Percutaneous coronary intervention (PCI) is established as one of the most common procedures for the treatment of coronary artery disease (CAD), based on considerable evidence. The advances in procedural techniques, stent materials and aggressive antiplatelet therapy have decreased the incidence of major periprocedural complications of PCI. However, periprocedural myocardial injury (PMI) is still one of the inevitable complications of PCI, resulting from distal embolization, side-branch occlusion, coronary dissection and disruption of collateral flow. A previous study has reported several risk factors for PMI, including more extensive disease, multivessel disease, complex lesion morphology, coronary thrombus and calcification, left ventricular systolic dysfunction and urgent procedures.¹ Both cardiac troponins and creatine kinase MB are sensitive and specific biomarkers for quantitative diagnosis of irreversible myocardial injury, and the release of these biomarkers is associated with increased risk of death and myocardial infarction (MI).² A previous report showed that a 5-fold post-procedural elevation of cardiac troponin T above normal levels is an independent predictor of a composite of death, MI, and revascularization at 1 year (hazard ratio, 2.39; 95% confidence interval, 1.09–5.26).³ Importantly, because PMI also occurs in a considerable

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**Figure 1.** Biological effects of remote ischemic preconditioning. (Reproduced with permission from reference 12.)
A meta-analysis of randomized trials revealed that RIPC is known as an opener of the K ATP channel with activation of the K ATP channels and inhibition of the oxygen species, protein kinases and nitric oxide (NO). Activation of the mitochondrial permeability transition pore, opioid receptors, reactive cardiac troponin levels still occurred in 29% of the patients subsequent meta-analysis has revealed that increased major cardiovascular events in patients with angina.

Oral administration of nicorandil reduced the incidence IPC has been investigated. The IONA study showed that a NO donor property; therefore, its effect as an agent of IPC acts protectively not only locally, but also protects remote organs, known as RIPC (Figure 1). A meta-analysis of randomized trials revealed that RIPC significantly reduced the increase of myocardial injury in patients undergoing coronary artery bypass graft surgery. The mechanisms underlying the cardioprotective effect of RIPC are similar to those reported for IPC; including neural and humoral signaling pathways (Figure 2). The RIPC procedure is safe and tolerable for patients, consisting of several cycles of inflation of a blood pressure cuff on the arm or leg to a pressure of 200 mmHg, followed by cuff deflation for a short period of time. The RINC trial showed no significant difference in the incidence of PMI following elective PCI, corresponding with the result of the meta-analysis. However, in the current subgroup analysis, the authors show that the incidence of PMI was similar between the control and RIPC groups of patients with simple coronary lesions, but notably lower in the RIPC group than in the control group of patients with complex coronary lesions. The COURAGE trial provided evidence that PCI does not improve mortality or reduce major cardiovascular events in patients with stable CAD, compared with optimal medical therapy. However, the prevention of PMI may lead to improved prognosis after PCI. Thus, RIPC may be a novel therapeutic option for high-risk patients undergoing PCI and improve long-term mortality. However, further studies are needed to evaluate its effect on death and cardiovascular outcomes.

**References**

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