The urine was scanty, varying from eleven to twenty-one ounces; it contained two-thirds of albumen and 166.5 grains of urea. The diarrhoea doubtless was the cause of the diminished quantity of urine.

Necropsy, October 4th, 1881. The body was very emaciated. The lower half of the ileum, and the whole of the large intestine, were occupied by extensive ulcers of well-marked tubercular form. The spleen (11 ounces) contained numerous sago-bodies, and some tubercle, as well as lardaceous material. The liver (3 lbs. 7 oz.) was apparently lardaceous. The kidneys (10 ounces) showed a wide cortex, and were markedly lardaceous. The lungs had large cavities at the apices, with foul contents, each cavity being of the size of an orange. The left lung also had a cavity in the axillary region. The lower lobes of both lungs contained caseous tubercle and purulent infiltration. The larynx was ulcerated.

REMARKS.—This was undoubtedly a case of lardaceous disease following on the exhausting discharge of expectoration and diarrhoea. As it came under notice only shortly before death, the observations taken were few; but, as far as they went, they indicated the presence of moderate temperatures where, considering the extensive ulcerative processes, we should have expected high pyrexial ones. The quantity of urine and urea excreted was decidedly small; this may have been due to the profuse diarrhoea.

CASE IX.—James R., aged 27, was admitted May 14th, 1878. He had cough and yellow expectoration, occasionally streaked, fourteen months, caused by exposure to cold and wet, followed by night-sweats, and emaciation. Since November, 1877, he had shown rapid emaciation and loss of strength. His cough was troublesome; expectoration was yellow, and copious. The tongue was furred. In the right chest, tubular sounds and crepitation, were heard in the first interspace. In left chest, undue resonance and cracked-pot sound were heard, with distant cavernous breathing from the clavicle to the fifth rib. Crepitation was audible over the lower third. There was dulness above the scapula.

Temperature-observations were taken nine days after admission, in consequence of rigors coming on and occurring daily, generally about 11 A.M., accompanied by a quick pulse (110 to 120), and hurried respiration. Vomiting occurred generally after breakfast for a week. Later on night-sweats appeared, and the rigors ceased. On June 20th, he complained of pain in the back; slight cedema of the legs was noted, and the urine was examined, and found to contain albumen occupying one-third of the test-tube. The temperature-averages of twenty-seven days were: at 8 A.M. 100.2°; at 11 A.M., 101.6°; at 2 P.M., 101.8°; at 5 P.M., 102°; and at 8 P.M., 102.4°. Maximum, 104.2°; minimum, 98.2°.

The urine varied in quantity 16 ozs. to 44 ozs., containing one-third albumen, and some granular casts. The pains in the back (renal region) continued, the vomiting and shivering having ceased, till June 28th, when they subsided. Unfortunately, the patient quitted the hospital two days later, and was lost sight of.

REMARKS.—This case is included, as the temperature-observations were carried on steadily for twenty-seven days; but the height and range of these were not explained by anything occurring in the lungs, or in any other organ, and as the patient left the hospital and was lost sight of, the cause of the pyrexia was not explained.

CASE X.—John T., aged 30, groom, was admitted December 22nd, 1874. Five years ago, being chilled by sleeping in a damp bed, he occasionally had cough and expectoration since, which had become constant in the last three years. He had severe hæmoptysis twice, each time lasting two weeks. He had lost three stone in weight. Scattered crepitation was audible over both lungs. He had cedema of the feet and ankles. The urine contained a considerable amount of albumen, was of specific gravity 1027, and averaged one and a half pints daily.

Observations on temperature were carried on from January 8th to February 4th, by Mr. H. L. Bernays. During five days, diarrhoea had prevailed. The following were the averages of nineteen days: at 8 A.M., 98.1°; at 11 A.M., 98.6°; at 2 P.M., 99.4°; at 5 P.M., 100°; at 8 P.M., 100.2°; maximum, 101.6°; minimum, 96°.

The patient became more and more dropsical, there being general cedema and effusion into the right pleura, and he sank on March 3rd.

The **necropsy** on the following day showed general anasarca. The right lung contained a large cavity at the apex, and tubercle scattered through the upper lobe. The left pleura contained a pint of serous fluid; the left lung showed a small cavity at the apex, and tubercle scattered through the lower lobe. Both cavities were of old date, and had cartilaginous linings. The right cavities of the heart were dilated. The kidneys were large, mottled, and their cortical structure was much increased.

CASE XI.—J. M. W., aged 28, was admitted in February 1875, with a history of phthisis, of two years' standing, and of diarrhoea of some months' duration. Cavernous sounds were audible over the greater portion of both lungs, with crepitation at the base of the left lung. The urine (in quantity forty-four ounces), loaded with albumen, contained a large number of casts, epithelial, hyaline, and granular, with much loose epithelium. There was considerable cedema. The patient gradually sank, and died on March 2nd, twenty days after admission. During the last five days of his life, Mr. Bernays took temperature-observations, which gave the subjoined results as the average: at 8 A.M., 100.6°; at 11 A.M., 99.3°; at 5 P.M., 101°; at 8 P.M., 101.8°; maximum, 103.8°; minimum, 94°.

The **necropsy** showed both lungs to contain large thin-walled cavities, with scattered tubercle in the lower lobes. The liver was pale and large. The kidneys were very pale; their cortical structure was greatly increased; the capsules peeled easily (chronic nephritis).

REMARKS.—The shortness of this case precludes any decided deductions being made from it, the temperature being only taken for five days. The course was a very fitful one, such as often accompanies the termination of destructive lung-disease. Unfortunately, no account is given of the kidneys being tested with iodine, and we must accordingly accept them as instances of chronic nephritis though the description is more that of lardaceous.

[To be continued.]

THE ÆTIOLOGY AND PATHOLOGY OF SPORADIC PUERPERAL SEPTICÆMIA.¹

By WILLIAM STRANGE, M.D.,

Senior Physician to the Worcester Infirmary.

In the year 1878, I published in the columns of the *Lancet* two papers on the subject of puerperal septicæmia. The principal point, amongst others, aimed at in those papers, was to establish the entire independence of true puerperal fever of the contagion of the specific zymotic diseases, such as scarlet fever, measles, small-pox, etc.; believing, as I then did, and do now, that the specific zymotic diseases, such as those above named, always breed true; and that, therefore, although the virus of any of those diseases may be introduced to a parturient woman, the disease which results will not be true puerperal septicæmia, but modified scarlet fever, measles, or small-pox, as the case may be.

This is a point in the etiology of puerperal septicæmia which is, however, still much mooted, and, by many, considered to be still *sub judice*; and, as such, it will fall to be considered in its proper place.

Since the publication of my former papers, it has fallen to my lot to be called upon to attend, and to attend regularly, in consultation, many fresh cases of this dire disorder, than which no disease to which flesh is heir spreads greater dismay among our patients and their friends, causes more anguish to the medical attendant, or extinguishes more hopes.

I do not propose to say anything in regard of that horrible form of contagious or endemic puerperal fever which, more often formerly than at the present day, decimated the parturient women gathered together in our lying-in hospitals. Also, I discard from present consideration those cases which are primarily due to an inflammatory process, set up, after parturition, in the peritoneum, or in the uterus, or in some of its appendages. The causes of these are well known, and their course is pretty definite, and they have little or no true pathological analogy with such cases as those with which we are now concerned.

By sporadic puerperal septicæmia I mean a form of disease, cases of which every now and then arise to perplex and astound the medical attendant, as frequently amongst the healthy and well-to-do as amongst the poorer and more sickly classes of the community, as well in the palace as in the cottage, in the country mansion as in the squalid court or alley. Neither can air or drainage be subjected to suspicion in many cases, nor can fatness or poverty, or anxiety of mind. Our text-books inform us that puerperal fever often seizes upon those unfortunate women who, at the time of childbirth, are under a cloud, whether from poverty, sickness, or mental anxiety. Yet the healthiest, happiest homes are not proof against the invasion of this horrible destroyer, as most of my own cases would show.

¹ Read at a joint meeting of the Worcestershire and Herefordshire and Gloucestershire Branches.

It is these facts which invest the etiology of this disease with a special difficulty; and, as we find in many other cases, it will be necessary to acquire a correct idea of its pathology, to be used, *pari passu*, in elucidating its genesis or etiology.

Again, the course and duration of such cases as I have seen, disagree with the text-books in several important particulars. In the text-books we learn that more or less of the inflammatory element is generally mixed up with the septicæmic process; whereas, in my experience, it is generally absent. The duration of the disease, when fatal, is said to be from a week to ten days, or even less; whilst I have generally found it running on to three, four, or seven weeks, and, in one case, to ten weeks, the fever continuing unsubdued all the time.

As might be expected, we get little aid from the morbid anatomy, or dead pathology, of puerperal septicæmia. Let us see if the living pathology will prove more helpful to us in elucidating its etiology. From the thirty or forty cases of sporadic puerperal septicæmia which of late years have fallen under my notice, I may summarise the following average of symptoms. From the second to the fifth day, or, very occasionally, later, after delivery, fever arises, and the temperature continues abnormally high throughout the whole course of the disease; for when it has become normal, and remains so for a few days, the disease, as far as the sepsis is concerned, is at an end. The temperature ranges from 100° to 103° , or 104° , slightly higher in the evening than in morning, but not regularly so. When death is impending, the temperature may run up to 106° or 107° . The pulse keeps pace with the temperature, generally exceeding 100 and going up to 120, and, when a fatal issue is impending, to 130, 140, or even to 150. There are morning remissions in the pulse as in the temperature. The next thing that happens is the suppression of milk and lochia. This may be sudden and complete, as in the more severe cases, or it may take place gradually in four or five days. This suppression of the discharges does not bear so heavily on the prognosis as do the state of the pulse and of the temperature. In some cases, and in some cases only, there is more or less of diarrhœa, sometimes in the earlier days of the illness, sometimes in the later. It is always of unfavourable augury.

The urine is generally high coloured and scanty at first, but soon becomes plentiful; and, except for a little albumen in some cases, fairly healthy. The appetite may be disturbed, with or without vomiting, for a few days at the beginning; but soon the power of digestion returns, and then, during the whole term of the illness, the patient is able to take an abundance of liquid, and sometimes of solid nourishment. The sleep is generally somewhat disturbed, but delirium is not frequent or violent.

Palpation of the abdomen scarcely gives pain unless the uterus be hardly pressed, and there is in most cases but little tympanites, at least during the early part of the illness. This symptom is much more marked in purely inflammatory cases. When death threatens, it nearly always becomes considerable. In a large proportion of cases there are remissions of all the symptoms about every six or seven days, when the temperature may fall nearly or quite to the normal; and if the pulse correspond, this remission may give rise to a hope, too often delusive, that the case is about to terminate favourably. As the disease proceeds towards its usual fatal termination, the temperature and pulse rise again, the respiration becomes hurried by the slightest exertion, the second sound of the heart being preternaturally sharp and dirotous. The tongue, which has been preternaturally red throughout, owing to the loss of its epithelium, becomes dry, baked, and brown.

Now it is, that is, about the end of the second or third week, that in some cases, not in all, red spots and streaks make their appearance upon the wrist, elbow, or ankle-joints, or along the course of the long bones, especially of the lower extremities. Sometimes superficial abscesses form, and suppurate; or deeper effusion into the same joints may occur. Along with this pyæmic condition, the skin, previously very pale, assumes a dusky tint, and the prostration becomes extreme. The lungs and other parenchymatous viscera may now become the seat of pyæmic deposits, or of emboli, and a low form of pneumonia or of jaundice may quickly carry off the patient. In some cases, there is also effusion into the cellular tissue of the fleshy portions of the arms, thighs, or buttocks.

Death takes place at a period varying from ten days to three or four weeks. In one remarkable case, the patient succumbed only after 70 days, ten weeks, of uninterrupted septic fever. The death, in my cases, has been owing to one of three causes, viz., asthenia, or exhaustion; secondly, pyæmic deposits in some of the vital organs; thirdly, thrombosis of the heart and pulmonary artery.

. This is the living pathology, or clinical history, which, with slight

variations in each case, I have now observed in from thirty to forty cases of puerperal septicæmia. What other morbid process does it resemble? Surgical pyæmia, septic lymphatic poisoning, typhoid inflammation, or any of the specific zymotic diseases? I think it resembles none of them. In the pyæmia of our surgical wards, the tendency to form pus is more marked, and ensues at an earlier period. The abscesses are larger, and the course of the disease is shorter. "Pyæmia," Dr. Burdon Sanderson says, "is a malignant process which goes on to its fatal end, and has always a connection with some local morbid process." Septic lymphatic poisoning runs a course more nearly resembling that of surgical pyæmia than that of puerperal septicæmia. Then, of inflammation, whether of an active or typhoid type, there is often no trace at all. Of the poison of the specific diseases, I shall speak presently when I come to the question of infection.

Now, in the second place, as to the sources of infection, and the kind of germ or other pathogenic matter causing it.

I have lately seen it stated that the most useful classification of cases of this disease is into the autogenetic and the heterogenetic, the self-infective and the imported case. But I fail to see that this division can be of any use to us, unless it be to remind us that cases may owe their beginning to either of these sources. For the question is, Do those cases, in which the patient is supposed to have infected herself, the autogenetic, differ essentially from those in which the poison has been brought to her from without, the heterogenetic? If not, this classification does not help us at all. If the poison which is brought to the patient produces exactly the same train of morbid processes as that which, we think, arises in the woman's own system, there can scarcely be any specific difference in the *materies morbi* in the two cases. And, to my mind, there is no question that this is so. Indeed, a doubt has often arisen in my mind, when called to one of these sad cases, whether we are not too ready to conclude that the poison is autogenetic, i.e., generated in the patient's own person, in order to cover some uncomfortable feeling of neglect on the part of some person, or as a cloak to our own ignorance. Strict inquiry will too often reveal the fact that the accoucheur has been lately making a *post mortem* examination, or has just come from the surgical wards of a hospital, without changing his clothes; or that the nurse or the medical man has just been attending a bad midwifery case; so that the suspicion necessarily arises whether a poison has not in some way been introduced from without, although we cannot quite make out in what way. If autogenesis were so easy, how is it that puerperal septicæmia is happily so rare a disease, where all the circumstances favouring such a mode of infection abound in the patient's own person? It is so rare a disease, that many busy accoucheurs go through life without meeting with a single case of it.

It is clearly impossible to go into the germ-theory of disease here. We cannot even discuss the question as to the various actions of the different kinds of germs, whether they be bacteria, micrococci, coccobacteria, or other kind of *hoc genus omne*. Those who have studied the conclusions to which Robert Koch has come from his own experiments, and from those of others lately published by the New Sydenham Society, must be satisfied that much remains for future experiment, and experiment of the most cautious and careful kind, before we shall be able to apportion to each form of bacillus, or micrococcus, its specific range of pathogenesis.

That the same germ-forms are generally found in the same diseased processes seems established; but, at the same time, these forms are not unmixed with other forms which, in different circumstances, produce distinctly other effects.

One thing seems to be clearly established; viz., that these organisms, in one or more of their forms, abound in the mucous lining of the passages in puerperal women affected with septicæmia, and that they are soluble in it; or, at least, by means of the fluids existing there, operate with facility. And we may safely conclude that in most cases, either in the hands or in the clothes of the accoucheur, or of the nurse, also by open windows in foul neighbourhoods, by the coming in and out of various individuals, not always of the cleanest, various, nay, every kind of septic germ, be they specific or non-specific, may get an introduction to the persons of lying-in women. And, if we adopt the commonly received theory that these germs are harmless until they meet with a suitable nidus for their development, are planted, in fact, in a soil in which they can grow and increase, such as the abraded surfaces of the genital passages, we may readily account for the symptoms of puerperal septicæmia; although we shall be as far off as ever from a solution of the questions as to what kind of germs they are which give origin to the disease, and what is the kind of soil they require; that is,

what is the exact morbid condition of the fluids of the woman's organs, upon which, when sown, these germs are found to thrive so well. Here, I think, you will agree with me, is work for the cultivator of germs.

Dr. Burdon Sanderson says that the poison of puerperal septicæmia, unlike that of pyæmia, does not multiply itself in the system. If this be so, how is it that a person may convey the disease from one lying-in woman to another without having introduced his hand into the passages of either? Surely the *materies morbi*, like that of scarlet fever, measles, etc., can be conveyed in clothing! If so, it must multiply in the woman's system, and be given off from her person, by her breath, her skin, or discharges. And here it is that an ounce of experience is worth a pound of theory or conjecture. I have seen a case where it was not possible to lay hold of any cause of the disease, except that the medical attendants had, a few days before, been attending a case of scarlet fever. Yet this patient never had a sign of scarlet fever, but died, after a month, of pure pyæmic septicæmia. In another case, which was thought to be owing to the state of the drains, the attendant had taken part in a nasty *post mortem* examination a few hours before being called to the confinement. In a third, no external source could be made out, and the case was decided to be autogenetic; but the medical attendant carried the infection from this case to another woman, who immediately took on the same disease, attended by the very same symptoms. In a fourth case, there was no parturition at all; but two surgeons introduced their hands into the unimpregnated uterus to remove a small growth, and the patient died within a week of acute septicæmia, in no way differing from such cases in lying-in women.

Now, as to the case of the specific zymotic diseases. We are told in the text-books that the poison of these, conveyed to a lying-in woman, may give rise to puerperal septicæmia. Is this the fact? In the case of each of the truly specific zymotic diseases, we are accustomed to find that each specific germ breeds true. Small-pox does not become scarlatina, nor measles typhoid fever. And, conversely, the septicæmic virus from a puerperal woman may give the same disease to another woman similarly situated; but we never find it setting up scarlet fever or typhoid in any one who may have come into contact with her. Suppose that a woman, just prior to her confinement, has been exposed to the contagion of some zymotic disease, What is it that we observe? A few hours or days after delivery, she becomes terribly ill; there are a hot skin, preceded by rigors, rapid pulse and respiration, with a tendency, in most cases, to speedy death. But some of the specific signs of zymotic disease will not fail to appear: dusky red skin and throat if it be scarlet fever, dusky papules if it be small-pox, plum-coloured blotches if it be measles, and so on. The specific disease will be modified, and, in most cases, intensified; but you will have no difficulty in recognising what it is, and you cannot mistake it for true puerperal septicæmia, such as I have now described.

In a word, is it not in the experience of us all that the germs of the truly specific diseases always breed true? There is, therefore, no analogy, except of the very loosest kind, between the symptoms, or living pathology, of the ordinary specific zymotic diseases, and those of true septicæmia, as it affects parturient women.

Whether disease-germs be all specific in character, or the contrary, cannot yet be decided. If we be guided by the phenomena of the specific contagious diseases, small-pox, scarlatina, measles, etc., we should say that they are. But if we look at the morbid processes in the surgical wards of our hospitals, we cannot believe them to be so; for we find that the poison of various diseases—erysipelas, scarlet fever, diphtheria, etc.—will excite pyæmic mischief amongst our wounded patients. The experiments of Pasteur, Koch, and others, also appear to me to support the latter view.

Turning to the other element, we say that it must be the condition of the *nidus*, of the bed to which the micro-organisms gain access, that determines the form of the disease; or else that the germ carries with it its own specific virus, and so determines the kind of septic fermentation set up. If we adopt the first view, then we must conclude that a woman gets septicæmia because she has been delivered of a child; the condition of her discharges, and of the part, determining the form of disease, whatever germs be applied. If we prefer the second, we ought to acknowledge as many forms of puerperal septicæmia as there are forms or species of germs which can gain access to her person. In favour of the first view, the researches of Nägeli and others tend to the conclusion that germs do acquire their particulate septic character by cultivation in albuminous fluids, and it is also found that the properties so acquired are capable of indefinite extension in the same form.

In the present state of our ignorance of the intimate character of various disease-carrying germs, we must be content to interpret facts by such light as we have. What I contend for is this—that sporadic puerperal, and sometimes non-puerperal, septicæmia, such as, in the cases I have described, attacks women in the best possible circumstances as to previous health and perfect sanitary surroundings. Also, I contend that the fact of the germs of the ordinary zymotic diseases getting to them, and inducing puerperal septicæmia, is not proven, and, in many cases, cannot possibly be entertained. Moreover, I contend that it is more in accordance with our experience, and with such knowledge as we possess of the action of disease-carrying germs, to conclude that, in most cases, a *materies morbi*, to call it by no more specific name, has been brought to the patient from another lying-in room, or from the wards of a hospital, or from a *post mortem* examination, or from some crowded assembly of human beings, or from some other unsanitary source, than that the disease has been set up *ab origine* in the woman's own person. The many known sources of a heterogenesis may well overwhelm the hidden and doubtful causes of autogenesis.

We may conclude, I think, from these propositions, that each specific germ will set up the same specific disease in a parturient as in a non-parturient person; and that non-specific germs—pathogenetic or zymotic matter—will give rise to such a form of disease as the particular condition of the patient is prone to; whether it be to puerperal septicæmia, to erysipelas, to typhoid fever, or diphtheria, all of which diseases I believe may be generated by septic matters, not yet rendered specific and particulate by cultivation in albuminous fluids. For recent experiments in the cultivation of germs would lead us to conclude that a simple zymotic *materies morbi* may, by successive cultivation in the bodies of patients, become, after a time, specific and particulate. And, again, such specialised germs may be afterwards diluted and toned down until they are able to produce only an ordinary non-specific zymosis, or blood-poisoning.

Those of you who listened to, or read, the very interesting Address in Pathology by Dr. Creighton, at Liverpool, will have noticed this idea developed at much greater length, and with all the author's acuteness of reasoning. If I am not mistaken, that address contains the germs of a large extension of our knowledge as to the origin of specific, pseudo-specific, and contagious diseases.

If you ask anything about treatment, I can say only that the almost sole indication is to keep the patient alive, in the hope that nature will eventually gain the victory over the destroyer. Along with attention to the state of the stomach and bowels, put your whole trust in quinine and iron in large quantities, with a sufficiency of nourishment and of stimulants, and persevere with them to the end. Never be led away by such mild affairs as Warburg's tincture, or tincture of aconite.

Since the above remarks were written, the Collective Investigation Committee have sent out a request for answers to certain preliminary questions on puerperal fevers. As I was one of the first to recommend this inquiry to the Committee, I cannot but rejoice that they have taken up such an important and interesting subject; but much fuller data are required than any that can be noted in this paper now issued.

BEQUESTS AND DONATIONS.—Mr. Thomas Mackrell, of Newton Abbot, has bequeathed £2,000 to the North Devon Infirmary, and £200 to the North Devon Dispensary, both at Barnstaple, his native place. —Mr. John K. Ford, of Portsmouth, bequeathed £2,000 to the Royal Portsmouth, Portsea, and Gosport Hospital, and a Southsea gentleman has given a similar amount.—The last quarterly report of the Hospital for Consumption and Diseases of the Chest announced bequests of £500 under the will of Miss Hayes, £500 under that of Miss Romney, £200 under that of Mr. Henry Couchman, £200 under that of Mrs. Perry, £100 under that of Miss Barlow, and £100 under that of Mrs. Maggs.—The Salisbury Infirmary has received £459 15s. 6d. under the will of Mrs. Fowler.—University College Hospital has received £80 further, making £400 for 1883, from the Peoples' Contribution Fund.—The North-West London Hospital has received £300 for the endowment of a bed in memory of A. D. K., St. Moritz.—The Halifax Infirmary has received £100 under the will of Mrs. Jane Stott, of Greetland.—The Grocers' Company have given £100 to the Dental Hospital of London, and £50 to the Royal Hospital for Children and Women.—The Goldsmiths' Company have given £100 to the Charing Cross Hospital.—Sir Erasmus Wilson has given £100 to the Dental Hospital of London.—Mr. S. G. Buxton has given £100 to the Norfolk and Norwich Hospital.