Ms Hazelbaker and her work on tongue-tie. However, we also stand by the integrity of our study.

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Postvasectomy Semen Analysis

To the Editor: I read with interest the article by Christensen and Maples[1] addressing postvasectomy semen analysis and the low compliance with instructions to confirm azoospermia. It prompted the following questions—when and how often is semen analysis required after a vasectomy, and is it *ever* necessary after a vasectomy? Furthermore, is it necessary to send the excised ends of the vas deferens for histopathological evaluation?

A review of the literature suggests that there is no definite agreement regarding the timing or the frequency of postvasectomy semen analysis. All ejaculates contain potentially fertile spermatozoa immediately after vasectomy, which become rapidly immobile within a few days, and usually by 3 weeks following the procedure.[2] The British Andrology Society guidelines requires patients to wait 4 months or have at least 24 ejaculations before semen analysis.[3] The society also recommends that patients not ejaculate for 48 hours prior to collection, collect semen by masturbation directly into the container, avoid condoms, and deliver the semen within an hour of collection.[3] The World Health Organization has different recommendations—one or 2 semen analyses after 12 weeks or 15 ejaculations.[4]

Azoospermia proven on a single semen analysis at 3 months is probably sufficient grounds for discontinuing other methods of contraception.[5] Further semen analyses should be required only if live sperm are present. Nonmotile sperm are probably not an indication for checking further semen samples.[6] Patient compliance is good if they are required to submit only one sample for analysis but decreases significantly when they are asked to provide a second sample.[5]

I suspect that postvasectomy semen analysis, though logical, is simply not necessary. Perhaps many patients (nearly 40% of my 360) realize this instinctively, wait the specified 3 to 4 months or, in many instances, 12 to 15 ejaculations, before commencing unprotected intimacy with their partners. A small percentage will undoubtedly have unintended issues, but humans gamble on success, and change will be difficult.

There is also no uniformity regarding histologic evaluation of the vasectomy specimens. One series from the United Kingdom showed that only three fourths of the surgeons followed this practice.[6] Provided that the vasa are confidently identified and sectioned, routinely evaluating specimens just adds to the cost. Of the patients requesting vasectomy in my practice, most pay for the procedure themselves, and they can ill-afford this added expense. Hence, I have tended to preserve the vas deferens specimens until azoospermia is established at 3 months, or for 1 year, after which time the specimens are discarded because of space constraints. This is explained to the patients before the vasectomy. I suspect many family physicians practicing in rural communities do the same.

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Author's Reply

To the Editor: Dr Ramakrishnan has raised some very good points. There is no absolute protocol for the number or timing of postvasectomy semen analyses. He suggests that a single 3-month postvasectomy semen analysis would probably suffice, which seems reasonable. My research, however, indicates that less than half the men returned at 3 months (25%) than returned for the 6-week check (58%). Because a semen analysis is the only way to know that one has achieved azoospermia—and that is the purpose of the vasectomy—then this noninvasive sampling is logical.

Our study followed Denniston and Pfenninger,[1] which suggested customary postoperative care, with the exception that we also encouraged a 12-month postoperative semen check, in which only 8% of men participated. I also agree that because 42% of my patients did not return for ANY postvasectomy semen analysis, there are a significant number of risk-takers getting a vasectomy. It has been our practice not to routinely send specimens of excised vas deferens to pathology, because it just incurs more cost and does not determine the success of the vasectomy.

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Excess Factor VIII and Hypercoagulability

To the Editor: The author of this article[1] states that "This is a report of 3 cases of thromboembolism not associated with conventional risk factors (trauma, cancer, or immobility). The patients were found to have elevated factor VIII activity without other evidence of a hypercoagulable state."

I would respectfully disagree that the patients show no evidence of any other hypercoagulable state. The antithrombin level was low in all 3 patients and could constitute a prothrombotic risk factor. In addition, the known risk factors, factor V Leiden and the prothrombin gene mutation, were not evaluated. Thus, we do not know whether the patients had these common risk factors.

The author also quotes sources to support the contention that "Elevated factor VIII levels have been found to persist over time and to be independent of the acute phase response."

This statement is a little misleading in the context of the current report. Previous studies found that even though factor VIII (FVIII) is an acute phase reactant, elevated FVIII levels persisted in some patients with thrombosis after an acute inflammatory stimulus had resolved. In addition, those authors compared FVIII levels with other acute phase reactants (ie, fibrinogen and C-reactive protein [CRP]) to determine whether there was evidence of concurrent acute inflammation. They only considered FVIII level to be an independent (not inflammation-related) risk factor for thrombosis when levels of other acute phase reactants were not elevated. The current study did not verify, by measuring CRP or fibrinogen levels, that the patients did not suffer from an inflammatory state that could have elevated FVIII levels.

Thus, it would have been very useful to know the CRP and fibrinogen levels for the patients reviewed in this report. This would allow firm conclusions to be drawn about whether the FVIII elevation was or was not related to a concurrent inflammatory state. The presence of an inflammatory state might suggest the presence of other factors predisposing to thrombosis. Thus, I do not believe that the author has clearly ruled out other risk factors for thrombosis in his patients and thus cannot attribute their thrombotic tendency to elevated FVIII levels.

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Author's Reply

To the Editor: In her critique of our article, Dr Hoffman states that factor V Leiden and the prothrombin gene mutation were not evaluated in the three patients. They were. The "NP" in the table means "not present" (see key under table).

That factor VIII can be an acute phase reactant seems to be common knowledge. However, I listed 3 references[1–3] that specifically examined this in patients with venous thromboembolism (VTE) and concluded that the increase in factor VIII was "persistent and independent of the acute phase response." O'Donnell et al[1] use that specific phrase in their title and find 94% of 35 VTE patients with elevated FVIII to have a persistent increase, independent of CRP and fibrinogen. O'Donnell et al[2] found elevated FVIII to be the single most common risk factor in 260 VTE patients and also stated that it did not correlate with CRP or fibrinogen. Kamphuisen et al[3] reached the same conclusion. ("Increased levels of FVIII and fibrinogen in patients with VTE are not caused by acute phase reactions.")

I would have liked to have had CRP and fibrinogen levels on my patients, but this was a retrospective study and none were done.

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