

Brains and mobile phones

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BMJ 2006;332;864-865 doi:10.1136/bmj.332.7546.864

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Notes

tions. We know next to nothing, for example, about the predictors of major bleeding on warfarin, or the characteristics that make it likely that the benefits of endarterectomy will outweigh the risks in a patient with asymptomatic carotid stenosis.

The lack of basic clinical research on the issues that matter most to patients and practising clinicians inevitably calls into question whether medical academia, as currently constituted and funded, is properly fit for purpose. Basic biological research and bench-to-bedside translation are obviously important, but why has so much critically important basic clinical research not been done? Whatever the causes (some possible ones are given in the box), medical academia must improve its performance or, less preferably, be forced by politicians to prioritise appropriately. The recent

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emphasis on the development of clinical research is welcome, as are the recent UK Department of Health proposals for future research funding, although there are potential pitfalls. Greatest of these is the tendency for clinical research to be defined too narrowly as being only bench-to-bedside translational research, large scale epidemiology, and pharmaceutical trials, with the lowest hanging fruit—observational research necessary for effective clinical practice—continuing to be neglected.

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Competing interests: None declared.

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Brains and mobile phones

The biggest risk to health from mobile phones is using them while driving

here are more than 50 million mobile phones in the United Kingdom, and more than 1 billion worldwide. Mobile phones allow people to communicate with flexibility and ease. In addition, having a personal and mobile means of communication has helped to save lives through quicker notification of accidents, trauma, and other dangers. But concerns about the safety of mobile phones have been raised.

In 2000 the UK Independent Expert Group on Mobile Phones (IEGMP) published the Stewart report. The report recommended a programme of research and a precautionary approach to the use of mobile phones, especially use by children. As a result of the recommendations a research programme was launched in 2001 with a budget of £7.36m (€10.5m; \$13m), jointly funded by government and industry. Two papers in this week's BMJ come out of this initiative. Two

Hepworth and colleagues (p 883) conducted a population based case-control study of 966 patients with gliomas and found that use of mobile phones, in the short and medium term, is not associated with increased risk of developing a glioma.³ The response rate of only 51% in this study, predominantly from patients with low grade tumours, may contribute to missing a real but small effect. The study illustrates the difficulty of estimating use of mobile phones over many years and with different technology (analogue

and digital), and thus the uncertainty in estimating exposure to radiofrequency radiation.

As there is no obvious biological mechanism for cancer to be caused by radiofrequency radiation, there is probably no relation between mobile phone use and development of gliomas. But the latency period for formation of gliomas could be longer than the period studied by Hepworth and colleagues, and longer surveillance will be necessary to reach more reliable conclusions. Greenfield's neuropathology textbook states: "Such an association [between radiofrequency radiation from mobile phones and malignant gliomas] would be surprising given the short time since the introduction of the widespread use of mobile phones: in adult humans, all known environmental carcinogens, including radiation, require a latency period of usually more than 20 and often more than 30 years."5 In Hepworth and colleagues' study only a small number of participants with glioma reported exposure of more than 10 years.

Some evidence indicates, however, that acoustic neuromas and salivary tumours may be related to use of mobile phones. Hepworth and colleagues' paper derives from an international collaborative study on use of mobile phones and risks of intracranial tumours, and perhaps these associations will also be studied.

Also in this week's *BMJ* (p 886) Rubin and colleagues examine the phenomenon of "electromagnetic hypersensitivity." This is a collection of

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symptoms such as headache, nausea, fatigue, dizziness, and loss of memory or concentration apparently precipitated by exposure to electromagnetic radiation. In Sweden it is accepted as a physical impairment, and a national scheme exists to improve home and work conditions for sufferers.

Rubin and colleagues conducted a double blind randomised within participants provocation study in a group of people who reported sensitivity to electromagnetic fields. The study failed to show that symptoms were associated with exposure to mobile phone radiation. People in the sensitive group had more severe symptoms (compared to controls), but their symptoms of electromagnetic hypersensitivity occurred with the same frequency when the mobile phone was switched on and during sham exposure. The authors describe this as a nocebo phenomenon, and suggest the role of psychological factors.

The IEGMP accepted that mobile phone radiation may produce biological effects, but it did not think that such radiation caused adverse health effects. In 2005 the National Radiological Protection Board updated the Stewart report and proposed that this conclusion still holds true. Hepworth's paper gives some further reassurance but, as the Global System for Mobile Communications (GSM) is now barely 10 years old, the question remains whether this technology has been in use for a sufficient period to allow recognition of an effect of exposure on the development of brain pathology. Rubin's study shows that some people develop symptoms to expected exposures even in the absence of such exposure. This finding does not necessarily preclude a real effect.

The evidence to date suggests that any risk to the individual mobile phone user of developing brain pathology is fleetingly small. The Health Council of the Netherlands even states that there is no reason to recommend that mobile phone use by children should be limited, and no need to apply the precautionary principle.⁷

The most important established risk of mobile phones to people is their use while driving. This is true for hand held phones as well as for hands free ones. Since 2003 it has been illegal in the United Kingdom to drive a car while using a hand held phone, but still legal to use a hands free one. It is time to correct this discrepancy.

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Competing interests: None declared.

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Strict glucose control in the critically ill

May not be such a good thing for all critically ill patients

In 2001 Van den Berghe et al reported the results of a randomised controlled trial comparing the mortality of critically ill surgical patients receiving insulin infusions to achieve "tight glycaemic control" (target blood glucose 4.4-6.1 mmol/l) with that of patients receiving conventional treatment, where insulin was infused only if the blood glucose exceeded 11.9 mmol/l and was adjusted to maintain values of 10-11.1 mmol/l.¹ The trial was stopped after 1548 patients had been enrolled because the mortality in the tight control group was 4.6% compared with 8% in the control group (32% corrected relative reduction; P=0.04). Ever since, tight glycaemic control has been standard practice, but there are now good reasons to question it.

It always seemed surprising that a simple change in blood glucose management reduced mortality more than other far more costly and complex interventions tested through randomised trials in the critically ill. The only corroborating evidence came from studies of glucose-insulin-potassium treatment in acute myocardial infarction outside a critical care setting² and an observational study of tight glycaemic control in a

general intensive care setting.³ The 2001 study was conducted on a relatively restricted population consisting mainly of post-surgical patients (63% after cardiac surgery) with low admission APACHE II scores and used an unusual feeding regimen. In spite of these limitations, tight glycaemic control rapidly became standard practice in critically ill medical as well as surgical patients in Britain⁴ and an internationally recommended standard of care in all patients with severe sepsis.⁵

However, confidence in the benefits of strict glucose control for all critically ill patients is being eroded. Last year the German SepNet group suspended a multicentre randomised controlled trial in medical and surgical patients with severe sepsis. Tight glycaemic control produced no reduction in mortality, but it did cause a higher incidence of hypoglycaemia (12.1% v 2.1%). Also last year the CREATE-ECLA trial, a study of insulin-glucosepotassium therapy in 20 000 patients with acute myocardial infarction, showed no benefits, removing some of the indirect support for tight glycaemic control. Finally, early this year Van den Berghe and colleagues reported another study of tight glycaemic

BMJ 2006;332:865-6