ABSTRACTS

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ATHEROSCLEROSIS


The energy to protein (E/P) ratio of the diet had a significant effect on serum cholesterol levels of rats. Corn oil in the diet was found to depress serum cholesterol levels in diets of high E/P ratio but not in diets of low ratios. A low ratio depressed serum levels regardless of the type of fat in the diet. It is suggested that the E/P ratio may represent the unknown factor in the equation of workers who have tried to relate the effects of dietary fats to serum cholesterol levels.

Aviado

CONGENITAL ANOMALIES


Three cases of congenital absence of a main branch of the pulmonary artery are added to the 40 cases recorded in the literature. In 1 patient, it was associated with cystic bronchiectasis of the same lung. In the second, an atrial septal defect was associated with absence of the left pulmonary artery. In the third, absence of the right pulmonary artery was associated with the Eisenmenger complex and a right pulmonary artery arising from the ascending aorta. When this anomaly was unassociated with bronchiectasis or intra-cardiac defects, the patient was usually asymptomatic. Typically, there was a lag in expansion and diminution of breath sounds in the lung with the absent pulmonary artery. Roentgenogram revealed this lung to be small with fine vascular markings, whereas the opposite lung was overdistended and well vascularized, and displaced the mediastinum. Angiocardiography was diagnostic. With bronchiectasis, there may be further pulmonary parenchymal disease leading to cough and a finding of rales. Diagnosis of absence of pulmonary artery when there are septal shunts is more difficult. Such patients are often ill with recurrent infections, dyspnea, and cyanosis. Conventional x-ray helps little; angiocardiography is diagnostic. Cardiac catheterization may be needed to localize the shunt. Differentiation of the isolated type from the complicated type is significant because of therapeutic and prognostic implications.

Kerald


This report deals with the unusual occurrence of atrial septal defect in a mother and 2 of her children. A third child had died of congenital heart disease—probably also atrial septal defect. The diagnosis in each of the patients was established by cardiac catheterization and was confirmed in 1 by an operation to close the defect. There is also a review of the literature dealing with the incidence of familial occurrence of congenital heart disease and the reports of families in which 2 or more instances of identical congeni-
tal heart disease had been present. The author believes that modern diagnostic methods that permit more accurate diagnosis and the routine registration of the family histories of patients with congenital heart disease will provide more adequate material for the study of the hereditary factor in congenital heart disease.

**BROTHERS**


The case records of 5 patients with congenital aneurysm of the aortic sinuses are presented in detail. Cardiac catheterization and thoracic angiography were performed on each patient. The latter procedure was of particular diagnostic value. Two patients had a communication from the aorta into the right heart. One of these had lived for 7 years without progression of symptoms. The other had symptoms of rapidly increasing right heart failure and died during attempted surgical intervention. Two other patients had regurgitation into the left ventricle with a hemodynamic picture resembling aortic incompetence. The fifth patient had no demonstrable abnormality of the circulation and only moderate symptoms. A review of the literature to determine the cause of death in congenital aneurysm of the aortic sinuses disclosed that death can be caused by rupture into 1 of the heart chambers, by compression of the bundle of His, by impairment of aortic valvular function, and by bacterial endocarditis. At the present time, the authors think that surgical repair of aneurysms of the sinuses of Valsalva should be considered only in the small group of patients in whom an aneurysm has ruptured into the right atrium, provided a considerable left-to-right shunt is present, there are no signs of a complicating interventricular septal defect, and there is no endocarditis, syphilis, or arteriosclerosis.

**BROTHERS**


A 7 year old boy presented the usual clinical and anatomic features of tetralogy of Fallot with the unique exception that the electrocardiogram repeatedly showed anomalous atrioventricular excitation. Rapid supraventricular tachycardia occurred as a cardiac catheter was placed in the right atrium. Open surgical correction of the defects, aided by total circulatory bypass, was followed by repeated cardiac arrest and death in 18 hours. Autopsy also showed endocardial sclerosis over the ventricular septum, which was considered possibly to be related to the electrocardiographic anomaly.

**ROGERS**


The forty-eighth instance of congenital mitral stenosis is reported. A 17 months old infant developed heart failure, and the basic disorder was indicated by the roentgenologic finding of a markedly enlarged left atrium. An attempted valvuloplasty by a closed technic was unsuccessful. Autopsy showed a contracted and focally calcified mitral valve that possibly was attributable to an associated fibroelastosis. The findings in this patient are discussed in relation to those of others previously reported.

**ROGERS**

**CORONARY ARTERY DISEASE**


Of 760 consecutive autopsies of cardiac patients, 212 disclosed major coronary sclerosis, and 70 of these patients had shown angina pectoris spontaneously or on exertion. In these 70 hearts, 78 of the major coronary branches (right, left common and anterior, and left circumflex) showed stenosis of 50 per cent to 90 per cent of the lumen, and 116 showed complete or incomplete occlusion; this corresponded to 2.15 occlusions per heart. Myocardial lesions were absent in 15, relatively minor in 29, and major in 26 hearts, which showed a total of 30 myocardial infarctions. Coronary and myocardial lesions had greatest incidence in patients with a very short evolution of angina (several days to 1 month), in those with angina of long duration, not affected by treatment or becoming worse in spite of it, and in those with status anginosus. They were more numerous in spontaneous angina than in angina of effort.

**LEPSCHERK**


The hospital records of all patients having a clear-cut acute myocardial infarction during a 1 year period ending in June 1955 were reviewed. The 66 patients who received anticoagulants (mainly Dicumarol) had the same mortality rate—47 per cent—as the 198 not so treated. The 2
groups may not have been closely comparable; for example, 88 per cent of the treated patients were "poor risk" while 74 per cent of the untreated ones were so designated. In the treated group, 1 instance of major and 4 of minor hemorrhage were encountered, suggesting that excessive anticoagulant effect did not often occur. The present findings are compared with those of private hospital patients who generally fare considerably better.

ROGERS


Iproniazid (Marsilid) recommended by Cesaroni for the treatment of angina was used in 30 patients. In every patient, the symptoms improved and tolerance to stress increased. In the majority of patients with hypotension, the blood pressure was reduced. However, electrocardiographic alterations attributable to ischemia remained unchanged. Side effects were frequent and consisted in orthostatic hypotension, bradycardia, perspiration, constipation, urinary difficulties, rigidity of spontaneous muscle movement, muscle tremor, and somnolence.

PICK


In 100 patients with myocardial infarction followed for 6 months to 5 years, the ballistocardiogram reflected the clinical condition much better than the electrocardiogram. In the 5 patients who died and in 6 who developed heart failure, the ballistocardiogram showed type III and IV. In the 35 patients who developed angina pectoris it usually showed type III, with type II appearing when the complaints subsided. In some patients a more abnormal ballistocardiogram was a sign of approaching death, even when the clinical condition seemed to indicate improvement. More than half the patients were able to return to their work, and in these patients both the ballistocardiogram and the electrocardiogram usually showed marked improvement. An interesting observation concerned the development of pointed upright T waves in 2 cases of anterior and 2 of posterior infarction; this pattern seems to have a good prognosis.

LEFESCHKIN


Four cases are reported of men with arteriosclerotic heart disease who experienced fatal attacks of coronary insufficiency soon after the ingestion of dye administered orally in preparation for gallbladder x-ray studies. In every case pain of coronary artery origin, either noted for the first time or recurrent, had been present 10 to 30 days before the test was performed, implying a new occlusion or myocardial infarction or both. These cases point out the fact that in patients with recent symptoms of coronary artery disease cholecystography may be a hazardous procedure and should not be performed without positive indications. The possibility of a vasovagal reflex as the mechanism of acute coronary insufficiency in these cases is raised and on this basis it is suggested that when cholecystography must be done in such patients premedication with atropine be employed.

SAGALL


Iproniazid or Isoniazid was employed in the treatment of 120 patients with angina pectoris and 12 patients with intermittent claudication and impending gangrene of the lower extremities. In one third of the patients with angina pectoris who were treated with Iproniazid and in one sixth of the patients treated with Isoniazid the attacks of angina subsided entirely or almost entirely after 3 to 7 days. In an additional similar number the frequency, intensity, and duration of attacks diminished although attacks of pain still occurred. These beneficial effects in 95 per cent of the patients lasted for as long as the drug was maintained. In the remaining 5 per cent pain recurred but was of less intensity and duration. The effect on pain due to intermittent claudication was less dramatic. Neither drug was found to have any effect on the natural course of angina pectoris or intermittent claudication. Side effects consisting of faintness, weakness, paresthesias, nervousness, syncope, impotence, or muscular twitchings were reported by two thirds of the patients, occurring in most cases after the third week of treatment. Side effects were more frequent and more intense in the older patients. The author concludes that these drugs are highly effective in the prevention of pain due to ischemia and presents details concerning the dosage, ways of administration, indications, and contraindications.

SAGALL
Vagal nerves on the coronary sinus flow and on cardiac metabolism.

The effect of the vagi and of the sympathetic nerves on the coronary sinus flow and on the oxygen uptake of the heart stimulated to increased work was investigated with methods previously reported. Section of the vagi led to disappearance of the increased coronary flow and oxygen consumption obtained in the intact heart. Sympathectomy had no effect on these parameters; the influence of the left vagus was greater than that of the right. It was concluded that a vagal reflex, presumably initiated by baroreceptors located in the cardiac chambers, was stimulated; the efferent pathway would be primarily constituted by coronary vasodilator fibers; it appeared that adrenergic fibers were not an important constituent of the efferent pathway. The modifications of coronary flow secondary to elevation of coronary sinus pressure were also studied. Gradual increase in coronary sinus pressure produced little changes in the intact or sympathectomized animal; when the vagi were severed, a marked decline in blood flow and also in oxygen uptake was noted. Sudden interruption of the venous flow caused a transitory decline in coronary flow, followed by an increase; after vagotomy, a more marked decrease in flow was noted, not followed by a reverse effect. These observations are interpreted as evidence of a vagal reflex initiated by pressure receptors in the coronary sinus and having a vagal vasodilator efferent pathway.

Calabresi


The nerve supply of the major thoracic and upper abdominal structures is reviewed, and the mechanisms and clinical appreciation of pain production therefrom are mentioned. Diagrams of the segmental innervation of the skin and of the cardiac plexus and a sample history form for chest pain evaluation are depicted.

Rogers


Twenty-six white and 26 Negro hospital patients between the ages of 20 and 47 and without evidence of cardiovascular disease were studied on a 3,000 calorie house diet containing 150 Gm. of fat. The studies were performed in the morning following a 10 hour fast. The subjects were given 100 ml. of 10 per cent cream per square meter of body surface area and hourly plasma specimens were taken. No other food was allowed during the 6 hour observation period. The plasma was separated by centrifugation at 2,500 r.p.m. for 15 minutes, and optical densities were determined using a Beckman DU spectrophotometer at a wave length of 650 mμ. Despite the fact that the incidence of proved myocardial infarction in Negroes is 45 per cent of that in Caucasians, in the area from which the studied subjects were taken, there was no difference in the degree of alimentary lipemia or in the number of individual abnormal responses found. The authors conclude that these data are not consistent with the view that intensified alimentary lipemia is a significant predisposing etiologic factor in coronary artery disease.

Maxwell


A review of 30 patients who experienced cardiac or cerebral infarction during the 30 days following operation or accidental trauma suggests a biological difference between these 2 processes. Sixty-three per cent of the myocardial infarctions occurred during the operation or within the first 3 postoperative or injury days. All but 23 per cent occurred within a week. On the other hand, the cerebrovascular accidents occurred at random. Preoperatively, two-thirds of the patients in this study had definite clinical evidence of cardiovascular disease. The type of surgery and anesthesia did not seem to bear on the incidence of these complications. However, hypotension did occur in 53 per cent of the patients during surgery and the authors stress this as the most important factor in reduced coronary blood flow. Since atheroslerotic coronary arteries cannot dilate appreciably, the reduction in blood flow associated with the hypotensive state undoubtedly is a major factor in the progression of ischemia. Unlike the heart, during adequate general anesthesia, the oxygen demands of the brain are reduced so that an absolute oxygen deficit rarely occurs. Therefore, a cerebral infarction, when it occurs, is usually not directly related to the surgery or anesthesia. The avoidance of anoxia, hypotension, and increased cardiac work load should prevent many myocardial infarctions. The biological and clinical factors underlying cerebrovascular accidents differ in not being specifically related to the period of trauma. Therefore, attempted prevention requires the empirical application of such measures as adequate oxy-

Of 760 consecutive autopsies in patients with heart disease, a coronary occlusion or a stenosis resulting in at least a 50 per cent reduction of the lumen was found in 211 hearts. In 193 of these a total of 517 occlusions was found: 211 in the left common and anterior descending branches, 167 in the right circumflex branch and 139 in the left. The occlusion was situated in a peripheral section of the artery in 60, in an intermediate section in 165, and in a proximal section in 292, and was caused by thrombosis in 236 and by arteriosclerosis in 281 instances. The average number of infarctions in each heart increased continuously from 0.66 in cases with 1, to 1.40 in those with 6 or more occlusions. Coronary stenosis of less than 50 per cent did not cause any myocardial lesions while 1 exceeding 50 per cent caused them only in part of the cases.

Lepeschkin

ELECTROCARDIOGRAPHY, VECTORCARDIOGRAPHY, BALLISTOCARDIOGRAPHY, AND OTHER GRAPHIC TECHNICS


The reliability and clinical usefulness of 2 phonocardiographic time relationships were tested in 49 patients with mitral valvular disease in whom the Q-1 (beginning of QRS to first heart sound) and 2 OS (second heart sound to opening snap) were compared with data obtained from left heart catheterization and at mitral valvuloplasty. The mean Q-1 interval was prolonged in the entire group with mitral valvular disease compared to that in a group of 23 control patients, but there was considerable overlap. There was little correlation between Q-1 and size of the mitral valve, the end-diastolic pressure gradient across the mitral valve, or the left atrial mean pressure. The interval became shorter in 19 of 21 patients following mitral valvuloplasty. An opening snap was demonstrable in 31 patients. Its absence was associated with extensive calcification of the valve with obliteration of leaflet mobility, predominant regurgitation, or minimal mitral stenosis. A 2 OS of 0.09 second or shorter indicated moderate or severe stenosis, but intervals longer than 0.09 second allowed no conclusion about the degree of stenosis. The 2 OS showed significant positive correlation with left atrial mean pressure and less correlation with the mean diastolic pressure gradient across the mitral valve. The 20S interval was lengthened in 10 of 20 patients following valvuloplasty. The phonocardiogram was of limited usefulness in the evaluation of mitral valvular disease.

Kurland


The electrocardiographic findings obtained by multiple epicardial direct unipolar leads in 55 patients are reported; in 1 patient it was possible to verify the electrode placement at autopsy. The electrocardiographic technic used is given. Many leads were obtained from the surface of the left ventricle; only a few from the surface of the right ventricle. On the anterior epicardial surface, in the normal human heart the intrinsic deflection appeared first over the right ventricle, than over paraseptal areas of both chambers, then over the free right ventricular wall, and finally over the left ventricle; the time sequence of activation of the surfaces of the 2 ventricles overlapped, however. A delay over the right ventricle was noted in patients with increased right ventricular load. No correlation was found between the electrocardiographic contour and the thickness of the subjacent myocardium. RS, rS, qRS, or qR patterns were obtained over different points of the left ventricle; there was however an orderly progression of the initial deflection over serial points of the left ventricle.

Calabresi


The electrocardiograms in 158 patients with right bundle-branch block were compared with the findings of right ventricular hypertrophy at autopsy. The electrocardiograms were classified according to the method of Lepeschkin: 78 belonged to type I, 29 to type II, and 51 to type III: no examples of types IV and V were found.
In 51 of the 78 cases of type I, right ventricular hypertrophy, pure or with dilatation, was found; in 16 the hypertrophy was bilateral, in 6 it was limited to the left ventricle, and in 5 there was no hypertrophy. Of the cases of type II, 7 showed right ventricular hypertrophy; in 7 the hypertrophy was bilateral, in 12 it was limited to the left ventricle; in 3 there was no hypertrophy. Right ventricular hypertrophy was found in 28 cases of type III; in 18 there was bilateral or left ventricular hypertrophy; no hypertrophy was found in 5. It was noted that a marked delay of the intrinsicoid deflection correlated well with right ventricular hypertrophy; this was noted also for a wide area of $S_{1}$, resulting from its voltage, but even more from its duration.

Calabresi


The intervals Q-I sound and the II sound-opening snap were measured in 57 patients with mitral stenosis in direct-writing records of the electrocardiogram and of the logarithmic phonocardiogram. In 9 patients the measurements were repeated after commissurotomy. Fifty normal subjects were studied as controls. In some individuals measurements were obtained also during the Azoulay legs-lifting test. In the normal subjects the Q-I sound interval never exceeded 0.05 second; its duration was a direct function of the cardiac rate. In all patients with mitral stenosis this interval was longer than 0.06 second, and in inverse relation to the cardiac rate; in most patients it became shorter after commissurotomy. The II sound-opening snap interval was directly proportional to the R-R interval; it was prolonged after commissurotomy. The Azoulay test had no demonstrable effect on these measurements.

Calabresi


Right axis deviation is frequently observed in patients with diseases causing increased load limited to the left ventricle. The hypothesis is proposed that this electrocardiographic pattern results from delayed activation of the posterior juxtaepicardial left ventricular musculature. To test this hypothesis a localized lesion was produced in dogs by curettage of the endocardial and subendocardial tissue, including the posterior arborizations of the left bundle-branch. The electrocardiographic changes were more evident in hearts in the horizontal position. Because of a late vector involving the posterior wall of the left ventricle and directed anteriorly and inferiorly, deviation of the electric axis to the right was produced. The electrocardiographic changes produced in these experiments were compared with those due to right bundle-branch block and also to posterior displacement of the apex of hearts in the horizontal position. The electrocardiographic changes resulting from similar lesions of the anterior arborizations of the left bundle were also described.

Calabresi


The fundamentals of the vectorial interpretation of the electrocardiogram and the objections to it are briefly discussed. An interpretation based on non-euclidian geometry is sketched; the electric moment is represented by a tensor rather than by the vector of classic electrocardiography. It is concluded, however, that, although the vector interpretation does not fully satisfy the requirements of reproducing each of the scalar electrocardiograms currently obtained and other criteria, the best vectorial systems of leads provide a satisfactory method of analysis of the electrocardiogram.

Calabresi


The characteristics of the electrocardiographic and vectorcardiographic changes in old and fresh myocardial infarctions obtained by a reconstruction method previously described by the author are presented in detail. The changes found by this method closely resembled those reported by previous investigators. In general, RS-T elevations in fresh myocardial infarction corresponded to Q phenomena and reduced R waves in old myocardial infarction, and RS-T depressions in fresh infarction corresponded to augmentation of R waves in old myocardial infarction. The typical changes in the vectorcardiogram comprised displacement of the QRS loops away from the infarcted areas in old infarction and displacement of the RS-T junction directed toward the in-
Lepeschkin


A patient with Wolff-Parkinson-White syndrome is presented emphasizing the fact that anomalous atrioventricular excitation may produce Q, S-T, and T changes which simulate those of myocardial infarction. Since the preexcitation phenomenon may also conceal the abnormal Q waves of myocardial necrosis in arteriosclerotic heart disease, normalization of the QRS-complex conduction in this syndrome is essential for the electrocardiographic diagnosis or exclusion of myocardial infarction. Various methods for normalizing excitation are discussed and the effects of atropine, exercise, and quinidine in this regard in this patient are illustrated. The author also points out that after normalization of conduction, the QRS complex remains the most reliable index of infarction and arteriosclerotic heart disease in this patient because the T-wave changes for a number of reasons are not reliable indices of coronary atherosclerosis.

Sagall


Phonoaudiograms of all patients with aortic stenosis observed by the authors showed increased amplitude of the systolic murmur after a long postextrasystolic pause, or after long pauses in absolute arrhythmia due to atrial fibrillation. This was especially well seen in patients who also had aortic regurgitation, and can be explained by the observation that the aortic pressure was much lower after a long than after a short pause, thus leading to a greater pressure gradient across the aortic valve during the following systole. In the illustrated case the systolic aortic pressure was slightly lower after a long pause. On the contrary, all patients with mitral insufficiency showed little or no change in the intensity of the systolic murmur after long pauses. This can be explained by the observation that the left atrial pressure was approximately the same after a long pause as after a short one. The different behavior after long pauses is an important clue in the differential diagnosis of aortic and mitral systolic murmurs. In 2 patients the systolic murmur was accentuated after a long pause at the base, but not at the apex, and in these patients clinical or anatomic study showed both aortic stenosis and mitral insufficiency.

Sagall


A patient with valvular pulmonary stenosis and normal position of the heart developed inversion of the T waves in leads I, II and V₆₆, 6 weeks after valvotomy as a result of aseptic pericarditis; the P waves at times became inverted in leads I and V₆₆ without appreciable change in the P-R interval, as a result of an atrial ectopic rhythm or an atrial conduction disturbance. A second patient with congenital heart disease but normal heart position showed similar P-wave changes, but the P-R interval was short whenever the P wave was inverted; the T wave remained diphasic or inverted in lead I. If only the tracings with inverted P waves in lead I were available, the pattern would have been indistinguishable from that of mirror-image dextrocardia.

Sagall


A unipolar lead from a needle insulated except for its point showed a Q-S deflection with only minimal S-T elevation when the point was in the cavity of the left ventricle and the inner two thirds of the wall; an R wave and marked S-T elevation appeared only in the external one third. A lead from a uninsulated needle showed marked S-T elevation, Q-S deflection only when the needle point was in the cavity, and gradual increase of the R wave as the point was withdrawn toward the epicardial surface. With the point in the cavity, the amplitude of the Q-S deflection was

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greatest in the apical region, least in the posterior wall. Areas of complete myocardial necrosis extending from the epicardial surface almost to the endocardial surface were produced by systematic infiltration with 95 per cent carbolic acid. Leads from the center of these areas showed marked elevation of the S-T segment but Q waves appeared in only 12 of 32 experiments; these Q waves did not exceed 3 mm. Ligation of coronary vessels supplying the apex did lead to the appearance of Q waves, especially in leads from the lateral portion of the cyanotic area. Cooling of the apex caused these Q waves, as well as normal Q waves, to become smaller (and also increased S-T elevation), while right bundle-branch block increased them. It is concluded that a necrotic region of ventricular wall cannot be compared to an electric "hole" in this wall, as its conductivity is smaller than that of a noninsulated needle electrode. Leads from the surface of this lesion reflect not only the cavity potential, but also that of normal selections of this wall surrounding them.

LEPESCHKIN


In 2 patients who had no other cardiac lesions ventricular tachycardia was initiated by atrial flutter with 1:1 conduction and left bundle-branch block. In the first of these, sinus rhythm was restored by quinidine while in the second atrial flutter appeared during the course of treatment of atrial fibrillation with quinidine. When the latter was discontinued, 2:1 atrioventricular block appeared; sinus rhythm was reestablished subsequently by digitalis. The third patient showed "arborization block"; paroxysmal tachycardia in this patient was initiated by single ventricular extrasystoles of the same configuration as that of the wide QRS complexes during tachycardia, and independent P waves could be seen superimposed on the ventricular complexes. This patient died suddenly after leaving the hospital.

LEPESCHKIN


After a review of the literature, 7 personal cases are presented, of which 4 are of interest. A patient with alkalosis showed moderately low T and tall U waves at a serum potassium level of 3.2, but increased slurring of QTs and huge U waves almost completely fused with T waves at a level of 2.2 mEq per L. Another patient with traumatic anuria showed very low P, pointed T, prolonged P-R, deep S waves in all leads, and a QRS duration of 0.13 second with a serum potassium of 8.5 and a magnesium of 3 mEq per L. After treatment with the artificial kidney potassium became 5.5 and magnesium 1.6 mEq per L and the electrocardiogram became completely normal. A third patient with sarcoidosis and a serum calcium of 6.3 mEq per L showed very short T waves with terminal inversion in leads V₁, through V₆, separated by a long isoelectric interval from normally shaped U waves, which appeared at the usual time for the heart rate. In another patient hyperkalemia and hypocalcemia caused the S-T segment to become prolonged but the T wave was short and pointed. It is concluded that the electrocardiogram represents a sensitive index of potassium and calcium imbalances, but it is probably influenced by the concentration gradient across the cell membrane rather than by the absolute concentration; extracellular fluid may influence this gradient by an action similar to that of cation exchange resins. However, because of the multitude of other factors that may affect it, the electrocardiogram must be interpreted with great caution.

LEPESCHKIN


The use of hypertonic saline in the detection of right-to-left shunting is described in 21 patients with congenital heart disease. The advantages over the Evans-blue technic are the unlimited number of determinations that can be made and the lack of interference with oximetric determination. The method suffers the same limitation as any indicator dilution technic in that only the presence but not the location of the shunt can be detected.

AVIADO


Serial electrocardiograms of 100 patients with anemia of at least 3 months duration and without other cardiovascular disease were studied. Abnormalities were found in 85 cases. In order of decreasing frequency of incidence these electrocardiographic abnormalities included inversion of T waves, depression of S-T, P changes, prolonged Q-T interval, left ventricular hypertrophy, left ventricular strain, atrial premature beats, ventricular premature beats, prolonged P-R intervals, U-wave changes, atrial tachycardia, and hypopotassemia. Persistence of abnormalities de-
spite cure of the anemia was seen in 36 cases and was found to have no relation to the age of the patient. In 28 of these, cardiac enlargement persisted. The abnormalities were found more frequently in the female patients. The incidence and nature of the electrocardiographic abnormalities seemed to be closely related to the hemoglobin level and the heart size on admission. The higher incidence of electrocardiographic abnormalities in this series was believed to be due to the greater intensity of the anemia in this group. Several mechanisms of production of the electrocardiographic abnormalities were suggested: temporary coronary insufficiency without actual cardiac damage, subendocardial infarction and reversible cardiac changes, persistent and probably irreversible cardiac changes, congestive heart failure per se, temporary myocardial fatigue due to tachycardia, electrolyte imbalance resulting from diarrhea, and probable abnormal myocardial thiamine metabolism.

Sagall


Of 11 patients with small defects (less than 1 cm. diameter, shunt less than 3 liters per minute and normal pulmonary arterial pressure) the electrocardiogram was practically normal in 10, but showed deep and slightly wide S waves in leads II and IIII; one patient showed splintered QRS complexes with deep Q waves in leads I and V_{4-6}. Of 7 patients with medium defects (1 to 1.5 cm., pulmonary pressure exceeding 40 mm.) 2 showed normal findings, 4 showed deep and wide S waves in leads I through III and qR or rSR deflections with tall R waves in V_{1-2} (right activation d-leaf) and 3 showed tall R waves in V_{4-6}. Pointed P waves in leads II and III were found in 2 patients. Of 37 patients with large defects and nearly equal pressures in both ventricles, 10 showed right activation delay and 16 a right ventricular hypertrophy pattern, which was accompanied by QS deflection in leads I through III in 3 patients. Incomplete right bundle-branch block was seen in leads V_{5-6} in 1, and a normal finding in 2 patients. P waves were normal in 20, elevated in leads II and III in 10, in 1 through III in 3, and showed the “mitral” pattern in 3 patients. A systolic murmur of diamond or band shape usually had a maximum at the origin of the fourth left rib and was loudest in small defects, where it could be the only sign. A split first sound was found in one third of the patients.

Lepeschkin


Two hundred patients with typical angina appearing only on effort and lasting less than 15 minutes, with a blood pressure less than 160/90 mm. Hg, and free of all other cardiac conditions or drugs that could affect the electrocardiogram, were studied. The pain was retrosternal in 178 and showed radiation (usually brachial, cervical, or dorsal) in 10 patients. Nitroglycerin had a good effect in 52 of 61 patients. A definitely abnormal electrocardiogram was found in 28 per cent; it was most common in patients who had symptoms for less than 1 month or more than 5 years and in those with cardiac enlargement. It included old myocardial infarction patterns (15), bundle-branch block (13), terminal inversion of the T wave (26), and left ventricular hypertrophy (2 cases). Minor abnormalities were found in 41 per cent, especially in women and in persons with a symptom duration of 1 to 5 years; they included low T waves (23) with concave S-T segments (9), peaked, symmetric T waves (17), absent Q waves in leads V_{5-7} (13), an angle between the QRS complex and T wave exceeding 40° (15), and prolonged Q-T intervals (6 cases). Completely normal electrocardiograms were found in 31 per cent; these were least common when the duration of symptoms was less than 1 year and most common when it was 1 to 6 months and the heart of normal size. Of 38 patients submitted to the exercise test, 25 showed an abnormal electocardiographic response, 4 a normal response with pain, and 9 a normal response without pain.

Lepeschkin


Electrocardiographic study of 342 patients with infarction showed that in anterior infarction the terminal vector of QRS was deviated to the left in 68 per cent, while in posterior infarction it was normal or deviated to the right in 88 per cent. Of 48 patients where electrocardiograms were available before and after infarction, those with anterior infarction showed left axis deviation of this vector previous to infarction in 66 per cent and after infarction in 84 per cent while those with posterior infarction showed none or right axis deviation previous to infarction in 75 per cent and after infarction in 82 per cent. The changes in the direction of the terminal component are attributed to a conduction disturbance or the boundary of the necrotic area.

Lepeschkin

As vectorcardiography did not prove suitable as a routine method of study, "sectorcardiography" was developed as a rapid and adequately exact method of designation for the spatial cardiace vectors from the standard 12 lead electrocardiogram. In this method, the direction of the highest vectors during P, Q, R, S-T, and T in the frontal plane is subdivided into 12 sectors, according to the direction of these waves in the 6 standard and unipolar limb leads. The direction of these vectors in the horizontal plane is subdivided into 4 sectors, according to the direction of the waves in leads V1 and V4. A more exact subdivision in this plane is superfluous because of the errors caused by the eccentric position of the heart. The application of the method is illustrated with several examples.

Lepeschkin


In 40 normal persons and 41 patients without heart disease leads V3r and V4r showed positive waves of QRS that did not exceed 0.5 mv. and were always smaller than the negative waves if these exceeded 0.5 mv. An rSR' pattern appeared in V3r, in 9 per cent and in V4r, in 10 per cent. The direction of the T waves in these leads was very variable. In several patients with right ventricular or biventricular hypertrophy R or R' in these leads (especially in V4r) exceeded the normal limits while other leads showed no signs of right ventricular hypertrophy. The dorsal lead V8, taken 2 interspaces lower than the conventional level, showed much less variability than the same lead at the conventional level. Persons with normal hearts never showed negative T waves or Q waves deeper than one third of R or wider than 0.04 second in this lead. These patterns may appear in this lead in posterior myocardial infarction even if leads III and aVp are not definitely abnormal. However, inversion of the T wave in the lower lead V8 may also appear in left bundle-branch block or in digitalis effect.

Lepeschkin


The electrocardiographic findings in 70 patients with rheumatic valvular defects are reported. Twenty of these patients had pure or predominant mitral stenosis, verified at surgery; 20 had apparently pure mitral regurgitation; 10 had double mitral defects with predominant insufficiency; 20 were patients with pure aortic regurgitation. In pure mitral stenosis the mean axis of the P wave is directed to the left and posteriorly; the P wave in V1 may be negative; it was of long duration, 0.12 second or more, in 30 per cent of the cases; these changes have never been seen in pure mitral regurgitation. Tall P waves in the standard leads, bifid and diphasic P waves in V1 were also more common in mitral stenosis than in regurgitation. In mitral stenosis there was right axis deviation of the QRS complex in the frontal plane and deflection to the right and forward in the horizontal plane; therefore the mean solid axis was directed to the right, downward, and anteriorly. The T axis was directed to the left and upward in the frontal plane, to the left and posteriorly in the horizontal plane; hence the solid axis was directed upward, to the left and backward. These findings were of greater significance when the QRS and the T axes were discordant. The transition zone was usually displaced to the left. These changes resulted from hypertrophy and dilatation of the right ventricle, clockwise rotation of the heart, and incomplete right bundle-branch block. In aortic regurgitation the QRS mean axis was directed to the left, upward, and posteriorly; the T axis was directed downward, anteriorly, and to the left. The transition zone was commonly displaced to the right. This was due to hypertrophy and dilatation of the left ventricle and also to counterclockwise rotation and incomplete left bundle-branch block. In pure or predominant mitral regurgitation the electrocardiographic findings varied between these extremes. The Q wave was frequently absent in the left precordial leads in mitral stenosis; this was interpreted as due to the greater participation of the right ventricle in the anterior surface of the heart and also to the "apex" and the left contour of the heart. The Q wave was instead frequently seen in V2-4 in aortic regurgitation and also in pure mitral regurgitation due to greater participation of the left ventricle in the anterior surface of the heart.

Calabresi


Data are presented concerning simultaneously obtained right ventricular endocardial and precordial leads in 50 patients who were studied during cardiac catheterization. This group consisted of 8 normal patients, 6 with pure aortic
ABSTRACTS


The fetal heart rate during labor and delivery was studied by electronic methods employing fetal and maternal electrocardiograms recorded simultaneously by a preamplifier and a cathode ray oscilloscope for careful positioning of the electrodes to obtain a good fetal electrocardiogram. It was found that the fetal heart rate determined throughout contractions provided more accurate information than the average rate determined between contractions. Fetal bradycardia during contractions with the cervix dilated 4 to 8 cm. is normal in vertex presentations. Fetal embarrassment was indicated by bradycardia with contractions at less than 4 cm. or more than 8 cm. dilatation in vertex or breech presentations. Bradycardia persisting between contractions is a late manifestation of fetal distress with serious damage to the fetus. With normal breech and most vertex presentations, fetal heart rate does not slow during contractions. In the minority of vertex presentations, bradycardia may occur with contractions with normal rate restored 10 to 15 seconds later. In these cases, fetal bradycardia is probably related to an increase in intracranial pressure, which in turn is related to the degree of cervical dilatation. It is probable that changes in the instantaneous fetal heart rate represent a sensitive index of fetal distress.

SHUMAN


In 60 patients operated on for mitral commissurotomy, 8 to 126 direct epicardial leads from various ventricular locations were obtained after opening of the pericardium. In each patient, the precordium had been explored before surgery by multiple unipolar leads and charted in an "electric map" of the chest. The precise location of the cardiac chambers in relation to the chest wall was verified in 28 instances with the help of frontal angioelectrocardiograms. Comparison of the 2 lead systems revealed considerable morphologic dissimilarities between direct epicardial leads and the corresponding precordial series. The discrepancy was particularly evident in cases with right ventricular strain. The results of this study failed to confirm Wilson's concept of similarity of epicardial and precordial leads as well as the equivalence of intrinsic and intrinsicoid deflections.

Pick


Electrocardiograms were obtained in 5 patients during prodromal stages of myocardial infarction. In all 5 patients the electrocardiogram was abnormal and showed alterations of the ST-T complexes. According to current concepts, these were interpreted as ischemic alterations involving in 2 patients the subendocardial layers, in 1 the posterior wall and in 2, the anterior wall. In 1 of the latter, the pattern changed to a pattern of anterior wall necrosis after the development of the typical clinical features of myocardial infarction.

Pick


A study on 30 infants under 10 days of age is presented. These infants were studied by means of serial electrocardiograms making primary use of unipolar lead B1. The infants were divided into several groups; all were normal at birth from physical examination. Several groups served as controls, and 1 group of infants was given periodically 10 per cent and 100 per cent
oxygen by mask, and another group was given epinephrine and acetylcholine. The electrocardiographic changes were well recorded. Alteration of the T wave in B1 took place over a 5 day period and in the older infants administration of epinephrine produced changes in the T wave of B1 similar to those found in young infants who normally had pulmonary hypertension. No significant change in the tracing was found by the administration of 10 or 100 per cent oxygen. Administration of acetylcholine to younger infants produced no significant changes in the pattern. It is concluded that it is difficult to make any positive statements regarding the nature of pulmonary hypertension in normal newborn infants from the electrocardiographic tracings alone.

Harvey


During an investigation of the cardiovascular system of Australian athletes, the author noted the frequent occurrence of an electrocardiographic abnormality consisting in splintering and widening of the terminal part of QRS, best seen in lead V1 or lead B of a special lead system reported previously. Invariably this was observed in middle distance runners and was most marked in 2 outstanding athletes 1 of whom later established the 1 mile world record. This electrocardiographic variety is attributed by the authors to a delay in activation of some parts of the basal portion of the left ventricular myocardium engendered by an altered distribution of the terminal ramifications of the conduction system. such a change in the sequence of ventricular activation can, in the author's opinion, enhance to a significant degree the pumping mechanism of the left ventricle; and thus increase cardiac efficiency under stress.

Pick

PHARMACOLOGY


In dogs anesthetized with phenobarbital, chlorpromazine in doses from 1 to 10 mg. per Kg. of body weight induced a transitory fall in blood pressure that was not influenced by vagotomy or by autonomic drugs; it was suggested that this effect resulted from direct action of the drug upon the heart or blood vessels; tachyarrhythmias and elevation of the T wave in the electrocardiogram, however, were abolished by vagotomy or atropinization. A single large dose of chlorpromazine (10 mg. per Kg.) caused a marked fall in blood pressure with transient bradycardia and inversion of the T wave: vagotomy or atropinization did not prevent these changes. No toxic cumulative action was noted: following prolonged administration of the drug, the animals died in a shoeklike state due to the persistent hypotension. Chlorpromazine in small doses antagonized the pressor effect of norepinephrine; the pressor effect of epinephrine was inverted. This inversion was not obtained however in cats similarly treated in which the brain had been destroyed; because of this, a central nervous system effect or a reflex mechanism was suggested. Chlorpromazine in small doses also prevented the cardiac arrhythmias and fibrillation following injections of epinephrine during chloroform inhalation; even in much higher doses the drug did not prevent the arrhythmias produced by pressor pituitary extract or by ouabain. It was suggested that the antagonism to chloroform-epinephrine arrhythmias was not a direct effect of chlorpromazine on the heart, but possibly involved a reflex or central nervous system action.

Calabresi


The mechanism involved in the cardiovascular effects of rauwolscine were studied in dogs and cats. It was found to be a short-acting, reversible type of adrenergic blocking agent. It produced reversal of the pressor response to epinephrine and reduction of the pressor response to levartrenol in both the dog and cat. Doses of rauwolscine that produced epinephrine reversal of the blood pressure did not reduce the pressor response to bilateral carotid occlusion. Much larger doses of rauwolscine were needed to inhibit contraction of the nictitating membrane than to produce epinephrine reversal. The drug exerted a peripheral vasodilator effect on both the innervated and denervated hind limb of the dog. It produced tachypnea consistently in the dog but not in the cat. The effects of rauwolscine on the heart rate were not marked. The cat showed no significant change in rate even during hypotension. The dog showed a transient bradycardia on occasion.

Rinzler

ABSTRACTS

In 13 dog heart-lung preparations it was observed that small doses of chlorpromazine had no effect, higher doses produced transient depression of the cardiac output and still larger doses produced cardiac arrest. These effects were not irreversible and could be antagonized by epinephrine. Small doses of chlorpromazine did not inhibit the cardiac effects of acetylcholine, and even high doses only diminished these effects. In the isolated and perfused rabbit heart chlorpromazine induced depression of heart contractions and coronary dilatation; the stimulating action of epinephrine or norepinephrine was not completely blocked even by large doses. In the isolated perfused frog heart the negative inotropic effect of chlorpromazine was also observed; this effect could not be prevented by epinephrine or norepinephrine; it could be transitorily overcome by the simultaneous perfusion of ouabain, but the ultimate toxic effect of ouabain was not prevented. It was concluded that chlorpromazine exerted no specific anti-adrenergic action on the dog, rabbit, or frog heart, but had a depressant effect on the myocardium and conduction system, and also on the musculature of the coronary vessels.


Three patients with Stokes-Adams attacks who failed to respond to customary measures were treated with intramuscular corticotrophin. All improved strikingly within a few hours. The mechanisms of improvement are unknown although in these 3 patients, age 35 to 45, the possibility of an underlying inflammation of the bundle of His was postulated.


The augmentation of the renal effect of meraluride by acetazolamide was studied in beagles. Acetazolamide, given at 0 time and at 12 hours, augmented the effect of meraluride when given at 24 hours and promoted the renal excretion of sodium, chloride, and water. This augmentation appeared to be due to prior excretion of carbonic acid following acetazolamide, which resulted in a metabolic acidosis that could be reflected in acidosis of the renal cells. This could occur in the presence of normal plasma chloride concentration. When the 2 drugs are given simultaneously, chloride excretion may be less than that following meraluride alone. For maximal chloruretic effects, it is advised that the 2 agents not be given together. The most effective regimen for a 48 hour net sodium and chloride removal is administration of acetazolamide on the first day and of meraluride on the second day.


This report deals with a systematic study of the cardiovascular effects of narcotine hydrochloride with some comparison to those of related compounds. It produces systemic vasodepression in dogs which is incompletely blocked by diphenhydramine and is not affected by atropinization, midcervical vagotomy or carotid sinus and body denervation. Narcotine in the intact dog proved a relatively potent vasodilator with a moderately long duration of action. It increased coronary and femoral blood flow. Tachyphylaxis to the vasodepressive effect of narcotine could be produced following intravenous administration. Tachyphylaxis to the systemic hypotensive action of papaverine hydrochloride, ethaverine hydrochloride, or dioxylinephosphate did not occur. Narcotine produced bronchoconstriction. These effects of narcotine were attributed to liberation of histamine.


Chlorisondamine chloride was used as a model for ganglionie-blocking agents to observe its effects on the response to the drug of different species of test animals, the presence and absence of barbiturate anesthesia, and the type of experimental hypertension. It was found that the blood pressure of the unanesthetized normotensive monkey and rabbit showed a definite and sustained fall in systolic and diastolic levels after intravenous administration of the drug. On the other hand, the blood pressure of the dog and rat under the same conditions did not respond with a persistent fall. In contrast, normotensive dogs, after pentobarbital, showed a fall in systolic and diastolic pressures associated with a relative bradycardia following the ganglionie-blocking agent administration; rats, after Dial-Urethane, likewise showed a fall in blood pressure following
injection of chlorisondamine chloride. The dog, in the malignant phase of renal hypertension, responded with a sustained systolic and diastolic decline following injection of the ganglionic-blocking agent. Studies of the cardiac output, mean blood pressure, peripheral resistance, and cardiac volume indicated that the barbiturates interfere with compensatory mechanisms that normally function in the normotensive dog and rat during ganglionic blockade and that these compensatory mechanisms appeared to be less predominant in the monkey and rabbit.

Rinzler


The origin of norepinephrine which was found in the walls of arteries is not definitely known. It has been suggested that it comes out steadily from the sympathetic nerve fibers. The authors offer evidence that the accumulation of norepinephrine can be dispersed by the action of reserpine in the course of several hours. When this occurs, nicotine and acetylcholine, which are usually vasoconstrictive, lose this action on the blood vessel involved. It might be that this action of nicotine, namely the release of norepinephrine from the vessel, accounts for the pain that occurs when a patient with thromboangiitis obliterans smokes. On the basis of their experiments, the authors suggest that reserpine might be of value in peripheral vasoconstrictive disease states. In this respect, the use of reserpine is limited by its effect on the central nervous system.

Krause


A 60 year old woman who had taken tablets of digitalis leaf for many years developed itching and a skin rash consisting in an exanthema, at first papular, later bullous in appearance, associated with eosinophilia. Idiosynrneres could be demonstrated to injection of a number of commercial digitalis preparations, including acetyl digitoxin, ecdilanid, and digoxin, but skin tests were negative. Antihistaminic drug failed to control the allergic reaction.

Pick

Physiology


There was a satisfactory reproductibility of the measurements of cardiac output in 167 unselected cases. The median error of 2 separate determinations using the Fick principle within a 15 minute time period was 8.6 per cent. The potential error due to phasic variations of blood oxygen content was of no major importance in the determination of cardiac output. In spite of the complexity of the procedures of cardiac catheterization, a satisfactory steady state occurred in the majority of patients.

Aviado


This report deals with the presence of norepinephrine and its congeners chiefly in splenic and lung vessels. Bovine splenic arteries and veins contain about 0.3 to 0.4 µg of norepinephrine per gram. Bovine pulmonary vessels with a diameter of 2 mm. and above contain amounts of catechol amines similar to those of splenic vessels. Lung vessels and other vessels from the dog, however, contain larger amounts of catechol amines than those from bovine sources. There is a differential distribution of amines even within the lung, for large bovine lung vessels contain 10 times more norepinephrine than peripheral lung tissue. Chromatographic separation of catechol substances in splenic and pulmonary vessels indicate the presence of dihydroxyphenylacetic acid, dihydroxymandelic acid, dopamine and norepinephrine which are similar for the 2 kinds of vessels but differ greatly from the relative and absolute amounts found in lung tissue.

Rinzler


Pulmonary ventilation, tidal volume, alveolar carbon dioxide tension, respiratory exchange, and heart rate in response to exercise of different intensities have been measured in 5 men, age 70 to 70 years, and compared to a group of students age 19 to 26. The older group showed a higher total ventilation for any given load attained by a higher respiratory frequency with a somewhat lower mean tidal volume and a little larger physiologic dead space. Alveolar carbon dioxide pressure was the same in the 2 groups. The difference in dead space diminished with increasing rate of work. The old men also showed a higher exercise heart rate at an oxygen uptake around 1500 ml. per minute due to a slow rise in rate between the end of the first half minute and the end of the fourth minute.

Kurland

The hydrogen ion concentration, oxygen saturation, and carbon dioxide tension of the arterial blood were investigated in 55 patients with cardiac dyspnea and, for comparison, in 55 patients with dyspnea due to chronic pulmonary disease. The majority of patients with pulmonary dyspnea showed reduced oxygen saturation, increased carbon dioxide tension, and a decrease in pH. Patients with cardiac dyspnea exhibited normal or slightly reduced oxygen saturation, normal or reduced carbon dioxide tension and a pH either normal or shifted toward the alkaline side. In cardiac dyspnea there was no significant difference in oxygen saturation and carbon dioxide tension between patients with mild or severe dyspnea at rest except in those with an arterial oxygen saturation greater than 93 per cent. In this latter group carbon dioxide tension was significantly lower in those with severe rather than mild dyspnea at rest. In patients with pulmonary dyspnea the oxygen saturation was significantly lower and the carbon dioxide tension significantly higher in the presence of severe dyspnea as opposed to mild dyspnea at rest. The dyspnea that occurs in congestive heart failure cannot be said to be due exclusively to changes in the blood chemical studies that were done. The authors conclude that cardiac dyspnea is probably due to alterations in the pulmonary arterial pressure.

SAGALL


In 71 patients with pure pulmonary stenosis, right atrial and ventricular pressure at the beginning of the QRS complex of the electrocardiogram was found to increase with the maximal right ventricular systolic pressure. A similar behavior was found for the amplitude of the atrial contraction wave in the right atrial pressure curve. The mean diastolic pressure showed no elevation unless there were signs of right ventricular failure. On the contrary, in 28 patients with atrial septal defects there was no definite relation between the increase in right ventricular output and the right atrial pressure at the end of diastole or the amplitude of the atrial contraction wave.

LEPESCHKIN


Dye-dilution curves were obtained in 4 dogs following injection at different sites and levels of flow and volume in order to show that, in normal animals, the anatomy of the circulation between injection and sampling sites was a determinant of the contour of the dye curve. Volume between injection and sampling sites was varied by altering the injection site, which increased the volume and the anatomic path length, and by bleeding, which altered the volume without changing the path length. When dye was injected into the pulmonary artery, the curves had a larger variance, an earlier appearance time, and a lower peak concentration for a given flow and volume than was observed following injection into the inferior vena cava. Reduction in mean flow and volume by bleeding did not alter the equations relating variance of the curves to flow and volume.

KURLAND

Experiments in dogs suggest that the increase in cardiac output following infusion of dextran solution is related to the anemia produced thereby and not to the expansion of whole blood volume or to the increase in cardiac filling pressure. This hypothesis is based on the observation that hypervolemia without anemia does not cause an increase in output, whereas hypervolemia with anemia increases output. The ultimate mechanism for the increase in output has not yet been identified but the most attractive one is the release of a cardiac stimulant humoral agent by anemia.

AVIADO


The authors reviewed the literature dealing with experimental and clinical observations on the relationship of neurovegetative stimuli and atrial flutter and fibrillation. Usually vagal stimulation predisposes to, or triggers the onset of these disorders of rhythm, but on rare occasions, with special conditions of reactivity of the myocardium, increased vagal tone may terminate the atrial arrhythmia. Two clinical examples are presented in which paroxysms of atrial fibrillation could be induced by parasympathetic stimulation. This was demonstrated in records revealing consistently slowing of the sinus rate prior to the onset of fibrillation, multiplication of atrial premature systoles and short bursts of atrial fibrillation under carotid sinus pressure, abolition of fibrillation subsequent to atropine injection and appearance and disappearance of fibrillation independent of changes in posture.

PICK


Aortic constriction in rats caused an immediate decrease in cardiac output. After 1 week, the hypertrophied heart maintained a nearly normal hemodynamic situation. The hypertrophied heart was not only enlarged but was characterized by an increase in "reserve force," i.e., the maximum output induced by infusion. The actual stimulus for the hypertrophy was not revealed by the experiments.

AVIADO


Extrasystoles appearing during the ejection phase of the preceding systole, as evidenced in the pulmonary artery pressure pulse, caused a rise in pressure superimposed on this systole and resulted in an increase in its amplitude and duration. Extrasystoles appearing during the phase of relaxation produced a rise in pressure of a greatly reduced amplitude and duration; showed an increased period of mechanical latency usually not accompanied by ejection into the great vessels. Extrasystoles appearing during the filling period were characterized by prolongation of the latent period and shortening of ejection. The postextrasystolic beat showed decreased latency period and increased duration and amplitude of contraction; these changes may be present also in the subsequent beat. No difference was found between atrial and ventricular extrasystoles. In atrial fibrillation the ventricular diastolic baseline was smooth, while in atrial flutter it showed deviations coinciding with the flutter waves.

LEPESCHKIN


It is calculated that the total resistance to flow is greater in the capillaries than in the arterioles, but this would not explain the fact that the decrease in mean arterial pressure is greater in arterioles than in the capillaries. Observation of blood flow in the ocular fundus shows that pulsation of the arterioles is largely transmitted to the venules that accompany them. This transmission could have great importance in facilitating blood flow in these veins, and would also account for the considerable pressure gradient in the arterioles. The transmission of arterial pulse to the veins would also result in an immediate increase of venous return whenever there is a sudden rise in heart output.

LEPESCHKIN


The injection of sodium salicylate (100 mg. per Kg.) consistently produced a prompt increase in heart contractile force in anesthetized dogs. The effect, however, was moderate if compared with typical stimulant drugs such as digitalis, xanthines, and sympathomimetic amines. There is little reason to consider that this limited increase in heart force has any significant clinical importance beyond the fact that this may be taken as
an indication that the usual doses of salicylates probably do not seriously depress myocardial contractility.

**Aviado**

**RENAL AND ELECTROLYTE EFFECTS ON THE CIRCULATION**


The renal response to postural changes on the tilt table was studied in 17 patients in chronic congestive heart failure. In normal individuals the acute adjustment to the standing position included a decrease in the renal plasma flow in the glomerular filtration rate and in the urinary rate of excretion of sodium and water. Patients with a mild degree of congestive failure showed a response similar to that of normal individuals. As congestive failure became more marked this orthostatic response was reduced and in certain patients with severe heart failure there was essentially no response to the tilt position.

**Sagall**


Renal biopsies were performed 5 to 17 days after the onset of illness in 15 patients with acute glomerulonephritis, and the clinical features were compared with the structural changes. In general, there was a broad measure of agreement regarding severity between the clinical and histologic features. Proteinuria and hematuria indicated structural lesions of the glomerular tufts, and hypertension was in general also related to glomerular disease. Changes in the tufts are accompanied by decreased creatinine clearance, which appeared to be a more reliable indication of structural abnormality than the blood urea. Impairment of the ability to concentrate was an indication of microscopically evident damage in the distal tubules. A sedimentation rate greater than 55 mm. in 1 hour was good evidence of widespread renal damage with interstitial and tubular lesions. Only 2 patients had proteinuria for more than a year; both had numerous tubular lesions.

**Kurland**


Quantitative studies of the isolated rabbit atrium have revealed that the onset of fibrillation is accompanied by a net loss of potassium. Fibrillation and its associated flux changes were inhibited by a decrease in temperature, a decrease in extracellular sodium concentration, an increase in intracellular potassium concentration, and the presence of quinidine. The data presented did not establish whether the permeability change was the cause or the result of the initiation of arrhythmia.


Based on data obtained from dogs poisoned by strophanthin, an attempt was made to differentiate between mild and severe digitalis intoxication. This was studied by means of the response to the antagonism of the potassium ion. It has been postulated that in digitalis intoxication, the primary defect may involve interference with active transport of potassium to the myocardial cells and this interference is in proportion to the dose of digitalis. When digitalis intoxication is mild, it seems that the use of potassium in any form will correct the ionic deficit. However, when digitalis intoxication is severe, the anion of the potassium salt may be a factor in determining its effectiveness. In particular, the authors were impressed with the results obtained when potassium was used as the glutamate. They speculate on the mechanism of potassium glutamate and glutamate reversal of strophanthin toxicity under circumstances where the chloride was ineffective. It is suggested that the glutamate ion may operate by its ability to promote active transport of potassium to the depleted cells.

**Krause**


Mercurial diuretics are an important and valuable adjunct in the treatment of congestive heart failure. However, they are potentially dangerous because of their action on the renal tubular epithelium. There have been scattered reports on the occurrence of the nephrotic syndrome after prolonged administration of mercurial diuretics. The authors present 5 cases of the nephrotic syndrome, 3 of them fatal, which appeared following the use of mercurial diuretics. The fatal cases were autopsied and each presented similar pathologic changes in the kidneys, resembling in quality those seen in acute poisoning with mercuric chloride. Furthermore, none of these cases had
evidence of kidney disease before the administration of the mercurial and each had a normal urine before treatment was begun. Additional convincing evidence was the fact that at autopsy, an excess of mercury was found in the renal tubules in 2 cases and, furthermore, no cause other than mercurial damage to the kidneys was found. Early recognition of this condition is of the utmost importance if a fatal outcome is to be avoided. Close supervision of patients treated with mercurial diuretics, including regular urine analysis is essential. The most useful evidence of tubular damage is the persistence of albumin despite a satisfactory diuresis in response to mercurial therapy.

KRAUSE


Isolated rabbit heart preparations were studied. The administration of epinephrine, norepinephrine, isoproterenol, or theophylline during perfusion via the aorta with normal Locke's solution generally increased the strength and rate of contraction, dilated the coronary arteries, and led to a net loss of potassium from the heart. Reduction in the potassium content of the Locke's solution by 75 per cent resulted in a greater degree of cardiac stimulation and coronary dilation when the above drugs were given; at the same time the mean potassium shifts from the hearts were not significantly changed. Perfusion with potassium-free Locke's solution regularly was followed by a rapidly progressive increase in rate and amplitude of contraction and increased coronary flow ending in ventricular fibrillation in 10 to 15 minutes. A number of related experiments are reviewed.

ROGERS


The effect of Diamox upon urine volume, body weight, and sodium and potassium excretions was evaluated using the diuretic index of Kattus. The drug was found to have virtually no effect upon normal pregnant patients with no edema or hypertension. In those with edema but without hypertension, Diamox produced a more rapid loss of fluid and electrolyte than did treatment with low-sodium diets (700 to 1000 mg.). However, unless sodium intake was restricted, the use of Diamox was associated with subsequent retention of sodium and water. In 20 patients with edema and hypertension a favorable response occurred with the use of Diamox; however, the enhanced excretion of sodium appeared to be unimportant in the management of these patients. There was no clinical improvement until after delivery. The patients receiving Diamox had no greater hypotensive response than those treated without specific therapy. The drug was ineffectual in patients with severe toxemia of pregnancy, eclampsia, reduced glomerular filtration, impaired renal function, or a profound electrolyte disturbance.

SHERMAN

RHEUMATIC FEVER


A comparison was made of the effectiveness in the treatment of 611 children of beta-hemolytic streptococcal infections of 1 form of parenteral penicillin (a single intramuscular injection of 600,000 units of benzathine penicillin G) and 5 different oral preparations (benzathine penicillin G tablets, penicillin G with probenecid suspension, penicillin G with probenecid tablets, buffered potassium penicillin G tablets, and penicillin V). Treatment with the oral preparations was maintained for 10 days. Penicillin in any of the forms used was found to be an effective agent in the immediate treatment of streptococcal infections. Study of the "cure rate" at the end of 2 months and the relapse rate within 25 days indicated that the single intramuscular injection of 600,000 units of benzathine penicillin G was superior to the oral forms of penicillin, therapeutically, prophylactically, in cost and in avoidance of the problem of oral administration of drugs to children. The chief disadvantage comprised the painful local reaction frequently observed and the psychologic trauma of an injection. Among the oral preparations no single preparation was found to be significantly superior to the others. There was no significant difference between the oral and intramuscular forms of penicillin in the allergic reaction rate. The authors conclude that the first choice of treatment of streptococcal infections in children is a single intramuscular dose of 600,000 units of benzathine penicillin G, but if an oral preparation is to be used cost factors would make buffered penicillin G in a dosage of 800,000 units daily for 10 days the first choice.

SAGALL

The results of the use of a single 600,000-unit injection of benzathine penicillin to navy recruits in an attempt to minimize streptococcal infection and rheumatic fever are reported. The injection was administered to 19,561 recruits arriving at one training station between November 22, 1955, and April 14, 1956. This program appeared to have reduced the incidence of streptococcal infections over that experienced in previous winters when prophylaxis either was not used or employed only after an outbreak had occurred. The incidence of rheumatic fever was also lower in those recruits who received prophylaxis. The single 600,000-unit injection of benzathine penicillin apparently afforded a high degree of individual protection against streptococcal infection for a period of about 3½ weeks. There were 145 clinical episodes adjudged to be sensitivity reactions after prophylactic benzathine penicillin. This rate of 0.74 per cent was very close to that found in a smaller previous experiment at this station and was about twice that observed after mass oral penicillin. No serious reactions were observed and the majority of sensitivity reactions were mild and transient with a good response to antihistaminic drugs.

SAGALL


The experience with a mass prophylactic program employing a single injection of benzathine penicillin to recruits at a naval training station during the fourth week of their 70 day stay is reported. Annual epidemics of streptococcal infections and rheumatic fever had previously been a problem at this station. The probable effectiveness of the prophylactic program with benzathine penicillin during the winter of 1956 and 1957 was indicated by the lowest incidence of streptococcal disease in the history of the station and by the fact that only 3 cases of rheumatic fever occurred among the 20,000 men at risk. Dosages of both 600,000 and 1,200,000 units of benzathine penicillin were used, but the larger dose did not appear to increase the degree or duration of protection. Adverse sensitivity reactions to penicillin were not a deterrent to the program.

SAGALL


Allergic penicillin reactions observed in 175 patients resulting from a total of 12,858 single injections of benzathine penicillin G given to military personnel for prophylaxis of streptococcal infections are described. This was a reaction rate of 1.3 per cent. The reactions observed ranged all the way from mild urticaria, lasting 1 or 2 days, to the full picture of serum sickness, lasting several weeks. Dermatologic manifestations only occurred in 21 per cent of this group while more severe reactions developed in 130 patients or 76 per cent. Fourteen patients (1 out of a 1,000 men injected) had reactions severe enough to require cortisone therapy. The reactions were of the delayed type and usually became manifest 8 to 15 days after the injection, although some occurred as early as the day of injection and some as late as 48 days after injection. The signs and symptoms of the allergic reactions tended to be prolonged and commonly recurred after they had temporarily abated, particularly in areas subject to trauma. Response to steroid therapy was prompt, but antihistamine therapy appeared to have only a slight effect in diminishing the length of illness caused by the allergy. Many reactions were observed to clear rapidly without therapy.

SAGALL


At a military hospital 5198 patients who received treatment with an antibiotic or sulfadiazine for streptococcal tonsillitis or pharyngitis were studied to determine the factors responsible for the development of rheumatic fever. Of this group 76 later developed acute rheumatic fever despite therapy. Eighteen of these patients were excluded from the study because the historical and bacteriologic evidence did not clearly indicate that the observed and treated streptococcal infection was the primary precipitating factor for their rheumatic fever. In 9 patients acute rheumatic fever developed during the first 72 hours of the acute streptococcal illness and treatment would not be expected to prevent rheumatic fever, since it was already present. Study of the various factors concerned in the remaining 49 patients with acute rheumatic fever showed that the primary cause of failure to prevent rheumatic fever was the fact that the infecting type of streptococcus was not eliminated by therapy. Other factors of lesser importance were the acqui-
sition of a new infection after therapy and a history of previous attacks of rheumatic fever or of recent streptococcal infections.

SAGALL


A report is presented of the pathologic changes found in the hearts of 30 patients with severe rheumatic fever who had been treated with cortisone or its analogues and died because of rheumatic endocarditis. Studies of other organs and also of nonspecific effects of cortisone will be reported separately. No clinical data were given, but the cases studied were said to be of comparable severity: the dosage of cortisone (or of prednisone or prednisolone) administered and the duration of treatment were tabulated. Doses considered adequate because of their systemic effects on the disease process were found to be without influence on the development of verrucae on the cardiac valves; the fibrinous substance of the verrucae was organized slowly; the underlying connective tissue exhibited an important fibroblastic reaction; infiltration of inflammatory cells was lacking. Fibrinous necrosis in the endocardial and myocardial connective tissue was found more frequently and more extensively in patients who had been treated with cortisone than in nontreated cases; the organization and reabsorption of the fibrinous substance was slower in the treated cases. The number of Aschoff's bodies decreased as an effect of treatment; the architecture of the nodules was altered in that the follicular cells resembled fibroblasts and elaborate collagen; the protoplasm of some of the Aschoff cells became strongly basophilic; scarred nodules were prominent. The morphologic characteristics of the Aschoff cells seen in these treated cases support the opinion that they are of fibroblastic origin.

CALABRESI

ROENTGENOLOGY


Possible toxic effects from Thorotrast administration were sought by studying 136 patients who had had carotid arteriograms for an average of 12½ years previously. The commonest effect, seen in 14 per cent, was fibrosis about the injection site (thorotrastoma), probably due to extravasation of the agent. This occurred despite the use of an open technic, but was usually small and asymptomatic. The only effect noted that was attributable to the prolonged systemic retention of radioactive material (thorium dioxide) was persistent roentgenologic density of the liver, spleen, and abdominal lymph nodes. Hemograms, chest and long-bone x-rays were available in a minority of patients and were nonrevealing. While more and more follow-up studies are needed, at present it appears that Thorotrust toxicity is largely local in nature.

Rogers


Two hundred consecutive patients with the clinical diagnosis of “stroke” or cerebral vascular accident admitted to the Cerebro Vascular Service of Detroit Memorial Hospital were studied angiographically. Angiograms were obtained by percutaneous injection of 8 to 10 ml. of 50 per cent Hypaque. From 2 to as many as 8 injections were given per patient with only 1 untoward reaction, an immediate fatality in a 60 year old woman who was critically ill. Autopsy in this case revealed neoplasm almost completely occluding the main pulmonary artery in the proximal portions of its branches. It appears that angiographic studies are quite safe in cerebral vascular disease. The combined clinical, neurologic and radiographic study on such patients has yielded much valuable information as to the etiology of symptoms. In addition to the more commonly described occlusions of the internal carotid and middle cerebral arteries, there is a relative frequency of occlusions of the vertebral-basilar artery system and of the anterior cerebral artery. Angiographic appearance of cerebral atherosclerosis without occlusion is described, and the importance of such findings is indicated. Complete occlusion of the internal carotid artery was encountered in 10.5 per cent of the 200 patients, of the anterior cerebral artery in 7.5 per cent, of the middle cerebral artery in 4.5 per cent, and of the vertebral-basilar arteries in 2.5 per cent. Significant narrowing of the internal carotid artery without complete occlusion was found in 10 per cent of this series. Fifty eight per cent showed some manifestation of atherosclerosis. A plea is made for visualizing the internal carotid artery down to its origin and for examining all vessels carefully for early signs of atherosclerosis.

KITCHELL

UNCOMMON FORMS
OF HEART DISEASE


A detailed report on 5 infants who died with glycogen-storage disease is presented. These
ABSTRACTS

children were all less than 3 months of age, were normal at birth and later developed upper respiratory tract infection, for which they were admitted to the hospital. In the course of observation they were discovered to have cardiac murmurs and cardiac enlargement. All developed muscular weakness and died a respiratory type of death. Autopsies were performed on all and in 2 muscules was submitted to Dr. Gerty Corey for analysis of glycoen and anylo 1-6 glucosi-
dase. An extensive discussion of the electrocardiographic changes in these patients is presented. All tracings showed a sinus tachycardia, QRS complexes were of high amplitude, the P-R interval was not prolonged, and the Q-T interval was within the normal range. There was no axis deviation. A prominent R wave occurred on the right side of the chest and the QRS complexes were diphasic on the left side of the chest. Depressed S-T segments were found in the limb leads, negative T waves were present in all standard limb leads, and late inversion of the T and negative T waves on the left side of the chest were demonstrable in some of the tracings in the chest leads. An extensive pathologic report is presented in 1 patient.

Harvey


The case of a 50 year old woman in whom successful surgical removal of a myxoma of the right atrium was accomplished by open heart operation, using a pump oxygenator and a stopped heart is reported. Arterial-blood oxygen studies demonstrated a right-to-left shunt (through a patent foramen ovale) varying with change in body position, affording an important clue to the correct diagnosis. The findings of cardiac catheterization closely resembled those of Ebstein's syndrome. The definitive preoperative diagnosis of myxoma of the right atrium was established by angio-cardiography, which demonstrated a large, lobulated filling defect in the right atrium. Since surgical cure of these lesions is now possible, the detection of intracardiac myxomas during life is important.

Sagall


The clinical aspects, arterial blood gases and pulmonary function were described in 24 persons with severe kyphoscoliosis and compared with comparable observations in 10 patients with congestive failure and 14 without congestive failure. The signs in the cardiovascular system of patients with heart failure were similar to those in emphysema with anoxie cor pulmonale. Cardiac decompensation was often precipitated by exacerbation of respiratory infection. Clinical examination of the heart was usually not striking but the periphery showed undue warmth and flushing, full abrupt pulse and severe central cyanosis. Cardiac catheterization in 3 patients showed a moderate rise in pulmonary artery pressure. Severe kyphoscoliosis with or without cardiac failure produced great reduction in total lung capacity, vital capacity, maximum ventilation, and large residual air, but little evidence of impaired mixing or bronchial obstruction. Reduced arterial oxygen saturation and retention of carbon dioxide were present in 8 of 9 patients with true kyphoscoliotic heart disease but in only 2 of the 14 patients without heart failure and provided a clearer distinction between the 2 groups than any lung studies.

Kurland

Vascular Disease


Thrombosis of the internal carotid artery may present the clinical picture of a space-occupying lesion. Five patients are presented in 4 of whom cerebral neoplasm was diagnosed and 1 cerebral abscess. The symptoms developed in 2 stages: first, the progressive or intermittent appearance of focal symptoms such as headache, convulsions, localized weakness, paresis or dysphagia and, second, the features of a space-occupying cerebral lesion. Radiography, lumbar puncture, electroencephalogram may not clarify the diagnosis. Certain differentiations can only be made by carotid angiography.

Kurland


On the basis of direct catheterization studies in man with pulmonic stenosis and aortic coarctation and hydrodynamic experimental observations, the authors offer an explanation for the pathogenesis of the hemodynamic paradox of poststenotic dilatation. They point out that any change
that elevates the blood pressure or results in weakening the wall of a vessel at the circum-
scribed area might cause widening of the artery at that particular section. Since in the direct
catheterization studies and in the model experiments localized increases in arterial blood pres-
sure were not found in the poststenotic region, the theory of an elevation of lateral pressure
below the level of the stenosis as the cause of the poststenotic dilatation can be eliminated. The
hemodynamic studies indicated that the turbulent flow and, perhaps more important, cavitation with
bubble formation in the poststenotic area were the factors causing severe injury to the vessel
wall and dilatation. These factors acted more severely when the stenosis was abrupt rather than
gradual. The poststenotic dilatation was an important differential diagnostic sign between steno-
sis involving a short or a long segment of a vessel. Poststenotic dilatation appears to be a favorable
factor in blood circulation because it improves the blood flow below the level of the stenosis
primarily enhancing the collateral flow and to a lesser degree the flow through the stenosis itself.

SAGALL

Chason, J. R.: Cerebral Infarction Secondary to
Occlusive Arterial Disease. Radiology 70: 811
(June), 1958.

Bland infarcts of the central nervous system
are of 2 forms. The more common is the anemic
type, where scant secondary hemorrhage is limited
to the periphery of the area of necrosis; the less
common is the hemorrhagic infarct, where blood
fills all or most of the area destroyed. After
the occurrence of an anemic infarct softening
and liquefaction gradually ensue. These changes
begin probably within 6 hours, but the radiologist
is most often confronted with such an infarct
that is in the second or third week. At this time
fluid accumulation and cell infiltration within the
necrotic region can produce enlargement suggesting
an expanding mass. Such progressive en-
largement ends when the rate of removal of the
necrotic material exceeds the rate of attraction
of fluid and reactive cells, usually at the end of
the third week. After this there is a gradual
decrease in the size of the infarct accompanied
by focal enlargement of the adjoining ventricular
and subarachnoid spaces. In hemorrhagic infar-
tion the story is similar to that of the anemic
infarct except enlargement occurs earlier and is
of greater degree and of longer duration. Vessels
in the infarct rapidly undergo necrosis. Other
vessels are subjected to a hypoxic state, which
stimulates hypertrophy and hyperplasia of the
endothelial cells. The lumens of these vessels
are decreased in size and their effective use as
collateral channels is impaired. Distal to the
point of arterial obstruction, arterial or capillary
emboli may form within the infarct and exert
a detrimental influence upon the utilization of
the vessels as part of the collateral circulation.
The occluding thrombus or embolus may enlarge
either distally or proximally and form a further
serious threat to the availability of the existing
channels.

KITCHELL

Meyer, J. S.: Theory and Rationale of Anticoagu-
lant Therapy in Occlusive Cerebral Vascular

It is generally conceded that long-term anti-
coagulant therapy is of benefit in arteriosclerotic
thrombosis and occlusion of the internal carotid
and basilar arteries associated with intermittent
ischemic episodes. Such therapy reduces the
incidence or abolishes entirely the attacks but
does not appear to affect significantly the course
of severe infarction once it has occurred. The
present report is concerned with an investigation
of the action of anticoagulant drugs in experi-
mental cerebral vascular disease. It appears that
the mechanism of localized changes in cerebral
vascular resistance following cerebral vascular
occlusion is first a slowing of local blood flow
with ischemic anoxia resulting in endothelial
damage and localized loss of fluid constituents
of the blood. This results in brain edema and
localized hemoconcentration. Hemoconcentration
is followed by increased adhesiveness of all
formed elements of the blood and of the endo-
theium. The administration of heparin and Die-
marol prevents the adhesiveness of red cells,
white cells, and platelets, and by this means pre-
vents increased cerebral vascular resistance and
promotes better collateral circulation.