Author's Reply

Invited comment: A new theory of anorectal function
(D. Chatoor, A. Emmanuel - issue 4, 2008)

We have been interested to study Drs Chatoor and Emanuel's analysis and comments on our work. We note that they do not comment overall on the musculo-elastic theory itself, or on the concepts underlying our series of publications. As we explained in the preamble we sought to test the musculo-elastic theory of pelvic function and continence by challenging its predictions, a method proposed as the basis of the scientific method by the late Sir Karl Popper, and regarded as the most rigorous test procedure. Thus the studies we reported were designed to test the musculo-elastic theory by seeking direct tests that would refute the theory.

For example, in Study No 1, if we failed to demonstrate the three predicted directional movements, then the Theory would be invalidated. Since the theory states that faecal incontinence (FI) is caused by lax suspensory ligaments, in this case, the pubourethral and uterosacral, and we found a >80% cure of FI on repair of those ligaments, then the theory remains valid, although not necessarily proven. Popper held that nothing in science is ever finally proven, but that any theory remains valid until it is refuted. The classic example of this principle in Physics is Newton's Laws of Gravity, which required modification in relation to Einstein's Theory of Relativity, yet still remain accurate enough that they are used to calculate satellite orbits.

We define idiopathic FI as a disorder of faecal continence despite a normal external and internal anal sphincter, and in the absence of any causative central neurological or other disorder.

Chatoor and Emanuel state that "the aetiopathogenesis of urinary and faecal incontinence is rarely a singular process. Insults to the pelvic floor are usually multiple (including chronic straining, increased intra abdominal pressure effects, parturition and the menopause)". This statement reflects generally held opinion but is essentially phenomenological rather than mechanistic. It does not lead to understanding of the abnormal functional forces acting on the pelvic floor leading to incontinence. Our musculo-elastic theory, on the other hand leads to predictions of abnormalities in pelvic floor function, and that their correction should mitigate FI when present. It also offers functional explanation of symptoms such as straining and abdominal pressure effects as secondary manifestations of connective tissue damage, especially to the suspensory ligaments, because this abnormality will cause unloading of muscle forces, and weakening of the vector acting across the pelvic floor during its normal function.

The comments made by Chatoor and Emanuel regarding collagen abnormalities as risk factors for the development of FI, and probably also applicable to stress urinary incontinence, regarding collagen abnormaities are entirely in accord with our perspective of these problems, and are especially valuable since they should be amenable to experimental testing.

We note that Chatoor and Emanuel have misinterpreted some of our ideas, and comment as follows:

Experimental study No 1.

We did not set out to alter the puborectal angle, which we agree is not in itself crucial to faecal continence. We set out to restore the functional integrity of certain intrapelvic ligaments, in order to restore muscle forces. Any resultant change in the puborectal angle would therefore be secondary to restoration of ligamentous function and muscle force vectors acting across the pelvic floor.

We regard the outer longitudinal muscle of the rectum (LMA) as contracting against, and angulating, the levator plate, which is attached to the ligamentous structure via its fascial coverings. We do not suggest the LMA merges with the uterosacral ligaments.

We have not claimed to quantify muscle forces, but rather to confirm the direction of these muscle forces. We agree that the position of the ligaments is necessarily only an estimate, but it is evident by studying the radiographic illustration of the normal subject with the pelvic floor in the "resting position", that there is a definite bend at midurethra, which is consistent with the anchoring point of the three muscle forces we have described. With regard to the uterosacral ligaments, we compared the straining to the resting data. The downward angulation of the tip of the levator plate is the directly in line with the position of the cervix, the insertion point of the uterosacral ligaments.

It is not possible to assess laxity in the living subject, even with MRI.

Experimental study No 2.

We have indeed suggested a possible mechanism; that is, restoration of the pubourethral anchoring point for levator plate contraction and we provide a diagram to illustrate this concept.

Experimental study No 3.

Chatoor and Emanuel offer an explanation of the phenomenon described on the basis of increased sensory input caused by digitation of the vagina, leading to an enhanced reflex response. We prefer the notion, based on clinical examination that this change in function was simply due to a mechanical change associated with restitution of abnormalities in function associated with lax connective tissue structures. This concept is testable by more detailed experimental work in affected patients.

Experimental study No 4.

The bladder was full of urine when the testing was done. Although we accept that there is discussion regarding the best techniques for measurement of abdominal pressure changes, any criticisms regarding the methods we used apply equally to both the squeezing and straining maneouvres, and so would apply to both.

Experimental study No 5.

Chatoor and Emanuel make a definitive statement "One of the frequent causes of faecal incontinence in the elderly is internal sphincter atrophy." This may or may not be so.

We must emphasize that we are suggesting a different approach to understanding the functional basis of faecal incontinence and stress urinary incontinence; i.e., that it may be due primarily to ligamentous laxity. This concept does not exclude a role for other factors, especially anal sphincter tears, and even internal anal sphincter dysfunction, but we do not think the latter is a likely cause of faecal incontinence as a unique and solitary abnormality. We agree entirely that we have taken an arbitrary 2 mm definition of internal anal sphincter thinning on the basis of advice from our Radiologist. What we did demonstrate was:

a) Only a minority of patients with FI had IAS thinning.

b) IAS thinning had no impact on whether such patients were cured following the tape insertion procedure.

One has to conclude therefore that IAS defect was not a major cause of FI in our patients.

As regards the ultrasound probe, a 7 Mhz probe may be less sensitive but its use was consistent, and this does not therefore alter our conclusions.

Once again we point out that our observations and interpretations are open to further experimental study, which will verify or refute them.

Experimental study No 6.

We note the comments. However, the presenting symptoms were relieved by the procedure, and we have presented our observations and interpretation of the mechanism of benefit.

Experimental study No 7.

We agree that the puborectalis is an important part of the anorectal closure mechanism; indeed, this has been known for more than 30 years. We regard the main function of the puborectalis muscle as anchoring the anorectum, so that closure of the anus can be effected by stretching the rectum backwards and downwards. A relevant analogy might be maintenance of urinary continence after excision of he distal part of the urethra. In that case, we consider it is the backward/downward stretching which narrows the urethral tube, exponentially raising its resistance according to the 4^{th} power law of Poiseuille.

Experimental study No 8.

The histological findings described were features that do not occur in normal subjects, as compared with historical data on the anatomy and histology of these perineal muscles. Biopsies from control subjects are clearly unethical, not least since normal subjects are not subject to surgical procedures. We have made the point that histological features in muscles in which muscle fibres could be detected were consistent, in part, with changes found in limb muscles after tenotomy. The essential feature of this abnormality is that it is due to unloading of muscle by the tenotomy, or by ligamentous laxity in the case of our pelvic floor biopsies, and that these changes are reversible.

Even a weak muscle, caused by direct injury, denervation or myopathy can function better if its ligamentous attachments are reinforced. People with severe muscle atrophy, as in inherited neuropathies, poliomyelitis or myopathy can remain mobile as long as joints and ligaments are intact. It is well-recognized that when there is stretching of ligaments, mobility may be lost.

We agree however, that other than stating that the tape provides a strong insertion point, we did not demonstrate how the contractility improved.

Experimental study No 9.

A strength of this report is that it represents the experience of a generalist gynaecologist who based his report on a preoperative questionnaire. Symptoms are important. Gradation of the severity of incontinence would not have contributed to the aim of testing the hypothesis although we agree that will be necessary in attempting to better define the indications for the procedure. The important point to note is that patients were cured of their FI symptoms. This report reflects the remarkably successful results of one practitioner.

Experimental study No 10.

Chatoor and Emanuel take issue with our algorithm. This is intended to reflect our experience. We agree that it differs in some respects from other algorithms of pelvic floor dysfunction and, of course, like all algorithms it certainly oversimplifies the issues. We draw attention to the various algorithms in the book "The Pelvic Floor", (Pemberton J, Swash M, Henry MM, WB Saunders 2002) which also represent summaries of ideas and practice. The unexpected placement of certain symptoms, such as nocturia, unexpectedly as a posterior defect represents our experience of its resolution following a posteriorly-directed, reconstructive tape-insertion procedure.

In reply to Chatoor and Emanuel we again point to the results described – a large percentage of these patients were cured or improved of their FI symptoms following operations that only repaired suspensory ligaments. The symptoms and their relation to the algorithm have been validated in two ways.

1. Use of 'simulated operations'; i.e., anchoring specific ligaments and observing the effect on symptoms such as urge, SI and pelvic pain.

2. Tracking pre-operative symptom fate after site-specific repair to the three zones in large numbers of patients. The surgical techniques are fully described elsewhere in the literature.

We agree with Drs Chatoor and Emanuel, that the algorithm may not have described the functional defects sufficiently for optimal cure of all patients in this group. For example, some patients required a second procedure directed to another pelvic floor ligament. However, because we wished to assess whether laxity in either the anterior or posterior suspensory ligaments (or both) caused FI, we followed the protocol described. The operations are minor in-patient procedures, so that an argument can be made for repairing both the anterior and posterior in all patients with FI.

We agree that it would have been helpful to repeat the tests in failed cases. However, this re-examination was dependent on patient consent.

Mean anal pressure and functional anal length were determined by standard methods, using balloon manometry, in the Dept of Colorectal Surgery at the Royal Perth Hospital.

Our results regarding the difficulty in securing reliability in pudendal nerve terminal motor latencies speak for themselves. The neurological co-author (M Swash), who introduced this technique as an experimental method in the 1980s never intended that it should be used in clinical practice in individual patients, for the very reason that the length of the terminal segment of the pudendal nerve over which the measurement was made could not be verified and might vary from test to test. At the time this method provided useful verification of damage to the pudendal nerve, when treated as a change in group data, but the standard deviation of the grouped results was always too great for application in individuals. Our results reported here reflected these comments.

With regard to "validated scoring sheets' we believe that nothing could be more validating than a patient stating she does not soil anymore. This is a yes/no response that answers the question absolutely.

Poisseuille's Power Law may be confusing. However, we set out to explain why some patients with no obvious anatomical defects were incontinent, or had emptying problems. By regarding the anorectum as a tube which is opened or closed by muscle forces, this explanation becomes rationalized in terms of physics. If we then understand that these same muscle forces effectively contract against suspensory ligaments, as do all muscles in the body, then the argument becomes rationalized also in biomechanical terms.

Experimental study No 11.

Once again, Chatoor and Emanuel have ignored our results – a large percentage of these patients were cured

or improved of their FI symptoms with simple out-patient, daycase, operations which repaired suspensory ligaments.

Cystocoeles were present in a number of these patients and required repair. This necessity afforded us the opportunity to test whether this repair also improved the FI cure rate; it did not.

We agree with Chatoor and Emanuel that it would have been helpful to add subgroup data in the paper. However, the numbers of patients involved were too small to allow this. We agree that the question, which ligament causes which symptom is of critical importance. Our approach as stated, was to restore all the anatomical defects, based on the principle of "restore the anatomy, and you will restore the function". Further work may yet address this question.

Experimental study No 12.

Chatoor and Emanuel make no comment on the major observation made in this report by Abendstein; that is, a large percentage of these patients were cured or improved of their rectal intussusception and obstructive symptoms by repairing the posterior suspensory ligaments, thus avoiding a major invasive abdominal procedure. From our perspective, this observation validates another of the musculo-elastic theory's predictions. All these patients were symptomatic prior to surgery.

Again, we do agree that subgroup data in this study would have been beneficial to our aims.

We believe overwhelmingly that psychological problems in this disorder are secondary to the incontinence. It is remarkable how many such problems disappear overnight after successful surgery.

Final comments

In their criticisms Chatoor and Emanuel have unmasked what is perhaps a major imperative for future research, close collaboration with colleagues whose knowledge and advice will provide more information, and better test the musculo-elastic theory.

We have presented information that supports a concept that emphasizes the role of ligaments applied to the muscle forces activating anorectal closure (continence) and evacuation. Our aim has been to indicate a new direction for treatment and research. We hope that the many doubts and questions raised by Chatoor and Emanuel will be taken up and used to further test the musculo-elastic theory. What we have done can only be considered a small beginning which, we trust, will unfold into a rich new era of research in anorectal function and dysfunction.

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