reperfusion but also on successful tissue reperfusion. Regarding this aspect, the contribution of further dynamic electrocardiographic patterns[6] and of serum cardiac markers will have to be established.

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References

[1] Yusuf S, Collins R, Peto R *et al.* Intravenous and intracoronary fibrinolytic therapy in acute myocardial infarction: overview of results on mortality, reinfarction and side-effects from 33 randomized controlled trials. Eur Heart J 1985; 6: 556–85.

[2] TIMI Study Group The thrombolysis in Myocardial Infarction (TIMI) trial; Phase 1 findings. N Engl J Med 1985; 312: 932–6.

[3] Shah PK, Cercek B, Lew AS *et al.* Angiographic validation of bedside markers of reperfusion. J Am Coll Cardiol 1993; 21: 55–61.

[4] Ellis AK, Little T, Zaki Masud AR *et al.* Early non-invasive detection of successful reperfusion in patients with acute myocardial infarction. Circulation 1988; 78: 1352–7.

[5] Jurlander B, Clemmensen P, Ohman EM *et al.* Serum myoglobin for the early non-invasive detection of coronary reperfusion in patients with acute myocardial infarction. Eur Heart J 1996; 17: 399–406.

[6] Ochiai M, Isshiki T, Hirose Y *et al.* Myocardial damage after successful thrombolysis is associated with the duration of ST re-elevation at reperfusion. Clin Cardiol 1995; 18: 324–8.

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Angina, exercise and food

See page 394 for the article to which this Editorial refers

For the normal individual, the cardiovascular challenge of eating is insubstantial. The original observation that angina may be worsened by eating was made more than two centuries ago by William Heberden[1]. Classical studies in 1934 suggested that exercise capacity fell by 25% after a meal[2]. In addition, Goldstein *et al.* found 11 of 12 subjects developed angina pain soon after eating[3]. Ingestion of even a modest meal increases heart rate and stroke volume, raising cardiac output by around 30% in patients with angina[4] and up to 60% in healthy volunteers[5]. Blood pressure remains unchanged as peripheral vascular resistance falls due to gut vasodilatation. A more prolonged elevation of cardiac output may occur with meals of a greater energy content[6]. High carbohydrate food results in a rapid haemodynamic response and fat or protein meals result in a delayed response. The increase in cardiac output post-prandially in patients with coronary artery disease represents a substantial proportion of their reduced cardiac reserve and the resulting raised myocardial oxygen demand may lead to anginal symptoms.

As a symptom of coronary artery disease, post-prandial exertional angina may denote the presence of more severe underlying disease than is present in patients without this symptom. Berlinerblau and Shani questioned 408 patients with chest pain who underwent coronary angiography[7]. Thirty-five patients (8·6%) had post-prandial angina, at rest and on exertion, most commonly after an evening meal. Post-prandial angina occurred mainly in men, was associated with rest angina and a high incidence of left main and three-vessel coronary artery disease. The ejection fraction was lower in patients with post-prandial angina (0·39 vs 0·47). Post-prandial angina is therefore an indication of severe coronary artery disease and such patients should be considered for coronary angiography.

This issue contains a report concerning the effects of eating meals of differing composition on effort tolerance in patients with angina[8]. The authors performed four different pairs of exercise treadmill tests at least one week apart, before and 30 min after meals of mainly fat, mainly carbohydrate, a balanced meal or water alone, in 14 patients with chronic stable angina. Cardiac output was estimated non-invasively using Doppler echocardiography. The authors report improvement of 72 s in the post-prandial exercise time, after a meal consisting mainly of fat in comparison with a balanced meal. Cardiac output increased significantly only following a balanced meal, compared with water alone.

One explanation offered for the lower exercise capacity in patients following food is that of

increased sympathetic activation relating to the carbohydrate component of the meal. However, whilst feeding activates the sympathetic nervous system[9], plasma catecholamines did not vary before and after food in one study[5]. Does treatment with beta-blockers attenuate the post-prandial response? In 1980, Delage and colleagues exercised 12 patients pre- and post-prandially after an 800 calorie meal[10]. Metoprolol slowed the time to development of post-prandial angina compared with placebo by 75 s, a similar improvement to that reported by the current study for a meal of mainly fat compared with a balanced meal.

Since many patients with angina are already taking beta-blockers, if this mechanism is the sole explanation for the post-prandial haemodynamic changes, then patients are receiving the correct therapy. However, the situation is doubtless more complex than this. Presumably, the release of locally produced vasoactive substances and the role of the gastrointestinal nervous system are also important. Vasoactive intestinal peptide for example is a potent vasodilator, infusion of which can cause reduction in peripheral vascular resistance, a fall in blood pressure and a rise in cardiac output. A further similar study comparing the effects of continuing to take beta-blockers compared with withdrawal beforehand might help to illuminate the postulated role of sympathetic activation. However, at present, the fine mechanisms controlling the haemodynamics of eating in patients with coronary artery disease remain unclear.

One possible conclusion from the current study might have been that patients should increase their dietary fat intake. For the sake of a 72 s improvement in exercise tolerance 30 min after a meal, exercise which the authors sensibly caution against, the associated potential deleterious effects of increased serum lipids do not seem worthwhile. One should not, therefore, recommend increased risk of progression in atherosclerosis for the sake of post-prandial exercise gain.

Should all patients with angina be told to rest for 30 min or an hour following a meal? If so, how much food is a meal and how much is a snack? These questions cannot be answered at present. Studies of the dose response to different amounts of food have not yet been performed. However, data from a study by Hung and colleagues provide some clues[11]. They compared the cardiovascular responses to combined static–dynamic effort, post-prandial dynamic effort and dynamic effort alone by upright bicycle ergometry in patients with ischaemic heart disease. The cardiovascular response to combined static–dynamic effort and to post-prandial dynamic effort became similar to that of dynamic effort alone, as dynamic effort reached a symptom limit. If significant ischaemic and arrhythmic abnormalities were absent during symptom-limited dynamic exercise testing, they were unlikely to appear during combined static–dynamic or post-prandial dynamic effort. Therefore, it appears that patients with a negative exercise test are at low risk of post-prandial angina. These patients might therefore be allowed to exercise following food, if they so wished.

Investigation of the mechanisms of post-prandial angina should continue. For the time being, that patients with angina should avoid exercise in the immediate period following a meal continues to be perfectly reasonable advice. Perhaps, more importantly, patients with post-prandial angina should be informed of the increased likelihood of the presence of prognostically significant coronary disease and coronary angiography should be recommended.

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References

[1] Heberden W. Some account of a disorder of the breast. Medical Transactions of the Royal College of Physicians. London 1772; 2: 59–67.
[2] Wayne EJ, Graybiel A. Observations on the effects of food, gastric distension, external temperature, and repeated exercise on angina of effort, with a note on angina sine dolore. Clin Sci 1934; 1: 287.
[3] Goldstein RE, Redwood DR, Rosine DR, Beiser GD, Epstein SE. Alterations in the circulatory response to exercise following a meal and their relationship to post prandial angina. Circulation 1971, 44: 90–100.
[4] Cowley AJ, Fullwood LJ, Stainer K, Harrison E, Muller AF, Hampton JR. Postprandial worsening of angina: all due to changes in cardiac output? Br Heart J 1991; 66: 147–50.
[5] Kelbaek H, Munck O, Christensen NJ, Godtfredsen J. Central haemodynamic changes after ingestion of a meal. Br Heart J 1989; 61: 506–9.
[6] Sidery MB. Meals lie heavy on the heart. J Roy Coll Phys Lond 1994; 28: 19–23.
[7] Berlinerblau R, Shani J. Postprandial angina pectoris: clinical and angiographic correlations. J Am Coll Cardiol 1994; 23: 627–9.
[8] Lam FY, Wilson AT, Channer KS. The effect of meals of differing composition on exercise tolerance in patients with angina pectoris. Eur Heart J 1996; 17. 394–8.
[9] Landsberg L, Young JB. Fasting, feeding and regulation of the sympathetic nervous system. N Engl J Med 1978; 298: 1295–1301.
[10] Delage F, Rouleau JR, Labelle JL, Dagenais GR. Effects of metoprolol on effort angina during the postprandial state. Clin Pharmacol Ther 1980; 27: 763–8.
[11] Hung J, McKillip J, Savin W *et al.* Comparison of cardiovascular response to combined static–dynamic effort, post-prandial dynamic effort and dynamic effort alone in patients with chronic ischemic heart disease Circulation 1982; 65: 1411–9.