physicians to come away with the message that obesity is not a health risk as long as traditional coronary atherosclerotic risk factors are normal.

Two other points concerning obesity deserve mention. First, not all obesity confers the same degree of abnormality in coronary heart disease risk factors. The patient with abdominal obesity (the 'apple' form of obesity often associated with males) is linked to insulin resistance and hence significant risk for the development of atherosclerosis. Thigh and buttock obesity (the 'pear' form of obesity often associated with females) confers minimal coronary heart disease risk probably because fat in this zone is not as metabolically active as abdominal fat. Another point made by Ashton *et al.* that deserves repeating is that modest weight loss is capable of remarkable improvement in abnormal coronary heart disease risk factors. It has often been observed that modest ($\sim$10% of body weight) loss of weight produces marked amelioration in elevated blood pressure, abnormal serum cholesterol, and hyperglycaemic tendency. I am in complete agreement with Ashton *et al.* when they advise moderate weight loss for obese patients. Demanding that the patient seek to reach their ideal body weight is often unrealistic and discourages compliance with the prescribed programme of diet and exercise. In the end, 'the enemy of good is perfect', that is, we should strive to enlist our patients in a programme that produces moderate, sustained weight loss rather than advising a draconian strategy that eventually fails to induce the patient to lose any weight at all. As recently pointed out by the National Task Force on the Prevention and Treatment of Obesity here in the U.S., the best strategy for weight loss would appear to be one of the moderate calorie restriction, increased activity (that is, regular exercise), and a supportive programme of behavioural modification to assist patients in remodelling their eating habits and style[2].

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European Heart Journal (2001) **22,** 11–14
doi:10.1053/euhj.2000.2226, available online at http://www.idealibrary.com on IDEAL®

The interrelation between carotid, femoral and coronary artery disease

See page 62 for the article to which this Editorial refers

Atherosclerosis and thrombosis are the main pathological processes involved in ischaemic stroke, coronary heart disease and peripheral arterial disease. It has long been known from pathological studies that atherosclerosis is a systemic disease[1]. Although certain areas of the vasculature are particularly prone to atherothrombotic disease, it is relatively unusual for individuals to have disease localized solely to one area. Moreover, clinical manifestations of disease in one arterial territory are strongly predictive of clinical events in other territories[2–5]. For example, patients presenting with cerebral ischaemic events have a 5-year risk of myocardial infarction of 10–25% and a 5-year risk of non-stroke vascular death of 10–15%[3–5]. These risks are 5–10 times higher than general population controls. Even the risk of recurrent events in the same vascular territory is increased in those patients with symptomatic disease in other territories[3,5].

Recent developments in non-invasive imaging have allowed the prevalence of disease at multiple sites (usually the carotid, femoral and coronary arteries) to be measured in large numbers of individuals during life. This has led investigators to consider the clinical usefulness of measuring disease in arteries distant from the vascular territory of immediate interest. Some studies have related carotid artery disease and peripheral vascular disease[6,7], but most work has

been related to carotid disease and coronary heart disease, and there have been four main areas of research. Firstly, the prevalence and degree of intima-media thickness in the carotid arteries has been related to risk factors for cardiovascular disease[8,9] and the prevalence of symptomatic coronary heart disease[10,11] in cross-sectional studies, and to the risk of development of coronary heart disease in cohort studies of symptomatic normal subjects[12–15]. These studies have demonstrated a strong association between disease at the two sites. Secondly, the prevalence of carotid plaque and peripheral vascular disease has been assessed in cross-sectional studies of patients known to have coronary heart disease already[2,16]. Nearly 50% of patients have either clinical or imaging evidence of vascular disease in one or more other territory. Thirdly, the value of non-invasive measurements of carotid and peripheral vascular disease in the prediction of severe coronary artery disease has been assessed in cohort studies of patients with suspected ischaemic heart disease[11,17]. Finally, the value of non-invasive measurements of carotid and peripheral vascular disease in the prediction of future coronary vascular events has been assessed in cohort studies of patients known to have coronary artery disease[18].

In this issue, Held *et al.*[19] report the results of a study in which they assessed the relative predictive value of intima-media thickness and the occurrence of plaque in the carotid and femoral arteries in patients with stable angina. They report data on a subgroup of 558 of 809 patients with stable angina in which intima-media thickness, lumen diameter and the presence of atherothrombotic plaque was assessed in the carotid and femoral arteries using ultrasound. These assessments were related to the risk of myocardial infarction or coronary vascular death and the requirement for coronary revascularization during median follow-up of 3 years. They found that after adjustment for other risk factors, the degree of carotid intima-media thickness was only a weak predictor of coronary events, whereas the presence of carotid plaque was a significant predictor. Intima-media thickness and plaques in the femoral artery predicted the requirement for revascularization, but were not significant predictors of coronary events. They conclude that coronary and femoral artery disease have different prognostic significance, and that although carotid intima-media thickness is the most commonly used surrogate marker for atherosclerosis, assessment of plaque in the carotid and femoral arteries is a better predictor of coronary events in patients with stable angina.

These results are interesting, but there are several provisos. Firstly, the number of coronary outcome events during follow-up was small and there may be an element of chance in the findings. Secondly, the authors did not assess whether the relationships between intima-media thickness or plaque and the various outcomes were statistically significantly different from each other. Some of the relationships were statistically significantly different from the null hypothesis and some were not, but was there any statistically significant heterogeneity *between* the relationships? Thirdly, the results may be biased by the fact that the assessments of femoral and carotid intima-media thickness were relatively limited, whereas the assessments of plaque were more detailed. Intima-media thickness was measured in the far arterial wall only, along a 10 mm long segment of the common carotid artery just proximal to the carotid bulb and along a 15 mm long segment of the femoral artery just proximal to the bifurcation. In both arteries, the occurrence of plaques was assessed over larger sections both proximal and distal to the bifurcations. It has been shown previously that a measurement of intima-media thickness that combines both common carotid artery and internal carotid artery is more strongly associated with cardiovascular risk factors and the prevalence of cardiovascular disease than either measure alone[20]. The combined measure is also a better predictor of subsequent coronary vascular events[15]. Moreover, the combination of near-wall and far-wall measurements of common carotid intima-media thickness is more predictive of subsequent coronary events than far-wall measurements alone[15]. It is possible, therefore, that Held *et al.*[19] have under-estimated the predictive value of intima-media thickness measurements by limiting assessment to the far wall of a short section of artery.

However, the findings of Held *et al.*[19] may, in fact, be correct. The majority of studies looking at the predictive value of carotid, or other intima-media thickness measurements, have been performed in cohorts of healthy individuals without symptomatic coronary heart disease[12–15]. Fewer data are available in patients with established vascular disease[11,18]. Intima-media thickness is clearly a useful measure of vascular pathology and hence future coronary disease in individuals with a relatively low prevalence of vascular disease, but it is likely to be of less prognostic value in cohorts of higher risk individuals all of whom already have established vascular disease. It is quite plausible that the frequency of atheromatous plaque might be more informative in such individuals or in other groups in whom the prevalence of vascular disease is higher. For example, it has been shown that Doppler ultrasound assessments of carotid plaque are highly predictive of clinically significant coronary stenosis on angiography in

asymptomatic hypercholesterolaemic individuals with a positive exercise ECG[17].

The observation that the presence of plaque in the carotid and femoral arteries might have different significance is less easy to explain. Having said that, the pathology of atheromatous plaques does vary depending on the vascular bed. For example, plaque instability and rupture are most common in severely stenosing lesions in the carotid artery[21], but are associated with less severe disease in the coronary arteries[22]. Further studies are required to improve our understanding of the differences in behaviour of atheromatous plaques in different vascular territories, and to determine how best to quantify atheromatous plaque. Most studies have simply used the maximum plaque thickness. However, it has recently been demonstrated in patients with established vascular disease that total plaque cross-sectional area measured on longitudinal views of the common, internal and external carotids using B-mode ultrasound is probably a better predictor of acute coronary events that either intima-media thickness or maximum plaque thickness[23].

The occurrence of plaque in one vascular territory tells us that an individual is likely to be prone to the development of plaques in other territories. However, the risks of the major thrombotic and thromboembolic complications of atherosclerosis are related more to the stability of atheromatous plaques than to the extent of disease. Stable angina is associated with smooth fibrous coronary artery plaques, whereas unstable angina, acute myocardial infarction and sudden cardiac death are almost invariably associated with irregular or ruptured plaques[24,25]. Similarly, in patients with carotid artery atherosclerotic disease, plaque irregularity and rupture are closely associated with the occurrence of cerebral ischaemic events[26,27], and patients with irregular or ulcerated plaques on carotid angiography have a higher risk of ischaemic stroke irrespective of the degree of stenosis of the vessel lumen[21,28]. It has been suggested that systemic factors, such as infection, autoimmune disease, or genes, might be partly responsible for plaque instability and rupture[29–31].

If plaque stability is influenced by systemic factors, then plaque morphology at one site might predict plaque morphology at a distant site. This hypothesis was investigated recently in a study of 5393 carotid bifurcation angiograms from 3007 patients with a recently symptomatic carotid stenosis[3]. The study determined the extent to which plaque surface irregularity and ulceration at one site, the symptomatic carotid artery, was associated with irregularity and ulceration at a distant site, the contralateral carotid artery, and the extent to which plaque morphology at these sites was associated with previous myocardial infarction and subsequent non-stroke vascular death (due mainly to coronary artery disease). Patients with plaque surface irregularity or ulceration in the symptomatic carotid artery were twice as likely as those with smooth plaque to have irregular or ulcerated plaque in the contralateral carotid artery. Patients with irregular or ulcerated plaques in one or both carotid arteries were more likely to have had a previous myocardial infarction than patients with smooth plaques and were more likely to suffer a non-stroke vascular death on follow-up. However, there was no difference in the risk of non-vascular death. These associations were not explicable on the basis of differences in traditional vascular risk factors. These findings, which have been confirmed in a second similar study[32], suggest that some individuals have a systemic predisposition to irregularity and rupture of atherosclerotic plaques that is independent of traditional vascular risk factors.

In conclusion, it is increasingly clear that imaging of the type and extent of vascular disease in peripheral large arteries, particularly the carotid bifurcation, provides information that is highly predictive of the risk of acute coronary events. More work is required in order to determine exactly which measures of vascular disease are of most predictive value in which populations of patients. This information will help to target the investigation of coronary artery disease in low risk populations and the prevention of coronary events in high risk populations.

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European Heart Journal (2001) **22,** 14–16
doi:10.1053/euhj.2000.2266, available online at http://www.idealibrary.com on IDEAL®

Ablation or modification of slow pathway: the final word has still to be uttered

See page 82 for the article to which this Editorial refers

Atrioventricular nodal reentrant tachycardia is the most frequent of paroxysmal supraventricular tachycardias. Several studies have reported the efficacy of radiofrequency catheter ablation in the treatment of this arrhythmia and have encouraged its use in clinical practice[1–3].

Understanding the mechanisms responsible for a disease and the development of techniques for its