Postoperative Management and Course. The separated twins were kept on two thermostatically controlled incubator cots, fitted with Kreiselman's infant resuscitators in an isolated air-conditioned nursery. Hydrocortisone (10 mg) was administered to each twin at four-hour intervals during the first 24 hours. A total of 150 mg of hydrocortisone was injected into the twins during operation and in the following 24 hours. The rationale of administering corticosteroids to infants was based on the assumption that one of the twins might have been dominant in providing cortisone for both; the other twin might then have had adrenocortical deficiency after separation. Furthermore, the anti-inflammatory action of steroids would help prevent laryngeal reaction following tracheal intubation and would help during stress. During the postoperative period the respiration, blood pressure, color and general condition of the twins were carefully watched. Oxygen (3 l/min) was administered through a plastic oxygen hood for an hour at two-hourly intervals. The oxygen in the hood was kept at around 40 per cent. Urinary output, electrolyte and fluid balance were attended to.

During the first 24 hours of the postoperative period, Twin II had a slight elevation in blood pressure to about 140–170 mm Hg, systolic. Both twins also had elevated blood urea, considered to be due to the pre-existing renal damage consequent to the renal vein thrombosis from which the twins had recovered.

The separated infants were kept for two days in a slightly head-up position to prevent collapse of the upper lobes of the lungs. Postoperative roentgenographic studies of the chest showed no areas of unaerated lung. The postoperative period gave no cause for anxiety. Both children are hale and hearty and are growing satisfactorily.

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Obstetrics and Pediatrics

FETAL ACIDOSIS AND HEART RATE Fetal base deficit values were correlated with fetal heart rate patterns in 139 fetuses of mothers in active labor. Normal fetal heart rates and early deceleration "type 1 dip" (thought to be due to head compression) were not associated with any appreciable abnormality in fetal *p*H. Mild and moderate variable deceleration (thought to be due to intermittent cord compression) and mild late deceleration "type 2 dip" (due to uteroplacental insufficiency) were associated with increases in fetal base deficit. Severe variable, moderate, and severe late deceleration patterns were associated with more pronounced increases in fetal base deficits. These fetal acid–base findings support the etiologic and prognostic significance assigned by the author to different fetal heart rate patterns. High correlations were seen in babies with low Apgar scores where ominous fetal heart rate patterns and fetal acidosis were both present. (*Hon, E. H., and Khazin, A. F.: Observations on Fetal Heart Rate and Fetal Biochemistry. I. Base Deficit, Amer. J. Obstet. Gynec. 105: 721 (Nov.) 1969.*)