was most pronounced in the smallest arteries. Control and hyperoxic artery groups were similar. Medial thickness decreased in all vessels with age; this decrease was greatest in the control and hyperoxic groups. This study demonstrates that maternal hypoxia produces quantitative changes in the fetal and newborn pulmonary arteries.

The effects of maternal hypoxemia on fetal and infant development. RUTH WHITTEMORE, GERALD ANDERSON, MARCELLO ORZALESI, and SHIRLEY DRISCOLL. *Yale Univ. Sch. of Med., Yale-New Haven Hosp., New Haven, Conn., and Harvard Univ., Boston, Mass.* (Intr. by Charles D. Cook).

Pregnant women with cyanotic cardiac defects have a high percentage of abortuses and infants small for gestational age. The primary aim of this study was to determine the effect of maternal hypoxemia, as measured by arterial blood gas tensions, on fetal and placental development, neonatal adaptation, and subsequent growth and development. 19 women with right-to-left shunts were studied and 21 of the 30 pregnancies resulted in live births. 11 of these infants were small-for-dates, all born to women who had no surgery or only palliative surgery over 10 years ago. Mothers with total correction of these shunts delivered infants of normal size. Urinary estriol excretion during pregnancy was studied in 6 patients; low urinary estriol levels and low arterial blood oxygen tensions ($P_{O_2} < 60$ mm Hg) were associated with low birth weight of the infants. Placental size was larger than expected on the basis of birth weight. Blood O_2 affinity and red cell 2,3-diphosphoglycerate (DPG) were studied in 2 cyanotic women and in their infants at birth. Blood O_2 affinity was significantly decreased and DPG markedly increased in both mothers; the same changes, although to a lesser degree, were observed in their infants, indicating intrauterine adaptation to hypoxia. The postnatal growth and development of all small infants have equalled or exceeded the normal standards.

Echocardiography in the diagnosis of neonatal congenital heart disease. RICHARD A. MEYER and SAMUEL KAPLAN. *Univ. of Cincinnati, Cincinnati, Ohio.*

Since its introduction in 1954, diagnostic ultrasound has been used to record the movement of atrio-ventricular valves, to measure atrial and ventricular dimensions, left ventricular free wall thickness, left ventricular outflow dimension and to detect the presence of pericardial effusions. In this study echocardiograms were obtained in 50 normal neonates (age 5–96 hours) as part of a control study. In every instance distinct mitral and tricuspid valve echoes were easily recorded. Left ventricular end-diastolic diameters were 1.5–2.0 cm. and the diameter of the left ventricular outflow tract was 0.8–1.2 cm. Echocardiograms were obtained from 16 infants with a variety of cardiac malformations (age 2–16 weeks) in whom diagnoses were confirmed either by cardiac catheterization or autopsy. In 2 babies the diagnosis of hypoplastic left heart syndrome was made because of absence or gross distortion of reflected ultrasound from the mitral valves, and left ventricular end-diastolic diameters which were far below the values observed in our normal neonates. At autopsy the mitral and aortic valves were atretic and the left ventricles hypoplastic in both patients. In 3 babies the clinical diagnosis of hypoplastic left heart syndrome was untenable because of normal mitral valve reflection and left ventricular dimensions. Subsequent cardiac catheterizations confirmed the ultrasonic findings. In another baby the echocardiographic diagnosis of tricuspid atresia was confirmed at autopsy. This technique provides a non-invasive method of evaluating the hearts of neonates. Infants suspected of having the hypoplastic left heart syndrome may be diagnosed by ultrasound without the necessity of cardiac catheterization.

The site of congenital and surgical heart block in children. KENNETH M. ROSEN, ASHWIN MEHTA, SHAHBUDIN H. RAHUIMTOOLA, and ROBERT A. MILLER. *Cook County Hosp. and the Univ. of Ill. Col. of Med., Chicago, Ill.*

Catheter recordings of His bundle (BH) electrograms were obtained in five children with asymptomatic congenital heart block (CHB) and two children with symptomatic surgical heart block (SHB). In the SHB patients, block followed repair of tetralogy of Fallot in one and of A-V canal in the other. One patient with CHB had an associated secundum atrial septal defect. A-V dissociation was present in all patients with atrial rates of 80–100 and ventricular rates of 36–52 per minute. In the patients with CHB, BH spikes were unrelated to P waves and preceded every QRS. Intraventricular conduction was normal in four patients with CHB, who had H-Q intervals of 35–40 msec. (normal 35–55) and QRS duration of less than 0.10 seconds. One patient with CHB had a wide QRS (0.12 seconds) with initial delay suggesting type B pre-excitation. In the latter patient, H-Q was only 25–30 msec and the right ventricular (RV) electrogram preceded that of the left, suggesting either anomalous insertion of BH into the RV or an accessory infranodal communication to the RV. In the patients with SHB, H spikes followed P waves with P-H of 125 and 115 msec respectively. In the latter two patients, there was complete block distal to BH.

In conclusion, CHB was characterized by block proximal to the BH recording site (RS), consistent with block in either the A-V node or proximal His bundle. This was in contrast to SHB, where block was distal to the BH RS consistent with a lesion in the distal His bundle.

The effect of respiratory frequency on gas exchange in asthma. ANDRE LAMARRE, ARTHUR C. BRYAN, ANTHONY MANSELL, and HENRY LEVISON. *Univ. of Toronto and Hosp. for Sick Children, Toronto, Ont., Canada.*

The presence of unequal time constants in the lung should lead to a progressive inequality of ventilation distribution and gas exchange failure at high breathing frequencies. However, in a group of 8 adolescent asthmatics we have not been able to demonstrate such a relationship between breathing frequency and gas exchange. These subjects were trained to alter respiratory rate (f) over a wide range while maintaining their arterial carbon dioxide tension ($PaCO_2$) within 3 mmHg of their resting values. At rest ($f = 14.7 \pm 4.8$) the alveolar arterial oxygen gradient ($AaDo_2$) was 37.0 ± 9.9. At an intermediate rate ($f = 30.0 \pm 3.77$) the $AaDo_2$ rose significantly to 39.4 ± 8.5. At high rates ($f = 104.5 \pm 18.9$) the $AaDo_2$ fell significantly to 34.1 ± 9.4. The validity of alveolar arterial gradients at high rates is doubtful, but a similar effect although not significantly different, was seen in the arterial oxygen tension: rest 72.3 ± 9.00, intermediate 70.4 ± 7.3, fast 73.3 ± 9.5. There was no significant difference in alveolar ventilation (rest 4.95 L/min $\pm$ 1.38, intermediate 5.31 ± 1.51, fast 5.76 ± 1.55). To explain this conservative behaviour, we have postulated that the regional resistances and compliances must be non-linear acting in a direction to equalize the ventilation distribution as frequency increases. In non-laminar flow, resistance is flow dependent and over-distention of regional units will reduce their compliance. Both these factors will tend to stabilize distribution.