Severe Anemia in Childhood Presenting as Transient Ischemic Attacks

RICHARD S. K. YOUNG, M.D., D. EUGENE RANNELS, PH.D.,* ANN HILMO, M.D., JAMES M. GERSON, M.D., AND DAVID GOODRICH, M.D.†

TRANSIENT ISCHEMIC ATTACKS (TIAs) may result from interruptions in the supply of oxygen to the brain.1 We recently cared for a child whose first manifestation of profound anemia was recurrent TIAs.

Case Report

This 2 year old girl was well until she awoke from a nap with left sided weakness. She was alert and aware of her deficit. Speech was not impaired. She was seen that afternoon by her pediatrician, but the hemiparesis (which lasted 15 minutes) had remitted by the time of examination. On the next day, she had a second 15 minute episode of left hemiparesis again following a nap. On the third day, she had another episode of left sided weakness and was hospitalized. Laboratory tests revealed severe normochromic, normocytic anemia (hemoglobin, 4 grams/dl; MCV, 82 FL; MCH, 29.7 PG; MCHC, 35.6%; reticulocyte count, 0.1%) while the white blood cell count, platelet count, and plasma glucose concentration were normal. She was transferred to The Milton S. Hershey Medical Center. The physical examination revealed an alert, but pale child; neurological examination was entirely normal. A bone marrow aspirate was hypocellular with virtual absence of red cell precursors. A diagnosis of transient erythroblastopenia of childhood was made on the basis of her age, normal hemoglobin F, and absence of erythrocyte i antigen.2 The patient received packed red blood cell transfusions and had no subsequent attacks of weakness. Right carotid arteriogram, EEG and CT brain scan were normal. She was treated with prednisone for two weeks. A repeat bone marrow aspirate showed increased erythroid precursors. Her hematocrit returned to normal levels within one month. Neurological examination 6 months later was normal.

Discussion

Anemic hypoxia is of particular interest since Brierley has suggested that it never causes brain damage.3 Moreover, we could not find a previous detailed report associating profound anemia with transient ischemic attacks or cerebral infarction.

It appears likely that this child's transient hemipareses were due to anemic hypoxia. Hypothetical calculations show that her brain oxygen requirement of approximately 50 mlO2/min (equation 1, fig. 1) was not being met (equations 2 and 3, fig. 1). These calculations assume that cerebral blood flow was near maximum, that oxygen extraction by the tissues was complete, that cardiac output was not compromised, and that PaO2 and hemoglobin saturation were normal. Any reduction of these parameters would have further compromised delivery of oxygen. Since the brain of a child less than four years of age may use up to 50% of the oxygen breathed, a four-fold reduction in hemoglobin levels could exhaust the margin of safety inherent in the oxygen transport system. It is possible that all three attacks began while she was asleep, because of the blunting of ventilatory drive and the increase in brain oxygen consumption which occurs during sleep.9 It remains uncertain how widespread metabolic defi-

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1. Brain Oxygen Requirement
   \[ \text{Cerebral metabolic rate} \times \text{brain weight (g)} \]

2. Arterial Oxygen Content
   \[ \text{Oxygen content (mlO2/ml blood)} \]

3. Total Oxygen Delivered to Brain
   \[ \text{Cerebral Blood Flow (ml/min/100g brain)} \times \text{Brain Weight (g)} \]

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From the Departments of Pediatrics, *Physiology, and †Anesthesiology, The Milton S. Hershey Medical Center, The Pennsylvania State University, Hershey, Pennsylvania 17033.

Address correspondence to: Richard S. K. Young, M.D., Department of Pediatrics, Division of Neurology, The Milton S. Hershey Medical Center, The Pennsylvania State University, Hershey, Pennsylvania 17033.

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ciency produces focal neurological deficits. However, hemiplegia may result from an insufficient supply of glucose as well as oxygen.

References

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Continuous Measurement of Regional Cerebral Blood Flow Using Krypton-81m

AKIRA OGAWA, M.D., YOSHIHARU SAKURAI, M.D., AND JIRO SUZUKI, M.D.

SUMMARY We have developed a new method for taking continuous measurements of rCBF by means of continuous infusion of $^{81m}$Kr. Using this method, it is possible to follow the sequential changes in blood flow of various brain regions continuously. The method makes it possible to observe the sequential changes in CBF following drug administration, motor activation and various kinds of stimulation, and, furthermore, to observe the CO$_2$ reactivity and autoregulation of cerebral vessels. It will undoubtedly prove useful in the investigation of various pathological states.

Method
Kr-81m solution was produced by pathing 5% glucose solution through a 10mCi Rb-Kr generator (Japan Mediphysics Co). The solution, with constant concentration, was infused continuously to the internal carotid artery (ICA) at a constant rate of 3ml/min through a catheter inserted into the ICA using a perfusion pump (Truth A-II). Under these conditions, lateral image of the head and neck were obtained by a $\gamma$-camera (Toshiba GCA 301). The obtained images were fed online into a data processor (Toshiba GNS 80A). Sampling time was 30 seconds a frame and 60 frames of the sequential data obtained every 30 seconds were collected in 30 minutes.

Theoretical Consideration
In the intracarotid Kr-81m infusion method, regional radioactivity of the brain can be obtained from the following equation:

$$Cieq \propto fi(\mu + fi/vi \lambda i)$$

where, $Cieq$ is the regional radioactivity obtained from the surface of the skull, $fi$ is the regional CBF, $\mu$ is the decay constant, $vi$ is the volume of brain tissue perfused by radioisotope, $\lambda i$ is the partition coefficient between blood and tissue.

Since the half-life of $^{81m}$Kr is as short as 13 seconds, the regional radioactivity ($Cieq$) is roughly proportional to rCBF ($fi$). Consequently, the equilibrium image of the brain infused with $^{81m}$Kr is said to express this regional distribution of CBF. However, in the case of
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R S Young, D E Rannels, A Hilmo, J M Gerson and D Goodrich

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