counterpulsation. The authors also measured atrial and brain natriuretic peptide levels. Unlike the Masuda et al. study, atrial natriuretic peptide levels were unchanged, but brain natriuretic peptide levels decreased after enhanced external counterpulsation treatment as they did in the study of Masuda et al. The authors concluded that enhanced external counterpulsation improves myocardial perfusion, exercise performance and left ventricular diastolic filling and decreases cardiac workload, providing yet more data to explain the mechanisms by which enhanced external counterpulsation achieves its antiischaemic benefits.

In summary, results from the Stony Book experience and the MUST-EECP trial clearly demonstrate the beneficial haemodynamic myocardial effects of external counterpulsation in patients with chronic refractory angina. Masuda et al. shed light on possible mechanisms responsible for these effects. In my opinion, this procedure should be considered as part of the contemporary clinician’s therapeutic armamentarium in selected patients, as recommended by Conti in his editorial accompanying the initial MUST-EECP report and by Lange and Hillis in their editorial questioning the role of transmyocardial laser revascularization. Lange and Hillis recommend that safer and less invasive therapies (such as enhanced external counterpulsation and neurostimulation) be the procedures of first choice in refractory chronic angina patients, rather than transmyocardial laser revascularization, and this seems an eminently sensible approach, especially in light of the new data provided by Masuda et al.

P. F. COHN
SUNY Health Sciences Center, Stony Brook, NY, U.S.A.

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have truly normal studies that documented widely patent coronary arteries with smooth luminal outlines. A number of hypotheses have been suggested to explain the phenomenon of myocardial infarction with normal coronary arteries. In some patients, coronary arterial vasospasm was documented or theorized. In others, intravascular ultrasound studies documented atherosclerotic plaques which failed to encroach on the lumen of the involved coronary artery. In these patients, it was hypothesized that one of these small, intramural atherosclerotic lesions had ruptured, thereby leading to coronary arterial thrombosis that subsequently lysed leaving the normal lumen intact. Other possible explanations for myocardial infarction with normal coronary arteries included small vessel disease, e.g. various collagen vascular diseases, hypercoagulable states, poor angiographic technique so that a significant coronary arterial stenosis was missed, and coronary arterial embolism, for example, following an episode of paradoxical embolism.

In the article published in this issue, DaCosta and co-workers observed that only a minority of patients were shown to have coronary arterial vasospasm (15-5%), a hypercoagulable state secondary to a congenital condition or oral contraceptives (13-9%), collagen vascular disease with presumptive small vessel disease (2-2%), or coronary arterial embolism (2-2%). Thus, no obvious etiology for myocardial infarction with normal coronary arteries was discernable in 66-2% of their patient population with this condition. Presumably, many of the remaining patients had had either a small atherosclerotic plaque that ruptured, or an as yet unknown cause for their transient coronary arterial obstruction. Of course, it is always possible that some of these latter patients had an atherosclerotic coronary arterial stenosis that was missed at catheterization. However, this seems unlikely to be the case for more than a rare individual. Furthermore, it is possible that additional patients had had an isolated episode of coronary arterial spasm for unknown reasons. Supporting the coronary arterial vasospasm hypothesis is the series of 21 patients with myocardial infarction with normal coronary arteries reported by Ammann et al. in which migraine syndromes were commonly reported. Also of interest in this series was an increased incidence of seropositivity for cytomegalovirus, Chlamydia pneumoniae, and Helicobacter pylori in patients with myocardial infarction with normal coronary arteries.

Similar to earlier workers, DaCosta et al. noted that patients with myocardial infarction with normal coronary arteries had fewer atherosclerotic risk factors and a better long-term prognosis when compared with infarct patients with obvious atherosclerotic coronary arterial lesions. These investigators also found that decreased left ventricular function and diabetes mellitus were adverse prognostic factors in patients with myocardial infarction with normal coronary arteries. Other observers have noted that patients with the condition tend to be younger, usually less than age 50, as compared with atherosclerotic myocardial infarction patients. In general, both genders are affected equally although DaCosta et al. observed more men with this condition in their series. Despite the lower prevalence of atherosclerotic risk factors in patients with myocardial infarction with normal coronary arteries as compared with infarct patients with evident atherosclerosis, it is my practice to reduce aggressively whatever risk factors do exist in myocardial infarction with normal coronary arteries patients, on the assumption that either atherosclerosis or one of the risk factors themselves has played a role in the genesis of the myocardial infarction.

Q wave and non-Q wave infarctions have both been reported in patients with normal coronary arteries. Sharifi et al. noted a bimodal age and gender distribution in a series of 12 patients (1% of their post myocardial infarction population) with myocardial infarction with normal coronary arteries. Younger patients were predominantly men with a strong smoking history and a tendency to develop Q wave infarction. Older patients tended to be women who smoked less and who tended to develop non-Q wave infarcts. It seems likely to me that the younger male population in this series had ruptured an intramural coronary arterial atherosclerotic plaque while the older female population had suffered an episode of transient coronary arterial vasospasm. Another interesting series of patients with myocardial infarction with normal coronary arteries was reported by Lip et al. who described six patients with recurrent myocardial infarction and angina with normal coronary arteriographic studies. Thus, whatever the etiology might be, it is possible for ‘lightning to strike twice’ in at least an occasional individual.

In conclusion, approximately 1% of patients who suffer an acute myocardial infarction will be subsequently found to have angiographically normal coronary arteries. These patients tend to be younger than routine atherosclerotic infarct patients with more moderate levels of atherosclerotic risk factors. Myocardial infarction patients with normal coronary arteries generally have an excellent prognosis. Nevertheless, I personally treat these patients...
aggressively with beta-blockers, aspirin, and hypo-
lipidaemic agents (usually a statin). If coronary
arterial spasm seems a likely aetiological agent in
such a patient, I also prescribe a vasodilator, i.e. a
calcium channel blocker, an ACE inhibitor, or an
angiotensin receptor blocker. These patients represent
a very interesting subset of individuals with myocar-
dial infarction. I am convinced that they will continue
to be the subject of clinical investigation in the future
in an effort to understand the pathophysiological
process that results in their transient episode of
coronary arterial obstruction.

J. S. ALPERT
Department of Medicine,
University of Arizona Health Science Center,
Tucson, Arizona,
U.S.A.

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Economics of coronary stenting and GPIIb/IIIa blockade

See page 1476 for the article to which this Editorial
refers

Multiple studies have shown that the use of GPIIb/
IIIa blockade can reduce cardiovascular events in the
setting of percutaneous coronary intervention[1–3].
The EPISTENT trial was particularly interesting and
timely[4]. In EPISTENT, 2399 patients undergoing
coronary revascularization were randomized to three
arms: stent plus placebo, stent plus abciximab, and
PTCA plus abciximab. The primary end-point
was the composite at 30 days of death, myo-
cardial infarction, or coronary ischaemia requiring
urgent revascularization. At 30 days and 6 months,
major adverse cardiac event-free survival was highest
in the stent plus abciximab group. Abciximab re-
sulted largely in a decrease in acute myocardial
infarction within 1 month while stents decreased
additional revascularization at 6 months. In diabetics,
abciximab reduced revascularization rates as well.

In the current issue, Zwart-van Rijkom and van
Hout extend the results of the EPISTENT trial to
include an economic analysis[5]. EPISTENT was con-
ducted in the United States and Canada, but the
economic study presented in this issue was based on
Dutch unit costs, priced in 1998 Euros. Costs at
6 months were highest in the stent plus abciximab
group and lowest in the PTCA plus abciximab group.
Cost effectiveness was evaluated as the incremental
cost per event averted by adding abciximab to stent
plus placebo and as the incremental cost per event
averted by adding a stent to PTCA plus abciximab.
The cost per event averted of abciximab was Euros
14 198 and of a stent Euros 2167 and of a stent Euros 8040. Due to uncertainty in the
cost and efficacy measures, abciximab may be cost
saving in diabetics. Studying the subgroup with
diabetes shows that the cost-effectiveness of therapy
can be much greater in higher risk subgroups and that
therapy can then be targeted to such subgroups[6].

The study by Zwart-van Rijkom and van Hout is
limited by the design of the EPISTENT study, as the
investigators well recognize. The use of clinical data
from one geographical area for use in another geo-
ographical area for an economic study assumes that
the patterns of care are similar. Often in economic
studies there are either no local or insufficient local
data to conduct the economic study, such that data
from other countries are all that is available. Where
there is a local data subset, this subset should be
compared to the non-local data for the patterns of

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