THE DYSFUNCTIONAL 'LAZY' BLADDER SYNDROME IN CHILDREN*

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The dysfunctional 'lazy' bladder syndrome is one of the common causes of recurrent urinary tract infections in children. A complete urological work-up in patients with this syndrome fails to identify underlying *obstructive* or *neurological* lesions. In a series of 114 cases where the bladder pathology was the main feature, 84 had the dysfunctional 'lazy' type of bladder, 12 had cord bladders, and 18 had demonstrable obstructive lesions. The detailed study of these patients and their follow-up, particularly from the physiological standpoint, has resulted in a better understanding of the pathogenetic mechanism.

The dysfunctional 'lazy' bladder occurred prevalently between 2 and 6 years of age and was twice as common in females as in males. Diagnosis was usually made late with an average delay of 18 months, probably as a result of minimal urinary tract symptoms. More than half of the cases initially presented with fever of unknown origin, and in a number of these patients, abdominal pain, nausea and vomiting were associated symptoms. They were treated before admission to the hospital with antibiotics on repeated occasions without a urological cause being suspected. Some patients were referred to the hospital because of an abdominal mass which subsequently proved to be an enlarged bladder. Symptoms referable to the urinary tract, when they did occur, varied from infrequent voiding to incontinence and occasionally frequency. Over 80% of the patients received one or more courses of antibiotics before the diagnosis was established. The bladder was palpable in the majority of cases; the kidneys were rarely palpable. No patient presented with uraemia.

Urine analysis on admission to the Boston Floating Hospital for Infants and Children showed gross pyuria, and the culture indicated significant infection. The most prevalent organism was *Escherichia coli* in about 70% of the cases, 12% of

which were resistant to the commonly used antibiotics, such as penicillin, streptomycin, tetracycline and chloramphenicol. The other organisms cultured, *Proteus vulgaris*, *Aerobacter aerogenes* and *Pseudomonas*, were usually encountered in patients treated with several courses of antimicrobial agents. They probably represented a super infection and were resistant in many cases to most antimicrobial agents.

Post-voiding catheterization commonly demonstrated a residual urine which ranged from 30 to 200 ml., and this finding was also confirmed by post-voiding cystograms. It should be emphasized that the presence of vesico-ureteral reflux may give a false estimation of the volume of residual urine. This is due to the fact that, after voiding, the urine which refluxes into the ureters returns into the bladder. The bladder on cystography was usually smooth in contour and enlarged, and ureteral reflux could be seen (Fig. 1). Occasionally, in early cases, reflux was observed only during micturition. Varying degrees of vesico-ureteral reflux occurred bilaterally in 40 patients and unilaterally in 24 patients. Cystoscopic examination revealed a normal bladder neck and rarely trabeculation. In 51 patients with reflux, the ureteral orifices were wide and gaping; in 13 they appeared normal. Excretory urography usually showed hydronephrosis and hydro-ureters (Fig. 2).

The function of the dilated ureters was evaluated by measuring their peristalsis manometrically. Many cases demonstrated decreased amplitude and frequency of peristalsis, while some ureters showed ineffectual peristalsis occurring particularly when there was severe active infection and reflux. In some of these patients normal peristaltic activity returned after treatment.

The cystometrographic studies using a Statham transducer and a polygraph were of particular interest because of their characteristic abnormal pattern. A normal cystometrogram was characterized by occasional small contractions and by a gradual elevation of intravesical pressure which initiated involuntary bladder contractions on reach-

* A paper read at meeting of the British Association of Paediatric Surgeons in Stockholm, September 1961.

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FIG. 1.—Cystogram demonstrating bilateral vesico-ureteral reflux as well as bilateral hydro-ureters and hydronephrosis.

FIG. 2.—Excretory urogram showing hydronephrosis and hydro-ureters.

ing approximately 15 to 20 cm. of water pressure. If the patient did not respond to the urge to void, the contractions disappeared and the intravesical pressure decreased temporarily to rise again gradually to the previous level at which involuntary contractions appeared. This phase is attributed to the ability of the bladder to adapt itself when the stimulus of emptying is ignored (Fig. 3). By contrast, in the dysfunctional 'lazy' bladder syndrome, the cystometrogram showed a gradual elevation of the intravesical pressure beyond the physiological limits which normally initiate involuntary bladder contractions. On further filling, partially effective involuntary contractions coinciding with the patient's urge to void could be obtained in early cases. At this point, by requesting the patient to strain, thereby increasing the intra-abdominal pressure, the complete emptying of the bladder was usually achieved (Fig. 4a). On the other hand, if the patient did not respond to the urge to void, the contractions disappeared and the intravesical pressure decreased temporarily to rise again with the possible appearance of less effective involuntary contractions and the urge to void. This phenomenon of adaptation partially simulates the one occurring in the normal bladder. It is necessary to emphasize that a cystometrogram was considered reliable only when vesico-ureteral reflux was absent or occurred at elevated bladder pressures. The cystometrogram of the dysfunctional 'lazy' bladder syndrome may vary from one characterized by a gradual elevation of intravesical pressure beyond physiological limits with partially effective involuntary bladder contractions to one with no involuntary contractions (Fig. 4b).

It should be noted that the cystometrogram of the dysfunctional bladder differs from the one associated with peripheral lesions of the parasympathetic system of the bladder. In the latter lesions the bladder may lack tone as well as involuntary contractions. The dysfunctional bladder, however, has normal tone as evidenced by its ability to adapt itself to variations in bladder volume.

From the cystometrographic studies of the dysfunctional 'lazy' bladder which demonstrated an elevated threshold for the initiation of bladder contractions as well as the ease and rapid adaptation of the bladder to inhibition of voiding, we believe that this condition is more related to a deranged bladder function rather than to an anatomical and/or neurological abnormality. The abnormally high threshold has developed from the habitual neglect of the patient to empty the bladder on getting the urge to micturate, which has resulted

FIG. 3.—Normal cystometrogram demonstrating normal contractions at 20 cm. H₂O pressure.

FIG. 4a.—Characteristic cystometrogram of dysfunctional 'lazy' bladder demonstrating minimal involuntary bladder contractions and abnormal intravesical pressure of 35 cm. H₂O.

FIG. 4b.—Cystometrogram of dysfunctional 'lazy' bladder demonstrating no involuntary bladder contractions.

FIG. 5.—Cystogram after therapy demonstrating no reflux.

in the bladder yielding and its subsequent enlargement. In other words, this closely simulates many children with chronic constipation who have also lost the urge to evacuate in response to the normal stimuli. Both conditions with further

TABLE 1
PERISTALTIC ACTIVITY OF THE URETERS

	Before Therapy	After Therapy	
		Improved	No Improvement
Normal	42	42	
Poor	26	20	6
Absent	16	9	7
Total number of cases ..	84	71	13

TABLE 2
URETERAL REFLUX

	Before Therapy	After Therapy	
		No Reflux	Reflux Present
No reflux	20	20	
Unilateral reflux	24	18	6
Bilateral reflux	40	26	14
Total number of cases ..	84	64	20

neglect develop into a vicious cycle. In chronic constipation we get megarectum and megacolon; whereas, in the dysfunctional 'lazy' bladder we get bladder enlargement and infection. The uretero-vesical junction ultimately becomes incompetent and vesico-ureteral reflux occurs, favouring the spread of infection and contributing to dilatation of the ureters. The secondary sequelae are far more serious in the urinary tract than they are in the cases of chronic constipation.

The exact role of infection in the pathogenesis of the dysfunctional 'lazy' bladder is not clearly defined. With the present diagnostic measures we can only say that infection accentuates the deranged bladder function. Further experimental and clinical study may eventually clarify the role of infection.

The management of these patients necessitates regular follow-up to evaluate the efficacy of therapy and the status of the urinary tract. Early cases with residual urine of less than 50 ml. and not associated with reflux are treated conservatively. They are trained to void voluntarily every three to four hours with the aim of re-establishing the urge to void at a low threshold stimulus and the return of normal bladder size. Infection should be treated with a specific antimicrobial agent during the acute episodes for no longer than five to nine days, to be followed by long-term suppressive therapy of either sulfisoxazole, methenamine mandelate, or nitrofurantoin. These patients are followed monthly with a urine culture and colony count. Cases complicated with minimal ureteral reflux are first managed conservatively. These patients are instructed to practise double or triple micturition to ensure complete emptying of the bladder.

If, after six months of conservative treatment, there is no response, then the medical measures may be rendered effective by diminishing the normal bladder neck resistance by performing a Y-V plasty of the bladder neck. Plastic repair of the ureteral orifice is indicated when ureteral reflux occurs at low bladder pressures and when peristalses are present.

Following the management outlined, out of the 42 patients with ineffectual ureteral peristalses, 29 showed an improvement of peristalsis (Table 1). No reflux could be demonstrated in 44 patients, while it persisted only during micturition or at elevated bladder pressures in the remaining 20 (Fig. 5 and Table 2).

Excretory urography showed regression of the hydronephrosis and hydro-ureter in those without reflux (Fig. 6). The cystometrograms in the improved cases demonstrated more effectual involuntary and voluntary contractions (Fig. 7).

Normal bladder function was also confirmed by a residual urine of less than 10 ml.

Conclusion

The dysfunctional 'lazy' bladder syndrome is characterized by progressive urinary tract changes. Once diagnosed, it is necessary to evaluate the extent of the disease in order to institute the proper therapy. Patients with minimal pathological changes respond favourably to conservative treatment. Advanced cases with high residual urine and severe degree of reflux necessitate prompt surgical therapy, consisting of Y-V plasty of bladder neck with occasional plastic repair of the ureteral orifices.

Summary

A disorder of bladder function neither obstructive nor neurological but secondary to habitual urinary retention and infection is described.

The delay in diagnosis is attributed to the minimal urinary tract symptoms. The cystometrographic pattern of the dysfunctional 'lazy' bladder syndrome is described. The pathogenesis of the condition based on the cystometrographic studies is postulated. The patients were followed up for six months to nine years. The results of therapy of 84 patients with dysfunctional bladder is reviewed.

FIG. 6.—Excretory urogram showing improvement of hydronephrosis and hydro-ureters.

FIG. 7.—Cystometrogram after therapy demonstrating more effectual involuntary bladder contractions and normal intravesical bladder pressure.