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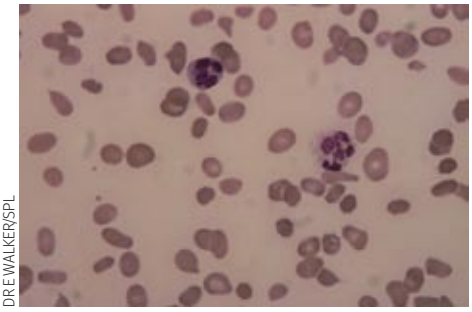
LETTERS

VITAMIN B-12 DEFICIENCY

Monitor for up to two years after symptoms resolve



DR E WALKER/SPL

Hudson presents a 10 minute consultation on vitamin B-12 deficiency.[1] Monitoring is required in patients with pernicious anaemia after symptoms resolve, particularly for the first few years after diagnosis. Data from large population studies on Chinese, European, and American patients show that the risk of cancer is greatest in the first one or two years after diagnosis.[2-5] The risk is greater in those with detectable intrinsic factor antibodies, but patients without detectable antibodies are also at risk.[2]

A large Swedish study of 4517 patients with pernicious anaemia and 20 years of follow-up found an excess risk of gastric and other cancers (standardised incidence 2.9, 95% confidence interval 2.4 to 3.5), the risk being particularly great in the first year after diagnosis (7.4, 5.3 to 10.1).[3] An American population of 5161 males with pernicious anaemia and a mean follow-up of 6.8 years also showed an excess risk of stomach cancer (3.2, 2.2 to 4.6), again most pronounced within the first year after diagnosis (7.6, 3.9 to 13.3).[4] A Danish cohort of 5072 patients with pernicious anaemia and up to 15 years' follow-up also reported an excess risk of stomach cancer (2.4, 1.7 to 3.1).[5]

Overall, these data recommend monitoring during ongoing follow-up for at least the first two years after diagnosis.

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Competing interests: None declared.

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Consider non-invasive follow-up and malabsorption in old age

Hudson describes the classic clinical signs of vitamin B-12 deficiency in an elderly patient and recommends traditional diagnostic procedures such as measuring vitamin B-12 values and determining antibodies against intrinsic factor and parietal cells.[1] However, newer non-invasive, functional blood tests such as measurement of total homocysteine, methylmalonic acid, and holo-transcobalamin II values would allow confirmation of the clinical diagnosis and even monitoring of the early response to treatment.[2]

Hudson focuses primarily on pernicious anaemia as the most likely cause of vitamin B-12 deficiency in elderly people. However, malabsorption of vitamin B-12 in food, which is closely associated with atrophic gastritis—a condition with a high prevalence in elderly people—may be the leading cause (in 60-70% of cases) of mild vitamin B-12 deficiency.[3] Long term atrophic gastritis may not only be associated with impaired ability to release vitamin B-12 from food products or its binding proteins but may also cause impaired iron absorption.[4] Awareness of the association between malabsorption of vitamin B-12 in food and impaired iron absorption is therefore clinically important, and iron status should be monitored during supplementation with vitamin B-12. Indeed, a rapid haematological response to vitamin B-12 supplementation may result in rapid depletion of iron stores.

Patients with malabsorption of vitamin B-12 in food respond perfectly well to oral vitamin B-12 treatment, which is cheaper and more convenient than parenteral supplementation.[3]

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Competing interests: None declared.

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PREDICTING CARDIOVASCULAR RISK

Using risk scores with patients

Scott focuses on the clinician as decision maker using cardiovascular risk scores.[1] However, I and many of my colleagues in general practice use Framingham and QRISK scores not only to decide on the need for primary prevention but also to try to communicate risk to patients, and persuade them to take potentially unpleasant drugs or change their lifestyle to reduce their risk when they do not have symptoms.

Scott states that the cardiovascular risk tools may not be readily accessible in busy practice settings and cites a 15 year old paper in support of his thesis.[1 2] Primary care has moved on in those 15 years, and all general practitioners in the UK have ready and routine access to these risk scores through integration of risk calculators with clinical computer systems. Other practitioners can use internet based scores or a paper version—for example, in the *BNF*. Lack of access to these tools in modern primary care in the developed world is not a reasonable excuse for guessing risk and prescribing potent preventive drugs.

Researchers are keen to develop the next risk assessment tool, but more effort is needed to get these tools into practical use by clinicians and to develop better and more effective ways of communicating the risks to patients with meaningful outcomes in terms of health improvement. Some methods of communicating risk using heart age or comparisons with normal risk have been mathematically modelled but not researched in primary care.[3] There is a pressing need to find ways of translating validated risk scoring into user friendly, patient friendly, understandable, and effective communication tools with real benefit.

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Competing interests: None declared.

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How many are missed?

Figure 2 in Collins and Altman's research article well illustrates the simple issue raised by Wald and Simmonds in their response: How many do we miss?[1 2]

My eye tells me that QRISK2 misses roughly two thirds, and Framingham misses roughly a half.

In my general practice simply targeting all men over 50 would pick up two thirds of the 155 people having a stroke or developing coronary heart disease before the age of 70.

Neither QRISK2 nor Framingham/NICE performs particularly well, in that they both miss most people destined for cardiovascular disease.

Nearly two thirds of the patients in my practice who develop cardiovascular disease before the age of 70 are in fact at calculated risks less than 20% by Framingham, and probably even more so by QRISK2. The table shows the distribution of known cardiovascular disease in Framingham risk cohorts.

How many angels are not able to dance on a poor predictor's pin?

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Competing interests: None declared.

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Distribution of cardiovascular disease by Framingham risk in one practice

	Framingham risk (%)							
Patients	0	10	20	30	40	50	60	70
All ages								
Cohort	3191	1248	754	377	161	48	9	2
With CVD	42	106	105	75	40	13	4	0
Age <70								
Cohort	3166	1025	445	138	43	4	0	0
With CVD	36	47	31	10	6	1		

CVD=cardiovascular disease.

VOLUME-MORTALITY FOR CYSTECTOMY

Centralisation of cancer services vindicated

Mayer and colleagues evaluated the relation between volume and mortality for radical cystectomy in England.[1] Their finding—that patients undergoing surgery in medium volume hospitals have a poorer outcome than those in low volume hospitals—contrasts with most published data.[2 3]

A weakness of Mayer and colleagues' study is that they report only short term outcomes after radical cystectomy—namely, in-hospital and 30 day mortality. Mortality after cystectomy has been reported to rise significantly beyond 30 days.[4]

To evaluate whether hospital volume influences longer term outcomes after cystectomy, we identified patients undergoing radical cystectomy, again within the hospital episode statistics database, but slightly more recently (2002-3 and 2007-8).

Like Mayer and colleagues, we found that 30 day mortality was not statistically different between the hospitals of different volumes (low 3.0%, medium 2.7%, and high 2.4%; P=0.22). However, when we evaluated 90 day mortality, high volume hospitals significantly outperformed both medium and low volume hospitals (low 7.6%, medium 6.2%, high 5.7%; P=0.007).

The National Institute for Health and Clinical Excellence (NICE) recommends that radical cystectomy be performed only in hospitals doing more than 50 pelvic cancer procedures a year.[5] Although we did not adjust for structural and processes of care as Mayer and colleagues did, our results support this guidance. When outcomes of pelvic cancer surgery are being evaluated, analysis of 30 day mortality may result in erroneous conclusions.

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Competing interests: JK is chair of the NCRI Bladder Clinical Studies Group.

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WHY MEDICINE IS OVERWEIGHT

Supplier induced demand

Martyn makes a mistake in thinking that patient demand alone is making medicine overweight.[1] He has ignored supplier induced demand.[2]

Jack Wennberg, "the father of variation studies," points out that every doctor who graduates in the United States immediately creates lifetime medical costs of around $5-6m. It's very uncomfortable for doctors and hospitals to think that they are creating demand, but they are. And they don't recognise it: read Gawande's article in the *New Yorker* in which he spoke to the doctors in McAllen, Texas, which has the highest health costs in the US.[3] We deceive ourselves if we think supplier induced demand is a US phenomenon. We have it here, it's just that the US has better data on costs and activity.

NHS costs have risen not just because patients want more but also because there are more doctors with more to offer—some of it, as Martyn points out, of dubious (or even negative) value.

Despite their promises during the election the government will have to make cuts in the NHS—even if it calls them "efficiency savings." I suggest cutting the salaries of everybody earning over £20 000 a year by 5% (or 10% if necessary) and closing at least one medical school.

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Competing interests: RS is an honorary professor in Warwick and on the council of St George's, University of London, so he would be sad if either of them closed, but he wouldn't lose financially as he is not paid for either position.

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