merits or demerits. So far as existing evidence seems to show, the successful cases have been examples of localized pus deposits, more in the nature of pelvic peritonitis, or, in a few cases, of general suppurative peritonitis. In the latter class the operation has been performed a considerable time after delivery, such as six weeks, but cases of this kind cannot with propriety be called true puerperal septicæmia. The few cases reported in which cœliotomy has been performed soon after the development of septic symptoms appear all to have ended fatally. This is exactly what one would have *à priori* expected. In acute septic infection, which is a general and not a local disease, there are, it is true, very often marked symptoms of peritoneal disease, such as tenderness, immense distention, and the like; but this is one only of many local phenomena. To open the abdomen in such cases would be rash in the extreme, and a most hopeless procedure; it might even be impossible to return the enormously inflated intestines. It has been said that cœliotomy to be of use in cases of this kind must be done early, but it is to be remembered that in the early stages of septicæmia the symptoms are not well marked. The hope of cutting them short has not been abandoned, and it would lead to deplorable results if advice of this kind should lead to opening the abdomen of puerperal patients as soon as suspicious symptoms arose. In the former class, however, it is certain that in well-selected cases cœliotomy, washing out of the abscess cavity or peritoneum, and drainage, offer by far the best prospects of recovery.

Such are the remedies most used in this disease. It is needless to say that it is quite impossible to lay down fixed rules for the management of any individual case; and it is obvious that, if puerperal septicæmia be not a special and distinct disease, its judicious treatment must depend on the general knowledge of the attendant and on a careful study of the symptoms each separate case presents.

CHAPTER VI.

PUERPERAL VENOUS THROMBOSIS AND EMBOLISM.

Puerperal Thrombosis and its Results.—Under the head of *thrombosis* we may class several important diseases connected with the puerperal state, which have received far less attention than they deserve. It is only of late years that some, we may probably safely say the majority, of those terribly sudden deaths which from time to time occur after delivery have been traced to their true cause, viz., obstruction of the right side of the heart and pulmonary arteries from a blood-clot, either carried from a distance or, as I shall hope to show, formed *in situ*. Although the result and, to a great extent, the symptoms, are identical in both, still a careful consideration of the history of these two classes of cases tends to show that in their production they are distinct, and that they ought not to be confounded. In the former we have primarily a clotting of blood in some part of the peripheral venous system, and the separation of a portion of such a thrombus due to changes undergone during retrograde metamorphosis tending to its eventual absorption. In the latter we have a local depositing of fibrin, the result of blood changes consequent on pregnancy and the puerperal state. The formation of such a coagulum in vessels the complete obstruction of which is incompatible with life, explains the fatal results. When, however, a coagulum chances to be formed in more distant parts of the circulation, the vital functions are not immediately interfered with, and we have other phenomena occurring, due to the obstruction. The disease known as phlegmasia dolens, I shall presently attempt to show, is one result of blood-clot forming in peripheral vessels. But from the evident and tangible symptoms it produces, it has long been considered an essential and special disease, and the general blood dyscrasia which produces it, as well as other allied states, has not been studied separately. I shall hope to show that all these various conditions, dissimilar as they at first sight appear, are very closely connected, and that they are in fact due to a common cause; and thus, I think, we shall arrive at a clearer and more correct idea of their true nature than if we looked upon them as distinct and separate affections, as has been commonly done. I am aware that in phlegmasia dolens, the pathology of which has received perhaps more study than that of almost any other puerperal affection, something beyond simple obstruction of the venous system of the affected limb is probably required to account for the peculiar tense and shining swelling which is so characteristic. Whether this be an obstruction of the lymphatics, as Dr. Tilbury Fox and others have maintained with much show of reason, or whether it is some as yet undiscovered state, further investigation is required to show. But it is beyond any doubt that the important and essential part of the disease is the presence of a thrombus in the vessels; and I think it will not be difficult to prove that in its causation and history it is precisely similar to the more serious cases in which the pulmonary arteries are involved.

It will be well to commence the study of the subject by a consideration of the conditions which, in the puerperal state, render the blood so peculiarly liable to coagulation, and we may then proceed to discuss the symptoms and results of the formation of coagula in various parts of the circulatory system.

Conditions which Favor Thrombosis.—The researches of Virchow, Benjamin Ball, Humphry, Richardson, and others have rendered us tolerably familiar with the conditions which favor the coagulation of the blood in the vessels. These are chiefly: 1. A stagnant or arrested circulation; as, for example, when the blood coagulates in the veins which draw blood from the gluteal region in old and bedridden people, or, as in some forms of pulmonary thrombosis, in which the clots in the arteries are probably the result of obstruction in the circu-

lation through the lung-capillaries, as in certain cases of emphysema, pneumonia, or pulmonary apoplexy. 2. A mechanical obstruction around which coagula form, as in certain morbid states of the vessels; or, a better example still, secondary coagula which form around a travelled embolus impacted in the pulmonary arteries. 3. And most important of all, in which the coagulation is the result of some morbid state of the blood itself. Examples of this last condition are frequently met with in the course of various diseases, such as rheumatism or fever, in which the quantity of fibrin is increased and the blood itself is loaded with morbid material. Thrombosis from this cause is of by no means infrequent occurrence after severe surgical operations, especially such as have been attended with much hemorrhage, or when the patient is in a weak and anæmic condition. This has been specially dwelt upon, as a not unfrequent source of death after operation, by Fayrer and other surgeons.[1]

Coagulation in the Puerperal State.—But little consideration is required to show why thrombosis plays so important a part in the puerperal state, for there most of the causes favoring its occurrence are present. Probably there is no other condition in which they exist in so marked a degree, or are so frequently combined. The blood contains an excess of fibrin, which largely increases in the latter months of utero-gestation, until, as has been pointed out by Andral and Gavarret, it not unfrequently contains a third more than the average amount present in the non-pregnant state. As soon as delivery is completed, other causes of blood-dyscrasia come into operation. Involution of the largely hypertrophied uterus commences, and the blood is charged with a quantity of effete material, which must be present in greater or less amount until that process is completed. It is an old observation that phlegmasia dolens is of very common occurrence in patients who have lost much blood during labor. Thus Dr. Leishman says: "In no class of cases has it been so frequently observed as in women whose strength has been reduced to a low ebb by hemorrhage either during or after labor, and this, no doubt, accounts for the observation made by Merriman, that it is relatively a common occurrence after placenta prævia."[2] An examination of the cases in which death results from pulmonary thrombosis shows the same facts, as in a large proportion of them severe post-partum hemorrhag has occurred. The exhaustion following the excessive losses so common after labor must of itself strongly predispose to thrombosis and, indeed, loss of blood has been distinctly pointed out by Richardson to be one of its most common antecedents. "There is," he observes, "a condition which has been long known to favor coagulation and fibrinous deposition. I mean loss of blood and syncope or exhaustion during impoverished states of the body."

Since, then, so many of the predisposing causes of thrombosis are present in the puerperal state, it is hardly a matter of astonishment that it should be of frequent occurrence or that it should lead to conditions of serious gravity. And yet the attention of the profession

[1] Edin. Med. Journ., March, 1861; Indian Annals of Med., July, 1887.
[2] Leishman: System of Obstetrics, p. 720. 2nd edition, 1876.

has been for the most part limited to a study of only one of the results of this tendency to blood-clotting after delivery, no doubt because of its comparative frequency and evident symptoms. True, the balance of professional opinion has lately held that phlegmasia dolens is chiefly the result of some morbid condition of the blood, producing plugging of the veins; but the wider view which I am attempting to maintain, which would bring this disease into close relation with the more rarely observed, but infinitely important, obstructions of the pulmonary arteries, has scarcely, if at all, been insisted on. Doubtless further investigation will show that it is not in these parts of the venous system alone that puerperal thrombosis occurs; but the symptoms and effects of venous obstruction elsewhere, important though they may be, are unknown.

Distinction between Thrombosis and Embolism.—I propose, then, to describe the symptoms and pathology of blood-clot in the right side of the heart and pulmonary artery. It may be useful here to repeat that this is essentially distinct from embolism of the same parts. The latter is obstruction due to the impaction of a separated portion of a thrombus formed elsewhere, and for its production it is essential that thrombosis should have preceded it. Embolism is, in fact, an accident of thrombosis, not a primary affection. The condition we are now discussing I hold to be primary, precisely similar in its causation to the venous obstruction which, in other situations, gives rise to phlegmasia dolens.

At the threshold of this inquiry we have to meet the objection started by several who have written on this subject,[1] that spontaneous coagulation of the blood in the right side of the heart and pulmonary arteries is a mechanical and physiological impossibility. This was the view of Virchow, who, with his followers, maintained that whenever death from pulmonary obstruction occurred, an embolus was of necessity the starting-point of the malady and the nucleus round which secondary deposition of fibrin took place. Virchow holds that the primary factor in thrombosis is a stagnant state of the blood, and that the impulse imparted to the blood by the right ventricle is of itself sufficient to prevent coagulation. It is to be observed that these objections are purely theoretical. Without denying that there is considerable force in the arguments adduced, I think that the clinical history of these cases strongly favors the view of spontaneous coagulation; and I would apply to the theoretical objections advanced the argument used by one of their strongest upholders with regard to another disputed point: "Je préfère laisser la parole aux faits, car devant eux la théorie s'incline."[2]

The anatomical arrangement of the pulmonary arteries shows how spontaneous coagulation may be favored in them; for, as Humphry has pointed out,[3] "the artery breaks up at once into a number of branches, which radiate from it, at different angles to the several parts of the lungs. Consequently a large extent of surface is presented to

[1] See especially Bertin: Des Embolies, p. 46 *et seq.*
[2] Bertin: Des Embolies, p. 149.
[3] Humphry: On the Coagulation of the Blood in the Venous System during Life.

the blood, and there are numerous angular projections into the currents, both which conditions are calculated to induce the spontaneous coagulation of the fibrin." We know also that thrombosis generally occurs in patients of feeble constitution, often debilitated by hemorrhage, in whom the action of the heart is much weakened. These facts of themselves go far to meet the objections of those who deny the possibility of spontaneous coagulation at the roots of the pulmonary arteries.

Results of Post-mortem Examinations.—The records of post-mortem examinations show also that in many of the cases the right side of the heart, as well as the larger branches of the pulmonary arteries, contained firm, leathery, decolorized, and laminated coagula, which could not have been recently formed. The advocates of the purely embolic theory maintain that these are secondary coagula, formed round an embolus. But surely the mechanical causes which are sufficient to prevent spontaneous deposition of fibrin would also suffice to prevent its gathering round an embolus; unless, indeed, the obstruction was sufficient to arrest the circulation altogether, when death would occur before there was any time for a secondary deposit. Before we can admit the possibility of embolism we must have at least one factor—that is, thrombosis—in a peripheral vessel, from which an embolus can come. In many of the recorded cases nothing of the kind was found, and although, as is argued, this may have been overlooked, yet such an oversight can hardly always have been made.

The strongest argument, however, in favor of the spontaneous origin of pulmonary thrombosis is one which I originally pointed out in a series of papers "On Thrombosis and Embolism of the Pulmonary Artery as a Cause of Death in the Puerperal State."[1] I there showed from a careful analysis of 25 cases of sudden death after delivery, in which accurate post-mortem examinations had been made, that cases of spontaneous thrombosis and embolism may be divided from each other by a clear line of demarcation, depending on the period after delivery at which the fatal result occurs. In 7 out of these cases there was distinct evidence of embolism, and in them death occurred at a remote period after delivery; in none before the nineteenth day. This contrasts remarkably with the cases in which the post-mortem examination afforded no evidence of embolism. These amounted to 15 out of the 25, and in all of them, with one exception, death occurred before the fourteenth day, often on the second or third. The reason of this seems to be that, in the former, time is required to admit of degenerative changes taking place in the deposited fibrin leading to separation of an embolus; while in the latter the thrombosis corresponds in time, and to a great extent no doubt also in cause, to the original peripheral thrombosis from which, in the former, the embolus was derived. Many cases I have since collected illustrate the same rule in a very curious and instructive way.

Another clinical fact I have observed points to the same conclusion. In one or two cases distinct signs of pulmonary obstruction have

[1] Lancet, 1867.

shown themselves without proving immediately fatal, and shortly *afterward* peripheral thrombosis, as evidenced by phlegmasia dolens of one extremity, has commenced. Here the peripheral thrombosis obviously followed the central, both being produced by identical causes, and the order of events necessary to uphold the purely embolic theory was reversed.

I hold, then, that those who deny the possibility of spontaneous coagulation in the heart and pulmonary arteries do so on insufficient grounds, and that we may consider it to be an occurrence, rare no doubt, but still sufficiently often met with, and certainly of sufficient importance, to merit very careful study.

History.—Dr. Charles D. Meigs, of Philadelphia, was one of the first to direct attention to spontaneous coagulation of the blood in the right side of the heart and pulmonary arteries as a cause of sudden death in the puerperal state. The occurrence itself, however, has been carefully studied by Paget, whose paper was published in 1855, four years before Meigs wrote on the subject.[1] It is true that none of Paget's cases happened after delivery, but he none the less clearly apprehended the nature of the obstruction. In 1855, Hecker[2] attributed the majority of these cases to embolism proper; and since that date most authors have taken the same view, believing that spontaneous coagulation only occurs in exceptional cases, such as those in which, on account of some obstruction in the lung or in the debility of the last few hours before death, coagula form in the smaller ramifications of the pulmonary arteries, and gradually creep back toward the heart.

Symptoms of Pulmonary Obstruction.—The symptoms can hardly be mistaken, and there seems to be no essential difference between the symptomatology of spontaneous and embolic obstructions, so that the same description will suffice for both. In a large proportion of cases the attack comes on with an appalling suddenness, which forms one of its most striking characteristics. Nothing in the condition of the patient need have given rise to the least suspicion of impending mischief, when all at once an intense and horrible dyspnœa comes on; she gasps and struggles for breath; tears off the coverings from her chest in a vain endeavor to get more air; and often dies in a few minutes, long before medical aid can be had, with all the symptoms of asphyxia. The muscles of the face and thorax are violently agitated in the attempt to oxygenate the blood, and an appearance closely resembling an epileptic convulsion may be presented. The face may be either pale or deeply cyanosed. Thus, in one case I have elsewhere recorded, which was an undoubted example of true embolism, Mr. Pedler, the resident accoucheur at King's College Hospital, who was present during the attack, writes of the patient:[3] "She was suffering from extreme dyspnœa, the countenance was excessively pale, her lips white, the face generally expressing deep anxiety." In another,

[1] Medico-Chirur. Trans., vol. xxvii, p. 162, and vol. xxviii, p. 352. Philadelphia Medical Examiner, 1849.
[2] Deutsche Klinik, 1855.
[3] Brit. Med. Journ., 1869, vol. i. p. 282.

which was probably an example of spontaneous thrombosis,[1] occurring on the twelfth day after delivery, it is stated: "The face had assumed a livid purple hue, which was so remarkable as to attract the attention both of the nurse and of her mother, who was with her." The extreme embarrassment of the circulation is shown by the tumultuous and irregular action of the heart in its endeavor to send the venous blood through the obstructed pulmonary arteries. Soon it gets exhausted, as shown by its feeble and fluttering beat. The pulse is thread-like and nearly imperceptible, the respirations short and hurried, but air may be heard entering the lungs freely. The intelligence during the struggle is unimpaired; and the dreadful consciousness of impending death adds not a little to the patient's sufferings and to the terror of the scene. Such is an imperfect account of the symptoms, gathered from a record of what has been observed in fatal cases. It will be readily understood why, in the presence of so sudden and awful an attack, symptoms have not been recorded with the accuracy of ordinary clinical observation.

Is Recovery Possible?—A question of great practical interest, which has been entirely overlooked by writers on the subject, is, Have we any ground for supposing that there is a possibility of recovery after symptoms of pulmonary obstruction have developed themselves? That such a result must be of extreme rarity is beyond question; but I have little doubt that in some few cases, entirely inexplicable on any other hypothesis, life is prolonged until the coagulum is absorbed and the pulmonary circulation restored. In order to admit of this it is, of course, essential that the obstruction be not sufficient to prevent the passage of a certain quantity of blood to the lungs to carry on the vital functions. The history of many cases tends to show that the obstructing clot was present for a considerable time before death, and that it was only when some sudden exertion was made, such as rising from bed or the like, calling for an increased supply of blood which could not pass through the occluded arteries, that the fatal symptoms manifested themselves. This was long ago pointed out by Paget,[2] who says: "The case proves that, in certain circumstances, a great part of the pulmonary circulation may be arrested in the course of a week (or a few days, more or less) without immediate danger to life, or any indication of what had happened." And after referring to some illustrative cases: "Yet in all these cases the characters of the clots by which the pulmonary arteries were obstructed showed plainly that they had been a week or more in the process of formation." If we admit the possibility of the continuance of life for a certain time, we must, I think, also admit the possibility, in a few rare cases, of eventual complete recovery. What is required is time for the absorption of the clot. In the peripheral venous system coagula are constantly removed by absorption. So strong, indeed, is the tendency to this, that Humphry observes with regard to it: "It appears that the blood is almost sure to revert to its natural channel in process of time."[3]

[1] Obst. Trans., 1871, vol. xii. p. 194.
[2] Op. cit., p. 358.
[3] Med.-Chir. Trans., vol. xxvii. p. 14.

If, then, the obstruction be only partial, if sufficient blood pass to keep the patient alive, and a sudden supply of oxygenated blood is not demanded by any exertion which the embarrassed circulation is unable to meet, it is not inconceivable that the patient may live until the obstruction is removed.

Illustrative Cases.—Such I believe to be the only explanation of certain cases, some of which, on any other hypothesis, it is impossible to understand. The symptoms are precisely those of pulmonary obstruction, and the description I have given above may be applied to them in every particular; and after repeated paroxysms, each of which seems to threaten immediate dissolution, an eventual recovery takes place. What, then, I am entitled to ask, can the condition be, if not that which I suggest? As the question I am considering has never, so far as I am aware, been treated of by any other writer, I may be permitted to state very briefly the facts of one or two of the cases on which I found my argument, some of which I have already published in detail elsewhere.

K. H., delicate young lady. Labor easy. First child. Profuse post-partum hemorrhage. Did well until the seventh day, during the whole of which she felt weak. Same day an alarming attack of dyspnœa came on. For several days she remained in a very critical condition, the slightest exertion bringing on the attacks. A slight blowing murmur heard for a few days at the base of the heart, then it disappeared. For two months patient remained in the same state. As long as she was in the recumbent position she felt pretty comfortable; but any attempt at sitting up in bed, or any unusual exertion, immediately brought on the embarrassed respiration. During all this time it was found necessary to administer stimulants profusely to ward off the attacks. Eventually the patient recovered completely.

Q. F., aged forty-four years. Mother of twelve children. Confined on July 6th. On the eleventh day she went to bed feeling well. There was no swelling or discomfort of any kind about the lower extremities at this time. About 3.30 A.M. she was sitting up in bed, when she was suddenly attacked with an indescribable sense of oppression in the chest, and fell back in a semi-unconscious state, gasping for breath. She remained in a very critical condition, with the same symptoms of embarrassed respiration, for three days, when they gradually passed away. Two days *after* the attack phlegmasia dolens came on, the leg swelled, and remained so for several months.

This case is an example of the fact I have already referred to, of phlegmasia dolens coming on *after* the symptoms of pulmonary obstruction had manifested themselves; the inference being that both depended on similar causes operating on two distinct parts of the circulatory system.[1]

C. H., aged twenty-four years. Confined of her first child on August 20, 1867. Thirty hours after delivery she complained of great weakness and dyspnœa. This was alleviated by the treatment employed, but on the ninth day, after making a sudden exertion, the dyspnœa returned with increased violence, and continued unabated until I saw the patient on September 4th, fourteen days after her confinement. The following are the notes of her condition, made at the time of her visit: "I found her sitting on the sofa, propped up with pillows, as she said she could not breathe in the recumbent position. The least excitement or talking brought on the most aggravated dyspnœa, which was so bad as to threaten almost instant death. Her sufferings during these paroxysms were terrible to witness. She panted and struggled for breath, and her chest heaved with short gasping respirations. She could not even bear anyone to stand in front of her, waving them away with her hand, and calling for more air. These attacks were very frequent, and were brought on by the most trivial causes. She talked in a low, suppressed voice, as if she could not spare breath for articulation. On auscultation air was found to enter the lungs freely in every direction, both in front and behind. Immediately over the site of the pulmonary arteries there was a distinct harsh, rasping murmur, confined to a very limited space, and not propagated either upward or downward. The heart-sounds were feeble and tumultuous." These symptoms led me to diagnose pulmonary obstruction, and I of course gave a most unfavorable prognosis, but to my great surprise the patient slowly recovered. I saw her again six weeks later, when her heart-sounds were regular and distinct and the murmur had completely disappeared.

[1] An interesting example of this occurrence has been kindly communicated to me by Dr. Neville, of Bristol. The patient, a primipara, aged twenty years, was suddenly seized with well-marked symptoms of pulmonary obstruction on January 24, 1892, three days *before* delivery. She was confined on the 27th, her condition from apnœa being then so critical that death was momentarily expected. Thirty hours *after* delivery symptoms of phlegmasia dolens, with painful swelling of both legs and thighs, occurred. After a protracted illness the patient gradually recovered. This case is of special interest, since the symptoms of pulmonary obstruction occurred before delivery. The only other instance of the same kind I know of has been recently recorded by Dr. Church (Trans. of the Obstet. Soc. of Edin., vol. xvii. p. 211), and that ended fatally.

E. E., aged forty-two years, was confined for the first time on November 5, 1873, in the sixth month of utero-gestation. She had severe post-partum hemorrhage, depending on partially adherent placenta, which was removed artificially. She did perfectly well until the fourteenth day after delivery, when she was suddenly attacked with intense dyspnœa, aggravated in paroxysms. Pulse pretty full, 130, but distinctly intermittent. Air entered lungs freely. The heart's action was fluttering and irregular, and at the juncture of the fourth and fifth ribs with the sternum there was a loud blowing systolic murmur. This was certainly non-existent before, as the heart had been carefully auscultated before administering chloroform during labor. For two days the patient remained in the same state, her death being almost momentarily expected. On the 21st—that is, two days *after* the appearance of the chest symptoms—phlegmasia dolens of a severe kind developed itself in the right thigh and leg. She continued in the same state for many days, lying more or less tranquilly, but having paroxysms of the most intense apnœa, varying from two to six or eight in the twenty-four hours. No one who saw her in one of these could have expected her to live through it. Shortly after the first appearance of the paroxysms it was observed that the cellular tissue of the neck and part of the face became swollen and œdematous, giving an appearance not unlike that of phlegmasia dolens. The attacks were always relieved by stimulants. These she incessantly called for, declaring that she felt that they kept her alive. During all this time the mind was clear and collected. The pulse varied from 110 to 130; respirations about 60; temperature 101° to 102.5°. By slow degrees the patient seemed to be rallying. The paroxysms diminished in number, and after December 1st she never had another, and the breathing became free and easy. The pulse fell to 80, and the cardiac murmur entirely disappeared. The patient remained, however, very weak and feeble, and the debility seemed to increase. Toward the second week in December she became delirious, and died, apparently exhausted, without any fresh chest symptoms, on the 19th of that month. No post-mortem examination was allowed.

I have narrated this case, although it terminated fatally, because I hold it to be one of the class I am considering. The death was certainly not due to the obstruction, all symptoms of which had disappeared, but apparently to exhaustion from the severity of the former illness. It illustrates, too, the simultaneous appearance of symptoms of pulmonary obstruction and peripheral thrombosis. The swelling of the neck was a curious symptom, which has not been recorded in any other cases, and may possibly be a further proof of the analogy between this condition and phlegmasia dolens.

Such Cases can only Depend on Pulmonary Obstruction.—Now it may, of course, be argued that these cases do not prove my thesis, inasmuch as I only assume the presence of a coagulum. But I may fairly ask in return, What other condition could possibly explain the symptoms? They are precisely those which are noticed in death from undoubted pulmonary obstruction. No one seeing one of them, or even reading an account of the symptoms, while ignorant of the result, could hesitate a single instant in the diagnosis. Surely, then, the inference is fair that they depended on the same cause. In the very nature of things my hypothesis cannot be verified by post-mortem examination; but there is at least one case on record in which, after similar symptoms, a clot was actually found. The case is related by Dr. Richardson.[1] It was that of a man who for weeks had symptoms precisely similar to those observed in the cases I have narrated. In one of his agonizing struggles for breath he died, and after death it was found "that a fibrinous band, having its hold in the ventricle, extended into the pulmonary artery." This observation proves to a certainty that life may continue for weeks after the depositing of a coagulum; and, moreover, this condition was precisely what we should anticipate, since, of course, the obstructing coagulum must necessarily be small, otherwise the vital functions would be immediately arrested.

Cardiac Murmurs in Pulmonary Obstruction.—There is a symptom noted in two of the above cases, and to a less extent in a

[1] Clinical Essays, p. 224 et seq.

third, which has not been mentioned in any account of fatal cases occurring after delivery, viz., a murmur over the site of the pulmonary arteries. It is a sign we should naturally expect, and very possibly it would be met with in fatal cases if attention were particularly directed to the point. In both these instances it was exceedingly well marked, and in both it entirely disappeared when the symptoms abated. The probability of such a murmur being audible in cases of thrombosis of the pulmonary artery has been recognized by one of our highest authorities in cardiac disease, who actually observed it in a non-puerperal case. In the last edition of his work on diseases of the heart, Dr. Walshe[1] says: "The only physical condition connected with the vessel itself would probably be systolic basic murmur following the course of the pulmonary main trunk and of its immediate divisions to the left and right of the sternum. This sign I most certainly heard in an old gentleman whose life was brought to a sudden close in the course of an acute affection by coagulation in the pulmonary artery, and to a moderate extent in the right ventricle.

Similar cases have, probably, been overlooked or misinterpreted. Many seem to have been attributed to shock, in the absence of a better explanation, a condition to which they bear no kind of resemblance.

Causes of Death.—The precise mode of death in pulmonary obstruction, whether dependent on thrombosis or embolism, has given rise to considerable difference of opinion. Virchow attributes it to syncope,[2] depending on stoppage of the cardiac contraction. Panum,[3] on the other hand, contests this view, maintaining that the heart continues to beat even after all signs of life have ceased. Certainly tumultuous and irregular pulsations of the heart are prominent symptoms in most of the recorded cases, and are not reconcilable with the idea of syncope. Panum's own theory is that death is the result of cerebral anæmia. Paget seems to think that the mode of death is altogether peculiar, in some respects resembling syncope, in others anæmia. Bertin, who has discussed the subject at great length, attributes the fatal result purely to asphyxia. The condition, indeed, is in all respects similar to that state, the oxygenation of the blood being prevented, not because air cannot get to the blood, but because blood cannot get to the air. The symptoms also seem best explained by this theory; the intense dyspnœa, the terrible struggle for air, the preservation of intelligence, the tumultuous action of the heart, are certainly not characteristic either of syncope or anæmia.

Post-mortem Appearances of Clots.—The anatomical character of the clots seems to vary considerably. Ball, by whom they have been most carefully described, believes that they generally commence in the smaller ramifications of the arteries, extending backward toward the heart, and filling the vessels more or less completely. Toward its cardiac extremity the coagulum terminates in a rounded head, in which respect it resembles those spontaneously formed in the peripheral veins. It is non-adherent to the coats of the vessels, and the blood circulates, when it can do so at all, between it and the vascular walls.

[1] Walshe: On Diseases of the Heart, 4th ed., 1873.
[2] Gesamm. Abhandl., 1862, p. 316. Virchow's Archiv, 1863

Such clots are white, dense, and of a homogeneous structure, consisting of layers of decolorized fibrin, firm at the periphery, where the fibrin has been most recently deposited, and softened in the centre where amylaceous or fatty degeneration has commenced. Ball maintains that if the coagulum have commenced in the larger branches of the arteries, it must have first begun in the ventricle and extended into them. According to Humphry the same changes take place in pulmonary as in peripheral thrombi, and they may become adherent to the walls of the vessels or converted into threads or bands. When the obstruction is due to embolism, provided the case is a well-marked one and the embolus of some size, the appearances presented are different. We have no longer a laminated and decolorized coagulum, with a rounded head, similar to a peripheral thrombus. The obstruction in this case generally takes place at the point of bifurcation of the artery, and we there meet with a grayish-white mass, contrasting remarkably with the more recently deposited fibrin before and behind it. It may be that the form of the embolus shows that it has recently been separated from a clot elsewhere; and in many cases it has been possible to fit the travelled portion to the extremity of the clot from which it has been broken. We may also, perhaps, find that the embolus has undergone an amount of retrograde metamorphosis corresponding with that of the peripheral thrombus from which we suppose it to have come, but differing from that of the more recently deposited fibrin around it. It must be admitted, however, that the anatomical peculiarities of the coagula will by no means always enable us to trace them to their true origin. In many cases emboli may escape detection from their smallness or from the quantity of fibrin surrounding them.

Treatment.—But few words need be said as to the treatment of pulmonary obstruction. In a large majority of cases the fatal result so rapidly follows the appearance of the symptoms that no time is given us even to make an attempt to alleviate the patient's sufferings. Should we meet with a case not immediately fatal, it seems that there are but two indications of treatment affording the slightest rational ground of hope.

1. To keep the patient alive by the administration of stimulants—brandy, ether, ammonia, and the like—to be repeated at intervals corresponding to the intensity of the paroxysms and the results produced. In the cases I have above narrated, in which recovery ensued, this took the place of all other medication. Possibly leeches, or dry cupping to the chest, might prove of some service in relieving the circulation.

2. To enjoin the most absolute and complete repose. The object of this is evident. The only chance for the patient seems to be that the vital functions should be carried on until the coagulum has been absorbed, or at least until it has been so much lessened in size as to admit of blood passing it to the lungs. The slightest movements may give rise to a fatal paroxysm of dyspnœa, from the increased supply of oxygenated blood required. It must not be forgotten that in a large proportion of cases death immediately followed some exertion in itself trivial, such as rising out of bed. Too much attention, then, cannot

be given to this point. The patient should be kept absolutely still; she should be fed with abundance of fluid food, such as milk, strong soups, and the like; and she should on no account be permitted to raise herself in bed, or attempt the slightest muscular exertion. If we are fortunate enough to meet with a case apparently tending to recovery, these precautions must be carried on long after the severity of the symptoms has lessened, for a moment's imprudence may suffice to bring them back in all their original intensity.

Bertin,[1] indeed, recommends a system of treatment very different from this. In the vain hope that the violent effort induced may cause the displacement of the impacted embolus (to which alone he attributes pulmonary obstruction), he recommends the administration of emetics. Few, I fancy, will be found bold enough to attempt so hazardous a plan of treatment.

Various drugs have been suggested in these cases. Richardson[2] recommended ammonia, a deficiency of which he at that time believed to be the chief cause of coagulation. He has since advised that liquor ammoniæ should be given in large doses, twenty minims every hour, in the hope of causing solution of the deposited fibrin; and he has stated that he has seen good results from the practice. Others advise the administration of alkalies, in the hope that they may favor absorption. The best that can be said for them is that they are not likely to do much harm. The inhalation of oxygen, which has been used with great success in severe pneumonia,[3] is obviously a hopeful remedy in this condition, and is well worthy of trial.

Puerperal Pleuro-pneumonia.—This is, perhaps, the best place to mention an important but little understood class of cases which I believe to be less uncommon than is generally supposed. I refer to severe pleuro-pneumonia occurring in connection with the puerperal state, but not distinctly associated with septicæmia. Two carefully observed cases of this kind are recorded by MacDonald, occurring in his practice; I myself have met with three very marked examples within the past three years, one of which proved fatal, the other two giving rise to most serious illness, from which the patient recovered with difficulty.

So far as my own observation goes there are marked peculiarities in such cases which clearly differentiate them from the ordinary course of pneumonia. The onset is sudden and unconnected with exposure to cold or other cause of lung disease; there is no definite crisis, but a continuous pyrexia of moderate intensity lasting a variable time; and the physical signs differ from those of ordinary pneumonia.

Physical Signs.—In MacDonald's case, as well as in my own, they were peculiar in this respect, that there was very slight crepitation, marked rusty sputum, and a wooden dulness, much more intense than in ordinary pneumonia, extending over a large lung space, with a very slight entrance of air into the lung tissue. It is also remarkable that a very large proportion of the cases were associated with phlegmasia

[1] Op. cit., p. 393.
[2] Heart Disease during Pregnancy, p. 209.
[3] "On the Use of Oxygen and Strychnia in Pneumonia," Brit. Med. Journ., January 23, 1892.

dolens. Thus it existed in one of MacDonald's two cases, and in two out of my own three. Like phlegmasia dolens, moreover, the disease generally commenced some weeks after delivery; my own cases, for example, occurred respectively fifteen, twenty-eight, and thirty-five days after labor. It is difficult to believe that there is not some connection between these two conditions, and there is much in their peculiar history to lead to the belief that such forms of lung disease depend, in fact, on the thrombotic or embolic obstruction of the minute branches of the pulmonary arteries, caused by conditions similar to those which have produced the phlebitic obstructions in the lower extremities. In the absence of careful post-mortem examination this hypothesis is clearly not susceptible of proof. MacDonald, while admitting that "a limited thrombosis of the pulmonary arteries would no doubt explain the facts of the cases," is rather inclined to "seek the chief explanation of their occurrence in the alterations which the pregnant and puerperal conditions impress upon the blood and the blood-vascular system."

I confess that to my mind the former hypothesis is not only the most definite, but the one which most readily explains all the peculiarities of these cases. I cannot, however, do more than suggest it, in the hope that further observations, and especially carefully conducted autopsies, may throw some light on this obscure and little-studied subject.

Treatment.—As regards treatment, it is obvious that it must be conducted on general principles, carefully avoiding over-severe measures, and supporting the patient through a trial to the system that must necessarily be severe.

CHAPTER VII.

PUERPERAL ARTERIAL THROMBOSIS AND EMBOLISM.

Arterial Thrombosis and Embolism.—The same condition of the blood which so strongly predisposes to coagulation in the vessels through which venous blood circulates tends to similar results in the arterial system. These, however, are by no means so common, and do not, as a rule, lead to such important consequences. The subject has been but little studied, and almost all our knowledge of it is derived from a very interesting essay by Sir James Simpson.[1] As I have devoted so much space to the consideration of venous thrombosis and embolism, I shall but briefly consider the effects of arterial obstruction.

Causes.—In a considerable number of recorded cases the obstruction has resulted from the detachment of vegetations deposited on the cardiac valves, the result of endocarditis, either produced by antecedent rheumatism or as a complication of the puerperal state. Sometimes the obstruction seems to depend on some general blood dyscrasia, similar to that producing venous thrombosis, or on some local change in the artery itself. Thus Simpson records a case apparently produced by local arteritis, which caused acute gangrene of both lower extremities, ending fatally in the third week after delivery. In other cases it has been attributed to coagulation following spontaneous laceration and corrugation of the internal coat of the artery.

[1] Selected Obstet. Works, vol. i. p. 523.

Symptoms.—The symptoms of puerperal arterial obstruction must, of course, vary with the particular arteries affected. Those with the obstruction of which we are most familiar are the cerebral, the brachial, and the femoral. The effects produced must also be modified by the size of the embolus, and the more or less complete obstruction it produces. Thus, for example, if the middle cerebral artery be blocked up entirely, the functions of those portions of the brain supplied by it will be more or less completely arrested, and hemiplegia of the opposite side of the body, followed by softening of the brain texture, will probably result. If the nervous symptoms be developed gradually, or increase in intensity after their first appearance, it may be that an obstruction, at first incomplete, has increased by the deposition of fibrin around it. So the occasional sudden supervention of blindness, with destruction of the eyeball—cases of which are recorded by Simpson—not improbably depend on the occlusion of the ophthalmic artery, the function of the organ depending on its supply through the single artery. The effects of obstruction of the visceral arteries in the puerperal state are entirely unknown, but it is far from unlikely that further investigation may prove them to be of great importance. In the extremities arterial obstruction produces effects which are well marked. They are classified by Simpson under the following heads: 1. *Arrest of pulse below the site of obstruction.* This has been observed to come on either suddenly or gradually, and, if the occlusion be in one of the large arterial trunks, it is a symptom which a careful examination will readily enable us to detect. 2. *Increased force of pulsation in the arteries above the seat of obstruction.* 3. *Fall in the temperature of the limb.* This is a symptom which is easily appreciable by the thermometer, and when the main artery of the limb is occluded the coldness of the extremity is well marked. 4. *Lesions of motor and, sensory functions, paralysis, neuralgia, etc.* Loss of power in the affected limb is often a prominent symptom, and when it comes on suddenly, and is complete, the main artery will probably be occluded. It may be diagnosed from paralysis depending on cerebral or spinal causes by the absence of head symptoms, by the history of the attack, and by the presence of other indications of arterial obstruction, such as loss of pulsation in the artery, fall of temperature, etc. The sensory functions in these cases are generally also seriously disturbed, not so much by loss of sensation as by severe pain and neuralgia. Sometimes the pain has been excessive, and occasionally it has been the first symptom which directed attention to the state of the