1980
YEAR BOOK OF
PEDIATRICS

OSKI / STOCKMAN

The YEAR BOOK of

Pediatrics

1980

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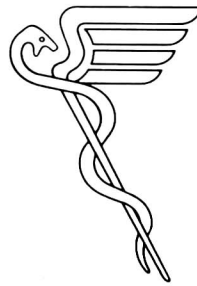

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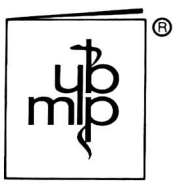

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The Newborn

Hypercapnia at Birth: Possible Role in Pathogenesis of Intraventricular Hemorrhage. To test the hypothesis that birth asphyxia has a role in the etiology of intraventricular hemorrhage, John D. Kenny, Joseph A. Garcia-Prats, James L. Hilliard, Anthony J. S. Corbet and Arnold J. Rudolph[1] (Baylor Univ., Houston) collected blood from the umbilical artery at birth in 28 infants born after 26–29 weeks' gestation. The samples were analyzed for hydrogen ion concentration, P_{CO_2}, standard bicarbonate level and lactic acid level. The infants were followed until a diagnosis of intraventricular hemorrhage could be made or excluded clinically or by autopsy.

Of the 28 infants, 14 had an intraventricular hemorrhage 1–184 hours (mean, 48) after birth and died 3–240 hours (mean, 98) after birth. The 14 infants with hemorrhage and the 14 controls were similar with respect to birth weight, gestational age and 5-minute Apgar score, but the 1-minute Apgar scores were 2.7 in infants with hemorrhage and 4.4 in controls ($P < 0.05$). The sex ratio and incidences of breech delivery, cesarean section, hyaline membrane disease and need for intermittent positive pressure ventilation were not significantly different in the two groups, but death occurred in all 14 infants with intraventricular hemorrhage and in only 2 controls ($P < 0.001$). The infants with intraventricular hemorrhage had higher P_{CO_2} values at birth than the controls ($P < 0.025$), but there were no significant differences in the other acid-base parameters.

Although the increase in P_{CO_2} in infants with intraventricular hemorrhage was relatively small, the increase during labor may have been comparatively large. Hypercapnia may be important in the genesis of intraventricular hemorrhage, possibly through increasing cerebral blood flow. **(1)**

▶ [I again asked Dr. Joseph Volpe, Professor of Pediatrics and Neurology, Washington University School of Medicine, to comment on this topic. For his previous comments see the 1979 YEAR BOOK, pages 41–42. This year, Dr. Volpe writes:

"Intracranial hemorrhage is now the most important neurologic problem encountered in the newborn. Of the four major varieties of neonatal intracranial hemorrhage, periventricular-intraventricular hemorrhage (PV-IVH), is the most common and clinically most important. Subdural hemorrhage, a traumatic lesion, has been rendered uncommon by improved obstetric care. Primary subarachnoid hemorrhage, though relatively common, is only infrequently of clinical importance. Intracerebellar hemorrhage affects the very small premature infant and is of uncertain clinical significance.

"The primary goal in *management* of PV-IVH is prevention. To accomplish this goal will require understanding of the pathogenesis. Recent studies of cerebral blood flow and the regulation thereof in the human newborn have provided major new insight into the pathogenesis of this lesion (see Volpe, J.: J. Pediatr. 94:170, 1979).

"The *pathogenesis* of PV-IVH must explain the strong association of the lesion with prematurity, an asphyxial event(s) and the periventricular locus of the lesion. The strong

(1) Pediatrics 62:465–467, October, 1978.

association with prematurity relates to three major factors. First, the subependymal germinal matrix is still present in the human premature infant and is not exhausted until term; this region provides very poor support for the many small vessels found within it. Second, the periventricular vessels of the premature infant are thin walled and, presumably, somewhat fragile. Third, there is an impairment of vascular autoregulation (see below). The relation to asphyxial event(s) is threefold. First, with asphyxia there is often increased intravascular pressure in brain, presumably secondary to hypoxic myocardial failure. Second, with hypoxic-ischemic insult a degree of endothelial injury may occur. Third, the impairment of vascular autoregulation is accentuated (see below). The characteristic periventricular locus of the lesion also relates to three major factors. First, this is the site of the subependymal germinal matrix. Second, in the periventricular location a peculiar hemodynamic situation exists which leads to increased intravascular pressure. Thus, the terminal, thalamostriate and choroidal veins, which drain anteriorly, converge in the periventricular region at the level of the foramen of Monro and the head of the caudate nucleus (the sites of origin of PV-IVH in 75–90% of cases) to form the internal cerebral vein, which then courses directly posteriorly. Thus, a peculiar U-turn in the direction of cerebral blood flow occurs, i.e., a good circumstance for venous stasis and, rarely, even thrombosis. Third, there is an unusual amount of fibrinolytic activity in the periventricular region of the human premature infant, and this may encourage bleeding.

"A critical role for *cerebral blood flow and the regulation thereof* in the genesis of PV-IVH is suggested by recent studies of normal and modestly asphyxiated infants by Lou et al. (ibid., p. 118). Utilizing the xenon clearance technique to measure cerebral blood flow, these workers demonstrated a linear relationship between cerebral blood flow and systolic blood pressure. The failure of cerebral blood flow to remain constant despite differences in blood pressure indicate that cerebral blood flow is not autoregulated in the human newborn. Thus, cerebral blood flow is "pressure-passive." When blood pressure rises, cerebral blood flow rises and exposes the periventricular capillaries to a burst of pressure that could result in rupture. This notion is supported by the demonstration that PV-IVH emanates from capillaries and not from veins, as previously thought (Hambleton and Wiggelsworth: Arch. Dis. Child. 51:651, 1976). Moreover, experimental observations demonstrate that asphyxia further impairs vascular autoregulation (Lou et al.: Acta Paediatr. Scand., in press, 1979) and that asphyxia most effectively results in PV-IVH in fetal sheep when *both* arterial blood pressure and venous pressure are raised (Reynolds et al.: Pediatr. Res. 11:1024, 1977). These data particularly direct our attention to the *arterial* side of the periventricular vascular network to understand the pathogenesis of PV-IVH. The most potent effector of cerebral blood flow in man is the arteriolar, perivascular hydrogen ion concentration, and arterial P_{CO_2} is the most effective means of raising that concentration. Indeed, with perinatal sheep, an approximately 7% increase in cerebral flow occurs for every mm of mercury increase in arterial P_{CO_2} (Purves and James: Circ. Res. 25:651, 1979). Thus the importance of the hypercapnia observed by Kenny et al. (see accompanying article) is apparent. Further work clearly is needed on that specific issue. Moreover, an incriminating posture might now be taken for the injudicious use of volume expanders, hypertonic solutions, pressor agents, etc. It is clear that *abrupt* changes in arterial blood pressure could have very dangerous consequences in the human premature infant, especially in the clinical setting of even modest asphyxia.

"This discussion should not be left without looking at the other side of the coin. Thus, the impairment of vascular autoregulation in the human premature infant means also that cerebral blood flow will fall as blood pressure falls. Consequently, the infant is very vulnerable to hypotension, with ischemic injury to brain (i.e., parasagittal cerebral injury in the full-term and periventricular leukomalacia in the premature) the unfortunate result. The neonatal physician is sitting on a rather sharp and fine edge—too little perfusion may cause ischemic brain injury and too much, PV-IVH. Moreover, assessing cerebral perfusion is not simply a matter of measuring arterial blood pressure. Nevertheless, this determination is obviously of great value, and *continuous* monitoring of blood pressure clearly is of major importance in the management of the sick newborn. Until we are able to measure cerebral blood flow directly, safely and conveniently, we must be particularly compulsive about monitoring arterial blood pressure."] ◀

Inappropriate Antidiuretic Hormone Secretion in Premature Infants with Cerebral Injury. The syndrome of inappropriate antidiuretic hormone secretion (SIADH) has been described in only 1 pre-

mature neonate. Fergus M. B. Moylan, John T. Herrin, Kalpathy Krishnamoorthy, I. David Todres and Daniel C. Shannon[2] (Massachusetts Genl. Hosp.) saw 11 premature infants with the syndrome among 854 admissions over $3^1/_2$ years to a newborn intensive care unit. The syndrome may be much more common among premature neonates than the scarcity of reports suggests.

Average age at onset of SIADH was 10 days (range, 5–16) and average duration was 6.4 days (range, 3–10). Mean gestation was 30.7 weeks (range 27–35) and mean birth weight was 1.5 kg (range, 0.9–2.2). All patients had apnea before onset of SIADH and 10 had signs of asphyxiation at birth. An acute fall of 10% or more in arterial blood hematocrit occurred in 8 infants, but this was corrected before SIADH developed. Seven patients had intracranial hemorrhage and 5 had one or more coagulation abnormalities.

Therapy included restriction of oral and parenteral fluids to 30–50 ml/kg per day, increased sodium intake from 3 to 4 to 6–8 mEq/kg per day and maintenance of potassium intake at 1 to 2 mEq/kg per day. Fluid restriction was continued until serum sodium concentration and osmolality returned to normal and then was increased slowly until intake of calories was adequate. All infants survived. Of the 9 followed for 2–48 months (mean, 18), 7 had serious neurologic sequelae.

Diagnosis of SIADH in premature neonates may be difficult due to the complexity of precipitating factors. Its differentiation from other conditions causing hyponatremia depends on review of the history, physical findings and weight curve. Hyponatremia may be due to excessive "free" water intake or to sodium depletion or to both (table). The syndrome in premature infants most often accompanies brain injury and is strongly associated with intracranial hemorrhage. Recognition of the syndrome should prompt a neurologic evaluation including cerebrospinal fluid analysis, frequent measurement of head circumference and a computerized tomographic scan when intracranial hemorrhage is suggested. **(2)**

DIFFERENTIAL DIAGNOSIS OF SIADH*

	Serum Na^+, mEq/Liter	Serum Osmolality, mOsm/kg H_2O	Urine Na^+, mEq/Liter	Urine Osmolality, mOsm/kg H_2O	Weight
Water overloading	< 130	< 280	Within normal limits	< 280	Increased
Appropriate antidiuretic hormone secretion (ADH)	Normal or > 150	Normal or > 300	< 20	> 300	Decreased
SIADH	< 130	< 280	> 80	> 300	May be normal, increased, or decreased
Hypoaldosteronism	< 130	< 280	> 80	> 300	Decreased
Renal sodium wasting	< 130	< 280	> 80	Normal or < 280	Decreased
Inadequate sodium intake	< 130	< 280	< 20	> 300	Decreased
SIADH + sodium depletion	< 130	< 280	< 20	> 300	Increased
Increased ADH and aldosterone	Normal or < 130	Normal or < 280	< 20	> 300	Increased

*Differentiation of SIADH from other conditions causing similar electrolyte picture depends on review of simultaneous serum and urine electrolyte values and osmolality, physical examination, history and body weight changes.

(2) Am. J. Dis. Child. 132:399–402, April, 1978.

Significance of Intracranial Bruits in Neonates, Infants and Young Children. Morris Cohen and Solomon E. Levin[3] (Johannesburg) examined children aged 1 day to 3 years chosen randomly from a children's hospital and from the neonatal section of a maternity hospital. The patients were divided into two groups. Group A consisted of 56 subjects who were normal neonates, premature infants or children with medical conditions but without cardiac murmurs. Group B consisted of 102 subjects with cardiac murmurs. The patients were further subdivided into three age ranges: 1 day to 1 month, over 1 month to 4 months and over 4 months to 3 years. Intracranial bruits (ICBs) were classified into three grades according to the classification of Mace et al.

In group A, 36 children were aged 1 day to 1 month and 7 were aged between 1 month and 4 months. None had ICBs. Of 13 older patients, 4 had ICBs; 1 was grade I, 1 was grade II and 2 were grade III. Two of the 4 patients were found to be anemic.

In Group B, 23 children had ICBs. Of 38 aged 1 day to 1 month, 2 had ICBs. Of 31 infants in the middle age range, 2 had ICBs. Among these 4 babies, the grade of intensity of the ICBs was I or II. Of 33 patients in the older age range, 19 had ICBs. Intensity varied from grade I to grade III; 6 patients had ICBs regarded as grade III. On closer analysis, it was found that all the 23 patients with ICBs in group B had precordial murmurs of intensity grade III/VI or greater.

In infants under age 4 months, even in the presence of a loud cardiac murmur, an ICB is rarely heard. The presence of an ICB, with or without signs of cardiac failure, strongly suggests an intracranial arteriovenous fistula.**(3)**

► [Listening for cranial bruits is not done frequently enough. It is just too inexpensive to be popular.—F.A.O.] ◄

Fulminant Necrotizing Enterocolitis Associated with Clostridia. Neonatal necrotizing enterocolitis (NEC) has become the most common surgical emergency of the newborn infant. It is characterized by crepitant necrosis of the gut, which may lead to perforation, sepsis and death. Ann M. Kosloske, John A. Ulrich and Howard Hoffman[4] (Univ. of New Mexico) in 1976–78 investigated the relation between clostridia and NEC in 17 infants in whom a diagnosis was based on abdominal distention, gastrointestinal bleeding and x-ray evidence of pneumatosis intestinalis. Initial management was with nasogastric suction, intravenous fluids and antibiotics, and gentamicin instillation into the gut. Surgical treatment was indicated for pneumoperitoneum or peritoneal fluid that was brown or contained bacteria on smear. Six infants were receiving antibiotics at the time of onset of NEC.

Peritoneal fluid was obtained by paracentesis in 14 infants and by lavage in 1. Five infants with negative peritoneal-fluid and blood cultures recovered with medical treatment. Twelve with positive cultures required surgical treatment (table). Eleven infants had resection of necrotic intestine. All specimens showed at least one focus of

(3) Arch. Dis. Child. 53:592–594, July, 1978.
(4) Lancet 2:1014–1016, Nov. 11, 1978.

CLINICAL AND BACTERIOLOGIC FINDINGS IN SURGICALLY TREATED NEC PATIENTS

Patient	Pneumatosis intestinalis*	Type of gangrene†	Peritoneal-fluid culture: Aerobic	Peritoneal-fluid culture: Anaerobic	Outcome
1	Mild	Focal	Klebsiellæ	N.G.	Survived
2	Moderate	Focal	Klebsiellæ	N.G.	Survived
3	Mild	Focal	N.G.	N.G.	Survived
4	Moderate	Focal	Klebsiellæ	N.G.	Survived
5	Mild	Focal	Klebsiellæ	*Bacteroides fragilis*	Survived
6	Severe	Focal	. .	. .	Survived
7	Severe	Extensive	Klebsiellæ *Staph. aureus*	*Clostridium* sp.	Died
8	Severe	Extensive	N.G.	*Cl. perfringens*	Died
9	Severe	Extensive	*Staph. aureus*	*Cl. perfringens* *Clostridium* sp.	Died
10	Moderate	Focal	N.G.	*Clostridium* sp. *Bacteroides fragilis*	Survived
11	Severe	Extensive	*Streptococcus fæcalis* α-hæmolytic streptococci	*Eubacterium* sp. *Cl. perfringens*	Died
12	Moderate	Focal	Klebsiellæ *Streptococcus bovis*	*Bacteroides* sp. *Cl. perfringens*	Survived

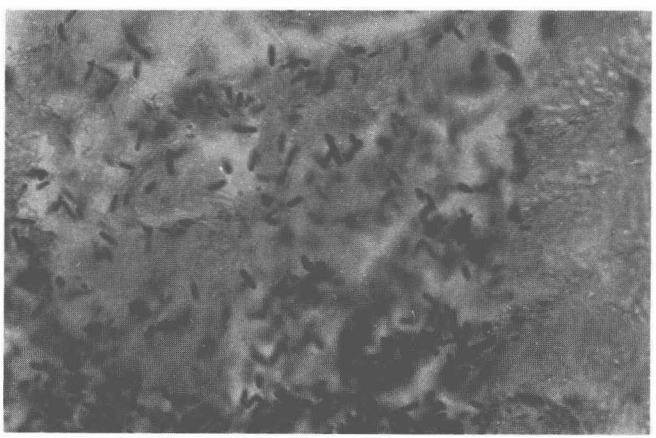
NG = no growth. Blood culture was negative in all patients except Patient 6 (clostridia), 7 *(C. butyricum)* and 11 *(Eubacterium sp.)*.

*Portal venous gas was found only in patients 7–9 and 11.

†Perforation was found only in Patients 4, 6 and 8–11.

bland ischemic necrosis. Gram staining showed bacteria in the gut wall in eight of eleven segments. The 7 clostridia-positive infants had a more severe course than the others. Of the 7 infants, 5 had intestinal perforation, and 4 died (Fig 1). Three of 4 infants who harbored *Clostridium perfringens* had fulminant disease, with early perforation and extensive gangrene of the bowel, and all 4 infants died.

Fig. 1.—Segment of ileum from Patient 8, showing abundant gram-positive rods typical of clostridia within the muscularis. Gram stain; reduced from ×657. (Courtesy of Kosloske, A. M., et al.: Lancet 2:1014–1016, Nov. 11, 1978.)



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