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 “reasonably sure” that their pain is not cardiac but this is of course not the end of the matter. Professor Mayou and others 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. 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, and they were clear that it was different pain from their angina.

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 mechanical, often allowing confident discharge without a backup medication and giving the patient a fairly clear explanation of the symptoms, which does far more than negative tests to avoid chronic ill health.

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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. We agree that in many patients with this condition AV node ablation 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. 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 longer term deterioration in left ventricular function, the development of mitral regurgitation, and symptoms of dyspnoea, tiredness, and exercise intolerance. Furthermore, to prevent complications, a combined AV node ablation and pacemaker insertion procedure is to deny a significant subgroup of patients the potential benefits of drug refractory ablation. We suggest that a trial period of rate responsive atrial pacing be undertaken in patients with drug refractory PAF before ablation. This will enable those patients who will benefit from pacing alone to be identified, and thus prevent them undergoing unnecessary AV node ablation.

We appreciate that there are potential cost implications to this staged approach; however, any increase in costs incurred may be offset by the decreased number of patients eventually requiring AV node ablation, and by the decrease in long term complications of ventricular pacing dependency.

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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 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 rendering 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 symptoms. 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 DDDR pacing alone. However, data from the same study also suggested that in general DDIR pacing does not prevent atrial fibrillation and indeed showed a higher recurrence compared to no pacing (pacemaker programmed to DDI at 30 beats/min). Other studies of pacing alone for PAF have been disappointing. While some have shown minor reductions in atrial fibrillation, some have shown no benefit. Therefore, it seems that pacing alone is not a satisfactory alternative to AV node ablation.
Management of polycythemia in adults with cyanotic congenital heart disease

Editor,—I welcome Thorne's editorial reiterating the pitfalls of overzealous venesection in young people with cyanotic congenital heart disease. As she states there is now a body of evidence highlighting the detrimental effects of inappropriate venesection. The evidence these conclusions are based on, however, is sparse and retrospective. This will unfortunately be a feature of a relatively new field such as adult congenital disease practice until multilocated collaboration and prospective studies are planned. Despite these limitations the work we have at present points towards the principles expounded by Thorne. A recent paper by Ammassi and Warnes,1 not mentioned in Thorne's editorial, provides further evidence regarding the lack of association between stroke and a high haematocrit. This study of cyanotic patients followed for 3135 patient-years did not identify an association between red cell mass and stroke. Of particular interest was the finding that iron deficiency and recurrent venesection were independent risk factors for cerebrovascular events. This study not only refutes the belief that we must treat haematocrits that are “too high” but positively demonstrates the dangers of venesection. This is reflected in the statement from Perloff et al that venesection should only be performed for “temporary relief of significant, intrusive hyperviscosity symptoms”.

Unfortunately, in the UK this message is not getting through, even to cardiac specialists let alone general physicians or haematologists who may only occasionally come into contact with this patient group. In our recent meeting of British cardiacologists in Scotland,2 there was a great variation in practice regarding the perceived indications for and techniques of venesection. Only a minority of consultants caring for these patients appeared to be aware of the dangers of inappropriate venesection or the appropriate management of iron deficiency. Many patients were still being submitted to regular venesection despite dubious indications with no fluid replacement and no monitoring for the onset of iron deficiency. In this specialist area, presenting simple guidelines such as those drafted by Thorne to a wider audience will hopefully redress this issue and improve patient care. It would be interesting to repeat a similar national survey of consultant practise in a year or so to determine whether this message is getting through.

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Myocardial infarction in young people with normal coronary arteries

Editor,—Williams and colleagues1 recently described two patients in their early 20s presenting with acute myocardial infarction (AMI) in the absence of significant coronary disease which demonstrated no evidence of atherosclerotic coronary disease. In their review of the pathogenesis of AMI in patients with “normal” coronary arteries, alcohol and cocaine are discussed as potential triggers for coronary spasm or local thrombus, but they do not mention the potential role of myocardial bridging.

Myocardial bridging is usually asymptomatic but has been related to AMI in patients as young as 15 years in the absence of risk factors for coronary artery disease and without evidence of coronary atherosclerosis.2 Although commonly a post-mortem finding,3 myocardial bridging is manifest as a segmental systolic kink in a coronary artery in up to 5% of patients undergoing diagnostic angiography. As coronary arterial blood flow is primarily diastolic, the relevance of bridging in clinical practice has been the subject of extensive debate.4 However, recent studies suggest that deep muscle bridges can twist the coronary artery and compromise diastolic blood flow5 and that this disturbance in flow at the site of the bridge might increase the propensity for intimal damage or platelet aggregation.6 Intravascular ultrasound and intracoronary Doppler studies have also indicated that bridging may play an important role in AMI or angina in some patients.4 Indeed, Williams and colleagues2 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.1 They conclude that the best investigation for assessing exercise capacity remains unclenched.

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 sub- optimal 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.

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

DDD is the best rate response system for subjects with normal sinus node function; compared with VVIR it offers improved quality of life and less pacemaker syndrome. The evidence that the haemodynamic improvement of DDD over VVIR automatically translates into an increase in treadmill exercise capacity is not so well established;1,2 and as such we do not accept the explanation of Somauoro and Connelly.

The purpose of our study was not to compare the benefits of various pacing modes, rather it was to use rate responsive pacing (in this case VVIR) as an instrument to compare the validity of various measurements of exercise capacity. It was methodologically unfortunate that some of our subjects were VVIR rather than DDD paced, but this was a reflection of the then accepted practice at the time when they had their original units implanted. The interesting observation we did make was that a treatment that is known to improve both symptoms and treadmill exercise capacity did not lead to an improvement in customary daily activity. This leaves us with the problem of divining the likely clinical benefit from the results of any study measuring treadmill exercise capacity. Just because someone can exercise harder in the laboratory does not mean that they will do so at home—but a quality of life questionnaire will show you if they do so with greater ease.

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 V (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.
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