Ischaemic heart disease

High incidence of undiagnosed diabetes mellitus in patients with AMI

Of 181 consecutive non-diabetic patients with acute myocardial infarction (AMI), 35% (95% confidence interval [CI] 28% to 43%) and 40% (95% CI 32% to 48%) had impaired glucose tolerance at discharge and after three months, respectively, while 31% (95% CI 24% to 38%) and 25% (95% CI 18% to 32%) had previously undiagnosed diabetes mellitus. Independent predictors of abnormal glucose tolerance at three months were concentrations of HbA1c at admission (p = 0.024) and fasting blood glucose concentrations on day 4 (p = 0.044).

Prevention of NIDDM with acarbose

Over 1400 patients with impaired glucose tolerance were randomly allocated to 100 mg acarbose or placebo three times daily. At a mean follow-up of 3.3 years, 211 (31%) of 682 patients in the acarbose group and 130 (19%) of 686 on placebo had discontinued treatment.

Tropinone is still predictive in patients with ACS plus renal failure

Tropinin T is renally cleared and so may remain elevated for long periods in patients with renal impairment. In the GUSTO IV trial, death or myocardial infarction occurred in 581 of >7000 patients. Among patients with a creatinine clearance above the 25th centile value of 58.4 ml per minute, an abnormally elevated troponin T concentration (>0.1 ng/ml) was predictive of an increased risk of myocardial infarction or death (7% v 5%; adjusted odds ratio [OR] 1.7, 95% CI 1.3 to 2.2; p < 0.001). Among patients with a creatinine clearance in the lowest quartile, an elevated troponin T concentration was similarly predictive of increased risk (20% v 9%; OR 2.5, 95% CI 1.8 to 3.3; p < 0.001).

 Syndrome X patients may have subendocardial ischaemia

Previous work suggests that there may be abnormal pain sensation or abnormal microvascular leakage in syndrome X patients. Now cardiovascular magnetic resonance imaging has demonstrated subendocardial hypoperfusion in the intravascular administration of adenosine, which is associated with intense chest pain. These data support the notion that the chest pain may have an ischaemic cause.

Unstable angina is not caused by a single vulnerable plaque

Previous studies suggest that there is more than one complex plaque in patients with unstable angina. This is backed up in this study which shows that the inflammation (as measured by neutrophil myeloperoxidase depletion) is present in aortic blood and in the venous drainage of the left coronary artery, irrespective of the site of coronary stenosis. There is a transmural gradient of activation in unstable angina not seen in stable angina or variant angina.


The end of in-stent restenosis? Restenosis after coronary stenting occurs in 20–30% of cases. The development of stents coated with sirolimus or paclitaxel looks like a promising new approach to inhibit smooth muscle proliferation within stents. The RAVEL trial suggests that restenosis rates are close to zero in the sirolimus coated arm and >25% in the standard stent arm. The benefits were maintained at one year. The disadvantages is that the coated stents may cost three times as much as ordinary stents.


High dose statins reduce MACE whether or not PTCA is done

A lot was made of the AVERT trial comparing 80 mg atorvastatin versus coronary angioplasty (PTCA) for low risk patients. Clearly lipid lowering reduced event rates more that just PTCA. The LIPS trial answers the obvious question of whether early lipid lowering after PTCA has additional benefits. The average low density lipoprotein cholesterol concentration was 3.4 mmol/l, and follow up was for four years. Major adverse cardiac events (MACE) were reduced from 26.7% in the placebo arm to 21.4% (relative risk [RR] 0.78, 95% CI 0.64 to 0.95; p = 0.01). There were trends to reduction in death, MI, and revascularisation rates. This suggests that early statin treatment should be given in nearly all patients undergoing PTCA.


10% annual rupture rate for AAA >5.5 cm that are left alone

Of 198 veterans whose abdominal aortic aneurysm (AAA) was left alone for medical reasons or refusal to have surgery, 57% had died after an average follow up of 1.5 years. The one year incidence of probable rupture by initial AAA diameter was 9.4% for AAA of 5.5–5.9 cm, 10.2% for AAA of 6.0–6.9 cm (19.1% for the subgroup of 6.5–6.9 cm), and 32.5% for AAA of 7.0 cm or more. Much of the increased risk of rupture associated with initial AAA diameters of 6.5–7.9 cm was related to the likelihood that the AAA diameter would reach 8.0 cm during follow up, after which 25.7% ruptured within six months. Pericranial insertion of covered stents may provide a non-surgical solution in the future.

General cardiology

Biventricular pacing improves symptoms in heart failure ▶
In a randomised controlled trial of 450 patients, those whose biventricular pacemakers were turned on had subjective (New York Heart Association class, quality of life) improvements in their condition as well as improvements in objective markers. There was an improvement in the distance walked in six minutes (39 m v 10 m, p = 0.005), time on the treadmill during exercise testing (81 s v 19 s. p = 0.001), and ejection fraction (+4.6% v -0.2% p < 0.001). In addition, fewer patients in the group assigned to cardiac resynchronisation than control patients required hospitalisation (8% v 15%, p < 0.05). The trial was not powered to detect a mortality advantage for this procedure. There is a risk with this procedure and two patients died as a result of the implantation.


Infective endocarditis still has an in-patient mortality of 16% ▶
A French registry of 390 cases of infective endocarditis in 1999 showed that, compared to 1991, mortality had reduced from 21.6% to 16.6%. In addition, patients without prior heart disease made up 4% of cases. The population incidence of infective endocarditis is 31 per million, except in New Caledonia, where it is 161 per million.


Tennis prevents heart disease but baseball does not ▶
In 1000 medical students followed for 40 years, sporting activity was related to cardiovascular health. After adjustment for father’s occupational, parental incidence of cardiovascular disease, serum cholesterol concentration, cigarette smoking, body mass index, and hypertension during follow up, the relative hazard of developing cardiovascular disease was 0.56 (95% CI 0.35 to 0.89) in the high activity group, compared with the no-activity group. Strangely, other sports did not have this effect, perhaps as they could not be maintained as one ages.


Basic science

How LVH turns to heart failure in long standing pressure overload ▶
Gq proteins couple membrane receptors for angiotensin II, endothelin-1 and adrenaline (epinephrine) to the cardiac hypertrophy response. Protein NIX is part of the apoptotic response that explains how pressure induced hypertrophy can turn to heart failure. It is strikingly induced in Gq-dependent and pressure overload hypertrophy and, when expressed in vitro, localises to mitochondria and causes rapid cell death with caspase-3 activation of apoptotic nuclear changes. Expressed in the in vivo mouse heart, NIX provoked a dilated cardiomyopathy that was invariably lethal because of massive cardiomyocyte apoptosis within days of detectable protein expression.


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3 Management of Marfan syndrome JCS Dean July 2002;88:97–103. (Education in Heart)

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7 Meta-analysis of randomised controlled trials of the effectiveness of antiarrhythmic agents at promoting sinus rhythm in patients with atrial fibrillation G Nichol, F McAlister, B Pham, A Laupacis, B Shea, M Green, A Tang, G Wells June 2002;87:535–43. (Cardiovascular medicine)


9 Development and structure of the atrial septum RH Anderson, NA Brown, S Webb July 2002;88:104–110. (Education in Heart)

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