**ABSTRACTS**

*Editor: Stanford Wessler, M.D.*

Abstracts

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**ATHEROSCLEROSIS**


Male rats given a high-fat diet plus cholic acid within 7 months developed multiple thrombi in small coronary arteries without marked arteriosclerotic change. The concomitant induction of hypertension by daily desoxycorticosterone injection markedly potentiated the thrombotic tendency, increased aortic atherogenesis, and raised their mortality rate. A certain degree of hypertension and hypercholesteremia seemed to be caused by the high-fat diet alone. Half of the hypertensive rats showed mesenteric lesions of periarteritis nodosa, but hypertensive-hyperlipemic animals had considerably fewer such lesions. It was suspected that hypertension may have a direct arteriolar effect leading to thrombosis.

Rogers


The authors have previously reported a positive correlation between type A behavior pattern (characterized by enhanced drive, competitiveness, ambitiousness, and frequent exposure to “deadline” time pressures) and clinical coronary heart disease and substantially higher average serum cholesterol levels than do paired subjects who do not exhibit such behavioral characteristics (behavior pattern B). In the current study 10 men who exhibited behavior pattern A, as determined by a group of lay selectors and the authors, were compared to 10 men of pattern B in respect to serum lipids and lipoproteins. The serum analyses were performed by an independent, out of state laboratory, unaware of the subject’s personality ratings. Significantly higher serum levels of triglycerides, phospholipids, and cholesterol and of serum and other low-density lipoprotein lipids, and significantly lower a/β-lipoprotein cholesterol ratios were found in the type A group. These differences were not due to any differences in diet, weight, or physical activity.

Rogers

**BLOOD COAGULATION AND THROMBOEMBOLISM**


Blood withdrawn from varicose veins had a clot-lysis time essentially the same as that from the brachial artery. However, venous blood withdrawn 15 seconds after the vein had been pinched just upstream showed the clot-lysis time to be shortened nearly by one half. Citrated plasma injected into an isolated venous segment for 2 minutes was found to have acquired considerable
fibrinolytic activity. These findings were explained by postulating a release of fibrinolytic activator from vein walls and it was conjectured that venous thrombosis similarly excites release of activator which determines the fate of the thrombus.

Rogers


A group of 300 decompensated patients with heart disease and chronic atrial fibrillation was studied in a Veterans' Administration Hospital. All patients were treated with conventional measures of bed rest, salt restriction, and administration of digitalis and diuretics. The control group of 100 patients received no additional therapy (group 2). One hundred patients were given Dicumarol and received quinidine to the point of reversion to normal sinus rhythm or to toxicity (group 1). One hundred patients received anticoagulants but no quinidine (group 3). The mortality and morbidity (thromboembolism) rates and persistent decompensation were highest in group 2 and lowest in group 1. The incidence of persistent cardiac failure was almost as high in the anticoagulated group who did not receive quinidine (group 3) as in group 2 (neither anticoagulants nor quinidine). All patients who successfully reverted to normal sinus rhythm achieved cardiac compensation. There was a significant reduction in mortality and morbidity rates in groups 1 and 3, indicating the usefulness of anticoagulants in reducing thromboembolic complications. Anticoagulants and conversion to normal sinus rhythm would seem to offer the best prognosis for patients with organic heart disease and chronic atrial fibrillation.

Helwig

CONGENITAL ANOMALIES


Studies were made on 10 patients with corrected transposition of the great vessels. Among the associated lesions found, were six instances of ventricular septal defects and two instances of left-sided Ebstein's disease. The association of any degree of heart block was very suggestive of corrected transposition. Clinical examination was helped when pulmonary murmurs were maximal low down or when the pulmonary component of the second sound was inaudible in the pulmonary area. The chest roentgenogram may show a straight or convex contour to the left upper portion of the heart shadow normally occupied by the pulmonary artery or the vascular pedicle may be narrow. The electrocardiogram may show Q waves in the right precordial leads, absent Q waves in the left precordial leads, and prominent Q waves in leads III and aVp. Cardiac catheterization and selective angiography demonstrated the essential characteristics of this condition.

Kalmansohn


Six infants, aged 6 days to 13 months at death, were studied clinically and pathologically. All were cyanotic since birth, and three had a systolic murmur in the midprecordium. Roentgenograms revealed pulmonary hypovascularity and, often, right heart enlargement. Electrocardiograms showed normal P waves at birth progressing to P pulmonale by age 4 months. Normal or right axis deviation and right ventricular preponderance or hypertrophy were the main clinical features distinguishing these patients from those with tricuspid atresia. Autopsy in all patients disclosed a patent foramen ovale, a stenotic hypoplastic tricuspid valve, a hypertrophic right ventricle with a small cavity not permitting angiocardiographic visualization by venous injection, pulmonary atresia, and hypoplastic pulmonary arteries with a small patent ductus arteriosus. A tiny ventricular septal defect was present in one patient, and transposition of the great vessels was not encountered. Attempted shunt surgery failed in all three instances.

Rogers

CONGESTIVE HEART FAILURE


Determinations of the circulating pulmonary blood volumes in 30 patients with heart disease and two patients with no cardiovascular abnormalities are reported. Decreasing pulmonary blood volume was related to increasing functional disability. The lowest values for pulmonary blood volume were found in groups III and IV patients. There was no definite relationship between the pulmonary blood volume and the degree of elevation in the pulmonary arterial pressure. There was a tendency for the pulmonary blood volume to decrease with increasing pulmonary vascular re-
sistance. There was no relationship between the heart volume or the presence of mitral valvular disease and the pulmonary blood volume. It was concluded that the characteristic pattern of the pulmonary circulation in patients with heart failure is not an increased pulmonary blood volume but an elevation of blood pressure in the pulmonary vessels. Consequent to the elevated pulmonary pressures along with interstitial edema there is impairment of the elastic properties of the lungs. The potential volume of the pulmonary vessels becomes restricted.

Rakita

CORONARY ARTERY DISEASE


This study attempts to evaluate the significance of qR or QR patterns in premature ventricular contractions with regard to the diagnosis of myocardial infarction. Leads aV_R and aV_L were excluded from consideration because their particular orientation in the electrical field allows them to exhibit a qR pattern normally. One hundred and forty-four electrocardiograms in 138 patients were available for analysis. The qR or QR pattern was present in 12 records from normal subjects, a “false positive” incidence of 8.3 per cent. On the other hand, the morphology of the premature beat failed to detect myocardial infarction 43 times in leads which were otherwise characteristic of myocardial infarction; a “false negative” incidence of 29.8 per cent. Finally, there were 16 (27.3 per cent) patients with normal electrocardiograms who had had unequivocal infarcts in which the premature beats exhibited qR or QR patterns. Unfortunately the report does not consider either the total incidence of “noninfarct pattern” premature beats in electrocardiograms otherwise pathognomonic of infarction, or the incidence of “noninfarct pattern” premature beats in nonspecific or normal electrocardiograms from patients with unequivocal infarction.

March


The authors point out the fact that at the present time (and in spite of the vast amount of written material) there is not enough warranty to consider coronary heart disease as an epidemiologic entity—at least like a “macroscopic” entity. The “macroscopic” type takes a gross look at the prevalence or incidence of a disease; for example, a comparison in various countries of dietary factors and of mortality from arteriosclerotic heart disease. Such a study must be based on statistical analysis. The statistics available pertaining to coronary heart disease are not dependable. The “microscopic” type makes an intimate personal study of a population. At the present time this is a safer approach to the consideration of this disease as an epidemiologic entity and it is possible to define it in terms of clinical syndromes. The most reliable clinical syndrome for this purpose should be myocardial infarction and sudden death. Angina pectoris is a purely subjective sensation and it is often difficult to detect or to define. The selection of population samples is another important feature for this study. In this way, epidemiologic studies which have been carried out to date have been amply rewarding and a large-scale established program should be justified.

Liotta


This report from the Cardiovascular Health Center, Albany, New York, is based on the observation of a population sample of 1,913 men in the age range 39 to 55. Increasing age and masculinity were accepted as the two major risk factors in ischemic heart disease. At the initial examination of a group of 1,913 men, 70 patients with ischemic heart disease were detected. During the 6-year period of observation reported in this study, 96 additional patients were observed in the group of 1,843 men originally free from this disease. Ischemic heart disease is defined as myocardial infarction, angina pectoris, abnormal electrocardiographic response to standard exercise, death attributed to arteriosclerotic heart disease, or sudden death with no other apparent cause. An effort was made to construct a coronary profile and a noncoronary profile based on the following data: serum cholesterol, blood pressure, electrocardiographic evidence of left ventricular hypertrophy, amount and duration of smoking, and obesity. Despite the fact that it is not possible to identify a specific coronary-prone individual with any precision, the average man with no clinical evidence of ischemic heart disease is slightly younger, less hypercholesteremic, less hypertensive, less obese, and smokes less than the average man with ischemic heart disease.

Liotta

Circulation, Volume XXIX, January 1964

Tetracycline analogues become fixed in diseased animal tissues, such as myocardial infarcts. A chlortetracycline (CTC) fluorescence technic was used to study the myocardium of dogs that had undergone either infarction from ligation of the anterior descending coronary artery or electrical defibrillation by a 1000-1500 V condenser discharge 3 to 5 minutes after induction of fibrillation by an alternating current. In the infarcted hearts, CTC was fixed in the muscle fibers and in the form of an intensely fluorescing demarcation line between healthy and diseased tissue. In the defibrillated hearts, single damaged fibers fluoresced. Unlike the free antibiotic, the CTC complex in the myocardium is stable on boiling, in an alkaline medium, and in formal. The development of fixed fluorescence is dependent on the presence of damaged muscle and of bivalent ions especially calcium. Labeling of the CTC by I$^{131}$ and its detection by precordial scanning may possibly have an application in the diagnosis of ischemic heart disease.

Marshall


Sixty-five cardiac output measurements were performed in 16 patients with acute myocardial infarction, principally during the early phase of the disease. Persistently low cardiac and stroke indexes were found only in fatal cases (seven). Two of the nine survivors had a low cardiac index initially, but all were subsequently normal. Ventricular gallop rhythm in 15 of 18 instances and tachycardia in all of 19 instances were associated with a low stroke volume (35 ml. per beat per M.² or less).

Rogers


Human hearts and aortas obtained from 150 cases of accidental deaths in the New Delhi area were analyzed for atherosclerotic changes utilizing the methods and criteria recommended by the World Health Organization Study Group. Significant atherosclerotic lesions in the coronary vessels of the Indian subjects appeared at least two decades later than the North American population and appeared to involve less of the intimal surface and demonstrated fewer fibrous plaques. No direct correlation was found between the aortic and coronary atherosclerotic changes. However, these changes began approximately two decades later and involved less intimal surfaces in the coronary vessels than in the aortic wall. The authors conclude that estimation of aortic atherosclerosis in itself will be inadequate to assess the degree of ischemic heart disease in the New Delhi area.

Karpman


Review of the necropsy files of the Western Infirmary, Glasgow, for the period 1940-60 disclosed 606 deaths due to myocardial infarction, of which 43 had ruptured. Rupture was slightly more common in women, occurring in 16 of 195 women with fatal infarcts. The patient age at rupture paralleled that of infarction in general. The most important factor found to be associated with rupture was hypertension persisting after infarction. This occurred in 70 per cent of the group with rupture and in 25 per cent of the patients with infarct, but without rupture. A second factor correlating with rupture was the presence of pronounced polymorphonuclear leukocytic infiltration into the infarct. No significant association was found between rupture and anticoagulant therapy, electrocardiographic pattern or myocardial fibrosis.

Rogers

ELECTROCARDIOGRAPHY, VECTORCARDIOGRAPHY, BALLISTOCARDIOGRAPHY, AND OTHER GRAPHIC TECHNICS


In anesthetized dogs, occlusion of the superior and inferior venae cavae caused an abrupt decrease in the magnitude of the QRS potentials recorded from surface leads. The magnitude of the QRS potentials returned to control values after restoration of venous flow. Identical results were obtained in dogs in which the heart rate was not controlled and in others in which vagotomy or atropinization had been carried out. Often
the QRS potentials were increased during venous occlusion in direct right epiventricular leads. Similar results were obtained when venous return was reduced by means of rapid withdrawal of large volumes of blood. The findings suggest that the major deflections of surface leads depend on potential vectors oriented approximately at right-angles to the intracardiac blood mass. In contrast, in the opened-chest preparation the major components of direct right epiventricular leads appear to depend on potential vectors oriented tangentially to the intraventricular blood mass.

Marshall


Phonocatheters were inserted into the aorta of anesthetized dogs to study the mode of sound transmission through blood vessels. The average velocity of transmission of the canine second heart sound was 5.7 meters per second. Murmurs, produced by compression of the aorta in dogs, were found to be transmitted in the form of a transverse vibration in the aortic wall. The authors conclude that most of the energy of cardiovascular sound in arteries is transmitted in a mode which is exactly analogous to the transmission of the pulse wave.

Helwig


Vagotonic influences may cause an increase in amplitude of the slurped delta component of the QRS complex in the Wolff-Parkinson-White syndrome. Vagolytic influences, such as atropine, can cause its descent to the isoelectric line and even result in its separation from the main part of the QRS complex. The author affirms that the P wave preceding the W-P-W ventricular complex is not structurally similar to the sinoatrial P wave. He has found that a brief “P-R” interval is not an essential feature; indeed, W-P-W complexes may be associated with first-degree heart block. Further, W-P-W complexes may occur in the absence of a preceding P wave.

Marshall


The fetal QRS is electronically recordable as early as 10 to 11 weeks following conception. An FM data-transmitting system has been developed for telemetric transmission and recording of this utilizing normal telephone circuits. The main receiving station is operationally located in Milwaukee, Wisconsin, and has received successful transmissions from Fort Wayne, Indiana (approximately 200 air miles). This development permits better and more economical maternal care in situations where the status of the fetus is in question, and also offers considerable research potential.

Sanctetta

ENDOCARDITIS, MYOCARDITIS, AND PERICARDITIS


A case of constrictive pericarditis operated on twice with an interval of 17 years between operations and with a successful result following the second procedure is reported. Six years following the second operation the patient presented with pronounced hypoproteinemia, hydrothorax ascites, and moderate edema. Recurrent diarrhea and hypalbuminemia persisted until death, 24 years after the original pericardectomy. A retrospective view revealed that hypalbuminemia had been present before the second operation and that an increase in serum albumin level had occurred after relief of the constriction. Radioactive studies revealed abnormal gastrointestinal permeability, a reduction in total exchangeable albumin due to an increased degradation, and a limited capacity for albumin synthesis. Lymphatic studies after cannulation of the thoracic duct were compared with the findings in a control subject. The pressure in the thoracic duct was 25 cm. in the patient and 6 cm. in the control subject. Lymph flow was almost 5 times as great in the patient as in the control subject. There was also an absolute reduction in the quantity of fat transported via the thoracic duct. In addition, there was a marked reduction in the albumin concentration of the lymph from the patient. Postmortem examination revealed numerous dilated lymphatics scattered along the entire length of the small intestine, and lymphangiectasia was seen on histologic examination. In addition, there was mild portal cirrhosis. It was suggested that the increased flow of thoracic duct lymph in constrictive pericarditis could result from increased pressure in the inferior vena cava or in the portal vein. The factors responsible for intestinal protein loss might include cardiac-inflow stasis with increased pressure in the inferior vena cava and the portal vein, increased intestinal capillary pressure causing increased production of
lymph with secondary dilatation of lymphatics, and loss of protein and fat by rupture or by escape of lymph by transudation under high pressure. In this particular patient impaired hepatic synthesis of albumin was probably also a factor.

Rakita

HYPERTENSION


Permanently reversible chronic hypertension was induced in male rats by excision of the right kidney and constriction of the left renal artery. Hypertension was ameliorated rapidly by removal of the constricting clip. The retinal vessels were photographed during control, hypertensive, and subsequent normotensive periods under light ether anesthesia. In mild or moderate hypertension, an indefinite diffuse narrowing was noted. In addition, those with marked hypertension, 220 mm. Hg and over, demonstrated a characteristic focal arterial narrowing. This was manifest in one or more vessels with dilatation of intervening parts and a tortuous appearance. The changes appeared early within weeks or more gradually in several months. Once present, the zones of narrowing remained constant in position and varied little in degree. Despite their fixed appearance, the areas of focal narrowing and dilatation were abolished when ether anesthesia was deepened. Lightening of the anesthesia caused return of the elevated blood pressure and reappearance of the localized arterial changes in the previously affected area. Although this cycle can be repeated, reversibility becomes more difficult with time. In rats with hypertension up to 18 months, vasoconstriction and tortuosity were abolished within days by removal of the renal artery clip. Some areas of overdilation did not return to normal. Some microscopic sections revealed several instances of focal arterial necrosis. In the vast majority there were no morphological findings to account for the altered vessel caliber. This lends support to the opinion that angiospasm is the cause for similar retinal changes in man. Structural change may become irreversible with time. It is inferred that labile progressive focal vasoconstriction may be the cause of retinopathy and the widespread circulatory changes of malignant hypertension.

Kaltman


This apparatus was developed to facilitate the self-registration of the blood pressure by patients on antihypertensive medication. It consists of a modified, standard, conventionally inflatable blood pressure cuff with an inserted pickup, two mercury-calibrated aneroid pressure gages for separate registration of systolic and diastolic (either fourth to fifth sound or sound disappearance) pressures, and appropriate amplifying, timing, and readout circuits. Pressure energy of the first detectable pulse is converted to electrical energy and the systolic pressure gage is arrested electromagnetically. On disappearance of the sounds, capacitance discharge occurs, arresting the reading on the diastolic gage. Tabulation is provided of the blood pressures simultaneously obtained by conventional auscultation and the apparatus, the range of pressures being 74 to 162 mm. Hg (systolic) and 61 to 99 mm. Hg (diastolic). Variation in comparative systolic measurements is negligible; in five of the 10 patients tested the diastolic pressures matched closely, in the other five the apparatus underestimated pressure by 9 to 18 mm. Hg. This is due to a 2-second lag in registration following capacitor discharge, and could presumably be avoided by very slow cuff deflation or a speedup in conduction of the RC discharge. Appropriate diagrams of the circuitry are provided.

Sancetta


The role of renin in experimental canine hypertension has been well established. In the current paper an extensive series of experiments with monkeys is reported. Hypertension was induced in monkeys by injection of monkey or human renin and by a 3:1 (volume-pulse ratio) constriction of a renal artery. Frequent direct arterial pressure measurements were recorded in all controls and the experimental group which were injected with monkey or human renins and antirenins (produced in dogs) or with various infraprimate (dog, hog and rabbit) renins and antirenins.

The following observations were noted: 1. Monkey and human renin produced hypertension in the monkey but the infraprimate renins did not. 2. Antirenin to human renin (but not to hog or dog renin) neutralized monkey renin. 3. Crude human renin (processed from human kidneys obtained at autopsy from Chicago area hospitals) was partially successful in treating chronic experimental monkey renal hypertension but crude hog renin was not. Passive immunization with dog serum containing antirenin to human renin (but not to hog renin) was similarly successful. 4. Prophylaxis of experimental monkey renal hypertension with crude human renin was partially suc-
cessful as long as adequate antirenin titers persisted. 5. No foreign protein nephritis was encountered. The results support the author’s hypothesis that renin (or a closely related protein) plays a role in the pathogenesis of primate experimental hypertension. The theoretic possibility of treating human essential hypertension is raised but a 3 months course of treatment would require 56,000 monkey kidneys and hence “a more feasible approach to the possible pathogenetic role of renin in essential hypertension might be the production of an ‘antimetabolite’ to angiotensin.”

HELWIG


The authors describe an in vitro method to determine the ability of plasma to inactivate synthetic angiotensin, “plasma angiotensinase activity” (PAAs). PAA was increased in “essential” hypertension complicated by severe cardiovascular disease, in chronic glomerulonephritis, in hypertension secondary to renal artery stenosis, and in pheochromocytoma (both before and after removal of the tumor). In all of these conditions, which may have in common an ischemic or obstructive renal vascular disorder, there is prior evidence for increased activity of the mechanism concerned with the secretion of renin. Increased PAA was also found in refractory edema and in pregnancy (normal and abnormal), both of which conditions are commonly associated with secondary hyperaldosteronism. It is suggested that the increased PAA in all these conditions may result from an increased rate of elaboration of angiotensin through a process of enzymatic adaptation. It is concluded, therefore, that previous failures to demonstrate consistent increases in angiotensin levels in blood in even the more severe stages of “essential” hypertension and in malignant hypertension do not exclude the possibility that the rate of elaboration of angiotensin is increased.

MARSHALL


Thirteen patients who had a stroke while under observation for hypertension in a clinic were compared with 99 nonstroke patients from the same clinic. The two groups were found to be similar except that 12 of the 13 stroke patients had not had a sustained and significant reduction in blood pressure prior to the stroke. In contrast, 79 of the nonstroke group had had a good pressure reduction, from an average systolic level of 198 mm. Hg to 162 and from an average diastolic reading of 119 mm. Hg to 96. It was suggested that effective hypotensive therapy may help prevent strokes.

ROGERS


The course of 104 patients with untreated diastolic hypertension of 120 mm. Hg or more was compared with that of 104 patients treated with ganglion-blocking drugs and with 79 patients given guanethidine. Reduction of diastolic pressure to 90 to 110 mm. Hg was attended by a distinct fall in the 3- to 5-year mortality rate, chiefly due to a decrease in incidence of strokes and uremia. It was suspected that both these complications resulted from fibrinoid arteriolar necrosis which was prevented or arrested by lowering blood pressure. The infrequent strokes that occurred despite pressure control were attributed to cerebral arteriosclerosis. Whether more could be gained by earlier treatment of hypertension and whether lesser degrees of hypertension should be treated were regarded as unanswered questions.

ROGERS


Retroperitoneal fibrosis is an uncommon disease of unknown etiology. Although most of the main retroperitoneal vessels were affected in the cases previously reported in the literature, none included involvement of the renal artery. The authors describe a 54-year-old man with hypertension in whom the left renal artery was occluded by a tunnel of fibrous tissue. After surgical removal of this constriction, the blood pressure returned to normal limits. A 3-month follow-up examination revealed continued normotension. Since the process did not involve the right renal artery, it is reported in detail as a case of unilateral hypertension caused by retroperitoneal fibrosis. Treatment is surgical and the approach must be bilateral. The aim of surgery is to free the ureters from their fibrous sheaths. In patients with hypertension, dissection of the renal pelvis is suggested to relieve possible renal artery compression.

KALTSMAN

Circulation, Volume XXIX, January 1964

Data on family history and blood pressure were collected in 1,989 men aged 40 to 55 free from known coronary heart disease. History of familial hypertension, heart disease secondary to high blood pressure, or cerebral vascular accidents was considered evidence of hypertension. Subdural hematoma, embolus, or uremia in relatives was excluded from this group. Correlation of a single casual measurement of the blood pressure obtained with the family history was subjected to several types of statistical analyses. To study the effect of age on blood pressure, the men were divided into three groups: 40 to 44, 45 to 49, and 50 to 55. Those with one or both parents hypertensive were separated from the population with negative family histories. The blood pressure of men with positive family histories was higher at each age group. Systolic and diastolic pressures rose with age at the same rate as those with and those without hypertensive families. This rate of increase was the same in men with parents surviving to old age as in those with a parent dead in middle age. The data do not support inheritance of essential hypertension as a simple Mendelian dominant trait. The mode of inheritance cannot be ascertained by this study, but it is more in keeping with Pickering’s doctrine of continuous distribution of blood pressure. Environment is not excluded as an important etiological factor. This is suggested by the rise in blood pressure with age independent of family history.

KALTMAN


Technics for recording the arterial blood pressure, other than sphygmomanometry or intra-arterial manometry, either permit only intermittent measurement of systolic pressure (ophthalmic artery occlusion, finger-type sphygmomanometers) or are still in the experimental stage (methods based on measurement of arterial distention or of pulse wave velocity). The authors describe the development and preliminary assessment of a transducer that permits continuous external recording of the arterial pressure pulse. Initially they used a transducer that recorded displacement of the skin overlying a superficial artery (e.g., brachial, radial, superficial temporal). However, problems of calibration and artifacts due to physiologic changes in skin and subcutaneous tissue around the artery led to adoption of the present technic, which consists in restraining arterial deflection by the transducer and measuring the restraining force. A mathematical model of the transducer-artery system was developed and used as a guide for the experimental prototype transducers. The blood pressure was measured simultaneously in 11 subjects by sphygmomanometry, with use of an electronic stethoscope (right brachial artery), and by the external transducer (left radial artery). The systolic pressure was 0 to 15 per cent less and the diastolic pressure 7 per cent greater to 19 per cent less by the latter method. Preliminary comparisons of external with simultaneous intra-arterial pressures in the cat provided even closer agreement. Also, records obtained during rapidly changing conditions of the blood pressure, such as the Valsalva maneuver, clearly demonstrated the anticipated changes in absolute pressures, heart rate, and wave form.

MARSHALL


A newly developed automatic blood-pressure recorder made possible half-hourly observations of pressure during sleep. Nearly all of the 16 benign hypertensive patients showed a fall in systolic and in diastolic mean levels, averaging 15.4 and 9.1 mm. Hg, respectively. In contrast, 11 of 12 malignant hypertensive patients showed no such pressure fall, the mean changes during sleep being 0.08 mm. Hg systolic and −2.6 diastolic. These differences in the groups were thought not likely to be due to a difference in severity of pressure elevation, since many of the patients in the benign group had pressures as high as those of the malignant group.

ROGERS


Twenty-two men with labile hypertension, free from evident vascular disease, were observed before and 4 hours after the intramuscular injection of 2.5 mg. of reserpine. The arterial pressure fell in all subjects, and both the cardiac output and peripheral arterial resistance fell when elevated. The pressor effect of infused norepinephrine was slightly greater than in control subjects, which was
regarded as evidence that reserpine had produced some catecholamine depletion. However, "re-
leased" epinephrine or norepinephrine could not be
detected in the plasma nor could an increased
urinary excretion of 3-methoxy 4-hydroxy man-
delic acid be demonstrated. Tilting to 60° for 10
minutes produced syncope in five of 11 patients,
two of these despite infusions of norepineph-
rine. Arterial epinephrine levels rose in six of
eight patients during tilt but did not rise in the
two of these who fainted.

ROGERS

Villamil, M. F., Yeyati, N., Enero, M. A.,
Rubianes, C., and Taquini, A. C.: Effect
of Long-term Treatment with Hydrochlo-
rothiazide on Water and Electrolytes of Mus-
cle in Hypertensive Subjects. Am. Heart J.

The intracellular and extracellular distribution
of water and electrolytes in muscle was studied in
five normal subjects and in eight hypertensive pa-
ients before and after the administration of hy-
drochlorothiazide (HCT). Muscle biopsies were
taken once in control subjects, and before and
after HCT treatment in the hypertensive patients.
All participants were on "standard" diets with the
addition of 2 Gm. of sodium chloride daily.
The treated patients received HCT 50 mg., twice
daily, for periods of 22 to 48 days. Sodium and
potassium in dried muscles and plasma were deter-
mined by flame photometry. Extracellular water
was derived from the chloride space. Normal val-
ues were in agreement with previous work. Hyp-
ertensive subjects showed higher contents of
total muscle and intracellular water and sodium,
and higher concentrations of intracellular sodium.
Total muscle potassium was normal, but there was
a relative reduction of intracellular potassium.
HCT caused reductions in total muscle and in-
tracellular water, sodium, and potassium.

MARCH

METABOLIC EFFECTS ON
CIRCULATION

Nelson, G. H., Bear, D. M., Dotson, T. O.,
and Krause, R. F.: Lipid Changes in Cardiac
Hypertrophy as Measured by Silicic
Acid Chromatography. Am. J. Physiol. 204:
297 (Feb.), 1963.

In view of the importance of lipids to the struc-
ture, function, and integrity of the myocardium it
was considered worth while to investigate the lip-
id changes that might accompany cardiac hyper-
trophy. Cardiac hypertrophy was produced in

adult, male, albino rats by prolonged exposure
to reduced atmospheric pressure in an altitude
chamber. The total lipid extracted from the heart
was separated into seven fractions by silicic acid
chromatography. Fractions II, IV, and VII were
found to contain reasonably pure triglyceride,
cholesterol, and phospholipids respectively. Fra-
cions I, III, V, and VI contained cholesterol esters,
free fatty acid, diglyceride, and monoglyceride,
respectively, but were contaminated with other
lipid material. With cardiac hypertrophy there
was no change in the weight of total lipid per
heart. As the heart increased in weight, the phos-
pholipid content increased and the weight of cho-
lesterol decreased. These results and those ob-
tained by others would indicate that with cardiac
hypertrophy the increase in heart water, glycogen,
or lipids was not significant and that the greater
portion of increase in weight is represented by
the increase of protein in the heart.

ABBoud

PATHOLOGY

Enos, W. F., Beyer, J. C., and Holmes, R. M.:
Arteriosclerosis of the Aortic Sinuses. Am.

Narrowing and closure of the coronary ostia
are generally attributed to syphilitic aortitis. Ar-
teriosclerosis of the sinuses of Valsalva is very
seldom cited as a cause of obliteration of the coro-
nary ostia. The authors believe that in some in-
stances the myocardial changes and the resulting
clinical symptoms are attributable to lesions of
arteriosclerotic origin surrounding the coronary
ostia. These lesions are more frequent in the
anterior than in the left posterior sinus of Val-
salva, probably due to the anatomic location of
the former and to hemodynamic factors function-
ing in this area. As pointed out by physiologists
and anatomists, the anterior sinus may receive
less external support than the other two sinuses
and therefore the intima may receive more
trauma. The obliteration of the ostium of the
conus artery eliminates an important source of col-
larateral circulation. The authors describe the clin-
cal and autopsy findings of two patients in whom
the cause of death was obliteration of the right
coronary ostia from an arteriosclerotic lesion in
the anterior aortic sinus.

Lopez

Mariu-Padilla, M., McGill, R. J., Jr., and
Shaka, J. A.: Diffuse Fatty Degeneration
of the Myocardium. Report of Two Cases.
The clinical course and autopsy findings of two

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patients with diffuse fatty degeneration of the myocardium and myocardial hypertrophy are described. In a review of 72 cases of idiopathic myocardial hypertrophy it was found that more than 50 per cent of the patients were young adults, without evidence of any of the usual causes of cardiac decomposition. At autopsy they had cardiomegaly with hypertrophy of individual fibers, mainly in the subendocardial region, and intracellular vacuoles were commonly seen on histopathologic studies. Intracellular lipid has been reported in association with a large variety of infectious diseases and with intoxication from phosphorus. Fatty degeneration has been found in experimentally induced shock in dogs that were sacrificed serially. This fatty degeneration was focal in distribution, occurred between the third and fifth days, and subsequently regressed. The degenerative changes were considered to be direct results of myocardial hypoxia. The degree of involvement and the diffuse distribution of the lipids in the two patients described are unique and could represent a spurious metabolic pathway that did not become manifest until it was stimulated by prolonged hypoxia.

**Lopez**

**PHARMACOLOGY**


The effects of alkalosis and changes in serum potassium concentration on cardiac arrhythmias are well known. In this study, performed on anesthetized dogs, the effects of acute metabolic acidosis (HCl infusion), acute metabolic alkalosis (NaOH infusion) and acute metabolic alkalosis with potassium infusion, on the cardiac arrhythmias produced by strophanthidin were evaluated. The amount of the rapid-acting digitalis required to produce cardiotoxicity was closely related to the serum potassium concentration and essentially unrelated to pH per se. The data are consistent with previous observations that infusion of a mineral acid results in a rise in serum potassium and fall in serum sodium due to intracellular ion shifts. Digitalis, in toxic doses, appears to block potassium uptake into cells and therefore serum potassium rises.

**Helwig**


Retinal photographs offer the opportunity of measuring the changes in the caliber of the retinal vessels in man. Seven normal subjects (age 27 to 35 years) were studied on separate occasions after infusions of angiotensin and norepinephrine. Retinal photographs were obtained during a control saline infusion, after a steady elevation of the blood pressure was reached by the infusion of the drug and, finally, half an hour after the blood pressure had returned to normal. The vessels were measured in the transparencies (Kodachrome I film) by means of a low-power microscope and by two observers independently. Measurements were made in arbitrary units. The average blood pressure before norepinephrine was 114/73 (mean 85) mm. Hg and during the infusion, 173/104 (mean 127) mm. Hg. Before angiotensin the average resting value was 115/72 (mean 86) mm. Hg and during the infusion it went up to 116/114 (mean 131) mm. Hg. There was a uniform reduction in the caliber of the arterioles. The reduction in the diameter of the arterioles average 7.3 units with angiotensin and 5.4 units with norepinephrine. A similar, but smaller, reduction was observed in the caliber of the veins. The reduction in size appeared to be greater in the smaller vessels than the larger ones, suggesting that the degree of fiber shortening in the small vessels was greater than in the larger ones. Other factors, like the ratio of the mitral wall thickness to the internal diameter, may influence the change by a given amount of fiber shortening.

**Lopez**


This study was based on simultaneous measurements of cardiac oxygen consumption, the coronary flow and the performance of the heart as affected by ouabain. Two preparations, the isolumic and coronary flow preparations, were employed to test the action of ouabain on the heart and its coronary vascular bed. A total of 13 mongrel dogs were studied. In the isolumic preparation ouabain produced an increased coronary flow, an augmentation in the magnitude of left ventricular contraction and cardiac oxygen consumption, and a diminution of oxygen extractions. In this preparation ouabain appeared to act primarily as a coronary dilator and the other changes were secondary. This suggested that ouabain may exert its beneficial effects by lessening the deficiency in coronary flow. In the coronary flow preparations small amounts of ouabain had no effect when cardiac oxygen consumption was related to the HR.BP index of effort. In larger
amounts ouabain increased the magnitude of this $O_2C$-to-HR.BP ratio. Still larger amounts of ouabain decreased the $O_2C$-to-HR.BP ratio at the same time as it depressed the myocardial oxygen extraction. These two effects resembled the diphasic action of ouabain on oxidative metabolism in heart muscle slices.

**ABBoud**


It has previously been demonstrated by one of the authors and others that the glycoside, ouabain, caused an increased uptake of Ca$^{45}$ in frog, rabbit, and guinea pig myocardium. The rat heart is very resistant to the effects of glycosides and the present study reports the effects of ouabain on calcium dynamics of rat atria. Ouabain induced a small but significant increase in Ca$^{45}$ uptake and a decrease in tissue Ca content in the rat atria during the positive inotropic effect but the concentrations of the glycoside required were 100 to 200 times greater than those required in guinea pig atria.

**HELwIG**

**PHYSICAL SIGNS**


Phonocardiograms were recorded routinely in 103 residents of a Masonic Home, all aged 80 or more. Systolic ejection murmurs were found in 59 subjects, mostly attributable to hemodynamically insignificant aortic valve disease. A single holosystolic apical murmur was observed. Also one decrescendo diastolic murmur was noted at the left sternal border. Systolic ejection clicks were recorded in 14 patients, in 10 of whom the origin was considered to be aortic, in two pulmonic. The second sound was widely split in seven cases, six of which were associated with right bundle-branch block. Third sounds were recorded in 27 cases and heard in 12 of these; only the latter showed evidence of congestive failure. Fourth sounds were recorded in 59 patients and heard in 43 of them; five of the latter had diastolic hypertension of 100 mm. Hg or greater.

**ROgers**

**PHYSIOLOGY**


The purpose of these experiments was to deprive the cardiovascular system of all neuromotor control. Seven dogs who ran well on a motor-driven treadmill were completely sympathectomized (including adrenal denervation) and subjected to unilateral vagotomy. After recovery, retraining, and completing the vagotomy a terminal experiment was performed during which direct Fick determinations of cardiac output and continuous recordings of mean arterial pressure, heart rate, and oxygen consumption were made at rest and during increasing exercise. The results were compared with those described by Barger et al. for normal dogs. With exercise the increases in cardiac output and oxygen consumption were very small compared to Barger's normal animals. Peripheral resistance was reduced equally in both groups but the blood pressure in the normal dogs increased while that in the treated dogs fell about 20 per cent. The heart rate in the treated dogs increased only slightly. The low oxygen consumption and the vigorous work performed lead to the conclusion that a large oxygen debt must have occurred during the ten-minute working periods. Changes in venous or left atrial pressure were not measured during the work. The animals without autonomic cardiovascular regulation were able to do a surprising amount of running over a 10-minute period with very little change in heart rate, cardiac output, or oxygen consumption.

**ABBoud**


Eight well-trained athletes (cyclists) with large dimensions of the circulatory system were studied with heart catheterization at rest and during exercise at 800 and 1600 kpm./minute both while sitting and supine. The results are compared with data from nonathletes. The total amount of hemoglobin averaged 993 Gm. (14.0 Gm. Kg. body weight), which is higher than found in nonathletes. The total blood volume averaged 7.51 liters (106 ml./Kg. body weight) which is higher than found in nonathletes. The heart rate at rest in the supine position was 63 beats/minute and increased to 115 on the first load and to 160 beats/minute on the second load; in the sitting position at rest it was 64 and the corresponding figures during exercise were 112

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and 159 beats/minute. The oxygen uptake at rest in the supine position averaged 31.7 per cent and sitting 49.9 per cent higher than the predicted basal value; during exercise it increased at 800 kpm./minute to an average of 1864 ml. in the sitting and 1769 ml. in the supine position. At 1600 kpm./minute the values were 3387 ml. and 3364 ml. in the sitting and supine position, respectively. The lactate concentration increased during exercise at the highest load to an average of 4.96 mEq./L. and 3.36 mEq./L. in the supine and in the sitting position, respectively. The arterial oxygen saturation at rest averaged 97.8 per cent, during the first load it decreased to 96.1 per cent both in the sitting and supine position, and at the heavier load to 95.1 per cent in the sitting and to 94.5 per cent in the supine position. The oxygen saturation of mixed venous blood at rest averaged 77.2 per cent in the supine and 66.8 per cent in the sitting position. During the first load it decreased to 47.1 per cent in the supine and to 40.8 per cent in the sitting position. At the second load the values were 31.4 per cent and 27.0 per cent, respectively. The arteriovenous oxygen difference at rest averaged 38.5 ml./L. in the supine and 60.7 ml./L in the sitting position. The cardiac output at rest averaged 9.18 L./minute in the supine and 6.61 L./minute in the sitting position; at 1600 kpm./minute it averaged 26.26 L./minute in the supine and 24.50 in the sitting position. The stroke volume at rest in the supine position was 149 ml. and decreased 43 ml. (29 per cent) on changing to sitting position. In the supine position during exercise the stroke volume increased by an average of 13 ml., or 9 per cent, to an average of 163 ml.; in the sitting position it increased by 49 ml., or 48 per cent, of the value at rest. The stroke volume at rest in the supine position averaged 2.01 per cent of the blood volume, the same as found in ordinary subjects. Right ventricular filling pressure was lower in the sitting than in the supine position at rest. This pressure increased during work, more in the sitting than in the supine position.

**Biot**


In a previous communication the authors had reported on a new differential flowmeter adapted to a catheter end (cross sectional area 0.178 cm.²) for measurements within suitable arteries and veins of intact large mammals, and had also presented the static calibration. In the current report dynamic calibration was performed in a semi-rigid system employing water as the perfusate. In 360 observations the degree of difference in known as against computed stroke volume varied from 0 to 11 per cent, with a mean difference of 3.8 and a standard deviation of ± 2.9. This held for stroke rates up to 100. At higher rates and with higher stroke volumes, the computed flow is progressively less than actual; at stroke rates of about 150, no flow is measurable. This may be due to the fact that the system employed in calibration was semi-rigid.

**Sancetta**


In anesthetized dogs a thermodilution technic, in which cold saline was injected into the left ventricle and sampled from the root of the aorta, was used to measure cardiac output and left ventricular stroke volume (SV), end-systolic volume (ESV), end-diastolic volume (EDV). Fast heart rates were produced by electrical pacemaking (using a catheter electrode) and slow rates by stimulation of the distal end of the severed right vagus nerve. The left ventricular volumes decreased during moderate tachycardia. During more extreme tachycardia, SV decreased proportionately more than ESV and EDV. At slow rates SV increased proportionately more than ESV and EDV. The decreased SV during tachycardia from electrical pacing confirms the findings of Warner and Toronto, and provides an interesting contrast with the effects of exercise in conscious dogs, in which comparable tachycardia is accompanied by little change or a slight increase in SV.

**Marshall**


The effects on intracardiac pressures and flows of angiotensin action were measured during cardiac catheterization utilizing indicator dilution technics in over 200 young patients, of which nine representative cases were presented. The injection of 1 to 10 µg. of angiotensin II into the central venous circulation was followed for approximately 3 minutes by little change in the pulmonary arteriolar resistance (with the possible exception of some instances of pulmonary arteriolar obstruction) although a slight rise in pulmonary blood pressure occurred passively; systemic arteriolar resistance increased, causing moderate rise in pressure including that of the left atrium, while cardiac output decreased little or not at all. Left-
to-right shunts increased while right-to-left shunts decreased. These changes were often pronounced and at times allowed detection of a shunt not shown by conventional oximetry. The possible therapeutic use of the drug in raising the arterial oxygen saturation during cyanotic spells in tetralogy of Fallot was mentioned. No untoward effect of angiotensin was observed in any of the 12 studies.

ROGERS


When the intrathoracic pressure is increased to 3 to 4 cm. water in the dog through forced insufflation of the lungs, the venous pressure rises but the arterial pressure falls to almost zero. When the intrathoracic and intra-abdominal pressures are increased to similar values by inflating a balloon placed in the abdominal cavity after bandaging the abdomen and thorax, the venous pressure rises but the arterial pressure rises also. This type of experiment, where abdominal blood is forced into the heart, is more like the Valsalva experiment in human subjects. In the latter, registration of intra-abdominal pressures from a high rectal balloon showed that during the Valsalva test the rise of intra-abdominal pressure is up to 20 mm. Hg greater than the rise of intrathoracic pressure, as registered by the manometer mouthpiece. In human subjects, the mean peripheral arterial pressure does not fall, while the diastolic pressure rises. However, the amplitude of pulse oscillations decreases, and there is a reduction of the radiologically determined cardiac area. Toward the end of the experiment the P wave of the electrocardiogram increases in amplitude and shows right axis deviation and the duration of the QRS complex increases. The S-T segment shows slight depression especially in lead V₆ while the T wave usually shows a marked decrease in amplitude and sometimes inversion. The maximal duration of the experiment in different individuals does not seem to depend on the arterial oxygen concentration alone.

LEPESCHKIN


The authors made hemodynamic observations of the effects of atrial and ventricular pacing in mongrel dogs. When the atrium and ventricle con-

tracted at about the same time, the ventricle was deprived of the atrial contribution to ventricular filling, mean atrial pressure rose in relation to the left ventricular end-diastolic pressure, the end-diastolic pressure was lower, and the ventricle performed less work.

KALMANSOHN


Little information is available about the actual rate of oxygen uptake of cardiac Purkinje fibers. This investigation was undertaken to measure the oxygen uptake of intact Purkinje fibers of the dog's heart by a flow respirometer and oxygen polarography. The tissue employed was a free-running Purkinje fiber from the left ventricle. The fiber was approximately 10 mm. in length and ranged from 0.4 to 0.75 mm. in diameter. At ambient oxygen concentrations of 60 per cent or higher, the rate of uptake was 0.739 cu. mm./mg. wet weight per hour at 35°C. Ambient oxygen concentrations greater than 60 per cent did not alter the oxygen uptake, but at lower oxygen concentrations the rate of uptake was depressed. This depression was probably the result of diffusion limitation. It should be recognized that in the present study no attempt was made to determine the amount of connective tissue present in the Purkinje bundle. These and other observations would indicate that Purkinje fibers have a much lower oxygen requirement than the adjacent myocardium.

ABBoud


A new technic of perfusing a papillary muscle has been developed. This has made it possible to study calcium exchange in the functioning myocardium of dogs. Basically the flux of calcium in the papillary muscle of dogs is similar to that of frog skeletal muscle and guinea-pig atria. An initial rapid phase followed by a slower phase has been described. The major component of calcium flux is independent of the rate of contraction and is determined not only by diffusion but also by other factors as yet unknown. A lesser component of calcium flux is related to the frequency of contraction. The present observations of calcium exchange provide additional support to the concept of the central role of calcium in excitation-contraction coupling.

CUCCI

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This study was concerned with a comparison of estimates of ventricular volumes in the same animal by indicator-dilution method and by cineangiography. In each of 12 mongrel dogs data for end-diastolic volume (EDV), end-systolic volume (ESV), and stroke volume (SV) of the left ventricle were obtained by the two methods. While the values for stroke volume showed good agreement, significant and directionally constant differences were found between the values for EDV and ESV calculated with the two methods. The values for EDV obtained by the indicator method were between 1.5 and 3.3 times higher than those obtained from the angiogram. Values for ESV were as much as 9.3 times greater than those from the angiogram. Differences were observed in dogs with fast heart rates as well as in dogs with heart rates to about 100 beats per minute. The authors interpret their findings as strongly suggesting that a fundamental error is present in estimation of volume based on the washout of an indicator dye.

ABBOND


With a recently developed experimental model these authors perfused directly the sinus node through its own artery in the intact heart of an opened-chest dog. They noted that injections into the artery were almost invariably accompanied by slowing of the sinus rhythm for the duration of the injection. The rate returned to control levels after completion of the injection. This phenomenon was observed in over 90 per cent of 800 experiments in 35 dogs. Studies to explain the phenomenon included control of temperature, pH, osmolarity, oxygen, and ionic content of injecting solutions. Neurogenic mechanisms were excluded by bilateral cervical vagotomy and by direct perfusion of the sinus node with atropine, hexamethonium, and trimethaphan. Because the sinus node completely surrounds its nutrient artery, the sinus bradycardia may simply be the consequence of distention of the sinus node artery. The authors indicate that if the rhythmic expansion of extracardiac arteries (carotid sinus) can be considered as contributing to the control of heart rate, the very artery supplying the cardiac pacemaker must be allotted similar consideration. Implications concerning autoregulation of the normal heart rate are discussed.

ABBOND


A transistorized, battery-powered impedance detector has been devised that is capable of localizing the conduction tissue of the living mammalian heart, beating or in arrest, within 1-mm. distance and at a depth of at least 3 mm. A three-terminal guard circuit is employed to obviate differences in impedance caused by blood and by changes in pressure as the probe is passed over the ventricular cavity. The use of a probe with gold electrodes, "aged" by prior immersion in saline activated by a small AC current, minimizes changes due to ionization. The principle is based on the fact that cardiac conduction tissue conducts an impulse faster than muscle tissue and hence has a lower impedance. By appropriate amplification, rectification and balancing of the current within the circuity of the apparatus, an area of low impedance (conduction tissue) is detected by an increase in the pitch of a constant audio signal. The latter can be adjusted to a frequency range that is not disquieting and that can be readily filtered by the human ear from the ambient operating-room noise. Using the device in more than 70 open-heart surgical cases where an interruption of the conduction system with heart block might be anticipated as a result of the surgical repair, the authors have reduced their occurrence of this complication from a previous incidence of 10 per cent to only 2 per cent in the current group of subjects.

SANCETTA


In this study the strength and the time course of isometric contractions of cat papillary muscles were determined at normal cardiac temperatures and at 14 regular frequencies of contraction. Increases in frequency of contraction were associated, over the entire range, with decreases in the time to peak tension and in the relaxation time and increases in the maximum rate of development of tension and in the peak tension developed. The changes in heart rate altered both the degree of activation of the contractile elements and the duration of their active stage. It was concluded
that the opposing effect of both of these changes must be considered in any analysis of the influence of alterations in rate or rhythm of the heart on the strength of contraction of the mammalian ventricular muscle. Decreased duration of the active state with increased heart rate correlated with the decrease in duration of action potential observed by others. The increased degree of activation resulted in an increase of strength contraction without excessive shortening of the time available for ventricular filling. Hence, these inherent responses of heart muscle to changes in heart rate are of great importance for the normal function of the heart.

**ABBOUD**

Lewartowski, B.: Functional Changes in Ventricular Muscle After Crushing the Sino-

Previous work by the author indicated that crushing the sinoatrial node of rabbit hearts was followed by shortening of the refractory period of the right ventricle. This effect was present in animals treated with reserpine. Stimulation of the vagus nerve did not affect the ventricular excitability cycle. Phystostigmine gave rise to the changes in excitability cycles similar to those that followed crushing of the sinoatrial node. In the present study the influence of imposed heart rate or crushing of sinoatrial node on the total cholinesterase activity and excitability cycle of the ventricular muscle was investigated. Experiments were performed on anesthetized rabbits. Cholinesterase activity was estimated with a potentiometric titration method. Acetylcholine bromide was used as a substrate. Crushing of the sinoatrial node was followed by a decrease in cholinesterase activity if the heart was driven at a rhythm exceeding a spontaneous rate by 100 beats per minute. Destruction of the node was followed also by shortening of the absolute refractory period if the accelerated driving lasted for at least 20 minutes. It was supposed that acetylcholine metabolism of rabbit ventricular muscle was influenced by the activity of the cardiac conductive system. Acetylcholine system being one of the important factors regulating the permeability of the cardiac cell membrane, this compound might influence the excitability cycle.

**ABBOUD**


Twenty-six dogs were studied to determine changes in regional myocardial blood flow produced by pulmonary embolism and pulmonary artery constriction. Rb\(^{86}\) was infused intravenously and continuous sampling of blood for Rb\(^{86}\) analysis was performed from a systemic artery, the coronary sinus (mainly representing the left ventricular flow) and an anterior cardiac vein (mainly representing right ventricular flow).

Pressure overload of the right ventricle (RV) resulted in an increase of flow to the RV from 0.52 ± 0.26 ml./minute to 1.05 ± 0.32 ml./minute. Isotope clearance, which is closely related to flow, increased by 25 percent in the right atrium. The increases in flow to various portions of the ventricles did not appear to be proportionate to increases in the hemodynamic load.

**HELWIG**

Mahon, W. A., and Mashford, M. L.: Pressor Effect of Tyramine in Man and Its Modifi-

Reserpine depletes catecholamines in isolated tissues and in pharmacological preparations of intact animals. The purpose of this study was to decide whether reserpine affects circulatory reflexes in intact man. The pressor response to tyramine, 0.2 mg. per Kg., was found to be significantly less in eight subjects after treatment with reserpine, 0.06 mg. per Kg. intravenously, than it was in the control state. These observations can be explained by the hypotheses that tyramine acts by releasing endogenous norepinephrine, and that therapeutic doses of reserpine decrease the response to tyramine probably by reducing tissue stores of catecholamines.

**MARSHALL**

Samet, P., Jacobs, W., Bernstein, W. H., and Shane, R.: Hemodynamic Sequelae of Idio-

Simultaneous systemic arterial and venous pressure recordings in two of three patients with complete heart block and electrical ventricular pacing were presented. When contraction of the atria preceded that of the ventricles, maximum arterial and pulse pressures with minimum venous pressure were achieved, but when P waves occurred during or immediately following QRS complexes, arterial and pulse pressures promptly fell while venous pressure rose, implying a decrease in cardiac output. Thus the importance of atrial contraction in ventricular filling was indicated, and the desirability of pacemakers preserving this sequence was theorized.

**ROGERS**

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ABSTRACTS
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