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Original Article

A Study of the Effect of Cutaneous Electric Stimulation on the Structure of The Anal Sphincter of Rat After Induction of Anal Muscle Injury

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ABSTRACT

Background: It is well known that the muscles of the anus perform a critical role in maintaining continence. Any defect in their structure can negatively affect the physiological control of feces. Anorectal electro-stimulation, proved to improve the anal musculature. The exact effect remains unclear

Aim of the work: The present work aimed to investigate the effect of cutaneous electrical stimulation upon regeneration of the injured anal sphincter in a rat.

Material and Methods: 30 adult female albino rats were divided into four groups. Group I (12 rats): control group (IA) and sham group (IB). The other 3 groups (6 rats each) were subjected to anal sphincter injury. Group II: rats were subjected to anal sphincter injury. Group III: rats were left for spontaneous healing for 3 weeks. Group IV: received cutaneous anal electric stimulation for three weeks. At the end of the determined period, rats were sacrificed, specimens from the lower part of anal canal were processed for paraffin and semithin sections and stained with Haematoxylin and Eosin, Masson's trichrome, Toluidine blue and immune-histochemical stains (anti-desmin and anti alpha-SMA- antibodies).

Results: Group II showed marked affection of both external and internal anal muscles. Group III showed disorganization of normal histological architecture of both anal muscles. On the other hand, group IV revealed findings indicating regeneration of the muscle fibers.

Conclusion: The use of the cutaneous electric stimulation proved to be useful in muscle healing with regaining almost the normal architecture of the injured external and internal anal muscles.

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Key Words: Anal sphincter, electric stimulation, injury.

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INTRODUCTION

It has been known that continence is maintained by the muscles of the anus, which is its critical role. The physiological control of feces can be negatively affected if any defect in their structure occurs.

Fecal incontinence is one of the most socially and psychologically devastating conditions in a healthy individual. It can lead to social isolation and loss of self-confidence.[1] True anal incontinence is the loss of the anal sphincter control leading to unwanted passage of feces or gas. This must be distinguished from other conditions that can lead to stool passing through anus such as hemorrhoids, enlarged skin tags and rectal mucosal prolapse.[2]

Vaginal delivery is accepted as the most common predisposing factor to fecal incontinence in young and healthy women.[3]

Review

Histology of the anal canal

The anal canal consists of an inner layer (mucosa), submucosa, anal sphincters (internal and external), and supporting fibromuscular tissue, in addition to dense neuronal networks (autonomic and somatic).[4]

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Enck *et al*., (2005)[5] described section through the anal canal that shows the following layers: the mucosa (about 0.5–1 mm), circularly arranged smooth fibers forming the internal anal sphincter IAS (about 0.5–1 mm), a layer of smooth fibers running in a longitudinal direction (longitudinal muscle LAM, about 1 mm); all the above three layers (mucosa, IAS & LAM) continue above as the layers of the ampulla of rectum. The external anal sphincter (EAS)) forms the outermost thick layer of striated muscle fibers (between 1 and 3 mm). Therefore, the anal canal is surrounded by internal and external anal sphincters, which are essential in the maintenance of fecal continence.

The mucosa:

The mucosa of the anal canal is organized into longitudinal folds, known as anal columns. These are joined together at their inferior ends by anal valves. Above the anal valves are small pouches which are called anal sinuses – these contain glands that secrete mucus.

All the anal valves together form an irregular circle – known as the pectinate line (or dentate line).[6]

Agarwal, (2012)[7] described the anal columns and sinuses as a structure resembling an umbrella or an accordion wall. This structural feature might play a role in smooth expansion of the anal canal without overstretching the epithelium for defecation as well as for continence of feces and gas.

According to the character of the epithelial lining, the anal canal is divided into three zones; the colorectal zone, the anal transitional zone and the squamous zone.

The submucosa:

Agarwal, (2012)[7] mentioned that the terminal branches of the superior rectal artery and the rectal venous plexus are present in the submucosa of the anal columns. Any increase in the venous pressure in the portal circulation (portal hypertension) results in enlargement of these submucosal veins forming internal hemorrhoids.

Muscles of the anal canal:

It has been known that the continence is maintained by the muscles of the anus, which is its critical role. Knowing that the physiological control of feces can be negatively affected if any defect in their structure occurs.[8]

External anal sphincter

The external anal sphincter (EAS) was described as a band of striated muscle that surrounding the anal canal in its lowest part. The area of pigmentation of skin around the anal verge corresponds approximately to the extent of the external anal sphincter. Rociu *et al*., (2000)[9] added that the external anal sphincter formed the main bulk of the anal sphincter complex. It is a striated muscle oval tube , composed mostly of type I slow twitch muscle fibers modified for prolonged contraction.

The uppermost (deepest) fibers intermingled with the lowermost fibers of puborectalis. Anteriorly, some fibers decussated into the superficial transversus perineal muscles. Posteriorly, fibers were fixed to the anococcygeal raphe. The majorities of the middle fibers of the external anal sphincter surrounded the the internal sphincter in its lower part and anteriorly they were attached to the perineal body, and to the coccyx posteriorly through the anococcygeal ligament. Some fibers from the sides of the sphincter decussated in midline to form the anterior and posterior commissure.[10]

A subcutaneous portion encircled the anal verge and forms the radial skin creases surrounding the anus. The area of the lower fibers lied below the internal sphincter and is separated by the submucosa from anal epithelium in the lowest part.[11]

The conjoint longitudinal anal muscle

The longitudinal anal muscle has been termed as a vertical layer of muscular tissue interposed between the internal and external anal sphincters. It was augmented in its upper part by striated muscle fibers from the medial aspect of levator ani. The muscle is particularly prominent in the fetus, where it is actually thicker than the internal anal sphincter.[12]

Macchi *et al*., (2008)[13] reported that with advancing age, there was gradual replacement of muscle by connective tissue, such that the layer became thin in the elderly and few muscle fibers were seen in its distal part.

Internal anal sphincter

The internal anal sphincter (IAS) is a muscular ring that encircled about 2.5 – 4.0 cm of the anal canal. The internal sphincter is not under voluntary

control. It is considered as the specialized, white, thickened terminal part of the inner circular muscle of the large intestine. It is formed by an aggregation of the involuntary circular fibers of the rectum.[14] An important morphological feature of the circular muscle layer of the IAS that it is divided into separate bundles separated by connective tissue septa which have been referred to as "minibundles." In rats, minibundles extend from the myenteric to the submucosal surface of the circular muscle layer with the entire width of the IAS consisting of about 5–8 minibundles[15]. It starts at the anorectal junction and ends above the anal verge; its lower border is palpable at the intersphincteric groove which is about 6 mm from the orifice of the anus, and which corresponds to the proximal limit of the subcutaneous part of the external anal sphincter. It is traversed by fibers passing medially from the conjoint longitudinal muscle into the submucosa.[16]

At rest, it was normally contracted but it relaxed as a consequence of a reflex activity, predominantly during defecation. Transient relaxation of the upper internal anal sphincter occurred in response to rectal distension (the recto-anal inhibitory reflex) and postprandial rectal contractions (the sampling reflex).[17]

Bharucha, (2006)[18] stated that the relaxation of the IAS allowed the passage of distal rectal contents into the upper anal canal, enabling a conscious perception of their physical nature; this was accompanied by sustained contraction of the distal internal anal sphincter and contraction of the external anal sphincter to sustain continence. The recto-anal inhibitory reflex was primarily mediated by the enteric nervous system, although spinal pathways might have a modulatory role.[1]

Intersphincteric space and anal glands

The intersphincteric 'space' is a potential space which lied between the conjoint longitudinal muscle layer and the external anal sphincter. It could be entered surgically to offer entrance to many operations (e.g. intersphincteric excision of the rectum and intersphincteric approach to fistulae).[19] Within this space lied the intersphincteric anal glands.

Anal incontinence is known to have important social and economic influence and significantly impairs quality of life.[20] The emotional consequences of anal incontinence often go beyond the physical manifestations. Many individuals reported withdrawing from their social lives and hide the problem from their families, friends, and even their doctors. This has led to difficulties for healthcare providers in identifying those affected by anal incontinence.[21]

Fecal incontinence is an unintentional loss of solid or liquid stool. True anal incontinence was the loss of the anal sphincter control leading to unwanted passage of feces or gas.[20] This was important to be distinguished from other conditions that can lead to loss of stool control from anus such as hemorrhoids, enlarged skin tags and rectal mucosal prolapse.[22]

Prevalence of fecal incontinence in the general population is 7 -15%.[23] This prevalence of fecal incontinence increases with age. Rate of fecal incontinence in women is as many as 13-25% after vaginal delivery.[24]

According to Pretlove *et al*., (2008)[3] the sphincteric causes of fecal incontinence might be structural (disruption or atrophy of part of the sphincter musculature) or neuropathic (damage to the nerve supply to the sphincters), or a combination of both. Obstetric anal sphincter injury was the most common causes of sphincter disruption, followed by the anal surgery (for haemorrhoids, fistula or fissure) and trauma.

Cutaneous electrical stimulation

The cutaneous electrical stimulation was defined as the skin having transcutaneous electrodes attached to it on the surface, which has the current transferred between the device and the muscle.[25]

From the literature, cutaneous electrical stimulation perform a main role in muscle function, as specified by numerous issued work. Transcutaneous electrical stimulation is becoming a vital tool of rehabilitation to patients for control of muscle function and mobility.[26]

Healing process that occurs normally without the interference of electrical stimulation usually might take more time. Currently with the help of this technique an injury could resolve earlier.[25]

Some of the important parameters that is essential to undertake evaluation in using the cutaneous electrical stimulation, are the pulse width, the frequency, the ramp period, the duty cycle and amplitude.[27]

The use of the cutaneous electrical stimulation as a muscle atrophy rehabilitation intervention, had been discussed in various research works. Evidence was proved that it could promote and develop muscle bulk alongside the continuous therapy.[28]

MATERIAL AND METHODS

30 adult female albino rats (weighing 180-250 grams) were divided into four groups.

Group (I): Twelve female adult rats were subdivided into two subgroups six in each.

IA: control group.

IB: sham group.

Group (II): Six adult female rats were subjected to induction of anal sphincter injury, and then histological specimens were taken after three days.

Group (III): Six adult female rats were subjected to induction of anal sphincter injury and were left for spontaneous healing for three weeks.

Group (IV): Six adult female rats were subjected to induction of anal sphincter injury and then received five sessions per week of cutaneous anal electric stimulation for three weeks.[29]

Induction of the anal sphincter injury:

The rats were put in the lithotomy position, and then the skin of the anal orifices was incised transversely at 9 o'clock position. Deepening of the lesion was done from the skin inwards along the whole thickness of the anal sphincters without interrupting the mucosa (a forceps were kept inside the lumen of the anal canal while doing the injury, to keep it opened to see the intact muscosa).[30]

Cutaneous ano-rectal electric stimulation:

The animals were subjected to one electro-stimulation session per day, five days a week, for three successive weeks with a total of fifteen sessions. Each session included two electrical stimulations for the animals having a rest for five minutes between them.[29] This was done using alpha – wave Healthtronic muscle stimulator (model BM- 1006 – DC- 6 V batteries, 0.6 W, serial no. 0001171, CE) supplied from the physiology department.[31]

By the end of the experimental duration, all animals were sacrificed. Extraction of the anal canal was done. Samples were taken and prepared for light microscopic study stained with Haematoxylin and Eosin, Masson's trichrome, Toluidine blue and immuno-histochemical stains(anti-desmin and anti alpha-SMA-antibodies) .

RESULTS

Light microscopic examination of the anal canal group I showed normal arrangement of all layers of the anal canal. The first layer, the epithelium consisted of three zones. The second layer was the submucosal layer with the anal glands. The third and outer layer was the musculosa formed of internal anal muscle, conjoint longitudinal anal muscle and external anal muscle arranged from inside outwards (Fig. 1). Examination of sections stained with anti-desmin and anti-alpha-SMA - antibodies revealed positive reaction in external and internal anal muscles respectively (Fig. 2).

Examination of anal canal sections of group (II) showed both external and internal anal muscles with marked infiltration by inflammatory cells and marked interstitial hemorrhage and cell deposition in between the muscle fibers at the site of induced injury, as well as congested blood vessels around the site of the lesion (Figs: 3, 4, 5, 6, 7 & 8). Vacuolations were evident in between the muscle fibers (Fig. 5). Examination of sections stained with anti-desmin antibody (Fig. 9) and anti-alpha SMA antibody (Fig. 10) of group (II) showed negative reaction at the site of the induced injury in the external anal muscle.

Examination of anal canal sections of group (III) which was left for spontaneous healing showed disorganization of the normal histological architecture of both anal muscles fibers. The internal anal muscle revealed inflammatory cellular infiltration, while the external anal muscles showed wavy fiber direction and loss of their striations (Figs. 11, 12 & 13). The nuclei of both muscles appeared either dense or vacuolated which indicate degenerative changes in the muscle fibers (Fig. 13). Semithin

sections stained with toluidine blue revealed few satellite cells seen along the muscle fibers (Figs. 14 & 15). Masson stained sections showed increased amount of collagen tissue present in between the anal musculature when compared to the control group (Fig. 16).

Examination of sections stained with anti-desmin antibody and anti-alpha SMA antibody of that group showed large areas giving negative immunoreactivity among EAS and IAS respectively (Figs. 17 & 18).

On the other hand, group IV revealed findings indicating regeneration of the muscle fibers as noticed by the regularly arranged fibers of both the external and internal muscles (Fig. 19). Most of the external anal muscle fibers regained their striated appearance, and their regenerating nuclei started to take a peripheral position. Few fibers were still splitted and wavy (Fig. 20). The nuclei of the internal muscle fibers appeared vesiculated and also regained a normal position (Figs. 20 & 21). Many satellite cells were seen along the anal muscle in the semithin sections stained with toluidine blue (Figs. 22 & 23). Masson's stained sections showed decrease in the amount of collagen tissue present in between the anal musculature when compared to group III. Few areas appeared devoid from muscle fibers (Fig. 24). Sections stained with anti-desmin antibody and anti-alpha SMA antibody of group (IV) revealed normal positive reaction in both EAS and IAS respectively (Figs. 25 & 26).

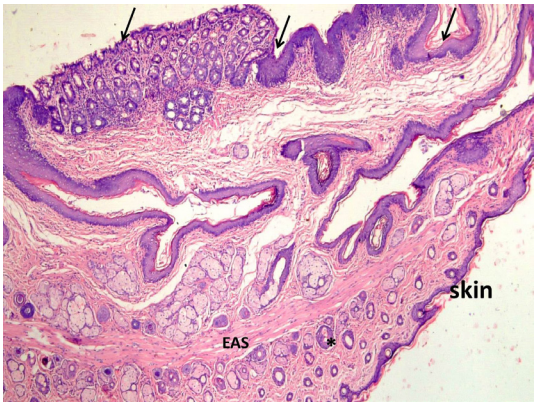

Fig. 1: A photomicrograph of a paraffin section of the anal canal of albino rat from the control group showing normal histological structure of the anal canal which consists of first layer: anal mucosa with its characteristic three zones (↑), underlying the second layer: submucosa and third layer: anal canal musculosa (EAS) extending towards the skin of the anus. Hx & E X40.

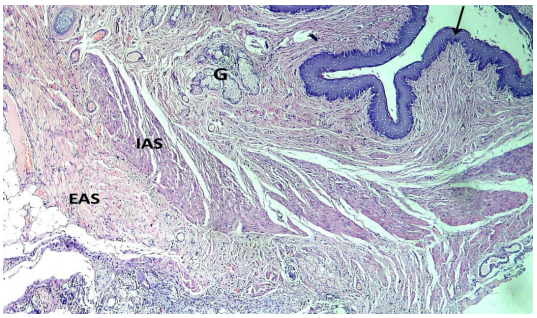

Fig. 2: A photomicrograph of a paraffin section of the anal canal of albino rat from the sham group (Ib) showing normal histological arrangement of the anal canal structures from inside outwards; first layer: anal canal mucosa (↑), followed by the second layer (submucosa) with the presence of the anal glands (G), and finally the third layer (anal canal musculosa) containing the internal anal sphincter (IAS) and external anal sphincter (EAS). Hx & E X40.

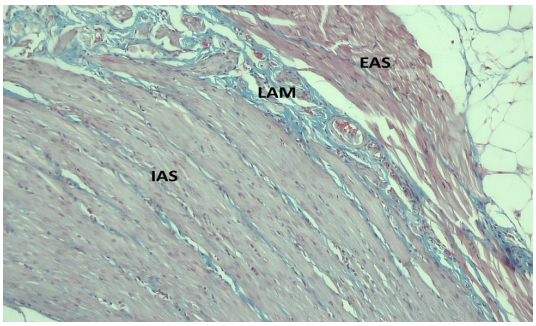

Fig. 3: A photomicrograph of a paraffin section of the anal canal of albino rat from the control group showing the usual histological appearance of collagen tissue in the anal musculature. IAS: internal anal sphincter, LAM: longitudinal anal muscle, EAS: external anal sphincter. Masson's trichrome X100.

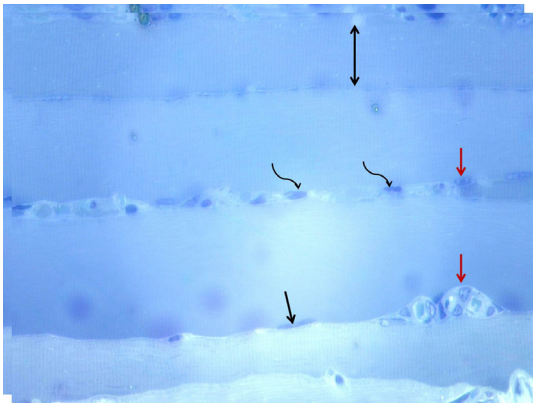

Fig. 4: A photomicrograph of a semithin section of the anal canal of albino rat from the control group showing the external anal muscle with apparent striation (double head arrow). The muscle fibers appear with multiple elongated nuclei situated at the periphery (black arrow). Note the presence of the satellite cells (red arrow) and the fibroblasts (zigzag arrow). Toluidine blue X400.

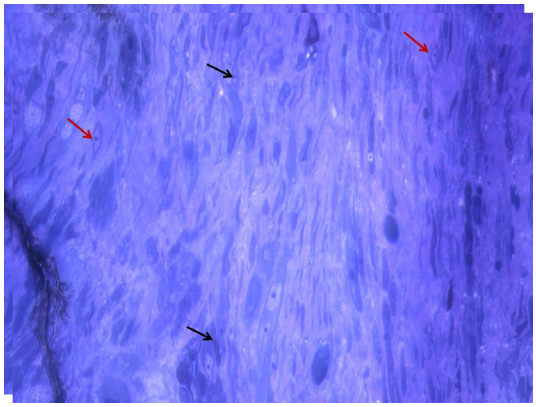

Fig. 5: A photomicrograph of a semithin section of the anal canal of albino rat from the control group showing the internal anal muscle fibers closely packed together and passing in one direction with central, flat and elongated nuclei (↑). Note the presence of the satellite cells along the muscle fibers (↑). Toluidine blue X400.

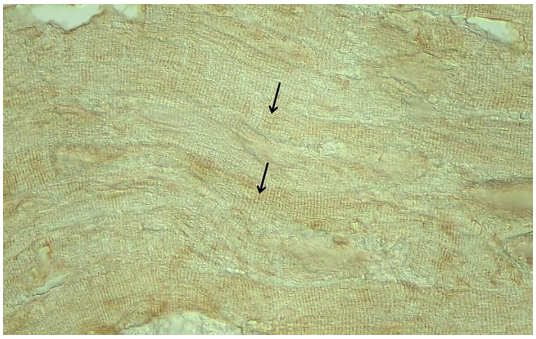

Fig. 6: A photomicrograph of the anal canal of albino rat from the control group showing a positive reaction of the external anal muscle after staining with desmin antibody (↑). Immune staining with with anti-desmin antibody X400.

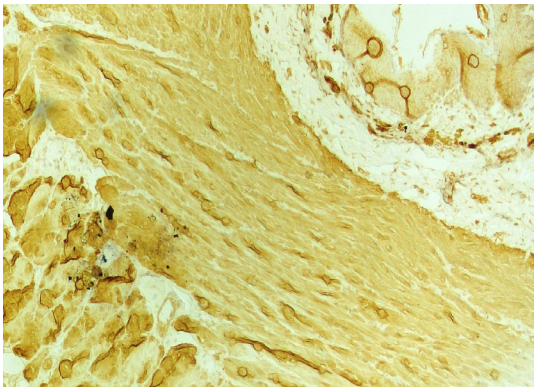

Fig. 7: A photomicrograph of the anal canal of albino rat from the control group showing a positive reaction of the internal anal muscle against alpha SMA antibody. Immune staining with with anti-alpha SMA antibody X400.

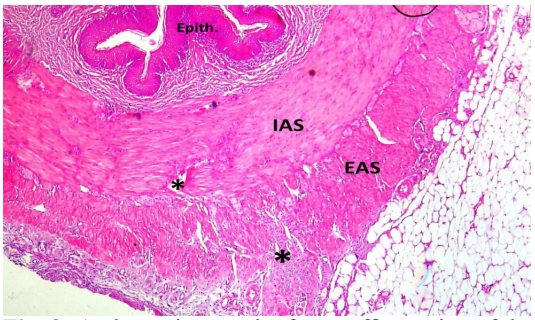

Fig. 8: A photomicrograph of a paraffin section of the anal canal of albino rat from group II showing normal epithelial layer. Inflammatory cells (*) traverse the lesion of internal anal sphincter (IAS) and external anal sphincter (EAS). Hx & E X40

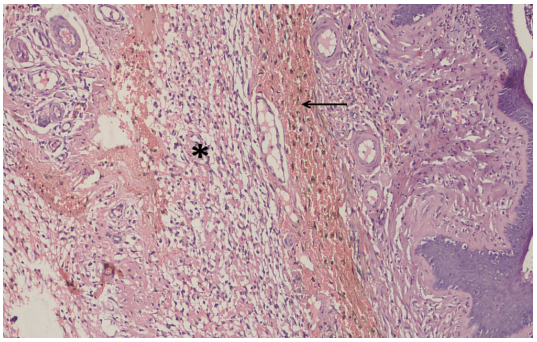

Fig. 9: A photomicrograph of a paraffin section of the anal canal of albino rat from group II showing massive hemorrhage inside the anal musculature (↑) with infiltrating inflammatory cells (*). Hx & E X100.

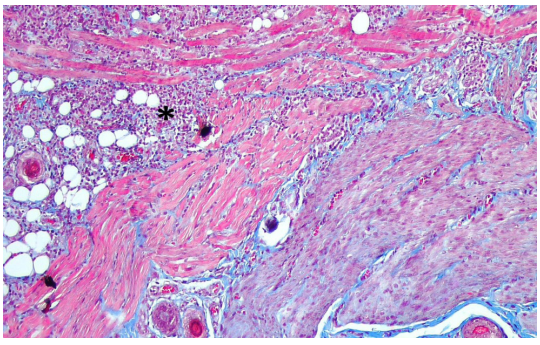

Fig. 10: A photomicrograph of a paraffin section of the anal canal of albino rat from group II showing inflammatory cells infiltrations and vacuolation at the site of the lesion (*) with minimal increase in the amount of the collagen tissue in between the anal musculature. Masson's trichrome X100.

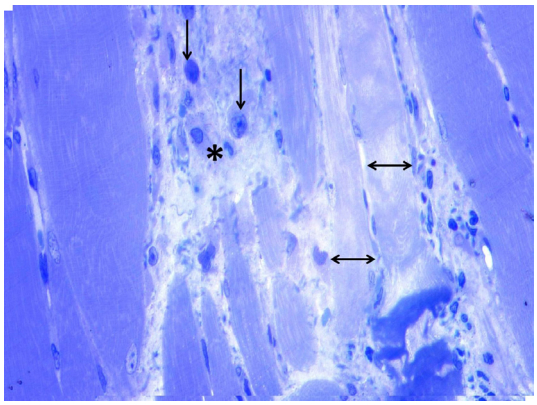

Fig. 11: A photomicrograph of a semithin section of the anal canal of albino rat from group II showing disrupted external anal muscle (*). Some muscle fibers show loss of striation (↔) Inflammatory cells infiltration is present at the site of lesion (↑). Toluidine blue X400.

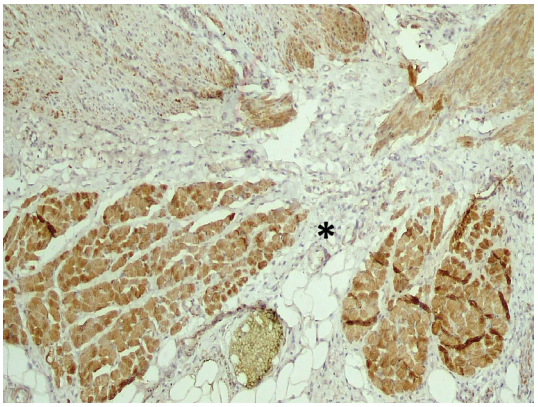

Fig. 12: A photomicrograph of the anal canal of albino rat from group (II) showing a negative reaction to the anti-desmin antibody at the site of the injury in the anal sphincters (*). Immune staining with anti-desmin antibody, X100.

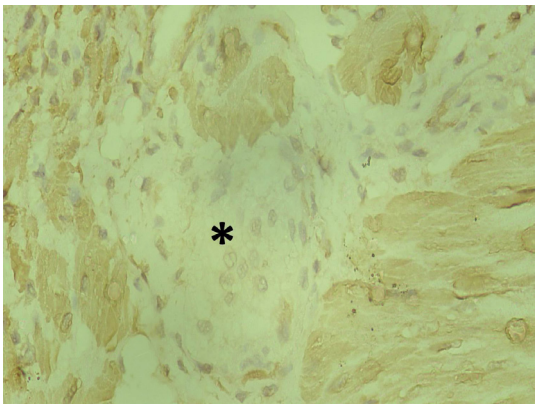

Fig. 13: A photomicrograph of the anal canal of albino rat from group (II) showing negative immunoreactivity of anti – alpha SMA antibody stain at the site of the induced injury (*). Immune staining with anti- alpha SMA antibody, X400.

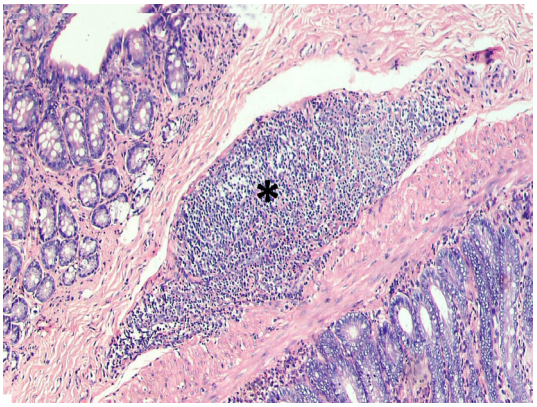

Fig. 14: A photomicrograph of a paraffin section of the anal canal of albino rat from group III showing a localized collection of inflammatory cells (*) present in between the muscle fibers. Hx & E X100.

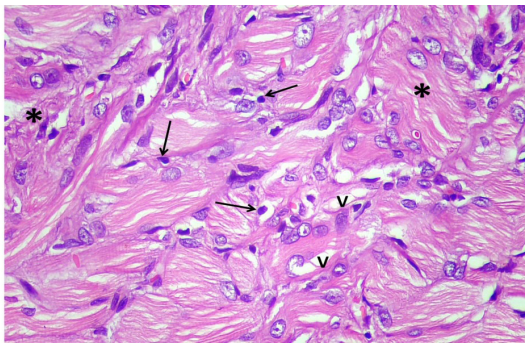

Fig. 15: A photomicrograph of a paraffin section of the anal canal of albino rat from group III showing disorganized muscle fibers that appeared splitted and wavy (*). Note the dense, deeply stained nuclei (↑). Note also the vacuolations between the muscle fibers (V). Hx & E X400.

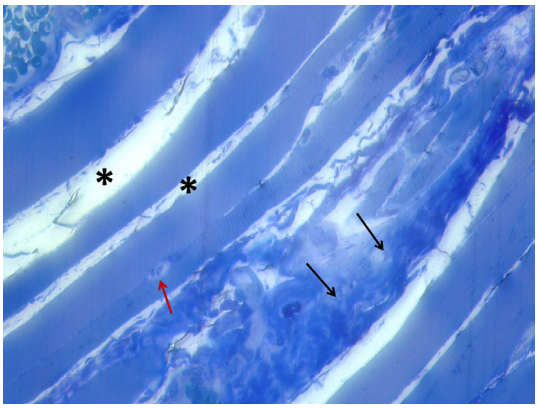

Fig. 16: A photomicrograph of a semithin section of the anal canal of albino rat from group III showing separation in between skeletal muscle fibers (*). Some fibers appeared raged (↑). Note the satellite cells (↑). Toluidine blue X400.

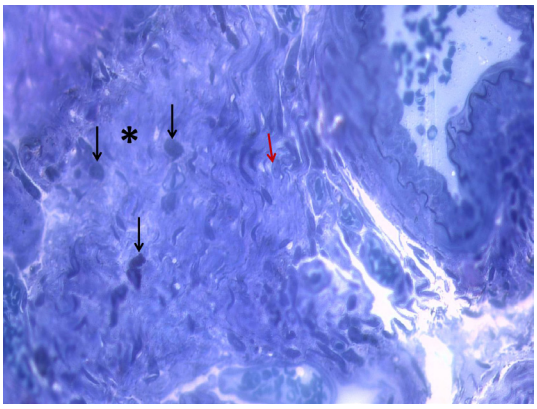

Fig. 17: A photomicrograph of a semithin section of the anal canal of albino rat from group III showing the internal anal muscle with large degenerated area (*). The nuclei appeared irregular in shape and degenerated (↑). Note the satellite cell (↑). Toluidine blue X400.

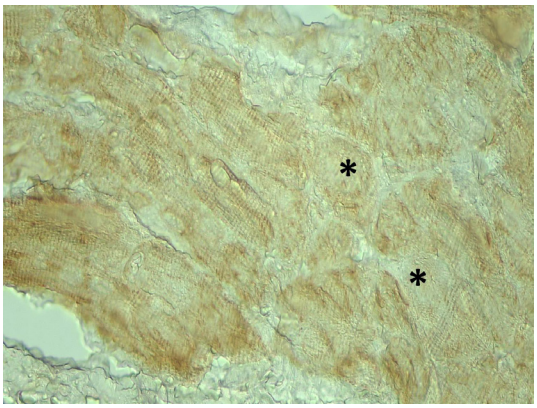

Fig. 18: A photomicrograph of the anal canal of albino rat from group (III) showing negative immunoreactivity to staining with desmin antibody in the external anal muscle. Loss of architecture and striations in some areas is evident (*). Immune staining with anti-desmin antibody, X400.

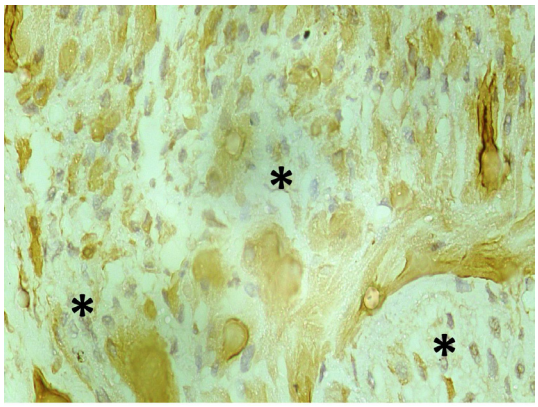

Fig. 19: A photomicrograph of the anal canal of albino rat from group (III) showing a large areas in the internal anal muscle with negative immunoreactivity to anti-alpha SMA antibody (*). Immune staining with anti- alpha SMA antibody, X400.

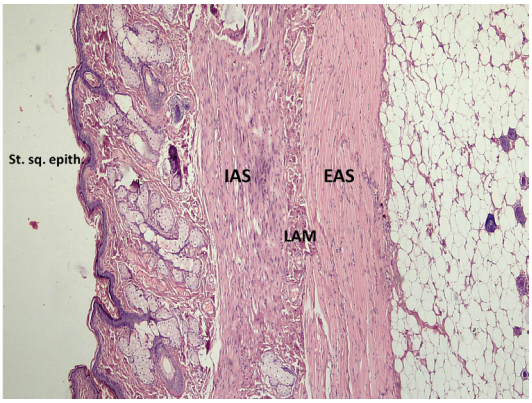

Fig. 20: A photomicrograph of a paraffin section of the anal canal of albino rat from group IV showing normal epithelial layer of the anal canal (st. sq. epith.). The three muscle layers appeared uniform and continuous. The internal anal muscle (IAS) is seen outside the submucosa followed by the longitudinal muscle layer (LAM). The outermost layer is the external anal muscle layer (EAS). Hx & E X40.

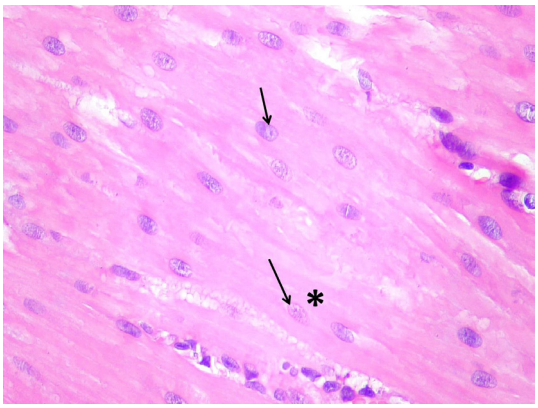

Fig. 21: A photomicrograph of a paraffin section of the anal canal of albino rat from group IV showing the internal anal muscle fibers with vesiculated nuclei and prominent nucleoli (↑). The cytoplasm of the regenerated fibers appeared pale (less basophilia) (*). Hx & E X400.

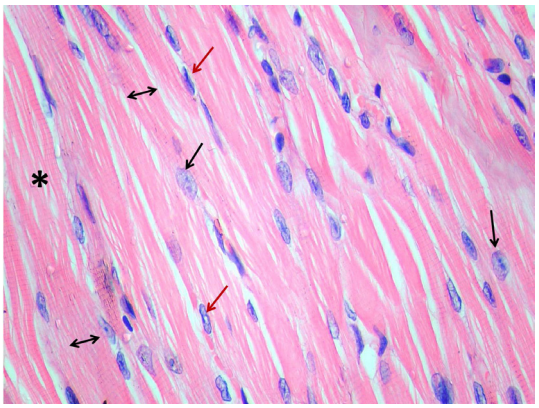

Fig. 22: A photomicrograph of a paraffin section of the anal canal of albino rat from group IV showing external anal muscle with vesiculated nuclei and prominent nucleoli (↑). The nuclei started to take peripheral position (↑). Muscle striations appeared in most of the fibers (↔). Note the presence of some splitted and wavy fibers (*). Hx & E X400.

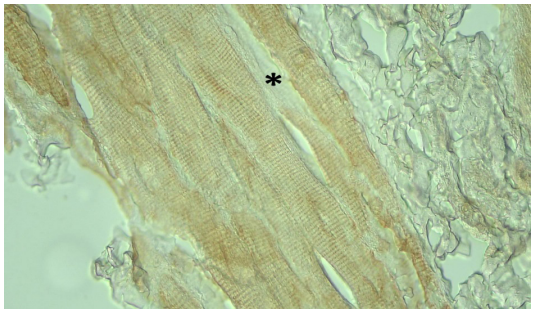

Fig. 23: A photomicrograph of the anal canal of albino rat from group IV showing positive reaction of the external anal muscle against desmin antibody. Note there is loss of striation in few fibers (*).

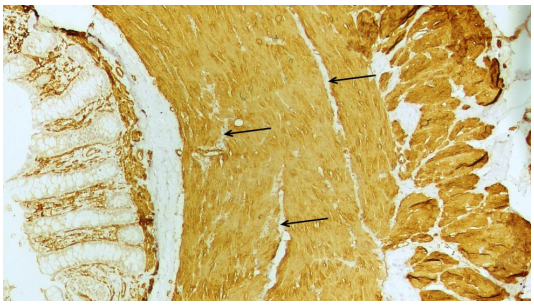

Fig. 24: A photomicrograph of the anal canal of albino rat from group IV showing positive reaction of the internal anal muscle against anti-alpha SMA antibody (*). Note there is some spacing in between the muscle fibers (↑). Immune staining with with anti- alpha SMA antibody, X400.

DISCUSSION

Fecal incontinence is a debilitating clinical symptom resulting from disruption of the anal sphincter complex. It has been known that the continence is maintained by the muscles of the anus, which is its critical role. Knowing that the physiological control of feces can be negatively affected if any defect in their structure occurs. Rate of fecal incontinence in women is as many as 13-25% after vaginal delivery.[24]

Restoring the anatomical and the physiological function is not easily achieved by treatment options. Therefore, induction of new treatment modalities can help in regaining the normal anatomical and physiological functions of the injured muscle and reducing fibrosis and scar tissue formation which can affect greatly the efficacy of the muscle contraction.[32]

The most recent non-invasive treatment is the anorectal electro-stimulation, which has been shown in different studies for local sensitivity enhancement, the defecation reflex control, and the rise in the pressure of the anal canal.[33]

Therefore the present work was designed to detect the effect of cutaneous electrical stimulation upon regeneration of the anal sphincter subjected to injury; that injury was done in a way to resemble the external and internal anal muscles injury resulting from a normal vaginal delivery in women.

In the present study the rats which were subjected to anal sphincter injury and examined after the injury directly (group II), showed huge interstitial hemorrhage along the muscle fibers at the site of the lesion. This could result from destruction of the muscular and vascular tissues induced by the injury.[34]

Again, polymorphic mononuclear inflammatory cells infiltration was found at the site of the induced lesion among this group (II). According to James (2005)[35] muscle injury starts a rapid and sequential invasion of the muscle by a population of inflammatory cells that can persist for several days up to weeks.

Neutrophils are the first invaders of the muscle after injury. They can stay in elevated concentrations for periods that can reach 5 days[36]. Neutrophils can share in the events following injury in two ways: (i) the invading neutrophils have a phagocytic function, clearing any necrotic debris from the site of the injury[37]; and (ii) they augment the process of inflammation through the release of pro-inflammatory cytokines such as IL-6 and TNFα.[38,39]

Although the neutrophil invasion and phagocytosis are important mechanisms in the early inflammatory response to muscle injury, they can cause more damage to the injured muscle, and may result in damage to the healthy surrounding tissue.[40]

In the current work, examination of the anal canal muscles of rats of group III (after induction of injury and left for spontaneous healing), revealed signs indicating the occurrence of degenerative changes. There were loss of striations, vacuolations around nuclei, dense and rounded nuclei. Also, mononuclear cellular infiltration was observed in between the muscle fibers and in the perivascular areas.

These signs of degeneration which were found among this group were similar with Bigard and Fink (2010)[41] who stated that an inflammatory degeneration might happen with the removal of all traces of the originally damaged myofibres.

Imbalanced degeneration-regeneration, possibly exacerbated by high levels of inflammation, is the primary driver of muscle loss.[42]

The muscle fibers of this group (III) were splitted and wavy, which was probably due to the incomplete fusion of the regenerating fibers within the same basal lamina[43,44]. There were also eosinophilic material infiltrate between the fibers which denoted the presence of immature collagen. The highest percentage of fibrosis was recorded in this group (III).

The muscle regeneration process after an injury depends on a balance between pro-inflammatory and anti-inflammatory factors.[45] Although the phases of the repair process are alike after different causes of damage, the kinetics and amplitude of each phase may depend on the exact muscle damaged, the extent of damage, or the damage model use.[46]

In the present study, in group III, the extent of the lesion according to RCOG, 2015[47] could reach up to grade 3 (injury to both EAS and IAS), and this leads to limited muscle regeneration. This was similar to Rodrigues *et al*., (2013)[45] who stated that despite the remarkable ability of the muscle to regenerate, it depends on the extent of the lesion. The regenerative capacity of the injured muscle might be limited due to the formation of the fibrotic tissue. This fibrotic tissue can lead to atrophy, contracture, pain and predispose to recurrent injury.

Smooth muscle injury resulted in an inflammation and the release of soluble factors which stimulated the surrounding smooth muscle cells to divide and replace the damaged cells. Inflammation after injury stimulates the deposition of fibrous connective tissue, or extracellular matrix. This extracellular matrix is known to play a role in early muscle development and smooth muscle regeneration. However, during disease or excessive build-up of fibrous material, the extracellular matrix could impede the smooth muscle regeneration.[48]

On the other hand, the endogenous repair mechanisms in skeletal muscles can only regenerate a limited amount of muscle tissue and can thus be overwhelmed by the deposition of fibrotic scar after the muscle loss resulting from trauma or large resection.[49]

Moreover, remodeling of connective tissue is an important step for regeneration of muscle.[50] In the early stage, the fibrotic response is useful, causing stabilization of the tissue and supports regeneration of myofibers. However, excessive synthesis of collagen following injury, results in an increase in the size of scar tissue over time which may impair the normal function of muscle.[51]

Few numbers of satellite cells were also noted among this group (III) in the healing skeletal muscles and can be recognized between the sarcolemma and the basal lamina. Relaix and Zammit, (2012)[52] & Sambasivan *et al*., (2011)[53] explained the presence of such cells. Muscle fibers are post-mitotic cells, which are not able to divide. Following any injury, the muscle fibers that are damaged can't be repaired in the absence of adult muscle stem cells, which are the satellite cells.

Following activation, proliferation of satellite cells generate the myoblasts that can differentiate to repair the fibers that are damaged.[54] Collins (2006)[55] added that if a cell is damaged to a greater degree than can be repaired by satellite cells, the muscle fibers are replaced by scar tissue in a process called fibrosis. As the scar tissue cannot contract, the muscle that has persistent significant damage, loses strength and cannot produce the same amount of power or strength as before the injury.

In the current study, it was found a decreased number of smooth muscle fibers among group III. It is generally accepted that inflammatory stimuli are important contributing factors to the catabolism of muscle tissue.[56] This fact is thought to be secondary to collagen deposition, resulting in smooth muscle atrophy and fibrosis.[57]

Moreover, the severity of illness and the higher degree of inflammatory response might result in increased in the level of different cytokines in the plasma involved in the inflammatory response.[58] Some of these cytokines such as tumor necrosis

factor-, interleukin-1, and interleukin-6 were reported to have a catabolic effect on muscle tissue.[59]

Therefore, the relatively high levels of inflammatory cytokines resulted after the injury might inhibit differentiation of satellite cells, and hence maintenance of the muscle, resulting in a loss of muscle mass and quality.[60]

On the other hand, the present work demonstrated improvement in the structure of the anal sphincters in group IV. That group was subjected to anal electrical stimulation sessions for three weeks. The structural improvement was demonstrated by regaining the normal architecture of the three muscle layers with their regularly arranged muscle fibers. Most of the external anal muscle fibers retained their striated appearance and their regenerating nuclei started to take a peripheral position. Few fibers were still splitted and wavy. The internal anal muscle fibers presented with vesiculated nuclei and prominent nucleoli with pale cytoplasm. Many satellite cells reappeared along the anal muscles.

In the present study a significant structural gain of muscle tissue was found among this group (IV) in the form of hyperplasia of the smooth muscle. The results obtained in the present study pointed to the role of the cutaneous electrical stimulation performed in the process and which helped regeneration and proliferation of muscle tissues.

Press and Bergfeld (2007)[61] found that stimulation of muscle with the lower frequency electrical current could increase the size of muscle fiber which was thought to be due to proliferation of nuclei of muscle cell.

Electrical stimulation has been shown to guide cells in wound healing. Willis (2015)[26] reported that electrical stimulation of intact anal sphincter significantly increased gene expression of CXCL12 and +CCL7, the two important cytokines for mesenchymal stem cell migration.

In the presence of damaged tissue, an endogenous electrical current of nerve stimulation is required for normal regeneration, through induction of mitotic processes, and cell signaling mechanisms guide. It also stimulates cell migration towards the injury by a phenomenon called electrotaxis.[62] Electrotaxis also acts on the distribution of integrins in the cell membrane of fibroblasts, stimulating their adhesion to the extracellular matrix protein. Therefore, according to Zhao *et al*., (2017)[63] the exogenous electrical stimulation can give the same effects of endogenous electrotaxis.

Smooth muscle hyperplasia (internal anal sphincter) caused by the cutaneous electrical stimulation might be related to the activation of the protein tyrosine kinase Ab 1 (Abelson tyrosine kinase), expressed in smooth muscle cells and has many functions.[64] From these functions regulating actin and adhesions of the cell, in addition to proliferation of cell in response to the activation of growth factors.[65]

Skeletal muscle fibers hypertrophy results from the increased muscle fiber due to the increase in intracellular protein synthesis as an adaptive process. The rate of synthesis and degradation of proteins and the number of myonuclei are responsible for this hypertrophy[66]. In cases of muscular atrophy diseases, there is gradual loss of myonuclei from the skeletal muscle fibers, resulting in lower protein synthesis.[67] However, the reverse can occur in the presence of an excitatory stimulus which was the cutaneous electrical stimulation of the anal muscles.(63) In this case, the satellite cells are activated and begin to form new muscle fibers that actively restore protein synthesis.[66]

The immunohistochemistry reaction to desmin antibody of the present work demonstrated negative immuno-reactivity to desmin antibody in group III. Group IV showed the normal positive reaction to the desmin antibody in the skeletal muscle fibers of the EAS. It has been known that the network of desmin filament involves a major cytoskeleton portion of the fibers of the skeletal muscle and therefore it is possible to perform a part in the stabilization and organization inside the cells. Moreover, desmin also links to the extracellular matrix at costameres, suggesting that it could have an effect on the organization and regulation of the matrix as well.[68]

The loss of desmin leads to more muscle injury during its regeneration. In response to injury, there is inflammatory response in the muscle leading to the regeneration process. However, since there is lack of desmin in the regenerated fibers, they will

still be vulnerable to further injury. Moreover, the inflammatory process is already known to induce fibrosis.[69,70]

The decrease in desmin level in muscles results in more stiffness, increased collagen, and increase expression of genes responsible for turnover of extracellular matrix. Moreover, in the absence of desmin, the skeletal muscle suffers an increase in inflammation and regeneration.This is evident by centrally nucleated fibers, higher inflammatory and regeneration state and increased inflammatory cell numbers.[71]

The immunohistochemistry reaction to alpha SMA antibody of the present work demonstrated negative reaction in group III and positive reaction in group IV. The expression of alpha-SMA in smooth muscles was associated with the activation of myofibroblasts.[72] Myofibroblasts are considered as a form of fibroblasts that partially become differentiated into a smooth muscle phenotype.[73] They use some of the cytoskeletal proteins to contract, which are found in the smooth muscle cells, particularly alpha-SMA. Speeding wound repair is done by these cells by contracting the wound edges. It is accepted that differentiation of fibroblast to form myofibroblast is the essential event for tissue repair and wound healing.[74]

CONCLUSIONS

The present study confirmed the importance of cutaneous electric stimulation as a non-invasive method in the treatment of anal incontinence. It was useful in regaining the normal architecture of the injured anal muscles. Moreover it appeared to be beneficial for the hypertrophy of muscles and hence for improving the function of the internal and external anal sphincters.

CONFLICT OF INTERESTS

There are no conflicts of interest.

REFERENCES

1. Rao S. Pathophysiology of adult fecal incontinence.; Gastroenterology. 2004; 126(1): 14-22.

2. Ternent C, Fleming F, Welton M, *et al*. Clinical Practice Guideline for Ambulatory Anorectal Surgery. American Society of Colon and Rectal Surgeons. Dis Colon Rectum. 2015; 58(10):22-915.

3. Pretlove S, Thompson P, Toozs-Hobson P, *et al*. Does the mode of delivery predispose women to anal incontinence in the first year postpartum? A comparative systematic review. BJOG. 2008; 115(4):421-34.

4. Kinugasa Y & Moriya Y. Intersphincteric Resection for Low Rectal Tumors.Ann Gastroenterol Surg. 2012; 1(1):24-32.

5. Enck P, Hinninghofen H, Merletti R, *et al*. The external anal sphincter and the role of surface electromyography. Neurogastroenterol Motil.2005; 17 (1): 60–67.

6. Mahadevan V. The anatomy of the rectum and anal canal. Elsevier surgery. 2010; 29(1):5-10.

7. Agarwal S. Anatomy of the Pelvic Floor and Anal Sphincters. JIMSA. 2012; 25(1):9.

8. Yu S and Rao S Anorectal physiology and pathophysiology in the elderly. Clin. Geriatr. Med. 2014; 30(1):95-106.

9. Rociu E, Stoker J, Eijkemans M, *et al*. Normal anal sphincter anatomy and age- and sex-related variations at high-spatial-resolution endoanal MR imaging. Radiology. 2000; 217 (2):395-401.

10. Kearney R, Sawhney R and Delancy J. Levator ani muscle anatomy evaluated by origininsertion pairs. Obstet Gynecol. 2004; 104(1): 73- 168.

11. Tsukada Y, Ito M, Watanabe K, *et al*. Topographic Anatomy of the Anal Sphincter Complex and Levator Ani Muscle as It Relates to Intersphincteric Resection for Very Low Rectal Disease. Dis Colon Rectum. 2016; 59(5): 33-426.

12. Lunniss P and Phillips R. Anatomy and function of the anal longitudinal muscle. Br J Surg. 1992; 79(9):882–884.

13. Macchi V, Porzionato A, Stecco C, *et al*. Histo-topographic study of the longitudinal anal muscle. Clin Anat. 2008; 21(5):447–452.

14. Rao S and Meduri K. What is necessary to diagnose constipation? Best Pract Res Clin Gastroenterol. 2011; 25(1):127-140.

15. Hall K, Ward S, Cobine C, *et al*. Spatial organization and coordination of slow waves in the mouse anorectum. J Physiol. 2014; 592(17):3813-3829.

16. David E, Patricia L, Theodore J, *et al*. The ASCRS textbook of colon and rectal surgery. Springer. 2007; 2:335-345.

17. Jose M, De-Ocampo S, Paulson J, *et al*. RECTO-ANAL REFLEXES AND SENSORI-MOTOR RESPONSE IN RECTAL HYPOSENSITIVITY. Dis Colon Rectum. 2010; 53(7): 1047–1054.

18. Bharucha A. Pelvic floor: anatomy and function. Neurogastroenterol Motil. 2006; 18(7):507-19.

19. Pankaj G. Intersphincteric Component in a Complex Fistula-in-Ano Is Like an Abscess and Should Be Treated Like One. Diseases of the Colon & Rectum. 2018; 61 (4): 26.

20. Landefeld C, Bowers B, Feld A, *et al*. National Institutes of Health state-of-the-science conference statement: prevention of fecal and urinary incontinence in adults. Ann Intern Med. 2008; 148(6):449-58.

21. Bharucha A, Dunivan D, Goode P, *et al*. Epidemiology, pathophysiology, and classification of fecal incontinence: State of the Science Summary for the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) Workshop. Am J Gastroenterol. 2015; 110(1): 127–136.

22. Amy E, Sarah B, and Michael D. Common Anorectal Disorders. Gastroenterol Hepatol (N Y). 2014; 10(5): 294–301.

23. Adil, E, Gena D, Patricia S, *et al*. Epidemiology, Pathophysiology, and Classification of Fecal Incontinence: State of the Science Summary for the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) Workshop. Am J Gastroenterol. 2015; 110(1):127–136.

24. Guise J, Morris C, Osterweil P, *et al*. Incidence of fecal incontinence after childbirth. Obstet Gynecol. 2007; 109(2): 8 - 281.

25. Mark I, Leica S, Peter H, *et al*. Transcutaneous electrical nerve stimulation (TENS) for fibromyalgia in adults. Cochrane Database Syst Rev. 2017; 9(10): 12-172.

26. Willis A. How Does Electrical Muscle Stimulation Work? 2015

27. De Kroon J, Ijzerman M, Chae J, *et al*. Relation between stimulation characteristics and clinical outcome in studies using electrical stimulation to improve motor control of the upper extremity in stroke. J Rehabil Med. 2005; 37(2):65–74.

28. Azman M and Azman A. The Effect of Electrical Stimulation in Improving Muscle Tone. IOP Conf. Ser.: Mater. Sci. Eng. 2017;260(2):12-20.

29. Hugo A, Maria G, Keyla P, *et al*. Electrical stimulation structurally affects the tissues of the rectum and anus of nulliparous rats. Journal of Anatomy. 2017; 231(3); 398-404.

30. Levilester S, Marc P, Margot D, *et al*. Functional Outcome After Anal Sphincter Injury and Treatment With Mesenchymal Stem Cells. Stem Cells Transl Med. 2014; 3(6): 760–767.

31. Bataa M, Ansam A, Enas A, *et al*. Effects of transcutaneous electrical stimulation of lower limb muscles on experimental fatty liver. Arab Journal of Gastroenterology. 2016;17(1): 20-28.

32. Benezech A, Bouvier M and Vitton V. Faecal incontinence: current knowledges and perspectives. World J Gastrointest Pathophysiol.2016; 7(1): 59–71.

33. De Sousa H, Silva M, Barbosa K, *et al*. Electrical stimulation structurally affects the tissues of the rectum and anus of nulliparous rats. J Anat. 2017; 231(3):398-404.

34. Schaap L, Pluijm S, Deeg D, *et al*. Inflammatory markers and loss of muscle mass (sarcopenia) and strength. Am J Med. 2006; 119(9): 517-529.

35. James G. Inflammatory processes in muscle injury and repair. Am J Physiol Regul Integr Comp Physiol. 2005; 288(2): 345–353.

36. Brooke, L, Megan S and John. The Role of Heparan Sulfate in Inflammation, and the Development of Biomimetics as Anti-Inflammatory Strategies.J Histochem Cytochem. 2018; 66(4): 321–336.

37. Tiidus P. Radical species in inflammation and overtraining. Can J Physiol Pharmacol. 1998; 76 (5): 450-533.

38. Tidball J. Inflammatory processes in muscle injury and repair. Am J Physiol Regul Integr Comp Physiol.2005; 288 (2): 53-345.

39. Cannon J and St Pierre B. Cytokines in exertion-induced skeletal muscle injury. Mol Cell Biochem. 1998; 179 (2): 159-67.

40. Smith C, Kruger M, Smith R, *et al*. The Inflammatory Response to Skeletal Muscle Injury. Sports Medicine. 2008; 38(11): 947–969.

41. Bigard A and fink E. Basic science of tissue healing and repair. Skeletal muscle regeneration after injury. Cellular and Molecular Events. 2010; 3: 35-51.

42. Michael C, Anshu S, Oke A, *et al*. Histological Evidence of Muscle Degeneration in Advanced Human Rotator Cuff Disease. J Bone Joint Surg Am. 2017; 99(3): 190–199.

43. Blaveri K, Heslop L, Yu D, *et al*. Patterns of repair of dystrophic mouse muscle: studies on isolated fibers. Dev Dyn. 1999; 216(3): 244-256.

44. David G, Nicholas P and Stanley C. Absence of Dystrophin Disrupts Skeletal Muscle Signaling: Roles of Ca2+, Reactive Oxygen Species, and Nitric Oxide in the Development of Muscular Dystrophy. Physiol Rev. 2016; 96(1): 253–305.

45. Rodrigues N, brunelli R, Parizotto N, *et al*. low level laser therapy (LLLT) (660nm) alters gene expression during muscle healing in rats. Journal of Photochemistry and Photobiology B: Biology. 2013; 120: 29-30.

46. Antonio M. The Basis of Muscle Regeneration. Advances in Biology. 2014; Article ID 612471:1-16 pages.

47. Royal College of Obstetricians and Gyneacologists(RCOG). The Management of Third- and Fourth-Degree Perineal Tears. 2015

48. Online site1:(https://education.seattlepi.com/muscle-cells-greatest-ability-regenerate-3768.html

49. Turner N and Badylak S. Regeneration of skeletal muscle. Cell Tissue Res. 2012; 347(3):74-759.

50. Darby I, Zakuan N, Billet F, *et al*. The myofibroblast, a key cell in normal and pathological tissue repair. Cell Mol Life Sci. 2016; 73(6):1145–1157.

51. Mann E, Perdiguero Y, Kharraz N, *et al*. Aberrant repair and fibrosis development in skeletal muscle. Skeletal Muscle. 2011; 1(1): 21.

52. Relaix F and Zammit P Satellite cells are essential for skeletal muscle regeneration: the cell on the edge returns centre stage. Development. 2011 ; 139(16): 2845–2856.

53. Sambasivan R, Yao R, Kissenpfennig A, *et al*. Pax7-expressing satellite cells are indispensable for adult skeletal muscle regeneration. Development. 2011; 138(17):3647–3656.

54. Dhawan J and Rando T. Stem cells in postnatal myogenesis: molecular mechanisms of satellite cell quiescence, activation and replenishment. Trends Cell Biol. 2005; 15(12):666–673.

55. Collins C. Satellite cell self-renewal. Curr Opin Pharmacol. 2006; 6(3):301–306

56. Sakuma K, Aoi W, Yamaguchi A, *et al*. Current understanding of sarcopenia: possible candidates modulating muscle mass. Pflugers Arch. 2015; 467(2):213-29.

57. Reid W, Rurak J and Harris R. Skeletal muscle response to inflammation—Lessons for chronic obstructive pulmonary disease. Crit Care Med. 2009; 37(10): 372–383

58. Riche F, Cholley B, Panis Y, *et al*. In flammatory cytokine response in patients with septic shock secondary to generalized peritonitis. Crit Care Med. 2000; 28(2):433–437.

59. Lang C and Frost R. Sepsis-induced suppression of skeletal muscle translation initiation mediated by tumor necrosis factor alpha. Metabolism. 2007 ; 56(1):49 –57.

60. Langen R, Van Der Velden J, Schols A, *et al*. Tumor necrosis factor-alpha inhibits myogenic differentiation through MyoD protein destabilization. FASEB J. 2004; 18(2):227–237.

61. Press J and Bergfeld D. Physical Modalities. Clinical Sports Medicine. 2007; 32(3): 207–226.

62. Balakatounisa K and Angoulesc A. Low-intensity electrical stimulation in wound healing: review of the efficacy of externally applied currents resembling the current of injury. Eplasty. 2008; 8(28): 283–291.

63. Zhao B, Shen P and Liu K. Perioperative statins do not prevent acute kidney injury after cardiac surgery: A meta-analysis of randomized controlled trials. Journal of cardiothoracic and vascular anesthesia. 2017; 31(6):2086-2092.

64. Cleary R, Wang R, Waqar O, *et al*. Role of c-Abl tyrosine kinase in smooth muscle cell migration. Am J Physiol Cell Physiol. 2014; 306(8): 753–761.

65. Jia L, Wang R and Tang D. Abl regulates smooth muscle cell proliferation by modulating actin dynamics and ERK1/2 activation. Am J Physiol Cell Physiol. 2012; 302(7): 1026–1034.

66. GuoY, Xiao L, Sun L, *et al*. Wnt/β-catenin signaling: a promising new target for fibrosis diaseases. Physiol Res. 2012; 61(4):337–346.

67. Mitchell P and Pavlath G. Skeletal muscle atrophy leads to loss and dysfunction of muscle precursor cells. Am J Physiol Cell Physiol. 2004; 287(6): 1753–1762.

68. Lovering R, O'Neill A, Muriel J, *et al*. Physiology, structure, and susceptibility to injury of skeletal muscle in mice lacking keratin 19-based and desmin-based intermediate filaments. Prosser BL, Strong J, Bloch RJ. Am J Physiol Cell Physiol. 2011; 300(4): 13-203.

69. Li Y, Foster W, Deasy B, *et al*. Transforming growth factor-beta1 induces the differentiation of myogenic cells into fibrotic cells in injured skeletal muscle: a key event in muscle fibrogenesis. Am J Pathol. 2004; 164(3):19-100.

70. Serrano A and Muñoz-Cánoves P. Regulation and dysregulation of fibrosis in skeletal muscle. Exp Cell Res. 2010; 316(18): 8-305.

71. Gretchen A and Richard L. Skeletal muscle fibrosis develops in response to desmin deletion. Am J Physiol Cell Physiol. 2012; 302(11): 1609–1620.

72. Nakatani T, Honda E, Hayakawa S, *et al*. Effects of decorin on the expression of alpha-smooth muscle actin in a human myofibroblast cell line. Mol Cell Biochem. 2008; 308(1-2): 7-20.

73. Elberg G, Chen L, Elberg D, *et al*. MKL1 mediates TGF-beta1-induced alpha-smooth muscle actin expression in human renal epithelial cells. Am J Physiol Renal Physiol. 2008; 294(5): 1116–1128.

74. Jarvinen T, Jarvinen T, Kaariainen M, *et al*. Muscle injuries: biology and treatment. Am J Sports Med. 2005; 33 (5): 745-64.

دراسة تأثير التحفيز الكهربائى الجلدى على هيكل العضلات الشرجية للفأر بعد القطع الجراحى لاصابة العضلات العاصرة الشرجية

ملخص البحث

من المتعارف عليه أن عضلات فتحة الشرج تؤدي دورًا حاسمًا في الحفاظ على السيطرة علي عملية الاخراج. أي خلل في تركيبتها يمكن أن يؤثر سلبا على السيطرة الفسيولوجية للبراز.تعتمد طرق العلاج المطبقة على حالات سلسال براز على كفاءتها. أحدثها هو التحفيز الكهربائي الجلدي عن طريق الشرج، و الذي ثبت تأثيره العام على العضلات الشرجية.

تم استخدام ثلاثون من إناث الجرذان البيضاء في هذه الدراسة:

وقد تم تقسيم الجرذان الي أربعة مجموعات.

المجموعة أ: تم تقسيم اثني عشر جرذ بالغ من الإناث إلى مجموعتين فرعيتين في كل منهما ستة جرذان.

١- مجموعة التحكم.

٢- مجموعة الشام

المجموعة ب: تعرضت ستة جرذان بالغات لإصابة قطعيه للعضلة العاصرة الشرجية.

المجموعة ج: تعرضت ست جرذان بالغات لإصابةقطعية للعضلة العاصرة الشرجية وتركت للشفاء التلقائي لمدة ثلاثة أسابيع.

المجموعة د: تعرضت ست جرذان بالغات لإصابة مستحثة للعضلة العاصرةالشرجية،ثم تلقت خمس جلسات أسبوعيًا من التحفيز الكهربائي الشرجي الجلدي لمدة ثلاثة أسابيع.

في نهاية التجربة، تم استخراج القناة الشرية. تم أخذ العينات ومعالجتها للدراسة بالمجهر الضوئي بعد صبغ العينات بصبغات مختلفة.

أظهرت فحوصات القناة الشرجية من المجموعة الثانية قطع في عضلات الشرج الخارجية و الداخلية على حد سواء مع ترشيح ملحوظ للخلايا الالتهابية و نزيف خلالي ملحوظ على طول العضلات في موقع الإصابة المستحثة. في حين ان المجموعة الثالثة قد فقدت التركيب النسيجي الطبيعي لألياف عضلات الشرج التي تركت للشفاء التلقائي. كشفت عضلات الشرج الداخلية عن تسلل خلوي التهابي، في حين أظهرت عضلات الشرج الخارجية اتجاه الألياف المتموجة وفقدانها للتشنجات. من ناحية أخرى، كشفت المجموعة الرابعة نتائج أشارت إلى تجديد الألياف العضلية فيشكل استعادة ألياف مرتبة بانتظام فى كل من العضلات الخارجية والداخلية. استعادت معظم ألياف العضلات الشرجية الخارجية مظهرها المخطط و بدأت نواتها المجددة في اتخاذ موضعها الطبيعي. ما عدا ألياف قليلة لم تزل متموجة. ظهرت نواة كلا من ا لألياف العضلية تحويصليّة و استعادت أيضًا وضعها الطبيعي.

وقد نوقشت هذه النتائج مع ما سبق دراسته في هذا الموضوع واستنتج أن استخدام التحفيز الكهربائي الجلدي مفيد في التئام العضلات مع استعادة معظم الهيكل الطبيعي و وظيفة عضلات الشرج الخارجية و الداخلية المصابة.