even if it is understandable. The steps Young proposed for remedying the situation, such as listing all programs that a candidate has applied to, would help solve the problem. They should be considered by the Canadian Resident Matching Service.

In our program we now give serious consideration only to those who have spent some elective time with us. In this way we learn about the students, and they learn about us. With only 1 position available per year, we consider this a vital aspect of the screening process. It may place a well-intentioned student who has not done a rotation with us at a disadvantage, but under the current circumstances we believe it is the most reliable method for ranking our applicants.

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Drug to treat obesity: editorial writer responds

We are concerned that the letter by Sana R. Sukkari (*Can Med Assoc J* 1997;156:768-9) misrepresents the relationship that Dr. Manson and I had with the pharmaceutical industry. Upon invitation, we wrote an editorial about pharmacotherapy for obesity.[1] In the process, a series of miscommunications and misunderstandings occurred between the *New England Journal of Medicine* (*NEJM*) and us.

As stated in our subsequent letter to *NEJM*,[2] we had briefly served as scientific consultants to Servier, the manufacturer of dexfenfluramine (Redux) and had submitted a proposed disclosure statement to *NEJM*. *NEJM*'s written disclosure policy statement had ambiguities, and our direct discussions with their editorial staff were misinterpreted. This led to a series of misunderstandings.[2]

Most important, we had and have no financial interest in any manufacturer of anti-obesity drugs, nor do we stand to gain from the commercial success of any of their products. The opinions that we expressed were entirely our own and independent of industry. The editorial was carefully written and was in no way intended as an endorsement of appetite suppressants. We urged long-term studies and cautious prescribing to patients with medically significant obesity who had failed an exercise and diet program.

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Managing benign prostatic hyperplasia

As a very busy urologic surgeon in Toronto, I found that after reading the article "Efficacy and safety of finasteride therapy for benign prostatic hyperplasia: results of a 2-year randomized controlled trial (the PROSPECT Study)" (*Can Med Assoc J* 1996;155:1251-9), by Dr. J. Curtis Nickel and colleagues, I was even more confused than before as to the appropriate management of benign prostatic hyperplasia (BPH).

Patients with symptomatic BPH usually require or request some treatment. To say that finasteride is a viable and safe alternative to watchful waiting is confusing and inappropriate. If one has embarked on watchful waiting, then there is an understanding between the patient and the physician that no intervention is necessary because the symptoms or signs of BPH are not significant. There should be no therapy, not even a "safe, nonoffensive therapy," that is not required.

If, however, one has determined that the symptoms (as defined by the symptom score), urinary flow or sequelae of BPH demand treatment, then one must prescribe the most effective, reliable and safe treatment. There is no golden pill that works for everyone, even if patients have the same size of prostate. In my hands, terazosin has been very safe and reliably effective.

I find it hard to reconcile the fact that, in a recently published study of BPH in veterans,[1] the investigators found no improvement in the patients taking finasteride compared with those taking a placebo. Even if we accept the retrospective analysis that finasteride, because of its mode of action, should be more effective in larger prostates, we still find significant discrepancies. In the subset of patients with prostate volumes greater than 50 mL, the urinary flow improved by 2.5 mL per second in the group taking finasteride v. 3.9 mL per second in the group taking terazosin. A similar trend was found in the symptom-score improvement. Another unexpected discovery in the study was that the prostate-specific antigen (PSA) level decreased in the group taking terazosin, but not in the group taking finasteride.

It seems logical that finasteride would work more effectively in larger prostates and that the patients' PSA level would decrease. However, this was not corroborated in the 2 studies.

Logically, as well, α-blocking agents should be more effective in the smaller prostates usually seen in younger patients; in such patients, the impotence that is a side effect of finasteride would be more troublesome.

At the primary care level, once one has decided that therapy is indicated

to treat symptomatic patients, cancer has been ruled out, and there are no significant contraindications, all studies have shown that α-blocking agents have a significant effect on all prostates, whereas finasteride has a beneficial effect only in men with significantly large prostates and major obstructive symptoms.

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[One of the authors responds:]

The trial involving veterans, published while our article was in press, concluded that terazosin was significantly more effective than placebo, whereas finasteride was not. However, it turned out that the patients enrolled in this study had a mean prostate size identical to that in a normal population of men.[2] A meta-analysis (incorporating the results of our Canadian study) subsequently confirmed that only patients with enlarged prostates had a response significantly better than those taking a placebo.[3] It is obvious now, but not when we designed our study in 1991, that a drug whose action is to shrink the prostate only works in men with large prostates. Many of us, including Dr. Barkin, are concerned about the unacceptable failure rate of drug therapy, particularly after several years. In a long-term study of terazosin,[4] twice as many patients with small prostates (32%) as with larger prostates (16%) were still available for study after 4 years. By contrast, more than 90% of patients taking finasteride who entered open-label trials (and who presumably had a favourable response secondary to shrinkage and stabilization of their prostates) were still taking the drug and were available for study 5 years later.[5] These new and important findings allow busy clinicians such as Barkin a less confusing and more efficient, durable and evidence-based approach to the treatment of his patients who do not choose watchful waiting, who have an indication for drug therapy or who are reluctant to undergo surgery. Most men with symptoms but with normal-size prostates (50% or more of Barkin's patients) can be expected to have a favourable and durable response to α-blocking agents. Both α-blocking agents and finasteride can achieve similar results in men with larger prostates. With finasteride, we can expect the response to be durable over the long term.

Barkin was also concerned about the confusing finding of the study involving veterans that the PSA level decreased in the terazosin group, but not in the finasteride group.[1] In fact, the result was precisely the opposite. This error had passed through proofreaders, editors and multiple authors. One must question everything one reads. Even the *New England Journal of Medicine* can make a mistake.

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Wiping out measles: When to vaccinate?

The measles outbreak reported in the article "Outbreak of measles in a highly vaccinated secondary school population" (*Can Med Assoc J* 1996;155:1407-13), by Drs. Penny A. Sutcliffe and Elizabeth Rea, is one of many such outbreaks during the last several years in North America. These outbreaks prompted our southern neighbour to switch to a 2-dose measles-vaccination strategy a long time ago. The article and the accompanying editorial "Elimination of measles in the Americas" (*Can Med Assoc J* 1996;155:1423-6), by Dr. John Furesz, support a 2-dose strategy to eliminate measles. However, the timing of the 2 doses is an issue that remains to be settled.

In all Canadian provinces, the first dose of measles–mumps–rubella (MMR) vaccine is administered at 12 months of age, except in PEI, where it is given at 15 months. In the new 2-dose strategy, a second dose is given at 18 months in Newfoundland, Quebec, Saskatchewan, BC, Yukon and the Northwest Territories, and at 4 to 6 years in PEI, Nova Scotia, Ontario, Manitoba and Alberta. Both schedules are consistent with the recommendations of the National Advisory Committee on Immunization.

Our studies of measles-vaccine response, vaccine failure and waning immunity shed some light on the timing of the 2 doses. Our data show that up to 16% of children who receive the first dose of MMR vaccine at 12 months do not respond adequately and remain without protective immunity after the first dose.[1,2] This lack of immunity cannot be attributed entirely to maternal measles