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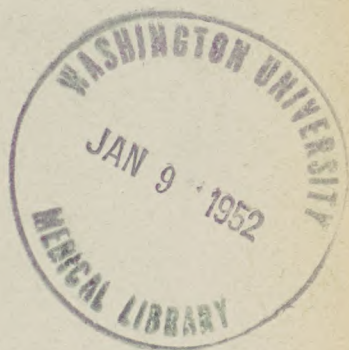
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- The Pathogenesis of Hypertension
Henry A. Schroeder, M.D.
- 1951 Alumni Reunion
- Dr. Carl A. Moyer, New Head of Department of Surgery
- Report on Dormitory Fund Campaign



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PATHOGENESIS OF HYPERTENSION*

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In a symposium conducted by The American Journal of Medicine on the subject of arterial hypertension a number of studies and reviews were presented which described some of the modern concepts of the disease and its various ramifications. Attempts to integrate the several concepts leave one somewhat confused, however, by the different points of view expressed. Understanding of the condition has progressed rather rapidly during the last fifteen years. Enough evidence is now at hand to formulate a tentative hypothesis and to organize the experimental and clinical facts in such a way as to arrive at a theory which will satisfy most of them. The purpose of this report is to develop such a hypothesis, to examine it and to evaluate it in the light of known facts and suggestive mechanisms.

Arterial hypertension, especially that variety called "essential," is a psycho-

somatic disorder. Therefore, it is necessary to examine first in what way the psyche differs from "normal"; second, in what way the psyche influences the soma; and third, in what way the soma responds to such influences. The various pathways through which these influences can act and the immediate and long term effects of their action must be considered closely. Furthermore, examination must be made of the several mechanisms by which a condition such as hypertension may be maintained, may fluctuate in intensity and may finally change in a direction incompatible with life.

The influences and their interaction will be discussed in the order in which they appear to act. They include: first, the influence of the psyche and of the nervous system, which appear to be common underlying etiologic factors; second, sustaining factors, which include neurogenic, nephrogenic and

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adrenocorticogenic influences concerned in maintaining elevation of the blood pressure; and third, the consequent effects of chronic hypertension to cause pathologic changes in arterioles. In addition an attempt will be made to indicate gaps in basic knowledge, the filling of which is necessary for a thorough understanding of these important and prevalent diseases. Various pathogenetic clinical types will be described. Those forms of arterial hypertension believed to be secondary to other conditions, e.g., disturbances of the endocrine glands (such as adrenal tumors), renal excretory insufficiency (from obstruction, glomerulonephritis or pyelonephritis) and types due to congenital anomalies (such as coarctation of the aorta) or to collagenous disease (such as polyarteritis nodosa) will be considered only insofar as they contribute to our understanding of "essential" hypertension.

ETIOLOGIC FACTORS

Psychogenic Factors.† There appears to be present in practically all cases of primary arterial hypertension certain alterations of personality which are quite consistent. These disturbances have not been fully appreciated until recently, although for many years the "nervousness," "tenseness" and emo-

† "Psychic" and "neurogenic" are fairly definite terms describing certain areas believed to be important in etiology. A more inclusive but less well defined term is the "constitutional factor." It would not be amiss to speak of the "hypertensive diathesis" as that combination of conditions, hereditary, psychic, nervous and vascular which predisposes an individual to the development of hypertension and/or the ability to react to stresses by means of vasoconstriction.

tional lability of hypertensive patients has been noticed. Under modern conditions in this country 40 to 50 per cent of the population appears to develop hypertension at some period of life, usually after the age of forty. If, as recent studies suggest, there is a disturbance or deficiency in the personalities of such individuals, the frequency of this deficiency does not make it a normal state.

Evidence for disturbances of personality is found in three fairly extensive surveys. Weiss, studying ninety-three patients suffering from "essential" hypertension, concluded that emotional factors were chiefly related to the onset of hypertension in fifty-three, and related in a minor way in thirty-three. In only seven cases was there apparently no relationship. Like others, he found that inhibited aggression ("chronic rage") was of great importance and suspected some specific relationship of this disturbance to hypertension. Binger et al. found that a group of twenty-four hypertensive patients carefully studied as to life history and personality showed a particular psychologic configuration. The outstanding elements were "exaggerated dependent strivings, submissiveness coupled with stubbornness, feeling of weakness and defenselessness, suppression of hostility, fear of injury and emotional detachment. In addition, there was a tendency to develop acute emotional disorders characterized mainly by anxiety and depression and often associated with temporary failure of the usual techniques of mastery of the environment. This acute failure of the integrative functions of

personality seems to result from the inefficiency of the patterns of defense against anxiety and a weakness of the repressive mechanisms."

The third study was made at this institution by Gressel et al. Six elements of the personality were particularly singled out for examination. Fifty hypertensive patients, fifty patients suffering from psychoneuroses without hypertension and approximately an equal number suffering from chronic diseases not believed to be psychosomatic and with normal blood pressures were included. Objectivity in the evaluation of personalities was stressed and the results analyzed statistically. Of the facets of the personality which Binger described only two were found to be significantly predominant in the hypertensive group: obsessive-compulsive tendencies and subnormal assertiveness. Anxiety and hysteria occurred frequently and similarly in both hypertensive and psychoneurotic patients to a significantly greater extent than in the other group. Impulsive traits were present with similar frequency in all groups, and no significant trends were found for the traits of excessive insecurity, depression and frustrated ambition. While it is impossible to quantitate a subjective impression, it is possible to ascertain by some of the better psychologic methods whether certain qualities are present or not, and to a certain extent to what degree they are present. Therefore, the implications of these two latter studies are that certain personality defects not common to the general population are present in individuals who develop hypertension. The extent of

these defects varies considerably from person to person. Similar personality structures were found by Wolf et al. using a group of asthmatics as controls.

Are these disturbances of personality secondary to hypertension or are they concerned in its causation? One-third of Binger's cases were in early mild stages of the disease. In addition, "evidences of this peculiar structure of personality are discernible before hypertension, or its prodromal symptoms, make their appearance." The same conclusions could be drawn by Gressel, for the manifestations of the "hypertensive personality" were present to an equal degree in early mild and late severe cases, and antedated by many years the onset of hypertension. Therefore, it is improbable that the changes were the result of hypertension; their presence implies a causal relationship. It is not known whether defects of personality such as these are acquired, that is, developed during childhood, youth and possibly adult life, or whether they are inherited. Our own view of the matter is that these defects are developmental, a result of either the stresses of the modern environment or of one considered hostile by the individual, and presenting problems incapable of solution with the capabilities at hand.

For the moment, therefore, let us assume that defects of personality of a specific nature exist which are in themselves conducive to conflict when an unfavorable environment impinges upon the psyche. This, of course, is common to everyone. The manner in which nervous tension resulting from the repression of these conflicts is resolved is not

the same in all individuals. In those predisposed to hypertension, however, it would appear that the sympathetic nervous system and in some the adrenal cortex respond to or are activated by these repressed conflicts. It would also appear that this method of reaction occurs in from 40 to 50 per cent of the population. Variations in degree, type and severity of reaction in different individuals suggests that this factor has wide differences of intensity while showing a common pattern.

The habit of transferring these conflicts to the autonomic nervous system probably becomes more firmly established as the years pass. Major calamities may be necessary to evoke reactions in most younger individuals; however, as this habit of reacting becomes established the trigger mechanism may be set off by smaller and smaller stimuli, and those minor irritations which are present in the lives of all of us become major ones insofar as their effects upon the body are concerned. According to this concept, hypertension is a disorder resulting from the socio-economic complexities of modern civilization and the stresses and conflicts which they impose upon man who is trying to adapt. Hypertension is therefore one of the prices man must pay because of his own deficiencies in coping with the environment of the civilization he has devised for himself. Because that civilization has resulted in conflicts which are beyond his capacities for solution by himself, a state of imbalance of his vegetative functions has resulted which leads to disease.

Hereditary Factor. An hereditary factor is suggested by the work of Hines who found hyper-reactivity of the vascular system to a painful stimulus (cold pressor test) a familial trait which predisposed to hypertension. Over 40 per cent of the off-spring exhibited vascular hyper-reactivity when one parent had hypertension or hyper-reactivity; when both parents were either hypertensive or hyper-reactors, 95 per cent of their children showed this trait. When both parents were hypertensive, about 90 per cent of their children eventually developed hypertension. That the hereditary trait is not a prerequisite is suggested, however, by the absence of a familial history in over half of cases of hypertension.

Neurogenic Factor. The neurogenic factor is mediated by that portion of the nervous system which reacts by peripheral manifestation to conflicts produced in the psyche. The essential parts involved in hypertension appear to be the hypothalamus and the sympathetic nervous system. While the exact pathways by which cortical impulses travel to the hypothalamus and the mechanisms by which conflicts can set off emotional discharges are not thoroughly understood, the end results of such discharges initiated by the cortex are well known. For some reason, either through development or inheritance, patients subject to hypertension appear to react to emotional tension and to conflicts by means of a discharge of the sympathetic nervous system. It is possible that this method of reaction, the result of internal "boiling over," is an inherited defect; other portions of the

population may react to analogous situations of conflict through, for example, the parasympathetic nervous system. An understanding of any psychosomatic disease involves not only a knowledge of the psyche and the soma but especially of the connections between them, of their effects and of their controls in the brain and by endocrine glands.

Evidence for hyperactivity of the sympathetic nervous system in hypertension is for the most part based upon clinical observations and reasoning. Patients in early or prehypertensive stages develop under proper stimuli manifestations suggesting discharges of their sympathetic nervous systems, with peripheral vasoconstriction. The cold pressor test demonstrates hyper-reactors to painful stimuli; hyper-reactors to cold or psychic stimuli usually develop hypertension and sympathetic hyperactivity offers the most probable explanation for this phenomenon. Many hypertensive individuals at times have cold, clammy, intensely constricted extremities, perspire profusely under emotional tension, especially on palms of hands, on feet and in axillae, and show vasomotor instability by means of flushing of the skin and variations in blood pressure and pulse rate. Acute emotional outbursts are occasionally seen, with watering of the eyes, palpitation, tachycardia, a blotchy erythema of the skin of the neck, chest and back, and further elevation of blood pressure. In some cases this tendency is so pronounced as to appear in acute attacks with signs suggesting that the hypothalamus has suddenly discharged via the sympathetic nervous system in a complicated man-

ner not thoroughly understood. This sequence of events has been termed the "hypertensive diencephalic syndrome." The intradermal injection of histamine will produce these discharges in susceptible individuals.

Further suggestive evidence is based upon the observations that autonomic blocking agents will lower blood pressure to varying levels presumably by interruption of neurogenic impulses in ganglia, that extensive sympathectomy sometimes abolishes hypertension for several years, that blood pressure levels may be extremely variable in some hypertensive patients independent of cardiac output and that blood vessels of the extremities are hyper-reactive to minor stimuli. The studies which have shown little neurogenic activity in hypertension may have been made in advanced cases; as will be discussed, other factors may operate in them to the partial exclusion of the neurogenic. When neurogenic influences are clinically evident, symptoms, signs and findings strongly suggest a form of sympathetic activity not dependent upon the release of epinephrine but possibly similar to that resulting from the action of norepinephrine. Undoubtedly the peripheral resistance varies in these individuals from time to time.

SUSTAINING FACTORS

Neurogenic* vasoconstriction and its effects can be profound and prolonged. When sympathetic discharges take place or when the sympathetic nervous system

* The term "neurogenic" is used in a broad sense, and includes the actions of chemical effector substances acting on the smooth mus-

is hyperactive for prolonged periods, the cardiovascular and other systems are affected. Of most concern to the problem of hypertension are the effects on 1) blood vessels, (2) kidneys and (3) endocrine organs.

Neurogenic vasoconstriction probably occurs in all organs and tissues of the body to some extent. When this is generalized, blood pressure rises. To what degree each local peripheral bed contributes to the generalized increase in peripheral resistance is not known, but the splanchnic area may be somewhat more labile in this respect than that of striated muscles, for example. Emergency responses of the cardiovascular system mediated through sympathetic nerves and the adrenal medulla are well understood. There is a tendency to shift blood away from splanchnic areas and into those used for emergencies, e.g., muscles. The output of the heart may increase transiently but is soon compensated for by the diminished blood flow resulting from the increased resistance. Renal vasoconstriction occurs and is prolonged. Epinephrine is discharged from the adrenal medulla and the cortex is stimulated either directly or via the pituitary's adrenocorticotrophic hormone. Sympathin E, which is probably nor-epinephrine, may be formed in increased amounts or may be activated excessively. The net result can be not only a transient hypertension but one

cles of blood vessels and on organs as well as those resulting from nerve-borne impulses. In this discussion "neurogenic" usually refers to the sympathetic component of the autonomic nervous system with its "adrenergic" effects, although parasympathetic actions and their cholinergic chemical effectors are also "neurogenic."

prolonged for many minutes or even hours, with residua lasting much longer, until the reaction wears off or compensatory functions come into play.

The two known chemical effectors of the sympathetic nervous system, epinephrine and nor-epinephrine, have quite different actions. Epinephrine increases cardiac output and decreases peripheral resistance in physiologic doses; minute doses can cause vasodilatation. Norepinephrine (1-arterenol) on the other hand acts as a generalized vasoconstrictor substance with little or no effect upon cardiac output. The hemodynamic profile produced by this naturally occurring substance is similar to that seen in chronic arterial hypertension with the exception that pulmonary vascular resistance is increased; in hypertension it is usually but not always normal. Nor-epinephrine could account, however, for part of the mechanism of neurogenic vasoconstriction.

As part of splanchnic vasoconstriction, renal blood flow is reduced, the efferent arterioles of the glomeruli being predominantly affected, although strong stimulation causes constriction of both efferent and afferent arterioles. This reduction may last a long time after epinephrine is injected or vasoconstriction is produced by alarm. The slow return to normal is worthy of emphasis. In Smith's original experiments appreciable reduction of renal plasma flow and increase in filtration rate were noticeable for an hour or longer after the initial emotional or chemical stimulus.

Wolf et al. explored the relation of the psychic to the somatic factor by

measuring renal blood flow and arterial pressure before, during and after traumatic interviews concerned with emotional problems. Most of their cases exhibited labile blood pressures which, while often at high levels, still retained the ability to drop to near normal ranges and were therefore not in late stages of the disease. Their studies clearly demonstrated the close dependency of arterial pressure and renal blood flow upon the immediate emotional situation. Discussion of topics involving personal conflicts caused prolonged rises in blood pressure, reductions in renal plasma flow, and renal hemodynamic pictures consistent with those seen in more severe essential hypertension. Studies made over long intervals likewise showed the relation of the environmental situation to the level of blood pressure. The psychoneurogenic mechanism of renal and peripheral vasoconstriction therefore seems clearly established. Furthermore, this pattern of reaction was abolished after lumbodorsal sympathectomy, indicating the sympathetic nature of the reaction.

Nephrogenic Factors. While blood pressure in susceptible individuals may rise acutely as a result of generalized neurogenic vasoconstriction, the other functions which control blood pressure more slowly probably account for most of the prolonged changes which occur. The kidneys particularly are implicated. Renal ischemia on a neurogenic basis may cause the kidneys to excrete into the circulating blood humoral pressor substances which act for a long time upon blood vessels, increasing periph-

eral resistance and raising blood pressure. There are many pressor mechanisms of humoral nature present in the body; at least sixteen different substances have been detected, identified, or believed implicated (Table I) of which eight are nephrogenic in origin. If nephrogenic substances are active, the results of their actions are known. Until the unknown pressor substance which has been postulated and detected by various methods has been identified chemically we shall call it the *Hochdruckstoff*. It is defined as a substance or group of substances with the following pharmacologic actions: (1) elevation of blood pressure; (2) generalized and relatively equal vasoconstriction of all vascular beds except the pulmonary; (3) renal vasoconstriction greater on efferent than on afferent arterioles; (4) no pronounced alteration of cardiac output or pulse rate; (5) no obvious sympathomimetic effects (sweating, pallor, spasm of sphincters, etc.); (6) no change in blood volume or blood viscosity. The hypothetical *Hochdruckstoff* is probably concerned in chronic hypertension, maintaining blood pressure at constantly elevated levels superimposed upon which neurogenic mechanisms operate. Prolonged action is unnecessary provided continuous formation is assured. Any naturally occurring substance having these actions is under suspicion as a causative agent in hypertension. Six naturally occurring substances are probably somewhat similar pharmacologically to the *Hochdruckstoff*. (Table I.)

The best known renal pressor mechanism is the one involving renin and

TABLE I
PRESSOR SUBSTANCES POSSIBLY RELATED TO HYPERTENSION

Name	Source	Accessory Conditions	Chemical Nature	Pharmacologic Action	Obtained Pure	Found in Hypertension	Similarity to <i>Hochdruckstoff</i>
Proteins:							
Renin	Kidney	Renal ischemia	Protein	Prolonged	No	Acute	Yes
Prolonged pressor substance	Kidney and blood	Hypotension (Renal ischemia?)	Protein	Very Prolonged	No	?*	Unknown
Peptides:							
Hypertension	α_2 Globulin	Renin	Peptide	Acute	No	Acute	Yes
Pepsitensin	α_2 Globulin	Pepsin	Peptide	Acute	No	No	Yes (?)
Serotonin	Blood	Standing	Peptide	Acute	Yes ?	No	Unknown
VEM	Kidney	Renal ischemia and anoxia	Peptide (?)	Prolonged	No	Yes	Unknown
Amines:							
"Amines"	Blood	Renal ischemia and anoxia	Amine	Acute	Yes	Yes	Possibly (mixture)
Nor-epinephrine	Tissue	Nervous stimuli	Amine	Acute	Yes	?	Yes (?)
Urosympathin	Urine	-----	Amine	Acute	No	Yes	Unknown
v. Euler's substance	Blood and tissues	-----	Nor-epinephrine?	Acute	No	No	Yes (?)
Victor's material	Kidney	Anaerobic autolysis	Tyramine?	Acute	No	No	Unknown
Pherentasin	Arterial blood	Hypertension (renal)	Amine	Prolonged	Yes	Yes	Unknown
Nicotine Bases:							
Lockett's base	Urine and blood	Renal ischemia	Complex alkaloid	Acute	No	Experimental only	Unknown
Urohypertensin	Urine	-----	Nicotine base?	Acute	No	Yes ?	Unknown
Others:							
Nephrin	Kidney	-----	Unknown	Prolonged	No	Yes	Unknown
Desoxycorticosterone	Adrenal cortex?	Salt and hypertension?	Steroid	Prolonged	Yes	Certain types?	Yes (?)

* Where a question mark (?) is shown, the characteristic of the material is in doubt. It is obvious that the chemical identification and the pharmacology of most of these substances has been insufficiently studied.

hypertensin. It is not necessary to explain at length the reactions which produce hypertensin, concerning which there is a large literature. Renin, a proteolytic enzyme in kidney, acts on a specific substrate, an α_2 globulin produced probably by liver, called hypertensinogen, to form a peptide, hypertensin. An enzyme in blood, hypertensinase, inactivates hypertensin. The renin pressor mechanism is active in shock, congestive heart failure and acute renal ischemia. Proof of abnormal activity in chronic hypertension is lacking. Here Renin is antigenic and to some extent species-specific, and anti-renin in some cases will lower the blood pressure of experimental hypertensive dogs. From the evidence to date it is probable that the renin mechanism acts in a slower and more prolonged manner than other emergency mechanisms, may be concerned in relatively rapid changes in blood pressure which are, however, still slower than those produced by neurogenic vasoconstriction, and may operate at all levels of blood pressure. Renin does not appear to be the primary effector substance in hypertension and, although similar in action, is probably not the *Hochdruckstoff*.

Enough evidence is now at hand to reinforce the belief that other, probably nephrogenic humoral substances are present. The brilliant work of Shorr and his group in demonstrating vasoexcitator and vasodepressor materials acting to maintain normal vasomotor tone has led to the discovery of increased amounts of both substances in the blood of hypertensive animals and human beings. The vasoexcitator mate-

rial was demonstrated indirectly, the vasodepressor directly. The latter has been identified as ferritin; the former appear in the anaerobic autolysis of kidney and after chronic renal ischemia. It is not hypertension but its general pharmacology is unknown.

An unidentified substance which we have named pherentasin* has been isolated from the arterial blood of hypertensive patients in this laboratory, using the blood pressure of the intact hypertensive rat for assay. When purified, it appears to be present in very small amounts, 10 to 20 γ per L. of blood. The pressor effect of pherentasin on blood pressure of the rat, unlike that of hypertensin, is delayed and prolonged. It contains free amine and carbonyl groups necessary for biological activity, is of small molecular size, non-protein and is probably an amine. Pherentasin is found to a large extent in the blood of patients with the more severe forms of hypertension, especially those who show evidence of minor renal damage, but it has not been proven to be renal in origin. Patients with "neurogenic" hypertension who have little vascular damage exhibit only small amounts in their blood. A depressor substance is also present which resembles pharmacologically and spectrophotometrically an adenylyl derivative. The pressor material has a reaction similar to Shorr's vasoexcitator material when his test methods are employed.

Although not a necessary characteristic, substances giving prolonged pres-

* $\Phi\acute{\epsilon}\rho\omega$ = hold up; $\xi\gamma\tau\alpha\iota\varsigma$ = pressure. The name was suggested by Henry A. Schroeder, Jr.

pressor responses have been of interest in this problem. The vasoexcitor material of Shorr and pherentasin have equally long actions. A prolonged pressor substance has been isolated from the blood of animals in shock; it resembles but is not renin. Other substances under suspicion are: nor-epinephrine which acts in many ways similarly to the theoretic *Hochdruckstoff*, serotonin isolated from red blood cells and amines. It has been shown that ischemic kidneys decarboxylate but do not deaminate certain amino acids; the amines so formed are pressor and are oxidized by a specific amine oxidase. "Amines" are increased above normal levels in extracts of hypertensive blood; this abnormality may represent a defect in the metabolism of certain amino acids, without reference to a specific pressor amine. None of these substances has been closely identified with human hypertension to date. (Table I.) The similarity of the action of nor-adrenalin to the *Hochdruckstoff* is of great interest.

Adrenocorticogenic Factors. The adrenal cortex also can be stimulated to discharge by sympathetic nerves, either through production of adrenocorticotrophic hormone from the pituitary or directly via sympathetic nerve endings in the medulla. A pressor mechanism which appears to act principally in hypertension is probably mediated by the adrenal cortex. Hypertension is common in adrenal cortical hyperfunction and this fact has led to a considerable amount of investigation on the role of this organ. Only a few facts are known: (1) Desoxycorticosterone acetate (DCA) injected daily raises blood pres-

sure in hypertensive subjects. The injection of this material, however, if continued loses its action on blood pressure, weight and urinary chlorides. In addition, blood pressure does not rise when dietary salt is also restricted. (2) DCA or desoxycorticosterone glucoside acts as a prolonged pressor substance when injected intravenously, but only in hypertensive subjects (and in some dogs and rats). (3) DCA probably does not act by stimulating a renal pressor mechanism. Dogs who respond to its intravenous injection by hypertension respond equally well after their kidneys have been removed. Renal plasma flow and glomerular filtration rate in patients are not altered during the pressor response in the direction of efferent arteriolar constriction. (4) Certain hypertensive patients maintain blood pressure levels which are high when the intake of salt is high and low when it is restricted. Blood pressure of these individuals is apt to be sensitive to DCA or DCG, a very prolonged effect following a single injection. We have been unable to isolate pherentasin from their blood by methods giving results in other cases. It is probable that patients of this variety exhibit a different pathogenesis, as will be discussed later, for the sodium and chloride content of their sweat is low.*

* There is little direct evidence that the adrenal cortex is in a hyperactive state in most individuals with hypertension. The urinary excretion of corticoids and 17-ketosteroids is not disturbed, there is no alteration in sensitivity of blood sugar to insulin; and the salt concentration of sweat is normal. A well defined group, however, does show abnormalities (vide infra).

Effects of These Pressor Mechanisms on Various Vascular Beds. Stimulation of sympathetic nerves leads to increased peripheral resistance especially of the splanchnic bed. The relative resistances of various local circulations in chronic hypertension have not been thoroughly studied and therefore little is known regarding the distribution of the effective peripheral resistance. This is an important question for its solution may give a clue to the identity of the *Hochdruckstoff* and to the relative part played by neurogenic and humoral mechanisms. Pickering and Prinzmetal and Wilson believed that humoral mechanisms acted to an approximately equal degree on all arterioles throughout the body. Others, notably Sheard, Abramson and Stewart et al. produced indirect evidence that certain areas might be more affected by vasoconstriction than others; these differences were altered by sympathectomy. To generalize from experiments made on the assumption that a single mechanism operates to elevate blood pressure is probably erroneous. In certain cases the neurogenic element predominates, in others one or more humoral ones. It is necessary, therefore, to know as exactly as possible where the resistance lies—in the splanchnic bed predominantly, in all beds equally, or in intermittent variation between them. By the use of a photoelectric rectovaginal plethysmograph, marked lability of an area of the splanchnic bed in some hypertensive women has been demonstrated.

Studies have principally concerned themselves with the renal vascular bed. Efferent arteriolar constriction mea-

sured by clearance technics is uniformly present in chronic sustained hypertension but may be absent during early stages. The conditions of the experimental procedure and the fluctuating nature of early hypertension, which usually contains a large neurogenic component, do not lend themselves to exact determinations of renal blood flow during the stresses of daily living. Therefore, the most that can be said is that *at rest* early hypertensives do not show renal vasoconstriction while well established ones do.

EFFECTS OF HYPERTENSION ON THE VASCULAR SYSTEM

The presence of prolonged arterial hypertension leads to changes in the arterioles throughout the body but more especially in those of the kidneys. This view is at variance with that of Goldblatt who believes that afferent renal arteriolar disease may come first and cause hypertension by producing renal ischemia. Evidence for the secondary nature of arteriolar disease in increasing. Renal biopsy studies on man have shown none to minimal renal vascular disease in 46 per cent of 500 hypertensive patients. The argument that the small size of the renal biopsy did not give a true picture of the whole kidney is not valid; bilateral biopsies were similar; few pathologists study more than one good section of kidney, and the consistency of the findings strongly suggested that a representative sample was obtained. Various unilateral renal affections in rats caused chronic hypertension with vascular disease, even arteriolar necrosis, in the opposite kidney.

Because dogs usually require constriction of *both* renal arteries to maintain hypertension, the experimental approach is difficult; however, in this laboratory by using special technics and choosing nervous, high-strung animals, we have produced hypertension which has slowly caused some arteriolar changes in a contralateral kidney; these studies are continuing. Unilateral renal affections with hypertension in man are associated with arteriolar disease in the opposite kidney. Removal of a unilateral ischemic kidney in the rabbit does not "cure" the hypertension caused by the ischemia. Therefore, in the rat, man and probably the dog arteriolar nephrosclerosis appears to be the result of, and not the cause of hypertension, although it must, by the very nature of the lesion, act to maintain renal ischemia and therefore contribute to an already elevated blood pressure.

There is further suggestive evidence that hypertension damages arterioles. The kidney of the dog on which there is a Goldblatt clamp is "protected" against vascular disease. Chronic pulmonary hypertension resulting from long-standing mitral stenosis or pulmonary emphysema is accompanied by pulmonary arteriolar — and arterial — lesions similar to those seen in peripheral hypertension. If lesions accompany hypertension in either the greater or the lesser circulation, are confined to that circulation and do not uniformly occur in its absence, the evidence becomes more than suggestive that high arterial pressure can cause vascular disease. In benign arteriolar sclerosis we can assume that the lesions are those

secondary to chronic strain and overwork, until proof is offered that the *Hochdruckstoff* causes changes in blood vessels. Strain may also predispose larger vessels to arteriosclerosis if the metabolic changes necessary for its development have occurred. The necrotizing lesions seen in the malignant stage of hypertension may be the result of toxic substances released by severely ischemic kidneys; experiments in dogs suggest that this may be so.

The Vicious Circle of Arteriolar Disease, Renal Ischemia and Hypertension. When hypertension has become well established through the mechanisms described previously and arteriolar nephrosclerosis has appeared as a result, the renal vascular changes themselves cause renal ischemia.* Superimposed on this "fixed" renal ischemia of disease is the intermittent renal ischemia of neurogenic vasoconstriction. When the latter is abolished or modified by surgical or chemical sympathectomy, or

* While actually meaning a lowered flow of blood through the kidneys, the term renal ischemia as used in this discussion also includes situations in which ischemia is potential; that is, absent or of minor degree when the head of arterial pressure is high but present when it is low. Structural changes in renal arterioles or arteries, for example, will lead to such a condition. The assumption is therefore made that renal resistance to blood flow is increased, that this increase leads to ischemia, that ischemia leads to the formation of nephrogenic pressor substances and therefore to a higher peripheral pressure which partly compensates, and that compensation leads to the establishment of the pressure-flow relationships of the kidney at a different level. For this level to be maintained we can assume that intermittent ischemia (or hypoxia) occurs in which flow is reduced below renal needs or requirements, and which recalls into action these regulative mechanisms for peripheral pressure.

by adequate rest, sedation and psychotherapy, a "floor" is reached; the level of blood pressure of this "floor" is probably maintained principally by humoral vasoconstrictor substances, with possibly a small contributing element due to organic arteriolar narrowing in the periphery. Therefore, complete reversal of the elevated blood pressure to normal levels by any means can occur only in the earlier stages which are associated with little or no organic renal vascular damage.

The superimposition of neurogenic vasoconstriction on organic, with intermittent episodes of greater degrees of renal ischemia, leads to the intermittent production of more *Hochdruckstoff*; the resulting higher arterial pressure gradually leads to greater degrees of nephrosclerosis, and the nephrogenic "floor" becomes fixed at higher and higher levels. After varying periods of time

some organ gives way to the constant strain and an accident occurs. It is not within the province of this discussion to describe the secondary damage to heart and brain. The cardiac hypertrophy is probably the result of overwork, and cardiac accidents secondary to overstrain or to coronary arterial diseases. Cerebral blood vessels rupture from strain on areas weakened by associated vascular disease or abnormalities because of the relative thinness of their adventitia. Renal insufficiency supervenes on three accounts: first, because (for unknown reasons) the nephrosclerosis progresses rapidly; second, because accessory renal parenchymal diseases are present; and third, because in some way, possibly through outside influences, this vicious circle becomes a rapidly descending spiral, renal ischemia becoming "decompensated" in spite of higher blood pressure. Signs of acute

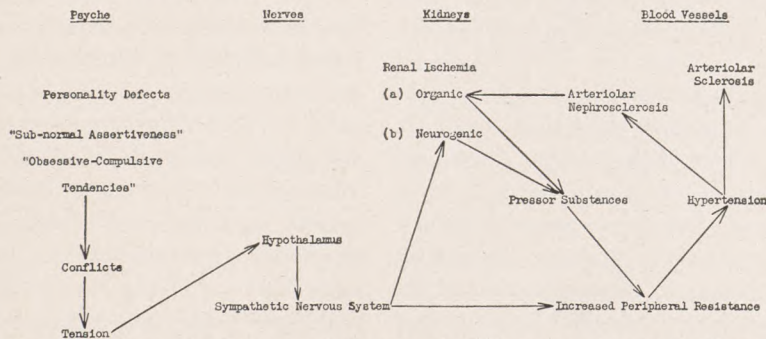


FIG. 1. Pathogenesis of neurogenic hypertension. Sequence of events leading to sustained hypertension when accessory etiologic factors are absent. Repressed emotional tension causes discharges of the sympathetic nervous system via the mid-brain leading to generalized vasoconstriction. The kidneys are included in the response. The resultant disturbance of intrarenal hemodynamics leads to the production of pressor substances. The replacement of humoral for neurogenic vasoconstriction sustains the hypertension for longer periods of time. Repeated discharges eventually lead to sustained hypertension which in turn causes changes in the walls of the arterioles, especially those of the kidneys. The organic renal ischemia caused thereby promotes a vicious circle in which the continued production of pressor substances is predominant. The neurogenic element is superimposed thereon.

vascular damage in the ocular fundi, progressive renal insufficiency and death from uremia characterize this "malignant stage" of hypertension, which sometimes may be variable in its course and occasionally is reversible.

PROBABLE PATHOGENESIS

Therefore, the pathogenesis of hypertension can be considered somewhat according to the following: Nervous stimuli arising in the brain from emotional tensions are discharged via the hypothalamus and the sympathetic nervous system. The diathesis which produces this type of discharge may be hereditary or developmental. As these discharges occur they produce generalized neurogenic vasoconstriction which includes the kidneys. Vasoconstriction in the kidneys results in the formation of blood-borne pressor substances which serve to maintain the elevated blood pressure. The results of such an emotional discharge in predisposed individuals may persist for some length of time, perhaps a matter of hours. Subsequent repetitive discharges result in establishment of the pattern of reaction, each period of renal vasoconstriction producing pressor substances. Eventually these pressor substances themselves cause changes in the architecture of the renal arteriolar bed which by their very nature result in organic renal ischemia. Thus hypertension which at first was intermittent becomes constant. (Fig. 1.) It is probable that almost all patients with hypertension not secondary to other diseases demonstrate to a greater or lesser extent this pattern of reaction. When the extent is considerable, "neu-

rogenic" or "psychoneurogenic" hypertension is the result. The various influences causing these changes can be modified by accessory conditions or diseases affecting the several organs or systems involved. The "vicious circle" of renal ischemia \rightarrow pressor substances \rightarrow hypertension \rightarrow renal arteriolar sclerosis therefore can be initiated by disturbances in intermediate pathways as well as of the whole pathogenic mechanism.

CLINICAL TYPES OF HYPERTENSION DEPENDENT ON ACCESSORY ETIOLOGIC FACTORS

Up to this point we have described the psychic and neurogenic aspects of arterial hypertension and its organic consequences. Other factors organic in nature can contribute to hypertension in addition to the nervous. These are: parenchymal disease of one or both kidneys, arteriosclerosis with narrowing of one or both renal arteries at their mouths or along their courses extra- and intrarenally, and disturbances of the endocrine glands, especially of the adrenal cortex. When accessory diseases are absent, usually the condition is pure "neurogenic" hypertension. When accessory conditions are present in addition to the psychoneurogenic, it appears preferable so to designate cases suffering from them, since course, prognosis and therapy may be quite different. While differential diagnosis between the various types may be difficult if not impossible when one examines a short segment of the course of the disease without a thorough knowledge of the patient's history, well documented cases usually can be classified by clinicians

with experience and understanding. A brief description of the points of differentiation of "pure" cases may be helpful although many are "mixed."

Renal Hypertension. While the nephrogenic factor probably operates in most cases of hypertension this term is used to designate those with renal parenchymal disease, including pyelonephritis, glomerulonephritis "masked" by hypertension, chronic urinary obstruction, calculi, etc., which are unsuspected until searched for thoroughly or the signs of which become less evident as nephrosclerosis advances, hypertension being the presenting condition. The urologic disease is probably the initiating factor. This view is at variance with that of Smith who believes that there is little evidence that the kidneys play a primary or initiating role since removal of one diseased kidney rarely "cures" the hypertension. When one accepts the concept, however, that arteriolar nephrosclerosis is the *result* of hypertension and can maintain renal ischemia and hypertension even when its causes are removed, the view that urologic disease may initiate hypertension in susceptible individuals becomes more tenable. When urologic disease is unilateral, failure of nephrectomy to lower the blood pressure therefore is explained by the presence of secondary contralateral arteriolar nephrosclerosis; favorable result can be expected in those relatively rare instances when arteriolar disease in the opposite kidney is minor or absent.

In a recent excellent review Smith listed only forty-seven cases of hypertension associated with urologic dis-

eases apparently "cured" by nephrectomy, commenting on the relative rarity of proven renal hypertension. It is interesting that the average age of these patients was just over twenty-seven years, that all but ten were under forty, and nineteen were under twenty when nephrectomy was performed, a relatively young group for advanced hypertension. To these can be added two cases of our own, previously reported, who have normal blood pressures more than ten years after nephrectomy. If unilateral urologic or renal affections can cause hypertension in man, as it can in rats, goats, rabbits and certain dogs, and if hypertension itself can cause renal arteriolar sclerosis, as it does in rats, and probably in dogs and man, an explanation is offered for the apparent discrepancy in results. Furthermore, if unilateral disease can give rise to hypertension, there is no reason to believe that bilateral disease will not; the experimental evidence is clear on this point.

Smith has clearly demonstrated that arterial hypertension is little more common when organic renal disease is present than when it is not. Bell intimates that he has come to the same conclusion. Analysis of his cases and those of Addison—1,047 patients with various types of organic renal disease—indicates that diastolic pressures of 90 or more were present in 52.9 per cent, which is little greater than the incidence for the general population. When patients probably or certainly exhibiting nitrogen retention were excluded, both with normal and with high blood pressures (745 cases), 43.4 per cent had hypertension.

(Fig. 2.) There are two differences, however, which appeared from these statistics. First, the age incidence of hypertension in renal diseases was lower, many severe cases occurring in childhood. Second, the course of the disease was usually shorter than that commonly seen in "essential" hypertension. To deny that renal diseases can be contributory to hypertension in man is to oppose the weight of voluminous experimental, clinical and pathologic experience. When definite experimental mechanisms have been evoked, and in part understood, the burden of proof must be placed on those who insist that these mechanisms have little if any clinical counterpart.

It is not surprising that the incidence of hypertension in urologic diseases is not much greater than in the general population; a situation such as this could be expected if both the predisposition or diathesis and the urologic disease were necessary to cause hypertension. Such seems to be suggested by

the fact that urologic and renal disease occur both with and without hypertension; two factors must therefore be at work. However, when both occur together the hypertension is apt to (1) be more severe, (2) begin earlier and (3) terminate earlier in renal failure. The clinical characteristics of a group of eighty cases of hypertension associated with previously unsuspected organic renal and urologic diseases were: (1) 64 per cent had a history of hypertension in their families; (2) the average duration of the disease from known onset to death was 6.9 years in males and 9.0 in females, or 7.4 years for the whole group; (3) the shortest duration was six months; (4) retinitis, that is, hemorrhages, exudates or papilledema, was present in 48 per cent; (5) a malignant course was common (21 per cent); (6) death from renal or renal and cardiac failure occurred in 68 per cent; (7) the diastolic pressure was often very high and appeared to be "fixed." These findings contrast with

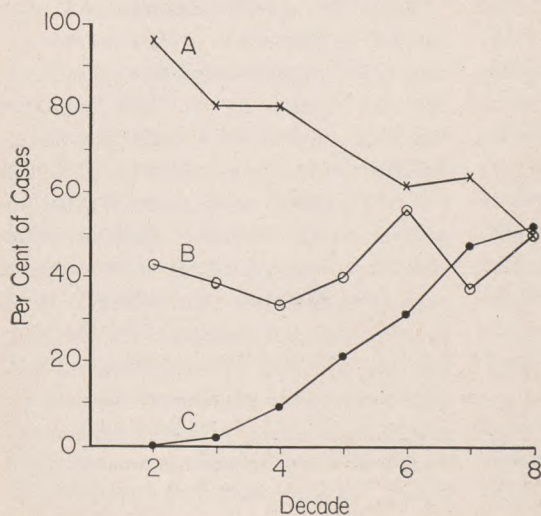


FIG. 2. Incidence of hypertension according to decade in patients with renal and urologic diseases compared with incidence in the general population. A, with renal excretory insufficiency (280 cases); B, without nitrogen retention (seventy-one cases); figures taken from Bell's and Addis' tables. C, incidence in general population (1,981 cases) averaged from Friedman et al. and Braasch et al., quoted by Smith. Although the series is small, the incidence in renal disease is approximately as high in the younger age groups as in the older, and much greater than that of the general population.

those of other observers analyzing groups of hypertensive patients without attempt at classification, a malignant course being rare (1.3 to 5 per cent), renal failure accounting for about 5 per cent of deaths and exudative retinitis being present in 1 to 19 per cent. On clinical grounds, therefore, it appears that when the constitutional or psychoneurogenic factor is present, and the patient develops organic renal disease, the result not infrequently may lead to severe hypertension with a tendency to develop renal insufficiency. When the former is absent, hypertension occurs possibly only with renal excretory insufficiency.

It is conceivable that moderate renal ischemia caused by organic processes may become severe through added neurogenic vasoconstrictor influences. Experiments in anesthetized dogs conclusively show that this is so although the point has not been demonstrated in man. Epinephrine reduces renal blood flow

in the dog only momentarily. However, when renal hemodynamics are previously altered by an adjustable clamp on the renal artery, the action of epinephrine on blood flow is greatly enhanced. Under these conditions one-hundredth the dose will cause renal ischemia lasting ten times as long. If the implications of these observations can be carried to man, summation of the effects of moderate renal ischemia and moderate neurogenic stimulation may affect renal blood flow profoundly. (Fig. 3.) Experiments done in this laboratory, however, have shown that the subcutaneous injection of 0.5 mg. of epinephrine reduced effective renal plasma flow, as measured by the clearance of para-amino hippurate, to a level of 71 per cent of the control values in three normotensive subjects and to a level of 66 per cent of control values in three hypertensive patients. The difference is not striking and the duration

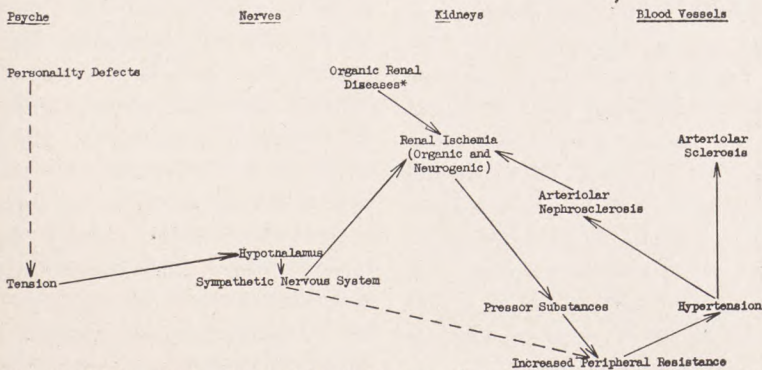


FIG. 3. Pathogenesis of renal hypertension. Sequence of events leading to nephrogenic hypertension without renal insufficiency. The influence of the psychoneurogenic factor may be of variable magnitude but is superimposed upon intrinsic changes in the circulation of the kidney produced by renal and urologic diseases. Humoral vasoconstrictor substances are formed as a result of the action of these two influences and the vicious circle is established. When arteriosclerosis obstructs partially the renal arteriolar tree, the sequence of events is similar except that less nephrosclerosis will result.

of action was similar (up to fifty minutes).

Neurogenic Hypertension. As opposed to the renal type, pure neurogenic hypertension has shown the following characteristics: (1) psychologic disturbances of more obvious nature; (2) fluctuating blood pressure even after years of apparently sustained hypertension, with a good response to sedation and sympatholytic or blocking agents (a lower nephrogenic "floor"); (3) little evidence of cardiac or renal damage; (4) the presence of the "hypertensive diencephalic syndrome," with reproduction of attacks by intradermal histamine; (5) evidences of vasomotor disturbances in symptoms and signs, such as palpitation, tachycardia, cold, clammy extremities especially during emotional stress; (6) a prolonged benign course; (7) slight diurnal variations in body temperature to febrile levels; (8) absence of accessory etiologic factors such as primary renal diseases, specific endocrine disturbances and little or no arteriosclerosis; (9) marked fluctuations of blood pressure occurring spontaneously and with respiration when measured by direct methods. The "histamine test" for producing the diencephalic blush and the response of the blood pressure to the intravenous injection of tetraethylammonium ion have been found most useful in differentiation.* In 62 per cent of a group

* The intradermal injection of histamine does not usually produce the blotchy, mottled blush so characteristic of neurogenic hypertension in subjects with other types. The "TEA floor," however, may occasionally be low in endocrine and arteriosclerotic types, even when the malignant stage has begun.

of forty-six such patients studied, headache was the most distressing symptom. Retinitis was rare. The condition occurred more often in females (84 per cent). While the diagnosis of neurogenic hypertension is made partly by exclusion, the aforementioned characteristics occur with less frequency and predominance in the renal type. The easy flushing of such patients when embarrassed or under the strain of an interview often suggests this condition.

Endocrine Hypertension. Of readily discernible nature is the so-called endocrine type of hypertension in which glandular disturbances are believed to predominate. While the adrenal cortex may be stimulated by activity of the sympathetic nervous system, on clinical grounds certain patients appear to be suffering from some form of intrinsic overactivity. Others have as yet undescribed endocrine diseases manifested by certain clinical findings.

Two definite groups can be described; one of which has been called pseudo-Cushing's syndrome, the other going by the cumbersome name of "nongoitrous thyrotoxic hypertension," both seen principally in women. The former syndrome is characterized by the presence of obesity of the trunk, thighs and upper arms, which usually develops rapidly, often after some physiologic endocrine disturbance (puberty, pregnancy, menopause) or an operative procedure (hysterectomy, oophorectomy); it is not uncommon after the third or fourth pregnancy. The central obesity is usually less well developed than that seen in Cushing's syndrome; the rapidity of onset is noteworthy, patients often

gaining 30 to 50 pounds within a year. Irregularities of menstruation are almost universal. The hypertension varies from mild to severe but is accompanied by little if any renal damage. Albuminuria is usually absent or of slight degree, but retinitis has been noticed in a number of cases, disappearing readily during treatment. Other signs and symptoms commonly but not universally encountered are: pale striae on thighs and sometimes arms, a "buffalo" type of hump on the back, tendency to easy bruising and ecchymoses, hirsutism, mottled cyanosis of extremities especially of legs, abnormal glucose metabolism with diabetic types of glucose tolerance determinations or mild diabetes which may regress spontaneously hyperchloremia, excessive sensitivity of the blood pressure to injections of desoxycorticosterone and the blood sugar to insulin, low excretion of 17-ketosteroids, cyclic disturbances of water metabolism with long periods of oliguria, and a dislike of salty foods. These patients lose weight with considerable difficulty on low caloric diets under controlled hospital conditions. The sodium and chloride concentrations of their sweat is always low. Of great clinical interest is the response of the hypertension and its manifestations to dietary restriction of salt (and calories). Dramatic response of the blood pressure, retinitis and symptoms may follow severe limitation of dietary salt; when salt is added to the diet, blood pressure rapidly rises. Clinical differentiation is therefore important, carrying with it therapeutic implications. The cardinal signs are

central obesity, menstrual disturbances and the low level of sodium and chloride in sweat. This last finding strongly suggests overactivity of the salt-retaining hormone of the adrenal cortex. Post-mortem findings have been scanty as the disease is benign, but in two cases the adrenal cortex was hyperplastic, a lesion not wholly unexpected.* (Fig. 4.)

"Non-goitrous thyrotoxic hypertension" is a condition described in the earlier literature characterized by severe hypertension often ending in the malignant phase, a continuously elevated basal metabolic rate which does not respond to thyroidectomy or iodine, the fairly frequent presence of the diencephalic syndrome, severe symptoms especially of headache and a relatively rapid course. Pathogenesis has not been defined.

Hypertension Associated with Arteriosclerosis. The theory that diastolic hypertension is dependent on generalized arteriosclerosis is the least proven of the various hypotheses. When gen-

* In a recent review Findley discussed the work of Heinbecker and others, and advanced the theory that hypertension is due to hypofunction of the neurohypophysis, which acts upon other endocrine organs leading eventually to a state of increased tissue sensitivity to the action of various pressor substances. As is evident, we do not agree that this hypothesis applies to the vast majority of cases of hypertension. It is possible, however, that some mechanism such as this may be concerned with the cases discussed here, and which probably account for 20 per cent of all hypertensive women. If the sodium and chloride content of sweat may be considered as an index of adrenal cortical hyperfunction, there is no evidence of hyperfunction in the usual case of renal or neurogenic hypertension. Proof of this hypothesis awaits pathologic demonstration of pituitary changes in hypertensive patients, and experimental production of hypertension in animals by reproducing these changes.

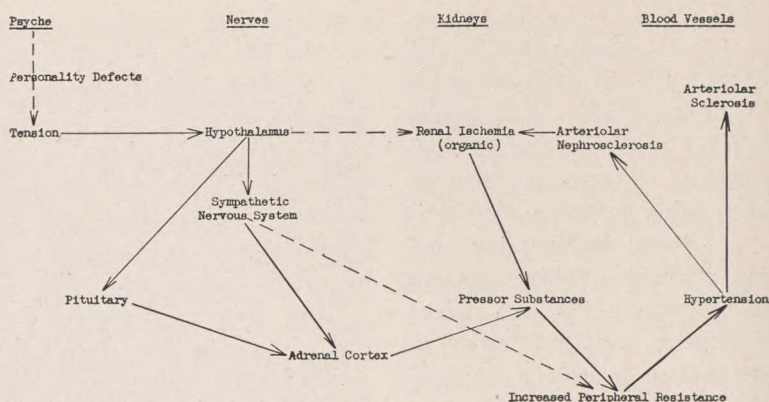


FIG. 4. Pathogenesis of endocrine hypertension. Possible sequence of events leading to endocrine hypertension. The role of the psychoneurogenic factor is not clearly established but may in some way influence the adrenal cortex to hyperactivity. Adrenal cortical hormones may themselves act as pressor substances but are probably not specific renal vasoconstrictors. The resultant hypertension leads to only moderate degrees of arteriolar nephrosclerosis and the institution of the vicious circle of renal ischemia \rightarrow pressor substances \rightarrow hypertension.

eralized arteriosclerosis occurs during later years, systolic pressure rises because of diminished vascular elasticity and diastolic pressure falls slightly. With this condition we are not concerned. The processes which cause diastolic hypertension may be construed theoretically as follows: When the psychoneurogenic factor is present to a slight degree (and a relatively slight degree must be postulated or neurogenic hypertension would have developed at an earlier age) little effect on blood pressure might be expected. If arteriosclerosis is located, however, in such a place as to interfere moderately with blood flow through the kidneys by obstruction of the orifices, lengths or intrarenal branches of the renal arteries, the combined action of both influences results in hypertension. Blackman's observations on the frequency of renal arterial narrowing from arteriosclerosis

are worthy of consideration. The aging process in the kidney probably does not contribute to this condition, for a gradual reduction in the number of nephrons, not a change in their internal anatomy, is most frequently seen in older normotensive individuals.

We are therefore in agreement with Goldblatt that "the possible contribution of this obliterative sclerosis of the large intrarenal arteries, and even of the main extra-renal artery, to the disturbance of intra-renal hemodynamics has been underestimated." When such a large supply of blood (500 to 600 cc. per minute) is going to a kidney through an artery (which never appears large enough) at a low resistance, it is obvious that minor stenosis of the artery would cause relatively profound peripheral changes. Experiments conducted with models and isolated limbs of dogs have demonstrated this to be hemody-

namically so.* By strict definition this type of case should be included in the group designated renal hypertension; they are separated because of their vascular aspect.

Clinically these cases can be differentiated by certain suggestive signs: the onset occurs later in life, usually in the fifth or sixth decades; the course is very variable, depending upon the degree and site of the major vascular changes, but the hypertension itself is benign and slowly progressive; retinitis is unusual; renal insufficiency is rare, death usually resulting from stroke or cardiac accident or failure; and severe symptoms referable to hypertension itself are unusual. The slowly progressive course can be explained by the nature of the lesions themselves and the relatively small part played by the neurogenic component. While comprising the most common type of hypertension, diagnosis can be made only by exclusion and by the presence of generalized arteriosclerosis.

* Either flow or pressure beyond a constriction is reduced, depending upon whether arteriolar constriction or dilatation occurs. Normally the kidney has a low resistance to flow. The resistance beyond a Goldblatt clamp in dogs' kidneys, however, is increased, indicating renal vasoconstriction. Therefore, the resistance offered by the arterial constriction is associated with high resistance in the arteriolar bed. Obviously, if renal arterial constriction causes renal arteriolar constriction, blood flow must fall to even lower levels than those resulting from the obstruction in the artery alone. As far as is known, the renal changes distal to a Goldblatt clamp are the following: increased vascular resistance, varying degrees of cortical hypoxia, acidification of the periphery of the cortex, possibly disturbances of electrolyte excretion and enzymatic constitution, and diminution of mean and possibly pulse pressure.

SECONDARY HYPERTENSION

Hypertension secondary to known pathologic conditions also can be caused by disturbances of four main systems: renal, nervous, endocrine and renal vascular. We do not intend to discuss these diseases at length. They fall into two general classes, those in which hypertension is associated and develops with renal excretory insufficiency, and those in which it does not. Among the latter renal failure sometimes supervenes. Elevated blood pressure accompanies renal insufficiency in more than two-thirds of cases (71.4 per cent of Bell's series) being more common in younger people. (Fig. 2.) The reason for the absence of hypertension in a large number of patients with renal insufficiency is not known. For example, it is absent in 38 per cent with polycystic disease, 50 per cent with urinary obstruction, and 30 per cent with glomerulonephritis. Its frequency suggests a causal relationship with the renal disturbance but this is not invariably so.

Careful analysis of cases of coarctation of the aorta indicates that true diastolic hypertension does not usually accompany the abnormality. In one series it was present in only five of twenty cases when blood pressure was measured directly in the femoral artery. It appeared to develop or become worse in one-third of another series and did not completely disappear after the aortic defect was repaired. These observations suggest that some patients have coarctation of the aorta alone (with often a systolic elevation above the constriction) while others have coarctation plus hypertension (with a dia-

stolic elevation below the constriction). Although the series is small, the possibility exists that chance occurrence of the hypertensive diathesis and coarctation is responsible for these discrepancies. The degree of coarctation did not correlate in these few subjects with the degree of true hypertension.

A similar argument may be valid for the explanation of the hypertension secondary to other diseases, notably renal. The large proportion of cases without hypertension but with renal diseases believed to be associated with it suggest that two factors again may be operating, the combination of which results in elevation of blood pressure. In some conditions both factors may be renal, as suggested by Bell, but in most the evidence indicates that the hypertensive diathesis, mild, moderate or severe, acts in conjunction with a renal factor. Any other explanation is inconsistent with the facts. When Cushing's syndrome and other endocrine diseases are examined from the same point of view, one is forced to the same conclusion, for hypertension is absent in a fair proportion. Data are not available to suggest whether hypertension is the result of two interacting influences, neurogenic and adrenal. The occasional absence of hypertension in this condition can also be explained by the interrelationships of the actions of the various adrenal cortical hormones which may be present in excessive amounts.

Pheochromocytomas produce intermittent hypertension which can lead to sustained hypertension. As far as is known, epinephrine and nor-adrenalin are the causative agents, and the condi-

tion is curable by surgery. And yet the same pathologic findings may be present in the kidneys of patients with long-standing pheochromocytoma as in those with long-standing neurogenic hypertension. Pathogenesis in this case follows a similar pattern. Although the psychogenic factor may be absent, the neurogenic one as exemplified by its chemical effector substances is strong.

Chronic glomerulonephritis without nitrogen retention may exist with or without hypertension. When hypertension is present it may be severe and in certain stages indistinguishable from that called "essential." Only at the beginning and end of the disease can a differential diagnosis be made, often only by necropsy. We can postulate therefore the interaction of the two factors to produce hypertension, the psychoneurogenic and the renal. When the former is absent, hypertension in chronic nephritis occurs with renal excretory insufficiency, possibly the result of retention of some pressor substance. The same may be true for polycystic disease of the kidneys in which hypertension is frequent but not universal. The pressor substance is probably not related to guanidine, phenol or creatinine.

COMMENT

When the four types of hypertension are considered as separate conditions having in common only two components, the psychoneurogenic factor in degrees from mild to severe and increased peripheral resistance, differentiation is not too difficult. It is important from both prognostic and therapeutic viewpoints. The analysis of any

procedure designed to test or alter hemodynamics must be made in the light of the conception that "essential" hypertension is not a single disease, just as diabetes is not. Much of the confusion in the literature on pathology, physiology and response to therapeutic measures is probably caused by the tendency to regard hypertension as having a single pathogenesis. The present disagreement as to the relative importance of either renal, neurogenic or adrenal pressor mechanisms is the result of this tendency. While there is general agreement that blood pressure can be elevated by many mechanisms, one is often considered implicated to the exclusion of others, in spite of the fact that hypertension is only a sign, analogous to fever, tachycardia, albuminuria or hyperglycemia.

Therapy, as in all conditions, must be directed against cause. The common factor appears to be psychoneurogenic. The relative weight of the factor varies from case to case except in neurogenic hypertension, and therefore the most successful psychotherapy can be expected to produce variable results. Psychotherapy is meant to include all measures which tend to relieve anxiety and tension and cause the patient to turn his welfare and his worries over to someone else. Religious or quasi-religious faiths, limitation of activities, strict regimens, dependency on non-specific drugs, severe operative procedures, prolonged hospitalization and frequent clinic or office visits come under this category and are of effect in some cases. Any therapeutic procedure vigorously pursued by both physician and patient

will show results in more moderate cases. Psychotherapy has sometimes altered the course of hypertension when used in the earliest stages. Psychotherapy can attempt to re-educate the individual so that he will react to his conflicts by overt action or by insight and logic and not resolve them by repression, emotional tension and subsequent discharge via his hypothalamus and his sympathetic nervous system. The deficiencies of personality, however, are so deeply ingrained and fundamental to the individual that intense psychotherapy of the wrong kind may make the situation worse.

The neurogenic factor, the effector mechanism of the psychic discharges, can be altered in two ways other than by changing its psychic cause. The newer sympatholytic agents are now used only for brief experiments to evaluate the degree of sympathetic activity. Their action often calls out reactions attributable to parasympathetic activity. So far they have been relatively ineffective for continued use. A drug specific for the neurogenic factor must be taken continuously, produce a sustained "chemical sympathectomy," should probably block the action of nor-epinephrine, and preferably should be active by mouth. When such a drug is developed, it will be useful in cases having predominantly neurogenic hypertension without severe arteriolar disease of the kidneys. Evidence for the reversibility of advanced arteriolar disease by sympathectomy is not available.

The other method is by extensive sympathectomy which has proven valu-

able in only a certain proportion of cases 10 to 20 per cent), although it does seem to alter the malignant phase into one more benign. The apparent failures of this method are explicable on four grounds: (1) If arteriolar disease is severe, removal of the neurogenic component will not result in the abolition of renal ischemia. (2) The neurogenic component is very variable from patient to patient. (3) Removal of nerves is incomplete. (4) Sympathetic nerves regenerate. We have seen one such example: a twenty-eight year old woman whose lumbar sympathetic chain and splanchnic nerves were removed in 1946, with temporary lowering of blood pressure. Two years later lumbodorsal sympathectomy was performed; the splanchnic nerves and lumbar chain had completely regenerated, even to the regrowth of distorted ganglia. The second procedure resulted in a normal blood pressure for at least two years. The inadequacy of limited denervation of the kidneys and splanchnic bed has been stressed by Smithwick. The innervation of splanchnic nerves and kidneys is very variable.

The other factor which must be controlled is humoral. Five approaches are possible: 1) destruction of the pressor material by enhancement of natural destructive agents or by artificial antagonists; (2) inhibition of its formation by enhancement of natural inhibitory processes; (3) "flooding" a system with a non-toxic competitive agent;*

* It is interesting, in this respect, that the pressor effects of iso-amyl amine in hypertensive rats is about one-half as strong as in normal rats; this pressor amine, which is relatively weak, usually lowers blood pressure

(4) increasing the concentration of a naturally occurring antagonist; (5) increasing renal blood flow by non-specific methods, thereby lessening the production of pressor substances. Identification of the pressor material is necessary before intelligent investigation can proceed along these lines. The problem is biochemical. Unless it turns out that the chemical mediators of both neurogenic and humoral vasoconstriction are identical, it is probable that both approaches will be necessary to control hypertension. There is already evidence that some patients do better when several therapeutic methods are applied together.

Advances along these lines are encouraging enough at present to justify considerable optimism for the near future. When the solution finally appears, it may be a relatively simple one. There are no grounds for the pessimistic attitude expressed by a number of workers in this field.

SUMMARY

Experimental and clinical observations on arterial hypertension are consistent with the theory that: (1) Repressed psychic disturbances of a more or less specific nature lead to increased activity of the sympathetic nervous system; (2) sympathetic stimulation may raise blood pressure acutely but also produces renal ischemia and stimulates

in hypertensive human beings, although raising it in normal subjects. Furthermore, thiocyanate ion has been found to act on renal tissue *in vivo* by inhibiting decarboxylation of those amino acids, the amines of which are pressor. Conceivably, the hypotensive action of this drug in man could be due to its ability to inhibit the formation of pressor amines from amino acids.

the adrenal cortex to activity; (3) renal ischemia leads to the production of pressor substances and therefore hypertension; (4) hypertension itself causes arteriolar sclerosis, especially in the kidneys, resulting in more renal ischemia; (5) adrenal cortical activity can lead by itself to hypertension. When organic renal or urologic disease is also present, the hypertension may be more severe. When the predominant influence arises in the adrenal cortex, the disease presents different clinical manifesta-

tions. When arteriosclerosis causes renal ischemia, the type of disease is also different. Variations in the degree of the causative factors, i. e., psychic and neurogenic, along with the presence and severity or absence of contributory factors, i. e., renal and endocrine diseases and arteriosclerosis, account for the wide variation in the course of different patients, and in the relative efficacy or inefficacy of various methods of treatment.

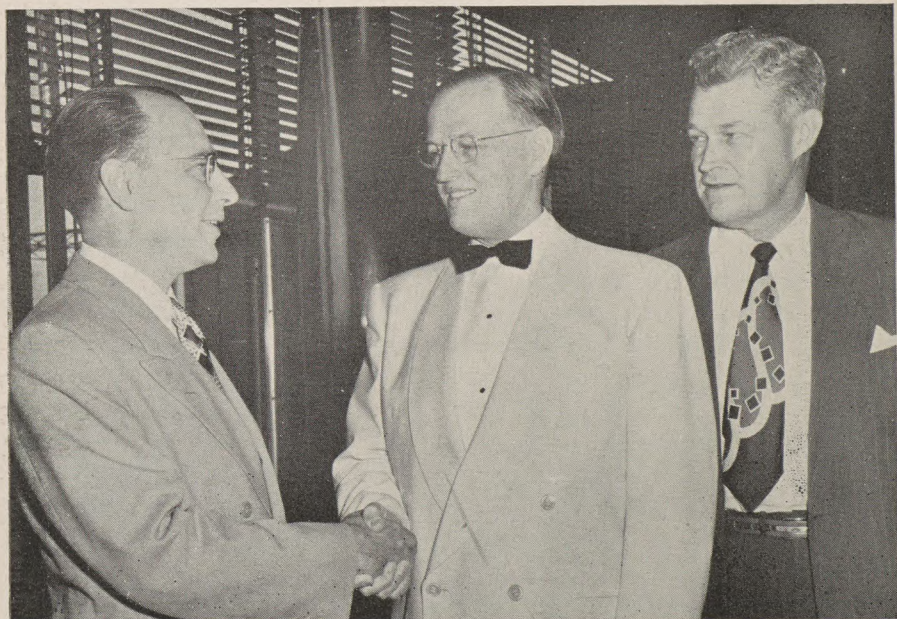
275 MEDICAL ALUMNI ATTEND REUNION DINNER; A. NORMAN ARNESON, '28, ELECTED PRESIDENT

Some 275 alumni of the School of Medicine, including 95 members of the 1951 class, attended the annual alumni dinner to hear Dr. Walter H. Judd, representative in Congress from Minnesota, speak on "What Are We to Believe About the Far East?" The dinner and preceding cocktail party were held at the Starlight Roof, Chase Hotel on Friday evening, June 1, with Dr. James Barrett Brown '23 presiding.

Among the guests present were — Chancellor Arthur H. Compton, Vice-Chancellor Charles Belknap, Harry Brookings Wallace, president of the Washington University corporation; Robert A. Moore, dean of the Medical

School; Dr. J. William Thompson, president-elect of the Missouri State Medical Association; Dr. LeRoy Sante, president of the St. Louis Medical Society; Elizabeth Garrett, president of the Nurses' Alumnae; Dr. Harry Gordon Fisher, president of the Dental Alumni; Franklin Ferriss, president of the Law Alumni, the heads of the various medical school departments, and other members of the university corporation and administration.

Twenty-two members of the class of 1926 returned for the silver anniversary of their graduation from the School of Medicine, which was marked by a separate pre-dinner cocktail party.



Talking with Dr. Judd (center) are Virgil Fish '30 and John Tidwell, 30.

Officers for the coming year were elected during a short business session. Dr. A. Norman Arneson '28 is president and will be aided by Drs. Wendell G. Scott '32, first vice-president; George W. Ittner '37, second vice-president; and Guy N. Magness '28, secretary-treasurer.

Four new members were elected to the executive committee of the Alumni Association and will serve until 1954. They are Drs. Robert B. Bassett '31, Vilray P. Blair, Jr. '39, Bruce D. Kenamore '35, and John F. Patton '28, all of St. Louis.

First Graduating Class Holds Golden Anniversary Reunion

Ten members of the class of 1901, the first class to be graduated from the Washington University School of Medi-

cine, together with two of their former teachers held their fiftieth anniversary celebration May 31 at the Chase Hotel.

The veteran physicians exchanged advice on how to keep healthy and reminisced about their med school days. They represented the majority of the 16 members of the class, which originally numbered 56, who are still living. Six of the ten are still practicing.

The two former teachers who celebrated with the class were Dr. Robert J. Terry, professor emeritus of anatomy and Dr. Robert E. Schlueter, both of the class of 1895.

Members of the class who attended the dinner were: Dr. Julius C. Bohn, Tacoma, Washington; Dr. Pierre I. Chandeysson, St. Louis; Dr. Wm. Carver Forder, St. Louis; Dr. Irwin J. Har-



The first graduating class at the School of Medicine at its fiftieth anniversary dinner at the Chase Hotel. Seated, left to right: John L. Lionberger, Julius Bohn, Robert Schlueter, R. J. Terry, Dean Robert A. Moore and Walter Kirchner. Standing, left to right, are: Wendel Scott, Irwin J. Harris, W. C. Forder, Melvin K. Wylder, Wm. G. Krenning, Raymond Holben, Pierre Chandeysson and Garrett Hogg.

ris, Clayton, Missouri; Dr. Garrett Hogg, Springfield, Missouri; Dr. Raymond E. Holben, Jacksonville, Illinois; Dr. W. C. G. Kirchner, St. Louis; Dr.

William G. Krenning, St. Louis; Dr. J. R. Lionberger, St. Louis and Dr. Mel-drum K. Wylder, Albuquerque, New Mexico.

DR. CARL A. MOYER APPOINTED TO SUCCEED DR. EVARTS A. GRAHAM

Dr. Carl Alfred Moyer, professor of surgery and dean of the Southwestern Medical College in Dallas, Texas, has been appointed Bixby Professor of Surgery and head of the department of surgery at the School of Medicine, succeeding Dr. Evarts A. Graham.

Dr. Graham will become professor emeritus of surgery and will remain in St. Louis, connected with Washington University and the affiliated hospitals in the Medical Center. Dr. Graham recently received an honorary degree during ceremonies celebrating the 500th anniversary of the University of Glasgow.

Chancellor Arthur H. Compton expressed appreciation of the long and distinguished service of Dr. Graham to Washington University. "We are all proud of what Dr. Graham has created here in St. Louis and look forward to continued progress under Dr. Moyer. It is a great satisfaction to us that Dr. Graham will remain in St. Louis as an active member of the staff."

Dr. Moyer, who is 42 years old, is well-known for his scientific work on anesthesia, respiration, and the physiology of electrolyte and fluid balance. He plans to assume his duties at Washington University in October.

Born in Baraga, Michigan, Dr. Moyer holds the A.B. degree from Northern

Michigan State Teacher's College, and his M.S. and M.D. degrees from the University of Michigan. He took additional work at Harvard University in 1940-41 as a National Research fellow.

He started his professional career as a teaching assistant at the Teacher's College while in school there, then was assistant in the departments of physiology, anatomy and pharmacology at the University of Michigan while earning his advanced degrees.

In 1941 he was appointed instructor in surgery at Michigan, and was promoted to assistant professor in 1943. From 1944 to 1946, he was surgical director of Wayne County General Hospital, Detroit.

Since 1946, Dr. Moyer has been professor of surgery at Southwestern Medical College in Dallas. He became dean there about two years ago.

Dr. Moyer holds fellowships in the American Surgical Association, American Physiological Society, American Society for Clinical Investigation, Southern Society for Clinical Investigation, and the Texas Surgical Society.

Dr. Evarts Graham first came to the Washington University staff 32 years ago, in 1919, when he was appointed professor of surgery following a high reputation gained in the Army Medical Corps during World War I. Since that

time Dr. Graham has become one of the world's leading figures in the field of surgery, and has been called the "father of modern chest surgery," by his associates.

He is perhaps best known for performing in 1933 the first operation for successful removal of an entire lung in one stage, for treatment of cancer of the lung. Among his many contributions to surgery are the development of a test for diagnosis of gall bladder disease, on which he worked with Drs. Sherwood Moore, Glover Copher, and Warren Cole.

Dr. Graham was chiefly responsible for the founding of the American Board of Surgery, which has become the accepted standard in this country of competence in surgery.

Many honors and awards have been given Dr. Graham in recognition of his outstanding work. Perhaps his greatest honor is the award of the 1942 Lister Medal, presented by the Royal College of Surgeons in England. Only one other American has ever received the Lister Medal.

Dr. Graham was elected to the National Academy of Science in 1941, and has served as president of the American College of Surgeons and the American Surgical Association. He received the St. Louis Award in 1942, and has been honored with medals from numerous medical and scientific associations.

W. U. to Exchange Medical Teaching Personnel with Thailand

With the signing of a contract between Washington University and the Economic Cooperation Administration,

the School of Medicine became the first school in the country to establish a program of teaching assistance with institutions in another country under the technical and economic assistance program administered by the ECA.

The contract will put into action a cooperative teaching program between the Medical School here and the two medical schools in Bangkok, Thailand, or Siam. Staff members from W. U. are to establish residence in Bangkok and teach in the medical schools there; a number of Thailand doctors will in turn come to the Medical Center here for advanced training and study. The exchange includes nurses, medical technicians and nurse anesthetists as well as physicians.

Chancellor Arthur H. Compton terms this program a "rare opportunity for Washington University to help in holding the line for the Western World by helping the people of Thailand raise their standard of living through improved health care and education." He stated that it "represents a new aspect of United States foreign policy in the effort to contain communism by strengthening the free nations of the world."

The School of Medicine will, in effect, "adopt" the two medical schools of Thailand, and will set the pattern for other U. S. medical schools to do the same sort of work with other nations which request such help.

Dr. Robert A. Moore, Dean, said in this connection, "The School of Medicine is proud that it has been given the opportunity of leading the way for other medical schools in the United

States to 'adopt' medical schools in other strategic countries."

The School of Medicine is receiving a grant-in-aid for a two-year period to support this cooperative teaching program. Faculty members who are to teach in Bangkok will come from both the pre-clinical and clinical medical departments and the School of Nursing. Eight staff members are planning to go to Bangkok for one year, and upon their return to this country, others will be sent in their places.

For almost twenty years, Washington University has been in close professional relationship with medical men in Thailand. This has been done mainly by Thailand doctors coming to St. Louis for part of their postgraduate training. The first of these was Dr. Seng Tongprasroeth, who came to study surgery under Dr. Evarts A. Graham in 1931-32. Since then, many Thailand doctors have followed Dr. Seng here.

Ten Staff Members to Participate in Thailand Exchange

The following medical school staff members have been recommended for appointments to participate in the Bangkok Medical Exchange Program from Washington University, subject to clearance by the Economic Cooperation Administration:

Dr. Ben Eiseman, instructor in surgery and assistant dean, is director of the program.

Dr. C. Read Boles, instructor in clinical pediatrics, to spend three months in Bangkok.

Dr. Arthur Knudson, visiting professor of biochemistry, from Union Uni-

versity, Albany Medical College, Albany, N. Y., for 12 months.

Miss Jean McCormick, assistant in nursing, for 12 months.

Miss Virginia Minnich, research associate in medicine, for 12 months.

Mrs. Honora Camden Obourn, assistant professor of nursing, for 12 months.

Miss Bonnie D. Perry, instructor in anesthesia, from Ellis Fischel State Cancer Hospital, Columbia, Mo., for 12 months.

Dr. Morris Scherago, visiting professor of bacteriology, from University of Kentucky, in Lexington, for 12 months.

Miss Barbara Sweikert, technician in pathology, for 12 months.

Dr. Frank Vellios, instructor in pathology, from Columbia University College of Physicians and Surgeons, in New York, for 12 months.

Thailand Presents Gifts to Chancellor, Six Med School Faculty Members

Two nielloware cigarette boxes were presented to Chancellor Arthur H. Compton of Washington University on behalf of the University of Medical Sciences, Bangkok, Thailand, and the Thailand Minister of Public Health.

The gifts, in appreciation of help given by the university in setting up a teaching assistance program at two Thailand medical schools, were presented to Dr. Compton by Dr. Ben Eiseman, assistant dean of the Washington University School of Medicine. Dr. Eiseman also relayed gifts to five Medical School faculty members.

Dr. Eiseman returned recently from Thailand, where he taught surgery for six weeks and directed the establishment of the teaching assistance program.

Medical school staff members who received gifts from Thailand are: Dr. Edmund V. Cowdry, Dr. Edward W. Dempsey, Dr. Eiseman, Dr. Evarts A. Graham (recently retired professor of surgery), Dr. Robert A. Moore and Dr. Harvey Lester White.

K. U. Medical School Dean Speaker at Award Program

Dr. Franklin Murphy, dean of the University of Kansas Medical School, addressed the graduating seniors of the School of Medicine during a special program on June 5. His topic was "The Public Responsibilities of the Modern Physician."

Dr. Murphy stressed the fact that physicians must help labor organizations, industry and farm consumer groups in working out realistic health insurance programs. He said the wholehearted acceptance of health protection insurance must be recognized and should be followed by a plan worked out, preferably, on a voluntary basis.

The speaker added "It is the responsibility of the physician to vigorously participate in affairs of organized medicine. The public has given, by law and tradition, certain privileges to our profession, but these privileges are balanced by responsibility. If we do not take care of the responsibility, we may well lose our privileges.

The Alpha Omega Alpha prize for the highest average for four years was

given to Margaret Ann Hunt of DeWitt, Arkansas at the award ceremony. Miss Hunt is interning in pediatrics at the University of Minnesota Hospital in Minneapolis.

Other awards given included—the Medical Fund Society prize for excellence in internal medicine, to John H. Knowles, St. Louis; and the George F. Gill Prize for superior scholarship in pediatrics to Alexis F. Hartmann, Jr., St. Louis, whose father is head of the medical school's department of pediatrics.

Four St. Louis Boys Win Jackson Johnson Scholarships

Four St. Louis boys have been awarded Jackson Johnson Scholarships to Washington University School of Medicine for the school term beginning next September. Two of the boys were graduated from Washington University with bachelor's degrees this year, and two have completed four years of work at Harvard University.

The scholarship winners are: Jules A. Kernen, 21, son of Mr. and Mrs. Jules H. Kernen, 5065 Oleatha ave.; Wolff Kirsch, 20, son of Mr. and Mrs. David Kirsch, 415 Midvale ave., University City; James N. McClure, Jr., 20, son of Mr. and Mrs. J. N. McClure, 2 Sappington Spur, Kirkwood; and Donald H. Tilson, Jr., 20, son of Mr. and Mrs. D. H. Tilson, 5334 Waterman ave.

Kernen and Tilson both were graduated from Harvard University with bachelor of arts degrees this month. Kernen was graduated from Southwest High School in 1946, and Tilson, from

St. Louis Country Day School in 1947.

Kirsch and McClure both received bachelor of arts degrees from Washington University on June 6. Kirsch is a graduate of University City High School (1948), and McClure of Kirkwood High School (1948).

Jackson Johnson scholarships provide up to \$1200 yearly, where needed, out of a \$250,000 fund bequeathed to the School of Medicine in 1930 under the will of the late Jackson Johnson, former president of International Shoe Company.

The fund was established to aid worthy and promising students in acquiring and completing their medical educations, awarding scholarships in recognition of exceptional ability and achievement in pre-medical work.

Recipients are chosen on a regional basis whereby the United States and its territories are divided into seven regions, with scholarships being awarded to students of merit attending colleges or universities in those areas. It is extremely unusual that as many as four recipients of the scholarships are St. Louisans, and this comes about because Kernan and Tilson were attending Harvard and were considered from the New England region.

Other men who are receiving Jackson Johnson Scholarships are:

Willard C. Schwartz, Jr., 21, son of Dr. and Mrs. Willard C. Schwartz of Manhattan, Kans., and graduate of Kansas State College, 1951.

Edward Lewin, 22, son of Mr. and Mrs. Morton Lewin of Denver, Colo., and a graduate of the University of Colorado, 1951.

David Murray, 21, son of Mr. and Mrs. William G. Murray of Ames, Iowa, and a graduate of Cornell University, 1951.

Herbert Spady, 22, son of Mr. and Mrs. George P. Spady, 4521 S. E. 47th Ave., Portland, Ore., graduate of U. of Oregon, 1951.

Frederick T. Kraus, 21, son of Lt. Col. and Mrs. William A. Kraus of Richmond, Va., and a graduate of the College of William and Mary, 1951.

Charles W. Markham, 21, son of Mr. and Mrs. Daniel R. Markham of Greenville, Miss., and a graduate of Millsaps College, Jackson, Miss., this year.

Anatomist Awarded Honorary Degree at Commencement Here

Dr. George B. Wislocki, professor and head of the department of anatomy, at the Harvard Medical School, was awarded an honorary doctor of science degree from Washington University at this year's commencement exercises.

The citation for Dr. Wislocki, which was presented by Dean Robert A. Moore, said he brought "distinction to himself and to his alma mater (Washington University A.B. '12) by outstanding contributions in his chosen field of anatomy." It also said he had added significantly to scientific knowledge in the fields of embryology and endocrinology.

Dr. Wislocki led a seminar at the School of Medicine on June 5 in the department of anatomy. The subject was "The Cytology and Histochemistry of Possible Neurosecretory Regions of the Brain."

Diplomats Among Those Expected to Attend "Old-Age" Congress Here

Dr. Antonio M. Araujo, Venezuelan Ambassador, and Dr. Dario Contreras, Minister Counselor of the Dominican Republic, will be among the more than 1,000 representatives expected to attend the Second International Gerontological Congress, which will meet in St. Louis at the Hotel Jefferson, September 9 through 14.

Forty-two countries will be represented at the Congress to which Washington University will act as official host. A representative of the United Nations will also be present. Dr. Edmund V. Cowdry, research professor of anatomy, is president of the Congress and several other staff members of the Medical School are serving as chairmen of various committees.

The objectives of the Congress are to promote research on aging and on the social consequences of an aging popu-

lation, and to stimulate utilization of existing knowledge in order to provide greater opportunity for older people. The Congress is a continuation of the First International Gerontological Congress held at Liege, Belgium in July 1950, and of the first national Conference on Aging called by the Federal Security Agency in August 1950.

Activities of the Congress will start on Sunday, September 9, with the setting up of exhibits and a radio broadcast of the University of Chicago Round Table from St. Louis. The exhibits will include 20 to 30 of a scientific nature and 60 in the commercial field.

The Congress is being sponsored by the International Association of Gerontological Societies, the Gerontological Society, Inc., and the American Geriatrics Society. Four sections have been established for discussion of the phases of old age study. These are: biology and medicine; sociology, psychology, education and religion; economics and welfare; and medical services, hygiene and housing.

FORMER MED. SCHOOL DEAN DIES IN LOS ANGELES

Dr. George Dock, dean of the Washington University Medical School from 1910 to 1922 and also professor of medicine, died May 30 at the age of 91 in Los Angeles, Cal. where he was the eldest member of the Huntington Memorial Hospital staff.

Dr. Dock was born in Hopewell, Pa., and was graduated in medicine at the University of Pennsylvania in 1884. He taught there and at Texas Medical College and Hospital, the University of Michigan, Tulane and the University of Southern California as well as here. He held honorary degrees from Harvard and SC, was presented the Gold Headed Cane in Great Britain, an award rarely made outside the British Empire, and in

1944 received the Distinguished Service Award from the American Medical Association.

He served as a surgeon in the Spanish-American War and in World War I, and from 1916 to 1917 was president of the Association of American Physicians. He was a member of the American Medical Association, Nu Sigma Nu, Chi Phi, social fraternity, the University Club in Pasadena, Cal. and the Los Angeles Athletic Club.

Dr. Dock leaves his widow, Miriam Gould Dock, Altadena, Cal.; two sons, George Dock Jr. and Dr. William Dock of New York; three sisters and three grandchildren.

Report of the Dormitory Fund Campaign

The Dormitory fund is slowly but steadily growing as additional alumni send in their contributions.

The total amount, including pledges, is now \$67,024.10, and our goal of \$100,000 from the medical school alumni is within reach.

The following list shows the names of the 749 alumni who have contributed. If you have not as yet made a contribution, please send in your check now to the alumni office. This project deserves your support.

Samuel B. Grant, Chairman

1950—Living Graduates, 85

Elmer B. Brown, Jr., New York, N. Y.
Edward T. Emura, St. Louis
Joseph D. O'Keefe, Nashville, Tenn.
Robert I. Pfeffer, St. Louis
Joseph V. Sharrotta, East Cleveland, Ohio
Richard L. Swarm, St. Louis

1949—Living Graduates, 96

Roger Bumgarner, Kansas City, Kans.
Eugene W. Pearce, Washington, D. C.
Russell D. Sheldon, Kansas City, Mo.
George S. Woodard, Jr., Washington, D. C.

1948—Living Graduates, 89

Virgil R. Bleisch, Boston, Mass.
Walter A. Fernau, Jr., Cincinnati, O.
David A. Guterman, Elgin, Ill.
Hugh R. Harting, St. Louis
Richard F. Huck, Jr., St. Louis
Robert Kiyasu, St. Louis, Mo.
Juro L. Shintani, Perry Point, Md.
Thomas N. Stern, Memphis, Tenn.

1947—Living Graduates, 98

Charles G. Clay, Rantoul, Ill.
Marvin Cornblath, St. Louis
William C. Dunckel, Charlottesville, Va.
Helen Hofsommer Glaser, St. Louis
Arnold Namrow, Newport, R. I.

Burnet W. Peden, St. Louis
Virginia H. Peden, St. Louis

1946—Living Graduates, 86

R. Robert Bates, Tucson, Ariz.
Drennan Bailey, Clayton, Mo.
R. Robert Bates, Tucson, Ariz.
Gladden V. Elliott, Richmond Heights, Mo.
Lawrence W. O'Neal, Webster Groves, Mo.
James O. Owen, Jr., Skiatook, Okla.
Theodore J. H. Smith, Temple, Tex.
Robert S. Spain, McKinney, Tex.
Frank Vellios, St. Louis
Leonard J. Wiedershire, Aurora, Colo.

1945—Living Graduates, 105

John T. Farrar, Boston, Mass.
Jay O. Gibson, French Camp, Calif.
Samuel B. Guze, St. Louis
John T. Johnstone, Jr., St. Louis
Donald E. Kilker, St. Louis
Louis O. Lambiotte, Salt Lake City, U.
Ceylon S. Lewis, Jr., Salt Lake City
Roscoe Maxwell, Punta Gorda, Fla.
George W. Prothro, Clovis, N. Mex.
Eugene T. Taylor, Mocksville, N. C.
John W. Ubben, Staunton, Ill.
Gary B. Wood, St. Louis
Betty Ben Geren, Boston, Mass.
Charles Wolfson, Amarillo, Tex.

1944—Living Graduates, 95

Guy D. Callaway, Jr., Seattle, Wash.
Albert B. Eisenstein, St. Louis
J. K. Frost, Centralia, Ill.
Robert D. Lange, Kirkwood, Mo.
Ervan Levine, Vandalia, Mo.
Clayton H. Manry, Syracuse, N. Y.
Francis E. Pennington, St. Louis
H. H. Perman, Forest City, Ia.
Richard S. Roberts, Ottawa, Kans.
John J. Rupp, Tucson, Ariz.
David E. Smith, St. Louis
Roy A. Walther, Jr., Overland, Mo.
Virgil Loeb, Jr., St. Louis
Marvin T. Pursell, Dinuba, Calif.
Louis Weisfuse, Brooklyn, N. Y.

1943—(Dec.)—Living Graduates, 113

John F. Blinn, Jr., Stockton, Calif.
J. P. Myles Black, Olive View, Calif.
C. Read Boles, St. Louis

- William P. Callahan, Wichita, Kan.
 Joseph B. Clay, Van Nuys, Calif.
 Terrell Covington, Jr., McKinney, Tex.
 Edward W. Czebrinski, St. Louis
 Del Roy R. Davis, Kingwood, W. Va.
 Harold Grant, McKinney, Tex.
 Mary Jordan, Ridley Park, Pa.
 James L. Petry, Port Arthur, Tex.
 Walter J. Kennedy, Yakima, Wash.
 Edward H. Kowert, St. Louis
 Elaine K. Lince, Pasadena, Calif.
 Torrence A. Makley, Jr., Columbus, O.
 Walter A. Rohfling, Fresno, Calif.
 Ernest T. Rouse, St. Louis, Mo.
 Ernest Schwartz, San Francisco, Calif.
 Burton Shatz, St. Louis
 Donald E. Smith, Salt Lake City, U.
 Tom G. Stauffer, Scarsdale, N. Y.
 Herbert C. Wiegand, St. Louis
 Frances C. Wilson, Tampa, Fla.
 Carl T. Woolsey, St. Louis
 Alfred H. Sudholt, St. Louis
 Harold E. Walters, St. Louis
- 1943—(March)—Living Graduates, 105
 DeWayne C. Anderson, Stanhope, Ia.
 Grace E. Bergner, St. Louis
 Raymond M. Charnas, St. Louis
 Gerald J. Conlin, Denver, Colo.
 David Feldman, St. Louis
 Harlan I. Firminger, Bethesda, Md.
 Melvin L. Goldman, St. Louis
 H. Clagett Harding, Portland, Ore.
 Stanley S. Kahn, Birmingham, Ala.
 F. C. Lawrence, Bartlesville, Okla.
 Albert N. Lemoine, Jr., Mission, Kans.
 Ira W. Liebner, Brooklyn, N. Y.
 Eichichi Masunaga, T. H.
 Roberts B. Pappenfort, New York, N. Y.
 Ernest S. Rogers, San Francisco, Calif.
 Daniel G. Santer, Milwaukee, Wis.
 Carvel T. Shaw, Hermann, Mo.
 David A. Stadtner, Stockton, Calif.
 H. A. Uhlemeyer, Jr., St. Louis
- 1942—Living Graduates, 92
 William M. Anderson, Richmond, Va.
 Ewald W. Busse, Denver, Colo.
 Hiraku Ishida, Los Angeles, Calif.
 Frances M. Love, Richland, Wash.
 French H. McCain, Birmingham, Mich.
- C. Barber Mueller, St. Louis
 William G. Reese, Perry Point, Md.
 Herman Rice, Temple, Tex.
 Frank O. Shobe, St. Louis
 Souther F. Tompkins, Oklahoma City, Okla.
 George L. Watkins, Farmington, Mo.
- 1941—Living Graduates, 93
 Cecil H. Blackburn, Selma, Ala.
 Bruce L. Canaga, Jr., Washington, D. C.
 Robert J. Cook, St. Louis
 Jane A. Erganian, Glen Rock, N. J.
 Charles E. Fildes, Poplar Bluff, Mo.
 B. W. Finkel, St. Louis
 Peter O. Fleming, Topeka, Kan.
 Anne T. Goetsch, Berkeley, Calif.
 Samuel W. Gollub, St. Louis
 Leon Kahn, Beverly Hills, Calif.
 Geo. Bruce Lemmon, Springfield, Mo.
 J. I. Moreland, Salem, Ore.
 Jane Matthews Day, Montgomery, Ala.
 Harold E. McCann, E. St. Louis
 V. A. Mueller, Wichita, Kan.
 C. A. Nielsen, Seattle, Wash.
 Joseph W. Noah, St. Louis
 Carol H. Rehm, Los Angeles, Calif.
 Allan M. Rossen, Los Angeles, Calif.
 William L. Topp, Seattle, Wash.
 Bernice A. Torin, St. Louis
 Mitchell Yanow, Clayton, Mo.
 Howard S. J. Walker, Jr., Mobile, Ala.
- 1940—Living Graduates, 92
 Robert R. Anschuetz, Alton, Ill.
 Donald S. Bottom, Alton, Ill.
 Seymour Brown, St. Louis
 Russell J. Crider, St. Charles, Mo.
 Roland R. Cross, Hines, Ill.
 A. T. Esslinger, St. Louis
 L. R. Fernandez, Laupahoehoe, T. H.
 James M. Foerster, Wausau, Wis.
 Robert L. Garrett, Vallejo, Calif.
 Otto H. Grunow, St. Louis
 R. N. Hirst, Ogden, Utah
 Robert H. Johnson, Tulsa, Okla.
 Robert E. Koch, St. Louis
 James Mann, Boston, Mass.
 Joseph J. Mira, Alton, Ill.
 Gordon F. Moore, Alton, Ill.
 Charles G. Obermeyer, St. Louis
 Willard D. Rowland, Portland, Ore.

Leo A. Sachar, St. Louis
 Llewellyn Sale, Jr., St. Louis
 Harry W. Sawyer, San Francisco, Calif.
 John S. Skinner, St. Louis
 Robert M. Smith, St. Louis
 W. L. Tomlinson, St. Louis

1939—Living Graduates, 96

Alfred K. Baur, St. Louis
 Irving L. Berger, Cleveland, Ohio
 Vilray P. Blair, Jr., St. Louis
 Leo J. Blum, Jr., Warner Robins, Ga.
 Joseph Borenstine, Kansas City, Mo.
 Sidney S. Boyers, W. New York, N. J.
 Mark J. Brockbank, Petaluma, Calif.
 Heinz E. Cron, San Francisco, Calif.
 John W. Dix, Miami, Fla.
 Leon J. Fox, St. Louis
 William B. Hildebrand, Menasha, Wis.
 Leonard H. Jacobson, Miami Beach, Fla.
 Benjamin Milder, St. Louis
 Edward H. Reinhard, St. Louis
 Minton D. Ritter, Margate City, N. J.
 R. J. Roscow, Evansville, Ind.
 Gerald A. Slusser, Silver City, N. Mex.
 O. W. Towers, St. Charles, Mo.

1938—Living Graduates, 92

Harry A. Baers, North Hollywood, Calif.
 G. W. Blankenship, Anderson, Mo.
 Robert D. Brookes, St. Louis
 Margaret A. Carter, St. Louis
 Kenneth L. Carter, Beloit, Wis.
 Adolph H. Conrad, Jr., St. Louis
 Marion J. Dakin, Los Angeles, Calif.
 Harold R. Gilbert, Wyandotte, Mich.
 Nathan Kimelman, St. Louis, Mo.
 Lawrence M. Kotner, St. Louis
 Harry Mantz, Alton, Ill.
 Robert G. Moles, Hanford, Calif.
 Alexander A. Mueller, Los Angeles, Cal.
 Anthony Piraino, Oberlin, Ohio
 Joseph H. Pollock, Los Angeles, Calif.
 Philip Rosenblatt, New York, N. Y.
 Samuel Schultz, Clayton, Mo.
 Roy W. Thomas, Redding, Calif.
 J. L. Doenges, Anderson, Ind.

1937—Living Graduates, 93

Thomas S. Boozer, Montgomery, Ala.
 Samuel Brady, Gary, Ind.
 Paul A. Brenner, Owensville, Mo.
 G. L. Calvy, Cleveland, Ohio

R. G. Carter, Austin, Tex.
 Martin A. Compton, Richmond, Va.
 John R. Connell, Denver, Colo.
 Samuel M. Day, Jacksonville, Fla.
 J. M. Dougall, Houston, Tex.
 J. A. Florito, New Haven, Conn.
 Edward A. Harris, Birmingham, Ala.
 Lester E. Haentzschel, Appleton, Wis.
 William H. Gray, Yakima, Wash.
 Carroll W. Huffman, Los Angeles, Calif.
 Arthur A. Kaplan, Utica, N. Y.
 Robert C. Kingsland, St. Louis
 Carl E. Lischer, St. Louis
 Edgar H. Little, New Orleans, La.
 Lewis E. Littmann, St. Louis
 Elizabeth Lowenhaupt, San Francisco
 M. S. McGrath, Weiser, Idaho
 A. E. Meisenbach, Jr., Dallas, Tex.
 John E. Miksicek, St. Louis
 Ralph C. Petersen, Glendale, Calif.
 Charles M. Polan, Huntington, W. Va.
 J. J. Pontier, Richmond, Calif.
 William J. Quinn, Alturas, Calif.
 Henry N. Reid, Rome, N. Y.
 Lloyd Rosenbaum, Anderson, Ind.
 Harvey S. Smith, Boise, Idaho
 Walter Stevenson, Jr., Quincy, Ill.
 H. L. Townsend, Louisville, Ky.
 B. C. Trowbridge, Kansas City, Mo.
 David R. Wall, Wichita, Kan.
 Ellsworth A. Westrup, Webster Groves,
 Mo.
 Marie H. Wittler, Wheaton, Ill.

1936—Living Graduates, 102

Wallace E. Allen, Oakland, Calif.
 Charles A. Brasher, Mt. Vernon, Mo.
 Lawrence Breslow, Chicago, Ill.
 James H. Bryant, St. Louis
 F. R. Crouch, Farmington, Mo.
 Norman W. Drey, St. Louis
 Stephen Ellis, Coffeyville, Kan.
 Curtis H. Epps, Springfield, Mo.
 John L. Horner, St. Louis
 W. H. Jacobson, Canton, Ohio
 Hyman Jaffe, Beverly Hills, Calif.
 Nathan R. Kahn, Brooklyn, N. Y.
 George M. Klingner, Springfield, Mo.
 Vernon Lundmark, Seattle, Wash.
 Frank McDowell, St. Louis
 W. T. McNew, Carthage, Missouri

James D. Morrison, Billings, Mont.
 R. A. Nussbaum, St. Louis
 Samuel Schneider, St. Louis
 William L. Sellers, Jr., Mobile, Ala.
 E. H. Trowbridge, Jr., Kansas City, Mo.
 Michael S. Wepprich, Washington, Mo.
 Warren B. West, Ogden, Utah
 Robert A. Wise, Houston, Tex.
 William R. Young, Salt Lake City, Utah

1935—Living Graduates, 88

K. M. Amlin, Honolulu, T. H.
 Arthur P. Echternacht, Fort Dodge, Ia.
 I. J. F lance, St. Louis
 Elmer G. Graul, St. Louis
 Heinz Haffner, St. Louis
 Alfred W. Harris, Dallas, Tex.
 A. Herman Hutto, St. Louis
 Norman M. Johnson, Clarinda, Iowa
 Jacob Katzeff, Brooklyn, N. Y.
 Bruce Kenamore, St. Louis
 Kenneth V. Larsen, St. Louis
 Ellen S. Loeffel, St. Louis
 Edward Massie, St. Louis
 Sidney Messer, Venice, Calif.
 Clark G. Porter, Three Rivers, Mich.
 Laurence G. Pray, Fargo, N. D.
 David Rothman, St. Louis
 Bernard Schwartzman, St. Louis
 Ben H. Senturia, St. Louis
 A. J. Steiner, St. Louis
 David O. Weiner, Brooklyn, N. Y.
 Irvin Weisman, Granite City, Ill.
 John W. Williams, Oak Grove, Mo.

1934—Living Graduates, 88

Helen M. Aff, St. Louis
 Edmund B. Alvis, St. Louis
 James M. Baker, Columbia, Mo.
 Garvey Bowers, Kokomo, Ind.
 Eugene M. Bricker, St. Louis
 John and Katherine Brown, Fulton, Mo.
 L. F. Bush, Danville, Pa.
 Everett S. Caldemeyer, Washington,
 D. C.
 T. C. Campbell, New Orleans, La.
 David Friedman, Granite City, Ill.
 Ben I. Frissel, Phoenix, Ariz.
 William W. Gist, Kansas City, Mo.
 Paul O. Hagemann, St. Louis
 Stanley Hampton, St. Louis
 Louis G. Jekel, Phoenix, Ariz.

Dorothy J. Jones, St. Louis
 Ralph R. Jones, Boise, Idaho
 Paul C. Kunkel, Newington, Conn.
 Morris D. Marcus, St. Louis
 M. Norman Orgel, St. Louis
 Jean F. Rogier, Bogota, Colombia, S. A.
 H. D. Rosenbaum, St. Louis
 John A. Saxton, St. Louis
 Edna Schrick, Oakland, Calif.
 James G. Telfer, Chicago, Ill.
 Martin P. Hunter, Kansas City, Mo.
 Fred C. Reynolds, St. Louis

1933—Living Graduates, 89

Henry C. Allen, St. Louis
 James W. Bagby, St. Louis
 Russell J. Blattner, Houston, Tex.
 Cecil M. Charles, St. Louis
 Lee W. Dean, Jr., St. Louis
 Truman G. Drake, St. Louis
 Wallace D. English, Cardwell, Mo.
 Charles H. Flynn, Clarinda, Ia.
 George E. Grim, Kirksville, Mo.
 Carl G. Harford, St. Louis
 John R. Haslem, Terre Haute, Ind.
 W. W. Herman, Cleveland, Ohio
 Joseph C. Jaudon, St. Louis
 F. Craig Johnson, Denver, Colo.
 A. A. Loverde, Chicago, Ill.
 R. R. Merrell, Pocatello, Idaho
 Alvin R. Miller, Seattle, Wash.
 Louis A. Motchan, Beverly Hills, Calif.
 Charles Oderr, New Orleans, La.
 Lyman K. Richardson, New Orleans, La.
 J. F. Roufa, St. Louis
 Richard Y. Sakimoto, Honolulu, T. H.
 Robert S. Smith, Boise, Idaho
 Robert T. Terry, Nashville, Tenn.
 Oreon K. Timm, Danville, Ill.
 R. M. Van Matre, Oklahoma City, Okla.
 Wirt A. Warren, Wichita, Kan.
 Lawrence M. Wilson, Olympia, Wash.
 J. J. Wimp, Kirksville, Mo.
 Lawrence M. Wilson, Olympia, Wash.
 George J. L. Wulff, Jr., St. Louis
 Frank G. Zingale, St. Louis
 George E. Zukovich, San Diego, Calif.

1932—Living Graduates, 85

Harry Agress, St. Louis
 Sim F. Beam, St. Louis
 Brian B. Blades, Washington, D. C.

- Louis T. Byars, St. Louis
 B. S. Clark, Spearfish, S. D.
 William Ehrlich, Newark, N. J.
 Leo Gottlieb, St. Louis
 John B. Grow, Denver, Colo.
 Kikoshi Inouye, Honolulu, T. H.
 Virgil E. Jeans, Joylin, Mo.
 D. H. Kaump, Detroit, Mich.
 Kenneth C. Kehl, Racine, Wis.
 Edward J. Kloess, Selfridge, Mich.
 Paul H. Lefkowitz, Spring Valley, N. Y.
 William H. Meinberg, St. Louis
 Carl V. Moore, St. Louis
 Paul B. Nutter, Spokane, Wash.
 Donald M. Paton, Houston, Tex.
 Sydney S. Pearl, Elizabeth, N. J.
 Joseph Rebillot, Litchfield, Ill.
 C. O'Neil Rich, Salt Lake City, Utah
 Wendell G. Scott, St. Louis
 Don J. Silsby, Springfield, Mo.
 Barrett L. Taussig, St. Louis
 Dwight H. Trowbridge, Fresno, Calif.
 Sam R. Wallis, Kauai, T. H.
 Helman C. Wasserman, St. Louis
 John C. Wilson, San Jose, Calif.
 Irving Wyle, Brooklyn, N. Y.
- 1931—Living Graduates, 73
 Delevan Calkins, St. Louis
 E. W. Cannady, E. St. Louis, Ill.
 Joseph Cieri, Piedmont, Calif.
 D. B. Elrod, Cape Girardeau, Mo.
 Ben Friedman, McKinney, Tex.
 W. Wallace Greene, Kansas City, Mo.
 A. W. Hankwitz, Milwaukee, Wis.
 W. E. Keiter, Kinston, N. C.
 Morris Krutchkoff, San Francisco, Calif.
 H. R. McCarroll, St. Louis
 Max Magnes, Paterson, N. J.
 Robert F. Monroe, Louisville, Ky.
 Mary Louise Newman, Jacksonville, Ill.
 Edwin C. Schmidtke, Columbia, Mo.
 John A. Schindler, Monroe, Wis.
 R. B. Wray, Nevada, Mo.
 Takeo Yamashita, Allentown, Pa.
- 1930—Living Graduates, 76
 Harold S. Bowman, Wichita, Kan.
 M. A. Brennecke, Waimea, Kauai, T. H.
 J. Paul Burgess, Hyrum, Utah
 M. A. Diehr, St. Louis
 Donald E. Eggleston, Macon, Mo.
- Virgin O. Fish, St. Louis
 Herbert H. Gass, India
 Joseph J. Gitt, St. Louis
 Stanley Harrison, St. Louis
 Alfred H. Hathcock, Fayetteville, Ark.
 Walter M. Howard, Joplin, Mo.
 James D. Horton, Springfield, Mo.
 I. D. Newmark, Chester, Ill.
 Garrett Pipkin, Kansas City, Mo.
 Austin C. Taylor, Spokane, Wash.
- 1929—Living Graduates, 71
 Grace Edwards Barar, Allahabad, India
 Carl S. Bickel, Wheeling, W. Va.
 Leslie C. Drews, Clayton, Mo.
 A. W. Freshman, Denver, Colo.
 Guerdan Hardy, St. Louis
 F. L. Harms, Salisbury, Mo.
 Craig B. Johnston, Philadelphia, Pa.
 Louis Kovitz, Kansas City, Mo.
 Sidney Pakula, Kansas City, Mo.
 V. L. Peterson, Charleston, W. Va.
 Frank B. Queen, Portland, Ore.
 A. P. Rowlette, Moberly, Mo.
 Jay Marvin Salzman, Springfield, Ill.
 Arthur E. Varden, San Bernardino, Cal.
 A. Ford Wolf, Temple, Tex.
- 1928—Living Graduates, 69
 A. N. Arneson, St. Louis
 William Brewer, Hays, Kans.
 Edward Burns, Toledo, Ohio
 Justin J. Cordonnier, St. Louis
 Roland F. Elkins, Springfield, Mo.
 John S. Harter, Louisville, Ky.
 H. R. Hildreth, St. Louis
 Laurence L. Howard, Great Falls, Mont.
 J. Ted Jean, St. Louis
 R. D. Kepner, Honolulu, T. H.
 Guy N. Magness, St. Louis
 L. A. Malone, Terre Haute, Ind.
 Earl L. Mills, Wichita, Kan.
 John F. Patton, St. Louis
 A. Victor Reese, St. Louis
 Paul R. Rollins, Seattle, Wash.
 Verne Ross, Stockton, Calif.
 W. A. Ruch, Memphis, Tenn.
 O. G. Schneidewind, New Athens, Ill.
 B. Wright Shelton, Miami, Okla.
 David M. Skilling, St. Louis
 A. Lloyd Stockwell, Kansas City, Mo.
 Jacob Stolar, St. Louis

- Vincent T. Williams, Kansas City, Mo.
George H. Wood, Carthage, Mo.
- 1927—Living Graduates, 70
Philip K. Allen, San Diego, Calif.
Louis F. Aitken, St. Louis
L. N. Claiborn, New Haven, Conn.
Everett C. Drash, Charlottesville, Va.
A. C. Fortney, Fargo, N. D.
William Goodlett, Olean, N. Y.
Paul H. Guttman, Sacramento, Calif.
Alfred G. Henrich, Los Angeles, Calif.
A. G. Klein, St. Louis
Irene A. Koencke, Halstead, Kans.
C. H. Leslie, Kirkwood, Mo.
W. R. Merrell, Brigham City, Utah
Alfred J. Metscher, Enid, Okla.
Kazuo Miyamoto, Honolulu, T. H.
Eugene O. Parsons, Kansas City, Mo.
Willard C. Schwartz, Manhattan, Kan.
Abigail E. Smith, Lexington, Mass.
Frances H. Stewart, St. Louis
Richard T. Taylor, Los Angeles, Calif.
Louis L. Tureen, St. Louis
Franklin Walton, St. Louis
W. B. Wilcoxon, Bowling Green, Mo.
George S. Wilson, Enid, Okla.
- 1926—Living Graduates, 73
Reno A. Ahlvin, Kankakee, Ill.
Herbert Anderson, Los Angeles, Calif.
Willard Bartlett, Jr., St. Louis
James L. Benepe, St. Paul, Minn.
H. M. Chandler, Waipahu, T. H.
Erich A. Cunningham, Louisiana, Mo.
Max Deutch, St. Louis
Charles W. Duden, St. Louis
Andy Hall, Jr., St. Louis
William M. James, St. Louis
William B. Kountz, St. Louis
John G. Manning, McMinnville, Ore.
G. Wendell Olson, Fullerton, Calif.
Walter R. Peterson, Trenton, N. J.
Bernard Rand, New York City
Henry A. Romberg, Oshkosh, Wis.
J. C. Schmidtke, Elgin, Ill.
E. H. Theis, Granite City, Ill.
- 1925—Living Graduates, 67
George P. Bailey, Lakewood, Colo.
Robert J. Crossen, St. Louis
H. M. Denny, Union, Mo.
James J. Donohue, E. St. Louis, Ill.
- B. Y. Glassberg, St. Louis
A. E. Hiebert, Wichita, Kan.
Richard K. Kimmel, St. Louis
James I. Knott, San Diego, Calif.
Jerome S. Levy, Little Rock, Ark.
Joseph Magidson, St. Louis
Carl H. Matthey, Davenport, Iowa
Loren D. Moore, Wellesley Hills, Mass.
James O. Nall, Marion, Ky.
Sam J. Roberts, Miami, Fla.
Melvin A. Roblee, St. Louis
Roland A. Slater, Peoria, Ill.
S. D. Soule, St. Louis
Winton T. Stacy, Fort Sill, Okla.
R. O. Stickler, Kirksville, Mo.
Gershom J. Thomson, Rochester, Minn.
Hugo O. Wagner, Great Lakes, Ill.
- 1924—Living Graduates, 68
Alfred O. Adams, Spokane, Wash.
Eugene S. Auer, Denver, Colo.
Roy F. Basket, Texarkana, Tex.
J. William Beckmann, New York, N. Y.
Harry J. Davis, Topeka, Kans.
Charles Drabkin, Los Angeles, Calif.
Perry E. Duncan, Springfield, Ill.
George H. Garrison, Oklahoma City, Okla.
H. V. Gibson, Great Falls, Mont.
William B. Gnagi, Monroe, Wis.
Scott Johnson, New York City
Louis H. Jorstad, St. Louis
Elizabeth E. Koppenaar, Elmhurst, Ill.
A. E. Meinert, Winona, Minn.
E. B. Pfefferkorn, Oshkosh, Wis.
Reuben M. Smith, St. Louis
O. Earl Whitsell, St. Joseph, Mo.
- 1923—Living Graduates, 47
Oliver Abel, Jr., St. Louis
William G. Becke, St. Louis
William L. Bradford, Rochester, N. Y.
James Barrett Brown, St. Louis
Ben M. Bull, Ironton, Mo.
I. Z. Davidoff, Milwaukee, Wis.
Walter J. Decker, Westfield, Pa.
George V. Feist, Kansas City, Mo.
Elias H. Schlomovitz, Milwaukee, Wis.
Ben D. Senturia, Chicago, Ill.
Charles Teel, Bellingham, Wash.
J. Wm. Thompson, St. Louis
Clair O. Vingom, Madison, Wis.

1922—Living Graduates, 44

K. S. Chouke, Philadelphia, Pa.
 Calvin Clay, St. Charles, Mo.
 James B. Costen, St. Louis
 Aphrodite J. Hofsommer, Webster
 Groves, Mo.
 Armin C. Hofsommer, Webster Groves,
 Mo.
 Kirby A. Martin, New York, N. Y.
 F. E. Sultzman, Hannibal, Mo.

1921—Living Graduates, 42

Lester J. Evans, Jackson Heights, N. Y.
 Oliver W. Lohr, Saginaw, Mich.
 J. C. McKitterick, Burlington, Iowa
 Harvey S. Rusk, Pueblo, Colo.
 Oscar C. Zink, St. Louis

1920—Living Graduates, 39

Robert L. Andrae, Louisiana, Mo.
 Clifton H. Briggs, Pasadena, Calif.
 Warren H. Cole, Chicago, Ill.
 Alfred Goldman, St. Louis
 Samuel B. Grant, St. Louis
 Guy H. Hopkins, Pueblo, Colo.
 William A. Hudson, Detroit, Mich.
 W. N. Jenkins, Port Gibson, Miss.
 Frederick E. Jostes, St. Louis
 P. H. Kennedy, Hubbard, Ohio
 Herman M. Meyer, St. Louis
 L. J. Owen, Lincoln, Neb.
 M. G. Peterman, Milwaukee, Wis.
 Royal W. Rudolph, Tucson, Ariz.
 H. W. Wellmerling, Bloomington, Ill.
 Harvey Lester White, St. Louis

1919—Living Graduates, 45

Duff S. Allen, St. Louis
 S. P. Funkhouser, Lake County, Calif.
 Howard H. Heuston, Bolder, Colo.
 Fred J. Hodges, Ann Arbor, Mich.
 Carl O. Kohlbry, Duluth, Minn.
 Marriott T. Morrison, Mt. Horeb, Wis.
 E. H. Munro, Grand Junction, Colo.
 Raymond L. Murdoch, Oklahoma City,
 Okla.
 Howard A. Plank, New York, N. Y.
 A. B. Raffl, Syracuse, N. Y.
 R. P. Roantree, Elko, Nev.
 A. L. Walter, Sedalia, Mo.

1918—Living Graduates, 26

Glover H. Copher, St. Louis
 Wilbur G. Gillett, Wichita, Kan.

Elmer N. Liljedahl, Hollywood, Calif.
 Arthur G. Mahle, Chicago, Ill.
 J. F. Pessel, Trenton, N. J.
 O. Sundwall, Murray, Utah
 James A. Tesson, Kansas City, Mo.

1917—Living Graduates, 25

Archie A. Skemp, La Crosse, Wis.
 J. E. Wattenberg, Cortland, N. Y.

1916—Living Graduates, 13

E. L. Dallwig, Milwaukee, Wis.
 Earl C. Sage, Omaha, Neb.
 Ray T. Woolsey, Salt Lake City, Utah

1915—Living Graduates, 20

F. F. Alsup, Honolulu, T. H.
 D. K. Rose, St. Louis
 J. E. Strode, Honolulu, T. H.
 W. T. Wilkening, Fort Scott, Kans.

1914—Living Graduates, 8

O. F. McKittrick, Linglestown, Pa.
 John T. McLarney, Brookfield, Mo.

1913—Living Graduates, 20

F. O. Kettelkamp, Colorado Springs,
 Colo.

1912—Living Graduates, 30

C. F. DeGaris, Oklahoma City, Okla.
 Roy G. Empson, Valmeyer, Ill.
 Edwin C. Ernst, St. Louis
 George S. Gilpin, Cleveland, O.
 W. N. O'Bannon, New Madrid, Mo.
 Wells C. Reid, Goodrich, Mich.
 A. P. Erich Schulz, St. Charles, Mo.
 George L. Watkins, Farmington, Mo.

1911—Living Graduates, 20

Thomas M. Davis, St. Louis
 Clyde P. Dyer, St. Louis
 William H. Fickel, Denver, Colo.
 Charles H. Hecker, Palo Alto, Calif.
 Arthur J. Wagers, Philadelphia, Pa.

1910—Living Graduates, 40

Stanley S. Burns, St. Louis
 Frederic Hagler, Springfield, Mass.
 Robert M. Hardaway, Wheatridge, Colo.
 John P. Keim, St. Louis
 Peter G. Moskop, St. Louis
 Claude D. Pickrell, St. Louis
 Frederick O. Schwartz, St. Louis

1909—Living Graduates, 29

James W. Barrow, Carbondale, Ill.
 Carey B. Elliott, Raton, N. Mex.

- W. N. Pugh, Salt Lake City, Utah
Richard S. Weiss, St. Louis
- 1908—Living Graduates, 29
W. A. Olds, Colville, Wash.
O. J. Raeder, Boston, Mass.
W. D. Moore, San Pedro, Calif.
- 1907—Living Graduates, 28
C. C. Nash, Dallas, Tex.
Grandison D. Royston, Hope, Ark.
Llewellyn Sale, St. Louis
Raymond M. Spivy, St. Louis
- 1906—Living Graduates, 33
Martin J. Glaser, St. Louis
Arthur Gundlach, St. Louis
T. A. Lawler, Taylorville, Ill.
S. P. Martin, East Pairie, Mo.
S. B. McPheeters, Goldsboro, N. C.
William H. Smith, Colfax, Calif.
- 1905—Living Graduates, 30
Jerome E. Cook, St. Louis
Walter Fischel, St. Louis
Harold M. Griffith, Pasadena, Calif.
J. M. James, Henning, Ill.
O. W. Knewitz, East St. Louis
- 1904—Living Graduates, 31
Paul Baldwin, Kennett, Mo.
W. Q. Conway, Kalispell, Mont.
N. M. Freund, St. Louis
Harry L. Jones, Kansas City, Mo.
Roy P. Scholz, St. Louis
J. H. Woodbridge, Pueblo, Colo.
- 1903—Living Graduates, 20
A. H. Myerdick, Mt. Pleasant, Iowa
Clive D. Scott, Louisiana, Mo.
- 1902—Living Graduates, 22
- 1901—Living Graduates, 14
John M. Bradley, St. Louis
W. C. Forder, St. Louis
Walter C. G. Kirchner, St. Louis
M. K. Wyler, Albuquerque, N. Mex.
- 1900—Living Graduates, 2
- 1899—Living Graduates, 34
Charles Arkebauer, Van Buren, Ark.
J. C. Caldwell, Wellington, Kans.
C. L. Lawless, Marshall, Mo.
R. O. Raymond, Flagstaff, Ariz.
Selden Spencer, St. Louis
Julian B. Woodson, Lowesville, Va.
- 1898—Living Graduates, 27
J. G. W. Fischer, Alma, Mo.
R. B. H. Gradwohl, St. Louis
John Q. Roane, Carlyle, Ill.
A. L. Stuttle, Williamsville, Ill.
- 1897—Living Graduates, 21
Theodore Greiner, St. Louis
Frederick E. Woodruff, St. Louis
- 1896—Living Graduates, 21
- 1895—Living Graduates, 21
H. A. Geitz, Monterrey, N. L., Mexico
Sandor Horwitz, Peoria, Ill.
Robert J. Terry, St. Louis
William A. Tolleson, Eufala, Okla.
- 1894—Living Graduates, 13
- 1893—Living Graduates, 12
Andrew Darling, St. Louis
R. Clarence Stephens, Plymouth, Ind.
- 1892—Living Graduates, 4
- 1891—Living Graduates, 15
- 1890—Living Graduates, 6
- 1889—Living Graduates, 6
- 1888—Living Graduates, 2
- 1887—Living Graduates, 3
- 1886—Living Graduates, 3
- 1885—Living Graduates, 2
E. F. Ellis, Fayetteville, Ark.
- 1884—Living Graduates, 2
- 1883—Living Graduates, 2
W. A. Fries, St. Louis
- 1882—Living Graduates, 1
- 1881—Living Graduates, 3
James A. Dickson, St. Louis
Willis Hall, St. Louis
- 1880—Living Graduates, 2
Joseph M. Long, St. Louis

OTHER DONORS

- Mrs. T. R. Akin, Clayton, Mo.
Mr. William M. Akin, St. Louis
Harry L. Alexander, M.D., St. Louis
Robert W. Bartlett, M.D., St. Louis
Leon Bromberg, M.D., St. Louis
J. J. Bronfenbrenner, Ph.D., St. Louis
Samuel S. Bukantz, M.D., St. Louis
Martin M. Calodney, M.D., St. Louis
Benjamin H. Charles, M.D., St. Louis

Alfred J. Cone, St. Louis
 Drs. Carl F. and Gerty T. Cori, St. Louis
 Gustave J. Dammin, M.D., St. Louis
 Morris Davidson, M.D., St. Louis
 Hallowell Davis, M.D., St. Louis
 Joseph E. Edwards, M.D., St. Louis
 Ben Eiseman, M.D., St. Louis
 Robert Elman, M.D., St. Louis
 Lee T. Ford, St. Louis
 Robert J. Glaser, M.D., St. Louis
 Harry N. Glick, M.D., St. Louis
 Drs. Evarts and Helen Tredway Graham,
 St. Louis
 G. E. Gruenfeld, M.D., St. Louis
 Miss Helen D. Harkness, St. Louis
 Leopold Hofstatter, M.D., St. Louis
 Mr. W. W. Horner, St. Louis
 Alex H. Kaplan, M.D., St. Louis
 J. Albert Key, St. Louis
 John Esben Kirk, M.D., St. Louis
 Paul E. Kubitschek, M.D., St. Louis

K. Cramer, Lewis, St. Louis
 Grover Liese, M.D., St. Louis
 Robert G. Loeffel, St. Louis
 Sedgwick Mead., M.D., St. Louis
 Albert I. Mendeloff, M.D., St. Louis
 Ivan N. Mensh, Ph.D., St. Louis
 William H. Olmsted, M.D., St. Louis
 Ernest H. Parsons, M.D., St. Louis
 Joseph C. Peden, Sr., M.D., St. Louis
 Lawrence T. Post, M.D., St. Louis
 M. Hayward Post, M.D., St. Louis
 Herman J. Rosenfeld, M.D., St. Louis
 Theodore B. Rosenthal, Ph.D., St. Louis
 Harold Scheff, M.D., St. Louis
 Arthur E. Strauss, M.D., St. Louis
 A. C. Stutsman, M.D., St. Louis
 Robert Votaw, M.D., St. Louis
 Theodore Walsh, M.D., St. Louis
 Carl R. Wegner, M.D., St. Louis
 Park J. White, M.D., St. Louis
 Ralph B. Woolf, M.D., St. Louis

DEPARTMENTAL NEWS

Anatomy

Dr. George B. Rowe, anatomy, and Dr. M. B. Roche, surgery, were awarded an honorable mention for their exhibit on "Separate Neural Arch" in the Orthopedic Section at the American Medical Association meeting in Atlantic City in June.

At the American Association of Physical Anthropologists meeting in Ann Arbor, Mich., March 19-20, Dr. Mildred Trotter, professor of gross anatomy, and Dr. Goldine Gleser and Mr. Oliver Duggins, research assistants, presented scientific papers. Dr. Trotter and Dr. Gleser gave a paper on changes in stature after maturity, and Mr. Duggins spoke on a procedure for determining refractive index of hair with some preliminary findings.

Biochemistry

Miss Anne Perley, instructor in pediatrics in biochemistry, resigned her position and left July 1 to become assistant professor of biochemistry at the University of Oregon in Portland.

Cancer Research

Dr. Eugene Roberts, research associate in cancer, left early in June to work at the Jackson Memorial Laboratory in Bar Harbor, Me., for the summer. This is the third summer he has worked in Bar Harbor.

Gerontology

Dr. William B. Kountz attended the meeting of the American Hospital Association Assembly in Atlantic City,

June 6-8. He presented a paper on protein requirements of elderly individuals before the American Geriatrics Society on June 8th.

Internal Medicine

Dr. W. Barry Wood, Jr., professor of medicine, attended meetings of the American Society for Clinical Investigation, Association of American Physicians, and the Armed Forces Epidemiological Board in Atlantic City and Washington between April 30 and May 4.

Dr. Richard Weiss and Dr. Clinton Lane attended the meeting of the American Dermatological Association in Hot Springs, Va., May 22-26. On June 5, Dr. Weiss and Dr. Adolph H. Conrad, Jr., addressed the General Practitioners Postgraduate Club of St. Elizabeth's Hospital in Washington, Mo., presenting a series of colored slides of common skin diseases, and discussed diagnosis and treatment of those diseases.

Dr. Paul O. Hagemann, Dr. Bernard Hulbert and Herman Rosenfeld attended the American Rheumatism Association meeting in Atlantic City, June 8-10.

Neuropsychiatry

Drs. Edwin F. Gildea, George Ulett, George Saslow, D. Wells Goodrich, Jr., George Winokur, Marvin Stein and Mrs. Janet Golden attended the meetings of the American Psychiatric Association in Cincinnati, May 7-11. Dr. Stein presented a paper on the subject "Physiological Response to Heat Stress and ACTH of Normal and Schizophrenic

Subjects," which was authored by him, with Dr. Ethel Ronzoni and Dr. Edwin Gildea.

Dr. Robert I. Watson, associate professor of medical psychology, visited the School of Aviation Medicine at Randolph Field, Texas, April 18-21 in connection with research on the psychiatric screening program for air force personnel which he is conducting for the Air Force. Dr. George Ulett, assistant professor of psychiatry, also visited at Randolph Field in connection with a contract for research which he is conducting on the use of the EEG in psychiatric screening.

Dr. Saul Rosenzweig, professor of medical psychology, attended the Thirteenth International Congress of Psychology in Stockholm, Sweden from July 15-21. He presented a paper entitled "Frustration, Growth and the Creative Process," and participated, by invitation, in a Symposium on "The Relationships between Psychoanalysis and Psychology."

Dr. James L. O'Leary, professor of neurology, was elected president of the American Electroencephalographic Society at its 5th annual meeting held in Atlantic City, New Jersey, on July 15, 16 and 17. Dr. O'Leary has been vice-president of the society for the past year and will now succeed Dr. Robert S. Schwab of Boston, Mass., in the presidency.

Obstetrics-Gynecology

Dr. Alfred I. Sherman and Dr. MacDonald Bonebrake attended the meeting of the American Radium Society in

Atlantic City, June 7-13, presenting a paper on the use of radioactive colloidal gold.

Dr. Willard C. Scrivner spoke on "Common Complaints in Obstetrics and Gynecology" at the meeting of the Illinois State Medical Association at Chicago, Ill., May 24.

Otolaryngology

Dr. Hallowell Davis, director of research at Central Institute for the Deaf and research professor of physiology, has been reappointed chairman of the Committee on Hearing of the National Research Council, it was announced by Dr. C. Winternitz, chairman of the Council's Division of Medical Sciences. Dr. Davis will serve in this capacity for a year effective July 1. He is also a member of the psychophysiology panel of the Office of Naval Defense.

Dr. Richard Silverman, director of Central Institute for the Deaf, was a special guest of honor and a principal speaker at the Second Latin American Congress of Otolaryngology and Broncho-Esophagology in Sao Paulo, Brazil, July 8-14. In the course of the Congress, Dr. Silverman delivered one major address and four other lectures.

Pathology

The Federal Civilian Defense Administration in Washington has appointed Dr. Gustave J. Dammin, head of the Department of Pathology, as an advisor to the group. He attended a meeting in Washington on May 3 to discuss laboratory organization for civilian defense needs. Prior to this meeting, he was in

Atlantic City for the American Society for Clinical Investigation.

The following members of the Department of Pathology attended the meeting of the American Association of Pathologists and Bacteriologists which was held in Cleveland, April 26-28: Drs. Gustave Dammin, Margaret Smith, Edward Smith, David Smith, Frank Dixon, Robert A. Moore, Zola Copper.

Dr. Robert A. Moore, dean of the Medical School and professor of pathology, gave the Mellon Lecture of the Society for Biological Research at the University of Pittsburgh on May 9. He spoke on "The Nature of Teratomatous Tumors." He returned to his alma mater, Ohio State University in Columbus, O. on May 15 and 16 to participate in the dedication and opening of the new Medical Center there. On May 10, Dr. Moore attended a meeting of the Committee on Pathology of the National Research Council in Washington, and he took part in a meeting of the National Board of Medical Examiners in Philadelphia the following day. He met with the examination committee of the National Board in Princeton, N. J. on May 12 and with the Cancer Control Advisory Committee in Washington on May 25.

Dr. Moore also attended the International Congress of Clinical Pathology which was held in London, July 16-20 and read a paper on "Histoplasmosis" at the conference. He is president of the American Association of Pathologists and Bacteriologists and also president of the American Board of Pathology.

Pharmacology

The following members of the Department of Pharmacology attended meetings of the Federation of American Societies for Experimental Biology held in Cleveland the week of April 30: Drs. Oliver H. Lowry, F. Edmund Hunter, Robert Furchgott, Eli Robins, Morris Friedkin, Jack Strominger, Byron Wenger and Helen Graham.

Physical Medicine

As area consultant in physical therapy, Dr. Sedgwick Mead, director of the division of physical medicine, visited Winterer Veterans Hospital in Topeka, Kansas on April 5. Dr. Mead presented an exhibit entitled "Electrotherapy" at the meeting of the American Medical Association in Atlantic City, N. J., June 11-15.

Miss Beatrice F. Schulz, technical director of the School of Physical Therapy, attended the annual convention of the American Physical Therapy Association, which was held June 18-22 at Glenwood Springs, Colo.

Radiology

Radiology Department staff members who participated in the American College of Physicians meeting here during April were: Drs. A. N. Arneson, Gladden Elliot, William B. Seaman, Wendell G. Scott and Michel Ter-Pogossian.

Surgery

Dr. James Barrett Brown, professor of clinical surgery, presented the Trimble Lecture of the Medical and Chirurgical Faculty of the State of Maryland in

Baltimore on April 24. He spoke on "Rehabilitation of War Injuries."

Dr. Charles Eckert, assistant professor of surgery, spoke on the principles of cancer surgery at the Polk County Cancer Institute which met in Des Moines, Iowa on April 18.

Dr. Evarts A. Graham, professor emeritus of surgery, received an award from the Homer G. Phillips Hospital Internes' Alumni Association on April 26 for outstanding contributions to the hospital and the alumni.

Dr. Henry G. Schwartz and Dr. Leonard Furlow attended the Society of

Neurological Surgeons' meeting at Rochester, Minn., in April.

Dr. M. B. Roche gave a paper at a meeting of the Midwest Orthopedic Club in Cleveland, O., June 1-2 on "Posterior Dislocation of the Hip, Complicated by Acetabular Fractures."

Dr. Robert Elman attended meetings of the American Gastroenterological Society and the American Medical Association at Atlantic City. At the former, he discussed a paper on pancreatitis, and at the AMA meeting, he discussed Dr. Frederick Stare's paper on intravenous fat.

ALUMNI NEWS

1894

Horace W. Soper and **Adolph Schlossstein** of St. Louis attended the reunion banquet.

1895

Robert Schlueter and **C. G. Ahlbrandt**, both of St. Louis and **Newton T. Enloe** of Chico, Calif., were present at the reunion banquet.

1897

F. E. Woodruff of St. Louis attended the reunion.

1899

C. L. Lawless of Marshall, Mo. returned for the alumni banquet.

1901

Walter C. G. Kirchner, **W. C. Forder**, **Irwin J. Harris** and **John R. Lionberger**, all of St. Louis, were present at the alumni reunion as were **M. K. Wylder** of Albuquerque, N. M. and **Julius C. Bohn** of Tacoma, Wash.

1902

J. Mather Pfeifferberger of Alton, Ill. attended the alumni banquet.

1906

William Weiss, **John F. Gallagher**, **Martin J. Glaser**, **August W. Peters**, and **Arthur Gundlach**, all of St. Louis, were present at the alumni reunion.

1907

C. C. Nash, Dallas, Tex. and **P. Vinyard** of St. Louis attended the alumni banquet.

1909

M. F. Arbuckle of St. Louis was present at the alumni banquet.

1910

R. W. Lhamon has moved to Asheboro, N. C., where his address is Rt. 3—Hidden Hill.

1913

LeRoy Sante of St. Louis attended the alumni banquet.

1917

Robert Mueller, who during World War II served as head of the Physicians' Procurement and Assignment Service in Missouri, has been named chairman of the Missouri Advisory Committee on the Selection of Doctors, Dentists, and Allied Specialists, under the Selective Service Act. The appointment is, in effect, the same as the one he held during World War II.

1919

M. T. Morrison, of Mount Horeb, Wis., represented his class at the alumni reunion.

1920

H. L. White, **H. W. Wellmerling**, **Leon Brombert** and **Alfred Goldman** attended the alumni banquet. Dr. Wellmerling is from Bloomington, Ill., the others from St. Louis.

1921

Oscar C. Zink and **Alexis Hartmann**, both of St. Louis, represented their class at the alumni reunion.

1922

James B. Costen and **Theo. H. Hanser**, both of St. Louis, and **Gilbert L. Chamberlain** of New Franklin, Mo., were present at the alumni banquet.

1923

James Barrett Brown and **J. W. Thompson** attended the reunion banquet. Both are from St. Louis.

1924

Beryl C. Shearer's new address is Route 1, Box 285, El Cajon, Calif.

Louis H. Jorstad of St. Louis attended the alumni banquet.

1925

Col. Winton T. Stacy reports that he is now with the 379th Evac. Hosp. Ambl. at Camp Atterbury, Ind.

James J. Donahue of East St. Louis, Ill. and **Ben Fox** of Carbondale, Ill., were present at the reunion banquet.

1926

For the celebration of its silver anniversary since graduation, the class of 1926 had a turnout of 22 members. In addition to the reunion banquet, the group had a private cocktail party just preceding the banquet.

Those who attended the reunion are: **Karl D. Dietrich**, Columbia, Mo.; **L. M. Shapiro**, Kansas City, Mo.; **Eric A. Cunningham**, Louisiana, Mo.; **E. H. Theis**, Granite City, Ill.; **Walter Peterson**, Trenton, N. J.; **Herbert Anderson**, Los Angeles, Calif.; **George Gay**, Ironton, Mo.; **Robert Evans**, Russiaville, Ind.; and the following from St. Louis: **LeRoy L. Robertson**, **Wm. M. James**, **H. P. Thym**, **Jos. A. Bauer**, **Andy Hall, Jr.**, **Willard Bartlett, Jr.**, **Wm. B. Kountz**, **Charles Duden**, **A. G. Heideman**, **H. H. Feller**, **Max Deutsch**, **John Hartwig**, **Elmer Schluer** and **Walter Siebert**.

1927

D. K. Webb reports that he is now working at the State Sanatorium, Oakdale, Iowa.

Louis F. Aitken, **Franklin E. Walton**, **John E. Hobbs** and **Lee Harrison**, all of St. Louis and **Walter Whitaker**, Quincy, Ill. and **Hugh M. Wilson**, New Haven, Conn., were present at the reunion banquet.

1928

News of the promotion of **Col. Wilford F. Hall** to the rank of brigadier general was received recently. Gen. Hall has been a medical officer since 1928, and now has the position of air surgeon for the Military Air Transport Service.

Class members attending the alumni banquet were: **O. G. Schneidewind** of New Athens, Ill., and **Guy N. Magnus**, **A. N. Arneson**, **F. R. Bradley**, **Samuel D. Soule**, **John F. Patton** and **A. V. Reese** of St. Louis.

1929

Guerdan Hardy of St. Louis represented his class at the reunion banquet.

1930

Virgil O. Fish and **Clyde E. Kane**, both of St. Louis and **J. W. Tidwell** of Herrin, Ill., attended the alumni banquet.

1931

R. B. Bassett, **H. R. McCarroll** and **Delavan Calkins** were present at the alumni reunion.

1932

Sim F. Beam, **Wendell Scott** and **Leo Gottlieb** attended the reunion banquet.

1933

Robert T. Terry recently moved to 734 Balboa Ct., San Diego, Calif.

Carl G. Harford, **James W. Bagby**, **C. M. Charles**, **George Wulff, Jr.**, **J. C. Jaudon**, **Wm. T. K. Bryan**, all of St. Louis, and **John D. Maddox** of Joplin, Mo., were present at the alumni reunion.

1934

Class members attending the reunion banquet were: **Fred Reynolds**, **Stanley F. Hampton**, **John Jensen**, all of St. Louis and **David Friedman** of Granite City, Ill.

1935

Eight class members gathered for the alumni banquet on June 1. They were: **D. G. Cariss** and **Irving Wiesman** of Granite City, Ill.; **Max Goldenberg**, East St. Louis, Ill. and **Richard A. Sutter**, **I. J. Fiance**, **Ellen S. Loeffel**, **Heinz Haffner** and **Alfred Fleishman** of St. Louis.

1936

Commander Wallace E. Allen sent in his contribution to the Medical Student Dormitory Fund in April with a note saying that for the past eight months he has been aboard the hospital ship, U.S.S. *Repose*, AH-16, F.P.O., San Francisco, which was in Korean waters.

The following class members were present at the alumni banquet: **Curtis H. Eppes**, Springfield, Mo.; **Robert W. Elliott**, Alton, Ill. and **Emmett B. Drescher**, **R. W. Kelly**, **John L. Horner**, **Frank McDowell**, **James H. Bryan** and **Ralph Earp**, all of St. Louis.

1937

Lt. Col. and Mrs. Martin A. Compton, M.C., U.S. Army, have announced the birth of a son, Charles Carson, at the Medical College of Virginia Hospital, Richmond, Va., on April 30. Dr. Compton has been PMS & T and assistant in medicine there for the past two years.

Arthur P. Brewer, Alton, Ill., and **George W. Ittner**, **John E. Miksicek**, **Lawrence E. Mendonsa** and **Robert C. Kingsland** of St. Louis attended the alumni reunion.

1938

Leo J. Wade has moved to No. 1 Mayflower Rd., Scarsdale, N. Y. from Mamaroneck, N. Y.

Class members present at the reunion banquet were: **John H. Wedig**, **Harry Mantz**, both of Alton, Ill., and **A. H. Conrad, Jr.**, **Robert D. Brookes** and **Joshua E. Jensen** of St. Louis.

1939

A. Waite Bohne's new address is the Ford Hospital, Detroit, Mich.

Walter Baumgarten, Jr. and **Robert E. Shank** represented their class at the alumni banquet.

1940

The new address of **Robert L. Garrett** is 327 Georgia St., Vallejo, Calif.

Albert A. Ackerman has moved to 25 Kenyon Avenue, Berkeley, Calif.

The following class members attended the alumni banquet: **Fred Biggs, Jr.**, Poplar Bluff, Mo.; **Robert R. Anschutz**, Alton, Ill. and **Seymour Brown**, **Llewellyn Sale, Jr.**, **Robert E. Koch**, **David Kerr**, **A. T. Esslinger**, **Robert M. Smith**, **John S. Skinner**, **Wm. McDonald**, **Lindell Kinman**, **Ben Jolly** and **Leo Sacher**, all of St. Louis.

1941

Jack L. Baughman is now at 6012 A.S.V., U.S.A. Hospital, Camp Stoneman, Calif.

The new address of **B. L. Canaga, Jr.** is Office of the Naval Attache, Moscow, Russia, c/o Navy Pouch Section, Navy Dept., Washington 25, D. C.

Howard S. J. Walker, Jr. has offices in the Van Antwerp Bldg., Mobile, Ala., where he is in the private practice of surgery. His home is at 28 South St., Mobile.

Fifteen class members returned to the Medical School for their tenth anniversary. They were **Jim Kinder**, Cape Girardeau, Mo.; **V. A. Mueller**, Wichita, Kans.; **Richard F. La Force**, Sterling, Colo.; **Cecil Blackburn**, Selma, Ala.; **H. E. McCann**, East St. Louis, Ill. and St. Louisans **Joseph W. Noah**, **Samuel W. Gollub**, **Samuel E. Schecter**, **Barney W. Finkle**, **Mitchell Yanow**, **Kenneth A. Koerner**, **Calvin E. Ellis**, **Jane E. Kandorian**, **Henry Schwarz** and **William F. McGinnis**.

1942

Alex Harrell and **W. B. Mills** represented their class at the alumni reunion.

1943

Parker R. Beamer was announced as winner of the Moore County Award at the meeting of the Medical Society of the State of North Carolina in Pinehurst, N. C. on May 8. The award is given annually for the paper judged best among those presented in sessions of the preceding year. The title of the paper presented by Dr. Beamer in 1950 which won this year's prize was "Studies on Experimental Leptospirosis." Dr. Beamer is now professor of microbiology and immunology, and associate professor of pathology at Bowman Gray School of Medicine of Wake Forest College, Winston-Salem, N. C.

Lawrence Alred has moved to 113 N. Bell, Dothan, Ala.

The new address of **Edward H. Dunn** is Oteen Veterans Hospital, Oteen, N. C.

Capt. Carlton G. Watkins, M.C., U.S. A.R. is now stationed at Sta. Hospital No. 2, Fort Bragg, N. C.

E. N. Snyder, Jr., presented a paper on "Spontaneous Perforation of the Esophagus" at the regular dinner meeting of the Los Angeles County Medical Association on May 11.

Dr. and Mrs. Byron G. Wilson (Francis Chappell) announced the birth of a daughter, Ann Lynn Wilson, on November 13, 1950.

Carl Woolsey has moved from Brentwood, Mo. to 710 Boston Bldg., Salt Lake City, Utah.

The following class members attended the reunion banquet: **Donald Huelsmann**, Boonville, Mo.; **Raymond Rose**, Dupon, Ill. and **Burton A. Shatz**, **Alfred F. Sudholt**, **Ernest T. Rouse**, **Wm. G. Klingberg**, **Edward H. Kower**, **Melvin Goldman**, **Herbert C. Wiegand**, **Harold E. Walters** and **Grace Bergner** of St. Louis.

1944

George Donnell recently moved to 4208 Franklin, Burbank, Calif.

The new address of **Patrick A. Lynch** is 722 S. 30th Ave., Yakima, Wash.

Homer C. Marshall, Jr. is now located in the Professional Bldg., Glenn-Turner Medical Clinic, Springfield, Mo.

Three members of the class attended the alumni reunion—**Wayne Simril**, **Roy A. Walther** and **Roland F. Neuman**, all of St. Louis.

1945

Thomas J. Fitzpatrick has moved to 220 South Eastern Ave., Joliet, Ill.

The new address of **Thomas K. Hood** is 597 - 4th St., Elko, Nevada.

Gary B. Wood recently moved from Webster Groves, Mo. to 637 S. Vassar, Wichita 17, Kansas.

Samuel Guze has been appointed an instructor in medicine at the School of Medicine.

1946

George M. Ewing is now located at 881 S. Hotel St., Honolulu, T. H.

The new address of **Mary Bubliss** is c/o Veterans' Hospital, Temple, Tex.

John M. McGrath recently moved from Tulsa, Okla., to 611 N. Main, Athens, Pa.

Sanford C. Snyderman of St. Louis attended the alumni reunion.

1947

Capt. Wm. C. Dunckel, Jr. is now located at 8522 Garland Ave., Takoma Park 12, Md.

Carroll Behrhorst of Alton, Ill. represented his class at the alumni banquet.

Arnold Namrow has been called to active duty by the Navy and is now working with the Psychiatric Screening Unit at the Newport, R. I. Naval Training Station. His address is MOQ KK 11, Naval Training Station, Newport, R. I.

1948

Stanley Schuman is now at the Mid-South Medical Center, P.O. Box 3027, Avondale Sta., Birmingham 6, Ala.

Frank Norbury has moved to 620 Regnault Ave., Gulfport, Miss.

David A. Guterman is now finishing nine months of training which included work in psychiatry at the Illinois Neuropsychiatric Institute, three months of work in child psychiatry at the Institute for Juvenile Research and three months of work in neurology at the I.N.I. His address now is Elgin State Hospital, 750 S. State Hospital, Elgin, Ill.

1949

Harold N. Fogel has moved from Detroit, Mich., to 4512 Main, Kansas City 2, Mo.

Lester H. Margetts, Jr. is now at the Mason Clinic, 1115 Terry Ave., Seattle 1, Wash.

Richard J. Puls' new address is O.S. U. Medical Center 371 W. 10th Ave., Columbus 10, O.

Lt. (j.g.) James D. Mills, Jr., M.C., U.S.N.R., is now stationed at the U. S. Naval Medical Center, Bethesda, Mr.

Robert E. Thomasson, **Chas. M. Lederer** and **Meredith Jorstad Payne**, all of St. Louis, were present at the alumni banquet.

In Memoriam

1880

Joseph M. Long of St. Louis died January 27, 1951 at the age of 95. Dr. Long practiced until 1942 when he retired.

1891

Andrew D. Miller of Sullivan, Ill., passed away December 7, 1949.

1892

John Birkhead Anderson died March 22, 1951, after a long illness. For many years Dr. Anderson served as city health officer at Spokane, Washington. He was commissioned senior surgeon in the U. S. Public Health Service in 1921 and served in several Veterans' Administration Hospitals. He was chief neuro-psychiatrist at Hines (V.A.) Hospital, Chicago and organized the Diagnostic Center at Hines. He was chief of the Center at the time of his retirement in 1938. He is survived by his widow, Sara E. Anderson.

1901

John M. Bradley, Sr., 78 years old, died May 3, 1951 in St. Louis. Dr. Bradley suffered a stroke on the fiftieth anniversary of his graduation from the Medical School. He was an instructor in mental diseases at the Medical School until his retirement about seven years ago. He is survived by his widow, a daughter and a son.

1904

Arthur H. Rohlfing of St. Louis passed away recently.

1906

Andrew C. Henske of St. Louis died recently.

1908

Harry Rich of St. Louis passed away recently.

1912

Roy C. Empson of Valmeyer, Ill. died December 16, 1950.

1919

Harry W. Bond of Wheeling, W. Va., died March 4, 1951 at the age of 59, following an extended illness. He was on the staff of several hospitals in Wheeling until retiring from active practice due to ill health. He is survived by his mother, two brothers and two sisters.

1924

Thomas K. Brown, 52 years old, died of heart disease in Rochester, Minn., April 30. A member of the Medical School staff since 1928, Dr. Brown was in Rochester to give a demonstration for the Minnesota State Medical Society. He was a professor of clinical obstetrics and gynecology at the Medical School, chief of staff at Homer G. Phillips Hospital and a member of the staff at Barnes, City, Children's and Maternity Hospitals. He was noted for his research on puerperal fever. He is survived by his widow, a daughter and a son.

1934

G. J. Nordenbrock of Dallas, Tex. died April 20, 1951.

1942

Caroline Kreiss Pratt of Atlanta, Ga. was accidently drowned in Florida while on a vacation.

WASHINGTON UNIVERSITY

Arthur H. Compton, Ph.D., Sc.D., LL.D., *Bridge Chancellor*
Leslie J. Buchan, Ph.D., *Vice-Chancellor and Dean of Faculties*
Thomas Edward Blackwell, Ph.B., M.S., J.D.,
Vice-Chancellor and Director of Business Administration

- The College of Liberal Arts
Thomas S. Hall, Ph.D., Dean
- The School of Engineering
Lawrence E. Stout, Ph.D., Ch.E., Dean
- The School of Architecture
Joseph D. Murphy, Dean
- The School of Business and Public Administration
R. Miller Upton, Ph.D., Dean
- The George Warren Brown School of Social Work
Benjamin E. Youngdahl, A.M., Dean
- The Henry Shaw School of Botany
Henry N. Andrews, Jr., Ph.D., Dean
- The Graduate School of Arts and Sciences
Carl Tolman, Ph.D., Dean
- The School of Medicine
Robert A. Moore, M.D., Ph.D., Dean
- The School of Law
- The School of Dentistry
Otto W. Brandhorst, D.D.S., Dean
- The School of Nursing
Louise Knapp, R.N., B.S., A.M., Director
- The School of Fine Arts
Kenneth E. Hudson, B.F.A., Dean
- University College
Willis H. Reals, Ph.D., Dean
- The Summer School
Stephen C. Gribble, Ph.D., Director
- The Henry Edwin Sever Institute of Technology
Lawrence E. Stout, Ph.D., Ch.E., Director