part was made out of the old operating theatre across the corridor. The cost of remodelling was about £500 000, but the authority saved £500 000 a year through closing the Wimbledon Hospital and got £1.5m from its sale.

Buildings as resources

The Medical Architecture Research Unit of the Polytechnic of North London, which is currently evaluating several nucleus hospitals, has extended the idea of treating buildings as resources in its studies on the use of space within hospitals.

MARU has always taken the line that hospitals should be seen not as static collections of rooms but as accommodating organisations. Indeed, one of its criticisms of early building notes was that they concentrated too much on room schedules instead of thinking about how an organisation worked and the need to generate spaces to house that activity. The idea is not new. The Nuffield report of 1955 on the function and design of hospitals pointed out that better timetabling in outpatient clinics would allow the waiting area to be much smaller.¹ But it is a lesson that still needs to be learnt.

Hospital provision tends to be calculated on the basis of norms—so many beds per 1000 population, so many surgical beds per theatre, and so on. In examining the use of operating theatres MARU discovered that the assumptions behind the norm of one theatre per 45 surgical beds were wrong and that many theatres were considerably underused. Policies on keeping a theatre free for emergencies and on routine maintenance had a large effect on the

overall use of the theatres, as did their location. One study showed, for example, that a hospital that was replacing eight scattered theatres by a central suite needed to build only seven theatres because they could be used much more effectively by being grouped together. The methods developed by MARU can be applied to other departments and will be included in DHSS guidance.

The drawback of using such methods, of course, is that they take more effort than simply applying a norm, and users need an incentive. The incentive can be only that there is a chance of saving money that can be spent on something else. And that in turn depends on the DHSS being rather more flexible than it is at present on interpreting cost allowances.

The NHS has taken a long time to get to grips with its estate. Only now are authorities taking a more sensitive approach to both new and existing hospitals, ironically at a time when expectations are much lower. During the boom years of the '60s and early '70s ambition was often compounded by ignorance, and a lot of things went wrong—as next week's article shows.

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Lesson of the Week

Insertion of intrauterine contraceptive device in a patient with a ventricular septal defect: need for antibiotic prophylaxis

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The development of infective endocarditis after insertion of an intrauterine contraceptive device is a rare occurrence,^{1,2} and hence the need for antibiotic prophylaxis in patients with pre-existing cardiac abnormalities is easily overlooked. We report the case of a young woman with a known ventricular septal defect in whom infective endocarditis occurred after insertion of an intrauterine device without antibiotic cover.

Case report

A 28 year old woman was admitted by her general practitioner with a three week history of lethargy, evening nausea, pains in the back and thighs, and

The insertion of intrauterine contraceptive devices carries a small but definite risk of infective endocarditis; antibiotic prophylaxis is required in susceptible patients

nocturnal sweats. She had had an intrauterine contraceptive device inserted four weeks earlier.

At the age of 5 the patient had been noted to have a pansystolic murmur; ventricular septal defect (maladie de Roger) was diagnosed clinically but no further investigations or treatment were considered necessary. She had, however, been advised about antibiotic prophylaxis for dental procedures. Five months before admission she had had a normal delivery, although dilatation and curettage had been required at seven days for postpartum haemorrhage. She had been given antibiotic cover with metronidazole and cephalixin for these events but had developed an allergic rash, presumed to be due to the cephalixin.

On examination she was flushed and feverish (38.2°C). She was in sinus rhythm at 72/min, blood pressure was 90/60 mm Hg, and jugular venous pressure was not raised. There was a pronounced systolic thrill over the precordium and auscultation disclosed a loud pansystolic murmur, loudest at the left sternal edge. Tip of the spleen was just palpable but she had no other sign of infective endocarditis. Vaginal examination showed the uterus to be normal and anteverted, the coil in situ, and no evidence of vaginal

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discharge. There was no microscopic haematuria. The contraceptive device was removed and sent for culture; occasional Gram positive cocci and Gram negative rods were seen on microscopy but no pathogens were isolated. Blood was taken for culture and treatment instituted with penicillin (3 MU four times a day intravenously) and gentamicin (80 mg thrice daily intravenously). *Streptococcus faecalis* was isolated from the blood cultures, and ampicillin (1.25 g four times daily intravenously) substituted for the penicillin. She made a rapid response to treatment and was discharged home within three weeks to continue oral pivampicillin 1 g thrice daily for a further three weeks. Subsequent two dimensional echocardiogram showed a small defect in the upper ventricular septum (figure).

Two dimensional echocardiogram (apical four chamber view) showing defect in membranous ventricular septum. RA=Right atrium. RV=Right ventricle. LA=Left atrium. LV=Left ventricle. VSD=Ventricular septal defect.

Comment

This patient had a documented ventricular septal defect (although it was considered to be unimportant haemodynamically) and was therefore at increased risk of endocarditis. Despite this, however, antibiotic prophylaxis was not given for the insertion of an intrauterine contraceptive device, although she had received prophylaxis during two previous deliveries. This suggests that insertion of an intrauterine contraceptive device is not generally considered to be a procedure likely to produce bacteraemia.

The recent survey of bacterial endocarditis by the Medical Services Study Group found that the portal of entry for the infecting organism was the genitourinary tract in 23 of 544 episodes (4.2%).³ Only four of these cases occurred in women and none in association with insertion of an intrauterine contraceptive device. That series,

however, was probably underreported, as it covered a two year period and the incidence of infective endocarditis has been estimated as roughly 900 cases a year.⁴ Indeed, only two definite cases of endocarditis in women fitted with these devices have been reported: both were associated with pre-existing valvular abnormalities and one was fatal^{1,2}; both occurred in young women in their 20s who had not previously been noted to have any cardiac problems.

The incidence of intrauterine infection after insertion of intrauterine contraceptive devices is reported as 2.5% in the first year in the United States⁵ and is thought to be even rarer in Britain.⁶ Insertion of an intrauterine contraceptive device is likely to introduce vaginal bacteria into the uterine cavity at insertion⁷ and hence provide a source of bacteraemia. In March 1984 the West Midlands Regional Health Authority Drug Information Service issued guidelines for the insertion of intrauterine contraceptive devices in patients at risk of endocarditis. These guidelines recommend that the devices should be fitted to such patients only after consultation between the gynaecologist and a cardiologist, and that the devices should never be fitted when there is a history of pelvic inflammatory disease. Full aseptic technique should be observed and parenteral antibiotic prophylaxis given.⁸

Although infective endocarditis is a rare complication of insertion of intrauterine contraceptive devices, it is an eminently preventable cause of morbidity and mortality. We suggest that all women attending for insertion should be examined for signs of cardiac abnormalities (even those considered not to be important haemodynamically—for example, small ventricular septal defects, mitral valve prolapse, etc) and if any doubt exists insertion should be delayed until cardiological opinion has been obtained; or if this is not practicable insertion of the device should be performed with full antibiotic prophylaxis—that is, gentamicin 1.5 mg/kg plus ampicillin 1 g, both intravenously 30 minutes before the procedure and repeated twice at eight hour intervals thereafter (if the patient is allergic to penicillin substitute erythromycin lactobionate 500 mg intravenously for ampicillin). A cardiological opinion should then be obtained as soon as practicable and urgent opinion obtained should an episode of fever occur.

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Many of the signs and symptoms of thyrotoxicosis resemble excessive adrenergic stimulation of the organ concerned—for instance, tachycardia, tremulousness, and sweating. It is also known that beta blockade can effectively treat these symptoms. Another major classic symptom of thyrotoxicosis is diarrhoea. This is somewhat paradoxical in the light of the observation that adrenergic stimulation decreases gut motility. Why is constipation not one of the presenting symptoms of thyrotoxicosis?

It is true that many of the symptoms and signs of thyrotoxicosis resemble excessive adrenergic stimulation. This is probably due to increased numbers of beta adrenergic receptors in hyperthyroidism, for the plasma concentrations of catecholamines fall rather than rise. It is also true that many patients with thyrotoxicosis have increased bowel movements, and this is paradoxical in the light of the fact that adrenergic stimulation reduces the motility of the gut. The reason for the tendency to diarrhoea in thyrotoxicosis is the rapid gastric emptying and intestinal transit time that are features of hyperthyroidism. Even steatorrhea is not uncommon in hyperthyroidism. This is not related to vagal stimuli but is probably a direct effect of thyroid hormone on the gut. Furthermore, prostaglandins and serotonin both stimulate small

intestine motility, and raised blood serotonin concentrations have been reported in hyperthyroidism. Thus although many of the symptoms and signs of thyrotoxicosis resemble excessive adrenergic stimulation, other symptoms and signs have alternative explanations.—C W H HAVARD, consultant physician, London.

Correction

The UK cardiac surgical register, 1977-82

Errors occurred in this article by T A H English *et al* (3 November, p 1205). In table III the signs for "less than" and "greater than" appeared the wrong way round. The first row of the table should have referred to the older age group. Also in table III, the column for 1982 should have read: greater than 1 year 1565 (7.0%); less than 1 year 447 (25.7%); total 2012 (11.2%). In table IV the column for 1982 should have read: coronary artery bypass grafts 6290 (3.3%); coronary artery bypass grafts and other procedures 942 (14.1%); procedures without graft 171 (22.8%); total 7403 (5.2%).