

1947

## Introduction to the aged patient

Edwin Theodore Cooke  
*University of Nebraska Medical Center*

This manuscript is historical in nature and may not reflect current medical research and practice. Search [PubMed](#) for current research.

Follow this and additional works at: <https://digitalcommons.unmc.edu/mdtheses>

---

### Recommended Citation

Cooke, Edwin Theodore, "Introduction to the aged patient" (1947). *MD Theses*. 1445.  
<https://digitalcommons.unmc.edu/mdtheses/1445>

This Thesis is brought to you for free and open access by the Special Collections at DigitalCommons@UNMC. It has been accepted for inclusion in MD Theses by an authorized administrator of DigitalCommons@UNMC. For more information, please contact [digitalcommons@unmc.edu](mailto:digitalcommons@unmc.edu).

AN INTRODUCTION

to the

AGED PATIENT

by

EDWIN THEODORE COOKE

Senior Thesis

Presented to

University of Nebraska

College of Medicine

Omaha, 1947



## CONTENTS

Introduction -----	i
Importance to the Physician:	
What is Aging?-----	1
Biological vs Chronological Aging -----	1
Senescence -----	2
Principle Aging Processes -----	4
Cause of Aging -----	5
Theories -----	5
Irreversible Changes? -----	5
Aging of Colloids? -----	5
Reticulo-endothelial Changes? -----	6
Factors Influencing Age -----	13
Heredity -----	13
Environment -----	14
Other Factors -----	15
Effects of the Nervous System -----	16
Effects of Infection -----	17
What is "Normal" Aging? -----	18
Statistics -----	21
Reliability of Present Statistics -----	22
Change in Characteristic Diseases -----	24
Heart Disease -----	25
Chronic Diseases -----	28
Neglect -----	29
History of Geriatric Medicine -----	34



The Problem of "Normality":

Cardiovascular System -----	38
Heart -----	40
Anatomical Changes -----	40
Physiological Changes -----	42
Arteriosclerosis -----	45
Blood Vessels -----	49
Anatomical Changes -----	49
Physiological Changes -----	53
Changes in the Blood -----	56
Respiratory System -----	59
Nervous System -----	63
Mental Changes -----	63
Importance -----	63
Motor Ability -----	64
Intelligence -----	65
Learning Ability -----	65
Etiology -----	66
Symptoms of Decline -----	68
Mental Change and Arteriosclerosis ----	71
Neurology -----	75
Anatomical Changes -----	75
Brain -----	75
Spinal Chord -----	76
Peripheral Nerves -----	77

Physiological Changes -----	78
Motor Ability -----	78
Sensation -----	78
Reflexes -----	79
Electro-encaphlogram -----	79
Special Senses -----	80
Ear -----	80
Eye -----	81
Digestive System -----	86
Nutrition -----	86
Age Changes in Digestive System -----	89
Salivary Glands -----	89
Stomach -----	90
Pancreas -----	91
Liver -----	92
Gallbladder -----	93
Small Intestines -----	93
Appendix -----	94
Constipation -----	94
Genitourinary System -----	96
Primary Kidney Changes -----	96
Vascular Changes -----	96
Secondary Degenerative Changes -----	97
Secondary Hypertrophic Changes -----	98
Prostate -----	100

Male Genetalia -----	101
Female Genetalia -----	102
Endocrine System -----	109
Hormones and Aging -----	110
Adrenals -----	112
Pituitary Gland -----	114
Thyroid -----	117
Female and Male Hormones -----	120
Other Systems -----	121
Muscular System -----	121
Smooth Muscle -----	121
Striated Muscle -----	121
Changes in Anatomy -----	121
Changes in Function -----	122
Changes in the Vessels -----	123
Changes in Chemistry -----	123
Integumentary System -----	124
Skeletal System -----	126
<b>Diagnostic Differences:</b>	
Difficulties in Clinical Diagnosis -----	128
Difficulties Inherent in the Disease ----	128
Difficulties Inherent in the Patient ----	135
Difficulties due to the Inadequacy of	
Clinical Methods -----	138
Difficulties Inherent in Diagnosis -----	140

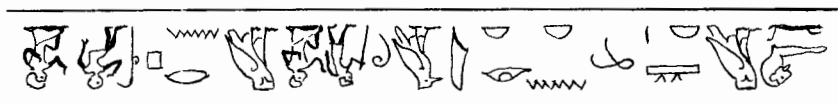
New Ideas of Therapy for the Old:

In Diseases Occurring in the Older Patient -- 143

Types of Treatment Best Suited for the Aging- 151

Summary ----- 153

Bibliography ----- 156



# INTRODUCTION

Why all this fuss over the aged patient? What makes him so different than any other patient the physician sees? If he is different, how is diagnosis and therapy influenced by this change from maturity to senility? Above all, what good can medicine do to a man already doomed to die? Why is there need for an introduction to the aged patient?

In the following pages, it has been attempted to answer some of these questions. In doing so, it is inferred that this paper is an argument in favor to the proposition that the aged pose a special problem to the medical profession and that this problem needs more emphasis than it has now. The main opposition is not an organized body of thought, nor any writer proposing an opposing view. The resistance arises out of apathy, indifference, ignorance and even outright avoidance of anything included under "old age". In spite of the fact that we are all, barring accident, going to become "old" very little attention is paid to the fact. Neglect is a hard word, but it seems applicable in this situation.

The terms and most of the information included in this thesis are general and much of the writing will seem philosophical. There is no disease taken up, with a few exceptions, to limit the length of the paper. It is the intention of the author to do just as stated in the title --- to give the reader a glimpse of the aged patient.

IMPORTANCE  
TO  
THE PHYSICIAN



## "WHAT IS AGING?"

How are we to define old age? What yard-stick is the proper one to use? Are we to judge by the tick of the clock, the turn of the calendar, the condition of the arteries, the metabolic rate, the alertness of the mind, the physical prowess of an individual, or by reaction to the early days of spring?

In the days of economic stress, the man over 40, especially in the business world, finds that he is beginning to pass the optimum of desirability. The man over 50 is something of an anomaly and ready for the traditional gold watch. We have here a paradox. He is young physically and mentally, but old economically.

In competitive sports, in combat warfare, in hazardous undertakings, the accent is strongly placed on youth. When a prize fighter gets to be 28 he is aging, and when he is 38 years he is old, physically perfect though he may be.

### Biological vs Chronological Aging

Age is relative. Aging changes do not conform to rigid calibration on the scale of chronological time. Biological and chronological age are rarely the same. There are many of us physiologically older than our elapsed years and some few younger than our chronological age. Furthermore, no one individual is of uniform physiological age throughout. The rate of aging varies with certain certain cell types and with functional units or



organs. For example, the cells of the epidermis are relatively short lived and are constantly being replaced, whereas the nerve cells of the cerebral cortex are not regenerated as long as the patient survives. The female organs of reproduction go through a rapid involution at the time of the climacteric. Biological age is a composite sum of the extent of aging of all the constituent structures of the body.

### Senescence

It is necessary to differentiate between senescence and senility. Senescence is a physiological aging and senility is abnormal old age. It is not at all possible to draw a sharp line between senescence and senility, since there is no definite connection between chronological and biological age. (182)

The human body is destined to decline in functional capacity as it grows old and finally must die. The body cells slowly undergo atrophy as life advances. The parenchymatous elements of the internal organs gradually suffer and the interstitial tissue of these organs undergo increase during senescence. The atrophy of the body cells that occurs in physiological old age is the morphological expression of the reduction of the energy processes of those cells. Whether this reduction is due to increase of specific growth inhibitory factors, or to decrease of specific growth stimulating factors is not as important as that it does occur and we can do little about it.

The general anatomical changes in the old body constitute a changed internal environment that attempts to maintain equilibrium with an unchanging external environment. When an organ has successfully escaped serious disease and can continue to work in harmony with other organs, the slow changes of age need not affect its' relative efficiency. However, disease in a tissue even at an early age may quickly simulate the total change that might, in the course of events, have taken place much later in life. Thus an old tissue and a tissue bearing the scars of disease may appear very similar.

Senescence, like growth and development, brings insidious and occult changes affecting all the structures and activities of the structures and activities of the individual. The elderly are not just "old people", they are physically and mentally different men and women than in the days of their young maturity. These changes are of the utmost significance to the practice of medicine. Normality, it must be realized, is not a fixed point but a series of variables which change with age. Recognition of this fact is essential in diagnosis, prognosis, and treatment. The early manifestations of incipient disease are readily confused with changes of senescence. Disease, after all, rarely introduces new phenomena; the manifestations arise from exaggerations or distortions of the normal reactive mechanisms.

## PRINCIPLE AGING PROCESSES

(148)

1. Gradual tissue desiccation.
2. Gradual retardation of cellular growth, reproduction and repair.
3. Gradual retardation of the rate of tissue oxidation.
4. Cellular atrophy, degeneration, pigmentation and fatty infiltration.
5. Gradual decrease in tissue elasticity and degeneration in elastic tissue.
6. Decreased speed, strength, and endurance of skeletal neuromuscular reaction.
7. Atrophy of the nervous system.

A glance at these fundamental changes will show that age would affect the etiology, pathogenesis, pathology, course and prognosis, and therapy of all disorders, whether they be characteristic of the age group or not.

## CAUSE OF AGING

### THEORIES

The underlying cause of aging remains as much a mystery as it has ever been but there are several theories that are plausible and act as aids in understanding the processes that do go on.

#### Irreversible Changes?

The ability of organs to assimilate, digest and excrete, while maintaining a specific function and structure is a characteristic of all living things. At the same time, however, within the metabolic process exists a small degree of irreversible change and a slow but steady accumulation of regressive factors. This accumulation results in the gradual loss of efficiency, often accompanied by atrophy or loss of structure. Since the alterations begin at birth, there is no period this regressive tendency is not present to some extent. In fact degeneration is most rapid early in life and slowest in later years. (121)

#### Aging of Colloids?

All living substance is now regarded as a very complicated colloidal system in which certain well defined processes of transformation take place in the course of time just as in any

gel. The basis of these processes is increasing dehydration with a transformation of finely dispersed phases into phases with coarser dispersion. (43)

#### Reticulo-endothelial Changes?

The following summary of the work by the Russian Bogomollets has been included to represent the last word in modern thinking upon the subject of changes related to age. His straight forward attempts to present his theory of the basis of these changes has been confused by popularization, prejudice, and misinterpretation.

The importance of the general and local reactivity of the body in the course of disease is well appreciated. This reactivity depends to a greater extent upon the functional condition of the connective tissue.

The connective tissue, for a long time, was regarded merely as a kind of elastic skeleton or stroma. At present, however, intensive study has revealed the influence of the connective tissue on vital processes in the body. The connective tissue, it has been found, forms a physiologic system possessing complex functional aspects. The elements of this tissue are varied. The term includes diverse active cellular elements of mesodermal origin; clasmatocytes, reticular cells, fibroblasts, adventitial cells, basophilic cells, oxyphilous plasma cells, fatty cells rich in lipase, and chromatophores. It includes further the

stellate cells of the liver, epitheloid and granulomatous giant cells, perivascular cells of various organs, reticulo-endothelial elements of the spleen and thymous, monocytes, lymphocytes and fibroblasts and osteoblasts.

The physiologic system in certain of these tissue possess the following functions;

a. Trophic function; regulation of cellular nutrition and metabolism and acts as a depot. Disorders in the trophic function result in various pathologic conditions and contribute in great measure to senescence of the body.

b. Plastic function; active participation in the healing of wounds, ulcers, fractures and in the regeneration of tissues.

c. Protective functions:

1. Participation in the reaction of the body to infection.
2. Active participation in the reaction of the body to the development of new growths.

d. Autoregulative function; autoregulation of the functions of the physiologic system of connective tissue.

e. Mechanical function: the osseous and elastic skeleton of the connective tissue. To a great extent it determines its reactivity both qualitative and quantative, therefore determining its constitution. The age and health of the body are determined

by the physiologic condition of its connective tissues. In the body as a whole, all the physiologic systems are in a state of interaction. The physiologic system of the connective tissue, reflecting various influences of the nervous and endocrine systems and other physiologic systems, itself exerts a great influence upon their conditions.

The connective cellular elements constitute the macrophagic system which has as its main activity the absorption and digestion of the absorbed particles. It is clear that they take active part in the transport and possibly in the supply of cells with protein and lipoid substances, in the metabolism of iron, and in the production of bile.

The cellular elements of the connective tissue produces fibrilles composed of various complex substances, which are either crudely or minutely dispersed, that fill the space between the capillaries and the parenchymal cells and form the so-called hemato-parenchymal barrier. It is believed that not only is this a barrier but a depot of various nutritive substances as well.

The nutrition of the adjoining parenchymal cells depend upon the condition of this barrier and the quality of the substances composing it. For example, since the cells of the microglia carry trophic functions in relation to the cells of the nervous system, it is conceivable of an accumulation around

them of constant supplies of energy and plastic substances and at the same time of a removal of the metabolic remains of nerve cells. It thus becomes apparent that a disorder in the function of the microglia can affect the functions of the nerve cells and in some cases lead to schizophrenia since it is an important pathogenetic factor of this disease.

The conditions of the hemato-parenchymal barrier formed by the cells of the connective tissue system to a large extent determines the functional condition of the highly differentiated parenchymal elements, the local and general reactivity of the body, and its health and longevity. There is no doubt that disorders of the physiologic system of the connective tissue play an important role in the origin of those pathologic conditions which prevent man from obtaining his normal longevity.

Contemporary clinical pathology tends to regard hypertension as a result of an accumulation in the body substances which, by acting through the vasomotor centers, are capable of producing a sustained elevation of blood. Many pressure cases may arise, however, not from this causation but as a result of an affection of the normal permeability of the hemato-parenchymal barrier. This disorder is usually the initial stage of presclerotic changes in the capillaries. Such changes can be broadly distributed through the whole body but may also be initially localized in some single organ, frequently the kidney. In these cases the resulting hypertension is a compensation for the increased impermea-



bility of the hemato-parenchymal system.

Later, hypertension itself causes pathologic changes. The effectiveness of anti-reticular cytotoxic serum is limited to those cases in which this change is not yet permanent.

The connective system also plays an important role in the resistance of the body to carcinogenic tumors. As soon as the macrophages penetrate the tumor tissue they destroy the carcinogenic cellular elements and the fibroblasts and macrophages form a strong line of demarcation around the cancerous foci.

The nervous system influences the cellular elements of the physiologic connective tissue. These influences undoubtedly exist and probably it is of a humeral origin. This substance is likely produced in the spleen or lymph nodes.

It was shown by investigators that injections of ARC serum increased the hemolysins and agglutinins in the blood and that this factor saved 70% of the lives of the mice infected with recurrent typhus. It was also found that in certain doses cancer transplants were stimulated and with lesser doses there was greater demarcation, fewer metastasis and even disappearance of the cancerous tissue.

Stimulating doses were shown to promote union of fractures and healing of many vascular skin lesions.

These experiments led to use of A.R.C. in treating human diseases where it was necessary to stimulate this trophic, plastic, or protective functions of the physiologic system of the connective tissue.

An A.R.C. for use in humans is produced by inoculating horses with cells of the spleen and bone marrow taken from a human cadaver.

The results from preliminary experiments are encouraging. The percentage of relapses of cancer and metastasia have been considerably decreased. Inoperable cancer has been treated relieving the patients of pain and adding several more years to their life. Infections such as scarlet fever, surgical infections, postpartum infections, acute rheumatism lung abscesses, sepsis and osteomyelitis have shown highly favorable reactions to treatment by A.R.C.

A.R.C. acts by stimulating the plastic, protective and trophic functions of the system of connective tissue. The earliest manifestations are increased permeability of the hemato-  
parenchyma barrier, dilation of the capillaries and relative lymphocytosis. The latter disappears after three to four hours and is replaced by negative monocytosis. Though monocytosis usually lasts a short time, the process of intense penetration of monocytes into the blood lasts for a number of days. The carcinolytic index is raised, complement in the blood increased

and the opsonin index elevated. The sedimentation rate is often returned to normal. (26)

## FACTORS INFLUENCING AGE

The genesis of aging may be divided into the hereditary tendencies or causes and the acquired or environmental causes. Of these the inherited qualities are probably the most important.

### Heredity

Many at birth are destined to senescence at an early age. For example: persons with congenital heart or kidney conditions, essential hypertension, glandular inadequacy such as diabetes, cretinism, stigmata of disease and early malignancies. The physical and nervous defects, and those with low resistance show the effects upon longevity of the hereditary factor. It apparently not only determines the particular disease acquired but also the age at which the disease first develops. What ever the inherited qualities are they cannot be changed. They can only be studied, guided, and protected. Naturally, one endowed at birth with a strong hereditary ability to survive, withstands environmental hardships far better than the weaker ones.

The germ plasm from which the body starts not only determines growth and development in earlier life but is also responsible for the involutinal changes that occur at different periods of life. Parts that are no longer of use undergo involution at certain times of life corresponding to the cessation of their importance as a functional part of the body as a whole.

In addition to so-called minor involutinal changes such as observed in the thymus and at menopause, we observe in later life a major involution of the organism as a whole. This major change is as inevitable as death itself. The chemical and histological changes in the body, especially in the nervous system, in the sense organs, in the endocrin glands, in the circulatory, respiratory and digestive organs are bound to occur in advanced life and are, in the large part, constitutionally determined.

In pathological or abnormal old age there is observed a premature breakdown of one or more of the organs or systems as the result of either bad inheritance or of harmful environmental influences. Arteriosclerosis, arterial hypertension, myocardial disease, vascular accidents, chronic kidney ailments, premature hypertrophy of the prostate, Parkinsonian syndrome, and senile dementia are among the commoner examples of disorders that accompany pathological aging.

#### Environment

The constitution of a person is also determined by his environment. Social, economic, and educational factors, familial habits of eating and living, occupation, the climate and composition of the soil and water, and of the food where he is born, where he grows up and where he spends his mature years, all have their effect. The heredity and environment may both contribute

factors which so modify cellular chemistry that they determine the type of involuntary change and the age at which senescence appear.

The involutinal biochemical processes which lead to senescence may be influenced by all factors which in anyway modify cellular metabolism. The heredity, health and nutrition of parents at the time of conception and of the mother during pregnancy and nursing, diseases that the individual may have during his life, the quality and quantity of the nutrition in relation to the body needs, the environment in which he lives and works, and various accidents play an important role in influencing the body metabolism and thus the onset of old age.

#### Other Factors

In the quest for longevity women have a better chance to live to a ripe old age than men. A person who increases his weight twenty five per cent above his normal actually doubles his chances of dying, and if he is twenty pounds overweight at the age of forty he has a thirty per cent greater chance of developing either diabetes or hypertension. (39)

Other disease or factors that definitely tend to shorten life are syphilis, uncontrolled diabetes, hypertension, worry, hard work, and excessive use of alcohol or tobacco.

## Effects of the Nervous System

It has been proposed by some that highly nervous systems, unable to relax from the problems of life, regardless as to whether present at birth or acquired later, are the most important cause in producing organic exhaustion ... the precursor of senescence. (169)

High nervous tension may be produced and maintained throughout life by such emotions as fear, worry, anxiety, indecision, uncertainty, or an inferiority complex. These, continued by the hour, day, week, and years plus physical exhaustion, lead to tissue starvation and impaired physiology.

Given a nervous, fearful child, slightly undernourished and emotionally misfit, with excessive physical activity and we can expect an adult with premature senescence, a victim of greying, wrinkling, high blood pressure, arteriosclerosis, with lessened cardiac, respiratory and renal reserves, whose vital capacities will succumb at an early age unless his mode of life can be changed.

It is reasonable to consider that increased nervous tension over a long period of time will produce an overproduction of pressor substance which in turn stimulates the cells and organs to excess, and causes tissue exhaustion, infiltration of fat and fibrous tissue with a loss of elasticity. Whether these stimuli

are contained within as from the pituitary and thyroid glands, or there is an extremely sensitive nervous system, or there are external stimuli, such as financial, sex, personality or family worries, the end results are the same. It is little wonder that there developed the old saying, "Whom the Gods would destroy, they first make mad".

#### Effects of Infection

The factor of infectious diseases during the life time of an individual should be emphasized in connection with environmental factors. The survivors of acute and chronic disease are doomed to carry throughout life scars of their illness which must result in some degree of physiological and or anatomical change causing a weakness or deficiency in the body organization.

An example of the foregoing statement is the person who, following a severe streptococcus infection, pneumonia, typhoid or similar disease, rapidly puts on weight and develops hypothyroidism. The factors producing the conditions are not understood, but probably there was a depression of function of glandular secretion which allowed the let down.

But far more important is that vast number of persons who harbor, in various parts of the body, foci of long standing infections. The absorption of toxic products of bacterial infections over forty years makes definite changes in the vital tissues. Once



these effects or changes have occurred, there is no method that can undo these damages except by slowing up and learning how to live within the body's limits.

#### WHAT IS "NORMAL" AGING

In describing the changes that occur with old age there immediately arises a problem of what shall be considered as the criteria for judging what is pathological and what is non-pathological. There is no change in the aging system that hasn't happened under different circumstances. In other words none of the structural changes observed in aging are peculiar to that state. Normal changes have never been seen. There are the many alterations which add their quota of disintegrating effect. First there are the biochemical changes which precede the "primary" tissue changes. These are inherent in the chemical composition of the human structural make up. The structural changes are repetitions of alterations that have operated in the phylogenetic and ontogenetic past. Lastly there are changes the result of "wear and tear" due to merely living. But how much is "wear and tear" and how much is pathology?

There are certain changes inherent in the structure of the human organism that predispose to senile changes. This is especially evident in the vascular system; For instance the size and shape of the coronary vessels and their differences between the sexes may cause a loss of function of the heart and a loss of

nutrition to the body. This, no matter how small, hastens senile changes.

The essential anatomical change in old age is that of atrophy of the parenchymal cells and an increase in the interstitial substance. The environment of these cells is the most logical place to look for the cause of such changes. This is controlled to a great extent by the blood supply. The most direct explanation of the vascular factor as a cause of atrophy is that an arterial lesion lessens the supply of nutriment to the cell so that it can no longer maintain itself. Many other factors are certainly involved, as lack of oxygen, without which the energy transformations of the cell become impossible. A failure in the removal of toxic metabolites is another result of circulatory insufficiency that may cause atrophy.

Normal growth, cellular differentiation, and even the involutions of embryonal and early life are in large part subject to hormonal control and from this fact alone it might be concluded that the same mechanisms would operate in the terminal and equally normal involution of senescence. The most striking examples of this control are seen in the waxing and relatively early wane in both function and structural differentiation in the female sexual cycle.

The question of what is normal aging must remain unanswered but act as a goad in further experimentation. Until such a time

as it is answered, certain assumptions and hypothesis must be regarded as a basis on which to start.

## STATISTICS

At the beginning of the century in the United States, the chances of survival to age 65 were slightly more than 40 per cent. Twenty years later, newborn infants already had about an 80 per cent chance to survive to this age. By 1938, chances of survival to age 65 had increased to 60 per cent. If this trend continues by 1980 a child should have a 75 per cent chance to see his 65th birthday. ( 81) (See fig. 1)

For many years the average age of the American population has been increasing, and there is every indication that this trend will continue in the future. The combination of high birth rates and heavy immigration in the past made us a very youthful people. The population was characterized by a large proportion of children and young adults, with a corresponding small proportion of older persons. Since the beginning of the century, however, our birth rate has dropped more than one third. Immigration, likewise, has been cut drastically, and has now virtually ceased. ( 104) (See figs. 3 & 10)

The net result of these changes has been to shift the weight of our population structure from youth toward the older ages. If 65 is taken as the threshold of old age, there were at the beginning of the century 3,080,000 persons, or 41 per cent of the total, in this bracket. Today, however, 9,000,000 persons, or 6.8 per cent of the total, are in this category. In addition another 22

per cent fall in the 45-65 age group. If the present trends continue, the proportion of persons 65 years of age or over will be more than 14 per cent by 1980, and their total number will be about 22,000,000. ( 127 ) (See fig. 4)

This shift in population combined with a tremendous decrease in infant mortality and improved treatment of infectious disease has resulted in an increase in the importance of those diseases characteristic of old age.

Under mortality conditions prevailing in 1938, half of the babies would eventually succumb to degenerative disease of the circulation system and the kidneys. The chances of dying from this group of diseases rises steadily through life so that, at age 60, the chances exceed 60 per cent. ( 70 )

#### Reliability of Present Statistics

Before going further in a discussion of disease in old age a word of caution is needed. Present official mortality and morbidity statistics for old age are inadequate for scientific and practical purposes. Reliable data can be collected only upon autopsy reports. An experimental survey of autopsy reports shows that cardiovascular diseases are less important than are the infectious diseases in primary causes of death and that correct clinical diagnosis was made in 46 per cent of the cases. ( 122 ) Further discussion in this paper must be viewed in this light.

The greater part of the gains in the expectation of life at birth may be attributed to the control of infant mortality, to the practical elimination of certain diseases of childhood, and to the curtailment of conditions once considered typical of adolescence and early maturity. Altogether our progress with the diseases of late maturity and old age has not been of any consequence. We observe savings only at the ages where lives were heretofore unnecessarily shortened by the impact of bacterial diseases. (See fig. 2)

## CHANGE IN CHARACTERISTIC DISEASES

(See Fig. 5)

The diseases of the senescent period differ widely from those common in youth. In youth disease is usually due to some exogenous injury or infection. The concept of specific cause, inflated by the earlier discoveries of bacteriology, has done much to retard medical thinking as it has simultaneously advanced knowledge of infective disorders. Cause is never singular. In geriatric medicine, perhaps more than anywhere else, the physician must be constantly conscious of the multiplicity of etiological factors involved in the degenerative disorders. Predisposing influences, provoking factors, and perpetuation factors all play a part in any disease process. This fact is especially true and prominent in the degenerative diseases of old age.

In the past conspicuous shifts in population structure have arisen as the result of destructive forces such as wars, famines, and pestilence, but always the changes were in the direction of a relative increase in youth. The less fit failed to survive. Today we are faced with the enhanced survival of less vigorous youths because of the protection of medical science and the lessened rigors of modern civilization. The apparent boon of longevity may become a curse. Longevity without health is more than an individual or personal tragedy. In the aggregate it becomes a dangerous economic burden upon the nation and potentially a political football.

The social burden of chronic physical and mental illness among senescent is already immense. Nearly five million aged are dependent upon relatives or the government. (94) However, longevity with health, prolonged productivity and usefulness can be made into an incalculably valuable asset, for there exists in the elderly an immense largely unutilized and little appreciated reservoir of accomplishment. Offsetting chronic illness with long and progressive disability and the failure of society to employ many functionally capable among the elderly are the potential assets of wisdom, judgment, accumulated skills and the desire to serve. These are available if health is maintained. This heavy responsibility rests squarely upon the medical profession, though the majority of its members are unaware of it.

The older persons have problems quite as specific for them as are the problems of infancy and adolescence. Since the reaction of the older body to disease is different than at other stages of life, the symptoms, which are an expression of this reaction, must be different. If the older body itself is different, the signs, both normal and in disease states, are changed requiring a modification of the rules of diagnosis. The changed chemistry of the older body means that accepted laboratory normals must be adjusted. Treatment must fit this changed and different reacting body.

#### Heart Disease

Measured in terms of sheer number of persons affected, heart



disease is our most important medical problem. There are today about four million people in the United States who have some form of heart disease, and the number of cases is steadily increasing. Heart disease ranks first in the list of causes of death. It is not surprising, therefore, that there is a widespread impression that the situation with regard to the disease is critical, that the outlook for the patient with heart disease is poor, and that the conditions of modern life are largely to blame. Actually the situation is much better than appears on the surface, and these current gloomy beliefs about the disease are not warranted by the facts. (165)

It is true that the crude death rate from heart disease has shown a steady increase over the years. (See fig. 8) It is also true that this is the most important problem in medicine today. The major part of this increase, however, simply reflects the rapidly increasing proportion of older persons in our population. The apparent increase in heart disease as shown in the accompanying figure is due in a large part to a change in diagnosis of the primary cause of death. Many cases formerly called chronic nephritis or cerebral hemorrhage are now recognized to have heart disease as a primary factor. The actual increase in rate is only 4 per cent above that for the year 1928. (164)

When all these influences are taken into account, it is doubtful whether there has been any real increase whatever in heart

disease, beyond what is expected from the increase in the number of older persons in our population. (See fig. 9) Indeed; it is likely that there has been an actual decrease in the mortality from heart disease at every age roughly parallel to the decline in the death rate for the aggregate of the cardiovascular-renal disease as shown in the chart.

When the factor of age is figured and the change in diagnosis is taken into consideration it is seen that the cardiovascular renal mortality rate is on the decline. This is well born out by the accompanying chart which shows for the older age group, 65-74 years of age, a decline of 21.9 per cent over the figures in the 1911-1915 group.

The problem of cancer is likewise of growing importance. (See fig. 7) The chances of eventually dying from cancer are now nearly one in nine, for white males and one in seven for white females. Even if the death rates at the various age periods remain at the present levels, there will nevertheless be growing numbers of cancer patients, because of the increasing proportion of older persons in the population. In fact, their number may nearly double within the next 25 years. (103)

## CHRONIC DISEASES

Senescence is an important factor in the causation of many chronic disease. These increase sharply in incidence with aging. The beginnings of chronic and progressive heart disease, arteriosclerosis, arthritis, diabetes mellitus, and the like occur with the beginnings of the major involutions of senescence. Characteristic of these degenerative disorders is their slow but inevitable progression, insidious onsets, and long periods of disability. The toll of disability in the fifth and sixth decades is a graver economic problem than deaths from acute infections. Brutal truth forces one to confess that a man in his forties or fifties disabled for many years by arthritis or coronary or cerebral arteriosclerosis is a greater social loss and heavier burden upon his family and society than a man quickly dead. Mortality tables alone are most misleading indexes of the social significance of disease.

A study of the accompanying chart (see fig. 6) will show the rapid rise in invalidism as age progresses. It also shows that where the greatest incidence of chronic disease is between 35 and 54, the highest percentage of invalids are above the age of 65. (92) Comparison of these charts with mortality charts and with the percentage of population composition will show how very important the problem is. Obviously a huge fraction of the disability in the senescent group arises from the progressive disorders so common twenty years earlier. This data dramatically emphasizes the concept that the sources of geriatric disability starts in the thirties and forties.

## NEGLECT!

The story of the rise of Geriatrics and Gerontology to a recognized place in medicine has been slow and due to lack of interest upon the part of the medical profession as a whole. In fact, if it was not because of factors other than the curiosity and benevolence of the profession this important development would still be in the throes of birth.

This is not to detract from the honor due to a small band of persistent men who stoutly maintain that the physiology and pathology, as well as the therapeutics, of the aged need as much if not more investigation and consideration than the study of pediatrics. The uniqueness of the elderly individual has been recognized for centuries, but mainly by philosophers and poets.

The reasons for the present general interest and the great increase in research are: the increase in population and in the number and percentage of elderly patients, pressure from pseudo-medical groups, pressure from political movements, and perfection of new technics to aid investigation in disease peculiar to old age.

But despite extensive publication, thirty-nine medical treatises published prior to 1800 and hundreds later (182), this field of medicine has never attracted the interest of the medical profession. Through the prefaces of many of the books on the subject runs a common theme of regret and wonder that a subject of

such importance and fruitfulness should be so neglected; a hope is expressed that the defects in experience and practice, the inadequacy of diagnosis and therapy will be, in a measure, alleviated by their writing, a desire to stimulate medical enthusiasm to further knowledge and to improve medical performance. Unhappily, the theme sounded in vain. Too often, even now, the senile patient is unwelcome in institutions, since he is considered neither a worthy object for intensive study nor is it thought rewarding to trouble about his complaints at the obvious end of life. In private practice, the physician screened himself behind a diplomatic obscure vocabulary of comforting misapplied generalities or he spoke of marasmus, senile decay or breakdown, arteriosclerosis, weak heart and the like. Quite clearly, behind this negativism consciously or unconsciously lurked his own insufficiency and lack of training in this segment of his practice.

Old age is a condition, a biological situation, and calls for as fresh and as special approach as does infancy. When we realize that a disease can be understood only in terms of its host, it becomes apparent that the disturbances common to all age groups change in characteristics in each group. It will be shown how the more prevalent diseases that have to be conquered lie in the field of the diseases of degeneration seen especially in the older age groups.

The man of forty to sixty-five lives little longer than he

did fifty years ago, and there is evidence that he is no healthier. (104) In other words, the present program of preventitive medicine is enabling a great many people to live to sixty-five years of age but is not promising them anything when they do. It seems only fair that if they are to live as long as this, life ought to be made happier and healthier for them than it is now. Old age has been what one might call tolerated by the medical profession and by the public in general. No one is enthusiastic about old age, certainly elderly people themselves are not particularly enthusiastic about it. In general society has taken a negative and an entirely defeatist attitude toward old age.

As long as this negative, defeatist attitude continues, nothing is going to happen.

One can easily account for the public neglect of the aged. Human sympathy is universal in scope, but not in its' application. Instinctively or subconsciously economic values, social relation, the esthetic sense, and other factors influence the directions in which the sympathy is applied. The aged become economically worthless and must remain so, while the child has a prospective and ever increasing economic value. Optimistic philosophers of all ages, from Cicero to Jean Finot, have described old age as being beautiful, but no philosopher has ever declared that the aged themselves are beautiful. From the purely esthetic standpoint, the aged are disagreeable, often repulsive, and fail to

arouse sympathy as long as they are not altogether helpless or in distress; from them may emanate disagreeable odors, offensive action; and they may justly be accused of peevishness, selfishness, willfulness, and suspicion. One finds consequently, that here is a universal tendency to shift responsibility and the care of the aged upon others, usually the community.

#### Medical Attitude

A large proportion of the medical profession is still ignorant of the peculiarities of the senescent organism. Physicians still treat aged persons as they do younger ones, perhaps diminishing dosage of drugs, without system or reason. When the patient dies under such treatment, the physician eases his conscience and satisfies the family by ascribing the death to old age. But old age is not a disease, and ordinarily it is not a cause of death.

Death from old age is conceivable. Sometimes there will be a gradual degeneration of all the organs and tissues and their functions will become more and more impaired, the cerebral activity and nervous and muscular activity will diminish gradually to the point of complete extinction. This is, however, exceedingly rare. It is far rarer than vital statistics indicate, since many deaths ascribed to old age are due to disease which the physician did not recognize, or in which, finding symptoms and signs he could not interpret, he resorted to the diagnostic placebo, "old age".

Medical neglect of the aged is as general as is public neglect. It is obvious that the aged will never appeal to physicians as children do because their chances of recovery are smaller, they are often temperamental and, their families are not always keenly interested in the fate of aged relatives. Nevertheless, minimum of care and attention will often have startling results.

The chief obstacle to an enlightened attitude is the power of the "old age taboo". Some young people fear the onset of old age and feel uncomfortable in the presence of the aged. Osler, in his younger days, declared that men over sixty were better dead. They would rather remain ignorant of the adjustments that they themselves will have to make in the future. Physicians are for the most part silent, despite the fact that many are well advanced in years. In 1930 10.2 per cent of the physicians were 65 years or over and this figure has increased since then. (182)



## HISTORY OF GERIATRIC MEDICINE

The interest in old age has been manifest almost as long as there has been literature. The problems of the aged have caused concern as far back as the Babalonian empire and the days of the Ming dynasty. Hippocrates, Galen and Socrates concerned themselves with the symptoms of the aged. In 44 B. C. Cicero wrote *De Senectus*, Roger Bacon wrote one of the first English volumes, but it remained for Sir John Floyer in 1724 to start the new subject of Geriatrics along its' way by publishing *Medicina Gerocomica*.

In the year of 1914 Nascher coined the term geriatrics to indicate the study of the senile organism. He has been called the father of Geriatrics and the founder of the science but he was not alone more did he suffer from lack of company. Day in 1849, Maclochlan in 1863, Humphry in 1889, Minst in 1906, and Saundy in 1913 wrote upon the subject in great detail. The French: Gillette in 1851, Chorcot's 1881, and German: Fishcer, in 1766, Geist in 1869, Methenheimer in 1863, and Muller in 1863, contributed to the general knowledge on the subject. (148)

### Definition

Geriatric medicine is that branch of medical science which treats of the elderly in all their physiological and pathological relations. (177)

## Need

Each period in life; infancy, adolescence, maturity, and involution presents a special problem for the physician. The study of infancy and its' diseases has given rise to the speciality of pediatrics, but a similar study of the involut-ional period has been neglected. From what has been said and from what evidence will become increasingly evident that there should be physicians who will make an intensive study of the needs and care required in this period. The diseases, the modification of their symptoms, their course when they occur in the aged should have special study and investigation. The geriatrician is the specialist who fills the bill.

The problems of geriatrics are not confined to those patients actually senile, for infirmities and disorders of later years arise insidiously far earlier than their clinical manifestations. Aging, as a process or series of processes, is continuous. It starts at conception and terminates only with death. To arrest the process would mean destroying life. Evolution, or growth, and involution, or atrophy occur simultaneously throughout the life span. These two antagonistic phenomena of aging, occurring asymmetrically at variable rates in different individuals and in different tissues, structures, and systems of the same individual at different times, determine the rate and character of senescence.

## Scope

It is necessary, in view of the nature of the problem, to consider the scope of the geriatrics as beginning at about the fortieth year of life. This does not imply that all people over forty are decrepit and infirm, but from this age onward occurs the insidious accumulation of involuntional phenomena which later results in the entity of senility. The so called degenerative disorders become conspicuously more frequent at this point in the life span. The menace of these chronic and progressive diseases is greatest from 40 to 60. If health can be maintained during these two critical decades, the likelihood of long disability and uselessness from chronic illness will be reduced immensely. Obviously far more can be accomplished for the aging than for the aged. Therefore geriatrics must include care and guidance during the period of senescence. An attitude of prophylaxis and preparation during maturity is essential.

To many the objectives of geriatrics are limited to the prolongation of life and the relief of suffering. This is a serious error. The normal life span has its' limits. There is no desire among medical men to arrest aging. Rejuvenation and the fountain of youth are a myth. Proper medical guidance, however, can prevent, retard, or even control disease so that disability may be postponed until true senility causes infirmity and thus adds to health, vigor and usefulness in later life.

If geriatrics were to limit its activities and its thinking to merely palliative measures for the infirm senile, it would appeal to very few minds. If, on the other hand, geriatrics takes the viewpoint that more may be accomplished for the aging than for the aged and emphasizes preventative measures during critical years where senescence begins, it may do much to diminish or retard infirmities of senility. Such a goal is dominant in the minds of those involved in this work.

There are many who resent the implication that geriatrics should attempt to prolong life. Many among the senile, and even more among the young, feel that most of the older members of our population are living too long as it is, that the vastly increasing numbers of the old are becoming nothing but a burden upon the community. In some ways they are justified in their claim.

Nearly 75 per cent of people over 65 years are wholly or partially dependent ( 50 ) and we have already noted what a large percentage are chronically ill. The aged are not up to par with the young in many fields of activity. However, these scoured egotists assume that the primary objective of geriatrics is to prolong life. Their assumption is erroneous. Equally in error are those who would assume that the primary objective of geriatrics was only to relieve suffering in the elderly. Such would be a most negative and defeatist approach. It is for science not only to add years to life, but more important, to add life to the years.

# EXPECTATION OF LIFE

1879 - 1889 ; 1911 - 1912 ; AND 1941 COMPARED

103

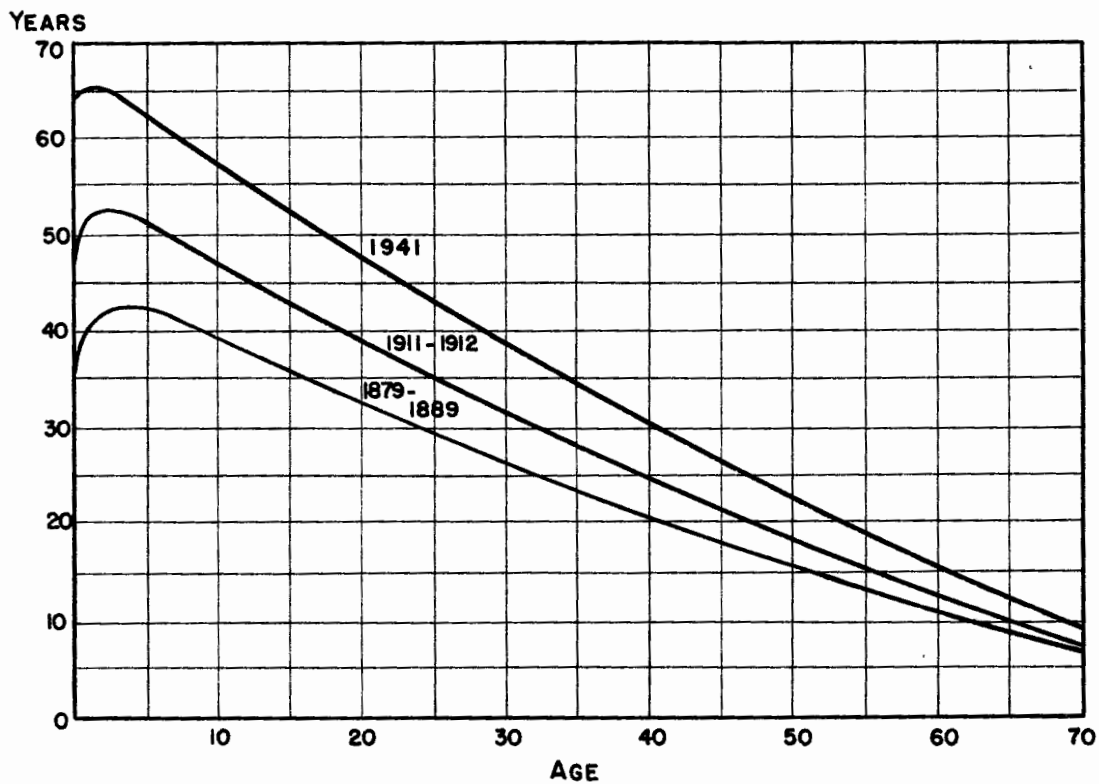


Fig. 1

NUMBER OF DEATHS IN THE WHITE POPULATION OF THE UNITED STATES DURING 1942  
FROM SELECTED CAUSES, COMPARED WITH THE NUMBERS EXPECTED  
IN THAT YEAR ON THE BASIS OF THE MORTALITY RATES  
BY SEX AND AGE PREVAILING IN 1900

103

CAUSE OF DEATH	DEATHS IN 1942		LIVES SAVED IN 1942 BY IMPROVEMENT IN MORTALITY SINCE 1900	LIVES LOST IN 1942 BY INCREASE IN RECORDED MORTALITY SINCE 1900
	ACTUAL	EXPECTED ON BASIS OF MORTALITY IN 1900		
ALL CAUSES -----	1,209,944	2,281,621	1,071,677	
INFLUENZA & PNEUMONIA -----	61,051	268,916	207,865	
TUBERCULOSIS, ALL FORMS -----	41,306	243,367	202,061	
DIARRHEA & ENTERITIS -----	11,815	160,471	148,656	
PRINCIPAL COMMUNICABLE DISEASES OF CHILDHOOD -----	4,349	76,363	72,014	
MEASLES -----	1,029	13,792	12,763	
SCARLET FEVER -----	418	9,590	9,172	
WHOOPING COUGH -----	1,800	12,397	10,597	
DIPHTHERIA -----	1,102	40,584	39,482	
NEPHRITIS -----	81,475	133,013	51,538	
TYPHOID & PARATYPHOID FEVER -----	504	37,274	36,770	
CEREBRAL HEMORRHAGE & SOFTENING -----	92,483	123,042	30,559	
PUERPERAL STATE -----	5,515	15,423	9,908	
EXTERNAL CAUSES -----	89,054	92,043	2,989	
ORGANIC HEART DISEASE -----	241,732	181,308		60,424
CANCER -----	152,357	101,266		51,091
DIABETES -----	31,624	16,419		15,205
ALL OTHER CAUSES -----	396,679	832,716	436,037	

Fig. 2

CHANCES PER 1000 OF EVENTUALLY DYING  
FROM SPECIFIED DISEASES OR CONDITIONS  
UNITED STATES 1938

102

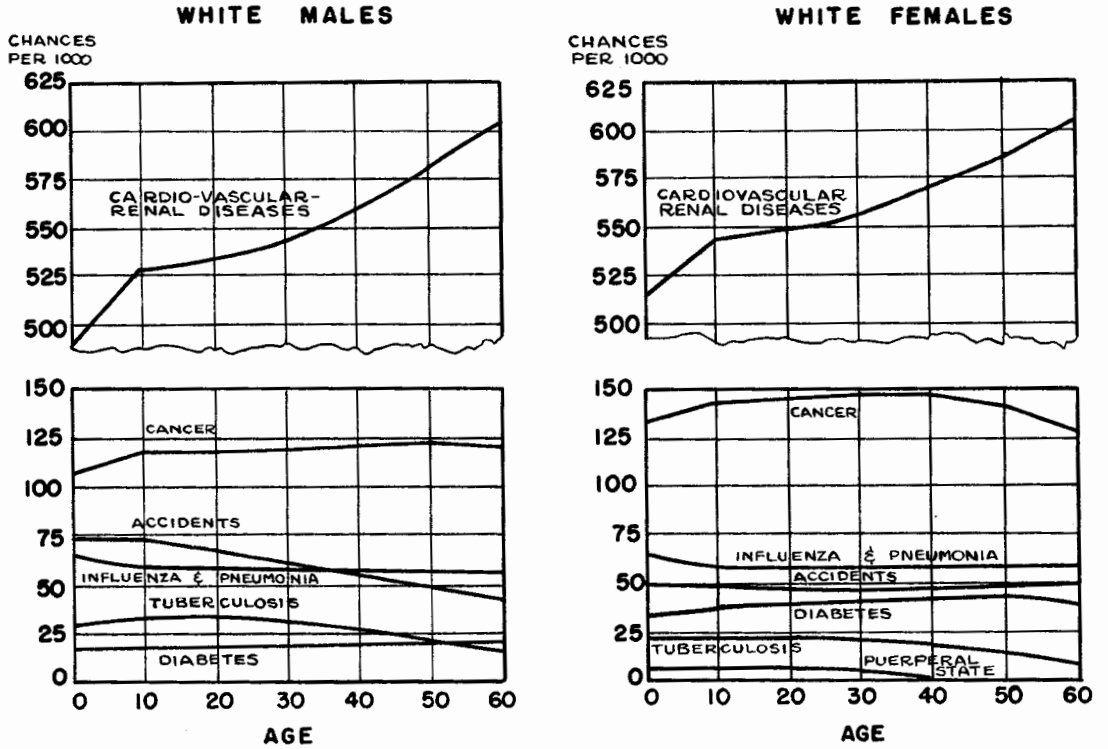


Fig. 3

PERCENT DISTRIBUTION  
OF  
TOTAL POPULATION BY AGE  
UNITED STATES 1850 TO 1980

50

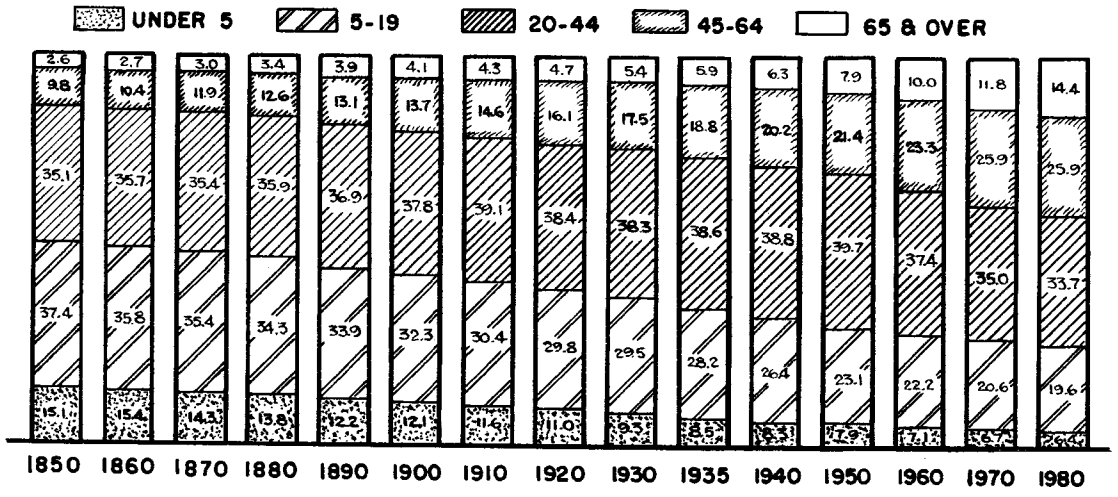


Fig. 4



**RANK & DEATH RATES FROM TEN LEADING CAUSES  
IN 1938 COMPARED WITH THE SAME CAUSES 1900  
UNITED STATES**

50

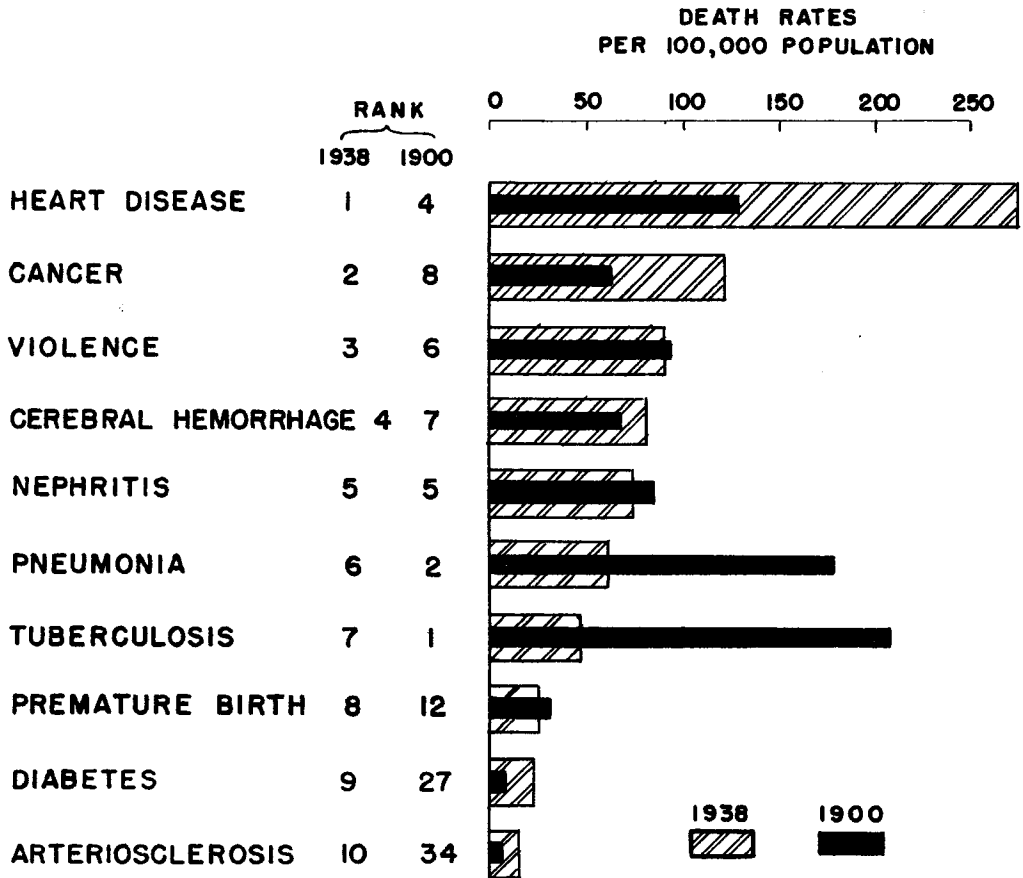


Fig. 5

INVALIDS PER 1,000 POPULATION ACCORDING TO AGE  
NATIONAL HEALTH SURVEY, 1935-1936

92

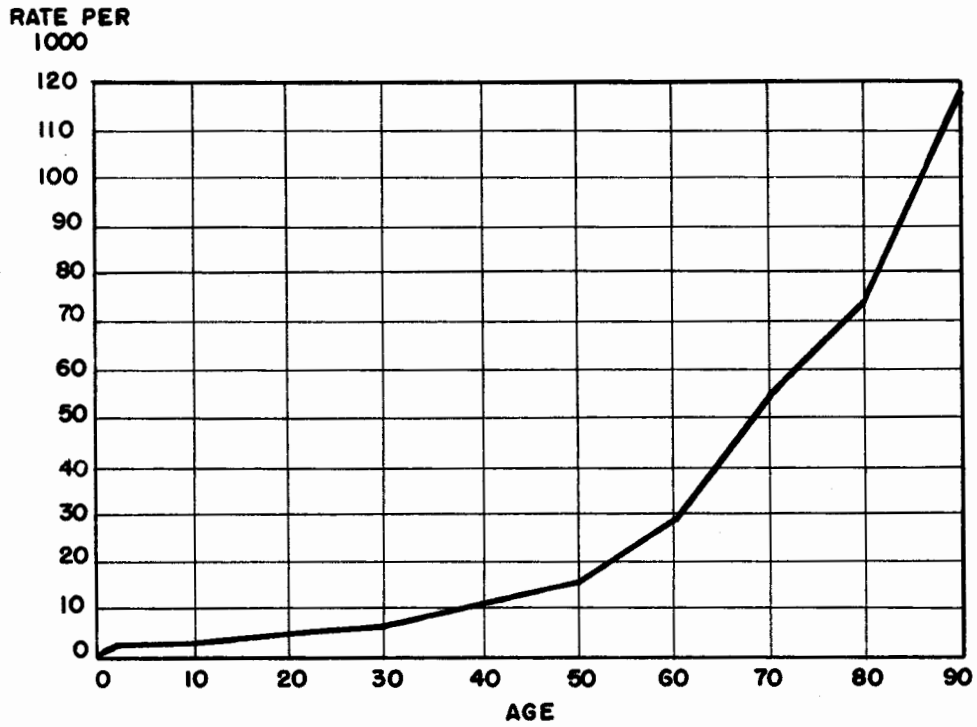


Fig. 6

**MORTALITY FROM CANCER BY SITE**  
**STANDARDIZED ANNUAL DEATH RATES PER 100,000**  
**WHITE PERSONS BY SEX AGES 1 TO 74 - 1911 TO 1940**  
 104

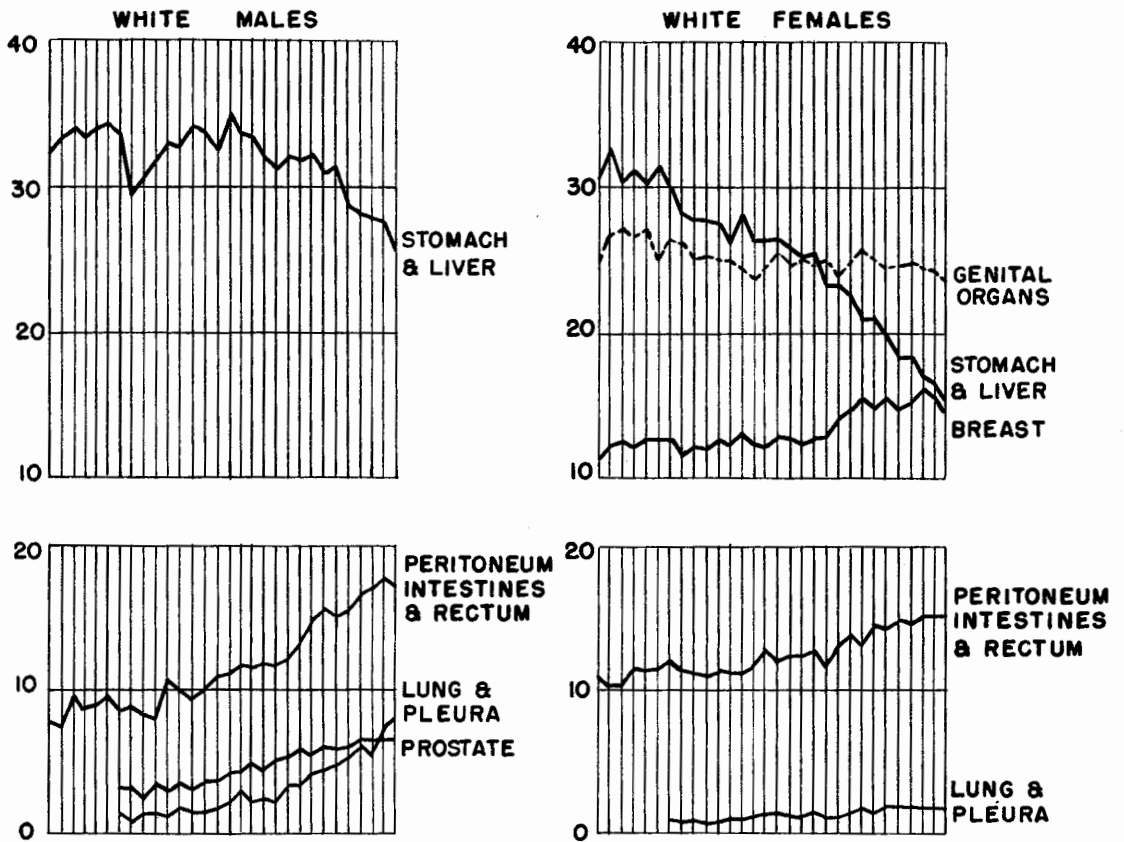


Fig. 7

AGE-ADJUSTED DEATH RATES PER 100,000 FROM TOTAL  
& SEPARATE CHRONIC CARDIOVASCULAR-RENAL DISEASES

1911-1945 AGES 1-74

104

DEATH RATES  
PER 100,000

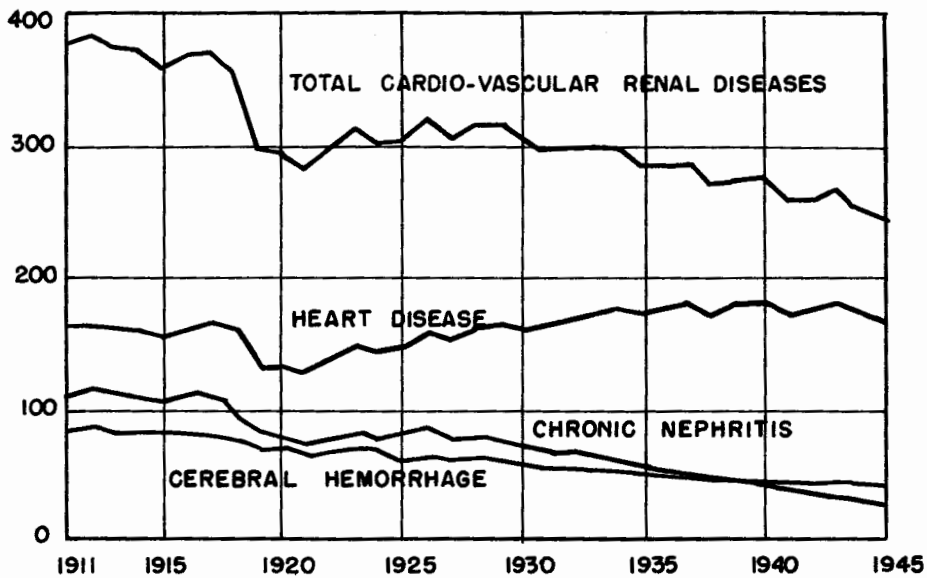


Fig. 8

**PRINCIPAL CARDIOVASCULAR-RENAL DISEASES  
ANNUAL DEATH RATES FOR WHITE PERSONS BY SEX AND AGE**

104

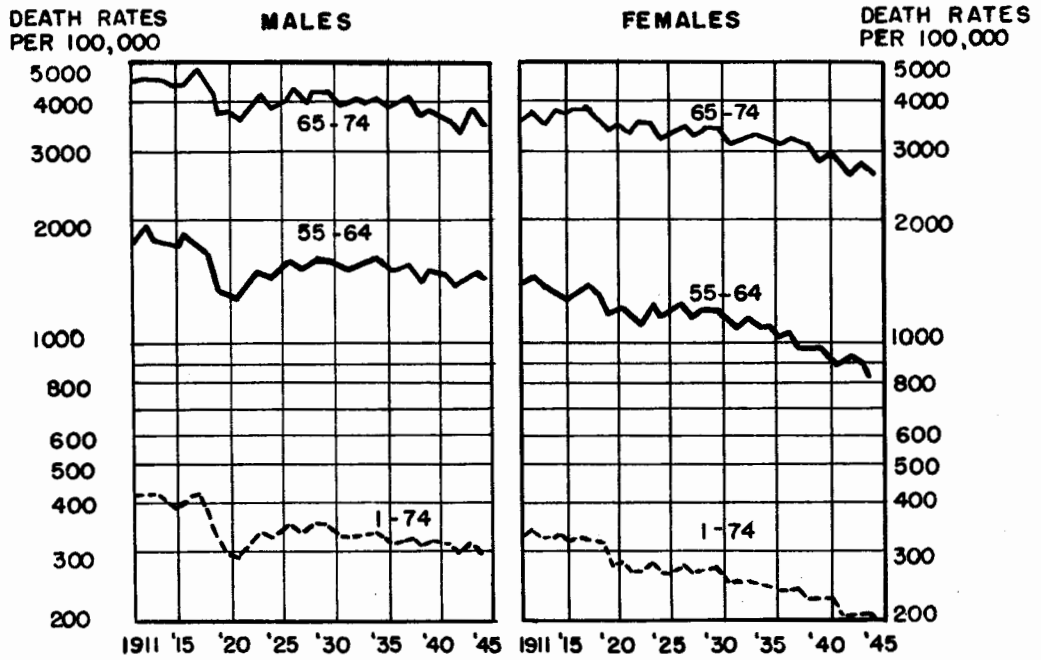


Fig. 9

# BIRTH RATES & DEATH RATES IN THE UNITED STATES

BIRTH RATES: OBSERVED FROM 1915 TO 1939  
ESTIMATED FROM 1940 TO 2000

DEATH RATES: OBSERVED FROM 1900 TO 1939  
ESTIMATED FROM 1940 TO 2000

RATE  
PER 1000

50

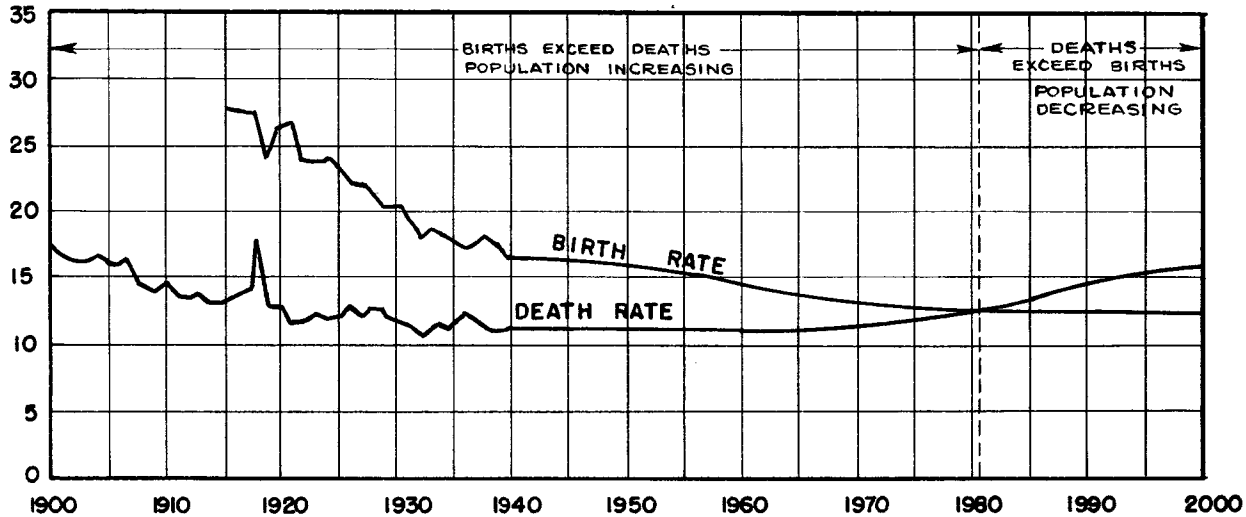
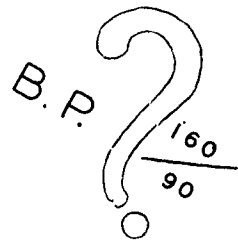
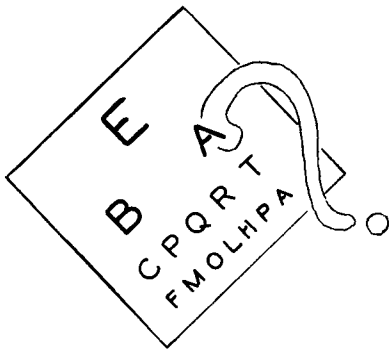
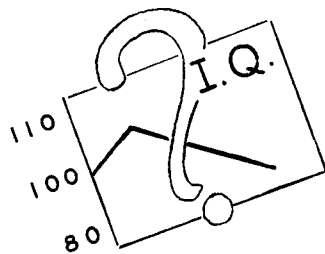
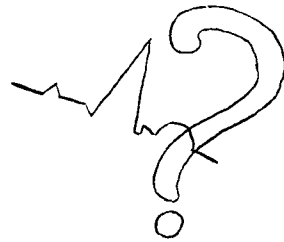


Fig. 10



# THE PROBLEM OF "NORMALITY"



# VASCULAR SYSTEM



The various changes attending the process of aging include processes which manifest themselves in the vascular system. In fact, the majority of complaints offered by individuals over 65 years of age are referable to the disorders of the cardiovascular system, the nervous system, the digestive tract, and the locomotor apparatus.

It has been said that "man is as old as his arteries". No doubt there is some truth in it, for there is hardly another system that shows the transition from the ascending to the descending period of life as plainly as the vascular system.

In discussing the "normal" Cardiovascular system there immediately appears a legion of contradictatory information. It will be shown the effects of arteriosclerosis must be omitted and only the heart that is relatively unaffected by disease considered. Methods of evaluation and collection of figures vary greatly. As far as possible all doubtful and incomplete data has been discarded in the following discussion and there is presented only that information which will help to give a clearer picture of the aging system.

It has been shown that a child at its' birth will have 45 chances in 100 of dying because of cardiovascular disease; a man at the age of 30 will have 53 chances out of 100; and, if a man reaches the age of 60 years, his chances of dying from this disease are 60 out of 100. Vascular disease in a hypertensive

individual will produce cardiac failure due to coronary occlusion in 60 percent of the cases. ( 60 )

However, heart disease maintains as high a place on the list of killers in old age as it does because of the comparative ease in diagnosis. The modern instruments of cardiology make the diagnosis much easier than before. The fact that heart disease, of one form or another, is least open to criticism in the absence of a definite diagnosis influences to some extent the available statistical information. Figures compiled from autopsy reports by Mueller-Dehman (122) and by Aschoff ( 7 ) show a mortality rate of less than 41 percent as compared with official mortality statistics which show a rate of 60 percent from cardiovascular disease.

HEART  
ANATOMICAL CHANGES

Weight

The absolute weight of the heart diminishes with age although the ration of heart weight to bodgy weight increases. ( 18) The formula of age plus three times the body weight plus 100 equals the heat weight has been used by some to indicate the normal weight of the heart in the senile patient. If the weight is 77 grams or more then the heart is considered to be hypertrophied.

Position

The position of the heart is changed somewhat. It is more lateral and there is a shifting upwerds and to the left of the left ventricle. ( 24) Upon x-ray examination there is noted an increase in the size of the left ventricle and a widening of the aortic arch.

Gross Changes

The pericardium tends to become opaque, particularly over the surface of the base of the right ventricle. The apicle portion of the left ventricle decreases in size. Subpericardical fat tends to increase along the grooves of the coronary vessels, especially in the auriculo-ventricular groove at the base of the right auricle. The valves become more rigid because of an increase in fibrous tissue and from the deposition of fats and calcium. ( 189)

There may also be calcification of the annulus fibrosa. The sinuses of Valsalva become deeper. Even without inflammatory attacks the papillary muscles tend to be thick. The portion of the left ventricle at the apex atrophies so that the infra-papillary space becomes smaller. (25)

### Microscopic

The striated muscle fibers undergo important changes although they are not reduced in number. (18) The individual cells become modified, coalesce and form a syncytium. The striation of the muscle gradually disappears, especially around the nuclei. The size of the individual fibers decreases in later years. Pigment, lipochrome in nature, begins to be laid down at the poles of the nuclei in the earlier decades, increasing continuously in amount afterward. Older hearts, exhibits this appearance, to which the term brown atrophy is applied, as a result of decrease in the concentration of water.

The microscopic changes of involution are fewer mitoses, atrophy of cells, condensation of the chromatin of the nucleus, nuclear pyknosis, vacuolation and karyolysis, and lipoid deposition in the cytoplasm may be observed. These changes are most marked where the blood supply is insufficient.

An increase in elastic tissue fibers mainly in the auricles have been observed. The amount of elastic tissue varies so much

with age and is so characteristic that, in normal hearts, (198) it may be possible to arrive at an estimate of age in terms of the amount present.

### Physiological Changes

#### E.K.G.

Certain changes are known to occur with sufficient frequency in the aged in the absence of other manifestations of cardiac disease to be regarded as normal. (60)

The most frequent of these changes in the electrocardiogram of the aged is a shift to the left. This has been observed in well over 50 percent of older patients. As associated but less frequent finding is a prominent q s wave in Lead 3, and an inverted T3. These changes are probably derived from the more transverse position of the heart secondary to the flattening of the diaphragm.

Extra systoles occur in about a third of elderly patients and, as in younger groups, they constitute the usual electrocardio-graphic arrhythmia's. Those of ventricular origin are about twice as common in occurrence as those of auricular origin. They assume pathological significance when frequent or they show coupling or other rhythmic changes occur or multiple foci of origin develop. (18)

In one study of patients with clinically normal cardiovascular systems over 70 years of age there was found: (52)

Axis deviation

Left ----- 62%  
Right ----- 0  
Normal ----- 38%

Voltage of major deflections

Abnormally low ---- 37%  
Normal ----- 63%  
Bradycardia ----- 13%  
Tachycardia ----- 1%  
Ectopic beats  
    Auricular ---- 5%  
    Ventricular -- 15%

A. V. conduction deflection

Left bundle branch block 6%  
Right bundle branch block 0  
Intermediate forms 6%  
"3" Type 8%  
Arboration 4%

Inversion or abnormal depression of T waves

Lead I 19%  
Lead I II 2  
Lead I II III %  
Lead IV 5%

Presence of Q wave in IV 18%

## Heart Sounds

Heart sounds are often normally decreased due to the general decrease in myocardial tone. There are systolic murmurs that are normal. The most common is the pulmonary systolic murmur which varies greatly with different phases of respiration, being maximal in full expiration. A systolic murmur at the aortic area may be explained by the change in the position of the aorta when the heart moves more horizontally in later life. This is a partial explanation for the pulmonic murmur. Cardio-respiratory murmurs heard best over the apex vary with the respiratory cycle and is blamed on the displacement of air in the lungs.

## ARTERIOSCLEROSIS

Arteriosclerosis is defined as a chronic disturbance of the vessels which is manifest by deposits of the most varied kinds in the vascular walls and which becomes irreversable on reaching its' climax in vessels impaired by changes attending the process of aging with resulting deformation of the lumen and brittleness of the vascular walls.

No single sign has been connected with old age more than arteriosclerosis. It has been found in 95 percent of males and 85 percent in females past the age of 70 years in the United States. The problem arises; then; as to whether arterioscler-osis and all its' effects are normal in old age or should be called a disease process. (60)

When this question was presented to Aschoff, his reply was indeed emphatic. "Are we justified in counting arteriosclerosis among the changes attending the process of aging? Does the description of the changes with age fully cover the term arterio-sclerosis? Most certainly it does not! The mere aging of the vessels results in distension and dilatation and consequently in increasing tortuosity. These conditions may be described as "infirmities" of old age. But mere aging never results in deformation of the vascular walls." (6)

Other writers however just as avidly cry that it is a



normal physiological process. (182) What then shall we use to judge what is normal or pathological. This is a most difficult task and impossible if an iron clad rule is sought. There are too many variables in the human system to include them under one rigid rule.

We cannot declare the average as normal, nor can we say majority makes it normal. It is obvious under this rule that if 95 percent of people have colds then the cold is normal. Obviously this is not true. The safest method of determining what this elusive word denotes is by using these criteria: the process must be orderly and progressive, it must fall within a narrow statistical range, it must be predictable, and it must run a course which can be modified but never reversed. Disease has a cause which has the effect of changing the expected course. The effects of disease are not in accord with other changes in the body.

If the process were orderly and progressive, it should be less common than it is before the age of 50 and the percentage should rise more rapidly after 50 years of age. Furthermore, lesions due to senility would be more likely to be diffuse instead of patchy as the lesions usually are in arteriosclerosis even in the same vessels. The changes which are definitely traceable to aging are of a diffuse nature.

Arteriosclerosis is not orderly. In scarctation of the

of the aorta there is marked sclerosis proximal to the lesion and little or no sclerosis distal to the stenosis. Sclerosis of the pulmonary arteries is far less common than in sclerosis of the systemic arteries and even when it is present there is almost invariably some process, such as mitral stenosis or extensive pulmonary disease, that has caused increased pressure in the pulmonary circulation. Sclerosis is found more frequently in the heart, brain and kidneys and is correlated to a large extent with a history of nephritis, rheumatic fever or some other acute infectious disease.

It does not appear with some degree of regularity among all people regardless of sex, race, occupation or age. It should be emphasized that even in ripe old age well marked arterio-sclerotic lesions need not necessarily be present in the blood vessels. This observation as well as the fact that in some individuals the disease occurs early in life, long before senile changes have set in, shows clearly that old age alone is not the most essential causative factor. Arteriosclerosis is by no means universal. Using the figure of 1 to denote the percentage of cases of arteriosclerosis in the United States, Japan's figure is

.56 and Sweden's is 1.12. It has been shown (198) that negroes have arteriosclerosis much less than whites and that those in the United States have more than the native negroes. Even among the urban and rural negroes there is a difference in incidence. Women show less evidence of it than men.

The changes due to arteriosclerosis are not predictable. Persons with extensive "hardening of the arteries" may live beyond the expected number of years and those with only a slight degree of arteriosclerosis may die young. Even with this same degree and distribution of the arterial change, the clinical symptomatology varies greatly.

Of course there are changes in the arterial wall with old age. These will be described but they are by no means arteriosclerotic. Age, it must be concluded, is a predisposing factor in arteriosclerosis but of little importance, if any, in the causation of this disease.

## BLOOD VESSELS

### Anatomical Changes

In the early forties a progressive loss of elasticity begins to take place in the vessels, and above all in the aorta, which had not made itself felt until then. (189) From the histological point of view it manifests itself by incipient changes in the structure of the aortic wall and more especially by an increase in its' connective tissue framework. This is most pronounced in the interna of the aorta, where the characteristic relation between abundant elastic and comparatively scanty connective tissue undergoes a progressive change in favor of the latter. At the same time, however, there is also a quantitative increase in the elastic tissue of the interna. This is especially pronounced in vessels like the radial artery. In such vessels there is a splitting up of the marginal elastic tissue between the interna and the media rather than a connective tissue proliferation. Increases of the elastic and connective tissue framework are more or less conspicuous also in the media.

All these processes taking place in the aging vascular system have one common feature inasmuch as they result in hardening and thickening of the walls of the vessels. This would necessarily bring about a narrowing of the lumen if it were not for a second process equally characteristic of aging vessels, namely the stretching of their walls, in both transverse and longitudinal directions.

( 6 )

This is linked with dilatation of the lumina and progressive tortuosity of all vessels belonging to the arterial system. No doubt its causes are to be found in changes in the histologic structures already referred to. This condition can be demonstrated most easily in the aorta. Whereas in the prime of life the root of the aorta generally has a circumference of about 50 mm., this increases to 70-80 mm. or even more in the course of advancing age. This holds true also for the thoracic and the abdominal aortas, which exhibits an increase of 15 and 10 mm., respectively, approximately in the same proportion, even though the absolute figures are smaller. (18) Owing to this general process of senile dilatation, the vessels of the arterial system not only become too wide, but also too long, so that they no longer fit into the usual channels, but are constrained to take a tortuous course. At the same time their elasticity becomes measurably diminished. Because of the increasing elastic resistance of their walls the vessels like the aorta will stretch less and less easily in longitudinal and transverse directions. The elasticity being no longer adequate, a vessel once it has been distended cannot regain its original size so that dilatation becomes progressive.

#### Coronary Arteries

The coronary arteries present an interesting picture. The ramus descendens anterior shows maturing five to ten years earlier than the posterior branch. Even in the single branch changes take

place at different times in different places. For instance, the point about 1 cm. from the origin of the anterior branch shows calcification and ultimately constriction and thrombosis more than any other area. ( 87 )

During infancy there is a relative equality in the thickness of muscle of the right and left ventricles, but as the child develops and becomes active, the muscles of the left ventricle develop more than those of the right. To supply this increased musculature, there has to be an increased blood flow. While the child has straight coronaries and lack of anastomosis, as the individual becomes older, the arteries become tortuous and anastomatic channels develop. In old age there is a marked increase in the vascularity of the heart due to this increase in anastomatic channels and an increase in diameter up to two and a half times.

( 58 )

#### Cerebral Arteries

With advancing age numerous alterations begin to occur in all layers of the cerebral arteries. One of the first structures to show change is the internal elastic lamina. It begins to show areas of reduplication as early as the fourth decade. This results in a pseudo-reduplication not to be confused with the true multiplication occurring in the fifth and sixth decade. It also loses its ability to stain normally and appears patchy and indistinct. Once in a while some segments of the lamina appear swollen and extend

into the media. The thickened pale staining membranes, instead of appearing as uniform structures, are split not only into longitudinal branches but also into transverse fragments. (10)

A complete reduction in the quantity of the elastic and muscular elements occurs in the media. The fibrosis of the media which follows makes it impossible to differentiate it from the adventicia. This change is followed by the loss of outline and hyalinization.

The extreme fibrosis and hyalinization of the vessel elements often fray the arterial wall and weaken it to such an extent that the erythrocytes break through and escape from the lumen to the peri-vascular spaces.

The smaller the vessel, the earlier the change is seen.

The histological picture shows increasing elastification of the media, elastic hyperplastic changes in the intima, fusion of the two layers, atrophy of the smooth muscle elements and development of irregular patches of connective tissue. Examples of these changes are found in the posterior papillary muscle of the left ventricle first; next in the interventricular septum, left ventricle and pulmonary conus, and last in the auricles.

## Physiological Changes

Blood pressure has undergone much discussion and the present conception is more conservative than previously advocated. In the past a rule of one hundred plus the age in years (158) or anything under 140 mm. Hg. have been called normal. Now 120 mm. Hg. is considered the average of normal. This is of course taken under basal conditions.

In averaging the blood pressure for the group many factors have led to error. The inclusion of hypertensive patients, lack of standardization of methods, selection of an unrepresentative group, negligence of obtaining a basal level by eliminating factors which tend to elevate the blood pressure and taking of the average, not the modal, all tend to give an erroneous picture.

It is generally conceded that patients with blood pressures over 140/90 are definitely in the hypertensive group, so eliminating these and obtaining as representative group as possible conditions will give us a more clear picture. It was found (150) 80.4 percent of all men and 71.1 percent of all women had blood pressures under 130 mm. of Hg.

Systolic blood pressure in the 60 plus year age group.

Systolic pressure under 130	60-64	70-74	75-79
	Male	74.	66.7
80.4	Female	66.6	
	71.1		



Diastolic pressure under 80

Male	79.7	85.7	88.9	83.4
Femal	68.5	80.	100.	100.

e

Added to the factor of the "normal" blood pressure in this group is the factor of arteriosclerosis which is present in 95 percent males and 85 percent of females. Thus it can be said that there is no rise in blood pressure in the "normal" group but a rise is found in those prehypertensive people.

Hypotension is neither a disease nor a disease entity; it is an ideal blood pressure level, in the absence of other findings. This is true of pressures that occasionally dip to the level of 80 systolic and 50 diastolic. The commonly described symptoms of the disease hypotension can be ascribed with equal statistical accuracy to any level of blood pressure.

The daily and yearly variation of normal blood pressure show proportionately greater and more erratic yearly variations. A blood pressure history of over 120 systolic and 80 diastolic over a ten year span is pathologic, and is a almost infallible sign of incipient hypertension. Even transient elevations of blood pressure should not be ignored but suspected of a further and more frequent and possible permanent rise.

High blood pressure is a long term disease having its beginning at an early age. It is not a disease that suddenly merges

with middle age. Slightly more than 40 percent of the adult population is either actually or incipiently hypertensive. The mortality rate rises with the systolic or diastolic pressure.

Venous pressure is within normal range in the aged. (32) The circulation time, as measured with the florescence and cyanide method, shows a definite trend toward slower rates with increasing age. On the other hand the velocity of blood flow through the lungs shows no constant relation to the aging of the patient. Studies of the pulse wave velocity of the radial artery and the aorta show an increase with age. (40) This is associated with a decrease in elasticity which may be halved at the age of sixty. The blood volume is somewhat decreased in the older patients.

The pulse rate generally increases in persons over sixty-five years of age. The difference is small so that even at eighty there is a difference of less than eight beats per minute. The rhythm of the heart tends to become more irregular with advancing years. Premature contractions and brief periods of auricular fibrillation are the most observed changes. The minute cardiac output shows no change in old age but with it is a 9.3 percent decrease in oxygen consumption. (40)

## CHANGES IN THE BLOOD

The cellular types in the bone marrow are about the same in the aged as in the middle aged group. There is greater variation in the blood platelet level but the variation is within normal levels. The ability to recover from loss of blood or blood elements is much slower than seen in the younger age group. (147)

The opsonic index of the serum and the phagocytic activity of the white blood cells have been reported to be diminished in amount. The sedimentation rate is accelerated, more markedly in aged women. (111)

Antisera production of different ages differs in quality. Sera of the same titer may have qualitative changes which allow the two (young and old) to act differently. (15&16)

In a two year study it was found there was a gradual lowering of the glucose tolerance. In 57 percent of people over sixty a glucose tolerance test showed either a delayed return to normal or a mild diabetic curve. (76) Although there is a very little difference in blood fasting levels the kidney threshold rises from 180 to 210 mgm. percent.

The cholesterol level in the blood has been debated, but it seems to be, on the average, about 215 mgm. percent. The female average, 237 mgm. percent, is higher than the male, 196 mgm. per

cent, throughout old age. In the eighth and ninth decade there is a drop in both sexes of from 30 - 40 mgm. percent. (91)

	Average	Male	Female
Hemoglobin	12.9 grams Mb.	13.1 $\frac{1}{2}$	12.5
Red Blood Cells	4,580,000 Gr. per cmm.	4,610,000	4,460,000
Hematocrit	41.4%	41.7%	40.4%
R.B.C. Diameter	7.5		
Leukocytes	7,220 per cu mm.		
Band Neutrophiles	4%		
Segmented Neutrophiles	60%		
Eosinophiles	2.8%		
Basophiles	1. %		
Lymphocytes	27.5%		
Monocytes	54. %		
Specific Gravity	1.054 (1.056 - normal at 30-40 yrs.)		
Lactic Acid	16 mgm per 100 cc		
Calcium	7.7 - 10 mgm per 100 cc.		
Oxygen carrying power	35 cc per kilogram body weight is considered normal	53 cc	
Volume Index		1.02	.88
Color Index		.99	.88
Packed Cell Volume		41.2	36.7
Mean. Corp. Volume		97.7	90.
Mean Corp. Hemoglobin		28.5	28.8
Mean. Corp. Hemoglobin Concentration %		29.6	32.3

Saturation Index	.98	1.
Reticulocyte Count %	.53	.64
Hemolysis starts at	.45	.42
Hemolysis completed at	.33	.35
(9 & 112)		

# RESPIRATORY SYSTEM

## RESPIRATORY SYSTEM

In dealing with respiratory system in old age, it must be remembered that the lungs are no longer young, and that they exhibit marked functional and anatomical digressions. More or less advanced pulmonary changes are discernable at almost every geriatric necropsy. The lung is light, diminished in substance, incompletely collapsed, and poorly elastic. Its dimensions are absolutely increased if the general body weight is unimpaired, or relatively increased with overlapping of the heart and lowering of pulmonary bases if the viscera are reduced by cachectic disease or by senile atrophy.

The primary pulmonary changes are: (a) loss of functional tissues, (b) the reduction of breathing surface by confluence of alveoli, (c) reduction of capillary volume by destruction of intervening alveolar walls in the formation of larger confluent alveoli, (d) decrease in the number of elastic fibers and impairment of their elasticity with consequent diminution of the retractive or expiratory forces of the lungs.

In addition to changes in the lungs, modifications appear in the bones, joints and muscles of the thoracic cage which are associated with deviations in the shape of the chest. There is lessened mobility of the thoracic wall due in part to a weakening of the intercostal muscles, a decrease in the range of motion in the coste-vertebral joints, and especially to a stiffening of the

attachments of the ribs. The water content of the costal cartilages decreases with age and the calcium content increases. Whereas only 125 milligrams percent of dry material is found in the second decade, there is 617 milligrams percent in the fifth decade and 1399 in the seventh. (184) Besides this greater rigidity of the thoracic wall there is the effect of lack of exercise and of indolent habits in the elderly which would tend to reduce the ability to engage in vigorous and ample respiratory movements lasting a long period of time.

The broad chests and short lungs of the pyknic and athletic types usually present a picture of a so called "barrel chest". It is characterized by enhanced volume, enlargement of the diameters at the base, and depression of the diaphragm. In these types the effect of the enlargement of the lower thoracic diameters is aggravated by a bulky fat omentum and a thick abdominal wall which handicap diaphragmatic depression.

The chest of the asthenic person is often periform in old age brought about by an increase in volume of the upper half of the chest. The length of the lungs checks the efficiency of the diaphragm. The diaphragm is accordingly depressed, and as it sinks, its dome is proportionately flattened. As a matter of fact, the base of the thorax becomes smaller when the diaphragm, now without its dome, contracts. Anchored to the chest walls the edges of the diaphragm then tend to approach the center of the



flat diaphragmatic sheet. As the walls converge the base of the chest decreases in size. Since the base of the chest is thus immobilized, the upper portion is the only portion in which adequate expansion occurs.

In the anteroposterior plane, extended space for expansion in the upper part of the chest is made available by the hunching of back (kyphosis) and by the raising of the sternum. The lateral diameter is enlarged by elevation of the upper ribs which become more or less horizontal.

The degree of calcification of the costal cartilages, a condition often seen in senile persons, is another determining skeletal factor. Calcified cartilage no longer retains the elasticity of normal cartilage, and calcification may be so far advanced as to produce complete rigidity or fixation of the chest. Hypertrophy of the accessory muscles of respiration such as those in the neck and at the sides of the chest, calls attention to the decrease in functional capacity. Although atrophy and degeneration of the muscles of the diaphragm are commonly present they are, of course, not clinically demonstrable.

Among the results of the changes in the chest and lungs are: (a) a limitation of respiratory movements, (b) a tendency for the chest to assume the inspiratory position, even at rest, (c) a noticeable diminution of vital capacity, (d) an increase in the amount of dead air space. These factors, added to the reduction

of alveolar and capillary surface, provide ample grounds for the inability of the breathing apparatus to respond, to increased demands upon it with resulting incomplete oxygenation of the blood passing through the lungs. The enhanced resistance in the pulmonary circuit is a further consequence of the fewer pulmonary capillaries, of the lesser flow of blood through the lungs in inspiration than in expiration, and the curtailment of respiratory movements with a resultant depreciation of venous suction.

Decrease of Vital Capacity with Age: (114)

Age	40	45	50	55	60	65	70	75	80	85	90
% of Normal	79	80	75	75	72.4	70.6	70.2	69.2	68.8	68.1	62

# NERVOUS SYSTEM

## NERVOUS SYSTEM

No field of medicine has felt the impact of geriatrics more than psychiatry. This impact has made itself felt in the great increase in the number of patients admitted to the state hospitals, an increase for which the facilities for care and treatment as well as study are entirely inadequate. From 1923 to 1945 there was a 150 percent increase in admissions of persons aged 60 or over to state hospitals, and a 116 percent increase in resident patients of the same age group. (133)

### Mental Changes

#### Importance

A dynamic dualism sets in when physical strength decreases with age and mentality remains strong, for old age does not always signify senility. During senescence the creative impulse may continue to exist, and frequently even surpasses that of earlier years. The gifted and intellectual personalities show a relatively slower degeneration with the onset of old age. Strong will and a capacity for making important and far reaching decisions are, like creative talent and lively sexual appetites, often undiminished by age. Generals of advanced years have carried their armies to victory against heavy odds. Matters of national and international importance are usually entrusted to elder statesmen.

So many individuals have made outstanding contributions in

advancing years that it seems reasonable to conclude that a life-time spent in activity, creation and self development need never cease to be fruitful if health is present. Men working in science enjoy a mental stimulation which increasing experience can only augment. In the chosen group age evolves wisdom, compassion, understanding and objectivity. On these qualities must the administration of justice depend, thus it is no rare chance that the high courts of the land are traditionally conducted by men of advanced age. Despite the apparent mental vigor displayed by such persons to the last, necropsy sometimes reveals low weight and an astonishing amount of brain deterioration and atrophic change.

#### Motor Ability

The measurement of the correlation between motor ability and age gives one index to activity in work, competence, and recreational adjustment in maturity. Decrement is greatest in the less practiced activities and in strength and quickness. It is least in the more experienced functions. Habits established through exercise, although sometimes spoken of as associated with the rigidity of the old age, have thus their favorable aspect in the better maintenance of experienced effort. (110) In complex manual activities, the characteristic speed or dexterity loss regularly appears as age advances. There is gradual slackening in speed from the twenties to the sixties, with a sharp decline in rate thereafter. Practice and experience contribute to speed of per-

formance. The person with mechanical training maintains adult speed rate into late maturity. The speed loss in men is gradual from decade to decade and is twice that of women. Womens loss is slower in the forties and fifties, but rapidly declines thereafter.

### Intelligence

The results from intelligence tests by many investigators give rather adequate evidence of: (1) score decline from young adulthood to old age; (2) greater decrement of the speed versus the "power" of intelligence; (3) better preservation with age of verbal as compared to the mathematical and manual functions; (4) wide individual differences in score at every age. The peak is reached in the late teens and early twenties. Until the middle forties the score is about the same, but then declines with increasing rapidity. A person with a mental age of nineteen at forty years will have one of about the level of seventeen. The average decade scores of men and women are generally about equal whatever test is used, and they describe approximately the same decrement curve. Information test of intelligence shows increment rather than decrement up to the late forties, with a decline after the age of fifty. (196)

### Learning Ability

There is a decrease in learning ability with age but no one is ever too old to learn. The measured rate of learning follows

the characteristic parabolic curve of rapid rise in childhood and youth to a young adult peak, with a slow decline thereafter to late age. Adults in the forties learn approximately 85 to 95 percent as efficiently as twenty year olds. Whatever his age, the learning individual improves with training in every type of exercise. Learning ability is closely correlated with native ability, practice and repetition.

Individual, social and cultural attitudes and interests differentiating the sexes show little age change for women in adulthood, while a marked trend inclines the men's decade scores gradually toward the women's mean. This tendency, although persistent from early to late age, never obscures the sex difference.

#### Etiology

(Other than purely neurogenic)

With age, responsibilities increase and may greatly color the older outlook on life. Insecurity develops when confidence in his body, or in his earning power or social desirability is shaken. This insecurity leads to a need for self-protection, a readiness to defend what he has achieved, caution and frequently indecision. These reactions are recognized in many of our social attitudes such as seniority rule and the policies of government. With this psycho-logical development is considered it is not surprising to find so often irritability, anger, anxiety, and resentment, emotions which interfere in social adjustment and have an undesirable physiological

influence.

Another expression of aging is the difference in emotional attitude towards the past and the future. There may be a tendency to remember the lost opportunities of youth and the failure to do justice to what one has gained from an active life. The desire to obtain what has been missed may lead to ill-considered adventures. The attitude toward death gradually becomes more philosophical. Death becomes accepted, but anxiety arises in connection with the responsibilities involved. Loneliness may become an important factor in the life of the unmarried person and in that of many mothers whose children have grown out of the home. A narrowing of interests and a tendency to more aversions and to intolerance make a social reorientation difficult.

With the increasing difficulty in making rapid adjustments, in carrying out fine movements, in remembering details, comes a tendency to feel insecure and uncertain. This may be compensated for by development of that judgment and wisdom which is so much valued by some of the more progressive employers in their older employees. It may, on the other hand, result in an increasing timidity and hesitation with related worry and anxiety over the security of the

One need only mention, in addition to the loss of friends and cronies, the realization that the physical and mental powers may be flagging and will continue to fail and the increasing sense of



loneliness, to realize that many of the many influences at work in the aging person to be sought in the mental realm. The attitude of the family may constitute a threat, just as it may a support. Children are often inconsiderate of the feelings of older people and unduly resentful of any advice. The feeling of not being wanted threatens the integrity and security of the aging individual. Our society in general tends to undervalue old age. It has come about that the term senile is almost a term of derrogation rather than merely an adjective "old".

#### Symptoms of Decline

Of the many physiological changes occurring during the period of senile involution, the aging mind presents the greatest vagarities.

Usually the first sign of mental impairment noted is impaired memory, forgetfulness of names and haziness of events and their sequence. (134) However, much earlier signs of mental impairment, though seldom noticed, are a general slowing down of all mental processes and rapid brain fatigue. The person speaks more slowly. He must make a conscious effort to recall even stand circumstances and fit them into problems of the immediate present. Another frequent sign of mental impairment is lessened attention, while prolonged attention brings on rapid brain fatigue.

The layman frequently speaks of the wonderful memory of some

old person who recalls events of his early life, but a study of the mentality of such a person would show that he cannot recall recent events. This is not due to impaired memory as much as to lessened attention and weakened visual and auditory impressions, due probably to anatomical change. The lessened attention may be nature's provision to prevent mental fatigue, the same as is found in those who fall asleep during an interesting sermon or play.

Among the vagaries of the aging mind are obsessions, delusions, morbid fears, and tenacious retention of antiquated ideas or customs.

Another early indication of the mental changes in the process of aging is inability to adapt and adopt oneself to new concepts of modesty and morality. The aging mind cannot readily accept the changing concepts of morality, birth control, unconventional social relationships, deviations from the golden rule and the ten commandments, which are generally accepted as necessary under modern conditions of life.

The most bizarre of the vagaries of the aging mind occur in the realm of imagination. The novelist employs his imagination deliberately to create the pen pictures of the supposed events he describes. In the aging mind such pictures arise unbidden and often with the vividness of reality. The individual knows that they are figments of the imagination and tries to dispel them by interposing real, serious problems or resorting to other mental

diversions. Aging persons do not like to speak of what they can recognize as silly notions, and only tactful questioning will elicit these silly notions, day dreams or whatever the individual chooses to call his unwelcome thought. However, if it is a pleasant picture that he has created he will consciously elaborate it, and may act upon it even though knowing it is only fantasy.

Women have bizarre imaginations but it is much more difficult to dig them out. The female climacteric creates a much greater change in the personality of the individual than the change in the male who passes through a similar stage. In the male the seminal vesicles fill more slowly, libido and potentia wane gradually the emotions of love which are found are weakened and may have disappeared entirely. In the female the much more rapid and profound changes wrought during the menopause are reflected in her changed mentality, intellect, emotions and will. While the aging mind permits wide diversions and variations in the intellect, the changes in the emotional sphere are in degree rather than in kind. The emotions are blunted, but the exhibitions of the emotions are pronounced. The individuals become more demonstrative, yet the emotion itself, love, hate, fear, grief, joy, etc. is shallow and soon passes away and is forgotten.

There may arise in the aging mind odd variations of the emotions, odd likes and dislikes, loves and hates, joys and griefs, hopes and fears, abnormal attachments, over reaction or fixation of emotion are seen in every case of older patients.

The will is the least affected of the mental functions of the aging mind. Aging people become more stubborn, adhere more to obsolete or preconceived notions, and are less susceptible to reasonable argument. Occasionally one will accept new ideas from one person after rejecting the same idea from another. (123)

#### Mental Change and Arteriosclerosis

There is a good deal of confusion about the incidence and importance of psychoses of senile cerebral arteriosclerosis and psychosis.

#### Opinions of Various Authors

Numerically the largest group of mental disorders of later maturity is that of psychosis with cerebral arteriosclerosis. The distinction between these and senile psychosis is perhaps in a large part academic. The prodromal signs are similar and they tend to occur in persons of 55, however, the senile group is likely to develop a decade or more later. (135)

Senile dementia, which is more frequent in women than men, usually comes after the age of 70, while dementia due to cerebral arteriosclerosis is more frequent in men and occurs after the age of 65. Arteriosclerotic dementia, as a rule, follow cerebral accidents, especially cerebral thrombosis. There is no sharp dividing line between mental change and senile dementia, one being a normal physiologic condition, the other pathologic. (156)

The only condition with which senile dementia may be confused with is cerebral arteriosclerosis. For many reasons, anatomical and pathological, as well as clinical, it is important to effect such a clinical differentiation and it should not prove too difficult. The age of onset in the senile group averages 75 years and in the arteriosclerosis group 66 years. (71)

Robinson (151) makes the claim that senile dementia and arteriosclerosis are not the common mental disorders in persons over 60. The common disorders and primary toxic delirious reactions and agitative depressive psychosis superseded them in frequency. These conditions can be cured readily by the modern methods at our disposal.

#### A Few Points in Favor of Differentiation

It is not within the scope of this paper to go into detailed discussion on this point nor is it appropriate to come to a definite conclusion in face of the wide spread disagreement. There has, however, been a tendency in recent years to throw into the arteriosclerotic group more cases than was formerly the practice, and in general it may be said that where evidence of arteriosclerosis, particularly with focal lesions, is prominent, the diagnosis of senile psychosis is usually not made.

It is a fairly common experience to find patients who have a fairly advanced degree of arteriosclerosis, perhaps even demonstra-

ble as affecting the vessels of the brain, yet who show extra-ordinarily little mental impairment. (133) This is true in a good many cases. In fact, one of the things which the studies of the pathologists have shown has been that the nature and the extent of the cerebral lesions are far from indicating the nature of severity of the impairment of the ability of the patient to adjust a situation. The well integrated individual may continue to make an effective adjustment in spite of serious lesions. On the other hand, the person who has shown unusual traits such as suspicious-ness, miserliness, seclusiveness, or a tendency to become depressed upon slightest provocation, may show an exaggeration of these trends when relatively little damage has been done to the brain.

Many persons pass through the earlier part of the so-called arteriosclerotic period with relatively little damage and some very old persons do show extraordinary elasticity of the blood vessels. In the aging person, particularly the one who has passed the age of 70, one may look for evidences of dotage.

In a number of series of autopsies it has been found that 90 percent of men and 85 percent of women over 60 years of age had arteriosclerosis. These statistics confirm the concept that pathological conditions are not the only factor in the dementia of old age. It is certain that 90 percent of the men and 85 percent of the women over 60 are not afflicted with mental symptoms. (195) If the mental symptoms shown by the patients were on a pathological

basis alone then attempts to cure would be useless, yet, according to various authors, from 24 percent to 66 percent made enough progress to be sent home with only 2 percent to 12 percent remissions. This percentage must be considered in the light of the usual number of cures at any age and the fact that any manifestation of personality change is harder to treat in the aged. (51)

## NEUROLOGY

### Anatomical Changes

#### -Brain-

There is an increase in the skull thickness and the brain shows atrophy both microscopically and macroscopically. The bones of the skull usually thicken uniformly but there may be senile atrophy of the parietal bones. This is indeed a rare condition but seen often enough to warrant mention when pathology is suspected in that area. In the parietal bones between the sagittal suture and the parietal prominence there is a triangular or quadrilateral flattened depressed area or there may be a trough like depression running longitudinally along both sides of the sagittal suture. Such thinning is very rarely found in other bones of the cranium. There has been described no clinical significance to such a finding. There may be focal osteophytic changes which is thought to cause clinical manifestations, Morel's syndrome. (166)

From the age of 40 to 50 there is a constant reduction in the weight of the brain. The loss is from 160 to 200 grams and is greater in females than males. Atrophy of the frontal lobes is the greatest. The brain substance is usually found hard but if softness is found it indicates a vascular lesion of some sort. The lateral ventricles are dilated and the walls of the ventricles may be studded with granulations and various markings. Scattered throughout the cerebral cortex as circular areas of densification



are senile plaques. There is no correlation between mental symptoms and the severity of pathologic changes.

Microscopic changes in the brain:

- (132)1. Numerical atrophy of nerve --- especially in the cell<sup>3</sup>rd cortical laminae and frontal lobe.
2. Diminution in size of neurones.
3. Excess in pigmentary elements.
4. Thickening of intercellular fibrils.
5. Widespread glial overgrowth.
6. Lipoidal changes and a mucocytic degeneration of the oligodendroglia.
7. Increase in hortegea cells.

The dura mater is thickened and may be adherent to the inner table of the skull. The choroid plexus undergoes cystic changes, proliferation of connective tissues, vacuolation, and pigmentary degeneration of the epithelium, with deposits of calcareous, hyaline, and psammous bodies.

#### - Spinal Cord-

Most of the atrophic changes in the spinal cord are microscopic. There are patches of perivascular demyelination, reduction in the number of nerve fibers, especially in the lateral and posterior columns, neuroglial proliferation, and wide spread changes in the ganglion cells.

In the spinal nerves there is a decrease in the number of dorsal and ventral root neurons. From an average number of neurons of 8,700 in the 8th and 9th roots in the second and third decade, there is a decrease to 5,700 in the sixth decade. There is also a decrease in the number of ganglia cells. In the case of the 8th and 9th thoracic dorsal root ganglia, there is a decrease from 37,000 ganglia cells in the fifth decade to 30,000 in the seventh decade. (62)

It is of interest to compare the loss of fibers in the olfactory nerves with that occurring in the spinal nerves. It has been shown above that the number of fibers in the dorsal and ventral roots of the 8th and 9th thoracic nerve roots does not begin to decrease until after thirty years of age, and that ultimately the loss does not exceed thirty percent. This contrasts sharply with the findings for the olfactory nerves in which the loss begins in the first year of life and amounts to seventy-three percent in old age. (167)

#### -Peripheral Nerves-

In the peripheral nerve trunks, the neurokerating network of the cells change from a network in younger persons to a clumped mass with geometric designs. (42)

The most outstanding change with age is the increase in the connective tissue elements, which is concomitant with reduction of

the potency of the blood vessels and destruction of nerve fibers. Not only do the normal collagenic elements increase, but there is a gradual invasion of the nerves by a fine, non-collagenic connective tissue. This replaces normal nerve fibers and itself degenerates frequently. Myelin and axic cylinders exhibit degeneration in surrounding areas. In the fifth decade, these areas of destruction are conspicuous and, in general, they increase with advancing age.

#### Physiological Changes

As a result of this aging of the nervous system, there are sensory and motor changes. These are not always obvious and may be often confused with pathological processes. A keen judgment is needed to use the knowledge of the normal changes in determining the pathological.

#### Motor Ability

Motor ability decreases from the thirtieth year onward in a characteristic parabolic curve. The greatest decline is noted in less practiced functions and in strength and quickness. Manual skill also suffered a similar loss. Here again practice and experience improved the scores. In women there is less loss of ability in the forties and fifties but then there is a rapid decline. (176)

#### Sensation

The diminution of sensation is very important to recognize in

old age. The decrease in pain will be discussed under another heading. In addition heat, cold and tactile discrimination is noticeably subnormal. Loss of sensation of touch may make it difficult to walk safely. Sense of position is often affected and a patient may give a suggestion of a positive Romberg sign. A progressive defect of vibratory sensibility is very common, evidenced first in the distal portions of the extremities.

### Reflexes

Reflex activities are likewise altered. There are sluggish pupillary reactions, sluggishness or even absence of ankle jerks. The plantar reflexes are often equivocal in old age and sometimes extensor responses appear without any associated pathology. The abdominal reflexes are lost early.

Changes in motility are due to muscular and joint changes as well as neurological in nature. A slight but generalized poverty of movement is characteristic and is associated with slowness of movement and an attitude of flexion and hypertonus.

### Electro-encephlogram

As a whole, the pattern of the electro-encephlogram of a very old person is more unstable, shows more distortion of wave forms and a large amount of slow wave forms and activity, giving the appearance of a broken down pattern.

## SPECIAL SENSES

### Ear

Though the ear reaches maturity twenty odd years before the body as a whole, it appears to inherit a functional expectancy terminated only by death. (57)

The external ear is of comparatively little functional importance but, like the other appendages of the body, shows changes with age. Beginning in the third decade there is atrophy, loss of elasticity, dehydration, pigmentation, growth of single long hairs from unexpected locations, wrinkling in front of the tragus and in men a flabbiness and lateral prominence. The drum membrane and skin of the meatus often show thinning. The external meatus may be enlarged from atrophy or narrowed due to bulging of the walls. There is a stiff thick growth of hair in the meatus which is often disagreeable. When the membrane is sufficiently thinned variable degrees of the middle ear can be seen, but this atrophy, unless accompanied by release of tension, does not impair hearing.

The eustachian tubes in aged persons are usually more patent than those in younger persons. The muscles, tendons and ossicles of the middle ear tend to atrophy, but almost imperceptibly.

Histologically there is no significant difference between the vestibular structures from the young and old people, but there is no question but that after 35 to 40 everyone shows signs of aging

such as delayed reaction, diminished security of orientation and equilibrium under sudden or unexpected changes in position. However, all tests for measurement of the changes in the vestibular apparatus caused by the aging process are unsatisfactory and no accurate statistics are available.

The first notable impairment in the auditory apparatus is noted in the frequency range about 4,000 cycles. As the person grows older he loses hearing for the extremes, the high and low. The higher frequencies are first to be lost. The hearing of speech, particularly in a crowd, is distinctly affected.

#### Eye

Eyelids - The skin of the eyelids, as does the skin over the rest of the body, changes with age. It becomes thin and wrinkled due to the loss of underlying fat and elasticity. One of the most common cosmetic defects is a bulging of the orbital fat above and below the palpebral fissure. This change is caused by a thinning of the septum orbitale. The defect may be sufficient to cause a drooping of the upper lid over the eye. With diminished muscle tone and laxity of the skin the lid may fall away from the eyeballs causing ectropion, etc. The palpebral fissure is narrower and there is a loss of the retrobulbar fat pad causing the eyes to sink deeper into the orbital cavity. (54)

Conjunctiva - The conjunctiva is thin and usually contracts.

It is also the frequent site of hyalin and lipoidal changes.

Cornea - The luster, found in youth, disappears with age. There is often a lipoidal degeneration which forms a white ring near the limbus which is called arcus senilis. The corneal diameter lessens with age. An actual decrease of 5 mm. in diameter takes place. There is also a flattening of the cornea, more in the vertical meridian with an increase in the perverse astigmatism. This increase is marked after 70 years. The Massal-Henle warts of the cornea can always be seen with the slit lamp. They increase with age, but have no functional effect. (68)

Sclera - The sclera takes on a yellowish color due to fatty degeneration of the whole sclera. The sclera become more rigid. A small yellow spot, the pinguecula, appearing at the lateral poles, gives the appearance of a neoplasm. Fat deposits also are limited by the Bowman's membrane superficially, but extend toward the deeper layers of the cornea.

Iris - The changes in the iris are characterized by a thinning of the whole structure with the loss of crypts and a sclerosis of the vessel walls. Connective tissue, sparse normally in the iris, is replaced by much hyalin tissue, sufficient at times to form a ring at the papillary margin. This latter may cause senile myosis. The vessels, often seen as fine white lines, are greatly thickened, but remain potent. Pigmentary degeneration

regularly occurs, with pigment becoming deposited on the posterior corneal surface, the surface of the iris and, more especially, in the pectinate ligament. With advancing age, the color of the iris fade, especially in blue-eyed persons. The pupillary margin pigment ring is frequently broken and sometimes everted. The pupils become smaller and less reactive to light and accommodation.

Lens - Changes in the lens occur at the same time as changes in the epithelial appendages. There is little change early in life but in the seventh decade a sclerosis of the lens nucleus occurs with a loss of the nuclei of the lens fibers. This causes the whole nucleus to become a homogeneous mass from the pressure of newly formed fibers in the lens cortex. Coincidentally, there is a loss of water, insoluble protein replaces the soluble and oxidative metabolism is lost. There may be a color change, the gray reflex, in the lens but whether this is pathological or not is still debated.

There may be extreme degrees of nuclear sclerosis with little effect upon the vision, except lenticular myopia, and a disturbance of color vision, especially blue. On the other hand, a slight sclerosis may reduce vision to a minimum, especially for reading, even while the fundus is clearly visible through the lens. These changes, in addition to being a factor in the loss of accommodation, and producing presbyopia, also cause other serious changes in the circulation of ocular fluids.



The loss of elasticity of the lens causes a decrease in the power of accommodation. While it is calculated to be 11 diopters at 20, 6 diopters at 40, it is reduced to 1 or less after the age of 60. In addition, 90 percent of people show some opacities in their lens.

The pectinate ligament or the trabeculae of the anterior chamber is also involved in the general sclerotic process. Proliferation of the collagenous material from which the ligament is derived, depositions of hyalin material, and sclerosis of the lamellae themselves are constantly seen.

The ciliary body - The degeneration of the ciliary body is a large factor in the production of presbyopia. In this process there is an atrophy of the muscle tissue, a deposition of connective and hyalin tissue between the fibers, and a complete hyalini-zation of the ciliary processes. In addition, the change involves the pectinate ligament as previously described. The ciliary processes themselves become larger, pushing toward the base of the and embarras the size of the posterior chamber.

In most cases fatty droplets occur in the fibers of the muscle itself, in the nonpigmented portion of the ciliary epithelium and in the interstice of the sclerosed tissue.

Retina - The retina becomes less transparent and has a duller appearance in advanced years. Senile macular degeneration and

colloid bodies are frequently observed. Arteriosclerotic and arteriolosclerotic changes have been well described.

The choroid - The sclerotic changes seen in the retina are also the chief senile variation in the choroid. Lipoidal in-filtration of the intima and muscularis causes a narrowing of the arteries and veins. Since all arteries are not affected, this atrophy of the choriocapillaris is patchy in distribution and compensatory dialation takes place in the unaffected areas. There is a fatty degeneration characterized by deposits of fat in the endothelial cells. The muscular coat is replaced by fibrous and hyalin tissue.

The vitreous.- Liquefaction in the vitreous gel results in an accumulation of the supporting fibers into strands. Liquefaction occurs most frequently in the anterior pole. At times highly refractile particles or floating bodies resembling cotton balls are seen in the vitreous. These are made up of cholesterol or calcium salts. They have little or no effect on vision.

Robertson and Yudkin show that dark adaptation, as measured by the final rod threshold, is impaired in old age. There is also a gradual decrease in visual acuity and a gradual narrowing of the visual field. (149)

# DIGESTIVE SYSTEM

As has been mentioned before, along with heredity and infections, nutritional factors play a major role in influencing the aging process. Nutrition is a factor in resistance to infection, alterations in the cardiovascular system and the nervous system. It plays an important part in glandular secretion and metabolic process of the human body. Diet in the youngster is a major topic of conversation, worry and experimentation among doctor dietitians and mothers but little attention is paid, with the exception of those who are vitamin conscious, to the adult diet and even less in old age. The aged person is "supposed to eat less and want less". They "naturally" are more finicky in their choice of foods. No more false conception could exist.

#### NUTRITION

The story of geriatric nutrition cannot be told until all the facts of the normal physiology of this stage of life have been determined. Not only does this involve a knowledge of the muscular capabilities of the old gastro-intestinal system, but it also encompasses the intrinsic and extrinsic glandular reserves and the end results in blood chemistry. A survey of these physiologic entities should uncover the characteristic required diet.

#### Causes of Poor Nutrition

There are many reasons for poor nutrition in the aged. Appetite is often affected due to such things as disturbances of

sight, smell and taste, as well as diminution in salivary and gastric secretions. The appetite is often perverted and hunger may be felt shortly after a heavy meal. This gnawing sensation has been attributed by some to an abnormal irritation of certain centers in the diencephalon. The aged are often childish in their likes and dislikes. They may gorge themselves on their favorite food and refuse to eat enough basic foods.

Lack of teeth or improperly fitted teeth is a major cause of digestive disturbances. A loss of teeth leaves the aged person with not enough grinding surface. There may be a large amount of decay in the mouth and poor oral hygiene is not unusual. This provides the proponents of focal infection as a causative agent in disease a good scapegoat.

Occasionally aged persons without teeth or dentures remain in good health, but they depend upon soft food. With the teeth decayed and mastication difficult, meat must be chopped or replaced by another source of protein.

Atrophy is the anatomical characteristic of the older muscle without giving the implication thereby that such muscles are inadequate to handle the average diet. Because there are certain somatic changes, it has often been assumed that there are correlating physiologic changes in the older system. Cerebrovascular sclerosis does not imply senile dementia, and gastric muscular

atrophy does not imply gastric dysfunction. It has been well

shown ( ) that the gastric musculature of the aged is capable of effective peristalsis and that gastric emptying time is not influenced by senescence regardless of gastric acidity. The gastric emptying time of a group of persons averaging over 79 years of age was 1.94 hours with a range of 1.33 to 2.75 hours. In the control group of younger persons studied, under identical circumstances, the gastric emptying time was 2.08 hours with a range of 1.03 to 3.08 hours.

#### Calorie Requirement

A moderately active person needs less in the way of calories than his younger counterpart. Between the ages of 20 and 59 years, 3,000 calories are considered adequate but over 60 years of age 2,700 calories are enough. ( 61 )

## AGE CHANGES IN DIGESTIVE SYSTEM

### Salivary Glands

In all the salivary glands there is atrophy, especially numerical and fibrous tissue. The cells lining the intermediate ducts show definite degenerative changes and may be the cause of the decreased volume of saliva. There are many large cells with acidophilic granules in their cytoplasm and pyknotic nuclei found in the salivary glands of elderly people. These have been termed "onkocytes".

In the aged, the concentration of ptyalin and the volume output of saliva are less and the specific gravity of the saliva is slightly greater than in the young adult. (105) The ptyalin concentration in the basal and stimulated secretion among the different individuals in the older group does not vary appreciable; greater variations do occur between individuals in the younger group. The basal secretion of ptyalin is quite constant in the individuals of both the old and young. The concentration of diastase in non-stimulated mixed saliva shows no significant change with age. The reaction of the secretion is either neutral or alkaline in patients without teeth.

In comparison to the 60 percent of the starch which is digested in the mouth, only 5 percent is digested in the old person. (109)

	Ptyalin Basal Secretion Units	Stimulated Units	S. P.	Volume
Young (average 25)	10.2 per cc	8.2 per cc	1.004	14.2 cc
Old (average 81) (107)	.30 per cc	.28 per cc	1.009	5.8 cc

#### Upper Part of Alimentary Tract

Senility brings not only loss of teeth but reduction of the lymphoid elements in the upper portion of the alimentary tract. The tongue is smoother as the papillae flatten. There is no important change in the esophagus except the general weakening of the musculature and a decrease in the resistance at the junction of the esophagus with the diaphragm.

#### Stomach

Contrary to what might be expected, there is an increase in the elastic tissue in the mucosa of the stomach and an increased amount of muscle tissue has been reported around the cardia and fundus. There is in general a loss of weight especially noted after the 70th year. Evidence of atrophic changes in the stomach are neither clear nor consistent and most of the information is in dispute.

The incidence of achlorhydria increases with advancing age. Vanzant's data (193) shows achlorhydria to be present in 17.4 per cent of females and 12.9 per cent of males. Although there is a greater number of females who have achlorhydria, there is a rela-



tively greater increase with age in ten aging males. The basal secretion of acid also suffers with advancing years. (107) Where at the age of 30 years the basal secretion of acid averages 54 units of acid, in the 70 year olds it averages only 34 units. When acidity alone is considered, there is evident a fall in males with age but not in females. The decline in the total amount of acid secreted is similar if both acidity and volume is taken into consideration. Histamine tests show a higher value for units of acid but the rate of decline is similar. (22)

The pepsin concentration in old persons is low. In the younger group it shows great variability and the range is between .1 and .03 mg. of tyrosine, but in old age the upper limit is about .02 mg. of tyrosine. (107)

There is a decrease in the irritability of the gastric motor mechanism concerned in hunger contractions of the stomach. The most apparent factor in the different age groups is that of the increased length of the periods of quiescence and the lengthened periods of activity and aging proceeds. The decrease in hunger motility of the stomach with age is due to the age of the muscle or motor mechanism or the decline in the quantity of chemical stimuli of the stomach in the blood stream. There is, however, no decrease in the emptying time of the stomach and in a case of achlorhydria, the time may be less than in younger adults. (191 & 192)

Pancreas

There is much controversy as to what happens in the pancreas in the aged. Histologically there are many changes, but it is not known which is physiological and which is pathological. There is, however, accurate information as to the secretion of the pancreas. There is no marked decrease in amylase but both the lipase and trypsin secretion are noticeably impaired. (106)

Using glucose tolerance as an indication of the amount of insulin secreted is a usually acceptable method, but its accuracy is questionable in old age. As age increases glucose tolerance decreases, but parallel with this tendency is a trend to diminution of insulin sensitivity. (75) In other words, the apparent decrease in secretion may be due in part to decrease in the sensitivity of the aging system.

The liver remains remarkable unchanged during the aging process. There is atrophy as evidenced by the decrease in the livers' weight but since the safety factor of the liver is large, there should be little or no evidence of hepatic impairment. Most of the changes, such as increase in connective tissue, are due to pathological changes.

In a study of liver function, Rafsky and Newman reported: (145)

1. I.V. Hippuric Acid Test - .21% showed abnormal function; .85 gm. in one hour was considered normal; Average age of group .94 gms. in one hour.

2. % Free Cholesterol - 44% or more was considered as abnormal; average for group 39.04 -- 32 % were abnormal.
  3. Total cholesterol - normal was considered 200 mgms. or less -- 66% were abnormal -- average of the group was 253.84 mgm. %.
  4. Oral Hippuric Acid - more than 2.5 gm. in 1 hour. Considered normal -- 6% abnormal in the group -- the average was 3.126.
  5. Cephalin Flocculation - 32% were abnormal.
  6. Bromosulphalein test - in 5 minutes 20% excretion was considered normal -- only 40% of group were normal -- the average for the group was 29.14%. In 30 minutes only a trace could be found -- 26% were found abnormal.
- 86% were found to be abnormal in at least one test.

#### Gallbladder

In old age the musculature in the walls of the gallbladder may undergo some hypertrophy and at the same time develop a tendency to sag, the result of lost elasticity and possible absorption of upper abdominal fat. The mucosa shows definite thickening. The rate of emptying the gallbladder is constant in females but in males is more rapid in the old age group than in young adults. There is no relationship between the size of the gallbladder and its emptying time. (155)

#### Small Intestines

The intestinal tract weighs less, not only because of loss of subserosal and submusosal fat, but also on account of atrophy of mucosa, lymphoid tissues and muscle. There is also some shortening of the intestines which is evidenced after the age of 40 years.

Using the galactose absorption method for measuring the intestinal absorption, it was found by Meyer, Sarter, Oliver and Nicheles that the milligrams of blood galactose rose rapidly in the young and quickly fell while in the older group there was a slight initial raise in thirty minutes which continued until sixty minutes passed then slowly fell. This points out the fact that the rate of absorption in old age is definitely slower in the older patients, but it is maintained for a longer period of time. (109)

#### Appendix

The lumen of the appendix shows a tendency to obliterate as age advances. This obliteration starts at the tip and gradually extends toward the base.

#### Constipation

Contrary to popular opinion constipation in old age is not increased to any important degree. Constipation in the normal ambulatory patient, with a well adjusted diet and adequate exercise, is not a common complaint. Indulgence in laxatives is frequently due to the impact of repeated advertisement, careless advice, or the misconceptions as to what the normal adequate evacuation is. (188)

( )

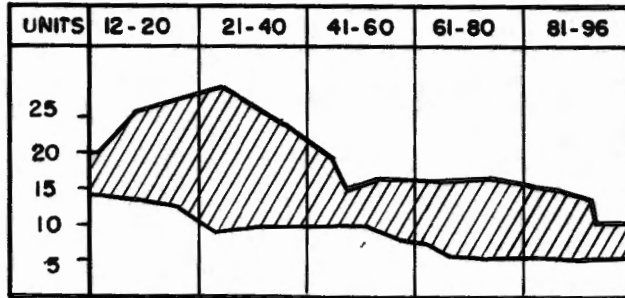
Where progressive vascular limitation occurs, ischemia results, so that, as in angina pectoris where the patient can be active within the limits of his coronary caliber, this type of patient can adjust his diet within the limits of his intestinal ischemia. In this pathological state small meals at frequent intervals is a necessity. Intestinal vascular sclerosis is a condition of the mesenteric blood vessels associated with old age and accompanied by disordered bowel function.

There is evidence of decrease in motility and thinning of the colonic musculature. These are slight and there is no evidence to show a decrease in the time taken by a meal to pass through the intestines. The complaint of constipation is, however, more frequent and should be treated as a complaint and not a physical sign.

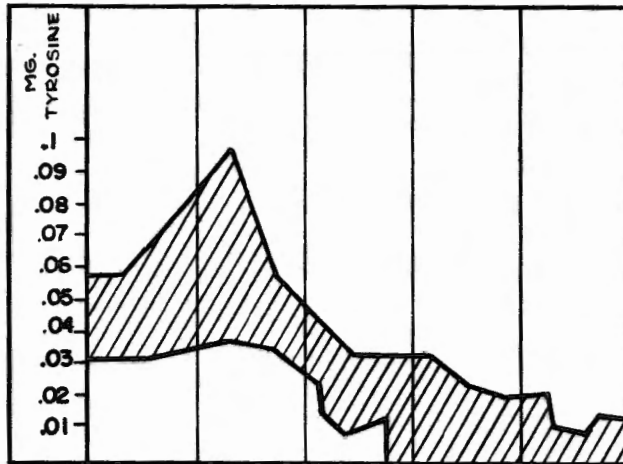
# SECRETION OF DIGESTIVE ENZYMES IN OLD AGE

106

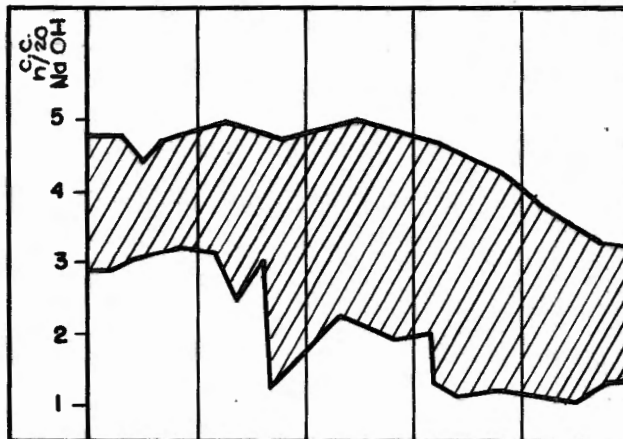
YEARS OF AGE



PANCREATIC AMYLASE



TRYPsin



PANCREATIC LIPASE

# GENITOURINARY SYSTEM

The changes encountered in the urogenital system in old age hold a minor position as far as causing death but are far more important in causing problems in nursing care and economic disability. Heart diseases and diseases of the digestive and nervous systems are not as disagreeable to the doctor, patient and relatives as those conditions causing incontinence, retention and frequency. The number of men suffering from urinary obstruction, the number of women suffering from menopausal and postmenopausal disorders and the incidence of prostatic hypertrophy and cancer and the incidence of carcinoma in the female genital system bear out the contention of the importance of those changes that do occur in this system.

#### PRIMARY KIDNEY CHANGES

The primary changes in the kidney brought about by the aging process is most often obscured by the secondary changes resulting from sclerotic alteration, of the arteries of the kidney. It has been conceded that this change does take place even in the face of vascular pathology but it itself is of very minor importance when the full reserve of the kidney is realized. Its' adaptive ability is impaired as shown by the results of McKay, McKay and Addis. (100) They found that the compensatory hypertrophy of one kidney caused by removal of the other was progressively less as the subject grows older.

#### Vascular Changes



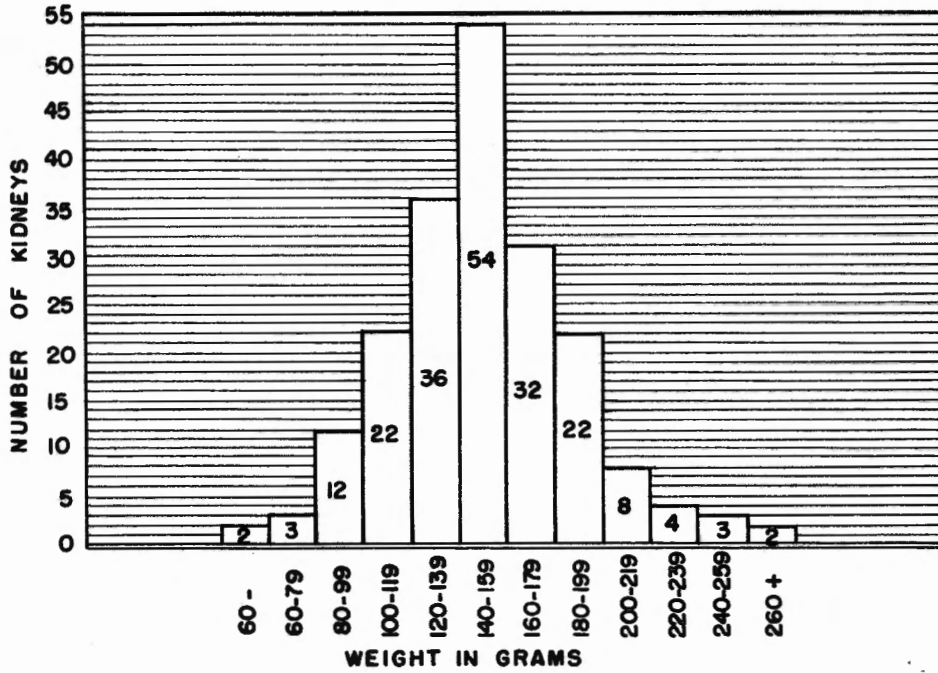
Just as there is no sharp delimitation between the "normal" and pathological changes of the vessels in the human cardiovascular system in old age, the effects of such changes in the kidney cannot be separated into these two categories with any degree of finality. The change inherent in the kidney is greatly overshadowed by the secondary change resulting from these vascular changes.

The large and middle sized arteries are frequently affected by sclerotic changes though the changes may extend into the arterioles. The alterations in the renal arteries are a result of a continuous change starting in infancy and ever increasing in degree with advancing years. One of the most striking anatomical expressions of this evolution is the duplication of the elastic fibrils and the development of a hyperplastic intimal thickening. The change in the terminal arterioles is of a simpler nature, consisting of a thickening and hyalinization of the vessel wall.

#### Secondary Degenerative Changes

The parenchymal modifications that follow the vascular changes are the result of disturbances in the nutrition of the tissues and may be considered examples of ischemic atrophy. The glomerulus is affected whether by collapse of its capillaries when the afferent arteriole is obliterated or by hyalinization which may extend into the tuft from the arteriole. By either process the tuft is transformed into a fibrous nodule and in the end may disappear. Quantitative examinations of the senile kidney have shown that in the seventh decade

POST MORTEM WEIGHT  
OF  
200 KIDNEYS FROM PATIENTS OVER 57 YEARS OF AGE



the number of glomeruli equals only two-thirds to one-half of the early adult count. (117) The ischemia due to vascular obliteration also causes atrophy of the glomerulus. The epithelial cells of the tubule decrease in size, and the tubule decrease in length and complexity and narrows to a greater or lesser degree. Tubular disruption occurs and the scars are filled with detached vesicles and cysts which lie among dense connective tissue.

#### Secondary Hypertrophic Changes

Not all changes are regressive, however, for there is hypertrophy of the proximal convoluted tubule. Hyperplastic lengthening of this segment may also increase its volume until it is twelve times as large as normal. There is no correlation between the size or even the pressure of the glomeruli.

Progressive change in the arteries takes the form of a growth and new development of vascular channels. Some of the new vessels will be seen to go directly to the intertubular network.

In many of the references read on the kidney in old age, there was a wide variation in the gross description of the "normal" kidney. Opinions varied from slight reduction in size to a small kidney not much larger than 60-90 gms. With the aid of Doctors Tollman and Myrabo of this hospital, I gathered data on two hundred kidneys from one hundred autopsy reports. (See Fig. 11) There was included no kidney that had signs of infection nor that was present in the face

of some urinary infection such as cystitis or perinephritic abscess. Kidneys with typical arteriosclerotic changes were included. The average age of the group was 66.4 years. The average weight of each kidney in the group was 158 grams. The average of the smaller of the pair was 145.25 grams and that of the larger was 165.5 grams. Using 150 grams  $\pm$  20 grams as normal, 62 kidneys were above normal and 38 kidneys were below. The most often encountered microscopic conditions were congestion, small fibrous scars and small cysts.

The change brought about in senescence shows little in functional disturbance but with the vascular change the picture is altered. The range of power of concentration of the urine is limited but well within the normal range. In 60 percent of the cases there is no albumin and most of the remaining show only a trace. Nycturia is a common complaint but seldom is significant. 75 percent of the people have no casts in their urine in later years and white blood cells are rare. The urea clearance that is 83% of aged patient's is normal. The nonprotein nitrogen level averages 30 mg % and the Phenosulfaphthalien test shows excretion of 27.8 in 15 minutes and 74.3 in 1 hours as an average. (97 )

In the aged subjects inulin clearance was reduced on the average to 60 percent of the average values observed in young adults. Average diodrast clearance in the aged was only 45 percent of normal. (162)

## Prostate

Obstructive prostatic enlargement in varying degrees is found in 4 out of every 10 men after the age of 60. (168) Occult carcinoma is found in 45 percent of the prostates of men over 65 years. ( 12) These two facts show the importance of the prostate in old age. In fact this is about the only time a man realizes he has one.

The age changes in the prostate which Moore (118) regards as normal are:

- a. Slight irregularity in the height of the epithelium begins between 40 and 45 years.
- b. Lobular atrophy begins between 45 and 50 years.
- c. The glandular epithelium loses its secretory activity between 50 and 60 years.
- d. Sclerotic atrophy first appears between 60 and 65 years.
- e. Atrophy of smooth muscle and relative or absolute increase of the fibrous tissue of the stroma is first seen between 60 and 70 years.
- f. Laminated corpora amylacea increase in number and size after 65 years.

There is a progressive increase in the size of the prostate with the greatest increase between the 20th and 30th year and a less important increase between the 60th and 70th year. Simmonds (163) gives these weights for the normal prostate; third decade, 15 gms., fourth 16 gms., fifth 17 gms., sixth 20 gms., seventh 23 gms., eighth 40 gms.

The outstanding characteristic of the senile gland is the variation in appearance of the same structure in different parts of the same prostate, in contrast with the uniform appearance of the adult prostate.

In the stroma, there is gradual atrophy of the smooth muscle fibers and relative or absolute increase in connective tissue. There is an increase of the collagenous tissue about each acinus. The smooth muscle fibers are smaller and pigmentation of the fibers is found. The cut surface is composed largely of stroma with discrete white, brown, or gray granules. Atrophy of the acinus usually involves the whole acinus without evident change in the stroma. At 80 years, more than one half of the acini are obliterated.

#### Male Genitalia

While the reproductive activity in the female ceases at the menopause, in the male this activity continues for a long time after the corresponding age in the female. Exner reported that in cases of men past 60, spermatozoa were present in 68.5 percent of men between the ages of 60 and 79, 59.5 percent between 70 and 89 and 48 percent of cases between 80 and 89. (53) It is probable that sexual activity decreases or is lost long before reproductive potentiality is lost.

With the cessation of spermatogenic action, there is at the

time an increase of the connective tissue of the tunica propria which causes a decrease in the size of the tubule. This may go on in the senile testis to complete tubular fibrosis and hyalinization. The spermatogenic cells change into small round cells of the type seen in undeveloped testis.

There is no evidence that an increased excretion of urinary gonadotrophius, such as is found in actual decrease in testicular secretion, is a constant finding in men. While a physiologic failure of the gonads, the male climacteric, has been described, it is by no means common and this failure is most often seen with some history of previous pathology.

The number of interstitial cells decreases from about the age of fifty. Pigmentation, fine golden brown granules, is found in the interstitial cells, efferent tubules, prostate and seminal vesicles. The presence of much free pigment in the lumenia of the efferent tubules, and its presence in some specimens of semen, suggest that the epithelium of the tubules have an excretory function.

#### Female Genitalia

The vulvae, being principally skin with accessory structures, show changes similar to those of other cutaneous areas. There is a loss of elasticity and firmness with a tendency to laxity and wrinkling. The skin becomes thinner and there is a diminution of

of hair and more or less atrophy of the accessory glands. The subcutaneous tissue is also less firm and increases the flabby appearance. The clitoris tends to atrophy, although it occasionally becomes more prominent, the introitus usually becomes smaller and the membrane thinner. Where there has been previous birth trauma, the parts may gape and the vaginal walls roll out. The microscopic appearance is that of a thinned epidermis and dermis with diminution in elastic fibers, atrophy and relative paucity of the glands. The vulvovaginal glands also show evidence of atrophy.

#### Vagina

The vaginal lumen tends to become smaller, especially in virgins and multiparae. The surface loses its folds and has a smoother appearance, the epithelial layer is thinner, the elastic and muscular fibers decrease in number, but the connective tissue increases. The blood supply diminishes due to obliteration of some vessels which show hyaline degeneration in their walls.

#### Cervix

The cervix as a whole atrophies. The epithelium of the portio undergoes changes similar to those of the vagina, the muscle fibers tend to be replaced by connective tissue, and the vessels diminish in caliber and number. The epithelium of the endocervix and of the glands loses its cilia, becomes flattened



and its' secretory function diminishes. There may be stenosis of gland ducts and of the cervical canal which, on one hand, leads to retention cysts and, on the other, to some obstruction of the canal with retained secretions and dilatation above. This may lead to pyometra.

#### Uterine Body

The retrogressive changes in the corpus uteri begin with the menopause and continue throughout the remainder of life. The uterine wall atrophies and becomes thinner due to a diminution in the muscular elements, and the vascularity decreases. The size of the cavity lessens, the mucous membrane thins, and the epithelial and glandular elements diminish in size and number, and the stroma with its vessels becomes atrophic.

Microscopically, the vascular lumina are of smaller caliber and the walls become hyalinized. The uterine walls are compact and consist primarily of connective tissue. The mucous membrane becomes thin, avascular connective tissue stroma with sparse, irregular and small glands lined with low epithelium, which is also distributed over the endometrial surface. Small areas may be completely denuded of epithelium and if such areas are in apposition, adhesions may occur. At times, such adherent surfaces may cause almost complete obliteration of the uterine cavity.

Kosmak ( 90) denies that atrophy alone is characteristic of

the aged uterus. Less than one half of the cases he studied showed the atrophic changes usually looked upon as typical of senile endometrium. 35 percent showed proliferation. This finding is no doubt connected with the well known fact that estrogen may be produced long after the menopause. The source of this postmenopausal estrogen is not definitely known, though it seems reasonably certain that it is not in the ovaries, but rather in some of the other endocrine glands, probably the adrenals. In this case, postmenopausal bleeding is based on a hormonal factor as before the menopause.

25 percent revealed a histological picture, which represents a retrogressive hyperplasia. It is characterized by a swiss cheese pattern; but with obvious evidence of retrogressive changes and inactivity, such as fibrosis of the stroma.

#### Fallopian Tubes

The fallopian tubes reflect the changes in the uterine body and with atrophy of the endometrium the walls become thinner and the lumen almost disappears. The plicae are smoothed out and the epithelium loses its cilia and changes from a columnar type to a flattened almost endothelial layer.

The round and other ligaments also atrophy and lose much of their muscular and vascular structure through degenerative change.

## The Ovary

There is no abrupt change in the pre-menopausal as compared to the early post-menopausal ovary. (73) In general, the aging of the ovary takes place in an orderly and progressive fashion resulting in a gradual loss of primordial ova and their follicular phases, a progressive thinning of the ovarian cortex which becomes increasingly wrinkled or gyrated, and a relative increase in the medulla which becomes the repository for corpora albicantia. The vessels of the hilum and medulla become progressively sclerosed.

When the menopause is fully established, the ovary then becomes a relatively static, although by no means an inactive organ. A post-menopausal patient may experience a second menopause if her ovaries are removed surgically. Morphologically this is due to the presence of active appearing cortex which may be present either as a thin regular layer beneath the germinal epithelium or as an irregular scalloped zone of cortical stroma about the periphery of the organ. The more active such ovarian cortices are, the more apt the patient is to have evidence of endometrial activity.

The increasing cortical gyration of the aging ovary, which usually begins in the early thirties, is often accentuated by areas of cortical stromal hyperplasia. In the depth of the sulci between the gyrations, there is undistributed germinal epithelium. Here the latter is cuboidal or even medium columnar in type, whereas on the surface of the gyri it is thin or even absent.

After the age of 50 there is a striking loss of all phases of follicular activity. Only rarely is there encountered evidence of follicular activity, promordial or otherwise. There are, however, the infrequent patients whose ovaries resemble those of the younger group. Evidence of cortical stromal hyperplasia is more apparent in the group.

There are structures in the cortex termed cortical granuloma. They appear to be estrogenic in that their associated endometria show more activity than the age group would justify. (55) They appear to start out as a single relatively large polyhedral cell or small group of such cells resembling the theca lutein type. They contain neutral fat as do the latter variety of cell. During the evolution of this structure, it becomes vascularized, attracts lymphocytes and finally ends up as a focal collection of multinucleated giant cells containing needle like clefts. More or less lymphatic response still remains about the periphery.

#### Breasts

After menopause, the breasts gradually lose their capacity for physiological activity and are subject to morphological changes. They consist of cutaneous and accessory structures, the subcutaneous tissues, chiefly fat, and the mammary ducts and glands. The skin changes are similar to those of other surfaces. There is a tendency for the subcutaneous fat to increase after the menopause. The breasts become larger and more pendulous, which increases the

intertrigo below the sagging breasts. At more advanced ages the adipose tissue may be absorbed and the whole breast structure become loose, flabby and pendulous.

The glandular tissue reverts to the type of lactiferous ducts lined with several layers of epithelium. The acinous structure may persist. The normally large amount of elastic tissue is markedly reduced and the collagenic connective tissue cells come to predominate.

ENDOCRINE SYSTEM

The amount of work and its' quality that has been done upon the endocrines in old age is surprisingly sparse and poor. Of course there are numerous factors inhibiting any progress along this line. The main one is the fact that the functions and relationships of these glands are by no means understood. Certainly it is of very little use to attempt to find what happens later in life if very little is known about the organs as they function in their prime.

Aging is only one of the factors that has influence on the endocrines and, from how the work that has been done, a relatively minor one at that. Heredity, nutrition, infection, blood supply and the interrelationships of the glands all produce notable changes both in structure and function.

## HORMONES AND AGING

Hormones may accelerate and intensify the earlier phases of growth in organs and tissues, but the subsequent phases in the time curves of these organs and tissues which lead to the retrogressive changes of old age, tend to undo these effects and to enhance the degenerative effects of the hormones. The popularly sought rejuvenation by the administration of hormones is of a temporary effect only and may be followed by a reversal of action. A hormone may thus exert various effects on the different phases of the "time curves" of the organs and tissues which constitute the organism. What kind of effect predominates will depend upon the amount of the hormone given, upon the phase of the time curve which the organ or tissue has reached when the hormone is administered, those effects having a better chance to prevail which tend normally to take place during this phase, and lastly, upon inherited characteristics of the individual on which the hormone acts. In general, the processes which are in accord with the inherited constitution of the tissues have a better chance to be promoted by the hormone than others which are not in accord.

While hormones affect directly only certain constituents of the body, indirectly their influence may extend to a greater variety of organs and tissues. However, as Loeb (98) says, there is no master hormone which directly controls senescence as a whole and duration of life. Hormones may increase the function of an



organ and give it a more youthful appearance, but the same hormone may affect different tissues and their metabolic processes in different ways, and what will be an adequate stimulus for one tissue may be unsuitable for another. It seems impossible, by means of growth stimulation, to control adequately that finely adjusted interaction of the various tissues and organs which result in the harmonious functioning of an individual.

## ADRENALS

### Anatomy

A decrease in size of the adrenals is noted in old age. This decrease in size affects both width and thickness, but not the length of the organ and is not associated with a loss in weight. (65) This increase in specific gravity is not accompanied by any gross pathological change (99) and indicates a loss of water and an increase in the solid matter. Thickening of the argentaftin reticulin is the outstanding microscopical alteration, in addition to the somewhat smaller size of the component cells. There seems to be some decrease in the output of the gland but there is no definite proof. (152)

There is also an increase in the amount of collagenous stroma with added hyalinization but without evidence of inflammation. The interstitial and parenchymatous changes connote a disturbance of hydration in which water passes from the intracellular to the extracellular phase.

### Function

The urinary excretion of ketosteroids provides a means of determining whether there is a decrease in the function of the adrenal. The average 17 ketosteroid output is 14 mg. of which 9 mg. are contributed by the adrenal cortex and 5 mg. by the gonads. (180) Fraser, et al. (59) found that in the age group

71-75 years that the 24 hour output varied from 1.8 to 4.8 mg. This shows that the sterol output of the cortex is probably diminished even if the testicular secretion has completely ceased. A decrease of 17 ketosteroid excretion in women well over the age of 60 years was shown by Hamblin et al. although during menopause there was a rise. (69)

(78 & 79)

Haskins/ noted a definite increase in physical activity after administering estrogens to rodents. It is possible that the reactions were referable to the cortico-mimetic effects of estrogen. Stimulation of the adrenal cortex by the administration of estrogen might have sufficiently increased the discharge of cortical hormones to permit increased activity of the aged animal. This is further bórned out by Ingle ( 83) who noted that in adrenalectomized rats adrenal extracts and synthetic compound would maintain work capacity while the estrogens would not.

## PITUITARY GLAND

### Anterior

Although there is much conflicting data on the weight of the pituitary in old age, the majority agree that there is a slight loss of weight after the age of 40. ( 35) There seems to be evidence that the decrease in weight is greater in females and in some cases there has been an increase in weight observed in males. This can not be construed as proof of involution of the gland.

Microscopic changes in the gland are numerous and again the subject of much conflicting material. It seems well established that there is an increase in the number of basophilic cells. (41) The acidophiles are large and fully granulated. There are many cells in which mitochondria are large and abundant. Many cells show a greatly enlarged Golgi apparatus. These cytological features are evidence that acidophiles are in the state of active elaboration and discharge of their secretory products.

The basophils are numerous and large and are not as vacuolated as they are in the younger group. They show a wide variation in coloring due to a variable content of mitochondria and basophilic granules. All of these cytological features indicate a high state of activity both in production and elaboration of secretory products.

The above description is for that of the female gland and is

in sharp contrast to that of the male. In the old man there is the increase in basophilic cells but it is a relative increase. There is a reduction in both the size and number of granular cells. The basophils are filled with vacuoles. The chromophobes are much more plentiful. The cells have lost their normal compact arrangement and are widely dispersed by increasing amounts of connective tissue. The cells appear inactive and have little to indicate that secretion is taking place.

There are many unanswered questions in the study of histology of this gland that must be answered before any conclusions can be drawn.

#### Posterior

There is an invasion of the posterior lobe of the pituitary by basophilic cells that roughly parallels the increase in age. There is also note an increase in pigmentation.

#### Secretions

The amount of gonadotrophic hormone in the pituitary gland varies widely with age and sex. Women over the age of 45 have the greatest amount in their urine, 1000-4000, rate units, while men of the same age group follow closely with 100-4000 R. U. This is compared with the amount in pregnant women which reaches 200 R. U. at the most. (202)

The absence of significant differences in thyrotrophic and

chromatophorotropic contents in all age groups is not surprising in view of the importance of these hormones for the metabolism of all ages. There is, however, an equally constant secretion of the lactogenic hormones which is not at all expected if the hormone has only one function.

## THYROID

The available material on the activity of the thyroid gland in old age is very inconclusive and extremely contradictory, although much work has been done. The following are the arguments for a decrease in the function of the gland.

1. The slow but gradual reduction in the basal metabolic rate in otherwise healthy patients after the age of 20 to 25. (96)

Age	40	50	60	70	80	90	100
Calories Sg.							
Meter Hour	36.4	35.7	35.	34.4	33.7	32.6	31.9

2. There is a resemblance between the impairments appearing in the skin and hair with advancing age and those induced by varying degrees of hypothyroidism. (47)
3. Marked hypothyroidism sometimes produces a lowered resistance to infections. This is seen with old age in certain types of infections.
4. The thyroid attains its maximal normal size at the age period of 15 to 20.
5. According to Cooper (41) the thyroid is reduced in size, the follicles and cells are smaller, the vascular supply is reduced, there is much less mitosis seen in the epithelial cells, the colloid is absent from many of the vesicles, and the colloid, when present, appears less dense. McCarrison (35) states that the human thyroid

after the age of 40 shows less colloid, sclerosed blood vessels, and increased connective tissue. Dogliotti and Missi-Nuti((49) report that the follicles are reduced in size and colloid content as well as showing fewer follicles. Hertzler (74 ) found in old age an increase in the basophilic properties of the colloid, some abortive attempts of the follicles to reproduce themselves, and changes involving the connective tissue. In general he regards the changes seen as frankly regressive.

There is some evidence indicating a reduced amount of iodine per weight of gland in the human thyroid.

The following are arguments in favor of the increased secretion of the thyroid in old age.

1. The decreased basal metabolic rate may be due to the decreased amount and efficiency of the body tissues, the increase in antihormones, perhaps from the adrenal cortex, or a decreased sensitivity to the thyroid hormone. The muscle tissue, as will be further discussed, undergoes a great degree of atrophy and fatty degeneration that is not reversed by the administration of thyroid and in the measurement of the metabolic rate the muscles play a major part.
2. While the hair on the skin shows degenerative changes that of the nares, auricle and eyebrows increase in amount and thickness. (146) Administration of thyroid substance



reverses the course of hypothyroid changes but not those of old age.

3. Many other factors can be logically blamed for this increase in susceptibility to infection.
4. There is no evidence that reduction in size means a reduction in hormonal secretion.
5. The histological picture of the senile thyroid, according to Dogliotti and Nizzi-Nuti ( 49), shows that this organ functions more actively in normal old age than in earlier adult years. In addition to the changes already described they found an increased number of granules. These changes, they conclude, demonstrate an increased activity of the gland and more intense reabsorption of the colloids.

Garau ( 4 ) found evidence of increased activity in old age in the thinner colloid and in the numerous follicles with high epithelial cells. This increased activity he considers as compensatory to the degeneration of follicles elsewhere.

In testing the activity of the thyroid gland, Vogeler ( 4 ) used a dried powder of the gland to induce transformation in the tadpole. By this method he found an increase in the activity of the thyroid in old men, and no apparent decrease in women.

No matter which view is taken, the administration of thyroid substance has no specific action in old age. When it is found to have real value in the aged its action is probably non-specific. The very aged may feel better temporarily after any stimulating therapy.

## Female Hormones

As has been mentioned previously, the ovaries cease to exert any influence upon the human system that is measurable, although there is continued ovarian activity long after menopause.

Under the section on the pituitary it has been shown that there is an increased secretion of the gonadotrophic hormones. Immediately after menopause there seems to be estrogenic activity in the adrenals.

## Male Hormones

The life span of the testis is longer than that of the ovaries and their decline is more gradual. The hormone production mechanism is more gradual. The hormone production mechanism in the testis, the factor involved in libido and copulation, appears to fail earlier than the spermatogonia and the sperm producing factors. At least in individual instances in the human male, sperm production has been described beyond the ninth decade of life.

OTHER SYSTEMS

## MUSCULAR SYSTEM

Of all the systems we have studied there is none in which the changes wrought by age point to the need of more investigation into the basic causes of aging. With this system we see some of those things that have been considered as a part of the aging process, but also the absence of some of the other.

### Smooth Muscle

Smooth muscle seems to be unaffected by age. Investigation conducted on the musculature of the human caecum found no age changes in this tissue. It also has been found that smooth muscle has the ability to regenerate despite the absence of mitosis and of amitosis.

### Striated Muscle

#### Changes in Anatomy

Myofibrils in the aged muscles appear in oblique, spiral and even annular orientation in addition to those in the parallel. The reticular sheathes of the fibers show no change. The collagenous fibers, pigmentation, and elastic fibers increase with old age. Fatty infiltration and brown atrophy occur as age advances.

X-ray studies show that the muscle shadows in persons over sixty are reduced in bulk and density. The muscle atrophy does not extend to the muscular attachments which are not affected by

age except that there is some bone hypertrophy and atrophy at the attachment. This gives a roughened appearance to the bone at the attachment site which increases with age.

#### Changes in Function

Measurement of muscle power seems to be a logical way to determine the functional loss occurring with age. There is a decline of muscle power in the aged. This decline is greatest in the biceps and muscles of the back. In the biceps, the strength declines to 54.1 percent at 65 of what it was at 35. The strength of the back muscles decline to 64.3 percent in men. In women the decline is even greater. The power of sustained effort appears to dwindle during later life in approximately the same manner as maximum power.

In strength, and speed of action and dexterity, however, there is surprisingly little decline in old age. There is no regularity in decline. The oldest often show a better score than younger persons.

These facts show a definite decline in muscle strength, but the structural changes are not as great as those of strength. There is evidence that the cause lies in the governing mechanism rather than in the structure. The Purkinje's cells of the cerebellum tend to disintegrate and disappear after 40 years of age and pigmentation appears in the dentate nucleus. Such changes in-

dicates that the nervous system may be responsible for those changes above described.

#### Changes in the Vessels of the Muscles

Throughout the body arteriosclerosis is seen as a factor in the process of aging but not in the vascular system of the muscles. ( 5 ) Although there is a small increase in the amount of fibrous tissue in the media of the vessels of the muscles, no intimal disease has been found at any age.

#### Changes in Chemistry

The chemical content of muscle shows an increase in water, chlorides, total base, sodium and calcium and a decrease in potassium, magnesium, phosphorus and ash. The increase of water is intercellular. ( 44 )

## INTEGUMENTARY SYSTEM

The first noticeable evidence of the onset of old age is usually seen in the skin. Although cutaneous changes appear insidiously and do not affect all persons uniformly, still the face of an individual is a fairly true index of his years. The changes brought by advancing years are in the turgor, color, thickness, smoothness and contour of the skin which results from the fact that as a normal process the specialized cells of the skin, rather than its connective tissue framework, are affected by age.

Heredity governs the extent, the rate of development, and the particular age at which skin changes become manifest. Whatever the traits, the conversions are inescapable and eventually the skin becomes inelastic like parchment, loses its velvety thickness and wrinkles develop from degeneration of the elastic tissue, muscle fibers and subcutaneous fat. With age, the skin gradually becomes more flaccid and less elastic and later thin, dry and harsh in texture. As the atrophic process progresses, the skin assumes a glossy appearance, the underlying blood vessels become visible and the color is tinged with a muddy yellow. These transformations are all of an involutional character and are followed by the appearance of degenerative changes, of which the first to develop are the pigmented spots so frequently observed on the face and backs of the hands. Mingled with them may be tiny atrophic white spots and small clusters of dilated blood vessels, the bright red senile angiomas.

The changes which take place in the hair are a striking, if not reliable, sign of advancing years. The tendency for the hair to whiten or to thin out even until complete baldness, particularly in men, is usually associated with old age, although it may occur prematurely. Conversely, it is known that the members of some families retain their hair in its natural state and color in extreme old age. As age progresses the hairs on the skin become thinner and less vigorous while the hairs of the nostrils and ears in contrast grow longer and stiffer, much like bristles. Often the eyebrows become bushy. In elderly women the hairs on the upper lip and scattered hairs on the chin increase in thickness in thickness and growth. Similarly hairs in moles tend to grow longer, and the moles themselves become more pigmented.

With age the nails become harder, thicker and brittle, often exhibiting longitudinal ridges and lacking in the luster of nails of a younger person.

A skin wound of 40 sq. cm. in a person twenty years old heals on the whole in forty days. The same size skin wound in a person forty years old requires for healing about eighty days. At the age of sixty, the same skin wound requires five times longer to heal than in a child of ten. (176)



## SKELETAL SYSTEM

In the bone the organic matter wastes, leaving an excess of mineral matter and causing the bone to become more brittle. Because of bone atrophy, the weight of the skeleton is reduced. The waste of the organic matter in the heads of long bones and the pelvis produces a spongy condition, or osteoporosis. The neck of the femur is depressed until the angle formed nears a right angle. Flat bones may become porotic and brittle. Bone marrow becomes gelatinous, harder, denser, and contains more fat than that of younger persons.

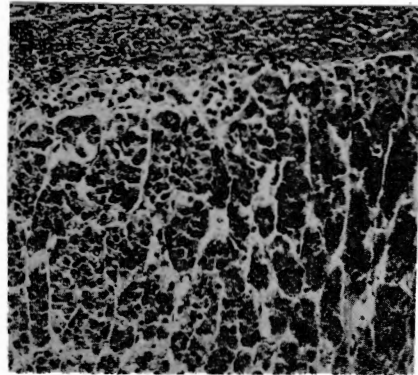
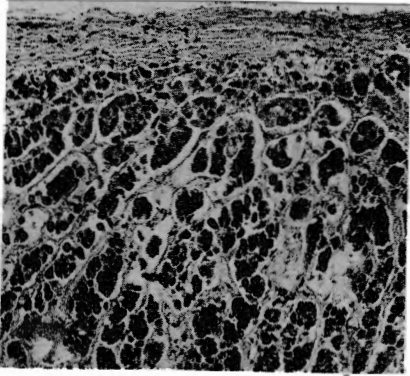
Marked bone changes appear in the skull, spinal column, thorax, pelvis, femurs, and feet. The cranial bones become thin and the mandible shows pronounced osseous waste. Some bones, especially the frontal, show hyperostosis. The chin becomes more pointed, and loss of teeth and absorption of the alveolar process, with general atrophy of bone, tend to make the face wizened.

The spinal column becomes shorter and curved and the intervertebral disks become contracted and thinner. There is, consequently, a loss of height. These processes begin about the fiftieth year and later on there is calcification of the disks. Vertebrae almost always show the presence of bony spurs which are often misdiagnosed as arthritis of the spine. Besides the calcification of cartilages, there is also noted flattening and bowing of the vertebrae and changes in the shape and size of the thoracic

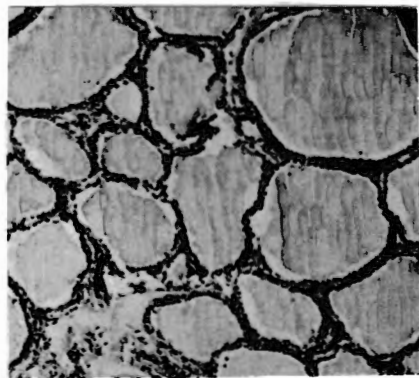
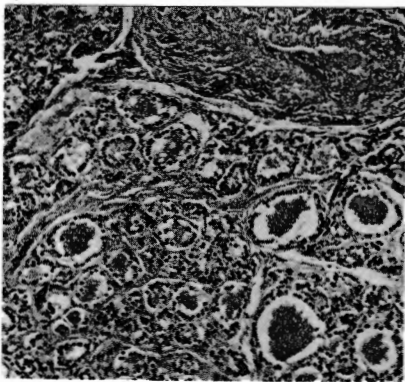
cage.

Cartilage of joints either atrophies through insufficient repair, becomes fibrillated and wastes through attrition, or becomes fibrotic and osseous. Costal cartilages suffer loss of elasticity and become calcified. These cartilages are brittle and are easily fractured. The synovial sacs become stiff and hard. These changes in the structure of the joint cause a stiffening, with diminished mobility. The cartilages may be gone completely and the aged use the bony surface of the joints in locomotion.

# ADRENAL



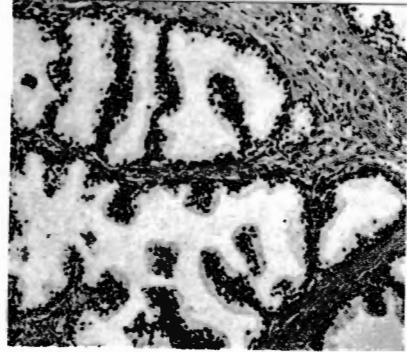
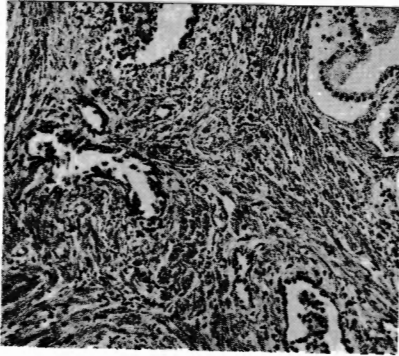
# THYROID



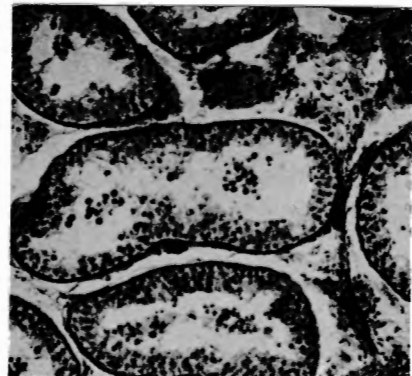
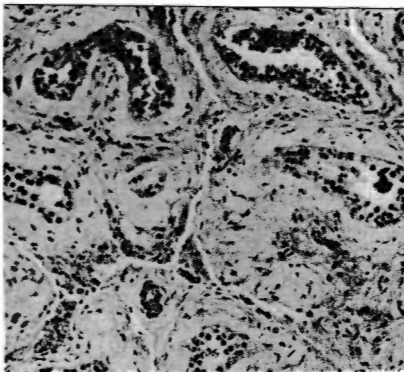
The rows of cortical cells of the 72-year-old organ (right) are narrowed due to the atrophy of the cells and there is an accompanying increase in the delicate connective tissue framework which contrasts with plump cords and scanty framework of the organ at 22 years (left).

Atrophy of follicles with scanty colloid and heavy interstitial fibrosis in the 70-year-old organ (right). There is also an extreme sclerosis of the arteries to the organ. The contrasting figure shows the organ at 25 years (left).

# PROSTATE



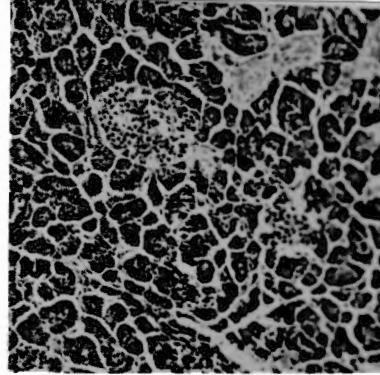
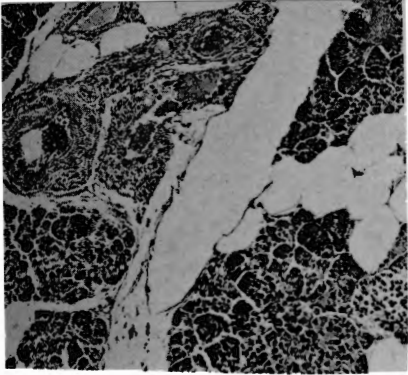
# TESTIS



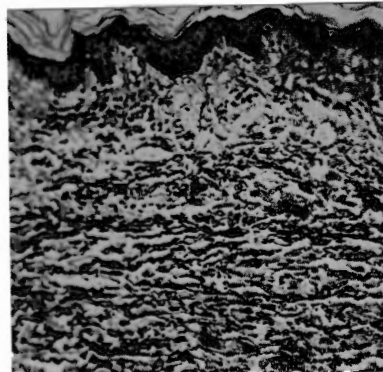
The uncomplicated senescent change of atrophy, not hypertrophy, is seen in the shrunken glands of the 78-year organ (right). The interstitial tissue is at least relatively increased in amount. Note not only the large size of the gland acini in the 22-year-old organ (left) but also the high epithelial cells which line them.

There is an extreme interstitial fibrosis with so great thickening of the basement membrane of the tubules in the 72-year-old testis (right) that the lumen of the seminiferous tubules is greatly reduced. Contrast the shrunken atrophied spermatogenic cells with their pyknotic nuclei and the absence of free spermatozoa with the proliferative activity in the tubules of the 20-year testis (left). Between the tubules of the senile organ are seen persisting interstitial cells.

# PANCREAS



# SKIN

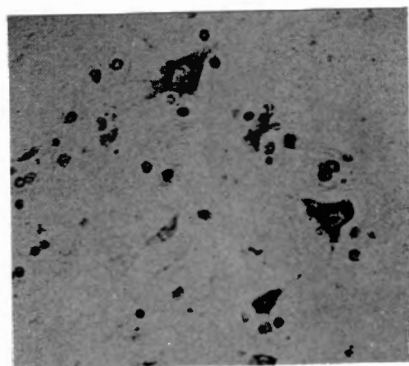
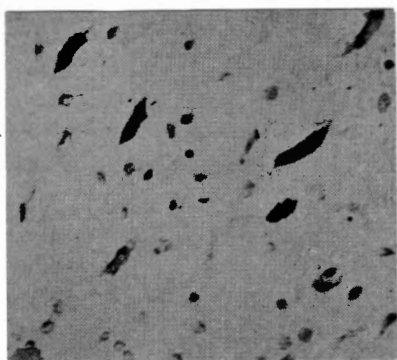


Some decrease in size of both secretory acini and islands of Langerhans is seen in the 75-year organ (right) as compared to that of 20 years (left). The latter are commonly reduced in total number in old age. More striking, however, is the great thickening of the arteries and the infiltration of the framework of the organ with fat.

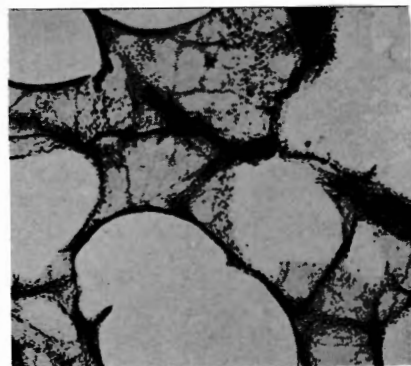
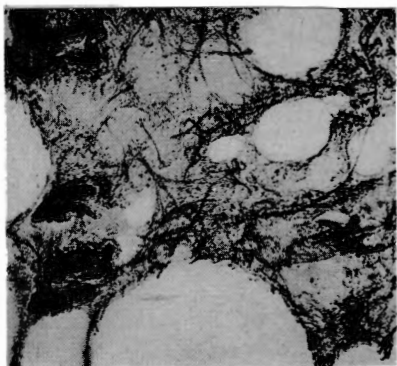
The most striking change in the 73-year old skin (chest) (right) is the disappearance of the black-stained elastic fibers from the deeper layers of the dermis as compared to the great number seen in the 22-year example (left) from the same region. There is also atrophy and disappearance of the papilla, though the epidermis shows no thinning in this example.



# BRAIN



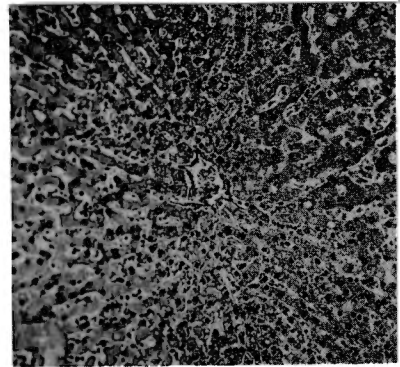
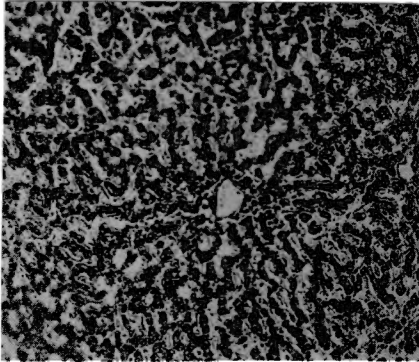
# LUNG



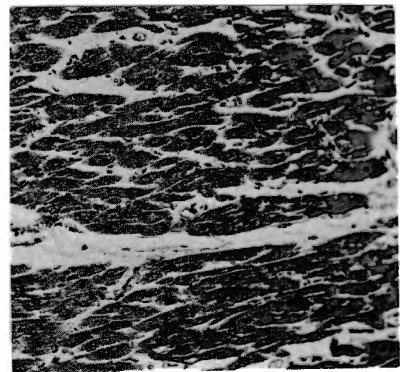
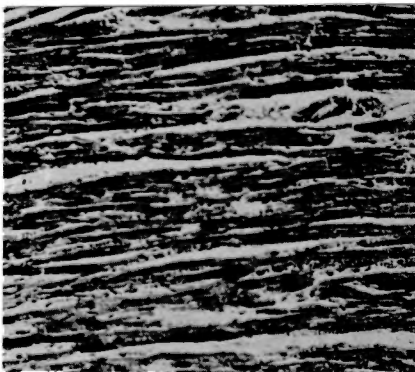
Atrophy and pigmentation of ganglion cells in the deeper layers of the cerebral cortex in a 78-year old brain (right) as compared to those of an 18-year old individual (left). The other prominent lesion of senility, proliferation of glia, is not shown in the figure.

The disruption of the atrophic elastic tissue framework in senile emphysema of the lung of 65 years (right) contrasts to the thick well-preserved pattern of the elastic fibers in the lung of 18 years (left). It will be noted that the degree of strain on the two sets of fibers must have been about equal as the air spaces are equally distended.

# LIVER



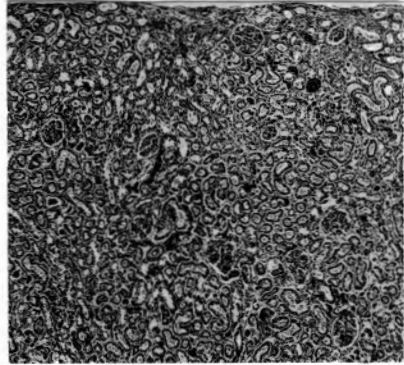
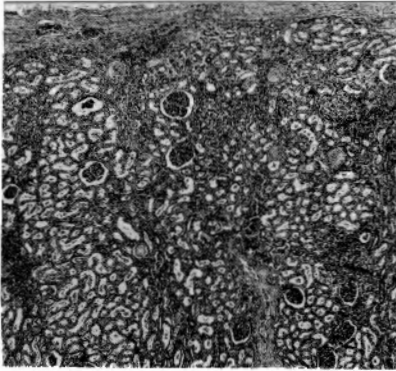
# MUSCLE



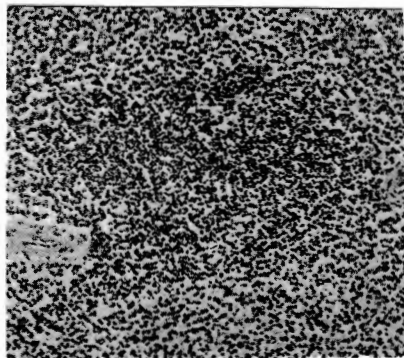
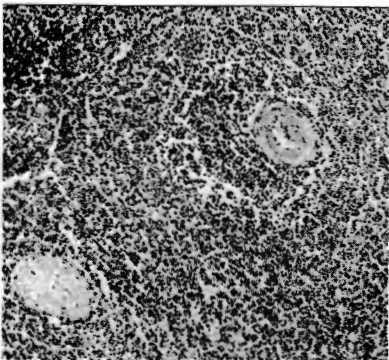
Little architectural change is observed in the 72-year liver (right) as compared to the 20-year organ left. There is a small reduction in the size of the liver cells and therefore in the breadth of the liver cords which radiate outward from the central vein. There is little or no increase in the connective tissue framework of the organ.

The reduction in size of the atrophic muscle cells of a 75-year heart (right) compared to those of an 18-year organ (left). In this form of "simple" or abiotrophic" atrophy there is little connective tissue proliferation. The hearts of most old individuals also show focal areas of fibrous scarring due to coronary sclerosis.

# KIDNEY



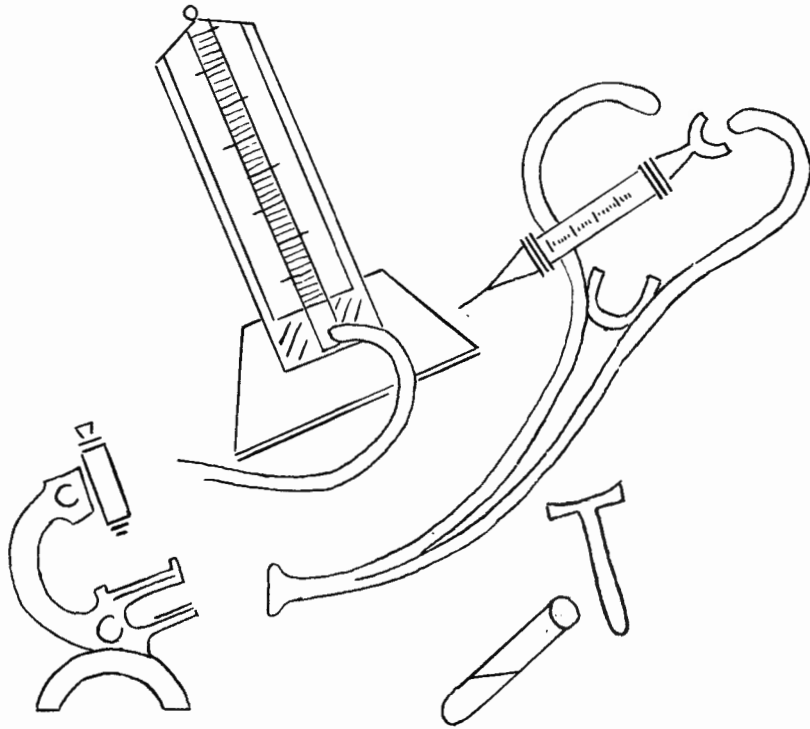
# SPLEEN



Senescent change in the spleen consists of atrophy of the lymphadenoid tissue with little increase in the connective tissue stroma, combined with a sclerosis of the terminal arterioles. Contrast the large Malpighian body of the 20-year spleen (right) with the small follicle of that of 60 years (left). Note the thickened central artery of the latter. Senescent arterial sclerosis occurs early in this organ and is not therefore necessarily indicative of and equally severe general arterial involvement.

The structural change of old age in the kidney is largely the result of senescent arterial sclerosis. Note how the even pattern of the 25-year kidney (right) is broken up by the large "arteriosclerotic scars" in the 70-year organ (left). Many well-preserved glomeruli and tubules persist, however, so that there is seldom clinical evidence of renal insufficiency. REF. 107-176

# DIAGNOSTIC DIFFERENCES



## DIFFICULTIES IN CLINICAL DIAGNOSIS

The cardinal goal of all medical science, whether it is experimental laboratory study, or bedside application, is not to define, or even to cure disease, but to return the patient to the maximum level of health possible. Diagnosis exists solely for the purpose of guiding treatment, so that this maximum level may be obtained, and not for the mere academic differentiation of pathological entities. Too often there has been more concern with the disease than with the patient who has the disease.

In order to facilitate further discussion, the classification of Stieglitz (178) will be used. His article gives four principle groups of difficulties encountered in old age especially when a clinical diagnosis is made. These are: difficulties arising out of the characteristics of diseases, difficulties arising out of the characteristics of the patients in which these disorders occur, difficulties arising out of inadequacy of present clinical methods and difficulties inherent in the process of diagnosis itself.

### Difficulties Inherent in the Disease

The first and most obvious consideration under this heading is the difference between the disease typical of youth and the degenerative disorders of old age. In the earlier years, the etiology is usually exogenous, obvious, specific (a single disease process) and of recent origin. The diseases of later years are usually endogenous



and occult in origin. They are cumulative and multiple as well as of long duration. (177)

The symptoms in youth are outstanding and easily recognized and deviations from the norm. The diseases in old age are more insidious and asymptomatic. Those diseases in youth are acute and often self limiting, giving immunity to another attack. This is in contrast to the chronic course, progressive disability, and increasing vulnerability typical of diseases in the aged. Then, too, the aged show great variation from individual to individual while this is not true in the earlier years.

These attributes make detection of the etiology much more difficult than in the florid and obvious infectious disorders of youth. It is most important to recognize that in each individual instance of a given disorder the disturbance results from a series of superimposed factors, each adding insult to injury, and that these factors are not necessarily identical in any two cases. Causation is never singular. Medical thinking is just now recovering from the retarding influence of the concept of "specific" etiology which came into being with the birth of bacteriology. Appreciation of the importance of other causative factors was delayed by the zealous advocates of specificity. With the excessive concern over the nature of the seed, it was largely forgotten that seed will not grow unless it falls on fertile soil.

Causative influences may be divided into three categories;

predisposing factors, provoking factors and perpetuating factors. The breakdown of etiology into these three types of factors has proven extremely helpful in understanding the ways and means by which the disease comes about.

The importance of comprehending causation as fully as possible cannot be over emphasized. Effective treatment, whether preventive or curative, is predicated upon thorough removal of etiologic factors. There can be no lasting therapeutic result if causation is neglected.

The second difficulty inherent in disease is the co-existence of multiple disorders, so common in senescence, introduces another most significant obstacle to accurate diagnosis. When the physician is faced with the diagnosis of disease in the senescent individual he must anticipate the presence of several disorders. If the illness is an acute one, he must still assume that there probably existed some chronic impairment prior to the onset of the acute illness. Thus, he is habitually faced with the question of deciding which of the physical signs and symptoms are due to the new disorder, and how many or how much to pre-existing disturbances. (66)

The whole group of disorders which have been called, "degenerative" overlap one another. They represent a single common denominator in the fact that all of them interfere with the nutrition of the parenchymal cells through changes in the inter-cellular matrix. Applying nutrition in its' broadest sense and

realizing that the "internal milieu" is the medium through which the cells are maintained, it is not difficult to appreciate that impairment may result from any one or more of several factors. These are inadequate nutritional supply, inefficient distribution, ineffective utilization of food elements and accumulation of injurious metabolic debris. (14)

The individual variations in the distribution and intensity of the lesions of arteriosclerosis emphasize the importance of individualization in diagnostic study of senescent individuals. Variability undoubtedly depends in some way on the fact that each individual has been subject to a different series of insults and injuries in different sequence. The cumulative effects of the innumerable intoxications, transient infections, physical and physical trauma which constitute the vicissitudes of our existence, probably play a major role in determining the vulnerability of the different tissues. We start very much alike, but soon differ. (119)

The third major source of confusion intrinsic in the character of the diseases is the frequently insidious onset of the disease and their essentially asymptomatic course. It should be stressed that none of these diseases present any truly primary subjective symptoms. We have no kinesthetic sense which informs us whether our blood pressure is above or below normal levels. There are no sense organs to signal fluctuations of the concentration of glucose in the blood nor any sensation in the arteries

of the brain to warn us of cerebral arteriosclerosis. As a matter of fact, the retarded cerebration of the senile arteriosclerotic brain would tend to prevent awareness of any changes in mental vigor and acuity.

Perhaps the subjective silence of these disorders is an asset. That which we do not know, does not hurt us. Certainly much distress is avoided by the absence of symptoms early in the course of these degenerative disorders. But, unquestionably, the lack of early symptoms is a curse as well as a blessing, for it definitely delays discovery, and therefore often retards the institution of treatment until it is too late to accomplish significant benefit.

The symptoms of these circulatory, metabolic and nutritional diseases are all secondary to the effects on parenchymatous tissues. They cannot do their work as effectively as they should, but functional depreciation does not produce specific symptoms or changes until actual functional failure intervenes.

The human being is provided with a tremendous functional reserve in all his systems and it is not until these reserves are depleted that definite symptoms make themselves manifest. The symptoms of functional failure, which characterizes the symptomatology in the aged patient, may have been preceded by years by the commencement of those pathological changes now causing the disturbance in health.

Fourthly, the similarity of symptoms in old age often causes

mistaken diagnosis. In fact, in some series of studies at post-mortem, the clinical diagnosis agreed with that at autopsy in only 45 percent of the cases.

For example; for years medical men have thought of strokes in terms of paralysis or other major changes. He has failed often to recognize the smaller stroke which give minor, hard to trace symptoms. There are also the larger strokes in the "silent" areas of the brain which give no neurological symptoms but cause some changes in personality or give vague symptoms often mistaken for other disorders. Upon finding such symptoms as vertigo, parasthesia or pain in the thorax or abdomen, slight bulbar palsy, sudden unexplained loss of weight, a bad tasting or burning mouth, pain in the face or head, diarrhea, shuffling gait, insomnia, over-emotionalism, parkinsonian syndrome, numbness, or personality changes with a sudden onset, a stroke must enter into consideration. It is almost pathognomonic of a stroke when it is found that an elderly person who formerly enjoyed good health, suddenly, at a certain minute of the day, fell seriously ill. Perhaps that he fell down and was unconscious or confused for a minute or two or he went to a hospital and was thought to have "intestinal flu" or some equally vague diagnosis. The most important point is that there came a decided change in character or temperament, sometimes with an increased irritability, a loss of efficiency, a loss of memory and interests. Sometimes the attack has been preceded by similar ones and more often a fairly high blood pressure has been found lowered

after the attack. (3 & 14)

The symptomatology of cranial tumors is vague at the best but in old age it imitates many of the degenerative diseases. In contrast to those manifestations found in the younger group, there is papilledema in less than one third of the cases, less than 10 per cent show optic atrophy, less than half have any perimetric symptoms and about one third have positive x-ray findings. The most common complaints of patients having cranial tumors are those found also in senile dementia or cerebral sclerosis. (116)

A diagnosis of congestive heart failure is frequently made on the slight cyanosis, basal rales, and a palpable liver edge. Pulmonary diseases such as emphysema, pulmonary fibrosis, mild chronic bronchitis, and atelectasis are much oftener responsible for these symptoms. Physical findings such as peripheral edema, especially occurring as an isolated finding, is more usually due to a nutritional deficiency, varicose veins, intra-abdominal tumor or obesity than to congestive heart failure. Chest pain is often consequent to cervical or thoracic osteoarthritis, hiatus hernia, or gall bladder disease rather than angina pectoris.

But left sided cardiac failure may occur in elderly patients without clinically manifest evidence of either prolonged hypertension, coronary disease, or valvular defects as would be expected in the younger age group. The possibility that these patients have underlying coronary disease cannot be excluded even in the absence

of angina. The clinical evidence of congestive failure may be represented in very attenuated form in contrast to the pronounced symptoms of earlier years and may consist merely of a cough, dyspnea, rales, or/and occasionally antemporary elevation of the blood pressure during the attack. (120)

#### Difficulties Inherent in the Patient

The second reason why diagnosis in geriatrics is difficult is the inherent difference in the patient's reaction to disease. It must be admitted that it is quite impossible to draw a line between the things that are normal and those that are abnormal in the senescent organism. But this is not essential to our discussion nor to the therapy applied after the diagnosis is made. As has been previously stated the object of diagnosis is to implement correct therapy and the object of therapy is to give the patient the best possible health compatible with his potentialities. This talk of normal and of health is never absolute but relative. As patient's hemoglobin may be at the lower limits of normal, yet iron and liver may elevate it and cause a relatively great improvement of "health" even though his hemoglobin was "normal" previously. (174)

It has been shown that aging brings change to the organism. Old age is not the ruin of youth plus years. There are structural, chemical, functional, psychological and immunological changes which alter the economy of the body considerably.

One of the greatest difficulties encountered in the older

group is the diminished symptomatic response to a pathological process, even those of a most severe nature. It is well known that in old age there is an increase thermostability. The patient may have severe pneumonia and yet show no rise in temperature. High fevers in old age is uncommon.

The mere fact that there is a left sided chest pain does not mean a heart attack nor does the absence of pain rule out the diagnosis of coronary occlusion; Coronary occlusion occurs without pain in 57.1 percent of cases between 71 and 80 years and 40.8 percent of cases between 61 and 70 years. Even in the sixth decade only 71 percent of patients had pain during the attack. (67)

Sensations produced normally within the organs as a symptom of some pathological process are seen less often in the older patient. As has been mentioned, coronary thrombosis without pain is more common in later life. The extra hepatic biliary passages may be filled with calculi, the common bile duct suddenly obstructed, a communicating fistula established between gall bladder and transverse colon, yet the patient make no complaints referable to the biliary system. Renal stones may pass down the ureter into the bladder without provoking colic and larger ulcerating lesions and carcinoma of the stomach may fail to produce either nausea or other complaints.

But in old age a lowered threshold to pain is neither general nor universal, nor is it found only in serous membranes or internal



visera. Marked distress may accompany diseases of joints, muscles, nerves, and blood vessels, while a severe tonsillitis or erysipelas possibly elicits little discomfort.

The localization of areas of hypersensitivity or tenderness on pressure is very unreliable. Response to pressure is often disturbed in the senile patient so that diagnosis is increasingly uncertain. Peritoneal tenderness is as much altered as peritoneal pain. Muscle spasm may be pronounced in some cases of diffuse peritonites or intra-abdominal disease and in others may be slight or even absent. Referred pain, zones of the skin hypersensitivity, and points of pressure are often undetectable.

Since the aged are often inaccurate, history taking is uncertain at best. Owing to faulty memory the senile patient's own description of his symptoms may be fragmentary and contradictory. Aged patients are apt to deny certain symptoms in order to dodge a disagreeable examination. Semi-consciousness and complete unconsciousness are more frequent in the older patient. In the presence of senile dementia, the confusion becomes worse, and even hopeless.

The pulse in old age is often misleading both in rate and quantity due to the sclerosed condition of the radial arteries.

Physical signs are also misleading. Emphysema and limited excursions of the chest alter and veil pulmonary lesions. In turn, the enlarged overlying emphysematous lungs handicap inspection and

palpation of the mediastinal contents such as the heart and vessels. It is, therefore, not easy to determine the exact site of the apical beat and the other signs of a normal cardiac and aortic enlargement. These conditions tend to exaggerate resonance and obscure dullness. Localization of areas of dullness is much more troublesome in aged. Necropsy now and then discloses surprisingly large areas of tuberculosis or pneumonia which evoked little or no dullness on percussion during life. Distinct loud tubular or bronchial breathing is rarely heard and the emphysematous condition may confuse or obscure symptoms of other chest pathology.

Obesity and intestinal dilatation impede abdominal examination, and these obstacles are aggravated by anatomical changes found in later life. Senile variations cause the abdominal cavity to decrease in size as the thorax and pelvis tend to approach each other. As a result palpation of the upper abdominal organs is hindered. Visceroptosis, on the other hand, greatly facilitates palpation of the organs.

#### Difficulties due to the Inadequacy of Clinical Methods

In order to discover degenerative disorders early enough to make treatment effective, discovery must precede the appearance of subjective symptoms by years. Clinical medicine has approached the problem by looking for positive evidences of abnormality. The early signs of trouble are not manifest in this way. By the time the evidence of abnormality is present the changes are far advanced. It

might be profitable to measure or search for evidences of diminished normality.

The young human systems have immense functional reserves. There is renal functional reserve capacity of 400 percent of the normal burden placed upon these structures. This is also typical of the liver and heart. In fact in all the homeostatic mechanisms of the body such as heat regulation, acid base balance, etc., have a tremendous ability to maintain equilibrium.

This reserve is depleted with advancing years and measurement of this capacity is necessary to adequately evaluate the patients state of health. There is not much change in blood sugar, pulse rate, body temperature, and various other so called "physiologic constants" in the aged, but when the load of disease is added the response is greatly impaired by the decreased reserve.

Reserve capacities are measurable only by stress tests. Examination under static conditions can reveal abnormalities only when they are of considerable magnitude. The heart at rest may yield essentially normal findings on physical examination, or even by electrocardiographic study, despite the fact that the reserve capacity may be so diminished that climbing one flight of steps induces distress. The reverse is often true; a noisy heart with murmurs and irregularities may have developed sufficient compensatory muscular hypertrophy so that it actually is more competent than a quiet one.

With advancing age the ability to maintain homeostatic equilibrium in the face of disturbing influences become diminished. This tendency toward retarded return to normal balance constitutes the basis for the majority of the clinical functional stress tests which are now available. These include the glucose tolerance test, the renal concentration test, and the measurement of ocular accommodation. There is great need for more such tests. Only a few of the tests available are adequate.

When there are tests devised for measuring the reserve function for the various organs in the body which are cheap, simple and harmless, when we can measure the degree of arteriosclerosis in the blood vessels of the body, when we can measure the amount of resistance to various infectious diseases with accurate methods, and when there has been devised a method of determining the presence of early neoplastic growth geriatric diagnosis will have firm footing.

#### Difficulties Inherent in Diagnosis

Diagnosis, as it has been used, means much more than giving a name to a disease, telling one disease from another, or determining the cause of a disease. Instead, it is used in a broader sense. Diagnosis is an analysis of the state of health of an individual for the purpose of guiding therapy and prognostication. An analysis is always changed with the addition of new facts and thus it is with diagnosis. It is an opinion always left open to revisions.

Diagnosis includes identification of the disease process, its etiology, severity and the rate of its progress.

#### Identification of the Disease State

The multiplicity of diseases, the confusion with normal signs of old age, the similarity of many symptoms of the diseases manifest in old age, the absence of symptomatic response and the confusion and unreliability of the history tend to make identification of the disease process difficult.

#### Etiology

Etiology in the diseases of old age is almost unknown. First, no one knows what is the underlying process of aging, secondly, the diseases appearing mostly in old age have little known about their etiology, for instance, what causes cancer, hypertension, or prostatic hypertrophy, lastly those disease that appear at all ages in the young are separate diseases with little else in the way of confusing pathology while in old age the disease may have had its origin years ago and only now does the destructiveness of its process show up. The etiology may be hidden in the great mass of abnormal or unusual responses. For instance, a person with pneumonia got because he had a broken leg which was caused by falling when he fainted from a cerebral embolism from a mural thrombus which resulted from fibrillatory auricle caused by rheumatic fever and the resulting heart lesions.

### Severity

It is hard and almost impossible at times to be fully aware of the severity of the disease in old age. There is often an absence of those signs which are depended upon in earlier years as indications of the severity of the disease. Pain, temperature, and white blood count often are greatly changed in relatively mild conditions and show little or no change in very severe ones.

### Progress

Since there is trouble in finding the severity of the disease it is to be expected that severity would also be difficult to determine. Many of the changes, for better or for worse, are so insidious that they are unrecognized. Some of the progress of a disease may be in the downward direction as measured by the criteria used in the young but may even be a form of compensation in the older person. Auricular fibrillation may be a good sign as well as a bad.

NEW IDEAS OF  
THERAPY  
FOR THE OLD



In diseases occurring in the older patient:

- There is a need for special treatment.

The time honored and tested methods of treatment used by the medical profession in many instances have to be altered due to the change that age brings in the physiology and pathology of the older patient.

In a previous section the changed physiology and anatomy of the aged has been presented. In addition to the changes there enumerated, there are others too numerous to mention in such a short space. In addition, there are many changes not yet discovered because of the limited work in this field and because methods for their discovery have not been perfected. From the few statements on the diseases of old age and disease in old age it seems obvious that the disease processes themselves show alteration.

One may say, and properly so, that many of these changes are so very minute. Yet in reply it should be pointed out that minute changes often determine the difference between health and disease. There are also those relatively untreatable, such as arteriosclerosis, that therapy must be instituted in spite of it.

In geriatrics one deals with low levels of tolerance to metabolic and physiologic disturbances accompanied by a concomitantly low level of regenerative power. The elderly patient presents a set of such inflexible structures and processes that



the stress and strain of either disease or therapy may result in catastrophe.

It must be remembered that one of the results of cerebral arteriosclerotic changes is an increased capillary permeability. Whereas an individual with an intact vascular system has a high tolerance for bromides, in the arteriosclerotic they diffuse more rapidly from the blood stream to the cerebrospinal fluid. This diffusion is so constant that it forms the basis for a test of vascular disease of the brain. Normally the blood bromide level is about three times that of the spinal fluid. In the arteriosclerotic, this ratio is much less.

Barbiturates must be administered with even more care than bromides. Barbital and phenobarbital are metabolized slowly and as a result have a prolonged effect. The rapid acting barbiturates are apt to produce excitement and psychotic behavior during the peak of the drug effect.

A minimum of sedation of any type is therefore preferable for the aged patient. If a hypnotic is required, it should be prescribed with an awareness of its' possible untoward effects. The same limitations which apply to bromides and barbiturates hold for almost all sedatives.

In addition to toxic psychoses due to sedatives are those resulting from other medication, the most common of which is digitalis. Often abnormal behavior is the first sign of overdose.

Antibody production increases with age in response to specific antigens. Aged persons frequently escape acute infections and childhood diseases because they acquired immunity by repeated exposures during early life. It is equally true that old patients can be successfully vaccinated against smallpox, typhoid fever, cholera, and rabies. Although allergic manifestations occur in persons more than sixty-five years of age, the incidence is a great deal lower than that in adults and children. Antitoxins and antisera can be administered with less possibility of serum sickness.

- In general, there is a change in the amounts of drugs needed.

Atropine, morphine, digitalis, adrenaline, ether, aminophylline, barbiturates and bromides often produce better results when given in smaller doses. In fact, an excess of such drugs may cause an opposite effect from the one desired.

Thyroid, insulin, liver extract, iron, belladonna protein preparations, ammonium chloride and other drugs are often seen to give better results in larger doses.

- There is a change in the type of therapy used.

Direct and severe methods of treating disease in the younger individual are successful and justified. However, in the aged neither is of any great value and perhaps have a negative value. It was observed at a famous clinic that the new doctors had a

higher mortality than the older or more experienced ones solely due to the fact that they over treated their patients.

Recently a patient in this hospital died after almost three gallons of fluid were taken from her abdomen. Her condition was described as shock-like after that event and she soon died. In a younger person, this probably would not have occurred.

Further discussion of the types of therapy will follow.

- There is a change in the nature of the patient which affects therapy.

The nature of the patient as well as his whole problem is often misunderstood or not comprehended at all because of the lack of training along these lines. Very few schools teach anything about the problems of the aged and rarely is there any emphasis on this subject in the physicians' supplementary training.

The individual who has so long outwitted nature may have faith in his own regulations and remedies and none in those a stranger tries to impose upon him. He may also be more conscious of the passage of time and, feeling his years are numbered, he is less patient with medication or a regimen that does not bring immediate symptomatic improvement. Simple but forceful explanation of the particular ailment is wise since the physician is faced with an individual who has probably formed his own diagnosis. Probably nothing induces greater cooperation from the patient than his

feeling that he is sharing the responsibility of the physician in promoting his cure. More than in any other instance the patient should be approached as an individual rather than as a problem.

Regardless of the particular medical problem at hand, the most frequent source of difficulty is the onset of psychotic behavior, in the presence of which medical care becomes almost impossible. One of the commonest causes of psychosis is the direct result of attempts at therapy; i.e. the indiscriminate administration of sedatives.

Diseases in old age can best be treated in the aging by helping them to develop a cheerful and complacent attitude. Doctors can be especially helpful by developing such an attitude themselves. It should be remembered that a common method of suggesting fear to a patient arises out of the practice of standing around a patient's bed and carrying on an earnest discussion of the signs, symptoms, laboratory reports and history. Deep in thought, the staff appears grave, serious, and concerned while the discussion is carried on in peculiar medical jargon. Then the session being finished and an order given to the resident, the group passes to the next bed without an explanatory word nor even a single reassuring glance for the subject of this formality. Out of this some kind of suggestion is conveyed to the patient and none is more likely than a sense of the gravity of his plight, alone and helpless, surrounded by unfriendly or uncomprehending people who tell him nothing and who hide their thoughts behind an enigmatic smile.

The old may not admit symptoms if they fear an examination. Others will show illness and think up symptoms in order to excite sympathy. Because of diminished sensibility, it is possible for an aged person to have appendicitis or even petonitis without pain.

Physical examinations of older people should be repeated at regular intervals, but with due consideration for the patient's feeling, in order to avoid any undue annoyance or fatigue. Physicians should make this an examination not only of the patient's physical status and physiology, but also of his attitude and habits. His headaches may be due to quarrels with his employer rather than vascular hypertension or his indigestion may be the result of a visit from a disagreeable relative rather than the pro syndrome of an ulcer. The number of hours he sleeps and the manner in which he spends his leisure time are more important than what his tissues show at the time.

Should the patient stay in bed? Frequently the course of an illness may be entirely changed by this critical decision. So important is the emotional outlook of the aged that many therapeutic procedures must be modified. Although unthought of for the same disease in a younger patient, it frequently is wise to keep an elderly patient sitting in a chair instead of in bed. In this connection one must consider not only the imminent dangers of hypostatic pneumonia subsequent to being confined to bed, but the profound mental depression the aged undergo when they become aware

that disease has at last conquered them and they must take to their bed.

Although from the point of view of the emotional attitude of the patient, it is highly desirable to keep the patient out of bed, careful consideration of the factors involved must be exercised. Perhaps the current ideas concerning putting elderly patients to bed arose from observations of the frequent downhill course of those elderly patients confined to bed because of a fractured hip. This obviously cannot be applied to all illnesses. A patient with cardiac decompensation may require only a few days in bed to achieve diuresis and compensation. (128 & 129)

-The diseases which are prevalent in old age are different than those encountered in those with no signs of old age.

Arteriosclerosis, heart disease based on hypertension, unilateral renal disease, vascular accidents, etc., are typical of old age and require therapy far different than scarlet fever, pneumonia, appendicitis and rocky mountain spotted fever.

In the elderly there is a multiplicity of diseases. Multiple diseases in the aged often tend to compensate for one another. There should be no attempt to treat them all simultaneously since nature may have effected a satisfactory balance and it may be unwise to interrupt it. Treatment should be supportive and supplementary rather than alterative in character. Over enthusiastic

treatment, whether with drugs, diet, or exercise, "will supply autopsy material". Neither is it recommended the palliative treatment suggested to King David in the Book of Kings; "Now King David was old and stricken in years, but he got no heat. Wherefore his servants said unto him, 'Let there be sought for my lord the King a young virgin; and let her stand before the king, and let her cherish him, and let her lie in thy bosom, that my lord the King may get heat.'" "

## TYPES OF TREATMENT BEST SUITED FOR THE AGING

The therapeutic methods open to the practitioner are prophylaxis, palliation, cure, control and constructive therapy. The effectiveness of prophylactic treatment, however, is conditioned by the precision and completeness of knowledge concerning the etiology of a disorder.

Here geriatrics is severely handicapped for the understanding of the etiological factors of most disorders of later years is very vague and incomplete. The same basic obstacle effects curative therapy, for cure of disease is based upon eradication of the causes. Furthermore, cure is obstructed by the diminution of the capacity of the senescent organism to repair itself after injury. Palliative therapy seeks merely to conceal distressing symptoms in otherwise hopeless situations. (86)

Control therapy is not curative nor palliation. Control is the type of therapy used in diabetes, anemia, gout, myxedema, arteriosclerosis and pernicious anemia. The controlled diabetic still has diabetes but he is physiologically well. Controlative measures seeks only to reestablish the normal balance disturbed by disease. Most chronic and progressive diseases of later years are not amenable to cure. They are, however, amenable to control and retardation. This is the form of therapy most appropriate to the majority of problems of geriatric medicine.

Another therapeutic objective in geriatrics is that of con-



structive therapy. There has been little of this applied to adults, but pediatricians have taken apparently well babies and made them healthier by scientific feeding and meticulous attention to hygiene and immunity. These healthier children become healthier adults. Similarly geriatrics has the opportunity to modify and retard the detrimental consequences of senescence. Health is relative, never absolute. There is always room for improvement.

The potentialities of constructive and controlative therapy for adults are immense. Conscientious application of existing knowledge in such fields as nutrition, endocrinology, hematology, and psychiatry can do much to increase well being during senescence. Such guidance must be applied individually. The wholesale methods of public health are decidedly inadequate for older age groups. The method of the mass approach has been directed toward keeping sources of injury away from the individual. No improvement in the intrinsic vigor, resistance and endurance of the individual can be expected from these methods.

This kind of medicine goes a step further than preventive medicine since it is an attempt to detect disease tendencies and to treat the patient before he reaches the symptom stage. This involves a close study of the person's hereditary background, constitutional type, racial factors, intellectual equipment, racial adjustment, reaction to climate, occupation and past diseases.

## SUMMARY

It has been long realized that the human organism at any stage of development has its' own characteristic advantages and disadvantages. These are the result of the process of aging. Both growth and decline are the result of this process. The influence of aging on all the functions of the body is taken into consideration in medicine to aid in diagnosis and therapy.

The causes of aging can only be conjectured. It can't even be accurately measured, for biological time and chronological time do not correspond by any manner of means. Many factors, however, do influence the course of the aging process. In fact anything altering the metabolism of the body may retard or hasten the onset of old age. This fact makes it exceedingly difficult to judge what is pathological and what is non-pathological in the changes that occur in old age.

With the declining birth rate, curtailed immigration, and improved morality in childhood and early adult, there has been an increase in the older population. The following generation will contain an even greater proportion of old people. This "aging of the nation" will have influence on every phase of the social and economical life of the country. There will have to be increased state support of the aged, politicians will woo the older, more conservative group, business will cater to the whims and the desires of an older clientel. Most important to members of the

medical profession is the increase of aged patients they will have to treat, the larger number of cases of degenerative diseases they will see, and their responsibility of maintaining health among this group.

In spite of the ignorance of the peculiarities of the senescent person prevalent among most of the population and a large proportion of the medical profession, there has been established an American Geriatric and American Gerontology Society, each with its' publication. The Readers' Digest, American Magazine, Saturday Evening Post, Coronet and Harper's have recently published articles concerning the problems of old age. The interest in old age has been manifest for centuries, but little in a scientific nature has been done until very recently.

Unfortunately for the subject of geriatrics, much of the writing which has been done on the subject has concerned itself with longevity instead of the real objective of geriatrics.

"To add life to years rather than years to life." That is the basic motive for campaigning for better understanding and further investigation of the aged and, more important, the aging process. What is sought is not a dragging out of a vegetative almhouse existance but the promotion of real efficiency.

There are many changes in old age. No system is unaffected, and no process is the same as it was at an earlier age. The

older individual is by no means the same individual he was years ago. In this thesis a few of the changes have been mentioned and there are many more, but in comparison to what remains unknown, they are relatively few. New methods of investigation are needed and thousands of experiments must be made to establish a picture of what really constitutes aging and its effects upon the human make up. Herein lies the answers to the question of cancer, arteriosclerosis, hypertension and other important problems.

As it is recognized that the old need special attention because of their physical and physiological make up, it is also realized that diagnosis and therapy will, of necessity, be changed. New methods of diagnosis are needed and new programs of therapy must be instituted in order to adequately provide for this growing segment of the population.

## BIBLIOGRAPHY

1. Adair, F. L. in Steiglitz: Geriatric Medicine, 1943, W. B. Saunders, Philadelphia.
2. Allen, E. B.: Functional personality disorders, North Carolina Med. J., vol. 5, p. 585, December, 1944.
3. Alvarez, W. C.: Small unrecognized strokes; common cause of illness, Penn. Med. J., vol. 47, p. 1042-1046, July, 1944.
4. Andrew, W. & A. V.: Senile involution of the thyroid gland; Am. J. Path., vol. 18, p. 849-851, Sept., 1942.
5. Andrus, F. C.: The relation of age and hypertension to the structure of the small arteries and arterioles in skeletal muscle. Am. J. Path., vol. 12, p. 635-652, 1936.
6. Aschoff, L., in Cowdry Arteriosclerosis; The MacMillan Co., New York, 1933.
7. Aschoff, L., in Mueller-Dehman; Morbidity Statistics; Geriatrics, vol. 1, p. 285-295, 1946.
8. Babbitt, J. A.: Relation of Otolaryngology to Geriatrics; Ann. Otol. Rhin. & Laryng., vol. 50, p. 1079-1084; December, 1941.
9. Bainbridge, W. S.: Our Debt to the Aging; North Carolina Med. J., vol. 5, p. 557-560; December, 1944.
10. Baker, A. B.: Structure of Small Cerebral Arteries and their Change with Age. Am. J. Path. vol. 13; p. 453-462; May, 1937.
11. Barker, L. F.: General Problems; Proceed. Interst. Postgrad. Med. Ass. of North America; p. 150-155; 1940.
12. Baron, E.: Incidence of Occult Adenocarcinoma of the Prostate after 50 years of age; Arch. Path. vol. 32, p. 787-789, Nov., 1941.
13. Barrow, W.: Acute Appendicitis; Am. J. Surg., vol. 53, p. 242-246; August, 1941.
14. Basse, P. M. & Blanton, B. N.: So-called Strokes; Tex. State Med. J., vol. 36, p. 670-673; Feb., 1941.
15. Baumgartner, L. Age and Antibody Production; J. Immunol., vol. 27, p. 407-427; Jan., 1934.

16. Baumgartener, L.: Age and Antibody Production. *J. Immunol.* vol. 33, p. 477-488, December, 1937.
17. Beaser, S. B., Rudy, A. & Seligman, A. M.: Capillary Fragility in Relation to Diabetes, Hypertension and Old Age. *Arch. Int. Med.*; vol. 7, p. 18-22, January, 1944.
18. Bell, E. T. in Cowdry: *Arteriosclerosis*, The MacMillan Co., 1933.
19. Benedict, F. G. & Root, H. F.: *Physiology of Old Age*. New England M. J.; vol. 211, p. 521-536.
20. Berens, C.: Aging Process in the Eye and Adnexa. *Arch. Ophth.*; vol. 29, p. 171-179, February, 1943.
21. Berghoff, R. S.: Aging Heart. *Proc. Insterst. Postgrad. Med. Ass. North Am.*; p. 1945.
22. Bloom, A. L.: Gastric Secretion with Advancing Years. *J. Clin. Investigation*; vol. 19, p. 61-63, March, 1940.
23. Boas, E. P.: Aging of the Cardiovascular System. *New York Acad. Med. Vol.* 16, p. 607-617, October, 1940.
24. Boas, E. P.: *Physiological and Clinical Phenomena of Aging*. *New Orleans Med. & Surg. J.*; vol. 97, p. 64, August, 1944.
25. Boas, E. P.: Aging Heart. *New York Med. No. 23*; vol. 1, p. 17-21, December 5, 1945.
26. Bogomolets, A. A.: Anti-Reticular Cytotoxic Serum as a Means of Pathogenic Therapy. *Am. Re. of Soc. Med.* ; vol. 1, p. 101-112, 1943.
27. Boler, T. O.: Urinary Disturbances. *Nebr. Med. J.*; vol. 30, p. 202-205, June, 1945.
28. Bortz, E. L.: Geriatrics - A New Light on Old Folks. *Clinics*; vol. 1, p. 386-405, August, 1942.
29. Bowman, K., Solomon, H. C., & Wortis, J.: Geriatrics. *Am. J. Psychiat.*; vol. 100, p. 537-540, January, 1944.
30. Breck, L. W.: Fractures. *Southwestern Med. J.*; vol. 25, p. 229-234, August, 1941.
31. Bruger, M. & Rosenbrantz, J. A.: Arteriosclerosis and Hypothyroidism. *J. Clin. Endocrinology*; vol. 2, p. 178-182, March, 1942.

32. Byrd, E. S.: Heart-Vascular Aging. *J. Med. Ass. Georgia*; p. 34-74, April, 1945.
33. Cameron, D. E. & Rosen, S. R.: Reactivity of Intecranial Vessels. *Am. J. Med. Sc.*; vol. 201, p. 871-876, June, 1941.
34. Cannon, W. B. in Cowdry; *Problems of Aging*. Williams & Wilkins Co., Baltimore, 1939.
35. Carlson, A. J. in Cowdry; *Problems of Aging*. Williams & Williams Co., Baltimore, 1939.
36. Carlson, A. J.: Physiology of aging. *Northwest. Med.*; vol. 46, p. 6-46, January & February, 1943.
37. Carr, J. G.: Diseases of the Lung. *Med. Clin. of North Am.*; vol. 24, p. 33-43, January, 1940.
38. Carr, J. G.: Diseases of Lungs. *Proc. Interst. Postgrad. Med. A. North Am.*; p. 92, 1941.
39. Chaney, W. C.: Problems of Old Age. *Memphis Med. J.*; vol 20, p. 66-68, May, 1945.
40. Cohn, A. E. in Cowdry; *Problems of Aging*. Williams & Wilkins Co., Baltimore, 1939.
41. Cooper, E. R. A.: *The Histology of the more Important Endocrine Organs at Various Ages*. Oxford Press, London-New York, 1925.
42. Cottrell, L.: Histologic variation with age in Apparently Normal Peripheral Nerve Trunks. *Arch. Neurol. & Psychiat.*, *Arch. Neurol. & Psychiat.*, vol. 43, p. 1138-1150, June, 1940.
43. Cowdry, E. V.: *Arteriosclerosis*. The MacMillan Co., New York, 1933.
44. Cowdry, E. V.: *Problems of Aging*. Williams & Wilkins Co., Baltimore, 1939.
45. Cowdry, E. V.: Physician's Opportunity to help Older People. *J. Am. Med. Ass.*; vol. 125, p. 402-405, June, 1944.
46. Critchley, M. in Cowdry; *Problems of Aging*. Williams & Wilkins Co., Baltimore, 1939.
47. Davis, N. S.: Factors Which May Influence Senescence. *Ann. Int. Med.*; vol. 18, p. 81-84, January, 1943.

48. Diethelm, O.; Psychological and Psychopathologic Aspects of Aging. North Carolina Med. J.; vol. 5, p. 583-588, December, 1944.
49. Dogliotti, G. C. & Mizzi-Nuti, G.; Thyroid and Senescence. Endocrin; vol. 19, p. 289-292, 1935.
50. Dublin, L. I.; Statistical and Social Implications in the Problem of our Aging Population. University of Penn. Bi-centennial Conference; p. 1-16, 1941.
51. Dublin, L. I. in Cowdry; Problems of Aging, Williams & Wilkins, Baltimore, 1939.
52. Eliaser, M. & Kondo, B. O.; Electrocardiogram in later life. Arch. Int. Med.; vol. 67, p. 637-646, March, 1941.
53. Engle, E. T. in Cowdry; Problems of Aging, Williams & Wilkins Co., Baltimore, 1939.
54. Fischer, F. P.; Changes in the Eye. Ophthalmologica; vol. 102, p. 226-230, October, 1941.
55. Fluhman, C. F.; Hormonal Relations of Menopausal Symptoms. J. Clin. Endocrin; vol. 4, p. 585-591, 1944.
56. Fowler, W. M., Stephens, A. L. & Stump, R. B.; Changes in Hematological Values in Old Patients. Am. J. Clin. Path.; vol. 11, p. 700-705, September, 1941.
57. Fowler, E. P.; Aging Ear. Arch. Otolaryng.; vol. 40, p. 475-482, December, 1944.
58. Frandsen, C.; Factors Influencing the Heart which Become Evident After 50 Years of Age. Neb. Med. J.; vol. 28, p. 75-78, March, 1943.
59. Fraser, R. W., Forbes, A. P., Albright, F., Sulkowitch, H. & Reifenstein, E. C.; The Excretion of Ketosteroids in Old Age. J. Cl. End.; vol. 1, p. 234-245, 1941.
60. Freeberg, A. S. & Lewis, H. O.; Geriatrics: The Normal Heart. New England Med. J.; p. 231-731, November 30, 1944.
61. Freeman, J. T.; Factors contributing to Geriatric Nutrition. Penn. M. J.; vol. 46, p. 595, March, 1945.
62. Freeman, J. T.; Status of Geriatrics. Penn. Med. J.; vol. 43, p. 813-816, March, 1946.



63. Gardner, E.: Decrease in Human Neurons with Age. *Anat. Rec.*; vol. 77, p. 529-536, August 26, 1940.
64. Geist, S. H., Salmon, U. J. & Walter, R. I.: Hormonal Aspects of Functional Urinary Disorders in Post Menopausal Women. *Urol. and Cutan. Rev.*; vol. 47, p. 275-279, May, 1943.
65. Goldzieher, M. A.: *The Adrenal Glands in Health and Disease.* F. A. Davis Co., Philadelphia, Penn., 1944.
66. Golob, M.: Geriatrics in Internal Medicine. *Am. J. Digest. Dis.*; Vol 11, p. 156-163, May, 1944.
67. Gorham, L. W. & Martin, S. J.: Coronary Occlusion with and without Pain. *Arch. Int. Med.*; vol. 62, p. 799-804, March, 1933.
68. Grant, H. W.: Eye Problems. *Journal Lancet*; vol. 64, p. 199-205, June, 1944.
69. Hamblen, E. C., Cuyler, W. K. & Baptist, M.: Excretion of 17-ketosteroids in Females. *J. Cl. End.*; vol. 1, p. 777-782, 1941.
70. Harding, T. S.: Statistics of Geriatrics. *Am. J. Pharm.*; vol. 112, p. 421-429, December, 1940.
71. Henderson, D. K.: Early Diagnosis and Treatment of Mental Disorders. *Practitioner*; vol. 149, p. 1-6, July, 1942.
72. Hendrick, J. W.: Hyperthyroidism. *Am. J. Surg.*; vol. 52, p. 466-471, June, 1941.
73. Hertz, A. T.: The Aging Ovary - A Preliminary Note. *J. Clin. Endocrin*; vol. 4, p. 581-585, 1944.
74. Hertzler, A. E.: Histological and Histochemical Structure of the Normal Thyroid Gland. *Arch. Surg.*; vol. 38, p. 417-427, 1939.
75. Himsworth, H. P. & Kerr, R. B.: Age and Insulin Sensitivity. *Clin. Sc.*; vol. 4, p. 153-157, 1940.
76. Hofstatter, L., Sonnenberg, A., & Kountz, W. B.: The Glucose Tolerance in Elderly Patients in *Bio. Symp.* (Moore)
77. Holender, A. R.: Histopathology of Nasal Mucosa. *Arch. Otolaryng.*; vol. 40, p. 92-95, August, 1944.

78. Hoskins, R. G., Levene, H. M. & Bewins, S.: Male Sex Hormones and Bodily Vigor in Senility. *Endocrin.*; vol. 25, p. 143-146, July, 1939.
79. Hoskins, R. G. & Bevin, S.: Possible Relationship Between Gonads and Adrenal. *Endocrin.*; vol. 27, p. 929-940, 1941.
80. Howell, T. H.: Progressive Cerebral Ischemia. *Brit. Med. J.*; vol. 2, p. 746, December 11, 1943.
81. Howell, W. E.: Gerontology, Geriatrics and Gerontology. *J. Tenn. Med. Ass.*; vol. 38, p. 1-4, January, 1945.
82. Huffman, O. D.: Renal Function. *Cal. & West. Med.*, vol. 50, p. 16-20, January, 1939.
83. Ingle, D. J.: The Work Performance of Adrenalectomized Rats Treated with Corticosterone and Chemically Related Compounds. *Endocrin.*; vol. 26, p. 472-477, 1940.
84. Ivy, A. C. in Cowdry; *Problems of Aging*. Williams & Wilkins Co., Baltimore, 1939.
85. Ivy, A. C., Schmidt, C. R. & Beazell, J. M.: The Gastric Digestion of Starches. *J. Nutrition*; vol. 12, p. 59-83, 1936.
86. Johnson, W. M.: Therapy in Older Patients. *North Carolina Med. J.*; vol. 4, p. 385-389, September, 1943.
87. Karsner, H. T. in Cowdry; *Arteriosclerosis*. The MacMillan Co., New York, 1933.
88. Kelemen, G.: Palatine Tonsil in the Sixth Decade. *Ann. Otol. Rhin. & Laryng.*; vol. 52, p. 419-424, June, 1943.
89. Kopelowski, J. C.: Diseases of the Gastrointestinal Tract. *J. Missouri Med. A.*; vol. 38, p. 55-59, February, 1941.
90. Kosmak, G. W.: Gynecological and Other Implications which Relate to Aging Female Population. *Am. J. Obst. & Gynec.*, vol. 44, p. 897-901, November, 1942.
91. Kountz, W. B., Somenberg, A., Hofstatter, L. & Wolff; Blood Cholesterol Levels in Elderly Patients (Moore). (#119)
92. Kwetschmer, H. L.: Problem of the Chronically Ill Patient. *J. Am. Med. Ass.*; vol. 127, p. 1025-1031, April, 1945.

93. Lawton, G.: After 65? *Ment. Hyg.*; vol. 25, p. 414-416, July, 1941.
94. Lawton, G.: Happiness in Old Age. *Ment. Hyg.*; vol. 27, p. 231-236, April, 1943.
95. Lee, R. I.: Geriatrics. *Rhode Island Med. J.*, vol. 28, p. 647-651, September, 1945.
96. Lewis, W. H., Jr.: Changes With Age in the Basal Metabolic Rate. *Am. J. Physiol.*; vol. 121, p. 500-515, 1938.
97. Lewis, W. H., Jr. & Alving, A. S.: Changes in Urine with Old Age. *Am. J. Physiol.*; vol. 123, p. 500-515, 1938.
98. Loeb, L.: Hormones and Their Place in Aging. *Harvey Lectures*; vol. 36, p. 228-250, 1941.
99. Loeb, R. F.: Glandular Physiology and Therapy. Adrenal Cortex Insufficiency. *J. A. M. A.*; vol. 116, p. 2495, 1941.
100. MacKay, L. L., MacKay, E. M. & Addis, T.: The Effects of Various Factors on the Degree of Compensatory Hypertrophy of the Kidney after Unilateral Nephrectomy. *J. Clin. Invest.*; vol. 1, 576-589, 1944.
101. Mazier, M. & Reisinger, J. A.: An Electrocardiographic Study of Cardiac Aging. *Am. J. Physiol.*; vol. 21, p. 645-652, 1944.
102. Metropolitan Life Insurance Co.: Medical Practice and Our Aging Population, *Statistical Bulletin*; vol. 22, p. 1-3, April, 1941. Chances of Reaching Age 65. *Statistical Bulletin*; vol. 22, p. 1, April, 1941.
103. Metropolitan Life Insurance Co.: Increase in Longevity. *Statistical Bulletin*; vol. 25, p. 3, May, 1942. Decrease in Expected Mortality. *Statistical Bulletin*, vol. 25, p. 7, November, 1944. Large Decline in Mortality from Degenerative Diseases; *Statistical Bulletin*; vol. 23, p. 5-8, March.
104. Metropolitan Life Insurance Co.: Longevity at Record High; *Statistical Bulletin*; vol. 27, p. 1-3, August, 1946. Our Aging Population. *Statistical Bulletin*; vol. 27, p. 3-7, March. Encouraging Trends in Heart Disease; vol. 27, p. 6-8, August. Incidence of Cancer by Sites; *Statistical Bulletin*; vol. 27, p. 9, July, 1942.
105. Meyer, J., Golden, J. S., Steiner, N. & Necheles, H.: Ptyalin Content of Human Saliva in Old Age. *Am. J. Physiology*; vol. 119, p. 600-602, July, 1937.

106. Meyer, J. & Necheles, H.: Clinical Significance of Salivary, Gastric, and Pancreatic Secretion. *J. A. M. A.*; vol. 115, p. 2050-2053, December, 1940.
107. Meyer, J., Spier, E., & Newalt, F.: Basal Secretion of Digestive Enzymes. *Arch. Int. Med.*; vol. 65, p. 171-177, January, 1940.
108. Meyer, J. & Saphir, O.: Peptic Ulcer. *Am. J. Digest. Dis.*; vol. 10, p. 28-32, January, 1943.
109. Meyer, J., Sorter, H., Oliver, J. & Necheles, H.: Intestinal Absorption in Old Age. *Gastroenterology*; vol. 1, p. 876-881, September, 1943.
110. Miles, W. R. & Miles, C. C. in Stieglitz; *Geriatric Medicine*. W. B. Saunders Co., Philadelphia, 1943.
111. Miller, I.: Blood Sedimentation Rates in Old Age. *J. Lab. & Clin. Med.*; vol. 21, p. 1227-1236, September, 1936.
112. Miller, I.: Normal Hematologic Standards. *J. Lab. & Clin. Med.*; vol. 24, p. 1176-1186, August, 1939.
113. Miller, I.: Blood Pressure Studies. *New York State J. Med.*; vol. 41, p. 1631-1636, August, 1941.
114. Miller, I.: Vital Capacity Studies. *J. Lab. & Clin. Med.*; vol. 27, p. 737-739, March, 1942.
115. Mitchell, W. J.: Treatment of Diabetes. *Penn. M. J.*; vol. 44, p. 1015-1019, May, 1941.
116. Moersch, F. P., Craig, W. M., & Kernohan, J. W.: Tumors of the Brain. *Arch. Neurol. & Psychiat.*; vol. 45, p. 235-245, February, 1941.
117. Moore, R. A.: The Total Number of Glomeruli in the Normal Human Kidney. *Anat. Rec.*; vol. 48, p. 153-168, 1931.
118. Moore, R. A.: The Evolution and Involution of the Prostate Gland. *Am. J. Path.*; vol. 12, p. 599-624, 1936.
119. Moore, R. A.: *Biological Symposis*. Jaques Cottell Press, Lancaster, Pa., 1945.
120. Moschcowitz, E.: Potential or Latent Congestive Failure. *J. Mt. Sinai Hosp.*; vol. 9, p. 663-667, November, 1942.

121. Mueller-Deham, A. & Robson, S. M.: Internal Medicine in Old Age. Williams & Wilkins Co., Baltimore, 1942.
122. Mueller-Deham, A.: Are Geriatrics Mortality and Morbidity Statistics Reliable. Geriatrics; vol. 1, p. 285-295, 1946.
123. Nascher, I. L.: The Aging Mind. Med. Record; vol. 157, p. 669-673, November, 1944.
124. Necheles, H., Plotke, F. & Meyer, J.: Active Pancreatic Secretion in Aged. Am. J. Digest. Dis.; vol. 9, p. 157-166, May, 1942.
125. Newman, B. & Gitlow, S.: The Erythrocyte in the Aged Male and Female. Am. J. of Med. Science; vol. 205, p. 677-689, 1943.
126. Niehaus, F. W.: Aging Population, its Influence on Cardiovascular Disease and Consideration of Possible Prophylactic Measures. Nebr. Med. J.; vol. 26, p. 348-351, October, 1941.
127. Norman, J. F.: Our Aging Population. Minnesota Med.; vol. 24, p. 1066-1071, December, 1941.
128. Norman, J. F.: General Care of Aged. Minnesota Med.; vol. 26, p. 876-881, October, 1943.
129. Norman, J. F.: Abuse of Prolonged Rest. Minnesota Med.; vol. 28, p. 803-809, October, 1945.
130. Northington, J. M.: Future of Geriatrics. South. Med. & Surg.; vol. 104, p. 386-389, July, 1942.
131. Novak, E. & Richardson, E. H.: Proliferative Changes in Endometrium. Am. J. Obst. & Gynec., vol. 42, p. 564-569, October, 1941.
132. Oliver, J. R. in Cowdry: Problems of Aging. Williams & Wilkins Co., Baltimore, 1939.
133. Overholser, W.: Psychiatric Problems in Old Age. Wisconsin Med. J.; vol. 44, p. 300-308, March, 1944.
134. Overholser, W. in Stieglitz: Geriatric Medicine. W. B. Saunders Co., Philadelphia, 1943.
135. Palmer, H. D., Braceland, F. J. & Hastings, D. W.: Somatopsychic Disorders. Am. J. Psychiat.; vol. 99, p. 856-860, May, 1943.

136. Papanicolaou, G. N. & Schorr, E.: Vaginal Smears in Post-Menopausal Women. *Am. J. of Ob. & Gyn.*; vol. 31, p. 801-831.
137. Paulus, D. D.: Chronic Digestive Disturbance. *J. Oklahoma Med. A.*, vol. 37, p. 297-301, July, 1944.
138. Pepper, O. H. P.: Principles of Diagnosis and Treatment. *Nebr. Med. J.*; vol. 24, p. 401-406, November, 1939.
139. Pepper, O. H. P.: Geriatrics and the Physician. *Uni. of Penn. Bicentennial Conference*; p. 27-35, 1941.
140. Perry, C. B.: Heart Disease. *Practitioner*; vol. 148, p. 117-121, February, 1942.
141. Pippin, B. N.: Teeth and Aging. *Journal Lancet*; vol. 64, p. 203-207, June, 1944.
142. Poer, D. H.: Hyperthyroidism. *J. M. A. Alabama*; vol. 10, p. 353-358, May, 1941.
143. Powers, J. H.: Acute Appendicitis. *Ann. Surg.*; vol. 117, p. 221-225, February, 1943.
144. Proetz, A. W. in Steiglitz; *Geriatric Medicine*. W. B. Saunders Co., Philadelphia, 1943.
145. Rafsky, H. A. & Newman, B.: Liver Function Tests in Old Age. *Am. J. Digest. Dis.*; vol. 10, p. 66-69, January, 1943.
146. Rattner, H. in Steiglitz; *Geriatric Medicine*. W. B. Saunders Co., Philadelphia, 1943.
147. Reich, C., Swirsky, M. & Smith, D.: Sternal Bone Marrow in the Aged. *J. of Lab. & Clin. Med.*; vol. 29, p. 508-509, May, 1944.
148. Robbins, I. L.: General Discussion of Problems of Old Age. *New Orleans Med. & Surg. J.*; vol. 93, p. 184-187, October, 1940.
149. Robertson, G. W. & Yudkin, J.: Age and Dark Adaptation. *J. Physiol.*; vol. 103, p. 1-8, 1944.
150. Robinson, S. C. & Brucer, M.: Blood Pressure Studies in Old Age. *Arch. Int. Med.*; vol. 64, p. 409-445, September, 1939.
151. Robinson, G. W.: Psychiatric Geriatrics. *J. A. M. A.*; vol. 116, p. 2139-2141, May 10, 1941.

152. Rogoff, J. M. & Stewart, G. N.: Studies on Adrenal Insufficiency. *Am. J. Physiology*; vol. 78, p. 683-710.
153. Rones, B. in Stieglitz; *Geriatric Medicine*. W. B. Saunders Co., Philadelphia, 1943.
154. Roper, F. A.: Chronic Bronchitis. *Practitioner*; vol. 148, p. 18-21, January, 1942.
155. Rosenthal, J.: Gall Bladder Disease. *Ann. Int. Med.*; vol. 20, p. 933-939, June, 1944.
156. Rothschild, D. & Sharp, M. L.: Origin of Senile Psychoses. *Dis. of Nerv. System*; vol. 2, p. 49-54, February, 1941.
157. Russek, H. I.: Blood Pressure. *Am. Heart J.*; vol. 26, p. 11-17, July, 1943.
158. Russek, H. I. & Zohman, B. I.: Normal Blood Pressure in Senescence. *Geriatrics*; vol. 1, p. 146-149, March, 1946.
159. Sebrell, W. H. in Stieglitz; *Geriatric Medicine*. W. B. Saunders Co., Philadelphia, 1943.
160. Segall, H. N.: Aging Process. *Indust. Med.*; vol. 9, p. 521-524, October, 1940.
161. Shook, N. W.: Age Changes in Alveolar CO<sub>2</sub> Tension. *Am. J. Physiol.*; vol. 133, p. 610-616, July, 1941.
162. Shook, N. W.: Kidney Function Tests in Aged Males. *Geriatrics*; vol. 1, p. 232-238, 1946.
163. Simmonds, M. in Thewlis; *The Care of The Aged*. C. V. Mosby Co., St. Louis, Mo., 1946.
164. Simms, H. S. & Stolman, A.: Changes in Human Tissue Electrolytes in senescence. *Science*; vol. 86, p. 269-270, 1937.
165. Simms, H. S.: Problems of Aging and of Vascular Disease. *Science*; vol. 97, p. 183-187, February 20, 1942.
166. Skinner, E. H.: Radiologist Looks at Aging Bones. *Journal Lancet*; vol. 64, p. 189-193, June, 1944.
167. Smith, C. G.: Loss of Olfactory Nerves with Age. *J. Comp. Neurol.*; vol. 77, p. 589-595, December, 1942.
168. Smith, K. J. & Jaffe, R. H.: Frequency of Prostatic Hypertrophy in White and Colored Races. *Urol. & Cutan. Rev.*; vol. 36, p. 661-662, 1932.

169. Soper, H. V.: Geriatrics: Early Senescence. Med. Rec.; vol. 153, August, 1941.
170. Soper, H. W.: Gerontology. Am. J. Digest. Dis.; vol. 12, p. 219-221, September, 1945.
171. Sprague, H. B. in Steiglitz: Geriatric Medicine, W. B. Saunders Co., Philadelphia, 1943.
172. Stalker, L. K.: Appendicitis among Individuals more than 60 years of Age. Surg. Gyn & Ob.; vol. 71, p. 54-59, July, 1940.
173. Stern, M. J.: Ophthalmic Geriatrics. Kentucky Med. J.; vol. 43, p. 202-204, August, 1945.
174. Stieglitz, E. J.: Relativity in Age and Health. New York Med.; no. 14; vol. 1, p. 19-22, July 20, 1940.
175. Stieglitz, E. J.: Potentialities of Preventive Geriatrics. New England Med. J.; vol. 225, p. 247-252, August, 1941.
176. Stieglitz, E. J.: Geriatric Medicine. W. B. Saunders Co., Philadelphia, 1943.
177. Stieglitz, E. J.: Pertinent Problems in Geriatric Medicine. Ann. Int. Med.; vol. 18, p. 89-93, January, 1943.
178. Stieglitz, E. J.: Difficulties in the Clinical Recognition of Degenerative Diseases in Moore. (#119)
179. Sutton, M. B.: Androgens and Prostatic Enlargement. J. Clin. Endocrinology; vol. 1, p. 882-886, November, 1941.
180. Talbot, N. B., Butler, A. M. & McLachlan, E. A.: The Colorimetric Assay of Total Alpha and Beta 17-Ketosteroids in Extracts of Human Urine. J. Biol. Chem.; vol. 132, p. 595-603, 1940.
181. Taran, L. M. & Kaye, M.: Electrocardiographic Studies. Ann. Int. Med.; vol. 20, p. 954, June, 1944.
182. Thewlis, M. W.: Care of the Aged. J. Am. Med. Ass.; vol. 120, p. 749-752, November, 1942.
183. Thewlis, M. W.: The Art of Geriatric Practice. North Carolina Med. J.; vol. 5, p. 580-585, December, 1944.
184. Thewlis, M. W.: The Care of The Aged. C. V. Mosby Co., St. Louis, Mo., 1946.



185. Thompson, C. Q.: Blood Pressure as seen in Aging Individuals. *Nebr. Med. J.*; vol. 28, p. 78, March, 1943.
186. Thompson, W. O.: Enderine Problems. *Med. Clin. North Am.*; vol. 24, p. 79-91, January, 1940.
187. Todd, T. W. in Cowdry: Problems of Aging. Williams & Wilkins Co., Baltimore, 1939.
188. Tuoghy, E. B.: Handbook of Nutrition, Feeding the Aged. *J. A. M. A.*; vol. 121, p. 42, 1942.
189. Ulrich, H. L.: Cardiovascular Deterioration. *Geriatrics*; vol. 1, p. 150-154, March, 1946.
190. Van de Heydt, R.: Visual Prognosis for Aging Lens. *Am. J. Ophth.*; vol 29, p. 576-578, May, 1942.
191. Van Liere, E. J. & Northrup, D. W.: Gastric Emptying Time in Old People. *Am. J. Physiol.*; vol. 43, p. 719-722, 1941.
192. Van Liere, E. J.: Effect on Gastric Emptying. *West Virginia Med. J.*; vol. 38, p. 114-120, March, 1942.
193. Vanzant, F. R., Alvarez, W. C., Eusterman, G. B., Dunn, H. L. & Berkson, J.: The Normal Range of Gastric Acidity from Youth to Old Age. *Arch. Int. Med.*; vol. 49, p. 345-359, 1932.
194. Wagener, H. P.: Changes in Choroid and Retina in Old Age. *Am. J. Med. Science*; vol
195. Warthin, A. S.: Old Age, The Major Involution. Paul B. Hoeber Co., New York, 1929.
196. Wechsler, D.: Measurement of Adult Intelligence, Williams & Wilkins, Baltimore; p. 55-71 & 118-129, 1941.
197. Weidman, F. D. in Cowdry: Problems of Aging. Williams & Wilkins Co., Baltimore, 1939.
198. Wells, H. G. in Cowdry: Arteriosclerosis. The MacMillan Co. New York, 1933.
199. Williamson-Noble, F.: Failing Sight. *Post. Grad. Med. J.*; vol. 10, p. 298-301, August, 1934.
200. Wilson, A. K.: Roentgenologic Findings in Old Age. *Am. J. Roent.*; vol. 51, p. 685-689, July, 1944.

201. Wissler, C. in Cowdry: Problems of Aging. Williams & Wilkins Co., Baltimore, 1939.
202. Witsch, E. & Gardner, M. R.: Quantitative Studies on the Hormones of the Human Pituitaries. Endocrin; vol. 26, p. 565-576, April, 1940.
203. Young, G. A.: Nervous Disorders, Nebr. Med. J.; vol. 25, p. 252-255, July, 1940.