parents given smoking advice were as a result of the child suffering with acute otitis media. In addition, we could only identify parents who were registered with our practice. There was no way of including adults in the home who were not parents or guardians.

This audit was presented to the partners, and a plan was made to put up reminders to discuss smoking with the parents of any children presenting with acute otitis media. The audit is to be repeated in 1 year to allow sufficient numbers of cases to present.

We realise that this is just one of many motivational factors that can be used to encourage patients to stop smoking, but smoking cessation is such a high priority that this window of opportunity should not be overlooked. Prevention rather than prescriptions must remain our ideal in the management of otitis media.

Ryan Kerstein
Royal Free Hospital, General Practice, Pond Street, London.
E-mail: ryan.kerstein@gmail.com

REFERENCES

1. Worrall G. Acute otitis media. *Can Fam Physician* 2007; **53(12):** 2147–2148.
2. Diagnosis and management of acute otitis media. *Pediatrics* 2004; **113**: 1451–1465.
3. Spiro DM, Tay KY, Arnold DH, *et al*. Wait-and-see prescription for the treatment of acute otitis media: a randomized controlled trial. *JAMA* 2006; **296(10)**: 1235–1241.
4. Uhari M, Myantysaari K, Niemelya M. A meta-analytic review of the risk factors for acute otitis media. *Clin Infect Dis* 1996; **22**:1079–1083.

DOI: 10.3399/bjgp08X280281

How can we remove barriers to HIV testing outside of a GUM setting?

Approximately 32% of patients infected by HIV in the UK remain undiagnosed.[1] Delayed HIV diagnosis is responsible for HIV presentation at lower CD4 T-cell count and such patients respond less well to antiretroviral therapy.[2] At least 35% of HIV-related deaths in 2005/6 in the UK were attributed to late diagnosis of infection.[3] Furthermore, delays in HIV diagnosis and initiation of antiretroviral therapy contribute to horizontal and vertical transmission of HIV infection.[4,5]

A recent study examined the factors which were significantly associated with GUM clinic patients (not exclusively attending for HIV testing) agreeing to GP contact. These factors included heterosexual orientation, initial GP referral, and not considering HIV testing to have negative implications for future mortgage and life insurance applications.[6]

Two factors have been reported to us that impair the ability of non-genitourinary practitioners, both in primary care and other specialist care settings, to perform HIV testing. The first is pre-test counselling. We would argue that, as in other disease areas, the pre-test counselling is no longer necessary as there are clear health benefits in knowing about an HIV diagnosis which outweigh perceived disadvantages. This is consistent with a general move towards 'opt out' HIV testing in GUM clinics and antenatal services.[7] In rare, high risk, or acutely unwell cases, pre-test counselling may be the preferred option, but for the majority of patients it is not required. The second barrier cited is that of transparency about HIV testing for insurance company medical reports. However, the GP and insurance applicants are not required to notify insurers when negative tests are performed.[8]

The life expectancy of a 25-year-old HIV-positive person, who is hepatitis C negative, has been estimated to be greater than 35 years[9] and this will increase as newer anti-retroviral drugs become available. Like other chronic and manageable conditions, an early diagnosis is essential to maximise individual and community health but this can only be achieved by the removal of barriers to widespread HIV testing across all hospital departments and primary care. We urge that the earlier diagnosis of HIV infection is made a clear priority and that the role of specialist genitourinary clinicians to enable better training, clear referral pathways, and the destigmatisation of testing in all care settings are key parts of the development of local sexual health networks.

Tristan J Barber
Specialist Registrar GUM/HIV

Anatole Menon-Johansson
Specialist Registrar GUM/HIV

Simon Barton
Clinical Director HIV/GUM Directorate
St Stephen's Centre, Chelsea and Westminster NHS Foundation Trust, London.
E-mail: simon.barton@chelwest.nhs.uk

REFERENCES

1. UK Collaborative Group for HIV and STI Surveillance. *A complex picture. HIV and other sexually transmitted infections in the United Kingdom: 2006.* London: Health Protection Agency, Centre for Infections, 2006.
2. Palella FJ Jr, Deloria-Knoll M, Chmiel JS, *et al*. Survival benefit of initiating antiretroviral therapy in HIV-infected persons in different CD4+ cell strata. *Ann Intern Med* 2003; **138**: 620–626.
3. Johnson M, Skinner C, O' Mahony C. Mortality Audit. BHIVA Audit and Standards Sub-Committee. http://www.bhiva.org/files/file1001379.ppt (accessed 14 Apr 2008).
4. Wawer MJ, Gray RH, Sewankambo NK, *et al*. Rates of HIV-1 transmission per coital act, by stage of HIV-1 infection, in Rakai, Uganda. *JID* 2005; **191:** 1403–1409.
5. Pilcher CD, Tien HC, Eron JJ, *et al*. Brief but efficient: acute HIV infection and the sexual transmission of HIV. *JID* 2004; **189:** 1785–1792.
6. Fernando I, Clutterbuck D. Genitourinary medicine clinic and general practitioner contact: what do patients want? *Sex Transm Infect* 2008; **84:** 67–69.
7. Collini P. Opt-out HIV testing strategies. BMJ, 2006. http://clinicalevidence.bmj.com/downloads/2.Opt-out HIV testing strategies.pdf (accessed 14 Apr 2008).
8. Association of British Insurers. *Gay men and insurance*. London: ABI, 2005. http://www.abi.org.uk./Display/default.asp?Menu_ID =1140&Menu_All=1,946,1140&Child_ID=656 (accessed 14 Apr 2008).
9. Lohse N, Hansen AB, Pedersen G, *et al*. Survival of persons with and without HIV infection in Denmark, 1995–2005. *Ann Intern Med* 2007; **146**: 87–95.

DOI: 10.3399/bjgp08X280308

Benzodiazepine tolerance, dependency, and withdrawal syndromes and interactions with fluoroquinolone antimicrobials

I investigated reports of an abnormally high incidence of adverse reactions to

fluoroquinolone antibiotics in patients dependent on and withdrawing from benzodiazepines. Participants of an online benzodiazepine withdrawal support group (www.thetrap.org.uk) who reported fluoroquinolone use were asked to fill out a structured questionnaire. Eleven participants reported severe or very severe adverse reactions, one participant reported a moderate adverse reaction, and a further participant reported no reaction to fluoroquinolone treatment. In most cases adverse symptoms resolved within 1 month of cessation of fluoroquinolone dosing. However, in some cases the symptoms persisted with gradual improvement for a period of several months. All participants reported adverse effects similar to those of acute benzodiazepine withdrawal which included depression, anxiety, psychosis, paranoia, severe insomnia, paraesthesia, tinnitus, hypersensitivity to light and sound, and tremors. Four patients became acutely suicidal.

One participant, a female aged 44 years, who had detoxed off high prescribed doses of benzodiazepines 3 months previously, experienced an acute psychotic reaction within 1 hour of commencing norfloxacin and attempted suicide. Her condition quickly deteriorated and she developed repeated seizures; this progressed to status epilepticus, which failed to respond to treatment in ICU. Worryingly, her medical attendants continued to prescribe norfloxacin while in ICU. Her seizures were only controlled after finishing her norfloxacin course.

Chronic use of benzodiazepines causes compensatory adaptions which cause GABA receptors to become less sensitive to GABA. On discontinuation of benzodiazepines, withdrawal symptoms typically develop which may persist for weeks or months.[1] Antagonism of the $GABA_A$ receptor is believed to be responsible for the CNS toxicity of fluoroquinolones affecting 1–4% of patients treated.[2] Fluoroquinolones have also been found to inhibit benzodiazepine receptor binding.[3] The results of this small study seem to confirm that adverse reactions to fluoroquinolones occur more frequently in the benzodiazepine-dependent population than the 1–4% seen in the general public and may be severe.

Possible explanations for the adverse fluoroquinolone-induced reactions in the current reported patient group include:

- Fluoroquinolones compete directly with benzodiazepines for the benzodiazepine receptor site displacing benzodiazepines and precipitating an acute withdrawal effect.
- Alterations in the $GABA_A$–benzodiazepine receptor complex (during benzodiazepine tolerance/dependency status) may increase fluoroquinolone-induced stimulation of the receptor complex.
- Benzodiazepine dose-tapering and/or cessation might be associated with GABAergic underactivity but rebound neuro-excitation following fluoroquinolone exposure.

Participants were asked in the questionnaire about medication and alcohol or drug usage at time of adverse reaction. None of the participants reported anything which could explain their adverse reaction apart from the introduction of a fluoroquinolone. Physicians should, wherever possible, avoid fluoroquinolones in patients who are dependent on or withdrawing from chronic benzodiazepines.

John Girvan McConnell
Ulster Hospital,Care of the Elderly, Dundonald, Belfast.
E-mail: girvin.mcconnell@setrust.hscni.net

REFERENCES

1. Ashton H. Protracted withdrawal syndromes from benzodiazepines. *J Subst Abuse Treat* 1991; **8:** 19–28.
2. Halliwell RF, Davey PG, Lambert JJ. Antagonism of GABAA receptors by 4-quinolones. *J Antimicrob Chemother* 1993; **31:** 457–462.
3. Unseld E, Ziegler G, Gemeinhardt A, *et al*. Possible interaction of fluoroquinolones with the benzodiazepine-GABAA-receptor complex. *Br J Clin Pharmacol* 1990; **30:** 63–70.

DOI: 10.3399/bjgp08X280317

Correction

In the April 2008 issue of the *BJGP* we incorrectly published the key of Figure 1 in the following letter:

> Thurlow VR, Bailey IR, Payne NM. Centralised pathology services. *Br J Gen Pract* 2008; **58(549):** 278–279.

In Figure 1 (Effect of temperature and phlebotomy on the incidence of hyperkalaemia) the labels for the key were transposed. We apologise for this error. The corrected version is available online.

DOI: 10.3399/bjgp08X280326