

PostScript

LETTERS

If you have a burning desire to respond to a paper published in the *JECH*, why not make use of our "rapid response" option?

Log on to our website (www.jech.com), find the paper that interests you, and send your response via email by clicking on the "eletters" option in the box at the top right hand corner.

Providing it isn't libellous or obscene, it will be posted within seven days. You can retrieve it by clicking on "read eletters" on our homepage.

The editors will decide as before whether to also publish it in a future paper issue.

Acute otitis media after forceps delivery

While running an ambulatory paediatric clinic, the mother of a crying baby wondered whether forceps delivered babies were more prone to otitis than other babies. To our knowledge, this association has not been reported. But, on the other hand, facial nerve injury is more common among those delivered by forceps¹; it is caused by compression of diploic bone of the mastoid process where the facial nerve is located superficially.² It is clear that this kind of extraction applies some pressure over the ear of the baby. The head vulnerability is well known: long term consequences have been recognised after mild head injury.³

To estimate the differences of proportion of acute otitis media (AOM) between children delivered by forceps and other babies we conducted an electronic medical records review of all singleton children attending a primary care paediatric clinic, born full term from 1 January 1996 to 31 December 2004. Table 1 shows the characteristics of the children. Sex was comparable, birth weight of non-operative vaginal delivery babies (NO) was lower, vacuum assisted babies (VA) were older at AOM diagnosis, and rates of neonatal admission were not comparable.

AOM was diagnosed by a history of acute onset of signs and symptoms and otoscopic examination of the eardrum.⁴

Of 1449 deliveries, there were: 754 NO, 217 F, 52 VA, and 426 CS (caesarean sections). AOM had been recorded in 234 (31.0%) of children born by NO, 87 (40.0%) of F, 15 (28.8%) of VA, and 124 (29.1%) of CS.

The analysis showed that forceps delivered babies were associated with an increase in proportion of AOM (odds ratio (OR) 1.48; 95% confidence intervals (CI) 1.08 to 2.03; $p = 0.015$) compared with NO.

There were no differences in AOM proportions between VA and NO (OR 0.85; 95% CI 0.46 to 1.58; $p > 0.3$). And there were no differences in AOM proportions between CS and NO (OR 0.91; 95% CI 0.70 to 1.18; $p > 0.3$).

Our finding could be confirmed or discarded by ongoing longitudinal studies. If it is confirmed, it will strengthen the resolve to pay careful attention to the comments of our patients.

It is already known that VA is at least as safe as forceps for the mother and the neonate. Long term consequences of operative vaginal delivery need to be explored: a prospective study should be undertaken to find if this association really exists.

Correspondence to: Dr S Verd, Paediatric Clinic, AV Alejandro Rosselo, 10, 07002 Palma de Mallorca, Spain; drsverd@terra.es

doi: 10.1136/jech.2005.034702

References

- Demissie K, Rhoads GG, Smulian JC, et al. Operative vaginal delivery and neonatal and infant adverse outcomes: population based retrospective analysis. *BMJ* 2004;**329**:24-9.
- Bonni A, Ross MG. Forceps delivery. *eMedicine*. <http://www.emedicine.com/med/topic3284.htm>.
- Anderson V, Catroppa C, Morse S, et al. Outcome from mild head injury in young children: a prospective study. *J Clin Exp Neuropsychol* 2001;**23**:705-17.
- American Academy of Pediatrics, Subcommittee on Management of Acute Otitis Media. Diagnosis and management of acute otitis media. *Pediatrics* 2004;**113**:1451-65.

Education and mortality: a role for intelligence?

In their report van Oort and others¹ clearly describe and empirically examine the potential mediating factors—broadly categorised by the authors as material, psychosocial, and behavioural—that might account for the well established inverse education-mortality gradient. We believe the role of intelligence (denoted here as IQ, and defined as a person's ability to learn, reason, and solve problems²) warrants mention, given its link with all cause mortality, other somatic health outcomes, and at least two (material and behavioural factors) of the aforementioned pathways.³

Recently reported findings from a series of cohort studies show an inverse association between IQ, assessed using psychometric tests, and later death, whether this "exposure" was quantified in childhood, early adulthood, middle age, or older age.³ Although fewer data are available for cause specific outcomes, similar gradients have also been reported for childhood assessed IQ in relation to adult risk of ischaemic heart disease (but not stroke), selected cancers, and accidents.³ IQ has also been linked with behavioural factors, including smoking patterns, such that adults with higher early life IQ scores are more likely to subsequently give up the habit than their lower performing counterparts.⁴ While these findings are comparatively recent, the suggestion that early life IQ might influence later life material measures of socioeconomic position—particularly income,² but also car and house ownership—has a long research tradition.

Based on these findings and using the authors' own conceptual model (figure 1; page 215¹), IQ may be regarded in at least three ways. Firstly, education may be a proxy for IQ. However, this is not to ignore potential interplay between IQ and education, such as mediation or moderation (effect modification), and the influence this might have on health. Secondly, in a related point, IQ might generate individual differences in educational attainment, in addition to being independently associated with material and behavioural factors. In studies that adjusted for education in the IQ-mortality relation, results are inconsistent with some investigators finding pronounced attenuation, while others do not.³ Thirdly, given that education may represent a cognitive archaeological "record" of pre-adult insults (for example, illness, nutritional privation, poor living conditions, psychosocial stress), it is probable that IQ, given its metric properties (education is normally quantified categorically), is a more sensitive marker of such exposures.⁵

In summary, there is new and persuasive evidence to link early cognitive ability and education with later health outcomes. Understanding the mechanisms that may underlie these associations should include an examination of whether education may be a partial mediator of, or a surrogate for, IQ differences.

Acknowledgements

We thank Marcus Richards for providing helpful comments on an earlier version of this correspondence.

Table 1 Patient characteristics

Feature	Non-operative delivery (n=754)	Forceps delivery (n=217)	Vacuum delivery (n=52)	Caesarean section (n=426)
Birth weight mean (SD)	3315.2 (400.7)*	3429.3 (388.2)	3496.2 (416.7)	3383.4 (467.8)
Ratio male:female	1.08	1.18	1.42	1.34
Newborns admitted to special care (%)	24 (3.1)*	11 (5.0)*	8 (15.3)*	25 (5.8)*
Age, months, at AOM diagnosis mean (SD)	18.2 (12.9)	17.4 (10.8)	23.7 (12.6)*	18.9 (13.0)

* $p < 0.05$.

G David Batty

MRC Social and Public Health Sciences Unit,
University of Glasgow, Glasgow, UK

G David Batty, Ian J Deary

Department of Psychology, University of Edinburgh,
Edinburgh, UK

Correspondence to: Dr G D Batty, MRC Social and
Public Health Sciences Unit, University of Glasgow,
4 Lilybank Gardens, Glasgow G12 8RZ, UK;
david-b@msoc.mrc.gla.ac.uk

References

- 1 **van Oort FV**, Van Lenthe FJ, Mackenbach JP. Material, psychosocial, and behavioural factors in the explanation of educational inequalities in mortality in the Netherlands. *J Epidemiol Community Health* 2005;**59**:214–20.
- 2 **Neisser U**, Boodoo G, Bouchard Jnr T, *et al*. Intelligence: knowns and unknowns. *Am Psychol* 1996;**51**:77–101.
- 3 **Batty GD**, Deary IJ. Early life intelligence and adult health. *BMJ* 2004;**329**:585–6.
- 4 **Taylor MD**, Hart CL, Davey Smith G, *et al*. Childhood mental ability and smoking cessation in adulthood: prospective observational study linking the Scottish Mental Survey 1932 and the midspan studies. *J Epidemiol Community Health* 2003;**57**:464–5.
- 5 **Whalley LJ**, Deary IJ. Longitudinal cohort study of childhood IQ and survival up to age 76. *BMJ* 2001;**322**:819.

Authors' response

We appreciate the comments of Batty and Deary on our paper reporting on the explanations of educational inequalities in mortality. We agree with them that there is evidence linking early cognitive ability to later health outcomes and that inclusion of education and early life intelligence may be important for studies of health inequalities. However, the latter seems to be dependent on the purpose of the study. It was the aim of our study to contribute to the debate on the importance of different mediating factors in the causal pathway of educational inequalities in health, and to provide more guidance to policy recommendations to reduce these inequalities, and it is less clear if inclusion of early life intelligence would have contributed much to these aims.

We believe that the most appropriate position for early life intelligence in our conceptual model would be preceding educational achievement. In this position early life intelligence could contribute to the understanding of differences in educational achievement. In contrast, its contribution to the understanding of causal pathways from education to health would be limited.

Inclusion of intelligence in a study like ours would increase understanding of the “core” roots of inequalities in health and should therefore be supported. Using the same arguments as the authors did however, this can be also be said from other factors, such as birth weight, parental socioeconomic position, and neighbourhood deprivation.^{1–3} These factors are also related to educational achievement, and do have mediating and interacting effects.

Finally, the connection of early life intelligence to policy recommendations is less straightforward than this is for education: recommending improvement of an individual trait as early life intelligence may be more difficult than recommending improvement of educational achievement, for example by

policies that maximise the chance that children will remain at school.⁴

Floor V A van Oort, Frank J van Lenthe, Johan P Mackenbach

Department of Public Health, Erasmus MC, University
Medical Centre, Rotterdam, Netherlands

Correspondence to: Dr F van Oort, Department of
Public Health, Erasmus MC, University Medical Centre
Rotterdam, PO BOX 1738, 3000 DR Rotterdam,
Netherlands; f.vanoort@erasmusmc.nl

References

- 1 **van Lenthe FJ**, Borrell LN, Costa G, *et al*. Neighbourhood unemployment and all cause mortality: a comparison of six countries. *J Epidemiol Community Health* 2005;**59**:231–7.
- 2 **Mheen van de H**, Stronks K, Bos van den J, *et al*. The contribution of childhood environment to the explanation of socio-economic inequalities in adult life: a retrospective study. *Soc Sci Med* 1997;**44**:13–24.
- 3 **Osler M**, Andersen AM, Due P, *et al*. Socioeconomic position in early life, birth weight, childhood cognitive function, and adult mortality. A longitudinal study of Danish men born in 1953. *J Epidemiol Community Health* 2003;**57**:681–6.
- 4 **Mackenbach JP**. Genetics and health inequalities: hypotheses and controversies. *J Epidemiol Community Health* 2005;**59**:268–73.

Offspring sex ratios of people exposed to electromagnetic fields

Saadat¹ wrote that there has only been one study on the association between human offspring sex ratio (proportion male) at birth and parental exposure to electromagnetic fields (EMF). He substantiated this claim with a reference to Irgens *et al*.² However, I³ cited six other such studies.^{4–9} These studies, although not unanimous, cumulatively suggest that both exposed men and exposed women tend to produce significant excesses of daughters. This suggestion is not much changed by the comparatively small samples adduced by Saadat.¹ The point may be illustrated by considerations of standard power analysis.¹⁰ Suppose that you wished to test that exposure has the effect of reducing the offspring sex ratio by, say, 10% (viz from an expected value of 0.515 to 0.465). Then, to stand 8 chances in 10 of detecting a difference at the 0.05 level (one way), you would require equal sized samples (of offspring of exposed and non-exposed subjects) each numbering 1236. The number of offspring of Saadat's¹ sample of exposed subjects was 110.

In my letter I cited³ evidence that exposure of men to EMF is associated with fatigue, headaches, dizziness, impaired memory, nausea, loss of strength in limbs, respiratory difficulties, sleep disturbances, and reduced libido. Such reports may be dismissed as subjective. However I also cited reports that exposure to EMF was associated with reduced sperm counts in men and rats: and of reduced testosterone levels in rats. And more recently, men's exposure to radiofrequency radiation has reportedly been linked to a lowered testosterone/gonadotrophin ratio.¹¹ And there are good grounds for suspecting this hormone profile to be causally associated with low offspring sex ratios.^{12–13} More data are urgently needed on the sexes of offspring of men and women who have been exposed to EMF.

Correspondence to: Dr William Henry James,
University College London, 4 Stephenson Way,
London NW1 2HE, UK; whjames@waitrose.com

References

- 1 **Saadat M**. Offspring sex ratio in men exposed to electromagnetic fields. *J Epidemiol Community Health* 2005;**59**:339.
- 2 **Irgens A**, Kruger K, Skorve AH, *et al*. Male proportion in offspring of parents exposed to strong static and extremely low frequency electromagnetic fields in Norway. *Am J Ind Med* 1997;**32**:557–61.
- 3 **James WH**. The sex ratios of offspring of people exposed to non-ionising radiation. *Occup Env Med* 1997;**54**:622–3.
- 4 **Nordstrom S**, Birke E, Gustavsson L. Reproductive hazards among workers at high voltage substations. *Bioelectromagnetics* 1983;**4**:91–101.
- 5 **Knave B**, Gamberale F, Birke E, *et al*. Long term exposure to electric fields: a cross-sectional epidemiologic investigation of occupationally exposed workers in high voltage substations. *Scand J Work Environ Health* 1979;**5**:115–25.
- 6 **Mubarak AAS**, Mubarak AAS. Does high voltage electricity have an effect on the sex distribution of offspring? *Hum Reprod* 1996;**11**:230–1.
- 7 **Larsen AI**, Olsen J, Svane O. Gender specific reproductive outcomes and exposure to high frequency electromagnetic radiation among physiotherapists. *Scand J Work Environ Health* 1991;**17**:324–9.
- 8 **Guberan E**, Campana A, Faval P, *et al*. Gender ratio of offspring and exposure to shortwave radiation among female physiotherapists. *Scand J Work Environ Health* 1994;**20**:345–8.
- 9 **Kolodynski AA**, Kolodynska VV. Motor and psychological functions of school children living in the area of the Skrunda radiolocation station in Latvia. *Sci Total Environ* 1996;**180**:87–93.
- 10 **Snedecor GW**, Cochran WG. *Statistical methods*. 6th ed. Ames, IA: Iowa State University Press, 1967:222.
- 11 **Grajewski B**, Cox C, Schrader SM, *et al*. Semen quality and hormone levels among radiofrequency heater operators. *J Occup Environ Med* 2000;**42**:993–1005.
- 12 **James WH**. Evidence that mammalian sex ratios at birth are partially controlled by parental hormone levels at the time of conception. *J Theor Biol* 1996;**180**:271–86.
- 13 **James WH**. Further evidence that mammalian sex ratios at birth are partially controlled by parental hormone levels around the time of conception. *Hum Reprod* 2004;**19**:1250–6.

Author's reply

I thank Dr James for his letter. He may well be correct for the citations of the related studies and also the estimation of the sample size. However, I wish to make some comments.

Not only in my report,¹ but also in other reports^{2–4} the sample size was much lower than that estimated by James. Among published data, the article of Guberan *et al*⁸ was based on 1781 births (508 and 1273 births from exposed and unexposed pregnancies, respectively), which is a comparatively large sample size and it is near to the required sample size, calculated by James. Guberan *et al* found that there was no statistically significant difference between exposed and unexposed pregnancies for offspring sex ratio.⁵ Irgens *et al* reported that offspring sex ratio of women in industries with electromagnetic fields was significantly reduced; while in men exposed to the fields the ratio did not show significant difference.⁶ On the other hand, experimental design studies showed that when rodents (mice and rats) were exposed to electromagnetic fields, the offspring sex ratio significantly increased⁷ or remained unchanged,^{8,9} compared with their

controls. Taken together it seems that the published data are not sufficient to conclude that the offspring sex ratio tends to produce significant excesses of daughters when parents are exposed to electromagnetic fields.

Correspondence to: Dr Mostafa Saadat, Department of Biology, College of Sciences, Shiraz University, Shiraz 71454, Iran; saadat@susc.ac.ir

References

- 1 Saadat M. Offspring sex ratio in men exposed to electromagnetic fields. *J Epidemiol Community Health* 2005;**59**:339.
- 2 Nordstrom S, Birke E, Gustavsson L. Reproductive hazards among workers at high voltage substations. *Bioelectromagnetics* 1983;**4**:91–101.
- 3 Knave B, Gamberale F, Bergstrom S, et al. Long-term exposure to electric fields. A cross-sectional epidemiologic investigation of occupationally exposed workers in high voltage substations. *Scand J Work Environ Health* 1979;**5**:115–25.
- 4 Larsen AI, Olsen J, Svane O. Gender-specific reproductive outcomes and exposure to high-frequency electromagnetic radiation among physiotherapists. *Scand J Work Environ Health* 1991;**17**:324–9.
- 5 Guberan E, Campana A, Favali P, et al. Gender ratio of offspring and exposure to shortwave radiation among female physiotherapists. *Scand J Work Environ Health* 1994;**20**:345–8.
- 6 Irgens A, Kruger K, Skorve AH, et al. Male proportion in offspring of parents exposed to strong static and extremely low-frequency electromagnetic fields in Norway. *Am J Ind Med* 1997;**32**:557–61.
- 7 Wang SM, Wang DW, Peng RY, et al. Effect of electromagnetic pulse irradiation on mice reproduction. *Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi* 2003;**21**:329–31.
- 8 Ohnishi Y, Mizuno F, Sato T, et al. Effects of power frequency alternating magnetic fields on reproduction and pre-natal development of mice. *J Toxicol Sci* 2002;**27**:131–8.
- 9 Ryan BM, Symanski RR, Pomeranz LE, et al. Multigeneration reproductive toxicity assessment of 60-Hz magnetic fields using a continuous breeding protocol in rats. *Teratology* 1999;**59**:156–62.

BOOK REVIEWS

Public health policy

David J Hunter. Cambridge: Polity Press, £50, pp 224. ISBN 0-74-562646-7

“Advancing the public’s health has always been the stated goal of healthy policy, even if in practice a systematic bias in favour of responding to ill health and tackling disease has remained all pervasive and remarkably impervious to change”.

While this quote doesn’t appear until halfway through *Public Health Policy*, it is from this perspective that Hunter approaches his analysis. The text details the history of the public health sector in the United Kingdom as well as the problems and issues that have faced and will continue to face the policy makers who enjoy responsibility for the public’s health, from the House of Commons to the NHS. While historically confining his analysis to the UK, the well organised history of the struggles faced in the UK parallel those faced by developed and developing countries. The pitfalls of the competing priorities of the “downstream” focus on current health care offerings (treatment) compared with the “upstream” positive effects of shifting focus to a public health perspective (prevention) are analysed on many levels and from varying viewpoints. Hunter offers potential solutions to some of the difficulties that face decision makers struggling to formulate health policy that reflects a shift in priorities. The book is well written and offers insights useful for all who have an interest in improving the public’s health, whether in the UK or elsewhere.

S Goldie

Ethics for health care, 2nd edition

Edited by C Barglund. Melbourne: Oxford University Press, 2004, pp 330. ISBN 0-19-5517-30-X

Oxford University Press has recently published a second edition of this popular and widely read primer on medical ethics. Designed as a teaching tool for a broad range of health care professionals, the text draws upon a series of contemporary problems as the author illustrates the key features of ethical thinking.

The chapters are designed in a sequential rather than encyclopaedic fashion, developing themes through a series of over 50 suggested tasks for the reader to engage in before moving further. Almost half of these individual and group exercises incorporating tutorial/problem based triggers are new to this edition, reflecting the author’s emphasis on helping the reader understand the material by placing it within their own context. Subsequent text then explores the issues, guiding the reader on the process of analysing everyday health care issues in an ethical framework, while focusing on the health care worker to client interaction.

Although many examples considered in the text are drawn from contemporary Australian sources, they are presented in an inclusive fashion for an international readership. In particular, the author considers the cultural, religious, and sociological contexts that inform ethical considerations, rather than limiting herself to any one theoretical framework.

In summary, this book provides an excellent programme of instruction for health care workers in applying ethical reasoning to the problems they encounter in everyday practice, leaving the reader with workable tools they can use on a daily basis.

S Margolis