search is made for them. The absence of other physical signs and the negative findings in roentgenograms prove that other cause does not exist for the history; and correction of these defects, with prevention of further intestinal or omental pinching, removes the gastric symptoms. Chronic pelvic inflammatory disease in women, and old adhesions involving the intestinal wall in a former peritonitis, are other conditions that at times may simulate ulcer in the way described. Pelvic examination, giving direct or positive evidence, and roentgenograms giving indirect or negative evidence, call attention to the site of the real pathologic condition.

6. Gastric Crises.—The hardest lesson to learn about abdominal disease is that of "gastric crises," due to no disease within the abdomen itself, but to disease in the spinal cord. These crises come at irregular intervals, weeks or months apart, with good health between. They last for days or weeks, and the attacks are characterized by intense pain and by vomiting, so that the patient fears to take food and cannot keep it if he does. No wonder the first thought with such a history is of some disease of the stomach; and commonly of ulcer as corresponding most closely in its manifestations. The physical examination of the abdomen is negative in such case; and the stomach contents most often show hyperchlorhydria after an attack is over. It is not surprising, therefore, if such symptoms have in the past misled us, even to the point of advising a gastro-enterostomy to prevent their repetition. Now, however, the negative roentgenographic findings come to our rescue, to save us from error; and at all times, even without roentgenograms, we have other definite signs to point out the real disease, if only we take the trouble to look for them. First, these signs consist in alteration of reflexes, in one pupil or both; in patellar or Achilles tendons, perhaps only on one side; or in the plantar reactions to stimuli. Second, and most important, lumbar puncture and examination of spinal fluid for cell count and Wassermann reaction afford the most constant clue to the real disease present.

Gastric Neuroses.—These are still mentioned in the textbooks, in the differential diagnosis of ulcer, but their existence outside of textbooks must be considered very dubious. A history resembling that of ulcer, with hyperchlorhydria, does not occur without some pathologic condition somewhere in the body, usually in the abdomen, to explain it. In times past this "acid dyspepsia" has been considered as a possible result of a disturbance of the nervous system only; but such a supposition, with our increased facilities for eliciting facts, is no longer tenable. The exact cause of the clinical picture may not always be obvious, but one always exists, and careful search will sooner or later reveal it.

CONCLUSIONS

We have heretofore been too ready to diagnose ulcer, when it did not exist, because the history was typical; but now we have learned how many other conditions may simulate this history, and we demand other data in addition to the patient's story. On the other hand, when the history was not typical, ulcer was not suggested by it and we were likely to overlook its existence because our other means of recognition were so meager. Now we have learned that the patient's story of his ailments is not always the same; that some feel less discomfort from an ulcer than others do; and that only a part of the classical symptoms may be present, even when hyperchlorhydria is found and roentgenograms show definitely a pyloric defect or a deformed cap. The only way to avoid error, therefore, is to trust to no one element in the diagnosis; but to collect our data by history, by physical examination, by laboratory reports, and by fluoroscopic examinations and roentgenograms; and then to piece these data together as a child does the parts of a picture-puzzle, to see what they will ultimately make. Fifty years ago Thomas Huxley wrote:

Sit down humbly before facts as a little child, be prepared to give up every preconceived notion, follow humbly wherever and to whatever abyss nature leads, or you shall learn nothing. I have only begun to learn content and peace of mind since I have resolved at all costs to do this.

THEORIES REGARDING BLOOD PRESSURE

HAROLD W. DANA, M.D.
Formerly Major, M. C., U. S. Army; Formerly Chief Cardiovascular and Lung Examiner, Medical Officer Training Camp, Camp Greenleaf, Ga.

BOSTON

For a number of years, there have appeared from time to time books by various authors purporting to give to the reader a complete understanding of blood pressure, the causation and the measuring of changes in the various factors, and the blood-pressure conditions to be found in all diseases. Independent observers have also put forth formulas for the determination, by means of blood-pressure estimation, of cardiac efficiency.

Recently I have had the opportunity of studying blood-pressure findings in a large number of army officers and candidates for commissions in the army, chiefly in men over 30 years of age, medical officers being in a large majority. I am not here presenting statistics as to the distribution of these cases among different age periods, or as to the classifying of the blood-pressure readings obtained; for, while these figures might be of interest, it does not seem to me that such statistics help us to understand the conditions presented by the individual. What I wish to do is to point out some of the conditions met in this mass of material, to discuss the interpretation of the findings, and to suggest a new point of view in the analysis of blood pressures.

Most of the medical officers examined came to camp from a considerable distance, and were examined the day after their arrival, without opportunity for rest after the long journey. Being physicians, they were almost universally very nervous over the ordeal of the examination. Most of them were naturally constipated, and this constipation was increased by the journey, by the change of routine, and by the change in diet. To many, sleep under camp conditions was at first difficult. From all of these causes, it was not surprising that a large number of the candidates showed an elevation of the systolic blood pressure. In a great majority of such cases, however, rest, catharsis, and the fact of becoming accustomed to the new routine of life, soon brought the blood pressure down to within normal limits. It served to demonstrate in a very striking way the effect of overwork, nervous strain, psychic stimulation, and constipation, in raising blood pressure.
One fact that impressed me particularly is the frequency with which one meets a familial hypertension. Such a condition of continued elevated systolic pressure, in which most members of particular families share, the tendency apparently being hereditary, does not seem in such families to cause invalidism or to shorten life. Indeed, it has seemed to me that many such individuals with a sustained hypertension continue to have better than normal health and robustness; and that the hypertension, if it were not actually the cause of this, at least went hand in hand with their abundance of strength.

With a superabundance of energy and an abnormal vitality, one physician, 48 years old, 6 feet tall, weighing 190 pounds, hard as nails and the picture of health, had a constant systolic pressure of 190 to 200, with a diastolic of 110 to 120. He told me that his father was over 80 years of age, vigorous and active, in spite of a systolic pressure that had been around 200 mm. Hg for years. The officer in question and his brother, three years his senior, had each of them presented similar pressures for years, yet had the best of health. This officer showed a negative urine, a normal heart, no thickening of the peripheral arteries, and normal eye grounds.

Such a condition shakes our faith in any preconceived standard for normal systolic pressures. These familial hypertension cases as a general thing, in my opinion, can be accepted as representing to all intents, and for that particular family, a condition free from serious organic disease. Another type in which there is difficulty in setting down a standard for normal blood pressure is the case in which the hypertension is compensatory to renal or arterial disease. In this general category come those cases in men of 50 years or over in which the blood pressure has assumed a probably normal and physiologic elevation. Taken by and large, our conception as to what represents an unduly high systolic blood pressure in a given individual must take a good many facts into consideration, must be highly individualized, and must have considerable latitude, both as to the standard accepted and the interpretation to be placed on deviation from the standard.

Certainly I am not at all willing to concede that a high blood pressure, for example, 200 mm., means necessarily any of the things that we have always agreed that it did mean. It does not seem to me a proved fact that marked hypertension necessarily causes apoplexy; that it necessarily increases the probability of apoplexy, or of renal or arterial disease, or of ill health of any kind. If marked hypertension means of a certainty any of these things, why do some men live to far beyond the average age, in spite of continued marked hypertension of long duration?.Granted that some pathologic condition would have been found present after death in these cases; granted that signs of nephritis or of arterial degeneration might have been present, proof is still lacking that the hypertension was the result of the lesions found; for, after all, if there were not some cause for the termination of life, these fortunate beings would have lived forever; and in my opinion any man who enjoys reasonably good health and an active life—as many men with marked, continued hypertension do—until past 75 or 80 years comes to his end for the reason that his body is not immortal and is constructed to last for only seventy years or thereabouts.

Why is it that a considerable proportion of men with sclerosis of the peripheral vessels have a normal blood pressure, and that in a similar proportion of cases of hypertension there are soft radial arteries? Is it not true that we are putting undue weight on the occasional coincidence of hypertension and arteriosclerosis?

We have had certain standards set down for us by the pioneers in sphygmomanometry, most of which we have accepted blindly. Unfortunately, there has been little opportunity for exact observation correlating physiology and pathology along this line, and it seems to me that most of the supposed facts of blood pressure that we accept are still open to proof and subject to doubt.

Probably we can accept without serious question that the systolic pressure represents the point at which sounds are first heard with the stethoscope over the cubital fossa when the pressure in the cuff is dropping, and that the diastolic pressure is the lowest point at which these sounds pass their maximum, that is, the beginning of the "fourth phase."

What does the systolic pressure represent? We have been taught that a constant high systolic pressure indicates a permanent change in the capillaries, particularly the renal capillaries. Yet, if such be the case, why is it a fact that some patients with high systolic pressure may have their blood pressure lowered and kept at a normal level by repeated treatments, either with high frequency currents or with radium emanations internally? Incidentally, I would point to this influence of the high frequency currents or radium emanations on blood pressure, through their action on metabolism, presumably in oxidizing or otherwise destroying toxic products in the body, as supporting my theories as to the causes of individual blood-pressure readings. If there is actual change in the renal circulation, of such a nature that the capillaries can no longer dilate, then it must be a fact that an increase in systolic pressure is necessary to drive the blood through the kidneys. But it is a fact that many patients with contracted kidneys and high systolic pressure may be freed from symptoms for years and have a blood pressure normal for their age, as the result of treatment. This, to my mind, is explained by the theory that the systolic pressure, while a measure of peripheral resistance, and as such, a compensatory mechanism in these cases, is not the result of permanent changes in the renal capillaries, but is the effect of vasocostriction, due to toxins circulating in the blood. Many cases of acute focal infection give rise to a temporarily increased systolic pressure, to be accounted for in the same way.

Similarly, many cases of hyperthyroidism present an elevated blood pressure. The effect of nicotine in raising systolic pressure is well known. It has been demonstrated to my satisfaction that the presence of constipation causes a rise in blood pressure. To my mind, there is a definite syndrome of slight cyanosis, increased aortic second sound, and increased blood pressure, that goes with many cases of intestinal stasis. Furthermore, I am certain that many persons leading a sedentary life can have daily bowel movements and still be constipated.

My argument is, that the systolic blood pressure is maintained by vasocostricting substances in the circulating blood, and that an abnormally high blood pressure indicates that the blood stream contains either
toxic substances (whether unexcreted products of metabolism or of focal purulent processes) or an excessive amount of the vasoconstricting secretion of particular glands of internal secretion. The functional test of cardiac efficiency suggested by the observation of Graupner that when a man has been put through a certain amount of exercise, and when following this his pulse rate has returned to normal, his systolic blood pressure as a rule rises, would seem to me to be better explained by the theory that there was an increase in the amount of vasoconstricting internal secretion, liberated perhaps by the thyroid or suprarenal glands as a result of the increased circulation following exercise, rather than on the basis of the condition of the heart muscle.

In further support of this argument is the condition that one finds present in many cases of small-lunged emphysema, usually with chronic bronchitis and asthma. In many cases there is present a constant hypotension. Certainly some patients with severe bronchial asthma are much benefited by repeated administration of epinephrin. While as yet I have had no opportunity of working out my theory along this line, the known facts would seem to favor the existence in such asthmatics of a deficient secretion of vasoconstricting substances.

With regard to the meaning of the diastolic blood pressure, I feel that our present conceptions are even more wide of the mark. Observers in general feel that the diastolic pressure represents the power of the heart to maintain the circulation. In aortic regurgitation, in hyperthyroidism, and in "irritable heart of soldiers," we may have a greatly lowered diastolic pressure without necessarily any actual failure of the muscle power of the heart. Many clinicians feel that a diastolic pressure above 100 mm. indicates a myocardial defect. With this point of view I cannot agree. I believe, as do many others, that a systolic blood pressure of 150 or 160 mm. may be normal for a man 50 years old. To my mind, the ratio 2:3 for the diastolic and systolic pressures, respectively, should be maintained by the normal heart regardless of the rise or fall of pressure; and with a pressure of 160 mm. systolic, I believe that an intact circulation would show a diastolic pressure of 105 to 110 mm. The relatively lowered diastolic pressure in thyrotoxicosis and in "effort syndrome" may perhaps be explained either as a vice of internal secretion, or perhaps in the case of "effort syndrome," as the result of intestinal stasis. In aortic regurgitation we may perhaps explain the low diastolic pressure as due to fatigue either of the vasoconstrictor centers or of the vasoconstrictor muscles. However, the explanation of the blood-pressure phenomena in aortic regurgitation seems more difficult than under the other conditions; it is a condition set off by itself, in which no rule seems to hold good. So far as any theory may be formed regarding aortic regurgitation, this condition helps merely to throw doubt on all other theories as to the significance and causation of changes in the diastolic pressure.

The more I study blood pressure, the less sure I become of any of the accepted interpretations regarding the test. Certainly, while I have as much respect for blood-pressure readings as ever, I feel that we must get a new conception as to the factors influencing the readings.


CONCLUSIONS

1. It is believed that increased systolic blood pressures indicate the presence in the circulating blood of either unexcreted putrefactive products absorbed from the intestine, from the kidneys, from focal infections in the dental alveoli, the nasal sinuses, the tonsils, the genito-urinary tract, or of secretions in abnormal amounts from the glands of internal secretion.

2. It is believed that in some cases at least, a lowered systolic blood pressure indicates a defective secretion of pressor substances, or an increased secretion of depressor substances by the ductless glands.

3. It is believed that the diastolic pressure, when it fails to conform to its normal ratio with the systolic pressure, is also influenced by abnormal products of metabolism or by abnormal amounts of ductless gland secretion in the blood stream.

4. It is not believed that either the systolic or the diastolic blood pressure gives any certain indication as to the condition of the cardiovascular renal system as such; and that when changes in the vascular system are accompanied by hypotension, neither condition is secondary to the other, both being held to be secondary to the presence of unexcreted toxic products of metabolism in the circulating blood.

A HOSPITAL EPIDEMIC OF STREPTOCOCCIC SORE THROAT WITH SURGICAL COMPLICATIONS

J. J. KEEGAN, M.D.

Lieutenant, M. C. U. S. Navy

CHELSEA, MASS.

The object of this report is to call attention to an unusual epidemic of throat infection in the surgical wards of a hospital, caused by a peculiar hemolytic streptococcus and sharply differentiated from the streptococcus bronchitis and broncho-pneumonia which assumed epidemic proportions during the winter of 1917-1918 and has been present to a less extent during the winter of 1918-1919.

PREVIOUS EPIDEMICS

Streptococcal sore throat, septic sore throat, epidemic sore throat and milk-borne sore throat are terms used synonymously to denote the type of epidemic disease here described. The first description of a similar disease in the United States was that of an outbreak in eastern Massachusetts in May, 1911, although in England writers had recognized and reported outbreaks previously. Further outbreaks in the United States were reported from Chicago in December and January, 1911-1912, and from Baltimore in February, 1912. All of these epidemics were attributed to an


