Literature Briefs

C. Philip Larson, Jr., M.D., Editor

Briefs were submitted by Drs. C. M. Ballinger, N. Bergman, R. B. Boettner, A. Boutros, D. R. Buechel, H. F. Cascorbi, R. B. Clark, D. Duncalf, W. H. Mannheimer, F. C. McPartland, D. H. Morrow, R. C. Morton, J. W. Pender, A. D. Randall, L. J. Saidman, and A. D. Sessler. Briefs appearing elsewhere in this issue are part of this column.

Circulation

AUTOLOGOUS BLOOD TRANSFUSION Sixteen patients undergoing thoracic surgery received a total of 26 units of autologous banked blood. Single units were drawn in routine fashion one week prior to surgery. When the need for two units of blood was anticipated, a double phlebotomy was performed using a double plasma pheresis pack, followed by reinfusion of a previously drawn autologous unit. When three units were requested for anticipated surgery, two units were obtained as before and exchanged on day 14 for units using a double plasmapheresis pack. On day 16, a single unit was obtained in the routine fashion. The net loss of blood to the donors at a single phelebotomy never exceeded one unit of blood; however, one, two, or three units of autologous blood were obtained by appropriate exchange for fresh blood. None of the units had been stored in excess of ten days when finally used at the time of surgery. Transfusion of autologous blood avoids the hazards of transmission of serum hepatitis and isoimmunization. It may be the only source of rare blood types or blood for members of religious sects which prohibit the use of homologous blood transfusion. (*Ascari, W. Q., Jolly, P. C., and Thomas, P. A.: Autologous Blood Transfusion in Pulmonary Surgery, Transfusion 8: 111 (March) 1968.*)

CEREBRAL METABOLISM The effect of respiratory alkalosis on cerebral carbohydrate metabolism was studied during passive hyperventilation of lightly anesthetized human volunteers. Reduction of Pa_{CO_2} to 19 mm Hg produced several alterations in cerebral carbohydrate metabolism, including a lesser proportion of cerebral glucose consumption which could be accounted for by oxygen utilization and increased conversion of glucose to lactate. Lowering of Pa_{CO_2} to 10 mm Hg caused a decrease in oxygen uptake and an increase in glucose uptake by the brain, as well as increased cerebral excess lactate. By inducing metabolic alkalosis superimposed on a constant level of respiratory alkalosis, it was demonstrated that the observed decrease in cerebral oxygen uptake was due both to diminished cerebral circulation and to the effect of alkalosis on the position of the oxygen dissociation curve of hemoglobin (Bohr effect). It was concluded that hypocarbia can alter brain metabolic pathways. (*Alexander, S. C., and others: Cerebral Carbohydrate Metabolism of Man During Respiratory and Metabolic Alkalosis, J. Appl. Physiol. 24: 66 (Jan.) 1968.*)

SHOCK In shock, the stress reaction is initiated by the pituitary adrenal axis to stimulate glycogenolysis for increased ATP production through the glycolytic, hexomonophosphate, and citric acid cycles. Concomitantly, amino acids and fatty acids are mobilized from muscle protein and fat tissue to enter the tricarboxylic acid cycle for additional ATP production. Continued loss of volume blocks the pyruvate–acetyl coenzyme A junction, anoxia disrupts the oxidative enzymatic systems for proper shuttling into the citric acid cycle, and lactic acid is increased. Acidosis is compounded by incomplete breakdown of fatty acids yielding acetone bodies, protein catabolism yielding aminoaciduria, and hyperphosphatemia. There is an increasing demand for energy, but less energy is produced. Ultimately, metabolic derangement of the vital organ system occurs and death ensues. (*Schumer, W., and Kukral, J. C.: Metabolism of Shock, Surgery 63: 630 (April) 1968.*)