THROMBOSIS OF THE MIDDLE CEREBRAL ARTERY.¹

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SUNDAY evening, June 27th, S. P., a medical student, aged twenty-five, was sitting on the door-steps of a friend, when he was observed to raise his hand suddenly to the left side of his head, and to give a suppressed groan. He immediately rose to his feet and staggered into the house, across a front room into a back room, and threw himself upon a lounge. He was unable to utter a word, but he called attention to his paralyzed right arm and leg by lifting them with the corresponding members of the left side. A few minutes later he vomited, and became restless. I saw him an hour after he fell. I found him tossing his head and body to and fro continuously, at one moment grasping his right wrist in his left hand, at another pulling a pillow over his head and face as if to protect them from the air. There was very marked pallor of the face. He was unable to make a single articulate sound, and he did not appear to comprehend anything said to him. He could not protrude his tongue nor swallow liquids. The entire right side of the body, including the face, was paralyzed. The pupils were moderately dilated; temperature normal; pulse 50, rather full; respiration irregular and sighing; perspiration profuse.

June 28th, nine A. M. Patient remained restless all night. He swallows liquids this morning; temperature normal; pulse 50. He urinates on being lifted to a vessel. Sensation is partially intact in the paralyzed parts. Eight P. M. He protrudes the tongue by request, which turns to the left.

June 29th, nine A. M. He appeared to sleep at intervals during the night; he voided urine twice when requested to do so; he recognized friends. On being requested to move his right hand he picked it up with the left one. When requested to draw up his right leg he worked the toes of his left foot under the right heel and attempted to comply. Temperature normal; pulse 50, and full; takes nourishment well. Eight P. M. He grew worse after noon; he does not comprehend anything said to him; very restless; respiration sighing; temperature normal; pulse 60.

June 30th, nine A. M. He was restless during the night. The breathing grew heavier up to six o'clock, when it became decidedly stertorous; the temperature increased perceptibly about midnight, and is now 101° F.; perspiration has ceased; he has not swallowed anything since last evening; voids his urine unconsciously; pulse 120, weak; flexor muscles contracted; extremities cold; he lies perfectly quiescent; eyes fixed, pupils widely and equally dilated.

He died two hours later, sixty-five hours after the attack.

Previous history. This patient was born cyanotic, but he gradually recovered at the age of four months. After this age he had what his mother described as "large purple spots" on various parts of the body. These spots appeared at irregular intervals up to the age of three years, after which they were not noticed. He was now taken to the West to live, where he had malarial fever in its worst phases. He became pale, weak, and dropsical. Two years later he was so feeble

it was found necessary to return to the East with him. He soon began to recover under the change of climate, and became a bright, active, healthy, and studious boy, entering college at the age of seventeen. From this time onward he pursued his academical and professional studies without interruption. He seldom complained of malaise of any nature whatever. He was, however, of a somewhat taciturn nature. He was a very sound sleeper, and he had the peculiarity of sleeping across the bed with his head hanging over the side. He was subject to attacks of profuse nosebleeds. These attacks frequently happened in the night. On several occasions during the six months previous to his death he had several attacks, the real nature of which he would never explain to his relatives. He appeared for a moment to be out of breath, placing his hand over the region of the heart as if in pain. He always recovered in a few moments, if not from the distress, at least his presence of mind, and said it was nothing. These attacks happened two or three times last spring while at the dinner table. He would arise suddenly, clap his hand to his left side, then in a moment resume eating, and turn the conversation from himself. He also had several attacks in the night, which caused him to spring from his bed. These were all that were observed by his friends. On two or three occasions during this period the middle finger of the right hand became numb, and as white as if every drop of blood had been extracted from it. At the same time severe pain shot up the forearm, sometimes reaching to the shoulder. After an hour of rubbing and heating circulation and sensation were restored. He confided to a friend his opinion that these phenomena were due to embolism of one of the arteries of the affected finger. He at the same time predicted that he should die of embolism!

For several weeks previous to this fatal attack, Mr. P. had been hard at work in a machine shop to earn money to meet the expenses of his last course of medical lectures the coming winter. The Sabbath on which he fell and the several previous days were excessively warm and oppressive.

Autopsy, thirty-six hours after death. The subject was of light complexion, medium height, rather spare, but of exceptionally fine and uniform muscular development. Abdominal organs normal; lungs normal; pericardium, normal; heart and large vessels filled with dark fluid blood; chambers of the heart absolutely free from any indication of disease, past or present; but there was no right auriculo-ventricular septum. The two chambers made one continuous conical cavity. The tricuspid valves were laid plaited out closely against the expanded walls, while their lower margins were pulled down smoothly by the tense chordæ tendineæ; but they were nowhere adherent nor in any way diseased. The fossa ovalis was normal. The pulmonary vessels and aorta, together with the other thoracic vessels, were perfectly healthy. The carotids were not examined. The left heart was certainly very muscular, and it seemed to me somewhat hypertrophied, but other physicians present at the autopsy thought its walls no thicker than they should be in a man so muscular and accustomed to vigorous exercise.

The brain. The dura mater was feebly adherent to the left second frontal convolution, evidently the result of very recent inflammation; sinuses free of clots; diffuse extravasation of blood and lymph beneath the sheath of the superior longitudinal sinus, extending

¹ Read before the Rhode Island Medical Society, September 18, 1880.

along two thirds its length, from the torcular forwards; nothing unusual in the appearance of the surface of the brain; a few drops only of serum either in the membranes or ventricles. The middle cerebral artery, with all its branches, was filled with coagulated blood. The clot was confined to those portions of the arteries lying in the fissure of Sylvius. The clot in none of the branches extended beyond the points of entrance into the brain substance. Beyond these points the vessels were empty and collapsed, but nowhere obliterated. The lining of all these arteries was of a bright red color, softened and wrinkled longitudinally. Just within the origin of the middle cerebral, about a quarter of an inch from the junction with the anterior cerebral artery, there was a small area studded with minute granular protuberances. They were five or six in number, of uniform size, about as large as a millet seed. They were subepithelial, and of the same microscopical appearance as the basement membrane (hyperplastic). The epithelial membrane was everywhere intact over these points, and presented no difference in color or texture from the rest of the membrane. The clot was a little firmer at this point, and slightly adherent to the granular surface. At all other points the coagulum rolled easily from the arteries after incision.

On section, nearly all those portions of the brain supplied with blood by these occluded vessels were found in an advanced state of yellow softening. The corpus striatum was completely broken down, forming a mere granular pulp. The softening in other portions, though less advanced, extended outwards to the temporal and second frontal convolutions, involving also the island of Reil. All other portions of the brain were normal.

Comments. Looking at this case without the light of the subsequent autopsy, what would be the most plausible explanation of the phenomena presented? Evidently, one of three things has happened — cerebral hæmorrhage, embolism, or thrombosis. Do the symptoms recorded indicate, with any degree of certainty, which of these three lesions has taken place? Frankly, nothing in my own experience, either in private or hospital practice, enabled me to feel certain as to the diagnosis. I find nothing in the age, sex, suddenness of attack, pallor, vomiting, temperature, pulse, unconsciousness, impairment of sensation, extent and location of the paralysis, nor contractions which might not obtain in either of the three lesions mentioned. Nor do I find in the differential diagnosis of authors any great degree of certainty. It is the absence of certain conditions, rather than the symptoms presented in this case, which determined my diagnosis. The chief factor in causation not being perceived, the reasoning led to error. Here was a young man, in apparent health, of excellent habits, and of fine physique, who had never been ill since early childhood, with no perceptible disease of the heart or blood-vessels, with no personal nor family history of rheumatism, scrofula, syphilis, tuberculosis, or carcinoma, with no impoverishment or contamination of the blood. In the absence of these conditions we should hardly expect either embolism or thrombosis. The diagnosis made was cerebral hæmorrhage in the region of the left corpus striatum.

The anomalous condition of the heart was not recognized during life. A soft, indistinct, hæmic murmur at the base, attending the systole only, was heard at in-

tervals, but this was not attributed to structural disease. I never examined the patient in health, and I do not know what auscultation would have revealed then. The effect of this state of the heart would be to increase both arterial and venous tension, and to diminish the velocity of the blood current, — two conditions favorable and adequate to the production of thrombosis. My first impression, on viewing the site of the lesion, was that a thrombus had originally formed in the trunk of the middle cerebral artery at the point of the granular protuberances; that shreds had washed from it into the connecting branches, producing emboli, and that the intervening clots were the result of infarction. I think it may be doubted, however, if this theory represents the true order of events. The attack was so sudden and unheralded, and the injury so profound, it seems probable that the entire clot was formed within a period of a few seconds. The clot, moreover, was of uniform color and texture throughout. If the clot was slightly adherent to the granular surface, it was probably due to the mere mechanical advantages of that locality, and not necessarily to an earlier formation. This appears to be a case of autochthonous thrombosis. It is true that the suddenness of the attack is remarkable and, I should say, somewhat exceptional. But there may have been premonitory signs which the reticent patient carefully kept from his friends. The numbness of the fingers referred to in the report, may have been one of them.

The stasis of blood was not due to an enfeebled heart, which, on the contrary, was remarkably vigorous, but to the antagonism of the venous and arterial currents. The point of greatest tension and of least velocity would be somewhere on the arterial side of the capillaries. If there was any element of causation other than the state of circulation, it was probably the depressing effect of the very hot weather at the time of the attack.

It is difficult to account for the granular protuberances, so limited in number and distribution. They were evidently not recent in origin. They were identical in their character and in relation to the arterial structures with those seen in cases of undoubted inflammation of arteries. But why here? I do not regard them as factors of causation of the thrombosis.

The extent, the rapidity, and the character of the softening are of great interest. There was no distinct line of demarcation between the healthy and the diseased brain substance. Nor should we expect any clearly marked area of diseased tissue. The softening would naturally begin first, and be the most complete and rapid in those portions supplied by terminal vessels. As there is no anastomosis here with other vessels, death of the parts is sudden and complete, and the decay partakes of all the characteristics of non-inflammatory softening. The entire mass of the corpus striatum and nearest adjacent parts were of this nature. Their pulpy, granular texture illustrated very perfectly that condition so familiar to pathologists. All this disorganization took place within the brief space of sixty-five hours. The cortical portions of the affected region were in a state much less advanced towards softening. Here collateral circulation very materially delayed decay.

The yellow softening was what would be expected under the circumstances, if the generally received opinion as to the cause of coloring be correct; namely, the staining by disintegrated blood corpuscles en-

trapped in the parts. The occlusion was so extensive, there was no possibility of regurgitation, or setting back of capillary circulation. The arterioles were found collapsed, and hence a large area of brain tissue was anæmic.

The only evidence of inflammatory action in all this wide area of disease was found in a small surface of the pia mater over the left second frontal convolution. The red, softened, and corrugated state of the lining membrane of the occluded vessels, no doubt, took place after the accident.

Aphasia was complete from first to last. Many interesting queries as to the mental state of this patient suggest themselves; but any adequate consideration of them would prolong this report to too great a length.

REPORT ON THE PROGRESS IN MATERIA MEDICA AND PHARMACY.¹

BY W. P. BOLLES, M. D.

THERE has been no important discovery in this line within the past six months, although some interesting investigations are in progress and will perhaps be ready for summarizing in a short time. Several of the newer drugs are being carefully analyzed by eminent workers, but evidence of their medicinal value is more wanting than the details of their composition.

JABORANDI.

In addition to pilocarpine, Messrs. Flarnack and Meyer have obtained a second alkaloid from this drug, which they propose to call jaborina: this alkaloid differs from pilocarpine in its sparing solubility in water and readier solubility in ether. It was separated from commercial pilocarpine, as a colorless amorphous body of strong basic properties, forming amorphous salts. Its action is said to resemble that of atropia upon a frog's heart; no other test appears to have been tried.

QUILLAIA IN EMULSIONS.

Nothing delights an apothecary like the prospect of making an "elegant" emulsion, so we have had emulsions with tragacanth, and other gums, with almonds, egg and pancreatine, in innumerable variations. Now quillaia in tincture appears as an emulsifying agent (it has, we believe, for some years existed in soda water syrups to make them foam). It will "extinguish" mercury, and holds oils, chloroform, copaiba, and resins in fair suspension. From one fourth to one eighth appear to be suitable proportions of the tincture for oils etc. Turpentine and copaiba require an equal quantity of the tincture, and resins still more; of course all with the addition of water.

CHIAN TURPENTINE.

Perhaps this substance received all the attention it deserved in the last report; but as Dr. Clay still maintains that he is justified in his claims for it, and as there is a constant, though moderate, demand for it, coupled with great difficulty in detecting the genuine from the numerous imitations, a word or two more may be accepted.

There are, it seems, only about a thousand old trees

¹ The Pharmaceutical Journal and Transactions. The American Journal of Pharmacy. The Pharmacist. New Remedies. Pharmaceutisches Centralblatt, etc.

in Cyprus, many of them very old and large. For many years little or none of the resin had been collected until last year, when the natives were surprised by the orders from England, and began to gather it again. The quantity from Cyprus can never be large, and collectors are already looking around in Asia Minor and Algeria for larger forests, so far without success being assured.

The description in the last report, although correct for a clean and pure product, does not correspond with the appearance of much of that at present in the market, and believed to be genuine.

Three specimens in the possession of the writer, with a satisfactory history of having come from Cyprus, are briefly as follows:—

(1.) Is a brownish-yellow, not quite transparent, honey-like, very sticky, liquid containing scattered grains of sand and bits of bark. Imported in cans.

(2.) Similar to number one, but darker, thicker, and dirtier.

(3.) Solid, brittle when cold, of a greenish or grayish-brown color, full of impurities (twenty-five per cent.). Imported in lined boxes. After softening with ether and straining the resin is deep yellow.

The odor of all the specimens is characteristic and similar, and the taste free from persistent bitterness.

Two specimens, believed to be factitious, have a coniferous odor, are clean and transparent, without bark, sand, or other obvious impurities, and leave a persistent, disagreeable resinous taste in the mouth. They were probably made from Canada turpentine. The present method of collecting the genuine article is such that a certain amount of bark and dirt is necessarily included, and a perfectly clean product, unless it comes with a history of being strained by reliable hands, should be avoided; but if the demand continues it is to be hoped that a more rational mode of gathering it will yield a more respectable product.

ACONITIA.

Mr. C. R. A. Wright, who has carefully investigated the constituents of the aconites, has given in detail the chemical distinctions between aconitia, pseudaconitia, and "Japaconitia," the two latter the alkaloids of Indian and Japanese species respectively. They are of too nice character for ready pharmaceutical tests, and will fail, therefore, directly to clear up the confusion in which these products have for some time been.

Crystalline aconitia has been for a number of years prepared according to the process of M. Duquesnel, but the alkaloids of the other two species can equally be prepared in crystals.

Dr. Wright considers aconitia to be the chief, if not the only, active ingredient of the roots of *Aconitum napellus*; that it occurs therein with other amorphous alkaloids of lower molecular weight, and containing a higher percentage of carbon. "If the amount of aconitia present relatively to the amorphous bases is not considerable it is often impossible to get it to crystallize at all; in any case a considerable amount of aconitia is retained in solution permanently by the agency of the amorphous alkaloids which thus cause considerable loss." Therefore crystalline aconitia can so far be prepared only at the expense of waste, and remains very costly. Amorphous aconitia, on the other hand, may be good in quality as a small portion of the uncrystallizable bases will suffice to prevent its crystallizing, but there seems to be no good way of determining it unless, pos-