dialysis may not be readily accomplished in some patients who have had recent abdominal surgery, multiple abdominal fistulas, or extreme adhesive peritonitis.

The technique described here provides a good compromise, in that those patients with acute renal failure may undergo early haemodialysis with a shunt. The vasculature which is enlarged by the fast flow of the shunt may then be converted to a fistula which can be used immediately for haemodialysis.

[1] Buselmeier, T J, *et al, Surgery*, 1973, **73,** 512.
[2] Buselmeier, T J, *et al, ASAIO*, 1973, **19,** 25.
[3] Quinton, W E, Dillard, D, and Scribner, B H, *ASAIO*, 1960, **6,** 104.
[4] Brescia, M J, *et al, New England Journal of Medicine*, 1966, **278,** 89.
[5] Kjellstrand, C M, *et al, Clinical Nephrology*, 1975, **4,** 37.

(*Accepted 18 May 1977*)

University of Minnesota Hospitals, Minneapolis, Minnesota 55455, USA

T J BUSELMEIER, MD, associate professor, medicine and surgery
J J RYNASIEWICZ, MD, fellow, department of surgery
R H HOWARD, MD, assistant professor, department of surgery
D E R SUTHERLAND, MD, assistant professor, department of surgery
T D DAVIN, MD, instructor, department of medicine
R L LYNCH, MD, instructor, department of medicine
E H HODSON, MD, fellow, department of medicine
R L SIMMONS, MD, professor of surgery and microbiology
J S NAJARIAN, MD, professor and chairman, department of surgery
C M KJELLSTRAND, MD, professor of medicine and surgery

Early antenatal diagnosis of small open spina bifida lesions

The measurement of α-fetoprotein (α-FP) in amniotic fluid is generally a reliable technique for the early antenatal diagnosis of neural tube defects.[1] It may, however, present difficulties if the fetus has only a small open spina bifida.[2] We have produced evidence that a careful examination of the morphology of the amniotic fluid cells, particularly those cells that adhere rapidly to glass or plastic surfaces in culture, can help in making such diagnoses.[3] We report here two cases that illustrate this point.

Case 1

A 24-year-old mother, para 0 + 1, with no history of affected children had three sequential α-FP values above the 95th percentile of the normal range (see table). Amniocentesis at 19 weeks gave an amniotic fluid α-FP concentration of 32·8 mg/l. The total cell count was 50×10^6/l; $25{\cdot}3 \times 10^6$/l were viable. There was no blood cell contamination. Nine per cent of the viable cells were adherent to glass after 20 hours' incubation and all these cells had an abnormal morphology.[3] The differential cell count showed that 49 % of adherent cells were large vacuolated cells, 14 % were long bipolar cells, and 9 % had multiple filamentous pseudopodia, while 28 % of the nuclei were contained in vacuolated giant multinucleated cells.

A second amniocentesis at 20 weeks gave an α-FP level of 24·2 mg/l. The total cell count was 51×10^6/l, of which $16{\cdot}4 \times 10^6$/l were viable. There were $1{\cdot}3 \times 10^9$ red blood cells/l, of which 20 % were fetal; 6·5 % of the viable cells were adherent, and while all these showed abnormal morphology (non-epithelioid), the long bipolar vacuolated cells were no longer present.

In view of the marginally raised amniotic fluid α-FP concentrations the pregnancy was allowed to go to term. The outcome was an infant with a severe lumbar myelocele, who died after four days.

Case 2

A 26-year-old mother, para 2 + 0, with no history of affected children had three sequential α-FP values above the 95th percentile of the normal range. Amniocentesis at 19 weeks, after ultrasound scan, gave an amniotic fluid α-FP value of 29·8 mg/l (see table). The total cell count was $45{\cdot}4 \times 10^6$/l, of which 18×10^6/l were viable. The liquor was contaminated with $2{\cdot}3 \times 10^9$ red blood cells/l, with 10 % of fetal origin. Cellular adherence was less than 3 %. Most of these cells were small with eccentrically placed nuclei, the remaining few nuclei being present in multinucleate cells which had no vacuolation or cytoplasmic inclusions. These cells are found only where the placenta has been traversed by the needle during amniocentesis.[3]

Serum α-FP, amniotic fluid α-FP, and proportions of rapidly adherent cells

Gestation (weeks)	Serum α-FP (μg/l)	Amniotic fluid α-FP (mg/l)	Rapidly adherent cells (%)
	Case 1		
16	158 (85)*		
18	118 (105)		
19	190 (115)	32·8 (21)†	9·0 (6)†
20		24·2 (17)	6·5 (6)
	Case 2		
17	115 (95)		
18	170 (105)		
19	125 (115)	29·8 (21)	2·1 (6)
20		22·0 (17)	<1 (6)

*95th percentile of normal range. †Upper limit of normal.

A second amniocentesis at 20 weeks gave an α-FP level of 22·0 mg/l. The total cell count was $58{\cdot}3 \times 10^6$/l, of which 20×10^6/l were viable. There were $2{\cdot}4 \times 10^9$ red blood cells/l, 10 % being fetal. Cellular adherence was less than 1 %. There were no cells with abnormal morphology, the very few cells that adhered to the coverslip being epithelioid. In view of the similarity of the α-FP concentrations to those in case 1, however, it was decided to terminate the pregnancy. The fetus had no neural tube defect or any other sign of external or internal abnormality.

Comment

In each of the two cases described here there were three serum α-FP values above the 95th percentile of the normal range and two amniotic fluid values above the defined upper limit of normal.[4] In particular the matched amniotic fluid values at 19 and 20 weeks were virtually indistinguishable. The small amount of fetal blood contamination in case 2, represented by about 2×10^9 fetal red blood cells/l, could have contributed little to the amniotic fluid α-FP concentration.

The results of cellular adherence studies were, however, more discriminating. In case 1 both amniocentesis samples yielded abnormal proportions of rapidly adhering cells with morphology characteristic of a neural tube defect.[3] In case 2 the first amniocentesis sample showed some adherent cells, but their morphology suggested that the placenta had been traversed rather than that the fetus was abnormal.[3] Examination of the second amniocentesis sample confirmed the suggestion that the fetus was normal, in that fewer than 1 % of adherent cells were seen and these had a normal (epithelioid) morphology.

[1] Brock, D J H, *British Medical Bulletin*, 1976, **32,** 16.
[2] Laurence, K M, *et al, Lancet*, 1976, **2,** 81.
[3] Gosden, C, and Brock, D J H, *Lancet*, 1977, **1,** 919.
[4] Brock, D J H, Scrimgeour, J B, and Nelson, M M, *Clinical Genetics*, 1975, **7,** 163.

(*Accepted 18 May 1977*)

Department of Human Genetics, University of Edinburgh, Edinburgh

D J H BROCK, BA, PHD, reader in human genetics

MRC Clinical and Population Cytogenetics Unit, Western General Hospital, Edinburgh

CHRISTINE GOSDEN, BSC, PHD, member of scientific staff

Induction of sex hormone binding globulin by phenytoin

In collecting material for a reference distribution of sex hormone binding globulin (SHBG) concentration, we identified a man who had an SHBG concentration considerably above that of the rest of the male population. After excluding thyroid or hepatic diseases, which are known to raise SHBG concentrations,[1] the only abnormality remaining which could have possibly caused the high SHBG concentration was epilepsy, for which he was receiving phenytoin. To check the possibility of phenytoin-induced increase in SHBG concentration we decided to study the SHBG concentration in women with epilepsy treated with phenytoin (Difhydan or Fenantoin).