ABSTRACTS

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ELECTROCARDIOGRAPHY,
VECTORCARDIOGRAPHY,
BALLISTOCARDIOGRAPHY,
and OTHER GRAPHIC TECHNICS

Gottsegen, G., and Bodrogi, G.: On the Mechanism
of the W-P-W Syndrome. Acta cardiol. 16:529,
1961.

The authors report a case of Wolff-Parkinson-
White syndrome associated with idioventricular
rhythm. Because of the time relationships of
the P-P intervals, the R-R intervals, the timing
of the delta wave, and the P-J intervals, it was
concluded that the mechanism of the syndrome
in this instance was related to two independent
pacemakers, one from the atria and the other
from the ventricle, discharging so as to form
fusion beats. It was proposed that the delta
wave was initiated by the impulse arising in
the ventricle and the terminal portion of the QRS
complex resulting from normal conduction of
the impulse via the usual pathway. The authors
point out that other mechanisms for this syn-
drome may be operative in other circumstances.

Rakita


The ordinary electrocardiogram yields but a
small sample of the electrical activity of the
heart in any 24-hour period. To secure electro-
cardiograms during exercise and other activity,
the radioelectrocardiogram was developed to
secure freedom from recording wires. However,
even this method had the drawback that a re-
corder had to be maintained in the vicinity of
the patient. The author has developed a com-
 pact electrocardiocoder, which may be carried
on the person and records a continuous electro-
cardiogram on tape for many hours. The recorder
is combined with an analyzer system which can
detect the occurrence of even a single ectopic
beat or ST-segment deviation and display them
for analysis.

Helwig

Howland, W. S., Schweizer, O., and La Due, J.
S.: Evaluation of Routine Postoperative Elec-
62:1941 (June 15), 1962.

Electrocardiograms were recorded on 782 pa-
tients on admission to and discharge from the
recovery room. Four hundred and sixty-one of
the patients showed evidence of abnormalities
of rate, rhythm, or conduction, or showed changes
suggestive of "myocardial disease." Ninety-nine
showed evidence of arrhythmias. Patients sub-
jected to operation in the regions of the breast
and perineum were more prone to develop brady-
cardia. Two hundred and seventy-one patients
were considered to have evidence of cardiac
pathologic conditions represented by flattening
or inversion of the T wave, depression of the ST
segment, delayed intraventricular conduction,
bundle-branch block, nodal rhythm, A-V disso-
ciation, atrial fibrillation, old myocardial infar-
etion, cor pulmonale, or left ventricular hyper-
tropy. Electrocardiographic abnormalities in 167 of these patients were not discovered prior to operation. Only two of these 167 patients showed electrocardiographic changes indicative of myocardial damage attributable to events occurring during the operative and postoperative period. In all the others the abnormalities of the electrocardiogram resulted from chronic heart disease. It is concluded that routine postoperative electrocardiography is not justified, since the information revealed was not greater than that found by restricting the procedure to selected groups of patients. However, a routine preoperative electrocardiogram is indicated in every patient scheduled for general or spinal anesthesia or major surgery.

RAKITA


Of the 24 patients in whom the diagnosis of endocardial fibroelastosis was proved by postmortem examination, electrocardiographic findings of left ventricular hypertrophy occurred in 90 per cent. Three patients showed evidence of isolated right ventricular hypertrophy. In addition, two of the patients with left ventricular hypertrophy showed evidence of right ventricular hypertrophy as well. Fewer than 30 per cent of the patients showed left axis deviation. Electrocardiographic evidence of left atrial enlargement occurred frequently. Very minimal descriptions of the vectorecardiograms from the five living patients are reported.

RAKITA


The authors report electrocardiographic changes in four patients with acute pulmonary embolism. In the first three cases ST depressions and T-wave inversions occurred in the left chest leads. In the fourth case electrocardiographic changes suggested a posterior myocardial infarct and at postmortem examination such an infarction was found although the coronary arteries were normal. The origin of the electrocardiographic changes in acute pulmonary embolism remains obscure. One possibility is altered right ventricular function due to the embolism. Other suggestions include an associated myocardial infarction or the simultaneous presence of peri-carditis. The demonstrated myocardial infarction in the presence of normal coronary arteries in the fourth case suggests a severe reduction in coronary flow because of pulmonary emboli. This lends weight to the hypothesis that the electrocardiographic changes in other cases were the result of myocardial ischemia.

KURLAND


Intra-cardiac phonocardiographic characteristics of the atrial sounds and the relation of these vibrations to mechanical and electrical events of the cardiac cycle were studied in 21 dogs. Simultaneous recordings of intracardiac (from right atrium, left atrium, right ventricle, and left ventricle) and external phonocardiograms and equi-sensitive intra-cardiac pressures confirmed that the atrial sound is commonly formed by two main sets of vibrations: the first coincident with the onset of the atrial "a" wave, the second with its highest pressure peak. It is suggested that the first group of vibrations is usually due to atrial contraction and the second to ventricular filling. A short reversal of presystolic atrioventricular pressures after atrial relaxation occurred in some of the cases, suggesting that here a valvular mechanism was involved in the production of the late component of the atrial sound. The rapid intravenous infusion of saline generally increased the amplitude of the atrial sound and made evident the presence of a second component. The administration of drugs inducing systemic or pulmonary hypertension failed to give consistent changes in the amplitude and timing of the atrial sound. An asynchronism between right and left atrial sounds was demonstrated, the right-sided presystolic vibrations preceding those of the left heart by 0.026 second (AS1) and 0.023 second (AS2), respectively, in accordance with the earlier electrical activation and contraction of the right atrium.

KAYDEN


Right bundle-branch block of various degrees was produced in 20 normal individuals by passing an electrode catheter against the septal surface of the outflow tract of the right ventricle. The delay thereby induced in time of onset of the
intrisicoid deflection from a right ventricular cavitory lead was 0.03 to 0.05 second and the electromechanical interval was delayed similarly, thus proving the existence of a conduction block in the right ventricle. Existing concepts of vectorecardiographic and electrocardiographic patterns of complete right bundle-branch block were generally confirmed. However, morphologic signs of incomplete right bundle-branch block were found with QRS intervals as brief as 0.07 second. In such instances, slowing of the QRS sE loop was not always present, and the relationship between electrical and mechanical systole frequently remained within normal limits.

ROGERS


Among 200 electrocardiographic tracings from patients with arterial hypertension due to multiple etiologies 19 per cent were normal, 7 per cent showed minor abnormalities suggestive of left ventricular involvement, 49 per cent manifested evidence of systolic overload of the left ventricle, 3.5 per cent diastolic overload of the left ventricle, 12 per cent combined systolic and diastolic overload of the left ventricle, and 9.5 per cent showed evidence of coronary artery involvement. aQRS occurred in the normal sextant in 46 per cent of cases and showed left axis deviation in 40 per cent, right axis deviation in 11.5 per cent but marked left axis deviation occurred in 2 per cent. AT was normal in 38 per cent, right axis deviation in 39.5 per cent, and left axis deviation in 22.5 per cent. The aQRS and AT angle exceeded 90° in 32 per cent. The ventricular gradient was normal in 80 per cent. In 19 per cent the gradient was deviated to the left. The authors conclude that with systemic arterial hypertension the variations of the aQRS, AT, and G vectors are not comparable to other electrocardiographic criteria which are ordinarily used to evaluate cardiac involvement. It is noted that normal ventricular gradients were observed in otherwise abnormal electrocardiograms and abnormal gradients were observed in the presence of normal electrocardiograms.

Sagall


Thirteen children were studied before and up to 20 months after open repair of a ventricular septal defect. Preoperative electrocardiograms showed right ventricular hypertrophy in 10 patients, left ventricular hypertrophy in one, and no abnormality in two; the QRS duration was 0.10 second or less in all. Vectorcardiograms gave similar results. Postoperatively the QRS duration had increased by 0.02 to 0.06 second; rR' or RSR' was seen in V_{1} in eight patients, rR' was found in two, R in two, and QR in one. Serial observations in a few patients revealed a gradual decrease in R or R' with the QRS duration remaining prolonged. Postoperative vectorcardiograms consistently displayed slowing of the terminal portion of the QRS loop and occasionally a terminal appendage was inscribed to the right and anteriorly. The vectorcardiogram was thought to be more helpful than the electro-

Pathologic, electrocardiographic, and clinical data were studied in 39 cases of myocardial infarction complicated by the presence of left bundle-branch block. The findings indicated certain electrocardiographic features which in the presence of left bundle-branch block might afford evidence of myocardial infarction. Substantial evidence of transmural anteroseptal myocardial infarction appeared to be afforded by Q-wave deflections in lead I or by Q-wave deflections or Q-wave equivalents in lead V_{6}. Supportive evidence of anteroseptal myocardial infarction appeared to be afforded by R waves in lead V_{3} or V_{4} lower in amplitude than those in V_{1} or V_{2}, or by a notch or slur with a duration of 0.05 second or more in the terminal portion of the QRS complexes in the precordial leads showing rS or QS deflections. Supportive evidence of posterior wall myocardial infarction appeared to be afforded by Q-wave deflections in leads II and III. Finally equivocal evidence of anteroseptal myocardial infarction appeared to be afforded by Q-wave deflections in lead aV_{1}. In three instances electrocardiograms were recorded with left bundle-branch block present both before and after infarction and in two the R waves in the standard leads were found to have decreased markedly in height after infarction. Changes in the S-T segments and T waves of a degree to be considered suggestive of myocardial infarction were found only in a minority of the cases of recent myocardial infarction.

Rakita

cardiogram in detecting right ventricular hypertrophy in the presence of right bundle-branch block.

ROGERS


Electrocardiograms were correlated with clinical findings in 56 patients, with clinical and hemodynamic findings in 50 patients and with clinical, hemodynamic, and autopsy findings in 36 patients with myocardial infarction. Anterior myocardial infarction was more common than posterior, and also showed a greater mortality and incidence of tachycardia, cardiac enlargement, and venous congestion. The mortality was higher in anterior infarction when atrial fibrillation was present and in posterior infarction when extrasystoles were present. Atrioventricular conduction disturbances were more common in posterior infarction, whereas paroxysmal ventricular tachycardia was more frequent in anterior infarction. Signs of heart failure or elevation of venous pressure corresponded to increased mortality, especially in anterior infarction. Signs of shock were accompanied by increased mortality in posterior but not in anterior infarction. Ventricular conduction disturbances appeared in 53 per cent of the cases with involvement of the septum, in 35 of those without it. In anterior infarction without septal involvement S-T elevation in V₃ was always smaller than in V₄, while in those with septal involvement it was greater in 45 per cent of the cases.

LEPESCHKIN


Leads II and V₃ of the electrocardiogram were used to provide patterns of wave form that were used to test the ability of properly programmed computers to duplicate accurately and automatically the steps involved in human pattern recognition and measurement. The electrocardiographic leads were converted from its analogue form to digital values and recorded in digital form on magnetic tape. Measurements were obtained at each 0.0016 second. The information was then fed into an LGP 30 computer. The clinical variables selected for measurement were the P, Q, R, S, T, and U waves, and P-Q, S-T, Q-T, and R-R intervals. Rules and definitions based on conventional electrocardiographic criteria were formulated and used to define wave onset, wave peak, wave termination, significant voltage fluctuation, and time intervals in the electrocardiogram. The point of greatest negative rate of change in the electrocardiographic signals was used as the stable reference point in the logic of recognition, because this parameter was found to be repeatable in any subject. The P-wave peak is searched for in a fixed interval before the maximum negative derivative. A base line is constructed as the straight line connecting the start of the P wave in the second heart beat with the start of the P wave in the third heart beat, and all amplitudes of the electrocardiographic signal are referred to this base line. Other steps in the logic of recognition are the location of the T-wave peak, searched in a fixed interval after the S- or R-wave peak, and the finding of the beginning and end of the P, Q, R, S, T, and U waves. The computer correctly measured 767 of 770 parameters in the electrocardiograms tested. These measurements agreed in the first two significant figures with manual measurements of standard clinical electrocardiograms. In one subject with a signal to noise ratio of 10:1 the computer program could not distinguish between the high degree of noise present and the P wave. Thus, the onset, peak, and end of the P wave were not determined by the computer. The computer was able to analyze successfully electrocardiograms with signal to noise ratio of 15:1 or greater.

LUCHI


The authors studied the electrocardiographic records of 849 normal individuals between the ages of 1 and 16 years, and calculated the arithmetic sum of the voltages SV₂ and RV₅. The mean value for this sum was found to be 38.0 mm., plus or minus 9.7 mm., and the range was 15 to 65 mm. It was noted that in both sexes prior to the age of 11 years voltages up to 60 mm. (i.e., the sum of SV₂ and RV₅) were frequently found in normal individuals. There were no statistically significant differences in the voltage in males before and after the ages of 11 years; however, the greater extent of deviation in the older boys made it necessary to regard values up to 65 mm. as being within the normal range. Between the ages of 11 and 16 normal values were
found to be as high as 55 mm, in females and 65 mm, in males. The authors concluded that it was unwise to rely on precordial voltage alone as an index of left ventricular hypertrophy unless the SV2 plus the RV3 values were greater than 60 mm, in children less than 11 years of age, or greater than 55 mm, in females and 65 mm, in males past 11 years of age. When values fell within those limits, axis deviation, intrinsicoic deflection time, the position of the transition zone, the comparison of QRS segment and T waves, and, when available, vector analysis had to be taken into account in order to diagnose left ventricular hypertrophy with any degree of accuracy.

Karpman

ENDOCARDITIS, MYOCARDITIS, AND PERICARDITIS


Endomyocardial fibrosis is commonly observed in both sexes of the natives of northern and western Africa, but it is quite uncommon in South Africans. The disease begins as a thickening of the endocardium at the apex of either or both ventricles and extends upward destroying the papillary muscle-chordae tendineae function, so that severe atrioventricular valvular regurgitation is the rule. When the right heart is involved in this way any concomitant left heart involvement is masked. The right atrium may become very large; 1 case was cited in which its volume was 600 ml. Pronounced venous hypertension with a prominent CV wave in the neck and cirrhosis with ascites typically are seen, making for a readily recognized syndrome in endemic areas. The fall in venous pressure following paracentesis, the usual absence of dependent edema, the preservation of good effort tolerance and of cardiac output until late in the course — all point away from the existence of congestive heart failure in the early stages. The pulmonary valve is not involved, but calcification of the endocardium of the right ventricular outflow tract is nearly pathognomonic of the disorder. The myocardium shows little disease, so the dip and plateau observed in right ventricular pressure recordings were ascribed to endocardial restriction of venous filling. Treatment was generally unsatisfactory although the use of digitalis was advocated in the patients with atrial fibrillation.

Rogers


In the 125 cases of "degenerative heart disease of pregnancy," previously reported in the literature congestive heart failure usually appeared just before or after delivery and was characterized by a protracted course and a poor response to the usual therapeutic agents. In this group about 20 per cent died, embolic phenomena were common, and pathologic studies showed a subendocardial myocardial degeneration without significant myocarditis. Two additional cases with the clinical and pathologic features similar to the previously reported cases are presented. One patient also showed the presence of pericardial effusion, a finding not previously reported. The etiology of the condition was considered and it was concluded that the clinical features and pathologic picture were not pathognomonic.

Sagall


This is the case report of a Belgian who developed a systemic illness with cutaneous and ocular lesions in Equatorial Africa. The diagnosis of filariasis was made on the finding of 85 per cent eosinophils and of microfilariae in the blood. The patient responded to treatment with nitidine. Six years later he presented with paroxysmal dyspnea, cardiomegaly, and cardiac failure. Catheterization showed the presence of severe pulmonary venous hypertension. Although eosinophilia was still present, attempts to demonstrate parasites in the blood and pleural fluid were unsuccessful. At autopsy there were a firm lamellae thickening of the endocardium of the left ventricle and foci of ischemia in the subjacent myocardium; the remaining chambers were normal. The authors believe that this finding represented the end result of an allergic reaction to infestation with filariasis.

Marshall


Fourteen consecutive patients with staphylococcal endocarditis were evaluated from the standpoint of combined antibiotic therapy. The
onset of the endocarditis followed surgery in three patients and a self-induced abortion in a fourth patient. One patient was infected with *Staphylococcus albus*, and the other 13 patients with *Staphylococcus aureus*, hemolytic, coagulase-positive. Nine strains were penicillinase producers. Of the four patients who died, two received no planned antistaphylococcal therapy and of the two patients who died with planned therapy, one died suddenly 5 weeks after completion of antibiotic therapy, and one represented a true therapeutic failure. Eighty-two of the patients who received planned antibiotic therapy were apparently cured.

**Kalmansohn**

**Kisch, B.: A New Type of Cardiac Parasite.**


Study of the ultramicroscopic structure of the frog’s heart performed on animals with red-leg disease has led to the discovery of very small parasites in the blood stream and cardiac tissue in two such animals. The parasites are a previously unknown type of protozoon and accumulated mainly in the subendocardial space, the space between the endocardial cells and the sarcolem of muscle fibers. The parasites, the largest of which measured 10 μ, are mainly in three forms: pyriform measuring up to 3 μ with a cilium at the tip, spindle or sausage shaped 2 to 5 μ in length with a cilium at each end, and a bizarre amoeboidal form up to 10 μ in size and having cilia irregularly distributed over the body. The cytoplasm of all three forms is surrounded by a membrane 200 to 300 Å thick, having a parallel striation with a periodicity of 500 Å, but there is no sturdy pellicle. In the cytoplasm is an ovoid or spherical nucleus containing a dark karyosome. Also seen was a striking parallel arrangement of fine lamellae having a double lining and droplets between them that may represent a Golgi apparatus, an ergastoplasm, endoplasmic reticulum, or possibly a primitive type of mitochondrium, since no mitochondria of the usual type were seen in any of these protozoa. The cilia show the typical inner structure of nine fibrils arranged in a circle around a central fibril. The parasite, among the smallest known, does not fit any known species of protozoa and the name *ultrazoon romarum* is suggested by the author who feels that it may not be the only representative of this class. It was also shown that the intruding parasites are destroyed through phagocytosis by the cells in the subendocardial space. These findings prove that the subendocardial cells of a vertebrate may have phagocytic properties.

It is suggested that these findings may have relevance to human cardiopathy, and electron microscopic studies of endocarditis and research into other protozoon diseases are indicated.

**Fox**


Rats pretreated with 9-alpha fluorocortisol plus sodium phosphate developed cardiac lesions following intravenous administration of saccharate iron oxide. This cardiopathy was characterized by “tigroid” streaks of myocardial necrosis with thrombosis in the capillaries and medium-sized veins of the heart.

**Kalmansohn**

**HYPERTENSION**


Pathologic changes observed in the arterial wall with advancing age include an increase in collagenous fibers, which results in a loss of distensibility, and a decrease in the unstretched length of these fibers due to cross linkages and adhesions. This latter change results in a more rapid decrease in distensibility with stretch and an increase in the rate of change in slope of pressure-volume curves toward the pressure axis. An indirect clinical index of this effect, called index of arterial rigidity, is obtained by measuring the intra-arterial blood pressure changes resulting from the inhalations of amyl nitrite before the onset of reflex tachycardia. The ratio of the change in pulse pressure times 100 to the related decrease in diastolic pressure during this period is the index. The relationship of hypertension to aging of the arteries was studied in 100 hypertensive patients by means of the amyl nitrite blood pressure test. Previous studies by these authors have established the limits of the index in normal subjects for separating the rigidity of aging from that of premature arteriosclerosis. There was no evidence of premature arterial aging in 71 of the 100 hypertensive patients when they were compared with normal subjects of the same age. The clinical features of the 29 hypertensive patients with abnormal rigidity indices suggested that arteriosclerosis and hypertension developed as two separate entities possibly potentiating each other. No evidence was found to suggest that hypertension might have been caused by arteriosclerosis extending.
to the peripheral vascular system. Of the 29 hypertensive patients in the group with high indices, seven had diabetes mellitus, seven had hypertension for more than 10 years, three were over 80 years of age, three had a family history of diabetes, and one had myxedema. In only four of the 100 hypertensive patients was there unexplained premature arterial aging.

**KAYDEN**


Blood pressure studies were performed on relatives of primigravidae delivered in hospital in Aberdeen. The sisters of 146 preeclamptic patients had a higher incidence of preeclampsia or hypertension in their first pregnancy than did the sisters of 273 primigravidae without preeclampsia or hypertension. In 87 mothers who had at least one daughter with eclampsia or hypertension in her first pregnancy the mean systolic and diastolic pressures were significantly higher than in the 149 mothers none of whose daughters developed hypertension in a first pregnancy. Siblings of 176 middle-aged women who had had preeclampsia or hypertension in the first pregnancy some 20 years earlier showed higher mean systolic and diastolic pressures, at all ages than siblings of 177 middle-aged women who had been normotensive throughout their first pregnancies. The findings suggest a strong familial tendency to preeclampsia and hypertension in pregnancy and support the hypothesis that preeclampsia does not cause hypertension in later life but develops more often in women with an inherited hypertensive tendency in whom the hypertension would later have become manifest anyway.

**SAGALL**


A group of women between 35 and 50 years of age were examined in regard to their blood pressure. One hundred and ninety-seven were nulliparous and 334 had their first pregnancy at least 15 years prior to this study. Of this latter group 149 had had preeclampsia during that pregnancy and 185 had been normotensive. It was found that the mean blood pressures of those who had had mild or severe preeclampsia were significantly higher than those who had not suffered preeclampsia. Higher mean blood pressures were found in the nulliparous women. The findings were interpreted as supporting the views that preeclampsia is not a direct cause of hypertension in later life; that in some women diagnosed as having mild preeclampsia the rise of blood pressure resulted from a temporary unmasking during pregnancy of a hypertensive tendency than subsided after delivery but returned in later life. It was also suggested that the proportion of women with severe preeclampsia who have this hypertensive tendency is no greater than average.

**SAGALL**


Twenty patients were treated with methyldopa in initial dosage of 250 mg. four times a day starting in the hospital and followed on an outpatient basis. All but one of the 20 patients showed a satisfactory initial blood pressure response during the first 2 weeks of treatment with methyldopa. Some slight subsequent increase was necessary to maintain the reduction in diastolic pressure. Four patients developed resistance. There was some reduction of pain in two patients who suffered angina pectoris. Significantly, a striking effect in the reduction of supine blood pressure was noted. This constitutes one of the great advantages of methyldopa together with the fact that it does not cause symptomatic postural hypotension or undue drop in blood pressure on exertion. The disadvantages of methyldopa are a lack of potency in severe hypertension, drowsiness, fluid retention, and the amount of the compound required to control blood pressure.

**KURLAND**


This report describes in detail the clinical and necropsy findings of two boys (ages 5 and 11) who died of severe hypertension. In both patients hyperplasia of the adrenal medulla was found at necropsy; this was the only lesion found in one of the patients, whereas the other patient demonstrated findings consistent with a severe degree of chronic pyelonephritis in addition to the medullary hyperplasia. In a review of 20 other cases of hypertension in children without evidence of cardiovascular disease or glomerulonephritis, the authors were able to find two proved cases and one suspected case of medullary hyperplasia. Careful study of the nephrons of the child who had died with associated pyelo-
nephritis revealed no evidence of nephron abnormalities that might predispose to the development of pyelonephritis. The author suspects that the hypertension of adrenal origin may have been the predisposing factor in the development of the pyelonephritis in this case. It is concluded that medullary hyperplasia is a disease entity because it was the only reason found at autopsy to account for the hypertension in one of the children, and because the hypertension persisted after excision of the ischemic kidney in the second case.

Karpman


Infusion of angiotensin into normal individuals had been previously found to cause an antidiuresis with marked diminution of inulin (C_{in}) and para-aminohippurate (C_{PAH}) clearances. The present studies were made in 21 patients having hypertension of various origins, employing infusions of angiotensin, 2.5 mg. per minute or less, for 10 to 20 minutes. Sixteen of the 17 subjects having a resting diastolic blood pressure of more than 120 mm. Hg exhibited a well marked and prompt diuretic response, usually with a slight reduction in C_{PAH} and little change in C_{in}. Considerable increases in urinary sodium and chloride excretion and a slight rise in urinary potassium generally occurred. The five remaining patients showed the normal antidiuresis. In two of three studies a diuretic effect was achieved with suppressor doses of angiotensin. Following successful medical or surgical antihypertensive treatment, all of seven patients showed a reversal of the diuretic response over a period of several months. While the mechanism of the induction of diuresis in severely hypertensive persons was not clear, it was suspected that a slight though inmeasureable increase in glomerular filtration rate and in perfusion pressure might be contributory.

Rogers


The biological half-life of Na^{22} was measured in seven hypertensive adults and in eight normotensive adults on equivalent sodium chloride intakes. Na^{22} has a physical half-life of 2.6 years in comparison to short-lived Na^{22} with a physical half-life of 15 hours. Counting was done by whole-body counting procedure. Dietary regimens were controlled for the duration of the study, and three levels of sodium intake, 34, 86, and 172 mEq. per day, were studied. At all three levels of sodium intake, hypertensive subjects were found to have a prolonged biological half-life for Na^{22}. It is suggested that the explanation for this observation is that hypertensive patients have a larger sodium pool than do nonhypertensive subjects.

Kayden


Whereas the effectiveness of sodium depletion in the treatment of “essential” hypertension is well established, most studies, including earlier work of these authors with use of isotopic dilution with Na^{22}, have failed to detect an increase in the sodium content of such patients. Finding of a linear increase in the prevalence of hypertension with increasing salt consumption in rats and man has caused the authors to restudy the body sodium in patients with and without hypertension. Fifteen patients, seven with “essential” hypertension and eight with unrelated diseases and normal blood pressures, were studied while on a constant diet containing 250 mg. of sodium chloride. Na^{22} (physical half-life 2.6 yr.) was given orally and the body content of Na^{22} determined every 1 to 3 days with use of a whole-body counter. Thereafter sodium chloride intake was increased by either 2, 5, or 10 Gm. for 2 to 4 weeks. Addition of this salt to the diet resulted in a prompt increase in Na^{22} loss from the body. However, at each of the three levels of salt ingestion, the hypertensive patients lost Na^{22} at a significantly slower rate (p 0.01) than those without hypertension. Of three possible explanations for the latter finding, the thesis that hypertensive patients have a larger “metabolic pool” of sodium most clearly warrants re-examination. The authors recently noted negligible losses of exchangeable sodium in obese hypertensive women despite weight losses of 20 to 50 Kg. Therefore, the authors’ earlier determinations of total exchangeable sodium in hypertensive patients, based on actual patient weight, were recalculated by two approximations of “lean body mass.” This resulted in the finding that hypertensive patients appear to have an increase in tissue sodium, supporting the thesis that sodium is intimately involved in the hypertensive process.

Fox

Fifty-nine hypertensive patients were treated with methyldopa, one of a series of compounds inhibiting the decarboxylation of dopa and other aromatic amino acids. The dose was 0.5 to 4 Gm. daily in divided doses. Action began after 5 hours and lasted for about 24 hours. Absorption from the intestine was good, and in the presence of normal renal function excretion was rapid. In uremia, however, care must be exercised because of delayed excretion. Satisfactory control of blood pressure was obtained in 32 of the 59 patients. Blood pressure control with methyldopa has less chance of being successful in patients with the severest grades of hypertension. Side effects included drowsiness, which is almost universal but wears off after the first week, nasal stuffiness, dryness of the mouth, soreness of the tongue. The most troublesome side effect was weight gain due to retention of fluid, occasionally accompanied by edema, raised jugular venous pressure, and even left ventricular failure. Methyldopa is an effective hypotensive agent whose properties place it between the powerful pressure-lowering agents such as the ganglionic blockers and the less potent agents such as reserpine and chlorothiazide. The relation between antidecarboxylation and hypotensive action is not clear. The bradycardia and postural hypotension suggests a sympathetic blockade.

KURLAND


There was no alteration in the corticosterone concentration in the plasma in the renal-hypertensive rats. In addition, the corticosterone content in the adrenal homogenates did not change. It is concluded that the activity of the adrenal glands is not significantly affected by hypertension.

RAKITA


A negative-pressure method of determining capillary resistance was used on 280 control subjects (140 men and 140 women) and on 234 hypertensive patients (115 men and 119 women). The interscapular area of the back was used and, by means of a two-way tap, a measured amount of negative pressure was applied to the skin for 30 seconds. All the control subjects had a diastolic pressure of less than 100 mm. Hg and were in good health. The hypertensive patients had a diastolic pressure of at least 100 mm. Hg or more on two recordings after at least a 10-minute rest. They were, however, free from any other pathology such as heart or kidney disease. The hypertensive patients were divided into three groups according to fundoscopic findings. Group I (161 patients with age range of 20 to 86 years) were asymptomatic and had normal ocular fundi or vascular changes only. Group II (49 patients with age range 35 to 69 years) had ocular exudates or hemorrhages, but no papilledema. Group III (24 patients with age range of 22 to 61 years) had hypertensive neuroretinopathy and papilledema. The authors found that sex was not a factor in the difference in capillary resistance at any age; also, capillary resistance in normal persons decreased with age. Patients in group I with symptomless hypertension had capillary resistance levels similar to the normal control group. However, patients in Groups II and III had lowered capillary resistance. The authors suggest that changes in the small blood vessels may occur when hypertension passes from the benign to the malignant phase.

KRAUSE


In six resting normal individuals, breathing 13 per cent oxygen for 20 minutes produced nearly a two-fold rise in pulmonary arterial pressure and a slight rise in heart rate and in cardiac output, but no significant changes in pulmonary wedge or systemic arterial pressures. Following the intrapulmonary arterial injection of 10 to 27 mg. of guanethidine, which was sufficient to abolish the arterial pressure overshoot after a Valsalva maneuver, there was essentially no change in these parameters while the subjects were either normoxic or hypoxic. It was concluded that pulmonary hypertension during hypoxia is not mediated by sympathetic nervous activity.

ROGERS


Alpha methyl dopa in doses varying from 0.75 Gm. to 3 Gm. daily for 16 weeks was administered to 15 patients with hypertension. The average falls in blood pressure were 36/23 mm. Hg standing and 31/18 mm. lying. The side-
Effects were mild, minor, and of short duration and included dry mouth, sleepiness, loss of energy, and indigestion. The results of this study indicate that alpha methyl dopa is a valuable new drug in the treatment of patients who do not have an adequate response to the milder hypertensive drugs such as phenobarbital, rauwolfia alkaloids, or chlorothiazide, but whose hypertension is not severe enough to warrant treatment with guanethidine. It is also useful in those patients who cannot tolerate guanethidine.

ROGERS


Some current views of and tests for renovascular hypertension are reviewed in this paper. Renal hypertension mimics other varieties of hypertension and probably accounts for 5 to 15 per cent of all cases of blood pressure elevation. Radioisotope renography plus excretory urography usually can detect disparity in kidney function, in which cases renal arteriography may be required. Treatment then depends on the over-all clinical situation, and it appears that most renal hypertensive patients respond to drug therapy. Surgical relief of renal ischemia, by one of a variety of technics, is generally desirable, although the precise indications for operation remain to be ascertained. Of 40 surgically treated patients observed for at least 1 year afterward, 19 had a blood pressure reduction to 140/90 mm. Hg or less, and 13 had a less pronounced reduction.

ROGERS


Observations on the blood pressure were made on three patients accidentally deprived of a single functioning kidney and kept alive for a period of 24 to 62 days thereafter. An additional patient had an elective bilateral nephrectomy prior to a successful transplantation of a normal kidney and was studied for 10 days prior to the placing of the kidney isograft. Unlike the experimental animal, the absence of renal tissue alone was not of itself sufficient to cause hypertension. There was no relationship between the presence or absence of hypertension and the serum sodium level. However, consistent with the experimental data, there was some relationship between the blood pressure levels and the body weight, with a subsequent fall in blood pressure following a dehydrating regimen. The authors thought that hypertension in man in the renoprival state was dependent upon exogenous factors.

KALMANSOHN

The clinical, laboratory, and radiographic studies of 50 patients with hypertensive disease who had upper abdominal murmurs are presented. These patients were selected from a hypertension clinic. In 66 per cent of the patients renal artery disease was found at lumbar aortography. About one third of the patients had a family history of hypertension, but about one half noted that the onset of hypertension was between the ages of 30 to 50 years. It is suggested that when an abdominal murmur of high pitch is found in a patient with hypertension, even with a normal intravenous pyelogram, renal artery stenosis is a good possibility.

Kayden


No significant differences in urinary aldosterone levels were noted among two groups of normotensive and hypertensive patients maintained on a daily sodium intake of 290 mEq. During restriction of sodium to 25 mEq a day, the normotensive group showed an increase in aldosterone excretion, peaking at 5 to 7 days and then returning to normal levels despite continued sodium restriction (12 days). In the hypertensive group the increase in aldosterone excretion was greater and remained sustained during the 12-day period of restriction. Following a sodium load, a greater reduction in aldosterone excretion was noted in the normotensive as compared to the hypertensive group. Essentially similar results were obtained by administration of 9-alpha fluorocortisol. It is concluded that a difference exists in the adaptive responses of aldosterone excretion between normotensive and hypertensive individuals.

Sancetta


The effects of synthetic angiotensin II on heart rate and blood pressure were determined in dogs under the influence of morphine and chloralose. Angiotensin in total doses of 2.5 to 20 µg rapidly injected intravenously in intact dogs caused an initial decrease in heart rate followed by a rise above the control level, despite the continued elevation of arterial blood pressure.


The extensive 6-year experience of the Cleveland Clinic Group with hypertension due to renal artery disease is reviewed. Six hundred and seventeen patients were selected for renal angiography during this period. Although no detailed criteria are presented for the selection of the hypertensive patient who should have angiography, the author makes the point that there


One day after nephrectomy, rabbits showed a slight rise in blood pressure but a marked (2 to 5 times) increase in the pressor reaction to epinephrine, nor-epinephrine, hypertensin, pituitrin, and serotonin. Soon afterwards the blood pressure fell and the animals died. After ureteral ligation there was a slight fall of blood pressure and no change in reactions to pressor substances. Nephrectomized rabbits maintained for 5 to 12 days by peritoneal dialysis developed marked hypertension and greatly increased reactivity to pressor substances. The hypertension was attributed to increased sensitivity to subliminal pressor hormones, and this in turn was related to the retention of sodium. Administration of deoxy-corticosterone and salt, which caused sodium retention, was also accompanied by hypertension and greatly increased reactivity to all of the above pressor substances.

Lepeschkin
was “a striking absence of the familial tendency of hypertensive disease among these patients.” Most of the patients were between 20 and 40 years of age and were equally divided between the sexes. Also of importance for selection of patients for renal angiography was the intravenous urogram, which had an accuracy of 70 to 80 per cent in predicting the presence of renal artery lesions. The diagnostic clues from the urogram include nonfunctioning kidney, delayed appearance of the radiopaque medium in one kidney, and a difference in size of the kidneys. Less obvious signs were paradoxical hyperconcentration, disparity in size of the collecting system of one kidney compared to the other, and partial atrophy of one kidney. Split renal function tests were of less value in predicting the presence or absence of unilateral renal artery disease, although at times they have proved helpful. Not enough experience with the radioactive renogram has as yet been accumulated to determine its reliability. Of the 617 hypertensive patients who were subjected to translumbar aortography, 173 were found to have occlusive disease of one or both renal arteries of whom 126 were treated surgically. Surgical treatment included nephrectomy, which is no longer commonly performed, segmental nephrectomy, endarterectomy, splenorenal anastomosis, and aortic renal artery bypass graft. Of the 126 patients, 10 died postoperatively and 76 patients were available for detailed follow-up evaluation. Of these 76 patients, 44 had a normal blood pressure 1 to 6 years postoperatively, three had only residual systolic hypertension, 12 had a “decreased blood pressure,” and 17 had an unchanged blood pressure. Successful remission of diastolic hypertension occurred in about 62 per cent of surgically treated patients and in another 16 per cent a considerable decrease, almost to normal, occurred in arterial pressure.


Blood pressures, antirenin titer, and renal renin concentration were determined in groups of normotensive, experimental hypertensive, and spontaneously hypertensive dogs, sacrificed at varying levels of neutralizing antibody (antirenin), which was produced by injection of hog kidney extracts containing renin. In both normotensive and hypertensive dogs a positive correlation was found between increasing titers of antirenin and renal renin concentrations. Thus, the authors postulate that antirenin neutralizes the endogenous renin of the experimental animals resulting in an increase of renal renin.


Subcutaneous administration of d-lysergic acid diethylamide (LSD) or 2-brom-d-lysergic acid diethylamide (BOL) markedly lowered the blood pressure of renal hypertensive rats. Drops of 50 to 120 mm. Hg below established hypertensive blood pressure levels occurred within 48 hours after administration of the drug. For the following 10 to 14 days the pressure fluctuated and then returned to original hypertensive levels. In normotensive control rats fluctuations of lesser absolute magnitude occurred within normal blood pressure ranges.


 Destruction of the central nervous system by pithing produced a much greater fall in pressure in the renal hypertensive than in the normal rat. The average postpith pressures in the two groups were identical. If pithing was done while angiotensin with pithing being infused into a normotensive rat the pressure fell more slowly and did not reach as low a level as it did following pithing in the hypertensive rat. Although it is not reasonable to interpret these findings as indicating that renal hypertension is caused by an increase in neurogenic tone, it appears unlikely that renal hypertension can be due to the direct pressor action of circulating angiotensin.


Attributing the failure of previous attempts to produce hypertension in humans by the administration of blood from toxemic patients to a lack of susceptibility of the recipients, the authors chose as recipients patients who had recently demonstrated their susceptibility to the hypertensive component of this disease. Five hundred milliliters of blood were obtained by phlebotomy at or near term from normal prenatal patients,
those with severe preeclampsia with no evidence of prior hypertension, and from patients previously diagnosed as having chronic hypertensive cardiovascular disease without evidence of superimposed toxemia. All blood was obtained and preserved by the usual blood bank technics. "Toxemia" blood was withdrawn 2.5 days prior to delivery and infused 6.3 days following delivery. The 500 ml of blood were given in approximately 55 minutes, blood pressure readings being taken by sphygmomanometer at 5-minute intervals. In one group of preeclamptic patients routine bank blood was substituted for their own blood for the infusion, while in another group 500 ml of each type of blood was given on different days in the puerperium. While both the systolic and diastolic pressures of preeclamptic patients receiving either bank blood or an autotransfusion rose, the pressure elevations following autotransfusion were significantly higher. Comparison of the pressure elevations following autotransfusion in toxemic patients and in the chronic hypertensive patients revealed the former to be significantly higher. While, as expected, a small pressure rise was also seen in normal subjects following autotransfusion, again this was significantly less than the rise following autotransfusion in toxemic patients. Finally, two puerperal patients who had completely normal pregnancies have been given "toxemic" blood during the immediate puerperium with no remarkable changes in blood pressure resulting.

Fox


In previous studies, the authors have demonstrated that the aortic wall of rats with various forms of experimental hypertension contains an increased amount of sodium, potassium, and water per unit of dry weight. The present study was to measure these substances in the arterioles. Fifty-five rats were made hypertensive by narrowing the renal artery of their one remaining kidney. Seven months later these hypertensive rats were divided into two evenly matched groups. The rats in one group were "cured" of their hypertension by releasing the arterial constriction. The rats in the other group had a comparable sham operation. One week after either procedure, a sample of arteriolar tissue from vessels of 0.025 to 0.14 mm. outer diameter was obtained. Arterioles from the rats with continuing hypertension had 16 per cent more sodium per unit of dry weight than those from the rats "cured" of hypertension ($p < 0.0001$). The potassium in the arteriolar wall was similar in the five groups and a difference in chloride content was of borderline significance ($p = 0.08$). At least part of the increment of sodium in the hypertensive arterioles appears to be located intracellularly. A 10 per cent increase in the total cation content, sodium plus potassium, in the hypertensive arterioles ($p = 0.0004$) may indicate that these vessels have about 10 per cent more water per unit of dry weight than the arterioles from rats with "cured" hypertension. The arterioles from the rats with either continuing or "cured" hypertension had undergone about the same degree of thickening; hence the difference in sodium content is probably not related to this. The extra sodium in a hypertensive arteriole may contribute to its narrowed lumen.

Kayden