over a dozen examples of ACTH secreting small cell carcinoma, starting with Wenk *et al* in 1978.[2] Regrettably, the consent for our patient's autopsy did not include examination of intracranial contents, so we were unable to examine the pituitary gland. ■

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2 Wenk RE, Bhagavan BS, Levy R, Miller D, Weissburger W. Ectopic ACTH, prostatic oat cell carcinoma and marked hypernatremia. *Cancer* 1977;40:773–8

Atrial fibrillation

Editor – Clayton and colleagues should be congratulated on focussing on an important cohort of patients within intensive care. The authors report a retrospective observational analysis in a cohort of patients who develop novel atrial fibrillation/atrial flutter within a generalised intensive care unit setting.[1]

However beyond the salient discussion and conclusion the data highlights further points of note.

Firstly within the reported demographics there is no description of contemporary left ventricular function as measured on echocardiography. The group of patients who developed new onset atrial arrhythmias had an increased incidence of atherosclerotic disease, hypertension and diabetes all of which increase the chance of incident left ventricular systolic dysfunction. Atrial fibrillation with uncontrolled ventricular rates may also in itself induce a tachycardia-associated cardiomyopathy. Finally, the patient admitted to intensive care is critically unwell with other pathology, eg sepsis, which may also induce myocardial dysfunction.[2]

Hence the development of atrial fibrillation within such patients should be an opportunity to consider a detailed cardiology review, consideration of cardiac monitoring, rationalisation of inotropes and potentially serial detailed assessments of left ventricular function using echocardiography.

Secondly the presence of atrial arrhythmias should be communicated following successful discharge from the intensive care unit to the ward. Beyond using short-term anticoagulant measures, prior to discharge the patient should have formal evaluation of their thromboembolic risk and bleeding risks documented. There should be consideration of the appropriateness of anticoagulation and correct method. This should also include the patient and family with full detailed anticoagulation counselling. It is imperative that such important clinical information follows the patient throughout the rest of their hospital stay, but following a complex lengthy admission it is not unusual to find that the ultimate discharge summary does not contain any mention of atrial arrhythmias nor the need to review anticoagulation. ■

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2 Ponikowski P, Voors AA, Anker SD, Bueno H *et al*. 2016 European Society of Cardiology guidelines for the diagnosis and treatment of acute and chronic heart failure: the task force for the treatment of acute and chronic heart failure of the European Society of Cardiology. *Eur Heart J* 2016;37:2129–200.

Response

We would like to thank Dr Guha for his supportive comments and express our agreement, at least in principle, with most of the issues he raises. We were careful in our article to keep our conclusions aligned with the current evidence and believe that further study is needed.

Left ventricular (LV) function was not assessed consistently across our population. Where it was known or assessed prior to or during the index episode it is included (insofar as it's relevant to thromboembolic risk assessment) in the CHADS2VASc score (4% with known or demonstrable LV dysfunction). We acknowledge the potential for critical illness, pharmacotherapy and atrial fibrillation (AF) itself to affect LV function, but while this is of course relevant to the clinicians managing their acute episode the relevance of transient LV dysfunction on long term thromboembolic risk is less clear.

We also agree that the occurrence of even a transient arrhythmia should be communicated to the ward teams and ultimately to the patient's general practitioner following discharge from the intensive care unit, and it certainly warrants further patient evaluation. In our population, the documentation of this was very limited, but this may be because conventional thinking was that transient AF in the setting of critical illness is a benign, almost inevitable, phenomenon with no longer term ramifications. We sincerely hope to have altered this perception.

There are, we believe, a great many issues that our article begins to raise. It does not yet provide sufficient evidence that these patients should definitely be managed the same as other groups with paroxysmal AF. Our overriding desire in writing was to bring these issues to the attention of a wider medical audience and begin a conversation about the management of these patients and we are grateful to Dr Guha for contributing to this. ■

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Feedback on CME haematology

Editor – The article by Brierley and Pavord[1] contains an unfortunate omission. On summarising secondary causes of immune cytopenias (Table 1), HIV infection is only mentioned as a possible contributor to autoimmune hemolytic anemia. Indeed, the most common presentation of HIV related cytopenias is immune thrombocytopenic purpura as published and supported by several published articles.[2,3]

General physicians' lack of appreciation of this fact has led to several cases of late HIV diagnosis. National Institute for Health and Care Excellence guidelines[4] now strongly recommend HIV testing of all patients with thrombocytopenia. I wonder if the table should be amended to reflect these facts. ■