CORRESPONDENCE

The method proposed by Stiff et al requires further assessment by other units before it is widely adopted. This is particularly important with peripheral blood stem cell transplantation where we are convinced that complete and sustained hematopoietic reconstitution following ablative therapy corresponds more closely to the CFU-GM dose than is the case of BM autotransplantation.1,2

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REFERENCES

RESPONSE

To the Editor:

We appreciate the interest of Haylock et al in our simplified bone marrow cryopreservation method.1 We regret that they have misinterpreted some of the data presented in our paper. First, as is clearly stated, we performed 72 transplants and listed the preparative regimens for each. The 12 patients who received both a second preparative regimen and marrow infusion had complete engraftment after the initial transplant. Second, while we had four deaths before engraftment, we feel it is inappropriate to consider the two deaths occurring before day 12 post-transplant which were due to progressive disease and pulmonary edema as “engraftment failures.” Our reported engraftment rate of 97% has not changed in over 150 transplants to date.

We agree that caution is required when considering long term storage of bone marrow cryopreserved in DMSO/HES at −80°C and clearly state this in our paper. We feel that it will remain difficult to verify the efficacy of long term storage at −80°C for our method as the overwhelming majority of autologous transplants performed here and elsewhere are completed within several months of the cryopreservation procedure. However, we recently thawed untransfused bone marrow from five patients, stored it in 300 mL aliquots at −80°C for 16, 22, 23, 24, and 27 months, and performed in vitro assays. The trypan blue viability was 83.5% ± 3.2% (SEM) and the CFU-GM recovery was 109.4% ± 12.5%. On the basis of both our previously reported small aliquot data2 and the above, it is appropriate to proceed with the clinical testing of our cryopreservation method using bone marrow stored for periods longer than 6 months.

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REFERENCES

CIRCULATING GLYCOSAMINOGLYCAN ANTICOAGULANTS ASSOCIATED WITH SURAMIN TREATMENT

To the Editor:

I read with interest the article by Horne et al1 in which they reported the development of a circulating anticoagulant in three cases treated with suramin. The authors hypothesized that the reason for the development of coagulopathy in their patients was the use of larger drug dosages and the presence of liver pathology in their patients. They stated that “if our hypothesis is correct it explains why coagulopathy has not been described previously as a consequence of suramin treatment despite widespread use of the drug for more than 60 years.” In fact, coagulopathy as a consequence of suramin therapy has been recognized for more than 60 years.1 Suramin, germanin, Bayer 205, and moranyl are brand names of the same drug.2 Evidence for anticoagulant action of this compound was summarized by A.J. Quick3 and he actually included germanin in the category of anticoagulants that inhibit coagulation through fibrinogen. He cited evidence indicating that germanin inhibits coagulation by combining with protein components, thrombin, and fibrinogen. Quick stated that the drug acts on plasma proteins, stabilizing them against flocculation by heat, acids, and by other precipitating agents. Germanin also shifts the isoelectric point of proteins by combining with plasma proteins.

Toluidin blue, although a smaller molecule, has a formula resembling portions of suramin. Like suramin, toluidine blue binds to many tissue and plasma proteins. It prolongs prothrombin time (PT) and activated partial thromboplastin time (APTT) and shortens the thrombin time (TT).4,5 The correction of abnormal coagulation tests
Circulating glycosaminoglycan anticoagulants associated with suramin treatment [letter]

AM Shojania