CONCLUSIONS.

The spontaneous modification or disappearance of pain is so large a subject that a lecture like this can hardly be made to contain more than a suggestion of its importance. What I have said, however, I hope will be sufficient to emphasize the three following cardinal points necessary to be borne in mind in its consideration:

1. No accurate estimate of the behaviour of pain is possible in the absence of a careful study of the temperament of the patient.

2. Sudden or rapid disappearance of pain should never be accepted without reserve on its own account as a sign of improvement.

3. Sudden disappearance or rapid diminution of pain, unless it be coincident with proportionate improvement in the associated symptoms, is often a sign of impending disaster, and not an indication of recovery.

An Address

ON

THE SO-CALLED FAMILY DISEASES:

PREMATURE PHYSIOLOGICAL SENESCENCE
LOCALIZED TO CERTAIN ORGANIC
SYSTEMS.

DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS
OF LONDON ON JUNE 22ND, 1908,

BY F. RAYMOND, M.D.,

MEMBER OF THE PARIS ACADEMY OF MEDICINE; PROFESSOR OF
CLINIC OF NERVOUS DISEASES IN THE FACULTY OF MEDICINE;
PHYSICIAN TO LA SALPÊTRIÈRE.

MR. PRESIDENT AND GENTLEMEN,—On February 18th last, in the name of the University of London, Sir Dyce Duckworth, President of the English Section of the Entente Cordiale Médicale, speaking in excellent French, gave in the large theatre of the Faculty of Medicine of the University of Paris a remarkable lecture on Diatheses and the Personal Factor in Disease.

No one was better qualified than the eminent physician of St. Bartholomew's for the task of expounding the traditional doctrines which the great clinicians of the past have built up bit by bit from the patient observation of the sick; and it was all the more necessary seeing that many physicians, in view of the marvellous discoveries of bacteriology, physiology, normal and morbid histology, etc., have thought, and still think, that nothing, or next to nothing, remains of the teachings of the past. They would fain have us believe that up to the time of these discoveries we had been living on mere words, on conventionalities, on systems that have crumbled to pieces one after the other. It was fitting that the author of classical works on gout—pre-eminently a constitutional disease—should put things in their proper place, and show that in every disease the *personal* factor has a prevailing influence, and that, beside the *germ*, we must take into account the *soil* in which it grows. Your distinguished countryman, with his great clinical sense, laid proper stress on this important fact. His success was great and justified.

To-day I, in my turn, have the honour of addressing you as President of the French Section of the "Entente Cordiale Médicale." This is really a return visit which the University of Paris is happy to pay to her sister, the University of London, and in her name I thank you for your warm welcome.

I must in the first place excuse myself for speaking in French, but my knowledge of your language is so imperfect that I dare not venture to lecture in English.

You would certainly be surprised were I not to select my subject from that special branch of medicine which has always engaged more particularly my attention, but it has occurred to me that I might interest you more by taking from the immense field of neurology a general subject which all physicians, whether specialized or not, should be familiar with and which—in my opinion at least—rises high above all other subjects. I refer to the so-called *family diseases of the nervous system*, and to the *part played by heredity in those diseases*.

The so-called family diseases are most interesting. They form a class of very peculiar maladies, a nosological group as important from its particular characters as from the considerable development it is deservedly attaining in pathology. Whether you consider their anatomy or their pathological physiology, their symptoms or their course, they stand out with such strong and personal features that they cannot be mistaken for any other disease. No trauma, no infection, no intoxication, no diathesis gives rise to them. From beginning to end they follow an inevitable course, as inevitable as the "Fatum" of the Ancients. To become the victims of these diseases the subjects need only be born of the same parents and reach the age marked out by Fate. Family diseases, unlike so many others, are not the expression of a struggle waged by the system against a morbid agent (and the word "struggle" implies possible victory)—they are the consequence of the original constitution of certain nerve tracts or cells, and so they have sometimes been called "evolution diseases." As a matter of fact, they can hardly be termed "diseases," for they are really *organic types abnormal from their very origin*.

I purposely say "family diseases" and not "hereditary diseases." The latter term includes too many complaints, and, moreover, pathological heredity is one with normal heredity; it is present in all sick persons—I was about to say in all men. We all know its enormous influence on the development of nervous diseases; its characteristic feature is its great dissimilarity. Moreover, an efficient morbid cause is always found working with it. Not so with the family diseases; they are exclusively created by the parents, by the ancestors, and they develop quite apart from any external influence.

Clinically, it is easy to realize the absence of any external influence when we see several brothers and sisters who have lived in widely different conditions developing at the same age the same disease, and one having as its fundamental characters that it is inevitable and progressive, and due to pathological changes which are not those of defence or inflammation.

Are these diseases always, strictly speaking, "family diseases"? That is to say, are they always repeated in several members of the same family? By no means. They are often met with as isolated cases, and it is by their own symptoms that they can be recognized.

Another general remark which must not be overlooked in connexion with these diseases is that the different clinical types which constitute them are not found to be identically the same in all cases. It seems as if each family had its own way of working out the same family disease, just as if the fact of developing in different families caused each morbid type to receive a somewhat different stamp. And thus it is that the classification of family nervous diseases is difficult. Every year, as it were, new diseases spring into existence, because authors describe too often as a special disease what is in reality a peculiar form of disease already well recognized.

In this respect nothing could be more instructive than the way in which were constituted certain morbid types of diseases, whether family diseases or not.

See, for example, what has taken place for *progressive muscular atrophy*. When for the first time this disease was described as a distinct nosological entity by Duchenne of Boulogne more than fifty years ago, this great observer described under that one name several diseases which have but one common feature—namely, slow and progressive wasting of the striate muscles. A first distinction was introduced between the primitive disease and those forms with contracture, paresis, sensory disturbances, exaggerated reflexes, etc. At the same time another separation was being achieved which led to the recognition of several varieties of muscular atrophy characterized by their inception at an early age, and by their development in several members of the same family. Thus it seemed that nothing would remain of Duchenne's primitive type. As a matter of fact this type does exist, but it is, comparatively speaking, scarce. It is a variety of progressive muscular atrophy, the "myelopathic" variety, an individual disease, characterized by slow and progressive disappearance of the large cells of the ventral horns.

To this type another form has been opposed—namely, the "myopathic" type, a family disease characterized by

its localization to the muscular system only, without any apparent change in the nervous system. The forms of myopathy became extremely numerous because at first their differential characters were alone considered, no account being taken of the essential bond which unites them together.

Later on other morbid types of progressive muscular atrophy were described. These completely differed anatomically from the two classical forms—the myelopathic and the myopathic types. Not only were there morbid changes described in the large cells of the ventral horns but also in the posterior columns (as in locomotor ataxia) and even in the peripheral nerves (Dejerine and Sottaz). And yet these were family diseases just like the myopathic cases. It became impossible, therefore, to draw a contrast between myelopathy and myopathy and to consider those two types as two distinct entities, and so I was led to proclaim once again in my lectures the unity of progressive muscular atrophy. At the present time, gentlemen, this unity has become an unquestionable fact owing to the variety of family progressive muscular atrophy described by Werdnig and Hoffmann, which forms without possible doubt the connecting link between the two great varieties of progressive muscular atrophy. There is, therefore, no well-defined limit between the different clinical types of progressive muscular atrophy. The disease is *one*; it is essentially due to the degeneration of certain organic segments of the neuro-muscular system.

Again, myelopathy is nearly always seen as an individual disease; and yet it has the same significance as the other purely family types. The same may be said of the “amyotrophic” lateral sclerosis of Charcot, of which myelopathy can be considered as a part. Definite proof of this opinion, which I have been maintaining for years, is found in a case reported by Professor Rovighi of Bologna, at the International Congress at Lisbon. In this case mother and daughter both died of amyotrophic lateral sclerosis duly verified with the microscope.

I think it is possible to go one step further. If we consider the work that has been done in Germany, England, the United States, and France, on “myasthenia,” “myotonia,” “myatonia,” we are bound to reach the conclusion, already arrived at by Messrs. Klippel and Villaret in 1906, that there is an evident relation, which we are just beginning to perceive, between all those morbid conditions, and also between them and myopathy. In fact, all those diseases seem to be due to a faulty development of the spino-muscular neurone at its very origin, kept up and perhaps exaggerated through deficient nutrition, due in turn to defective working of certain ductless glands.

We have, then, a series of well-defined syndromes, some of which constitute obviously family diseases; others that we consider as such by reason of their clinical similarity with the members of the first group. The two groups unite to form one class, which bears the name of progressive muscular atrophy.

The same demonstration could be made in regard to other varieties of family diseases, especially the group of “hereditary ataxias.” In my lectures on Friedreich’s Disease I have demonstrated the existence of hybrid forms of hereditary ataxia. These forms have so increased in number that the pure type which was first described is now the exception rather than the rule. As a matter of fact there is no well-marked boundary between the two extreme types of hereditary ataxia: the spinal form and the cerebellar form. Friedreich’s disease and the cerebellar heredo-ataxia of Pierre Marie, with the intermediate types, have as a common feature the changes in the cerebellar system both in its afferent and efferent paths, and in my lectures of 1905 I proposed the following classification:

- A. A *spinal form* with loss of reflexes, scoliosis, and club-foot added to the common syndrome. This would be the *Friedreich type*.
- B. A *cerebellar form* with atrophy of the optic nerves, vertigo, mental disturbances, added to the common syndrome, forming the *Marie type*.
- C. A *bulbar form* with vomiting, dyspnoea, and cardiac arrhythmia.
- D. A *bulbo-pontine form* characterized by auditory disturbances.
- E. A *generalized form* as in Menzel’s case.

These are all different types, but they are all linked together by the common cerebellar syndrome. This same

common link has been found in those types described by Pauly and Nonne, by Lorrain, and which are intermediate between family ataxia and the spastic paraplegia of Strümpell. It also appears in the recent cases published by Mademoiselle Pesker in her *Thesis* and where the cerebellar syndrome is associated at the same time with symptoms of mental failing with spastic paraplegia, the anatomical basis of this condition being well-marked hypoplasia of the whole cerebro-spinal axis, without sclerosis. In all those cases disease is the direct outcome of heredity, and this fact should be borne in mind when attempting a nosological classification.

Family diseases can, however, be considered from a higher and more general standpoint. I use the word *general* advisedly, for this question is by no means confined to the nervous system.

I have no intention of making a lengthy digression in the domain of general medical pathology. I may be allowed, however, to recall the excellent work that has been done of late years on family diseases of the digestive system (merycismus, periodical vomiting), of the liver and bile ducts (as for example family cholaemia, so well described by Gilbert and Lereboullet), of the kidney (Charrin and Delamarre, Castaigne and Rathery), of the circulatory apparatus, of the skin, the organs of sense, the blood (haemophilia), the bones (achondroplasia, cleidocranial dysostosis), etc.

It is most important to recognize accurately the family character of a disease, whatever be the disease and its localization. By this I mean that we should not give the name of “family diseases” indiscriminately to all congenital or hereditary diseases. Thus we separate from the group of family diseases those maladies where heredity manifests itself by the transmission of some infection such as syphilis or tuberculosis. Several children born of a syphilitic father may be afflicted with the symptoms of hereditary syphilis without our considering this in the least as a “family disease.” If, however, the children exhibit dystrophic stigmata, the distinction is more difficult. These remarks about syphilis apply equally well to tuberculosis, diabetes, gout, intoxications such as lead poisoning, or alcoholism.

It becomes very difficult to apply the qualification of family disease when in a family heredity creates a *locus minoris resistentiae* which is to be found in all its members. It is in this sense that Gilbert and Lereboullet understand the family character of cholaemia. In the patients to whom the authors’ description applies, there is a special predisposition to infection; hence, at the first opportunity a morbid condition arises in the bile ducts.

I fully recognize the analogy between the pathogeny of those diseases and that of the family diseases as I understand them. But we must bear in mind that in those conditions described by Messrs. Gilbert and Lereboullet the disease is not fatal; it does not develop at the same age in all the members of the same family; finally, it depends necessarily on the intervention of some external agent—microbe, poison, or toxin. Now all these characters are essentially different from those ascribed to family diseases proper. Under this heading I place only those diseases which affect in an identical form several children in a same generation, which make their appearance at the same age in all of them, and which originate without the intervention of any morbid agent, whether extrinsic or intrinsic.

Of the three fundamental characters just mentioned, the two first may be wanting, as there are isolated and aberrant cases; these are all the more difficult to identify that the most remarkable peculiarity of family diseases is a great variableness in the symptoms. We must not expect to find the same type everywhere, each family has its own. Even the best known of these diseases—as, for example, Friedreich’s hereditary ataxia—does not always appear with the same features. The absence of all known efficient cause is therefore the principal character of family diseases.

Nevertheless the morbid anatomy, the symptoms, the course, are sufficiently peculiar to identify the *nervous* family diseases, the only ones I have in view here.

The *morbid anatomy* is characterized not by localized degeneration to one or other of the fibre groups or cell masses, but by strict localization to one or several systems. No inflammatory products are found, whether nodular or in streaks and extending to the several tissues of one same

region. Macroscopically we simply notice the disappearance of certain parts which constitute, as we know, one functional whole. Microscopically we only meet with simple atrophy of the nobler elements, with hypertrophy of the interstitial-tissue. In some cases this hypertrophy is limited to the replacement tissue. In other cases such as pseudo-hypertrophic myopathy or in the interstitial neuritis of Déjerine and Sottaz it reaches an abnormal development. But in all those cases one very important fact is noticed, and that is the absence of any inflammatory reaction. The system has not to repulse an attack from the outer world or from the depths of its own being. There is no trace of a struggle, and therefore no evidence of defence. The lesion consists simply in the disappearance of a system of cells or fibres which dissolve as it were without leaving a trace. Connective-tissue growth then occurs and takes the place of the cellular system that has disappeared, but this is only to fill the void.

This process, therefore, is very different from that which modern conceptions ascribe to diseases. These are likened to a real battle with its hand-to-hand struggles, its defenders and assailants strenuously fighting against one another, and destroying one another amid much havoc. Again, the process is very different from the less boisterous but none the less earnest attack of poisons or toxins causing our cells to decay, and modifying our body fluids. Nor can it be compared to the traumatic changes such as are found in the neighbourhood of a tumour or of a haemorrhagic area, nor to the massive necrosis of ischaemia, whether due to an embolus or to arterial stenosis.

The symptoms of the family nervous diseases depend entirely on the physiological functions of the tracts or cells that are doomed and are disappearing gradually. Any part of the neuro-muscular system may be involved. We must remember, however, that even when the morbid changes are well limited the functional disturbance points nevertheless to an involvement of the whole neuro-muscular system. The proof of this is in the frequency of the mental syndrome. The sphincters, however, remain as a rule untouched until the very last period of the disease.

The course of the disease is the most important point.

The onset is slow and insidious, so that it is impossible to state accurately at which moment the trouble has begun. The course is always progressive. There is never any retrocession; never does a symptom fade or disappear when once it has appeared. Sometimes the course is hastened under the influence of certain causes (nearly always toxic or infectious). But the rule is an imperceptible and continuous progression, running on very slowly and very smoothly till the complete disappearance of the affected system. The disease may not necessarily prove fatal, the affected system may not be indispensable to life or else its function may be carried on by another system, so that death will not come about without the interference of some other disease. Death is not, therefore, the direct, immediate, and fatal outcome of family diseases proper.

It now remains for us to see how the family diseases develop. What is their pathogeny? What place should they occupy in a classification of diseases?

The problem is not only of a speculative interest; it has also a great practical bearing.

When describing the inevitable course of family diseases I made no reservation, the patient is doomed from the very instant of his conception, as soon as spermatozoon and ovum have united. Now this we can prevent by forbidding marriage to those individuals who bear a hereditary stigma. We must never forget that a physician must try to prevent at least as much as cure. Moreover prevention is easier than cure. From the standpoint of prevention it may be therefore of the highest practical importance to know the circumstances which give rise to family diseases.

In the first place, we must inquire into the etiological factors such as we find them in the detailed reports on which has been built the history of these diseases. Unfortunately, at the present time we can trace but their outline. The problem may, however, be somewhat simplified by the following considerations, which, it seems to me, have not sufficiently engaged the attention of observers hitherto.

The perfect development of a germ supposes in the first place the existence in that germ of its own attributes. In

the human species the germ arises from the union of the spermatozoon with the ovum, and these must necessarily possess certain qualities for the germ to be able to develop. Further conditions are also necessary; thus a favourable medium is particularly indispensable, for one cannot separate the germ from the medium in which it is about to develop, and, after all, development is but the outcome of the conflict between the cell and the medium.

In relation to family nervous diseases, the plain facts are as follows: An organic system—the nervous system—comes into life, develops itself, performs its functions, then fades away, dies, and disappears at a far earlier period than the other systems of the body; we see it atrophy, lose its functions, and undergo sclerotic changes long before the death of the individual.

The mechanism of this isolated and premature disappearance of an organic system is rather obscure. Is there in each cell a certain dose of energy which gradually gets exhausted until it entirely disappears? This is a somewhat vague and philosophical conception. At any rate, it is no explanation to us physicians so long as the word "energy" does not apply to something tangible and well defined. Is there, on the contrary, a poisoning of the nerve cells through some modification in their medium—as for example the formation of a special cytotoxin? Again, are the sclerotic changes brought about by an accumulation of waste products the results of cell activity?

This last hypothesis is rather tempting, for it enables us to understand why it is that the onset of the disease takes place at about the same age in the several members of a same family—that is to say in individuals whose cells have the same chemical characters and the same medium. We can understand, then, that fibrous degeneration appears at the same moment in the members of a same family, just like fermentation of yeast cells stops in a given medium at the end of a certain time, always the same for the same kind of yeast, whereas by altering the medium the period of senescence of the yeast is changed.

Now our patients represent the same primary germ, the same maternal medium, and most likely the same individual medium (as the latter depends on the working of all the organs).

As an example of the duration and family character of an organ or function, nothing could be more striking and instructive than the result of some careful observations I have just made at the Salpêtrière on the duration of genital life in certain families. I have found twelve families who have been able to give me accurate information as to the time when menstruation appeared and ceased either in the mother and daughter, or in several sisters, or at the same time in the mother and in several daughters. The duration of genital life, as characterized by menstruation, is very variable. We have found it to last from twenty-one to forty-two years with all the intermediate figures. In those twelve families the duration has proved the same in mother and daughter with a possible difference of one year.

Let us now return to the family diseases of the nervous system and their pathogeny.

The nerve cell naturally takes its share of the qualities of the primary germ cell born of the union of the spermatozoon with the ovum. Now, one or other or both of those cells may undergo some influence that may modify their normal characters simply because the father and mother suffer from a pathological disturbance of their tissues and body fluids. Now, I insist on the fact that up to the very time of conception the modifications of the parent cells may have a very decided influence on the daughter cell, the germ. To bring about a change in the spermatozoon or ovum it is not necessary for the father or mother to suffer from the slow and prolonged action of a chronic disease; remember the baneful consequences when conception takes place one of the genitors being in a condition of drunkenness. Apart, however, from inebriety, which may affect several members of the same family, we can imagine similar results arising from the use of other exciting poisons, such as cantharides or even from any other exciting cause. It has been said that suggestion may cause certain disturbances in the development of the fetus; a great mental shock or even the memory of such

a shock may perturb the parent cell. Now, if we consider the action of drugs, have we not good reason to be struck with the truly magical effects of mercurial treatment on syphilitics before conception? Take a syphilitic man. He marries but begets no children, or else his wife cannot bring to term a single pregnancy. We give a few grains of mercury and conception takes place; the child is born at term and his development is normal. In view of this fact, is it not wonderful that in our daily life there should be such a uniformity of our inner medium that spermatozoa and ova are only exceptionally modified and deviated from the normal type? We are bound to realize that the variations of our inner medium are of the slightest, and we may remark also that by the union of the two cells of opposite sexes there takes place, as it were, a levelling of defects, so that the average type of the species is maintained. Here, again, pathology enforces its laws. It is necessary that the pathological peculiarities of the germs which go to form the embryo be of a different kind so as to neutralize each other. If the peculiarities are of the same nature—synergetic—they will add themselves, or even multiply, and the new being will be not of the normal but of a pathological type.

However great the influence of synergy or opposition of parental defects or stigmata this influence is reduced to nought when the stigmata of one parent are marked enough to remain predominant. One parent may in that case unite with different partners and reproduce every time the same pathological type. And so in a careful etiological inquiry, in case of a second marriage, we are led to examine whether the children born of the second union present the same morbid peculiarities as those born of the first.

This is not all. After the union of the spermatozoon with the ovum the evolution of the future being is far from settled. The new germ cell arising from the fusion of the two parental cells must now multiply. This is only possible under special conditions of nutrition, temperature, and surroundings. If the conditions differ too widely from what they should be the cell will perish and will give rise to no living being. If the conditions are moderately different a new being will come into existence, but it will be an abnormal one. In man, of course, the maternal medium is necessary for the development of the ovum. Anything that may perturb the maternal medium may therefore exercise an effect on the developing ovum. It is therefore impossible to exaggerate the necessity of placing the mother in as healthy conditions as possible.

Now, it so happens that pregnancy exercises a marked disturbing influence on the organic functions. This influence on the organic functions may differ in intensity and in nature. Here, again, is a subject for important researches to be made amongst mothers who have borne children suffering from family diseases.

The child being born will now develop in the outer medium; it will have to withstand external influences, such as defective airing or clothing, and, above all, faulty nourishment, and we all know what extraordinary food is given sometimes to children in certain classes of society.

An inquiry into the causes of family diseases is a most difficult enterprise, for it must go carefully into the circumstances that have modified the health of the parents before they conceived; it must include the question of opposition or synergy of hereditary taints; it must find out the possible disturbances of the maternal medium during pregnancy; it must examine the hygiene of the children, etc. All those points must be minutely investigated if we are to get at the truth.

When all those causes have been brought to light, we must go still deeper into the subject. We must try and find out why a given part of the system is attacked in preference to another, why in one case the muscles are involved, in another the posterior or the lateral columns of the cord, in another the cerebellum, in another the optic nerves. Then another problem remains to be solved: Why do the cells disappear at a certain age and not at another?

If we consider the nervous system we see that, despite all the precautions taken by Nature to place the nerve-cells beyond the action of any traumatic or toxic influence, they are still very fragile, for they feel the action of a few whiffs of chloroform, of a glass of alcohol, of a few centigrams of morphine, of a few milligrams of strychnine or

of prussic acid. Nothing is to prevent us from thinking that in the course of development there are thrown into the circulation of the individual, at certain periods of his life, substances that are very toxic to his nerve cells—as, for example, those cytolytins the existence of which we are just beginning to suspect. I am alluding now to the most powerful of the internal secretions—as, for example, those that are connected with the development of the genital system; the influence of these is such that puberty and castration bring about a veritable revolution in the system. Now, is it not chiefly at the time of puberty that the family diseases of the nervous system become manifest? Whether they be brought about in this way or by other means, heredity governs their etiology.

In my lectures on heredity (1894) I have shown that family diseases represent veritable degeneration stigmata in the race. Sachs, comparing all the family nervous diseases and considering them as caused by an arrest in the development of the centres (brain or cord) in one of their constituent parts, has defended the same opinion.

That is why I think that for the purposes of a general classification these diseases should be grouped under the following heading: "Premature physiological senescence of certain organic systems." On one hand one often sees, as I have already remarked, isolated and aberrant cases of these family diseases; on the other hand, there are certain diseases which we never meet as family diseases, or exceptionally so: such are "myelopathy," amyotrophic lateral sclerosis, etc.

We need not be surprised at meeting constantly, so to speak, with these isolated cases. I have upheld the opinion that, like the others, they are due to a condition of degeneration, only they manifest themselves later and without any apparent hereditary influence. I consider these nervous diseases as showing the bad quality of certain parts of the nervous system, as a hereditary malformation, total or partial, of the cerebro-spinal axis; one can easily understand that under such circumstances an infection or any other cause may annihilate structures insufficiently resistant and badly protected against the ordinary causes of wear and destruction.

Considerations of this order inspired your eminent neurologist Sir William Gowers when he created the terms "abiotrophy" and "abiotrophic diseases." Analogous ideas have been set forth by Edinger when defending his theory of functional wear.

Duclaux has rightly said of heredity that it is the great force which governs the world. To us physicians morbid heredity should be as a breviary—always open in front of us, to instruct us with all possible certainty as to the prospects of a given case as regards its evolution, its termination, its possible consequences for the individual or for his descendants.

In this the country of Darwin, who has worked out with such sagacity the laws of normal heredity (and those of morbid heredity are the same), I need not insist on this fact that the stigmata or defects of the parents or of the ancestors are transmitted, weakened or strengthened, to the descendants. This pathological heredity, however, is not necessarily fatal. By proper measures we can prevent its consequences all the more effectively that we have penetrated more deeply in the intimate process of degeneration, especially in its relationship with internal glandular secretions and with general nutrition.

Meanwhile we should be fully impressed, and we should impress others, with the principle that to prevent diseases due to degeneration we must only allow marriage between fit individuals who seek in marriage not wealth but health. Physicians should never tire of repeating and spreading this truth. We must teach it to all, rich and poor, and never will compulsory education be applied to a more important subject.

Gentlemen, in concluding this lengthy discourse allow me to thank you for your kind attention. I have attempted to bring into prominence a particular factor in the causation of morbid conditions of a very special nature. This factor I have termed "premature physiological degeneration."

This degeneration is not an acquired one; it is inborn, and dates from the very conception of the individual; it is therefore a "physiological" degeneration as regards that individual.

This physiological senescence is quite independent of any external factor; its date of onset varies with the different subjects. It may be premature—that is, it may manifest itself at a period of life which is not that of normal senescence of the individual. I do not think this process is limited to the large group of family diseases of the nervous system, nor even to that of family and hereditary diseases considered as a whole. I am convinced that this is a far-reaching process, which deserves to be placed on a par with intoxication and infection, the two factors which nowadays govern medical pathology.

Nowhere could I have discussed these questions of nervous and general pathology before an audience better prepared to hear them. You belong to a nation which has achieved conspicuous distinction in general medicine, and especially in neurology. Do you not reckon among your great men of the past Sydenham, Fothergill, Charles Bell, Brodie, Abercrombie, Parkinson, Todd, Graves, Gull, Waller, Little, Lockhart Clarke, and Sir William Broadbent, who died recently, and who was the first President of the English Section of the Entente Cordiale Médicale? And at the present time can you not be justly proud of your brilliant phalanx of neurologists, who would not forgive me were I to sing their praises? In the name of French medical science I greet them as the worthy successors to a glorious past.

An Address

ON THE

PRESENT POSITION OF ABDOMINAL HYSTERECTOMY FOR FIBROIDS IN LONDON.

DELIVERED TO THE GLOUCESTER BRANCH OF THE
BRITISH MEDICAL ASSOCIATION,

BY JOHN BLAND-SUTTON, F.R.C.S.ENG.,

SURGEON TO THE MIDDLESEX HOSPITAL; SENIOR SURGEON TO THE
CHELSEA HOSPITAL FOR WOMEN.

It is surely a triumph of surgical art to improve an operation which in 1896 was attended with such a high mortality that few surgeons performed it, and then only in highly favourable, uncomplicated, and carefully selected cases; yet by 1906 the mortality of this operation in some hospitals in London had fallen to less than 2 per cent. This is the history of abdominal hysterectomy for fibroids in London.

In the year 1896 the results of abdominal hysterectomy for fibroids in eight hospitals in London were as follows:

St. Bartholomew's	7	with 3 deaths.
St. George's	1	0 "
St. Thomas's... ..	5	" 2 "
Middlesex	6	" 1 "
University College	3	" 0 "
Hospital for Women (Soho) ...	1	" 0 "
Samaritan	17	" 4 "
Chelsea Hospital for Women ...	9	" 1 "
	49	11

Ten years later—that is, in 1906—the latest returns from the same sources, and the New Hospital for Women, are:

St. Bartholomew's	26	with 4 deaths.
St. George's	8	" 0 "
St. Thomas's... ..	40	" 2 "
Middlesex	50	" 0 "
University College	21	" 1 "
Hospital for Women (Soho) ...	60	" 1 "
Samaritan (for Women)	37	" 2 "
Chelsea (for Women)	80	" 1 "
New Hospital for Women	26	" 0 "
	348	11

In regard to my own results: During the years 1906 and 1907, at the Middlesex Hospital and the Chelsea Hospital for Women, I performed abdominal hysterectomy for fibroids on 101 patients; they all recovered.* I performed abdominal hysterectomy in these two hospitals for other morbid conditions and the patients recovered.

* The short notes of the 101 hysterectomies for fibroids, and the subsequent history of the patients, are published in the *Journal of Obstetrics and Gynaecology of the British Empire*, May, 1908.

The notes concerning the operations in this series have been compared with the hospital books by the Registrars, Mr. Somerville Hastings, F.R.C.S. (Middlesex Hospital), and Mr. Stanley Dodd, M.B., M.A. (Chelsea Hospital for Women), and these gentlemen allow me to state that they have verified them.

It will be gathered from a perusal of these statistics that the removal of the uterus by the abdominal route, when it is occupied by troublesome fibroids, has become a fairly safe proceeding, nevertheless, hysterectomy, like all other major surgical operations, is attended with risks, even when it is carried out with all the careful surroundings which form such a prominent feature of modern surgical operations.

Setting aside the risks and sequelae common to operations in general, such as dangers from the anaesthetic, pulmonary complications, and the like, there are a few special dangers connected with it which require careful consideration at the hands of those who are daily occupied with this class of surgery. In this communication I shall deal with some of them in detail. I shall also consider the question which is much debated at the present time—namely, the advantages and disadvantages of the two methods in vogue for removing the uterus, subtotal and total hysterectomy, especially in connexion with an important matter, the occurrence of cancer in the cervical stump left after subtotal hysterectomy. The fate and value of belated ovaries is also well worth consideration, for some recent writers in America seem to attach little importance to the retention of ovarian tissue.

In addition to careful statements on these matters, which are of high importance for the subsequent comfort of patients, I propose to discuss certain matters concerning the pathology of uterine fibroids in relation to carcinoma and sarcoma, and to some secondary changes which affect them, especially in relation to pregnancy. These observations, more or less of a pathological kind, have become of great value in assisting us in the clinical side of our work; and they have helped to convince even the most experienced that "fibroids," which were commonly supposed to be the most easily recognized species of pelvic tumour, are not only simulated by other tumours, but in the secondary changes to which they are prone, mimic other well-known morbid conditions of the internal pelvic reproductive organs and lead to startling errors in diagnosis.

It is no part of my intention this evening to discuss details of technique, because the operation of hysterectomy, as a radical method of treating uterine fibroids, is now practised extensively throughout the civilized world; but I am anxious to put in a plea for the employment of sterilized rubber gloves by those who perform this operation or take any part in it, whether they be surgeons, assistants, or nurses. It not only reduces the mortality of the operation to a minimum, but the unpleasant sequelae are almost abolished also, especially trouble with the ligatures and sutures.

There is a risk which attends the total removal of the uterus, whether by the abdominal or the vaginal route, which should be referred to, namely, the liability of injuring one or both ureters. This subject I have discussed recently so that I need only refer to it here, but I mention it because it is an accident which demands the anxious consideration of all surgeons who perform hysterectomy.¹

Unpleasant sequelae, and especially that form of sudden death during convalescence which is usually attributed to pulmonary embolism, are more frequent in the practice of those who habitually operate slowly. It is a natural inference that a patient whose operation is completed in thirty minutes by a dexterous and careful surgeon, has a better chance of recovery and less liability to post-operative complications than the patient whose operation is performed by one who requires two hours for a simple hysterectomy. Everyday experience teaches that celerity in operating counts in favour of the patient.

TOTAL AND SUBTOTAL HYSTERECTOMY.

The great success which followed the use of the short ligature in ovariectomy induced several surgeons to apply the same principle to the cervical pedicle when removing the uterus for fibroids. The result was dismal failure. Matters improved somewhat after Koeberlé introduced the serre-neud, and this continued the safest method until