Early Descriptions of Possible Angina Pectoris

Notwithstanding the conditions prevailing before the eighteenth century, there were undoubtedly some few individuals who had the potential for development of ischaemic heart disease. This can occur in the absence of exogenous risk factors for coronary arteriosclerosis, for example, in rare homozygous subjects with familial abnormalities of cholesterol metabolism.[3] Congenital coronary arterial anomalies can also very occasionally cause angina independently of arteriosclerotic disease.[4] Either of these could have accounted for Heberden's twelve-year-old patient with exertional chest pain. The present work does not therefore attempt to prove that angina pectoris was a totally new syndrome emerging in the mid-eighteenth century in the way, for example, that Acquired Immune Deficiency Syndrome (AIDS) first became manifest in the late 1970s. It *is* being suggested that before the mid-eighteenth century angina pectoris was too rare to have been recognisable as a distinct clinical entity by any one physician.

Coronary Heart Disease Frequency in the Absence of Traditional Risk Factors

In mid-twentieth century studies, the frequency of symptomatic coronary heart disease occurring in the then apparent absence of risk factors may have been overestimated. When the association of lipid abnormalities with increased risk of coronary arterial disease was first recognized, "normality" of serum cholesterol was based on results of measurements that were made in apparently healthy subjects. Levels that were then accepted as normal would now be regarded as pathologically high and a consequence of the excessively fatty diets and possibly resulting obesity that has been all too common in western societies. As late as 1998, the CARE study, an investigation of the effect of pravastatin (a cholesterol lowering medication) on the incidence of coronary events among survivors of a first myocardial infarction was initiated because total serum cholesterol levels in the range of 5.2 to 6.2 mmol/L were then considered to be unproven as risk factors and therefore regarded as normal. Indeed the ethical decision to allow randomizing half the study population to placebo treatment was based on this premise. The term hypercholesterolaemia was reserved for levels above 6.2 mmol/L. The results of the CARE study itself showed that serum cholesterol concentrations earlier categorized as "average" should now be considered pathologically high, even 5.2 mmol/L being considered excessive.[5]

A measure of the incidence of overt coronary heart disease in populations with total serum cholesterol levels that are acceptable as normal by present standards

[3] Peter H Jones and Antonio M Gotto, 'Assessment of lipid abnormalities in the heart', in J Willis Hurst and Richard C Schlant (eds), *The heart, arteries and veins*, 7th ed., New York, McGraw Hill, 1990, p. 378.

[4] J Noble *et al.*, 'Myocardial bridging and milking effect of the left anterior coronary artery. Normal variant or obstruction?', *Am J Cardiol*, 1976, **37**: 993–9, p. 997.

[5] F M Sacks *et al.*, 'Relationship between plasma LDL concentrations during treatment with pravastatin and recurrent coronary events in the Cholesterol and Recurrent Events trial', *Circulation*, 1998, **15**: 1446–52, p. 1447.