most serious defficiency of the simulations, because it could greatly inhibit the processes tending to concentrate the gas into the centre.

Because of their limited resolution, the simulations provide information neither on how a central black hole might be formed, nor on how an already existing black hole might be supplied with gas. There have been various proposals for the latter process. Norman and Scoville[8] have suggested that gas drag or dynamical friction causes massive gas clouds to spiral down to a 10-parsec radius, where the gas forms a dense star cluster. Gas shed by stars in this cluster as they die is assumed to form and fuel the black hole. An alternative picture[9] is that a bar instability develops in the central gas disk once it becomes sufficiently massive. The bar loses angular momentum by gravitational torques, again carrying the gas down to a small volume, after which turbulent viscosity sets in, driving accretion onto the black hole.

As the rate of star formation decays with the consumption or dispersal of the gas and dust surrounding the central source, the external appearance of the central region will change: at first, the light from young stars, re-radiated by dust, will be seen; then there will be thermal emission from dust and non-thermal continuum from the active nucleus, both in the infrared; finally, with dust removed, optical emission from the active nucleus could become apparent if there is still sufficient accretion onto the black hole. Thus, one may have the evolutionary sequence of starburst → Seyfert → classical quasar. If all that is needed to trigger this sequence in a spiral galaxy is for it to have a moderately sized companion, many spiral galaxies may have gone through such a phase at some time. □

Cedric Lacey is at the Department of Astronomy, University of California, Berkeley, California 94720, USA.

1. Telesco, C.M. *A. Rev. Astron. Astrophys.* **26**, 343 (1988).
2. Hernquist, L. *Nature* **340**, 687–691 (1989).
3. Scalo, J.M. in *Starbursts and Galaxy Evolution* (eds Thuan, T.X. *et al.*) 445–465 (Frontieres, Paris, 1987).
4. Scoville, N. in *Galactic and Extragalactic Star Formation* (eds Pudritz, R.E. & Fich, M.) 541–549 (Kluwer, 1988).
5. Soifer, B.T., Houck, J.R. & Neugebauer, G. *A. Rev. Astron. Astrophys.* **25**, 187–230 (1987).
6. Simkin, S., Su, H. & Schwartz, M.P. *Astrophys. J.* **237**, 404–413 (1980).
7. Larson, R.B. in *Starbursts and Galaxy Evolution* (eds Thuan, T.X. *et al.*) 467–482 (Frontieres, Paris, 1987).
8. Norman, C. & Scoville, N. *Astrophys. J.* **332**, 124 (1988).
9. Shlosman, I. *et al. Nature* **338**, 45–47 (1989).

MARROW TRANSPLANTATION

Can cord blood be used?

David C. Linch and Leslie Brent

TRANSPLANTATION of HLA-matched bone marrow is currently the treatment of choice for selected patients with aplastic anaemia, leukaemia and inherited disorders of the bone marrow. The availability of HLA-identical sibling donors limits this option to roughly a quarter of otherwise suitable patients, so that panels of HLA-typed individuals willing to donate bone marrow have been set up in many countries. The panels need to be very large — a pool of 250,000 donors is estimated to have a 59 per cent chance of providing a compatible donor for any one patient, and with increasing pool size there is a disproportionally smaller increase in the number of successful matches[1]. In a recent report, Broxmeyer *et al.*[2] make the intriguing suggestion that the neonatal blood retained in the placenta contains sufficient blood stem cells to serve as a transplant inoculum. Neonatal blood could be collected, largely from the umbilical cord, and cryopreserved to build up a large bank, thus overcoming some of the difficulties associated with volunteer panels. The logistical problems associated with the creation and maintenance of such a bank are, however, quite formidable.

It was shown a quarter of a century ago that the blood of fetal mice contains large numbers of haemopoietic stem cells detectable in spleen colony-forming assays[3]. Likewise, second-trimester human fetal blood has a high incidence of haemopoietic progenitor cells estimated by *in vitro* colony-forming assays[4]; although the frequency is known to decline during the course of gestation, umbilical cord blood contains a higher number of progenitor cells per unit volume than adult blood, and it is roughly equivalent, in this respect, to adult bone marrow. This is amply confirmed by Broxmeyer *et al.* in over 100 cord blood samples. It is an article of faith that when there are large numbers of haemopoietic stem cells there will be large numbers of transplantable stem cells, but this is a reasonable assumption.

The novel data reported by Broxmeyer *et al.* concern the volume of cord blood that may be extracted: over 50 ml on average, but nearly 200 ml using an "optimized" method involving removal of blood from the maternal end of the transected cord whilst the placenta is still *in utero*, to which is sometimes added blood obtained by needle aspiration of engorged vessels on the fetal surface after expulsion of the placenta. This figure is surprisingly high — previous estimates[5] of fetal blood volume in the placental vessels immediately after birth have been of the order of 75–125 ml.

A 100-ml sample of cord blood can be expected to contain about 2×10^6 myeloid (GM-CFC) progenitor cells and 1×10^6 erythroid progenitor cells, which should be sufficient for reconstitution after allogeneic transplantation[6] provided that the stem-cell/progenitor-cell ratio is not appreciably less than in adult bone marrow. Only clinical studies can prove this point.

There are, however, two possible difficulties with the strategy proposed by Broxmeyer *et al.*, both relating to the possible development of graft-versus-host (GVH) disease. First, it is common practice when transplanting matched unrelated bone marrow to deplete it of mature T lymphocytes in order to minimize the GVH response; we are, however, told by Broxmeyer *et al.* that any fractionation of neonatal blood results in unacceptable losses of progenitor cells. This could be surmountable.

More worrying is the second possibility — that neonatal blood is contaminated by maternal cells. Some degree of leakage from fetus/neonate to the mother occurs in up to 50 per cent of births, usually during parturition, and occasionally clinically significant leakages occur from mother to fetus[6]. The incidence of relatively minor leakages from the maternal to the fetal circulation during parturition is not known, but it could be high. The magnitude of maternal blood contamination could be increased by the vigorous venesection from placental vessels before and after delivery of the placenta that seems to be required for the collection of optimal amounts of blood. Although adult blood possesses relatively few stem cells, the transfer of mature T lymphocytes incompletely matched with the histocompatibility antigens of the recipient could contribute unacceptably to GVH disease. Neonatal blood is virtually as immunocompetent as adult blood so far as the generation of cytotoxic T lymphocytes is concerned[7] and therefore has the potential for inducing GVH disease.

Finally, Broxmeyer *et al.* suggest that cryopreserved cord blood cells could be used for autologous bone marrow transplantation. The prospect of selected individuals (presumably the babies of the wealthy) having their cord blood stored as an insurance against the ravages of leukaemia or nuclear accident in adult life is more than a little disturbing. □

David C. Linch is in the Department of Haematology, University College and Middlesex School of Medicine, London, WC1E 6HX and Leslie Brent is in the Department of Immunology, St Mary's Hospital Medical School, London, W2 1PG, UK.

1. Bradley, B.A. *et al. Bone Marrow Transplantatn* **2** (suppl. 1), 79 (1987).
2. Broxmeyer, H.E. *et al. Proc. natn. Acad. Sci. U.S.A.* **86**, 3828–3832 (1989).
3. Barnes, D.W.H. & Loutit, J.F. *Lancet* **I**, 1395–1396 (1964).
4. Linch, D.C. *et al. Blood* **59**, 976–979 (1982).
5. Oski, F.A. & Naiman, J.L. in *Haematological Problems of the Newborn* 8-10 (W.B. Saunders, Philadelphia, 1982).
6. Gorin, N.C. *Clinics Haematol.* **15**, 19-48 (1986).
7. Rayfield, L.S. *et al. Clin. exp. Immunol.* **42**, 561 (1980).