☐ IN REPLY TO PASSQUIRE ET AL.

☐ To the Editor:

We appreciate your interest in our article and thoughtful comments. We believe you are quite correct that the most likely cause for the disparity in our clinical outcomes lies in selection bias that was inherent to both of our studies. We believe there is ample evidence of a dismal prognosis for patients with traumatic cardiac arrest (TCA) who present without signs of life on examination and no organized electrical activity on electrocardiogram (ECG) (1–3). Our system in Montgomery County follows the American College of Surgeons/National Association of EMS Physicians joint position statement on withholding or terminating resuscitation to guide our clinical protocols. This position statement suggests that resuscitation may be withheld in patients presenting after blunt or penetrating injury, pulseless and apneic without other signs of life on examination or electrocardiography (4). Our series included only patients in whom resuscitation was attempted and excluded any patients from whom the medics in attendance withheld efforts in patients meeting our criteria, no signs of life, and without organized electrical activity on ECG. We believe further studies involving larger numbers of patients with TCA will better illustrate both the efficacy of prehospital chest drains for TCA and other factors associated with favorable neurologic outcomes in these patients.

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☐ "CONTROVERSIES IN MANAGEMENT OF HYPERKALEMIA:" REMARKS ON THE RISK OF HYPERGLYCEMIA

☐ To the Editor:

We read with much interest the American Academy of Emergency Medicine Clinical Practice paper by Long et al. on controversies in management of hyperkalemia (1). The authors should be congratulated on the efforts to clarify this important topic. Although we agree with many of their conclusions, we would like to make two remarks on the risk of hypoglycemia in connection with the treatment of hyperkalemia. We believe they may contribute to further insight into this important area.

Our major concern is related to the statement that factors associated with hypoglycemia after the administration of insulin to treat hyperkalemia include no prior diagnosis of diabetes and no use of diabetes medication prior to hospital admission, leading to the conclusion that diabetes "per se" diminishes the risk of hypoglycemia in the above-mentioned type of insulin treatment. The study cited in relation to this statement was a retrospective one conducted on patients with hemodialysis (2). The authors, and in addition, several other studies with similar findings, treated diabetes mellitus in the analysis as one disease, although diabetes mellitus is a group of metabolic diseases resulting from various pathogenic defects and may be treated in different ways, with varying risks of hypoglycemia (3,4). Looking at the results of the study, it seems much more likely that the real reason for the lower rate of hypoglycemia found in diabetes patients was actually the higher glucose level prior to hyperkalemia treatment, which was another study finding (not statistically tested) also mentioned in the article by Long at al. (1). As there was no protocol for hyperkalemia treatment in the hospital where the study was conducted, another possible reason for the lower rate of hypoglycemia among diabetes patients could be the less aggressive treatment approach and more frequent glucose monitoring in these patients.

We have recently expressed our speculation based on clinical experience that the higher risk of hypoglycemia after insulin administration due to treatment of hyperkalemia may be associated with type 1 diabetes, as this disease is or may be accompanied by: 1) failure to clear circulating insulin during hypoglycemia; 2) lower glucose threshold for release of counterregulatory hormones, and loss of normal pancreatic alpha cell response; 3) impaired awareness of

hypoglycemia that may be present in 30% and even more patients; 4) usually good insulin sensitivity (making the patients more sensitive to even smaller doses of insulin, especially if added to their regular daily doses) (5). We believe that in these patients, special attention should be paid when administering insulin in the treatment of hyperkalemia, especially if the glucose level prior to insulin administration is in or close to the normal values. As some of the above-mentioned features could be present in type 2 diabetes patients, we also advise that special attention should be paid to those treated with insulin or sulfonylurea derivates.

Our second remark is related to the use of insulin analogues in the treatment of hyperkalemia in patients with renal failure. The authors correctly noted that their half-lives are not prolonged in renal insufficiency patients because they are not eliminated chiefly via renal excretion: some studies therefore showed a lower risk of hypoglycemia. But, as the gluconeogenesis is impaired in the kidneys of those patients, a higher risk of hypoglycemia is, to some extent, still present (2).

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☐ RISKS OF HYPOGLYCEMIA WITH INSULIN THERAPY FOR HYPERKALEMIA

☐ The Reply:

Thank you, Dr. Brož, for your insights regarding the risk of hypoglycemia with insulin therapy for hyperkalemia. Dr. Brož brings to light several important considerations regarding hypoglycemia in his discussion of insulin therapy for hyperkalemia, especially in patients with diabetes and end-stage renal disease (1–3). Several studies suggest that risk factors for hypoglycemia with insulin therapy for hyperkalemia include lower patient weight (<60 kg), patients without prior history of diabetes, and patients with lower pretreatment glucose levels (1,4). As Dr. Brož states, one major contributor to hypoglycemia is lower serum glucose levels in patients without diabetes, as the elevated serum glucose levels in diabetic patients may protect them from hypoglycemia (5). End-stage renal disease also results in prolonged insulin duration of action due to reduced insulin clearance (1–3).

Dr. Brož, in his letter, further states that patients with insulin-dependent diabetes (formerly type 1 diabetes) demonstrate difficulty clearing circulating insulin and possess a higher sensitivity to even small doses of insulin, decreased glucose threshold for counterregulatory hormone release, and impaired awareness of hypoglycemia (5). In non-insulin-dependent diabetic patients (formerly