Athero-embolism occurs in all vascular beds both as a consequence of natural disease processes and in the course of invasive diagnosis and treatment. As a profession, we have tended to underestimate or minimize the incidence of this problem particularly in relation to invasive diagnostic and interventional therapies. This may be partly due to the fact that not all episodes of athero-embolization lead to readily recognized clinical events, causing difficulties in diagnosis when a high index of suspicion is not maintained. Symptoms and signs may be multiple and varied, often mimicking a systemic disease, with a definitive diagnosis being dependent on the histopathological demonstration of cholesterol crystals in tissue biopsies. Clinical presentation will vary dependent upon the sensitivity of the involved end target organ and the actual particle load that impacts at this site over a period of time. Cerebral events will often be immediately apparent and can usually be readily recognized and characterized; renal embolism, on the other hand, usually presents late with recognition often being delayed until rising levels of blood urea and creatinine are seen with biochemical testing. Recently, there has been a renewed interest, usually of a focal nature, in this problem due to the availability of distal protection devices for use in the brain, heart and kidney. This interest has occurred although many reported single institution or grouped series of procedures such as carotid stent angioplasty have suggested that the clinical consequences of such events are very low. If athero-embolism of clinical significance is therefore so uncommon in the most sensitive of all the vascular structures in which we intervene, need we worry about the use of these devices in order to better what are already reportedly good results?

**Background.** Embolization of vascular beds by material comprised principally of cholesterol crystals has often been regarded as a rare event; there is, however, an increasing body of evidence to suggest that this may be less common than previously reported. Two modes of presentation (with some overlap) can be recognized: a polymorphic form resulting from disruption of aortic plaque and a local form confined to a single vascular site. Both types may be spontaneous or iatrogenic in nature, with the latter increasing in frequency, and the triggering factors often being arteriography, angioplasty, surgery, anticoagulation (including low-molecular-weight heparin) or fibrinolytic therapy. Multi-organ embolization is often the most difficult to diagnose, with clinical presentation often occurring at a time remote from the triggering event or events. Episodes of embolization need not be singular, but rather may be multiple and accumulative in terms of effect. Disruption of aortic atherosclerotic plaque is the most common precipitating event, with the vascular beds at risk being dependent upon their relationship to the left subclavian artery; disrupted plaque distal to this area will not cause cerebral or ocular symptoms. Plaque disruption in the ascending thoracic aorta has the potential to not only embolize the brain but also all other distal organs. Clinical features can be misleading with a pseudo “poly-arteritis nodosa” presentation being common; however, more focal problems such as isolated gangrene of the gall bladder, stomach, large and small intestine have also occurred. Most frequently, noted features are transient eosinophilia (about 80%), cutaneous signs (present in up to 70%) and acute renal failure (approximately 30%). Elderly males account for the majority of patients; mortality in this group approaches 80%. Demonstration of cholesterol crystals in skin, muscle or renal biopsies gives confirmatory diagnosis.
Recommended treatment regimes are often contradictory with general systemic support appearing to be the best option.

Local forms of embolization are more easily recognized and include acute myocardial infarction, stroke, transient ischemic attack, amaurosis fugax, renal infarction and “blue toe” syndrome.

The above is a somewhat simplified classification and not uncommonly the pleomorphic form may initially present with predominantly local signs and symptoms. While aortic plaque disruption is the least common of the two, it is arguably more commonly induced by invasive medical procedures. Local atheroembolism also accompanies attempts at diagnosis and intervention. Diagnostic carotid imaging studies supposedly have a low rate of cerebral events, yet audited stroke rates of up to 4% per annum have been reported in relation to carotid angiography alone. This has led to an increasing use of less invasive diagnostic studies, with computerized tomographic angiography, magnetic resonance angiography and duplex ultrasound replacing traditional arteriography in many non-cardiac vascular applications.

Current issues: Carotid angioplasty and carotid surgery. Two randomized studies — the North American Symptomatic Carotid Endarterectomy Trial (NASCET, 1991) and the European Carotid Surgery Trial (ECST, 1991) — demonstrated the superiority of surgery compared to medical treatment in patients with symptomatic carotid artery stenosis showing 70% or greater diameter reduction.

Spontaneous events, while common, seem to attract less attention than iatrogenic incidents since these are readily related to specific attempts at treatment. Transient and fixed cerebral events are well recognized in relation to surgical and endovascular interventions for carotid artery stenosis. Single-center studies in relation to both types of treatment report a low incidence of fixed neurological deficit; however, in general such results have not to date been supported by randomized independently audited trials comparing these modes of therapy. The only completed trial comparing carotid surgery with angioplasty at the time of writing is CAVATAS; the comparative stroke rates were 9.9% for the surgically treated group and 10.0% for the interventional group. Notably, only 26% of the angioplasty group were stented. Needless to say, it would appear that room for improvement exists in relation to both modes of treatment.

There is little doubt that the less invasive methods of therapy employed in the treatment of cardiovascular diseases have a place in treatment. Despite some limitations, they offer an alternative to traditional surgical procedures; in many instances, they may be superior to surgery. Surgery and interventional therapy are mutually compatible and need not be mutually exclusive; our current concerns should focus upon making both forms of treatment safer for patients.

Following both cardiac and peripheral stenting, observation or measurement of deterioration in target organ function may occur despite an excellent anatomical result at the site of the treated lesion. Electrophysiological mapping of ventricular function following successful coronary stenting may show decreased electrical and mechanical activity in the area perfused by the treated vessel, suggesting an adverse rather than a positive functional outcome. Whether this represents cholesterol embolization, platelet aggregation or both remains unclear; the effect seems to be largely abolished with the use of a IIb/IIIa platelet inhibitor in association with coronary stenting, suggesting that platelets have a role to play. The potential contribution of atheromatous particulate matter is less clear.

Problems associated with treatments of degraded coronary artery saphenous vein grafts are well recognized, with distal embolization of atheroma, thrombus and other material being a complication of angioplasty. Deterioration of renal function also occurs following successful renal artery stenting. In this instance, the cause can be more readily proven since renal biopsy will demonstrate the presence of cholesterol crystals in tissue samples; however, renal contrast nephro-toxicity may also contribute to the problem of deteriorated renal function. Current biochemical measurements of renal function lack sensitivity, tending to become abnormal only when there is a significant loss of functioning renal parenchyma. Since many patients undergoing this procedure are elderly and therefore probably possess little renal reserve, the embolic load need not be large.

Controversy in relation to angioplasty and stenting for atherosclerotic stenosis of the internal carotid artery is unlikely to be resolved in the short term. In terms of an unfavorable outcome in relation to atheroembolism, this remains the most sensitive end organ currently treated. The major proponents of stenting are likely to be cardiologists with the strongest opposition being voiced by vascular surgeons since, in most instances, each alone can currently offer but one mode of treatment. Under such circumstances, controversy in relation to stenting versus endarterectomy is likely to continue for some time to come. Distal protection devices have the potential to reduce adverse events.

Angioplasty of degraded saphenous vein grafts, renal artery and carotid artery stenoses are the sites where distal protection devices are likely to prove beneficial in limiting end organ damage.

Low-profile devices, such as occlusion balloons, are more readily placed across severely stenosed lesions.
without the need for preliminary angioplasty, but suffer from the drawback that not all patients will tolerate internal carotid artery occlusion. A further potential drawback is that the small caliber monorail suction devices used to aspirate atheromatous debris may not adequately capture larger particles.

Filters that allow continuing cerebral perfusion are attractive in theory but may prove problematic in practice. These devices, when compared with balloons, are both higher in profile and more rigid. Preliminary angioplasty with a small-profile balloon may be necessary in order to pass the protection device beyond the lesion; this results not only in the need to cross the lesion twice, but also in losing guidewire access after the initial angioplasty. Prior to deployment, filters are quite rigid and thus may be difficult to pass through tortuous or kinked vessels. Manipulations necessary to gain distal access may be traumatic to the artery endothelium, thereby risking distal embolization. Filters are effective once deployed, but on occasion may be occluded due to the amount of material captured; this event may result in significant cerebral irritability or loss of consciousness. Fortunately, short periods of vessel occlusion do not necessarily relate to irreversible neurological deficits. Improvements in the design of distal protection devices are likely to continue making them both safer and easy to deploy.

**Conclusion.** Non-interventionalists often express concern that there exists a high likelihood of distal embolization with all interventional procedures. While such concerns are often ill-founded, they should not be ignored since the possibility does exist in any invasive diagnostic or interventional treatment. An awareness of this risk is often the best protection, particularly in regard to possible aortic plaque rupture. When possible, non-invasive imaging should be used for diagnosis. Excessive catheter manipulation needs to be avoided particularly in the aortic arch. Distal protection devices are likely to proliferate in both type and use, and appropriate application during interventional treatment is likely to reduce adverse events due to athero-embolism in vulnerable vascular structures.