siderable emotional and practical support at this time.[6 7] After discharge they are, like the patient, uncertain about how to behave, how protective they should be, and the long-term implications.

Relatively simple measures will probably be of substantial benefit in increasing understanding and reducing unnecessary social morbidity, and more complex rehabilitation should be reserved for the few who remain unnecessarily disabled. Awareness of the needs of patients and families, allocation of the responsibility for giving information, written guidance, and improved communication with the general practitioner should all be possible without making overwhelming demands on hospital staff.[1] There is a need for evaluation both of such measures and of exercise programmes and perhaps the use of nurses to supervise and co-ordinate convalescence.

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References

1 Working Party of the Royal College of Physicians and British Cardiac Society, *Journal of the Royal College of Physicians*, 1975, **9**, 281.
2 Ley, P, and Spelman, H S, *Communicating with the Patient*. London, Staples Press, 1967.
3 Dominian, J, and Dobson, M, *British Medical Journal*, 1969, **2**, 795.
4 Mayou, R A, Foster, A, and Williamson, B, *Journal of the Royal College of General Practitioners*. In press.
5 Cartwright, A, *Human Relations and Hospital Care*. London, Routledge and Kegan Paul, 1964.
6 *Lancet*, 1975, **2**, 355.
7 Shelton, M, and Dominian, J, *British Medical Journal*, 1973, **2**, 161.

Hospital Topics

Hepatitis B in retreat from dialysis units in United Kingdom in 1973

Public Health Laboratory Service Survey*

British Medical Journal, 1976, **1**, 1579-1581

Summary

A prospective study of hepatitis in over two-thirds of the dialysis units in the United Kingdom has continued since 1968. After the introduction of a control and preventive programme in 1970 there was a sustained decline in the incidence of hepatitis B to the low level of 0·3% among patients and 0·1% among staff in 1973—a greater than tenfold decrease in four years. Comparisons with data from other countries suggest that the almost complete elimination of hepatitis B from dialysis units in the UK was due to the preventive programme.

Introduction

A prospective survey of the incidence of hepatitis in haemodialysis units that began in 1968 was combined in January 1970 with a hepatitis B prevention programme, based on regular tests for hepatitis B surface antigen (HBsAg) of sera from patients and staff before entry to the units and at regular intervals afterwards, dialysis in isolation of infected patients, and appropriate cross-infection precautions. The study results showed a threefold decrease in the incidence of hepatitis B infections among patients and staff from 1970 to 1972. The results of the survey in 1973 are reported here.

Method

The method has been described elsewhere.[1 2] In addition, in 1973 records were completed for all patients who had been treated in the units and who were being maintained by dialysis at home or by transplants.

*The survey was co-ordinated and the report prepared by Sheila Polakoff, MD, MFCM, consultant epidemiologist, Epidemiological Research Laboratory, Central Public Health Laboratory, London NW9 5HT. The collaborators in the study are listed in the appendix.

Results

In 1973, 33 of the 48 haemodialysis and transplant units in the United Kingdom were included in the survey. Formerly records had not been returned for staff concerned with transplantation, and, although it was planned that this staff group would be included in 1973, few units returned the relevant records, so transplant staff continued to be excluded from estimates of incidence.

Hepatitis B outbreaks—There were no new outbreaks of hepatitis B in 1973, and hepatitis B infections developed in only three patients, who were all in one unit in which a hepatitis B outbreak had been in progress since 1970. This outbreak ended in the unit in mid-1973. Six months after the last HBsAg carrier was dialysed in the unit it was cleared of patients and equipment, thoroughly cleaned, disinfected, and re-equipped before patients, known to be HBsAg-negative, were admitted. In the associated transplant unit, however, HBsAg carriers continued to undergo transplantation, and, in contrast to the absence of infection among the staff of the haemodialysis unit, two nurses in the transplant unit developed hepatitis B infections in 1973. No hepatitis B infections of patients or staff in either the haemodialysis or the transplant unit were reported in the following year.

Introduction of HBsAg to units—HBsAg was not detected in the serum of any patient in the 32 units without outbreaks of hepatitis B. HBsAg was, however, introduced into a unit by a doctor who began duty without undergoing a HBsAg screening test; he worked in the unit for 12 weeks before he was identified as a symptomless HBsAg carrier and transferred to another department. No evidence of hepatitis B infection of patients or other staff of the unit was found then or later. This finding is in keeping with the outcome of two similar incidents in 1971, in each of which a nurse who was a HBsAg carrier worked in a unit for about three months without transmitting infection.

Incidence of hepatitis B—The incidence of hepatitis B infection in the survey units as a whole decreased to 0·3 per 100 patients, compared with 1·4 in 1972, 3·5 in 1971, and 4·9 in 1970 (table I). Among the staff of haemodialysis units the incidence rate, which rested solely on the HBsAg carrier doctor described above, was 0·1 per 100 persons, compared with 0·4 in 1972, 0·8 in 1971, and 1·3 in 1970.

TABLE I—*Incidence of hepatitis in patients and staff of haemodialysis units in 1970-3*

	Year	No of units surveyed	No of people in unit during year (for ≥1 week)	No of person years in unit	No of hepatitis infections			Incidence rate				
								HBsAg associated		Other	All	
					HBsAg associated	Other†	All	per 100 persons	per 100 person years	per 100 persons	per 100 persons	per 100 person years
Patients	1970	28	770	376	38	20	58	4·9	10·1	2·6	7·5	15·4
	1971	29	886	481	31	20	51	3·5	6·4	2·3	5·8	10·6
	1972	29	978	497	14	9	23	1·4	2·8	0·9	2·4	4·6
	1973	33	1034	458	3	45‡	48	0·3	0·7	4·4	4·6	10·5
Staff	1970	28	1421	835	19	2	21	1·3	2·3	0·1	1·5	2·5
	1971	29	1456	961	11	1	12	0·8	1·1	0·1	0·8	1·2
	1972	29	1372	979	6	1	7	0·4	0·6	0·1	0·5	0·7
	1973	33	1355	940	1*	1	2	0·1	0·1	0·1	0·1	0·2

*Two transplant nurses also infected in outbreak (see text).
†Clinical hepatitis non-B or serum aminotransferase or bilirubin levels above the upper limits of normal.
‡42 had abnormalities of serum aminotransferases only (see text).

Other types of hepatitis—The number of patients with abnormalities that might have been due to viral hepatitis rose to 45; in 42 cases the suspicion was based only on raised serum aminotransferase levels, but three patients had abnormal serum bilirubin levels also. None had any evidence of hepatitis B infection. The patients were reported from 11 units; one from each of five, two from two, and more than two from the remaining four. Of the four units with many cases, one was the unit with the hepatitis B outbreak, the end of which was complicated by the finding of abnormal aminotransferase levels in sera from some patients, though evidence of hepatitis B infection could not be detected in serial samples of their sera (taken at two-weekly intervals and tested by both immunoelectro-osmophoresis and radioimmunoassay). Another of the four units was one in which an outbreak of hepatitis, non-B, began before 1968[3] and in which patients with abnormal serum aminotransferase levels continued to be detected throughout 1969-73. Human normal immunoglobulin was used regularly for prophylaxis in only one of the 11 units. None of the staff of the 11 units developed clinical hepatitis. Among the staff of all 33 units there was only one case of clinical hepatitis, non-B; the infected nurse worked in a unit in which none of the patients showed any evidence of hepatitis. The incidence rate of other types of hepatitis among patients was higher in 1973—4·4 per 100 patients—than in the previous peak year, 1970, when the corresponding rate was 2·6 per 100 patients. Among staff the incidence rate of clinical hepatitis, non-B, remained the same—0·1 per 100 persons—during the four years 1970-3.

Prevalence of HBsAg carriage among patients—In 1973, 1913 patients with chronic renal failure were being maintained under the care of the 33 units, 1034 by dialysis in the units, 520 by dialysis at home (including one in hospital isolation), and the remaining 359 by transplanted kidneys. Of the 1913 patients 33 were HBsAg carriers in 1973 and a further eight—not tested in 1973—had been persistent carriers previously; if these eight are assumed to have continued HBsAg carriage the prevalence rate of HBsAg among all patients in 1973 was 2·1 per 100 patients (table II). The difference in the carrier rates between patients dialysed in the units and patients in the other two groups resulted from the policies adopted to control and prevent hepatitis B outbreaks. These 41 HBsAg carriers were the remainder of 106 infected patients who were involved in hepatitis B outbreaks or incidents in the 33 units during the years 1968-73. Of the 65 who did not appear in the prevalence rates in 1973 nine had clinical attacks—not confirmed by HBsAg tests—in hepatitis B outbreaks, 31 died, one emigrated before 1973, and 24 no longer had HBsAg in their sera in 1973. The 24 who became HBsAg-negative were maintained by dialysis; all 16 transplanted patients who were tested in 1973 remained HBsAg carriers.

TABLE II—*Prevalence during 1973 of HBsAg carriage among patients with chronic renal failure under care of survey units. Figures in parentheses include three patients on home dialysis and five with transplants who were previously HBsAg carriers but were not tested in 1973*

	Place and type of treatment in 1973			
	Dialysis		Transplanted	Total
	In unit	At home		
No of patients	1034	520†	359	1913
HBsAg carrier patients:				
No	2*	15 (18)	16 (21)	33 (41)
%	0·2	2·9 (3·5)	4·5 (5·8)	1·7 (2·1)

*Infected in 1973 during outbreak, then transferred to home dialysis.
†One HBsAg carrier in hospital isolation included.

Hepatitis B among home contacts—Three relatives—a husband, a wife, and a daughter—of three infected patients were reported to have developed clinical hepatitis B in 1973. The two spouses had been exposed to the HBsAg carrier patients for long periods before their attacks; in one case the patient had been a HBsAg carrier since 1970 and had undergone dialysis at home since 1971, and in the other case the patient was a HBsAg carrier who had had a functioning transplant since 1971. The daughter's illness, however, appeared four months after the onset of clinical hepatitis in her mother and three months after her transfer to home dialysis.

Blood transfusions, which had been reduced from an average of 6·5 units per patient year in 1970 to 2·7 units in 1972, were further reduced to 1·9 units per patient year in 1973.

Discussion

A decline in the incidence of a disease after a prevention programme has been introduced must, if a controlled study has not been made, lead to the question: Was the improvement due to the action taken? The only available approach to an answer is to compare the hepatitis B incidence rates found in this study with data available from dialysis units in countries in which similar prevention programmes were not used. Formerly such comparisons would have been considered biased by differences between countries in HBsAg carrier rates of blood donors. It is now generally accepted, however, that the role of blood transfusions has been exaggerated and that cross-infection within units is the chief means of perpetuating hepatitis B.[4 5] The data show that hepatitis B is still rife in dialysis units in Europe and in the USA. Out of about 11 000 patients in European units in 1973 10% had acquired HBsAg previously, and HBsAg was detected in the sera of a further 10% during that year.[5] Infection rates of staff in these units were not available. In over 20 units in the USA during the period mid-1972 to 1974 clinical hepatitis B or symptomless HBsAg developed in 24% of patients who had had no evidence of hepatitis infection at entry, who received no prophylaxis other than human normal immunoglobulin, and who remained in the units for a year. The corresponding attack rate for staff was 11%.[6] Compared with these rates, the reversal of a rising trend and the greater than tenfold decrease in the incidence of hepatitis B infection among staff and patients in the four years of the prevention programme in the UK is dramatic. Indeed, the programme might well be considered "a winner"[7] of so real and considerable an effect that the inclusion of a control group in the study was not essential.

An effective hepatitis B prevention programme should not only reduce infection in the units but also, by limiting cross-infection, keep to a minimum the pool of HBsAg carriers among all patients with chronic renal failure under the care of the units, including those being dialysed at home or maintained by transplants. These results show that out of almost 2000 patients being maintained by the survey units in 1973 only 2%

were HBsAg carriers. This compares favourably with the corresponding rates in 1973 for Europe as a whole: out of about 16 000 patients 18% were HBsAg carriers.[5] Similar data are not available for the USA, but a point prevalence study made in 15 centres in the USA during 1972-3 showed that HBsAg was detected in the sera of 17% of almost 600 patients[4]; this suggests that the prevalence in the USA is similar to that in Europe as a whole.

Another means of preventing hepatitis B in haemodialysis units is afforded by immunoglobulin prepared from human sera with high titres of antibody to HBsAg. Results of controlled trials with this material, performed in the USA[6] and Belgium,[8] showed that it had a substantial protective effect among patients. Nevertheless, among participants in the USA who received two doses of the material at four monthly intervals and who remained in the units clinical hepatitis B or HBsAg developed in no less than 4·9% of patients and 5·5% of staff within eight months of entry to the trial. Furthermore, there was no evidence of a reduction of persistent HBsAg carriage among those who were infected.

Regular prophylaxis with "specific" immunoglobulin should undoubtedly reduce the incidence of hepatitis B in heavily infected units, but this method used alone is unlikely to lead to the low level of infection observed in the survey units in the UK in 1973.

Analyses of the survey results for 1974-5 are incomplete, but the records suggest that hepatitis B incidence rates remain as low as in 1973. This being so, routine prophylaxis with "specific" immunoglobulin seems unnecessary in the UK, but supplies of the material, held by the Public Health Laboratory Service, are available to unit directors as an additional precautionary measure when an antigenaemic patient is detected in a unit or when staff or home dialysis assistants sustain any of the following accidents with material containing HBsAg: inoculation, ingestion, contamination of mucous membranes, or cuts or abrasions of skin.

Exclusion of staff who are HBsAg carriers from work in the units is part of the prevention programme, though there is no clear evidence that a hepatitis outbreak has ever originated from a healthy carrier among the staff in a dialysis unit in the UK. The fact that in 1973, for the third time during the survey, a healthy HBsAg carrier worked in a unit for three months without sequelae suggests that such staff present little hazard of infection to even the most vulnerable patients. The danger of hepatitis B outbreaks in dialysis units is so great, however, that unit directors are unlikely to accept staff who are carriers until there is further evidence that they are innocuous.

The remaining question raised by the results in 1973 is whether the increased number of reports of patients with abnormal serum aminotransferase levels indicate that viral hepatitis, non-B, is now becoming a problem in the units. Though continued surveillance is certainly required, there are two reasons for believing that there is as yet no need for alarm. Firstly, some of the increase is probably artificial: the Department of Health and Social Security's advisory report,[9] which became available in July 1972, recommended that serum aminotransferase and bilirubin levels in patients' sera should be measured each month in addition to HBsAg tests; a large increase in the number of tests made probably revealed transient abnormalities that formerly remained undetected. Unit directors ascribed many of these abnormalities to causes other than viral hepatitis, but by definition in this survey any patient with a serum aminotransferase level above the upper limit of normal is included in the group of those who may have other types of hepatitis. Secondly, none of the staff of the 11 units in which the patients with abnormal serum aminotransferase levels were treated developed clinical hepatitis even though none—except in one unit—was given human normal immunoglobulin prophylactically. If these abnormalities among patients are due to viral hepatitis the absence of obvious illness among the staff cannot be accounted for unless it is assumed that either the causal viruses rarely cause clinical illness or they are so common that all the staff have acquired immunity from previous exposures.

We are grateful to the dialysis unit staff who completed the records and dispatched the specimens. We thank Mrs J Miller and other members of the staff of the Epidemiological Research Laboratory for helping with the co-ordination of the survey.

Appendix

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References

1. Polakoff, S, Cossart, Y E, and Tillet, H E, *British Medical Journal*, 1972, **3,** 94.
2. Public Health Laboratory Service, *British Medical Journal*, 1974, **4,** 751.
3. Eastwood, J B, *et al*, *Annals of Internal Medicine*, 1968, **69,** 59.
4. Szmuness, W, *et al*, *Journal of the American Medical Association*, 1974, **227,** 901.
5. Parsons, F M, *et al*, *Proceedings of the European Dialysis and Transplant Association*, 1974, **11,** 3.
6. Prince, A M, *et al*, *New England Journal of Medicine*, 1975, **293,** 1060.
7. Hill, A, *Principles of Medical Statistics*. London, Lancet, 1966.
8. Desmyter, J, *et al*, *Lancet*, 1975, **2,** 377.
9. Department of Health and Social Security, *Report of the Advisory Group on Hepatitis and the Treatment of Chronic Renal Failure, 1970-2*. London, DHSS, 1972.

Over 50 strains of Salmonella alachua have been isolated in two hospitals in an Indian city from the cerebrospinal fluid in 40 cases of neonatal meningitis and in some cases of diarrhoea in babies aged under 6 months. What are the clinical features of this infection in man, particularly neonates?

Bacterial meningitis may occur after infection with almost any salmonella serotype, and I am not aware that some serotypes are more likely than others to cause this complication. Some salmonellas (other than those causing typhoid) are said to be especially likely to produce systemic infection, but *Salmonella alachua* is not recognised as one of these. If the neonates were born in the two hospitals mentioned it is more likely that *S alachua* was endemic in the neonatal or children's wards and that the infections were acquired by cross-infection. The continuous existence of a common source of the organism such as the local milk supply should also be considered. In salmonella infection the risk of meningitis is greater in neonates than in adults or children,[1 2] and this could explain the unexpectedly high number of cases due to this particular salmonella. It would be of interest to know if *S alachua* has been isolated from any mothers, members of staff, or the environment in these wards.

1. Saphra, I, and Winter, J W, *New England Journal of Medicine*, 1957, **256,** 1128.
2. Riley, H D, and Diardoff, M, *Journal of the Oklahoma State Medical Association*, 1962, **55,** 10.