Acute myocardial infarction (AMI) is mostly caused by atheroma plaque rupture with thrombosis formation in the lumen of a coronary artery, which partially or completely disrupts blood flow to perfuse the myocardium. In general, the ischemic myocardium becomes necrotic if there is no collateral coronary blood flow and the culprit lesion of the target vessel is not patent within 12 hours. Primary percutaneous coronary intervention (PPCI) is the most often used management for AMI nowadays and the in-hospital mortality rate approximates around 5% for those with AMI undergoing PPCI in high-volume cardiac laboratories. Blood pressure (BP) plays the central role in hemodynamics, which is closely related to the cardiac performance during AMI. Previous studies have shown that admission systolic BP and pulse BP referring to the pulsatile component of BP have a J-shaped or U-shaped relationship with in-hospital mortality in patients with AMI after PPCI.1,2 In this issue, Shiraishi and colleagues examined the association of mean BP, reflecting the steady component of BP, with in-hospital mortality in Japanese patients with AMI following PPCI. They found that admission mean BP < 79 mmHg might be associated with in-hospital mortality but other BP categories were not.3

The lowest mean BP level at admission associated with the highest risk of in-hospital mortality in patients with AMI is reasonable and not unexpected. Several mechanisms could explain this finding. First, the lowest mean BP group certainly included most cases of cardiogenic shock, which was found to be an independent predictor of in-hospital mortality in their report.1,5 Second, lower cardiac power output, the product of cardiac output and mean BP, has been associated with in-hospital mortality in patients with AMI or cardiogenic shock.4,5 Although cardiac output, the product of stroke volume and heart beat rate, was not measured in this study, cardiac output in the lowest mean BP category may be lower than other groups because of the greater proportion of culprit lesions in the right coronary artery, possibly leading to a slower heart beat rate and right ventricular infarction which decrease the stroke volume. Third, a low mean BP level may reflect the reduced cardiac reserve when the myocardium was injured before PPCI. Cardiac reserve has been recognized as another strong predictor of in-hospital mortality in cardiogenic shock.6 In contrast, the highest mean BP level at admission was not associated with the risk of in-hospital mortality in patients undergoing PPCI. This result was contrary to previous findings of the prognosis of systolic BP and pulse BP levels in AMI. Acute elevated BP levels may trigger inflammatory reaction, procoagulant system activation, and platelet aggregation,7,6 which may increase the risk of re-infarction, and mortality after PPCI. In contrast, high mean BP levels at admission may represent high cardiac reserve in AMI, which would offset its hazardous effect.

In the real world, mean BP levels are easily obtained at admission and convenient for the cardiologist to use as a prognostic factor in patients with AMI. Since the study lacks some important data such as heart rate at admission, post-procedural left ventricular function, myocardial ischemic time, and peak cardiac enzymes that were not collected prospectively, the findings regarding the relationship could be affected by these unadjusted confounders. In addition, the clinical utility of low mean BP levels is somewhat overlapped with and limited by the status of cardiogenic shock. Therefore, further well-designed prospective studies are needed to clarify the association of high mean BP with in-hospital mortality.

Disclosure
Conflict of interest: None declared

References

From the 1 Department of Medicine, Hualien Armed Forces General Hospital, Hualien, Taiwan and 2 Department of Preventive Medicine, Northwestern University Feinberg School of Medicine, Chicago, IL, USA.
Address for correspondence: Yuichiro Yano, MD, Department of Preventive Medicine, Northwestern University Feinberg School of Medicine 680 North Lake Shore Drive, Suite 1400, Chicago, IL 60611, USA. E-mail: yyano@jhchi.jp or yuichiro.yano@northwestern.edu
Received for publication May 1, 2016. Accepted May 6, 2016.
Released in advance online on J-STAGE August 16, 2016. All rights reserved by the International Heart Journal Association.


