Living with chest pain

Doctors are encouraged to follow evidence-based guidelines in the assessment and management of chest pain. Sometimes following these guidelines conflicts with clinical experience and even common sense, as is shown by the contrasting approaches (and outcomes) to this case.

Richard A Best

A clinical approach

A 52-year-old plumber (AB) was moving a heavy sink one morning. He sat down for a tea break, bent forward to reach a mug of tea, and developed a sudden, severe pain in the left side of his chest. His mate noticed that he was pale and a bit sweaty and was holding himself rather rigidly; worried that he might be having a heart attack, he drove him to his general practitioner's (GP) surgery, which fortunately was nearby.

When he arrived he was feeling a little better and the pain had almost gone but the receptionist, who had been told that all patients with acute, sudden or unexpected chest symptoms should be referred immediately to the doctor, called through to his GP, who apologised to the patient with him, came out and asked AB to go into another room so that he could examine him. While he quickly finished seeing the patient in consultation, he asked the practice nurse to run an electrocardiogram (ECG) and get out the notes.

The GP noted that AB was a smoker and was taking bendroflumethiazide 2.5 mg once daily for hypertension. He then asked for details of the symptoms. He found that the pain had been sharp in the left chest in the costo-chondral region. When it came on, AB felt that he needed to sit hunched forward and that when he took a deep breath it hurt. He was otherwise fit and asymptomatic. The general practitioner then examined him and found that passive rotation of his thoracic spine to the left reproduced a milder version of the pain and to the right tended to relieve it. He also found that cervical spinal flexion seemed to relieve it, and that cervical extension and left rotation made it worse.

The ECG which had been taken while the pain was fading was unremarkable except for voltage criteria for left ventricular hypertrophy.

The GP reassured AB that his pain was clearly and demonstrably mechanical, pointed out that rib rotation on a painful or displaced joint will cause pain and explained that he was short of breath because rib rotation is required in breathing. He also explained referred pain and that sudden pain, even if severe, does not necessarily result from a serious condition and drew the parallel of a dentist drilling into a dental nerve. He explained that severe pain produces adrenaline secretion, which will result in pallor and sweating.

He recommended a visit to the practice physiotherapist, who would discuss with him postural training and exercises; he also took the opportunity to encourage weight loss, regular exercise, stopping smoking and, ignoring the slightly raised blood pressure as being due to pain, nevertheless arranged a repeat visit in a week's time to make sure that blood pressure control was adequate in view of the evidence of left ventricular hypertrophy on the ECG. AB went back reassured to plumbing in a new sink but he did take the message seriously about weight, smoking, exercise and posture and six months later he was a much fitter man.

Following the guidelines

A 52-year-old plumber (AB) was moving a heavy sink one morning. He sat down for a tea break, bent forward to reach a mug of tea, and developed a sudden, severe pain in the left side of his chest. His mate noticed that he was pale and a bit sweaty and was holding himself rather rigidly; worried that he might be having a heart attack, he drove him to his GP's surgery, which fortunately was nearby.

When he arrived the receptionist told him that his GP and two of the other partners were in a multi-disciplinary committee meeting that morning and couldn't be disturbed. The fourth GP had appointments all morning and couldn't see him. She suggested if he had chest pain that he should go to the A & E Department, and his mate therefore took him there. Speed of management had been emphasised here and he was seen in just three minutes' time by the triage nurse, who filled in the chest pain protocol form, ticking the boxes for acute chest pain within one hour, related to exertion, accompanied by sweating and dyspnoea together with a past history of hypertension and smoking. She took an ECG, which showed voltage changes of left ventricular hypertrophy and rather high ST take-off in v2 and v3 with up-sloping ST segments slightly concave upwards. She immediately rang the medical SHO on duty for admissions, summarising her checklist. She asked him to come quickly, reminding him of the forthcoming audit of door-to-needle times in which the hospital had recently scored rather poorly.

While she was waiting she inserted a Venflon, gave the patient 300 mg of aspirin and reflected aloud to a colleague as to why the GP surgery had not given him aspirin immediately and why they had allowed his mate to drive him to hospital rather than sending for an ambulance. AB asked why they should have done this and she explained that should he have had a cardiac arrest in the car on the way to hospital it would have been very difficult for his mate to know what to do. This brief conversation not only destroyed what remaining confidence he had in his general practice but so frightened him with the severity of his condition that he tensed up and the pain, which had settled, recurred in mild form.

When the SHO arrived he correctly introduced himself and compassionately asked how the pain was. When told that it had returned he asked the nurse to give the patient intravenous diamorphine, which she did, resulting in sedation, confusion, vomiting and later constipation. The SHO decided that the ST elevation, which was consistent just with left ventricular hypertrophy, met the (2 mm ST elevation in consecutive chest leads) criteria for thrombolysis. Realising how recent the pain had been, he ordered a newer thrombolytic drug which, in his hospital's protocol, was indicated in early myocardial infarction in preference to streptokinase, which does not produce such early patency of arteries but has half the risk of cerebral haemorrhage and is about six times less expensive. Fortunately this caused no problems; indeed, when he reached a bed some three hours later, tired, confused and frightened, he met the consultant doing a post-take ward round who commented learnedly on the number of lives saved by early thrombolysis, which in this case had clearly prevented even a rise in troponin.

Five days later AB returned home, devastated at his change in life and warned that he would not be able to work for six weeks and that he would need to do only light physical work after that. How this was possible for a plumber was not explained. He returned three weeks after his chest pain for an exercise test. He didn't feel very well, perhaps because of the beta blockers, aspirin, statins and angiotensin-converting enzyme (ACE) inhibitor with which he was blessed, and he was not surprised that with a combination of fatigue and some return of his chest pain (which had been niggling whenever he bent forward or breathed in since he left hospital), he could only do four and a half minutes on the treadmill.

He was impressed when the cardiologist appeared while the leads were being removed but horrified when he was told that after thrombolysis the duration of a stress test was an important guide to prognosis and he was at significant risk of serious damage to his heart. It was suggested that he went for angiography and he accepted expert advice and was put on the list.

Seven months later

He waited for seven months for this test. It was, of course, known that those with unstable coronary syndrome and continuing pain had a high likelihood of infarction or death within the next six months but nobody could shift the waiting list. Very reasonably, in order to avoid any suggestion of blame from the relatives should something go wrong, he was advised that he should not work until he'd had his angiogram and things were sorted out. Unfortunately, the firm for which he worked was unable to keep him paid but not working, so he lost his job.

When he was admitted for his angiogram the doctor who was going to do the test, a third year specialist registrar, explained in detail all the potential risks and AB signed the appropriate form to say that he accepted these. Unfortunately, during the procedure a small plaque of atheroma in the aorta was detached through no fault of the operator and was carried up the carotid to the brain, where it caused a degree of permanent damage and a left-sided hemiplegia which remained with him life-long. On angiography no plaque was found in excess of > 40% stenosis and his case provided an interesting demonstration to the students that infarction was caused in 60% of cases by small stenoses that might not even be detectable on angiography.

All credit in the audit meeting to his doctors that some five years later, when he was finally discharged from out-patients, he was still taking aspirin together with a beta blocker, an ACE inhibitor and a statin and had not developed any further problems. Since he had stopped work he had not even had a recurrence of his chest pain except, strangely, when he bent forward in his armchair in front of the television to pick up a cup of tea.

Acknowledgement
With acknowledgements to Dr Rory CM McGouran. Dying with dignity. *Clinical Medicine* 2002;**2**(1):43.

Conflict of interest
None declared.

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Br J Cardiol 2005;**12**:85–9

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Has PEACE declared war on EUROPA?

Dear Sirs,

A recent report from the American Heart Association Meeting included a summary of the PEACE trial.[1] Given that the results – showing that angiotensin-converting enzyme (ACE) inhibitors were of no benefit in stable coronary artery disease – appear to be inconsistent with those from EUROPA, it is perhaps inevitable that PEACE will provoke controversy.

In the original paper,[2] the PEACE investigators attempted to explain the differences between the results of the two studies on the basis that the patients recruited to PEACE and EUROPA[3] were dissimilar. The arguments that they put forward are unconvincing. Both studies specifically described their patient population as being at 'low risk' of cardiovascular events and the baseline data – taken collectively – suggested only minor differences.

The authors of PEACE placed great emphasis on differences in outcome between the placebo groups of the two studies. This, however, is not supported by the data which showed little difference in non-fatal myocardial infarction (5.3% *vs.* 6.2%), cardiovascular mortality (3.7% *vs.* 4.1%), total mortality (8.1% *vs.* 6.9%), or the common combined end point (8.6% *vs* 9.9%) in the placebo groups of PEACE and EUROPA respectively.

Why did the PEACE investigators strive so hard to account for the differences rather than simply accepting that their study failed to demonstrate any benefit from ACE inhibitors? And would they have searched so eagerly for these differences if the results of PEACE and EUROPA had been consistent? In this context, it is pertinent to draw attention to the conflicts of interest reported by some of the authors of the PEACE trial. In any case, regardless of their motives, their efforts to explain the differences are in vain. The notion that one large-scale trial can verify or falsify the results of another is a myth.[4,5] Strictly speaking, different trials are not comparable.

What, then, can be said about the use of ACE inhibitors in 'low risk' patients with ischaemic heart disease? Firstly, despite many thousands of patients being treated for more than four years in both EUROPA and PEACE, these drugs had no effect on either cardiovascular deaths or total mortality. And, secondly, in terms of preventing myocardial infarction, EUROPA reported only a trivial absolute reduction of 1.4%, while PEACE failed to show any difference. Surely a reasonable conclusion would be that ACE inhibitors have no role in the long-term management of this subgroup of patients with ischaemic heart disease?

Conflict of interest
None declared.

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Yours faithfully,
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