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A BIOMECHANICAL MODEL OF THE HUMAN DEFECATORY SYSTEM TO INVESTIGATE MECHANISMS OF CONTINENCE

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Abstract

Introduction: This paper presents a method to fabricate, measure and control a physical simulation of the human defecatory system to investigate individual and combined effects of anorectal angle and sphincter pressure on continence. To illustrate the capabilities and clinical relevance of the work the influence of a passive-assistive artificial anal sphincter (FENIX^{TM}) is evaluated.

Methods: A model rectum and associated soft tissues, based on geometry from an anonymised CT dataset, was fabricated from silicone and showed behavioural realism to the biological system and ex-vivo tissue. Simulated stool matter with similar rheological properties to human faeces was developed. Instrumentation and control hardware were used to regulate injection of simulated stool into the system, automate balloon catheter movement through the anal canal, define the anorectal angle and monitor stool flow rate, intra-rectal pressure, anal canal pressure and puborectalis force. Studies were conducted to examine the response of anorectal angles at 80°, 90° and 100° with simulated stool. Tests were then repeated with the inclusion of a FENIX device.

Results: Stool leakage was reduced as the anorectal angle became more acute. Conversely, intra-rectal pressure increased. Overall inclusion of the FENIX reduced faecal leakage, while combined effects of the FENIX and an acute anorectal angle showed the greatest resistance to faecal leakage. These data demonstrate that the anorectal angle and sphincter pressure are fundamental in maintaining continence. Furthermore it demonstrates that use of the FENIX can increase resistance to faecal leakage and reduce anorectal angles required to maintain continence.

Conclusions: Physical simulation of the defecatory system is an insightful tool to better understand, in a quantitative manner, the effects of the anorectal angle and sphincter pressure on continence. This work is valuable in helping improve our understanding of the physical behaviour of the continence mechanism and facilitating improved technologies to treat severe faecal incontinence.

KEY WORDS

Faecal incontinence, fecal, physiological model, incontinence device

1. Introduction

Faecal Incontinence (FI) is the inability to carry out controlled defecation and leads to the involuntary passing of bowel content, including flatus, mucus and liquid and solid faeces. Stigma and social taboo are associated with FI, leading to its underreporting (1). Despite this the known prevalence of FI in adults is high, estimated at 11-15% and increasing with age, where approximately 33% of people living in retirement homes (or similar institutions) are affected (2). Overall, FI is a condition with profound consequences for individuals, their family/friends, and the wider healthcare system (3). Unfortunately, treatment options for FI are limited and there is a consequent need to develop new understanding and technology to help address this deficit.

1.1 Anatomy and Physiology of Continence

Continence relies on the coordinated function of the nervous system, gastrointestinal tract, and anal sphincter and pelvic floor musculature (4-8). Figure 1 shows key parts of the anatomy associated with continence. The rectum, which stores faecal matter prior to defecation, is a hollow muscular tube approximately 13cm in length and composed of a continuous layer of longitudinal muscle that interlaces with the underlying circular muscle (9). The distal end of the rectum joins to the anal canal, a muscular tube 2.5-4cm in length which ends at the anus (10).

The anal sphincter complex (internal and external sphincters) applies pressure over the length of the anal canal, enabling it to be occluded. The puborectalis (PR) and levator muscles are anchored at the pubis and loop around the bottom of the rectum. They act to support this structure and can also occlude the top of the anal canal. The PR also acts to mediate the angulation between the anal canal and the rectum, termed the anorectal angle (ARA). The presence of an acute ARA has been considered important in maintaining continence (11, 12). At rest, the anal canal forms an angle of approximately 105° (13) with the axis of the rectum. During voluntary hold the ARA becomes more acute, whereas during defecation, the angle

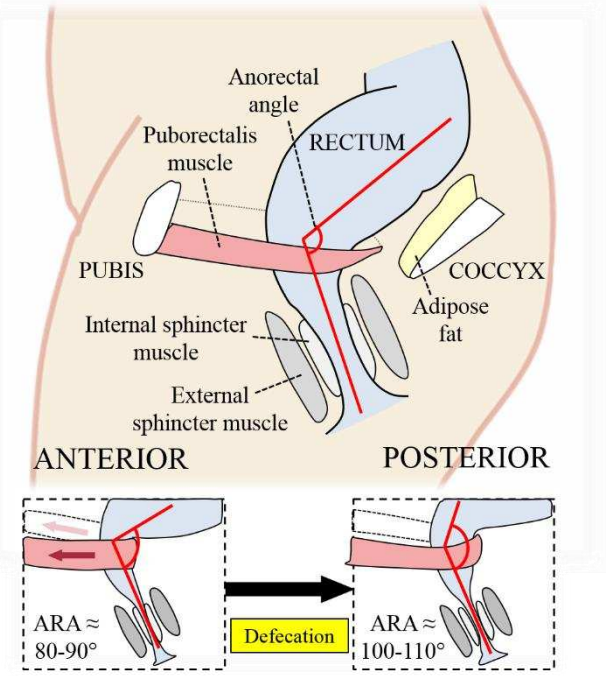

Figure 1 *A schematic showing key components of the physiology of the defecatory system and their action.*

becomes more obtuse.

During defecation, evacuation of faecal matter is promoted by minimising resistance to its passage while applying motive pressures. Relaxation of the anal sphincters minimises occlusion of the anal canal while relaxation of the PR enables the ARA to straighten so the bend is less acute. In conjunction abdominal pressures are elevated and the rectal wall muscle contract to force faeces through the rectum and anal canal until it is expelled at the anus. Dysfunction of any one of these components can result in FI, with common causes including diarrhoea, obstetric trauma, spinal cord injury and rectal prolapse (14).

1.2 Clinical Treatment of FI

Treatment to address FI is a complex process, a reflection of the multifaceted, interlinked causative factors and the wide array of physiological mechanisms used to maintain continence. Conservative methods such as dietary modifications, lifestyle alteration, constipating drugs, suppositories and biofeedback therapies (15) are effective at treating mild cases of FI. As symptoms become more severe, treatment modalities move toward surgical intervention.

Sacral nerve modulation and sphincteroplasty are the commonest surgical modalities for the treatment of FI, but their efficacy deteriorates in the longer term. There is therefore a need for treatments with more durable benefit. For worse cases of FI, efforts have been made to use technology developed for urinary incontinence in which an implantable, manually inflatable cuff is used to occlude the urethra (16, 17). Unfortunately, using a similar approach to treat FI by occluding the sphincter (18, 19) has been plagued with complications including local ischaemia due to the occlusive pressures necessary to maintain continence (7, 20-22). Currently only a small number of implantable devices are available to treat patients with severe FI and these focus on augmentation of the anal sphincter. Two treatments currently on the market include the passive FENIX^{TM} (Torax Medical, Minnesota) (23) system and the active Acticon Neosphincter^{TM} (24), for which studies have shown success rates (for people with a functioning device) of 65%, at a mean follow up of 26.5 months (25). A previous, but less often used, strategy is the post-anal repair operation for idiopathic FI, designed to correct an overly obtuse ARA (5) by reducing the angulation (26, 27). More recently, the TOPAS posterior sling is designed to restore anorectal angulation, although in practice it has not shown to be particularly effective, with an improvement seen in 16.1% of patients at a mean follow up of 24.9 months (28).

The paucity of commercially available, clinically viable systems to treat FI reflects the difficulty of designing medical technologies to meet the multi-faceted challenges surrounding this complex condition. A key failure mode in many attempts at new technology has been when device-tissue interaction causes tissue erosion, resulting in device migration or rejection (29, 30). Alternative strategies to sphincter augmentation have also been explored. Notably, in-vitro studies have shown that increasing ARA reduces the occlusion pressure required to hold back solids and semi-solids (31, 32). Similarly, another study reported increased retention of semisolid material when

increasing ARA in an ex-vivo porcine rectum, but no effect for water (32). The question of whether the ARA or sphincter occlusion pressure is a greater contributor to continence remains inconclusive, despite previous comparative studies (33, 34). However, it is evident that modulating ARA is a key feature in maintaining continence and that this provides a complementary strategy to sphincter augmentation. Unfortunately, there are currently no clinically available devices that exploit these features.

1.3 Modelling FI

There is a clear clinical need to develop improved technology to treat FI, and a promising opportunity to exploit mechanisms around ARA modulation. To further advance this work requires an in-depth biomechanical understanding of the associated physiological continence mechanisms and the effect of rectal disorders to capture their complex behavior and interaction.

There is a dearth of research in this area. Existing work is dominated by the use of computational models to simulate aspects of the pelvic floor system. Finite element models of the pelvic floor have been developed in attempts to understand its function in the urinary and faecal continence mechanisms. One model has been developed to investigate the effect of stool consistency on continence (35), whilst another looks at the effect of damaged ligaments on stress urinary continence (36). Computational models have also been developed to characterise the global behaviour of the pelvic floor muscles (37-41). However, there are large quantitative differences between the models and parameters used (42).

Whilst computational studies have been developed, a physical model provides opportunities to further understand the biomechanics of FI to help develop and optimise new systems for treatment. In particular, physical models can readily simulate the complex physical properties of faecal matter and the physical interactions between faecal matter and different tissues. Furthermore, they provide a convenient means to evaluate new treatment concepts. Accordingly, our research concerns the development of a physical model to investigate the effect of ARA on continence for the future development and evaluation of novel FI technologies.

This paper presents a biomechanical model of the human defecation system with an exploratory study to illustrate its capabilities and relevance. Section 2 details the approach and constituent physical models of the anatomy combined with computational measurement and control. An exploratory study using this model is defined in Section 3 which aims to firstly investigate the effects of rectal compliance and changing ARA on continence and secondly explores the clinical relevance of the work by evaluating the influence of a passive-assistive artificial anal sphincter (FENIX). Results from the study are then reported in Section 4 and discussed in Section 5, with particular consideration of their relevance to inform future treatment options for FI.

2. Model Development

Our approach in developing a physical model of the faecal system is to combine soft silicone representations of key parts of the anatomy, computerised control and instrumentation to objectively monitor and regulate physiologically relevant parameters and a stool simulant to obtain a realistic flow regimen in the system.

2.1 Anatomical Representation

The full biological continence mechanism is complex and consists of the coordinated

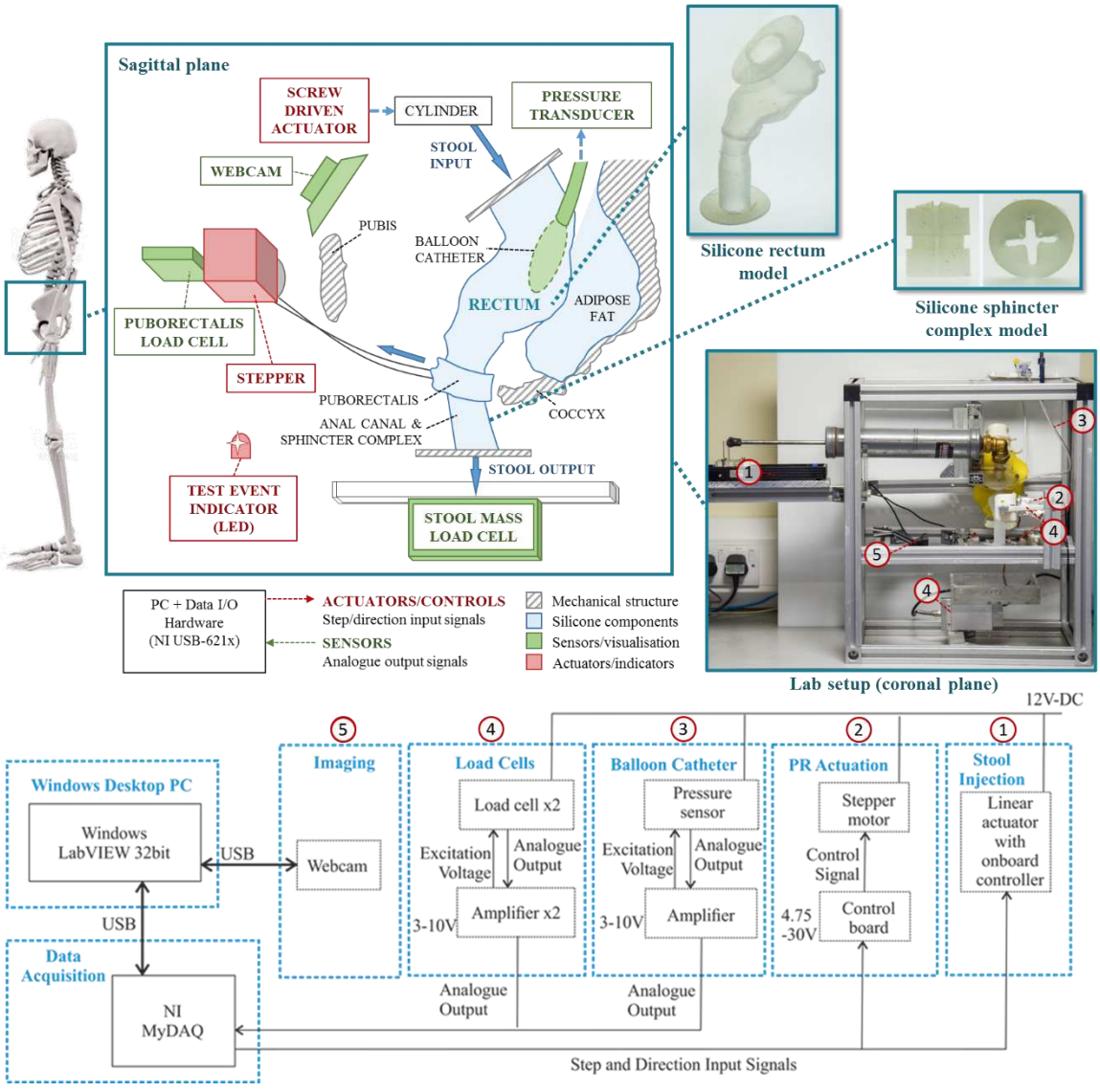

Figure 2 *Key components of the defecation model*

function of the nervous systems, gastrointestinal tract, and anal sphincter and pelvic floor musculature. Our current model is focused on investigating the effects of varying ARA and sphincter pressure, and accordingly we have simplified the system to facilitate fabrication and detailed analysis of these parts of the anatomy.
The model has been based on data for a 50th percentile adult male, although the methods and principles would extend to other percentiles, ages or gender. The rectum, adipose fat and PR muscle components are simulated by cast, 1:1 scale, silicone models, anatomically positioned within a housing linking these elements to control and instrumentation, as shown in Figure 2. The system is driven through a stool injection mechanism (detailed in section 2.4) while the ARA is regulated through an active PR muscle as part of the continence mechanism. By varying the pressure exerted by the PR muscle on the rectum, the ARA can be controlled and its effects on faecal leakage are observed during influx of simulated stool. The anal canal is represented within the rectum geometry with passive occlusion from an anal sphincter cuff. The anal sphincter occludes the anal canal by producing mucosal folds in the wall of the rectum phantom. This allows for expansion without elastic deformation of the rectal wall, to observe effects of sphincter pressure on the system.

2.2 Modelling Soft Tissues

A biomechanical representation of the soft tissue components in the model was achieved using a silicone casting process in which their geometry and mechanical properties were approximated.

Rectum Model

The rectum represents the most complex component in the model. The 3D geometry, shown in Figure 3a, was obtained from the open source 3D-IRCADb database (43) which contains a wide-range of high-fidelity anatomical structures, segmented from medical imaging by clinical experts, in 3D form. The particular dataset used here consists of segmented CT data from a 44 year old male patient with focal nodular hyperplasia of the

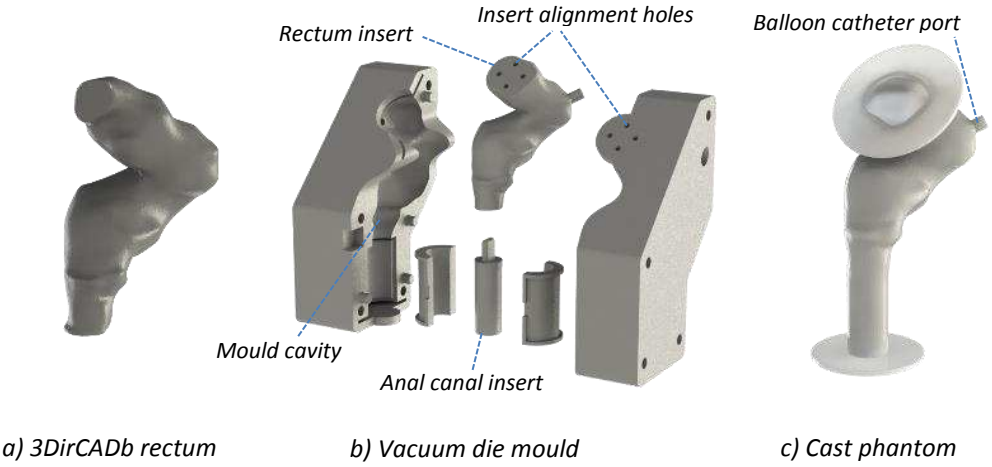

Figure 3 *Fabrication process of the rectum model with a) the segmented geometry b) the 3D printed vacuum injection mould and c) a cast rectum model in silicone*

liver, but no condition relating to FI. This model showed close agreement with other published works (9, 44) on the size and shape of the human rectum. However, it should be noted that this component could be interchanged with alternate geometries if required (e.g. to represent different anatomy).
To fabricate the rectum as a hollow silicone shell a custom mould was required. Firstly, the 3D geometry was imported into a CAD package (SolidWorks^{TM}, Dassault Systèmes) and modified to add flanges for mechanical fixation and interfacing with adjoining components. A 3D mould, Figure 3b, was then constructed using the modified rectum geometry. The mould consisted of two halves with an insert. Fixation points allowed the rectum insert to be correctly aligned within the mould cavity such that a uniform wall thickness was achieved. Lastly, a material reservoir and inlet ducts were added to the mould to enable fabrication by vacuum casting. With pre-mixed, de-gassed silicone in the material reservoir the mould was positioned in a vacuum chamber for 4 hours. When a vacuum is applied, air in the mould cavity is displaced with silicone where it cures, and the rectum model is de-cast (Figure 3c).

Soft tissues like the rectum exhibit highly non-linear mechanical behaviour which would be challenging to fully represent using a homogenous silicone material. However, based on the assumption that these tissues are operating within normal physiological conditions we found a good approximation could be achieved using commercial grades of silicone. To select this we compared the stress-strain response of passive human rectum tissue (45) within this limited strain regime [0-35%] to a range of commercially available silicone elastomers. Since the rectum is ‘active’ and modulates its contraction upon defecation we selected three variants of silicone whose compliance represent the rectum during contraction in healthy and diseased states (Dragon Skin 10A, 20A & 30A, Smooth-On Inc., Easton, USA).

Anal Canal and Sphincter Complex

The anal canal and sphincter complex are located at the distal end of the rectum (shown in Figure 2) and are modelled as a passive assembly, Figure 4, consisting of an inner silicone tube (the anal canal) and an outer constraint layer used to represent the combined occlusive action of the sphincter complex. The anal canal was modelled in a distended

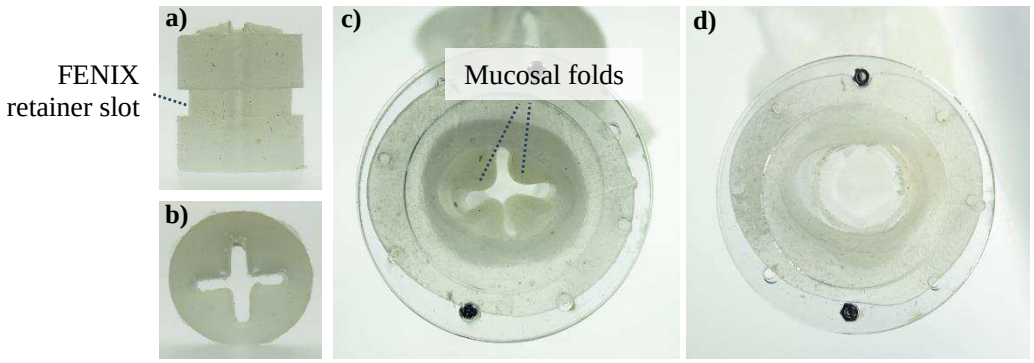

Figure 4 *The model sphincter showing a) side view; b) top view; c) simulated mucosal folds along the anal canal and d) the anal canal with the sphincter distended*

state (as during defecation) which is then constrained by the passive sphincter element to produce an occluded cross-section with features representing mucosal folds. The dimensions of these features were obtained from anatomical studies (46) and the 3D-IRCADb database (43) discussed above. A 1mm × 3mm retaining groove was added to the outer wall of the sphincter to locate the FENIX device and prevent the device moving longitudinally along the canal during use.

The anal canal and sphincter complex were fabricated from silicone elastomer (Ecoflex 00-30, Smooth-On Inc., Easton, USA), selected experimentally such that the resultant distensibility of the anal canal approximated that of a healthy adult in a rest state. A clinical anal manometry system (the EndoFLIP®, Crospon LTD (47)) was used to develop and validate this aspect, using the Distensibility index (DI) measure[1] (48, 49). The DI of the modelled anal canal complex was calculated at 4.18, in line with the ranges reported for healthy adults (DI=0.3-10.4, N=40) and those with FI (DI=0.7-12.1, N=34) (48).

Puborectalis Muscle

The puborectalis is part of the sheet-like *'levator ani'* musculature which forms a key part of the pelvic floor, anchored about the pelvis. It is the primary component of the levator ani associated with modulation of the ARA and therefore this model focuses solely on the PR, representing this structure as a simplified 'band' which wraps around the base of the rectum from anchor points at the pubis (40-42).

The key geometry of the PR in this model is its contact area at the rectum which was approximated from anatomical studies (46, 50) and defined as 18mm in width. The length of the PR is varied through an actuation mechanism described in the next section. The band was fabricated using a fine inextensible mesh (fiberglass mesh) embedded within a soft silicone elastomer (Ecoflex 00-50, Smooth-On, Inc.) to provide a soft interface between PR and rectum.

Connective and Supportive Structures

A range of elements were made to hold and support the functional parts of the defecation model (the rectum, anal canal and sphincter complex). An adult male pelvis model (Male Pelvis Skeleton, 3B Scientific, Hamburg, Germany) was used to house all the components and provide visual anatomical reference points for later analysis. Adipose fat was modelled using a soft silicone (Ecoflex 00-20, Smooth-On Inc., Easton, USA) to approximate the mechanical properties in healthy adults (51). The most distal part of the anal canal and the proximal end of the rectum were fixed relative to the pelvis using the soft silicone flanges and an adjustable aluminum framework (Rexroth, Bosch), positioned such that the combined rectum structure assumed a resting anatomical position (see Figure 1).

2.3 Modelling Faeces

[1] DI is defined as the cross sectional area at the narrowest point of the canal divided by catheter bag pressure at 50 ml inflation volume

Tests to determine the physical properties of faeces have shown that they vary considerably in viscosity, hardness and consistency. A pharmaceutical grade smectite clay, (VEEGUM R, Magnesium Aluminum Silicate NF Type IA, Vanderbilt Company), was selected as the stool medium for the simulation. It forms a homogenous solution with water that can be adjusted to obtain similar physical properties of density and viscosity comparable to those reported for soft faeces (52). This material is also used as simulated stool for nuclear medicine proctographic studies, enabling future comparative studies.
The formulation of the stool solution was determined through experimental analysis of its rheological properties. A range of samples were made by adding measured amounts of magnesium silicate powder to distilled water to produce 7, 8 and 9wt% magnesium silicate suspensions. Samples were dispersed using a chemical homogeniser for 2 minutes before being transferred immediately to the rheometer. Following homogenisation, samples were transferred immediately to a rheometer vessel to obtain shear rate-apparent viscosity flow curves for varying clay moisture contents. Interpolated viscosity was then plotted against moisture content (at a shear rate of $1s^{-1}$). The measured moisture contents of human faeces range from 58.5% to 88.7% by mass, with apparent viscosities at $1s^{-1}$ ranging between 52.8 and 3306.3 Pa.s based on a power law relationship. In this study the clay formulation was selected at 90.5% water content, producing an apparent viscosity of 47.065 Pa.s similar to high moisture-content semisolid faecal samples (52).

2.4 Measurement and Control

Instrumentation and control systems were integrated into the model to quantitatively measure key aspects of the model and to provide repeatable automation of the defecation process, as shown in Figure 2.
A central PC was used to coordinate the measurement and control components using a commercially available data interface (NI USB-621x, National Instruments Ltd.) in conjunction with a custom control program on the LabVIEW^{TM} platform (National Instruments). The control program is used to define the operating configuration of the defecation model (e.g. parameters such as stool injection rate), to initiate experiments and to record subsequent data streams with reference to a hardware-timed clock.
Modulation of the ARA was driven using a stepper motor (RS Pro, 535-0366) and spool assembly, controlled by a host PC. The simulated PR muscle is connected to the spool through an inextensible nylon cord and tightened against the anorectum through rotation of the spool. The stepper motor was mounted to a load cell (RS, Model 1004) connected to an amplifier (DR7DC, RDP UK Ltd.) allowing the forces acting on the anorectum by the PR to be measured.
Stool simulant was introduced to the system by controlled injection using a lead-screw linear actuator (SMC, PSAA-60 W) which drove a 500ml syringe containing the stool simulant. Stool leakage from the anal canal is collected in a tray mounted to a second load cell (RDP, RLS005kg) connected to an amplifier (RDP, DR7DC) such that mass, and mass flow rate, can be determined.
A balloon catheter (Medi Plus, 2309) was located within the rectum and fed to a pressure transducer (Utah Medical, Deltran® 6199) connected to an amplifier (RDP, DR7DC) to obtain dynamic measures of pressure inside the rectum during simulated defecation.

A high definition universal serial bus webcam (C920 HD Pro, Logitech) was mounted on the model's supportive framework to provide a sagittal plane video-stream of the rectum at 30 Hz throughout each experiment. The video stream was used to monitor ARA (as shown in Figure 5) and was recorded by the control program for post-hoc analysis.

3. Experimental Methods

A study was defined to investigate the effects of ARA, rectum compliance and sphincter augmentation (using a FENIX device) on continence using the defecation model. This was achieved using an experimental matrix in which the controlled experimental variables were ARA (80°, 90° and 100°), rectum compliance (material was 10A, 20A or 30A DragonSkin) and sphincter state (baseline, with FENIX fitted). The FENIX was fitted for the extremes of the ARA values tested. A series of tests were defined to evaluate each permutation of these experimental parameters across 10 repeats. The control program was used to measure and record Intra-rectal (IR) pressure, PR force and stool mass leakage at 100Hz and the webcam stream at 30Hz.

All experiments were performed at room temperature (25°C). During the tests, 100ml of stool simulate was delivered to the system at a constant flow of 9.26 ml/s, a typical flow rate for stool being passed during defecation (53). Stool simulant was prepared using the same technique as during rheology tests.

The desired ARA was achieved by varying the PR length (through the control program) then analysing the webcam image of the rectum using ImageJ^{TM} (National Institutes of Health) to measure the augmented ARA, as shown in Figure 5. This process was iterated until ARA was obtained within a tolerance of 0.5°. Subsequent repeats at this ARA used the same PR configuration to help ensure consistency.

The FENIX was fitted and configured as specified in the clinical guidance provided with the device. A supplied sizing tool was used to measure the sphincter circumference and thus determine the appropriate length of the device. It was then applied around the recess in the sphincter complex, as shown in Figure 5.

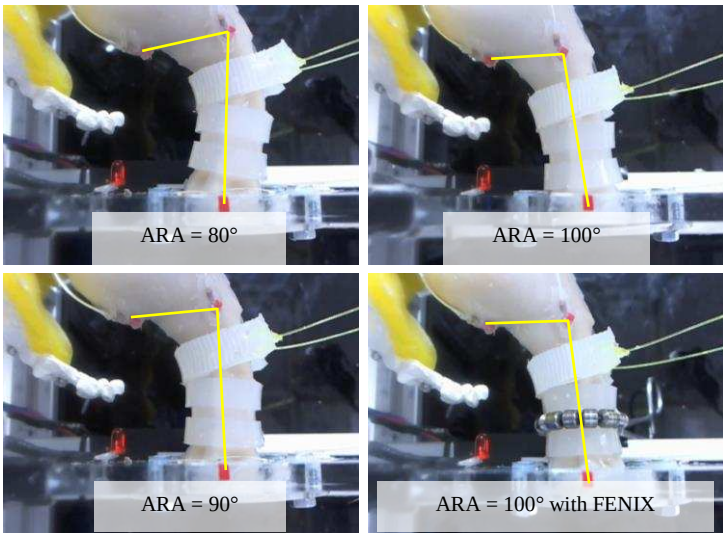

Figure 5 *View of the model rectum for the range of ARA values and sphincter configurations used in the experimental study*

The following protocol was followed for each experiment:

1. Initialise System: Prime the rectum with stool simulant, using a rigid rectum covering shell to prevent distention, until leakage from the anal canal occurs (thus filling the rectum without inducing wall strain).

2. Configure Experiment: Adjust the PR muscle length. Apply the FENIX is fitted if required

3. Initiate Recording: The control program is used to begin recording all sensor and webcam data to a time-stamped datafile.

4. Run Test: The syringe driver is started to inject a pre-metered volume of stool simulant into the rectum at a controlled rate

5. End test: 10 seconds after the system reaches steady state (with respect to stool mass) all data recording is stopped and saved to disk)

4. Results

The full study procedure was successfully completed for each specified experimental configuration. Figure 6 shows typical data obtained from the system for faecal mass passed and IR pressure during simulated defecation, in this case without the presence of sphincter augmentation.

From each experimental dataset, metrics for peak mass, pressure change and time at leakage were calculated, summarised in Table 1 and illustrated in Figure 6. These data demonstrate an effect of ARA on the resultant faecal leakage, evident in the reduction of total faecal mass passed decreasing from 0.0597 kg at 100° to 0.0109 kg at 80°. The associated IR pressures show a similar increase during the initial phase of stool injection

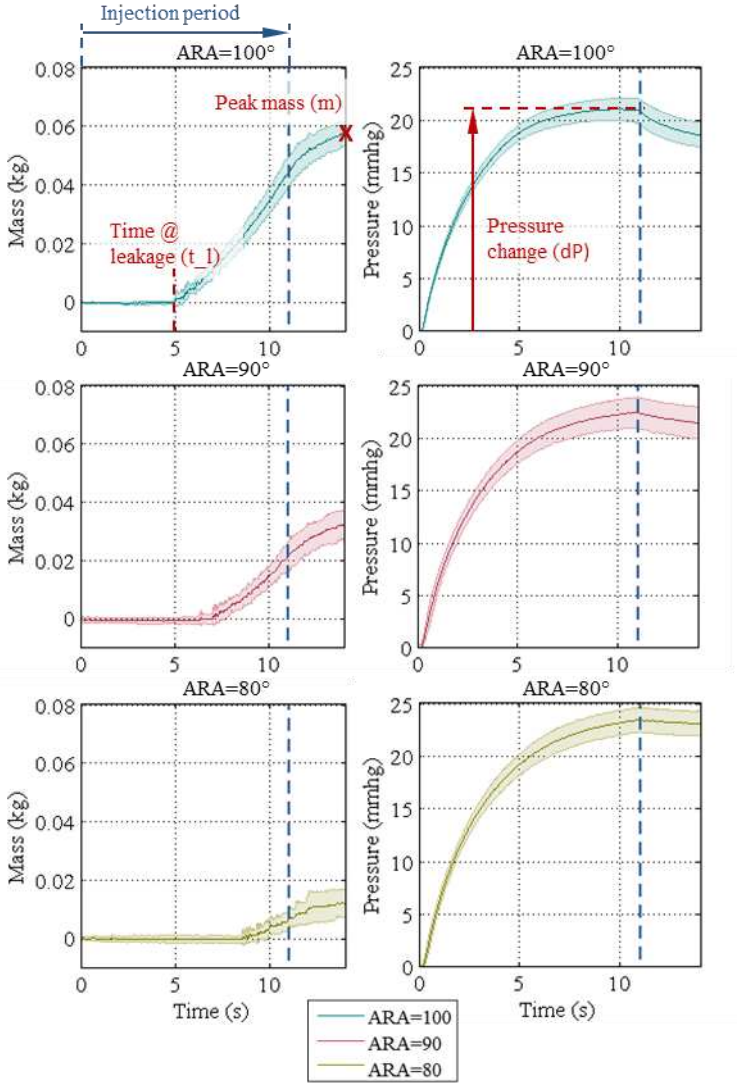

Figure 6 ***Left;*** *Faecal mass passed and* ***Right;*** *IR pressure versus time for different ARA configurations. Each plot shows mean (N=10) in solid with 1 STD as shaded region.*

but diverge as the process approaches steady state, with higher pressures observed for lower values of ARA.
The metrics shown in Figure 7 reveal how the effects of rectal compliance and sphincter augmentation (through the FENIX device) couple with changing ARA.

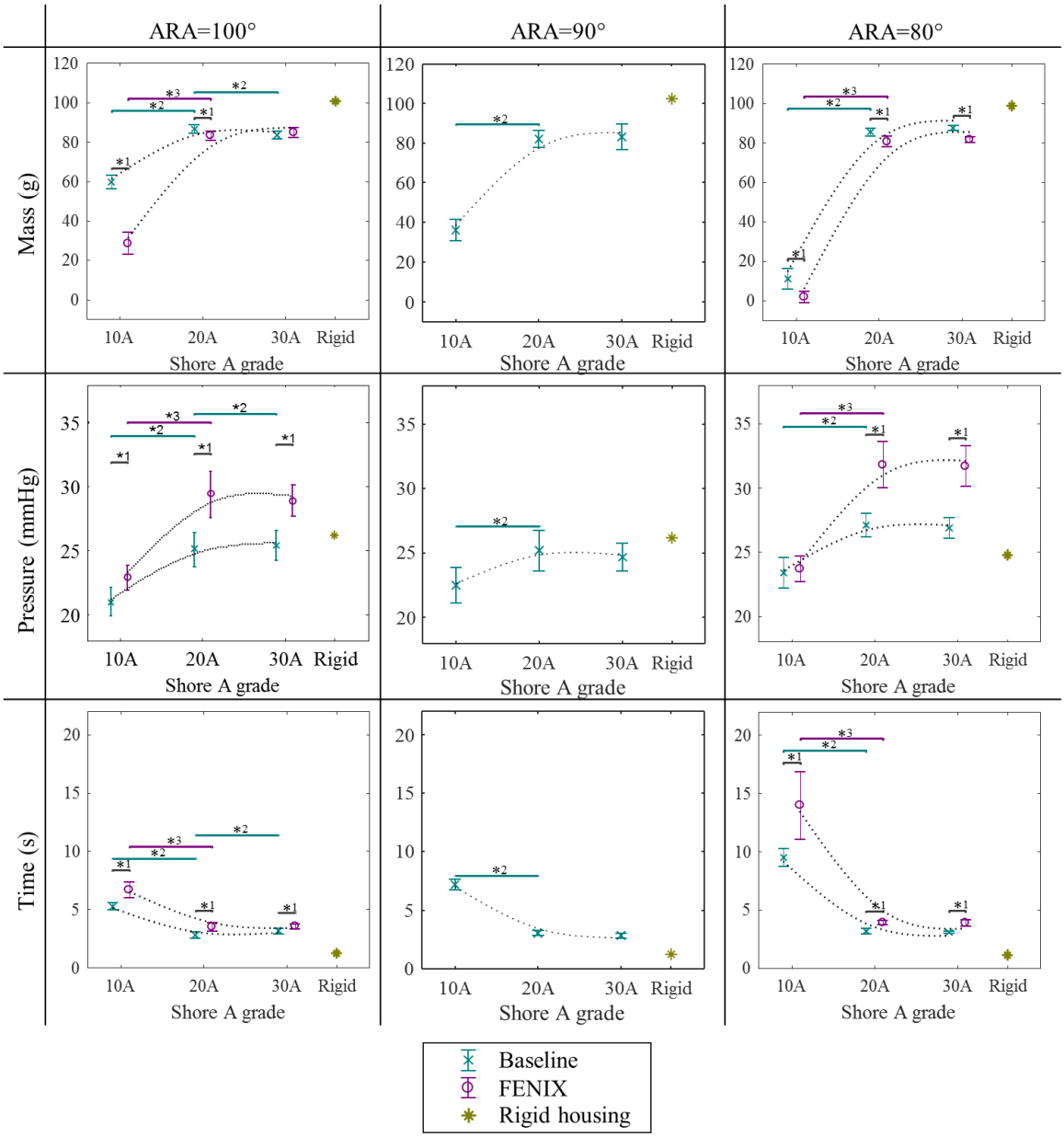

Figure 7 Effects of rectal compliance on faecal mass passed **(top)**, IR pressure change **(middle)** and leakage time **(bottom)**. *Each plot shows mean (N=10) with 1STD error bars. Statistical significance (P<0.05) is shown between configurations of sphincter state (*1), compliance without the FENIX (*2) and compliance with the FENIX (*3)*

Effect of sphincter occlusion on faecal leakage is most pronounced and significant ($p<0.05$) when rectum has a high compliance (10A) and the ARA is obtuse. As shown by

ARA	Compliance	Sphincter config.	m (g)	Sig.	dP (mmHg)	Sig.	t_l (s)	Sig.
100°	10A	Baseline	59.7±3.6	P<0.01	21.0±1.1	P<0.01	5.25±0.29	P<0.01
		FENIX	28.5±5.6		22.9±1.0		6.67±0.70	
	20A	Baseline	86.6±2.2	P<0.05	25.1±1.3	P<0.01	2.79±0.27	P<0.01
		FENIX	83.2±2.2		29.4±1.8		3.50±0.36	
	30A	Baseline	83.4±2.0		25.4±1.2	P<0.01	3.14±0.24	P<0.01
		FENIX	84.7±2.6		28.9±1.2		3.53±0.22	
90°	10A	Baseline	36.3±5.4		22.5±1.4		7.19±0.46	
	20A		82.1±4.4		25.2±1.6		3.04±0.23	
	30A		83.2±6.3		24.7±1.1		2.83±0.22	
80°	10A **(n=9)**	Baseline	10.9±5.2	P<0.01	23.4±1.2		9.49±0.79	P<0.01
		FENIX	1.8±2.9		23.7±1.0		13.96±2.9 **(n=8)**	
	20A	Baseline	85.5±2.0	P<0.01	27.1±0.9	P<0.01	3.17±0.23	P<0.01
		FENIX	80.6±2.8		31.8±1.8		3.91±0.20	
	30A	Baseline	87.4±1.3	P<0.01	26.9±0.8	P<0.01	3.05±0.09	P<0.01
		FENIX	81.5±1.4		31.7±1.6		3.87±0.26	

Table 1 *Mean vales ± 1SE (n=10) for stool injection tests, reporting peak mass (m), pressure change (dP) and time at leakage (t_l) for ARAs of 80° and 100°, significance is reported between the different sphincter states for each ARA*

a reduction of total faecal mass passed from 0.597 kg without the FENIX to 0.0285 kg with the FENIX, with good statistical significance ($p<0.001$). Effect of sphincter occlusion on faecal leakage is least pronounced and insignificant ($p>0.05$) for low rectal compliance (30A) and when the ARA is obtuse. Rectal compliance has a significant effect on faecal leakage for the range of ARA's observed between 10A and 20A. However little variation is observed between total mass passed at rectal compliances of 20A and 30A.

Effect of sphincter occlusion on IR pressure change is least pronounced and least significant for high rectal compliances (10A) and acute ARA's. Effect of the FENIX compared with baseline sphincter occlusion on IR pressure change is small but significant for high rectal compliance (10A) and obtuse ARA's. Effect of sphincter occlusion on IR pressure change is most pronounced and significant for lower rectal compliances (20A & 30A), with little effect apparent from the ARA at these compliances. Tests with a rectal compliance of 20A revealed no significant difference in the total faecal mass passed with and without the FENIX.

The effect of sphincter occlusion on time at faecal leakage is most pronounced and significant for high rectal compliance (10A) and acute ARA's. The effect of sphincter occlusion on time at faecal leakage is less pronounced for a rectal compliance of 20A, but highly significant ($P<0.0005$) for both ARA's. The effect of sphincter occlusion on time at faecal leakage is least pronounced and least significant for low rectal compliances (30A), particularly for obtuse ARA's.

5. Discussion

The results obtained from this study reveal the complex dynamics of the defecation process and the interplay between the mechanisms involved. A particular benefit of this model is the ability to control and time the processes involved, revealing the temporal characteristics of defecation. Once simulated stool starts to be introduced into the system (t=0s) there is a notable time lag before leakage of feacal matter which tends to occur after approximately two seconds have passed. This delay is due to rectal filling whilst holdback pressures are great enough to overcome pressures produced by elastic energy stored in the rectal walls. Consequently this delay varies as a function of rectal compliance, with longer delays observed from more compliant rectum models (which overcome the holdback pressure more slowly as they fill with stool simulant). This has a clinical analogue in those patients with low rectal muscle tone (and so compliance) who find it difficult to generate sufficient driving pressure to defecate.

The effect of the PR modulating the ARA is notable in this study. Upon varying the ARA, a notable difference in leakage was observed between an ARA of 80° and 100°, increasing from 0.0109 to 0.0597 kg. This demonstrates that as the ARA becomes more acute, a greater amount of stool is contained within the rectum during a controlled influx of stool. It would also appear that if a threshold ARA is exceeded, the amount of leakage is drastically reduced, whereas at more obtuse ARA values, small changes in angle have little effect on leakage. This signifies that more acute ARAs produce an elevation in the apparent hold back pressure, and that if this is sufficient in relation to induced IR pressures, faecal leakage will be reduced. Fluctuation of the mass flow rate is apparent for all ARA values tested, with the phase of the fluctuation appearing larger at more acute ARA values and lower flow rates. These are formed as the semisolid exits the system in fluid globules, characteristic of viscous fluids with low surface tension under shear.

In the simulation, augmentation of the sphincter complex using the FENIX device exhibits a similar effect to making the ARA more acute. Additional pressure applied to the anal canal by the FENIX causes a restriction to flow and thus greater retention of faecal matter in the rectum, with consequent increases in IR pressures. The FENIX was particularly effective when used with more compliant rectum models (10A), where a significant difference ($p<0.01$) was observed in peak masses passed from 0.0597 to 0.0285 kg and generated IR pressures of 21.0 and 22.9 mmHg respectively. However, the effect diminishes as variations were observed for less compliant rectum models (20A, 30A) although effects were still significant ($p<0.05$). This demonstrates that while sphincter augmentation can be effective at reducing faecal leakage it does not have universal application.

To defecate effectively requires a reduction in occlusive pressure at the sphincter and achieving a less acute ARA (i.e. straightening the rectum-canal configuration), as observed during proctographic studies (33). These traits are reflected in this study, particularly evident in tests using a low compliance rectum (30A), and an obtuse ARA (100°) for which case there is no statistical significance (p.0.05) for faecal mass passed at baseline (0.0834 kg) and with FENIX (0.0847 kg).

This study demonstrates that to effectively reduce faecal leakage, both anorectal angulation and occlusion pressure at the sphincter should be enhanced. Furthermore, it shows that to retain semisolid material in the rectum, it is not necessary to completely occlude the sphincter. Angulation of the rectum alone provides sufficient resistance to reduce stool leakage. Mean biological ARA values for healthy, nulliparous patients are measured at 104.5±10.3° at rest and 84.5±14.2° during squeeze (13). These values are in agreement with the ARA's observed for the reduction in leakage in this study. This highlights the potential to develop new technologies for FI which do not rely solely on occlusion of the anal canal to maintain continence but also include modulation of ARA. Too much of either mechanism would result in obstructed defecation. A combined and modulated strategy would allow a reduction in occlusive pressures and thereby help to mitigate against the issues of soft tissue erosion and device migration that have previously plagued implantable technology for FI.

To the best of our knowledge there are no other studies reported in literature on the use of physical simulations to understand mechanisms associated with continence, or for the modelling of FI disorders. While computational studies have been developed to model ligament damage on continence (36) and to provide an understanding of the biomechanics of the pelvic floor (37), fundamental mechanisms of continence have not been addressed, such as the ARA and sphincter pressures. This is probably due to the complexities of such modelling parameters. In contrast, use of a physical model allows complex interactions to be modelled with relative modelling ease, and establishes a basis around which refinements can be made in terms of biomechanical properties and physiology. Due to the high variability and complexity of biological systems, the faecal system model has some limitations. The non-linear, anisotropic behaviour typically found in human soft tissue have been approximated with an isotropic silicone model. Furthermore, complex surface interactions which occur between the between the rectum, pelvic floor, bladder and other surrounding tissues have been neglected. The anal canal closure mechanism is complex due to its interaction with adjoining tissue bodies, of particular relevance here is that contraction of the PR effects forces which act to occlude the anal canal, in conjunction with the EAS, due to connectivity of neighboring tissues. These features are only partially approximated in the current model. Lastly, the current model uses a passive model, the active musculature in the rectum and sphincter have been neglected, most significantly the intrinsic contraction of the rectum and anal sphincter complex have not been included. Despite these simplifications it is evident that the behaviour of the model is informative and in agreement with that found in human subjects. Further refinements to this model will help increase its fidelity. In particular, continence relies upon the effects of ARA being augmented with anal sphincter contraction when IR pressures are elevated, and these aspects will form the basis of future enhancements to the model, with the inclusion of intra-abdominal pressures and anisotropic material properties for the soft tissues, and inclusion of tissues adjoining the sphincter, PR muscle and rectum to the pelvis.

6. Conclusion

The physical model has given an insight into the biomechanics of the human faecal system and the combined effects of the ARA and sphincter pressure on continence. As stool simulant is fed into the rectum, the volume expands as elastic potential energy is stored in the rectal walls. When the contraction of the rectum leads to IR pressures which are sufficient to overcome holdback pressures incurred by PR muscle forces, leakage from the anal canal occurs. As pressures reach an equilibrium, stool flows steadily from the anal canal. When the influx of stool into the rectum ceases, leakage continues at a reduced rate until the holdback pressure is sufficient to contain any remaining faeces in the rectum.

This work has shown that, in this simulation, decreasing the ARA increases continence, and augmenting sphincter function improves continence. The study provides rationale that modulation of the ARA could help relieve symptoms of chronic leakage associated with more severe cases of FI, complementing occlusion of the anal canal by existing technology like the FENIX. Future work will increase the fidelity and scope of the physical simulation, as a means to develop new technologies for the treatment of FI.

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References

1. Saga S, Vinsnes AG, Mørkved S, Norton C, Seim A. Prevalence and correlates of fecal incontinence among nursing home residents: a population-based cross-sectional study. BMC geriatrics. 2013;13(1):1.
2. Stoker J, Halligan S, Bartram CI. Pelvic Floor Imaging 1. Radiology. 2001;218(3):621-41.
3. Macmillan AK, Merrie AE, Marshall RJ, Parry BR. The prevalence of fecal incontinence in community-dwelling adults: a systematic review of the literature. Diseases of the colon & rectum. 2004;47(9):1341-9.
4. Read N, Bartolo D, Read M. Differences in anal function in patients with incontinence to solids and in patients with incontinence to liquids. British journal of surgery. 1984;71(1):39-42.
5. Parks A. Royal Society of Medicine, Section of Proctology; Meeting 27 November 1974. President's Address. Anorectal incontinence. Proceedings of the Royal Society of Medicine. 1975;68(11):681.
6. Yoshioka K, Keighley M. Critical assessment of the quality of continence after postanal repair for faecal incontinence. British journal of surgery. 1989;76(10):1054-7.
7. Christiansen J, Lorentzen M. Implantation of artificial sphincter for anal incontinence. The Lancet. 1987;330(8553):244-5.
8. Dubrovsky B, Filipini D. Neurobiological aspects of the pelvic floor muscles involved in defecation. Neuroscience & Biobehavioral Reviews. 1990;14(2):157-68.
9. Snell RS. Clinical anatomy for medical students: Little, Brown Medical Division; 1995.
10. Williams P, Warwick R, Dyson M, Bannister L. Splanchnology. Gray's Anatomy, 36th edn Churchill Livingstone, London. 1980;1318.

11. Parks A, Porter N, Hardcastle J. The syndrome of the descending perineum. Proceedings of the Royal society of Medicine. 1966;59(6):477.
12. Ma S, Leu S-Y, Fang R-H. Reconstruction of Anorectal Angle After Abdominoperineal Resection of Rectum and Anus-An Animal Model. Annals of plastic surgery. 1989;23(6):519-22.
13. Piloni V, Fioravanti P, Spazzafumo L, Rossi B. Measurement of the anorectal angle by defecography for the diagnosis of fecal incontinence. International journal of colorectal disease. 1999;14(2):131-5.
14. Arnold Wald MD, Paul Hyman, M.D., Diane Darrell, A.P.R.N., William E. Whitehead, Ph.D. Bowel Control Problems (Fecal Incontinence) 2013 [Available from: https://www.niddk.nih.gov/health-information/health-topics/digestive-diseases/fecal-incontinence/Pages/facts.aspx.
15. Emmanuel A, Krogh K, Bazzocchi G, Leroi A, Bremers A, Leder D, et al. Consensus review of best practice of transanal irrigation in adults. Spinal cord. 2013;51(10):732-8.
16. Burton JH, Staehle BG. Inflatable artificial sphincter. Google Patents; 1987.
17. NURSE DE, Mundy A. One hundred artificial sphincters. British journal of urology. 1988;61(4):318-25.
18. Sofia C, Rush Jr B, Koziol J, Rocko J, Seebode J. Experiences with an artificial sphincter to establish anal continence in dogs. The American Surgeon. 1988;54(6):390-4.
19. Satava RM, King GE. An artificial anal sphincter. Phase 2: implantable sphincter with a perineal colostomy. Journal of Surgical Research. 1989;46(3):207-11.
20. Christiansen J, Lorentzen M. Implantation of artificial sphincter for anal incontinence. Diseases of the colon & rectum. 1989;32(5):432-6.
21. Christiansen J, Sparsø B. Treatment of anal incontinence by an implantable prosthetic anal sphincter. Annals of surgery. 1992;215(4):383.
22. Christiansen J. Advances in the surgical management of anal incontinence. Baillière's clinical gastroenterology. 1992;6(1):43-57.
23. Torax® Medical I. The FENIX® Continence Restoration System 2014 [Available from: http://www.toraxmedical.co.uk/fenix/.
24. Gregorcyk SG. The Current Status of the Acticon® Neosphincter. Clinics in colon and rectal surgery. 2005;18(1):32.
25. Devesa JM, Rey A, Hervas PL, Halawa KS, Larrañaga I, Svidler L, et al. Artificial anal sphincter. Diseases of the colon & rectum. 2002;45(9):1154-63.
26. Bartolo D, Jarratt J, Read M, Donnelly T, Read N. The role of partial denervation of the puborectalis in idiopathic faecal incontinence. British journal of surgery. 1983;70(11):664-7.
27. Mahieu P, Pringot J, Bodart P. Defecography: II. Contribution to the diagnosis of defecation disorders. Gastrointestinal radiology. 1984;9(1):253-61.
28. Mellgren A, Zutshi M, Lucente VR, Culligan P, Fenner DE, Group TS. A posterior anal sling for fecal incontinence: results of a 152-patient prospective multicenter study. American journal of obstetrics and gynecology. 2016;214(3):349. e1-. e8.
29. Wong WD, Congliosi SM, Spencer MP, Corman ML, Tan P, Opelka FG, et al. The safety and efficacy of the artificial bowel sphincter for fecal incontinence. Diseases of the colon & rectum. 2002;45(9):1139-53.
30. Congilosi S, Spencer M, Madoff R, Jensen L, Wong W, Rothenberger D. The artificial bowel sphincter: long-term experience at a single institution. Dis Colon Rectum. 2002;45:A26.
31. Hajivassiliou C, Finlay I. Effect of a novel prosthetic anal neosphincter on human colonic blood flow. British journal of surgery. 1998;85(12):1703-7.
32. Hajivassiliou C, Carter K, Finlay I. Anorectal angle enhances faecal continence. British journal of surgery. 1996;83(1):53-6.
33. Shorvon P, McHugh S, Diamant N, Somers S, Stevenson G. Defecography in normal volunteers: results and implications. Gut. 1989;30(12):1737-49.
34. Bartolo D, Miller R, Mortensen N. Sphincteric mechanism of anorectal continence during Valsalva manoeuvres. Coloproctology. 1987;9:103-7.
35. Chanda A, Unnikrishnan V, Roy S, Richter HE. Computational Modeling of the Female Pelvic Support Structures and Organs to Understand the Mechanism of Pelvic Organ Prolapse: A Review. Applied Mechanics Reviews. 2015;67(4):040801.

36. Brandão S, Parente M, Mascarenhas T, da Silva ARG, Ramos I, Jorge RN. Biomechanical study on the bladder neck and urethral positions: simulation of impairment of the pelvic ligaments. Journal of biomechanics. 2015;48(2):217-23.
37. d'Aulignac D, Martins J, Pires E, Mascarenhas T, Jorge RN. A shell finite element model of the pelvic floor muscles. Computer Methods in Biomechanics and Biomedical Engineering. 2005;8(5):339-47.
38. Martins J, Pato M, Pires E, Jorge RN, Parente M, Mascarenhas T. Finite element studies of the deformation of the pelvic floor. Annals of the New York Academy of Sciences. 2007;1101(1):316-34.
39. Zhang Y, Sweet RM, Metzger GJ, Burke D, Erdman AG, Timm GW. Advanced finite element mesh model of female SUI research during physical and daily activities. Stud Health Technol Inf. 2009;142(1):447-52.
40. Bhattarai A, Frotscher R, Sora M-C, Staat M. A 3D Finite Element model of the female pelvic floor for the reconstruction of urinary incontinence. Rev Urol. 2014;16(5):S2-S10.
41. Janda Š, Van Der Helm FC, de Blok SB. Measuring morphological parameters of the pelvic floor for finite element modelling purposes. Journal of biomechanics. 2003;36(6):749-57.
42. Silva M, Brandão S, Parente M, Mascarenhas T, Natal Jorge R. Biomechanical properties of the pelvic floor muscles of continent and incontinent women using an inverse finite element analysis. Computer Methods in Biomechanics and Biomedical Engineering. 2017;20(8):842-52.
43. 3Dircadb. 3D image reconstruction for comparison of algorithm database June 2013 [Available from: http://www.ircad.fr/softwares/3Dircadb/3Dircadb.php?lng=en.
44. Dall F, Jørgensen C, Houe D, Gregersen H, Djurhuus J. Biomechanical wall properties of the human rectum. A study with impedance planimetry. Gut. 1993;34(11):1581-6.
45. Christensen MB, Oberg K, Wolchok JC. Tensile properties of the rectal and sigmoid colon: a comparative analysis of human and porcine tissue. SpringerPlus. 2015;4(1):1-10.
46. Liu J, Guaderrama N, Nager CW, Pretorius DH, Master S, Mittal RK. Functional correlates of anal canal anatomy: puborectalis muscle and anal canal pressure. The American journal of gastroenterology. 2006;101(5):1092-7.
47. Sørensen G, Liao D, Lundby L, Fynne L, Buntzen S, Gregersen H, et al. Distensibility of the anal canal in patients with idiopathic fecal incontinence: a study with the functional lumen imaging probe. Neurogastroenterology & Motility. 2014;26(2):255-63.
48. Gourcerol G, Granier S, Bridoux V, Menard J, Ducrotté P, Leroi A. Do endoflip assessments of anal sphincter distensibility provide more information on patients with fecal incontinence than high-resolution anal manometry? Neurogastroenterology & Motility. 2016;28(3):399-409.
49. Alqudah M, Gregersen H, Drewes A, McMahon B. Evaluation of anal sphincter resistance and distensibility in healthy controls using EndoFLIP©. Neurogastroenterology & Motility. 2012;24(12).
50. Li D, Guo M. Morphology of the levator ani muscle. Diseases of the colon & rectum. 2007;50(11):1831-9.
51. Alkhouli N, Mansfield J, Green E, Bell J, Knight B, Liversedge N, et al. The mechanical properties of human adipose tissues and their relationships to the structure and composition of the extracellular matrix. American Journal of Physiology-Endocrinology and Metabolism. 2013;305(12):E1427-E35.
52. Woolley S, Cottingham R, Pocock J, Buckley C. Shear rheological properties of fresh human faeces with different moisture content. Water SA. 2014;40(2):273-6.
53. Lestár B, Penninckx FM, Kerremans RP. Defecometry. Diseases of the Colon & Rectum. 1989;32(3):197-201.