a molecule-dependent target specificity: mutations in *parC* are generally selected by pefloxacin, ciprofloxacin, and levofloxacin, and those in *gyrA* are selected by sparfloxacin, gatifloxacin, moxifloxacin, gemifloxacin, and garenoxacin (*5*). In second-step mutants, mutations are present in both *parC* and *gyrA* and confer resistance to the antistreptococcal FQs levofloxacin, moxifloxacin, and gatifloxacin.

FQ resistance in GBS has been reported in Japan, the United States, and Spain (*6*–*8*). Up to now, all FQ-resistant GBS strains described were highly resistant because of point mutations in *gyrA* and *parC* QRDR; a *parC* mutation at position 79 was present in all strains. These strains were isolated from elderly adults who, in some cases, had received quinolone therapy. Low-level resistance to FQ in GBS CNR0717 was associated with a Ser 79 → Tyr mutation in *parC*. Therefore, although the FQ sensitivity of this strain is unknown, a first-step mutant could have been selected in vivo as our patient was treated with levofloxacin for 2 weeks.

GBS is an unusual cause of acute bacterial exacerbation of chronic bronchitis compared with other respiratory pathogens such as *S. pneumoniae,* but pathologies associated with this bacterium are changing. Clinical microbiologists should be aware of these changes and test isolates of *Streptococcus* spp. for susceptibility to FQs. This report indicates that FQ resistance among streptococci is a growing concern and that levofloxacin can select in vivo *parC* first-step mutants that will facilitate emergence of high-level FQ-resistant GBS strains, as demonstrated in vitro for *S. pneumoniae* (*9*). Finally, although FQ treatment is recommended for high-risk groups with acute exacerbations of chronic bronchitis, these antimicrobial drugs must be reserved for situations in which there are no effective alternative drugs to treat infections caused by multidrug-resistant bacteria. For susceptible strains, β-lactams, which still constitute the first-line recommended antimicrobial drugs, should be used for treatment of these patients (*10*).

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### References

1. Blasi F, Ewig S, Torres A, Huchon G. A review of guidelines for antibacterial use in acute exacerbations of chronic bronchitis. Pulm Pharmacol Ther. 2006;19:361–9.
2. Schuchat A. Group B streptococcus. Lancet. 1999;353:51–6.
3. Clinical and Laboratory Standards Institute. Performance standards for antimicrobial susceptibility testing. Approved standard M100–S17. Wayne (PA): The Institute; 2007.
4. Hooper DC. Fluoroquinolone resistance among gram-positive cocci. Lancet Infect Dis. 2002;2:530–8.
5. Houssaye S, Gutmann L, Varon E. Topoisomerase mutations associated with in vitro selection of resistance to moxifloxacin in *Streptococcus pneumoniae.* Antimicrob Agents Chemother. 2002;46:2712–5.
6. Kawamura Y, Fujiwara H, Mishima N, Tanaka Y, Tanimoto A, Ikawa S, et al. First *Streptococcus agalactiae* isolates highly resistant to quinolones, with point mutations in *gyrA* and *parC.* Antimicrob Agents Chemother. 2003;47:3605–9.
7. Miro E, Rebollo M, Rivera A, Alvarez MT, Navarro F, Mirelis B, et al. *Streptococcus agalactiae* highly resistant to fluoroquinolones. Enferm Infecc Microbiol Clin. 2006;24:562–3.
8. Wehbeh W, Rojas-Diaz R, Li X, Mariano N, Grenner L, Segal-Maurer S, et al. Fluoroquinolone-resistant *Streptococcus agalactiae*: epidemiology and mechanism of resistance. Antimicrob Agents Chemother. 2005;49:2495–7.
9. Perez-Trallero E, Marimon JM, Gonzalez A, Ercibengoa M, Larruskain J. In vivo development of high-level fluoroquinolone resistance in *Streptococcus pneumoniae* in chronic obstructive pulmonary disease. Clin Infect Dis. 2005;41:560–4.
10. Hayes D, Meyer KC. Acute exacerbations of chronic bronchitis in elderly patients: pathogenesis, diagnosis and management. Drugs Aging. 2007;24:555–72.

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## Dengue and Relative Bradycardia

**To the Editor:** In a recent letter to Emerging Infectious Diseases, Lateef and colleagues identified a relationship between dengue and relative bradycardia in patients in Singapore. They stated that "To our knowledge, this sign has not been previously associated with dengue" (*1*). Unfortunately, the association of dengue fever with relative bradycardia has already been well established and is certainly not a new finding (*2*,*3*). Despite this, however, there is no harm done in reinforcing an often forgotten clinical sign that can assist in the diagnosis of dengue, especially in those countries with limited resources.

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### References

1. Lateef A, Fisher DA, Tambyah PA. Dengue and relative bradycardia. Emerg Infect Dis. 2007;13:650.
2. Senanayake S. Dengue fever and dengue haemorrhagic fever — a diagnostic challenge. Aust Fam Physician. 2006;35: 609–12.
3. Wittesjo B, Bjornham A, Eitrem R. Relative bradycardia in infectious diseases. J Infect. 1999;39:246–7.


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## Importation of Poliomyelitis by Travelers

**To the Editor:** In July 2007, an Australian traveler imported polio from Pakistan to Australia (*1*). He was a 22-year-old man who had immigrated to Australia and had traveled to his country of origin (Pakistan) to visit friends and relatives. Pakistan is one of 4 countries (Afghanistan, India, Nigeria, Pakistan) where polio is still endemic. A diagnosis of polio was made shortly after his return to Australia. Australia was certified as polio-free in 2000. Australia will not be the last industrialized country affected by importation of polio. All countries are at risk until polio has been completely eradicated.

Between 2003 and 2006, polio was imported by travelers (e.g., refugees, pilgrims, traders) to 24 polio-free countries (*2*). The origin of these importations was largely the 4 countries where polio transmission was never completely interrupted. The importations resulted in about 1,400 secondary cases (*2*). The resurgence of polio by international spread was a setback to the Global Polio Eradication Initiative that had successfully decreased the number of polio-affected countries to only 9 in 2002.

The revised International Health Regulations, IHR (2005) (*3*), entered into legal force on June 15, 2007. These regulations provide the legal framework for coordination of the international effort to reduce or prevent international spread of diseases of public health concern. IHR (2005) (*2*) lists polio as one of the diseases of public health emergencies of international concern. Preventing importation of polio into polio-free countries is therefore a test case for the revised International Health Regulations (*4*). Compared to the previous IHR (1969), IHR (2005) has moved away from the definition of fixed maximum measures relating to specific diseases and instead focuses on the issuance of context-specific recommendations, made either on a temporary emergency basis (a temporary recommendation) or routinely for established ongoing risks of disease spread (a standing recommendation).

One strategy to protect polio-free countries from reintroduction of wild poliovirus is by requiring proof of polio vaccination for all incoming travelers from polio-endemic countries. This was proposed by the Advisory Committee on Poliomyelitis Eradication in October 2006. The rationale is similar to that used for yellow fever, currently the only disease for which proof of vaccination may be required for travelers as a condition of entry to a country. The proposal of the Advisory Committee of Poliomyelitis Eradication was discussed at the World Health Assembly in May 2007 (*5*). Although the main strategy for polio eradication continues to be attaining high vaccination coverage against polio in all countries, the 193 member states have also adopted the resolution to "continue to examine and disseminate measures that member states can take for reducing the risk and consequences of international spread of polioviruses, including, if and when needed, the consideration of Temporary or Standing Recommendations, under the International Health Regulations (2005)" (*3*).

The recent polio importation by an inadequately vaccinated traveler would add impetus to such considerations. However, this case also shows that focusing on travelers from polio-endemic countries alone may not be sufficient. Immigrants from developing countries to industrialized countries who subsequently return to their home countries to visit friends and relatives may also be at increased risk if traveling to polio-endemic countries, in particular as many may not have received adequate childhood vaccination including vaccination against polio (*6*). Targeting those visiting friends and relatives is therefore a potential additional strategy to reduce the risk for the worldwide spread of polio.

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### References

1. Department of Health and Public Aging. Public health alert over positive case of polio in Australia media release. Australian Government [cited 2007 Jul 13]. Available from http://www.health.gov.au/internet/wcms/publishing.nsf/Content/health-mediarel-cmo-130707
2. Conclusions and recommendations of the Advisory Committee on Poliomyelitis Eradication, Geneva, 11–12 Oct 2006, Part I. Wkly Epidemiol Rec. 2006;81: 453–60.
3. World Health Organization. International Health Regulations 2005 [cited 2007 Nov 9]. Available from http://www.who.int/csr/ihr/en/
4. Hardiman M, Wilder-Smith A. The revised international health regulations and their relevance to travel medicine. J Travel Med. 2007;14:141–4.