patients with complex care needs. Frank showed that readmission is more likely in those living alone, while Woodard and Conroy echo our findings that readmission is more prevalent in frail older patients, who generally have increased lengths of stay. Heydtmann introduces a further important group – those admitted with alcoholic liver disease. It is distressing to read of such high readmission and mortality rates in this cohort.

Our article discussed the merits of a multidisciplinary approach in caring for high-risk patients, who have been shown to benefit from adequate discharge planning and aftercare initiatives.[2,3] We read with interest Woodard and Conroy's description of an acute frailty unit, which will likely improve the standard of care provided to older patients, and we await with further data from their experience.

Patients at risk of readmission have been shown to be older and sicker with less social support than other inpatients. They have medical, psychological and social needs that are complex and significant. Our ageing population means the size of this cohort will only increase. Caring for these patients requires a multidisciplinary, holistic approach that seamlessly coordinates care across diverse locations.

As well as adequate medical provision, further investment in effective discharge planning and aftercare strategies, such as the care attendants scheme discussed by Frank,[4] will need to be implemented. This will require better cooperation between the medical profession and social services, as well as the political will to implement this change. The challenge is significant, but the reward is happier, healthier patients.

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Quality of care

Editor – I am moved by Professor Allan's elegy to bygone NHS virtues of 'calm caring and gentle pace of clinical life…and all the time in the world to deliver compassionate care' (*Clin Med* October 2009 p 407). One's immediate instinct would be to say 'Ah, but times have changed' – only to find the Editor extolling the same high level of care in a hospice in present-day England.

One can attempt to blame the unforeseen rise in the level of patients seeking emergency medical care and requiring acute hospital admission; the physical limits to what one can do with an outmoded hospital infrastructure; inadequate handover mechanisms; over-politicisation and micro-management of the delivery of patient care; and the list goes on.

I wish to argue that this regrettable gear shift in patient care is in no small part due to a disenfranchised clinical workforce in general. We are urged to explore new ways of working and improve efficiency in order to provide care for an ever increasing number of patients by a depleted workforce. Of course efficiency must be increased, but a workforce that is plagued by low morale and a poor sickness record is in no position to do such a thing.

I plead with our clinical leaders and politicians to work hard to re-energise our clinical workforce. The professional hierarchy must no longer delay tackling the sickness record in the NHS head-on, improve staff recruitment and retention, reward those who work hard, retain good senior nurses on the ward rather than an automatic channel to management and re-empower ward senior nurses (can we stop calling ward sisters or matrons 'ward managers'?).

A new hospital can address many of the shortcomings mentioned by Professor Allan. But a caring environment is still a numbers game: a small handful of nurses, however good they may be, cannot emulate the level of care recalled in the editorial when asked to look after a busy ward of more than a dozen of the infirm. Likewise, a dizzying day-to-day shift of a medical team provides only fragmented care. Things must be going wrong when I found myself presenting the case history of every patient on a Monday morning ward round to my foundation doctors and registrar who had all come back from various leave and shifts, and that was not the August changeover! How do we restore the firm structure and team spirit in the shadows of the European Working Time Directive and budget cuts without increasing the number of doctors? I do not think we can. We create rotas of complex shifts for our doctors, and that is what they will continue to be, shift workers.

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Applications of pharmacogenetics: importance in the treatment of diabetes

Editor – It is with interest that I read the recent article by Munir Pirmohamed (*Clin Med* October 2009 pp 493–5). The article explained how genotype testing might guide drug choice. I would like to highlight how detection of individual gene mutations is being used to influence drug therapy within the specialty of diabetes.

The realisation that some forms of diabetes occur as a result of monogenic mutations has allowed clinicians to optimise patient therapy by choosing drugs that are more likely to overcome the consequences of particular mutations.

Mature Onset Diabetes of the Young (MODY) is an inherited form of diabetes that often presents before the age of 25 years. Identification of genes causing MODY has allowed alternatives to insulin treatment to be offered to patients. Hepatocyte nuclear factor 1α (HNF-1α) and glucokinase mutations are the most common causes of MODY.[1] HNF-1α

diabetes has a marked sensitivity to sulphonylureas, to the extent that patients can have improved diabetes control with a switch from insulin treatment to sulphonylurea therapy if the mutation is identified. Patients with MODY due to heterozygous glucokinase mutations often do not require insulin treatment at all and may be able to come off treatment altogether.

Permanent neonatal diabetes is a non-autoimmune condition that usually presents before the age of 18 months. It can be caused by monogenic mutations in the KCNJ11 and ABCC8 genes that encode constituents of the β-cell KATP channel. Detection of these mutations allows clinicians to offer treatment with sulphonylureas, which act directly on the KATP channel in an attempt to overcome the clinical effects of the mutation. This allows a more readily administered oral treatment with improved disease control in neonates.[2,3]

The specialty of diabetes has had the good fortune to be able to provide concrete examples of how detection of genetic mutations might influence drug choice in small subgroups of patients; it must be hoped that the application of pharmacogenetics will become more widespread within all areas of medicine to allow improved patient care in the near future.

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Should thrombolysis have a greater role in the management of pulmonary embolism?

Editor – We read with interest the review article by Jenkins *et al* exploring the question of thrombolysis in acute pulmonary embolism, particularly considering the subgroup of patients with right ventricular strain but no haemodynamic compromise (*Clin Med* October 2009 pp 431–5).

They acknowledge the technical problems associated in some patients in obtaining good quality transthoracic echocardiographic images, however this review article, the studies upon which it is based[1–6] and the British Thoracic Society[7] guidelines on pulmonary embolism management do not discuss some of the major limitations of transthoracic echocardiography in the evaluation of right ventricular size and function and estimations of pulmonary artery pressure. This may be part of the reason why the studies investigating thrombolysis in this subgroup of patients have been inconclusive.

Anatomically, the right ventricle has a very complex geometrical shape.[8] Physiologically, its mechanism of contraction is also complicated and not fully understood, with longitudinal and circumferential motion occurring. This makes assessment of its size and function using two-dimensional (2D) and M mode echocardiography less reliable than that of the left ventricle. In our experience, cases of severe right ventricular dilation or systolic functional impairment are usually reliably identified on 2D and M mode imaging, but more borderline cases are more difficult to assess accurately. However, one author had experience last year of a case of an acute ventricular septal defect (VSD) opening following myocardial infarction. Echocardiography allowed excellent images of the right ventricle to be obtained in multiple views and did not suggest any right ventricular dilatation. The patient was severely haemodynamically compromised and underwent emergency surgery on the same day during which a massively dilated right ventricle was found, so large that the surgeon was unable to close the chest following the surgery. We suspect that this was missed by the echocardiogram because 2D views were probably obtained that were off axis to the 3D direction in which the ventricle had dilated.

Measurements of the pulmonary artery pressure can also be misleading if the echocardiographer is not vigilant in their technique. The most common way to evaluate this variable utilises the presence of a tricuspid regurgitation (TR) jet found in over 90% of the population.[9] If performed correctly, the maximum velocity of this jet added to the right atrial pressure (calculated from the size and amount of collapse during inspiration of the inferior vena cava on echocardiography) gives a reliable estimate of the pulmonary artery pressure. The maximum velocity is calculated by placing a line of continuous wave Doppler through the TR jet. The equation used to calculate the maximal velocity of flow assumes that the line of continuous wave Doppler is placed by the echocardiographer absolutely parallel to the flow of the jet. Any misalignment will result in an underestimation of the pulmonary artery pressure. We were interested to find that in one study discussed in Jenkins *et al*'s review, the maximum TR velocity was assessed using the subcostal echocardiographic window.[1] In many of the patients that we have assessed over many thousands of studies, it is uncommon for this view to allow parallel intercept with the TR jet, and hence it is likely that this study underestimated this variable.

Most of the studies discussed in the review regarding the role of echocardiography in right heart assessment were performed many years ago and used a variety of differing techniques to assess this structure, making direct comparisons difficult. Echocardiographic equipment has improved substantially since many of these studies were published, leading to much improved image quality. There are also numerous more modalities available that allow the right ventricle to be more accurately assessed.[10] These include 3D echocardiography, tissue Doppler and strain imaging. However, these modalities will not be routinely used in most non-research centres as yet.

In summary, we would ask general physicians to be aware of the limitations of