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MASSACHUSETTS MEDICAL SOCIETY

DEATHS

MALONE — Stephen Joseph Malone, M.D., of Pocasset, died on September 20. He was 94.

Dr. Malone graduated from Tufts College Medical School in 1927. He was a member of the American Medical Association and a 50-year member of the Massachusetts Medical Society.

OSTERHELD — Roger Golden Osterheld, M.D., of Palmer, died on September 27. He was 88.

Dr. Osterheld graduated from Boston University School of Medicine in 1924. He was a member of the American Psychiatric Association and a 50-year member of the Massachusetts Medical Society.

ROSENBLUM — Harry Aaron Rosenblum, M.D., formerly of Worcester, died on January 22. He was 79.

Dr. Rosenblum received his degree from the Kansas City University of Physicians and Surgeons in 1932. He was a member of the American Medical Association, the American Academy of General Practice, and the American Geriatrics Society.

CORRESPONDENCE

Letters to the Editor are considered for publication (subject to editing and abridgment), provided that they are submitted in duplicate, signed by all authors, typewritten in double spacing, and do not exceed 40 typewritten lines of manuscript text (excluding references). Submission of a letter constitutes permission for the Massachusetts Medical Society, its licensees, and its assignees to use it in the *Journal's* various editions (print, data base, and optical disk) in anthologies, revisions, and any other form or medium. Letters should not duplicate similar material being submitted or published elsewhere, and they should not contain abbreviations. Financial associations or other possible conflicts of interest should always be disclosed.

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LIVER TRANSPLANTATION IN A HEMOPHILIAC

To the Editor: A cure rather than a treatment has long been the goal of those caring for and those suffering from hemophilia. Encouraging results were obtained some years ago with the transplantation of normal livers into a dog with mild hemophilia^{1,2} and into four others with severe hemophilia.³ Two dogs given transplants survived more than 100 days and produced coagulation factor VIII in quantities sufficient to maintain normal levels.

The first "cure" in a human being appears to have been achieved, at least temporarily, in a 15-year-old boy with hemophilia and severe chronic active hepatitis, who received a liver from a 9-year-old "normal" donor on March 5, 1985. The patient was given the diagnosis of hemophilia A in early childhood and has been followed at the Hemophilia Center, Children's Memorial Hospital, Northwestern School of Medicine, Chicago, by Dr. John Paul Scott. In 1969 he had severe hepatitis B; positivity for hepatitis B surface antigen and antibody to hepatitis B e has persisted. In addition, therapy-related non A, non B hepatitis may have contributed to his liver disease. About four months before liver transplantation, a splenectomy was performed for chronic thrombocytopenia, which resolved postoperatively, but a residual pancreaticogastrocutaneous fistula remained. Liver transplantation and repair of the fistula were accomplished with less than the average blood loss. Before, during, and for 18 hours after liver transplantation, the patient's factor VIII level was maintained at 0.54 to 1.60 U per milliliter by intermittent infusions of heat-treated factor VIII concentrate. From 18 hours after operation to the time of this writing (April 17), his level has been sustained at 1.0 to 3.0 U per milliliter without exogenous treatment. The high postoperative levels of factor VIII are similar to those seen after transplantation in patients without hemophilia. Eight days after operation the patient underwent repair of a bile duct. There was no increased hemorrhage, and he required no factor VIII treatment.

Although in this case, at least temporarily, internal production of factor VIII has been achieved by the implanted liver, liver transplantation cannot be recommended as a "cure" for hemophilia. This drastic operation is justified only for end-stage liver disease because of the risks of lifelong immunosuppressive therapy, rejection, infection, and recurrence of disease. The duration of production of factor VIII is unknown. This case suggests that the antihemophilic activity (factor VIII:C) of the large factor VIII complex, synthesized in endothelium,^{4,5} is acquired in the liver.

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BLOOD DONATION BY PERSONS AT HIGH RISK OF AIDS

To the Editor: In March 1983, the U.S. Public Health Service published guidelines on the prevention of the acquired immunodeficiency syndrome (AIDS), including a recommendation that persons in groups at high risk of AIDS voluntarily refrain from donating blood.¹ The incubation period in adult cases of transfusion-associated AIDS ranges from two to five years.² Thus, it is not surprising that the number of cases of transfusion-associated AIDS has increased from 9 to 115 since the guidelines were published.¹⁻³ In January 1985, the Public Health Service recommended that all donated blood be tested for antibodies to human T-cell lymphotropic virus Type III (HTLV-III),⁴ the primary etiologic agent of AIDS.⁵⁻⁸ Since HTLV-III is occasionally isolated from antibody-negative sexual partners of patients with AIDS,⁹ the original guidelines must still be followed strictly.

Two groups of homosexual men who had been enrolled in private physicians' offices in Manhattan and Washington, D.C., during the spring of 1982^{8,10,11} were surveyed about their blood-donation practices during the previous two years. Eight (4 per cent) of the 199 homosexual men had donated a total of 18 units of blood between January 1982 and June 1984; seven men donated 11 units during 1982, three donated 4 units during 1983, and two donated 3 units during 1984. None of the eight blood donors had HTLV-III antibodies detected in serum samples obtained in 1982, and none of them has acquired AIDS. One subject had lymphadenopathy, and another had both lymphadenopathy and a low helper T-cell count at the initial examination. These two men were the only blood donors who had HTLV-III antibodies by 1984. They had each donated once, in August 1982 and May 1982, respectively.

Homosexual men are apparently continuing to donate blood, albeit at a reduced rate. Although the assay for HTLV-III antibodies is very sensitive and specific¹² and seropositivity is closely associated with lymphadenopathy and low helper T-cell counts both in homosexual men and in patients with hemophilia,^{8,13} lymphadenopathy does occasionally precede seroconversion,^{8,14} supporting the existence of an antibody-negative carrier state.⁹ The public should be educated about the potential harm of transfusing blood from possible carriers of HTLV-III, including not only persons from known risk groups but also their heterosexual partners.¹⁵ As the evidence mounts that sexual transmission of HTLV-III is continuing, consideration may need to be given to recommending that promiscuous heterosexual men and women, particularly prostitutes, and their contacts also refrain from donating blood.¹⁶

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ON VASCULAR NON-DISEASE OF THE FOOT IN DIABETES

To the Editor: We were interested in the paper by LoGerfo and Coffman on foot disease in diabetes (Dec. 20 issue).¹

In a prospective study, we recently measured skin oxygenation of the foot in diabetic patients with ulcerative foot lesions and retinal microangiopathy but without macrovascular occlusive disease inducing permanent ischemia.

Systolic blood pressure in the great toe was measured with a mercury strain gauge on the toe tip and an inflatable thin plastic cuff around the proximal phalanx (Plethysmograph SP2, Medimatic, Copenhagen).² Transcutaneous measurement of partial pressure of oxygen (PO₂) was performed with a modified Clark electrode, heating the skin at 44°C. All measurements were performed on the dorsum of the foot between the first and second metatarsals (Oxygen Monitor, Kontron, Roche).³ Arterial PO₂ was measured by puncture of the femoral artery. The PO₂ gradient between arterial blood and skin was calculated.

We selected 23 diabetic patients in whom systolic blood pressure in the great toe was above 50 mm Hg, in order to eliminate those with ischemia of the foot due to macrovascular occlusive disease. Thirteen of these patients had chronic ulcerative lesions of the feet. Eight nondiabetic subjects constituted the control group.

The PO₂ gradient between arterial blood and foot skin measured 24.6 ± 18.1 mm Hg (mean ± 1 S.D.) in diabetic patients with foot ulcerations; 20.4 ± 10.5 mm Hg in diabetic patients without skin ulcerations, and 20.1 ± 10.6 mm Hg in normal subjects. Differences were not statistically significant.

These data strongly suggest that diabetic microangiopathy does not provoke macrovascular occlusive disease and induce a severe skin hypoxia. This study is in good agreement with LoGerfo and Coffman's conclusion that diabetic neuropathy, rather than macrovascular disease, is the main cause of ulcerative lesions in patients without foot ischemia due to macrovascular occlusive disease.

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