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.)
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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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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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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

or hypercapnic that the haemoglobin-oxygen dissociation curve is shifted well to the left. In either case a saturation of 95% should start alarm bells ringing.

The lesson to be learnt is that for patients with normal lungs breathing high inspired concentrations of oxygen the doctor should not be comforted by an oxygen saturation of less than 98-99%. A lower saturation without obvious cause should alert the doctor to measure the arterial blood pressures. Pulse oximetry remains an invaluable aid if its results are interpreted correctly.

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Misunderstanding leads to dangerous practice

EDITOR,—A recent editorial and lesson of the week have highlighted the potential limitations of pulse oximetry and the dangers of inappropriately relying on it to identify ventilatory failure.[1,2] We endorse the cautions recommended with regard to its use during anaesthesia and recovery. We have recently become concerned about a potentially dangerous lack of understanding when pulse oximetry is used to titrate oxygen treatment when patients are transferred by ambulance.

In the past six months five patients with infective exacerbations of chronic obstructive pulmonary disease have been admitted with hyperoxia and dangerous hypercapnia. On each occasion the patient had high concentrations of oxygen administered in the ambulance while arterial oxygen saturation was maintained above 90%. On arrival each patient was hyperoxic (range 18·0-40·0 kPa) and severely hypercapnic (range 10·0-22·0 kPa). Three patients were stabilised with a reduction in fractional inspired oxygen, one required doxapram, and another needed intermittent positive pressure ventilation.

We believe that these patients' condition was made worse by the inappropriate use of pulse oximetry due to lack of knowledge. Although there are many circumstances in which monitoring of arterial oxygen saturation may save lives, it must be used with caution in patients with chronic obstructive lung disease. The principles of controlled oxygen treatment in ventilatory failure are well known to every medical student and should not now be forgotten.

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1 Davidson JAH, Hosie HE. Limitations of pulse oximetry: respiratory insufficiency—a failure of detection. *BMJ* 1993; 307:372-3. (7 August.)
2 Hutton P, Clutton-Brock T. The benefits and pitfalls of pulse oximetry. *BMJ* 1993;307:457-8. (21 August.)

Use your nerve stimulator

EDITOR,—J A H Davidson and H E Hosie highlight the dangers of relying on arterial oxygen saturation to indicate adequacy of ventilation.[1] We wish to comment on several aspects of their management.

Muscle relaxation was achieved with alcuronium, but the authors do not state whether a peripheral nerve stimulator was used to monitor the degree of neuromuscular blockade intraoperatively. It is common practice to monitor neuromuscular blockade both during surgery, to ensure good relaxation leading to a near bloodless field, and during reversal of the neuromuscular blockade.

The authors administered neostigmine and glycopyrrolate and stated that incomplete reversal was apparent; presumably this was on clinical grounds. A further dose was given and reversal of neuromuscular blockade was judged adequate, as was respiratory function, and the patient was extubated. No comment was made regarding the variables used to assess respiratory effort.

It is notoriously difficult to assess the adequacy of reversal of neuromuscular blockade clinically. A patient with clinical signs indicating full reversal—for example, normal muscle power and an effective cough—may still have 70% of the acetylcholine receptor sites occupied by muscle relaxant.[2] A peripheral nerve stimulator used at this point would have indicated incomplete reversal despite clinically adequate respiratory effort.

After the patient's arrival in the recovery ward her respiratory function seems to have deteriorated steadily until help was summoned, when she was in a near moribund state. Arterial blood gas pressures indicated an extreme respiratory acidosis, which was unlikely to be corrected by a respiratory stimulant such as doxapram. The only appropriate action in this case was immediate intubation and ventilation.

It is well known that oxygenation may be maintained by diffusion despite apnoea—a fact often used during anaesthesia for rigid bronchoscopy. The authors recognise that pulse oximetry will not detect respiratory failure, but their statement that it will detect hypoxaemia is not true in all situations. A pulse oximeter measures functional haemoglobin oxygen saturation, which may be appreciably different from the percentage total haemoglobin oxygen saturation if other haemoglobin types are present. For example, a pulse oximeter will give a misleadingly high haemoglobin oxygen saturation in patients with appreciable amounts of carboxyhaemoglobin, who may be hypoxic, and results may also be misleading in patients with haemoglobinopathies. Thus not only does a pulse oximeter fail to measure adequacy of ventilation but it does not always reflect the true state of oxygenation.

The authors have presented an interesting case that highlights one of the pitfalls of overreliance on pulse oximetry in assessing respiratory function. The patient's deterioration might have been avoided if neuromuscular blockade had been monitored earlier.

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Pulse oximetry a poor guide to limb perfusion

EDITOR,—Although principally concerned with the failure of pulse oximetry to detect hyperventilation in patients receiving supplementary oxygen, Peter Hutton and Tom Clutton-Brock's editorial also alludes to the poor performance of pulse oximetry when it is used to assess peripheral perfusion.[1] The sensitivity of pulse oximeters allows pulse signals to be detected when pulse pressure is too low to provide adequate tissue perfusion and in the presence of proximal arterial occlusion.[2] This point deserves further emphasis, particularly as pulse oximetry has been advocated for use in assessing limb perfusion after trauma.[3]

Severinghaus and Spellman showed persistence of the pulse oximeter signal with normal saturations during experimental complete clamp occlusion of the brachial artery. They also observed the absence of digital blood flow (determined by plethysmography) while the pulse oximeter continued to function, although at this point saturations often fell slowly.[2]

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Ventilate immediately in severe respiratory acidosis

EDITOR,—J A H Davidson and H E Hosie provide a useful account of postoperative respiratory depression occurring in a patient with apparently normal arterial oxygen saturation.[1] Failure of theatre recovery staff to comprehend the limitations of pulse oximetry contributed to a delay in diagnosis and appropriate therapeutic intervention.[2,3]

I was most concerned, however, by the description of the subsequent management of the patient as effective resuscitation was delayed while naloxone and doxapram were administered. Furthermore, after the patient was intubated tidal volumes were measured, resulting in a further delay before intermittent positive pressure ventilation was started.

As a potentially life threatening respiratory acidosis had been proved and the patient was unresponsive it would have been more appropriate to protect the airway by intubation and start assisted ventilation immediately, in accordance with recently published guidelines for advanced life support.[4] Though measuring tidal volume and assessing the response to naloxone are important in this clinical situation, resuscitation should have been started without delay.

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Transcutaneous carbon dioxide monitoring useful in children

EDITOR,—As paediatricians dealing with respiratory failure and disorders of respiratory control, we were not surprised by the lesson in J A H Davidson and H E Hosie's paper.[1] We were surprised, however, that the authors did not mention the use of transcutaneous monitoring of partial pressure of carbon dioxide. This is used routinely alongside pulse oximetry in our unit for infants and children with moderate to severe respiratory failure. In addition, it is used for all patients in our paediatric intensive care unit and those receiving high dependency care, so that arterial blood gas sampling is required less frequently.

We use Hewlett Packard or Kontron monitors with the sensor heated to 42°C, allowing the sensor to be resited every 12-18 hours. An adjustment to