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

to prazosin having a potentiating effect on the other antihypertensive drugs also being employed at the same time. Clearly this aspect requires further study.—I am, etc.,

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Hernias in Children

SIR,—I have read with interest your leading article on the above subject (31 August, p. 540), and though only an "ordinary" general and not a paediatric surgeon I would like to make three points. These may not be altogether out of place as the majority of hernias in neonates probably reach general and not special hospitals.

Firstly, I cannot agree that spontaneous disappearance never occurs. Over the years I have seen several intermittent neonatal hernias which have ceased to appear after a few months. One has to make up one's mind either to take the very small risk of strangulation occurring or, on the other hand, of doing a possibly unnecessary operation. My own practice for many years has been to leave those hernias which occur only intermittently and to operate on those which are down most of the time. I see the former ones at monthly intervals, and if there is no improvement after 3-4 months will then treat them surgically. I have never yet had one "strangulate on me."

My second point concerns the treatment of the possible contralateral hernia. Some years ago I tried enthusiastically in several cases to demonstrate the other sac with a curved director but was totally unsuccessful. Perhaps I was over-cautious. I now feel that searching for a sac which may not be there, and even if it is may not cause symptoms for many years, is really not justified. After all, this operation is not entirely without complications and one does not want to prolong it unnecessarily.

My third and last point is a plea to my fellow consultants. In experienced hands removing a hernial sac in a tiny baby is the simplest of operations, taking only 10-15 minutes. The inexperienced can get hopelessly lost, especially where "puppy fat" is excessive. My plea, therefore, is that never, never should this operation be delegated to a house surgeon or inexperienced registrar unless competent supervision is immediately available.—I am, etc.,

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Age Dependence of T-Cells

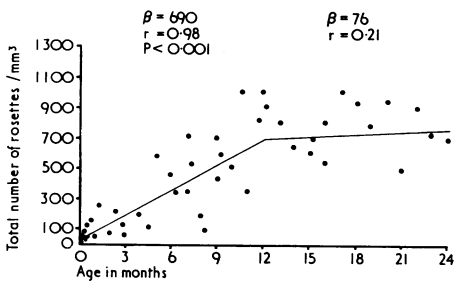
SIR,—In 1967 we reported[1] our finding that the mitotic activity of peripheral lymphocytes was lower in children under 2 years of age than in adults. Since then spontaneous rosette formation on incubation with sheep erythrocytes has been found to occur with lymphocytes derived from the thymus (T-cells).[2 3] We therefore used this technique in a further study of cellular immunity in infants.

The number of rosettes formed from at least 300 lymphocytes was counted in 1971 healthy children and adults. The results (see table) were expressed as the number of rosettes per 100 lymphocytes as well as the absolute number of rosettes/mm³ of peripheral blood. The number of rosettes increased gradually with age to reach about adult levels at 2 years if expressed per 100 lymphocytes or at 12 months if expressed as number/mm³ of peripheral blood. This discrepancy is due to the relative decrease of lymphocytes/mm³ after 12 months of age.

Number of Rosettes formed by T-Cells in 171 Healthy Children and Adults

Age	No. of Children	Rosettes/100 Lymphocytes		Rosettes/mm³ Peripheral Blood	
		Mean	Range	Mean	Range
1-30 days	10	1·9	1-4·5	88	13-273
–6 months	11	3·9	1-10	243	56-600
–12 "	15	9·9	4-20	618	123-1,044
–2 years	14	16·0	9-26	792	528-1,056
–4 "	16	20·0	10-48	988	491-1,452
–6 "	15	24·0	16-43	1,036	490-1,513
–9 "	21	29·0	14-62	953	468-1,520
–12 "	14	32·0	20-60	978	483-1,499
–16 "	15	32·0	19-59	1,110	624-1,526
>16 "	40	40·0	21-77	1,124	517-1,776

The increase of rosette formation with age during infancy is better illustrated by plotting the regression line which represents the age dependence of the total number of rosettes/mm³ of peripheral blood (fig.). The inclination factor (β = 690) is significantly high. It shows that the increase in the number of rosettes during the first year of life is about 50 per month, and $P<0.001$ makes this finding statistically highly significant. On the other hand, the increase during the second year of life is 10 times smaller, which is not significant.



These findings show that T-cells, as identified by their formation of rosettes with sheep erythrocytes, are few at birth and slowly and steadily increase in number each month until they reach about adult levels at 1 year of age.—We are, etc.,

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Saudi Arabian Medical School

SIR,—Our next-door neighbour in Riyadh has several daughters, three of whom are married. We have no social relations with this family other than an occasional visit to my wife made by the youngest of these three girls, who is perhaps now in her late teens. She has a small child, but she remains in full-time education as the corporate family looks after the child while she attends classes. I have never met the girl, as custom dictates that such visits are made when I am out of the house, but my wife inquired on one occasion of the girl's views on the difficulty of continuing to gain an education at the same time as being a wife and mother. The reply was that she considered herself fortunate; she had been born young enough to have routine access to education, and marriage was a minor handicap. Her elder sisters had been less lucky; at every step they had been in the first cohort of female education, so that when they became due for secondary school they had to await the completion of the buildings and curricula. Likewise their entry into university had been delayed for the same reasons.

I was reminded of this girl when I read the letter from Dr. Susan Barlow (17 August, p. 473). I have no connexion with the University of Riyadh, but I have lived in Saudi Arabia for the past six years. During that time I have seen enormous progress, but progress taken at a rate that is acceptable to a society which, until a few years ago, had maintained an unchanged pattern of life for centuries. That pattern still imposes many restrictions upon social behaviour, but it also still guarantees that the elderly and infirm are cared for by their family and that theft and violent crime remain at levels almost unknown elsewhere in the world.

There are so few areas today where British help is actively sought, and we should be proud that London University is held in such esteem that its guidance is behind the development of Riyadh's medical school. Of course there are female doctors in Saudi Arabia, and in due course there will be female lecturers too. It is not for us to stipulate when that should be, but Dr. Barlow might reflect on the spirit that drives girls such as our neighbour to overcome obstacles that have never blocked her own path.—I am, etc.,

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Women in Medicine

SIR,—In your leading article (7 September, p. 590) you state that "those [women] who opt for two careers cannot normally expect to reach the highest points in medicine. They must be satisfied with achieving less in medicine than they would have done had they foregone marriage and family and devoted themselves to medicine full time."

Sir, I am astonished. Is fatherhood less important than motherhood in family life? I do not think so, but we do not judge the quality of a father by how much time he spends physically at home; nor do we