ABSTRACTS

Editor: STANFORD WESSLER, M.D.

Abstracters

FRANCOIS M. ABBoud, M.D., Iowa City
CESARE E. Cucci, M.D., New York
JACQUES DESCOTES, M.D., Lyon, France
DANIEL DEYKIN, M.D., Boston
JOHN HELwig, Jr., M.D., Philadelphia
ROBERT KALMANsohn, M.D., Los Angeles
ALFRED J. KALTMAN, M.D., New York
HAROLD KAPLAN, M.D., Beverly Hills
HERBERT J. KAYDEN, M.D., New York
GEORGE S. KURLAND, M.D., Boston
EUGENE LEFESCHKIN, M.D., Burlington, Vt.

SALVATORE M. SANCETTA, M.D., Cleveland

Domingo Liotta, M.D., Houston
Jose Lopez, M.D., Chicago
Robert J. Luchi, M.D., Philadelphia
Harold W. March, M.D., San Francisco
Robert J. Marshall, M.D., Morgantown, W. V.
Henry N. Neufeld, M.D., Tel Aviv, Israel
Louis Rakita, M.D., Cleveland
Stanley M. Reimer, Ph.D., New York
Wayne R. Rogers, M.D., Portland, Ore.
Lawrence R. Ross, M.D., Salt Lake City
Miltiades Samartzis, M.D., Athens, Greece

PHYSIOLOGY


This study was directed at the question of whether it is the rhythmic pulsations of the atrium or the reduced mean right atrial pressure that increases venous return. Artificial pulsations were produced in the right atrium by having a mechanical pulsator cause rapid inflow of blood to and outflow of blood from the right atrium not synchronously with cardiac action. Mean right atrial pressure was able to be controlled independently of the degree of the pulsations. By this means one could determine whether the pulsations themselves or the changes in mean right atrial pressure had a beneficial or harmful effect on venous return. The phenomenon of rectification is described and it is shown how a pulsation in the right atrial pressure fluctuating positively and negatively (± 20 mm. Hg) about zero pressure and hence resulting in a mean right atrial pressure of zero produces a mean pressure in the veins before entry into the thorax of 6.5 mm. Hg. Similarly it is seen that whereas a nonpulsating, mean, right atrial pressure of −2 mm. Hg results in a venous return of 1,200 ml. per minute, a pressure pulsating cyclically (from +2 to −6 mm. Hg) about the same mean reduced the venous return to 1,030 ml. per minute. The gradual collapse of the great veins when mean right atrial pressure falls to −4 mm. Hg is discussed. It is shown that whereas extreme pulsations have no effect on venous return when the right atrial pressure is very negative, even an instantaneous elevation of right atrial pressure above the critical level of −4 mm. Hg, and especially above 0 mm. Hg, decreases venous return. Increasing the volume of fluid moved per pulse lessened the venous return. As the pulse volumes were increased, the venous return began to decrease at lower and lower mean right atrial pressures. The effects of varying pulse volume and mean right atrial pressure were independent of pulse frequency between 60 and 240 c.p.m. It is concluded that the artificially induced right atrial pressure pulsations had a consistently depressant effect on venous return, whereas even a slight decrease in mean right atrial pressure down to the pressure level at which the veins collapse completely increased venous return markedly.

Fox


A phonocardiographic device utilizing a gating circuit was developed for the beat-to-beat measurement of fetal heart rate. Maternal and fetal heart rates were recorded simultaneously and continuously on paper tapes. The responses of future mothers and their fetuses to various stimuli, e.g., inhalation of 100 per cent and 12 per cent oxygen, intravenous administration of atropine, epinephrine or norepinephrine, cigarette smoking, and a frightening experience (in one instance), were studied. No change in fetal
heart rate was noted after the administration of epinephrine or norepinephrine. Atropine produced a tachycardia in the mother, which was followed in approximately 12 minutes by a fetal tachycardia. This long delay is believed to be due to the time required by the drug to cross the placenta. On the other hand, cigarette smoking produced a maternal tachycardia followed by a fetal tachycardia after a very brief interval. A similar brief interval between the maternal and fetal tachycardia was noted after a frightening experience. In both of the latter instances the fetal tachycardia was believed to be the result of vasomotor changes in the placental bed.

**ABSTRACTS**


Six healthy physicians in the supine seated position were subjected to a total of 80 exposures to 2, 3 1/2, and 5 g. levels of forward acceleration for periods up to 10 minutes. In comparison to 35 control determinations, there was at the higher acceleration levels, a slight increase of the heart output (dye-dilution method) an increase in heart rate, mean aortic pressure (catheter method), and total peripheral resistance; and a decrease in stroke volume. The most dramatic hemodynamic change was an increase in mean right atrial pressure (catheter method) averaging more than 20 mm. of mercury at the onset of exposure to a 5 g. acceleration and gradually declining toward the end of the exposure.

**SANCETTA**


The authors compared in dogs the progression of the activation wave and the contour of the atrial complexes in electrical flutter induced from the sinoatrial area with those of electrical tachycardia and aconitine flutter induced from the same point. The data support the hypothesis that electrical flutter is caused by an ectopic focus located at or near the sinoatrial or atrioventricular node. Two different patterns of atrial activation in electrical flutter are described, which probably correspond to each of the two locations. Both patterns can be obtained in the animal by stimulating a single area of the atrial myocardium. The electrocardiographic records obtained cannot be explained by the theory of a cireos movement.

**KAYDEN**


It has been suggested by others that cardiac arrest following multiple transfusions of banked blood is variously due to the acidosis thus created or to the attendant hyperkalemia. This was tested by measuring isometric myocardial contractions of the turtle (Chrysemys picta) heart, immersed in Howell's solution containing 6 mEq. potassium ion per liter. The pH was varied by the addition of 0.1N hydrochloric acid. With pH ranges from 7.8 to 6.1 no change in contractility was noted, and some variable decrease in heart rate. With extreme acidification, at pH 4.3, a 25 per cent decrease in the strength of contractility was noted, again with slight slowing of the heart rate. It was concluded that cardiac arrest produced in experimental animals by over-transfusion of banked blood is due to causes other than acidosis.

**LEPESCHKIN**


This article is from the Laboratory of Physiology and Pathology of the Heart, of the Moscow
Institute of Normal and Pathological Physiology. When hyperfunction of any organ occurs in healthy individuals as a reaction to physiologic stress, it is usually transient and disappears after the cessation of the stress. Compensatory hyperfunction of the heart caused by destruction of the valves or by persistently high arterial pressure, however, is protracted, for the lesions are irreversible and the continuity of hyperfunction is necessary for life. Animal studies were carried out in 450 rabbits and in 12 dogs by the production of stenosis of the aorta distal to the aortic valve. The study of myocardial function, metabolism, and structure showed that during the period of compensatory hyperfunction the heart passes through three main stages. The first or transient breakdown stage characterized by symptoms of left ventricular failure with pulmonary congestion, hydrothorax, aseites, and death of 20 per cent of the animals. The contractile insufficiency is caused by acute cardiac strain and a deficiency in certain enzymes. There is temporary mobilization of glycogen and creatine phosphate and the resynthesis of adenosine triphosphate (ATP) is by the inefficient anaerobic pathway. The second or protracted stage of relatively stable hyperfunction is characterized by absence of cardiac insufficiency and by arrest of cardiac enlargement. Hypertrophy of muscular fibers, compact disposition of myofibrils, and moderate focal cardiосclerosis are present. The glycogen, creatine phosphate, and ATP content are normal, and myocardial lactic acid may be doubled. The latter phenomenon is ascribed to moderate myocardial hypoxia due to a decrease in the number of coronary capillaries per unit of mass. In the third or protracted stage of progressing cardiосclerosis there is cardiac insufficiency, progressive myocardial fibrosis, focal fatty degeneration, a deficit in deoxyribonucleic acid, a decrease to one half the rate of protein synthesis, and a decrease in ATP level. In acute cardiac insufficiency oxidative phosphorylation becomes insufficient to compensate for the increased loss of ATP brought about by strain. Chronic cardiac insufficiency is a result of a decrease in the capacity of actomyosin to transform the energy of the phosphate bonds of ATP into the kinetic energy of cardiac contractions. The nature of the change in actomyosin is being studied.

KAYDEN


Dogs with surgically induced heart block of 4 to 24 months' duration were studied both under anesthesia and without anesthesia. An electrode catheter was placed in the right ventricle to stimulate the heart. Cardiac output determinations were obtained with indocyanine green. In other dogs, aortic root pressures and cinefluorographic estimates of ventricular volume were recorded. It was found that with heart rates below 60 per minute, stroke volume was maximal and relatively constant, and the cardiac output was largely rate dependent, whereas these relationships did not exist with ventricular rates above 60 beats per minute. Decreasing diastolic ventricular filling with increasing ventricular rates was demonstrated cinefluorographically. The cardiac output decreased at very slow and at very fast ventricular rates. The former was due to rate alone; the latter was attributed to a decreased diastolic filling period and resistance to ventricular distention.

HELWIG


Direct hemodynamic measurements were made in open-chested, anesthetized dogs whose heart actions were electrically paced. Mean left atrial pressure (MLAP) roughly paralleled left ventricular end-diastolic pressure (LVED) at slower heart rates; but, above 160 to 180 beats per minute, MLAP became considerably higher. This observation was related to relatively earlier atrial contraction at higher rates until, at very high rates, much of atrial systole occurred while the mitral valve was closed. This phenomenon was more pronounced at higher ventricular stroke volumes, in which the duration of ventricular systole was increased. When atrial contractility was depressed by stimulation of the distal end of the cut vagus nerve, MLAP was higher for a given level of cardiac output, thus demonstrating the importance of atrial systole in ventricular filling. Stimulation of the isolated left stellate ganglion shortened ventricular contraction, and, therefore, at high heart rates, allowed the atrial beat to fill the ventricle better, resulting in a higher cardiac output per MLAP level. It was suggested that depressed atrial function (failure) might contribute to the elevated venous pressure of congestive heart failure.

ROGERS

Moret, P. R., Pattay, J., and Megevand, R.: Pulmonary Circulation. I. Relationship of Cardiac

Using a bypass procedure, the authors controlled the venous return to the right atrium of anesthetized dogs. The systemic, diastolic, and mean pulmonary artery pressures varied in the same direction as the venous return. The relationship was not linear in that at levels of 110 to 130 ml. per Kg. per minute there was an increase in the slope of the pressure curve. Similar results were obtained when the venous return was decreased. Although no experiments were carried out with the venous return at 0, it is the authors' impression that under these circumstances the pressure in the pulmonary circulation would not reach 0. The relationship of the left atrial pressure was also not linear with the venous return, although the changes in left atrial pressure were a function of the venous return. The pulmonary artery-left atrial pressure gradient also varied with the venous return but, again, this relationship was not linear. It is suggested that at a venous return of 0, a gradient of approximately 6 to 5 mm. would still exist. The pulmonary vascular resistance varied in its response to changes in the venous return. At low levels of venous return, the resistance was markedly elevated. At levels of venous return from 50 to 100 ml. per Kg. per minute the pulmonary vascular resistance decreased to values between 200 to 700 dynes sec. cm. At venous returns above these levels, the resistance again rose. The authors discussed the relationship of the cardiac output to the central pressures and the pulmonary vascular resistance. The nonapplicability of Poiseuille's law to the problem was mentioned. The factors involved in the nonlinear responses of the pressures to the changes in the cardiac output were considered. Other interesting facets of the relationships between the left atrial pressure and the pulmonary-vascular resistance were discussed. The significance of the pulmonary vascular resistance and the concept of the "critical closing pressure" in the pulmonary vascular bed and its relationship to the distribution of the blood flow within the vascular bed were reviewed.

**Rakita**


A method for estimating left-to-right and right-to-left intracardiac shunts from a single arterial dye dilution curve is described. The mathematical formulae and procedures required to extract the data from the curves are presented. This dye method was more sensitive than oximetry. Existence of a significant right-to-left shunt may lead to erroneous results in the evaluation of coexisting left-to-right and right-to-left shunts.

**Rakita**


Studies were made on the biochemical activity of mitochondria and of homogenates obtained from normal hearts and from hearts after experimentally induced failure. The principal types of failure investigated were "chronic" congestive failure and acute failure in the guinea pig. The "chronic" failure was induced in from 2 to 10 days by a partial constriction of the ascending aorta; the acute type of failure was induced by a more severe aortic constriction of a similar type. Various physiologic parameters were studied. The "chronic" animals exhibited tachycardia, elevated right ventricular systolic pressure, and a significant depression of myocardial contractility. In addition, a highly significant increase in cardiac tissue mass was observed. Passive congestion of the liver, spleen, kidneys, and lungs was evident. Mitochondria isolated from the hearts of guinea pigs in "chronic" failure exhibited a significant depression of metabolic activity. Thus, phosphorylation associated with the oxidation of glutamate, succinate, or a-ketoglutarate was uncoupled in mitochondria isolated from the "failed" heart. Cardiac glycosides administered to animals with experimental cardiac failure did not alter the uncoupled state of the mitochondria, although these agents effected a significant improvement in cardiac contractility. The adenosine triphosphatase activity of the "failed" mitochondria was normal. Experiments concerning the localization of the site or sites of uncoupling of oxidative phosphorylation in the "failed" mitochondria revealed that the defect probably resided in the phosphorylation step associated with the electron transfer between cytochrome C to oxygen. Mitochondria isolated from acutely failed guinea pig hearts exhibited a "mild" uncoupling of oxidative phosphorylation manifested as a decrease in responsiveness to a phosphate acceptor system. This was a graded effect, increasing in severity with increasing duration of aortic constriction. On the basis of the present study, it is possible that uncoupling of oxidative phosphorylation in heart mitochondria may play a role in the development of congestive cardiac failure.

**Kayden**

_Circulation, Volume XXVII, March 1962_

Force-velocity relations were studied in the cat papillary muscle. As with skeletal muscle, a characteristic relation has been demonstrated between the velocity of shortening (V) and the force developed (Po). Two generalities have been shown to pertain. First, increasing initial muscle length increases the maximal developed force (Po) without a change in the maximal velocity of shortening (Vmax). Secondly, at any one muscle length, changes in frequency of contraction and chemical environment (increased calcium and norepinephrine) increase Vmax with a variable change in Po. Changes in Vmax thus help to characterize an inotropic intervention (altered contractility). Work and power, at any one muscle length, are functions of afterload, with maxima when the load is approximately 40 per cent of isometric tension. With increasing initial muscle length, the work and power at any one afterload as well as the maximal work and power of the muscle are both increased. At constant initial length, positive inotropic interventions (increased frequency, increased calcium, and norepinephrine) increase the work at any one afterload as well as shift the maximal work potential to a higher afterload. Work performance thus depends on muscle length, the prevailing force-velocity curve, and the afterload at which the muscle is operating.

Kayden


Evidence that people indigenous to the tropics have low forearm blood flows and low systolic blood pressures in relation to their surface and deep-body temperatures prompted this study to determine whether the cardiovascular systems of heat-acclimatized persons are relatively insensitive to increases in body temperature. If the latter were true, the relationship between heart rate and sublingual temperature of man in the tropics might be expected to differ from that reported by Tanner in nonacclimatized subjects. Heart rates and sublingual temperatures were measured in 32 male Asian subjects in Singapore at a mean environmental temperature of 28.9 ± 1.0 C., dry bulb, and 25.6 ± 0.9 C., wet bulb. Heart rates were counted from electrocardiograms and sublingual temperatures were measured by means of a copper-constantan thermocouple. The correlation and regression coefficients for heart rates and sublingual temperatures of the heat-acclimatized subjects in Singapore did not differ significantly from values for those coefficients published by Tanner for young English subjects. Thus no evidence has been obtained in this study for the thesis that acclimatization to heat is associated with a significant change in the sensitivity of the tissues of the cardiovascular system to temperature.

Fox

PULMONARY DISEASE


In 53 experiments pulmonary embolism was produced in dogs by the intravenous injection of a suspension of barium sulfate. The response was characterized by a sudden marked increase in pulmonary arterial and right atrial pressures and a precipitous fall in systemic arterial and pulmonary venous pressures. These changes showed a direct dependence on the degree and suddenness of pulmonary obstruction, but exhibited great individual variability attributed to a corresponding variability in functional adjustments such as coronary and pulmonary vaseroxenostrietion, peripheral vasodilatation, and changes in cardiac rhythm and contractility. After section of the vagi and the spinal cord, the fall of systemic blood pressure was more pronounced and precipitous. An essential role in the production of collapse was played by the heart and its ability to withstand the increased requirements arising from pulmonary obstruction. Rapid death from pulmonary embolism was usually the result of acute cardiac failure, peripheral vasodilatation having only an accessory role.

Lepeschkin


The clinical signs, symptoms, and course along with the electrocardiographic and hemodynamic findings are described in 14 patients with primary pulmonary hypertension. Therapy is discussed. The authors consider this disease entity to be a clear indication for therapeutic abortion. It is of interest that in four of the patients the symptoms started during childhood at ages ranging from 18 months to 12 years.

Rakita
RENAL AND ELECTROLYTE EFFECTS ON THE CIRCULATION


The effect of the calcium ion on two different preparations of the intact heart of the dog was studied. The first preparation permits the measurement of total coronary flow (with the exception of the small Thesbian drainage into the left side of the heart) while the rate of blood flow into the heart is controlled. The second preparation is an isovolumic beating left ventricle and permits the simultaneous measurement of myocardial oxygen consumption and the left ventricular pressure at controlled left ventricular volumes. Calcium, as the 10 per cent gluconate, was rapidly infused into the venous circulation of the dog coronary flow preparation. It was also infused into the aortic circulation perfusing the heart of the isovolumic preparation, in which an otherwise empty, beating left ventricle was filled with a known volume of fluid contained within a slack Latex balloon. In the coronary flow preparation, calcium was found to increase heart rate, leave aortic pressure unchanged, increase the velocity of left ventricular rise, decrease the circumference of the left ventricle, and increase the coronary flow and myocardial oxygen consumption per beat in relation to the existing mean aortic pressure. In the isovolumic preparation calcium increased the peak ventricular pressure at a given balloon volume, but had no effect on the ratio relating myocardial oxygen consumption to heart rate, and left ventricular pressure developed. In both preparations, oxygen extraction was decreased. It is suggested that in addition to the action of calcium on the energetics of the heart and its hemodynamic performance, there is presumably a direct action of calcium on the coronary vasculature that leads to an increased coronary flow.

Kayden


Local effects of major cations on coronary vascular resistance were studied in the beating dog heart. This was accomplished by shunting the blood around the heart and lungs, clamping the arch of the aorta and perfusing arterial blood at a constant rate into the ascending aorta. Perfusion pressure was measured during intra-
coronary infusion of isotonic solutions of NaCl, KCl, CaCl₂, MgCl₂, and MgSO₄ and hypertonic solutions of NaCl and KCl at rates that raised cation concentrations in coronary blood without significantly affecting concentrations generally within the body. Coronary vascular resistance decreased as a function of the infusion rate of isotonic solutions of KCl, MgCl₂, and MgSO₄ and increased as a function of the infusion rate of an isotonic solution of CaCl₂. Isotonic NaCl had no effect. Resistance changes occurred before measurable change in the proportion of a minute spent in electrical systole by the ventricle. These findings, together with those previously reported for the dog foreleg, suggest that the coronary vascular bed is actively dilated by localized slight increase in plasma concentration of potassium or magnesium and actively constricted by localized slight increase in plasma concentration of calcium.

Kayden


Administration of chlorothiazide to rats for 9 weeks produces an increase of intracellular sodium and a decrease of intracellular potassium in skeletal muscle. However, in cardiac muscle, in the wall of mesenteric arterioles, in aortic wall, and in kidney there is no significant alteration in the amount of sodium, potassium, or chloride per unit of dry tissue weight. The water content of heart muscle, skeletal muscle, and kidney is not altered by chlorothiazide. The intracellular concentration of sodium and potassium in heart muscle is likewise unaltered by chlorothiazide. However, chlorothiazide produces a highly significant, 44 per cent, increase in the granularity of the juxtaglomerular cells. The data in general suggest that chlorothiazide decreases the volume of extracellular fluid, but does not reduce the content of intracellular sodium. Extracellular potassium is reduced as well as the potassium inside skeletal muscle fibers. However, the amount of potassium inside cardiac muscle fibers is unchanged by chlorothiazide.

Kayden


The effects of severe calcium deficiency and calcium excess on transmembrane potentials in

Circulation, Volume XXVII, March 1963
isolated frog ventricular strips have been investigated. Resting potential rose about 5 mv above normal during perfusion with three times normal calcium concentration and fell about 4 mv below normal during exposure to calcium-free Clark's solution. Mean overshoot rose about 3 mv during calcium lack, but was unaffected by calcium excess. Maximum depolarization rate increased about 20 per cent during calcium deprivation and fell a similar amount during high calcium perfusion. However, the membrane potential at the moment of maximum depolarization rate was unchanged from normal by either experimental solution. High calcium augmented the "spike" and "plateau" during repolarization, whereas calcium deficiency abolished the spike, producing "hump-backed" action potentials with prolonged membrane reversal. These results are discussed, especially in relation to possible permeability changes of potassium during upstroke of the action potential.

**RHEUMATIC FEVER**


In a group of 250 children with acute rheumatic fever, who were being treated with steroids, diffuse weakness occurred in five. The weakness was of variable severity and tended to involve the proximal musculature more than the distal segments of the limbs. Respiratory and pharyngeal functions were unaffected but the anterior neck muscles were weakened in two patients. Moderately severe cerebral symptoms were noted in one child, which cleared within a month after omission of the steroids. Muscle biopsies revealed changes consistent with a primary myopathy and not those of rheumatic fever myopathy. No instances of weakness occurred in patients treated with ACTH or cortisone. An absolute statement could not be made regarding the relative potency of the various drugs (i.e., prednisone, dexamethasone) used that produced myopathy; however, in all cases the lesion was clinically reversible within a period of months. The authors concluded that primary myopathy was a definite risk in patients receiving steroid therapy.

**KARPMAN**


Data are presented concerning 300 native-born adult Puerto Ricans with rheumatic fever who were admitted to the medical service of one of the charity hospitals over an 8-year period (3.8 per cent of the total number of admissions). The observations with regard to incidence, pathogenesis, clinical picture, and morbidity in this indigent population of a tropical area with no significant climatic alterations were not strikingly different from the manifestations of rheumatic fever and rheumatic heart disease in the temperate zones.

**SAGALL**


The duration of rheumatic fever was measured in 1,169 consecutive hospital admissions over a 7-year period. This report from Irvington House, a hospital and convalescent home for rheumatic children, reviews in detail 76 children whose attacks lasted longer than 223 days. The authors have previously stated that the mean duration of rheumatic fever is 109 ± 57 days. The chronic group were those children whose attack lasted longer than the mean plus twice the standard deviation. In six attacks, the diagnosis of chronic rheumatic fever was incorrect and other diseases were present (systemic lupus erythematosus, endocardial fibroelastosis, infections arthritis, or recurrent rheumatic fever from a new streptococcal infection). In 16 patients the only evidence of rheumatic inflammation beyond 223 days was an elevated erythrocyte sedimentation rate. Follow-up examination 2 to 7 years later in 12 children showed no change in cardiac status and no new rheumatic manifestations. Fourteen patients had chorea as the sole clinical abnormality, appearing 7 months after acute onset. In eight children it had appeared before but extended into "chronic" period, in six children chorea started during the "chronic" period. Follow-up studies in this group indicated a good prognosis and no change in cardiac status. The 40 remaining patients (3 per cent) had other clinical manifestations of rheumatic fever during the chronic period. In 21 patients congestive heart failure was present. It appeared that the frequency of persistence of the attack into a "chronic" period increased with the number of attacks of rheumatic fever and with the presence and severity of heart disease in the initial attacks. Of the 40 patients described as "true
chronic," 13 died, of whom 11 were autopsied. Although Aschoff bodies were present in only one subject, fresh nontuberculous endocarditis was present in three patients and recent pericarditis in another two.

**Kayden**

**ROENTGENOLOGY**


Biplane angiocardiography was used to determine left ventricular volume in different phases of the heart cycle. The contrast material was injected into the pulmonary artery or, by retrograde arterial catheterization, into the left ventricle. The stroke volume in the left ventricle was determined from the maximum and minimum left ventricular volumes, and the minute volumes during angiocardiography were then determined from the stroke volume and cardiac rate. There was fairly good agreement between the stroke volume obtained by the angiocardiographic examination and the Fick method.

**Kalmansohn**


The authors report 11 patients with supravalvular stenoses of the pulmonary arteries diagnosed by selective angiocardiography. The murmur in all patients was systolic in timing; in four patients the murmur was best heard at the right base and transmitted to the axilla and back. One patient had a valvular aortic stenosis in addition to the pulmonary artery stenoses. No continuous murmurs were heard in the absence of a patent ductus arteriosus. The electrocardiograms usually showed right ventricular enlargement unless the disease was minimal, although the findings were influenced by associated lesions. The findings on chest roentgenograms were gradually inconclusive; right ventricular hypertrophy and dilatation may be present. The stenoses themselves were impossible to locate even when compared to the angiograms. In patients with very marked poststenotic dilatations of the peripheral pulmonary arteries, the pulmonary vessels may have the appearance of a left-to-right shunt. Moderate dilatation of the main pulmonary artery may be present. All patients had elevated systolic pressures in the right ventricle, the highest pressures being recorded in patients with combined lesions. In a few patients the diagnosis was suspected by passing the catheter beyond the area of constriction. Selective angiocardiography from the right ventricle or pulmonary artery was successful in all patients.

**Kalmansohn**


In 41 cases of pure mitral stenosis and nine cases of combined stenosis and regurgitation, the state of the pulmonary veins was assessed from roentgenographs taken in the anteroposterior and left oblique projections. The veins of the left lower lobe were identified in an extreme left oblique view in 70 per cent of cases. When the degree of stenosis is mild, and when regurgitation is also present, the veins of the left lower lobe are normal or slightly increased in caliber and the pulmonary arteries appear normal. The increased caliber of the veins, when present, is probably due to increased venous pressure in the lower lobe. With more severe degrees of stenosis, especially when pulmonary hypertension was severe, the veins were narrowed as were the peripheral arteries. Arterial narrowing causes radiolucency of the lower lung fields, which permits the veins to be clearly seen. Venous narrowing may be caused by reduced blood flow secondary to constriction or organic narrowing of the peripheral arteries. The veins of the upper lobe are occasionally normal in mitral stenosis, but more often are dilated. This dilatation is thought to be due to increased flow in the upper lobe secondary to the reduction of flow in the lower lobe, and possibly also to increased pulmonary venous pressure.

**Marshall**


The authors are dissatisfied with the use of conventional cardiac catheters for angiocardiography. In their experience, the pressure required to provide adequate detail of intracardiac structures may lead to rupture of the catheter or of the myocardium. They used polyethylene tubing (1.0, 1.8 to 3.5 mm.) with a cored-screw adapter. In all children and in most adults the tubing is introduced under general anesthesia, and usually via the right internal saphenous vein. For injections into the right ventricle, the tubing is initially molded with a U-bend; it is then introduced by a semi-rigid steel wire as a stilet; when the

*Circulation, Volume XXVII, March 1963*
tip has been advanced to the atrium, the wire is withdrawn and the tube manipulated through the tricuspid valve. On only seven of 124 occasions did the tubing recur during the injection into the right atrium. For left ventriculography, the authors have found the Seldinger technic of percutaneous insertion of a plastic catheter into the femoral artery to give unsatisfactory results in adults and to be technically difficult in children. They introduce their larger polyethylene tubing by open femoral arteriotomy, and employ a Seldinger guide-wire as an aid in manipulation. The left ventricle was entered on all 18 occasions in which this technic was used.

MARSHALL


A 28-year-old man showed decreased physical performance but no signs of cardiac failure; the heart shadow showed bilateral enlargement, while arterial pulsation in the left arm was much greater than in the right arm and especially the legs. Aortography through the left brachial artery showed that the descending aorta was on the right side, and was supplied from the ascending aorta through abdominal branches of the left internal mammary artery and through two arteries that represented a connection between the left brachiocephalic trunk and the descending aorta. The right upper part of the body was supplied through a left brachiocephalic trunk and through the inferior thyroid artery and the thyroid plexus. This case represents atresia of a right aortic arch, bypassed through persisting portions of the fetal circulation.

LEPESCHKIN


The authors have found angiocardiography via a needle inserted by the posterior percutaneous route (Bjorek) into the left atrium useful in assessing patients with pure or predominant mitral stenosis; however, it was of no value in patients with mitral regurgitation. They used retrograde aortic catheterization by the Seldinger technic for left ventriculography, but experienced technical difficulties and some instances of pseudoregurgitation from transient disturbance of valve function due to the effects of the injection. They now use anterior percutaneous puncture of the left ventricle with a needle which has a closed tip and side holes, and report their experience with 80 cases. There were no serious complications. In five instances there was a small pericardial effusion; arrhythmias were transient; accidental injection into the myocardium was surprisingly well tolerated. Cases of mitral regurgitation were subdivided into four grades of severity, depending on the opacity of the left atrium as compared with that of the ventricle, the extent to which the aorta was opacified when the atrium showed a maximal concentration of dye. The paper includes detailed descriptions of the appearance of the normal valve, of various types of valvular deformity including that due to rupture of chordae tendineae, and of "jet lesions" in the atrium. It is emphasized that ventriculography is intended to augment, not to replace, careful clinical assessment.

MARSHALL


Transverse (horizontal) laminography was applied to 181 patients with cardiac or vascular abnormalities and proved of value in the diagnosis and treatment. This was especially true in the case of positional anomalies of the aorta and pulmonary vessels, errors in torsion of the truneus and transposition of the pulmonary veins. Laminography at the level above the diaphragm was especially valuable in demonstrating the size and configuration of the individual cardiac chambers and in showing rotation of the heart about its vertical axis. The plane just above the diaphragm was the most useful location in all types of cardiovascular disease except coarctation of the aorta, where the plane of the pulmonic arch was best, and aortic aneurysm, where the plane of the aortic arch was more useful. In addition to these planes, however, the planes at the height of and below the pulmonary hilus should be also taken in every case. A detailed exposition was made of the characteristic patterns seen in the five planes for every type of cardiovascular abnormality; these were illustrated by numerous laminograms.

LEPESCHKIN


Twenty-one cases of idiopathic myocardial hypertrophy, of which 17 had adequate clinical and roentgenographic data for analysis, were studied. The patients ranged in age from 28 to 57 years with a mean age of 40 and most pre-
sented with a similar clinical and radiologic picture. The typical patient is a relatively young male with congestive heart failure, cardiomegaly, gallop rhythm, and an absence of significant cardiac murmurs. Systemic and pulmonary emboli with no apparent origin are common. On chest x-rays, all the patients showed cardiomegaly always including the left ventricle and frequently including the left atrium and right ventricle. The aorta was frequently normal or hypoplastic, a differential point in distinguishing this syndrome from the large heart in patients with so-called “burnt out” hypertension in whom the aortic knob is invariably large. Angiocardiography, when performed, revealed only marked dilatation of the heart, and cardiac catheterization data were consistent with congestive heart failure. In some patients it was difficult to distinguish pericardial effusion or myocarditis from idiopathic myocardial hypertrophy. The prognosis was poor and at autopsy cardiac hypertrophy and dilatation were found with or without intramural thrombi. The coronary arteries were usually normal and no valvular disease except for dilatation was found.

HELVIG


The vascular anatomy of the hilum of the right lung was studied in 48 normal controls, 33 patients with mitral valve disease, and 45 patients with left ventricular failure. In the controls, the smaller “upper half” of the hilum is formed largely by the upper lobe veins and the larger “lower half” mainly by the descending pulmonary artery. The pulmonary artery was found to be enlarged in those cases of mitral stenosis with pulmonary arterial hypertension. A similar enlargement in over 50 per cent of patients with left ventricular failure suggested the existence of pulmonary artery hypertension. In most patients with either mitral valve disease or left ventricular failure, the lateral border of the hilum was straight or convex due to enlargement of the upper lobe veins. This sign, in addition to the presence of a clearly defined oblique lower border of the superior pulmonary vein, is helpful in assessing hilar changes in diseases associated with pulmonary venous hypertension.

MARSHALL


The diameters of specific veins were measured at specific sites in tomograms of the right lung in normal control subjects, patients with mitral stenosis, and patients with left ventricular failure. Cuts were made at 1 cm. intervals between 7 and 13 cm. from table top. The diameters of a pair of veins in the upper lobe and of a pair in the lower lobe were measured and a ratio (“venous index”) was established of upper to lower lobe venous size. Independent measurements by three observers were usually in good agreement. In 50 controls, the venous index was between 0.6 and 1.0; similar values were obtained from pulmonary phlebograms in 15 controls. Of 36 patients with mitral valve disease, 29 had a venous index greater than 1.0; that is, the lower lobe veins were relatively narrow. All nine patients with wedge pressures of 20 mm. Hg or more had an index greater than 1.0, while all three patients with a wedge pressure less than 10 mm. Hg had an index of less than 1.0. In the third group there were 42 patients with left ventricular failure of variable severity due to hypertension, aortic valve disease, ischemic heart disease, and myocardopathy; 18 had a venous index greater than 1.0. Changes were less striking and less consistent than in the group with mitral stenosis. These findings are of interest in view of the demonstration by Doyle et al. of constriction of the lower lobe arteries at autopsy in patients with mitral stenosis and by Dolly and West of a relative reduction in blood flow through the lower lobe in patients with left ventricular failure. The occurrence of diminished blood flow in the lower lobes in both these conditions may be the result of a homeostatic mechanism that protects the patient from the occurrence of pulmonary edema.

MARSHALL


Because previous roentgenologic techniques have been notably unsuccessful in the demonstration of coronary artery calcification, and because of previous observations by other investigators that postmortem coronary calcification indicates significant arteriosclerotic coronary disease, the authors employed cinefluorography of the heart. A group of predominantly white male patients from a Veterans Administration Hospital were studied. A control group of 60 patients without clinical heart disease in various age groups and a heart disease group of 630 consecutive patients...
who had known or suspected heart disease, mostly valvular in type, were studied. A 5-inch Westinghouse image intensifier was employed and six film series were taken on each patient. The authors found that they could visualize coronary calcification in 25 per cent of the control group and also in 25 per cent of the heart disease group. However, 58 per cent of the 150 patients with coronary calcification were found to have arteriosclerotic heart disease when the cinefluorographic study was correlated with clinical and autopsy material, whereas only 30 per cent of the comparable group had clinical coronary disease without coronary calcification. The authors conclude by stating that although cinefluorographic evidence of coronary calcium by itself appears to be unreliable for predicting the presence of symptomatic ischemic heart disease, it may prove to be a good indicator of potential ischemic myocardial disease.

Hedwig


The renal blood flow was recorded in cats during renal arteriography with two contrast media, sodium acetazolamid (Triul) and sodium diatrizoate (Hypaque). Sodium acetazolamid produced no change or a slight increase in renal blood flow in single small doses. When larger doses were injected, a decrease in blood flow occurred and finally almost ceased with repeated injections. Postmortem examination showed a marked increase in the weight of the injected kidney, which was edematous and cyanotic. In comparable doses, sodium diatrizoate produced no effects on the renal circulation.

Kalnansohn


The authors used an image intensifier with a 16-cm. field and a 16-mm. cine camera taking 25 frames per second for 10 to 20 seconds. The total radiation dose did not exceed 300 m.a. at 100 to 120 kv. The films were viewed in motion, and individual frames were magnified and studied as stills. The main indications for cine films were study of the dynamics of diseased valves, assessment of ventricular hypertrophy and of pulmonary infundibular and subaortic stenosis, and the delineation of shunts. Cineangiocardiology does not replace the classical methods of angiocardiography but is a useful adjunct.

Marshall


In interstitial, extra-alveolar pulmonary edema the lymphatic vessels of the pulmonary parenchyma are distended and filled with edema fluid. The widened interlobular septa appear as fine peripheral lines (Kerley's B-lines), particularly in the costodiaphragmatic angles. In the upper lobes they form fine central lines radiating from the hilum (A-lines). Edema of the subpleural tissues leads also to thickening of the interlobar tissues; in the region of the minor fissure this appears as an ill-defined horizontal line. Edema of the perivascular tissues results in lack of definition of the vascular shadows and clumping of the perihilar region. Interstitial pulmonary edema can be caused by defective lymph drainage, by increase in pulmonary venous and capillary pressure, and by increased capillary permeability. The septal lines seen in mitral stenosis indicate interstitial edema resulting from a marked increase of the pulmonary venous pressure. As interstitial edema cannot be recognized clinically, radiologic examination is particularly important for its diagnosis.

Lepeschkin


Coronary arteriography was carried out in anesthetized dogs by means of a supravalvular injection of 10 to 15 ml of 90 per cent diatrizoate-M during cardiac asystole induced by metacholine chloride. In a control group of animals conventional roentgenographic technic was employed by means of an x-ray tube having a 1.5-mm. focal spot and a 40-inch target-object distance. In this group the dog's chest lay on the x-ray table close to the film. In the study group, the x-ray tube had a 0.3-mm. focal spot, and the animal lay on a radiolucent table midway between the tube and the film. Fine roentgenographic magnification of the coronary tree was thereby achieved so that vessels 100 μ or less in diameter could be well seen. This method had the disadvantage of requiring a half-second exposure time and hence cardiac arrest was mandatory. Photographic magnification of the radiograms of the control dogs did not provide sufficient definition to improve the visualization of the smaller vessels.

Rogers

In 27 patients with mitral disease, direct or retrograde injection of contrast medium into the left ventricle enabled an accurate diagnosis and determination of the degree of mitral insufficiency on the basis of the reflux of the medium into the left atrium. If reflux opacification of the left atrium was confined to the region near the ostium, the regurgitation was slight; if it extended throughout the atrium, but was definitely less intense than that of the ventricle, regurgitation was moderate; if it became more intense than in the ventricle during systole, regurgitation was severe. If atrial filling occurred only when the entire thoracic aorta was filled, the regurgitation was slight; if complete filling occurred when half the descending aorta was filled, it was moderate; and if it took place when the entire, the first half, the initial portion, or no portion of the ascending aorta was filled, the regurgitation was severe. The degree of regurgitation was also proportional to the number of ventricular contractions required to clear the contrast medium from the left atrium and ventricle, but this also depended on the heart rate, the force of ventricular and atrial contraction, and the presence of aortic valvular lesions. This was also true of the left ventricular volume at the end of diastole; however, the left ventricular pressure at this time was more nearly parallel to the degree of mitral regurgitation. The thickness of the left ventricular wall and the diameter of the left compared with the right coronary artery, as measured during levoangiography, also reflected the degree of mitral regurgitation as did the degree of systolic decrease in left atrial volume in the presence of sinus rhythm. Contrast filling of the pulmonary veins and entry of the catheter into the left atrium were additional signs of marked regurgitation. In addition to an evaluation of the degree of mitral insufficiency, selective levoangiography gave information concerning the anatomic configuration of the valvular defect; it could demonstrate predominant reflux at the posteroomedial or anterolateral commissure or shrinkage of the mitral valve leaflets.

LEPESCHKIN


Tricuspid stenosis is not so rare as was once thought. The incidence varies from 5 to 14 per cent in large series of autopsied cases of rheumatic heart disease. The authors selected 20 patients with tricuspid stenosis after the diagnosis had been confirmed by cardiac catheterization and studied the chest films and angiocardiograms (in nine patients). They conclude that a prominent right atrial bulge on the overpenetrated frontal chest film is the most reliable sign of right atrial enlargement. Angiography is very useful in demonstrating prolonged opacification of a large right atrium as well as in excluding other conditions, such as pericardial effusions, pericardial cysts and tumors, and congenital anomalies, which may mimic a large right atrium.

HELGIG


The valvular system of the azygos vein, situated in the middle or posterior third of the azygos arch, produced characteristic images during angiocardiographic studies. The radiologic visibility of the valvular system of the azygos vein in the anteroposterior projection appeared to constitute an important sign of prolonged stasis in the right heart due mainly to tricuspid stenosis. However, the sign was not pathognomonic of high pressure in the right atrium. The finding was not present in 14 of 20 patients with tricuspid stenosis.

KALMANSOHN


Partial filling of the common iliac artery on the affected side with the formation of a very faint pocket of contrast material over the course of the affected artery was noted in three patients with aortic dissections. This has been termed the "cul-de-sac sign" and is considered pathognomonic of aortic dissection.

Rakita
ABSTRACTS
STANFORD WESSLER

Circulation. 1963;27:463-474
doi: 10.1161/01.CIR.27.3.463

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/27/3/463.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/