

# Levator ani muscle activity in pregnancy and the postpartum period: a myoelectric study

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## Summary

The levator ani (LA) is a muscle of evacuation and acts as well to support the pelvic viscera. An increase of the intra-abdominal pressure beyond the physiologic limits and visceral overload are speculated to interfere with LA functional activity. This consideration was a stimulus to study the effect of pregnancy on the LA muscle.

The EMG activity of the LA muscle was recorded before and during pregnancy and after delivery in 36 women (mean age  $27.2 \pm 3.1$  years, 20 multigravida, 16 primigravida). A needle electrode was inserted into the muscle and LA activity was recorded at rest, and on squeezing and straining in both the erect and recumbent position.

In the erect position, the resting and squeezing EMG activity during the first 8 weeks of pregnancy, showed no significant difference ( $p > 0.05$ ) from that before pregnancy, and after that, increased progressively and significantly until delivery. On straining, the EMG activity showed no significant difference from that before pregnancy in the first 8 weeks, and after that, decreased progressively and significantly till delivery. In the recumbent position, the LA EMG registered similar activity to that in the erect position but with significantly lower values ( $p < 0.05$ ). The reduction in the LA EMG activity was more evident in the multi- than in the primigravida. In the postpartum period, no LA EMG activity was recorded in the first month; the activity increased progressively after that time to reach the pre-pregnancy level in the fourth postpartum month.

In conclusion, pregnancy interferes with EMG and functional activity of the LA from the 8th week onwards due to the progressively increasing size and weight of the uterus. This effect was most marked in the last 12 weeks. Delivery seems to maximally inhibit the LA activity in the first postpartum month. Excessive LA traumatization may eventually lead to levator dysfunction syndrome.

**Key words:** Pelvic floor; Electric waves; Levator dysfunction; Squeeze; Strain; Primi-multigravida.

## Introduction

The pelvic floor muscles include the levator ani (LA) and puborectalis [1]. The LA muscle is funnel-shaped with a transverse part called the levator plate and a vertical one called the suspensory sling [2, 3]. It is connected to the structures passing through the levator hiatus by means of the hiatal ligament [2, 3] (Fig. 1). Upon contraction, the levator plate is elevated and laterally retracted with a resulting pull on the hiatal ligament which pulls open the intra-hiatal structures leading to their evacuation (rectum or bladder; Fig. 2), thus, the LA is a muscle of evacuation. Furthermore, it supports the pelvic viscera and contracts upon straining to oppose any increase in intra-abdominal pressure [4]. The puborectalis is a U-shaped muscle which together with the deep external anal sphincter forms the top loop [5]. It is a muscle of continence.

Under normal physiologic conditions, the main brunt of increase in intra-abdominal pressure caused by straining for any reason is borne by the levator plate and in particular the rectococcygeal raphe which is its most dependent and durable part [3, 6, 7]. The levator hiatus being plugged by the hiatal ligament, is immune to

increased intra-abdominal pressure. The hiatus ligament attachment to the intrahiatal structures (Fig. 2) firmly seals the pelvic floor and prevents intra-abdominal pressure from leaking to the infralevator structures [6, 7].

In pregnancy, the size of the uterus progressively increases and the intra-abdominal pressure (IAP) is elevated [8]. Pregnancy, thus, appears to have an effect on the function and integrity of the LA muscle. The current communication investigates the effect of the gravid uterus and the associated increased IAP on the functional activity of the LA muscle.

## Material and Methods

Thirty-six women (mean age  $27.2 \pm 3.1$  SD years; range 24-32) were investigated after giving an informed consent. Sixteen were primigravida and 20 multigravida with 2-4 previous deliveries. Our Faculty Review Board and Ethics Committee approved the study.

Physical examinations including neurologic assessment as well as laboratory work were normal. The EMG activity of the LA muscle was recorded before pregnancy, every four weeks during the 36 weeks of pregnancy and in the first five months of the postpartum period.

The LA EMG activity was studied by means of a concentric needle electrode using the method previously described [7]. The EMG recordings were done at rest (basal) and on squeeze and straining in the erect and recumbent position.

## Results

No adverse effects were encountered during the study and all the women were evaluated.

**LA EMG activity in the erect position:** The LA EMG activity before and during pregnancy at rest, and on squeezing and straining of the 36 women in the erect position is shown in Figure 3. The resting activity in the first 8 weeks of pregnancy showed no significant difference from that before pregnancy ( $p>0.05$ ) and increased progressively and significantly from the 12th to the 36th week, the highest increase being registered in the last 12 weeks ( $p<0.01$ , Fig. 3). On squeezing, the LA EMG showed an activity similar to that at rest with no significant difference ( $p>0.05$ , Fig. 3). On straining, the LA myoelectric activity exhibited no significant changes from that before pregnancy in the first eight weeks of pregnancy ( $p>0.05$ , Fig. 3). After that time, EMG activity was continuously and significantly decreasing, in particular during the last 12 weeks. The diminished EMG activity was more manifest in the multigravida than in the primigravida and in the women with four previous deliveries than in those with two; however, the difference was not significant ( $p>0.05$ ).

**LA EMG activity in the recumbent position:** Figure 4 shows the LA myoelectric activity in the recumbent position, before and during pregnancy, at rest, and on squeezing and straining. The activity before and during pregnancy recorded significantly lower values ( $p<0.05$ ) than those in the erect position when compared month by month. In the pregnant women the LA EMG activity at rest increased significantly from the 20th week till the 36th, while on squeezing showed no significant change from that at rest ( $p>0.05$ ). On straining, a significant reduction in the LA EMG activity occurred starting from the 12th week of pregnancy, the decrease being more significant in the last 12 weeks. The reduction in EMG activity was more evident in the multigravida than in the primigravida and in the women with four previous deliveries than in those with two.

**Postpartum LA EMG activity:** In the first postpartum month, the EMG recorded no LA activity in the erect or recumbent position, at rest and on squeezing, and an activity on straining which was significantly lower than that

before pregnancy ( $p<0.01$ , Table 1). The LA resting activity returned on the 2nd postpartum month, but was significantly lower than that before pregnancy ( $p<0.01$ ). The activity, at rest and on squeezing and straining, reached the pre-pregnancy level four months postpartum (Table 1).

## Discussion

The current study demonstrated that during pregnancy significant changes occurred in the myoelectric activity of the LA muscle. Normally, the LA has resting electric activity [9]. This activity, in contrast to that of the puborectalis muscle, does not change on squeezing but increases on straining due to muscle contraction [2, 3]. The LA supports the pelvic viscera including the uterus which during pregnancy is progressively growing in size and weight causing increased LA EMG activity. No changes were recorded in the first 8-12 weeks of pregnancy compared to the EMG activity before pregnancy probably due to the fact that neither the size nor the weight of the uterus during this period had increased to the extent to affect the muscle integrity.

Previous and recent studies have shown that the LA muscle consists of type 1 (slow-twitch) and type 2 (fast-twitch) muscle fibers [10, 11]. The type 1 fibers are probably activated during an increase of the intra-abdominal pressure. They seem to be responsible for maintaining the LA tone and the associated resting myoelectric activity, which probably have the function to support the weight of the pelvic viscera.

The significant increase in the resting LA EMG activity after the first 8-12 weeks of pregnancy is suggested to guard the LA against, and to enable it to cope with the overload effected by the continuous increase in weight and size of the uterus – a kind of defense mechanism to prevent muscle subluxation and sagging.

Before pregnancy the LA EMG exhibited a higher activity in the erect than in the recumbent position due probably to the extra load thrown on the LA by the viscera in the erect position. Likewise was the effect of pregnancy on the resting EMG activity of the LA more manifest in the erect than in the recumbent position.

On straining, the diminished LA EMG activity in comparison to the pre-pregnancy levels presumably denotes a decrease in the contractile power of the muscle. This

Table 1. — Postpartum LA EMG activity in the erect and recumbent position, at rest, and during squeezing and straining\*.

POST-partum month	Potentials ( $\mu V$ )											
	Erect						Recumbent					
	REST		SQUEEZING		STRAINING		REST		SQUEEZING		STRAINING	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range
1	0	0	0	0	214.6 $\pm$ 38.2	160-248	0	0	0	0	196.4 $\pm$ 25.3	122-220
2	43.2 $\pm$ 8.4	24-56	44.6 $\pm$ 8.8	26-60	362 $\pm$ 36.4	283-394	32.2 $\pm$ 7.5	18-42	33.1 $\pm$ 7.6	18-44	318.3 $\pm$ 30.2	266-338
3	84.3 $\pm$ 16.6	48-94	82.3 $\pm$ 15.9	50-92	442 $\pm$ 39.3	364-492	62.4 $\pm$ 13.6	40-78	66.4 $\pm$ 14.2	43-80	408.8 $\pm$ 40.6	312-442
4	116.6 $\pm$ 21.3	78-128	118.5 $\pm$ 22.1	80-122	598.3 $\pm$ 43.1	482-642	94.5 $\pm$ 18.6	68-116	98.3 $\pm$ 17.5	70-118	512.6 $\pm$ 44.2	413-584
5	123.3 $\pm$ 28.5	88-139	121.6 $\pm$ 27.9	92-142	623.6 $\pm$ 53.3	582-674	98.6 $\pm$ 21.5	74-124	100.2 $\pm$ 22.2	77-130	556.2 $\pm$ 50.1	473-612

\* Values were given as mean  $\pm$  SD

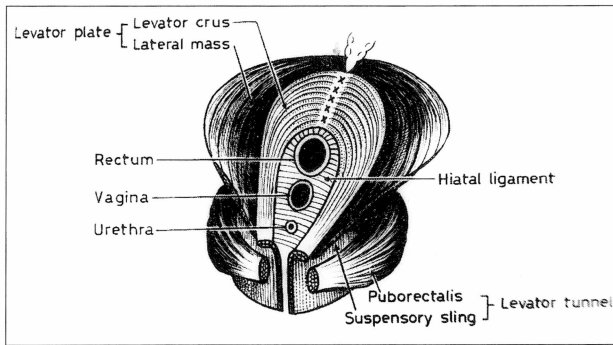


Figure 1. — Diagram illustrating the levator plate, levator hiatus, intra-hiatal structures as well as the hiatal ligament (from Shafik [3]).

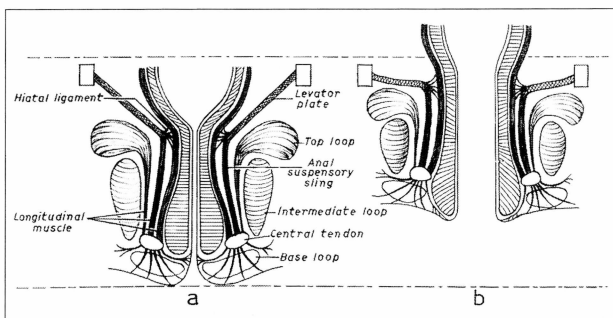


Figure 2. — Diagram illustrating the mechanism of rectal evacuation (defecation) upon levator muscle contraction (from Shafik [3]).

a) Levator muscle at rest.

b) On levator contraction, the levator plate becomes elevated and laterally retracted and pulls on the hiatal ligament, which pulls open the anorectal junction. Contraction of the suspensory sling of the levator muscle leads to widening and shortening of the anal canal.

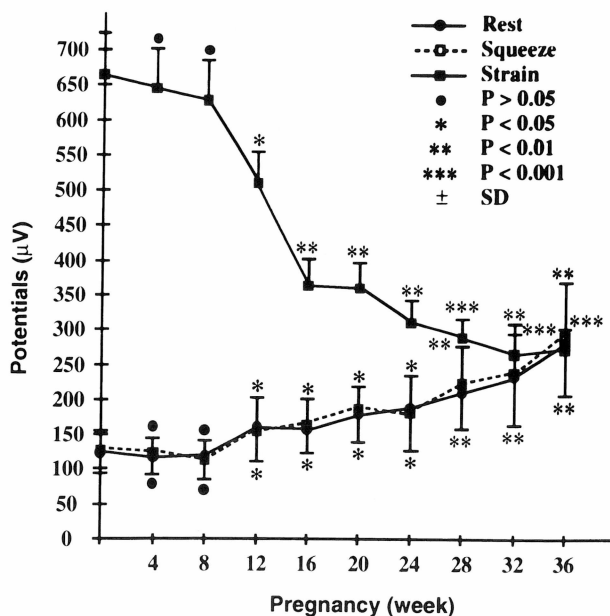


Figure 3. — The levator ani EMG activity during pregnancy in the erect position, at rest, and during squeezing and straining.

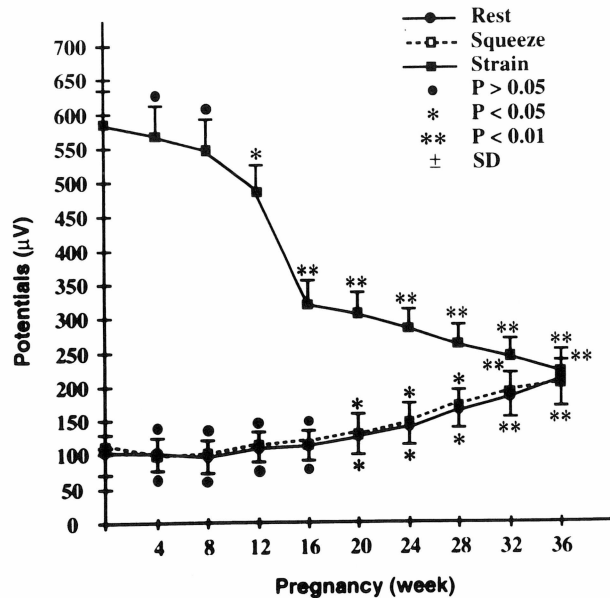


Figure 4. — The levator ani EMG activity during pregnancy in the recumbent position, at rest, and during squeezing and straining.

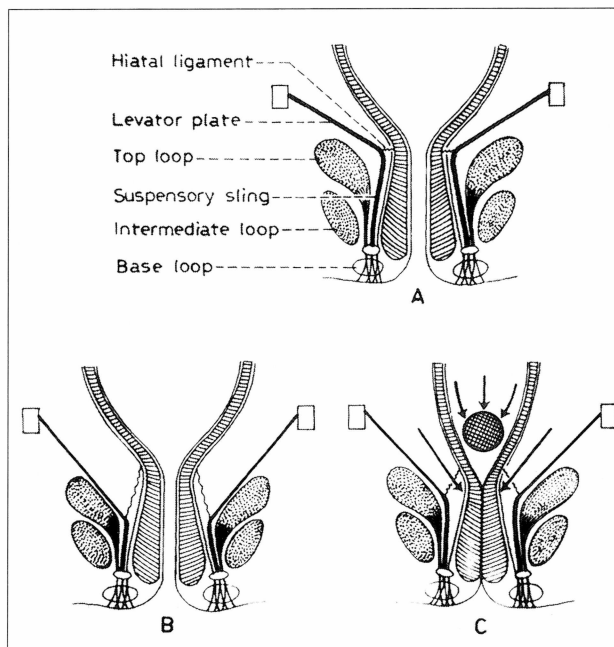


Figure 5. — The levator ani muscle in the levator dysfunction syndrome (from Shafik [6]).

a) Normal findings

b) Pathological findings in levator dysfunction syndrome: sagging of the levator plate as well as hiatal ligament and suspensory sling subluxation. Levator hiatus is widened and lowered so as to expose the anal canal to the intra-abdominal pressure.

c) Levator dysfunction during straining: on straining, the sagging levator plate and the subluxated suspensory sling are too weak to effect opening of the intra-hiatal structures (anal canal and vesical neck). The increased intra-abdominal pressure leaks through the abnormally wide levator hiatus, to the intra-hiatal structures leading to their obstruction.

diminished activity became more evident with advancement of pregnancy. The LA muscle contracts on straining [2, 3]. It is postulated that the diminished LA contraction is due to the increasing weight of the gravid uterus imposing an overload on the levator plate and inhibiting its contractile power. Furthermore, the size of the gravid uterus seems to interfere mechanically with the LA contractile activity. The LA muscle in the last 12 weeks of pregnancy is most likely overstretched and mechanically obstructed by the huge-sized and heavy gravid uterus so that efficient muscle contraction on straining cannot be achieved.

**LA EMG activity in the postpartum period:** The absence of the LA EMG activity, at rest and on squeezing in the erect and recumbent position, in the first postpartum month, is presumably due not only to the trauma induced directly to the LA muscle by the fetus during delivery, but also to the excessive dilation of the levator hiatus. On straining, the muscle showed EMG activity, which was, however, significantly below that before pregnancy. The LA activity returned in the 2<sup>nd</sup> postpartum month and increased gradually until it reached the pre-pregnancy level in the 4th postpartum month. It seems that during the 4-month period, the LA muscle traumatization disappeared and the muscle regained its pre-pregnancy integrity.

**Levator dysfunction syndrome:** It is postulated that excessive LA muscle traumatization during delivery by, for example, a prolonged 2<sup>nd</sup> stage or the application of a forceps, might induce irreversible LA damage. The main brunt of injury seems to fall on the levator muscle bundles, rectococcygeal raphe and the hiatal ligament [2, 6]. The latter two structures, both being tendinous, become overstretched and subluxated. The levator plate may sag down and appears to acquire a vertically oblique position (Fig. 5). The levator hiatus, consequently, is overwidened and lowered so that most of the intrahiatal structures lie above it and are exposed to the direct effect of the intra-abdominal pressure with its deleterious effects [3, 6] (Fig. 5). The levator dysfunction syndrome eventually occurs [3, 6].

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