2 Murtagh FEM, Preston M, Higginson I. Patterns of dying: palliative care for non-malignant disease. *Clin Med* 2004;4:39–44.
3 Hockley J, Clark D. *Palliative care for older people in care homes*. Buckingham: Open University Press, 2002.

In response (1)

Thank you for the opportunity to respond to the letter by Kafetz and Atkin. They state that our paper 'shows the limitations of an approach grounded in the specialty of palliative care that deals with death from a single pathology, when analysing deaths on acute wards'. First and foremost, we would like to highlight the fact that palliative care does not just deal with death from a single pathology. The World Health Organization definition describes palliative care as 'an approach that improves the quality of life of patients and their families facing the problems associated with life-threatening illness, through the prevention and relief of suffering by means of early identification and impeccable assessment and treatment of pain and other problems; physical, psychological and spiritual'.[1] Of note, there is no reference to diagnosis; the definition encompasses the care of patients with a spectrum of illnesses and prognoses. As clinicians working in this specialty we have daily encounters with patients with complex needs from many 'pathologies' other than cancer.

As suggested in our article, we agree the Liverpool Care Pathway (LCP) is not particularly helpful in 'diagnosing dying' for patients with many non-cancer illnesses. For example, patients with stroke and dementia may be bed bound and unable to swallow tablets, but not dying. For this reason, we used a pragmatic approach for our audit using the LCP criteria and/or case note documentation to determine whether and when a patient had been 'diagnosed as dying'. Kafetz and Atkin suggest many patients over the age of 80 dying of pneumonia have concomitant dementia; in our audit, of the 49 individuals who fell into this age category, 16 had a primary diagnosis of a chest infection and four of these had a documented diagnosis of dementia.

The authors suggest, 'The culture of specific wards for the care of elderly people is to look for what is remediable and palliate what is not'. Surely this is how medicine should be practised across all specialties and all ages, not just for the elderly? They propose that 'geriatric medicine and palliative medicine find ways to make clinicians more confident in 'diagnosing dying''. We agree, and acknowledge that the diagnosis of dying is difficult to make.[2,3] Our current research into end-of-life care on acute hospital wards suggests that a huge cultural shift is needed away from the concept of death as failure, and towards open discussions about death as a possible outcome so that it can be anticipated and planned for. We therefore still believe the key approach is 'to assist clinicians in identifying those patients who might die during their current hospital admission thereby enabling active treatment where appropriate alongside symptom relief and advanced care planning for the future'.[2]

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References

1 World Health Organization. www.who.int/cancer/palliative/definition/en/
2 Gibbins J, McCoubrie R, Alexander N, Kinzel C, Forbes K. Diagnosing dying in the acute hospital setting; are we too late? *Clin Med* 2009;4:116–9.
3 Higgs R. The diagnosis of dying. *J R Coll Physicians* 2000;33:110–2.

Diagnosing dying in the acute hospital setting (2)

Editor – Gibbins and colleagues show that providing end-of-life care is a challenge in hospitalised patients (*Clin Med* April 2009 pp 116–9). We conducted a similar audit in acute medical patients and reviewed case notes of 50 patients who died following admission to the department. We excluded patients who died within seven days of admission as we felt that the clinical uncertainty during this period would be very high. Our results are similar to the findings in the article with 62% (31/50) of patients being identified as having end-stage disease and only 54% (17/31) of them being offered end-of-life care. The Liverpool Care Pathway (LCP) was used in 13 patients. Five of these also received specialist palliative care input. Four patients had specialist palliative care input without the use of the LCP.

We agree with the authors that the uncertainty in diagnosing dying is perhaps a major contributor to patients not receiving palliative care but we feel that other factors, such as frequent transfers of patients between wards, which occurred in 27/50 patients in our audit, and reduced continuity of care owing to shorter shift patterns and frequent junior staff changes, also contribute to delay or denial of end-of-life care.

The majority of the patients in our audit were admitted with an infection and in the majority of the patients the cause of death was infection. It is often thought that infections can be treated despite the presence of other significant co-morbidities. There is little recognition among healthcare staff, patients and relatives that an infection is often the terminal event in most end-stage diseases. A number of such patients would have had previous admissions with similar infections in the past and recovered, which adds to uncertainty about diagnosing the terminal event. In our audit 15 patients had previous admissions within the last two months.

We feel that it is important to discuss with patients and relatives the role of infection as a terminal event in chronic illness, so that they are informed and not alarmed when healthcare staff decide not to treat infection actively. We also feel that the LCP should indicate that in cases of uncertainty it may be appropriate to give antibiotics despite the decision to provide end-of-life care as we feel that this will help healthcare staff to allay their own and their patients' anxieties in instances of clinical uncertainty, thereby promoting wider use of the LCP.

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In response (2)

We thank Wallis and Guptha for sharing their audit results. Their results show that the majority of their patients were admitted with an infection, which was also stated as the cause of death. Our results revealed that infection was the primary reason for admission in 32% of the cohort.[1] Symptoms arising as a result of infection can be similar to those of someone who is dying, and as healthcare professionals working in an environment which focuses on cure, we sometimes treat infections without recognising that the patient is actually dying from their underlying illness.[2–4]

However, we fully agree that the treatment of an infection can be entirely appropriate for patients who may be entering their last days of life – either for symptom control or because the prognosis is uncertain (especially in those with non-malignant disease who have unpredictable disease trajectories). The current version of the Liverpool Care Pathway (LCP) for the dying does not stipulate that all antibiotics should be stopped, but that inappropriate antibiotics should be discontinued.[5] Thus in those patients where appropriate, it is possible for the patient to be on the LCP while receiving antibiotics. However, as suggested by our audit and the work of others, the 'diagnosis of dying' can be difficult to make and thus the 'right time' to place a patient onto the LCP or other end-of-life care pathway can be challenging. We therefore advocate that until sensitive and specific prognostication tools are available, as stated above, we should 'assist clinicians in identifying those patients who might die during their current hospital admission thereby enabling active treatment where appropriate alongside symptom relief'.[1] This approach would enable those patients who require appropriate antibiotics to receive them, while allowing 'healthcare professionals to allay their own anxieties in instances of clinical uncertainty'.

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References

1 Gibbins J, McCoubrie R, Alexander N, Kinzel C, Forbes K. Diagnosing dying in the acute hospital setting; are we too late? *Clin Med* 2009;4:116–9.
2 SUPPORT Principle Investigators. A controlled trial to improve care for seriously ill hospitalised patients: the study to understand prognoses and preferences for outcomes and risks of treatment. *JAMA* 1995;274:1591–8.
3 Ellershaw J, Ward C. Care of the dying patient: the last hours or days of life. *BMJ* 2003;326:30–4.
4 Miller F, Fins J. A proposal to restructure hospital care for dying patients. *N Engl J Med* 1996;334:1740–2.
5 Liverpool Care Pathway. www.mcpcil.org.uk/liverpool_care_pathway

Investigation of the patient with pleural effusion (1)

Editor – Rahman and Munavvar's paper on investigating the patient with pleural effusion (*Clin Med* April 2009 pp 174–8) made no mention of the utility of quantifying pleural fluid adenosine deaminase (ADA) as a diagnostic parameter. Assay of ADA is simple and inexpensive, coupled with a relatively high sensitivity and specificity in identifying tuberculous pleuritis, enhanced further when the effusion in question is richly lymphocytic.[1,2] While the positive predictive value of ADA may be lowered by its use in areas of low tuberculosis (TB) prevalence, its negative predictive value should remain unaffected, retaining its overall value as a subsidiary 'rule out' test for suspected TB pleural disease.[3] Furthermore, ADA levels falling below the diagnostic cut-off threshold (around 40 U/l) have been shown to virtually exclude TB as a cause of pleural effusion even when the collection is lymphocytic.[3,4] We suspect that many physicians continue to find the analysis of ADA useful in the setting of suspected TB pleural sepsis while eagerly awaiting the emergence of newer immune-based tests of pleural fluid.

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References

1 Valdes L, Alvarez D, San Jose E *et al.* Value of adenosine deaminase in the diagnosis of tuberculous pleural effusion in young patients in a region of high prevalence of tuberculosis. *Thorax* 1995;50:600–3.
2 Burgess LJ, Maritz FJ, Le Roux I *et al.* Combined use of adenosine deaminase with lymphocyte/neutrophil ratio. *Chest* 1996;109:414–9.
3 Jimenez Castro D, Diaz Nuevo G, Perez-Rodriguez E, Light RW. Diagnostic of adenosine deaminase in nontuberculous lymphocytic pleural effusions. *Eur Respir J* 2003;21:220–4.
4 Lee YC, Rogers JT, Rodriguez RM, Miller KD, Light RW. Adenosine deaminase levels in nontuberculous lymphocytic pleural effusions. *Chest* 2001;120:356–61.

Investigation of the patient with pleural effusion (2)

Given the fact that culture of pleural fluid (using Lowenstein medium) has only 36.6% sensitivity for diagnosis of tuberculous pleural effusion versus 79.8% sensitivity obtained with the more invasive modality of histological identification of caseating granuloma,[1] among the non-invasive 'special tests' for evaluation of pleural effusion,[2] mention should also have been made of adenosine deaminase (ADA)[3] and interferon-gamma (IFN-gamma),[4] so as to complement strategies such as staining and culture for acid-fast bacilli.[2] In a systematic review of 63 studies ADA was characterised by positive likelihood ratio 9.03 (95% confidence interval (CI) 7.19 to 11.35), negative likelihood ration 0.10 (95% CI 0.07 to 0.14) for diagnosis of tuberculous pleural effusion.[3] Correspondingly, in a systematic review of 22 studies, diagnostic accuracy of IFN-gamma was characterised by positive likelihood ration amounting to 23.45 (95% CI 17.31 to 31.78), and negative likelihood ratio 0.11 (95% CI 0.07 to 0.16).[4] In the setting of tuberculous effusion prevalence of 5%, post-test probability of a negative ADA test has been estimate to be 0.4%, increasing to 2.4% where tuberculous pleural effusion has a 25% prevalence. For INF-gamma, corresponding post-test probabilities are 0.22% and 1.2% respectively.

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