

I am speeding around the community doing rheumatology clinics, how can I manage my rehabilitation unit? Multi-tasking is possible on one site, but not on many. Trainee teaching will collapse as the diversity of case-mix, a virtue of service concentration, will be lost. The opportunity to collect cases for clinical trials will dissipate. Departmental morale will collapse. A two-tier service of superspecialist care in big teaching centres and barefoot specialist care elsewhere will develop. These are not my arguments alone; I asked my chronic disease 'focus group' (our local National Rheumatoid Arthritis Society network group) whether they would prefer to be seen by me in the hospital clinic or in their own GP surgeries. Without exception they expressed a preference to be seen at the hospital, citing many of the above concerns. Ask the wrong people and you get the wrong answer.

Concentration brings benefits. The clearest example of this is surgical; in the first world war facial injury care for Great Britain and the Dominions was concentrated in one hospital (mine, as it happens) and the advances in plastic surgery thereby generated were unmatched on the continent where facial injury was dealt with in a fragmented way. Furthermore the patient support that grew from this obviated the need for a self-help group, whereas in France 'les gueules cassées' developed because of the isolation and dispersion of sufferers.³ To create a specialist diaspora will recreate the disadvantages of dilution. We must learn from history.

Lastly, Care in the Community often means very little, or no, care. As social service budgets contract and input from carers diminishes we have already seen the adverse effects and must do everything we can to avoid this in medicine.

That is not to say that hospital-based care is cheap or that we should not look for ways of making it cheaper, for example by running telephone clinics for those on long-term follow-up. As Patterson points out, hospitals are encouraged to maximise income, while PCTs try to limit access because Payment by Results (PbR) tariffs are unaffordable. But we do not need to disperse specialists to address this; as the musculoskeletal services in Stoke and Bolton have shown it may be possible to avoid sub-

stantial transactional costs by changing management from acute trust to PCT without necessarily altering the physical structure of the service. We should also remember that those services turning a profit in an acute trust (rheumatology outpatients is one) will prop up the loss leaders (acute medicine is one). So pulling out profitable services may compromise the whole of acute hospital-based care – unless the purchaser-provider split is abolished, which, for me, would be the essential and final outcome of Teams without Walls.⁴

I firmly believe that care closer to home is a concept based on flawed research and the turning of a blind eye to economic reality. Specialist medicine as a whole will be seriously damaged if we fail to examine its risks.

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Conflict of interest

AB is employed by an acute trust.

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- 3 Harold Gillies: surgical pioneer. *Trauma* 2006;8:143–56.
- 4 Royal College of Physicians. *Teams without walls. The value of medical innovation and leadership*. London: RCP, 2008.

In response to both

I agree with Dr Cohen that general practitioners (GPs) with a special interest have an important role. It is essential that they feel part of the specialist service and participate in audit, continuing professional development and so on with specialist colleagues.

They also need to be able to discuss patients easily with consultants and to access more specialist opinion when needed, as well as bringing their expertise as to how patients can be managed in the community.

Dr Cohen also makes the point that the challenge is not to deliver more of the same just in a different location – which very well answers Dr Bamji's concerns. Moving expertise into the community, working more closely with GP colleagues, community nurses and other professionals to deliver consultant input in a different way is not just an argument about geography. There are undoubtedly logistical difficulties in providing services in different places, but these can be overcome (and many consultants already deliver outpatient services in locations away from their home base). The point of consultants working in community settings is to develop better pathways of care which are more joined up across the old primary–secondary care boundaries and to truly build Teams without Walls.

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Managing capacity and demand across the patient journey

Editor – Walley and colleagues recently highlighted the problem of reduced bed capacity which has an impact on coping with healthcare demand (*Clin Med* February 2010 pp 13–5).

I would like to comment on the long-term planning and that bed requirements are based on average demand and average length of stay, the author felt that this can create a problem as once there is random variation in demand and staff capacity, bed shortages will occur. I do not feel that we have a bed shortage in England. However, the discharge process is patchy and lengthy and there is a lack of coordination between hospital staff or secondary care and primary care as well as between NHS and social services.

I agree with the author that a 'systems' approach is the only solution where healthcare staff and social services, primary and

secondary care work collaboratively. We may need a unified bed management team in every primary care trust which can allocate the patient after initial assessment to an acute hospital, a community hospital or to intermediate care. This requires training and commitments from all staff. The whole health and social care system should plan together how to meet the demand of the increasing elderly population. I should also emphasise the importance of multidisciplinary teams in each trust for effective discharge planning.

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'With a pinch of salt' revisited

Editor – I read with interest but some concern the recent lesson of the month by Gangopadhyay *et al* (*Clin Med* February 2010 pp 86–7). The lesson highlights a case of severe hyponatraemia which the authors attribute to excessive sweating, poor fluid consumption and low salt intake in a hot environment. There is little evidence to suggest that low salt intake would contribute to this event and the authors have not explored alternative likely explanations. During evolution mankind has survived with very little salt in the diet. Even in modern times, this evidence is detectable in the Yanomano and Xingu Indians living in the humid and hot environment of the Amazon jungle.¹ Their average salt intake, when measured by 24-hour urine collections, varies between 1 and 10 mmol/day. These levels of salt intake, however, are almost unseen in the western world due to the high salt intake we are exposed to, even when adhering to a low salt diet. Under conditions of exercise in a hot environment, a low salt intake does not impair the ability to exercise, and it does not cause changes in plasma sodium, potassium, osmolality or sweat rate, although the salt content of sweat is reduced on a low salt diet.² They misquote the evidence in athletes and the military where the high morbidity from hyponatraemia is due to overhydration (ie too much water) rather than a low salt intake. The case presented here is clearly a case of diuretic abuse, surreptitious vomiting or laxative abuse, as we described in the

past in a different scenario.³ While hyponatraemia may possibly have been caused by water intoxication, it would not have caused plasma potassium to fall so low, or the renin–angiotensin system to be so stimulated. The authors do not seem to have considered screening for diuretics. Diuretic abuse would explain hyponatraemia, alkalotic hypokalaemia, and activation of the renin–angiotensin–aldosterone system as described here. A moderate reduction in salt intake (up to 3 g per day) does not raise cause for concern and should be recommended to everyone to prevent cardiovascular disease and other common conditions like kidney stones and osteoporosis.⁴

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Serum sodium disorders: safe management

Editor – I suspect that Wakil and Atkin were set an impossible task, in reviewing the aetiology, assessment and acute management of hyponatraemia and hypernatraemia in three pages (*Clin Med* February pp 79–82)! The 2007 American guidelines on hyponatraemia alone run to 21 pages, including 120 references.¹ However, the authors of the CME acute medicine review covered this complex topic in a readily accessible manner, for which they should be commended. However, I fear that a number of important and clinically relevant points were not highlighted due to space limitations. Firstly, the contribution of excessive

water intake to hyponatraemia should be stressed. Although classically presented as a psychiatric condition of psychogenic polydipsia, water intoxication is an important differential diagnosis for hyponatraemia. It can also cause a diagnostic challenge and contributes to many cases of hyponatraemia. This was evident in the lesson of the month, published in the same edition of *Clinical Medicine*, where a young man presented with hypovolaemic hyponatraemia.² Secondly, it should be stressed that in older patients with low serum sodium levels, there are often multiple contributing factors. Diuretic therapy may promote hypovolaemia; co-morbidities such as chronic kidney disease or heart failure cause a tendency to hypervolaemia. At the same time, underlying diseases or other medicines such as tricyclic or selective serotonin-reuptake inhibitor antidepressants may cause inappropriate antidiuretic hormone (ADH) secretion. However, the authors' advice that, where there is doubt, isotonic saline should be given is probably valid; but the response to this therapy may be unpredictable. Finally, I worry that the review lacked sufficient detail on pharmacological therapy, advocating the use of new aquaretic drugs but without mention of demeclocycline, which is still commonly prescribed. However, I would strongly counsel against the use of such agents in the acute setting and only where there is a clear diagnosis (with an underlying cause for) inappropriate ADH secretion.

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In response

We thank Aspray for the comment on our article. In answering the first point we