

Paradoxical outcome after use of hyaluronate barrier to prevent intra-abdominal adhesions

J P Trickett FRCS R M Rainsbury MS FRCS
R Green MB MRCPATH

J R Soc Med 2001;94:183–184

Intra-abdominal adhesions are the commonest cause of small-bowel obstruction and secondary female infertility. At operation, strategies to prevent adhesions include the use of absorbable barriers. Seprafilm (Genzyme BV, Naarden, Netherlands) is a sterile translucent membrane comprising sodium hyaluronate and carboxymethylcellulose; it temporarily separates potentially adherent surfaces, turning to a gel within 24 h and being cleared from the abdominal cavity in 7 days. Sepracoa (Genzyme BV) is a solution of hyaluronidase in phosphate buffered saline, reabsorbed from the body cavity and excreted in 5 days. In animal models and patients, these agents have reduced adhesion formation by about 44% without any apparent excess of adverse events^{1–3}.

CASE HISTORY

A woman aged 71 developed small-bowel obstruction which resolved with conservative management. She had undergone four previous laparotomies—for hysterectomy, for salpingo-oophorectomy and for adhesiolysis (two). She continued to experience episodic abdominal pain but contrast studies showed no evidence of obstruction. At laparotomy, performed four months later to identify the cause of her recurrent symptoms, the entire small bowel was found to be affected by multiple adhesions, with partial obstruction. The adhesions were carefully divided throughout the small bowel, polyglactin (Vicryl) ligatures being used for haemostasis. Sepracoa was instilled into the abdominal cavity in accordance with the manufacturer's instructions. One sheet of Seprafilm was placed under the midline incision before closure with polydioxanone (PDS) sutures. Salbutamol was required at extubation for bronchospasm.

Normal bowel sounds returned on the third post-operative day. On day 14 a barium meal and follow-through examination was performed because of high-volume nasogastric aspirates. This showed a distended duodenum

and proximal jejunum, but contrast passed through an undilated distal small bowel in 80 min.

A second laparotomy was performed 21 days later because of unresolved high intestinal obstruction. This revealed a dense, thick, glue-like mass involving 95% of the small bowel and part of the transverse colon, anchoring the abdominal contents to the anterior abdominal wall. An attempt to release the small-bowel loops was abandoned because of the extent and density of the encasing mass. 90% of the small bowel was resected and a jejunocolic anastomosis fashioned in two layers of chromic catgut. During the dissection a serosal tear was made in the mid transverse colon which was repaired by suturing it to the serosal surface of the ascending colon. The patient's condition deteriorated three days later with a faeculent bloodstained wound discharge, signs of peritonitis and renal failure. Further surgical intervention was deemed inappropriate and she died the following day.

At necropsy the jejunocolic anastomosis was found to be intact, but closure of the defect in the transverse colon had broken down, causing faeculent peritonitis. Examination of the resected specimen showed multiple loops of small bowel matted together within an extensive mass of densely fibrotic tissue (Figure 1). On microscopy the serosal surface was congested and encased in adherent fat and fibrous tissue containing numerous foreign-body-type giant-cell granulomata. Birefringent foreign material was identified in the giant cells, appearing as small particles and short fibres.

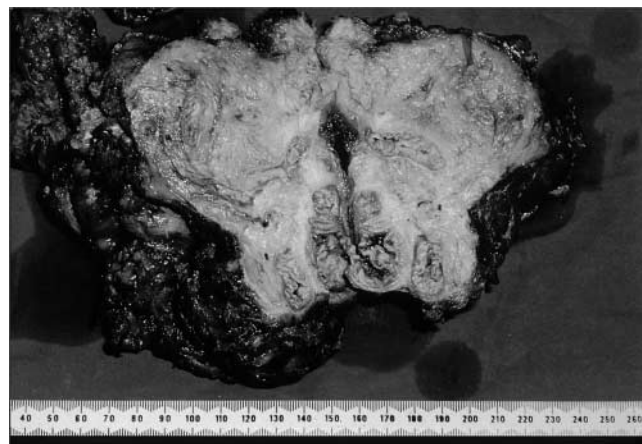


Figure 1 Resection specimen showing fibrotic mass

COMMENT

Foreign-body granulomata may be related to suture material¹, but the extent and severity of the changes observed in this case suggest the possibility of another cause. At the first laparotomy hyaluronidase was instilled throughout the abdominal cavity and as a film between the bowel and anterior abdominal wall. The only other materials used were a small number of Vicryl ligatures

and PDS sutures for closure. Reaction to suture material, however extreme, would be expected to occur in the immediate vicinity of the sutures, rather than cause the widespread changes seen in this case. The second laparotomy did not reveal any evidence of leakage or sepsis that could have caused such a diffuse reaction. The dense fibrotic reaction, containing foreign-body-type giant cell granulomata, was florid and universal affecting the whole serosal surface of the small bowel.

Concerned that these remarkable findings might represent an idiosyncratic response to Septrafilm and Sepracoat, the operating surgeon filed an adverse incident report with the Medical Devices Agency. Four similar cases of foreign-body-type granulomatous reaction following the use of a hyaluronate barrier had been reported to agencies in the USA and the UK. Three involved placement of the barrier following adhesiolysis, and the fourth occurred after repair of a paraduodenal hernia. All patients required subsequent surgery within 6–13 days for small-bowel obstruction, two undergoing extensive small-bowel resection. These five cases in total since marketing approval would represent an occurrence of less than 0.1% for Septrafilm and 0.01% for Sepracoat.

REFERENCES

- 1 Becker JM, Dayton MT, Fazio VW, *et al.* Prevention of post operative abdominal adhesions by a sodium hyaluronate based bioresorbable membrane: a prospective randomised double blind multicentre study. *J Am Coll Surg* 1966;**183**:297–306
- 2 Burns JW, Colt MJ, Burgees LS, Skinner KC. Pre-clinical evaluation of Septrafilm bioresorbable membrane. *Eur J Surg* 1997;**577**(Suppl.):40–8
- 3 Diamond MP. Reduction of post surgical adhesions by intraoperative pre coating with Sepracoat® (HAL-C) solution: a prospective randomised, blinded, placebo controlled multicentre study. *Fert Steril* 1998;**69**:1067–74

Computer game delusions

Rachel Forsyth MRCPsych Rachel Harland MRCPsych¹
Thomas Edwards MRCPsych²

J R Soc Med 2001;**94**:184–185

Patients with schizophrenia have described being controlled by and entwined with the Internet^{1,2}, and others have perceived themselves to be characters in a film, with life

Heathfields, 30 Denmark Road, Gloucester GL1 3HZ, UK; ¹University of Bristol Division of Psychiatry, 41 St Michael's Hill, Bristol BS2 8DZ; ²Psychopharmacology Unit, School of Medical Sciences, University of Bristol, Bristol BS8 1TD

Correspondence to: Dr Rachel Forsyth
E-mail: samnbunny@cableinet.co.uk

played out for the cameras³. We have found no report of a computer game as the basis for a delusion.

CASE HISTORY

A young man was admitted from prison to a psychiatric facility after reports that he had been acting in a bizarre manner. He had been arrested for stealing motor vehicles and assaults with weapons. At interview he was found to be experiencing the delusion that he was a player inside a computer game (adult-certificate game, widely available) in which points are scored for stealing cars, killing assailants and avoiding police vehicles. Psychotic symptoms had emerged slowly over two years. His family had noticed him becoming increasingly withdrawn and isolated from social activities. He developed delusions that strangers were planning to kill him and also experienced auditory hallucinations, constantly hearing an abusive and derogatory voice. Previously a computer enthusiast, he began to play computer games incessantly. He felt that the games were communicating with him via the headphones. In a complex delusional system he came to believe he was inside one of these games and had to steal a car to start scoring points. He broke into a car and drove off at speed, believing he had 'invulnerable' fuel and so could not run out of petrol. To gain points he chose to steal increasingly powerful vehicles, threatening and assaulting the owners with weapons. Later he said he would have had no regrets if he had killed someone, since this would have increased his score.

After arrest and while in prison he continued to believe he was in the game, despite initial medication. When he was admitted to hospital six weeks later, part of ward management was to deny him access to computer games. Nothing abnormal was found on physical examination, blood investigations, drug screen, electroencephalography or a computed tomographic brain scan. Paranoid schizophrenia was diagnosed and he responded well to further treatment with antipsychotic medication.

COMMENT

Schizophrenia is characterized by abnormalities of thinking, perception and emotion⁴. Paranoid schizophrenia is the commonest subtype and initial symptoms typically include bizarre delusions, auditory hallucinations and interference with thinking. In the chronic form, blunting of emotion, social withdrawal and avolition can become more obvious. Age at onset is usually the late twenties or early thirties with equal distributions between the sexes. Current cultural beliefs have been consistently found to determine the content of delusions in schizophrenia⁵. Often the background is religious ('I am God') but a common secular delusion is that of being pursued by secret agents.

In this report we are not suggesting that computer games can be the cause of psychosis; but it does seem likely that, with the growing use of computers for relaxation, game scenarios will be incorporated increasingly into delusional systems. A worrying aspect is that, in many of these games, points are scored for acting violently or even killing. If the game is transposed into the real world by a delusional state, the risk of subsequent violence is high—particularly if violence is not perceived to be illegal or morally wrong.

REFERENCES

- 1 Catalano G, Catalano MC, Embi CS, Frankel RL. Delusions about the Internet. *South Med J* 1999;92:609–10
- 2 Tan S, Shea C, Kopala L. Paranoid schizophrenia with delusions regarding the internet. *J Psychiatry Neurosci* 1997;22:143
- 3 Tomison AR, Donovan WM. Dangerous delusions: the Hollywood phenomenon. *BMJ* 1988;153:404–5
- 4 World Health Organization. *ICD 10 Classification of Mental and Behavioural Disorders*. Geneva: WHO, 1992
- 5 Ahmed SH. Cultural influences on delusion. *Psychiatr Clin (Basel)* 1978; 11:1–9

Nipple excised and areola retained after total mastectomy (NEAT)

A B Gordon FRCS N Nasiri FRCPATH
G P H Gui MS FRCS N P M Sacks MS FRCS

J R Soc Med 2001;94:185–186

The nipple is part of the lactiferous system¹ whereas the areola is a form of pigmented skin, anatomically close but functionally different. For mastectomy, common surgical practice varies between complete excision and preservation of the nipple–areola complex. Two examples of these extremes are given, both giving unsatisfactory results, and a third case in which the differences between nipple and areola were exploited in the NEAT procedure—nipple excised and areola retained.

CASE HISTORIES

Case 1

A woman aged 51 had extensive microcalcification on her left mammogram. She was treated by total mastectomy and

subpectoral implant with complete preservation of the areola and nipple. Histological examination showed complete clearance of high-grade ductal carcinoma-in-situ (DCIS) with no evidence of tumour in the separately submitted subareolar tissue. All twelve axillary lymph nodes were free of tumour. One year later she developed left nipple discharge and there were adenocarcinoma cells on the smear. The nipple, excised alone, showed extensive high-grade DCIS of the comedo type with large areas of central necrosis, associated with calcification.

Case 2

A woman of 51 was referred with nodularity in the upper and outer quadrant of the right breast. On mammography there was widespread malignant microcalcification, and clinical examination showed carcinoma cells. A Patey mastectomy was performed with insertion of a subpectoral implant. Skin closure was tight and with adjuvant radiotherapy the aesthetic result was not ideal. On histological examination there was extensive high-grade DCIS with a comedo pattern, with several small foci of grade III invasive carcinoma, the largest 1 cm in diameter. The nipple and areola showed no intrinsic abnormality. Fourteen of nineteen axillary lymph nodes showed metastatic carcinoma.

Case 3

A woman aged 68 had a breast lump which proved to contain a duct papilloma with associated papillomatosis. Five years later a second right breast mass was removed, which contained a papilloma. Further biopsies from the right breast at eight and nine years also showed papillomatosis. At eleven years from the original presentation a benign duct papilloma was removed from the left breast. Two years after this, another right-sided papilloma was excised, and the surrounding breast tissue contained a 3 mm focus of high-grade DCIS with incomplete margins. The patient was counselled about the risk of breast cancer associated with bilateral papillomatosis. After considering the options, she opted for bilateral mastectomy with excision of the nipple but retention of the areola. The breasts were reconstructed with subpectoral bi-dimensional anatomical permanent expander implants (Figure 1).

COMMENT

Preservation of the entire nipple–areola complex has been reported safe after mastectomy³, but concern persists because of the presence of mammary ducts within the preserved nipple and areola⁴. The principle behind the NEAT procedure is that these tissues are functionally different; in particular, the accessory mammary glands in the areola are not connected by ducts with breast tissue².

Academic Surgery (Breast Unit), Royal Marsden NHS Trust, Fulham Road, London SW3 6JJ, UK

Correspondence to: Mr Angus B Gordon, FRCS. E-mail: ggui@cwcom.net



Figure 1 Bilateral mastectomy with NEAT and immediate reconstruction using submuscular implants

The technique is especially suitable for prophylactic mastectomy. Relative contraindications are deep-sited central tumours and extensive multifocal disease; absolute contraindications are Paget's disease, *peau d'orange*, clinical evidence of subdermal infiltration, and locally advanced breast cancer.

In a case of breast cancer, what is the likelihood that carcinoma is present or will develop in the retained areola? The risk is likely to be greatest in patients with large primary tumour size and retro-areolar location, multifocal disease and dermal invasion despite apparent clinical areola sparing⁵. The key to success is careful selection.

NEAT is a simple modification of an accepted mastectomy technique that facilitates tension-free skin closure for immediate breast reconstruction, in particular for implant breast reconstruction without a myocutaneous flap. In breast reconstruction including a myocutaneous flap, the presence of the pigmented areola, incorporated into the transposed flap or as part of the principal surgical incision, can improve the aesthetic result without compromising the principles of cancer surgery.

REFERENCES

- 1 Rosen PP, Tench W. Lobules in the nipple. Frequency and significance for breast cancer treatment. *Pathol Annu* 1985;20:317-22
- 2 Neville MC, Neifert MR. Lactation. *Physiology, Nutrition and Breastfeeding*. New York: Plenum, 1983: 23-47
- 3 Bishop CC, Singh S, Nash AG. Mastectomy and breast reconstruction preserving the nipple. *Ann R Coll Surg Engl* 1990;72:87-9
- 4 Schnitt SJ, Goldwyn RM, Slavin SA. Mammary ducts in the areola. Implications for patients undergoing reconstructive surgery of the breast. *Plast Reconstr Surg* 1993;92:1290-3
- 5 McCarty KS, Kesterson GHD, Barton TK, Seigler HF, Georgiade NG. Selection of patients for heterotopic implantation of the areola and nipple. *Surg Gynecol Obstet* 1980;150:545-7

Superficial thrombophlebitis followed by pulmonary embolism

Patrick Kesteven PhD FRCPATH
Brian Robinson BSc FIBMS

J R Soc Med 2001;94:186-187

Superficial thrombophlebitis is often thought of as a benign self-limiting disorder, warranting only symptomatic treatment with non-steroidal anti-inflammatory drugs. Lately, however, several reports have suggested an association with deep-vein thrombosis, with one study finding an unexpectedly high rate of pulmonary embolism in patients with saphenous vein thrombosis and no obvious deep-vein involvement¹.

CASE HISTORY

A student aged 23 was seen with a five-day history of left calf swelling which began after 6 hours of immobility at her computer. She gave no relevant medical history apart from the fact that she was awaiting varicose vein surgery. She was a non-smoker with a moderate alcohol intake (ten to twelve units per week) and had been on the combined oral contraceptive pill for the past 2 years for painful irregular periods. There was no history of recent travel. On further questioning she mentioned that her mother had had a deep-vein thrombosis post partum. The patient had not experienced haemoptysis, chest pain or shortness of breath and the findings on physical examination, electrocardiography and chest radiography were unremarkable. She was afebrile and normotensive with a slightly raised D-dimer level of 0.4 mg/L (normal 0.0-0.3). The left calf had a 2 cm greater circumference than the right and a thrombosis was palpable in the short saphenous vein with mild surrounding erythema and tenderness. An ultrasound scan of her left leg confirmed the presence of thrombus in the short saphenous vein but no thrombosis was seen in the deep venous system. She was discharged on non-steroidal anti-inflammatory medication, having been given a contact telephone number in case symptoms worsened.

Six days later the patient was admitted after a collapse at home and subsequent episodes of hyperventilation. She had been out dancing the previous night and had consumed about eight units of alcohol. For the past two days she had

Department of Haematology, Freeman Hospital, Newcastle upon Tyne NE7 7DN, UK

Correspondence to: Mr B Robinson

been increasingly short of breath, with pleuritic pain around the left shoulder but no haemoptysis, sputum production or cough. On systemic examination the only obvious abnormality was shortness of breath at rest (respiration 26 per minute). Her left calf was still swollen though the calf measurements had not changed. Arterial blood pH was 7.46, PCO_2 3.8 kPa and PO_2 10.9 kPa, other values being within normal limits. D-dimer was now 2.8 mg/L and a VQ scan revealed multiple pulmonary emboli. A subsequent ultrasound scan of her left leg showed that thrombus had extended into the popliteal and lower superficial femoral veins. The patient was given tinzaparin and started on warfarin. On anticoagulation therapy she recovered without further incident. A thrombophilia screen revealed no genetic cause for her venous thromboembolism.

COMMENT

Acute superficial thrombophlebitis is a common vascular disease which is usually expected to run a 'benign' clinical course². Treatment includes non-steroidal anti-inflammatory drugs, other analgesia and rest. An association with deep venous thrombosis, however, has been reported, with frequencies of 12–44%³, and there have been several reports of pulmonary embolism in thrombophlebitis. In one study, 43 of 50 patients with superficial thrombophlebitis of the great saphenous vein had pulmonary emboli². Seemingly, complications are more likely with superficial thrombophlebitis of the long saphenous vein rather than the short one⁴ though this is a contentious issue⁵. There are several reports of thrombus extending from the short saphenous vein into the deep-vein system^{5,6}.

Should patients with superficial thrombophlebitis of the short and long saphenous veins receive anticoagulants? This might be justifiable not only as a way to limit progression to deep-vein thrombosis and the hazard of pulmonary embolism but also in terms of symptom relief.

REFERENCES

- Verlato F, Zucchetta P, Prandoni P, *et al.* An unexpectedly high rate of pulmonary embolism in patients with superficial thrombophlebitis of the thigh. *J Vasc Surg* 1999;**30**:1113–15
- Markovic MD, Lotina SI, Davidovic LB, *et al.* Acute superficial thrombophlebitis: modern diagnosis and therapy. *Srp Arch Celok Lek* 1997;**125**:261–6
- Bounameaux H, Reber-Wasem MA. Superficial thrombophlebitis and deep vein thrombosis: a controversial association. *Arch Intern Med* 1997;**157**:1822–4
- Lutter KS, Rerr TM, Roedersheimer R, *et al.* Superficial thrombophlebitis diagnosed by Duplex scanning. *Surgery* 1991;**110**:42–6
- Jorgensen JO, Hanel KC, Morgan AM, Hunt JM. The incidence of deep vein thrombosis in patients with superficial thrombophlebitis of the lower limbs. *J Vasc Surg* 1993;**18**:70–3
- Blumenberg RM, Barton E, Gelfand ML, Skudder P, Brennan J. Occult deep vein thrombosis complicating superficial thrombophlebitis. *J Vasc Surg* 1998;**27**:338–43

Pulmonary embolism after intravenous immunoglobulin

C Alliot MD J P Rapin MD M Besson MD
F Bedjaoui MD D Messouak MD

J R Soc Med 2001;**94**:187–188

The best-known adverse effects of high-dose intravenous immunoglobulin (IVIg) are nephrotoxicity and anaphylactic reaction. Only a few cases of thrombosis have been reported.

CASE HISTORY

A woman aged 63 with a history of osteoporosis, hypertension, aspirin-induced gastritis and thrombophlebitis in the left leg 4 years ago was admitted to the hospital because of bleeding gums, spontaneous haematomas and severe thrombocytopenia (platelet count $2 \times 10^9/L$). A coagulation screen was normal, as was viral serology; screening for autoimmunity was negative; bone-marrow smears were normal. She was treated unsuccessfully with prednisone, the platelet count reaching $18 \times 10^9/L$ after one month. IVIg, 1 g/kg, was then infused progressively to a maximal flow of 3 mL/kg per hour, and three days later the platelet count had risen to $143 \times 10^9/L$. The patient was readmitted seven days after this infusion because of dyspnoea. Temperature was $38.1^\circ C$, blood pressure 150/80 mmHg, pulse 102/min, respirations 24. She was cyanosed but examination of the heart and lungs, including electrocardiogram and chest X-ray, revealed nothing abnormal. There was no evident thrombophlebitis. Arterial blood showed the following: pH 7.4, PO_2 6.9 kPa, PCO_2 3.7 kPa, HCO_3 21.3 mmol/L. Haemoglobin was 16 g/dL, platelets $227 \times 10^9/L$, serum protein 83 g/L, D-dimers >4 mg/L (normal <0.4). Prothrombin time, partial thromboplastin time and fibrinogen concentration were normal. Ultrasonography revealed thrombophlebitis in the right popliteal vein. A work-up for hypercoagulable state (including antithrombin III, protein C, protein S, heparin cofactor II, homocysteine, factor V Leiden mutation and antiphospholipid antibodies) was negative. A spiral computed tomographic scan of the chest revealed bilateral pulmonary embolism. She was treated initially with heparin

Department of Internal Medicine, General Hospital of Annemasse, BP 525, 74107 Annemasse Cedex, France

Correspondence to: Dr C Alliot, 8 rue du Chablais, 74100 Annemasse, France
E-mail: chiab@sante.hautesavoie.net

and was discharged on day 26 taking fluindione. Further episodes of thrombocytopenia proved resistant successively to disulone, vincristine and colchicine. The patient underwent splenectomy after four courses of IVIg, given on the same schedule as before but under anticoagulant cover. There was no further thrombotic event.

COMMENT

We have found thirteen previously reported cases of IVIg-related thrombosis, including myocardial infarction in five instances, stroke in four and spinal cord ischaemia in one¹. Deep venous thrombosis has been reported in only three instances—a woman of 52 with lymphocytic lymphoma complicated by immune thrombocytopenia, an immobilized patient treated for myasthenia gravis and a man aged 74 with renal carcinoma and immune neutropenia who died from pulmonary embolism^{1–3}. Perhaps the explanation for these events is that IVIg not only induces platelet activation but also increases plasma viscosity⁴.

REFERENCES

- 1 Go RS, Call TG. Deep venous thrombosis of the arm after intravenous immunoglobulin infusion: case report and literature review of intravenous immunoglobulin-related thrombotic complications. *Mayo Clin Proc* 2000;**75**:83–5
- 2 Brannagan TH III, Nagle KJ, Lange DJ, Rowland LP. Complications of intravenous immune globulin treatment in neurologic disease. *Neurology* 1996;**47**:674–7
- 3 Alliot C, Barrios M, Tabuteau S, Desablens B. Autoimmune cytopenias associated with malignancies and successfully treated with intravenous immune globulin. *Thérapie* 2000;**55**:371–4
- 4 Delakas MC. High-dose intravenous immunoglobulin and serum viscosity: risk of precipitating thromboembolic events. *Neurology* 1994;**44**:223–6

Dementia with a prosthetic aortic valve

D Dutta MB MRCP C E Ashton MB FRCP

J R Soc Med 2001;**94**:188–189

SECTION OF GERIATRICS AND GERONTOLOGY, 23 NOVEMBER 1999

To reduce the incidence of thromboembolism, a cloth-covered version of the Starr–Edwards aortic valve was introduced in 1968 (Figure 1)¹. A new complication then arose—systemic embolism due to cloth tears^{1–3}.

Department of Medicine for the Elderly, Worcester Royal Infirmary, Newton Branch, Worcester WR5 1JG, UK

Correspondence to: Dr C E Ashton
e-mail: dipankar.dutta@virgin.net

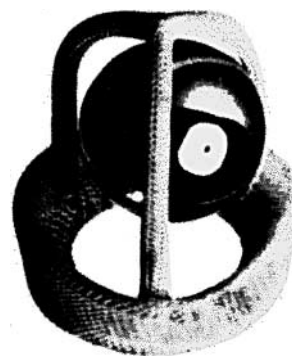


Figure 1 Cloth-covered Starr–Edwards model 2310/2320 aortic prosthesis used from 1968 to 1975. Reproduced from Ref. 1 by permission

CASE HISTORY

A woman aged 76 reported progressive memory loss over the past year. The onset had been insidious with initial loss of short-term memory followed by difficulties in social interaction and visual hallucinations. In 1971 she had had an aortic valve replacement with a cloth-covered Starr–Edwards prosthesis for rheumatic disease. There was no history of stroke or transient ischaemic attacks. On examination her abbreviated mental test score was 1/10; she had expressive dysphasia but no other focal neurodeficit. She was normotensive and in controlled atrial fibrillation. Prosthetic heart sounds were heard along with an ejection systolic murmur.

Blood tests were unremarkable. Her records showed that, since the valve replacement, she had almost always been adequately anticoagulated. Electrocardiography confirmed atrial fibrillation. Computed tomography of the brain showed a degree of cerebral atrophy. Transthoracic echocardiography showed subnormal left ventricular function with a dilated left ventricle and reduced septal movement. There were no thrombi or vegetations. On transoesophageal echocardiography the prosthetic valve appeared normal and no thrombus was seen in the left atrium. Magnetic resonance imaging was not possible because of her prosthetic valve. Cerebral perfusion scintigraphy and single-photon emission tomography showed a defect in the left frontoparietal cortex and thinning in the occipitoparietal perfusion consistent with vascular dementia. Several falls and infections punctuated her clinical course and she died suddenly in a nursing home.

At necropsy there were multiple areas of microcystic degeneration around the lateral ventricles and one larger (1 cm) area of infarction in the left cerebellar hemisphere. There were mild atherosclerotic changes in the aorta and carotid arteries. On examination of the Starr–Edwards valve *in situ*, the cloth covering the struts was seen to be frayed and broken down, hanging from the cage. There were no vegetations on the valve, and the atria and its

appendages were clear. Brain tissue was not examined microscopically.

COMMENT

Complications related to the ball-in-cage heart valves include infective endocarditis, haemolytic anaemia, systemic arterial embolization, thrombotic occlusion of the prosthesis and paraprosthetic leak². The one hazard unique to the cloth-covered prosthetic valve is wear of the cloth covering the struts, leading to systemic cloth fibre emboli¹⁻³ and haemolytic anaemia⁴.

To the best of our knowledge, there have been no previously reported cases of dementia associated with a damaged prosthetic heart valve. Though we did not look for microscopic evidence of material from the valve within the multiple infarcts, the absence of vegetations, of clots and of severe atherosclerosis makes the damaged prosthetic valve the most likely source of embolic material. Hypertension and diabetes being absent, small vessel disease causing multiple lacunar infarcts is unlikely.

In patients with cloth-covered prosthetic valves, cloth wear should be suspected as a cause of recurrent embolic events when anticoagulation has been adequate³. Exclusion of other sources of systemic emboli and infective endocarditis is necessary. Transthoracic and transoesopha-

geal echocardiography may be useful in detecting cloth tears, which have to be differentiated from vegetations and thrombi^{5,6}. Some patients with sequelae of cloth wear have benefited from replacement with porcine xenografts².

Acknowledgments We thank Dr Mike Davis for help with the patient and Dr P J S Dunn for conducting the necropsy.

REFERENCES

- 1 Starr A, Grunkemeier GL, Lambert LE, Thomas DR, Sugimura S, Lefrak EA. Aortic valve replacement: a ten year follow-up of non-cloth-covered vs cloth-covered caged ball prostheses. *Circulation* 1977; **56**(suppl 2):133-9
- 2 Shah A, Dolgin M, Tice DA, Trehan N. Complications due to cloth wear in cloth-covered Starr-Edwards aortic and mitral valve prosthesis and their management. *Am Heart J* 1978; **96**:407-14
- 3 Borouchow IB, Ramsey HW, Wheat MW Jr. Complications following destruction of the cloth covering of a Starr-Edwards aortic valve prosthesis. *J Thoracic Cardiovasc Surg* 1971; **62**:290-3
- 4 Feinberg MS, Sagie A, Freimark D, *et al.* Echocardiographic detection of severe prosthetic valvular cloth wear. *Am Heart J* 1996; **131**:1229-31
- 5 Shapira Y, Feinberg MS, Hirsch R, Nili M, Sagie A. Echocardiography can detect cloth cover tears in fully covered Starr-Edwards valves: a long term clinical and echocardiographic study. *Am Heart J* 1997; **134**:665-71