Though the smoking habits of our patients were carefully determined at entry and after two years by direct interview, data on subsequent smoking habits of the groups have not been taken into consideration in this report. Nevertheless, if some of those who had stopped smoking resumed the habit and some of those who continued to smoke subsequently gave up this would be expected to conceal an even greater divergence of the mortality curves. In our experience patients who have stopped smoking two years after a coronary attack seldom resume the habit although those still smoking at this time may eventually stop, particularly if they suffer a further non-fatal coronary episode.

A strong interaction between stopping smoking and the severity of the initial attack was apparent in terms of total mortality and mode of death. The effect of continued smoking on total mortality was greatest in those with unstable angina, suggesting that antismoking advice should be at least as compelling in those with less severe attacks. The greater benefit of stopping smoking in patients at lowest risk after infarction was also noted by Salonen.⁶ An earlier analysis of our patients suggested a greater benefit from stopping smoking in subjects with a complicated myocardial infarction.³ This finding, however, seems to have been heavily influenced by a small number of cases who died soon after discharge from hospital and re-examination of the data raises doubts about the accuracy of information on their smoking habits. In contrast, the choice of the two year follow up examination for the present report has yielded reliable information on smoking habits.

In terms of mode of death smoking had its strongest effect on sudden death in the patients with unstable angina and on fatal reinfarction in patients with complicated myocardial infarction. This may suggest that different pharmacological interventions may be appropriate in groups defined by severity of illness and smoking habit. There is no doubt, however, that stopping cigarette smoking is the most effective single action in the management of patients with coronary heart disease. Future trials of drug and surgical treatment in those surviving myocardial infarction should provide details of smoking habits at presentation and at follow up.

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SHORT REPORTS

Acupuncture needles as a cause of bacterial endocarditis

The incidence of infective endocarditis has remained unchanged during the past 40 years.¹ There are several reasons for this. Many patients at risk of infective endocarditis are unaware that they have a cardiac lesion, and others have poor dental health despite knowing of their susceptibility to infective endocarditis.² Medical and dental practitioners could do more to extend dental prophylaxis.³ Physicians should also be aware, however, of possible new ways of inducing endocarditis in at risk patients, so that appropriate advice and prophylaxis may be given. We report the development of bacterial endocarditis after the insertion and manipulation of acupuncture needles.

Case report

A 57 year old woman with a prosthetic Starr-Edwards valve in the mitral position presented at the outpatient department with a 10 day history of night sweats, fever, fast irregular palpitations, anorexia, lassitude, and increasing dyspnoea. She also complained of sudden weight gain with ankle and abdominal swelling. She had contracted rheumatic fever as a child and undergone mitral valvotomy in 1970 with valve replacement in 1972. Eighteen days before presentation acupuncture needles had been inserted in both ears in an attempt to stop her smoking. These needles had remained in situ for one week and had then been replaced by a second set after which she complained of irritation and a discharge from the skin around the needle.

On examination she was feverish, in fast atrial fibrillation and gross right heart failure, and with an enlarged pulsatile liver and tricuspid incompetence. There was also appreciable splenomegaly. An ejection systolic murmur was heard in the mitral area with clear prosthetic valve sounds. Two splinter haemorrhages and two Osler's nodes were noted, but no Roth spots were seen. Microscopic sterile haematuria was found. The erythrocyte sedimentation rate was 68 mm in the first hour. *Pseudomonas aeruginosa* (sensitive to gentamicin) was grown from two out of eight blood cultures, although no growth was obtained from an ear skin swab culture. Serological tests for other causes of endocarditis yielded negative results. An echocardiogram (M mode and sector) suggested two possible vegetations around the valve. Bacterial endocarditis was diagnosed. Treatment was started with increased diuretics and intravenous antibiotics (penicillin 3 MU every four hours and gentamicin 80 mg every eight hours). The gentamicin and intravenous penicillin were continued for four weeks and then oral amoxycillin was given to complete a six week course.

She made a full and uneventful recovery and was well six months later with no heart failure.

Comment

The patient was well aware of the need for prophylaxis against bacterial endocarditis and regularly received antibiotics before dental treatment. She had not considered acupuncture to be a procedure needing antibiotic cover; nor, presumably, had her acupuncturist. There was little doubt that clinically she had bacterial endocarditis. The source of infection was probably the acupuncture needle site, which was clearly inflamed. It is perhaps important that the needles were in situ for a long period and had been manipulated several times each day on instruction. The relevance of the growth of *P aeruginosa*

is uncertain, but this organism was assumed to be the cause of the endocarditis as no other organism was cultured. The endocarditis may have been caused, however, by a penicillin sensitive organism.

Bacterial endocarditis secondary to acupuncture has not previously been reported, and the incidence of local infection after acupuncture is also not recorded. With the increasing popularity of acupuncture physicians may need to consider advising antibiotic prophylaxis to patients with cardiac lesions who intend having acupuncture. If *Pseudomonas* was the causal agent in the patient reported a normal prophylactic regimen would not have been adequate. We suggest, however, that oral amoxycillin would be suitable for skin prophylaxis for most patients although flucloxacillin might be considered for those exposed to resistant staphylococci.

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Department of Cardiology, Guy's Hospital, London SE1 9RT

D B JEFFERYS, BSC, MRCP, registrar

S SMITH, BSC, MB, senior house officer

D A BRENNAND-ROPER, MA, MRCP, consultant cardiologist

P V L CURRY, MD, FRCP, consultant cardiologist

Correspondence to: Dr D B Jefferys.

Persistent atrial tachycardia in pregnancy

Sustained tachycardias are a rare complication of pregnancy especially in the absence of heart disease. We report an example of persistent atrial tachycardia in an otherwise healthy primigravida.

Case report

A 20 year old white woman noticed a persistently fast pulse rate in the initial weeks of her first pregnancy. She was booked into the antenatal clinic at 15 weeks, when a tachycardia was recorded. Apart from this there was no evidence of a cardiac abnormality. She had never suffered from palpitations and there was no history of rheumatic fever. On examination she was found to be normotensive and there were no physical signs of heart disease. Her only medication was promethazine for morning sickness, and the tachycardia persisted despite withdrawal of the drug. An electrocardiogram (figure) showed an atrial tachycardia of 160-180 beats/min with occasional periods of variable atrioventricular conduction resulting in a slightly slower ventricular rate. A chest radiograph, thyroid function values, and an echocardiogram were normal.

The pregnancy proceeded normally and at no stage did the patient develop signs of left ventricular impairment. She was admitted at 28 weeks for cardioversion, which proved unsuccessful. At 38 weeks she went into labour spontaneously and delivered a normal boy weighing 3000 g. There were no obstetric complications in the puerperium. Ten days post partum her heart rate suddenly reverted to normal. An electrocardiogram showed sinus rhythm at 70 beats/min. There was no recurrence of the tachycardia during more than one year of follow up.

Comment

Although both atrial and ventricular premature beats may occur during pregnancy,^{1,2} sustained tachycardias are rare especially if there is no structural heart disease. Most instances of sustained "supraventricular" tachycardia during pregnancy are related to anomalous atrioventricular conduction pathways of the type found in the Wolff-Parkinson-White syndrome. In our patient, however, the absence of ventricular pre-excitation and the finding of variable atrioventricular conduction during tachycardia excluded such pathways. Atrioventricular nodal re-entry was also unlikely. Hence the tachycardia apparently originated in the atrial myocardium. The P wave axis was difficult to determine but appeared to be normal in leads I, II, aVR, and aVF. This would be consistent with a focus in the high right atrium. In the general population these tachycardias

occur most commonly in association with a wide variety of organic heart diseases, none of which was detected in our patient.

The cardiovascular changes during pregnancy are well documented.³ There is an increase in blood volume together with a raised cardiac output and heart rate. These occur in the presence of high circulating concentrations of progesterone and oestrogen. Although the relevance of these changes to the aetiology of the atrial tachycardia must remain speculative, the sequence of events strongly suggests that pregnancy was an important factor in the initiation and maintenance of the arrhythmia. The incessant nature of the tachycardia and the failure

Twelve lead electrocardiogram about three months before delivery. QRS complexes were normal. Atrial rate was roughly 180/min. Periods of variable atrioventricular conduction were recorded in all limb and augmented leads.

to respond to cardioversion may imply abnormal atrial automaticity as the basic mechanism.

The pharmacological treatment of supraventricular tachycardias in pregnancy is similar to that in non-pregnant women.⁴ The most commonly used agents are digoxin, quinidine, and beta adrenergic blocking drugs, all of which appear to be safe with regard to fetal development. Nevertheless, although the risk of interference with fetal development or physiology is small it is preferably avoided. We therefore chose DC cardioversion, which is not contraindicated in pregnancy and does not harm the fetus.⁵ Although this failed to terminate the tachycardia, attempts at pharmacological termination and long term treatment were not considered in view of the lack of symptoms or objective evidence of left ventricular impairment.

We conclude that persistent atrial tachycardia may be uniquely associated with pregnancy. In the absence of symptoms or associated heart disease treatment may not be necessary, provided that careful monitoring is undertaken.

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Cardiac Department, Brompton Hospital, London SW3
W N HUBBARD, MRCP, senior house officer

Royal Cornwall Hospital, Truro, Cornwall
B A G JENKINS, MD, FRCP, consultant physician

Cardiology Department, St George's Hospital, London SW17
D E WARD, MD, senior registrar

Correspondence to: Dr D E Ward.