samples from various laboratories in the south east that used a commercial latex agglutination assay. These were referred to us for confirmatory testing by indirect enzyme linked immunosorbent assay (ELISA)²; 11 gave discrepant results (table). If equivocal results are treated as negative then seven patients (four with negative and three equivocal results in the latex assay) would have been wrongly considered to be negative for the antibody if confirmation had not been sought (a false negative rate of 7/32 (22%). Only five serum samples that yielded a positive result in the latex assay were referred to us, and these were all positive on ELISA.

Comparison of results obtained with latex test and ELISA for antibodies for varicella zoster virus

	Result of ELISA			Total
	Negative	Equivocal*	Positive	
Result of latex test:				
Negative	16	1	4	21
Equivocal	3	0	3	6
Positive	0	0	5	5
Total	19	1	12	32

*Equivalent results of ELISA should be regarded as negative for clinical purposes.

A working party of the Public Health Laboratory Service is currently assessing commercial assays to detect IgG antibody to varicella zoster virus. Although latex agglutination has compared well with immunofluorescence in the United States,³ we recommend that, until the results of the working party's study are available, all serum samples that yield negative results on latex testing should be retested with more sensitive assays. This will save unnecessary use of zoster immunoglobulin and acyclovir in the event of exposure. Even in urgent cases—that is, those in which testing is done after exposure—there is usually sufficient time to refer serum samples to laboratories that can offer rapid confirmatory assays since zoster immunoglobulin can be given up to 10 days after exposure,⁴ not 72 hours as stated by Rice and colleagues.

We question the acceptability of a 1-2% false positive rate when better tests are available. A false positive result may have serious consequences for an immunocompromised patient. Health care workers are also a large group screened for immunity to chickenpox; unexpected chickenpox in a health care worker can cause considerable problems of infection control.⁵

JUSTIN BENDIG
Consultant microbiologist
VALERIE MEURISSE
SHARON CHAMBERS

Public Health Laboratory,
West Park Hospital,
Epsom,
Surrey KT19 8PB

- 1 Rice P, Simmons K, Carr R, Banatvala J. Near fatal chickenpox during prednisolone treatment. *BMJ* 1994;309:1069-70. (22 October.)
- 2 Patou G, Midgeley P, Meurisse EV, Feldman RG. Immunoglobulin prophylaxis for infants exposed to varicella in a neonatal unit. *J Infect* 1990;20:207-13.
- 3 Klevjer-Anderson P, Anderson LW. Comparison of a new latex-agglutination assay with indirect immunofluorescence to detect varicella-zoster antibodies. *Diagn Microbiol Infect Dis* 1993;17:247-9.
- 4 Department of Health, Welsh Office, Scottish Office Home and Health Department, Department of Health and Social Services (Northern Ireland). *Immunisation against infectious disease*. London: HMSO, 1992.
- 5 Gurevich I, Jensen L, Kalter R, Cunha BA. Chickenpox in apparently "immune" hospital workers. *Infection Control* 1990; 11:510-1.

Authors' reply

EDITOR,—Paul Buss and Isobel Burnett express concern about our recommendation that varicella-zoster immunoglobulin should be given to non-

immune patients exposed to chickenpox who have received systemic steroids within the previous three months, a recommendation made by the Committee on Safety of Medicines earlier last year.¹ Depression of cell mediated immune responses occurs within four to six hours of administration of steroids. Therefore a short course of steroids given within the incubation period of chickenpox might impair host responses.² Even if short courses of steroids (less than five days) are given during the incubation period of chickenpox, severe and sometimes fatal infection occurs more commonly.³

We are therefore reluctant to alter our recommendations, although we accept Buss's and Burnett's point that the recommendations by the Committee on Safety of Medicines are likely to have considerable implications for supplies of varicella-zoster immunoglobulin. Although a history of chickenpox or detection of antibody will allow prophylaxis to be targeted more effectively, augmentation of these supplies should be considered. Blood donors are altruistic and could be reminded to donate blood if they have recently had chickenpox or shingles, thereby increasing supplies of varicella-zoster immunoglobulin. Alternatively, if the varicella vaccine now under review by the Food and Drug Administration in the United States was licensed in Britain the incidence of chickenpox would decline.⁴ This vaccine would also protect susceptible health care workers, who might otherwise transmit infection to others, including those receiving corticosteroids.

Donna Ellender and colleagues state that we failed to emphasise that patients who have had chickenpox are not at risk and therefore do not require prophylaxis with varicella-zoster immunoglobulin. We not only stated that a history of chickenpox should be ascertained, as this correlates reliably with past infection and immunity, but also emphasised this by stating that patients shown to be seronegative must be told that they are at risk of serious complications of chickenpox. This point was reiterated in the lesson. The Medicines Control Agency and Department of Health propose to issue a warning regarding chickenpox and other infections in a new patient held steroid card; serious complications of chickenpox have been known for a long time, and the recommendations are long overdue.

Cost-benefit analyses seem to have convinced the American Academy of Pediatrics and the Advisory Committee for Immunisation Practices to recommend vaccination of susceptible children, adolescents, and adults if the vaccine is licensed.⁵ This would overcome the increased morbidity and mortality associated with chickenpox among children receiving only modest doses of corticosteroids for asthma.

PHILIP S RICE
Senior registrar in virology

Guy's and St Thomas's Hospital Trust,
St Thomas's Hospital,
London SE1 7EH

JANGU E BANATVALA
Professor of clinical virology

ROBERT CARR
Senior lecturer in haematology

KATY SIMMONS
Lecturer in haematology

United Medical and Dental Schools of
Guy's and St Thomas's Hospitals,
St Thomas's Campus,
London SE1 7EH

- 1 Committee on Safety of Medicines and Medicines Control Agency. Severe chickenpox associated with systemic corticosteroids. *Current problems in pharmacovigilance*. 1994;20:1-2.
- 2 Fauci AS, Dale DC, Balow JE. Glucocorticosteroid therapy: mechanisms of action and clinical considerations. *Ann Intern Med* 1976;84:304-15.
- 3 Dowell SF, Bresee JS. Severe varicella associated with steroid use. *Pediatrics* 1993;92:223-8.
- 4 Plotkin SA. Vaccines for varicella-zoster virus and cytomegalovirus: recent progress. *Science* 1994;265:1383-5.
- 5 Lieu TA, Black SB, Rieser N, Ray P, Lewis EM, Shinefield HR. The cost of childhood chickenpox: parents' perspective. *Pediatr Infect Dis J* 1994;13:173-7.

Managing ulceration of the breast

EDITOR,—The article on the management of breast infection includes, under the heading "skin associated infection," a photograph of a previously irradiated breast showing longstanding telangiectasia within the radiotherapy field and a central area of ulceration of the skin.¹ Although this is described as cellulitis, the features are typically those of cutaneous radionecrosis,² which may be further complicated by an underlying osteoradionecrosis. We are concerned that the article is misleading as management with antibiotics and dressings would rarely produce a stable wound. We suggest that consideration should therefore be given to excision of the area affected and reconstruction with a local or distant flap.³

CHRIS STONE
Senior house officer
KEN DUNN
Senior registrar

Department of Plastic and Reconstructive Surgery,
Withington Hospital,
Manchester M20 8LR

- 1 Dixon JM. Breast infection. *BMJ* 1994;309:946-9. (8 October.)
- 2 Lipsztein R, Dalton JF, Bloomer WD. Sequelae of breast irradiation. *JAMA* 1985;253:3582-4.
- 3 Slavin SA, Love SM, Sadowsky NL. Reconstruction of the radiated partial mastectomy defect with autogenous tissues. *Plast Reconstr Surg* 1992;90:854-65.

Author's reply

EDITOR,—The example of cellulitis after radiotherapy in my article (reproduced in the figure (top)) was from a patient who in 1983 was treated for a cancer of the right breast by mastectomy, immediate reconstruction with a subpectoral prosthesis, and postoperative radiotherapy. In 1993 she presented with three areas of ulceration over the implant; the prosthesis was protruding through one of these areas and was removed in the outpatient clinic. The figure (top) shows cellulitis affecting the chest wall after the prosthesis was removed. Treatment with co-amoxiclav produced resolution of the cellulitis within two weeks (figure (bottom)).

Culture of material from the ulcerated areas yielded *Staphylococcus aureus*, which confirmed the clinical impression that infection was present. Although excision of the damaged area was

Cellulitis of right breast before (top) and after (bottom) a course of co-amoxiclav. Note resolution of erythema. Areas of ulceration healed after resolution of infection