16.7% of a cohort of 253 patients followed up over 10 years.²

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- 1 Schröder FH. Microscopic haematuria. *BMJ* 1994;309:70-2. (9 July.)
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Cystoscopy has little value in children

EDITOR,—Fritz H Schröder does not emphasise that his editorial on microscopic haematuria relates to adult and not paediatric practice. There is little point in children with haematuria undergoing cystoscopy, yet each year I see several children who have been investigated in urology departments and, after cystoscopy has given a negative result, are referred for a paediatric medical assessment. It would have been helpful if the editorial had emphasised this.

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Is rarely significant if asymptomatic

EDITOR,—Fritz H Schröder's editorial on microscopic haematuria seems unduly alarmist.¹ He quotes a prevalence of 12% of life threatening conditions in men and 34% in women over 50 with haematuria. However, the prevalence of serious disease depends on how the haematuria comes to light. In a paper from the Mayo Clinic Mohr *et al* found "serious" urological disease (including calculi) in 2.0% of patients with asymptomatic haematuria compared with 1.2% of controls.² The prevalence of urothelial tumours and renal carcinomas was 0.5% in the asymptomatic haematuria group (zero in the controls). These were men over 34 and women over 54 living in Rochester, Minnesota, undergoing routine urine screening.

The much higher figures quoted by Schröder must include patients who have urinary symptoms or signs. In the absence of such symptoms or signs it is arguable whether asymptomatic haematuria with less than nine red blood cells per high power field justifies investigation. In a paper from Chicago the estimated cost of diagnosing one case of renal or urothelial carcinoma in patients with asymptomatic haematuria was \$75 000-\$86 000.³

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Dipsticks may pick up haemoglobinuria

EDITOR,—Fritz H Schröder provides a logical set of practical guidelines for the investigation of microscopic haematuria.¹ Because his algorithm starts from a dipstick analysis, it is important to remember that a positive dipstick result is not synonymous with haematuria. The commonest explanation for a positive result with no red cells in

the urinary sediment is haemoglobinuria. This is much more rare than haematuria, but recognising haemoglobinuria is important because it signals, rather than renal disease, intravascular haemolysis. This in turn is the final common pathway of red cell destruction in different conditions.² Haemoglobinuria can be acute and massive, and therefore unlikely to be confused with haematuria, in haemolytic anaemias associated with glucose 6-phosphate dehydrogenase deficiency, and in rare cases of malaria ("blackwater fever") and septicaemia (caused by *Clostridium welchii*); but it can be more insidious in certain forms of autoimmune haemolytic anaemia and in paroxysmal nocturnal haemoglobinuria.

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- 1 Schröder FH. Microscopic haematuria. *BMJ* 1994;309:70-2. (9 July.)
- 2 Luzzatto L. Glucose 6-phosphate dehydrogenase deficiency and hemolytic anemia. In: Nathan DG, Oski FA, eds. *Hematology of infancy and childhood*. Philadelphia: W B Saunders, 1993: 674-91.

Prevalence in young men underestimated

EDITOR,—Fritz H Schröder's fine editorial and algorithm¹ is no less welcome if I cavil at his underestimate of the problem in those aged under 40 years. In the *BMJ* 10 years ago, Froom *et al* showed that about a third of fit young men in the Israeli air force had microscopic haematuria when examined over 15 years²: the sort of cumulative incidence that is not disclosed by prevalence studies.

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Paternal treatment for sterility and neonatal neuroblastoma

EDITOR,—Robaire and Hales have discussed the possible effects of paternal exposure to chemicals before conception.¹ We report a case of neuroblastoma diagnosed as stage IVS at birth (with manifestations in the skin and liver accompanied by lumboaortic adenopathy) in a male infant born in France whose father had been treated for sterility.

Metaiodobenzylguanidine scans yielded negative results, and N-myc oncogene was not amplified. The first two courses of chemotherapy with vincristine and cyclophosphamide according to the NB90 protocol of the French Society for Paediatric Oncology led to a good partial remission. This was followed by recurrence in the brain and massive pulmonary metastases, which did not respond to treatment with etoposide and carboplatin. The child died of respiratory failure at 3 months. Necropsy was not performed.

The father, aged 29, had been diagnosed as having hypogonadotrophic hypogonadism. He had been treated for seven years, until the third month of the pregnancy, with alternating human chorionic gonadotrophin, human menopausal gonadotrophin, androgens, and luteinising hormone releasing hormone. A few months before the pregnancy modest spermatogenesis had been noted. No other paternal or maternal treatments could be identified, and the parents had not been exposed to other recognised toxic compounds.

Recently, neuroectodermal tumours have been reported in children whose mothers had been

treated for infertility—in particular, after the use of drugs to induce ovulation.^{2,3} Other cases of tumours and a case of intestinal fibromatosis of the newborn have also been described after treatment with human gonadotrophins or oral contraceptives or after induction of ovulation. Gamete intrafallopian transfer has been associated with retroperitoneal haemangiolymphangioma in one case, and in vitro fertilisation with congenital malformations.⁴ Treatments for sterility affect germinal cells; therefore their role in the occurrence of genetic modifications that might play a part in the development of tumours cannot be excluded. The role of paternal occupational exposure—whether to hydrocarbons, paints, or radiation—in the aetiology of childhood cancer is still unresolved.

To our knowledge, no publication has addressed the issue of childhood cancer and medical treatment of the father. In our case the infant had a neuroectodermal tumour. As a carcinogenic mechanism has been considered to be plausible with regard to maternal exposure, a similar mechanism linking the treatment of paternal sterility and childhood cancer should not be excluded. Thonneau *et al* estimated that in 39% of infertile couples both partners have reduced fertility.⁵ It would therefore be worth investigating, in the cases already reported, whether the father had also received any fertility treatment.

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Treating hypertension after stroke

EDITOR,—In their editorial on treating hypertension after stroke,¹ Janice E O'Connell and Christopher S Gray refer to the use of sublingual nifedipine, citing an editorial in the *Lancet*,² which in turn refers to the work of Angeli *et al*.³ Sublingual nifedipine is often prescribed in severe hypertension of various aetiologies. The fact that it works, however, has nothing to do with it being sublingual.⁴ O'Connell and Gray allude to this by suggesting nasogastric administration as an alternative route, and, crucially, in Angeli's study the patients were instructed to swallow their saliva after placing a perforated nifedipine capsule under their tongue.⁵ Since therapeutic amounts of nifedipine are absorbed only after the drug is swallowed the capsules should be bitten to release the