of the endometrioma wall (5), in which endometriosis and fibrosis were found in 54% of the specimens, whereas endometriosis, fibrosis, and ovarian tissue were present in 46% of the specimens. Scurry et al. (4) try to link their finding to existing pathogenetic theories, which we did not do (1, 5).

Also, Hachisuga and Kawarabayashi (6) were not able to "histopathologically demonstrate the secondary involvement of functional ovarian cysts in the process of endometriosis," because "no ovarian endometriotic cyst showed both luteinized and epithelial lining" in their series of 73 ovarian endometriomas.

As to the depth of penetration of endometriosis in the cyst wall, we report a mean maximal depth of 0.6 mm. However, this is not the thickness of the entire cyst wall, which also contains fibrosis and ovarian tissue, if present. The mean cyst wall thickness, in fact, is reported as 1.4 mm (1). The cyst wall gets thicker (2.4 mm in our study) where the endometriosis penetrates more deeply into the tissue underneath. Therefore, the figure of 0.6 mm does not necessarily contrast with the figure of 4 to 5 mm that was given by Nezhat et al. in their letter.

In conclusion, it may well be that our study included only type I and late type II endometriomas, as described originally by Nezhat et al. (3) and as subsequently modified with the simplification into two types instead of three. However, if late-stage type II means that a type II endometrioma has lost the initial functional lining, the existence of a true type II, according to the existing literature on the histological analysis of endometriomas, still is unproven. In our study, anyhow, we did not intend to make endometriomas fall into any preset categories or to fit our findings in any existing pathogenetic theory. We intended simply to describe the histology of the endometrioma wall that was excised with the stripping technique.

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Perspectives on oocyte research

To the Editor:

In response to the findings presented by Moayeri et al. (1), we oblige the authors' request for a larger series of data to confirm their conclusions.

Our analysis of 922 clinical pregnancies resulting from blastocyst transfers over a 56-month period revealed a similar decline in the monozygotic (MZ) twinning rate with time. A significant drop in the rate of MZ twinning resulting from blastocyst transfers was demonstrated when comparing the 29-month period before 2005 (18/412, 4.4%) with the subsequent 27 months beginning in 2005 (10/510, 2.0%; $P<.05$). An even larger decline was found in the MZ rate after, specifically, donor oocyte pregnancies during the same time periods (6/99 [6.1%] vs. 3/122 [2.5%]), but this difference did not reach statistical significance because of the limited sample size.

Our results occurred over a shorter time period, <5 years, with a larger number of cases (922), lending great support for the authors' hypothesis of an experience factor. There were no obvious changes in laboratory technique, protocols, or culture media during the experimental period, supporting the findings that experience, including very highly controlled culture conditions, may play a larger role in the MZ rate drop. We also analyzed all the MZ twinning cases at our center over the entire study period and could not find a significant correlation with mean oocyte age, method of fertilization (intracytoplasmic sperm injection vs. IVF), use of assisted hatching, or media lot number used for embryo culture to day 3 or blastocyst.

Our supportive finding that the rate of MZ twinning declines with time and experience also is difficult to explain. Although we do not have a cause to directly relate to the decline, we have eliminated a large number of proposed causative factors in our lab, including ICSI and assisted hatching. General improvements in blastocyst culture systems (media-manufacturer related), the grading of blastocysts, and the process of embryo selection for transfers all may have improved over the past 5 years, and that learning curve may help account for our findings. Analysis of other potential causes

for these rare pregnancies, such as epigenetic regulation changes caused by in vitro culture, or possible growth factors found, or lacking, in media or protein supplements, will need to be investigated to determine why IVF increases the risk of these types of pregnancies over natural conception. We believe that our findings, involving more cases in a shorter time period, verify these authors' published results.

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Reply of the Authors:

We appreciate the letter that Barritt et al. wrote in response to our study (1). We found a significant decline in the incidence of monozygotic twinning with blastocyst transfer when we compared our recent experience with our early experience with this modality.

It is reassuring to us to learn that a similar trend was observed by Barritt et al. in New York, among a large number of pregnancies after blastocyst transfer. We are grateful to those authors for obliging our request for other investigators to share their recent experience with blastocyst transfer to help confirm our findings. Their data provide valuable information to the IVF community by verifying our results in their large series.

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Erratum

In the article "Assisted reproductive technology in the United States: 2001 results generated from the American Society for Reproductive Medicine/Society for Assisted Reproductive Technology registry" (June 2007;87(6):1253–66), the "Participants" section of the abstract should say that data were collated after November 2002 so that outcomes of all pregnancies would be known. A corrected version appears online.