vated by 6M urea. The final step of treatment with formalin inactivates hepatitis B virus, as well as many other viruses, including parvoviruses, retroviruses, and the delta agent.[6] Human T cell lymphotropic virus (HTLV III or LAV) is a retrovirus and there is strong evidence of a causal association between this virus and AIDS; but as might be expected from the above data AIDS has not been associated with the vaccine.

The hepatitis B plasma derived vaccine, then, meets the WHO requirements revised in 1983 and its safety is now established. The priorities for immunisation against hepatitis B are not the same for each region or country. These needs are dictated by epidemiological patterns, socioeconomic factors, cultural and sexual practices, and the environment. Immunisation against hepatitis B is recommended for six main groups in Britain.

Firstly, health care personnel should be vaccinated if they have frequent contact with blood or needles, if they are staff of residential institutions for the mentally handicapped, take part in direct patient care in units treating carriers, or work in haemodialysis, haemophilia, and other centres providing maintenance treatment with blood or blood products. Dentists and ancillary staff with direct contact with patients, laboratory workers regularly exposed to increased risk from infected material, and personnel on secondment to countries with a high prevalence of hepatitis B, if they are directly concerned in patient care, should also be vaccinated. Personnel accidentally pricked with needles used for patients with hepatitis B should be given the vaccine either alone or in combination with hepatitis B immunoglobulin at a contralateral site.

Secondly, the patients who should be vaccinated include first entrants into residential institutions for the mentally handicapped, those treated by maintenance haemodialysis or by frequent transfusion of blood or blood products, those having surgery requiring multiple transfusions or treatment with blood products, or both, and those with chronic renal damage when it appears likely that treatment by haemodialysis or transplantation will ultimately be required.

Thirdly, vaccination should be offered to certain contacts of patients: the sexual partners of patients with acute hepatitis B or carriers and other family members in close contact.

Fourthly, vaccination is recommended for infants born to hepatitis B carriers or HBsAg positive mothers as a result of recent infections, particularly if e positive or without anti-e. The optimum time for immunisation in combination with hepatitis B immunoglobulin, however, is not yet established.

Fifthly, other groups at risk include staff at reception centres for refugees and immigrants from areas where hepatitis B is very common, such as South East Asia; individuals who frequently change sexual partners—particularly promiscuous male homosexuals and female and male prostitutes; and narcotic drug abusers.

Finally, there are groups at "lower risk," including long term men prisoners; staff of custodial institutions, ambulance, and rescue services; and selected police personnel.

Failure to immunise these people places them at unnecessary and unjustified risk.[7]

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Ethics in clinical chemistry

At first sight there might not appear to be much scope for ethical brooding among the analytical machines and gadgets of a clinical chemistry laboratory. Yet an extensive discussion of ethical issues has in fact been filling sizable portions of the *Association of Clinical Biochemists' News Sheet* for the past several months.

The inspiration came from a plenary lecture on the subject given in Vienna by BenGershôm at the International Federation of Clinical Chemistry's congress in 1981. Dr BenGershôm is head of the clinical chemistry department at the Sophia Children's Hospital, Rotterdam. His lecture delineated various topics in which, at least by his observation, barriers of professional etiquette deter the clinical biochemist, who may have special skills, from offering advice to clinicians on the investigation, treatment, or counselling of patients. For example, the biochemistry of some rare inherited disorders may be better known to the clinical chemist than to an individual clinician encountering such a case for the first time. In BenGershôm's view ethical considerations should, but rarely do, take precedence over etiquette and should oblige the clinical chemist to participate fully in the clinical decision making process.

With some trepidation the Association of Clinical Biochemists took up the challenge through one of its working parties—the one most concerned with the interaction between clinicians and the laboratory. Instead of attempting to draw up an ethical code of practice on behalf of the association (which everyone realised would be unlikely ever to secure general agreement) the working party drew up a list of questions on ethical matters which were then put to four leaders in clinical chemistry, who were encouraged to answer them purely according to personal conviction. The recorded interviews have now been published, perhaps unfortunately in somewhat ephemeral form (*Association of Clinical Biochemists' News Sheet*, issues of April to July 1984).

The replies showed a striking degree of concordance. British clinical chemists, whether medically qualified or not, are evidently ready and willing to shoulder the responsibility for facing ethical problems together with the relevant clinician, while unhesitatingly recognising that in case of disagreement the final decision lies with the consultant in charge of the case. All four clinical chemists interviewed regarded coaxing clinicians into using the laboratory more sparingly as an ethical duty. There was less agreement on how strong a stand a chemist should take if he finds himself engaged in a supportive role in clinical research whose ethics seem—usually at some distance and on somewhat flimsy evidence—to be dubious. Attitudes on whether industrial action (especially in support of other groups of staff) is ever justifiable also varied, as in other medical and paramedical professions.

Most of those interviewed were sympathetic to the notion of redeploying to other duties any member of staff who

objected to work connected with programmes they found morally objectionable—for example, screening programmes which necessarily lead to the termination of some pregnancies. The ethical dilemma posed by the need on the one hand to establish reference ranges for chemical measurements in the blood of healthy children but to avoid invasive venesection on the other was tackled with scrupulous sensitivity coupled with a forthright determination to improve the welfare of future patients.

Some of the issues raised were surprising. Who would have thought that deciding whether to telephone laboratory results might pose ethical problems? It turns out to do so because vital results may need to be conveyed quickly, but there is a risk that the message may never reach its proper destination or get so garbled along the way that the wrong treatment is started. Yet another item whose *ethical* importance has not been emphasised is the vexed question of whether clinical chemists should try to prevent the use of a blood gas analyser by an anaesthetist or a glucose meter by a diabetologist if they consider that these instruments are or may be the source of misleading results and consequent harm to patients.

Many of the matters discussed border on what most people would regard as questions less of ethics than of professional responsibility—for example, the amount of effort that should be given to quality control within the laboratory or how best to deal with staff members hooked on NHS ethanol; but then the Association of Clinical Biochemists' working party quite rightly reminds us that in every profession professional integrity is ultimately an ethical matter.

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Diarrhoea, dehydration, and drugs

Oral rehydration therapy is effective in treating over four fifths of episodes of diarrhoeal dehydration,[1] and in developing countries which have adopted this form of treatment hospital admission rates and mortality have decreased by about half.[2] Nevertheless, the logistics of getting oral rehydration fluids to children throughout the world to treat the 500 million attacks of diarrhoea that they experience each year pose vast problems. At present probably fewer than 10% of these children have access to oral rehydration fluids, and of these fewer than half actually receive them. Furthermore, the beneficial effects of oral rehydration on diarrhoeal mortality will not be matched by a decreased attack rate unless other public health measures are introduced and breast feeding is encouraged.

Current recommendations for the treatment of diarrhoeal dehydration include intravenous plasma or saline for circulatory failure followed by oral fluids to complete rehydration. In the absence of shock or a contraindication to giving fluids by mouth, such as ileus, oral rehydration may be started.[1] A well proved oral rehydration regimen entails giving a solution of sodium 90 mmol(mEq)/l and glucose 111 mmol/l (20 g/l), with potassium 20-30 mmol(mEq)/l and base 20-30 mmol(mEq)/l for the first four hours, followed by water for a further two hours.[3] The total volume of fluid administered should be twice the estimated fluid deficit. If hyponatraemia is present oral rehydration solution is given without extra water,[4] and in hypernatraemia the rehydration period is extended to 12 hours.

The addition of glycine[5] or substitution of rice powder for glucose[6] is a useful adjunct to the standard regimen because it may reduce the stool volume and the duration of diarrhoea. The beneficial effect of adding nutrients such as powdered rice may explain the observations of Isolauri and Vesikari, who found that if children resumed their normal diet immediately after oral rehydration the duration of diarrhoea was a third as long as that in children whose usual diet was introduced gradually.[7] Other studies of early feeding have shown similar benefits and such an approach should diminish the nutritional deprivation imposed by recurrent attacks of diarrhoea.[1 3-5 8]

Not all diarrhoeal dehydration responds to oral rehydration, and vomiting of rehydration fluid may be a cause of failure. This may be alleviated by giving the fluid in frequent small amounts—for example, by cup and spoon or nasogastric infusion. Carbohydrate malabsorption is another cause, and should be suspected if the patient continues to pass voluminous watery stools. In rotavirus diarrhoea up to a third of the glucose in the oral rehydration fluid may appear in the stools,[9] but an adequate clinical response may still be achieved, the success rates in rotavirus diarrhoea being as high as those in bacterial diarrhoea. Rehydration solutions such as that recommended by the World Health Organisation are less likely to cause malabsorption of carbohydrate because they contain half the concentration of carbohydrate of the commercial rehydration fluids. Treatment by mouth may also fail if stool loss exceeds 10 ml/kg/hour. Treatment of the child with dysenteric diarrhoea may be particularly difficult, because even if rehydration is achieved the child may die from the effects of systemic spread of invasive pathogens such as *Shigella*, *Salmonella*, and *Campylobacter*.

What other forms of treatment may be deployed to tackle the problems? Antibiotics have a limited part to play in treating specific infections such as cholera, severe shigellosis, giardiasis, amoebiasis, and typhoid and when there is diagnostic doubt whether the infant has gastroenteritis or septicaemia.[10] Hill *et al* used oral gentamicin and cholestyramine to treat children in South Africa with protracted diarrhoea that was presumed to be infective.[11] Nevertheless, bacterial resistance to antibiotics is increasing, and clinical trials of oral rehydration therapy have shown that antibiotics are usually unnecessary.[1 3-9]

Of the antidiarrhoeal drugs, loperamide appears to be the most effective when used to treat chronic diarrhoea of varied aetiology,[12] excess ileostomy output,[13] and faecal incontinence.[14] Sandhu *et al* have used high doses successfully in selected infants with severe protracted diarrhoea.[15] The effect of loperamide on stool output seems to be exerted by its opiate like action on the motility of the bowel and enhanced absorption of chloride.[16] Though apparently less toxic than its predecessor Lomotil (diphenoxylate and atropine), loperamide may still cause opiate poisoning and ileus.[15 17]

The results of a multicentre trial of loperamide as an adjunct to oral rehydration in the treatment of acute diarrhoea are reported in this issue (p 1263). The duration of the diarrhoea was reduced significantly, but the effect of loperamide on stool output was not recorded. Other antidiarrhoeal drugs that have been similarly investigated