LUMLEIAN LECTURES

ON

THE CONVULSIVE DISEASES OF WOMEN.

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LECTURE II.

Epilepsy in Pregnancy and Lactation; Cases.—Chorea and Ague in Pregnancy; Cases; Conditions determining the Convulsion.—The Influence of Ovarian Nisus.—Vomiting in Pregnancy; Three Groups of Cases; Complication with Poisoned Blood.—Tetanus.—Whooping-Cough.—The Rise of Convulsive Diseases in Pregnancy puts to test the various Theories of the Genesis of these Diseases.—Influence of the Shock of Convulsion in inducing Structural Change in Nervous Centres; in altering Periodicity or Susceptibility.

MR. PRESIDENT AND GENTLEMEN,—Since our last meeting, one of the two women whom I described as suffering during pregnancy from uncontrollable spasmodic twitchings or “jumpings” of the legs, has been delivered, labour having come on prematurely. The fact is interesting, in illustration of the law of augmented centric nervous irritability. The immediate provoking cause of the labour seemed to be a distressing cough which the patient caught a week before at Hastings. The commotion caused by the cough destroyed the harmony or balance of nerve-supply and demand; and the irritation, beginning under its influence, was propagated to the part of the cord which rules over the dynamics of parturition. When the nervous centres are in a state of exalted tension, irritation telling upon any part of the brain through the emotions, or of the cord through reflex action, seems liable to spread and involve the whole cord. And if one part, as in pregnancy, be especially irritable, this part will be excited to its special action. Or we might express it in another way. We may imagine the spinal cord to be a conductor along which an impression made at any point is transmitted rapidly to the rest, testing and calling into action any part which is in an especially excitable condition.

Thus, as in the present case, cough excites labour; but under other conditions of special influence, as of blood-poisoning, or of proclivity from hereditary disposition, a cough might set up convulsion.

This law of the propagation of excitation from one part of the spinal cord to another part in a state of peculiar susceptibility, is admirably illustrated in a remarkable clinical experiment recorded by Harvey. “It seems to me,” says our immortal physiologist, “on deep investigation, that the throes of childbirth, just as sneezing, proceed from the motion and agitation of the whole body. I am acquainted with a young woman who, during labour, fell into so profound a state of coma that no remedies had power to rouse her; nor was she, in fact, able to swallow. When called to her, finding that injections and other remedies had been employed in vain, I dipped a feather in a powerful sternutatory, and passed it up the nostrils. Although the stupor was so profound that she could not sneeze, or be roused in any way, the effect was to excite convulsions throughout the body, beginning at the shoulders, and gradually descending to the lower extremities. As often as I employed the stimulus the labour advanced, until at last a strong and healthy child was born without the consciousness of the mother.” It is in the highest degree probable that in this instance there was albuminuria and uræmia. But how well must Harvey have been acquainted with the reflex function, when we see him thus turning it to practical account to accelerate the course of labour.

But the practical unity of the cerebro-spinal axis is not seen alone in the action of physical peripheral irritation; it is equally demonstrated by the influence of the mind. It is familiarly known to obstetric practitioners that depressing emotions experienced during labour will often stop the “pains,” and that a feeling of confidence and courage will impart to them activity. It is greatly through the blotting out of the sentiment of fear, that chloroform acts so like a charm in promoting the healthy course of labour in women of delicate and susceptible organisation.

There is yet another illustration of the law we are now discussing, which I may be excused for introducing. It is seen in that vivid sym-

pathy which springs up between pregnant women. Thus, if a woman far advanced in pregnancy assist at the labour of another, she seems herself to catch every pain that seizes upon her suffering sister; and cases are known—I have recently seen one—where labour has in this way been actually excited. I have read somewhere, but I cannot lay my hand upon the reference, that the same thing is observed amongst pregnant mares and cows, so that those who have the charge of pregnant animals take care to separate from the rest any one which may be taken in labour.

I will now take up again the thread where I laid it down at the close of last lecture. Pursuing the method adopted at the beginning, I propose to work out the remaining relations of pregnancy and labour to convulsions, before discussing convulsion in the non-pregnant state.

Although there is no condition to parallel that marked by albuminuria in the proclivity to convulsion, yet we find other conditions which strongly predispose to similar outbreak. The very difference in nature of these conditions supplies further evidence of the truth of the fundamental law, that in pregnancy there is a special and intense accumulation of nerve-force.

EPILEPSY.

I shall best illustrate the relations of epilepsy to pregnancy by brief reference to cases.

CASE I.—A young woman had had three labours at term, and three abortions; all the children were born dead excepting one, and that lived only seven weeks. She had not, as far as could be ascertained, had fits before marriage. The history and present evidence of syphilis were distinct. She had been delivered a week. Convulsions had appeared during pregnancy, and fits were frequent after labour. She started convulsively on any noise or movement, showing a highly sensitive state of the nervous centres. There was no œdema; no albumen in the urine. There was general debility.

CASE II.—F. F., aged 32, had had three children and two abortions, the last child three years ago; she was seven months pregnant. She had several fits when suckling the last child; she had been only three days delivered when the first fit occurred; she had had fits in increased frequency since being pregnant; she lost consciousness. She looked anæmic; the pulse was small; there was œdema in the feet and legs; venectasia of legs; palpitation. The urine contained no albumen; its specific gravity 1,014.

CASE III.—G., aged 26, had one child one year and ten months old; she came under my care at the London Hospital. She had previously been under the care of my colleague, Dr. Andrew Clark, who had advised her to wean after six months' suckling. The fits broke out a month after labour, lasting four minutes. Since weaning she had a fit once a month only. She had not menstruated since labour. Palpitation was very distressing; she had “flushes to the head” preceding syncope; moaning followed, but there were no spasms now. The pulse was weak; there were increased impulse of heart, and slight anæmic sound. She had intense inflammation of the neck of the uterus. Under treatment for this, and taking acetate of iron, she got much better. The fits returned when she left off her medicine, and were “kept off” by taking it. In three months she menstruated regularly. Having been free for some time, whilst menstruating she was struck by her husband; this brought on a fit, and the menstruation ceased suddenly. The fits then returned, with sickness and headache. Soon after this she was lost sight of.

CASE IV.—*Anæmia from protracted lactation; epilepsy.*—M.S., aged 36, has had four children, the last eighteen months ago, and was still suckling. She had a fit, the only one, last week; was two hours unconscious; was told she was “convulsed.” She had nothing of the kind after previous labours; had suckled a previous child for two years. She had great pain under the left breast; short breath; some prolapsus and subinvolution of the uterus.

CASE V.—A pluripara, had had epileptic fits at rare intervals when not pregnant. She had a convulsion just before delivery. The urine was acid; specific gravity 1020; it contained no albumen, the full proportion of urea, and a little sugar.

CASE VI.—On the 9th June, 1869, I saw a young woman who had been married about a year. She had been delivered on the 2nd inst. of a live child, after an ordinary labour. Next day she had an epileptic fit; the face remained much congested for three days, but there was no return of fits. The urine was not albuminous. At times she was delirious. On the third day after my visit there was a sudden attack of secondary hæmorrhage, from the effect of which she died. She was apparently a healthy young woman. No history of previous nervous disorder could be elicited, but her husband, a young man, was seized with a paralytic stroke a little time before her labour. The shock of this event had caused her considerable agitation.

CHOREA IN PREGNANCY.

CASE 1.—E. D., aged 22, came under my care at St. Thomas's Hospital. She had had two children, and suckled both for twelve months; she was now between three and four months pregnant. She had rheumatic fever at seventeen; chorea appeared three or four months afterwards, and reappeared with each pregnancy in the early months; she got better on quickening. The movements were all on the right side. After taking zinc and iron for a month she became much better as to the choreic movements, but still complained of headache. There was still some systolic murmur.

CASE 2.—C. E., aged eighteen, seven months gone in her first pregnancy, had a fright when three months gone; she had chorea when a child, five years ago, not since until the fright; then the "jumpings" and chorea came on, and continued ever since. She had typhoid some time ago, and was anæmic. The uterine souffle was heard as a strong whistle in left hypogastrium.

The relation between a fit of ague and convulsion is too striking to be overlooked. Pregnancy and labour seem to exercise the same influence in causing a relapse as they do in the case of chorea.

CASE 1.—In 1863, I saw, with Mr. Corner of Poplar, Mrs. W. She had had several children. She had had ague in more or less distinct form for two years. She was delivered five weeks ago, and is suckling the child, but the milk is not copious. She seemed doing well, when five days ago she was seized with a marked ague-fit. This left her much prostrated; appetite impaired; heart's action feeble; no intumescence of liver or spleen. We advised removal to a high gravelly soil. She took quinine, then cobweb, which, Mr. Corner said, was followed by marked benefit.

If we now analyse these cases, we find, amongst other lessons which they teach, the following.

1. There is the fundamental state of nervous tension or erethism due to pregnancy and labour.
2. There is a degraded state of the blood, often vaguely called anæmia, but which is more strictly a form of toxæmia.
3. In some cases, to a degraded state of the blood is added a morbid diathesis, which in all probability involves a peculiar organic modification of the nervous centres.
4. Then there comes an emotional or physical shock, or a peripheral irritation, which, acting upon a system prepared to explode, determines the convulsion.

Thus in Case I there was the syphilitic diathesis—a condition well known to be capable of evoking, or leading up to, epilepsy in men. But in this case labour was necessary to determine the convulsions.

In Case II, epilepsy followed on the exhaustion produced by suckling. It is in cases of over-suckling that we see most strikingly the influence of exhaustion—say, of anæmia. The thin blood, deficient in red corpuscles, can hardly afford proper nourishment to the nervous centres. But the term "anæmia", or "spanæmia", imperfectly describes the kind of blood-impairment found in these cases. It would lead us away too far from our present theme to examine the reasons which seem to me to prove that a state of pure spanæmia, or simple dilution of the blood, can hardly exist. Blood which, from its poverty, is incapable of nourishing the organs, and of stimulating them to the adequate performance of their functions, necessarily soon becomes contaminated by excrementitious matters. Spanæmia, then, implies toxæmia. Excretion is always imperfectly performed. Almost any animal poison in the blood seems to possess the property of unduly exciting the nervous centres. There is hardly a fever which does not manifest evidence of cerebrospinal irritation, by delirium, or by that form of convulsion known as *subsultus tendinum*. The poison of spanæmia is different from the specific poisons of fevers; but, like them, it irritates the nervous centres, and, like them, it is likely to cause various irregular involuntary muscular actions, recognised as spasms, cramps, twitches. To return, then, to our case of epilepsy, provoked or evoked by over-suckling. It is a type of a considerable class. The mother goes through perhaps one or two pregnancies and lactations without serious ailment; but under repeated wear and tear, as in this case, at the third lactation, she breaks down. The blood is degraded, the nervous centres are ill-nourished; but, the function of lactation going on, there is maintained an inordinate centric irritability. Under these conditions, convulsion is near at hand. If there be present a convulsive diathesis, it is pretty sure to break out. If she have at any time been "subject to fits", it is almost certain that they will reappear now. That convulsions should occur, even where they had never occurred before, and where we can trace no evidence of convulsive diathesis, affords very strong presumptive evidence of the independent adequacy of the conditions which we are discussing to produce them.

It may be objected that, when pregnancy and labour are past, there

is no longer equal necessity for that preponderant activity of the cerebrospinal axis which I have invoked. But there are abundant facts to prove that lactation, which, like pregnancy, imposes upon the mother the double duty of supporting two beings, really does imply a corresponding increase of nervous energy. Nor is this all. Throughout this work—of itself sufficient to tax the mother's powers to the utmost—the ovarian stimulus is commonly acting. In married women, especially, the ovaries can hardly be kept in abeyance: they are always struggling to renew the cycle of the function of reproduction. The effort at ovulation frequently determines menstruation in spite of lactation; and even where the blood-flow—the outward manifestation of ovulation—does not occur, there is reason to believe that ovulation, more or less complete, goes on. This state, added to the excitation of the sexual act, causes a constant renewal of exaggerated cerebrospinal irritability. Hence it is that we see, in some cases, a periodical recurrence of epileptic fits, although there may be no visible menstruation. Illustration of all this is seen in Case III, in which epileptic fits recurred monthly.

It is remarkable, as bearing upon this argument, that protracted lactation, apart from ovarian excitement, is not nearly so likely to lead to nervous exhaustion, or to convulsion or insanity. Thus it is no uncommon thing for women who lose their husbands whilst they have a child at the breast, to go on suckling for an indefinite time. While they concentrate all their emotional and other nervous expenditure upon this vicarious duty, the ovaries are kept in subjection, and there is no excessive development of spinal irritability. Thus I have known poor women, badly fed, hardly worked, continue to suckle for two, three, and even seven years; exhausted, it is true, in strength, but free from convulsion or insanity. The nervous diathesis was wanting.

But it is in pregnancy, when the tension of the nervous centres is at the maximum, that we see the most striking proclivity to epilepsy. I do not know a recorded case in which this is more forcibly shown than in one which I saw several times with Dr. Sharpe of Norwood, the history of which he has drawn up for me. It is altogether one of the most interesting I have ever known, and I wish I could read it in detail. The main features are as follows.

Mrs. T. Many of her blood-relations were strumous or phthisical; her grandfather died of apoplexy; her father had hæmoptysis, and was dying of Bright's disease; her elder brother had been epileptic from birth, and died at thirty-two. She herself was healthy, although she had never menstruated when she married at eighteen. Her first pregnancy ended in labour at term, and she suckled for ten months, when menstruation returned. During the next five years she aborted four times, generally at eight or ten weeks, suffered much from hæmorrhage and great prostration, and great mental anxiety. The sixth pregnancy went to term; she nursed for eleven months, when she became pregnant again, and aborted at six weeks. Two years after this, being pregnant again, six weeks after a day of much fatigue, she had a fit in the night while asleep. She was unconscious for a long time; had a succession of fits; aborted after two or three. It was ten months before she recovered from this illness. During these ten months she menstruated regularly, having a slight fit before each period, on recovering from which she would find herself unwell. At the end of these ten months she was pregnant again, and at once "took fits", and had them frequently during the whole pregnancy, which lasted nine months. When labour came on, fits came on. She had no fits after this child was born. She nursed it for thirteen months, having plenty of milk. For three years there was no pregnancy, and she remained very well. Then, being pregnant for the tenth time, she had no fits; was delivered at term; was nursing, when at the end of seven months she had a fit, and found herself pregnant for the eleventh time. During this pregnancy she had many fits at varying intervals, went to term, had no fit during labour, but they broke out immediately afterwards, severe and protracted, she being semi-comatose between. She nursed for twelve months. When pregnant for the twelfth time, she aborted at six weeks, had much hæmorrhage, and fainted, but "had no fits." This account Dr. Sharpe, in my opinion, rightly doubts. He suspects that the two "faints" were two fits. In about eighteen months after this she was pregnant for the thirteenth time, and went on very well for three months, when she "quickened", having had the day before a very severe fit. A fortnight after this, she found her right leg paralysed. She took to bed, having fits from time to time until she was five months and a half gone, when the fits became very severe, and the coma so prolonged as to cause great anxiety; the paralysis of the leg continued. Labour was induced by rupturing the membranes. She had no fits during the labour or in childbed. She continued pretty well, except that the paralysis remained, and is now pregnant for the fourteenth time. She had another slight fit at the beginning, one at the end of four months, and one at the end of the fifth month.

Such is the record, not yet complete, of this "strange eventful history." It illustrates, in several repeated series of experiments instituted by Nature, almost all the points upon which I have dwelt.

1. There was probably an hereditary predisposition to nervous disease. But Dr. Sharpe specially records that this woman is the reverse of hysterical; she is a woman of surprising bodily and mental energy, of steady, determined will, ruling her own household firmly, and, having still energy to spare, rules those of sons-in-law as well.

2. She never had fits unless when pregnant, except during the ten months between her eighth and ninth pregnancies, when she had a slight fit at each monthly period.

3. The menstrual or ovarian nixus produced fits, shown by periodicity of recurrence during lactation and pregnancy.

4. She had fits during the climax of coition, but never except when pregnant. This last fact or experiment affords striking evidence of the increased efficacy of irritation during the inordinate spinal erethism of pregnancy.

5. The increasing preternatural irritability of the spinal cord is manifested in the repeated abortions following the first pregnancy.

6. These abortions and the protracted lactations induced a gradual blood-deterioration, the effect of which culminated, after six years of reproductive troubles, in the first epileptic fit during the nervous tension of pregnancy.

The urine, examined at four different times, never showed any albumen. The case was one of pure epilepsy.

Although, as in this case, epilepsy will frequently excite abortion, it is not nearly so likely to bring on labour as eclampsia, showing that the blood-poison in the latter case is a special cause in augmenting the irritability of the nervous centres.

VOMITING IN PREGNANCY.

The morning sickness of pregnancy is proverbial. It requires no treatment. Up to a certain point it even seems to fulfil a useful physiological purpose. It is usually the first evidence of the cerebro-spinal erethism produced by pregnancy. Its action may be compared to that of a safety-valve discharging the superabundant nerve-force, which might otherwise result in convulsion, abortion, or other mischief. Its constant occurrence in the morning seems to imply that at this time there is a maximum of central nervous irritability, so that comparatively slight peripheral causes will then act with more effect. There are many facts which lend support to this hypothesis. The immediate irritating cause I believe to be the stretching of the uterine muscular fibre under the eccentric pressure of the growing ovum, and the turgescence of the uterine vessels. In many cases, after three or four months, the balance is restored, and the vomiting either ceases or is so moderate as to be easily tolerated. It is remarkable, however, that in not a few cases vomiting appears to set in almost from the moment of conception. These cases may be explained by the facts—(1) that conception has taken place, as it most commonly does, at a menstrual epoch—that is, when there is an exalted central nervous tension; (2) by the stimulus or irritation of coition.

In some cases I have seen, the vomiting was so distressing, so prostrating, within the first three months, as to bring the patient to the most critical position, and even to prove fatal.

In other cases, the vomiting, less urgent at the beginning, being continuous, and increasing in severity, brings the patient into danger at the fourth, fifth, sixth, or seventh month.

In yet other cases, the first half of pregnancy may have been got over without much distress. The vomiting may have even stopped altogether; when, about the sixth or seventh month, it becomes uncontrollable, quickly exhausting the patient, even threatening her life.

These three groups of cases demand separate consideration. The first group, that comprising cases of severe vomiting within the first three months, includes cases of women who have borne several children, as well as primiparæ; although probably the primiparæ are the majority. In these, the preponderating condition is the extreme convulsive tension of the nervous centres. The subjects are generally "nervous," susceptible to emotional and physical impressions. In some, there are predispositions of a kind similar to those which operate in the production of convulsions. In some, and this may be especially predicated of those women who have already borne children, there is probably blood-deterioration. Sooner or later, indeed, blood-deterioration surely supervenes; but the influence of this does not appear necessary to produce vomiting in primiparæ in the first month.

In the second group, including cases of continuous vomiting increasing in severity, the initial conditions are those which mark the first group. But very soon another condition arises: obviously, continuous vomiting implies impaired or arrested nutrition. The influence of this seems to be to increase the irritability of the nervous centres. It is a

matter of observation that, if the strength can be roused, the susceptibility is diminished. But this is not all. The condition is not simply the negative one of want of nutrition. If food be not supplied from without, the starved system feeds upon itself. Absorption goes on actively. The proceeds of tissue-change find their way into the blood, and empoison it. At this point the danger is extreme. The blood-poison still further increases the irritability of the nervous-centres; it oppresses the brain; delirium supervenes; and utter prostration is at hand. Every fit of vomiting acts as a shock, and leaves the system more open to the next attack. At this point the slightest emotional impression, the gentlest touch on the skin, the offer of food or drink, will act as they will upon the sufferer from tetanus. At this stage diarrhoea is not uncommon. It bears further evidence of toxæmia. The indications of danger are—extreme emaciation; a pulse small, easily put out, exceeding 130 in the minute; hollow, staring eyes; hippocratic aspect; and delirium. I have not seen a patient recover in whom the last symptom had persisted for a few days, supervening on the rapid pulse. The vomiting may now subside; even premature labour may take place; but the patient will sink notwithstanding.

In some cases, and the observation applies particularly to these first two groups, the urine is albuminous. This was pointed out by Sir James Simpson. Where this complication exists, the case bears close affinity to the class of albuminuria or uræmic eclampsia. Or the albuminuria may be a secondary result of the vomiting brought about by the poisoned condition of the blood. But the fact is that this complication is exceptional, and therefore not essential.

In the third group of cases—that in which the vomiting becomes obstinate in the latter stages of pregnancy—the etiology is sometimes pretty clear. For example, I have seen cases in which, in the latter half of pregnancy, the uterus has rapidly, almost suddenly, undergone excessive distension. The cause of distension has been the undue secretion of liquor amnii or the growth of twins. At this time vomiting has set in. The explanation I would offer is this: normally, the uterus grows *pari passu* with the development of the embryo. The adaptation is so well balanced that there is no strain. But if the contents of the uterus be suddenly augmented, the harmony of correlation is destroyed. The uterus cannot suddenly grow to keep pace with the eccentric pressure within; its fibres are stretched, in some cases torn, and vomiting results. Stretching of the uterine fibre is enough to cause vomiting, even without pregnancy; *a fortiori*, it is enough when the irritability of the nervous centres is exalted by pregnancy.

In these cases I have seen the vomiting cease almost abruptly, and recovery commence from the moment the uterine tension was taken off by puncturing the membranes and emptying the uterus. In other cases, there are unmistakable evidence of blood-poisoning. Jaundice attends, sometimes preceding, sometimes apparently induced by, the vomiting. Whenever the blood is charged with matter which ought to be excreted by the liver or kidneys, vomiting is pretty sure to be aggravated. The most striking example of cholæmic vomiting is that connected with acute atrophy of the liver.

Again, I have seen cases of obstinate vomiting where a dead foetus or a diseased placenta probably supplied the poisonous element. Perfectly relates a case of obstinate vomiting for several days during the retention of the head of the foetus *in utero*. A retained placenta, or clots giving rise to the invasion of the blood by septic matter, cause vomiting. This phenomenon is, indeed, a frequent result of the absorption of septic matter during childbirth.

In a case that terminated fatally through exhaustion and irritative fever, the patient had been addicted to drinking. I shall again have occasion to refer to the pernicious influence of this habit in increasing the disposition of women to vomit.

It will now be useful to trace roughly some of the common features of resemblance or of relationship between the different forms of convulsive disease which occur in pregnancy. In this comparison or analysis we ought to include the relationship of syncope, vertigo, migraine, apoplexy, paralysis, delirium, insanity. They often form links of one chain or of different chains. Syncope and vertigo should be studied in their frequent relations to epilepsy; apoplexy in its occasional relations to uræmic eclampsia; paralysis in its relations with apoplexy and epilepsy; and insanity in its relations with epilepsy, eclampsia, and chorea. A vast field of inquiry is here before us. I scarcely dare to touch it. But I think I see enough to justify me in asserting that all these diseases, or symptoms, or pathological results, must be studied together in order that their true individual and cognate significance may be understood. Leaving aside the deeply interesting subject of puerperal insanity in its more usual forms, I may call to mind that all the convulsive diseases may culminate in mania or dementia.

What is it, then, that determines epilepsy in one case, vomiting in a

second, chorea in a third, tetanus in a fourth? We must of necessity invoke a peculiar antecedent condition of the nervous centres, lying dormant, probably quite unknown or unsuspected, until it declares itself under the magical ordeal of pregnancy. In the case of epilepsy this is almost certainly so; and this is proved by those cases in which the subject had previously been known to be epileptic herself, or to have come of epileptic parents. That pregnancy should reproduce epilepsy in those known to be predisposed to it is strong presumptive evidence that there has been a similar predisposition, although hitherto latent, in those in whom epilepsy appears for the first time during pregnancy.

In the case of chorea, this dormant morbid something is even more clearly proved. In a considerable majority of the cases in which chorea has broken out in pregnancy, the disease had existed in childhood. Pregnancy could only be regarded as a renewing cause.

But the postulate of an antecedent condition is most indisputably settled by the case of ague. We cannot conceive the possibility of ague being evolved solely out of the conditions of pregnancy. We know that other debilitating causes may also act in reproducing ague in persons who have once been under the influence of the paludal poison.

But it is not so clear that an antecedent predisposing cause is necessary to the production of eclampsia, or obstinate vomiting or tetanus. In some cases of eclampsia, at any rate, it has been impossible to trace a history of convulsive affection, or to establish hereditary taint; whilst, in many instances, the convulsion has begun and ended with one particular pregnancy, the recovery being perfect, and leaving no mark of special nervous diathesis behind. Obstinate vomiting, I believe, is more common in women of known so-called nervous or susceptible temperament; but I have not been able in some cases to make out any presumable nervous proclivity.

The influence of loss of blood in producing convulsions has often been dwelt upon, and an essential factor has been thought to be found in anæmia. I am persuaded that clinical observation will not support this hypothesis—at least, not in its general application.

The convulsive movements sometimes, but by no means constantly, observed in subjects dying from hæmorrhage, differ materially in character from the ordinary epileptic fit. The observations of animals bled to death have only a limited application. Yet it appears to be chiefly from such observations that the advocates of this theory have drawn their conclusions.

Probably few practitioners who do not practise obstetrics, and not even, it appears, many who do, have seen several cases of bleeding to death. It has been my fortune to see a considerable number. In most of them death was preceded by general tremor, a kind of universal shuddering; consciousness was sometimes not abolished, and in none was there trachelismus or congestion of the face. There was often vomiting, always distressing dyspnoea, and the pulse was rapid, scarcely felt, or even extinct. These signs constitute a picture differing essentially from convulsions.

In the following case the convulsion more nearly resembled epilepsy than usual. A woman pregnant with her eleventh child had profuse flooding from placenta prævia. Two or three convulsive fits followed the hæmorrhage. Next day she was very prostrate, but there were no more fits; no abdominal pain, but still frontal headache. On the fourth day her tongue continued colourless, and there was still intense frontal headache. She recovered. Here, possibly, there existed the epileptic diathesis.

In all these convulsive disorders the nutrition of the nervous centres is affected by some abnormal state of the blood; but the abnormal state is not the same in all. In epilepsy, in chorea, in hysteria, and, up to a certain stage, in vomiting, the alteration probably consists chiefly in that degradation which results from the impoverishment caused by excessive demand, and in the empoisonment of defective excretion. In eclampsia, supervening upon this, there is undoubtedly a special empoisonment by the elements of urine.

For the evolution of any one of these convulsive disorders, the development of a peculiar tension of the *vis nervosa* seems to be a fundamental condition; for, unless pregnancy supervene to produce this peculiar condition, neither epilepsy, eclampsia, vomiting, nor chorea, would, in most cases, ever appear.

Intimately, I might say inseparably, associated with exalted reflex irritability, is a peculiar tension or irritability of the psychical organs.

Convulsion may be determined by irritation starting either from the centripetal nerves, or from the brain.

Marshall Hall's contention that convulsion may also have a centric origin, must, I would venture to submit, be received with some qualification. It is very true that some poisons carried in the blood to the spinal cord, as well as to the rest of the body, will so exalt the irritability of the cord, that convulsions are easily determined. But it may

be questioned whether the *primum mobile*, the exciting cause of the convulsion, starts from the chord. Strychnine will enormously exalt the central irritability; but, so long as the animal is kept perfectly free from emotional or peripheral irritation, there may be no convulsion. This, Dr. Marshall Hall has shown me himself.

Strychnism resembles the excessive centric irritability of pregnancy; and, more nearly still, that greater irritability seen in tetanus. And in strict connexion with our theme, we must not forget that true tetanus is one of the convulsive diseases of pregnancy. In hot countries, as in the West Indies, in India, and in the Southern States of America, it is not uncommon in connexion with abortion and labour. Mr. Waring (*Indian Annals*, 1855), recorded 232 cases observed in India. Its occurrence has generally been thought to be sufficiently explained by comparing the condition of the uterus after labour to a surgical injury. But this, according to our view, is only a partial explanation. It accounts for the source of the peripheral irritation only.

The *tetanic state* consists in intensely exalted irritability of the spinal cord, a condition which may be regarded as a morbid exaggeration of the normally increased irritability of pregnancy. I have often seen in labour evidence of such extreme reflex and emotional irritability that I have expressed it to myself as tetanoid. But I have not seen a case of true tetanus in a pregnant woman which I could identify with what I have seen of tetanus after surgical operations, or of so-called idiopathic tetanus. Sir James Simpson (*Selected Obstetrical Works*, 1871), collected twenty-eight cases of tetanus connected with abortion or labour. In some of these there was no unusual lesion; in some there had been hæmorrhage; in some there had been plugging of the vagina to arrest hæmorrhage, and this has appeared to cause peculiar irritation. One observation made by Simpson is undoubtedly true. It is, that tetanus in women is extremely rare independently of pregnancy. Dr. Wiltshire has related (*Obstetrical Transactions*, 1872), two cases, both in pregnant women. Of the twenty-eight cases collected by Simpson, only six recovered. Both Dr. Wiltshire's died. Here I am tempted to mention a case of tetanus induced in an infant by whooping-cough. It will serve as an illustration of the influence peripheral irritation brought to bear upon the nervous centres of a child, naturally irritable, and brought to still higher tension by a morbid poison, may exert in causing convulsion.

Whooping-cough in an infant inducing tetanus.—On January 16th, 1866, I met Mr. Giles, now of Henley-on-Thames, on the case of a boy, aged nine months. He had been weaned two months, and was taking cows' milk and Robb's biscuits. He had had whooping-cough three weeks, having taken it from his mother. During the last three days a singular train of nervous symptoms appeared: first trismus, then emprosthotonos, the hands touching the feet, then the body arched back into opisthotonos. The fits seemed the first expression of the impulse to cough, the coughing coming on soon after the fit. Anything in a spoon excited cough or fit. The child was not much wasted, but there was some degree of anæmia; the bowels had been out of order, the stools pale. Calomel and rhubarb had corrected this condition. He had had belladonna. We recommended goat's milk, solution of perchloride of iron, and gave a favourable prognosis. The child recovered.

Such a case must be studied in connexion with the *trismus nascentium*. It proves very clearly the intimacy of the association between whooping-cough and convulsive diseases.

The facts related, and the propositions deduced from them, have a valuable application to the pathology of convulsive diseases. It may be said, that the relations of these diseases to pregnancy bring the accuracy of various theories as to the essential conditions of these diseases to the test of clinical experiment. Many are the theories, and discordant, of the pathogeny of epilepsy, of chorea, of tetanus, and of hysteria. Now, be these essential conditions what they may, anæmia, congestion, change of structure of the nervous centres, embolism, pregnancy must produce them all, and all must vanish suddenly with labour. Are these conditions so created and so disposed of? Great as I believe the pathogenic potency of pregnancy to be, I cannot go so far as this. We cannot, at any rate, admit that a gross structural change in the nervous centres is of the essence of epilepsy or chorea, when we see complete recovery ensue as soon as the pregnancy is over.

We are, then, driven to conclude, with Voisin and others, that the visible alterations found in the brain and cord, in persons who have died of epilepsy or chorea, are consecutive on, not antecedent to, the disease.

These structural alterations, I submit, are more strictly connected with the ulterior superadded symptoms of the disease than with the initiatory or proper symptoms. For example, when the disease has long endured, when the fits have become frequent and severe, the brain commonly shows signs of impairment, and exhaustion, paralysis, dementia, or mania is the result. In a memoir which I wrote some years

ago on Chorea in Pregnancy (*Obstetrical Transactions*, 1868), I adduced reasons for concluding that the graver symptoms—the paralysis, the mania, death—were produced by the repeated shocks of convulsion.

No one who has watched a case of puerperal convulsions, or of obstinate vomiting, or tetanus, or who has himself experienced the torture of sea-sickness, can have failed to observe how each repetition of the fit weakens the power of resistance, rendering the nervous centres more and more susceptible to those impressions which started the affection. This increasing susceptibility is obviously the result of shock, aggravated no doubt by exhaustion from want of food.

In the case of tetanus, it can hardly be questioned that the fatal prostration is almost purely the result of the repeated shocks. The blood-poison, if any exist, and I believe it does, mainly acts by increasing central nervous irritability; it scarcely complicates the problem; and the mind often remains singularly clear. In uræmic convulsions, again, notwithstanding the complication with blood-poisoning, the effect of shock is clearly seen in the exhaustion following the fit, in the general muscular resolution, in the relaxation of the sphincters, and in the gradual return of nervous power during the intervals, under the influence of rest. I have seen almost sudden death in labour which could be ascribed to no other cause than the shock of pain, and the convulsive action of the uterus. In some cases of paraplegia arising in labour it seems reasonable to attribute the paralysis to exhaustion or shock upon the spinal cord. It is certain that some of these cases cannot be accounted for on the theory of pressure upon the nerves in the pelvis.

The increasing or progressive influence of successive shocks partly explains what is comprised under the idea of "habit." "Habit" is a vague term. In the case of convulsive disease it implies—first, increased susceptibility to external and to emotional impressions; second, altered periodicity in the renewal of nerve-force. This increased susceptibility seems to be produced by repetition of shock or fit. We have one very interesting practical application of this law in obstetrics. When we induce labour prematurely in a woman for the first time we often find the nervous irritability insufficient to complete the labour promptly, and it becomes desirable to supplement defective natural power by operative aid. But in each succeeding pregnancy the nervous centres respond more and more easily to the artificial provocation, so that when labour has been brought on three or four times at the same stated period of pregnancy, it seems as if that stated time had become the normal term of gestation. Such is the altered periodicity in the preparation of the requisite nerve-force produced.

It is greatly by the influence of shock that I would account for the cerebral disorder which often attends the progress of puerperal convulsions, of epilepsy, and of chorea. The fits act as repeated shocks which stun the nervous centres. These shocks are equivalent to concussions. The *ictus epilepticus* is as real a blow as the apoplectic stroke. They exhaust and divert the nervous force, and after a time impair the nutrition of the nervous substance.

In the case of chorea proceeding to mania, we have to note that the cerebral disease is almost always *secondary and progressive*. In cases in which the chorea is evoked by fright, some mental disturbance may be noticed at the onset, whilst in other cases there may at first be little or no such disturbance. But soon irritability of temper, a certain peevishness or waywardness, a loss of balance, impairment of memory, sometimes, of articulation, follow. An aspect of stupidity betrays a real loss of intelligence. At first these defects are slight, but they gradually increase in severity, occasionally to the extent of ending in delirium or furious mania.

In cases of puerperal mania breaking out after labour, where there has been no convulsion, it may seem that some other factor than shock must be invoked. In some of these cases there is albuminuria: that is, there is blood-poisoning; and this may be taken to be the chief factor. But in other cases there is no albuminuria. But in all there is the shock of labour, with its attendant exhaustion, its severe physical and psychological revolution, acting upon a nervous system wrought up to a climax of irritability. It seems to me that convulsions, collapse, insanity, are not, indeed, interchangeable or convertible, but that the issue in any one of these conditions is determined by idiosyncrasy, or antecedent peculiarity existing in the nervous centres.

THE PRIZES OF THE PARIS FACULTY OF MEDICINE.—The Faculty of Medicine in Paris has divided the Chateaullard prize of 2,000 francs equally between Dr. Luys, for his *Researches on the Structure of the Encephalon*, and Dr. Legrand du Saulle, for his work on the *Delirium of Persecutions*. The Lacaze prize has been awarded to Dr. Pidoux, for his *General and Practical Studies of Phthisis*; and M. Lépine receives honourable mention for his treatises on *Casous Pneumonia* and on the *Unity of Phthisis*.

CLINICAL LECTURES

ON

MENTAL AND CEREBRAL DISEASES.

By J. CRICHTON BROWNE, M.D., F.R.S.E.,

Medical Director, West Riding Asylum; Lecturer on Mental Diseases to the Leeds School of Medicine; etc.

IV.—CANCER OF THE BRAIN.

GENTLEMEN,—On the table before me lies a brain presenting some unusual appearances. You see, on looking at it from above, that it is very unsymmetrical, and that there is a remarkable bulging or swelling in the middle of the left hemisphere. In this swelling are involved the whole of the convolutions of the left parietal lobe—the ascending frontal and the posterior extremities of the three tiers of frontal convolutions. These have all an expanded and flattened appearance, as if they had been pushed out and distended by some force acting from within, and together form a tumour, not well defined in front, but accurately bounded above by the median fissure, below by the horizontal limb of the fissure of Sylvius, and behind by the external parieto-occipital fissure. This tumour, which is of the size of an orange, feels firm and elastic to the touch in some places, and pulpy in others, especially in its lower third, where its surface has an eroded ulcerated appearance, and is of a reddish-grey colour. On its surface, the outlines of the convolutions involved in it are distinctly visible beneath the arachnoid and pia mater, which are much attenuated, and which cannot be traced over the eroded or ulcerated area. Apart from this tumour, there is nothing very remarkable in the brain externally. The arachnoid is not thickened nor cloudy; the pia mater strips freely; the convolutions are not wasted—on the contrary, they have everywhere a compressed or flattened appearance; there is no atheroma of vessels. On removing the upper half of the hemispheres by a section which has been made through the centre of the tumour, in a line a little below the centrum ovale of Vieussens, so that the lateral ventricles are exposed, you at once see that the tumour is somewhat rounded in shape, that it involves the whole thickness of the hemisphere, and encroaches to some extent upon the optic thalamus and corpus striatum, which are also displaced inwards, so that they obliterate the third ventricle, and are in immediate contiguity with the corresponding ganglia of the opposite side. In its centre the tumour is firm and resilient; towards its margins, however—and this is particularly the case as regards its inner and deeper margins—it is softened and broken down. Its substance is of a pinkish-white colour, and is variegated by patches of yellowish translucent glue-like matter, by dark red blotches and stains, and by a few small irregular lacunæ or cavities and open mouths of vessels. When the section is scraped, a creamy juice is collected on the scalpel—and this is especially abundant towards its outer boundaries. The left ventricle has been greatly diminished in size by the growth of the tumour, and the right has undergone a proportional dilatation, its anterior and middle horns being singularly capacious.

Beside the brain lie two lungs, and to these I must next direct your attention. Taking the right first, you notice that it is covered externally by a layer of dense fibrous tissue, the remains of some former pleurisy, by which it was firmly fastened to the thoracic wall. At its apex there is a nodule about the size of a walnut, of stony hardness, and of a greenish-black colour; and below this there is a mass of matter, of a dirty-white colour, about the size of a hen's egg; this is of rather irregular shape, and is not of uniform density. The outer portion is of cartilaginous consistence, and creaks beneath the knife, but its core is soft and disintegrated. It is not defined nor sharply demarcated from the pulmonary tissue, which is somewhat hard, consolidated, and dark coloured, wherever they come into contact. Turning to the left lung, you find that it has also an external fibrous envelope, and that at its apex there is a white calcareous nodule about the size of a bean, around which the lung-substance for the space of half an inch is black and hard. The bronchial glands connected with both lungs are enlarged, black, and of stony hardness—in these respects exactly resembling the mesenteric and the mesocolic glands, which are also submitted to your inspection.

Now, this brain, these lungs, and these glands, have been taken from the body of a male patient who died recently in this asylum. They present to you a series of pathological changes easily recognised and named. It is clear that we have here a case of malignant disease to deal with, and that the cerebral tumour, upon which I desire chiefly to fix your