Biofeedback training in chronic constipation

M A Benninga, H A Büller, J A J M Taminiau

Abstract

Twenty nine patients, aged 5–16 years, were studied to evaluate whether biofeedback training is effective in treating children with chronic constipation and encopresis; the clinical outcome at six weeks and 12 months was also evaluated. Patients received on average five biofeedback training sessions. The existence of external anal contraction or decreased rectal sensation in 16 (55%) and eight (27%) of the children, respectively, was identified on manometry. After biofeedback training, 26 (90%) of the patients learned to relax the external anal sphincter; 18 (63%) normalised rectal sensation. The training resulted in a significant increase in defecation frequency and a significant decrease in encopresis. At six weeks, 16 (55%) of the patients were clinically symptom free. At follow up after 12 months the results were sustained. Only three patients showed a relapse within six months, of whom two were successfully treated with one extra training session. Biofeedback training might be a useful therapeutic approach in children with chronic constipation and encopresis.

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Although constipation is not a disease, it often constitutes a major problem for the child and his or her family. Similarly, it poses an enormous challenge to the physicians and other care givers responsible for the treatment of affected children. By definition, constipation implies an infrequent, sometimes painful, defecation occurring over at least a three month period with two or fewer bowel movements weekly.1 Often constipation is accompanied by abdominal pain, poor appetite, or enuresis. Importantly, the only symptom of constipation may be encopresis: the involuntary deposition of loose or formed stools in the underwear after the age of 4 years. The prevalence of constipation is approximately 3% in an average paediatric practice.2 3 The mechanism responsible for constipation has not yet been elucidated. Many different hypotheses have been put forward: change from breast to formula feeding, too strict or too early toilet training, aberrant child-parent interaction, or low fibre or liquid intake.

Children with constipation are conventionally treated with laxatives for prolonged periods in combination with habit training and adequate fibre and fluid intake. However, in 15–40% of the patients this treatment is not effective.1 3 Recent studies, using anorectal manometry, showed abnormal defecation dynamics in 36–52% of children with chronic constipation and encopresis.4 5 These patients close their anal canal by contracting rather than relaxing the external anal sphincter during defecation. Because this sphincter is a striated muscle, it is possible to instruct these children how to control this muscle by using biofeedback training. The aim of this study was to evaluate whether biofeedback training is effective in treating children with chronic constipation and encopresis.

Patients and methods

PATIENTS

Twenty nine otherwise healthy children (5–16 year of age) with constipation and/or encopresis from the outpatient clinic of a tertiary academic teaching hospital were enrolled consecutively. Constipation was defined as two or fewer bowel movements per week. Encopresis was defined as the loss of faecal material in the underwear two or more times per week. By definition, encopresis as the presenting symptom was diagnosed in children only after the age of 4 years. All children with Hirschsprung's disease, spinal and anal anomalies, surgery of the colon, metabolic diseases, mental retardation, or who used drugs other than laxatives were excluded. The study was approved by the medical ethical committee. Written informed consent was obtained from patients and their parents. Controls consisted of 13 healthy children (8–16 years, average 12 years; there were eight boys and five girls) and were recruited from siblings and friends of the medical staff and from siblings and friends from paediatric patients. A written informed consent was obtained from all subjects.

METHODS

Each child underwent a complete work up that included a detailed medical history and a thorough physical, including rectal, examination.

Manometry

A manometric anal probe of 4·8 mm outer diameter and 0·8 mm inner diameter was used. Two side holes spacing 3 cm were perfused with sterile water at a rate of 0·5 ml/min by a hydraulic infusion system (Arndorfer). A rectal distending balloon with a high compliance was tied to the tip of the probe, 3 cm above the first side hole. Pressures were measured by transducers situated in each perfusion line and
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connected to PC Polygraf HR preamplifiers (Synectics Medical). The signals from the preamplifier were digitally converted by an analog-digital converter and information was transmitted via a fibreoptic cable to a personal computer.

Electromyography (EMG)
Electrical activity of the external anal sphincter was recorded by one indifferent and two differential electrodes connected to a bioamplifier II and the PC Polygraf HR. Pregelled disposable neonatal electrocardiography electrodes overlying the subcutaneous part of the external anal sphincter were used. The indifferent electrode was located on a thigh.

Manometry and EMG
Without bowel preparation, manometry and EMG were performed with the patient in the left lateral decubitus position. Maximal anal resting tone (MART) and maximal squeeze pressure were measured by stationary pull-through at a rate of 1 cm/min. Sensory threshold was defined by the smallest reproducible volume of rectal distention sensed. The defection dynamics, the manometric profile obtained by expelling the rectal balloon, was assessed during at least five simulated defection trials. Defecation was defined as normal if the integrated EMG of the external anal sphincter showed a decrease or no change during expulsion of the balloon in at least two of five defection attempts. Defecation dynamics were defined as abnormal if manometric and myoelectrical increase occurred during bearing down in four out of the five defection attempts. The rectoanal inhibitory reflex was measured in response to balloon distention to exclude (short segment) Hirschsprung’s disease.

Biofeedback training
Patients were shown normal manometric and EMG tracings, and were trained to recognise the responses of the external anal sphincter during contraction and simulated defection. After insertion of the anorectal probe, sensory threshold was determined by inflating the balloon and requesting the patient to contract the external anal sphincter whenever rectal sensation was perceived. Subsequently, the balloon was inflated with 20 ml of air and the patient was instructed to increase intra-abdominal pressure and to relax their external anal sphincter. If the child did not understand the relaxation procedure, the rectal balloon was filled with 50 ml of air to demonstrate the tracing of a relaxation on the monitor. Verbal reinforcement was given while the patient experimented and tried to modify the response. As part of the training, visual feedback was withheld by blocking the patient’s view. Sphincter exercises were prescribed and the patient was encouraged to employ the techniques at the slightest sensation of rectal fullness. Each session lasted 35–45 minutes once a week until relaxation of the external anal sphincter without visual feedback was accomplished.

Patients were divided in three groups based on symptoms. The first group consisted of children who fulfilled the criteria of constipation without encopresis (group I). The second group had symptoms of constipation in the presence of encopresis (group II). The third group consisted of patients who did not meet the criteria of constipation, but only complained of encopresis (group III). Laxative use was diminished only when clinical symptoms improved. Parents and children kept a diary of bowel movements, encopresis, and laxative use. Evaluation of patients occurred six weeks and 12 months after the last training. Patients were considered to have clinically recovered if they did not use any laxatives for at least four weeks and met the following criteria (a) two or more bowel movements per week and (b) two or fewer soiling episodes per month.

Statistical methods
Data were collected using an integrated patient database (Patfile). Statistical methods included the Wilcoxon paired signed ranks test, with significance accepted at 5% level. Results were expressed as mean (SEM).

Results
PATIENTS
All 29 patients enrolled in the study finished the training period and all except two were available for follow up. Twenty of the 29 patients (68%) were on laxatives for at least five months before the beginning of the study period. Table 1 shows the number of boys and girls and their respective ages at the beginning of the training in the three different groups. The majority of patients demonstrated encopresis with or without signs of constipation. Furthermore, the age of onset of symptoms and the genders of the patients did not vary significantly.

### Table 1 Number (%) of patients, boys and girls, age of onset of symptoms and mean (range) ages are given in the three different groups (total children=29)

<table>
<thead>
<tr>
<th></th>
<th>Constipation (group I)</th>
<th>Constipation and encopresis (group II)</th>
<th>Encopresis (group III)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Children in group</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boys</td>
<td>5 (17)</td>
<td>13 (45)</td>
<td>11 (38)</td>
</tr>
<tr>
<td>Girls</td>
<td>2 (40)</td>
<td>8 (62)</td>
<td>7 (64)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>3 (60)</td>
<td>5 (38)</td>
<td>4 (36)</td>
</tr>
<tr>
<td>Age of onset of symptoms (months):</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6-12</td>
<td>3 (60)</td>
<td>6 (46)</td>
<td></td>
</tr>
<tr>
<td>&gt;12-48</td>
<td>1 (20)</td>
<td>2 (10)</td>
<td></td>
</tr>
<tr>
<td>≥48</td>
<td>1 (20)</td>
<td></td>
<td>11 (100)</td>
</tr>
</tbody>
</table>

Presenting Clinical Symptoms
In table 2 the clinical symptoms at the time of the first training are summarised for the different groups. The defection frequency in children with constipation alone (group I) was fewer than three bowel movements weekly and was similar in children with constipation and encopresis (group II). In contrast, the group of children with encopresis alone (group III) showed a normal defection frequency. Encopresis was
not present in group I and was similar in frequency in group II and III. Abdominal pain and poor appetite was present in at least a quarter of all patients. Enuresis was not found in group I but present in approximately 40% of the patients in the two groups with encopresis. Pain during defection was only seen in a minority of the patients. An abdominal or rectal palpable mass was found in group I and II but not in group III.

ANORECTAL MANOMETRY
Table 3 shows the manometric findings. The MART and maximal squeeze pressure at the first and last biofeedback training were not significantly different in the three separate groups. In all groups there was a tendency of a lowering of the sensory threshold, only in group III did this reach statistical significance. In the total group there was a significant decrease in MART and sensory threshold. In addition, the defecation dynamics in the total group showed an increment from 45% before training to 90% after intervention. All children exhibited a normal rectoanal inhibitory reflex upon balloon distension.

Table 2  Clinical symptoms in the three different groups (total children=29)

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Constipation (group I, n=5)</th>
<th>Constipation and encopresis (group II, n=13)</th>
<th>Encopresis (group III, n=11)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (SEM) defection frequency</td>
<td>2.6 (1.2)</td>
<td>1.1 (0.3)</td>
<td>9.6 (3.2)</td>
</tr>
<tr>
<td>Mean (SEM) encopresis</td>
<td></td>
<td>8.1 (1.9)</td>
<td>7.5 (2.5)</td>
</tr>
<tr>
<td>No (%) with:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>2 (40)</td>
<td>3 (23)</td>
<td>3 (27)</td>
</tr>
<tr>
<td>Poor appetite</td>
<td>4 (80)</td>
<td>5 (38)</td>
<td>5 (46)</td>
</tr>
<tr>
<td>Enuresis</td>
<td>0</td>
<td>5 (38)</td>
<td>5 (46)</td>
</tr>
<tr>
<td>Pain during defection</td>
<td>2 (40)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Abdominal or rectal palpable mass</td>
<td>1 (20)</td>
<td>4 (31)</td>
<td>0</td>
</tr>
</tbody>
</table>

Table 3  Mean (SEM) manometric findings before (-1) and after (-5) biofeedback training in the different groups for MART, maximal squeeze pressure (MSP), and sensory threshold and percent of normal for defecation dynamics (DD)

<table>
<thead>
<tr>
<th>MART-1</th>
<th>MART-5</th>
<th>MSP-1</th>
<th>MSP-5</th>
<th>ST-1</th>
<th>ST-5</th>
<th>DD-1 (%)</th>
<th>DD-5 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>43.6 (7.8)</td>
<td>34.6 (7.9)</td>
<td>110.4 (24.9)</td>
<td>124.4 (29.2)</td>
<td>37.0 (9.4)</td>
<td>33.0 (7.0)</td>
<td>60</td>
<td>80</td>
</tr>
<tr>
<td>46.4 (5.4)</td>
<td>35.2 (2.5)</td>
<td>130.0 (10.8)</td>
<td>122.4 (9.4)</td>
<td>43.0 (7.1)</td>
<td>37.3 (7.3)</td>
<td>59</td>
<td>92</td>
</tr>
<tr>
<td>42.8 (4.9)</td>
<td>39.4 (3.6)</td>
<td>124.3 (15.0)</td>
<td>125.6 (7.5)</td>
<td>35.0 (3.8)</td>
<td>25.0 (3.3)</td>
<td>46</td>
<td>91</td>
</tr>
<tr>
<td>44.5 (3.2)</td>
<td>36.7 (2.1)</td>
<td>124.7 (8.3)</td>
<td>124.0 (6.7)</td>
<td>38.9 (3.8)</td>
<td>31.8 (3.7)</td>
<td>45</td>
<td>90</td>
</tr>
<tr>
<td>55.3 (4.5)</td>
<td>182.6 (16.7)</td>
<td>192 (3.2)</td>
<td>92</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 4  Mean (SEM) frequency of defection and encopresis for the different groups before and after training

<table>
<thead>
<tr>
<th>Defecation frequency:</th>
<th>Constipation (group I, n=5)</th>
<th>Constipation and encopresis (group II, n=13)</th>
<th>Encopresis (group III, n=11)</th>
<th>Total (n=29)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before</td>
<td>2.6 (1.1)</td>
<td>1.1 (0.3)</td>
<td>9.6 (3.2)</td>
<td>4.6 (1.4)</td>
</tr>
<tr>
<td>After</td>
<td>5.6 (0.7)</td>
<td>5.3 (0.6)</td>
<td>8.5 (1.4)</td>
<td>6.5 (0.6)</td>
</tr>
<tr>
<td>Follow up</td>
<td>4.0 (1.2)</td>
<td>5.1 (0.7)</td>
<td>10.4 (1.0)</td>
<td>6.9 (0.7)</td>
</tr>
</tbody>
</table>

Encopresis: Before | 8.1 (1.9) | 7.5 (2.5) | 6.5 (1.3) | 1.5 (0.5) |
After | 0.9 (0.4) | 3.0 (1.2) | 0.8 (0.6) | 0.3 (0.1) |
Follow up | 0.4 (0.2) | 0.4 (0.4) | 0.2 | 0.2 |

Discussion
For years, chronic constipation in children has challenged paediatricians, but little progress has been made regarding the underlying mechanism. In addition, the primary treatment is still symptomatic. The development of manometry, however, has shed some new light on the possible role of the anal sphincters in this disorder. This study describes the use of

EFFECT OF BIOFEEDBACK TRAINING AND CLINICAL OUTCOME AT FOLLOW UP
Patients received on average five biofeedback training sessions.
As shown in table 4 the training had an important clinical effect, as illustrated by a significant increase in defection frequency in group I and II. Similarly, the training contributed to a significant decrease in encopresis. At follow up after 12 months the results were sustained. Only three patients showed a relapse within six months, of which two were successfully treated by one extra training session. Laxative use decreased from 68% to 24% in the total group at follow up. When all patients were assessed using the clinical criteria mentioned under study protocol 16 (55%) were successfully treated. In 11 of the 13 patients who did not respond to the training, lack of motivation, no ability to wifully relax their anal sphincter, or no improvement of their rectal sensation formed the reason of failure. In the others the symptoms of constipation and encopresis disappeared but laxative use could not be stopped. No attempt was made to assess the possible negative effect or feeling of punishment by the insertion of rectal probes as part of the biofeedback training on the outcome in these patients.
Biofeedback training in chronic constipation

biofeedback training in the treatment of chronic constipation in children. On manometry we observed the existence of two distinct phenomena. One was an inadequate contraction of the external sphincter during defecation causing an outlet obstruction and resulting in constipation and/or encopresis. The second was a decreased rectal sensation as illustrated by increased sensory threshold, also resulting in constipation and/or encopresis. These different manometric entities were both treated effectively with biofeedback training. The training was in essence similar, but the emphasis was on either rectal sensation or sphincter relaxation in the two different groups.

In 90% of all children in the present study defecation dynamics upon manometry normalised after biofeedback training. In the majority of these patients this resulted in a partial or total clinical improvement, with a significant increase in defecation frequency and decrease of encopresis episodes. Using strict clinical criteria, namely normal defecation without encopresis and no use of laxatives, complete remission was achieved in 55% of all children enrolled. This percentage of success was maintained after a 12 month follow up.

The age of onset of constipation in this study was often long before the beginning of toilet training, emphasising the minor part toilet training plays in the pathogenesis of constipation. Most children in this study had encopresis indicating the prevalence of this socially unacceptable behaviour. It is generally believed that encopresis, which is not observed in adults, is the result of constipation. However, encopresis with normal defecation frequency and no other signs of constipation, as this study shows, might be the only complaint.

The normal defecation frequency and lack of physical signs of constipation in the group with only encopresis (group III) compared with the abnormal frequency in the group with constipation and encopresis (group II) suggests a different pathophysiology. One mechanism might be differences in rectal control or sensitivity, as indicated by the significant decrease in sensory threshold in group III after biofeedback training. Furthermore, the absence of abdominal or rectal palpable mass in group III underscores this suggestion and makes overflow incontinence, the mechanism in group II, less likely. Another explanation for the encopresis in group III might be the absence of discipline or denial to go to the toilet when the urge to defecate disturbs their daily activity. In group II, the encopresis was due to primary distention of the rectal wall with absent or lowered rectal sensation. The values found for MART and maximal squeeze pressure in all groups confirms the existence of a normal resting tone and squeeze function of the anal sphincters.

In accordance with other studies, we found that on average 55% of the children with chronic constipation and/or encopresis have abnormal defecation dynamics. A larger study is currently underway to assess the frequency of abnormal dynamics in a less selected population of children with constipation.

Although this study describes the manometric profiles and results of biofeedback training in a small group of children, the findings are encouraging, and a larger controlled study is now being conducted to establish further the therapeutic effect of biofeedback training and possibly elucidate the underlying mechanisms of constipation and encopresis.

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