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Letters to the Editor

☐ BLOOD PRESSURE CONTROL AND ENDOVASCULAR THERAPY

☐ **To the Editor:**

We read with keen interest the comprehensive review by Rezaie et al. regarding the paradigm shift in the management of acute ischemic stroke (AIS) (1). Their review provides a comprehensive overview of the currently accepted standards for managing large vessel occlusion (LVO) through endovascular therapy (EVT) (1). A strength of their review is the acknowledgment of the heterogeneity of AIS disease based on infarct core and size of ischemic but salvageable brain tissue, assessed using advanced imaging. In addition, the recognition as to the role of advancing imaging in EVT patient selection, subsequently aiding in the delivery of positive clinical trials, is clearly highlighted.

Rezaie et al. kindly provide the latest iteration of the American Heart Association/American Stroke Association guidelines that provide recommendations and strength of supporting evidence for pre- and post-EVT care (1,2). Elevated blood pressure (BP) poststroke is a key risk factor for poor stroke outcome (3,4). The guidelines state "in patients who undergo mechanical thrombectomy (EVT), it is reasonable to maintain the BP $\leq$ 180/105 mm Hg during and for 24 hours after the procedure (Level IIa evidence)" (2). However, the conflicting evidence for BP control post-EVT is very much understated within this article and reference to the lack of relevant prospective randomized controlled trial (RCT) data, the effects of general anesthesia during EVT, and the importance of individualized assessment based on reperfusion status could be outlined.

First, with reference to BP control post-EVT, a post hoc analysis of the Multicenter Randomized Clinical Trial of Endovascular Treatment (MR CLEAN) trial data demonstrated that BP does not affect the benefit or indeed safety of EVT in AIS caused by LVO (5). However, systolic BP was correlated with functional outcome in a U-shaped manner, with high and low BP associated with the worst outcomes (5). Second, with reference to anesthesia during EVT, further post hoc analyses from the MR CLEAN study demonstrated that a decrease in BP during intervention under general anesthesia as compared to baseline BP is associated with worse stroke outcome (6). Lastly, with reference to reperfusion status, those who failed to recanalize post-EVT had worse outcomes if their BP demonstrated significant variation from their mean BP during the first 24 h post-EVT (7).

In our view, there exists significant variation in approach to BP management post-EVT. This is demonstrated by a lack of standardized protocols for post-mechanical thrombectomy BP management across institutions and interinstitutional heterogeneity in the "target" BP post-mechanical thrombectomy (8). There is evidence that the majority are targeting <180 mm Hg; however, whether they are adequately positioning their BP control strategy within the well-defined "U-shaped" curve remains unclear. Fortunately, RCT data from studies assessing intensive versus standard blood pressure lowering in AIS are available, and though the study was neutral for the primary outcome, participants in the intensive group were less likely to suffer intracranial hemorrhage (9). Future research directions should now begin to examine for BP thresholds for favorable outcomes when using EVT for LVO specifically.

Lastly, it is worth adding an additional consideration to the authors comment on the modified Rankin scale score used in stroke trials. There are data demonstrating reliability to determine the modified Rankin scale category score when assessed via consultation or remotely via telephone interview (10). In addition, statistical sensitivity is increased by assessing the whole scale via ordinal methods, thereby increasing the likelihood of detecting a treatment effect.

We thank the authors for addressing this important aspect of acute stroke care and commend the wide variety of literature they have drawn on. However, with an increasing body of evidence supporting BP control in pharmacologic reperfusion circumstances, there is a necessity to answer similar questions during EVT therapy. Ultimately, further research data, ideally generated through randomized studies of BP control strategies, is required to greater improve the focussed care we give those with LVO requiring EVT.

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☐ SHOULD TARGET GLYCEMIC RANGE BE EXACTLY THE SAME FOR PATIENTS WITH ACUTE MYOCARDIAL INFARCTION VERSUS WITHOUT DIABETES?

☐ **To the Editor:**

Pronounced hyperglycemia by itself is a dangerous condition, but it is particularly dangerous when accompanied by ketoacidosis or a hyperglycemic hyperosmolar state (1). The measurement of glycemia is universally available and inexpensive. Glycemia is often measured in the emergency department (ED); in almost 20% of patients, glucose concentration in serum is evaluated, and capillary glucose measurement is required in an additional large number of patients (2). Another rationale for measuring glycemia in the ED is the fact that every fourth patient in the ED has diabetes mellitus (DM) (3). Glycemic control (and therefore the target range) are important things to be aware of in the ED (2,4–17). High serum glucose concentration in the ED is a valid marker of in-hospital morbidity and mortality (4,5). Despite the prognostic significance of hyperglycemia in the ED, its management in the ED is clearly suboptimal (5). It may be advantageous to start proper hypoglycemic treatment in the ED because it results in faster achievement of the glycemic target(s).

The *Journal of Emergency Medicine* published the Rush Emergency Department Hyperglycemia Intervention protocol for ED treatment of hyperglycemia using subcutaneous insulin before hospital admission or discharge. This protocol was safe and reduced the in-hospital length of stay (8). The Rush Emergency Department Hyperglycemia Intervention protocol was cited as valid in the Consensus Statement on Inpatient Glycemic Control of the American Association of Clinical Endocrinologists and the American Diabetes Association (13). Furthermore, Bernard et al. have demonstrated that use of the standardized insulin protocol in the ED and in the hospital can provide better results compared with the usual care (9). In addition, as far as acute myocardial infarction (AMI) is concerned, the Immediate Myocardial Metabolic Enhancement During Initial Assessment and Treatment in Emergency Care (IMMEDIATE) trial that was performed before admission and in the ED has demonstrated that glucose infusion with insulin and potassium reduced the size of the myocardial infarction (MI) after 30 days of follow-up (18). Moreover, a triple endpoint (cardiac arrest, mortality, or heart failure), or hospitalization within 1 year of follow-up was reduced in ST-elevation AMI patients in this trial (18). Therefore, parameters that are already measured in the ED, such as glycemia, ought to be used for treatment. For example, in a patient with DM and chest pain (without ST elevation or other electrocardiographic abnormalities typical of non-ST-segment elevation acute coronary syndrome), who has a high capillary glucose level measured, insulin treatment can be started in the ED while waiting for the troponin result. Indeed, marked hyperglycemia (with or without ketoacidosis or a hyperglycemic hyperosmolar state) is much more dangerous in the AMI setting without insulin. The numerous studies published to date have confirmed the rational expectation that hyperglycemia, both at admission (admission blood glucose), fasting, and during the hospital stay, is a valid prognosticator of poor in-hospital outcome in patients with AMI (19,20).

Arterial blood gases can be a valid prognostic marker for patients with AMI, even for a follow-up of 20 years (21). Also, AMI has been known for a long time (since 1931) to worsen glycemic control (22). There is little doubt that acute control of hyperglycemia in AMI is beneficial. For example, in the Japanese Acute Coronary