Journal of Behavioral Medicine, Vol. 11, No. 3, 1988

The Effects of Blood Sugar Level Changes on Cognitive Function, Affective State, and Somatic Symptoms

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Accepted for publication: July 24, 1987

In an attempt to find out whether decreased blood sugar level is associated with impaired cognitive function, adverse emotional changes, or somatic symptoms, 35 subjects who suspected that they had hypoglycemia were given 5-hr glucose tolerance tests (GTTs). Nine blood samples were taken during the GTT, and the subject's mood, Serial Sevens Test (SST) performance, and somatic symptom reports were recorded on each occasion of sampling. The subjects reported significantly more negative affect and somatic symptoms after glucose nadir than before nadir. SST performance deteriorated at glucose nadir. These effects were more pronounced for subjects with high hypoglycemic index scores than for subjects with low index scores. The impairment in SST performance was greater for subjects who showed rapid decreases in blood sugar than for subjects who showed slow decreases. Dividing subjects by high and low nadirs did not reveal any differences in symptomatology.

KEY WORDS: blood sugar; hypoglycemia; somatic symptoms; affective state; cognitive function.

INTRODUCTION

The purpose of this study was to investigate the relationship between changes in blood sugar level and cognitive function, affective state, and so-

This research was conducted while the first author held a Medical Research Council Studentship; this assistance is gratefully acknowledged.

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0160-7715/88/0600-0279$06.00/0

matic symptoms. The secondary aim was to evaluate various methods of determining the presence or absence of reactive hypoglycemia.

Blood sugar levels increase after substantial amounts of glucose are ingested and then fall below fasting levels 2 to 5 hr later. Reactive hypoglycemia is an abnormal condition in which the person's blood sugar level rises after eating and then falls significantly lower than normal within 2 to 5 hr. This excessive overreaction is associated with transient symptoms such as sweating, palpitations, trembling, weakness, dizziness, nervousness, irritability, fatigue, hunger, and confusion, all of which have been noted to occur during this period of low blood sugar (e.g., Cataland, 1986; Hale *et al.*, 1982; Hare, 1986). However, there is no agreement on the parameters of this overreaction and no agreement on the diagnostic criteria for reactive hypoglycemia.

The most frequently used diagnostic criterion is a glucose nadir (i.e., the lowest level of blood sugar reached) that falls below a predetermined cutoff point; unfortunately there is no agreed cutoff point for this nadir. Values from 40 mg/dl (e.g., Hofeldt *et al.*, 1972; Young and Karam, 1983) to 70 mg/dl (e.g., Budd, 1981) have been used. A quick rate of fall in blood sugar has also been proposed as predictive of hypoglycemic symptoms (e.g., Budd, 1981; Cataland, 1986; Hamburger *et al.*, 1984), but Johnson *et al.* (1980) found that the symptoms were related to slower rather than faster rates of decline. Finally, Hadji-Georgopoulos *et al.* (1980) developed a hypoglycemic index that is calculated by dividing the decrease in blood sugar during the 90-min period before nadir by the absolute glucose nadir. Subjects with index values above .8 became symptomatic (Hadji-Georgopoulos *et al.*, 1980; Uhde *et al.*, 1984), but there is also some evidence that the hypoglycemic index does not differentiate symptomatic from asymptomatic subjects (Lev-Ran and Anderson, 1981). In light of this confusion and disagreement, there is an evident need for a reliable, discriminative, and valid diagnostic measure.

The prevalence of reactive hypoglycemia is also uncertain. The popular literature on low blood sugar contains claims that hypoglycemia is a disease of epidemic proportions (e.g., Brennan and Mulligan, 1975; Budd, 1981), and hypoglycemia has been said to cause many symptoms and disorders including depression, panic attacks, alcoholism, emotional outbursts, nightmares, hyperactivity, aggression, and schizophrenia. There is little basis for either of these claims, but there is some acceptable evidence of a relationship between lowered blood sugar levels and symptoms. Low blood sugar has been related to impaired performance on the Serial Sevens Test (Hale *et al.*, 1982); increased reaction time and decreased number of additions calculated (Holmes *et al.*, 1983); impaired performance on a pegboard task, fewer words recalled, and increased time to solve problems (Russell and

Rix-Trott, 1975); and somatic symptoms (Pennebaker *et al.*, 1981; Schweizer *et al.*, 1986; Uhde *et al.*, 1984). On the other hand, it has been reported that hypoglycemia is very rare and that many people experience low blood sugar levels without symptoms (e.g., Anderson and Lev-Ran, 1985; Hofeldt, 1975; Johnson *et al.*, 1980; Lev-Ran and Anderson, 1981).

The controversy over whether or not lowered blood sugar levels produce symptoms and the absence of satisfactory diagnostic criteria for hypoglycemia prompted the present study. The primary hypothesis was that lowered blood sugar levels are associated with impaired cognitive performance, adverse emotional changes, and somatic symptoms. The subsidiary hypotheses were that the cognitive impairment, adverse emotional changes, and somatic symptoms are greater (a) the lower the glucose nadir, (b) the more rapid the decrease in blood sugar, and (c) the higher the hypoglycemic index score (Hadji-Georgopoulos *et al.*, 1980).

METHOD

In order to test the hypotheses, the following data were needed: blood sugar levels, cognitive performance, mood state, and somatic symptoms presence. Blood sugar level was manipulated with the procedures of the 5-hr glucose tolerance test (GTT), the standard test for assessing hypoglycemia. Cognitive performance was assessed by performance on the Serial Sevens Test (SST; Hayman, 1942; Ruesch, 1942), mood state by scores on the Profile of Mood States (POMS; McNair *et al.*, 1971), and somatic symptoms by scores on the Physical Symptom Scale (PSS).

Subjects

Thirty-five subjects (25 female and 10 male) who believe or suspected that they had hypoglycemia participated in the experiment. They were recruited by notices posted in the community. Subjects were between 21 and 66 years of age (M = 32.4 years) and had between 12 and 21 years of education (M = 15.5 years). Informed consent was obtained in writing from each subject.

Blood Sugar Manipulation

It was of primary interest to observe subjects after their blood sugar level had been lowered. To accomplish this, GTTs were conducted at a local hospital. Subjects fasted for 12 hr overnight before arriving at the hospital.

During the GTT, subjects had their blood sugar level measured in the fasting state and then ingested a 75-g glucose solution (Glucodex). Blood sugar level was then measured at regular intervals over a 5-hr period. The usual response to this manipulation is a peak in blood sugar within the first 45 min, followed by a fall below fasting blood sugar levels after 2 to 5 hr. Blood sugar then returns to fasting level. The blood sugar samples were taken via finger pricks (i.e., whole blood) and analyzed in a YSI 23A Glucose Analyzer (Yellow Springs Instrument Company, Yellow Springs, Ohio) which provides a digital display of blood sugar level as milligrams per deciliter.

In order to test the hypotheses, the following values were calculated for each subject:

(1) glucose nadir (LOWPOINT)—the lowest level of blood sugar achieved after the ingestion of glucose;
(2) speed of fall in blood sugar (SPEED)—the drop in blood sugar between peak and nadir, divided by the time between peak and nadir, yielding a score as milligrams per hour; and
(3) hypoglycemic index score (INDEX)—the decrease in blood sugar during the 90 min preceding nadir, divided by the nadir.

Dependent Measures

Serial Sevens Test. The SST is a task in which subjects are given a number and instructed to count backward by sevens from it (orally) until told to stop. The test is scored as the time taken to complete 14 subtractions (SST-Time). Hayman (1942) reports that factors interfering with mental efficiency are reflected by increased time taken to complete the SST.

In the present study, different starting numbers were used on each trial to prevent subjects from memorizing their answers. The order of presentation of the numbers was randomized. A preliminary study had indicated that subjects improve their performance with repeated trials on the SST but that this practice effect ceases to produce significant differences between scores after two trials. Therefore, subjects were given two pretrials on the SST before commencing the experiment, in order to attenuate the effect of improvement with practice.

Profile of Mood States. The POMS (McNair *et al.*, 1971) is a self-report instrument consisting of 65 words or phrases describing common feelings. Subjects indicate how they "are feeling right now" by placing a mark on a 5-point scale. The POMS is intended to assess transient fluctuating affective states. It yields six scores: anxiety, depression, anger, vigor, fatigue, and confusion. The sum of these scale scores (with vigor negatively weighted) provides a total mood disturbance score (POMS-Total).

Physical Symptom Scale. This scale was developed in the study for the purpose of measuring somatic symptoms that are said to occur during hypoglycemia. The scale consists of the following symptoms: trembling, pounding heart, flushed face, lightheadedness, sweating, hunger, weakness, headache, blurred vision, dizziness, and nausea. These 11 descriptors were selected on the basis of a review of the literature. Subjects place a mark on the dashed line to indicate how they are feeling "right now." The dashes were scored from 0 to 9, with the far-left dash being labeled "not at all" and the far-right dash being labeled "a great deal" or "extremely." The items were summed to provide a total symptom score (PSS-Total).

Procedure

Demographic data were collected from each subject and the two pretrials of the SST were administered. Subjects then had their first blood sugar sample taken and completed the first set of tests. Next, they ingested the glucose solution. Subsequently, blood sugar was sampled and the tests were given at 45 min and 2, 2.5, 3, 3.5, 4, 4.5, and 5 hr post-glucose ingestion. Each time, the order of measures was blood sugar sample, PSS, SST, POMS; this battery of tests took between 5 and 10 min to complete.

Between testing periods, subjects sat, read, and/or went for short strolls. Eating and drinking (except water) were not allowed. Subjects and the experimenter were blind with respect to blood sugar levels throughout the study but were aware that a GTT was being conducted and that symptoms might occur.

RESULTS

A repeated-measures design, across nine time points, was used. The point of most interest was that at which subjects' blood sugar was lowest (glucose nadir). Because nadir occurred at different times for different subjects, the following five measurement points were used in the analyses: 1 hr before nadir (ONEBEF), 0.5 hr before nadir (HALFBEF), nadir (NADIR), 0.5 hr after nadir (HALFAFT), and 1 hr after nadir (ONEAFT). In order to differentiate the effects of blood sugar changes from linear effects of order or practice, it was necessary to record the symptoms before and after nadir. The time of nadir was distributed as follows: four subjects at 2.5 hr, nine subjects at 3 hr, seven subjects at 3.5 hr, eight subjects at 4 hr, one subject at 4.5 hr, and six subjects at 5 hr. Subjects whose nadirs occurred at 4.5

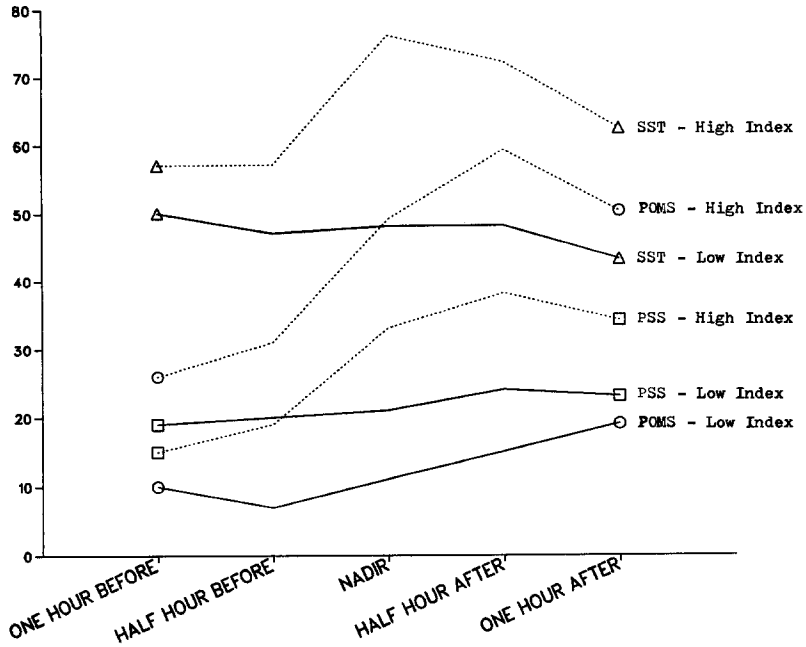

Fig. 1. Symptom changes as a function of time from glucose nadir for subjects with high versus low hypoglycemic index scores. SST, Serial Sevens Test; POMS, Profile of Mood States; PSS, Physical Symptom Scale.

or 5 hr could not be used in the analyses, as HALFAFT and ONEAFT could not be measured.

The typical response to the GTT was not shown by all of the subjects. Two subjects had flat curves, five had M-shaped curves, and two had their peak blood sugar level at 2.5 hr (versus the usual 45 min). It was decided to exclude these nine subjects from the analyses, as the manipulation had not produced the expected physiological changes, and therefore, it was unclear whether or when symptoms could be expected to occur. Also, it was not possible to calculate the diagnostic criterion variables (i.e., LOWPOINT, SPEED, INDEX). Thus, the analyses were based on the 22 subjects for whom the manipulation was successful and for whom there were data available for all five measurement points.

Primary Hypothesis

To test the hypothesis that lowered blood sugar levels are associated with symptoms, a repeated-measures MANOVA was conducted on the three dependent measures across the five measurement points. The MANOVA was

Table I. Means (Standard Deviations) for POMS-Total, PSS-Total, and SST-Time Across Five Measurement Points[a]

Measure	ONEBEF	HALFBEF	NADIR	HALFAFT	ONEAFT
POMS-Total	16.27	18.55	29.55	34.27	31.23
	(36.17)	(33.21)	(33.88)	(39.64)	(37.05)
PSS-Total	16.46	19.18	26.59	29.59	27.45
	(18.00)	(17.90)	(19.73)	(22.77)	(21.24)
SST-Time	52.86	51.50	60.27	57.96	52.09
	(16.56)	(17.33)	(24.95)	(25.57)	(20.39)

[a]Figures based on $N = 21$.

significant [$F(16, 235.88) = 4.39$, $p < .0001$], and therefore, repeated-measures ANOVAs were conducted (with Bonferroni-corrected probability levels of .0125). The means for the three dependent variables are presented in Table I. The effect for POMS-Total was significant [$F(4,80) = 7.85$, $p < .0001$], with subjects reporting significantly more mood disturbance at HALFAFT than at ONEBEF or HALFBEF. (All multiple comparisons were conducted via the Tukey HSD method, .01 level of significance). The PSS-Total effect was also significant [$F(4,80) = 9.03$, $p < .0001$]. Subjects reported significantly more somatic symptoms at NADIR, HALFAFT, and ONEAFT than at ONEBEF; only symptoms at HALFAFT were significantly greater than at HALFBEF. Thus, for both POMS-Total and PSS-Total, scores were highest half an hour after nadir.

The ANOVA for SST-Time was significant [$F(4,80) = 3.42$, $p < .0125$], but none of the multiple comparisons reached significance. However, the pattern of results is clearly different from the usual pattern of steady improvement in SST performance across trials.

In order to get a preliminary idea of which symptoms were the most affected by the GTT, repeated-measures MANOVAs were conducted on the 6 POMS scales and 11 PSS items. The MANOVA on the POMS was significant [$F(24,276.81) = 1.88$, $p < .01$]. Subsequent ANOVAs (corrected probability, .0083) were significant for anxiety [$F(4,84) = 8.98$, $p < .0001$] and confusion [$F(4,84) = 4.93$, $p < .002$]. The MANOVA on the PSS was also significant [$F(44,285.06) = 2.35$, $p < .0001$]. The ANOVAs, with a corrected probability level of .0045, showed significant effects for trembling [$F(4,84) = 13.94$, $p < .0001$], pounding heart [$F(4,84) = 4.19$, $p < .004$], hungry [$F(4,84) = 22.01$, $p < .0001$], and weak [$F(4,84) = 5.82$, $p < .0004$].

Subsidiary Hypotheses

In order to test the subsidiary hypotheses, subjects were divided into high- and low-scoring groups for each of the diagnostic criterion variables (i.e., LOWPOINT, SPEED, INDEX). The 11 subjects with the highest scores

Table II. High- Versus Low-LOWPOINT Group Means (Standard Deviations)[a]

Variables	LOWPOINT group	ONEBEF	HALFBEF	NADIR	HALFAFT	ONEAFT
POMS-Total	High	19.55	16.27	26.73	33.36	31.55
		(44.01)	(37.78)	(38.61)	(40.35)	(39.92)
	Low	15.50	20.40	31.60	38.80	35.50
		(28.26)	(31.18)	(31.55)	(41.18)	(34.28)
PSS-Total	High	17.27	19.09	25.82	28.18	26.36
		(24.10)	(22.70)	(23.74)	(24.53)	(22.19)
	Low	16.80	20.10	28.00	32.90	30.30
		(9.60)	(12.92)	(16.59)	(22.18)	(21.56)
SST-Time	High	57.09	55.09	59.09	59.82	52.09
		(13.01)	(10.45)	(19.97)	(19.34)	(9.95)
	Low	49.40	48.80	63.50	58.50	52.30
		(20.02)	(23.20)	(30.89)	(32.09)	(29.31)

[a]Figures based on $N = 21$; 11 subjects with high nadirs and 10 subjects with low nadirs. LOWPOINT = glucose nadir (lowest level of blood sugar reached).

constituted one group, and the 11 subjects with the lowest scores comprised another group. Subjects in the high-LOWPOINT group had nadirs ranging from 61 to 75 mg/dl ($M = 65$ mg/dl), while those in the low-LOWPOINT group had nadirs ranging from 48 to 60 mg/dl ($M = 53$ mg/dl). High-SPEED subjects' rates of drops in blood sugar were between 35.6 and 73.1 mg/hr ($M = 48.7$ mg/hr); low-SPEED subjects' rates were between 21.2 and 34.7 mg/hr ($M = 30.3$ mg/hr). Finally, subjects in the high-INDEX group had index scores between 1.14 and 2.10 ($M = 1.44$), and subjects in the low-INDEX group had scores between 0.61 and 1.06 ($M = 0.84$).

For the subsidiary hypotheses, 2 × 5 between–within MANOVAs were conducted in which the high- and low-diagnostic criterion variable groups formed the two levels of the between-group factor, and the measurement points (HALFBEF, ONEBEF, NADIR, HALFAFT, ONEAFT) formed the five levels of the within-group factor. The interaction between group membership and measurement point was of interest.

For the analysis of LOWPOINT, this interaction was not significant [$F(16,223.66) = 0.71$, $p < .7846$]. The means are shown in Table II.

The interaction was significant for SPEED [$F(16,223.66) = 1.78$, $p < .05$]. Subsequent ANOVAs were significant for SST-Time [$F(4,76) = 5.79$, $p < .0005$] but not for POMS-Total [$F(4,76) = 2.35$, $p < .0617$] or PSS-Total [$F(4,76) = 2.20$, $p < .0772$]. Means for these effects are presented in Table III. Multiple comparisons for SST-Time indicated that high-SPEED subjects took longer to do the SST at NADIR and HALFAFT than at ONEBEF and HALFBEF. Performance was also poorer at NADIR than at ONEAFT. No comparisons were significant for the low-SPEED group.

The interaction between group and measurement point was also significant for INDEX [$F(16,223.66) = 2.29$, $p < .005$]. ANOVAs were sig-

Table III. High- Versus Low-SPEED Group Means (Standard Deviations)[a]

Variables	SPEED group	ONEBEF	HALFBEF	NADIR	HALFAFT	ONEAFT
POMS-Total	High	20.80	24.10	42.20	50.30	42.20
		(25.36)	(26.34)	(34.10)	(35.88)	(30.33)
	Low	14.73	12.91	17.09	22.91	25.45
		(45.46)	(40.27)	(31.97)	(40.23)	(41.04)
PSS-Total	High	10.70	14.30	27.40	29.60	25.90
		(7.24)	(9.06)	(21.02)	(18.52)	(15.02)
	Low	22.82	24.36	26.36	31.18	30.36
		(23.23)	(23.22)	(20.38)	(27.32)	(26.56)
SST-Time	High	53.60	54.50	74.00	68.60	56.00
		(10.71)	(8.84)	(20.70)	(19.08)	(9.45)
	Low	53.27	49.91	49.55	50.64	48.73
		(21.40)	(23.09)	(23.86)	(28.38)	(27.64)

[a]Figures based on $N = 21$; 10 subjects with high speed of drop in blood sugar and 11 subjects with low speed of drop in blood sugar. SPEED = rate of decrease in blood sugar.

nificant for POMS-Total [$F(4,76) = 4.14$, $p < .005$], PSS-Total [$F(4,76) = 5.40$, $p < .0008$], and SST-Time [$F(4,76) = 4.97$, $p < .002$]. The means for these effects are presented in Table IV. Multiple comparisons showed that POMS-Total scores were significantly higher at NADIR, HALFAFT, and ONEAFT than at ONEBEF for the high-INDEX group. Similarly, for this group, subjects reported more mood disturbance at HALFAFT and ONEAFT than at HALFBEF. No comparisons were significant for the low-INDEX group. On the PSS-Total, high-INDEX subjects reported significantly more symptoms at NADIR, HALFAFT, and ONEAFT than at ONEBEF and HALFBEF. Again, no comparisons were significant for the low-INDEX group. Finally, high-INDEX subjects' performance on the SST was significantly poorer at NADIR and HALFAFT than at ONEBEF and HALFBEF. There were no significant comparisons for the low-INDEX group.

DISCUSSION

The first hypothesis—that lowered blood sugar levels are associated with impaired cognitive performance, adverse emotional changes, and somatic symptoms—was supported. Subjects reported greater mood disturbance and more bodily symptoms half an hour after glucose nadir than an hour or half an hour before nadir. It is of interest to note that these symptoms were highest not at nadir, but half an hour after nadir. Some of the negative findings in the literature may have arisen because symptoms were sought only at nadir.

The subsidiary hypotheses received varying amounts of support. No support was obtained for the hypothesis that symptoms are greater at lower

Table IV. High- Versus Low-INDEX Group Means (Standard Deviations)[a]

Variables	INDEX group	ONEBEF	HALFBEF	NADIR	HALFAFT	ONEAFT
POMS-Total	High	25.90	30.50	49.10	59.40	49.80
		(19.27)	(19.35)	(25.27)	(27.62)	(25.11)
	Low	10.09	7.09	10.82	14.64	18.55
		(46.89)	(41.08)	(32.51)	(37.87)	(39.75)
PSS-Total	High	14.90	19.40	33.20	37.60	34.10
		(8.80)	(11.89)	(18.88)	(20.35)	(19.23)
	Low	19.00	19.73	21.09	23.91	22.91
		(24.20)	(23.21)	(20.40)	(24.19)	(22.81)
SST-Time	High	56.70	57.40	76.10	72.00	62.40
		(18.94)	(20.10)	(27.43)	(27.94)	(24.75)
	Low	50.45	47.27	47.64	47.55	42.91
		(14.76)	(14.04)	(12.90)	(16.92)	(11.05)

[a]Figures based on $N = 21$; 10 subjects with high hypoglycemic index scores and 11 subjects with low hypoglycemic index scores. INDEX = hypoglycemic index score.

glucose nadirs. This negative finding is of potential significance because cutoff values of glucose nadir are the most frequently advocated diagnostic criterion for reactive hypoglycemia (e.g., Benson and Fredlund, 1985; Young and Karam, 1983). It remains possible that below a certain level of blood sugar (one lower than that reached by the low-nadir subjects), all subjects experience symptoms. However, even if this is found to be true, a different diagnostic criterion will be needed for those subjects with higher nadirs. The present findings indicate that defining low blood sugar levels solely on the basis of nadir values is not advisable. Other aspects of the blood sugar response appear to be better predictors of subjects reporting symptoms at times of lowered blood sugar.

One such aspect is the rate of the fall in blood sugar. Some support was obtained for the hypothesis that symptoms would be greater with more rapid decreases in blood sugar. The strongest support was found for the hypothesis that symptoms are greater with higher hypoglycemic index scores. The hypoglycemic index takes into account the level of nadir, the amount of decrease in blood sugar, and the speed of the drop in blood sugar. Its success at taking these variables into account is corroborated by correlations between the index and the nadir ($r = -.399$, $p < .05$) and the index and speed ($r = .768$, $p < .0001$).

The results of the present study are relevant to the debate between those who claim that hypoglycemia causes many psychological changes (e.g., Brennan and Mulligan, 1975; Budd, 1981) and those who claim that hypoglycemia is a spurious explanation for psychologically caused problems (e.g., Cahill and Soeldner, 1974; Yager and Young, 1974). The present results suggest that the claims of both of these groups may be exaggerated.

The symptoms claimed to be attributable to hypoglycemia in some cases—such as family violence, hyperactivity, and schizophrenia (Brennan and Mulligan, 1975)—are unlikely to be caused by low blood sugar. Subjects in the present study reported feeling anxious or confused, but no bizarre behavior, thoughts, or feelings were observed or reported. Similarly, the proportion of the normal population who is said to experience significant symptoms from lowered blood sugar levels may well be exaggerated. In the present study, only half of the subjects, all of whom believed or suspected that they have hypoglycemia, experienced an increase in symptoms at or after nadir.

The findings are congruent with the evidence of a relationship between low blood sugar levels and symptoms. Uhde *et al.* (1984) found that nine patients with panic disorder experienced either an increase in anxiety or two or more somatic symptoms (i.e., hunger, light-headedness, sweating, or palpitations) 3 to 5 hr after glucose ingestion. These four somatic symptoms, plus trembling, pounding heart, and sleepiness, were found by Pennebaker *et al.* (1981) to be associated with low blood sugar levels in diabetic patients. Hale *et al.* (1982) found that subjects with low nadirs experienced greater regressions in SST performance at nadir than did subjects with high nadirs. Similarly, lowered blood sugar levels have been associated with slower performance on tasks (Holmes *et al.*, 1983; Russell and Rix-Trott, 1975). That such diverse methods (GTTs, daily symptom–glucose correlations, insulin challenge, and artificial insulin/glucose infusion) yield similar results adds support to the hypothesis that lowered blood sugar levels are associated with impaired cognitive performance, adverse emotional changes, and somatic symptoms.

As described under Results, 9 of the 35 subjects exhibited atypical responses to the GTT. Flat curves have been reported to occur (e.g., Budd, 1981), and Burns *et al.* (1965) found that—with continuous blood sugar sampling—two to four peaks and falls in blood sugar level may be observed. On the basis of the literature, however, the number of atypical curves observed was completely unexpected. Research is needed to determine whether, or when, atypical blood sugar responses are associated with cognitive, somatic, or affective impairments.

The main weakness in the present study is the lack of a placebo control group to rule out order or practice effects and subject expectancy as alternative explanations of the results. Several arguments render these alternative explanations implausible. Subjects' nadirs occurred at different times; therefore, nadir was not completely confounded with time of testing. Also, the effects observed differed depending on whether the subjects had high or low index scores. The subjects with low hypoglycemic index scores can be regarded as a post hoc control group which provides an indication of the changes that might occur even in the absence of an abnormal response to glucose ingestion.

In future research in this area, a randomized double-blind experiment with glucose and placebo tolerance tests would be informative, as would a test–retest evaluation of the present findings. Also, the diabetes literature indicates that it may be important to do within-subject analyses because of individual differences in symptom–blood glucose relationships (e.g., Cox *et al.*, 1983; Pennebaker *et al.*, 1981).

If progress is made in this research, it should be possible to move on to practical applications, including evaluations of the effects of various types of food on blood sugar level and symptom reactions. This would lay the basis for an empirical approach to the dietary treatment of hypoglycemia. In the meantime, clinicians can assist their hypoglycemic patients in developing individually tailored self-management programs, consistent with the practice of modern behavioral medicine. People experiencing symptoms from lowered blood sugar levels can use glucometers (individually operated portable glucose analysis instruments) to observe their symptom responses to changes in blood sugar level. They can learn which foods cause symptoms to arise, the time interval between eating and symptom onset, and which symptoms can be attributed to low blood sugar levels. They can then develop self-control of their symptoms by systematically observing the effects of diet on their symptoms.

ACKNOWLEDGMENTS

The authors wish to thank Dr. K. Dawson, Dr. D. Lawson, and Ms. Kusum Menon for their help with this research. Thanks are also given to the anonymous reviewers of this article.

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