# ABSTRACTS

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Abstracters

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## ENDOCARDITIS, MYOCARDITIS, AND PERICARDITIS


In a period of 3½ years, 17 infants and children (2 to 3 per 1,000 admissions) suffering from pericarditis with effusion were studied at the University of Oklahoma Medical Center. The etiology was rheumatic fever, four cases; purulent infection, three; virus infection, one; metastatic tumor, one; uremia, one; rheumatoid arthritis, one; fibrocystic disease, one; and postcardiomyopathy syndrome, five. Technics used to diagnose pericardial effusion in the absence of a friction rub include cardiac catheterization, angiocardiography, and pericardial paracentesis, which the authors regard as reliable but difficult, tedious, and potentially hazardous, and also fluoroscopy, kymography, and isotope scanning, which, although safe, they regard as unreliable. Since accuracy of diagnosis is essential for satisfactory management, the choice of diagnostic procedures requires astute clinical judgment.

**Marshall**

## HYPERTENSION


Urinary catecholamine excretion was estimated biologically in over 500 hypertensive patients. More than 96 per cent of these were under 50 mg. per 24 hours, and values above this level in the authors' laboratory were considered diagnostic of pheochromocytoma. There was a considerable day-to-day variation in the amount of catechols excreted by normal individuals (as well as those with chromaffin tumors), and distinctly elevated values were observed after surgery. Twelve patients had medullary pheochromocytomas. All nine patients tested showed markedly increased catechol excretion, but 3 revealed normal excretion values on single days. Eight of these patients had estimates both of epinephrine and norepinephrine excretion, and four were found to excrete a mixture of the two catechols. Four patients had paraganglionic pheochromocytomas. All of them passed increased amounts of catecholamines, 100 per cent of which were norepinephrine. Thus, the hormone excretion pattern provided in some instances preoperative indication of the tumor site. There was no significant correlation between tumor weight and amount of catechols in the tumor or excreted in the urine. There was a correlation between the excretion of vanillyl mandelic acid and total catecholamine in hypertensive patients without tumor but not in those with pheochromocytoma.

**Rogers**

Stokke, H., and Mathisen, H.: Guanethidine and Chlorthalidone in Hypertension. Long Term Treatment of Hypertension with the
Combination of Guanethidine (Ismelin) and Chlorthalidone (Hygroton). Cardiologia 42:349 (June), 1963.

Sixty-three patients with diastolic hypertension, in many cases severe, were treated with a combination of guanethidine and chlorthalidone for 3 to 24 months. The maintenance dose of guanethidine averaged about 25 mg. daily. There was a satisfactory fall in blood pressure in 81 per cent of the treated patients. Side effects attributed to guanethidine were encountered in 44 per cent, and comprised weakness, dizziness, loose bowel movements, and failure of ejaculation. In 17 per cent chlorthalidone caused asymptomatic hypokalemia. Increased tolerance to guanethidine did not occur.

Marshall

PATHOLOGY


Porous prostheses constructed from woven Nylon, Terylene, and Courlene, and imporous prostheses from Nylon and polyethylene sheet fused together, were used to replace excised segments of aorta in dogs. The histologic response was studied by light and electron microscopy 1 to 6 years after implantation. The response to porous fabrics was an orderly deposition of tissue on both sides of the prosthesis. Electron microscopy showed that its cellular components were smooth muscle cells and macrophages. However, the intimal lining of the impermeable prostheses was incomplete, the central part being composed only of a fibrinous material. It was concluded that there are two sources for the intimal lining of prostheses, namely, growth in continuity across the anastomosis from the intima of the host artery, which rarely extends more than 1 cm., and penetration of cells and fibers through the interstices of woven fabrics. This process is impossible in imporous fabrics.

Marshall

PHARMACOLOGY


Bradycardia is a well-known sign of thiamine deficiency. In theory this could be due to decreased rate of synthesis of intracellular acetylcholine or to increased vagal activity. The author compared the effect of acetylcholine on the heart rate in the isolated hearts of normal and of thiamine-deficient rats. The thiamine-deficient heart in vitro had a regular normal beat and amplitude. The inhibitory effect of acetylcholine was identical in the normal and the thiamine-deficient heart. Addition of thiamine to the perfusing fluid did not alter the inhibitory effect of acetylcholine. Since the behavior of the thiamine-deficient heart in vitro so closely resembles that of the normal heart, it is unlikely that thiamine deficiency is directly the cause of bradycardia.

Marshall


Electronic differentiation of the pressure pulse obtained from the right or left ventricle by means of a catheter-tip transducer was employed in order to measure the maximum rate of increase of pressure during early systole (peak dP/dT). Observations were made in the right ventricle of four patients without heart disease and two with minimal cardiac abnormalities. After infusion of 0.3 to 0.6 mg. of ouabain, the peak dP/dT increased by 10 to 75 (mean 32) per cent. Observations were made in the left ventricle of four patients with uncomplicated ostium secundum atrial septal defects. Ouabain caused an increase of 26 to 49 (mean 36) per cent in the peak dP/dT. Although quantitation was not possible, ouabain also appeared to increase the rate of change of pressure during atrial contraction. These findings, all in intact, unanesthetized subjects, indicate that ouabain is capable of increasing the contractility of the normal (and nonfailing) heart.

Marshall


In this study the effects of serotonin on cardiac output and blood flow distribution to the various organs were studied in rats. Cardiac output was measured with Evans-blue dye administered intravenously, and samples of blood were taken from the carotid artery every 0.66 second. With use of Sapirstein’s isotope indicator-dilution technic (Rb 86) the fraction of cardiac output going to each organ was calculated from the rate of uptake of Rb 86. Serotonin was injected intraperitoneally and intravenously in various doses. Fifteen to 30 minutes after intraperitoneal injections cardiac
output was not changed while blood pressure was significantly reduced. Increase in blood flow was noted in the myocardium, pulmonary parenchyma, and in the "carcass" (skeletal muscle, bone, central nervous system), whereas kidney and skin flows were decreased. Splanchnic blood flow was unchanged. Conversely, intravenous infusion of serotonin produced an increase in cardiac output, blood pressure, and cutaneous blood flow.

**ABBoud**

**PHYSICAL SIGNS**


The auscultatory and phonocardiographic differentiation of the physiologic ejection murmur from pathologic systolic murmurs was investigated in 80 healthy children. A pansystolic murmur always indicated the presence of organic disease and was distinguished by its occurrence immediately after the valve closure had occurred. An ejection systolic murmur was much less specific, since a physiologic ejection murmur was so common in healthy children. These murmurs were often noted to be quite loud in patients with increased stroke volume (i.e., as with exercise, excitement, anemia, etc.) or where a flat chest wall was present. The decision as to whether a pulmonary ejection systolic murmur is physiologic or pathologic depends on analysis of the second sound; if the second heart sound becomes single in the expiratory phase of continued respiration, pulmonary stenosis and left-to-right shunting atrial septal defect are excluded. Delayed pulmonary closure in expiration may be due to pulmonary valve or infundibular stenosis, anomalous venous return with a large atrial septal defect, dilatation of the pulmonary ring, or to delay in electrical activation or spread from complete right bundle-branch block. An aortic origin of the ejection systolic murmur was suggested, if the murmur was louder in the aortic area than the pulmonary area and was well transmitted to the mitral area; aortic stenosis was diagnosed by finding the appropriate murmur and correlating the murmur with the slow rise and prolonged ejection in the carotid pulse. The differentiation of the physiologic ejection murmur in children from one due to minimal roughening of the aortic valve without significant stenosis or regurgitation was very difficult and one usually had to rely on the intensity of the murmur, particularly in the aortic area.

**KarPMAN**

**PHYSIOLOGY**


Cardiac hemodynamics were carefully studied in 83 babies who were catheterized via the umbilical vein. The left atrial pressure was found to decrease soon after birth and became lower than the adult left atrial pressure. However, the right atrial pressure in the newborn infant rapidly became equivalent to that observed in the adult. The pressures in both atria were initially higher when additional blood was transferred to the circulation from the placenta and, in fact, slight cardiac dilatation was noted to occur, but this was not observed in vigorous infants who had been subjected to little or no asphyxia at birth. The heart was noted to be larger in infants with clinical and biochemical evidence of greater asphyxiation even when they received no additional blood and when their atrial pressures were low. The normal fall in the left atrial pressure was thought to be related to contraction of the ductus arteriosus with a subsequent fall in the volume of blood flow through the lungs. The evidence suggested that cardiac dilatation after birth was related to the degree of asphyxiation and that the principal factor in producing cardiac dilatation is the effect of asphyxiation on the myocardium. Other hemodynamic factors such as hypervolemia may contribute to the end result noted.

**KarPMAN**


The cardiac output was measured by the Fick method in 126 infants and children most of whom had congenital heart disease. It was found that there was no significant difference in the mean resting cardiac index for patients with body surface areas under 1.0 M.² regardless of the presence or severity of the heart disease. Patients with heart disease who were larger than 1.0 M.² had significantly lower mean cardiac indices than the control group. Although the normal increase in cardiac output during exercise was greater for children than for adults, 47 per cent of children with heart disease showed a low cardiac output to exercise.

**Kalmansohn**

Fourteen sedentary men aged 19 to 26 years were studied before and after a 2-month strenuous basic military training period. Slight increases were observed in heart volume, stroke and cardiac index before and after exercise and in left ventricular stroke work index. Slight decreases were noted in pulse rate, brachial artery pressure, total peripheral resistance, and tension-time index. Left ventricular work was unchanged but, in view of the decrease in tension-time index, the work was performed at a smaller oxygen cost. These data were regarded as representing a transitional state in the development of an endurance athlete.

Rogers


Height, weight, and body surface area were compared to the cardiac output, stroke volume, and peripheral vascular resistance in 77 subjects ranging in body size from 0.2 to 2.0 M$^2$ body surface area. Correlation between cardiac output and stroke volume with height, weight, and body surface area was high. The data supported the validity of the mean cardiac index and mean stroke index as a mechanism for eliminating the influence of body size from the hemodynamic data. The cardiac index was slightly higher and the stroke index was slightly lower in the younger age groups but both the blood pressure and the systemic resistance index demonstrated an increase up to the age of 15 years.

Karpman


The ratio of the external work of the heart to its energy requirements ("cardiac efficiency") is not a precise criterion of myocardial efficiency. The best approach to this is the ratio of the total work of the heart to the energy utilization. The total work of each ventricle was calculated from the product of the stroke volume and the mean intraventricular pressure. In 10 dogs the ratio of external to total work (mechanical efficiency) of the left ventricle was, on the average, 94 per cent while the left ventricular myocardial efficiency was 27.5 per cent, resulting in a total cardiac efficiency of 25.8 per cent. After creation of aortic stenosis the mechanical efficiency decreased to 22.9 per cent, the myocardial efficiency to 17.5 per cent, and the cardiac efficiency to 4 per cent.

Lepeschkin


Hemodynamic measurements were made following acutely induced changes in hematocrit levels in 20 anesthetized and chronically splenectomized dogs that had either a normal or an increased blood volume. In both normovolemic and hypervolemic dogs, cardiac output was inversely related to the hematocrit value. Also, at comparable hematocrit values, cardiac output was about twice as high in the hypervolemic as in the normovolemic dogs. In both volume groups, alterations in cardiac output were related more to changes in stroke volume than to changes in heart rate. Also, in both groups the pulmonary vascular resistance progressively increased with increase in hematocrit value. Arterial oxygen saturation tended to increase in normovolemic dogs as the hematocrit value increased, while in hypervolemic dogs it decreased by a statistically significant degree. The quantity of oxygen delivered to the tissues was, at comparable hematocrit levels, approximately twice as great in the hypervolemic as in the normovolemic dogs. It is concluded that both the blood volume and the hematocrit level have independent effects on the circulation. The changes observed in any animal are the resultant of the separate effects of the two factors.

Marshall


This study identifies by a method, independent of those previously employed, the existence of anaerobic metabolism of the heart during inhibition of its oxidative metabolism and demonstrates that the heart can convert anaerobically liberated energy to mechanical work. Energy liberated from substrates of heart muscle metabolism appeared as mechanical work and heat. External mechanical work and heat production of the left ventricle were compared with its oxygen consumption in intact dogs. Left ventricular coronary flow was determined by the nitrous oxide method and cardiac output was determined by the Fick method. The temperatures of coronary arterial and venous blood were measured with thermistors attached to catheters in the ascending aorta and great cardiac vein. Under control conditions, within the range of accuracy possible, the sum of work and heat was equal to energy from oxidative metabolism. Intravascular administration of cyanide in-
creased heart work but reduced its rate of aerobic metabolism. During the cyanide effect work plus heat exceeded the energy available from oxidative metabolism. The difference represented myocardial anaerobic metabolism. Since the energy of mechanical work output alone was greater than the myocardial aerobic energy source, a portion of the anaerobic energy being liberated must have been converted to mechanical work. Possible inaccuracies in the methods and their effects on the conclusions made from the data are discussed in detail.

**ABBOD**


The authors have utilized strips of human atrial appendage removed during mitral valvulotomy, to measure in vitro metabolism of human heart muscle at various temperatures. Oxygen consumption and the ability to metabolize succinate were determined at a control temperature of 37 C. and at 26 C., 15 C., and 8 C. Following cooling, the atrial strips were rewarmed to 37 C. and oxygen consumption and the ability to metabolize succinate were again measured. The experiments demonstrated a reduction in oxygen consumption to 72 per cent of the value obtained at 37 C. when the tissue was maintained at 26 C. and to 40 and 30 per cent of the value obtained at 37 C. when the tissue was maintained at 15 and 8 C., respectively. The ability to metabolize succinate decreases with the declining temperature and ceases by the time 8 C. is reached. The specimens from the bath at 8 C., however, showed a rapid return of the ability to metabolize succinate when placed in the bath at 37 C.

**DEYKIN**


Vasoconstrictor stimuli and bacterial endotoxin activate an inducible form of histidine decarboxylase present in mammalian tissues. Histamine produced from the activation of this enzyme has been postulated to oppose the vasoconstrictor action of the catecholamines. The experiments were performed to test the hypothesis that the unique reaction of rabbit kidney to endotoxin, that which produces some alteration in the glomerular capillaries leading to the generalized Schwartzman reaction, might involve an anomalous response of kidney histidine decarboxylase. The activity of the enzyme might be suppressed rather than stimulated by endotoxin. The results indicate that in the kidney of endotoxin-treated rabbits there was an anomalous response; histidine decarboxylase was strongly reduced rather than activated. This response might result in suppression of induced histamine synthesis at a time when there is increased catecholamine release leading to potentiation of the action of the catecholamine vasoconstrictor on the kidney to the point of causing tissue damage. The authors discuss various possibilities that indicate this event might play a part in the duration or development of the generalized Schwartzman reaction following endotoxin.

**ABBOD**


A dual study of an isolated cat papillary muscle preparation and an in situ innervated, isovolumetric canine cardiac preparation was undertaken to determine the mechanics of isometric contraction of the heart. The canine heart was isolated from the circulation by means of cardiopulmonary bypass. The inferior vena cava, the azygos system, the pulmonary artery, and the bronchial circulation were ligated. The flow from the superior cava cava was returned to the thoracic aorta after isothermic heart lung bypass and the aortic pressure kept constant at a level calculated to prevent aortic valve opening. Coronary sinus and Thesbian vein flow was returned to a reservoir. A balloon was inserted into the left ventricle and balloon and aortic root pressures were recorded under varying distending pressures. The effects of heart rate changes and sympathetic stimulation, as well as the effects of intra-aortic norepinephrine, calcium, and acetyl strophanthidin infusion were determined. The maximum rate of development of isometric tension (dp/dt) and the integrated isometric tension (IIT) were calculated on a beat to beat basis. The results of these experiments suggest that the ratio dp/dt/IIT is a quantitative measure of the contractility of the myocardium.

**HELVIG**


The purpose of this study was to determine the effect of sympathetic stimulation on ventricular distensibility and on ventricular end-diastolic pressure-volume relations. The authors indicate at the
outset that the term pressure-volume relation refers to the relation between pressure and volume whether or not the ventricle is relaxed, while the term distensibility refers to the relation between pressure and volume only when the ventricle is relaxed. Their experiments were carried out on nine isovolumically contracting canine left ventricle preparations. The left ventricle was isolated hemodynamically from the circulation and a fluid-filled rubber balloon was attached to a rigid metal cannula and inserted into the left ventricular cavity through the apical dimple. Stellate ganglion stimulation did not induce any alteration in ventricular distensibility at various ventricular volumes. When the heart was paced at high rates with bipolar electrodes on the left atrium, diastole was sufficiently curtailed to make ventricular relaxation incomplete and increase end-diastolic pressure thus changing end-diastolic pressure-volume relation of the ventricles. When sympathetic stimulation was carried out at these high rates, systole was shortened and the rate of relaxation was accelerated, thus allowing more time for adequate ventricular relaxation to occur and restoration of the relaxed pressure-volume relation without any indication, however, that ventricular distensibility was changed.


This study was performed to establish the functional status of the heart at different degrees of coronary embolization with radioactive microspheres. Blood was taken from the right atrium and passed through a pump and a rotameter and delivered into the pulmonary artery. A blood reservoir allowed the rapid transfusion or bleeding of the animals. The radioactive plastic microspheres with an average diameter of 285 μ were injected proximal to an inflated balloon placed at the root of the aorta; the occlusion of the aorta being maintained for 30 seconds after the injection of the microspheres. Left ventricular function curves were obtained by plotting the mean left atrial pressure against cardiac output or left ventricular minute work. In each of 11 experiments both the maximum cardiac output and maximum minute work were decreased progressively with increasing quantities of microspheres, indicating that the functional ability of the heart can be expressed very well by the maximum level of these function curves. It appeared that once the coronaries were emblazoned at a certain critical degree the heart would enter a vicious cycle of progressive deterioration. The average volume of microspheres sufficient to cause death of the animals was 6.5 mm³.


A denervated in situ canine heart-lung preparation was devised in which stroke volume (by means of measured pump inflow to the pulmonary trunk), aortic pressure (by controlling aortic resistance), and heart rate (pacemaker) could be varied independently and the duration of isovolumic and total left ventricular ejection time was determined before and after infusion of norepinephrine and acetyl strophanthidin. Increasing the stroke volume prolonged ejection, shortened the isovolumetric phase, and had little or no effect on the duration of total systole. Increasing mean aortic pressure had the reverse effect while total systole was unchanged or slightly decreased. Increasing the heart rate or administration of acetyl strophanthidin or norepinephrine shortened all phases of systole.

ABBOUD


The purpose of the investigation was to study the circulatory effects of rapid expansions of blood volume in intact unanesthetized dogs and to determine whether Starling's law of the heart is operative under these circumstances. Seven mongrel dogs were studied. An electromagnetic flow meter was placed on the ascending aorta. Changes in left ventricular dimensions were recorded with mercury resistance gages. Left ventricular diastolic and intrapleural pressures were measured simultaneously. The chest was closed and the dogs were permitted to recover from the anesthesia and to breathe spontaneously. Alteration in left ventricular size and the effect of filling pressure were produced by rapid transfusion or bleeding. Left ventricular stroke work, peak values of aortic blood velocity and acceleration, and left ventricular power all increased as left ventricular size and pressures were augmented. At any given filling pressure and ventricular size all these indices of ventricular performance increased during infusion of norepinephrine. These experiments support the view that Starling's law and the ventricular function curve concept are...

Isolated papillary muscles from the cat were exposed in respirimeters to gas mixtures containing carbon dioxide and oxygen. In muscles exposed during the second of three periods to 25 per cent oxygen, the volume of oxygen consumed was reduced by half; the oxygen consumption returned to the control level during a third period, in which the concentration of oxygen was 98 per cent. The work capacity was not significantly altered if the partial pressure of oxygen was low.


Observations were made on the association of polyuria and paroxysmal tachycardia. This relationship occurred in any paroxysmal arrhythmia except nodal rhythm or heart block; the polyuria did not occur if the attacks lasted less than 20 minutes or if the rates were less than 110 per minute. The polyuria most commonly occurred in patients with atrial fibrillation (68 per cent) and least commonly in patients with atrial tachycardia (25 per cent). Acute left ventricular failure appeared to prevent the polyuria. The diuresis usually began 20 to 30 minutes after the onset of the attack and was remarkably constant for each patient; the diuresis was essentially a water diuresis. It was suggested that the polyuria develops because of an increase in the volume of blood in the left atrium.


The heart of an adult moth is a chamberless tubular structure which extends from the thorax to the abdomen. It is composed of striated muscle without conducting tissue. Bioelectric activity was measured in situ; two types of action potentials were recorded. The one from the cephalic region of the heart resembles the action potentials of the vertebrate atrium. The other, obtained from the abdominal region of the heart, has a contour similar to the ventricular action potentials of vertebrates. No histological difference between the two cardiac regions exists. Typical pacemaker potentials occur but could not be topographically localized. Omission from the perfusion fluid of sodium produced no effect on resting and action potentials; omission of potassium caused hyperpolarization, lack of calcium prolonged the repolarization and, conversely, lack of magnesium prolonged the depolarization phase. The major current-carrying ion remains to be identified, but its effectiveness is greatly influenced by the presence of magnesium.


Intravenous injections of a lipopolysaccharide extract of gram-negative bacilli were given to 10 men free from obvious cardiopulmonary disease in order to provoke a pyrogenic response. Four clinical phases followed the injections: prodrome (period of stable temperature); chill (rising temperature); flush (peak temperature); defervescence (falling temperature). No change in monitored cardiovascular functions occurred during phase 1. During phases 2, 3, and 4 there were increases in oxygen consumption, minute and alveolar ventilation, respiratory rate, and heart rate. During phase 3, the cardiac output increased, "total pulmonary resistance" was unchanged, and systemic vascular resistance decreased. These changes in cardiovascular function induced by a pyrogen could be enough to cause cardiac failure in patients with impaired reserve.


The cardiac ganglion of the "Squilla Oratoria" (mantis shrimp) is composed of neurons tightly linked together functionally. The response to an applied stimulus is all-or-none, indicating that excitation spreads very rapidly from one neuron to all the others. Probably the principal usefulness of such a system is the safety factor when a part of it is injured. The present experiments suggest that transmission takes place electrically rather than chemically. The impulse invades the soma from two directions, one from the stimulated side, the other via parallel axons. When the parallel axons are cut, conduction takes place across the soma with a greatly reduced safety factor and is delayed. The electrical potential of the whole
heart is composed of two different elements: a rapid one due to the spike in the nerve trunk and a slow one due to the muscle action potential. The pacemaker is probably located in the rostral part of the ganglion. In fact, the stimulation of this cardiac region produced impulses conducted rostro-caudally like those following spontaneous discharge.

**CUCCI**


This study reports the effect of changes in carotid sinus pulse pressure without a concomitant change in mean arterial blood pressure on the blood levels of catecholamines. "Elasticity" bottles were inserted bilaterally into the common carotid arteries in order to change the magnitude of the pulse pressure in the carotid sinus area without changing the mean arterial blood pressure. It was observed that a marked decrease in the pulse pressure resulted in significant increases in heart rate and in catecholamine plasma levels. It was clear that the exposure of carotid sinus to a decreased pulse pressure per se was sufficient to elicit sympathetic adrenalin stimulation and to increase the catecholamine release in blood vessels. The mechanism and role of this increase in plasma levels of catecholamines were discussed.

**ABBOWD**


This report is concerned with the mechanism of myocardial norepinephrine depletion which has been previously reported following guanethidine and reserpine administration. Sixteen anesthetized open-chest mongrel dogs were studied. Coronary sinus and femoral artery blood was analyzed for norepinephrine (NE). Right ventricular contractile force (Walton-Brodie strain gage), femoral artery pressure, heart rate, and the electrocardiogram were recorded. The right atrial appendage was biopsied and the tissue concentration of NE was assayed. Guanethidine infusion resulted in measurable NE release into coronary sinus blood which persisted for 2 or 3 hours and paralleled the systemic adrenergic effects. NE content of the atrial appendage was reduced by only 24 per cent in 4 hours and to extremely low levels in 24 hours. Thus the major portion of atrial NE depletion occurred after the period of measurable release of NE into the blood. Reserpine infusion resulted in no detectable release of NE into the coronary sinus blood in three of four dogs and a smaller adrenergic systemic response, but the NE content of the atrial appendage was reduced by 65 per cent in 4 hours. The data suggest a different mechanism of myocardial NE depletion for guanethidine than for reserpine.

**HELWIG**


Reserpine or guanethidine prevents the response of effector organs to direct stimulation of postganglion sympathetic nerves. The experiments presently reported compare the mechanisms of the adrenergic blockage induced by these two drugs. The chronotropic response to cardioaccelerator nerve stimulation and norepinephrine (NE) content of left atrial appendage was determined in four control dogs, 25 reserpine treated dogs, and five guanethidine treated dogs. Guanethidine produced complete blockade of the cardiac accelerator response before producing measurable myocardial NE depletion, whereas reserpine produced blockade only after marked reduction of myocardial NE. Infusion of NE did not restore the normal response in either guanethidine or reserpine-treated dogs. These data suggest that the interference with adrenergic transmission produced by guanethidine is independent of changes in the level of stored adrenergic transmitter. The reserpine-induced blockade of adrenergic transmission may ultimately be dependent upon the depletion of adrenergic transmitter, but almost complete depletion of stored adrenergic transmitter must occur before reserpine-induced adrenergic blockade occurs.

**ROENTGENOLOGY**


X-ray examinations of the right hilum of 79 patients with left ventricular disease and 70 control subjects were correlated with left atrial pressures. The normal right hilum has a concave lateral margin where the descending superior pulmonary vein crosses the descending pulmonary artery. In early left ventricular failure, distention of veins of the upper lobe obliterates this normal hilar

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concavity. With further venous distention, this contour is completely effaced and, in severe failure, the greatly dilated superior vein may present a convex shadow. Clearly abnormal hilar shadows and normal contours of the control subjects were easily identified. Twenty-eight per cent of patients with left ventricular insufficiency had minimal changes with diminution of the hilar angle and straightening of the lateral border. Many of these patients had normal resting left atrial pressures. Response of these pressures to exercise was not determined, but these relatively asymptomatic patients had other evidence of definite left ventricular disease. It is suggested that these minimal changes in right hilar contours may be helpful in the roentgen diagnosis of early left ventricular failure.

**Kaltman**


Fifty-eight renograms were obtained in 32 women who were free from cardiovascular and renal diseases. 

There was no apparent disparity between the two kidneys. Competitive inhibition of tubular transport by para-aminohippuric acid resulted in a flattening of the renogram contour. Partial ureteral obstruction produced by an abdominal compression board reversibly increased the renogram parameters. The renogram contour in all of the experiments appeared to be dependent upon the rates of urine flow and tubular transport; sensitivity to changes in urine flow appeared to be too low to permit accurate measurement of flow from the separate kidneys by this technic.

**Kalmansohn**

**SURGERY AND CARDIOVASCULAR DISEASE**


A series of 100 consecutive patients with abdominal aortic aneurysms was analyzed. Other manifestations of atherosclerosis were usually present. These included hypertension in 37 per cent, heart disease in 10 per cent, and both in 16 per cent. The average age was 65. The majority of the patients presented with the complaint of abdominal pain. Evidence of rupture, or expansion was considered an absolute indication for surgery; aneurysms larger than 7 to 8 cm. were removed after preparation. Smaller aneurysms were evaluated carefully. There were 66 nonruptured and 34 ruptured aneurysms with an operative mortality of 15 per cent in the former and 59 per cent in the latter. Seven patients died since surgery and 56 patients 7 years following surgery, are enjoying normal lives.

**Kalmansohn**


One complication of the closure of ventricular septal defects and endocardial cushion defects is surgical heart block. Early operations for the repair of ventricular septal defects resulted in a high incidence, approximately 12 per cent, of complete heart block. Recent studies of the position of the conduction system and improvement of surgical and perfusion technics contributed to reduce the over-all incidence of surgical heart block to an estimated 5 per cent. During the past 36 months the authors have operated upon 175 patients with ventricular septal defects and 21 patients with endocardial cushion defects; only two patients developed permanent complete heart block. The authors' experience confirms previous evidence with regard to the extreme vulnerability of the postero-inferior margin of the ventricular septal defect. Factors other than direct surgical trauma to the conduction system have been implicated in the production of the heart block. Among these are myocardial damage by potassium arrest, severe pulmonary hypertension, and postoperative metabolic acidosis.

**Cucci**


Three patients with severe mitral incompetence resulting from bacterial endocarditis were successfully treated surgically by simple reattachment of the ruptured chordae tendinae or by mitral valvuloplasty with a heart-lung bypass. Two of the three patients probably had valvular changes secondary to rheumatic heart disease prior to the bacterial endocarditis; however, the valve function prior to the infection was not significantly abnormal and therefore did not interfere with the surgical correction. The author concluded that mitral incompetence secondary to bacterial endocarditis probably had a more favorable surgical prognosis than did rheumatic mitral incompetence.

**Karpman**

The report deals with 52 of the 288 patients from a previous series of 1,000 patients subjected to surgery for mitral stenosis and in whom symptoms recurred after at least 1 year of significant improvement. The relative degrees of mitral stenosis and insufficiency were assessed tentatively by clinical methods, and a “corrected diagnosis” was made after hemodynamic investigation and reoperation. For comparison, similar clinical and “corrected” diagnoses were made in 50 patients undergoing initial valvulotomy. Twenty-two of the patients previously operated upon had mitral stenosis alone, 12 had mitral stenosis with mild to moderate insufficiency, and 18 had mitral insufficiency alone. Qualitative estimates of regurgitation by dye curves agreed closely with the subsequent surgical findings. But clinical assessment of the predominant lesion was correct in only 73 per cent of the patients previously operated upon, in contrast to 94 per cent accuracy with new patients. The constellation of mitral stenosis tended to persist although significant insufficiency had supervened, and failure to detect significant or predominant insufficiency was ascribed to these features: absent left ventricular thrust in eight, absent apical systolic murmur in three, failure of electrocardiogram to show left ventricular hypertrophy in 13, and normal left ventricle at fluoroscopy in 12. On the other hand in 5 of these patients with mitral stenosis only, there was a left ventricular thrust, the electrocardiogram indicated left ventricular hypertrophy or combined hypertrophy, and the left ventricle was enlarged fluoroscopically. Pansystolic murmurs were heard in 14 patients with mitral stenosis only. There were four deaths in the 39 reoperated patients. Sixteen of the 22 with stenosis only had “excellent” to “good” results, whereas 11 of the 17 with significant or predominant insufficiency had “fair” to “poor” improvement.

MARCH


Phonocardiographic studies of six patients with Starr-Edwards ball-cage mitral valve prostheses revealed that the interval between the Q wave and the closing of the mitral sound (CVS) and between the aortic second sound (S2) and the opening valve sound (OVS) vary with the preceding R-R interval. Increasing of the Q-CVS interval and decreasing of the aortic S2-CVS interval without a change in the preceding R-R interval suggest an improvement of the cardiac status—a decreased left ventricular end-diastolic pressure.

Kalmansohn

UNCOMMON FORMS OF HEART DISEASE


Cardiac size as determined by the chest x-ray was correlated with electrocardiographic changes in 53 patients with myxedema. Typical electrocardiographic changes of myxedematous heart disease were observed in 27 patients, whereas only 11 had minor electrocardiographic abnormalities, six had evidence of coronary artery disease, two had left ventricular hypertrophy, and two had a pattern of left bundle-branch block; only five patients were found to have normal records. Radiologic evidence for cardiac enlargement was detected in 29 patients and was noted to be present especially in those patients who were older or who had raised systemic blood pressures; fourteen of the patients were found to have typical electrocardiographic evidence of myxedematous heart disease with a normal-sized heart. The authors concluded that patients with established myxedema may have normal-sized hearts despite electrocardiographic evidence of myxedematous heart disease; in addition, enlargement of the heart in myxedematous patients is usually associated with an abnormal electrocardiogram.


The authors describe an intramural fibroma of the heart that caused the sudden death of a 17-month-old child with no previous symptomatology. The heart was twice normal size and the fibroma occupied the musculature of the anterior left ventricle and extended into the interventricular septum. The atria were not involved. Primary tumors of the heart represent 0.15 per cent of 480,000 autopsies. Twenty-seven cases of intramural fibromas of the heart have been published. Tumors involving the atria have been excluded. No sex predisposition was noted. Of these 27 tumors, 21 occurred in children and two caused death in newborns. Sudden death was described in nine of these.
patients and an antemortem diagnosis was made in only two patients. Most intramural fibromas are located in the interventricular septum or in the anterior wall of the left ventricle. The right ventricle is very seldom involved. Histologically they are benign tumors and nonencapsulated. Some authors prefer to call these tumors fibroelastic hamartomas. 

LOPEZ
KARPMAN


The cardiac findings in 75 patients with muscular dystrophy are presented. Of 16 white males with the Duchenne type of muscular dystrophy only four had normal electrocardiograms. The predominant abnormality was right ventricular conduction delay. The electrocardiograms in four patients with facioscapulohumeral dystrophy were all essentially normal. In 26 patients with limb girdle type 3 had a history of heart failure; seven had normal electrocardiograms, whereas 12 showed right ventricular conduction delay. Of the 27 patients with dystrophia myotonica, one third had abnormalities in the electrocardiogram.

KALMANSOHN

VALVULAR HEART DISEASE


Thirty-nine mongrel dogs were studied without thoracotomy. Six animals were used as controls, 18 were studied before and after induction of various degrees of aortic insufficiency (A1), and 15 were studied only after induction of aortic insufficiency. Detection of immediately appearing indicator via a catheter in the left ventricle following its injection about 1 cm. above the aortic valve was found to be a reliable index of the presence or absence of aortic regurgitation. The ratio of the area of the left ventricular curve to the area of the simultaneously recorded femoral artery curve bears a close positive correlation to the severity of aortic insufficiency as estimated by back perfusion of the valve at necropsy.

HELWIG


Forty-five children aged 2 to 16 years had peak systolic pressure gradients measured by catheter-pullback technic before and during the intravenous infusion of 80 mcg per cent isoproterenol solution. An increase in gradient was found in all except two subjects, both of whom had clinically minor lesions. The increase in gradient was generally of larger magnitude in patients whose basal gradients were higher. One patient with surgically treated pulmonic valvular stenosis had a surprisingly large gradient rise from 28 to 84 mm Hg with isoproterenol, suggesting the possible need for reoperation at a later date. It was concluded that isoproterenol infusion provided a safe and effective means of simulating the effects of exercise as an aid to the study of obstructive heart lesions.


The association of valvular and myocardial disease with chronic deforming arthritis was discussed. It was suggested that there was a spectrum ranging from classical rheumatoid arthritis with valvular and myocardial disease to rheumatic heart disease with chronic joint deformity (Jaccoud's arthritis). Ankylosing spondylitis with the associated aortitis was excluded from the spectrum. Rheumatic heart disease was characterized by advanced rheumatoid arthritis with widespread formation of rheumatoid granulomas, high titers of rheumatoid factor, the presence of myocardial disease, and often aortic insufficiency. Patients with Jaccoud's arthritis presented a history of severe recurrent rheumatic fever with advanced rheumatic heart disease and gradual painless deformity limited to the hands.

KALMANSOHN
ROGERS


The authors studied the frequency of atrial fibrillation and the significance of various factors for its occurrence in 617 patients with rheumatic mitral valve disease. There were marked differences between the various types of mitral lesions.
The arrhythmia was present in 17 per cent of 325 cases of pure mitral stenosis, 25 per cent of 129 cases of combined mitral disease with mild insufficiency, and 72 per cent of 98 cases of combined mitral disease with marked insufficiency, its overall frequency being 26 per cent. There was no fibrillation in 65 patients with pure mitral insufficiency, despite considerable atrial dilatation in one fourth of them. Age of the patient and duration of the lesion were dominating factors in all three groups, more so in pure mitral stenosis. The latter was also affected by the degree of stenosis. The size of the left atrium was a significant factor in all three groups, more decisive in combined mitral disease with marked insufficiency. Combined mitral lesions with mild insufficiency were intermediate between pure stenosis and combined lesions with marked insufficiency.

Samartzis


Mitral insufficiency accompanying a tight mitral stenosis usually causes no additional clinical signs or symptoms and does not change the indications for commissurotomy but often leads to a less satisfactory postoperative improvement. Mitral regurgitation accompanying a mild stenosis is more important. Compared to tight stenosis, this combination appears more often in men and after typical rheumatic fever, and leads earlier to impairment of heart function. A marked systolic murmur, systolic left atrial expansion, and roentgenologic or electrocardiographic signs of left ventricular hypertrophy not accounted for otherwise from the best clinical signs, but often even these do not allow definite diagnosis. Micromanometry, selective left ventricular angiography, radioangiography, and dye-dilution do demonstrate regurgitation but do not allow the determination of whether or not the mitral orifice is tight unless possibly they are combined with an exercise test. The presence of a functionally significant mitral insufficiency without tight stenosis is a contraindication to simple commissurotomy and requires an operation with extracorporeal circulation.

Lepeschkin


Twenty-six of 51 patients undergoing mitral commissurotomy had recurrence of symptoms. Eight died, 10 were reoperated on, and eight patients with clinically diagnosed restenosis were not submitted to surgery. Eight patients who had noncalcified mobile valves had satisfactory commissurotomies and no restenosis. Inadequate splitting of the valve and recurrence of symptoms in the other 43 patients were related to the extent of calcification and deformity of the valves noted at surgery. Forty-nine of 51 patients were men. The unusually high incidence of calcification, 66 per cent, in this predominantly male series is unexplained. There was a mean interval of 5 years between operation and recurrence of severe disability. Examination of these patients was clinical, and hemodynamic studies were performed only when considered necessary to confirm a clinical diagnosis of restenosis. Since neither clinical nor cardiac catheterization data are included in this paper, the reasons for deterioration of these patients, attributed to restenosis by the authors, cannot be evaluated.

Kaltman

VASCULAR DISEASE


Venographic studies on normal legs in contracted and relaxed states demonstrated that muscular contraction had an occlusive effect on the communicating veins; a reverse effect took place in limbs with varicose veins.

Kalmansohn


Varicose saphenous veins in 13,352 patients have been treated in a special clinic by an injection-compression technic. The varicosities and incompetent perforating veins were first detected by physical examination and were located by marking the overlying skin. Approximately 0.5 ml. of a 3-per cent solution of sodium tetradecyl was then injected into each of one to three points of incompetence while a 5-cm. segment of vein about each site was isolated by manual compression for 30 seconds. (Subsequent palpation and biopsies have shown complete thrombosis of this segment.) The leg was bandaged from the site of injection to the toes, and this was covered by an elastic stocking—both of which were maintained for 6 weeks while the thrombus in the treated vein thoroughly organized. Review of an unselected group of 760 patients treated over 6 years revealed that 85 per cent had satisfactory results. No
ABSTRACTS

serious complications of this treatment were encountered, and pregnancy and previous deep-vein thrombosis were not regarded as contraindications.

ROGERS


Three personal observations and a review of the literature show that gangrene following a thrombophlebitis is not unusual, and is characterized by edema with no loss of temperature of the extremity, normal or exaggerated arterial pulsation, and later cyanosis. Generalized febrile reaction, pulmonary embolism and shock are especially frequent. Arterial spasm is inconstant and has no major significance in the pathogenesis. Accordingly, surgical relief of the venous obstruction when cyanosis appears in thrombophlebitis is the most satisfactory method of prevention of the gangrene.

LEPESCHKIN


Venous occlusion plethysmography was used to assess the significance of the blood flow in the calf at rest and in response to timed arrest of the circulation in normal individuals and in patients with intermittent claudication of the calf due to obliterative arteriosclerosis. The blood flow in the calf at rest was found to be the same in the normal and ischemic limbs. In several instances after reconstructive surgery, resting flow remained unaltered or was decreased. The reactive hyperemia test was thought to be unsafe for periods beyond 20 minutes. This test demonstrated a marked difference between the normal and diseased circulation. In the latter, there was a negligible increase in peak flow in response to arrest of the circulation to the limb. Further attributes of the reactive hyperemia test in evaluation of ischemia and in controlling therapy are presented. The importance of estimating the relationship between the first minute flow following the onset of reactive hyperemia and the flow during the rest of the hyperemia is discussed.

RAKITA

OTHER SUBJECTS


Extensive studies of the circulation were conducted in approximately 10 normal and 14 hypertensive adults. Synthetic angiotensin I and angiotensin II appeared to have similar potency and action. Intravenously administered angiotensin produced in normal subjects a prompt and sustained rise in systolic and diastolic arterial pressure (the mean pressure increasing an average of 26 to 37 per cent) and a rise in pulmonary arterial pressure (averaging 12 per cent mean) and in pulmonary capillary pressure. The pressor effect subsided 3 to 5 minutes after cessation of the infusion, while alterations in regional blood flows persisted for as long as 30 minutes. Other effects were bradycardia, unchanged cardiac stroke output, increased right atrial pressure, little or no change in heart size, slightly increased skeletal muscle blood flow, decrease in skin flow in comfortable individuals—increase in hand flow in the hot subject, and decrease in liver and kidney blood flows. The action of angiotensin was compared with that of noradrenaline, which, on a weight basis, is one seventh as potent in raising arterial pressure.

ROGERS


A modified direct-current defibrillator (Cardioverter) shock restored sinus rhythm in 20 of 22 patients. Three patients reverted to atrial fibrillation in a brief follow-up period. The indications for this form of treatment were considered tentative and in general consisted of the persistence of the arrhythmia in a symptomatic patient already treated conventionally or, in one who had had an embolus. The technic included pretreatment usually with digitalis but seldom with quinidine or an anticoagulant, premedication with pethidine, anesthesia with thiopentone and occasionally posttreatment with quinidine. No complication was observed clinically, electrocardiographically, by white-cell count, transaminase measurement, or by alteration in sedimentation rate. In 10 patients, the cardiac output usually did not change immediately after conversion, but in seven instances was found to have increased substantially 3 to 16 days later. While the proper place of this therapy was believed not yet established, this early experience emboldened the authors to widen its scope of application.
ABSTRACTS
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