Pressure to Publish in the Premedical Years

Ware G. Kuschner

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Editors and Owners of Scientific Journals

To the Editor: Certainly, as Dr Rennie suggests,1 the Massachusetts Medical Society (MMS) is very much aware of its responsibilities as steward of the New England Journal of Medicine. We know that our guarantee of editorial independence is a necessary condition for the integrity of content. Furthermore, as Rennie points out, the editorial independence of the journal’s editors has never been in dispute. Rather than dictate content, our role as publishers is to provide an environment in which editors and their colleagues are free to explore all areas of medicine and to follow the truth, wherever it leads them.

However, even in an age when it is fashionable to believe the worst about people, commentators better serve their readers and their publication when they move beyond reflexive cynicism. Moreover, those who enjoy editorial independence must use their bully pulpit responsibly. Unfortunately, in his discussion about the recent departure of Jerome P. Kassirer, MD, from our journal, Rennie has chosen, with little foundation, to believe the worst about the motives of the MMS. A closer study of our words—and our deeds—should finally retire all such unfair speculation.

Contrary to the assertions of cynics and critics, our work is not, nor will it ever be, about commercialism. Our activities are solely an expression of our 200-year-old mission to educate and advocate for patients and physicians; everything we do is focused on those purposes. All 13 of our physician-edited publications meet this expectation.

We are facing a new world of electronic publishing, so we must remain open-minded and responsive to this ever-changing environment if our publications are to remain financially viable and, thus, survive. We want to foster creativity because there are many unmet needs for reliable medical information, and we are among the few publishers who can meet these needs and still provide material that is not beholden to any commercial or ideological agenda. Importantly, all new ideas undergo an intensive process of investigation and discussion, by which their merits and their conformity to our mission are carefully studied. Only the best are implemented.

These bedrock values of editorial excellence, independence, and integrity will never change. This is not only my commitment as president of the MMS; it is the commitment of all of our members as well.

Jack T. Evjy, MD
Massachusetts Medical Society
Waltham


To the Editor: The Council of Science Editors, which includes both scientific editors and publishers, views with deep concern the second firing within one year of the editor of a major medical journal.1 The involuntary separations of George Lundberg, MD, from JAMA and Jerome Kassirer, MD, from the New England Journal of Medicine raise troubling questions about editorial freedom, the reputations and cultures of scientific publications, and the proper relationship between a journal and its owners.

We recognize that editors, publishers, and societies have increasingly complex goals aside from publicizing scientific advances, including political and financial agendas.2 The Lundberg and Kassirer incidents, together with the proposal by Harold Varmus, MD, of the National Institutes of Health for a free electronic repository of biomedical research reports,3 may cause some to conclude that the most important issues in scientific publishing today have less to do with specific persons and the boundaries of their editorial responsibility than with money, power, and control, especially control over dissemination of and access to information, both within and beyond the scientific community. Nevertheless, journals and their editors must focus on—must be allowed to focus on—their primary mission: publishing the highest-quality peer-reviewed articles for the needs of scientists, clinicians, and, ultimately, patients.

Monica Bradford, PhD
Annette Flanagan, RN, MA
Michael Held, MA
Pat Huston, MD, MPH
Cheryl Iverson, MA
Tom Lang, MA
Faith McLellan, PhD
Blaire Mossman
Ted Parker, MBA
Board of Directors of the Council of Science Editors
Reston, Va


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Letters Section Editors: Phil B. Fontanarosa, MD, Deputy Editor, Margaret A. Winker, MD, Deputy Editor, Stephen J. Lurie, MD, PhD, Fishbein Fellow.
In Reply: Dr Evjy finds me guilty of “reflexive cynicism.” I have chosen, he claims, “with little foundation, to believe the worst about the motives of the MMS.” He continues, “A closer study of our words—and our deeds—should finally retire all such unfair speculation.”

To avoid any such speculation, I did indeed study Evjy’s deeds. The executive of the MMS forced the departure of his editor, Dr Kassirer, a distinguished academic clinician, prematurely and against his wishes, very largely over a fight about branding unrelated products with the journal’s name. That’s an impressive deed. Several other deeds followed as a direct consequence: resignations, in protest, from the New England Journal of Medicine’s editorial board, from among the editors, and from the Committee on Publications. These expressions of dismay by people I know and greatly respect helped me feel even more confident that my strong objections to Kassirer’s removal were based on solid foundations.

I am glad to read the list of “bedrock values” expressed in Evjy’s letter. But since I believe that actions are more impressive than words, I am skeptical. Those who want more information should read the correspondence in the October 21, 1999, issue of the New England Journal of Medicine.1 Dr Evjy would do well to listen to those readers who expressed sadness, shock, and outrage. They are the subscribers.

The New England Journal of Medicine is a precious resource. Fortunately, the editors who are left have a strong track record for scholarship and integrity. The MMS, facing the resignation of all its editors, has reached an interim agreement on a set of principles with the interim editor-in-chief, Marcia Angell, MD.2 In view of Kassirer’s statement that “the crisis is not over,”1 there is reason to be concerned that the protections afforded Angell under this agreement might not outlive Angell’s tenure. Kassirer notes that the body to whom the editor reported, the Committee on Publications, was bypassed in the process leading to his departure. Kassirer therefore urges that their search committee not merely select the next editor-in-chief, but first devise some protection for future editors. He points to the agreement the American Medical Association recently struck with the JAMA Search Committee as being a suitable model.3 I was part of the negotiations that led to the latter agreement, and I strongly endorse this idea.

The MMS now has a unique chance to strengthen the New England Journal of Medicine and rebuild the trust so necessary for the running of such an enterprise.4 I hope the society will grasp it and rebuild the trust so necessary for the running of such an enterprise. The world will be watching the deeds of the MMS very closely.

Drummond Rennie, MD
Deputy Editor, JAMA
Chicago, Ill


The Hospitalist’s Role in Advance-Care Directives

To the Editor: Dr Pantilat and colleagues1 described ethical issues presented by the hospitalist system. They describe a patient who desires “only comfort care” when her condition worsens. Her primary care physician wrote an outpatient do-not-resuscitate (DNR) order that stated “does not want [cardiopulmonary resuscitation] CPR for cardiac arrest.” In our opinion, this order, in isolation, did not adequately reflect the wishes of the patient and created the hospitalist’s dilemma. When the patient’s condition became unstable, and required mechanical ventilation and vasopressors, the hospitalist overrode the DNR order by initiating these treatments. There are 2 separate issues in this case. First, there was no report of a cardiac arrest, but the patient did need treatment and support for a potentially reversible condition (pulmonary embolism) despite her underlying malignancy. Second, little documentation was available to the hospitalist about the meaning of the DNR order or its implications for other treatment decisions.

It is not clear that the hospitalist overrode the DNR order. In fact, his action seems prudent given the limited information he had been provided. In medical literature as well as in practice, the meaning of the DNR order can range from do not treat aggressively to do not initiate CPR or advanced cardiac life support in the event of cardiac or pulmonary arrest.2 However, in 1983 the president’s commission stated “any DNR policy should ensure that the order not to resuscitate has no implications for any other treatment decisions. Patients with DNR orders on their charts may still be quite appropriate candidates for all other vigorous care, including intensive care.”3 We think the hospitalist’s actions were appropriate based on the information at hand because other treatment limitations are not inherent in the DNR order.

Advance directives, as Pantilat et al acknowledged, have not solved the problem of clear documentation of patients’ wishes. At our institution, we are pilot-testing a project that requires a “Physician Documentation of Preferred Intensity of Treatment” form to document discussions with patients (or their surrogates) about the patient’s wishes for end-of-life care. This form can be easily copied and transferred between facilities (ie, acute hospital, clinic, and skilled nursing facilities). In this case, the preferred intensity treatment form may have provided guidance to the hospitalist.

Leslea Brickner, MD
Theresa Drought, RN
Kaiser Permanente
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In Reply: The loss of information during the transition from outpatient care to the hospital creates ethical dilemmas for physicians and patients. As pointed out by Dr Brickner and Ms Drought, discussions about resuscitation status and end-of-life care are particularly susceptible to confusion. A readily available form that clearly documents a patient's wishes could help resolve ambiguities about the patient's goals for care.1 The preferred intensity of treatment form described by Brickner and Drought represents a creative approach. Such a document may encourage physicians and patients to discuss preferences for care and could guide the content of these conversations. Nonetheless, even a thorough and revealing discussion with a trusted primary care physician may not address an issue such as the massive pulmonary embolus experienced by the patient in our case.

In addition to creative approaches, such as the preferred intensity of treatment form, which attempts to make the content of prior discussions available to the inpatient physician at the point of care, we see no substitute for hospitalists routinely discussing preferences for treatment with every patient for whom they care. Such discussions allow the hospitalist to be certain that the patient has not rethought prior preferences in light of recent events2 and validate the crucial role of the primary care physician. Moreover, compared with outpatient conversations, discussions at the time of admission could focus on specific scenarios.

Hospitalists can do much to resolve ethical dilemmas raised by discontinuity of care. Solutions that respect the central role of the primary care physician but also take advantage of the opportunities presented by a hospital admission may be most likely to respect patient preferences and to promote the highest quality care.

Steven Z. Pantilat, MD
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Robert M. Wachter, MD
University of California, San Francisco


Accurate Ascertainment of Child-Abuse Mortality

To the Editor: The article by Dr Herman-Giddens and colleagues1 contributes to the literature on the scope of the child abuse problem by providing an estimate of underreporting in vital statistics. Similar studies2 provide estimates of reliability for other causes of death.

Two reasons account for differences in estimates of child abuse deaths between the vital statistics system and those of the authors' comprehensive case fatality review: (1) the availability of information on circumstances of the death and (2) definitional differences. Herman-Giddens et al reviewed a broad range of information from which child abuse could be inferred, including medical examiners' reports, autopsy reports, toxicology reports, and case notes. In contrast, ascertainment of child abuse in vital statistics depends entirely on the cause of death reported by the medical examiner, coroner, or attending physician. This is the only information available to the mortality medical coder to make judgments or inferences about the circumstances of death.

Differences in definition are also critical. The authors propose a definition of child abuse that depends on information about the caregiver who was responsible for the child's health or welfare. Such a definition does not lend itself to vital statistics, because information about the caregiver is rarely reported on death certificates. In 1997, such information was reported on death certificates for only 1 of 10 deaths classified as due to child abuse.3

The authors question the vital statistics coding practice that, in the absence of the terms “abuse” or “maltreatment,” relies on terms consistent with prior or repeated abuse, beating, or other maltreatment. These coding procedures were established by the National Center for Health Statistics in the 1980s in consultation with child abuse experts from the US Department of Health, Education, and Welfare to address the absence of uniform national coding guidelines for child abuse. Prior to those rules, each state coded child abuse according to its own criteria, resulting in statistics that could not be compared among states.

Opportunities exist to bring vital statistics data into closer alignment with the case review estimates through educating medical examiners about certifying these deaths.4 However, it is unrealistic to expect a broad-based data system such as vital statistics to provide the same estimates as an intensive case review system.5 The strengths of the vital statistics system are in providing timely, consistent, and uniform information on cause of death to illuminate geographic differences, trends over time, and sociodemographic differences in risk of death. Studies such as those by Herman-Giddens et al are a useful complement to mortality data from vital statistics.

Harry M. Rosenberg, PhD
Mary Anne Freedman
National Center for Health Statistics
Hyattsville, Md


In Reply: Dr Rosenberg and Ms Freedman stress 2 of the causes we cited for differences in the number of child abuse deaths recorded by the vital statistics system and the number obtained in our study. We appreciate their further elaboration on the limitations under which nosologists function and their rec-
Estrogen-Receptor Status in Breast Cancer

To the Editor: Estrogen-receptor (ER) levels are an important predictor of breast cancer prognosis. When combined with other prognostic information, ER status is predictive of disease-free survival.\(^1\) Although ER status is commonly used in the medical literature, its cutoff value is still arbitrary. For example, in the Early Breast Cancer Trialists’ Collaborative Group’s study,\(^2\) ER-positivity was defined as at least 10 fmol ER per milligram of cytosol protein, while Clark et al\(^3\) defined ER positivity as at least 3 fmol/mg of protein-specific binding sites. Without a consensus on the cutoff point, it is difficult to make medical decisions based on these research findings.

Cummings and colleagues\(^4\) concluded that raloxifene decreased the risk of ER-positive breast cancer by 90%. Even though their results are promising, the definition of ER status was not provided. Inclusion of the ER cutoff point would have made this study more valuable.

In addition, the relative risk (RR) calculations were not appropriate. Only ER-positive patients can have invasive ER-positive breast cancer. Therefore, the RR should have been based only on the patients who had ER-positive test results. Cummings et al calculated the RR as 0.10 by mistakenly including all of the patients in each group. The same error occurred in the RR calculation for ER-negative invasive breast cancer. Estrogen-receptor levels of all subjects should have been measured before performing these calculations.

Yen-Hong Kuo, ScM, MS
Meridian Health System
Neptune, NJ


In Reply: The ER status of breast cancer was determined based on clinical reports from the numerous sites that participated in this international study; it was not feasible to mandate a single method. Variability in the assessment of ER status might have led our study to underestimate the differential effect of raloxifene on ER-positive and ER-negative cancer; however, we found that these effects of raloxifene on risk of ER-positive and ER-negative cancer were extremely different.

We disagree with Mr Kuo’s assertion that the RR calculation should be based only on participants who had ER-positive cancer. We believe that the RR of ER-positive and ER-negative cancer was appropriately calculated by comparing the rates of each type of cancer in the treatment group vs the placebo group.

Steven R. Cummings, MD
Deborah Grady, MD
Dennis Black, PhD
Prevention Sciences Group
San Francisco, Calif

The federal government, including the Centers for Disease Control and Prevention, which includes the National Center for Health Statistics, would need to be the vanguard.

Specifically, we suggest the following steps: (1) creating a child-specific death certificate with prompts so that available space could be used to capture information, including sociodemographic data, pertinent to children that would support improved risk of death analyses for all causes and manners of death; (2) revising the rules for coding child abuse fatalities; (3) improving training in medical schools and residencies on the correct process for completion of a death certificate; (4) increasing involvement of forensic pathologists in ascertaining causes and manners of death; (5) continuing advocacy for transitions from lay medicolegal death investigation systems to those staffed by medical professionals; and (6) continuing development, implementation, and evaluation of systematic, continuous child death review programs, with the charge to these panels to report on a regular basis the contribution of maltreatment to children’s deaths.

Nonetheless, we recognize that the vital statistics system can never be as accurate as an individual record review. More deliberate practices must be implemented for rare causes of death because misclassification can lead to a proportionally greater underascertainment of the total impact, particularly when the health and safety of children are involved. Moreover, inexact cause-of-death classification impairs the development of appropriate funding and intervention policies, assessment of the efficacy of prevention programs, and the ability to quickly address epidemics.

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Gail Brown, MD, MPH
Sarah Verbiest, MSW, MPH
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The American Medical Association requires that individuals disclose all relevant conflicts of interest. Disclosures are available at www.jama.com.

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Letters are the opportunity for readers to express opinions and other viewpoints. The opinions expressed do not necessarily reflect the views of the editors or the American Medical Association. Submitted authors are encouraged to submit letters electronically through the website www.submitjama.com.
Drs Rochon and Gurwitz1 document specific
underuse of proven therapies and properly encourage application of effective agents for “seniors (65 years and older).” However, for purposes of pharmacological management, all elderly patients should not be grouped together because they fall roughly into 2 cohorts, those younger than 75 years and those of the rapidly increasing population aged 75 years or older. For the older group, reliable prospective studies are rare because they are complicated by multiple morbidities, frequent compromise of 1 or more organ systems, and substantial interindividual variability of general health. Because of these pathophysiological problems and sparse data, most decisions for older elderly patients will continue to be individualized and less accurately defined.

These issues are well illustrated in decisions for blood pressure management in persons aged 75 years or older. Rochon and Gurwitz point out that elevated blood pressure levels in this group are often clinically unattended. However, there is sound basis on which to make such decisions for those older than 75 to 80 years. In the largest randomized studies of long-term hypertensive treatment of elderly persons, mean ages were 72,2 64,3 and 704 years; only 1 (the Systolic Hypertension in the Elderly Program [SHEP] study cited by the authors) included a few patients older than 74 years.2 In the smaller, more pertinent Swedish Trial in Old Patients With Hypertension (STOP-H; age range, 70-85 years, with 16% older than 80 years),3 treatment reduced the overall incidence of several adverse events and death. However, after age 74 years, the benefits were no longer statistically significant, and after age 82 to 83 years, there were (nonsignificantly) more adverse events in treated subjects.3 In addition, none of these reports reflect the overall population: subjects were living independently, and free of major illnesses; these criteria led to exclusion of about 50% of hypertensive patients in the STOP-H study.

Such considerations illustrate the continuing difficulties for the practitioner in prescribing for patients aged 75 years or older. Randomized studies pertain primarily to those younger than 75 years. In Western countries, most members of this younger elderly group remain active and have minimal morbidities; for this group, with individual exceptions, it is reasonable to use available data or perhaps extend information from middle-aged subjects for therapeutic decisions. However, it would be imprudent to extrapolate evidence from such trials to older elderly patients. In the absence of more complete information, clinicians must continue to be cautious and light-handed in treating patients older than 75 to 80 years.

Gerson T. Lesser, MD
Leslie S. Libow, MD
The Jewish Home and Hospital
New York, NY

In Reply: Drs Lesser and Libow highlight the challenges of prescribing for persons of advanced age in the context of limited efficacy and safety data.

Older people certainly are poorly represented in trials. Our work illustrates the magnitude of this problem. Among the 9664 subjects identified in our systematic review of trials of nonsteroidal anti-inflammatory therapy for arthritis treatment,1 only 14 people were aged 75 to 84 years and none were 85 years or older. Poor representation is likely compounded for women, who are disproportionately represented in the oldest age group. Our systematic review of myocardial infarction trials demonstrated that less than 25% of participants were women and virtually none were of advanced age.2 These findings are inconsistent with the US Food and Drug Administration guidelines3 that encourage investigators to evaluate drug therapies in the types of patients that will use them in clinical practice.


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Despite poor representation of older persons in trials, treatment decisions need to be made. The relatively few older people included in any single trial make it difficult to conduct a meaningful subgroup analysis. Subgroup analyses are possible using meta-analysis, as illustrated by a recent review of hypertension trials. Guéyffier et al\(^4\) evaluated 1670 patients who were at least 80 years of age participating in 7 major hypertension trials. Antihypertensive treatment relative to placebo reduced the risk for stroke, major cardiovascular events, and heart failure, but did not affect mortality. This meta-analysis supports treatment of hypertension in people of advanced age.

The key clinical question may not be whether a drug therapy should be administered to an older person, but how best to prescribe that therapy. For example, while β-blocker therapy has proven beneficial in patients with heart disease, the vast majority of older people are prescribed dosages lower than were evaluated. Among 10,991 myocardial infarction survivors dispensed β-blocker therapy in Ontario, 9458 (86.1%) were dispensed a lower than evaluated dose.\(^3\) Older people may not tolerate dosages of drug therapies that were tested on younger, fitter, and male patients. Barron et al\(^6\) demonstrated that use of lower dosages of β-blocker therapy was associated with similar mortality reduction relative to higher dosages.

We fully support the need to increase the representation of older patients in clinical trials. In the absence of adequate data on drug efficacy and safety, we agree that therapeutic decisions need to be individualized in very old patients.

Paula A. Rochon, MD, MPH, FRCPC
University of Toronto
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Pressure to Publish in the Premedical Years

To the Editor: Many premedical students seek experience assisting with biomedical research. For some, a summer doing research is a way of bringing career plans into greater focus. For many students, however, their minds have been made up; they want to go to medical school, and they are keenly aware that doing research on a medical school campus can help them achieve that goal.

Letters of recommendation from academic physicians and biomedical researchers are valuable currency in the competitive pursuit of gaining admission to medical school. Moreover, for a few volunteers, research can lead to coauthorship on a journal article. After research grant money, publications carry more weight in academic medical centers than virtually any other marker of accomplishment. That message is broadcast so loudly that it now resonates on the undergraduate campus.

In an era of $300 per hour SAT (Scholastic Aptitude Test) tutors,\(^1\) the aggressive pursuit of impressing admissions committees comes as no surprise. Nevertheless, I recently received a curriculum vitae (CV) that seemed to set a new standard for prolific achievement in biomedical research by a young adult. This 20-year-old college student had 11 publications listed on his CV, most of which addressed the surgical management of a type of cancer. Some were published when the author was 16 years old.

Ten of the 11 articles included another author, usually the senior author, with the same last name. That a well-connected family member engaged in medical research might want to help a young student strengthen his or her CV, irrespective of the student’s actual contribution to the research, is understandable although lamentable. That a medical school admission committee might actually be impressed by such distortion is troubling.

The pressure to “publish or perish” in higher education pushes faculty to generate new knowledge. This pressure should not, however, be visited on college students who have not even completed basic premedical course work. For the undergraduate who ultimately attends medical school, there will be plenty of time to publish research if doing so becomes a genuine career interest. Most physicians, however, ultimately pursue a career in taking care of patients.

The message to premedical students from medical schools should be clear and consistent: participating in biomedical research for the sake of impressing admission committees is probably a mistake. Rather, demonstrating passion, commitment, and integrity in academics and extracurricular pursuits is impressive and will be rewarded.

Ware G. Kuschnier, MD
Stanford University School of Medicine
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Human Papillomavirus Antibody and Risk of Prostate Cancer

To the Editor: Human papillomavirus type 16 (HPV 16) is the HPV type most frequently detected in human cancers and is associated with an increased risk of cervical and lower ano-
Methods. The association of prostate cancer risk with the presence of HPV 16 antibody was examined in a nested case-control study within the Child Health and Development Study cohort, which enrolled members of the Kaiser Foundation Health Plan residing in the Oakland, Calif, area between 1959 and 1966. Cancer cases were identified from more than 13,000 men in this cohort by linkage with the California Cancer Registry through October 1993 (estimated to have been 97% and 75% complete, statewide, through 1991 and 1992, respectively). There were 48 incident prostate cancer cases, from the time of enrollment through October 1993, occurring an average of 26 years (range, 12-32 years) after enrollment. Control subjects were 63 men in the cohort who had been cancer-free as of October 1993, frequency matched with cancer cases by decade of birth (mean age at enrollment was 36 years) and race (53% white, 36% black, 3% Hispanic, and 8% other). Presence of HPV antibody in stored serum samples (obtained by phlebotomy at time of enrollment) was determined in 1998 using an enzyme-linked immunosorbent assay (ELISA) against HPV 16 viruslike particles as previously described. Optical density (OD) of 1.017 or greater was considered positive and OD less than 0.904 was considered negative. Indeterminate values (0.904 ≤ OD < 1.017) were excluded from the analysis. The relative risk of prostate cancer was estimated by the odds ratio (OR) and 95% confidence intervals (CIs) from multiple logistic regression analysis.

Results. Overall, 20 (42%) of 48 cancer subjects and 19 (30%) of 63 control subjects were HPV 16 antibody positive. The prevalence of HPV 16 antibody was not significantly different by race/ethnic groups. Because few cancer cases (n=4) were of race other than white or black, individuals of other races were excluded from the analysis. With adjustment for age at serum sampling and race, HPV 16 antibody positivity was marginally associated with an increased risk of prostate cancer (OR, 2.7; 95% CI, 0.9-7.9). The association remained unchanged with additional adjustment for educational level and smoking status.

Comment. The presence of antibody to HPV 16 has been associated with an increased risk of cervical cancer in the present study cohort (M.H. et al, unpublished data). An increase in prostate cancer risk among subjects with HPV 16 antibody compared with those without the antibody is consistent with the recent report by Dillner et al, in which a 2.4-fold risk (95% CI, 0.8-7.6) increase was reported. However, the results from these prospective studies conflict with the results from case-control studies that failed to detect an association. Furthermore, we may have underestimated the prevalence of HPV infection in both subjects and controls, because infections that occurred after phlebotomy would have been missed. The role of HPV infection in the etiology of prostate cancer warrants further investigation, with serial measurements of antibody levels in a prospective study.

CORRECTION

Transposed Data: In the Original Contribution titled “Cost-effectiveness of Vaccination Against Pneumococcal Bacteremia Among Elderly People,” published in the October 23/29, 1997, issue of the Journal (1997;278:1333-1339), several rows of data in Table 3 on page 1337 were transposed. Entries for “No vaccination” and “Vaccination” in the column “Total Effectiveness (Quality-Adjusted Life-Years)” were reversed for the following: “Age ≥ 65 y,” “Metropolitan Atlanta, Ga,” and “Monroe County, New York.” Figures in all other columns were correct as printed.
† There were originally 51 subjects in this group, but the blood draw was unsuccessful in
the majority of subjects in both groups. The conjugated meningococcal C conjugate vac-
cine was well tolerated by both groups. The antibody responses to the conjugated meningococcal vaccine are summarized in the TABLE. The conjugated meningococcal vaccine induced titers of bactericidal antibody considered to be protective (≥1:8) in the majority of subjects in both groups. However, at a mean age of 50 months, there was still evidence of immune hyporesponsiveness to the meningococcal C polysaccharide component among the children who previously had been found to be hyporesponsive at 34 months.

**Table.** Serum ELISA and Bactericidal Responses to Immunization With Conjugated Meningococcal C Vaccine*

<table>
<thead>
<tr>
<th></th>
<th>Group 2 (n = 51)</th>
<th>Group 3 (n = 47)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous doses of plain meningococcal polysaccharide vaccine, No.</td>
<td>3</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>IgG anticapsular antibody, geometric mean (µg/mL) (95% CI)</td>
<td>0.74 (0.48-1.1) 0.93 (0.6-1.5)</td>
<td>.46</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>48† 47</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Month after immunization</td>
<td>6.6 (4.5-9.6) 12 (8.2-18.0)</td>
<td>.03</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>49 46</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bactericidal antibody responses 1 month after immunization</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1/Geometric mean (95% CI)</td>
<td>16 (9.8-25) 36 (25-59)</td>
<td>.01</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>49 46</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Titer ≥1:8, % (95% CI)</td>
<td>69 (55-82) 78 (64-89)</td>
<td>.23</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>49 46</td>
<td></td>
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</tr>
</tbody>
</table>

*ELISA indicates enzyme-linked immunosorbent assay; CI, confidence interval.
†There were originally 51 subjects in this group, but the blood draw was unsuccessful in 3 subjects.

children induces a hyporesponsive state to meningococcal C polysaccharide has been confirmed in multiple studies.\(^1-3\) We found that these children also are relatively hyporesponsive when given meningococcal C conjugate vaccine, and hyporesponsiveness can be detected 18 months after receipt of the last dose of the plain meningococcal polysaccharide vaccine. The molecular mechanism of this immune hyporesponsiveness is unknown.

Evaluation of this phenomenon in children and adults with known terminal complement component deficiency who have previously received plain meningococcal quadrivalent polysaccharide vaccine may be important because these patients are at increased risk of recurrent meningococcal disease.

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**Previous Presentation:** Presented in part at the 1999 Annual Meeting of the Pediatric Academic Societies, San Francisco, Calif, May 1999.


**CORRECTIONS**

**Error in Affiliation:** In the Letter to the Editor entitled “Treatment of Hypotremic Encephalopathy” published in the December 22/29, 1999, issue of THE JOURNAL (1999;282:2298-2299), there was an error in one of the affiliations on page 2299. The affiliation for Dr Sterns should read “University of Rochester School of Medicine and Dentistry, Rochester, NY.”

**Word Omitted:** In the Letter to the Editor titled “Prescribing for Elderly Persons” published in the January 19, 2000, issue of THE JOURNAL (2000;283:339-340), the word “no” was omitted from the first letter on page 339, altering the meaning of the sentence. In the second paragraph, the third sentence should have read, “However, there is no sound basis on which to make such decisions for those older than 75 to 80 years.”