Non-cardiac chest pain: a useful physical sign?

EDITOR,—Non-cardiac chest pain is a major problem in general practice, in outpatients, and on the wards. Some patients are “reassured” that their pain is not cardiac but this is of course not the end of the matter. Professor Mayou and others 1 have demonstrated that these patients are still in trouble at follow up. Some are given the label “musculoskeletal” but this is not very convincing without a clear explanation. I suggest that this diagnosis can be supported in many cases by demonstrating reproduction of the pain by passive spinal movements. Production of symptoms by passive movements is well known to orthopaedic specialists. 2 Flexion, extension, lateral flexion, and rotation of the thoracic spine are the basic movements, and sometimes the position has to be held at the extreme for a least five seconds to reproduce symptoms—but I do not know whether the method of subluxation is not well known. Fifteen consecutive medical senior house officers and eight casualty officers were asked how they would demonstrate that chest pain was musculoskeletal and the best that they could do was percussion or direct pressure.

In six months of cardiology outpatients there were 27 patients (16 women and eight men) with a provisional diagnosis of angina whose history indicated another diagnosis. Their discomfort was either too unpredictable, too long or too short in duration, perhaps with paraesthesia in the fingers, or did not respond appropriately to antianginal drugs. One patient seemed to have oesophageal spasm and reflux and responded to appropriate treatment. One had pain reproduced by pressure on a costochondral joint. Of the remainder all but three had their discomfort reproduced in part or completely by passive spinal movements. Two patients also had their symptoms reproduced by percussion over the spine; both had vertebral collapse and insidious on osteoporosis and one from a secondary deposit. Two patients said that they were pale and sweaty during the pain, and pallor and sweating were reproduced by passive thoracic rotation and relieved by return to normal; a concurrent ECG was unchanged. Seven patients had exercise tests, two of which were positive and they await catheterisation; their pain has not responded to antianginal medication so there is doubt that they have angina.

Of 20 consecutive patients with typical angina and positive exercise tests, only three had some discomfort in the chest on thoracic spinal rotation and they were clear that it was a different pain from their angina. Review of the 27 patients with non-anginal pain at one year showed that seven had definite relief from non-steroidal anti-inflammatory drugs and one from physiotherapy. None had been admitted to hospital and those that still had pain seemed resigned to their symptoms but not anxious.

The exact origin of musculoskeletal pain remains obscure. It seems likely that the pain is referred along the intercostal nerves where it is exacerbated by inspiration, presumably from pain reflexes.

Coronary disease and inflammatory spinal disease can co-exist and this physical sign does not exclude angina but its presence in the absence of any objective evidence of myocardial ischaemia—for example, no ECG changes during pain, is helpful in the differential diagnosis. Perhaps more important it demonstrates that the pain is mechanised, often allowing confident discharge without a backup plan by the general practitioner and giving the patient a fairly clear explanation of the symptoms, which does far more than negative tests to avoid chronic ill health.

RICHARD A BEST
Department of Cardiology, Burnley General Hospital, Ganton Avenue, Burnley BB10 2PQ, UK


AV node ablation and implantation of mode switching dual chamber pacemakers: effective treatment for drug refractory PAF

EDITOR,—Marshall et al state that the combined procedure of atrioventricular (AV) node ablation and permanent pacemaker insertion for medically refractory paroxysmal atrial fibrillation (PAF) is justified on the basis of their study results. 1 We agree that in many patients with this condition AV node abolition and pacemaker insertion can improve the perceived quality of life; however, we feel that it is in the patient’s best interest that this procedure be performed in a staged manner with at least one month between pacemaker insertion and ablation. Lau et al previously identified a group of patients with drug resistant PAF in whom DDDR pacing prevented the need for subsequent AV node ablation. 2 Their conclusion was that up to a third of patients with drug refractory PAF may derive benefit from sensor driven atrial pacing alone and that this treatment can result in an improvement in patient perceived quality of life, without additional AV node ablation.

Permanent blockade of the AV node results in lifelong ventricular pacemaker dependency. This can result in long term deterioration in left ventricular function, 3 the development of mitral regurgitation, 4 and symptoms of dyspnoea, tiredness, and exercise intolerance. 5 In the absence of any objective evidence of myocardial ischaemia—for example, no ECG changes during pain, is helpful in the differential diagnosis. Perhaps more important it demonstrates that the pain is mechanised, often allowing confident discharge without a backup plan by the general practitioner and giving the patient a fairly clear explanation of the symptoms, which does far more than negative tests to avoid chronic ill health.

RICHARD A BEST
Department of Cardiology, Burnley General Hospital, Ganton Avenue, Burnley BB10 2PQ, UK


This letter was shown to the authors, who reply as follows:

Levy et al raise an important question. The timing of AV node ablation in relation to pacing is currently the subject of debate, although most published series describe ablation and pacing as a single procedure. However, the consensus of a recent discussion group report 6 was that ablation and pacing for PAF could be offered as a staged procedure to allow patients to pass the early high risk period for pacemaker lead displacement before removing them pacemaker dependent.

There is also increasing interest in pacing alone as a mode of treatment for drug resistant PAF and this raises the suggestion that dual chamber pacing should be carried out as a standalone procedure with later ablation only if there is no improvement. In contrast to pacing for sinus node disease (our study specifically excluded patients with significant bradycardia) the data to support pacing alone for PAF are far from clear. Levy et al refer to early data from the PA3 study in which some patients’ symptoms improved with DDIR pacing alone. 7 However, data from the same study also suggested that in general DDIR pacing does not prevent atrial fibrillation and indeed showed a trend for increased recurrence compared to no pacing (pacemaker programmed to DDI at 30 beats/min). 8 Other studies of pacing alone for PAF have been disappointing. While some have shown minor reductions in atrial
fibrillation frequency, none has demonstrated quality of life benefits. We accept that alternative atrial pacing sites and novel pacing algorithms may allow pacing to improve quality of life without the need for AV node ablation.

With regard to the complications of AV node ablation cited, the mitral regurgitation reported in two patients by te TOURNEAU et al was moderate before ablation and became severe post-ablation. Improved ventricular filling (achieved by good rate control) may well worsen mitral regurgitation and we advocate that patients with moderate mitral regurgitation undergo valve surgery before ablation. In patients with PAF this may well reduce left atrial pressure sufficiently to reduce the frequency of atrial fibrillation episodes. Possible deterioration of left ventricular function associated with long term right ventricular apical pacing has to be weighed against the possibility of patients developing tachycardia induced left ventricular dysfunction if they continue to be exposed to frequent prolonged episodes of rapid atrial fibrillation. Several studies have shown improvement in left ventricular systolic function after ablation and pacing for PAF. In addition, alternative sites for right ventricular pacing (or even biventricular pacing) may reduce the potential for long term left ventricular dysfunction.

In summary, we feel that either a one or two stage procedure is acceptable. A pragmatic approach might be to offer patients the choice of ablation and pacing at one sitting, which will improve symptoms but require pacemaker dependency, or a two stage procedure, which may avoid the need for ablation but may result in a second admission for symptomatic improvement. Given the high symptom burden of PAF patients being considered for ablation and pacing, we suspect many will choose the first option.

Management of polycythaemia in adults with cyanotic congenital heart disease

EDITOR,—I welcome Thorne’s editorial reiterating the pitfalls of overzealous venesection in adults with cyanotic congenital heart disease. As she states there is now a body of opinion highlighting the detrimental effects of inappropriate venesection. The evidence these conclusions are based on, however, is sparse and retrospective. This will unfortunately not only refute the belief that we must treat cocaine are discussed as potential triggers for myocardial infarction. Indeed, de Winter and colleagues recently reported a case of recurrent AMI caused by a soft atheromatous plaque within a myocardial bridge. This plaque was invisible during coronary angiography and could only be imaged using intravascular ultrasound. Thus, although myocardial bridging is usually clinically irrelevant, in selected cases it can be the culprit for acute coronary syndromes. In young patients with AMI particularly, documentation of bridging during angiography may be inadequate and complete evaluation using intravascular ultrasound and Doppler is advisable.

Value of improved treadmill exercise capacity

EDITOR,—Staniforth et al’s paper comparing exercise capacity in VVIR and VVI pacing modes in 12 patients with complete AV block showed that rate responsive pacing improved some but not all measures of exercise capacity. They conclude that the best investigation for assessing exercise capacity remains unclear.

On the contrary, we feel that what is at fault is the pacing mode rather than the investigation. It is widely accepted that the preservation of AV synchrony is optimal for patients with sinus rhythm and complete AV
block and is indeed recommended by the British Pacing and Electrophysiology Group. Maintaining AV synchrony with a physiological AV interval increases cardiac output both at rest and on exercise with normal and impaired left ventricular function. Therefore, the use of VVIR and VVI pacing is inappropriate for six of the 12 patients in their study who were in sinus rhythm with complete AV block.

DDD (or DDDR) pacing would have been more appropriate in these patients; therefore, this study compares two suboptimal pacing modes. Dual chamber pacing would almost certainly have resulted in better exercise capacity. The loss of AV synchrony with VVI or VVIR modes probably also accounts for the heterogeneity of the results assessing exercise capacity in this small group of patients. If dual chamber pacing had also been compared the results may well have shown an improvement in exercise capacity across the board. The results of the UK-PACE trial comparing VVI, VVIR, and DDD modes in higher degrees of AV block are awaited, but as with previous studies are expected to show that preservation of AV synchrony is the preferred mode on symptomatic, among other, grounds.

J D SOMAUROO
Specialist Registrar in Cardiology
D T CONNELLY
Senior Lecturer in Cardiology
The Cardiothoracic Centre—Liverpool NHS Trust,
Thomas Drive, Liverpool L14 3PE, UK


CORRECTION

Effects of reconstructive surgery for left ventricular anterior aneurysm on ventriculoarterial coupling (Heart 1999;81:171–76).

The incorrect Figure 5 was published in this paper. The correct figure and caption are shown below.

Figure 5 Pressure–volume loops in a single case before and after endoventricular circular patch plasty (EVCPP) repair. Straight lines indicate the Emax and Ea slopes. Emax increased slightly (from 1.3 to 1.63 mm Hg/ml), while Ea decreased (from 1.64 to 1.41 mm Hg/ml). SV did not change (from 69 to 71 ml), neither did V0 (from −16.7 to 23.3 ml). End systolic pressure decreased (from 112.85 to 100.0 mm Hg). Note the different time of maximum systolic pressure before and after surgery.