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Costs of infection control in endoscopy units

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Bronchoscopists face problems in reconciling hygienic practices with service demands and limited resources. We prospectively assessed the costs of three different approaches to controlling infection in endoscopy units.

Methods and results

The table gives details of the three policies; they each lasted four weeks and were implemented consecutively in our unit. The first policy relied on infected patients being identified before extra precautions were adopted. The second and third policies assumed all body fluids to be infected with HIV and hepatitis B virus, the third policy differing from the second only in having longer disinfection times to meet the Department of Health's recommendations on mycobacteria.[1] Costs of maintaining and repairing bronchoscopes for the preceding four years were obtained from the suppliers (Keymed). Screening all patients for hepatitis B before bronchoscopy was mandatory under hospital policy. The policy was to warn the nurse if a patient had hepatitis or suspected mycobacterial infections. Actual mycobacterial infections and compliance with hepatitis screening were determined retrospectively from laboratory records.

Forty two, 40, and 44 bronchoscopies were performed during the first, second, and third policies respectively. Three or more patients underwent bronchoscopy consecutively on 10 occasions. On six of these occasions, during the third policy, three bronchoscopes were used to avoid a delay of half an hour between the second and third patients. All other lists were completed without delays with two bronchoscopes. Gown and gloves were worn almost universally during the first policy, though their use was discretionary. Compliance with all three policies was excellent.

The capital cost of each policy was essentially that of a bronchoscope (£7756), biopsy forceps (£261), and reusable cytology brush (£199). Two of each were required for the first and second policies and a third for the third policy. The mean cost of maintaining and repairing each bronchoscope was £86.40 a year.

The mean cost of consumables and laundry per bronchoscopy was £17.50, £15.40, and £12.70 for the three policies respectively. The first policy proved more expensive because of special provision made for the one patient positive for HIV antibody. This entailed replacing a suction bag and gown and linen, though they were not soiled and were normally reused (cost £7.80); and discarding part of the cytology brush (cost £99). These two factors contributed £2.54 to the cost per bronchoscopy under the first policy; the cost for each non-infected patient under this policy was therefore £14.96. Further differences in cost were largely unrelated to infection control and were due, for example, to differences in drugs and the number of samples taken.

In addition to the cost of consumables under the first policy the cost of screening for hepatitis and HIV (72p and £1.15 respectively for each patient screened) should be added.

Screening for hepatitis B, though mandatory, was performed on only 79 of the 126 (63%) patients, all of whom were negative. The bronchoscopy nurse was not informed of any possible mycobacterial infections. One patient during the first policy, however, had *Mycobacterium tuberculosis* in her sputum at bronchoscopy, and another, who underwent bronchoscopy during the third policy, had *M xenopi*.

Summary of policies for infection control

	Policy 1	Policy 2 (all patients except those with tuberculosis)	Policy 3 (all patients)
Endoscopist and assistant	Gown* (optional) Gloves (optional) Gown, gloves, mask† Eye protection†	Gown,* gloves, mask Eye protection	As for policy 2
Patient	Paper roll on couch Linen pillow case* Blanket* Paper drape† on couch Paper pillowcase† Blanket†	Paper roll on couch Linen pillow case* Blanket*	As for policy 2
Cleaning and disinfection	Clean in detergent Sonicate brushes and biopsy forceps Disinfect in 2% glutaraldehyde for >5 minutes; rinse in 70% alcohol Bleach (10%) to surfaces and floor at end of day Disinfect in 2% glutaraldehyde for >60 minutes† Bleach (10%) to surfaces and floor†	Clean in detergent Sonicate brushes and biopsy forceps Disinfect in 2% glutaraldehyde for >30 minutes routinely, for 60 minutes if tuberculosis suspected; rinse in 70% alcohol Bleach (10%) to surfaces and floor at end of day or after spill of body fluid	As for policy 2 but disinfect for 60 minutes routinely
Equipment	Silicon suction tubing changed for each case New suction bag for each list Suction tubing and bag†	Single pack plastic suction tubing for each case New suction bag for each list	As for policy 2
Linen	Dispose in skip Put in plastic bag†	Dispose in skip; put in plastic bag if soiled with body fluids	As for policy 2
Specimens	Cytology brush and biopsy forceps reused Transported in self sealing plastic bag Central wire from cytology brush discarded† Put in two bags with hazard label†	Cytology brush and biopsy forceps reused Transported in self sealing plastic bag	As for policy 2

*Item reused until soiled.
†Procedure for infectious patients, such items used only once.

Comment

Since the emergence of HIV a uniform policy has been advocated as the only rational approach to controlling infection.[2,3] For endoscopy units a uniform policy is less disruptive and costs no more than current two tier policies; indeed, with increasing numbers of infectious patients two tier policies become more expensive. The effect of length of procedure and time for disinfection on the number of instruments needed has been discussed.[4] Some units would need to buy extra fibrescopes and to replace older, non-immersible models with immersible ones. Over half of the units already have three or more bronchoscopes and over a third have two; a fifth reserve a bronchoscope for infected patients (S E Church, personal communication). A uniform policy could release these instruments for general use.

The policies detailed here are illustrative. The British Thoracic Society has recently circulated its members with

specific recommendations for a uniform policy of infection control (*BTS Newsletter*, winter 1988).

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Carpal tunnel syndrome and type of dialysis membrane

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The carpal tunnel syndrome is commonly seen in patients who have received haemodialysis for a long time.[1,2] Compression of the median nerve, flexor tenosynovitis, and destructive arthropathies are associated with hypertrophic material, which generally contains amyloid derived from β_2 microglobulin. The pathogenesis of the syndrome is unknown, although possibly the dialysis membrane has an effect. The syndrome has been reported in patients treated with cellulose membranes non-permeable to β_2 microglobulin. The prevalence of the syndrome increases with the duration of dialysis, and nearly all patients treated for more than 18 years require a decompression operation.

Few data have been reported on the occurrence of amyloidosis due to dialysis in patients treated with synthetic membranes permeable to β_2 microglobulin.[1] Since the early 1970s we have used the highly permeable polyacrylonitrile AN 69 membrane for dialysis. We report the prevalence of the syndrome in our patients.

Patients, methods, and results

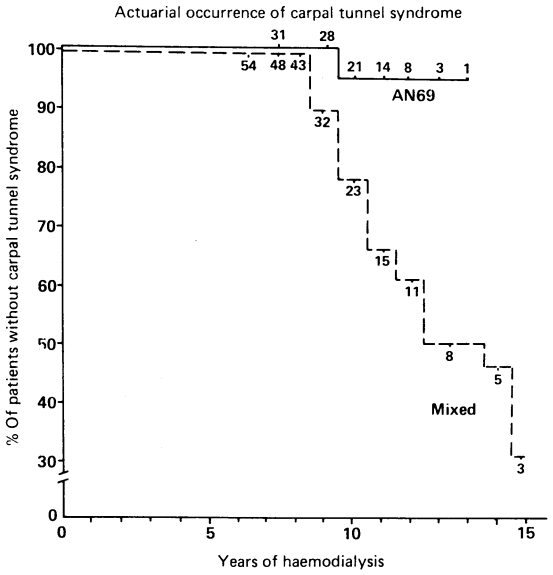
Eighty five patients who had received dialysis for more than seven years were entered into the study. Two groups were selected: group 1 comprised 31 patients who had been treated exclusively with AN 69, and group 2 comprised 54 patients who had been treated with cuprophane for an average of 5·7 (SD 3·9) years and subsequently with AN 69. The mean duration of dialysis was 124·2 (33·0) months in group 1 and 126·9 (28·6) months in group 2. The mean time for which patients had been exposed to the artificial membrane was 5533 (1200) hours and 7580 (1984) hours respectively ($p<0·05$). The mean age of the groups was 54 (3) and 49 (14), and the mean serum β_2 microglobulin concentration was 49 (15) mg/l and 45 (13) mg/l.

The carpal tunnel syndrome was considered to be present only when a decompression operation was needed, thus avoiding clinical bias by using motor nerve conduction velocity as reduced velocity may be due to uraemic toxicity without entrapment of the median nerve. After 10 years of dialysis only one patient in group 1 had had the operation, compared with 16 in group 2 ($p<0·001$). Amyloidosis derived from β_2 microglobulin was proved in the only patient in group 1 with the syndrome and in eight out of 12 patients in group 2 from whom synovial specimens were available.

Cumulative survival analysis with the Mantel-Cox model applied to the syndrome at the time of operation showed a significant difference ($p=0·0118$) between the two techniques of dialysis. The proportion of patients without the syndrome after 10 years was predicted to be 94% (n=18) in those treated with AN 69 and 77% (n=21) in those treated with several membranes; that at 12 years was predicted to be 94% (n=8) and 61% (n=11) respectively.

Comment

The carpal tunnel syndrome was less common in patients treated exclusively with the highly permeable membrane AN 69 than in patients who had been exposed to cuprophane for at least five years. Preliminary studies have indicated a similar trend for juxta-articular cystic bone defects, which have been assumed to be related to amyloidosis derived from β_2 microglobulin.[3] Except for the type of membrane used and the time for which patients had been exposed to the membrane there were no clinical or biological differences between the two groups.



Cumulative percentage of patients without carpal tunnel syndrome, calculated from life tables and survival analysis, in patients given dialysis with AN 69 membrane (———) and with several membranes including cellulose membrane (÷÷÷). Figures are numbers of patients. Date of onset of carpal tunnel syndrome was considered to be date of surgery

There are several ways in which AN 69 might prevent or postpone the development of the carpal tunnel syndrome. The concentration of β_2 microglobulin substrate may be insufficient to allow amyloid to form after 10 years of dialysis. With a sieving coefficient of 0·40 for β_2 microglobulin up to about 22·5 g of the protein is extracted each year,[4] but despite this serum β_2 microglobulin concentrations remain raised and it would be naive to assume that the serum β_2 microglobulin concentration alone causes amyloidosis in the absence of other promoting factors such as changes in the structure of the precursor protein in the serum[5] and transformation of the precursor protein in situ by some local process. Alternatively, because AN 69 is more biocompatible than cellulose mem-