EDITORIALS

Are we counting the right thing?

“Of course, there are some cases, which I fear:
Do not admit of diagnosis clear”

In the 1950s, it was not routine to subject an acute admission to extensive testing, yet doctors were usually able to make a diagnosis, often using observation alone. More sophisticated diagnostic tools, an ever-greater expectation for accuracy in diagnosis, pressure from well-informed patients and relatives along with pressure for an accurately coded diagnosis all add to the demands of a modern admission. Indeed, from a medical point of view, the purposes of admission might be to make a diagnosis, to initiate treatment, and to return the patient to a primary care setting with adequate information to continue treatment.

In this month’s journal, Dr Walsh and colleagues from Southampton have used the nationally collected data on hospital admissions to highlight a growing trend in the coding of older peoples’ admissions to hospitals, that is, the rise of no particular diagnosis but a catchall of ‘signs symptoms and abnormal laboratory findings’ [1]. Increasing age is rejected as a cause as is the possibility of a rise in finished consultant episodes (FCEs). It is considered to be due to the increase in hospital admissions, changes in reporting patterns, and, possibly, lack of appropriate community facilities/services.

We need to be clear. Those patients with ‘signs, symptoms and abnormal laboratory findings’ as their primary diagnosis, are patients whom we have failed.

This worrying trend needs further assessment and, until more information is available, it should make those of us responsible for admitting older people, look at our own services from the front door through to the episode being coded. Diagnosis remains key in the treatment of older people, particularly with reference to the intermediate care services that are purported to be able to care for this group.

So what are the problems that might exist in the system to cause this?

In the acute admission setting, one-third of acute medicine physicians responding to the RCP Consultant Census 2006 declared that they also worked as geriatricians, though only one-fifth of this growing specialty have a Certificate of Completion of Training in Geriatric Medicine (RCP London 2006). The increased pressure for reduced length of stay may also push older people out at this stage with no diagnosis. We must be certain that those making early decisions to deal with older people are equipped to do so, and that appropriate follow-up exists to make a diagnosis where it is not done in hospital.

Add to this the fact that elderly patients are often complex, with problems of co-morbidity, more ‘noise’ from such things as positive troponin or d-dimer, and often more difficulty in getting the history.

Although the rise in FCEs is not the statistical cause for the rise of the undiagnosed patient, there may be another effect. FCEs are likely to go up where patients on admission units are subsequently moved to wards operating a ward-based system. Suddenly, continuity of care becomes fragmented, the original history mutated and the thrust of the admission lost. The quest for a diagnosis is superceded by the need to get the patient medically fit for discharge.

FCEs may also increase because of the push to make sure that each component is assessed appropriately, once again losing sight of the whole and merely dealing with the symptom. The cardiologist, neurologist and gastroenterologist may all be needed in the admission, but we must make sure that holistic care is not lost amongst this segregated care.

And the European Working Time Directive creates teams that rarely act as one, thereby leaving continuity of care with the one person who often sees the patient least: the consultant. Once again the quest for a diagnosis is often lost between ward rounds, in favour of discharge.

Next, lengths of stay are falling still and the pressure to discharge is high. We do not have the time for leisurely observation of the evolution of symptoms that we may have done in the past.

Once the patient is discharged, the coding system kicks in. All episodes must be coded to achieve maximum cost effectiveness. The discharge summary is key. Too often, when written by a junior doctor, it is a list of the symptoms that brought the patient in, and not a diagnosis with an easy-to-follow treatment plan. It would be interesting to look at the disparity in codes (and income) in groups of patients where coding is taken from the junior doctors’ discharge summaries, as opposed to those done by consultants.

Some will not be worried about this increasing trend in their locality because they have systems in place that mean that the rapidly discharged patient with no clear diagnosis is seen in the community by an appropriately medically supported intermediate care team, or in the outpatients department. However, it would be revealing to take a look at the cohort described in this paper and see how many were further followed up to make a diagnosis.

Does it matter that we do not fit each patient with a diagnosis?
Androgens, ageing and vascular function

We will never be perfect in achieving a diagnosis in every patient, and the challenge of geriatrics is how difficult it can be in some patients, and indeed, whether it is appropriate in others. An audit of our own ill-defined diagnoses showed that re-admission rates, at 1 month, were 10%, very similar to our better defined patients, and the great majority did not return to hospital within the next year. But that should not make us complacent.

Walsh’s paper should make us all reflect upon the systems that operate for our older people, and the continuum of care from the front door to after-discharge, including the information (clinical and managerial) that goes along with it. We were trained as geriatricians and should continue to ensure that older people get the care they deserve at the time they deserve it, in the place they deserve it.

MATT THOMAS
Consultant Physician, Poole Hospital NHS Foundation Trust, Poole BH15 2JB, UK
Tel: +44 (0)1202 448160; Fax: +44 (0)1202 442993
Email: Matt.Thomas@poole.nhs.uk

Reference

Androgens, ageing and vascular function

Circulating testosterone in males is well known to decline progressively with advancing age, a decline that is paralleled by a number of physiological changes including loss of bone and muscle mass; increased fat mass; impairment of physical, sexual and cognitive functions; loss of body hair; and decreased hemoglobin levels. Indeed, early-onset androgen deficiency produces similar changes [1].

In addition, androgens have an important influence on vascular function: the human androgen receptor is expressed in all vascular tissues, including endothelium, smooth muscle and myocardium [2]. Besides traditional modifiable cardiovascular risk factors, androgens have also been implicated in the relatively higher rate of cardiovascular disease in men. Excess exogenous androgens certainly seem to increase cardiovascular morbidity [3], however, there is growing evidence that at physiological levels androgens appear to be associated with reduced cardiovascular risk [2, 4].

Arteriosclerosis is associated with increased large artery stiffness (or reduced compliance), which inhibits optimal coronary and peripheral perfusion [5]. Simultaneous endothelial degeneration, with impaired release of vasoactive substances has an effect mainly on peripheral vessels where smooth muscle content is higher. These processes interact to contribute to increased cardiovascular morbidity and mortality (Figure 1).

Arteriosclerosis is associated with increased large artery stiffness (or reduced compliance), which inhibits optimal coronary and peripheral perfusion [5]. Simultaneous endothelial degeneration, with impaired release of vasoactive substances has an effect mainly on peripheral vessels where smooth muscle content is higher. These processes interact to contribute to increased cardiovascular morbidity and mortality (Figure 1).

Non-invasive measurement of arterial properties that reflect the severity of arteriosclerosis allows both safe and meaningful assessment of the extent of vascular damage and overall vascular performance. Pulse wave velocity (PWV), which measures large artery stiffness, is the most widely used and independently predicts cardiovascular morbidity and mortality [6]. Other methods for measuring large artery stiffness include augmentation index (AI) [7], systemic arterial compliance (SAC) [8], and measurement of QKD (ECG Q wave to Korotkoff sound) [9]. Impaired endothelial function, assessed non-invasively, has also been shown to predict cardiovascular risk [10]. Endothelial function may also influence large central arteries, and thus, measurements such as PWV and endothelial function are closely related, but not necessarily interchangeable, as they measure different aspects of vessel function [11, 12].

Because arterial stiffness is closely related to chronological age, any deviations from age-predicted normal