healthcare systems.2 This is noteworthy, since a variety of additional variables may contribute to adverse risk besides the elevation of cardiac biomarkers and the presence of suggestive electrocardiogram changes.7,8 Our results support this hypothesis, underlining that a strategy aimed at rationalizing troponin testing, such as that proposed by Ramsay et al., based on the application of a validated risk assessment, would be far more cost-effective for identifying patients at higher risk for subsequent cardiac events, especially those presenting at the emergency department, than simply doing an isolated troponin measurement at admission.2

With pressure from cost containment and reimbursement policies worldwide, expenditure on non-diagnostic results also highlights the need to identify diagnostic strategies based on something more concrete than simple clinical suspicion. In addition, a positive troponin result in a patient in whom the test was inappropriate in the first instance, may generate other unfavourable consequences, including decreased specificity, costs and health risk from further investigations, and unnecessary patient anxiety.9

G. Lippi
G.L. Salvagno
M. Montagnana
G.C. Guidi
Sezione di Chimica e Microscopia Clinica
Dipartimento di Scienze Morfologico-Biomediche
Università degli Studi di Verona
Verona
Italy
email: ulippi@tin.it; giuseppe.lippi@univr.it

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Estimated glomerular filtration rates (eGFR): predicting progression is dependent on accuracy

Sir,
In a recent paper, Hemmelgarn et al. introduced a potentially important clinical risk score for the prediction of progression amongst patients with chronic kidney disease (CKD).1 The use of readily available clinical data to allow risk stratification of patients would represent a significant advance in the practical management of CKD. However, the study has a number of potentially problematic methodological issues. The authors chose to limit their study population to those with an eGFR <90 ml/min/1.73 m², because of concerns regarding the validity of the MDRD eGFR estimate above that level of function.2 These validity concerns continue to be a problem when using the MDRD estimation equation between 60 and 90 ml/min/1.73 m², and this concern has become so prominent within the nephrology community that the equation’s creators have recommended reporting eGFR estimates as simply >60 ml/min/1.73 m², rather than giving specific estimates above that level.3

Recent advances have improved the accuracy of the MDRD equation (traceable by isotope dilution mass spectrometry IDMS),4 but (as recently discussed in QJM) these corrections are dependent on central laboratory validation and provision of correction factors.5 Regardless of the methodology, there remains significant concern over the clinical relevance of an estimated GFR between 60 and 90 ml/min/1.73 m², particularly in the
elderly female population. Including these patients in the analysis increases the likelihood of misclassifying anomalies in creatinine measurement and eGFR estimation as progression. Previous studies have indicated the potential variance of eGFR estimation in the same person over time within the same laboratory, and more recently we have come to realize the impact of simple daily activities, such as a cooked meal, on eGFR estimations.

In epidemiological studies assessing CKD progression, it is crucial to limit methodological errors in creatinine measurement and eGFR estimation, if we are to gain a reliable understanding of the natural history of CKD. Cystatin C may represent the most exciting potential solution to estimating renal function accurately in early CKD, but its expense and lack of availability currently limit its use; until this changes, we must be aware of the limitations of our currently used estimates.

M. Quinn
A. Rainey
K.J. Cairns
A.H. Marshall
D.G. Fogarty
The Queen’s University
Belfast
email: mquinn05@qub.ac.uk

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