from a standpoint of hand ischemia, and ulnar artery structure. In reality, these 2 datasets (1,2) are complementary, increasing the strength of the evidence underscoring the efficacy of prophylactic ulnar compression in RAO prevention across a broad array of patient subsets, and regardless of the hardware used to compress the ulnar artery.

We agree with Dr. Koutouzis and colleagues that a dedicated device with dual and precise compression capabilities would be ideal and will allow wide adoption of this important technique, improve patient comfort and outcomes.

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Do We Need Individualized Therapeutic Strategies in High-Risk Patients Treated With Hypothermia?

We congratulate Shah et al. (1) for their excellent analysis of a very important question concerning stent thrombosis and hypothermia. However, from our point of view, there are some concerns that should be considered in detail.

First, the investigators did not define the different antiplatelet therapy strategies in detail, as prasugrel has been available since 2009. At present, the influence of hypothermia on the effectiveness of the newer adenosine diphosphate inhibitors such as prasugrel and ticagrelor is not sufficiently understood. The newer adenosine diphosphate inhibitors seem to be more effective, as the effectiveness of clopidogrel is reduced in therapeutic hypothermia (2,3).

Furthermore, it was shown that individually guided clopidogrel therapy significantly reduced adverse thrombotic events in patients undergoing coronary stent implantation (4). Therefore, it would have been of great interest to know whether the patients in the present study received tailored antiplatelet therapy with different loading doses.

Second, the investigators did not provide information about the kinds of drug-eluting stents implanted. As shown in the DAPT (Dual Antiplatelet Therapy) trial, there is increased risk for stent thrombosis with the use of paclitaxel-eluting stents compared with, for example, everolimus-eluting stents. This risk is further reduced with the use of prasugrel, as indicated in more intense platelet inhibition.

Taken together, a third issue must be discussed critically in this context. With an incidence of stent thrombosis of about 4% in the present study, related to a high proportion of patients with cardiogenic shock, the question is raised whether to treat coronary lesions simultaneously or only culprit lesion. Given that the incidence of complications is higher because of cardiogenic shock and hypothermia, a staged procedure that treats only culprit lesions seems to be the most feasible therapy. The solution may partially be found in ongoing trials such as the CULPRIT-SHOCK (Culprit Lesion Only PCI Versus Multivessel PCI in Cardiogenic Shock) trial (5).

The relatively high incidence of stent thrombosis in the present study again demonstrates that hypothermia treatment itself defines a high-risk patient, who needs more individualized antiplatelet therapy and a differentiated revascularization strategy.

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REPLY: Do We Need Individualized Therapeutic Strategies in High-Risk Patients Treated With Hypothermia?

We thank Dr. Jellinghaus and colleagues for their interest in our article (1). We have read their commentary with great interest and would like to respond to the points raised by them.

Dr. Jellinghaus and colleagues mention the potential impact of use of newer $P2Y_{12}$ inhibitors such as prasugrel and ticagrelor. Although prasugrel has been available in the United States since 2009, it was not readily available in all hospital pharmacies and therefore not used in a widespread fashion from the outset. An analysis from the National Cardiovascular Data Registry reveals that the rate of prasugrel use in patients with acute coronary syndromes went from 3% in 2009 to 18% in 2012 (2), indicating that even after 3 years, the rates of prasugrel utilization remained low. Moreover, 21.4% of subjects in our overall study population, and 11.9% of subjects among those undergoing hypothermia were older than 75 years of age, which would be a contraindication for prasugrel use. Furthermore, Jellinghaus and colleagues assert that newer $P2Y_{12}$ inhibitors would reduce stent thrombosis rates; however, this is not true in cardiac arrest patients. In fact, a 2015 study showed surprisingly higher rates of stent thrombosis with newer $P2Y_{12}$ inhibitors compared to clopidogrel in survivors of out-of-hospital cardiac arrest (3).

Although intuitively a tailored approach with different loading doses of clopidogrel proposed by Dr. Jellinghaus and colleagues seems attractive, the GRAVITAS (Gauging Responsiveness with A VerifyNow assay-Impact on Thrombosis and Safety) trial in 2011 (4) showed no difference in cardiovascular death, nonfatal myocardial infarction, or stent thrombosis at 6 months with high dose compared to standard dose clopidogrel in patients with high on-treatment platelet reactivity after percutaneous coronary intervention.

Our study database is an administrative database and does not contain information about the specific type of drug-eluting stent (DES) implanted as suggested by Jellinghaus and colleagues; however, it does allow us to compare DES with bare-metal stents (BMS), and shows no difference in stent thrombosis rates with DES compared to BMS (4.8% vs. 4.6%; $p = 0.70$) (1).

Although our study shows that stent thrombosis rates are higher in patients with cardiogenic shock and with increasing number of stent implantations (1), the American College of Cardiology percutaneous coronary intervention guidelines in 2011 recommend revascularization of noninfarct arteries in patients with cardiogenic shock to maximize myocardial perfusion and improve hemodynamic stability (5). If a complete revascularization strategy serves to stabilize patients with refractory cardiogenic shock, higher stent thrombosis rates should not discourage operators from applying this strategy in emergent situations. Patients who are sicker tend to have higher stent thrombosis rates, and although it is important to be cognizant of this risk, it is hardly justified to withhold life-saving therapies for the fear of stent thrombosis.

Finally, we agree that the cardiac arrest state presents a high-risk situation, and optimal antiplatelet, anticoagulation, and revascularization strategies need to be determined in these patients.

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