Letters continued from p. 195

Sessions "every week or two," as described in your article, are already somewhat diluted; with a follow-up frequency of once a month, one would expect very little. The finding that 50 percent of the patients in the latter condition were depression-free is thus astounding; one wonders whether even better effects would have been seen with sessions every other week.

A third problem is the implication that blood-level monitoring, "presumably including . . . fluoxetine (Prozac)," prevents suicide. For antidepressants where blood level bears some relation to clinical effects (and fluoxetine so far is not one of these), blood levels can help a practitioner ensure that the patient has had an adequate trial of medication. But not all patients respond to the first medication tried, even at adequate levels, and those who do may still be at risk for suicide as their energy levels increase but they still have periods of hopelessness.

Finally, the presentation of placebo studies as the ultimate in "scientific" double-blind studies raises an issue seldom addressed. Because the side effects of antidepressants (especially imipramine) are quite obvious even when tolerable, no person monitoring such patients can remain ignorant of the condition of treatment, and I suspect that few of the patients themselves are fooled. Until researchers utilize a placebo pill with very similar side effects, knowledge of treatment or lack of treatment is bound to confound the results of such studies.

Although there are still "medicine-only" and "psychotherapy-only" extremists, most practitioners today acknowledge that both medication and psychotherapy have a place in the treatment of depression, as has been borne out in studies of depression over the last several years at the National Institute of Mental Health.

To imply that the field is a battle between biochemical treatments and psychoanalysis is not only a distortion but an anachronism.

Marilyn L. Kohl
Clinical Psychologist
University of Illinois
Urbana, Ill.

Most clinicians do use both drugs and psychotherapy, as noted in the article. But take a look at the letters to the editor in the January AMERICAN JOURNAL OF PSYCHIATRY, *responding to debate over the Osheroff case, if you think the split between biochemically and psychoanalytically oriented clinicians is merely an anachronism.*
— B. Bower

I was very surprised to read that the National Task Force on Women and Depression did not find evidence to support a biological predisposition to depression in women.

Prozac, an extremely successful antidepressant, is a serotonin agonist, suggesting that disturbances of this potent neurotransmitter may be involved in severe depression. In recent research on migraine patients, it has become increasingly clear that migraine episodes may also involve serotonin. Given that 80 percent of migraine patients are women, it comes as no surprise to me that twice as many women as men suffer from depression. If serotonin is involved in depression, as it is in migraine, shouldn't we have expected this type of gender distribution?

Ann Molnar
Dobs Ferry, N.Y.

Heart attack hypotheses

As a social scientist pondering the difference in mortality between men and women following hospitalization for a heart attack ("The Safer Sex?" SN: 1/19/91, p.40), I find one hypothesis to be glaringly obvious and easily testable: that marital status and gender-role performance account for a substantial amount of the mortality difference.

I would hypothesize that the men are far more likely to be married than the women (more women in the age groups studied are widowed) and that the married women recovering from a heart attack do more physically and emotionally taxing work than the recovering men, the underlying hypothesis being that women are more likely to protect their spouses from physical and emotional stress than are men.

Judith Lempert Green
Psychologist
Ann Arbor, Mich.

Is it not conceivable that while women's coronary arteries were protected by high estrogen levels, men were forming collateral circulation within the substance of the heart muscle? Thus, when a coronary artery was closed off, women's hearts were not cushioned and the attack was worse.

David Goldstein
New York, N.Y.

News of the week continued from p. 199

Air pollution: A respiratory hue and cry

High levels of air pollution may foster respiratory symptoms in otherwise healthy individuals, a new analysis suggests. An even more provocative study indicates that air pollution, at least in Los Angeles, may begin permanently "deranging" the lung's cellular architecture by the time a person reaches age 14.

Environmentalists say both studies underscore the need for stepped-up efforts to bring areas with substandard air quality into compliance with the Clean Air Act. Researchers presented the new findings this week in Arlington, Va., at the annual conference of the Society for Occupational and Environmental Health.

Bart Ostro and his co-workers reexamined data collected 12 years ago in the north-central Los Angeles area, looking for associations between respiratory health and local measurements of air pollutants. The team focused on 320 generally healthy, nonsmoking men and women who had kept daily logs of their respiratory symptoms over a six-month period as part of another study.

A preliminary analysis indicates that runny noses, sinusitis, sore throats, head colds and other upper-respiratory symptoms arose most often on the haziest days or on days with the highest peaks in smog-ozone, reports Ostro, an epidemiologist with the California Department of Health Services in Berkeley. The bad smog episodes also coincided with more lower-respiratory symptoms, such as coughs, phlegm, wheezing and chest colds — but only among participants whose homes lacked air conditioners, which can limit indoor ozone levels. In addition, the researchers uncovered hints that when sulfates reached high levels in the city's air, lower-respiratory symptoms emerged the following day.

Daily one-hour peaks in ozone were high throughout the study period, averaging 0.1 parts per million and sometimes reaching 0.43 ppm — well above the federal limit of 0.12 ppm. For each 0.1 ppm elevation in peak ozone, participants logged "roughly a 10 percent increase in upper or lower respiratory symptoms, and increases of 30 percent in eye irritation," Ostro reports.

Russell P. Sherwin and his colleagues at the University of Southern California in Los Angeles detected surprising signs of "an ecologic problem at the cell level" in the lungs of 85 Los Angeles residents, aged 14 to 25, who had died in homicides or traffic accidents. The team's detailed cellular assays uncovered chronic bronchitis, inflamed bronchial submucosal glands and microscopic holes — subtle but "pretty severe" lung damage that would elude detection by most coroners, Sherwin says.

Some of these lung abnormalities probably reflect damage from smoking, drugs and previous respiratory diseases. However, Sherwin says, "we know ozone is involved [too]," because studies have shown that nonhuman primates develop identical lesions when exposed to ozone levels exceeding the federal standard. Parts of Los Angeles exceed the ozone standard "every other day," he notes.

Overall, 98 percent of the young victims showed some form of chronic bronchitis. In 55 percent of the cases, the researchers observed atrophy, or even some outright loss, of submucosal glands. Three-quarters of the youths also suffered centriacinar-region lung disease — a condition characterized by chronic inflammation, and often structural aberrations, in the smallest, terminal "twigs" of the bronchial tree.

Sherwin says such damage indicates that the deceased youths had "certainly [been] heading for a lot of trouble" with emphysema and perhaps early heart attacks. If their lungs turn out to be bellwethers of air pollution's effects on the population at large, "we're all heading for a lot of trouble," he warns. *— J. Raloff*