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.

ADRIAN CARO

Dereham, Norfolk

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.

MARY N SMITH
RICHARD G WILSON

Department of Paediatrics,
Kingston Hospital,
Kingston upon Thames

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.

JOHN WALLACE

Glasgow and West of Scotland
Blood Transfusion Service,
Law Hospital,
Carlisle, Lanarkshire

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.

H L F CURREY
C G BARNES

Department of Rheumatology,
The London Hospital,
London E1

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 $\mu\text{mol/l}$ %	4	4	0	3	7	35	48	67	78
	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 $\mu\text{mol/l}$ %	0	3	1	0	4	9	13	13	7
	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.