CPAP Therapy of Obstructive Sleep Apnea in Type 2 Diabetics Improves Glycemic Control During Sleep

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Background: Type 2 diabetes and obstructive sleep apnea (OSA) are frequently comorbid conditions. OSA is associated with increased insulin resistance, but studies of continuous positive airway pressure (CPAP) have shown inconsistent effects on glycemic control. However, endpoints such as hemoglobin A1c and insulin sensitivity might not reflect short-term changes in glycemic control during sleep.

Methods: We used a continuous glucose-monitoring system to measure interstitial glucose every 5 minutes during polysomnography in 20 patients with type 2 diabetes and newly diagnosed OSA. The measurements were repeated after an average of 41 days of CPAP (range 26-96 days). All patients were on a stable diet and medications. Each 30-second epoch of the polysomnogram was matched with a continuous glucose-monitoring system reading, and the sleeping glucose level was calculated as the average for all epochs scored as sleeping.

Results: The mean sleeping glucose decreased from untreated (122.0 ± 61.7 mg/dL) to treated (102.9 ± 39.4 mg/dL; p = 0.03 by Wilcoxon paired rank test). The sleeping glucose was more stable after treatment, with the median SD decreasing from 20.0 to 13.0 mg/dL (p = 0.005) and the mean difference between maximum and minimum values decreasing from 88 to 57 mg/dL (p = 0.003). The change in the mean hemoglobin A1c from 7.1% to 7.2% was not significant.

Conclusions: Our study is limited by the lack of a control group, but the results suggest that sleeping glucose levels decrease and are more stable after patients with type 2 diabetes and OSA are treated with CPAP.

Keywords: Sleep disordered breathing, diabetes mellitus, continuous glucose monitoring, glucose variability

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Obstructive sleep apnea (OSA) and type 2 diabetes mellitus are frequently comorbid conditions. Clinic-based estimates of the prevalence of OSA in type 2 diabetes range from 18% to as high as 36%1,3 and some 50% of patients with OSA have type 2 diabetes or impaired carbohydrate metabolism.4 Population surveys, the Wisconsin Sleep Cohort,4 and the Sleep Heart Health Study5 estimate the prevalence of type 2 diabetes in patients with OSA (apnea-hypopnea index [AHI] > 15) to be about 15%. Obesity is common in both conditions, but OSA is associated with increased insulin resistance independent of obesity.7,8

If OSA increases insulin resistance, then one might expect treating it to increase insulin sensitivity and to improve glycemic control. However, studies of the effect of treating OSA on insulin sensitivity in diabetics and nondiabetics have yielded inconsistent and mostly negative results.9,10 However, Babu et al11 reported an improvement in postprandial blood glucose after patients with type 2 diabetes and OSA were treated with continuous positive airway pressure (CPAP) for about 3 months.

OSA causes intermittent hypoxia and sleep fragmentation, which are capable of activating the sympathetic nervous system and the hypothalamic-pituitary axis.9 By decreasing this activation, CPAP therapy might result in short-term improvements in glycemic control during sleep in patients with OSA that would be reflected less in daytime values or in slowly responding measurements such as insulin sensitivity and hemoglobin A1c (HbA1c).

In this study, we used a continuous glucose monitoring system (CGMS) to measure glucose levels during polysomnography recordings of sleep in patients with type 2 diabetes and moderate to severe OSA, first before treatment and then after 4 to 13 weeks of CPAP therapy.

METHODS

Subjects

We selected patients with type 2 diabetes who were on a stable diabetic regimen. All were recruited at the time of their initial consultation with a sleep physician. They were considered eligible if the history and physical examination, supplemented by overnight oximetry, indicated that the patients were likely to have at least moderate OSA. All were newly diagnosed with OSA, and none had any previous experience with CPAP. They were instructed to maintain a constant diet and not to attempt to
lose weight during the study. The study received approval from the Scripps Clinic Human Subjects Committee.

**Procedure**

The interstitial glucose level was monitored during a full-night diagnostic polysomnogram. Patients whose AHI was greater than 15 were eligible to continue the study. They were provided with an autoadjusting CPAP unit for 1 week (REMstar Auto with Encore Smart Card, Respironics, Pittsburg, PA) and then were switched to fixed-level CPAP set at the 90th percentile of the pressure required during the autoadjusting period. CPAP was continued for at least 3 more weeks, during which patients were seen weekly to monitor their adherence and to deal with any problems in using the equipment. We required that, for patients to be eligible to continue the study, they had to use the CPAP for an average of at least 4 hours per night from the time they began fixed-level CPAP. These patients had a second polysomnogram with recording of interstitial glucose while they were on fixed-level CPAP. Subjects were seen during the second day after their polysomnogram to remove the CGMS and to download the data so that we could obtain the minimum of 36 hours of glucose recording.

**Interstitial Glucose**

Glucose levels were monitored using the CGMS System (Medtronic-MiniMed, Northridge, CA), which records the interstitial glucose level every 5 minutes for up to 72 hours. The sensor was inserted subcutaneously in the lower abdomen in the afternoon or early evening before the study night at least 3 hours before lights out. The CGMS was calibrated against a finger-stick blood glucose, the results of which were entered 3 to 4 times daily.

Subjects were sent to the hospital cafeteria with instructions to take their evening meal at the same time and in the same amount on the 2 nights of polysomnography.

**Polysomnography**

Polysomnography was done using the Compumedics E-series system (Compumedics USA, Ltd., El Paso, TX). We followed the guidelines of the American Academy of Sleep Medicine using 2 channels of electroencephalography (C3-A2, O1-A2), 2 electrooculogram channels, submental electromyography, surface anterior tibial electromyography, electrocardiography, nasal air flow by thermistor and/or nasal pressure transducer, respiratory movements (thoracic and abdominal belts), snoring, body position, and pulse oximetry. The American Academy of Sleep Medicine 1999 criteria were used for identifying apneas and hypopneas.

**Data Analysis**

The sleep stage was scored for each 30-second epoch of the polysomnogram. To account for the time lag between the blood glucose and the interstitial glucose levels, 5 minutes were added to the time of each polysomnography epoch. Since the epochs were 30 seconds each, whereas the CGMS reported the glucose level every 5 minutes, the same glucose level was attributed to a block of 10 polysomnography epochs. The actual difference between the polysomnography epoch time and the CGMS time could range from 4.5 to 9.5 minutes. This is consistent with published estimates of the time delay between the interstitial glucose level recorded by the CGMS and the plasma glucose level.

The primary endpoint was the mean interstitial glucose for all epochs scored as sleep from lights out to lights on (Gsleepmean). Secondary endpoints were the average glucose from lights out to lights on (Gnocmean), the average glucose during epochs scored as awake after sleep onset (Gwaso), the standard deviation (SD) of the sleeping glucose (Gsleeppmax), the maximum sleeping glucose (Gsleeppmax), and the difference between the maximum and minimum sleeping glucose (Gsleepmax).

We also calculated the mean and SD of the 24-hour interstitial glucose and the postprandial level after breakfast. We determined the postprandial glucose by finding the peak interstitial value during a 2-hour window after the time the subject recorded for beginning breakfast. The values were then averaged over the period 30 minutes before and 30 minutes after the peak time. This should be similar to the postprandial value that Babu and associates described as decreasing after CPAP therapy. They recorded the mean values over a 1-hour period “on the basis of correlation with the food diary.”

As an additional assessment of glucose variability, we calculated MAGE, the mean amplitude of glycemic excursions, as described by Service et al. MAGE is designed to include major and to exclude minor fluctuations in the glucose level by averaging the peak-to-trough differences of changes exceeding 1 SD of the values observed over 24 hours. MAGE has shown a close correlation with urinary 8-iso prostaglandin F2α, an indicator of oxidative stress, and predicts it better than does the 24-hour coefficient of variation of glucose levels. We also calculated MAGE over the 8-hour interval from 2300 to 0700 during which our subjects were fasting and were sleeping most of the time, though we recognize that all of the published data on MAGE have calculated it over 24 to 48 hours.

**Statistical Analysis**

Most of the data were not normally distributed, and so we used 2-tailed nonparametric tests—Wilcoxon signed rank test for comparisons and Spearman r for correlation. We used GraphPad Prism version 5 for Macintosh (GraphPad Software, San Diego, CA) with an α of 0.05 to reject the null hypothesis.

**RESULTS**

**Subjects**

A total of 28 subjects completed the first night of the study. One was dropped because her AHI proved to be less than 15. Two were dropped because the CGMS recording was unsatisfactory. Three additional subjects were not able to sleep with CPAP for the required 4 hours per night. This left 22 subjects who then completed the second study night while using CPAP. One of these was dropped from the analysis because he showed many central apneas during his night on CPAP with no improvement in the AHI. One additional subject recruited early in the study was excluded from the final analysis because she was the only type 1 diabetic and the only subject on an insulin pump. That left 20 subjects who completed the 2 study nights, and their data
were included in the analysis. Their characteristics are described in Table 1. Eighteen of the 20 had a body mass index greater than 30, and 10 had a body mass index greater than 40. Sixteen of the 20 had severe sleep apnea (AHI > 30). In 8, the diabetes was not well controlled (Hb\textsubscript{A1c} > 7.0). Eighteen were on only oral medications, and 2 were on insulin plus oral medications.

### Clinical and Polysomnography Findings with CPAP

The clinical data are summarized in Table 2. Twelve subjects gained weight from the first to the second study night, 6 of them more than 2 kg. Four subjects had an AHI above 10 with CPAP, though the AHI was greatly improved from before treatment (74 to 113 before treatment decreasing to 23 to 29 after). In 1 of the 4, all of the events on CPAP were central apneas, whereas, in the other 3, they were a mixture central events and hypopneas that were difficult to characterize as obstructive or central.

The polysomnography results are summarized in Table 3. After treatment, the subjects showed an increased total sleep time with less time awake after sleep onset and less time in stage 1 and more time in stage 3-4 and in stage REM. The number of arousals was significantly reduced with treatment.

### Interstitial Glucose Levels

The sleeping and nocturnal glucose levels are summarized in Table 4. The mean sleeping glucose level decreased with CPAP treatment, and it was less variable, as indicated by the decreased SD and the reduced difference between the maximum and minimum sleeping values. There was no much difference between Gno\textsubscript{mean} and Gsleep\textsubscript{mean}, and the latter also decreased significantly on the CPAP night. The mean sleeping glucose level decreased in 10 of the 11 subjects whose Gsleep\textsubscript{mean} was greater than 100 mg/dL (5.5 mmol/L), and, in the other, it increased by only 2 mg/dL (0.11 mmol/L). There was no decrease in the 9 subjects whose Gsleep\textsubscript{mean} was less than 100 mg/dL (untreated 84.3 mg/dL; treated 85.1 mg/dL), but, even in these subjects, there was a tendency for Gsleep\textsubscript{max} to decrease (untreated 140.7 mg/dL (7.74 mmol/L and with CPAP, 118.2 mg/dL [6.50 mmol/L]; p = 0.07). There was no significant correlation between the change in Gsleep\textsubscript{mean} and the AHI (r = -0.02, p = 0.95).

Gsleep\textsubscript{max} was also significantly decreased on the treatment night. On the untreated night, there was a nonsignificant tendency for Gsleep\textsubscript{max} to be greater than Gsleep\textsubscript{mean} (p = 0.055), whereas Gsleep\textsubscript{max} was no greater than Gsleep\textsubscript{mean} on the treatment night.

### DISCUSSION

Sleeping and nocturnal hyperglycemia were reduced and the sleeping interstitial glucose level was less variable during CPAP treatment in this group of mostly obese type 2 diabetics with moderate to severe, mostly severe, OSA.

We also found a decrease in the mean 24-hour glucose but not in the Hb\textsubscript{A1c}. The lack of change in the Hb\textsubscript{A1c} may be explained in part by the fact that only 3 of our subjects had 60 days or more of CPAP before the second PSG. Babu and associates showed a significant reduction in Hb\textsubscript{A1c} in those subjects with a baseline level greater than 7%. However, among their subjects who adhered well to CPAP therapy, the Hb\textsubscript{A1c} decreased consistently only in those who were treated for 60 days or more. The diabetes was less well controlled in the subjects of the Babu study than in ours, with a mean baseline Hb\textsubscript{A1c} of 8.3%, but we found no decrease with CPAP therapy even in our 8 subjects whose baseline was above 7%.

### Abbreviations

- BMI refers to body mass index; AHI, apnea-hypopnea index; CPAP, continuous positive airway pressure; PSG, polysomnogram; Hb\textsubscript{A1c}, glycated hemoglobin.

### Table 1—Characteristics of the 12 Men and 8 Women Who Took Part in the Study

<table>
<thead>
<tr>
<th>Part in the Study</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>59.8</td>
<td>10.2</td>
<td>34-75</td>
</tr>
<tr>
<td>BMI, kg/m(^2)</td>
<td>39.6</td>
<td>8.0</td>
<td>23-59</td>
</tr>
<tr>
<td>Duration of diabetes, y</td>
<td>9.8</td>
<td>7.7</td>
<td>0.3-25</td>
</tr>
<tr>
<td>AHI, no./h</td>
<td>63</td>
<td>30.4</td>
<td>19-113</td>
</tr>
<tr>
<td>CPAP use, h/day</td>
<td>5.8</td>
<td>1.0</td>
<td>4.6-7.3</td>
</tr>
<tr>
<td>Days on CPAP before second PSG</td>
<td>41.0</td>
<td>17.0</td>
<td>26-96</td>
</tr>
<tr>
<td>Hb\textsubscript{A1c} %</td>
<td>7.1</td>
<td>1.3</td>
<td>4.8-10.3</td>
</tr>
</tbody>
</table>

### Table 2—Changes with CPAP Therapy

<table>
<thead>
<tr>
<th></th>
<th>Untreated</th>
<th>CPAP</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight, kg</td>
<td>117.9</td>
<td>118.9</td>
<td>0.02</td>
</tr>
<tr>
<td>ESS, score</td>
<td>11.2</td>
<td>4.9</td>
<td>0.001</td>
</tr>
<tr>
<td>AHI, no./h</td>
<td>63</td>
<td>30.4</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Hb\textsubscript{A1c} %</td>
<td>7.1</td>
<td>7.2</td>
<td>1.3</td>
</tr>
</tbody>
</table>

### Table 3—Polysomnography Results

<table>
<thead>
<tr>
<th></th>
<th>Untreated</th>
<th>CPAP</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total sleep, min</td>
<td>367</td>
<td>372</td>
<td>NS</td>
</tr>
<tr>
<td>WASO, min</td>
<td>71</td>
<td>61</td>
<td>NS</td>
</tr>
<tr>
<td>Sleep stage, min</td>
<td>43</td>
<td>27</td>
<td>NS</td>
</tr>
<tr>
<td>1</td>
<td>241</td>
<td>233</td>
<td>NS</td>
</tr>
<tr>
<td>2</td>
<td>34</td>
<td>37</td>
<td>NS</td>
</tr>
<tr>
<td>3</td>
<td>46</td>
<td>69</td>
<td>NS</td>
</tr>
<tr>
<td>Sleep efficiency, %</td>
<td>79</td>
<td>85</td>
<td>NS</td>
</tr>
<tr>
<td>Arousal index, no./h</td>
<td>51</td>
<td>2</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Total</td>
<td>64</td>
<td>17</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

Data are presented as median (interquartile range). CPAP refers to continuous positive airway pressure; WASO, wake time after sleep onset.

The mean 24-hour glucose, beginning at midnight of the polysomnography night, also decreased significantly during CPAP treatment. However, the change with CPAP in the mean daytime glucose from 0700 hours to 2300 hours (167.4 ± 52.9 to 159.3 ± 51.6) was not significant (p = 0.36). We found no significant change in the variability of the 24-hour glucose levels when assessed by either the SD or MAGE. There was a tendency for MAGE, calculated from 2300 to 0700 hours, to decrease with CPAP therapy, but the change was not statistically significant. There was no significant change in the Hb\textsubscript{A1c} or in the breakfast postprandial glucose level.

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The improvement in these nocturnal disturbances of glucose metabolism might have relatively little effect on daytime glucose metabolism and might not be reflected by measurements of insulin sensitivity or by a change in the Hb\textsubscript{A1c}.

Tasali et al have shown that, in normal subjects, selective suppression of slow-wave sleep produces increased insulin resistance and decreased glucose tolerance.\textsuperscript{24} Though our subjects showed an increase in slow-wave sleep on the CPAP night, the increase was small and not statistically significant.

We found that glucose levels tended to be greater during wake after sleep onset than during sleep on the untreated night but not on the CPAP night. This suggests that CPAP therapy may have reduced short-term rises in glucose levels provoked by arousals and respiratory events.

Our data suggest that glucose levels decreased with CPAP therapy only during the night. However, Babu et al found that, with CPAP, postprandial glucose levels were lower after all 3 meals,\textsuperscript{11} and so it is likely that there are persistent effects of CPAP therapy on daytime glycemic control that our study lacked the power to demonstrate.

Even if CPAP did not decrease the 24-hour mean blood glucose, it could have a beneficial effect on the course of type 2 diabetes by reducing the nocturnal fluctuations in the glucose level. It has been shown that variability of the fasting blood glucose level is an independent risk factor for mortality and for retinopathy in type 2 diabetes.\textsuperscript{25, 26}

The major limitation of our study was the lack of a control group. West et al\textsuperscript{10} included a group treated with sham CPAP, and they pointed out that glycemic control might be improved during a study if people modified their behavior, knowing that they were being monitored. It is not likely that our subjects were more adherent to their diets during the study because there was actually a statistically significant gain in weight from the untreated night to the night on CPAP. We instructed our subjects not to attempt to lose weight, and these obese diabetics probably interpreted this as a license to ease up on their usual efforts to diet. However, we cannot be certain that they did not take their medications more consistently. Another limitation of our study was our inability to ensure that subjects had the same calorie intake at the same time on the 2 polysomnography nights.

### Table 4—Interstitial Glucose Levels

<table>
<thead>
<tr>
<th></th>
<th>Untreated Mean</th>
<th>SD</th>
<th>CPAP Mean</th>
<th>SD</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean sleeping (Gsleep\textsubscript{mean})</td>
<td>120.7</td>
<td>61.1</td>
<td>101.9</td>
<td>39.0</td>
<td>0.03</td>
</tr>
<tr>
<td>SD of sleeping values (Gsleep\textsubscript{sd})</td>
<td>20.0</td>
<td>13.1-31.5</td>
<td>13.0</td>
<td>9.3-18.2</td>
<td>0.004</td>
</tr>
<tr>
<td>Maximum sleeping value (Gsleep\textsubscript{max})</td>
<td>173.7</td>
<td>71.1</td>
<td>135.5</td>
<td>42.6</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Maximum - minimum sleeping value (Gsleep\textsubscript{min})</td>
<td>86.9</td>
<td>46.5</td>
<td>56.0</td>
<td>23.8</td>
<td>0.002</td>
</tr>
<tr>
<td>Mean lights out to lights on (Gnoc\textsubscript{mean})</td>
<td>123.9</td>
<td>62.0</td>
<td>103.9</td>
<td>39.1</td>
<td>0.03</td>
</tr>
<tr>
<td>Mean awake after sleep onset (Gwaso\textsubscript{mean})</td>
<td>128.1</td>
<td>62.7</td>
<td>99.7</td>
<td>38.4</td>
<td>0.02</td>
</tr>
<tr>
<td>Postprandial breakfast</td>
<td>198.0</td>
<td>63.4</td>
<td>196.6</td>
<td>59.6</td>
<td>NS</td>
</tr>
<tr>
<td>24-hour mean value\textsuperscript{a}</td>
<td>153.7</td>
<td>52.0</td>
<td>142.2</td>
<td>43.9</td>
<td>0.03</td>
</tr>
<tr>
<td>24-hour SD\textsuperscript{a}</td>
<td>33.1</td>
<td>25.3-54.3</td>
<td>32.7</td>
<td>30.0-47.7</td>
<td>NS</td>
</tr>
<tr>
<td>MAGE 8\textsuperscript{a}</td>
<td>44.8</td>
<td>38.9-77.5</td>
<td>43.0</td>
<td>31.5-53.1</td>
<td>0.06</td>
</tr>
<tr>
<td>MAGE 24\textsuperscript{a}</td>
<td>77.6</td>
<td>44.1-122.6</td>
<td>82.2</td>
<td>66.3-95.2</td>
<td>NS</td>
</tr>
</tbody>
</table>

Results are presented as mg/dL; to convert to mmol/L, divide by 18. MAGE refers to the mean amplitude of glycemic excursions; MAGE 8, the MAGE calculated over 8 hours from 2300 to 0700; MAGE 24, the MAGE calculated over 24 hours beginning at midnight.

\textsuperscript{a}Median and interquartile range

\textsuperscript{b}Data from 2 subjects excluded because of missing data

*West and associates\textsuperscript{10} also reported that there was no significant change in the Hb\textsubscript{A1c} and no change in insulin sensitivity after 3 months of CPAP therapy. The diabetes of their subjects was also less well controlled than the diabetes of the subjects in our study, with a mean Hb\textsubscript{A1c} of 8.5% before treatment.

Our study was not designed to evaluate the effect of treatment on postprandial glucose levels but, because of the report by Babu et al that postbreakfast levels were lower after treatment,\textsuperscript{11} we analyzed our data to see if we could show a similar improvement. We found no significant change with treatment. Though our subjects fasted until 0700 when they had blood drawn, we did not require them to take breakfast at a specific time or to eat a standard meal. Therefore, any effect of CPAP on the postprandial glucose could have been obscured by variations in the amount and timing of the meal.

Harsch and associates showed an improvement in insulin sensitivity after 3 months of CPAP therapy in a group of obese type 2 diabetics with OSA.\textsuperscript{18} However, they had only 8 subjects in their series after excluding data from 1 who had a significant loss of weight during the study period.

Hassaballa and associates reported a significant decrease in the Hb\textsubscript{A1c} after at least 3 months of CPAP therapy in a group of obese type 2 diabetics, but their findings were based on a retrospective chart review.\textsuperscript{19} They also reported a weight gain in the small number of their subjects for whom the information was available.

The studies cited above evaluated the effect of 3 months of CPAP therapy on glycemic control, but Pallayova et al recently reported that the mean and SD of the overnight interstitial glucose decreased rapidly with the initiation of CPAP therapy. They found a significant improvement even during a CPAP titration night.\textsuperscript{20}

The intermittent hypoxemia associated with OSA has been shown to produce surges of sympathetic activation,\textsuperscript{21, 22} whereas arousals during sleep can induce bursts of cortisol release in normal humans.\textsuperscript{23} Punjabi and associates suggested that intermittent hypoxemia and sleep fragmentation alter the autonomic, hypothalamic-pituitary, and somatotropic axes and also increase the levels of inflammatory cytokines and adipokines, thereby affecting glucose metabolism.\textsuperscript{9} These short-term effects of sleep apnea could worsen hyperglycemia and cause fluctuations of the blood glucose during the night that would diminish with CPAP therapy.
Only 3 of 28 subjects recruited to the study (11%) were dropped because of nonadherence to the CPAP therapy, an adherence rate that is much better than is typically reported in the literature. Adherence was encouraged by the weekly visits with our research nurse, but probably more important was that this was a highly motivated group of subjects who participated in the study without any financial incentive. The study was further biased toward compliant subjects by our exclusion of the data from the 3 subjects whose use of CPAP was less than 4 hours per night. The new diagnosis of sleep apnea might also have encouraged closer adherence to their diabetic regimen. Because our study included only the most compliant subjects, our conclusion that CPAP therapy of diabetics with OSA improves their glycemic control may not be generalizable to other patients who have the 2 conditions.

Our findings need confirmation by a larger study that randomly assigns subjects to sham-CPAP and effective-CPAP arms, but our findings suggest that screening type 2 diabetics for OSA and treating those with moderate to severe sleep-disordered breathing could improve the management of their hyperglycemia and might favorably influence their long-term prognosis.

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DISCLOSURE STATEMENT

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