using larger numbers of patients, and with comprehensive laboratory coverage.

My thanks are due to Dr. P. J. Sequeira and Dr. J. Tobin for the virus studies, Dr. E. Busill Jones for clinical help, and Dr. A. W. Galbraith, of Geigy (U.K.) Limited Pharmaceuticals Division, for supplying the drug and placebo.

—I am, etc.,

J. T. LEEMING.

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Withington Hospital,
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REFERENCE

- ¹ Sabin, A. B., *J. Amer. med. Ass.*, 1967, 200, 943.

Plasminogen-activator-producing Tumour

SIR,—The identification by Dr. J. F. Davidson and others (11 January, p. 88) of a plasminogen-activator-producing tumour is fascinating. However, on the evidence produced I am doubtful about two of the conclusions.

There is no evidence that the tumour was rapidly growing—that is, multiplying, extending, and invading during the time that it was producing plasminogen-activator. The proximal extension of the swelling revealed only serosanguineous material which most likely reflects an accumulation of tumour fluid. It is conceivable, and perhaps likely, if one preserves the view that tumours may require a fibrin network as a matrix for growth, that this secondary tumour with its demonstrated thromboplastic activity grew rapidly first and then produced plasminogen-activator.

It cannot be concluded that a rare tumour of lung showed pathological plasminogen-activator production. This property can only be ascribed to the peripheral secondary carcinoma, presumably arising from a lung primary. No assessment is described of the lung primary's activity, and it may be inferred that the primary did not in fact produce plasminogen-activator, for, although haemorrhagic, it did not bleed. Equally, the other secondary tumours, similarly haemorrhagic, did not bleed.

It would seem worth while to comment that lung tissue is known to contain substantial amounts of tissue plasminogen-activator.¹ Malignant cells arising from the lung may therefore have a potential for producing large quantities of plasminogen-activator. The rarity in this report may be the circumstances under which a secondary from a giant-cell carcinoma of lung showed such an activity.

—I am, etc.,

Beaconsfield, Bucks.

A. J. JOUHAR.

REFERENCE

- ¹ von Kaulla, K. N., *Chemistry of Thrombolysis: Human Fibrinolytic Enzymes*, 1963. Springfield, Illinois.

Treatment and Prevention of Poisoning

SIR,—I was interested to read your leading article (28 December, p. 787) concerning the treatment and prevention of poisoning.

Approximately 75% of adults who have ingested poison present in hospital within six hours.¹ The same percentage of children present within two hours. If we subtract the length of time an ambulance takes to bring the patient to hospital then the earliest time

that treatment may be undertaken is even shorter. An important early stage in the treatment of poisoning is getting the poison out.² Karlsson and Norén³ have successfully used dilute solutions of copper sulphate as an emetic in poisoned children. Ninety-eight per cent. of 100 children vomited within 14 minutes. Twenty-eight per cent. vomited twice. They found copper sulphate safe and more reliable than ipecacuanha.

In most British hospitals it is the custom for the nursing staff to perform lavage.¹ Inducing emesis in a conscious patient is probably a less hazardous procedure. About 20% of adults are admitted to hospital unconscious; children are rarely unconscious on admission. If suitable packs of dilute copper sulphate solution were carried as part of the standard equipment in ambulances, these could be administered to co-operative conscious patients by ambulance personnel, as soon as the patient was collected. With this arrangement, by the time the patients arrived at hospital they would probably have vomited. It would be necessary to carry a suitable receptacle in the ambulance.

With the increasing number of cases of ingested poison I wonder will the day come when as well as the obstetric flying squad and coronary ambulance we may have the "overdose" ambulance? Or will we eventually evolve a system where there is a fleet of multipurpose ambulances equipped to start the early treatment of patients suffering from any acute medical emergency endangering life?—I am, etc.,

MICHAEL BURKE.

Gateshead,
Co. Durham.

REFERENCES

- ¹ Burke, M., *Practitioner*, 1968, 201, 786.
² Mathew, H., Mackintosh, T. F., Tompsett, S. L., and Cameron, J. C., *Brit. med. J.*, 1966, 1, 1333.
³ Karlsson, B., and Norén, L., *Acta paediat. scand.*, 1965, 54, 331.

Cerebral Haemodynamics and Metabolism in Hypnosis

SIR,—We have read with interest the articles on the value of the use of hypnosis in medicine and on the neurophysiological interpretations of the hypnotic state (12 October, pp. 67 and 71).

We have carried out a study of cerebral haemodynamics and metabolism in five normal subjects in deep hypnotic trance with analgesia and amnesia at the arousal. The study included the determination of cerebral blood flow, cerebrovascular resistances, and cerebral metabolic rate of oxygen, according to the methods previously described.¹ In comparison with a group of normal but alert subjects it was found that the hypnotic state does not cause any modification of the examined parameters (see Table). However, we

Cerebral Haemodynamics and Metabolism in Normal Subjects (Mean values $\pm$ S.D.)

	No. of Cases	Cerebral Blood Flow (ml./100 g. Tissue/min.)	Cerebrovascular Resistances (mm. Hg./ml./100 g./min.)	Cerebral Metabolic Rate of O ₂ (ml./100 g. Tissue/min.)
Alert subjects	6	54.9 $\pm$ 10.8	1.90 $\pm$ 0.49	3.68 $\pm$ 0.77
Hypnotized subjects	5	51.0 $\pm$ 4.7	2.08 $\pm$ 0.31	3.57 $\pm$ 0.59

wish to stress the fact that during the hypnotic trance the values of the standard deviation for each of the above-mentioned parameters were clearly below the levels of the normal and alert subjects.

We think that the difference which resulted between the hypnotized and the alert subjects may be due to a state of anxiety and fear caused by the method of examination used; as in fact this state was present only in the alert subjects, even if clinically not evident.

This work is part of a wider investigation, and a full report is in preparation.

—We are, etc.,

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e Metodologia Clinica (II),
Milan University, Italy.

REFERENCE

- ¹ Della Porta, P., Maiolo, A. T., Negri, V. U., and Rossella, E., *Metabolism*, 1964, 13, 131.

C.S.F. Rhinorrhoea

SIR,—Your leading article on C.S.F. rhinorrhoea (18 February, p. 137) contains the following: "... Galen thought that the C.S.F. was formed by the brain, stored in the ventricles, and normally excreted through the nose."

It is now generally accepted that the C.S.F. was never described in antiquity and in fact the first certain reference to it was made by Berengario da Carpi as late as 1521. The first clear description of it was given by another Italian anatomist, Massa, 15 years later.¹ Galen was in fact referring to the animal spirits or vapours of the soul, which were thought to be formed in the vascular network said to be present in man at the base of the brain—the *rete mirabile*—but found in fact only in ungulates. The resulting waste products were excreted through the pituitary fossa and the olfactory plate if fluid, and, if gaseous, through the cranial sutures. There is, however, no suggestion whatever that the former may have been C.S.F.

I am sure that you would wish to avoid inaccuracies in scientific data submitted to your journal, and I see no reason why historical information should not be equally precise.

—I am, etc.,

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Sub-Department of the History
of Medicine,
University College,
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REFERENCE

- ¹ Clarke, E., and O'Malley, C. D., *The Human Brain and Spinal Cord*. University of California Press, Berkeley, California, 1968.

SIR,—The leading article on C.S.F. rhinorrhoea (18 January, p. 137) was very interesting because it reminded the readers of an unusual clinical problem. We have much to learn about the anatomy of the roof of the nose, as well as the abnormalities. A patient of mine suffering from spontaneous C.S.F. rhinorrhoea was found to have a congenital fistula between his subarachnoid space and the roof of one of his frontal sinuses. The concept of a focal atrophy of the cribriform plate is also interesting. It would explain why a patient develops meningitis after removal of