30 Goldberg DP. *The detection of psychiatric illness, by questionnaire. Maudsley monograph No 21.* Oxford: Oxford University Press, 1972.
31 Goldberg JD, Williams P. *A user's guide to the general health questionnaire.* Windsor: NFER-Nelson, 1988.
32 Snaith RP, Bridge GWK, Hamilton M. The Leeds scales for the self-assessment of anxiety and depression. *Br J Psychiatry* 1976;128:156-65.
33 Brooks DN. Psychological deficits after severe blunt head injury: their significance and rehabilitation. In: Osborne DJ, Gruneberg MM, Eiser JR, eds. *Research in psychology and medicine.* Vol 2. London: Academic Press, 1979:469-766.
34 Mahoney FH, Barthel DW. Function evaluation: The Barthel index. *Maryland State Medical Journal* 1965;14:61-5.
35 Jennett B, Bond MR. Assessment of outcome after severe brain damage: a practical scale. *Lancet* 1975;ii:480-4.
36 Livingstone MG, Livingston HM. The Glasgow assessment schedule: clinical and research assessment of head injury outcome. *International Rehabilitation Medicine* 1985;7:145.
37 SPSS. *Statistical package for the social sciences.* PC+version 3·1. Chicago: SPSS, 1989.
38 Everitt BS, Dunn G. *Applied multivariate data analysis.* London: Edward Arnold, 1991.
39 Statistics and Epidemiology Research Corporation. *EGRET: epidemiological graphics, estimation and testing.* Seattle, WA: Statistics and Epidemiology Research, 1990.
40 Scottish Agricultural Statistical Service. *REML user's manual.* 1987.
41 Intagliata J. Improving the quality of community care for the chronically mentally disabled: the role of case management. *Schizophr Bull* 1982;8:655-74.
42 Henderson NG, Wallack SS. Evaluating case management for catastrophic illness. *Business in Health* 1987 Jan: 7-11.
43 Matson JD. Independent third party case management. In: Rapp D, ed. *Brain injury casebook. Methods for reintegration into home, school, and community.* Springfield, IL: Thomas, 1986.
44 Dixon TP, Goll S, Stanton KM. Case management issues and practices in head injury rehabilitation. *Rehabilitation Counselling Bulletin* 1988;31:325-43.
45 Bush GW. Catastrophic case management: thoughts from a teacher/consumer/advocate. *Brain Inj* 1989;3:91-100.
46 McKeganey N, Hunter DJ. "Only Connect....": tightrope walking and joint working in care of the elderly. *Policy and Politics* 1986;14:335-60.
47 Friend JK. Planning—the act of responsible scheming. *Linkage* 1983;7:8-15.
48 Ovretveit J. Case management: notes for the perplexed. *Clinical Psychology Forum* 1991;36:3-7.
49 Bachrach LL. Case management: toward a shared definition. *Hosp Community Psychiatry* 1989;40:883-4.
50 Kanter J. Clinical case management: definition, principles, components. *Hosp Community Psychiatry* 1989;40:361-8.
51 Holloway F. Case management for the mentally ill: looking at the evidence. *Int J Soc Psychiatry* 1991;37;2-13.
52 Prigitano GP, Fordyce DJ, Zeiner H, eds. *Neuropsychological rehabilitation after brain injury.* Baltimore, MD: Johns Hopkins University Press, 1986.
53 Burke WH, Wesolowski MD, Guth MC. Comprehensive head injury rehabilitation: an outcome evaluation. *Brain Inj* 1988;2:313-22.
54 Wehman PH, Kreutzer JS, West MD, Shevron PD, Zasler ND, Groah CH, *et al.* Return to work for persons with traumatic brain injury: a supported employment approach. *Arch Phys Med Rehabil* 1990;71:1047-52.
55 Franklin JL, Solovitz B, Mason M, Clemons JR, Miller GE. An evaluation of case management. *Am J Public Health* 1987;77:674-8.
56 Brooks DN, McKinlay WW, Symington C, Beattie A, Campsie L. Return to work within the first seven years of severe head injury. *Brain Inj* 1987;1:5-19.

(Accepted 17 February 1994)

Errors in blood transfusion in Britain: survey of hospital haematology departments

D B L McClelland, P Phillips

Edinburgh and South East Scotland, Blood Transfusion Service, Royal Infirmary of Edinburgh, Edinburgh EH3 9HB
D B L McClelland, *director*
P Phillips, *medical audit coordinator of Scottish National Blood Transfusion Service*

Correspondence to:
Dr McClelland.

BMJ 1994;308:1205-6

Blood transfusion laboratories aim to provide a high quality service with minimum risk to patients. British guidelines for good practice in transfusion medicine exist,[1] and most hospitals have local protocols. If these procedures fail incompatible blood may be transfused, which could lead to potentially fatal haemolytic reactions. As no system of collecting data centrally exists in Britain failures of the transfusion process are not documented. In contrast, in the United States the Food and Drug Administration requires all establishments that are registered to process blood to report all errors and deaths associated with transfusion. We aimed to investigate the incidence of recognised transfusion errors in Britain in 1990 and 1991 and the cause and clinical outcome of these errors.

Methods and results

A short questionnaire about errors in blood transfusion procedures and the outcome of these errors was sent to the 400 hospital haematology laboratories in Britain in August 1992. In all, 245 (61%) laboratories responded: these supplied 3·3 million red cell units for transfusion (about three quarters of all the red cell and whole blood units collected annually in Britain). A third of responding laboratories reported incidents in which patients received the wrong blood. The table shows the results of the survey.

Comment

The error rates that we found are similar to those reported in studies from the United States.[2,3] Our data do not allow the calculation of error rates per patient transfused, which must be substantially higher than the rates in the table since most patients receive several units of blood. Several respondents indicated that multiple errors had contributed to the wrong blood being transfused; similar findings have been reported elsewhere.[4]

Twenty respondents reported (without having been asked in the questionnaire) 100 incidents in which the wrong blood sample was submitted in the compatibility tube and the error was detected in the laboratory because of a previous blood sample on the same patient. On the basis of this information and comments volunteered by other respondents, we estimate that the incidence of wrong blood being submitted in tubes is about 1/6000 red cell units issued.

Only a third of unmatched transfusions are incompatible with ABO blood groups; of these, only about a tenth are associated with a fatal outcome.[4] We should not, however, be complacent as these figures emphasise that data on mortality and morbidity, even if complete, can give only a substantial underestimate of the incidence of important failures in the transfusion process.

The data available are inadequate to determine the true incidence of errors in transfusion. All the errors found in this survey were reported by only a third of the responding laboratories; it would be surprising if the remaining laboratories had experienced no errors over two years.

We propose several ways of improving the quality and safety of the blood transfusion process in Britain. Firstly, a national system should exist for reporting critical transfusion incidents, especially those in which the wrong blood is transfused and "near misses." Regular reports to transfusion laboratories and hospital transfusion committees could be incorporated in the national external quality assurance scheme.

Results of questionnaire about errors in blood transfusion sent to 400 hospital haematology departments, of which 245 responded

	Wrong blood was transfused
Source of information (No of laboratories):	
Memory	126
Memory and written records	87
Written records	11
Not known	21
No of incidents (incidence per units supplied for transfusion)	
Total	111* (1/29 000)
Cause or place of error:	
Wrong blood in tube	23
Laboratory	6
Ward or theatre staff checked or transfused wrong blood	82
Outcome of error:	
Death	6 (1/550 000)
Morbidity	12 (1/275 000)
No adverse effect	93 (1/36 000)

*These were reported by 79 laboratories.

Secondly, all hospitals should establish clear and coordinated managerial responsibility for the transfusion process. Thirdly, transfusion laboratories should be required to have a procedure for recording all transfusion errors and the corrective action taken and to report regularly to the proposed national scheme. Fourthly, statistics on the number of blood units transfused and the number of patients who receive transfusions should be collected and reported nationally to provide denominators for any reporting scheme. Finally, pilot projects should be set up to identify and report cost effective ways of improving the safety of the clinical transfusion process.

We thank the organisers of the national external quality assurance scheme in haematology, which distributed the questionnaire and the final report to all hospital transfusion laboratories in Britain.

1 Roberts B, ed. *Standard haematology practice.* Oxford: Blackwell Scientific, 1991.
2 Sazama K. Reports of 355 transfusion-associated deaths: 1976 through 1985. *Transfusion* 1990;30:583-90.
3 Linden JV, Paul B, Dressler KP. A report of 104 transfusion errors in New York State. *Transfusion* 1992;32:601-6.
4 Murphy WG, McClelland DBL. Deceptively low morbidity from failure to practise safe blood transfusion: an analysis of serious blood transfusion errors. *Vox Sang* 1989;57:59-62.

(Accepted 17 February 1994)

Pneumocystis carinii in bronchoalveolar lavage fluid and bronchial washings

Simon P Matusiewicz, Ronald J Fergusson, Andrew P Greening, Graham K Crompton, Sheila M Burns

Respiratory Unit, Western General Hospital, Edinburgh EH4 2XU
Simon P Matusiewicz, *registrar*
Ronald J Fergusson, *senior registrar*
Andrew P Greening, *consultant physician*
Graham K Crompton, *consultant physician*

Regional Virus Laboratory, City Hospital, Edinburgh EH10 5SB
Sheila M Burns, *consultant virologist*

Correspondence to: Dr Matusiewicz.

BMJ 1994;308:1206-7

Antibodies to *Pneumocystis carinii* develop in early childhood,[1] although no associated illness has been identified. The classic concept is that *P carinii* pneumonia in immunocompromised patients represents a reactivation of dormant childhood infection. Studies with DNA amplification, however, have failed to detect pneumocystis in sputum[2] or specimens of lung tissue taken at necropsy[3] from immunocompetent patients. Reinfection rather than reactivation may therefore be more relevant. To see whether *P carinii* exists as a commensal organism of the lower respiratory tract we examined bronchoalveolar lavage fluid and bronchial washings in apparently immunocompetent patients undergoing routine diagnostic (or research) bronchoscopy by using an immunofluorescent antibody test.

Subjects, methods, and results

We studied 220 patients with various clinical diagnoses (table). Patients were considered to have no active disease if investigations, including bronchoscopy, failed to show any lung disease. Patients with known HIV infection or suspected pneumocystis pneumonia or those taking immunosuppressive drugs were excluded. In all, 116 patients had bronchoalveolar lavage, specimens being obtained in aliquots of 10 ml from 240 ml lavages (8×30 ml) of the lingula or middle lobe; 104 patients had bronchial washing, saline being introduced into and aspirated from the lung with disease diagnosed radiologically or seen to be abnormal at bronchoscopy. All specimens were examined for pneumocystis by a virologist (SB) without access to clinical data.

Details of 220 patients examined for presence of Pneumocystis carinii in bronchial washing or bronchoalveolar lavage specimens

	Bronchial washings	Bronchoalveolar lavage specimens
No of patients	104	116
Mean (range) age (years)	65 (22-83)	59 (24-84)
Diagnoses:		
No active disease	18	22
Asthma or chronic obstructive lung disease	6	12
Bacterial infection	10	8
Interstitial lung disease	3	13
Lung cancer*	67	61

*Cell types: squamous, 37; adenocarcinoma, 10; large cell, 16; small cell, 27; bronchiolo-alveolar, 1; carcinoid adenoma, 2; lymphoma, 1; metastatic, 12; histology unknown, 22.

Cytospin preparations of samples were fixed (50% methanol-acetone) and stained with murine monoclonal antibodies labelled with fluorescein that react with cysts and trophozoites (Genetic Systems Corporation, SYVA). Samples were considered positive if cysts or trophozoites were seen in three or more high power fields.

Of 220 specimens examined, only five (three lavage specimens and two washings) were positive for *P carinii* by the fluorescent antibody test. The five positive samples all had cysts and trophozoites in more than three high power fields. All were from patients with small cell lung cancer. No cysts or trophozoites were seen in any fields of samples defined as negative. No patients were thought to have clinical or radiological evidence of *P carinii* pneumonia at the time of bronchoscopy. Two positive specimens were available for staining by an alternative less sensitive method (toluidine blue O), and both were positive. Four of the five patients were clinically well. Two patients had evidence of macroscopic extrahemithoracic disease at presentation. Two patients had additional causes for impaired immunity—namely, previous cytotoxic treatment and ectopic secretion of andrenocorticotrophic hormone.

Comment

We were unable to detect *P carinii* by fluorescent antibody testing of bronchoalveolar lavage fluid or bronchial washings in 215 of the 220 patients studied and therefore consider that it may not be a commensal organism of the lower respiratory tract. Although the antibody test requires interpretation by a skilled pathologist, it is more sensitive and specific than silver staining. Theoretically, DNA amplification techniques are able to detect one or two cells but have failed to detect organisms in sputum[2] or postmortem samples of lung[3] in normal subjects. Not all studies, however, have shown any increase in diagnostic sensitivity of DNA amplification over fluorescent antibody testing in bronchoalveolar lavage samples.[4] Our findings support the concept that reinfection rather than reactivation leads to disease in susceptible patients. No environmental source has been identified, although infection rates show a seasonal variation. Infection was first identified in debilitated young and elderly subjects. These groups, in addition to patients infected with HIV, may act as a reservoir for pneumocystis.

Subclinical *P carinii* infection was found in five out of 27 patients with histologically proved small cell lung cancer. This may reflect a local defect in immune fuction associated with small cell lung cancer, which may have prognostic implications. Previous colonisation with pneumocystis may be clinically relevant for patients who subsequently receive cancer chemotherapy as pneumocystis pneumonia is a recognised