TO THE EDITOR: Qaseem and colleagues (1) recently presented clinical practice guidelines on HIV screening in health care settings. One of the authors (Dr. Owens) and 2 of the acknowledged commentators (Drs. Walensky and Paltiel) have had cost-effectiveness analyses of routine HIV testing published, but this is not the entirety of all researchers (myself included) who have written about this topic. Qaseem and colleagues classify only the economic evaluation work of the authors and commentators as good quality and inaccurately characterize the literature they did not generate. For instance, they dismiss my peer-reviewed cost-effectiveness analysis of a variety of counseling and testing strategies (2) because the targeted counseling and testing scenario assumes that high-risk clients can be identified at no additional cost. I made this assumption in the base-case analysis because targeting does not need to isolate individual clients for testing but can use existing surveillance information and other data to identify venue types and geographic areas with a higher background HIV seroprevalence. Still, I had a sensitivity analysis published that explores this specific assumption and found that even if one third of the available budget was spent on expenses related to targeting (and other highly conservative assumptions were also used), a targeted testing strategy still yields more HIV diagnoses and linkages to care than the Centers for Disease Control and Prevention’s opt-out approach (3).

The cost-effectiveness articles that Qaseem and colleagues judged to be of good quality (4–8) have limitations. First, those articles do not fully estimate the costs and consequences of a national implementation of the Centers for Disease Control and Prevention’s opt-out testing recommendations, but Qaseem and colleagues generalize from them to national policy. In addition, those articles compare screening with the absence of screening or with the status quo. Because screening is not compared with a variety of other counseling and testing strategies or even with other types of HIV prevention interventions, these articles do not inform policy questions, such as how to optimize the health benefits from limited HIV prevention resources (9), or how to quickly maximize the number of persons aware that they are living with HIV and help them access treatment.

When developing practice guidelines, the American College of Physicians should either have the literature reviewed by a completely independent group of researchers (as the Institute of Medicine might do) or include all persons publishing in the field to avoid mischaracterizations.

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Potential Financial Conflicts of Interest: None disclosed.

References


**IN RESPONSE:** We thank Drs. Van Allen and Kohlwees and Dr. Holgrave for their comments regarding the American College of Physicians’ recent clinical guidance statement on screening for HIV. Drs. Van Allen and Kohlwees are concerned about the potential for false-positive test results with widespread screening. Their example notes a specificity of 99.99% and estimates a substantial number of false-positive test results. In well-controlled testing programs using traditional HIV tests (enzyme immunoassay followed by Western blot) (1), the specificity of the sequence of antibody tests is as high as 99.9994% (1); therefore, false-positive results will be rare and far fewer than in their example. However, the specificity of rapid HIV tests may not be as high as that of traditional testing, and as we noted in our guidance statement, relatively high false-positive rates have been reported with an oral rapid test (2). We share the concern of Drs. Van Allen and Kohlwees about the impact of false-positive results. Patients and clinicians should be aware that HIV results with rapid tests are preliminary and must be confirmed with traditional antibody testing. The benefit of rapid testing is that more patients receive their test result.

We thank Dr. Holgrave for the clarification about the results of his work. We agree with him about the importance of independent literature reviews in the development of clinical guidelines. In our clinical guidance statement, we used literature reviews prepared independently from our committee by the U.S. Preventive Services Task Force (3, 4) and the Centers for Disease Control and Prevention (5). Current evidence has not shown targeted screening to be successful in reducing transmission of HIV in the United States or identifying patients early in the course of disease. We await evidence demonstrating the benefits of targeted screening and, until then, recommend adoption of routine screening for HIV. We also note that the cost-effectiveness of HIV screening was only 1 of several factors that we considered in making our recommendation for routine screening.

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**Potential Financial Conflicts of Interest:** None disclosed.

**References**


**The Professional Testing Center: Past and Present**

**TO THE EDITOR:** Bedgood (1) and Elon (2) highlight 2 very different testing experiences for American Board of Internal Medicine certification.

Like Elon, I remember taking my recertification examination with a pencil and paper. Taking the examination with my colleagues was a collective and reinforcing experience. We could support each other during breaks, tell jokes to cut the tension, and make new acquaintances who shared the same areas of specialization.

But many of the security concerns and measures of today existed then as well. When I took my geriatrics examination, proctors walked up and down the aisles, and we had to follow a specific schedule for completion of each section and for breaks—you could not complete the examination at your own pace. We had to raise our hands to go to the bathroom and be escorted out of the test room to the restroom and back. We signed the same pledge of honesty we agree to when we register for an examination today.

Twenty years later, the examination experience has changed, but not the need for security measures. The shift to computer-based testing provides an opportunity for physicians to take the examination at their own pace, and allows the American Board of Internal Medicine to offer the examination at many more locations. Pearson VUE has more than 200 testing centers across the United States and in 23 countries—including Iraq, where, despite some logistical challenges, Bedgood was able to successfully complete his certification examination in gastroenterology. In the old days of paper-and-pencil examinations, there were only 50 testing centers, and administration of an examination in Iraq would have been impossible. The
computer-based testing environment also allows for different testing elements (like video and audio) that are not available in the old model.

The professionalism and integrity of most examinees is unquestioned. But improper behavior from 1 examinee would compromise the results for an entire group. Pearson VUE’s security procedures are designed to help maintain examination integrity—but they come with tradeoffs, many of which are highlighted by Elon. We hear from physicians who prefer the new computer approach and from others who prefer the old pencil-and-paper model. Overall, most candidates believe that the security procedures are appropriate.

The test-taking environment is not perfect, and we are committed to improving the testing experience of all our diplomates. To that end, we appreciate the insights of Elon and Bedgood.

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Potential Financial Conflicts of Interest: Employment: Dr. Cassell is President and Chief Executive Officer of the American Board of Internal Medicine.

References

Does B-Type Natriuretic Peptide Testing Affect Outcome and Management of Patients With Acute Dyspnea?

TO THE EDITOR: Schneider and colleagues (1) describe how a group of Australian emergency department physicians treat patients with severe shortness of breath. They evaluate their patients on the basis of their symptoms and promptly institute therapy based on their clinical impression.

Their study examined whether advising physicians that “a BNP [B-type natriuretic peptide] level less than 100 ng/L made the diagnosis of heart failure unlikely, whereas a BNP level greater than 500 ng/L made heart failure likely,” would alter management of those patients (1). No other decision methodology, tree, or algorithm was instituted. The results show no difference between being informed and not being informed.

This comparison contrasts physician behavior, not the utility of BNP testing. Why patients were blinded to the B-type natriuretic peptide value is unclear, but it seems that physicians also blinded themselves to this. The results clearly show no difference between ignoring the test result versus not being aware of the test result. This study is not about the utility of BNP testing but the bravery of these Australian emergency department physicians, who can march on with their decisions despite the lack of clear, objective, laboratory-proven evidence.

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Potential Financial Conflicts of Interest: None disclosed.

Reference

TO THE EDITOR: Schneider and colleagues (1) reported on a well-conducted randomized trial on the impact of BNP testing on management of patients with dyspnea who present to the emergency department. They examined the impact of BNP measurement on clinical outcomes among patients with acute dyspnea. It is a well-powered study, given the large sample size.

Schneider and colleagues noted that, within the BNP-tested group, patients with a final diagnosis of heart failure had markedly elevated BNP values compared with patients without heart failure and that BNP testing accurately discriminated between the groups. However, it is very important to know what the initial diagnosis was, how BNP testing was used in arriving at it, and how well it correlated with the final diagnosis. This distinction may be crucial because accuracy of initial diagnosis and initiation of appropriate treatment may affect outcome and length of stay. Results of BNP testing are valuable in establishing or excluding diagnosis of congestive heart failure in patients presenting with acute dyspnea, with a diagnostic accuracy of 83% at a cutoff value of 100 pg/mL (2). Furthermore, addition of BNP values to clinical judgment improves the evaluation of acute dyspnea (3). Schneider and colleagues recommended the use of a heart failure nomogram (3) but did not provide further information on how accurately it was interpreted and applied in decision making, especially in view of the limited experience of the emergency department physicians at the participating hospitals—the test was ordered by cardiologists or performed only with approval from chemical pathologists.

It is difficult to conclude that BNP testing does not alter clinical outcomes without knowing how well BNP values were incorporated in arriving at the initial diagnosis and whether any discordance existed between initial and final diagnoses.

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References
TO THE EDITOR: We read with interest the article by Schneider and colleagues (1) about the use of BNP testing in emergency department patients with dyspnea. However, we believe that several key issues need clarification.

Schneider and colleagues reported no significant differences between BNP-tested and control groups in emergency department use of medications. It would be interesting to analyze how BNP-tested and non–BNP-tested groups were treated on the basis of final diagnosis to evaluate whether knowledge of BNP results (≤100 or >500 ng/L, used as cutoff values for exclusion or confirmation of heart failure diagnosis, respectively [2]) influenced therapeutic approach. If no differences are found between BNP-tested patients with heart failure and control participants, we propose 3 possible interpretations. First, as Schneider and colleagues suggest, the results of BNP analysis could have been available too late to influence physician decision making. Second, BNP testing was a new technique for emergency department physicians, and although knowing its theoretical meaning, they could have been insufficiently experienced with it to be influenced by its results. Third, as shown in another study (2), BNP testing is especially useful for intermediate-risk patients (pretest probability of heart failure between 21% and 79%), which amounted to 28% of participants with dyspnea enrolled in that study. Supposing a similar patient distribution in the study by Schneider and colleagues, there were probably too few intermediate-risk patients to reach statistical significance.

Finally, because BNP testing is used in clinical practice for the differential diagnosis of dyspnea, we think that enrolling patients either with a low dyspnea severity (who are discharged without doubt) or with a serious respiratory impairment (who are admitted regardless of the cause of dyspnea) could have masked a substantial reduction in admission rates, which would have been obtained by enrolling only patients with intermediate respiratory problems. It would be interesting to stratify patients into 3 severity subgroups according to respiratory rate or oxygen saturation and analyze whether BNP testing is useful in the intermediate-severity subgroup.

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Potential Financial Conflicts of Interest: None disclosed.

References
**Letters**

**Clinical Observation**

**A Novel Maneuver to Prevent Median Nerve Injury in Phlebotomy**

**Background:** Among complications associated with phlebotomy, nerve injury is relatively rare but potentially serious (1–3). Median nerve injury in particular is critical for patients (4, 5), because both motor and sensory neurons innervating the hand are involved. Appropriate practices to prevent median nerve injury have yet to be established, other than avoiding deep insertion of needles and excessive probing for veins (6) (although the antecubital fossa is the most common site for phlebotomy).

**Objective:** To use ultrasonography to determine the precise location of the median nerve in relation to major blood vessels in the antecubital fossa.

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### Table. Use of Medication in the Emergency Department for Patients With and Without Heart Failure Between BNP-Tested Group and Control Group

<table>
<thead>
<tr>
<th>Medication</th>
<th>Heart Failure (n = 274), n (%)</th>
<th>No Heart Failure (n = 338), n (%)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BNP Group (n = 148 [48.4%])</td>
<td>Control Group (n = 126 [41.2%])</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bronchodilator</td>
<td>39 (26.4)</td>
<td>32 (25.4)</td>
<td>0.86</td>
</tr>
<tr>
<td>Diuretic</td>
<td>94 (63.5)</td>
<td>87 (69.1)</td>
<td>0.34</td>
</tr>
<tr>
<td>Vasodilator</td>
<td>42 (28.4)</td>
<td>38 (30.2)</td>
<td>0.75</td>
</tr>
<tr>
<td>Antibiotic</td>
<td>41 (27.7)</td>
<td>32 (25.4)</td>
<td>0.67</td>
</tr>
<tr>
<td>Steroid</td>
<td>17 (11.5)</td>
<td>11 (8.7)</td>
<td>0.45</td>
</tr>
<tr>
<td>Morphine</td>
<td>10 (6.8)</td>
<td>7 (5.6)</td>
<td>0.68</td>
</tr>
<tr>
<td>Digoxin</td>
<td>6 (4.1)</td>
<td>6 (4.8)</td>
<td>0.75</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>0 (0)</td>
<td>2 (1.6)</td>
<td>0.12</td>
</tr>
<tr>
<td>ACE inhibitor</td>
<td>3 (2)</td>
<td>3 (2.4)</td>
<td>0.84</td>
</tr>
<tr>
<td>Noninvasive ventilation*</td>
<td>32 (21.6)</td>
<td>14 (11.1)</td>
<td>0.02</td>
</tr>
</tbody>
</table>

**ACE = angiotensin-converting enzyme; BNP = B-type natriuretic peptide.**

* Noninvasive ventilation includes bilevel positive airway pressure and continuous positive airway pressure.

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**References**


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**Potential Financial Conflicts of Interest:** None disclosed.

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**Methods:** A total of 134 arms of 67 healthy volunteers with a mean age of 39 years (range, 22 to 60 years) were investigated after informed consent was obtained. Two trained medical technologists conducted the examinations by using an ultrasonography scanner (Aplo 80, Toshiba Medical, Tokyo, Japan) with a 12-MHz linear transducer. To reproduce the usual conditions of venipuncture, the participants placed their arms in the supine position on the phlebotomy stand with slight arm-down positioning with or without tourniquet application. The following values were measured by ultrasonography: maximal sagittal and transverse thickness of the median nerve, distance between the median nerve and the brachial artery (value at the elbow crease and maximal value), distance between the median nerve and the closest major superficial vein at the elbow crease, and minimal distance between the median nerve and the skin (Figure). The institutional ethics committee approved our study.

**Results:** The median nerve thickness ranged from 1.4 mm to 2.4 mm in the sagittal direction and from 2.7 mm to 5.3 mm in the transverse direction. At the level of the elbow crease, the median nerve was located on the ulnar side of the brachial artery in all groups examined. The median distance between the radial edge of the median nerve and the ulnar edge of the brachial artery was 3.1 mm (range, 0 to 7.6 mm) with a tourniquet applied and 1.4 mm (range, 0 to 8.4 mm) without. No statistically significant differences were seen according to age, height, elbow circumference, or arm dominance. Scanning of the median nerve in the distal direction showed that the median nerve accompanies the brachial artery until finally submerging under muscles at the point where the brachial artery becomes impalpable. From the level of the elbow crease to this point, the distance between the median nerve and brachial artery remained less than 1 cm (range, 0 to 8.4 mm). The median distance between the median nerve and the closest major superficial vein at the elbow ranged from 0.3 to 32.4 mm (median, 5.2 mm) with a tourniquet applied and from 0.5 to 27.8 mm (median, 5.2 mm) without. The median nerve was located within 5 mm of the closest vein at the elbow in nearly one half of the groups examined (48%). The minimal distance between the median nerve and the skin ranged from 2.9 to 13.0 mm (median, 5.9 mm) with application of the tourniquet.

**Discussion:** Our study showed that the median nerve is frequently located close to the major blood vessels and the skin at the elbow. Taking the diameter of the median nerve into account, the
distance of 1.5 cm ulnar to the brachial artery in the antecubital fossa, where the brachial artery is palpable, is a “danger zone” for median nerve injury.

**Conclusion:** We strongly recommend that phlebotomists palpate the brachial artery before venipuncture and avoid puncture within 1 finger-width ulnar to the artery in the antecubital fossa.

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**References**

**Corrections**

**Correction: Prevalence of Cognitive Impairment Without Dementia**

In a recent article (1), an error was discovered in the total population estimate for the age group older than 90 years that was used to calculate the sample weights and the total number of individuals with cognitive impairment without dementia for 2002 in the United States. The sample weights have been recalculated by using the corrected population estimate, and the results in the article have been revised. A revised Table 3 (reprinted here) shows a slightly lower estimate of total prevalence of cognitive impairment without dementia due to the corrected smaller size of the age group older than 90 years. The revised weights do not change the conclusions reached by the authors, but they do slightly change many numeric values in the article. The revised sample weights and an explanation of the reason for their change can be found at http://hrsonline.isr.umich.edu/site/docs/userg/adams/ADAMSSampleWeights_Jun2009.pdf.
An error was also noted in the calculation of the association between apolipoprotein E and progression to dementia in the multivariate logistic regression model reported on page 432 in the Results section of the article. The revised analyses showed that the presence of at least 1 apolipoprotein E4 allele was significantly associated with progression to dementia (odds ratio, 4.05 [95% CI, 1.12 to 14.72]).

Reference

Correction: In The Clinic: Tuberculosis
The recent In The Clinic issue on tuberculosis (1) contained several misnumbered reference citations. On page ITC6-2, the citations for references 2 and 3 on lines 4 and 5 should be switched. On page ITC6-8, line 5 of the second column, the citation for reference 36 should be removed. On page ITC6-9, line 9 of the first column, reference 38 should be reference 32, and on line 30 of the first column, reference 37 should be reference 38. On page ITC6-10, line 51 of the second column, reference 39 should be reference 36. On page ITC6-11, line 48 of the first column, reference 43 should be reference 44, and on line 55 of the first column, reference 44 should be reference 45. On page ITC6-13, the Clinical Bottom Line box refers to Table 2, but this should be Table 5. The entry for reference 47 (on this same page) incorrectly lists the volume as 59, when it should be 58. Finally, there is a typographical error in the Toolkit on page ITC6-14. The IDSA is the Infectious Diseases Society of America, not the Infectious Disease Society of American College of Physicians. These errors have been corrected in the online version.

Reference

Table 3. Estimated National Prevalence of Cognitive Impairment Without Dementia, by Age Category*

<table>
<thead>
<tr>
<th>Age</th>
<th>All Cognitive Impairment Without Dementia (n = 241)</th>
<th>Prodromal Alzheimer Disease (n = 98)†</th>
<th>Vascular Cognitive Impairment Without Dementia and Stroke (n = 54)†</th>
<th>Medical Conditions (n = 55)†</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Prevalence (95% CI), %</td>
<td>Population Estimate (95% CI)</td>
<td>Prevalence (95% CI), %</td>
<td>Population Estimate (95% CI)</td>
</tr>
<tr>
<td>71–79 y</td>
<td>16.0 (11.5–20.5) 2.29 (1.65–2.94) 5.5 (2.6–8.4) 0.79 (0.37–1.20) 3.5 (1.4–5.6) 0.50 (0.20–0.80) 4.7 (1.2–8.3) 0.67 (0.17–1.19)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>80–89 y</td>
<td>29.2 (24.3–34.1) 2.41 (2.00–2.81) 9.7 (6.4–13.1) 0.80 (0.53–1.08) 10.1 (6.4–13.9) 0.83 (0.53–1.15) 5.4 (2.1–8.7) 0.45 (0.17–0.72)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥90 y</td>
<td>38.8 (25.6–52.0) 0.62 (0.41–0.84) 22.1 (11.8–32.3) 0.35 (0.19–0.52) 2.7 (0.0–6.0) 0.04 (0.0–0.10) 9.4 (0.2–18.6) 0.15 (0.04–0.30)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>22.0 (18.5–25.5) 5.33 (4.48–6.17) 8.1 (6.3–9.8) 1.96 (1.52–2.37) 5.7 (3.8–7.6) 1.38 (0.92–1.84) 5.3 (2.5–8.0) 1.28 (0.60–1.94)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Proportions are weighted. Population estimates in millions (and their CIs) were calculated by using the Aging, Demographics, and Memory Study population sampling weights. 
† “Prodromal Alzheimer Disease” includes amnestic mild cognitive impairment subtype. The 3 right columns are a subset of the column “All Cognitive Impairment Without Dementia.” The percentages across rows for these 3 subgroups do not equal the percentage for the “All Cognitive Impairment Without Dementia” group because the latter group includes additional subgroups not presented in the table.