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# Biofeedback in Treatment of Urinary Incontinence in Stroke Patients[1]

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*Urinary incontinence can occur poststroke owing to weakness or incoordination of sphincter muscles, impaired bladder sensation, or hyperreflexic, neurogenic bladder. Four male subjects who had urinary incontinence associated with a stroke that had occurred 8 months to 10 years earlier, and who averaged 1.6 to 7.5 accidental voidings per week, participated in an outpatient study with a 4-week scheduled-voiding baseline, 2 to 5 sessions of biofeedback-assisted bladder retraining, and 6- to 12-month follow-up. Training sessions included stepwise filling of the bladder and manometric feedback display of bladder pressure, abdominal pressure, and external anal sphincter pressure. Training procedures were designed to teach subjects to attend to bladder sensations, inhibit bladder contractions, and improve voluntary sphincter muscle control. All four subjects achieved and maintained continence regardless of substantial differences in subject characteristics,*

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*including laterality of stroke, degree of sensory impairment, and independence in daily activities.*

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Incontinence is a common problem in patients referred to rehabilitation programs, and it is frequently seen in patients with sensory or motor deficits due to neurological diagnoses such as stroke, partial spinal cord injury, and multiple sclerosis. The standard approach to treating these problems is through bowel and bladder training programs that emphasize scheduled voiding and habit training in combination with dietary control, bulking agents, and medication. Patients are taught to make use of voiding reflexes to establish regular voiding habits and thereby minimize incontinence.

Bowel and bladder training programs seldom directly address such problems as sphincter muscle weakness, reduced awareness of bladder or rectal sensations, or poor voluntary inhibition of voiding reflexes. Yet these factors cause, or contribute to, incontinence in many patients. One reason for the current lack of emphasis on sensorimotor retraining for improved voluntary control is that existing bowel and bladder training methods are effective for many patients and indeed are the only feasible method for some categories of patients, such as those with complete spinal cord injury, who do not have the neurological capacity for regaining voluntary control. A second reason is that workable methods have not been available for directly evaluating and retraining these sensorimotor deficits.

Biofeedback procedures, which provide audio or visual feedback to guide sensorimotor retraining, have been highly successful in treatment of fecal incontinence (Engel, Nikoomanesh, & Schuster, 1974; Whitehead, Burgio, & Engel, 1985; Whitehead et al., 1986; Whitehead & Schuster, 1987a, 1987b) and urinary incontinence (Burgio & Burgio, 1986; Burgio, Robinson, & Engel, 1986; Burgio, Whitehead, & Engel, 1985; Middaugh, Whitehead, Burgio, & Engel, 1985a, 1985b). These procedures provide a potentially valuable methodology for treatment of incontinence in selected rehabilitation patients who (1) have the cognitive capacity to follow instructions during treatment sessions and retain learning from session to session and (2) have the neurological capacity for reacquiring voluntary control.

At present, there is relatively little information available on the suitability or effectiveness of biofeedback procedures for treatment of urinary or fecal incontinence in patients with neurological diagnoses commonly seen in rehabilitation settings. A few neurological patients with fecal incontinence have been icluded in clinical trials with some reported success. Cerulli, Nikoomanesh, and Schuster (1979) for example, included 3 stroke patients in their series of 50 consecutive ambulatory patients with fecal incontinence

who were treated with manometric biofeedback procedures. All 3 stroke patients were reported as showing a good response to biofeedback training. Such reports, however, do not provide the kind of information—on either the medical history or the neurological characteristics of the individual patients—that is needed to develop optimal biofeedback procedures for patients with major neurological diagnoses or to develop guidelines for selection of appropriate biofeedback candidates.

One recent study by Whitehead et al. (1986) does provide information relevant to rehabilitation. In this study, children with spinal cord injury due to myelomeningocele, who had fecal incontinence that had not responded to previous bowel training programs, were given controlled clinical trials compairing two behavioral procedures, habit training alone versus habit training plus manometric biofeedback. Data were provided on the neurological status of all patients, including level of lesion, rectal sensation, and capacity for voluntary contraction of the external anal sphincter. Habit training, which included behavioral methods for obtaining the cooperation of the child, reducing constipation, and establishing regular voiding schedules, made a major contribution to clinical gains. Habit training alone could account for the improvements made by those children who had higher cord lesions (above L2) and relatively low accident rates prior to treatment. These were children who tended to have poor rectal sensation, little capacity for voluntary sphincter contractions, and high resting sphincter tone due to spasticity, and who were relatively sedentary. Biofeedback procedures made a significant, positive contribution to treatment outcome in a subset of children (27% of participants) who had lower, less complete cord lesions and high pretreatment accident rates. These were children who tended to have relatively good sensation, greater preserved capacity for voluntary sphincter contractions, and little spasticity (which can augment sphincter tone), and who were physically very active. Such children have typically not responded well to standard methods of bowel and bladder training (Dietrich & Okamato, 1982).

These studies indicate that biofeedback procedures are potentially important adjuncts to bowel and bladder training programs for selected rehabilitation patients. However, the neurological characteristics of the individual patients are very relevant and need to be investigated as part of biofeedback trials. The following study of urinary incontinence in stroke patients was carried out as part of an ongoing research program on biofeedback-assisted procedures for treatment of urinary and fecal incontinence in patients with major neurological diagnoses treated in rehabilitation settings.

Urinary incontinence can be a consequence of stroke because the striated sphincter muscles may be weak or uncoordinated, the bladder may be hyperreflexic (spastic), and bladder sensations constituting the early warning system may be reduced or misinterpreted. The biofeedback procedures

in this study were designed to remedy these three problems in an attempt to reduce urinary incontinence poststroke. For many stroke patients, this problem clears up quickly and spontaneously; for others, there is a continuing problem, which may require the regular use of a catheter or may involve only occasional loss of control. However, incontinence even once or twice a month can be highly distressing to the patient and socially limiting.

While urinary incontinence following stroke is a well-recognized problem, it has been difficult to determine the scope of this problem. Perhaps the best data on prevalence to date come from a survey by physicians in Manchester, England, of all stroke patients seen over a period of 6 months who had hemiparesis lasting longer than 24 hours and who survived 2 weeks or longer (Brocklehurst, Andrews, Richards, & Laycock, 1985). Of a total of 135 patients in this survey, 51% experienced fecal or urinary incontinence associated with their stroke. For most stroke patients, the incontinence apeared, and also cleared, within the first 2 weeks to 2 months poststroke. From 2 months to 6 months poststroke, the rate of spontaneous resolution slowed considerably, and no one who was incontinent at 6 months was found to be continent at 12-month follow-up. At 1 year poststroke, 15% of survivors had unresolved urinary incontinence and 8% had unresolved fecal incontinence, indicating that urinary incontinence was more prevalent and more persistent than fecal incontinence. Incontinence was related to severity of stroke, but not to laterality of brain damage.

The above data indicate that the prevalence of persistent incontinence poststroke is relatively low when considering all stroke survivors, including many with mild strokes and relatively complete recovery. However, because of the large number of strokes annually, stroke patients constitute a relatively high proportion of the incontinent elderly and a high proportion of those with urinary incontinence in nursing homes (Brocklehurst, 1984; Brocklehurst & Dillane, 1966). An excellent, recent survey by Ouslander, Uman, Urman, and Rubenstein (1987), for example, found that stroke patients accounted for 53% of those with urinary incontinence in one Veterans Administration nursing home. Incontinence is also a common problem among stroke patients seen in rehabilitation settings, most of whom have had relatively severe strokes and incomplete recovery.

One can conclude, therefore, that new treatment methodologies that are effective in treatment of incontinence—expecially urinary incontinence—poststroke would constitute a significant clinical advance in treatment of incontinence.

## METHODS

### *Subjects*

Subjects in the present study were four men, all living in the community, with a clear history of persistent urinary incontinence associated with a

**Table I.** Subject Characteristics

| | Subject | | | |
|---|---|---|---|---|
| | 1 | 2 | 3 | 4 |
| Age (years) | 69 | 65 | 61 | 66 |
| Duration of incontinence | 2 years | 10 years | 8 months | 4 years |
| Laterality of brain damage | Right | Right | Left | Right |
| Stroke stage | III | III | II/III | III |
| Spasticity | Mild | Marked | Moderate | Moderate |
| Sensory status | | | | |
| Arm and leg | Not tested | Impaired | Normal | Normal |
| Bladder | Impaired | Impaired | Normal | Normal |
| Ambulation | Walker<br>Wheelchair | Cane | Quad cane<br>Wheelchair | Short leg brace |
| Self-care | Dependent | Independent | Assistance | Independent |
| Mini-Mental State (30 possible) | 19<br>Illiterate<br>Mild cognitive impairment | 29 | 23<br>Mild expressive aphasia | 27 |
| CESD score (60 Possible) | 12 | 18 | Not tested | 0 |

stroke occurring 6 months or more prior to the study. All four were given a urological examination and a cystometrogram by a urologist at the time of admission to the study and were diagnosed as having hyperreflexic neurogenic bladders (detrusor muscle instability) (Bates et al., 1979; ICS Committee on Standardization of Terminology, 1984). Other causative factors, such as prostatic hypertrophy and bladder infection, were ruled out. None was using an indwelling catheter, but two were using an external collection device (condom catheter) when outside the home (subject 1 and subject 3). All four had the ability to comprehend and follow simple verbal instructions, as determined by administration of the Mini-Mental State Exam (Folstein, Folstein, & McHugh, 1975) and by the subject's ability to follow directions during the following additional test procedures. The CES-D scale for depression was administered (Comstock & Helsing, 1976), and subject 2 was categorized as mildly depressed (CESD = 18: a score of 16 or higher on a 60-point scale is indicative of depression). Sensation (light touch and pressure) and joint proprioception were tested on the hemiparetic side, and the Brunnstrom (1970) stage of recovery poststroke was determined. These test results and other relevant subject characteristics are given in Table I.

A preliminary report of this study has appeared in abstract form (Middaugh et al., 1985a, 1985b). These subjects were recruited and treated through the Geriatric Incontinence Clinic, Gerontology Research Center, National Institute of Aging, Baltimore, and were included as a special-diag-

nosis subset of a large series of patients treated in this NIA clinic (Burgio et al., 1985).

## *Procedure*

In an initial visit, subjects were interviewed, given the tests described above, instructed on a home program of scheduled daytime voiding every 2 hours, and asked to keep daily records of voiding and accidents for a 4-week baseline period. Biofeedback training sessions were then implemented once every 2 to 4 weeks over a period of 3 months, and follow-up sessions were scheduled for 6 months and 12 months posttraining.

The recording and feedback system used in each biofeedback training session is illustrated in Figure 1 (from Whitehead, Burgio, & Engel, 1984). A catheter was placed in the bladder and connected through a pressure transducer to a Beckman dynagraph. Bladder contractions increased bladder pressure and were seen as an upward pen deflection (Figure 1, top tracing). In addition, a rectal tube was inserted with three balloons, two of them also connected to pressure transducers. One balloon measured pressure at the external anal sphincter (EAS). Voluntary sphincter contractions increased pressure and produced an upward pen deflection, while sphincter relaxation resulted in a downward pen deflection (Figure 1, middle tracing). The second balloon, situated at the internal anal sphincter, served to hold the tube in place. The third balloon measured rectal (and abdominal) pressure. Tensing of the abdominal muscles produced a rise in abdominal/rectal pressure and an upward pen deflection (Figure 1, bottom tracing). This

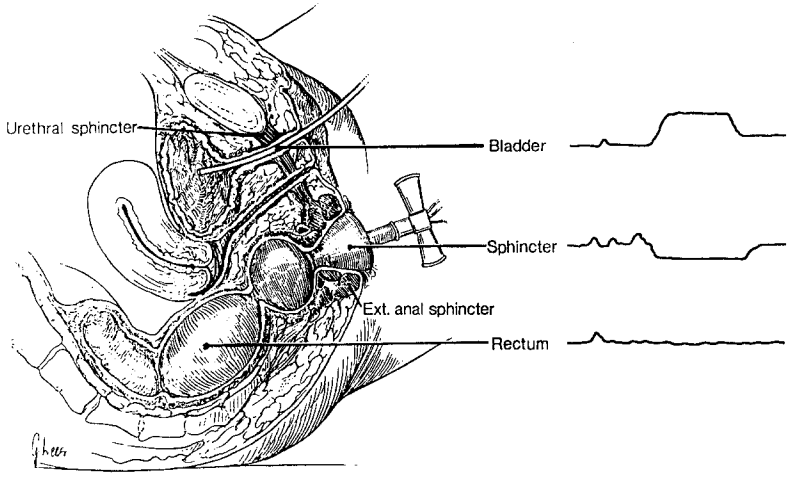


**Fig. 1.** Diagram of pelvic anatomy showing the devices used to provide biofeedback information to the patient. Sample polygraph tracings are shown at right.

three-balloon recording system has been described in detail elsewhere (Whitehead & Schuster, 1983). Manometric feedback was provided visually by having the subject (side-lying) observe a continuous display of these three channels on the chart writer during biofeedback training sessions.

Each session consisted of three trials in which the bladder was filled by injecting 20 ml of sterile water through the catheter at 1-minute intervals. During filling, subjects were instructed to attend to bladder sensations, visually monitor bladder pressure, keep their abdominal muscles relaxed, and concentrate on avoiding (inhibiting) bladder contractions. When a bladder contraction occurred—seen as a steep rise in bladder pressure on the feedback display—subjects were asked to voluntarily contract the external anal and urinary sphincters to (a) reflexly inhibit the bladder contraction and (b) interrupt the stream. At the same time, subjects were to avoid generalized muscular bracing and breath holding to avoid counterproductive increases in abdominal pressure (monitored via the rectal balloon).

Normal individuals can actively inhibit, and thereby prevent, reflex bladder contractions during filling and can voluntarily contract the external urinary sphincter to interrupt the urinary stream when voiding. In addition, voluntary contractions of the pelvic floor muscles (which include the external anal and external urinary sphincters) can reflexively inhibit bladder contraction through a pudendal-to-pelvic nerve reflex. In contrast, maneuvers that increase abdominal pressure, such as breath holding and tensing of abdominal muscles, can reflexly augment and trigger bladder contractions (Bagley & O'Shaughnessy, 1985; Teague & Merrill, 1977; Whitehead & Schuster, 1987b). Normal individuals can voluntarily initiate bladder contractions without such somatomotor activity (Lapides, Sweet, & Lewis, 1957).

Each session also included 10 to 20 sphincter-training trials to increase strength and control of these important muscles. Subjects were provided with manometric feedback from the two channels indicating external anal sphincter contractions and rectal (abdominal) pressure. On each trial the subject was asked to voluntarily contract the external anal and external urinary sphincters and hold the contraction for 10 seconds while keeping the abdominal muscles as relaxed as possible. These sphincters are both innervated by the pudendal nerve and commonly contract together. Since the external anal sphincter is by far the more accessible, it has been used, quite effectively, for biofeedback training to improve urinary sphincter control (Burgio et al., 1985, 1986; Maizels & Firlit, 1979; Maizels, King, & Firlit, 1979; Perkash, 1980; Sugar, 1983).

Between sessions, subjects were asked to carry out a daily home program. They were asked to do the following: (1) Continue with scheduled voiding and record keeping as in baseline; (2) practice sphincter exercises, typically 10 to 25 contractions several times a day for a daily total of 50 contractions; (3) attend to bladder sensations and start for the toilet early, rath-

er than waiting for a strong urge to void; (4) use several voluntary sphincter contractions to inhibit the bladder prior to standing and stay as relaxed as possible on the way to the commode; and (5) practice sphincter control by interrupting the stream during voiding. Subjects were asked to continue keeping daily records throughout training.

At 6 months and 12 months after biofeedback training, subjects were contacted and asked to keep daily records for 1 month and return for a test session. At 6-month follow-up, data were obtained by telephone interview and home records for subjects 1 and 3. Subjects 2 and 4 also returned for a test session. At 12-month follow-up, subject 1 refused to provide data (the daily records and information for this subject were provided by the wife, who was in poor health at the time of follow-up and did not want to participate), and subject 4 was deceased. Subjects 2 and 3 kept records and returned for a test session.

## RESULTS

As shown in Table II, all four subjects showed substantial, clinically relevant improvements during training, and all four became continent (no accidental voidings in the previous month) either by the end of training (subjects 3 and 4) or by 6-month follow-up (subjects 1 and 2). These gains were well-maintained in the two subjects who were available for 12-month follow-up.

Although this sample of four subjects was small, it was heterogeneous with respect to patient characteristics reported in Table I. These data provide some initial indication of factors that may be important in urinary incontinence poststroke and response to biofeedback training. All four patients benefited from biofeedback training in spite of substantial differences in laterality of brain damage, sensory status (of arm, leg, and bladder), and functional status (independence in ambulation and self-care). Interestingly, sensory status of the arm and leg on the paretic side was found to be consistent with sensory status (impaired vs. unimpaired) of the bladder. Rectal sensation was not tested. Neither mild cognitive impairment (subject 1) nor mild depression (subject 2) was an impediment to improvement.

The use of diuretics by three of the four subjects did not prove to be a problem. Subject 2 did show exceptionally high rates of urine formation in several biofeedback sessions, and this information was used to emphasize to the patient the importance of regular voiding, regular fluid intake, and limited use of substances such as caffeine and beer.

Fecal incontinence, although not the primary focus of this study or the biofeedback training procedures, was reported by three of four subjects during the initial interview and verified on daily home records either during

**Table II.** Results of Biofeedback Training

| | Subject | | | |
|---|---|---|---|---|
| | 1 | 2 | 3 | 4 |
| *N* biofeedback training sessions | 3 | 5 | 4 | 2 |
| Diuretics | + | + | + | – |
| Urinary incontinence rate and % improvemente | | | | |
| Baseline | 1.9/week | 7.5/week | 5.1/week | 1.6/week |
| Posttraining | 0.1/week 95% | 2.8/week 63% | 0/week 100% | 0/week 100% |
| 6-month follow-up | 0/month 100% | 0/month 100% | 0/month 100% | 0/month 100% |
| 12-month follow-up | Refused | 0/month | 0/month | Deceased |
| Fecal incontinence | | | | |
| Pretreatment | + | + | – | + |
| Follow-up | – | – | – | – |

baseline or during the course of treatment. Since EAS exercises were part of the study protocol, subjects were encouraged to use these exercises to improve strength and control of the anal sphincter, attend more closely to early warning bowel sensations, and use voluntary EAS contractions to maintain control when necessary. Frequency of fecal incontinence was reported to be "occasional" for subjects 1 and 2, occurring approximately once a month, usually in association with diet-related diarrhea or gas. Subject 4 reported more frequent problems, "once or twice a week" and recorded one fecal accident per week during baseline. All subjects reported improvements over the course of treatment and had no fecal incontinence in the month preceding their last follow-up.

Table III provides data on bladder characteristics, for the first and last training session, for each of the four subjects. These data indicate that uninhibited bladder contractions at a relatively low bladder capacity, less than 350 ml, was a major factor that contributed to urinary incontinence in these subjects. All four subjects showed uninhibited bladder contractions at relatively low levels of bladder filling during biofeedback training trials. This finding was consistent with patient reports and with the urologist's diagnosis. This bladder hyperreflexia was aggravated by a number of factors that increased spasticity in general on the hemiparetic side, such as physical exertion and cold. For example, subjects 2 and 4 reported particularly severe problems with driving home in cold weather: The exertion of getting out of the car and walking, and the sudden exposure to the cold aggravated their bladder problems. The additive effect of such factors was clearly demonstrated by subject 2. This patient had marked spasticity of the left arm and leg and reported that most accidents occurred while on his feet, either while getting to a standing position, walking to the bathroom, or standing at the commode adjusting his clothes. This individual was independent in these functions and did not take an unusual amount of time to accomplish these activities. However, he showed little evidence of uninhibited bladder contractions during the initial cystometrogram or during training trials in the first two biofeedback sessions in which the bladder was filled up to 600 ml. These procedures were carried out in a side-lying position, and all muscles were relatively well relaxed under those conditions. In session 3, a bladder-filling trial was carried out with the subject standing. In the standing posture, the picture was very different; spasticity was markedly increased in the arm and leg, and an uninhibited bladder contraction occurred at 300 ml. This finding was consistent with the subject's verbal reports.

As indicated in Table III, impaired bladder sensation was a second factor that contributed to urinary incontinence. This was a serious problem for two subjects. During biofeedback sessions, subject 1 invariably reported his first sensation as an urge to void just 15 to 30 seconds prior to involuntary voiding, and this combined event occurred at a low level of bladder fill-

**Table III.** Bladder Characteristics

| | Subject | | | |
|---|---|---|---|---|
| | 1 | 2 | 3 | 4 |
| First urge to void | | | | |
| Pretreatment | 130–150 ml | 240 - > 350 ml | 100 ml | 100 ml |
| Posttreatment | > 230 ml | 320 - > 350 ml | 100 ml | 100 ml |
| Bladder capacity | | | | |
| Pretreatment | 130–150 ml | > 350 ml | 200–250 ml | 320–340 ml |
| Posttreatment | 230 ml | > 350 ml | 180–240 ml | 200 ml |
| Ability to interrupt urinary stream | | | | |
| Pretreatment | – | – | – | – |
| Posttreatment | – | + | + | + |

ing, in the 130- to 250-ml range. This subject, and his caretaker, reported that he had very little warning time prior to voiding and sometimes voided without realizing that he had done so. Subject 2 also had reduced sensation and typically reported his first urge to void at 240 ml. Since his bladder capacity was high (600 ml or more, as noted above), he had ample warning time while sitting or lying down. However, this sensory threshold was quite close to the bladder contraction threshold (300 ml) that was found for this individual in the standing position. Two subjects, 3 and 4, were able to detect bladder filling at normal levels; however, their bladder capacity was also low, and so warning times were relatively short.

The data in Table III also indicate that poor urinary sphincter control was a third factor that contributed to urinary incontinence. At evaluation, none of the four subjects was able to use the pelvic floor muscles effectively to interrupt the urinary stream while voiding. Judging by anal sphincter responses to digital examination and biofeedback training trials, absolute sphincter strength was less of a problem than poor voluntary control. The external anal sphincter muscles of all four subjects were capable of a relatively strong voluntary contraction (10–20 mm Hg). However, control of this pelvic floor muscle was initially poor. The EAS was slow to develop a maximum contraction (slow recruitment is a characteristic of paretic muscles that has been noted before; Hammond, Kraft, & Fitts, 1988; Middaugh & Miller, 1980; Middaugh, Miller, Foster, & Ferdon, 1982). There was also a pronounced "warm-up" effect; i.e., EAS contractions improved substantially over the course of repeated 10-second trials. Subjects usually needed three to five trials to "get the muscle going," and then could produce relatively strong, well-sustained voluntary contractions. Digital examination showed good internal anal sphincter tone for all four subjects, which is consistent with spastic hemiparesis and heightened reflex activity. Across train-

ing sessions, all four subjects showed improvements in EAS control. Three subjects ultimately reported being able to interrupt the urinary stream during home practice and also reported good success with the strategy of using voluntary sphincter contractions to postpone voiding. The fourth, subject 1, did not demonstrate an ability to stop the urinary stream in sessions. This subject was not a good reporter and did not provide information on strategies employed to achieve continence at home.

## DISCUSSION

The results of this study indicate that three major factors contributed to urinary incontinence in these subjects: (1) uninhibited bladder contractions at a relatively low bladder capacity, less than 350 ml, (2) impaired bladder sensation, and (3) poor sphincter control. The biofeedback-assisted training program was directed toward countering these three problems. All subjects showed improvements in urinary incontinence during such training, and all ultimately achieved urinary continence.

An interesting finding was while the subjects improved in bladder control, there were no measurable changes in bladder sensory threshold or bladder capacity as a result of training, indicating that the physiological characteristics of the bladder did not change (Table III). Subjects did not learn to alter the sensory threshold of the bladder or to inhibit bladder contractions encountered at the upper limit of bladder capacity under standard conditions—while resting quietly. Instead, the findings in this study, together with experimenter observations and patient reports, indicate that subjects improved by learning to manage their low-capacity, hyperreflexic bladder more effectively in the following ways:

1. By attending more closely to available bladder sensations and possibly interpreting sensations more appropriately.
2. By using early bladder sensations as a cue to void (rather than waiting for a strong urge to void) and thereby allowing more time to get to the toilet; and by using regularly scheduled voiding (independent of urge to void) to keep bladder filling below contraction thresholds, particularly when this contraction threshold is lowered by standing and walking.
3. By using several, repeated 10-second voluntary sphincter contractions to gain control prior to standing up and starting for the toilet in order to (a) "warm up" the sphincter muscles so they can be used quickly and effectively if needed and to (b) use voluntary sphincter muscle contractions to temporarily inhibit voiding reflexes.

4. By staying relaxed on the way to the toilet in order to minimize spasticity in the arm, leg, and bladder that is aggravated by physical exertion and anxiety.

Manometric biofeedback training, therefore, offers a promising new methodology for treatment or urinary incontinence poststroke. All four participants, all with urinary incontinence associated with a stroke occurring 6 months or more prior to the study, achieved (and maintained) continence. The biofeedback procedures in this study did not alter the physiological status of the bladder but did teach these stroke patients effective methods for managing a low-volume, hyperreflexic bladder and preventing precipitous, uncontrolled voiding.

Neither scheduled voiding nor spontaneous changes over time could account for these improvements. All subjects were placed on a scheduled voiding program for 1 month prior to biofeedback training, and none became continent with scheduled voiding alone. This was not surprising since the subjects reported frequent toileting, sometimes as often as every 30 to 60 minutes, prior to entering this study. In addition, the response to biofeedback-assisted bladder retraining (Table II) was measured in terms of change from the scheduled-voiding baseline and so was independent of gains that may have occurred with the scheduled voiding regimen alone. Spontaneous recovery was also not likely to have been a factor in this study. All four participants were 6 months or more poststroke and therefore had minimal chance of spontaneous improvements in urinary incontinence (Brocklehurst et al., 1985). All four had stable histories of persistent urinary incontinence and showed improvements coinciding with the onset of biofeedback training.

The improvements made by these stroke patients can be attributed to the biofeedback-assisted sensorimotor retraining procedures implemented in this study. However, these procedures had a number of different components, and it is not possible to say which aspects of the treatment program were most essential or even contributory. The four subjects differed considerably with regard to bladder characteristics and their cognitive and functional capacities, and it is possible that different components of the treatment package were primary for different subjects. The procedures used in this initial study were designed to be clinically comprehensive, and to provide objective measurements of bladder sensations and bladder contractions for purposes of evaluation as well as biofeedback training. Less comprehensive, simpler procedures may be equally effective, at least for some individuals.

The fact that subjects did not alter their bladder threshold for sensation or bladder capacity during the stepwise bladder-filling procedure across sessions (Table III) suggests that direct bladder pressure feedback may not be necessary. If this is indeed the case, it may be possible to simplify the

treatment procedures considerably and focus on biofeedback-assisted sphincter retraining procedures. However, the bladder-filling and bladder-feedback procedures in this study did offer several distinct advantages that are likely to have contributed to the effectiveness of the treatment program in this initial study. The stepwise bladder-filling procedure was very useful in teaching subjects to identify, interpret, and employ their full range of available bladder sensations. The home program emphasized early action based on early warning signals that occur before the urge to void, and this sensory retraining process may be important, particularly in patients with sensory abnormalities.

The bladder-filling and monitoring procedures also provided a wealth of information on the sensory status and motor function of the bladder of individual subjects, which made it possible to tailor the treatment approach precisely to the needs of each. Such individualization is likely to enhance treatment effectiveness. For example, during the bladder pressure feedback procedure, it was evident that subject 1 consistently had only 15 to 30 seconds' warning time prior to reflex voiding and could not use early bladder sensations to prevent incontinence. His treatment program therefore emphasized the use of voluntary sphincter contractions to inhibit the bladder and maximize sphincter control in combination with continued adherence to scheduled voiding.

The information derived from bladder monitoring also led to improved understanding of the various sensorimotor deficits that contribute to stroke-associated urinary incontinence. This led, in turn, to important refinements in the overall treatment protocol. For example, as noted above, subject 2 showed a flaccid bladder-filling pattern while lying down during cystometrogram, although his verbal report was consistent with a diagnosis of hyperreflexic bladder. In the course of biofeedback training it was possible to observe, and document, that his bladder threshold for uninhibited contractions was markedly lower when standing than while lying down. This objective finding was consistent with verbal reports by all four subjects that the process of standing and walking to the toilet posed special problems, even for those who could walk independently and with reasonable speed (subjects 2 and 4). Procedures were added to the protocol to address this problem: Subjects were educated about this aspect of bladder function and were taught to use voluntary contractions of the pelvic floor muscles to inhibit bladder reflexes *prior* to standing and walking to the bathroom. The information provided by physiological monitoring procedures, therefore, can function as "feedback for the clinician and researcher"; such information is vital in developing optimally effective treatment procedures for urinary incontinence for patients with neurological diagnoses. At the same time, it is possible that these treatment procedures, when they are fully developed, may not require bladder monitoring to provide "feedback for the

patient" or may require such biofeedback for selected patients only. That remains to be seen.

In the present study, the external anal sphincter feedback appeared to make a strong contribution to clinical outcome. Prior to training, all patients had the neurological capacity to produce a relatively strong, sustained contraction of the pelvic floor muscles. However, these key muscles were characteristically very slow and inconsistent in developing a maximal contraction. Three of the subjects had some degree of fecal incontinence, and none was able to use the pelvic floor muscles effectively to interrupt the urinary stream while voiding. All four subjects showed improvements in EAS control across training sessions, fecal incontinence cleared, and three subjects reported being able to interrupt the urinary stream during home practice and also reported good success with the strategy of using voluntary sphincter contractions to postpone voiding.

An important implication of this study is that urinary incontinence can occur, and persist, in stroke patients who are cognitively and physically very functional. Incontinence following a stroke often clears spontaneously in the course of neurological recovery, and it is often assumed that those who are left with residual incontinence do not have the capacity to reacquire voluntary control owing to neurological limitations, cognitive deficits, or problems with mobility and self-care. The participants in this study had serious strokes that left them with residual spastic hemiparesis. However, all had been good rehabilitation candidates and had benefited from rehabilitation hospitalizations. All returned home still partially incontinent (three with some degree of double incontinence) in spite of standard bowel and bladder training. These individuals responded well to biofeedback training and achieved continence.

It is not possible to tell, on the basis of this initial study, how typical these subjects are or how many other patients there are with incontinence associated with stroke who could similarly benefit from these procedures. Such information will only come with additional research studies and more extensive clinical trials. This study does provide evidence that there are stroke patients with persistent urinary incontinence who are capable of regaining effective control with behaviorally oriented, biofeedback-assisted treatment procedures. Since stroke is a major contributor to urinary incontinence in the elderly, and the costs of this medical problem are enormous (Hu, 1986), further development of this promising new treatment approach should be vigorously pursued.

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