In Reply  We agree that our results are based on a small number of respondents who met criteria for major depressive disorder and mood disorders. We were transparent about this and noted sample size as the study’s primary limitation. As stated in our article, the National Survey of American Life (NSAL) remains one of the best data sets for this study. We considered using National Comorbidity Survey Replication data; however, it contained a limited number of rural cases. Given the sample size limitation, our article emphasized the importance of future research examining major depressive disorder and mood disorders among African American individuals with other samples.

Second, Keyes and colleagues’ statement that “the national sample weights cannot be assumed to be appropriate for approximating regional-specific estimates...” is not a valid statement for the NSAL. Because the nonresponse adjustment factors in the NSAL weights are computed separately for NSAL respondents from the South region and primary stage sampling units with the design strata representing the South, the weights (including the implicit nonresponse) are uniquely determined for the South and the weight computation process is not influenced by the response experience or the patterns of depression in other regions of the country. We are confident that the NSAL weights are computed “appropriately” for both national estimates and analyses for subpopulations.

Third, Keyes et al criticize us for speculating on our findings. However, that is the purpose of a Discussion section. They minimize the importance of religiosity as a potential mechanism to explain lower rates of major depressive disorder among African American women. However, research consistently reports higher rates of religiosity among African American individuals, and that religiosity is protective for depression. Krause’s extensive body of work suggests religion and church-based social support are protective of mental and physical health, including mortality. Most of his studies demonstrate that older African American individuals are more likely than older white individuals to reap mental and physical health-related benefits of religion owing to higher levels of religious participation and church-based support networks. Additionally, Reese et al found that when controlling for religious service attendance, the black-white difference in depression was no longer significant. Clearly, our speculation about the importance of religious participation is “fraught with problematic assumptions lacking empirical support” as stated by Keyes et al.

Last, Keyes et al argue that our focus on religiosity and social ties risks minimizing “the role of structural, economic, and sex discrimination experienced by African American women, while potentially reifying stereotypes.” This is by no means the case. In fact, much of the contemporaneous and historical research on black-white differences in the social sciences has taken a deficit approach and has not adequately considered African American women’s strengths. That is, despite a history of slavery and Jim Crow laws, high levels of poverty, segregation and low-quality education, and substandard housing, African American women in the rural South continue to cope. We take a strengths-based approach and argue that low rates of depression among rural African American women are potentially owing to their high levels of religiosity and their family ties.

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Error in Calculating Main Outcome in Gamma Ventral Capsulotomy for Obsessive-Compulsive Disorder Randomized Clinical Trial

To the Editor  In many ways, the Lopes and coauthors randomized clinical trial on gamma ventral capsulotomy (GVC) in patients with treatment-refractory obsessive-compulsive disorder stands out. Ethics approval and informed consent pro-
cerebrospinal fluid was collected after surgery. Patients were followed up for an exceptionally long period. The article is among the few in psychiatry documenting binding of raters and patients, although such data are not only important, as presented, at follow-up but particularly immediately after surgery (as apparently ascertained). Further, the authors make their study transparent in providing a wealth of outcome data.

It appears from these data that a small error occurred in calculating the mean outcome: Table 1 and the P value for comparing the decrease in Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) scores between treatment and control indicate the baseline Y-BOCS score of the third patient in the treatment group (ATa3) was not 36 but 30. With a follow-up score of 22, the decrease was 26.7%, rather than 38.9%, as stated in Table 2. Apparently, however, Lopes et al concluded from the latter figure that patient 3 was a responder because the decrease exceeded 35%. Without this patient, the number of responders under GVC stands at 2 of 8, with no responder among controls (Fisher exact test: P = .47). The absolute risk reduction, therefore, is reduced from statistically significant 37.5% (95% CI, 4% to 71%) to 25% (95% CI, −5% to 55%; nonsignificant), and number needed to treat increases to 4 (95% CI, 1.7 to ∞). Given group sizes of 8, Y-BOCS score change, when compared as a continuous variable, should be analyzed using the exact Mann-Whitney U test and not its asymptotic version. Thus, P slightly rises from .046 to .050 (or to .0499, to be exact). Similarly, when recalculated, the P value for change in Dimensional Y-BOCS score increased from 0.01 to 0.02. These differences are not entirely moot in a study with a host of outcomes and not adjusted for multiple tests. As a consequence, corrected figures for both dichotomous and continuous outcomes (calculated with Open Epi and SPSS version 22 [IBM]) suggest that chance cannot be ruled out as a factor in explaining the results.

The corrections do not lessen the merit of this exceptional study, yet the bottom line of the randomized clinical trial may be this: We cannot be sure, but it seems as if GVC reduced symptoms in treatment-resistant obsessive-compulsive disorder. Two of 8 patients responded to treatment, and another patient developed delirium, likely as a consequence of GVC. Depression, anxiety, and quality of life were not improved.

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In Reply On behalf of our coauthors, we are grateful to Dr Baethge for his detailed reading of our study. He is correct that for the primary outcome, we inadvertently misclassified a patient (ATa3) as a responder when indeed that patient was a nonresponder. When preparing the table, we inadvertently included in our computation of treatment response a Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) score of 36 (the patient’s first measure) instead of 30 (the second measure). We defined the baseline Y-BOCS score for this study as the last value obtained before surgery. At that point, all patients knew they would receive the procedure.

Thus, instead of 3 of 8 patients responding in the treatment group at 12 months, 2 of 8 responded compared with 0 of 8 in the control group. Because of this error, the analysis has been redone and we have requested that JAMA Psychiatry retract and replace the original article.

In the corrected article, corrections have been made to the Abstract; Results, Discussion, and Conclusions sections of the text; Tables 2 and 3; Figure 2; and eFigure 3 and eTables 5, 6, and 7 in the Supplement. The article now concludes, “In this preliminary trial, patients with intractable OCD [obsessive-compulsive disorder] who underwent GVC [gamma ventral capsulotomy] may have benefitted more than those who underwent sham surgery although the difference did not reach statistical significance. Additional research is necessary to determine if GVC is better than deep-brain stimulation.”

We regret the errors caused by this misclassification as well as the confusion it caused for JAMA Psychiatry, readers, and potentially patients.

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