Methods and results

A brief questionnaire with a letter requesting cooperation was circulated to all 1250 general surgeons in the United Kingdom on the register of a commercial mailing house. The surgeons were not required to identify themselves. Information was obtained on experience with the Angelchik prosthesis by the questions: "Have you ever used the Angelchik prosthesis? How many prostheses have you put in? Have you had any complications?"

Specific questions were included about severe dysphagia, erosion into the gastrointestinal tract, and any other complications. The surgeons were asked if they had removed any Angelchik prostheses and, if so, how many and for what reasons. They were also asked how many operations for hiatus hernia and reflux they did yearly.

Five hundred and thirty (42%) surgeons replied, of whom 134 (25%) had used the prosthesis and implanted a total of 1013 (about three quarters of the total sales in the United Kingdom at that time). The mean (2 SE) number of prostheses implanted by each surgeon was 7.6 (1.8). Surgeons who used the Angelchik prosthesis performed a mean of 8.2 (1.2) antireflux operations yearly whereas non-users performed 3.6 (0.4) operations.

Forty six surgeons reported that complications had occurred in 91 (9%) patients; severe dysphagia was by far the commonest complication (66 patients), but nine patients had developed the serious complication of erosion. Thoracic migration was seen in six patients and distal migration in one. Disruption of the tapes, which was a problem with an early version of the prosthesis, was seen in three patients. Only two patients apparently developed appreciable sepsis. Oesophageal perforation early in the postoperative period, as opposed to late erosion, was seen in one patient. Fatal pericarditis due to erosion of the prosthesis was seen in one patient and atrial compression in one.

Fifty two prostheses (5.1%) were removed, and again by far the commonest reason was dysphagia (27 cases), followed by erosion (nine), migration (seven), disruption of the tapes (three), and sepsis (two).

Four prostheses were removed because of pain, oesophageal perforation, atrial compression, and pericarditis.

The experience of the surgeon did not seem to relate to postoperative complications or rate of removal. The surgeons who had implanted five prostheses or fewer had a rate of removal of 7% compared with 5.5% for surgeons who had used more than 20.

Comment

With the increasing use of the Angelchik antireflux prosthesis complications are now being reported. We believe that the results of new procedures should be assessed in widely based studies such as ours rather than solely on the results of experienced enthusiasts operating in teaching centres. Although only 42% of surgeons replied to our questionnaire, those using the prosthesis accounted for about three quarters of the total number of prostheses sold in the United Kingdom at that time, and this is by far the largest collection of such data in the world.

Surgeons who used the Angelchik prosthesis performed more antireflux procedures each year than non-users, and the prosthesis accounted for only a part of their experience. More than half of the prostheses were inserted by surgeons who had performed the procedure 20 times or less often, but the rate of removal in this group was not different from that among the 18 surgeons who had done more than 20 procedures and were thus more experienced. The serious complications seen included severe dysphagia, erosion, and migration; these complications and other less frequent problems necessitated the removal of 5% of the prostheses. This is comparable with the revision rate after the Nissen procedure. The long term effects of the Angelchik procedure are not yet known, and the rates of complication and removal may increase with time. Long term follow up of these patients is clearly required, and we seek the continued cooperation of surgeons in the United Kingdom.

We thank all the surgeons who took the time to help with this survey and Forth Medical and the Mentor Corporation for financial support.

- Kosarek RA, Phelps JE, Sanowski RA, Grobe JL, Fredell CH. An anti-reflux prosthesis in the treatment of gastro-oesophageal reflux. *Ann Intern Med* 1983;98:310-5.
- Morris DL, Jones J, Evans DF, et al. Reflux versus dysphagia—an objective evaluation of the Angelchik prosthesis. *Br J Surg* 1985;72:1017-9.
- Ubhi C, Morris DL, Hardcastle JD. Dysphagia following Angelchik insertion. *Thorax* 1986;41:655-6.
- Mansour KA, McKeown PP. Disastrous complications of the Angelchik prosthesis. *Am J Surg* 1983;99:616-8.
- Lackey C, Potts J. Penetration into the stomach. A complication of the anti-reflux prosthesis. *JAMA* 1982;248:350.

(Accepted 1 May 1987)

Department of Surgery, University Hospital, Nottingham NG7 2UH

D L MORRIS, FRCS, MD, senior lecturer in surgery
C S ROBERTSON, FRCS, research registrar
J D HARDCASTLE, FRCS, FRCP, professor of surgery

Correspondence to: Mr Morris.

Does screening high risk dental patients for hepatitis B virus protect dentists?

Markers of past or current infection with hepatitis B virus (HBV) are present in up to 24% of dental surgeons and technicians¹; the risk of infection correlates with age and the nature of exposure.² Current recommendations are that patients from well defined high risk groups should be screened before having dental treatment so that appropriate precautions can be taken.³ We evaluated the efficiency of such screening in a dental clinic in an area with a high prevalence of drug abuse and a substantial proportion of patients who were born, or descended from people born, in places where HBV is endemic.

Methods and results

Dentists at the King's College School of Dentistry were advised to screen patients from high risk groups for infection with HBV according to recommended guidelines.³ Altogether 2000 consecutive requests for screening (September 1983 to July 1986) were analysed for the reason that the request was made and whether hepatitis B surface antigen (HBsAg) was present in serum. Over the same period 53 000 patients were treated on 409 000 occasions without screening. Patients known to be positive for HBsAg were excluded from the analysis. HBsAg was sought by radioimmunoassay and positive results confirmed by reverse passive haemagglutination.

Sixty two (3.1%) of the high risk patients were positive for HBsAg (table),

Reasons for requesting screening for HBV in 2000 dental patients, and presence of HBsAg in serum

Reason for request	No tested	No (%) positive for HBsAg
Tattoos	801	3 (0.4)
History of hepatitis or jaundice (>12 months previously)	537	10 (1.9)
Racial origin (from South East Asia and Far East)	408	40 (9.8)
Intravenous drug addiction	51	1 (2.0)
Racial origin (from Africa, Caribbean, and Mediterranean)	48	3 (6.2)
Current hepatitis, liver disease, or jaundice (not known to be positive for HBsAg)	16	3 (18.7)
Alcoholism	12	0
Haemophilia	5	1 (20.0)
Homosexuality	3	0
Others or unstated	119	1 (0.8)

which was a lower proportion than we had expected. The two main reasons for requesting screening—that the patients had tattoos or a history of hepatitis or jaundice—identified only 13 carriers of HBV in 1338 patients screened; of these, three were later found to be homosexuals or intravenous drug addicts.

Comment

Estimates of the prevalence of carriers of HBV in the United Kingdom are based on results from blood donors, which may not accurately reflect the contribution of race and excludes people with liver disease. Thus estimates of the prevalence as being 0.1% to 0.4% may underestimate the problem, particularly in inner city areas; up to 1.5% of unselected patients admitted to North American hospitals⁴ and 2.1% of pregnant women in Holland⁵ are positive for HBsAg. If we make the conservative estimate that 0.4% of the local population are positive for HBsAg the contrast between the expected number (220) and the actual number (62) of carriers of HBV identified is disconcerting, and the true difference is probably even greater. The problems in identifying three important groups probably account for the discrepancy: only three homosexuals were screened in 55 000 patients; more people from South East Asia were screened than from Africa, the Caribbean, or the Mediterranean, which is the inverse of the local population distribution; and, finally, although 51 drug addicts were screened, only one was positive for HBsAg, yet in our experience as many as 58% of drug addicts show evidence of past or current infection with HBV.

The reasons for inadequate screening are not clear but may include a failure to ask specifically about drug abuse or homosexuality; embarrassment at screening on grounds of race; concern at the time spent awaiting results; and even deliberate concealment by patients fearing that disclosure might adversely affect treatment. The screening must be regarded as unsatisfactory as appropriate precautions were not taken when unscreened patients were seen, many of whom were probably infected with HBV.

Our results show that neither a tattoo nor a history of hepatitis or jaundice more than 12 months previously is associated with an increased prevalence of infection with HBV; efficiency of screening would be improved, therefore,

by no longer screening patients in these two categories. Our results, however, question the validity of screening to protect dentists. A more logical approach would be to encourage vaccination of all staff at risk of infection with HBV at the earliest possible opportunity and to assume that all patients are a potential source of infection. We have recently adopted this policy.

- 1 Schiff ER, de Medina M, Woodman S, Katz B, Kline S. VA co-operative study on hepatitis and dentistry. *Hepatology* 1982;2:688.
- 2 Epstein JB, Bucher BK, Bouchard S. Hepatitis B and Canadian dental professionals. *Journal of the Canadian Dental Association* 1984;7:555-9.
- 3 Scully C. Hepatitis B: an update in relation to dentistry. *Br Dent J* 1985;159:321-8.
- 4 Linneman CG, Hegg ME, Raymundo N, Schiff GM. Screening hospital patients for hepatitis B surface antigen. *Am J Clin Pathol* 1977;67:257-9.
- 5 Mazel JA, Heijunk RA, Schalm SW. Gecomineerde passieve en actieve immunisatie van zuigelingen van HBsAg moeders. *Ned Tijdschr Geneesk* 1985;129:150.

(Accepted 15 May 1987)

Liver Unit and Department of Primary Dental Care, King's College Hospital and School of Medicine and Dentistry, London SE5 8RX

HEATHER M SMITH, BSC, senior biochemist
GRAEME J M ALEXANDER, MRCP, honorary lecturer
WARREN BIRNBAUM, BDS, FDSRCS, associate specialist
ROGER WILLIAMS, MD, FRCP, director, liver unit

Correspondence to: Dr Williams.

Does nifedipine ameliorate cyclosporin A nephrotoxicity?

Nephrotoxicity is a serious disadvantage of cyclosporin A and may lead to irreversible loss of renal function.¹ Recent evidence suggests that it may be a haemodynamic phenomenon due to intrarenal vasoconstriction.^{2,3} Studies in animals show that the calcium channel blocker nifedipine may reverse the effects of long term treatment with cyclosporin.⁴ We report the influence of nifedipine on graft function in a retrospective study of renal transplant recipients given cyclosporin.

Patients, methods, and results

We reviewed 106 patients who had stable transplant function at least three months after grafting and were taking cyclosporin. Cyclosporin (usually combined with low dose prednisolone) was given in maintenance doses of 5-8 mg/kg/day, and trough whole blood cyclosporin concentrations were measured by high pressure liquid chromatography. Renal function was assessed by measuring the serum creatinine value. Hypertension (diastolic blood pressure ≥ 95 mm Hg requiring drug treatment) was usually treated by β blockers combined with vasodilators; since 1984 prazosin and hydralazine had increasingly been superseded by nifedipine. Choice of blood pressure treatment was based on individual clinical decision. All rejection episodes had been confirmed histologically.

Hypertension was present in 90 (85%) of the patients taking cyclosporin (compared with 54% of our patients receiving azathioprine; $p < 0.001$). For further analysis patients receiving nifedipine ($n = 58$) were separated from those who were hypertensive but not taking nifedipine ($n = 32$) and from the normotensive group ($n = 16$) (table). The three groups did not differ in distribution of primary renal disease leading to end stage renal failure, incidence of bilateral nephrectomy, and use of prednisolone.

Those receiving nifedipine had their grafts for a shorter mean period ($p < 0.001$), which was reflected in a significantly higher mean dose of cyclosporin ($p < 0.02$) and higher whole blood concentrations of the drug ($p < 0.05$). Despite this, renal function was better in those receiving nifedipine compared with

hypertensive patients not receiving nifedipine (serum creatinine concentration 153 (SEM 10) v 183 (13) $\mu\text{mol/l}$; $p < 0.05$) and similar to that in normotensive patients taking cyclosporin (154 (11) $\mu\text{mol/l}$). Blood pressure was controlled to an equivalent, acceptable degree in both hypertensive groups.

Rejection was infrequent during treatment with cyclosporin, only 33 of the 106 patients having suffered a rejection episode. More patients in group 2 had suffered rejection episodes (14/32; 43.8%) compared with group 3 (15/58; 25.9%) and group 1 (4/16; 25%) ($p < 0.05$). Analysis of the 71 patients who had never had a rejection episode, however, showed that the serum creatinine concentration among those receiving nifedipine was still lower than in hypertensive patients not receiving nifedipine (141 (6.8) v 175 (22) $\mu\text{mol/l}$; $p < 0.05$) and comparable to that in normotensive patients.

Comment

This retrospective analysis has identified a cohort of renal transplant recipients receiving nifedipine for cyclosporin related hypertension who, owing to shorter graft duration, high cyclosporin dosage, and higher blood concentrations of cyclosporin, would have been at risk of more severe cyclosporin nephrotoxicity. These patients, however, had better graft function than equivalent hypertensive patients receiving other drug treatment. This finding was sustained even when the possible influence of previous graft rejection was excluded.

Though cyclosporin may produce irreversible loss of renal function,¹ the more characteristic pattern of a raised serum creatinine concentration rapidly falling when cyclosporin dosage is reduced suggests a haemodynamic abnormality. Studies in rodents suggest that cyclosporin produces intrarenal vasoconstriction,² and isotope studies in patients with renal transplants have shown increased renal vascular resistance which is corrected with a parallel fall in serum creatinine concentration when cyclosporin is replaced by azathioprine.³

The role of vasodilator agents to reverse cyclosporin nephrotoxicity has not been studied in man. Animal studies suggest that nifedipine is superior to α adrenergic blockade, converting enzyme inhibition, and manipulation of prostaglandins in reversing cyclosporin induced intrarenal vasoconstriction.⁴ It is not clear if this is a specific effect of calcium channel blockade, though there is some evidence that calcium channel blockers prevent cyclosporin uptake by kidney cells in culture.⁵

Nifedipine appears to be of potential value as a cyclosporin nephroprotective agent, and further prospective haemodynamic studies are justified.

- 1 Myers BD, Ross J, Newton L, Luetscher J, Perloth M. Cyclosporine-associated chronic nephropathy. *N Engl J Med* 1984;311:699-705.
- 2 Dieperink H, Leyssac P, Starklint H, Kemp E. Nephrotoxicity of cyclosporin A. A lithium clearance and micropuncture study in rats. *Eur J Clin Invest* 1986;16:69-77.
- 3 Curtis JJ, Luke RG, Dubovsky E, et al. Cyclosporin in therapeutic doses increases renal allograft vascular resistance. *Lancet* 1986;ii:477-9.
- 4 Dieperink H, Leyssac P, Starklint H, Jorgensen KA, Kemp E. Antagonist capacities of nifedipine, captopril, phenoxylbenzamine, prostacyclin and indomethacin on cyclosporin A induced impairment of rat renal function. *Eur J Clin Invest* 1986;16:540-8.
- 5 Nagineni CN, Yamagawa N, Musra B, Lee DBN. Cyclosporin A-calcium channels interaction: a possible mechanism for nephrotoxicity. *Transplant Proc* 1987;19:1358-62.

(Accepted 26 May 1987)

Departments of Nephrology and Surgery, Leicester General Hospital, Leicester LE5 4PW

J FEEHALLY, DM, MRCP, senior registrar in nephrology
J WALLS, FRCP, consultant nephrologist
N MISTRY, BSC, MPHIL, senior medical laboratory scientific officer
T HORSBURGH, PHD, principal biochemist
J TAYLOR, FRCS, transplant fellow
P S VEITCH, FRCS, consultant surgeon
P R F BELL, MD, FRCS, professor of surgery

Correspondence to: Dr Feehally, department of nephrology.

Characteristics of normotensive and hypertensive renal transplant recipients and influence of nifedipine. Mean values expressed with SEM in parentheses

	No	Age (years)	Graft duration (months)	Mean arterial blood pressure (mm Hg)	No (%) of patients not receiving corticosteroids	Cyclosporin dose (mg/kg/day)	Whole blood cyclosporin concentration ($\mu\text{g/l}$)	Serum creatinine ($\mu\text{mol/l}$)
Group 1: normotensive	16	49.3 (3.8)	21.8 (3.0)	98.3 (2.9)	3 (19)	5.7 (0.4)	293 (39)	154 (11)
Group 2: hypertensive, not taking nifedipine	32	39.1 (2.2) $\S$	28.9 (2.8)	101.5 (2.1)	6 (19)	5.7 (0.2)	270 (22)	183 (13) $\dagger\dagger$
Group 3: hypertensive, taking nifedipine	58	42.5 (2.0)	16.7 (1.4)**	97.4 (1.2)	10 (17)	7.2 (0.4) $\ddagger$	327 (17)*	153 (10)

* $p < 0.05$ compared with group 2.

** $p < 0.001$ compared with group 2.

$\dagger p < 0.02$ compared with groups 1 and 2.

$\ddagger p < 0.05$ compared with groups 1 and 3.

$\S p < 0.02$ compared with group 1.

(Differences compared by Student's t test.)