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TRANSIENT ATTACKS OF APHASIA AND PARALYSES IN STATES OF HIGH BLOOD PRESSURE AND ARTERIO-SCLEROSIS

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HEADACHE, vertigo, convulsions, aphasia, paralyses, and a progressive dementia are among the cerebral manifestations of arterio-sclerosis. Death "at the top" may be slow as in the old oak with which Dean Swift compared himself; or it may be sudden, when a vessel ruptures, or more gradual if thrombosis occurs. These may be called the major manifestations, but there are others less serious, but of great importance as their significance may be overlooked or misinterpreted. To headache and vertigo I will not refer since everyone now recognizes how common they are as early symptoms of arterio-sclerosis in the young, and more constant features in the aged. It is more particularly to the transient aphasias and paralyses, cerebral crises as they have been called, occurring in states of high blood pressure and in arterio-sclerosis, to which I wish to call attention. Within a few weeks of each other I have recently seen two cases which illustrate the character of the attacks, and the first case is unusual since so far as could be determined only high blood pressure existed.

A well-built, active man of forty-three, who had driven his engines at a maximum speed for twenty-five years—keenly occupied in business, using tobacco freely, and intensely devoted to Bacchus, Venus, and Vulcan,—returned on the afternoon of March 1st, 1910, to his hotel, rang the bell for the servant and found that he could not speak. Perfectly conscious, he could not say a word, and was very much upset, and still more so when he found he could

not write. He was a little dazed mentally, as he could not tell the time. He became emotional, and the doctor found him crying and still unable to speak. In a few hours he could say a few words, but incoherently. The next day he could talk, but not quite freely. There was no paralysis, no disturbance of vision, and no headache. Within three or four days he was quite well, and could talk perfectly. The blood pressure was found to be 212 mm., and the attack was regarded as possibly a slight hæmorrhage. I saw him on May 18th, 1911, nearly fifteen months after the attack. Very well, except that he has become very nervous and apprehensive, he has given up work, and has wandered about, and has been under the care of a great many doctors.

On examination he was a very healthy looking man of good colour, and good physique. The radial arteries, with the blood current flowing, could easily be rolled under the fingers. The pulse was recurrent, with practically no difference in the fulness of pulsation beyond the point where the artery was compressed. In a section of the emptied radial no arterial wall could be differentiated with the finger. It was the same with the temporals and the brachials. With the blood current flowing they could be rolled under the fingers. Emptied they were not palpable. Neither the brachials nor the femorals were sclerotic. The retinal arteries looked prominent. The apex beat was inside the nipple line. There was no evidence of enlargement of the heart; the second aortic sound was ringing. The blood pressure was 220 mm. in spite of the fact that he had been taking for more than a year nitrites and potassium iodide. He has had no other cerebral attack. His general condition was very good, but he was morbidly apprehensive about his condition.

A very different picture was presented by Mr. ————, aged sixty-two, seen June 6th, 1911; a man who had worked hard in many parts of the world, but had not been a heavy drinker. A wiry, tough-fibred man, he had always kept himself in very good condition, but had used tobacco to excess. One afternoon, just a year ago, while waiting for tea, he went out to say something to the gardener, and to his surprise found it impossible. He did not feel giddy or dazed, and five minutes later he could speak quite well. He returned to the house, and about an hour afterwards some people came in, and to his surprise he could not say how-do-you-do, could only nod and give a grimace. He could see the people were very much upset, and he was mortified to feel that perhaps they thought he had been drinking. He was greatly embarrassed as he could not say a word. The gentleman urged him to see a doctor at once.

He walked out to the garden gate with them but could not say goodbye. He then went across the road to the doctor, but could tell him nothing. He had no headache and he felt quite clear in his mind. In the course of ten to fifteen minutes he began to say a few words, though not quite clearly; in a day or two he could say everything. He remained in bed for a couple of days on low diet. His blood pressure was found to be 200 mm., and for the first time it was found that his arteries were sclerosed. He was a healthy looking man of good colour. He had lost more than twenty-five pounds in weight, and had been very much worried about the high blood pressure. There was an extreme degree of arterio-sclerosis. Brachials, radials, and ulnars were visible in their entire course, with forcible pulsation. The pulse was recurrent; the radial wall, very much thickened. There was not much difference in the sensation given to the finger between the vessel full and empty. Blood pressure, 130 mm. He had worried incessantly about the high blood pressure and had become greatly depressed. This is not the first instance in which I have known worry and loss in weight to be the most effective means of lowering high tension.

My introduction to this condition occurred under peculiar circumstances: As a young man in Montreal there were two doors I never passed,—47 and 49 Union Avenue; going up I called on Dr. Palmer Howard, and if he was not in or was engaged I called on Dr. George Ross; going down, the reverse. Any growth in virtue as a practical clinician I owe to an intimate association with these two men, in whom were combined in rare measure enthusiasm and clear vision. One morning I had a shock, the first of the kind I had ever felt—I realized that my dear friend George Ross was seriously ill. He had always seemed well and strong, though one hot day, in 1878, at the old Savile Club in London, he had an attack of shortness of breath. This day he told a strange story: he had been awakened by the night bell, and, attempting to put out the right hand to get the match-box, he found he had lost power in it. With his left hand he struck a match and rang the bell. When the servant came he could not speak. He realized perfectly what had happened—that he had had a stroke; but to his surprise in a few hours power had returned to his arm, and he could speak, but not quite clearly. When I saw him he was quite himself—no trace of paralysis, and the speech was clear. Arteries like whip-cord—apex beat out—the usual story that we now know so well. This was the first of a series of transient attacks of aphasia, monopelgia, and hemiplegia extending over four or five years, with intervals of good health during

which he lectured and carried on his practice. Once on his return from Europe with Dr. Roddick and Dr. Alloway he had an attack of partial paraplegia and had to be helped off the steamer, but it disappeared in the course of a couple of days.

These not uncommon features of arterio-sclerosis have had an abiding interest ever since. In the first edition of my text-book, 1892, I mentioned that: "transient hemiplegia, monoplegia, or aphasia may occur in advanced arterio-sclerosis. Recovery may be perfect. It is difficult to say upon what these attacks depend. Spasm of the arteries has been suggested, but the condition of the smaller arteries is not very favourable to this view. Peabody has recently called attention to these cases, which are more common than indicated in the literature." The subject had been brought before the Association of American Physicians by Dr. George Peabody at our meeting in 1891, in a very thorough study of the relation of arterial and visceral changes (Transactions of the Association of American Physicians, Vol. VI, p. 170). In one of his cases a man, aged fifty-six, with well-marked arterio-sclerosis had an attack of transient hemiplegia without loss of consciousness. Then in the course of ten days he had four of five attacks in which he lost the power of speech, and had incomplete paralysis of the right side. He died in a very severe attack in which he had complete right hemiplegia with unconsciousness. Extensive arterio-sclerosis was found in the cerebral vessels, but there was no local lesion, no areas of special œdema, or any foci of hæmorrhage or softening. So far as I know Dr. Peabody was the first to offer a reasonable explanation of the condition:

"It seemed to me that there might perhaps have been a spasmodic contraction of the muscular coat of the middle cerebral artery, or of several of its branches; which, in addition to the encroachment upon its lumen, produced by the new growth, was sufficient to cut off blood supply to the parts to which it was distributed; that this had occurred several times, causing each time temporary ischæmia of important brain centres; and that in the final attack it had lasted long enough to produce death, but that it was not complete enough, or of long enough duration, to cause softening."

Peabody urged that as spasm could be seen in the retinal vessels, with transient loss of vision, the same very probably occurred in local vascular areas in the brain causing ischæmia and loss of function. No one has stated the case more clearly, and I am glad to refer to this important, initial bit of work which has not received

recognition except in my text-book. Since then I have seen a score or more cases, which fall into three catergories: (a) Healthy individuals with high blood pressure, but without signs of arterial disease. The first case mentioned in this paper had no obvious sclerosis of the palpable or visible arteries. I have seen only two other patients in which hyperpiesis existed alone—one a man, aged fifty-one, who had numbness of the left side and hemianopia, which passed away in the course of a day; the other a young man, aged thirty-one, who had not had syphilis but who had high pressure and angina and in several attacks loss of power in the left hand with numbness. (b) Patients with well-marked arterio-sclerosis, in whom the cerebral attacks have come on without warning, sometimes as the signal symptom. A majority of my cases come in this group. (c) In advanced sclerosis with cerebral changes, manifested by progressive mental and muscular weakness, all possible types of these transient seizures, including convulsions, may occur. The attacks are most frequent in the aged, but men in the fifth and sixth decades are also affected.

The symptoms are extraordinarily varied, but tend in individual cases to repeat themselves in the attacks. *Transient aphasia* is one of the most common. The account given by the two patients whose cases are here reported is singularly accurate—inability to talk, consciousness of it, no paralysis, emotional disturbance, and, within a few hours, complete recovery. One patient had at least twenty attacks, all of very much the same type. Loss of the power to write and hemianopia may be present.

Sensory disturbances rarely occur alone, but one patient had day-long attacks of numbness of the face and right hand with loss of the finer movements of the fingers. Paræsthesiæ may exist with the aphasia.

Motor paralysis is the most common symptom, and may be hemiplegia, or only the face and hand or arm may be involved. The paralysis, rarely complete, has a transient character, which with the recurrences give it a peculiar stamp. Complete recovery is of course seen in monoplegias and hemiplegias of organic origin, but not in a few hours or in a day. I have had a letter in the evening from a man who at 9 a.m. could not button his shirt collar. In one instance paraplegia of brief duration occurred.

The *mental features* are interesting. Confusion of thought is common and emotional disturbances, which are very natural under the circumstances. The transient attacks of mental aberration—forgetfulness or slight delirium—seen sometimes in arterio-sclerosis

may be the psychical counterparts of the motor attacks, and cases have been reported by Edgeworth and William Russell. Loss of consciousness has not been common in my experience. I saw a patient in Washington whose attacks were always ushered in by a short cry, followed by fainting, and on recovery the right arm and face were paretic and there was transient loss of the power of speech. He had a pulse of sixty, and the question of Stokes-Adams disease was considered.

The mode of origin of these cerebral crises in arterio-sclerosis has been much discussed of late years by William Russell in his work on Arterial Hypertonus, etc., (1907), by Edgeworth[1], Parker[2], Langwill[3] and Heard[4], Allan[5], and others.

An interesting discussion in the *British Medical Journal*, 1909, II, followed a paper by William Russell on "Intermittent Closing of the Cerebral Arteries." When Peabody brought forward the view that in these cases a transient arterial spasm occured, I was doubtful how far this was possible in sclerotic vessels; but I have since come round to his view and I do not think any other explanation is more plausible than that these attacks represent vascular crises.

We have plenty of evidence that arteries may pass into a state of spasm with obliteration of the lumen and loss of function in the parts supplied. In the peripheral arteries in Raynaud's disease we can sometimes feel the spastic, cord-like vessel; in the retina we can sometimes see the arteries contracted. Both in Raynaud's disease and in the remarkable thrombo-angitis described by Buerger the obliteration may persist until necrosis occurs, but in many instances it is only transient and the circulation is restored. A case of Raynaud's disease[6], with recurring attacks of aphasia, hemiplegia, and loss of consciousness, some occuring coincidently with the local asphyxia and necrosis, convinced me that intermittent closing of the cerebral vessels could occur, and the transient nature of the attacks with the complete recovery seems to offer no other explanation so satisfactory. And we know now that there are neither anatomical nor physiological objections to this view as applied to the cerebral arteries.

Transient paralyses in uræmia may be due to œdema, as suggested by Traube (*Gesammelte Beitrage*, Bd. 2, p. 551); but the condition is rarely transient and is more often a terminal event. It is possible that there are cerebro-spinal manifestations in angio-neurotic œdema, as in the extraordinary case which I reported[7] of a physician, aged twenty-nine, who had right hemiplegia and aphasia at the age of

nine, and, within a year, five or six attacks of transient hemiplegia, subsequently migraine, and well-marked attacks of angio-neurotic œdema. Howland[8] has recorded a case of this disease with spinal symptoms. The association of migraine with cerebral symptoms is well known, and Mitchell Clarke[9] has reported a familial form with which hemiplegia occurred in three generations.

The *diagnosis*, usually easy, is based on the existing conditions of high tension or sclerosis or both, the slight and transient character of the attacks, and the recurrences. Slight paralyses due to hæmorrhage or softening rarely pass away so quickly, and it may be weeks before a patient speaks clearly or uses the hand freely. Numbness, tingling, and slight weakness of one side with headache may be precursors of a "stroke" in which case the symptoms are not transient but progressive. In sclerosis of the cerebral arteries small foci of softening are not rare and some of these may produce symptoms. An artist friend motoring in the neighbourhood of Oxford felt so badly in his head that his wife insisted upon coming at once to my house. He was a bit dazed and could not sit at luncheon, but there did not seem very much the matter. He said, however, that he felt "queer in his head," and could not see properly. I could find nothing wrong with the retinæ but I did not take the fields. Two days later he consulted Mr. Lawford who found a quadrantic hemianopia, which has never cleared up. Here no doubt was a definite lesion.

The *prognosis* is largely that of the sclerosis. Patients may live for years and be very comfortable in the intervals. While writing this I had a visit from my old friend, Dr. Litchfield, of Pittsburg, who happened to mention the case of a Mr. L., whom I had seen with him eleven years ago with transient attacks of what Dr. L. called "mutism," often associated with numbness and tingling on the right side. The patient had arterio-sclerosis, and I remember we discussed the possibility of the condition being due to angiospasm. He has had in all ten or twelve of such attacks; they pass off in a few days; associated with the feeling of fulness and headache. Once or twice he has had a transient diplopia. In the intervals he is pretty well, though it is difficult to keep down his blood pressure. An interesting point of which Dr. Litchfield reminded me was that this man's father had had similar attacks, beginning when he was a comparatively young man, and lasting until he was over seventy. Of considerable moment, as illustrating the necessity for a more widespread recognition of this condition, is the fact that Mr. —— a few years ago consulted a well-known heart

specialist, who said that he had chronic meningitis, gave him bromides, and his friends a hopeless prognosis.

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2. *British Medico-Chirurgical Journal*, March, 1909.
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4. *Edinburgh Medical Journal*, November, 1910.
5. *Glasgow Medical Journal*, July, 1910.
6. *American Journal of the Med. Sciences*, November, 1896.
7. *British Journal of Dermatology*, Vol. XII.
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"ONE of the most puzzling problems that confronts a physician is to determine the cause of continued fever in a sick child. An examination of the urine—a catheterized specimen if necessary—should never be neglected. It is surprising how frequently, especially in girl babies, an infection of the urinary tract will be found. In one hundred and twenty-one cases, reported by Jeffreys (*The Quarterly Journal of Medicine, April*, 1911), sixty-seven were due to coliform organisms, thirty-seven to staphylococcus, ten to streptococcus, three to pneumococcus, and four to other organisms. Of sixty cases reported by the same author, fifty-three were in females and seven in males. This preponderance of cases in the female sex, together with the fact that the infection of the urinary tract often follows bowel trouble of one kind or another, suggests strongly an ascending infection from the urethra, the genitalia being soiled from the napkins. Another possible cause is by the lymphatics from the bowel. The right kidney is much more frequently infected than the left and the lymphatic connexion between the former and the ascending colon, cecum, and appendix is very close. This Jeffreys believes occurs quite commonly and he quotes a number of cases which seem to bear out his contention. A third mode of infection is by the blood stream, but this certainly must be quite rare. Infection of the urinary tract in children must not be looked upon as a trivial complaint, as the average mortality is about ten per cent. The treatment consists in the administration of alkalis, urinary antiseptics, the best of which is urotropin, and in some cases local treatment to the bladder, It is very important to treat the bowel condition. In stubborn cases the writer has seen good results from the vaccine treatment."—*The Cleveland Medical Journal.*