THE CAUSES OF SUDDEN DEATH OF PUERPERAL WOMEN.

AN ADDRESS

IN

OBSTETRICS AND DISEASES OF WOMEN AND CHILDREN,

DELIVERED BEFORE THE

AMERICAN MEDICAL ASSOCIATION, JUNE 5, 1878.

BY

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THE rule of the Association concerning one of the duties of Chairmen of Sections is, that they "shall prepare and read in the general session of the Association, papers on the advances and discoveries of the past year in the branches of science included in their respective Sections." The strictest rendering of this law gives the Chairman of a Section limited power of electing a subject for his address. As in this instance I have deviated somewhat from the letter of the law, I deem it proper that I should mention as my chief reason for it that the *American Journal of Obstetrics*, for several years past, and but a short time prior to our annual meetings, has published an excellent and exhaustive annual *résumé* of the latest views and more recent discoveries pertaining to obstetrics and gynæcology obtained from all parts of the civilized world. I was assured that it was the intention of the editor of that Journal to do this year as in preceding years.

I have, then, for very obvious reasons, chosen not to go over the entire field of obstetrics and gynæcology for the composition of this paper, and yet, adhering to the spirit of the law, I have selected as my topic one which has of late elicited the deepest interest of obstetricians and obstetrical writers. The subject of this paper is "The Causes of Sudden Death of Puerperal Women."

Were I to limit my paper simply to a review of all that has been published upon this subject within the last twelve months alone, I could dismiss it with but a few remarks. Indeed, the literature of the past which bears upon my theme, contains amid a mass of theories and reports of many cases, very little from

which one can deduce positive knowledge, as close analysis shows that there has been a paucity of facts.

The systematic works upon midwifery and puerperal diseases, many of which allude to the importance of the subject, contain only meagre chapters or brief allusions to the causes of sudden death of puerperal women.

Unexpected or sudden death after gestation was alluded to and the histories of cases given by the older obstetrical and medical writers, among whom were Mauriceau,[1] Peu,[2] Morgagni,[3] De La Motte,[4] and Madame Lachapelle.[5]

In 1814 Ramsbotham[6] attributed sudden death above all other causes to moral emotions.

Our distinguished countryman, the late Prof. Charles D. Meigs,[7] published in 1849 a memoir upon the sudden death of lying-in women from heart-clot. He was one of the first to direct attention to the possibility of spontaneous coagulation of the blood in the right side of the heart and pulmonary arteries as a cause of death in the puerperal state.

Paget had taken a similar view concerning a case of sudden death in 1845, although none of his cases happened after delivery.

In 1850 McCormac[8] attributed the sudden death of puerperal women to the entrance of air into the circulation by the uterine veins.

McClintock admitted and afterwards rejected this hypothesis as a cause of sudden death, in an excellent paper written in 1852.[9]

Hecker[10] in 1855 was of the opinion that unexpected or sudden death occurred most frequently as the result of obliteration of the pulmonary artery, an embolism proper.

Moynier[11] and Mordret[12] each in 1858 wrote upon this subject.

In 1863 Olhausen[13] wrote upon the introduction of air by the uterine veins as a cause of sudden death.

[1] Traité des mal. des femmes et de celles qui sont accouchées, Paris, 1668–94.
[2] La Pratique des accouchemens, Paris, 1694.
[3] De sedibus et causes morborum, Lond. 1763.
[4] Traité des accouch. naturels, non naturels et contre nature, Paris, 1715.
[5] Pratique des accouch., Paris, 1825.
[6] London Medical Repository, 1814.
[7] Philadelphia Medical Examiner, 1849.
[8] Provincial Medical and Surgical Journal, 1850.
[9] Dublin Medical Press, 1852.
[10] Deutsche Klinik, 1855.
[11] Mém. Acad. de Méd., Paris, tome xxii.
[12] De la mort subite dans l'état puerperale, Thèse, Paris, 1858.
[13] Monatsch. für Gebur., tome xxviii.

Hervieux, who already in 1864 and 1868 had published two articles upon sudden death after accouchement, devoted a long chapter to the subject in his voluminous work upon puerperal diseases in 1870.[1] He attributes sudden death of puerperal women to lesions either of the circulatory, respiratory, or nervous systems, but gives the greatest prominence to puerperal poisoning.

The same year M. Challier de Grand Champ,[2] in a thesis announced that sudden death after gestation was most often due to pre-existing causes, the principal one being an alteration of the blood.

In 1873, M. Lamy[3] wrote upon sudden deaths of puerperal women, which he believed most frequently due to an alteration of the blood, or to an organic affection itself aggravated by parturition, or to nervous susceptibility.

Coste,[4] in his memoir, in 1876, upon puerperal myocarditis, announces in the most emphatic manner that this disease is the principal cause of sudden death of puerperal women—"*que la mort subite après l'accouchement est presque toujours le resultat d'une myocardite.*"

Sir James Simpson and our distinguished countryman, Prof. Fordyce Barker, in his excellent work upon the subject,[5] have each added valuable contributions to the literature of the subject. Winckel[6] also devotes a short chapter to it.

Through the *Transactions of the Obstetrical Society of London* are scattered papers upon the causes of sudden death of puerperal women.

In the medical journals of late years under various titles are many cases noticed of sudden death of puerperal women, with different theories given as to the causes of death. Many of these contain no pathological facts, and are not deserving of any special notice. The most noticeable and worthy articles of which I have any knowledge, that have not been alluded to, will be mentioned later in this paper.

[1] Traité clin. et prat. des malad. puerp. suites de couches, Paris, 1870.

[2] Thèse, Paris, 1870.

[3] De la mort subite, des femmes en couches. Thèse, Paris, 1873.

[4] De la myocardite puerpérale, Paris, 1876.

[5] Puerperal Diseases. By Fordyce Barker, Clinical Prof. of Midwifery in the Bellevue Hospital Medical College, etc. etc. New York, 1878.

[6] The Pathology and Treatment of Childbed. By F. Winckel, Prof. in Univ. of Rostock. Translated by James R. Chadwick, M.D., of Boston. Philadelphia, 1876.

DISEASES OF THE CIRCULATORY SYSTEM AS A CAUSE OF SUDDEN DEATH IN A PUERPERAL STATE.

Among the causes of sudden death of puerperal women, will be considered, diseases of the circulatory system.

Of the valvular lesions of the heart, mitral insufficiency seems to imperil the puerperal woman least, and mitral stenosis most. McDonald[1] has collected eight puerperal cases complicated with mitral insufficiency. In these cases there were three deaths, and five recoveries. Only two of the deaths could be called sudden, or 25 per cent. The same author has collected twelve cases of chronic cardiac disease, in which mitral stenosis was the leading heart lesion.

A brief abstract of these cases will better enable us to estimate the relation of this lesion to sudden death in puerperal women.

Case I. Mitral stenosis, hæmoptysic, palpitation, dyspnœa, at end of sixth month of first pregnancy; pulmonary and general œdema in the second pregnancy; labor supervening spontaneously about the end of eighth month; death sudden on sixth day after delivery.

Case II. Mitral and tricuspid stenosis, with insufficiency; dropsical symptoms in 1873, a month after confinement; embolism of branch of left middle cerebral artery, with hemiplegia of the right side three months afterwards; recovery. Pregnancy again in 1878, with great aggravation of previous symptoms of dropsy and dyspnœa. Labor premature at beginning of ninth month; easy. Death from exhaustion; contraction of both auricular-ventricular orifices; enlargement of both ventricles; atrophy of posterior half of left corpus striatum.

Case III. Mitral stenosis; hemiplegia, with a certain degree of aphasia; labor premature in consequence of accidental hemorrhage on 21st of February, 1877, being about three weeks too early. Urgent symptoms during delivery, which was ended by forceps. Recovery.

Case IV. Mitral stenosis; labor at full term; threatened symptoms of weakness and iregularity of the pulse during the second stage. Delivery by forceps. Recovery.

Case V. Mitral stenosis; miscarriage about fifth month, and death thirty-six hours afterwards.

Case VI. Mitral stenosis; breathlessness from the age of

[1] Obstet. Journ. Great Britain and Ireland, 1877.

fifteen; symptoms much worse after marriage. Miscarriage about fourth month of pregnancy, and sudden death immediately after. *Post-mortem* revealed great mitral contractions. Both auricles and right ventricle dilated and hypertrophied.

CASE VII. Extreme stenosis of mitral valve; violent palpitation and severe breathlessness for three or four years; sudden death from pulmonary engorgement, immediately after the patient's fourth labor. *Post-mortem* examination revealed the lungs healthy, but engorged with blood. Mitral valve only admitted tip of little finger.

CASE VIII. Stenosis of the left mitral of moderate amount. Severe chest symptoms (œdema and congestion of the lungs) during the later months of second pregnancy. Premature labor suggested, but came on spontaneously before arrangements could be completed, and patient died during confinement of suffocative œdema of lungs. *Post-mortem* proved internally œdematous. Heart ordinary size. Aorta *strikingly small;* pulmonary artery dilated; mitral valve contracted. This case is one of Hecken's, and was one of the first well-described cases directing the attention of obstetricians to this important subject.

CASE IX. Mitral stenosis, with insufficiency; dilatation of the right side of the heart. Severe chest symptoms from œdema of the lungs. Symptoms during labor very critical; venesection. Liquor amnii excessive. Recovery.

CASE X. Mitral stenosis with insufficiency; no special trouble before the sixth month; then breathlessness, palpitation, cough, expectoration of rusty sputum, and occasionally blood. Labor premature during seventh month, with unconsciousness and extremely alarming symptoms. After this, remission of urgent symptoms. Death nine months afterwards with Bright's disease. *Post-mortem* examination revealed a pericardium distended with fluid; heart twice the ordinary size; both auricles and right ventricle greatly dilated; left ventricle natural size; left auriculo-ventricular opening admitted only one finger. Lungs partly collapsed, partly emphysematous and œdematous.

CASE XI. Mitral stenosis; slight attack of acute rheumatism after second confinement, followed by severe illness involving heart and kidneys. Admitted to the hospital in seventh month of third pregnancy, with symptoms referable to mitral stenosis, with dilatation and venous congestion. Induction of premature delivery thought of toward end of eighth month, but labor super-

vened spontaneously, patient being in a state of unconsciousness. Death three months afterwards. *Post-mortem* examination showed great dilatation of right heart and left auricle; slight enlargement of left ventricle. Mitral opening too small to allow a finger to pass; valves thickened, evidences of pulmonary infarction recent, and of old-standing hydrothorax.

Case XII. Mitral stenosis and insufficiency, with aortic insufficiency; enlargement of left side of heart, consequent upon an acute attack following the tenth pregnancy, which was a placenta prævia. Aggravation of symptoms during the latter half of the eleventh pregnancy. Spontaneous premature labor in the eighth month whilst patient was in a state of unconsciousness. Cardiac symptoms much aggravated therewith, and death forty-eight hours afterwards. *Post-mortem* examination showed enlargement of left heart. Left auriculo-ventricular valve thickened and nodulated. Mitral opening allowed one finger to pass. The right heart only moderately dilated.

Of these twelve cases nine were fatal, and six of them during abortion or delivery, or within a few hours afterwards. In other words, seventy-five per cent. of the cases were fatal and fifty per cent. *suddenly* fatal.

Four of the cases were primipar ; and of these three died, and two very *suddenly*.

Three were pregnant for a second time; of these two died very suddenly. The other five cases, at the time they were under observation, were respectively confined for the third, fourth, sixth, tenth, and twelfth time; all died but the last, and two *very suddenly*.

In no case was death caused by embolism, but rather by suffocative œdema of the lungs, congestive bronchitis with or without actual pulmonary hemorrhage, extreme dyspnœa, and cardiac irregularity, culminating in abortion and death, either during or shortly after delivery.

From these facts we are bound to expect the above circle of phenomena to occur in a pregnancy complicated with a badly compensated mitral stenosis.

Dr. MacDonald, in the article quoted from, records five cases of aortic incompetency, of which the following is a brief abstract:

Case I. Aortic insufficiency, with mitral obstruction; pulse extremely irregular during labor; great tendency to faint. Delivery by forceps. Recovery.

Case II. Extreme aortic insufficiency, with great mitral obstruction; hæmoptysis; frequent cough, dyspnœa, and vomiting. Premature labor came on spontaneously at end of eight months. Dyspnœa and repeated attacks of syncope during labor. Death three weeks afterwards from suffocation, congestion, and œdema of the lungs. *Post-mortem* examination revealed the mitral orifice so contracted as to admit only the passage of a little finger. Aortic valves reduced to mere stumps; heart dilated and hypertrophied; lungs œdematous and congested.

Case III. Aortic insufficiency, with mitral stenosis, referable to an attack of rheumatic fever five years before. Suffered from a severe cold about the end of the third month of pregnancy. Severe symptoms began about the sixth month. Pain in left chest, palpitation, tendency to faint, hæmoptysis, nephritis, convulsions, parturition; *sudden death*. *Post-mortem* examination revealed aortic valves much diseased and acutely inflamed, and the mitral much stenosed.

Case IV. Severe case of aortic insufficiency. Violent precordial pain and dyspnœa at about the twenty-eighth week of pregnancy. Improvement under treatment for a time; but a return of the distressing symptoms on slight exertion. Pregnancy interrupted by spontaneous premature labor about the thirty-fourth week. Labor easy, and remission of urgent symptoms rapid.

Case V. Aortic insufficiency; slight case; dyspnœa and palpitation apparent about the sixth month. Under treatment the patient got better, but premature labor came on in the eighth month, and was followed by recovery.

Of the two suddenly fatal cases in this group, it will be observed that both were complicated with mitral stenosis, so that from what has been previously shown we may reasonably refer the fatal results mainly to the mitral obstruction.

In the *Revue des Sciences Médicales*, July, 1877, is given an exhaustive review of this subject, which includes Marty's table[1] of two hundred and four puerperal cases complicated by valvular lesions of the heart. In sixty-five cases there was mitral insufficiency; twenty-nine premature confinements, with frequent and grave complications; and three sudden maternal deaths. In

[1] Influence de la grossesse et des couches sur l'appareil circulatoire et troubles gravido-*cardiaques*, par Porak.

forty cases there was mitral stenosis, with twelve premature confinements; and three sudden maternal deaths. In fifty one cases there was mitral insufficiency and stenosis, with twelve premature confinements, and four sudden deaths. In fifteen cases there was aortic insufficiency, with but one complication, and no death. In five cases there was aortic stenosis, with two sudden deaths. In three cases there was aortic and mitral insufficiency, with one sudden death.

This table shows also that the liability to sudden death is far greater in mitral stenosis than in any other form of valvular disease.

ENDOCARDITIS AS A CAUSE OF SUDDEN DEATH IN THE PUERPERAL STATE.

Simpson was about the first to note the influence of the puerperal state upon endocarditis. This influence he supposed to be exerted through the presence of lactic acid in the blood of the pregnant woman.[1]

Virchow[2] regards puerperal endocarditis as a form of puerperal fever to be classed with the peritoneal forms, as both affect a serous membrane. At the time of his report upon the subject he had within a year and a half eighty-three cases of *sudden* death from this cause. All the cases were remarkable by the absence of lesions of the genital organs or of other parts of the body which could account for the intensity of the fever and other symptoms. His observations forced him to the conclusion that the disease of the lining membrane of the heart was the origin of other pathological changes. More frequently he found evidences of recent endocarditis which had caused a degeneration of the mitral valves. Particles of these broken-down valves had been loosened and carried into the circulation. These obstructed the capillaries and produced circumscribed inflamed spots.

Finally, Virchow found in the vessels supplying blood to these spots a plug composed of the *débris* of the softened valves. In one case death was determined by softening of the entire heart.

In one case of Virchow's, cited by Vast, and later by Martineau, of ulcerative puerperal endocarditis, a *post-mortem* exami-

[1] Obs. Memories, etc., Edin., 1856, page 69.

[2] Gynæcological Society of Berlin, March 9th, 1858.

nation revealed the uterus perfectly sound, but in a great number of organs were found the so-called metastatic spots, viz., in the kidneys, spleen, liver, eyes, etc. In each of these spots was found an artery plugged with detritus of the broken-down endocardium. Hence in all cases of puerperal disease in which there exist these metastatic spots, and the condition of the abdominal organs does not account for the great frequency of the pulse and general disorder, a careful examination of the heart is advised.

Ahlfeld[1] records a case of acute endocarditis, attended with vegetation on mitral valves, leading to capillary cerebral embolism, convulsions, delivery by forceps, and sudden death. *Post-mortem* examination revealed vegetations of the mitral valves from the size of a millet-seed to that of a pea. Extravasation of blood under the pia mater; hundreds of capillary apoplexies in the gray substance of the brain, some about the size of a millet-seed or pea, one the size of a lentil in the pons Varolii.

Lebert[2] reports a case of acute articular rheumatism in the fourth month of pregnancy; there was an abortion in the sixth month, followed by ulcerative endocarditis and embolism of the left subclavian, the abdominal aorta, and common iliac artery; sudden death. *Post-mortem* examination showed a flabby, dilated, large fatty heart, extensive ulceration of the mitral valves, more especially of the posterior cusp, which was completely destroyed. Emboli were found in left subclavian, abdominal aorta, and both iliacs.

Of four cases recorded by Simpson,[3] in which a *post-mortem* examination proved the embolism to be due to the escape of cardiac vegetations, one of the patients had, a year previous to the fatal pregnancy, suffered from rheumatic endocarditis. Another had an attack of acute rheumatism during the pregnancy, and died a few days after delivery. A third suffered from endocarditis supervening only after her premature delivery. A fourth was due to ulcerative puerperal endocarditis. These cases especially illustrate with great force the risk of severe embolism and sudden death which cases of recent rheumatic endocarditis run by becoming pregnant, and so lighting up a fresh and more severe endocarditis. The practical deduction clearly is, that all

[1] Archiv für Gynäk., Bd. iv. S. 158.
[2] Ibid., Bd. iii. S. 10 und foly.
[3] Selected Obstet. Works, p. 525.

women who have suffered from endocarditis should be discouraged from marrying or becoming pregnant, as the peculiar changes of the pregnant and puerperal state render it probable that an ulcerative endocarditis will be excited and bring with it embolism and sudden death.

Fatty Heart.—That a fatty heart should be a cause of sudden death in puerperal women we may readily admit, since the most reliable observations of all deaths in persons affected with this disease place the sudden deaths at two-thirds of the entire number. But as it occurs during the latter half of life, and is one of the degenerative changes belonging to that period, it is obvious that it must be relatively infrequent in puerperal women.

Of the positive observations I have been able to find but two cases in which a sudden death was due to a fatty heart.

The first case was reported by myself.[1] Patient aged thirty-six; had been out of health for some years. Was safely delivered of her third child with no particular difficulty, but died suddenly about three hours afterwards. *Post-mortem* examination revealed a markedly fatty heart and kidneys. Uterus was contracted and normal. Careful microscopic examination was made of the heart, demonstrating beyond a doubt its fatty degeneration.

The other case is reported by Winckel:[2] Woman, aged thirty-five, was delivered of her fifth child. The labor was difficult; the funis prolapsing, and forceps being required. Ulcers on the cervix uteri were the source of a parametric exudation on the right side as large as one's fist; there was also a continued fever. Patient died quite suddenly on the sixth day wi h symptoms of pulmonary œdema. The autopsy showed fatty degeneration of the heart.

We may fairly suspect that a closer *post-mortem* examination of all the cases of sudden death in the puerperal state would many times reveal a fatty heart as the organ at fault.

Coste,[3] in his monograph before mentioned, has especially emphasized myocarditis as a very common cause of death in puerperal women. But of the five reported cases on which he bases his conclusion, at least four of them seem to us more nearly to

[1] Detroit Review of Medicine and Pharmacy, vol. vi. p. 345.

[2] Winckel on Childbed, 1876, p. 221.

[3] Op. cit., p. 73.

fall under the head fatty heart; and even in the remaining one, it is not at all clear that the sudden death was caused by myocarditis alone. As it is almost impossible to diagnosticate the disease during life, we pass it with this brief mention as a possibly rare cause of sudden death in puerperal women.

DISEASES OF THE ARTERIES AS A CAUSE OF SUDDEN DEATH IN PUERPERAL WOMEN.

Local arteritis as a cause of sudden death in puerperal women has not been very carefully studied. All admit that it speedily ends in the formation of an obstructing fibrinous or sanguineous concretion in the tube of the inflamed artery.

Simpson[1] reports a case observed with the most scrupulous exactness. The patient was a primipara, and two weeks after delivery was brought into the hospital suffering from gangrene of the lower extremities. Death occurred soon. An autopsy revealed an entire absence of disease in the heart, its walls, valves, or cavities. But an inch and one-half above the bifurcation of the aorta was found a fibrinous plug occluding the entire artery and extending along the common iliac, so as to obstruct both the external and internal iliacs on each side. The upper part of the plug was flattened and closely adherent to the arterial walls. At the most inflamed part the calibre of the artery was contracted.

More positive observations are needed in making autopsies of those suddenly dying in the puerperal state to determine the structure of the existing plugs and the exact condition of the arterial wall at the point of obstruction.

With such observations we may more accurately estimate the actual importance of this cause of sudden death.

That arteritis may produce sudden death by forming and setting free emboli, we have the most abundant evidence from the records of Simpson, Meigs, Playfair, Paget, Baron, Dubin, Richelet, and others. The special locality in which they observed the most fatal results from this source, was the pulmonary artery.

Whether death is caused through syncope, as held by Virchow, or through cerebral anæmia, as thought by Panum, or through asphyxia, as claimed by Bertin, may be still a question. It is

[1] Obstetrical Works, p. 539.

evident that all the results are due to the fact that the blood is kept from the air.

The connection of phlebitis with sudden death in the puerperal state has long been recognized, especially since the fact has been made clear that fragments of the plugs caused by the phlebitis may be set free and form obstructions in the vessels elsewhere, particularly in the pulmonary circulation.

Simpson[1] records four cases of plugging of the pulmonary arteries in the puerperal state, in which death occurred with fearful suddenness and rapidity. In all of these instances uterine or cerebral phlebitis preceded the pulmonary obstruction.

Winckel, Levy, Hecker, Makinder, Charcot, Hervieux, Steele, Chantreuil, and others, report similar cases, in which an early and sudden death followed the detachment of a portion of the thrombus.

It is of interest to note that in most of these cases the cause of the sudden embolism is ever-exertion, such as lifting, stooping, rapid movements, etc. Thus a piece of the primary thrombus is broken off, and in a few moments carried to the pulmonary artery.

The relation of hemorrhage, ante- and post-partum, to sudden death is so fully dwelt upon in all obstetrical works, and lectures, and magazine contributions, that we pass it with a simple mention.

Few obstetric practitioners are devoid of terrible experiences fully illustrating the relation of such hemorrhage to sudden puerperal deaths.

As a cause of puerperal diseases in general, *septicæmia* has given rise to widespread and long-continued discussion and difference of opinion. That it has been an influential cause of sudden death in puerperal women there can be no doubt. As it has appeared in hospitals or epidemics in various places, it has produced appalling lists of sudden death.

You will remember that in London during the years 1760, 1768, and 1770, it so prevailed that in some lying-in hospitals almost every patient died. It is said of the Edinburgh Infirmary in 1773 that "almost every woman, as soon as she was delivered, was seized

[1] Obstetrical Works, p. 346.

with this disorder, and all of them died." Nor did the lying-in establishments on the Continent fare better. Thus the *Maison d'Accouchements* of Paris during a number of different years presented a mortality of one in three or even greater. In Vienna during 1823, nineteen per cent. of the cases died, and in 1842 sixteen per cent. In Berlin, in 1862, hardly a single patient escaped.

Numerous other instances here and abroad will occur to you all and sufficiently emphasize the statement that septicæmia in its most malignant forms is one of the chief causes of sudden death in puerperal women. To illustrate this matter Prof. Barker[1] reports the following:—

A primipara during the seventh month of her pregnancy began rapidly to fail in health, lost strength, became dull and sleepy; had slight fever; vomiting; offensive diarrhœa. Refusing to have labor brought on at once, she rapidly sank, so that in four days she became unconscious. Pulse rapid; skin cold. In a short time the membranes were ruptured; labor came on; and with little trouble she was delivered of a putrid fœtus. She died a few hours after. In this case the source of infection was the dead fœtus.

Equally suddenly fatal cases are on record of puerperal women dying from a blood poison derived from broken-down portions of the placenta, or gangrenous destruction of a part of the neck or other portion of the uterus.

So also cases of sudden death are well authenticated from a poison carried to the women by a nurse or physician, or carried from cases of erysipelas, scarlet fever, diphtheria, etc. The sources of the poison are both autogenetic and heterogenetic.

In short, it matters little whether the poison be engendered in some part of the generative tract of the woman or carried to it from without; if only she is in proper condition to be profoundly affected by it, sudden death is almost inevitable.

The suddenness of the death is regulated by two factors, the susceptibility of the patient and the virulence of the poison. When both factors exist to a considerable extent, remedial agents are as futile as in cases struck dead by lightning, or as in cases overwhelmed by any other powerful poison, as morphia, atropia, or arsenic, etc. Apparently the human protoplasm may be de-

[1] Puerperal Diseases, p. 402.

prived of its essential properties with great rapidity by the peculiar form of poison under consideration.

The relations of septicæmia to thrombosis and embolism are not fully worked out.

Recent investigations respecting the phenomena of coagulation of the blood seen to indicate a plausible explanation of one phase of this relationship.

Many years since Prof. Schmidt demonstrated that no fibrine exists in the living healthy body; and that the fibrine of the blood-clot is derived from the union of the fibrino-plastin and fibrinogen of the blood. Later he demonstrated that in order to produce a union of these two factors a third substance was necessary. This third substance has been traced to the white blood-corpuscles; but in order that it may act in producing fibrin it must be set free by the disintegration of these corpuscles.

The reason for believing that the white blood-corpuscles contain the ferment necessary to the coagulation of blood are well stated by Dr. Burdon Sanderson.[1] First he shows that certain white blood-corpuscles disintegrate from the moment that they leave the blood stream. The experimental proof of this is as follows: Blood from an artery or vein is caught in a tall jar in which it is rapidly cooled by a freezing mixture placed about it. Thus treated, the blood remains uncoagulated, the red blood-disks sink to the bottom, the white ones rise to the top, and serum separates the two. If under a microscope we examine almost immediately the top layer, we shall find countless white blood-corpuscles. But if a few moments have elapsed a certain number of these corpuscles will be observed in all stages of disintegration.

In the process of disintegration the corpuscles first break up into granules, but eventually disappear. From these granules the first formed filaments of fibrine are seen to take their origin. Further, if the white blood-corpuscles be withdrawn, coagulation is arrested. Proof of this statement is rendered possible by the fact that leucocytes at the temperature of freezing acquire such firmness and consistence, that they are held back by the ordinary methods of filtration.

Thus by appropriate apparatus, uncoagulated blood can be freed from its white blood-corpusles. The filtrate thus obtained is absolutely transparent and deprived of its power of coagu-

[1] British Medical Journal, Jan. 12, 1878.

lation. If the white blood-corpuscles on the filter be washed and added to the decorpusculated plasma, the latter is restored to its original coagulability.

With these facts before us, and those cases in which cerebral, pulmonary, and other embolisms occur to cause sudden death in the puerperal state, we get an intelligent suggestion as to the mechanism by which the fatal result is brought about.

The uterine sinuses and the internal surface of the uterus are loaded with breaking-down blood-corpuscles, white and red. Usually the balance of nutritive forces is such that all the effete materials are carried out of the uterus, and discharged from the body as lochia. In certain exceptional cases this balance is lost, and more or less of the effete material is carried into the venous circulation. This material, by its contact with the fibrinogen and fibrino-plastin of the blood, is able to produce blood-clots, and so emboli more or less numerous, according to the circumstances of the case. We have no doubt that very many cases of embolism have this direct origin.

Meantime other elements of the effete material are producing their depressing and disorganizing effects upon other protoplasmic elements of the body. For one purpose it matters not whether we regard the poison of puerperal fever as one *sui generis* or not. Under definite circumstances we very certainly observe but one result, viz., sudden death. As we have indicated, this may be brought about, *first*, by the formation of emboli in the blood, through the action of the disintegrated white blood-corpuscles; *secondly*, by the paralyzing influence of other poisonous elements upon other protoplasmic structures essential to life; and *thirdly*, by the simultaneous formation of emboli and general poisoning.

If we accept the view that broken-down white blood-corpuscles circulating in the blood may determine the formation of blood-clots, we can readily understand how a ferment derived from the uterine sinuses may pass through the capillaries of the lungs, and afterwards produce an embolus in the cerebral arteries, or capillaries, or in the liver, kidneys, or any part of the general capillary or arterial systems.

We look for researches which will render equally clear the constitutional effects produced by the other elements of puerperal poisoning, which are doubtless more far reaching in their effects and equally as fatal.

The introduction of air into the uterine veins is an occasional cause of sudden death in puerperal women. McClintock cites six examples which were probably due to this cause. Lachapelle[1] relates two; Baudelocque[2] two; Simpson, Lionel, and Hervieux, each one; Bussy[3] of Birming am, in fifty cases of sudden death in the puerperal state, found air in the heart and uterine veins. Smith[4] reports one case; Leven[5] reports three cases of similar nature. In most of these cases a careful *post-mortem* examination confirmed the diagnosis.

The chief symptoms produced by this accident are due to the more or less complete stoppage of blood in the pulmonary capillaries. Unquestionably after delivery the uterine sinuses are nearly as well adapted as the veins of the neck to facilitate the entrance of air.

If in any manner air enters the uterus it is easy to understand how the alternate contraction and relaxation of the uterus would open the mouths of the sinuses and force air into the veins. Doubtless the entrance of air is facilitated by an exhausting hemorrhage, such as would temporarily lower the venous blood-pressure.

Dr. Graily Hewitt[6] refers to a case in which sudden death occurred shortly after the removal of an adherent placenta, and under symptoms that rendered it certain that the cause was the entrance of air into the veins. The introduction of the hand into the uterus for turning or other intra-uterine operations is occasionally the cause of sudden death by facilitating the introduction of air into the veins.

Tetanus is an occasional cause of sudden death in the puerperal state. Simpson[7] has collected twenty-five cases. Of the nature of the disease it cannot be said to differ at all from that which ocurs in the ordinary surgical practice. Here as elsewhere, three conditions seem essential to determine its occurrence: *first*, a wound; *second*, an undetermined sensitive condition of the nervous system; and *third*, a peculiar state of the atmosphere. There is good evidence for believing that a neuritis is originated in the sensitive nerves at the point of the wound, and that this

[1] Op. citat., p. 76.
[2] Op. citat., p. 16.
[3] Arch. für Gynæc., Bd. iii.
[4] British Med. Journal, 1856.
[5] British Med. Journal, 1857.
[6] Lond. Obstet. Trans., vol. x. p. 28.
[7] Obstet. work, p. 510.

extends upward to the motor tract of the spinal cord, producing in the latter a hypersensitive condition. Be this as it may, the fact remains undoubted that tetanus may cause sudden death in the puerperal state.

Another cause of sudden death of lying-in women is *puerperal convulsions*. The older obstetrical teachers were accustomed to say that one-half of the patients attacked with this disease died. Barker, from statistics of published cases collected in 1855, showed that thirty-two per cent. of all cases occurring before and during labor, and twenty-two per cent. of those after delivery ended fatally. From his own practice and in consultation, Dr. Barker has seen since 1855 sixty-five cases of puerperal convulsions, with a mortality of nine cases, or about fourteen per cent. Of course these cases had the advantage of the most recent and skilful modes of treatment, so that we may not expect to find better results elsewhere. No doubt the increased frequency with which chloroform and chloral have been used has greatly diminished the death rate.

For our purpose it is needless to discuss the causes of puerperal convulsions. Obstetrical literature is largely occupied with such diseases.

Other infrequent causes of sudden death in the puerperal state, which lack of time forbids our dwelling upon at length, are: *Rupture of the heart; rupture of the uterus; apoplexy; profound moral emotion*, and *mental excitement; shock*, and *traumatism*.

To conclude, we may remark (1) The causes of sudden death fall naturally under four heads: (*a*) Lesions of the circulatory system; (*b*) Lesions of the respiratory system; (*c*) Lesions of the nervous system; and (*d*) Puerperal septicæmia.

(2) In its wide-reaching relations to the other causes of sudden death, puerperal septicæmia stands pre-eminent, as very many lesions of the circulatory, respiratory, and nervous systems are the direct or indirect results of blood poisoning.

(3) It is more than probable that small and large emboli may be formed in any part of the circulatory system by the action of disintegrated blood-corpuscles upon the fibrinogen and fibrino-

[1] Puerperal Diseases, p. 125.

plastin. The minuteness of the particles of white blood-corpuscles enables them to pass with the greatest freedom through capillaries of the smallest diameter and afterwards by their ferment acting upon other elements to produce concretions in the vessels. Obviously the plain duty of every obstetrician is to prevent the absorption of any decomposing materials from the uterus. Emphatic as was this statement of old, it gathers new meaning and greater force from the new researches respecting the agency of decomposing white blood-corpuscles in producing blood coagulation.

(4) Of the various valvular lesions mitral stenosis is clearly the most dangerous to the pregnant or puerperal woman. In fact the liability to sudden death of those affected with this heart lesion is frightful.

(5) The usual mode of sudden death in puerperal women affected with valvular lesions is by the damming back of blood upon the lungs, causing pulmonary œdema, etc.

(6) Endocarditis, old or recent, if existing in a woman at the beginning of pregnancy, is extremely liable to be re-kindled by the changed blood of the pregnant woman, and to run the course of ulcerative endocarditis. Thus material for countless emboli is furnished, and the woman's life placed in imminent peril.

(7) Endocarditis may result as one of the forms of the so-called puerperal fever, just as the peritoneum may become involved in the same diseased process by the same blood poisoning.

(8) Arterial degeneration may produce death in two ways: (*a*) The white blood-globules caught on the roughened surface of the vessels may be broken in pieces, and the ferment set free, which unites in a blood clot the fibrinogen and fibrino-plastin; (*b*) An increase of the arterial pressure may rupture the diseased vessel, and so cause sudden death.

(9) Arteritis is a very rare occasion of sudden death.

(10) Phlebitis, on the other hand, is a very frequent source of the gravest complication of the puerperal state, viz., sudden death.

(11) No more certain cause of sudden death exists than the introduction of large quantities of air into the uterine sinuses. The anatomical relation of the mouths of the uterine sinuses are such that the relaxation of the uterus leaves them more or less open. If air be present in the uterus it will fill the partially open sinuses, and by the next uterine contraction be forced into the

venous circulation, and so to the right side of the heart and pulmonary capillaries.

Relatively, however, the entrance of air into the veins is a very infrequent cause of sudden death in the puerperal state.

(12) Of the causes of sudden death to puerperal women operating mainly through the nervous system, puerperal eclampsia is the most conspicuous. But its nature, its origin, and its treatment have been so unfolded during the last twenty years, that its death-rate has been vastly diminished.

(13) Finally, every physician should feel himself bound by a professional obligation to investigate with the most painstaking minuteness the pathological anatomy of every case of sudden puerperal death that falls under his observation. With a broader scope of reliable data, the conditions underlying each cause of sudden death may be so thoroughly made known that most sudden deaths may be prevented.

As already stated, the mortality of puerperal convulsions during the last twenty years has been reduced from thirty-two to fourteen per cent. This is but one illustration of what has been done by our science and art in the past, but it is a significant prophecy of what we may expect in the future.