during inspiration, thus increasing the efficiency of the functional hemidiaphragm. Finally, while an increase in the expiratory intercostal muscle tone will tend to decrease rib cage volume, by virtue of the linkage between the abdomen and rib cage postulated by Goldman and Mead,[10] it may be that the increased expiratory abdominal pressure could tend to maintain rib-cage volume.

We conclude that in patients who can generate an adequate ΔP_{di} during an obstructed inspiratory effort, a trial of CPAP may permit time for functional recovery without the need of either IPPB ventilation or surgical diaphragmatic plication. Since there is no published information available to answer the question of whether surgical plication in the infant or child with a temporary hemidiaphragmatic paralysis may have long-term adverse effects on the normal growth and development of the lung, it appears prudent to refrain from surgical intervention until permanent injury is confirmed. With convincing evidence of phrenic-nerve injury causing permanent hemidiaphragmatic paralysis, surgical plication is indicated when it has also been demonstrated that the infant can do well with CPAP alone, *i.e.*, the functional hemidiaphragm will allow adequate ventilation when the paralyzed hemidiaphragm is stabilized.

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References

1. Schifrin N: Unilateral paralysis of the diaphragm in the newborn infant due to phrenic nerve injury, with and without associated brachial palsy. Pediatrics 9:69–76, 1952
2. Sethi G, Reed WA: Diaphragmatic malfunction in neonates and infants. J Thorac Cardiovasc Surg 62:138–143, 1971
3. Mearns AJ: Iatrogenic injury to the phrenic nerve in infants and young children. Br J Surg 64:558–560, 1977
4. Gould L, Kaplan S, McElhinney AJ, et al: A method for the production of hemidiaphragmatic paralysis. Am Rev Resp Dis 96:812–814, 1967
5. Loh L, Goldman M, Newson Davis J: The assessment of diaphragm function. Medicine 56:165–169, 1977
6. Bates DV, Macklem PT, Christie RV: Respiratory Function in Disease. Philadelphia, W. B. Saunders, 1971, p 29
7. West JB: Respiratory Physiology—The Essentials. Baltimore, Williams and Wilkins, 1974, pp 89–115
8. Hagan R, Bryan AC, Bryan MA, et al: Neonatal chest wall afferents and regulation of respiration. J Appl Physiol 42:362–367, 1977
9. Lai-Fook SJ, Hyatt RE, Rodarte JR: Effect of parenchymal shear modulus and lung volume on bronchial pressure—diameter behavior. J Appl Physiol 44:859–868, 1978
10. Goldman MD, Mead J: Mechanical interactions between the diaphragm and rib cage. J Appl Physiol 35:197–204, 1973

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Serum Potassium Levels Following Transfusion of Frozen Erythrocytes

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The role of frozen, deglycerolized erythrocytes is assuming greater importance in the management of patients receiving massive transfusions.[1,2] Advantages cited by the proponents of frozen erythrocytes over blood with added citrate–phosphate–dextrose (CPD) are: longer shelf life,[3] prevention of alloimunization,[4] decreased risk of transfusion hepatitis,[5] and improved viability of function of erythrocytes.[6] Hyperkalemia, which might occur with transfusion of stored CPD blood[7] is noticeably absent with transfusion of frozen erythrocytes. Although it has been demonstrated that intracellular potassium levels in frozen, thawed, deglycerolized, and washed erythrocytes tend to decrease,[8] serum potassium values in patients receiving frozen erythrocytes have not previously been reported. This investigation was undertaken to study the effect of transfusion of frozen erythrocytes on serum potassium values in patients who receive such transfusions.

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Materials and Methods

Twenty-eight adult patients, 11 women and 17 men, whose ages ranged from 48 to 61 (mean 54) years,

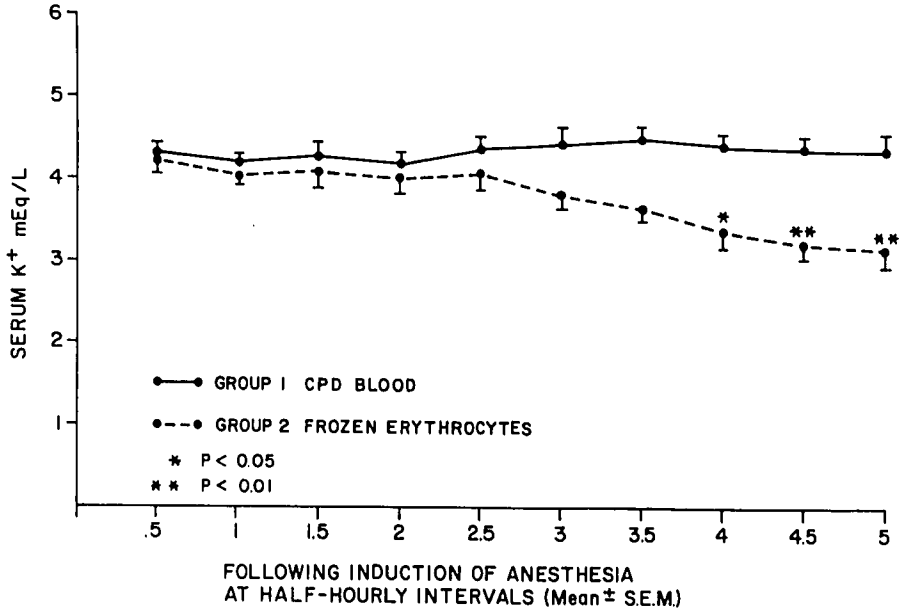

FIG. 1. Serum potassium changes following transfusion of CPD blood and frozen erythrocytes.

were studied. All the patients underwent radical resections of the gastrointestinal tract for cancer treatment, and they were free of respiratory, cardiovascular, hepatic and renal disease. The patients were not taking any drugs on a regular basis. The patients were randomly divided into two groups. Patients in Group I (n = 15) received CPD-preserved whole blood eight to 13 days old, and those in Group II (n = 13) received four-to-eight-month-old frozen, thawed, deglycerolized, and washed erythrocytes that had been frozen within 24 hours after harvesting from donors. Preoperative serum electrolyte, hemoglobin, blood glucose, and arterial blood-gas values were within normal limits in all patients in both groups. Anesthesia was induced with thiopental, 275 mg, iv, and tracheal intubation was facilitated with pancuronium, 8 mg. Anesthesia was maintained with nitrous oxide, 60 per cent, in oxygen, and fentanyl, given intravenously as needed. Ventilation was mechanically controlled to maintain arterial blood P_{CO_2} values between 35 and 40 torr. Rectal temperature was continuously monitored and maintained between 36.5 and 37.5 C.

The patients in both groups received intravenous fluid therapy at a rate of 10 ml/kg/h for the first two hours and 6 ml/kg/h thereafter. The first two liters consisted of dextrose, 5 per cent, in lactated Ringer's solution only. All surgical procedures lasted at least five hours, with a mean duration of 6.4 hours. Every 30 min following induction of anesthesia, arterial blood samples were analyzed for *p*H, P_{O_2}, P_{CO_2}, serum sodium and potassium, glucose, hemoglobin, and hematocrit. Arterial blood *p*H, P_{O_2}, and P_{CO_2} values were measured with a Corning 175 blood-gas machine; serum sodium and potassium with an Orion SS30, and blood glucose with a Yellow Springs 23A. When hematocrit decreased to 28 per cent, blood that had been warmed to 37 C by use of a blood warmer was transfused into the patient. All patients needed blood transfusions during the second to fourth hour of anesthesia, when blood loss was maximum. By four hours after the induction of anesthesia, blood loss was minimal, and blood transfusions were not given thereafter.

For comparison purposes, the values for arterial blood samples drawn 30 min following induction of anesthesia were compared with those for blood drawn four and a half hours after induction. The data were subjected to statistical analysis by use of the paired *t* test for within-group comparisons and non-paired *t* test and chi-square test comparisons between the two groups. Values are expressed as mean ± standard error of the mean (±SEM).

RESULTS

Patients in Group I received 3.8 (±0.2) units of stored CPD-preserved blood, while patients in Group II received 3.7 (±0.2) units of frozen erythrocytes. The amounts of blood transfused into patients in the two groups were not significantly different. Prior to transfusion, there was no significant difference between blood glucose, arterial blood *p*H, P_{O_2}, hemoglobin, and hematocrit, and serum sodium and potassium values in Group I and Group II. There was no significant change in blood glucose, arterial blood *p*H, P_{O_2}, P_{CO_2}, hemoglobin, or hematocrit, or serum sodium, before or after transfusion of CPD blood or frozen erythrocytes. The only significant

change was in serum potassium. In the patients in Group I who received CPD-preserved blood, the post-transfusion serum potassium level was not significantly different from the pretransfusion level, while in patients of Group II, who received frozen, thawed erythrocytes, serum potassium levels were significantly lower after transfusion. In four of 13 patients in Group II, serum potassium values decreased to less than 3 mEq/l. Two patients had serum potassium values of 2.4 and 2.6 mEq/l, respectively, and ventricular premature beats developed in these patients. Following the intravenous administration of potassium chloride, 30 mEq/l, in 250 ml of dextrose, 5 per cent, serum potassium values returned to normal, and the dysrrhythmias disappeared. Thus, four of 13 patients in Group II (31 per cent) needed supplemental potassium therapy, while no patient in Group I needed potassium.

Discussion

Storage of CPD-preserved blood at 4 C causes egression of potassium ions from the erythrocytes into plasma, reaching abnormal levels within the first seven days of storage.[9] Few of these potassium ions re-enter the erythrocytes on rewarming to body temperature.[10] Following transfusion of this stored blood, excess extracellular potassium ions may be excreted by the kidney, re-enter the original erythrocytes, or pass into the erythrocytes, muscle cells or other tissues of the recipient, or remain in the plasma of the recipient.[11] Thus, transfusion with CPD-preserved blood might produce transient hyperkalemia in the recipient prior to redistribution of the egressed potassium ions.[7]

However, it has been shown that frozen, thawed, deglycerolized, washed and resuspended erythrocytes have decreased levels of intracellular potassium.[8] The potassium content of erythrocytes is about 7 mEq/10^{12} erythrocytes. In frozen, thawed, deglycerolized and washed erythrocytes, however, potassium values have been as low as 4 mEq/10^{12} erythrocytes.[8] When these erythrocytes are transfused into a patient, potassium ions from the recipient's plasma could move into these erythrocytes, which are low in intracellular potassium, thereby decreasing the serum potassium levels of the recipient. This investigation demonstrates the occurrence of hypokalemia when frozen, thawed erythrocytes are transfused into patients with normal serum potassium levels. Other factors known to influence serum potassium levels, including alkalemia, hypocarbia, and blood glucose changes, did not play a role in the effects seen in this investigation.

The decrease in serum potassium following the use of frozen erythrocytes could be more important in patients needing massive blood transfusions to maintain blood volume and oxygen transport, or in premature infants and neonates, where exchange transfusion is performed using frozen erythrocytes.

In conclusion, when frozen, thawed, deglycerolized, washed and resuspended erythrocytes are transfused, serum potassium levels of the recipient should be monitored, and if hypokalemia occurs, potassium supplementation therapy should be instituted to prevent the occurrence of dysrhythmias.

References

1. Moss GS, Valeri GR, Brodine CE: Clinical experience with the use of frozen blood in combat casualties. N Engl J Med 278:747–752, 1968
2. Bryant LR, Wallace ME: Experience with frozen erythrocytes in a private hospital. Transfusion 14:481–485, 1974
3. Blagdon J: The long term storage of blood by freezing. Br J Hosp Med Equip Supplies 12:23–34, 1972
4. Meryman HT, Hornblower M: Red cell recovery and leukocyte depletion following washing of frozen, thawed red cells. Transfusion 13:388–393, 1973
5. Tullis JL, Hinman J, Sproul MT, et al: Incidence of post transfusion hepatitis in previously frozen blood. JAMA 214:719–723, 1970
6. Valeri CR, Szymanski I, Runck AH: Therapeutic effectiveness of homologous erythrocyte transfusion following frozen storage at −80°C for up to seven years. Transfusion 10: 102–112, 1973
7. Miller G, McCoord AB, Joos AH, et al: Studies of serum electrolyte changes during exchange transfusion. Pediatrics 13:142–417, 1954
8. Valeri CR: Blood banking and the use of frozen blood products. Cleveland CRC Press 1976, pp 177 and 192
9. Crawford H, Mollison PL: Reversal of electrolyte changes in stored red cells after transfusion. J Physiol 129:639–647, 1955
10. Eurenius AB, Smith RM: The effect of warming on the serum potassium content of stored blood. Anesthesiology 38:482–484, 1973
11. Valeri RC, Hirsch N: Restoration of *in vivo* erythrocyte adenine triphosphate, 2,3,diphosphoglycerate, potassium ion and sodium ion concentrations following the transfusion of acid citrate dextrose stored human red blood cells. J Lab Clin Med 73:722–733, 1969