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Is the natural history of Clostridium tetani such that protective levels of immunity might be maintained in those—for example, gardeners and builders—who by the nature of their work are continually suffering minor lacerations. How ubiquitous is the organism in its active and latent forms? Is there any good evidence that active immunisation should be at five rather than 10 year intervals?

Tetanus is due to the production of toxin by *Clostridium tetani* under the anaerobic conditions present in wounds. Even those who have recovered from tetanus do not gain reliable immunity and active immunisation is therefore essential especially for those continually suffering minor lacerations that may be contaminated. The organism is widespread in soil contaminated with animal excreta (including wild animals) making, for example, football pitches a source of infection as well as domestic gardens and fields grazed by farm animals. A full basic course of adsorbed vaccine (three "primary" doses in infancy followed by a preschool and teenage booster) induces durable immunity. Following this, in Britain, it is currently recommended that further reinforcing boosters should be given after injuries but not normally more frequently than every five years, unless the wound is particularly dirty, deep, or likely to have been contaminated. Whether routine reinforcing doses are needed for adults in other circumstances is debatable but many

cases of tetanus occur without a history of preceding injury. It would seem reasonable therefore that especially those with occupational or other increased risks of infection should receive boosters at around 10 year intervals. The precise timing of boosters required to maintain protective antitoxin levels is likely to vary with individuals and it is better to be "safe than sorry."—ERIC WALKER, lecturer in infectious diseases, Glasgow.

Joint Committee on Vaccination and Immunisation. *Immunisation against infectious disease*. London: DHSS, 1984:47-9.

Correction

Use and misuse of a digoxin assay service

We regret that an error occurred in this article by Dr Ian Gibb and others (13 September, p 678). In the abstract it was stated that "Treatment in 64 patients (22%) was changed either while awaiting the assay result or after receiving it. . . ." This should have read "Treatment in 64 patients (22%) was changed after the assay result was received."