

5. Babor TF, Kranzler HR, Lauerma RJ. Early detection of harmful alcohol consumption: comparison of clinical, laboratory, and self-report screening procedures. *Addict Behav* 1989;14:139-57.
6. Pokorny AD. Comment: validity of self-report in alcoholism research (letter). *Alcohol Clin Exp Res* 1989;13:299-300.
7. Forman SG, Linney JA. Increasing the validity of self-report data in effectiveness trials. *NIDA Res Monogr* 1991;107:235-47.
8. Brown RL. Identification and initial management of alcohol and drug abuse. In Fleming MF, Barry K, editors. *Addiction medicine: a practitioner's guide*. St Louis: Mosby Year-Book, 1992:25-43.
9. Brown RL. Alcohol and drug abuse. In Schiebeler LP, Mengel MB editors. *Ambulatory primary care handbook*. 2nd ed. Stamford, Conn: Appleton & Lange Publishing, 1996:509-16.

To the Editor: The recent article "Chronic Opioid Analgesic Therapy for Chronic Low Back Pain" by Brown et al¹ and the accompanying editorial by Terence Murphy² offer interesting views regarding the debate about the use of opioids for the treatment of chronic back pain. Brown et al appear to base their conclusion that chronic opioids should be considered a legitimate treatment of chronic low back pain on the self-reported responses to surveys given to patients on chronic opioid therapy. Is proper measure to be used, however, to assess the effectiveness of chronic opioids? Alcoholics in the midst of addiction will report that a drink calms them down, improves their thinking, and makes them feel better. Patients when first attempting to stop smoking will frequently have considerable deleterious mental and physical symptoms. If the patient's sense of well-being is the criterion upon which physicians should base treatment decisions, then the argument can be made that physicians should not advise patients to discontinue alcohol or tobacco if using these substances make them feel better. Most physicians would recognize that an alcoholic's or smoker's view of how the drug is affecting his or her life is unreliable at best.

Dr. Murphy in his editorial touches upon what should be the true measure of the effects of opioids on chronic pain. His position discouraging the use of opioids is based upon his observations that patients who enter his chronic pain program are frequently impaired in their ability to participate in rehabilitation because of their medication use. Functional ability, not self-reported relief of symptoms, should be the benchmark by which opioid usage is measured. A more convincing argument supporting the use of opioids would have been made had Brown et al shown that the use of opioids decreased lost work days, improved rehabilitation potential, or returned previously disabled individuals to the work force. The data presented by Brown et al do not appear to address these issues.

It is important periodically to reexamine commonly held beliefs to see whether these beliefs continue to hold up under the scrutiny of our continually expand-

ing body of knowledge. Brown et al make the case that opioids might need to be considered in the treatment of chronic low back pain, but the evidence to support its effectiveness is lacking. Until studies showing improved functional abilities of those treated with opioids are forthcoming, I will continue to approach the use of narcotics for chronic conditions with extreme caution and skepticism.

Jerry Ryan, MD
University of Wisconsin-Madison

References

1. Brown RL, Fleming MF, Patterson JJ. Chronic opioid analgesic therapy for chronic low back pain. *J Am Board Fam Pract* 1996;9:191-204.
2. Murphy TM. Chronic opioids for chronic low back pain—solution or problem? *J Am Board Fam Pract* 1996;9:225-8.

To the Editor: The abstract and the concluding summary of the paper of Brown et al on the prescription of oral opioid analgesics for chronic backache patients suggest that the authors feel comfortable with recommending wider use of this treatment modality.¹ The intervening 10 pages of text, however, contain numerous important caveats about their use, along with warnings about patient subgroups for whom maintenance opioid therapy is clearly contraindicated. Additional reservations are presented in a related editorial in the same issue of the *Journal*.²

Risks and restrictions aside, Brown et al offer little evidence that opioids help backache or other chronic pain patients. They note a lack of adequate published studies on the subject, and they describe the outcome assessment in the principal relevant uncontrolled publication³ as "vague."

A recently published double-blind crossover study using oral sustained-release morphine showed statistically significant benefits and would appear, at first glance, to support the position of Brown et al.¹ Careful inspection of the figure accompanying the paper of Moulin et al,⁴ however, reveals a disturbing pattern: patients reported striking improvement during an initial 3-week titration period, but this benefit appeared to diminish gradually but inexorably during the subsequent 6-week evaluation period. It appears likely that the ratings would have approached placebo levels by 12 weeks after treatment onset had the study been continued for that length of time. This observation is consistent with the known tendency for the benefits of opioid treatment to diminish with time unless dosage is escalated.

Given the known salience of psychosocial factors in pain disorder,⁵ the weakness of the evidence for sustained efficacy of opioid use in these patients, the increasing reluctance of third party payers to underwrite treatment of unproved value, and the time-tested prescription of interventions that might harm patients, more liberal use of opioids in this population seems unwise. Physicians who choose to try it might be wise

to address each case as an "n of one experiment" requiring regular and rigorous assessment for beneficial and adverse effects.

Robert D. Gillette, MD
Poland, Ohio

References

1. Brown RL, Fleming ME, Patterson JJ. Chronic opioid analgesic therapy for chronic low back pain. *J Am Board Fam Pract* 1996;9:191-204.
2. Murphy TM. Chronic opioids for chronic low back pain—solution or problem? *J Am Board Fam Pract* 1996;9:225-8.
3. Taub A. Opioid analgesics in the treatment of chronic intractable pain of nonneoplastic origin. In: Kitahata LM. *Narcotic analgesics in anesthesiology*. Baltimore, Williams & Wilkins, 1981:199-208.
4. Moulin DE, Iezzi A, Amireh R, Sharpe WKJ, Boyd D, Merskey H. Randomised trial of oral morphine for chronic non-cancer pain. *Lancet* 1996;347:143-7.
5. Eisendrath SJ. Psychiatric aspects of chronic pain. *Neurology* 1995;45(12 Suppl 9):S26-34.

The Dynamically Evolving Field of Cervical Cancer Screening

Cervical cancer screening is a dynamic and changing field. At the 9th World Congress of Cervical Pathology and Colposcopy held in May in Sydney, Australia, five relatively new methods were presented as potential ancillary tools for cervical cancer screening. These included human papillomavirus (HPV) testing, cervicography, speculscopy, polar probe, and immunofluorescence. In addition to ancillary tools, there was a prototype of a video colposcope with superior illumination. Cytology screening is also undergoing a technical revolution with automated systems for processing, quality assurance, and preliminary screening. As technology becomes more accessible and understood, and as research tools are modified, the nature of cancer screening in general changes.

Unfortunately, Holman chose to use an outdated technology for HPV typing in his study.¹ The Virapap and Viratype tests were the first commercially available prototype for HPV testing, testing for a limited number of mucosotropic HPV types. As with any technology, initially there are difficulties translating the collection and detection devices to widespread clinical practice. These difficulties have been documented previously for this first-generation HPV test.²⁻¹⁰

The second-generation HPV detection kit approved by the Federal Drug Administration, Hybrid Capture, has been available since March of 1995. This test¹¹⁻¹⁴ shows marked improvement compared with the first-generation test. The low-risk groups detectable include types 6, 11, 42, 43 and 44. The intermediate- and high-risk groups are combined together to detect types 16, 18, 31, 33, 35, 39, 45, 51, 52, 56 and 58. In addition, the Hybrid Capture method has a chemiluminescent reaction that quantifies the amount of HPV DNA present.

The third-generation HPV detection kit is not yet

commercially available, as it is still undergoing testing. It promises to improve the detection and quantification of HPV DNA oncogenic types.

Whether HPV testing is used as a triage tool¹¹ or as a primary screening tool,¹⁵ the utility of HPV testing has yet to be proved in a general clinic for all age ranges. The ALTS trial sponsored by the National Cancer Institute will attempt to answer this question in the United States. Women entering this trial will be randomized to immediate colposcopy, repeat cytology, or HPV testing after an initial ASCUS or LSIL Papanicolaou smear. Two similar trials are currently being conducted in Canada. The form of the final technology for HPV testing is still evolving, as are the methods for taking Papanicolaou smears, cytologic processing, and reading. Current research must evaluate the most evolved technologies for improved clinical performance.

Diane M. Harper, MD, MPH
University of Missouri-Kansas City

References

1. Holman JR. Detection of human papillomavirus DNA in patients referred to a family practice colposcopy clinic. *J Am Board Fam Pract* 1996;9:162-6.
2. Lorincz AT, Temple GF, Patterson JA, Jensen AB, Kurman RJ, Lancaster WD. Correlation of the cellular atypia and human papillomavirus deoxyribonucleic acid sequences in exfoliated cells of the uterine cervix. *Obstet Gynecol* 1986; 68:508-12.
3. Reid R, Greenberg MD, Lorincz AT, Jensen AB, Laverly CR, Husain M, et al. Should cervical cytologic testing be augmented by cervicography or human papillomavirus deoxyribonucleic acid detection? *Am J Obstet Gynecol* 1991; 164:1461-9.
4. Cole HM. Human papillomavirus DNA testing in the management of cervical neoplasia. *JAMA* 1993;270:2975-81.
5. Moscicki AB, Palefsky JM, Gonzales J, Schoolnick GK. The association between human papillomavirus deoxyribonucleic acid status and the results of cytologic rescreening tests in young, sexually active women. *Am J Obstet Gynecol* 1991; 165:67-71.
6. Weintraub J, Redard M, Seydoux J. The comparative test performance of dot filter hybridization (Viratype) and conventional morphologic analysis to detect human papillomavirus. *Am J Clin Pathol* 1992;97:46-57.
7. Burner GC, Parker JD, Bates J, East K, Kulander BG. Comparative analysis of human papillomavirus detection by polymerase chain reaction and Virapap/Viratype kits. *Am J Clin Pathol* 1990;94:554-60.
8. Meyer MP, Carbonell RI, Mauser NA, Kanbour AI, Amortegui AJ. Detection of human papillomavirus in cervical swab samples by ViraPap and in cervical biopsy specimens by in situ hybridization. *Am J Clin Pathol* 1993;100:12-7.
9. Corliss J. Utility of ViraPap remains to be established. *J Natl Cancer Inst* 1990;82:252-3.
10. Guerrero I, Daniel RW, Bosch FX, Castellsague X, Munoz N, Gili M, et al. Comparison of ViraPap, Southern hybridization, and polymerase chain reaction methods for human papillomavirus identification in an epidemiological investigation of cervical cancer. *J Clin Microbiol* 1992;30: 2951-9.