

Repeatedly reactive specimens were confirmed in second-generation recombinant immunoblot assays (RIBA, Ortho Diagnostics). Discordant results between serum and urine led to retesting.

18 serum samples (22%) and their urine pairs were repeatedly reactive by ELISA. Mean OD/CO ratios were 5.14 (SD 1.21) and 4.24 (1.53), respectively. There were no discrepancies between serum and urine pairs by ELISA. 16 serum and 15 urine specimens were confirmed positive by RIBA. 2 of the 18 ELISA-reactive serum samples and 3 of the urine samples were indeterminate by RIBA. Only 1 of these urine samples had an indeterminate result with a confirmed positive in the serum. This sample was reactive (++) only to c33c antigen (with +/- to c100-3) by RIBA, while being reactive in serum to all four antigens (++). Efforts to demonstrate reactivity to the other bands in this urine samples by RIBA with a highly sensitive chemiluminescence substrate⁷ were unsuccessful. 38% (6/16) of the confirmed positive samples were from drug abusers.

The modified second-generation ELISA detected antibodies to HCV in urine with 100% sensitivity and specificity in this post-mortem group with a high frequency of HCV infection. Confirmation with RIBA resulted in 1 specimen of 16 (6%) being classified as indeterminate. The advantages of urine rather than serum specimens include cost savings, ease and safety of sampling, and the use of a convenient, non-invasive collection technique. Larger studies in high-risk and, especially, low-risk live populations are warranted.

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Carbon-adsorbed mitomycin prophylaxis against recurrence of gastric cancer

SIR,—Dr Hagiwara and colleagues (March 14, p 629) report the efficacy of intraperitoneal carbon-adsorbed mitomycin (M-CH) in patients resected for gastric cancer. We are concerned about the mechanism of action of prophylaxis with M-CH and the population of cancer patients to which these results can be extended.

Since the probability of survival at 3 years in the M-CH group is 68.6%, 16 of 24 patients would be expected to survive. If we accept that 6 of the 24 have been cured because of surgical treatment alone (26.9% of survivors in the control group), survival of the remaining 10 patients may be attributed to the effect of M-CH. This means a cure rate due to M-CH of 56% (10/[24-6]). However, according to Takahashi et al,¹ the efficacy of M-CH in patients with peritoneal carcinomatosis is only 50%, and this would imply that in these 18 patients peritoneal carcinomatosis was the sole cause of failure.

To our knowledge, however, these high values of peritoneal carcinomatosis are not supported by the few studies on systematic second-look operations or necropsy investigations on postoperative patients, the only studies that can show the true prevalence of the single patterns of recurrence of the disease. Gunderson and Sosin² have reported, in a planned reoperation series of cancer grade S₂ (definite invasion of serosa) to S₃ (invasion of contiguous structures), and node-positive, that peritoneal seeding alone accounted for only 2% of the failure rate (although it was a

component in 40% of the overall failure rate). Noda et al³ estimated that peritoneal dissemination was the prominent cause of death in 36.9% of patients, but they did not record exactly the frequency of the single patterns of recurrence. Papachristou and Fortner⁴ in a necropsy study of radically resected patients who died because of early postoperative complications, found a prevalence of distant visceral metastases of 58.8%, and did not mention any case of peritoneal seeding.

Other studies are less reliable because they included patients who had not been operated on or were in a very late stage of disease,^{5,6} or the site of failure was not examined alone or as a component of the overall failure rate.^{3,7} It is impressive, however, that in some of these series 70% non-peritoneal metastases have been reported.^{3,7}

We think that when results of a trial conflict with those of others or are unexpected, this would mainly depend on patient selection criteria. Correct selection of patients was probably a real difficulty in this trial since the degree of infiltration of the serosal surface or of the neighbouring organs, a prerequisite for randomisation, cannot be assessed intraoperatively,⁸ whereas randomisation had obviously to be done at operation. Moreover, a mean of only 1.1 patients were randomised per month (less than 15% of patients operated by the same institution according to a previous report¹), and, finally, the survival in controls is much lower than that reported (52.7%) by Koga et al⁹ in similar patients having much the same treatment.

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Latex dummies as allergens

SIR,—Latex allergy is well known in adults, causing contact urticaria, eczema, asthma, and anaphylaxis.¹ We report three infants with latex allergy caused by dummies (comforters).

Three boys, aged 0.7, 1.0, and 2.7 years, were admitted for severe atopic eczema. All had a positive family history of atopy, and all had food allergy (cows' milk, cereals, and other). Despite an elimination diet, eczema persisted. All used dummies. Thus, they were assessed by skin prick test (SPT) for latex. The antigen was prepared from surgical gloves (Exona, Semperit, Austria).² Histamine 10 mg/ml was used as a positive control and 50% glycerol as a negative control. Latex-specific IgE was measured with the Pharmacia Cap-system.

The first boy had positive SPT (4/3, diameters of the wheals in mm caused by latex/histamine) and positive radio allergosorbent test (RAST), 1.5 IU/l. The second boy had negative SPT (0/5) but positive RAST, 1.2 IU/l. The third boy also had negative SPT (0/6) but positive RAST, 3.5 IU/l; he underwent a provocation test. A finger of a latex glove (Triflex) was kept on a moistured finger of this patient and a finger of a vinyl glove on the same finger of the opposite hand. After 11 min, flush and urticaria developed in the finger exposed to latex. After stopping the use of latex dummies, all patients improved.

The presence of latex allergens in a dummy made of natural gum (Infra, "special safety dummy", Nordisk Forbindstof, Denmark) and in a silicone teat for bottles (Esska, Sweden) was analysed in an immunospot system (modified dot blot). Extracts from the dummies, and also from latex gloves and centrifuged rubber tree sap as controls, were applied to nitrocellulose sheets. One sheet was incubated with a pool of sera from latex-RAST-positive patients. A serum with a negative latex-RAST but positive cereal RAST was used as control. After washing, the plates were incubated with radioactive anti-IgE (Pharmacia) before autoradiography. IgE antibodies from the latex-RAST-positive serum were bound to the latex dummy, but not to the silicone teat. IgE was also bound to the control extracts (glove and sap). The control serum did not show any IgE binding. There are reports of latex allergy in children exposed to balloons.³ Children with congenital urological abnormalities have an increased risk of intraoperative anaphylaxis, probably due to latex allergy from frequent exposures to rubber catheters.⁴ Because babies' dummies contain natural latex, exposure to natural latex allergens is very common. Our findings show that sensitisation to latex can occur even in infancy. In eczema, urticaria, or anaphylaxis in babies, latex allergy should be borne in mind. Gloves, catheters, and other latex products could cause anaphylaxis during surgical procedures. Thus, tests for latex allergy might be recommended for surgical patients with allergy.

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Latex allergy and repeated graft rejections

SIR,—Immediate-type IgE-mediated allergy to latex can cause a wide range of reactions from localised contact urticaria to systemic anaphylaxis.^{1,2} We present a patient in whom contact with latex during surgical procedures was probably the reason of repeated rejections of autologous grafts. In a 15-year-old boy with a combined lip-jaw-palate-cleft, attempts at reconstructive surgery at age 8, 9, and 11 had all ended with rejection of transplant. A reason such as bacterial infection could not be found, despite investigations including histopathological studies of rejected tissue. Before a planned fourth operation, the boy was referred because of face swelling that had occasionally developed after dental procedures and had persisted for several hours. This reaction had been known about for more than 8 years. The swellings were not associated with the use of a particular local anaesthetic.

There was a history of atopy (hay fever since age 5, bronchial asthma at 5-10 years). Prick testing with common inhalant allergens yielded positive reactions to cat epithelium and housedust mite; specific IgE antibodies could also be demonstrated by radioallergosorbent test (RAST). Total serum IgE was 300 kU/l (normal 100 kU/l). Prick testing with local anaesthetics and patch testing with contact allergens were negative. Prick tests with solutions of natural brown and white latex were strongly positive (such reactions were not found in controls). The swelling resulting from latex skin prick testing persisted for more than 6 h. Application of a piece (1 cm²) of a latex surgical glove on the inner forearm produced urticarial swelling within 5 min. High inhibition of specific IgE antibodies against natural latex with commercial latex discs could be demonstrated (RAST class 4).

An IgE-mediated allergy to latex was diagnosed. All previous reconstructive operations were done with latex-containing surgical gloves. Because this might have been the cause of graft rejections, it was proposed that for the fourth attempt of reconstruction, a latex-free procedure should be used. This was done, and the grafts were accepted without complications for the first time.

It is common in surgery that post-traumatic oedema must resolve before reconstruction can begin. This especially applies to grafts,

which are at high risk for rejection when applied while oedema is present or increasing. It is probable that in our case, localised urticarial reaction resulting from contact with latex gloves was responsible for repeated graft rejections. In cases of transplant rejection of unknown cause, allergy should be considered.

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Laparoscopic pericardial fenestration for malignant pericardial effusion

SIR,—We have used laparoscopy to treat malignant pericardial tamponade palliatively. This approach has not, to our knowledge, been described previously. A 69-year-old woman was admitted with a 3 week history of rapidly worsening dyspnoea. In 1989 she had undergone a left upper lobectomy for a squamous bronchial carcinoma and local and mediastinal recurrence had occurred. On admission she had cardiac tamponade (hypotension, raised jugular venous pressure [JVP], pericardial rub, peripheral oedema). Kussmaul's sign was positive and a large pericardial effusion was confirmed ultrasonically. Under ultrasound guidance, a pericardial drain was inserted via the perixiphoid approach. 300 ml bloodstained fluid was aspirated after which the patient became normotensive. However, the raised JVP and peripheral oedema remained and she continued to be dyspnoeic after even slight exertion. Cytological examination of the pericardial fluid revealed no malignant cells. To avoid future recurrence of the pericardial effusion, the more definitive procedure of laparoscopic pericardial fenestration was done.

Under general anaesthesia, a pneumoperitoneum was introduced via a subumbilical incision, through which laparoscopy was done. Laparoscopy trocars (11 mm and 5 mm) were placed to the left of the abdomen for instrumentation. No intra-abdominal evidence of malignancy was seen. However, the position of the pericardial attachment to the central tendon of the diaphragm was easily visualised, over the left lobe of the liver, by presence of cardiac pulsation and bulging caused by pericardial fluid. A 3 cm incision made into this area with a diathermy hook resulted in a gush of bloodstained fluid into the peritoneal cavity. This incision provided decompression of the tamponade and prevention of fluid reaccumulation. Through the pericardial window, secondary deposits were visible on both the visceral pericardium and the myocardium. The procedure lasted 30 min, during which the patient's vital signs remained stable. She made a rapid and largely uneventful recovery. On the evening of the operation, atrial fibrillation developed but settled quickly on digoxin. By 48 h she said she felt more comfortable and was much less dyspnoeic, had a normal JVP, and very little peripheral oedema. She had no problems from the laparoscopic procedure, was pain-free, and was sufficiently mobile to be discharged home.

Malignant effusions bring unpleasant symptoms that may be difficult to palliate. Percutaneous drainage allows short-term relief of tamponade but effusions may reaccumulate. Pericardial fenestration offers a definitive solution but its use in patients with malignant disease has been limited by the need for laparotomy, which is often unacceptable in such patients. Moreover, the time to full recovery from a laparotomy erodes the already short-life expectancy of these patients and seriously reduces the palliative benefits of the procedure. Fenestration via laparoscopy gives definitive pericardial decompression and symptom relief with a quick recovery. The palliation achieved is excellent, and we suggest that laparoscopic fenestration is the treatment of choice.

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