

"ASPECTS OF HEART DISEASE IN OLD AGE, 35
WITH SPECIAL REFERENCE TO
HYPERTENSION."

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P R E F A C E

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S U M M A R Y.

INTRODUCTION.

PART I. The Blood Pressure in Old Age.

PART II. The Disabilities Associated with Hypertension.

PART III. The Cardio-vascular System in Old Age.

DISCUSSION.

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INTRODUCTION

(i)

Recently, problems concerning aging and the aged have come to occupy a prominent position in the thoughts of scientific, medical, and social workers. Poets and philosophers have, indeed, from the earliest times been interested in the changes in man's physical and mental outlook which come with advancing years, as part of the mystery of life and death, the eternal and greatest problem of mankind, and in the early literature of many languages are found descriptions comparable with the allegorical trembling of the "keepers of the House" and "bowing" of the "strong men" of Ecclesiastes.

Every society has required to make provision for its aged, and the attitude of different peoples has varied from abandonment among some of the nomad tribes, for example, the Greenland Eskimos and Tasmanians, to extreme veneration and care among certain Oriental races. Our own occidental civilisation, its philosophy partly derived, perhaps, from the Greek adoration of bodily fitness, has tended, in the past, towards vocational and social "abandonment" of the aged and contrasts unfavourably, in this respect, with the deeply ingrained reverence for age shown by the Chinese with their specially accorded privileges to the old. Thus the West emphasises decline in aging, and the East development of maturity.

(ii)

This paradoxical situation, namely, that old age implies both maturity and decline, has been emphasised again and again. George Bernard Shaw's "Back to Methuselah" expresses the view that only after the first hundred years of his life are past, does man gain "sense" to contribute significantly to the progress of humanity. It is not merely a coincidence that pride of place in counsel and government has always been given to the elderly, and without the maturity of the old age of men like Titian, Goethe, Verdi, and Darwin, we should have lost the climax of their life's work. Yet "that un hoped serene which men call Age" has perhaps been dreaded more than it has been extolled. The "sorry breaking-up" of the "immortal age" of Tithonus and Swift's Struldbruggs stand out in vivid contrast to Shaw's conceptions, and there must be few, who perceiving the less attractive aspects of aging, have not at some time echoed Robert Louis Stevenson's words: "Does not life go down with better grace foaming in full flood over a precipice than miserably straggling to its end in sandy deltas?"

Human sympathy, instinctively influenced by economic values, has directed our first sociological experiments towards the preservation and education of the young, and as a very consequence of the new developments in preventive medicine and industrial welfare (coincident with the population/

(iii)

population changes caused by the fall in the birth-rate) we now have among us a greatly increased, and increasing number, of elderly folk. Crew (1946) quotes figures which indicate that between 1861 and 1939, while there was an increase of 63% in the total population of Scotland, the increase in the numbers of persons over 65 years of age was 186.1%, and that of persons under 15 years was, by contrast, 11.4%. According to the Beveridge Report, men over 65 and women over 60 (i.e. those of pensionable age) who in 1941 comprised 12% of the population, will in 1971 comprise 20.8%.

Were the majority of these old people vigorous and well, we might have cause to rejoice in the advantages of their presence among us. But, whether they are well or ill, if they do not support themselves, the burden of their maintenance must fall on those in the so-called productive age-groups. Their needs and desires must be met, and until something is known of the disabilities from which they suffer, their medical requirements cannot be adequately dealt with. Knowledge of geriatrics, also, adds something to the science of gerontology.

The wards of a municipal hospital afford an opportunity of observing some of the infirmities of elderly people. True, the picture is neither balanced nor complete. Surveys like Sheldon's in Wolverhampton give a more accurate description/

description of the capacities of old people, and of the minor ailments to which they are subject. Yet information about some of the diseases causing major incapacity in the elderly is not wholly divorced from the biological changes in senescence which are fundamental to the political, economic, and cultural issues.

My work, then, was based on patients admitted to the medical wards of a municipal hospital. To these wards come a fair proportion of acutely ill patients, and also a great variety of people of all ages belonging to the category of the "chronic sick" as described by Warren (1946). A considerable proportion are over the age of 60, and these are often admitted to hospital, not primarily for medical reasons, but simply because there is no-one to tend them or because the physical, mental, or financial resources of their relatives are not inexhaustible. These patients often remain in hospital, unbefriended by their sons and daughters, until death grants them release from a life which has become narrow, querulous, and essentially selfish.

It was immediately apparent that the number of patients admitted with so-called "myocardial degeneration" or "arteriosclerotic heart disease" was very considerable. Ferguson's figures (1948) for the chief causes of invalidism among old people in Glasgow receiving regular domiciliary nursing care from the Queen's Institute of District Nurses place/

place cardio-vascular troubles high on the list, both in men and in women. While it has been estimated (Dewey, 1942) that, as causes of disability, mental disease and rheumatism probably rank higher than cardio-vascular disease, among my selected group of cases admitted to medical wards with so-called "medical" complaints, as opposed to "surgical" or "mental", cardio-vascular disease seemed very important indeed.

It became clear that the incidence of heightened blood-pressure in my patients over 60 years was very marked, and this led me to examine the records of all the patients, old and young, admitted to the wards between 1937 and 1945, in an endeavour to ascertain how often hypertension occurred in sick people in the various decades.

An examination of the fluctuations of the blood pressure in old people counselled care in the interpretation of these results.

A relatively large number of elderly patients seemed to have asymptomatic and uncomplicated hypertension, and, since I found that histories taken by myself did not differ substantially from the recorded histories of other patients taken routinely in the wards (although details of physical examination did differ markedly), I examined the records of/
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of some of the hypertensive patients admitted to the wards during the years noted above, and contrasted some aspects of hypertension in the old and the young, and in men and women. In addition, I obtained from the records of patients with hemiplegia, some details about prodromal symptoms and prognosis.

Changes in the heart, upsets of cardiac rhythm, and other electrocardiographic abnormalities were found to be related rather to sclerotic changes, as measured by palpation of the limb arteries, than to high blood pressure. Yet not all could be so related.

A very bad prognostic sign for all old patients with hypertension was a sudden progressive lowering of the blood pressure. This brought on clinical symptoms denoting a condition which has been called "progressive cerebral ischaemia", and heralding the approach of death.

In practice there were three main groups of results:-

1. blood pressure readings obtained from a large series of patients
2. an analysis of hypertensive cases
3. a survey of clinical and other findings in elderly patients.

Although results were obtained from these various groups simultaneously, and although the findings sometimes overlap, for the sake of clarity I have kept the three groups/

groups of cases almost entirely separate, and have devoted a Part of the thesis to each group. In Part 1, I have also included a discussion of the nature of hypertension, being a summary of recent experimental work.

"We might be free of an infinity of maladies", said Descartes, "and even of the infirmities of old age, if we had sufficient knowledge of their causes and remedies." It is in the hope that a further statement of the "infirmities of old age" may help ultimately to knowledge of their causes and so their remedies, that the following facts are presented.

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P A R T . 1.

BLOOD - PRESSURE IN OLD AGE.

Chapter 1. The Nature of Hypertension.

Chapter 11. Blood Pressure changes with advancing age.

Chapter 111. The Fluctuations of the Blood Pressure
in Old Age.

CHAPTER 1.

THE NATURE OF HYPERTENSION.

The phenomenon known as raised arterial pressure seems to have been suspected by Richard Bright, who, in 1836, described 52 cases of marked hypertrophy of the left ventricle, in 22 of which there was no obvious lesion such as valvular or aortic disease. He suggested that the cause of the ventricular hypertrophy in those cases might be the altered quality of the blood which "so affects the minute and capillary circulation as to render greater action necessary to force the blood through the distant subdivisions of the vascular system."

Yet though over 50 years have passed since, in 1896, Riva-Rocci introduced his pneumatic cuff for the measurement of blood pressure, we know comparatively little of the causes of arterial hypertension, or of the mechanisms by which it operates. It is part of the symptom complex of diseases as varied in their primary defect as co-arctation of the aorta, pituitary and adrenal dysfunction, and the whole range of kidney diseases. Yet, in spite of Platt's recent demonstration (1947) that there are not many cases of "malignant" hypertension in young persons which are not secondary to some other disease, it probably still remains true that patients with known or suspected causes for their hypertension constitute a small percentage of the total number.

Though arterial hypertension is, like a raised temperature, a sign of disease rather than the disease itself, /

itself, it still remains important to separate "normal" and "high" levels. In this determination the view has gained ground that the diastolic pressure is all-important. Hines, in 1940, suggested that the level of only the diastolic blood pressure is of value in determining whether hypertension will develop in after years. He set as the significant level 85 mm Hg., whereas White and his colleagues (1944-45), in a roughly similar investigation, set their critical level at 90 mm Hg., and stated their belief that elevation of the diastolic blood pressure above 100 mm Hg. was of the greatest prognostic significance.

This belief is in line also with the experimental work which has established that, since in hypertension the cardiac output and the volume and the viscosity of the blood are normal, the fundamental haemodynamic alteration is an increase in the resistance in the peripheral circulation. This increased resistance produces increase in the impelling force in the circulation during cardiac diastole - in other words, the diastolic blood pressure (Goldring and Chasis 1944)

If this be true, then elevation of the systolic blood pressure with a "normal" diastolic blood pressure does not constitute "hypertension." In practice, we find this picture in aortic incompetence, where the valvular insufficiency, allowing reflux of blood into the heart, accounts for the rapid emptying of the arterial system and the fall in diastolic/

diastolic pressure, with its subsequent increased systolic discharge. It is found also in hyperthyroidism, where excessive vasodilatation causes excessive emptying of the arterial system during diastole, and in complete heart-block, where the slow cardiac rate compels an increased cardiac output. But it has also been described as occurring in a particular group of people characterised by their age. Fahr et al. (1932) and Wiggers (1932) have suggested that the development of this so-called "Systolic Hypertension" is compensatory to the loss of elasticity that occurs in aging arterial walls. In health the large arteries are stretched during cardiac systole, and the elastic recoil during diastole helps to maintain the diastolic blood pressure and the peripheral blood flow. If the arteries become more rigid, the "accessory pump" is less effective and there must be a higher systolic blood pressure to maintain the peripheral blood flow.

The systolic pressure in diastolic hypertension is also influenced by structural and functional changes in the arterial walls. Elevation of the diastolic blood pressure in hypertensive patients is usually accompanied by an exalted elevation of the systolic blood pressure, resulting in increased pulse pressure. How fundamental is this change in the pulse pressure was shown when Bradley and Parker, in 1941, injected hypertensin into a normal man, and produced an increase in systolic and diastolic blood pressure with widening of the pulse/

pulse pressure. Wiggers (1940) summarising the available experimental and clinical evidence, believed that in clinical hypertension the circulatory changes were the result of the combined effects of the increased peripheral resistance and decreased elasticity of the aorta. In 1942, he suggested that elevation of the diastolic pressure acts not only on the arterioles, but also causes contraction of the walls of the larger arteries with changes in aortic capacity and elasticity, to which the increase in pulse pressure is due. Steele (1937), on the other hand, believed that the increased pressure in the aorta itself produces changes in aortic distensibility. Either concept might explain the increased pulse pressure and, if sclerotic changes are added, widening of the pulse pressure could be attributed to this anatomical change.

Pickering in 1939 and Oppenheimer and Prinzmetal in 1937 have summarised the evidence by which, from measurements of the intravascular pressure at various points along the vascular tree, the conclusion has been reached that the increased peripheral resistance, referred to above as the cause of increased diastolic blood pressure, is confined to the arteriolar bed. Subsequent experiments by Goldring and Chasis, and their colleagues, using new methods of estimating cardiac output, have but served to confirm this hypothesis.

Pickering (1943), in his review of the circulation in hypertension, /

hypertension, showed that in essential hypertension the vasoconstriction is generalised, but probably more severe in the kidney than elsewhere. He showed, moreover, that the circulatory changes are not quite the same in the various diseases in which hypertension occurs. In the one type - essential hypertension, chronic nephritis, coarctation of the aorta - the hypertension is due to arteriolar constriction. In acute nephritis, on the other hand, constriction has not been demonstrated in the vessels either of the hand or of the kidney. Furthermore, Pickering showed that while adrenaline is not concerned in the production of hypertension as above, it is directly concerned in the production of paroxysmal hypertension in pheochromocytoma of the adrenal medulla.

Fatty hyaline thickening of the intima in the afferent glomerular arterioles occurring commonly in essential hypertension - though now shown not to be the cause of hypertension, and to occur in the arterioles of other organs and in elderly people with normal blood pressure - led Goldblatt to perform his classical experiments (published in 1934) in which he showed that the production of renal ischaemia in the dog led to the development of hypertension. By clamping both renal arteries a permanent hypertension could be produced, with arteriolar changes throughout/

throughout the body, but not in the kidneys. Later, in 1939, Wilson and Byrom showed that, in the rat, permanent hypertension could be produced by clamping one kidney, and that if, after changes resembling those of malignant hypertension in man were well established, the clamped kidney was removed, the hypertension persisted and increased, showing that renal ischaemia had been established in the second kidney. This is analogous to the findings of Ellis (1938) that in malignant hypertension acute fibrinoid necrosis and endarteritis of the arterioles of the kidney and other organs (the necrosing arteriolitis of Fahr) is minimal in the early stages and severe in the later stages. The "arteriolitis" is an effect of hypertension, and not its cause, and this explains why different renal disorders, in the late stages, show similar clinical and histological pictures, for the factor common to all is seen to be renal ischaemia leading to hypertension and so to further vascular lesions and more renal ischaemia.

As a result of their work in elaborating new chemical methods for the accurate estimation of renal function, and investigating, by such methods, 60 cases of essential hypertension in various degrees of severity, Homer Smith and his colleagues (1941) have demonstrated that renal ischaemia is the result of increased resistance, beyond the glomeruli, in the efferent glomerular arterioles.

It/

It seems not unlikely that in chronic kidney diseases - pyelonephritis, hydronephrosis, renal tuberculosis and congenital cystic kidney - the mechanism of the production of the hypertension may be the same as in experimental renal hypertension of animals, i.e. the result of renal ischaemia, and this supposition has been strengthened by the discovery of a few cases of hypertension which have been associated with unilateral renal disease and cured by removal of the diseased kidney.

In essential hypertension, the direct connection with renal ischaemia is not so immediately clear. Ellis, in the Croonian lectures, published in the Lancet in 1942, stated; "The evidence that essential hypertension is due to primary renal vascular lesions is at present insufficient. It is quite possible that the mechanism of production of essential hypertension is renal ischaemia, but if this is so, it seems more probable that it is first functional, due to some extra-renal factor, and only later aggravated by renal arteriolo-sclerosis."

Further evidence that the arteriolar lesion is probably at first functional, is provided by Castleman and Smithwick (1943), who examined renal biopsies from 100 patients with well-established hypertension, and found that in 28% of the patients there was no vascular disease, and in another 25% only mild changes. Moreover Prinzmetal and Wilson (1936) have/

have shown that in hypertension the peripheral vessels can dilate in response to heat. This is in keeping, too, with the clinical finding that in essential hypertension, the blood pressure readings are labile and only become fixed when malignant changes supervene i.e. when renal arteriolitis becomes evident.

Much experimental evidence exists to show that the mechanism by which this vasoconstriction is produced is humoral and not neural. For instance, Page (1935) showed that the experimental hypertension of renal ischaemia could not be relieved by denervation of the kidney, and that it could be produced even in animals where the kidneys had previously been denervated. Blalock and Levy (1937) showed that hypertension could be produced even by an ischaemic kidney transplanted into the neck or groin, and Goldblatt, in the same year, showed that no rise of blood pressure was produced in the experimental animal if the renal vein was obstructed at the same time as the renal artery. In 1943, came Pickering's experiments demonstrating that the changes in the circulation in essential hypertension are different from those produced by neural vasoconstriction, where the skin also is depleted of blood.

In 1898, Tigerstedt and Bergmann first extracted from renal cortex a heat-labile, non-dialysable, pressor compound. During the next 40 years, however, only Bingle and Strauss (1909) were able to reproduce the observations of Tigerstedt and Bergmann. Goldblatt's experiments in 1934 once more aroused interest in renin. In 1936, the hypertensive effect of extracts of ischaemic kidney were observed independently by (1) Harrison, Blalock and Mason, and (2) Prinzmetal and Friedmann. In 1938, after much work on kidney press juices, autolysates and extracts, purification of renin was attained by two groups of workers, one in North and one in South America.

Renin is an enzyme stored, or formed, in the proximal convoluted tubules, and liberated from the renal cortex into the blood stream. Here it reacts with a - globulin in the plasma - 'hypertensinogen' (Braun-Menendez and colleagues) or 'angiotonin activator' (Page and Helmer) - to form a thermo-stable, dialysable, pressor compound - 'hypertensin' or 'angiotonin'. There is evidence also of the presence of a hypertensinase, an enzyme which destroys the activity of hypertensin, and it may be that the hypertensive subject is deficient by being less capable of destroying pressor substances, since in experimental animals it has been shown that the degree of hypertension after renal ischaemia is less when the animal possesses one normal kidney.

Now/

Now in man, the action of renin has not been fully investigated, because animal renin is inactive in man and human kidneys are a poor source of renin. But Helmer and Page, having produced hypertensin in a form suitable for injection in man, its effects were investigated by various groups of workers in 1941, and it was shown that, although other pressor agents such as adrenaline and tyramine as well as renin and hypertensin produce constriction of the efferent glomerular arterioles of the kidney, only renin and its product, hypertensin, produce hypertension without so constricting the vessels of the skin that much blood is diverted from it. Thus they are the only agents known which produce the effects of essential hypertension.

In man, so far, no correlation has yet been achieved between hypertension and hyperactivity of the renin pressor mechanism, and it may be that some forms of hypertension may have to be explained in a different way. Yet, at the same time, the renin hypothesis seems the most likely, taking into account the strong indications of a chemical mechanism in hypertension, with the appropriate physiological effects of renin on the circulation.

The initial stimulus producing renal ischaemia - Ellis's extra-renal factor - has not yet been found. Recent work on the renal circulation supposes that in hypertension there is a chronic "shunt" diverting blood to the tubules from the glomeruli, /

glomeruli, producing cortical ischaemia. This cortical ischaemia, it is considered, may be the stimulus for renin production. This work is still unconfirmed, and how far it is true with respect to hypertension, is still to be seen.

It should not, however, be forgotten that endocrine and nervous factors play a part in the regulation of normal blood pressure, and it is not impossible that abnormality of their function may have some influence on the production of hypertension in disease. For instance, although Pickering's experiments indicate that in essential hypertension the circulation differs from the vasoconstriction produced by the adrenal and posterior pituitary hormones, Page and Sweet have shown that the removal of the suprarenals and hypophysis in experimental animals reduces the level of the blood pressure. There is some evidence, moreover, that the suprarenal cortex may play some part in maintaining the production of hypertensinogen.

We have seen that it is unlikely that the vasoconstriction which underlies essential hypertension is of nervous origin, and Pickering has shown that only in acute nephritis does it appear possible that over-action of the vasomotor nerves might have to do with the origin of the hypertension. Yet it is along the lines of nervous control of hypertension that explanations of the changes in blood pressure with age have so far been made. Two different mechanisms have been advanced/

advanced to indicate the way in which the central nervous system produces overactivity of the sympathetic system, and so vasoconstriction. The first rests on the fact that in 1927 Koch, Mies and Nordmann demonstrated that in the experimental animal a persistent hypertension could be produced by resection, or bilateral denervation, of both carotid sinuses and section of the aortic depressor nerves. Pickering, Rothschild, and Kissin (1936), however, showed that in essential hypertension the carotid sinus mechanism was still active. The second view was first put forward by Cushing, in 1902, who concluded, as a result of his experiments in raising the intracranial pressure above the diastolic blood pressure, that blood pressure regulation is a function of the medullary vasomotor centres, set in action by the anaemia induced in elevation of the intracranial pressure above the diastolic blood pressure. Later it was found that injection of kaolin into the cisterna magna to increase the intracranial pressure, produced the so-called "kaolin hypertension." Arteriosclerotic changes producing diminution of the cerebral blood flow are said to produce analogous changes, and the recent experiments of Fishback (1943) would support this suggestion. Stimulation of certain areas of the hypothalamus (Ranson and Magoun) 1939, and of discrete portions of the cerebral cortex (Hott and Green) 1936 have also been shown to produce hypertension.

But/

But whatever may be the intimate nature of the pressor mechanism, its activation in the individual is largely determined by factors of heredity and environment. For instance, Ayman (1934) found that essential hypertension was much more common in the children of hypertensive parents. Examining 1,524 members of 277 families, he found that where neither parent had hypertension 3.1% of the children had hypertension, but where one parent and two parents, respectively, had hypertension, 28.3% and 45.5% of the children had hypertension. This family predisposition has also been commented on by O'Hare, Walkers, and Vickers (1924), and by many others. Platt (1947) believes that the tendency to hypertension is a Mendelian dominant with a rate of expression of 90%. Thus it is not surprising that the frequency of hypertension varies in different races, being particularly high, for instance, in the American negro.

Lastly, the influence of psychogenic factors - stresses and strains, whether environmental or due to personality traits and habits must be considered. Moschowitz (1929) described a definite type of person with certain physical and psychic complexes, in which essential hypertension was apt to occur. The effect of anxiety and strong emotional upsets on the blood pressure has often been caricatured in/

in the lay press, where the strong rages of the man with the terrible temper are ready to pull the trigger which will send up his blood pressure, so that each moment becomes, for himself and his associates, a kind of "imperilled eternity." Alexander and Saul (1939) however, emphasise the importance of repressed aggressions and anxieties. But even Fahrenkamp writing in Dunbar's book "Emotional and Bodily Changes" and believing that psychic factors play a predominant role, does not accept the existence of functional hypertension of purely psychic origin.

Conclusions.

Arterial hypertension is a sign of disease. It occurs in several different types of diseases, and can be produced in various ways.

Systolic hypertension is to be differentiated from conditions where the diastolic pressure is also elevated.

In essential hypertension, as in chronic nephritis and other kidney diseases (except acute nephritis), renal ischaemia, leading through a humeral mechanism to a generalised increase of peripheral resistance, is the most probable cause of hypertension. In essential hypertension, the ischaemia is at first functional, the primary stimulus being probably extra-renal, but later organic changes are produced which aggravate the ischaemia so that the blood pressure eventually becomes/

becomes fixed at a high level.

In the present state of our knowledge, nervous factors cannot be wholly excluded.

Heredity seems to play a large part in the production of hypertension, and repressed aggressions and anxieties also seem to act as aggravating, if not causal, factors.

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CHAPTER II.

BLOOD PRESSURE CHANGES WITH ADVANCING AGE.

- Section (1) Introduction.
 - Section (2) Systolic Blood Pressure.
 - Section (3) Diastolic Blood Pressure.
 - Section (4) Pulse Pressure.
 - Section (5) Systolic Hypertension.
 - Section (6) Discussion.
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SECTION (1)

I N T R O D U C T I O N .

That a high blood pressure was a frequent finding in my elderly patients was apparent from even the most cursory survey of routine general examinations. A more detailed examination of the incidence of hypertension at various age-groups seemed to be indicated as the first step towards the determination of its part in the production of sickness in old age.

The results of several surveys were found in the literature. As long ago as 1917, Bowes found among 150 patients, whose ages ranged from 65 to 94 years, 22 who had a systolic blood pressure above 200 mm mercury (Hg), 28 with a diastolic blood pressure over 100 mm Hg., and 30 with a pulse pressure greater than 100 mm Hg. In 1930, Blackford, Bowers, and Baker, examining the records of 17,500 patients, found that the 401 cases of hypertension, defined by them as those patients having a systolic blood pressure over 175 mm Hg., were distributed so that while of patients under 40 years of age only 2.2% had hypertension, the proportion increased till it reached a maximum of 42.6% hypertensives among patients between 60 and 70 years.

In 1931 Willius, and in 1932 Willius and Smith published the results of examination of 700 persons over 75 years of age and 381 over 70 respectively. They found the majority to have a systolic blood pressure over 140 mm Hg., and a pulse pressure of over 60 mm Hg., while 40% had a diastolic pressure of over 90 mm Hg. Howell, examining in 1942 120 Chelsea Pensioners aged 65 - 92, found that 50 had a systolic blood pressure greater than 160 mm Hg.

Later surveys, involving larger numbers of patients and being perhaps wider in their scope, take both systolic and diastolic pressures into account. Masters et al. (1943) examined 15,000 men and women over 40 years of age. They claimed that their material was representative of the general population, and all their blood pressure readings were taken according to the standard method laid down in 1939 by the American Heart Society and the Cardiac Association of Great Britain and Ireland. They made seven different definitions of high blood pressure, but when the significant levels were taken as 150 systolic and 100 diastolic, more than 30% of the men aged 50 - 59, and more than one half of the men over 60 had hypertension, while for women the incidence was higher still. In the same year Russek examined a group of 1,000 retired seamen, aged 60 - 95 years, and in 1946 Russek, Rath, Zohman, and Miller, /

Miller, publishing the results of this (already published) and other enquiries (all in persons in reasonably good health) found a steady progressive decrease in incidence of "normal" blood pressure (defined as below 150 systolic and 95 diastolic) from 87.2% in 40 - 44 age-group to 27.8% in 85 - 95 age-group.

Perusal of the literature thus showed that in general it could be said that the incidence of high blood pressure increased as age advanced. In addition it became clear that there were three possible sources of confusion in the interpretation of the results of a particular survey. Firstly, the method of taking blood pressure readings had to be stated; secondly, the population from which the sample had been drawn had to be defined; and thirdly, the dividing line between "high" and "normal" blood pressure had to be determined.

1. Method of taking blood pressure readings.

I cannot claim that the blood pressure readings listed below form an ideal basis for study. They are imperfect because taken from hospital records and made by different observers. I have shown above what led me to examine the case-records of all patients in six medical wards during 1937 - 45. The readings selected were those taken several days after the admission of the patient, and from series of daily readings the lowest one was chosen. Despite their/

their defects, however, I have set them down as an introduction to, or perhaps a statement of, the problem which I have tried in subsequent studies to elucidate.

II. Population.

In the introduction I have described the patients from whom these readings were taken. If we assume that hypertension causes disabilities which bring people to the medical wards of a hospital, we may infer that among my cases there is a higher - perhaps much higher - proportion of hypertensives than would be found among the general population. These figures in their absolute sense are therefore not comparable with those from healthy groups.

III. "Normal" and "High" Blood Pressure.

To determine where the dividing line should be drawn is difficult, and made more so because it is by no means certain that what is abnormal in youth is also abnormal in old age.

It was shown originally by Bowes (1917) and Lewis (1938) (Lewis's cases, though small in number, being carefully examined under standardised conditions) that the "average" systolic pressure and pulse pressure increased with advancing age, the diastolic pressure remaining relatively constant. This view was confirmed by the larger/

larger series of Miller (1941), Howell (1942), Russek (1943), and Russek et al. (1946), these last-named workers coming to the conclusion that the old maxim (100 + age in years) may actually be a fair index of normal blood pressure.

Some, however, would not concede this relationship between "normal" and "average." Alvarez and Stanley (1930), though the average blood pressure in their older patients was higher than in the younger, considered the most frequent pressure for each age-group to be the important figure, and believed that a pressure of 140 mm Hg. was as abnormal in an old man as in a young one. Robinson and Brucer (1939), though doubt has been thrown on the validity of their statistical calculation (Freloar 1940), also took 140 mm Hg. as their ceiling level for "normal" systolic pressure, and discarding all pressures above that, calculated that the mean level of blood pressure for all ages became the same.

Russek et al. (1946), however, analysing their figures for "normal" blood pressure, which comprised those patients whose systolic pressure was below 150 mm Hg. and diastolic below 95 mm Hg., found a big increase in the frequency of systolic pressures between 140 and 149/

149 - from 18.6% in the age-group 40 - 49, to 40.9% between 80 and 95, and concluded that the upper limit of normality for systolic blood pressure is a figure considerably above 140 mm Hg.

From the literature available to me, I have compiled table 1. showing the levels of average blood pressure in different age-groups, above the age of 60 years as determined by various investigators. With these figures before me, I at first chose 150 mm Hg. as the significant level for systolic blood pressure, and divided the range of systolic pressure from 100 mm Hg. - 250 mm Hg. into groups of 25 mm Hg., in this way placing each patient in his appropriate age and systolic pressure group. Later, however, as my examination of old people proceeded, I considered that 150 mm Hg. was too low a level, and that some "normal" people were thereby being included among the hypertensive group. Eventually, therefore, I fixed a significant level for my own purposes at 160 mm Hg.

Similarly, as regards the diastolic pressure, with the object of including abnormal patients only, I set the level of "high" diastolic pressure at 100 mm Hg. though this is much higher than the level suggested by Hines (1940) or White and his co-workers (1944 & 1945).

By/

TABLE 1.

AGE AND BLOOD PRESSURE

Age	S.B.P./ D.B.P. in mm Hg.						
yrs.	Bowes 1917	Lewis 1938	Robinson & Brucer 1939	Miller 1941		Howell 1942	Russek 1943
				M.	F.		
60-64	-	124/83	-	142/86	156/84	-	147/82
65-69	151/82	134/75	139/75	146/84	174/93	149/80	153/86
70-74	160/86	141/75	137/77	148/84	161/88	157/83	154/85
75-79	166/86	153/76	154/80	154/84	165/90	166/87	156/86
80-84	175/84	154/85	-147/87	154/85	176/94	158/84	157/85
85-89	170/90	158/76		157/86	145/83	-147/79	160/86
Over 90	142/81	147/78	-	-	158/75		-
" 100	-	136/75	-	-	-	-	-

By means of a series of charts, a diminutive specimen of one of which is attached below, patients were separated according to their sex, age, systolic and diastolic pressures, and pulse pressures, and eventually from these figures a series of tables was compiled. These tables are appended below.

SPECIMEN OF CHART:

Age Group Sex Systolic B.P..... mm Hg.

Diastolic B.P. mm Hg.	< 90			91 - 99			> 100		
Pulse Pressure mm Hg.	<40	40-80	>80	<40	40-80	>80	<40	40-80	>80

Table 2 shows the number of men and women in each age - group.

TABLE 2.

NUMBER OF PATIENTS IN EACH AGE-GROUP

	Age - Groups								Total
	20	20-39	40-49	50-59	60-69	70-79	80-89	90-99	
MALE	190	388	348	406	607	528	92	7	2566
FEMALE	199	504	244	353	511	335	114	20	2280

SECTION (2).

Systolic Blood Pressure.

Tables 3 A. and B. show the range of systolic pressure from under 100 mm of mercury to over 250 mm of mercury, divided into groups of 25 mm. Hg.

Certain points emerge from a study of these two tables:-

- (1) Among men, in the age-groups below 50 years, the greatest number of patients was found in the group having a systolic blood pressure between 101 and 125 mm Hg. In the other groups, that is, in each decade from 50 onwards, the greatest number was between 126 and 150 mm Hg.
- (2) Among women, in the age-groups below 40 years, the systolic pressure most commonly fell between 101 and 125 mm Hg. and in each decade after 40, between 126 and 150 mm Hg.
- (3) There was a very small number of persons aged 90 years and over, but in contrast to the statement of Richter (1925) that a very low blood pressure is frequently seen in very old people, the proportion of men and women with a relatively high blood pressure was quite considerable.
- (4)/

TABLE 3.

SYSTOLIC BLOOD PRESSURE - LEVELS IN DIFFERENT AGE GROUPS.

A. MALES:

S.B.P. mm Hg.	Age of patient in years							
	20 -	20-39	40-49	50-59	60-69	70-79	80-89	90-99
Under 100	38	31	28	25	33	27	-	-
101-125	112	206	143	129	124	78	12	2
126-150	40	134	135	151	190	159	28	2
151-175	-	11	18	41	100	114	19	-
176-200	-	3	13	31	95	91	27	2
201-225	-	-	7	15	40	39	7	1
226-250	-	2	3	10	21	19	-	-
Over 250	-	1	1	4	4	1	-	-
TOTAL	190	388	348	406	607	528	92	7
*Total % age above 150	Nil	4.4	12.1	24.8	42.9	51.7	56.6	

B. FEMALES:

S.B.P. mm Hg.	Age of patient in years							
	20 -	20-39	40-49	50-59	60-69	70-79	80-89	90-99
Under 100	35	62	14	15	30	7	-	-
101-125	138	261	69	86	65	31	19	-
126-150	25	151	100	104	147	97	32	5
151-175	-	11	23	59	101	85	29	3
176-200	-	8	20	44	83	62	26	12
201-225	1	9	13	16	45	27	-	-
226-250	-	2	4	18	30	18	7	-
Over 250	-	-	1	11	10	8	1	-
TOTAL	199	504	244	353	511	335	114	20
* Total % age above 150	0.5	6.0	24.9	41.9	52.7	59.8	55.6	

* expressed as a percentage of the total number of patients in the age group.

(4) Choosing arbitrarily, for the reasons given above, 150 mm Hg. as a significant level, we find that in both men and women the proportion of the total number of patients in each age-group who had hypertension increased with each successive decade, until the age-group 70-79 was reached, and indeed, these differences were statistically significant,

(5) In the 40's, 50's, 60's, and 70's, the proportions of women having hypertension were significantly larger than those of men.

SECTION (3).

Diastolic Blood Pressure

Tables 4 A and B show the numbers of men and women whose diastolic blood pressure was (a) lower than 90 mm Hg., (b) between 91 and 99 mm Hg., and (c) above 100 mm Hg.

(1) Among men, until the age of 60 years, the proportions of patients with diastolic hypertension increased significantly with each decade.

(2) The same was true of women until the age of 50 years (i.e. a decade earlier than men).

(3) After these ages (i.e. 60 years in men and 50 years in women), the proportions in each group were approximately the same.

(4) /

TABLE 4.

DIASTOLIC BLOOD PRESSURE - LEVELS IN DIFFERENT AGE GROUPS.

A. MALES:

D.B.P. mm Hg.	<20	Age of patient in years						
		20-39	40-49	50-59	60-69	70-79	80-89	90-99
Under 90	187	361	288	286	374	339	56	4
91 - 99	1	15	26	36	39	27	15	-
Over 100	2	12	34	83	194	162	21	3
TOTAL	190	388	348	405	607	528	92	7
* Total % above 100	1.05	3.09	9.77	20.49	31.96	30.68	22.82	42.85

B. FEMALES:

D.B.P. mm Hg.	< 20	Age of patient in years						
		20-39	40-49	50-59	60-69	70-79	80-89	90-99
Under 90	194	450	175	209	307	184	69	14
91 - 99	2	18	24	22	29	34	7	5
Over 100	3	36	45	122	175	117	38	1
TOTAL	199	504	244	353	511	335	114	20
* Total % above 100	1.50	7.14	18.44	34.56	34.25	34.93	33.34	0.20

* Expressed as a percentage of the total number of patients in the age group.

(4) Up to the age of 60 years, a significantly larger number of women than of men had hypertension. Over the age of 60, there was no significant difference between the two sexes.

SECTION (4).

Pulse Pressure.

It will be realised, if the findings of the changes in systolic and diastolic pressures are compared, that there was a difference in their behaviour as age advanced. After the age of 60 in men and 50 in women, the proportion of patients with a high diastolic pressure remained constant, while that of patients with a high systolic pressure continued to increase.

This change can be shown more clearly by examining the pulse pressure. Each patient had been charted as having a pulse pressure of either below 40 mm Hg., between 40 and 80 mm Hg., or over 80 mm Hg. It is realised now that those levels are not well chosen, for a patient might have a blood pressure whose systolic level was over 150 or 160, and diastolic under 100, yet not come into the category of those whose pulse pressure is greater than 80 mm Hg. Yet I have set down the appropriate figures because I wished rather to show the trend of events than to lay down absolute criteria, which I did not feel justified in drawing from my particular data. /

data.

From Tables 5 A & B, then, we obtain the following information:-

1. In men, as age advances there is a gradually increasing proportion of patients with a pulse pressure greater than 80 mm Hg., the difference between the proportions in the age groups 40-49 and 50-59, 50-59 and 60-69, and 60-69 and 70-79 being significant.
2. In women, there is also a gradually increasing proportion of patients with high pulse pressure, but here the increase is not significant after the age of 60 years.
3. In the forties, fifties, and sixties, a high pulse pressure occurs significantly more often in women than in men.

In Tables 6 A & B, I have set down the number of patients with a pulse pressure over 80 mm Hg., according to whether they had a diastolic pressure of below 99 mm Hg. or above 100 mm Hg., and I have compared them with the total number of patients (and the number in each age-group) whose diastolic pressure fell within these limits.

1. Among patients with a diastolic pressure below 99 mm Hg., in all 2055 men and 1741 women, 224 men (10.9%) and 216 women (12.4%) had a pulse pressure over 80 mm Hg.

Among the diastolic hypertensives, in all 510 men and 539 women, 261 men (51.2%) and 278 women (51.6%) had a pulse pressure of above 80 mm Hg.

TABLE 5.HIGH PULSE PRESSURE - INCIDENCE IN VARIOUS AGE GROUPS.A. MALES

mm Hg.	20	20-39	40-49	50-59	60-69	70-79	80-89
Under 40	99	160	136	118	135	86	7
41 - 79	85	211	186	221	311	270	51
Over 80	6	17	26	66	161	172	34
TOTAL	190	388	348	405	607	528	92
%AGE above 80	3.2	4.4	7.5	16.3	26.5	32.2	36.0

B. FEMALES

mm Hg.	20	20-39	40-49	50-59	60-69	70-79	80-89
Under 40	123	239	43	90	71	32	9
41 - 79	72	249	155	177	272	179	58
Over 80	4	16	46	86	168	124	47
TOTAL	199	504	244	353	511	335	114
%AGE above 80	2.0	3.2	18.8	24.3	32.9	37.0	41.3

TABLE 6. DIASTOLIC PRESSURE IN PATIENTS WITH HIGH PULSE PRESSURE

A. MALES:

(1) Low D.B.P. (under 99 mm Hg.)

	20	20-39	40-49	Age-groups		70-79	80-89	90-99	TOTAL
				50-59	60-69				
No. high PP	6	14	16	16	60	86	25	1	224
Total with low D.B.P.	188	376	314	323	413	366	71	4	2055
% age with high P.P.	3.2	3.7	5.1	4.9	14.5	23.5	35.2	25.0	10.9

(2) High D.B.P. (over 100 mm Hg.)

	20	20-39	40-49	Age-groups		70-79	80-89	90-99	TOTAL
				50-59	60-69				
No. high PP	-	3	10	50	101	86	9	2	261
Total with high D.B.P.	2	12	34	82	194	162	21	3	510
% age with high P.P.	0	25	29.4	60.9	52.1	53.1	42.9	66.7	51.2

B. FEMALES:

(1) Low D.B.P. (under 99 mm Hg.)

	20	20-39	40-49	Age-Groups		70-79	80-89	90-99	TOTAL
				50-59	60-69				
No. high PP	3	7	26	28	71	59	19	3	216
Total with high D.B.P.	196	468	199	231	336	216	76	19	1741
% age with high P.P.	1.5	1.5	13.1	12.1	21.1	27.3	25.0	15.8	12.4

(2) High D.B.P. (over 100 mm Hg.)

	20	20-39	40-49	Age-Groups		70-79	80-89	90-99	TOTAL
				50-59	60-69				
No. high PP	1	9	20	58	97	65	28	-	278
Total with high D.B.P.	3	36	45	122	175	119	38	1	539
% age with high P.P.	33.0	25.0	44.4	47.5	55.4	54.6	73.7	-	51.6

There is a significant difference in the proportions between the two above groups.

2. Amongst those whose diastolic pressure is under 99 mm Hg., the proportion who have a pulse pressure greater than 80 mm Hg. increases with each decade, both in men and in women.

We see then that in diastolic hypertension, a pulse pressure of over 80 mm Hg. is not very uncommon at any age; nor does the advent of age necessarily induce an increased number of diastolic hypertensives to develop a large pulse pressure. Among patients with a low diastolic pressure, however, a proportion, much lower initially but constantly becoming larger with each decade, have a pulse pressure greater than 80 mm Hg.

SECTION (5).

Systolic Hypertension.

In Tables 7 A & B are set down the number of patients in each age-group whose systolic pressure was greater than 150 mm Hg., yet whose diastolic pressure was less than 100 mm Hg. I have referred above to the various conditions associated in practice with elevation of the systolic pressure and a normal diastolic pressure. When, however, the number of patients with (a) Aortic Incompetence, and
. (b) /

TABLE 7.

SYSTOLIC HYPERTENSION - The number of patients in each age-group with B.P.>150/<100. Also the incidence (for comparison) of Aortic Incompetence and Hyperthyroidism.

A. MALES:

	Age of patient in years							
	< 20	20-39	40-49	50-59	60-69	70-79	80-89	90-99
Systolic Hypertension	-	6	19	33	85	111	33	-
Aortic Incompetence	-	-	5	3	14	1	-	-
Hyperthyroidism	-	7	2	1	1	-	-	-
Total number of cases	190	388	348	405	607	528	92	7
% patients with S.H.	-	1.55	5.46	8.15	14.01	21.02	35.87	0

B. FEMALES:

	Age of patient in years							
	< 20	20-39	40-49	50-59	60-69	70-79	80-89	90-99
Systolic Hypertension	-	9	21	40	102	91	25	4
Aortic Incompetence	-	1	2	2	-	-	-	-
Hyperthyroidism	2	15	14	4	1	1	-	-
Total number of cases	199	504	244	353	511	335	114	20
% patients with S.H.	-	1.79	8.61	11.33	19.96	27.16	21.93	69.98

(b) Hyperthyroidism is compared with the total number of patients with systolic hypertension, it is evident that among my patients they represent only a small proportion of the whole, and that over the age of 50, even if we deduct the total number of patients with these two conditions, there would not be any marked change in the incidence of systolic hypertension.

There is, with each decade after 50, a significant increase in the proportion of patients with "systolic hypertension", except, for some reason, in women between the 70-79 and the 80-89 age-groups. Furthermore, in the fifth, sixth, and seventh decades, this "systolic hypertension" occurs significantly more often in women than in men, but in the eighth decade the finding is reversed and men predominate. Russek (1943) and Russek and co-workers (1946), in their surveys (whose populations have already been described) found a progressive increase in the incidence of systolic hypertension, (which they defined as a systolic blood pressure greater than 150 mm Hg. with a diastolic blood pressure less than 95 mm Hg.). For instance, in the 1946 survey, of those between 40 and 44 4.2% had systolic hypertension and 8.5% had diastolic hypertension, while in 85-95 age-group, 45.2% had systolic hypertension and 27% had diastolic hypertension.

Fineberg (1927) described a certain group of patients with elevated systolic pressure and relatively low diastolic pressure. They were of advanced age (38 being over 80 years), and, compared with diastolic hypertensives, they were less likely to develop cardiac failure. More important, perhaps, was that they had a much more favourable life expectancy. Russek (1943) confirmed Fineberg's findings with regard to prognosis, and showed that his cases of systolic hypertension had a prognosis relatively similar to that of patients with a normal blood pressure and very different from the prognosis of diastolic hypertensives.

I have already indicated above that Fahr et al. (1932) and Wiggers (1932) believed that systolic hypertension developed as a result of loss of elasticity and increased rigidity of the walls of the great vessels. Fahr and Davis (1930) have shown that increased rigidity of the arterial system, by itself, does not impose any added burden on the heart, and, though Wiggers (1938) thought that systolic hypertension might represent long-standing diastolic hypertension complicated by sclerosis of the aorta, systolic hypertension with marked arteriosclerosis of the aorta is frequently associated with a small heart.

Finally, the much more favourable life expectancy of systolic hypertensives would support the view that they form an independent entity.

SECTION (6).

DISCUSSION.

It has been shown that with advance in age, there is a marked decrease in the proportion of patients with a "normal" blood pressure. There is also, with advance in age, a marked progressive increase in the proportion of those with systolic hypertension. This systolic hypertension is believed to be due to changes in the elasticity of the aorta, and is not "true" hypertension.

There is, however, also an increase in the incidence of diastolic hypertension with age. This increase occurs during middle life up to the age of 60 years in men and 50 years in women, but after these ages the incidence remains at a constant high level. This may be due to the fact that diastolic hypertensives are much less long-lived, and that those who have diastolic hypertension at 60 are unlikely to live long enough to be also among the community at 80. It seems, then, that in considering the etiology of diastolic hypertension we cannot wholly neglect this factor of age.

The cold-pressor test of Hines and Brown (1936) was elaborated, and has been widely used, as a test to determine vascular responsiveness to a pressor stimulus - in this case, immersion of the hand in ice-cold water for one minute. Hines believes that hyperreactivity of the vessels represents an antecedent or latent phase of essential hypertension. But, Russek/

Russek (1943), using the cold-pressor test, has demonstrated that the incidence of vascular reactivity increases progressively as age advances.

This increase in vasopressor response with age is attributed by Raab (1941) to increasing irritability of the cerebro-medullary vasoconstrictor centres - a consequence of cerebral ischaemia due to arterio-sclerotic changes. Raab has suggested that this increased central vasomotor irritability might contribute to the vasoconstriction which results in hypertension.

Critchley (1942) has shown that much senile behaviour is due, not to a vague "cerebral arterio-sclerosis", but, to disseminated cortical atrophy of the cerebrum. Dock (1945) has suggested that this trophic loss of neurones might account for, not only the tremors and rigidity of senescence, but also the increased amount of diastolic hypertension in old age. Analogous changes, he thought, might also account for the increasing incidence with age of cold-pressor test hyper-reactors.

Both these theories of the causation of diastolic hypertension are based, as I have already indicated, on the assumption that the vasoconstriction of the arterioles, which causes hypertension, is mediated by nervous means. However, we have already seen that, on the basis of our present knowledge, this assumption is not likely to be correct.

If/

If we accept renal ischaemia as being the cause of the hypertension, then renal ischaemia and the renin mechanism must be more easily produced and set in motion as age advances. Mosenthal (1941) has suggested the presence of some unknown factor in the aging kidney.

Whatever may be the reason for it, there was no doubt about the large number of hypertensives amongst the group covered by the survey. If hypertension causes sickness, then, here is an important cause of sickness in elderly people.

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It is a well-known fact that variations occur constantly in the blood pressure levels of normal individuals - often from minute to minute, and sometimes from hour to hour. The fall in blood pressure on standing, the increase on eating or with muscular effort, the diurnal variations - all these are well recognised. Every physician is aware of the rise in blood pressure which occurs with an emotional stimulus, such as a medical examination, and the response of the blood pressure to the exhibition of certain pressor stimuli (e.g. cold, as in Hines' cold-water test) is employed directly as a test of this property of the blood pressure - its lability.

In such conditions as acute nephritis, toxæmia of pregnancy, and during the paroxysmal hypertension of adrenal pheochromocytoma, the variations in the level of the blood pressure do have some relation to underlying changes in the etiological process. We can hardly assume that the relatively large fluctuations in the normal person are conditioned in the same way, but rather that, superimposed on whatever process is operating primarily on the blood pressure, are incidental variations attendant upon the physiological factors controlling the normal blood pressure levels.

Addis (1922) first attempted to find a "basal" level for blood pressure by taking readings before patients rose from/

from bed in the morning.

Alam and Smirk (1943) set down their technique for the achievement of basal blood pressure. The blood pressure measurement was made 10-12 hours after the last meal on the previous evening, and after the patient had been resting comfortably in a warm and quiet room for 30 minutes. No conversational distractions were indulged in, and the patient was made accustomed to blood pressure readings by having them performed every two minutes during the half-hour. The conditions were such that most patients became somnolent during the test, though few actually fell asleep. Alam and Smirk likewise defined the "casual" blood pressure - the reading taken under ordinary conditions, and called the difference between casual and basal readings - the "supplemental" pressure. This supplemental pressure is a measure of the degree of elevation of the blood pressure due to emotional, nervous, or experimental factors and physical exertion.

Alam and Smirk (1943) and Kilpatrick (1948) found the supplemental pressure in essential hypertension to be much greater than in patients with normal blood pressure, and Kylin (1921), Ayman (1931), and Gatman, Nassif Amin and Smirk (1943) found that patients with chronic nephritis were much less likely to show wide variation, suggesting that the blood vessels of patients with renal hypertension are less reactive to vasomotor impulses than are those of patients with essential hypertension.

Now/

Now, if readings of blood pressure from case-records are to be used, some idea of their variability, and thus of their accuracy, should be obtained. It was decided also to try to determine whether the variability of the blood pressure in old people with essential hypertension is as great as it is in young subjects. Blood pressure readings were taken daily in 28 subjects - all from the one ward. The largest number of readings from one patient was 94, the smallest 6.

No attempt to obtain basal conditions was possible. The daily routine in the ward was as follows:- Before 9 a.m., patients were bathed, fed, and had their beds made. From 9.15 a.m. till almost 11.00 a.m. they were accustomed lying quietly in bed while clinical teaching of students was in progress, or while ward-rounds were made by the visiting staff, who were well known to the patients. Immediately after 11 a.m., though not an ideal time, was judged the best time at which to make daily blood pressure readings. On Sunday the ward was open to visitors from 10.30 to 11.30 a.m., and as there was much excitement, both before and after the visiting-hour, no blood pressure readings were taken on that morning. At each examination, three determinations of the blood pressure were made within a few minutes of one another, and the lowest figures were recorded.

For each patient the maximal and minimal readings for both systolic and diastolic pressure were noted, and these figures/

figures are recorded in the appendix to this chapter. All the patients over the age of 60, and only one under 40, as it happens, had diastolic hypertension, so we can compare the range of pressures obtained in hypertensive patients between the ages of 40 and 90 years.

Between 40 and 59 years, there were six cases of essential hypertension. The difference between maximal and minimal readings of the systolic pressure varied from 85 to 16 mm Hg., with an average of 55 mm Hg., and for the diastolic blood pressure the difference varied from 55 to 20 mm Hg., with an average variation of 40.3 mm Hg.

Between 60 and 90 years, there were 11 cases of essential hypertension, among whom the average range for systolic pressures was 47.5 mm Hg., with a range of 80 mm Hg. - 0 mm Hg.; the average range for diastolic pressures was 40.5 mm Hg., with a range of 75 mm Hg. - 10 mm Hg. Thus daily variations of the blood pressure were as marked in old people as in the age-group 40-59 years.

I have appended two charts, one showing the daily variations of blood pressure over a few days of (a) a woman of 44, and (b) a woman of 83, and the second showing the falls in blood pressure of (a) a woman of 29 and (b) a woman of 67, when sodium amytal was given to test the lability of the blood pressure and the suitability for sympathectomy. The use of this test, and of sodium nitrite and pentothal in/
in/

in similar circumstances, is not now believed to have the value formerly assigned to it (Allen and Adson, 1940). Nevertheless, these two charts are still an indication of the lability of the blood pressure in these two particular patients, and are included here as an illustration of the degree of such lability in an elderly patient compared with a younger one.

It is therefore just as important to ensure reasonable freedom from emotional and other stimuli when examining old people as when examining a more middle-aged group. The blood pressure in old people is no less labile, and age does not necessarily diminish vasomotor reactivity.

Caution must therefore be used in interpreting blood pressure readings from case-records. Readings in old people are just as likely to be too high as are readings from middle-aged groups. But the figures set down in Chapter II are still of interest in illustrating the changing trends as age progresses.

A P P E N D I X.

- 1) List of patients giving maximal and minimal blood-pressure readings and number of observations.
- 2) Chart showing daily variations in (a) a woman of 44 years (b) a woman of 83 years.
- 3) Chart showing fall in blood-pressure of (a) a woman of 29 years (b) a woman of 67 years when sodium amytal was given to test the lability of the blood-pressure.

A P P E N D I X.

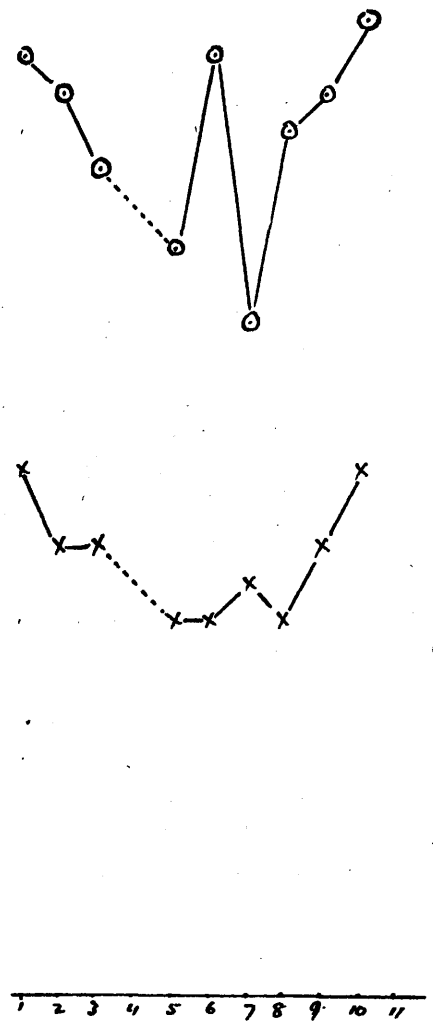
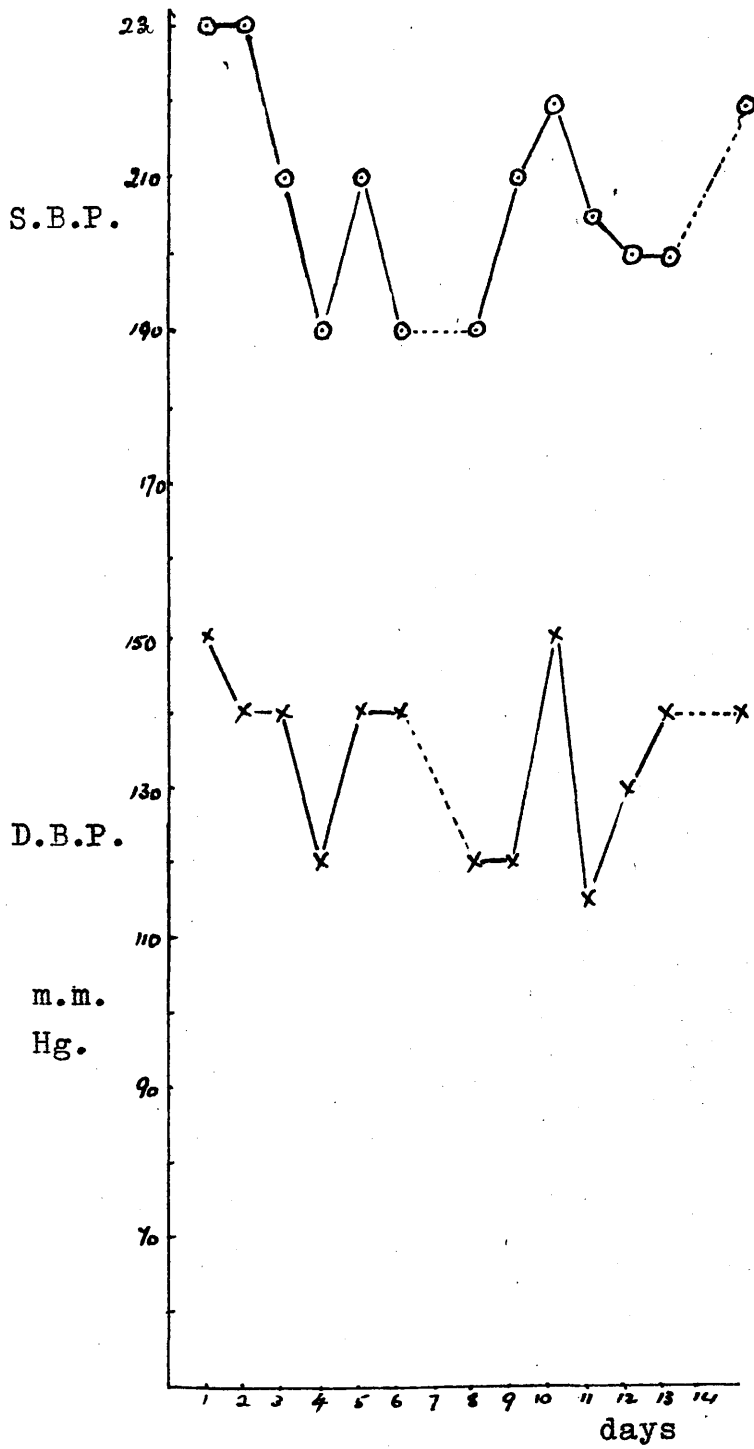
- 1) List of patients giving maximal and minimal blood-pressure readings and number of observations.
- 2) Chart showing daily variations in (a) a woman of 44 years (b) a woman of 83 years.
- 3) Chart showing fall in blood-pressure of (a) a woman of 29 years (b) a woman of 67 years when sodium amytal was given to test the lability of the blood-pressure.

No.	Age.	S.B.P. readings			D.B.P. readings			No. of observats.
		Highest	Lowest	Range	Highest	Lowest	Range	
1.	10	138	104	34	100	80	20	10
2.	13	170	140	30	70	60	10	10
3.	16	230	200	30	188	155	33	20
4.	18	125	100	25	85	70	15	24
5.	23	150	100	50	104	72	32	6
6.	24	150	130	20	104	80	24	10
7.	26	122	102	20	82	62	20	10
8.	29	140	110	30	96	60	36	21
9.	37	164	110	54	112	76	26	10
10.	40	212	150	62	120	82	38	19
11.	44	230	190	40	150	118	32	18
12.	46	136	120	16	90	80	10	6
13.	47	160	125	35	100	80	20	24
14.	54	210	190	20	140	100	40	19
15.	54	225	140	85	135	80	55	31
16.	58	240	155	85	155	100	55	94
17.	58	200	135	65	140	80	60	19
18.	63	190	145	45	120	84	36	15
19.	66	210	160	50	120	90	30	10
20.	67	250	250	0	180	170	10	7
21.	69	176	132	44	110	70	40	10
22.	70	220	140	80	135	60	75	30
23.	70	200	150	50	126	70	56	39
24.	75	210	150	60	130	80	50	24
25.	78	176	110	66	96	70	26	22
26.	78	198	160	38	122	90	32	10
27.	83	176	146	30	120	80	40	10
28.	84	250	190	60	150	100	50	24

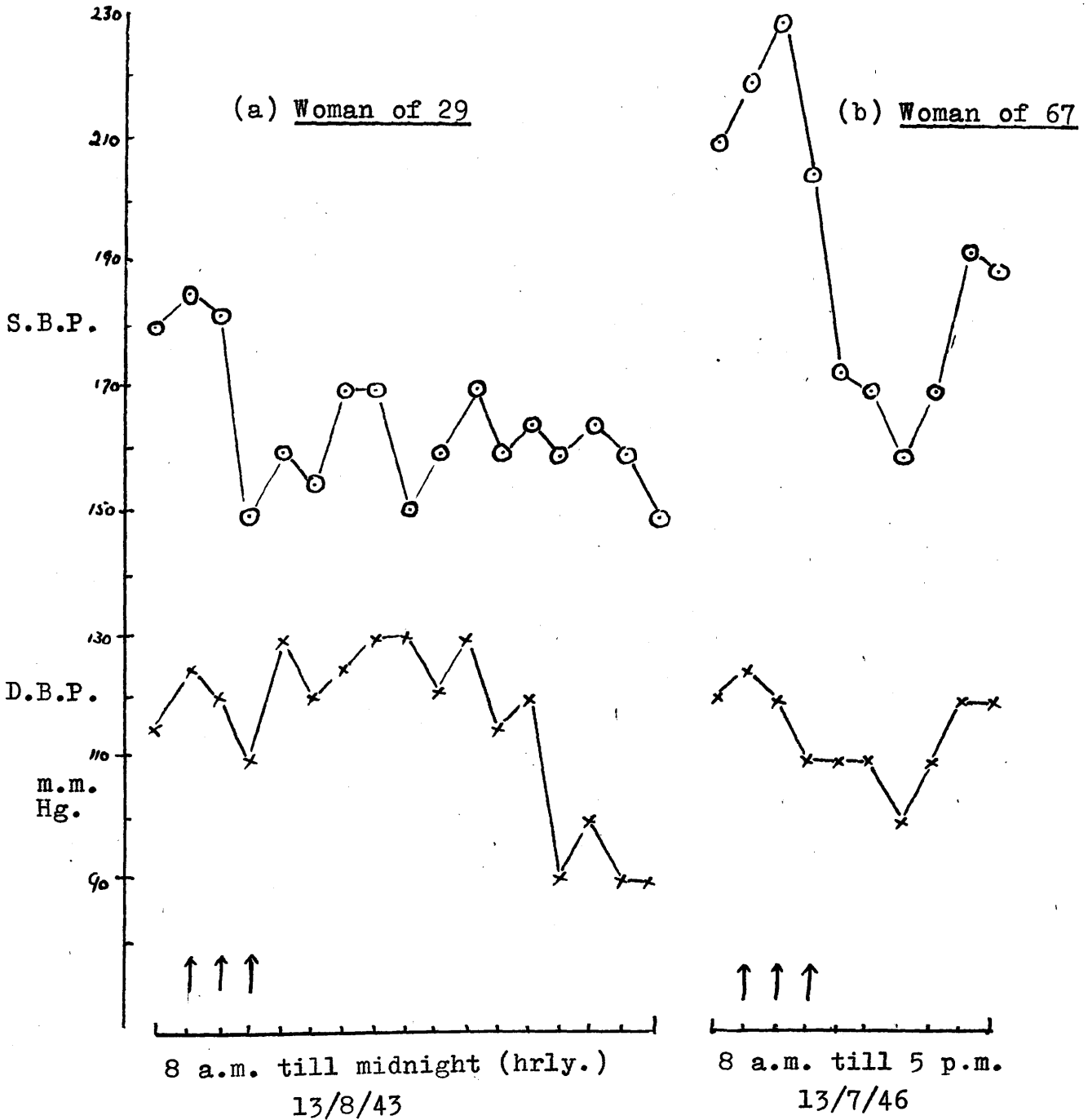
DAILY VARIATIONS IN BLOOD PRESSURE

(a) Woman of 44

(b) Woman of 83



EFFECT OF SODIUM AMYTAL ON BLOOD PRESSURE



↑ Sod. Amytal gr.iii given.

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P A R T II.

P A R T II.

THE DISABILITIES ASSOCIATED WITH HYPERTENSION.

- Chapter I. A description of the cases employed
and a discussion of the survey in Part II.
- Chapter II. The Causes of Death in Hypertension.
- Chapter III. The Incidence of "Complications."
- Chapter IV. Hemiplegia.
- Chapter V. Congestive Cardiac Failure.
- Chapter VI. a) Malignant Hypertension
b) Retinitis
c) Renal Failure
d) Subarachnoid Haemorrhage
e) Hypertensive Encephalopathy
f) The Lungs in Hypertension.
- Chapter VII. The Symptoms Associated with Hypertension.

P A R T II.

CHAPTER 1.

**A DESCRIPTION OF THE CASES EMPLOYED AND A
DISCUSSION OF THE SURVEY IN PART II.**

In Part I, I have shown that high blood pressure becomes more frequent with each succeeding decade, and that, over the age of 60 years, not only is there reputed to be a large number of hypertensives in the general population, but of patients admitted to the general wards in a municipal hospital over 30% had a diastolic pressure greater than 100 mm Hg., and over 50% had a systolic pressure greater than 150 mm Hg. It seemed pertinent, then, to enquire what were the disabilities associated with hypertension. As I proceeded with the examinations of elderly people (which I shall describe in full in Part III), I came to the conclusion that a relatively large number of my patients seemed to have asymptomatic and uncomplicated hypertension. For instance, it seemed to me that the prognosis in essential hypertension must be less gloomy than the outlook generally assigned to it. Secondly, my patients seemed to include relatively few with complaints of severe headache or dizziness, and in these several respects, the clinical picture in my elderly patients appeared to be different from that described in the literature, and different, too, from the ideas I had acquired in the wards of voluntary hospitals. Because of these apparent differences, I felt that some more precise definition was required of the extent and the type of incapacity which could accrue in my patients as a result of hypertension.

It seemed that the best method of undertaking this investigation was to make a comparison between the manifestations of hypertension in old age and in youth or middle-life. Having discovered that in the details of history-taking my own observations did not differ very markedly from those made routinely in the wards, I proceeded to list from the case-records of 514 male and 431 female patients symptoms and gross signs of disease.

These patients were selected on the basis of their blood pressure readings. Case-sheets of patients with readings higher than either 160 mm Hg. systolic or 100 mm Hg. diastolic were collected and analysed. A few patients with blood pressures below those levels were also included, and have been retained for comparison. It was at this stage that I decided to use 160 mm Hg. rather than 150 mm Hg. as the significant level above which I should describe systolic pressure as "high". Had I continued to include patients with a systolic pressure between 150 and 160 mm Hg. among the hypertensives, many more "uncomplicated" cases of hypertension would have been included.

Patients whose blood-pressure readings were above 160 mm Hg. systolic and 100 mm Hg. diastolic are hereafter referred to as having DIASTOLIC HYPERTENSION, while those with so-called SYSTOLIC/

SYSTOLIC HYPERTENSION had a systolic blood-pressure greater than 160 mm Hg., but a diastolic blood-pressure less than 100 mm Hg.

The 945 patients were grouped, as follows, according to their blood-pressure:-

	<u>Male</u>	<u>Female</u>
Diastolic Hypertension	334	329
Systolic Hypertension	113	80
S.B.P. & D.B.P. below the required limits)	47	15
With chronic nephritis	12	5
Blood pressure unknown	<u>8</u>	<u>2</u>
	514	431
	—	—

The 12 men and 5 women with chronic nephritis all had a systolic blood pressure above 160 mm Hg. and a diastolic blood pressure above 100 mm Hg., but they have been excluded from the diastolic hypertension group. They are the only cases, so far as is known, of "secondary" hypertension. All the patients in the diastolic hypertension group could, therefore, be said to have "essential" hypertension.

Paullin, Bowcock, and Wood (1927) have enumerated twenty-six "complications" of hypertension referring to the heart, the central nervous system, the eyes, the lungs, and the kidneys. (Strictly speaking, each symptom or sign of disease, /

disease, occurring along with essential hypertension and causally related to it, is a "complication" rather than a "symptom" of hypertension - but that is a quibble over terms). I have adopted a relatively simple system of classification, comprising, firstly, those who had no symptoms or complications associated with their hypertension; secondly, those who had symptoms, but no gross signs of disease which could be connected with their hypertension; thirdly, those in whom serious disease had supervened - cardiac failure, hemiplegia, renal failure, etc. In this third category, under their own separate heading, I have also included those who had chronic bronchitis or pulmonary emphysema, because while in practice it was difficult, even on complete physical examination, to decide whether or not some degree of left-sided heart-failure aggravated dyspnoea in these patients, merely from the reading of case-records this differentiation was impossible.

The findings are shown in the Tables 8 A & B, 9A & B, and 10 A & B, and from these tables comparison was made between patients with systolic and diastolic hypertension, between patients over and under 60 years of age, and between men and women. In subsequent chapters of Part II, I shall briefly comment on each manifestation of hypertension in turn.

Some explanation seems desirable of why 60 years was chosen as the dividing line between middle and old age. There is,/

TABLE 8 - A.

RESULT OF DIASTOLIC HYPERTENSION - MALES.

	AGE GROUPS - YEARS.											TOTAL	PERCENT %
	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85-89		
No signs or symptoms	-	-	2	2	1	12	8	6	11	1	1	44	13.15
Symptoms only	-	-	4	1	15	9	12	15	12	6	-	74	24.55
Cardiac Failure	2	1	4	9	9	15	18	12	2	-	-	72	21.56
Cardiac Failure + Auricular Fibrill ^o	-	-	-	1	2	4	3	1	1	1	-	13	
Cardiac Failure + Hemiplegia	-	-	-	1	-	-	2	-	-	-	-	3	
Cardiac Failure + Auric. Fibrill ^o + Hemiplegia	-	-	-	-	-	-	-	2	-	-	-	2	
Auricular Fibrillation	-	-	-	-	-	-	-	2	-	-	-	2	
Auricular Fibrill ^o + Hemiplegia	-	1	-	-	-	-	-	-	1	-	-	2	
Coronary Thrombosis	-	-	2	1	-	4	-	1	-	-	-	8	
Hemiplegia only.	1	3	-	6	11	14	18	7	12	4	1	77	23.02
Renal Failure	-	-	-	-	-	-	1	1	-	-	-	2	
Subarachnoid Haemorrhage	-	1	-	-	2	1	1	-	-	-	-	5	
Chronic Bronchitis + Emphysema	-	2	1	1	3	4	1	8	5	5	-	30	9.28
TOTAL	3	8	13	22	43	63	64	55	44	17	2	334	

TABLE 8 - B.

RESULT OF DIASTOLIC HYPERTENSION - FEMALES

	AGE GROUPS - YEARS															TOTAL	
	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85-89		90-94
No signs or symptoms	-	-	1	-	2	2	4	8	10	10	11	9	5	5	3	-	70
Symptoms only	-	-	-	3	2	1	5	5	11	15	24	14	10	4	1	-	95
Cardiac Failure	-	-	-	-	1	-	6	5	4	8	2	5	2	2	-	-	35
Cardiac Failure + Auric. Fibrillation	-	-	-	-	-	-	-	1	1	1	3	2	3	2	-	-	13
Cardiac Failure + Hemiplegia	-	-	-	-	-	-	2	1	1	-	2	-	-	-	-	-	6
Card. Failure + Auric. Fibrill. + Hemiplegia	-	-	-	-	-	-	-	1	1	-	-	-	-	-	-	-	2
Auricular Fibrillation	-	-	-	1	-	1	-	1	1	-	-	-	-	-	-	-	4
Auricular Fibrillation + Hemiplegia	-	-	-	-	-	1	-	-	-	-	-	-	-	-	1	-	2
Coronary Thrombosis	-	-	-	-	-	-	1	-	1	1	-	1	-	-	-	-	4
Chronic Nephritis with Cardiac Failure	-	-	-	-	-	-	-	2	-	-	-	-	-	-	-	-	2
Hemiplegia only	-	-	-	-	-	1	2	6	10	15	15	16	3	2	-	-	70
Subarachnoid Haemorrhage	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	1
Malignant Hypertension	1	-	1	1	1	-	-	-	-	-	1	-	-	-	-	-	5
Renal Failure	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	-	1
Retinitis only	-	-	-	-	-	-	-	1	2	-	1	-	-	-	-	-	4
Hypertensive Encephalopathy	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	1
Chronic Bronchitis with Emphysema.	-	-	-	1	-	-	-	2	1	2	1	3	2	-	-	-	12
TOTAL.	1	0	2	6	6	6	20	33	43	54	60	50	26	15	5	0	327

TABLE 9A RESULT OF SYSTOLIC HYPERTENSION - MALES

	AGE yrs.												TOTAL		
	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84		85-89	90-94
No symptoms or signs	-	-	-	2	2	-	1	1	10	3	4	3	-	-	26 (23.00%)
Symptoms only	-	-	-	-	-	-	-	2	3	8	10	3	-	-	26 (23.00%)
Cardiac Failure	-	-	-	-	-	1	2	3	4	11	2	-	-	-	23 (20.35%)
C.F. + Auricular Fibrillation	-	-	-	-	-	-	-	1	-	1	-	-	-	-	2
C.F. + Hemiplegia	-	-	-	-	-	-	1	-	1	-	-	-	-	-	2
Hemiplegia	-	-	-	-	-	-	-	3	8	3	2	2	-	-	18 (15.92%)
Subarachnoid Haemorrhage	-	-	-	-	1	-	-	1	-	-	-	-	-	-	2
Chr. Bronchitis + Emphysema	-	-	-	-	-	1	2	-	2	2	5	2	-	-	14 (12.39%)
TOTAL	.	.	.	2	3	2	6	11	28	28	23	10	.	.	113

TABLE 9B RESULT OF SYSTOLIC HYPERTENSION - FEMALES.

	AGE yrs.												TOTAL		
	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84		85-89	90-94
No symptoms or signs	-	1	-	1	2	3	2	4	3	6	6	3	-	1	34 (61.98%)
Symptoms only	1	-	1	1	3	1	2	3	2	4	-	-	-	-	18 (22.22%)
Cardiac Failure	-	-	-	-	-	1	1	2	2	1	2	2	-	1	12 (14.81%)
C.F. + Auricular Fibrillation	-	-	-	-	-	-	-	1	-	1	-	-	-	-	2
Auric. Fibrill ⁿ + Hemiplegia	-	-	-	-	-	-	-	-	-	-	-	-	1	-	1
Coronary Thrombosis	-	-	-	-	-	-	-	1	-	-	-	-	-	-	1
Hemiplegia	-	-	-	1	-	2	-	1	2	2	2	-	-	-	10 (12.34%)
Chr. Bronchitis + Emphysema	-	-	-	-	-	-	-	-	2	-	-	-	1	-	3
TOTAL	1	1	1	3	5	7	5	12	11	16	10	5	2	2	81

TABLE 10.A. COURSE IN NON-HYPERTENSIVES - MALES

AGE.	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	TOTAL
No symptoms or signs	5	3	1	-	3	3	2	-	17
Symptoms only	-	1	1	2	1	-	1	2	8
Cardiac Failure	2	-	1	4	1	1	1	1	11
C.F.+Auricular Fibrillation	-	-	-	-	1	-	-	-	1
Hemiplegia	-	-	1	2	-	3	-	-	6
Subarachnoid Haem.	-	-	-	-	-	1	-	-	1
Chr. Bronchitis+Emphysema	-	3	-	-	-	-	-	-	3
TOTAL	7	7	4	8	6	8	4	3	47

TABLE 10.B. COURSE IN NON-HYPERTENSIVES - FEMALES.

AGE.	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	TOTAL
No symptoms or signs	-	-	1	4	-	1	2	1	9
Cardiac Failure.	-	-	-	-	-	1	-	-	1
Chronic Bronchitis	-	-	-	1	2	1	-	-	4
Eye signs	1	-	-	-	-	-	-	-	1
TOTAL	1	0	1	5	2	3	2	1	15

is, of course, no moment of time when one can state, for oneself or for large numbers of people in the community, that here middle-life ends and here old age - the "grey remainder of the evening", which is to be sat out in idleness and musing - begins. Officially, for Old Age Pension purposes, old age begins in men at 65 and in women at 60, and this lower figure was accepted as my limit, so that I might compare similar age-groups in the two sexes.

That patients without hypertension should have cardiac failure, hemiplegia, or symptoms such as commonly occur in hypertensive patients, supports the view that during the course of a long immobilising illness like cardiac failure, the blood-pressure may become depressed. On the other hand, it cannot always be assumed that because a patient has both hypertension and cardiac failure, the latter is necessarily a result of the former.

It should be remembered that the population from which these figures are drawn is a hospital one, and therefore selected. Firstly, one would expect that the number of patients who were seriously ill as a result of hypertension would be increased disproportionately in comparison with the number of patients whose symptomless hypertension was accidentally discovered. Secondly, in this particular hospital it is to be expected that patients with more chronic types/

types of illness will predominate, and those acutely ill will tend to be excluded. In the absolute sense, then, it cannot be guaranteed that another group of hypertensive patients, selected another way, would not include a very different proportion with, for instance, cardiac failure. But for comparative purposes, it has been assumed that there was no large discrepancy between the methods of selection of hypertensive and non-hypertensive, young and old, male and female - and it is in this comparison that we are primarily interested.

This assumption may not necessarily be correct. For instance, the old are more likely to be alone in the world and to be brought into hospital because there is no-one to look after them outside. But on the other hand, the middle-aged, bearing the burden of maintenance of a family, and perhaps parents, are also unlikely to find it easy to get some one to nurse them while they are ill. Men are perhaps more commonly nursed at home by their wives than women by their husbands. On the other hand, women with home responsibilities may struggle on with their work and remain ambulant when men would succumb. Without more precise knowledge it is difficult to legislate for all the factors governing reasons for admission to hospital.

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CHAPTER II.

THE CAUSE OF DEATH IN HYPERTENSION.

Cause of Death in Hypertension.

As well as causing disability, hypertension shortens life. Masing in 1934, showed that for his 161 cases of hypertension, followed and observed in private practice for over 20 years, the average age on the first detection of hypertension was 57.3 years, and the average age at death 64.9 years. He contrasted with these findings the calculations of German life insurance societies that the expectation of life for a healthy man of 57 was 14.3 years.

Several series of cases have been observed for a relatively long time, and the resultant mortality rates are briefly summarised in Table 11. It will be seen that the various investigators differ widely in their results, but that where the figures for men and women were observed separately, each group was in agreement that the proportion of men dying within the given follow-up period was much higher than that of women.

Among the diastolic hypertensives in my group (vide Table 12) - 128 out of 334 men (38.30%), and 94 out of 327 (28.75%) died while in hospital. In 90 men (26.95%) and 62 women (18.96%), death was probably directly attributable to the result of hypertension. Between the figures for men and women there is a statistically significant difference, and it is in the group under 60 years that this difference primarily/

TABLE 11.

MORTALITY IN ESSENTIAL HYPERTENSION - VARIOUS AUTHORS.

Author & Year	No. of cases	Period of observation	% mortality		
			Men	Women	Men & Women
Janeway (1913)	458	1-10 yrs	53	33	{ 5 yr-50 10 " -75
Paullin (1926)	76	5-17 yrs	48.7	9.2	-
{ Blackford, Bowers & Baker (1930)	202	5-11 yrs	70	39	50
{ Blackford & Wilkinson (1932)	202	10 yrs	82	50	-
Masing (1934)	161	20 yrs	-	-	50
{ Keith, Wagener, & Barker (1939)	219	5-10 yrs	93	88	91

TABLE 12.

DIASTOLIC HYPERTENSION - CAUSE OF DEATH.

A. MALES:

Cause of Death.	Age in 5-yr. groups											Total	
	15-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84		85-89
Card. Failure	-	1	-	1	6	5	7	9	10	5	-	-	44
Cerebral Haem.	-	1	-	-	3	1	4	7	7	7	3	1	34
Coronary thrombosis	-	-	-	1	-	-	2	-	-	-	-	-	3
Uraemia	-	-	-	-	-	-	2	-	1	-	-	-	3
Subarachnoid Haem.	-	-	-	-	-	-	-	1	-	-	-	-	1
Grad. failure after apoplexy	-	-	-	-	-	4	-	1	-	-	-	-	5
Infections	-	-	-	-	-	1	-	-	-	2	3	-	6
Uncertain	-	-	-	-	-	-	2	8	4	4	3	-	21
Other causes	-	-	-	-	1	-	2	2	5	-	1	-	11
No. of deaths	-	2	-	2	10	11	19	28	27	18	10	1	128
No. patients	None	3	8	13	22	43	63	64	55	44	17	2	334

TABLE 12.

DIASTOLIC HYPERTENSION - CAUSE OF DEATH.

B. FEMALES:

Cause of Death.	15-19	20-24	25-29	Age in 5-yr. groups									75-79	80-84	85-89	Total
				30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74				
Card. Fail.	1	-	1	1	1	1	-	1	3	5	2	4	2	3	-	20
Cerebral Haem.	1	-	1	1	1	1	1	1	2	6	6	5	1	1	-	23
C. Thrombosis	1	-	1	1	1	1	1	1	1	1	-	-	-	-	-	1
Uraemia	1	-	1	1	1	1	1	1	1	1	1	1	1	-	-	1
Subarch. Haem.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Grad. failure after apoplexy	-	-	-	-	-	1	1	1	-	-	1	3	2	-	-	9
Infections	-	-	1	1	1	1	1	1	1	1	1	1	-	-	1	3
Uncertain	-	-	-	-	-	-	2	-	2	3	5	4	2	5	-	23
Other causes	-	-	-	-	-	-	-	-	3	1	-	-	1	1	-	6
Malign. Hypert.	1	-	-	-	1	-	-	-	-	-	-	-	-	-	-	2
Cerebral Embol. in Aur. Fibn.	-	-	-	-	-	1	-	1	1	1	1	-	-	1	-	6
No. of deaths	1	-	-	-	1	2	4	4	11	17	16	17	9	11	1	94
No. patients	1	-	2	6	6	26	20	33	43	54	60	50	26	15	5	327

primarily exists. Among people under 60 years of age, the mortality rates were - men 23 out of 89 (25.84%), and women 16 out of 117 (13.68%). The corresponding figures for the age-groups over 60 were - men 67 out of 245 (27.35%), and women 46 out of 210 (21.91%). There is no statistically significant difference in this last group of figures.

In the cases of both the men and the women, there was no significant difference in the mortality rate due to hypertension between those under 60 and those over 60. Although, therefore, in men under 60 more often than in women under 60, hypertension led to death, in old age (i.e. over 60 years) both in men and in women death was directly due to hypertension as often as under 60 years.

Such figures as I have been able to find in the literature for the relative incidences of various causes of death in hypertensive patients illustrate the discrepancies which occur between the figures of various groups of investigators, and I have set them down in tabular form for comparison with my own figures (Table 13).

TABLE 13.

CAUSE OF DEATH IN HYPERTENSIVE PATIENTS.

Author	Year of publicn.	Cause of Death - % mortality			
		Cardiac Failure	Cerebral Haem.	Uraemia	Other causes
Janeway	1912	29	14	15	42
Christian	1926	32	25	4.5	38.5
Bell & Clawson	1928	60.4	19.3	8.5	11.0
Granger	1929	52	31.5	5.5	11.0
Murphy I	1933	23.3	-	-	76.7
Murphy II	1933	56.4	14.9	7.6	21.1
Murphy III	1933	24.1	17.2	55.2	3.5
Self (Males)		34.4	31.2	2.3	32.8
(Females)		21.3	34.0	1.1	43.6

I have included in my subsequent comments about the various manifestations of hypertension, brief notes about their importance as a cause of death.

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CHAPTER III.

THE INCIDENCE OF "COMPLICATIONS."

INTRODUCTION.

- 1) Effect of Hypertension.
- 2) Changes with Age.
- 3) Difference in the course of Hypertension in
Men and Women.
- 4) Effect of Pregnancy in Women.

Conclusion.

INTRODUCTION

In this and each of the following chapters of Part II, I have considered one manifestation of Hypertension, and have compared its incidence in diastolic and systolic hypertension, in the age-groups under and over 60 years, and in men and women. I have also discussed briefly the nature of congestive cardiac failure, hemiplegia, etc., and their relation to hypertension.

The present chapter deals with the first group of cases listed in Tables 8, 9, and 10 - in other words, patients whose high blood pressure was an incidental discovery causing to appear neither symptoms nor signs relative to hypertension. I have then in subsequent chapters examined the other "complications" of hypertension in turn, leaving to the end a consideration of the symptoms of which many patients complained.

Comparisons between groups of figures have been made by the use of the χ^2 method to determine whether differences were statistically significant. Throughout the rest of the thesis, the word 'significant' indicates statistical significance.

1. Effect of Hypertension.

Among men, 23% with systolic hypertension (26 out of 113 patients) and 36.16% without hypertension (17 out of 47 patients) were free from complications. (Vide tables 9A and 10A) As there is no statistically significant difference between these figures, they can be added together and the joint incidence contrasted with the 13.17% of diastolic hypertensives (table 8A) without manifestations of disease (44 out of 334 patients). The difference in incidence is statistically significant.

Similarly, in women (tables 8B and 9B) there was a statistically significant difference between the 22.0% of diastolic hypertensives (70 out of 327 patients) and the 41.98% of systolic hypertensives (34 out of 81 patients) who were free from hypertensive disease.

In other words, as one would expect, among patients with diastolic hypertension, there was a significantly larger proportion with "complications" or symptoms of hypertensive disease, compared with systolic hypertension.

2. Changes with Age.

In Tables 8A & B, the patients with diastolic hypertension have been separated into 5-year age-groups.

It/

It was particularly noticeable that a very large proportion of male patients under the age of 60 suffered from some complication. Indeed, only 5.6% (5 out of 89) of those under 60 years were free from manifestations of disease. A significantly higher proportion of those over 60 years had no complications - there being 39 out of 245 patients (15.2%) without any manifestations of disease.

In female patients, however, the same relationship did not obtain. 27 of 117 patients (22.89%) under 60 years, and 43 of 210 patients (20.88%) over 60 years were symptom-free, and there is no significant difference between these figures.

Thus it would seem that hypertension in men under 60 is more virulent and more likely to be associated with complications than in older men, while there is little difference between similar groups in women.

3. Difference in the course of hypertension in men and women.

When, on the other hand, male and female patients of similar age-groups were compared, it was found that after the age of 60, hypertension was symptom-free in more or less equivalent proportions of men and women, while below this age a very much higher proportion of men than of women showed signs or symptoms.

Under/

Under 40 years of age, the number of men was not sufficient for an effective comparison to be made, but when hypertension in men was compared with that in women between 40 and 59 years of age (the menopausal ages) it was found that its manifestations in women were relatively benign compared with men of a similar age-group.

Above the age of 60, there seemed to be no difference between the proportions of men and women who being hypertensive were seriously incapacitated by the disease.

4. Effect of pregnancy in women.

Of the 327 women with diastolic hypertension, 175 were known to have had children, 80 were known to have had none, and in 72 cases whether they had or had not was unknown. 40 of the patients (22.8%) who had had children were free of complications and 18 of the childless patients (22.2%). There is no significant difference between these proportions.

Conclusions:

Patients with diastolic hypertension were more likely to be incapacitated by diseases accounted as hypertensive "complications" than patients with systolic hypertension. Also a greater proportion of men under 60 had "complications" than those over 60. In women, however, age did not make any difference.

Sex/

Sex had no effect on the incidence of incapacity amongst those over 60 years of age, but men under 60 years were more affected than women under 60 years.

The fact that a woman had borne children did not appear to render her more liable to disability from hypertension.

CHAPTER IV.

HEMIPLEGIA.

- 1) The Nature and Cause of Hemiplegia.
 - 2) Incidence of Hemiplegia in my cases.
 - 3) The Relation of Hemiplegia to Hypertension.
 - 4) The Importance of Age.
 - 5) The Significance of Sex.
 - 6) The Relation of Hemiplegia to Cardiac Failure.
 - 7) The Side Affected.
 - 8) The Prognosis in Hemiplegia as regards Life.
 - 9) Prognosis and Duration in Hospital of Survivors.
 - 10) Prodromal Signs in Apoplexy.
 - 11) Reasons for Admission to Hospital.
- Conclusions.

HEMIPLEGIA

Section (1).

Introduction - The Nature and Cause of
Hemiplegia.

The development of a hemiplegia is a disabling disaster at any time of life. It follows on a lesion interrupting the cerebral course of the fibres of one or other motor tract, such a lesion being more likely to produce widespread havoc where the pyramidal tract fibres are close together, as in the internal capsule. Trauma and new growth involving this area may account for a few cases of hemiplegia, but the overwhelming majority are due to cerebro-vascular disease - haemorrhage, thrombosis, or embolus. The relationship between apoplexy and cerebral haemorrhage has been known since the days of Johann Jakob Wepfer (1620-98), who was especially interested in diseases of the head. The proximal cause of cerebral haemorrhage (or thrombosis) shares, however, the obscurity surrounding the whole subject of vascular diseases.

It is said (Lippmann, quoted by Fishberg 1939) that the vast majority of cases of cerebro-vascular catastrophe occur in the presence of hypertension. Baker (1937) has shown that the average small cerebral artery has little elastic or muscle tissue/

tissue in its medial coat. According to James (1926), the branches from the middle and anterior cerebral arteries to the thalamus and corpus striatum are especially small and thin-walled, and since they arise directly from the large arterial trunks, they receive the full impact of all changes in the blood pressure. Charcot called one of these branches "the artery of cerebral haemorrhage." Fishberg mentions also, however, in his discussion of the causation of cerebral haemorrhage, an experiment of Lampert and Mueller (1926) who found that a pressure of 1520 mm Hg. ruptured the cerebral arteries in only two out of 30 cadavers. They concluded that high blood pressure was not the only factor to be contended with.

Russell Brain makes the assumption that the artery which is ruptured or thrombosed is an atheromatous one. The cerebral arteries frequently show arterio-sclerosis, but the exact relationship between cerebral arterio-sclerosis and cerebral haemorrhage is still as undetermined as the relationship between hypertension and cerebral arterio-sclerosis.

But even in the absence of precise knowledge of its cause, it is still important to enquire into the amount of disability resulting from cardio-vascular catastrophe, to endeavour to discover whether its onset could be prevented, and especially to determine how best patients can be treated and/

and full function of their paralysed limbs restored.

Section (2). The Incidence of hemiplegia in my cases.

The following table (Table 14) shows how the hemiplegic patients were distributed:-

TABLE 14. INCIDENCE OF HEMIPLEGIA

A. MALES:

Complication	D.H.	S.H.	non-H.	no infor.	Total
Hemiplegia only	77	18	6	1	102
Hemiplegia & Cardiac Failure	3	2	-	1	6
Hemiplegia & C.F. & Aur. Fibr.	2	-	-	1	3
Hemiplegia & Aur. Fibrillation	2	-	-	-	2
Total in each group	334	113	47	8	

B. FEMALES:

Complication	D.H.	S.H.	non-H.	Total
Hemiplegia only	70	10	-	80
Hemiplegia & Cardiac Failure	6	-	-	6
Hemiplegia & C.F. & Aur. Fibr.	2	-	-	2
Hemiplegia & Aur. Fibrillation	2	1	-	3
Total in each group	327	81	15	

A very few patients (10 men and 11 women) had in addition to hemiplegia, cardiac failure, or auricular fibrillation or both.

Section (3).

The Relation of Hemiplegia to Hypertension.

An examination of Tables 14 A & B, shows that the proportions of patients with diastolic hypertension who developed hemiplegia (83 out of 334 men (24.85%) and 80 out of 327 women (24.46%)) were significantly greater than the corresponding proportions of those with systolic hypertension (20 out of 113 men (17.70%) and 11 out of 81 women (13.58%)). It must be remembered that all the blood pressure readings were made after the occurrence of apoplexy, and it is not impossible that rest in bed, enforced immobility, and perhaps also enfeebled mental effort had caused some lowering of the original figures. The proportions of hypertensives developing hemiplegia are higher than the 7.2% of Paullin, Bowcock, and Wood (1927), 14% of Janeway (1912), and 12% of Marshall (1932) though Marshall's cases were also from hospital in-patients.

Section (4).

The Importance of Age.

Tables 8 A & B show how hemiplegics were distributed in the various age-groups. Though it has been stated that apoplexy largely comes on in late middle life, among my cases hemiplegia was found in every 5-year group from the thirties to the eighties, both in men and in women. 23 of the men were under 60, and 61 were over 60 years of age, while the total/

total numbers in these respective age-groups were 89 and 245. Thus 25.84% of the hypertensive patients under 60 (and 24.90% of those over 60) had developed hemiplegia.

Among women, the figures of those who developed hemiplegia were 22.22% (26 out of 117) under 60 years of age, and 25.71% (54 out of 210) over 60 years. Thus the greater number of patients with hemiplegia were over 60 years, and hypertensives were just as liable to develop hemiplegia in old age as in middle life. But hemiplegia may also occur early in life, and presumably also early in the history of the hypertension. If it is true that hemiplegia has some relation to cerebral arterio-sclerosis, then in some subjects this must develop rapidly and at an early age.

Section (5).

The Significance of Sex.

No significant difference was discovered between the incidences in the two sexes, neither under nor over 60 years of age.

Section (6).

The Relation of Hemiplegia to Cardiac Failure.

Fishberg has stated that the enforced diminished physical activity after hemiplegia tends to spare the heart, so that the greatest danger for the recovered patient is recurrence of his apoplexy. As I shall show, in Tables 20 A & B., of all/

all the hemiplegic patients, only four men and two women were having their second apoplectic attack. Only a small number of patients had both hemiplegia and cardiac failure; and in all but two, cardiac failure preceded the onset of hemiplegia. A brief summary of the history of these two patients is given, because although their number is small in comparison with the total number of hemiplegic patients, their stories do suggest that cerebral haemorrhage or thrombosis does not necessarily indicate immunity from cardiac failure.

The first patient, a woman aged 58, developed hemiplegia in September 1943 and was admitted to hospital in June 1944, having had dyspnoea on exertion for five months. The second woman, aged 65, had an interval of two years between the onset of her hemiplegia and her admittance to hospital in December 1945 with attacks of dyspnoea.

Section (7).

The Side Affected.

The following table shows the incidence of left and right-sided hemiplegia, in men and in women:-

TABLE 15./

TABLE 15. HEMIPLEGIA - SIDE AFFECTED.

SIDE	Males				Females			
	Imp.	ISQ	Died	Total	Imp.	ISQ	Died	Total
Left	23	3	21	47	16	2	17	35
Right	26	6	30	62	27	6	22	55
Both	1	-	3	4	-	-	1	1
Total	50	9	54	113	43	8	40	91

Imp. = Improvement. ISQ = No change.

In both sexes, right-sided hemiplegia occurred more often than left. Now one would expect the right-handed individual who has developed a right-sided hemiplegia and aphasia to be more severely handicapped than the right-handed person with a left-sided hemiplegia, and this increased incidence of right-sided paralysis in my cases may not necessarily reflect an increased incidence in the community, but a greater need for hospital treatment because of greater incapacity. It would be of interest to undertake a more thorough investigation into the differences between the effects of left and right-sided lesions. We can, in part, deal with the question by discussing any difference of prognosis in the two groups.

Section (8).

The Prognosis in Hemiplegia as regards life.

Table 15 shows the number of hemiplegic patients who died, and the number who recovered sufficiently to go out of hospital. 54 male (47.78%) and 40 female (43.96%) patients died. The prognosis then, as regards life, was no different in the two sexes.

Similarly, there was no significant difference between the prognosis in left and right-sided lesions, for in men 48.39% of the right-sided hemiplegics and 44.68% of the left-sided, died in hospital. For women the figures were 40.00% and 48.61% respectively.

From Table 16, it can be determined that in the under-60 age-groups, 13 male patients out of 26 (50.00%) and 12 female patients out of 29 (41.38%) died in hospital, and in the over-60 age-groups 41 male patients out of 87 (47.13%) and 28 female patients out of 62 (45.16%) died in hospital. There was thus, as regards life, no difference in prognosis in the two age-groups.

In Tables 16 & 17 is set forth the duration of stay in hospital of those who died - analysed according to age (table 16) and side affected (table 17). Newbill (1940) has analysed the records of 296 cases of cardio-vascular accident in which autopsies were made. He showed (a) that sudden death (in less than two hours) was rare, (b) that within 24 hours, 64 patients had/

TABLE 16.

AGE-GROUPS - LENGTH OF STAY IN HOSPITAL (Fatal Cases)

A. MALES:

	Less than 1 wk.	1-2 wks.	2-3 wks.	3-4 wks.	1-2 mths.	2-3 mths.	3-6 mths.	6-9 mths.	9-12 mths.	12-15 mths.	15-18 mths.	18-21 mths.	21 mths - 2 Yrs.	Over 2 Yrs.	Total.
Under 60	4	1	-	2	-	3	2	-	1	-	-	-	-	-	13
Over 60	15	2	2	2	14	3	-	1	-	1	1	-	-	-	41
Totals	19	3	2	4	14	6	2	1	1	1	1	-	-	-	54

B. FEMALES:

	Less than 1 wk.	1-2 wks.	2-3 wks.	3-4 wks.	1-2 mths.	2-3 mths.	3-6 mths.	6-9 mths.	9-12 mths.	12-15 mths.	15-18 mths.	18-21 mths.	21 mths-2 Yr	Over 2 Yrs.	Total.
Under 60	3	1	2	-	4	-	-	-	-	1	-	-	1	-	12
Over 60	10	3	3	3	3	-	3	1	1	1	-	-	-	-	28
Totals	13	4	5	3	7	-	3	1	1	2	-	-	1	-	40

TABLE 17.

HEMIPLEGIA - LENGTH OF STAY IN HOSPITAL (Fatal Cases)

A. MALES:

Side Affected \ Duration of stay	Duration of stay													Total
	Less than 1 wk.	1-2 wks.	2-3 wks.	3-4 wks.	1-2 mths	2-3 mths	3-6 mths	6-9 mths	9-12 mths	12-15 mths	15-18 mths	18-21 mths	21 mths - 2 yrs.	
Left side only	7	2	-	1	6	2	-	1	1	-	1	-	-	21
Right " "	12	1	1	3	6	4	2	-	-	1	-	-	-	30
Both sides	-	-	1	-	2	-	-	-	-	-	-	-	-	3
Total	19	3	2	4	14	6	2	1	1	1	1	-	-	54

B. FEMALES:

Side Affected \ Duration of stay	Duration of stay													Total
	Less than 1 wk.	1-2 wks.	2-3 wks.	3-4 wks.	1-2 mths	2-3 mths	3-6 mths	6-9 mths	9-12 mths	12-15 mths	15-18 mths	18-21 mths	21 mths - 2 yrs.	
Left side only	7	1	1	2	2	-	2	1	1	-	-	-	-	17
Right " "	6	3	3	1	5	-	1	-	-	2	-	-	1	22
Both sides	-	-	1	-	-	-	-	-	-	-	-	-	-	1
Not stated	-	-	-	-	-	-	-	1	-	-	-	-	-	1
Total	13	4	5	3	7	-	3	1	1	2	-	-	1	40

had died, (c) that of the 18 patients who lived for more than a year, only one had haemorrhage as the basis of his symptoms, while seventeen had had thrombosis, and (d) that the average survival period of females was much longer than that of males.

Among my cases, 19 males and 13 females died within the first week of admission into hospital. The other fatal cases lingered on, some for a very long period, and the actual cause of death was sometimes exceedingly difficult to determine. Twelve men and eight women were in hospital for more than two months before they died. The factors which determine the course of these long-term, unimproved hemiplegias are likely to be complex, as illustrated by the following case:-

A woman of 44, single, but not by any manner of means friendless, had a right-sided hemiplegia with aphasia. Before the onset of the "shock" on 21.4.45, she had, according to her relatives, been quite active and healthy. After consciousness returned, however, she made no attempt to get well. She seemed dazed and unintelligent, sat propped up in bed but took no interest in her surroundings. Although the function of her paralysed limbs improved, she could not be induced to get out of bed. After recovering from a pulmonary infection, she began to develop a painful oedema of the paralysed arm and hand, for which no cause could be found. There was no other evidence of myocardial insufficiency, though occasionally extrasystoles were heard. No urinary albumin was found though the/

TABLE 18.

AGE-GROUPS - LENGTH OF STAY IN HOSPITAL (Survivors)

A. MALES:

	Less than 1 wk.	1-2 wks	2-3 wks	3-4 wks	1-2 mths	2-3 mths	3-6 mths	6-9 mths	9-12 mths	12-15 mths	15-18 mths	18-21 mths	21-24 mths	Over 2 yrs	Totals
Under 60	-	1	2	1	2	3	3	1	-	-	-	-	-	-	13
Over 60	-	1	-	3	18	11	10	1	1	1	-	-	-	-	46
Totals	-	2	2	4	20	14	13	2	1	1	-	-	-	-	59

B. FEMALES:

	Less than 1 wk.	1-2 wks	2-3 wks	3-4 wks	1-2 mths	2-3 mths	3-6 mths	6-9 mths	9-12 mths	12-15 mths	15-18 mths	18-21 mths	21-24 mths	Over 2 yrs	Totals
Under 60	2	-	-	-	5	6	3	-	1	-	-	-	-	-	17
Over 60	2	3	2	3	15	2	3	1	-	-	1	1	-	1	34
Totals	4	3	2	3	20	8	6	1	1	-	1	1	-	1	51

TABLE 19.

HEMIPLEGIA - LENGTH OF STAY IN HOSPITAL (Survivors)A. MALES:

Side Affected	Duration of Stay													Total	
	Less than 1 wk.	1-2 wks	2-3 wks	3-4 wks	1-2 mths	2-3 mths	3-6 mths	6-9 mths	9-12 mths	12-15 mths	15-18 mths	18-21 mths	21-24 mths		Over 2 Yrs
Left side only	-	-	2	2	10	5	6	1	-	-	-	-	-	-	26
Right " "	-	2	-	2	9	9	7	1	1	1	-	-	-	-	32
Both sides	-	-	-	-	1	-	-	-	-	-	-	-	-	-	1
Total with Hemiplegia.	-	2	2	4	20	14	13	2	1	1	-	-	-	-	59
*All other survivors	12	14	21	32	109	34	36	5	4	1	-	-	1	-	269

B. FEMALES:

Side Affected	Duration of Stay													Total	
	Less than 1 wk.	1-2 wks	2-3 wks	3-4 wks	1-2 mths	2-3 mths	3-6 mths	6-9 mths	9-12 mths	12-15 mths	15-18 mths	18-21 mths	21-24 mths		Over 2 Yrs.
Left side only	2	1	-	2	4	4	2	-	-	-	1	1	-	1	18
Right " "	2	2	2	1	16	4	4	1	1	-	-	-	-	-	33
Both sides	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total with Hemiplegia	4	3	2	3	20	8	6	1	1	-	1	1	-	1	51
*All other survivors	6	12	25	31	118	26	21	10	3	1	-	-	-	-	253

* The duration of stay in hospital of all those in the survey who did not die in hospital - for comparison with those with hemiplegia.

the serum protein level was rather low (5.25 gms.%). The blood pressure level, hitherto very high (210/130) began to fall on 28.5.46. On 31.5.46 the patient was comatose, giving no response to painful stimuli, and having large dilated pupils, which, however, reacted to light. There was marked muscular rigidity, but on lumbar puncture a clear, colourless fluid was obtained, not increased in pressure, and not abnormal in any particular. Eventually, on 3.6.46 death ensued, after fourteen months in hospital, but unfortunately no autopsy was obtained.

Section (9).

Prognosis and Duration in Hospital of Survivors.

It will be seen from Table 19, that the time in hospital for hemiplegics was often longer than that of the other patients in the survey. Of hemiplegic patients who survived, 52.54% (31 out of 59) men and 37.26% (19 out of 51) women were in hospital for more than two months, while for the other surviving patients in the survey, the proportions were 30.12% (81 out of 269) men and 24.02% (61 out of 254) women. The proportion of the surviving hemiplegics who remained in hospital for over two months was significantly more than that of the other patients. If, however, we examine the figures for hemiplegia once more, we find that by the end of three months only 17 men and 11 women, and by the end of six months only four/

four men and five women still remained in hospital. Warren (1948), outlining her scheme for the rehabilitation of hemiplegic patients, mentions that they may show "very satisfactory progress in three months", and my figures would seem to confirm this statement.

Of those who survived (table 18) in the under 60 groups, there were 13 males and 17 females, and in the over 60 groups, 46 men and 34 women.

7 of the men under 60 were in hospital for more than two months, and 24 of the men over 60. Among women, 10 out of the 17 under 60 and 9 out of the 34 over 60 were in hospital for longer than two months, and between these two groups of figures for women there is a significant difference.

Men, on the whole, perhaps, tended to be a little longer in hospital than women, but there was no significant difference between the proportion of those with right-sided lesions who were in hospital for over two months, and the proportion of those with left-sided lesions who were in hospital for more than two months. The relevant figures are:

a) Right hemiplegia - men (12 out of 26) 46.15%

Women (9 out of 18) 50%

b) Left hemiplegia - men (19 out of 32) 59.39%

women (10 out of 33) 30.31%.

Section (10).

Prodromal Signs in Apoplexy.

Most contemporary discussions of hemiplegia (Page & Corcoran, Fishberg, Cowdry) mention that signs and symptoms referable to the central nervous system can precede the development of a cardio-vascular catastrophe. Though I can find no reference to it in the literature, I have seen attempts made to determine whether or not a patient is likely to develop hemiplegia on the basis of measurements of the pulse pressure - a high pulse pressure and a high diastolic pressure being regarded as indicative of the probable development of hemiplegia. Some means of assessing prognosis might be of value in giving assurance to those who are not likely to be involved in such a possibility. Taylor and Page (1945), examining 40 patients who died of hypertension, found five signs and symptoms constantly present in those dying of cerebral haemorrhage, and rare or absent among those patients dying from other causes. These premonitory signs and symptoms were :-

- 1) severe occipital or nuchal headaches
- 2) vertigo or syncope
- 3) motor or sensory neurologic disturbances
- 4) nose bleeds
- 5) retinal haemorrhages in the absence of papilloedema or exudates.

Lange (in Cowdry - Arteriosclerosis. 1933) refers to paralytic affections, vasomotor disturbances, and functional disturbances affecting the part of the brain in which the hæmorrhage is going to occur.

Tables 20 A & B. attempt to discover the past history of diastolic hypertensives who have developed hemiplegia, and these patients' complaints can be compared with those of all the other patients, as shown in Tables 26 A & B.

Out of the total of 77 male and 70 female hemiplegics, 25 men and 28 women were too ill on admission or too confused to give any history. Of the remainder, 21 (46.38%) men and 21 (50.00%) women had had no symptoms indicative of hypertension previous to the onset of the hemiplegia. There was no significant difference in the proportion of those without any prodromal symptoms in those over and under 60 years of age. Four men and two women had had a previous cerebrovascular catastrophe. The remaining 27 men and 19 women had had some previous symptoms, which were collected and listed in the method described in Chapter VII.

Among these patients, the main complaints, in order of frequency, were - 1) headache. 2) vertigo. 3) blurring of vision, and 4) dyspnoea (equal in frequency to 3.). This is in contrast to the order of frequency of symptoms in hypertensive patients as a whole where dyspnoea was much the commonest/

TABLE 20. SYMPTOMS IN DIASTOLIC HYPERTENSION WITH HEMIPLEGIA

A. MALES:

	35- 39	40- 44	45- 49	50- 54	55- 59	60- 64	65- 69	70- 74	75- 79	80- 84	85- 89	Total
Headache	-	-	-	-	2	4	4	1	3	1	-	15
Dyspnoea	-	-	-	-	2	1	4	-	-	-	-	7
Vertigo	-	1	-	-	-	3	2	1	3	-	-	10
Blurring/vision	-	-	-	-	-	3	2	-	4	1	-	10
Praecordial pain	-	-	-	-	-	-	-	-	-	-	-	-
Palpitation	-	-	-	-	-	-	-	-	-	-	-	-
Epigastric discomfort	-	-	-	-	-	1	-	-	-	-	-	1
Weakness	-	-	-	-	1	-	2	-	-	-	-	3
Fatigue	-	-	-	-	1	-	-	-	1	-	-	2
Depression	-	-	-	1	-	-	-	-	-	-	-	1
Insomnia	-	-	-	1	-	-	-	-	-	-	-	1
Nervousness	-	-	-	-	-	-	-	-	-	-	-	-
Paraesthesia	-	-	-	-	-	1	-	-	-	-	-	1
Trans. paresis	-	1	-	-	1	1	1	-	1	-	-	5
Urinary frequency	-	-	-	-	-	1	2	-	1	-	-	4
Epistaxis	-	-	-	-	-	-	-	-	-	-	-	-
Haematuria	-	-	-	-	-	-	-	-	-	-	-	-
Haemoptysis	-	-	-	-	-	-	-	-	-	-	-	-
Haematenesis	-	-	-	-	-	-	-	-	-	-	-	-
Blood/ rectum	-	-	-	-	-	-	-	-	-	-	-	-
Total with symptoms	-	1	-	1	5	5	6	1	6	2	-	27
History unobtainable	1	1	-	2	6	2	7	2	3	1	-	25
No previous symptoms	-	1	-	2	-	6	5	4	1	1	1	21
Previous cerebral thrombosis	-	-	-	1	-	1	-	-	2	-	-	4
Total in each age-group	1	3	-	6	11	14	18	7	12	4	1	77

TABLE 20. SYMPTOMS IN DIASTOLIC HYPERTENSION WITH HEMIPLEGIA

B. FEMALES:

	40- 44	45- 49	50- 54	55- 59	60- 64	65- 69	70- 74	75- 79	80- 84	Total
Headache	-	-	1	1	3	4	2	-	-	11
Dyspnoea	1	-	-	1	1	2	2	-	-	7
Vertigo	1	-	1	1	1	-	2	-	-	6
Blurring/ vision	-	-	1	1	1	-	1	-	-	4
Praecordial pain	-	-	-	-	1	-	-	-	-	1
Palpitation	-	-	-	-	-	-	-	-	-	-
Epigastric discomfort	-	-	-	-	-	-	-	-	-	-
Weakness	1	-	1	-	-	2	1	-	-	5
Fatigue	-	-	1	1	-	1	-	-	-	3
Depression	-	-	-	1	-	-	-	-	-	1
Insomnia	-	-	-	-	1	-	-	-	-	1
Nervousness	-	-	-	-	-	-	-	-	-	-
Paraesthesia	-	-	-	-	-	-	-	-	-	-
Trans. paresis	-	-	-	-	-	-	2	-	-	2
Urinary frequency	-	-	-	-	-	1	1	-	-	2
Epistaxis	-	-	-	-	1	-	-	-	-	1
Haematuria	-	-	-	-	-	1	-	-	-	1
Haemoptysis	-	-	-	-	-	-	-	-	-	-
Haematemesis	-	-	-	-	-	-	-	-	-	-
Blood/rectum	-	-	-	-	-	-	-	-	-	-
Blood / uterus	-	-	-	-	-	-	-	-	-	-
Total with symptoms	1	1	2	2	4	6	3	-	-	9
History unobtainable	-	-	-	6	4	6	8	2	2	28
No previous symptoms	-	-	4	2	7	2	5	1	-	21
Previous cerebral thrombosis	-	1	-	-	-	1	-	-	-	2
Total in each age-group	1	2	6	10	15	15	16	3	2	70

commonest symptom. Apart from dyspnoea, however, the incidence of these other symptoms is the same in hemiplegics and in hypertensives as a whole. This is shown in the accompanying Table 21.

TABLE 21. COMPARISON PRE-HEMIPLEGIA AND HYPERTENSIVE SYMPTOMS

Symptom	Males		Females	
	Hemiplegia	Total with DH	Hemiplegia	Total with DH
Headache	15 (28.8%)	61(21.9%)	11(26.2%)	63(21.5%)
Vertigo	10 (19.2%)	58(20.8%)	6(14.3%)	43(14.6%)
Blurring of vision	10 (19.2%)	22(7.8%)	4(9.5%)	25(8.5%)
Dyspnoea	7 (13.4%)	134(49.6%)	7(16.7%)	128(43.6%)
Total in each group	52	278	42	293

These findings, therefore, do not corroborate those of Taylor and Page. There are apparently no symptoms which can be interpreted as giving warning of an imminent cardio-vascular disaster. Dyspnoea, however, presaging cardiac failure, is an uncommon prodromal complaint. This may mean that patients already having incipient cardiac failure are less prone to cardio-vascular accidents.

Section (11).

Reasons for Admission to Hospital. /

Section (11).

Reasons for Admission to Hospital.

It seemed of interest to discover how many patients were admitted to hospital primarily because of their hemiplegia and how many for other reasons. Tables 22 A & B show the reasons for admission in the 182 patients with hemiplegia uncomplicated by cardiac failure or auricular fibrillation, 73 men (71.57%) and 57 women (71.25%) were admitted directly because of the occurrence of the hemiplegia. 17 men (16.67%) and 20 women (25.00%) came in because of other medical conditions, and the remaining 12 men (11.76%) and 3 women (3.75%) were admitted because of the inability of those who were looking after the patient at home to continue to do so. Neither age nor sex had any influence on the proportions of patients who were admitted for social reasons.

Amongst those who came in for social reasons or because of some other medical condition, the average time since the onset of the hemiplegia was two years in men, and three years and nine months in women, with a range from one month to 17 years. In a considerable number of these patients there was still a marked disability.

Conclusions:

Hemiplegia is most commonly found in patients with hypertension. In this survey it occurred with equal frequency in/

TABLE 22.

HEMIPLEGIA - REASON FOR ADMISSIONA. MALES:

	35- 39	40- 44	45- 49	50- 54	55- 59	60- 64	65- 69	70- 74	75- 79	80- 84	85- 89	To- tal
Hemiplegia	1	1	-	6	6	14	18	9	12	5	1	73
Other med. condition	-	-	-	-	5	5	4	3	-	-	-	17
Surgical condition	-	-	-	-	1	-	-	-	-	-	-	1
Social condition	-	2	-	-	-	1	4	2	2	1	-	12
Total	1	3	-	6	12	19	26	14	14	6	1	102

B. FEMALES:

	35- 39	40- 44	45- 49	50- 54	55- 59	60- 64	65- 69	70- 74	75- 79	80- 84	85- 89	To- tal
Hemiplegia	-	1	2	4	9	13	13	10	3	2	-	57
Other med. condition	-	1	-	3	1	-	4	6	-	-	-	15
Surgical condition	-	-	-	-	-	1	-	2	2	-	-	5
Social condition	-	-	-	1	-	2	-	-	-	-	-	3
Total	-	2	2	8	10	16	17	18	5	2	-	80

in men and women, and in patients of either sex under and over the age of 60 years. Hemiplegia occurred rather more often on the right than on the left side.

There were no symptoms which specifically gave warning of the likelihood of apoplexy. In approximately half of the patients whose history was known, the onset of apoplexy was the first sign of hypertension. This was true for both men and women as well as for the over-60 and the under-60 age-groups.

Dyspnoea was complained of significantly less often by hemiplegic patients, and it was postulated that cardiovascular catastrophe might occur less often where incipient cardiac failure was already present. Congestive cardiac failure, however, did occur along with hemiplegia in a few cases. In two of these, the onset of hemiplegia preceded that of cardiac failure, showing that the apoplectic patient is not always immune from the cardiac consequences of hypertension.

The majority of the patients were admitted into hospital because of the hemiplegia, and only a few were admitted for social reasons or because of some other medical condition. Some of those in the latter category had had hemiplegia for several years, and in some cases there was still a very marked disability.

About half of those admitted with hemiplegia died in hospital/

hospital, some after many months. Of the survivors, the majority had recovered sufficiently by the end of three months to be discharged from hospital, and only nine were kept in for more than six months. The prognosis as regards life and duration of stay in hospital was as good in men as in women, and in those with right as in those with left-sided hemiplegia.

Warren has recently emphasised how important to hemiplegic patients are a hopeful and encouraging environment and active treatment. She has outlined her scheme of treatment, and has stated that there is probably no field where interest and ingenuity on the part of medical, nursing, and physiotherapy staffs reap a greater reward. In Stobhill Hospital, great interest has been shown by the nursing and physiotherapy staffs in the re-education and rehabilitation of hemiplegic patients. While they remain in hospital longer than do patients with other forms of hypertensive disease, their period of treatment compares not unfavourably with the three months in which, Warren says, satisfactory progress may be made.

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CHAPTER V.

Congestive Cardiac Failure

1. The Incidence of Cardiac Failure in Hypertension.
2. The Influence of Age.
3. The Significance of Sex.
4. The Relation to Hypertension.
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Chapter V.

Congestive Cardiac Failure.

1. The Incidence of Cardiac Failure in Hypertension.

"Congestive cardiac failure is the commonest cause of death among patients with Hypertension." So begins a chapter in a recent review on Hypertension by Page and Corcoran. The authors do not indicate, perhaps wisely, what is the incidence of cardiac failure. For here again, the problem of sampling of the population becomes important. Thus Paullin, Bowcock and Wood in 1927 found cardiac failure in only 6.6% of their cases gleaned from private practice. In 1912, Janeway stated that 29 out of his 100 deaths were due to cardiac insufficiency, and Murphy in 1933, who divided his patients into three groups depending on the type of hypertension, found that the proportions of patients in whom heart disease was the cause of death varied in these groups from 23.3% to 56.4%.

Among my cases, of 334 men with diastolic hypertension 75 developed congestive cardiac failure and another 15 had auricular fibrillation in addition to cardiac failure - a total of 90 or 26.95%. Among the 327 women, the corresponding figures were 41 and 15 - a total of 56 or 17.13%.

2. The Influence of age.

The incidence of cardiac failure in each of the 5-year age-groups is shown in Tables 8 A and B. Among men, the incidence in the under 60's was 32.58% (29 out of 89 patients), and in those over 60 years of age it was 24.90% (61 out of 245). The incidence among women was for those under 60 - 20.51% (24 out of 117); for those over 60 - 15.24% (32 out of 210). There is no statistically significant difference between the proportions under and over 60, either in men or in women.

3. The Significance of Sex.

When, however, the proportions of men and women developing cardiac failure are compared, it is found that there is a significant difference between them. Both in the age-group under 60 and that over 60, the proportion of men who had cardiac failure was significantly greater than that of women.

4. The Relation to Hypertension.

Among systolic hypertensives, 27 out of 113 men (23.90%) and 14 out of 81 women (17.28%) developed congestive cardiac failure. Between the proportions of patients with diastolic hypertension and those with systolic hypertension who developed cardiac failure, there was no significant difference. Whereas apoplexy is almost exclusively associated with hypertension, /

hypertension, congestive cardiac failure can result from many causes and in every decade. Even so, however, Howell (1944) found that high blood pressure is the commonest cause of cardiac failure in old age. In Order to examine more closely the relation of hypertension to cardiac failure, all the cases of cardiac failure which had occurred in the medical wards of the hospital during the years to which the survey applied, were examined and classified according to age and cause of failure (as far as could be ascertained). The results are shown, for men and women separately, in Tables 23 A & B.

As age increased, those whose cardiac failure had its origin in rheumatic valvular disease disappeared, and after the age of 30 hypertension began to become prominent. Auricular fibrillation could produce cardiac failure at all ages. Syphilitic aortic incompetence causing cardiac failure appeared in middle life. Again I have included patients with chronic bronchitis and emphysema.

There remained a large group of cases to which a cause for failure could not be assigned - whether these patients could be labelled "arteriosclerotic heart failure" or whether they represented patients whose blood-pressure, originally high enough to produce cardiac failure, had now fallen, I am able only to speculate.

The/

TABLE 23. ETIOLOGY OF CARDIAC FAILURE WITH REGARD TO AGE.

A. MALES:

Cause	10-14	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85-89	90-94	Total
With D.H.	1	1	1	1	1	2	1	4	10	20	17	20	16	11	1	1	1	103
With S.H.	1	1	1	1	1	1	1	1	1	3	3	5	11	4	1	1	1	27
Rheumatism	10	10	8	4	2	1	3	1	1	1	1	1	1	1	1	1	1	38
Aur. Fibr.	1	3	1	1	3	3	4	5	5	2	7	7	4	4	2	1	1	51
Subac. Bact.	1	1	2	1	3	1	1	1	1	1	1	1	1	1	1	1	1	6
Ao. Incomp.	1	1	1	1	1	1	1	5	2	1	8	6	1	1	1	1	1	23
Chr. Bronc.	1	1	1	1	1	1	1	9	13	10	7	6	3	1	1	1	1	49
Ac. Infect.	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Indeterminate	1	1	1	1	4	3	11	17	18	17	26	33	26	22	6	2	1	186
Total	10	13	11	5	13	9	19	40	49	55	68	77	61	42	9	2	1	484
	Under 60 = 224										Over 60 = 260							

B. FEMALES:

Cause	10-14	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85-89	90-94	Total
With D.H.	1	1	1	1	1	1	1	8	9	5	8	16	15	8	2	1	1	73
With S.H.	1	1	1	1	1	1	1	1	1	1	3	2	1	4	2	1	1	15
Rheumatism	2	2	1	1	1	6	2	1	3	1	1	1	1	1	1	1	1	19
Aur. Fibr.	1	2	4	3	9	9	16	10	3	2	5	2	4	1	1	1	1	70
Subac. Bact.	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Ao. Incomp.	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	5
Chr. Bronc.	1	1	1	1	1	1	1	1	2	2	3	9	4	2	3	1	1	25
Ac. Infect.	1	1	1	1	1	1	1	1	1	1	1	1	3	3	3	1	1	7
Indeterminate	1	1	1	1	1	2	4	5	9	3	13	15	14	11	6	3	1	85
Total	3	4	5	4	11	18	24	25	28	15	32	44	41	25	16	4	1	300
	Under 60 = 137										Over 60 = 163							

The most frequent cause of cardiac failure after the age of 60, in both sexes, was hypertension. Hypertension was the cause of cardiac failure significantly more commonly in women than in men in the older age-group (in 51 out of 163 women (31.29%) and in 65 out of 260 men (25.00%)).

This may reflect the fact that hypertension is commoner in women than in men.

5. Cardiac Failure as a cause of death in Hypertension.

Table 12 shows that of 128 deaths in men with diastolic hypertension, 44 were caused by cardiac failure, and among women 20 out of 94 deaths were due to this cause. Of deaths under the age of 60 years, 13 out of 25 (52.00%) in men and 4 out of 23 (17.39%) in women were due to cardiac failure. Over 60 years, 31 deaths out of 103 (30.10%) in men were due to cardiac failure, and we may therefore state that in older men, death from cardiac failure was not so likely as in younger men. In women, there was no significant difference between the proportions of deaths due to cardiac failure in persons under and over 60 years. (Among women over 60 years, 16 out of 71 deaths - i.e. 22.53% - were due to cardiac failure.)

Men significantly more often than women died of cardiac failure, and the difference was seen especially between men and/

and women in the younger age-group. This was due to the fact, however, that cardiac failure occurred more often in men, for after it had developed, the prognosis, as regards life, was found to be the same in both sexes.

6. The Prognosis in Hypertensive Cardiac Failure.

90 men had cardiac failure, and of these 44 (48.89%) died, while of 56 women who had cardiac failure, 20 died (35.72%). There is no significant difference between these figures.

In men, death occurred in 13 out of 29 patients (44.82%) under 60, and 31 out of 61 (50.83%) over 60. There is no significant difference here, but the younger women did not so commonly die from hypertensive cardiac failure, for there were 4 who died out of 24 aged less than 60 (16.63%), but 16 out of 32 (50.00%) aged more than 60.

From the figures given above, gleaned from Table 24, it will be realised that the prognosis as regards life in hypertensive cardiac failure is not good. Daley, Ungerleider, and Gubner (1943) indicated increased mortality where left ventricular hypertrophy (determined by electrocardiographic means) became more marked. Flaxman (1937) discussed the course of hypertensive heart disease, and found that of 623 "uncomplicated" cases, 189 had died - 85% of these within one year of the onset of cardiac failure. In 1938, he stated that, although cardiac/

TABLE 24.

DURATION IN HOSPITAL OF FATAL CASES HYPERTENSIVE
CONGESTIVE CARDIAC FAILURE.

	Less than 1 wk.	1-2 wks.	2-3 wks	3-4 wks	1-2 mths	2-3 mths	3-6 mths	6-9 mths	9-12 mths	12-15 mths	15-18 mths	Total
MALES	16	8	6	4	4	3	2	-	1	-	-	44
	38 (86.36%)					6 (13.64%)						

FEMALES	4	1	2	2	4	2	5	-	-	-	-	20
	13 (65.00%)					7 (35.00%)						

TABLE 25.

DURATION IN HOSPITAL OF NOT-FATAL CASES HYPERTENSIVE
CONGESTIVE CARDIAC FAILURE.

	Less than 1 wk.	1-2 wks.	2-3 wks.	3-4 wks.	1-2 mths	2-3 mths	3-6 mths	6-9 mths	9-12 mths	12-15 mths	15-18 mths	Total
MALES	2	2	1	3	20	8	7	2	1	-	-	46
	28 (60.87%)					18 (39.13%)						

FEMALES	2	1	3	3	11	2	5	2	1	-	-	30
	20 (66.67%)					10 (33.33%)						

cardiac failure might not develop till the patient had had essential hypertension for many years, after its appearance the duration of life was very short - 77% of his patients dying within six months.

From Table 24, it can be seen that almost all of the fatal cases among my series had died within six months, the majority within two months.

Among non-fatal cases (Table 25), most were able to go home by the end of two months; a few patients (and Flaxman also mentions this possibility) after adequate digitalisation remained able to go about and even to perform light work.

7. The Prognosis of Hypertensive Cardiac Failure compared with Heart Failure of other origin.

The prognosis in all types of congestive cardiac failure is poor. Boyer, Leach, and White followed 748 patients with congestive cardiac failure (from many causes) for three months. They found that over 50% of these patients died within that time or remained in failure at rest, no matter what their age or the precipitating cause of the cardiac failure. The prognosis over the age of 70 years was very bad indeed. Reviewing the underlying causes and precipitating factors in 1,000 patients with congestive cardiac failure, they indicated the importance

of/

of the precipitating factor in prognosis, for its nature is a measure of myocardial reserve before the onset of congestion. This need not be stressed further, for the difference between the prognosis of congestive cardiac failure in a child where it is likely that the rheumatic infection is still active, and that in an adult where it is quiescent, is well known.

Examination of Table 23 shows that there is only one other cause of cardiac failure affecting approximately the same age-groups as does hypertension - namely, aortic incompetence. A comparison of the mortality rates in these two conditions is of interest. Among men between the ages of 45 and 75, there were 87 cases of hypertensive heart failure, 53 of whom died in hospital. On the other hand, there were 23 cases of aortic incompetence in the same age-groups, and of these, 8 died in hospital. The difference between these figures is statistically significant. In women, there were only 5 cases of aortic incompetence - an inadequate number for an informative comparison. Among those men who improved, 10 out of the 33 survivors with hypertensive heart disease were in hospital for more than two months. The corresponding figures for those with aortic incompetence are 6 out of 15, and there is no significant difference between these figures.

8. Conclusions.

Over the age of 60 years, cardiac failure, especially in women, /

women, was probably most often caused by hypertension, and this was directly due to the large number of cases of hypertension over the age of 60 years, for cardiac failure occurred as a sequel to hypertension just as often under as over 60. It was, however, more often a cause of death in hypertension in men under 60 than in men over 60 years.

Cardiac failure occurred in hypertensive men more often than in hypertensive women.

The prognosis as regards life was poor when hypertensive cardiac failure had developed, except perhaps in women of the younger age-group. Most patients had a stay in hospital of less than two months.

Death occurred relatively more often in men with hypertensive cardiac failure than in men of the same age-groups having cardiac failure as the result of aortic incompetence.

9. Discussion - The Cause of Cardiac Failure in Hypertension

It is not difficult to conceive that when there is an increase in the resistance offered by the capillaries to the passage of blood through them, the heart must beat more vigorously to produce the rise in arterial pressure which will allow the circulation in the capillaries to be maintained. If the heart undertakes increased work for a considerable/

considerable time, hypertrophy and hyperplasia of the muscle cells will follow, and the mass of cardiac muscle will become enlarged.

The cardiac muscle is supplied with blood by the coronary arteries, and in cardiac hypertrophy the arteries cannot carry a blood supply increased to be commensurate with the needs of the hypertrophied muscle. The myocardium of the hypertrophied heart is therefore ischaemic and rendered less capable of responding when, on exertion, an increased energy output is required of it. In health, the coronary arteries can dilate in some measure under the influence of various reflex and chemical stimuli, but, as it happens, the coronary arteries are peculiarly liable to become the seat of "arteriosclerotic" changes early in life. There is much disagreement about the exact changes designated by the term "arteriosclerosis." Suffice it to say for the present that in the coronary arteries, it is not unusual to find in the third decade atheromatous plaques encroaching on the lumen of the vessels and that by middle life the tunica intima of the arteries is also thickened. Later the elastic layer fragments, the arterial walls become, in large measure, fibrous, and eventually calcification may ensue. These changes, involving encroachment on the lumen of the vessels and hardening/

hardening of the vessel walls, we can for the moment call "coronary arteriosclerosis." Should they occur in a normal heart, myocardial ischaemia might be produced; in a hypertrophied heart, this is much more likely.

Davis and Klainer (1940) found on examination of 61 patients with angina pectoris, 40 of whom had hypertension, and 21 of whom had none, that an extreme degree of coronary sclerosis was seen in 95% of those without hypertension, and in only 39% of hypertensives. If the occurrence of angina pectoris is an index of myocardial ischaemia, then where there is no sclerosis of the vessels myocardial ischaemia is more easily produced in the presence of hypertensive cardiac hypertrophy.

The basic defect, then, is a disproportion between the myocardial need for blood and the ability of the coronary arteries to supply that need. The fault may be myocardial, if there is excessive strain and demand for work, or arterial, if the capacity to supply the required flow of blood or to increase it during effort is reduced.

Should myocardial ischaemia persist, fibrosis of the myocardium develops - the so-called "chronic myocarditis". Christian (1918) defined chronic myocarditis as chronic heart disease developing after the age of 40 due to primary failure of the heart muscle and unassociated with valvular defect./

defect. Clawson (1923) showed that this process had nothing to do with inflammation, but was found only when coronary sclerosis was present.

10. The Manifestations of "Chronic Myocarditis."

As Pardee stated in 1934, there are varied initial manifestations of arteriosclerotic heart disease. The slow development of shortness of breath and fatigue leading to congestive cardiac failure, is, in chronic myocarditis, rendered likely, from incapacity of the damaged heart muscle to deal with the required amount of work. Depending upon the part of the myocardium involved, there may supervene rhythm changes or alterations in the conduction of cardiac impulses. Should auricular fibrillation develop, it probably hastens the onset of cardiac failure. If the conditions in the coronary arteries are suitable for the development of a thrombus, the process of fibrosis may be interrupted by the occlusion of an artery or one of its branches and the production thereby of a myocardial infarct; or should there occur marked vasoconstriction of a sclerotic coronary artery or temporarily increased myocardial demand, anginal pain may result.

The incidence of coronary sclerosis is much higher in men than in women, especially below the age of 60 (Davis and Klainer, 1940) and this finding is paralleled by the varying incidences/

incidences of coronary thrombosis in men and women, for only 25% of the patients in the series of Baer and Frankel (1944) and only 31% in that of Rosenbaum and Levine (1941) were women.

Hypertension has been found to be a frequent precursor of chronic myocarditis (so-called) and of coronary thrombosis. Fahr (1923), defining chronic myocarditis as Christian defined it above, concluded that "hypertension heart" was its most common form and that chronic or acute infection played a minor role in the production of heart muscle disease. Chambers (1947) and Baer and Frankel (1944) have shown that the incidence of antecedent hypertension in coronary thrombosis is greater than in the ordinary population. Furthermore, while coronary thrombosis is not uncommon in men, it seldom occurs in women except in the presence of hypertension. In the series of cases of coronary thrombosis of Rosenbaum and Levine (1941) 86% of the women and 44% of the men had hypertension, and similarly statistically significant figures are reported by Baer and Frankel (1944) for their series.

Congestive cardiac failure in hypertension is almost invariably associated with coronary sclerosis. For instance, Bell and Clawson (1928) reported that only 10% of the hypertensive hearts were unassociated with coronary disease.

Auerbuck, /

Auerbuck, in 1936, examined the clinical histories and post-mortem material of 70 cases of essential hypertension, of whom 40 had had cardiac failure in their last illness, while the remaining 30 had died from other causes. In the hearts of both groups there was left ventricular hypertrophy, but in the great majority of those who had had hypertensive cardiac failure, there was consistent involvement of the coronary arterial system with correspondingly severe myocardial changes, while in the non-cardiac failure group, the coronary system was only slightly affected. Davis and Klainer (1940), also, examining the coronary vessels of 407 autopsied cases, found the incidence of coronary sclerosis markedly increased where there had been hypertension during life.

Perhaps, then, the confusion existing at present between the terms "hypertensive heart disease" and "arteriosclerotic heart disease" is not so important as would appear, if when persons with hypertension develop cardiac symptoms, the clinical picture, as Pardee observed in 1934, is similar to that in coronary disease.

The fact that coronary sclerosis develops more commonly in men than in women explains why, among the hypertensive patients discussed above, congestive cardiac failure was more common in men. Judging from my figures, however, it would appear that coronary sclerosis develops in the hypertensive patient as often under as over the age of 60 years.

Congestive cardiac failure among my patients may have developed as the result of any of the initial manifestations of coronary sclerosis, as described by Pardee.

(a) Shortness of Breath.

Auricular fibrillation or coronary thrombosis may, as I shall show, help to precipitate cardiac failure, or the slow process of the development of myocardial strain and fibrosis may carry on to its inevitable end. The failure of the diseased myocardium to maintain the circulation becomes first apparent during effort, but later there is failure to maintain the circulation even when the patient is at rest. Allbutt calls this "cardiac defeat", and it is the physician's task to hold this in check, if he can. As we have seen, the prognosis is poor for the hypertensive patient when congestive cardiac failure has developed.

In Chapter VII I shall deal with the frequency of dyspnoea in hypertension. Among my patients, dyspnoea on effort was the commonest indication of the presence of "hypertensive heart disease."

(b) Auricular Fibrillation.

In Table 23 (Etiology of Cardiac Failure with Regard to Age), it has been shown that in all age-groups were to be found a small proportion of patients whose congestive cardiac/

cardiac failure was associated with auricular fibrillation. I use the word "associated" because auricular fibrillation often supervenes on rheumatic carditis or in hyperthyroidism - and the clinical picture evoked by the aggravation, in dramatic fashion, of cardiac decompensation when auricular fibrillation develops is too well known to need description. In Part III, I shall discuss the clinical features of auricular fibrillation in old age. The absence of incapacity in old age has been reported by several authors.

Auricular fibrillation is evidently not a "complication" much to be feared in essential hypertension. Although Flaxman (1937) found it in 158 out of 623 cases of so-called "uncomplicated" hypertensive heart disease, it is mentioned only, but not hailed as important, in reviews on hypertension, (Page and Corcoran: 1945). It may arise after coronary thrombosis or it may arise without it, as the result of myocardial fibrosis. Among my hypertensive patients it was not very common, occurring only in 19 male (5.69%) and 21 female (6.42%) patients. Four of the men and six of the women had auricular fibrillation without signs of cardiac decompensation.

When the proportions of diastolic and systolic hypertensives having auricular fibrillation were compared, there was found to be no significant difference between them. Two male patients (1.77%) and three female patients (3.70%) in/
in/

in the systolic hypertensive group had auricular fibrillation. Between the proportions of hypertensive patients having auricular fibrillation in the age-groups over and under 60, there was no significant difference, for of the 19 male patients 4 (4.49%) were under 60 and 15 (6.12%) were over 60. Among the female patients, the corresponding figures were 9 (7.69%) and 12 (5.72%). (The percentages refer to the percentage of the total in each age-group). Nor did the sex of the patients affect the numbers who developed auricular fibrillation.

Fahr (1935) and Flaxman (1937) were of the opinion that in some patients, but not necessarily all, auricular fibrillation adversely influenced the course of hypertensive heart disease.

(c) Coronary Thrombosis.

Coronary atherosclerosis, whether latent or marked by angina pectoris or congestive failure, may be complicated by thrombotic occlusion whenever the conditions for intravascular clotting, i.e. slowing of the blood stream, and atheromatous change in the tunica intima, are fulfilled. Horn and Finkelstein (1940) did not ever find a thrombus in a normal coronary artery. The area of myocardium denuded of blood undergoes infarction, and the heart's action is impeded to a variable degree, depending upon the size and site of the thrombus./

thrombus. The beat ceases in the infarct, which often dilates in systole. These myocardial changes are attended by characteristic physical signs and sequelae. Pain is perhaps the most marked individual feature, and is presumed to be due to the stimulation of the nerve endings at the edge of the infarct by accumulated metabolic products.

Much of our knowledge of coronary thrombosis is of relatively recent origin, and it is now considered to be much more frequent than was formerly recognised. Hedley (1939) gives figures for the percentage of deaths from heart disease caused by coronary thrombosis - the peak figure being 26% in the 50-59 age-group.

Strangely enough, in spite of the fact that, as I shall show later, the degree of arteriosclerosis in the radial artery continues to increase with advancing years, and that Willius and Smith (1932) found on autopsy examination moderately severe arteriosclerosis in all their 381 patients over the age of 70 years, coronary thrombosis is not primarily a disease of old age. In Hedley's cases, which were men only, the maximum incidence of coronary thrombosis was in the seventh decade, and in Rosenbaum and Levine's cases (1941) the maximum incidence for men was in the sixth decade, and in the seventh decade for women.

Many/

Many of the patients who recover after coronary thrombosis develop congestive cardiac failure - Rosenbaum and Levine suggest that cardiac failure ensues in 75% of patients and the figures of Baer and Frankel are 40 and 60 percent for men and women respectively. This observation of Baer and Frankel regarding the greater incidence of cardiac failure after coronary thrombosis in women fits in with that of Boyer, Leach, and White that a greater number of hypertensive patients than of non-hypertensives will develop congestive cardiac failure after coronary thrombosis.

In view of the importance given to coronary thrombosis in the literature, it is notable that in my series of cases it occurred so seldom - in all in 8 men and 4 women. The method of selection of cases is partly responsible, those patients manifesting acute symptoms being more likely to gain admittance to a voluntary hospital. It is possible that among my patients sensitivity to pain is rather less than among other reported groups, and that some patients had a "silent" coronary thrombosis.

Dressler (1944) has discussed unusual modes of onset in coronary thrombosis. Thrombosis without pain, i.e. "silent" thrombosis, has been reported on many times, though there is disagreement about its incidence. Rosenbaum and Levine thought that 3% of their cases might have a painless onset. Gorham and Martin (1938) suggested a figure between
38/

38 and 61%, Stroud and Wagner (1941) reported "silent" thrombosis in 13 of their 49 cases, and Kennedy (1937) in 4% of his. Thewlis (1941) suggests that coronary thrombosis is more liable to occur without pain in old people.

(d) Angina Pectoris.

The typical history of the patient with this disorder is of recurrent attacks of discomfort in or near the chest, induced by conditions which impose an additional burden on the heart. The mechanism is believed to be that of arterial vasoconstriction and the pain is due to disturbances of the oxidative processes in the myocardium. The vasoconstriction may be caused by reflex stimulation from the skin (e.g. exposure to cold), from the respiratory passages (e.g. breathing cold air, walking against the wind), or from disorders of the gastrointestinal tract.

Summers (1948) in his review of angina pectoris, emphasises the importance of hypertension and of coronary sclerosis as predisposing factors, especially in women.

The story in my cases was so often not clear-cut, that I have included patients with angina pectoris among other types of præcordial pain, and have dealt with them in Chapter VII, Part 2.

11. Summary.

Myocardial hypertrophy, the result of hypertension, induces a relative ischaemia of the myocardium, which may give rise to angina pectoris. At the same time, hypertension seems to have some effect in predisposing to coronary sclerosis, which leads to "chronic myocarditis", and so to congestive cardiac failure.

From the clinical point of view, the development of chronic myocarditis may be marked in four different ways.

It follows that patients without hypertension, if they have arteriosclerosis of the coronary arteries, may also develop clinical manifestations as described above. Thus the confusion existing between the terms "hypertensive heart disease" and "arteriosclerotic heart disease" is justifiable.

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CHAPTER VI.

- a) Malignant Hypertension.
 - b) Retinitis.
 - c) Renal Failure.
 - d) Subarachnoid Haemorrhage.
 - e) Hypertensive Encephalopathy.
 - f) The Lungs in Hypertension.
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a). Malignant Hypertension.

Volhard and Fahr in 1914 were the first to describe a rapidly developing type of hypertension, and in 1928 Keith, Wagener, and Kernohan presented their classical description of 81 patients with hypertension and characteristic retinitis, in whom the outcome was rapidly fatal. Murphy and Grill in 1930 published the results of their studies, confirming and extending the observations of their predecessors. But the chief difficulty in the way of full recognition of the nature of this syndrome lay in the fact that the histological picture in the kidney (the so-called "chronic interstitial nephritis") could not be distinguished from that in chronic nephritis.

The clinical and pathological work of Ellis (1942) and the experimental work of Wilson and Byrom (1941) have gone far to elucidate the problem. Wilson and Byrom showed that severe hypertension itself, independent of any toxic or inflammatory process, could produce the necrosis and endarteritis of arterioles and tubular atrophy, which lead on to the changes characteristic of the end-stages alike of hypertension and of nephritis.

Ellis has shown that, in patients dying in the early stages of malignant hypertension, arterial changes in the kidney may be very slight - which is consistent with the view that renal vascular changes are the result and not the cause/

cause of hypertension. At present, so far as is known, the factor determining the malignant character of hypertension can be attributed only to excessive individual response of the renal and extrarenal arterioles to some unknown stimulus (probably the renin-hypertensin mechanism).

Page (1939) has recently reviewed 30 cases of malignant hypertension, which show clearly the patients' comparative youth, headache as the predominant complaint (13 patients), and uraemia as the main cause of death (10 cases). Ellis in 1938 emphasised the importance of papilloedema as a diagnostic sign, and has stated that the diagnosis of malignant hypertension should not be made unless papilloedema is present.

In my survey, there were five cases of malignant hypertension, all of whom were women. Four of them were young (under 40), and one was over 60 years of age. All had headache and dyspnoea, all had papilloedema, and all died of renal failure. In each case, the course of the illness was very short, not exceeding twelve months.

b). Retinitis.

Four female patients, three under 60 and one over, came under notice because of arteriosclerotic retinitis. Two of these younger patients had no sign of renal impairment, though they complained of severe headache and were dyspnoeic on/

on moving about the ward. The other two patients died of renal failure.

The changes observed in the retinal vessels have been given importance by Keith, Wagener, and Barker (1939), who graded hypertensive patients, according to the retinal changes, into four categories with reference to the severity of the disease and prognosis.

Group I. The retinal changes are slight, and there are no marked alterations in the general health.

Group II. The retinal changes are more marked, but there is no retinitis. The disease is more progressive, but the general health is good and cardiac and renal functions are satisfactory.

Group III. Retinitis is present, and the hypertension is high and sustained. There may be alterations in cardiac and renal functions, and symptoms, such as nervousness, headache or vertigo may appear.

Group IV. The changes characteristic of malignant hypertension are present.

The retinal changes in Group I are usually those ascribed to retinal arteriosclerosis, which I shall describe in more detail in Part III. "Retinitis" is constituted by the occurrence of flame-shaped haemorrhages and "cotton-wool" exudates.

Among/

Among my patients, considering the cases of malignant hypertension and severe retinitis together, we find that only two over 60 years of age were affected, compared with seven in the younger group, i.e. these two conditions occurred about eight times more frequently in those under 60 than in those over - a difference that is also statistically significant, in spite of the small numbers involved.

It is, therefore, unlikely, though not improbable, that when hypertension occurs in old age, it should be of severe and rapidly progressing type.

c). Renal Failure.

Among the patients in this series, two men and one woman died of renal failure, all being over 60 years of age.

In discussing the etiology of hypertension, we have seen how it is that renal arteriolar constriction begets hypertension and renal arterio-sclerosis. This arterio-sclerosis by means of its restriction of the kidney's blood flow, produces loss of renal functional tissue; and so surely and inexorably progress is made towards renal failure. In fact, renal failure is the irrevocable end for every hypertensive patient whose course is not cut short by the development of another abnormality. On the other hand, it must be admitted that patients with chronic nephritis can tolerate severe renal injury and survive for long periods compared with/

with malignant hypertensives whose renal injury is relatively slight (Corcoran and Page, 1944), and Page and his co-workers (1944) have suggested that this difference is due to the more severe heart disease and more general blood vessel disease in malignant hypertension.

We have seen that the uraemia (the name given to the clinical syndrome resulting from renal failure) of malignant hypertension is brief and stormy, and, as Rosenberg has shown, complicated by cerebral injury. But the uraemia which my three patients showed was a slow, deliberate process - the foul breath, the gastric disturbances, the headache, the apathy of the initial stages being followed only after several weeks by marked evidences of dehydration and of acidosis. Ellis (1942) has described a few cases of this kind, who seemed to develop a chronic renal lesion - pathologically a "chronic interstitial nephritis" - following on essential hypertension. Fishberg mentions that in older subjects, occasional cases occur in which renal arteriosclerosis from high blood pressure leads to such extensive renal atrophy that renal failure results.

d). Subarachnoid Haemorrhage.

Rapid prostration with severe headache of sudden onset, meningitic irritability and diminution of tendon reflexes with absence of specific paralyses was seen in five men and one/

one woman, two men and the woman being over 60 years of age. In all these cases, blood was present in the cerebro-spinal fluid. It should not be forgotten that in intracerebral haemorrhage, blood sometimes tears its way into the subarachnoid spaces, and without post-mortem examination an exact diagnosis is difficult. The course of the disease in the woman was characteristic; two or three days after admission she was conscious, intelligent, and co-operative, with no evidence of any motor or sensory lesion, but deeply depressed, and complaining of very severe headache. In her case, recovery was eventually complete.

In two of the men, one under 60 and the other over, the sudden onset of subarachnoid haemorrhage was the first indication of any abnormality. (see Table 33A).

e). Hypertensive Encephalopathy.

Hypertensive encephalopathy, as described by Fishberg, resembles an epileptic seizure. Only one of the patients in my series had symptoms and signs of this nature. She was a stout lady, aged 64, who came into hospital complaining of thirst and polyuria of about two weeks' duration. She was discovered to be a diabetic. Six days after admission, symptoms of excessive irritability appeared, with insomnia at night. On the seventh day, epileptiform convulsions occurred several times, the patient being comatose in/

in the intervals between them. Venesection of one and a half pints of blood was performed. Complete recovery from the episode ensued.

The pathogenesis of the syndrome is uncertain. There is evidence to suggest that excessive vasoconstriction occurs during the paroxysm, but its mechanism is obscure. During the years with which this survey deals, hypertensive encephalopathy was observed four times in young children (10-14 years of age) with acute nephritis, and only once (on this occasion) in an adult.

f). The Lungs in Hypertension.

Emphysema: Gull and Sutton (1872) suggested that pulmonary emphysema was an intrinsic manifestation of chronic Bright's Disease. This observation serves to illustrate how commonly are the two conditions associated. There has been much debate about the pathogenesis of emphysema, but it is now more or less accepted that the hypertrophic type results from the effects of forced expiration and coughing, which cause distension of the alveoli, and the atrophic type from "senile" changes in nutrition of the lungs and in the character of the chest walls - whatever the intimate nature of these "senile" changes may be. Even though the two conditions may occur together there is, therefore, no reason for the assumption that hypertension of the greater circulation predisposes to emphysematous/

emphysematous changes. On the other hand, partly because of stretching and compression of the interalveolar arterioles, and partly by destruction of part of the pulmonary capillary bed, emphysema is likely to induce an increased peripheral resistance and so an increased blood pressure, in the lesser circulation, and to lead to right ventricular hypertrophy and failure.

Chronic Bronchitis: Recent writings on this subject have emphasised how seldom is chronic bronchitis the result of the acute form, and how often are its signs and symptoms the result partly of circulatory insufficiency, or pulmonary tuberculosis, or bronchiectasis (Howell 1943; Thewlis 1946). Emphysema nearly always results, sooner or later, and Howell has shown that the prognosis of chronic bronchitis, especially in elderly patients, is often bad because of complicating congestive cardiac failure.

Pulmonary Congestion: In a discussion on dyspnoea in hypertension (Part II, Chapter VII), I have very briefly commented on its cause and suggested that it usually portrays loss of cardiac adaptation. Cardiac dyspnoea is largely, if not wholly, due to the increased rigidity of the lung which follows pulmonary congestion (Christie. 1938). The lungs, then, become very frequently involved as failure of/

of the left ventricle commences. Marked pulmonary congestion is also a feature of the intense nocturnal paroxysmal dyspnoea, which is not uncommon in elderly hypertensive patients - though the trigger mechanism here is still not fully understood.

The onset of loss of cardiac adaptation in hypertension is then marked by dyspnoea, which is an index of pulmonary congestion. As the pressure in the pulmonary circulation rises, right ventricular hypertrophy takes place with, eventually, failure - and at this point dependent oedema is seen. Cardiac failure in hypertension often begins with a period of intense dyspnoea, which disappears when oedema commences. The rapid terminal course of the following patient's illness is an illustration of this:-

A woman of 63 was admitted to hospital on 16.7.46, complaining of increasing breathlessness over the past few months. She had previously always enjoyed good health. She was pale and thin on admission, and suffering from extreme and distressing orthopnoea. There was no oedema. There was no apparent cardiac enlargement on clinical examination, the apex-beat being 9 cms. from the mid-line. The heart-sounds were of good quality, and there were no bruits. The blood pressure was 210 mm Hg. systolic and 140 mm Hg. diastolic, and the limb arteries were grossly thickened and tortuous. Respiration sounds/

sounds over the whole chest were very harsh and prolonged, and accompanied by dry sibilant and sonorous rhonchi.

The patient was treated with sedative and with cardophyllin Tabs. 6 daily (0.6 Gm.). The dyspnoea remained very distressing, until on 29.7.46, oedema commenced in the dependent parts and this was accompanied by a marked diminution in the dyspnoea. Digitalisation was commenced, and as oedema became more marked, Neptal was given, but without appreciable diuretic effect. By 6.8.46 the blood pressure had begun to fall (168/100). On 13.8.46 the blood pressure was 156/96 mm Hg., and the liver was enlarged and tender on palpation. The patient's condition steadily deteriorated with a falling blood pressure, an increasing systemic congestion, a rising blood urea (138 mgms. % on 13.8.46), and after the appearance of a dry, dirty tongue, diarrhoea and abdominal pain, she died on 17.8.46.

Autopsy revealed a hypertrophied and dilated heart, especially on the right side, calcification of the coronary arteries, and gross congestion of lungs, liver, and other organs.

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It is often a matter of difficulty to decide in the elderly patient, whether emphysema, "chronic bronchitis", circulatory failure (or even pulmonary tuberculosis) is the cause of his dyspnoea, coughing, increased bronchial secretion, and chest signs. In Table 8, I have included under the heading/

heading "chronic bronchitis and emphysema" all those patients with such a doubtful diagnosis. 30 men (8.98%) and 12 women (3.67%) with diastolic hypertension, and 14 men (14.14%) and 3 women (3.70%) with systolic hypertension came under this heading. There was no significant difference between the incidences in diastolic and systolic hypertension, or between those in age-groups under and over 60 years. Seven of the men (7.87%) and four of the women (3.42%) among the diastolic hypertensives were under 60, and 23 men (9.39%) and 8 women (3.81%) were over 60. (The percentages refer to the total number of patients in each age-group).

Men, however, both in the under-60 and the over-60 age-groups, more often could be placed in this category than could women. Congestive cardiac failure, as we have seen, is more common in men, and emphysema, also, is described as being more often seen in the male sex (Young and Beaumont: 1937).

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C H A P T E R VII.

The Symptoms Associated with Hypertension.

1. Introduction. A discussion of the nature, character, and incidence of the symptoms in Hypertension.
2. The Mode of Onset of Hypertension.
3. A Discussion of each symptom in turn -
 - 1). Headache
 - (a) The Incidence of Headache
 - (b) The Cause of Headache
 - (c) What Constitutes Headache
 - (d) The Findings in my group of patients.
 - 2). Dyspnoea
 - (a) The Definition of Dyspnoea
 - (b) The Incidence and Cause of Dyspnoea
 - 3). Vertigo
 - (a) The Definition of Vertigo
 - (b) The Cause of Vertigo
 - (c) The Incidence of Vertigo
 - 4). Blurring of Vision
 - 5). Praecordial Pain
 - 6). Palpitation
 - 7). Epigastric Discomfort
 - 8). Weakness
 - 9). Fatigue
 - 10). Depression
 - 11). Insomnia
 - 12). Nervousness
 - 13). Paraesthesiae & 14). Transient Pareses
 - 15). Urinary Frequency
 - 16). Haemorrhages
4. Conclusions.

1. Introduction - A Discussion of the Nature, Character, and Incidence of the Symptoms in Hypertension.

It has been shown, in Tables 8 A & B, that a high blood pressure may be accompanied by gross and easily recognisable disease, or by no signs or symptoms of disease; and again, that even although there may be no gross lesion, there may be subjective complaints. Into this last category fell 22.16% (74 out of 334) of the men and 29.05% (95 out of 327) of the women with diastolic hypertension - proportions not significantly higher than the 23.00% (26 out of 113) of men and the 22.22% (18 out of 81) of women with systolic hypertension who had corresponding complaints and no physical signs of disease. Among the patients with diastolic hypertension, significantly more women than men had symptoms without other signs of disease, and it was above the age of 60 that the distinction took place. (68 out of 210 women, or 32.4%) and (54 out of 245 men, or 22.0%). The relative incidence, however, of those who had symptoms without physical signs in the under-60 and the over-60 age-groups showed no significant difference, among either men or women.

Yet this group does not exhaust the number of patients with subjective complaints, for, although not shown in Table 8, symptoms of varying nature also occurred in patients with heart-failure, hemiplegia, or other manifestations of hypertension./

hypertension. Before, then, it was possible to enumerate the symptoms associated with hypertension, a new subdivision of my cases had to be made, and will be found at the foot of Tables 26 A & B, 27 A & B, and 28 A & B. There were, of course, patients who had no indications of hypertension, and to these I have already referred in Chapter 3. Some patients had signs of disease, which fitted them into one or other category in Table 8, but were so ill that a detailed history of the illness could not be taken. A few patients had had no symptoms of any upset, until the sudden onset of coronary thrombosis, hemoplegia, or in two cases, subarachnoid haemorrhage. The remainder of the patients could be divided into -

- i. those who had symptoms, but no gross disease
- ii. those who had gross disease and symptoms.

The case-records of all the patients were scrutinised, and all the symptoms complained of by each patient were noted - except where these seemed clearly related to a known concurrent disease; for instance, thirst in a diabetic patient with hypertension was considered to be related to the diabetes, but fatigue in the same patient was included in my list. In Tables 26, 27 and 28 will be found the completed lists, classified according to blood-pressure, sex, and age. In Table 29 is listed the symptoms of patients with diastolic hypertension belonging to group (i) above.

The/

The question arises: should, in considering the frequency of symptoms, group (i) alone be taken into account, or, if the symptoms in both groups (i) and (ii) are observed, will the symptoms of the patients in the second group bear on the complications of hypertension as well as on the presence of hypertension itself? Table 8 shows that the most frequently occurring "complications" were hemiplegia, cardiac failure, and bronchitis. Hemiplegia, by itself, is unassociated with symptoms, but in both cardiac failure and bronchitis dyspnoea is a predominant symptom. When the proportion of patients having individual symptoms was compared in the two groups, it was found, as one would expect, that dyspnoea occurred significantly more commonly in the second group than in the first, but that the proportions of those with headache, vertigo, praecordial pain (the symptoms next in order of frequency) showed no statistically significant difference. I have decided, accordingly, to include in this survey of symptoms every patient from whom a history had been taken, and to consider each symptom in such terms. From Tables 26 A & B, 27 A & B and 28 A & B it becomes apparent that the number of patients from whom the history of the illness was received were as follows:-

TABLE 26. SYMPTOMS IN DIASTOLIC HYPERTENSION

A. MALES

	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85-89	TOTAL
Headache	-	-	1	5	15	7	16	10	5	2	-	61
Dyspnoea	2	4	6	14	20	25	27	24	9	7	-	138
" , noct.	2	1	2	5	11	9	5	8	3	2	-	48
Vertigo	-	2	2	2	12	9	11	13	5	2	-	58
Blur./vision	-	-	2	1	5	2	4	5	2	1	-	22
Precord.pain	-	-	1	3	4	5	3	4	1	-	-	21
Palpitation	-	1	1	-	1	2	-	2	2	-	-	9
Epig. discomf	-	1	3	-	-	3	1	-	-	-	-	8
Weakness	-	-	-	3	2	2	6	6	2	2	-	23
Fatigue	-	1	1	1	1	2	3	3	2	1	-	15
Depression	-	-	1	-	3	1	-	1	-	-	-	6
Insomnia	-	-	1	1	2	2	-	-	1	-	-	7
Nervousness	-	-	-	-	2	-	1	-	-	-	-	3
Paraesthesia	-	1	1	-	4	1	-	-	-	1	-	8
Trans.paresis	-	1	1	-	1	1	1	-	1	-	-	6
Urinary freq.	-	2	1	5	10	5	11	12	5	2	-	53
Epistaxis	-	-	-	-	2	1	2	-	-	-	-	5
Haematuria	-	-	-	-	-	-	-	-	-	-	-	-
Haemoptysis	1	1	1	-	-	2	-	-	-	-	-	5
Blood / rectum	-	-	-	1	-	-	-	-	-	-	-	1

No signs or symptoms	-	-	2	2	1	12	8	6	11	1	1	44
Signs but no symptoms	-	1	-	2	1	6	6	4	2	1	1	24
Symptoms but no signs	-	-	4	1	15	9	12	15	12	6	-	74
Symptoms and signs	2	5	7	14	16	29	26	25	6	6	-	136
Signs but no history avail	1	2	-	3	10	7	12	5	13	3	-	56
TOTALS	3	8	13	22	43	63	64	55	44	17	2	334

TABLE 26. SYMPTOMS IN DIASTOLIC HYPERTENSION

B. FEMALES

	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85-89	TOTAL
Headache	1	-	1	1	3	-	4	7	11	13	11	6	4	-	1	62
Dyspnoea	1	-	1	2	3	4	9	15	19	19	23	18	8	5	1	128
" ,nocturnal	1	-	-	1	2	1	1	2	-	4	2	2	1	-	-	17
Vertigo	-	-	-	-	-	1	4	2	6	5	11	10	3	-	1	43
Blur./vision	-	-	-	1	1	-	4	3	6	4	3	3	-	-	-	25
Precord. pain	-	-	-	1	-	-	-	2	1	7	2	2	3	-	-	18
Palpitation	-	-	-	1	-	2	1	5	3	3	1	-	2	-	-	17
Epig. discomf.	-	-	-	1	-	-	-	-	-	-	-	3	-	-	-	4
Weakness	-	-	-	-	-	1	4	1	5	3	7	7	2	-	-	30
Fatigue	-	-	1	1	1	2	5	1	5	5	4	8	1	-	-	34
Depression	-	-	-	1	1	-	-	-	5	1	-	1	-	-	-	9
Insomnia	-	-	-	-	-	1	-	1	1	3	1	3	-	-	-	10
Nervousness	-	-	-	1	1	-	1	2	3	1	-	2	-	-	-	11
Paraesthesia	-	-	-	-	-	1	-	1	2	1	1	1	-	-	-	7
Trans. paresis	-	-	-	-	-	-	-	-	-	-	-	2	-	-	-	2
Urinary freq.	-	-	-	-	2	-	3	5	5	6	2	13	3	-	-	39
Epistaxis	-	-	-	-	-	-	1	1	2	3	3	2	-	-	-	12
Haematuria	-	-	-	1	-	-	-	-	1	-	1	-	-	-	-	3
Haemoptysis	-	-	-	1	-	-	-	1	-	-	1	-	1	-	-	4
Haematemesis	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	1
Blood / rectum	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
" / uterus	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	1

No signs or symptoms	-	-	1	-	2	2	4	8	10	10	11	9	5	5	3	70
Signs but no history avail.	-	-	-	-	-	-	-	1	7	4	6	8	5	3	-	34
Signs, but no symptoms	-	-	-	-	-	-	-	4	2	7	2	5	1	-	-	21
Symptoms, but no signs	-	-	-	3	2	1	5	5	11	15	24	14	10	4	1	95
Sympts.&signs	1	-	1	3	2	3	11	15	13	18	17	14	5	3	1	107
TOTALS	1	-	2	6	6	6	20	33	43	54	60	50	26	15	5	327

TABLE 27 SYMPTOMS IN SYSTOLIC HYPERTENSION

A. MALES

	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85-89	
Headache	-	-	-	1	-	-	2	4	8	1	-	16
Dyspnoea	-	-	-	2	4	4	9	14	10	1	-	44
", nocturnal	-	-	-	-	-	-	-	1	-	-	-	1
Vertigo	-	-	-	-	1	1	1	5	7	1	-	16
Blurring / vision	-	-	-	-	-	-	-	2	1	-	-	3
Praecordial pain	-	-	-	-	-	-	-	-	3	1	-	4
Palpitation	-	-	-	-	-	-	-	2	5	-	-	7
Epigastric discomf.	-	-	-	-	-	-	-	1	-	-	-	1
Weakness	-	-	-	-	2	-	3	1	3	-	-	9
Fatigue	-	-	-	-	1	-	1	2	-	-	-	4
Depression	-	-	-	-	-	-	-	-	-	-	-	-
Insomnia	-	-	-	-	-	-	-	-	-	-	-	-
Nervousness	-	-	-	-	1	-	-	-	1	-	-	2
Paraesthesia	-	-	-	1	-	-	-	-	-	-	-	1
Trans. paresis	-	-	-	-	-	-	-	-	-	-	-	-
Urinary frequency	-	-	-	-	-	1	3	6	3	1	-	14
Epistaxis	-	-	-	-	-	-	-	-	1	-	-	1
Haematuria	-	-	-	-	-	-	-	-	-	-	-	-
Haemoptysis	-	-	-	-	-	-	-	-	-	-	-	-

No signs or symptoms	-	2	2	-	1	1	10	3	4	3	-	26
Signs, but history not available.	-	-	1	-	-	3	5	-	2	3	-	14
Signs, but mental § confusion ...	-	-	-	-	(1)	(1)	(3)	(1)	(1)	-	-	(7)
Signs, but no symptoms	-	-	-	-	1	2	-	3	-	-	-	6
Symptoms, but no signs	-	-	-	-	-	2	3	8	10	3	-	26
Both signs and symptoms	-	-	-	2	4	3	10	14	7	1	-	41
	-	2	3	2	6	11	28	28	23	10	-	113

TABLE 27 SYMPTOMS IN SYSTOLIC HYPERTENSION

B. FEMALES	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85-89	90-94	Total
Headache	-	-	-	1	1	2	-	2	2	2	-	-	-	-	10
Dyspnoea	1	-	-	-	3	2	3	4	4	7	2	2	1	1	30
", nocturnal	-	-	-	-	1	-	-	-	-	-	1	-	-	-	2
Vertigo	-	-	-	-	-	2	2	2	1	4	1	-	-	-	12
Blurring / vision	-	-	-	-	1	-	1	-	1	3	-	-	-	-	6
Praecordial pain	-	-	-	-	1	-	1	-	1	-	-	-	-	-	3
Palpitation	-	-	-	-	2	-	1	-	2	1	-	-	-	-	6
Epigastric discomf.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Weakness	-	-	-	-	2	2	1	1	1	4	2	-	1	1	15
Fatigue	-	-	-	-	-	2	-	-	-	3	-	-	1	-	6
Depression	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Insomnia	-	-	-	-	-	-	-	-	-	-	1	-	-	1	2
Nervousness	1	-	1	-	1	2	-	1	-	-	-	-	-	-	6
Paraesthesia	-	-	-	-	-	-	-	1	-	-	-	-	-	-	1
Trans. paresis	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Urinary frequency	-	-	-	1	1	2	1	2	1	3	-	-	-	1	12
Epistaxis	-	-	-	-	-	-	1	-	1	-	-	-	-	-	2
Haematuria	-	-	-	-	-	-	-	1	-	-	-	-	-	-	1
Haemoptysis	-	-	-	-	-	-	-	-	-	1	-	-	-	-	1

No signs or symptoms ..	-	1	-	1	2	3	2	4	3	8	6	3	-	1	34
Signs, but history not available	-	-	-	-	-	-	-	1	2	2	2	-	-	-	7
Signs, but mental confusion ...	-	-	-	(1)	-	-	-	(1)	(1)	-	(2)	-	-	(5)	
Signs, but no symptoms	-	-	-	-	-	2	-	-	-	-	-	-	-	-	2
Symptoms, but no signs	1	-	1	1	3	1	2	3	2	4	-	-	-	-	18
Both signs and symptoms	-	-	-	1	-	1	1	4	4	2	2	2	2	1	20
Total	1	1	1	3	5	7	5	12	11	16	10	5	2	2	81

TABLE 28.

SYMPTOMS IN PATIENTS WITH NO HYPERTENSION.A. MALES

Symptoms	45-49	50-54	55-59	60-64	64-69	70-74	75-79	80-84	Total
Headache	-	-	-	2	1	-	1	1	5
Dyspnoea	2	3	3	4	2	-	2	2	18
Vertigo	-	1	-	-	-	-	-	2	3
Blurring of Vision	-	-	-	1	-	-	-	-	1
Praecordial pain	-	1	-	-	-	-	-	-	1
Palpitation	-	-	-	-	-	-	-	1	1
Epigastric discomf.	1	-	-	-	1	-	-	-	2
Weakness	-	-	-	1	-	-	-	-	1
Fatigue	-	-	-	2	1	-	-	2	5
Depression	-	-	-	-	-	-	-	-	-
Nervousness	-	-	-	-	-	-	-	-	-
Paraesthesia	-	-	1	-	-	-	-	-	1
Transient paresis	-	-	-	-	-	-	-	-	-
Urinary frequency	1	-	1	-	1	-	-	1	4
Epistaxis	-	-	-	-	-	-	-	-	-
Haematuria	-	-	-	-	-	-	-	-	-
Haemoptysis	-	-	-	-	-	-	-	-	-
Blood / rectum	-	-	-	-	-	-	-	-	-
Total in each group	7	7	4	8	6	8	4	3	47
" with case hist.	7	7	3	8	6	5	4	3	43
No symptoms or signs	5	3	1	-	3	3	2	-	17
Signs, but no hist. available	-	-	1	-	-	3	-	-	4
Signs, but mental confusion	-	-	-	-	-	-	1	1	2
Signs, but no symptoms	-	-	-	1	1	2	-	-	4
Symptoms, but no signs	-	1	1	2	1	-	1	2	8
Signs and symptoms	2	3	1	5	1	-	-	-	12
Totals	7	7	4	8	6	8	4	3	47

TABLE 28.

SYMPTOMS IN PATIENTS WITH NO HYPERTENSION.

B. FEMALES

SYMPTOMS	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	Total
Headache	-	No PATIENTS.	-	-	1	-	-	-	1
Dyspnoea	-		-	-	2	-	-	-	2
Vertigo	-		-	-	-	-	-	-	-
Blurring/vision	-		-	-	-	-	-	-	-
Praecord. pain	-		-	-	-	-	1	-	1
Palpitation	-		-	-	-	-	-	-	-
Epigast. discomf.	-		-	-	-	-	-	-	-
Weakness	-		-	-	-	-	-	-	-
Fatigue	-		-	-	-	-	-	-	-
Depression	-		-	-	-	-	-	-	-
Nervousness	-		-	-	-	-	-	-	-
Paraesthesia	-		-	-	-	-	-	-	-
Trans. paresis	-		-	-	-	-	-	-	-
Urinary freq.	-		-	-	-	-	-	-	-
Epistaxis	-		-	-	-	-	-	-	-
Haematuria	-		-	-	-	-	-	-	-
Haemoptysis	-	-	-	-	-	-	-	-	
Blood / rectum	-	-	-	-	-	-	-	-	
No. of patients	1		-	5	2	3	2	1	15

No signs or Sympt.	-		1	4	-	1	2	1	9
Signs & symptoms	1		-	1	2	2	-	-	6
Total patients	1		1	5	2	3	2	1	15

TABLE 29

SYMPTOMS IN DIASTOLIC HYPERTENSIVES (GROUP WITH SYMPTOMS ONLY)

A! MALES.

Symptoms	AGE			- G R O U P S					Total
	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	
Headache	-	-	5	-	4	1	-	-	10
Dyspnoea	1	1	6	3	1	6	7	4	29
", nocturnal	-	1	-	-	-	-	-	-	1
Vertigo	1	-	7	3	5	6	1	2	25
Blur./vision	-	-	2	-	1	1	-	-	4
Praec. pain	1	-	1	2	-	4	-	1	9
Palpitation	-	-	-	-	-	-	-	-	-
Epig. discomf.	1	-	-	-	-	-	-	-	1
Weakness	-	-	3	1	2	4	1	1	12
Fatigue	-	-	1	1	2	-	-	1	5
Depression	-	-	2	1	-	1	-	-	4
Insomnia	-	-	1	-	-	-	1	-	2
Nervousness	-	-	1	-	-	-	-	-	1
Paraesthesia	-	-	2	-	-	-	-	1	3
Trans. paresis	-	-	-	1	1	-	-	-	2
Urinary freq.	-	1	3	1	3	4	1	2	15
Epistaxis	-	-	1	-	2	-	-	-	3
Haematuria	-	-	-	-	-	-	-	-	-
Haemoptysis	-	-	-	-	-	-	-	-	-
Haematemesis	-	-	-	-	-	-	-	-	-
Blood/rectum	-	-	-	-	-	-	-	-	-
Total in each age-group	4	1	15	9	12	15	12	6	74

TABLE 29.

SYMPTOMS IN DIASTOLIC HYPERTENSIVES (GROUP WITH SYMPTOMS ONLY)

B. FEMALES

Symptoms	Age-groups												Total
	20-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85-89	
Headache	1	1	-	1	3	4	6	4	1	3	-	1	25
Dyspnoea	2	1	1	3	4	5	8	14	3	5	1	-	47
" ,noct.	1	-	-	-	-	-	-	-	-	1	-	-	2
Vertigo	-	-	-	2	1	1	2	-	-	1	-	-	23
Blur./vision	1	1	-	1	1	1	3	2	2	-	-	-	12
Precord.pain	1	-	-	-	1	1	2	1	-	2	-	-	8
Palpitation	-	-	1	-	1	1	2	-	-	1	-	-	6
Epig.discomf	-	-	-	-	-	-	-	-	-	-	-	-	-
Weakness	-	-	1	2	1	2	3	4	4	2	-	-	19
Fatigue	-	-	1	1	1	1	3	4	3	1	-	-	15
Depression	1	1	-	-	-	2	1	-	-	-	-	-	5
Insomnia	-	-	-	-	1	1	2	-	1	-	-	-	5
Nervousness	1	1	-	1	1	2	1	-	1	-	-	-	8
Paraesthesia	-	-	-	-	-	2	-	-	1	-	-	-	3
Trans.paresis	-	-	-	-	-	-	-	-	1	-	-	-	1
Urinary freq	-	-	-	1	1	3	3	1	2	1	-	-	12
Epistaxis	-	-	-	1	-	2	1	3	1	-	-	-	8
Haematuria	1	-	-	-	-	-	-	-	-	-	-	-	1
Haemoptysis	-	-	-	-	1	-	-	1	-	-	-	-	2
Haematemesis	-	-	-	-	-	-	-	1	-	-	1	-	2
Blood/rectum	-	-	-	-	-	-	-	-	-	-	-	-	-
Blood/uterus	-	-	-	-	1	-	-	-	-	-	-	-	1
Total in each age group	3	2	1	5	5	11	15	24	14	10	4	1	95

TABLE 30.

SYMPTOMS IN DIASTOLIC HYPERTENSION - VARIOUS INVESTIGATORS

Symptoms	Douthwaite 1928	Paullin, Bowcock, & Wood 1927	Riseman & Weiss 1930	Aymans & Pratt 1931	Marshall 1932	Self	
						M.	F.
Headache	44.2 %		43.3 %	72 %	62 %	21.9	21.5
Dyspnoea	55.4	11.0 %	27.7	34	42	49.6	43.7
Vertigo	23.3	3.6	40.3	66	15	20.9	14.7
Blur./vision			4.0		19	7.9	8.5
Precord.pain	4.6	8.4	5.3		11	7.6	6.1
Palpitation	51.2		13.1	18	15	3.2	5.8
Epig.discomf					30	2.8	1.3
Weakness			11.6	65	19	8.3	10.2
Fatigue				55		5.5	11.6
Depression						3.2	2.0
Insomnia			5.6	63	11	2.5	3.4
Nervousness			13.2	67		1.1	3.7
Paraesthesia			4.4			2.5	2.4
Trans.paresis						2.2	0.7
Urinary freq		49.0	25.9			19.1	13.3
Epistaxis			5.6		12	1.8	4.1
Haematuria	13.9	5.4				4.0	7.2
Haemoptysis							
Haematemesis							
Blood/rectum							
Blood/uterus							

Kauffman (48 cases) quoted by Riseman & Weiss	"Rheumatism"	68.4%
	Dizziness	58.2
	Hypersensitivity to warmth	47.9
	"Migraine"	43.8
	Morning headache	33.3

TABLE 31.

SYMPTOMS IN MALIGNANT HYPERTENSION

FEMALES

Symptom	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69
Headache	1		1	1	1						1
Dyspnoea	1		1	1	1						1
Blurring/v.	1		1	1	1						1
Praecord. p.	-		-	-	-						1
Urin. freq.	1		1	1	1						1
Total in age-group	1		1	1	1						1

TABLE 32

SYMPTOMS IN CHRONIC NEPHRITIS

A. MALES

Symptom	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	Total
Headache				1	2	1	2	1	2	10
Dyspnoea				-	1	2	3	1	2	9
Weakness				1	-	-	-	-	-	1
Fatigue				1	-	1	-	-	-	2
Praecord pain				-	-	-	1	-	-	1
Urinary freq.				-	1	2	1	-	1	5
Epistaxis				-	1	-	-	-	-	1
Total/age group				1	2	2	3	1	3	12

B. FEMALES

Symptom	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	Total	
Headache	-					2			2	1	5
Dyspnoea	1					2			2	1	6
Weakness	-					1			-	1	2
Fatigue	-					-			-	1	1
Urinary freq.	1					2			1	1	5
Paraesthesia	-					-			1	-	1
Total/age group	1					2			2	1	6

TABLE 33

FIRST SIGN OR SYMPTOM IN DIASTOLIC HYPERTENSION.

A. MALES

	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85-89	Total
Hemiplegia	-	1	-	2	-	6	5	4	2	1	1	22
Subarach. Haem.	-	-	-	-	1	-	1	-	-	-	-	2
Coronary Thrombosis	-	-	1	1	-	3	-	1	-	-	-	6
Headache	-	-	-	-	2	1	2	-	1	-	-	6
Dyspnoea	2	4	5	12	18	22	27	21	9	7	-	127
Vertigo	-	1	2	-	2	4	6	10	4	2	-	31
Blurring/vision	-	-	-	-	1	-	-	1	-	-	-	2
Praecordial pain	-	-	1	-	2	3	-	2	-	-	-	8
Palpitation	-	-	-	-	-	-	-	-	-	-	-	.
Epigastric discomf.	-	-	-	-	-	2	-	-	-	-	-	2
Weakness	-	-	-	1	3	2	1	3	4	-	-	14
Fatigue	-	-	1	-	-	-	1	-	-	1	-	3
Depression	-	-	-	1	-	-	-	-	-	-	-	1
Nervousness	-	-	-	-	-	-	-	-	-	-	-	.
Mental confusion	-	-	-	-	-	-	1	2	-	-	-	3
Paraesthesia	-	-	-	-	2	-	-	-	-	-	-	2
Transient paresis	-	-	-	-	-	1	-	-	-	-	-	1
Urinary frequency	-	-	-	-	-	-	-	-	-	2	-	2
Epistaxis	-	-	-	-	1	-	-	-	-	-	-	1
Haematuria	-	-	-	-	-	-	-	-	-	-	-	.
Haemoptysis	-	-	1	-	-	-	-	-	-	-	-	1
Blood per rectum	-	-	-	-	-	-	-	-	-	-	-	.

Signs or symptoms as above	2	6	11	17	32	44	44	44	20	13	1	234
No signs or symptoms	-	-	2	2	1	12	8	6	11	1	1	44
No hist. available	1	2	-	3	10	7	12	5	13	3	-	56
Mentally confused	.	.	.	(2)	(2)	(5)	(4)	(7)	(2)	(1)	.	(30)
TOTAL	3	8	13	22	43	63	64	55	44	17	2	334

§ History accordingly unreliable. The signs and symptoms are already included in the above tables.

TABLE 33. FIRST SIGN OR SYMPTOM IN DIASTOLIC HYPERTENSION

B. FEMALES

	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85-89	Total
Hemiplegia	-	-	-	-	-	-	-	4	2	7	2	5	1	-	-	21
Coronary Throm.	-	-	-	-	-	-	1	-	-	1	-	-	-	-	-	2
Headache	-	-	-	1	-	-	1	-	1	6	4	2	-	-	1	16
Dyspnoea	1	-	1	2	3	2	7	12	14	19	16	16	6	5	1	105
Vertigo	-	-	-	-	-	-	1	-	3	-	9	2	1	-	-	16
Blur./vision	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	1
Precord. pain	-	-	-	-	-	-	-	-	-	3	-	3	-	-	-	6
Palpitation	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	1
Epig. discomf	-	-	-	1	-	-	-	-	-	-	-	1	-	-	-	2
Weakness	-	-	-	-	-	2	2	2	-	1	3	3	-	-	-	13
Fatigue	-	-	-	1	1	-	2	1	1	-	3	1	-	-	-	10
Depression	-	-	-	1	-	-	-	-	1	-	-	-	-	-	-	2
Insomnia	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	1
Nervousness	-	-	-	-	-	-	-	2	1	-	1	-	-	-	-	4
Paraesthesia	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	1
Trans. paresis	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Urinary freq.	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	1
Epistaxis	-	-	-	-	-	-	1	1	2	1	1	1	-	-	-	6
Haematuria	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	1
Haemoptysis	-	-	-	-	-	-	-	1	-	-	1	-	1	-	-	3
Haematemesis	-	-	-	-	-	-	-	-	-	-	1	-	-	1	-	2
Blood / rectum	-	-	-	-	-	-	-	-	-	-	-	-	1	-	-	1
Mental confusn.	-	-	-	-	-	-	1	-	-	-	-	-	6	1	-	8

Signs or sympt- -oms, as above	1	-	1	6	4	4	16	24	26	40	43	33	16	7	2	223
No signs or symptoms	-	-	1	-	2	2	4	8	10	10	11	9	5	5	3	70
No history available	-	-	-	-	-	-	-	1	7	4	6	8	5	3	-	(34)
With mental * confusion	-	-	-	-	-	-	1	-	4	5	4	3	6	4	-	
TOTAL	1	-	2	6	6	6	20	33	43	54	60	50	26	15	5	327

* History accordingly unreliable. The signs or symptoms of these patients are already included in the above table.

	<u>Male</u>			<u>Female</u>		
	<u>under 60</u>	<u>over 60</u>	<u>Total</u>	<u>under 60</u>	<u>over 60</u>	<u>Total</u>
Diastolic hypertension	73	205	278	109	184	293
Systolic hypertension	12	87	99	23	51	74
No hypertension	17	26	43	2	13	15

It has long been known that the symptoms of hypertension are peculiar in that they lack the specificity of dyspnoea in heart disease or of thirst in diabetes. Indeed, Riseman and Weiss (1930), enumerating the symptomatology of 1090 "uncomplicated" cases having hypertension (most of them having blood pressures greater than 160 mm Hg systolic and 100 mm Hg diastolic), found the complaints of hypertensive patients to be identical with the complaints of others (whose blood pressure was normal) but who were obese, or psychoneurotic, or of menopausal age.

In addition, it has been shown many times that the symptoms of hypertension may be completely relieved without change in the blood-pressure. Ayman in 1931 reported such relief in 82% of his 40 patients when an impressive therapeutic regime was instituted. Palmer (1937), claiming a fall in systolic blood pressure in 50% of mild cases on a regime of careful and thorough medical treatment, reports also symptomatic relief in 90% of such cases. Robinson (1937) reports a case of hypertension where there was a sudden disappearance of symptoms/

symptoms without change in the blood pressure when the patient was relieved of emotional strain. In this case, even the dyspnoea, thought to be of organic origin, disappeared, and Robinson calls attention to the value of looking for psychic influences in the production of symptoms even where there may seem to be a satisfactory explanation for them in organic disease. Evans and Loughnon(1939), making clinical trials of 33 drugs for the treatment of 70 patients having blood pressure levels over 160/100, found that an improvement in the severity of symptoms occurred as often with a rise as with a fall in blood pressure, and, moreover, that in relieving symptoms inert placebos were often as efficacious as reputed hypotensive drugs.

It is difficult to avoid the conclusion that the symptoms of hypertension are of psychic origin and relieved by the suggestion inherent in any form of treatment. Moschowitz (1929) has gone so far as to describe a definite type of person with certain physical and psychic complexes in which essential hypertension is apt to occur.

Yet there is some similarity between the complaints of individual hypertensive patients, and in the findings of independent investigators. The first list was made by Janeway (1913), and he headed his list with headache as the most frequent symptom. Douthwaite (1928) describing the symptoms/

symptoms found in his 43 cases, found dyspnoea and palpitation most frequently, and headache and dizziness next in order. Paullin, Bowcock and Wood (1927) analysed symptoms according to the system of the body to which they referred. The most common symptoms of Riseman and Weiss (1930) were headache, dizziness, aches and pains, and dyspnoea, and those of Ayman and Pratt (1931) were headache, "pain", nervousness and dizziness. Headache was first in Marshall's list (1932), while Weiss in 1942 divided his patients into two groups, a larger group who complained of headache, dizziness, fatigue, and constipation, and a smaller whose complaints were pain in the heart with palpitation, dyspnoea, and fatigue. In Table 30, I have listed the findings of these investigators and compared them with my own. Only among Douthwaite's cases is there a pattern similar to that in mine, i.e. dyspnoea occurring more frequently than headache. However, different surveys cannot be compared too closely, as the exact definition of each term employed varies from survey to survey, if not indeed from patient to patient.

For comparison with essential hypertension, I have set down in Tables 31 and 32 the frequency and character of the symptoms complained of by the few patients with malignant hypertension and chronic nephritis.

2. Mode of onset of hypertension.

Ayman and Pratt in 1931 declared that the symptoms of hypertension could be divided into (a) early, and (b) late. The early symptoms they noted were:- headache, dizziness, weakness, fatigue, nervousness. In agreement with Sir Clifford Allbutt's statement that when dyspnoea occurred the "fifth act of the play was in progress", they found dyspnoea a very late symptom. True vertigo, which was secondary to cerebral sclerosis, and nocturia, secondary to renal insufficiency, were also among the late symptoms.

I have endeavoured to determine what were the first indications, in my patients, that a state of hypertension might exist, and have set down my results in Table 33.

It was noticed that, in both sexes, in only a small proportion (in 30 out of 278 men, and 23 out of 293 women) did illness suddenly descend without warning. The predominant mode of onset was the commencement of dyspnoea on exertion - in 127 out of 278 men (45.7%), and in 105 out of 293 women (35.8%). The next commonest symptom was dizziness, which occurred in 31 out of 278 men (11.1%), and in 16 out of the 293 women (5.4%), and in men, especially, it became commoner as age increased. Headache was the first symptom complained of by 6 out of the 278 men (2.1%), and by 16 out of the 293 women (5.4%).

A very large number of people, therefore, noticed no abnormality until dyspnoea on exertion appeared, indicating loss of cardiac adaptation.

3. A Discussion of each Symptom in Turn.

1. HEADACHE

a). The incidence of headache.

Headache has been associated with hypertension since 1913, when Janeway first called attention to the fact that it was the most frequent symptom complained of by his patients. Later other workers such as Riseman and Weiss (1930), Ayman and Pratt (1931), and Marshall (1932) also found that in their series of cases headache was the most frequent complaint.

A glance at Table 30, wherein are listed the complaints of various reported groups of hypertensive patients, shows the widely differing figures given for the incidence of headache. The highest incidence of 72% is in the series of Ayman and Pratt, and the lowest figure, 33.3%, is recorded in Kauffman's series (as reported by Riseman and Weiss, 1930), though here migraine was listed as a separate symptom.

Other observations that have been made on headache and hypertension include the following -

a./

- a. Headache occurred as one of the earliest of symptoms (Ayman and Pratt. 1931).
- b. Headache, present in 50 out of 93 cases, was associated with dizziness, fatigue, and constipation (Weiss. 1942).
- c. Migraine was five times commoner in hypertensive patients than in a control group (Gardner, Mountain and Hines. 1940).

I have been unable to find a record of the occurrence of headache in the ordinary population. Weiss and English (1943) record that it has been estimated to be present in 50% of hospital patients. Riseman and Weiss (1930) found that headache occurred often in psychoneurosis, and its incidence was not so different from the incidence in hypertension. Weiss (1942) suggests that symptoms which in normal people are referred to the emotions are in hypertension referred to the high blood pressure, and, indeed, the emotional origin of headache is so well known as to have crept into everyday language, where a source of vexation and anxiety is referred to in terms of 'headache'. In addition, however, it must not be forgotten that headache occurs in association with serious organic disease.

b). The Cause of headache.

Formerly, the most widely held hypothesis as to the genesis of headache ascribed the pain to stretching of the parietal dura from increased intracranial pressure, and it was on the basis of this assumption that lumbar puncture was often resorted to for its/

its relief when very severe. Northfield (1938) has, however, shown that the dura is insensitive except around the arteries, the large venous sinuses, and some areas at the base of the brain. Pickering, writing in 1939, suggested, on the basis of his own and Hess's observations on histamine headache and Northfield's writings on headache of intra-cranial tumour, that it seemed probable that most headaches of intracranial origin were due to tension round the cerebral vessels - the intra-cranial arteries when these dilate, as in histamine headache and the headache of pyrexia, or the great venous sinuses, when these are pulled on and displaced in intracranial tumour.

It is an attractive hypothesis, then, that headache in hypertension is due to dilatation of the cerebral arteries. The intrinsic muscular weakness of the tunica media of the cerebral arteries renders it possible that hypertension, though a disease characterised by constriction rather than by dilatation, should predispose to a state of passive dilatation of the cerebral arteries - or allow, if they were dilated from some other cause, e.g. stress or fatigue - an increased degree of stretching of the arterial walls, and so of the pain-sensitive areas of the dura mater around them.

Wolff, writing in 1944, referred to the demonstration that headache in hypertension can be abolished by ergotamine tartrate, digital pressure on several branches of the external carotid, or ligation of the middle meningeal or temporal arteries. He inferred/

inferred that headache in hypertension was chiefly due to dilatation of the branches of the external carotid artery, and that its mechanism was thus comparable with that of migraine. He indicated, however, that by headache in hypertension, he meant not the hypertensive encephalopathy of Fishberg but the frequent headaches suffered by hypertensive patients who might otherwise be free of symptoms. This offers an explanation in physical, as opposed to psychosomatic, terms for Gardner, Mountain, and Hines' findings as above, though, of course, it may be true, as Weiss and English have pointed out, that rage has something to do with the mechanism of both.

This explanation of its mechanism infers that in hypertension there is a predisposition to headache. Relaxation of arterial walls is one factor in the production of hypertensive headache, and the second is the high level of blood pressure causing greater distension of the relaxed arteries than would occur if it were low.

c). What constitutes "headache".

What exactly is meant by the term "headache" is something difficult of definition. Weiss, while indicating that a particular type and location of headache is common in hypertension, also described a great variety of pains, discomforts, and fullness in the head, with or without vertigo, which were also called "headache" and attributed to hypertension./

hypertension. Page and Corcoran in their review of hypertension, suggested that hypertensive headache generally occurred in the morning, and was often located in the occipital region. Unilateral headache associated with marked general upset has been termed "migraine" by most writers.

d). The Findings in my group of patients.

1. In contrast to the findings quoted above and shown in more detail in Table 30, concerning the incidence of headache in the hypertensive cases of other investigators, headache occurred in remarkably few of the patients in this survey who had diastolic hypertension. In Table 30, the percentage of patients in whom headache occurred ranges from 72 to 33.3. Out of the patients in my survey who had diastolic hypertension (in all 278 men and 293 women) 61 men or 21.95%, and 63 women or 21.50% complained of headache. Furthermore, there was no significant difference between those figures, and the proportion of systolic hypertensives who had headache - 16 out of 99 men (20.34%) and 10 out of 74 women (13.52%). Five men out of the 43 with no elevation of blood pressure had headache (11.62%) and between this figure and the preceding ones, there is no significant difference.

2. When we consider whether or not there is any change in the incidence of headache in different age-groups, we find that in men 21 out of 73 patients (28.77%) younger than 60 suffered/

suffered from headache, and 40 out of 205 (19.51%) over 60. In women, the figures were 28 out of 109 (25.69%) under 60 and 35 out of 184 (19.02%) over 60. Neither in men nor in women was there a statistically significant difference between the two groups.

3. Thirdly, there was no significant difference between the incidences of headache in men and in women. However, headache was the first indication of hypertension in 6 men (2.16%) and 16 women (5.49%), and this difference is statistically significant.

4. From Table 31, it can be seen that 5 women were classified as having malignant hypertension, and that they all complained of headache. In spite of the small numbers involved, there is a significant difference between these figures and the proportion of women with diastolic hypertension who complained of headache.

5. Twelve men had chronic nephritis (Table 32 A), and of these twelve, ten (or, 83.33%) complained of headache, and the comparison with the group of diastolic hypertensives gives a statistically significant difference. Similarly, of the six women with chronic nephritis (Table 32B), five had headache, a figure which contrasts markedly, and significantly, with that in diastolic hypertension.

Thus/

Thus it seemed that among my cases, while essential hypertension of the benign type did not induce an increased incidence of headache, patients with malignant hypertension or chronic nephritis, in vivid contrast, had headache significantly more commonly, and indeed as almost a constant finding.

This may form part of the explanation for the low incidence of headache among my cases as a whole - namely that other surveys may have included a larger number of persons with more rapidly advancing types of hypertension.

Having this low incidence in mind, I attempted to acquire refutation or confirmation of this finding by carefully interrogating the 148 elderly men who formed my third major group of cases (they will be fully described in Part III). After personally taking from each man a careful history, I asked him directly whether he had headaches, and one would expect the incidence of the complaint to be rather more in such circumstances. Among these patients there were altogether 42 men from whom an adequate history could not be obtained. Of the remaining 106 men, 52 had diastolic hypertension, 12 had systolic hypertension, and 42 had no hypertension. In these three groups, those with headache numbered 11 (21.15%), 1 (8.33%), and 3 (7.14%) respectively. There is no significant difference between these three groups, nor is this incidence markedly/

markedly different from that in the cases already quoted. Only a few of these patients gave a history of consistent morning headache or of a particular site for headache - occipital or frontal. All pains in the head seemed to be included in this term.

While these patients are not necessarily representative of the general population, the figures serve, on the whole, to confirm the finding that in my group of cases, whether suffering from hypertension or not, the incidence of headache was very low (about 20%). This figure does not seem to be related in my patients to age or sex. It would be of interest to know how this group compared with a similar group, say, among the professional classes. For apart from the fact that my patients could be distinguished from the rest of the world by being ill (and this would increase the incidence rather than diminish it) they could also, I think, quite fairly be distinguished on the grounds that their responsibilities - family, social, and financial - weighed more lightly upon them than upon the average member of society. Even if the patients in this survey had fewer strains or worries, or had a higher threshold for pain (a point which was discussed in my section on Coronary Thrombosis) than the general populace, or other groups, we cannot avoid the conclusion that in essential hypertension there are a large number/

number of patients who do not make the complaint of headache. On the other hand, headache is an almost constant finding where one expects a rapidly rising, and non-labile high blood-pressure. This would seem to suggest that the vast majority of my cases belonged to a relatively benign type of essential hypertension.

2). DYSPNOEA.

a). The Definition of Dyspnoea.

The word "dyspnoea" in its etymological sense means difficult, bad, painful, or in other wise disordered breathing, and it is in such a sense that most clinicians use the term - including within its meaning not only the patient's sensations, but also difficult breathing observed by objective means. Here however I have not included objective observation in my analysis of records, but only the patients' stories, and so "dyspnoea" as far as the great majority of my cases are concerned, refers only to what the patient himself described.

b). The Incidence and cause of Dyspnoea.

The most striking observation in connection with dyspnoea is that in patients with diastolic hypertension it is the most common symptom by far, occurring in 138 of 278 (49.65%) males and 128 of 293 (43.68%) females. But dyspnoea is a symptom of many disorders, and in this series of cases a not inconsiderable number of patients (90 males and 56 females) had varying degrees/

degrees of cardiac failure, for which they were under treatment, whilst 30 males and 12 females had emphysematous changes. Nor can acidosis (from renal failure or diabetes) be entirely eliminated for although renal failure occurred only in a very small number of cases, diabetes was more common (8 males and 33 females).

Yet even when only the small group of cases with only symptoms and no signs of disease are considered, (Table 29) dyspnoea is still very commonly found - in 28 out of 74 males (37.84%) and in 47 out of 95 females (49.8%). To what can it be ascribed in those patients?

In some, perhaps, it has a psychoneurotic origin, as in Robinson's case, quoted above. In others - diabetes ? 2 males and 13 females were diabetics. Incipient renal failure or cardiac failure? Of those cardiac failure is the most likely. In hypertension, as we have seen, the heart is working at a disadvantage as its load increases and its blood supply diminishes.

Lewis (1937) has said : "The first indication of cardiac failure is to be found in a diminished tolerance of exercise. Of the very numerous tests of cardiac efficiency and inefficiency that have been devised there is none that approaches in delicacy the symptom breathlessness." Dyspnoea is, by far, the commonest first indication of hypertension/

hypertension and if it be true that dyspnoea in hypertension is commonly of cardiac origin, then the breakdown of cardiac adaptation is the first indication of abnormality in very many cases.

Dyspnoea occurred in 138 out of 278 (49.65%) diastolic hypertensives and 44 out of 99 (44.45%) systolic hypertensives (men). Of these, 46 out of 73 (63.02%) and 6 out of 12 (50.00%) respectively were not yet 60 years of age. In this younger age-group the difference between the proportions was significantly different - probably reflecting the seriousness of hypertension for men under 60 years. Similarly, in diastolic hypertensives (men) there is a significant difference between the 46 out of 73 (63.02%) under 60, and the 92 out of 205 (44.87%) over 60 who complained of dyspnoea.

Among the corresponding incidences in women there are no significant differences. The figures are as follows:-

	<u>Below 60</u>	<u>Over 60</u>
Diastolic hypertension	54-109 (49.55%)	74-184 (40.22%)
Systolic hypertension	9- 23 (39.13%)	21- 51 (41.17%)

There are no significant differences between the figures for men and for women.

3. VERTIGO.

a). The Definition of Vertigo.

As in the case of headache, here again we have difficulty in defining precisely what is meant by the term "vertigo". As Simonton in 1941 pointed out, patients call anything dizziness or giddiness which ranges from the slightest sensation of rotation to disorientation in space so extreme as to produce collapse. According to Page and Corcoran, the term "dizziness" is restricted to an abnormal sensation of unsteadiness characterised by a feeling of movement within the head. In the view of Soma Weiss, "dizziness" is often a 'forme fruste' of syncope.

Diagnosis of the symptom of vertigo ideally demands a sensation of rotation - as opposed to syncopal states which do not. I cannot claim for my cases that all who are labelled as having "vertigo" did in fact feel this disordered orientation as opposed to other sensations of instability, and I therefore with diffidence and reservation set down the figures which follow.

b). The Cause of Vertigo.

The maintenance of an appropriate position of the body in space depends upon several groups of afferent impulses - from retinae, labyrinth, and proprioceptive impulses from different groups of muscles. These impulses are mutually related by central mechanisms (cerebellum, vestibular nuclei, red/

red nuclei), which send impulses to the parietal cortex, so influencing voluntary movement. Thus vertigo may result from disturbance of function of cortex, eyes, cerebellum, brain-stem or ear.

Also Brain (1938), in his article on Vertigo, mentions psychogenic influences, namely, the effect of bringing into consciousness elements in spatial orientation which are normally unconscious. In such a case the patient uses a physical relationship as a symbol for a psychological relation between himself and his environment which is a source of difficulty to him. Such a process of symbolism, according to Brain, is well illustrated by the double meaning (physical and moral) of such terms as "lapse", "fall", "downfall". Riseman and Weiss (1931) have indicated the frequency of vertigo in psychoneurosis, and Weiss (1942) states that in his opinion vertigo is often a result of psychic stress - a symbolic representation of insecurity.

Guild, writing in Cowdry's 'Problems of Ageing' (1942), interprets the absence of reports (until that date) on the effect of aging on the vestibular apparatus, to mean that there is no decline in its function with advancing years. But Sheldon (1948) in his Wolverhampton survey, has shown that vertigo is a most important and distressing symptom in old age. He found that it occurred much oftener in women than in men, and that its highest incidence was apportioned in/
in/

in different age-groups in the two sexes. The maximum incidence in men was in the age-group 75-79, and in women the incidence went on increasing as age advanced.

Sheldon concluded that different mechanisms participated to different degrees in the two sexes. In very old women, he found that 68% were deaf, 70% had vertigo, 70% were liable to tumble, and 90% had difficulty in the dark, and he concluded that these were manifestations of decline in the function of the whole inner ear and its central connections.

Since vertigo can result from such a variety of stimuli, it has proved difficult to ascertain its mechanism in the hypertensive patient, and no satisfactory pathological explanation has as yet been found, though Page and Corcoran state that its presence usually indicates that haemorrhages, albeit small ones, have occurred in the brain.

c). The Incidence of Vertigo.

Once again we find that although "dizziness" is referred to in each list of symptoms in Table 30, its incidence varies from the 66% (Ayman and Pratt. 1931) to 23.3% (Douthwaite. 1928). Furthermore, Riseman and Weiss (1930), Ayman and Pratt (1931), and Weiss (1942) have mentioned that dizziness is frequently found in association with headache. How the two symptoms connect up, and what is their common pathology/

pathology seems somewhat obscure.

In my patients, vertigo was complained of by 20.86% (58 out of 278) men and 14.68% (43 out of 293) women. I found that vertigo did not occur more commonly in diastolic hypertension. These figures do not show a significant contrast with the 16.16% (16 out of 99) men and 16.22% (12 out of 74) women with systolic hypertension.

However, out of the group of cases who were personally interrogated, 25.00% (13 out of 52) with hypertension complained of vertigo, while of 42 without hypertension, none had vertigo. This difference is statistically significant. What those patients meant by "dizziness" is again difficult of definition. They were, for the most part, not used to describing sensations, and their evidence must be accepted with reservation - especially as all, when examined, were safely ensconced in bed.

To return to the larger group of cases, in the under-60 age-groups, 24.66% (18 out of 73) men and 11.92% (13 out of 109) women were included in those who complained of vertigo, and in the over-60 groups, 19.63% (40 out of 205) men and 16.30% (30 out of 184) women. There was no significant increase in the latter figures over the former.

Amongst/

Amongst the diastolic hypertensives, the proportions having vertigo in the two sexes were just not significant. Over the age of 60 years, there was no difference between men and women, but under 60, vertigo was complained of more frequently by men than by women. Furthermore, 11.15% (31 of 278) men and 5.46% (16 of 293) women began their hypertensive course with vertigo, and these also are significantly different figures.

Without further and careful investigation, then, it would appear difficult to come to a conclusion (a) about whether vertigo does appear more commonly in hypertension (b) if so, what mechanism is involved, (c) whether vertigo increases in incidence in elderly people, and (d) whether hypertension has anything to do with such an increase, if it were present.

I can only with diffidence put forward my figures, which tend to suggest:-

1. that in old age, vertigo is more common among hypertensives than non-hypertensives.

2. that vertigo is more common in the male sex in hypertension.

4. Blurring of Vision.

In the reviews of the symptomatology of hypertension which I have cited, not much importance is attached to disturbances of/

of vision. Only Riseman and Weiss mention "spots before the eyes", and in a proportion of 4% of their cases.

It is believed that the condition of the retinal vessels is of great importance with regard to prognosis in hypertensive cases. Keith, Wagener, and Barker (1939) based their method of grading the severity of hypertension on the condition of the eye vessels, and Ellis (1938) regards papilloedema as the essential requirement before the diagnosis of malignant hypertension can be made.

I personally found that "blurring of vision" did not occur without changes in the retinal vessels, though I am not prepared to say that changes in these vessels were not specially looked for when the complaint of blurring of vision was made.

I did not find disturbances of vision to be very frequent (in 22 out of 278 men, or 7.91% : in 25 of 293 women, or 8.53%), and there was no significant difference between its incidence in diastolic and systolic hypertension, in the over- and under-60 age-groups, or between men and women. The relevant figures are:-

	<u>Men</u>	<u>Women</u>
Under 60 yrs.	8 out of 73 (10.96%)	15 out of 109 (13.73%)
Over 60 "	14 " " 205 (6.83%)	10 " " 184 (5.44%)

In only three patients (two men and one woman) was it the first symptom of disease.

Blurring/

Blurring of vision, then, is not a frequent symptom in hypertension, and it is not a common presenting symptom. But when it occurs, although disturbances of vision can have a psychogenic origin, it usually indicates some change in the retinal vessels. This is important, as the degree of change in the vessels can give a great deal of information about the severity of the disease process, and indeed is so used by several authors.

5). PRAECORDIAL PAIN.

Bishop and Bishop (1931) have estimated that about 25% of the patients attending a cardiologist have cardiac pain as the main complaint. Pain in the praecordium may, of course, be the result of several factors. If sudden and severe it may be the result of coronary thrombosis, pulmonary embolism, rupture of the aorta, dissecting aneurysm, spontaneous pneumothorax, pericarditis, or angina pectoris which has generally a history of clearly outlined attacks with a precipitating factor. Then there are the other vague syndromes and pains which might be attributed to fibrositis, intercostal neuralgia, digestive upsets, or gall-bladder disease.

Finally there is the problem of the obese woman of menopausal age, who suffers from severe praecordial pain, dyspnoea, and fatigue - a syndrome very like the syndrome of "pain in the heart with palpitation, dyspnoea, and fatigue" which/

which Weiss (1942) claims to have been the opening statement, as it were, of what was, in 38 of his 98 patients, to unfold itself in the future as hypertension. Douthwaite found angina in 4.6% of his cases, and Riseman and Weiss in 5.3% of theirs.

Again, there is difficulty in interpreting exactly what is meant by these authors when they refer to anginal pain. Among my cases, pain over the praecordium was found rather more often - in 7.55% of men (21 out of 278) and 6.14% of women (18 out of 293). There was no significant difference between the proportions involved of diastolic and systolic hypertensives, of under 60 and over 60 age groups, or of men and women. In these groups, patients with praecordial pain were distributed as follows:-

	<u>Men</u>	<u>Women</u>
Systolic Hypertension	4 - 99 (4.04%)	3 - 74 (4.05%)
D.H. - under 60 yrs.	8 - 73 (10.96%)	4 - 109 (3.67%)
D.H. - over 60 yrs.	13 - 205 (6.34%)	14 - 184 (7.61%)

6). PALPITATION.

By palpitation is meant the consciousness of the cardiac impact against the chest wall regardless of whether there is an increase in its rate or not, or of whether or not the heart's action is regular.

According/

According to Weiss (1942), palpitation is commonly associated with praecordial pain in his second group of hypertensive symptoms. It does occur in association with cardiac irregularities, and it is also a very frequent symptom in cardiac neurosis. In a susceptible patient a very slight irregularity in the heart's action may be sufficient to call the patient's attention to his heart and from that point in time he may suffer from palpitation.

Once again my figures show an incidence strikingly less than in the surveys I have quoted. Palpitation was found in 3.24% (9 out of 278) men and 5.80% (17 out of 293) women - which is in contrast with the incidence of 51.2% (Douthwaite), 13.1% (Riseman and Weiss), and 18% (Ayman and Pratt).

When this incidence was compared with the incidence of palpitation in systolic hypertension, no significant difference was found either among the men or among the women. When the effect of age and sex upon the incidence was examined, it was seen that palpitation occurred relatively more often in the group of women under 60 than in men under 60 or women over 60 - the differences being significant. The incidence of palpitation in these various groups were:-

	<u>Men</u>	<u>Women</u>
Systolic Hypertension	7 - 99 (7.07%)	6 - 74 (8.11%)
D.H. - under 60 yrs.	3 - 73 (4.11%)	11 -109 (10.09%)
D.H. - over 60 yrs.	2 -205 (2.93%)	6 -184 (3.26%)

Palpitation is not an early sign of the disease, being the initial complaint in only one woman.

7). EPIGASTRIC DISCOMFORT.

This occurred so seldom that it need not be considered in detail. It was found in 8 men and 4 women with diastolic hypertension. In three of these cases it probably originated in enlargement of the liver due to cardiac failure - in the others, perhaps in some digestive disturbance.

8). WEAKNESS.

Weakness is another term whose definition is difficult. For the majority of patients, it is probably synonymous with fatigue. There was no difference between its incidence in diastolic or systolic hypertension, nor was it significantly affected by age or sex.

The figures are as follows :-

	<u>Men</u>	<u>Women</u>
S.H. - under 60	2 - 12 (16.67%)	5 - 23 (21.74%)
S.H. - over 60	7 - 87 (8.05%)	10 - 51 (19.61%)
Total S.H.	<u>9 - 99 (9.09%)</u>	<u>15 - 74 (20.27%)</u>
D.H. - under 60	5 - 73 (6.85%)	11 - 109 (10.09%)
D.H. - over 60	18 - 205 (8.78%)	19 - 184 (10.33%)
Total D.H.	<u>23 - 278 (8.27%)</u>	<u>30 - 293 (10.23%)</u>

9). FATIGUE.

This is a vague term, not easy to define from the medical point of view, and seldom even discussed as a medical problem, except by those interested in industrial health and psychology - or those who have studied athletic or military problems. It has been defined as a decrease in potential or actual capacity for work. Studies of metabolic function in industrial workers have shown that the normal human being possesses a comfortable margin of metabolic reserves and develops normal fatigue only after prolonged activity which is well beyond the amount indulged in by the ordinary person.

It has been established that the amounts of constituents in blood are maintained continually within a relatively narrow range, and that variation above or below these narrow limits frequently results in fatigue. Apart from the physical causes, however, fatigue may be an expression of mental and emotional conflicts.

In my cases, fatigue occurred among 5.40% (15 out of 278) of the men, and 11.61% (34 out of 293) of the women. There is no significant difference between the proportions in diastolic or systolic hypertension, nor was the incidence affected by age or sex.

	<u>Men</u>	<u>Women</u>
Systolic Hypertension	4 - 99 (4.04%)	6 - 74 (8.11%)
D.H. - under 60 yrs.	4 - 73 (5.48%)	11 - 109 (10.09%)
D.H. - over 60 yrs.	16 - 205 (7.80%)	18 - 184 (9.78%)

10). DEPRESSION.

Depression is a mood of dejection or gloom which may arise in a normal person as the result of an adequate stimulus, or be a symptom of emotional or nervous disorder.

15 patients among the diastolic hypertensives complained of depression, and four of these (two men and two women) were over 60 years of age. It did not occur among the systolic hypertensives, but these figures are too small for effective comparisons to be made.

11). INSOMNIA.

Again, the incidence of insomnia was very low. An incidence of 2.54% in men (7 out of 278) and 3.41% in women (10 out of 293) contrasts oddly with that of the 55% of Ayman and Pratt's cases - though in the survey by Riseman and Weiss, it occurred in 5.6%.

This incidence did not appear to be affected by age or sex, and it was not significantly different from the incidence of insomnia in systolic hypertension.

	<u>Men</u>	<u>Women</u>
Systolic Hypertension	0 - 99 (0 %)	2 - 74 (2.7%)
D.H. - under 60 yrs.	4 - 73 (5.48%)	3 - 109 (2.19%)
D.H. - over 60 yrs.	3 - 205 (1.46%)	7 - 184 (3.81%)

The figures are too low to enable much to be said about the importance of insomnia in hypertension.

12). NERVOUSNESS.

"Nervousness" again eludes precise definition. It implies a state of mental or physical restlessness in which the capacity for purposeful activity has become impaired, and the patient is uneasy, apprehensive, and anxious.

The relationship of this symptom to hypertension is interesting because it has so often been stated, as Page and Corcoran do in their book, that hypertensives seem to be highly strung, emotional people. Alexander and Saul likewise say that strong anxiety is part of the temperament of the hypertensive person.

Whether for this or for other reasons, many authors have associated Hypertension with Hyperthyroidism. Keith and his associates (1928) showed that, in out of a total of 39 cases of malignant hypertension, eight had a basal metabolic rate greater than 20%, and Boothby and Sandiford (1920) that of 170 patients with a high blood pressure, approximately 90% had a B.M.R. between +15 and -15%. On the other hand, Weiss and Ellis (1930) had one case, and Becker (1932) 16, in which thyroidectomy or X-ray treatment of the thyroid gland produced reduction in the high basal metabolic rate without influence on the blood pressure. Crile and McCullagh (1930) considered that the increased B.M.R./

B.M.R. in hypertension was the result of the increased cardio-vascular effort required to maintain the circulation against a high diastolic pressure. More recently, Mountain, Allen, and Haines (1943) have analysed 827 cases of essential hypertension from the point of view^{of} B.M.R. and thyroid status. As the severity of the hypertension increased, the proportion of patients with a high B.M.R. increased. Also, where there was hyperthyroidism as well as hypertension, thyroidectomy did not necessarily produce a fall in B.M.R.. In addition Treusch, Kepler, Power, and Haines (1944) have shown that the hypertensive patient has a much lower urinary creatine level than the majority of hyperthyroid patients. In general, then, it would appear that essential hypertension is not associated with hyperthyroidism, but that in some cases of severe hypertension the basal metabolic rate may be raised. No adequate explanation for this is forthcoming up to the present.

So far as I know, no writer has made mention of any resemblance in the clinical pictures of Hyperthyroidism and Hypertension. Nervousness and anxiety are predominant symptoms of hyperthyroidism, and by the authors previously mentioned and by Keith, Wagener, and Barker (1939) they have been considered important symptoms of hypertension.

In the papers of the various authors quoted, the incidence of nervousness varies enormously, from Ayman and Pratt's 67% to Riseman and Weiss's 13.2%. Douthwaite classes nervousness among/

among his miscellaneous group. Among my patients the incidence was very small indeed - in 3 out of 278 (1.08%) of men and 11 out of 293 (3.75%) of women. There was no significant difference between those figures and those for the incidence in systolic hypertension. There was no significant difference between the sexes, but nervousness occurred in young women significantly more often than in old women. The comparative incidence of nervousness was:-

	<u>Men</u>	<u>Women</u>
Systolic Hypertension	2 - 99 (2.02%)	6 - 74 (8.11%)
D.H. - under 60 yrs.	2 - 73 (2.74%)	8 - 109 (7.34%)
D.H. - over 60 yrs.	1 - 205 (0.48%)	3 - 184 (1.09%)

13). PARAESTHESIAE & 14). TRANSIENT PARESES.

The incidence of these two conditions is so low that they need only be mentioned and passed over. Paraesthesia occurred in 7 men (2.52%) and 7 women (2.40%), and pareses in 6 men (2.16%) and 2 women (0.68%) - equally distributed between the under-60 and the over-60 age-groups. Pareses which clear up rapidly are thought to be due to arteriospasm and to presage the onset of hemiplegia. This was not necessarily true of my cases.

15). URINARY FREQUENCY.

I have much hesitation in drawing any conclusions from the incidence of this urinary abnormality. Not only are there/

there differences in the patients' personal judgments to be overcome (as with other symptoms) but frequency of micturition may occur with almost every kidney, bladder and prostatic disturbance. Even nocturia need not necessarily be an index of renal impairment.

Urinary frequency, however, was complained of by 53 men (19.06%) and 39 women (13.30%) - figures not significantly different from the 14 men (14.15%) and 12 women (16.22%) with systolic hypertension who had this complaint. Neither age nor sex had any significant effect on the incidence of this complaint.

	<u>Men</u>	<u>Women</u>
D.H. - under 60 yrs.	18 - 73 (24.66%)	15 - 109 (13.73%)
D.H. - over 60 yrs.	35 - 205 (17.07%)	24 - 184 (13.04%)

16). HAEMORRHAGES.

Obvious haemorrhage must partake of the nature of a "sign" of disease, rather than a symptom. The occurrence of bleeding, however, was, in the vast majority of instances, related by the patient rather than observed by the doctor, and I have therefore included it as a "symptom".

As in the case of other manifestations of hypertensive disease, there is much disagreement about incidence and importance of haemorrhages. Fishberg in his review of essential hypertension, says that haemorrhage is "a striking feature",/

feature", whereas Page and Corcoran do not mention it. Kauffman and Ayman and Pratt (1931) make no mention of haemorrhage. Douthwaite gives 13.9% as the incidence of haemorrhage from all sources, and Riseman and Weiss (1930) found epistaxis in 5.6% of their cases.

In my cases, haemorrhage from all sources occurred in 11 men (3.96%) and 21 women (7.17%) - and of those, 5 men (1.80%) and 12 women (4.10%) had epistaxis.

Between these figures and those for systolic hypertension (1 man out of 99, and 4 women out of 74) there is no significant difference.

With regard to age and sex, haemorrhage occurred oftener in young men than old, and also in old women oftener than old men. The comparative incidences were:-

		<u>Men</u>		<u>Women</u>
D.H. - under 60 yrs.	6 - 73	(8.23%)	9 - 109	(8.26%)
D.H. - over 60 yrs.	5 - 205	(2.41%)	12 - 184	(6.52%)

4.

CONCLUSIONS.

Dyspnoea was the only symptom consistently observed in hypertension. In many cases it was related to obvious cardiac failure, and in others it may have been due to psychic stress or strain, but in the majority of cases, it probably indicated the development of myocardial strain and/

and fibrosis with the onset of cardiac insufficiency. In a great many cases, dyspnoea was the first indication of illness, and we may therefore conclude that diastolic hypertension often remains unnoticed until myocardial insufficiency begins.

Headache was the next most common symptom, observed consistently only in rapidly progressing hypertension.

The incidences of the other symptoms were, for the most part lower than those given in other surveys. None of the symptoms listed was specific for hypertension; in none was there a significant difference between the proportions in diastolic and systolic hypertension.

Differences between age-groups were not important, though in men of the under-60 age-group, haemorrhages were more often observed than in the older group, and nervousness and depression were commoner in women in the under-60 group. Neither were there important differences between the sexes, though as a presenting symptom, vertigo was more often complained of by men, and headache and haemorrhage by women. There is thus no difference in the character of the symptomatology of hypertension in young or old, male or female.

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P A R T III.

P A R T III.

The Cardio-vascular System in Old Age.

Chapter I. A Plan of the Study.

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in the Study.

- 1) Method of Selection
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- 5) Reasons for Admission to Hospital
- 6) Duration of Stay in Hospital
- 7) Diseases suffered from.

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- 2) Hypertension and Obesity
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 - 4) Serum Proteins
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CHAPTER I.

PLAN OF THE STUDY.

Plan of the Study.

The aim of the investigation was to determine whether the presence of high blood-pressure made any difference to the old man or old woman, and, to this end, an endeavour was made to note why he was brought into hospital, what disease or diseases he suffered from, what symptoms he complained of, whether or not he had arteriosclerosis, how alert he was in mind, what fundal changes he presented, and especially, whether or not he suffered from cardiac damage.

A careful history, as detailed as the patient's mental condition allowed, was first obtained. Attention was paid to the patient's civil state, to the details of his domestic life, and to the reason (medical or sociological) for his admission to hospital. At first, inquiry was made into the health of parents, brothers, and sisters, but this was soon abandoned since a large number of patients had lost touch with all relatives except their married partners and children (even, at times, with them). A history of previous illnesses was elicited, but, replies were often rather vague. The symptoms complained of by the patient were ascertained, and, in the first few cases, direct questions were avoided. However, /

However, as the enquiry proceeded simultaneously with the examination of the records of other patients with hypertension, and it became clear that headache and other cerebral symptoms occurred relatively rarely, patients were asked directly whether they complained of headache or dizziness.

While this examination was going on, I attempted to make an assessment of the patient's mental state, and accordingly divided patients into three categories:-

- 1) Mentally confused: those who did not know where they were and could not answer simple questions.
- 2) Dull: those who could interpret simple commands and look after their own bodily processes, without necessarily being able to construct a reasonable story.
- 3) Alert: those who could tell their own story, and enjoy conversation with others.

A careful clinical examination was then performed, with the object of reaching a diagnosis, which was later confirmed by discussion with the medical officer in charge of the ward. Particular attention was paid to the cardio-vascular system, and a clinical estimation of the size of the heart was made. The position of the apex beat was noted as accurately as possible, and its distance from the mid-sternal/

mid-sternal line was measured. This was particularly difficult when emphysema was present, and, as I shall show, my measurements were not always very accurate. Attention was also paid to the ventricular rate, and to the character of the heart sounds. Towards the end of the investigation, fluoroscopic examination of the heart was undertaken in 61 cases, to measure the size of the heart, and to determine whether cardiac enlargement was present.

The advisability of an assessment of the degree of arterio-sclerosis by means of palpation will be discussed later. Since my work was begun before Howell's article (1945), defining various arterio-sclerotic groups, was published, I have not been able to use a strictly comparable classification. But probably that would in any case be a matter of difficulty with regard to decisions which must in the last resort rely on personal judgment. My decision was made after examination of the radial, brachial, temporal, femoral, and dorsalis pedis arteries. The amount of tortuosity of the brachial or temporal arteries was regarded as not so important as the degree of thickness of all the arteries on palpation. Each patient was placed in one of four categories:-

Nil/

Nil - no palpable thickening of the vessel walls.

+ - vessel walls just palpable.

++ - vessel walls firm and easily palpable.

+++ - gross thickening and tortuosity.

In 66 cases, fluoroscopic examination of the right arm and thigh was undertaken, to reveal the presence or absence of arterial calcification.

The blood pressure was examined by the method laid down by the Cardiac Society of Great Britain and Ireland, in 1939. Three estimations were made, at intervals of a few minutes, and, of these, the lowest readings were chosen. If there was a marked difference between the first and the third readings, the estimation was repeated on the following and subsequent days. Estimation of the blood-pressure at the end of a clinical examination can be criticised on the grounds that a basal level is then probably not obtained. But it was thought that since the patient was, by this time, used to a strange doctor, and that fears regarding the nature and the extent of the examination were allayed, he would be less anxious than if the estimation of blood pressure had been made at the beginning of the interview.

Finally, an examination of the fundus was undertaken. A note was made of any retinal abnormalities, and particular attention was paid to the state of the retinal vessels and to/

to whether there were arterio-sclerotic changes present.

Later, a carefully standardised electrocardiographic record was taken.

Soon after admission, a sample of blood was collected from each patient. From this, the haemoglobin level was estimated by a photoelectric absorptiometer method, as described by Bell, Chambers, and Waddell (1945). I have kept a record of these results in my patients.

In the first 63 patients, a sample of venous blood was withdrawn, and collected so that, (a) 5 ml. of oxalated blood were available for the estimation of blood urea, by the direct Nesslerisation Method, as described by King (1946); and of blood cholesterol, by the Sackett Method (described in the same volume), and (b) 5-6 ml. of serum were available for estimation of serum proteins, by the Biuret Method (Harrison : 1947); of serum calcium, by the Clark Collip modification of the Kramer-Tisdall Method (Hawk and Bergeim : 1938); and of serum inorganic phosphate, by the Youngburg Method (Hawk and Bergeim : 1938).

In/

In a further 46 patients, estimation of the blood urea only was carried out.

These biochemical estimations, made after the other examinations had been completed, took place, in almost all cases, after the patient had been in hospital on a regular regime and ward diet for 7-14 days or sometimes longer.

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C H A P T E R II.

A Description of the Patients in this Study.

- 1) Method of Selection
- 2) Sex Distribution
- 3) Civil State
- 4) Domestic State
- 5) Reasons for Admission to Hospital
- 6) Duration of Stay in Hospital
- 7) Diseases suffered from.

The observations in the following chapters are based primarily on 175 patients admitted to medical wards in Stobhill Hospital, and 18 patients admitted to similar wards in the Eastern District (Duke Street) Hospital. It was intended that all should be over 60 years, but 6 cases between 50 and 60 years have also been included.

1) Method of Selection.

Patients were chosen in a completely arbitrary fashion from among those present in the wards, during the year September 1945 - September 1946. They were examined in the latter part of the morning, or in the early afternoon. First, one ward of 40 patients was selected, and, when all the patients over the age of 60 had been interviewed, I passed to the second ward. After the round of five wards had been made in this manner, I returned again to the first ward - perhaps as long as a fortnight later. Such a method of selection tended to exclude patients who were in hospital for only a very short time. The only variation in the method of selection related to my own ward - a female ward - where almost all the patients admitted, whether for a long or short time, are included in the survey. Patients in the Eastern District Hospital were chosen in a similar way.

I did, however, deliberately exclude from my survey several patients of the required age who were very ill, and who/

who could not be subjected to additional examination, or so confused as to be difficult to handle.

2) Sex Distribution.

I have included only 45 women as compared with 148 men. This was partly because more male than female wards were available for my purpose, and partly because the turn-over among elderly male patients was much greater than among elderly females. Male patients, as it happened, could be more easily disposed of to other institutions, while women perhaps tended to live longer, and so occupy available beds for a longer time.

3) Civil State.

It became clear, very early in the survey, how deeply sociological and psychological factors enhanced or retarded recovery among this group of patients. Both these factors are difficult to evaluate in practical terms, and can be only hinted at as tendencies. For instance, among both men and women, almost twice as many patients in this group were single, widowed, or separated, as were married. (Table 34). This might seem to indicate that there are more old people in the community single and widowed than married. The district medical officers for the City of Glasgow, in their/

their survey of old people (from whom my hospital population was drawn), found the figures for married, single, and widowed, to be as follows:-

Conjugal Status (Curran et al.)

State	Males	Females
Married	143	153
Single	35	68
Widowed	95	507

In this table of Curran et al. (1946), single and widowed men equalled the number of married men, though the widows among the women greatly outnumbered the other groups. If these figures are a gauge of the distribution among (a) the outside (Curran et al.), and (b) the hospital population (my figures), then the single and widowed men either more commonly become ill, or, when ill, more commonly require hospitalisation than the married - indicating perhaps, that the married, who have spouses or children able and willing to look after them in illness, do not so commonly require treatment in hospital.

4) Domestic State.

This tendency can also be shown in a slightly different way, by examining the mode of life of the patients concerned (Table 35). When this table is compared with the following table by Curran et al. (1946), it will be observed that a much higher proportion of my patients lived alone, or in a model lodging-house or other lodgings.

Domestic Life (Curran et al.)

	Married	Single	Widow(er)	Total
Tenant	275	57	437	769
Lodger	21	33	138	192
Inmate of Lodging-house	-	13	27	40
Alone	10	26	243	279

Either, then, those who do not live in their own family circle are (a) more prone to illness, or (b) more likely to require treatment in hospital when illness assails them.

5) /

5) Reason for Admission to Hospital.

Moreover, a large proportion of those who do live with their own families require hospitalisation because they cannot be adequately nursed outside, rather than for investigation and treatment of disease.

The reasons for admission are tabulated in Table 36.

TABLE 36. REASON FOR ADMISSION.

Reason	Males	Females
Medical condn. only	77	30
Med. cond. + home cond.	55	8
Home condn. only	13	2
No def. information	3	2
Total no. patients	148	42

A little more than half of the patients came in mainly for medical reasons, while the others were admitted for an admixture of reasons, but all having some relation to sociological factors.

A comparison was now made of the reasons why the patients in the several domiciliary groups had found their way into hospital, and the results are contained in Table 37. It/

It was discovered that among those who lived at home with their spouse, in a significantly smaller number was the home condition a vital factor in necessitating admission (except for group 5, in which the total number of patients is small).

TABLE 37. DOMICILIARY STATUS AND REASON FOR
ADMISSION.

	MED. Cond.	Med. & Home Cond.	Home Cond.	No inform.	Total
Living at home - alone	14	9	5	-	28
Living at home & wife	35	14	-	-	49
Living at home & friends	14	14	9	-	37
In model	17	16	4	-	37
In other lodgings	8	6	-	-	14
No information	19	4	-	5	28
Totals	107	63	18	5	193

6) Duration of Stay in Hospital.

This being the case, and since so many patients looked not for cure, but for "an hiding-place from the wind and a covert from the tempest", it follows that the length of their/

their stay in hospital was very considerable. I have already explained why it is that this survey of 193 patients rather tends to omit patients who were in hospital for a period of less than two or three weeks. There are among these 193 patients more very long-term hospital inmates, than there were among the patients dealt with in Part II. The duration of stay in hospital is shown in Table 38, and it is seen that while only 19 had been dismissed at the end of one month, the majority of patients were out of hospital by the end of three months. 18 patients were in hospital for 18 months - 2 years, and 7 for over 2 years.

7) Diseases suffered from.

A small group of cases such as this gives no indication of the type of disease suffered by the aged. Suffice it to say that among this group were cases of acute pneumonia and acute cystitis, as well as chronic diseases like rheumatoid arthritis, lingering illnesses like pulmonary neoplasm, and long-standing but severe fever such as in pulmonary tuberculosis of the aged.

TABLE 38./

TABLE 38. DURATION OF STAY IN HOSPITAL.

TIME	No. Patients
Less than 1 week	- } 19
1 - 2 weeks	3 })
2 - 3 weeks	9 } :
3 - 4 weeks	7 })
4 wks. - 2 months	55
2 mths - 3 mths.	28
3 mths - 6 mths.	39
6 mths - 9 mths.	19
9 mths - 12 mths.	8
12 mths - 15 mths.	9
15 mths - 18 mths.	2
18 mths - 21 mths.	2
21 mths - 2 yrs.	5
Over 2 years	7

TABLE 34.CIVIL STATE

	Males	Females	Totals
Single	39	7	46
Married	45	14	59
Widowed	40	17	57
Separated or divorced	5	-	5
Not stated	19	7	26
Totals	148	45	193

TABLE 35.DOMESTIC STATE

	Males	Females
At home alone	26	8
Living with spouse	29	12
Living with friends or relatives	25	15
In motel	38	-
In other lodgings	11	2
No information	19	8
Totals	148	45

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CHAPTER III.

ARTERIOSCLEROSIS.

1. Introduction. A brief account of the nature of arteriosclerosis, and the definition of the term as employed in subsequent chapters.
2. Arteriosclerosis and Age
3. Arteriosclerosis and Sex
4. Arteriosclerosis and Calcification of Vessels
5. Conclusions.

1). INTRODUCTION.

No considerations of the alterations in the course of a man's life which may result from changes of the pressure within the blood vessels, would be complete without some account being taken of the changes in the walls of the vessels. In spite of much histological investigation into the origin of the tissue changes and chemical alterations occurring in the vessel walls as man passes through his "seven ages" of development, much yet remains to be clarified. It is not yet clearly defined what constitutes arteriosclerosis, and whether there are not in fact several processes at work coincidentally, and not necessarily related to one another.

Aschoff (1933) believed that arteriosclerosis was not a phenomenon associated with aging, but a disease of the vessels manifesting itself during senescence. Aging, he said, was associated with dehydration of tissues all over the body, and this involved in the vessels, loss of elasticity, progressive hardness, and increasing brittleness. In the tunica intima, it produced, inevitably, splitting of the internal elastic lamina, and increase in connective tissue. Similar changes in the tunica media produced stretching of the vessel walls with dilatation of the lumina and progressive tortuosity of the vessels. Atheromatosis, the deposition/

deposition in the tunica intima of lipoids, collagenous material, hyalin, and calcium, is now believed (Hueper, 1947 : Wolffe, 1947.) to be a systemic condition of abnormal metabolism, manifesting itself in the arteries, (and, as such, is differentiated from the hypertrophy and fibrosis of the tunica media, which leads to more hardening and stiffening of the vessel walls), is called "true arteriosclerosis" by some (Muir, 1941), and is believed (Moschowitz, 1942) to be directly related to the presence of intravascular tension over a long time.

From the clinical point of view, as Howell (1945) has pointed out, very little definition of arteriosclerosis has been attempted. Indeed, to attempt it is fraught with difficulty, for no estimation of the effects of arteriosclerosis is possible unless an easy clinical method of determining its presence is available. Palpation of the vessel walls is the obvious method, but there are several disadvantages. In the first place, only the muscular arteries of the limbs and the temporal arteries are accessible to palpation, and it has been shown that arteriosclerotic changes proceed in different parts of the body at different rates (Sappington and Cook, 1936.: Ophuls. 1933). Furthermore, Sydenstricker's figures (from Cowdry. 1933), correlating the degree of arterial thickening with the percentage/

percentage amount over or under the average weight, suggest that underweight people have arteriosclerosis more commonly than the overweight. Yet it has been shown (Dublin 1931) that there is a definite association between overweight and increased mortality from arterial disease. There are two possible explanations for this apparent contradiction. Firstly, Dublin has not clearly defined what he means by "arterial disease", and his paper may rather concern the relation between obesity and hypertension, but, on the other hand, Sydenstricker's findings might be criticised on the grounds that to compare palpation of the arteries in thin and fat persons may not produce strictly comparable results. Thirdly, Moschowitz (1942) observed that, in transient hypertension, there is also a transient sensation of thickening in the radial arteries. Fourthly, Fischer and Schlayer (quoted by Moschowitz) have shown that the feel of the vessel wall depends almost entirely on the condition of the media, and is not affected by that of the adventitia or intima. If this is so, only when there is fibrous change, muscular hypertrophy, or calcification of the media will there be detectable alteration on palpation. Lastly, judgment as to the presence and degree of arteriosclerosis is subjective, and therefore will vary from one observer/

observer to another. However, it was felt that if these things were borne in mind when the results of the investigation were examined, and, if the examination were conducted by one observer using the same careful technique in each case, the relative simplicity of the palpation method was sufficient justification for its use.

In the following pages, then, the term "arteriosclerosis" refers not to a particular pathological or histological concept, but to the thickening of the large limb arteries which was perceptible on palpation.

2). Arteriosclerosis and Age.

To state that arteriosclerosis is a very common condition which increases in frequency and severity with advancing age is commonplace. So much so, that, in fact, as I have indicated, it is not yet decided whether or not arteriosclerotic changes are part of an involuntary process beginning at birth. Arteriosclerosis has been demonstrated early in life. Sydenstricker's figures indicate its incidence up to the age of 60, but his final group includes patients of 60 or over, although it is after 60, as Howell has pointed out, that arteriosclerosis becomes of paramount importance. Howell (1945), examining the arteries of 341 healthy Chelsea pensioners, claimed that there was a steady drift towards the more/

more severe degrees of arteriosclerosis as age increased,

Table 39 shows the distribution of my cases, in 5-year age-groups, in respect of the degree of arteriosclerosis present. As age increased, both men and women showed a statistically significant increase in the severer degrees of arteriosclerosis.

3). Arteriosclerosis and Sex.

Table 40 compares the distribution of arteriosclerosis in men and in women. 22 men out of 144 (15.3%), and 17 women out of 45 (37.8%) had no arteriosclerosis. This is a statistically significant difference. Secondly, male patients more commonly showed the severer degrees of arteriosclerosis, and again the figures are such that the difference is unlikely to have arisen by chance. Furthermore, at each age-group, the severer degrees of arteriosclerosis were significantly more common among men than among women (vide Table 39).

This finding is supported by Sydenstricker's figures, which also suggest that the rate of occurrence of arteriosclerosis is greater in men than in women. It cannot be accepted entirely without question, however, for the differing amount of subcutaneous fat in the two sexes may lead to differing interpretations of the sensation in the vessels.

TABLE 39.

A. MALES: DEGREE OF ARTERIOSCLEROSIS AND AGE.

Degree A/s	<60	60-64	65-69	70-74	75-79	80-84	>84	Total
Nil	-	3	6	6	6	1	-	22
+	1	13	6	11	4	-	-	35
++	1	12	14	13	10	5	3	58
+++	-	3	4	7	7	3	5	29
No inform.	-	-	1	2	-	-	1	4
Total	2	31	31	39	27	9	9	148

B. FEMALES:

Degree A/s	<60	60-64	65-69	70-74	75-79	80-84	>84	Total
Nil	2	3	7	3	-	2	-	17
+	2	5	6	-	1	3	-	17
++	1	1	-	2	2	-	-	6
+++	-	1	-	2	1	1	-	5
Total (females)	5	10	13	7	4	6	-	45
Total (males)	2	31	31	39	27	9	9	148
ALL CASES	7	41	44	46	31	15	9	193

TABLE 40.

DEGREE OF ARTERIOSCLEROSIS AND SEX.

Degree A/s	Males	Females	Total
Nil	22	17	39
+	35	17	52
++	58	6	64
+++	29	5	34
Totals	144	45	189

If the changes in the feel of the vessel wall are afforded, as Fischer and Schlayer indicated, by the condition of the media only, and if medial hypertrophy and fibrosis, consequent on hypertension, are more common than atheromatous inroads into medial tissue, it is difficult to reconcile the fact that men over 60, more commonly than women over 60, have palpable arterial vessel walls, while equal numbers of men and women over 60 years of age have hypertension. (In this case, 66.67% of the women and 52.70% of the men had diastolic hypertension - a difference which is not statistically significant).

On the other hand, the statement is common in textbooks that atheroma is "much more common in men than in women." This is supported by the observations of Lake, Pratt, and Wright (1942) who, examining groups of men and women in the same occupations, found that men had a much higher incidence of "arteriosclerosis" than had women. Further, Ludden, Bruger, and Wright (1942), estimating the effects of testosterone propionate and oestradiol dipropionate on experimental arteriosclerosis in rabbits, decided that there was a definite difference between the two sexes in the reaction of the arterial wall to atheromatous change.

4). Arteriosclerosis and Calcification of Vessels (as seen on X-ray.)

51 male patients and 15 female patients (Table 41) had the right arm and right thigh examined radiologically to determine whether or not calcification of the vessel walls was present. The relation of the deposition of calcium to arteriosclerotic change in the vessel wall is not clear. It is deposited in atheromatous plaques in accordance with the general principle that any area of dead or devitalised tissue will become infiltrated with lime salts if it cannot be absorbed. Such deposits are part of the atheromatous change, as lipoid deposits also are part. The so-called Monckeberg's Sclerosis, described first in 1903, is a primary necrosis and calcification of the media not necessarily associated with intimal change. This type of calcification is observed in the arteries of the extremities, which it converts into solid tubes with the calcareous material often deposited in rings. Monckeberg (1903) believed that this type of sclerosis was quite different from ordinary "arteriosclerosis", but this is not an opinion universally held. Indeed, it could, according to the views of some writers, be regarded as an important manifestation of "true" arteriosclerosis.

Calcification/

TABLE 41.

ARTERIOSCLEROSIS AND CALCIFICATION OF VESSELSA. MALES:

Degree A/s	Arm only	Thigh only	Arm & Thigh	None	Total	% A/s with calc.
Nil	-	1	-	5	6	14.44%
+	-	3	1	6	10	40.00
++	-	11	3	11	25	56.00
+++	1	5	2	2	10	80.00
Totals	1	20	6	24	51	52.94

B. FEMALES:

Degree A/s	Arm only	Thigh only	Arm & Thigh	None	Total
Nil	-	2	-	2	4
+	-	2	-	4	6
++	-	-	-	3	3
+++	-	1	-	1	2
Totals	-	5	-	10	15

Calcification was seen on X-ray in 27 male patients and 5 female. Thus, calcification occurred more frequently in males, but the numbers observed were not sufficient to indicate a significant difference. Among the male patients, the proportion of those showing calcified vessels, increased progressively as the degree of arteriosclerosis became more severe, although the increase was not statistically significant. On the other hand, occasionally patients, who did not appear to have much arteriosclerosis clinically, showed calcification radiologically.

Calcification of thigh vessels was much more frequent than that of arm vessels.

5). Conclusions.

There are then doubts about the nature of the arterio-sclerotic process, and its relation to processes inherent in aging. There are doubts about the validity of a method of examination based on palpation. Bearing these things in mind, it is found that increase in palpable thickness of the limb vessels is related to age directly, and is more marked in men than in women. In addition, it is probably related to the deposit of calcium in the limb vessels as seen on X-ray examination.

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CHAPTER IV.

HYPERTENSION AND ARTERIOSCLEROSIS.

1. Hypertension in Old Age. - being a brief summary of the findings in Part II.
 2. Hypertension and Obesity.
 3. Hypertension and Arteriosclerosis.
 4. Mental Changes in Old Age, and their relationship to Arteriosclerosis and Hypertension.
 5. Fundal Changes in Old Age, and their relationship to Arteriosclerosis and Hypertension.
 6. Hemiplegia and Arteriosclerosis.
- - - - -

1). Hypertension in Old Age.

It has been shown, in Part I, that demonstration of a high blood pressure is a feature of the examination of a large proportion of patients over the age of 60 admitted to the medical wards in a municipal hospital. Another large group have pressures which can be defined as systolic hypertension. In Part II, it has been shown that certain "complications" are associated with diastolic hypertension, and that these (hemiplegia, especially) occur more often than in patients with systolic hypertension. Old men are less liable to develop "complications" than men younger than 60 years, though between these same age-groups in women there is no difference in the "virulence" of the hypertension. Although until the age of 60 years, diastolic hypertension occurs more often and is "milder" in its manifestations in women than in men, over the age of 60 years, frequency and severity are the same in men and in women. Hemiplegia and cardiac failure are just as likely to ensue in old age as in middle life, and there is very little difference between the character and incidence of symptoms in the two age-groups. Rapidly progressive hypertension, with severe retinal and renal involvement, is rare in old age, but there does occur a type of slowly progressive renal failure unknown in younger age-groups, and pointing to the inevitable development of renal ischaemia in all degrees of hypertension.

It remains to compare the elderly man and woman with and without hypertension, and in the following chapters this comparison has been made, with respect to mental state, changes in fundus, heart, electrocardiograph, haemoglobin content of the blood, and blood chemistry.

In these determinations, arteriosclerotic changes in the vessel walls must also be taken into consideration - and its relationship to the changes outlined above is also discussed.

2). Hypertension and Obesity.

The belief is widely prevalent that hypertension occurs more commonly in obese persons. Employment of the ordinary sphygmomanometer cuff for measurement of the pressure gives a higher reading in stout persons because of the increased pressure required to compress the larger mass of tissue.

52 male patients over the age of 60 years were carefully weighed several times, at weekly intervals. All were patients who could get about easily, and who could stand on ward scales. No patient with any evidence of cardiac failure was included, and all were weighed in dressing-gown and slippers and at the same time of day. There were very marked variations in the individual measurements, but there was no significant difference between the weight of the hypertensive/

hypertensive patient and the non-hypertensive, although on the average the former was 6 pounds heavier. In old men, then, diastolic hypertension and increased weight do not appear to be associated. (Table 42).

3). Hypertension and Arteriosclerosis.

Hypertension, as we have seen, is associated with arteriolar vasoconstriction, and subsequently with arteriolar sclerosis. But the relationship (if there is one) between hypertension and arteriosclerosis of the large arteries has not yet been defined. Traube, in the very early days (1856), discussing the relation between arteriosclerosis and cardiac hypertrophy, came to the conclusion that as a result of the arteriosclerosis, the blood pressure was increased, and this doctrine held sway until accurate methods of measuring blood-pressure were evolved, and it could be conclusively demonstrated that arteriosclerosis and hypertension did not, by any manner of means, always occur together. Now from Traube's view, the "wheel has come full circle", and Moschowitz (1942), has stated his belief that hypertension, by virtue of the extra wear and tear on the arterial walls, does increase the possibility of atheromatous changes developing. He bases this belief on his observations in the arteries of the pulmonary circulation, where, he says, "arteriosclerotic changes/

TABLE 42.

HYPERTENSION AND WEIGHT (IN POUNDS).

Blood - pressure mm Hg.		
+160/+100	+160/-100	-160/-100
128	120	138
119		
140	116	133
175		131
126	134	
154		121
129	105	105
127		131
120	133	
142		121
98	134	
133		161
131	132	122
124		
120		92
98		115
126		
132		133
162		152
180		
119		77
128		102
98		133
131		133
		133
		118
		147
		100

Av. weight 130.8

124.9

124.2 pounds

changes develop only when the pressure in the pulmonary circulation is increased. This view receives support from the fact that it is a common pathological finding that atheromatous changes are frequent in the lower part of the abdominal aorta and the lower limb arteries which are subject to frequent changes in blood pressure, (Page, 1945). Further, it does seem to be true that hypertension predisposes to coronary artery sclerosis.

But hypertension certainly is not the only factor involved, for gross arteriosclerosis is well-known to exist in the absence of hypertension - a fact confirmed once more in Table 43, where the relationships of arteriosclerosis and hypertension are dealt with. There is no significant relationship between arteriosclerosis and hypertension in my figures.

4). Mental Changes in Old Age.

The cerebral vessels are affected by arteriosclerosis oftener than any other part of the arterial system, except the aorta and the coronary arteries (Cobb and Blain, 1933). It is a matter of great difficulty to decide whether the ensuing changes in cerebral blood supply or merely the involutionary processes consequent upon aging of nervous tissue are responsible for changes such as defect in vibratory sensibility and absence of ankle-jerks, and for senile/

TABLE 43.

BLOOD-PRESSURE AND ARTERIOSCLEROSIS.

A/scl.	-160/-100		+160/-100		+160/+100		TOTALS	
	M.	F.	M.	F.	M.	F.	M.	F.
Nil	11	6	4	2	7	9	22	17
+	10	2	3	1	22	14	35	17
++	19	1	9	2	30	3	58	6
+++	9	1	5	-	15	4	29	5
No inform.	-	-	-	-	4	-	4	-
Totals	49	10	21	5	78	30	148	45

senile cerebellar and amyostatic syndromes, and the senile phychoses. Because a patient has generalised arteriosclerosis, it does not follow that any neurotic symptoms he may show are due to the cerebral vessels being affected, and Eysenck (1946) has shown that, out of her groups of patients, lack of social integration was the factor most powerfully averse to mental health.

Howell (1945) believed that a high blood pressure mitigated the effects of cerebral arteriosclerosis, and, that among patients with grossly sclerotic cerebral vessels, a high blood pressure is beneficial, in that it overcomes the ischaemic effects of the arteriosclerosis.

I have described how in my patients an attempt was made to assess the mental state. In Tables 44, 45 and 46, its relationship to arteriosclerosis and hypertension is shown. There was no correlation between the degree of arteriosclerosis and the patient's mental state; nor was there any relationship between the blood pressure level and the mental state. In Table 40, I gathered together the patients in the two groups with most severe arteriosclerosis, and attempted to establish a relationship between blood pressure level and mental capacity, but from these figures no significant difference could be established between the amount of mental confusion in the various blood pressure groups.

I thus cannot confirm Howell's finding.

TABLE 44. MENTAL STATE AND ARTERIOSCLEROSIS.

A/scler- osis	Mental State				Totals
	Alert	Dull	Confused	Not stated	
Nil	26	8	5	-	39
+	28	11	12	1	52
++	37	14	12	1	64
+++	18	5	10	1	34
No inform	1	-	3	-	4
Totals	109	38	39	3	193

TABLE 45. MENTAL STATE AND BLOOD PRESSURE.

B.P. mm Hg.	Mental State				Totals
	Alert	Dull	Confused	Not stated	
-160/-100	40	10	7	2	59
+160/-100	13	4	9	-	26
+160/+100	57	24	26	1	108
Totals	109	38	39	3	193

TABLE 46. MENTAL STATE, ARTERIOSCLEROSIS AND
BLOOD PRESSURE

B.P.	Mental State			Total
	Alert	Dull	Confused	
-160/-100	20	5	4	29
+160/-100	10	1	5	16
+160/+100	25	13	13	51
Totals	60	19	22	101

5). Fundal Changes in Old Age.

The fundi were examined in 117 patients. Each one was examined personally, generally on a different day from that on which the arteries had been examined, and with no regard to the other findings in the same patient.

The fundus is the only part of the body whose vessels can be examined by sight. Thus the state of the retinal vessels is of special importance, even although those vessels are under special pressure conditions, and may not always reflect exactly the changes in the other vessels of the body.

Certain anatomical peculiarities of the retinal vessels explain the special pathological changes which have been observed. The walls of the arteries are extremely thin, and, for this reason the whole intraocular course of the central retinal artery is morphologically equivalent to the terminal arterioles of other organs, and can be affected by arteriolar sclerosis. On the other hand, the internal elastic lamina of this artery, though reduced "to a mere thread", is still present, and atheromatous changes can take place within the tunica intima. Furthermore, the retinal vessels are confined within a single layer of the retina, and thus the arteries and veins are brought into intimate contact, their tunicae adventitiae becoming closely united at the point of contact.

The changes occurring in the retinal vessels in hypertension have, as we have seen, been used as the basis for Keith,/

Keith, Wagener, and Barker's very useful method of grading essential hypertension so as to give some idea of its severity and course. These authors and Castleman and Smithwick (1943) have shown that there is a correlation between retinal and renal arteriolar change.

Of the 117 patients examined, nine had abnormalities judged not to be connected with arteriosclerotic processes. In the remaining cases, 45 had no abnormality, and 63 had arteriosclerotic changes. The changes accepted as criteria of retinal arteriosclerosis were as follows:-

1. localised constriction in the arterial walls (due to localised atheromatous deposits).
2. narrowing of the calibre of the arteries (due to constriction of the lumen nearer the heart).
3. constrictions at the arterio-venous crossings (due to thickening of the vessel wall).

Tables 47 and 48 show the relation of the occurrence of these changes to general arteriosclerosis and hypertension.

TABLE 47. FUNDAL CHANGES AND ARTERIOSCLEROSIS.

A/sclerosis	Fundus		Total
	Normal	A/s changes	
Nil	14	11	25
+	14	17	31
++	13	22	35
+++	3	13	16
No inform.	1	-	1
Totals	45	63	108

TABLE 48. FUNDAL CHANGES AND BLOOD PRESSURE.

Blood Pressure mm Hg	Fundus		Totals
	Normal	A/s changes	
-160/-100	14	14	28
+160/-100	6	9	15
+160/+100	25	40	65
	45	63	108

There is no significant difference between the proportions of patients with retinal changes in the various hypertensive groups. While the figures in Table 47 are not statistically significant, it will be observed that with increased limb arteriosclerosis there is an increase in the proportion of those having retinal changes, and there would seem to be some correlation between retinal arteriosclerosis and arteriosclerosis in the limb arteries.

6). Hemiplegia and Arteriosclerosis.

I have already discussed, (in Part II, Chapter IV), the relationship between hemiplegia and blood pressure. The figures in this series of cases (vide Table 50) are in keeping with the previous finding, that hemiplegia occurs more commonly in patients with diastolic hypertension.

When/

When however, the frequency of hemiplegia in the various arteriosclerotic groups is considered (vide Table 49), as far as my figures go, there is no relationship between general arteriosclerosis and the occurrence of hemiplegia.

TABLE 49. HEMIPLEGIA AND ARTERIOSCLEROSIS.

A/sclerosis	Hemiplegia	None	Totals
Nil	7	32	39
+	18	34	52
++	12	52	64
+++	6	28	34
No inform.	-	4	4
	43	150	193

TABLE 50. HEMIPLEGIA AND HYPERTENSION.

B.P. mm Hg	Hemiplegia	None	Totals
-160/-100	9	50	59
+160/-100	5	21	26
+160/+100	29	79	108
	43	150	193

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CHAPTER V.

1. Introduction. - The effect of arteriosclerosis on the heart.
2. Heart Sounds.
3. Cardiac Enlargement.
4. Cardiac Decompensation.
5. Discussion.

1). Introduction.

In Part II, Chapter V, I have discussed the question of coronary arteriosclerosis and its effects, in so far as they relate to hypertension. I have shown that coronary sclerosis leads to a disturbance of the myocardium, known as "chronic myocarditis", and that the slow development of this disorder may be interrupted by the sudden onset of thrombosis in one of the sclerosed coronary vessels - or that in certain circumstances, and especially if the myocardium is hypertrophied and relatively ischaemic, angina pectoris may result - or that, whether or not these manifestations of coronary disease take place, congestive cardiac failure may gradually develop. I have shown also that so-called "hypertensive heart disease" is almost always due to coronary sclerosis, and that existing confusion between the terms "hypertensive heart disease" and "arteriosclerotic heart disease" is therefore probably justified. Moreover, the presence of hypertension seems to encourage the development of coronary sclerosis.

But wide-spread atheromatous change in the coronary arteries can occur without hypertension, especially in men (Davis and Klainer. 1940), - and hypertension is not nearly such an important pre-existing factor in men with coronary thrombosis as in women. (Rosenbaum and Levine, 1941, found hypertension/

hypertension in 44% of their male patients with coronary thrombosis and 86% of their female patients). Hypertension is therefore not the only factor of importance in the production of coronary sclerosis.

There is a second way in which arteriosclerotic change may affect the heart and perhaps produce cardiac embarrassment. Endocardial changes may take place analogous to the changes in the tunica intima of the arteries, and the valves may become more rigid as a result of increase in fibrous tissue and changes in the quality of the elastic tissue, while atheromatous deposits may also be laid down. Valvular incompetence or stenosis may result, with corresponding circulatory changes.

Each of the 193 patients in this series was examined with a view to determining whether any clinical changes, referable to the heart, were to be associated with the development of arteriosclerosis and hypertension. Since sometimes, where there was marked emphysema, the borders of cardiac dullness were difficult to define accurately, 61 of the later cases were examined radiologically. Attention was paid to the character of the heart sounds and to the presence or absence of adventitiae. Note was also taken of the degree of cardiac compensation. Electrocardiographic records were obtained from 129 patients.

2). Heart Sounds.

These varied very much in quality, but owing to the not infrequent presence of bronchitis or emphysema, it was judged impracticable to attempt any classification of the results. Accentuation of the second sound at the aortic area, so marked in well-compensated hypertensive cases, was not a feature of this series.

Murmurs, occasionally accompanied by thrills, were heard in 42 patients. All occurred in systole. Most were loud and harsh, or musical in quality. Two were very soft. Four were best heard at the base, and 40 best heard at the apex. One of the patients with a basal murmur had signs typical of aortic incompetence, which was probably due to a syphilitic aortitis. In none of the other three cases with a basal murmur was there typical signs of aortic stenosis. The endocardial changes predominantly affected the mitral valve. None of these patients showed any sign or gave any history of rheumatic or syphilitic disease, and the inference is then that the majority, if not all, of these cardiac murmurs were due to endocardial arteriosclerosis.

As the severity of arteriosclerosis in the limb arteries increased, the percentage of patients with a systolic murmur increased also (vide Table 51), and there was a difference, which was statistically significant, between the proportions of/

of those with no arteriosclerosis and those with arterio-
sclerosis, who had cardiac murmurs.

TABLE 51. CARDIAC MURMURS AND ARTERIOSCLEROSIS.

Heart-sounds	Degree of arteriosclerosis				
	-	+	++	+++	No Inf.
Pure	35	40	43	22	4
With murmurs	4	12	18	10	
Not stated	-	-	3	2	
	39	52	64	34	4

The presence or absence of hypertension (Table 52) made
no difference to the incidence of murmurs.

TABLE 52. CARDIAC MURMURS AND HYPERTENSION.

Heart sounds	Blood-pressure mm Hg.			No Inf.
	-160/-100	+160/-100	+160/+100	
Pure	46	17	77	4
With murmurs	11	8	25	-
Not stated	2	1	2	-
	59	26	104	4

Among the 44 patients with cardiac murmurs, 16 (36.36%) had evidences of congestive cardiac failure. Among the whole 193 patients, 42 (21.76%) had congestive cardiac failure. There is a statistically significant difference between the proportions of patients with and without cardiac murmurs, who had congestive cardiac failure. This would suggest that such murmurs are not so innocuous as is commonly supposed, and that the endocardial changes which they represent, may be one factor tending towards the production of cardiac decompensation in the aged.

3). Cardiac Enlargement.

As I have described, the apex-beat was sought in each case by palpation and by auscultation; when its position was determined by palpation, it was taken as the outermost and lowest point of vigorous pulsation, when by auscultation (a much more un-certain procedure), the point of maximum sound. The distance of the apex-beat from the mid-line was measured, and, if greater than 12 centimetres, the heart was considered to be enlarged.

In 61 cases, the chest was X-rayed to determine whether or not cardiac enlargement was present. The film was taken with a distance of 6 metres between the screen and the X-ray tube. From the film was measured the transverse diameter of the heart, being the sum of the maximal distances of the right/
left

right border from the mid-sternum and of the left border from the mid-sternum. At the same time, the long diameter of the heart was measured, being the distance from the junction of the shadows of the right auricle and great vessels on the right heart border to the point of the cardiac apex. If the transverse diameter were more than 14 cms. or the long diameter more than 15 cms., the heart was considered to be enlarged.

When, however, a comparison was made of patients showing clinical cardiac enlargement and radiological cardiac enlargement, there was a significant difference between them (Table 53). It was therefore thought prudent to consider as being enlarged only those hearts which showed radiological evidence of enlargement. Although the proportion of cases with cardiac enlargement was increased among those with the grosser degrees of arteriosclerosis, the difference was not a significant one (Table 54). Nor is there a significant difference between the amount of enlargement in the various hypertension groups (Table 55).

4). Cardiac Decompensation.

I have already shown that probably the most common cause of cardiac failure in old age is hypertension. But if hypertensive heart disease is nearly always related to coronary sclerosis, there seems no good reason why cases of coronary sclerosis without hypertension, producing chronic myocarditis, should/

TABLE 53.

CARDIAC ENLARGEMENT

(comparison of methods of measurement)

Cardiac Enlargement	Method of examination		Total
	(a) Clinical	(b) X-ray	
Present	47	12	59
Not present	74	49	123
No inform.			(11)
Total	121	61	193

TABLE 54. CARDIAC ENLARGEMENT AND ARTERIOSCLEROSIS

Cardiac Enlargement	Degree of A/sclerosis				Total
	-	+	++	+++	
Present	1	2	4	5	12
Not present	5	13	23	8	49
Total	6	15	27	13	61

TABLE 55.

CARDIAC ENLARGEMENT AND HYPERTENSION

Cardiac Enlargement	Blood-pressure mm Hg.			Total
	-160/-100	+160/-100	+160/+100	
Present	3	2	7	12
Not present	19	7	23	49
Total	22	9	30	61

should not lead eventually to congestive cardiac failure, and so explain some of the cases of cardiac failure whose cause is not obvious by other means. But how is arteriosclerosis of the coronary system to be measured? Sappington and Cook (1936), in their histological study of radial and coronary arteries from the same individual, found very little correlation between the degree of arteriosclerosis in each. Indeed, they stated that the amount by which arteriosclerosis had developed in the radials at 65 years was comparable to that shown in the coronaries at 20 years. Certainly among my patients, there was no increase in the incidence of cardiac failure in the presence of limb arteriosclerosis, or with increase in its severity (Table 56), and, although Table 57 shows an increased incidence of cardiac failure in diastolic hypertension, the increase is not a significant one.

TABLE 56.

CARDIAC FAILURE AND ARTERIOSCLEROSIS.

A/sclerosis	Cardiac Failure			Total
	with	without	Not known	
Nil	8	31	-	39
+	15	37	-	52
++	14	49	1	64
+++	5	29	-	34
No inform.	-	4	-	4
Totals	42	150	1	193

TABLE 57.

CARDIAC FAILURE AND HYPERTENSION.

B.P. mm Hg.	Cardiac Failure			Total
	with	without	Not known	
-160/-100	10	49	-	59
+160/-100	4	22	-	26
+160/+100	28	79	1	108
Totals	42	150	1	193

5). Discussion.

We have seen (Part II, Chapter V) that hypertension, exerting its effect on the myocardium through the medium of coronary artery sclerosis, is the commonest cause of cardiac failure in old age.

But coronary sclerosis can occur without hypertension. There is, however, no easy means of determining its presence - except when cardiac decompensation, coronary thrombosis, or angina pectoris appear, for no relationship has been discovered between the degree of arteriosclerosis in the limb arteries and that in the coronary system.

There does, however, seem to be a relationship between the degree of limb arteriosclerosis and endocardial changes of an atheromatous nature leading to the production of cardiac murmurs. Furthermore, patients with such murmurs are more prone to develop the signs of cardiac decompensation than are other patients. Generalised systemic arteriosclerosis, per se, may therefore play a part in the production of cardiac failure in old age.

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CHAPTER VI.

The Electrocardiograph in Old Age.

- 1). Introduction.
- 2). Heart Rate.
- 3). Rhythm.
- 4). Alterations in Individual Waves and Intervals.
- 5). Conclusions.

1). Introduction.

Electrocardiograph records were made in 129 patients. All the records were taken by myself, each lead being separately calibrated, and the calibration recorded so that an electromotive force of 1 millivolt produced a deflection of 10 m.m. on the record. Leads I, II, and III were the standard leads, while lead IVR was taken with the left arm electrode at the extreme outer border of apex beat as the exploratory electrode, and the right arm electrode as the distal indifferent electrode - following the recommendations of the joint report of the Cardiac Society of Great Britain and Ireland and American Heart Association, in 1939. In several cases, two separate recordings were made at intervals of a few weeks, but in no case did any major event intervene between the recordings, nor was there any important difference between the tracings.

Each record was analysed with regard to the heart-rate and rhythm, to the voltage and duration of the P, QRS, T, and U waves, to the duration of the PR and ST intervals, and to the elevation or depression of the PR and ST segments above or below the iso-electric line. Measurements of amplitude were made in all the leads, and of duration in lead II only - due regard being, however, paid to the fact (as White, Leach, and Foote pointed out in 1941) that the QRS complex may have an isoelectric onset in Lead II, and that this may lead to an error/

error in measurement if the other leads are not also examined. Finally, a comment was added regarding the axis deviation (if any), and the normality or abnormality of the record.

Lewis and Gilder (1912) first constructed tables giving the amplitude of the various waves and the length of the various intervals as determined by their observations on fifty healthy subjects. More recently, electrocardiograph records of patients in late middle life have been studied by Jensen, Smith, and Cartwright (1931-32). Shipley and Hallaran (1936) analysed the records from 200 normal men and women between the ages of 20 and 35, and Hoskin and Jonescu (1940) from 50 women medical students aged 20-27. Chamberlain and Hay (1938) discussed the characteristics of the normal electrocardiogram in patients up to and including the seventh decade of life. Changes with age, they found, were slight - amounting to a slight increase in the amplitude of the Q waves, and a decrease in that of the R waves, and an increased proportion of patients with left axis deviation.

Levitt (1939) examined the records of 100 men and women of 71 years of age or over, excluding patients with any symptoms or signs of cardiovascular-renal disease, or blood pressure above 170/90. Eliaser and Kondo (1941) selected 100 persons over 70 years of age, said to have no evidence/

evidence of cardio-vascular disease but including patients with systolic mitral and aortic murmurs. Fox, Klements, and Mandel (1942) included in their series the records of 300 men and women between the ages of 60 and 100, all ambulatory and some fit for light work, but containing 44.4% of patients with diastolic hypertension, and 61.6% with X-ray evidence of cardiac enlargement. The 102 cases of Taran and Kaye (1944) were aged 60-90 years, and were selected from patients with no history or physical signs of cardiovascular disease. Finally, Fox, Weaver, and Francis (1948) made a further survey of electrocardiographic changes in 300 male inmates of a Farm Colony in New York.

As the standards vary, and the exact definition of "no cardio-vascular disease" is not given, these surveys are not of much help in laying down criteria of normality for elderly people. They are, however, of interest as indicating the type of electrocardiographic change which most commonly occurs in old age. I have assessed "normality" in my records according to the standards laid down for adults in the text-books of Katz (1941) and White (1944), modified, in some instances, by the findings of Chamberlain and Hay (vide supra).

The results of the analyses of electrocardiographic records will be found below :-

2). Heart Rate.

The heart rate is subject in old age, as in adults or children, to many physiological and pathological variations. Table 58 indicates the rate in beats/minute of the 129 patients in the survey.

TABLE 58. HEART RATE

	Rate in beats/min.									Total
	Below 50	50- 59	60- 69	70- 79	80- 89	90- 99	100- 109	110- 119	120 129	
No. patients	1	2	17	29	32	27	13	5	3	129

I have examined more closely the two groups of patients whose heart rate was likely to indicate disease (a) those patients in whom it was rapid, and (b) those in whom it was very slow.

(a) Of the 8 patients whose heart rate was faster than 110 beats/minute, one had auricular fibrillation and cardiac failure, and three had chronic pulmonary infections. Of the other four patients, two had hypertension with hemiplegia, one was a diabetic, and the other a hypertensive patient whose reason for coming into hospital, affirmed by herself, was that "she hadn't the courage to face life". These four patients when re-examined on a subsequent occasion at rest and in the ward, had a heart rate of between 70-80 beats/min.. Thus, none/

none of this group of patients exhibit any peculiarity, with regard to heart rate, which would not be met in ordinary adult practice.

(b) Three patients had a heart rate of less than 60 beats/min. One, aged 64, was in hospital with a rectal neoplasm. He had a true sinus bradycardia with low QRS complexes and a prolonged PR interval, - a phenomenon remarked on by Eliaser and Kondo as occurring "frequently" in their 13 patients with sinus bradycardia. The second, a man aged 75 years with hypertension, hemiplegia, and pulmonary emphysema, had a well-marked sinus arrhythmia. The third patient, aged 79, had auricular fibrillation with a mild degree of congestive cardiac failure. They belonged respectively to the non-hypertensive, diastolic and systolic hypertensive groups, and all three had "Group ++" arteriosclerosis. (With regard to the third patient, I shall add a comment later on auricular fibrillation in old age.)

Seventeen patients had a heart-rate between 60 and 70 beats/min. Of these, one had sinus arrhythmia, and two had auricular fibrillation.

All the 20 patients with a heart rate of less than 70 beats/min have been analysed according to age, degree of arteriosclerosis and blood-pressure, and the results set down in Tables 59, 60 and 61. It was observed that the percentage of patients having a low heart rate increased with the/

TABLE 59.

HEART RATE AND AGE

Pulse Rate	Age-groups					Total
	Under 60	60-69	70-79	80-89	90-99	
Under 70	-	6	12	2	-	20
Over 70	6	48	40	14	1	109
Total	6	54	52	16	1	129
% under 70	0	11.11	23.08	12.50	0	15.5

TABLE 60.

HEART RATE AND ARTERIOSCLEROSIS

Heart Rate	Arteriosclerosis					Total
	nil	+	++	+++	No inf.	
Under 70	1	4	11	4	-	20
Over 70	25	35	31	17	1	109
Total	26	39	42	21	1	129
% under 70	3.85	10.26	26.18	19.05		15.63

TABLE 61.

HEART RATE AND BLOOD PRESSURE

Heart Rate	Blood Pressure			Total
	-160/-100	+160/-100	+160/+100	
Under 70	5	4	11	20
Over 70	33	10	66	109
Total	38	14	77	129
% under 70	13.16	28.58	14.29	15.50

the degree of arteriosclerosis (except the most severe grade). There is actually a relationship here due not merely to chance.

It would appear then, in spite of their absence from the series of Fox, Klements, and Mandel, and Taran and Kaye, that patients with a slow heart rate are not uncommon in old age, and that this phenomenon is significantly more common when there is arteriosclerosis present. Furthermore those whose heart rate is very slow show phenomena characteristic^{of}/geriatric rather than of ordinary adult practice.

3). Changes in Rhythm.

A. Sinus Arrhythmia.

Sinus arrhythmia is commonly associated with youth only. It was found in 46% of Shipley and Hallaran's young adults. It occurred 5 times in Fox, Klement, and Mandel's series, and 6 times among my patients. Though four of my patients had hypertension with hemiplegia, and all had arteriosclerosis, five being "++", - they had no other common characteristics. They were of all ages, up to 79 years of age, and none had cardiac enlargement or cardiac failure. Sinus arrhythmia in my cases was not counted an abnormality.

B. Extrasystoles.

B. Extrasystoles.

The occurrence of extrasystoles was determined by auscultation, since the heart sounds could be listened to for a longer period than could be recorded on the electrocardiographic record. Extrasystoles are said to be more common in the aged. They occurred in 20 of Eliaser and Kondo's 100 cases, and in 38 out of the 300 cases of Fox, Klement, and Mandel (12.7%). They have, of course, both cardiac and extra-cardiac causes, and, though Fox, Klements, and Mandel found them to be associated with abnormal tracings rather than normal, their presence is usually no indication of the condition of the heart, and they were not counted an abnormality. Extrasystoles occurred in 28 of the 193 patients, and Tables 62, 63, and 64 indicate their frequency with reference to age, arteriosclerosis, and blood-pressure. There was no significant relationship with any of these.

TABLE 62. EXTRASYSTOLES AND AGE.

Extrasystoles	Age-groups				Totals
	Under 60	60-69	70-79	Over 80	
Present	3	6	17	2	28
Absent (or not stated)	4	79	60	22	165
Total	7	85	77	24	193
% with E/s	42.86	7.06	20.78	8.33	13.83

TABLE 63. EXTRASYSTOLES AND ARTERIOSCLEROSIS

Extrasystoles	Degree of arteriosclerosis					Totals
	Nil	+	++	+++	No. inf.	
Present	9	2	9	7	1	28
Absent	30	49	52	26	3	160
Not stated	-	1	3	1	-	(5)
Totals	39	52	64	34	4	193
% with E/s	23.08	3.92	14.75	21.21	-	14.68

TABLE 64. EXTRASYSTOLES AND BLOOD-PRESSURE

Extrasystoles	Blood-pressure			Total
	-160/-100	+160/-100	+160/+100	
Present	6	5	17	28
Absent	52	19	89	160
Not stated	1	2	2	5
Total	59	26	108	193
% with E/s	10.35	20.84	16.04	13.83

C. Auricular Fibrillation.

While discussing the heart-rate in old age, I have suggested that auricular fibrillation may have different clinical features - in the shape, for instance, of a slow heart/

heart rate rather than a fast. Brill (1937) affirms that, in old age, auricular fibrillation may occur in an otherwise normal heart, and Boas (1941) describes it as occurring as the sole cause of cardiac abnormality - causing little disturbance, and neither shortening the patient's life nor impairing his efficiency. White and Jones (1928), out of 346 cases with organic heart disease and auricular fibrillation, collected 74 (21.4%) to whom no abnormality save arteriosclerosis could be assigned as the cause of fibrillation.

As regards the incidence of auricular fibrillation in old age, Willius (1931) found that it occurred in 68 (17.9%) of his 700 cases over the age of 75 years (selected from patients who had undergone electrocardiographic examination). Eliaser and Kondo (1944), from their 100 persons over 70 years, found three asymptomatic cases of auricular fibrillation; and Taran and Kaye (1944) found in their patients, aged 60-69, 9% with conduction disturbances (auricular fibrillation and ventricular premature contractions being included with other disturbances), and 20.5% in those aged 80-89 years.

Among my cases were 15 patients with auricular fibrillation. Their distribution in age-groups, and their relation to arteriosclerosis, blood-pressure, cardiac enlargement, and cardiac failure are set down in the following tables :-

TABLE 65.

AURICULAR FIBRILLATION AND AGE

Auric. Fibr.	Age-group					Total
	Under 60	60-69	70-79	80-89	90-94	
With	-	6	6	2	1	15
Without	6	48	46	14	-	114
Totals	6	54	52	16	1	129
% with A.F.	0	11.1	11.5	12.5	0	11.6

TABLE 66.

AURICULAR FIBRILLATION AND ARTERIOSCLEROSIS

Auric. Fibr.	Degree of Arteriosclerosis					Total
	Nil	+	++	+++	No inform	
With	3	2	6	4	-	15
Without	23	37	36	17	1	114
Totals	26	39	42	21	1	129
% with A.F.	11.5	5.1	14.3	19.0	0	11.6

TABLE 67.

AURICULAR FIBRILLATION AND BLOOD-PRESSURE

Auric. Fibr.	Blood-pressure mm Hg.			Total
	-160/-100	+160/-100	+160/+100	
With	4	2	9	15
Without	34	12	68	114
Totals	38	14	77	129
% with A.F.	11.1	14.3	11.7	11.6

TABLE 68.

AURICULAR FIBRILLATION AND CARDIAC ENLARGEMENT

Auric. Fibr.	Cardiac Enlargement		Total
	With	Without	
With	4	0	4
Without	11	37	48
Total	15	37	52

TABLE 69.

AURICULAR FIBRILLATION AND CARDIAC FAILURE

Auric. Fibr.	Cardiac Failure		Total
	With	Without	
With	7	8	15
Without	14	100	114
Total	21	108	129

Auricular fibrillation, then, occurred in all the age-groups, and was not decreased or diminished in incidence as age progressed. There was no significant difference between the proportions of patients in the various arterio-sclerotic groups who had auricular fibrillation, nor did patients with hypertension, more often than those without, have auricular fibrillation. As one would expect, however, cardiac enlargement and cardiac failure were found among patients with auricular fibrillation more commonly than among the other patients.

Seven of the patients with auricular fibrillation had cardiac decompensation, and the average pulse rate of these patients was 90.0 beats/minute, though since all were receiving digitalis therapy, too much emphasis cannot be placed on this figure. Among the cases without cardiac decompensation, the average pulse rate was 73.8 beats/minute. These patients were very little upset by the cardiac irregularity, even though, in addition, three had intra-ventricular block. Two were slightly dyspnoeic, but the others walked about the ward and were completely unaware of any cardiac abnormality.

4). Changes in individual waves and intervals.

D. P Wave.

Katz and White in their respective textbooks have stated that in a normal person, the P wave may be 0-3 mms in amplitude and up to 0.10 sec& in duration. Also, that in Lead III,

the P wave in a normal heart may be diphasic or inverted.

None of my patients had P waves taller than 2 mms., except on a few occasions when in Lead IV their height reached 4 mms.. In my cases, the P wave in lead I was often isoelectric but never inverted. P_2 was usually well-marked. P_3 was diphasic or inverted in 11 cases. There was, of course, no P wave in patients with auricular fibrillation.

E. P-R Interval.

The P-R interval - defined by White as being from the upstroke of the P wave to the beginning of the QRS wave, whether this be an upstroke or a downstroke - is a measure of the time taken for an impulse to be conducted from the sino-auricular node to the auriculo-ventricular node and through the ventricular bundles to the ventricular muscle fibres. In the normal adult, (Katz : White) this process usually takes from 0.12 - 0.20 secs. Chamberlain and Hay (1938) found however, that in some of their normal healthy individuals this conduction time might be as long as 0.22 secs. If prolonged beyond this time, however, we have constituted one form of auriculo-ventricular block.

Fox, Klements, and Mandel (1942) found that among their 300 men and women over the age of 60 years, 40 patients (13.3%) had a prolonged PR interval without clinical impairment. A higher percentage (40%) was found by Eliaser and Kondo (1944) in/

in their patients over the age of 70 years, - while among the group examined by Fox, Weaver, and Francis (1948), there were 5%.

A PR interval of more than 0.20 secs. was found in 43 of my patients, being 33.33% of those who had electrocardiographic records taken. In view of this, and of Chamberlain and Hay's finding of PR intervals of 0.22 secs in normal people, the following tables relate to those patients in whom the PR interval was longer than 0.22 secs., and show the distribution with respect to age, arteriosclerosis, blood-pressure, cardiac enlargement, and cardiac failure.

Four of the patients with an excessively long P-R interval had also a slow pulse rate.

Excessive prolongation of the P-R interval occurred significantly more commonly among the more severe degrees of arteriosclerosis, but did not seem to be directly related to age, to the level of the blood-pressure, or to the presence of cardiac enlargement or cardiac failure.

F. Depression or Elevation of P-R Segment.

Hahn and Langerdorf (1939) found that signs typical of right auricular stress often occurred in arterial hypertension, and later Hahn (1940), defined the signs of right auricular stress as displacement of the P-R segment 0.5 mV. below the iso-potential level. Examining the electrocardiograph records of 200 patients with arterial hypertension, chronic glomerulo-nephritis/

TABLE 70.

PROLONGED P-R INTERVAL AND AGE

PR Interval secs	Age-groups					Total
	60	60-69	70-79	80-89	90-	
Over 0.22	1	6	6	3	-	16
Under 0.22	5	48	46	13	1	113
Total	6	54	52	16	1	129
% with PR over 0.22	16.67	11.11	11.54	18.75	-	12.40

TABLE 71. PROLONGED P-R INTERVAL AND ARTERIOCLEROSIS

PR Interval secs	Degree of S/sclerosis					Total
	Nil	+	++	+++	No inf.	
Over 0.22	1	4	7	4	-	16
Under 0.22	25	35	35	17	1	113
Total	26	39	42	21	1	129
% with PR over 0.22	3.85	10.26	16.67	19.05	-	12.40

TABLE 72. PROLONGED P-R INTERVAL AND BLOOD-PRESSURE

PR Interval secs	Blood-pressure			Total
	-160/-100	+160/-100	+160/+100	
Over 0.22	4	2	10	16
Under 0.22	34	12	67	113
Total	38	14	77	129
% with PR over 0.22	10.53	14.29	12.99	12.40

TABLE 73. PROLONGED P-R INTERVAL AND CARDIAC ENLARGEMENT

PR Interval secs	Cardiac Enlargement		Total
	Present	Absent	
Over 0.22	5	7	12
Under 0.22	10	30	40
Total	15	37	52
% with PR over 0.22	33.33	18.92	23.08

TABLE 74.

PROLONGED P-R INTERVAL AND CARDIAC FAILURE

PR Interval secs	Cardiac Failure		Total
	Present	Absent	
Over 0.22	2	14	16
Under 0.22	19	94	113
Total	21	108	129
% with PR over 0.22	9.52	12.96	12.40

glomerulo-nephritis, or aortic valvular disease, Hahn found pathological depression of the PR segment in 148 cases, whose average age was 55 years. Comparing those patients with the patients without PR segment changes, he found that patients with changes were rather older, showed a higher incidence of left ventricular preponderance, and of "ischaemic heart disease", and probably were rather more severely ill. Hahn suggested that these changes might result from arteriosclerosis of the auricular arteries, causing an insufficient blood supply to the auricular muscle.

PR segment changes of the kind described by Hahn occurred in my patients as follows :-

Lead I	2
Lead I & II	2
Lead II	14
Lead II & III ...	12
Lead III	5
Total	<u>35</u>

Elevation or depression of the PR segment was measured against the iso-electric line, taken as just before the commencement of the P wave. Of the 35 cases, 21 had depression of the ST segment also in one or two leads. Three patients had well-marked Q waves, and one had right bundle-branch block. Four had left ventricular preponderance.

The relation of the 35 patients to age, arteriosclerosis, blood-pressure, cardiac enlargement and cardiac failure is shown in the following tables :-

Age	Arteriosclerosis	Blood-pressure	Cardiac enlargement	Cardiac failure
15	1	1	1	1
20	2	2	2	2
25	3	3	3	3
30	4	4	4	4
35	5	5	5	5
40	6	6	6	6
45	7	7	7	7
50	8	8	8	8
55	9	9	9	9
60	10	10	10	10
65	11	11	11	11
70	12	12	12	12
75	13	13	13	13
80	14	14	14	14
85	15	15	15	15
90	16	16	16	16
95	17	17	17	17
100	18	18	18	18
Total	150	150	150	150

Age	Arteriosclerosis	Blood-pressure	Cardiac enlargement	Cardiac failure
15	1	1	1	1
20	2	2	2	2
25	3	3	3	3
30	4	4	4	4
35	5	5	5	5
40	6	6	6	6
45	7	7	7	7
50	8	8	8	8
55	9	9	9	9
60	10	10	10	10
65	11	11	11	11
70	12	12	12	12
75	13	13	13	13
80	14	14	14	14
85	15	15	15	15
90	16	16	16	16
95	17	17	17	17
100	18	18	18	18
Total	150	150	150	150

Age	Arteriosclerosis	Blood-pressure	Cardiac enlargement	Cardiac failure
15	1	1	1	1
20	2	2	2	2
25	3	3	3	3
30	4	4	4	4
35	5	5	5	5
40	6	6	6	6
45	7	7	7	7
50	8	8	8	8
55	9	9	9	9
60	10	10	10	10
65	11	11	11	11
70	12	12	12	12
75	13	13	13	13
80	14	14	14	14
85	15	15	15	15
90	16	16	16	16
95	17	17	17	17
100	18	18	18	18
Total	150	150	150	150

TABLE 75.

	PR segment present	changes absent	Total	% with PR segt. changes
Under 60	1	5	6	16.67
60-69	13	41	54	24.07
70-79	14	38	52	26.93
80-89	7	9	16	43.75
90-94	-	1	1	
Total	35	94	129	27.13

TABLE 76.

Nil	6	20	26	23.08
+	9	30	39	23.08
++	13	29	42	30.95
+++	6	15	21	28.58
No inform.	1	-	1	
Total	35	94	129	26.57

TABLE 77.

-160/-100	13	25	38	34.21
+160/-100	5	9	14	35.72
+160/+100	17	60	77	22.08
Total	35	94	129	27.13

TABLE 78.

Present	2	13	15	14.29
Absent	15	22	37	40.54
Total	17	35	52	33.33

TABLE 79.

Present	6	15	21	28.58
Absent	29	79	108	26.85
Total	35	94	129	27.13

Neither age, degree of arteriosclerosis, height of blood pressure make any difference to the proportions of patients who had PR segment changes such as Hahn described.

Since Hahn found a higher incidence of PR segment depression in patients with left ventricular preponderance, I have collected patients who exhibited this change (the criteria for which I shall discuss in a subsequent section), and have compared the proportions of patients having PR segment changes and left ventricular preponderance with the proportions of those in which the two conditions were not associated.

TABLE 80. L. VENTRICULAR PREPONDERANCE AND THE PR SEGMENT

L. VENTRIC. Preponderance	PR segment changes		Total	% with PR changes
	Present	Absent		
L.V.P. (Type 2)	4	24	28	14.29
None	31	55	86	36.04
Bundle Branch Block	-	15	15	
Totals	35	94	129	

Thus, in fact, from these figures it appears that significantly fewer patients with left ventricular preponderance have PR segment changes such as Hahn described.

It should be remembered, regarding the PR segment, that after the P wave, there may occur what is called an auricular T wave. The reason for its occurrence in some normal cases and not in others is not known, but it may cause a depression of 1 mm in the PR segment. My 35 cases on review contained only two cases where the PR depression was greater than 1 mm. One of those was a man of 65, reasonably healthy, with no arteriosclerosis of his limb vessels and no high blood pressure. He was a diabetic patient, admitted to hospital for stabilisation of his diabetic condition. The other was a woman of 60 admitted to hospital with pneumonia. She had arteriosclerosis of the "+" variety, but no hypertension. She went out of hospital very well, at the end of four weeks. Neither of these patients, therefore, had gross signs of cardiovascular disease. Where, therefore, PR segment changes were the only electrocardiographic abnormalities, I have counted these records as "normal".

G. Q Wave.

Einthoven called the initial deflection of the QRS complex the Q wave when downward and the R wave when upward.

An initial downward ventricular deflection, or a Q wave, is not always an abnormal finding, and the point at which its presence indicates abnormality has not been clearly defined. Kossman, Shearer, and Texon in 1936, examining/

examining 178 young adults, found that the maximum normal Q wave had an amplitude of 0.2 mV in lead I, 0.25 mV in lead II, and 0.3 mV in lead III. Chamberlain and Hay (1938) found that the maximum Q in their 302 subjects, who were free from heart disease, was 4.5 mm. Apart from the association of an increased Q wave with coronary insufficiency (Pardee: 1930), and with myocardial disease (Edeiken and Wolfert, 1932), the significance of this abnormality remains unknown.

When examining my cases, I have listed as having an abnormally large Q wave those, the amplitude of whose Q wave was greater than 2 mm. or greater than 25% of the largest QRS deflection. All my patients had, as Pardee's (1930) criteria for abnormality also required, normal or left axis deviation. In all, 15 patients had an increased Q wave; it occurred in lead I in 2 cases, in leads I and II in 6 cases, in leads II & III four times, and in lead III three times.

A Q wave of abnormal amplitude might occur in any age-group (see following table). While it occurred less commonly in those without hypertension, the relationship was not a significant one. The Q wave might be seen, both in patients with cardiac failure and those without, and there was no obvious specific change in the clinical condition of those whose electrocardiograph showed a Q wave. It seemed to/

to occur most commonly in those with the lower grades of arteriosclerosis, and less commonly in the severer grades.

H. QRS Complex.

The usual nomenclature accorded to the waves of the QRS complex is that the first small downward projection (which often is not seen at all) is known as the Q wave, the high upward projection is known as the R wave, and the succeeding variable downward projection as the S wave. McGinn and White (1934) have pointed out the inconsistency of this nomenclature, for the Q of lead I may often coincide with the R of lead III, and the R of lead I with the S of lead III. McGinn and White suggest that the QRS wave may be described as consisting of positive and negative deflections above and below the iso-electric line, and Katz in his book does so designate these waves. Nevertheless, the generally accepted nomenclature still is that given above.

Shiple and Hallaran (1936), Chamberlain and Hay (1939), Hoskin and Jonescu (1940), found that the amplitude of the R and S waves varied extremely in health. Steuer (1933) considered their amplitude abnormal when projecting less than 7 mms. from the iso-electric line, and reviewed 50 cases with a voltage of less than this - all being cases which went to autopsy. All showed severe myocardial changes of rheumatic/

TABLE 81.

		Q wave		Total	% with Q wave
		with	without		
Age	Under 60	2	4	6	33.33
	60 - 69	6	48	54	11.11
	70 - 79	4	48	52	7.69
	80 - 89	3	13	16	18.75
	90 - 94	-	1	1	
	Totals	15	114	129	11.62

TABLE 82.

Arterio-sclerosis	Nil	4	22	26	15.39
	+	9	30	39	23.08
	++	1	41	42	2.38
	+++	1	20	21	4.76
	No inform.	-	1	1	
	Totals	15	114	129	11.62

TABLE 83.

Blood-Pressure	-160/-100	2	36	38	5.27
	+160/-100	2	12	14	14.29
	+160/+100	11	66	77	14.29
	Totals	15	114	129	11.62

TABLE 84.

Cardiac Enlargement	With C.E.	2	13	15	13.33
	Without "	5	32	37	13.52
	Totals	7	45	52	13.46

TABLE 85.

Cardiac Failure	With C.F.	2	19	21	9.52
	Without "	13	95	108	12.03
	Totals	15	114	129	11.62

rheumatic, syphilitic, or arteriosclerotic character, Shipley and Hallaran (1936) had 3 cases among their healthy subjects, the amplitude of whose waves was less than 7 mms. Katz considers the waves abnormal when they project less than 5 mms. from the iso-electric line, and the limit set by White is 3 mms.

Levitt (1939) showed that "decreased" ventricular complexes were common in elderly persons. Fox, Klements, and Mandel (1942) found low QRS complexes in 11 out of 300 persons over 60 years of age, and Fox, Weaver and Francis (1948) in 6 out of 300 cases. Among Eliaser and Kondo's (1941) patients, 37% had "decreased" ventricular complexes.

Very low QRS complexes were seen three times in my series. One patient was he previously mentioned, whose electrocardiograph^{ic} record showed sinus bradycardia and a PR interval of 0.20 secs.. Of the other two patients, one had acute pulmonary tuberculosis and the other bronchiectasis. Neither had any clinical myocardial lesion. All the patients belonged to the group without hypertension.

I. Second Positive Wave of QRS₃.

Peel (1939) discussed the significance of a "second positive/

positive wave of QRS" in lead III. He showed that organic heart lesions were more common in patients whose sole electrocardiographic abnormality consisted of "a second positive wave of QRS" in lead III, than in patients whose electrocardiographic records were completely normal, and that the increased frequency of organic heart lesions consisted almost entirely of patients with coronary artery disease, hypertension, or arteriosclerosis.

Among my patients, a second positive wave of QRS₃ was observed twice. The first was in a man, aged 73, who had a hemiplegia without hypertension, but who had "++" arteriosclerosis. The other was in a woman of 51, who, as far as could be ascertained, did not have arteriosclerosis, but who had a grossly elevated blood-pressure, and who, during her stay in hospital, developed hypertensive encephalopathy. She had a slight degree of cardiac enlargement.

Neither patient had any gross electrocardiographic abnormality. The first had an S wave in lead II (left ventricular preponderance, type 1), and the second showed simply left axis shift. Both showed slight depression (1 mm) of the ST segment in lead I.

These cases bear out Peel's contention that such E.C.G.'s often occur in abnormal conditions.

J. Electric Axis Deviation.

The determination of the electric axis of the heart by the electrocardiograph is, at best, only a rough measurement. Einthoven expressed it as an angle, and Carter, Richter, and Greene, modified Einthoven's method to a graphic method based on Einthoven's equilateral triangle. Throughout this study, I have used Katz' empirical method of diagnosing axis shift - using the criteria shown below.

Meek and Wilson (1925) first showed that the changes in the anatomic relationships of ventricles and septum, which occur in hypertrophy of one or other ventricle, are duplicated by rotation of the heart about its longitudinal axis - rotation of the apex to the left and up, producing the changes characteristic of left axis shift, and to the right and down, the changes of the right axis shift.

Kaplan and Katz (1941), and Schnur (1941) have shown by comparison of teleoroentgenograms and post-mortem material with electrocardiograms that, as the heart enlarges, the T wave progressively becomes inverted. 92% of the patients of Kaplan and Katz, in whom the T wave in lead I was negative, had enlarged hearts (i.e. cardiothoracic ratio greater than 1 : 2), whereas there was no case of inversion in the normal heart. Kaplan and Katz then/

then concluded that the presence of ST and T deviations were the distinguishing features of prolonged left ventricular strain, and suggested as their cause changes in the conducting system of the hypertrophied heart, or chronic coronary insufficiency, or insufficiency of the capillary supply to the hypertrophied muscle.

Both sets of investigators found hypertension the commonest cause of left ventricular preponderance.

Langendorf, Hurwitz, and Katz (1943) emphasised that the picture of left ventricular preponderance, as obtained electrocardiographically, indicates not unilateral ventricular hypertrophy, but the site of greater strain - so that the absence of electrocardiographic evidence of ventricular preponderance in the presence of radiological or clinical evidence of cardiac enlargement is presumptive evidence of combined ventricular strain.

The criteria of left and right axis shift and left and right ventricular preponderance were accepted, for my purpose, as described by Katz, and as such are set down here -
Left axis shift.

1. QRS waves of normal duration but mainly, or entirely, inverted in lead III without similar inversion in lead II.
2. No abnormalities in ST segments or T waves.

Left/

Left Ventricular Preponderance.

Type I.

1. QRS complexes mainly or entirely inverted in lead II as well as in lead III.
2. No abnormalities in ST segments, or T waves.

Type II.

1. QRS as in first type.
2. Depression of ST segments and inversion of T waves in lead I or in leads I and II.

(Katz also describes a "concordant" type of left ventricular preponderance, where the ST changes occurred as in type II, but without the QRS changes, indicating either a change in the axis of the heart, or concomitant right ventricular hypertrophy.)

Right axis shift.

1. In lead I very small QRS complex - of 2 mms. or less - or small QRS complex with an S wave where the inverted second phase of the complex is less than the upright first phase.
2. No abnormalities of ST segments or T waves.

Right ventricular preponderance.

1. QRS waves mainly or entirely inverted in lead I.
2. T waves upright in lead I and inverted in lead II or in leads II and III.

Electrocardiographic/

Electrocardiographic studies on old people emphasise the infrequency of right axis shift and the frequency of left axis shift - Taran and Kaye (1944): Eliaser and Kondo (1944). Klainer (1940) showed that out of 36 collected cases of arteriosclerotic and hypertensive disease with right axis shift, 26 had had recent attacks of coronary thrombosis, and in 7 cases the right axis shift eventually altered to left axis shift.

Only one case in my survey had right axis shift. He was a man of 65 with diastolic hypertension and no palpable arteriosclerosis, who complained of breathlessness on exertion and anginal pain for about five years before his eventual admission to hospital with gross congestive cardiac failure. On clinical examination, he had marked cardiac enlargement. With adequate digitalisation and rest, his condition became greatly improved, and he was dismissed from hospital after six weeks. He did not return to the Cardiac Follow-up Clinic, and his subsequent progress is unknown.

The relationship of left ventricular preponderance to arteriosclerosis, blood-pressure, cardiac enlargement, and cardiac failure is set down below in the following tables. 15 cases, having intraventricular block, are not included.

TABLE 86.

		Axis shift			L.Vent P.B.-B.			Total	% with LVP II
		Right	nil	Left	I	II	Block		
Age	Under 60	-	2	2	2	-	-	6	-
	60 - 69	1	12	10	9	14	8	54	25.92
	70 - 79	-	7	12	18	11	4	52	21.15
	80 - 89	-	1	4	6	3	2	16	18.75
	90 - 99	-	-	-	-	-	1	1	-
	Totals		1	22	28	35	28	15	129

TABLE 87.

Arterio-sclerosis	Nil		10		8	5		23	21.74
	+		16		9	9		34	26.46
	++		17		13	7		37	18.92
	+++		8		5	6		19	31.58
	No inform.		-		-	1		1	-
	Totals			51		35	28		114

TABLE 88.

Blood-Pressure	-160/-100		16		10	5		33	15.16
	+160/-100		5		4	2		11	18.18
	+160/+100		30		21	21		72	29.17
	Totals			51		35	28		114

TABLE 89.

Cardiac Enlargement	CE present		6		4	4		14	28.58
	CE absent		13		15	5		33	15.17
	Totals			19		19	9		47

TABLE 90.

Cardiac Failure	CF present		12		2	10		24	41.67
	CF absent		39		33	18		90	20.00
	Totals			51		35	28		114

There was perhaps a tendency for drift towards an increased amount of left axis shift and left ventricular preponderance (Type I) as age advanced. The proportion of patients with preponderance (Type II) had no evident relationship to age, degree of arteriosclerosis, blood-pressure readings, or to cardiac enlargement. Patients with this type of preponderance were more likely to develop cardiac failure.

K. Intraventricular or Bundle-Branch Block.

This is generally an unexpected finding made solely on electrocardiographic examination. It sometimes occurs transiently as in an acute infection, or acute coronary insufficiency, or in acute heart failure; but, in the great majority of cases, it is chronic in character and associated with organic heart disease - rheumatic, syphilitic, or, particularly, arteriosclerotic in origin.

Any QRS complex of more than 0.10 secs. duration in limb leads is considered abnormally prolonged (Shipley and Hallaran ; Chamberlain and Hay.). The term 'intraventricular block' has been applied to complexes of duration 0.10 - 0.12 secs., while those greater than 0.12 secs. have been called bundle-branch block. I have included both types among my 15 patients. Right bundle-branch block indicates those with a downward deflection of the QRS complex/

complex in lead I, while left bundle branch block almost always produced an upright broad deflection in lead I. The T waves in both types were in opposite directions to the QRS waves.

In their review of a 100 patients over the age of 70, Eliaser and Kondo in 1944, found 6 examples of left bundle-branch block, while Fox, Klements and Mandel (1942) found it in 39 (13%) of their patients, and Fox, Weaver, and Francis in 8.7% of their 300 patients. In none of the affected patients in my series were there symptoms of any consequence. Among my 129 E.C.G.'s, there were 15 examples of intraventricular block, 6 of the right branch, and 9 of the left. All had a QRS interval greater than 0.12 secs.

Bundle-branch block occurred at all age-groups - it occurred in the oldest patient in the survey.

TABLE 91.

		I.V. Block	None	Totals	% with IV Block
Age	60 - 69	8	46	54	14.80
	70 - 79	4	48	52	7.61
	80 - 89	2	14	16	12.50
	90 - 94	1	-	1	
	Totals	15	108	123	

TABLE 92.

Arterio-sclerosis	Nil	3	23	26	11.54
	+	5	34	39	12.82
	++	5	37	42	11.91
	+++	2	19	21	9.52
	No inform.	-	1	1	
	Totals	15	114	129	11.72

TABLE 93.

Blood-Pressure	-160/-100	7	31	38	18.42
	+160/-100	3	11	14	21.43
	+160/+100	5	72	77	6.49
	Totals	15	114	129	11.63

TABLE 94.

Cardiac Enlargement	C. Enlargt.	1	14	15	6.67
	None	4	33	37	10.81
	Totals	5	47	52	9.62

TABLE 95.

		I.V.Block	None	Total	% with IV Block
Cardiac Failure	C. Failure	6	24	30	20.00
	Without "	9	90	99	9.09
	Total	15	114	129	11.63

TABLE 96.

* Clinical Abnormality	With clin. abnorm.	11	65	76	14.47
	Without clin. abnorm.	4	49	53	7.53
	Total	15	114	129	11.63

TABLE 97.

Auricular Fibrillation	With A.F.	3	12	15	20.00
	Without "	12	102	114	10.53
	Total	15	114	129	11.63

* Note: 9 out of 15 had complaints with some reference to the circulatory system, and two had a symptomless auricular fibrillation.

There was no significant difference between the proportions of patients with intraventricular block in the various degrees of arteriosclerosis and hypertension. Patients with intraventricular block did not have cardiac enlargement or cardiac failure, nor did they exhibit disease of the cardio-vascular system more often than others.

It was noted, however, that only in one patient with right bundle-branch block, a patient with chronic nephritis, could there be said to be any suggestion of disease of the cardio-vascular system. The others were admitted to hospital because of - 1.) pneumonia, 2.) rheumatoid arthritis, 3.) bilateral inguinal herniae, and symptomless slow auricular fibrillation, 4.) enlarged prostate with overflow incontinence. The fifth patient, an old man of 94, with a symptomless slow auricular fibrillation, was in hospital because he had nowhere else to go.

On the other hand, of patients with left bundle-branch block, 4 had hemiplegia, 3 had congestive cardiac failure, one had gangrene of the left foot, and one had well developed bone changes characteristic of Paget's disease.

This suggests that left bundle-branch block is of more serious import than right, and is in accord with the findings of Perera, Levine, and Erlanger (1942), who showed that the prognosis for patients with right bundle-branch block is much better than for left.

After the electrocardiographic diagnosis had been made, in each of the 15 cases, the cardio-vascular system was carefully re-examined. No clinical indications of any specific cardiac lesion could be found, and, in particular, neither reduplicated heart sounds nor gallop rhythm were ever heard.

L. The ST Segment.

The ST segment and the T wave represent phases of the same process during cardiac systole. In left ventricular preponderance and intraventricular block, we have already seen, characteristic changes take place in both the ST segment and the T wave, which are deviated in the same direction, away from the predominant QRS wave. Gubner and Ungerleider (1943) suggest that these changes are due to relative ischaemia of the deeper layers of the left ventricle.

The measurement of elevation or depression of the ST segment from the iso-electric line has been discussed by Shipley and Hallaran (1936), who pointed out that if the auricular T wave persists for 0.34 - 0.42 secs. as stated by Sprague and White (1925), then the ST interval, as well as the PR interval, will be invaded by it and depressed, and the level of the PR segment should be the correct point from which to measure any superimposed deviations of the ST level. On the other hand, the auricular P wave, during the ST interval, may have reached its lowest point and be coming back to the iso-electric/

iso-electric level. I have made all measurements, as in the PR segment, from the iso-electric line before the P wave begins.

The New York Heart Association (1939) has laid it down that elevation or depression of the ST segment of more than 1 mm., occurring in any two leads, is significant of myocardial disturbance. This degree of deviation of the ST segment occurred in 9 patients, in 6 of whom the T wave was inverted in the same two leads (I & II). All six patients had arteriosclerosis, diastolic hypertension, and congestive cardiac failure. All had left ventricular preponderance.

Of the other three patients, two had diastolic hypertension with left ventricular preponderance of the first type (not yet showing change in the T waves). The third was a patient with auricular fibrillation, bronchitis and emphysema, and who was grossly dyspnoeic. All three patients could, therefore, be said to have "abnormal" hearts.

Inversion of the T wave in leads I and II was also found to be due to changes well recognised. It occurred 28 times with left ventricular preponderance, and three times it could be explained by the exhibition of digitalis. There was no indication of T wave inversion, in any case, being due directly to coronary insufficiency or coronary thrombosis.

M. U Wave.

This interpretation of U, the sixth wave of the electrocardiogram, is not yet understood. It is a small, smooth undulation occurring between the T and P waves, and apparently (according to Papp : 1940) being independent of T and also independent of the heart rate, the R - U period remaining constant despite variation of the heart rate. It was found in lead IV R in 33 of my 129 cases, and in the limb leads in 13 cases. Only three times could it be distinguished in all the leads; most commonly, apart from lead IV R, it occurred in lead II. Its maximum height in the limb leads was 1 mm., and in the fourth lead, 2 mms. It occurred at all ages, up to 88 years.

Papp found that of 100 consecutive E.C.G.'s in which U was higher than 0.5 mm in the limb leads, nearly half the patients had a systolic blood pressure above 160 mm., and 40% had, on radiological examination, an enlarged left ventricle. Of my 129 patients, 5 out of 38 (or 13.16%) of those with no hypertension had U waves in the limb leads, and 2 more (or 18.42%) had U waves in lead IV. Of those with systolic hypertension, 2 out of 14 (14.29%) showed U waves in the limb leads and 3 in lead IV (21.43%), while of those with diastolic/

diastolic hypertension, 6 out of 77 (7.79%) had U waves in the limb leads and 23 (or 29.86%) had U waves in lead IV. There was no significance in these differences between patients with hypertension and those without.

51 of the patients had had a radiological examination of the heart. Of those, 14 had cardiac enlargement, and 37 had not. Of those with cardiac enlargement, 3 showed U waves in the limb leads, and 6 in lead IV, while of the 37 without cardiac enlargement, 5 showed U waves in the limb leads, and 11 in the IVth lead. There is no significant difference between the numbers of those with or without cardiac enlargement who show U waves either in the limb leads or in lead IV alone.

Similarly, there is no significant difference between those with left ventricular preponderance and those without, with regard to the frequency with which U waves are seen.

Nor is there any apparent relationship with the degree of arteriosclerosis observed, nor with the patient's clinical condition. Those in whom the U waves was observed did not develop cardiac failure more frequently than the others - they were a representative group.

A. Tables with Reference to U Wave in Lead IV R.

TABLE 98.

		U Wave		Total	% with U wave
		+	-		
Age	Under 60	2	4	6	33.33
	60 - 69	15	39	54	27.78
	70 - 79	12	40	52	23.10
	80 - 89	4	12	16	25.00
	90 - 99	-	1	1	
	Totals	33	96	129	25.58

TABLE 99.

Arterio-sclerosis	Nil	9	17	26	34.61
	+	8	31	39	20.51
	++	10	32	42	23.81
	+++	6	15	21	28.58
	No infor.	-	1	1	
	Totals	33	96	129	25.77

TABLE 100.

Blood-Pressure	-160/-100	7	33	38	18.43
	+160/-100	3	11	14	21.43
	+160/+100	23	54	77	29.86
	Totals	33	96	129	25.58

TABLE 101.

Cardiac Enlargement	C.Enlargt	6	9	15	40.00
	Without	11	26	37	30.42
	Totals	17	35	52	34.61

TABLE 102.

Cardiac Failure	C.Fail.	6	15	21	28.58
	Without	27	81	108	25.00
	Totals	33	96	129	25.58

TABLE 103.

		U WAVE		Totals	% with U wave
		+	-		
Left Ventric. Preponderance	None	12	39	51	23.53
	L.V.P.(I)	9	26	35	25.71
	L.V.P.(II)	10	18	28	35.72
	(B-B.Block)	(2)	(13)	(15)	(27.01)

B. Tables with Reference to U Wave in Limb Leads.

TABLE 104.

		U WAVE		Totals	% with U wave
		+	-		
Age	Under 60	-	6	6	
	60 - 69	3	51	54	5.56
	70 - 79	8	44	52	15.39
	80 - 89	2	14	16	12.50
	90 - 99	-	1	1	-
	Totals	13	116	129	10.08

TABLE 105.

Arterio-sclerosis	Nil	3	23	26	11.53
	+	4	35	39	10.26
	++	4	38	42	9.53
	+++	2	19	21	9.53
	No inform.	-	1	1	
	Totals	13	116	129	10.16

TABLE 106.

		U WAVE		Total	% with U wave
		+	-		
Blood- Pressure	-160/-100	5	33	38	13.16
	+160/-100	2	12	14	14.29
	+160/+100	6	71	77	7.79
	Totals	13	116	129	10.08

TABLE 107.

Cardiac Enlarge- ment	With C.E.	3	12	15	20.00
	Without "	5	32	37	13.52
	Totals	8	44	52	15.38

TABLE 108.

Cardiac Failure	With C.F.	2	19	21	9.52
	Without "	11	97	108	10.19
	Totals	13	116	129	10.08

TABLE 109.

Left Ventric. Preponderance	None	4	47	51	7.86
	L.V.P.(I)	5	30	35	14.29
	L.V.P.(II)	1	27	28	3.57
	(B-B Block)	(2)	(12)	(14)	
	Totals	10	104	114	8.77

5). Conclusions.

(A) Coronary Sclerosis correlated with Clinical Findings.

In 1942 Katz and his colleagues correlated autopsy reports in 54 patients with electrocardiographic records taken less than 2 months before death. They found -

1. that old healed myocardial infarction may be indistinguishable from left ventricular preponderance without infarction.

2. that coronary sclerosis and myocardial infarction do not give rise to any particular pattern in the electrocardiograph. Of their 50 cases of coronary sclerosis and myocardial fibrosis, 9 showed intraventricular block, 21 ventricular strain patterns, and 20 other non-specific abnormalities. Katz found, however, like Pardee and Price (1938), that marked coronary disease always produced electrocardiographs with definite abnormalities.

Standards of normality, as accepted for my cases, have been discussed in the section above. I shall briefly list them below.

Findings not considered pathological.

1. Heart rate faster than 60 beats/minute.
2. Sinus arrhythmia, or extrasystoles.
3. P wave iso-electric, or up to 3 mms. in amplitude, or 0.1 secs. in duration.
4. P-R interval up to 0.22 secs. in duration.

5./

5. Q wave smaller than 2 mms. or 25% of amplitude of largest R or S wave.
6. QRS complexes greater than 5 mms. amplitude all leads.
7. QRS less than 0.10 secs., even if showing slurring or notching.
8. Left axis shift or left ventricular preponderance (type I),
9. ST segment elevated or depressed less than 1 mm. in two leads.

According to these standards, 52 of the electrocardiographs were normal, and 77 abnormal. The normal electrocardiographs occurred with almost equal frequency among those who had high blood pressure as among those with "normal" blood pressure, nor was there any significant relationship between the proportion of normal to abnormal records and the degree of arteriosclerosis.

It is, however, interesting to note that, in the groups over 70 years of age, the numbers of normal and abnormal records were approximately equal; while in the groups below 70, there were approximately two abnormal records to every "normal" one. The difference, however, is not statistically significant.

Abnormalities/

Abnormalities occurred more commonly among patients with severe arteriosclerosis, and among those with diastolic hypertension, but there was no statistical significance between the proportions; they did not conduce to the appearance of cardiac enlargement or cardiac failure.

If the presence of some non-specific abnormality in the electrocardiograph was an index of coronary sclerosis, its presence could not then be foretold with accuracy by any of the means of clinical cardiac examination indicated above - or by all of them taken into consideration together.

(B) Changes associated with Generalised Arteriosclerosis.

We have seen that a slow pulse rate and a prolonged P - R interval often occurred in association with generalised arteriosclerosis.

(C) Changes associated with Hypertension.

Hypertension in old age induced few electrocardiographic changes - an increase in incidence of left ventricular preponderance being the most important.

Tables relative to E.C.G. abnormalities (as a whole).

TABLE 110.

		E. C. G.		Total	% with ECG abnormal
		Abnormal	Normal		
Age	Under 60	5	1	6	83.33
	60 - 69	36	18	54	66.67
	70 - 79	27	25	52	51.94
	80 - 89	8	8	16	50.00
	90 - 99	1	-	1	
	Totals	77	52	129	59.69

TABLE 111.

Arterio-sclerosis	Nil	17	9	26	65.37
	+	24	15	39	61.53
	++	25	17	42	59.53
	+++	10	11	21	47.62
	No inform	1	-	1	
	Totals	77	52	129	60.22

TABLE 112.

Blood-Pressure	-160/-100	21	17	38	55.26
	+160/-100	8	6	14	57.15
	+160/+100	48	29	77	62.33
	Totals	77	52	129	59.69

TABLE 113.

Cardiac Enlargement	With C.E.	11	4	15	73.33
	Without "	19	18	37	51.36
	Totals	30	22	52	57.69

TABLE 114.

Cardiac Failure	With C.F.	16	5	21	76.19
	Without "	61	47	108	56.48
	Totals	77	52	129	59.69

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CHAPTER VII.

Progressive Cerebral Ischaemia.

Howell in 1941 described a syndrome, progressing towards a fatal termination, and involving changes in mentality and behaviour, a falling blood pressure, and evidence of peripheral circulatory failure. The relation between a falling blood pressure and the appearance of cerebral symptoms in arteriosclerotic subjects suggested to him that the cause was successive ischaemia of cerebral cortex, hypothalamic area, and medulla.

In several of my cases, the approach of death was carefully observed, and I record for each a brief summary of the observations made.

Case 1.

A patient, aged 63, was admitted to hospital on 16.7.46, complaining of increasing fatigue and breathlessness. She had been well until the previous year, when she first felt breathlessness on exertion.

She was a pale thin woman, grossly orthopnoeic on admission, but showing no other signs of congestive cardiac failure. She belonged to the group having most severe arteriosclerosis.

On/

On 29.7.46, oedema was first noticed, and in spite of diuretic therapy, this increased and other signs of congestive failure became manifest.

The following are the successive observations made on blood pressure and blood urea -

<u>Date</u>	<u>B.P.</u>	<u>Blood Urea.</u>
16th July	210/140	60 mgms. %.
20th "	200/120	66 " "
24th "	200/120	78 " "
29th "	180/120	76 " "
2nd August	184/100	94 " "
6th "	168/ 96	138 " "
7th "	196/100	206 " "
8th "	186/100	240 " "
9th "	170/100	280 " "
11th "	166/100	320 " "
13th "	156/ 96	326 " "
15th "	150/ 90	400 " "
17th "	140/ 90	481 " "

This patient showed no signs of mental confusion and no signs of peripheral circulatory failure.

Case 2./

Case 2.

A woman of 72 was admitted to hospital on 1.3.44 because of an attack of vertigo 10 days before. She had a left facial palsy, of upper motor neurone type, and a grossly contracted bladder, the result of long-standing bladder infection. She was one of those unfortunates who had been abandoned by her relatives, and she was always disgruntled and difficult.

One week before her death, on 18.2.46, she became very confused. At that time (11.2.46) her blood pressure was 180/90, and the blood urea estimation showed a concentration of urea of 61 mgms./100 ml. blood. Two days later, the patient lay semi-comatose in bed, all her limbs were blue and cold, pulsation in brachial and dorsalis pedis arteries had disappeared, the blood pressure was 130/90, and the blood urea 286 mgms.%.

Case 3.

A man of 74 was admitted to hospital on 22.1.46 because of increasing swelling of the lower limbs. There were also congestion of the neck veins, enlargement of the liver, and other signs of congestive cardiac failure present on admission. His blood pressure was 175/90 and the/
the/

the blood urea 86 mgms.%.

By 5.2.46, the patient had become confused and noisy at night, getting in and out of bed for no apparent reason. His dementia increased, his congestive failure also increased, and the following readings of blood pressure and blood urea were made :-

<u>Date</u>	<u>B.P.</u>	<u>Blood Urea.</u>
5th February	175/ 60	132 mgms.%.
11th "	160/ 50	184 " "
27th "	140/ 60	284 " "
1st March	130/ ?	344 " "
2nd "	-	472 " "

Here were three patients, therefore, who exhibit some of the phenomena which Howell has described. All were hypertensive patients originally, and two of them, (1) and (3), belonged to the group of patients with severe arteriosclerosis. Cerebral symptoms, except in case (3) were not so marked as were signs associated with peripheral circulatory failure.

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CHAPTER VIII.

THE BLOOD CHEMISTRY IN OLD AGE

1. Haemoglobin
2. Blood Urea
3. Blood Cholesterol
4. Serum Proteins
5. Serum Calcium
6. Serum Inorganic Phosphate.

1. Haemoglobin.

In 173 of the patients in this survey, the haemoglobin was estimated as oxy-haemoglobin, by the photo-electric absorptiometer method (Bell, Chambers, and Waddell : 1945).

The Medical Research Council's Committee on haemoglobin surveys found that in men the haemoglobin level tended to fall with advancing age, while in women it tended to rise when menstruation ceased. Their average figure for the level of haemoglobin for men over 60 was 98.7% Haldane (13.62 gms. haemoglobin per 100 ml. blood), and for women over 60, it was 92.4% (12.75 gms/100 ml).

The levels of haemoglobin obtained in my cases are shown in the following Tables :-

TABLE 115. AGE AND HAEMOGLOBIN LEVEL.

A. Males:

Age	Haemoglobin gms. %.					Total
	Under 11	11-13	13-15	Over 15	Not stated	
Under 60	-	-	-	1	1	2
60 - 69	10	16	19	15	2	62
70 - 79	12	17	13	13	11	66
Over 80	3	4	8	-	3	18
Totals	25	37	40	29	17	148

B. Females:

Age	Haemoglobin gms. %.					Total
	Under 11	11-13	13-15	Over 15	Not stated	
Under 60	1	1	2	-	1	5
60-69	3	7	10	1	2	23
70-79	2	5	3	1	-	11
Over 80	2	3	-	1	-	6
Totals	8	16	15	3	3	45

No significant difference is apparent on comparison of the proportions of men and women, or the proportions in different age-groups having haemoglobin (a) greater than 15 gms.% or (b) less than 11 gms.%.

Tables 116 and 117 show the relation of the haemoglobin level to arteriosclerosis and hypertension. There is no significant difference between the proportions in different arteriosclerotic and hypertensive groups who have high or low levels of haemoglobin.

Table 116/

TABLE 116. ARTERIOSCLEROSIS AND HAEMOGLOBIN LEVEL

A/scl.	Haemoglobin gms. %.					Total
	Under 11	11-13	13-15	Over 15	Not stated	
Nil	5	13	14	2	5	39
+	6	8	18	18	2	52
++	15	22	10	10	7	64
+++	7	10	12	2	3	34
Not stated	-	-	1	-	3	4
Total	33	53	55	32	20	193

TABLE 117. BLOOD PRESSURE AND HAEMOGLOBIN LEVEL

B.P.	Haemoglobin gms. %.					Total
	Under 11	11-13	13-15	Over 15	Not stated	
-160/-100	14	13	29	6	7	59
+160/-100	5	10	5	5	1	26
+160/+100	14	30	31	21	12	108
Total	33	53	55	32	20	193

The following are the causes of anaemia, so far as can be ascertained, in the 25 men and 8 women with haemoglobin levels below 11 gms. %.

<u>Men</u>		<u>Women</u>	
Pernicious anaemia	1	Hypochromic microcytic anaemia	2
Scurvy	3	Rheumatoid arthritis	1
Bronchiectasis	2	Carcinoma cervix	1
Pulmonary tuberculosis	4	No obvious cause	4
Carcinoma - rectum	1		
- stomach	2		
- lungs	2		
Bleeding haemorrhoids	1		
Living alone for many yrs. Sent into hospital for social reasons	5		
No obvious cause	1		

2. BLOOD UREA.

The ordinary clinical methods of measuring renal impairment are well-known to be inaccurate, and to yield results only when gross impairment has taken place. During the last 5-10 years, Homer Smith and his colleagues have elaborated diodrast and inulin clearance tests, for the measurement of renal blood flow and total glomerular filtrate, but they are somewhat elaborate for wide clinical use./

use. Estimations of blood urea were made in 95 of my patients. The urea mechanism is somewhat complicated, and the level in the blood depends not only on glomerular function, but also on the amount reabsorbed from the glomerular filtrate in the tubules. This latter amount is dependent, in turn, upon the urinary volume. Furthermore, the level in the blood fluctuates fairly widely in normal people (between 20 and 40 mgms.%), but the fluctuation is caused by changes in metabolism, and when a patient is in bed in hospital under a regular regime, even a slight rise betokens impairment of the ability of the kidney to eliminate urea.

Stewart (1947) showed that in old age, the average blood urea is increased, and that this is due to an increase of impairment of renal function. He suggested that since renal failure is much commoner in the aged, it may be bound up with changes consequent upon aging, though secondary merely to more fundamental changes. Laroche (1933) and his colleagues found that gradual failure of the kidneys in old age was reflected in a higher level of indoxyl and urea in the blood.

TABLE 118. BLOOD UREA IN OLD AGE

Blood Urea mgms. %.	Age in 5-yr. groups					
	60- 64	65- 69	70- 74	75- 79	80- 84	85- 89
Average value	46.7	36.5	45.3	39.3	50.5	46.7
No. under 40	10	18	13	8	3	1
No. over 40	9	3	14	9	5	2
Total	19	21	27	17	8	3

Table 118 shows that among my patients the average blood urea level did not increase with advancing age, nor did the number of patients with an urea level higher than 40 mgms per 100 ml. blood alter significantly. If, however, we examine more closely those patients with a relatively high blood urea level, in order to determine if possible its cause, we find a picture such as is illustrated in Table 119.

TABLE 119. CAUSE OF HIGH BLOOD UREA IN OLD AGE

CAUSE	Age in 5-yr. groups					
	60- 64	65- 69	70- 74	75- 79	80- 84	85- 89
Cardiac Failure	7	1	1	1	-	-
Renal Failure	-	-	-	1	-	-
Pyuria	-	-	3	-	1	-
Prog. cerebral isch. with periph. circ.: failure	1	-	2	-	-	-
No obvious reason	2	2	8	8	4	2
Total with high blood urea	10	3	14	10	5	2
Total examined	20	21	27	17	8	3

Cardiac failure is, then, an important cause of high blood urea in my elderly patients. The proportion of patients in whom no obvious reason could be found for their high blood urea increased significantly with advancing years, from 9.76 in the sixties, to 54.55% in the eighties. These patients belonged to all grades of arteriosclerosis (see Table 120). Many of them were hypertensives, there being a significantly larger/

larger proportion (38.46%) of patients with hypertension among them than among the series as a whole.

TABLE 120. DEGREE OF ARTERIOSCLEROSIS AMONG PATIENTS WITH HIGH BLOOD UREA OF NO OBVIOUS CAUSE.

A/Sclerosis	No. with High blood urea	Total investigated	% with elevation
Nil	5	20	25.00
+	10	34	29.41
++	9	32	28.13
+++	2	10	20.00
Totals	26	96	27.08

TABLE 121. HYPERTENSION IN PATIENTS WITH HIGH BLOOD UREA.

Hypertension	Blood Urea		Total	% with high blood urea
	high	normal		
With	20	32	52	38.46
Without	6	38	44	13.63
Totals	26	70	96	27.08

Hypertension is thus an important factor in inducing the impairment of renal function which occurs in old age.

3. BLOOD CHOLESTEROL.

The blood cholesterol level was ascertained in 54 patients, using the Sackett Method, as described by King (1946). One observation only was made in each patient, generally 7-14 days after his admission to hospital. The figures for the observations are therefore, in a sense, inadequate in that they have not been checked by further observations. But all have been taken after the patient had been on a reasonably stable dietetic regime for at least a week.

The following tables show how the values varied, in my patients, with sex, age, degree of arteriosclerosis, and hypertension. As the normal blood cholesterol level varies between 100 and 200 mgms/100 ml. blood, for the purposes of comparison between the various groups, I have considered the number of patients whose cholesterol level was above 150 mgms.% in terms of the total number.

TABLE 122. BLOOD CHOLESTEROL AND SEX

	No.	Av. level mgms.%.	No. over 150 mgms.%.
Male	40	137.13	11
Female	14	142.07	5

TABLE 123. BLOOD CHOLESTEROL LEVEL AND AGE

Age	Av. level mgms.%. %	No. over 150 mgms. %	Total	% age
60-64	136.2	4	10	33.33
65-69	152.7	4	14	
70-74	131.1	3	13	28.57
75-79	149.4	3	8	
Over 80	111.3	1	7	

TABLE 124. BLOOD CHOLESTEROL LEVEL AND ARTERIOSCLEROSIS

A/s	Av. level mgms.%. %	No. over 150 mgms.%. %	Total	% age
Nil	129.7	3	13	23.08
+	137.5	6	24	25.00
++	144.3	4	12	33.33
+++	155.4	3	5	60.00

Neither age nor sex make any difference to the average cholesterol level (in my patients), or to the number whose blood cholesterol level was in the higher "normal" group.

Although the figures in Table 124 are not significant, they do suggest that with a larger group of patients it might be shown that the patients with higher degrees of arteriosclerosis also had higher blood cholesterol levels.

TABLE 125. BLOOD CHOLESTEROL LEVEL AND BLOOD PRESSURE

B.P.	Average level	No. over 150 mgms.%	Total	% age
-160/-100	116	1	6	16.67
+160/-100	135.8	2	8	25.00
+160/+100	145.1	13	40	32.50

These figures are not significant.

Cholesterol is widely distributed in the cells of the body, including the red blood cells, as free cholesterol and cholesterol esters. It is ingested in the food, absorbed by the intestinal lymphatics, conjugated in the liver, and excreted in the bile and faeces, a small amount being re-absorbed in the duodenum. The thyroid gland exercises a regulatory effect upon its utilisation and deposition.

In Chapter III, I have briefly discussed the nature of the changes which take place in arteriosclerosis, and whether these are in fact consequent upon aging, or alternatively whether they result from disease of the vessels occurring mainly in later life. I have likewise discussed the differentiation between the hardening and fibrosis of the tunica media (believed to follow on hypertension), and the development of atheromatous deposits in the tunica intima. It is the presence of atheromatosis in the coronary arteries which is responsible for the narrowing of their lumina and for the production of coronary thrombosis.

Much evidence exists to suggest that an increased cholesterol level in the blood is important in the production of atheromatous lesions. Hueper (1947) and Page (1941) instance, in support of this belief, the early and excessive atherosclerosis which occurs in man and animals in hypothyroidism, diabetes mellitus, or other conditions where the plasma has an excessive lipid content. Hueper has shown that experimental cholesterol atheromatosis occurs only after hypercholesteremia has been produced. Dry and Hines (1941) have commented on the role of diabetes in the production of atheromatosis, and the markedly higher incidence of occlusive peripheral arteriosclerosis among diabetic patients as well as the significantly higher incidence of the more severe complications of arteriosclerosis.

In peripheral sclerosis, some observers have reported high values of blood cholesterol (Barker, 1939), while others have found them not significantly elevated (Nuzum and Elliott, 1936; Jacobi, 1937). The majority of observations suggest that where there is coronary arteriosclerosis, the serum cholesterol level is high. Poindexter and Bruger (1938) found, on comparing the total cholesterol content of the plasma in normal subjects and patients with rheumatic and arteriosclerotic heart disease with or without hypertension, that the concentration of cholesterol in the plasma was much increased in patients with arteriosclerotic heart disease.

Steiner/

Steiner and Domanski (1943) found that the average serum cholesterol values for patients with coronary arteriosclerosis were increased significantly above those with none. Davis and his colleagues (1937) found an increase in blood lipids and cholesterol in patients with angina pectoris. On the other hand, Wolff and his colleagues (1945) found that in male patients with coronary sclerosis, the cholesterol values were not different from those in other male patients.

Age, itself, seems to have no influence on hyperlipaemia (Page et al. - 1935) - though Wolff found the highest values between the ages of 61 and 70 years. Bruger and Chassin (1941), however, found that the cholesterol content of the thoracic aorta increased with age though that of the main renal artery remained fairly constant throughout life. They also found that the degree of pathological change in the kidney, or heart, varied directly with concentration of cholesterol in main renal artery or thoracic aorta. Wolff found cholesterol levels higher in female than in male patients.

Hulse (1926) and Poindexter and Bruger (1938) discovered a relation between hypercholesterolaemia and hypertension. Page, Kirk, and Van Slyke (1936) denied any such relationship. Bruger and Chassin found the cholesterol content of both the main renal arteries and the thoracic aorta greater in essential hypertension.

The/

The mechanism of production of atheromatosis and deposition of lipid material is not known. The theory of Virchow (1871) and Aschoff (1932) assumed that the lipids from the plasma were absorbed directly through the endothelium to the intima. Virchow believed that mechanical strain was the significant factor inducing this change, and Aschoff incriminated the relationship of cholesterol esters and cholesterol in the blood and tissues.

Winternitz (1938) believed that local haemorrhages and exudations in the intimal wall were important contributory factors, and Hueper (1947) suggested that the primary change was "hypoxia" and impairment of the nutrition of the vascular wall, and that subsequently phagocytic, proliferative, and degenerative changes occurred on these as base.

The effect of the amount of ingested cholesterol on the blood cholesterol level is also a matter of controversy. Page and Farr (1936), and Turner and Steiner (1939) have been unable to influence blood cholesterol levels by dieting. Steiner and Domanski in 1941 were, however, able to raise the blood cholesterol appreciably in 10 patients by giving large amounts of egg-yolk powder daily for several weeks, and Rabinowitch (1935) presents strong evidence in favour of the view that administration of high carbohydrate - low fat diets in diabetes delays the development of atherosclerosis.

Chronic diabetic and de-pancreatised dogs maintained
on/

on insulin develop fatal fatty changes of the liver, due to the upset in cholesterol metabolism. Allen and others found that this could be prevented by feeding fresh whole pancreas. Subsequently dried pancreas, then extracts of pancreas, then insulin-free de-cholesterizing extracts of pancreas, then choline and methionine were used for the same effect.

In conclusion, then, it is not unlikely that, as Wolffe (1947) suggests, atheromatosis is a systemic condition of abnormal metabolism manifesting itself in the arteries. If this is true, there are three possible ways of delaying its advance, though none of these ways is as yet proven to be effective -

1. Administration of a low-fat diet.
2. Administration of iodine or potassium iodide or thyroid hormone.
3. Administration, in some form, of the internal secretion of the pancreas.

4. SERUM PROTEINS.

The serum protein level was estimated in 56 patients by the Biuret Method, as set forth by Harrison (1947).

Stewart (1947) inferred that the concentration of serum proteins diminished slightly as age increased. The values in my patients were as follows:-

TABLE 126. SERUM PROTEIN LEVELS AND AGE

<u>Age</u>	<u>Av. serum protein level (gms. per 100 ml.)</u>
60 - 64	6.70
65 - 69	6.85
70 - 74	6.75
75 - 79	6.90
Over 80	6.34

TABLE 127. SERUM PROTEIN LEVELS AND ARTERIOSCLEROSIS

<u>A/sclerosis</u>	<u>Av. serum protein level (gms. per 100 ml.)</u>
Nil	6.83
+	6.87
++	6.42
+++	6.81

TABLE 128. SERUM PROTEIN LEVELS AND BLOOD PRESSURE

<u>Blood Pressure</u>	<u>Av. serum protein level</u>
mm Hg.	gms. per 100 ml.
-160/-100	6.33
+160/-100	6.61
+160/+100	6.84

Age, degree of arteriosclerosis and hypertension made no significant difference to the serum protein level in my cases.

5. SERUM CALCIUM.

The serum calcium level was estimated in 55 patients by the Clark-Collip Modification of the Kramer-Tisdall Method, as described by Hawk and Bergeim (1938). The results are briefly shown below:-

TABLE 129. SERUM CALCIUM AND AGE

Age	No. patients	Av. level Ca. mgms. %.	No. below 9.5 mgms. %
60-64	9	10.80	2
65-69	12	10.46	1
70-74	14	10.33	3
75-79	9	10.03	2
80-84	6	9.83	1

TABLE 130. SERUM CALCIUM AND ARTERIOSCLEROSIS

A/s	No. patients	Av. level Ca. mgms. %.	No. below 9.5 mgms. %
Nil	12	10.3	1
+	23	10.5	3
++	15	10.5	3
+++	5	9.4	2

TABLE 131. SERUM CALCIUM AND BLOOD PRESSURE

B.P. mm Hg.	No. patients	Av. level Ca. mgms.%	No. under 9.5 mgms.%
-160/-100	7	10.93	1
+160/+100	9	9.96	2
+160/±100	36	10.31	7

These figures show no significant change in the serum calcium level with age, arteriosclerosis, or blood pressure. The series is much too small, and contains too many diseased persons. The figures for the mean levels in the various age-groups do, however, suggest lowering of the calcium level with advancing age. Reports in the literature on this subject are conflicting. Major (1929) and Gresheimer, Johnson, and Ryan (1929) suggest that the serum calcium level does tend to decrease with advancing age, while Robertson (1941) in 15 healthy old people found no difference from the range of values seen in younger groups, and Page and his colleagues (1935) affirmed that up to the age of 85, the plasma calcium level falls within the range characteristic of adult men.

6. SERUM INORGANIC PHOSPHATE

The serum inorganic phosphate was estimated by Youngburg's Method. Between the mean values in the various age-groups, arteriosclerotic groups, and hypertensive groups, there was no significant difference.

TABLE 132. SERUM INORGANIC PHOSPHATE AND AGE

<u>Age-Group</u> (yrs.)	<u>Mean Value</u> (mgms. per 100 ml. blood)	<u>Number of</u> <u>patients</u>
60-64	3.75	8
65-69	3.54	13
70-74	3.43	15
75-79	3.20	9
Over 79	4.12	6

TABLE 133. SERUM INORGANIC PHOSPHATE AND ARTERIOSCLEROSIS

<u>Arteriosclerosis</u>	<u>Mean Value</u>	<u>Number of</u> <u>patients</u>
Nil	3.63	12
+	3.59	23
++	3.47	12
+++	3.42	5

TABLE 134/

TABLE 134. SERUM INORGANIC PHOSPHATE AND BLOOD PRESSURE

<u>Blood Pressure</u> mm. Hg.	<u>Mean Value</u> (mgms. per 100 ml. blood)	<u>No. of</u> <u>patients</u>
-160/-100	3.74	7
+160/-100	3.44	9
±160/±100	3.54	36

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DISCUSSION.

DISCUSSION:

Among patients admitted to the medical wards in a municipal hospital, there was a markedly increased incidence of high blood pressure in those over 60 years compared with those under 60 years. An impression was gained that hypertension in older age-groups was more often asymptomatic and uncomplicated, but on further examination this was found to be not quite accurate, for only in men was hypertension more often associated with symptoms or complications under the age of 60 years. There was another difference between the sexes under 60, for in those age-groups, hypertension was much more common in women than in men, though less severe in its effects. Over the age of 60 years, not only was the incidence of hypertension in the two sexes more nearly equal, but also there was not so much difference between the occurrence of complications.

There was also a difference between the two age-groups in the way in which hypertension manifested itself. Cardiac failure and hemiplegia occurred equally frequently in middle-aged and elderly, but in the younger age-group severe progressing hypertension was more often to be found, while on the other hand, in old age, a slowly progressing type of renal failure was occasionally seen.

A remarkable feature of the whole series of patients, however, was that rapidly progressing hypertension was so infrequently found. There was a very large number of hypertensive/

hypertensive persons in whom headache, retinitis, and their associated phenomena did not occur.

Cardiac failure in hypertension is associated intimately with arteriosclerosis of the coronary system. The occurrence of hemiplegia probably has some relationship with arteriosclerotic changes in the cerebral vessels. The nature of arteriosclerosis is still unknown, and its terminology is very confusing, but it is likely that atheromatous changes in the coronary and cerebral vessels are of major importance as far as the above disorders are concerned. Yet the relationship between atheroma and hypertension is obscure. Atheromatosis, we have seen, is now believed to be due to defect in lipid metabolism, and there is only doubtful evidence of such defect in hypertension.

Over 60 years, and especially in men, the walls of the limb and temporal arteries were firm and rigid on palpation, to a varying degree, in a large proportion of my patients, and the incidence of severe "arteriosclerosis" varied directly with age. Some writers have believed this change to be due solely to changes in the tunica media of the vessels, namely hardening and fibrosis consequent upon hypertension. But it is difficult to reconcile this with the fact that, while hypertension occurs equally commonly in the two sexes, changes on palpation are much more common in men. Atheromatous changes in the tunica intima of the arteries have been shown to/

to be more common in men, and it may be that either per se, or by means of involvement of the intima, atheroma plays some part in determining the condition of the vessel wall on palpation.

No relationship between coronary arteriosclerosis and changes in the radial vessels has been established.

I have not been able to confirm Trevor Howell's finding that high blood pressure lessened the incidence of mental confusion due to cerebral ischaemia in patients with gross arteriosclerosis.

I found that, among my elderly patients, changes in the fundus characteristic of retinal arteriosclerosis were related more closely to the degree of arteriosclerosis of the limb vessels than to the presence of hypertension. This would suggest that, in old age, retinal arteriosclerosis is not such a good indicator of the severity and degree of hypertension as it is in younger groups - and it suggests the possibility, also, of severe retinal arteriosclerosis occurring without hypertension.

Hypertension was the commonest cause of cardiac failure in old age. Systolic murmurs at mitral and aortic area occurred in some of my patients and more commonly with increase in severity of arteriosclerosis. Patients with murmurs showed an increased incidence of cardiac failure.

The/

The electrocardiograph in old age revealed changes which do not occur in youth, notably, in some patients, a slow heart rate, and prolongation of the auriculo-ventricular conduction time. Both were associated with severer degrees of arteriosclerosis. My selection of electrocardiographic records, as would be expected in a group of sick people, showed a high incidence of abnormalities. Non-specific abnormalities, such as a large Q wave, intraventricular block, or elevation or depression of the ST segment without change in the T wave, were taken as evidence of coronary sclerosis, but no relationship was found between such abnormalities and the presence of hypertension or arteriosclerosis of the limb vessels.

In old age, there was an increased incidence of blood urea levels higher than the ordinary adult ones. These occurred more often in patients with hypertension, indicating an increased frequency of impairment of renal function in hypertension.

Figures for blood cholesterol and serum proteins and calcium showed no significant changes.

In conclusion, then, arteriosclerosis probably increases the likelihood of cardiac decompensation, and it is associated with the slow pulse rate and lengthening of the auriculo-ventricular conduction period which occur in old age. It is more/

more often associated with retinal sclerosis than is hypertension. The suggestion that atheroma is a defect of cholesterol metabolism raises hopes of delaying or preventing its onset, and thus that of "arteriosclerotic heart disease".

Hypertension renders the old man or woman liable to congestive cardiac failure or to hemiplegia. Also, it produces an increased incidence of impairment of renal function, which, as we have seen, may lead to death from renal failure. No satisfactory explanation is available for the increased incidence of hypertension in old age, and, until the reason for hypertension is elucidated and such explanation found, treatment can be directed only to amelioration of its effects. But the onset of cardiac failure can be delayed, and the victim of hemiplegia can once more take his rightful place in society.

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N O T E.

THE STATISTICAL METHODS EMPLOYED IN THIS STUDY.

For the calculation of the "significance" of differences between proportions, the " X^2 " test has been used throughout. For each table, whatever the number of degrees of freedom, when the probability was that less than once in twenty times we might have reached merely by chance the differing proportions actually observed, the differences were said to be "significant". In other words, when Fisher's table of X^2 was used, P of less than 0.05 was accounted as "significant".

The "t" test was employed in the calculation of differences between means in Table 42 - Hypertension and Weight.

REFERENCE

Table of X^2 , as quoted by Hill, A.B. Principles of Medical Statistics. The Lancet Ltd. (London. 1942).

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