



Response to: Relation between cortisol and admission blood glucose in patients admitted with myocardial infarction but without hyperglycemia!



We thank Dr. Chattopadhyay and Dr. John for their comments. Whilst the Endocrine Society did not specifically label a BG > 7.8 mmol/L as “stress hyperglycemia”, the guideline indicates that this is hyperglycemia, and have recommended this as a threshold for ongoing monitoring and intervention.¹ We therefore believe that it is reasonable to use this cut-off to define stress hyperglycemia in our study.

Within the 45 subjects without known diabetes but with an admission glucose ≥ 7.8 mmol/L, there were 11 who had an HbA1c in the diabetic range (≥ 48 mmol/mol). Three of these subjects were not classified as abnormal glucose tolerance (AGT) by our a priori glucose parameters. We have in fact already provided the re-analysis with these 3 subjects classified as having AGT, though in the paper it was erroneously stated that these 3 subjects did not have an admission glucose ≥ 7.8 mmol/L, when it should have read that these 3 subjects did have an admission glucose ≥ 7.8 mmol/L.² This did not alter the relationship between admission glucose and serum cortisol.

As acknowledged in the paper, we agree that it is possible that some subjects with a normal admission glucose level could be found to have AGT had they undergone follow-up testing. However it is unlikely that there would be many such patients when the stress of myocardial infarction did not cause hyperglycemia. Dr. Chattopadhyay and Dr. John have suggested that stress may lead to a modest rise in glucose which does not necessarily cross the cut-off used to define stress hyperglycemia. We agree that this is indicated by the linear relationship seen between admission glucose and cortisol level. Indeed, admission glucose has been shown to be predictive of mortality amongst myocardial infarction patients in an incremental manner, from as low a glucose level as 6.1 mmol/L.^{3,4}

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