Hepatitis B vaccination

Non-responders must be detected...

EDITOR,—Andrew J Hall's editorial on hepatitis B immunisation addressed infection with hepatitis B virus solely as a public health issue.[1] Laudable though this approach may be, it fails to take account of the needs of individual vaccinees.

We were dismayed that Hall queried whether the adequacy of the hepatitis B surface antibody response should be shown after immunisation. Hadler *et al* showed that hepatitis B virus infection occurred in 55 vaccinees with a poor antibody response after immunisation. Two became carriers of hepatitis B, both of whom had been non-responders.[2] Though hepatitis B vaccine elicits a protective immune response in most healthy people, a small proportion either fails to respond or responds only poorly to the primary course of immunisation. These people may well respond to booster doses.[3]

Determining the surface antibody level after immunisation means not only that poor responders and non-responders may be offered a booster but also that, if a health care worker is accidentally exposed on a single occasion to material infected with hepatitis B virus, post-exposure prophylaxis may be tailored to his or her needs. In our opinion it is important that health care workers who are poor responders or non-responders and are exposed to such material should be offered post-exposure prophylaxis with hepatitis B immunoglobulin.

Surface antibody levels decline significantly within five years of immunisation with hepatitis B vaccine.[3] The editorial states, "currently no reason exists for recommending booster vaccinations as a public health measure." This may be correct in the narrow sense but is of little comfort to clinical virologists. Current concerns about nosocomial transmission of infectious agents and a move to greater accountability put the prevention of hepatitis B virus infection in health care workers clearly in the public interest. While we concede that people with a surface antibody level of around 10 IU/l may in theory be protected against hepatitis B virus infection, this level is not protective from a laboratory point of view as many serum samples may give non-specific reactions of this magnitude.

Maybe it is a sign of the times for an editorial on an issue of major importance with regard to public health and resource management to conclude, "Whether antibody responses after vaccination should be verified and subsequent decay documented will depend on local resources." The editorial was depressing to those of us who invest considerable time in educating health care workers on the need for hepatitis B immunisation, testing after immunisation, and rapid reporting of accidental exposure to potentially infected material.

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1 Hall AJ. Hepatitis B vaccination: protection for how long and against what? *BMJ* 1993;307:276-7. (31 July.)
2 Hadler SC, Francis DP, Maynard JE, Thompson SE, Judson FN, Echenberg DF, *et al.* Long term immunogenicity and efficacy of hepatitis B vaccine in homosexual men. *N Eng J Med* 1986;315:209-14.
3 Jilg W, Schmidt M, Deinhardt F, Zachoval R. Hepatitis B vaccination: how long does protection last? *Lancet* 1984;ii:458.

Advice to authors

Priority will be given to letters that are less than 400 words long and are typed with double spacing. All authors should sign the letter. Please enclose a stamped addressed envelope for acknowledgment.

...and given booster injections

EDITOR,—Andrew J Hall's editorial on hepatitis B vaccination advises that "currently no reason exists for recommending booster vaccinations as a public health measure."[1] There is no specific reference to protecting health care workers from occupational risks of hepatitis B. With the present state of our knowledge it would be unwise to suggest that antibody levels of 10 IU/l give adequate protection for all occupational exposures. Health care workers at risk who have antibody levels below 100 IU/l should receive booster injections. If levels remain low, especially below 50 IU/l, adequate protection against occupational exposure cannot be assumed.

The editorial does not address the implications to health care workers undertaking invasive procedures of the Department of Health's document *Hepatitis B Infected Health Care Workers: Occupational Guidance for Health Care Workers, Their Physicians and Employers.*[2] This recommends that those who carry out invasive procedures should be required to show that they have antibodies to hepatitis B virus. I do not believe that a level of 10 IU/l can be accepted as adequate in this context until experts have provided more reassurance that such a level is as protective as a level of 100 IU/l. Meanwhile I suggest that, as with all other inactivated vaccines, booster injections should continue to be given at appropriate intervals.

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1 Hall AJ. Hepatitis B vaccination: protection for how long and against what? *BMJ* 1993;307:276-7. (31 July.)
2 Tonks A. NHS adopts tougher measures against hepatitis B. *BMJ* 1993;307:522. (28 August.)

Author's reply

EDITOR,—J A Lunn and R S Tedder and colleagues raise the issue of health care workers and occupational risk of hepatitis B. In this situation testing after vaccination to determine the peak antibody response is important. This is because it influences the action to be taken when a health care worker suffers a needlestick injury. In contrast to this, testing after vaccination is not done in the more than 40 countries that have implemented universal vaccination programmes, whether in infancy or in adolescence. Although Lunn considers that booster vaccination doses are indicated for health care workers, this is not the opinion of the United States Immunization Practices Advisory Committee on the basis of the same evidence quoted in the editorial. This committee recommends booster doses for people who have abnormal immunity, most notably patients receiving haemodialysis.[1]

A second issue raised in both letters is the protective level of surface antibody. It is important to specify what the protection is against. In studies in west Africa the peak antibody level correlated with protection against infection.[2] There was a gradient of risk of infection, with 6% of children with a peak response of >1000 IU/l infected as reflected by core antibody conversion. Although information was not available on the antibody level at the time of infection in these children, these data suggest that protection against infection is not an all or nothing phenomenon but is a probability function. Therefore there cannot be an absolute protective level of antibody against infection. In contrast, all studies have shown that protection against carriage is absolute in those who mount any antibody resonse.

Tedder and colleagues express concern that resources should play a part in determining vaccination policies. My comments about resources were not intended to refer solely to Britain. The groups at highest risk of hepatitis B infection and long term carriage with subsequent death are children in Asia and Africa. Although hepatitis B vaccination is highly cost effective (comparable to the other routine vaccinations of childhood[3]), few children in the truly high risk populations of the world have access to it. In these situations purchase and delivery of vaccine clearly take priority over serological testing.

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1 Hepatitis B virus: a comprehensive strategy for eliminating transmission in the United States through universal childhood vaccination. Recommendations of the Immunization Practices Advisory Committee (ACIP). *MMWR* 1991;40:1-25.
2 Whittle HC, Inskip H, Hall AJ, Mendy M, Downes R, Hoare S. Vaccination against hepatitis B and protection against chronic viral carriage in The Gambia. *Lancet* 1991;337:747-50.
3 Hall AJ, Robertson RL, Crivelli PE, Lowe Y, Inskip H, Snow SK, *et al.* Cost-effectiveness of hepatitis B vaccine in The Gambia. *Trans R Soc Trop Med Hyg* 1993;87:333-6.

False reassurance of pulse oximetry

Take note of inspired oxygen concentration

EDITOR,—J A H Davidson and H E Hosie rightly point out the perils of overreliance on pulse oximetry as an indicator of adequate ventilation.[1] In the case they report, however, the pulse oximeter did tell them that something was wrong. According to the alveolar gas equation, the patient must have been breathing an oxygen concentration of at least 50% through the Hudson mask (37·4 kPa carbon dioxide pressure+14·2 kPa oxygen pressure, assuming a normal respiratory quotient). If the patient had normal ventilation and normal lungs this should have given an arterial oxygen pressure of at least 40 kPa and a haemoglobin oxygen saturation of 99-100%. The measured oxygen pressure in their patient (14·2 kPa) should have produced a haemoglobin oxygen saturation of 98-99%. The saturation recorded was only 95%, which is not normal and suggests an oxygen pressure of 10 kPa or less in a patient with a normal haemoglobin concentration.

A relatively low saturation despite the patient breathing a high oxygen concentration suggests that either the patient has a high degree of ventilation-perfusion mismatch or, as in this case, the patient is not hypoxic but so severely acidotic