neonatal lambs. Metabolic acidosis counteracts the cardiac response but does not influence the rate of oxygen consumption.

42. Effect of hypercapnia on a blood-brain barrier in fetal and neonatal sheep. C. A. N. Evans, J. Reynolds, M. Reynolds, N. R. Saunders, and M. B. Segal. *University College, London, England.*

Penetration of sucrose from blood into brain which is very slow in the adult has been used to investigate the development of the blood-brain barrier (BBB) in fetal and neonatal sheep. Approximately constant blood levels of ^{14}C sucrose have been maintained by an intermittent intravenous injection technique over 1½ hr, after which cerebrospinal fluid and brain were removed, their ^{14}C activity was measured and compared with that in the blood during the 1½-hr injection period. Fetuses as young as 100 days gestation have a barrier to ^{14}C sucrose which is similar to that found in adult animals. However, fetuses at term (140 days) and newborn lambs show an increase of about 300% in sucrose penetration when made severely hypercapnic (arterial $pCO_2 > 90$ mm Hg). A smaller effect occurs at lower levels of pCO_2. The effect of CO_2 is also less in older animals. In other experiments, acidosis induced by slow intravenous infusion of ^{2}N-lactic acid has little effect on sucrose penetration, although arterial pH was as low as that in the hypercapnic animals. Asphyxia produced by intermittent cord occlusion in the fetus or underventilation in newborn lambs did not affect sucrose penetration unless arterial pCO_2 showed a sustained rise above about 60 mm Hg. A possible explanation for the effect of hypercapnia is that the increase in cerebral blood flow which occurs in hypercapnia may cause more sucrose penetration because of the increased surface area for exchange across the BBB. Krypton-85 clearance has been used to estimate superficial cortical blood flow. Preliminary observations suggest a correlation between cortical blood flow and sucrose penetration into cortical tissue since both appear to be proportional to arterial pCO_2. Other possibilities being considered are (*1*) a direct effect of CO_2 upon the permeability of the barrier, (2) an increase in the size of the brain extracellular space.

43. Plasma amino acid ratio in infants born after pregnancies complicated by toxemia, placental infarction, impaired umbilical circulation and chronic maternal diseases. J. Mestyán. *Univ. of Pécs, Hungary.*

The ratio of the plasma concentration of glycine + serine + glutamine + taurine to the concentration of leucine + isoleucine + valine + methionine has been studied by Whitehead's method using paper chromatography. The mean postnatal rise in the ratio was found to be significantly higher than that observed previously in infants born to mothers with uncomplicated pregnancies. The higher ratio observed in the different series of examinations was due to a pronounced opposite response in the plasma level of the two groups of amino acids included in Whitehead's ratio test. The results will be discussed in relation to conditions interfering with uteroplacental functions.

44. Combined cortical and medullary adrenal hyporesponsiveness in hypoglycemia of infancy and childhood. R. P. Zurbrügg, H. U. Tietze, K. A. Zuppinger, E. E. Joss, and H. Käser. *Univ. of Berne, and Swiss Ctr. for Clin. Tumor Res., Berne, Switzerland.*

In contrast to healthy subjects a group of children with hypoglycemic attacks do not respond with an appropriate increase in urinary epinephrine (E) during the insulin tolerance test, ITT (Broberger *et al*, 1959, 1961). The significance of the E deficiency regarding the disturbance of blood glucose regulation is still uncertain. Therefore, the possible importance of growth hormone (GH) and especially cortisol (F) was evaluated under various stimuli. The eight patients investigated were suffering from hypoglycemia with an insufficient or virtually absent urinary E response.

During ITT the clinical signs of hypoglycemia were lacking. The mean plasma glucose fell to a significantly lower minimum and the rise toward normalization, as expressed by the recovery index, was significantly slower. There was no impairment of GH response.

The most impressive finding during ITT was a deficient response of plasma F which could be shown to be independently impaired from the lacking E excretion. F and E response to intravenous glucagon was also found to be impaired in the same group of patients.

The simultaneous and independent occurrence of both an adrenomedullary and adrenocortical disturbance gives evidence for an impairment of hypoglycemia-sensitive centers for E as well as F regulation located most probably in the hypothalamus. In addition, a delayed response to metopirone, a lack of sweating during hypoglycemia and a positive history for birth injury in all patients also are in favor of a central nervous system dysregulation.

45. Incomplete renal tubular acidosis with hypercalciuria in siblings. K. Schärer. *Children's Univ. Hosp., Heidelberg, Germany.*

Two female siblings were first seen at the age of 7 and 9 years with a recent history of pyelonephritis. On admission, polyuria, hypercalciuria (up to 10 mg/kg 24 hr), and advanced nephrocalcinosis were noticed. Growth was normal. Renal function studies revealed leukocyturia, proteinuria, moderately decreased glomerular filtration rate, impaired concentrating capacity, an acidification defect (minimal urinary pH 5.4 3 days after 150 mEq/m²/24 hr of NH_4Cl), a normal HCO_3 threshold, and a low citrate excretion. Under basal conditions, acidosis was never observed. Potassium metabolism and amino acid excretion were normal. Radiological examination showed slight osteoporosis. In a kidney biopsy patchy interstitial fibrosis and glomerular sclerosis were reported. Increased calcium absorption by the gut was excluded by following the calciuria during prolonged starvation and the activity of Ca^{47} (200 nCi *p. os*) in the whole body and in the stools. Phosphate reabsorption in the kidney rose as in normal subjects after a Ca infusion, but insufficiently after administration of parathyroid hormone. Treatment with a diet low in Ca resulted in a fall of the Ca excretion with a negative Ca balance. By a salt restricted diet, given over 6 months, the balance increased considerably and the calciuria became normal. Hydrochlorothiazide provoked hypocalcemic tetany. The father of the siblings is a past stone former, has a high Ca excretion, but can acidify his urine normally. It is believed that these girls present an incomplete familial form of (distal) tubular acidosis distinct from other types of tubular acidosis and from idiopathic hypercalciuria.

46. Origin of the X chromosome in patients with XO Turner's syndrome. S. N. Pantelakis and A. Karakelis. *Aghia Sophia Children's Hosp., Athens, Greece.*

The frequency of maternal *versus* paternal origin of the single X chromosome in cases of XO Turner's syndrome has not been decided with certainty. Additional data from informative