Biofeedback-Assisted Relaxation in Type 2 Diabetes

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OBJECTIVE — The objective of this randomized controlled study was to determine the effects of biofeedback and relaxation on blood glucose and HbA$_{1c}$ (A1C) in patients with type 2 diabetes.

RESEARCH DESIGN AND METHODS — Patients with type 2 diabetes were randomized to either 10 sessions of biofeedback (electromyograph and thermal) and relaxation or 3 sessions of education. All sessions were individual. A total of 39 participants were entered, and 30 completed the 3-month protocol. Average blood glucose, A1C, forehead muscle tension, and peripheral skin temperature were assessed, and inventories measuring depression and anxiety were administered prerandomization and after completion of treatment/control.

RESULTS — Biofeedback and relaxation were associated with significant decreases in average blood glucose, A1C, and muscle tension compared with the control group. At 3-month follow-up, the treatment group continued to demonstrate lower blood glucose and A1C. Both groups decreased scores on the depression and anxiety inventories. Patients with depression had higher blood glucose levels and tended to drop out of the study.

CONCLUSIONS — This study supports the use of biofeedback and relaxation in patients with type 2 diabetes for up to 3 months after treatment. Further research is necessary to determine the long-term effects of biofeedback and the effects of mood on patients' responses to treatment.

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Management of type 2 diabetes requires continuous monitoring and multiple interventions to prevent long-term complications (1). One of the contributing factors in the etiology of glucose intolerance and poor glycemic control in individuals with diabetes is the stress response (2,3). Based on this premise, several studies have evaluated stress management techniques as adjunctive in the treatment of patients with diabetes. Stress management is a generic term that may encompass biofeedback, relaxation, cognitive behavioral therapy, and imagery. Biofeedback is a therapeutic technique involving an instrument that provides information about physiological activity, such as skin temperature or muscle tension, with the objective of learning control over maladaptive responses to stress. Biofeedback is commonly coupled with relaxation therapy, which can comprise deep breathing, progressive relaxation, or imagery (4). Jablon et al. (5) examined the effects of progressive relaxation training and electromyograph (EMG) biofeedback on patients with type 2 diabetes. Patients demonstrated significant reductions in stress levels but were unable to improve diabetic metabolic control as measured by fasting blood glucose, 2-h postprandial blood glucose, and fructosamine.

Surwit et al. (6) tested the effects of education with and without stress management in 108 patients with type 2 diabetes. None of these patients had a psychiatric diagnosis. After 1 year, a small but significant decrease was observed in glycohemoglobin in the group that received education with stress management. In a controlled study of biofeedback-assisted relaxation therapy (BFRT) in type 1 diabetes (7), decreases in blood glucose were found in the treated group compared with a wait-list control group. However, patients with symptoms of depression did not benefit from BFRT and had minimal decreases in blood glucose.

The moderating effects of mood on glycemic control highlight the complex relationship between depression and diabetes (8). The prevalence of mood and anxiety disorders is higher in individuals with type 2 diabetes compared with the general population (9). Depressive symptoms are associated with hyperglycemia, poor glycemic control, and more serious complications in type 1 and type 2 diabetes (10–12).

Given the conflicting results of previous studies, this study was designed to test the hypothesis that BFRT would decrease blood glucose and HbA$_{1c}$ (A1C) in patients with type 2 diabetes compared with an education group that was not expected to change significantly. Since depression and anxiety are known to affect glycemic control in type 2 diabetic patients, this study also evaluated those parameters as modulators of diabetic control.

RESEARCH DESIGN AND METHODS

Thirty-nine patients with type 2 diabetes were recruited using handouts distributed at diabetes education sessions or were referred from local physicians. During the intake session, patients signed an institutional review board–approved consent form and privacy (HIPAA) documents. A brief history was taken regarding length of illness, prior medical therapy of diabetes, and self-care habits such as exercise and diet,

Table 1—*Comparison of dropouts and protocol completers*

	Dropouts	Completers
Age (years)	50.33 ± 9.69 (9)	52.63 ± 76 (30)
Pretest blood glucose (mmol/l)*	11.3 ± 3.5 (5)	8.3 ± 2.1 (30)
Pretest trait anxiety	47.4 ± 10.8 (8)	41.8 ± 13.3 (30)
Pretest depression*	22.9 ± 12.6 (8)	12.5 ± 9.2 (30)

Data are means ± SD (*n*). *Significant difference between groups, $P < 0.05$.

daily routine such as occupation, current living situation, support system, major sources of stress, and prior psychiatric history. The patients' diabetes care providers were contacted for written permission for their patient to participate.

Pretest (baseline) measurements for each subject were recorded during a 4-week period. Patients were asked to log morning and evening blood glucose, yielding 20–30 values. Two sets of psychophysiological assessments were taken and the values averaged. These comprised forehead muscle tension taken using a J & J M-57 Electromyograph (EMG), finger skin temperature using a T 808A Bio-Logic device, blood pressure, and weight. Assessments for depression and anxiety were carried out using the Beck Depression Inventory II (BDI-II) (13) and Spielberger State-Trait Anxiety Inventory (14), respectively. If a BDI-II score ≥11 was reported, the subject was asked to schedule an interview with a psychiatrist, and if recommended, prescribed 50 mg sertraline for the duration of the study. The 50-mg dose was stabilized for at least 2 weeks before the subject entered the pretest measurement period and continued on in the study; a blood sample was drawn for A1C, an indicator of metabolic control for the previous 10–12 weeks. Reference range in our laboratory was 4.0–6.2%.

After pretest, patients were randomly assigned to the BFRT treatment group or the diabetes education control group. BFRT consisted of 10 once-a-week, individual, 45-min sessions. Feedback consisted of either EMG (five sessions) or thermal (five sessions) biofeedback and relaxation training (4,15). Patients were presented with fluctuating sounds and numerical values that indicated their muscle tension level or finger temperature. A 15-min audiotape was provided to the patients for a recommended twice-daily home practice of relaxation. Part of each session fostered a problem-solving approach to stress management and self-care, emphasizing that the subject could decrease the stress response. As part of the learning process, patients were encouraged to experience a sense of control over their physiological responses to stress. The feedback was used to demonstrate that the subject could in fact control facial muscle tension and skin temperature by utilizing relaxation and simple imagery. It was suggested that similar control over blood glucose was possible.

Diabetes education consisted of three individual sessions of 60–75 min held 3–4 weeks apart. Each session was taught by a registered nurse and addressed the topics of different types of diabetes, portion control, glycemic index, exercise, monitoring blood glucose, effects of medication, effects of stress, prevention of complications of diabetes, and the need for consistent medical check-ups. Patients were given take-home handouts of this information.

Four weeks after the completion of the treatment/control group sessions, all psychophysiological measurements were repeated and recorded as posttest data. Average blood glucose was again averaged from 20–30 values logged during 4 weeks, similarly to pretest. Finally, two follow-up sessions, each 1 month apart, were attended by the BFRT patients. During each of those sessions, there was a brief discussion of patients' current management of diabetes and stress. A BFRT session was offered. The psychophysio-logical measurements were recorded as follow-up data at the second booster session or 3 months after completion of BFRT. Since the control group patients were offered BFRT after their participation as controls (4 weeks after the last education session), they were not available for follow-up.

Of the initial 39 participants, three men and six women (seven Caucasians, two African Americans) dropped out before completing all requirements of the study. Table 1 shows the characteristics of dropouts and the patients who completed the protocol. Seven patients discontinued participation after the interview and before randomization, and two dropped out during the first 2 weeks of BFRT. Dropouts most frequently expressed concerns about the time commitment necessary for participation. Of the nine dropouts, seven were considered depressed because they scored ≥11 on the BDI-II. Three were interviewed by the psychiatrist; four dropped out before this evaluation. Of the three who were interviewed, one was prescribed 50 mg sertraline, and two chose to increase dosage of current antidepressant medication through their physicians. Completers included 30 patients: 7 men and 23 women (28 Caucasian, 1 Hispanic, and 1 "Other.") Twelve of the completers scored ≥11 on the BDI-II at pretest. Three of the twelve were prescribed 50 mg sertraline, whereas the others were stabilized on ongoing selective serotonin reuptake inhibitor antidepressants through their physicians.

Pretest, posttest, and follow-up data were analyzed using SPSS version 10.5. ANOVA with repeated measures, Pearson correlations, and *t* tests were performed.

RESULTS — Table 2 illustrates the average blood glucose values in the BFRT and control groups at pretest, posttest, and follow-up (no follow-up for control subjects). In the BFRT group, average

Table 2—*Self-reported average blood glucose and A1C in treatment and control groups*

	Blood glucose (mmol/l)*†			A1C (%)†‡		
	Pretest	Posttest	Follow-up	Pretest	Posttest	Follow-up
Treatment (BFRT)	8.3 ± 2.0	7.5 ± 1.4	7.4 ± 1.3	7.4 ± 1.4	6.8 ± 1.0	6.6 ± 2.1
Control (education)	8.3 ± 2.3	8.5 ± 2.8	—	7.0 ± 1.4	7.2 ± 1.4	—

Data are means ± SD. *Significant difference between groups pretest compared with posttest, $P < 0.05$; †significant difference between follow-up and pretest; ‡significant difference between groups pretest compared with posttest, $P < 0.01$.

Table 3—*Physiological and psychological factors in the treatment and control groups at pre- and posttest*

	n	Muscle tension (μvolts)		Finger temperature (F°)		Depression Score		Trait anxiety*	
		Pretest	Posttest	Pretest	Posttest	Pretest	Posttest	Pretest	Posttest
Treatment (BFRT)	16	3.7 ± 2.9	2.2 ± 1.0	89.8 ± 5.0	90.9 ± 5.2	11.7 ± 7.7	5.4 ± 6.0	39.9 ± 13.7	34.6 ± 12.6
Control (education)	14	2.3 ± 1.2	1.9 ± 0.7	91.9 ± 2.6	92.5 ± 3.3	13.6 ± 10.9	8.4 ± 10.3	43.9 ± 13.3	42.5 ± 12.2

Data are means ± SD. Significant differences between groups, $P < 0.05$. *Significant differences within subjects, $P < 0.01$.

blood glucose values decreased from pretest 8.3 ± 2.0 to posttest 7.5 ± 1.4 mmol/l (± SD). The mean blood glucose values in the education group increased slightly from pretest 8.3 ± 2.3 to posttest 8.5 ± 2.8 mmol/l. ANOVA with repeated measures showed a significant interaction between group and pretest/posttest: [$F(1,28) = 7.3; P = 0.012$]. Post hoc t tests showed that only the BFRT group sustained significant decreases ($P = 0.001$) in blood glucose. After 3 months, the BFRT group continued to demonstrate significantly lower blood glucose values compared with pretest ($P = 0.001$). There was no significant effect of sex on pre- or posttest blood glucose.

Table 2 also shows the hemoglobin values for both groups at pre- and posttest. The A1C levels in the BFRT group decreased from pretest 7.4 ± 1.5 to posttest 6.8 ± 1.0%. The average A1C levels in the education group increased slightly: pretest 7.0 ± 1.4 and posttest 7.2 ± 1.4. A significant difference was observed between the groups [$F(1,27) = 12.3, P = 0.002$]. Post hoc t tests showed that only the BFRT group sustained significant decreases in A1C ($P = 0.01$). Three months after BFRT, A1C values remained significantly different from pretest ($P = 0.001$).

Table 3 summarizes data for muscle tension, finger temperature, anxiety, and depression. There was a significant interaction between group and time [$F(1,28) = 4.7, P = 0.039$] in tension. Only the BFRT group decreased muscle tension from pre- to posttest ($P = 0.006$). There was no significant interaction or main effect for temperature. With regard to the psychological measures, Table 3 also shows a significant main effect within groups in BDI-II scores ($P = 0.0001$) and Spielberger State-Trait Anxiety Inventory ($P = 0.005$). Both groups decreased depression scores, but only the BFRT group significantly decreased trait anxiety ($t = 0.037$).

Then, the relationship between blood glucose, depression, and anxiety in those patients who completed the protocol was explored. Pretest scores on the BDI-II were significantly correlated with pretest levels ($r = 0.44, P = 0.015$) and posttest A1C levels ($r = 0.53, P = 0.003$). Pretest scores on the anxiety inventory were correlated with posttest A1C ($r = 0.40, P = 0.031$). Two-way ANOVA with repeated measures did not show a significant interaction between group, depression code (pretest BDI-II score ≥11 or <11), and pretest/posttest blood glucose. The significant interaction between group and time was confirmed.

Finally, completers and dropouts were compared on blood glucose, depression, and anxiety scores. Blood glucose and BDI-II scores were higher in the dropouts compared with those patients who completed either BFRT or education [$F(1,33) = 7.1, P = 0.012; F(1,36) = 6.8, P = 0.013$], respectively. There were no significant differences between groups in age or pretest anxiety scores.

CONCLUSIONS — This study indicated that patients with type 2 diabetes could significantly improve glycemic control through the use of BFRT. The significant decrease in A1C observed in this short-term, controlled study supports the 1-year findings of Surwit et al. (6). However, there are differences between the designs of the two studies. Our protocol offered biofeedback in addition to relaxation in 10 weekly individual sessions in contrast to group stress management sessions held during 1 year. It seems that the intensive, one-to-one techniques used in BFRT may have led to early benefits for the patients. Although education is recognized as a mainstay of diabetes treatment, education control groups in both studies experienced a worsening of their A1C levels over time. This can be related to the long-term nature of our patients' diabetes, their previous exposure to diabetic education, and the passive nature of the edu-

cation process. Reeducating these patients regarding illness management was not associated with improvements in diabetes control. This type of education acted as a true control with patients showing some deterioration over time.

BFRT fosters both physiological and psychological changes, which may explain the process by which it assisted patients in the BFRT group to lower blood glucose and A1C compared with the education control group. Research on the physiological effects of BFRT suggests that it decreases indicators of chronic stress responding such as muscle tension, peripheral vasoconstriction, anxiety, heart rate, skin conductance, cortisol, and catecholamines (4). In previous studies, increased skin temperature and decreased anxiety mediated the response to BFRT in type 1 and type 2 diabetic patients (7,16). Although our current patients were unable to demonstrate significant skin temperature changes when pretest and posttest were compared, they did show increases in temperature during the individual thermal biofeedback sessions. It is not clear why these patients preferentially lowered muscle tension without a significant decrease in adrenergically mediated vasoconstriction.

During the stress response, activation of the sympathetic nervous system also increases cortisol release and insulin resistance (17). Modulation of the sympathetic nervous system and subsequent reduction of plasma cortisol levels following BFRT would lead to improved glucose control. Although measurements of these variables were not performed in this study, effects of relaxation on blood glucose and plasma cortisol have been shown in other studies (18). Specific assessment of sympathetic nervous system activity and cortisol levels would be useful in determining the direct physiologic effects of BFRT in patients with type 2 diabetes.

The psychological effects of BFRT may have also improved blood glucose

levels and A1C. In contrast to the more passive aspects of medical management, the experience of BFRT fosters a sense of control over physiological responses to stress. This view is supported by the significant decreases in anxiety and depression scores in the BFRT group. Positive reinforcement from the instrument provides "evidence" of improvement, such as lower facial tension, which the patients are encouraged to apply to other relevant physiological functions. The psychological effects of BFRT often include learning to set realistic goals, an improved sense of control, and better problem solving. These strategies, although not formally tested as outcome variables in this study are similar to the empowerment model described by Anderson et al. (19). In that controlled trial, patients with type 2 diabetes increased self-efficacy and A1C after six 2-h sessions of patient education centered on problem solving, coping, stress management, and self motivation.

Patients with diabetes and psychiatric illness have a poorer overall prognosis and more diabetes-related complications compared with patients who are never emotionally ill. In this investigation, pretest depressive symptoms were correlated with pre- and posttest A1C levels. However, Miyaoka et al. (20) found that depressive state did not correlate with serum level of A1C, perhaps because of varying psychopharmacologic therapy offered to patients. Depressed patients exemplify lack of motivation and hopelessness about the future in addition to physical manifestations of fatigue, insomnia, and disruptions in appetite. When depressed patients are confronted with requirements to measure blood glucose, take multiple medicines, participate in regular exercise, and attend to food intake, they often feel overwhelmed, anxious, and more depressed. Lustman et al. (21) showed that depressed patients with fewer symptoms of depression after treatment with fluoxetine also improved blood glucose. But the 50-mg dosage of sertraline was not sufficient to significantly improve depression scores in some of our more depressed study patients. Most depressed patients dropped out after the initial interview and before randomization, supporting the contention that the depressed patients could not make the commitment to the demands of the research protocol.

There are a number of limitations to this study. The BFRT intervention was more intensive than the education arm of the study and required more effort on the part of the patients. The total number of weeks for the BFRT group was 10 weeks of active treatment, 4 weeks posttest, and an additional 2 months of follow-up, whereas the education sessions were spread over 9–12 weeks. The lack of long-term follow-up information in the BFRT group and lack of any follow-up data for the control subjects limits our ability to predict ongoing effectiveness of the BFRT treatment. The study is also limited by the relatively small sample size and the low minority representation. Patients were all volunteers, leading to a potential bias in the sample. There were unequal numbers of depressed and nondepressed patients, and a relatively small number of depressed patients completed the protocol. In addition, the depressed patients were not treated to remission, which further limits interpretation of the results comparing outcome in depressed and nondepressed patients. The predominant evidence for mediation of improved blood glucose and A1C in the BFRT group was decreased muscle tension and less anxiety, but we cannot address the issue of the timing of improvement in glycemic control.

In summary, BFRT may be a useful adjunct to standard medical care of type 2 diabetes. For optimal long-term improvement in A1C, individual and group sessions could be combined and supplemented by biofeedback. Physicians and other health care providers caring for patients with diabetes should consider screening for depression and anxiety and instituting psychopharmacological or psychotherapeutic management, when appropriate. With major increases in both type 2 diabetes (22) and mood disorders (23) predicted in the general population, it is important to develop therapies that address the treatment of both of these conditions.

References
1. American Diabetes Association: Standards of medical care in diabetes (Position Statement). *Diabetes Care* 27 (Suppl. 1):S15–S35, 2004
2. Rosmond R: Role of stress in the pathogenesis of the metabolic syndrome. *Psychoneuroendocrinology* 30:1–10, 2005
3. Surwit RS, Schneider MS, Feinglos MN: Stress and diabetes mellitus. *Diabetes Care* 15:1413–1421, 1992
4. Schwartz NM, Schwartz MS: Definitions of biofeedback and applied psychophysiology. In *Biofeedback: A Practitioner's Guide.* 3rd ed. Schwartz MS, Andrasik F, Eds. New York, Guilford Press, 2003, p. 27–42
5. Jablon SL, Naliboff BD, Gilmore SL: Effects of relaxation training in glucose tolerance and diabetic control in type II diabetes. *Appl Psychophysiol Biofeedback* 22:155–169, 1997
6. Surwit RS, van Tilburg MAL, Zucker N, McCaskill CC, Parekh P, Fienglos MN, Edwards CL, Williams P, Lane JD: Stress management improves long-term glycemic control in type 2 diabetes. *Diabetes Care* 25:30–34, 2002
7. McGrady A, Horner J: Role of mood in outcome of biofeedback assisted relaxation therapy in insulin dependent diabetes mellitus. *Appl Psychophysiol Biofeedback* 24:79–88, 1999
8. Talbot F, Nouwen A: A review of the relationship between depression and diabetes in adults. *Diabetes Care* 23:1556–1562, 2000
9. Anderson RJ, Freedland KJ, Clouse RE, Lustman PJ: The prevalence of comorbid depression in adults with diabetes. *Diabetes Care* 24:1069–1078, 2001
10. Lustman PJ, Griffith LS, Clouse RE, Cryer PE: Psychiatric illness in diabetes mellitus: relationship to symptoms and glucose control. *J Nerv Ment Dis* 174:736–742, 1986
11. deGroot M, Jacobson AM, Samson JA, Welch G: Glycemic control and major depression in patients with type 1 and type 2 diabetes care. *J Psychosom Res* 46:425–435, 1999
12. Goodnick PJ, Henry JH, Bukik VM: Treatment of depression in patients with diabetes mellitus. *J Clin Psych* 56:126–136, 1995
13. Beck AT: *Beck Depression Inventory.* San Antonio, Texas, Harcourt Brace & Company, 1978
14. Spielberger CD, Gorsuch RL, Lushene RE: *STAI Manual.* Palo Alto, CA, Consulting Psychologists Press, 1968
15. Davis M, Robbins Eshelman E, McKay M: *The Relaxation and Stress Reduction Workbook.* Oakland, CA, New Harbinger Publications, 1995

16. McGrady A, Graham G, Bailey B: Biofeedback-assisted relaxation in insulin-dependent diabetes: a replication and extension study. *Ann Behav Med* 18:185–189, 1996

17. Chrousos GP, Gold PW: A healthy body in a healthy mind and vice versa: the damaging power of uncontrollable stress. *J Clin Endocrinol Metab* 83:1842–1845, 1998

18. Surwit RS, Feinglos MN: Relaxation-induced improvement in glucose is associated with decreased plasma cortisol. *Diabetes Care* 7:203–204, 1984

19. Anderson RM, Funnell MM, Butler PM, Arnold MS, Fitzgerald JT, Feste CC: Patient empowerment: results of a randomized controlled trial. *Diabetes Care* 18:943–949, 1995

20. Miyaoka Y, Miyaoka H, Motomiya T, Kitamura S, Masahiro A: Impact of sociodemographic and diabetes-related characteristics on depressive state among non-insulin-dependent diabetic patients. *Psychiatry Clin Neurosci* 51:203–206, 1997

21. Lustman PJ, Freedland KE, Griffith LS, Clouse RE: Fluoxetine for depression in diabetes. *Diabetes Care* 23:618–623, 2000

22. Boyle JP, Honeycutt AA, Venkat Narayan KM, Hoerger TJ, Geiss LS, Chen H, Thompson TJ: Projection of diabetes burden through 2050: impact of changing demography and disease prevalence in the U.S. *Diabetes Care* 24:1936–1940, 2001

23. Michaud CM, Murray CJ, Bloom BR: Burden of disease-implications for future research. *JAMA* 285:535–539, 2001