Chronic endarteritis, and its clinical and pathological effects (chronic Bright's disease) / by Arthur V. Meigs.

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EXTRACTED FROM THE

TRANSACTIONS OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA.

THIRD SERIES, VOLUME XI.

CHRONIC ENDARTERITIS, AND ITS CLINICAL AND PATHOLOGICAL EFFECTS.

(CHRONIC BRIGHT'S DISEASE.)

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[Read June 5, 1889.]

Chronic endarteritis is as much a disease by itself—a separate entity—as are typhoid fever and measles. It is in the highest degree important that this fact should be recognized, and that the disease should be transferred from the position it now occupies among the obscure and little understood pathological curiosities, to the rank it is sure soon to claim for itself among the common and well-known complaints of every-day life. Until clinicians ordinarily are prepared to diagnosticate it during life and to announce it as a cause of death among their patients, its frequency of occurrence and importance will not be fully perceived. The name by which a disease is called is not an unimportant matter, for, if a name be accurately descriptive it is well received, and those who hear it easily comprehend what is meant, and thereby knowledge is rapidly diffused. The name "arterio-capillary fibrosis," proposed a good many years ago by Gull and Sutton, has never been commonly accepted, for the complaint cannot justly be called a fibrosis, and disease of the capillaries has never been proved to be an essential part of the process, and therefore their description of the pathology was incorrect. The fact that our knowledge of the complaint is still most incomplete is no more reason for longer delaying to receive it

among ordinarily recognized diseases than it would have been right for Bright to withhold his great addition to the sum of pathological knowledge because there was still more to be learned in the same line of thought. Since there has come to me the feeling that I have more understanding of the pathology of the disease than formerly, the clinical management of cases often occasions a good deal of difficulty by placing me in an awkward position with my patients when they or their friends ask: "What is the disease with which they are suffering?" To say Bright's disease in every case in which albumen and casts are found in the urine would be very far from a correct expression of my understanding of the malady, for the instant conclusion of the public is that the patient has a disease which is as incurable as cancer, and one which will terminate in death almost as soon; whereas in many cases I *believe* that complete recovery, so far as the further progress of organic change is concerned, takes place, and I *know* that in many others the patients live in comfort for long periods of years. Such being the case, a name is needed by which to designate the disease, and it must be one from which the word kidney will be excluded, for it may be asserted that it is now known that the disease does not necessarily involve kidney change, and, if it does, that such change is only a very small part of the whole, and is subsidiary to something else. The pathological lesion which is always present, and which, so far as investigations made up to the present time show to be most widely diffused through the organs and tissues of the body, is an inflammatory thickening of the *intima* of the arteries and arterioles, and, whether the future will show this to be the primary and original change and the true cause or not, it is so widely diffused and so important that to call *it* the disease seems to me much more correct and descriptive than any other that has been proposed. Chronic endarteritis, a simple inflammation of the lining of the arteries, is the process by which the disease begins and thence extends to almost any degree throughout the arterial system, to some degree into the veins, and probably into the capillaries, as has been proved by the

observations of others and my own.[1] This name should be modified to meet the needs of individual cases, as the pathological lesion attacks one or another or several organs, by saying—chronic pulmonary endarteritis, chronic cerebral endarteritis, chronic cardio-renal endarteritis, or other combinations as may be required to make the name accurately descriptive of the variations of one case from another.

To illustrate somewhat the clinical aspect of the subject, a few cases will be, as briefly as possible, given to show that the disease may be recognized during the life of the patient by physical and rational signs as well or better defined than are the diagnostic features of many well-known diseases. The cases will be grouped strictly according to their clinical features and the order of succession of symptoms, showing the disease to have taken possession of one or another or several of the important organs of the body:

Case I.—A man about fifty years of age after a year or two increasing very much in weight and becoming inactive, though previously very strong, was seized in the night with copious epistaxis, a thing he had not previously had for thirty years. Examination of the urine showed the presence of a trace of albumen and very numerous casts, and a month later he had cerebral hemorrhage, of which he died in three days.

Case II.—A man about sixty-seven years of age, who had always been typically strong and healthy, was taken with severe epistaxis several times within a day or two, a thing he had not had in fifty years. Physical examination of heart and lungs was negative, but there was some stiffening of the radial arteries. There were neither casts nor albumen in the urine, but slight œdema of the ankles was present. This observation was just made, and what shape the disease will take remains for the future to decide.

Case III.—A woman of about sixty-five, very strong and well preserved, was seized with severe epistaxis; examination of the urine demonstrated the presence of albumen and casts. From this time she enjoyed quite good health except that occasionally she would be vaguely out of sorts when albumen and casts would be present in the urine, though at others absent, until she was seventy-seven, when she had slight right facial paralysis, with partial aphasia and much distress and confusion of mind, of which she was perfectly conscious. The paralysis soon passed entirely away. After this there was gradually increasing loss of memory though no perversion of

[1] The Medical Record, July 7, 1888, and Trans. College Phys. Phila., vol. x., 1888.

mental function, and at seventy-nine she was seized with sudden violent pain in the head, radiating down the back and into the limbs, particularly the lower ones; stupor came on, increasing to coma, and in a week she died.

Case IV.—A woman about fifty-nine years of age, who had always been unusually strong except that she was subject to take cold and have quite violent cough, was seized with epistaxis which recurred several times within a week or two. Examination of the urine revealed the presence of a trace of albumen, and after careful search by two observers, one of them found one or two casts. Physical examination was negative, the lung and heart sounds being natural, though when she had cold and cough, bronchial râles would be present. From that time to the present she has been very nervous, complaining of many vague sensations and having became visibly much older. She cannot walk freely and with comfort, as her steps are always short and uncertain and she tires easily. Now, two years later, there is a distinct and rather harsh aortic murmur which is systolic in time, and this, though she has never had any acute illness in the two years that have elapsed. A trace of albumen is constantly present in the urine, and occasionally a few casts.

Case V.—A man of about sixty, in the hospital ward, suffering with typical chronic Bright's disease—dropsy, albumen and casts in the urine, hydrothorax, and great hypertrophy of the heart—declared upon most careful cross-examination that the first deviation from health with which he had suffered was a dispostion to take cold readily and have violent cough, and this for some time before any of the other conditions mentioned came upon him.

Case VI.—A man about sixty-six, was for a year or two subject to violent spasmodic cough with much expectoration; he had bronchitis and some enlargement of the heart and œdema of the ankles, but neither albumen nor casts in the urine; five years later he is still living, but there has been in that time no opportunity to see or examine him.

Case VII.—A man of fifty-six, previously healthy, except that he had at times passed gravel, had bronchitis and coryza with more oppression than the physical signs seemed to account for. Examination of the urine revealed the presence of albumen and casts. For four years subsequently albumen and casts were found from time to time upon examination, but since that period there has been no opportunity to test the urine. With the exception of much rheumatic stiffness and a tendency to take cold readily he is still living and healthy at seventy-one years of age.

Case VIII.—A man, previously very healthy, had, when about sixty, an attack of pneumonia of subacute character. Since that time he has never been the same, having lost his activity and strength, and now, at sixty-seven years of age, his condition is as follows: He has lost a good deal of weight; some of the arteries are rigid; he is very easily fatigued, and complains that his legs are almost paralyzed, so great an effort is it to him to walk, though to all

appearance his gait is natural enough, except that it is rather shuffling. The heart-action is rapid and feeble. Neither albumen nor casts have been found in the urine.

Case IX.—A woman of about sixty-five complained of not being well, though she was not confined to bed. Physical examination revealed dulness over one lung posteriorly and very harsh bronchial respiration, but no moist sounds or râles of any sort and no expectoration—plainly a case of pneumonia. There was a slight mitral valve murmur, and the urine contained albumen and casts. This condition of affairs continued, and she gradually failed, though at no time having any urgent or alarming symptoms, and after four months died very quietly.

Case X.—A lawyer of fifty-four, in full practice, had an attack of acute catarrhal pneumonia. He was ill for several months, and during the whole time there were albumen and casts in the urine, and for between two and three years afterward. After that they disappeared, and eight years subsequent to the attack the urine was found to be healthy; since then there has been no opportunity to examine it, but the patient is still living, appearing to be healthy, and practising his profession at sixty-eight years of age.

Case XI.—A man, of about sixty-six or sixty-seven, who had always been well, for a few months did not seem quite himself in strength, and finally had some cough. Examination revealed the presence of subacute pneumonia and albumen and casts in the urine, though there were not then nor subsequently any symptoms which could be attributed to faulty renal action. There was extreme atheroma, so much so that one radial pulse was almost extinguished and the vessel very hard. After a few weeks the lungs cleared and he got about again, though continuing very weak. In about three months there was a return of the pulmonary trouble, with cough and expectoration, and the breathing was quite rapid, though he declared he had no pain, or even oppression. In a few weeks he died, fading out very quietly, without violent or urgent symptoms of any kind.

Case XII.—A man, who is now about eighty-four, and, for his age, quite active, has for the past fifteen years had very frequently albumen and casts in his urine, and during most of that time his breathing has been somewhat wheezing, with bronchial and asthmatic râles to be heard in the chest.

Case XIII.—A man, who inherited gout, was, for ten or twelve years, subject to the arthritic form. When between fifty-seven and fifty-eight years of age he began to have some shortness of breath, and six months later had one night an attack of oppression so violent that it was thought he would die. Albumen and casts were found in the urine, and from this time he failed, and died at sixty years of age, having spent the last eight months of his life in the house in most shocking suffering from general dropsy and great oppression, and latterly quite insane (not delirious), being very suspicious and having marked delusions. The heart was greatly hypertrophied

and the arteries diseased (see Figs. 1 and 2), and once he had a slight uræmic convulsion.

Case XIV.—A woman, of about sixty, had for a good many months been getting less and less able to walk. Examination revealed the presence of albumen and casts in the urine, stiffening of the mitral valve, and a tumor in the right hypochondrium, which was thought to be due to gall-stones in the bladder; this, however, she had had for a good many years, and it seemed quite latent so far as giving rise to any irritation was concerned. Many of the arteries were stiff. She walked very feebly and with a shuffling gait, hardly lifting the feet at all, though there was no true paralysis. The feebleness in walking increased, and the legs wasted until the calves could almost be spanned with the thumb and forefinger, and it was with difficulty that she could even cross a room. One day she fell upon the floor, simply losing her balance, and broke the neck of one femur, and never was able to walk again. After this, and before too, her intellectual powers became less and less, she lost memory to a great degree, and at last became mildly insane, having delusions. She failed gradually, and after two years died, without having had any violent or active symptoms beyond those mentioned, except that for some time before death there was marked general hyperæsthesia.

Case XV.—A woman of about sixty-two has, during the past six years, been subject to attacks of sharp fever which prostrate her very much and keep her from one or two days to a week in bed. At these times albumen and casts have very frequently been found in the urine. She has the signs of slight mitral valve stiffening. Except when suffering with an acute attack she lives a very active life, doing a great deal of charitable work. During the last eight months she has suffered with most persistent prickling sensations (pins and needles) in the hands and feet and becomes easily fatigued, complaining that her legs are often so heavy that she can hardly lift or drag them along.

Case XVI.—A man thirty-two years of age, had a little more than two years before his death an attack of acute rheumatism, after which he had always shortness of breath and palpitation on exertion. During the last seven weeks of his life he had all the commonly accepted signs of Bright's disease—dropsy, albumen and casts in the urine, frequent micturition, cough with bloody expectoration, enormous hypertrophy of the heart with aortic regurgitation, great oppression and difficulty in breathing, violent vomiting, and pain in the belly. He died in great suffering. (See Fig. 3.)

Case XVII.—A man who during all his life has been very strong was taken at about sixty-three years of age with attacks of vertigo. Examination showed that his heart acted with the most extreme irregularity, but that there was no sign of valvular disease and the urine contained neither albumen nor casts. These symptoms continued for about one year, but during the last year, he being now about sixty-five, the condition is much improved, as he seldom has any vertigo and the heart is no longer very

irregular though occasionally intermittent in its action. There is some stiffening of the arteries.

CASE XVIII.—An infant that was deserted by her mother when between two and three weeks old, having already before this time lost weight and failed in general appearance and condition instead of gaining, was placed in an institution. The urine was never examined. In spite of all that could be done for her she soon presented the appearances of marasmus in a most typical manner, and at last, after having for a good while looked like an old, dried-up woman, she died at five months of age. (See Fig. 4.)

These cases were selected because they presented, in a typical manner, the clinical symptoms it has been my desire to group together so that anyone who will study the subject can readily and with precision make a diagnosis of the disease. In Cases XIII, XVI, and XVIII autopsies were made, and the arteries figured in the plates were taken from them. I could have given a larger number in which autopsies were made and the lesion found, and such specimens are still in my possession, but the three only were introduced because they are fair types of the lesion and the introduction of more would have made this paper too long, especially as my desire has been to deal now more with the diagnostic and prognostic features of the disease, and to touch upon the pathology, with which I have dealt more at length elsewhere (*Trans. College of Physicians of Philadelphia*, vol. x., 1888, and *Medical Record*, July 7, 1888), only so much as was necessary to make plain my meaning, without going so deeply into pathological technicalities as to fatigue the ordinary practitioner. Only three of the cases (V, XVI, and XVIII) were hospital patients, the rest being taken from the records of private practice, and were selected because they were under observation in almost every instance for long periods.

Cases I, III, VII, X, and XII have already been alluded to in a paper which appeared in the *Transactions of the College of Physicians of Philadelphia*, vol. vi., 1882, entitled "Clinical Observations on Albuminuria, based upon a study of sixty-two cases seen in private practice."

One of the cases (XVIII) may be thought to have been improperly placed in the group, as it was one of ordinary in-

fantile wasting, or, if the name be not objectionable, marasmus, and it presented no unusual clinical features or symptoms; but it has so long seemed to me that there must be some parallelism between the disease under consideration in adults and some of the cases of infantile wasting that, upon finding the pathological lesion so typically developed (see Fig. 4), I added the case to the group; where from the pathological standpoint certainly it justly belongs. Infants dying in this way grow to look so exactly like those who die of old age that every one who has written upon the subject has remarked upon the similarity of the appearances presented, though the two modes of death are the antipodes of human existence. Upon previous occasions I have published three papers on this subject, and it will be my endeavor not to repeat unnecessarily what will be found in them. They are as follows: "Clinical Observations on Albuminuria, based upon a study of sixty-two cases seen in private practice," *Trans. College of Physicians Phila.*, vol. vi., 1882; "Cardiac Degenerations and Bright's Disease the Result of Changes in the Intima of the Arteries and Veins," *American Journal of the Medical Sciences*, June, 1888; "A Study of the Arteries and Veins in Bright's Disease," *Trans. College of Physicians Phila.*, vol. x., 1888; and the *Medical Record*, July 7, 1888.

Symptoms and Diagnosis.—The symptoms of this disease are of such a nature as, in marked cases at least, to make the diagnosis an easy matter. Perhaps these symptoms cannot be better elucidated than by passing them in review and trying to collect them together in such groups as they would seem most naturally to fall into, for to my mind certainly they present a clinical picture which is most striking and characteristic. Hemorrhage, whether it appear in the form of epistaxis or of cerebral apoplexy, is a marked symptom and one which should always rouse the physician to investigate whether endarteritis has not taken possession of some part of the vascular system of the patient. Epistaxis was the earliest symptom in the first four cases narrated to draw attention to any deviation from health in the patients. Any externally distinguishable stiffen-

ing of the arteries, commonly called atheroma, is to my mind positive evidence of the presence of endarteritis, for I have yet to examine an atheromatous artery exhibiting diseased middle and external coats and a healthy intima. Many of the commonly received symptoms of kidney disease, though they do most certainly indicate that that organ has undergone grave pathological change, causing damage to a greater or less degree, and which will be permanent and irreparable, show much more, if the warning is but heeded, that extensive and often much more dangerous degenerations are taking place in other and perhaps far-away organs and tissues of the body. Occasionally in patients affected in this way there may be from time to time attacks of fever, and at such times albumen and casts may be found in the urine, as in Case XV.

The various manifestations of disturbance of cardiac function are among the most constant accompaniments of endarteritis in any form, and as far as present knowledge leads us to a comprehension of the subject, among the most important and of greatest gravity. It has long seemed to me the merest folly in all cases of demonstrable cardiac valvular disease to set it down as caused by rheumatism or chorea, or even to over-exertion of the body, as is so commonly done by clinicians, or, on the other hand, to believe that rheumatism commonly induces endocarditis and stops at that. Cases IV and XVI are types of what may happen in these respects, and as they afford positive evidence of what sometimes does take place, they are of more value than many negative cases, for it is an axiom in logic that it is hard to prove a negative. In Case IV the patient had no cardiac valvular disease two years ago, now she has positive evidence of aortic stiffening, and yet during that time she has never had any acute attack of illness, but has most positive signs of endarterial inflammation—stiffening of the superficial arteries, albumen and casts in the urine, the valvular heart disease, great nervousness, with loss of muscular power, and general rapid aging in every respect, all coming on after a violent series of attacks of epistaxis which ushered in the loss of health. Most certainly the valve change was not

due to rheumatism or chorea or overexertion of the body in this case, for no one of these possible causes has obtained, and if it be not granted that the inflammation that certainly has existed in the lining of the arteries has crept slowly and silently into the heart, there to accomplish the same fell work that it has undoubtedly done in the vessels, no other more reasonable explanation at any rate can be offered. The history in Case XVI seems equally positively to show that the effects of rheumatism by no means stop after having injured the endocardium and valves, for in that case, from the time of the acute attack, the patient was no longer well, and in about two years died of Bright's disease, with a typical development of the symptoms and pathological lesions (see Fig. 3). Looking at the case from my own standpoint, I feel quite satisfied that the man had rheumatic endocarditis, and at the same time or soon after, and as a continuation of the same process of disease, endarteritis, at first acute and then continuing in a chronic form after he seemed well, and that by this avenue and in this way almost all his organs became gradually involved. It would be easy to accept the old mode of explanation—rheumatic endocarditis, aortic regurgitation, cardiac hypertrophy, increased arterial tension, hypertrophy of the muscular coat of the arterioles, venous congestion of the kidneys, and thus Bright's disease secondary to the rheumatic heart disease. This as an explanation has long seemed to me more than merely theoretical—it is positively fanciful and visionary, and besides, it totally fails to explain many of the most evident and important of the pathological lesions. For the sake of argument, suppose we accept the hypothesis as affording an adequate explanation of the lesions so far as the heart and kidneys are concerned, how are the diseased conditions of the other organs to be explained? The lungs were much infiltrated with cellular overgrowth and their vessels were markedly thickened, especially the intima, the liver in a state of granular disorganization, the bloodvessels of the spleen very thick, and the aorta (though the man was only thirty-two) very atheromatous, beside which the heart exhibited fibroid degeneration and its arteries were

in the last stages of degeneration, not hypertrophy (see Fig. 3). As an explanation for all this is it not more easy to believe, and more consonant, too, with our knowledge of the migratory habits of inflammation, that an endarteritis, rheumatic in origin, acute at first, and then continuing as a chronic process, took possession of the lining of the arteries, and creeping from place to place by avenues so widely open as actually to invite invasion, thus gradually produced the result, than to distract our brains by trying to follow the intricacies of the older explanation, which is so complicated that from the time it was originally proposed it has always been looked upon with suspicion?

Œdema coming on without discoverable organic lesion sufficient to account for its presence is often an indication of the existence of endarteritis (Case II), and the same may be said of vertigo and irregular heart action in some instances (Case XVII).

The fact that symptoms connected with pulmonary disorder or actual lung diseases are often the earliest departures from health in those who afterward die of what has been called chronic Bright's disease, was one of the things which first caused me to feel any doubt of the sufficiency of that mode of explanation of the train of symptoms. Among the cases detailed this is plainly illustrated, for in eight at least, Cases V to XII inclusive, the mode of origin was an attack of pulmonary disease, and six of them certainly were cases that every one would agree to call Bright's disease. This phase of the complaint may declare itself in the form of an attack of bronchitis, either by cough and expectoration without physical signs, or with a full development of bronchial râles, as the case may be, or of catarrhal pneumonia, a disease which is capable of taking upon itself very many and varying aspects, or of asthma or emphysema; or, lastly, there may be one of the forms of oppression, either sudden and violent, or coming on gradually and being very persistent, which are such common and well-known accompaniments of chronic disease of the kidney. These manifestations of pulmonary disorder may be acute and accompanied by great fever

and general disturbance of the system of such a character as to constitute an acute illness confining the patient to bed, or their onset may be so insidious as to attract little or no attention until a physician discovers that his patient is going about with a lung half solid from pneumonia (Cases IX and XI).

Functional disturbance of the liver is not uncommon, and the frequency with which the bloodvessels are thickened shows it to be likely that it has here too some causative relation. The spleen is incapable of inducing clinical symptoms of which at the present time we have knowledge, but it is one of the organs in which the pathological change of the vessels is most commonly found.

Symptoms manifesting a disordered state of the nervous system have long been well recognized as a common consequence of this disease, and of late several interesting and valuable communications upon the subject have appeared in different medical journals,[1] but the earliest attempt probably to arrive at a scientific understanding of their cause was made in the paper of Gull and Sutton.[2] In these symptoms of disordered nervous function do we find what affords perhaps the strongest confirmatory evidence that the disease has its origin in the vascular system, and at the same time the best proof that the old theory of origin is totally untenable. There has never been even an attempt made to explain the cerebral apoplexy and many other less formidable symptoms of nervous disorder that are so common, and clinically so fully recognized, except in a general way to allude to the many things that increased arterial tension is capable of effecting. The most common of these symptoms—as has already been somewhat brought out in the detail of cases given—are: cerebral apoplexy, general muscular weakness, with shuffling gait but without actual paralysis, and, in some instances, wasting of the

[1] Chronic Bright's Disease (Arterio-capillary Fibrosis) in its Relations to Insanity. By E. A. Christian, M.D. The Journal of the American Medical Association, March 23, 1889.

[2] On Changes in the Spinal Cord and its Vessels in Arterio-capillary Fibrosis. Trans. Path. Soc. London, vol. xxviii., 1887.

muscles; change of temper and disposition, nervousness and decline of mental power, especially loss of memory, and even mild insanity; and in old people in the latter stages of the disease, most commonly after they have got so weak as to be confined to bed, a curious general hyperæsthesia which does not seem to be confined to the skin, for there is great pain caused by any movement.

Rheumatism and gout seem to be near relatives of endarteritis, or at least closely bound up with it. In one of the cases (Case XVI) rheumatism, and in another (Case XIII) gout, seemed to set fire to the train that finally led to the fatal result, and in both, as has already been alluded to, post-mortem examinations were made, and the lesions of chronic endarteritis in a typical state of development found (see Figs. 1, 2, and 3), and they both died with all the ordinarily recognized symptoms of chronic Bright's disease.

Prognosis.—In the clinical management of cases there is nothing which occasions more difficulty than the question of prognosis; for, to any one who has had to deal much with the disease, one thing at any rate is very plain, that many cases that a few years ago would have been looked on as necessarily fatal, recover entirely, and others regain sufficient health to live for many years in comfort. It is wonderful how differently two cases, that at the outset seemed very closely parallel, may terminate, the one perhaps living for many years, and the other dying within a few months; this is exemplified in Cases XI and XII. It does not seem possible to doubt that in Case X, for instance, entire recovery has taken place, though, as albumen and casts were present in the urine and the patient was in ill-health for more than two years, he certainly had chronic Bright's disease; and Case VII is one in which, even if it cannot be positively asserted that complete recovery has occurred, at any rate there has been such a physical condition that for fourteen years the man has had a very enjoyable life. It seems possible to explain this, so far as the kidney is concerned, only by the belief that there was, at the time of the acute attack, a wedge-shaped spot or spots of inflammation with

round-cell infiltration, the broad base of the wedge being toward the cortex, such as are so commonly seen in sections cut from the organs of persons dying in this stage of the disease, and that this has in the course of time healed, after having destroyed a greater or less number of tubules, and left in their place a small scar. Why it should so long have been supposed that the kidney was incapable of getting over the effects of an inflammatory process which is in no wise different from what occurs elsewhere, when it is perfectly well known that every other organ and tissue in the body can undergo regeneration to a greater or less degree, is a thing which is incomprehensible! In view of these facts we should, as physicians, be very slow to give a fatal prognosis, except in cases in which the morbid changes have progressed so far as to make our ground very certain. There is no combination so bad, and none which so fully seems to justify a very gloomy prognosis, as the presence of albumen and casts in the urine, with general dropsy and a heart evidently greatly enlarged, with much shortness of breath. Of these symptoms, to individualize them, there is none which has anything like the grave significance of enlargement of the heart with heaving impulse and constant shortness of breath; people with this combination seldom live more than a few months. Experience has long since taught me to believe that the presence or absence of the signs of valvular change is, comparatively speaking, of very secondary importance, and should have little influence in determining our decision of the probable duration of life, but that all our efforts should rather be turned to endeavor to form an estimate of the size of the heart and state of its walls.

Treatment.—It would make this paper much too long if any attempt was made elaborately to discuss the treatment; beside which, the question does not properly come under its scope as defined in the title. It need, therefore, only be said that the disease is so widespread in its results and produces such varying effects, as the morbid process takes possession of one or another part of the organism, that no special plan of treatment could be laid down which would cover all the dif-

ferent aspects it may present. Drugs which have seemed to me to be capable of effecting much good in certain phases of the complaint are carbonate of ammonium with small doses of digitalis—these should be given three or four times a day and their use persisted in for long periods, often for as much as a year or more. The advantages of this mode of treatment were exemplified in Case XVII.

Pathology.—As was stated, the pathology of this disease has been discussed by me in papers already published, and time fails, nor would it perhaps be desirable to go fully over the subject again. There are, however, a few points to which allusion must be made. To my mind it seems established as a fact and beyond the reach of doubt that many cases of chronic Bright's disease, if not most of them, are in truth cases of arterial disease; this was years ago asserted by Gull and Sutton; now, as a result of more recent investigation, I believe it to be established that endarterial inflammation is an important part of the disease. In reaching this conclusion one more step has been made in the direction of a final understanding of the process, though whether we are now near to this goal or still far away is a question which the future only can determine.

It would be easy, but of no present advantage, to speculate upon whether the endarterial inflammation is but an extension of disease from the blood, the living and moving tissue with which the lining of the arteries is always in contact, or whether the process starts in the connective tissues, in which the arteries lie and of which they are in fact a part, or whether again the inflammation has its true origin in the arterial intima. The probability is that the future will show the arterial inflammation to be but an extension and consequence of some other preëxisting change. Outside of the assertion that the disease begins in the blood—and this is a theory which though exceedingly plausible and not unlikely to be correct, is without a single fact of observation to establish its truth, so little understanding have we at the present time of the blood or its diseases—there has as yet been but one hypothesis offered to explain

an earlier beginning of the disease. This hypothesis, for hypothesis only at present has it a right to be called, was mentioned in one of my earlier papers (loc. cit.), and is that which was in the first instance propounded by Drs. Da Costa and Longstreth[1] and has been further elaborated by Dr. Da Costa in a later paper.[2] These authors, after making post-mortem investigations both macroscopic and microscopic, assert that there is constantly present a lesion of the renal nerve plexus, and Dr. Da Costa in his later paper states that lesions were also found in the cardiac ganglia in cases of Bright's disease. These investigations are of the highest scientific interest and exceedingly suggestive, for from a purely theoretical point of view it is fully as reasonable to suppose that the morbid process might begin in the nervous system as in the lining of the arteries or elsewhere. Investigation of the histological condition of the nerve trunks and ganglia of the sympathetic system offers a field of the greatest interest, but at the same time great difficulty, for, if the truth be told, but little is really known at the present time of its ordinary condition in human adults—that is to say, few if any pathologists have been in the habit of examining the sympathetic so frequently as to be very familiar with its ordinary condition. Further study of this matter is much to be desired, but even if such investigation should demonstrate that a lesion of the sympathetic is as constantly present as the endarteritis, it would still remain an open question which, if either, was the causative lesion.

A point of great pathological significance, and to which allusion has already been made, is that valvular disease of the heart, as recognized by the ordinary clinical signs, may come on in the course of this disease without the patient's having at any time an acute attack of illness, and this is proved by what took place in Case IV, the woman developing valvular heart lesion in the course of an attack of mild chronic Bright's dis-

[1] Researches on the State of the Ganglionic Centres in Bright's Disease. American Journal of the Medical Sciences, July, 1880.

[2] The Middleton-Goldsmith Lecture on the Relation of the Diseases of the Kidney, especially the Bright's Diseases, to Diseases of the Heart. Medical News, May 5, 1888.

ease lasting two years, during which time she had no severe illness or febrile attack—at the beginning being free from valve change, and now having its clinical evidences most unmistakably developed; showing, it seems to me, most positively that the endarterial inflammation has crept into the heart to twist and distort the valve.

It is a curious fact, and one worth recording, though at present I can offer no explanation nor draw any deductions from it, that in several different sorts of tissue removed by operation by surgeons as incurably diseased, I have found upon microscopic examination precisely the same endarterial inflammation and thickening, in some vessels so great as entirely to close their calibre, as found almost universally throughout the body in the cases of chronic endarteritis or chronic Bright's disease that have been described. These have been ovaries and Fallopian tubes removed by gynecologists, a vulvo-vaginal retention cyst, a cancerous uterus, and, I think, other tissues.

The relation of old age to this process of chronic endarteritis, or Bright's disease, I have discussed in former papers, and it does not seem possible longer to doubt that the conditions are in many respects parallel if not identical, and a further confirmation of this view is afforded by the observation I have recorded that a similar condition of the lining of arteries has been found in the heart of an infant dying of marasmus at five months of age (see Fig. 4). The arteries of young infants, just as all the rest of their tissues, present histological appearances which are different from those of adult life, and they may offer, therefore, perhaps an opportunity to learn the true origin of this process of atheroma, as it has been called, which could never be had from the study of adult tissues. The plicated membrane or fenestrated membrane of Henle presents itself in the arteries of young infants, when examined in cross-section, as a folded line somewhat thicker relatively than it is in the tissues of older persons, and in healthy vessels appears to be entirely structureless, containing no nuclei which will take stain; in the diseased artery from

the heart it seems as if this had, owing to some irritation, become inflamed, and there are many proliferated nuclei in the membrane itself, or lying inside of it, producing swellings in the lining of the artery, just as is so commonly seen in the atheromatous processes of adult life (see Fig. 4). It is impossible to avoid drawing a parallel between these appearances and what is so well known and has been so much studied in inflammation of the cornea.

The drawings were made for me by Dr. B. Alexander Randall, and they admirably represent the appearances intended to be shown. As they were to be reproduced by photographic process, they were necessarily made somewhat diagrammatic, for many of the finer lines and shadings in a drawing cannot have justice done them by the photographic method as here applied.

It was my desire in this paper to deal more with the clinical than the pathological side of the subject—to try to formulate the symptoms so that a diagnosis may be more easily made; and last, though by no means least, it is high time to give the disease a name, so that it may be commonly recognized and admitted to our every-day classifications of diseases. To me it seems plain enough, in the present state of clinical and pathological knowledge, that the morbid process is "chronic endarteritis"—and so the disease should be named.

Fig. 1.

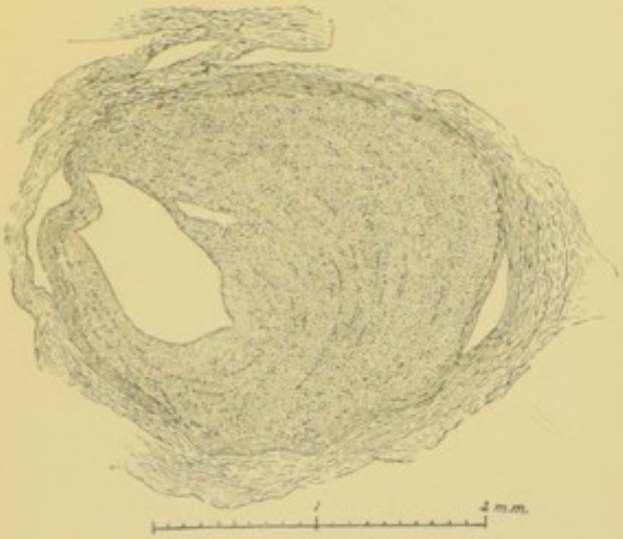


Fig. 2.

Fig. 3.

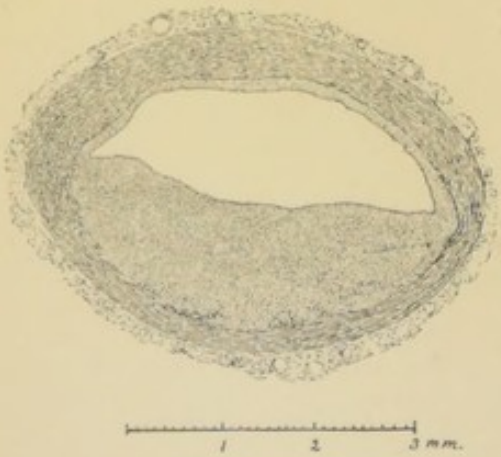
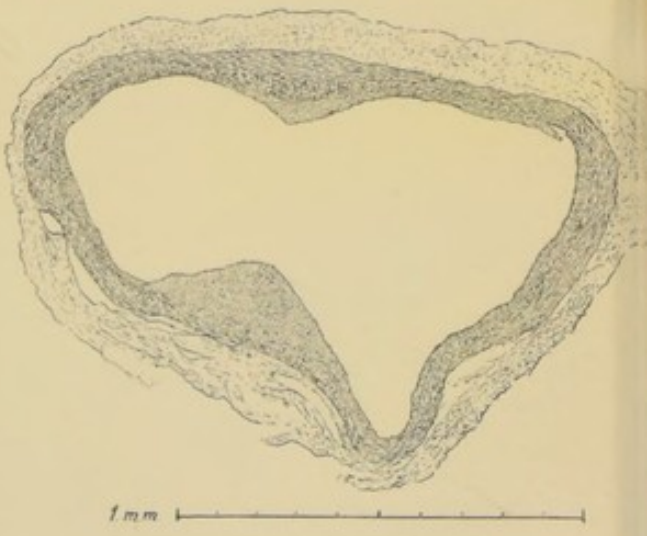


Fig. 4.



EXPLANATION OF PLATES.

Fig. 1, × 17 diameters. Is from Case XIII, and represents the large branch of the left coronary artery of the heart, which descends in the interventricular septum. The calibre of the vessel is almost closed by overgrowth of the intima, the opening which remains being represented toward the left in the picture. The other spaces were either the result of tearing in mounting, or were filled with some material which did not stain and was transparent. The muscular coat is represented by lines, and the adventitia by lighter ones outside; all the material inside these, represented by dots, is the proliferated intima. The part of the vessel represented here contained no calcareous material, though elsewhere there was abundant mineral deposit.

Fig. 2, × 9 diameters. Is also from Case XIII, and represents one of the first branches of the renal artery as it entered the hilus of the kidney. The great and irregular thickening of the intima is very marked, and the plicated membrane is distinctly visible around the greater part of the circuit forming the boundary between the intima and muscular coat.

Fig. 3, × 10 diameters. From Case XVI, and representing also the branch of the left coronary artery of the heart which descends in the interventricular septum. The enormous and irregular thickening of the intima is very evident; the plicated membrane is distinctly visible around half the circuit upon the side of least thickening; upon the same side also the muscular coat is nearly twice as thick as upon the other. There are two explanations for this irregularity of thickness of the muscular coat: the one, that it is hypertrophied upon the side on which it is thickest; the other, that upon the opposite side it has been partially destroyed by the invasion of disease.

Fig. 4, × 40 diameters. The infant was five months old. This is the left coronary artery of the heart just after its origin in the aorta, from Case XVIII. The irregularity of thickening is very evident, and it seems equally positive that the process of disease belongs to the lining of the vessel, for the muscular coat is distinctly visible outside of the area of disease, and does not appear to be very much altered, except that in some places the process seems to have invaded it as well as having produced masses inside the vessel. The plicated membrane is represented by two fine lines with a white space between them, and is visible around the entire circuit, except in the thickening which is represented in the lower part of the picture, where the process of disease has involved the muscular layers to a great extent.