5 µV (range 508-863 µV) with 1 ml distension. The increased external anal sphincter EMG activity was associated with anal and lower pelvic pain. No increased activity occurred on ureteric distension with volumes lower than 0.75 ml. The external anal sphincter EMG actvity returned to the basal level upon release of distension. These findings also occurred with no significant difference ($p > 0.05$) on distension of the middle and lower third of the ureter.

Slow renal pelvic or ureteric distension caused no significant change in the EMG activity of the external anal sphincter ($p > 0.05$); no anal pain was felt.

4.4 Effect of renal pelvic and ureteric distension on the anesthetised renal pelvis, ureter and external anal sphincter

Rapid and slow distension of the renal pelvis or ureter, 20 minutes after having been anesthetised did not cause significant pressure changes in the renal pelvis, ureter, rectum or anal canal ($p > 0.05$). No flank or anal pain was felt. Distension after two hours, when the anesthetic effect had worn out, produced pressure response as well as flank and anal pain similar to that observed before anesthetisation with no significant difference ($p > 0.05$). Distension of the renal pelvis or ureter after saline administration produced pressure responses in the renal pelvis, ureter, rectum and anal canal similar to those before saline administration ($p > 0.05$).

The external anal sphincter EMG activity, as well as rectal and anal canal pressures did not respond to rapid or slow renal pelvic or ureteric distension 20 minutes after external anal sphincter anesthetisation; the subjects did not feel anal pain. Two hours later, when the anesthetic effect had waned, the external anal sphincter showed increased activity; the anal canal pressure was elevated on rapid renal pelvic or ureteric distension as mentioned previously and flank and anal pain was felt.

In the current study, we performed rapid renal pelvic and ureteric distension in an attempt to simulate the distending effect of a calculus impacted in the ureteropelvic junction or ureter. The effect of slow renal pelvic or ureteric distension could be compared with that of partial obstruction of the renal pelvis or ureter.

Rapid renal pelvic or ureteric distension caused an increase of both the anal pressure and external anal sphincter EMG activity as well as flank and anal pain. The flank pain

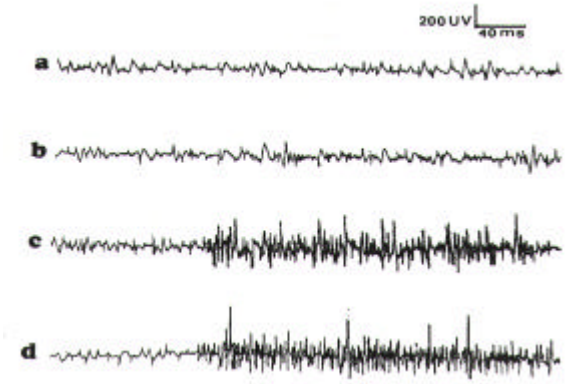

Figure 6: The EMG activity of the external anal sphincter on rapid ureteric distension with a) 0.25, b) 0.5, c) 0.75 and d) 1 ml of saline.

occurred with significant distension of the renal pelvis and appears to be due to the increase of the renal pelvic pressure. The anal pain is presumably caused by external anal sphincter contraction as evidenced by the increase of its EMG activity on renal pelvic or ureteric distension. Alternatively, it might be caused by the high anal pressure which occurs on renal pelvic or ureteric distension. The elevated anal pressure and the anal pain may be due to increased contractile activity of the internal anal sphincter. However, the fact that the anal canal pressure did not increase and the anal pain was not felt on renal pelvic or ureteric distension while the external anal sphincter was anesthetised, does not support such a conclusion and confirms that the external anal sphincter is the source of these findings. Furthermore, the increased EMG activity of the external anal sphincter on renal pelvic or ureteric distension is another evidence that this sphincter is the source of the elevated anal canal pressure.

The present findings demonstrate a previously unrecognized relationship between renal pelvic or ureteric distension and the external anal sphincter . The external anal sphincter response to renal pelvic or ureteric distension affirms the hypothesis of the possible involvement of a reflex which we term "reno-anal reflex". This reflex relationship is evidenced by reproducibility and its absence on anesthetizing either of the renal pelvis and ureter or the external anal sphincter , both presumably representing the 2 arms of the reflex arc.

We do not know yet the role of the reno-anal reflex which might be evoked by rapid renal pelvic or ureteric distension as for example by means of a calculus impacted in the ureteropelvic junction or in the ureter. The external anal sphincter contraction might confine the bowels and thus could explain the abdominal distension which occurs in subjects who are passing a stone down the ureter or have an impacted calculus in the ureteropelvic junction. Although in such cases, the urinary symptoms commonly overshadow the anal pain and abdominal distension, the latter symptoms could be misleading when outstandingly present.

Slow distension of the renal pelvis or ureter did not cause anal pressure elevation or pain, nor an increase of the external anal sphincter EMG activity.

5. ACKNOWLEDGMENT

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