Dear Doctor

The reorganisation within the Department of Health resulted in the development of the Health Protection Programme. Future issues of the newsletter will be sent from the Programme. This issue contains details on the expanded hepatitis B immunisation programme and a variety of items which I hope you will find useful and informative.

1 DEPARTMENT OF HEALTH EXPANDED HEPATITIS B IMMUNISATION PROGRAMME

I should like to acquaint you with the details of the new, expanded programme which will be introduced early in 1987, and also to ask for your assistance and co-operation in the successful implementation of it.

(1) Current Policy

At present, and since September 1985, immunisation against hepatitis B has been available on a nationwide scale from the Department of Health only for newborn babies of mothers found to be highly infectious, i.e. those mothers with both the hepatitis B surface antigen (HBSAg) and the hepatitis B e antigen (HBeAg).

(2) Future Policy

The vaccine will be made available free in early 1987 for the following:
all newborn babies, nationwide, of mothers found to be infected carriers of Hepatitis B at ante-natal screening, ie HBsAg Positive.

(Obviously, the newborn babies of highly infectious mothers with HBeAg will also be included in this category.)

(b) All newborn babies born in:
Northland Area Health Board
and the following health districts:

(i) Takapuna   (iv) Rotorua
(ii) Auckland  (v) Napier
(iii) South Auckland (vi) Gisborne

NOTE: The cost of hepatitis B immunisation for any individuals other than those above will not be borne by the Department of Health, but is the responsibility of the individual/parent/guardian.

(3) Dosage Details

I should also like to recommend to practitioners the particular schedule to be employed for infants eligible within the National Expanded Hepatitis B Immunisation Programme:

(a) All Newborn Babies of Infectious Mothers (HBsAg+)
Within 12 hours of birth give:

(i) 100 international units (usually, though not always, in 0.5ml) of hyperimmune hepatitis B immunoglobulin intramuscularly into the antero-lateral thigh.

(ii) 10 micrograms of H-B-VAX intramuscularly into the other antero-lateral thigh.

NOTE: Hepatitis B vaccine should not be injected into the baby's buttock. Studies have demonstrated poor antibody response when the vaccine is injected into fatty tissue and it is not always possible to be certain of injecting into muscle tissue and not fatty tissue in a young baby's buttock.

At six weeks of age give:

10 micrograms of H-B-VAX intramuscularly into antero-lateral thigh.

At five months of age give:

10 micrograms of H-B-VAX intramuscularly into antero-lateral thigh.
(b) Babies Born in Northland Area Health Board, Takapuna, Auckland, South Auckland, Rotorua, Napier and Gisborne Health Districts to Non-Carrier Mothers.

Same as preceding schedule, except that Hepatitis B immunoglobulin should not be given.

Please note:  (1) The vaccine is available in different formulations.

(2) The vaccine to be used in this programme, Merck Sharp and Dohme H-B-VAX, is supplied in rubber- stoppered ampoules containing a volume of 0.7ml of fluid. 0.5ml of this contains 20 micrograms of vaccine (one adult dose); the dose for infants is 0.25ml. For accuracy, 0.5ml Diabetic Syringe, or alternatively a 1.0ml Tuberculin Syringe can be used. Provided the usual aseptic procedure is followed, a second paediatric dose should be taken from the ampoule at any time up to the expiry date shown on the package. To maintain potency of the vaccine, ampoules must be kept at a temperature of 20-8°C and not frozen or allowed to warm up.

(4) Ante-Natal Screening

It is recommended that all pregnant women to be screened for HB surface antigen, not only for medical and nursing staff to take suitable precautions, but also to ensure that the baby receives an immunoglobulin injection and the first dose of HB vaccine within twelve hours of birth.

(5) Informed Consent

Attending medical practitioners should obtain informed consent from the mother before arranging administration of the vaccine to the newborn baby.

(6) Supplies of Vaccine

Practitioners should be aware of when eligible babies within their care become due for their 2nd and 3rd injections of HB vaccine. Arrangements should be made through district health offices or area health boards to obtain supplies of the vaccine.
Practitioners wishing to offer immunisation to patients who are not covered by the Department of Health Programme must obtain vaccine directly from an appropriate pharmaceutical outlet.

(7) Low Dose Vaccine

Lower doses of HB vaccine are being used in some areas and this option was considered. However, the Department of Health considered that there is insufficient evidence at present to proceed with a national immunisation programme using lower than manufacturers recommended doses of vaccine.

2 INFECTIOUS DISEASES ORDER 1986 : RHEUMATIC FEVER NOTIFIABLE

Rheumatic fever has been added to the list of diseases under Section B of Part I of the first schedule of the Health Act. It became notifiable to the medical officer of health as from 4 September 1986.

There is recent evidence that there are deficiencies in diagnosis and in primary and secondary management of rheumatic fever in New Zealand. Progress in reducing the impact of the disease depends on effective co-ordination and delivery of services by means of rheumatic fever registers. This in turn implies notification of cases.

For practical purposes the following conditions should be notified:

(1) acute rheumatic fever and/or
(2) sydenhams chorea and/or
(3) presumed rheumatic heart disease under the age of 20 and/or
(4) patients on secondary antibiotic prophylaxis for rheumatic fever.

Therefore all new and suspected cases of rheumatic fever including chorea, and all patients on secondary prophylaxis will be notifiable ensuring the patient is added to a rheumatic fever register through which the secondary prophylactic penicillin injection programmes will be administered in future.

The register will be administered through the medical officer of health and further details in relation to the prophylaxis programme will be issued in the next circular letter to medical practitioners.

3 TREATMENT OF ANTICOAGULANT POISONING

Item 6 in the Circular Letter to Medical Practitioners PH 1/86 issued in April 1986 discussed ICI (NZ) Ltd’s booklet which deals with the treatment of anticoagulant poisoning. After an approach from Dr Paul Ockelford, Department of Immunobiology,
University of Auckland Medical School, who pointed out a potentially confusing statement relating to the relative toxicities of the anti-coagulants difenacoum and brodifacoum when compared with the more familiar warfarin, the Department of Health contacted ICI. This resulted in a change to the booklet.

The alteration reads "The major difference between warfarin and difenacoum/brodifacoum is that both difenacoum and brodifacoum can cause increased bleeding tendency for a longer period of time than warfarin" (rather than "increased bleeding time"). "It is therefore important to understand that it may be necessary to give vitamin K1 for weeks rather than days".

Extra copies of the revised booklet are available on request from Mr W F Leonard of ICI (NZ) Ltd, P O Box 1592, Wellington.

4 MENINGOCOCCAL MENINGITIS

This epidemic appears to be caused predominantly by Group A organism and continues to be spreading throughout Auckland and particularly in south, northwest and central districts of Auckland. Approximately three quarters of the cases occurring in children under 14 years. It is an infection occurring predominantly in winter and spring and particularly where there are crowded living conditions.

Early treatment is essential as delays of only a few hours can be dangerous and even fatal.

The incubation period for this disease is short. Adequate protection of contacts cannot therefore be provided in time by immunisation. If, however, general practitioners wish to provide some protection for individual patients, especially in the Auckland area it should be noted that although the vaccine does not currently have consent to market in New Zealand, it can be obtained on a named patient basis from Smith, Kline and French.

All general practitioners should consider rifampicin prophylaxis for contacts of cases in consultation with the medical officer of health (see C.L.M.P. 2/85).

Yours sincerely

J C J Stoke
Manager
Health Protection Programme
RECOMMENDATIONS FOR PREVENTION OF HEPATITIS B VIRUS INFECTION
IN NEW ZEALAND BY THE USE OF VACCINE.

Prepared by Hepatitis Subcommittee for Communicable Disease
Control Advisory Committee, New Zealand.

INTRODUCTION

The term 'viral hepatitis' includes hepatitis A, hepatitis B, non-A non-B hepatitis and delta hepatitis. Hepatitis A, formerly infectious hepatitis, is caused by hepatitis A virus (HAV) for which there are reliable serological markers. HAV is spread almost exclusively by the faecal-oral route, a carrier state or chronic infection does not occur, and there are no long term sequelae. Hepatitis B, formerly serum hepatitis, is caused by hepatitis B virus (HBV) for which there are reliable serological markers also. HBV is spread primarily by parenteral transfer of infected serum, intimate, especially sexual, contact and perinatally. A carrier state may develop and can be responsible for chronic liver disease. Non-A, non-B hepatitis is probably caused by at least two different agents and remains a diagnosis by exclusion as there are no specific diagnostic tests. There are reliable serological markers for delta virus which is dependent on HBV infection and may occur as a co-infection with HBV or as a superinfection of a hepatitis B carrier. Delta virus infection may compound the severity of both acute and chronic hepatitis B.

POTENTIAL TO ERADICATE HBV AND DELTA VIRUS INFECTIONS

HBV infection is potentially eradicable as infected humans are the only reservoir and almost all susceptible humans could be protected by vaccine if its administration were fiscally and logistically feasible. Protection against HBV also implies protection against delta virus.

REASONS TO PREVENT HBV AND DELTA VIRUS INFECTIONS

In surveys conducted in the U.S.A. approximately 25% of those infected with HBV become ill with jaundice and of these 0.5% die of fulminant disease. 6-10% of those infected become chronic carriers and of these some 25% develop chronic active hepatitis which is associated with a high risk of cirrhosis and hepatocellular carcinoma.

Although epidemiological data on the natural history of HBV infection in New Zealand are less complete and available data suggest that chronic carriage after acute hepatitis B is less common than in the USA, chronic hepatitis is commonly diagnosed and has been reported to be due to HBV infection in
30-40% [1]. Non-Europeans in Auckland have a nine times greater incidence than resident Europeans of hepatocellular carcinoma and in these patients HBV infection is common [2]. Its eradication and coincidentally that of delta virus are therefore desirable.

THE PREVALENCE OF HBV INFECTION IN NEW ZEALAND

The epidemiology of HBV infection in New Zealand has been investigated mainly in restricted occupational, geographical and social groups thought to be at high risk [3] and by a national survey of 2001 sera collected from children and adolescents aged 0-21 years during a three month period beginning in November 1978 [4]. This last study was undertaken as part of a regular national seroprevalence survey conducted by the Department of Health using 100-200 sera collected within each of New Zealand's eighteen health districts. There was a prevalence of hepatitis B antibody in Europeans of 10% with a 2% carriage rate and in Maoris of 25% with a 6% carriage rate. The prevalence in the north of the North Island was approximately three times that of the South Island with that in the south of the North Island being intermediate. These data contrast with the estimated life-time risk of HBV infection in the USA of only 5%. It is apparent that New Zealand has a particular problem with HBV and this has been further emphasised by a regional study in Kawerau where the prevalence of all hepatitis B markers in adolescents aged 15-19 years was 61.6% for Europeans and 74.5% for non-Europeans [7].

TRANSMISSION OF HBV

Transmission occurs via percutaneous or permucosal routes from carriers and persons with acute infection. The highest concentration of HBV is in blood, serum and plasma and lesser concentrations are in other body fluids such as saliva and semen [5].

Prevalence studies have indicated the following settings of increased risk.

1 Perinatal - infants born to HBsAg positive mothers. This is especially important in the subgroup born to mothers with HBeAg, as 90 percent of these infants become chronic carriers at one year if they are untreated [6].

2 Exposure to infected blood:
   (1) occupational eg, health care workers;
   (2) iatrogenic eg, haemodialysis patients;
   (3) sharing of needles by parenteral drug abusers.

3 Sexual contact with an infected person:
   (1) infected partner,
   (2) multiple heterosexual or homosexual partners.
Occult transfer, probably of blood:

(1) household contacts of infected persons,

(2) children in geographical areas where infection is prevalent,

(3) institutions, especially those for disturbed and retarded patients and prisons.

The prevalence of hepatitis B serological markers in various population groups in the USA is shown in the table [5] and illustrates the magnitude of risk for several of these settings. The concept that schools may be an important setting for transfer of HBV is suggested by the Kawerau study [7] in which a sharp increase in prevalence was noted in the early school age group.

WHO SHOULD BE VACCINATED AGAINST HEPATITIS B IN NEW ZEALAND?

In New Zealand priority was initially given to neonates born to hepatitis B surface antigen (HBSAg), hepatitis B e antigen (HBeAg) positive mothers. This group is known to have the greatest risk of carriage and epidemiologically acts as a continuing source of community infection. These individuals carry a high risk of eventual hepatocellular carcinoma. Subsequently vaccination was also offered to mothers who were HBSAg positive but HBeAg negative. As a corollary of that decision there is no need for all mothers to be HBeAg tested.

By mid 1985 data from the Kawerau studies had become available and shown that in that area of New Zealand and by implication at least, in other similarly structured New Zealand communities, acquisition of HBV was more common in the 5-9 year old group than at birth: these authors have subsequently shown that important transmission occurs even before school entry [Milne A, personal communication]. There would thus seem a need to vaccinate children at an earlier age than school entry, at least in those parts of the country where this is the local epidemiological pattern of spread.

There are problems in devising a hepatitis B vaccination programme for preschoolers. The three dose regimen recommended does not fit neatly into the routine New Zealand Paediatric vaccination schedule. There is also the issue of whether individuals should be screened for hepatitis markers before being vaccinated: in general the greater the prevalence of infection in the community the more likely is screening to be cost effective. In young children blood tests are often technically difficult and may adversely influence subsequent vaccination acceptance.

A programme aimed at all children soon after birth and conducted without prior screening would resolve most of these difficulties. Children would be protected from the earliest age and the scheme could be included with the present routine paediatric schedule, but a catch-up programme would be
necessary for those at risk, and especially children at risk, in the remainder of the population. Of many possible options the Communicable Disease Control Advisory Committee (CDCAC) has recommended the following measures for national eradication of hepatitis B.

Vaccination of:

1. All neonates
2. All susceptible household or sexual contacts of HBsAg positive individuals.
3. All children entering primary and intermediate schools and not already vaccinated or known to be immune. [This programme would cease after 5 years].
4. Others at risk ie, certain employees, certain patients and other institutionalised persons, parenteral drug abusers and those likely to have sexual contact with infected individuals.

These recommendations remain long term goals, but because of costs, are not immediately feasible. It is the opinion of the CDCAC that if restriction on nationwide neonatal vaccination is needed for economic reasons, then it would be appropriate to vaccinate all North Island neonates in the first instance. In the event the option initially chosen by the Department of Health as offering best use of existing resources has been vaccination of all neonates, regardless of maternal HBsAg status, in certain, very limited, geographically defined areas of high risk. This is in addition to the programme of vaccination of all neonates born to known HBsAg positive mothers.

Extension of the current programme to include susceptible household contacts of all HBsAg positive individuals would allow for vaccination of most children now at significant risk and not protected by the neonatal programme.

The need for and frequency of booster doses remains to be determined. There is a vital need that the data on vaccinations be documented for individuals in a way that allows for easy later access: at the moment this is the responsibility of every doctor who arranges administration of hepatitis B vaccine.

It is also vital that there be a continuing coordinated national surveillance programme.

S D R Lang FRACP, FRCPA
R B Ellis-Pegler PRACP
D R Lennon PRACP
REFERENCES


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