Surgical vs Conventional Therapy for Weight Loss Treatment of Obstructive Sleep Apnea: A Randomized Controlled Trial

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Obesity is a major risk factor for obstructive sleep apnea (OSA). The estimated prevalence of OSA in obese adults varies from 42% to 48% in men and 8% to 38% in women. Obstructive sleep apnea is strongly related to obesity and associated conditions such as type 2 diabetes and hypertension. Individuals with OSA are at greater risk of stroke, cardiac disease, psychosocial morbidity, cognitive dysfunction, and impaired quality of life.

Therapy for symptomatic OSA focuses on methods to reduce upper airway resistance and prevent obstruction, such as continuous positive airway pressure (CPAP), oral appliances, and upper airway surgery. However, in severely obese patients, none of these options treats the accompanying obesity. Observational studies of major weight loss following bariatric surgery suggest substantial remission of OSA symptoms in up to 60% to 80% of patients. However, in the studies where repeat polysomnography was available, greater reduction in AHI despite major differences in weight loss.

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Surgical Program

Participants underwent 2 weeks of VLED to reduce liver size prior to placement of an LAGB (LAP-BAND System, Allergan Health) via the pars flaccida pathway by 1 of 3 experienced surgeons (P.E.O. and W.B. and Stewart Skinner, MBBS[Hons], PhD, Department of Surgery, Monash University), within 1 month of randomization. Adjustments to band volume were made using standard clinical criteria.18

Patients in both programs had open access to a bariatric physician, sleep physician, and diettitian, and had their progress reviewed every 4 to 6 weeks throughout the 2 years. The management of OSA, the intensity, and nature of the lifestyle program were common to both groups.

Outcome Measures

The primary outcome measure was the change in AHI, measured by diagnostic laboratory polysomnography from baseline to 2 years. Polysomnography was performed using standard electroencephalogram, electrooculogram, electromyogram, nasal pressure cannulae, oronasal thermistor, respiratory inductance plethysmography, finger oximetry, electrocardiography, and video monitoring for body position. Diagnostic polysomnography at years 1 and 2 was performed after a 48-hour CPAP washout at the same institution as the initial test, scored by staff blinded to randomization group, and using the same precise AHI scoring criteria for each study.19

One of the secondary measures was CPAP adherence, which was monitored with 3 monthly computerized downloads recording mean CPAP adherence (in hours) and pressure (in cm H2O). Other diagnostic polysomnography measures included arousal index, percentages of rapid eye movement and slow wave sleep, and the mean and minimum oxygen saturation. We also assessed measures of cardiometabolic risk (office blood pressure, resting heart rate, 6-minute walk test, levels of fasting plasma glucose, insulin, triglycerides, total and high-density lipoprotein cholesterol, and calculated low-density lipoprotein cholesterol); measured weight, waist, hip, and neck circumference; and reported metabolic syndrome status.20

Other secondary outcomes included the following functional status measures: Epworth Sleepiness Scale score,21 which was scored from 0 to 24 with 10 or more considered abnormal, Short Form-36 Health Survey,22 and Beck Depression Inventory.23 Adverse events were recorded throughout the study.

Statistical Analysis

The sample size was selected to provide a statistical power of at least 80%
to detect a between-group difference of 50% in AHI (mean of 20 events/hour for conventional program vs 40 events/hour for surgical program with an SD of 20 events/hour) at 2 years (P < .05), with modeling based on our observational data and expectations of other devices. Twenty-five patients were needed to provide a 95% confidence interval of this difference with a power of 0.80 or more (2-tailed). Recruitment size was therefore set at 60 to allow for a dropout rate of up to 20% or for failure to follow the randomly assigned allocation.

Univariate statistical analysis was performed using SPSS statistical software version 18 (SPSS Inc), with baseline comparisons made using χ² tests for equal proportion, t tests for normally distributed outcomes, or Mann-Whitney tests. A multivariate longitudinal analysis was performed using the PROC Mixed procedure in SAS version 9.2 (SAS Institute Inc), with each patient treated as a random effect.

Longitudinal models were fitted using main effects for group, time, and an interaction between group and time to ascertain if the groups behave differently over time. Covariance structures were chosen to minimize the corrected Akaike and Bayesian information criteria. Multiple imputation was generated for missing data using SAS version 9.2 and was based on the assumption that data were from a multivariate normal distribution and were missing at random. The regression method was used for imputation with 5 imputed data sets used for each variable. The post hoc relationship between changes in weight and AHI were explored by dividing weight loss into quintiles and using comparisons of Pearson correlation coefficients and 95% confidence intervals performed using the Fisher r-to-z transformation. A locally weighted smoothing (loess) curve was used to indicate nonlinear trends in the scatter plots.

All data were analyzed using the intention-to-treat principle. Continuous variables were expressed as means and standard deviations with differences expressed as means and 95% confidence intervals. Sensitivity analyses examining assigned only completer and per-protocol completer were performed. A 2-sided P value of .05 was considered to be statistically significant.

RESULTS

Study participant flow and reasons for exclusion are shown in Figure 1. There were no statistically significant between-group differences in the baseline characteristics (TABLE 1). In the surgical group, 4 participants did not consent to surgery but continued in the study and received conventional therapy; complete 2-year follow-up data were available in 28 (93%) of the 30 participants. Of the 30 participants in the conventional group, the 2-year program was completed by 28 (93%), and all data were collected for 26 (87%). One conventional group participant elected to undergo LAGB surgery soon after randomization. Another conventional therapy participant was unable to undergo a repeat diagnostic polysomnography because he or she was unable to sleep without the CPAP device.

All bands were placed laparoscopically with a mean (SD) procedure time of 70 (14) minutes (range: 40-105 minutes). The lengths of hospital admission were 1 day for 24 participants (92%) and 2 days for 2 participants (8%). In the conventional group, 17 participants chose to use an intensive VLED program for 4 to 6 weeks, another 5 used a more conservative initial program; 11 of these 22 elected to use a partial or intermittent intensive VLED meal replacement throughout the study.

Weight Loss

The surgical group achieved a significantly greater mean weight loss of 27.8 kg (95% CI, 20.9-34.7 kg) from 134.9 kg to 107 kg or a mean of 20.6% (95% CI, 15.4%-25.7%) of initial body weight.
at 2 years compared with 5.1 kg (95% CI, 0.8-9.3 kg) from 126.0 kg to 121.8 kg or a mean of 2.9% (95% CI, 0.6%-7.3%) among the conventional group (P = .07; Table 2, Figure 2, and eFigure 1 at http://www.jama.com). This represents a reduction of body mass index from 46.3 to 36.6 with surgery vs 43.8 to 42.3 with conventional therapy.

Apnea-Hypopnea Index

Both groups had a significant decrease in total AHI between baseline and 2 years, with a decrease of 25.5 events/hour (95% CI, 14.2 to 36.7 events/hour) from 65.0 events/hour to 39.5 events/hour or a mean of 31.4% (95% CI, 12.7% to 50.2%) (calculated for individuals) in the surgical group and 14.0 events/hour (95% CI, 3.3 to 24.6 events/hour) from 57.2 events/hour to 43.2 events/hour or a mean of 13.5% (95% CI, −5.8% to 32.9%) in the conventional group (Table 2 and Figure 2). The between-group difference was −11.5 events/hour (95% CI, −28.3 to 5.3 events/hour; P = .18) or percentage change in AHI of −15.9% (95% CI, −44.3% to 8.5%; P = .18). The sensitivity analysis confirmed that the results were not altered by inclusion of only completers when those in their assigned or per-protocol group followed the program. Thus, despite the substantially greater weight loss with surgical therapy, there was not a greater improvement in AHI. An AHI of less than 15 events/hour, which indicates mild OSA, was achieved by 8 participants in the surgical group (27%) and by 2 participants in the conventional group (7%) (P = .04); only 1 participant in the conventional group achieved an AHI of less than 5 events/hour, which indicates OSA remission.

A post hoc analysis showed a significant positive relationship between the change in weight and change in AHI (r = 0.45 [95% CI, 0.22 to 0.63]; P < .001). However, when examined in the individual groups, the relationship was demonstrated only in the conventional group (r = 0.69 [95% CI, 0.44 to 0.84]; P < .001); in the surgical group, the analysis yielded r = 0.33 (95% CI, −0.03 to 0.62; P = .07) (conventional group r = 0.69 vs surgical group r = 0.33; P = .06). Scatterplots (with loess curves) of change in weight and change in AHI, and percentage weight loss and percentage change in AHI (Figure 3) demonstrate an attenuated benefit beyond the first 8% to 10% of weight loss, and great variability of any change with weight loss (eFigure 2). Table 2 details the treatment effects on other polysomnography variables.

CPAP Usage and Adherence

Within the surgical group (n = 30), 28 participants had CPAP initiated; however, at 3 months, only 20 participants were adherent (mean pressure: 11.9 cm H2O used for 4.9 hours/night) and at 2 years, only 14 participants were adherent (mean pressure: 11.6 cm H2O for 5.2 hours/night). Within the conventionally treated group (n = 30), 25 participants had CPAP initiated; however, at 3 months, only 21 participants were adherent (mean pressure: 11.7 cm H2O used for 3.9 hours/night) and at 2 years, only 18 participants were adherent (mean pressure: 11.5 cm H2O used for 5.6 hours/night). There were no significant between-group differences in CPAP adherence or mean pressure delivered.

Table 1. Baseline Characteristics of Participants

<table>
<thead>
<tr>
<th>No. (%) of Participants</th>
<th>Surgical Treatment (n = 30)</th>
<th>Conventional Weight Loss Program (n = 30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male sex</td>
<td>17 (67)</td>
<td>18 (60)</td>
</tr>
<tr>
<td>Hypertension*</td>
<td>15 (50)</td>
<td>17 (57)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>10 (33)</td>
<td>10 (33)</td>
</tr>
<tr>
<td>Depression</td>
<td>12 (40)</td>
<td>11 (37)</td>
</tr>
<tr>
<td>Metabolic syndrome†</td>
<td>19 (63)</td>
<td>24 (80)</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>47.4 (8.8)</td>
<td>50.0 (8.2)</td>
</tr>
<tr>
<td>Body mass index*</td>
<td>46.3 (6.0)</td>
<td>43.8 (4.9)</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>134.9 (22.1)</td>
<td>126.0 (19.3)</td>
</tr>
<tr>
<td>Waist circumference, cm</td>
<td>136.1 (13.1)</td>
<td>126.8 (13.1)</td>
</tr>
<tr>
<td>Neck circumference, cm</td>
<td>46.8 (4.9)</td>
<td>46.8 (3.8)</td>
</tr>
<tr>
<td>Blood pressure, mm Hg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>137.6 (18.7)</td>
<td>142.2 (16.9)</td>
</tr>
<tr>
<td>Diastolic</td>
<td>83.1 (8.2)</td>
<td>86.8 (8.8)</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>73.2 (7.1)</td>
<td>70.2 (8.7)</td>
</tr>
<tr>
<td>Apnea-hypopnea index, events/h</td>
<td>65.0 (32.8)</td>
<td>57.2 (30.3)</td>
</tr>
<tr>
<td>Available hemoglobin saturated with oxygen, % During sleep</td>
<td>94.6 (2.7)</td>
<td>94.7 (2.7)</td>
</tr>
<tr>
<td>Lowest level</td>
<td>72.5 (13.1)</td>
<td>72.4 (19.2)</td>
</tr>
<tr>
<td>Arousal index, events/h</td>
<td>41.6 (31.8)</td>
<td>38.4 (26.0)</td>
</tr>
<tr>
<td>Fasting plasma glucose, mg/dL</td>
<td>107.4 (30.6)</td>
<td>103.7 (23.5)</td>
</tr>
<tr>
<td>Hemoglobin A1c, %</td>
<td>6.25 (1.1)</td>
<td>6.26 (1.1)</td>
</tr>
<tr>
<td>Fasting plasma insulin, µU/mL</td>
<td>32.0 (22.0)</td>
<td>26.8 (19.1)</td>
</tr>
<tr>
<td>Cholesterol, mg/dL</td>
<td>194.4 (42.2)</td>
<td>192.2 (36.1)</td>
</tr>
<tr>
<td>Total</td>
<td>44.8 (10.4)</td>
<td>43.6 (10.4)</td>
</tr>
<tr>
<td>High-density lipoprotein</td>
<td>163.7 (83.1)</td>
<td>195.6 (96.9)</td>
</tr>
</tbody>
</table>

*Conversion factors: To convert glucose to mmol/L, multiply by 0.0555; hemoglobin A1c to proportion of total hemoglobin, multiply by 0.01; high-density lipoprotein and total cholesterol to mmol/L, multiply by 0.0259; insulin to pmol/L, multiply by 6.945; triglycerides to mmol/L, multiply by 0.0113.
†Defined as blood pressure of 140/90 mm Hg or higher or current use of antihypertensive medication.
‡Compliance assessed by self-report.
§Determined by the US National Cholesterol Education Program Adult Treatment Panel III requirements of at least 3 of the following to be met: waist circumference: greater than 102 cm for men and greater than 88 cm for women; triglycerides level: 150 mg/dL or greater; high-density lipoprotein cholesterol: less than 40 mg/dL in men and less than 50 mg/dL in women; blood pressure: systolic, 130 mm Hg or greater and diastolic, 85 mm Hg or greater; fasting plasma glucose: 110 mg/dL or greater.
**Other Health Outcomes**

eTable 1 shows changes in clinical and laboratory measures of health at 2 years. There were no between-group differences in blood pressure, fasting glucose and insulin, or plasma lipid profile. There were no within- or between-group differences in the use of antihypertensive, diabetes, lipid-lowering, or antidepressant medication between baseline and 2 years. Twenty participants had type 2 diabetes at baseline (10 in each group) and a further 2 in the conventionally treated group developed type 2 diabetes during the study. For those with diabetes (n = 22), the mean hemoglobin A1c levels were 7.2% (95% CI, 6.4% to 7.9%) for those in the surgical group (n = 10) and 7.3% (95% CI, 6.4% to 8.1%) in the conventional group (n = 12) at baseline and 6.0% (95% CI, 5.6% to 6.4%) and 7.0% (95% CI, 6.3% to 7.7%), respectively, at 2 years. There was a significant within-group change of −1.26% (95% CI, −1.97% to −0.55%; P = .003) in those with diabetes in the surgical group compared with −0.3% (95% CI, −1.16% to 0.5%; P = .46) in the conventional group; however, the between-group difference of −0.96% (95% CI, −2.04% to 0.12%; P = .08) was not statistically significant.

**Functional Outcomes**

Baseline health-related quality-of-life and summary scores on the Short Form-36 scale were not statistically different between the 2 groups. The surgical group had greater improvement in scores between baseline and 2 years for physical role, general health, vitality, and the physical component summary (mean, 9.3 [95% CI, 0.5 to 18.0]; P = .04 for physical component summary score; eTable 2). Both groups experienced a reduction in daytime sleepiness as measured with the Epworth Sleepiness Scale, reductions in symptoms of depression, and improvement in the 6-minute walk test, but there were no significant between-group differences.

**Adverse Events**

There was no mortality in either group. One patient experienced an acute proximal gastric pouch dilatation causing obstructive symptoms and requiring elective laparoscopic replacement of the LAGB 1 month later. This patient’s hospital stay was less than 1 day and there were no complications (eTable 3).

### Table 2. Longitudinal Analysis With Multiple Imputation for Missing Data for Polysonomography Variables and Weight at 2 Years

<table>
<thead>
<tr>
<th>Mean (95% CI)</th>
<th>Surgical Treatment (n = 30)</th>
<th>Conventional Weight Loss Program (n = 30)</th>
<th>Between-Group Difference</th>
<th>No. (%) Missing at 2 y</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Weight, kg</strong></td>
<td>107 (99 to 116)</td>
<td>121 (113 to 129)</td>
<td>−27.8 (−34.7 to −20.9)</td>
<td>2 (3)</td>
<td>.001</td>
</tr>
<tr>
<td><strong>Apnea-hypopnea index, events/h</strong></td>
<td>39.5 (28.4 to 50.5)</td>
<td>43.2 (34.9 to 51.9)</td>
<td>−3.7 (−17.4 to 10.0)</td>
<td>1 (2)</td>
<td>.52</td>
</tr>
<tr>
<td><strong>Total sleep time, min</strong></td>
<td>373 (348 to 399)</td>
<td>333 (297 to 370)</td>
<td>26 (−31.7 to 8.0)</td>
<td>1 (2)</td>
<td>.60</td>
</tr>
<tr>
<td><strong>Sleep latency, min</strong></td>
<td>18.6 (11.3 to 26)</td>
<td>24.5 (15.0 to 34.1)</td>
<td>−5.9 (−18.7 to 7.0)</td>
<td>1 (2)</td>
<td>.31</td>
</tr>
<tr>
<td><strong>Sleep efficiency, %</strong></td>
<td>79.8 (75.8 to 83.9)</td>
<td>72.4 (65.3 to 79.6)</td>
<td>7.4 (−11.3 to 26.1)</td>
<td>1 (2)</td>
<td>.29</td>
</tr>
<tr>
<td><strong>Supine sleep, %</strong></td>
<td>47.1 (35.5 to 58.6)</td>
<td>37.0 (25.1 to 48.8)</td>
<td>−10.1 (−21.2 to 0.1)</td>
<td>1 (2)</td>
<td>.05</td>
</tr>
<tr>
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<td>1 (2)</td>
<td>.05</td>
</tr>
</tbody>
</table>

aIndicates significant within-group improvement (P < .05).

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COMMENT
Comparing surgical and conventional weight loss therapy for the management of OSA demonstrated greater weight loss in the surgically treated group, however, this did not translate into significantly greater improvements in OSA. Our study did confirm that weight loss is associated with an improvement in AHI, but we found great variability in the individual effect. The pattern of improvement in AHI suggests that much of the benefit is associated with mild to moderate weight loss, with limited additional benefit with further weight loss.

Our findings support the 2 independent meta-analyses by Greenburg et al9 and Chang et al27 of before and after case series trials, in which they concluded that only a minority of obese patients with OSA are free of OSA after bariatric surgery. This contrasts with a meta-analysis of bariatric surgical treatment groups that described resolution of OSA symptoms in 85.7% of patients. However, only 4 of 24 studies (17%) reported polysomnography or AHI outcomes.9

Data from the present study (and that from the systematic reviews) do not support routine cessation of CPAP or other therapies for OSA following bariatric surgery.9,27 Formal assessments, including polysomnography, should be considered before informing a patient that he or she may cease CPAP therapy. The clinical picture can be confounded by patients self-reporting improved quality of life, sleepiness, sleep quality, and exercise tolerance after bariatric surgery.

Our findings support a complex, rather than pure mechanical load, pathogenesis of OSA in obese individuals. The nonlinear relationship may indicate the importance of the state of weight loss rather than the extent of weight loss in generating an altered neurohumoral and metabolic-inflammatory milieu influencing defects in neuromuscular responses to mechanical load.9,28 Population studies indicate the influence of weight on OSA varies with age and sex, and additional factors such
as boney structures are also contributors to risk.30,31 Thus the relationship of the extent of weight loss with improvement of OSA needs further investigation. However, the benefits of modest weight loss have several other major public health implications12,33 that should not be diminished by the outcomes of this study.

Several limitations of this study need to be recognized. First, the study may have been underpowered given the extraordinary variance in the effect of weight loss on AHI. We relied on data from our observational study that may have been biased because it relied on patients volunteering for a follow-up polysomnography study.23 However, the clinical relevance of a much larger study, even if a significant effect of weight loss on AHI were demonstrated, would be limited because the effect of weight loss is highly variable and incomplete. Second, we used LAGB surgery, which is associated with a slower weight loss and a lower mean weight loss than gastric bypass and bilipancreatic diversion at 2 years following surgery.24 However our data do not provide a signal that greater weight loss would achieve a better response. Third, we restricted the study to those with a body mass index of 35 to 55 with moderate to severe OSA. Results in those in a lower weight range or with less severe OSA may be different.33 In addition, this study was not powered for safety, the wide range of secondary outcome measures, or to detect differences in end points such as mortality or cardiovascular events.

In conclusion, greater weight loss in the surgical group did not translate into better effect as a therapy for OSA compared with conventional weight loss therapy. While there is a clear overall response to weight loss, the individual effects are variable, incomplete, and appear to be related to modest weight loss. This study suggests caution be used in counseling patients about the expected benefits of weight loss for OSA, and that patients losing weight be assessed appropriately before ceasing effective therapy.

**Author Contributions:** Drs Dixon and Bailey had full access to all the data in the study and take responsibility for the integrity and the accuracy of the data analysis.

**Study concept and design:** Dixon, Schachter, O’Brien, Naughton.

**Acquisition of data:** Dixon, Schachter, O’Brien, Jones, Grima, Brown, Naughton.

**Analysis and interpretation of data:** Dixon, Schachter, O’Brien, Jones, Grima, Lambert, Brown, Bailey, Naughton.

**Drafting of the manuscript:** Dixon, Schachter, O’Brien, Jones, Grima, Bailey, Naughton.

**Critical revision of the manuscript for important intellectual content:** Dixon, Schachter, O’Brien, Jones, Grima, Lambert, Brown, Bailey, Naughton.

**Statistical analysis:** Dixon, Bailey, Naughton.

**Obtained funding:** Dixon, O’Brien, Naughton.

**Administrative, technical, or material support:** Dixon, Schachter, O’Brien, Jones, Grima, Lambert, Brown.

**Study supervision:** Dixon, O’Brien, Jones, Grima, Lambert, Naughton.

**Conflict of Interest Disclosures:** The authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Dr Dixon reported being supported by a National Health and Medical Research Council Senior Research Fellowship; his laboratory receives educational grants from Medtronic Inc; serving as a consultant for Medtronic Inc, Bariatric Advantage, and Intellicon, and being a member of the editorial board for Sleep Medicine; serving on the speakers bureau for Eli Lilly and iNovo Pharmaceuticals; developing educational material for Novartis and iNovo Pharmaceutical; and receiving travel assistance from Gilead sciences for an educational meeting. Dr Schachter reported being the medical director of Sleep Services Australia, which uses continuous positive airway pressure machines and masks from Resmed Australia, Fisher and Paykel New Zealand, and Phillips Respironics United States. Dr O’Brien reported receiving compensation as the national medical director for the American Institute of Gastric Banding since 2007; receiving compensation as the author of The Lap-Band Solution: A Partnership for Weight Loss; serving as the emeritus director of the Centre for Obesity Research and Education (CORE), which receives a grant from Amgen Medical toward educational programs; and receiving a grant from Allergan Inc for investigator-initiated research. Dr Lambert reported being supported by an National Health and Medical Research Council Senior Research Fellowship, his laboratory currently receives commercial research grant funding from Medtronic (formerly ARIDIAN Inc), Servier Australia, Abbott (formerly Solvay), and Allergan Inc; serving as consultant for Medical Information Systems; travel support for presentations at Pfizer, Wyeth Pharmaceuticals, Servier, and Medtronic. Dr Brown reported receiving an honorarium from Servier Inc for attending a scientific advisory panel in London in 2009; serving as the director of CORE, which receives a grant from Allergan Inc for research support but the grant is not tied to any specified research projects and Allergan has no control of the protocol, analysis, and reporting of any studies; and that CORE receives a grant from Applied Medical toward educational programs. Dr Naughton reported being a recipient of competitive funding from Resmed and Respironics to assist in investigator-directed research; serving on the Australian Medical advisory boards of Resmed, Medtronics, Covidian and Fisher, and Paykel, but that he and his family hold no stock or have any financial interest in any company related to sleep apnea or obesity. Drs Jones and Bailey and Ms Grima did not report any disclosures.

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**Role of the Sponsors:** The funding sponsors played no part in the design and conduct of the study; collection, management, analysis, and interpretation of the data; and preparation, review, or approval of the manuscript. Sponsors had no access to the data and did not perform any of the study analysis.

**Online-Only Material:** eFigures 1 and 2 and eTables 1 through 3 are available at http://www.jama.com.

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**REFERENCES**


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