The value of continuous ST segment monitoring in patients with unstable angina

See pages 1991 and 1997 for the articles to which this Editorial refers

Approximately 75% of all episodes of transient myocardial ischaemia (usually identified by ST segment depression) in patients with stable and unstable angina are silent[1]. Despite the lack of symptoms, the importance of detecting transient episodes of myocardial ischaemia is obvious to most cardiologists. But to others, the value is not so clear and must be continually re-proven. To those of us with long-standing investigative interests in silent myocardial ischaemia, this is not a new challenge. Historically, there was an initial need to authenticate the existence of the syndrome itself, either by exercise testing or by Holter monitoring, the latter serving to reliably record the numerous episodes of transient ischaemia that occur during daily life, thereby accurately estimating its prevalence. Second, it was important to document the prognostic importance of these episodes in asymptomatic individuals and, more importantly, in patients with known coronary artery disease and either stable or unstable angina pectoris. Third, the effect of antiischaemic agents and revascularization procedures in reducing the frequency of these episodes had to be established and their role in improving subsequent morbidity and mortality proven. The latter aspect of this saga is still ongoing, but it is the prognostic value with which two articles in the current issue are concerned.

In the United States, the pioneering studies of Gottlieb et al. in 1986[2] and 1987[3] first documented the adverse progress associated with transient ischaemia in patients with unstable angina. In their studies, 70 patients admitted with unstable angina were followed first for 30 days[2] and then for 2 years[3]. All patients received appropriate medical therapy for that era and their symptoms usually subsided, but Holter monitoring performed before discharge revealed two distinct prognostic patterns. For example, at the 2 year mark, 21 of the 37 patients (57%) with transient ischaemia on Holter monitoring had either cardiac death, a non-fatal myocardial infarction or a coronary revascularization procedure, compared to six of 33 patients (18% P<01) without evidence of transient ischaemia. These results were confirmed in a subsequent study in 1987 by Nademanee et al.[4], who also pointed out the added prognostic importance of having more than 60 min of ischaemia per 24 h of Holter recordings. In Europe, Arnim et al., in 1988[5] reported similar adverse prognostic implications of transient ischaemia detected by Holter monitoring.

In the current issue, studies from the United Kingdom and from The Netherlands provide new data to confirm the importance of detecting transient ischaemia in unstable angina patients and to possibly explain its pathophysiological basis. The report by Akkerhuis et al.[6] is a very ambitious project, a meta-analysis of three studies involving nearly 1000 patients. The authors used data obtained from CAPTURE, PURSUIT, and FROST, three trials that evaluated antiplatelet agents in patients with unstable angina. Patients were monitored for 24 h with 12 lead ECG or VCG monitoring devices that computed ST segment shifts. The prior unstable angina studies cited — as well as more recent ones — had relatively small numbers of patients and were unable to demonstrate a direct relationship between number of ischaemic episodes and increased risk of cardiovascular events. In the meta-analysis, transient ischaemic episodes were found in 27% of the patients, a smaller number than in the studies from the 1980s, but this time a direct relationship could be established. The authors concluded that the ‘relative risk of death or myocardial infarction at 5 and 30 days increased by 25% for each additional ischaemia episode per 24 h’.

While the prognostic importance of continuous 12-lead ECG evidence of ischaemia is emphasized by this report, thus confirming results of smaller studies performed by previous investigators with Holter systems, the pathophysiological basis for the poor prognosis was not addressed. Is it the ischaemia per se, or is the ischaemia merely a marker for certain types of coronary vascular lesions? This is where the second study by Patel et al.[7] from the United Kingdom is helpful. This prospective observational study of 101 patients with refractory unstable angina involved evaluation by Holter monitoring in a London teaching hospital prior to cardiac catheterization. Unlike recurrent chest pain, which was often atypical and not associated with ECG changes, ST segment depression (indicating transient and usually silent myocardial ischaemia) was a powerful predictor of complex coronary lesion morphology or intracoronary thrombus formation when subsequent coronary arteriography was performed. The authors
postulated that this association may explain why recurrent transient ischaemia predicts a poor outcome in unstable angina patients who appear to be well treated with antiischaemic agents.

Both sets of authors urge that intensive therapeutic approaches be considered in the more ischaemic patients. Specifically, Akkerhuis et al. suggest that continuous on-line ST segment monitoring can influence the triage and risk stratification of patients with unstable angina. Enhanced antithrombotic treatment and revascularization can then be used in those patients with persistent episodes of transient ischaemia who were initially thought to be at low risk for future cardiac events. Patel et al. also favour using revascularization procedures in their ‘refractory’ patients. The continuing instability of the vascular endothelium is a source of ongoing concern and probably warrants other approaches as well. I am thinking specifically of the decrease in transient ischaemia (and subsequent cardiac events) reported with statin therapy in patients with stable angina.

This class of drugs makes the endothelium less vulnerable to the kinds of lesions associated with increased cardiovascular morbidity and mortality and may be especially valuable when a full regimen of ‘traditional’ antiischaemic agents (nitrates, beta-blockers, calcium blockers, antiplatelet drugs) are already being employed. There is preliminary data that ACE inhibitors may also be helpful. In the final analysis, however, it may be that coronary revascularization — and especially bypass surgery — offers the best prognosis for patients with ongoing ischaemia, whether stable or unstable, silent or symptomatic.

References


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All stents are not alike or is the difference in the eye of the observer only?

See pages 2007 and 2015 for the articles to which this Editorial refers

Coronary artery stents have emerged as the preferred tool for percutaneous coronary interventions during the past decade. Their ubiquitous acceptance results from the ease and speed of applicability in an ever-broader array of anatomical lesions, the improved safety by elimination of abrupt closure and need for urgent coronary artery bypass grafting (CABG), and the angiographically pleasing result. The basic principle underlying the acute therapeutic value is common to all coronary artery stents and consists of (1) increasing the arterial lumen by scaffolding...