

REMARKS

ON

PUERPERAL FEVER

BEFORE THE
LIBRARY

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BY

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(Phonographically reported by Dr. Geo. F. SHRADY, of the New York Hospital.)

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REMARKS BY DR. BARKER.

The regular meeting of this body was held October 7th, the President, Dr. VALENTINE MOTT, in the Chair. A continuation of the discussion on Puerperal Fever having been made, by previous vote of the Academy, the special order of this meeting, a large concourse of the Fellows was brought together.

Prof. Barker, being called upon by the President to continue the discussion, rose and said :

MR. PRESIDENT : I should feel some hesitation in offering a few remarks on puerperal fever, after the subject has been discussed for three evenings, by gentlemen whose character, position, and experience, carry with their expression of opinion the greatest weight and authority, were it not for the importance and difficulty of the subject, the opposite opinions which have been entertained by distinguished practitioners in regard to it, its liability to occur in the practice of every physician, and its terrible fatality. It is a disease which cannot be investigated by the study of a few sporadic cases, or of a single epidemic, or of different epidemics in the same locality. Neither is it a disease to be studied in the dead-house, for valuable as are the researches which have been made into the minute anatomy of this affection, important as it is, for the complete elucidation of the subject, that everything should be known as to the autopsic lesions which are to be found ; yet their variety in different epidemics, the absence of everything like constancy or uniformity in these autopsic lesions, the frequent want of everything like correspondence between the severity of the symptoms during life, and the amount of the morbid appearances found after death, prove, as I think, that these lesions should be regarded as results of the disease, but not as the

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disease. And it seems to me that there is some danger of our giving undue prominence to these autopsic lesions. We might as well seek to find out the cause of the fire, in a minute chemical and microscopic examination of the ashes of a conflagration, as rely upon the appearances found after death, to determine the character of a disease which results from the absorption of a morbid poison. But is this such a disease? That is the grand question; and it can be answered correctly, not by studying the disease from one stand-point alone, but by a comprehensive examination of all that has been found out as to its phenomena and laws, in different epidemics and varied localities, as recorded by different observers. Naturally enough, the opinions, especially the scientific opinions of men, have for a basis what they have seen and observed for themselves, and such opinions justly carry with them the most weight. Hence, in puerperal fever, we have had the finest minds and the brightest geniuses in medicine, as exponents of exclusive, special, restrictive views as to its pathology and its therapeutics, their ideas resting entirely on the peculiar type of the disease which they have seen for themselves. The character of this disease, bringing death and desolation into those families which have just exulted in the joy and gladness of a new birth, the medium of infection being sometimes the physician, whose steps are regarded, and should ever prove as those of a ministering angel of comfort and hope; its sudden onslaught, its speedy results, and its terrible fatality, all have combined to bring out the highest talent, where talent has before lain dormant in the ordinary routine of practice, and has contributed to give us a richer literature on this subject, than is to be found of any other disease. This statement will surprise some whose attention has not been specially called to this subject, but I believe it will bear close scrutiny. In this day of progressive medicine, in our anxiety to bring it up to the perfection of a physical science, it is possible that in our search after what is new and original, we may overlook what is old and proven, and thus that erroneous deductions may be drawn from the limited experience and observation of a few, which would have been corrected, had advantage been taken of the enlarged experience of the many. We have greatly the advantage of our predecessors in studying this disease. With all the acumen, and careful observation, and extensive experience of Gordon, and Armstrong, and Collins, and Gooch, and Ferguson, they had but a limited knowledge of its pathology and therapeutics, compared with what we may have, who can bring together the aggregate results of all their labors, without assuming to place ourselves

on the same intellectual level with them. What is puerperal fever? Is it a local phlegmasia? It was believed to be inflammation of the uterus by Mauriceau, Astruc, Denman, and others—to be inflammation of the omentum and intestines, by Hulme, Leake, and others. It was regarded as peritonitis by Gordon, Hey, Armstrong, Mackintosh, and Collins—to be inflammation of the veins and lymphatics, by Dance, Duplay, and others. Prof. Meigs, more comprehensive in his pathology than the authors I have named, considers puerperal fever as metritis, metrophlebitis, peritonitis, or ovaritis, or two or more of these phlegmasiæ combined. He distinctly avows as the object of his work, “to prove that it is a simple state of inflammation in certain tissues of pregnant women, and of women lately confined, and that the fever that attends it is a natural effect of intense constitutional irritation from the local disorders.”

In the discussion before the Academy, I think I am not wrong in asserting that we have had presented inferentially, if not directly, two entirely distinct, not to say opposite views, as to the essential character of this disease. Prof. Smith, in his very able and complete paper on its causes and modes of propagation, very plainly announces his belief that it is an idiopathic fever. In the interesting and valuable contributions to its morbid anatomy, made by my friend and colleague, Prof. Clark, although he has not definitely expressed his opinion as to the pathology of the disease, yet I think the legitimate deduction from what he has said must be, that it is a local phlegmasia. He says, “Puerperal fever has four principal lesions, and many of a secondary character; inflammation of the peritoneum, inflammation of the veins of the body of the uterus, inflammation of the inner surface of the uterus, or endo-metritis.” He expresses his belief that, in every case, one of these lesions will be found. He distinctly asserts that these lesions are inflammatory. He also states his conviction, “that those cases described by Gooch, Locock, Simpson, and others, as without lesion, were cases of pyæmia, and that the pyæmia has its source in the inflammation of the inner surface of the uterus, and the facility with which the uterine sinuses could convey the pus into the system was shown.”

Without stopping at the present moment to discuss the question, whether pus, the product of simple ordinary inflammation, if absorbed or washed into the circulating blood, will produce the symptoms which we call pyæmia, or whether, in fact, another element besides laudable pus is not essential for the production of these phenomena, it is sufficient for my present purpose to remark that *inflammation* is

claimed distinctly to be the essential characteristic. In other subsequent remarks, Prof. Clark declares that "the *primary* lesions are in the organs of generation, the *secondary* are in the blood, and are found, indeed, in almost every organ of the body." It seems to me that this is equivalent to a distinct avowal of the doctrine of local phlegmasia. The whole question of contagion must turn upon this point, for if it can be established that puerperal fever is simply a local phlegmasia, modified only by the puerperal state, I think we shall be compelled to give up the doctrine of contagion. Besides, the pathology of the disease must form the basis on which to rest all intelligent discussion of its treatment. As preliminary to an expression of views in regard to the pathology of the disease now under consideration, I may remark, that important as is pathological anatomy, now so zealously cultivated, its prominence in the present age has resulted in some evils. It has led to exclusive *solidism* in medical doctrines, as is strikingly manifest in the opinions held by many in regard to other of the zymotic diseases, as typhus fever and cholera, as well as puerperal fever.

It seems to me a very important question to determine whether, in puerperal fever, the *primary* lesions are in the organs of generation, and the *secondary* are in the blood, or whether the order of phenomena is not reversed! This involves the whole question in discussion. It is only asking, in other terms, whether puerperal fever be a local phlegmasia or a zymotic disease. In as brief terms as may be consistent with clearness, I will assign my reasons for believing that puerperal fever is a zymotic disease, having an essentiality altogether distinct from inflammation of any tissue or structure of the body, even in a puerperal woman. Here I wish to remark that the puerperal state, *per se*, cannot alter, in any sense, the laws which govern inflammation. It may, and undoubtedly does increase, under certain circumstances, the susceptibility to inflammatory action, but it may, and often does, produce a condition of the system directly antagonistic to inflammation,—such a condition as must notably decrease the tendency to inflammation; and it is precisely in this latter condition that we find the most virulent, the most intractable forms of puerperal fever. What do we mean by the puerperal state? It can only mean the physiological and pathological organic changes induced by gestation, the organic changes produced by parturition, and the physiological changes which follow it. Now there are nothing in these changes which can essentially change the laws of inflammation, when we have inflammation alone. Occurring under

these conditions, I think I shall subsequently show that it does not materially differ from inflammation occurring in the non-*puerperal* state. Now, in giving my reasons for believing that *puerperal* fever has an essentiality, that it is something entirely distinct from local *phlegmasiæ*, it will be seen that I adopt a line of argument parallel with that followed by Dr. Stokes in his masterly lectures on typhus fever.

First, then, *puerperal fever has no anatomical character*. That there are a great variety of structural lesions found, all will admit. The four principal lesions are, as Dr. Clark has said, those of the peritoneum, of the veins of the body of the uterus, of the lymphatics, of the inner surface of the uterus. And then we have lesions of the pleura, of the lungs, and pus in the liver, in the muscles, in the joints, and pus in the blood. But where we have a group of symptoms so resembling each other that they are almost identical, we do not have constant or uniform structural lesion. They are inconstant in their seat and their amount. In the same epidemic we have the greatest possible variety in their seat and their amount. Lesion of the peritoneum may be present or absent,—so of the uterus, so of the lymphatics, and so of the veins. In those cases, which Osiander, Gooch, Locock, and Simpson, suppose to be cases of *puerperal* fever without lesion, conceding the correctness of Prof. Clark's view, that with a proper examination pus might have been found in the veins of the uterus,—that they really were cases of *pyæmia*, what does this prove? In legal parlance, I should put in a demurrer. Admit the fact, will any one claim that all cases of *puerperal* fever are cases of *pyæmia*, and that inflammation of the inner surface of the uterus, or of its veins, is an essential anatomical characteristic of *puerperal* fever?

2. *These lesions are often not sufficient to influence the progress of the disease, or to explain the cause of death*. The most malignant form of the disease, that in which a fatal result occurs the most speedily, offers the fewest and the least striking structural lesions. The longer the disease continues, the more prominent and the more manifest are the organic lesions. Does not this prove that the lesions are consecutive or secondary,—that there is a primitive source, an original cause of vital depression, which sometimes destroys life so rapidly that there is no time for the development of the secondary morbid alteration. The symptoms are not, then, the result of these lesions, but the result of some specific agent—some morbid poison, which subsequently develops the autopsic lesions. Sometimes this

morbid poison so overwhelms the system, that the patient dies in a few hours, without any reâctional symptom. It is not in these cases that we have the most marked structural lesions.

3. *We may have inflammation, even to an intense degree, of any of the organs in a puerperal woman, in which the principal lesions of puerperal fever are found, and yet the disease will lack some of the essential characteristics of puerperal fever.* I mean to say, we may have peritonitis, or phlebitis, or metritis, in the lying-in woman, and yet the disease will be quite distinct in its mode of attack, in its symptoms, in its morbid anatomy, and in its treatment, from puerperal fever. Take peritonitis, for example : it may be excited by a difficult and protracted labor, by the application of cold to prevent hæmorrhage, by improper exposure, and by other well known exciting causes. But puerperal fever, with the peritoneal lesion, may attack the patient after most favorable delivery, and without any obvious cause. Then the symptoms of the disease show that it has a special character, for in the puerperal fever, with the peritoneal lesion, the symptoms of the first stage of peritonitis are absent ; the peritoneal symptoms are those of the second stage, or that of collapse, as for example, we have diarrhœa very frequently instead of obstinate constipation. The pain in peritonitis commences in the region of the uterus. In puerperal fever the pain often commences at the epigastrium. In peritonitis the pulse corresponds in character with the local symptoms, increasing in frequency as the local symptoms increase, diminishing as they disappear. In puerperal fever the pulse bears no relation, or at least a very slight one, to the local symptoms. In puerperal fever it is the pulse which tells the story, as to the exact condition of the patient, not the local symptoms. So also we may have phlebitis in the puerperal woman, and not have puerperal fever. No one, at the present day, would use the term phlegmasia alba dolens, and puerperal fever, as synonymous. Yet this is a phlebitis, a circumscribed, adhesive inflammation of the vein, to be sure, but nevertheless a phlebitis. But I go farther : we may have uterine phlebitis, and not have puerperal fever. The same contrast could be drawn between the two, as regards the mode of attack, symptoms, and treatment—the difference being, that the one disease follows the laws of ordinary inflammation, and that in the other the toxæmic origin of the disease gives it quite a different character. These differences were strikingly illustrated in the recent epidemic at Bellevue Hospital. In the beginning of the epidemic I had charge of the lying-in wards. I must ask permission to read a short extract

from the April number of the *AMERICAN MEDICAL MONTHLY*, giving some clinical remarks of my own, made at Bellevue Hospital on the 7th of February last :

“ In the latter part of January, succeeding a period of almost unparalleled cold, came that long spell of warm, damp, close, foggy weather. This change had scarcely set in, when one after another, as the women were delivered—these wards having been previously healthy—they began to develop, one pelvic cellulitis, another peritonitis, another ovaritis, another metritis, all of the asthenic type, and with an early tendency to gangrene or suppuration, while scarce one escaped without a threatening at least, of those terrible torments of nursing women, sore nipples or mammary abscess. Indeed so well established did this state of things become, that a pulse of one hundred and twenty and a flushed cheek were looked for as matters of course on the morning after confinement, and the pleasant, soft pulse, and cool skin of the physiological recovery, were luxuries which the attendant physician dwelt lovingly and long upon, when at long intervals they presented themselves. These cases, notwithstanding that they bore the outward semblance of inflammations, were yet, in their mode of progression, constitutional effects and indications for treatment so different from the ordinary phlegmasia as to lead Dr. Barker to announce his belief in the specific character of these diseases; that the quasi inflammatory processes taken on by these organs were, in reality, the results of the action of a poison infused into them through the blood, and stirring up its peculiar excitement wherever it found the proper amount of combined irritation and exhaustion to insure it a nidus, just as the typhoid poison awakens its deceptive pseudo-inflammations in the brain, the lungs, the intestines. ‘Treat these cases,’ said he, ‘as idiopathic inflammations, and you must inevitably kill your patients.’ Most of these cases were treated successfully, by early local derivation or depletion, followed or even accompanied by profuse general stimulation. Three, however, terminated fatally, two by suppuration into the pelvic cavity and purulent absorption, in one of which a large number of abscesses, from the size of a walnut down, were found in the lungs—and one by gangrene of the cervix, extending to the mucous membrane of the body, and involving to a slight extent, the posterior walls.”

The next point, to which I wish to call attention, is *that the lesions themselves differ materially from those having an inflammatory origin*. Professor Murphy has so clearly pointed out these

distinctions that no apology is necessary for quoting them :—

“ In *peritonitis* all the arterial capillaries are highly injected : hence the intestines are streaked with bright red lines of capillaries that encircle them. In *puerperal fever* the *venous* capillaries predominate : hence the livid hue of the intestines, and the dusky red color of the patches and streaks on their surface. In *peritonitis* the lymph which is poured out is adhesive, uniting the different parts like glue. If removed from the surface of the intestine on which it is deposited, the strings of this lymph are broken across, and the surface is rough ; the quantity of serum poured out is not great, and, being lodged in the cavity of the pelvis, may at first escape observation. In *puerperal fever*, that which we call lymph is not adhesive : it is much more abundant than adhesive lymph, covering the fundus of the uterus, the intestines, the liver, the diaphragm ; it is found, also, in the *plenra* : its color varies from a dusky brown to a pale yellow : it may be peeled off the liver, the intestines, or the uterus, quite easily : the surface from which it is taken is smooth, and that of the intestines is a dark red color. The quantity of serum is equally profuse ; and this substance being dissolved in it, gives it a lactescent appearance, like pus : hence it is called sero-purulent fluid. Thus, when the abdomen is opened, a large quantity of this fluid always escapes. It will be objected that this sero-purulent fluid is also met with in *peritonitis*. This is perfectly true ; but it is necessary to note the stage of the inflammation in which it is observed. I have never met with it unless in the second stage of the attack. When a patient died in the first stage there was none of it. I conclude, therefore, that in the former instance (the second stage) such effusions only occurred when the constitution was sinking under the attack ; but in the latter, when death took place from a different cause, the effusions noticed were the true products of inflammation. In *puerperal fever* the greater the intensity of the seizure the less the chance of meeting anything like lymph. In the most intense forms no effusion at all may take place. In a degree less intense, a large quantity of serum, colored brown by blood, is found in the peritoneum and throughout the tissues : the lymph poured out is of the same color, having no adhesion to the surface on which it lies, as if the fibrine of disorganized blood had been deposited there. In the next degree, the same kind of lymph, or fibrine is found, of a yellow color, with a quantity of sero-purulent fluid. And lastly, in those cases in which the constitution for a time struggles successfully against the fever, some adhesive lymph will be

met with, mixed up with a larger quantity of what I have just described."

The next argument which I adduce in proof of the doctrine that puerperal fever is a zymotic disease, and not a local phlegmasia, is *that simple inflammatory diseases are not communicable from one patient to another through the medium of a third party*. It may be objected that this argument assumes that puerperal fever is thus communicable, which is not proven, and is one of the points now under discussion. With all due deference to those present who may differ from me, if any such there be, I must be allowed to say that I think no one fact in medicine is better established than this. The question of contagion is not one of abstract reasoning, but one of facts; and of these facts, a few, amounting to positive demonstration, must be conclusive. Negative testimony is utterly worthless in settling such a question. I should almost feel that I insulted the intelligence of those present by entering into an argument on this point at the present day. Prof. Oliver Wendell Holmes, in his essay on the *Contagiousness of Puerperal Fever*, has brought together an array of facts which must, I think, be convincing to every unprejudiced mind. For myself I would say, with Dr. Blundell, that I had rather those I esteem the most should be delivered, unaided, in a stable, by the manger-side, than that they should receive the best help, in the fairest apartment, but exposed to the vapors of this pitiless disease. I would heartily concur with the emphatic declaration of Dr. Holmes, that "if, on this point, there is any voluntary blindness, any interested oversight, any culpable negligence, even in such a matter, and the facts shall reach the public ear, the pestilence-carrier of the lying-in chamber must look to God for pardon, for man will never forgive him." Now, then, if this disease is thus communicable, is there any other local phlegmasia that is thus communicable? It may be objected that dysentery is sometimes contagious. I think I have myself been through an epidemic of dysentery which was evidently contagious, but I should answer, first, that it remains to be proved that this form of dysentery is simply a local phlegmasia; and secondly, that there is no evidence that a healthy person can communicate this disease from one person to another.

My next argument is that *the prophylaxis of puerperal fever is not the prophylaxis of local inflammation*. In the large hospital of Vienna, from 1840 to 1846, one in every ten mothers delivered perished, chiefly from puerperal fever. In May, 1847, Dr. Semelweiss prevented students from touching parts at the autopsies, and directed

all of them to wash their hands in a solution of chlorine, before and after every vaginal injection; and the mortality from this time so far diminished that in 1848, not above 1 in 74 mothers died. Does not this fact prove the toxæmic origin of the disease in these cases, and that the local lesions are secondary—reactive, and have less pathological value than the change which precedes it?

It may be objected that the views which have been advanced as to the pathology of puerperal fever, entirely ignore the existence of an epidemic influence, and that the epidemic influence may give a specific character to the local phlegmasia. From Sydenham, we have learned the phrase, "type of the season," and another phrase has come into use, meaning nearly the same thing, viz: "epidemic constitution." Now what is meant by these terms? Clearly they must refer to certain atmospheric or telluric influences which modify the susceptibility of the system to disease, or which increase the virulence of the poison which develops disease. That this influence really exists, acting in both ways, I think there can be no doubt. It sometimes produces its influence wholly on the system, diminishing the vital resistance to disease, and rendering inflammatory action, asthenic in its type; or the opposite result may be produced. So also, it may increase the virulence of the poison which gives rise to the zymotic diseases. Puerperal fever is most notably susceptible to an epidemic influence. I have thus given my reasons for believing that puerperal fever is an essentiality, that it is a zymotic disease, resulting from the absorption of a specific poison, and that its anatomical lesions are secondary.

The etiology of this disease has been so fully and so ably brought before the Academy by Prof. Smith, that I should not expect to be listened to with patience if I attempted to add anything to what he has said. So also in regard to its semeiology, it would be presumptuous in me to attempt to add anything to what is already known to the profession. The question of contagion, is one on which the profession is divided. My own views on this point have already been sufficiently clearly expressed. I certainly would not, at this day, seek to change the views of any one who has intelligently formed a different opinion, for I should deem it a hopeless task. As I do not intend again to occupy the time of the Academy during this discussion, I will beg your indulgence while I make a few remarks in regard to the treatment. This is the grand aim to which our discussion should tend, and its practical value rests on the bearing which it has on the therapeutics of the disease. The statistics

of the disease show that when it prevails in an epidemic form, about one in three die. It is to be hoped that in the progress of medicine, the resources of art will prove successful in greatly diminishing this frightful mortality. In some cases, the morbid poison is so intense as to overwhelm at once the vital powers. Just as in some cases of malignant scarlet fever, there is no capacity for reaction, and the patient dies in a few hours after the attack. In such cases as these, art must stand back appalled. The treatment of puerperal fever has afforded quite as fruitful ground for controversy as its pathology. I do not propose to review the various plans which have in different epidemics been supposed by violent partisans to be the most successful. I shall only refer to those general principles which should in my estimation govern the treatment—and give a few illustrations of some special methods of treatment. There are no specific therapeutics for puerperal fever. The sooner this idea is dismissed from the mind, the more probable is it that the treatment adapted will have a rational and philosophical basis. No one method is adapted to all types of the disease. It must vary according to the virulence of the epidemic or special poison, according to the condition of the system as to its vital powers when the poison is received, and according to the intensity and severity of its secondary lesions. I should say in general terms, that the indications are: *First, to eliminate from the system as much of the morbid poison as is possible by means of depletion and the other evacuants, as purgatives, emetics, diuretics, &c.*

Unfortunately this indication, owing to the peculiar character of this disease, can rarely be fulfilled, except to a limited degree. In the opinion of some, venesection is the grand remedial agent for puerperal fever, although, at the present day, the advocates of this measure, as essential to its successful treatment, are few in number. It proved to be the most efficient remedy in the epidemics met with by Gordon, Hey, Armstrong, and in one seen by Gooch. But in other epidemics, we have the testimony of equally sagacious observers, that it could not be borne. It proved an agent of destruction. Prof. Murphy has well made the point, that it may prove useful, where it can be tolerated, as a means of evacuating a certain amount of the materies morbi, thus relieving the central organs in which this poisoned blood has accumulated. It has not been my lot to see those epidemics which would tolerate blood-letting, yet I believe there are some. In sporadic cases it is, doubtless, much more frequently indicated. It seems to me that the principle which should

govern our practice in this disease, should be the same as would govern our practice in any other disease. Venesection should never be resorted to simply because the case is one of puerperal fever, but because the symptoms indicate that depletion is necessary. Admitting the pathological view, that "the fever is a natural effect of intense constitutional irritation, from local inflammation," it does not follow that blood-letting will be the remedy, or even that it can be tolerated. Rejecting this pathological view, it does not follow that venesection is not sometimes of great importance in the treatment of this affection. I hold that it is equally absurd to insist upon this as the cardinal remedy, as to denounce it as never applicable in the management of this disease. Common sense, not theory, must be our guide in regard to the use of this measure. The same general principles should govern us, in resorting to purgatives, emetics, diuretics, &c. Each of this class of agents has had warm advocates, and have, undoubtedly, been specially indicated, and proved eminently successful in certain epidemics. They are neither to be excluded wholly, or relied upon entirely, in the treatment. They may be indicated in certain cases, but they are only to be resorted to when there are special indications for their use. We are to remember that it is not generally our rôle to put out the fire, but to treat the burn, and hence the importance of the second indication, viz :

To control the vital disturbances resulting from réaction. These are principally vascular excitement and nervous irritation. It is unnecessary for me to enter into an elaborate argument, to prove the importance of these indications, for it is obvious that by vascular excitement and nervous irritation, the vital powers are exhausted, and death follows. It has been before remarked that it is the pulse which indicates the condition of the patient, much more than the local symptoms. Of the agents for reducing vascular action, we have first, venesection, when it can be borne, which is rarely the case. This means of reducing vascular excitement involves a loss of vital power. But we have, in the *Materia Medica*, an agent lately brought prominently before the profession, which acts specifically as an arterial sedative, without depressing the vital powers. I refer to the *veratrum viride*. We are indebted to Dr. Tully, of New Haven, for our first knowledge of its medicinal properties, and the profession in certain parts of Connecticut were familiar with its use long before Dr. Norwood's name was associated with it. It is simply and solely an arterial sedative. By it the pulse can be brought under volun-

tary control. For more than twelve years I have been accustomed to use it for this purpose, and for several years I have used it in puerperal fever, and in no disease have I seen its value more strikingly exhibited. It is an agent which requires care in its use, and in those cases where its full effects are required, I never allow them to be left without careful medical watching. The patient must be seen at short intervals. I have never seen any unfortunate results from its use, but I have seen it give rise to very severe temporary depression.* As an illustration of its action, I will read the report by Dr. Cobb, House Physician, of a very severe case of puerperal fever, which occurred in my service at Bellevue Hospital :

“ Kate Short, aged 23 years, fell in labor in full term at 2 o'clock P. M., Feb. 25, and was delivered of a healthy child at 8½ o'clock on the morning of the 26th. Nothing unusual occurred in her labor, except that the second stage was somewhat prolonged. Placenta came away in due time, and was not followed by hæmorrhage. First pregnancy.

February 28th, at 8 A. M., she was seized with a very severe chill, followed by increased frequency of pulse, and pain over hypogastric region, extending as high up as the umbilicus. This pain was very much increased by taking a full inspiration, or by the application of pressure. Tympanitis very considerable. The discharge abundant and very offensive. Pulse 140. Respirations 24.

At 1 o'clock P. M. Dr. Barker saw her, and recommended that she should be transferred to the Fever Wards, and put on the use of the Tinctura Veratri Viridis.

At 2 o'clock P. M., after having been removed to the Fever Wards, her pulse was 140. Respirations 24. Pain over hypogastric region intense. Tympanitis very considerable. Discharge abundant and very offensive. No mammary secretion. Dr. Barker

* There is a marked difference in the power of the article grown at the South, as compared with that grown at the North, which should not be forgotten in prescribing it. When I removed to this city, in 1850, the tinc. veratrum virid. was not kept by the druggists here, and I therefore procured some from Norwich, Conn. I was accustomed to prescribe this (a saturated tincture made from the article growing in Connecticut) in doses of from 12 to 20 drops. In the first case in which I made use of the tincture now found in the shops here, alarming prostration was produced, and I soon learned that I must diminish, very decidedly, the dose. Prof. Dickson, of Charleston, S. C., informs me that 7 drops is a large dose of the tincture used at the South.

requested that she should be seen hourly by one of the House Staff, and that her condition, as to the state of the pulse, respiration, and other symptoms, and the dose of the veratrum viride given, should be recorded at each visit. The following is the record thus kept :

February 28th.	Hour.	Pulse.	Resp.	Drops.	
	2 P.M.	140	24	10	
	3	127	22	10	
	5	140	22	10	
	6	132	12	10	
	7	120	20	10	
	8	80	20	9	Bowels moved once.
	9	75	16		Vomited a greenish colored fluid. Bowels loose.
	10	68	16	4	Vomiting ceased. Bowels moved once.
	11	65	22	7	
	12	58	13	2	
March 1st.	1 A.M.	64	52	6	Respiration vary irregular. Inclined to sleep.
	2	58	25	2	Sleeping.
	3	59	21		Hiccough and headache.
	4	60	18	1	Hiccough still continues.
	5	66	20		Severe headache. Vomited a greenish colored fluid.
	6	66	21		Headache severe, and very restless. Vomited several times within last hour. Hiccough.
	7	58	20		Vomited once since last visit. Vertigo and headache.
	8	52	28		Sleeping.
	9	60	19		
	10	68	21	1	Slight hiccough.
	11	70	23	2	
	12	80	28	3	Tenderness over abdomen, marked. Tympanitis somewhat diminished. Discharge dark, bloody, and very offensive.
	1 P.M.	80	20	4	Visit of Prof. Barker.
	2	92	24	8	
	3	76	24	9	Face flushed.
	4	76	28	9	Sleeping.
	5	88	28	8	Sleeping.
	6	66	28	8	
	7	68	26	6	Slight hiccough. Bowels moved once.
	8	66	18		Vomited a greenish colored fluid.
	9	68	24		Vomited once since last visit.
	10	60	28		Sleeping.
	11	64	28		Still sleeping.
	12	66	28	2	Sleeping still.
March 2d.	1 A.M.	56	32		
	2	70	24	3	Complaints of pain in left thigh. There is slight swelling, and along its internal surface, over the course of the veins and lymphatics, the tenderness is so great that she can scarcely bear the lightest touch. Tenderness over abdomen still continues. Slight Tympanitis. Discharge abundant, dark, bloody, and very offensive. No mammary secretion.
	3	76	24	4	
	4	65	20	3	Sleeping.
	5	78	22	8	
	6	68	22	4	
	8	64	24	4	
	9	72	24	6	
	10	64	28	2	Bowels moved once.
	11	72	28	6	
	12	70	24	5	
	1 P.M.	64	24	3	
	2	60	20		
	3	64	24		
	6	68	28	3	
	7	72	28	5	
	9	80	28	6	Face flushed.
	10	80	26	6	
	11	80	28	8	
	12	80	28	10	Sleeping.
March 3d.	1 A.M.	80	29		Vaginal discharge now ceases to be offensive. No mammary secretion. Tympanitis still remains. Tenderness over abdomen still continues, though not so well marked. Tenderness and swelling in left thigh still continues.
	2	78	28	10	
	3	80	28	8	Slight hiccough.
	4	72	20	4	
	5	68	28		Vomited a greenish colored fluid. Headache. Hiccough. Bowels moved twice.
	6	64	24		
	8	60	24		
	9	68	24	5	
	10	64	24	3	
	11	68	28	6	

March 3d.	Hour.	Pulse.	Resp.	Drops.	
	1 P.M.	80	25	6	
	2	80	25	6	
	3	76	23	4	
	4	76	26	5	
	5	73	23	4	Sleeping.
	7	64	23	2	
	8	72	23	5	
	9	68	20	4	
	10	68	23	5	
	11	72	23	5	
	12	70	20	7	
March 4th.	1 A.M.	72	22	8	Sleeping. Tenderness over abdomen not so intense. Slight tympanitis. Vaginal discharge now appears to be natural. Tenderness and swelling on internal surface of left thigh now seems to be diminishing. No mammary secretion.
	2	70	20		
	3	64	23	2	
	4	64	23	2	
	5	60	24	2	
	6	60	23	2	
	7	60	23	2	Bowels moved twice.
	8	55	23		
	9	60	23		
	10	66		2	
	11	64	23	2	
	12	72	24	4	
	1 P.M.	78	23	6	
	2	80	23	8	
	3	80	24	8	
	4	80	20	8	
	5	80	23	8	Sleeping.
	6	60	23	8	
	7	64	24	6	
	8	60	24	2	
	9	60	23	2	
	10	60	24	2	
	11	60	26		
	12	68	24		
March 5th.	1 A.M.	60	22	8	She now says she feels much better. Her countenance looks much brighter, and she appears to be much improved in every respect. The tenderness which has been so intense over the abdomen, now is scarcely noticeable. Tympanitis very slight. Discharge very scanty, but normal. No mammary secretion. The swelling and tenderness on the internal surface of the thigh, in the course of the veins and lymphatics, has now disappeared altogether.
	2	68	26	4	Sleeping.
	3	60	23	2	
	4				
	5				
	6	70	20	6	
	7	64	24	4	
	8	76	24	6	
	9	76	24	6	
	10	72	23	6	
	11	64	24	3	
	12	68	24	6	
	1 P.M.	64	23	5	
	2				
	3	56	23		
	4				
	5	64	24	5	
	6				
	7				
	8	68	26	4	
	9				
	10	72	24	4	
March 6th.	8 A.M.	70	24	6	Feels well; improvement marked. No tenderness on pressure over abdomen. No tympanitis. Discharge still scanty, but normal. Slight mammary secretion.
	11	76	24	4	
	12				
	1 P.M.	72	24		
	5	78	23	8	
	6				
	7	76	26		
	8				
	9				
	10	72	24	4	
March 7th.	9 A.M.	76	24		She says she feels well and hearty. No tenderness over abdomen. No tympanitis. Vaginal discharge healthy. No tenderness or swelling in left femoral region. Appetite good. Bowels regular. Continues to improve very fast.
March 8th.	10 A.M.	76	24		

From this time she continued to improve, and in a short time was discharged as well and hearty as she ever was.⁷⁷

Now here is a case occurring in a hospital, at the time of an epidemic, presenting a combination of symptoms which all familiar with the disease would pronounce truly alarming. By the *verat. virid.* the pulse was brought down from 140 to 60 per minute, and it was never permitted to rise above 80. The quantity administered varied according to the condition of the patient, two, three, or four drops being frequently sufficient to control the vascular excitement. No other medicine was used. In many other puerperal cases, I have seen equally striking results. I will briefly mention one which I saw, in consultation with Dr. Sayre, the tenth day after confinement. She was a primipara, and her convalescence seemed perfectly normal, until the sixth day, when she began to exhibit some appearance of mental disturbance. She was especially anxious in regard to her religious condition. Gradually a high state of nervous excitement was developed, with insomnia, and when seen by myself, she had been decidedly maniacal for more than twenty-four hours. Her respiration was short and hurried, her pulse very rapid, her countenance anxious and frightened; she was incessantly talking and starting with apprehension, from the slightest movement in the room. No physical exploration could be obtained, but there were no local symptoms indicating pelvic trouble. She sat up in bed, and moved from one part to another with great rapidity. The *verat. virid.* was now given, and by its influence the pulse was brought down below 70 per minute, the respiration became slower, the mind tranquil, and she was enabled to sleep. I am informed by Dr. Sayre, that in the course of a few days there was developed, in the pelvic cavity, an extensive abscess, which pointed externally, near the sacrum. Her convalescence was somewhat prolonged, but she eventually recovered.

One of the most important indications we are called upon to fulfil, in the management of this disease, is to allay nervous irritation. There is no doubt that the most frequent of all the lesions of puerperal fever, are those of the peritoneum, and that the disturbance to the nervous system is much more severe than from any other local cause. It is well known to most of the profession, that within the last four or five years, this has been treated by heroic doses of opium, or of some of its preparations. It has long been used by the profession in the treatment of peritonitis, and Graves and Stokes have demonstrated its great value in idiopathic, or traumatic peritonitis, but to Prof. Clark belongs the honor of fully testing it, in what he calls puerperal fever, with peritoneal lesion. This is his own ground,

and I will not encroach upon it, inasmuch as we hope to have, in detail, the results of his enlarged experience. I have treated a few, a very few compared with him, by this method. It is astonishing to see to what extent patients will tolerate opium, where the peritoneal lesion predominates, but it is only in this form of fever that this great tolerance exists. The quantity given in some cases, without producing narcotism, is enormous. But there is one point to which I wish to call attention, and that is a test whether the action of this drug is proving beneficial or not. If opium be pushed to incipient narcotism, or a point little short of it, a gradual decrease in the frequency of respiration results. In some of my cases, the respiration went down to 14, 12, and 10 per minute. Now, then, the opium treatment is acting beneficially, when, in connection with the reduction of the frequency of the respiration, there is a corresponding decrease in the frequency of the pulse, but if the opium is pushed to the point of incipient narcotism, the respiration growing slower and slower, without a corresponding decrease in the pulse, I should say the opium treatment is to be abandoned at once. In one case that occurred at Bellevue Hospital, some two years since, the opium had been pushed to such an extent, that galvanism had been resorted to to make her breathe. When I saw her, the respirations were 10 and 11 per minute, while the pulse was about 140 per minute. Seeing this slow respiration, with the frequent pulse, I suggested that no more opium should be administered, as I thought its continued use would be likely to overwhelm the vital powers. The *veratrum viride* was then given, and in a few hours the pulse came down below 80. This patient eventually recovered. I will state, then, as my conviction, that in that class of cases where the peritoneal lesion predominates the opium treatment has proved successful to an extent which no other has.

In many cases, to control the vital disturbances resulting from reaction, it will be necessary to use a variety of agents to accomplish this end. Venesection, *veratrum viride*, opium in full doses, camphor, all may be indicated, and prove eminently serviceable in the same case. In illustration of this, I will mention a case which occurred in my private practice. The patient, a primipara, was delivered by the forceps, after a very severe labor, on the 4th of July last.

On Sunday, the 5th, everything seemed to be going on in the most favorable manner. I saw her again on Monday morning, and there was no indication of disturbance of the general system, except that she complained somewhat of nausea, her breath smelled like raw

beef, and the tongue was covered with a pasty, white coat. I was sent for to see her again that day, between 5 and 6 P. M. I found that she had been seized, a little time before, with a violent rigor, her countenance was pale and haggard, wearing an anxious, despondent look. She complained of intense pain over the lower part of the abdomen, particularly over the left iliac region. Her pulse was about 132 per minute. Here was a case calculated to excite the gravest apprehension. The following prescription was made, and it will at once be apparent what indications it was designed to fulfill :

R. Pulv. G. Camphor, - - - ʒss
 Sol. Morphiaz (Majend.), - gtt.lxxx
 Tinc. Aconite, - - - gtt .xvj
 Mucill. G. Acaciae, - - - ʒjv
 M. S. A tablespoonful every second hour.

Turpentine fomentations were applied to the abdomen. She was seen again by me late that evening. The pain was decidedly less, but the pulse continued very frequent. She obtained no sleep that night. The mixture was continued the next day, at intervals of four hours. On the third day from the attack, fifth after delivery, she was seen by my colleague, Dr. Peaslee, who found that the pain had ceased, skin soft, pulse 92, but there was still this pasty coat upon the tongue. She subsequently had some swelling and pain in the left leg, along the track of the crural vein, but this continued but two days. The mixture was continued in diminished doses for several days afterwards, as the pulse continued frequent after all the local symptoms had disappeared. She had no mammary secretion. Under this treatment alone she perfectly recovered. I will give a brief outline of another case, which was to me most interesting and instructive. This patient was confined about the 1st of August, and in this case also the forceps were necessary on account of the position of the head. It was the right occipito-iliac posterior, the occiput rotating back to the sacrum, instead of anteriorly. After the labor was completed, I gave her a full dose of opium, as I usually do, when it has been severe. For two days after delivery, everything went on favorably, but on the evening of the third day, she had a slight rigor, and was seized with a most intense pain in the lower part of the abdomen and in the vagina, so severe that although a person of great self-control, she shrieked out with agony. Her pulse was very rapid. Turpentine fomentations were applied to the lower part of the abdomen, and Majendie's Sol., in full doses, was given until the pain subsided. The pulse continuing very rapid,

I then gave the *verat. virid.* in 12 drop doses every hour, until the pulse was brought down below 80, and there it was my aim to keep it. On the evening of the fifth day after confinement, I was sent for in great haste, when I found her with symptoms of cerebral congestion of the most alarming character. The attack had come on suddenly, without premonition. She complained of asphyxia, her countenance was livid and turgid, and every appearance was such as to indicate the most imminent danger. I should mention that, although not a person of full habit, I had found it necessary to bleed her a few weeks before confinement.

I now opened a vein and abstracted about 30 $\bar{3}$, which at once relieved her of her cerebral symptoms. Previous to her confinement the urine had been tested for albumen, but none was found. The blood now drawn was examined by my friend, Prof. Doremus, and found to contain urea. I say after bleeding there was entire and complete relief from all the cerebral symptoms, but still the pulse remained rapid and frequent, and there was still a tendency to pain in the vagina and pelvic cavity, requiring the occasional use of Majendie's solution. The point that I wish to call attention to is, that the second day after venesection the local symptoms disappeared in a great measure, but if the *veratrum viride* was not continued the pulse would become extremely rapid, and this patient required the constant and steady use of this remedy for thirteen days. It may be said by some that these were not cases of puerperal fever, and I have anxiously asked myself the question whether they were so or were cases of local phlegmasia. I answer that they were cases of puerperal fever, and I will mention only one reason for believing so, viz : after all the local symptoms had disappeared there was still left evidence of poison in the system as shown by the rapid pulse.

3d. *To combat the local secondary lesions which may be developed.* I will not take up the time of the Academy in enlarging upon this part of the treatment. Local depletion, counter-irritation, fomentations to the abdomen, turpentine endermically, opium to subdue pain, chlorinated injections—the value of all these measures where special indications for their use exist, has long been settled by the profession. The discriminating physician will employ each or all of these methods as adjuvants to the radical treatment of the case.

I will only add one other indication, viz : *to sustain the vital powers of the system.* In other words, keep the patient alive. There are a certain class of cases where the system seems to be overwhelmed, and yet life will be preserved by the heroic use of stimulants and

good nutrition. I believe many are permitted to die from the neglect of these resources. It seems to me that after a patient with puerperal fever has lived for forty-eight hours, there is constant encouragement for effort, and that the danger is, in a certain sense, diminished in proportion to the duration of the disease. Without enlarging upon this topic, I will read a brief abstract of another case, reported by Dr. Cobb, which occurred in my service at Bellevue Hospital, which I think will illustrate my ideas better than argument. This patient was so utterly prostrate by the disease, and had such a variety of secondary lesions, that she was regarded by myself and all who saw her as past praying for.

“Matilda Smith, aged 21 years, first pregnancy, was delivered, in the lying-in wards of Bellevue Hospital, of a healthy child, at full term, at 8 P. M., February 11th. For the first few days after delivery she appeared to be doing well. Nothing unusual occurred to call attention to her case until February 17th, when she had a severe chill, with a quick, rapid pulse, and intense pain over the region of the uterus. The vaginal discharge was profuse, very dark colored, and excessively offensive. A large blister was applied over the region of the uterus, and Dover’s powder, calomel, and camphor were administered, and she was removed to the fever wards.

This treatment was continued for forty-eight hours, but without benefit. On the contrary, her symptoms were constantly growing worse. Her pulse was above 140, weak and irritable. Exquisite tenderness over the uterus, the vaginal discharge abundant, very black, and extremely offensive. She vomited frequently a greenish colored fluid, and became somewhat deaf. Dr. Barker now ordered porter, milk punch, beef tea, as much as the stomach could take care of, and a full opiate at night. For ten days her condition varied but little from that above described. The stimulants were pushed to the point of tolerance, but the pulse continued very weak, rapid, and irritable. Her whole aspect was as bad as possible. Quinine was tried, but it could not be borne, as it induced severe headache.

February 28th she had an attack of capillary bronchitis, accompanied with profuse perspirations and coldness of the surface. This was relieved by extensive dry-cupping over the front and back, and the liberal administration of Carb. Ammonia. A few days after an abscess made its appearance in the right mamma, which, when opened, gave exit to at least two pints of very offensive pus. She also had a large bed-sore. These three complications, capillary bronchitis, mammary abscess, and bed-sore, made their appearance about the

same time. Diarrhœa set in March 3d, which was found very difficult to control. On the 4th it is recorded that she took a moderate quantity of beef tea, two bottles of porter, and 30 $\frac{3}{4}$ of port wine. March 5th, she appears somewhat better. Pulse varying from 125 to 135. Perspirations still very profuse, and vaginal discharge was still very offensive. Tenderness over the uterus not so intense; diarrhœa ceased. From this time she gradually improved, but her convalescence was greatly retarded by the extensive suppurations in the mamma and bed-sore. Early in April she was discharged cured."

This case was watched with great interest by the students in attendance from the different Colleges, and I need not add that her recovery was as gratifying as unexpected. In the interesting history of the recent epidemic of puerperal fever in the Dublin lying-in-hospital, by Dr. McClintock, the present able Master, it will be observed that he found it necessary to make a liberal use of stimulants. Apologizing for the length of my remarks, I will occupy the time of the Academy no longer.

Prof. Clark then rose and stated, that he did not rise to continue the discussion. He congratulated the Academy upon hearing such an interesting paper from Dr. Barker. If he was not witty himself, he seemed to have been the occasion of wit. It had proved a good fortune to the Academy that Dr. Barker was not present when this subject had been brought up before. The Academy would agree with him that it was a very connected and substantial argument to prove his point. It did not differ very materially from his own views on the subject, and he would propose that it be continued for discussion at the December meeting, when he hoped to be present. At the time of the November meeting, he thought it probable that he should be obliged to be absent from the city.