PREGNANCY AND THE LOWER URINARY TRACT: PART III

THE SURGICAL TREATMENT OF URINARY STRESS INCONTINENCE RESULTING FROM PREGNANCY

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The results of the investigations as described in Parts I and II lead to the conclusion that hormonal influences are the cause of the common urinary symptoms of pregnancy and that infection plays a minor part. Although pressure effects have not been assessed, they are thought to be of transient nature and not to be of importance in the causation of persistent postpartum symptoms. Of the latter, stress incontinence is one of the most distressing. It often persists after pregnancy without obvious anatomical evidence to explain its occurrence. The purpose of this paper is to show how the knowledge gained from the described investigations could be applied in order to formulate a rational approach to the surgical treatment of stress incontinence.

It is therefore proposed to describe the rationale and technique of a surgical procedure, together with its results, which was undertaken for the treatment of stress incontinence in the female without associated genital prolapse. In the discussion of the operation a short review of modern knowledge and thought on the problem of stress incontinence will be made. Supporting evidence, also from the literature, will be given for the concept on which the operation is based. Lastly, the formulation of a suggested approach to the treatment of all cases of stress incontinence in the female will be attempted.

It has been shown that the extensive collagenous component of the lower excretory apparatus, not only the ureters but also the bladder in the vicinity of the urethrovesical junction, is profoundly influenced by hormonal factors during pregnancy.

If the connective-tissue layers in the bladder wall increase by approximately 100% in thickness, as it appears to be from the foregoing investigations, it would lead to an over-all increase in thickness of the bladder wall of approximately 25%. Microscopical evidence also seems to indicate that the hypertrophy of the connective-tissue layers is associated with an increased elasticity.

It is thought that the hormonal modifications of the bladder wall result in a 'relaxation' of the firm holding function of the inelastic investing fascial collar surrounding the urethrovesical junction. There is thus an increase in diameter of the lumen of the proximal urethra which promotes urinary frequency, and in certain cases leads to loss of urethrovesical competence with resultant stress incontinence. Whereas after pregnancy the vast majority of cases return to normal, it is thought that in some the condition persists for some unknown reason. This course of events could then explain the aetiology of patients presenting with stress incontinence without obvious anatomical defects.

The purpose of the operation to be described is to restore the integrity of the investing fascial collar surrounding the urethrovesical junction.

MATERIAL AND METHOD

In the 7-year period January 1960 - 31 December 1966, I operated on a total of 65 patients. They all presented with urinary stress incontinence not associated with pelvic floor prolapse. All the patients in this series had a history of previous pregnancy.

Diagnosis

In the clinical diagnosis of urinary stress incontinence the history is important so as to distinguish the nature of the condition, i.e. between continual stress incontinence or the symptom occurring only in conjunction with a full bladder. Also it is necessary to eliminate urgency incontinence which complicates urinary infection. The latter condition creates a problem because its existence does not exclude a true stress incontinence. However, if after adequate treatment of a urinary infection stress incontinence persists, such a case is regarded as one of true stress incontinence. In the physical examination it is important to test for stress incontinence not only in the recumbent

position but also in the erect position. In the majority of instances a voluntary sphincteric defect is, of course, also revealed in the physical examination and presents as a urethrocele with or without cystocele. (Cystocele does not necessarily indicate urethrovesical incompetence.) Difficulty arises when there is no obvious anatomical cause for the condition. In these cases lateral cysto-urethrography is helpful, although correct interpretation of such radiographs is not easy. Experience, however, shows that most of these cases are the result of urethrovesical junction incompetence. As all the cases in the series were in the latter group, additional radiographic investigations were performed in about one-third of the cases. Successful radiographs showed distinct loss of angulation at the urethrovesical junction on the lateral views.

There is one form of urinary incontinence which can easily be confused with stress incontinence. This is seen in cases of severely diminished bladder capacity, sometimes associated with previous surgery or severe infection resulting in marked pericystic fibrosis. In such cases the maximum bladder capacity is in the vicinity of 150 - 200 ml. If such a condition is suspected, preliminary cystometric studies are indicated.

Operative Procedure

The operation evolved from, and is a modification of, the Marshall-Marchetti-Kranz[1] procedure. In the same way as the latter, it is equally useful as an adjunct to an intraperitoneal operation such as abdominal hysterectomy, or as a primary extraperitoneal procedure. After the insertion of a Foley's catheter into the bladder, the cave of Retzius is opened, preferably through a Pfannenstiel incision. (There is no objection to a midline incision.)

This operation is not concerned with the urethra or the peri-urethral tissues or the pubocervical fascia, as it is not thought that the approach through the cave of Retzius is indicated for simple urethrocele which is more properly dealt with by conventional urethroplasty.

With blunt dissection the bladder is dissected clear of the perivesical tissues. The urethrovesical junction is defined with the help of the Foley's catheter with the bulb inflated. On each side of the urethrovesical junction and just superior to the bulb, sutures are inserted through the 'collar' of the investing fascia which surrounds it. These sutures are placed according to circumstances, but cardinal principles in applying them are:

1. The sutures must be at the level of the urethrovesical junction on each side, and they must be correctly opposed on each side.

2. Beginning as far posteriorly as possible, good 'bites' of investing fascia must be taken; preferably two 'bites' for each suture. Blood-vessels must be avoided. Each 'bite' incorporates approximately ¼ inch of investing fascia, and the distance between the 'bites' is also approximately ¼ inch. The investing fascia (fibrosa) of the bladder is relatively thick and there is little danger of penetrating the bladder wall right through the mucosa. After taking two 'bites' the posterior suture is tied tightly and the investing fascia is then closely approximated, resulting in plication of the fibrosa. The number of sutures inserted in this fashion depends on circumstances, in order to satisfy the need of restoring an adequate collar of connective tissue. Two or 3 sutures are usually enough (Fig. 1).

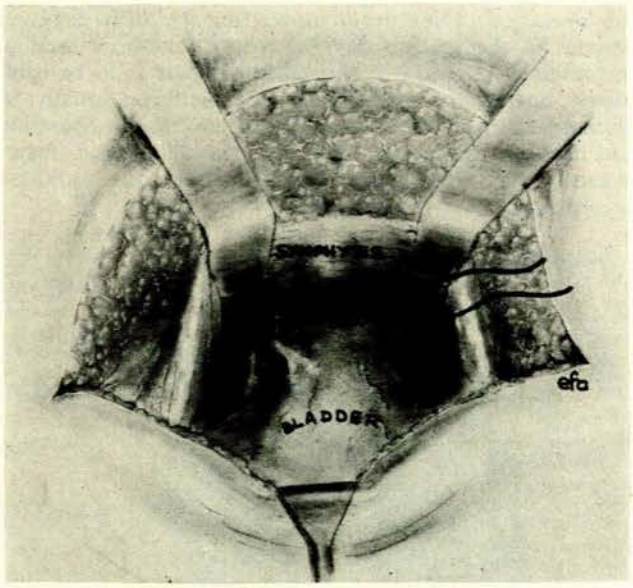

Fig. 1. The round-bodied Mayo needle takes two 'bites' of investing fascia of the bladder on the lateral aspect of the urethrovesical junction.

3. On the anterolateral aspect of the urethrovesical junction a similar suture is placed. The ends of this suture are not cut, and, should there be marked sagging of the urethrovesical junction as a result of tearing of the urethral suspensory ligament, they may be attached to the posterior symphyseal ligament. There should never be difficulty in approximating the investing fascia to the posterior symphyseal ligaments, as this would create abnormal tension. It must be possible to tie knots with ease, and no taut strings of gut must stretch between symphysis and bladder. Should there be difficulty in obtaining adequate plication of the urethrovesical junction, two of the more lateral sutures can also be passed through the symphysis. It is important to stress that the purpose of this operation is not to approximate the bladder to the symphysis primarily but only to plicate the urethrovesical junction in such a fashion as to reduce the diameter of the fibrosal 'collar' which surrounds it. If, however, the latter sags far posteriorly as a result of tearing of the urethral suspensory ligament, it is imperative to restore normal anatomy (Figs. 2 and 3).

4. Suture material must be absorbable. In the present series No. 2 Plain London Hospital catgut was used with a round-bodied Mayo needle. Plain catgut was preferred because it promotes local fibrosis which binds the fascia with collagen. Plain catgut absorbs rapidly, and if there is no infection in the perivesical tissues there is very little chance of imprisoning the bladder in a rigid cage of fibrous tissue which would negate the purpose of the approach, namely to restore normal morphology with as little interference as possible.

Postoperative Treatment

Usual routine postoperative care is taken. Constant

sterile urinary drainage through a Foley's catheter is important for the first 48 hours. In view of this, antibiotic cover is essential. Ambulation is encouraged 24 hours after the operation. Clips are removed on the fifth day and sutures on the seventh day. If a drain has been used in the cave of Retzius, it is shortened daily so as to be completely out by the fifth day. The patient can usually be discharged on the seventh or eighth day. If the operation has been performed in conjunction with another major intraperitoneal procedure, circumstances are adjusted accordingly.

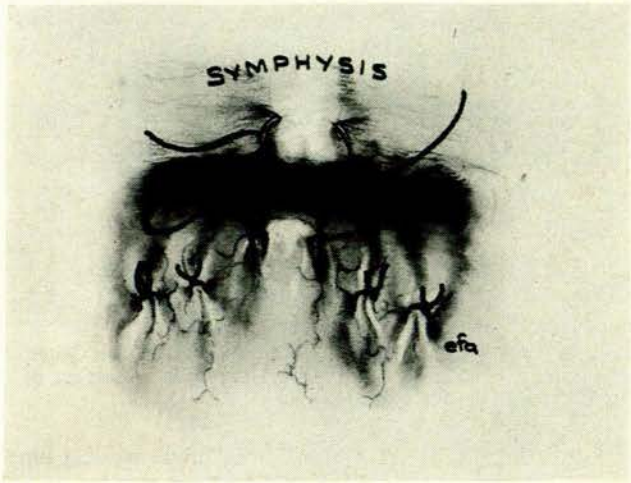

Fig. 2. The plicated urethrovesical junction is approximated to the posterior symphyseal ligament.

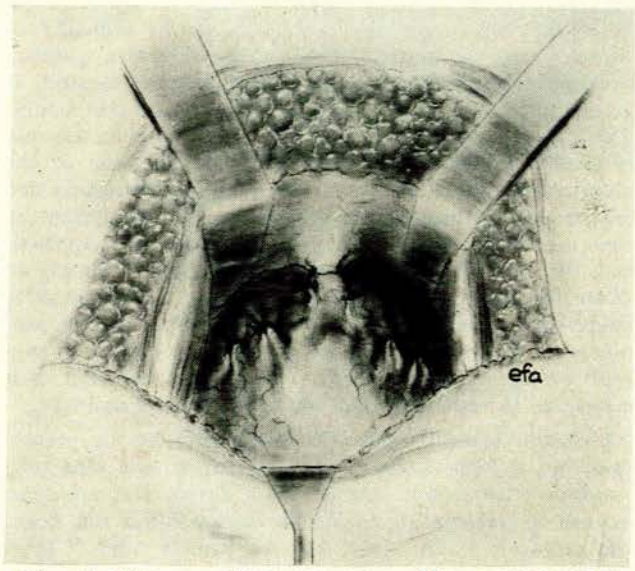

Fig. 3. The completed operation. The urethrovesical junction is plicated and firmly attached to the posterior symphyseal ligament.

Complications

In this series complications have been few and of relatively minor importance: Haematuria of minor nature was noticed in 2 cases. In both cases catheter drainage was prolonged for one week under antibiotic cover and after removal of the catheter urinary function was normal. Haemorrhage and oozing in the cave of Retzius was the most troublesome complication. In 9 cases the haemorrhage was difficult to control during the operation and drainage with corrugated rubber was instituted. In all these cases the drains were removed on the fifth day without subsequent problems. In one case the cave of Retzius was quite dry at the end of the operation and drainage was not instituted. Subsequent haemorrhage, however, occurred and a huge perivesical haematoma formed within a matter of hours. The wound was opened and the haematoma evacuated. One of the sutures had perforated a small vein which had kept on bleeding. After the bleeding was controlled, the sutures were found to be still intact. Drainage was instituted and this patient made an uneventful recovery.

Serious postoperative infection was never a problem.

Leakage of urine never occurred at the operation site.

A breakdown of sutures at the urethrovesical junction or at the attachment of the latter to the symphysis is impossible to assess clinically. The likelihood of this occurrence should, however, be kept in mind, especially if the patient is suffering from a chronic respiratory infection. With marked postoperative coughing this is very likely to occur. It is suspected that the failures which did occur resulted from breakdown of sutures, or, less likely, from inadequate plication of the urethrovesical junction.

Follow-up Examination

After discharge from hospital the patients were examined at a routine 6-weeks postoperative examination, and again 3 months after the operation. On this occasion they were instructed to report any recurrence of the symptom and also to report annually either by letter or by telephone. Some of the earlier cases did not receive precise instructions and were difficult to trace later on. Of these, 4 were lost to follow-up. After 3 years the follow-up was discontinued in those cases where the patients were not seen regularly for other conditions. The results are therefore based on a 3-year follow-up period.

It is interesting to note that, except for a single instance, there was no recurrence of the stress incontinence after the third postoperative month; in fact all the other failures reported a recurrence of the condition at the 6-weeks examination. In this connection it is notable that the cases were all relieved of their symptoms in the immediate postoperative period and that failure was sudden and complete. It is thought that this was a result of breakdown of sutures or a breakdown of early collagen in the plicated fibrosa before fibrosis was established.

RESULTS

During the 7-year period from January 1960 to December 1966, operations were performed on 65 patients. For comparative purposes the cases were grouped into 4 categories, the first being those cases that were operated on 3 and more years before this report. The second group comprised the cases operated on between 2 and 3 years preceding the report, and the third group comprised the cases done between 1 and 2 years previously. The last

group consisted of the cases who had had their operation during the last year.

In the first category (i.e. 3+ years) a total of 35 cases were operated on, of which 28 were complete cures. There were 3 outright failures and 4 patients were lost to follow-up. In this group there were 2 patients who were previously operated on for stress incontinence, one patient having had a vaginal repair 1 year before the operation and another patient having had a vaginal hysterectomy and repair 6 months before this operation.

In the 2 - 3-year group 15 cases were operated on. There were 14 cures and 1 failure. None of these cases was lost to follow-up. One of these cases had two previous failed vaginal attempts at repair, one previous Aldridge sling repair which failed and one Marshall-Marchetti repair which failed. These operations were done in the 5-year period preceding this operation, and the patient is now cured.

In the 1 - 2-year group, 6 patients were operated on, in whom there were 6 cures and no failures. None was lost to follow-up. One of these patients had stress incontinence to such a marked degree that it verged on total incontinence. Another one of these patients had a very marked stress incontinence after only one previous pregnancy, which had resulted in a premature delivery at 18 weeks. This patient had no anatomical defects whatsoever, and presented the appearance of a nulliparous female.

In the final group (i.e. those 0 - 1 year) 9 cases were submitted to surgery, and so far there have been 6 cures and 1 which is regarded as a doubtful failure, as well as 2 outright failures. The doubtful failure was the exceptional case of a 63-year-old multiparous woman who had had an abdominal hysterectomy 7 years previously. With this operation a Marshall-Marchetti was done which was unsuccessful. She was subsequently investigated and treated in different institutions and as far as it could be ascertained another Marshall-Marchetti was performed on her. Stress incontinence was clinically demonstrable. In addition to this stress incontinence she complained of total incontinence which occurred approximately every hour after emptying her bladder. This occurred only in the erect position, and when the bladder voided itself there was a sudden gush of a large volume of urine which was absolutely uncontrollable. Cystometry showed that the patient had a bladder capacity of only 175 ml. At operation a gross pericystic fibrosis was found. In addition, the bladder wall, in the vicinity of the urethrovesical junction, was tightly adherent to the pubic rami on both sides and it was under noticeable tension. The effect was of two 'pterygiae' of bladder on each side of the urethrovesical junction stretching towards the pubic rami. The bladder had to be freed from the bone by means of a sharp dissection and an attempt was made to restore normal anatomy as far as possible. It was also noted that the diameter of the urethra in the vicinity of the urethrovesical junction was markedly enlarged, and in this area only the plication was done. Postoperatively her stress incontinence was cured provided she emptied her bladder regularly at hourly intervals. If, however, she did not do this, she would experience a sudden gush of incontinence. It was thought that this was the result of diminished bladder capacity. Although the stress incontinence in this patient was cured, she is recorded as a failure.

The absolute cure rate of all 65 cases operated on is 83·0% and the corrected cure rate is 89·1%. The absolute cure rate of the cases in the group 3+ years is 80·7% and the corrected cure rate is 89·7%. In the group 0 - 3 years the absolute and the corrected cure rate is 86·6%. These results are tabulated in Table I.

TABLE I. RESULTS OF SURGERY IN 65 CASES OPERATED ON FROM JANUARY 1960 TO DECEMBER 1966

Group (years)	*No.*	*Cures*	*Failures*	*Lost to follow-up*	*Cure rate*	
					Absolute	*Corrected*
0 - 1	9	6	3	0		
1 - 2	6	6	0	0	86·6%	86·6%
2 - 3	15	14	1	0		
3+	35	28	3	4	80·7%	89·7%
Total	65	54	7	4	83·0%	89·1%

DISCUSSION

For maintenance of continence the importance of adequate angulation between the bladder and urethra, as seen on lateral cysto-urethrography, has been stressed repeatedly over the past decade by most investigators in the field.[2-10] Marchetti[11] has most emphatically stressed the importance of maintenance of this angle in order to conserve urinary continence, regardless of bladder position in the pelvis or orientation to the long axis of the body such as found in prolapsed conditions, e.g. cystocele. This he has repeatedly shown in a large number of cystographic studies inclusive of ciné studies and voiding cysto-urethrograms.

In the interpretation of loss of angulation the conclusion is implicit that dysfunction exists in the region of the urethrovesical junction. This finding has to a large extent been responsible for subsequent investigations on the urethrovesical junction or, as it has been accepted in the past, the 'internal sphincter'. Ball in 1950[12] and Langreder [13,14] developed ingenious theories on sphincteric function at the urethrovesical junction. However, the anatomical studies of Kranz[15] and Woodburn,[16] together with the urethrovesical pressure studies of Enhorning[17] and Hodgkinson and Cobert[18] and the ciné studies of Lund *et al.*,[19] Gardner *et al.*[20] and Ardran *et al.*,[21] all indicate that no sphincter exists at the urethrovesical junction.

Lapides *et al.*,[22] as well as Kranz[15] and Woodburn,[16] emphasize that it is the normal tone of the urethra which primarily effects normal closure of the urethra. All these workers completely reject the idea of an internal sphincter. In addition Hodgkinson and Cobert[18] have shown that in a patient with normal detrusor control, intravesical pressure does not rise as the bladder fills and that the pressure influences recorded in the bladder and in the proximal two-thirds of the urethra are essentially the result of transmitted intra-abdominal pressure. Enhorning[23] demonstrated that leakage of urine incidental to stress does not occur unless the pressure in the bladder equals or exceeds that recorded in every portion of the urethra, and that urine will not leak, regardless of the stress, if the pressure at any

one point in the urethra exceeds that recorded in the bladder. The idea was thus supported that the entire urethra acted as a sphincter and that there was no circular sphincter at the site of the urethrovesical junction. Thus Lapides[24] actually regards the urethra as synonymous with the 'urinary sphincter'.

Lapides then introduced the Law of Laplace to explain the mechanism of urethrovesical control. According to the Law of Laplace, pressure (P) exerted on the wall of a tube by its fluid contents is directly proportional to the tension (T) in the wall and inversely proportional to the radius (r) of the tube, i.e.

$$P = \frac{T}{r}$$

As the tension (T) in the wall of a tube is a variable, and in direct proportion to the length of the tube, he postulated that, with an adequate urethral length, the pressure in the tube would be greater than the pressure in the bladder and in this way would act as a cork to the bladder. From this concept, then, he declared that the aetiology of stress incontinence was actual or functional shortening of the urethra. In 1960 he reported upon 22 patients with stress incontinence whose urethrae were measured directly with the calibrated Foley's catheter method, and from these observations he concluded that stress incontinence would arise in a urethra of less than 30 mm. in length. He further advised that cure of this condition depended upon the urethra being lengthened to a value greater than 30 mm.

Based on previous work by himself and others, Lapides in 1961[24] listed what were in his opinion 4 important milestones in the evolution of the modern concept of the physiology of micturition:

'(*a*) Bladder smooth muscle possesses inherent tonicity; (*b*) bladder smooth muscle is under direct cortical control; (*c*) bladder smooth muscle is activated solely through the parasympathetic nerves, and (*d*) the urinary sphincter is a tubular structure.'

Hodgkinson and his co-workers[25] tested Lapides's thesis and the corollary: (*i*) that abnormal shortening of the female urethra was the basic aetiology of stress incontinence, and (*ii*) that the correction of this shortness constituted the fundamental requirement for cure. By means of the radiographic metallic bead chain technique they measured the length of the urethra in a series of 164 patients, and the data consisted of 496 urethral measurements. They could demonstrate no correlation between incidence, severity or cure of urinary stress incontinence and a short urethra. They advanced the view that alteration of physical relationships between the urethra and the bladder changes the functional work load of the urethra. If, as a result of parturition or the attrition of ageing, the support of the bladder base weakens and allows the open-ended internal urinary meatus to be placed at the lowest level of the bladder during the straining effort, the bladder urine assumes the capacity of a dilating hydraulic wedge and continually tests the proximal urethra from within. Based on the findings of Kranz[15] and Woodburn[16]—who have shown that the detrusor extends uninterruptedly from the bladder to the external urethra—and on the inherent property of smooth muscle to adapt to different degrees of physical stress, they advised altering the position of the urethrovesical junction in such a way that the physical stress is less trying, in order to promote recovery of the tone of the smooth muscle of the urethra. For these reasons they conceived of the cause of urinary stress incontinence as the result of altered physiology rather than the result of the pathogenic effects of trauma.

Hodgkinson and his co-workers[26] studied voluntary voiding in 184 patients by simultaneously recording intravesical pressure, intra-urethral pressure, and the precise times of starting and stopping urine flow. They found that after voiding was discontinued the intravesical—intra-urethral pressure relationships were reversed and the intra-urethral pressure exceeded that in the bladder.

Cameron[27] speculated on the urethrovesical closing mechanism. He advanced the view that closing at the urethrovesical junction is achieved by folds of bladder mucosa at the urethral meatus supported by the underlying detrusor which is continuous with the urethral muscle. Should the normal closing mechanism fail, allowing urine to leak into the urethra, continence would depend on voluntary contraction of the external sphincter which would exert a pinchcock action on the urethra and so compress the urethral walls. The normal opening mechanism would be contraction of the detrusor which pulls the urethrovesical junction upwards, analogous to 'taking up' of the cervix. With this shortening, the urethra in effect becomes the bladder. With this action the mucosal folds are pulled apart, allowing urine into the urethra, and the posterior angle would disappear. Failure of the urethrovesical mechanism would force urine into the urethra and continence would only depend on voluntary control. Incompetence of the closing mechanism would be due to: (*i*) atrophy, oedema or inflammation of the mucosal folds, or (*ii*) abnormal enlargement of the urethrovesical 'ring'. This could result from menopausal atony or pregnancy.

Cameron explained that the success of all surgical procedures lay in the fact that the 'ring' around the urethrovesical junction was narrowed. Failures of these procedures were due to inadequate narrowing, atrophic mucosal flaps or inflamed flaps, and the presence of too much scar tissue.

It is quite clear that, to date, there is no single concept which adequately and satisfactorily explains urinary incontinence, although recent advances have led to a fuller understanding of the physiology of micturition. The Law of Laplace, in its pure context, cannot alone explain urinary continence. The very existence of voluntary striated muscle in the urogenital diaphragm, able to compress the urethral walls, is witness against it. It also requires the attachment of a hollow tube (urethra) to the bladder of adequate length and, if not of absolute, at least of relatively uniform lumen (2r). It is therefore essential, for the Law of Laplace to function at all, that the proximal attachment of this tube to the bladder (urethrovesical junction) be of constant diameter in the resting or inactive phase. This is dependent, if not on a muscular sphincter, on at least an inelastic collar or 'ring'. Serious objection to, if not total invalidation of, the application of the Law of Laplace in

the ensurance of urinary continence has been provided by clinical observation.

In Johannesburg recently a radical vulvectomy was performed for carcinoma of the vulva in which the whole length, excepting perhaps $\frac{1}{8}$ in., of the urethra was excised. Postoperatively this patient was totally continent of urine for approximately 4 hours at a time. Incontinence arose only with overfilling of the bladder. Also, many cases have been reported in which urethrae have been severely shortened but no type of urinary incontinence has developed. There appears to be general acceptance of the fact that there is no true internal sphincter in the muscular sense. It appears to be extremely difficult to adequately explain the non-existence of a urethrovesical closing mechanism. Hodgkinson and Drukker[20] acknowledge the weakening of the support of the bladder base as a result of parturition or atrophy, and presume funneling at the site of the urethrovesical junction by sagging of the internal urinary meatus to the lowest level of the bladder. This is synonymous with stretching of the collagenous collar around the urethrovesical junction. They advocate the establishment of salutary urethrovesical relationships as the common denominating factor in successful surgical repair such as retropubic urethropexy and vaginal plastic procedures. This, in turn, implies reconstruction of the 'collar'.

There is no longer any doubt that the establishment of the Marshall-Marchetti-Kranz procedure in 1949 was a milestone in the treatment of urinary stress incontinence. Judging by the spate of reports in the literature on this procedure, it appears to have received world-wide acceptance. The operation which Lapides himself devised to lengthen the urethra appears to have been inspired by the Marshall-Marchetti-Kranz procedure.[22] It would seem that the success of the Marshall-Marchetti procedure evolves not so much from the fixation of the urethra to the posterior symphysis, but from the plication effect of the two posterior sutures in the vicinity of the urethrovesical junction. Similarly, urethral sling procedures, such as the Aldridge sling operation, may be successful because of either their constrictive effect around the urethrovesical junction or a secondary fibrosis in this area which then provides adequate support and functional narrowing of the fascial collar.

In addition to an adequate 'ring' around the urethrovesical junction, other factors must be involved in closing the bladder at this site. It otherwise becomes extremely difficult to explain continence in cases of amputation of the urethra. The concept of mucosal flaps which close the proximal end of the urethra and the urethrovesical junction as described by Cameron, appears to be meritorious. A clinical observation which might be of significance is the fact that 5 cases came to notice where fulguration of the 'bladder neck' for chronic trigonitis resulted in intractable stress incontinence. It would appear that some of the 'fronds' seen in chronic infection of the bladder base may be mucosal flaps essential for the maintenance of normal bladder physiology.

Traditionally, labour and delivery have been implicated as major aetiological factors concerned in the development of stress incontinence. Delivery, and by implication intrapartum trauma to the musculature of the pelvic floor, appears to have been overemphasized as a causative factor; and by contrast, the importance of pregnancy itself has been either minimized or completely overlooked. There is no doubt that this condition is extremely common during pregnancy even in primiparae. It is important to note that during pregnancy conditions exist which produce true stress incontinence at a time preceding any trauma to the pelvic floor. The symptom also manifests itself, if not frequently then at least significantly, during the first trimester of pregnancy when pressure effects from the growing uterus on the bladder are either minimal or absent. Winifred Francis,[2] during a prospective study of 400 women during and after pregnancy, did not find a single case in which stress incontinence made its first appearance in the puerperium. It always began during, if not before, pregnancy.

In the series investigated in South Rand Hospital, the symptom first made its appearance during pregnancy in every case. It appears, from the histological evidence as described before, that stress incontinence during pregnancy results from urethrovesical deformity which is secondary to hormonal effect on the collagenous component of this organ. The fibrous-tissue stroma, poor in elastic fibres but rich in cells, which support the detrusor muscle fibre arrangement, may be of prime importance. Should this stroma become infirm, thick and amorphous, the whole basis for the adequate function of the supported muscle becomes infirm and deranged. Whereas normally the connective-tissue investing fascia (fibrosa) of the bladder is inelastic and does not stretch, thus maintaining urethrovesical morphology, during pregnancy the urethrovesical junction becomes deformed and stretched under the influence of pregnancy hormones. Urethrovesical competence is lost.

It is not impossible that hormonal influences adversely affect the voluntary sphincter. The fibres of the constrictor urethrae are imbedded in collagen tissue, especially the transverse fibres arising from the descending ramus of the pubis and the transverse ligament of the perineum. The transversus perinei profundus which arises from the junction of the rami of pubis and ischium in the form of a rounded tendon is made up of a large proportion of connective tissue. After delivery the vast majority of cases return to normal. Where this does not occur the reason may possibly be: 'Subinvolution' of the investing fascia of the bladder with subsequent sagging and distortion of the urethrovesical junction, or otherwise intrapartum trauma to the anterior suspensory ligament of the urethra with resultant sagging and tearing of the investing fascia of the urethrovesical junction and postpartum sagging and loss of posterior angulation at the site of the urethrovesical junction.

It is a speculation that hormonal influences during pregnancy may have a protective action on the lower urinary system by inducing flexibility in the rigid connective tissue and so preventing lacerations during labour. In this connection it is interesting to note that the relaxin content of the blood increases tenfold as pregnancy advances towards term. This may be related to the low incidence of stress incontinence in the first trimester of pregnancy as

observed in the previously described experimental series, namely 5% (or 3 cases out of a total of 59) as compared with 52·5% in the third trimester (31 out of 59 cases).

The above is confirmed by Francis and Jeffcoate[2] who admit pregnancy as an aetiological factor in contrast to the relative unimportance of childbirth.

It is difficult to escape the conclusion that in the matter of urinary continence there is 'double insurance':

(*a*) Involuntary control of continence is dependent on maintenance of normal morphology at the urethrovesical junction. With filling of the bladder to a critical volume leading to intravesical pressure in excess of intra-urethral pressure, continence is totally dependent on voluntary control.

(*b*) Voluntary control in turn is entirely dependent on the integrity of the striated muscle in the urogenital diaphragm.

Urinary stress incontinence arises if there is failure of one or both of these control mechanisms. Failure of the involuntary control mechanism would lead to the most distressing variety of urinary stress incontinence. The striated muscular sphincter is overtaxed, and with sudden sharp rises in intravesical pressure such as occurs with coughing or sneezing, the tired muscle gives way and leads to leakage of urine, regardless of intravesical urinary volume.

In those instances where there is only failure of the voluntary control mechanism the stress incontinence is not of such a serious nature. It occurs only in association with filling of the bladder to a critical volume. If at that stage there is a sudden sharp increase of intravesical pressure, leakage of urine cannot be voluntarily controlled and it continues until such time as the competence of the urethrovesical junction is regained.

In combined failure of these two mechanisms, stress incontinence is of such a serious nature as to approach total incontinence of urine.

Adequate cure is logically thus dependent on correct diagnosis of failure of the particular controlling mechanism (or both) involved in the causation of the condition.

There is confusion in the exact interpretation of operative results because some procedures are designed to reconstitute both the voluntary and the involuntary control mechanisms. In these cases the vaginal approach is utilized which can ensure absolute cure of voluntary control and only partial cure of involuntary failure by strengthening the urogenital diaphragm. The most common procedure of this nature is urethroplasty with plication of the urogenital diaphragm, or anterior repair with Kelly stitches. This type of operation, by ensuring excellent voluntary control, often masks failure of the involuntary mechanism, especially if the latter is not gross. This results in at least a 60 - 80% cure rate.[2] It is doubtful if the above figure can ever be improved upon by these procedures, because of the inherent difficulty in reconstituting adequate urethrovesical morphology where the latter is markedly distorted.

Operations which are primarily designed to reconstitute voluntary control only, such as the Martius sling operation, often fail because of failure in diagnosing a purely urethrovesical junction defect. Because of the modern stress on the importance of involuntary urinary control, too much importance is attached to the over-energetic reconstitution of the posterior angle at the urethrovesical junction, with resultant abuse of the so-called 'sling' procedure.

There is a traditional and incorrect assumption that stress incontinence is mostly associated with prolapse. The antenatal investigation of 300 pregnant females strikingly underlines the occurrence of this symptom in the absence of prolapse, and supports Frances and Jeffcoate in their statement that it is necessary to dissociate the two conditions. Unfortunately, prolapse is commonly associated with ascending urinary tract infection as a result of chronic cervicitis, or chronic urinary retention in a cystocele pouch with infection and resultant trigonitis. Frequency and urgency incontinence is thus often confused with true stress incontinence. Subsequent repair of the prolapse either by vaginal hysterectomy and repair or by the Fothergill operation leads to a cure of the bladder infection by reconstitution of adequate urinary drainage and removal of the septic focus. The postoperative disappearance of urinary symptoms is thus mistakenly regarded as cured urinary stress incontinence.

It seems logical to plan reparative procedures on correct diagnosis of the aetiological factor involved in the causation of the condition. A rational approach to the surgical correction of stress incontinence would thus be:

In the case of a failure of voluntary control a good urethroplastic procedure. Good results are obtained by any of the recognized procedures which strengthen the urogenital diaphragm, provided that dissection is not so wide as to cause a peri-urethral fibrosis to such an extent as would convert it into a rigid tube.

In the case of failure of involuntary control alone (which apparently is rarely associated with prolapse) it seems logical to restore normal bladder morphology. It is important to realize that the latter is independent of the *position* of the bladder relative to the pelvis or urethral orientation to the symphysis pubis. Although displacement of the urethrovesical junction is more often than not associated with tearing of the suspensory ligament and morphological distortion, it is not necessarily so.

Any operation which restores normal morphology seems to be successful; and this fact explains the success of a urethrocystopexy and also the urethral sling operations if performed with this fact in mind. Any sling procedure which disturbs normal physiological function by increased fibrosis in the peri-urethral tissues, which imprisons the urethrovesical junction and upper urethra in a rigid cage, or forms a ridge with exaggerated angulation posteriorly may lead to failure or may cause retention and chronic urinary obstruction. For this reason the described operation is based on the belief that normal morphology must be restored with as little interference as possible in order not to cause excessive fibrosis in the region of the urethrovesical junction.

SUMMARY

An operation evolved from the Marshall-Marchetti-Kranz procedure is described together with its rationale. The latter is based on histological and laboratory investigations which appear to indicate that the majority of cases of urinary stress incontinence without urogenital prolapse result from hormonal onslaught during pregnancy.

In addition to operative details, attention is paid to diagnosis, postoperative treatment and the complications of the procedure.

The over-all cure rate in the 35 cases followed up for more than 3 years is 80·7% and the corrected cure rate is 89·7%. The corrected cure rate for all 65 cases is 89·1%.

Recent research on the problems of urinary stress incontinence and bladder physiology is briefly reviewed. Evidence, both from the literature and from clinical practice, leads to the conclusion that in the ensurance of urinary continence two separate factors are involved, viz., involuntary and voluntary control mechanisms.

With regard to satisfactory involuntary control the following demands must be complied with: (*a*) for the Law of Laplace to function, a hollow elastic tube—and not a funnel—must be attached to a sphere; (*b*) should the adequate approximation of mucosal folds at the urethrovesical junction be of importance (as it well may be), a urethra of small enough diameter must be attached to the bladder. Both these demands are met by ensuring the integrity of the fibrosal 'collar' surrounding the urethrovesical junction.

Concerning satisfactory voluntary control—the lesser problem—this is dependent on adequate function of the striated muscle in the urogenital diaphragm.

An improvement in the over-all results following on surgical treatment can only be obtained with full awareness of the aetiology, and with selective and correct implementation of operative procedures.

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REFERENCES

1. Marshall, V. F., Marchetti, A. A. and Kranz, K. E. (1949): Surg. Gynec. Obstet., **88**, 509.
2. Francis, W. J. A. and Jeffcoate, T. N. A. (1963): *Modern Trends in Gynaecology*. London: Butterworths.
3. Bailey, K. V. (1954): J. Obstet. Gynaec. Brit. Emp., **61**, 291.
4. *Idem* (1956): *Ibid.*, **63**, 663.
5. Jeffcoate, T. N. A. and Roberts, H. (1952): *Ibid.*, **59**, 685.
6. *Idem* (1952): Amer. J. Obstet. Gynec., **64**, 721.
7. Kellar, R. J. (1956): Proc. Roy. Soc. Med., **49**, 657.
8. Malpas, P., Jeffcoate, T. N. A. and Lister, U. M. (1949): J. Obstet. Gynaec. Brit. Emp., **56**, 949.
9. Roberts, H. (1953): *Ibid.*, **60**, 621.
10. Yousseff, A. F. and Mahfouz, N. M. (1956): *Ibid.*, **63**, 19.
11. Marchetti, A. A. (1962): Personal communication.
12. Ball, T. L. (1950): Amer. J. Obstet. Gynec., **59**, 1243.
13. Langreder, W. (1956): Zbl. Gynäk., **78**, 56.
14. *Idem* (1961): *Gynakologische Urologie*. Stuttgart: Georg Thieme.
15. Kranz, K. E. (1951): Amer. J. Obstet. Gynec., **62**, 374.
16. Woodburn, C. R. T. (1960): J. Urol. (Baltimore), **84**, 79.
17. Enhorning, G. (1960): Proc. Roy. Soc. Med., **53**, 1019.
18. Hodgkinson, C. P. and Cobert, N. (1960): Amer. J. Obstet. Gynec., **79**, 648.
19. Lund, C. L., Benjamin, J. A., Tristan, T. A., Fullerton, R. E., Ramsay, T. H. and Watson, J. J. (1957): *Ibid.*, **74**, 896.
20. Gardner, S. H., Campbell, J. H., Garrett, R. A. and Schell, H. R. (1961): *Ibid.*, **82**, 1112.
21. Ardran, G. M., Simmons, C. A. and Stewart, J. H. (1956): J. Obstet. Gynaec. Brit. Emp., **63**, 26.
22. Lapides, J., Mitsuya, H., Asai, J., Suyama, K., Ushida, T. and Hosoe, K. (1960): J. Urol. (Baltimore), **83**, 86.
23. Enhorning, G. (1961): Acta chir. scand., **276**, 1.
24. Lapides, J. (1961): Brit. J. Urol., **33**, 284.
25. Hodgkinson, C. P., Drukker, D. H. and Hershey, G. J. (1963): Amer. J. Obstet. Gynec., **86**, 16.
26. *Idem* (1966): *Ibid.*, **94**, 791.
27. Cameron, M. D. (1964): Brit. J. Urol., **36**, 526.
28. Hodgkinson, C. P. and Drukker, D. H. (1966): Amer. J. Obstet. Gynec., **94**, 791.