patients exhibit a constrictor response in distal coronary vessels so enhanced that it causes myocardial ischaemia at rest, remain unknown.

Third, even a careful, sophisticated analysis of the vasoconstrictor response at the site of angioplasty failed to differentiate restenosed (>50%) from non-restenosed coronary lesions submitted to percutaneous transluminal coronary balloon angioplasty. Yet this differentiation is demanded urgently, as demonstrated by growing excitement about the possibility that the risk of restenosis can be reduced by the application of stents or by the use of special catheter systems capable of delivering locally 'anti-restenosis' agents.

As the authors state at the beginning of their article, from 50 to 70% of dilated lesions do not exhibit flow-limiting restenosis. Progressively shrinking health care budgets worldwide prevent the cardiological community from proposing the use of costly techniques to reduce further the risk of restenosis in all treated lesions, including the 50 to 70% that would not restenose following standard percutaneous transluminal coronary balloon angioplasty.

So far, the explanation for the cause of restenosis has not come from animal studies as they consistently produce a similar local vascular proliferative response in all lesions, largely related to the severity of the injury. In such models inhibition of proliferation actually reduces the extent of the physiological repair process to vascular trauma. In patients the trauma of percutaneous transluminal coronary balloon angioplasty does not appear to be a sufficient explanation for the development of flow-limiting restenosis because, as stated above, this unfavourable outcome does not occur in as many as 50 to 70% of cases. Although all stenoses seem to develop at least some degree of restenosis[4], flow-limiting restenosis is likely to be due to mechanisms present locally only in some patients and only in a minority of lesions as, in multivessel percutaneous transluminal coronary balloon angioplasty, only some lesions restenose, particularly those responsible for instability[5]. Therefore, the identification of those lesions likely to develop flow-limiting restenosis (and hence deserving additional procedures) is more likely to come from clinical research than from animal studies.

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References

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Strength training may be a valuable adjunct to dynamic exercise rehabilitation

See page 854 for the article to which this Editorial refers

The article by Wosornu et al. published in this issue brings again into focus an important aspect of cardiac rehabilitation: while adequate data attest to the beneficial effects of dynamic exercise, the aspect of power training, which to a large extent includes an isometric element, has not been adequately stressed.

If during power efforts, the heart is stressed inordinately and manifests more severe ischaemic manifestations than those evidenced by dynamic exercise, then this type of training should be avoided. However, an obvious benefit of power training would
be to obviate the everyday difficulties encountered by untrained patients who sometimes have to carry significant weights during everyday life. This may represent the case of the university professor climbing the underground stairs with a heavy attaché case, more often resembling a suitcase, full of books. Moreover, because few patients are fortunate enough to devote to rehabilitation programmes more than a finite portion of their everyday life it would be important to know whether the rehabilitation time devoted to power training has adequate beneficial effects of cardiorespiratory performance and fitness.

The article satisfactorily answers the last aspect: these authors adequately showed that 6 months of power training improved exercise capacity. Here it should be noted that dynamic exercise already showed a significant increase at 3 months. However, if one looks at the baseline values, the group of patients undergoing power training had higher baseline values, which may have rendered changes more difficult to achieve, especially in the face of the small numbers of patients in the three groups. In any case, this group also achieved a significant gain in fitness.

The training sessions were not monitored so as to detect any untoward changes during power training. However, this question has been addressed before. De Busk et al. showed, 17 years ago, that ischaemic ST depression and ventricular ectopy were more common with dynamic than with static exercise in patients aged 34 to 63 years soon after a myocardial infarction. Kelemen et al. also commented on a lack of significant cardiac arrhythmias during circuit weight training, while blood pressure immediately after exercise was only slightly higher after power than after dynamic training.

Thus, by any criterion, power training of a logical intensity is not dangerous for the heart. The authors did not measure haemodynamic responses during the training sessions, but Kelemen et al. have pointed out that power exercise produces a lower double product. We have shown that the emergence of ischaemia very closely depends on the double product.

A last aspect concerns the actual benefit to be expected from power training as regards the reason for which it is employed, i.e. improvement of upper body strength. McCartney et al. have shown that arm curl strength can increase by 42% and leg strength by 21 to 25%, while repetition endurance can increase by more than three-fold. These are substantial gains and are similar to those observed by other authors; thus, Kelemen et al. recorded a 24% gain, while Hiatt et al. who subjected patients with intermittent claudication to strength leg training versus dynamic exercise, noted an increase of 28% of the gastrocnemius of the more diseased legs but a 31.5% increase in the less diseased legs. These strength gains could be significant in improving static load tolerance in everyday life. It is not known, however, whether they would subject the heart of untrained individuals to a lesser strain. In a study where both arms and legs were trained concurrently by endurance training, peak power output increased 24% in the arms and 23% in the legs, while maximal oxygen consumption increased 13% during arm and 11% during leg ergometry.

A point worth mentioning is that skin-fold thickness decreased significantly only with power training. Dynamic exercise has traditionally been shown to decrease subcutaneous fat accumulation, a problem especially concerning older patients. If the combination of dynamic and strength training is shown to produce a greater decrease of subcutaneous fat than either modality alone, this might be an additional welcome effect in this age of great importance of the gracious form.

A final comment should be made on the biochemical and haemostatic profile results. The authors found no changes in lipid levels. Perhaps the sample numbers were too small, or the exercise intensity level may have been too low. However, a new aspect should give some pause for thought: too strenuous exercise on a bicycle ergometer may sensitize platelet adhesiveness and aggregability, while moderate exercise has a suppressant effect. Previous work from the same authors only addressed fibrinogen and factor VII concentrations. Thus, further observations on platelet behaviour with power training could be of potential interest.

Of course, comparisons of purely dynamic or exclusively strength training regimes are academic. It is natural to expect that an effective programme would encompass both dynamic and power exercise. As shown by McCartney et al. this is the combination effecting the greatest aerobic improvement. The current article by Wosornu et al. shows that this combination is practical.

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References
Left ventricular ejection fraction: an important but incomplete determinant of long-term outcome after coronary bypass surgery

See page 874 for the article to which this Editorial refers

In this issue of the journal, Risum et al. report that pre-operative left ventricular ejection fraction, in particular, left ventricular ejection fraction <40%, is associated with an increased risk of early and late mortality after coronary bypass surgery. In 80 patients with left ventricular ejection fraction <40% versus 854 patients with left ventricular ejection fraction >40%, 10-year mortality rates were 55 and 79%, respectively. The findings confirm earlier reports of the importance of pre-operative left ventricular function as a determinant of early and long-term outcome after coronary bypass surgery and highlight the concept that the mortality risk continuum augments significantly when left ventricular function is severely impaired. In the Coronary Artery Surgery Study (CASS) registry, the average operative mortality was 2.3% and ranged from 1-9% in patients with left ventricular ejection fraction ≥50% to 6-7% for patients with left ventricular ejection fraction <19% in 6630 patients who underwent isolated coronary bypass surgery. Operative mortality was increased in older patients, those with congestive heart failure, left main coronary disease, and in patients who required an emergency procedure.

Long-term outcome is affected by a multitude of factors in addition to pre-operative left ventricular function that were not examined in the Risum study. Peri-operative myocardial infarction, incomplete revascularization, and low use of internal mammary artery conduits (only 12% of patients in the Risum study received an arterial conduit) are known to affect late mortality adversely. Saphenous vein graft atherosclerosis increases 7 or more years following coronary bypass surgery, just around the average 7-4 year duration of follow-up in this report. Mortality rates are also influenced by the coronary atherosclerotic burden manifest by disease extent. Although coronary disease extent was similar in patients with a left ventricular ejection fraction <40 or >40 in the Risum report, in the CASS trial, 15-year survival rates were 66 and 69% in men and women who had single-vessel disease compared to 45 and 32% when three-vessel coronary disease was present in an 8213-patient series involving coronary bypass surgery. The 15-year survival rate was only 32% for men and 21% for women in patients with three-vessel disease and moderate-to-severe left ventricular dysfunction (left ventricular contraction score >10(3)). Other variables known to impact morbidity and mortality adversely after coronary bypass surgery include older age, persistent smoking, and uncontrolled hypercholesterolaemia.

Risum et al. did not find that pre-operative left ventricular ejection fraction predicts recurrent angina or late non-fatal myocardial infarction. In CASS, angina recurrence was noted in 40% of patients 6 years following coronary bypass surgery. Significant multivariate predictors of angina recurrence included the presence of minimal coronary disease, peri-operative angina, use of only saphenous vein grafts, previous myocardial infarction, incomplete revascularization, female gender, persistent smoking,