ATHROSCLEROSIS


Carotenoid pigments are colored lipids distributed widely throughout nature. Certain carotenoids, chiefly those without hydroxyl groups, can be converted to vitamin A. Studies have indicated that carotenoids cannot be synthesized by man. It was thought advisable, therefore, to study the occurrence of these lipids in atheromatous plaques, as evidence that dietary lipid may be deposited in the lesions. The concentrations of total carotenoids and total cholesterol were studied in 30 human aortas with varying degrees of atherosclerosis. With increase in the severity of the lesions, there were accompanying significant increases in carotenoid and cholesterol contents. The carotenoid-cholesterol ratio was relatively constant. Although cholesterol in the plaque may accumulate either inside the plaque or outside, it is generally accepted that carotenoids are derived from exogenous sources. Although it is unknown whether carotenoid pigments are concerned with the pathogenesis of atherosclerotic lesions, the present findings give evidence that one dietary lipid is present not only in atherosclerotic plaques but accumulates in direct proportion to age and extent of the lesions.

WAIFE


Twelve hundred healthy males and females in Staten Island (New York) between the ages of 2 and 17 were examined and their sera analyzed for cholesterol and phospholipids to determine the average lipid levels for this population. Cholesterol and phospholipid levels for men remain constant to age 19, increase from age 20 through 33, and then remain constant to age 60. Levels for women remain constant through age 32 and increase sharply and continuously from age 33 through 58. The difference in the age trends between men and women may perhaps be related to the well-known preponderance of men with coronary artery disease in the younger decades and the proportionate increase in women with coronary atherosclerosis after the age of 50.

It is interesting to note that the period of marked increase of serum lipid levels that occurs physiologically in both sexes starts 13 years later in women than in men and lasts 12 years longer.

KITCHELL


Fourteen patients with atherosclerosis and pronounced hypercholesterolemia were given phenylethylacetic acid amide, 2 Gm. daily for 2 weeks and 3 Gm. daily thereafter for periods of 3 to 10 weeks. In 13 patients, a significant reduction (ranging from 71 to 5 per cent) of plasma cholesterol was found among the 10 patients who received the drug for 8 to 10 weeks, the mean reduction at the end of treatment was 43.3 per cent. The mean plasma cholesterol value dropped to normal for the major part of the period of treatment, and even below normal during the tenth week. One month after the drug was discontinued the cholesterol values had risen to near the initial prestudy levels.

SAGALL

Circulation, Volume XVI, October 1957

The authors report the results of controlled experiments on metabolically normal, physically healthy schizophrenic men in the 32 to 54-year-old age group who were maintained on diets that were constant in calories but differed in their protein content in order to evaluate the influence of dietary protein on the production of lowered blood cholesterol levels. The response of the total serum cholesterol to a change from a low- to a high-fat intake was the same in 1 group of men receiving 83 Gm. of protein daily as in a matched group on the same diet except for the isocaloric substitution of an extra 43 Gm. of skimmed-milk protein for carbohydrate in the diet. Two groups of subjects were maintained on a low-protein intake, were changed for 4 weeks to a high-protein intake and then were changed back to the low-protein intake, all at a constant fat intake. No significant change in the serum cholesterol level was found in either group at any time. Also, an increase of 1,000 mg. per day in the dietary cholesterol intake, maintained for 8 weeks, had no significant effect on the serum cholesterol level.

SAGALL

Arch. Ophth. 57:190 (Feb.), 1957.

Twenty-three patients having more or less advanced senile macular degeneration received heparin therapy designed to suppress underlying atherosclerosis. One hundred milligrams of a concentrated aqueous solution was administered intravenously semiweekly in 5- to 10-week periods for as long as 3½ years in an office practice. Seventeen of the 23 improved, while 3 each were stationary or became worse. The disciform type of lesion seemed to respond better than the arteriosclerotic type. No untoward reaction was noted from more than 1,000 heparin injections.

ROGERS


Essential hyperlipemia is a clinical state of unknown cause characterized by an increase in the blood neutral fat. Four additional patients are presented, 3 of whom presented initially with pure abdominal pain. Each patient showed a marked increase in blood neutral fat but also increased serum cholesterol, phospholipid, and fatty acids. Moderate impairment of glucose tolerance was demonstrated. On a low-fat diet, symptoms subsided promptly and blood lipid concentrations decreased.

KURLAND


Two siblings, aged 11 and 10, who had a very strong family history of hypercholesterolemia were put on a diet, moderately low in cholesterol and were given 20 ml. of a 20 per cent suspension of beta-sitosterol 3 times daily. Measurements were made of the serum cholesterol and serum phospholipids in the individuals at frequent intervals. The initial levels were elevated over the normal. A significant reduction in the concentration of cholesterol in the serum of these individuals was effected by the use of sitosterol administered 3 times daily. Adequate figures are given.

HARVEY


The authors studied the effect of reduction of thyroïdal activity upon atherogenesis in patients treated with radioiodine by following Gofman’s “atherogenic index” and the total serum cholesterol. Mild rises in both parameters occurred in hyperthyroid patients rendered euthyroid. These involved only minimal added risk of atherogenesis by Gofman’s calculations. Similar changes followed therapeutic ablation in euthyroid patients not presenting atherosclerotic heart disease. In atherosclerotic patients greater rises in serum total cholesterol and atherogenic index were sometimes observed. Among the patients with heart disease, radioiodine therapy dramatically relieved two thirds of those with incapacitating anginal pains. However, results in the patient presenting cardiac insufficiency secondary to pulmonary dysfunction and in several other such patients not included in this study have been less favorable. In patients thyroïdectomized for cardiac insufficiency resulting from atherosclerotic heart disease, changes in lipoprotein spectrum did not always parallel changes in total cholesterol and lipoprotein changes were not restricted to the S1 0-12 class. The excellent results obtained by Blumgart and the authors in rehabilitating patients through the relief of cardiac decompensation, congestive failure and anginal pain certainly seem well worth any added small risk of increased atherogenesis.

HARRIS


Hemodynamics has been presented as a primary factor in the development of atherosclerosis. A physical basis for occlusive coronary artery disease is offered. A mechanism is described for intimal ulceration, intramural hemorrhage, and dissecting.
hemorrhage occurring as complications during the pathogenesis of atherosclerosis. Atherosclerosis would appear to be a sequel primarily of fluid dynamics applied to the natural conditions in the circulatory system. A clinical and pathologic correlation of cardiac stress is discussed.

BERNSTEIN


Hemorrhage into an isolated atherosclerotic lesion of the right middle cerebral artery was found at autopsy to be the cause of encephalomalacia in a 28-year-old laborer. The remaining cerebral vessels were normal, and only minimal atherosclerosis was noted in the aorta and coronary vessels. A review of the literature emphasizes the rarity of clinically significant artery atherosclerosis in the younger age group.

BERNSTEIN


Numerous studies of experimental atherosclerosis have provided ample evidence of differences in metabolism of cholesterol in different species of animals. While the rat is very resistant to its production by cholesterol feeding: a marked increase in adrenal cholesterol resulted from feeding with rape oil. This contrasts with results obtained with rabbits, guinea pigs, chickens and dogs in which dietary cholesterol tends to be deposited more readily but in which rape oil is ineffective in causing a rise in adrenal cholesterol. The differences in response do not appear to be due to species differences in digestibility of rape oil but are probably related to differences in metabolism of phospholipids.

AVIADO


When cholesterol-fed rabbits are killed at successive periods gross, discrete atherosclerotic lesions appear first in the aortic arch and only much later in the rest of the aorta. The descending aortic limb has significantly lower total lipid, primarily due to decreased neutral fat concentration, compared with the arch. The specific activity of phospholipid in the descending aorta is significantly greater than that of the aortic arch of normal rabbits but this distinction is not found in the rabbits on a high-cholesterol diet. This is due to an increased rate of formation of phospholipid on the aortic arch (in fed rabbits) and probably is related to the development of atheromatous lesions. Although these facts suggest some fundamental metabolic differences between certain areas of the aorta, their possible relationship to the pathogenesis of atherosclerosis must await further studies of arterial tissue metabolism.

BLOOD COAGULATION AND THROMBOEMBOLISM


A series of 47 patients with mesenteric thrombosis were observed during a period of 17 years. In 42 patients the diagnosis was confirmed at operation or autopsy. Occlusion of the superior mesenteric vessels was present in all instances. Arterial thrombosis or embolism was present in 37 patients and venous thrombosis was found in 5. There were 5 unverified cases, 3 of whom were given anticoagulants and all of whom survived. Cardiac disease was the causative factor in 28 patients, 21 of whom had atrial fibrillation. Malignant tumors were present in 4 patients and inflammation was present in 7. In some patients the infarction developed insidiously without any alarming symptoms for several days and in 5 patients there were no abdominal symptoms. Fourteen patients were operated upon and 3 survived. The authors believe that if the extent of the lesion permits, the treatment of choice in this disorder is resection of the involved portion of the bowel. Adequate fluid therapy in the postoperative period is very important. Anticoagulant therapy after operation is also important in order to prevent the development of new emboli or an extension of the thrombosis and intestinal gangrene. If resection is not possible, the condition apparently responds in some instances to anticoagulant therapy alone. The authors believe that if hemostasis is carefully performed at the time of surgery, anticoagulant treatment, preferably with heparin, can be begun on the day after operation.

ROSENBAUM


Of 63 routine autopsies on persons subjected to venipuncture, thrombosis of the cubital veins was found in 80 per cent. The thrombosis was attributed to mechanical lesion of the venous wall, combined with changes in blood composition due to injection and probably also circulatory stasis preceding death. Morphologic lesions of the endothelium caused by injected substances could not be found. In 14 patients organization of thrombi was present. Embolism of cubital venous thrombi could not be proved with certainty, but small pulmonary embolisms appeared likely.

LEPESCHKIN

Tuller, M. A.: Amniotic Fluid Embolism, A fibrinogenemia, and Disseminated Fibrin Thrombosis.

The syndrome of sudden dyspnea, cyanosis, and extreme shock occurring intrapartum or immediately post partum in association with a strenuous labor and clinical and pathologic findings of cor pulmonale is described as attributable to amniotic fluid embolism. Afibrinogenemia has been demonstrated in 1 patient and presumed to be present due to incoagulability of the blood in 4 others in the medical literature. The present case was a 38-year-old gravida 3, para 2 who developed afibrinogenemia during Pitocin induction complicated by shock, oliguria and death on the eighth post partum day. At autopsy, multiple fibrin emboli in the small vessels of most organs were found. Extensive necrosis of the pituitary gland was present. Afibrinogenemia was described as resulting from intravascular coagulation attendant upon release of thromboplastic material into the circulation.

Shuman


The mechanism of blood coagulation was examined serially in 20 normal pregnant patients from the thirty-fourth week through delivery and the early part of the puerperium. The levels of fibrin and platelets were found to decrease near term. Clot retraction time and prothrombin time were reduced in early labor. A sharp stimulus for platelet production was noted during labor. The utilization of fibrinogen for hemoostasis during delivery is parallel to that noted during surgical procedures. During the puerperium, there was an increase in platelets, prothrombin, and fibrinogen. These changes may contribute to the embolic phenomena that are common at this time. The normal fluctuations during parturition of the factors involved in blood coagulation would not have caused any major bleeding difficulties.

Shuman


The plasma of 10 women with hemorrhage due to afibrinogenemia were examined for fibrinogen content, fibrinogenolytic and fibrinolytic activity, the inhibitors of such activity, and the hydrolytic products of enzyme action. Significant levels of fibrino- and fibrinogenolytic activities were found in most of the patients. Fibrinogen levels were reduced to less than 25 mg. percent in 6 patients, 3 were found between 55 and 89 mg. per cent, 1 had a minimum level of 170 mg. per cent. After fibrinogen therapy bleeding usually showed a marked decrease within 30 to 60 minutes and changed to a normal lochia within 3 hours, at which time fibrinogen levels had risen to 120 mg. per cent or higher. The lysis of serially diluted plasma clots disclosed considerable fibrinolytic activity in all cases. This change disappeared rapidly after intravenous fibrinogen infusion. Inhibitor levels showed a concomitant return to normal. It is suggested that proteolytic activity may be at least partially responsible for the fibrinogenopenia observed in these patients.

Shuman


In 33 patients the anticoagulant phenindione was employed on a long-term basis to maintain the prothrombin time at therapeutic protective levels (between 26 and 36 seconds originally, but between 20 to 30 seconds in the latter part of the study). The dose of phenindione required usually decreased during the first 3 months, but thereafter remained constant in 50 per cent of the cases and required little changes in the others. Because of its steadier effect on the prothrombin time with resultant less dosage adjustment and less close supervision being necessary, phenindione is considered by the authors to be superior to Dicumarol in long-term anticoagulation. In this series nearly all patients noted minor evidences of a bleeding tendency, and significant bleeding occurred in 27 per cent of the cases. During 565 patient months of treatment thromboembolic episodes occurred twice in 2 patients. Other than bleeding no toxicity was observed.

Sagall


The present report is based on the routine use of phenindione in 200 consecutive hospitalized patients in whom anticoagulant therapy was indicated. In the average patient the initial dose was 200 mg., followed by 100 mg. in 12 hours. For predictable effects the maintenance dose had to be given every 12 hours. The dosage varied and ranged from 29 to 400 mg. to keep the prothrombin activity between 5 and 10 per cent of normal. Changes in dosage requirement were seen, as was a cumulative effect with the same dose in some instances. The dissipation of effect usually occurred within 72 hours and, oftentimes, within 36 hours. Stabilization of dose did not occur for 6 days or more. In 21 patients (10.5 per cent) bleeding occurred, but this was slow and readily responded to vitamin K emulsion. The present study indicated some superiority of phenindione overbishydroxycoumarin in clinical use.
only as a result of the more rapid onset and dissipation of its effect.

Sagall


The authors report 22 instances recorded in the literature and 21 patients from their own clinic in whom there was evidence suggesting a familial occurrence of thrombosis, thrombophlebitis, or thromboembolic disease. These observations are believed to be of some importance in the problem of the etiology of thromboembolic disease and to argue for a more liberal prophylactic use of anticoagulants in patients with a familial history of thrombosis.

Rosenbaum


Fresh thrombi were found in various parts of the ciliary veins of 10 stillborn or newborn infants autopsied since 1950. Three of them had thrombi in other organs. Review of various data regarding the infants and their mothers gave no adequate explanation for occurrence of the ciliary vein thrombosis, a previously unobserved phenomenon.

Rogers


The daily oral administration of Sintrom to 42 patients, most of whom had acute myocardial infarction or acute coronary insufficiency, was guided by daily standardized clotting times (SCT), the normal value being 9.69 to 0.55 minutes. Therapeutic prolongation of the SCT to 15 to 20 minutes was reached after an average induction period of 4.3 days and was obtained on 71 per cent of the total of 866 treatment days. Neither hemorrhagic accident nor thromboembolism was noted while the SCT was in the therapeutic range, and it was concluded that Sintrom is a satisfactory anticoagulant for short-term therapy.

Rogers


An example of massive pulmonary embolization followed by instantaneous death in a 71 year-old white man following transurethral prostatic resection is presented. The embolization was composed of bone marrow and metastatic tumor to the bone marrow. It was assumed that bone “conclusion” of the vertebrae, caused by the strain of cystoectomy or moving from the bed, or both, resulted in medullary fracture of bone marrow previously weakened by carcinomatous metastasis. No comparable case has been noted in the literature.

Maxwell

CONGENITAL ANOMALIES


It is possible to define 3 clinical types of persistent common atrioventricular canal. The first is atrial septal defect (ostium primum type) without valvular insufficiency. Hemodynamically this lesion is identical with atrial septal defect. Significant pulmonary hypertension may be associated with this lesion but seems to be relatively infrequent. The second is atrial septal defect (ostium primum type) with valvular insufficiency (both atrioventricular valves or only the mitral valve). Pulmonary hypertension is more frequently present than in the first type. The third is atrial and ventricular defect with atrioventricular valvular insufficiency. Pulmonary hypertension is frequently present. Roentgen examination has not been particularly helpful in differentiation in most patients. In adult patients the cardiac silhouette has not been larger than in patients with usual atrial septal defects. The cardiac silhouette may be only slightly or markedly enlarged. The pulmonary artery shadow and pulmonary vascular markings are increased. Cardiac enlargement involves predominantly the right side of the heart, a fact that makes mild degrees of left ventricular and left atrial enlargement difficult to detect. The electrocardiogram is an important tool of the clinician. This aspect of the clinical appraisal of the patient with this defect has proved a most important diagnostic aid. From a careful evaluation of the authors experiences with this lesion in the past year they believe that in the future they shall be able to suspect this defect in most instances on the basis of clinical data. With the additional aid of hemodynamic data from the catheterization laboratory a decision regarding surgical treatment in an individual case can usually be made on a sound basis.

Simon


A case of a 14 year old boy who had aplastic anemia and a coarctation of the aorta is reported. The child died of a massive cerebral hemorrhage secondary to thromboctopenia. Speculation is presented on a possible relationship between the congenital cardiovascular abnormality and the bone
marrow deficit. It is suggested that both may represent a "forme fruste" of the Fanconi syndrome.

**Harvey**


A 21-year-old soldier who died suddenly showed at autopsy complete fusion of the posterior and incomplete fusion of the septal leaflet of the tricuspid valve with the endocardium. The man had no complaints, and all function tests except those of Lian and Demeny were normal. A nonpropagated mesocardiac systolic murmur could be heard, and the electrocardiogram showed only left axis deviation.

**Lepeschkin**


Nearly complete atresia of the pulmonary artery with intact ventricular septum and persistence of the foramen ovale and the ductus arteriosus was seen in 2 girls, comprising about 1 per cent of all congenital heart disease and 6 per cent of all trilogies of Fallot. In the first case the pulmonary orifice barely admitted a pin at autopsy; in the second case this orifice admitted a 1-mm. bougie during a valvulotomy operation, after which the extreme cyanosis disappeared almost completely, but the size of the heart increased. The electrocardiogram in both cases showed marked right axis deviation, tall monophasic R with inverted T in V1 and deep S in V5-V6.

**Lepeschkin**


When the pulmonary veins are transposed, to drain anomalously only into the right side of the heart, it is evident that the left heart would be bloodless unless there was a communication between it and the right side. In its simplest form, this communication is a patent foramen ovale. As would become evident in a study of any diagram of the fetal circulation, the anomalous circulation associated with total pulmonary venous drainage causes no disturbance in the fetus, but in postnatal life a gross inefficiency in the mammalian-type circulation exists.

In any differentiation of types of total anomalous pulmonary connection, the presence or absence of pulmonary hypertension is of paramount importance. Those with pulmonary hypertension could be subclassified into those in which the pulmonary hypertension is related to obstruction of the outflow of blood from the lungs, where a high "wedge pressure" in a pulmonary artery could be recorded, and those in which pulmonary arteriolar obstruction occurs pari passu with increased pulmonary blood flow. The clinical picture in infants is different from that in older children or adults and most often is manifested by pulmonary and systemic venous congestion (heart failure) appearing in the early neonatal months. While the infants may be intermittently cyanotic, cyanosis is not a characteristic of the condition at birth and even not during the course of the failure except at its termination.

While the size of the foramen ovale or interatrial orifices which allow blood to reach the left side may be critical, it would appear that this is not always the main factor in causing the heart failure, but rather, the right heart may fail in relationship to its efforts to maintain a high flow of blood against an increasing pulmonary resistance. One clinical syndrome that has been suggested that might tip one off to the presence of this total anomalous drainage into an abdominal vein would be the evidence of pulmonary congestion when a child strains.

In concluding these remarks, a definite conflict of opinion may be registered with those who would recommend that surgical therapy of total anomalous drainage be restricted to those patients with progressive disability. When symptoms are progressively severe and the patient is disabled, notably would operative risk be high, but the chances of restitution of normal circulatory function would be meager because of nonregressive pulmonary vascular changes. When the diagnosis can be made, the recommendation for surgical intervention exists if severe pulmonary hypertension is absent, and the surgeon then picks the time when technically it can be most competently and safely performed.

**Simion**


In 4 patients in whom a tetralogy could be proved by cardiac catheterization (entrance of the catheter into the aorta, large pressure gradient at the pulmonary artery) the pulmonary vessels were dense and pulsed in 2 patients. In 2 instances a partial explanation of these unusual findings could be given by assuming a persistent ductus arteriosus. Another explanation is the development of a pulmonary collateral circulation intense enough to show vascular pulsation. A third possibility is pulmonary regurgitation. Finally, it is possible that in some cases of tetralogy the intraventricular communication is more important than the pulmonary stenosis.

**Lepeschkin**

In 4 patients (interventricular communication, intraatrial communication, aortico-pulmonary fistula, and pentalogy of Fallot) cardiac catheterization disclosed that the pulmonary pressure showed no appreciable change, while the systemic pressure was always lowered whenevercyanosis was present. Oxygen inhalation did not improve the arterial oxygen saturation, but infusion of norepinephrine raised the systemic pressure and improved this saturation immediately.

LEPESCHKIN


This male patient with early cyanosis showed an only slightly enlarged heart, a vertical QRS axis with wide R' in leads V1-V2, a transition zone at V6, absent Q waves, pointed T waves in V3-V4 and elevation of P in leads II and III. Autopsy showed a single ventricle with a common truncus-pid A-V valve and large atrial septum defect; the wide aorta originated anteriorly while the very narrow pulmonary artery originated from the same ventricle posterior to it. The relatively long life of the patient is attributed to extensive pulmonary anastomoses. The most striking observation is that normal right and left ventricular patterns were seen in precordial leads in the presence of a single ventricle of nearly uniform thickness.

LEPESCHKIN


The authors report their findings in 50 infants with congenital heart disease studied by simultaneous heart catheterization and angiocardiography. All examinations are done under anesthesia. Contrast substance, 1.1 to 1.9 ml. per Kg. body weight, is injected within 1 second through a catheter passed through the saphenous vein.

The authors recognize that these studies are not performed under physiologic conditions. The pressure readings are unstable. A difference of at least 1 volume per cent in oxygen saturation must be present to be diagnostic of a shunt between the pulmonary artery and right ventricle or left atrium and right atrium. Between the right atrium and vein cavae, the difference must be at least 2 volumes per cent. The left atrium was catheterized in 15, only 11 of whom had signs of an intravascular shunt. In 5, the aorta was catheterized through the ductus arteriosus.

Direct intracardiac injection of contrast substance permits excellent visualization of that part of the heart which is of special interest.

This combined technic, together with the clinical findings, permits a satisfactory degree of certainty of the pathologic, anatomic, and physiologic cardiac abnormalities.

SOLOFF


Lung sections from autopsy and biopsy material from a large number of patients with congenital heart disease were studied in comparison with sections from 100 normal patients. The pulmonary vascular changes in small muscular pulmonary arteries associated with, or lying clearly apart from, small bronchioles was evaluated. The total transverse diameter of such a vessel was measured. The thickness of each layer, adventitia, media, and intima, was recorded. The diameter of the lumen was divided by twice the thickness of the media and intima added together, thus giving a ratio of lumen size to wall thickness. The lumen:wall ratio was placed against the age of the patient. Microscopic examination of the lungs of 50 patients over 2 months of age in whom a clear-cut anatomic pulmonic stenosis was present revealed an essentially normal pattern of evolution of the pulmonary vascular bed. The small pulmonary arteries were thin-walled with wide lumens and were similar to those in normal lungs.

RINZLER


Detailed clinical findings are presented in 69 patients with congenital arterial venous fistulas seen at the Mayo Clinic from 1935 to 1953. The authors point out that arteriography merely provided corroborative information and that correct interpretation of arteriograms required extensive experience. No cardiac symptoms were thought to be referable to the fistula. Treatment of these lesions has been difficult and must be individualized. Some patients were successfully managed with control of edema and healing of ulcerations through the use of elastic stockings and heel lifts. In others, ligation and stripping of varices and a direct attack on the fistula were performed. An appreciable number of patients required major amputation.

WESSLER


The results of cardiac catheterization in 60 in-
individuals with atrial septal defect were analyzed in an attempt to understand the nature of this disorder and some of its complications.

When the defect is small, left atrial pressure is greater than right atrial. When the defect is large, the pressures in each atrium during diastole are equal. Flow continues from right to left and mostly during diastole because of greater distensibility of the right atrium. In diastole, there is a free communication between respective ventricles. A small right to left atrial flow may also be present.

The left ventricular output is not above normal. The right ventricular and pulmonary arterial pressures are normal unless flow is greater than 10 liters per minute per M.², which raises pressures slightly, or unless there is pulmonary vascular disease, which may raise pressures considerably.

The incidence of cyanosis increases with age. The severe grades of cyanosis are associated with the smallest right ventricular outputs.

Minor degrees of cyanosis are explainable on mixing of systemic and pulmonary venous blood streams in the common atrium. This mixture is enhanced when the right ventricular pressure approaches the systemic. A high right ventricular pressure is associated with the smallest right ventricular outputs and with high pulmonary vascular resistance.

The cause for increase in pulmonary vascular resistance is not clear but it may be due to pulmonary thrombosis or embolism either of which is a common event late in the natural history of atrial septal defect.

Left ventricular output remains fairly constant even with rising end diastolic pressures. On the other hand, the right ventricular output may be large or small, regardless of the height of its end-diastolic pressure. Digitalis can increase right ventricular flow without changing its end-diastolic pressure. It is therefore concluded that right ventricular failure is manifested by a decrease in right ventricular flow without change in its end-diastolic pressure. On the other hand, left ventricular failure is characterized by increase in its end-diastolic pressure, which is transmitted to both atria and to the right ventricle.

The right ventricle fails because (1) of the increasing work it performs (pressure and flow), (2) of the appearance of pulmonary vascular disease, and (3) of the production of functional tricuspid incompetency.

The left ventricle fails because of the presence of relatively minor lesions imposing a burden on the left ventricle such as mitral valvulitis, hypertension, and endocardial fibroelastosis of the left ventricle.

Finally, Lutembacher's syndrome is rare and can be diagnosed only by demonstrating a pressure gradient between the left atrium and the left ventricle.

SOLOFF


The complete and partial varieties of persistent common atrioventricular canal appear to represent different degrees of malformation in relation to the atrioventricular endocardial cushions. In each instance, there is an interatrial communication. In the complete variety interventricular communication is frequent, while in the partial variety this is uncommon. Incompetence of the mitral valve may be an integral, though not necessary, feature of the malformation in the partial variety. In the complete form, insufficiency of both atrioventricular valves may exist. Death at an early age is usual in the complete variety, while longer survival is more frequent in the partial variety.

SIMON


The hemodynamic data and findings of diagnostic value obtained during cardiac catheterization are presented in 9 proved cases of persistent common atrioventricular canal. Four of the 5 adults were found to have normal pulmonary artery pressures and pulmonary vascular resistances. Pulmonary hypertension was present in 1 of the 5 adults and in all 4 children studied. The systemic blood flows were found to be normal in all cases. The increased pulmonary blood flows were due to large left-to-right shunts occurring at both the atrial and the ventricular levels, but in most cases chiefly at the atrial level.

The catheterization features that assist in the differentiation of persistent common atrioventricular canal from atrial septal defect of the usual variety are: (1) the low position of the shaft of the catheter in the cardiac silhouette when the catheter tip has been manipulated into the left ventricle; (2) evidence of additional arterIALIZATION in the right ventricle over that in the right atrium; and (3) the absence or decreased degree of preferential left-to-right shunting of blood from the right lung.

SIMON


The diagnosis of total anomalous pulmonary venous drainage rests upon the demonstration that the saturation of pulmonary artery blood equals or exceeds that of systemic artery blood, and that by dye-dilution curves the right ventricle represents a site that is functionally upstream to the right.
atrium in the pathway to the systemic circulation. The anatomic basis for these findings may be either a common atrium or an anomalous connection of all the pulmonary veins to the right atrium or one of its tributaries. In the latter instance, the presence of abnormal elevation of the oxygen saturation of blood samples drawn from the inferior or superior vena cava, innominate vein, or coronary sinus, or the course of a cardiac catheter when entering a pulmonary vein may assist in arriving at a correct anatomic diagnosis.

SIMON


The authors present a case report of a 13 month old cyanotic child with periodic respiratory distress, cardiac enlargement to the left, good first and second heart sounds in all areas, and a soft systolic murmur. Roentgenograms indicated an enlarged left ventricle, no evidence of left or right atrial enlargement, and a distinctly small right ventricle, as judged by failure of the cardiac shadow to project toward the anterior chest wall in the left anterior oblique position. The electrocardiogram showed the pattern of left ventricular hypertrophy.

At autopsy the following were noted: tricuspid atresia with a rudimentary right ventricle; atretic pulmonary artery trunk with almost normal-sized primary branches fed via a patent ductus arteriosus; large atrial septal defect; and a small ventricular septal defect with an overriding but otherwise normal aorta. The atrial tips were juxtaposed to the left of the aorta. Death apparently was precipitated by thrombosis of the left pulmonary artery.

SCHWEDEL


Six young adult men 19 to 29 years of age were studied mainly because of the presence of a systolic murmur. Poststenotic dilatation, as demonstrated by chest roentgenograms in the left anterior oblique view with barium swallow, was present in all cases, and in 2 instances the area of coarctation could be seen on the plain posteroanterior chest roentgenogram. The heart size was normal by x-ray in 3 cases and slightly enlarged in the other 3. The electrocardiogram was normal in 5 patients and showed left ventricular hypertrophy in 1. In all but 1 case (systolic pressure was 155 mm. Hg) the brachial artery pressure by direct measurement was normal. By palpation, the femoral artery pulsations were considered to be normal in 1 patient, slightly decreased in another, decreased in 3, and feeble in the remaining patient. The author concludes that when a murmur is heard at the level of the second to the fourth intercostal space beneath the sternum, and especially when this murmur is well heard in the back, coarctation of the aorta should be suspected, despite the level of the brachial blood pressure by sphygmomanometry. This becomes especially pertinent if the femoral pulses, by palpation or measurement, are considered to be present but diminished.

RINZLER


The author describes a 42-year-old woman with what was fairly typical clinical mitral stenosis with additional signs of tricuspid regurgitation. At operation the surgeon discovered a large left superior vena cava draining the left superior pulmonary vein, joining the left innominate vein and presumably carrying blood that found its way eventually to the right atrium via the normal right superior vena cava. The pulmonary artery was enormous. Tight mitral stenosis in a mobile, non-calciﬁed valve was found but no regurgitation. Satisfactory commissurotomy was possible. After operation improvement was in general satisfactory. The apical diastolic rumble disappeared but signs of tricuspid regurgitation, a pansystolic murmur at the lower end of the sternum, and an opening snap (? mitral) persisted. Cardiac catheterization data obtained after operation are presented.

It is of interest that the preoperative radiograph presented by the author shows much greater radiolucency of the left upper lung field, which was not subjected to the congestive effects of pulmonary venous hypertension.

MCKUSICK


The rarely reported cases of death during or following cardiac catheterization are reviewed. The authors describe a 10-year-old girl who at autopsy was found to have the right subclavian and jugular veins entering the right atrium and left subclavian and jugular veins entering the left atrium. No innominate vein connected the left and right superior venae cavae. There was a patent ductus arteriosus. The left atrium, left ventricle, and aorta were small, but the aorta became larger beyond the patent ductus.

Clinically the patient had universal cyanosis, clubbing of the toes but not the fingers, a systolic murmur at the apex and left sternal border, hepatomegaly.

Cardiac catheterization was attempted from the left arm because the right arm had been used for

Congenital malformations of the heart have been studied in 1,395 patients. In the vast majority, a clinical diagnosis could be made without angiocardiography and without cardiac catheterization. All that was usually necessary in order to arrive at a clinical diagnosis was to correlate the history, physical, fluoroscopic, roentgenologic, and electrocardiographic findings. In this series of cases, however, in addition to the above-listed examinations, the diagnoses were confirmed by 850 angiocardiograms, 450 cardiac catheterizations, as well as 300 autopsies. The order of frequency of the 13 noncyanotic types reported in this series was ventricular septal defect, patent ductus arteriosus, atrial septal defect, coarctation of the aorta, isolated pulmonary stenosis, aortic and subaortic stenosis, primary endocardial fibroelastosis, idiopathic dilatation of the pulmonary artery, vascular rings, anomalous left coronary artery, aortic septal defect, ruptured sinus of Valsalva, and glycogen storage disease. The order of frequency of the 9 cyanotic types was as follows: tetralogy of Fallot, complete transposition of the great vessels, pulmonary stenosis with atrial and ventricular septal defect, Eisenmenger complex, tricuspid atresia, persistent truncus arteriosus, levocardia, Taussig-Bing heart, and anomalous drainage of all pulmonary veins.


A case is reported of a 7-week-old male infant who died of heart failure from unknown cause. He was normal in all respects by physical examination except for slight cyanosis due to enlargement of the heart. Autopsy revealed an enlarged heart with right ventricular hypertrophy, and a 2-chambered left atrium separated by a thick membrane through which there was a very small opening 1 mm. in diameter. The pulmonary veins emptied into the posterosuperior chamber. The anteroinferior chamber communicated with the left ventricular chamber through a normal mitral valve. No other congenital abnormalities were present. The literature on this anomaly is reviewed. The clinical features are rapidly developing heart failure, absence of murmurs, tachycardia, right axis deviation, and right-sided hypertrophy. Opinion is expressed on the embryologic development of this anomaly. A plea is made for consideration of this anomaly in infants with heart failure presenting the above-listed clinical features. It might well lend itself to surgical amelioration. It is suggested that the surgical approach, when suspected, be made through the pulmonary artery to avoid entering the normal-appearing anteroinferior chamber, which would cause one to miss the lesion.


The authors discuss the findings in 9 cases of Ebstein’s malformation seen at the Mayo Clinic and review the data of 71 reported cases in the literature. Cyanosis was present in 84 per cent of the previously reported cases and was present at some time in each of the authors’ cases. Most patients showed cyanosis at birth or in early infancy, but in over one third of the patients there was a substantial delay (up to 52 years) in its appearance. It was practically always associated with an atrial septal defect.

The basic deformity of the malformation consists of a downward displacement of the posterior leaflet of the tricuspid valve, with adhesion to the ventricular wall, while the anterior leaflet may form a long veil-like structure. The size and thickness of the right ventricle are variable. The tricuspid orifice may be small enough to amount to a true stenosis. The large tricuspid leaflet may be pushed toward the free wall of the ventricle, occluding its cavity and preventing filling. During systole, blood behind the leaflet may be regurgitated into the atrium proper.

Auscultatory findings are variable. Double murmurs can be detected in almost half the patients,
sound is present in about one third of the cases. Phonocardiograms in 7 patients exhibited systolic and diastolic murmurs in all, murmurs of atrial contraction in 4, and loud third heart sounds in 3 instances.

Electrocardiographic findings are generally characteristic. Right bundle-branch block, usually atypical, is the chief finding. The tracings are apt to show R'R' deflections in right chest leads but frequently with low voltage of R and excessive splitting of R'. Increased sharpness and amplitude of P waves, and A-V conduction defects are also common. Supraventricular arrhythmias are fairly frequent.

The roentgenographic appearance is rather uniform. The heart is moderately to greatly enlarged, with a globular shape and a relatively narrow vascular pedicle in the posteroanterior view. Angiocardiograms have been used to demonstrate the location of the R-L shunt so commonly present. In addition to showing the atrial septal defect a huge thinned-walled right atrium can be shown. Because of stasis in this chamber the pulmonary circulation is poorly outlined.

Cardiac catheterization data of 27 cases were reviewed by the authors. Typically, the catheter enters a huge "atrial" chamber that occupies the space normally taken by the right ventricle. Mean atrial pressures are normal or increased. Right ventricular and pulmonary artery pressure curves are usually normal. Evidence of a R-L shunt at an atrial level is found in almost all patients with arterial oxygen unsaturation. Only 1 instance of a ventricular septal defect has been reported.

Among 32 autopsied cases, heart failure was present in one third. Seven patients died suddenly with no obvious cause. Pulmonary tuberculosis, cerebral abscesses, and other infections occurred in one third of the cases. Paradoxic embolism was proved in 2 other cases. Of 4 cases operated upon at the Mayo Clinic 1 survived and is greatly improved.

ENSIEBERG


The characteristic picture of embolic occlusion of a patent foramen ovale is illustrated by a case report. This syndrome should be suspected in a person without apparent heart disease who develops peripheral thrombophlebitis followed by pulmonary and later systemic embolism with persistent cyanosis and subsequently dies abruptly with intensification of the cyanosis and bulging neck veins. The electrocardiogram may show signs suggestive of pulmonary embolism.

When sudden closure of the foramen ovale occurs in a person with pre-existing pulmonary hyper-tension a further rise of pressure within the right heart ensues, producing abruptly bulging neck veins and marked cyanosis. Additional pulmonary emboli may further intensify the picture.

BERNSTEIN


Necropsy cases in patients 65 and 48 years of age are described. Previously, 10 patients surviving to 40 years of age or more have been described. The author points out with quotation and portrait, that Nicolas Steno (Niels Stensen) of Stensen's duct fame, described Fallot's tetralogy in 1673. Pre-Fallot contributions by British cardiologists, especially James Hope (1839) and Thomas Peacock (1866), are reviewed.

McKUSICK


Two patients with single coronary arteries were discovered accidentally during autopsy examinations. Both were white men, one 64 years of age and the other 56. Neither showed clinical evidence of absence of a coronary artery. In 1 patient the cause of death was arteriosclerotic heart disease with coronary occlusion and myocardial infarction. In the second patient no cardiovascular disease was clinically demonstrable. A single coronary artery, if not accompanied by evidences of cardiovascular disease or other anomalies of the heart, does not result in cardiac dysfunction. In individuals who have attained adulthood the condition of a single coronary artery is generally unassociated with any other cardiac abnormality. In children who die with a single coronary artery, it would appear that the premature death is attributable to the presence of other cardiac abnormalities. Longevity is apparently not altered by the presence of a single coronary artery. There is 1 case reported in the literature of an individual who died at the age of 80 years. The average age in adults is 45 years. Among 27 cases reported in adults, only 9 manifested evidences of heart disease as a cause of death. Even in these cases death was not connected with the presence of a single coronary artery. The total number of reported cases of single coronary arteries in adults and children is currently 43.

WENDKOS


In 12 patients with common atroventricular canal, operation has been performed at the Clinic. In the first 3, the atrial-wall technic was used. In
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the last 9, operation has been performed, since extracorporeal circulation through a mechanical pump-oxygenator had been developed and this technic was employed for them. In all cases, a prosthesis of ilaven sponge was utilized to close the defect. Six patients were male and 6 female. The ages varied from 10 months to 27 years, 5 patients being more than 20 years of age. All patients were asymptomatic, the severity of symptoms varying from a 10-month-old child desperately ill to a 20-year-old man experiencing only mild exertional dyspnea and occasional bouts of paroxysmal tachycardia. All patients underwent cardiac catheterization preoperatively. In 7 of the 12 patients mitral insufficiency was demonstrated at operation. Nine patients have survived operation. Seven of these have not as yet undergone cardiac catheterization postoperatively, although in 5 a normal dye curve was obtained at the end of the operative procedure. By clinical evaluation all these appear to have an excellent result. Another patient had a complete closure of the defect proved by cardiac catheterization 6 months following operation. In one patient in whom the atrial-well technic was used, cardiac catheterization 7 months postoperatively revealed a residual left-to-right shunt of 35 per cent at the ventricular level. Preoperatively, the over-all shunt had been 76 per cent. The patient seemed clinically improved. There were 3 deaths in this group of 12 patients, 2 in 10-month-old children and 1 in a 5-year-old boy. One of these children died 12 hours postoperatively. Severe mitral insufficiency was identified at operation, before repair. The atrioventricular canal was repaired but nothing was done to the eleft in the mitral valve. The evidence is good that the death resulted from this unrelieved mitral insufficiency. Common atrioventricular canal presents a grave problem to the patient afflicted. Fortunately, progress has been made both in diagnosis and in surgical treatment. Although problems in surgical therapy remain, there is reason for encouragement in the fact that repair has been accomplished in 12 patients, with 9 surviving and benefited by operation.

SIMON


A case report is presented of a female infant, who lived for 2½ months with persistent cyanosis, choking spells, and an increase in the size of the heart. At autopsy, a variant of the Taussig-Bing malformation was found. The aorta rose from the right ventricle, and the pulmonary artery arose from the right and left ventricle above a ventricular septal defect; about three quarters of the vessel arose from the left ventricle. The first branch of aortic arch was a common vessel that divided into right and left common carotid arteries. The second branch of the aortic arch was the left subclavian artery. Immediately beyond this was a coarctation, but arising from the posterior wall of the aorta just above the coarctation was an anomalous right subclavian artery. The right subclavian artery passed behind the esophagus to the right arm, and caused compression of the posterior aspect of the esophagus. In addition, a single coronary artery, which arose from the posterior aortic sinus, was present. Comment is made on the rarity of the occurrence of the right subclavian artery origin being proximal to a coarctation of the aorta. A review of some of the recently reported cases of the Taussig-Bing syndrome is presented.

Harvey


A very thorough and thoughtful study of fibroelastosis among the infants and children who came to autopsy at the New York Babies Hospital over a 20-year period, is presented. One hundred and ninety-nine hearts were available for analysis, of which 126 were instances of major congenital malformation. The authors state that the cases of endocardial fibroelastosis without associated malformation present a fairly uniform clinical picture, while the cases of fibrosis associated with congenital malformations present a varied clinical and pathologic picture. It seemed to the authors that the endocardial fibrosis in the latter group was secondary to varying pressures or currents in the flow of intra-cardiac blood caused by the malformation. All the congenital anomalies were classified, and an analysis of the sites of endocardial thickening in the types of congenital malformations was made. In considering the blood flows in these various groups, it was quite obvious to the authors that the elastosis occurred as a result of increased intracardiac pressure in a given area. The thickened valves could be reasonably ascribed to vibration, a result of eddy currents. In addition to the mechanical factors, the authors feel that partial anoxia contributes to the development of the condition. In none of the specimens was there any evidence of infection that could be considered the cause of the elastosis or the congenital malformation. An excellent review of the literature on this subject is presented.

Harvey


These authors report further on their study of congenital endocardial fibroelastosis. In a review
of the cases of fibroelastosis at the New York Babies Hospital, 17 instances were found in which the fibroelastosis occurred without any other congenital cardiac malformation. Among these 17 cases, were 2 in which there was a familial occurrence of the disease. The symptoms and clinical course were reviewed, as well as the physical findings and pathologic findings. In general, nonspecific symptoms appeared at about 6 months of age, and these were failure to gain weight, cough, labored breathing, and tachycardia. A terminal event was usually tachycardia with vomiting. Death occurred any time after the onset of the disease, during an acute attack, or at some later date after symptoms had been present for a period of time. In all instances the pathologic findings were essentially the same; the heart was large and dilated mainly because of hypertrophy and dilatation of the left ventricle. The fibroelastosis was more common in the left ventricle, and characteristically showed an increase in the fibrous tissue beneath the endocardium with an extension of fibrous strands into the myocardium. A review of the literature was presented, as well as a hypothesis for the cause. The authors feel that the familial instance suggests that there may be some genetic metabolic defect resulting in either deficiency or abnormality of an enzyme concerned with metabolism of the myocardium and that this defect leads to the development of the fibroelastosis. They consider the familial occurrence a strong point in this hypothesis.

Harvey


In 576 patients with congenital heart disease diagnosed by angiocardiography and cardiac catheterization, growth was considerably retarded, especially as far as weight was concerned. This retardation was especially great in patients with large left-to-right as well as right-to-left shunts and was attributed to tissue anoxia. In aortic coarctation localized anoxia in the lower half of the body may have been responsible. Retardation of growth, as well as the decrease in life expectancy, was parallel to the degree of cyanosis, but not to the pulmonary blood flow, and was much greater in boys than in girls.

Lepeschkin


As a result of observations in 14 patients, the conclusion is reached that the most important attribute of the syndrome is pulmonary hypertension due to increased flow, and that dilatation of the pulmonary artery is secondary to this. The major factor responsible for pulmonary hypertension is the interventricular communication, and the only surgical procedure logically possible for correction of the malformation is closure of this communication.

Coronary Artery Disease


Infarction of the interventricular septum was found in 30 of 62 patients studied. The most frequent localization was anterosetal (23 patients), whereas 5 were posteroseptal and 2 were anteroseptal and septal in location. Electrocardiographic localization was quite accurate with anterosetal infarction but it was faulty with posteroseptal lesions. Of the 30 patients with septal infarction, 11 died. It is believed that although this mortality figure is incorrect because of the selection of cases, infarction of the septum alters the prognosis of myocardial infarction unfavorably. Of 24 cases of bundle-branch block studied with special reference to the problem of septal involvement, one half the cases were found to have recent or old infarctions of the septum on postmortem examination.

Rosenbaum


A series of 166 cases of acute myocardial infarction was treated with a combination of heparin and Dicumarol as anticoagulants. The heparin was given intramuscularly in a solution containing 1.25 per cent carboxymethylcellulose. There was little discomfort and no hemorrhage at the site of injection of this material. The heparin was given until the Dicumarol began to be effective. The mortality in this series of 166 patients was 25.9 per cent. Hemorrhagic complications occurred in only 5 patients and in all of them it was mild. Hemorrhage into the myocardium occurred in 1 patient who died, but it was thought unlikely that the hemorrhage contributed to the death. There were 5 instances of thromboembolism among the 166 patients but none of these appeared at a time when the patients were believed to be under the full effect of the anticoagulant. In 22 per cent of the patients the condition of the patient changed from good to poor risk some 24 hours or more after admission. The author is of the opinion that such patients would be denied the advantages of anticoagulant therapy if the criteria of Russek were used in the selection of patients.

Rosenbaum

Malic dehydrogenase activity was determined in the serum of 21 healthy adults and 7 patients with myocardial infarction. Serum lactic dehydrogenase activity was measured in 42 healthy adults, 23 patients with a variety of clinical disorders and 22 patients with acute myocardial infarction. Serum zinc was measured in 8 patients with acute infarction. Lactic dehydrogenase activity of the serum was elevated in all patients with acute myocardial infarction rising on the first day, reaching a peak on the second or third day and falling to normal by the seventh to eleventh day. The malic dehydrogenase activity followed much the same pattern and the time course and degree of elevated activity were much the same as that described for serum oxaloacetate transaminase activity. The elevations of both enzymes were 2 to 10 times those found normally. The serum zinc concentration was significantly reduced in patients with myocardial infarction, falling on the day of infarction and remaining decreased for as long as 2 weeks; this constituent of the serum is also reduced in hepatic cirrhosis and pernicious anemia. Serum lactic dehydrogenase activity was not elevated in angina pectoris, coronary insufficiency or myocardial ischemia. It did rise in renal necrosis and parenchymal liver disease, renal infarction and subacute glomerulonephritis. In some patients with myocardial infarction complications caused a secondary rise of activity and delayed the return to normal. The changes in these metalloenzymes and in the serum zinc concentration are believed to occur early enough in the disease and to be sufficiently characteristic of myocardial infarction to be valuable adjuncts in the diagnosis of the disorder.

Rosenbaum


The occurrence of a C-reactive protein could be demonstrated in 94 per cent of 50 patients with myocardial infarction and typical electrocardiographic patterns. The positive reaction was as frequent as an elevation of the sedimentation rate and more common than leukocytosis and elevation of the temperature. It appeared before the rise of the sedimentation rate and disappeared before the latter had reached its peak levels. In cases of suspected acute coronary disease with nonspecific electrocardiographic alterations or a normal electrocardiogram, the reaction became positive only in the presence of an elevation of the sedimentation rate. The authors feel that a test for the presence of C-reactive proteins is a sensitive indicator and represents a valuable addition to other methods used to determine an active myocardial process in coronary disease.

Pick


According to the author persistence of the QS deflection in at least 4 precordial leads is suggestive of anterior ventricular aneurysm, while posterior aneurysm is characterized by persistent S-T segment elevation in leads II and III. Immediately after anterior infarction, giant positive T waves with slight S-T segment elevation may occur in the first 4 precordial leads. In the Wolff-Parkinson-White syndrome, the QS deflections in leads II and III may simulate old posterior infarction, while in chronic pulmonary disease a QS deflection localized to lead V, may simulate anterior infarction.

Lepeschkin


Histologic studies of the coronary arteries were made in 40 persons who died while having symptoms of coronary insufficiency and in 15 persons who died suddenly from other causes. The first group showed saturation with plasma of the walls of small arteries, hemorrhage into arteriosclerotic plaques in the larger arteries, intramural hemorrhages, stasis, and paretic dilatation of the smaller vessels. These are interpreted as due to coronary spasm, which is facilitated by the presence of arteriosclerosis. Coronary thrombosis is considered to be usually secondary to the functional spasm.

Lepeschkin


A comparison of health habits has been made between a test group of 100 patients with manifest coronary disease and a control test group of 200 people with the same distribution of ages and occupations but without coronary disease. Twenty-seven per cent of the test patients had average diets with a variety of foods and an apparent balance between caloric intake and energy output, while 60 per cent of the control test group had such findings. Seventy per cent of the test group had no regular exercise patterns, either at work or away from it, as compared with 30 per cent of the control test group. No influence of tobacco or alcohol was evident, but among the patients who survived coronary attacks 71 per cent had been using alcohol in moderation for from 1 to 10 years since the attack and claimed beneficial effects with less anxiety. There was evidence that the real culprits in coronary disease are not hard work, overexercise, or occupational stress, but sedentary living and poor health habits. Technologic advances are freeing more and more men from distasteful and onerous labor and more men will have time and money for leisure. This is
our modern challenge. We must guide men in the intelligent use of their abilities, resources, stamina, and interest in life if we would help them lead satisfying and happy existences. If we, as part of a creative minority, fail, we will find ourselves as part of a civilization unprepared for the economic riches it fought so hard to obtain.

Kitchell


Thirty-three patients with severe disabling angina pectoris were subjected to resection of the anginal pathway. Three patients (9 per cent) died of the operative procedure. Eighteen of the surviving patients had complete relief of anginal pain for from 1 to 11 years. Eight patients were believed to have had satisfactory results, and 4 obtained no relief. Although resection of the anginal pathways is not the final answer to disabling angina pectoris, the procedure does offer relief to certain carefully selected patients. The individual with angina and hypertension in whom the procedure is combined with extensive sympathectomy and splanchicectomy obtains the best results. The operation may offer great relief to patients with angina decubitus who have not had a recent coronary occlusion and who do not have a great functional overlay.

Kitchell

ELECTROCARDIOGRAPHY, VECTORCARDIOGRAPHY, BALLISTOCARDIOGRAPHY, AND OTHER GRAPHIC TECHNICS


The electrocardiograms in 16 patients with defects of the atrioventricular canal area, proved either at operation or post mortem, have shown a basic uniformity and may be highly distinctive in the differential diagnosis from usual types of atrial septal defect. The characteristic record shows a delayed excitation of the right ventricle of the partial right bundle-branch configuration, in general a left axis deviation and a QRS loop, as projected on the frontal plane, rotating counterclockwise, often placed superior to the isoelectric point. In some instances, a flattened, horizontally disposed figure-of-eight configuration of the QRS loop in the frontal plane may be seen. The basic pattern is modified by the presence of pulmonary hypertension and gross left ventricular enlargement caused by mitral insufficiency. The P-R interval is frequently prolonged.

Simon


The authors report that they have used the suprasternal puncture technic in 140 patients for hemodynamic studies. There were 68 patients with predominant mitral stenosis and, of these, 5 were considered to be in the very early stages of the disorder. The left atrial curve was characterized by a high and peaked curve, believed to result from an increase in the atrial systolic force, a well-developed intrasonic dip attributed to the suction influence of the left ventricle, and an angulation on the descending limb of the second sound wave regarded as the effect of obstruction to emptying of the left atrium. The pulmonary artery wave showed a presystolic pressure rise due to retrograde transmission of the large atrial a wave, and a broadening of the head of the curve with a displacement of the incisure to a higher level on the catacrotic limb so that the pressure response in the pulmonary artery resembled that of the aorta. The aortic curves showed a low and narrow head, a poorly developed incisure and rebound wave; these were features that the authors have encountered in all patients with mitral valve disease and that are thought to be caused by the reduction in cardiac output.

Rosenbaum


Electrocardiographic observations were made in 84 normal children and 41 normal adults at rest, in the upright position and during and after standardized heavy exercise. The exercise electrocardiogram seldom showed any significant disturbances in rhythm. The P-Q interval was not prolonged during exercise and in no case was it prolonged by more than 0.01 second above the resting value. The ventricular gradient as well as the mean electric axis of QRS tended to show a shift to the right immediately after exercise and then a shift to the left 4 minutes after exercise with T wave changes probably secondary to these axis shifts. After exercise the T waves decreased in amplitude initially and then increased in amplitude during the recovery period. When the T waves increased in amplitude, the increase was usually greater in children than in adults and when they decreased in amplitude, the fall was usually less in children. In orthostatic tests, electrocardiographic changes were less often found in children than in adults.
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