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ATHEROSCLEROSIS

One hundred forty-six males between the ages of 20 and 59 years underwent an oral fat tolerance test to determine whether the degree of lipaemia following the feeding of fat differed between three racial groups, the Bantu, the Cape colored, and the European, and to compare the findings with those obtained in age-matched patients with ischemic heart disease. The comparison of the two age-matched white groups showed that in males with established ischemic heart disease there was a greater and more prolonged postprandial lipaemia than in control subjects, manifest mainly at 6 hours and 7½ hours after feeding. There was no difference between the three racial groups in their tolerance to a test meal of fat. When a fat emulsion was administered intravenously, no difference in degree of lipaemia or rate of removal was found between the patients and the controls.

KURLAND


Various indices of lipid levels after fat ingestion were studied in a group of healthy medical students, a group of healthy middle-aged men, and a group of male patients with ischemic heart disease. After ingestion of radioactive-labeled fat, it was found that the group with ischemic heart disease demonstrated more marked and more prolonged lipaemia than the normal subjects, most striking 9 hours after fat ingestion. However, the 9-hour values for plasma optical density, total esterified fatty acids and lipid I\(^{131}\) did not completely separate the healthy from the diseased patients. In four patients with ischemic heart disease an abnormal fat-tolerance test, the simultaneous administration of heparin during a second test resulted in a much improved or normal response. In healthy subjects systemic use of heparin had no significant effect on fat clearance. It is suggested that the delayed clearance from the blood of ingested fat observed in patients with ischemic heart disease offers a means of separating healthy from diseased individuals more completely than fasting lipid levels and, therefore, may be of value as a screening procedure.

SAGALL


Six healthy men were given in sequence a cholesterol-free diet, a diet containing cholesterol from egg yolk, a cholesterol-free diet, and a diet with added crystalline cholesterol. These diets were identical in calories, fat, protein, carbohydrate, minerals, and vitamins. The fatty acid composition of the fat was also equivalent for all diets. Each dietary period was of 3 weeks' duration, and the diets were administered through an intragastric tube. Blood samples were analyzed for total cholesterol, total phos-
pholipid and triglyceride concentrations. During the initial cholesterol-free period, the serum cholesterol fell in each subject (mean 58 mg. per cent), the phospholipid fell (mean 44 mg. per cent), and the triglycerides fell (mean 47 mg. per cent). The addition of dietary cholesterol in the form of egg yolk caused a significant increase in the serum cholesterol concentration (mean 69 mg. per cent), in phospholipid concentration (42 mg. per cent), and a less significant rise in serum triglyceride concentration (33 mg. per cent). The serum cholesterol and phospholipid concentrations decreased greatly again when egg yolk cholesterol was removed from the diet. Amounts of dietary cholesterol from 475 to 1,425 mg. per day (two to six egg yolks) produced similar effects. When crystalline cholesterol was added to the cholesterol-free diet, less striking but significant increases in the serum cholesterol and phospholipid occurred.

Kayden


Serum cholesterol values from 95 normal male subjects were compared to those from 88 patients with coronary heart disease. The greatest difference between mean cholesterol values for these two groups was in the fourth decade, and this difference diminished with increasing age through the sixth decade. The authors suggest that the elevated values in respect to prediction of coronary heart disease may be more meaningful in subjects under the age of 40.

Sheps


The first part of this article compared the cholesterol values obtained by four different methods on 29 identical samples. The modified method of Zuckerman-Natelson was chosen because of its simplicity, reproducibility, and good recovery. The method involves direct extraction of serum by chloroform and sulfuric acid and development of a green color with acetic anhydride-sulfuric acid reagent added to an aliquot of the chloroform extract. The total serum cholesterol levels were measured by this method in 434 healthy farmers from 13 to 69 years of age in rice-producing districts. In these districts there is a high incidence of cerebral hemorrhage. The cholesterol levels were compared with the levels obtained in urban laborers and office workers in the same districts. The serum cholesterol levels were relatively low; this was presumed to be primarily due to the low-fat intake of the men in these districts. In men aged 40 to 49, the mean cholesterol level of the farmers was nearly the same as that of the urban laborers, but significantly lower than that of the urban office workers. The proportion of obesity in the farmers was smaller in comparison with that in the urban people, and the cholesterol levels of the fatter of the farmers and the fatter of the laborers were similar to those of the office workers with normal body weight. In males aged 13 to 19, there was no significant difference between the mean cholesterol levels of the farmers and of the urban population.

Kayden


Total serum cholesterol levels and low-density lipoprotein distributions were measured in men under 65 years of age. The results in 46 men, who were considered to have had myocardial infarction, were compared to the results in 48 men who had angina pectoris without myocardial infarction. The mean age, weight, and height of the two groups were not significantly different. The group with myocardial infarction had a somewhat higher level of total serum cholesterol (301 mg. per cent) than the group with angina pectoris (291 mg. per cent). The level of low-density lipoproteins was significantly higher in the patients with myocardial infarction than in those with angina pectoris. This difference was most apparent in the fraction Sf 12-400. The authors suggest that this may be due to a higher triglyceride content in men with myocardial infarction compared to men with angina pectoris.

Kayden


Rabbits made hyperlipemic by cholesterol feeding did not respond to intravenous infusions of inositol phosphatides with immediate rises in serum neutral fat and cholesterol levels as do normal rabbits. A delayed rise in neutral fat seen after 24 hours suggested that phosphatide infusions represent a lesser stimulus to lipid mobilization in hyperlipemic than in normal rabbits. Earlier work elsewhere could not be confirmed in that infusion of phosphatides intravenously weekly or semweekly for the initial 6 weeks of cholesterol feeding failed to alter the
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Development of mild aortic atherosclerosis. Nor
was regression of atherosclerotic plaques evident
when phosphatide infusions were begun 2½
months after cessation of cholesterol feeding.
Instead, increased aortic lipid deposit was noted
in most of the infused animals.

WHITE

Steinberg, D., Avigan, J., and Peigelson, E. B.: Efects of Triparanol (MER-29) on Cholesterol

Triparanol (MER-29) has been shown to inhibit cholesterol synthesis in animals, and the
serum and tissue levels of cholesterol in MER-29 treated animals were reduced. In animals treated
with MER-29, there was an accumulation of desmosterol (24-dehydrocholesterol) in the tissues,
and isotopic studies established that the major site of action of MER-29 was in the last step in cholesterol biosynthesis, the reduction of desmosterol to cholesterol. In nine patients, treatment
with MER-29 was always associated with the appearance of desmosterol in the serum, which accounted for from 19 to 43 per cent of the total circulating sterols. The free-to-total ratio for circulating desmosterol was not significantly different from that of cholesterol. Although there was a marked drop in serum cholesterol concentration (average 34 per cent) for all patients, the total sterols were depressed by only about 15 per cent. A method for determining desmosterol in the presence of cholesterol was presented; the usual analytic procedures yielded low total sterol levels when desmosterol was present. After intravenous injection of 2-14C-
mevalonate in patients under treatment with MER-29, the specific radioactivity of serum desmosterol was at least an order of magnitude higher than that of cholesterol. The specific radioactivities of the two sterols did not become equal until 2 weeks after injection of labeled mevalonate. The specific radioactivity of esterified desmosterol, low during the early hours, rose and became equal to the specific activity of free desmosterol at 24 to 48 hours after injection of labeled mevalonate. After injection of 2-14C-mevalonate, the peak specific radioactivity and total radioactivity in the unfractonated serum sterols during treatment with MER-29 were significantly lower than during control periods. These observations suggest that the mechanism of MER-29 in man is to inhibit the reduction of desmosterol to cholesterol and that the depression of serum cholesterol levels and total serum sterol levels is due to this inhibition.

Kayden


Previous work did not support the idea that tissue mast-cell depletion promotes hyperlipemia or atherosclerosis. In the present report, the author found in experiments on rats that hypercholesteremia and coronary lipoidosis did not affect the myocardial mast-cell count and that total-body depletion of mast cells did not interfere with the ability of rats to correct an experimentally produced hyperlipemia. The author concluded that the myocardial mast cell is probably not involved in the early stages of vascular lipid infiltration.

Kalmansohn

BLOOD COAGULATION AND THROMBOEMBOLISM


Lowenberg has considered intolerance to discomfort to a sphygomanometer cuff pressure of 150 mm. of mercury or less to be indicative of venous thrombosis and a tolerance to 180 mm. of mercury or greater as normal. Of 230 patients examined on admission to hospital, 25 were found to tolerate pressures of less than 180 mm. of mercury. None of these had or subsequently developed deep venous thrombosis. Among 107 patients evaluated postoperatively, the daily performance of this test was of little value. Twenty-two patients had false-positive results on the day of admission. The remainder showed wide variations in day-to-day readings and in the relative readings of the two legs. In no instance did this test assist in the establishment of an early diagnosis of deep venous thrombosis. Fifty extremities were tested in which phlebograms were also obtained. Acute deep calf vein thrombosis was present in 23 and absent in 27 extremities. The pressure-pain test was falsely negative in 22 per cent and falsely present in 26 per cent of the cases in which the correct diagnosis was reasonably certain. This test was proposed as an objective measurement of a subjective symptoms-tenderness but careful examination for calf tenderness was a more reliable sign. The cuff test is a benign one, and in some patients with equivocal tenderness a positive test on one leg with a normal response in the other may assist in the correct diagnosis. False-positive results were seen in patients with chronic venous disorders, degenerative disk disease, arterial disease, variety of bone disorders, acute superficial phlebitis, muscle tears, contusion, etc.

The clinical electrocardiographic and radiologic findings were reported in 72 patients with pulmonary embolism. The symptoms were due to diminished cardiac output or to infarction of the lung and consisted of faintness, substernal discomfort, and breathlessness without pleural pain or hemoptysis. Tachycardia was nearly always present. Arrhythmias, particularly atrial fibrillation, developed. A rise in jugular venous pressure was the commonest sign of massive embolism. Every patient with a significant fall in blood pressure had an obvious rise in venous pressure. Twenty patients had a fall in systemic blood pressure. Auscultation of the heart was of little value in making a diagnosis. Patients with massive embolism usually had pale, cold, cyanotic skin. Signs of pulmonary infarction included rapid, shallow breathing because of pleuritic pain but also dyspnea in the absence of pain. Fine rales at the bases, pleural friction rub, loss of resonance, and bronchial breathing were also present. In patients without evidence of circulatory change, the electrocardiogram was often normal. In the presence of massive embolus, the commonest electrocardiographic sign was inversion of T wave. Right ventricular delay and evidence of a positional change in the electrocardiogram were also noted. Radiologic changes included elevation of the dome of the diaphragm, clouding of the costophrenic angle, a density in the overlying lung, and changes in the pulmonary vascularity. Of 53 patients treated with adequate anticoagulation for 2 weeks, one died of acute renal failure, one suffered a recurrence of embolism during treatment, and two had recurrence of embolism after completion of treatment.

Kurland


The procedure described in this article for the measurement of prothrombin time was carried out with disposable equipment and prepared reagents. A suspension of thromboplastin with calcium and sodium chloride was prepared. The pipets employed in the study were either the usual white blood cell counting pipet or a 12-in. length of polyethylene tubing (internal diameter 0.023 in., and external diameter 0.038 in.) marked at 2 and 4 in. from its distal end. Blood for the test was obtained from freely flowing fingersticks made with disposable lancets. Blood was drawn into the pipet to the 2-in. mark and the thromboplastin solution then drawn in until the mixture reached the 4-in. mark. The mixture was then expelled onto the surface of a glass slide. The slide was rotated and tilted once per second over a white background. The reaction time was obtained from a stopwatch, which was started when the mixture was expelled onto the glass slide and stopped when definite slowing was observed in the movement of the mixture. Animal experiments were also carried out in dogs that received anisindone as the anticoagulant. The prothrombin times obtained by the simple test were compared with those obtained by the Quick method. The data indicated that both tests were approximately equal in precision, being reproducible to ± 4 per cent. The comparison of 136 concurrently performed Quick and rapid polyethylene tube and slide tests on 16 normal and anticoagulant-treated patients revealed that Quick test prothrombin times were 0.19 second (± 0.12 second) longer than the fingertip test.

Kayden


Postoperative venous thrombosis and pulmonary embolism are rare in African and Indian patients; higher levels of fibrinolytic activity have been shown in Africans compared to whites. It was thought that this low incidence of postoperative thromboembolism might be explained by differences in blood clotting and blood lysis in the three racial groups. Fibrinolytic activity in these three groups in Durban was therefore studied, with special reference to changes during and after operation. Fibrinolytic activity was determined as the euglobulin-lysis time (ELT). The mean ELT in whites was 152 minutes—a level higher than in either an African-staff control group eating an approximate white diet, or the poorly fed African patient control group. Sex, the presence or absence of shock, and length or severity of operation were not found to influence the over-all change in fibrinolytic activity. Plasma fibrinogen levels in the three groups fell within normal levels for white patients. During operation, there was no significant change in fibrinolytic activity in Indian and African patients. In all groups of patients, there was an increase in ELT during the first two postoperative days, followed by a fall in ELT during the next 6 days. Postoperatively, fibrinogen levels increased in African and white patients. Since no cases were studied in which postoperative thromboembolism developed, it was difficult to correlate the findings with the incidence of thromboembolism.

Kurland

Circulation, Volume XXV, May 1962

A strictly controlled clinical trial of long-term anticoagulant therapy in the treatment of cerebrovascular disease was undertaken in which the progress of patients receiving treatment was compared with that of a similar group not so treated but managed in the same way in all other respects over the same period of time. It was attempted to decide whether anticoagulant therapy increased life expectancy, decreased the incidence of further cerebral accidents, and influenced the functional capacity of patients. Admission to the series was made without knowledge of pretreatment group, and assigned by a randomization table. The patients did not know to which group they belonged. Aim of treatment in the high-dosage group was to maintain the prothrombin time by the Quick one-stage method at a level two to two and one-half times the control value. The incidence of further nonfatal cerebrovascular accidents did not differ significantly between the two groups, but there were four deaths from cerebral hemorrhage—one from hemopericardium in the high-dosage group compared with none in the low-dosage group. The trial was therefore stopped in its present form earlier than had been anticipated. The present study strongly indicates that the general use of anticoagulant therapy in patients with cerebrovascular disease, selected as in this trial, is hazardous because of the risk of cerebral hemorrhage. This risk is present even when anticoagulant therapy is carefully controlled and the prothrombin time maintained at a level which is generally accepted to be safe.

KURLAND


Coumarin drugs in addition to depressing prothrombin and factor VII (accelerator) levels, depress factor IX (plasma thromboplastin component) and factor X (Stuart-Prower factor). These latter two factors are needed for generation of normal plasma thromboplastin, and deficiencies in these factors due to coumarin drugs might be expected to show prolongation of whole blood silicone clotting time. Detailed coagulation studies were performed on 28 patients who received either bishydroxycoumarin or warfarin sodium by the oral route. The technic for whole-blood clotting time involved siliconized needles and collection of the blood through polyvinyl tubing directly into silicone-coated glass tubes. The whole-blood silicone clotting time was prolonged in 26 of 28 patients, whereas the clotting time in plain glass tubes was slightly prolonged in only 10 patients. The comparison between silicone whole-blood clotting time and prothrombin time indicated that prolonged one-stage prothrombin values almost always have prolonged clotting times. The two-stage prothrombin measurement did not correlate as well with silicone whole-blood clotting time. There was little or no correlation between accelerator levels and prolongation of clotting time. Patients with prolonged silicone clotting time had impaired thromboplastin generation. No difference was found between bishydroxycoumarin and warfarin in the effect upon depression of clotting factors. These studies indicate that factor IX and factor X deficiencies correlate directly with prolongation of the silicone clotting time in patients receiving coumarin drugs. It is suggested that such deficiencies rather than depressed prothrombin and factor VII levels may be largely responsible for the antithrombotic activity of these drugs.

KAYDEN


One-stage prothrombin times were carried out, utilizing as thromboplastin saline extracts of brain, heart, lung, femoral arteries and veins, pulmonary arteries and veins, thoracic aorta, and vena cava of dogs. It was found that brain and lung were approximately 10 times more active than heart, femoral artery and vein, and pulmonary vein, and 50 times more active than pulmonary artery, thoracic aorta, and vena cava. The dilution curves were parallel for the members of the muscular and elastic vessel groups respectively. Comparison of thromboplastic activity of saline in which pieces of tissue had been soaked indicated that the solubility of thromboplastic substances from intact muscular vessels was 20 to 40 times greater than from elastic vessels. The author suggested that the factor that determines whether or not thrombosis takes place adjacent to an injured or cut vessel is a local thromboplastin concentration. This thromboplastic substance arises from the wall of the blood vessel with muscular vessels liberating a large amount, fibrous vessels an intermediate amount, and elastic vessels a small amount. The actual local concentration of thromboplastin is dependent upon the rate of liberation and the rate of dilution and removal by the blood stream. Thus, stasis acts to promote thrombosis by decreasing the rate of removal of thromboplastins.
This occurs whether the stasis is general, as in an anastomosis in a vessel with an unrelieved distal block, or local, as in the case of pockets or wrinkling from a marked size discrepancy between two vessels or inaccurate suture technique. It is proposed that the local thromboplastin concentration is the important factor determining the immediate success of vascular suture, anastomosis, graft, or endarterectomy rather than mechanical considerations based on vessel size.

**Sheps**


The thrombotest method of Owren for the control of anticoagulation was evaluated and compared with the Quick one-stage prothrombin time determination in a series of patients. The thrombotest method was found to be a practical, simple, inexpensive, reliable method for the control of anticoagulation therapy. For the most part the results were similar to those obtained with the prothrombin time determinations, but a comparison of the level of anticoagulation obtained with the tests revealed that cases within the therapeutic range according to prothrombin time measurements frequently fell beyond that recommended by Owren. If future data substantiate Owren's criteria for a therapeutic range of anticoagulation as effective in the prevention of further thromboembolic episodes, then patients could be kept more safely on smaller doses of anticoagulants than now employed.

**Sagall**


Ligation of the inferior vena cava was carried out on 20 patients over a period of 7 years. The majority of the patients had some form of heart disease and varying degrees of congestive heart failure. Four patients had emboli occurring during effective anticoagulation treatment, but details are lacking. Nine patients sustained emboli following femoral vein ligation. The vena cava contained thrombus in four patients, and in two of these thrombectomy was necessary because the thrombus extended above the level of the proposed ligation. In the postoperative period, the foot of the bed was elevated and the patient was given leg exercises. Early ambulation was carried out with the legs in elastic bandages. These supports were maintained as long as necessary to prevent swelling of the ankles. This period varied from 2 months to 21/2 years. One patient died on the fourth postoperative day in severe congestive heart failure. Another patient died of a fresh myocardial infarction on the sixteenth day after the operation. None of the patients had a recurrent embolus after ligation of the inferior vena cava. The number of patients treated with anticoagulants postoperatively is not given. Most patients developed edema of one or both legs immediately after the operation, and some complained of discomfort. This edema, if unilateral, did not necessarily affect the leg involved before the operation. One patient sustained a migrating superficial thrombophlebitis the month following operation. Studies revealed no underlying disease, and phlebitis did not recur. Ten patients were followed for periods varying between 6 months and 7 years (average 2½ years). All complained of edema of the ankles and heaviness in the legs at the end of the day. This was relieved by elevation and could be controlled by elastic support. Two patients developed ulcera.tions, and in one this was resistant to treatment. Most patients developed prominent superficial veins and two patients developed varicosities, which became "inflamed" from time to time. Ligation of the inferior vena cava is recommended as the most effective method of treatment in pulmonary embolism.

**Sheps**


Platelets were tagged with diisopropyl fluorophosphate containing P32 in 29 atherosclerotic subjects receiving Dicumarol therapy, and in 31 atherosclerotic controls. The patients receiving Dicumarol had elevated values for whole-blood clotting time, prothrombin time, plasma thromboplastin time, and platelet clumping time. Their platelet adhesive index was significantly lower than the control value, as well. The mean platelet turnover, 38,500 platelets per mm.3 per day in the treated group was significantly lower than that of the control group, which had a turnover of 58,200 platelets per mm.3 per day. The half-life of platelets in the treated patients was 3.84 days as compared to 2.86 days in the controls. There was no significant difference between the platelet counts in the two groups of patients. Of the various parameters measured, platelet adhesive index correlated best with the rate of platelet turnover. It is suggested that Dicumarol has an influence primarily on the external environment of the platelet rather than on its internal milieu, at least in the atherosclerotic individual.

**Ross**

*Circulation. Volume XXV, May 1962*

Methods for the partial purification of Hageman factor and plasma thromboplastin antecedent were described. A crude fraction, rich in activated Hageman factor and apparently deficient in other known clotting factors, was prepared from plasma believed deficient in plasma thromboplastin antecedent. In the same way, a crude fraction containing plasma thromboplastin antecedent was prepared from plasma deficient in Hageman factor. In the process of purification, Hageman factor seemed to be converted from an inactive to an active form. When partially purified activated Hageman factor and plasma thromboplastin antecedent were incubated together, clot-promoting activity evolved. Studies suggested that plasma thromboplastin antecedent was converted during this procedure from an inactive to an active form by an enzymatic process. Activated Hageman factor and activated plasma thromboplastin antecedent were inhibited by di-isopropyl fluorophosphate and by a fraction of normal plasma. These observations were in agreement with the hypothesis that activated Hageman factor initiated clotting by an enzymatic process. The results also may explain the asymptomatic nature of the Hageman trait.

Kayden


Blood coagulation time (Lee-White) was found to have decreased an average of 16 to 35 per cent intraoperatively in 30 patients having major surgery under general anesthesia, while a decrease of 7 to 11 per cent was observed in 11 patients having surgery under spinal anesthesia. Concomitantly, the platelet count (Fonio) increased an average of 29 to 44 per cent in the former group and 11 to 42 per cent in the spinal group. Three patients having thromboembolism postoperatively had particularly striking platelet and coagulation alterations. Previous studies had indicated that megakaryocytes in the pulmonary capillaries may produce platelets and that this production may be increased during stress. Therefore it was suggested that during major surgery a demonstrable temporary hypercoagulable state often occurs and that in selected instances anti-coagulation may be called for to prevent thromboembolism.

Rogers


Data are presented of an experimental study designed to separate the anatomic effects of pulmonary hypertension from the effects of multiple pulmonary emboli. The studies were performed in dogs and multiple unilateral pulmonary emboli were produced with autologous whole blood clots, autologous fibrin, and inert plastic beads. The animals receiving fibrin or clotted blood emboli developed after 24 hours several distinct types of pulmonary vascular lesions. These consisted of organized or partially organized clots in the lumen of arteries with minimal lesions in the wall, with the exception of a peculiar vacuolated aspect of the endothelium that was in intimate contact with clots, and peripheral arteriolitis without alteration in the arteriolar wall, accompanied by focal hemorrhage, usually localized at the branching sites of vessels. There were no anatomic traces of pulmonary hypertension discovered and no such anatomic changes were found following plastic bead emboli. It was concluded that in this type of experiment, the arteriolitis found was probably the result of a fibrin factor. Furthermore, pulmonary hypertension and hypersensitivity could be eliminated as the causal agent of the vascular lesion of these experiments.

Sagall


In two groups of cases with pulmonary embolism the behavior of serum lactic dehydrogenase (LDH) activity was examined. The 14 cases in the first group had pulmonary embolism or infarction and, in six of these, measurement of serum LDH activity was made during the period between the thromboembolic episode and death. A significant rise of LDH was found in all six instances. In the second group, 11 patients were studied who showed strong presumptive evidence of pulmonary infarction. The diagnosis in these cases was based on clinical, roentgenographic, electrocardiographic, and laboratory data. In each instance an increase of serum LDH activity following the pulmonary embolism or infarct was observed. This increase in serum LDH activity appears to present an objective criteria for diagnosis of pulmonary infarct. It is recognized that
increased serum LDH activity is not specific, since it appears also in other diseases, particularly in myocardial infarction. Therefore, an elevated serum LDH activity must be correlated with other manifestations of pulmonary infarction. Increased serum LDH activity, hyperbilirubinemia, and normal serum glutamic oxaloacetic transaminase activity in a patient with pulmonary or cardiac symptoms should be considered to be indicative of pulmonary infarction.

**NEUFELD**


In this paper the author's main concerns were the reasons for varying results in different series of patients used for evaluation of anticoagulant therapy. The following criteria for planning of studies and evaluation of results in treatment of thromboembolic disease with anticoagulants were suggested. Selection of patients is one of the most important problems. Only random sampling of cases can be significant for evaluation and conclusions. The proportion and percentage of each subgroup in the studied as well as the control series should be indicated. The number of cases in the different subgroups has to be large enough to warrant statistical significance for conclusions. Only physicians who have had experience with anticoagulant treatment should be involved in these studies. The clinician must know the optimal levels as determined by the laboratory he is using. This is important, since investigation of various studies has revealed that in many instances the patients were actually receiving the therapeutic range a very small percentage of the time. The technic used for controlling the anticoagulant therapy is of great importance. The Quick test with Link-Shapiro modification and a 12.5 per cent dilution is recommended. Of the commercially available thromboplastin preparations, Acuplastin is recommended as the most satisfactory. In cases in which hemorrhage occurs, within the usual therapeutic range, a search for bleeding points should be made. In a study dealing with strokes, inclusion of a substantial number of hypertensive patients will influence the figures because of an increased incidence of bleeding. In investigations dealing with strokes, special emphasis should also be made on the percentage of embolization, thrombosis, and hemorrhage. Evaluation of treatment in patients with angina pectoris is very problematic, since statistical data are difficult to obtain from subjective symptoms.

**NEUFELD**


Coagulograms, as described by Horn, Kovacs, and Altman, were performed in patients with congenital and acquired heart disease. The coagulograms were graded as increased, decreased, or normal on the basis of a series of tests of coagulation. These tests consisted of (1) recalcification time, (2) prothrombin time, (3) thrombin time, (4) thrombin time (toluidine blue) (5) serum accelerator factor, and (6) fibrinogen B. One hundred forty-two patients (390 coagulograms) have been studied before and after surgery; 52 were men and 90 were women and the ages ranged between 21 to 50 years. Sixty-eight patients were class II of cardiac functional capacity (according to A. N. Bakulakew's classification), 38 were class III, and 36 were class IV. Among the patients of class II, 36 had coagulograms with decreased coagulation and 11 with normal coagulation. This is explained on the basis of high incidence of young patients in this group (38 below the age of 31) with a relatively short history of the disease. Among the 38 patients of class III, 21 showed diminished, 15 increased, and only two patients had normal coagulograms. Among the 36 patients of class IV, 20 showed increased coagulograms. Thrombi were found at surgery in the right atrium or appendage in 20 cases of class IV. The incidence of increased coagulability of blood is believed to be in direct proportion to the severity of the cardiac disease. Among 16 patients with normal coagulograms, 11 belonged to class II. In the same time in cases of classes III and IV the coagulogram was only occasionally normal. It is believed that coagulograms are important in the choice of the anticoagulant and its dosage.

**NEUFELD**

**CONGENITAL ANOMALIES**


Four cases of Ebstein's disease were studied. A short protodiastolic sound, best heard at the lower end of the sternum and radiating to a variable extent, was heard in all cases. This occurred 0.11 to 0.16 second after the second sound and may have represented a late tricuspid opening snap. A presystolic sound was also heard, occurring 0.12 to 0.17 second after the onset of the P wave. With rapid heart rates, these extra sounds may fuse, resulting in a summation gal-
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The authors report two cases of arteriovenous aneurysm of the great vein of Galen occurring in infants who died in the newborn period. A review of 24 previously reported cases revealed that the symptomatology included dilated face and scalp veins, proptosis, convulsions, headaches, the effects of subarachnoid and intraventricular hemorrhage, mental retardation, focal signs of the central nervous system, and failure to thrive. Death occurred under the age of 2 in the majority of cases and the patients infrequently lived to reach adulthood. The arteriovenous shunt may have a marked effect upon the cardiovascular system; both patients reported upon in this article died in the newborn period of heart failure. One infant had persistent hypoglycemia, which had not previously been described. Surgery appears to hold out the possibility of cure in some cases, provided that the diagnosis is made before the patients start to decline rapidly.

Karpman


Four types of coarctation are presented. The fetal coarctation complex consists of a wide nonconstricting coarctated area associated with a small left side of the heart and a large right side related to pulmonary hypertension. Most of these children died early from the effects of the lesion, but some who survived and developed constriction of the lesion entered a transitional stage. Survival from this phase leads to what has been termed the converted adult coarctation. In this instance there is no right ventricular hypertrophy; and clinically the appearance is that of adult coarctation. At surgery, however, these patients have a long area involved. The adult type of coarctation consists of a constrictive lesion in the region of the ductus arteriosus with subsequent effects on the left side of the heart. Surgery is delayed until the age of 5 or 6, if possible. When the infant presents with severe heart failure or there is a lesion complicated by intracardiac or extracardiac shunt, then surgery may be necessary at an earlier age. A clinical experience with 100 cases of what was diagnosed clinically as adult coarctation was reviewed. There were only three deaths.

Sheps


One hundred and eighty-six children with atrial septal defects were evaluated to determine whether complicating lesions or defects in atypical sites could be recognized. Of the 136 patients with ostium secundum lesions, 13 had pulmonary stenosis, 10 had partial anomalous pulmonary venous drainage, two had mitral stenosis, one had rheumatic mitral incompetence, and 14 had an associated ventricular septal defect. Of the 96 patients with isolated atrial septal defects, fatigue was the most common symptom. Three patients had mild congestive
heart failure. Splitting of the second heart sound was heard in all patients but varied with respiration, particularly in patients with smaller defects. A faint thrill was described in two patients. The left atrium was slightly enlarged in four patients, and the hilar vessels were prominent in all but five patients. Pulmonary hypertension was present in five patients, all of whom had significant cardiac symptoms; in the latter group, the intensity of pulmonary valve closure was noticeably increased, the heart size tended to be larger, and the height of the R wave in V1 was greater than 16 mm. in every case. In patients with ostium primum defects, enlargement of the left atrium was more common, clinical evidence of mitral and tricuspid insufficiency and left ventricular enlargement were common, and the electrocardiogram tended to show left axis deviation with a counterclockwise frontal vector. Patients with common atrioventricular canal had more disability and cyanosis, pulmonary hypertension, and right ventricular enlargement.

**Kalmansohn**


Study of four cases of transposition of the great vessels in children aged 1 1/2 to 5 1/2 years showed that a hemodynamic diagnosis may be made in the following circumstances: The presence in the pulmonary artery of blood with higher oxygen saturation than that in a systemic artery, the presence in the left ventricle (where the highest oxygen saturation of blood is found) of a systolic pressure exceeding that in the right ventricle, and demonstration by selective angiocardiography, with injection of contrast medium into the left ventricle, that the pulmonary artery arises from this chamber. Catheterization of the aorta via the right ventricle, or selective angiocardiography with injection into this chamber, often provides only presumptive evidence by showing displacement of the aortic root anteriorly and to the right.

**Marshall**


The clinical and laboratory features of corrected transposition of the great vessels are briefly reviewed. The authors describe a further case with both atrial and ventricular septal defects in a woman who has had an uneventful pregnancy. They draw attention to signs of inverted activation of the ventricular septum obtained from intraarterial electrocardiography. An initial negative deflection in the "right ventricle" and an initial rS complex in the "left ventricle" indicate a right-to-left depolarization of the septum, the converse of the usual finding.

**Marshall**


The clinical characteristics of familial obstructive cardiomyopathy were studied in 23 persons covering two generations. None had unequivocal evidence of heart disease. Three patients died at 18, 21, and 40 while the living patients ranged from 4 to 37 years. There was an approximately equal distribution between the sexes. One man died suddenly at the age of 40 of congestive heart failure with a history of cardiomegaly at 35 and chest pain at 39. An 18-year-old man died suddenly with a negative past history. A 21-year-old woman had a history of rheumatic fever at 8 and 13 without evident organic residue, a fruitless laparotomy at 19 for abdominal pain, and sudden exitus at 21 while running for a bus. Only one patient had been in congestive heart failure; two patients developed atrial fibrillation; all had normal blood pressures. Extra sounds were common in these patients. Systolic murmurs were heard in five patients and diastolic murmurs in four patients. A right atrial P wave was found in five patients, left bundle-branch block in one, left axis deviation in two, and abnormal T waves in several patients with inversion being present in lead III in all patients without left axis deviation. Seven of the nine affected patients had cardiomegaly by x-ray with right atrial enlargement in six patients, left atrial enlargement in seven patients, and left ventricular enlargement in five patients. This condition simulates Ebstein's syndrome but without a pattern of right bundle-branch block. Cardiac catheterization and angiocardiography were not performed because of the danger of arrhythmia and sudden death.

**Kalmansohn**


The jet of blood traversing the narrow orifice in congenital pulmonary stenosis raises a shallow bulge at the point of impact with the anterior wall of the pulmonary artery; this is visible in
ABSTRACTS

angiocardiograms. A second, higher projection may be seen in pulmonary stenosis and especially in Fallot's tetralogy, and has been attributed to the reflection of the pericardium from the dilated pulmonary artery. The authors believe, however, that the second projection is due to contrast in a blind ductus arteriosus; that is, a ductus that has undergone obliteration in its central part, leaving pouches or diverticula arising from both aorta and pulmonary artery.

MARSHALL


The clinical findings in peripheral pulmonary stenosis were analyzed on the basis of 47 cases described in the literature and 11 cases observed by the authors. In 38 cases the stenosis was associated with other cardiac malformations, and the clinical findings were dominated by the latter. Of the 20 cases with isolated stenosis, five had stenosis of the smaller branches, 10 stenosis of the main branches, two had combinations of both, and three had a supravalvular stenosis of the main pulmonary artery. An abnormal murmur with an atypical localization can be an important diagnostic clue; it was systolic in nine cases of peripheral stenosis, whereas in four cases it showed a continuation into the diastole. Roentgenologic and electrocardiographic signs of right ventricular hypertrophy appear only in cases showing marked elevation of pulmonary pressure, and the diagnosis can be made with certainty only by means of cardiac catheterization or selective angiocardiography. The latter demonstrate areas of localized narrowing, accompanied by poststenotic dilatation in peripheral but not in supravalvular stenosis. Stenosis at the origin of a main branch can be demonstrated only in a slightly oblique position. A characteristic sign is a localized pressure gradient; if this gradient is present also during diastole, the presence of multiple stenoses became probable. Marked poststenotic hypertension is common in multiple stenosis of smaller branches, but rare in that of the main branches or in supravalvular stenosis. Intracardiac phonocardiography usually demonstrated a localized murmur peripheral from the stenosis.

LEPESCHKIN


Analysis of roentgenologic signs of aortic coarctation from 589 published cases showed that signs suggestive of the condition are a wide as-

cending aorta, with a prominent or calcified knob or even a prestenotic aneurysm; these signs were present in less than one third of the cases. Further suggestive signs are a small or absent aortic knob, left ventricular hypertrophy, or a combination of the two, especially in young persons; these signs were present in one third to one half of the cases. Characteristic signs are esophageal displacement by a poststenotic dilatation of the aorta, a visible internal mammarian artery, dilated and varicose lateral thoracic arteries, or a notched collum sepalae; these signs were present in less than one third of the cases. Further characteristic signs are a dilated left subeliac artery or a double contour of the aortic knob (present in one third to two thirds of the cases) and indentations in the ribs (present in more than two thirds of the cases). Definite diagnostic signs are a notch in the external contour of the aorta at the site of the stenosis (present in less than one third of the cases) and large pulsations in the left subeliac artery in the presence of small pulsations in the descending aorta in the roentgen kymogram (present in one third to two thirds of the cases). In one personal case the only sign of coarctation was dilatation of the entire aorta in a young girl, while in another case it was a dilated, slightly calcified poststenotic descendent aorta.

LEPESCHKIN


Four hundred and fifty patients with ventricular septal defects were studied over a 10-year period to determine the natural course of the disease. Serial catheterizations were performed in 32 patients; of these 20 were below the age of 2, 11 were between 2 and 7 years of age, and one was 15 years old at the time of the initial catheterization. In 26 patients the interval between the first and second catheterization was 2 to 5 years. In the first 2 years of life there was a high incidence of pulmonary hypertension and a marked increase in the pulmonary blood flow. There was a tendency for the pulmonary blood pressure and flow to decrease after the first year or two of life. In three patients, signs of infundibular stenosis appeared; the latter was thought to occur in a heart that had the morphologic characteristics of tetralogy of Fallot during infancy, but with the right ventricular obstruction becoming more severe with age. A progressive rise in pulmonary resistance occurred in only three
patients. The results suggested that surgery should not be attempted before the second or third year of life.

Kalmanson


The authors reviewed the clinical, physiologic, and surgical aspects of a series of 61 cases of patent ductus arteriosus in infancy. In 14, all of whom had a typical continuous murmur, there were no complicating factors. Thirty-seven had pulmonary hypertension, with pulmonary artery systolic pressures between 30 and 95 mm. Hg; in none of these was the shunt predominantly veno-arterial. Five had major associated anomalies, including ventricular septal defect, pulmonary stenosis, and deformity of the mitral valve. The remaining five died from cardiac failure, and necropsy showed that the ductus was the sole congenital lesion. In general, the clinical picture of patent ductus arteriosus in the infant differs considerably from that in the older child. The symptoms are indistinguishable from those of ventricular septal defect. In many cases the diagnosis can be made on clinical findings. Cardiac catheterization, however, is essential in the more severe cases in order to determine operability. Cases in which there is evidence at catheterization of additional arterIALIZATION at the ventricular level present a difficult problem. In such cases a balloon-tipped catheter may be used to produce temporary closure of the ductus. Operation was uneventful in the 14 uncomplicated cases. There were six operative or postoperative deaths in the group of 37 with pulmonary hypertension; all but one of the remaining 31 had satisfactory results from surgery. In view of the likelihood of gradual deterioration, every poorly tolerated case should be submitted to surgery. The earlier the operation, the more quickly does the heart return to normal size.

Marshall


Intensive clinical and laboratory investigations were performed on four children varying in age from 5 months to 26 months with typical features of the Marfan syndrome. All four patients had radiographically demonstrable aneurysms of the aortic sinuses of Valsalva and dilatation of the ascending aorta and the main pulmonary artery. Phonocardiography demonstrated accentuated pulmonic closure, mid or late systolic murmurs, and in one case an apical pansystolic murmur. The accentuated second sound was attributed to the proximity of the dilated pulmonic artery to the anterior chest wall. The authors stressed that cardiovascular complications are the leading cause of death in this syndrome, but note that respiratory complications may be one of the serious fatal causes, especially in the pediatric age group. They emphasized that the cardiac lesions of the Marfan malformation may have their onset at a very early period of life, and may produce only minimal clinical cardiac findings.

Karpman


Two patients with congenital supravalvular aortic stenosis are described clinically and pathologically, apparently the sixth and seventh to be reported. Both were mentally retarded white males. One was known to have a loud systolic murmur since shortly after birth, otherwise asymptomatic, and was examined at necropsy after a post tonsillectomy hemorrhage at age 13. The other had a similar murmur, some features of the Marfan syndrome, and died at age 24 of congestive failure and nephritis progressive for 13 years. At necropsy, the left ventricles of both hearts were hypertrophied. In the first heart, a sharply outlined annular constriction lay at the upper margins of the sinus of Valsalva, producing stenosis of about 50 per cent. Microscopically, it consisted of hyalinized fibrous tissue. In the second heart a more constricting and irregular ring was found in a similar position, with the additional findings of nodular, thickened commissures of the aortic valve and a hypoplastic aorta. A 250-mlg. parathyroid adenoma was also noted in the second patient. Hyalinized fibrous tissue was again evident microscopically in the ring. The basic lesion of true supravalvular aortic stenosis is described as a defect in the intima and media at the level of the plica about the sinuses of Valsalva, with secondary hypertrophic and degenerative changes. Distinctions are drawn between this lesion and supravalvular membranes.

White


Diagnostic catheterizations were performed in 24 patients with atrial septal defect as the sole cardiac defect. Various parameters relating to
the left-to-right shunt were studied and are reported. It was found that in descending order of correlation between the measured or calculated parameter and shunt these were right ventricular as compared to left ventricular distensibility, left ventricular minus right ventricular systolic pressure, systemic minus pulmonary vascular resistance, and systemic minus pulmonary mean arterial pressure. In these relations allowance must be made for spurious mathematical correlation.

**SAGALL**


In some cases ventricular septal defect (VSD) coexists with infundibular or valvular pulmonary stenosis, which, by limiting pulmonary blood flow, protects the lung vessels. Twenty of a total of 140 cases of VSD studied by the authors were in this group. Recurrent chest infections were less common than in 20 cases of "unprotected" VSD with large left-to-right shunts. The electrocardiogram showed left ventricular diastolic overload in nine cases, with no cases of systolic overload, as compared with 16 and seven cases in the "unprotected" VSD group. The mean systolic pressure gradient across the pulmonary valve was 59 mm. Hg and the pulmonary "capillary" pressure averaged 9 mm. Hg as compared with 15 mm. Hg in the "unprotected" cases. The condition differs from Fallot's tetrad in that the predominant shunt remains from left to right. With the passage of time, however, a gradual evolution may occur with eventual transformation into the picture of the tetrad.

**MARSHALL**


During the course of 1 year the authors saw five patients whose aortas showed two separate curved portions with a kink corresponding to the ligamentum arteriosum between them. In the anteroposterior projection the lower curvature could be mistaken for a mediastinal tumor, but the lateral tomogram permitted a correct diagnosis. That the kink did not correspond to a coarctation of the aorta could be shown by means of roentgen kymography, which demonstrated equal amplitude of pulsation below and above the kink. Of 40 patients with true coarctation, all showed a definite difference in this amplitude except two patients who had developed extensive anastomoses. That the kinking was not caused by senile dilatation of the aorta, which was held back by the ligament, was shown by the fact that two of the patients were young persons with other congenital anomalies; this condition probably corresponds to an abortive aortic coarctation. A sixth patient, who had a shallow notch in the region of the aortic isthmus without kinking, also did not show any clinical or kymographic signs of coarctation.

**LEPESCHKIN**


Although the association of Friedreich's ataxia and heart disease is well known, only approximately 100 such cases have been reported in the literature. The authors report two children with Friedreich's ataxia in whom the cardiac manifestations preceded the neurologic symptoms. Pathologically, the myocardium and conduction system are usually involved in a toxic, chronic, progressive myocarditis and the pericardium is occasionally noted to be involved with thickening, petechiae, and effusion. Although the coronary arteries have at times been found to have all stages of intimal proliferation to the point of obstruction, the endocardium and valves have never been found to be affected. The authors suggested that the central nervous system and cardiac pathology is probably caused by some metabolic defect that is transmitted as a recessive trait.

**KARPMAN**


The authors carried out 102 retrograde angiographies of the left ventricle from the femoral artery without any complications except persistent ventricular extrasystoles in one case. This method permits accurate anatomic localization of ventricular septal defects with left-to-right shunts even if the defect is too small to be recognized by means of oxygen analysis. In large shunts the subvalvular defect can be seen best at the beginning of systole, when the amount of contrast medium in the right ventricle is still small. In defects of the membranous septum, flow of the medium is usually directed caudally in the dorsal portion of the right ventricle while the right ventricular blood is ejected orally in the ventral portions of the ventricle. Peculiarities of this flow are demonstrated by means of numerous examples. A common atrioventricular
canal can be demonstrated by direct opacification of an enlarged right atrium from the left ventricle; in this case angioarchiography also allows the determination of whether or not the ventricular septum is closed.

**Lepeschkin**


Three patients with the acyanotic form of Ebstein's disease are described. In only one patient was a slight right-to-left shunt found. The hemodynamic consequences of the anomaly are extensively discussed. Phonocardiographically in all patients a holosystolic murmur, a diastolic murmur, and a presystolic murmur were observed and in one also a protodiastolic murmur. In all cases a third heart sound was recorded and in one patient a fourth heart sound. The dye-dilution curve proved of great value for the diagnosis of tricuspid incompetence and of a possible shunt. In all cases the pressures in the right ventricle and pulmonary artery were normal. The pressure curves of the right atrium did not show an X-dip or only an indication of it. Moreover, the ventricular pressure curve showed a postsystolic dip with higher enddiastolic values, as found in constrictive pericarditis. This configuration can be explained from the small capacity of the distal half of the right ventricle.

**Brachfeld**

**CONGESTIVE HEART FAILURE**


During cardiac catheterization, two patients with pure mitral stenosis, one patient with mitral stenosis and systemic hypertension, one patient with mitral stenosis and mild aortic insufficiency, and one patient with aortic insufficiency developed acute pulmonary edema. The data obtained before, during, and after the episodes were presented as well as the effect of hexamethionium infused through the catheter. The principal findings included a marked rise in pulmonary artery and pulmonary wedge pressures, the latter rising abruptly to above 35 mm. Hg. Low cardiac output, increased heart rate, and decreased diastolic filling period were also observed. It is suggested that the precipitating causes for pulmonary edema in these patients were the maintenance of supine position for a prolonged period, with redistribution of blood from the peripheral circulation to the lungs and sympathetic stimulation due to anxiety, resulting in tachycardia, with decreased diastolic filling period and peripheral vasoconstriction. Marked elevation of systemic blood pressure and increase in right ventricular diastolic pressure were observed in two patients. Hexamethionium in doses from 6.5 to 25 mg. administered via the cardiac catheter was given to four patients. All showed a dramatic response to the drug with regression of the abnormal findings and prompt clinical improvement. The exact mechanism of action of hexamethionium is not known but it is suggested that, if the pulmonary veins can constrict, relief of this constriction by the drug would reduce pulmonary capillary pressure.

**Kayden**


The author has chosen a number of processes in various human organs to demonstrate the increased capacity to performance when subjected to increased load. This functional hypertrophy has limits beyond which the increased capacity becomes inadequate for the increasing load. At this point, failure of function becomes evident, despite output levels greater than normal. The limits of normal and pathologic states to an increase in capacity for a given function were illustrated by examples. For each process a normal rate, the high-output failure rate in certain disease states, and the pathologic limits to maximal hypertrophy of capacity were given. Organs and functions reviewed included the bone marrow and hemoglobin synthesis, the heart and cardiac output, the liver and albumin synthesis, the bone osteoid and osteoid calcification, the pulmonary vasculature and blood flow conduction, the kidney and ammonium synthesis, the thyroid and iodide organification, the liver and aldosterone inactivation and the pancreatic islets and insulin synthesis. In addition, the author suggested some other examples in man that might represent high-output failure and proposes normal and pathologic limits to increased capacity. For this group, however, physiologic technics for quantitating function are not yet available.

**Kayden**


It was observed that in the presence of congestive heart failure crepitant rales at the lung
bases could be made to shift from one side to the other by appropriate posturing of the patient. Cases were presented in which this feature was of diagnostic help. Observations during cardiac catheterization were referred to briefly. It is suggested that this phenomenon is an exaggeration of a normal phenomenon known as Buschnell's reversible crepitation.

**Other Subjects**


The carotid sinus syndrome is defined as the occurrence of spontaneous symptoms precipitated by a hypersensitive carotid sinus reflex. Sixteen male patients were studied with the reflex elicited by simple massage of the carotid sinus, first on one side and then on the other, without obstructing carotid blood flow. Testing was performed in the recumbent position and stopped as soon as cardiac standstill or blood slowing or sudden pallor occurred. The main presenting complaints of the patients were vertigo, syncopal attacks, focal attacks, and mental changes. A striking feature of many case histories was the dramatic and abrupt first onset of symptoms. Some patients improved spontaneously. The arterial pressure changes during induced attacks were recorded in 13 patients. In every instance, carotid sinus stimulation induced both disturbance of the heart rhythm (abrupt slowing or standstill) and a sharp fall in the mean arterial pressure and pulse pressure. The broad pattern of behavior of the blood pressure was similar in the cases of standstill, sinus bradycardia, or atrial standstill. In all 13 patients the hypotensive effect of carotid sinus stimulation outlasted the effect on the heart rate, whatever the mechanism of slowing. It was therefore assumed that the sharp reduction in mean arterial pressure and pulse pressure during slowing or after a systole was due either to an associated fall in peripheral vascular resistance or to a reduced cardiac output. Medical treatment consisted in anticholinergic or sympathomimetic drugs. In the severe case, the most satisfactory form of treatment was surgical denervation of the affected sinus, but if the onset was sudden and the history was short, operation should be deferred because spontaneous remission may occur.

Kurland

In 4 dogs with experimental chronic complete heart block atrioventricular conduction was re-established by electronic means. With the method described, the atrial complexes were fed into an electrocardiograph machine and then amplified so that they could be used to energize a relay system that discharged a stimulus into the ventricles. The pacemaker thus was placed under the control of the sinoatrial node, a more physiological approach than delivering a fixed rate of stimulation. Utilizing this technie the rheobase and chronaxies of the ventricular myocardium were determined; asystolic periods with Stokes-Adams seizures were experimentally reproduced; the observation was made that asystolic periods could be prevented by gradual slowing of the pacemaker to the rate of the idioventricular focus; the principal action of isoproterenol (Isuprel) was found to be that of making the idioventricular focus more sensitive so that it could take over at once, when higher pacemakers failed to stimulate the heart; and reversible congestive heart failure was produced experimentally. Based upon the data found so far an instrument applicable to human beings with chronic complete heart block is under construction.

SAGALL


When cardiac arrest occurs, the circulation must be restored promptly to prevent irreversible damage. There are 2 techniques that may be used in the emergency. One is to open the chest and massage the heart directly and the other is to accomplish the same end by a new method of closed-chest cardiac massage. This method is simple to apply: only the human hand is required. The heart is compressed by pressure on the sternum forcing it toward the spine and ejecting blood. The relaxation of the pressure allows the heart to fill again. In unconscious and anesthetized adults, the thoraclic eage is surprisingly mobile. With the patient in a supine position, preferably rigidly supported, the heel of one hand, with the other hand on top of it, is placed on the sternum just cephalad to the xiphoid. Firm pressure is applied vertically downward about 60 times per minute. At the end of each pressure stroke the hands are lifted. The operator should position himself so that he can use his body weight to apply pressure and the sternum should be moved 3 or 4 cm. toward the vertebral column with each stroke. Although some ventilation of the lungs is provided by this method, if there are two or more persons present one should massage the heart while the other gives mouth-to-nose respiration. In the 10 months prior to this writing, the method was applied to 20 patients aged 2 months to 80 years. In three of the 20 patients the hearts were in ventricular fibrillation and were defibrillated by a closed-chest A.C. defibrillator shock. Of the 20 patients resuscitated, 14 are alive at the time of this writing without central nervous system damage and without undergoing thoracotomy. The real value of this method lies in the fact that it can be used wherever the emergency arises.

KITCHELL


In a study of 157 patients with cardiac arrest the conclusion was reached in 50 patients that the sole cause was massive transfusion of hyperpotassemic bank blood. In 18 others transfusion contributed to the cardiac arrest. Bank blood contains high concentrations of extracellular potassium and these concentrations rise with the age of the blood. Endogenous release of potassium from the liver during hemorrhagic hypotension may further complicate the problem. Fresh blood, or blood treated with ion-exchange resin, has a low potassium content and is safe. Digitalis and calcium antagonize potassium effect. Refrigeration and the transfusion of ice-cold blood enhances the sensitivity of the heart to arrhythmias and raises the potassium content of the blood. Peripheral venipuncture may not show elevated potassium levels but such levels may be considerably elevated in blood obtained by cardiac puncture. Isopropylterenol (Isuprel) rather than epinephrine should be used as a cardiac stimulant.

KITCHELL


Variations of death rates in the United States from state to state concerning cardiovascular diseases are not explained on racial, dietary, or social bases. Potable water is one variable environmental influence to which all persons are exposed. Statistical analyses of water hardness and death rates from cardiovascular diseases show a highly significant correlation. This was
not noted in death rates from nonecardiovascular causes. Comparisons of known water hardness and constituents of water with death rates from degenerative cardiovascular disease showed definite negative correlations with contents of magnesium, calcium, bicarbonate, sulfate, fluoride, dissolved solids, specific conductivity of water, and pH. In all cases, higher values were associated with lower death rates. Stated in another way, softer waters are associated with higher death rates. It appears that some substance either present in hard water, or missing or entering soft water affects death rates from degenerative cardiovascular disease.

**Kitchell**

**REVIEWS IN CARDIOVASCULAR DISEASE**


