one side of the road connecting the brain and the heart, ignoring the possibility that poor cardiac function leads to depression rather than the other way round.

The study by van Melle et al.\(^4\) is very important because it shows that the severity of left ventricular dysfunction is significantly related to the severity of depressive symptoms in patients with myocardial infarction. This at least suggests the possibility that heart damage may be primary, and the mood disturbance follows. We agree with Kounis et al. that characterizing the mechanisms connecting the brain and the heart—as well as their direction—is critical, because the implication that left ventricular dysfunction might lead to depression is that if the former is treated appropriately, the latter may improve.

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Markers of myocardial ischaemia

We read with interest the article by Giannitsis and Katus\(^1\) on markers of myocardial ischaemia. Although their comments are authoritative and scholarly, we feel that the authors failed to objectively summarize the evidence available for ischaemia-modified albumin (IMA) as a marker of myocardial ischaemia. Research from our unit has shown that IMA is an early marker of ischaemia in patients undergoing coronary angioplasty.\(^2\) We have also shown that IMA has a higher sensitivity than the 12-lead ECG and cardiac troponin (cTn) T levels for the diagnosis of acute coronary syndrome (ACS) in chest pain patients attending the emergency department within 3 h of the onset of pain.\(^3\) The combined use of IMA, cTn, and a negative ECG has a high negative predictive value for ACS, which may be extremely useful to rule out ACS in the emergency room. In fact, the FDA has recently approved the use of IMA for this indication.

The use of IMA as a marker of ischaemia in the clinical setting, however, has limitations at present. (i) IMA has a relatively low specificity for myocardial ischaemia as it may increase in patients with stroke, end-stage renal disease, liver disease, and some neoplasms. (ii) Increased endogenous lactate levels appear to reduce IMA concentrations, which raise concern about the true significance of a negative IMA result in patients with sepsis or renal failure where lactate may be present in the circulation. (iii) A further limitation is that the ACB test (the test currently used to measure IMA) is a colormetric assay, and therefore an indirect test currently used to measure IMA. Required to assess the true role of IMA as a marker of myocardial ischaemia. Incorporating the ACB test into the decision-making process of a typical emergency room population may be a good way forward to ascertain whether IMA has a diagnostic and risk stratification role in the clinical setting. Importantly, however, as with other cardiac markers, IMA cannot be used effectively without fully considering the clinical circumstances of the patient.

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