Reduction in plasma concentrations of N terminal pro B type natriuretic peptide following percutaneous coronary intervention

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Baseline NTproBNP concentration was 257 (170–771) fmol/ml in the PCI group and 188 (106–550) fmol/ml in the control group (p = 0.5). In the PCI group plasma NTproBNP concentrations decreased over time (analysis of variance (ANOVA) p < 0.0001). Concentrations at 1 hour (281 (192–721) fmol/ml) and 6 hours (246 (137–688) fmol/ml) were similar to baseline (both p > 0.1), but a significant reduction was seen by 24 hours (207 (53–588) fmol/ml, p = 0.006 v baseline). A further reduction in NTproBNP was seen at 6 months (145 (66–369) fmol/ml, p = 0.0003 v baseline).

In the control group NTproBNP concentrations increased between 1 and 6 hours following angiography (232 (138–551) v 271 (177–726) fmol/ml, respectively, p = 0.036). In contrast, baseline concentrations were similar to those at 24 hours (188 (106–550) v 197 (115–344) fmol/ml, respectively, p = 0.24). Data were not available for three of the patients at day 7. In the remaining 10 patients NTproBNP concentrations did not change significantly between baseline and 7 days (210 (109–547) v 300 (140–596) fmol/ml, p = 0.15). In the patients whose baseline NTproBNP was not raised (14 (14–26) fmol/ml), plasma concentrations did not change over time (ANOVA p = 0.22). These results were not materially altered by sensitivity analysis using the 75th or 95th centiles as the threshold for high NTproBNP.

Abbreviations: BNP, B type natriuretic peptide; PCI, percutaneous coronary intervention; NTproBNP, N terminal pro B type natriuretic peptide
DISCUSSION
Previous studies with patients without raised BNP concentrations at baseline have shown a transient increase in BNP following PCI, thought to be related to myocardial ischaemia during balloon inflation. In contrast, we have found that in patients with raised NTproBNP at baseline, PCI was associated with a decrease in concentrations within 24 hours. This reduction is maintained, or even augmented, at six months.

The degree to which the reduction in NTproBNP is determined by improved left ventricular function and geometry, or by a reduction in ischaemic burden, remains to be established. Plasma NTproBNP concentrations are strongly related to left ventricular function in patients with recent myocardial infarction, but it has also been demonstrated that NTproBNP is elevated in patients with stable angina, even with preserved ventricular function, compared to healthy controls, suggesting that chronic or recurrent acute ischaemia may contribute to raised concentrations of NT proBNP. The absence of consistent sequential assessment of ventricular function is a limitation of this study.

While PCI is an effective means of alleviating symptoms in stable coronary disease, data supporting consistent mortality benefits are lacking. The finding that PCI can reduce raised plasma NTproBNP concentration may have important clinical implications. Raised plasma BNP and NTproBNP predict adverse prognosis in a number of clinical states, and it has been suggested that the assessment of BNP may identify high-risk patients with acute coronary syndromes who could benefit from early aggressive intervention. In heart failure, treatment titrated to reduce plasma NTproBNP improves clinical outcome compared to standard clinical practice. We have demonstrated a significant and sustained reduction in NTproBNP in patients undergoing PCI who had raised concentrations before the intervention. Given the wealth of evidence supporting NTproBNP as a prognostic indicator, and emerging evidence that measures which reduce NTproBNP are associated with improved clinical outcome, it is tempting to speculate that a reduction in plasma NTproBNP concentrations towards the normal range following revascularisation might indicate a prognostic benefit.

This study shows that raised NTproBNP concentrations in patients with stable angina may be reduced by PCI. It was not designed to elucidate the prognostic importance of the reduction, or the pathophysiological mechanisms behind it. The findings are both exciting and challenging, with further evaluation required to determine whether this primarily relates to a reduction in ischaemic burden or changes in left ventricular function, although these are tightly interrelated. The effect on outcome of the reduction of NTproBNP and the potential to use it to target the use of PCI needs to be addressed in larger prospectively collected populations.

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Accepted 23 January 2004

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