that the word "clinical," as applied to our Society, should be taken in the broadest sense—as relating not merely to the recording of cases, but to all that wide field of therapeutics which may enable us to relieve our patients *cito, tuto, et jucunde*.

THE LETTSOMIAN LECTURES ON SYPHILIS AND THE NERVOUS SYSTEM.

Delivered before the Medical Society of London, January 21st, 1889.

By W. R. GOWERS, M.D., F.R.S.

LECTURE II.

[Concluded from page 171.]

THE inadequacy of the clinical history of a syphilitic lesion, when that history is based only on certain evidence, is conspicuous in the case of the disease of the walls of the arteries. The classical researches of Heubner, who first placed our knowledge of this subject on a secure footing, were based, of necessity, to a large extent on fatal cases. The clinical picture delineated from those facts, faithful as it is so far as regards the effects of extensive and severe disease, is yet imperfect as a representation of the slighter and more common consequences of the morbid process. Passing over the former, because well known by description, although now seldom met with, I would ask your attention to some facts regarding these slight forms. The importance of the early diagnosis of this condition is unequalled even among syphilitic lesions. There is no luetic process which is so likely to cause grave and lasting damage, no process the effects of which, when once produced, are so entirely beyond our power, and yet can be so surely averted by prompt treatment. For this reason I think it is wise to devote more time to this than to the other special questions, and also because it is a disease in which the diagnosis during life can generally be made with sufficient confidence to give a high value to clinical facts.

The chief mechanism by which this process is effective in causing symptoms, as we have seen, is by the sudden closure of an artery. The suddenness of the closure is no doubt due to the fact that the narrowed cavity is finally occluded by clot, and in this respect the syphilitic disease does not differ from the other common form of arterial disease—atheroma. The closure is probably not often that of the main artery whose wall is diseased, but of a branch that comes off at the spot, the orifice of which is narrowed, so that the blood in the part beyond tends to stagnate and to clot. The closure has the usual consequence: sudden anæmia and necrosis of the brain tissue, unless there be sufficient anastomoses to permit a collateral circulation. This may be either complete, restoring function, or so incomplete that the continuity of the tissue is preserved only by a process which results in sclerotic induration.

The characteristic symptoms are those of a sudden focal lesion of the brain. Until the degenerative period of life is reached, such a sudden lesion is excessively rare, except from embolism. Embolism can usually be excluded if there is no valvular heart disease or other source of embolism, and if the patient has not recently suffered from a disease known to cause endocarditis. Hence it follows that the occurrence of such symptoms in an adult under 45, who has had syphilis and has not heart disease, may generally be held to indicate syphilitic disease of a cerebral artery, and this diagnosis may be made with a confidence possible in few other syphilitic lesions. This confidence is confirmed by the occasional opportunities for a verification. When, under such conditions, the diagnosis has been made, and the patient has subsequently died, the arterial disease has been found—invariably, so far as my own experience and reading have gone. It is true that, in rare cases, certain other causes of arterial closure must be excluded. I will mention these presently; they do not really lessen the validity of the statement just made, since the need for considering them is not only rare, but is also generally obvious. This confidence can, however, exist only before the degenerative period. When this period arrives, other causes of arterial closure come into operation

—causes which we cannot exclude with the readiness and confidence with which we can exclude embolism. This introduces an element of doubt into the diagnosis of cases over 45 or 50 years of age. Hence, in endeavouring to ascertain the history of these cases from mere clinical observation, it is desirable to confine our attention to those in which the diagnosis can be made with most confidence. The disease does occur during the later period of life; we have to recognise the fact in our practical diagnosis. It may be that this renders some of our conclusions from the earlier cases imperfect, but we must accept this imperfection in order to obtain as much certainty as possible.

The following conclusions are based on a series of cases that have come under my observation, excluding those over 45 years of age. The cases are 50 in number, but on some points (owing to omissions) the precise number available for comparison is somewhat less. The patients were over 25, with the exception of one, aged 21. Males were to females as three to one, a proportion that may not be far from the incidence of syphilis itself.

Many observers have noted how various are the intervals between the primary disease and the cerebral lesion, and the variation is conspicuous in this series. In one quarter, the lesion occurred during the first two years after infection, and the remainder were pretty evenly distributed during the next twelve years: 1 to 2, nine cases; 3 to 5, seven cases; 6 to 10, nine cases; 11 to 15, eleven cases; 16 to 20, four cases. There was an interval of nineteen years in a man who had well-marked syphilis at 18; and at the age of 37 an attack of right hemiplegia, while a year later paraplegia came on in a subacute manner. Eighteen years intervened in a woman who at 16 married a man of unsteady habits; her first two children were born dead, and hemiplegia came at 35, with an onset characteristic of arterial thrombosis. The shortest interval was apparently only three months, but the case does not seem otherwise open to doubt; a man, at the age of 21, had, for the first time, a chancre—a distinct hard sore—followed by indolent enlargement of the glands. Three months after the appearance of the sore, he had sudden hemiplegia, which slowly passed away, but was followed six months later by a second attack on the same side. In these cases, as indeed in all those compared, careful search revealed no other cause. In the only other case in which the interval was less than a year, the attack occurred six months after infection.

By far the most common effect of the lesion is hemiplegia, indicating disease of the middle cerebral artery, and probably the closure of a branch to the central ganglia. In only one case did convulsions at the onset suggest that the damage was limited to the cortex. (Simultaneous central softening necessarily prevents such special indication of a cortical lesion.) In three cases, hemianopia suggested that the arterial disease was in the posterior cerebral. It is evident, however, that these cases present only some of the effects of vascular disease, and do not present at all its gravest consequences. Lesions may occur in parts of the brain where they cause no focal symptoms; and, on the other hand, disease of the basilar and vertebral arteries, sufficient to cause definite symptoms, is seldom survived. These grave effects, however, are well known, and I therefore pass them over. In probably nineteen cases out of twenty, the Sylvian artery is that by which arterial disease causes symptoms—a fact easily explained—and of such disease this series probably presents a fair example.

The degree of the paralysis and its course present variations as great as, and similar to, those met with in embolism. It may be slight and transient, or severe and then often lasting, with only such recovery as occurs from compensation—a slow return of power in the leg and some in the upper arm, the hand remaining powerless, or almost powerless, with late rigidity. I have a few times known two attacks of hemiplegia to occur, at intervals of months. Very rarely symptoms of some other syphilitic process coincide with the onset. It is only in such a case, in which there is reason to suspect a gumma, that I have seen optic neuritis. In several cases the patients have, at some other time, suffered from paraplegia, such as might be caused by a gumma pressing on the cord.

The onset occurred during sleep in a third of the cases, a proportion nearly corresponding to the time spent in the sleeping state. During the waking state, the onset is seldom attended by loss of consciousness; it was so attended in only one-tenth of the cases—a fact of much importance. Giddiness and vomiting were occasional accompaniments. Of great significance also is the fact that the onset, in more than half the cases, was preceded by headache. The pain was usually great, and was either general or

chiefly on the side of the subsequent lesion. It preceded the onset sometimes for only a few days or a week, often for several weeks, rarely for two or three months. It is apparently in some way due to the arterial disease itself. Occasionally, for a day or two before the onset, there was a slight tingling or other sensation in the side afterwards paralysed. Thus one patient, after ten days of severe headache, had repeated attacks of tingling down the side during two days before the onset. The attack itself was often quite sudden, but in some cases it was deliberate or occurred in two stages. The following are instances of the modes of onset: 1. The arm and leg became weak a few minutes, then recovered, and half an hour later became suddenly powerless. 2. Slight weakness came on, and continued for six days before the sudden hemiplegia. 3. The leg became weak in the evening, and during the night the arm became powerless. 4. The patient woke up one morning with loss of power in the leg, and during the next two days the palsy spread gradually to the arm and face. 5. The arm became suddenly paralysed, and six days later aphasia came on, with a convulsion, doubtless from the occlusion of a cortical branch.

The diagnosis of this disease chiefly depends on the causal indications, negative and positive—the absence of other causes, and the presence of syphilis. The evidence of therapeutics here fails us. The syphilitic process in the artery, and the secondary thrombus that results, are wholly separate from the simple process in the brain—the necrotic softening, on which the symptoms directly depend. Treat the patient as you will; remove as speedily and as completely as possible the disease of the arterial wall—you cannot restore the circulation, nor can you avert the resulting necrosis of the brain tissue when once the blood has clotted in the vessel. Compare two series of cases of hemiplegia, one due to embolism and the other to this disease, and you will find that their general course corresponds, however wise and thorough may have been the treatment adopted in the syphilitic series. In each set we meet with cases in which the palsy is slight or transient; but these are cases in which the motor structures have not been involved in the softening, and have only been interfered with for a time, because the lesion was adjacent to them. In each series we find cases of severe paralysis, because the motor path was interrupted or the centres were destroyed. Hence, by your treatment you may save the patient from a fresh attack, or from an extension of the old mischief, but you cannot save him from destruction of tissue or avert its effects. Recognise any warning there may be, anticipate the onset—and the attack may perhaps be prevented; but once the vessel is occluded, your treatment is powerless over the accomplished injury. Thus, these are cases in which the result of treatment has no diagnostic significance, and if a positive result seems traceable, we may be sure that the improvement is a coincidence and not a consequence.

An illustration may make this important fact more definite. If the illustration is hypothetical, it will nevertheless be felt, I think, to be within the bounds, not only of the possible, but of the probable. A man is seized with sudden hemiplegia due to embolism, which has caused a small spot of softening near, but not involving, the motor structures. The function of those is temporarily impaired, as we know commonly happens in such a case. He has undetected mitral obstruction, a short presystolic murmur having escaped recognition—also not beyond the limits of experience. His medical attendant, who knows that the patient has had constitutional syphilis, believes that the hemiplegia is syphilitic, and at once gives him iodide of potassium. In two or three days there is a distinct return of power in the limbs. The improvement goes on, and in a month the patient is practically well. What will be the strength of the conviction on the part of the practitioner that the hemiplegia was due to syphilis? Will it not be almost impregnable? And yet it would be wholly wrong. Note, moreover, that had the same lesion of the brain been due to syphilis, his reasoning from the effect of the treatment would have been equally wrong, and yet his conclusion would have been right. A great deal of error in medical science (and not in medical science alone) is due to the fact that correctness of a conclusion is transferred to the reasoning by which it has been reached.

The frequency of premonitory symptoms, and of a more or less deliberate onset, without loss of consciousness, furnish diagnostic indications that are often of great importance. They are especially important when some other cause of arterial occlusion co-exists, and especially when a source of embolism can be found. Sometimes heart disease is left by rheumatic fever; the individual contracts syphilis, and afterwards has an attack of sudden hemiplegia. We are then unable to avail ourselves of the causal indica-

tion, unless we find evidence that one influence or the other has recently been active in the system, as by recent embolism or recent syphilitic processes elsewhere. A deliberate onset is strongly in favour of thrombosis, and, therefore, of syphilitic disease rather than of embolism. Most important of all, however, are the premonitory symptoms, and especially the headache. This, if severe, shows that a morbid process within the skull preceded the onset, whereas, in embolism, the intracranial condition is normal until the obstructing particle is suddenly transferred from the heart to the cerebral artery. The practical value of this indication is increased by the fact that it is not only a common, but a prominent, symptom.

Apart from embolism and injury, hemiplegia, coming on between 25 and 45, is, I believe, very seldom due to any other cause than syphilis, and its occurrence may justly raise a strong suspicion of this disease, even in a case in which there is no positive evidence. But this suspicion is stronger in the case of men than women, because in women, less rarely than in men, sudden hemiplegia, clearly due to an organic lesion, sometimes comes on when we can trace no source of embolism and when we can exclude syphilis. Now and then the onset is accompanied by that form of pyrexia which suggests septicæmia, and actual proof of this may be furnished by the characteristic retinal hæmorrhages. A softened clot, in some situation, is the most frequent cause of the symptoms, and the hemiplegia may be regarded as embolic. But there are other cases in which the onset is unaccompanied by any symptom to suggest embolism. I have seen several such cases in females between 18 and 30, in whom, from history, character, and life, inherited or acquired syphilis could be absolutely excluded. The hypothesis of thrombosis in a healthy artery, due to some general condition, seems perhaps the least improbable explanation of such cases; but if the subject of such a palsy happens to be a girl of the lower classes, especially if she is good-looking, the diagnosis of probable syphilis is not unlikely to be made, and the more readily because we cannot exclude the risk of infection, in the case of unmarried women, as we can in unmarried men.

With the advent of the degenerative period of life comes an enormous accession of difficulty in diagnosis. After the age of 45 or 50 is passed, we can never feel sure that we are not in the presence of the changes in the arteries which cause cerebral hæmorrhage or cerebral softening. The difficulty is further increased by the fact that syphilitic disease becomes not only relatively but absolutely less frequent, so that the likelihood of it does not help the observer as at an earlier period of life. Hence, when it does occur, it is more likely to be unsuspected and unrecognised. This is indeed one of the chief practical risks of error, for the diagnosis can still be made in most cases, although less surely. The diagnostic indications that are often distinct and important in the earlier period of life are equally common and equally significant in the later period. The significance of some of them is indeed not so strong as in early adult life, but their relative importance is greater, since on them, and especially on those that are still unequivocal, the diagnosis chiefly depends. Their significance is strengthened by a history of syphilis, but this history alone has far less value than at an earlier age. Further, as we cannot exclude syphilis merely because there is no history of it, so now we cannot exclude the degenerative lesions merely because degeneration is not manifest. Hence arises the extreme importance of the diagnostic indications afforded by the mode of onset, and by the premonitory symptoms, taken of course in combination with the general diagnostic indications that apply to all sudden lesions in late life. The absence of coma, the frequent occurrence of prodromata, or a deliberate development of the palsy, together with the state of the heart, commonly renders hæmorrhage unlikely, and the chief difficulty is the distinction from atheromatous softening. In this, as in syphilitic disease, a morbid state of the arteries exists before the sudden onset, and this may cause slight premonitory symptoms, analogous to those we have been considering; but headache is not very common, and is never so considerable as it so often is in syphilitic disease. Atheromatous softening, moreover, is essentially a senile malady. Apart from kidney disease or an early degenerative tendency, usually conspicuous, it is not often met with under 60, and it becomes more frequent as life advances, while the syphilitic lesion becomes more and more rare, till at last it is only to be thought of when special circumstances suggest its possibility.

The application of these principles, varying as it does in detail

I must leave. But I may mention to you a case which shows very well their use and value, and which—one of the best unverified illustrations of this particular diagnostic difficulty I have ever met with—came under my notice as I was finishing this part of my lecture. Dr. Barnes, of Ewell, asked me to see with him a woman, 42 years of age, in whom, two days before, right hemiplegia had come on in the course of a few hours, without loss of consciousness. The face was not affected on the right side, and this, with distinct weakness of the left side of the face, tingling of the left hand, and frequent hiccup, made it probable that the lesion was in the pons. The urine contained albumen and granular casts, and the albuminuria had existed for years. In the left eye were the remains of syphilitic choroiditis, and one child had been born dead without traceable cause. For several months she had suffered much from headache of increasing severity, and had become much stouter.

Here, then, we had evidence of a lesion which was probably hæmorrhage and not softening, on account of its position and of the deliberate onset without loss of consciousness—hæmorrhage into the pons causing more severe initial symptoms, which are seldom survived for more than a few hours. There was no source of embolism, and hence the most probable lesion was disease of the basilar artery with thrombosis in some of its branches. For such arterial disease we had two adequate causes: renal mischief sufficient to render atheroma probable even at her age, and syphilis. There was no evidence of recent active syphilis, and the balance of probability was strongly in favour of atheroma, by reason of the greater average frequency of this affection under these circumstances and causal conditions. But there was one symptom of a positive character, the significance of which no mere probability or improbability could be allowed to influence—the severe and persistent headache. Kidney disease will cause headache, but not such headache as this patient had suffered from, which had kept her awake at night repeatedly, and was often more severe on the left side of the head than on the right. It was a symptom that suggested more than mere atheroma, and distinctly turned the balance of evidence in favour of syphilitic disease. The case was clearly one of urgent danger, as every case is in which there is an active morbid process in the pons, and I confess I had little hope that any treatment would avail. But we decided to treat the patient for a time on the assumption that the disease was syphilitic, and to increase the iodide—which she was already taking in small doses—to 45 grains a day, adding a little digitalis to steady the circulation. If no improvement could be observed at the end of a few days, the dose of iodide should be reduced again, lest, if no good could be done to the wall of the vessel, the drug might do harm by increasing the tendency of the blood to clot. But on the third day the patient was distinctly better; the improvement steadily went on, and I hear now from Dr. Barnes that she has fair power in the arm and leg, is free from headache, and has no symptoms to suggest anxiety. Of course, the diagnosis may have been wrong; but its method may yet be instructive, and the result appears to afford confirmation both to the principles and to their application. At least, we may see in this case how essential it is to arrive at a precise diagnosis of the character of the lesion before the causal indications are considered, and that this need, always present, is especially obvious in those whose age or state involves the possibility of several lesions. A hasty diagnosis of cerebral hæmorrhage in this case would probably have prevented any significance being attached to the indications of previous syphilis. We may see also how necessary it is to give its due weight to each indication, how the significance of one may be strengthened by the concurrence of others, and how paramount is the necessity of limiting the influence of mere probability, and not permitting this to counterbalance any distinct and positive indication. Although, as I said, recovery does not prove that the diagnosis was correct, this reservation must be made whenever a patient happily survives. The reservation applies only, moreover, to solitary cases. The test of the reasoning afforded by the result may not be valid in the individual case, but in a number of cases combined it will not mislead.

Another point in the differential diagnosis of these cases should, however, be specially mentioned, because its peculiar and unusual difficulty occasionally leads astray even those who have had much experience. It is the distinction of the slighter forms of this hemiplegia or other disturbance from the unilateral symptoms that are common in general paralysis of the insane, even in the early stage of the disorder. Whatever be their immediate cause, they

do not seem to depend on an organic lesion, but the symptoms of such a lesion are sometimes simulated by them in a puzzling manner. The difficulty is increased by, and, indeed, is largely due to, the fact that so many of the subjects of general paralysis have had syphilis.

Cases are not rare in which the disease comes on while the syphilis is manifestly active, and it may even supervene on some actual syphilitic lesion of the brain. The hemiplegia due to syphilitic vascular disease is occasionally followed by degenerative symptoms of various kind and degree. After recovery from the palsy, complete or incomplete, there may distinctly develop such symptoms as failure of memory, excitability, alteration of mental and moral characteristics, changes in articulation in various degrees of intensity. These symptoms may commence during recovery from the hemiplegia. They may continue for a time, and then pass away, or may persist stationary or increasing. But an attack of hemiplegia which seems at the time to be due to the common vascular disease is occasionally followed, in the course of a few months, by symptoms of general paralysis of the insane quite typical in character and course. For instance, a man about eight years after primary syphilis had an attack of sudden hemiplegia, severe, and preceded by headache. In the course of a month he recovered almost completely, and those who knew him observed very little difference from his state before the attack. A little later, however, a slight change in articulation attracted their notice, coupled with some scarcely definable alteration in his mental characteristics. In a few weeks more optimistic delusions suddenly burst out; he was about to make his own fortune and the fortune of his friends, the thousands and hundreds of thousands poured from his lips in the flow of magniloquence we know so well—a flow that was soon rendered almost unintelligible by the usual characteristic failure of articulation, and the disease ran a rapid course to its common termination. In such a case, if the initial hemiplegia is slight and transient, it may be difficult to say whether the attack is due to an organic lesion, or is one of those unorganic attacks that occur in the course of this disease. I think that we must regard the hemiplegia as primary and general (1) whenever there are no preceding symptoms, slight or considerable, suggestive of general paralysis; (2) when the palsy is so considerable that the symptoms last for more than a week; (3) when a distinct interval separates this attack from the chronic symptoms; and (4) when there are no recurring attacks of similar character.

Another difficulty is due to the fact that in general paralysis attacks occur in which, instead of the sudden weakness of hemiplegic character, there are unilateral symptoms characteristic of a local "discharge" in the brain. When such discharge is so great and so situated as to cause actual convulsion, there is less risk of this particular error, but sometimes there are other sudden symptoms, more equivocal in character, such as tingling, beginning in some part and spreading through the side, and accompanied, it may be, by inhibitory weakness or with temporary aphasia. Such attacks have often been ascribed to syphilitic vascular disease. The latter, however, does not cause such definite limited "discharges" as a first symptom. These always suggest a chronic process, and not a sudden lesion, and therefore, in the absence of signs of tumour or meningitis, the degenerative malady should always be thought of. This is true also if there have been similar attacks, or change in mind or speech, however slight.

Among the innumerable diagnostic problems that invite, and almost demand, consideration, I can notice only one other group, those that relate to the palsies of the ocular muscles. These are among the most frequent manifestations of the influence of syphilis on the nervous system. The long and varied course of the ocular nerves exposes them to many kinds of specific damage. Arterial disease seldom interferes with their functions, and when it does so it is only by causing a sudden muscular lesion, separable from all others by the complex associated derangement produced, and by the actually sudden onset. Gummata and local meningitis may damage the nerves at the base of the brain, and the locality of the lesion is then indicated by the grouping of the nerves affected; the two processes are sometimes distinguishable by the greater interference with the central organs or with the motor tract in the crus and pons, and also by the greater frequency of optic neuritis in gummata than in meningitis of the posterior part of the base. Optic neuritis may be readily caused by meningitis when this is in the front part of the base, so that this distinction must depend on the evidence of the position of the disease. Gummata on the nerve-trunks themselves can scarcely

be distinguished from inflammation. In any part of their course a syphilitic neuritis may occur, and this is probably the most common cause of an isolated affection of a single nerve or of a single branch. Thus every diagnosis involves the careful consideration of the symptoms and their combinations, as well as of their mode of onset and associations. The subject is too large for me to do more than thus indicate the lines on which it must be approached. It needs, indeed, to receive a thorough investigation in the light of the pathological facts that are scattered through medical literature, and which need to be greatly increased in number before our diagnosis can be exact.

The greatest difficulties arise from the fact that ocular palsies are caused by some of the effects of syphilis that are not specific in their nature. An isolated palsy of one third nerve, in a young man known to have suffered from the constitutional disease, was naturally supposed to be truly specific. But treatment, although prompt and thorough, had no influence on it. One day he suddenly became unconscious, and died in a few hours. We can scarcely doubt that an aneurysm, due to syphilis, caused the palsy and his death. But the most frequent non-specific causes of these symptoms are the consecutive degenerations. These may set in before the period of true specific lesions has passed, and the ocular nerves constitute one of the regions in which the symptoms of the two kinds most often coincide. More frequent than actual coincidence is the doubt produced by it. Is this palsy due to a true specific lesion, or is it part of nerve degeneration that is a non-specific sequel to syphilis? The question is of great importance in regard both to prognosis and to treatment.

In cases of locomotor ataxy that occur comparatively early, this difficulty is especially frequent. The distribution of the symptoms may furnish an answer, as we shall see in a moment. Sometimes the suddenness of the onset raises a strong presumption that the palsy is tabetic and not specific. The same conclusion is indicated when symptoms quickly lessen without treatment and soon afterwards return. The nature of many of these tabetic palsies is doubtful, but some are certainly due to nuclear degeneration. These, as we saw in the first lecture, are often sequelæ of syphilis. Their distinction from a true specific lesion is another problem of much difficulty, a difficulty further increased by the fact that the nuclear affection seems sometimes to follow a peripheral palsy so directly that the symptoms of one pass into those of the other.

The most important characteristics of the nuclear palsy are the association of muscles according to function, its bilateral distribution, and its persistence in spite of treatment. In most true specific lesions, prompt treatment has an influence which is absent in the degenerative maladies. I need not trouble you with instances of these features. They are important distinctions, both in the isolated degeneration and in that associated with tabes. I may add to these distinctions the therapeutic test, which is often of great use in the diagnosis, but is of negative significance only when the proper treatment has been employed early.

I have said that the nuclear degeneration sometimes seems to follow a specific palsy—chiefly when, from the lateness of treatment, this palsy has lasted long and recovered imperfectly. A curious speculation is suggested by this—a speculation which I may indulge myself by starting, although I may not pursue it. Is the semblance of causation a real indication? Conduction through motor fibres is arrested; this arrest may—perhaps must—modify the action of the cells from which the fibres conduct, for the fibres do not carry force like tubes, but by an action similar to that of the cells, but far lower in degree. The disturbance of the centre must entail changes in nutrition, as every functional state does. We see the effects of prolonged arrest of conduction in the altered action of the facial muscles after disease of the nerve-trunk. It is conceivable that, given a predisposition to degeneration, the changes in nutrition excited by the arrested action may acquire an independent energy, and assume an independent course. The conclusion is, moreover, not without indirect support from some of the facts of physiology.

A tendency is often to be observed in the present day to under-rate diagnosis, or at least the elaborate diagnosis of which the diseases of the nervous system furnish so many examples. In the face of the urgent needs of suffering humanity, with its mute or uttered cry for the relief we cannot always give, our precise distinctions and elaborate processes may seem like an ingenious

device for interesting us while the patient suffers. That such an impression is wholly wrong I need not say to those who hear me now; but the tendency is real; it is reflected beyond our own ranks; and I may give one warning—a warning to myself as well as to others—that we should be always on our guard lest we do anything, by word or manner, that may excite or foster the feeling to which I refer. Diagnosis must come before treatment, but this should make us careful lest we produce an impression that we regard the order of the two in time that also of their importance—an impression easily produced when, as often, the treatment is plain and its methods familiar, while the diagnosis is complex and its processes strange.

Surely the diseases we are now considering supply a strong reproof to those who consider that, "to say the least, diagnosis is somewhat overdone." A diagnosis that can be described as "rough and ready" may serve to make our treatment effective in the majority of cases, but there are many in which only a most careful and even elaborate diagnosis will enable us to do all that can be done, and there are not a few in which the utmost degree of care and elaboration is needed to save us from being wrong in our treatment—often wholly wrong, and sometimes disastrously wrong. I could give many instances of this, striking and sad, but it is needless to do so, and it may be unwise. My own conviction is that diagnosis in itself cannot be overdone. To permit diagnosis to be studied and employed at the expense of treatment is a proceeding for which no words are too strong. To cultivate, at the expense of its application, that which has no other end, should in medicine be a sin beyond the reach of pardon; but, as a guide to treatment and prognosis, we cannot well know too minutely the seat of the maladies we are treating, and assuredly we cannot know too certainly their nature. Our art may be all-powerful to save, but without guidance it is useless—a hand to use without an eye to see. When the disease is beyond our present means of combating it, the same guidance is still needed to save the patient from that which, without power of good, may not be without capacity for harm; and I believe that there is no point in diagnosis, however elaborate it may be or superfluous it may seem, which is not or will not be, in some case or at some time, of definite and even essential service in enabling life to be saved or health restored.

THE

PAST AND FUTURE OF PATHOLOGY.

Being a Portion of the Presidential Address delivered at the Annual Meeting of the Pathological Society of London on January 29th, 1889.

By W. HOWSHIP DICKINSON, M.D., F.R.C.P.,
President of the Society.

AFTER some preliminary remarks in the course of which he thanked the Society for the honour done him, Dr. Dickinson proceeded as follows:—

Pathology is of great antiquity, but is apparently far from being exhausted. The science of the nineteenth century has touched it with new vitality, and, with the help of the microscope and chemistry, it is now developing and bearing fruit in a manner which has no parallel in any former part of its long history.

The ancients were acquainted with some of the larger facts of pathology, and turned them to practical use. They learned at a very remote date to connect pus in the pleura with its external signs, but, on the whole, their observation was superficial, more upon the living than the dead. I cannot find that before the seventeenth century there was any knowledge of conditions to us so obvious as cerebral hæmorrhage and hepatisation of the lung, though the symptoms in connection with them were matters of ancient experience.

So great indeed have been the acquirements of modern times, that we of this day can scarcely imagine the state of medicine even at a date so recent as to be within the life and possibly within the memory of some who are still with us. Bright published his observations on the kidney in 1827. How confused must have been the ideas of dropsy and coma before that time, and how much of enlightenment was due to his observations in morbid anatomy and his association of them with certain obvious symptoms.