and disadvantages of care closer to home. What patients want may not be what they need, nor what the nation can afford. Peripatetic specialists are inefficient and re-inventing the cottage hospital network (now termed polyclinics) is unaffordable – have we forgotten that many cottage hospitals disappeared in the 1980s because they were too expensive to run? Clinicians should only support systems that are financially sound. It is illogical to argue that emergency care must be centralised on fewer sites to improve results while potentially disseminating all other care.

Services may be provided more cheaply 'in the community' but they may not be better. There is nothing wrong with hospitals as specialist bases – to maintain clinical research; high-quality training; and good clinical governance. Professional isolation is avoided and multidisciplinary teamwork facilitated. In my own area the primary care trust (PCT) has (without discussion with its local specialists) commissioned a rheumatology service within general practice run by a retired consultant. It is one-third to one-half the cost of the current Payment by Results (PbR)-based secondary care alternatives. The reduction in hospital workload if patients are diverted to such cheaper alternatives threatens the viability of existing services.

The Royal Colleges of Physicians, Paediatrics and Child Health, and GPs agree that positive incentives are required in the NHS. The barriers between primary and secondary care must be removed and we must talk of general and specialist care, between which patients can move seamlessly, unhindered by financial issues. This is the principle behind Teams without Walls and requires vertical integration of provider services.[1] All competing services will then be on a level playing field. Conflict between GPs and specialists disappears. PbR, which forces PCTs to pay prices they cannot afford to hospitals that cannot reduce their prices without going bust, collapses. There are positive models already. In Stoke-on-Trent the musculoskeletal service is now managed by primary care (with substantial cost savings) but the current pattern of delivery has been maintained.

Do we need practice-based commissioning? The integration of general and specialist care removes its raison d'être. It allows local accountability but adds a huge layer of financial management to a bankrupt system and hinders the process of specialist referral.

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References

1 Royal College of Physicians, Royal College of General Practitioners, Royal College of Paediatrics and Child Health. *Teams without Walls: the value of medical innovation and leadership*. London: RCP, 2008.

Conflict of interest

At the time of writing, the author was President of the British Society for Rheumatology and a member of Council of the Royal College of Physicians, London.

In response

Many thanks for your reply to my article. I share your concerns and one of the great difficulties of practice-based commissioning (PBC) is that it is politically driven and the 'goal posts' are continually moving. The article I wrote was to summarise my current understanding of PBC, but not my opinion of the process.

In the article I cited two major concerns. Firstly, with the future possible role of private providers and so a plurality of providers, would PBC lead to improved efficiency or a path to further privatisation and de-unification of the NHS? Secondly, general practitioners (GPs) and consultants are becoming opponents in a bidding war, rather than colleagues with different areas of competence who currently cooperate in the management of patients.

It is therefore of particular concern to all of us who work as traditional NHS providers to observe this process which has no obvious evidence base and could potentially fragment and destabilise the NHS.

Bamji has made a very important observation and will perhaps be quoted for years to come on the concept of moving from the terms; primary and secondary care, to generalist and specialist care and therefore working as a 'team without walls'. It is imperative that we use these skills appropriately for the benefits of patients in the environment of the patient's choice and not those based on political visions. Competition between potential service providers should be avoided, but rather ensuring that the most appropriate practitioner is made available to treat a patient with a particular illness. A service provider based on cost rather than identified health need and the practitioner most appropriately skilled to meet that need, will compromise patient care.

There is a myth that care in the community is cheaper and equivalent and that it should be run by GPs with a specialist interest (GPwSIs). This has yet to be researched fully and like many recent changes in the NHS it should be piloted before it is implemented. Bamji is right, we should avoid 'reinventing the wheel' and concentrate in the areas of generalism and specialism where we endeavour to excel and the associated environments where that care is provided. We are currently losing the concept of shared care which is being undermined by locally enhanced PBC services which can actively discourage primary care from involving secondary specialist services, eg, in diabetes care.

Phrases often quoted that I find difficult are, 'we live in interesting times' and 'shaping the future'. In relation to the NHS it would be more true to say that as GPs and consultants we live in worrying times, particularly for our patients. I do share your concerns and thank you for writing.

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Lesson of the month (1)

Editor – I read Chirwa *et al's* lesson of the month (*Clin Med* February 2008 pp 107–8) with interest but would like to raise a few points. Modern computed tomography-based radiotherapy planning systems allow accurate calculation of the dose to specified areas of the brain making the phrase 'it was possible that radiation spread to the brain' imprecise and unnecessary. Most radiotherapy treatments for tonsillar carcinoma are carefully planned to avoid significantly irradiating the brain unless tumour location makes this necessary. No staging details are given to suggest why any volume of the central nervous system (CNS) should have

been irradiated to any significant dose. Without supporting dose/volume information, brain irradiation as an aetiological factor remains just a possibility.

It may be that authors feel that the significant pathophysiology relates to irradiation of major blood vessels outside the CNS although the article fails to clearly make this distinction. If so, there is no need to invoke the notion of radiation 'spreading' to the brain. If this is the hypothesis, then knowledge of the patient's smoking habits and alcohol intake (both strongly associated with squamous cell carcinoma of the upper aero-digestive tract in a 71-year-old male who also has diffuse white mater changes suggestive of ischaemia) would be pertinent, as would the presence or absence of carotid bruits. None of these details are mentioned. The time course of the events makes this possibility less likely as large vessel damage after radiotherapy often takes years to develop rather than the weeks in this case.

It may be that radiotherapy contributed to events but the evidence presented fails to support this assertion. I would therefore question whether this does constitute the first case of serotonin syndrome secondary to fluoxetine precipitated by a radiation induced cerebral vasculopathy to be described. Was the treating clinical oncologist consulted?

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Lesson of the month (2)

Editor – I read with interest Chirwa *et al*'s lesson of the month (*Clin Med* February 2008 pp 107–8). I feel, however, that the authors' premise of the underlying pathophysiology of radiation-induced cerebral vasculopathy is flawed.

Insufficient clinical details were given in order to determine the specifics of the radiotherapy their patient may have received. In general, radiotherapy for a tonsillar carcinoma is delivered to the primary tumour, or tonsillar bed if given post-operatively, and to the cervical lymph nodes at risk. These would include the ipsilateral level Ib-IV nodes (T1–2, N0) or bilateral Ib-V nodes if T3–4 or N+.

Radiotherapy is precisely delivered to a specific target volume. The target volume as defined above would usually be delineated on planning a computed tomography scan and it would be unusual to include any cerebral tissue within the treatment field.

Radiotherapy has both early and late effects. Early normal tissue toxicity occurs during radiotherapy and in the 90 days following radiotherapy. Late toxicity is defined as changes persisting or occurring more than 90 days after completion of radiotherapy.[1]

Stenosis and occlusion of vessels has been reported as late toxicity and would usually develop within the first eight years after treatment, with a latent period reported as 2–25 years.[2] Although this gentleman may be at risk of carotid artery stenosis and thus potential cerebral hypoxia, we would not anticipate the development of this within the time frame of 12–16 weeks post treatment.

The authors cite the paper by O'Connor *et al* in support of the argument for cerebral vasculopathy.[3] This paper, however, discusses radiotherapy for central nervous system neoplasms and cerebral arteriovenous malformation and as outlined above we would not expect the cerebral vasculature to receive a significant radiotherapy dose in the treatment of a tonsillar carcinoma.

I would therefore challenge the authors' claims that radiation induced vasculopathy is the underlying mechanism responsible for the development of their patients acute neurological deterioration.

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2 Bitzer M, Topka H. Progressive cerebral occlusive disease after radiation therapy. *Stroke* 1995;26:131–6.
3 O'Connor M, Mayberg M. Effects of radiation on cerebral vasculature: a review. *Neurosurgery* 2000;46:138–51.

In response

Many thanks to Button and Dorey for comments on our article on serotonin syndrome. We are grateful for the points raised. I agree that no mention of our patient's smoking and alcohol ingestion habits were mentioned, as well as the dose/volume of radiation administered for the treatment of the tonsillar ca and these were important in this case. I can, however, say that the patient was an ex-smoker and clinically he had no carotid bruits.

The point you made of large vessel damage due to radiation being a delayed response is entirely true hence the 12–16 week period in this case could not account for this. There are case reports where the large vessel vasculopathy occurred as long as twelve years later. However, it is also well known that endothelial cells are perhaps the most radiation-vulnerable elements of the mesenchymal tissue and that the injuries occur often in capillaries, sinusoids and small arteries in that order. Our hypothesis therefore is that the radiation precipitated these events by the injury caused to small vessels with subsequent damage to the endothelium hence affected serotonin metabolism.

The conclusion in our discussion paragraph states that cerebral vasculopathy precipitated the events and this was drawn from this hypothesis.

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