We have re-examined the radiographs of the last six cases of pneumomediastinum from the intensive care unit of the Institute of Neurological Sciences and six from the intensive care unit of the Victoria Infirmary, Glasgow. Of the 12, four show the continuous diaphragm sign. All four patients had suffered chest trauma and were examined supine. It is probable that the sign is seen more readily when the patient is in this position. It is only when there is no overlap of heart on the dome of the diaphragm that the sign will be seen clearly (see fig.). We have had one case where there was such overlap and the air under the heart was projected below the level of the upper surface of the diaphragm.

Appreciation of the significance of this sign will add to the likelihood of pneumomediastinum being diagnosed, especially as only a frontal view of the chest is taken in most cases of trauma. So many specialties are involved now in the management of patients in intensive care units that it is felt that this sign ought to be more widely appreciated. It is one which is easily seen if one thinks to look for it.—We are, etc.,

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1 Levin, B., *Clinical Radiology*, 1973, 24, 337.

Doctors, Drivers, and Confidentiality

SIR,—May I comment on your interesting medicolegal article (2 March, p. 399) on confidentiality?

I am glad to learn that in the motoring case the General Medical Council would not have acted had the doctor revealed the patient's name and address. It seemed to me at the time that, though he was acting from the best of motives, it was carrying confidentiality to an unreasonable extent. If a psychiatrist or a venereologist reveals that a patient has attended, this might do him harm, but what harm can result simply from the knowledge that someone attended a general practitioner's surgery?

It also always seems to me that there is an immense difference between revealing some harmless finding such as an injury and revealing that the patient has a chancre, tuberculosis, or a psychosis. I at least,

especially as a psychiatrist, feel that it is not right that one can be forced in court to reveal information which a patient gave under the impression that it was in strict confidence (though I agree with your legal correspondent that the courts, especially nowadays, are very understanding of our problems). However, I must admit that even outside court there can be a grave conflict as to the right course—for example, when (as befell a colleague) a schizophrenic patient confesses to a murder, which could be a delusion but might be fact, or when a psychopath announces that he is going to commit some serious offence. I would add that though this whole matter is mostly dealt with as an ethical problem basically it is a very important practical matter, because unless the patient believes he can talk under the veil of secrecy he may withhold vital information which may both impair his own treatment and mean that the doctor does not know of some condition which may make the patient a public danger.

Incidentally it seems to me that one method we sometimes use to resolve our dilemma—notifying the medical officer of the patient's employers—is in effect equivalent to informing the employers if, as in the case of a transport worker, the medical officer has a duty to notify them that an employee is unfit for his job. Might it not be an idea for the B.M.A. or the General Medical Council to set up some body or committee to which the doctor could refer his problem and which would take the responsibility of passing on the information if it considered this to be necessary? At present the medical defence organizations give us excellent advice, but the responsibility of accepting it still remains with the doctor.—I am, etc.,

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Death during Dental Anaesthesia

SIR,—With reference to your medicolegal report (2 February, p. 207) and the letter from Dr. D. Blatchley (2 March, p. 391), I feel this is a typical example of that old adage, "a little learning is a dangerous thing." I am more than ever firmly convinced that dental anaesthesia should be practised only by anaesthetists.—I am, etc.,

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Pre-eclampsia and the Kidney

SIR,—In your leading article (16 March, p. 468) you refer to certain views I hold on pregnancy nephropathy in which it had appeared that I indicated that renin was "the aetiological factor" in the production of this condition.

What I have always maintained is that a myometrial resistance to stretch can become responsible for the condition by causing ischaemia of the outer cortex of the kidney, which in turn produces hypertension and the other signs of the syndrome. Renal cortical ischaemia is the underlying aetiological state.

Of the various hypertensive products there produced, renin is one and in that context was signalled out for mention to draw attention to the ischaemic state. Its absence does not exclude other hypertensive products of ischaemia.—I am, etc.,

JOHN SOPHIAN

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SIR,—Your leading article (16 March, p. 468) is a fair and comprehensive assessment. Pre-eclampsia has no features of an immune complex disease but is a particular form of hypertension. The features are best explained by a theory linking uteroplacental ischaemia to hypertension, in which increased sympathetic tone is associated with secondary low-grade intravascular coagulation. Recent work on acute renal failure¹ also links vasoconstrictor effects with intrarenal coagulation. An unidentified mineralocorticoid may be the answer; there are certainly increased levels of prolactin in this state.

First might I add a few comments on intravascular coagulation, as some of my recent work which is highly relevant to pre-eclampsia has escaped your notice. My conclusions have been that intravascular coagulation, which itself can be productive of vasoconstrictor products, is secondary to hypertension occurring in the context of pregnancy,² and that it can also arise from placental damage,³ in which latter situation escape of phospholipid procoagulant into the circulation has been detected.⁴ A crucial finding has been that the inhibition of fibrinolysis that facilitates intravascular coagulation is probably due to the action of placental lactogen.⁵ That there is increased fibrin formation in the placenta in pre-eclampsia can be detected by fibrinogen chromatography of patient's plasma, when high-molecular-weight fibrin polymers can be shown.⁶ Furthermore, the non-selectivity of proteinuria has been shown by animal experiments to be due to intrarenal coagulation rather than functional vasoconstriction.²

As for histology I must make the points, firstly that mesangial hyperplasia can be due to fibrin alone, and secondly that the histology of pre-eclampsia is not specific, for it can be seen in other types of nephrosis in pregnancy. What worries me about the report by Dr. O. M. Petrucco and his colleagues (16 March, p. 473) of IgM and complement in the mesangium and arterioles in pre-eclampsia is that immune complexes of this composition would cause what Germuth⁶ has designated "mesangiopathic changes." Class II immune complexes of this size would cause focal glomerular necrosis or capillary loop lesions, as are seen in systemic lupus erythematosus. That these histological appearances are not seen in pre-eclampsia suggests that the accumulation of these proteins reflects only increased endothelial permeability. Increased transudation of proteins through arteriolar walls is well known in hypertension, and would be increased by local endothelial permeability factors, as for example platelet amines from local intrarenal coagulation.⁷ I think myself that these pre-eclamptic fluorescent changes should have been compared with those of non-pregnant patients with essential malignant hypertension.