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Antidepressants and Pregnancy: *Now* What Should We Tell Our Patients?

Are antidepressants hazardous to a neonate's health? The FDA implied as much this past summer, when they issued a new precaution and required all antidepressant makers to include something like this text (for Effexor) in their package inserts:

"Neonates exposed to SSRIs or SNRIs late in the third trimester have developed complications requiring prolonged hospitalization, respiratory support, and tube feeding. Such complications can arise immediately upon delivery. Reported clinical findings have included respiratory distress, cyanosis, apnea, seizures, temperature instability, feeding difficulty, vomiting, hypoglycemia, hypotonia, hypertonia, hyperreflexia, tremor, jitteriness, irritability, and constant crying. These features are consistent with either a direct toxic effect of SSRIs and SNRIs or, possibly, a drug discontinuation syndrome. It should be noted that,

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Focus of the Month:
Pregnancy and Psychiatric Treatment

Treating Postpartum Depression: A Look at the Evidence

What is postpartum depression (PPD), and why does it occur? PPD is generally defined as major depressive symptoms occurring within three months of childbirth. Don't confuse PPD with the very common postpartum blues, a phenomenon that occurs in over half of women who give birth, peaks about four days after delivery, and fully remits by 10 days postpartum (*N Engl J Med* 2002; 347:194-199).

About 13% of women experience depression after giving birth, which is significantly higher than the overall one-year incidence of depression in women. Women with a prior history of PPD have about a 30% risk of depression after subsequent pregnancies (*Acta Psychiatr Scand* 1985; 71:451-457).

Clearly, the postpartum period is a significant, specific trigger of depression in women. But is it a psychosocial or hormonal trigger? Most of us explain PPD to patients as a hormonal problem, but is there any solid evidence that this is true? Fortunately, there is. Sixteen women (eight with a history of PPD, eight with no such history) were enrolled in a study in which they were given leuprolide, an anti-hormone that causes estrogen and progesterone to plummet to post-partum levels. Five of the eight women with PPD histories developed depression, while none of the other women did. This implies that certain women are specifically vulnerable to the hormonal changes accompanying childbirth (*Am J Psychiatry* 2000; 157:924-30).

Do antidepressants work in PPD?

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Learning objectives for this issue: 1. Explain the degree of risk associated with SSRI use in pregnancy. 2. Identify effective treatments for postpartum depression. 3. Cite specific drug interactions and side effects involved with the use of anticonvulsants in women.

This CME activity is intended for psychiatrists, psychiatric nurses, and other health care professionals with an interest in the diagnosis and treatment of psychiatric disorders.

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in some cases, the clinical picture is consistent with serotonin syndrome. When treating a pregnant woman with Effexor XR during the third trimester, the physician should carefully consider the potential risks and benefits of treatment."

This is the stuff your patients will be reading, and many pregnant women will be discontinuing their antidepressants faster than you can say, "But wait--let's look at the data!" We all need to understand the evidence supporting this warning in order to give good advice to our patients.

How dangerous are SSRIs and SNRIs during pregnancy? How good is the evidence? And how can we best communicate the degree of risk to patients?

Risk of major malformations. Until a week before this issue went to press, this sentence was going to read: "The good news is that there is virtually no increased risk of major malformations with SSRI use." However, GlaxoSmithKline just sent a letter to healthcare professionals announcing the resuts of a retrospective study of malformation risk in women taking Paxil during the first trimester. Paxil incurred double the risk of malformations compared with other antidepressants – this amounted to a 4% risk for Paxil vs. a 2% risk for other ADs. The baseline rate is malformations is generally considered to be 1-3%.

It's unclear what to make of this data, since at least 15 controlled studies have been published assessing the effects on the fetus of exposure to SSRIs, and none have shown a higher than baseline incidence of major malformations. A recent review reported that the drug with the most safety data is Prozac, with over 1600 Prozac-exposed women showing no increased risk of teratogenicity; numbers for the other common SSRIs are: Zoloft, 269; Paxil, 330; Celexa, 397 (*J Clin Psychopharm* 2005; 25:59-72). Given the recent announcement from GSK, you'd be wise to steer women away from Paxil and toward Prozac if they are considering an AD during pregancy.

What about Wellbutrin? While a recent study of 136 women showed no major malformations, that same study did report a relatively high rate of spontaneous abortions (*Am J Ob Gyn* 2005; 192:932-936). However, since depression alone can increase the risk of miscarriage, it is not clear whether Wellbutrin was responsible.

Neonatal Complications and Third Trimester Use

Recently, an excellent literature review (*JAMA* 2005; 293:2372-2383) focused on the evidence linking third trimester SSRI use with neonatal symptoms. The authors systematically guide us through three different levels of evidence: case reports, case series, and controlled cohort studies.

Case Reports. Case reports make lousy statistics, but are great for painting a vivid picture of a syndrome, and the *JAMA* authors identified 18 published case reports of third trimester SSRI-related neonatal signs. The most common symptoms reported in these cases were tremors/jitteriness/shivering (present in 11 of the 18 cases), increased muscle tone (11), feeding/digestive disturbances (9), irritability/agitation (9), and respiratory distress (7). Interestingly, these clinical signs sound more like serotonin syndrome than like serotonin withdrawal, even though they are usually framed as signs of withdrawal. This is controversial because many of the symptoms of SSRI discontinuation are subjective, like dizziness and shock-like sensations, and you're not likely to find a neonate capable of describing feelings like that! Does it matter? It does, because some authorities advocate giving the baby a dose of Prozac at birth to ease discontinuation symptoms--precisely the wrong thing to do if the problem is serotonin overstimulation.

Case Series. Next up on the hierarchy of clinical evidence are published case series. Mostly, these have been derived from databases of adverse events reported by physicians to the FDA (in the U.S.) or the WHO (World Health Organization, in Europe). This spontaneous reporting makes for pretty poor statistics as well, both because adverse events tend to be under-reported (when was the last time *you* reported an adverse event to the FDA), and those that are reported are the most serious cases.

Nonetheless, the advantage here is that these case reports are entered into government computers to create huge databases including millions of case records. Researchers can use these databases to perform a new type of statistical maneuver called "data mining."

Recently the WHO adverse events database was mined for information on neonatal adverse effects of antidepressants, and the results were published (*Lancet* 2005; 365:482-487). The researchers used data-mining techniques to sift through 3 million records, looking for an association between adverse drug reactions (ADRs) in neonates and maternal SSRI use. The statistic used to report the strength of the association is the confusingly named "information component" (IC). An IC of 0 means that the number of ADRs reported in

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"Of course they do," you answer. And while most of the experts in the field would agree with you, you'd actually be on very shaky ground according to evidence-based psychiatry. Incredibly, only two placebo-controlled trials of antidepressants for PPD have been published. Both have the same first author, Katherine Wisner, and they yielded absolutely contradictory results.

In the first study, which was the larger of the two, non-depressed women who had previous histories of PPD were randomly assigned to receive either nortriptyline (titrated to a mean serum level of 83 ng/mL; the mean dose was not reported) or placebo. Women began treatment immediately postpartum and continued for 20 weeks. The results? Of 26 women on nortriptyline (NT), six (23%) suffered a recurrence of PPD, and of 25 women on placebo, six (24%) suffered recurrence. Thus, NT yielded no benefit in terms of preventing a recurrence of PPD (*J Clin Psychiatry* 2001; 62:82-86).

The second study had the same research design, but compared Zoloft (sertraline) rather than NT to placebo. There were far fewer subjects in this study, but the results were more heartening. Of 14 women on Zoloft (with the dose beginning at 25 mg QD for four days, increasing to 50 mg QD through week four, then 75 mg QD through week 17), only one had a recurrence (7%). Of eight women on placebo, there were four recurrences (50%). Despite the small number of subjects, this difference was statistically significant (*Am J Psychiatry* 2004; 161:1290-1292).

Why Zoloft should work better than NT isn't entirely clear. There was a briefly fashionable notion that women in general respond better to SSRIs than to tricyclics, based on a much publicized study in 2000 (*Am J Psychiatry* 2000; 157:1445-1452). However, a later data synthesis of nine studies showed no gender differences in response to Prozac or tricyclics (*Am J Psychiatry* 2002; 159:1848-1854), and a more recent meta-analysis of 30 randomized placebo-controlled trials that included men and women showed no overall gender differences in response to tricyclics (*Am J Psychiatry* 2004; 161:370-372).

The only other controlled study of antidepressants in PPD wasn't a clean placebo-controlled study but in some ways may be more useful. The investigators reasoned that in the real world, a woman with PPD will never be given an antidepressant without some therapy, if only a supportive chat with their PCP. Thus, rather than comparing Prozac alone with therapy, they randomly assigned 87 women with PPD to four different treatment arms: Prozac plus one cognitive behavior therapy (CBT) session, Prozac plus six CBT sessions, placebo plus one CBT session, placebo plus six CBT sessions. At 12 weeks, Prozac plus one session yielded similar benefits to placebo plus six sessions, implying that CBT may work as well as Prozac; Prozac plus six sessions didn't work any better than Prozac plus one (*BMJ* 1997; 314:932-936).

In a prior issue of *TCR* (Sept 2004), we reviewed another study endorsing psychotherapy for PPD, that time interpersonal therapy (IPT), which helped relieve symptoms of depression far better than no treatment at all in the waiting list control (*Arch Gen Psychiatry* 2000; 57:1039-1045).

In the end, no matter how inconclusive the evidence, we need to do what we can to treat a problem that can cause damage to both mother and infant, and this will usually involve a combination of SSRIs and psychotherapy. ❖

TCR VERDICT: *Zoloft, Prozac, IPT, CBT: They are all worth a try in postpartum depression.*

Q&A With the Expert

This Month's Expert: Victoria Hendrick, M.D., on Drug Interactions and Drug Side Effects Specific to Women

Associate Professor of Psychiatry
UCLA School of Medicine and Olive View-UCLA Medical Center

Dr. Hendrick has disclosed that she was/is a member of the speakers bureaus for GlaxoSmithKline and Pfizer Pharmaceuticals, and has given talks on the reproductive safety of Paxil (GlaxoSmithKline) and Zoloft (Pfizer). Because of these industry relationships, the editors of *The Carlat Report* have edited the interview in order to resolve any possible faculty conflicts of interest regarding this educational activity.

TCR: Dr. Hendrick, thanks for your return visit to the pages of *TCR*! I'm wondering whether, as a specialist in the psychiatric treatment of women, you can tell us about particular drug interactions that you feel we should be more aware of in treating women.

Dr. Hendrick: One of the really important interactions is with estrogen-containing birth control pills. The one that many people are aware of is with carbamazepine (Tegretol), which can induce the metabolism of hormones and thereby reduce the efficacy of the birth control pill, leading to the risk of an unwanted pregnancy.

TCR: And what are some of the other meds that can cause this interaction?

Dr. Hendrick: Oxcarbazepine (Trileptal), topiramate (Topamax), and St. John's Wort can all speed up the metabolism of the birth control pills. Unplanned pregnancy is a problem for many reasons, including the fact that we don't know much about the safety of the newer anti-epileptic drugs in pregnancy.

TCR: For women who are doing well on one of these inducing medications, are there any options short of changing contraceptive methods? Can the dose just be increased to account for the increased metabolism? Can lab tests determine whether the birth control pills are at effective levels?

Dr. Hendrick: No, you really can't rely on blood levels of hormones. At the very least, you can avoid low-dose estrogen formulations that contain only 20 micrograms of estrogen. Those are, of course, the highest risk formulations to be on if a woman is going to be on, say, Trileptal. So these women should take one of the higher-dose estrogen pills. But the safest strategy is to use a different form of contraception altogether.

TCR: Are there any other interactions to be aware of regarding the anticonvulsants?

Dr. Hendrick: Yes, it is important to be aware that, once women reach menopause, anticonvulsants can play the same havoc with hormone replacement therapy. If women are on hormone replacement therapy and they start carbamazepine or oxcarbazepine or topiramate, they might start noticing that they are getting a recurrence of hot flashes and night sweats because of induced metabolism, and they might need higher doses of hormone replacement.

"If women are on hormone replacement therapy and they start carbamazepine or oxcarbazepine or topiramate, they might start noticing that they are getting a recurrence of hot flashes and night sweats because of induced metabolism, and they might need higher doses of hormone replacement."
– Victoria Hendrick, M.D.

TCR: Are there any interaction concerns regarding Lamictal (lamotrigine)?

Dr. Hendrick: Yes, and this is a different type of interaction. The estrogen in estrogen-containing oral contraceptives can increase the metabolism of the Lamictal, and Lamictal levels can be reduced very dramatically – by 40-60%. This interaction can pose particular problems while women are starting the medication, because Lamictal must be gradually titrated to minimize the risk of Stevens Johnson syndrome. If women are starting or coming off the birth control pills, it is important for clinicians to be aware that blood levels can vary quite widely. A particular danger would be that a women discontinues the Pill midway through a titration, which could result in a dramatic increase in serum Lamictal levels.

TCR: So what are your recommendations?

Dr. Hendrick: What I recommend to patients is that during the period of titration on Lamictal, they make no changes to their contraception regimen. If they are going to get on the birth control pill, they should do it before they start the

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Q & A With the Expert

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Lamictal, and if they are going to come off it, they should do that before they start the Lamictal. I recommend that they don't make the change during that six-month titration period.

TCR: Moving now to the issue of medication side effects, are there any important ones that are specific to women?

Dr. Hendrick: One of the most important ones, and it has been a controversial one, is the relationship between polycystic ovary syndrome (PCO) and valproate (Depakote). A majority of the studies have found evidence that there is a connection between use of valproate and an increased incidence of irregular menses, elevated androgen levels, and polycystic ovary syndrome. So even though the jury is still out on this, I think it is still worthwhile for clinicians who are treating young women on valproate to be vigilant for PCO-like changes.

TCR: Specifically, what should we be looking for?

Dr. Hendrick: Briefly, PCO is defined by chronic anovulation and evidence of elevated androgen. Women will develop irregular menses, hirsutism and acne. On labs, androgen levels will be elevated, including testosterone, androstenedione, and DHEA. Readers should know that there have been some inconsistencies in the literature, but over the last decade there have been more and more studies endorsing this valproate-PCO connection.

TCR: And what is the presumed pathophysiological mechanism?

Dr. Hendrick: Valproate appears to lead to both weight gain and insulin resistance, and elevated insulin levels inhibit the enzymes that transform testosterone into estrogen. The elevated testosterone then initiates the cycle of hormonal dysregulation that leads to PCO.

TCR: What are your specific clinical recommendations?

Dr. Hendrick: I recommend that clinicians ask women on valproate to keep a menstrual diary, in order to see if there is any irregularity in the menstrual pattern after beginning the medication. We should also ask about facial hair and temporal pattern balding, both of which are evidence of hirsutism. If necessary, order androgen levels, making sure to include all three androgens on the lab slip: testosterone, androstenedione, and DHEA.

TCR: We have also heard a lot of concerns about Depakote as a teratogen as well.

Dr. Hendrick: Yes, and there has been some new literature on this. It has been known for a while that there is a link between Depakote and neural tube defects. The risk was quoted at about 1-3 percent, which is a pretty substantial risk. But now the risk has been raised to closer to 5-6 percent, so it looks like it is even more teratogenic than what was previously believed. The mechanism there seems to be the anti-folate properties of Depakote, since women need folate early in pregnancy for the formation of the embryo's neural tube. That is what makes Depakote so teratogenic, the anti-folate properties, and even giving women folate supplementation doesn't seem to be sufficient to prevent this risk. So the best thing is to avoid Depakote in women of reproductive age who are not using contraception reliably.

TCR: Any other side effects that have come up specific to women?

Dr. Hendrick: The other side effect that you see mostly in the neurologic literature but that I think is worth pointing out for us in psychiatry is the effect that anticonvulsant drugs like carbamazepine (Tegretol) can have on vitamin D metabolism. Women, especially as they get to be menopausal, can be at greater risk for bone loss if they are on medication that speeds up the metabolism of vitamin D, and carbamazepine is one of these medications. This could be a risk for men as well, but menopausal women are more vulnerable to osteoporosis and they might not be ideal candidates to be on a medication like carbamazepine.

TCR: Do both oxcarbazepine (Trileptal) and topiramate (Topamax) also induce vitamin D metabolism?

Dr. Hendrick: Yes.

TCR: Since we have a bit of space left, let's change tracks. Are there any new studies relevant to medications and pregnancy that clinicians should be aware of?

Dr. Hendrick: There was a paper that came out a few weeks ago on antipsychotic medication which is real boon to the field because there has been very little on antipsychotic medications (*J Clin Psychiatry* 2005; 66:444-9). It covered Zyprexa (olanzapine), Risperdal (risperidone) and Seroquel (quetiapine), so we finally have some published data on some of these newer antipsychotic agents. It showed that there were no adverse outcomes linked with any of the newer antipsychotic agents. Until that came out, most of the data we had was from the Eli Lilly registry on Zyprexa. But now we have about 50 exposures in this paper on Risperdal , which is not a ton of data, but it is enough to allow me several options in a patient who has a need for antipsychotic medication in pregnancy.

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association with a given drug is no higher than what you'd expect. A positive IC means the side effect is disproportionately common, while a negative IC means it's unusually *un*common.

You can probably guess the punch line. The ICs of SSRIs and Effexor were all positive for neonatal symptoms, including neonatal convulsions. Most cases were with Paxil, which also had the highest IC, leading the authors to conclude: "Thus paroxetine should not be used in pregnancy or, if used, should be given at the lowest effective dose."

Cohort Studies. In cohort studies, groups of pregnant women who have been prescribed SSRIs are enrolled, and are compared with control groups. In many of the studies reviewed, the control group consisted of women who discontinued SSRIs after the first trimester, making them ideal studies for assessing specific dangers of *third* trimester exposure. Don't confuse these studies with "placebo-controlled clinical trials," in which patients are randomly assigned to treatment or placebo. Such studies of pregnant women would never win approval from research ethics panels, meaning that cohort studies represent the best quality evidence we'll ever see.

The *JAMA* researchers located nine good quality cohort studies, but chose to focus on five that were particularly good in that they systematically defined and measured a specific neonatal SSRI syndrome. They crunched the numbers of these five studies and came up with an overall estimate of the risk of neonatal complications due to late SSRI exposure.

Their summary relative risk is easy to remember: 3.0. This means that babies are three times more likely to have neonatal complications if exposed to third trimester SSRI than with early or no exposure. But this is probably not the best way to describe it to your patients.

Why not? Because "relative risk" can be a misleading statistic. You've certainly seen the term bandied about quite a bit in the literature, because it's cleaner and quicker than discussing the raw numbers. But if a bad event has a large relative risk, but the baseline risk is very small, then the actual absolute risk of something occurring is still quite small. With regard to neonatal SSRI syndrome, though, both the relative and absolute risks are actually pretty substantial, and providing your patients with both quantities is informative.

Numbers Used to Calculate Absolute Risk as Reported in *JAMA* Article

Study	Relative Risks of Late SSRI Exposure	Absolute Risks	
		3rd Trim. SSRI	Early or No SSRI
Costei	3.6	12/55 = 22%	3/54 = 6%
Oberlander	3.4	14/46 = 30.4%	2/23 = 9%
Chambers	3.5	23/73 = 31.5%	9/101 = 9%
Cohen	3.3	16/53 = 30.2%	1/11 = 9%
Averages	3.4	65/227 = 29%	15/189 = 8%

The table above lists the numbers used to calculate the absolute risks reported in the *JAMA* article. The relative risk is easy to calculate: just divide the absolute risk of neonatal complications in the third trimester exposure group by the absolute risk in the early or no exposure group. (You'll notice we included only four studies, because the fifth study used a very different methodology that makes it difficult to compare with the others.

For this reason, our number for overall risk is slightly higher than the *JAMA* number.)

The overall relative risk of complications is 3.4 – a pretty intimidating number if related simply as a 3.4-fold higher risk of complications. But the absolute risk is 29%, meaning that out of 100 babies exposed to third trimester SSRIs, about 29 will have neonatal complications; out of 100 babies with either early or no SSRI exposure, 8 will have similar symptoms. Framed more positively, 71 out of every 100 late-SSRI babies will have no complications whatsoever.

Suddenly, the numbers don't seem quite as awful, particularly when you consider that all of these babies recovered fully, and that not a single baby in any of these studies had severe or life-threatening complications. Most of the affected babies were admitted to the NICU for a day or two and then sent home with mom.

Ultimately, the decision is your patient's. Providing her with the right numbers will make her job a little bit easier. ❖

TCR VERDICT: *SSRIs in pregnancy: the numbers tell the story*

CME Post-Test

To earn CME credit, you must read the articles and complete the quiz below, answering at least four of the questions correctly. Mail a photocopy or fax the completed page to **Wolters Kluwer Health, Office of Continuing Education, 530 Walnut Street, 8th Floor East, Philadelphia, PA 19106**; **fax: (215) 521-8637**. **For customer service, please call (215) 521-8635.** Only the first entry will be considered for credit and must be received by WKH by September 30, 2006. Acknowledgment will be sent to you within 6 to 8 weeks of participation.

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Please identify your answer by placing a check or "X" mark in the box accompanying the appropriate letter.

1. Regarding SSRIs in pregnancy:
- [ ] a. They may cause cleft palate but no other malformations.
- [ ] b. They are only harmful at high doses.
- [ ] c. Paxil may be linked to a higher rate of congenital anomalies than other SSRIs.
- [ ] d. They are contraindicated by the FDA for use during pregnancy.

2. Third trimester SSRI exposure causes:
- [ ] a. A three-fold higher incidence of serious neonatal complications.
- [ ] b. A 29% risk of non-critical complications, vs. an 8% risk otherwise.
- [ ] c. Seizures in 30% of infants.
- [ ] d. No measurable effects in 97% of infants.

3. Several SSRIs show unequivocal effectiveness for postpartum depression.
[ ] a. True [ ] b. False

4. Lamictal interacts with birth control pills by:
- [ ] a. Inducing their metabolism, rendering them ineffective.
- [ ] b. Inhibiting their metabolism, leading to menstrual irregularities.
- [ ] c. Lamictal does not interact with birth control pills.
- [ ] d. Birth control pills induce Lamictal's metabolism, decreasing levels dramatically.

5. According to Dr. Hendrick, Depakote is unlikely to be related to polycystic ovary syndrome.
[ ] a. True [ ] b. False

Keep it Coming!
We appreciate your feedback. If you have any suggestions on future topics, improvements to the website, or anything else, please email Dr. Carlat through our website at www.TheCarlatReport.com, or call him at 866-348-9279.

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1. Did the content of this activity meet the stated learning objectives? [ ] Yes [ ] No
2. On a scale of 1 to 5, with 5 being the highest, how do you rank the overall quality of this educational activity?
   [ ] 5 [ ] 4 [ ] 3 [ ] 2 [ ] 1
3. As a result of meeting the learning objectives of this educational activity, will you be changing your practice behavior in a manner that improves your patient care? Please explain.
   [ ] Yes _____
   [ ] No _____
4. Did you perceive any evidence of bias for or against any commercial products? Please explain.
   [ ] Yes _____
   [ ] No _____
5. How long did it take you to complete this CME activity? ___ hour(s) ___ minutes
6. Please state one or two topics that you would like to see addressed in future issues. _____

Tales from the History of Psychiatry

Tom Cruise versus Brooke Shields: A Strange Chapter in Psychiatry

"You can use vitamins to help a woman through those things," said Tom Cruise, denigrating Brooke Shields's use of an SSRI to treat her postpartum depression, which she detailed in her recent book *Down Came The Rain: My Journey Through Postpartum Depression.* Cruise's vocal antipathy towards psychiatry correlates with his high-profile association with the Church of Scientology, founded in the fifties by science-fiction writer L. Ron Hubbard. Hubbard first set out his odd ideas about psychological treatment ("auditing" to clear "engrams" in the "thetan") in *Dianetics* (1950), and, in the face of opposition from the American Psychological Association, relabeled his form of psychotherapy a philosophy (Scientology, 1952) and then a religion (the Church of Scientology, 1953) that controversially received nonprofit status from the IRS in 1993. Although many of Scientology's claims are laughable (it holds psychiatry responsible for World War I, the rise of Hitler and Stalin, the decline in education standards in the United States, the wars in Bosnia and Kosovo, and even the September 11th attacks), it has had a few frightening near-successes in curtailing the role of mental health professionals. Its anti-psychiatry arm, the benign-sounding Citizens Commission for Human Rights, was able to push a bill through the Utah state legislature (ultimately vetoed by the Governor) that labeled it a crime for schoolteachers to suggest to parents that their child get a psychological assessment. Similar bills have been filed in New Hampshire. With the renewed focus on Scientology raised by the Brooke Shields/Tom Cruise conflict over postpartum depression, it may be worth reminding ourselves of the potential for distorted perceptions of our work.

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This Month's Focus:
Pregnancy and Psychiatric Treatment

Next Month in *The Carlat Report*: Genetics and Psychiatry, with the latest on the genetics of psychiatric disorders, genetic counseling, the utility of P450 testing, and an interview with Samuel Barondes, M.D., of UCSF School of Medicine.