AN ADDRESS

DELIVERED AT THE OPENING OF

THE SECTION OF PATHOLOGY.

At the Annual Meeting of the British Medical Association, held in Newcastle, August, 1893.

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DEFENSIVE MECHANISMS.

GENTLEMEN,—It is my first and most pleasant duty to express my thanks to the British Medical Association for having done me the honour of appointing me President of this Section. In undertaking the honourable duties of this office I was well aware that they do not necessarily include the preparation of an introductory address. In the case, however, of a subject which is advancing with such bewildering rapidity as is the pathology of the present day, it is, I believe, possible that a short review of some subject of general pathological interest may sometimes be of use on an occasion such as the present.

All departments of pathology have undergone great development in recent years, in none perhaps more strikingly than that part which concerns itself with the parasites which we now know to be the causes of so many diseases. But although bacteriology offers plenty of scope for an address of this kind, I consider that it will be more profitable if I deal with a subject which is to my mind equally interesting and important, and which has been a little apt to be overlooked in recent years in the attention which we have all given to the microbic causes of disease. The part which I wish to deal with is that part of pathology which concerns itself, not so much with the causes of disease, as with the means of defence which the system possesses for counteracting and warding off possible sources of injury or death to the body.

Now these DEFENSIVE MECHANISMS can be distinguished by this, that they are only called into activity when a part or whole of the system is endangered. They sometimes constitute reactions of mechanisms which are in ordinary use in the normal unendangered animal, but in other cases they are due to the activity of an anatomical substratum which, so far as is known, acts only when the system requires to be defended.

Another characteristic of the protective mechanisms consists in the fact that they are most numerous and most highly differentiated in those parts of the body which are most liable to attack. Some of them, moreover, are for local defence, and serve to protect individual systems of the body, while others protect the economy as a whole. Let me, for illustration' sake, refer to a few of the defences of the body which have to protect a particular part. I might take any or all of the systems of the body, but for shortness' sake will confine what I have to say for the present to the respiratory and digestive systems. The lungs and air passages are obviously open to injury in a variety of ways.

It will be sufficient for my purpose if I refer to the dangers to the system which may result from interference with the bronchial tubes. Through them with each inspiration passes a current of air bearing with it under ordinary conditions microbes which in a moist, warm, organic medium, such as the fluid which bathes the bronchial mucous membrane, are capable of growing, and of producing harmful chemical substances. To protect the system against these we have been provided by Nature with a host of defensive means.

In the first place, there are being continuously extruded between the epithelial cells vast numbers of eosinophile and other leucocytes, which, as in the process of inflammation, destroy and digest any microbes with which they come in contact; while at the same time the conflicting cells and microbes with the chemical products of the latter are continuously being conveyed upwards out of harm's way by the movements of the cilia of the epithelium. Over and above this, however, the air passages are protected by a number of other mechanisms, which only come into play, so far as is known, when there is special necessity for defence against mechanical or chemical substances—by far the most important of these latter being those produced by microbes which have gained some headway against the protecting leucocytes. The bronchial membrane can, at any moment, be rapidly covered by a layer of mucin-bearing fluid, the viscidity of which is apparently of importance in preventing the penetration to the epithelium of both mobile and non-mobile micro-parasites. The accumulation of mucin, with the irritant substances which have caused its secretion, can then be expelled by two mechanisms which would not profitably be called into play in the absence of an increase of the amount of fluid which is normally present on the surface of the bronchial membrane.

These are, firstly, the *coughing* process, which has a co-ordinating centre in the spinal cord, and secondly, the contractions of the bronchial muscular layer. These and other mechanisms then defend the air passages, but, leaving out of account the protective arrangements in the lungs themselves and in the upper air passages, there is yet another and important mechanism which protects the system against an imperfect supply of air, whatever be its cause.

In the phenomena accompanying dyspnœa or asphyxia, we find an elaborate arrangement in which the nutrition of those parts of the body which can be temporarily interrupted is so interrupted, the available oxyhæmoglobin being sent only or chiefly to those organs, such as the central nervous system and heart (whose functions cannot without danger to life be even temporarily interrupted), and to those respiratory and other voluntary muscles which may require to be used to avoid or combat the cause of the dyspnœa or asphyxia. You will please to observe that I am by no means attempting to give a complete list of the defensive mechanisms connected with the respiratory apparatus. I am only giving enough to serve a particular purpose, which I will presently make clear.

Still less will I try to detail the manifold protective mechanisms by which the digestive system is hedged round, and, indeed, it will suffice if I recall to you the importance to the system of *vomiting* in ejecting from the system harmful substances which have found their way into, or been produced in, the stomach; or, again, the increased peristaltic action of the intestines, which, in ordinary diarrhœa, frees the system from peccant matters; or, again, the secretion by the gastric glands of hydrochloric acid, whose value as an antiseptic in destroying microbes which have been swallowed with the food we have recently learned to recognise.

Of greater interest than any of the protective mechanisms which I have just briefly referred to are the great defensive processes by which all the tissues of the body are defended from injury or death by the invasion of pathogenic micro-parasites. Among these, INFLAMMATION takes a prominent place, and on this subject I should like to say a few words. In the first place I must remind you that the much-abused term "inflammation" can no longer be so freely used as has hitherto been the case, and that we must now exclude from this species many pathological processes which have in the past been considered and called inflammations. For instance, many of the so-called chronic interstitial inflammations are, we now know, due to local anæmia, from narrowing of the blood vessels or other cause, whereby the blood supply is rendered insufficient to maintain the relatively active metabolism of certain tissue elements, which are replaced by fibrous tissue whose nutritive changes are less active. The same is the case with most, if not all, of the parenchymatous inflammations, which are mostly due to malnutrition, or to chemical destruction of the functionally active tissue elements, and which in many cases leads to their being replaced by fibrous tissue, in other words, to interstitial inflammation so-called, following so-called parenchymatous inflammation. We must also exclude from the term those processes of repair of tissues where the increased nutritive activity simulates the true inflammatory process.

Leaving these and other cases which I might mention aside, we find that at the present day the inflammatory process is narrowed down to the results of a localised invasion of pathogenic micro-organisms. How large a field this covers it is unnecessary for me to specify here, and I will only remind you that, although by chemical and other means a process apparently identical with what may be called the normal inflammatory reaction can be produced, yet that we must look upon these as cases where the inflammatory pro-

cess has been called for by other than what we may safely describe as its legitimate cause—the cause, that is to say, to meet which it has been evolved in the animal kingdom, namely, a local invasion of pathogenic microparasites.

Now, as regards the process itself, it is essentially to the physiologist a local increase in metabolism. The heat, redness, and swelling cannot be distinguished in kind from the nutritive congestion and the other phenomena which accompany localised increase in metabolism. It is distinguished from this, however, by the pain and hindrance of function, but the congestion and the exudation of plasma are without doubt due to the mechanism which normally regulates the supply of nutriment to a tissue in accordance with the requirements of that tissue. Besides the pain, to which I will presently have occasion to refer under another heading, *inflammation* is distinguished from the increased metabolism which results from such a cause as warming a part of the body a few degrees above its normal temperature by the presence and behaviour of the leucocytes. These, as you know, leave the vessels in increased numbers at the seat of inflammation. That this is no accidental thing resulting from the congestion or other cause is shown by the fact that the leucocytes of the blood are increased in number and in the inflammation due to some microbes, namely, the pyogenic ones, enormously so.

Kanthack and Hardy have found that the amœboid cells which first appear at the seat of invasion are distinguished by the fact that they contain a large number of granules which have a very characteristic appearance, and which Ehrlich has named, from their colour reactions, eosinophile granules. Kanthack and Hardy have shown that, in the presence of certain pathogenic microbes, these granules leave the leucocytes in which they have been formed, and, being dissolved in the lymph or plasma, in some way injure the vitality of the microbes with which they have come in contact. These observers have shown that the leucocytes which form and extrude eosinophile granules may be looked upon as unicellular amœboid glands, which play a *rôle* of apparently great importance in protecting the system against the invasion of pathogenic microbes. They have shown us also how we can bring into a harmonious whole the two opposing theories of a defence of the system against microbes by means of chemical substances present in the plasma and lymph, and the great work of Metschnikoff, which seemed at first to indicate that the system is defended against microbic invasion by phagocytic digestion only.

Kanthack and Hardy have never, so far as I am aware, for one moment disputed the importance of phagocytosis. What they find is that there is something more than this, and their facts are obviously fitted to explain what we know of the bactericidal and globulicidal action of the serum, apart altogether from the living cells which it may contain.

The enormous numbers of eosinophile cells in the early stages of the inflammation, due to many of the pathogenic microbes, is of itself an evidence of the importance of the part played by these cells in defending the system. Of the importance in inflammation of the process of phagocytosis it is unnecessary for me to speak. Metschnikoff's work on the subject is now being more fully understood than was at one time the case, and its limits are gradually being defined.

Much still remains to be done on the functions and relationship of the different forms of leucocytes, and it may well be that some of these cells have as their function the production of a substance which has the power of oxidising or otherwise destroying the toxins which are produced by pathogenic micro-organisms, and by which they tend to destroy the tissues of their host.

Other leucocytes, again, possibly take part in the process of regeneration which so often follows on inflammation proper. Besides this elaborate system of local protection there is, as you know, a second line of defence in the lymphatic glands.

I have said that PAIN is one of the features of inflammation which distinguish it from increased metabolism due to other causes than the invasion by microbes or other influence which simulates such an attack. In *pain* we have another of the great protective mechanisms of the body which is distinguished from some of the others, in that it has a fixed anatomical substratum apart altogether from the tissues which regulate the usual nutrition or specialised function of the economy.

The nerves which convey the impression of pain are, as we know, distinct from those which convey tactile impressions. Their function is, without doubt, to call into play the powers of the system as a whole, in order to aid in protecting against local attack. The reflex to the voluntary muscles we know of from physiologists, but in the case of the pain of inflammation the gain to the system is by no means so obvious; yet no one who has studied the animal economy with any care will believe *a priori* that the throbbing pain of an inflamed part is a profitless and gratuitous torture to the individual. That this is not so need hardly be said. Continuous pain, as Hilton has shown, acts differently from temporary pain, which calls into play reflexly the voluntary muscles; the continued pain of inflammation tends to inhibit the muscles, which would otherwise be reflexly excited, and calls for *rest*. This is by no means the only case where a defensive mechanism over-rides what is usually called normal function.

But the centripetal influences which pass from an inflamed part to the nerve centres do more than call the attention of the patient to danger, and do more than demand at the same time rest for the affected tissue. The vasomotor system is also called into play, and responds by cutting off or diminishing the blood supply of those organs or tissues whose nutrition can, without great harm to the body, be temporarily arrested in order that the endangered part may receive the increased blood supply which is required to meet the augmented metabolism. Whether the vasomotor centres are called into play by those nerves which convey impressions of pain, or by others which are intended solely to regulate nutrition, is not at present known, and in any case does not concern us here.

Inflammation, as I have said, calls into play in pain a protective reminder to the consciousness, and also calls on the vasomotor mechanism to provide the increased blood supply which is required for the active metabolism which is inseparable from the inflammatory process. As I have said, the lymphatic glands constitute a second line of defence; but the defences of the economy in respect to microbic invasion do not appear to be limited to these. Within or above them there is the febrile process, on which I must here say something. FEVER has been looked on by physiologists as an accidental derangement of the mechanism by which the temperature of warm-blooded animals is regulated—as a profitless and harmful process—in other words, as a part of the price which we must pay for the complicated mechanism by which heat regulation is carried on in the normal animal. This view does not recommend itself to pathologists, whose knowledge of the causes of fever is rapidly extending, and who are *a priori* disposed to see something more than accident in a reaction which so commonly accompanies invasion of the body by certain forms of pathogenic micro-organisms, and which goes hand in hand with the struggle between the system and the invading parasites.

Here I must say a few words regarding the causes of fever. Most fevers are associated with the presence in the system of microbic poison. There is another and quite distinct category of fevers—those due to injury or derangement of the nervous system—and on these I will have a few words to say presently, and will try to show that the existence of these does not really modify the fact that fever is essentially a reaction to the poison of certain microbes. How many in number these latter are I need not stop to say, and will only note that they appear to produce a considerable number of chemical substances which can produce fever.

Of the chemical composition of the fever-producing poisons not very much is known, but Sidney Martin has found that some pathogenic microbes form a fever-producing *albumose*, others an *acid organic body*, and others again a *basic organic body*, both of which produce fever on injection into the system.

We know, then, that all fevers are not due to the same chemical substance, but we do not yet know the full number of those chemical products of microbic life which can cause it. We know, however, that the fever-producing substances may some of them be distinguished from the toxins which are produced by most pathogenic microbes, and to the action

of which is presumably due the difference in the characters of ordinary infective fever, as compared with the fever due to derangement or disease of the nervous system.

The *fever process* itself appears to be the same in all cases. The rise in temperature of the body seems, if we are to accept the work of Rosenthal and his pupils, to be simpler in nature than has generally been supposed, and to be primarily due to a diminution in the amount of heat given off by the skin during the cold or initial stage when the temperature begins to go up. As soon, however, as the temperature of the body rises above the normal, this of itself causes increased metabolism and production of heat, while the amount of heat given off by the skin at the height of the fever may be greater, or may be less, than was given off by the same animal before the fever was produced. Rosenthal's facts have this great recommendation, that they bring the increased heat production and metabolism of fever into satisfactory accordance with what we know of the mechanism for heat regulation in the non-febrile individual, and do not require us to assume the existence of trophic nerves, which were by some supposed to be capable of directly increasing the oxidation of the tissues, and apart altogether from increase in their functional activity. Rosenthal's work is specially interesting in connection with the observations of Pembrey, who has worked recently in Burdon Sanderson's laboratory on the effect of lowering the temperature on the heat production, which, as we know, causes an increased production of heat, supposed by many to be due to the action of trophic or thermic nerves, causing increased metabolism. Pembrey finds that in the chick (with the eggshell still unbroken) before the muscles are capable of contracting, the effect of lowering the surrounding temperature is a diminution of the metabolism, and therefore the heat production, exactly in the same way as lowering of temperature affects a cold-blooded animal, and that the increased heat production in man and other warm-blooded animals, when they are exposed to cold, is apparently due to the shivering and other muscular contractions which take place under these conditions.

We see, then, that both the normal heat regulation and the increased heat production of fever can be satisfactorily explained without requiring to go beyond the simple facts of what has long been known regarding the heat regulation by the muscles and skin, and what is equally well known regarding the effect of a rise in temperature increasing the metabolism of the tissues as a whole.

The other phenomena of fever, the increased respiratory exchanges of gases in the lungs, and the increased amount of the products of metabolism in the excreta can all be explained by the rise of temperature, as can also the changes in the heart's action and in the circulation as a whole.

Fever occurs, so far as is known, in all warm-blooded animals, and apparently also, under certain conditions, in some cold-blooded animals as well. It occurs most readily and certainly as a result of the invasion of those microparasites which most frequently invade the body and cause disease, namely, in those which cause catarrh and suppuration.

Pathologists have learned to look upon it as a measure, in some respects, of the struggle between the system and the invading parasite. It is not a measure of the virulence of the invading microbe, for very often the more severe the symptoms of poisoning by a given parasite the less does the febrile reaction show itself. Fever, again, is characterised by this, that in a given case of an infective disease, such as measles or typhoid, the rise of temperature is confined within comparatively narrow limits, which is not what we would expect were fever an accidental derangement of the heat regulating mechanism.

But it may be urged that if fever be essentially a defensive mechanism, which the animal economy has gradually evolved and which helps it to cope with the microparasites which most frequently invade it, surely there ought to be no difficulty in producing direct evidence of this. Nor is there, I believe, any insurmountable difficulty in finding such direct evidence of the defensive nature of the febrile reaction.

I will not seek to give more than one or two examples—that, first, of *Asiatic cholera*, in which disease it has been commonly noted in the last, as in previous, epidemics that some cases had a fairly marked fever, and others little or no rise of temperature, and that in cases of apparently equal severity those with fever have much more chances of recovery than non-febrile cases. Another, and perhaps better, evidence of the benefit to the system of the febrile process, is to be found in the fact that, in the case of certain animals which are normally immune to certain pathogenic microbes, this immunity disappears if a dose of antipyrin be given at the time of the inoculation, or if, by cold applied externally, the temperature of the animal be kept low. I might give other examples to the same effect.

But I am not concerned here in trying to convince you that fever is essentially a protective mechanism, nor am I by any means sure that *complete* proof of such a view is at present producible. I am rather seeking to indicate to you the general line along which our knowledge of fever is, as it seems to me, developing. Nor are we at present in a position to say in what way the system is aided by the febrile reaction. It is possible, as Ehrlich finds from the colour reactions of certain tissues in the bodies of febrile as compared with non-febrile animals, that in the former there is not only increased oxidation going on, but also increased power of oxidation, so that substances, such as possibly some *toxins*, which would remain undestroyed at the normal temperature, can be oxidised during the fever process. It may also be that the rise of temperature increases the activity of those cells which produce antitoxins, or of those which acts as phagocytes, or it may be of both kinds.

But if we are to take some such provisional view of ordinary infective fever, namely, that it is a defensive or protective process, what view can we take as to the nature of the fever which is due directly to the nervous system in the absence of any poisous to be combated or destroyed? It is obviously possible that nervous fever, such as sometimes occurs in injury or disease of the upper part of the spinal cord, as well as in other ways, is due to an accidental derangement of the heat-regulating mechanism. I do not know of any gain, direct or indirect, that these fevers can bring to the system. They seem to come under the pathology of fever rather than its physiology, if we are to assume (as we must) that the heat regulation is mainly governed by the nervous system.

Before I leave the subject of fever it may be as well to note that the frequent association of fever and inflammation points—since the latter is unmistakably protective—to the probability that the former comes under the same category.

It would require a chapter for itself were I to deal at any length with the interesting subject of the relation which exists between defensive and nutritive mechanisms in the annual economy. In the "functio læsa" of an inflamed part, in the weakening of the digestive process in fever, or in the combustion of the tissues in the same process, in the indirect injury to nutrition of pain, in the rejection of food by the stomach as well as harmful substances when the latter are present, in the phenomena of asphyxia, etc., we will find no want of evidence that the processes of nutrition are habitually subordinated to those of defence when the welfare of the system, as a whole, requires such temporary subordination. In the animal economy, as in communities of men or certain animals, the ordinary avocations of daily life are suspended as far as may be whenever it is necessary to employ the whole powers of the economy or community to render defence against attack effective.

I come to a point when I can say a few words on the general position of the protective mechanisms in the economy. Their importance cannot, I imagine, be logically denied. Some idea of their place in Nature may be gathered from a short consideration of their history in the animal kingdom as compared with that of nutrition and reproduction, which are in any case first postulates in animal as well as vegetable life. It would be too much to expect me to go deeply into this subject here, even were the materials at my disposal sufficiently complete and well arranged in my own mind to allow me to do so with any satisfaction. The work of Metschnikoff and others has, however, shown that defensive mechanisms can be traced almost as far back as the history of animal life. In the amœba can be found phagocytosis which seems to be more than nutritive, and in the ordinary reactions of amœboid protoplasm we see functional characters which show the existence of automatic defensive mechanisms.

In the more differentiated protoplasm of the tissues of the invertebrata as a whole we find that protection from attack has differentiated and developed not less fully than have the mechanisms of nutrition. For example, to take one of many, in the earthworm, as Lim Boon Keng has recently found, the protective mechanisms have reached a very high degree of development, not only in the presence in their cœlomic fluid of phagocytes and eosinophile cells, which we have learned to associate with defence against microbes in the case of animals more highly placed in the scale of organisation, but also in other ways.

In higher animals, amongst the vertebrata, the elaborate organisation for defending the system against attack makes itself manifest, as I have, in my previous remarks, sought to remind you. In the evolutional history of the animal kingdom, then, defence advances more or less equally with elaboration of nutrition and reproduction.

Can we perhaps learn anything of the position in Nature of the protective mechanisms from their pathology, that is, from those cases where they are called into play uselessly and accidentally, and where they interfere with normal nutrition or reproduction they are harmful? The purely nervous fever of which I have already spoken, or the pain of neuralgia where nothing is gained from the pain, or the asthma which constricts the bronchial tubes to a degree which has no apparent connection with the importance of the cause of the asthma, or the vomiting of pregnancy which—of no apparent benefit even in ordinary cases—may be in some instances so excessive as to endanger the patient's life, or the cough which from a trivial cause may be so persistent as to prevent the patient sleeping, and thereby reduce his strength to perhaps a dangerous degree—these and many other examples will illustrate what I mean by the pathology of the protective mechanisms. Pathologists have some of them an idea that those functions of the body which are most likely to go wrong are less fundamentally important in the economy than those which are less liable to derangement—that they are less old in the history of the development of the animal kingdom. I am not sure that this idea has ever been clearly expressed, or that, if expressed, it can be proved. I may, however, say that I do not think that if looked at in this light the defensive mechanisms will be found to come in importance far behind those of nutrition and reproduction.

It would rather seem that the liability to derangement in the protective mechanisms, as in those of nutrition and reproduction, advances with the elaboration of the organisation of these respective mechanisms.

It will not, I trust, be out of place if I remind you that questions such as I have imperfectly sketched in the above remarks have an important bearing on the treatment of disease as we meet with it at the bedside, and that a medical man who unwittingly seeks to treat a protective reaction as a disease may put himself in the unpardonable position of the inexperienced whist player who trumps his partner's "best card," or, still worse, who plays against his own partner.

As a last word I may note that in what I have said I have drawn no sharp line between physiology and pathology. I have not sought to show that between health and disease there is a boundary that can be delimited. I do not believe that such a boundary can naturally be defined, for I am of opinion with Virchow that "pathology is physiology."

The Duke of Westminster has sent a donation of £500 towards the funds of the Metropolitan Hospital, Kingsland Road.

The Certificate in Psychological Medicine of the Medico-Psychological Association has been granted to the following after examination held July, 1893:—Aberdeen—A. Low, P. J. Henderson, J. W. Myers, A. Rose, C. G. Cowie. Edinburgh—S. Edgerley, J. Macmillan, R. S. S. Strong, L. Grant. Glasgow—D. R. T. Strong, R. D. Hotchkis.

The St. John Ambulance Brigade on the Royal Wedding Day.—The Home Secretary has sent a communication to Sir Edward Lechmere, testifying to the excellent work done by the St. John Ambulance Brigade in London on the occasion of the recent royal marriage. The force employed consisted of 200 ambulance officers and men, 10 surgeons, and 30 nursing sisters; 1,544 cases were relieved.

AN ADDRESS

DELIVERED AT THE OPENING OF

THE SECTION OF OPHTHALMOLOGY.

At the Annual Meeting of the British Medical Association, held in Newcastle, August, 1893.

By G. E. WILLIAMSON, M.A., F.R.C.S.,
Honorary Surgeon and Surgeon to the Eye Department, Royal Infirmary, Newcastle-on-Tyne.

ON REFRACTION TESTING AND ON MEDICAL EDUCATION IN OPHTHALMOLOGY.

Gentlemen.—I have great pleasure in offering you a hearty welcome to Newcastle, and I assure you that no effort on the part of the local men will be wanting to make your visit both pleasant and profitable.

This was all I intended to say, but, on thinking over the matter, two subjects came into mind which I am sure must also have occupied the attention of members of the Section.

The Testing of Refraction.

The first is the question of testing for glasses in refraction cases. The great spread of education, the increasing knowledge of the people, and the large amount of minute work due to division of labour, have brought home, even to the working classes, the necessity of having vision aided when it is not perfect. In addition to this, sight tests are exacted in various departments of labour. Now the question I wish to raise is, Who is to cope with the rapidly increasing mass of testing work that has now to be done? The members of this Section will, I am sure, agree with me in answering that it must be done by ophthalmologists. A great part has hitherto fallen to opticians and jewellers, but it is clear that if they are still to do it they must have some special training in the subject, so that the matter is not left to hazard. But even if they are trained as far as it is possible, there are innumerable cases with which they are unable to deal, and where there is disease they are bound to fail. I would urge that every person should have his eyes examined, tested, and reported upon, so that he may know their value and strength as optical instruments.

The public generally are not aware how far the mechanism of the eye is understood, how thoroughly its interior can be explored, how accurately its defects can be gauged, and how often those defects can be remedied. If some knowledge of optics were general, we should not need to insist upon this, nor should we see the public deluded by specious advertisements of certain kinds of glasses superior to any others. The prejudice against glasses, which in some cases is so strong that a man may not wear them at work, even when they give him perfect sight, would disappear, and the increase of those using lenses would be in proportion to increasing knowledge and civilisation. Indeed, what with the growing use of the eyes for close work, and with scientific zeal in correcting errors of refraction, we may ultimately reach a position in which a man who goes about with his eyes naked will be so rare that the sight of him will almost raise a blush. We should, I think, urge upon medical men generally the desirability of sending patients with whose defective vision they are themselves, perhaps, unable to cope, to oculists, as the public naturally receive a wrong impression when educated and qualified practitioners recommend them to be tested by opticians, or even to proceed to a jeweller's to select for themselves the glasses with which they see best. Take, for instance, those cases of refraction headaches which are now so frequently referred to us by physicians; many of them have good vision, and protest that they require no aid; yet the wearing of correct lenses relieves their headaches, and restores to them the full use of their eyes, I need not cite the instances of disease that are so constantly coming under our notice in those who think that all they require is the aid of a pair of spectacles. Whilst fully recognising the trouble taken by many opticians, I think that their part of the work will ultimately be the carrying out of orders; and this will come the sooner when everyone, before he begins his life's work, has his eyes seen to and reported upon by the ophthalmologist.