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EXTERNAL ANAL SPHINCTER PLICATION: A NOVEL SURGICAL TECHNIQUE TO ENHANCE THE ANAL CANAL CONTINENCE FUNCTION IN AN EXPERIMENTAL ANIMAL MODEL

Hypothesis /aims of study

Our recent studies show that the external anal sphincter muscle (EAS) operates at the low end of ascending limb of the length tension curve (low sarcomere length) *in-vivo (1).* In the present study, we tested a novel hypothesis that by surgically adjusting the sarcomere length to its optimal length, the EAS muscle tension and anal canal pressure can be enhanced**. Aims:** The goal of our study was to determine the effects of EAS plication on the sarcomere length, anal canal pressure and EAS muscle tension.

Study design, materials and methods

In rabbits, we performed EAS plication involving different muscle lengths (13, 20, 28 and 35% of the EAS circumference) and determined its effect on the active EAS muscle tension during electrical stimulation as well as passive anal canal tension. Animals (n=25) were anesthetized and subjected to either sham (group I; n=5) or EAS plication (groups II-V; n=5 in each group) surgery. The circumference of the anal canal was first measured and an incision was made to expose the EAS muscle. Sutures (4.0-gauge polypropylene) were placed at 2 points on the EAS muscle, at a distance of 13, 20, 28, and 35% of the circumferential length of the anal canal for the animals in groups II, III, IV, and V respectively. The two ends of the sutures were tied together (EAS plication) and the skin incision was closed. Anal canal pressures were recorded using a 3 mm sleeve sensor catheter, before and after the plication surgery. Effect of EAS plication on the anal canal pressure during electrical stimulation (1-6mA, pulse duration 5 ms and pulse frequency 50Hz) of the EAS (active EAS muscle contraction) and after the administration of neuromuscular blocking agents (pancuronium bromide 0.4 mg/kg) and smooth muscle relaxant (sodium nitroprusside; 1.5µg/kg), (passive anal canal pressure) were recorded. Anal canal was harvested at the end of pressure recordings and the EAS muscle was removed for the determination of the sarcomere length using the laser diffraction technique.

Results

Electrical stimulation of the EAS muscle resulted in a stimulus dependent increase in the anal canal pressure and EAS muscle tension. Maximum increase in the anal canal tension (g/cm^2) was observed with 20% plication (95%; $P < 0.05$) (Maximal electrical stimulus). The passive anal canal tension was not different between the sham and sphincter plicated animals.

Table: Effect of Plication on Anal Canal Tension (g/ cm^2) and EAS Sarcomere Length (µm) ;(mean± SE)

Procedure	13%	20%	28%	35%
Pre-plication	629 ± 20	656 ± 120	662 ± 82	757 ± 91
Post-plication	854 ± 54 P< 0.05	1045 ± 72 P< 0.05	814 ± 53	798 ± 156
Passive Anal Canal Tension	25 ± 2	40 ± 11	25 ± 3	22 ± 3
Post-plication sarcomere length	2.24±0.03 µm	2.64±0.05 µm	2.72±0.01 µm	2.81±0.04 µm

Interpretation of results

Plication resulted in the length dependent increase in the sarcomere length (see table). The EAS muscle tension generated at the 20% plication is consistent with the optimal sarcomere length calculated by the previously determined thin filament measurement of 2.45 – 2.59.

Concluding message

Our results suggest that the EAS plication is feasible and can change the sarcomere length of the EAS muscle. Optimal sarcomere length and maximum improvement in EAS muscle tension and canal pressures were achieved with 20% plication without affecting passive anal canal tension.

References
1. Gastroenterology (2007) **132** (Supply 2), 590.

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What were the subjects in the study?	**ANIMAL**
Were guidelines for care and use of laboratory animals followed or ethical committee approval obtained?	**Yes**
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