

possibility exists that in the 1970s women died of Creutzfeldt-Jakob disease but the cause of death was incorrectly attributed. Evidence that a transmissible agent caused Creutzfeldt-Jakob disease was first published in *Science* in 1968.⁵ It took several years, however, for to make the connection between Creutzfeldt-Jakob disease and growth hormone. No doubt the same may apply to gonadotrophin derived from human pituitaries.

NAOMI PFEFFER

London N16 0BB

- 1 Dyer C. Growth hormone victims seek compensation. *BMJ* 1993;306:607. (6 March.)
- 2 Pituitary hormones for infertility [editorial]. *BMJ* 1965;ii:316.
- 3 Crooke AC, Butt WR, Palmer RF, Morris R, Logan Edwards R, Anson CJ. Clinical trial of human gonadotrophins. *Journal of Obstetrics and Gynaecology of the British Commonwealth* 1963; 70:604-35.
- 4 Cochius JJ, Hyman N, Esiri MM. Creutzfeldt-Jakob disease in a recipient of human pituitary-derived gonadotrophin: a second case. *J Neurol Neurosurg Psychiatry* 1992;55:1095.
- 5 Gibbs CJ, Gajdusek DC, Asher DM, Alpers MP, Beck E, Daniel PM, et al. Creutzfeldt-Jakob disease (spongiform encephalopathy): transmission to the chimpanzee. *Science* 1968;161: 388-9.

Parental irradiation and excess childhood leukaemia

EDITOR,—Eve Roman and colleagues' study investigating the relation between parental employment in the nuclear industry and childhood leukaemia and non-Hodgkin's lymphoma¹ was first proposed by the Committee on Medical Aspects of Radiation in the Environment in 1989.² After the publication of the report by Gardner *et al* in 1990³ the committee recommended that the study should be completed as soon as possible.⁴ As one responsible for providing occupational health advice to radiation workers, I am surprised that the paper was not accompanied by a formal statement from the committee. When work is recommended by a national advisory group, in a subject known to be of enormous and instant interest to the media, advice to workers is greatly facilitated if expert assessment is available. Any delay can contribute to anxiety. In any work on transgenerational issues the provision of information to workers and their families is of the highest priority.

Roman and colleagues state that their study "was set up to investigate whether the excess [cases of childhood leukaemia in West Berkshire and North Hampshire] was related to parents' employment in the nuclear industry." On this basis, I wonder why its most fundamental conclusion is not stated as early as possible as "it has been shown that the cluster in Hampshire and Berkshire does not result from external radiation exposure of workers at the atomic weapons establishments." As the fathers of under 8% of the cases were employed at the sites it seems hard to imagine that any other conclusion can be reached. This important finding, however, is only alluded to in the final sentence of the abstract's conclusion, without any real emphasis. It is not raised overtly in "editor's choice" in the issue of 6 March, nor can I find reference to it in any coverage in the media. On this occasion, however, I find it hard to blame the press for the omission.

The more detailed statistics on the few cases with fathers employed in the industry are of interest but of limited value in relation to the nationally coordinated study of transgenerational risk which has been under way for some time, only part of which is mentioned in the paper. Gardner's name has become synonymous, among both the public and workers, with a postulated link between childhood leukaemia and paternal irradiation of 100 mSv in total and 10 mSv in the six months before conception. With the data presented indicating maximum doses of less than 5 mSv in total and zero in the six months before conception there seems to be nothing in the work to support such a

hypothesis. The statement that "our results can be interpreted as supporting Gardner and colleagues' finding" is, I suppose, based on statistical markers and associations rather than causal hypotheses but is open to misinterpretation. In examining their findings, the authors go on to speculate that the effect could be due to internal contamination by radioactive substances or some other exposure. Their text indicates that only one father of a case was monitored for contamination, and therefore no data are provided to support such a conclusion.

CHRIS KALMAN

Scottish Nuclear,
East Kilbride G74 5PR

- 1 Roman E, Watson A, Beral V, Buckle S, Bull D, Barker K, et al. Case-control study of leukaemia and non-Hodgkin's lymphoma among children aged 0-4 years living in West Berkshire and North Hampshire health districts. *BMJ* 1993; 306:615-21. (6 March.)
- 2 Committee on Medical Aspects of Radiation in the Environment. *Third report. Report on the incidence of childhood cancer in the west Berkshire and north Hampshire area, in which are situated the Atomic Weapons Research Establishment, Aldermaston and the Royal Ordnance Factory, Burghfield.* London: HMSO, 1989.
- 3 Gardner MJ, Snee MP, Hall AJ, Powell CA, Downes S, Terrell JD. Results of case-control study of leukaemia and lymphoma among young people near Sellafield nuclear plant in West Cumbria. *BMJ* 1990;300:423-9.
- 4 House of Commons official report (Hansard). 1990 April 2;170: col 433. (No 84.)

Kidney donation after paracetamol overdose

EDITOR,—We wish to correct the impression given in S Jackson and colleagues' letter that hearts and corneas are the only organs salvageable from patients who die after paracetamol overdose.¹ Over the past two years we have transplanted five kidneys from such donors. All recipients received routine immunosuppression with prednisolone, azathioprine, cyclosporin, and antilymphocyte globulin. The results in these five unselected recipients have been at least as good as those in our "normal" transplant population, and we have encountered no special difficulties in management or follow up. The table gives details of the donors and outcomes.

It has been estimated that 160-200 patients die in the United Kingdom each year from paracetamol overdose, of whom perhaps half might be suitable donors of kidneys. Accurate figures for paracetamol overdose are not available from the United Kingdom Transplant Service, but only 19 kidneys were donated from suicide victims in 1992 (personal communication). Thus a large pool of potential donor organs is not being used.

One explanation for this is the mistaken belief that patients presenting with acute renal failure after overdose are not suitable for organ donation. The renal changes associated with paracetamol overdose are, however, fully reversible, and the kidneys can be safely transplanted in the absence of other confounding factors. This point is well illustrated by case 1 (table): the patient was oligoanuric for at least three hours before organ retrieval, had a plasma creatinine concentration of 213 $\mu\text{mol/l}$, and might not have been offered

for transplantation in many units; outcome was excellent.

If there is any doubt regarding current or previous renal function a valuable guide to tissue viability is the findings on microscopic examination of a frozen section of renal cortex taken at or before donor nephrectomy. We have twice used this technique, both times with excellent results.

We recommend that all patients dying of paracetamol overdose should be regarded as potential kidney donors. Without prejudice to their survival the patients should be managed in intensive therapy units so that optimum renal function is preserved. Evidence should be sought of previous renal status and the transplant coordinator informed at an early stage. Only in this way will we see an increase in organ donation from this hitherto underused source.

P A ANDREWS

C G KOFFMANN

Departments of Nephrology and Transplantation,
Guy's Hospital,
London SE1 9RT

- 1 Jackson S, Nightingale P, Shelly MP. Organ donation after paracetamol overdose. *BMJ* 1993;306:718. (13 March.)

Screening for prostatic cancer

EDITOR,—Fritz H Schröder discusses the many problems related to screening for prostatic cancer.¹ He emphasises that both the incidence of and mortality from prostatic cancer are increasing and that early detection might help to reduce the mortality from cancer. We agree when he argues that the conditions to do this for prostatic cancer have not yet been met. This is mainly because of the poor validity of the three screening tests (digital rectal examination, transrectal ultrasonography, and prostate specific antigen concentration). In addition, as Schröder mentions, until now separating latent from aggressive prostatic cancers has not been possible, which carries a considerable risk of overtreatment.

We disagree that prospective studies should be carried out urgently because screening for prostatic cancer has already become widespread. Screening that was initiated as part of research has now become routine, with a decreasing emphasis on thorough evaluation. What is needed is a rigorous analysis of existing screening programmes: by analysing data from these programmes, combined with population data on prevalence and prognosis, it should be possible to answer four important questions.

Firstly, is it possible to separate latent from progressive prostatic cancer when the tumour is detected at an early stage?

Secondly, is the prognosis of progressive tumours influenced by early treatment?

Thirdly, is a suitable (combination of) screening test(s) available?

Fourthly, does screening for prostatic cancer reduce mortality from the disease?

Despite the current economic recession we fear that both the public and policymakers will continue

Details of five patients who donated kidneys after death from paracetamol overdose and of recipients

	Case 1	Case 2	Case 3	Case 4	Case 5
Donor:					
Age (years)	21	17	28	47	47
Sex	F	F	F	F	F
Recipient:					
Age (years)	35	17	25	49	51
Cause of renal failure	Spina bifida	Reflux	Vasculitis	Small kidneys	Obstruction
Haplotype match	2	0	1	0	0
Time to independence from dialysis (days)	7	0	0	0	20
Rejection episodes	0	1	1	0	3
Follow up (months)	2	1	12	10	10
Current creatinine ($\mu\text{mol/l}$)	65	84	93	83	134