Comparison of Intracoronary Bolus Injection and Intracoronary Continuous Infusion Methods for inducing Hyperemia

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Background: The method to induce coronary hyperemia with maximal and steady state is important to evaluate microvascular circulation by coronary flow reserve (CFR) and fraction flow reserve (FFR). We studied the method of inducing hyperemia with intracoronary (IC) adenosine bolus injection and continuous infusion.

Methods: CFR was evaluated in thirty-six coronary lesions (LAD 23, LCX 5, RCA 8) in 23 patients (male 18, mean 58+12; SA 2, UA 12, AMI 9) and FFR was evaluated in twenty lesions (LAD 13, LCX 6, RCA 1) in 10 patients (male, 8, mean 60+11; YA 1, SA 7, AMI 2). To induce maximal coronary hyperemia, two methods were applied to same patient: Method A (IC adenosine bolus injection (RCA 24µg, LCA 48µg)) and Method B (IC adenosine continuous infusion (240µg/min)). CFR was defined as a ratio of hyperemic (hAPV) to baseline APV (bAPV). FFR was defined as a ratio of mean aortic pressure to mean coronary pressure of distal lesion during maximal hyperemia.

Results: All patients were tolerable during maximal hyperemic period. During maximal coronary hyperemia, hAPV and bAPV were different between the two methods. Transient AV block occurred in 2 patients with method A and in 2 same patients with method B. The duration of maintenance of hyperemia was mean 24.6±5.4 seconds in method A and all patient except 2 patients were maintained stable hyperemia with method B during continuous infusion of adenosine. CFR and hAPV were significantly higher by using method B than those of method A (0.76±0.17 vs. 0.80±0.15, p<0.01; 52.5±23.4 vs. 45.7±19.9, p=0.01, respectively). FFR was significantly lower by using of method B than that of A (0.76±0.17 vs. 0.80±0.15, p=0.01).

Conclusion: Compared with intracoronary adenosine bolus injection method (24-48µg), intracoronary adenosine continuous infusion method (240µg/min) was more effective and stable for inducing maximal hyperemia.

T120-64 High Left Ventricular Mass Does Not Limit the Utility of Fractional Flow Reserve for the Physiologic Assessment of Lesion Severity


Background: Fractional Flow Reserve (FFR) has been shown to be a useful invasive physiologic index of coronary lesion severity. Increased left ventricular mass is known to impair microvascular reserve. However, whether increased LVM sufficiently impacts flow reserve to affect clinical FFR measurements is not known.

Methods: FFR was calculated from contrast left ventriculography in 84 patients using a modified Rackley method, which correlates well with LVM measured by cardiac MRI in 17 patients who had undergone both tests (r=0.80, p<0.001). The cohort was divided into normal and high left ventricular mass index (LVMI) groups based on published normal values. Cardiac risk factors and lesion FFR were compared in 22 vessels of patients with high LVM to 62 angiographically matched lesions in vessels of patients with normal LVMI.

Results: LVM was 84±21 g/m² and 126±21 g/m² in the normal and high LVMI groups, respectively. There were no differences in age (59±58 yrs, p=ns), presence of diabetes (20% vs 27%, p=ns), hypertension (80% vs 73%, p=ns), dyslipidemia (64% vs 64%, p=ns) or angiographic LVEF (58% vs 62%, p=ns) between groups. Importantly, in lesions with similar angiographic characteristics, there was no difference in FFR between groups. (Table).

Conclusion: FFR of lesions in patients with high LVM is no different to FFR of angiographically similar lesions in patients with normal LVM. These findings suggest that increased LVM should not limit the utility of FFR as a physiologic index of lesion severity.

T120-65 Emboli Protection Improves Thrombolysis in Myocardial Infarction Perfusion Score in Saphenous Vein Graft Intervention

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Background: Use of emboli protection devices (EPD) during saphenous vein graft percutaneous coronary intervention (SVG-PCI) has been proven to reduce major adverse cardiac events (MACE) specifically the composite of myocardial infarction, urgent target vessel revascularization, and death. However, the impact of EPD on the microcirculation using TIMI myocardial perfusion score (TMP) has not been fully characterized. We sought to analyze TMP in both unprotected- and EPD- SVG-PCI and its impact on 30-day MACE.

Methods: From August 2001 to December 2002, 305 patients had a SVG-PCI suitable for EPD; 247 (81%) had an angiogram appropriate for TMP evaluation. Of those, 49 (20%) had an EPD deployed during the coronary intervention. Both groups were similar regarding most demographic features, but use of GP IIb/IIIa inhibitors was more frequent in the EPD group (87% vs 74%; p=0.007).

Results: TMP 3 score was obtained in 87% of the EPD group vs 56% of the unprotected SVG-PCI (p<0.01) (Figure 1). MACE was 4.2% in the EPD group vs. 8.1% in the unprotected SVG-PCI group (p=0.04). Unprotected SVG-PCI patients with TMP scores lower than 3 had a trend towards increased total post-procedural CK (177 U/L vs 133 U/L, p=0.07), and CK-MB (21 ng/mL vs 6 ng/mL, p=0.07).

Conclusions: EPD SVG-PCI improves TMP score when compared to unprotected SVG-PCI. This finding was associated with a decrease in post-procedural MACE.