THE BOWMAN LECTURE

ON

OPHTHALMOLOGY AND DISEASES OF THE NERVOUS SYSTEM.

Delivered before the Ophthalmological Society, Friday, November 13th.

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BESIDES acknowledging the honour of being asked to deliver an address before this Society, I wish to say that I consider it an additional gratification that the address is the "Bowman Lecture." Praise from me to Bowman would be impertinence. But here is my opportunity, not only for thanking Sir William Bowman for the benefit which I, like the rest of my profession, have derived from the basic work he has done, but of also tendering him my warmest personal thanks for much encouragement given me in the work I was, many years ago, trying to do. At the same time, I gratefully acknowledge that to his successor in the presidential chair (Mr. Hutchinson) I feel highly indebted for my earliest instruction in ophthalmology, as well as for great help of very many kinds for many years.

It was long ago said that division of labour, or, more generally, differentiation, is an universal law. Things become increasingly numerous and more different, or, in other words, more complex. Differentiation is well seen in the development of animal organisms, and is seen, too, in the social organism. It would be very remarkable if there were an exception in the case of one part of the social organism, the body medical—if in so great a field of work as the medical there did not arise more and more different workers in different parts of that field. There is no exception. The body medical is now very complex; there are alienist physicians, obstetric physicians, neurologists, ophthalmic surgeons, aural surgeons, dentists, physiologists, chemists, etc.; the speciality of each comes out of, is a differentiated part of, a wide general knowledge.

Specialists have to justify themselves—to justify their differentiation. Differentiation is not the whole of the modern doctrine of evolution. The factors in progressing evolution, according to Spencer, are increasing (1) differentiation, increasing (2) definiteness, increasing (3) integration, and increasing (4) co-operation.¹

Increasing differentiation without increasing definiteness would be only confusion. That the ophthalmic surgeon has justified himself in the second factor of evolution needs no proof. I will instance, however, his highly definite work on paralyses of ocular muscles and on abnormalities of refraction. In neurology, I may instance the work of Charcot. And here I must say, as the examples are intended to imply, that by definiteness I mean definiteness which connotes exactness.

Now for the third factor. Each different definite worker is working for the whole body medical; his work leads to the greater integration of medical knowledge of other workers. Each different worker helps all the others. Many different workers in the medical field are seriously indebted to the ophthalmic surgeon in this way. To give but one example, and making again an arbitrary limit. Argyll Robertson has given to neurologists, in the pupil-symptom which is called by his name, not merely a new symptom, but a means of investigation of several important diseases. This debt, neurologists, Westphal and Erb, working in a different field, have paid back in showing the diagnostic value of loss of the knee-jerk.

There is yet another factor in evolution—increasing co-operation. Each different worker, knowing one subject best, and having great integration of different, definite medical knowledge, of necessity cannot have the precise knowledge of other subjects which other different workers have. Division of labour necessitates the co-operation of labourers. The whole of one disease is better understood by bringing to bear on its direct investigation and treatment different workers in different fields. To give but one illustration of the need of integration of different kinds of definite medical knowledge, and yet of the need, too, of co-operation of workers; the neurologist ought, at

least, to be able to suspect hypermetropia as a cause of head-troubles, but only the skilled ophthalmic surgeon can estimate it precisely, and correct it accurately.

I think we often err by underating the complexity of some nervous diseases. I take epileptic paroxysms and their after-conditions as being, together, an illustration of the morbid nervous affection of greatest complexity of symptoms. When its symptomatology is fully displayed, it shows, I submit, the imperative necessity of (1) different, (2) definite, (3) wide knowledge, and of (4) co-operative work. I speak of the "genuine epilepsy" of nosologists, not of epileptiform seizures. Epilepsy is a disease of the "organ of mind"—that is to say, of the highest and most complex, etc., centres. The symptomatology of the paroxysm is probably an universal symptomatology, is demonstrably nearly so. It may be said that insanity is more complex than epilepsy; not so, since epileptic paroxysms are not unfrequently followed by temporary insanity. The study of epilepsy, therefore, involves the study of some cases of insanity. These insanities are what I have called the after-conditions of the epileptic paroxysms. We ought, of course, to speak of epilepsies as we should do of insanities; for certainly, if two epileptics have different "warnings" of their fits, the discharging lesions" are in different parts of the highest centres in the two cases. There are many epilepsies, and many insanities. But now I give *en bloc* the symptoms of many severe epileptic fits, of many slight fits, and the symptoms of epileptic fits (or, I should say, epileptiform seizures), artificially produced in dogs; all are, at any rate, symptoms produced by cortical discharges. Among them are very important eye-symptoms.

At the climax of a severe fit, we see the algebraical sums of the co-operations and antagonisms of strongly developed movements of all the muscles of animal life accessible to observation—universal convulsion. As to wide involvement of sensory elements in the epileptic discharge, we can say that this is implied by "warnings" of crude and excessive smells, colours, sounds, tastes, by tinglings, etc., of the hands, and by many crude and excessive systemic sensations. Of what happens in the sensory sphere after loss of consciousness, we can say nothing. Besides colour-development, there is another eyesymptom—a vertigo—in which external objects seem to move, implying discharge, direct or indirect, of motor cerebral elements representing ocular movements. We find dilatation of the pupils, perhaps, sometimes contraction; there are great pallor of the face, increased flow of saliva, perspiration, alterations of pulse and respiration, passage of urine and feces, and erection of the penis.

Vulpian curarised a dog, thus putting the muscles of animal life out of the reach of an artificially induced discharge of the dog's left sigmoid gyrus (artificial respiration). Speaking almost only of effects of cortical discharges, which I have not yet mentioned, there were slowing and irregularity of the heart, raised arterial pressure, increased flow of bile, a little paling of the kidneys, and diminution of their secretion, contraction of the bladder, and contraction of the spleen.

So we go a long way towards showing that the epileptic paroxysm has an universal symptomatology, having shown that sensations are referred to many different parts of the body, and that effects by efferent (motor and inhibitory) nerves are produced in different ways in very numerous parts, "from the eyes to the feet" (strictly, I suppose, "from nose to feet"). We can add to the list of parts represented by the cerebral cortex.

"RocheFontaine and Lépine, on stimulating several points, especially in the neighbourhood of the sulcus cruciatus in the dog, observed increased secretion of saliva, slowing of movements of the stomach, peristalsis of the intestine, contraction of the spleen, of the uterus, of the bladder, and increased respiration." (Landois' *Physiology*, vol. ii, p. 942, Stirling's Translation). Bufalini has observed the secretion of gastric juice.

I do not think I need apologise for speaking in this lecture of the non-ophthalmological symptoms of epileptic paroxysms, especially of those of the organic parts. The neurologist and the alienist physician must take into account all the symptoms. They are an important part of the evidence towards showing that the "organ of mind" (highest centres) represents or co-ordinates all parts of the body. Unless retinal impressions corresponding to colour of objects, and ocular movements corresponding to shape of objects, are represented in the highest centres, how are we to account for the physical bases of visual ideation? If the organic parts are not represented in the highest centres, an emotional manifestation—say, of fear—is unaccountable; and so, too, are the physical symptoms of some cases of melancholia, the insanity of fear (anxiety, depression, etc., being only "fear spread out thin"). Whilst a man is thinking, or even dreaming, of a brick, he is having a purely psychological state; the correlative physical

¹ I am using terms more familiar to medical men than those Spencer uses; for this change, of course, Spencer is not answerable, nor must he be held responsible for my applications of his formula of evolution. I should consider it a great calamity were any crudities of mine imputed to a man to whom I feel profoundly indebted.

state is discharge of some nervous arrangements of his highest centres representing parts of his body—certain retinal impressions and particular ocular movements. Again, when a man is afraid, he is having a purely psychical state; the correlative physical state is discharge of some nervous arrangements of his highest centres, representing parts of his body—notably, the organic parts. There are clinical reasons why the ophthalmic surgeon should be interested in symptoms in organic parts. Often enough, in other diseases of the cerebrum, does he see effects produced in organic parts. To say nothing of vomiting, common with optic neuritis from cerebral tumour, there are acute stages in such cases, in which there are alterations of pulse and respiration, retracted belly, and constipation. Consider also the migrainous paroxysm, which is no doubt owing to a cortical discharge, often beginning by elaborate visual projections, and often ending by vomiting. Eye-symptoms from disease of the cerebrum cannot, in many cases, be studied isolatedly from organic symptoms.

We have not yet done with the complexity of the epileptic paroxysm. We must note, in slight fits, movements of chewing or tasting, swallowing, vomiting, and writhing movements of the arms during arrest of respiration. I do not believe that these movements result directly from the epileptic discharge, holding that such a discharge, so far as it spreads, puts an end to all movements properly so called, "runs them up" into convulsion. I suggest that an important eye-symptom at, or near to, the onset of some epileptic fits, the apparent alteration in size or distance of external objects, is, on the physical side, a phenomenon of the same order as the chewing movements; that both are the indirect (reflex) results of epileptic discharges of sensory elements. Ferrier produced, by faradising a monkey's "taste-centre," movements like the chewing and tasting movements which some epileptics or their friends describe. It is exceedingly important to distinguish movements proper from convulsion, which is a contention of many movements. I suppose everyone would believe that spitting, rubbing one hand with the other, clutching at the throat (in rapid suffocation!), are movements too elaborate to result from such a discharge as that which produces convulsion. There is yet more complexity; when, in severe fits, respiration is arrested, there is carbonic acid poisoning. Asphyxia renders a dog's cerebral cortex unexcitable (Hitzig, Franck and Pitres); but some other lower centres, or parts they supply, or both, are stimulated; thus there will be a multiplication of effects. Asphyxia produces dilatation of the pupils, besides other effects (salivation, sweating, etc.) But we must bear in mind that the centres which are directly stimulated by carbonic acid, or overacting from lack of oxygen in simple asphyxia, will be, more or fewer of them, to some degree, in an epileptic paroxysm already engaged by the epileptic discharge; and that, when the fit is over, there will be a degree of exhaustion of them corresponding to the degree of prior discharge, and so far an unsusceptibility to be acted on. The pupils are small after severe epileptic fits.

Now for the mental symptoms of, or rather during, the discharge beginning in part of the "organ of mind." During the epileptic discharge, there is defect or cessation of consciousness. Some think that also during it, at the outset of the fit, there arise the exactly opposite mental states of "overconsciousness," "dreamy state," alterations in size and distance of external objects, "seeing faces" and "hearing voices." I believe these superpositive mental states, and, adding to the list, fear—different miniature insanities—at the outset of different epileptic attacks, to arise during slightly raised discharges of healthy nervous arrangements untouched by the epileptic discharge.

We must be very careful, both in ophthalmological and neurological studies, not to confound sensations—colours, for an example—which are states of mind, with activities of sensory elements, which are states of body. We must not speak of any mental states as occurring *from*, but as arising *during*, nervous discharges. Only physical effects, such as movements, arise *from* nervous discharges. We must also distinguish different degrees of elaborateness of different mental states. "Seeing faces" is a greatly more elaborate mental state than colour-projections at the onset of an epileptic fit. What I have called the "dreamy state" is vastly more elaborate than "seeing faces." Whilst I think that colour-projections, at the onset of an epileptic fit, arise *during* the epileptic discharge, I believe that so elaborate a state as "seeing faces," and the still more elaborate mental state, the "dreamy state," do not, but that they arise *during* discharges only slightly stronger than normal. The "dreamy state" is a very voluminous mental state; it is commonly called an "intellectual aura." As I have implicitly said, I do not believe it to be an aura or warning; not a thing, I mean, of the same meaning as the crude sensations of colour, etc. Admitting that crude sensations arise *during*, and that convulsions occur *from*, the epileptic discharge,

I urge that elaborate mental states arise *during*, and that movements, properly so called, occur *from*, but slightly raised discharges of nervous arrangements.

[The lecturer then spoke of the different association of different sense-warnings in epilepsy, believing that the "subjective" sensations, smell, taste (or chewing, etc., movements) and the "epigastric" sensation most often occur in those cases of epilepsy, in which there is the "dreamy state;" and that the cortical lesions in these epilepsies are in parts of the cortex in the region of the posterior cerebral artery, which vessel supplies, among other parts, Ferrier's centres for smell and taste. He suggested that some cases of epilepsy, with colour and sound warnings (with which he had never known the "dreamy state" to be associated) were owing to cortical disease in some part of the region of the middle cerebral artery, which vessel supplies, among other parts, Ferrier's centres for sight and hearing. He considered that the nervous changes (the "discharging lesion") in epilepsies were not primarily, but only secondarily nervous; that in most cases they were secondary to embolism or thrombosis of small arterial branches; that there were "arterial cortical lesions" in most epilepsies. He had held that some epileptiform seizures have that pathology since 1864 (*London Hospital Reports*, vol. i, p. 465), and considered that some cases recently published by different physicians proved that hypothesis. The researches of Duret and Heubner, on the detailed arterial supply of parts of the brain, will enable us to be more definite in our arterio-cortical localisations of "discharging lesions," especially in epileptiform seizures; some cases of this kind were evidently owing to blocking of branches of the middle cerebral. Another way of putting part of the foregoing, is to say that epileptiform seizures do not always depend on tumour. This brings us again close to ophthalmology. If anyone is thinking of removing a brain-tumour in a case of epileptiform seizures, he ought to refrain from operating, if he does not find optic neuritis, perhaps with one exception. Of course there are epileptiform seizures from brain-tumour, in which (for years at least) there is no optic neuritis, but without this eye condition we cannot (with perhaps one exception) be sure of tumour. It was from both neurological and ophthalmological knowledge that Dr. Hughes Bennett made the double diagnosis of seat and nature of the disease in the case of tumour which Mr. Godlee removed from the patient's brain.

The lecturer then spoke of certain important cases of epilepsy, complicated with some particular definite and persisting eye-symptoms, pointing to intracranial tumour; the investigation, by the ophthalmic surgeon, of paroxysms of epilepsy so complicated is most desirable. A man had occasionally smells in his nose, heralding in slight attacks of epilepsy with the "dreamy state;" there were left-sided "shakings" in the paroxysms. There was double optic neuritis, but no defect of sight; the neuritis passed off under mercurial inunction and iodides, sight remaining good. The patient died, apoplectic, with left hemiplegia; his case illustrates the dictum that a man with optic neuritis is to be considered as being in imminent danger of death; that this patient was in any such danger, no one would have surmised who did not find out that there was optic neuritis. Whilst the optic neuritis is the best indication for treatment, and of most prognostic value, such an important variety of epilepsy as this patient had deserves most careful investigation.

In this association, the lecturer urged that the great thing in the diagnosis of epilepsy is not so much the "quantity or severity of the symptoms" as their paroxysmalness; and that the slightest a paroxysm, the more necessary, both for the patient's sake and for medical science, is its minute investigation. Attacks of epilepsy with the "dreamy state," when very slight, as they often are, are sometimes attributed to hysteria, or to stomach and liver derangement. To the uninstructed, the accounts given by the patients seem "fanciful." That optic neuritis is often overlooked in its pre-amaurotic, most curable state, is certain. The case mentioned was the only case of epilepsy with "dreamy state" the lecturer had seen complicated with optic neuritis. There was no necropsy. The presumption is, that death occurred by hæmorrhage from a vascular tumour in the right cerebral hemisphere. Another case of this kind of epilepsy (bitter taste, "dreamy state," and right-sided numbness), investigated by Dr. James Anderson, was complicated by simple atrophy of the optic nerves; there was loss of sight of the left eye, of the right field of the right eye, almost loss of smell in the left side, defect of taste more on the left side (the paroxysmal one-sided symptom (numbness) was right-sided, and the patient was not left-handed; in most cases of this variety of epilepsy, the lecturer thinks the one-sided phenomena, if any, are left-sided). Having regard to an important case recorded by Mr. Nettleship (*Transactions of the Ophthalmological Society*, vol. iv, p. 285) like this in the optic symptoms (necropsy by Dr. Sharkey), in which

there were slight epileptic fits, with a feeling of suffocation referred to the nose and mouth, the lecturer thought that in Dr. Anderson's patient's case, there was a basal tumour causing the epileptic fits by involving, or by in some way inducing changes of instability in the left middle cerebral lobe. Cases with such fixing-eye symptoms, implying gross organic disease, for obvious reasons deserve most careful and minute investigation, not only of the eye-symptoms, but of the epileptic fits also.]

Speaking now of the after-conditions of epileptic paroxysms very generally, we have insanity, according to the severity of the fits, in two degrees at least,—in three, I think. There is so-called "loss" of consciousness with actions (postepileptic "unconsciousness" with mania, for example), and after very severe paroxysms, acute dementia (coma). Here I remark that it is vain to attempt the realistic study of epilepsy and its after-conditions, or that of any other insanity, without psychological knowledge. Confounding psychology with physiology of the highest centres leads to crude metaphysical explanations, such as that in postepileptic coma a man does not move because he is unconscious; although, in a slighter postepileptic condition, epileptic mania, there is movement enough, when, according to the opinions of most, consciousness is lost. Taking postepileptic coma after a very severe fit, there is exhaustion of more or less of the highest centres implied by the negative affections of consciousness, and, in some slighter degree, of more or fewer of the lower (more organised) centres. Westphal and Gowers have noted transitory absence of the knee-jerks after epileptic paroxysms, thus showing there to be sometimes exhaustion of lumbar nuclei, some lowest motor centres. Deep post-epileptic coma is, psychically, dementia; but is, on the physical side, nothing else than some universal, almost total, paralysis—paralysis, not only of animal, but of organic parts also—proportionate to the degree of the prior epileptic discharge upon them.² This contention of mine is, however, denied; let us say, what cannot be denied, that the patient is nearly dead. I dare say my calling the psychical side of the condition insanity (dementia or amentia) will be objected to. Let us say that the patient is, or is nearly, mentally dead; this cannot be denied. Moreover, the patient is suffering from carbonic acid poisoning, from slow respiration, from imperfect circulation; thus there will be a multiplication of effects. Possibly the excess of carbonic acid helps somewhat in re-evolution, by strongly stimulating the respiratory and other partially exhausted (lowest) centres.

There is one after-effect which is certainly paralytic—transitory lateral deviation of the eyes, observed by Beevor instantly after severe attacks. Again, the exaggerated knee-jerks and foot-clonus after some fits (Beevor) imply paralysis, signify exhaustion of fibres in the lateral columns, and possibly also of inhibitory centres, in the cord itself (Gowers). Here we have after-effects at the two extremes—eyes (paralytic), feet (implying some paralysis); it would be remarkable if the rest of the symptoms were not paralytic.

I submit that it would not be possible for a neurologist to scientifically study this exceedingly complex disease without availing himself of the work done by different specialists. We require many different kinds of definite or technical knowledge; all the sagacity in the world will not avail, either for practical ends, or for the scientific investigation of complex problems, without technical knowledge. No one man can have, from his own working, enough different, definite knowledge to thoroughly investigate the universal symptomatology of epilepsy.

It is vain to begin the careful analysis of this complex problem without aid from psychology. Could the neurologist analyse the physical condition without help from the physiologist? How, without that help, is he to put in any reasonable order symptoms related to the organic parts, slowing of the heart, raised arterial pressure, and the rest? Recent researches by Gaskell will, I think, help us greatly in our analysis of the organic symptoms. I submit the hypothesis that, in some cases, the first effects of the epileptic discharge on the organic parts are by intermediation of Gaskell's leucocentric (inhibitory) fibres. Does not the neurologist need the help of the alienist physician? Witness the definite work on epileptic insanity by Falret. To the ophthalmic surgeon the neurologist is very directly indebted. Here I will pick the eye-symptoms out of the heap.

Sometimes, but yet very rarely, we have optic neuritis (common in epileptiform seizures), or simple optic atrophy, as I have illustrated. To speak only of the paroxysm; in some cases there are colour-warnings, occasionally followed by the greatly more elaborate mental state, "seeing faces." There are pupillary affections. In one case

of uræmic convulsions I saw the fundus easily during the fits, when the pupils were widely dilated, but could get no glimpse of it in the intervals. Vulpian found that the pupils in his curarised dog enlarged during the artificially induced paroxysm, and that, after the fit, they were smaller than before the fit. We have vertigo at the onset of some paroxysms. Vertigo is consciousness ceasing; that variety of it in which objects seem to move to one side is an eye-symptom; it implies discharge, primary or secondary, of centres representing particularly the most special of ocular movements. If we have not clear ideas on vertigo in epilepsy, we shall work sad havoc in the investigation of some important varieties of epilepsy. Here the neurologist should avail himself of the definite knowledge the ophthalmic surgeon can give him from cases of paralyses of ocular muscles, in which vertigo is seen in its simplest form, before he studies it in the vastly complex circumstances of the epileptic paroxysm. After that, he should take heed of what the aural surgeon can tell him of ear-vertigo. I have seen, so to speak, vertigo during an attack of ear-vertigo. Speaking more correctly, I saw the patient's eyes jerk to one side whilst he saw objects jerking to one side (*Brain*, April, 1879). In our *Transactions*, vol. iii, I have recorded a case (essentially like one previously published by Schwabach) in which pressure on a diseased right ear produced vertigo and movements of the eyeballs to one side. Such cases are very valuable in the interpretation of vertigo. The variety of vertigo mentioned occurs, no doubt, in an epileptic fit along with turning of the eyes to one side. Beevor has noted that, after some epileptic fits, the eyes deviate from the side to which they were strongly turned in the prior paroxysm. This is a matter of great importance. That movements of the eyes are represented in the highest motor centres (frontal lobes, motor division of the "organ of mind"), one might infer *a priori*. Ocular movements are the most representative of all movements. Most mentation is carried on in visual perceptions and ideas; and, as there is an element of, or symbolising shape, in every visual perception and idea, there is, of necessity, a representation of ocular movements in the physical bases of these ideas, that is, in the highest centres.

Ferrier and Gerald Yeo find, by experiments (ablations) on monkeys, that movements of the eyes and head are represented in the frontal lobes. There is another important eye-symptom in epilepsy; apparent alteration in the size and distance of objects. It occurs, I think, most often in those epileptic attacks in which there is the "dreamy" state. It is physically a "motor affair;" that size and shape are not simply "retinal affairs" is easily proven. Here we avail ourselves of the definite knowledge of the ophthalmic surgeon (the micropia after instillation of atropine; in paralysis of an internal rectus; the opposite condition after eserine). But we must not apply the knowledge derived from such simple cases to alterations in size of external objects at the onset of epileptic fits without great caution, the situation being an exceedingly complicated one. I have already stated an hypothesis regarding the mode of production of these phenomena. Dr. Gowers has considered them, with his characteristic ability, in his valuable work *On Epilepsy*, page 64, a work rich in ophthalmological knowledge.

Although I have spoken of epilepsy, there are really very many epilepsies, just as there are many epileptiform seizures, and thus, illustrating by the eye-symptoms (vertigo, colour, alteration of size or distance of objects), just mentioned, we should try to find the particular sequence of symptoms of other kinds of which each of the eye-symptoms is the prelude. Epilepsy is not a complex thing when it is empirically regarded as a clinical entity, which for some purposes it should be. It is comparatively easy to note the "causes," "warnings," etc., "of epilepsy;" much good work has been done in that way. But, besides investigating in this way, we have to "turn ourselves round," and try to ascertain what different symptoms result from differently seated "discharging lesions" of the highest centres. We have the large question to answer, what is the constitution of the "organ of mind" (highest centres) by which it results (1) that discharge, beginning in different parts of it, produces different epilepsies, and produces, if severe enough, nearly universal effects, no doubt in different degrees and sequences; whilst (2) other kinds of disease of different parts of the "organ of mind" produce different insanities. (3) In one case we may have first, in the paroxysm, cessation of consciousness with universal convulsion and equivalent effects in the organic field, and then, after the paroxysm, insanity, acute temporary dementia (coma), or "loss" of consciousness, with mania. After epileptic fits of different degrees of severity we have different degrees of insanity, from "confusion of thought" to coma. Indeed, some physicians go so far as to say that an attack of mania sometimes occurs instead of an epileptic convulsion; as I have implicitly said when

² Of course, we cannot say that such superpositive phenomena as foot-clonus, passage of faeces, etc., after epileptic fits, are paralytic, but they signify exhaustion of "controlling" nervous elements, are indirect evidences of paralysis.

speaking of movements properly so-called, and of elaborate mental states, I do not hold this hypothesis. (4) We have to find a means of studying epilepsies and insanities (diseases of highest centres) with diseases of lower centres.

It is impossible even to begin to work methodically towards answering the compound question put, if we confuse psychology with the anatomy and physiology of the nervous system; manifestly, referring to (4), we cannot reasonably compare and contrast loss of consciousness from disease of the highest centres with monoplegia from disease of the middle centre, nor with any other physical symptom.

We should follow the method of science, and investigate by the use of hypotheses. This may seem a strange remark to those who erroneously suppose an hypothesis to be a conclusion in which we may rest. It is only used for the methodising of work by observation and experiment. I submit that we should adopt the hypothesis of evolution, according to which the whole nervous system is a sensori-motor system representing all parts of the body. I suggest that all parts of the body are represented in each of three levels of evolution (representative, re-representative, and re-re-representative). Strictly, we should speak of four levels; as, when tracing the "ascent" (*vide infra*) from eye-muscles to most complex ocular movements, we should begin at the periphery. Representation increases in (1) differentiation (complexity), (2) definiteness (speciality), (3) integration (intricacy), and (4) in number of interconnections (co-operation) from lowest centres to highest centres ("organ of mind"). The "organ of mind" is nothing else than a series of centres representing, or, what is the same thing, co-ordinating, all parts of the body, "from eyes to feet," in greatest complexity, etc. Let me compare and contrast the lowest level of evolution with the highest, ignoring the middle level. (The lowest level is cerebro-cerebellar.) Each lowest centre represents, most nearly directly, some (3) limited particular region of the body (least integration), each some region in (1) fewest least different, and (2) least definite combinations; there are (4) fewest interconnections between these centres. The supposition is that each of the highest centres represents triply indirectly (through middle and lowest centres) (3) all, or very wide, regions of the body; that each represents in (1) most numerous, most different, and (2) most definite combinations; and that there are (4) most numerous interconnections between these centres. (Each highest centre represents all parts, or wide regions of the body; no two represent all parts, or the same parts, in the same way.) The evolutionist, it must be added, does not attempt the marvellous feat of "getting the mind out of the body;" he only tries "to get" the physical bases of mind (highest centres) out of the rest of the body. Mental states are only concomitant with nervous states. Taking mental symptoms to be only signs of what is not going on, or, of what is going on wrong, in the highest parts of a great sensori-motor mechanism, we may, I hope, find an answer to the large compound question put. We cannot expect to find an answer if we are not thoroughly materialistic as to what is purely material, the nervous system.

Taking an ophthalmological illustration, and yet artificially simplifying it by considering only the first factor in evolution, we might try to trace an ascending complexity of representation from the nerve-supply to ocular muscles, up to representation of ocular muscles in exceedingly complex movements in the physical bases of visual ideas and other mental states. We should use the "experiments" which disease makes (dissolutions) in endeavouring to trace that "ascent": 1, paralysis of ocular muscles from lesions of their nerve-trunks (periphery); 2, ophthalmoplegia externa and interna (lowest motor centres); 3, lateral deviations of the eyes in lesions, negative and positive, of the middle motor centres, or of a plexus (internal capsule) just below them (Vulpian and Prevost). In some limited epileptiform seizures, we see development of movements of the eyes in very important associations. Possibly the interesting and remarkable paralyzes of particular ocular movements, which Priestley Smith has described, are monoplegias owing to negative lesions of some middle centres; 4, deviations of the eyes in and after epileptic fits; these are, I presume, developments and losses of the most complex movements which are represented in the physical bases of visual ideas and other mental states. These "experiments" are very rough, but some of them can be supplemented by the very definite experiments, properly so called, of Hitzig and Ferrier. I refer again to the proof which Ferrier and Gerald Yeo have given of the representation of ocular movements, or of the most special of them, in the frontal lobes; these parts of the "organ of mind" I call highest motor centres.

It may be said that such a wide way of studying disease tends to vagueness. I think we may guard ourselves against this.

In the epileptic paroxysm (dissolution being effected), the universal symptomatology is suddenly presented, and is transitory. But whilst working at this disease of the highest level of evolution, and at insanities, other diseases of it, we may, at the same time, work at different diseases of the lowest level (ignoring the middle in this illustration), in which, taking numerous different cases, the symptomatology is of nearly, if not of all, parts of the body. I remind you that the hypothesis is, that the lowest level of evolution represents all parts of the body; that the middle level is the lowest over again, and so to speak, "raised to a higher power;" that the highest level is the middle over again, "raised to a still higher power."

In different progressive muscular atrophies we have symptoms nearly, if not quite, all along the lowest level, from ophthalmoplegia externa downwards (some forms of "bulbar paralysis," the common variety, Duchenne-Aran, etc.), "from eyes to feet."

Here is an important ophthalmological matter. It is well known that, with destructive lesions of the brachial plexus, we have smallness of the pupil on the side of the injury. Ferrier finds that, in the monkey, and presumably it is so in man, the dilator fibres of the iris, contained in the cervical sympathetic, are derived from the anterior root of the second dorsal nerve. In the monkey, the second dorsal sends a communicating branch to the brachial plexus, and so it does in most cases in man (Cunningham). Now the second dorsal root (Ferrier) supplies also the intrinsic muscles of the hand. In one case I have observed a small pupil (inability to dilate when shaded) in a case of progressive muscular atrophy, at a stage when the hand-muscles were almost solely those atrophic. Here again the neurologist has common ground with the ophthalmic surgeon. So much for atrophy of the animal parts. The separation is, of course, not absolute; bulbar paralysis is evidently a morbid affection of parts, mixed in function, or rather, having both kinds of function.

I submit, quite hypothetically, that we have atrophies of cells of centres for organic parts, like the atrophies of cells of anterior horns, and their higher homologues, which produce progressive muscular atrophies; thus I submit that (pernicious) diabetes may be a nuclear atrophy, progressing atrophy of cells of that part of the great vaso-motor centre which especially governs the hepatic artery. I would venture to suggest that Graves' disease is of the same kind of central pathology. (Similarly, *mutatis mutandis*, for myxoedema; central atrophic changes leading to atrophy of the thyroid. Dr. Ord says that marked bulbar paralysis has been found in two cases.)

In Graves' disease, we have an important eye-symptom—that, on the patient's looking down, the lid does not follow the globe; perhaps, by this symptom, we may hope some time to fix the seat of the changes in the centres in cases of Graves' disease. Warner and Bristowe have recorded a case of Graves' disease in which there was a form of progressive muscular atrophy, ophthalmoplegia externa. This case, so complicated, renders my hypothesis a little plausible. Of course, I do not mean that all the superpositive phenomena in Graves' disease are the direct consequences of atrophy of cells. The engorgement of the thyroid body may be owing to such atrophy of some part of the great vaso-motor centre. Some other symptoms, for example, the palpitations, may be indirect consequences of atrophy of cells of inhibitory centres. There is no reason why atrophy should not begin in cells of Clarke's column and its higher and lower representatives, and in spinal and bulbar "regulating centres."

In tabes dorsalis, we have, taking numerous different cases, symptoms "all along the line," from optic atrophy and pupillary affections downwards, "from eyes to feet." In these cases, we have morbid affections of many organic parts, among others, gastric crises (Charcot, Buzzard, etc.), bladder-troubles, and impotence; sometimes loss of smell and hearing, but the most important sensation-defect is of resistance, a defect clearly seen in cases where there is the feeling of "padding" of the soles.

Diphtherial paralysis is another disease on the lowest level of evolution, probably (Vulpian, Déjérine, Abercrombie, Percy Kidd) a myelitis of parts of anterior spinal horns and of their higher homologues (possibly, the ptomaine generated during the diphtheria acts, somewhat like curare and poisons endings of motor nerves). In this disease, we have, in severe cases, paralyzes from eye (ciliary muscle) downwards—"from eyes to feet." We must not forget the occasional implication of the organic parts—death after slowing of the pulse (implication of the accelerating centre?).

We may study the nearly universal symptomatology of different kinds of disease on the lowest level of evolution made up by the symptoms of the cases mentioned and by those of other diseases on that level. The ophthalmic surgeon sees eye-symptoms from lesions at or near to the "top" of the lowest level of

² I use numbers to indicate the particular factors of evolution previously stated. It is convenient here to state the factors in different order.

evolution, and in cases which, superficially regarded, are uncomplicated. But having great integration of medical knowledge of other kinds, he does not regard them as eye-diseases only, but finds what they mean, what wide symptomatology they are important elements of. By co-operation of many different workers, each with definite or special knowledge of particular kinds, and each with great integration of more general knowledge, we may hope to thoroughly analyse three very complex symptomatologies. The symptoms are slowly produced, are presented in detail, are persistent or permanent, and, so to speak, are comparatively simple analysing experiments by disease on the lowest level of evolution (simple dissolutions). But now regarding these symptoms taken *en bloc* as signs of negative lesions of (nearly) all parts on the lowest level of evolution, let me compare and contrast them with symptoms from negative lesions on the highest level.

The condition of a patient after a severe epileptic fit is one of nearly universal, and almost total, paralysis (dissolution effected). He has loss of consciousness, but this has nothing to do with his not moving. The symptomatology is, so to speak, the "evolutionary sum" of all, or nearly all, the symptoms of all the diseases on the lowest level of evolution. This statement needs obvious qualifications; one is important. Since the epileptic discharge, beginning in the highest centres, produces effects in nearly all (onto- and epi-) peripheral parts, currents from this primary discharge must have traversed and discharged more or less of the middle and lowest centres, in order to "get at" the periphery. Hence the paralytic condition after a very severe fit will be a very compound one, a very compound dissolution, all orders of centres being somewhat exhausted, but in different degrees.

Thus the neurologist, by availing himself of the different definite work which ophthalmic surgeons, aural surgeons, laryngologists, etc., are doing at different points all along the (1) lowest level of evolution, by working himself at different diseases on the (2) middle level (epileptiform seizures, monoplegias, migraine, etc.), by working with the alienist physician at diseases of the (3) highest centres (epilepsies and insanities) may hope to justify his differentiation. The several workers may hope to add (1) different (2) definite knowledge to, and further the (3) integration of general medical knowledge, and to lead to a higher and more methodical (4) co-operation of different workers. I say once more that this wide comparative study cannot be methodically undertaken, if we confuse psychology with the anatomy and physiology of the nervous system—not if we take the *organ* of mind to be mind. The "*organ of mind*" is simply a series of centres re-representing impressions and movements of all, literally all, parts of the body.

LACTIC ACID AND TUBERCULAR LARYNGITIS.—A discussion has taken place in the Berlin Medical Society on the value of lactic acid in laryngeal tuberculosis. Dr. Krause stated that, of fourteen undoubted cases he had treated in this way, some were improved, and some completely cured. The least satisfactory cases were those where the posterior wall of the larynx was affected; also those where there was a lesion below the vocal cords which could not be well brought under the influence of the application. The voice improved, also the power of swallowing, and the general condition of the patients. Dr. Rosenberg confirmed the statements of Dr. Krause as to the good effects of lactic acid, but, for his own part, was still more satisfied with the results of a twenty per cent. solution of menthol. He was also treating pulmonary phthisis with menthol-inhalations. Dr. Lublinski had had a certain amount of success with lactic acid, but found that, after the ulcers had healed, they again broke out. Professor Virchow pointed out that when these ulcerations healed, a cicatrix only was formed, not true mucous membrane. He hoped that further observations on this subject might be made, especially on cases that had healed for some time.

WEST KENT MEDICO-CHIRURGICAL SOCIETY.—The first meeting of the thirtieth session was held on Friday, November 6th, when the following officers were elected. *President:* Robert E. Carrington, M.D. *Vice-Presidents:* C. H. Hart, L.R.C.S.I.; F. B. Jessett, F.R.C.S. *Treasurer:* Prior Purvis, M.D. *Secretary:* H. W. Roberts, M.R.C.S. *Librarian:* J. B. Saundry, M.D. *Council:* J. Brindley James, M.R.C.S.; W. C. S. Burney, L.K.Q.C.P.; H. G. Cable, M.R.C.S.; W. Collingridge, M.D.; A. Forsyth, M.D.; John Marshall, M.R.C.S.; Frederick Moon, M.B.

RUSSIAN PROFESSORSHIPS.—Dr. A. Bruyeff, of the Military Medical Academy of St. Petersburg, has been appointed Professor of Syphilography and Dermatology in the University of Kharkoff. Professor Danilevski, of Kazan, has been appointed to the Chair of Physiological Chemistry in Kharkoff.

INAUGURAL ADDRESS ON MEDICAL TREATMENT.

Delivered before the Midland Medical Society, November 11th, 1885.

By SAMUEL WILKS, M.D., LL.D., F.R.S.,
Consulting Physician to Guy's Hospital, etc.

Slow Progress of Disease.—*Duty of the Medical Man as regards the Study of its Causes and its Prevention.*—*The Voice of the People as the Medical Man's Guide.*—*The Study of Disease and its Causes the true Foundation of Medical Practice.*—*Importance of Studying the Natural History of Disease.*—*Origin of Treatment by Drugs and other Agents.*—*Therapeutics as a Science.*—*Limitation of Value of Observations on Healthy Subjects.*—*Influence of Fashion on Modern Changes in Treatment.*—*Multiplicity of Medicines undesirable.*—*Specialism.*—*Increased Closeness of Relations between Medicine and Surgery.*—*Antisepsis.*—*Summary.*

[Continued from page 903.]

Influence of Fashion on Modern Changes in Treatment.—The difference of treatment from a former age is due, partly to an advance, but more to a change in fashion. Then it was thought that disease implied some over-activity in the part of the body affected, and it was to be knocked down by depleting measures—such as bleeding, general or local, assisted by medicines of the nature of purges, emetics, and salines, combined with low diet. Now, the theory of modern medicine can be best exemplified by turning to the advertising pages of the medical journals, in which you will perceive that there is no insertion of any medicine of a lowering nature, and, if there were, not a single dose would be sold. Every patient is too low, and all the chemist's powers are exercised to produce strengthening remedies. After the clever combination of quinine and iron had existed for some time, strychnine was successfully added to them, and large was the number of people swallowing the compound. This continued until some one suggested the addition of phosphorus, as necessary for weak brains. As a fortune seems to be soon made with these invigorating compounds, we need not lament that it was soon to be superseded by a similar mixture, combined with cod-liver oil. Then some wary rival found that these would not digest without lactic acid or pepsine, and a new compound was framed. Then, the articles of diet are all made on the same principle; the strongest essences of meat are sold, so that a spoonful is equal to a chop or pound of beef, and a little maltine is as invigorating as, and less injurious than, a pint of beer. To prove the value of these things, I propose that a healthy chemist be taken, and made to live for a month, if possible, on his own essence of meat and maltine, with perhaps a few peptones thrown in. If he fall into a bad way, he should then be revived by chemical food—such as the lacto-phosphates, Siegel's syrup, or some good blood-restorer. I have intended to imply, as you no doubt comprehend, that we are too much in the hands of the chemists and druggists; and that, instead of administering simple remedies of known effect, we have been content, through laziness, to prescribe these popular strengthening compounds. In this way, the therapeutic art will assuredly not advance; and it is equally evident that, if it were placed on a scientific basis, nothing could be more conducive to the death of all quackery. The latter could not exist if the prescribing a medicine implied a knowledge of its action and the proper occasion for its employment.

Since the discovery by Bernard of the regulating power of nerves over the smaller blood-vessels, hopes have arisen that we had therein a clue to the operation of medicines, as well as an explanation of many morbid processes. A paralysis of the vessels, or their increased tension through the influence of the nerves, could explain almost every action in the human body. It was thought by one enthusiastic physician that in belladonna and strychnine might be found the universal remedies; the same idea was simplified by the substitution of heat and cold. It was certainly interesting to observe how the system could be made applicable both to surgical and to medical treatment. If the physician, by giving an internal medicine, could, by acting on the vaso-motor nerves, increase or diminish the supply of blood to a part, and so arrest the morbid process, in like manner the surgeon could apply an astringent to an inflamed conjunctiva, or paint nitrate of silver on an erysipelatous surface, or, by a more mechanical way, arrive at the same end by strapping an inflamed joint or testis, or arrest a growth in a limb, by tying the main artery which supplied it.

To me it seems strange that such a simple system should ever have