ADDRESS IN OBSTETRIC MEDICINE.

BY

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ON PUERPERAL FEVER.

MEETINGS such as those of the great Association under whose auspices we are here assembled have always appeared to me as having their most useful function in presenting opportunities for reviewing the work we have been doing; estimating the value of the results attained; eliminating, so far as may be, the erroneous or defective; and so getting plainly before us certain standpoints from which further advances may be made. I propose, then, in endeavouring to accomplish the duty imposed on me by your Council when they selected me for the high and honourable office of delivering an address on obstetric medicine, to take Puerperal Fever as my subject, and to treat it in accordance with these principles. In selecting this subject I have been influenced not only by its importance—its vital importance—to one and all of us, but also by the great advances that have recently been made in our knowledge of its pathology, and in some measure perhaps by the feeling that in our enthusiastic pursuit of the new paths that have been opened to us we are in danger of losing the clue that is to lead us out of the labyrinth in which we have so long been wandering. I shall ask you to consider the disease mainly from a clinical point of view, referring to the laboratory experiments of scientists and pathologists to illustrate and explain the facts observed at the bedside, where alone disease can be studied. In doing this I shall not attempt to present any new or original doctrines. My task must be the more humble one of stating, with what clearness I may, the questions at issue, and the facts on which their solution must be founded—endeavouring to arrange and balance the evidence, so that its true value may be more fully apprehended.

Much of the difficulty that has always attended the study of this disease arises from the similarity of the symptoms during life, and of the pathological appearances found after death in its various forms. Its dependence on the great act of childbirth, and the peculiar condition of the patient's system at this period, sufficiently account for this similarity. The enlarged and highly organised uterus suddenly emptied of its contents after violent muscular exertion, and becoming a mass of effete material, whose constituent elements have to be absorbed into the blood as a preliminary to their being eliminated from the system; the exposed state of its vessels; the torn and contused tissues; the great current of blood diverted from the course it has so long been pursuing; the altered condition of the fluid itself, and the highly strained nerve-tension, all tend to concentrate the diseased action on one set of organs, and to impress on it this similarity of development. The attempts, consequently, that have from the earliest times been made to differentiate the various forms of the disease, founded on its morbid anatomy and its symptoms, have failed to simplify its study or in any great degree elucidate its nature. A classification founded on the conditions under which the disease arises, and the nature of the exciting cause adopted by some writers, as by Barnes in modern times, has proved more useful and done more to promote accuracy. One founded on this principle will probably best serve the requirements of the present occasion.

A writer of the last century, Kirkland, upbraids his contemporaries with including "a thousand disorders" under the one head of puerperal fever. It requires but a very elementary knowledge of the subject to see that a large proportion of these disorders arise from traumatic causes. These, then, will constitute the first group I will ask you to consider, and perhaps not the least important, seeing that so much can be done for their prevention.

Traumatic Cases.—In tedious and difficult labours, more especially in first labours, the soft parts of the mother are liable to be injured at every stage—the cervix may be lacerated, the lips may be caught between the head of the child and the side of the pelvis, and contused; the vaginal walls may be compressed till sloughing be induced, so as to lay open the bladder or the rectum, or both; the perinæum may be

torn, or some of the blood-vessels may be lacerated, and blood be effused into all the surrounding tissues, giving rise to hematomæ, more or less extensive; and yet, formidable as the enumeration seems, we all know that, in the great majority of cases, the reaction is moderate, the inflammation set up is localised; if the damage done be not excessive, it is repaired, and the patient recovers. But if the patient be in an unhealthy condition, or if her surroundings be unwholesome, then a new train of symptoms may arise, not necessarily depending on the extent or gravity of the wounds, but influenced by them. The inflammation, instead of being normal in character, becomes abnormal; it may extend to surrounding parts, and peritonitis or pelvic cellulitis may be set up; or more distant parts may be affected, and the disease will manifest itself in the pleura or the serous membranes of the heart or of the joints; or the blood in the veins may be coagulated, giving rise to thrombi and emboli, and subsequently pyæmia.

We have now to inquire what is the nature of this change. The researches of modern pathologists and scientists have proved that it is due to the introduction of a poison into the system, and that certain vegetable organisms (bacteria) are essential agents in the production and dissemination of this poison. They are not agreed, however, as to the exact part played by these organisms. Some hold that the most severe injuries may be inflicted without producing inflammation, and that when inflammation takes its origin from a wound, all that the wound does is to play the part of the door of entrance for the real *noxa*—water-germ, air-germ, or germ which sticks to the surface of the weapon or missile which inflicts the injury. They assert that, in the genesis of every inflammation, however definite may be the relation between the antecedent damage and consequent reaction, this relation is, after all, merely secondary. The true cause is the introduction into the damaged tissue from the atmosphere of invisible germs. Where there are no germs, there is no possibility of inflammation. Others, as Dr. J. Burdon Sanderson, to whom we are indebted for most of our exact knowledge on the subject, teach that inflammation is the direct physiological effect of injury, and that the only inflammations to which minute organisms stand in relation, are those which from their proved dependence on previously existing inflammation may properly be termed secondary or infective, and that consequently the organisms in question were in all probability not so much mischief-makers as mischief-spreaders; that is to say, although an inflammation may come into existence without their aid, their presence communicates to it, after it has come into existence, the power of reproducing itself in previously healthy tissues, whether by extension or dissemination. During the ten years which have elapsed since 1872 (when he first taught this), Dr. Sanderson says the question of the intervention of micrococci, which was then strange even to professed pathologists, has become familiar to everyone, and now the tendency exists to believe that germs explain everything; so that, whereas formerly one had to vindicate the very existence of such things as parts of pathological processes, it has now become a part of one's business to protest with all needful vehemence against the attribution to them of functions they do not possess.¹

In support of his theory, he proves a series of propositions on an experimental basis that seem to leave no room for rational doubt. He shows that the introduction into the circulating blood of a relatively large quantity of exudation, which is the most important effect of a considerable local inflammation, does not produce secondary inflammation elsewhere, and is not attended with any serious constitutional disturbance. He further shows that no organisms endowed with inflammation-producing, phlogogenic particles, exist in the atmosphere or in the ordinary aqueous liquids with which our bodies come into contact.

It had been proved by Hiller that bacteria collected from fluids in an advanced state of putrefaction might, when washed and purified, be introduced into the circulating blood without producing any injurious effect, though proved to be as lively and capable of development as ever. Hiller² even inoculated himself with them. Dr. Wegner³ showed that the subcutaneous tissue of a rabbit may be kept distended for days by frequently renewed injections of unpurified air without producing inflammation, and that the effect was not more serious if the same tissue were ventilated for a number of hours by a continuous current of air, even when that air was derived from a *post mortem* theatre. When water, charged with septic bacteria, was injected into the peritoneum of a rabbit, in quantity less than two ounces, it pro-

¹ Lumlleian Lectures on Inflammation. Delivered before the Royal College of Physicians. By J. Burdon Sanderson, M.D., etc. BRIT. MED. JOURNAL, April 8th, 1882, p. 493.

² *British Medical Journal*, January 5th, 1878, p. 2.

³ *British Medical Journal*, April 15th, 1882, p. 527.

duced no effect whatever. When the animal was killed, the fluid had been absorbed and the lymphatics of the diaphragm were full of bacteria, but neither in them, nor in the peritoneum, were there any signs of inflammation. Similarly it was found, that when a current of the fluid was passed through the peritoneum for many hours, so that the membrane was exposed for a long time to whatever phlogogenic influences the liquid conveyed, no effect whatever was produced. These experiments with watery liquids are even more conclusive than those with air, for we have here to deal with media, in every particle of which we can very readily prove the actual existence of germs in such a state of viability as to only require a suitable soil to insure their development.

Having thus proved that the exudation of a normal inflammation is not infective, and that no organisms endowed with inflammation producing "phlogogenic" particles exist in the atmosphere or in the ordinary aqueous liquids with which our bodies come into contact, Dr. Sanderson proceeds to show that, whenever an inflammation becomes infective, it owes that property to a chemical change in the exudation-liquid, of which the presence of microzymes is a necessary condition, and that, conversely, septic organisms, which are infectious, owe their infectiveness to the exudation-soil in which they have grown. He refers to experiments he made a dozen years ago, which showed that, in rodent animals, peritonitis induced by the injection of irritant liquids is always infective, notwithstanding that the irritant (tincture of iodine) was not only free from germs, but destructive of them. This was, he says, understood to mean that the first effect of the iodine was the effusion of liquid into the peritoneal cavity; the second, the infection of this liquid by formed ferments which found their way into it, not from the atmosphere or from external media of any kind, but from the alimentary canal. In another experiment, water which had been boiled so as to remove all germs that might have been in it, was, when cool, injected with antiseptic precautions, in so large a quantity that it could not be rapidly absorbed. When thus accumulated in the serous cavity, it charged itself by diffusion, first with the salts, subsequently, in increasing proportion, with the proteids of the blood, and thus became a most favourable soil for the development of septic organisms, which in their turn acted infectively or phlogogenically on the blood-vessels and living tissues with which they came in contact. A septic peritonitis ensued, which killed the animal by septicæmia in twenty-four or thirty-six hours. It is thus shown that, though the seed is indispensable to the coming into existence of the infective process, the question whether that seed becomes morbid or not, depends not on the seed itself, but on the soil. Other experiments prove the same thing, and the whole result is to establish that the presence of septic organisms (provided that they have not been bred inside the organism) is not necessarily attended with any danger, but that they become sources of danger at once when they are brought into relation either with devitalised organs, or with extravasated serous fluids of any kind, and particularly with inflammatory exudation.

The sum of our knowledge of these organisms seems to be this: They are found everywhere—in the air, in water, in the soil; in the bodies of the most healthy animals, on the surface where most exposed to the air; in the entrances to the alimentary and air-passages; in the stomach and upper portions of the alimentary canal, but not frequently here, for acid secretions kill them; in the lower portions of the canal frequently; possibly, also, in the tissues of solid organs, as in the pancreas, liver, kidney, muscle, but this is still a disputed question. In the circulating blood of healthy animals they are never found. They enter the system by the absorbents of the mucous membrane of the small intestines, the lacteals, by the pulmonary mucous membrane, by recent wounds, by the uterine surface after delivery. They exist in the animal body without interfering with health. If they meet with a proper soil, such as exudation-liquid, or dead tissue, they set up putrefaction, and, in the process of putrefaction, a poison is produced which, mingling with the blood, poisons it, and gives rise to septicæmia. This poison they carry into the system, but the bacteria themselves are not poisonous.

Thus, then, we must have two factors for the production of the poison: (1) the material capable of putrefaction; and (2) bacteria to effect putrefaction.

Now, how are we to apply this to our subject? It is well known, as Billroth remarks, that in most open wounds, especially in contused wounds, shreds of connective tissue die, and in many spontaneous inflammations the circulation ceases in some parts of the inflamed tissues, and they die. Here, then, we have the material from which the poison is to be elaborated, and the inference is plain that the first step to be taken to avoid this form of puerperal fever is to so conduct the labour as to lessen the amount of injury done, to be careful that

the membranes be not ruptured too soon, and that the head be not allowed to press so long on the soft parts as to injure them, and, above all, that no violence be used.

In the second place, to cut off so far as possible the access of bacteria to the injured parts, by cleanliness, by ventilation, by disinfection; but the experiments of Chauveau and of Kocher, of Berne and of Sanderson, prove that even where all access from without is cut off, organisms will pass from the intestinal canal or other sources to the point where dead or dying tissue exists, and there set up putrefaction and the elaboration of the septic poison, and consequently a third indication remains of equal, if not greater, importance than the others—that is, to remove the products of decomposition as quickly as possible. Cleanliness, the adage says, is next to godliness. Mr. Simon has modified this, and tells us that of all our present removable causes of disease uncleanness is the most deadly; and Burdon Sanderson tells us that the only scientific definition of filth is that it consists of the products of bacterial evolution. If the septic poison be not introduced too quickly, too frequently, or in too large a quantity, the living blood and tissues will decompose and neutralise it. They are the best colytics. All collections of blood and liquids must be prevented. Liston did this by leaving the wounds after his operations exposed to the air for five or six hours, or covered with lint dipped in water, till all oozing had ceased and the surfaces were glazed over, and then brought them into apposition. This was before the days of antiseptic surgery, yet the wounds were not poisoned by atmospheric germs. Lister leaves drainage-tubes in the wound to carry off the fluids as formed. In obstetric practice we must depend on the natural drainage and on frequent washings by the syringe. Billroth says that in ordinary wounds the secretion poured out within the first forty-eight hours is especially active, and that the stinking products of tissue-decomposition are possibly the least noxious. This does not seem to me to hold good of the discharges after delivery, but let it be so or not we must ever bear in mind that uncleanness must be reckoned as the deadliest of our present removable causes of disease.

Organic Substances Retained in the Uterus.—The next group we have to consider is so closely connected with the former that it will not be necessary to dwell on it at any length. Since the days of Hippocrates it has been known that portions of the placenta, or membranes, or clots, retained within the uterus will cause fever. As in the laboratory, infusions made of fragments of organic matter, animal or vegetable, afford the material on which bacteria may work and elaborate septic poison, so will organic substances retained in the uterus afford the conditions for the formation of septic poison, which, when it gets into the system, will set up fever. Harvey, of whom it has been said that if he had not been a great physiologist, he would have been a great obstetrician, washed out the uterus under these circumstances; but it is to Dr. Braxton Hicks we are indebted for formally recommending the practice. The rapidity with which the fever subsides when this is sufficiently done proves that the fever depends on the absorption of the poison.

Phlebitis.—Closely connected with these groups is another set of cases where the disease takes its origin in varicose veins, which inflame, and in which thrombi form, giving rise to emboli and pyæmia, with metastatic abscesses. Bacteria make their way into these thrombi, from within the system, and cause them to disintegrate and break into fragments, which, being carried off in the current of the blood, get arrested at certain points, and give rise to secondary inflammations and metastatic abscesses.

Epidemics.—The foregoing groups belong to the class Dr. Barnes calls autogenetic. Autogenetic they are so far as the primary factor for their production is concerned, but for their development they require the presence of organisms to carry on the manufacture of the poison—that is, the active agent in their production. The cases we have next to consider present a different clinical history, and seem to depend on entirely different causes. Those we have been considering occur only after labour, and have their origin in conditions caused by labour. They occur in isolated cases, are not infectious, and do not spread through whole communities. In the cases we have now to consider, the disease may manifest itself before labour has set in. It seizes great numbers at the same time; it observes cycles of development and decline, sometimes disappearing altogether for long or short periods; at its onset, it proves fatal to almost all who are attacked by it; as it declines, it becomes more mild in its character, and many of those attacked recover. In all these respects, these cases resemble diseases specific in their nature, and dependent for their development on the action of a specific epidemic influence.

The occurrence of fever before labour, which rapidly develops after delivery into puerperal fever of a very fatal character, has been noted and described by many observers. In the following case, all the facts

seem to be so fully verified, as to leave no room for doubt: A lady, mother of several children, residing in a healthy situation, in a large and well kept house, when expecting her confinement, had one of her children under the care of Mr. Wheeler for club-foot. Not feeling very well, she spoke to Mr. Wheeler about herself, and he prescribed a diaphoretic mixture. Next day, Mr. Wheeler, finding the fever increasing, and knowing that she was in hourly expectation of her confinement, advised her to send for her accoucheur. Dr. William Smyly now saw her, April 14th, 1882. He found her skin dry and hot; her pulse 120; her temperature 104° Fahr. The fever continued to increase till the morning of the 15th, when perspiration occurred, and labour set in. The child was born at 1.30 P.M. on the 15th; a healthy child, still living. The fever continued; though the temperature never again reached 103°, the pulse was abnormally and increasingly fast. The lochial discharge became fetid, and the abdomen tympanitic. During the night of the 18th violent diarrhoea set in, with great pain. I saw her the following morning; she had then a very rapid and small pulse; the abdomen was tympanitic; the breasts flaccid; the skin relaxed; and she was gulping up mouthfuls of coffee-ground fluid, and was apparently dying, but rallied a little, and lived till the next day—the sixth after her confinement. There was no history of exposure to any zymotic disease. Her infant was born alive and healthy, and continued healthy. There was no disease in the house or family. The child under treatment for club-foot was wearing a boot, and running about in perfect health. The fever manifested itself before either accoucheur or nurse was in attendance, or labour had set in, and ran its fatal course with all the characters of puerperal fever.

Epidemics of puerperal fever have been so frequently and so fully described, that it is almost unnecessary to do more than refer to them. But there is one epidemic on record whose whole history can be so clearly traced that it is exceptionally worthy of attention. It occurred in the Rotunda Lying-in Hospital in 1854 and 1855, and has been described by the late Dr. M'Clintock¹ with all the faithfulness, accuracy, and vividness of detail characteristic of him. Unfortunately, time will not admit of my giving more than a brief outline of the history. It was the second epidemic Dr. M'Clintock had witnessed in the hospital, the first being in 1845, when he was assistant under Dr. Charles Johnson, and this one in 1854-5, when he was himself Master.

The epidemic unequivocally declared itself in the first week of December 1854, but not unexpectedly, Dr. M'Clintock says, for there had been twelve or fourteen cases of puerperal peritonitis and phlebitis, together with a few isolated cases of scarlatina and typhus, in the house during the preceding nine months.

From the beginning of December to the 14th of February, 182 women were confined. Not half the average, as admissions, except in extreme cases, were stopped in the end of December and all January.

Of the 182, 38, or 1 in 5, were unequivocally affected, and, of the 38, 17 recovered and 21 died, making the proportion of fatalities 1 in 8 of all admitted, more than tenfold the average of the hospital. In 3 of these cases, the puerperal disease was complicated with scarlatina; of these, 2 died. One woman, who was slowly recovering from scarlatina, which came on soon after delivery, was seized with symptoms of peritonitis on the tenth day, under which she rapidly sank.

"It rarely happens," Dr. M'Clintock says, "that puerperal fever breaks out in the hospital without its contemporary appearance in private practice; and it never happens, I believe, that it prevails to any extent outside the hospital without appearing among the patients within its walls. On the late occasion, I had reason to know that several deaths had occurred from the disease among women confined at their own homes, and lacking neither comfort nor attention, before it visited the hospital. Nor, since then, were its ravages confined to the poor inmates of our wards, for many women, among even the upper classes of society, were carried off under its fatal influence. During the months of December and January, no less than twelve of such deaths, in and about Dublin, came to my own knowledge, and I have heard of four or five more in the beginning of last month" (February 1855).

On the outset of the fever in the hospital it displayed uncommon virulence, and the first seven patients who were attacked fell victims to its malignity.

The progress of the epidemic is shown by the number attacked on different successive days. Thus, on each of the following days, namely, the 1st, 3rd, 4th, 6th, 9th, 10th, 11th, and 12th December, there was one woman seized with the disorder, three on the 13th, and three on the 15th, and on no subsequent days of this month were there

more than one, and on many days not one was attacked; but, in February, two were attacked on the 8th, two on the 9th, and two on the 10th.

With respect to the period after delivery at which the patient was seized, the following are the general results: Two were attacked in three hours from the completion of labour, one in four hours, one in twelve, one in fourteen, one in seventeen, and one in twenty-two hours. Each of these seven cases terminated fatally. One was seized in twenty-two hours after delivery, and one in twenty-three; the former recovered and the latter died. Thus, we see, of nine patients in whom the complaint manifested itself on the first day of childbed, eight died; twelve were affected on the second day, six of whom died; ten were attacked on the third day, and of this number the complaint proved fatal to three.

Here, then, is a disease which approaches gradually; which suddenly acquires such force as to make the mortality of an hospital nearly tenfold; which, at its onset, caused the death of all who were attacked by it; which affected the city at large; which manifested itself sometimes within three hours of the patient's delivery; and which gradually exhausted its violence and ceased. Is not this the history of an epidemic? Does it not remind one of the history of cholera, or of smallpox, epidemics of which are also characterised by their gradual approach marked by sporadic cases, their furious onslaught, and their gradual subsidence indicated by diminished violence and the increased number of recoveries?

Epidemic diseases vary as to the mode of their commencement. Generally their advent is heralded by the occurrence of isolated cases at varying intervals. It was thus in the epidemic of 1854-5, of which we have been speaking, and in that of 1826, described by Dr. Collins. In the epidemic of 1845, also described by Dr. M'Clintock, there were no indications of its approach. Its invasion was sudden and unexpected. It sometimes disappears abruptly, as in that described by Dr. Collins: "In the year preceding my appointment as Master," he says, which took place in November 1826, puerperal fever prevailed in the hospital to an alarming extent. In the succeeding year, 1827, the mortality from this disease was slight. Typhus fever was, during these periods, very prevalent in Dublin, and many cases of it appeared in hospital. In 1828, the attack of puerperal fever was much more severe, proving fatal to twenty-one women. It continued to increase in violence considerably in the months of January, February, and March, after which it disappeared, and, for the four remaining years of my Mastership, we did not lose a single patient from this disease." During these four years, 10,785 patients were delivered in the hospital, of whom only 58 died, which is nearly in the proportion of 1 in 186, or 0.54 per cent.—the lowest mortality on record in an equal number of a similar class of patients, as Dr. Collins, writing in 1836, remarks, and now, in 1884, the same remark, I believe, holds good. The mortality of lying-in hospitals may be taken as depending on the extent to which puerperal fever prevails. For short periods, when fever is absent, the average is low; but, when the observation extends over a longer term, the average is equalised. The whole mortality of the Rotunda Hospital, from its opening in 1757 to the close of Dr. Shekleton's Mastership in 1854, nearly one hundred years, was at the rate of 1.21 per cent. As we have just seen, during the four years after the epidemic of 1826, it fell to 0.54; but, during the whole seven years of Dr. Collins's Mastership, it was 0.96. During the seven years of Dr. Evory's Mastership, 1794-1800, it was lower, 0.75; from 1781 to 1786, under Dr. Rock, it was 0.76; and, in the report just published for the year 1883, it was 0.55.

Epidemics of other diseases, small-pox for example, sometimes subside as completely as did that of puerperal fever in 1826; but they more frequently linger on, the epidemic influence getting less and less active, till a new wave approaches and overlaps that which was disappearing. This seems to have been the case in the epidemics of 1845 and 1854. The report of the hospital, published by Johnston and Sinclair, for the seven years preceding Dr. M'Clintock's Mastership, enables us to trace it. Unfortunately, we have no details for 1846; but in 1847, when Dr. Charles Johnson's Mastership terminated, and Dr. Shekleton's began, the disease was still prevalent, or probably there had been a recrudescence of the epidemic. During the months of November and December of that year, with which the report commences, the total mortality was at the rate of 7.02 per cent. In the following year, it fell to 1.81, and continued to fall till 1852, when it reached the minimum of 0.56. The new wave now manifested itself. In 1853, the mortality was 0.88; in the beginning of 1854, it was 1.13; and in the latter months of that year, the epidemic, as we have already seen, was in full force, and the mortality reached 11.53 per cent.

The following tables, calculated from Johnston and Sinclair's

¹ Proceedings of Dublin Obstetrical Society, *Dublin Quarterly Journal of Medical Science*, vol. xix, p. 456.

report, show the total mortality, and the mortality from puerperal fever.

TABLE I.—*Total Mortality.*

1847	Nov. and Dec.	17 in	242 deliveries = 7.02 per cent., or 1 in	14.23
1848	.. 33	1,823	.. 1.81	.. 55.2
1849	.. 37	2,063	.. 1.79	.. 55.7
1850	.. 17	1,982	.. 0.85	.. 114.0
1851	.. 14	2,070	.. 0.67	.. 147.8
1852	.. 11	1,963	.. 0.56	.. 178.0
1853	.. 17	1,926	.. 0.88	.. 113.3
1854	.. 19	1,879	.. 1.13	.. 88.3
Total 1652				

TABLE II.—*Puerperal Fever.*

1847	Nov. and Dec.	10 cases of puerperal fever, 9 died = 90	per cent.
1848	.. 43	.. 23	.. 53.48
1849	.. 29	.. 19	.. 65.55
1850	.. 15	.. 2	.. 13.33
1851	.. 10	.. 5	.. 50
1852	.. 3	.. 2	.. 66
1853	.. 8	.. 5	.. 62.5
1854	.. 11	.. 10	.. 90.9

In Dr. M'Clintock's report of the epidemic of 1854-5 it appears the hospital was closed in the latter part of December and all January. It is not directly so stated, but it would appear that it was opened again in February, but the disease showed itself among the new set of patients—2 were attacked on February 8th, 2 on the 9th, and 2 on the 10th. We had a similar experience in the Coombe Hospital in the early part of 1880. The hospital was completely rebuilt and newly furnished three years before. Fever manifested itself in the early part of the year, and in March the hospital was closed. The whole house was cleaned, the walls and ceilings scraped and whitened, the floors saturated with wax and tuppentine, and everything thoroughly cleaned. It was re-opened in April, every alternate bed being left unoccupied; again the fever showed itself, and again the house was closed. The hospital consists of two buildings, about twenty yards apart from one another. The second building is used for gynæcic cases. The patients were transposed—the gynæcic cases were moved to the labour-wards, and all new labour-cases were taken into the gynæcic wards; but again the fever manifested itself among the labour-cases. The other patients (transferred to what was supposed to be an infected house) remained perfectly healthy. The evidence here seems conclusive that the disease was not the result of "hospitalism."

Another feature of the disease of almost equal importance manifested itself on this occasion, as showing its true nature. As the epidemic subsided, and the patients who were attacked began to recover, they got pelvic cellulitis; and at the same time many cases of the same kind came into the house from among the patients who had been confined at their homes. At first, suppuration and the formation of abscesses was the rule in these cases; but, after a little, the majority got well by resolution. The epidemic had expended its force.

In many of the recorded epidemics, the occurrence of cases of typhus fever and of scarlatina at their beginning is noted, and it might be said that they were epidemics of these diseases, and not puerperal fever at all.

The eighty-nine cases recorded by Dr. Braxton Hicks in his paper on Puerperal Diseases² may lead some to adopt this view; but a careful examination of his cases will show that they prove only that scarlatina and other diseases may, and frequently do, occur in the puerperal state. They occur in a similar manner after surgical operations, and seriously influence the result, but do not prove that puerperal fever is scarlatina, or the converse. If you sow thistles, you will grow thistles; and if you sow scarlatina, the crop will be scarlatina. One case mentioned by Dr. M'Clintock affords evidence, however, of the clearest character that the disease was not scarlatina. "A woman recovering from scarlatina, which attacked her soon after delivery, was seized with symptoms of peritonitis (phrase often used by Dr. M'Clintock as synonymous with puerperal fever) on the tenth day, under which she rapidly sank." It must be admitted that it is in the highest degree improbable that, if the epidemic disease was scarlatina, it would affect a patient in this manner who was only just recovering from the same disease. It is worthy of remark that, though he makes special allusion to the health of the children, and to the absence of trismus and convulsions, which had been of frequent occurrence in the epidemic of 1845, Dr. M'Clintock does not

describe a single case of scarlatina as having occurred among the children of the women attacked by puerperal fever. Nor can it be said the patients died too soon to allow of the appearance of the characteristic eruption. The eruption of scarlatina appears on the second day of the fever. None of the patients died within this time; one died in fifty hours from the period of invasion, one in sixty, and two in seventy-two. These were the most rapid cases. Four or five days was the average time that patients lived after being seized with a fatal attack of the fever.

The foregoing evidence seems to me to prove irresistibly the existence of a specific epidemic influence. Much more might be adduced to the same effect. Dr. Lusk⁴ tells us that, in the year 1871, the mortality from childbed in New York was 399; in 1872, 503; in 1873, 431; in 1874, 439; and in 1875, 420. The excess in the deaths for 1872, he says, was due wholly to an increase in the cases of metria, those from ordinary accidents remaining nearly the same as in the preceding years. The disease certainly did not extend into the city from the hospitals serving as foci, for the mortality at Bellevue Hospital was scarcely more than half the usual average. There was no especial mortality that year from either diphtheria, erysipelas, or scarlatina, but the aggregate mortality was the largest known in the history of the city.

Dr. Fordyce Barker, in the recent discussion at the New York Academy of Medicine, alluded to the great puerperal mortality of 1873, and attributed it to epidemic influence. In five of the best wards of the city, he said, in which are the residences of a great proportion of those of wealth, and few of the class of dwellings known as tenement-houses, with a population of 307,046, there were eighty deaths from puerperal fever, while in the remaining wards of the city, with a population of 605,245, there were but sixty-three deaths—that is, nearly one-third less, in proportion to the population, than in the best parts.

Epidemic disease is defined to be "a disease prevalent among a people or a community at a special time, and produced by some special cause or causes not naturally or generally present in the affected locality, as distinguished from an endemic disease."⁵ The facts now mentioned seem to me to prove that there is a large group of puerperal cases that come strictly within this definition, and that the epidemic thus constituted is a specific disease.

SEPTICÆMIC THEORY.—It is taught, however, by many, that puerperal fever is nothing else but poisoning with septic matter from the genital organs.⁶

This theory seems to me to be defective, inasmuch as it does not embrace the foregoing facts.

As stated by Schroeder, it involves the following propositions:

1. Puerperal fever is caused by the absorption of septic matter from the genital organs. It is nothing but poisoning with septic matter.
2. The infecting matter is found everywhere where organic compounds decompose. It may be derived from dead bodies, suppurating wounds, disintegrating neoplasms, and especially the secretions of diseased and sometimes also of healthy puerperal women.
3. Fresh wounds are necessary for the absorption of septic matter. Wounds in a state of granulation do not absorb, but if the granulations be absorbed, or the surface of the wound broken, then it becomes an absorbing surface.
4. The septic matter is never absorbed through the intact skin or mucous membrane, through the lungs or intestinal canal.
5. The septic matter may be generated within the patient herself—self- or auto-infection; or be introduced from without—external or hetero-infection.
6. Self- or auto-infection is most likely to occur when the wounds are fresh—that is, when, at the birth of the child, there are already decomposed materials, as from a decomposed fetus, gangrene of the soft parts established before delivery, or when new growths, as carcinoma, rapidly undergo decomposition. Retained membranes and placenta are rarely the cause, for by the time they begin to decompose the wounds have ceased to be in an absorbing condition.
7. Hetero-infection, or infection from without, arises from the direct application of septic matter to recent wounds in the genital organs by means of the sponge or linen used in cleaning the parts, by instruments, or by the examining finger. It takes place most frequently through lacerations in the cervix.
8. It may possibly be caused by septic matter floating in the air, but there are no cogent reasons for such an assumption.
9. The definition shows there is nothing specific in puerperal fever. It is the same state which is frequently observed in sur-

² An error of 2, owing to a misprint in the Report. Total mortality from all causes was only 163 = 1.85 per cent.

³ *Transactions of the Obstetrical Society of London*, vol. xii.

⁴ *Science and Art of Midwifery*. London: H. K. Lewis. 1884.

⁵ *New Sydenham Society's Lexicon*.

⁶ *A Manual of Midwifery*, by Karl Schroeder.

gical wards, and designated as erysipelas, pyæmia, ichoræmia, and septicæmia.

Such is the septicæmic theory as to the nature of puerperal fever. Those who hold it, look on the disease as preventable, and assert that its occurrence is due either to ignorance of the precautions necessary to prevent it, or to failure in seeing that such precautions are rigorously carried out. These precautions are of a very elaborate character. They have been very fully set out in a paper read before the New York Academy of Medicine in December last, by Dr. Gaillard Thomas. They are to be put in force in private dwellings of all classes, as well as in hospitals. When a confinement is expected, the patient's room is to be carefully prepared, all curtains and upholstery to be removed, the ceilings and floors are to be washed with a solution of carbolic acid or bichloride of mercury, the bedstead and mattress to be sponged with the same solution. The nurses and physician are to take care that all their clothing is free from septic infection. If they have been exposed to the effluvia of septic diseases, such as typhus or erysipelas, their clothing is to be changed, and their bodies thoroughly sponged with a saturated solution of boracic acid. As labour sets in, the nurse is to wash her hands, remove the dirt from under her nails, and give the patient an antiseptic injection every four hours. The physician is also to wash his hands and attend to his nails; and, after washing, both doctor and nurse are to soak their hands for several minutes in a solution of bichloride of mercury. Labour being complete, the third stage being carefully attended to, the physician is to examine the vulva of the patient; if the perineum have been ruptured, it is to be closed by suture; if slight lacerations have occurred, they are to be dressed with a solution of carbolic acid and persulphate of iron, and painted over with collodion. The patient is to take ergot three times a day for a week, to have the vagina syringed out with an antiseptic lotion every eight hours—twice as often if the labour has been an instrumental or difficult one—and, after each syringing, a pessary of iodoform and cocoa-nut butter is to be introduced into the vagina, the nurse being careful to disinfect her hands before every approach to the genital tract of the woman.

In corroboration of their views, the advocates of the theory appeal to the results which they have obtained by adopting some such mode of preventive treatment as that laid down by Dr. Thomas. But the history of great hospitals, such as that of Dublin or Vienna, shows that puerperal fever will prevail and cause sad mortality at times, and disappear again, and that the conditions governing its movements have not yet been discovered. Dr. Collins's precautions, compared to those recommended by Dr. Thomas, were very simple indeed. "All the beds in the hospital are composed," he says, "of straw, nor is any one used more than a second time without the cover having been washed and the straw renewed. In every instance where the patient dies, this is at once done, and, should the most remote symptom of fever have been present, every article connected with the bedding is instantly scoured and stoved; the woodwork and floor washed with the chloride of lime solution, and the entire ward whitewashed."

To these precautions, along with strict attention to ventilation, he attributes the complete disappearance of puerperal fever, and the fact that his mortality during the last four years of his mastership was the lowest that had ever been recorded. The gentleman who immediately succeeded Dr. Collins in the mastership was his near relative, his pupil and assistant, and, it may be fairly assumed, did not relax his attention to the means that had been so successful with his predecessor; yet we find that the mortality during his mastership was nearly double the total mortality of Dr. Collins's mastership, more than three times that of Dr. Collins's four years' exemption from fever, and the highest recorded in the hospital during the first hundred years of its existence.

The experience of the Vienna hospitals is not less remarkable. "It is familiarly known," Dr. Lusk tells us, "that after Semelweiss had introduced the practice among the physicians attending patients at the large lying-in hospital at Vienna of washing the hands in a solution of chloride of lime, there was a great diminution in the mortality which prevailed, notwithstanding which G. Brann reports, however, that in 1857, in the month of July, in 245 deliveries there were 17 deaths. The following month Professor Klein gave orders to suspend the use of disinfectants. By chance, in August there were only 6 deaths out of 250 confinements; and in September, of 275 patients, none died. From 1857 to 1860, the mortality was slight, though disinfectants were not used; while, during the three following years, in spite of the systematic and persistent employment of those agents, the death-rate once more assumed formidable proportions."

Such experiences are, no doubt, discouraging. The precautions of the present day are, however, much more elaborate than those of Collins

and of Semelweiss; and, let us differ as to our theories as we may, we will all unite in the earnest desire that they may prove not only still more efficient, but more permanent in their effects.

AN ADDRESS

DELIVERED AT THE OPENING OF

THE SECTION OF MEDICINE,

At the Annual Meeting of the British Medical Association, held in Belfast, July and August, 1884,

By JAMES W. T. SMITH, M.D.,

Physician to the Belfast General Hospital; Consulting Physician to the Belfast Lying-in Hospital, and Belfast Children's Hospital.

OLD AND NEW VIEWS IN MEDICAL SCIENCE.

My first duty is to thank the members of the Association for the great honour they have done me in appointing me to preside over the Section of Medicine. I am conscious that my selection is due to the kind feeling of the members, and not to any qualifications of mine for the office. It has been customary for the President to open the Section with a few observations of a preliminary kind, the general addresses already delivered absolving him from the necessity of entering at any length upon the present state of medicine.

Since our last meeting, nothing has occurred, that I am aware of, which requires special notice. The year has been distinguished by no great achievement in the domain of medicine, nor by marked advance in any one direction. But, as in previous years, so has it been in the past one. Some new truths have been established; some doubt confirmed, some fallacy laid bare; some general advance made towards a horizon which is still distant.

When I remember the many distinguished physicians who have preceded me in this chair, I feel I am under a great disadvantage in undertaking to address you, and, in the few observations I propose to make, I intend to touch only on some points which have occurred to me in connection with this meeting.

During the last quarter of a century, our general ideas have materially changed in regard to disease. Many affections which were then looked upon as of the gravest character have now come to be regarded with much less apprehension. It is not so very many years since heart-disease was considered a most dangerous if not inevitably fatal affection. The revelation of a cardiac murmur by the stethoscope was like a sentence of death to the patient, and the facilities which this instrument afforded for detecting these lesions, at a time when their importance was greatly exaggerated, must have revealed to many that they were suffering from a disease, of the existence of which they might otherwise have lived and died in happy ignorance. It may indeed be inferred that the anxiety such knowledge gave rise to must often have been more fatal to the patient than the disease itself; for we have now learned that confirmed valvular disease is far less fatal than was supposed, and that the frequency of sudden death from this lesion has been unduly overrated. In the examination of hospital and other patients, we frequently discover a valvular lesion, which has never given any indications of its presence, and which years after will be found unchanged. It is, in fact, a matter of common observation, that persons affected with such lesions will reach the usual term of human life, unconscious of their ailment. In life-assurance cases, no doubt, healthy lives are annually declined, as it may be assumed that a cardiac murmur would be looked upon as a disqualification. But even acute cases of heart-disease, now that we no longer contend against them with the weapons we formerly used, are found to be far less fatal than was at one time supposed. It is not only in reference to cardiac disease that our opinions have changed; a more matured experience and a more judicious system of treatment have enabled us to entertain a more favourable opinion of rheumatic fever, of pulmonary consumption, and other diseases.

Our views have also changed with regard to many acute diseases. We do not often find nor expect to find acute inflammations occurring in healthy persons. The numerous ailments whose names end in *itis*, are rarely met with in practice as primary diseases, except when supervening upon morbid changes which have been at work often for a long time before. In pericarditis we look for the poison of rheumatic fever or Bright's disease, in peritonitis we do not rest satisfied with attributing the attack to cold, but look for some of those pre-existing lesions which morbid anatomy has taught us to expect. It may be said that acute pulmonary inflammations, the result of exposure, are well marked ex-