Transmission of HPV

John W. Sellors and colleagues[1] have described human papillomavirus (HPV) infection in women, relating this infection to various risk factors, including number of previous sexual partners. It should be pointed out, however, that HPV is not a typical sexually transmitted infection.

As the recent paper by Winer and associates[2] highlighted, sexual contact is not necessary for the transmission of HPV. Although these authors showed that the cumulative incidence of HPV over the first 4 years after first sexual intercourse was about 50%, they also showed that HPV infection was acquired by virginal women at a cumulative rate of 7.9% over 2 years. According to these authors, abstaining from penetrative sex did not protect women from HPV transmission, and they proposed that skin-to-skin contact during nonpenetrative sexual contact may be a primary mode of genital HPV transmission.

Furthermore, no protective effect has been associated with condom use.[3] This failure to prevent HPV may be related to the poor validity of self-reported condom use, condom breakage, slippage and incorrect use, but it may also be caused by the ability of biological material to pass through condoms.[4]

Perhaps researchers should move away from collecting data on the number of previous sexual partners a woman has had, especially given that data of this type help to stigmatize HPV as a virus affecting only promiscuous women who have unprotected penetrative sex.

Sarah Giles
Class of 2005
Dalhousie University
Halifax, NS

References

1. Sellors JW, Karwalajtys TL, Kaczorowski J, Mahony JB, Lytwyn A, Chong S, et al, for the Survey of HPV in Ontario Women (SHOW) Group. Incidence, clearance and predictors of human papillomavirus infection in women. *CMAJ* 2003;168(4):421-5.
2. Winer RL, Lee SK, Hughes JP, Adam DE, Kiviat NB, Koutsky LA. Genital human papillomavirus infection: incidence and risk factors in a cohort of female university students. *Am J Epidemiol* 2003;157:218-26.
3. Steiner MJ, Feldblum PJ, Padian N. Invited commentary: condom effectiveness — will prostate-specific antigen shed new light on this perplexing problem? *Am J Epidemiol* 2003;157: 298-300.
4. Macaluso M, Lawson ML, Duerr A, Hortin G. Macaluso et al. respond to "Condom effectiveness and prostate specific antigen." *Am J Epidemiol* 2003;157:301-2.

[Four of the authors respond:]

As Sarah Giles points out, nonpenetrative sexual activity is associated with increased risk of genital HPV infection.[1] We did not define sexual activity when we asked women to report the number of their sexual partners.[2] However, it is likely that at least some respondents included partners with whom sexual activity was nonpenetrative.[3]

We also agree with Giles that evidence for the protective effect of condoms against HPV infection is lacking.[4]

Giles raises an important issue regarding the potential stigmatization of women with genital HPV infection. Approximately 65% of women (or more) have been infected with HPV sometime in their lives, the vast majority of these infections being transient.[5] It is reasonable to assume that the same proportion of men are infected, given that the risk factors for genital HPV infection are similar in men and women.[6] Such infection is therefore widespread and common, especially at younger ages. To assume that any particular infected individual has had numerous sexual partners is wrong. Although an increasing number of partners does increase the risk of infection, sole contact with one infected partner can lead to acquisition of genital HPV.[7]

Nevertheless, as research has shown, a certain proportion of women and men with HPV infection have had numerous sexual partners.[2,6,7] A MEDLINE search for the period January 1966 to March 2003, using "human papillomavirus" as a subject heading and "promiscuity" as a keyword, identified 7 articles that used the word "promiscuous" in the abstract when referring to such a sexual history in people infected with genital HPV. We believe that terms such as this one are morally charged and judgement laden, and that they should be avoided by physicians and researchers.

Alice Lytwyn
Department of Pathology
Joseph Brant Memorial Hospital
Burlington, Ont.
Janusz Kaczorowski
Department of Family Medicine
McMaster University
Hamilton, Ont.
Attila Lorincz
Digene Corporation
Gaithersburg, Md.
John W. Sellors
Program for Appropriate Technology in Health
Seattle, Wash.

References

1. Winer RL, Lee SK, Hughes JP, Adam DE, Kiviat NB, Koutsky LA. Genital human papillomavirus infection: incidence and risk factors in a cohort of female university students. *Am J Epidemiol* 2003;157:218-26.
2. Sellors JW, Karwalajtys TL, Kaczorowski J, Mahony JB, Lytwyn A, Chong S, et al, for the Survey of HPV in Ontario Women (SHOW) Group. Incidence, clearance and predictors of human papillomavirus infection in women. *CMAJ* 2003;168(4):421-5.
3. Sanders SA, Reinisch JM. Would you say you "had sex" if....? *JAMA* 1999;281:275-7.
4. Manhart LE, Koutsky LA. Do condoms prevent genital HPV infection, external genital warts, or cervical neoplasia? A meta-analysis. *Sex Transm Dis* 2002;29:725-35.
5. Koutsky LA. Epidemiology of human papillomavirus infection. *Am J Med* 1997;102(5 Suppl 1):3-8.
6. Svare EI, Kjaer SK, Worm AM, Osterlind A, Meijer CJLM, van den Brule AJC. Risk factors for genital HPV DNA in men resemble those found in women: a study of male attendees at a Danish STD clinic. *Sex Transm Infect* 2002;78: 215-8.
7. Burk RD, Ho GY, Beardsley L, Lempa M, Peters M, Bierman R. Sexual behavior and partner characteristics are the predominant risk factors for genital human papillomavirus infection in young women. *J Infect Dis* 1996;174:679-89.

ECT for Parkinson's?

Mark Guttman and associates,[1] in their review of diagnosis and management of Parkinson's disease, make no reference to electroconvulsive therapy (ECT) as an option for patients with insufficient response to pharmacotherapy.

The most contemporary and authoritative review of psychiatric practice in this field[2] strongly endorses the use of ECT for the management of refractory Parkinson's disease, citing numerous references from the neurology and psychiatry literature in support of this endorsement. Many psychiatrists who administer ECT are aware of this literature.

I would appreciate the authors' comments on the available evidence for the effectiveness of ECT in Parkinson's disease. If warranted, ECT should then be given its appropriate place in the treatment algorithm for this illness.

B.A. Martin
Head, ECT Service
Centre for Addiction and Mental Health
Toronto, Ont.

References

1. Guttman M, Kish SJ, Furukawa Y. Current concepts in the diagnosis and management of Parkinson's disease. *CMAJ* 2003;168(3):293-301.
2. Task Force on ECT (Weiner RD, chairperson). *The practice of electroconvulsive therapy: recommendations for treatment, training and privileging.* 2nd ed. Washington: American Psychiatric Association; 2001.

[One of the authors responds:]

Although we did not mention ECT in our article,[1] we agree that it may have a role in the treatment of specific symptoms of Parkinson's disease.

Parkinsonian patients who are severely depressed and whose condition is refractory to antidepressant therapy are candidates for ECT to treat their depression. Patients with drug-induced psychosis that is resistant to atypical neuroleptic medication who cannot tolerate reductions in their antiparkinsonian medication may also be candidates for ECT. However, ECT should not be offered to patients with dementia because there is the potential that such treatment may cause worsening of cognition and may induce delirium. There is insufficient evidence to suggest that motor symptoms related to Parkinson's disease should be treated with ECT, and in our opinion this should not be considered an indication for its use.

Mark Guttman
Departments of Medicine and Psychiatry
University of Toronto
Toronto, Ont.

Reference

1. Guttman M, Kish SJ, Furukawa Y. Current concepts in the diagnosis and management of Parkinson's disease. *CMAJ* 2003;168(3):293-301.

The return of "negative" trials?

I was surprised that several important issues were not addressed in the original reports[1,2] and editorial[3] about rate versus rhythm control in atrial fibrillation published in the *New England Journal of Medicine*, or in the review[4] and editorial[5] published subsequently in *CMAJ*.

The Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM) investigators found no statistically significant difference between rhythm control and rate control.[1] However, one cannot rule out the possibility of a type II error, given that a sample size of 5300 was planned[6] but only 4060 patients were enrolled in the study.

In the noninferiority study by Van Gelder and associates,[2] the efficacy of rate control was within the upper bound of the 95% confidence interval of that of rhythm control. However, 3 concerns must be addressed.

First, it is not clear if the rhythm control strategy is a suitable active comparator. Neither the authors nor the practice guidelines cited[7] provided details on any earlier trials that showed rhythm control to be consistently better than placebo. Thus, it is not possible to assess the similarity of the current trial to those earlier trials, the expected effect size of rhythm control relative to placebo[8] or the consistent responsiveness to rhythm control of the composite endpoint components[9] used in the current trial.