a case under a doctor's care suddenly choose to "go it alone" at some point?

Probably the forthcoming report of the Brodrick committee will favour less rather than more formality in relation to coroner's inquiries. All the same, a doctor might well feel a little uneasy about giving his usual ready co-operation with a coroner in a case where a health visitor has been involved if he thinks she might be going to demonstrate publicly that she regards such co-operation as a breach of professional confidence. Such a demonstration would not seem to favour happy relations between the health visitor and doctor. The obstruction and discourtesy to the coroner who finds himself presiding over such a situation is also an unfortunate matter, and it seems to me it would be a good idea for the representatives of all concerned to get together and consider the question.

I would hope that the Health Visitors' Association could be persuaded to withdraw from its present position, as the association in my view is letting down its members badly and prejudicing good relations between them and the doctors.—I am, etc.,

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Eye,
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¹ *Health Visitors and the Courts*. London, Health Visitors' Association, May 1971.

Fibrin/Fibrinogen Degradation Products in Glomerulonephritis

SIR,—Dr. A. R. Clarkson and others (21 August, p. 447) have produced evidence that the urine concentration of fibrin/fibrinogen degradation products (F.D.P.) provide a reliable and sensitive index of activity, progression, and natural history in proliferative glomerulonephritis. They suggest that the F.D.P. are derived from lysis of intra-glomerular fibrin deposits and limited proteolysis of fibrinogen filtered through an abnormally permeable basement membrane.

Some years ago¹ we had demonstrated the presence of fibrinogen in the urine of certain patients suffering from the nephrotic syndrome due to glomerulonephritis. This fibrinogenuria was estimated in the range of 0.057–0.230 g/24 hrs with a clearance two to ten times smaller than the total proteins clearance. In this work, as well as in a previous paper,² we had studied the different factors including proteolysis, which may, in certain circumstances, have a suspensive effect on the fibrinogenuria, resulting from the passage of plasma fibrinogen through the damaged glomerulus.—I am, etc.,

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General Clinic of Athens,
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¹ Yatzidis, H., *Nature*, 1964, **201**, 187.

² Yatzidis, H., and Richet, G., *Revue Française d'Etudes Cliniques et Biologiques*, 1957, **2**, 717.

Lung Transplantation

SIR,—Reference was made in a paper entitled "Lung Transplantation in a Patient with Fibrosing Alveolitis" (14 August, p. 391) to the use of antilymphocytic globulin labelled with radioactive iodine-125. It was suggested (p. 395) that this material was "eliminated in two days," which could imply that the binding of the label to the globulin

was very poor. In fact globulin-bound radioactivity was followed in the blood plasma for nine days, at which time the radioactivity per ml was 10% of its initial value. A graph of the change in plasma radioactivity with time was approximately mono-exponential, after the initial equilibration period, with a biological half-life of 70 hours.—We are, etc.,

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Arteriosclerosis, Duodenal Ulcer, Blood Group, and Secretor Status

SIR,—The association of peptic ulcer and abdominal aortic aneurysm has been emphasized^{1,2} and a similar link suggested between arteriosclerosis and peptic ulcer by Barabas *et al.* (26 June, p. 768). Two series of patients suffering from occlusive peripheral arteriosclerosis have contained an excess of blood group A subjects,^{3,4} yet it is well known that duodenal ulcer is associated with blood group O.⁵

	N	O	A	B	AB
Male arteriosclerotics	554	216 (39.0%)	268 (48.4%)	52 (9.4%)	18 (3.2%)
"Proved" D.U.	81	31	37	10	3
"Possible" D.U.	28	12	13	3	—
Total D.U.	109	43 (39.4%)	50 (45.9%)	13 (11.9%)	3 (2.7%)
Blood donors (Leeds region)	98,054	45,399 (46.3%)	41,006 (41.8%)	8,510 (8.7%)	3,149 (3.2%)
Male peptic ulcer (York)	1,526	846 (54.8%)	557 (36.5%)	87 (5.7%)	36 (2.4%)

This dilemma may be clarified by our findings in 554 men with occlusive peripheral arteriosclerosis recently subjected to retrospective analysis in relation to blood group incidence. Because the surgeon contributing the majority of cases (Shucksmith, personal communication) had long recognized a clinical association between peptic ulcer and claudication, most patients had been asked about dyspepsia. A patient was classified as having a "proved" duodenal ulcer if it had been demonstrated by a prior barium meal examination, or by inspection of the duodenum during an intra-abdominal vascular operation, or by post-mortem study, or if he had had an operation for duodenal ulcer. Information was also collected with regard to patients who gave a suitable history of dyspepsia or of haematemesis or melaena but in whom definite evidence was lacking that the peptic ulceration was duodenal in site. Known gastric ulcers were excluded.

In all, 14.6% of 554 men were proved to have or to have had duodenal ulcer, and a further 5.1% fell into the "possible" category. The blood group distribution of the duodenal ulcer subgroup followed closely that of the larger arteriosclerotic group, but was markedly different from the blood donor contrast data and from a typical series of male peptic ulcer patients in York.

Our figure of 19.7% total duodenal ulcer cases is in close accord with the finding by Allan and Dawson⁶ of 19% duodenal ulcer in 353 male Aberdeen myocardial infarction survivors though they noted an excess of duodenal ulcer in the group O patients.

Because of the known deficiency of secretors in duodenal ulcer⁷ saliva from 66 currently attending survivors from the original 554 patients was tested for the ABH substances.⁴

Assuming a frequency of 77% secretors,^{7,8} this is a significant deficit of arteriosclerotic secretors ($\chi^2=19.1$, $n=1$, $P<0.001$). It may be that the non-secretor is not only liable

	O	A	B	AB	Totals
Sectors	12	20	2	2	36 (54.6%)
Non-sectors	13	15	2	—	30 (45.4%)

to duodenal ulcer but also to arteriosclerosis, and this could explain the paradoxical associations of blood groups O and A respectively in the face of the clinical association of the two diseases.

We wish to thank Dr. J. D. Crossland for the blood donor data, Dr. C. N. Pulvertaft for the York peptic ulcer blood group data, and Mr. K. Cumberbatch for technical assistance in testing saliva samples for ABH substances.

—We are, etc.,

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¹ Jones, A. W., Kirk, R. S., and Bloor, K., *Gut*, 1970, **11**, 679.

² *British Medical Journal*, 1971, **1**, 129.

³ Kingsbury, K. J., *Lancet*, 1971, **1**, 199.

⁴ Hall, R., Bunch, G. A., and Humphrey, C. S., *Arteriosclerosis Research*, in press.

⁵ Aird, I., Bentall, H. H., Mehigan, J. A., and Roberts, J. A. F., *British Medical Journal*, 1954, **2**, 315.

⁶ Allan, T. M., and Dawson, A. A., *British Heart Journal*, 1968, **30**, 377.

⁷ Clarke, C. A., *et al.*, *British Medical Journal*, 1956, **2**, 725.

⁸ Camps, F. E., Dodd, B. E., and Lincoln, P. J., *British Medical Journal*, 1969, **4**, 457.

Dermatoglyphics and Chromosomes in Cat-eye Syndrome

SIR,—In reply to Drs. C. W. Darby and D. T. Hughes (3 July, p. 47) I should like to report the results of the dermatoglyphic analysis I carried out¹ in a patient who was extensively studied by Curcio.² The patient was a newborn baby displaying several of the typical cat-eye syndrome features: anal atresia, rectovestibular and pre-auricular fistulae, presence of short metacarpal bones, and 13 ribs. In this patient, as in other published cases,^{3,4} there was an extra chromosome, about half the size of those in group G. As in the case reported by Drs. Darby and Hughes, the patient's father had a normal karyotype, but the mother was found to have the same marker chromosome. The dermatoglyphic pattern of the patient¹ was characterized by the presence of 10 ulnar loops on digits; distal t (axial) triradius; and a whorl pattern in the proximal hypothenar area on the right palm, and a radial loop in the hypothenar area on the left palm.

As suggested by Drs. Darby and Hughes it is possible that the peculiar chromosome anomaly of cat-eye syndrome affects the growth pattern of dermal ridges. However,