[5] Jacobson S, Bagley C, Rehin A. Clinical and social variables which differentiate suicide, open and accidental verdicts. *Psychol Med* 1976; **6**:417-21.
[6] Barraclough BM. Are the Scottish and English suicide rates really different? *Br J Psychiatry* 1972;**120**:267-73.
[7] Bagley C. Correspondence on the validity and meaning of suicide statistics. *Sociology* 1974;**8**:313-6.
[8] Barraclough BM, White SJ. Monthly variation of suicide and undetermined deaths compared. *Br J Psychiatry* 1978;**132**:275-8.
[9] Barraclough BM, White SJ. Monthly variation of suicidal, accidental and undetermined poisoning deaths. *Br J Psychiatry* 1978;**132**:279-82.
[10] Holding TA, Barraclough BM. Undetermined deaths—suicide or accident? *Br J Psychiatry* 1978;**133**:542-9.
[11] Sainsbury P, Barraclough B. Differences between suicide rates. *Nature* 1968;**220**:1252.
[12] Surtees SJ. Suicide prevention by the Samaritans. *Lancet* 1977;ii:872.
[13] Barraclough BM. Differences between national suicide rates. *Br J Psychiatry* 1973;**122**:95-6.
[14] McCarthy PD, Walsh D. Suicide in Dublin. *Br J Psychiatry* 1975;**126**: 301-8.
[15] Atkinson MW, Kessel N, Dalgaard JB. The comparability of suicide rates. *Br J Psychiatry* 1975;**127**:247-56.
[16] World Health Organisation. *Suicide and attempted suicide.* Public Health Paper No 58. Geneva: WHO, 1974.
[17] Anonymous. The suicide profile. *Br Med J* 1975;ii:525-6.
[18] Capstick A. Recognition of emotional disturbance and the prevention of suicide. *Br Med J* 1960;i:1179-82.
[19] *Re Davis (dec'd) (1968)* 1 QB:82D.
[20] *R v Cardiff City Coroner ex parte Thomas* (1970) 1 WLR 1475.
[21] Surtees SJ. Tales of Beachy Head. *Eastbourne Medical Gazette* 1978;**2**: 71-8.
[22] Sainsbury P. *Suicide in London.* Maudsley Monograph No 1. London: Chapman and Hall, 1955:65.
[23] Barraclough BM, Shepherd D, Jennings C. Do newspaper reports of coroners' inquests incite people to commit suicide? *Br J Psychiatry* 1977;**131**:528-32.
[24] Read CS. The problem of suicide. *Br Med J* 1936;i:631-4.
[25] Simpson MA. The great suicide epidemic of 1933. *World Medicine* 1978;**13**:77-9.
[26] Ashton JR, Donnan SPB. Suicide by burning—a current epidemic. *Br Med J* 1979;ii:769-70.
[27] Surtees SJ, Taylor DC, Cooper RW. Suicide and accidental death at Beachy Head. *Eastbourne Medical Gazette* 1976;**2**:22-4.
[28] Capstick A. The methods of suicide. *Medico-Legal Journal* 1961;**29**:33-8.
[29] Wells N. *Suicide and deliberate self-harm.* London: Office of Health Economics, 1981:6.
[30] Swinscow D. Some suicide statistics. *Br Med J* 1951;i:1417-23.
[31] Surtees SJ. Tales of Beachy Head. *Eastbourne Medical Gazette* 1980;**2**: 135-41.

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Clinical Topics

Hepatitis B virus infection in medical and health care personnel

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Abstract

An analysis of 51 cases of hepatitis B virus infection in health care workers admitted as patients to the liver unit over seven years showed three healthy carriers of hepatitis B virus, seven cases of fulminant hepatic failure, 13 cases of acute hepatitis, six cases of chronic persistent hepatitis, 17 cases of chronic active hepatitis (of whom 11 had cirrhosis), and five cases of hepatocellular carcinoma. To date 11 of these patients have died. Only 15 of the 51 patients had a history of direct occupational exposure and only three patients could recall specific inoculation injuries. In contrast, the source of infection was apparent in 32 of 50 consecutive cases of fulminant hepatic failure or acute hepatitis B in non-medical staff.

Since specific inoculation injuries are not the usual mode of infection in medical staff and since only a few of the patients who are hepatitis B virus carriers will be detected by selective screening of "high-risk" patients, the overall risk of infection can be reduced only by stricter precautions in the handling of *any* patient's blood and by the use of hepatitis B virus vaccines for medical staff at high risk.

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Introduction

Even before the identification of antigens of the hepatitis B virus, homologous serum jaundice was thought to be an occupational hazard in medical staff,[1] and the recent studies using antibody to hepatitis B surface antigen (anti-HBs) as a marker of previous exposure to hepatitis B virus show without doubt that medical staff are more often exposed than the general population. The highest prevalence of anti-HBs is among surgeons and pathologists, although nurses, laboratory staff, workers in renal units, blood procurers, and dentists are also affected.[2-4] Prevalence increases with age, but among medical staff the most rapid rise is in the early years after entering clinical work.[2] Outbreaks of acute hepatitis B in renal and oncology units[5 6] and in relation to emergency surgery[7] provide further evidence that medical staff can acquire hepatitis B virus infection from patients.

The availability of specific immune globulin for use after incidents of accidental inoculation and the very recent introduction of hepatitis B virus vaccines[8] have opened new approaches for prevention, and because of this we decided to review our experience of liver disease related to hepatitis B virus among health care workers with particular reference to the time and source of infection, the findings being compared with 50 consecutive cases of acute type B hepatitis in non-medical staff.

Series of patients

The series of 51 patients comprised 39 men and 12 women referred to the liver unit in the past seven years in whom hepatitis B virus infection was proved. Serum was examined for HBsAg and anti-HBs

and the e antigen system (HBeAg and anti-HBe) by radioimmunoassay (Abbott Laboratories). Most patients were surgeons, physicians, or nurses; 24 were Caucasians born and working in the United Kingdom, while 27 were from overseas, mainly South Europe, the Arab countries, and the Far East (table I). Seven of these foreign patients had been working in Britain for several years before their presentation with liver disease. They are considered below according to specific types of disease identified (table II).

TABLE I—*Occupations of 51 health care workers with hepatitis B virus infection*

	UK born	Foreign	Total
Nurses	9	6	15
Surgeons	5	9	14
Physicians	5	5	10
Laboratory staff	2	2	4
Radiologists	1	1	2
Anaesthetists	0	2	2
Dentists	2	0	2
General practitioners	0	2	2
Total	24	27	51

TABLE II—*Diseases related to hepatitis B virus in 51 health care workers*

	UK born	Foreign	Total
Healthy carriers	2	1	3
Fulminant hepatic failure and acute hepatitis	14	6	20
Chronic persistent hepatitis	3	3	6
Chronic active hepatitis	4	13	17
Hepatocellular carcinoma	1	4	5
Total	24	27	51

TABLE III—*Mode of exposure to hepatitis B virus in patients born in the United Kingdom with fulminant hepatic failure or acute hepatitis. Comparison of health care personnel with non-health care personnel*

	Health care personnel (14 cases)	Non-health care personnel (50 cases)
Occupational	4	0
Intravenous drug abuse	0	17
Blood transfusion	0	1
Tattooing	0	1
Heterosexual	1	4
Homosexual	0	9
Total known exposure	5 (35·7%)	32 (64%)

HEALTHY CARRIERS

Three HBsAg-positive patients with normal liver function test results and histology were classified as healthy carriers. In two patients the source of infection was unknown. The third patient, a retired theatre sister, probably acquired the infection from her husband, a general surgeon, who died of chronic active hepatitis and is included in the series. One patient was HBeAg positive.

FULMINANT HEPATIC FAILURE AND ACUTE HEPATITIS

There were seven patients with fulminant hepatic failure, of whom four survived, and 13 with less severe acute hepatitis. The 17 survivors returned to work after periods of one to six months. None has developed chronic liver disease. HBsAg was eliminated from the serum, and 11 of 12 who were tested more than six months after the onset of their illness had developed anti-HBs.

Of the 14 patients in this group who were born in the United Kingdom, four had a history of recent occupational exposure to HBsAg-positive patients: a theatre sister had been exposed during an emergency operation, a physician while working in a renal unit, one nurse while working in an intensive care unit, and another nurse while working on a medical ward. Non-occupational exposure could be implicated only in the case of a female patient whose regular sexual partner had recently had acute hepatitis B (table III).

Of the six foreign patients in this group, five had a history of recent occupational exposure. One surgeon had suffered an accidental inoculation injury with a safety pin during an operation, two other surgeons had recently operated on patients with hepatitis, and two nurses had nursed HBsAg-positive patients on medical wards.

In contrast to the British medical personnel, the source of hepatitis B virus infection was apparent in a large proportion (64%) of the 50 consecutive cases of acute hepatitis B virus infection in British residents who were not medical staff. None of these patients was referred directly from a drug addiction or venereology clinic, but the commonest sources of infection were intravenous drug abuse (17) and male homosexual activities (9) (table III).

CHRONIC ACTIVE HEPATITIS AND CHRONIC PERSISTENT HEPATITIS

Of the 17 patients with HBsAg-positive chronic active hepatitis, 11 had cirrhosis and three died—two in retirement and one while still in practice at the age of 54. Of the 14 patients alive, four were able to work only part time. Only two patients cleared HBsAg from the serum and developed anti-HBs, and of the others, who remained HBsAg positive over follow-up periods of six months to five years, eight were also HBeAg positive.

The six patients with HBsAg-positive chronic persistent hepatitis were clinically well and worked full time, although a medical physics technician in the cardiopulmonary bypass team had to change his duties because of hospital policy. These six patients were HBsAg positive for two to seven years and two were also HBeAg positive.

Of the seven patients in this group born in the United Kingdom, two had suffered accidental inoculation injuries with needles used to draw blood from patients, and a psychiatric nurse had recently been nursing patients with acute hepatitis B in a hospital for mental subnormality. Non-occupational exposure seemed likely in only one—a man who admitted frequent homosexual intercourse.

Of the 16 foreign patients in this group, three surgeons developed acute hepatitis B after operating on patients who were known to be hepatitis B virus carriers or who shortly afterwards became ill with acute hepatitis. Progression to chronic active hepatitis was documented nine months to three years later.

HEPATOCELLULAR CARCINOMA

All five cases of HBsAg-positive hepatocellular carcinoma had an underlying cirrhosis and died within a short period. Three presented when the carcinoma was developing; the other two had been seen earlier with cirrhosis and were reasonably well until the appearance of the carcinoma one to three years later. In none of these five patients was there an obvious source of hepatitis B virus infection.

Discussion

The incidence of clinical virus hepatitis is four times higher among American hospital employees than in the general population,[9] and 10 times higher among British surgeons than in the general population of London.[10 11] Also, in reported series of patients with acute hepatitis, hospital staff have been represented in high frequency—from 7% in the United Kingdom[12] and United States[13] to 23% in Switzerland.[14] The exact mode of hepatitis B virus infection in these medical staff, however, was ascertained only occasionally, and in this series only one of the 20 with acute hepatitis B virus infection and two of the 28 with chronic liver disease could recall a definite inoculation injury and in only one case was it certain that HBsAg-positive blood was implicated (table IV). This accords with the experience of the Public Health Laboratory Service, which reported that only two of 52 hospital workers with acute hepatitis B could remember a specific inoculation injury.[10] In contrast to the medical staff, in our series of non-medical personnel with acute hepatitis B virus infection, where only non-occupational exposure was possible, the actual source of infection was usually apparent. Probably, therefore, occupational rather than non-occupational exposure was responsible for the infection in many of the medical staff in whom the actual source was not apparent from the history.

Contact with a particular HBsAg-positive patient was identified in 15 (29·4%) of our 51 patients, and this applied to

similar numbers of the British patients and those born abroad (table IV), which suggests that a similar proportion of the hepatitis B virus infection in medical staff in areas of high and low hepatitis B virus prevalence was due to occupational exposure.

In an epidemiological study Pattison *et al*[15] showed that contact with patients' blood was the main risk factor for hepatitis B virus infection in hospital staff and not direct patient contact, even though saliva, ascites, and other body fluids may contain hepatitis B virus. In view of our findings it seems likely that hepatitis B virus infection may be acquired by contact with infected blood without specific inoculation injury. Evidence in support of this comes from the observation that renal dialysis workers are less likely to become HBsAg positive if they wear gloves when dialysing patients.[3] Although this does not protect against inoculation injury, it does reduce simple skin contact.

The regular testing of staff and patients in renal dialysis units and the isolation of HBsAg-positive individuals has, in the United Kingdom, resulted in an overall reduction in the prevalence of hepatitis B virus in patients and incidence of hepatitis among the staff.[16] Routine HBsAg testing of other high-risk groups might be thought to reduce similarly the risk of hepatitis B virus infection in medical staff. The study of Maynard[17] shows, however, the inadequacy of *selective* HBsAg screening. Among 6216 consecutive patients admitted to hospital, 59 were found to be HBsAg positive, of whom only 10 (17%) would have merited screening on clinical grounds. Cossart[18] has also drawn attention to the false sense of security that may occur in handling blood that has not been screened for HBsAg and is therefore assumed to be negative. The adoption of special precautions for handling only those blood samples shown to be HBsAg-positive on the basis of selective screening of high-risk patients will probably have little effect on the overall incidence of hepatitis B virus infection in medical staff. Only by taking precautions in all intravenous procedures and with all blood specimens reaching the laboratories will the risk of hepatitis B virus acquisition by staff be minimised. Unfortunately, in some circumstances, such as major gastrointestinal bleeding or major trauma, it is difficult to avoid contamination of staff members if the patients are to receive effective emergency treatment.

Three of the patients in this series may have transmitted the hepatitis B virus infection to family contacts. The son of one of the dentists and the wife of a surgeon (both from the group born in the United Kingdom) were found to be HBsAg positive when the respective families were tested and have remained healthy carriers of hepatitis B virus. The son of one of the foreign doctors developed hepatitis shortly after his father. The risk of medical staff transmitting hepatitis B virus infection to their patients is, however, quite small. Although 11 patients in this series had HBeAg in the serum, indicating high infectivity, there was no evidence that they had transmitted infection to their patients. Other reports indicate that medical and dental staff in the incubation period of acute hepatitis B have treated patients without apparently transmitting hepatitis B virus infection to any of them.[19 20] There are other reports, however, where health care workers who are chronic hepatitis B virus carriers, including a nurse,[21] a physiotherapist,[22] dentists,[23 24] and a gynaecologist,[25] have been responsible for the transmission of infection.

Treatment that inhibits virus replication and renders the blood non-infective would be of particular value to medical staff, and early results with adenine-5-monophosphate are encouraging.[26] Only the prophylactic use of hepatitis B virus vaccines at the start of clinical training is likely to reduce the risk of acquiring hepatitis B virus infection. In a controlled trial among medical and nursing staff in French renal units[27] hepatitis B virus vaccination was shown to confer protection against infection, and these encouraging results indicate that immunisation with hepatitis B virus vaccines should be of value in other health care workers with a lower risk of infection.

TABLE IV—*Comparison of occupational exposure to hepatitis B virus in UK born and foreign health care staff*

	UK born (n = 24)	Foreign (n = 27)
Accidental inoculation injury	2	1
Operations on HBV carriers	1	5
Working in renal unit with HBV carriers	1	0
Nursing HBV carriers on medical ward or intensive care unit	2	2
Nursing patients with hepatitis B in mental hospital	1	0
Total	7 (29·2%)	8 (29·6%)

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Requests for reprints should be addressed to RW.

References

1 Trumbull ML, Greiner DJ. Homologous serum jaundice. *JAMA* 1951; **145**:965–7.
2 Denes AE, Smith JL, Maynard JE, Doto IL, Berquist KR, Finkel AJ. Hepatitis B virus infection in physicians. *JAMA* 1978;**239**:210–2.
3 Pattison CP, Maynard JE, Berquist KR, Webster HM. Serological and epidemiological studies of hepatitis B in haemodialysis units. *Lancet* 1973;ii:172–4.
4 Mosley JW, Edwards JM, Casey G, Redeker AG, White E. Hepatitis virus infection in dentists. *N Engl J Med* 1975;**293**:729–34.
5 Jones PO, Goldsmith HJ, Wright FK, Roberts C, Watson DL. Viral hepatitis: a staff hazard in the dialysis units. *Lancet* 1967;i:835–40.
6 Wands JR, Walker JA, Davis TT, Waterbury LA, Owens AH, Carpenter CCJ. Hepatitis B in an oncology unit. *N Engl J Med* 1974;**291**:1371–5.
7 Shanson DC. Hepatitis B outbreak in operating-theatre and intensive care staff. *Lancet* 1980;ii:596.
8 Szumness W, Stevens CE, Harley EJ, *et al*. Hepatitis B vaccine. Demonstration of efficacy in a controlled trial in a high risk population in the United States. *N Engl J Med* 1980;**303**:833–41.
9 Byrne EB. Viral hepatitis: an occupational hazard of medical personnel. *JAMA* 1966;**195**:362–4.
10 Anonymous. Hepatitis B virus infection among surgeons. *Lancet* 1980;ii: 300.
11 Stewart JS, Farrow LJ, Clifford RE. A three-year survey of viral hepatitis in West London. *Q J Med* 1978;**187**:365–84.
12 Public Health Laboratory Service. Acute hepatitis B. *Br Med J* 1975;iii: 603.
13 Grady GF, Bennet AJE, Culhane PD, Forrest JN, Iber FL. Eight years of surveillance of patients hospitalised with hepatitis. *J Infect Dis* 1972; **126**:87–91.
14 Gassner M, Grob PJ. Hepatitis epidemiologie in der Schweiz. *Schweiz Med Wochenschr* 1973;**103**:1829–33.
15 Pattison CP, Maynard JE, Berquist JR, Webster HM. Epidemiology of hepatitis B in medical personnel. *Am J Epidemiol* 1975;**101**:59–64.
16 Polakoff S. Problems of protection against virus B hepatitis. *Postgrad Med J* 1976;**52**:580–3.
17 Maynard JE. Viral hepatitis as an occupational hazard in the health care profession. In: Vyas GN, Cohen SN, Schmid R, eds. *Viral hepatitis*. San Francisco: Abacus Press, 1978:321–31.
18 Cossart YE. *Virus hepatitis and its control*. London: Ballière Tindall, 1977: 206.
19 Alter HJ, Chalmers TC, Freeman BM, *et al*. Health care workers positive for hepatitis B surface antigen. *N Engl J Med* 1975;**292**:454–7.
20 Williams SV, Pattison CP, Berquist KR. Dental infection with hepatitis B. *JAMA* 1975;**232**:1231–3.
21 Garibaldi RA, Rasmussen CM, Holmes AW, Gregg MB. Hospital acquired serum hepatitis (report of an outbreak). *JAMA* 1972;**219**: 1577–80.
22 Syndman DR, Hindman SH, Wineland MD, Bryan JA, Maynard JE. Nosocomial viral hepatitis B: a cluster among staff with subsequent transmission to patients. *Ann Intern Med* 1976;**85**:573–7.
23 Rimland D, Parkin WE, Miller GB, Schrack WD. An outbreak of hepatitis B traced to an oral surgeon. *N Engl J Med* 1977;**296**:953–8.
24 Levin ML, Maddrey WC, Wands JR, Mendeloff Ai. Hepatitis B transmission by dentists. *JAMA* 1974;**228**:1139–40.
25 Collaborative Study. Acute hepatitis B associated with gynaecological surgery. *Lancet* 1980;i:1–6.
26 Basendine MF, Chadwick RG, Salmeron J, Shipton U, Thomas HC, Sherlock S. Adenine arabinoside therapy in HBsAg positive chronic liver disease: a controlled study. *Gastroenterology* 1981;**80**:1016–22.
27 Maupas P, Chiron JP, Barin F, *et al*. Efficacy of hepatitis B vaccine in prevention of early HBsAg carrier state in children. *Lancet* 1981;i: 289–92.

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