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SUDDEN DEATH FROM EMBOLISM.¹

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DURING the past two years I have had the opportunity of observing the anatomical results of immediately fatal embolism in several instances, and under such circumstances as seem to make it desirable to call renewed attention to this cause of death, already so well recognized.

The histories of these cases show that a marked feature is the sudden and unexpected nature of the attack, whether taking place during the progress of disease or in persons apparently in good health. Combined with this is the evident impossibility of furnishing any effectual relief from the distressing symptoms, except by the administration of anæsthetics, which in most cases represent merely a change in the agent eventually employed, as the evidence of carbonic-acid poisoning very rapidly becomes manifest.

That the employment of such agents may be recommended it is desirable that a relatively correct diagnosis should be made, and although there are no symptoms pointing absolutely to embolism, there are certain suggestive ones. The comparative rarity of the occurrence in the experience of any one physician is practically likely to cause considerable doubt when the occasion finally arises, and a consideration of these symptoms becomes, therefore, all the more important.

To the kindness of the several gentlemen in whose practice the following cases occurred I am indebted for the clinical histories which give to this paper its chief value.

CASE I. The first case was under the charge of Dr. J. T. G. Nichols, of Cambridge, who reports as follows: —

“A lady, thirty-three years of age, of unusual strength and endurance, had always been well with the exception of having suffered from an attack of Panama fever twelve years ago, and from an acute laryngitis eight years previous to her last illness. She had been troubled of late by bleeding piles, which had annoyed her from time to time for several years. A mild attack of typhoid fever began October 5, 1874, though she did not take to her bed till eleven days later, when I was first sent for. She had a slight cough, which became more severe three days

¹ Read before the Boston Society for Medical Observation, December 18, 1876.

after my attendance began. It was paroxysmal in character, and she described her sensation as though the windpipe were constricted and she must die unless speedily relieved. During these paroxysms her face grew red, and there was severe headache. She became quite nervous in consequence. There was no alteration of the voice, nor was there any expectoration or dyspnoea. Opiates were given, and the cough disappeared on the 20th of October. During this interval there was a slight irregularity of the heart's action, of which the patient was conscious, and she could not lie easily upon the left side. Physical examination of the heart and lungs, made daily, revealed nothing abnormal. When the cough and irregular action of the heart disappeared, a sense of weakness and sinking was complained of, but there was no change in color, pulse, or temperature corresponding with this sensation, which was relieved by the moderate administration of stimulants. Five days after the first paroxysm of coughing there was a return of this symptom in a less severe form, and it was again relieved within twenty-four hours by opium. Two days later she was seen in the morning, in good spirits, and expressed her confidence as to a speedy recovery. Her pulse was 90, and the temperature 100°. She was breathing naturally, and there was nothing in her appearance to give rise to any fear of impending danger. At noon she sat up in bed to take medicine; fifteen minutes later, while being read to by her sister, she made some remark in a natural tone of voice, and immediately began to struggle. The blood rushed to her face, and the respiration became difficult. She referred her suffering to the chest and to the right iliac region. Profuse sweating came on, the skin rapidly became livid and cold, and in less than an hour she died. Just before her death she was seen by Dr. Morrill Wyman; there was then no pulse to be felt, the respiration was very imperfect, and death took place during a slight convulsion."

The autopsy was made twenty-four hours later. The right side of the heart was distended with fluid blood. A dark-red, firm, elastic thrombus was found in the main pulmonary artery, extending nearly to the valves. It was doubled upon itself, the opposed surfaces being moderately agglutinated, and formed a mass nearly as large round as the thumb. The angle thus formed pointed towards the heart, and this portion of the thrombus presented a conical shape. It extended into the main pulmonary arteries of both lungs as far as the tertiary divisions. The oldest portion was in one of the tertiary divisions of the right pulmonary artery, the age being indicated by a firmer adherence to the wall, a softer consistence, and a reddish-gray color. There was no evidence of local disease of the pulmonary artery, and the surrounding tissue was not essentially altered.

The lower lobes of both lungs were extensively injected, and the right upper lobe was œdematous. There was no hæmorrhagic infarction.

The kidneys and liver were injected; the cystic duct was obstructed by a gall-stone, and there was cystic dropsy of the gall-bladder in consequence. The spleen was acutely enlarged, and Peyer's patches, near the ileo-cæcal valve, were moderately swollen, opaque, œdematous, and of a bluish slate color. There were three small typhoidal ulcers in the cæcum in the process of healing. A source for the embolism of the pulmonary artery was not ascertained, circumstances preventing a prolonged search.

The cause of death in this case seems to have been a primary embolism of a small branch of the pulmonary artery, and its extension towards the heart by secondary coagulation, until it actually protruded into the main pulmonary artery. The protruded part was then bent backwards, doubled upon itself, and was continued into the pulmonary artery of the other lung. It has long been evident that in typhoid fever there are certain predisposing elements to thrombus-formation. The enfeebled action of the heart, the confinement in bed, and the muscular rest act as favoring causes in producing the marantic thrombus, usually in the veins of the extremities or in the vesical plexus. The existence of hæmorrhoids naturally calls attention to the latter region as affording an additional factor in the production of thrombi, but none were found in these veins.

As the febrile attack was a mild one, during the first eleven days not necessitating confinement in bed, there seemed to be nothing in the general condition of the patient to call attention to the possible presence of a thrombus. In the light of the result, however, there are certain symptoms which seem of considerable importance, and Dr. Nichols has called very direct attention to them. These are the paroxysmal cough, the sensation of constriction and feeling of anxiety, without alteration of voice or dyspnœa, associated with temporary irregular action of the heart. Some days after the disappearance of this attack another similar one, though less severe, occurred, which was more speedily relieved. The absence of physical signs in the chest, and of special symptoms calling attention to the larynx, is of importance in withdrawing attention from the hypostatic pneumonia and laryngeal ulcerations which form so frequent a complication of typhoid fever.

The anatomical evidence of an antecedent embolus was quite sufficient, and if these paroxysmal attacks can be regarded as resulting from the transfer of emboli, it becomes evident that an original autochthonal thrombus must have been formed before the close of the second week of the fever, at a time when the patient had been in bed but three days.

CASE II. The following case was reported by Dr. E. Chenery at a meeting of the Suffolk District Medical Society.¹

¹ Vide JOURNAL, 1876, xciv. 396.

A lady, fifty-two years of age, was first seen by Dr. Chenery on August 26, 1875. She was in robust health till a short time before, when she had occasional attacks of diarrhoea. She had become weak and anæmic, complained of pain in the back and pelvis, and was constipated, though troubled with frequent mucous discharges from the rectum. During the following month she became steadily worse. On September 27th she was suddenly seized with dyspnoea, and became cold and clammy; the pulse was rapid and weak. In the course of two hours she rallied, but soon relapsed and died.

The autopsy showed that the immediate cause of death was a thrombus of the primary pulmonary artery extending into the secondary divisions; the thrombus was firm, gray, and laminated. Its source was not discovered. There was also cancer of the right ovary, filling the pelvis.

This patient, though previously robust, had become rapidly cachectic, apparently in consequence of the progressive cancerous affection and the frequent intestinal discharges. There seem to have been no symptoms from which an existing thrombus could be suspected, and no evidence of embolism preceding the fatal attack.

CASE III. Dr. R. L. Hodgdon, of Arlington, presented to the Obstetrical Society of Boston the account of the following case, which occurred in his practice.¹

The patient, twenty-five years of age, had already borne one child. The labor was a tedious one, and it was necessary to apply forceps. Before and after this event her health had been good. At her second confinement, January 22, 1875, a healthy female child was delivered by turning, there being but little loss of blood. During the subsequent twenty-four hours the after-pains were severe. A few hours after delivery she had a chill and pain in the left iliac region. Pain and tenderness in this region, accompanied by general febrile excitement, persisted for several days, when the symptoms subsided, and an apparently favorable convalescence followed. There was no œdema of the legs observed, nor was there any cough. On the twenty-fourth day after delivery the patient went to the bed from her chair, remarking, "I never felt better in my life." She lay down, and at once began to breathe with great difficulty. Dr. Hodgdon saw her ten minutes later; she was tossing about on the bed, suffering intensely, and complaining of pain in the epigastric region. The face was livid, the tongue blue, and the respiration labored, 80 per minute. The pulse was rapid, small, and thread-like, and but one sound of the heart was heard. The chest was resonant, and air could be heard entering it. Her condition remained the same during the following ten or fifteen minutes, when death took place.

The body was well nourished, but the organs were generally anæmic.

¹ Vide JOURNAL, 1875, xciii. 73.

A soft, reddish-gray thrombus, of the size of the forefinger, extended from the right iliac vein into the inferior vena cava for two inches. Thrombi were also found in the right ovarian vein and in the vesical plexus. The uterus was in a state of normal retrogression. The tricuspid orifice contained a club-shaped embolus one and a half inches in length, the larger end being the size of the tip of the forefinger. Both primary pulmonary arteries were plugged with emboli; also their branches which passed to the upper lobes of both lungs. These lobes were œdematous, while the lower ones were injected.

Although in this case there was clinical evidence of a pathological process in the pelvis, yet there were no special symptoms to call attention to its exact nature, nor was any evidence found after death to explain such symptoms. Certainly, during the last two weeks of the patient's life the case differed in no respect from perfectly normal cases of convalescence after labor. Whatever may have been the cause of these symptoms, a thrombosis could least be suspected, though it may have been a result. From the well-known fact that extensive thrombosis may take place without symptoms, and from the absence of the most common and prominent symptom of puerperal thrombosis, milk-leg, there seems to have been but little to suggest even the idea of embolism.

CASE IV. A man fifty years of age entered the Massachusetts General Hospital January 5, 1876, to be treated by Dr. Cabot for an ulcer of the leg. Many years previous he had been injured by a boat-hook, but the wound had never healed; at one time it even extended from just below the knee to the ankle, though at the time of his entrance it was about the breadth of two fingers only. For the two weeks preceding his admission he had been annoyed by considerable dyspnœa and by a slight cough, though there had been but little expectoration. He had also complained for some time of a fluttering sensation in the region of the heart. He was very weak; the face was pale and the lips blue, and he had an attack of faintness while being carried to the ward. The pulse was fair, though rather quick; the heart's action tumultuous, and the impulse felt over an abnormally large area, but no souffle could be detected. Examination of the lungs disclosed nothing but a few moist râles at the base. The urine had a specific gravity of 1013, and contained one half per cent. of albumen. Granular and hyaline casts, blood, pus, and oxalate of lime crystals were seen with the microscope.

At the morning visit, two days after his admission, he said he was feeling better, but about 4.30 P. M. he suddenly became oppressed in breathing, with paroxysms of unconsciousness; the face became livid, the pulse failed rapidly, and in about a quarter of an hour he died suddenly in one of the attacks of unconsciousness.

At the autopsy, seventeen hours after death, the left foot was found to be œdematous. The brain was examined, but nothing abnormal was found beyond an increased density of the posterior lobes. In each pleural cavity was about a pint of clear yellow fluid, and there were about four ounces in the pericardium. The heart was considerably enlarged from dilatation and hypertrophy. The aortic orifice permitted slight regurgitation, the valves being thickened and slightly retracted. The mitral orifice was enlarged, and the line of apposition of the valves was slightly roughened. The cavities of the heart contained post-mortem clots, and the right side was dilated, the left contracted.

The right and left pulmonary arteries were filled completely with firm red thrombi extending into the tertiary branches. On the left side they were moderately adherent to the walls, while on the right they were connected with an adherent, partially decolorized, and somewhat softened embolus. At the periphery of the right upper lobe were two wedge-shaped nodules of infarction of a reddish-gray color, about an inch and a half broad at the base.

Six ounces of fluid were found in the abdominal cavity. Both liver and kidneys presented the appearances of chronic passive congestion, nutmeg atrophy in the former, cyanotic induration in the latter; degenerative changes had also taken place in the renal epithelium.

The left femoral vein contained a recent thrombus extending from below the middle of the thigh into the iliac vein. From Poupart's ligament downwards it almost completely filled the vein, while above this point it represented a continued partial thrombus.

The various conditions favoring the production of a marantic thrombus were present here to a marked degree, as well as a local predisposing cause for the origin of the thrombus. The dyspnoea and cough which existed previous to the patient's entrance into the hospital are of but little value in relation to embolism, on account of the evident disturbance to the pulmonary circulation from the chronic valvular disease of the heart.

Although the attack of faintness may have been connected with the transfer of an embolus, it is evident anatomically that several transfers had taken place, from the nodules of infarction in one lung and the adherent embolus in the other.

However numerous may have been the emboli which preceded the fatal attack, a distinct interval of several days existed between the occurrence of any symptoms pointing to serious disturbance of the circulation and those immediately preceding death.

CASE V. On or about the 1st of May, 1876, I received from Dr. I. G. Porter, of New London, Ct., the thoracic organs of a patient who had died suddenly. From the published account of the case ¹ it

¹ American Journal of the Medical Sciences, 1876, cxliv. page 436.

appears that the patient, sixty-one years of age, was of robust and plethoric appearance, and of extremely active habits. His pulse was always small and weak. For two weeks previous to his death he complained of unusual weakness, and had a slight bronchial cough. He was able to attend to his business, however, and drove out the day before his death, managing the horses himself. After a restless night he arose, took a bath as usual, and went to the observatory at the top of the house as was his custom. "But he returned almost immediately, became faint and breathless, and barely reached the lower floor, when he almost fell into the arms of his family. I reached him very soon (at 7.45 A. M.), and found him cold and in profuse perspiration; countenance pale and ghastly; pulse rapid, irregular, and scarcely perceptible. He was very restless, though without true pain, and complained earnestly of being faint and of having no breath, although at the time filling his lungs completely at every inspiration. The difficulty was not increased by the horizontal position, and yet he preferred to sit up, supported by friends. Stimulants conferred momentary relief, and sinapisms and external warmth were freely used, but in about half an hour his head fell on his breast, and he was gone, remaining conscious to the last."

The organs arrived in a fresh condition; the heart had been opened; but had contained fluid blood, according to the accompanying letter. There was moderate dilatation with hypertrophy due to chronic changes at the aortic orifice, also thickening and contraction of the valves, which produced insufficiency. One of the aortic valves was almost wholly obliterated. The heart was not in a state of fatty degeneration.

In one of the secondary branches of the pulmonary artery of the left lung, an adherent and slightly decolorized thrombus was found, which had probably been in position several days. A more recent but still ante-mortem coagulation extended from this towards the main pulmonary artery of both lungs, and was continued into the right pulmonary artery, filling its primary branches. An arborescent thrombus was thus formed, with its ends rounded and pointed and passing from an inch to an inch and a half into the primary branches. Beyond its ends in the lung the blood was still fluid. No source of embolism was found in the heart.

In the report of the case referred to, Dr. Porter states that "some eighteen months previous to his death the patient made an overland journey to California, and, as he thought, through long and persistent confinement in the cars, his left leg and foot became very painful and swollen; so much so that for some time after his arrival he was disabled for business, and was under the care of a surgeon, and by him kept very quiet in a horizontal position. The pain left him after a while, but the limb remained swollen quite to the time of his death,

though it did not particularly incommode him in walking." About a fortnight before the patient's death he strained violently at the health-lift, after which he complained of feeling ill. Dr. Porter suggested that at this time, through powerful muscular action, a fragment may have been detached from a possible thrombus then existing.

CASE VI. A man fifty-five years of age, of marked cachectic appearance, entered the Massachusetts General Hospital to be treated by Dr. J. C. Warren for caries of the wrist, of two years' standing. During this period he had been unable to work on account of the local trouble. When he arrived at the hospital the wrist was much swollen, and a mild attack of erysipelas came on, which had almost subsided at the time of the fatal attack. Early in the morning of October 3, 1876, eight days after his entrance, a patient in an adjoining bed called the nurse's attention to him on account of apparent difficulty in breathing and the expression of his face. He had become unconscious almost instantly. Fifteen minutes later he was pulseless and breathless. There was no lividity, nor had any spasm or convulsion taken place.⁶ He had been in bed during his stay in the hospital, and the night before had expressed himself as feeling very much better than when he entered.

At the autopsy, made thirty-two hours after death, the heart was found distended and contained mainly fluid blood. A thrombus nearly two inches in length and as large round as the little finger extended from the right ventricle into the pulmonary artery. It was dark red and moderately firm, evidently having formed some hours before death. Several thrombi which had apparently been contained in the small pulmonary vessels were removed from the thorax. The entire lower lobe of the left lung was in a state of atelectasis, of relatively recent origin, and the right lung was œdematous. The kidneys and liver were unusually injected.

A source of embolism was looked for but was not found.

Whether the decided swelling of the wrist, evident at the patient's entrance into the hospital, resulted from a thrombosis or not, was not ascertained. A minute dissection of the veins of the arm was not made, although those were opened which were large enough to contain an embolus of the size mentioned.

A marked peculiarity in the symptoms of this case is furnished by the absence of lividity and spasms. The record of the symptoms immediately preceding death is a very brief one, the negative facts having been obtained several days after the death of the patient. The appearance of the organs suggested that death was the result of suffocation, but the recorded symptoms do not point in the same direction.

Although the element of enfeeblement is common to all of these cases, the degree is so varied that its importance as a general feature becomes comparatively limited. In the puerperal and typhoid cases it

was by no means sufficient to cause any anxiety ; both were regarded as convalescing favorably, and the condition of the organs indicated that such was the case. The mere fact that under these circumstances an accident of so grave a character may take place is sufficiently suggestive of a guarded prognosis even when a rapid recovery is anticipated. In Dr. Porter's case the theory of the cause and effect is certainly a very plausible one. Although the patient was far from being looked upon as enfeebled, yet the occurrence of a probable thrombo-phlebitis, in connection with confinement, indicates that a relatively trivial factor was alone required to bring about a diseased condition. After such an attack his health could hardly be regarded as fully up to the average. Of great energy and engaged in active commercial pursuits, his tendency was to add to rather than to withdraw from the routine of his daily life.

The symptoms of cardiac disturbance were insufficient to attract special attention to the heart ; nevertheless, some time after his return from California, he made a summer trip to Europe on account of overwork.

In the three other cases the cachectic condition was strikingly apparent, of extremely rapid origin in the cancerous patient, and of more protracted character in the cases of chronic ulcer and caries.

It may be considered that in all the cases, except perhaps in Dr. Porter's, there were no symptoms by which an existing thrombus could be directly diagnosticated. There are certain points, however, in the histories of some of them which seem to be of value in calling attention indirectly to such a condition. These are such as may be attributed to antecedent embolism.

In Dr. Nichols's case embolism of a tertiary branch of the right pulmonary artery, without resulting infarction, had occurred. In Dr. Cabot's case there was an embolism of a tertiary branch of the right pulmonary artery, also without infarction, in addition to the two nodules of hæmorrhagic infarction seated near the periphery of the right upper lobe. In Dr. Porter's case an embolus of a secondary branch of the left pulmonary artery had previously taken place. In three of the cases, therefore, there is anatomical evidence of antecedent embolism, while in the other three cases the only embolism was that immediately proving fatal.

It is to be noticed at the outset that the anatomical results of the embolism in these three cases were not alike. In one case only were nodules of infarction, embolic pneumonia, present, while in the other two cases no such condition was apparent. This discrepancy in the results of pulmonary embolism is one not unfrequently observed, and has recently been made the subject of experimental study by Cohnheim and Litten.¹ According to these observers an abnormal sluggish-

¹ Virchow's Archiv, 1875, lxxv. 99.

ness of the capillary current or an increased resistance to the escape of blood from the pulmonary veins must be present that infarction may occur. The former takes place in connection with multiple embolism of the lungs and from diminished action of the right side of the heart, as in protracted fevers and fatty degeneration. The latter is produced by valvular disease in the left side of the heart. Although the valvular disturbance found in Dr. Cabot's patient was incompetency rather than obstruction, yet the evidences of chronic passive congestion found in the liver and kidneys indicated obstruction to the passage of blood through the lungs, which obstruction had no evident source in these organs.

The question of practical importance is, were there any symptoms in these cases from which embolism could be inferred? Jürgensen¹ states that the obstruction of a small branch of the pulmonary artery usually gives rise to no symptom and that it is undetermined how large an infarction must be to give rise to symptoms. These are an increased frequency of respiration, even actual dyspnœa, little or no fever at the outset, although it may appear later, and a chill which is about as often absent as present. Cough arises, followed sooner or later by bloody expectoration in small quantities, and lasting several days; then there is pain when the infarction is seated at the periphery of the lung. The results of a physical examination of the lungs are dependent upon the size and seat of the embolus. He further adds: "Although it is very easy to describe these signs theoretically, it is very difficult to make them out practically." Gerhardt,² in addition, calls attention to sudden fainting attacks when a considerable portion of the pulmonary circulation is obstructed, and further to convulsive or spasmodic movements and disturbed cerebral function, all of which symptoms are attributed to anæmia of the nervous centres.

In neither of these three cases is bloody expectoration recorded, nor is there mention of a chill or of fever to be attributed to embolism. Dyspnœa is prominent though its character is not always fully described.

In two of the cases cough was observed, coming on suddenly in one case, in the other regarded as due to influenza although perhaps of equally sudden origin. In Dr. Cabot's case the sudden fainting attack may have been the symptom of embolism, though the dyspnœa and cough for two weeks previous are more likely to have resulted from the heart disease. In one of the cases a feeling of anxiety, in the other of uneasiness, is described as coming on suddenly. Temporary irregularity of the heart's action is noted in the case where the heart was healthy.

The special feature of importance seems to be the *sudden* occurrence

¹ Ziemssen's *Handbuch*, v. 251.

² *Sammlung klinischer Vorträge*, xci. 726.

of symptoms pointing to the lungs and brain. If these are associated with ascertained chronic disease of the heart, or with evidence of an existing thrombus, their value becomes considerable in exciting suspicion. This suspicion receives confirmation and becomes strengthened into probability as the other symptoms previously enumerated make themselves apparent.

The importance of ascertaining the existence of a pulmonary embolism is all the more apparent as the mechanical production of death in these cases is considered. Although in four cases the embolus may be regarded as sufficiently large to produce direct, rapid suffocation, in two cases this result was arrived at indirectly by the growth towards the heart of the thrombus which formed upon the antecedent embolus. The end projecting into the main pulmonary artery was bent backwards and forced into the unobstructed artery. Even in one of the four cases this combination of continued thrombosis, by cutting off the blood supply to one lung, was of great importance in necessitating sudden death when the pulmonary artery of the other lung received its obstructing embolus. A new element of danger thus becomes associated with the continued thrombus, that of giving rise to immediate and fatal obstruction without detachment, as well as through embolism.

The symptoms of the immediately fatal attack may be grouped together as follows: sudden extreme dyspnoea with open tubes, cough and thoracic pain, lividity or pallor, rapidly failing pulse, cold sweats, intense anxiety, and attacks of fainting or unconsciousness with or without spasms.

These symptoms may be referred to the lungs, heart, and nervous system, and are evidently due to the instantaneous elimination of more or less of the pulmonary circulation. The immediate effect of this is twofold, an accumulation of blood in the right side of the heart and nervous system in general, and a diminution of blood in the left side of the heart and arterial system, from the cutting off of the supply through the lungs. This arterial blood is not only diminished in quantity but in quality also, being poor in oxygen and containing more carbonic acid owing to the obstruction in the pulmonary vessels; and for the same cause the venous blood throughout the body contains an excess of carbonic acid. These changes in the composition of the blood are spoken of as suffocation, and it is of this condition that the patients so earnestly complain. Despite the respiratory efforts the blood becomes insufficiently aerated, and the fainting and spasms result from the causes producing the dyspnoea. The heart's action becomes enfeebled and irregular from dilatation of the right side and from diminished and insufficient blood supply to its walls, as well as from eventual paralysis of the pneumogastric nerves, and death is thus likely to result from asphyxia.

It is to be observed that in two of the cases death occurred with great rapidity, in fifteen minutes even. In one the pulmonary valves were apparently prevented from closing, and the face was not livid; in the other it seems probable that one pulmonary artery having been previously obliterated, the other became almost instantaneously closed. The immediate result must have been the same, non-oxygenation, and according to Pflüger¹ an insufficiency of oxygen kills much more rapidly than the immense accumulation of carbonic acid.

In the other cases where the symptoms of the fatal attack lasted from a half hour to two hours, sufficient opportunity seems to have been allowed for both factors to take effect, — the diminution of oxygen and the accumulation of carbonic acid.

In conclusion it may be stated that in general the symptoms of suffocation are considered as resulting from the prevention of the entrance of air into the lungs, from an obstruction to the pulmonary circulation, and from the removal of oxygen from the blood. In the differential diagnosis, in so far as it concerns the subject of this paper, it becomes therefore of importance to eliminate from the first two series the productive causes and to combine such elements as may add positively to the diagnosis.

The suddenness of the conditions being their chief interest here, all those forms requiring time for their production may be disregarded, and there remain —

(1.) Closure of the greater air passages or of a large number of small ones, from without or from within. (2.) Nervous lesions, particularly intracranial, affecting respiration and circulation. (3.) Obstruction to the pulmonary circulation from emboli, of blood and air particularly, fat being more gradual in its effects.

The first series may be eliminated by the physical and rational evidence of open air passages.

When an intracranial origin of suffocation exists, the predominant early symptoms are those of cerebral anæmia, namely, pallor, relaxed muscles, disturbed hearing and vision, contracted pupils, fainting, and convulsions. Although dyspnœa may at times precede these symptoms, it is not usually of so severe a character as in the other series.

In favor of the embolic source of the disturbance is the history of an antecedent thrombus or of a disease of the heart likely to be associated with thrombosis. The primary brief disturbance of cerebral function is rapidly, at times almost instantaneously, followed by extreme dyspnœa (Gerhardt²) while later the symptoms of cerebral anæmia again become prominent.

Briefly, if in suffocation the symptoms of cerebral anæmia predomi-

¹ Wagner's General Pathology, 563.

² Loc. cit.

nate, the source should be sought for in internal hæmorrhage or in intracranial disturbance; if the thoracic symptoms are most prominent, the air passages or the circulatory apparatus are to be questioned, and the former can most readily be excluded. The only positive evidence in favor of the latter is to be derived from the previous history of the patient.

A MODIFICATION OF THE OPHTHALMOSCOPE.¹

BY O. F. WADSWORTH, M. D.

DURING the last few years the direct method of ophthalmoscopic examination (upright image) has come more and more into favor. While it enables the inspection of the background of the eye under a much greater magnifying power than by the indirect method, and is free from the disturbing reflections from the surfaces of the auxiliary lens which attend the latter, it also permits the tolerably close determination of the refraction of the observed eye independently of the subjective impressions of the patient. For the patient the direct method has the important advantage that a far less amount of light need be thrown into his eye, and the often disagreeable dazzling caused by the indirect method is thus avoided.

To the generalization of this method the modification of Loring, which consisted in placing a rotating disk containing lenses of different focal power behind the mirror, has chiefly contributed. In his ophthalmoscope, as in nearly all the various modifications of it which have since appeared, the mirror stands in a plane parallel to that of the disk behind it. To this there is an objection. With such an arrangement, in order to throw the light from the lamp into the observed eye, the mirror, and also the correcting lens behind it, must be placed at an angle to the direction in which the observer looks and which the rays coming from the fundus of the patient's eye to enter his follow. The effect of this oblique position of the lens on the rays which traverse it is as if there were added to it a cylindrical lens, its axis corresponding to the axis on which the spherical lens is inclined. The lens is made practically astigmatic, and the image of the fundus seen through it is distorted as it would be if the observed eye were astigmatic. The degree of astigmatism increases with the increase of obliquity of the lens, and for a given degree of obliquity is proportionate to the strength of the lens. When the mirror is placed in suitable proximity to the observed eye, an inclination of 20° is as little as can well be given to it in order to reflect the light into the eye in such a way that the fundus may be seen. For the lower degrees of ametropia the amount of astigmatism caused

¹ Read before the Boston Society of Medical Sciences, December 26, 1876.