Confidentiality and life insurance

SIR,—As a general practitioner I frequently receive letters from life insurance societies stating, "We have received a proposal for insurance on the life of the above person who has given your name as medical attendant and consented to our referring to you regarding his or her medical history." Although most life insurance societies are trustworthy and are unlikely to say such things as that permission has been given when it has not, I feel that these associations and societies should enclose a photocopy of the signature of the patient giving this permission in the same way as the Ministry of Transport do when requesting information about the medical history of a potential driver. Although medical confidentiality is important, certain formalities such as permission from a patient rather than from the patient's agent to divulge information seem to be slipping by the wayside. I always request, before filling in the form, that the insurance society sends me a photocopy of the permission that they have. This should not create great problems as most firms have photocopying facilities.

Since writing the above I have seen the letter from Dr T M Pickard (10 December, p 1544), who echoes many of the feelings I have on this subject. I should be glad to read the views of others who feel strongly about it.

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Oxytocin and neonatal jaundice

SIR,—Dr Louise Friedman and her colleagues (5 November, p 1223) doubt whether there has been an increase in neonatal jaundice. A record of all neonatal abnormalities has been kept in this department since 1967. It includes all infants noted to have jaundice on more than one day in the nursery daily record book as well as those noted in the neonatal record. Hyperbilirubinaemia has been defined as over 250 $\mu\text{mol/l}$ (14.6 mg/100 ml) and figures for over 206 $\mu\text{mol/l}$ (12 mg/100 ml) are complete only for recent years, when more bilirubin estimations have been done. The incidence and severity of neonatal jaundice started to increase in the winter of 1972-73 (see table).

Phototherapy was introduced in 1974 and is given at levels above 250 $\mu\text{mol/l}$ to high-birth-weight infants and 200 $\mu\text{mol/l}$ (11.7 mg/100 ml) (or less) to low-birth-weight infants. This, together with phenobarbitone given to mothers before delivery, probably accounts for the fall in incidence and severity in low-birth-weight infants.

A statistical study of all high-birth-weight infants (except those discharged early) delivered during the past four years is in progress. Preliminary results suggest that

oxytocin is one of several factors which are each associated with a small increase in incidence and severity of neonatal jaundice. There are two observations which suggest that there is some other cause not yet considered. Firstly, about one-third of artificially fed infants delivered normally following spontaneous labour with no maternal drugs develop jaundice. Secondly, the incidence varies periodically, with peaks lasting about one week every few weeks.

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Anti-e and vertical transmission of hepatitis B

SIR,—The presence of hepatitis Be antigen (HBeAg) in the serum of chronic carriers of hepatitis B surface antigen (HBsAg) generally indicates persistence of hepatitis B virus (HBV).¹ Dr A K R Chaudhuri and his colleagues (26 November, p 1416) wisely stress that detectable anti-HBe in a carrier does not necessarily mean non-infectivity in respect of HBV. However, their evidence that an infant with overt type B hepatitis acquired HBV from a carrier mother with anti-HBe is equivocal.

The father was eliminated as the source of HBV by the observation that his serum contained neither HBsAg nor anti-HBs. This very observation, however, raises doubts about the alleged infectivity of the mother. Co-habitation with a chronically infected person is attended by a significant risk of infection, but the father lacked serological evidence of repeated exposure to HBV. Alternatives to the mother as a source of infection should be considered. Higher rates of infection with HBV are observed in the families of persistent carriers than in the rest of the community.¹ The mother of the affected infant was a chronic carrier of HBsAg and there may be clustering of HBV in her family. A dotting maternal relative who was HBeAg positive may have infected the infant.

No mention is made by Dr Chaudhuri and his colleagues of liver function in the mother with anti-HBe who is thought to have transmitted HBV to her infant. The presence of anti-HBe generally suggests normal hepatic function as well as non-infectivity. Demonstration of abnormal liver function in the presence of anti-HBe would have supported the contention of infectivity.

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¹ World Health Organisation, *Advances in Viral Hepatitis*, Technical Report Series No 602. Geneva, WHO, 1977.

Complicated polymyalgia

SIR,—Dr E C Huskisson and his colleagues (3 December, p 1459) rightly draw attention to the importance of excluding serious underlying diseases when making a diagnosis of polymyalgia rheumatica.

We are surprised that the authors consider this association to be "neither well known nor well documented." In our experience the initial presentation of diseases such as rheumatoid arthritis, polymyositis, and myelomatosis as "polymyalgia rheumatica" is relatively common and we have been teaching this for some years.¹ Clearly it is important to seek the appropriate clues: a positive rheumatoid factor test may point to a "myalgic" presentation of rheumatoid arthritis, while a less than dramatic clinical response to corticosteroids is an important clue to myelomatosis.

Polymyalgia rheumatica is, as the authors point out, a distinct clinical entity. But it is not defined as a disease. Until the condition is better understood we believe it is helpful to regard it as a reaction pattern in the elderly which may be the presenting picture in a number of diseases: "cranial" arteritis, rheumatoid arthritis, myelomatosis, and perhaps an "idiopathic" form. This at least encourages one to remain alert to the possibility that corticosteroid therapy may be masking symptoms of an underlying disease requiring different treatment.

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¹ Barnes, C. G. in *An Introduction to Clinical Rheumatology*, ed R M Mason and H L F Currey, 1st ed. London, Pitman, 1970.

Peritoneal dialysis for removal of copper

SIR,—The use of peritoneal dialysis for the treatment of the copper-induced renal failure of Wilson's disease has obvious attractions. The therapeutic trials conducted by Professor Sheila Sherlock and her colleagues (10 September, p 660) have demonstrated that small amounts of copper will diffuse across the peritoneum into dialysis fluid. Penicillamine, given either intravenously, orally, or through the dialysate, had no effect, presumably because of strong intravascular binding to albumin. This complements other work which suggests that intravascular copper is transported as a ternary complex with albumin and free amino-acids.¹

Recently we had a similar opportunity to study the efficacy of peritoneal dialysis when a 2-year-old boy presented with acute copper sulphate poisoning, haemolytic anaemia, and anuric renal failure.² He was started on peritoneal dialysis and on the third day 25 g/l salt-poor albumin was added. Dialysate copper increased from 0.633 $\mu\text{mol/l}$ (comparable to that measured by Professor Sherlock and her colleagues) to 5.98 $\mu\text{mol/l}$ and the dialysate: serum ratio increased from 0.02 to 0.35. Clinical improvement was noted, but anuria persisted for three weeks before recovery.

Although Wilson's disease is a more complex and chronic process, the manifestations of acute copper intoxication are identical with those resulting from accidental ingestion. A trial of peritoneal dialysis with added albumin and penicillamine (more closely mimicking the physiological mechanism)

Incidence of neonatal jaundice at Kingston Hospital, 1968-76

	1968	1969	1970	1971	1972	1973	1974	1975	1976
<i>Over 2.5 kg</i>									
Total No of infants	1850	1866	2073	2092	2040	1980	1939	1900	1712
% with jaundice	13	17	11	15	16.5	23.5	39	37	41
Bilirubin >250 $\mu\text{mol/l}$ { No	4	4	0	3	7	35	48	67	78
>250 $\mu\text{mol/l}$ { %	0.2	0.2	0	0.14	0.34	1.77	2.5	3.5	4.6
<i>2.5 kg and under</i>									
Total No of infants	118	131	138	98	156	116	113	123	129
% with jaundice	15	30	14.5	18	18.5	46	59	48	35
Bilirubin >250 $\mu\text{mol/l}$ { No	0	3	1	0	4	9	13	13	7
>250 $\mu\text{mol/l}$ { %	0	2.3	0.7	0	2.6	7.8	11.5	10.6	5.4

Conversion: SI to traditional units—Bilirubin: 1 $\mu\text{mol/l}$ $\approx$ 5.9 mg/100 ml.

would seem to be indicated and might improve the poor prognosis of severe copper intoxication complicated by acute renal failure.

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¹ Cole, D E C, and Lirenman, D S, *Pediatric Research*, 1977, 11, 548.

² Evans, G W, *Physiological Reviews*, 1973, 53, 535.

Allopurinol and urinary stones

SIR,—I read with interest your leading article on the use of allopurinol in urinary tract stone disease (19 November, p 1302). A male patient presented to this department in November 1976 with a stone impacted at the right ureteric orifice which required Dormia extraction. Analysis of this stone showed it to consist of calcium, urate, oxalate, and a trace of ammonia. He had previously passed a stone spontaneously, but as he neglected to retrieve it no information about its composition is available. He had no evidence of parathyroid disease. His blood and urine urate levels were normal.

In accordance with the protocol of a trial we are conducting in the department he was started on allopurinol 100 mg thrice daily. However, he formed another stone, which was removed by left upper ureterolithotomy in November 1976. Analysis of this stone showed that it consisted of calcium ammonium phosphate; neither urate nor oxalate could be detected. He is still on allopurinol and remains well and free from stone.

The patient is one of a group of subjects currently being studied with regard to the effects of allopurinol on urinary oxalate excretion. Most subjects within the group have shown a significant reduction in urinary oxalate values. It is interesting that in the case described there has been a change in stone composition and in particular that no oxalate has been found, although this is a common constituent of stones within this area.¹

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¹ Sutor, D J, and Wooley, S E, *British Journal of Urology*, 1971, 43, 268.

Crush injuries

SIR,—Your leading article on this subject (12 November, p 1244) states that "despite the advent of effective dialysis as treatment for post-traumatic oliguric renal failure, the overall mortality of 60-70% has not changed substantially from that reported 25 years ago."

In 1974 Flynn² reported the UK overall mortality for post-traumatic acute renal failure to be 50%. At Halton we have compared two periods, 1957-64 and 1965-75, with regard to mortality in trauma patients with acute renal failure. While mortality in the first period was 64%, this was reversed in the second period to 34%.^{2,3} The most important difference in management was the institution of daily dialysis, high-calorie feeding, and subsequently total parenteral nutrition in the latter group. Lordon⁴ talks about frequent dialysis in his patients (on whom your statistics are based) but then goes on to say that dialysis in those who died was once every 2-6 days.

He also states that improvement in parenteral nutrition might have been beneficial because of the marked catabolism in his patients. Adequate parenteral nutrition is not possible without daily dialysis because of the obligatory volumes required to provide the necessary calories and protein.

Control of catabolism and adequate dialysis will improve healing⁵ and reduce the susceptibility to sepsis.⁶ If early and adequate attention is paid to debridement and amputation, daily dialysis with total parenteral nutrition to control catabolism offers a much better prognosis than one might be led to believe from your article.

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¹ Flynn, C T, in *Acute Renal Failure*, ed C T Flynn. Lancaster, MTP, 1974.

² "Renal Problems in Trauma," Seminar, R N H Haslar, February 1977. Unpublished.

³ Rainford, D J, *Anaesthesia*, 1977, 32, 277.

⁴ Lordon, R E, and Burton, J R, *American Journal of Medicine*, 1972, 53, 137.

⁵ Teschan, P E, et al, *Annals of Internal Medicine*, 1960, 53, 992.

⁶ Parsons, F M, et al, *Lancet*, 1961, 1, 129.

IUCDs—a contraindication to removal

SIR,—I report here a case which shows that if an intrauterine contraceptive device (IUCD) is to be removed for reasons other than cessation of contraceptive measures it should not be removed at the time of expected ovulation.

A 32-year-old woman presented recently with six weeks' amenorrhoea and was found to be pregnant (last menstrual period 17 September 1977). She had had four previous pregnancies, all of which had been normal resulting in normal deliveries at full term, the children's ages spanning 8 to 2 years. She had had no serious illnesses in the past other than two episodes of pneumonia in the last three years, the last attack being in 1975. Investigations had not shown any underlying cause, though she was known to have a sensitivity to house dust which caused rhinorrhoea and occasional wheezing on exposure to dust.

For contraception in the past she had relied mostly on the vaginal diaphragm. Between her first and second pregnancies she had taken oral contraceptives, but these had caused unacceptable side effects and had been discontinued. Following the birth of her last child in November 1975 she again used a diaphragm, but when she had finished breast-feeding the infant, and on resumption of menstruation, she requested a change and in October 1976 she was fitted with a Gravigard Copper-7 IUCD. Her normal menstrual cycle came every 25-28 days, each period lasting six days with a moderate loss. Over the next few months her periods lengthened to 10 days and by September 1977 this had further extended to two weeks and it was decided that her IUCD should be removed. This was done on 30 September, whereupon she resumed the use of her diaphragm. She is a sensible and responsible person who had used this form of contraception successfully for some years previously, but despite this she presented five weeks later and was found to be pregnant.

Close questioning revealed that, although she had been scrupulous in observing contraceptive measures since the removal of the IUCD, she had had intercourse 48-72 h before this was done. Removal was effected 13 days after the first day of her last menstrual period and it therefore seemed likely that the IUCD had been removed just in time to allow implantation of a fertilised ovum. In view of all these circumstances she was referred to a consultant gynaecologist, who agreed to terminate the pregnancy.

Of course it is possible that this unfortunate sequence of events did not occur and that she

became pregnant simply because of failure of her diaphragm. Nevertheless, it is possible that it did occur in the manner suggested and this possibility should be borne in mind in future. In the large majority of women—that is, those with regular menstrual cycles—such an unwanted pregnancy can be avoided simply by ensuring that IUCDs are not removed during a time, say, seven days each side of midcycle. So far as I can tell from inquiries made so far this is not standard practice. I believe that there is a good case for observing this precaution in future.

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Drug-related red-cell aplasia

SIR,—The interesting report by Drs G Reid and A C Patterson (3 December, p 1457) of a case of red-cell aplasia apparently due to gold therapy illustrates the difficulties encountered in identifying cause and effect in this and other marrow hypoplasias. Wintrobe¹ lists 13 drugs believed to cause red-cell aplasia, while another review includes 16.² In some instances the evidence rests on only a very few anecdotal reports. The situation is also confused by the fact that in some cases the patient's underlying disorder was one itself occasionally associated with red-cell aplasia. In the present case the patient had received at least two other drugs known to cause marrow depression and was suffering from rheumatoid arthritis, one of a spectrum of connective tissue disorders in which red-cell aplasia may occur,³ presumably on the basis of autoimmune disease.³ The suspicion that a particular drug is responsible is naturally increased when withdrawal of a single drug, any others being continued at their previous dosage, is followed by remission of anaemia, though for obvious reasons this is rarely, if ever, justifiable in practice. Yet another difficulty is the tendency of red-cell hypoplasia to remit spontaneously without treatment; indeed, it is possible that many cases go unrecognised unless the patient is under haematological surveillance.⁴

These diagnostic pitfalls are further illustrated in the following case, seen in this clinic. A man of 72 was referred by Dr R V Stone because of a history of anaemia of six weeks' duration. There was no history of exposure to drugs apart from chlorotrianisene (TACE), which the patient had taken for 10 years after a clinical diagnosis of carcinoma of the prostate, and prednisolone (40 mg/day), which had been given for a month after the onset of the anaemia. Clinical examination showed mucosal pallor and a moderately enlarged prostate but no other signs. There was no evidence of metastatic disease, and the blood creatinine concentration and serum acid phosphatase activity were normal. The blood count on 21 July 1977 was: haemoglobin 6.6 g/dl, leucocytes $13.5 \times 10^9/l$ ($13\,500/mm^3$) (neutrophils 84%), platelets $224 \times 10^9/l$ ($224\,000/mm^3$), reticulocytes 0.02%. Marrow aspiration confirmed profound red cell hypoplasia, granulopoiesis and thrombopoiesis being normal. Since high-dose oestrogen therapy may diminish erythropoietin production⁵ and there is a report of marrow depression in animals following prolonged oestrogen administration⁶ it was postulated that the patient's red cell aplasia was due to this therapy, which was stopped, as was the prednisolone, to which there had been no response. A blood transfusion was given. Further investigation showed that his serum contained a small quantity of paraprotein, identified as IgG-kappa type, but there was no other evidence of lymphoma or immunoproliferative disease and it was decided that his was an example of "benign monoclonal gammopathy."