formation of anticoagulant fibrinogen degradation products aggravates the haematologic defect. Moreover, to control the replacement of fibrinogen, as is suggested, is difficult, since the laboratory tests commonly employed in acute fibrinolytic conditions (thrombin time, Fibrindex, etc.) are at best semi-quantitative. Even if a rapid method of fibrinogen estimation is used[6] the findings can be misleading, for the blood level of fibrinogen may be higher than that indicated by the test owing to the presence of anticoagulant fibrinogen degradation products.[7] In our experience in obstetrical patients[4 5] the clinical condition of the patient may be a better guide to therapy than the fibrinogen level determined in the laboratory, and fresh whole blood is a better substitute than fibrinogen alone.

Lastly, the risk of E.A.C.A. causing intravascular thrombosis (apart from that presented by the original stimulus for defibrination) when it is given for a relatively short period (as in acute obstetrical conditions) is negligible. This is because the drug is excreted through the kidneys very rapidly.—I am, etc.,

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Polycythaemia and Chorea

SIR,—Your leading article (23 December 1967, p. 696) referring to Gautier-Smith and Prankerd's two cases of progressive chorea due to polycythaemia rubra vera prompts me to summarize a similar case.

An old lady of 77 was referred to me at Barnet General Hospital in October with a six-months history of fidgety movements. Examination revealed that she was very arteriosclerotic, and the blood pressure was 190/100 mm. Hg. She showed grimacing movements of the face and writhing movements of the tongue, and had a moderately severe dysarthria. There were marked choreic movements to the trunk, leading to a lurching gait, and typical choreiform movements of the fingers and toes. Apart from extensor plantar responses the rest of the neurological examination was normal.

Regarding her as a probable case of senile chorea due to cerebral atheroma, treatment was started with thiopropazate (Dartalan) 10 mg. three times a day. At her next attendance one month later she was remarkably improved, and her daughter, an excellent witness, stated that the movements had ceased completely two days after the drug was commenced. However, the blood taken at the first attendance showed that the haemoglobin was 18.5 g./100 ml. and packed cell volume was 66%. A full blood count was therefore ordered, and revealed: R.B.Cs 8,000,000/cu. mm., haematocrit 64%, M.C.H.C. 29%, M.C.V. 80 cu. mm., platelets 320,000/cu. mm., white blood count 8,800/cu. mm., neutrophils 58%, eosinophils 3%, basophils 2%, lymphocytes 36%, and monocytes 1%.

Re-examination at this time showed no enlargement of spleen, and this, together with a normal white cell count and platelet count, suggested that the polycythaemia was probably of secondary type, perhaps due to chronic bronchitis. It was thought advisable to withdraw 1 pint (0.6 l.) of blood, but this had no noticeable effect on the patient. Because of the side-effect of drowsiness the thiopropazate was progressively withdrawn, but at a dose of 10 mg. daily the choreiform movements returned, although not so severe as when she was first seen. She is now well controlled on thiopropazate 20 mg. daily together with orphenadrine 100 mg. daily.—I am, etc.,

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Transplant Lung

SIR,—We feel that the authors of the article on transplant lung (13 January, p. 80) are premature in describing this as a new syndrome. Previous reports by Rifkind[1] and Hamburger[2] have referred to "transplant pneumonia." The discussion by Mr. Slapak and others, however, on the pathogenesis of this syndrome in terms of alveolar-capillary block is extremely speculative for two reasons. Firstly, they produce no histological evidence of a thickened alveolar membrane; in the literature they have cited there is one illustration[1] showing hypertrophied alveolar cells in a case which also shows the alveoli filled with a fluid containing many cysts of *Pneumocystis carinii*. Secondly, their evidence that the dysfunction in lung physiology is a decrease in oxygen diffusion is weak because they do not report the effect of breathing 100% oxygen, a procedure which should overcome a diffusion block.[3] In addition, it has been recognized for some time that the commonest cause of hypoxaemia, hypocapnia, and a low CO_2 diffusing capacity is imbalance of pulmonary ventilation and perfusion.[3 4]

The sentence "Our patient's vital capacity was 100% of the predicted value but his F.E.V.$_1$/V.C. was 78%, showing a mild degree of restriction," appears contradictory to us. Restrictive lung disease can only be diagnosed when the vital capacity is reduced. The F.E.V.$_1$/V.C. ratio is a measure of airways obstruction; the value quoted is normal.

We do not feel the authors have fully assessed the role of alveolar and interstitial oedema, especially since the chest *x*-rays of their cases are similar to those found in pulmonary oedema. The evidence for an immunological basis for this syndrome is extremely circumstantial. The decline in lung function was not accompanied by any deterioration in renal function, as might be expected if cross-reacting antibodies are involved. It is also surprising that investigators have so far been unable to reproduce this condition experimentally in the dog (and in our experience we have not encountered it either), since in many other respects the dog, after renal transplantation, has shown similar changes to those described in man.

There is little evidence for the conclusions these authors have reached or the hypotheses they have proposed.—We are, etc.,

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Rinsing the Dialyser

SIR,—I read with interest the paper by Dr. D. B. Evans and others (16 December 1967, p. 651) on different techniques of washing-back the blood from the two-layer Kiil dialyser at the end of dialysis. Evidence is presented that a rinse with 1 l. of normal saline (after tipping the dialyser vertically during half an hour) yields best results. However, with this technique the patient receives 500–700 ml. of 0.9% saline at the end of each dialysis. In patients on twice-weekly haemodialysis this equals an extra weekly sodium load of approximately 150–214 mEq. This seems to be highly undesirable in chronically dialysed patients who need rigorous dietary sodium restriction, which is (together with appropriate ultra-filtration during dialysis) the cornerstone in correction of hypertension.

I refer therefore to a recently published air-rinse technique[1] which yields equally excellent results without flushing extra water and sodium chloride into the patient. Results (approximately 5 ml. residual blood loss in the dialyser) were confirmed in our unit. Dangers of air embolism are virtually zero in nurse-supervised hospital dialysis and might be eliminated in home dialysis procedures by a rather simple device which blocks automatically the outflow bloodline immediately before the venous cannula when the first air bubble passes a fotocell.—I am, etc.,

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Elastic Band Injuries

SIR,—I was interested to read the series of letters following the description of artifact ulcers produced by elastic bands by Mr. I. D. Kitchin and others (22 April 1967, p. 218). Dr. L. Cohen (6 May, p. 376) described the result of allowing an elastic band to slip over a tooth, and Dr. J. G. B. Thurston (same page) reminded us of his previous description of ulcers produced around fingers by elastic bands. Mr. J. P. Turney (13 May, p. 445) described an ulcer in the upper arm produced by a group of three elastic bands, and Dr. K. Dawson-Butterworth (20 May, p. 510) described skin ulceration with a deeply buried elastic band in the lower third of the