Treatment of Stress Incontinence by Maximum Perineal Electrical Stimulation

THOMAS MOORE,* M.D., M.S., F.R.C.S. ; PHILIP F. SCHOFIELD,† M.D., F.R.C.S.

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Stress incontinence is one of the commonest symptoms to occur in the female. It has been estimated that more than 50% of nulliparous women have incontinence when severe stress is placed on the bladder (Nemir and Middleton, 1954). However, it is only when incontinence occurs on minor stress such as coughing or walking that the patient presents for advice.

Jeffcoate (1961) suggests that the main factor is the loss of the normal urethrovesical angle. Lapidès *et al.* (1960a, 1960b) felt that the principal defect was shortening of the urethra due to absence of the lengthening effect of the pelvic floor muscles. These views are probably not divergent, as descent of the bladder base can produce both the alteration in the angle and urethral shortening. Vincent (1959, 1966) has shown how mechanical raising of the perineum by an external appliance can tighten the pelvic floor muscles and the urethra and abolish incontinence.

On theoretical grounds incontinence occurs from either the uncontrolled action of the bladder muscle or some defect in the normal resistance of the bladder-neck and urethra. Beck, Hsu, and Maughan (1965) showed low urethral pressures due to general laxity of the urethral musculature in stress incontinence.

Perineal muscle exercises and faradism are known to bring improvement in this type of case (Jones, 1963). Some years ago one of us (T. M.) carried out studies of the electrical activity of the urethral muscles in all types of incontinent females by placing needle electrodes along the urethral wall. In one patient, in whom the anaesthesia was not deep enough, reflex contraction of the whole pelvic floor muscles followed introduction of the electrodes. Afterwards the patient's incontinence was mysteriously much improved.

Because of this observation it was decided to treat incontinent females empirically by causing maximal contractions of the pelvic floor muscles by electrical stimulation, under anaesthesia.

Method

Ten patients, who had only cystoscopy and measurement of urethral length, were used as a control group. Eighteen patients with marked stress incontinence had cystoscopy, measurement of urethral length, and maximal perineal faradism (see Table). Ten patients with other types of incontinence, four cases of enuresis, three cases of urge incontinence, and three cases with gross structural changes in the urethra had the same examination and treatment.

The faradism was given by electrodes—a mobile one placed over the perineal body and a stationary one under the sacrum—by a selective treatment unit (Stanley Cox Ltd.). The faradic surge consisted of pulses of one millisecond and the whole surge lasted for two to three seconds. There was then a pause of two to three seconds before a further faradic surge. The intensity of the stimulation was increased until all the voluntary musculature gave a maximal contraction. From four to six maximal tetanic contractions were produced in each patient.

* Consultant Surgeon, Department of Urology, Manchester Royal Infirmary.

† Senior Surgical Registrar, Department of Urology, Manchester Royal Infirmary. Present address: Cleveland Clinic, Ohio, U.S.A.

Classification of the results is difficult, but these have all been assessed after treatment by one of us (P. F. S.) and the patients who have no further stress incontinence in the first three months after treatment are placed in the "cured" category. It was accepted that patients may be classified as improved if they stated that the treatment was worth while, that the amount of protective pads worn per day was fewer than three, and if the assessor was convinced from the history and examination of the urethra when the patient was straining that there was improvement. All other patients were classified in the failure group.

Results

No other type of incontinence showed any improvement. One of the group of 10 patients who had only cystoscopy showed improvement but relapsed at four months. Ten out of 18 patients with stress incontinence were either considerably or completely relieved of their symptoms (see Table).

Investigation and Results of Treatment of Stress Incontinence

Age	Cystoscopy	Urethral Length in cm.	Neurological Signs	Result
41	Cystitis cystica	2.1	—	Failure
46	None	2.7	—	Cured
46	"	2.0	—	Improved
50	"	1.8	—	Failure
31	"	2.3	—	"
43	Trabeculation	2.2	—	" Cured
55	None	—	—	Failure
52	Trabeculation	2.3	—	Improved
38	None	2.0	—	Cured
27	"	3.5	—	"
32	"	1.9	—	"
21	"	2.5	—	Failure
26	"	2.4	—	"
38	"	1.9	—	Improved
37	"	1.6	—	Failure
26	"	—	—	Improved
47	"	2.5	—	Failure
44	"	2.1	—	Cured

Four patients relapsed partially by six months, but two of them responded to further faradism under general anaesthesia and the other two did not feel their symptoms were severe enough to require treatment. Six patients have remained much relieved for one year.

Six of the eight patients who failed to respond were treated by Lapidès's (1961) operation; five of these were relieved of all symptoms, but one still had stress incontinence.

Discussion

Urinary control is a learned function, and, as with any learned lesson, there are various degrees of success. Urinary control in the normal female is often not perfect; in fact, more than 50% of normal nulliparous females have slight incontinence on occasions of severe and sudden stress (Nemir and Middleton, 1954). More gross degrees of loss of control are only too distressingly common.

In the past, treatment has taken two main lines: (1) operation to alter the orientation of the bladder-neck and urethra or to

elongate the urethra; and (2) physical treatment by exercises and electrical stimulation to improve the perineal musculature.

Electrical stimulation to the perineum has been carried out for many years in the treatment of minor stress incontinence, and electrical devices have recently been used to treat incontinence of both urine and faeces (Caldwell, 1963; Hopkinson and Lightwood, 1966; Caldwell, Flack, and Broad, 1965). Caldwell *et al.* (1965) delivered a square wave pulse to the urinary sphincter in a patient with urinary incontinence after spinal injury, with gratifying results. It is interesting to note that some patients recover sufficient sphincter power to discard the electrical apparatus in time.

The cause of stress incontinence is an imbalance between the pressure in the bladder and the resistance in the urethra. Normally all conditions of stress causing an increase of intra-vesical pressure are accompanied by a corresponding increase in the urethral resistance. The mechanism of this reaction is that increase of abdominal muscle tone is always accompanied by an increase in the tone of the pelvic floor muscles and a consequent elongation of the urethra by the contraction of the pubococcygeus muscle. Lapidès *et al.* (1960a, 1960b) and Lapidès (1961) stated that in the female a urethral length of 3 cm. was necessary for continence. Personal observations have confirmed that in most cases of stress incontinence the urethra is less than 3 cm. long and have also shown that in cases of stress incontinence standing causes a shortening of the urethra. This is due to loss of tone of the pelvic floor muscles, which do not contract in response to the increased intra-abdominal pressure and elongate the urethra. In normal females without stress incontinence standing causes an increase in the length of the urethra by 1 cm. or more. We have also shown that during maximal stimulation under general anaesthesia the urethra lengthens by 1 cm. or more, to return to its original length when the electrical stimulation is stopped.

We presume that when the anaesthetic has worn off the tone of the perineal floor muscles returns and is greater than before treatment. The change in the length of the urethra and possibly some narrowing of the urethral lumen associated with the return of tone of the perineal muscles increases the urethral resistance, in accordance with Laplace's law.

In successful cases we have not remeasured the urethra to confirm its elongation because of a fear of causing further symptoms. The success of the treatment we presume is because maximum perineal contraction of the perineal muscles causes a maximum physiological rehabilitative response and is better than long-continued intermittent faradism or voluntary exercises. The improvement that follows the stress incontinence occurring directly after childbirth by pelvic floor muscle exercises is well known. In our view it is likely that such patients would recover more quickly if treated with maximal perineal electrical stimulation.

It will be noted from the Table that improvement in stress incontinence occurred in some cases in which the urethra was much shorter than 3 cm., though it was more likely to occur in those cases in which the urethra was more than 2 cm. long. The fact that such results can be obtained with very short urethrae shows how effective the method can be.

The encouraging results in which 6 (33%) out of 18 patients with stress incontinence have been completely relieved of symptoms and a further 4 (22%) considerably improved merit further studies. We are so convinced of the efficiency of the method and by its safety and simplicity that we now tend to try it in any case of urinary incontinence. In some cases of neurogenic bladder it might well have a most beneficial effect in restoring better power to muscles not completely paralysed by the nervous disease. It should, however, be tried in all cases of true stress incontinence before operation is advised, particularly in those patients with a short history and a urethra shorter than normal in the standing position only by 1 cm. or less.

Summary

Stress incontinence of urine in the female is usually due to loss of tone of the pelvic floor muscles with consequent shortening of the urethra. In the erect posture the urethra must be at least 3 cm. long.

Cases were treated by causing maximal contraction of these muscles by electrical stimulation under general anaesthesia. Of 18 cases so treated 33% were completely relieved and 22% much improved.

During contraction of the pelvic floor muscles the urethra lengthens by 1 cm. or more, to return to its previous length when the contraction ceases.

It is suggested that in the successful cases the maximal perineal muscle electrical stimulation has restored the postural tone of the pelvic floor, so that all increases of intra-abdominal pressure are again accompanied by the normal pelvic floor contraction and lengthening of the urethra. Possibly one maximum contraction of a muscle restores power better than repeated submaximal contractions, the more usual method.

This treatment should always be tried before operative measures are advised, particularly when investigation has shown that in the erect posture the urethra is only 1 cm. or less short of the critical length (3 cm.).

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