Day after Admission	Aprotinin (Trasylol) Therapy	Investigation*							
		Prothrombin Time (12-15 sec)	Thrombin Time (12-15 sec)	Fibrinogen Titre (1/256-1/512)	Fibrin Degradation Products (<1/32)	Platelet Count (150-400 × 10^3/mm^3)	Euglobulin Lysis Time (2-7 hr)	Factor V (50-200%)	Factor VIII (50-200%)
Case 1									
2	+	29	29	1/256	—	110	—	—	—
3	+	20	18	—	—	66	—	—	—
4	+	19	20	1/256	1/64	79	26	60	750
5	–	16	22	1/512	1/128	28	>5 <18	100	270
6	–	—	—	—	1/256	53	—	—	—
12	–	16	14	1/256	1/16	Ample	>5 <21	—	—
Case 2									
0	–	48	27	1/128	—	43	—	6	40
3	+	20	24	—	—	31	—	—	—
5	+	18	—	—	—	84	—	—	—
6	–	17	16	1/512	1/32	132	>5 <18½	100	770
7	–	15	17	—	1/128	Ample	—	—	—
11	–	18	—	—	1/16	Ample	—	—	—

*Normal values for each test in parentheses.

Three cases of disseminated intravascular coagulation in patients with acute pancreatitis have been reported.[1] Two of the three patients died, and at necropsy renal cortical necrosis was found associated with multiple microthrombi in many organs. The patient who survived was treated with heparin. The same authors reported[1] that a trypsin infusion in dogs produced significant defibrination with numerous microthrombi deposited in the lungs and kidneys. Acute respiratory distress syndrome, ascribed to the effects of fluid overload[2] or an alveolar-capillary block,[3] is a well-recognized complication of pancreatitis. Pulmonary fibrin deposition does not seem to have been considered as a possible cause of lung damage in pancreatitis, though it is common in disseminated intravascular coagulation[4] and probably plays a significant role in the shock lung syndrome.[5]

Both our patients had good laboratory evidence of a consumptive coagulopathy associated with acute pancreatitis. Aprotinin is a very active fibrinolytic inhibitor and 100 times more potent than tranexamic acid in terms of molar concentration.[6] If fibrin deposition has a significant role in the production of organ damage in pancreatitis any inhibition of fibrinolysis would obviously be harmful. Some reports have suggested that heparin or even streptokinase would be a more logical form of treatment.[7] The deterioration in pulmonary function in our patients associated with aprotinin therapy and the recovery on stopping the infusion may have been fortuitous. Nevertheless, there was a definite reduction in plasma lytic activity in one patient during the infusion and a striking rise in fibrin breakdown products in each case after the treatment ceased. Far from being a harmless remedy, aprotinin may well be contraindicated in acute pancreatitis when there is evidence of disseminated intravascular coagulation.

I thank Mr. L. T. Cotton and Dr. R. W. Williams for permission to report the cases of patients under their care.

—I am, etc.,

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Alpha-fetoprotein in Amniotic Fliud in Early Normal Pregnancy and Intrauterine Fetal Death

SIR,—A number of investigators have recently stressed the importance of the assay of alpha-fetoprotein (A.F.P.) for the prenatal diagnosis of congenital malformations. The radioimmunoassay method, which is used most often, is rather complex and unsuitable for small laboratories with restricted equipment.

The simple and rapid immunodiffusion method of Mancini as modified by Fahey and McKelvy[1] has been applied by us for evaluation of the A.F.P. levels in amniotic fluid which has been previously concentrated in 50% gum arabic.

The levels of A.F.P. in amniocentesis samples taken from 37 normal pregnant women at the 10th-12th week of pregnancy and from six patients in whom intrauterine fetal death had been diagnosed are shown in the table. The average A.F.P. value (29 μg/ml) in the amniotic fluid from normal pregnancies was in agreement with those reported by Brock and Sutcliffe[2] and by Nevin *et al.*[3] In the samples of the fluid from the cases of intrauterine fetal death, however, the mean value (55·4 μg/ml) was significantly higher.

Group	No. of Cases Studied	Alpha-fetoprotein Concentration in Amniotic Fluid (μg/ml)	
		Mean ± S.D.	Range
Normal pregnancy	37	29·0 ± 7·5	17·5-41·5
Intrauterine fetal death	6	55·4 ± 32·0	24·6-100·0

The results obtained indicate that the method applied by us can be useful for routine assays of A.F.P. in material derived from both normal and abnormal pregnancies. Thus it could be of great importance in the antenatal diagnosis of congenital defects. The concentration of the amniotic fluid makes estimations possible even in cases in which the levels of A.F.P. are low.—We are, etc.,

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Prazosin in Patients with Chronic Renal Failure

SIR,—I was interested to see the letter from Drs. Priscilla S. Kincaid-Smith and A. S. P. Hua (24 August, p. 520) on beta-adrenergic blocking agents in renal failure and would like to add a few comments with particular reference to the use of prazosin in patients with chronic renal failure.

Prazosin, a relatively new vasodilator drug, is a valuable addition to the range of drugs now available for the treatment of hypertension. I have found it valuable when used in combination with other drugs including propranolol, methyldopa, clonidine, frusemide, and chlorothiazide in the treatment of severe hypertension in patients with chronic renal failure. In the patients studied the initial ^{51}Cr-EDTA clearances varied from 28 to 47 ml/minute. The addition of prazosin to the antihypertensive regimen produced a significant fall in the blood pressure which was associated with an average fall in the ^{51}Cr-EDTA clearance of 8 ml/min (4-13 ml/min). Thereafter the ^{51}Cr-EDTA clearance stabilized. I would, however, like to add a note of caution when prazosin is employed in patients with chronic renal failure. I have found that such patients may be very "sensitive" to the drug, and small doses such as 1 mg twice daily should be used initially. In the patients I have studied the maximum daily dose has not exceeded 6 mg.

It is claimed that prazosin does not produce any significant postural effects. I have found, however, that the addition of prazosin to the antihypertensive regimen produces significant postural falls in both systolic and diastolic blood pressure. It was not possible to determine in my study whether the observed postural falls in blood pressure were due to the direct effect of prazosin or perhaps