Electrophysiologic Remodeling of the Atrium in Patients with Atrial Fibrillation
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Previous studies have shown that sustained atrial fibrillation [AF] causes atrial electrophysiologic remodeling. Whether remodeling affects the atrium uniformly and consistently in patients [pts] with paroxysmal AF [PAF] and chronic AF [CAF] is unclear. We measured: 1) atrial conduction time [CT] at multiple sites in right atrium [RA] (mid & low) and left atrium [LA] (proximal, mid & distal coronary sinus [DCS]) during sinus rhythm; 2) effective refractory periods [ERPs] at high RA [HRA], low RA [LRA] and DCS at 400 & 600 ms drive cycle length (±4 x threshold) in 11 pts (mean age:61±15 yrs) with CAF (mean AF duration:19±6mths) after successful transvenous defibrillation; 8 pts (mean age:47±14 yrs) with PAF and 10 controls (C) (mean age:50±12 yrs)

Results: No significant difference in RA CT was observed. CAF pts had significantly prolonged LA CT vs C. CAF pts had significant shortening of DCS ERPs and paradoxically prolonged LRA ERPs as compared with C. Both PAF and CAF pts had lost normal adaptation of ERPs at HRA & DCS, but was preserved in LRA.

Conclusions: 1) In CAF, electrophysiological abnormalities mainly occur in LA. 2) Electrophysiological remodeling seen in pts with AF affects the atrium nonuniformly with shortening or prolongation of ERPs in different parts of atrium, which may increase the heterogeneity of atrial electrophysiological properties and contributes to perpetuation of AF. 3) PAF is associated with a modest prolongation of LA CT and maladaptation of ERPs in high RA and DCS, suggesting progressive atrial electrophysiologic remodeling.

COST-EFFECTIVENESS OF SECONDARY PREVENTION WITH STATINS IN NORMOCHELSTEROLAEMIC PATIENTS
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The Cholesterol and Recurrent Event (CARE) study showed a significant reduction in recurrence of myocardial infarction (MI), but not overall mortality, in patients with normal cholesterol levels. Treatment with 40mg pravastatin daily for 10,405 patient years prevented 50 MIs and 98 revascularisation (coronary angioplasty and bypass surgery) procedures. Our aim was to analyse the cost-effectiveness of lipid lowering therapy using CARE criteria.

Cost of drugs and lipid measurements were determined. Cost per quality-adjusted life year (QALY) gained was calculated after deduction of benefits and savings. Benefits include potential increase in earnings from longer life expectancy and QALY gained from MI prevention. Savings include prevention of acute admission and lifetime follow-up after MI, revascularisation procedures and stroke. The mean life expectancy after MI and quality of life (QOL) after MI were taken as 8.71 years and 0.85 of normal respectively. Stroke was assumed to decrease life expectancy by half.

Costs of drugs and lipid measurements amounted to HK$7,948 per patient per year. After considering QALYs gained from lives saved, non-fatal MI prevented and stroke prevented, cost per QALY gained was $237,653. Benefits and savings were estimated to be $11,331 per patient per year. Net saving was $101,136 per QALY gained. If the benefits due to stroke prevention were excluded, the net saving became $45,499 per QALY gained. Sensitivity analysis showed that there was a net saving under a wide range of parameters in our economic model.

The cost-effectiveness of lipid lowering therapy by CARE criteria is acceptable. When all benefits to patients and savings to health care providers were taken into account, there was net benefit in treating patients with a statin after MI according to CARE criteria.