Marital Stress and Coronary Heart Disease

To the Editor: In their observational study, Dr Orth-Gomér and colleagues[1] reported an association between psychosocial adversity and poorer cardiovascular health. They found that among women with a history of coronary heart disease (CHD), the chances of a recurrent coronary event (cardiac death, recurrent symptoms, or a revascularization procedure) were 3 times higher in those who reported severe marital stress. If this relationship is causal, then it may be mediated through the psychoneuroendocrine mechanisms that the authors invoke and may even suggest that "specific preventive measures be tailored to the needs of women with CHD." The health policy implications of this latter conclusion are substantial. It is therefore important to consider alternative explanations for these intriguing findings.

First, it is well recognized that reporting bias (resulting from a tendency to overreport both marital stress and cardiac symptoms) can result in problems of interpretation of this relationship.[2] It would be interesting to see if the association between stress exposure and the most objective component of the composite outcome—cardiac death—was weaker than that with the other components.

Second, it is probable that the experience and reporting of marital stress are related to other social and demographic variables. For instance, if higher levels of marital stress are associated with social disadvantage, then an automatic, noncausal association between higher marital stress and poorer health will be generated since social disadvantage is associated with poorer health.[3] Adjustment for educational attainment might attenuate such an effect, but would be unlikely to abolish it. Again, it would be helpful if the authors described the social patterning of the exposures they studied. If marital stress was associated with social disadvantage in this population, then it seems likely that confounding by social position contributed to their findings. However, if levels of marital stress showed no social gradient or if the gradient was such that higher stress was associated with social advantage, then these findings would be difficult to attribute to confounding of this nature.

Third, many of the contributors to the authors' marital stress measure relate to sexual behavior. Higher levels of sexual activity are known to be associated with a lower risk of CHD, probably because this is an indicator of health status.[4] It is thus possible that marital stress is actually an index of health status and that the association between lower marital stress and better health reflects this.

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1. Orth-Gomér K, Wamala SP, Horsten M, Schenck-Gustafsson K, Scheiderman N, Mittleman MA. Marital stress worsens prognosis in women with coronary heart disease: the Stockholm Female Coronary Risk Study. *JAMA*. 2000;284:3008-3014.
2. Watson D, Pennebaker JW. Health complaints, stress and distress: exploring the central role of negative affectivity. *Psychol Rev*. 1989;96:234-254.
3. Davey Smith G, Harding S. Is control at work the key to socio-economic gradients in mortality? *Lancet*. 1997;350:1369-1370.
4. Davey Smith G, Frankel S, Yarnell J. Sex and death: are they related? findings from the Caerphilly Cohort Study. *BMJ*. 1997;315:1641-1644.

In Reply: Mr Macleod and Dr Davey Smith suggest that overreporting may have produced a spurious relationship between marital stress and the 5-year experience of recurrent cardiac events in women. If overreporting of stress were associated with a disposition to complain about symptoms and to be more persuasive toward the treating cardiologist, this may have led to a higher incidence of revascularization procedures, but would not affect "hard" end points like cardiac death.

In the Fem Cor Risk Study, we analyzed the impact of self-reported stress both from work and from marital relationships, first in a case-control comparison[1] and later in a 5-year follow-up of patients.[2] In both analyses, marital stress was a stronger predictor than work stress. In the longitudinal study, a composite end point that included revascularization procedures was used to obtain a large enough group for prognostic analyses. As the number of "hard" events during 5-year follow-up was small (N=14), results for both stressors were nonsignificant, but in the same direction as for composite end-point analyses (hazard ratio [HR]=1.7, 95% confidence interval [CI], 0.7-6.1 for marital stress; HR=1.4, 95% CI, 0.5-4.0 for work stress). Extended follow-up analyses are under way to further examine this relationship.

Macleod and Davey Smith further suggest that sexual problems may reflect severity of CHD rather than marital stress. In separate item analysis, the prognostic impact of these items was statistically nonsignificant (HR=1.2, 95% CI, 0.7-2.0). When questions about sexual relations were excluded from the Stock-

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Letters Section Editors: Stephen J. Lurie, MD, PhD, Senior Editor; Jody W. Zylke, MD, Contributing Editor.

holm Marital Stress Scale, this did not substantially change the results.

As almost all Swedish women are employed outside the home, their social patterning is dependent on both their education and occupation. Socially disadvantaged women in nonprofessional jobs more often reported severe marital stress (53%) compared to women with professional jobs (36%) ($P=.004$). Additionally, among women in nonprofessional jobs, severe marital stress was associated with a higher increase in risk of recurrences.[3] Overall multivariate adjustment, including both education and occupation, however, only marginally decreased the risk ratios.

Potential health policy implications of these findings are being considered in Sweden, in particular for socially disadvantaged women. In a 1-year structured psychosocial intervention program, marital and work stress management, along with lifestyle intervention, are combined with improved access to resources from community and neighborhood. Whether this method prevents recurrences of heart disease and whether it can be used on a national level remain to be evaluated.

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1. Orth-Gomér K, Moser V, Blom M, Wamala SP, Schenck-Gustafsson K. Kvinnostress kartläggs: hjärtsjudkom hos Stockholmskvinnor orsakas i lika hög grad av stress i familjen som i arbetet [Family and work stress, as markers for coronary disease risk in Stockholm women]. *Läkartidningen.* 1997;94:632-638.
2. Orth-Gomér K, Wamala SP, Horsten M, Schenck-Gustafsson K, Scheiderman N, Mittleman MA. Marital stress worsens prognosis in women with coronary heart disease: the Stockholm Female Coronary Risk Study. *JAMA.* 2000;284:3008-3014.
3. Wamala SP. Stora sociala skillnader bakom kvinnors risk för kranskälssjukdom: okvalificerat jobb och slitningar i familjen avgörande faktorer [Social inequalities in coronary disease risk among women: low occupational status and family strain are crucial factors]. *Läkartidningen.* 2001;98:177-181.

Training in Psychiatric Consultation

To the Editor: From his review,[1] it is clear that Dr Viederman did not enjoy my book, *Sigmundoscopy*.[2] This is his prerogative. He also appears not to favor the use of levity as an enhancement to medical education, which is also his prerogative. Residents, for whom this book is written, need a framework or "road map" so that they can master the basics and feel confident in developing a style that suits them. I recognize that a book the size and scope of *Sigmundoscopy* has its limitations and I am grateful for constructive criticism that will strengthen the text for future editions. Unfortunately, I cannot find anything in Viederman's review to assist me with this task.

He fails to describe key sections from chapters, as well an entire chapter, which give a detailed analysis of the recommendations most likely to be implemented by consultees. Furthermore, *Sigmundoscopy* was intended to be a supplemental textbook. At 182 pages, my book cannot cover all of the areas to which Viederman makes reference. Instead, it aims to answer the question that every resident has when starting a consultation psychiatry rotation: "How do I conduct a consult?" A comprehensive outline is clearly needed. There are only a handful of articles describing how consultations should be carried out. The *Textbook of Consultation Liaison Psychiatry*,[3] widely considered a "gold standard" reference text, devotes only 5 of 1171 pages to the topic; the *Comprehensive Textbook of Psychiatry*[4] does not even address the topic.

Viederman criticizes my example of a comprehensive consultation note by saying it is too long and unlikely to be read. Is he suggesting that important psychiatric information should be neither obtained nor documented because a consultee may not read the entire note? While the consult note provided in the book is admittedly an ideal one, as a psychiatric educator I cannot provide an example that encourages trainees to take short cuts in their interviews or documentation. He also criticizes my explanation of the term *splitting*. What I describe is the situation in which a psychiatric consultant, who may be able to spend more time talking with patients than the referring source, is seen as the "good" clinician and the less available consultee as the "bad" clinician. I realize there are other uses of the term *splitting*, but I am using it in the above context, which is consistent with standard usage, such as that found in the *Synopsis of Psychiatry*.[5]

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1. Viederman M, reviewer. *JAMA.* 2000;284:1580. Review of: Robinson DJ. *Sigmundoscopy: Medical-Psychiatric Consultation-Liaison—The Bases*.
2. Robinson DJ. *Sigmundoscopy: Medical-Psychiatric Consultation-Liaison—The Bases*. Port Huron, Mich: Rapid Psychler Press; 1999.
3. Rundell JR, Wise MG, eds. *Textbook of Consultation Liaison Psychiatry*. Washington, DC: American Psychiatric Press Inc; 1996:13-17.
4. Sadock BJ, Sadock VA, eds. *Comprehensive Textbook of Psychiatry*. 7th ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2000.
5. Kaplan HI, Sadock BJ, eds. *Synopsis of Psychiatry*. 8th ed. Baltimore, Md: Williams & Wilkins; 1998:780.

In Reply: My criticism of Dr Robinson's book reflects my view that it represents exactly what can be wrong in psychiatry and unfortunately tends to pervade much of psychiatric teaching. It presents the approach to the patient in a shorthand, stereotyped, technical way that misses the essence of what psychiatry at its best is all about. Psychiatry should be as much an education about human behavior as a training in procedural skills. I have had wide experience teaching medical students and house staff in psychiatry, as well as medicine, and what fascinates them still are the nuances of human behavior. Admittedly it takes years of experience to develop a personal style and a personal view. Yet, it is of critical importance that what Robinson calls "the neophyte consultant," or the neophyte psychiatrist for that matter, know that there is something in the interaction with a patient that is not only a source of fascination, but the only real vehicle of establishing contact with a patient. This is the task of the physician and does not contradict the need for basic knowledge. No attention is paid to this in *Sigmundoscopy*. For a psychiatrist to ignore this feature is to risk sterility for the entire enterprise.

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RESEARCH LETTERS

Linezolid and Reversible Myelosuppression

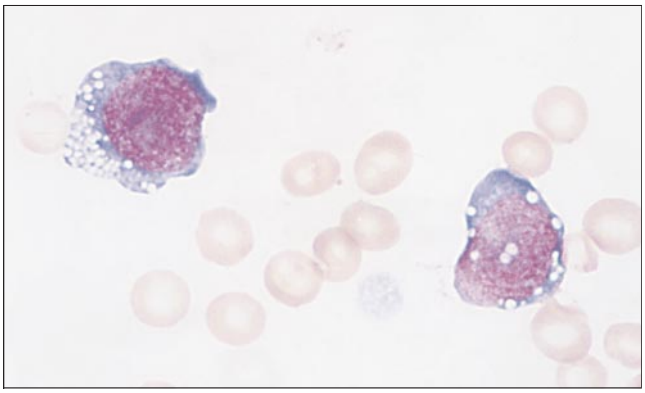
To the Editor: Linezolid is the first oxazolidinone antibiotic with labeling approved to treat methicillin-resistant *Staphylococcus aureus* (MRSA) infection. We report 3 cases of myelosuppression with red cell hypoplasia that occurred following therapy with linezolid. Although reversible thrombocytopenia has been reported in patients receiving more than 2 weeks of therapy,[1] we found additional bone marrow changes that appear similar to those seen in reversible chloramphenicol toxicity.

Report of Cases. The first patient was a 70-year-old man who received linezolid, 600 mg twice daily for 4 months, for an MRSA infection in a femoral-popliteal Gore-Tex graft. During this period, the platelet count decreased from 215×10^3/µL to 60×10^3/µL, and hemoglobin decreased from 14.3 g/dL (143 g/L) to 7.0 g/dL (70 g/L), with a reticulocyte count of 0%. Vitamin B_{12} and folate levels were normal, serum iron was 158 µg/dL (28.3 µmol/L) with a total iron binding capacity of 166 µg/dL (29.7 µmol/L), and iron saturation was 95%. Examination of the bone marrow demonstrated 30% hematopoietic tissue, increased iron stores, megaloblastoid white blood cell maturation, abundant megakaryocytes, and erythroid aplasia (myeloid/erythroid ratio = 174:1) with vacuolated erythroblasts (FIGURE). Ten days after discontinuing linezolid, platelet counts and hemoglobin levels returned to normal, the iron saturation fell to 25%, and the reticulocyte count increased to 5.8%.

The second patient was a 43-year-old woman who received linezolid, 600 mg twice a day for 6 weeks, for a chronic MRSA infection of a facial sinus. During this period, the platelet count decreased from 399×10^3/µL to 206×10^3/µL, and hemoglobin concentration decreased from 14.1 g/dL (141 g/L) to 12.8 g/dL (128 g/L), with a reticulocyte count of 0%. Parvovirus B-19 IgM and IgG titers were undetectable. Vitamin B_{12} and folate levels were normal, and serum iron was 214 µg/dL (38.3 µmol/L). Total iron binding capacity was 286 µg/dL (51.2 µmol/L) with an iron saturation of 74%. Linezolid was discontinued at this point, and 5 days later a bone marrow biopsy specimen showed decreased iron stores and rare vacuolated erythroblasts. One week later, platelet counts and hemoglobin levels returned to normal, the iron saturation decreased to 9%, and the reticulocyte count increased to 2.4%.

The third patient was a 61-year-old woman with type 2 diabetes who received linezolid, 600 mg twice daily, for MRSA metatarsal osteomyelitis. During 2 weeks, reticulocytes declined from 1.4% to 0% and iron saturation increased from 22% to 65%. The drug was discontinued for 2 weeks, then, with the patient's consent, continued for 4 more weeks. During that time, the reticulocytes decreased again from 4.5% to 0%, platelets from 294×10^3/µL to 125×10^3/µL, hemoglobin from 9.9 g/dL (99 g/L) to 8.0 g/dL (80 g/L), and iron saturation increased from 10% to 96%. Similarly to the other 2 patients, all values returned to normal levels after linezolid discontinuation.

Figure. Vacuolated Erythroblasts in the Bone Marrow of Patient 1 After Receiving Linezolid for 4 Months



Pathology performed with Wright stain (original magnification ×1000).

Comment. These 3 cases illustrate features strikingly similar to those of the reversible form of chloramphenicol toxicity, which is a dose-dependent reversible pancytopenia usually observed after 2 weeks of therapy. With both of these drugs, increasing levels of serum iron and iron saturation appear to precede the decrease in peripheral blood cell counts, and erythroblast vacuolization appears to occur.[2] The 3 patients described herein concurrently received other medications that do not usually cause myelosuppression. Linezolid was the only drug they had in common, and there was a strong temporal association between linezolid use, hematologic changes, and recovery. Since linezolid may potentially replace vancomycin in situations requiring long-term or outpatient therapy, it is important for clinicians to be aware of this toxicity and to closely monitor iron levels and reticulocyte counts in patients receiving linezolid for prolonged periods.

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Financial Disclosure: Dr Green was a clinical investigator in trials of linezolid using a research grant from Pharmacia, the manufacturer of the drug. None of the patients described in this letter were participants in those clinical trials and all reported events occurred after Dr Green's financial relationship with Pharmacia concluded.

1. Linezolid (Zyvox). In: *The Medical Letter*. 2000;42:45-46.
2. Williams DM. Pancytopenia, aplastic anemia, and pure red cell aplasia. In: Lee GR, Foerster J, Lukens J, Paraskevas F, Greer J, Rodgers G, eds. *Wintrobe's Clinical Hematology*. 10th ed. Baltimore, Md: Williams & Wilkins; 1999:1455-1459.

Methylmercury and Neurodevelopment: Reanalysis of the Seychelles Child Development Study Outcomes at 66 Months of Age

To the Editor: There is ongoing controversy about the effects of methylmercury (MeHg) exposure from fish consumption on child development. We previously reported no adverse devel-

opmental outcomes in children tested at 5.5 years of age following both prenatal and postnatal exposure from fish consumption in our longitudinal study in the Seychelles Islands.[1] A similar study in the Faeroe Islands reported subtle adverse effects associated with prenatal exposure from consumption of both fish and pilot whales.[2]

A National Academy of Sciences panel recently reviewed the evidence for health risks from dietary MeHg exposure.[3] The review included both the Seychelles and Faeroes studies. The panel concluded that both studies were well designed, controlled, and executed, and that a variety of differences, including methodological, might account for the

Table. Multiple Regression Reanalysis of 66-Month Test Scores*

	Regression Coefficient (SE) [*P* Value]					
Parameter	McCarthy GCI	PLS Total Language	Bender Gestalt Errors	Woodcock-Johnson Applied Problems	Woodcock-Johnson Letter Word Recognition	CBCL Total T Score
Maternal MeHg	–0.024 (0.153) [.88]	0.130 (0.055) [.02]	. . .	0.027 (0.034) [.43]	–0.007 (0.022) [.74]	–0.261 (0.176) [.14]
Child MeHg	0.318 (0.206) [.12]	0.152 (0.074) [.04]	. . .	0.097 (0.046) [.04]	0.053 (0.030) [.08]	–0.042 (0.235)[.86]
Sex, female	2.149 (0.680) [.002]	0.540 (0.250) [.03]	. . .	0.441 (0.153) [.004]	0.260 (0.100) [.01]	–0.989 (0.785) [.21]
Maternal MeHg, female	. . .	. . .	–0.070 (0.046) [.13]	. . .	. . .	. . .
Maternal MeHg, male	. . .	. . .	0.044 (0.047) [.36]	. . .	. . .	. . .
Child MeHg, female	. . .	. . .	0.094 (0.065) [.14]	. . .	. . .	. . .
Child MeHg, male	. . .	. . .	–0.164 (0.061) [.008]	. . .	. . .	. . .
Maternal age	0.08 (0.13) [.54]	0.01 (0.05) [.77]	–0.02 (0.03) [.48]	–0.01 (0.03) [.82]	0.002 (0.02) [.91]	–0.52 (0.14) [<.001]
Birth weight	2.23 (1.45) [.12]	0.75 (0.53) [.15]	–0.31 (0.31) [.32]	0.85 (0.32) [.01]	0.41 (0.21) [.06]	1.33 (1.66) [.42]
Child's age at test	17.39 (6.93) [.01]	8.87 (3.24) [.01]	0.001 (1.41) [>.99]	3.28 (1.72) [.06]	0.23 (1.13) [.84]	–8.20 (8.45) [.33]
Child's medical history (positive)‡	–0.21 (1.98) [.92]	0.14 (0.69) [.84]	0.54 (0.42) [.19]	–0.67 (0.43) [.12]	–0.55 (0.29) [.06]	2.43 (2.22) [.28]
HOME (*P* for overall test)	.001	.001	.002	.01	.001	.01
Low	–3.37 (1.00) [.001]	–1.38 (0.36) [<.001]	0.67 (0.22) [.002]	–0.43 (0.23) [.05]	–0.45 (0.15) [.002]	2.17 (1.15) [.06]
Medium	–0.73 (0.97) [.45]	–0.26 (0.36) [.47]	0.04 (0.21) [.86]	–0.27 (0.22) [.22]	–0.43 (0.14) [.003]	1.43 (1.13) [.21]
High	4.10 (1.04) [<.001]	1.64 (0.38) [<.001]	–0.71 (0.22) [.002]	0.70 (0.23) [.003]	0.88 (0.15) [<.001]	–3.60 (1.20) [.003]
SES (*P* for overall test)	.08	.08	.48	.05	.001	.12
Unskilled	–2.83 (1.25) [.02]	–1.20 (0.46) [.01]	0.41 (0.27) [.13]	–0.57 (0.28) [.04]	–0.58 (0.18) [.002]	2.40 (1.44) [.10]
Semiskilled	–0.94 (1.11) [.40]	–0.04 (0.40) [.92]	0.08 (0.24) [.73]	–0.20 (0.25) [.43]	–0.59 (0.16) [<.001]	1.89 (1.28) [.14]
Skilled	0.34 (1.29) [.79]	0.33 (0.48) [.49]	–0.18 (0.28) [.52]	–0.20 (0.29) [.49]	0.47 (0.19) [.01]	–0.43 (1.49) [.77]
Professional	3.43 (1.61) [.03]	0.91 (0.59) [.12]	–0.31 (0.35) [.37]	0.97 (0.37) [.01]	0.69 (0.23) [.003]	–3.85 (1.84) [0.04]
Caregiver IQ (*P* for overall test)	.02	.007	.005	.001	.67	.43
Lowest third	–2.65 (1.04) [.01]	–1.05 (0.38) [.01]	0.62 (0.22) [.01]	–0.90 (0.23) [<.001]	–0.10 (0.15) [.52]	1.16 (1.20) [.33]
Middle third	0.26 (0.93) [.78]	–0.07 (0.34) [.85]	0.08 (0.20) [.68]	0.11 (0.21) [.60]	–0.04 (0.14) [.78]	0.36 (1.07) [.73]
Highest third	2.39 (1.03) [.02]	1.11 (0.38) [.003]	–0.70 (0.22) [.002]	0.79 (0.23) [.001]	0.13 (0.15) [.38]	–1.52 (1.20) [.20]
Hearing level (*P* for overall test), dB	.87	.97	.05	.30	.68	.88
0-25	1.01 (2.05) [.62]	0.14 (0.83) [.87]	1.17 (0.47) [.01]	–0.71 (0.47) [.14]	0.06 (0.30) [.83]	–0.23 (2.34) [.92]
26-35	–0.24 (2.61) [.93]	–0.13 (1.04) [.90]	0.26 (0.58) [.66]	0.06 (0.60) [.91]	–0.31 (0.38) [.42]	1.43 (3.01) [.64]
>35	–0.77 (3.60) [.83]	–0.01 (1.49) [.99]	–1.43 (0.84) [.09]	0.64 (0.84) [.44]	0.24 (0.53) [.65]	–1.19 (4.13) [.77]
Examiner (*P* for overall test)	.001	.001	.62	.001	.61	.57
1	–3.25 (0.99) [.001]	–1.31 (0.35) [<.001]	–0.01 (0.21) [.95]	0.48 (0.22) [.03]	0.03 (0.15) [.82]	–1.20 (1.13) [.29]
2	–1.67 (0.96) [.08]	1.16 (0.34) [<.001]	–0.17 (0.21) [.42]	–0.80 (0.21) [<.001]	–0.13 (0.14) [.34]	0.17 (1.09) [.88]
3	4.92 (0.95) [<.001]	0.15 (0.35) [.66]	0.18 (0.21) [.38]	0.33 (0.21) [.13]	0.10 (0.14) [.48]	1.03 (1.40) [.46]

*The McCarthy GCI (General Cognitive Index) estimates cognitive ability; PLS (Preschool Language Scale) Total Language test measures language ability; Bender Gestalt Errors measures visual-spatial ability; Woodcock-Johnson Applied Problems estimates arithmetic achievement; Woodcock-Johnson Letter Word Recognition measures reading ability; the CBCL (Child Behavior Checklist) Total T Score is a reflection of the child's social and adaptive behavior; HOME (Home Observation for Measurement of the Environment) measures the quality of the home environment; and SES, assessment of socioeconomic status (based on the Hollingshead instrument). MeHg indicates methylmercury. For references for these tests see the original article in *JAMA*.[1]

†Outliers have been removed. Coefficients for individual categories of covariates were constrained to sum to 0. For binary covariates, we included only 1 of the 2 available coefficients. For models with significant sex × methylmercury interactions, main effects of methylmercury and of sex are omitted from the table.

‡Evidence of a postnatal threat to the child's health or development was noted in the medical record.

difference in results. The panel made 2 suggestions concerning the analysis of our 5.5-year test scores: (1) the use of raw test scores instead of standardized scores and the inclusion of the child's age at testing as an additional covariate in the analysis would provide better control for the age of the child at testing; and (2) adjustment for which of the 3 staff members administered the test battery would improve the precision of the analysis.

Methods. In the original study, all children were tested at a mean (SD) age of 66 (6) months and standardized test scores afforded inherent control for age, and intertester reliability among the 3 testers was consistently high. We reanalyzed the data from the 6 primary end points at 66 months, using the same statistical procedures reported earlier, except that raw test scores were used, and age at testing and tester were included as additional covariates in the regression analyses.

Results. The reanalyses confirmed our previous findings. The TABLE summarizes the results in the same format as our earlier report.[1] The significance and direction of the parameter estimates for prenatal and postnatal exposure are nearly identical to those reported earlier. We did find significant effects of both age at testing (to be expected since scores were no longer scaled) and tester (not unlikely since testers did not have 100% agreement). There continues to be an association among both prenatal and postnatal exposure and the Preschool Language Scale Total Score, as well as an association in males only among postnatal exposure and scores on the Woodcock-Johnson Applied Problems and the Bender Gestalt drawing and copying errors.

Comment. These associations continue to suggest beneficial effects with increasing mercury levels that may reflect dietary benefits of fish consumption. In a population exposed to MeHg from consumption of ocean fish, we continue to find no evidence of adverse effects.

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1. Davidson PW, Myers GJ, Cox C, et al. Effects of prenatal and postnatal methylmercury exposure from fish consumption on neurodevelopment: outcomes at 66 months of age in the Seychelles Child Development Study. *JAMA*. 1998;280:701-707.
2. Grandjean P, Weihe P, White RF, et al. Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury. *Neurotoxicol Teratol*. 1997;19:417-428.
3. National Research Council. *Toxicological Effects of Methylmercury*. Washington, DC: National Academy Press; 2000.

CORRECTION

Incorrect Hospital Name, Incorrect Numbers, and Rounding Errors: In the Original Contribution entitled "Handheld Cellular Telephone Use and Risk of Brain Cancer" published in the December 20, 2000, issue of THE JOURNAL (2000;284:3001-3007), a hospital was identified incorrectly, incorrect statistics appeared in the "Methods" section, and rounding errors appeared in 2 of the tables. On page 3001, Dr Neugut's affiliation should have read "Department of Medicine, Mailman School of Public Health, Columbia University, New York, NY." On page 3002, the first sentence in the "Methods" section should have read "New York Presbyterian Hospital" not "Columbia University Presbyterian Hospital." Also on page 3002, the second to last sentence in the second column should have been "In the first 2 years of the study, 100 of the case interviews (21%) and 22 of the control interviews (5%) were conducted with proxies." Also on page 3003, in Table 2, the corresponding values for "Musculoskeletal disorder" should have been "71 (23.9)." On page 3005, in Table 4, the corresponding values for "No. (%) Who Ever Used a Cellular Telephone" for "Cerebrum (not lobes)" should have been "36 (5.6)."