subjects and 10 asthmatics as controls. Instantaneous changes in lung volume, air flow and esophageal pressure were measured and simultaneous flow-volume and flow-pressure curves recorded. Striking differences were noted between asthma and CF: 1. below 95 % of the total lung capacity, pressures in excess of those required to produce maximum expiratory flow rates depressed flow in CF but not in asthma; 2. forced expiration was associated with a transient reversal in the slope of the single breath nitrogen curve in CF and not in asthma. Cineradiographic evidence of collapse of bronchiectatic airways during forced expiration and cough with retention of contrast medium distal to the collapse was provided. It is concluded that in CF: 1. airway obstruction is less uniform and involves larger airways than in asthma; 2. increased expiratory pressure collapses larger airways. This study emphasizes the necessity of combining postural drainage with liquefaction of bronchial secretions in CF. (SPR)

8 *Influence of Intracardiac Shunting on Left Ventricular Muscle Mechanics in Tetralogy of Fallot.* MOUAZZA M. JARMAKANI*, MADISON S. SPACH, SAM B. EDWARDS*, RAMON V. CANENT, Jr.*, M. PAUL CAPP*, VISHNU JAIN* and ROGER C. BARR*, Duke University Medical Center, Durham, N.C.

The need continues for better quantitation of left ventricular function. Previous studies have indicated the nature of intracardiac shunting in tetralogy. Simultaneously recorded left ventricular pressure and biplane cines were analyzed to construct continuous left ventricular function curves in 'normales' and in tetralogy patients. The original measurements throughout 3–6 consecutive beats were analyzed by a digital computer with numerical and graphic outputs of: LV pressure, LV volume, rate of volume change (flow), LV mid-circumference and its instantaneous velocity, tension at the endocardial surface, work and power. The force-velocity-length and pressure-volume-flow relationships of the left ventricle were depicted in three dimensional plots during the active state (systole) of the LV muscle for normals and tetralogy patients. In normals, maximum flow and peak velocity of shortening occurred at high tension levels in mid-systole; whereas in tetralogy, peak flow and velocity occurred during 'isovolumic' contraction at lower tensions. The continuous' function curves' were markedly abnormal in tetralogy with peak rate of flow and of circumference shortening occurring prior to opening of the aortic valve. The results indicate that the left ventricle in tetralogy functions more efficiently than normal and unloads 15 to 40 percent of its stroke volume during isovolumic contraction. This results in lower than normal stroke work values. (SPR)

9 *Studies of Left Ventricular Function in Children by Increasing Peripheral Resistance with Angiotensin.* L. JEROME KROVETZ, THOMAS G. MCLOUGHLIN* and GEROLD L. SCHIEBLER, Univ. Florida Col. of Med., Gainesville, Fla.

While satisfactory catheterization techniques for detection of shunts and abnormal movements of heart valves are available, assessment of myocardial function remains difficult. In normal human subjects angiotensin, produces systemic vasoconstriction which results in increased left ventricular stroke work (LVSW). Ross and BRAUNWALD studied 18 patients using graded infusions of angiotensin and showed a good correlation of clinical severity and LV function curves. To learn the value of this technique in children, LV function was assessed using graded infusions of angiotensin (2 to 3.8 mcg/min) in ten normals (age range 5 to 15 years) and 21 patients with LV abnormalities (age range 2 months to 15 years). The best method of graphing this data is to plot LV end-diastolic pressure versus the ratio of calculated to predicted LVSW. Predicted LVSW (in newton-meters), based on 24 normal children, is given by the formula: $= 0.745 + 0.0133$ wt (kg) $- 0.00243$ ht (cm) $- 0.00384 \times$ heart rate. All nine patients with primary LV endocardial fibroelastosis had abnormal LV function curves. Four of eight patients with Hunter-Hurler syndrome had depressed LV function as did two of three patients with idiopathic myocardial hypertrophy. One 2-month-old male with cardiac glycogenosis had a markedly depressed LV function curve, LV stroke work decreasing with increasing LV end-diastolic pressure.

Of the 16 abnormal LV function curves, only 7 had resting elevated LV enddiastolic pressures. An additional 5 had low resting LVSW ranging from 17 to 43 % of predicted. Thus, abnormal LV function was detected only following angiotensin infusion in one-fourth of these patients. (SPR)

10 *Neurohumoral Mechanisms of Ventricular Tachycardia in Experimental Heart Block.* HERBERT D. RUTTENBERG*, ROGER A. HURWITZ* and IWAO KANDA*, UCLA Sch. of Med., Los Angeles, Cal. (introduced by Leonard M. Linde).

The purpose of this study was to investigate the pathways by which electrical stimulation of the hypothalamus produces paroxysmal ventricular tachycardia (PVT) in dogs with chronic complete atrioventricular (A-V) block. Left thoracotomy was performed in 20 dogs to pass wire loops around the stellate ganglion and ansa subcalvia (cardiac nerves). Two weeks later, right thoracotomy was performed for placement of wires around the right cardiac nerves for production of complete A-V block by transatrial injection of formalin into the A-V node. One week later, under chloralose anesthesia, monopolar electrodes were placed into the lateral hypothalamic areas for 20 sec stimulations of 20 V, 1 msec, 50 cps stimulations. Aortic pressures (AP), left ventricular pressures (LVP) and ECG were recorded. In all 20 dogs, stimulation in the posterolateral hypothalamus (fields of Forel) caused an immediate depressor response followed by an increase in AP (systolic, diastolic and pulse pressure) with moderate increase in ventricular rate (VR). In 8 of these dogs, the pressure rise was followed by PVT with an average latency of 23 sec, a change in VR from 56 (resting VR) to 140, and a change in site of pacemaker. Production of PVT was not prevented by bilateral ablation of cardiac nerves. The long latent period suggested a humoral pathway. Administration of epinephrine HCl 5 to 10 μg/kg body weight produced PVT in 10 of 13 dogs with a latent period of 25 sec. This humorally-induced PVT was not prevented by ablation of cardiac nerves or α-adrenergic receptor blockade but was blocked by β-adrenergic receptor blockade. These studies suggest that adrenergic humoral mediation is a major pathway for production of PVT in heart block. (SPR)

11 *The Effect of Human Growth Hormone (HGH) on Red Cell Glucose Metabolism.* FRANK A. OSKI and ALLEN ROOT*, Dept. of Pediatrics, Univ. of Pa. Sch. of Med., Philadelphia, Pa.