at several frequencies (F) from 1–15/sec, duration 5 msec, and 10–15 V. Slowing was a function of F, but was unaltered by changes of PO_2, PCO_2 or pH up to an F of 10/sec. At F 15 the bradycardia was often enhanced by hypercapnia. It is concluded that hypoxemia and lactic acidemia do not alter adrenergic (A) or cholinergic (C) transmission, but that hypercapnic acidosis may reduce A, and enhance C during maximal stimulation. This suggests a mechanism whereby autonomic control of the heart may be altered in neonatal asphyxia.

34 *The Role of the Adrenergic Nervous System in Regulating Systemic Arterial Pressure and Venous Return.* RICHARD M. SCHIEKEN, GEORGE J. PECKHAM, ARMAN BILGIC and WILLIAM J. RASHKIND, The Children's Hosp. of Philadelphia, Philadelphia, Pa.

Total cardiopulmonary bypass was achieved in closed chest dogs. Systemic venous return (SVR) was directed through a recording electromagnetic flowmeter. Systemic arterial (SAP) and central venous pressure (CVP) were also monitored continuously. Alpha adrenergic agents caused a mean increase in SAP of 74% without any change in SVR. Beta adrenergic agents produced a mean fall of 34% in SAP and a mean increase of 9% in SVR and a mean increase of 4% in CVP. Alpha adrenergic blockage simulated beta adrenergic effect with a mean fall in SAP of 52% and a mean increase of 8% in SVR and a mean increase of 3% in CVP. No significant changes occurred as a result of beta adrenergic blockade. These data demonstrate the dominent effect of the beta adrenergic nervous system in increasing systemic venous return by an increase in venomotor tone.

35 *Increased Oxygen Release in Hypoxemia and Heart Failure.* WILLIAM W. MILLER, FRANK A. OSKI, MARIA DELIVORIA-PAPADOPOULAS, Dept. of Pediatrics, Medical School of the University of Pennsylvania, Philadelphia.

Red cell 2,3-diphosphoglycerate (DGP), a primary regulator of oxygen-hemoglobin affinity, was measured in 55 infants and children with congenital cardiac diseases. The levels were compared with those from 20 normal patients and were analyzed with regard to patient age, arterial oxygen tension (P_aO_2), and the presence of heart failure. In 14 patients the oxygen dissociation from whole blood was also measured, and values for DPG were correlated with those for P 50, the PO_2 for 50% oxygen saturation.

Elevated levels of DPG were found in 17 of 18 infants and children with heart failure, regardless of age or P_aO_2.

Thirty-seven patients had subnormal P_aO_2, but no heart failure. Among those > 3 months of age, DPG was elevated in 26 of 28 with P_aO_2 = 16–56 mm Hg, and it was normal in 5 with P_aO_2 > 56 mm Hg. In these 33 older patients, the level of DPG was inversely related to P_aO_2 ($r = -0.656$). In four hypoxemic infants < 3 months, DPG was normal.

The close correlation found between elevated values for red cell 2,3-DPG and for whole blood P 50 ($r = 0.7142$) supports *in vitro* studies indicating that DPG is a primary regulator of oxygen release from human blood. The correlation between levels of DPG and of P_aO_2 in patients > 3 months supports *in vitro* studies demonstrating deoxyhemoglobin control of red cell DPG synthesis. Among newborn infants the elevated levels of DPG measured in those with heart failure and the normal concentrations found in those with hypoxemia indicate two different control mechanisms for synthesis of this important modulator of oxygen homeostasis.

36 *Effects of Chronic Hypoxemia on the Electrophysiological Properties of Right Atria Tissue in Children.* HENRY GELBAND, HARRY L. BUSH, GERARD A. KAISER, JAMES R. MALM, ROBERT J. MYERBURG and BRIAN F. HOFFMAN, Coll. of Physicians and Surgeons, Columbia Univ., NY. (introduced by O. R. Levine).

The purpose of this study was to determine the electrophysiological properties of normal right atrial tissue in children and to evaluate the role of chronic hypoxemia in the genesis of atrial arrhythmias. Using standard microelectrode techniques we studied the electrophysiological properties of preparations of right atrial tissue obtained during open heart surgery in two groups of patients. Group A consisted of 6 patients with uncomplicated ventricular septal defects. Group B comprised 6 patients with tetralogy of Fallot with arterial O_2 saturations of 80–84%. All patients underwent cardiovascular diagnostic studies and had normal right atrial pressures and no evidence of atrial shunting. In Group A mean resting potential (RP) was -86 ± 5.7 mV, action potential duration was 210 ± 8 msec at a cycle length of 1,000 msec and conduction velocity was 0.45 ± 0.05 M/sec. The relationship between the rate of rise of phase 0 of the action potential (AP) and the membrane potential at which the AP was elicited (membrane responsiveness) was 'S' shaped. Cells were unresponsive at a mean RP of -56 ± 1.6 mV and maximum rate of rise occurred at a mean RP of -86 ± 5.7 mV. An increase in extracellular [K+] above control caused a decrease in the RP which was linearly related to log [K+]. When [K+] was reduced below control levels, RP decreased slightly. There were no significant differences ($p = > 0.05$) between Group A and Group B for all properties measured. Results from Group A can be used for 'normal' reference values. Chronic hypoxemia does not seem to alter the electrophysiological properties of right atrial tissue. This is consistent with the clinical observation of the lack of atrial arrhythmias in cyanotic congenital heart disease when there is no pressure or volume overloading of the right atrium.

37 *Evaluation of Left Ventricular Contractile State in Children With a Chronic Left Heart Pressure Overload.* THOMAS P. GRAHAM, JAY M. JARMAKANI and RAMON V. CANENT, Duke Univ. Med. Center, Dept. of Ped., Durham, NC (introduced by Madison S. Spach).

Recent animal investigations have indicated that myocardial hypertrophy may be accompanied by a depression of muscle function as analyzed in terms of length-tension and force-velocity relationships. Myocardial function was evaluated in these terms in 14 children with left ventricular hypertrophy (LVH) secondary to aortic stenosis or coarctation of the aorta and 10 children with normal left hearts who were undergoing diagnostic cardiac catheterization. Serial left ventricular volumes were calculated from biplane cineanangiocardiograms exposed at 60/frames sec, and left ventricular pressure was recorded simultaneously using a catheter tip transducer. Left ventricular circumferential stress (LVS), LV circumference at the equator