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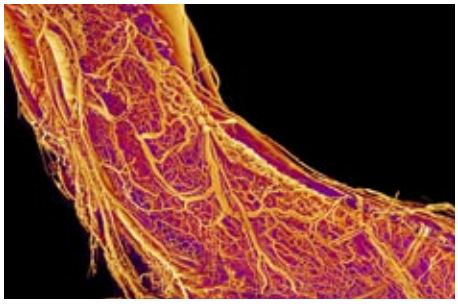
LETTERS



SUSUMU NISHINAGA/SPL

BENEFITS OF SCREENING

Erectile dysfunction predicts cardiovascular risk in men

I am disturbed to see an article on cardiovascular risk that totally ignores the massive evidence base linking erectile dysfunction with cardiovascular risk.[1] For years, I have seen patients referred with erectile dysfunction after a coronary event to hear that they developed erectile dysfunction two to three years beforehand, went to their general practitioner, but were dismissed. The facts are as follows.

Men live seven to eight years less than women. Erectile dysfunction is the manifestation of vascular disease in smaller arteries and gives a two to three year early warning of myocardial infarction. Erectile dysfunction carries a 50% additional risk of coronary events, a level comparable to moderate smoking or positive first degree relative family history. Erectile dysfunction in type 2 diabetes is a better predictor of coronary risk than HbA1c, hypertension, microalbuminuria, or hyperlipidaemia. Over 50% of men with type 2 diabetes are hypogonadal, which carries a 60% additional risk of early cardiovascular death.[2-5]

Despite this evidence we don't even screen for erectile dysfunction or low testosterone in type 2 diabetes or patients with coronary heart disease. We prescribe drugs for coronary heart disease that make erectile dysfunction worse, even though there are drug treatments as effective which improve it, and then make the patients pay privately because we treat erectile dysfunction as a recreational or "lifestyle" issue.

Continuing to ignore these issues on the basis that cardiologists feel uncomfortable mentioning the word erection to their patients is no longer acceptable and probably clinically negligent.

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Competing interests: None declared.

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TRANSIENT ISCHAEMIC ATTACK

TIA or stroke with transient overt signs?

Rapid and direct assessment in secondary care is the key to successful management of transient ischaemic attack (TIA) and stroke.[1] A continuing challenge is the persistent notion that such critical events should be channelled through primary care, with attendant delays, in a way that would be unthinkable for a "major" stroke.[2]

A radical rethink of our descriptors of stroke disease is timely. Unhelpful terminology can obstruct treatment and cloud priorities.[3] In addition to dropping the misleading couplet "minor stroke", we should also abandon TIA, a term that predates modern knowledge of stroke and sophisticated neuroimaging.[4]

Increasingly, the benefit of urgent assessment and intervention,[2] and evidence of subtle persistent neurological deficits after TIA,[5] are eroding the distinction between stroke and TIA. Stroke with transient overt symptoms (STOS) would provide a better match with patient needs, accurate definition of the syndrome, and ideally prompt urgent assessment and management directly with stroke specialist services.

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Wrong conclusion

Lasserson and colleagues show that people trust their own general practitioners more than NHS Direct or out of hours services organised by the primary care trust.[1] No surprise there. But the researchers' conclusions are wrong.

Strokes and transient ischaemic attacks (TIA) cannot be investigated or treated in a standard general practice surgery; most of us do not have a computed tomography scanner or access to intravenous thrombolysis. The best place for these patients is a hospital, and as quickly as possible. Arranging to see your GP is merely a delay when time is of the essence.

The correct conclusion is that we need to educate people about the symptoms of a stroke/TIA (now sometimes referred to as a brain attack) and encourage them to head directly to hospital. In the current atmosphere of government led GP bashing the authors' conclusion is at best mistaken.

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Competing interests: None declared.

1 Lasserson DS, Chandratheva A, Giles MF, Mant D, Rothwell PM. Influence of general practice opening hours on delay in seeking medical attention after transient ischaemic attack (TIA) and minor stroke: prospective population based study. *BMJ* 2008;337:a1569. (18 September.)

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WHAT HAPPENED TO THE POLYPILL?

Why is there more heat than light concerning the polypill?

Watts's question—why are there still so few polypill trials?—is reasonable.[1]

The big issue is not the finer points of the initial target population, the components, or professional opinion on the right balance

between the perfect and the possible. The big issue is the huge gap in funding for research and development of affordable "applied innovations." Formulation, manufacture, testing, and clinical trials of a polypill to European Medicines Agency (EMEA) and Federal Drug Administration (FDA) requirements will cost well over £10m. Generics pharma is geared to high volume, low margin products, but these margins typically cannot support research and development on this scale. Big pharma is unlikely to develop a pill that could be available for a few pence a day. No other sector has yet begun to address this funding gap.

As well as changing the economic incentives for industry, we need large scale, coordinated, public funding. Filling the research and development gap is a major challenge for translational research.

The other major issue brought to the fore by the polypill is the strange reticence about preventing cardiovascular disease. Can you imagine such delay if leading scientists proposed a credible solution to preventing most forms of cancer?

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Competing interests: AR and AP are involved in raising funding for research on affordable medicines.

1 Watts G. What happened to the polypill? *BMJ* 2008:337; a1822. (26 September.)

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News of the polypill

Watts asks what happened to the polypill for primary prevention of cardiovascular disease.[1] We can provide an answer.

The University of Birmingham and Tehran University of Medical Sciences have completed a randomised controlled trial of a four component polypill in approximately 500 men aged 50 to 79 and women aged 55 to 79. This is a pilot study, with the aim of starting a fully powered clinical trial if this is successful. The polypill consists of aspirin, a statin, an angiotensin converting enzyme inhibitor, and a thiazide diuretic. The trial is registered with www.controlled-trials.com.[2] We aim to determine the effects of treatment on blood pressure and lipid concentrations. Recruitment began in 2006 in Golestan (northeast Iran), and follow-up was completed earlier this year. We are currently analysing data.

Our pilot study keeps close to the original spirit of the polypill proposed by Wald and Law[3] by including only people who were not currently eligible for antihypertensive and lipid lowering treatment. Furthermore, we have located our trial in a developing country, where uptake of preventive treatments is far from ideal.

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Competing interests: None declared.

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Cite this as: *BMJ* 2008;337:a2160

TROPHOBLASTIC DISEASE

Analytical problems associated with HCG assay

Sebire and Seckl state that gestational trophoblastic disease is monitored by measuring serum human chorionic gonadotrophin (HCG), but they do not discuss the analytical problems associated with this assay.[1]

Numerous reports of interference have been made since 1984, the most important by Rotmensch and Cole in 2000.[2] They reported on 12 women who had a false diagnosis, 11 of whom had been subjected to needless, and in some cases quite extensive, surgery or chemotherapy. The problem has not disappeared and we still uncover cases of assay interference in women who have serial HCG measurements for pregnancy testing. The frequency of HCG interference is unknown, but our study of thyroid and gonadotrophin assays found clinically relevant interference in 0.5% of all assays.[3]

A further problem is in the variation between assays provided by different manufacturers. Mitchell and Seckl clearly showed this in a patient with tumours whose HCG was detectable in some assays but undetectable in others.[4]

HCG assays are approved by the regulatory authorities for pregnancy testing alone, and the risk to patients with trophoblastic disease or germ cell tumours will continue until assays are formally validated for these diseases.

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Competing interests: None declared.

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TECHNOLOGY COMES TO THE RESCUE

A web based computer program using video clips offers help

J NORDEN/IBL/REX

Paddock et al do not mention that there is a national shortage of British Sign Language (BSL) interpreters.[1] With approximately 70 000 registered BSL users and only around 400 qualified BSL interpreters, the chance of booking a BSL interpreter for the same day is small.

SignHealth, a healthcare charity for deaf people, has therefore developed a web based computer program—SignTranslate—to be used by clinicians when a qualified interpreter is not available. The program translates over 300 medical questions into BSL by means of short video clips. It also translates the questions into 12 foreign spoken languages. It offers an exceptional chance to improve communications between patients and healthcare professionals.

SignTranslate has been endorsed by the Department of Health and is currently available free of charge to all general practices in England until 31 July 2009. An additional benefit is that for more complex consultations it is possible to access via the internet fully qualified BSL interpreters by using a simple webcam.

As part of an initiative to improve access to primary health care for deaf patients, SignTranslate can be accessed instantly from an internet link in the menu bar of the EMIS clinical system. This automatically opens the homepage, where you will be asked to key in your national practice code to gain access. Non-EMIS users can still access the program for free via the SignTranslate website (www.signtranslate.com).

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Competing interests: None declared.

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Cite this as: *BMJ* 2008;337:a2164