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Original Article

Pelvic Floor and Abdominal Muscle Interaction: EMG Activity and Intra-abdominal Pressure

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Abstract: Pelvic floor muscle exercises prescribed for the treatment of incontinence commonly emphasize concurrent relaxation of the abdominal muscles. The purpose of this study was to investigate the interaction between individual muscles of the abdominal wall and the pelvic floor using surface and intramuscular electromyography, and the effect of their action on intra-abdominal pressure. Four subjects were tested in the supine and standing positions. The results indicated that the transversus abdominis (TA) and the obliquus internus (OI) were recruited during all pelvic floor muscle contractions. It was not possible for these subjects to contract the pelvic floor effectively while maintaining relaxation of the deep abdominal muscles. A mean intra-abdominal pressure rise of 10 mmHg (supine) was recorded during a maximum pelvic floor muscle contraction. These results suggest that advice to keep the abdominal wall relaxed when performing pelvic floor exercises is inappropriate and may adversely affect the performance of such exercises.

Keywords: Abdominal muscles; Electromyography; Intra-abdominal pressure; Pelvic floor muscles

Introduction

Isolation of the pelvic floor muscles, specifically eliminating abdominal muscle activity, is inherent in many guidelines for pelvic floor muscle (PFM) exercises [1–3]. Promotion of an isolated PFM contraction is to avoid an increase in intra-abdominal pressure (IAP) [4] which may provoke or exacerbate symptoms of stress urinary incontinence (SUI) and prolapse [5].

Pressure in the abdominal cavity is determined by the combined action of the pelvic floor, the abdominal wall and the diaphragm [6]. A number of studies investigating spinal stability have examined the relationship between IAP and activity of the abdominal muscles, in particular the transversus abdominis (TA), but only one study of lifting in males has demonstrated a relationship between PFM activity and IAP generation [7]. Despite the prevalence of SUI and prolapse in the female population [8] and the need for corrective surgery in many cases, there are no studies of the relationship in women between the muscles surrounding the abdominal and pelvic cavities and pressure generated by their contraction.

In the literature on pelvic floor training for incontinence, reference is commonly made to 'the abdominal muscles' without recognition of the individual muscles or their discrete actions [9]. Furthermore, there has been little research into the interaction between the different layers of the abdominal wall and the pelvic floor. In one small study of nulliparous women the rectus abdominis was investigated and shown to be activated during a strong pelvic floor muscle contraction [10]. In 7 parous women, intramuscular EMG was used to demonstrate different patterns of coactivation of the abdominal muscles when the pelvic floor muscles were contracted in three positions of the lumbar spine [11]. Activation of the pelvic floor muscles was verified by digital palpation. In addition, in 2 subjects activity of the pelvic floor during isometric abdominal exercises was also investigated using intramuscular EMG, and an increase in PFM EMG activity was demonstrated. Recruitment of the striated urethral sphincter and PFM was investigated in

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functional activities [12]. Coactivation of the PFM, but not the striated urethral sphincter, was shown when subjects performed exercises commonly known as sit-ups and pelvic tilts. The EMG activity of the abdominal muscles was not studied in these same subjects. None of these studies have addressed the effect of abdominal and PFM activity on IAP generation, although Sapsford et al. [11] concede that consideration of IAP is warranted.

Changes in PFM function have been demonstrated in women with stress urinary incontinence (SUI), including loss of the reflex activity of the pelvic floor [13] and PFM weakness [14]. PFM exercises form the mainstay of the conservative management of SUI [15], and although different training regimens have been described, specific strength training has been shown to be effective and is recommended [16]. Strength training of muscles relies on overload, i.e. the development of maximum or near maximum tension in the target muscles [17]. When this principle is applied to pelvic floor training, together with instructions to relax the abdominal wall, clinically it appears that adequate pelvic floor muscle recruitment may not be achieved.

Other studies suggest that a specifically timed PFM contraction just prior to and during a cough reduces incontinence in older women with SUI [18]. Because the abdominal wall is activated during coughing and other activities causing a rise in IAP, an isolated PFM contraction may be ineffective for bladder control during such activities. Despite the prevalence of instructions to avoid abdominal muscle contraction, there have been no studies investigating the efficacy of such an isolated PFM contraction.

We conducted this study to investigate the relationship between activity in the four abdominal muscles and the pelvic floor, and the effect of this activity on IAP generation during a maximal PFM contraction, during a PFM contraction with instruction to relax the abdominal wall, during common abdominal exercises, and during coughing and forced expiration. Tests were performed in both supine and standing positions.

Materials and Methods

Written informed consent was obtained from 4 nulliparous women aged 25–42 years (mean 34 years), who were tested on two occasions 1 week apart. Ethical approval for the study was obtained from the Human Ethics Research Committee of the University of South Australia. Exclusion criteria were a skinfold thickness of >2.5 cm, a history of low back pain within the last 6 months requiring time away from work or sport, known or suspected pregnancy, urinary incontinence, urinary tract infection, vaginal infection, and surgery involving incision of the left abdominal wall.

Electromyographic and Intra-abdominal Pressure Recordings

Purpose-designed equipment simultaneously recorded six channels of EMG and two channels of pressure. EMG activity from the abdominal and pelvic floor muscles was recorded using a combination of surface and fine-wire intramuscular EMG electrodes, with simultaneous recording of IAP from an intravaginal sensor. Test–retest analysis of the method was shown to be highly reliable for all measures except IAP in standing, which will not be reported.

Electromyography

Lean subjects with an abdominal and suprailiac skinfold thickness of <2 cm were recruited to facilitate lodgement of the wires in TA and to reduce EMG artefact due to interposed adipose tissue between the surface electrode and the target muscle. Abdominal and suprailiac skinfold thickness was measured with Harpenden Skinfold Calipers using a standard technique [19].

Intramuscular EMG. The technique was similar to that described by Hodges and Richardson [20]. Bipolar fine-wire electrodes were fabricated from Medwire Teflon-coated silver wire with the distal 2 mm of insulation removed, and inserted into a hypodermic needle (22 G $\times$ $1\frac{1}{2}$ inch). The receptive ends were bent back 3 mm and 5 mm, respectively, against the needle tip and staggered to avoid contact with each other. The skin at the wire insertion site was prepared with EMLA anesthetic cream and the electrode was inserted into the left TA 2 cm medial to the midpoint of a line from the anterior superior iliac spine (ASIS) to the costal margin. The needle was inserted under real-time ultrasound guidance, which provided visualization of the needle tip and allowed exact deposition of the fine wires into the muscle. The three muscle layers could be easily visualized on the ultrasound screen. The muscle thickness and depth from the skin surface were measured using a built-in on-screen calliper. The wires were inserted with the subject lying on her side, after which she was carefully moved into a supine position. Once positioned, the needle was withdrawn, leaving the wires in situ. The external portion of the wires was taped to the skin to prevent dislodgement during trunk movement. Change of testing position between supine and standing was effected with a tilt table to reduce the risk of traction on the wires (Fig. 1).

Surface EMG. Surface EMG electrodes (Meditrace infant pellet electrodes) with an interelectrode distance of 1.5 cm were applied to the abdomen after standard preparation to reduce skin impedance [21]. Electrodes were placed over left lower rectus abdominis (RA) (2 cm lateral and caudal to the umbilicus), left obliquus externus (OE) (over the tip of the eighth rib and angled diagonally in the direction of the muscle fibers)

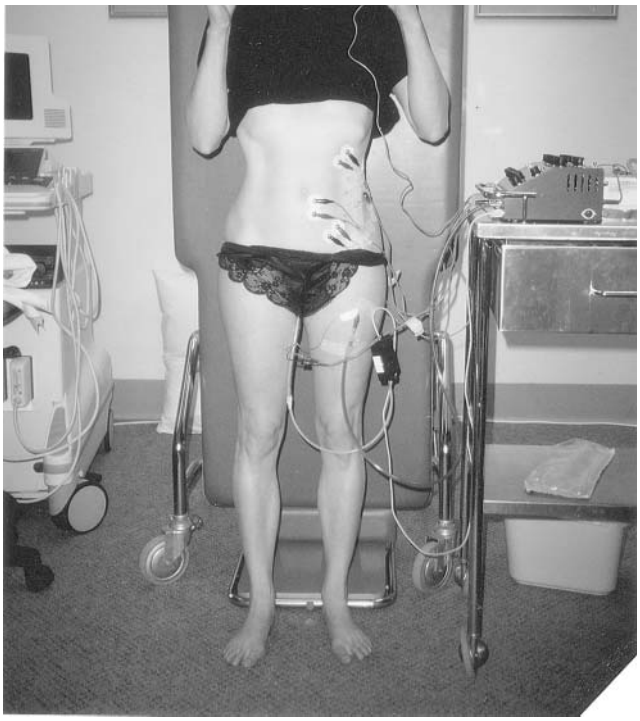

Fig. 1. Standing test position.

and left obliquus internus (OI) (2 cm proximal to the midpoint of a line from the ASIS to the symphysis pubis). A lip clip was used as a ground electrode [21].

Vaginal surface EMG was recorded from a pair of 3M TENS self-adhesive electrodes, trimmed to 1 cm in circumference and placed on the opposite sides of an air-inflated 7 mm HiLo endotracheal tube (Fig. 2). The electrodes were held in place with OpSite Flexifix with a 5 mm circular cut-out over the electrode to permit conduction of the EMG signal. The endotracheal tube was inflated to conform to the vaginal dimensions and inserted so that the SEMG electrodes were 3.5 cm from the vaginal introitus, to align with the pelvic floor muscle [22]. A rubber ring, placed on the endotracheal tubing 3.5 cm distal to the midpoint of the vaginal electrode, marked its location against the perineum, ensuring that the vaginal SEMG electrodes were maintained in the optimum position.

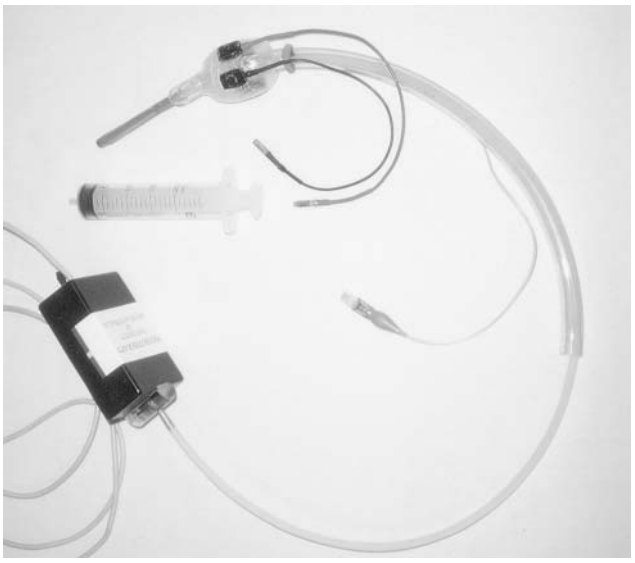

Fig. 2. Inflated endotracheal tube with IAP sensor and vaginal surface EMG electrodes in situ.

Intra-abdominal Pressure

Intra-abdominal pressure was recorded in mmHg using a specially designed sensor (Aviation Acrylic Mouldings) 1 cm × 2 cm × 0.25 mm, made of fluid-filled silicone, that was passed through the endotracheal tube and positioned in the posterior fornix of the vagina [23].

Signal Processing

EMG data were sampled at a rate of 2000 Hz with analog to digital conversion and bandpass filtered at 20–1000 Hz for intramuscular and 20–500 Hz for surface EMG. All data were rectified, integrated and stored on a computer for later analysis. Fast Fourier transform was performed to ensure that only frequencies representing voluntary muscle activity were recorded and analysed. Baseline EMG activity was subtracted and all data were normalized before analysis. The raw data were viewed offline to confirm different onsets of activity at the TA and OI recording sites as evidence of correct placement of the fine-wire electrode in TA, and not in the thicker and more superficial fibers of OI.

Procedure

Subjects were asked to void prior to testing in an attempt to standardize bladder volume. Subjects were taught how to perform the tests correctly before testing, and correct PFM action was confirmed [10].

Supine Test Position. Tests were performed in the supine position with the hips and knees extended, and with one pillow under the head. A Chattanooga pressure biofeedback device was inflated to 10 mmHg and placed centrally under the lumbar spine to maintain a neutral curve and to monitor changes in the pressure exerted by the lumbar spine due to spinal movement in relation to the supporting surface.

Standing Test Position. Subjects were moved to a vertical position on a tilt table. They then stepped forward off the table on to the floor to ensure a relaxed standing position.

Tests

The supine abdominal test was performed in the supine position, and the other five tests were performed both supine and standing. The starting position was randomly assigned to either supine or standing, and was only changed once during the testing procedure.

1. *Supine abdominal test.* With the hands behind the head, subjects were asked to simultaneously lift the head and shoulders off the bed and lift both feet 10 cm off the table, and to sustain the position for 3 seconds.
2. *Maximal pelvic floor muscle contraction.* Subjects were instructed to contract the muscles around the vagina 'like a drawstring' and to lift them internally. No posterior tilt of the pelvis was allowed and this was monitored by observation of the pressure in the biofeedback device under the lumbar spine. There was no instruction to either use or not use the abdominal muscles.
3. *Isolated pelvic floor muscle contraction.* Subjects were instructed to contract the PFM as in the previous maximal test, but were instructed specifically *not* to contract the abdominal muscles.
4. *'Belly-in'.* Subjects were instructed to draw in the lower abdominal wall towards the spine, an action which specifically activates TA [24]. The subject was required to breathe in a relaxed manner. No movement of the lumbar spine was allowed, and compliance with this request was monitored in the supine position by observation of the pressure in the biofeedback device, which was not allowed to fluctuate.
5. *Cough.* Subjects performed one maximal cough.
6. *Forced expiration.* The procedure for forced expiration was standardized by asking the subjects to blow into a plastic tube connected to a manometer, elevating the pressure to 40 mmHg as quickly as possible.

Evaluation Procedure, Including Statistical Methods

Subjects were tested twice 1 week apart. Two trials were performed for each test, with 1 minute's rest between trials. The mean normalized EMG scores of each of the target muscles for all four subjects are reported. Baseline EMG recordings were made and monitored both supine and standing, to ensure minimal activation of the target muscles at rest and to calculate the rise in EMG activity from baseline. The subjects performed the supine abdominal test to maximally recruit all the abdominal muscles. The EMG activity from this test was used to normalize the data from the four abdominal muscles during all other tests. The EMG activity from a maximal contraction of the PFM was used to normalize the data for the PFM for all tests. Patterns of muscle recruitment were evaluated by comparing rises in EMG activity above baseline, expressed as percentages. Mean scores and ranges (mmHg) are reported for IAP in the supine position.

Results

Recruitment Patterns

There were individual differences in the relative recruitment of the individual abdominal muscles and the pelvic floor muscles between the four subjects. However, each subject performed consistently for both trials and on the two test occasions.

1. *Supine abdominal test.* The abdominal muscles were strongly activated in the supine abdominal test. The abdominal EMG values from this test were used to normalize subsequent tests and are thus expressed as 100%. All other abdominal tests are expressed as a percentage of this. The increase in normalized PFM activity was 44% more than for a maximal PFM contraction (Fig. 3).
2. *Maximal pelvic floor muscle contraction.* TA and OI EMG activity was increased to 66% of the level during the supine abdominal test, with minimal recruitment of OE (6%) and RA (5%) (Fig. 4). A similar pattern was observed in the standing position (Fig. 5).
3. *Isolated PFM contraction.* The PFM was contracted to 26% of the maximum voluntary contraction when the subjects attempted to isolate the PFM, i.e. without concomitant activity in the abdominal wall. There was a rise in EMG activity in all of the abdominal muscles of 3%–12%, but most in OI and TA (Fig. 6).
4. *'Belly-in'.* 'Belly-in' produced an increase of 112% in TA EMG activity, with a rise of 84% in OI and 40% in the PFM (Fig. 7).
5. *Cough.* Coughing produced a rise of 179% in TA EMG activity and 136% in OI. An increase of 64% was recorded in PFM EMG (Fig. 8).
6. *Forced expiration.* An increase of EMG activity of 141% was recorded in TA and 93% in OI during forced expiration. The PFM (57%) and OE (21%) had smaller rises in EMG activity (Fig. 9).

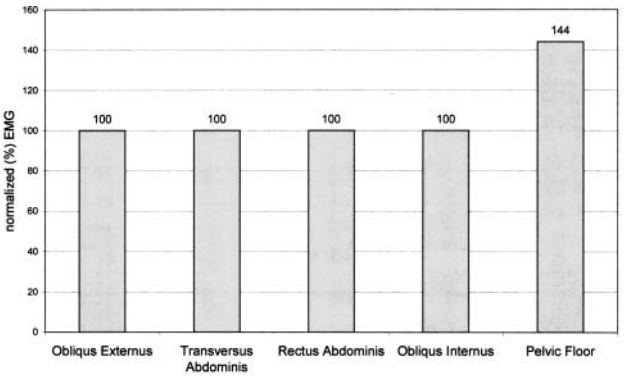

Fig. 3. Supine abdominal test.

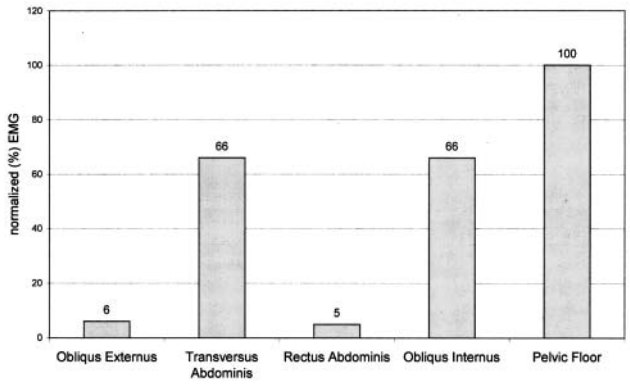

Fig. 4. Maximum pelvic floor muscle contraction (supine).

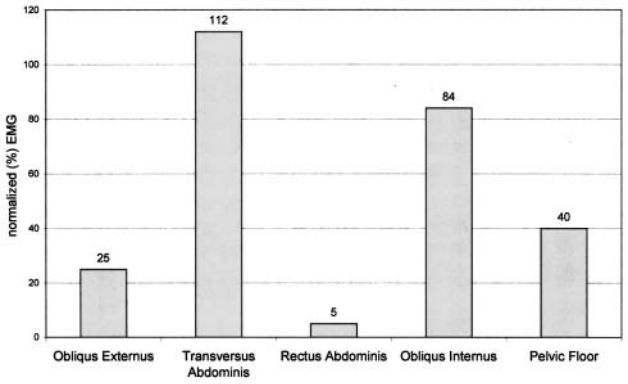

Fig. 7. Belly-in.

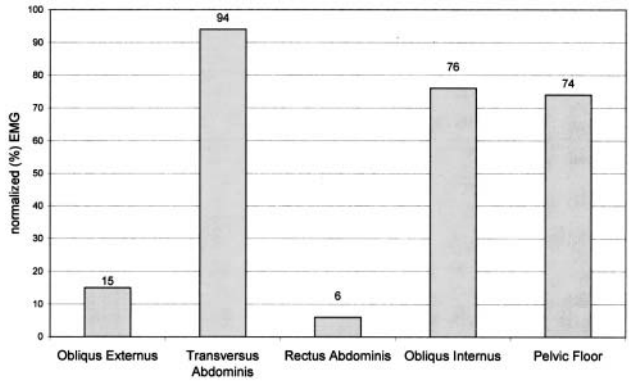

Fig. 5. Maximum pelvic floor muscle contraction (standing).

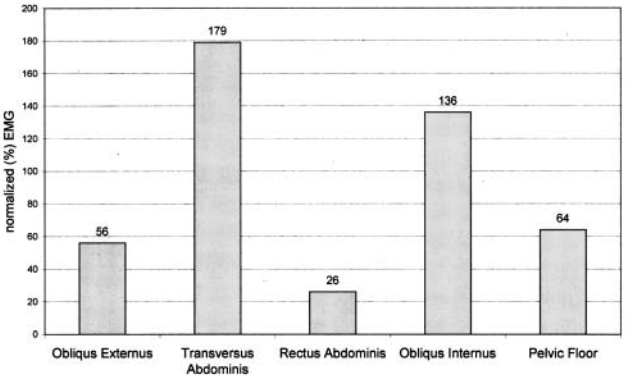

Fig. 8. Cough.

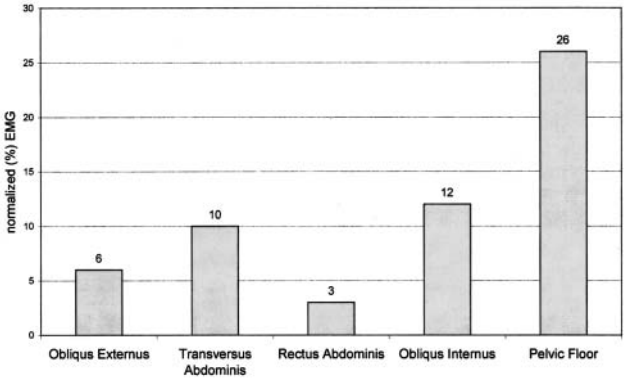

Fig. 6. Isolated pelvic floor muscle contraction.

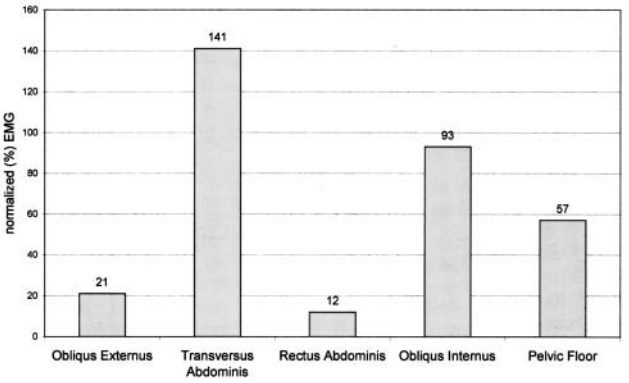

Fig. 9. Forced expiration.

Intra-abdominal Pressure

Rises in IAP were greatest during coughing (mean 46 mmHg) and then during forced expiration (36 mmHg). Maximum PFM contraction produced a mean rise of 9 mmHg (Table 1).

Table 1. Intra-abdominal pressure recorded in the supine position

Intra-abdominal pressure	Mean (range) mmHg
Supine abdominal test	27 (11–34)
Maximum PFM	9 (2–19)
PFM isolated	6 (1–14)
Belly-in	6 (1–18)
Cough	46 (37–55)
Forced expiration	36 (33–52)

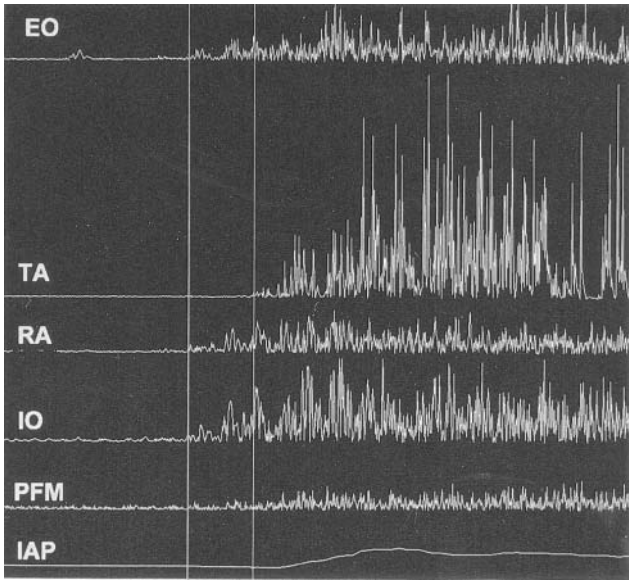

Fig. 10. Raw EMG data and IAP pressure curve of supine abdominal test to show different onset of activity of TA and OI. The vertical markers are set 200 ms apart. EO, obliquus externus; TA, transversus abdominis; RA, rectus abdominis; IO, obliquus internus; PFM, pelvic floor muscles; IAP, intra-abdominal pressure.

Onset of Activity

Observation of different onsets of EMG activity in each channel confirmed that electrodes recorded activity in different muscles (Fig. 10).

Discussion

Strong PFM contraction invariably activated the abominal wall muscles, although patterns varied between, but not within, subjects. TA and OI were recruited predominantly, with little or no activity in RA and OE in all subjects. The only other comparable study of abdominal muscle activity found a significant increase in activity in all abdominal muscles except RA during PFM contraction [11]. Furthermore, their results suggested that recruitment of all the abdominal muscles, except OE, was independent of the position of the lumbar spine.

Attempts to maintain a relaxed abdominal wall during a PFM contraction were unsuccessful and resulted in only 25% of maximum voluntary contraction of the PFM. This suggest that attempts to inhibit abdominal muscle activity may in fact reduce PFM recruitment to such a degree that the requirements of strength training, as for stress incontinent patients, are no longer met. The poor outcomes of some pelvic floor training programs may thus be explained if all concurrent abdominal muscle activity was indeed eliminated [3].

In this study a correctly performed PFM contraction did not result in a marked rise in IAP. A mean rise of 10 mmHg was recorded during a maximum pelvic floor contraction in the supine position. Much higher pressures were recorded during coughing and forced expiration, with strong recruitment of TA and OI. Although these muscles are considered to be important in the generation of IAP, the contribution of the diaphragm should not be overlooked. The recording of almost identical pressures orally with the manometer and intravaginally with the pressure sensor indicates the direct and equal deflection of IAP generation on to the pelvic floor and its associated structures. In all subjects the pelvic floor was recruited during the supine abdominal and belly-in tests, when no specific instruction was given to contract the pelvic floor. This supports the findings of other studies [11,12], suggesting coactivation of the PFM during abdominal muscle exercise. In the supine abdominal test, which strongly activated the abdominal wall, there was 44% more EMG activity in the PFM than during a voluntary maximum PFM contraction. Thus, as Bo and Stein [12] suggest, abdominal exercise may strengthen the PFM in normal subjects. However, parous subjects with PFM pathology should be advised how to avoid a rise in IAP when performing abdominal exercise, to reduce the risk of prolapse or stress urinary incontinence. The results of our study suggest that training TA and the lower fibers of OI may help to recruit the PFM preferentially without producing a marked rise in IAP.

Activity in TA was anticipated during the 'belly-in' test, but concurrent activity was also observed in OI. As there may be fibers of TA lying deep to OI at the site of the OI electrode, it is possible that there was some cross-talk from TA contributing to the EMG activity recorded. However, when the raw data were viewed offline there were clear and different onsets of activity in OI and TA, indicating specific recording from these two muscles (Fig. 10). It must be emphasized that EMG technique only detects muscle activity in the area of the recording electrode. Thus we recorded deep abdominal muscle activity on the lower part of the abdominal wall from OI and possibly TA, and lateral to the umbilicus from TA. It is not possible from the results of this study to comment on activity in other parts of the muscles distant from the electrodes.

A strong cough resulted in recruitment of all abdominal muscle groups, but particularly TA and OI. Forced expiration also produced intense EMG activity in TA and OI. The pelvic floor was moderately recruited in both tests, with similar recruitment patterns noted in both supine and standing positions for both tests. Because loss of PFM reflex activity has been documented in women with SUI, and a voluntary PFM contraction just prior to coughing is effective in reducing urine loss, our results suggest that it would be more appropriate for stress incontinent women to learn specific coactivation of the deep abdominal muscles concurrent with PFM contraction.

The complex interactions of diaphragm, abdominal muscles and pelvic floor have not been described or defined. Investigation of the interaction of these muscles is warranted, as the pelvic floor has to withstand rises in

IAP during many activities, such as coughing, sneezing, jogging, squatting, lifting and defecation. Severe symptoms associated with its dysfunction are prevalent. The role of the PFM during lifting has implications in the workplace and for occupational health and safety.

We would caution against extrapolation of these results and conclusions about the coactivation of abdominal and PF muscles to populations of women with a weak pelvic floor or with stress incontinence or prolapse. In particular, it should not be assumed in these populations that the pelvic floor muscles will be recruited normally during abdominal exercise, coughing or forced expiration, as reflex recruitment of motor units in the pelvic floor may be absent or delayed [13]. Thus inappropriate abdominal exercise, as well as incorrectly performed pelvic floor muscle exercises, may contribute to symptoms of SUI and prolapse in women with PFM weakness.

An incidental finding of this study, when viewing the onset of EMG activity offline, was that the onset of TA and the PFM did not precede the onset of the other abdominal muscles in any subject for any test (Fig. 10). In their study of spinal stability, in which the abdominal muscle activity of normal subjects performing arm movements was investigated, Hodges and Richardson [20] found an involuntary precontraction of TA before the onset of activity in the other abdominal muscles. The pelvic floor muscles were not investigated. We would suggest some caution in extrapolating their results to other conditions of spinal movement until the extent to which this precontraction occurs has been more fully investigated. Furthermore, a precontraction of the PFM with the TA should not be assumed, as no such precontraction was demonstrated in our study.

Our sample was small and limited to young, fit asymptomatic women, so that trends rather than definite conclusions can be drawn from our results. The results challenge the view that the abdominal wall muscles can be considered as a single muscular unit, and suggest that a more specific approach is required for PFM exercise, allowing and possibly training concurrent activity in TA and OI. Most specifically, the results challenge the view that the abdominal wall should be relaxed during PFM exercise. Further studies are warranted to investigate the interactions between abdominal and pelvic floor muscles in larger samples of asymptomatic and symptomatic women.

Conclusion

A strong PFM contraction resulted in strong and simultaneous recruitment of TA and OI but not of OE and RA in all of the four subjects, who were all asymptomatic and nulliparous. The pelvic floor was recruited during abdominal exercises, but this coactivation of the PFM during abdominal exercise and during rises in IAP should not be assumed in parous or symptomatic women.

It was possible to reduce but not eliminate abdominal muscle activity when contracting the PFM. However, attempts to inhibit abdominal muscle activity resulted in low-intensity PFM contractions, which would not satisfy the principles of strength training. Contrary to claims in the literature, a correctly performed PFM contraction resulted in activation of the deep abdominal muscles but did not result in a marked rise in IAP. The small numbers in this study and the sample of fit subjects without incontinence do not allow extrapolation to other populations. Nevertheless, our results challenge the view that the abdomial wall muscles can be considered as a singular unit that should be relaxed during PFM exercise. Further investigation of the interaction between the muscles surrounding the abdominal cavity is needed.

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